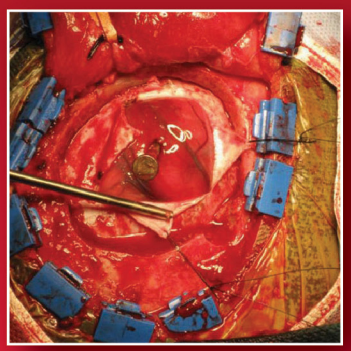

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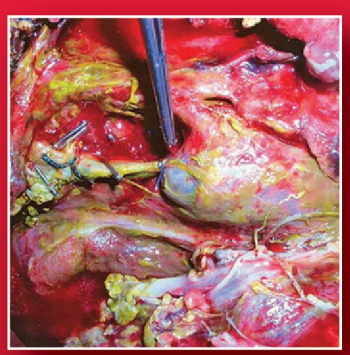

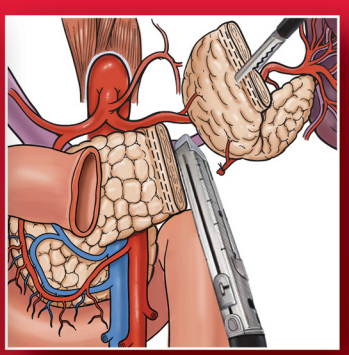

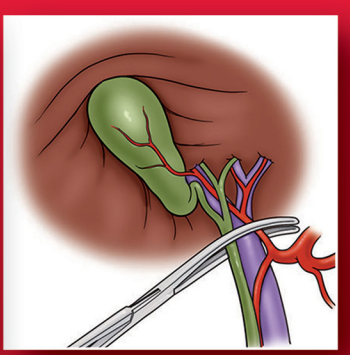

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The Founding Co-Editors dedicate the Ninth Edition to our families; to our surgical and medical colleagues; to our former fellows, surgical residents, and medical students; and to fallen and wounded warriors.

To my wife, Grace F. Rozycki, MD, MBA, for her unwavering love and support.
To my sons, David Joseph Feliciano and Douglas Donald Feliciano, JD.
In memory of my parents, Vincent Feliciano, MD, and Anita Hessler Feliciano.

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To my wife, Camella June Mattox, whose love and support began even before medical school. To my daughter, Kimberly Renee Toth, and her husband, Dan Toth.
To my grandchildren, Charlie, Alex, and Kelsey.
In memory of my parents, Bill and Carrie Mattox.

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To my wife, Sarah Van Duzer-Moore, MD, for her unconditional love and support.
To our sons, Hunter B. Moore, MD, PhD, and Peter K. Moore, MD, and daughters-in-law, Brook Brenneman and Tiffany Tello, MD.
To our granddaughters, Estelle and Simone.
In memory of my parents, Ernest Moore, MD, and Sarah Moore.

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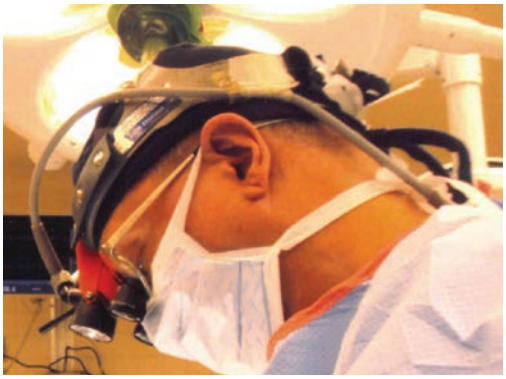

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PREFACE

This Ninth Edition of *Trauma* is another milestone in the now 35-year journey of the textbook. Originally conceived at a time (1984) when prospective randomized and multicenter studies were rare in the field, the management of injured patients was based on the following: history/physical examination/routine laboratory tests/routine x-rays; decreasing role for diagnostic peritoneal lavage; increasing usage of computed tomography (CT); and clinical guidelines passed on from teachers and mentors.

The evolution in the field of trauma since that time has been extraordinary. The following advances have truly changed the evaluation and management of injured patients during the history of the textbook: goal-directed resuscitation; surgeon-performed ultrasound; multidetector CT and CT angiography; magnetic resonance imaging; nonoperative management of injured viscera and vessels; damage control surgery; endostents and stent grafts; advances in surgical critical care and increased numbers of surgeon-intensivists; and the development of a formal training pathway in acute care surgery.

The major issue for the Founding Co-Editors (D.V.F., K.L.M., E.E.M.) when organizing and editing a "new" textbook every 4 years or so has been updating the contents enough to justify another edition. Changes in the Ninth Edition have been substantial, and many have been based on the comments from readers of previous editions. Most important has been the addition of six Associate Editors—Hasan Alam, Chad Ball, Kenji Inaba, Rosemary Kozar, David Livingston, and Marty Schreiber. All of these individuals have made substantial contributions to the knowledge behind evaluation and management of injured patients, all have been in leadership positions in trauma professional societies, and all were well known to us. We thank the Associate Editors for their efforts in making *Trauma,* Ninth Edition, an improved reference book in the field.

Other changes in the Ninth Edition include two new chapters, Chapter 28: Chest Wall and Lung, and Chapter 64: Trauma and Global Health; a reorganization of the chapters in the book; added content including a special section on injuries to the larynx in Chapter 25, now entitled Neck and Larynx; and revisions in the Atlas. In addition, there is now a section entitled Endovascular Commentary after Chapter 25: Neck and Larynx, Chapter 38: Abdominal Vessels, and Chapter 45: Peripheral Vessels. The Trauma Video section organized by Demetrios Demetriades and Kenji Inaba has been retained as well. Finally, over 45% of the chapters in the Ninth Edition have new senior authors.

We thank all of our chapter authors for updating the content and Mike de la Flor, PhD, for his excellent art work in the Atlas. Also, we thank our colleagues in the Medical Publishing Division of McGraw Hill, our longtime publisher, especially Andrew Moyer, Senior Editor, and Christie Naglieri, Senior Project Development Editor. And, of course, we offer special thanks to our administrative assistants for their endless work and tolerance: Josanne Walker (D.V.F.) and Mary Allen (K.L.M.).

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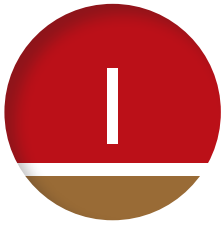

TRAUMA OVERVIEW

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1 Kinematics

Carrie A. Sims • Patrick M. Reilly

KEY POINTS

- A body in motion stays in motion, moving at the same speed and direction, unless acted on by a force.
- The energy of an object and its potential to do damage are directly proportional to its mass and the square of its velocity ($E_k = \frac{1}{2}MV^2$).
- The total energy of a system cannot be destroyed, although it may be transferred between objects or transformed into work.
- All injuries can be explained by four different types of strain (tensile, shear, compressive, and/or overpressure).
- If a force causes a decrease in the original volume of a fluid- or air-filled structure, there must be a concomitant increase in the pressure.
- The shape (and thus the sectional density) of a bullet is subject to change within the tissue and directly impacts the degree of tissue damage.
- A projectile creates both a permanent cavitation or "bullet track" and a hydrostatic pressure wave that stretches and damages adjacent tissues.
- In explosions, more energy is transferred if the shock wave travels through a medium of similar density (eg, water and the human body).

INTRODUCTION

Although patients suffer what initially may appear to be a unique constellation of injuries, all trauma can be described using physical principles. As such, understanding how the physical properties of force, mass, and velocity contribute to energy transfer can help the treating surgeon anticipate, identify, and subsequently treat injuries based on mechanism. This chapter will discuss the basic laws of physics that dictate the interaction between the injuring mechanism and the victim. This is followed by a discussion of blunt trauma, with special consideration of specific body regions and populations. Finally, the science of ballistics and the injuries caused by firearms and explosions are presented.

BASIC PRINCIPLES OF MOTION

In the late 17th century, the English physicist Sir Isaac Newton (1642–1727) first described how mechanical events could be explained using laws of motion and gravity. This section will provide a brief overview of Newtonian physics and how its principles directly apply to injury.[1]

Newton's first law is as follows: Every object persists in its state of rest or in uniform motion in a straight line unless it is compelled to change that state by forces impressed on it.

According to Newton's first law, a body in motion stays in motion, moving at the same speed and direction, unless acted on by a force. The state of being in motion, otherwise known as momentum (p), is described by both the object's mass (m) and its velocity (v).

$$p = mv$$

A motorcycle crash provides a classic example of this principle. Both rider and motorcycle have their individual mass and are traveling at the same velocity. When the motorcycle encounters an object, its velocity becomes zero. In contrast, the rider continues in a straight line until acted on by a force, usually the ground or another vehicle.

In order to change an object's momentum, a force must be introduced that either causes the object to speed up or slow down. When a force causes a change in momentum, it is referred to as an impulse. This is, however, a bidirectional exchange. A force causes a change in momentum and, conversely, a change in momentum will generate a force.

The momentum of an object is also a measure of its kinetic energy (E_k). In other words, the energy an object possesses is due to its motion and is directly proportional to both its mass and its velocity.

$$E_k = \frac{1}{2}MV^2$$

This principle can be used to explain the mortality differences from falls. For any given mass, a five-story fall is associated with greater velocity than a fall from standing. Therefore, higher falls are theoretically associated with more energy and, thus, more tissue damage on impact. This principle holds true until air resistance prevents further acceleration and terminal velocity is reached. For humans, this translates to a terminal velocity of 53 m/s and is achieved by falling 450 m in 12 seconds.

Newton's second law states the following: The acceleration of an object produced by a net force is directly proportional to the magnitude of the net force, in the same direction of the net force and inversely proportional to the mass.

Stated differently, force (F) equals mass (m) times acceleration (a), where acceleration is the change in velocity over the change in time.

$$F = ma$$

Newton's third law states: *For every action, there is an equal and opposite reaction*.

When two objects of equal velocity and mass strike each other, their velocities are reduced to zero at the moment of impact. Each exerts a force on the other and, because these forces are exactly equal and opposite, the net force is zero. Therefore, the net change in momentum is zero. This means that these two objects would change their direction and "bounce" in opposite directions at the same velocity and momentum if 100% of the energy were transferred. In real trauma scenarios, however, collisions are inelastic. Although they conserve momentum, their kinetic energy "does work" by deforming the materials they encounter.

Work (W) is defined as a force exerted over a distance, but can also be defined in terms of a change in kinetic energy. Therefore, the work being done by one object in motion on another equals the kinetic energy of the object before the interaction minus the kinetic energy after the interaction. In other words, the work done is equal to the change in kinetic energy of the first object. When this interaction sets the other body in motion, the second body now has kinetic energy of its own equal to the work that has been done, assuming no deformation. The greater the momentum of the first object, the greater the magnitude of work that can be done.

We can understand Newton's third law using the example of two cars colliding in various ways. Figure 1-1A represents a head-on collision of two vehicles with equal mass and velocity and, thus, equal kinetic energy and momentum in opposite directions. The total momentum for the system is zero prior to the crash and, by the law of conservation of momentum, must be zero after the crash. If the cars were made of a perfectly nondeformable material, all kinetic energy would be exchanged and the cars would bounce in opposite directions at the exact same speed. In reality, however, these vehicles will be deformed by this interaction. Because the final velocity is zero, all the kinetic energy is converted to work that stops the other car and causes deformation such as broken glass, bent metal, or physical intrusion into the passenger compartment.

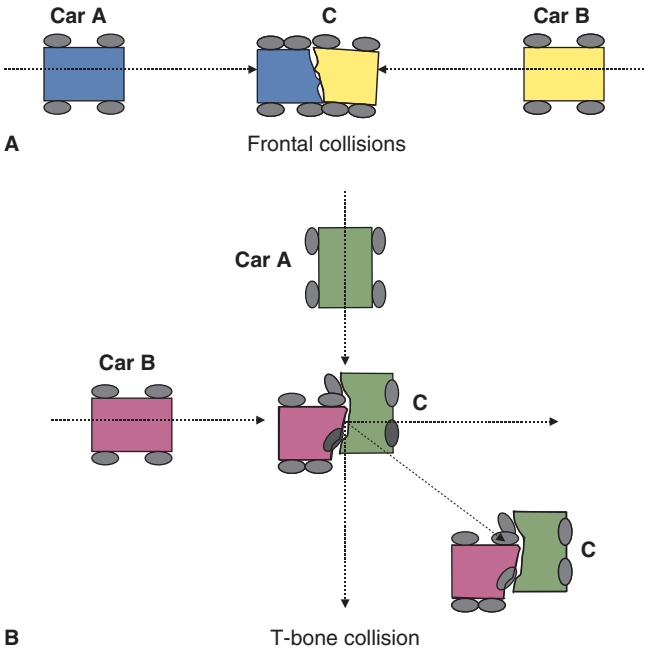

FIGURE 1-1 Energy and momentum available in various motor vehicle crash scenarios. (**A**) Frontal collisions have the greatest change in momentum over the shortest amount of time and hence the highest forces generated. (**B**) T-bone collision. When cars A and B collide, their resultant momentum directs them toward their final position C; the individual momentums in the x and y axes are dissipated over a greater time, resulting in smaller forces than in head-on collisions.

Assuming that both cars come to rest as a single entangled mass (referred to as object C), the change in momentum represents a force, which is equally applied to both cars. If the momentum of car A was greater than that of car B, the resultant mass C would have the momentum, and direction, of car A prior to impact. As such, the kinetic energy transferred to the occupants of car A would be less than that transferred to the occupants of car B. In order to illustrate this point, let's assume car A is an SUV and car B is a compact. The collision of these two vehicles results in predictable damage and consequences.

In T-bone type crashes, the directions of the momentum of cars A and B are perpendicular and momentum is conserved in a third direction, C (Fig. 1-1B). Because kinetic energy is partly conserved in this new momentum, less energy is transferred to the vehicles (and their occupants) with less deformity. Finally, in rear-end collisions, the energy exchange is a function of the net difference in momentum, not absolute momentum. The more momentum conserved by the

conglomerate of the two vehicles (mass C), the less energy is transferred into deforming the vehicles or their occupants.

STRESS, STRAIN, AND BIOMECHANICS

Stress and strain are two biomechanical principles that directly impact the degree of physical injury.[2] Stress, defined as a force per unit area or load, causes deformation of a given material. Strain, on the other hand, is the distance (or depth) of the deformation divided by the length over which the stress is applied. Injuries can be caused by four different types of strain as follows: tensile, shear, compressive, and overpressure (Fig. 1-2). Tensile strain occurs when applied forces are opposing and concentrated upon a *shared* point. As such, the integrity of the structure is literally pulled apart. Shear strain, on the other hand, occurs when opposing forces are applied to *different* points within a structure. Shear strain can either be caused by opposing *external* forces or arise from a relative difference in the change of momentum *within* a single structure (or between structures that are attached). Compressive strain is the direct deformation that occurs as a result of an impact. The energy transferred on impact does work on the structure, causing a crushing-type injury with disruption of the organ's structural integrity. Finally, overpressure is a type of compressive strain that is applied to a gas- or fluid-filled structure, causing deformation and a decrease in the volume of the structure. According to Boyle's law:

$$P_1V_1 = P_2V_2$$

the product of the pressure (P) and volume (V) before a force is applied must equal the product of these two variables afterward.[3] Therefore, if a force causes a decrease in the original volume, there must be concomitant increase in the pressure. This change in pressure (ie, force) could overcome the tensile strength of the structure, causing it to rupture.

How well a tissue tolerates a specific insult varies with the type of force applied and the properties of the tissue. The higher the density of a particular tissue, the less elastic it is and the more energy is transferred. For example, the lung is air filled, is extremely elastic, and tends to dissipate energy easily. In contrast, solid organs such as spleen, liver, or bone tend to absorb energy and will have greater tissue destruction as a consequence. Notably, air-filled structures such as the lung and bowel tend to be injured in explosions because the blast wave causes a localized pressure increase that overcomes the organ's structural failure point.[4]

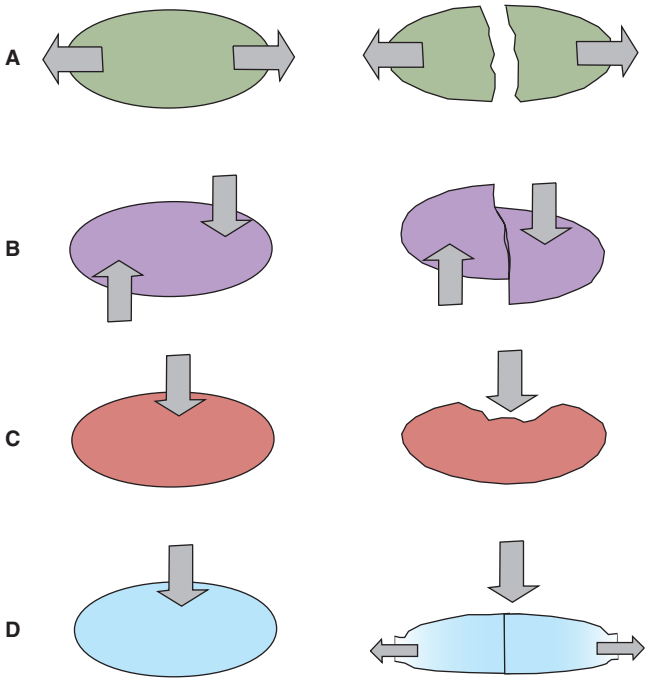

FIGURE 1-2 Biomechanical mechanisms of injury. (**A**) Tensile strain—opposite forces stretching along the same axis. (**B**) Shear strain—opposite forces compress or stretch in opposite direction but not along the same axis. (**C**) Compressive strain—stress applied to a structure usually causing simple deformation. (**D**) Overpressure—a compressive force increases the pressure within the viscus passing the "breaking point" of the wall.

BLUNT TRAUMA

The transfer of energy and application of forces in blunt trauma are more complex than in penetrating trauma. The most frequent mechanisms of blunt trauma include motor vehicle crashes, motor vehicles striking pedestrians, and falls from a significant height.

Mechanisms

MOTOR VEHICLE CRASHES

Understanding the changes in momentum, forces generated, and patterns of energy transfer between colliding vehicles can help predict how occupants of the passenger compartment will respond.[5,6] In frontal collisions, the front of the vehicle decelerates as the unrestrained front-seat passengers continue to move forward in keeping with Newton's first law. Lower extremity loads, particularly those to the feet and knees, occur early in the crash sequence. Because both the occupants and floor/dashboards are still moving forward, the relative contact velocity and change in momentum are still low. Contact of the chest, the steering column, and head with the windshield occur later in the crash sequence; therefore, contact velocities, change in momentum, degree of deceleration, and contact force are higher. Compression and continued movement of solid organs result in lacerations to the liver or spleen. Compression of the chest can result in rib fractures, a blunt cardiac injury, or a pneumothorax as the lung is popped like a paper bag. Finally, the sudden deceleration can cause shear forces on the descending thoracic aorta, resulting in a partial or full-thickness tear. As the driver is launched up and over the steering wheel, the head becomes the lead point, striking the windshield with a starburst pattern. The brain can sustain direct contusion or can bounce within the skull, causing

brain shearing and a contrecoup injury. Once the head stops, forces are transferred to the neck, which may undergo hyperflexion, hyperextension, or compression injuries, depending on the angle of impact. Once the head and neck stop, the chest and abdomen strike the steering wheel.

Lateral collisions, specifically those that occur on the side of the occupant, can be devastating because of the small space between the striking car and the passenger. If the side of the car provides minimal resistance (ie, no airbag), the passenger can be exposed to the entire change in momentum. These loads are usually applied to the lateral chest, abdomen, and pelvic acetabulum. As such, injuries to the abdomen and thorax are more frequent in lateral collisions.[7]

Rear-end collisions are classically associated with cervical injuries and provide an excellent example of Newton's first law. When the victim's car is struck from behind, the body, buttressed by the seat, undergoes a forward acceleration and change in momentum that is not experienced by the head. The forward push of the vehicle is associated with a sudden backward movement of the head, causing hyperextension of the neck. This injury pattern can also be seen in frontal collisions, where a sudden decelerating vehicle is associated with a continued forward movement of the head causing hyperflexion of the neck.[8,9]

Although there are frequently confusing vectors in a motor vehicle crash, mortality is directly related to the total amount of energy (or change in velocity) and direction of force. In a review of data collected by the Crash Injury Research and Engineering Network, higher speeds (40–80 km/h) were associated with greater mortality (17% vs 9%) and unrestrained patients were more likely to die than those who were restrained (17% vs 9%).[10] Interestingly, although frontal collisions have been historically associated with the greatest risk of death, new data suggest lateral impacts are actually more lethal (17% vs 11%).[9] Moreover, after adjusting for age, gender, and body mass index, the risk of death in a lateral collision has an odds ratio of 3.06.

In contrast, rollover crashes have a lower than expected mortality (<1%) because the momentum is dissipated and the forces projected to the passenger compartment are random.[11] That being said, mortality increases significantly regardless of mechanism if the vehicle's occupant is ejected. Ejected patients have the same velocity as their vehicle when they are ejected and typically strike a relatively immobile object such as the ground. When compared to restrained patients, ejected victims are three times more likely to sustain a significant traumatic brain injury, four times more likely to require admission to an intensive care unit, and five times more likely to die of their injuries.[12]

PEDESTRIAN INJURIES

Pedestrians struck by automobiles demonstrate a well-described pattern of injury depending on the size of the vehicle and the victim. Most adults struck by a car will have injuries to the lower extremities. If the car attempts to stop before hitting the pedestrian, the front bumper dips downward, striking at the level of the patient's knee. If the car does not stop, the force is transferred to the thigh. During the pedestrian–car interaction, the force applied to the knee (or thigh) causes an acceleration of the lower portion of the body that is not shared by the torso and head, which, by Newton's first law, tend to stay at rest. As the lower extremities are pushed forward, they will act as a fulcrum, bringing the trunk and head forcefully down on the hood of the car, applying a secondary force to those regions. As the car abruptly decelerates, the victim is then typically thrown to the pavement. This typical injury mechanism results in a tibia/fibula fracture, injuries to the torso such as rib fractures or a splenic laceration, and injuries to the skull and brain. If the patient is a child or if the vehicle involved is a truck, the transmitted force frequently results in serious injuries to the pelvis, abdomen, and/or chest.[13,14]

FALLS

Falls from height can result in a large amount of force transmitted to the victim. The energy absorbed by the victim at impact will be the kinetic energy at landing and reflects the potential energy prior to the fall, the height of the fall, the mass of the victim, and the gravitational acceleration. Given that the mass and gravitational acceleration are constant for the falling body, its kinetic energy is directly related to height. Therefore, the injuries sustained will depend on the elasticity of the ground and how the victim strikes.[3]

The majority of free falls in urban settings occur from moderate heights (≤20 ft). Fractures are the most common injury and occur in 76% of victims. Nearly 25% of victims sustain spinal column injuries, with 3.7% demonstrating a neurologic deficit. Intra-abdominal injuries are surprisingly rare.[15]

Specific Blunt Injuries

TRAUMATIC BRAIN INJURY

Traumatic brain injury (TBI) represents the single most important factor contributing to death and disability after trauma. TBI accounts for over 282,000 hospitalizations and over 56,000 deaths annually.[16] Our knowledge of the biomechanics of brain injury comes from a combination of experiments conducted with porcine head models, biplaner high-speed x-ray systems, and computer-driven finite element models.[17,18] Surrounded by a hard skull and bathed in a layer of cerebrospinal fluid, the brain is composed of different tissues with different densities. Loads applied to the head, therefore, result in different deceleration forces between the different components of the brain. For example, a brain contusion can result from direct impact and with associated compressive strain. The brain, however, is only loosely attached to the surrounding cranium. After a load is applied, the skull has momentum along the line of force but the brain lags behind. As the skull comes to rest (or even recoils), the brain is still moving along the line of the initial load and will strike the calvarium on the opposite side, creating a *contrecoup injury*.

Similarly, the forward acceleration of the brain relative to the skull can create tensile strain in the subdural bridging veins, causing their laceration and the formation of a hematoma.

Diffuse axonal injury (DAI) is a more complicated phenomenon. Although DAI can be explained by differences in shear strain between different parts of the brain in the setting of linear and/or rotational acceleration, there is increasing interest in how blunt (and overpressure) trauma can cause spatiotemporal pressure variations or "waves" within the brain.[19,20] Following trauma, the skull vibrates. The concavity of the cranium focuses these vibrations as multiple waves of energy to a focal point deep within the brain while sparing more superficial surface structures. Importantly, this "wave propagation" can disrupt the structural integrity of deeper structures, such as the reticular-activating system, leading to loss of consciousness.[21]

THORACIC INJURY

Musculoskeletal injury of the chest is dependent on both the magnitude and rate of the deformation of the chest wall and is usually secondary to compressive strain.[22] With blunt force to the chest, the sternum is deformed and rib cage compressed. Depending on the force and rate of impact, the ribs may fracture from compressive strain applied to their outer surface and consequent tensile strain on their inner aspects. Indirect fractures may also occur at the lateral and posterolateral angles of the rib due to the concentration of stress.

In addition to fractures, blunt chest trauma is associated with overpressure injuries. A direct load applied to the chest can result in a pneumothorax because lung compression increases the pressure within this air-filled structure beyond the failure point of the alveoli and visceral pleura. This overpressure mechanism may also be seen in the heart. As a blood-filled organ, compression causes an increase in pressure, leading to blunt cardiac rupture, which typically occurs in the thinnest portion of the heart, the right atrium.[23]

Patterns of injury for the internal organs of the thorax frequently reflect the interactions between organs that are fixed and those that are relatively mobile. This arrangement allows for differentials in momentum between adjacent structures that can lead to compressive, tensile, and shear stress. Blunt rupture of the descending thoracic aorta during deceleration provides a classic example. In frontal and lateral impacts, the heart moves in a horizontal motion relative to the aorta, which is fixed to the spinal column by ligamentous attachments. This causes a shear force applied at the level of the ligamentum arteriosum.[24] When the stress is applied in a vertical direction, such as a fall from a height, there is also a relative discrepancy in momentum, and a tensile strain is generated at the root of the thoracic aorta (Fig. 1-3).

Injury to a major bronchus is another example of deceleration shear injury. Depending on the direction of the load applied to the chest, there is a difference in the momentum between the relatively pliable and mobile lung compared to the tethered trachea and carina. This creates a shear force at the level of the mainstem bronchus (Fig. 1-4) and explains

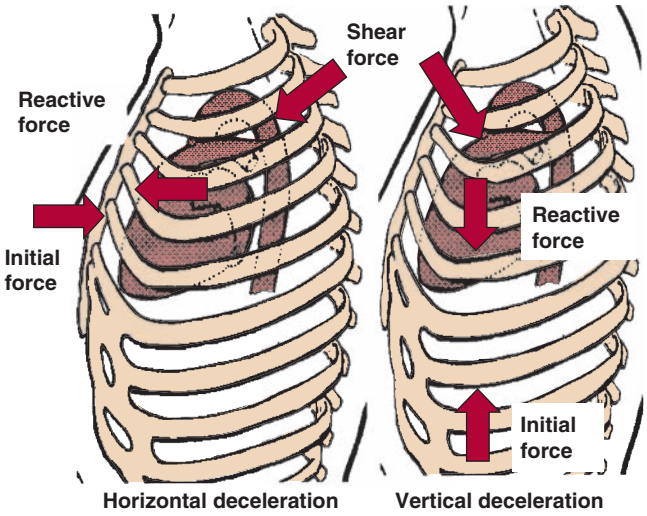

FIGURE 1-3 Various mechanisms of injury to the thoracic aorta. In a horizontal deceleration, the heart and arch move horizontally away from the descending aorta, causing shear strain and tearing at the ligamentum arteriosum. A vertical deceleration causes caudad movement of the heart, causing a strain at the root of the ascending aorta.

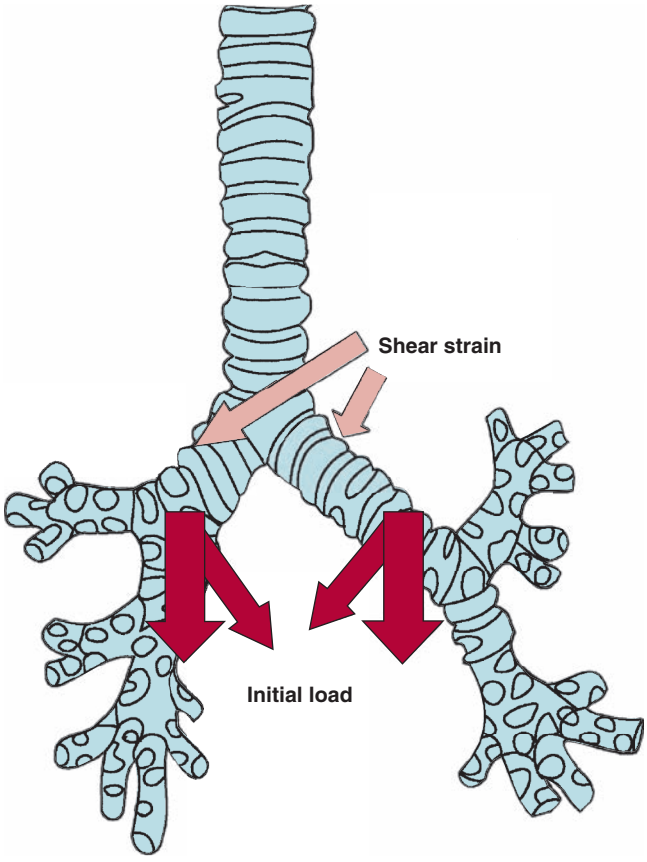

FIGURE 1-4 Mechanisms of injury for bronchial injury. The carina is tethered to the mediastinum and spinal complex, whereas the lungs are extremely mobile, setting up shear strain in the main stem bronchus upon horizontal or vertical deceleration.

why the majority of blunt bronchial injuries occur within 2 cm of the carina.[25]

ABDOMINAL INJURY

Abdominal organs are more vulnerable than those of the thorax, and a number of different mechanisms account for the spectrum of injury observed following blunt trauma. Without protection from the ribs and sternum, direct compressive force can lead to parenchymal destruction of the liver, spleen, or kidney. Shear strain at points of attachment can also lacerate these organs. As previously described, strain force requires a point of attachment in order to exacerbate a difference in momentum. This can occur at the falciform ligament, the hepatic veins, the splenic hilum, or the ligamentous attachments between the kidney and diaphragm. The body's tolerance to such biomechanical forces decreases with a higher speed of impact, resulting in an injury of greater magnitude with higher velocity collisions.[26]

Shear injury can also occur at vascular transitions, leading to intimal disruption, with thrombosis or complete transection. For example, the renal artery is attached proximally to the abdominal aorta, which is fairly immobile secondary to its attachments to the spinal column, and distally to the kidney, which is more mobile. A discrepancy in momentum between the two results in shear stain on the renal artery and a tear in the intima (or intima-media) approximately 3 cm from the aorta.

Injuries to hollow viscera are relatively rare in blunt abdominal trauma and occur in approximately 0.3% of patients.[27] Most injuries to the small bowel result from shear stress, typically occurring within 30 cm of the ligament of Treitz or ileocecal valve or at the site of adhesions. Although injuries do occur away from these points of fixation, they are infrequent and occur in the setting of "pseudo-obstruction" with overpressure.[28] The most common example of this injury pattern is blunt duodenal rupture where the closed pylorus and acute angle of the ligament of Treitz create a "pseudo-obstruction." When a compressive load is applied, the retroperitoneal duodenum is compressed. Because gas cannot adequately escape, high pressures develop and the duodenum ruptures.

Another important example of overpressure injury is rupture of the diaphragm. A large blunt abdominal force, such as the impact of a steering wheel, can cause a temporary deformation of the peritoneal cavity and a concomitant decrease in its volume. The subsequent abrupt increase in peritoneal pressure ruptures the diaphragm. Because the right hemidiaphragm is protected by the liver, the left side is the preferred route of pressure release.[29]

MUSCULOSKELETAL INJURY

By far, musculoskeletal injuries are the most common type of injury following blunt trauma, with orthopedic procedures outnumbering all other surgical interventions by nearly fivefold.[30] Although seatbelt laws, improved restraint systems, and airbags have decreased the incidence of intracranial and intra-abdominal injuries, the incidence of lower extremity trauma, in particular, has increased. It is possible, however, that this increase merely represents a survival bias. In the past, these patients may have suffered fatal injuries to the brain or torso, perhaps dying at the scene. Their associated extremity fractures, therefore, would not have been included in the overall list of injuries.[31] Alternatively, the final forces applied to the lower extremity are changed when airbags are used *without* seatbelt restraints. In simulated models of crashes with airbag deployment, muscle activation significantly increased the axial force and bending moment of the femur and tibia, and thus increased the risk of significant extremity trauma (Abbreviated Injury Scale score ≥2) by more than fivefold.[32]

Although each fracture is probably a consequence of multiple stresses and strains, there are three basic biomechanisms (Fig. 1-5).[33] *Lateral loads* provide compressive strain that leads to bowing and the subsequent development of tensile strain of the contralateral cortex. Initially, small fractures will occur in the cortex undergoing tensile strain because bone is weaker under tension than it is under compression. Once the failure point on the contralateral cortex is reached, the compressive

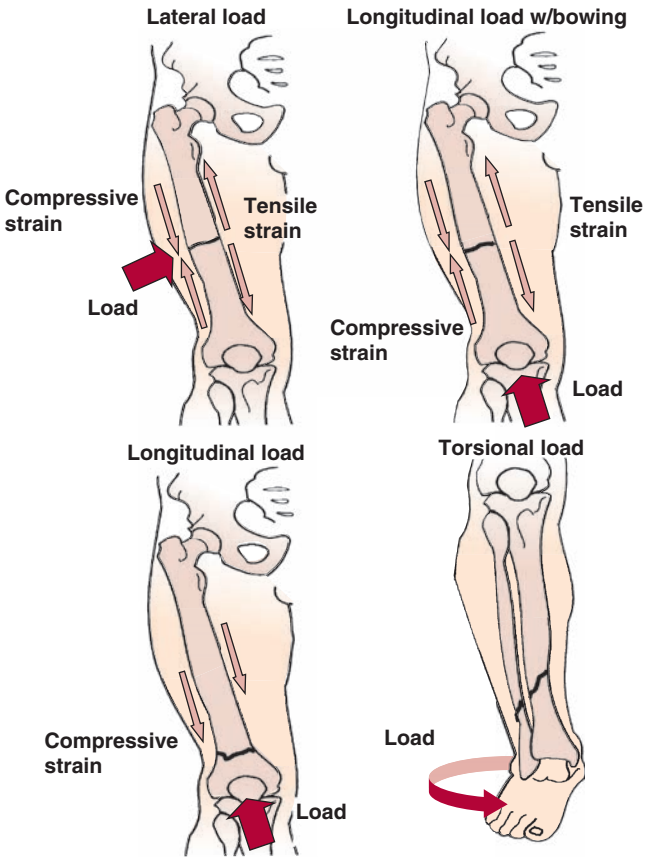

FIGURE 1-5 Fracture mechanics. A lateral load causing "bowing" will create tensile strain in the cortex opposite the force and compressive strain in the adjacent cortex. If a longitudinal stress causes "bowing," a similar strain pattern occurs. If no bowing occurs, the strain is all compressive. A torsion load will cause a spiral fracture.

strain on the ipsilateral cortex increases markedly. When its failure point is reached, a complete fracture occurs.

Similarly, *longitudinal loads* result in compressive and tensile strain patterns. If bowing does not occur, then compressive strain leads to fracture. For example, in falls or head-on collisions, the shaft of the distal femur is driven into the condyles. In contrast, the twisting force applied in a *torsional load* creates shear stress that is perpendicular to the torque axis, resulting in a spiral fracture pattern.

The type and extent of associated injuries to soft tissue are determined by the momentum and kinetic energy associated with the impact, the underlying tissue characteristics, and the angle of stress. High-energy injuries can involve extensive loss of soft tissue, neurovascular compromise, and highly comminuted fracture patterns. Low-energy injuries are often associated with a compressive strain that crushes soft tissues and results in simple fractures. Tensile and shear strain mechanisms, on the other hand, result in degloving and avulsion injuries, respectively.

INJURY TO THE SPINE

Injury to the spine can be devastating and is frequently the result of a complex combination of specific anatomic features and transmitted forces. Deceleration forces, such as those observed in motor vehicle crashes, create inertial differences between the head and torso. Injuries are related to the degree of flexion, extension, and lateral rotation; the extent of loading; and the magnitude of tension or compression forces generated during impact of the head.[34] The cervical spine is most frequently injured, due to its relatively unprotected position compared to the thoracic and lumbar regions.[35] A flexion mechanism can result in anterior atlantoaxial subluxation, an odontoid fracture, or a flexion teardrop fracture. Facet dislocation and fractures can occur when flexion is combined with rotatory forces. Similarly, traumatic spondylolisthesis of the axis, otherwise known as a hangman's fracture, results from extension and distraction of the cervical spine. Extension injuries can also lead to odontoid fractures with posterior displacement or extension teardrop fractures. Alternatively, axial compression of the cervical spine maintained in neutral position can lead to a C1 burst fracture or compression fractures of the lower vertebral bodies. Finally, distraction injuries can result in occipitocervical dissociation, a rare but frequently lethal event.[36] In contrast to trauma to the cervical spine, injuries to the thoracic or lumbar spine are more likely related to compressive mechanisms.

Whiplash refers to a musculoligamentous sprain typically seen following rear-end motor vehicle collisions where the momentum of the head creates an acceleration–extension injury, with or without rotational force. Patients typically experience neck pain and muscle spasm, although injury to cervical nerve roots or the spinal cord can occur.[37] Although whiplash can been treated with immobilization, routine activity, or active physical therapy, no treatment strategy appears to decrease long-term symptoms, with nearly 50% of victims reporting pain and disability 1 year after injury.[38]

Special Populations

PEDIATRICS

There are significant differences in patterns of injury between adults and children. Compared to adults, any given force is more widely distributed throughout the body of a child, making multiple injuries significantly more likely to occur. In addition, children also have a greater head-to-body ratio with thinner cranial bones and a less myelinated brain. Therefore, despite sustaining comparable degrees of blunt trauma, children are more likely to suffer a serious TBI and less likely to incur thoracic, spinal, and pelvic injuries.[39]

Although injury to the spinal cord is rare in children (~1%–2% of pediatric trauma), there are several anatomic features that impact the biomechanics of this injury. The pediatric spinal column is incompletely ossified, has flatter facet joints with a more horizontal vertebral orientation, and demonstrates significant ligamentous laxity. Moreover, the proportionally larger head and less developed neck musculature of younger children (10 years old) result in more torque and acceleration stress. Thus, younger children have high rates of transient upper cervical dislocation, resulting in spinal cord injury without radiographic abnormality (SCIWORA). In contrast, older children have a lower fulcrum of cervical motion (C5–C6), more ossification of the vertebral bodies, and more developed interspinous ligaments. Thus, they are more likely to have fractures in the lower cervical spine and less likely to present with SCIWORA.[40]

Another significant difference between adults and children is the compliance of the chest wall. In children, the chest wall is significantly more compressible and can absorb a greater impact while demonstrating less external signs of injury. As such, rib fractures, flail chest, hemopneumothorax, and injury to the thoracic aorta occur less frequently in children. The presence of these injuries, therefore, suggests significant blunt force. In contrast, the incidence of pulmonary contusion is higher because the compliant chest wall transmits force to the underlying compressible lung.[41]

Finally, a number of anatomic differences predispose children to more serious abdominal injuries than adults given the same degree of force. Children have proportionally larger and more anterior solid organs with less subcutaneous fat and poorly developed abdominal musculature. Their flexible cartilaginous chest wall allows for more pressure to be displaced to the abdomen and more compression of its internal organs. In addition, children also have relatively larger kidneys with fetal lobulations that predispose them to renal injury.[39]

PREGNANCY

On review of the National Trauma Data Bank of the American College of Surgeons, the majority of pregnant women are injured in the context of motor vehicle crashes (70.4%).[42] Several studies have demonstrated that the most common cause of fetal demise in motor vehicle crashes is placental abruption. The biomechanics of this injury involves the generation of tensile and shear forces within the circumferential uterine wall, which then induce a shear strain across

the placental surface. The resulting placental strain leads to a separation from the uterine wall (ie, abruption). The use of restraints and airbags decreases the force applied to the occupant, thereby increasing survival in both pregnant women and fetuses.[43]

GERIATRICS

Over 3 million Americans age 65 years or older are affected by trauma annually, and trauma is the seventh leading cause of death in this age group.[44] Falls from the standing position represent the most common mechanism of injury in the elderly, with an annual incidence of approximately 30% in those over 65 and approximately 50% in those over 80 years of age. Because aging is associated with a loss of bone density, falls frequently result in hip fractures. Unfortunately, elderly patients are also at a higher risk of a TBI given age-related cerebral atrophy and increased strain along subdural bridging veins.[45] Nearly 30,000 elderly patients die annually after falling, and a TBI accounts for the majority of these deaths.[46] Of interest, an estimated 25% of elderly patients will die within 120 days of discharge, signifying the important relationship between falls and geriatric frailty.[45]

Kinematics in Prevention of Blunt Trauma

By understanding the forces and patterns of energy transfer in motor vehicle crashes, automotive engineers have created a number of features designed to reduce injury. In order to decrease the force transmitted to the passenger compartment, Mercedes-Benz began making cars with "crumple zones" in 1959. This novel design allowed the front (and rear end) of a car to collapse upon impact. The change in momentum experienced by the passenger compartment, therefore, occurs over a longer period of time. Because more "work" is done on the vehicle, passengers experience less kinetic energy. Another feature designed to decrease the force transmitted to the passenger includes the redirection of the engine and transmission. Rather than intruding, the engine block in modern vehicles is directed downward and not into the passenger compartment.[47]

Passenger restraint systems, which include safety harnesses and child car seats, keep the passengers' velocity equal to that of the car and prevent the passengers from generating a differential in momentum and striking the interior of the car. In addition, these devices more evenly distribute loads applied to the victim across a greater surface area, thus decreasing stress.

Even with restraint systems, passengers can develop relative momentum and, therefore, kinetic energy during a crash. Airbags can dissipate this momentum and energy by converting it into "work" (ie, using the energy to compress the gas within the airbag device). Many studies have demonstrated the benefits of using seatbelts and airbags, with mortality reductions of 51% for seatbelts, 32% for airbags, and 67% for both.[48] Seatbelts and airbags have also significantly reduced the incidence of injuries to the brain, maxillofacial region, and cervical spine by keeping the forward momentum of the passenger to a minimum and preventing the head from striking the windshield.[18] Similarly, in rear-end collisions, headrests prevent a difference in momentum between the head and body and significantly decrease hyperextension of the neck. It is estimated that headrests decrease the incidence of whiplash-type injury by 70%.[49] As such, the US National Traffic Safety Administration mandated that all new vehicles be equipped with headrests in 1969.

Despite their effectiveness, restraint devices can also cause injuries. Lap seatbelts, particularly if worn incorrectly, can cause compressive injuries such as rupture of the bowel, pelvic fractures, and mesenteric avulsions. They can also act as a fulcrum for the upper portion of the trunk, leading to hyperflexion injuries such as compression fractures of the lumbar spine. As a consequence, newer automobiles are required to have three-point restraints with both lap and shoulder harness style belts. Unfortunately, shoulder harnesses can also cause intimal tears or thrombosis of the great vessels of the neck and thorax, particularly if the shoulder strap is malpositioned or the victim slides down under or out of the restraint system during lateral impact. Even when worn correctly, shoulder restraints have been associated with fractures of the clavicle, chest wall trauma, and injuries to hollow viscera secondary to compression.[50]

Approximately 10 to 20 deaths per year are thought to be directly attributable to the use of airbags. Over 90% of these are associated with the improper restraint of small adults or children or infants in front-seat locations.[51] In addition, there are a number of minor injuries that can occur as the result of airbag deployment including corneal abrasions, facial lacerations, and burns.[52]

PENETRATING TRAUMA AND BALLISTICS

Ballistics is the study of objects, or projectiles, in flight. *Internal ballistics* refers to the study of the objects themselves, whereas *external ballistics* is the study of how wind speed, drag, and gravity impact the objects. What happens when projectiles reach their target is known as *terminal ballistics.*

A projectile in motion is influenced by three forces. These include the force of the propellant (eg, a discharge or release of a bowstring), the force of gravity, and the force of resistance caused by the medium (eg, air, water, or tissue). How much damage a projectile imparts depends not only on its construction, mass, and velocity, but also on the physical properties of its target. Thus, if one knows all of these variables, projectiles have predictable trajectories and effects. This section will discuss how projectiles create injury in human tissues.

Physics of Penetrating Trauma

Both the mass of a projectile and its velocity determine its kinetic energy according to the following equation:

$$E_k = \frac{1}{2} MV^2$$

TABLE 1-1: Velocity and Kinetic Energy Characteristics of Various Guns

Caliber	Velocity (ft/s)	Muzzle energy (ft-lb)
Handguns		
0.25 in	810	73
0.32 in	745	140
0.357 in	1410	540
0.38 in	855	255
0.40 in	985	390
0.44 in	1470	1150
0.45 in	850	370
9 mm	935	345
10 mm	1340	425
Long guns/military weapons		
0.243 Winchester	3500	1725
M-16	3650	1185
7.62 NATO	2830	1535
Uzi	1500	440
AK-47	3770	1735

Given this equation, it is easy to see why a 9-mm bullet traveling at 935 ft/s has less kinetic energy than a similar round fired from an AK-47 rifle traveling at 3770 ft/s (Table 1-1). Because the energy of a system must be conserved, some (or all) of the kinetic energy of the projectile is transferred as its momentum is lost. It is the transfer of this kinetic energy that disrupts and damages tissues. How much energy is actually transferred, however, depends on the interaction between the projectile and the medium through which it travels. This interaction, also known as resistance, can be described by the formula for drag:

$$\frac{CD \times d \times v^2}{M/A}$$

where CD is the drag coefficient of the projectile, d is the density of the medium, v is the velocity of the projectile, M is the mass of the projectile, and A is the cross-sectional area. The drag coefficient, however, is difficult to measure, and the ballistic coefficient (BC) is more frequently used. The BC is directly proportional to the sectional density of the projectile, or the ratio of the surface area of its leading edge (the arrow, bullet, or fragment tip) relative to its overall mass. The higher the sectional density, the higher is the BC, and the less impedance the projectile has going through a medium. In contrast, projectiles with low sectional density will be subject to greater drag that retards their movement, thus losing their kinetic energy to the surrounding environment.

Although bullets generally travel through the air in a straight trajectory and strike their target tip first, they do not necessarily continue on a straight course in the victim. In addition, the degree to which a projectile's tip deviates from its flight path is its yaw, and this motion directly impacts its sectional density (Fig. 1-6). For example, an arrow remains stable with minimal yaw and thus has very high sectional density. As such, it passes easily through tissues. In contrast, a short bullet is much more prone to extreme yaw, flipping on its axis as it slows and "tumbling" through the medium. This motion produces an immediate decrease in the bullet's sectional density, increasing drag and thus releasing more kinetic energy into the surrounding tissue. Importantly, this transfer of energy occurs almost instantaneously. Given that force is a function of both mass and the change in speed over time (acceleration), destruction occurs because tissues are displaced extremely rapidly. The overall degree of tissue damage depends on both the velocity of the projectile and its surface density along the track.[53]

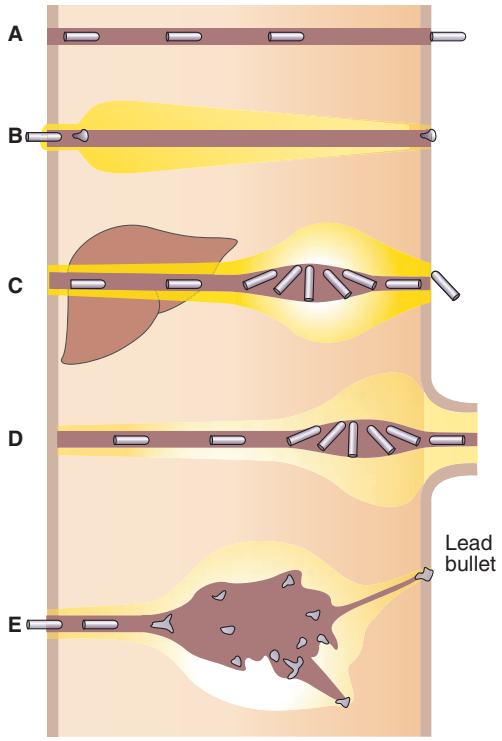

FIGURE 1-6 Wound profile of (**A**) nonfragmenting bullet through tissue with minimal yaw, (**B**) mushrooming bullet, (**C**) bullet tumbling through solid organ, (**D**) bullet tumbling with large exit skin flaps due to hydrostatic pressure wave, and (**E**) bullet fragmenting.

The shape (and thus the sectional density) of a bullet is subject to change within the tissue and directly impacts the degree of tissue damage. Projectiles that are small, hard, and fine-tipped produce minimal cavitation when compared to those that are large, soft, and broad. This important difference was noted by British soldiers in the late 1890s after their munitions were changed to hard-jacketed sharp bullets. To their disappointment, these new bullets were not as successful at stopping an advancing enemy. The bullets were subsequently modified by cutting off the tip and exposing the soft lead core. These expanding or mushrooming bullets were originally developed by the Dum Dum Arsenal in India and

became known as *dum-dums*. They were so destructive that the 1899 International Convention at the Hague banned their use in warfare.[53]

The most extreme change in sectional density occurs when the bullet breaks up into smaller pieces or fragments that then produce separate injury tracks and patterns. In their experimental model, Fackler et al[54] noted that fragmentation of a bullet caused far greater tissue injury than did a single bullet in striated muscle. The greater injury was due to the greater number of "projectiles" lacerating and crushing tissue.

As a projectile traverses the medium, the tissue is crushed and displaced, creating a permanent cavitation or "bullet track" (Fig. 1-6). The bullet also creates a hydrostatic pressure wave as it travels, stretching the adjacent tissues and creating a temporary cavity. In the 1890s, Sir William MacCormack described the destructive nature of this hydrostatic wave in a series of ballistics experiments in which a metal can filled with water was shot. When the metal pieces of the can were examined, it was obvious that the bullet exited first, creating a small hole. However, the hydrostatic pressure wave that followed created a larger and more irregular final exit hole that tore the can asunder.[53,55]

Unlike water, the human body is composed of tissues with variable density, elasticity, and compliance. Thus, the transfer of energy is far less perfect and harder to accurately predict. Moreover, each tissue has unique thresholds of stress and strain beyond which permanent destruction occurs. In general, tissues with low density (eg, fat, lung) do not impart much resistance. As such, a projectile will not fragment or yaw significantly and passes through these tissues with minimal damage. In contrast, projectiles traversing highly inelastic and dense tissues (eg, liver, kidney) create significant tissue destruction.

Blast Injuries

Blast injuries are among the most dramatic and devastating wounds treated by the trauma team. Although the vast majority of these injuries occur in the context of war on foreign lands, the United States is not immune, with 699 explosions reported by the Bureau of Alcohol, Tobacco, and Firearms in 2016.[56]

Blast injuries are broadly categorized as primary, secondary, tertiary, quaternary, and quinary, based on a taxonomy developed by the Department of Defense.[57] The trauma surgeon should be familiar with and able to predict associated injuries from each category (Table 1-2).

The damage imparted by the initial shock wave after an explosion is called the *primary blast*. Injuries are caused by overpressurization and typically affect gas-filled structures such as the lungs, intestines, and middle ear. The force of a primary blast is determined by the type and size of the explosive charge, the distance from the charge to the target, the medium through which the wave passes, and the composition of the target.

The strength of the charge is typically benchmarked against TNT (trinitrotoluene), with a detonation velocity of approximately 6900 m/s. Some modern plastic explosives such as Semtex and C-4 have detonation velocities in excess of 8000 m/s. The medium through which the primary blast wave travels will also determine how much energy reaches the target. Air is extremely compressible and will absorb much of the energy of the initial blast. Water, being relatively incompressible, transmits much more of the energy.

The characteristics of the target are important for several reasons. First, when the shock wave travels through a medium less dense than the target, such as air versus a human body,

TABLE 1-2: Department of Defense Classification of Blast Injuries from Explosive Devices

Classification	Definition	Common injuries
Primary	Blast overpressure injury (blast wave) Direct tissue damage from the shock wave Air-filled organs at highest risk (ears, lungs, gastrointestinal tract)	Tympanic membrane rupture Blast lung Gastrointestinal tract perforation/hemorrhage Ocular Concussion
Secondary	Primary fragments—from the exploding device (either from pieces of the device itself or from projectiles placed intentionally into the device to increase the lethality of the device) Secondary fragments—from the environment (glass, small rocks, etc)	Lacerations Penetrating injury Significant soft tissue injury (including traumatic amputations) Ocular
Tertiary	Acceleration/deceleration of the body onto nearby objects or displacement of large nearby objects onto an individual	Blunt trauma Traumatic amputation Crush injury
Quaternary	Injuries due to other "explosive products" effects—heat, toxidromes from fuel and metals, and so on	Burns Inhalation injury
Quinary	Clinical consequences from postdetonation environmental contaminants including bacteria, radiation, and tissue reaction to fuels and metals	Radiation Sepsis

much of the wave is reflected and not absorbed by the victim. In contrast, when the medium and the target have similar densities, such as water and the human body, the energy of the sound wave is almost entirely transmitted to the victim. Underwater explosions, therefore, are severalfold more dangerous than those occurring on land. Although blast injuries are less common in open terrain, the blast wave can reflect off the walls in contained spaces, increasing the incidence of injury to more than 90%.[58] Whether on land or under water, when the blast wave goes from one medium to a less dense medium, rarefaction waves cause shear stress within the tissues and cavitation.

Although the most common injury associated with primary blast is ruptured tympanic membranes, the most fatal injury is "blast lung." With this injury, the blast wave causes tissue disruption at the capillary–alveolar interface, resulting in pulmonary edema, pneumothorax, parenchymal hemorrhage, and, occasionally, air embolus from alveolovenous fistulas.[58] Clinical diagnosis of blast lung injury is dependent on the presence of hypoxia, respiratory distress, and the pathognomonic central "butterfly" infiltrates on a chest x-ray. The infiltrates are usually present on admission and can worsen with aggressive fluid resuscitation. Management is supportive and includes positive-pressure ventilation if needed, minimization of positive end-expiratory pressure (PEEP), and judicious fluid resuscitation. Fluid management in these patients can be challenging due to associated injuries from secondary and tertiary blast effects.

Secondary blast injuries are created by debris from the explosive device itself or from the surrounding environment. Many improvised explosive devices contain additional munitions such as nails, pellets, ball bearings, and scrap metal and are designed to increase the lethality of the explosion. Secondary blast injuries are more common than primary because the debris and fragments travel further than the primary shock wave. Lacerations, penetrating injury, and significant soft tissue defects are the most common injuries seen from secondary blast.[57]

Tertiary blast injuries are caused by the body being physically thrown a distance or from a solid object falling onto a person as a result of the explosion. Most tertiary injuries are the result of blunt trauma. Finally, *quaternary* and *quinary blast* injuries have only recently been defined. They are miscellaneous blast injuries caused directly by the explosion but often due to other mechanisms, such as burns, inhalation injuries, and radiation effects.[57]

Contact Shots

Gunshot wounds where the muzzle is in contact with the skin are termed *contact shots* and have a very different wounding profile than projectiles. In addition to discharging a projectile, the muzzle releases expanding gases that are contained by the skin and subcutaneous tissue.[59] Given that the blast velocity of gunpowder is upward of 20,000 ft/s, this contained explosion is far more destructive than the temporary cavity of a passing projectile (<4000 ft/s). As such, the tissue injury beneath the skin surface may be significantly greater than expected. In 1984, American television actor Jon-Erik Hexum killed himself on set when he fired a revolver loaded with blanks at his temple. Despite the absence of an actual projectile, the muzzle blast was contained by the skin and focused into his temple with sufficient force to cause a devastating brain injury that killed him instantly.

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2

Epidemiology

Ashley D. Meagher • Ben L. Zarzaur

KEY POINTS

- Injuries are the leading cause of death for Americans age 1 to 44 years and the third leading cause of death overall.
- Disease of injury has a bimodal age distribution of mortality, with peaks for those 16 to 40 years old and for those over 65 years old.
- Injury deaths have risen in the United States over the past 2 decades, from 52 per 100,000 in 2000 to 72 per 100,000 in 2016.
- The International Classification of Diseases, Tenth Revision, Clinical Modification external cause of injury code is known as an E-code and is used when a patient presents to a health care provider with an injury.
- Deaths from drug overdose now account for 27.4% of injury-related deaths in the United States, more than from motor vehicle collisions.
- Approximately 50% of all traffic fatalities (driver, occupant, bicyclist, or pedestrian) have been found to have a blood alcohol concentration of 0.08 g/dL or greater.
- Firearm-related mortality is eight times higher in the United States than in other high-income countries in the world.

INTRODUCTION

Injury is not an "accident" but rather a disease, much like infectious diseases such as malaria or tuberculosis or health diagnoses such as cancer, diabetes mellitus, or heart disease. Injury, like other diseases, has variants such as blunt or penetrating. It has degrees of severity, rates of incidence, prevalence, and mortality that can differ by race and other sociodemographic factors. In addition, injuries have a predictable pattern of occurrence related to age, sex, alcohol, illicit or prescription drugs, and, again, sociodemographic factors, among others. Finally, they have a predictable prognosis, based on age, sociodemographic factors, and injury severity.

This characterization of injury as a disease is an important one and a matter of more than just semantics. When public health concepts are applied to injury, the first step after recognition is to characterize the disease such that control strategies can be applied. Epidemiology is the study of patterns of disease occurring in human populations and the factors that influence these patterns.[1]

Descriptive epidemiology refers to the distribution of disease over time and place as well as within or across specific subgroups of the population. It is important for understanding the impact of injury in a population and identifying opportunities for intervention. The burden of injury can be described as the most common, most fatal, most debilitating, or most costly within a specific population.

In contrast, analytic epidemiology refers to the more detailed study of the determinants of observed distributions of disease in terms of causal factors. The epidemiologic framework traditionally identifies these factors as related to the host (characteristics intrinsic to the person), the agent (physical, chemical, nutritive, or infectious), and the physical or sociocultural environment (characteristics extrinsic to the person that influence exposure or susceptibility to the agent). The importance of this epidemiologic approach is the direction it gives to injury prevention efforts as well as to areas requiring further research.[2]

Injuries can result from acute exposure to physical agents such as mechanical energy, heat, electricity, chemicals, and ionizing radiation in amounts or rates above or below the threshold of human tolerance. The transfer of mechanical energy accounts for more than three-fourths of all injuries.[3] The extent and severity of injury are largely determined by the amount of energy outside the threshold of human tolerance. Both the exposure to energy and the consequences of

TABLE 2-1: Analysis of a Motor Vehicle Collision Using Haddon's Matrix

	Human (or host)	Vector	Physical environment	Socioeconomic environment
Pre-event	Substance misuse, poor driving habits	Faulty brakes, bald tires	Slippery road due to rain	Social acceptance of high levels of alcohol use by males
Event	Not wearing a seatbelt	No airbag	Tree too close to the road	Ineffective enforcement of DUI offenses
Postevent	Elderly man, preexisting medical condition		Slow emergency response, poor rehabilitation program	Little help for reintegrating rehab patients into society

DUI, driving under the influence (of alcohol).

that exposure are greatly influenced by a variety of factors both within and beyond individual or societal control.[4]

The public health approach applied to injury control seeks to modulate factors related to the host and agent and/or their interactions within the environment using a number of strategies. These strategies encompass engineering, education, the enactment and enforcement of laws, and economic incentives and disincentives.

The public health approach as it applies to injury was first conceptualized by the late William Haddon in the late 1960s.[2] He developed and promulgated a phase-factor matrix that incorporated the classic epidemiologic framework of host, agent, and environment in a time sequence that encompasses three phases: pre-event, event, and postevent. This allows for simultaneous consideration of the factors and the stages of an event. Factors (eg, host, agent, or environment) in the pre-event phase determine whether the event will occur. Factors in the event phase determine whether an injury will occur as a result of the event and the degree of injury severity. Factors in the postevent phase influence the outcome from, or consequences of, any injuries of any severity that do occur. An example of the Haddon Matrix applied to a motor vehicle collision is depicted in Table 2-1.[5]

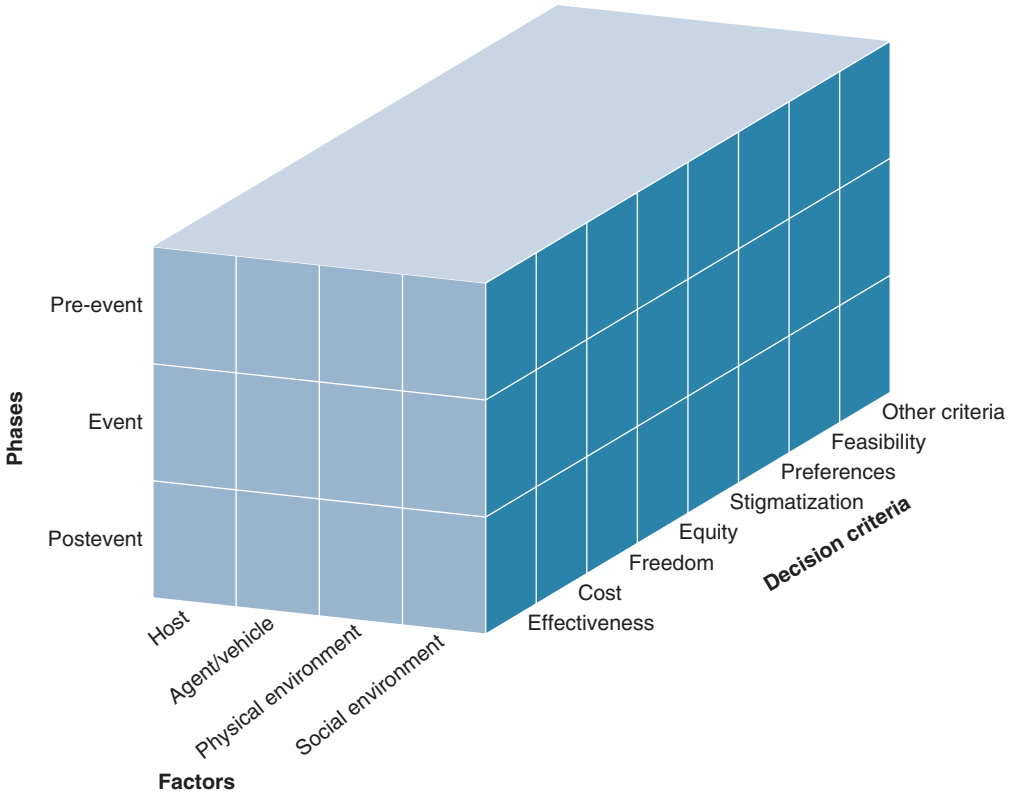
Although the Haddon Matrix is the foundation of injury epidemiology, it is not enough to direct robust injury prevention and control efforts. The addition of potential control strategies to the matrix in a three-dimensional fashion results in an "injury control cube." This suggests that injury prevention and control are not unidimensional or unifactorial and that the greater the number of sections of the "cube" that are addressed, the greater the control of the injury event. Runyan[6] has proposed using this third dimension of the matrix to incorporate and analyze the factors and value relative to each policy option (Fig. 2-1).

OVERVIEW OF INJURY EPIDEMIOLOGY IN THE UNITED STATES

Injuries are the leading cause of death for Americans age 1 to 44 years and the third leading cause of death overall. In 2016, 231,991 persons died in the United States as a result of an injury, up from approximately 190,000 in 2013 and resulting in an age-adjusted injury rate per 100,000 population of 68.78 (Fig. 2-2).[3] The predominance of injury deaths among the young results in another measure of the burden of injury—years of productive life lost. This measure makes the assumption that individuals are most productive to society before the age of 65; however, given the ever-increasing length of productive life, this is frequently an incorrect assumption. Nevertheless, it does give some measure of comparison of the effect of various causes of mortality. In 2016, all causes of death contributed to nearly 12 million years of productive life lost (YPLL). Deaths from unintentional injury were responsible for 23% of YPLL, more than any other individual category. The next two categories, malignant neoplasm and heart disease, accounted for 14% and 11% of YPLL, respectively.[7] As the American population ages, there is an increase in injury deaths for those over the age of 65. In 2016, 62,904 persons older than age 65 years died (127.7 per 100,000), up from 43,153 in 2006 (116.1 per 100,000).

There has been an intensive effort over the past 3 decades to decrease the rate of injury-related death in the United States. Some of these interventions have included the development of trauma systems, increased safety mechanisms in motor vehicles, and educational programs aimed at at-risk populations. The effect of these interventions can be seen in the mortality trends over time. These trends can also reveal areas in need of further intervention. For example, deaths from motor vehicle collisions have trended from 27% of injury-related deaths in 2001 to 16.7% in 2016 (Fig. 2-3).[8] Alarmingly, deaths from drug overdose have increased from 12.3% of injury-related deaths in 2001 to 27.4% in 2016, more than motor vehicle collisions.

The timing of deaths due to injury, classically described as trimodal, has changed due to advances in resuscitation and intensive care that have essentially eliminated the last peak of deaths from multisystem organ failure.[9-12] The majority of all deaths still occur within minutes of the injury, either at the scene prior to arrival of the emergency medical services (EMS), en route to the hospital, or in the first hours of care. A recent analysis found that 26% of all in-hospital deaths occurred within 1 hour of hospital arrival and 59% occurred within 24 hours.[12] These immediate deaths are typically the result of massive hemorrhage or severe neurologic injury.



Steps to Complete the Three-Dimensional Haddon Matrix	
Step	Activity
1	Use community needs assessment data to determine the problem in need of intervention.
2	Define dimension #1 (columns) of matrix as the targets of change.
3	Define dimension #2 (rows) of matrix by delineating the precise event and phases of change.
4	Define dimension #3 (depth) of matrix by delineating value criteria, defining each in clear terms.
5	Determine weights to be applied to each value listed in dimension #3.
6	Brainstorm potential interventions and fill in cells formed by columns and rows.
7	Use data to assess each criterion for each intervention under consideration.
8	Assess each intervention according to its attributes relative to each value criterion.
9	Conduct an overall assessment using weights for each value criterion across the set of interventions and criteria.
10	Make decisions about best options.
11	Explain decisions based on criteria applied and assessment of each intervention option according to the criteria.
12	Document the assessment process to assist future reanalyses.

FIGURE 2-1 Three-dimensional Haddon matrix as described by Runyan,[6] and steps to complete the matrix.

The second peak of the bimodal death distribution occurs within several hours to days of the event, with 78% of deaths by postinjury day 3. These deaths are primarily due to injury to the central nervous system (CNS) (Fig. 2-4).

Deaths represent only one small aspect of the injury disease burden. Each year, over 32 million people suffer a nonfatal injury. The vast majority of these patients are seen in emergency departments (EDs) or urgent care centers without requiring hospital admission; however, over 2.6 million people require hospitalization. Many of these nonfatal injuries have far-reaching consequences with the potential for reduced quality of life and high costs for the health care system, employers, and society. In 2013, the estimated total lifetime costs associated with both fatal and nonfatal injuries amounted to over $671 billion, including $457 billion for nonfatal injuries and $214 billion for fatal injuries (Figs. 2-5 and 2-6).[13-15]

The costs associated with injury deaths account for a disproportionate share of total injury costs. Estimates show that deaths account for less than 1% of all injuries, but are responsible for 31% of total injury costs. The remaining 69% of costs due to injury are associated with nonfatal injuries. These costs include direct expenditures for health care (30%) and other goods and services purchased as a result of the injury. The value of lost productivity due to temporary and permanent disabilities is also taken into account and represents 41% of the total costs. These are merely the financial costs

	Age Groups										
Rank	**<1**	**1–4**	**5–9**	**10–14**	**15–24**	**25–34**	**35–44**	**45–54**	**55–64**	**65+**	**Total**
1	Congenital Anomalies 4,816	Unintentional Injury 1,261	Unintentional Injury 787	Unintentional Injury 847	Unintentional Injury 13,895	Unintentional Injury 23,984	Unintentional Injury 20,975	Malignant Neoplasms 41,291	Malignant Neoplasms 116,364	Heart Disease 507,118	Heart Disease 635,260
2	Short Gestation 3,927	Congenital Anomalies 433	Malignant Neoplasms 449	Suicide 436	Suicide 5,723	Suicide 7,366	Malignant Neoplasms 10,903	Heart Disease 34,027	Heart Disease 78,610	Malignant Neoplasms 422,927	Malignant Neoplasms 598,038
3	SIDS 1,500	Malignant Neoplasms 377	Congenital Anomalies 203	Malignant Neoplasms 431	Homicide 5,172	Homicide 5,376	Heart Disease 10,477	Unintentional Injury 23,377	Unintentional Injury 21,860	Chronic Low. Respiratory Disease 131,002	Unintentional Injury 161,374
4	Maternal Pregnancy Comp. 1,402	Homicide 339	Homicide 139	Homicide 147	Malignant Neoplasms 1,431	Malignant Neoplasms 3,791	Suicide 7,030	Suicide 8,437	Chronic Low. Respiratory Disease 17,810	Cerebro-vascular 121,630	Chronic Low. Respiratory Disease 154,596
5	Unintentional Injury 1,219	Heart Disease 118	Heart Disease 77	Congenital Anomalies 146	Heart Disease 949	Heart Disease 3,445	Homicide 3,369	Liver Disease 8,364	Diabetes Mellitus 14,251	Alzheimer's Disease 114,883	Cerebro-vascular 142,142
6	Placenta Cord. Membranes 841	Influenza & Pneumonia 103	Chronic Low. Respiratory Disease 68	Heart Disease 111	Congenital Anomalies 388	Liver Disease 925	Liver Disease 2,851	Diabetes Mellitus 6,267	Liver Disease 13,448	Diabetes Mellitus 56,452	Alzheimer's Disease 116,103
7	Bacterial Sepsis 583	Septicemia 70	Influenza & Pneumonia 48	Chronic Low. Respiratory Disease 75	Diabetes Mellitus 211	Diabetes Mellitus 792	Diabetes Mellitus 2,049	Cerebro-vascular 5,353	Cerebro-vascular 12,310	Unintentional Injury 53,141	Diabetes Mellitus 80,058
8	Respiratory Distress 488	Perinatal Period 60	Septicemia 40	Cerebro-vascular 50	Chronic Low. Respiratory Disease 206	Cerebro-vascular 575	Cerebro-vascular 1,851	Chronic Low. Respiratory Disease 4,307	Suicide 7,759	Influenza & Pneumonia 42,479	Influenza & Pneumonia 51,537
9	Circulatory System Disease 460	Cerebro-vascular 55	Cerebro-vascular 38	Influenza & Pneumonia 39	Influenza & Pneumonia 189	HIV 546	HIV 971	Septicemia 2,472	Septicemia 5,941	Nephritis 41,095	Nephritis 50,046
10	Neonatal Hemorrhage 398	Chronic Low. Respiratory Disease 51	Benign Neoplasms 31	Septicemia 31	Complicated Pregnancy 184	Complicated Pregnancy 472	Septicemia 897	Homicide 2,152	Nephritis 5,650	Septicemia 30,405	Suicide 44,965

Data Source: National Vital Statistics System, National Center for Health Statistics, CDC.
Produced by: National Center for Injury Prevention and Control, CDC using WISQARS™.

Centers for Disease Control and Prevention
National Center for Injury Prevention and Control

FIGURE 2-2 Leading causes of death in the United States, 2016. (Data obtained from Centers for Disease Control and Prevention Web-Based Injury Statistics Query and Reporting System data, https://www.cdc.gov/injury/wisqars/index.html. Reproduced from the Centers for Disease Control and Prevention, National Center for Injury Prevention and Control.)

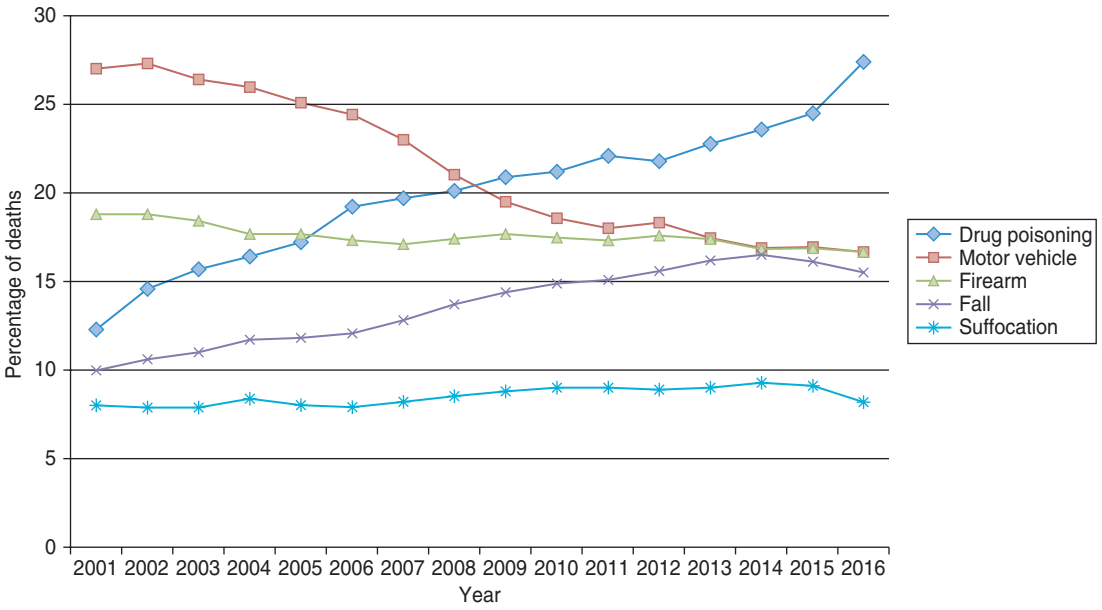


FIGURE 2-3 Injury-related causes of death in the United States from 2001 to 2016, by percentage. (Data obtained from Centers for Disease Control and Prevention Web-Based Injury Statistics Query and Reporting System data, https://www.cdc.gov/injury/wisqars/index.html.)

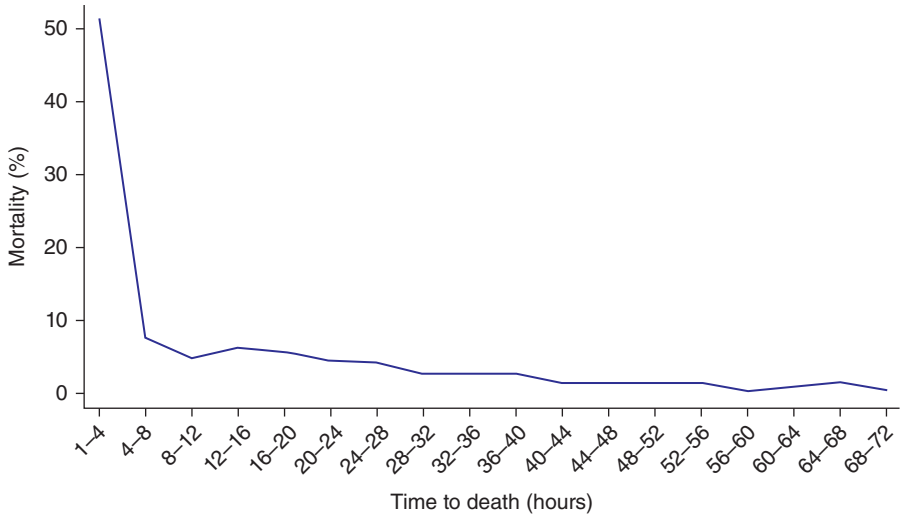


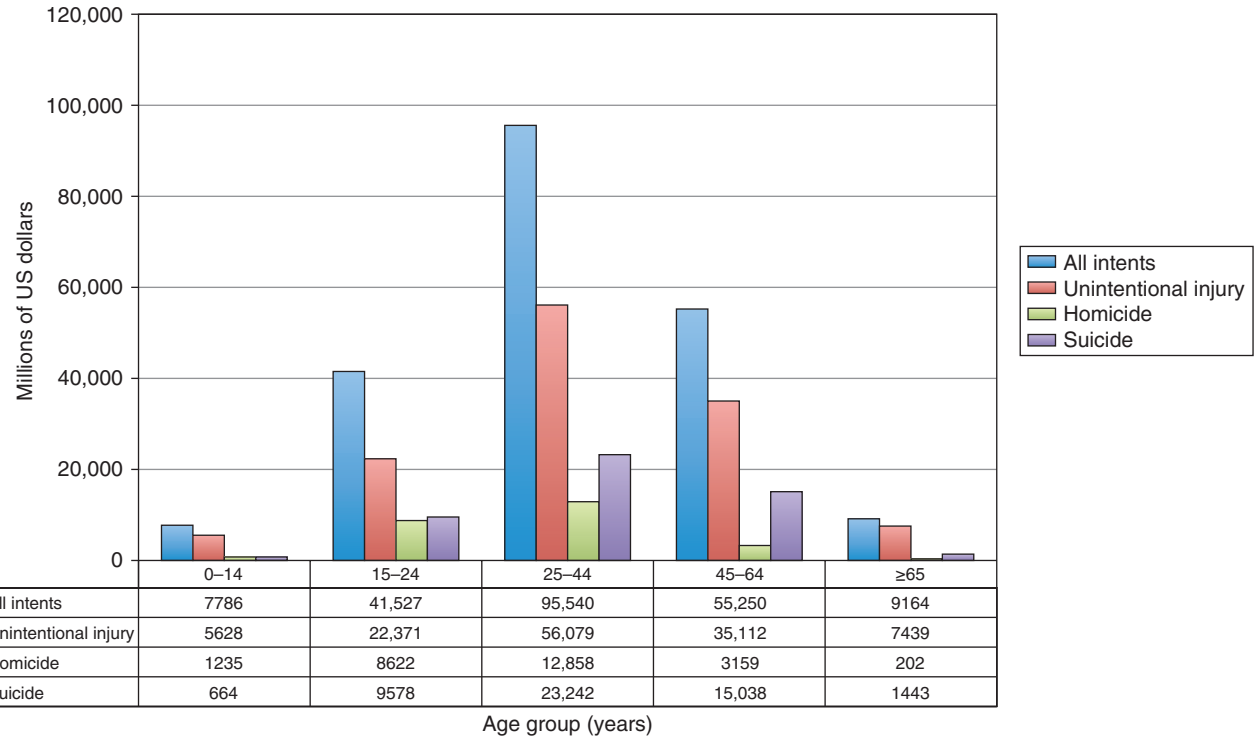
FIGURE 2-4 Temporal distribution of trauma mortality, in-hospital deaths.

and do not reflect the pain and suffering of patients, their families, and associates after nonfatal injuries.[14-18]

AGE-RELATED INJURY EPIDEMIOLOGY

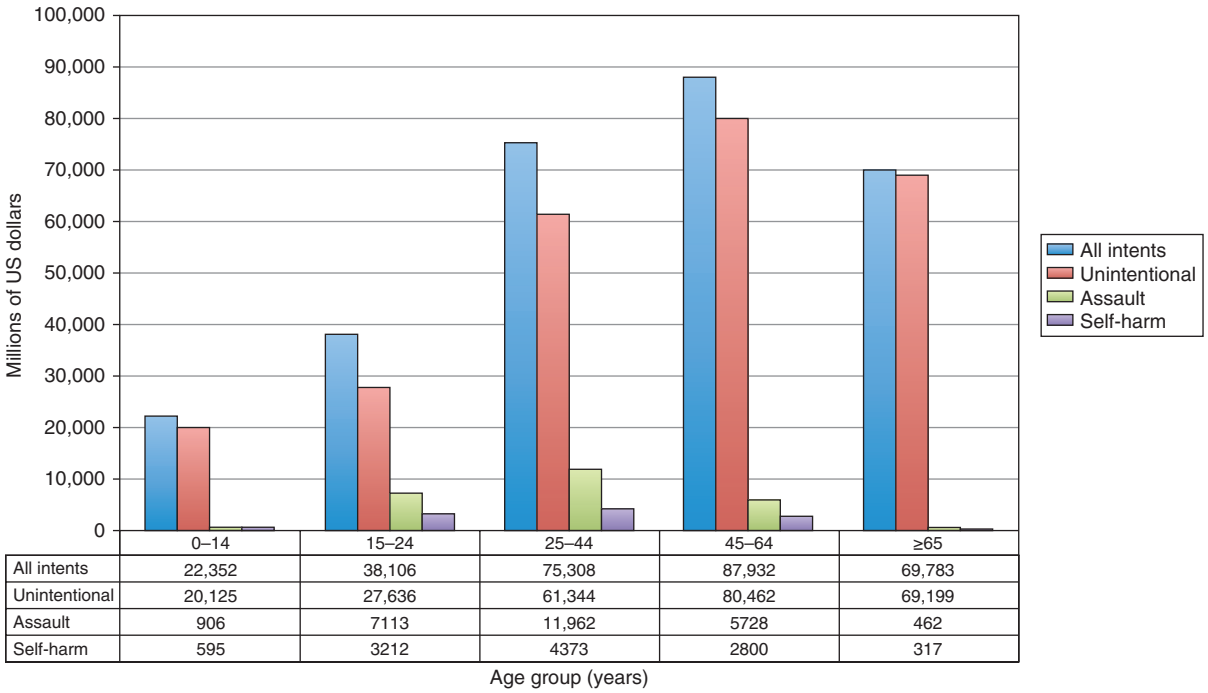
Injury is a disease predominantly affecting young males, including 70% of deaths and more than 50% of nonfatal injuries. For nonfatal injuries, males are only 1.3 times as likely as females to be affected. This gender-related risk reverses after the age of 65, with females 1.3 times as likely as males to suffer nonfatal injury. Therefore, injuries account for more premature deaths than cancer, heart disease, or HIV infection.[8,18]

The disease of injury has a bimodal age distribution of mortality that peaks for both genders in the 16- to 40-year-old age group and then again in those older than 65 years of age (Fig. 2-7). Persons under the age of 50 account for 51%



	0–14	15–24	25–44	45–64	≥65
All intents	7786	41,527	95,540	55,250	9164
Unintentional injury	5628	22,371	56,079	35,112	7439
Homicide	1235	8622	12,858	3159	202
Suicide	664	9578	23,242	15,038	1443

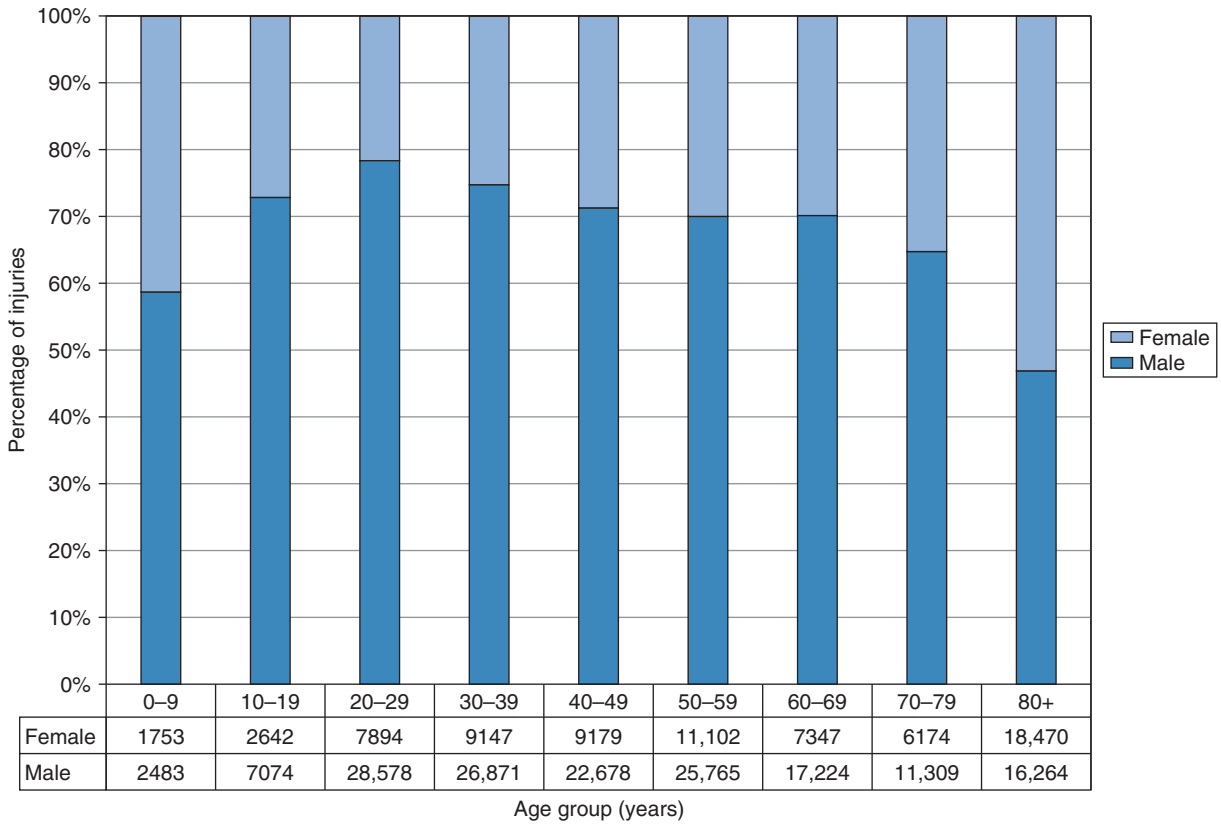
FIGURE 2-5 Estimated lifetime medical and work-loss costs, fatal injuries, 2013. (Data obtained from Centers for Disease Control and Prevention Web-Based Injury Statistics Query and Reporting System data, https://www.cdc.gov/injury/wisqars/index.html.)



	0–14	15–24	25–44	45–64	≥65
All intents	22,352	38,106	75,308	87,932	69,783
Unintentional	20,125	27,636	61,344	80,462	69,199
Assault	906	7113	11,962	5728	462
Self-harm	595	3212	4373	2800	317

Age group (years)

FIGURE 2-6 Estimated lifetime medical and work-loss costs, nonfatal injuries, 2013. (Data obtained from Centers for Disease Control and Prevention Web-Based Injury Statistics Query and Reporting System data, https://www.cdc.gov/injury/wisqars/index.html.)



	0–9	10–19	20–29	30–39	40–49	50–59	60–69	70–79	80+
Female	1753	2642	7894	9147	9179	11,102	7347	6174	18,470
Male	2483	7074	28,578	26,871	22,678	25,765	17,224	11,309	16,264

Age group (years)

FIGURE 2-7 Fatal injuries by sex and age group, 2016. (Data obtained from Centers for Disease Control and Prevention Web-Based Injury Statistics Query and Reporting System data, https://www.cdc.gov/injury/wisqars/index.html.)

of all injury fatalities, just over 50% of hospitalizations, and nearly 80% of ED visits.[3,9] Hospitalizations and ED visits follow this pattern of a bimodal peak related to age and a male predominance, as well.

The number of elderly (age ≥65 years) individuals in the United States is rapidly growing and is projected to reach 90 million adults by 2050.[19] Although patients over age 65 represent less than 25% of trauma admissions, they are more likely to die from injuries with a lesser severity than their younger counterparts.[19,20] In addition, the costs associated with geriatric trauma patients are higher than those of their younger counterparts, as they frequently have longer hospitalizations and need posthospital rehabilitation.[20-23] Representing 15% of the US population, those over the age of 65 account for approximately 27% of all injury deaths and 36% of all injury-related hospitalizations, and it is expected that the elderly will account for approximately 40% of all injury deaths and hospitalizations in the near future.[8,18] Current research is focusing on identification of risk factors for poor outcome as well as projecting posthospitalization needs for these elderly trauma patients.[21,24,25] Discussion continues on the need to alter triage criteria, admission protocols, and admission location as the physiology of aging results in different responses to injury.[26,27]

EPIDEMIOLOGY BY MECHANISM AND INTENT

Injuries are typically categorized by their mechanism, intent, and place of occurrence. Mechanism refers to the external agent or particular activities that were associated with the injury (eg, motor vehicle related, falls, firearm related). Intent of the injury is classified as either unintentional or intentional.

Injuries that are intentionally inflicted can be further subcategorized into interpersonal (eg, homicide) or intrapersonal (eg, suicide); however, intent may not always be determinable. Injuries resulting from legal interventions and operations of war are typically classified separately as an "other intent" category.

The classification system most often used in describing the specific mechanism and intent of injury is the International Classification of Diseases (ICD). This classification system was developed and promulgated by the World Health Organization and is now in its 10th edition.[28] The E-code, or external cause of injury code, provides detailed information about the circumstances associated with injury-related ED visits and hospitalizations. These codes are essential to the epidemiology of injury and its accurate study. They provide critical public health information for monitoring health status, setting injury prevention priorities, and developing and evaluating injury prevention programs at the local, state, and national levels.

E-codes can also be useful for quality initiatives associated with injury-related claims (eg, motor vehicle crash–related injuries) that may assist the Centers for Medicare and Medicaid Services (CMS) in making payment decisions. Hence, numerous professional organizations including the American College of Surgeons Committee on Trauma, the American College of Emergency Physicians, the Emergency Nurses Association, and the National Safety Council have published position statements to endorse the need for improving E-coding in state mortality and morbidity data systems. In addition, they have urged that at least three fields be captured as part of the E-code. Two of the E-code fields can be used for coding the precipitating and immediate causes (eg, the mechanism/intent of injury such as falls, motor vehicle traffic, fire/burn, cut/pierce, assault, self-harm), and one other field is used to delineate the place of occurrence (eg, home, street/highway, residential, institution). Unfortunately, E-codes are not mandated by all payors, all states, or CMS as a part of ICD-10.

The distribution of injuries by mechanism varies for deaths, hospitalizations, and ED visits. The leading mechanisms of fatal injury are drug overdose, motor vehicle collisions, and firearms. Although nearly 30% of all injury deaths are violence related, 93% of nonfatal injuries and 69% of fatal injuries are unintentional.[3]

The Bureau of Labor Statistics within the US Department of Labor tracks injury in the workplace. A total of 5190 fatal work injuries were recorded in the United States in 2016, representing 3.6 fatal work injuries per 100,000 full-time equivalent workers, a concerning uptrend over the past 5 years. Overall, transportation-related incidents accounted for the majority (40%) of occupational injury deaths. Assaults and violent acts accounted for 16% of fatalities, contacts with objects and equipment 14%, and falls 16%. Homicides accounted for 9% of occupational-related deaths in 2016.[29]

In addition to the fatalities associated with work-related activities, there were a total of 2.9 million nonfatal injuries recorded by the Bureau of Labor Statistics (2.9 cases per 100 workers). Of interest, 71% occurred in service-providing industries, and nearly half produced disability.[30]

LEADING MECHANISMS OF INJURY

Drug Overdose

The United States is in the midst of an opioid epidemic, with over 72,000 drug poisoning deaths in 2017, up 6.6% from 2016.[31] Drug overdoses have more than tripled since 1999 and, as previously noted, exceeded motor vehicle–related injury as the leading cause of injury-related death in 2011.[8] In 2016, there were 21.3 drug overdose deaths per 100,000 population, up from 12.3 per 100,000 in 2010.[32] As the number of drug overdose deaths has increased, so have the number of nonfatal drug overdoses. In 2010, there were 355 per 100,000 nonfatal overdoses, whereas there were 613 per 100,000 in 2016.[18]

The opioid epidemic is multifactorial, driven by a rise in the availability of prescription opioids as well as increased availability of illicit opiates such as heroin and fentanyl. Currently, the sharpest increase in opiate-related deaths involves heroin and synthetic opiates other than methadone. In 2017, 35 states (including Washington, DC) saw an increase in

drug overdose deaths, 7 states were stable, and 9 states saw a decrease.[31]

Similar to other mechanisms of injury, males are two-thirds more likely to die from a drug overdose as compared to females. Overdoses remain a disease primarily affecting adults and are the leading cause of injury-related death for adults age 25 to 64 years. Of interest, approximately 5% of drug overdose deaths are in adults over the age of 65, and 8% of drug overdose deaths are in young adults age 15 to 24.[8]

It is important to note that the impact of drug-associated injuries is not included in these data. Prior to the onset of the opioid epidemic, 33% of motor vehicle crashes and 8% of fall-related fractures were associated with illicit drugs.[33,34] As opiate use has increased, it is probable that injuries and deaths secondary to drug abuse disorders are increasing. It is important that health care providers recognize the potential for drug abuse disorders in injured patients and offer intervention.

In 2017, the opioid crisis was declared a national public health emergency, with the aim of releasing federal funds, developing harm reduction strategies, and tightening regulations on opiate prescribing practices among physicians.[35] Harm reduction strategies range from safe syringe programs, provision of naloxone to known heroin abusers and first responders, and improving access to treatment for opiate disorders. Increased attention on physician prescribing has led to restrictions on licensed prescribers, increased prescription drug monitoring programs, and more guidelines for the treatment of pain.[32,35] This epidemic will only abate with a multifaceted approach, integrating public health experts, physicians, and public and private partnerships.

Motor Vehicle–Related Injury

Traffic-related incidents involving motor vehicles were the second leading cause of injury-related death in 2016 and rank second as a cause of nonfatal injury in the United States. It is the leading cause of injury-related death in the 5- to 24-year age group and the second leading cause of injury-related death in the 25- to 84-year age group. Each year, more than 4 million people are injured in traffic-related crashes, resulting in over 40,000 deaths.[8]

Adolescents and young adults are at the highest risk for both fatal and nonfatal injuries due to motor vehicle collisions. Their rates of death, hospitalization, and ED visits are approximately twice the rate for all ages combined. White males age 15 to 24 are at particular risk. For black males in that same age group, traffic-related injury ranks second as a cause of death, behind firearm-related injuries. Individuals age 75 and older are also at relatively high risk for dying from motor vehicle accident–related injury.

Males are more than twice as likely as females to die from motor vehicle crashes. Males under the age of 45 are also more likely to be hospitalized as a result of motor vehicle–related injuries, although the gender differential is not as great as for fatalities. Males and females age 45 and older, in contrast, are equally likely to be hospitalized.

Determinants of injury occurrence and severity in a motor vehicle–related incident include speed of impact, vehicle crashworthiness, and the use of safety devices and restraints including safety belts, airbags, and helmets. This is an area where epidemiologic analyses have truly had an impact in reducing the burden of injury. When used, restraint devices have been shown to reduce fatalities to front-seat occupants by 45% to 60% and the risk of moderate-to-critical injury by 50%. It is estimated that over 2000 deaths could be prevented with appropriate use of restraint devices. Currently, usage rates for restraint devices in the United States range from 69% in South Dakota to 98% in Oregon, with a national average of 87% in 2013.[36] This is an increase of 1% over 2012 and 6% over 2006. Mandatory restraint device laws are present in 49 states. In 34 states, these are primary enforcement laws, meaning police may stop vehicles solely for restraint law violations.[37] The presence of an airbag in front of a belted driver provides increased protection, resulting in an estimated 51% reduction in fatality rate.

Despite some success in reducing the role of alcohol in motor vehicle collisions, it remains a major factor in fatal crashes among adolescents and young adults. Approximately 50% of all traffic fatalities including the driver, occupant, bicyclist, or pedestrian have been found to have a blood alcohol concentration (BAC) of 0.08 g/dL or greater. The proportion of fatally injured drivers with elevated BAC varies with age. For all age groups, it has remained relatively constant, at approximately 26% for women and 43% for men, since 1999.[38] In contrast, the proportion of drivers under the influence of other drugs, including narcotics, depressants, and cannabinoids, has increased significantly.[39]

Firearms

In 2016, there were approximately 39,000 intentional (98%) and unintentional (2%) gun-related deaths in the United States. This represents 107 firearm-related deaths per day, or 12 deaths per 100,000 population. The majority of firearm-related deaths among males age 15 to 34 in the United States (67%) are homicides.[3] Suicide deaths and firearm deaths have been increasing over the past 5 years. In addition, nonfatal firearm injuries have increased as well. In 2016, there were 116,414 reported nonfatal injuries caused by firearms, or 36 per 100,000 population. This represents an increase from 23 per 100,000 in 2010.[18]

Firearm-related injuries disproportionately affect males (85%) and younger people. In the 15- to 34-year age group, firearm-related death rates for males are nearly seven times that for females. Firearm-related injuries are the leading cause of death in black males age 15 to 34 and are the third leading cause of injury-related death in those age 10 to 84. From a global and cultural standpoint, firearm-related mortality is eight times higher in the United States than other high-income countries in the world.[3,8]

There has been a significant increase in the suicide rate in the United States, with 45,000 deaths and 50% of these being firearm related. Suicide in those age 65 or greater is

also a significant problem, with 5756 firearm-related suicides in 2016, representing 25% of all firearm-related injuries for both genders and all ages. Over 90% of the suicides in the elderly population were among males.[8]

Both fatal and nonfatal firearm injuries accounted for just over $39 billion in 2010 in direct medical costs and productivity losses.[16] This is related to the fact that firearms of some type are present in about 30% of all US households, including 46% of rural and 19% of urban residents.[40] Studies suggest that those who live in homes that have a firearm are more likely to die from homicide and suicide in the home than are residents of homes without firearms.[41,42]

Data on fatal and nonfatal firearm-related injuries are not as complete as data from motor vehicle collisions. This is secondary to the political and emotional environment that surrounds the discussion about gun violence. Since 1997, with the addition of the Dickey Amendment to the Omnibus spending bill, federally funded research on gun violence has been curtailed.[43] This has led to inadequate data collection and hindered public health professionals from developing and implementing prevention strategies. In 2015, eight medical professional groups, including the American College of Surgeons, released an organized call for repeal of the Dickey Amendment and increased federal funding into research on gun violence and prevention.[44]

Falls

There were 34,673 fatal falls in 2016; falls represent the third most common cause of injury-related deaths and 15% of all injury deaths. In addition, there were 9.2 million nonfatal injuries following falls. Falls account for over one-third of all injury hospitalizations, resulting in over $95 billion in lifetime costs.[8,13,18]

Unlike the other common mechanisms of injury, falls primarily occur in younger and older age groups; however, the severity profile in the two groups is quite different. Falls are the leading cause of injuries for all children age 0 to 14, although these are rarely fatal or cause severe injuries. Approximately 8000 children are treated daily in United States for fall-related injuries, totaling almost 2.8 million children each year; however, less than 3% of these need hospitalization.[45] Approximately half of all pediatric falls occur in the home and one-quarter occur at school. Falls in children age 0 to 4 years are most commonly from furniture or stairs. In older children, falls are commonly from standing and/or associated with recreational activities related to playground equipment, bicycling, or sports.

In adults of working age, most fatal falls are from buildings, ladders, and scaffolds. Falls on stairs increase in significance starting at age 45.[3] ED visit rates for falls have an interesting trend, with males predominating in the 0- to 19-year age groups, whereas the incidences in the sexes are nearly even from ages 20 to 44. Females then predominate above age 44, accounting for nearly 70% of nonfatal injuries in the age group over 85.[18] This finding is consistent with the increased fracture risk in women after menopause and, specifically, those with osteoporosis.

In the elderly, falls are a significant cause of mortality and morbidity and are the leading cause of injury-related deaths for those 65 and over. Falls account for 22% of injury-related deaths in those age 65 to 74 years and 65% in those age 85 years or older. The death rate from falls after age 85 is over three times that for people age 75 to 84 years.[8] Falls are also the most common cause of nonfatal injury in the elderly, accounting for nearly 60% of injury-related ED visits and approximately 80% of injury-related hospitalizations for persons age 65 years and older.[18] In the United States, 1 in 5 people over the age of 65 will sustain a fall annually. Of these, about one-quarter will be injured and another quarter will restrict their daily activities for fear of another fall.[46] The economic impact of falls in the elderly is sizable and was more than $50 billion in 2015, with an average hospitalization costing $30,000.[13,46]

Major risk factors for falls among the elderly include the following: those related to the host (eg, advanced age, history of previous falls, hypotension, psychoactive medications, dementia, difficulties with postural stability and gait, visual disturbances, cognitive and neurologic deficits, or other physical impairment) or environmental factors (eg, loose rugs and loose objects on the floor, ice and slippery surfaces, uneven flooring, poor lighting, unstable furniture, absent handrails on staircases). The risk of falling increases linearly with the number of risk factors present, and it has been suggested that falls and some other geriatric syndromes may share a set of predisposing factors. All of these factors are potentially modifiable with combinations of environmental, rehabilitative, psychological, medical, and/or surgical interventions.[47]

EPIDEMIOLOGY BY NATURE AND SEVERITY

Cataloging and analyzing the distribution of injuries by their nature and severity is important to efforts at establishing priorities for prevention as well as treatment, trauma system development, and research. The International Collaborative Effort on Injury Statistics was formed through the Centers for Disease Control and Prevention and National Center for Health Statistics to develop a uniform framework for using ICD codes to categorize injury diagnosis by the body region involved and the specific nature of the injury.[48] This collaboration aims to develop injury statistics, such as the Abbreviated Injury Scale (AIS) and the Injury Severity Score (ISS), that are internationally comparable to allow information exchange among researchers.[49] Several other systems for classifying the nature and severity of injury exist, and a number of these are described elsewhere in this textbook.

The American College of Surgeons has led the effort to aggregate trauma registry data in order to provide insight into the nature, severity, and types of injuries occurring across the United States.[50] Trauma centers are mandated to submit their registry data into the standardized National Trauma Data Bank (NTDB), allowing researchers to query a large database of quality injury-related data. Since the adoption of the

National Trauma Data Standard and with increasing adoption of electronic medical records, this data set has increased in quality and utility.[51] In the past, some of what has been known about the overall nature of trauma deaths was based on a limited number of studies conducted in selected geographic regions using coroners' reports and autopsy records; unfortunately, these types of records, much like the death certificates, are variable in completeness, accuracy, and utility.[52]

Frequently, injury patterns are mediated by the mechanism of injury. This allows for monitoring of injury types in relation to mechanisms. For example, recognition of the rate of traumatic brain injury in sports-related injuries has led to increased efforts at diagnosis and prevention.[53] Certain types of injuries carry a higher likelihood of death and are targets for aggressive treatments and prevention, such as hemorrhage. The American College of Surgeons Committee on Trauma, in collaboration with The Hartford Consensus, has launched a nationwide campaign to teach hemorrhage control to lay people, in order to save lives prior to arrival of first responders.[54] This campaign arose after analysis of several mass casualty events in which lay people were the only available initial "medical" providers. "Stop the Bleed" is an example of an initiative aimed at empowering public stakeholders with training and education.

INJURY BY GEOGRAPHIC LOCATION

The overall incidence and patterns of injury vary between urban and rural populations across the country (Fig. 2-8).[3] Unintentional injury and motor vehicle death rates are highest in rural areas, whereas homicide and firearm death rates are several times higher in urban areas. Evaluation of injury by rural versus urban is a difficult undertaking. There are numerous ways to divide a locale in order to designate rural, suburban, or urban.

Injury death rates also vary by region of the country. For example, death rates for unintentional injury tend to be highest in the west and south, whereas suicide rates are highest in the west and homicide rates highest in the south. There is, however, substantial state-by-state and even county-by-county variation. It is important to know that the observed differences related to geographic location and population

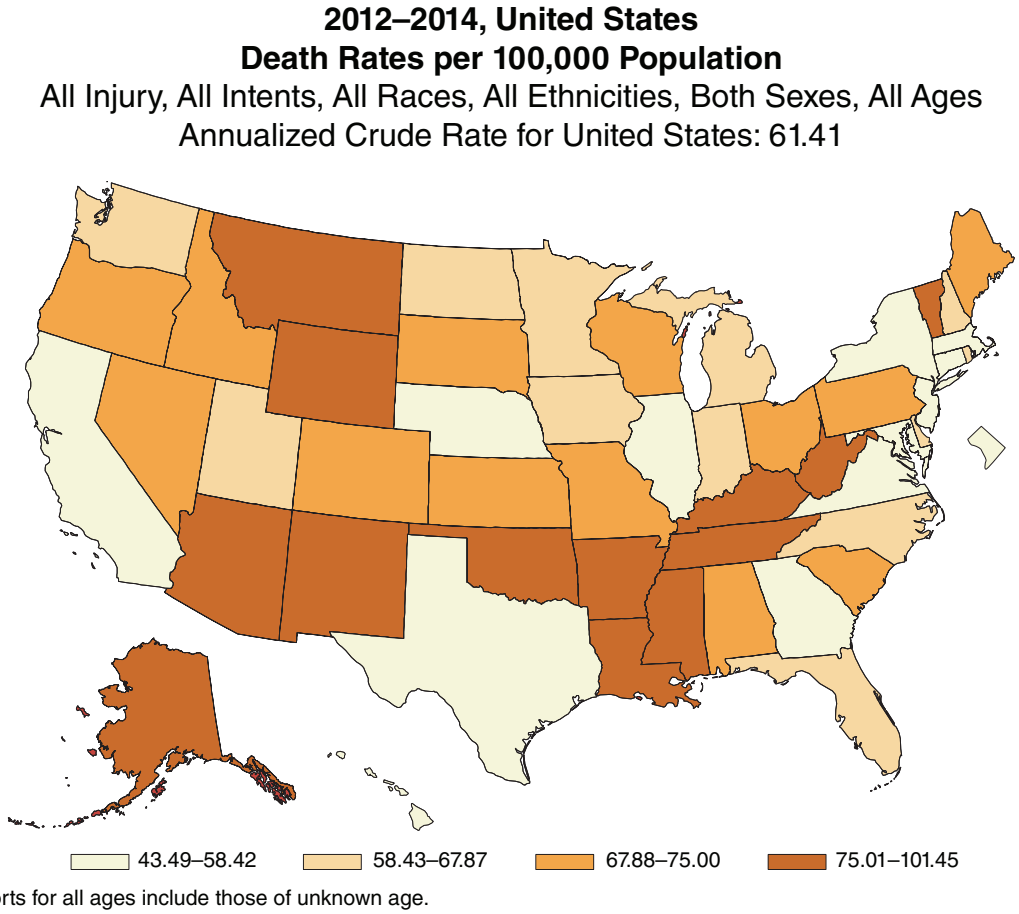


FIGURE 2-8 Death rate per 100,000 population, by state. (Data obtained from Centers for Disease Control and Prevention Web-Based Injury Statistics Query and Reporting System data, https://www.cdc.gov/injury/wisqars/index.html. Reproduced from the Statistics, Programming, and Economics Branch, National Center for Injury Prevention and Control, Centers for Disease Control and Prevention.)

density may be a function of a number of confounding factors such as access to care, economic disadvantages, education level, or even geographic barriers.[55,56]

CONFOUNDERS

There are a number of confounding factors that may influence results and, more critically, the interpretation and conclusions drawn from epidemiologic analyses of injury. These include race, ethnicity, culture, socioeconomic status, access to health care, mental health, and alcohol and other drugs. Due to their number and multiplicity, adequate control for these confounding factors is difficult and requires forethought and caution prior to embarking on epidemiologic research. Similarly, publications need to be critically appraised to avoid erroneous conclusions.

Data that are stratified by race and/or Hispanic origin must be interpreted carefully. In data often used to populate registries and in-hospital discharge data, the patient is often assigned a race by administrative staff. The patient does not chose which race he or she identifies with most. The color of the skin or patient language may be more of a determining factor than the patient's own preference when race and ethnicity are chosen by administrative means. This can lead to confounding through misclassification.[57,58] Additionally, the population that is used as the denominator in the calculation of the death rate generally comes from US Census Bureau estimates, whereas the characteristics of those who died (numerator) come from data collected by funeral directors or medical examiners. This results in inconsistent results due to the inherent errors in each set of data. Bias in estimates by race and ethnicity also can result from undercounting of specific populations in the census, thereby potentially producing an overestimation of death rates.

Differences in health status by race and Hispanic origin also are known to exist and may be explained by factors including socioeconomic status, health practices, psychosocial stress and resources, environmental exposures, discrimination, and access to health care.[59] As these factors are not routinely collected or controlled for, analysis of injury mortality and morbidity by race and ethnicity may lead to incorrect inferences. With specific regard to data on violence, estimates may be misinterpreted because attention may have been directed to the victim rather than to the perpetrator, for whom sufficient data are not routinely collected.

Although this chapter emphasizes the concept of injury being a disease entity in and of itself, data suggest that, for a significant number of trauma patients, injuries may be an unrecognized symptom of underlying alcohol or drug abuse. Therefore, it may be that injury is actually a comorbidity of a disease, which is alcohol or substance abuse. Nearly 50% of injury deaths are alcohol related, and injuries account for roughly the same number of alcohol-related deaths as cirrhosis, hepatitis, pancreatitis, and all other medical conditions associated with excessive alcohol use combined. A multicenter study that included data on more than 4000 patients admitted to 6 trauma centers demonstrated that 40% had some level of alcohol in their blood upon admission and up to 60% of patients tested positive for one or more intoxicants.[38,60] Therefore, it is clear that alcohol and substance abuse must be considered in the epidemiology of injury as well as in the equation leading to effective injury control. Recognition of this fact, along with evidence that intervention to address alcohol use disorder in the acute setting was beneficial, helped drive the recommendation from the American College of Surgeons Committee on Trauma to provide screening and brief intervention for alcohol use disorder at Level I and Level II trauma centers.[61]

DATA SOURCES

With the increasing use of electronic medical records (EMRs), there has been a significant increase in administrative data on health information. Administrative data refers to the fact that these data are originally collected for administrative purposes, rather than specific research questions. As such, there are frequently numerous variables available, such as demographic details, health diagnoses, complications, outcomes, and procedures. These databases have the potential for evaluating a patient's course through the hospital or health care system, as derived from the EMR. Most nationally available data sets are deidentified to protect patient and hospital privacy and typically require a fee for access, as well as a research proposal and clearance by an institutional review board (Table 2-2).

Selection of the appropriate administrative data set for research requires consideration of the population and outcome of interest. Administrative databases typically aggregate data from hospital billing and coding in the EMR. They are generally less useful when attempting to analyze detailed clinical issues, such as specific complications or severity of illness, despite available methods to estimate injury severity using ICD codes.[69,70] Use of hospital-level data may limit the generalizability of a study, whereas use of a national data set may decrease the granularity of available data. An additional confounder in the reliability and accuracy of administrative databases is the extent, prioritization, and accuracy of ICD coding. The fidelity of the data is only as good as the coding that is entered. There are frequently examples of "upcoding"—making a patient seem sicker than he or she is. In addition, if codes are omitted, then the patient may appear less sick. Also, it should be pointed out that these data sets only represent the population of hospitalized patients. They do not capture all deaths and will not ever include patients with minor injuries not seeking treatment at hospitals.

A number of the publicly available data sources have been used in the production of this chapter and are referenced. Table 2-2 gives a broad description of the most commonly used national data sets, as well as access information. National data can be used for drawing attention to the magnitude of the injury problem, for monitoring the impact of federal legislation, and for examining variations in injury rates by region of the country and by rural versus urban/suburban environments. They can also be useful in aggregating sufficient numbers of cases of a particular type of injury to analyze causal

TABLE 2-2: Administrative Databases Available for Injury Research

Database Name	Administration	Data Sources	Details	Website
Web-Based Injury Statistics Query and Reporting System (WISQARS)[3,8,13,18,62]	Centers for Disease Control and Prevention (CDC), National Center for Injury Prevention	National Vital Statistics System, National Center for Health Statistics, National Electronic Injury Surveillance System, National Violent Death Reporting System	Publicly available, interactive, state level. Filters for subpopulations.	https://www.cdc.gov/injury/wisqars/index.html
Healthcare Cost and Utilization Project (HCUP)[63]	Agency for Healthcare Quality and Research	Administrative data from state, hospital, and federal resources	National resource of encounter-level health care data. Numerous databases available through application and purchase.	https://www.hcup-us.ahrq.gov/
National Health Interview Survey (NHIS)[64]	CDC, National Center for Health Statistics	Cross-sectional household interview survey administered annually	Sample of approximately 35,000 households. Provides information on nonfatal injuries that do not result in hospital admission.	https://www.cdc.gov/nchs/nhis/index.htm
National Ambulatory Medical Care Survey (NAMCS)[65]	CDC, National Center for Health Statistics	Data abstracted from physician offices, emergency departments, and ambulatory surgery centers	A nationwide sample of physician, hospital outpatient, and emergency department services; the conditions most often treated; and the diagnostic and therapeutic services rendered, including medications prescribed.	https://www.cdc.gov/nchs/ahcd/index.htm
National Trauma Data Bank (NTDB)[50]	American College of Surgeons	Deidentified incident-level data submitted to national registry by institutions	Clinical registry of incidents of traumatic injury and hospital course. Data are available through application and purchase.	https://www.facs.org/quality-programs/trauma/tqp/center-programs/ntdb
Trauma Quality Improvement Program (TQIP)[66]	American College of Surgeons	Institutional-level quality improvement initiative	Provides institutions with risk-adjusted benchmarking that provides national comparisons across similar level trauma centers. Data access requires submission to TQIP as an institution.	https://www.facs.org/quality-programs/trauma/tqp/center-programs/tqip
National Emergency Medical Services Information System (NEMSIS)[67]	Federal funding with state-level cooperation; administered through University of Utah	Standardized emergency medical services (EMS) data registry from every state	30 million EMS activations in 49 states. Data available through application and purchase.	https://nemsis.org/
Uniform Data System for Medical Rehabilitation (UDSMR)[68]	Not-for-profit organization affiliated with the University at Buffalo and The State University of New York	Data submission by rehabilitation centers across the United States	Patient functionality throughout rehabilitation course. Data available through application and purchase.	https://www.udsmr.org/

patterns and clinical or other outcomes on an individual or systems basis. Often, however, these national data sources are not appropriate for developing and sustaining injury prevention programs at the state and local level.

State and local data are more likely to reflect specific injury problems and are, therefore, more useful in setting priorities and evaluating the impact of policies and programs. Additionally, local data may be more useful when advocating for local policy change or funding of injury control programs. Some of the national databases described in Table 2-2 do provide subsets of data at the state or even county level; however, many do not. Availability, accuracy, and completeness of local injury data vary substantially by state and county. Vital statistics and death certificate data are generally available for all injury-related deaths; however, these data are limited in the information they provide about the nature and circumstances of the injury, cause of death, and risk factors associated with the death. Medical examiner and coroner reports can be a useful adjunct to death certificate data, but, once again, the completeness and quality of these data vary substantially from state to state. Autopsy rates are equally variable and are generally biased toward being performed in cases of suspected homicide.

State and local data on trauma hospitalizations are generally available from two principal sources, either uniform hospital discharge data or trauma registries. Trauma registries typically include more detailed information regarding the cause, nature, and severity of the injury than standard hospital EMR data. Trauma registries suffer from selection bias and inconsistent inclusion criteria, as well as variable data integrity. In both types of databases, ICD coding is not uniform. In addition, trauma registries do not include patients not admitted to nontrauma hospitals. Hospital discharge databases and trauma registries do not include information on trauma deaths that occur at the scene or in transport, nor do they routinely include patients who are treated and released. It is important to reemphasize that caution should be exercised in using these databases for describing the epidemiology of trauma, as neither is population based. Uniform data on trauma patients treated and released from EDs, hospital clinics, and physicians' offices are generally even less accessible than hospital-level EMR data.

The utility of existing data at the state and local level can be significantly enhanced by linking data across multiple data sources. Single data sources are often limited in their content or scope of coverage, or both. Techniques have been developed and are continually being improved to facilitate linkage of these databases to avoid the high costs of gathering new data. Several states have now linked hospital discharge data, vital statistics, police crash reports, and prehospital run sheet data.[71,72]

Local, state, and national data are extremely expensive to collect, and there is more expense to ensure they are reliable and accurate. As a result, funding for registry or database initiatives is often hard to come by, and those resources supported by federal dollars are ever at risk for budget cuts. Despite the limitations listed earlier, administrative data are incredibly important because they form the cornerstone for research, advocacy, and prevention efforts in trauma patients.

CONCLUSION

In summary, injury imposes a heavy burden on society in terms of both mortality and morbidity, along with its sizable economic burden on the health care system. Largely unrecognized is the fact that many fatal and nonfatal injuries are preventable using specific strategies guided by the analysis of injury epidemiology. Hence, there is no societal level of intolerance and fear of incidence as there is for HIV or West Nile virus and H1N1 influenza. Yet, these diseases contribute much less to the burden of public health disease than do injuries.

Risks of injury death vary by age and gender. The majority of injury deaths are unintentional, with elderly people at a particularly high risk. Considering intentional injuries overall, suicide greatly exceeds homicides, but rates again vary by age, gender, and urban or rural residence. Mechanisms of injury death also vary by age. The risk of injury death on the job varies by occupation. From a global perspective, the United States compares less than favorably with other countries in terms of fatal injury, particularly those related to firearms (Figs. 2-4 and 2-5).

Injury deaths have risen in the United States over the past 2 decades from 52 per 100,000 in 2000 to 72 per 100,000 in 2016. There is significant variation by mechanism of injury, with a gradual decline in motor vehicle–related deaths, but sharp increases in drug overdoses and fall-related deaths. Injury morbidity rates have demonstrated declining trends among all age groups except the elderly. Alcohol and other drugs continue to be intimately associated with all types and mechanisms of injury.

In conclusion, although significant strides have been made in reducing the rate at which injury occurs, trauma remains a major public health issue. More efficient ways of treating injuries as they occur, or tertiary prevention, should and will continue to be the major thrust of clinical care providers and researchers. It is equally and perhaps more important, however, that efforts to develop appropriate programs and policies that prevent injuries be prioritized. Education of policymakers and the public that this public health epidemic can and must be controlled is an essential component of this effort. Accurate, easily obtainable, and understandable data are a key first step in this process.

Integrated efforts at primary, secondary, and tertiary prevention, along with public information and education programs, are the only means to effect injury control and reduce the burden of injury on individuals, the health care system, and society at large. Understanding the importance of high-quality data as a building block for the study of injury science is important for all who participate in the care of the injured. Studying the epidemiology of injuries provides the opportunity for understanding how, when, and with whom to intervene.

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3

Injury Prevention

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KEY POINTS

- Injury is responsible for more deaths worldwide than HIV, tuberculosis, and malaria combined.
- Injury remains the leading cause of death in the United States in people age 1 to 46 years old.
- The financial cost of injury is tremendous. Road traffic injuries alone cost most countries 1% to 2% of their gross national product.
- The social determinants of health, including poor employment opportunities, food deserts, poor education, and poverty, contribute significantly to risk of sustaining injury; addressing these factors represents a targeted strategy to reduce injury and recidivism.
- Evaluation of injury prevention programs begins at inception with feasibility and incorporates quantitative and qualitative measures.
- Broad public health principles can be applied to prevention of intentional and unintentional injuries.
- Prevention programs focus on risk reduction and thoughtful evaluation.

INTRODUCTION

Injury is a leading cause of death, disability, and health care costs worldwide. The Global Burden of Disease Study, which creates a unique framework to assess national trends in all-cause and cause-specific mortality and morbidity, has shed light on the burden of injury relative to the denominator of all morbidity and mortality.[1] This research and other prominent publications have been instrumental in moving injury to a level of recognition commensurate with its level of disease burden. Injury has begun to gain recognition as a prominent public health issue as thought leaders, researchers, and clinicians are vigorously studying the issues within a framework by which prevention efforts, trauma systems, and advocacy strategies can be developed and maintained.

Approximately 5.8 million people die around the world annually from injury-related causes. As a consequence of inadequate surveillance in many parts of the world, that number is likely to be much higher. Injury is responsible for more deaths worldwide than HIV, tuberculosis, and malaria combined. The impact is projected to increase over time relative to other leading causes of death (Table 3-1).[2] Greater than 90% of injury deaths occur in low- and middle-income countries, and within individual countries, vulnerable populations tend to be of lower socioeconomic status. This further hampers the progress of already struggling communities.

In several ways, the burden and demographics of injury in the United States provide an example of the patterns seen worldwide: injury is most prevalent in communities of lower socioeconomic status, rates of injury are higher in men than women, and young people are disproportionately affected by injury. According to the Centers for Disease Control and Prevention (CDC), in 2016, unintentional injury remained the leading cause of death in the United States for persons age 1 to 44 years. Unintentional injury, suicide, and homicide are the first, second, and third leading causes of death in those age 15 to 34 years, respectively. Homicide remains the leading cause of death in the United States in African Americans age 15 to 34 years old and the second cause in Hispanics of the same age. Unintentional injury is the third leading cause of death of all Americans, with motor vehicle crashes and falls, particularly in the elderly, having a significant impact.[3] As the population ages in the United States, the contribution of falls to the burden of unintentional injury will continue to rise, with an estimated 1 in 4 adults over the age of 65 expected to sustain a fall each year, resulting in an estimated $31 billion in annual Medicare costs. Furthermore, it is believed that approximately 50% of people over 65 years of age who sustain a fall will fall again within a year.[4]

Death is the "tip of the iceberg" in understanding the impact of injury. In order to understand the true magnitude

TABLE 3-1: Injury Deaths Rise in Rank, Leading Causes of Death, 2012 and 2030 Compared

Total 2012	Total 2030
1 Ischaemic heart disease	1 Ischaemic heart disease
2 Stroke	2 Stroke
3 Chronic obstructive pulmonay disease	3 Chronic obstructive pulmonay disease
4 Lower respiratory infections	4 Lower respiratory infections
5 Trachea, bronchus, lung cancers	5 Diabetes mellitus
6 HIV/AIDS	6 Trachea, bronchus, lung cancers
7 Diarrhoeal diseases	7 Road traffic injuries
8 Diabetes mellitus	8 HIV/AIDS
9 Road traffic injuries	9 Diarrhoeal diseases
10 Hypertensive heart diseases	10 Hypertensive heart diseases
11 Preterm birth complications	11 Cirrhosis of the liver
12 Cirrhosis of the liver	12 Liver cancer
13 Tuberculosis	13 Kidney diseases
14 Kidney diseases	14 Stomach cancer
15 Suicide	15 Colon and rectum cancer
16 Birth asphyxia and birth trauma	16 Suicide
17 Liver cancer	17 Falls
18 Stomach cancer	18 Alzheimer's disease and other dementias
19 Colon and rectum cancers	19 Preterm birth complications
20 Alzheimer's disease and other dementias	20 Breast cancer
21 Falls	21 Endocrine, blood, immune disorders

Source: Reproduced with permission from World Health Organization. *Injuries and Violence: The Facts*. Geneva, Switzerland: World Health Organization; 2014.

of injury, it is important to look at disability sustained, along with the age at which a person is killed or injured. Calculations of disability-adjusted life-years (DALYs), or the sum of years of life lost due to premature death and the years of productive life lost due to disability, are performed using a standard disability weight for each particular type of injury such as amputation, paralysis, posttraumatic stress disorder, and so on.[5] Although the DALY calculation has been criticized in its global applicability, it highlights the impact of injury on those who are either very early in their lives or in the middle of their years of economic productivity.

To round out the picture of the impact of injury, cost should be taken into consideration. Direct health care costs can be calculated and modeled. Indirect costs, including the psychological impact, loss of productivity affecting entire families, and societal costs of injury, are more difficult to calculate, but there is a growing understanding of their magnitude. Road traffic injuries alone cost most countries 3% of their gross national product.[6,7] A study in Ghana found that 25% of households affected by injury had a decline in their food consumption.[8] The release of the Lancet Commission on Global Surgery included a discussion on catastrophic expenditures from surgical disease, defined as out-of-pocket payment for treatment services exceeding 10% of annual household income.[9] This is a critical issue in surgical care, and injury is a leading part of that. In the United States, lifetime medical and work loss costs associated with fatal and nonfatal injuries were $671 billion in 2013.[10]

A chapter focused on injury prevention cannot properly be discussed without first understanding injury's influence on health and wealth. Understanding the global, national, and local impact of injury by mechanism and demographics is a critical start to launching into a strategy of targeted injury prevention appropriate for a particular location and population. A detailed investigation into these components along with understanding risk factors and protective factors allows us to understand the target population for prevention efforts. It can also be the cornerstone for creating a strategic plan for appropriate measures in education, engineering, and enforcement. In addition, understanding the public health impact of injury allows targeted advocacy for legislation and financial resources necessary to initiate many prevention plans. Understanding the direct and indirect costs of injury creates opportunities to conduct studies in cost-effectiveness, a compelling tool when advocating for prevention measures, particularly in an environment of limited resources.

THE IMPORTANCE OF PREVENTION IN TRAUMA SYSTEMS

The importance of injury prevention efforts is pointed out by trauma mortality patterns. Approximately one-third to one-half of trauma deaths still occur in the field, before any access to even the most advanced trauma treatment system.[11,12] Such deaths can only be decreased by prevention efforts. In terms of severely injured persons who survive long enough

to be treated by prehospital personnel, very few "preventable deaths" occur in a modern trauma system with well-run emergency medical services and designated trauma centers. Even among patients who survive to reach the hospital, a significant portion of in-hospital deaths are directly related to injuries to the brain and occur despite optimal available therapy. In one study of 753 consecutive deaths, over 50% were deemed possibly preventable only by prevention efforts, with only 13% due to pulmonary embolus, multiorgan failure, or sepsis.[13] Hence, injury prevention is critical to further significantly reduce the toll of death caused by trauma. Moreover, prevention efforts can also decrease the severity of injuries and the likelihood of disability that arises after trauma.

The American College of Surgeons (ACS) recognizes the critical role prevention plays in reducing the societal burden of injuries. The Committee on Trauma (COT) has mandated that all Level I trauma centers and recommended that Levels II through IV centers include a prevention program whose priorities reflect the dominant injury epidemiology in their community.[14] Additionally, Level I centers are mandated to have a prevention professional, who holds a separate position from the trauma program manager. This individual coordinates injury prevention efforts, including partnerships with community stakeholders and local governmental leaders. Level I trauma centers are also required to demonstrate that the trauma medical director has an active role in injury prevention, identification of priorities, and developing interventions to address root causes of injury. Currently, there is a working group within the ACS COT that is revising prevention criteria based on updated research and best practices review.

This chapter provides the historic and scientific framework by which prevention efforts are implemented today. Although the list is not comprehensive, topics that cover both unintentional and intentional injury and strategies that represent best practices and some newer promising practices in the United States are discussed. Many of these practices have evaluation and cost-effectiveness built in from program inception and stand as examples of the scientific principles presented in the chapter. Additionally, disparities in access to trauma care and the role social determinants of health play in injury incidence and treatment are important issues to address at a systems level and will also be addressed. Finally, the global implications of injury are reviewed. This includes injury as it affects low- and middle-income countries and developing strategies in surveillance, prevention, and injury control necessary to make a difference among populations at greatest risk.

SCIENCE AND ADVOCACY

History of Injury Prevention

Historically, injuries were seen as "accidents" that could not be predicted and, therefore, could not be prevented. This limited perspective resulted in an unaggressive and restricted approach to injury prevention that had little effect.[15,16] Over the past 100 years, several visionary individuals had successive insights that established the public health basis for injury prevention. These frameworks resulted in a rational approach that now guides effective injury prevention.

The epidemiologist John E. Gordon pointed out that injuries can be evaluated using the standard epidemiologic framework of host, agent, and environment. Just like any other condition affecting human health, Gordon explained that injuries were not random, but occurred with recognizable patterns across time and populations.[17] This was a paradigm shift from single-cause explanations that inadequately described the injury event and, therefore, limited prevention opportunities, to a multifactorial understanding of the components of injury. This would allow injuries to be studied from several perspectives and opportunities for prevention to be identified.[18]

The fundamental work done by Gordon applying public health principles to injury set the stage for the most notable of the early pioneers of injury prevention, William Haddon. He was the first director of the National Highway Traffic Safety Administration (NHTSA) and is most well-known for his expansion of Gordon's epidemiologic framework for injury prevention. This was by incorporating a temporal element to the host-agent-environment schema, which ultimately became known as Haddon Matrix (Table 3-2).[19]

TABLE 3-2: Application of Haddon Matrix to a Common Injury (elderly falls)

	Human factors	Agent factors	Environmental factors[a]
Pre-event	Visual impairment, agility Prevention of weakness or depression	Padding or softening of floor or other surface Kinesthetically friendly stairs	Handrails Removal of slippery rugs/low objects Adequate lighting
Event	Prevention of osteoporosis, social isolation	Removal of sharp objects potentially in the way Easily accessible alert system	Short emergency medical services response time Family members present
Postevent	Optimize nutrition Early and aggressive physical rehabilitation	Repair damage to home	Mitigation of health care costs Support for possible loss of independence (assisted living)

[a]Environmental factors can include social, political, and cultural factors. Some versions of Haddon Matrix include this element separately as a fourth factor (sociocultural environment).

Source: Adapted from Haddon W Jr. Advances in the epidemiology of injuries as a basis for public policy. *Public Health Rep*. 1980;95(5):411-421.

The pre-event phase allows us to examine the factors surrounding host, agent, and environment that influence the likelihood that an event capable of producing an injury will occur (eg, a motor vehicle crash). An example of a host factor in the preinjury phase would be alcohol impairment, agent factors could include brakes or maintenance, and an environmental factor could be road condition. During the event phase, there are factors influencing the probability that the event (ie, motor vehicle crash) will result in an injury and, if so, to what extent. A host factor during the event could be seatbelt use, an agent factor might be crush resistance of the car, and an environmental factor could be the presence or absence of dividers that would keep the car from ricocheting into ongoing traffic. In the postevent phase, these three components (host, agent, and environment) can be evaluated for factors that influence the ultimate consequences of injury.

This conceptual framework was further leveraged by Haddon to develop 10 strategies that formed the foundation of most current injury prevention and control efforts (Table 3-3).[19] Haddon's work marks the most pronounced shift in the transition from a simplistic, single-cause, individual-level perspective of injury events to complex, multifactorial, societal-level causation. Haddon's approach also integrated multidisciplinary involvement into injury prevention, including clinicians, epidemiologists, engineers, law enforcement agencies, policy experts, educators, and mental health experts.

Principles of Injury Prevention

Most interventions can be thought of as either being active or passive on the part of the person being protected. *Active* interventions involve a behavior change and require people to perform an act such as putting on a helmet, fastening a seatbelt, or using a trigger lock for a handgun. *Passive* interventions require no action on the part of those being protected and are built into the design of the agent or the environment, such as airbags or separation of vehicle routes and pedestrian walkways. Passive interventions are generally considered more reliable than active ones[19,20]; however, many interventions that are considered passive still inherently carry an active component, even if it is at the societal or political level, such as passing legislation to require certain safety features in automobiles.

Another framework often applied to injury prevention strategies is that of the following "three E's": (1) enforcement and legislation; (2) education and behavior change; and (3) engineering and environmental modifications. Initially, education was the main area of focus for injury prevention. If applied uncritically without a strong framework and thorough evaluation, behavior change through educational interventions in isolation can be difficult to achieve. A comprehensive report has suggested that the most effective interventions are engineering/environment, followed by enforcement, and lastly by education.[20] Educational interventions are usually most effective when complemented with modalities from the

TABLE 3-3: Haddon's 10 Strategies That Form the Foundation of Most Current Injury Prevention and Control Efforts

Phase	Strategy	Example
Pre-event		
	1. Prevent the creation of the hazard; prevent the development of the energy that would lead to a harmful transfer.	Prevent manufacture of certain poisons, fireworks, or handguns
	2. Reduce the amount of the hazard.	Reduce speeds of vehicles
	3. Prevent the release of the hazard that already exists.	Place a trigger lock on a gun
Event		
	4. Modify the rate or spatial distribution of the release of the hazard from its source.	Seatbelts, airbags
	5. Separate in time or space the hazard being released from the people to be protected.	Separation of vehicular traffic and pedestrian walkways
	6. Separate the hazard from the people to be protected by a mechanical barrier.	Protective helmets
	7. Modify the basic structure or quality of the hazard to reduce the energy load per unit area.	Breakaway roadside poles, rounding sharp edges of a household table
	8. Make what is to be protected (both living and nonliving) more resistant to damage from the hazard.	Fire- and earthquake-resistant buildings, prevention of osteoporosis
Postevent		
	9. Detect and counter the damage already done by the environmental hazard.	Emergency medical care
	10. Stabilize, repair, and rehabilitate the damaged object.	Acute care, reconstructive surgery, physical therapy

Source: Adapted from Haddon W Jr. Advances in the epidemiology of injuries as a basis for public policy. *Public Health Rep*. 1980;95(5):411-421.

other "E's"; that is, the most effective injury strategies typically have components of all three. An example is the child safety seat, an engineering solution for injury prevention, which was only successfully implemented through successful education campaigns and careful law enforcement.[21]

Other factors that must be considered when choosing and implementing injury control strategies are fidelity versus adaptability. Fidelity refers to the measure to which a program is implemented as intended and has been found to influence the measured effectiveness of an outcome.[22] While fidelity to the program's intended implementation is critical to achieving desirable outcomes, contexts may differ widely in a number of ways. These range from socioeconomic characteristics of the population served to cultural nuances that may influence implementation of the program. Adaptability is the ability of a program to be modified so that it is applicable in a specific context. An effective injury control program needs to strike an appropriate balance between fidelity to established, evidence-based methodology, while being adaptable enough to maintain relevance to the specific population being served. Often, the fidelity and adaptability of a specific program will influence its prioritization among potential interventions in injury control.

Prioritization of targets in injury control for intervention depends on multiple factors. The frequency and severity of a type of injury are fundamental to whether investments should be made to prevent or improve treatments for that injury; that is, having a solid base of evidence for the epidemiology of injury is key to prioritization. The cost of injuries in terms of direct health care costs and indirect societal and economic effects must also be considered. Effective arguments for implementing an injury control program can be made if savings in terms of averted injury-associated costs are demonstrated. Awareness of the importance of cost-effectiveness analyses and their potential as a tool for advocacy is steadily increasing.[22-25] Understanding of the resources available to fund and sustain the intervention is of primary consideration, as well, and will clearly influence the intervention chosen. Finally, less easily quantifiable but equally important are the acceptability and feasibility (including political) of a program in the community. When several strategies for injury control are available and found to be acceptable as potential interventions, prioritizing them may be difficult. Sustainability of a potential program is essential if it is to provide long-term effect. Therefore, assessing the ability of a program to become ultimately accepted and sustained may play into the decision as to whether or not to adopt it. An "institutionalized" program is one that achieves ongoing support and commitment from the agency, organization, or community in which it is based.[15]

Certain common characteristics run through many successful injury prevention programs. These include a multidisciplinary approach and community involvement, and there should be ongoing evaluation of both the process and outcome of the program. Depending on the targeted injury type, a program might involve contributions from the following: health care professionals, public health practitioners, policymakers, engineers, urban planners, epidemiologists, psychologists, manufacturers, traffic safety and law enforcement officials, experts in biomechanics, educators, and individuals associated with the media, advertising, and public relations. Health care professionals might include those in primary care, such as pediatricians, and those involved in acute trauma care. Finally, individual members of the public might be involved.[19,26]

Social Determinants of Health

The World Health Organization (WHO) defines "social determinants of health" as "the conditions in which people are born, grow, live, work and age," and these are largely determined by the unequal distribution of resources and power among populations.[27] Social determinants of health include factors such as education level, local availability of healthy foods, wealth attainment, employment opportunities, and gender equity, and all contribute to health inequities seen nationally and globally. One stark example of health inequity in the United States is the grossly unequal distribution of youth violence, which disproportionately affects African American and Latino communities. This can be tied to social factors such as disparities in housing, education, criminal justice, and economic development, among others.[28,29] This known inequity is further compounded in communities where trauma centers are located geographically far from those populations most affected by violence, resulting in prolonged transport times and potentially increased mortality.[30] Understanding the contribution of social determinants of health to a population's vulnerability to certain injury mechanisms and the limitations to access to care are critical. These identify risk and protective factors that inform coordinated prevention efforts to support those populations at highest risk. Any injury prevention initiative should incorporate a thorough assessment of the social determinants of health influencing the communities affected by injury and include these elements from program design through evaluation. Many of these social determinants, on an individual and ultimately on a community level, are modifiable. Efforts in modifiable risk factors often represent the backbone of injury prevention programs.

Injury Control: From Surveillance to Dissemination

The public health approach can be applied to injury prevention and control as it is applied to any problem at the population level. This approach is composed of the components described below:

1. Surveillance
2. Risk factor identification
3. Ascertaining natural history
4. Intervention
5. Evaluation
6. Dissemination

The components of a comprehensive injury prevention program are discussed in Table 3-4.

TABLE 3-4: Components of an Injury Prevention Program

Components	Role of the surgeon
1. Problem identification and targeted intervention	
Focus on severe and/or common problems (significant morbidity and mortality)	Evidence-based prioritization of problems
Identify potential intervention	Exploration of injury epidemiology through hospital-based data
Evaluate available information on the problem and possible interventions	
Choose appropriate and effective intervention	
2. Stakeholder engagement	
Identify potential coalition members, including clinicians, public health practitioners, community partners, government agencies, related industry, among others	Partner with injury prevention efforts
Identify one of the partners as the lead agency	Provide testimony describing the personal consequences of injury to engage stakeholders
	Connect stakeholders to patients (with their consent) for prevention efforts
3. Data gathering	
Identify potential challenges/obstacles (eg, lack of political will, opposition by interest groups)	Contribute to injury metrics (hospital-based data)
Choose metrics (process, outcome, surrogate)	Quantification of the direct cost of injury
Cost-effectiveness analysis	
4. Reduction of barriers to implementation/use of intervention	
A public information campaign to change a dangerous behavior	Advocacy based on human toll of injuries
A change in a law or the enforcement/application of a law	Empower and encourage our patients to be advocates
Change in the availability or characteristics of a product	
Change in a hazardous environment	
5. Funding	
Explore funding sources: community, foundation, governmental, industry, etc.	Collaborating in grant applications
Secure adequate seed funding and establish basis for future funding	
6. Advocacy	
Placing the burden of injury relative to other public health issues	Leverage societal role to call for collective action toward injury reduction
Creating partnerships between trauma centers and "champions" of a particular injury cause to generate groundswell	Experience-based advocacy through media, legislators, health departments, and hospital administration for injury prevention prioritization
7. Surveillance	
Identification of data sources (eg, police, hospital, autopsy, traffic safety administration)	Participation through trauma registry data
Comprehensive surveillance system	
Ongoing data collection	
8. Education/dissemination	
Identify public forums by which to inform: schools, health commission meetings, town hall gatherings, seminars, community organization meetings	Patient education
	Community advocacy
9. Evaluation	
Process measures (eg, legislation or policy change, change in the built environment, educational goals met, increased use of devices)	Participation through trauma registry data
	Continued injury surveillance
Outcome measures (reduction in injury events, deaths, or severity)	
10. Institutionalization (sustainability)	
Program becomes a regular part of the function of government or other groups	Patient education
	Advocacy to interest groups
Guard against successful programs being rolled back by opposing interest	

PROGRAM EVALUATION

Evaluation of an injury prevention program allows implementers to assess program effectiveness and make appropriate improvements. It also provides quantitative information for funders, increasing the program's accountability and support, and ensures that resources are being used in a beneficial and cost-effective way.[31,32] Although the science of program evaluation is extensive, a comprehensive review is beyond the scope of this discussion. Therefore, a brief overview of the necessary components and underlying standards is provided.

Program evaluation should be built into the program from inception, including during the development of theoretical frameworks supporting the program's premise. For example, a logic model developed in anticipation of forming a program should be assessed for the validity of underlying assumptions before implementation of the program.[32] Early stakeholder engagement is key to this process, as stakeholders can often identify gaps in the theoretical basis of a proposed program and help supply alternatives or solutions. As a program is developed, it is important to have discrete, agreed-upon metrics by which to assess the program, so that progress can be measured and seen by all those involved.

Program evaluation should focus on two broad areas including formative evaluation and summative evaluation (results, impact, and outcome evaluation).[32] Formative measures may include measurable goals inherent to the program's development, such as acquisition of human and capital resources or construction of program components. Summative evaluation measures are specific to the delivery of the program, outcomes of interest that the program is intended to influence, and the impact that the program is having on the community.[32] These measures can be a combination of process measures, impact measures, and outcome measures.[33] Process measures include measuring the success of delivering the program's services to the intended community.

Selection of these measures should be done early in program development, so that evaluation is built into the infrastructure of the program. In order to address these two areas, a combination of qualitative and quantitative evaluation metrics is often needed. When areas of research are novel and established quantitative metrics are unavailable, qualitative evaluation can be very helpful in exploring themes that can then be used to inform future quantitative evaluation measures. This approach helps to avoid investigator bias in terms of what is valued as a meaningful metric by the population served. Qualitative evaluation is also helpful in capturing program effects that are challenging to track quantitatively. One example of this is evaluating the influence and impact of culturally competent peer mentorship for violence intervention programs. The effect of this mentorship may not be evident through standard quantitative evaluation, but allowing program participants to speak to their experience in their own words may allow this theme to emerge.

For injuries, data sources used to evaluate outcome measures vary in terms of capture and resource expenditure (Fig. 3-1). Well-designed community-based surveys with

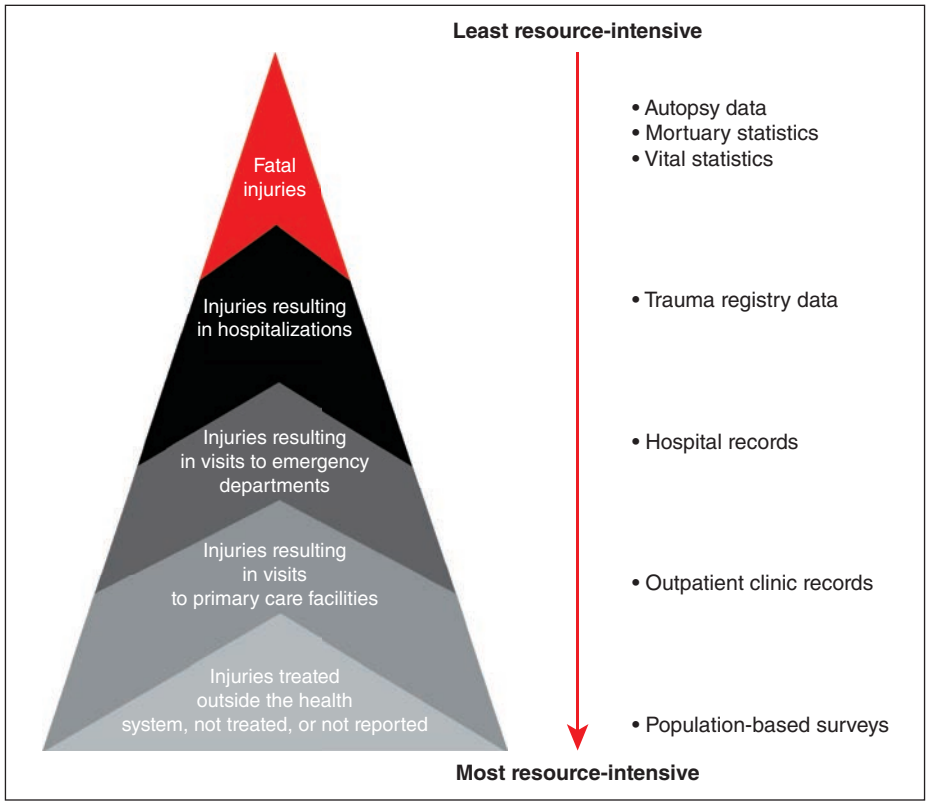

FIGURE 3-1 Examples of data sources used for capturing the burden of injury at different levels of the injury pyramid spectrum. (Reproduced with permission from World Health Organization. *Injuries and Violence: The Facts*. Geneva, Switzerland: World Health Organization; 2014.)

sophisticated sampling strategies are costly and not sustainable on a longitudinal basis, but they provide a good estimate of injury incidence per population. Trauma registries are extremely useful tools for injury surveillance and are present in all Level I trauma centers; however, the population captured is subject to selection biases based on severity and geography. Understanding the nature of the data and its inherent selection biases is critical to building and interpreting an injury surveillance system.[34] Trauma registries can be complemented by other sources of injury data, such as police and emergency medical services (EMS) data, to create a database that approaches representation of the "true" underlying injured population for surveillance purposes. Examples of augmenting individual data sources with complementary data sources through probabilistic linkage and other techniques demonstrate both the utility of this approach and the limitations of each individual data source on its own.[35-37]

Additional data sources that are extremely useful in injury surveillance and prevention efforts include the following:

1. CDC's Web-Based Injury Statistics Query and Reporting System (WISQARS)—https://www.cdc.gov/injury/wisqars/index.html
2. CDC's Wide-Ranging Online Data for Epidemiologic Research (WONDER)—http://wonder.cdc.gov
3. CDC's National Violent Death Reporting System (NVDRS)—https://www.cdc.gov/violenceprevention/nvdrs/index.html
4. ACS's National Trauma Data Bank (NTDB)—https://www.facs.org/quality-programs/trauma/ntdb

The steps in program evaluation have been well defined by the CDC, among others (Fig. 3-2).[38-41] The six steps are listed in Fig. 3-2.

The standards underlying the six steps in Fig. 3-2 strive to ensure that the program evaluation will be useful to users and stakeholders, ethically conducted, and accurate and feasible.[32,33] Ultimately, program evaluation should result in improvement of the program, identification of successful and unsuccessful components, and investment in strategies that work while discontinuing those that do not. For those interested in a more comprehensive review of program evaluation or further in-depth reading on the techniques and tools of program evaluation, references are provided.[32-34,39-41]

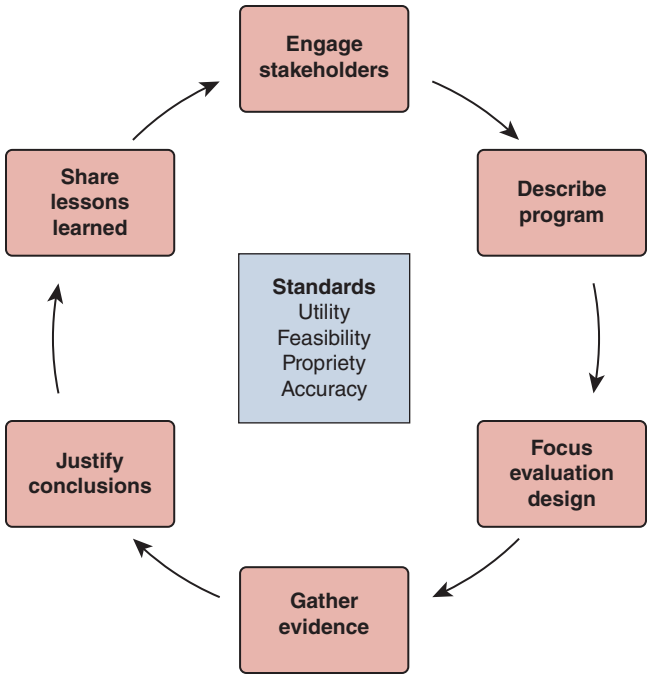

FIGURE 3-2 Example framework for program evaluation.
1. Engage stakeholders (people involved in program operations, the population served, funders, and others affected).
2. Describe the program (needs assessed, expected effects, context, logic model/theoretical framework, activities).
3. Focus the evaluation (identify the measurable effects that are most important to stakeholders).
4. Gather the evidence (indicators, data sources, quality and quantity of data available).
5. Justify conclusions (correlate conclusions using evidence gathered and assess this conclusion against predetermined standards set by the stakeholders).
6. Share lessons (dissemination of results and lessons learned improves the likelihood that the program will be used).

(Adapted from the Centers for Disease Control and Prevention's Framework for Program Evaluation at http://www.cdc.gov/eval/framework/index.htm and the Community Tool Box's Framework for Program Evaluation at http://ctb.ku.edu/en/table-of-contents/evaluate/evaluation/framework-for-evaluation/main.)

SPECIFIC STRATEGIES TO PREVENT UNINTENTIONAL INJURY

Motor Vehicle Safety—Occupants

Motor vehicle safety and injury prevention measures provide a comprehensive example of proven active interventions, passive engineering, legislation, and enforcement strategies that have been modified over decades. In addition, due to detailed surveillance over time, some newer strategies have recently been implemented to address current risk factors such as use of cell phones and targeted strategies for teenage drivers, the highest risk group.

Much has been accomplished to make motor vehicles safer. Engineering features such as "crash avoidance" make it less likely for a vehicle to crash. Other safety features include brakes, headlights, triple brake lights, and signals. Automotive safety also includes engineering features that make occupant injury less likely in the event of a crash. This is referred to as crashworthiness and includes such features as collapsible steering columns, shatterproof glass, and improved side impact protection. These improvements have resulted from both improved car design on the part of the automobile manufacturers and regulations from NHTSA, in the form of Federal Motor Vehicle Safety Standards (FMVSS).

One of the greatest advances in automotive safety was the realization that a significant component of the injuries sustained in crashes was due to secondary collision of the

occupant with the vehicle interior and to ejections. This understanding led to the development of seatbelts to allow occupants to "ride down" the crash, dissipating their kinetic energy more slowly and in a controlled fashion. It is estimated that seatbelt use reduces serious crash-related injury and death by about half. Adding (*not substituting*) airbags provides even greater protection for adults.[41,42] Despite the major progress overall as a result of engineering and enforcement, there remains a population of people least likely to wear seatbelts—teenagers. Of the teenagers who died in motor vehicle crashes in 2012, 55% were not wearing seatbelts.[43] Seatbelt use is an active and necessary prevention measure. Primary enforcement and education by parents and health care providers are strategies that can work, even when facing the challenge of the teen still in a steep behavioral development phase in cognitive growth.

Graduated Licensing

Teen drivers have the highest crash risk per miles traveled of any group; however, newer prevention strategies, most notably graduated licensing, are having positive results on rates of serious motor vehicle crashes. Graduated licensing laws are now present in all 50 states and the District of Columbia and allow for teens to mature and develop skills during an intermediate step. The initial license, for example, may restrict night driving or having young passengers except family members in the car with the new driver. Along with enforcement of drinking and driving laws and properly fitted child car seats, graduated licensing has significantly improved the rate of serious motor vehicle crashes in the United States.[44]

Strategies Regarding Distracted Driving

Along with the convenience of cellular phones for conversations and texting comes the risk of distracted driving. Distracted driving is thought to be responsible for more than 9 deaths and 1100 motor vehicle injuries daily in the United States, an issue that appears to be much worse than in other places with as much cellular access, such as Europe.[42,45] With the raised awareness of the dangers of distracted driving, many states have enacted strict laws regarding texting and driving and have legislated for "hands-free" cellular phone use while driving. A number of laws limiting cellular phone use among federal employees and train operators have also been enacted.

Motor Vehicle Safety—Pedestrians

Injury prevention for pedestrians exemplifies the importance of comprehensive strategic planning among the community, government, and health care agencies. In 2012, over 4700 pedestrians were killed and another 76,000 were injured in the United States. Vulnerable populations include the elderly, children, and the mentally ill.[46] As low- and middle-income countries industrialize at a rapid rate, improvements in road traffic engineering have lagged behind, causing a growing threat to pedestrians in many countries. Passive strategies such as complete separation of pedestrians from motor vehicles have had proven success at reducing risk, but may not be feasible. Other engineering strategies to create a safer environment such as countdown signals (particularly around senior facilities), flashing crosswalks, pedestrian lead time signals, and "bulb outs" have had varying rates of success.

Prevention of traffic injuries often requires a multidisciplinary approach involving community activists, engineering experts, public health departments, police and fire departments, legislators, and public health researchers. An example of a promising practice is the strategic plan called the Vision Zero Initiative. The initiative was created in Sweden and based on the public health principle that "we are human and we make mistakes," but the environmental design should keep us safe, and no loss of life due to traffic injury is acceptable.[47] As such, the initiative places the main burden for safety on comprehensive system design. Plans first enacted in Sweden in 1994 have led to clear reduction in pedestrian deaths and injuries, despite growing amounts of traffic. The initiative has now been adopted in multiple cities worldwide and over 30 cities in the United States, including New York City, Chicago, Los Angeles, Austin, and San Francisco. Central to strategic planning for Vision Zero is surveillance.[48] More recently, Vision Zero has been rolled out in medium-sized cities as well, focusing on implementation of Vision Zero principles, such as speed reduction and road rechannelization projects.

Trauma centers in the United States can play a major role in this initiative, given the evidence that police data only capture a portion of pedestrian and bicycle injuries.[35,37] Trauma center data can serve to augment these data sources to provide more comprehensive injury surveillance and better identify areas of prioritization for strategic planning and policy advocacy. Along with comprehensive plans for road traffic safety, analysis of cost-effectiveness should be rolled into the long-term planning. Therefore, strategies that are cost-effective must include knowledge of the direct and indirect costs of injury.

Prevention of Falls

More than one-third of adults over 65 years of age fall each year in the United States, and for over half, it is not the first fall. One in 10 falls are deemed serious, and over 10% of emergency department visits are due to falls. Individuals who are mentally ill are also at higher risk for falls, but this section will focus on the elderly patient. Risk factors are well known and include visual impairments, polypharmacy leading to disequilibrium and gait disturbances, home hazards such as loose rugs and steps, and lack of grab bars. The costs of falls in the elderly, both personally and financially, are very significant. Severe traumatic brain injury, particularly with increases in usage of antiplatelet therapies, warfarin, and novel oral anticoagulants, poses a serious threat to life and independence.[49,50] The direct medical cost of elderly nonfatal falls exceeds $20 billion annually.[51]

The risk factors listed, however, are preventable. A number of programs in prevention that have reduced falls and

fall recidivism have been implemented over the past decade. Many of these programs focus on home assessments and implementation of safety measures such as securing rugs, creating ramps, and installing grab bars. In addition, physical therapy programs working specifically on gait and balance, reviews of medications and potential medication interactions, and vision screening have all played a successful role in reducing risk. Hospital-implemented efforts in fall prevention, including screening and implementation, have been led in part by the Veterans Administration. These have proven to be promising and have been a way that trauma centers with a high burden of elderly falls can fulfill their injury prevention mandate and provide an active prevention strategy. These are developed in conjunction with senior community resource centers and other groups focused on risk reduction.[52-54]

SPECIFIC STRATEGIES TO PREVENT INTENTIONAL INJURY

Firearm Violence

The leadership of the ACS COT has made prevention of firearm violence a major priority and has adopted a public health approach. Initially, a survey of the ACS Board of Regents and members of the ACS COT and American Association for the Surgery of Trauma was conducted to understand the perspectives and values of surgeons.[55] Subsequent critical initiatives have included engaging owners of firearms in order to promote safety without infringing upon the rights afforded by the Second Amendment. The October 2017 ACS *Bulletin* was devoted entirely to violence prevention and intervention efforts.[56] Members of the COT's Injury Prevention and Control Committee (IPCC), under the leadership of Dr. Deborah Kuhls, have worked in the fields of policy and advocacy and development of a safe storage toolkit. In addition, they have partnered with the Health Alliance for Violence Intervention (HAVI) and supporting groups such as the American Foundation for Firearm Injury Reduction in Medicine (AFFIRM), which works to fund firearm injury research. The development of this group is in part a result of the dearth of federal funding for gun violence research with the passing of the 1996 Dickey Amendment. The COT's IPCC has recently produced a downloadable brochure, "Gun Safety and Your Health," as a guide to keeping families and communities safe.[57] Specific injury prevention strategies are described later in this chapter.

There is an association with the proximity of firearms and risk of death in a variety of populations along the life cycle. Sixty-three percent of firearm deaths in the pediatric population are from suicide, and the availability of a firearm is an independent risk factor.[58] In contrast, the horrific uptick of mass shootings in schools accounts for less than 1% of firearm deaths and unintentional shootings for 2%. Strategies to reduce risk include engaging parents and children in screening for at-risk behavior and providing guidance for safe storage practices. Victims of a domestic dispute are five times more likely to be murdered if a firearm is present. Senior citizens, as with children, are more likely to die from a suicide attempt if a firearm is present in the home.

As outlined in a recent article by leaders of the ACS COT, "Freedom with Responsibility: A Consensus Strategy for Preventing Injury, Death, and Disability from Firearm Violence," there are many Americans who have polarized views about firearms, often leading to a "war of words" when it comes to efforts to reduce firearm injury.[59] One common goal between the groups would be to make firearm ownership as safe as possible. The article presents broad, reasonable policy proposals that have the capacity to bring the two sides together for the health and welfare of the population of the United States.[59]

Prevention of Suicide

In 2016, suicide was the 10th highest cause of death for all ages in the United States. It is estimated that 800,000 people die worldwide from suicide annually. Globally, risk factors are most often present in marginalized and discriminated groups. In many countries, including the United States, risk factors include mental illness, prior suicide attempts, personal loss, financial loss, chronic pain, substance abuse, and trauma. In addition, establishing protective factors and resiliency often requires long-term care and counseling and are dependent upon access to that care.[60] A comprehensive strategy for suicide prevention is well outlined by the CDC in the guide "Promoting Individual, Family, and Community Connectedness to Prevent Suicidal Behavior," along with focused treatment of mental illness and substance abuse.[61] In 2017, the CDC published "A Technical Package of Policy, Programs, and Practices" for suicide prevention.[62] This is an amalgam of best practices and avenues for advocacy in suicide prevention. The trauma center can serve as a portal by which individuals who have attempted suicide can be provided with resources and the means to reduce harm through partnerships with mental health services. The National Suicide Lifeline is another excellent resource.[63]

Prevention of Child Abuse and Neglect and Elder Abuse

According to Child Protective Services, approximately 676,000 children were abused or neglected in the United States in 2016. These toxic experiences are harmful not only to the development of the young brain, but also to the immune system of the child. Similar to the technical guide for suicide prevention, the CDC has published a guide for the prevention of child abuse and neglect.[64]

Physical or sexual abuse, neglect, and financial or emotional deprivation are all examples of elder abuse. The Institute on Aging has teamed with Adult Protective Services in San Francisco to develop a model Elder Abuse Prevention Program.[65] As obligate reporters of child and elder abuse, practitioners need to remain vigilant and aware of the warning signs.

Prevention of Youth/Interpersonal Violence

It has been over 2 decades since the surgeon general at the time, C. Everett Koop, recognized interpersonal or youth violence as a public health issue. Since that time, health providers have adopted a public health model. This has included assessment of individual risk and protective factors and development of strategies for reduction in harm by implementing, evaluating, and exporting programs that are promising. In the United States, there are over 30 hospital-based violence intervention programs that target youth and young adults who have been violently injured. The public health approach follows the principles of Trauma Informed Care as follows: (1) realizes the widespread impact of trauma and understands potential paths to recovery; (2) recognizes the signs and symptoms of all trauma in clients and families; (3) responds by fully integrating knowledge about trauma into policies, procedures, and practices; and (4) seeks to actively resist trauma recidivism.[66] Conversely, programs based on "scared straight" approaches have not been successful.[67] Three specific guiding components for implementation in many hospital-based programs are as follows: (1) recognition that when an individual is injured, there is a "teachable moment" during which time that individual is most open to risk reduction strategies; (2) culturally aware and competent case managers/intervention specialists approach the individual at the bedside and provide long-term follow-up care; and (3) intervention specialists/case managers assess needs and assist individuals over time to find resources for risk reduction available in the community. The majority of these efforts in risk reduction are a reflection of the social determinants of health discussed earlier in this chapter. Evaluation, including feasibility, utility, intermediate- and long-term results, and cost-effectiveness studies, have been conducted. Results have been promising, with demonstration of successful enrollment and retention (feasibility), risk reduction when looking at intermediate outcomes, reduction in the injury recidivism rate when looking at long-term outcomes, and demonstration of cost-effectiveness.[23,68,69] Some programs have been successful at reducing injury in the most vulnerable populations by securing funding within the city budget through working with legislators and departments of public health. Technical support and advocacy are available for trauma centers interested in implementing a violence intervention program through the HAVI (www.thehavi.org). The ACS *Bulletin* published a "Primer" for starting and sustaining a hospital-based violence intervention program (HVIP).[56] HVIPs have teamed with Cure Violence, a successful community-based violence interrupter strategy, in some cities as part of a comprehensive strategic plan for violence prevention. These partnerships are critical because it often "takes a village" to tackle this complex societal issue.

Intimate Partner and Sexual Violence

Intimate partner violence (IPV) is one of the most common forms of violence in the United States. Up to 1 in 4 women and 1 in 7 men report having been physically assaulted by an intimate partner over their lifetime, a burden disproportionately experienced by women and racial minorities.[70] Of women who are murdered, 55% are victims of an intimate partner. Because an encounter with the health care system is often one of the few times a victim of IPV may be able to access help, the ACS has determined that the trauma surgeon plays an important role in screening for IPV and potentially preventing further harm or even death.[71] Additionally, in centers where IPV has a high incidence, prevention programs should be specifically tailored to identify potential victims and provide supportive risk reduction services. IPV is part of a broader spectrum of gender-based violence (GBV) and sexual violence, which can occur between a man and a woman, between members of the same gender, or among any individuals along a gender spectrum. The hallmark feature of GBV is that the violence is incurred by the victim in association with a power imbalance due to his or her gender or gender identity.[72] A number of prevention strategies have been described to prevent GBV, including advocacy, legislation, and community-based interventions aimed at promoting gender equity. A specific form of GBV that can be "hidden in plain sight" of the health care community is human trafficking.[73] Often, the health care system is one of the few institutional encounters a human trafficking victim will have, providing an opportunity for acute intervention, if health care providers are prepared to recognize the signs. The US Department of Health and Human Services has resources to aid health care providers in identifying and supporting victims of human trafficking.[74]

Horizontal Relationship of Violence

The different forms of violence often have distinct and separate resources available for intervention and prevention. Although separated in text, these forms of violence have significant crossover. Victims of one form of violence are frequently exposed to other forms. Programs across the spectrum of violence often have fidelity in their approach of addressing root causes and risk factors. It is also critical to understand that violence is cumulative in vulnerable populations, across the life spectrum. A trauma-informed care approach to victims of violence is important given the recognition that this not often just a singular event in an individual's life.

Mass Shootings in the United States

Approximately 5% of the world's population resides in the United States, yet 31% of all mass shootings occur here.[75] By definition, mass shootings are events that kill four or more people. Although mass shootings make up less than 1% of all homicides in this country, they are deadlier and are increasing in frequency. These horrific events galvanize people to take action through following advocacy for changes in legislation, political representation, funding for gun violence research, Second Amendment freedoms, mental health care, and educational endeavors. As with other aspects of violence prevention, approaching mass shootings with a public health

approach to drive at the root causes is essential. As championed by the article by Stewart et al,[59] it is only together that we can ultimately protect our freedoms and our safety.

INJURY AS A GLOBAL HEALTH PROBLEM (SEE ALSO CHAPTER 64)

Burden of Injury: Global Disparities

Growing attention has been placed on the burden of injury worldwide and highlighting trauma as a global public health issue. As countries industrialize, road traffic increases along with the risk of injury, especially in contexts where commensurate improvements in road safety have not been achieved.[2] As mentioned at the beginning of this chapter, over 90% of the 5.8 million deaths each year due to injury occur in low- and middle-income countries, where trauma systems are least developed.[76] The etiology of this is likely multifactorial. First, most people in the world live in low- and middle-income countries.[77] In addition, industrialization is associated with an increase in vehicle ownership, traffic density, and traffic mix, all of which contribute to an increased incidence of injury.[78] Additionally, the injury case fatality rate in low- and middle-income countries is higher than in high-income countries, likely due to weaker implementation of prevention efforts and less well-developed prehospital and hospital care. A person injured in a low- or middle-income country has twice the likelihood of dying from his or her injuries than a similarly injured person in a high-income country.[11] As surgery is an integral component of trauma care, it is informative to discuss the distribution of surgical services across countries of different economic strata. Although only 15% of the world's population lives in wealthy countries, 60% of the operations done each year are in these countries. Only 3.5% of the operations performed globally each year are in poor countries, which includes 35% of the global population.[79]

Decreasing the Global Burden of Injury: Opportunities and Challenges

The magnitude of the global burden of injury is clearly great, yet trauma is often not discussed among the world's global health priorities. Of the $4 billion budgeted by the WHO each year, less than 1% is spent on injury. This amount is grossly disproportionate to the 12% of the world's disease burden attributable to injury.[80]

Recent attention on the importance of trauma as a public health priority has led to several encouraging initiatives. The WHO has published several guidelines geared toward low- and middle-income countries that establish the essential components of trauma care and offer guidance toward implementing cost-effective programs in trauma quality improvement.[81,82] These were largely in response to a landmark World Health Assembly (WHA) resolution on trauma care (60.22) calling for improved technical support from WHO for stakeholder countries to address the growing burden of trauma.[83]

In 2015, the growing appreciation of the burden of surgically treated diseases in low- and middle-income countries sparked several landmark developments. As previously mentioned, *The Lancet* created a Commission on Global Surgery, representing contributions from 110 countries. This resulted in a series of reports, publications, and recommendations to guide improvements toward equitable access to affordable surgical care globally.[9] In March 2015, the third edition of the Disease Control Priorities was released, and this featured a renewed focus on surgical issues. Additionally, the WHO and the United Nations launched a Decade of Action for Road Safety in 2011 in over 100 countries, emphasizing the importance of prevention of injuries related to road traffic and treatment of associated trauma.[84] With increasing attention on road traffic injuries as a major contribution to death and disability globally, effective prevention measures are needed. The evidence is mounting that legislation, speed reduction measures, public awareness and advocacy, enforcement, and, ideally, multifaceted interventions incorporating more than one of these approaches can result in significant reductions in crash events, injuries, and mortality.[85]

INJURY CONTROL IN LOW- AND MIDDLE-INCOME COUNTRIES

The basic principles of injury prevention previously outlined in this chapter are universal; however, the application of these principles may vary by context. For example, the distribution of injuries related to road traffic has been shown to vary by country, with pedestrians and drivers of two-wheel vehicles found to be particularly vulnerable in certain low- and middle-income countries.[86-89] Some reports estimate that nearly 55% of road traffic deaths in Africa are due to pedestrian injury, compared to 15% or less in the United States or Europe.[90,91] Similarly, scald burns appear to be more frequent in high-income countries than in low- and middle-income countries, whereas flame burns are more frequent in low- and middle-income countries.[92-95] These differences in the patterns of injury underscore the importance of comprehensive and robust injury surveillance systems to inform evidence-based interventions. In high-income countries, a cornerstone of these systems has been hospital-based trauma registries, a surveillance tool that is increasingly used in low- and middle-income countries.[96-100] In addition to injury surveillance data that are regionally accurate, effective injury prevention efforts can only be successful with early engagement of stakeholders and careful assessments of feasibility to tailor interventions to local contexts.

Speed Bumps. An example of a cost-effective method of reducing highway speeds is the strategic placement of speed bumps. One speed bump is estimated to cost about $1000 to build and is expected to last 10 years. When modeled for the world's most dangerous intersections where 10% of deaths occur, speed bumps are among the most effective and cost-effective public health interventions available, costing approximately $100 for life saved and only $3.26 per DALY achieved.[101]

Helmet Use. Helmet laws are another intervention in injury prevention that has been studied in low- and middle-income countries. In Thailand, enactment of a helmet law was associated with a fivefold increase in helmet use among motorcyclists and a concomitant reduction in head injuries by 41% and deaths by 21%.[102] The cost-effectiveness of this intervention was estimated to be $467 per DALY averted.[103] In Vietnam, a helmet law was adopted more recently after it was found that 60% of the over 11,000 annual road traffic fatalities in that country occurred in motorcycle users. Preliminary work suggested that survey respondents were willing to pay for helmets.[104] This public health problem was addressed through a combination of legislation, social marketing, and enforcement, and was ultimately associated with significant decreases in injury events, serious injuries, and deaths captured through hospital and police data sources.[105]

Seatbelts. Improving the use of seatbelts through policy and legislation is another injury prevention strategy recommended by the WHO to improve traffic safety.[2] While seatbelt use has become widespread in high-income countries like the United States, the practice and enforcement in low- and middle-income countries are less prevalent. As part of the Bloomberg Road Safety in 10 Countries project, enforcement campaigns advocating for seatbelt use and speed control in several low- and middle-income countries have shown promise both in increased utilization and decreased injuries.[106,107] Initial interim reports from Russia demonstrate a significant increase in use of seatbelts after implementation of this initiative.[108]

One important concept when considering interventions in road traffic safety is that implementing a multifaceted intervention tends to be both more effective and more cost-effective than implementing a single intervention. For example, implementing a helmet law in combination with a seatbelt law in the same region would not only result in a greater reduction in injuries and deaths, but also be more cost-effective than only one of these interventions. This is because the infrastructure for enforcing one intervention is shared by the other, mitigating the cost.[109] An example of this is the previously mentioned Bloomberg Project, the second phase of which will focus on the following seven interventions: helmet use, seatbelt use, prevention of drunk driving, speed reduction, infrastructure improvements, sustainable urban transport, and improving vehicle transport.

Tertiary Prevention

With the increased focus on injuries as a global public health problem has come an evolving awareness of the importance of strengthening trauma care in resource-constrained settings. The WHO has published several resources aimed at providing technical support to health care providers, policymakers, and hospital administrators to improve trauma care in their context, regardless of resources.[81,82,110] Several programs in low- and middle-income countries have demonstrated improvements in the prehospital care of trauma patients using context-appropriate innovations.[111-115] In the hospital setting, quality improvement (QI) programs have been used as cost-effective means to improve trauma care in all countries.[81] Specific applications of QI techniques, including creation of a trauma registry, the definition of key performance indicators, application of audit filters, and root cause analysis have been associated with a reduction in both preventable deaths and overall mortality in trauma patients in Thailand.[116-118] Similarly, in Pakistan, the utilization of QI principles led to the creation of trauma services. This intervention was associated with significantly decreased odds of both death and complications after implementation.[119] The WHO has also led the implementation of a trauma care checklist that was associated with an improvement in 18 of 19 process measures and a reduction in mortality among the most seriously injured cohort.[120]

CONCLUSIONS: THE SURGEON'S ROLE

The field of injury prevention and control has made dramatic inroads into reducing the toll of death and disability. Particularly in high-income countries, prevention measures against active and passive injury have been successful in increasing the safety of road users, populations at risk for falls or burns, and potential victims of violent injury. Through comprehensive evaluations and the right mix of fidelity and adaptability, efforts in decreasing both intentional and unintentional injury are demonstrating intermediate- and long-term successes. With over 90% of injury deaths occurring in low- and middle-income countries, appropriate attention is now shifting to worldwide surveillance and strategic planning for injury prevention. It is only through this work that the burden of global injury can be improved.

Surgeons have a unique opportunity to participate in all components of prevention from surveillance to programming to evaluation and advocacy. Surgeons see patients during that unique "window of opportunity" after injury, making them powerful advocates not only to individuals and community organizations, but to legislators as well. The injury event, and similarly the hospital itself, is a promising portal of entry into prevention programs. Surgeons have spearheaded projects that have clarified the mechanisms of particular injuries and have implemented and studied programs that can have impact. Surgeons are a vital partner in the multidisciplinary approach necessary to make an appreciable difference in injury-related death and disability globally.

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4

Trauma Systems, Triage, and Transport

David J. Ciesla • Andrew J. Kerwin • Joseph J. Tepas III

KEY POINTS

- Overtriage of minimally injured patients will impair trauma center efficiency, whereas undertriage of severely injured patients will increase the risk of preventable death and disability.
- The major goal of an inclusive trauma system is complete control of all aspects of injury, from effective prevention to successful societal reintegration of injury victims.
- Only 60% of the United States has statewide trauma systems, and approximately 20% has no system at all.
- The numbers and levels of trauma centers should reflect population distribution and the burden of injury within the region.
- Level I trauma centers are distinguished from Level II centers by admission volume requirements, presence of a surgically directed critical care service, educational leadership, and trauma-related research.
- Most current data estimate that 63.1% of the population of the United States can reach a major trauma center within an hour by ambulance, a figure that increases to 90.4% with the inclusion of helicopter services.
- A multiple-casualty incident may exceed the resources of one hospital trauma center, but care can be provided by adjacent hospitals within the city or region without outside help.
- The Simple Triage and Rapid Treatment (START) triage system developed in California includes assessments of the patient's ability to ambulate and the patients' respiratory function, systemic perfusion, and level of consciousness.

TRAUMA SYSTEM DEFINITION

Trauma System Mission

A trauma system is an integrated collaboration of health care providers, agencies, and institutions dedicated to the control of the entire spectrum of injury from effective prevention to efficient societal reintegration of injury survivors. At the system's core is coordinated and comprehensive care of acutely injured patients within a defined geographic area.[1-4] Its services are multidisciplinary and comprehensive and encompass a continuum that includes all phases of patient need.[2,3,5-7]

Trauma System Goals

Because geographical coverage exceeds that of a single hospital or health system, trauma systems provide seamless regionalization of resources to facilitate efficient use of available and qualified health care facilities. Cost-efficient and efficacious coordination of resources is a defining characteristic of an effective trauma system, as is disaster preparedness. The institutions and agencies that form the trauma system provide the regional response to care for victims of natural or manmade disasters. The US Department of Health and Human Services has compiled a set of guidelines and standards for Model Trauma System Planning and Evaluation.[8,9] This comprehensive document was initially released in 2006 and remains an excellent source of information for every aspect of trauma system design and planning. More recently, the National Academies of Sciences, Engineering, and Medicine published its report entitled *A National Trauma Care System: Integrating Military and Civilian Trauma Systems to Achieve Zero Preventable Deaths After Injury*.[9-11] This document recognizes that although there are variations in patterns of injury in civilian and military environments, the spectrum of injury, when assessed across a population, demands access to every appropriate resource from both military and civilian sectors, especially when dealing with a mass casualty event. The document encourages the United States to commit to a national aim of achieving zero preventable deaths after injury and minimizing trauma-related disability. It recommends that the secretaries of Health and Human Services and Defense, together with their governmental, private, and

academic partners, work jointly to ensure that military and civilian trauma systems collect and share common data spanning the entire continuum of care. Within that integrated data network, measures related to prevention, mortality, disability, mental health, patient experience, cost, and clinical outcomes should be made readily accessible and useful to all relevant providers and agencies.

Trauma System Objectives: Transforming from Exclusive to Inclusive Trauma Systems

A modern trauma system is committed to control of the impact of injury on population health. While this is obviously achieved by effective prevention, the system's immediate daily function is focused on the elimination of preventable death. This is predicated on (1) ensuring expeditious access by acutely injured patients at risk of death or disability to the resources of a designated trauma center, and (2) accurate identification of patients who warrant care at these facilities. Although trauma systems evolved as networks of high-capacity centers available for all injury victims, those patients at greatest risk of mortality were the primary focus of triage protocols. As understanding of the requirements for efficacious population-based management of acute trauma has progressed, it has become clear that effective control of this disease requires participation of the entire community.[12] To achieve optimal control of injury on any defined population, trauma systems that had been developed for the exclusive care of the most severely injured required expansion to an inclusive regional enterprise that could address all phases of injury control. Paramount to this is effective integration and collaboration of designated trauma centers with all regional health care facilities. As the American health care system shifts from reactive acute therapy of patient disease to preemptive management of population wellness, all trauma systems must be configured as inclusive. This actually enhances the importance of accurate triage systems. Overtriage of minimally injured patients will impair trauma center efficiency, whereas undertriage of severely injured patients will increase the risk of preventable death and disability.

TRAUMA SYSTEM DEVELOPMENT

The modern timeline of development of comprehensive trauma care began in 1949 when the American College of Surgeons Committee on the Treatment of Fractures, which had been established in 1922, was expanded to become the American College of Surgeons Committee on Trauma (ACSCOT). In 1961, a dedicated trauma unit was opened at the University of Maryland under the leadership of R Adams Cowley, MD, FACS. The National Academy of Sciences and the National Research Council published *Accidental Death and Disability: The Neglected Disease of Modern Society* in 1966.[13] This redefinition of injury as a preventable and treatable disease was a major stimulus to the development and propagation of systems of trauma care.[14]

By 1973, Dr. Cowley's initiative emerged as the Maryland Institute of Emergency Medicine, which became the nation's first completely organized statewide regionalized trauma system. A similar initiative that included designation of trauma centers by state law was initiated by Illinois in 1971.[15] Virginia followed in 1981 and established a statewide trauma system based on volunteer participation and compliance with national standards as defined by ACSCOT.

In 1973, the Emergency Medical Service Systems Act became Public Law 93-154, thereby adding a section to the Public Health Services Act of 1944 (PL 78-410) that encouraged development of comprehensive area-wide emergency medical systems. State and local agencies instituted prehospital care systems to deliver patients to major hospitals where appropriate care could be provided. Prehospital provider curricula were standardized, and training programs were established for paramedics and emergency medical technicians (EMTs).

The American College of Surgeons published the first edition of the *Optimal Hospital Resources for the Care of the Seriously Injured* in 1976, establishing a standard for comprehensive delivery of trauma care. This document was the first to define specific criteria for the categorization of hospitals as trauma centers. The document has been periodically revised to reflect current thought and is recognized nationally and internationally as the standard for hospitals aspiring to be trauma centers. The most recent sixth edition, *Resources for Optimal Care of the Injured Patient*: *2014*, establishes evidence-based criteria to guide prehospital and trauma care personnel and emphasizes the importance of ongoing performance and quality improvement.[5]

ACSCOT developed the Advanced Trauma Life Support (ATLS) course in 1980, motivated in part by the personal experience of a surgeon involved in the crash of his private airplane. His experience and his observations of the care provided to members of his family made him realize that more education about resuscitation and care of the injured patient was critically needed. The resulting ATLS course has contributed to the uniformity of initial care, has developed a common language for all care providers, and has been successfully promulgated throughout the world.

In 1985, the National Research Council and the Institute of Medicine published *Injury in America: A Continuing Health Care Problem*.[16] This document concluded that there had been little progress toward reducing the burden of injury despite considerable funding committed to develop trauma systems. It also recommended investment in epidemiologic research and injury prevention. As a result, the Centers for Disease Control and Prevention (CDC) was designated as the national research center to coordinate efforts in injury control, injury prevention, and all other aspects of trauma care.

In 1987, ACSCOT initiated the Verification/Consultation Program to provide resources for trauma center verification. More recently, ACSCOT added additional focus on systems development with publication of *Consultation for Trauma Systems* to provide guidelines for trauma system evaluation and enhancement.[17] Concomitant with these efforts, the

American College of Emergency Physicians (ACEP) published "Guidelines for Trauma Care Systems."[18] This document also addressed the continuum of trauma care and identified essential criteria for trauma care systems.

In 1988, the National Highway Traffic Safety Administration (NHTSA) established the Statewide Emergency Medical Services (EMS) Technical Assessment Program and the Development of the Trauma Systems Course, both important tools to assess the effectiveness of trauma system components as well as support for ongoing system development. NHTSA also developed standards for EMS quality assessment, including trauma care. These standards required that the trauma care system be fully integrated into the state's EMS system and be supported by appropriate enabling legislation (Table 4-1). In 1990, the Trauma Systems Planning and Development Act created the Division of Trauma and EMS within the Health Resources and Services Administration to improve EMS and trauma care. The program was not funded between 1995 and 2000, as many states were in the process of developing trauma systems. The two most important initiatives from this legislation were provision of competitive planning grants for statewide trauma system development and publication of the Model Trauma Care System Plan as a consensus document. The Model Trauma Care System Plan established an apolitical framework for measuring progress in trauma system development and set the standard for the promulgation of systems of trauma care. The program was again funded in fiscal year 2001 but lost funding in 2006. Presently, the 115th Congress is considering H.R. 880, The Military Injury Surgical Systems Integrated Operationally Nationwide to Achieve ZERO Preventable Deaths (MISSION ZERO) Act. It would establish a grant program for partnerships between the military and civilian trauma centers to allow qualified military personnel to provide trauma care in civilian trauma centers and provide $15 million annually for grants to eligible civilian trauma centers through fiscal year 2022.

TABLE 4-1: Criteria for Statewide Trauma Care System

Criteria	Description
Legal designation authority	State governmental authority to legally designate, certify, identify, or categorize trauma centers
Formal designation process	Formal process for hospital selection, designation, and periodic review
Designation based on American College of Surgeons (ACS) standards	Designation of a hospital's trauma center as Level I through IV based on ACS definitions
On-site verification of standards	On-site external review to verify compliance with trauma center standards
Limited number of trauma centers	Limited number of major trauma centers based on community need to concentrate expertise and scarce resources at key hospitals
Prehospital triage criteria	Prehospital protocols allowing emergency medical services (EMS) to bypass nondesignated hospitals for major trauma patients
Process to measure systems outcomes	Formal process to monitor system-wide performance, which includes a trauma registry and external committee that monitors patient outcomes
Full geographic coverage	Coordination of EMS and hospital resources to ensure access to trauma resources independent of proximity to trauma centers

Source: Data from West JG, Williams MJ, Trunkey DD, Wolferth CC. Trauma systems: current status—future challenges. *JAMA.* 1988;259:3597.

Modern Challenges

The evolution of trauma systems reflects two very critical elements of modern health care. First is the obvious mandate to ensure that properly trained and experienced personnel with the right resources are immediately available for every injured patient. Linked to this is the reality that this extensive and intensive investment in personnel and resources demands accountability defined by better outcomes, more cost-effective and efficacious care, and good stewardship of expensive resources. Injury care is expensive, and the impact of ongoing chronic care costs and lost human productivity is enormous. The true impact of this global health problem is not completely measurable, especially when diminished quality of life for survivors and their families is considered.

The current transformation of the health care system in the United States is focused on increased patient coverage, improved quality of care, and lower cost. This is often defined in terms of the triple aims of better care, lower cost, and improved population health.[19] From the perspective of trauma systems, these goals demand careful and ongoing review of the overall performance and effectiveness of every component of the system. The critical factor that guides planning of a trauma system is the balance of injury risk versus total cost of disease control provided to the specific population served. While the ideal concept might be a designated trauma center within 5 minutes of ground transport for every citizen, the cost of maintaining such a network and the dilution of skills in management of severe, multiorgan injury that would occur with this model would increase societal cost with no expectation of improved clinical outcomes. Some evidence suggests that too many Level I trauma centers in a region actually *increased* the risk of death in some patients.[20] Moreover, the likelihood of inexperience driving unnecessary or avoidable cost is significant. The converse is also problematic. Too few trauma centers can lead to an increase in preventable mortality, especially if access requires long-distance transport. Careful planning of trauma systems must consider surrounding community resources, traffic patterns, modes of transport, and trauma density to avoid transport delays and achieve the best outcomes for patients.[21]

From a population-based perspective, trauma system performance must be measured in terms of efficacy in maintaining wellness (injury prevention) and efficiency in treatment of injury victims (outcome). The population of injured patients is skewed toward a majority with non–life-threatening injury for whom timely, appropriate care should produce optimal recovery and avoidance of unnecessary expense. To this is added a complex core of severely injured who demand multidisciplinary care from qualified providers whose experience can contribute to optimal outcome in a cost-effective manner. Thus, in the emerging, modern health care environment, an accountable trauma system must balance the competing mandates of immediate access to the care of qualified, experienced personnel in designated facilities against unnecessary cost to the entire population generated by inappropriate replication of these expensive resources.[22,23] In a statement released in February 2015, the American College of Surgeons (ACS) emphasized the importance of controlling allocation of trauma centers. An expert panel was convened by ACS in August 2015 to develop a statistical basis for determination of trauma center need and location. The resulting "Needs Based Assessment of Trauma Systems" attempts to apply objective metrics of patient volume, capability, and support to determine appropriateness of trauma centers.[24] The group's report begins by emphasizing that designation of trauma centers based on regional population need has been recognized as an essential component of trauma system design since the 1980s.[25] To date, few trauma systems have operationalized these concepts, especially when faced with real or potential challenges that stem from powerful health care institutions or providers.

Essential Components of Trauma System Development

The critical milestones necessary to support statewide EMS and trauma system development as initially defined by West et al[1] are listed in Table 4-1. Attainment of these criteria is a stepwise progression that begins with documentation of need.[1,5] Historically, this has been accomplished by reviewing regional trauma case experience with special focus on preventable deaths.[25,26] This is often the critical driver that establishes legal authority for system development. This authority usually proceeds from legislation at a state or local level that authorizes an appropriate agency to define criteria for participation, establish a trauma registry for system quality assurance, and implement processes for verification and designation. The authorizing legislation must also include adequate appropriation of resources for ongoing system management, including periodic needs assessment, assurance of fiscal solvency, and continuous focus on clinical outcomes. The surgeon's leadership and commitment to optimal care in articulating need and in guidance of system planning are critical. Designation requires continuous reporting of objective metrics of accountability from all participating entities. After verification of decreasing injury incidence, improved patient care, and improving outcomes, one of the most critical components to be assessed in the deployment of a trauma system is cost (Table 4-2). The major goal of an inclusive trauma system is complete control of all aspects of injury from effective prevention to successful societal reintegration of injury victims; however, reality dictates that each component of this system have an adequate financial base to support its mission. The ideal is that all injured patients reach definitive care in a time frame and at a level that are appropriate to meet their injury needs. Unfortunately, the cost of the ideal must be balanced against what is financially sustainable. The determination of need for a trauma center must first reflect its contribution to the overall capability of the trauma system to meet the population's demand. Part of this assessment must include overall taxpayer cost. As previously stated, too few trauma centers do not adequately serve the population, resulting in a more profound effect of injury on human productivity and longitudinal cost of care. Too many trauma centers produce unnecessary overhead to population health care costs, avoidable duplication of services, and potential dilution of provider skills in management of complex cases.[27] Trauma system funding parallels the challenges of providing coverage for individual patients. An unregulated "free-market" approach that enables hospitals to establish trauma programs places the burden of financial viability on the hospital. Fiscal solvency would be sensitive to payer mix, resulting in financial stress to those facilities that serve unfunded patients. Subsidizing these hospitals with public funds to cover gaps in reimbursement adds an additional financial burden to all taxpayers, since a portion of all tax dollars already pays for the medical coverage of the uninsured. Because hospitals try to cover reimbursement deficits by raising prices, the overall cost of care for all citizens increases. This cost shift creates a triple taxpayer burden of higher health insurance premiums, additional taxation to support care of those without adequate health insurance, and reallocation of existing tax revenue to provide special subsidies to many of the institutions that are the default providers of care to these underfunded patients. The provision of comprehensive trauma care is a noble cause but must be supported by a financial margin that does not undermine the fiscal solvency of the entire American health care system.

TABLE 4-2: Emergency Medical Service System Components

Regulation and policy
Resource management
Human resources and training
Transportation
Facilities
Communications
Trauma systems
Public information and education
Medical direction
Evaluation

Source: National Highway Traffic Safety Administration. *Development of Trauma Systems (DOT).* Washington, DC: National Highway Traffic Safety Administration; 1988.

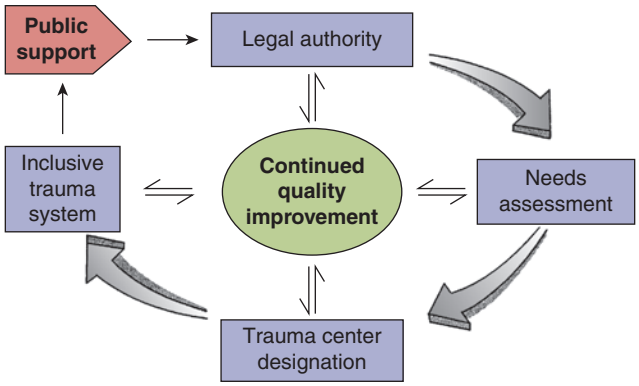

FIGURE 4-1 Regional trauma system development must progress in a sequential fashion; a comprehensive needs assessment is a pivotal early step. (Reproduced with permission from Moore EE. Trauma systems, trauma centers, and trauma surgeons: opportunity in managed competition. *J Trauma.* 1995;39:1.)

TRAUMA SYSTEM COMPONENTS

Despite the demonstrated efficacy of trauma systems, recent data show that only 60% of the United States has statewide trauma systems, and about 20% has no trauma system at all.[25,28-31] The functional elements of a trauma system are disease surveillance, system access, prehospital care, hospital inpatient management, rehabilitation, prevention, disaster medical planning, patient education, research, and accountable financial planning. Prehospital communications, a transport system, trained personnel, and qualified trauma specialists for all phases of care are essential for a system's success (Fig. 4-1).

Quality assessment and performance improvement is a vital component of the system because it guides identification of best practice and constantly evaluates the system's performance and needs.[32-35]

Based on the concept of the "inclusive system" (Fig. 4-2), trauma *centers* are identified by their ability to provide definitive care to the most critically injured, and the trauma *system* is assessed by its ability to manage all injuries.[36-38] Only about 15% of all injured patients will benefit from the resources of a Level I or II trauma center. Therefore, an inclusive system must encourage participation and enhance capabilities of the smaller hospitals. Surgical leadership is essential to development of trauma systems. In fact, trauma systems cannot develop without the commitment of the surgeons of a hospital or community. The model inclusive system is defined within a geographic area, and all health care resources are included in its planning. A centralized Level I trauma center concentrates highest risk injuries, whereas more distant, lower level centers care for less severely injured according to their capabilities and can serve as an access point for patients needing immediate care. EMS knits the system together.

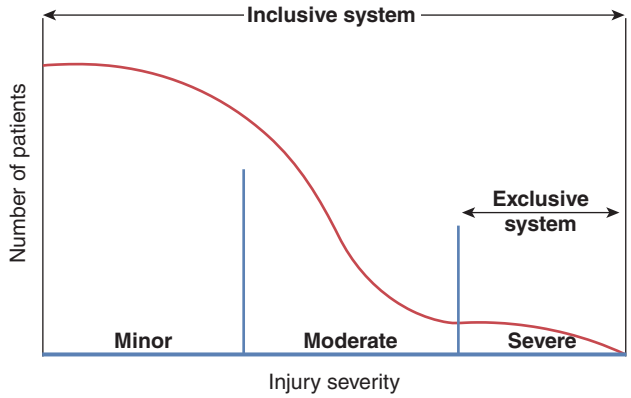

FIGURE 4-2 Diagram showing the growth of the trauma care system to become inclusive. Note that the number of injured patients is inversely proportional to the severity of their injuries.

Public Information, Education, and Injury Prevention

Death following injury occurs in a trimodal distribution defined in minutes, hours, and days. Effective trauma programs must also focus on injury prevention, since more than half of the deaths occur within minutes of injury and will never be addressed by acute care. Because trauma is not considered an important public health problem by the general population, efforts to increase awareness of the public as well as to instruct the public about how the system operates and how to access the system are important and mandatory. A Harris Poll conducted by the American Trauma Society showed that most citizens value existence of a trauma system with the same importance as fire and police services. Continuous epidemiologic surveillance to define interventions that will likely reduce both injury occurrence and severity requires trauma systems to focus on injury prevention. Identification of risk factors and high-risk groups, development of strategies to alter personal behavior through education or legislation, and other preventive measures have the greatest impact on trauma in the community and, over time, will have the greatest effect on all trauma victims.[19,22,24] If prevention is the only effective vaccine against injury, effective public education is the most critical ingredient of the vaccine.

HUMAN RESOURCES

Because the system cannot function optimally without qualified personnel, a high-quality system ensures comprehensive and continuing education to its providers. This includes all personnel along the trauma care continuum: physicians, nurses, EMTs, therapists, psychologists, and others who impact the patient and/or the patient's family. In all trauma centers, there must be a defined trauma resuscitation team consisting of predesignated personnel with specific assignments. The team's main purpose is rapid patient assessment, provision of immediately lifesaving interventions, initiation of comprehensive resuscitation, expeditious diagnostic workup, and provision of definitive care. Critical to the resuscitation team's function is linkage to EMS and timely prehospital notification. For major trauma patients identified in the field, the resuscitation team must be preassembled and

immediately available upon patient arrival. The constituents, role, and capabilities of the resuscitation team depend on the level of trauma centers.

PREHOSPITAL

Trauma care prior to hospital arrival has a direct effect on survival. The system must ensure prompt access and dispatch of qualified personnel, appropriate care at the scene, and safe and rapid transport of the patient to the closest, most appropriate facility. The primary focus is on education of paramedical personnel to provide initial resuscitation, triage, and treatment of trauma patients. Effective prehospital care requires coordination between various public safety agencies and hospitals to maximize efficiency, minimize duplication of services, and provide care at a reasonable cost. There are many comprehensive and standardized courses covering virtually every aspect of prehospital care. Most are the products of national and international organizations dedicated to emergency medical care and should be considered as the preferred alternative to "home-grown" courses or periodic symposia that provide inconsistent educational impact. Also of note is the fact that many injured patients are brought to hospitals via private vehicles. All hospitals should have a system to assess these patients and accurately triage them as appropriate.

COMMUNICATIONS SYSTEM

A reliable communications system is essential for providing optimal trauma care. Although many urban centers have used modern electronic technology to establish emergency systems, most rural communities have not. A communications system must include universal access to emergency telephone numbers (eg, 911), trained dispatch personnel who can efficiently match EMS expertise with the patient's needs, and the capability of EMS personnel at the trauma incident to communicate with prehospital dispatch, the trauma hospital, and other units. Post hoc review of many of the most recent mass casualty shootings has determined that multiple organizations responding from adjacent areas are often controlled by communication centers that cannot communicate with each other. Access also requires that all citizens know how to access and actually use the system. This can be achieved through public safety information and school educational programs designed to inform health care providers and the public about emergency medical access.

MEDICAL DIRECTION

Medical direction provides the operational matrix for care provided in the field. It grants freedom of action and limitations to EMTs who must rescue injured patients. The medical director is responsible for the design and implementation of field treatment guidelines, their timely revision, and their quality control. Medical direction can be "off-line" in the form of protocols for training, triage, treatment, transport, and technical skill operations or "online," given directly to the field provider. Simulation is an important part of ensuring appropriate training for prehospital personnel.

DEFINITIVE CARE FACILITIES

Acute care hospitals are the foundation of the regional trauma system. An inclusive trauma system integrates acute care facilities of all levels to provide the full spectrum of injury care within its region. Central to trauma system planning is the designation of definitive trauma care facilities to meet community need. The number and levels of trauma centers should reflect population distribution and the burden of injury within the region. In principle, the designating authority is responsible for determining the number and level of trauma centers needed to provide optimal care in its region. In practice, trauma centers and acute care hospitals should coexist within a region and cooperate to ensure appropriate distribution of patients based on resource needs. Every institution should contribute data to trauma system registries and participate in system performance improvement programs. Level I trauma centers should serve as the lead agency within a region to ensure that all EMS agencies, acute care hospitals, and trauma centers provide coordinated care within the region.

Trauma Center Facilities and Leadership

Hospital care of the injured patient requires commitment from specific facilities to provide administrative support, medical staff, nursing staff, and other support personnel. The network of institutions that is the core of a trauma system usually includes a variety of facilities whose capabilities reflect the local health care needs of their community. Efficient management of the system is predicated on effective communication and interaction among these participants. Part of this process is determination of level of function expected from each designated center. For most parts of the country, this is accomplished by designation of specific levels of capability based on hospital mission. Level I and Level II centers are committed to comprehensive management of all injured patients, whereas Level III and IV centers usually represent smaller community hospitals committed to optimal care of any injured patient with potential triage to a higher facility of severely injured patients with complex injury. As enhanced commitment to rural health care has evolved, many rural and critical access hospitals have become incorporated into regional trauma systems as Level IV centers. ACSCOT maintains a continuously updated compendium of qualifications and performance criteria for every component of a trauma system. In all trauma centers, there must be a defined trauma resuscitation team consisting of predesignated personnel with specific assignments. The team's main purpose is rapid patient assessment, provision of immediately lifesaving interventions, initiation of comprehensive resuscitation, expeditious diagnostic workup, and provision of definitive care. Critical to the resuscitation team's function is linkage to EMS and timely prehospital notification. For major trauma patients identified

in the field, the resuscitation team must be preassembled and immediately available upon patient arrival. The constituents, role, and capabilities of the resuscitation team depend on the level of trauma centers.

The key concepts below underscore these requirements. Regardless of level of capability, the common factors across all levels of designation include the following:

- Consistent provision of the necessary personnel and resources to deliver acute care to the injured patient
- Surgical leadership in every aspect of clinical care, including resuscitation, critical care, and definitive multisystem injury management
- A trauma medical director, trauma program manager, trauma registry managed by trained registrars, performance improvement specialist(s), and data analyst(s)
- Seamless integration within the trauma system, especially as related to EMS prehospital communication, interfacility transfer, data sharing, and system performance improvement

LEVEL I CENTER

From the perspective of clinical care to injured patients, Level I and Level II trauma centers are identical. Level I trauma centers are distinguished from Level II centers by the following characteristics:

- Admission volume requirements
- Presence of a surgically directed critical care service
- Educational leadership with graduate medical education programs in multiple specialties
- A productive program in trauma-related research

The Level I trauma center is a regional resource and tertiary care facility capable of providing immediate definitive and comprehensive care to all injured patients regardless of severity or complexity. It should function as the cornerstone of the regional trauma system. The Level I trauma center has a major responsibility for providing leadership in system planning, research, education, and training of trauma care providers. Level I trauma centers are generally located in large, population-dense areas and are typically affiliated with university teaching hospitals. They function as a regional resource center and generally serve large cities or population-dense areas, which is necessary to provide enough experience to develop clinical expertise, train new providers, and fulfill essential research and education missions. A Level I trauma center is often the lead hospital for a regional health care system. In larger urban areas, more than one Level I trauma center may be needed to ensure availability of appropriate resources and appropriate transports to the highest level of care.

LEVEL II CENTER

The Level II trauma center provides definitive care to the injured patient and functions in two distinct roles recognized by ACSCOT.

- A Level II center in a population-dense area may supplement the clinical activity and expertise of the regional Level I center, thereby augmenting capacity.
- In less populated areas distant from the regional Level I center, the Level II center may serve as the lead hospital for its region and provide support to local minor trauma centers in the same service area.

All Level II centers include the specialty services needed to provide definitive care to the severely injured, although clinical capabilities may not be as comprehensive as at Level I centers. Despite clinical capabilities similar to Level I, Level II centers may treat a lower volume of severely injured patients. Although graduate education and research are not required functions of Level II trauma centers, they are required to provide effective access to continuing medical education for all involved professional providers. The trauma medical director must have authority for determining each team member's ability to participate on the trauma panel based on an annual review. Qualified attending surgeons must be present in the emergency department for major resuscitations, to make major therapeutic decisions, and to participate in operative procedures and must be actively involved in the critical care of all seriously injured patients.

LEVEL III CENTER

The Level III center is an entry point to the regional trauma system usually in communities that are remote from major trauma centers. The presence of general surgery capability differentiates the Level III from the Level IV center, with the expectation that a general surgeon will be present in the emergency department to lead resuscitation upon arrival of every major trauma patient. The Level III center provides definitive care to the moderately injured and initial stabilization for the major trauma patient, which may include operative hemorrhage control to ensure safe transfer to a major trauma center. As with the Level IV center, predefined plans for transfer of patients to the major trauma centers are essential.

LEVEL IV CENTER

Level IV trauma centers expand the trauma system to sparsely populated, geographically isolated, and often medically underserved rural communities. These hospitals are typically the only source of medical care for many miles and function as an initial point of evaluation and treatment of injured patients. In principle, the Level IV center serves as an initial access point to the regional trauma system. The Level IV trauma center must have 24-hour emergency coverage and provide initial evaluation and stabilization of injured patients, most of whom will be transferred for definitive treatment. A defining difference between Level IV centers and higher level trauma centers is the absence of continuous surgical and/or orthopedic coverage. Thus, the Level IV trauma center must have an organized trauma resuscitation team that follows standardized protocols and predefined transfer plans for patients needing a higher level of care.

SPECIALTY TRAUMA CENTERS

Regional specialty facilities concentrate expertise in a specific discipline and serve as a valuable resource for patients with critical specialty-oriented injuries. Examples include replantation, pediatric trauma, burns, spinal cord injuries, and hand trauma. Where present, these facilities provide a valuable resource to the community and should be included in the design of the system. Most importantly, the unique capabilities of each must be seamlessly woven into the process of care so that the required specialty care is available at the appropriate time in the continuum of management of the patient. A pediatric trauma center is expected to have the same resuscitative capabilities as any center receiving acutely injured patients from the field. The most recent edition of *Resources for Optimal Care of the Injured Patient* defines pediatric criteria from the perspective of free-standing children's hospitals as well as supplemental capabilities for designated adult trauma centers.[5] A replantation or burn center, on the other hand, is usually reliant on comprehensive initial evaluation of the patient by a referring center with which it must have established transfer guidelines and protocols.

Acute Care Facilities Within the Trauma System

Many general hospitals exist within a trauma care system but are not officially designated as trauma centers. Circumstances often exist in which less severely injured patients reach these hospitals and appropriate care is provided. The trauma system should develop and manage protocols for interfacility transfer of patients whenever a major trauma patient is inappropriately triaged to an undesignated facility. Moreover, the trauma system's registry must be able to identify and track these injured patients managed at nondesignated facilities so that their care can be reviewed as part of a mature performance improvement and patient safety program.

All acute care facilities play important roles in the regional trauma system. Most patients with minor injuries receive effective and definitive care in acute care hospitals in their home communities (Fig. 4-3). This helps minimize the burden on the patient and preserves major trauma center resources for the care of the more seriously injured. Major trauma centers, especially Level I centers, are typically large tertiary referral centers located in population-dense areas and serve as the primary hospital for their communities. As such, major trauma centers also tend to attract many mildly and moderately injured patients simply because of proximity to the population and large market share.[36] Effective trauma system planning must recognize this phenomenon to ensure the proper balance of trauma center numbers and levels within a region. Sufficient minor trauma resources should be available to care for the minorly and moderately injured, and sufficient major trauma center resources should be available to not only ensure access to definitive care for the severely injured, but also concentrate severe injury volume in a limited number of major trauma centers to optimize clinical competency through experience and maximize efficiency

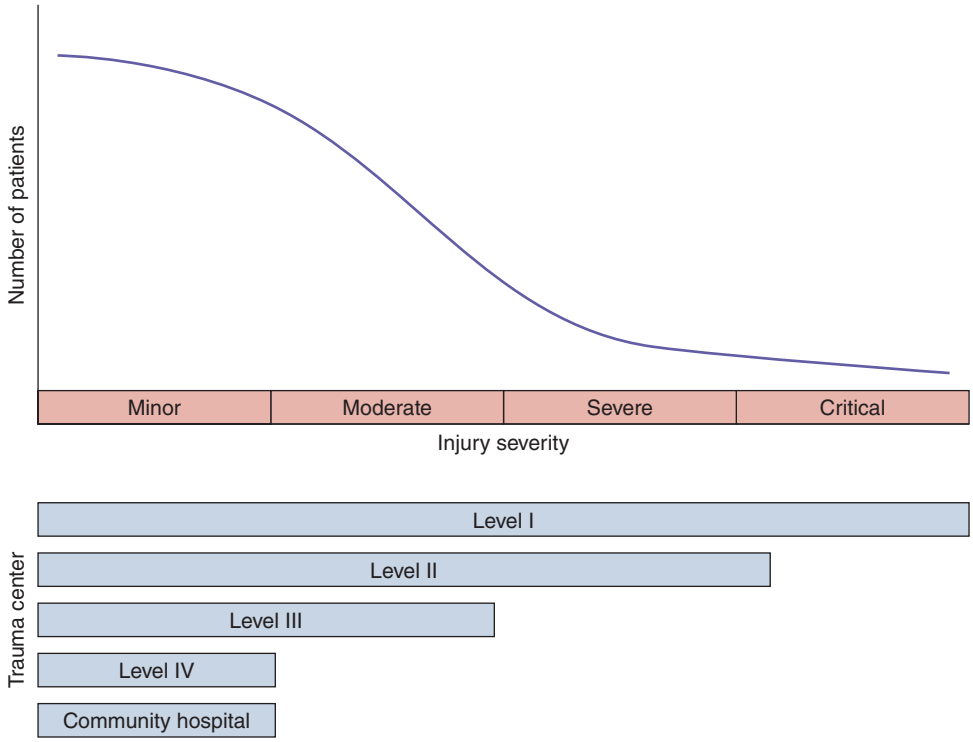

FIGURE 4-3 Distribution of trauma volume and severity within an inclusive trauma system. Proper allocation of resources should focus the bulk of the mission in the higher acuity centers and is predicated on effective communication and interfacility transfer.

through conservation of scarce, high-value human and material resources.

Rehabilitation

Effective control of injury involves a continuum of care that begins with failure of prevention and ends with complete societal reintegration of a recovered patient. In reality, injury can be an affliction that never really ends. The issues of long-term neurocognitive dysfunction and early onset of degenerative disorders are well defined in current literature. Less well understood are the impacts of chronic musculoskeletal disorders, intractable chronic pain, and numerous other impairments that undermine quality of daily living and social function. Rehabilitation is as important as prehospital and acute hospital care. Early and aggressive rehabilitation has been shown both to accelerate and enhance recovery, yet it is often least considered and incompletely available. Many insurance policies do not even cover rehabilitation, leading to a major additional burden for the patient whose coverage ends with hospital discharge. Although it is critically important to reintegrating the patient into society, only 1 of 10 trauma patients in the United States has access to an adequate rehabilitation program. Rehabilitation is often the longest and most difficult phase of care for both patient and family. It can be provided in a designated area within the trauma center or by agreement with a freestanding rehabilitation center, but it must be provided! Although it is not the purpose of this chapter to address all of the deficiencies of the health care system, it is important that trauma system planners understand this deficiency and establish processes that provide immediate involvement of rehabilitation experts as well as case managers to optimize timing and effect of patient rehabilitation. From another perspective, such service will limit excessive acute care hospitalization necessitated by lack of rehabilitation access. This latter phenomenon is detrimental to optimal recovery, adds additional financial burden to the trauma center, and denies acute care beds to other injury victims.

Psychological Services

In addition to the enormous physical burden of injury, the emotional toll can be even more debilitating. Many injured patients have preexisting mental illness, and the burden of injury adds to this toll. Inclusion of psychological services to a trauma program can help injured patients and their families manage these postinjury mental health issues in an attempt to achieve the maximum recovery and reintegration. Depression and posttraumatic stress disorder (PTSD) are prevalent problems for injured patients, especially in the year following their injury.[39] To enhance recognition of this additional threat, a simple and quick screening method for identifying injured patients at risk of developing depression and PTSD has been developed.[40] The most recent edition of *Resources for Optimal Care of the Injured Patient* recommends that trauma centers should include an effective program to screen and manage these disorders as part of their complete rehabilitation program. Support groups may be a useful adjunct as well.

TRAUMA SYSTEM FUNCTION

Emergency Response and Trauma System Access

A critical function of the regional trauma system is to ensure that all injured patients within its geographic boundaries have access to definitive care to meet their injury needs. Access to the trauma system is dependent on the availability of prehospital transport services, the proximity and availability of definitive care resources, and the processes that direct the injured patient to definitive care. Ideally, the geographic distribution of trauma centers matches that of the population, and prehospital resources are organized to assure timely access to a trauma center regardless of distance. In 2005, it was estimated that 69.2% of US residents could access a major trauma center within 45 minutes and 84.1% could access one within 60 minutes of injury.[41] The remainder lived primarily in rural areas. States with the most urban populations had the highest proportions that could access a trauma center within an hour. More current data estimate that 63.1% of the US population can reach a major trauma center within an hour by ambulance, and the inclusion of helicopter services increased this proportion to 90.4%. The University of Pennsylvania, in consort with the American Trauma Society and with support of the US Department of Health and Human Services, manages a comprehensive website (https://www.amtrauma.org/page/FindTraumaCenter) that is periodically updated and provides a continuous overview of trauma system coverage in the United States.

Triage and Transport

Access to trauma care is not only dependent on the availability of EMS and hospital physical resources, but also the processes that govern the delivery of the patient to definitive care. Triage is the process by which injured patients are sorted to ensure that each has timely access to the appropriate level of care based on medical need and availability of resources. A critical function of the regional trauma system is to triage major trauma patients to major trauma centers. This often requires bypass of a closer hospital in favor of a major trauma center for major trauma patients. Triage should be selective so that medical resources are allocated to the patients who will realize the most benefit. Triage is a dynamic process that occurs continually at every phase of patient contact. Initial triage decisions are often revised as more information becomes available. Triage priorities may also be modified based on the balance between demand and availability of medical resources, as in mass casualty scenarios.

DEFINING THE MAJOR TRAUMA PATIENT

There is no standard that defines the major trauma patient. In the prehospital setting, clinical assessment and provider experience determine which patients should be transported directly to the trauma center. This assessment process evaluates physiologic metrics, mechanism of injury, and specific

patient factors. Additional factors may include injury pattern, need for specialized resources, or standardized local criteria. Part of the process of trauma system performance improvement includes periodic retrospective analysis of triage accuracy based on mortality prediction thresholds derived from discharge diagnoses and other data sources. The major trauma patient is defined differently along the continuum of care, which can make generalizations beyond a specific context problematic. What is apparent acutely in the field and what has been determined at discharge by addition of additional factors can be very different.

PREHOSPITAL IDENTIFICATION OF THE MAJOR TRAUMA PATIENT USING LIMITED INFORMATION

The challenge of trauma triage is to anticipate definitive care needs using limited information available at the time when triage decisions must be made. Of all trauma patients, only 7% to 15% have injuries that benefit from care at a major trauma center. Although clinical recognition of the major trauma patient is usually straightforward, serious, even life-threatening, injuries are sometimes occult and not discovered until after a comprehensive workup. Because mistriage is an inherent threat, triage must be viewed as a continuous, flexible, and error-tolerant process with contingencies for mistriage.

DEFINITIONS

Primary (Field) Triage. Primary or field triage directs transport from the scene to the highest level of care within a reasonable transport distance for major trauma patients. This usually enables rapid transport directly to major trauma centers in urban and suburban environments. Because the extent of injury is not always evident in the field, prehospital triage guidelines are established to identify patients at risk for severe injury and aid prehospital decision making. These tools are designed to be simple and applied broadly; the main purpose is to determine if the patient's estimated risk warrants transport to the nearest major trauma center rather than the nearest hospital. Risk of severe injury is estimated using information based on physical examination, mechanism characteristics, preinjury patient characteristics that are easily obtained by EMS on initial patient contact, or EMS provider judgment. Criteria for field triage are ordered with physiologic derangements indicating highest risk followed by anatomic findings, energy transfer mechanisms, preinjury patient comorbidities, and EMS provider judgment, in decreasing order.

BRIEF REVIEW OF METHODS PROPOSED FOR FIELD TRIAGE SCORING

To be useful in the field, a triage protocol must meet certain criteria.[42] The components of the scoring scheme must be credible and have some correlation with the injuries being encountered. The triage scoring method must correlate with outcome, although its primary purpose is identification of immediate patient risk. A better correlation with outcome results in a lower mistriage rate within a trauma care system. Outcomes for major trauma patients are usually classified as death, need for urgent/emergent surgical intervention, length of intensive care unit (ICU) and/or hospital stay, and major single-system or multisystem organ injuries.

The scoring scheme must also have interobserver and intraobserver reliability; that is, it should be able to be consistently applied between observers and by the same observer at another point in time with the same results, recognizing that estimation of patient risk may change as more information is obtained. Finally, the scoring scheme must be practical and easily applied to trauma victims for a variety of mechanisms by a variety of personnel without the need of specialized training or equipment.

Although most of the field triage criteria are based on physiologic criteria, there are other methods for assessing the risk potential of an injured patient. As shown earlier in the chapter, mechanism of injury, anatomic region and type of injury, preexisting illnesses, and paramedic judgment are important considerations in providing additional information in the field to help determine whether a patient requires transport to a designated trauma center. Combination field triage methods make use of this additional information by including it in the initial evaluation of the injured patient. As trauma systems have evolved, the determination of which variables are most effective in attaining the accuracy required for optimal system function has resulted in numerous proposed methods. Table 4-3 lists a brief history of some of these approaches. All were designed to assist rescue personnel in determining which patient required transport to a trauma center.[43-49] The Pediatric Trauma Score (PTS) is the only approach developed specifically for assessment of children.[50] As the work detailed in Table 4-3 developed, ACSCOT, in collaboration with the CDC, compiled all of this experience into a single process and developed the ACS Field Triage System. This is a more complete, advanced triage scoring scheme that is described in the *Resources for Optimal Care of the Injured Patient* and reflects years of productive collaboration with the CDC.

CURRENT TRIAGE RECOMMENDATIONS

The first ACSCOT field triage guidelines, "Field Triage Decision Scheme," was published in 1986 and updated in 1990, 1993, and 1999. In 2005, the CDC, with financial support from the NHTSA, collaborated with ACSCOT to convene a National Expert Panel on Field Triage to revise the decision scheme, which was published in 2006 by ACSCOT as part of *Resources for Optimal Care of the Injured Patient: 2006.* In 2009, the CDC published a detailed description of the scientific rationale for revising the field triage criteria R2t. The CDC reconvened the panel in 2011 to review the 2006 guidelines in the context of recently published literature, assess the experiences of states and local communities working to implement the guidelines, and recommend any modifications to these guidelines.[51]

As has been the case from the beginning, the intent of these triage guidelines is to assist prehospital care providers in identifying individual injured patients who would benefit from specialized trauma center resources. The process is

TABLE 4-3: Review of Proposed Triage Protocols

Method	History	Components	Status	Reference
Trauma index	First reported in 1971 by Kirkpatrick and Youmans	Blood pressure, respiratory status, central nervous system (CNS) status, anatomic region, and type of injury	Some correlation with injury severity Never saw widespread use	52, 89, 90
Glasgow Coma Scale (GCS)	Teasdale and Jennett first introduced	Eye opening, motor response, verbal response	Intended as a description of the functional status of the CNS, not as a prehospital assessment tool; motor component of the GCS is almost as good as the Trauma Score and better than the Injury Severity Score in predicting mortality	91, 92
Triage index, Trauma Score, Revised Trauma Score (RTS)	Described in 1981 as index SBP, respiratory effort added 1982	Respiratory rate,[a] respiratory effort, SBP,[a] capillary refill, GCS[a]	Central idea was that the leading causes of traumatic death were related to dysfunction of the cardiovascular, respiratory, and CNS systems Revised in 1989 because of concerns about accurate assessment of capillary refill and respiratory effort	93-98
CRAMS Scale	Proposed as a simplified method of field triage	Circulation, respirations, abdomen, motor, speech	Retrospective and prospective studies indicate that CRAMS triage is accurate in identifying major trauma victims with high specificity and sensitivity Easy to use	99
Prehospital Index (PHI)	Introduced in 1986 as field triage tool	Blood pressure, pulse, respiratory status, level of consciousness	PHI is accurate in predicting the need for lifesaving surgery within 4 hours and death within 72 hours following injury	100
Trauma Triage Rule	Proposed by Baxt et al, 1990	Blood pressure, GCS motor response, anatomic region, type of injury	Major trauma victim identification sensitivity and specificity of 92% Reduced overtriage while maintaining an acceptable undertriage rate Not widely adopted	101
Pediatric Trauma Score (PTS)	Introduced in 1985, designed to follow ATLS initial assessment scheme	Patient size, level of consciousness, airway patency, SBP, long bone fracture, open wound	Still in use throughout world, frequently as risk adjuster in outcomes research	102

[a]Components of final revision (RTS).

ATLS, Advanced Trauma Life Support; SBP, systolic blood pressure.

designed to guide assessment of an individual patient and is not intended as a triage tool to be used in a situation involving mass casualties or disaster. Based on the extensive history of critical thinking, evaluation, review, and revision detailed earlier, the current recommended process of assessment proceeds in four phases as listed in Fig. 4-4. The process begins with physiologic assessment for the obvious reasons; however, it is essential to remain aware of the common presentation of "found down," which may reflect an evolving medical emergency rather than the effect of acute injury.

Step 1: Physiologic Criteria. The first step is rapid identification of critically injured patients by assessing level of consciousness (Glasgow Coma Scale [GCS]) and measuring vital signs (systolic blood pressure [SBP] and respiratory rate). Vital sign criteria have been used since the 1987 version of the ACS Field Triage Decision Protocol. These criteria demonstrate high predictive value for severe injury. Of 289 references identified from the CDC panel's structured literature review, 82 (28%) were relevant to step 1. SBP less than 90 mm Hg and respiratory rate less than 10 or greater than 29 remain significant predictors of severe injury and the need for a high level of trauma care. Multiple peer-reviewed articles published since 2006 support this threshold.

Recommended criteria for transport to the highest level of care are:

- GCS less than or equal to 13
- SBP of less than 90 mm Hg
- Respiratory rate of less than 10 or greater than 29 breaths per minute (<20 in infants age <1 year) or need for ventilatory support

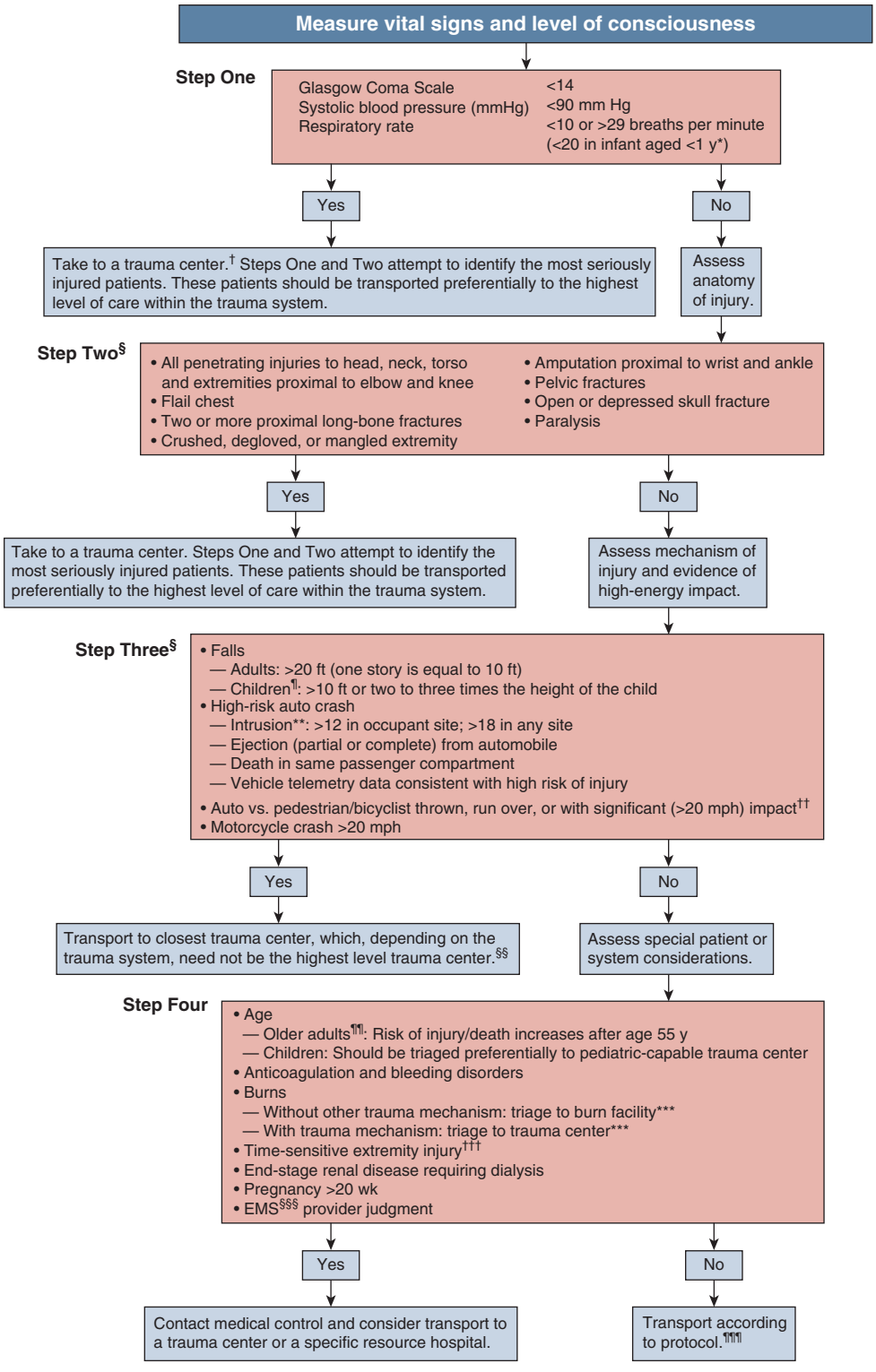

FIGURE 4-4 Centers for Disease Control and Prevention triage protocol. (Reproduced from Centers for Disease Control and Prevention. Guidelines for field triage of injured patients: recommendations of the National Expert Panel on Field Triage, 2011. *MMWR Recomm Rep*. 2012;61(RR-1):1-20. Adapted with permission from American College of Surgeons Committee on Trauma. *Resources for Optimal Care of the Injured Patient*. Chicago, IL: American College of Surgeons; 2006.)

*The upper limit of respiratory rate in infants is >29 breaths per minute to maintain a higher level of overtriage for infants.

†Trauma centers are designated Level I to IV, with Level I representing the highest level of trauma care available.

§Any injury noted in steps 2 and 3 triggers a "yes" response.

¶Age <15 years.

**Intrusion refers to interior compartment intrusion, as opposed to deformation, which refers to exterior damage.

††Includes pedestrians or bicyclists thrown or run over by a motor vehicle or those with estimated impact >20 mph with a motor vehicle.

§§Local or regional protocols should be used to determine the most appropriate level of trauma center; appropriate center need not be Level I.

¶¶Age >55 years.

***Patients with both burns and concomitant trauma for whom the burn injury poses the greatest risk for morbidity and mortality should be transferred to a burn center. If the nonburn trauma presents a greater immediate risk, the patient may be stabilized in a trauma center and then transferred to a burn center.

†††Injuries such as an open fracture or fracture with neurovascular compromise.

§§§Emergency medical services.

¶¶¶Patients who do not meet any of the triage criteria in steps 1 through 4 should be transported to the most appropriate medical facility as outlined in local EMS protocols.

Step 2: Anatomic Criteria. The second step considers that certain patients may initially manifest normal physiology but have an anatomic injury at risk of rapid deterioration and therefore may require the highest level of care. Of the 289 references reviewed by the panel, 57 (20%) were relevant to step 2.

Current recommendations for transport to a facility that provides the highest level of care include:

- All penetrating injuries to head, neck, torso, and extremities proximal to elbow or knee
- Chest wall instability or deformity (eg, flail chest)
- Two or more proximal long-bone fractures
- Crushed, degloved, mangled, or pulseless extremity
- Amputation proximal to wrist or ankle
- Pelvic fractures
- Open or depressed skull fractures
- Paralysis

Step 3: Mechanism of Injury. Step 3 addresses mechanism of injury (MOI) from the perspective of assessment of magnitude and vectors of force. An injured patient who does not meet step 1 or step 2 criteria should be evaluated in terms of MOI to determine the potential for severe but occult injury. Evaluation of MOI will help to determine if the patient should be transported to a trauma center.

Step 4: Special Considerations. In the fourth step, EMS personnel must determine whether persons who have not met physiologic, anatomic, or mechanism steps have underlying conditions or comorbid factors that place them at higher risk of injury or that aid in identifying the seriously injured patient. Persons who meet step 4 criteria might require trauma center care. A retrospective study of approximately 1 million trauma patients indicated that using physiologic (step 1) and anatomic (step 2) criteria alone for triage of patients resulted in a high degree of undertriage, implying that using special considerations for determining trauma center need helped reduce the problem of undertriage.[52] Among 89,441 injured patients evaluated by EMS providers at 6 sites, physiologic, anatomic, and MOI criteria identified 4049 (70.8%) patients with an Injury Severity Score greater than 15; step 4 of the guidelines identified another 956 seriously injured patients (16.7%), with an increase in overtriage from 25.3% to 37.3%.[53]

"INTERNAL" TRIAGE AND THE TRAUMA RESUSCITATION TEAM

The hospital response to prehospital notification is tiered according to the initially estimated need.[54] Highest risk patients require full trauma resuscitation team activation with all members present on or within 15 minutes of arrival. Lower risk patients may need a more limited resuscitation team activation, whereas other patients who are physiologically stable may be transported to trauma center and evaluated by an emergency medicine physician, with the resuscitation team or other surgical subspecialty services consulted as needed. The overarching factor governing this process is estimated injury need at the time of evaluation. However, it must be recognized that unnecessary mobilization of expensive resources robs them from other critical missions and is often wasteful.[53] Thus, the process of internal triage is predicated on how well a trauma center can balance its extensive and expensive resources against estimated patient need and logistical reality. The process may vary among centers and reflect unique capabilities or services available at different institutions. The most recent edition of the *Resources for Optimal Care of the Injured Patient: 2014* includes ACSCOT recommendations regarding major resuscitation criteria. Regardless of how the internal triage process is developed, it must be data driven, be continuously assessed in the program's Performance Improvement and Patient Safety program, and support the overall effectiveness of the trauma system.

SECONDARY (INTERFACILITY) TRIAGE

Secondary, or interfacility, triage directs transfer of patients whose needs exceed the capabilities of the initial receiving facility to a higher level for definitive care. This commonly occurs when patients who do not meet primary triage criteria are transported to a minor trauma center or community hospital and are subsequently found to have injuries that are beyond the capabilities of that facility.[55,56] In remote or rural environments, secondary triage serves to connect minor trauma centers to the major trauma centers after providing initial evaluation and stabilization of the major trauma patient. Like the field triage guidelines, interfacility (secondary) transfer guidelines are designed to identify patients at high risk of morbidity or mortality who might benefit from early transfer to a trauma center. Occasionally, patients who meet neither field triage nor secondary transfer guidelines are found to have injuries that exceed the capabilities of the initial treating facility. The Emergency Medical Treatment and Labor Act (EMTALA) intends that such patients have access to a higher level of care and ensures transport to the nearest trauma center with capacity and capability of managing their injuries.

MEASURING TRIAGE ACCURACY

The regional trauma system's ability to deliver the right patient to the right place at the right time and make the best use of available resources is expressed as triage accuracy.[57] Mistriage (overtriage and undertriage) occurs when a patient's triage decisions are not commensurate with the patient's clinical needs. Triage accuracy is dependent on compliance with established triage tools and the ability of those tools to predict the outcome of interest. The most recent edition of the *Resources for Optimal Care of the Injured Patient: 2014* recommends an overtriage rate of 30% to 40%, whereas the undertriage rate should be less than 5%. Despite these recommendations, there is still much work to be done in this area because several authors have demonstrated undertriage rates much higher than 5% and low sensitivity and specificity of existing triage protocols.[58,59] Because triage is a continuous, dynamic process, opportunities for mistriage can occur during any phase

of injury care. Field triage destination decisions are made using the best information available at the time. In-hospital triage and trauma resuscitation team activation decisions are made using the information provided from the field. Secondary triage and interfacility transfer decisions are made with more complete information but are influenced by the availability of resources at the referring facility. At any point, early decisions may appear to have been incorrect once more information is obtained. Consequently, retrospective evaluation of early decisions, using more information than was available at the time, introduces inherent methodologic mistriage. This error is worsened when real-time identification of a major trauma patient using field triage tools is evaluated using a different retrospective definition of a major trauma patient based on another system such as Injury Severity Score or a mortality prediction model. Thus, what looks like mistriage may, in part, be the combined effects of the retrospective evaluation of real-time decisions using disparate definitions.

Overtriage is a triage decision that incorrectly classifies a patient as needing a trauma center but retrospective analysis suggests that such care was not needed, and undertriage is a triage decision that classifies a patient as not needing trauma center when, in fact, the patient does need a trauma center. Although intuitive, operationalizing these definitions into objective quality metrics is problematic. There is no retrospective standard that defines which patients need trauma centers and which do not. The term *severe injury* is commonly used to refer to patients who need trauma centers and is often applied based on meeting an Injury Severity Score or mortality prediction threshold or consuming specific hospital resources such as operative or ICU care. This assumes that all patients and only patients who meet these definitions require trauma center care. In reality, there are many patients who do not meet such definitions but need major trauma center care, typically because definitive care resources may not be available in the community. Likewise, there are many patients who meet these definitions who may receive high-quality injury care at minor trauma centers or community hospitals.

Equivocation of terms when expressing over- or undertriage as rates also complicates trauma systems research. For example, the field undertriage rate could be expressed as the number of major trauma patients who should have received, but did not receive, the highest level of trauma team activation relative to the total number of major trauma patients. It could also be expressed relative to the total number of all injured patients. Each conveys important but different information; the former reflects the proportion of major trauma patients who were undertriaged, and the latter the proportion of all patients who were undertriaged. The same issues occur with equivocation of overtriage terms. The need to differentiate field triage, which reflects primary destination decisions, and system triage, which reflects the final patient distribution within the system, further complicates terms because over- and undertriage occur at both the field and system levels.

An approach to minimize equivocation of terms is to apply standard contingency table terminology to both field and system triage (Table 4-4). Given that triage accuracy is the number of patients appropriately triaged relative to the total number of patients, then inaccuracy or mistriage is the number of inappropriately triaged patients relative to the total. Because mistriage is a reflection of both over- and undertriage, then: 1 = accuracy + overtriage + undertriage. Here, accuracy, overtriage, and undertriage have precise meanings at both the field and system levels. Sensitivity, specificity, and positive and negative predictive values convey meaningful information at the field level since field triage is expected to predict high-risk patients. At the system level, it is descriptive but less predictive because the final distribution of patients depends not only on field triage risk assessment, but also the proximity of the patient population to the major trauma center, which also serves as the community hospital for a large portion of an urban population. Utilization refers to the proportions of low- and high-risk patients discharged from major trauma centers and other hospitals.

The study of triage accuracy is conceptually, linguistically, and technically complex. Field triage, secondary transfer, EMTALA regulations, and the role of major trauma centers as large community hospitals all influence access to resources in the regional trauma system. Application of system regulations and the hospital capabilities determine the final distribution of injured patients. Inherent methodologic errors introduced by retrospective evaluation of treatment decisions, equivocation of terms, and generalizations between phases of triage must be taken into account when making conclusions on overall triage performance and setting system triage benchmarks.

TABLE 4-4: Definition of Terms for Field and System Triage

Field/System		Risk: Low	Risk: High	Total
	–	A	B	A + B
	+	C	D	C + D
	Total	A + C	B + D	A + B + C + D
Sensitivity				D/B + D
Specificity				A/A + C
Positive predictive value				D/C + D
Negative predictive value				A/A + B
Accuracy				(A + D)/(A + B + C + D)
Mistriage				(B + C)/(A + B + C + D)
Overtriage				C/(A + B + C + D)
Undertriage				B/(A + B + C + D)
Over/under				C/B
LR utilization				C/(A + C)
HR utilization				D/(B + D)

HR utilization, major trauma center utilization by high-risk patients; over/under, overtriage to undertriage ratio; LR utilization, major trauma center utilization by low-risk patients.

DISASTER MANAGEMENT

Terrorism is the emerging weapon of modern civil strife. Terrorism events now occur almost weekly in various countries around the world and are usually designed to inflict as much damage as possible to innocent bystanders and then to strike again when rescuers arrive. Manmade events such as these and natural disasters such as Hurricane Katrina (New Orleans), Hurricane Harvey (Houston), Hurricane Maria (Puerto Rico), Superstorm Sandy (New Jersey and New York), and the endless stream of floods and devastating tornadoes that seem to increase in frequency with every passing year should crystallize the resolve of all medical personnel to become educated and proficient in disaster management. The approach to disasters, whether natural or manmade, requires a coordinated relief effort of EMS, hospital, fire, police, public works personnel, and often the military. This multiorganizational operation can effectively manage a crisis only if it is well directed and controlled. The ability to assess a disaster scene and mobilize appropriate personnel to provide damage control, fire management, rescue operations, and crowd control is dependent on an organization structure that permits dynamic information processing and decision making based on adequate planning and accurate vital scene information.

The military uses the concept of command and control for its combat operations. Key personnel continually monitor and manage the battlefield situation. The Fire Service of the US Department of Forestry, in 1970, adapted command and control into an incident command structure. Within this framework, a centralized group of disaster personnel commands and controls all of resources at the disaster site. Dynamic disaster scene information is processed at a predesignated incident command center where decisions regarding deployment and mission of rescue resources are implemented.

The incident command center structure is composed of seven key groups. If the disaster is small in scope, a single person may fill all seven areas. As the scale of the disaster increases, more personnel are required to perform these functions. The incident commander is responsible for the entire rescue or recovery operation. Under the direction of the incident commander are the seven group commanders: operations, logistics, planning, finance, safety, information, and liaison. Each of these section commanders has well-defined areas of authority and responsibility. Continuous on-scene information will be communicated to the command center. This will enable the incident command center to plan and direct the rescue or recovery operation. Thus, limited resources and key personnel will be directed to produce the greatest benefit.

The disaster scene is typically divided into zones of operation. Ground zero is the inner hazard zone where the fire and rescue operations occur. EMS and other nonessential personnel are kept out of this area. Rescued victims are brought out of this area to the EMS staging area. This is the second zone, a primary casualty receiving area, and it is here that EMS personnel perform triage and initial care for the patient. Disposition directly to the hospital may occur, or the patient may be sent to a distant receiving area for care and ultimate triage and transport.

The distant casualty receiving areas provide for additional safety in the environment. This downstream movement of injured patients prevents the primary triage sites from being overrun. Transportation of the wounded from the primary receiving site is reserved for the most seriously injured patients. Thus, a tiered triage approach is developed. A temporary morgue is also set up at a distant site.

Typically, groups of patients, the walking wounded, will migrate toward the nearest medical treatment facility. This process is called convergence. Medical facilities often set up a triage area in front of the emergency department to handle these patients. Current medical philosophy and federal regulations mandate an emergency assessment and treatment for stabilization of any patient who arrives at an institution's emergency department. In mass casualty situations, however, this can quickly overwhelm facility function and actually diminish effectiveness of care for all patients. Appropriate community disaster planning must recognize this potential problem and establish processes to direct certain groups of these patients to secondary medical facilities. The use of outpatient surgery centers and freestanding emergency medical centers, which are proliferating throughout the country, may be a valuable resource for this purpose. The final operational zone of the disaster site is the outer perimeter. Police permit only essential personnel access into the disaster site. Crowd and traffic control ensure the safety and security of the disaster scene as well as provide emergency vehicles rapid transit to and from the site.

Disasters may be of a small scale such as a building fire or explosion and may remain only a local or regional problem. As was demonstrated in the wake of the World Trade Center attack and Superstorm Sandy, the magnitude of a local disaster was of such proportions that activation of the National Disaster Medical System was necessary to address the rescue and recovery efforts. Analysis of more recent natural disasters demonstrates that approximately 10% to 15% of the survivors were seriously injured. The remaining victims either were dead or had mild to moderate injuries. Thus, overall effectiveness of disaster response is predicated on rapid sorting of survivors to determine the level of care needed by each patient.

The initial scene casualties from the World Trade Center attack were the result of planes striking the building. Fire and rescue personnel could not reach these patients. With the collapse of the first tower, rescue operations were aborted and attempts to evacuate rescue personnel became paramount.[60] After the building collapsed, victims injured in the street or from the surrounding buildings required medical treatment. As rescue operations resumed, injured rescue workers began to arrive at medical treatment facilities. Unfortunately, there were only 5 survivors of the Twin Tower collapse, with over 3000 fatalities, which included civilians and rescue personnel.

Israel's experience with terrorist attacks has demonstrated that rapid and accurate triage is critical to minimize mortality. Therefore, it has been suggested that the best triage officer, at least in bombings and shooting massacres, which are the most common form of terrorist violence, is the trauma surgeon.

This is important to guarantee that those in real need of immediate surgical attention are recognized and treated in a timely fashion without inundating the hospitals with patients who can be treated at a later time or those beyond salvage.

Many critical concepts have been learned from the Israeli experience. These include rapid abbreviated care, unidirectional flow of casualties, minimization of the use of diagnostic tests, and periodic relief of medical teams to maintain quality and effectiveness in care delivery. The concepts of damage control should be liberally applied in the operating room to free up resources for the next "wave" of injured individuals.[61-64] During mass casualty events, hospitals become overwhelmed very easily. Therefore, communication between hospitals and the incident commander is critical to distribute the casualties efficiently. All surgeons should be familiar with the basic principles of mass casualty management so that they can participate in the development of mass casualty management plans. Trauma surgeons should be the leaders in this field, since trauma systems serve as a template for the triage, evacuation, and treatment of mass casualty victims.[65]

APPLICATION OF TRIAGE PRINCIPLES FOR MULTIPLE PATIENT VICTIM EVENTS

Identification of major trauma patients in need of transport to a trauma center is the core mission of every triage protocol. In mass casualty events, however, a completely different process of patient assessment must be deployed. Triaging a single trauma victim is relatively straightforward, as described previously. For multiple casualty incidents, such as seen with multiple cars involved in a large-scale crash, the same essential principles apply; however, decisions must be made in the field as to which patients have priority. A multiple casualty incident can be defined as any situation where the volume of patients with injury severity may exceed hospital resources. Patients who are identified as major trauma victims by field triage criteria have priority over those who appear less injured. All major trauma patients should be transported to a trauma center as long as the trauma center has adequate resources to manage all the patients effectively. Because this situation can stress local resources, a properly conceived regional or state disaster management plan should include provision for possible diversion of the less critically injured to another trauma center or appropriately equipped hospital. Monitoring transports with online computer assistance allows for contemporaneous determination if one trauma center is overwhelmed.

MASS CASUALTIES

Triage in this situation is unique in that priorities are different from those in the single- or multiple-victim scenarios. As described earlier, in the instance of mass casualties, the resources of the designated trauma center, as well as the regional trauma system, are overwhelmed. When resources are inadequate to meet the needs of all the victims, priority shifts from providing care to those with the most urgent need to providing care to those with the highest probability of survival. A severely injured patient, who would consume a large amount of medical resources when not part of a mass casualty event, is now a lower triage priority. Despite the potential salvageability of this patient, the medical resources are focused on other patients who would benefit from advanced medical and surgical care. This method provides the greatest good for the greatest number of people. Field triage in this situation is probably the most difficult to perform as one has to make choices of quantity over quality with very limited amounts of information. These issues are further complicated when dealing with children.[66]

The most experienced and best-trained personnel available should make these field triage decisions. Physicians may be the best qualified to make these triage decisions; however, if they are the only clinicians available, direct patient care should take precedence and triage decisions should fall to other personnel. Patients are identified according to a triage code, based on the severity of injuries and likelihood of survival, and are treated accordingly. Occasionally, there may be an indication for a specialized surgical triage team with the capability to render acute lifesaving care of an injured, trapped patient.[67] In some disaster scenarios, moving intensive care capabilities into a disaster zone may be beneficial when evacuation of patients may be unrealistic due to logistical reasons.

In order to optimize patient care in these situations, it is important for regionalized systems to stage periodic mock disaster drills. These drills allow for the proper training of all individuals who might be involved as well as the identification and correction of potential problems. With increasing terrorist activity, specific triage algorithms have been developed for specific scenarios such as biologic, chemical, radiologic, or blast attacks.[68]

DISASTER TRIAGE: SIMPLE TRIAGE AND RAPID TREATMENT

In the event of a mass casualty or disaster, EMS personnel may use the Simple Triage and Rapid Treatment (START) triage system initially developed to be used in earthquakes in California. The object of this system is to triage large numbers of patients rapidly. It is relatively simple and can be used with limited training.[69] The focus of START is to evaluate four physiologic variables: the patient's ability to ambulate, respiratory function, systemic perfusion, and level of consciousness. It can be performed by lay and emergency personnel. Victims are usually divided into one of the four groups with color codes according to the timing of care delivery based on the clinical evaluation as follows: (1) green—minor injuries (walking wounded); (2) red—immediate; (3) yellow—delayed; and (4) black—unsalvageable or deceased.

If the patient is able to walk, he or she is classified as a delayed transport, but if not, ventilation is assessed. If the respiratory rate is greater than 30, the patient is an immediate transport. If the respiratory rate is less than 30, perfusion is assessed. A capillary refill time of greater than 2 seconds will mandate an immediate transport. If the capillary refill time is less than 2 seconds, the patient's level of consciousness

is assessed. If the patient cannot follow commands, he or she is immediately transported; otherwise, he or she is a delayed transport. Although capillary refill has been considered a reliable reflection of perfusion, in fact it can vary widely in different circumstances. This is especially true for children.[70] Therefore, findings from assessment of capillary refill should be verified by palpation of pulse. If the only palpable pulse is in the neck or groin, the patient should be transported. In light of the concerns about the predictive accuracy of capillary refill, some systems link the START method with severity scores, which may add unnecessary complexity and delay to the process of assessment: a Revised Trauma Score (RTS) of 3 to 10 is categorized as immediate, RTS of 10 to 11 is considered urgent, and RTS of 12 is categorized as delayed (nonurgent). This additional assessment requires providers to be familiar with the RTS and converts a binary finding to three options. Simplicity and expediency would dictate that central versus concomitant central and peripheral pulse palpation be the validator of capillary refill.

Mass casualty triage principles are the same for children and adults. However, because of differences in physiology, response to physiologic insult, ability to talk and walk, and anatomic characteristics, disaster triage in the pediatric age group is not as straightforward. Whenever possible, decisions regarding disposition of children should include consideration of availability of parental support.

A major benefit of the START system is accurate identification of severely injured trauma patients who may be able to be transported by air or ground ambulances to more distant trauma centers where the lower number of victims will assure that resources are available to provide optimal care.

MEASURING TRAUMA SYSTEM PERFORMANCE

Overview

As stated at the beginning of this chapter, trauma system sustainability is based on accountability. Accountability is phrased first in optimal outcome for every injury victim. Concurrent with this goal is confirmation of optimally achievable cost efficiency in delivering this care and ameliorating the financial burden of injury on the population. This process of accountability must also be able to support adequate analytics to define and deploy best practice. To achieve this goal, reliable system-wide data collection and analysis is an absolute necessity. Accurate, objective, and relevant data are the glue that keeps the trauma system functioning and the fuel that drives its continuous improvement.[32-35,71,72] A statewide trauma registry is necessary to assure commonality of critical data terminology; however, as the proliferation of electronic medical records continues to transform the clinical data ecosystem, adequate connectivity and interoperability of multiple data sources will be necessary to ensure that the trauma system is a self-learning, data-driven process of continuous quality improvement. Information from each phase of care is important and must be linked with every other phase. This level of data compatibility from different phases of care is especially important to determine the effects of certain interventions on long-term outcome. Constant system evaluation is necessary to identify where the system falls short operationally and stimulate strategies for improvements in system design.[73,74] This feedback mechanism must be part of the system plan for evaluation and must include designation of the agencies that will be responsible for data system management, definition of accountability metrics, and determination of best practice. Because each of these, and many subcomponents thereof, may fall within the purview of different stakeholder groups and may require special data security arrangements, this process should be defined as part of the regional trauma system plan. ASCOT has developed multiple programs and quality assessment processes that can guide this program as well as provide objective external review.[5]

Analysis of Trauma System Performance

The data on trauma system effectiveness published in the literature are difficult to interpret due to great variability in study design, type of analysis, and definition of outcome variables. Different study designs have been used to evaluate trauma system effectiveness. The most common scientific approaches include panel review of preventable death studies, trauma registry performance comparisons, and population-based studies. Panel review studies are conducted by experts who review trauma-related deaths to determine preventability. Well-defined criteria and standardized definitions regarding preventability have been used, but significant methodologic problems can lead to inconsistencies in the results and interpretation of the data.[26,33,75] The meta-analysis of trauma system assessment by Celso et al[76] provides an extensive review of various methods applied to the processes of assessment of the function and effect of trauma systems.

In an attempt to review the existing evidence on the effectiveness of trauma systems, the Oregon Health Sciences University, with support from the NHTSA and the National Center for Injury Prevention and Control of the CDC, organized the Academic Symposium to Evaluate Evidence Regarding the Efficacy of Trauma Systems, also known as the Skamania Symposium.[25] Trauma care providers, policymakers, administrators, and researchers reviewed and discussed the available literature in an attempt to determine the impact of trauma systems on quality of patient care. The available literature on trauma system effectiveness does not contain class I (prospective, randomized controlled trials) or class II studies (well-designed, prospective or retrospective controlled cohort studies, or case-controlled studies). There are several class III (panel studies, case series, or registry based) studies that were reviewed and discussed during the symposium. According to Mann et al,[31] review of the published literature in preparation for the Skamania Symposium supported the conclusion that the implementation of trauma systems decreases hospital mortality of severely injured patients. Independent of the methodology used (panel review, registry based, or population based) and despite the previously mentioned limitations

of each study design, a decrease in mortality of 15% to 20% has been shown with the implementation of trauma systems.[30,77] This has been most recently determined in analysis of the impact of the first 5 years of function of the Arkansas trauma system. Mortality decreased, as seen in other regions, and inpatient preventable mortality decreased significantly. The leaders of the Arkansas system are currently evaluating the cost savings generated by the system's ability to triage the most severe patient to the most appropriate center.[78] The participants of the Skamania Symposium also emphasized that not only mortality but also functional outcomes, financial outcomes, patient satisfaction, and cost-effectiveness should be evaluated in future prospective, well-controlled studies.[24,25,27]

Registry studies are frequently used to compare data from an individual trauma center, a trauma system against a national reference norm, between trauma centers within the same system, or at the same trauma center during different periods. The Major Trauma Outcome Study (MTOS) was among the first used as the national reference, although several of its limitations compromise the reliability of the comparison with data from other systems or centers.[79] Currently the National Trauma Data Bank and the more rigidly controlled data sets supporting the Trauma Quality Improvement Program (TQIP) are available for benchmark analysis in both adult and pediatric trauma centers. TQIP provides for risk-adjusted analysis of outcomes. In an effort to drive performance improvement across trauma systems, TQIP collaboratives of both hospital systems and states have been developed. Participation in TQIP collaboratives has been shown to improve system performance in states such as Michigan and Tennessee.[80-83] The advantages of registry-based studies include a detailed description of injury severity and physiologic data acquired by trained registrars. The disadvantages are related to inconsistency of data submission and the need to account for missing data using imputation and often propensity scoring in many multi-institutional analyses.

Population-based studies use information obtained from death certificates, hospital discharge claim data, or the Fatality Analysis Reporting System on all injured patients in a region. These methods of data collection and analysis are important to evaluate changes in outcome before and after or at different time periods following the implementation of trauma systems in a defined region. Because this is administrative and/or claims related information, there is little or no information on physiologic data, injury severity, and treatment. The limitations of the most commonly used databases in population-based studies are described in Table 4-5.

Despite differing approaches to study design, one area of rising interest has been comparison of outcomes of inclusive and exclusive systems. As described previously, in an inclusive system, care is provided to all injured patients and involves all acute care facilities, whereas in exclusive systems specialized trauma care is provided only in high-level trauma centers that deliver definitive care. In inclusive systems, patients may be transferred to a higher level of care (trauma center) after initial stabilization based on the availability of resources and expertise in the initial treating facility. Two associated variables can affect overall inclusive trauma system performance: (1) delay in transfer and (2) dilution of trauma centers' experience. Utter et al[38] have investigated whether mortality is lower in inclusive systems compared to exclusive systems. They concluded that severely injured patients are more likely to survive in states with the most inclusive trauma system, independent of the triage system in place. A possible explanation for these findings includes better initial care in referring hospitals.[38] A more recent study confirms a mortality reduction of 25% in patients under the age of 55 years.[28]

One of the benefits of continued analysis of trauma system function has been the identification of specific areas of injury where significant improvements in care and outcomes have emerged by evaluation of similar cohorts of patients across multiple institutions. Major advances have occurred in management of traumatic brain injury, including the benefit of high-volume experience in terms of patient survival and quality of recovery.[84] Continuous analysis of trauma center data aggregated at the state level affirms the importance of adequate clinical experience to optimize chances for good outcome. Racial and social disparity studies have defined specific

TABLE 4-5: Limitations of Current Trauma System Evaluation Studies

Panel studies
Inconsistent definition of preventability
Case mix of the population
Size, composition, and expertise of the panel
Process and criteria to determine preventability
Inconsistent report of prehospital and autopsy data
Registry-based studies
Missing or incomplete data sets
Coding inconsistencies and errors
Inconsistent report of autopsy data
MTOS limitations
Outdated data set
Data are not population based
Mostly blunt trauma
Differences in trauma centers' level of care
Inconsistencies in trauma registry inclusion criteria
Lack of data on comorbid factors
Lack of data on long-term follow-up
Population-based studies
Mechanism of injury and physiologic and anatomic data usually not available
Autopsies not performed consistently in all trauma deaths
Limited number of secondary diagnoses in claims data
Autopsy findings not always included in claims data
Hospital discharge data are inaccurate in transfers and deaths in the emergency department
Inconsistencies in obtaining AIS scores
Outcome measure is in-hospital mortality
No long-term or functional outcomes data available

AIS, Abbreviated Injury Scale; MTOS, Major Trauma Outcome Study.

issues that directly impact outcomes in these populations and, more importantly, document no discriminatory barrier to trauma system access.[85] Conversely, system assessment clearly underscores the importance of accurate triage and affirms that excessive overtriage makes treatment of minor injury inappropriately expensive and potentially disruptive of the process of care for more severely injured patients.

The greatest modern challenge for which careful system performance monitoring will be critical is management of the elderly. As the American population ages, the proportion of patients over 65 years who sustain significant injury continues to increase. These individuals represent a small volume of the overall trauma patient population, yet generate a disproportionate share of the cost of care.[85,86] In addition to disproportionate cost, multiple other factors influence how an effective trauma system must adjust to optimize care for the injured elderly. Despite being more active than their predecessors, most of these patients are afflicted with the usual comorbid conditions associated with aging. The most common MOI is falls, usually in the home environment. Next most frequent are motor vehicle crashes. The desire to be treated by the physicians or health system with which they are familiar often results in inappropriate triage. Inability to determine whether the patient is at risk because of being injured and elderly versus elderly and acutely injured often results in delayed management of injuries or incomplete assessment of deteriorating chronic comorbid conditions. Of even greater importance than simple survival is quality of life after recovery. Transformation of a reasonably healthy, active senior citizen to an impaired dependent is yet another burden of the disease of injury and demands careful planning, comprehensive case management, and compassionate counseling of victims and their families.

Practical Considerations in Trauma System Performance Monitoring

As is apparent in the review of the evolution of trauma systems, the major driver of this process has been commitment to eliminate preventable death. Because any injury represents a failure of prevention, the scope of effort has broadened to the concept of an inclusive trauma system that enhances prevention education and integrates all resources into a population-based, coordinated enterprise. The milestones of this evolution have been defined by a constant focus on clinical outcomes; what works, what doesn't, and what needs to be improved. This culture of outcome-oriented performance improvement has become the model for similar efforts across all aspects of American health care. The process of quality assessment is well defined elsewhere in this text; however, from a trauma system's standpoint, the following factors represent the core of what is required for accurate assessment and accountability. As the American health care system continues its transformation and as the clinical data ecosystem evolves to a level that supports the full gamut of health care delivery, system performance and clinical outcome measurement will drive better patient care, greater efficiency, and improved population wellness. The following items are some of the major factors that will be measured and will drive continuous quality improvement for the entire trauma system.

The three major domains of trauma system function are:

- Clinical effectiveness in reduction of the burden of injury
- Efficient use of resources
- Disaster response infrastructure

Clinical effectiveness is not simply stated in terms of decreased mortality. It must reflect continued focus on elimination of preventable death and optimization of potential recovery for all injury victims. This includes effective prevention of any injury, expeditious care for those at risk, and comprehensive long-term support through full reintegration into society. Trauma system function is predicated on the availability of extensive and expensive resources. These must be used in a cost-efficient and efficacious manner. Processes to measure this and provide objective accountability to society for its investment in citizens' safety is critical to assure sustainability of the trauma system. Just as the commitment to quality and optimal patient care forged by trauma systems has become a model for clinical care in general, so also has the trauma system collaborative infrastructure that coordinates availability and deployment of critical resources from disparate sources emerged as the infrastructure that will support effective disaster response.

Critical system factors necessary to address the earlier statements in a manner that defines trends and identifies improvement opportunities can be categorized as structure, process, and outcome elements according to the Donabedian conceptual model of performance assessment.[87] Table 4-6 identifies multiple aspects of trauma system function and theoretical metrics that could be developed to generate a data-driven, objective process for continuous performance improvement. The list is by no means complete and is provided as a starting point for thought and as a framework for transformation of relevant questions into objective milestones of success.

CONCLUSION

An effectively functioning trauma system is a physiologic, sociologic, financial, political, psychological, and cultural entity that is in continuous flux. It is an identifiable social commitment by a population to the welfare of all of its citizens. It is an investment of enormous expense in talent and treasure that must be continuously monitored to ensure full accountability for every aspect of its mission.[88] This chapter has reviewed the evolution of this fascinating and critical health care phenomenon and described the features that are necessary for implementation and deployment of an effective system. The two final characteristics that define every trauma system's pathway to survival and success are sustainability and agility. A trauma system will be sustainable if it continues to preserve lives and diminish the burden of injury in terms of cost, lost productivity, and avoidable misery. A trauma system's agility in managing all of its constituent components to anticipate and preemptively adjust to new challenges in

TABLE 4-6: Potential Metrics for Continuous Trauma System Performance Surveillance

Component	Relevance	Theoretical benchmark
Structure		
EMS coverage and access	Effectiveness is predicated on adequate population coverage and access	95% of citizens are within 30 minutes of transport
Distribution of EMS resources relative to DTCs	Optimize response time against out of service during active transport time	Predicated on incident response times
Estimated time to DTC (see https://www.amtrauma.org/page/FindTraumaCenter)	By any conveyance should be within 60 minutes	Running trend below 60 minutes
Number and distribution of trauma centers	Most efficacious and cost-effective mix of complex resources and appropriately staffed system entry points	No delays in access, no delays to definitive care related to resource availability
Occupancy	Adequate bed availability for every indicated level of care	No delays along the spectrum of care related to bed or provider availability
Diversion	Trauma service must be provided continuously, and when not, appropriate alternatives identified	Facility not available <2% of time
Post-acute care facilities occupancy	Immediate and continuous bed availability within 20 miles of trauma center	Proportion of bed days spent waiting for transfer should be <5% of total trauma bed days
Process		
Triage accuracy—overtriage and undertriage	Accurate risk recognition must drive correct field triage and expeditious transfer of severely injured patients from receiving facilities to the appropriate trauma center	Overtriage rate <15% Undertriage rate <5%
Cost-effectiveness	Avoidance of unnecessary service repetition and laboratory tests/imaging that increase radiation risk, prolong resuscitation, and often spawn unnecessary follow-up exams	100% documentation of need for imaging, laboratory, or clinical service Proportion of use and violation of clinical pathways
Outcome		
Mortality	Avoidance of preventable death	Application of standardized, evidence-based process for mortality prediction to identify preventable mortality
Morbidity	Elimination of care-related adverse events associated with long-term impairment; optimal care to ameliorate impact of acute injury on functional recovery	Incidence of adverse events and failure to rescue should approach zero
Disparities	No identifiable limitation to access along any aspect of the continuum of care	No delays or barriers to indicated therapy along continuum of care
Epidemiology as a measure of prevention effectiveness	Identification of most significant threats and determination of best practice for effective control	Decrease in incidence, severity and cost over time
Burden of cost	Per-capita cost of the disease of injury in terms of expended medical resources and lost human productivity	Population-based determination of decrease over time System response to spikes in incidence or severity of specific injury patterns, especially as related to violence-related mechanisms

DTC, designated trauma center; EMS, emergency medical services.

the American economy and health care system is essential for its sustainability. Many changes are fast approaching. The aging population is changing not only patient need, but also the epidemiology of injury. Violence remains a major problem in our culture; however, the impact of effective secondary prevention by better focus on environmental safety has diminished the incidence of devastating injuries related to high-energy transfer. Finally, our population continues to shift, and with it the burden of effective disease control can quickly exceed resources available. The tax base that shifts with this migration is often inadequate to support expansion of the infrastructure required to provide increased coverage. Thus, it is imperative that a trauma system be recognized as a living human endeavor under constant scrutiny to assure

optimal return on investment by continuously responding to its population's needs. It is the quintessential example of a self-learning health system.

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5

Injury Severity Scoring, Modeling, and Outcomes Research

Lynne Moore • Howard R. Champion

KEY POINTS

- Collecting reliable, accurate, and timely data on injury care is central to injury research and surveillance.
- Injury severity score (ISS) selection should be based on a clear sense of what one wants to measure and why and on a good understand of the score's strengths and limitations.
- Combination injury severity models attempt to combine the three concepts of risk: (1) preinjury physiologic reserve (eg, age, comorbidities); (2) physiologic status of the injured patient (eg, Glasgow Coma Scale [GCS]); and (3) anatomic injury severity (eg, ISS). The optimal injury severity model will depend on the data available, the study population, the exposure of interest, and, in particular, the outcome under evaluation.
- Injury outcomes research aims to improve our understanding of the determinants of optimal injury outcomes with the ultimate goal of reducing the societal burden of injury. Scientific ethics require a demonstration that addressing the research question will sigadvance current knowledge.
- Researchers are increasingly looking to assess outcomes that are important to patients. For hemorrhagic shock, that may be mortality, but for brain, spinal cord, and orthopedic injuries, function in daily activities and quality of life are more likely to be meaningful.
- Outcomes research must be based on a comprehensive, integrated, and end-of-grant knowledge-translation strategy. Practitioners, policymakers, decision makers, and patient/family advocates should be involved in all phases of research projects, and results should be distributed to all stakeholders, not just in the form of scientific articles but as policy briefs, clinical guides, and decision rules. We should also work to strengthen international collaborations to pool resources, avoid duplicating research projects, and generate results with maximum impact.
- To improve our understanding of the complex associations underlying the burden of injury, we need to improve the quality and coverage of injury data; employ more sophisticated analytical methods; improve the knowledge translation-to-action cycle of research results; look toward comparing outcomes across health care systems in high-, middle-, and low-income countries; and adapt our methods to the changing demographics of trauma populations.

INTRODUCTION

Injuries have long been classified in terms of severity. The world's oldest known surgical document, the Edwin Smith Surgical Papyrus (ca. 17th century BC), classified 48 traumatic injuries from ancient Egyptian battlefields and construction sites as successfully treatable, possibly curable, or untreatable.[1] Such predictions about patient outcomes, and attempts to quantify the severity of injury, are today the realm of more than 50 published injury severity scores, scales, and models.

Injury severity scoring quantifies the risk of an outcome following trauma and provides metrics based on elements of clinical acumen and statistical and mathematical modeling to describe aspects of the patient condition after injury. The primary outcome of interest is commonly survival or a measure of morbidity (eg, complications) or resource use (eg, hospital or intensive care unit [ICU] length of stay [LOS]) but may be any primary end point of interest (eg, compliance to an evidence-based clinical practice).

The injury scores and models discussed here are generally applied using retrospectively collected data. They are therefore suitable for case mix adjustment or risk prediction in groups of patients but are not appropriate for individual patient predictions to guide specific clinical management decisions. They have a host of applications including quality improvements in

patient care, trauma systems and health care delivery, injury prevention initiatives, and epidemiologic studies of injury.

DEFINITIONS

This chapter provides a concise description of injury severity scoring and modeling, with particular emphasis on application to outcomes research and quality improvement. Because there can be confusion about the precise meaning of terminologies used in this process, a few definitions are warranted to distinguish the qualitative from the quantitative.

Scales: Scales are a graduated range of values forming a standard system for measuring or grading something. Examples include the Glasgow Coma Scale (GCS), which has three components (verbal, eye, and motor) ranging from 1 (no reaction) to 5 (normal reaction), and the Abbreviated Injury Scale (AIS), which ranks severity from 1 (minor) to 6 (virtually unsurvivable, Table 5-1). Scales should be as objective as possible, and their assignment should be supported by inter- and intrarater reliability studies.

Scores: Injury severity scores place numerical values on injury descriptions, thus enabling standardized communication about the injury state. They are often generated by a standardized mathematical treatment of scales (scaled scores). Examples include the GCS score, which is the sum of the three components of the scale (ranging from 3 to 15) and the Injury Severity Score, which is the sum of squares of the AIS severity digit in the three most severely injured body regions (ranging from 1 to 75).

Scales and scores can be:

- Nominal: The values are not numerical and have no order.
- Ordinal: The values are not numerical but have an order (eg, increasing or decreasing injury severity).
- Interval: The values are numerical, and the distance between the levels is consistent both within and across the system of scoring.

Models: Modeling is defined here as the use of scoring or scaling systems to create processes for determining probabilities of risk for specific outcomes (eg, mortality, morbidity). The process of modeling is a means of combining or integrating multiple attributes, for example, multiple injuries or anatomy and physiology. Examples include generalized linear modeling with probability and link functions adapted to the outcome of interest (eg, logistic or log-binomial models for mortality and log-linear or gamma models for LOS). These models are used to formulate coefficients of risk for specific elements within a scale or risk associated with group diagnosis. Alternatives include machine-learning algorithms such as Bayesian networks[2] and neural networks.[3]

Outcomes research: Outcomes research is defined as a method of creating "empirically verified information" to better understand how variables in the real-world setting (from injury to treatment) affect a wide range of outcome variables (from mortality to satisfaction with care).[4]

Risk adjustment: Risk or case-mix adjustment entails isolation of the effects to be studied from the effects of other factors that can influence the outcome. Risk adjustment is essential for appropriate outcomes analysis. In injury outcomes research, severity scores are essential for risk adjustment because they allow accurate comparisons among disparate patient populations with varied degrees of risk. The goal is to compare populations with similar degrees of injury so that modifiable risk factors (eg, time to treatment, injury prevention interventions, mitigation equipment) may be properly isolated to examine their relationship to particular outcomes. Risk adjustment might be as simple as defining classes of a variable to stratify risk groups or as complicated as using a risk adjustor in a multivariable regression model.[5]

TABLE 5-1: Abbreviated Injury Scale (AIS)

AIS severity	Ordinal description
1	Minor injury
2	Moderate injury
3	Serious injury
4	Severe injury
5	Critical injury
6	Virtually unsurvivable injury

INJURY DATA

Collecting reliable, accurate, and timely data on injury care is central to injury research and surveillance and is the key to reducing the societal burden of injuries. Ideally, routinely collected data would be linked throughout the care continuum from prehospital care through community care. However, in most health care systems, injury data sets are limited to trauma registries and hospital discharge (administrative) databases, both based on the hospital phase of care.

Trauma Registries

One of the earliest civilian trauma registries was the Illinois Trauma Registry, which was created to benchmark and track improvements in trauma center implementation in a small geographic area.[6] However, it was preceded by an "accidental" trauma registry comprising the Wound Data and Munitions Effectiveness Team (WDMET) data collected in Vietnam from 1967 to 1969. About 15 years later, the WDMET database was rescued from destruction by a young naval officer and has provided a basis for our understanding of combat injury and future combat trauma registries.[7] The next significant step was the Major Trauma Outcome Study (MTOS),[8] which collected data from more than 100 hospitals in North America and Canada and serves as a model for all trauma registries established since the mid-1990s around the world.

Trauma registries are designed to provide information for quality improvement activities within trauma systems. They generally contain data on patient demographics, injury circumstances, prehospital care and transport (in some cases), anatomic injury, physiologic measurements on arrival,

emergency department (ED) and in-hospital interventions, comorbidities, complications, outcomes, and patient destinations. The information is generally coded from patient charts by trained data abstractors, and injuries are usually described using the AIS lexicon.

Trauma registries can be limited to a single provider but are most useful for quality improvement when they contribute to regional, state, national, and multinational registries. Trauma registries with a national reach include the US National Trauma Data Bank (NTDB), run by the American College of Surgeons (ACS), the United Kingdom (UK) Trauma Audit and Research Network (TARN), the German Trauma Register Deutsche Gesellschaft für Unfallchirurgie, and more recently, the Australian Trauma Registry and the New Zealand Trauma Registry. Because of the circumstances in Israel and the integration of military and civilian medical systems, the Israeli Defense Forces and Israeli trauma centers have a database that captures both civilian and military injuries, including those resulting from terrorist incidents. Trauma registry inclusion and exclusion criteria vary, with some registries including all injury admissions and ED fatalities and others applying inclusion criteria based on injury severity, trauma team activation, or LOS.

The main objective of most trauma registries is quality assurance or benchmarking, whereby provider performance is monitored over time or compared to an internal or external standard to identify hospital outliers. Trauma registries are also used for resource allocation planning, injury prevention, and outcomes research, to confirm specific hypotheses but also to generate them through exploratory analyses.

The major advantage of trauma registries is the fact that they include detailed clinical data that is essential for accurate benchmarking and outcomes research. Disadvantages include the facts that they are based on retrospectively collected patient chart data subject to data quality issues, they only cover the index acute-care admission for injury, and they are not usually population based. Indeed, trauma registries are generally limited to patients treated in designated trauma centers, which can represent a small proportion of regional injury admissions, particularly in exclusive trauma systems. However, trauma registries based on mandatory data collection and/or inclusive trauma systems such as the Victorian State Trauma Registry, the TARN, the Pennsylvania Trauma Registry, the Korean Emergency Medical Services Treated Severe Trauma Registry, and the Quebec Trauma Registry generally offer high population coverage.[9] Nevertheless, most trauma registries exclude a high proportion of injury-related deaths (particularly prehospital deaths) and minor injuries treated in the ED and discharged home. In addition, to maintain a focus on severe injury, many specifically exclude groups of patients, such as those with isolated hip fractures. Another important limitation of trauma registries is the investment required in terms of efforts and resources. In 2004, the Victorian State Trauma Outcomes Registry and Monitoring Group estimated the cost of the Victorian State Trauma Registry at approximately AUD$100 (US$84) per patient.[10] Despite the fact that this represents a fraction of a trauma patient's health care costs, it is usually perceived as an onerous financial burden and is a major barrier to trauma registry implementation and maintenance. Finally, most trauma registries have no data on outcomes that are important to patients such as functional capacity and quality of life and do not collect data on outcomes beyond discharge. One of the most complete trauma registries in the world is the Victorian State Trauma Registry in Australia, which provides 3-, 6-, and 12-month follow-up data on functional status and quality of life for injury patients using an opt-out consent strategy that would not be possible in most jurisdictions.[11] In summary, trauma registries present an incomplete but deliberately focused picture of injury, which may or may not even capture all severely injured admitted patients in a given geographic area.

Other Injury Databases

The US Department of Transportation National Highway Traffic Safety Agency (NHTSA) maintains two massive databases on vehicular-related injury in the United States, which are discussed in further detail later in this chapter: the Fatality Analysis Reporting System (FARS) and the National Accident Sampling System (NASS).

Military Trauma Registries

Despite the fact that military campaigns have often resulted in voluminous "after action reports" describing the type and number of injuries, systematic data collection did not occur until the Vietnam WDMET database. Seven teams of researchers were sent to Vietnam by General Creighton Abrams, then Army Chief of Staff, to comprehensively collect data on injuries for the purpose of assessing the wounding capabilities of various weapons. This database of some 8900 (an approximate 5% sample) combat injured and killed provided a useful source of data to characterize the nature and severity of modern combat injury (largely performed by Dr. Ron Bellamy). Other combat trauma databases include a complete set of data from the UK Hostile Casualty Reporting System (predominantly Northern Ireland) and databases on British casualties from the Falkland and Kosovo conflicts. Russian casualties in Chechenia were also databased. In the late 1990s, the US Special Operations Command (SOCOM) initiated efforts to database SOCOM casualties. An effort was also made to translate the injuries in the WDMET database to modern taxonomies. As Operation Enduring Freedom (OEF; Afghanistan) and Operation Iraqi Freedom (OIF) developed, the effort to database combat injuries picked up. Formats were field tested in Afghanistan. Finally, five trauma registries were in development through the early 2000s. As a result of these efforts, essentially census data were obtained on all Americans with severe combat injuries or killed in OEF/OIF. Data were generally excluded for those who returned to duty within 3 days. There have been numerous important publications from these sources of data. In addition, the information on the injured and injuries has been linked to operational data, and these classified data sets have been used to inform not only military tactics, but also body armor and vehicle design.

Hospital Discharge Data

Hospital discharge data are routinely collected in most high-income countries to meet administrative and reimbursement needs. These data sets generally include patient demographics, diagnoses, comorbidities, surgeries, and complications. Injury diagnoses and mechanisms of injury are coded using the International Classification of Diseases (ICD) lexicon. Advantages of hospital discharge data include population coverage of all hospital admissions and the fact that patients can be tracked through the system via a unique identifier, enabling researchers to obtain information on transfers, readmissions, and pre- or postinjury use of health care services. Disadvantages include the lack of clinical information, the absence of injury severity scores, and the omission of ED fatalities. In addition, discharge data are designed for administrative purposes and may not include all injury diagnoses. Hospital discharge data sets are most useful when they can be linked to trauma registry data. Systems that enter shared data simultaneously into both databases are the most efficient.

Prehospital Data

A major challenge of data acquisition in trauma systems relates to prehospital clinical data. At least half of all trauma deaths are prehospital. Few trauma registries capture these data, which, because of the emergency nature of prehospital intervention, are particularly hard to acquire and accurately record. For example, most prehospital providers do not ascertain patients' GCS scores except by gestalt. Further, although they record interventions, abstracting and converting the information into a database is particularly challenging. This is an important hurdle because at least half of all civilian trauma deaths occur in the prehospital period. This percentage is greatly increased in low- and middle-income countries. Military data reveal that at least 90% of combat deaths occur prior to reaching a medical treatment facility.[12] The 75th Ranger Regiment instituted a process of complete capture of prehospital data for the decade they were involved in Afghanistan and Iraq.[13] This is a fine example of prehospital data collection that has influenced the process of care and the speed with which care is rendered and has led to a reduction in preventable deaths. The Department of Transportation NHTSA, FARS, and NASS collect and database extensive prehospital data.

Other Data Sources

Other useful sources of injury data include police reports, road traffic or workplace injury insurance data sets, rehabilitation facility databases, physician billing records, and community care databases. Unfortunately, there are considerable barriers to linking these data sources, such as lack of unique identifiers, data-protection acts, and lack of harmonization across data sets.[14]

The US Department of Transportation NHTSA houses two large injury databases related to vehicular crashes, based on linkage of multiple data sources. FARS is a near-census population of deaths from vehicular-related injury in the United States, which contains data from police reports, emergency medical services, and EDs. NASS is a sample of vehicle crash patient data obtained from prehospital and hospital sources throughout the United States. Both of these databases, although imperfect, have been populated for decades and are a valuable resource for tracking, among other information, prehospital and in-hospital deaths from vehicle crashes (Fig. 5-1).[15]

We must continue to work toward solutions for linking routinely collected data on injury care. This would enable us to build injury life cohorts with amazing potential to inform primary, secondary, and tertiary injury prevention initiatives to alleviate the human and financial burden of injury.

Data Quality

Trauma registries generally have rigorous data quality assurance programs to ensure the accuracy and completeness of data extraction and entry. Mechanisms include initial and ongoing abstractor training, query forums, built-in electronic data error filters, retrospective data cleaning, and re-abstraction of random samples of charts. However, the quality of information in the medical charts themselves remains an important problem. Examples of common data quality issues in trauma registries include underreporting of comorbidities and complications, underestimation of injuries and their severity for early deaths, and missing data on physiologic parameters such as the GCS and respiratory rate (RR). Many clinicians ignore these data quality issues and mistakenly believe that large numbers of patients in a data set ensure quality. However, whatever the sample size, systematic variations in data quality across providers can lead to

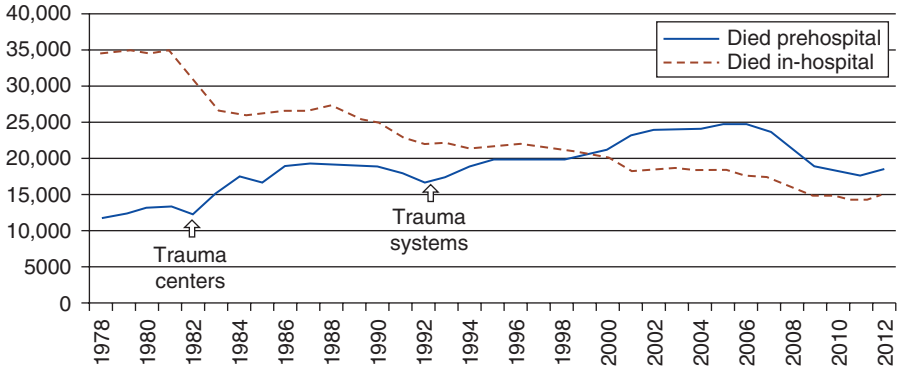

FIGURE 5-1 US vehicle crash deaths: 1978–2013.[15]

selection or information bias, whereby observed differences in performance across injury groups are due to differences in coding practices rather than quality of care. These data quality problems can be partly addressed in the analysis phase through sensitivity analyses and strategies to simulate missing data (eg, multiple imputation). However, analytical solutions will never replace high-quality data, and every effort should be made to ensure the accuracy and completeness of data collected in trauma registries.

INJURY SCORING AND MODELING

Trauma researchers must be familiar with injury scoring schemes to accurately risk-adjust injury group comparisons in an attempt to isolate the effects of an independent predictor variable on a dependent outcome variable. Four types of severity scores are typically used: (1) anatomic injury scores, (2) physiologic scores, (3) comorbidity scores, and (4) combinations of the three and other factors. The scores vary considerably in complexity of calculation and ease of use. Injury severity score selection should be based on a clear sense of what one wants to measure and why, and a good understanding of the score's strengths and limitations.

Anatomic Injury Scales, Scores, and Models

Anatomic injury scales, scores, and models describe the site of injury and extent of damage. Many anatomic scales, scores, and models have been proposed in the literature, but this review is limited to those that have gained practical acceptance. We will not review the huge number of specialty classification and grading systems such as the American Orthopaedic Foundation/Orthopaedic Trauma Association Classification of Fractures and Dislocations. Most general scoring algorithms are designed to predict mortality and are not specifically validated for other outcomes, such as resource utilization or disability, although moderate correlations may exist. The majority of scores are based on either the trauma-specific AIS coding classification or the more general ICD taxonomy.

ANATOMIC INJURY CLASSIFICATION SYSTEMS

Abbreviated Injury Scale. The AIS was first conceived as a system to define the type and severity of injuries arising from aircraft and motor vehicle crashes.[16] It began with a dictionary of 73 injuries in 1971 and now has close to 2000 injury codes, adding to its precision but also to the cost and complexity of coding.[16] Major revisions to the AIS occur infrequently; the latest revisions were in 1998 and 2005.[17,18] They aim to expand and create new codes, delete obsolete ones, and update severity scores in line with improvements in patient survival. Minor updates occur every few years with the latest ones in 2008 and 2015. To calculate AIS scores, medical records are transcribed into specific codes that capture individual injuries. The AIS is a proprietary classification system requiring specialized training for coding personnel. Because of this, AIS is not captured at every hospital.

AIS divides the body into nine regions: head, face, neck, thorax, abdomen, spine, upper extremities, lower extremities, and external. The AIS code consists of two numerical components separated by a decimal point. The first is a six-digit injury descriptor "pre-dot" code, which classifies the injury by region, type of anatomic structure and specific structure injured, and level of injury. The "post-dot" component is a severity score ranging from 1 (minor) to 6 (theoretically unsurvivable), as shown in Table 5-1. AIS severity scores are consensus-derived assessments assigned by a group of experts. The maximum AIS (MAIS), which is the highest AIS severity among all of a patient's injuries, is often used to quantify a patient's injury severity. This score is highly correlated with mortality but ignores information on concomitant injuries,[19] which may be significant in some patients and for specific outcomes.

AIS has many intrinsic shortcomings, including anatomically incorrect body regions, inability to code combat injuries,[20,21] and inconsistent scaling of severity across body regions.[22] For example, an AIS of 4 to the head and neck is associated with a higher probability of mortality than an AIS of 4 to the chest or abdomen.[23] Even when performed by experts, AIS coding has low interrater reliability, and 65% to 82% of AIS codes are not used, even in the largest registries.[24] Further, the limitations of the AIS are carried forward and multiplied when they are used as the basis for models (eg, the Injury Severity Score, discussed later).[25] A simplified version of the AIS has recently been developed for civilian injury with 450 codes, which greatly facilitates injury coding in resource-constrained environments.[26]

International Classification of Disease. Owned by the World Health Organization, the ICD is not injury specific, but is a general, all-purpose diagnosis taxonomy for all health conditions. It is well over a century old and is currently in its 11th clinical modification (ICD-11). In most countries, the 10th clinical modification, ICD-10-CM, has been used for nearly a decade.[27] In the United States, ICD-10-CM is in the process of being introduced.[28] In the ICD-10 lexicon, ICD codes exist for more than 68,000 medical conditions, and more than half of them describe physical injuries.[29,30] For trauma researchers, AIS codes are generally preferred over ICD codes because of their greater specificity of injury description (the pre-dot classification) and the availability of an injury severity classification (post-dot code).

ICD Injury Matrices. The Barell Injury Diagnosis Matrix (ICD-9) and the Injury Mortality Diagnosis Matrix (ICD-10) are matrices of ICD codes for classifying injury diagnosis by type and anatomic region into "injury profiles."[31] These matrices enable epidemiologic, management, and clinically oriented analysis by serving as a standard for case-mix comparison and characterization of injury patterns. The Barell Matrix consists of 12 columns based on ICD-9-CM sequence representing nature of injury (eg, fractures, amputations) and rows with varying levels of detail relating to body region (eg, head and neck, traumatic brain injury). Using this matrix, data can be aggregated into summary reports that indicate injury distribution, enabling descriptive comparisons.

ANATOMIC INJURY SCORING SYSTEM MODELS

Current anatomic injury models are based on the AIS or the ICD. The former has an injury scoring system derived by expert consensus (AIS injury severity score described earlier), whereas for the latter, empirically derived severity scores are available (probabilities of mortality calculated using large injury databases). AIS-based consensus models include the Injury Severity Score (ISS), the New Injury Severity Score (NISS),[32] and the Anatomic Profile (AP).[33] AIS-based empirical models include the Trauma Registry Abbreviated Injury Score (TRAIS) and the Trauma Mortality Prediction Model (TMPM). ICD-based models are all derived empirically and include the ICD Injury Severity Score (ICISS) and its derivatives.[34-37]

Injury Severity Score.

In 1974, Baker and colleagues[38] first posited a multi-injury score by introducing the ISS. Injuries in each AIS region are given an AIS score, and the highest AIS scores in the three most severely injured regions are squared and summed to form the ISS. The ISS range is an ascending scale of severity from 1 (least severe) to 75 (originally designated as unsurvivable). An AIS of 6 automatically translates to an ISS of 75.

ISS correlates with mortality, but the association is neither smooth nor monotonic.[39] Further, it only considers one injury in each body region and thus ignores important injury information, especially in penetrating and ballistic injury.[21,40] It also carries over the problem of nonuniform severity across body regions described earlier for the AIS. The ISS is probably most well known for its use in the definition of major trauma. Traditionally in the United States, an ISS of greater than 15 has been used to define major trauma. However, the major revision of the AIS in 2005 led to a reduction in severity for many injuries, in line with improved survival since the 1998 version. Consequently, it has been suggested that the definition of major trauma should be changed to an ISS of greater than 12.[41,42]

New Injury Severity Score.

The NISS was formulated to address ISS shortcomings in quantifying multiple occurrences of serious injuries within the same body region.[32] NISS is the sum of the squares of the three most severe AIS injuries, regardless of body region and, as with the ISS, an AIS of 6 automatically translates to an NISS of 75. This permutation offers a slight predictive advantage but has several of the same shortcomings as the ISS.

Anatomic Profile.

The AP, developed by Sacco and colleagues, uses AIS severity scores with an adjustment for differences across body regions.[21] Three modified components are weighted to form a single scalar based on anatomic location of all serious injuries (AIS >3). The AP overcomes many modeling defects inherent in ISS and NISS and avoids the excessive complexity and nontransportability in scores such as the TMPM.[33] The fact that the AP has not supplanted the ISS, however, is probably a reflection of the general lack of understanding of injury severity modeling in the injury research community.

ICD Injury Severity Score.

The ICISS took an empirical estimation approach to injury severity scoring with the formulation of ICD-9 survival probability (P_s).[34] P_s is an ICD code-specific estimate of the survival probability associated with that particular injury. The traditional ICISS is calculated as the product of P_s for as many as 10 injuries and ranges from 0 (unsurvivable) to 1 (null probability of death). Other versions of the ICISS include P_s of the worst injury and independent P_s calculated on patients with isolated injuries. A similar approach was used in the 1970s, with each ICD code being given a conditional and definitive P_s related to the presence of other injuries.[35]

The ICISS offers several advantages over other anatomic scores. First, because it is based on the ICD coding lexicon, it can be used in any clinical setting, including smaller centers that typically do not perform AIS coding. Second, unlike the consensus-derived AIS severity scores, the ICISS empirical approach enables powerful statistical estimates of injury-specific survival if the patient population used to derive them is large enough. Consequently, unlike the ISS and NISS, the ICISS is a smooth, if nonlinear, function of mortality.

The ICISS does, however, have limitations. First, although it resembles an overall probability, the ICISS can only be considered a scalar because P_s is often "contaminated" by patients with multiple injuries. Independent P_s can be calculated as mentioned earlier, but these are not available for all codes because many injuries rarely occur in isolation.[40] Second, P_s is database specific, and the degree to which it is applicable in injury populations with different patient characteristics remains uncertain.[41] Third, because empirically based injury severity scores are based on observed all-cause mortality, the ICISS is not an independent measure of anatomic injury but inherently incorporates physiologic reserve and physiologic reaction to injury, which vary across injury diagnoses. Finally, the ICISS has been validated for ICD-10 in a large aggregation of seven countries (Australia, Argentina, Austria, Canada, Denmark, New Zealand, and Sweden; 4 million observations) but will not be available for US data for several years.[43]

Trauma Registry Abbreviated Injury Score.

ICD codes are nominal, meaning they are unordered, qualitative categories not ranked by severity. If one ignores the AIS severity score, AIS codes can also be treated nominally, taking advantage of their specificity in injury classification. As such, AIS injury descriptor codes can be used to calculate P_s, similar to the ICISS. The TRAIS is the product of AIS-derived P_s. Kilgo and colleagues showed that ICISS and TRAIS behave similarly in a large group of patients coded both ways and that TRAIS predicts mortality more accurately than its AIS counterparts ISS, NISS, and AP.[44] Although ICISS and TRAIS are derived from two different coding systems, they behave similarly in terms of their association with mortality. This suggests that empirical approaches might obviate the inherent structure of the coding systems.

Trauma Mortality Prediction Model. The TMPM is also based on P_s associated with AIS codes, generated using NTDB data. It adds a level of complexity because it is calculated as a weighted sum of coefficients from two probit models of mortality: one based on AIS pre-dot codes and one on body region injured.

ICD-MAP. To address the lack of an injury severity scoring mechanism in the ICD classification system, MacKenzie and colleagues developed an algorithm to convert ICD-9-CM codes to AIS90 codes.[45] The algorithm has more recently been updated to map ICD-9 or ICD-10 codes to AIS05 (update 2008) codes.[46-48] Mapped AIS codes can be used to calculate AIS consensus-based severity scores including the ISS, NISS, and AP. This algorithm has proven useful for quantifying injury severity in administrative databases, but mapping is not achieved for all ICD codes, and the performance of mapped AIS injury severity scores for predicting mortality is significantly inferior to directly coded AIS severity scores and ICD-based scores (eg, ICISS).[19,49]

Trauma clinicians, outcomes researchers, and hospital administrators may ask which of these approaches is best. There is no consensus, and many publications every year continue to debate this question. Several large studies, including ones by Sacco[49] and colleagues and Meredith and colleagues,[19] compared these anatomic scores in terms of their ability to predict mortality. Both studies found that AP and ICISS better discriminate survivors from nonsurvivors than ISS, NISS, and the ICD-MAP versions of ISS, NISS, and AP. A surprising finding was that MAIS performed better than its multi-injury counterparts ISS and NISS. Based on this result, Kilgo and colleagues[44] showed that the patient's worst injury, regardless of coding lexicon (ICD-9 or AIS) or estimation approach (AIS severity consensus or empirical P_s), was a better predictor of mortality than multi-injury scores. Harwood and colleagues[50] reported that the NISS was better than the ISS and equivalent to the MAIS in the prediction of mortality in blunt trauma patients.

However, many of these studies have not accounted for other risk factors inherently incorporated into empirical scores (eg, age, comorbidities, physiologic reaction). Studies that do not account for these risk factors when comparing consensus-based and empirical scores unfairly disadvantage the former.[51] As mentioned earlier, through the large body of research on injury scoring systems, we have learned that each of these scores has strengths and limitations, and the choice of score should be adapted to the outcome, data, and study population at hand.

Physiologic Scoring Systems and Models

Anatomic injury unleashes a set of physiologic and biochemical consequences that require modulation and mitigation as part of the patient's treatment. The concept of integrating physiology into injury severity modeling recognizes the dynamic and time-dependent changes to physiologic status following injury. A patient with a ruptured spleen who is seen within 1 hour of injury might be normotensive and, when appropriately and promptly treated at a trauma center, will have a very low risk of dying. A patient with the identical anatomic injury who is seen 4 hours later, for whatever reason, may present with a systolic blood pressure (SBP) of 60 mm Hg and a significant risk of death, despite availability of the same system of in-hospital care. The hospital should not be penalized if the patient dies (but perhaps the trauma system should). Integration of physiologic parameters on arrival with other data is therefore essential for accurate case-mix adjustment and outcome prediction in injury research.

Early death following injury occurs as a result of central nervous system (CNS) impairment, hemorrhage, respiratory causes, or a combination of these. Clinical markers, including RR, SBP, base deficit, and reaction to stimuli/state of consciousness, are important prognosticators of outcome and are routinely used in clinical management. However, unlike anatomic injuries and preexisting comorbidities, which are fixed at the time of hospital admission, physiologic parameters are ever-changing, both spontaneously and in response to therapy. Thus, it is necessary to obtain a "snapshot" of physiologic status at one point in time, usually immediately upon ED or trauma center arrival. The main physiologic scores currently in use in injury research include the GCS and the Revised Trauma Score (RTS), which is a composite of the GCS, RR, and SBP.

GLASGOW COMA SCALE

The GCS was first proposed by Teasdale and Jennett as a means to directly triage brain-injured patients and to monitor postoperative craniotomy patients.[52,53] The GCS was subsequently integrated into the Trauma Score/RTS to describe level of consciousness without using subjective terms such as *semicomatose* and *lethargic* to classify head injury following trauma.[54] GCS measures brain function via three components: (1) motor (GCS-M), (2) verbal (GCS-V), and (3) eye opening (GCS-E), each with ordinal characterizations of severity (Table 5-2). The sum of scale components ranges

TABLE 5-2: Descriptors of Glasgow Coma Scale (GCS) Components

Function	Description	GCS scaled value
Eye	Spontaneous	4
	To voice	3
	To pain	2
	None	1
Verbal response	Oriented	5
	Confused	4
	Inappropriate	3
	Incomprehensible	2
	None	1
Motor response	Obeys commands	6
	Localizes pain	5
	Withdraw (pain)	4
	Flexion	3
	Extension (pain)	2
	None	1

from 3 (completely unresponsive) to 15 (fully conscious), and the GCS score has been shown to be strongly correlated with survival.[55] The motor component alone has been shown to be almost as powerful as the full GCS score for predicting mortality[56] and can be evaluated even when the patient is sedated and/or intubated. However, the verbal and eye components discriminate noncomatose patients and are thus valuable for predicting nonfatal outcomes.[55]

REVISED TRAUMA SCORE

The Trauma Score, later updated to the RTS, was designed by Champion and colleagues as an approach to combining clinical and observational physiologic data into one score.[57,58]

Two forms of the RTS exist, one for triage (Triage-RTS) and one for outcomes evaluation and risk adjustment (RTS). Both are based on the GCS, SBP, and RR (Table 5-3). The Triage-RTS is calculated by summing the coded values for each of the three variables and ranges between 0 and 12. The RTS equation for outcomes evaluation computes indexed values of GCS, SBP, and RR by weighting their coded value with logistic regression coefficients and summing them.

The RTS ranges from 0 to 7.84, with lower scores translating into more physiologic derangement. RTS correlates strongly with mortality[55] and remains important in injury scoring through its contribution to the Trauma Injury Severity Score (TRISS) model (discussed later in this chapter). Studies have also shown that the combined use of SBP and GCS-M is just as effective at predicting patient survival as the RTS.[59] Disadvantages of the RTS include the fact that coefficients are based on MTOS data and have not been updated and that categories of the GCS, SBP, and RR used to calculate the RTS often have very sparse data.[55] The 2009 version of the TRISS did not use the RTS but modeled the GCS, SBP, and RR separately.[60]

Comorbidity Scoring Systems

Injury outcomes research has long recognized the importance of comorbidities in outcome prediction models. For that reason, comorbidities were integrated into the ACS Committee on Trauma (ACSCOT) field triage decision scheme developed by Champion.[61] Morris and colleagues,[62] among others, identified several preexisting conditions that worsen prognosis following trauma, most notably liver cirrhosis, chronic obstructive pulmonary disease, congenital coagulopathy, diabetes, and congenital heart disease. Morbid obesity has now been added to this list.[63] The incorporation of preexisting conditions into injury severity models is difficult because so many potential comorbidities exist, each of which may occur with variable severity. Further, many are relatively rare and may be inconsistently recorded.

Specific comorbidity adjustments, such as the Charlson Comorbidity Index (CCI) or the Elixhauser Comorbidity Index, which are widely used in other disciplines,[64] are frequently used in injury severity models in an attempt to enhance their predictive abilities. Results, however, have been poor.[65,66] This may be because such scores are not adapted to acute injury populations.[66] Indeed, the CCI, a weighted sum of 17 preexisting conditions, is based on coefficients derived in a population of general admissions (mostly chronic diseases) using a Cox proportional hazards model in 1994 (updated in 2004)[67] and is therefore suboptimal for injury research. The number of Charlson comorbidities has been shown to predict injury mortality as well as the CCI.[66] The Elixhauser Comorbidity Index is based on 30 preexisting conditions, including hypertension, obesity, weight loss, and psychiatric disorders that are not included in the CCI. Other approaches include using the presence of individual comorbidities or classes of conditions (ICD ranges) or simply using patient age as a surrogate for comorbidities. Injury-specific comorbidity indices include the Mortality Risk for Trauma Comorbidity Index,[68] which achieves predictive accuracy for mortality equivalent to the CCI with just six items. With the aging of trauma populations, comorbidity and multimorbidity will increase, and accounting for these factors in injury research will become increasingly important. Further efforts should therefore be made to develop injury comorbidity indices for mortality but also for nonfatal outcomes, including complications, functional capacity, quality of life, and resource utilization.

TABLE 5-3: Revised Trauma Score (RTS)

Coded value	GCS	SBP (mm Hg)	RR (breaths/min)
0	3	0	0
1	4–5	1–49	1–5
2	6–8	50–75	6–9
3	9–12	76–89	>29
4	13–15	>89	10–29

GCS, Glasgow Coma Scale; RR, respiratory rate; SBP, systolic blood pressure.

Injury Outcome Scales

Outcomes following injury are widely measured in terms of mortality, complications, and resource utilization. However, there are several scales that can be used to measure functional outcomes and quality of life.

The Glasgow Outcome Scale is designed to provide an objective measure of recovery following brain injury.[69] The extended version ranges from 1 (death) to 8 (upper good recovery), and scores of 5 or more (lower moderate disability to upper good recovery) are considered to be consistent with a favorable outcome.

The Functional Independence Measure aims to assess functional status through the rehabilitation phase of care. It is a propriety scale that scores motor and cognitive function in 18 categories to assess independence in activities of daily living.[70]

Quality of life following injury in adults is often assessed with the Short Form Health Survey (SF-36 or SF-12)[71] or the EuroQol instrument EQ-5D.[72]

Combat Injury: A Special Case

Since the addition of descriptors for coding penetrating injuries with the AIS 1985 edition, researchers have had a tool for evaluating both blunt and penetrating injuries. The descriptors of penetrating injuries included in AIS versions since 1985 describe low-kinetic-energy injuries treated in civilian trauma centers and hospitals. Subsequent iterations have been used to code military combat injuries as well.[73] These codes, however, did not adequately describe commonly seen penetrating combat injuries such as multiple and massive soft tissue fragment wounds, high-velocity penetration, blast overpressure injuries (mutilating or nonmutilating), and/or bilateral and multiple injuries[20,21] that result from explosive devices, including improvised explosive devices, which account for 55% to 75% of combat injuries.[12] They also did not account for, nor were they designed to, some of the injury phenomena associated with mass casualty incidents, for example, crush injuries in earthquake disasters.

ABBREVIATED INJURY SCALE–MILITARY EDITION

To address these issues, a committee of military physicians was formed to work with the International Injury Scaling Committee of the Association for the Advancement of Automotive Medicine to propose guidelines for developing a version of the AIS specifically for coding combat injuries. These physicians represented all three services of the US military (Army, Navy/Marines, Air Force) as well as a spectrum of medical specialties relevant to combat casualty care including emergency medicine and trauma, orthopedic, neurosurgery, and general surgery. AIS 2005-Military is used for coding of all injuries in the three combat trauma registries: (1) the Department of Defense Trauma Registry (formerly the Joint Theatre Trauma Registry) based in San Antonio, Texas; (2) the Navy/Marine Combat Trauma Registry Expeditionary Medical Encounter Database based in San Diego, California; and (3) the Mortality Trauma Registry based at the Office of the Armed Forces Medical Examiner. These registries also code in AIS 2005 (civilian version) and AIS 1998 for future comparisons with civilian trauma registry data.

Development of AIS 2005-Military coincided with the revision efforts that would culminate in publication of the civilian AIS 2005, which included additional expanded descriptors for orthopedic trauma based on the Orthopaedic Trauma Association scale and expanded bilateral injury codes, particularly for vessel injuries. The same consensus model used in determining changes to each injury description by the International Injury Scaling Committee was used to determine AIS 2005-Military scores.

MILITARY COMBAT INJURY SCALE

Despite revisions that culminated in the AIS-Military, numerous combat injuries, such as those caused by explosive devices, still could not be coded or adequately described. Trying to adapt AIS was only moderately successful. Therefore, the Military Combat Injury Scale (MCIS)[24] was drafted by a large panel of military and civilian experts. First, a more anatomically correct and militarily relevant set of body regions was developed (head and neck, torso, arms, legs, multiple), five combat severity levels were determined (minor through likely lethal), and combat-relevant injury descriptions were tabulated.

Using these new body regions, severity levels, and injury descriptors, a five-digit MCIS coding scheme was developed, and 269 codes were assigned. Digit 1 indicates injury severity; digit 2 indicates body region; digit 3 indicates the type of tissue involved; and digits 4 and 5 together indicate the specific injury when combined with digits 1, 2, and 3. This coding scheme allows for injuries to the skull and brain to be identified separately from injuries to the face or neck and for injuries to the chest, abdomen, and pelvis to be separately identified despite being assigned to the same body region. It also allows for identification of unilateral or bilateral injuries, right or left for specific injuries, and easy identification of junctional area vascular injuries.

MILITARY FUNCTIONAL INCAPACITY SCALE

The Military Functional Incapacity Scale (MFIS) was developed at the request of military personnel to correlate immediate functional impairment with MCIS injury severity for ground troops and later for shipboard environments.[24] The ground operational requirement is based on the ability of an injured combatant to (1) communicate, (2) move, and (3) fire a weapon. The MFIS was developed as an ascending scale of functional impairment with four levels, as follows: (1) able to continue mission, (2) able to contribute to sustaining mission, (3) lost to mission, and (4) lost to military.

MFIS levels of incapacity were linked directly to MCIS injury severity. MCIS Severity 1 injuries are not associated with immediate functional incapacity, and casualties are able to continue with the mission; MCIS Severity 2 injuries usually result in immediate functional impairment with the potential for the casualty to contribute to the mission; and MCIS Severity 3, 4, or 5 injuries require medical treatment—casualties who sustain one or more of these injuries are lost to the mission or to the military. Specific Army and Navy scales have been developed.

Combination Injury Severity Models

Combination injury severity models attempt to combine some or all of the three concepts of risk described by MacKenzie[74]: (1) preinjury physiologic reserve (eg, age, comorbidities), (2) physiologic status of the injured patient (eg, GCS, RR, SBP), and (3) anatomic injury severity (eg, ISS, NISS, MAIS, ICISS).

Injury severity models are generally used to quantify patient case mix or to perform adjusted comparisons across injury groups. To date, most injury severity models have been based on mortality, but models based on nonfatal outcomes (eg, complications, readmissions, resource use) have more recently been proposed.[75-77] The most common use is probably for institutional benchmarking.

The most well-known injury severity model is the TRISS.[78] Documented limitations of TRISS include the fact that age is modeled in just two categories (equivalent mortality risk is assumed in all patients 55 years of age or older) and it does not account for comorbidities; in addition, it has other limitations inherent to the ISS and RTS (discussed earlier).[79] Further, the original coefficients are more than 20 years old, although they were updated in 2009 using NTDB data.[60]

In an attempt to address these limitations, many other injury mortality prediction models have been proposed. A Severity Characterization of Trauma (ASCOT) includes age modeled in five categories and uses the AP instead of the ISS.[80] The Harborview Assessment for Risk of Mortality (HARM) is based uniquely on hospital discharge data and includes anatomic injury descriptors (ICD), mechanism, comorbidities, and injury intent.[81] The Revised Injury Severity Classification (RISC) II model uses the AIS severity score of the two worst injuries, head injury, age, sex, pupil reactivity and size, preinjury health status, blood pressure, base deficit, coagulation, hemoglobin, and cardiopulmonary resuscitation.[26] The Trauma Risk Adjustment Model (TRAM) includes comorbidities and transfer status and employs flexible modeling techniques to use all information on continuous covariates (ie, age, GCS, RR, SBP) and to preserve their nonlinear associations with mortality.[82] Several studies have compared the predictive accuracy of injury severity models, and results indicate that the most complex models offer significantly better predictive accuracy and change the results of trauma center benchmarking analyses.[19,49,51] However, these more complex models have trouble supplanting the TRISS.

To address the problem of limited injury data available in low- and middle-income countries, the Kampala Trauma Score (KTS) has also been proposed. Originating in Uganda, the KTS is a "simplified composite of the RTS and the ISS, closely resembling TRISS,"[83] and is calculated along a descending scale of severity (ie, 5–10 = severe; 11–13 = moderate; and 14–16 = mild). Importantly, the KTS does not include anatomic injury description (AIS or ICD) because this would be unfeasible in a low-income setting.

Development of Injury Severity Models

Development of an injury severity model implies rigorous statistical methods in line with guidelines proposed for prediction models.[84,85] The purpose of the model must be clearly defined, the model should be derived on a large sample of (representative) patients subject to the highest standards of care, the choice of potential risk factors should be based on literature review and expert opinion in line with a conceptual model, and the model should allow for nonlinear associations with the outcome (eg, the probability of mortality does not increase linearly with age or the ISS, and associations with SBP and RR are nonmonotonic). The internal, temporal, and external validity of the model should be evaluated. Some injury severity models published to date have been evaluated in terms of apparent performance (discrimination and calibration on the sample used for derivation), but relatively few have been subject to rigorous internal and external validation.

Evaluating Predictive Models

The performance of models is evaluated according to their capacity to accurately predict the outcome of interest. Injury severity models based on binary outcomes (eg, mortality, readmission, complications) are generally based on the logistic regression model. The predictive accuracy of logistic models is evaluated by calculating measures of discrimination and calibration.

MODEL DISCRIMINATION

Model discrimination describes the accuracy of the model for distinguishing between survivors and nonsurvivors and is generally measured using the area under the receiver operating characteristic curve (AUC). This area varies between 0 and 1, where 0.5 indicates a model that discriminates no better than chance alone (noninformative) and 1 indicates a model that discriminates perfectly. Discrimination depends on the frequency of the outcome but, unlike calibration, tends to be relatively stable across similar populations. For example, injury severity models generally have excellent discrimination for predicting mortality (AUC >0.9)[82,86] and good discrimination for complications (AUC = 0.81),[77] but poor discrimination for unplanned readmission (AUC = 0.65).[75] This indicates that baseline risk (physiologic reserve, physiologic parameters on arrival, and anatomic injury severity) explains mortality well but that complications and unplanned readmissions are explained to a greater extent by other factors, such as quality of care. Discrimination is usually considered to be more important than calibration because it cannot generally be improved by modeling strategies.

MODEL CALIBRATION

Model calibration (or goodness of fit) indicates how well the model fits the data or how closely model risk estimates approximate observed event rates across different levels of risk. Good model calibration is dependent on the data at hand and can, to a large extent, be ensured by appropriate model specification, respecting clinically plausible associations between each independent variable and the outcome of interest.

Calibration is often quantified using the Hosmer-Lemeshow (HL) statistic,[87] based on the difference between observed and predicted probabilities of the outcome of interest in prespecified risk groups. The HL statistic has several limitations, including the fact that it is sensitive to sample size (a large, statistically significant value does not necessarily indicate poor model fit), is dependent on the risk groups used (deciles or other), and cannot be compared over different patient samples.[84,88-90]

Calibration should therefore also be evaluated using other strategies, the most useful of which is Cox's calibration curve. This curve is based on plotting predicted against observed probabilities of the outcome, thus providing a global

impression as to how the model fits the data, and enabling the analyst to identify areas where the fit is problematic. The intercept α and slope β of the calibration curve, which should be as close to $\alpha = 0$ and $\beta = 1$ as possible, are useful summary indicators of calibration.[91]

Models can also be evaluated in terms of explanatory power using, among others, *r*-squared adapted to binary outcomes, the Akaike information criterion,[92] and the Brier score.[93]

MODEL VALIDATION

Because the performance of predictive models tends to be overoptimistic in the sample used to derive them, predictive models should be validated in a sample of the study population from which they were derived (internal validation or temporal validation) and in a completely independent sample (external validation).

The internal validity of a model may be evaluated using split-sampling, cross-validation, or bootstrapping. In split-sampling, the model is derived on a random sample of the study population (eg, two-thirds), and it is validated by fitting the same model to remaining observations and calculating metrics of discrimination and calibration. In cross-validation, the sample is split in *k* samples of equal size. The model is repeatedly derived on one or several subsamples and its predictive accuracy evaluated on the remaining subsamples. In bootstrapping, the whole sample is used to derive the model, and it is validated on repeated random samples drawn from the original sample with replacement. The advantage of split sampling is that the validation sample is theoretically independent from the derivation sample (although in practice it has the same characteristics, as it is a random sample). However, bootstrapping has been found to be equivalent to split-sampling and is generally preferred because it uses all observations to derive the model, thereby increasing model precision.[94] The temporal validity of the model can then be evaluated by fitting the model to data collected in the same population at a different time. If the model has acceptable internal and temporal validity, model performance should then be evaluated on a completely independent sample (external validity).

SUMMARY

Current documented limitations do not invalidate the available injury severity models. Indeed, empirical validation studies provide strong evidence that all available models yield risk estimates of acceptable accuracy for groups of patients. The ongoing concerns are how to determine which model is best and how to improve available models. Several trends in recent modeling efforts provide initial answers to both questions. Models that reduce the weight given to secondary injuries relative to primary injuries,[95] incorporate interactions between injuries, and utilize better body region information are examples of promising directions for improving the accuracy of outcome predictions.[96-98] Multilevel modeling and methods that smooth the risk function (eg, spline regression, fractional polynomials) demonstrate directions for analytic refinement.[44,56,99,100] Data simulation techniques such as multiple imputation improve the feasibility of adding physiologic variables to the current anatomic/demographic models without excluding observations.[101] The growing access to extensive databases, improvements in analytic tools, and increased sophistication of substantive models lead to a straightforward conclusion: Today's models are good; tomorrow's will be better. However, analogous to severity scoring systems, the optimal injury severity model will depend on the data available, the study population, the exposure of interest, and, in particular, the outcome under evaluation.

INJURY OUTCOMES RESEARCH

Injury outcomes research aims to improve our understanding of the determinants of optimal injury outcomes with the ultimate goal of reducing the societal burden of injury. Patient outcomes were at one point focused solely on survival, but contemporary injury outcomes research has integrated nonfatal outcomes, including measures of morbidity and resource use. We are also gradually moving away from a predominant focus on intrahospital objective outcome measures toward patient-reported outcome measures,[102] such as health-related quality of life and function in daily activities, in line with patient-oriented research. These changes reflect a trauma community that has begun to embrace the World Health Organization's definition of health as a "state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity."[103] The objective of this section is to provide an overview of the basic elements of injury outcomes research.

Basics of Outcomes Research

A comprehensive programmatic approach is essential to answering the complex research questions that arise in injury research. Such an approach should include a systematic review of the literature with or without meta-analysis (scoping or rapid reviews may also be appropriate), analysis of retrospective cohort data, pilot data collection, and progression to a randomized controlled trial (RCT) or a well-designed prospective cohort study when an RCT is not possible. The first step in successful research is the rigorous elaboration of a strong research protocol in collaboration with methodologic experts and key stakeholders. There is an increasing trend toward registering and publishing study protocols.[104] One example of injury research that demonstrates the challenges of conducting studies in injury populations and has defeated many over the past decades is acute resuscitation. Significant challenges include obtaining sufficient sample sizes (participation from multiple institutions is often required), randomizing patients in the acute care phase, and defining appropriate end points. The insistence on 30-day end points by the US Food and Drug Administration when the vast majority of deaths occur within the first 3 to 12 hours following injury represents a significant barrier to interpretable research results and increases cost and risk of confounders.

According to Kane,[4] outcomes research comprises the following five essential steps:

1. Define a research question.
2. Develop a conceptual model.
3. Identify the critical dependent and independent variables.
4. Identify appropriate measures for each.
5. Develop an analysis plan.

RESEARCH QUESTION

Central to the success of a research project is the translation of a research question into carefully thought out research objectives and hypotheses. Rigorous research requires significant financial and human resources. Scientific ethics therefore require a demonstration that addressing the research question will significantly advance current knowledge. To do so, the researcher must be able to show, usually through a systematic literature review, that the question has not been adequately addressed in the past and that the research has the potential to have a positive direct or indirect impact on patient outcome. One important aspect in defining the research question is to establish whether the intention is to predict an outcome from a series of independent variables or to explain the association between one or more exposures and the outcome of interest. The injury severity models mentioned earlier (eg, TRISS, ASCOT) were derived to calculate predicted probabilities of mortality for benchmarking purposes, but injury outcomes research often aims to explain the association between an exposure variable (eg, intervention) and an outcome by estimating a measure of association (eg, odds ratio). Predictive and explicative research goals require very different analysis plans. The derivation and validation of predictive models has been covered earlier in this chapter. In this section, we will concentrate on explicative modeling of injury data in observational studies.

CONCEPTUAL MODEL

Each step of outcomes research is critical, although none more so than refining a research question through the elaboration of a conceptual model. Prior to building the conceptual model, investigators should carefully define the target population for the study, that is, the population to which they wish to generalize results. For example, in the study of the effect of surgical delay on complications for long-bone fracture fixation, the target population may be all patients 16 to 64 years of age admitted to a not-for-profit acute care center in a high-income country and requiring long-bone fracture fixation. The conceptual model should be elaborated as a causal diagram using directed acyclic graphs[105] (DAGs) and based on information from literature review and expert opinion. DAGs will provide the foundation for understanding how the association of interest between exposure variable(s) and outcome(s) is influenced by confounding, mediating, and moderating variables. In the example shown in Fig. 5-2, the association between trauma center care (exposure) and mortality (outcome) is of primary interest. Physiologic reserve, physiologic reaction, and anatomic injury severity are confounders, and interventions are mediators.

Study Sample. Prior to collecting information on the variables of interest, one must identify the study sample that will be used to address the research question. Careful choice of the study sample with clear inclusion and exclusion criteria in line with the target population is important for the external validity of a study. Ideally, the study population would be a random sample of the target population, but this is rarely possible. A convenience sample is therefore often used. For example, when studying the association between surgical delay for long-bone fracture fixation and complications, data availability may lead us to include only patients eligible for participation in the trauma registry when, ideally, we would include all admissions for long-bone fracture fixation within a health system. The consequences of the choice of study sample on the generalizability of results should be carefully thought out and discussed.

OUTCOME/DEPENDENT VARIABLES

The dependent variable represents the outcome of interest that we want to predict or explain with independent variables in the model under study. According to the quality of health care model proposed by Donabedian,[106] outcomes that should be evaluated to improve patient care include mortality, adverse events, unplanned readmission, resource use,

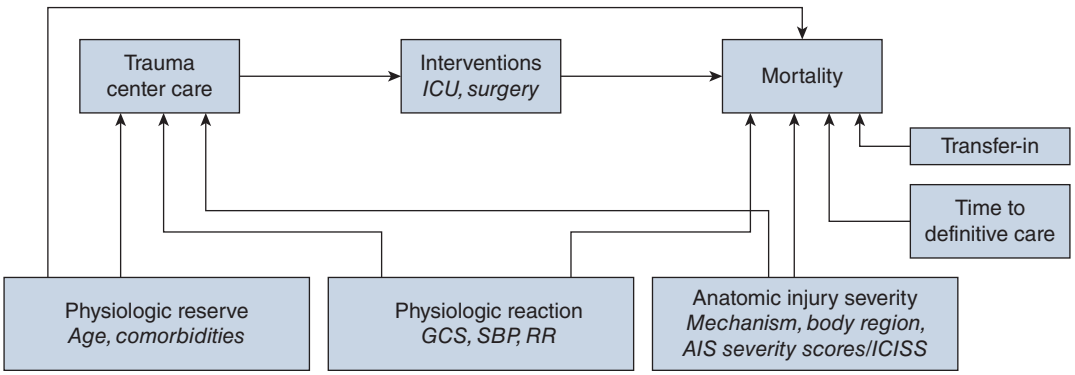

FIGURE 5-2 Causal diagram of the association between trauma center (vs nondesignated center) care and injury mortality. AIS, Abbreviated Injury Scale; GCS, Glasgow coma scale; ICISS, International Classification of Diseases Injury Severity Score; ICU, intensive care unit; RR, respiratory rate; SBP, systolic blood pressure.

quality of life, and ability to function in daily activities. A 2013 systematic review identified 14 nonfatal outcomes that have been used to evaluate injury care quality.[107] The most common were complications (35 of 40 studies) and hospital/ICU LOS (34 studies). Only three studies evaluated ability to function in daily activities, four evaluated unplanned readmission, and none used quality of life.

Outcome variables are frequently dichotomous in injury research because they represent the presence or absence of a health state (eg, mortality, complications). Depending on the design of the study, the outcome may be measured in terms of prevalence, incidence proportion, or incidence rate. For example, in a transversal study on community-acquired infection, the outcome may be measured as a prevalence (eg, presence or absence of infection on arrival). In a longitudinal study on hospital-acquired infection, the outcome may be measured as an incidence proportion or incidence rate (development of the infection over the hospital stay). In injury research based on trauma registry/hospital discharge data, retrospective cohort studies are common, and hospital outcomes are generally measured in terms of incidence proportions because events of interest often occur shortly after injury and time to event is of little interest. Incidence rates are common in chronic disease populations and may be more appropriate than incidence proportions for longer-term injury outcomes where loss to follow-up is an issue. Incidence proportions should be based on evaluation of outcome over a fixed period of time (eg, 30-day mortality), but information on postdischarge events is rarely available. Previous research has shown that hospital mortality, 30-day in-hospital mortality, and 30-day in-hospital and postdischarge mortality agree well, at least for patients less than 65 years of age.[108] However, the same is unlikely to be true for complications, which are a common reason for unplanned hospital readmission.[109]

The choice of outcome variable and the timing of outcome evaluation should be carefully adapted to the research question and target population. Hemorrhagic shock and/or brain injury are responsible for 90% of deaths following injury, and 80% of these deaths occur on the scene or in the hospital within 4 hours.[110,111] Therefore, as mentioned earlier, in resuscitation research, 4- or 24-hour mortality may be a more appropriate primary end point than 30-day mortality (Fig. 5-3).[112] The latter includes deaths due to comorbidities and late effects of injury (eg, complications), which may confound intervention evaluations. As mentioned earlier, the inclusion of prehospital deaths, which compose more than 50% of all injury deaths,[113] represents a significant advantage when systems of care or prehospital interventions are being evaluated.[114-116] Researchers are increasingly looking to assess outcomes that are important to patients.[117] For hemorrhagic shock, that may be mortality, but for brain, spinal cord, and orthopedic injuries, function in daily activities and quality of life are more likely to be meaningful.

INDEPENDENT VARIABLES

If the goal is to predict outcome, independent variables will be risk factors of the outcome, with consideration for the availability of information in populations intended for the prediction model. If the goal is to explain the association between an exposure(s) and outcome, independent variables will include the exposure variables of interest and covariates that influence the exposure(s)-outcome association(s).

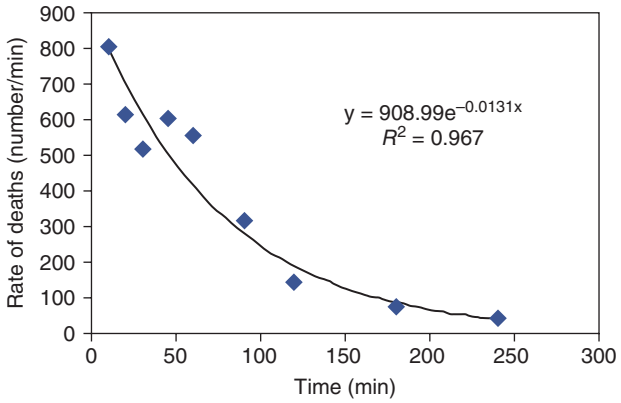

FIGURE 5-3 US vehicle-related deaths 5 minutes to 4 hours after injury, 2003–2005.[112]

COVARIATES

In an explicative design, covariates are independent variables that are known to influence the exposure-outcome association but whose relationship to the outcome is not of primary interest. They may be confounders, mediators, or modifiers and will be identified by the conceptual model (Fig. 5-2) and confirmed empirically during the analysis stage.

Confounders. Confounding variables are risk factors of the outcome that are also associated with the exposure (Fig. 5-2). Not adjusting for confounder variables in the statistical model will lead to a biased estimate of the exposure-outcome association. For example, if we evaluate the association between treatment in a designated trauma center (exposure) and mortality (outcome) and patients treated in a trauma center are sicker than patients treated in nondesignated hospitals, not accounting for injury severity would lead to an underestimation of the beneficial effect of trauma centers on injury mortality. The measure of association between trauma center designation and mortality (eg, odds ratios or relative risks) would thus suffer from confounding bias. In injury research, potential confounders are often identified among risk factors of the outcome that commonly cover physiologic reserve (eg, age, gender, comorbidities), physiologic reaction to injury (eg, GCS, SBP, RR), and anatomic injury severity (mechanism of injury, body regions injured, AIS- or ICD-based severity scores).[74] Randomized, double-blinded, controlled clinical trials should not suffer from confounding bias because risk factors are evenly distributed across exposure groups so the confounder-exposure association in the study sample is null.

Mediators. Mediator variables are caused by exposure and in turn cause the outcome. For example, in the association

between trauma center care and mortality, acute care (in-hospital) interventions would act as mediators. Some or all of the effect of the exposure on the outcome may pass by the mediator. In outcomes research, we usually aim to evaluate the total effect of exposure on outcome. It is therefore important not to adjust for mediator variables; otherwise, we will obtain an estimate of the direct effect only, which is an underestimation of the total effect. In the example, if we adjusted for in-hospital interventions, we would underestimate the effect of trauma center care on mortality. The distinction between mediator and confounding variables is critical to valid injury research.

Modifiers. Effect modifiers are variables that modify the association of interest. For example, we may expect trauma center care to reduce mortality for major trauma but to have no effect for minor trauma. If this is the case, injury severity would be said to modify the trauma center–mortality association. In outcomes research, potential effect modifiers are identified a priori, and usually, only one or two are specified because effect modification implies estimating the exposure-outcome association for each category of the modifying variable, thus increasing necessary sample size and the probability of a type I error due to multiple comparisons. Note that effect modification is not a bias but rather a natural phenomenon, which is present even under randomization. Global estimates of the exposure-outcome association are valid even under effect modification. Frequent effect modifiers in injury research are age (pediatric, adult, geriatric), injury severity, and type of injury (eg, TBI, blunt multisystem, penetrating). Stratifying analyses by injury type is particularly informative due to the heterogeneous nature of injury. For example, the pathophysiologic sequence to death in patients with isolated head injury is completely different from that for hemorrhagic shock; estimating a global exposure-mortality association for both patient populations may therefore be less meaningful.

ANALYSIS PLAN

Injury outcomes research is widely based on generalized linear models. The scale of measurement for the outcome variable will determine which model should be used. For outcomes measured on a continuous scale (eg, LOS, costs), a linear model will generally be appropriate. However, variables such as LOS or costs are right-skewed (many patients have an LOS <7 days and a few have LOS >90 days), so when sample sizes are small (ie, <100 observations per independent variable), log-linear models or gamma models may be more appropriate.[118] Sensitivity analyses should be conducted to evaluate the influence of outliers (eg, truncate LOS at the 95% percentile) and model specification on study results. Another issue with nonfatal outcomes such as LOS and costs is the competing risks of death, particularly in populations with high mortality (eg, severe TBI). This may need to be addressed with competitive risk models to avoid survival bias (discussed in the later section "Common Pitfalls and Mitigation Strategies in Injury Outcomes Research"). For ease of interpretation, results from simpler models should be preferred over those from more complex models when similar results are obtained.

For injury outcomes measured on a dichotomous scale (eg, mortality, readmission, complications), logistic or log-binomial models are commonly used. Logistic models are easier to use because convergence is rarely a problem (the logit of a proportion is not bounded, whereas the log of a proportion has a maximum of 0). However, odds ratios (logistic model) overestimate relative risks (log-binomial model), and this overestimation increases as the outcome becomes more common. For this reason, log-binomial models are generally preferred when the outcome occurs in more than 10% of patients.[119] The convergence problems encountered in log-binomial models can generally be overcome by using a Poisson model with robust variance estimates.[119] Finally, if long-term outcomes are of interest (eg, mortality up to 12 months after injury), incidence rates may be more appropriate than incidence proportions, in which case the Cox proportional hazards model is usually the model of choice.

Once the analysis model has been chosen, particular attention should be paid to the specification of independent variables. This should be based on a priori hypotheses about the functional relationship between each independent variable and the outcome using literature review and expert opinion. This stage is essential if we are to build a model with good face validity and to respect the postulate of a linear relationship between the independent and dependent variables, required for most parametric regression models. If this postulate is not respected for exposure variables, associations may be underestimated or missed, and for confounding variables, the exposure-outcome association may be subject to residual confounding, which can lead to over- or underestimation of the association(s) of interest. In injury research, most continuous independent variables do not have a linear relationship with mortality. For example, for general injury admissions, mortality risk is stable for patients 16 to 54 years of age and increases exponentially thereafter.[86] For SBP and RR, the change in mortality risk is not monotonic because risk increases both for patients who are hypotensive/hypoventilate and for those who are hypertensive/hyperventilate. These variables can therefore not be modeled using simple linear terms but should be specified using dummy variables on categories or more complex functions such as fractional polynomials or splines.[120]

All of these modeling strategies have advantages and disadvantages. Dummy variables on categories are easy to understand, operationalize, and present. However, disadvantages include difficulty identifying appropriate cut points, categories with small sample sizes, and nonintuitive step changes in risk. Fractional polynomials or splines offer more flexibility and therefore better model fit, but are complex and difficult to present. Simulations suggest that covariables modeled with dummy variables on at least four categories offer equivalent confounding control to fractional polynomials or splines.[121] However, the latter are interesting tools for graphically describing the functional relationship between the exposure variable(s) and the outcome. One often overlooked point is

that when scores based on probabilities (eg, ICISS) are modeled as independent variables in a logistic (log-binomial) model, they should be logit (log)-transformed to preserve the linearity assumption.[122]

As discussed earlier, an explanatory model contains exposure variable(s) and all potential confounders of the exposure-outcome association, commonly risk factors of the outcome. However, when sample size is an issue, a more parsimonious model may be required. This may involve a manual backward model selection approach, whereby potential confounders are removed from the model one at a time, starting with those that have the weakest association with the outcome (highest *P* value). Covariables are reintroduced into the model if their removal leads to a change of more than 10% in the exposure-outcome association estimate (eg, odds ratio, relative risk).[123]

MULTILEVEL MODELS

The generalized linear models described earlier are only valid under the assumption that outcome probabilities are independent. In many injury research contexts, this assumption is not respected. Common examples include study samples in which the same patient can be counted more than once for different injury events, samples that include several hospitals or trauma systems (cluster design), or studies in which the outcome variable is measured repeatedly over time on each patient (repeated measures design). If traditional methods are used to analyze such study samples, effect estimates (eg, odds ratio, relative risk) will be accurate, but variance estimates will generally be too low (increased chance of rejecting the null hypothesis when it is true).

The first example is not generally a problem in injury research because repeat visits for different injury events usually represent less than 10% of injury populations. Cluster samples are problematic, however, and generally require multilevel modeling techniques to correctly estimate variance.[109,124,125] Multilevel models are a simple extension of the traditional models described earlier, which allow for estimation of different intercepts and/or slopes for different clusters (eg, hospitals). These models can be useful for simultaneously modeling patient-level effects, such as age or injury severity, and hospital-level effects, such as volume or designation level. Generalized estimating equations are commonly used to account for intraindividual dependence in outcomes in repeated measures designs.[126]

OTHER STATISTICAL MODELS FOR INJURY RESEARCH

The traditional explicative models described earlier may not control well for confounding, especially with small sample sizes. Several new methods have been proposed to address this problem, including propensity scores and instrumental variables.

Propensity scores are obtained by modeling the exposure variable on all confounding variables and obtaining a score that is the probability of being exposed given confounders.[127] This score can then be used for matching, or simply included in the exposure-outcome model as a covariate to control for confounding. Propensity scores have been used to evaluate the hypothesis that intracranial pressure monitoring leads to a reduction in mortality in severe TBI patients[128] and to evaluate the advantage of helicopter transport over ground emergency medical services in the same patient population.[129] The propensity scores approach is based on the same confounder information as a traditional model so cannot theoretically reduce residual confounding. However, it does offer an advantage in terms of statistical power, particularly when the exposure is common and the outcome is rare.

Instrumental variables are intended to act as a proxy for unmeasured confounders.[130] Including them in the analysis model should therefore reduce residual confounding if they are chosen carefully. For example, Prada and colleagues[131] used the proportion of resident population served by helicopter ambulance services (at the state level) as an instrumental variable for evaluating the influence of Level I trauma center treatment on return to work, and Haas and colleagues[132] used county-level trauma center transport rate to evaluate the benefit of direct trauma center transport in a regional trauma system. However, choosing appropriate instrumental variables is not a simple task because they should be highly correlated with exposure but only associated with the outcome through the exposure variable.

The major problem with the methods described so far is that they are based on simple unidirectional associations with a single outcome. To improve our understanding of the influence of trauma systems and their components on multiple short- and long-term outcomes, more sophisticated statistical models are required. Examples of statistical methods that can better account for the multidimensional time-varying associations in injury research include marginal structural models,[133] structural equational models,[134] and microsimulation models.[135] These models are promising for gaining a better understanding of complex causal patterns and for evaluating the potential impact of modifying interventions on outcomes. However, all of these methods rely on the availability of high-quality, multidimensional data.

SAMPLE SIZE GUIDELINES

Sample size and power analysis can quickly become extremely complex with many hypothetical parameters to estimate, particularly for multivariable models and further still for multilevel models. However, accurate sample size calculations are essential to ensure studies have sufficient power to detect clinically meaningful associations. Several rules of thumb based on simulations are available to simplify the sample size estimation process. In general, for logistic, log-binomial, or Cox models, 10 outcome events per independent variable are necessary to obtain estimates with acceptable bias and precision.[136] For example, in a study sample of 1000 patients with 10% mortality, no more than 10 independent variables should be included in the model (dummy variables on categories count!). More recent research has suggested that five outcome events per independent variable are sufficient.[137] For

linear models, suggested sample size requirements are 10 to 25 observations per independent variable.[138] In injury outcomes research based on specific pathologies (eg, patients in hemorrhagic shock), studies based on single institutions will never reach sufficient statistical power to be meaningful and current. Collaborative multicenter research is thus essential to achieving sufficient sample size and representivity to confirm research hypotheses (explanatory analysis), rather than simply generate them (exploratory analyses). Researchers should also bear in mind that very large sample sizes like those seen in national or international trauma registries may lead to statistically significant associations that are not clinically important or even relevant.

COMMON PITFALLS AND MITIGATION STRATEGIES IN INJURY OUTCOMES RESEARCH

As in any research domain, injury research is vulnerable to bias, which should be considered in study design, analytical approaches, and interpretation of results. The following are some of the more common challenges of injury outcomes research:

- *Evaluating long-term outcomes*. Studies based on injury cohorts are often subject to high loss to follow-up over time as patients are more exposed to precarious living conditions than in chronic disease populations. They are also less likely to comply with treatment instructions or follow-up visits.
- *Obtaining patient consent*. Obtaining consent from the patient or family in a critical care situation is often very difficult. Ways to circumvent this problem include deferred and community consent.
- *Multiple admissions due to transfer*. Acute injury care often implies multiple hospital admissions due to transfer, particularly in an inclusive trauma system. Trauma registries and hospital-based cohort studies often fail to incorporate information on multiple admissions. This can lead to an underestimation of mortality and complication rates and resource use, including interventions. In many systems, linkage between registry- or hospital-specific databases and hospital administrative data enables investigators to track patients through multiple admissions.[139]
- *Survivor bias*. Injury research based on nonfatal outcomes is complicated by survivor bias.[140,141] For example, LOS is not fully observed (right censored) in fatalities. Therefore, if deaths are included in group comparisons of mean LOS, the latter will be underestimated in groups with higher mortality. If deaths are excluded from analyses, groups with lower mortality will have a high proportion of critically injured patients (those who did not die) and, therefore, higher mean LOS. In both scenarios, analysis will unfairly favor groups with higher mortality rates. Survivor bias may lead to biased measures of associations between interventions and outcomes, as fatalities may die before they can receive the intervention. It also leads to the detection of more injuries, comorbidities, and complications in survivors than in fatalities. Survival bias can be addressed with competitive risks models such as the subdistribution hazards model, introduced by Fine and Gray (more appropriate for predictive modeling), and the cause-specific hazards model (more appropriate for explicative modeling).[142] The former is mathematically equivalent to attributing the worse outcome (eg, longest LOS or highest cost) to deaths in a proportional hazards model. However, measures of association are difficult to interpret; the Fine and Gray model of LOS generates hazard ratios of hospital discharge.
- *Heterogeneity in injury coding*. The validity of intergroup comparisons in injury research is dependent on homogeneous injury coding across groups. Injury coding is particularly susceptible to heterogeneity across data collection sites, due to local coding conventions, and over time, due to changes in coding conventions. More obvious examples of the latter include changes in AIS versions; injury severity scores calculated with AIS 2008 are lower on average than injury scores calculated with AIS 1990, as discussed earlier.[143] AIS 2005/2008 to AIS 1990 conversions are available but are imprecise. In such a situation, analyses should be stratified by time period, and temporal comparisons should be conducted with caution. For comparisons between trauma centers or systems, analysts should be aware of intersite or intersystem differences in coding conventions and perform appropriate sensitivity analyses.
- *Missing data on physiologic parameters*. The GCS and RR are frequently used as covariates for risk adjustment in injury research. However, they are missing in 15% to 50% of patient files (and thus in trauma registries) due to prehospital sedation and/or intubation or lack of evaluation in patients with minor extracranial injury.[103,144] Excluding these variables from analysis may lead to confounding bias, whereas excluding patients with missing physiologic data may lead to selection bias and reduced statistical power. Possible solutions to this problem that have been proposed include using just the motor component of the GCS (can be evaluated in sedated/intubated patients) and simulating missing data using imputation techniques.[145] Perhaps the most promising technique proposed to date is multiple imputation (MI), whereby multiple plausible values are generated by simulation for each missing data value based on an imputation model that includes all independent and dependent variables to be used in the analysis model.[145] This technique takes account of the uncertainty surrounding the real data value in subsequent variance estimates. Much work has been done to evaluate the validity of MI for missing physiologic data in injury research, and simulations suggest that when the MI model is specified correctly, MI leads to valid effect estimates.[146,147] However, these solutions will never be as good as complete data and should not be used as a replacement for efforts to improve data quality.
- *Missing anatomic data*. Without autopsy records, anatomic data for early deaths are not fully available, and in many jurisdictions, autopsies are not frequently performed.[147] This results in an underestimation of injury severity in

early deaths, which leads to information bias in the evaluation of associations between exposure(s) and mortality.

SENSITIVITY ANALYSES

Since no observational study is exempt from selection, information, or confounding bias, sensitivity analyses should be used to evaluate the extent of the influence of these biases and any other analytical assumptions on study results. Robust sensitivity analyses plans should be developed a priori and integrated into the study protocol. For example, in evaluating the influence of trauma center care on mortality, sensitivity analyses may consist of (1) excluding deaths within 6 hours of arrival to account for underestimation of injury severity in early deaths and survival bias, (2) restricting analysis to patients age less than 55 years to assess the potential influence of underreporting comorbidities, (3) excluding deaths transferred in with GCS of 3 on arrival to address the problem of interfacility transfer for organ donation, (4) excluding patients age 85 or over to assess the potential influence of do-not-resuscitate orders, and (5) excluding patients transferred out to another acute care center to address varying rates of repatriation across hospitals. These sensitivity analyses will not provide definitive answers but will give an idea of the potential influence of bias on study results that can be used in formulating study conclusions.

BENCHMARKING

Comparing quality metrics within or across trauma centers or systems is central to the process of quality improvement. Benchmarking in injury care was initiated with the TRISS model, which has been used for decades to inform trauma care quality improvement activities worldwide. As mentioned earlier, despite its important contribution to trauma care quality, the TRISS has several documented limitations, including lack of adjustment for comorbidities and transfer status, consideration of age in just two categories, and the fact that performance statistics (W, Z, or standardized mortality ratios) cannot be used for interhospital comparisons.[148] Perhaps the most important limitation is the standard used for comparison. The MTOS cohort is now outdated, and the NTDB cohort used to generate the most recent published coefficients (2009) was based on voluntary participation with nonuniform inclusion criteria and nonstandardized coding practices. These limitations have been addressed in more recent benchmarking models.

Injury benchmarking has now moved beyond TRISS mortality calculations, and the injury research community has embraced the quality of care model introduced in 1966 by Donabedian.[106] Donabedian's model is based on the evaluation of structure, process, and outcome (Fig. 5-4),[149] where structure refers to physical characteristics of the health care provider (eg, presence of closed ICU), process refers to the use of evidence-based clinical interventions (eg, antibiotic prophylaxis for open fractures within 1 hour of presentation), and outcome refers to the result at the end of the care process (eg, mortality, morbidity, resource use). According to this model, improvements in structure lead to improvements in compliance with evidence-based processes, which in turn have a positive impact on patient outcomes.

Benchmarking under Donabedian's model implies the derivation and validation of a series of quality indicators (QIs) that cover the three quality domains. The American College of Physicians Performance Measurement Committee recently developed criteria to evaluate performance measures.[150] According to these criteria, QIs should be important (have a meaningful clinical impact), measure appropriate care (overuse, underuse, and appropriate timing), have a clinical evidence base, be clearly defined with good validity and reliability and appropriate risk adjustment, and be feasible and applicable. QIs should be developed in close collaboration with clinical and methodologic experts, knowledge users, and stakeholders according to the following steps: (1) identify structures, processes, and patient outcomes on which health care providers should be evaluated; (2) develop a risk-adjustment strategy, when appropriate; (3) establish a benchmark to which providers can be compared; and (4) evaluate the internal and external validity of QIs.

Step 1. The goal is to identify structures and processes of care that have a positive impact on patient outcome and measures of outcome that appropriately represent the burden of injury in terms of mortality, morbidity, and resource utilization. These should be supported by the best available evidence (identified using a systematic literature review) and expert opinion (expert consensus process involving clinicians, health care administrators, measurement experts, and patient-partners). This step is crucial if benchmarking results are to be used to improve care; previous research has provided evidence that there is little correlation between certain quality metrics and actual quality of care.[151]

Step 2. Structure or process QIs rarely require case-mix adjustment because they are based on the careful definition of providers/patients eligible for each structure/process (eg,

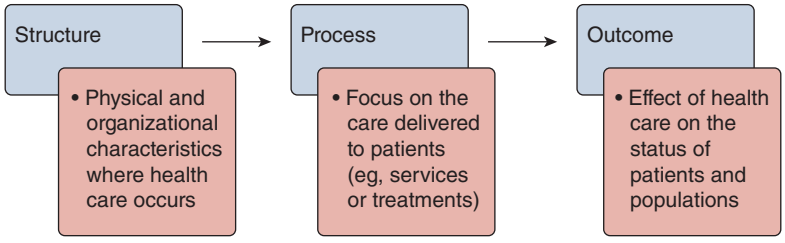

FIGURE 5-4 Donabedian's quality of care model.[149]

all Level I trauma centers should have closed ICUs, and all patients with GCS <9 on arrival should be intubated in the ED). However, appropriate risk adjustment is central to the process of benchmarking outcomes. Development of outcome QIs therefore implies the additional step of deriving and validating a risk adjustment model, as discussed earlier.

Step 3. The benchmark can be based on (1) a consensus-based standard (eg, all patients with major trauma should have an ED stay <1 hour); (2) an empirical, internal standard (eg, global mean in the system under evaluation); or (3) an empirical, external standard (global mean in an external population, such as the NTDB used for the TRISS model). The external standard has the potential to provide valuable information for quality control programs but should be based on an injury population with similar characteristics, subject to exemplary quality of care and high-quality data.

Step 4. The evaluation of QI validity implies providing evidence of face validity, content validity, criterion validity, discriminant validity, and predictive validity (Table 5-4).[152]

The selection of QIs through literature review and expert consensus should ensure both face and content validity. Criterion validity can be evaluated by assessing the correlation between QIs measuring the same quality construct (structure, process, outcome). Discriminant validity can be evaluated by assessing whether a QI detects statistically significant variation in performance across providers. Finally, predictive validity can be evaluated by assessing the correlation between structure-process QIs and process-outcome QIs (under Donabedian's model, structure influences process, which, in turn, influences outcome). Predictive validity should also be verified by assessing forecasting properties (ie, care provider results on QIs in one time period correlated with those in a following period). Once the internal validity of QIs has been demonstrated using all these metrics, their external validity should be evaluated in a new, independent patient sample.

TABLE 5-4: Measures of Validity[152]

Type of validity	Definition	How to measure
Face	On the face of it, do the metrics seem to be credible measures of the construct in question?	Expert consensus
Content	Does the metric evaluate the appropriate content and breadth of content?	Literature review and clinical peer review
Criterion	How well do the test scores reflect performance as measured by other accepted or widely used measures of the same concept?	Correlation with other quality indicators
Discriminant	To what extent do the inferences from a test's score accurately reflect the construct the test claimed to measure?	Discrimination between providers
Predictive	How well do the test scores predict the properties that they are designed to measure?	Correlation with outcomes
External	To what extent can the results of a research study be generalized to individuals and situations beyond those involved?	Validity in a completely independent sample

Several research groups have proposed QIs to evaluate injury care. In 1993, the ACSCOT introduced 22 audit filters to evaluate trauma care in terms of clinical processes.[153] More recently, Stelfox and colleagues[154] performed a systematic review to identify evidence-based QIs followed by an international expert consensus study according to University of California at Los Angeles (UCLA)/RAND guidelines. They generated a list of 31 QIs that experts consider should be used to evaluate the quality of injury care.[154,155] Inspired by this work, Moore, Stelfox, and colleagues derived and validated a comprehensive tool to evaluate the quality of acute injury care. The tool includes (1) a composite QI for evaluating trauma center structure including elements of commitment, trauma program, and procedural protocols based on ACS verification criteria[156]; (2) 15 clinical process QIs as well as composite process QIs based on compliance with best practice guidelines[157,158]; and (3) outcome QIs to evaluate mortality, complications, unplanned readmission, and hospital LOS.[75,76,159] The face, content, criterion, discriminant, and predictive validities of these QIs have been demonstrated, but their external validity has yet to be evaluated. Investigators have provided evidence that their injury care quality evaluation tool reproduces the structure-process-outcome associations of Donabedian's conceptual model.[160] In a mature, inclusive trauma system, trauma centers with higher scores on the composite structure QI also had higher scores on the composite process QIs, and centers with high scores on the process QIs also had lower risk-adjusted incidence of mortality, complications and unplanned readmission, and mean LOS. In addition, a significant positive correlation was observed among all QIs on outcomes; hospitals with lower risk-adjusted mortality also tended to have lower rates of readmission and complications and lower mean LOS.

Perhaps the most widely used QIs are those proposed under the Trauma Quality Improvement Program (TQIP) model. Many trauma centers/systems have subscribed to the TQIP benchmarking system, a for-profit organization that receives and processes institutional data to produce benchmarking results for hospital mortality, LOS, major complications, and selected clinical processes against the national average using the NTDB.[161] TQIP is based on standardized inclusion criteria, rigorous initial and ongoing training of data coders at participating sites, and extensive data quality-control mechanisms. Collaboratives allowing comparisons within hospitals of the same region have been formed, and drill-down tools to help identify root causes of quality problems are available.

Details on how TQIP models were derived have been published,[162] but information on model coefficients and validation of risk adjustment models is currently not available.

QIs are largely disseminated as part of an audit-feedback intervention, whereby providers (usually hospitals) receive a quality report, identify root causes of any quality problems (by discussions with stakeholders or drilling down data), propose quality improvement strategies, and are reevaluated. An approach based on feedback reports, educational material, local quality improvement teams, and quality improvement plans is associated with higher success rates than other audit-feedback approaches.[163-165]

International Injury Care Comparison

Benchmarking within a trauma system is an essential part of quality improvement activities, but international comparisons across health care systems provide a much greater opportunity to improve injury care and reduce the global burden of injury. There is clear evidence that trauma systems improve mortality, morbidity, and functional outcome following injury,[140,166-168] but it is unclear which aspects of trauma systems lead to optimal patient outcomes.[169] Many countries have or are in the process of adopting a trauma system model, but trauma systems worldwide have very different structures and levels of integration. International comparisons of injury care and injury outcomes therefore provide a unique opportunity to identify which components of trauma systems improve patient outcomes and which offer no advantage. Gabbe and colleagues have made considerable progress in international comparisons of trauma systems by comparing mortality and functional injury outcomes in Victoria, Australia, to those in the United Kingdom[170] and Hong Kong.[171] In addition, using the Utstein template to develop a pan-European trauma registry, Ringdal and colleagues[172] now have tools to compare injury outcomes across Europe. However, to truly improve our understanding of how differences in structures and processes of care across systems influence patient outcomes, efforts should be made to build an international injury data set including high-, middle-, and low-income countries. We also need to develop a standardized methodology to benchmark injury structures, processes, and outcomes on an international scale. A group of international injury researchers is currently working toward this goal. The first steps are to conduct a systematic review of international injury comparisons, to compare population-based injury outcomes using World Health Organization data, and to establish a framework for an international trauma registry.[169]

Challenges of Benchmarking

Many health care administrators and policymakers are moving toward making providers accountable for their performance by publishing benchmarking results (eg, health care report cards) or linking them to financial incentives such as pay for performance. However, rather than stimulating performance improvement, research has shown that the threat of institutional stigmatization or losing resources often leads to data gaming (eg, overestimation of injury severity, underreporting of complications), refusal of high-risk patients, and concentration on quality elements that are measured to the detriment of those that are not.[164,173] In addition, systematic variation in results across providers may be due to provider-level quality of care that can be modified by the provider, but may also be due to system-level problems or poor data quality. Therefore, it has been suggested that benchmarking activities should be used on an informative basis to identify potential problems and implement solutions as part of a quality loop (plan-implement-evaluate).[174]

FUTURE DIRECTIONS

We have made giant steps in injury severity scoring and outcomes research over the past three decades. The MTOS of the late 1980s[8] was the first to compare institutional outcomes of trauma patients while controlling for case mix. This process is now instantiated in trauma systems throughout the world and, despite some hiccups, has resulted in the NTDB, which contains data on more than 7 million patients, and the ACS TQIP, which has more than 200 subscriber institutions. However, there are more challenges to be faced in the future to address the growing global burden of injury.[156] To improve our understanding of the complex associations underlying the burden of injury, we need to improve the quality and coverage of injury data; employ more sophisticated analytical methods; improve the knowledge translation-to-action cycle of research results; look toward comparing outcomes across health care systems in high-, middle-, and low-income countries; and adapt our methods to the changing demographics of trauma populations.

Injury research is currently based largely on the acute (hospital) phase of care in trauma centers. Research has shown that the introduction of trauma systems within a public health care model leads to better short-term patient outcomes. However, we need to improve our understanding of which components of trauma systems drive optimal patient outcomes. To do so, population-based data from all components of the care continuum including prevention, prehospital, emergency, acute care, rehabilitation, and community care are needed. Some of these data are already available in many systems, but data protection acts and lack of unique identifiers often prevent database merging. Fortunately, fast progress is being made in methodology related to the acquisition, linkage, and treatment of big data, which should lead to breakthroughs in injury data coverage in the near future.[175-177] The ultimate goal may be to build injury life cohorts, which would have amazing potential to improve our understanding of the short-, medium-, and long-term consequences of injury. The immediate consequences of injury are the tip of the iceberg. To advance knowledge on the significant long-term burden of injury, we need to obtain information on long-term outcomes including quality of life and functional status.

Obtaining high-quality, high-coverage injury data represents a much greater challenge than employing appropriate analytical techniques, yet injury outcomes research is fraught with inappropriate statistical models. Despite our preference for simpler statistical models, traditional regression analysis is unlikely to be sufficient to fully understand the complex nature of trauma systems. Therefore, future research needs to look toward more sophisticated analytical methods such as causal inference and microsimulation models to better understand the determinants of optimal injury outcomes. In addition, whereas data coverage and quality may be improved, no observational study is exempt from selection, information, and confounding bias. We thus need to develop methods to evaluate the influence of such biases on observational injury data.

Efforts should be made to improve translation of research results into quality-of-care improvements as part of the quality loop. This is the essence of the public health model. Outcomes research must therefore be based on a comprehensive, integrated, and end-of-grant knowledge-translation strategy.[178] Practitioners, policymakers, decision makers, and patient/family advocates should be involved in all phases of research projects, and results should be distributed to all stakeholders, not just in the form of scientific articles but as policy briefs, clinical guides, and decision rules. We should also work to strengthen international collaborations to pool resources, avoid duplicating research projects, and generate results with maximum impact.

Injury severity scoring and biometric modeling remain fertile research fields. These include biometric markers and models for head injury and impending shock states, as well as a variety of biochemical markers being considered to augment models for identifying at-risk patients, including lactate, bicarbonate, and tissue oxygenation. Ultimately, these processes of risk evaluation of trauma patients will improve the general strategy and link in with therapeutic innovations, which include improved resuscitation therapies and energy-directed cellular-level therapies of the future.

Measuring and comparing structures, processes, and outcomes across health care systems is key to identifying areas for improving injury outcomes on a global scale. Despite challenges in terms of obtaining comparable data, an international repository for injury data would have the potential to greatly improve our understanding of the drivers of optimal injury care.

Finally, the demographics of injuries are changing rapidly. Traditionally designed to manage major blunt multisystem trauma and penetrating wounds, trauma systems must now adapt to trauma populations dominated by low-kinetic, isolated injuries in the elderly. We will therefore need to work toward adapting injury severity scoring systems, outcome measurement, and analytical strategies to these changes.

Fundamental to the process of evaluating innovation and the inexorable progress toward improving quality of care is a firm foundation of understanding of the power and limitations of injury severity scoring systems and biological, biometric models and an ability to adapt them to changing realities.

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6

Coding and Billing

R. Lawrence Reed II

KEY POINTS

- Medicare (established in 1965) provides health care benefits to Social Security beneficiaries (ie, previously employed individuals over age 65).
- Medicare hospital payments are payments based on the patient's admitting diagnosis in diagnosis-related groups that may be affected by documented comorbidities and quality metrics.
- Medicare physician payments: For each *Current Procedural Terminology* (CPT) code, a previously determined assessment is made of the amount of physician work (~50%), practice expense (~40%), and malpractice costs (~10%) associated with that service or procedure. The amount of work is assessed in comparison to the work already assigned to other similar procedures.
- The Relative Value Scale Update Committee (RUC) is a committee of the American Medical Association that assesses physician work for procedures and services and provides recommended relative value unit (RVU) valuations to the Centers for Medicare and Medicaid Services (CMS). Although the RUC provides recommendations, CMS makes all final decisions about what the Medicare payments will be.
- Conversion factor = Dollars paid/RVU.
- Billing Concept 1: Identify why physician does something for a patient by using codes for diagnoses from the International Classification of Diseases, 10th edition, Clinical Modification.
- Billing Concept 2: Identify what the physician does for the patient by using codes for services or procedures from CPT (updated annually).
- The provision of critical care services is specifically *excluded* from a global surgical package in the setting of trauma and burns.

INTRODUCTION

Although most physicians know that payment for their services must usually be coded and billed for monetary reimbursement to occur, their traditional medical education and training have not explained how this actually is accomplished. And, although it is true that most physician practice groups have employed personnel trained in medical professional coding and billing, those personnel are completely dependent on the quality of physician documentation in the medical record. The rules for coding personnel usually dictate the specific requirements that must be in the medical record to enable them to code appropriately for any given service. Therefore, physicians who are knowledgeable regarding documentation requirements will make it easier for their coders to apply the correct codes and then see better reimbursement. The purpose of this chapter is to provide the information and guidance for trauma and acute care surgeons to optimize their professional reimbursement due to a better understanding of the documentation, coding, and billing rules.

HISTORICAL BACKGROUND

Prior to the 20th century, physicians in the United States were paid for their services directly by the patient. Because of new developments and discoveries, the ability to provide life-saving remedies became increasingly available but was accompanied by increasing costs. The concept of using an insurance model (designed to pay for catastrophic events that were unlikely to occur) first evolved in the private sector in the late 1920s.[1] Initially limited to few people, private health care insurance received a significant boost during World War II, when a restrictive wage freeze forced industries to compete with other options for the few able-bodied men who were present stateside. Since they were prohibited from

boosting wages, employers were forced to compete by offering employee health care as a benefit of their employment. Employer-based health care insurance ultimately became the norm in American society with few exceptions.

Upon retirement, however, such employer-based health care insurance was no longer available to retirees, who were now forced to pay for their increasingly expensive health care needs out of their fixed Social Security income. Throughout the 20th century, there was a progressive movement to socialize American medicine and provide health care benefits for all citizens. A major milestone in this effort was the implementation of Medicare in 1965, which provided health care benefits to Social Security beneficiaries (ie, previously employed individuals over the age of 65).

Initially, patients submitted health care claims to Medicare and then received payments from Medicare to turn over to their physicians. Because many patients did not transfer their claim payments to their physicians, however, Medicare began to offer physicians the option of accepting "assignment," meaning that physicians would accept payment directly from Medicare instead of directly from the patient. With this benefit came the requirement that the physician would agree to accept Medicare's allowed amount as payment in full. Ultimately, private insurers followed suit, with actual payments by patients becoming minimized to the level of deductibles and copays, which were designed to prevent excessive or unnecessary use of health care resources by patients. Current-day health savings accounts seek to provide patients with the same disincentives toward needless health care expenses while simultaneously providing them the potential for additional financial support during retirement.

MEDICARE ADOPTION OF THE RESOURCE-BASED RELATIVE VALUE SCALE

The modern era of payments to physicians began in the 1980s, when assessments of the financial health of the Medicare program indicated that it was on course to bankrupt the Social Security system. Consequently, cost-cutting measures were instituted over several years, primarily aimed at lessening payments to physicians and hospitals.

For hospital payments, the Medicare program switched from a fee-for-service system to a "prospective" payment system. Realistically, this system is only prospective in that the payment that is ultimately provided is based upon the standard payment for the patient's admitting diagnosis, grouped into diagnosis-related groups (DRGs). The payment to the hospital, however, is not actually made prospectively (ie, at the time of admission), but is only paid after the patient is discharged. This system had the potential to reduce unnecessary tests and treatments that would not be paid separately as had previously been the case.

On the physician side, the concept of a Resource-Based Relative Value Scale (RBRVS) was developed as a means to quantify physician services on a relatively objective basis.[2] The American Medical Association's *Current Procedural Terminology* (CPT), an annually produced catalog of services and procedures performed by physicians, served as the structural basis for the RBRVS. For each CPT code, a previously determined assessment is made of the amount of physician work, practice expense, and malpractice costs associated with that service or procedure. These assessments are summed for each CPT code, and the resulting totals are ranked in a standardized scale to produce relative value units (RVUs).

RELATIVE VALUE UNITS DEFINED

RVUs serve as the fundamental currency for physician services. The entire concept and structure for physician RVUs under Medicare is defined in the US Code of Federal Regulations.[3] Although the valuation of the amount of physician work, practice expense, and malpractice costs varies slightly for each CPT code, physician work valuation composes about 50% of the total RVU payment, with practice expense averaging roughly 40% and malpractice expense averaging about 10%.

Physician work, in turn, is determined based on assessments of the time spent, the technical skill and/or physical effort required, the amount of mental effort and judgment required, and the stress arising from any potential risk to the patient from performing the procedure or service. These assessments are developed by the Relative Value Scale Update Committee (RUC), which is composed of representatives from physician specialty societies and the American Medical Association. The RVU valuations are recommended to the Centers for Medicare and Medicaid Services (CMS), which in turn produces a quarterly update of RVU valuations on its website.[4]

Physician reimbursement is directly tied to RVUs through the *conversion factor*, which specifies the number of dollars paid for each RVU. Unfortunately for physicians, the conversion factor has not kept pace with inflation. In 1999, the conversion factor was $34.73/RVU, whereas in 2018, the conversion factor was $35.9996/RVU. If the conversion factor had kept pace with inflation, it would now be $48.93/RVU. Although the difference of $13.18 may seem trivial, a general surgeon with an average production of 7000 RVUs annually now only earns $250,250, instead of the $342,510 he or she would have earned if the conversion factor had kept pace with inflation, representing an annual loss of $92,260.

Payments to physicians are made for two types of care: services and procedures. *Services* represent patient interactions such as clinic visits, emergency department visits, consultations, inpatient admissions, daily rounds, critical care services, and others. Although there may be some procedures bundled into these services (such as is the case with critical care services), the majority of this work on the physician's part involves evaluation and management (E&M) of the patient's condition, which are primarily cognitive in nature. As opposed to E&M services, *procedures* include surgical operations, bedside procedures, anesthesia services, radiologic interpretations and procedures, and performance

or supervision of laboratory procedures. In addition, in contrast with cognitive-only E&M services, procedures involve actual physical work by the physician in addition to cognition.

FUNDAMENTAL BILLING CONCEPTS

Most patients do not pay their health care bills directly out of their checking account or credit card, as they do with other purchases. Instead, health care services are usually paid by third parties, usually an insurance company or a government entity such as Medicare or Medicaid. These entities seek to ensure that they are paying for actual and appropriate services. Therefore, they demand to know *what* was done for the patient and *why* it was done before they will process a payment to the physician.

The "why" takes the form of one or more coded diagnoses, currently using the diagnoses enumerated by the World Health Organization in the International Classification of Diseases (ICD). Since the early 1980s, CMS and other third parties have required the use of a clinical modification (CM) of version 10, ICD-10-CM, for this purpose. Previously, ICD-9-CM had been in use since 1979 but was transitioned to ICD-10-CM on October 1, 2015.

The "what" is contained in one or more CPT codes, which are revised and produced annually by the American Medical Association. CPT provides a system that lists and codes the majority of procedures and services provided by physicians. CMS has established the CPT system as level I of the Healthcare Common Procedure Coding System (HCPCS). Level II HCPCS codes apply to nonphysician services that are billed to CMS, such as ambulance services, prosthetic devices, and rehabilitative services. In addition to identifying the procedure or service performed, the CPT code is the entity to which RVUs are assigned.

It is not uncommon for multiple procedures to be performed during the same operation, and in many cases, they can all be reimbursed. In an effort to counteract procedural "unbundling," however, the Health Care Financing Administration (HCFA)—CMS's previous name—established the Correct Coding Initiative (CCI) in the early 1980s. The CCI defines those procedures that cannot be billed simultaneously.[5] For example, the higher paying splenorrhaphy (CPT 38115, 21.88 work RVUs) cannot be reported at the same time as a splenectomy (CPT 38100, 19.55 work RVUs). Reporting both as having been performed during the same operation will result in payment for the lower-paying splenectomy alone. While seemingly reasonable, there have been several instances where the prohibition of simultaneous billing makes little sense.[6] In such cases, appeals can be made with proper documentation and, often, with modifiers.

Modifiers indicate that there is a change in the fundamental assumption about the service or procedure represented by the CPT code. To ensure appropriate reimbursement, CPT codes may need to be reported with one or more modifiers. As will become apparent, modifiers play an important role for trauma surgeons because of the multiple conditions being managed during surgical global periods. For example, most procedure codes are considered to have some degree of E&M services incorporated into the payment for the code. Therefore, payment for any additional E&M services that are unrelated to the procedure must be justified by the addition of a modifier.

DOCUMENTATION REQUIREMENTS

Appropriate and thorough documentation in the medical record is necessary for effective coding and billing. As previously noted, professional coding and billing personnel are completely dependent upon appropriate and specific physician documentation. The coders simply cannot generate any ICD-10 or CPT codes unless the basis for those code assignments exists in the physician documentation. The documentation must provide information regarding the condition(s) or disease(s) being treated and the procedures or services used in the treatments. Coders translate the documentation into ICD-10 codes for the diagnosis and into CPT codes for the procedures or services. In addition, the procedures or services must be appropriate for the conditions being treated. For example, if a patient admitted with a sacral fracture (ICD-10 code S32.1) requires insertion of a central venous catheter (CPT code 36556 for a nontunneled catheter in an individual >5 years old), there will need to be a separate diagnostic code to justify reimbursement for the central venous catheter because a sacral fracture by itself will not provide that justification. Thus, conditions such as hypovolemia (ICD-10 code E86) or traumatic shock (ICD-10 code T79.4) should be identified as the reason for the central venous catheter. Many of the coding software programs used by billing personnel have cross-coding applications that alert coders when a procedure may not be appropriate for a specific diagnosis, or vice versa. Fortunately, or unfortunately, there are no universal standards for these code pairs. Therefore, there have been situations in which the coding office is telling the physician they have identified an inappropriate diagnosis for a particular procedure and yet the physician knows that his or her diagnosis and procedure code pairing is clinically appropriate. In such circumstances, the physician will need to provide the appropriate clinical guideline, policy, or medical literature to educate the coding staff and provide effective clinical feedback to the cross-coding information provider.

Again, although professional coders can find the proper codes to apply, it is the physician's responsibility to provide adequate documentation for the coders to do their job well. The physician must realize that his or her note provides the information for invoicing the third-party payers. The documentation requirements under ICD-10 are significantly greater than under ICD-9 due to the need to describe conditions and injuries with greater specificity. Thus, it will no longer be adequate to identify an open fracture of the femoral shaft; rather, the fracture must be identified as to the body side (right or left), whether it is displaced or nondisplaced, the type of open fracture, whether the patient encounter for this problem is the initial or subsequent encounter, and, if

the latter, whether the healing has been routine, delayed, or complicated by malunion or nonunion.

SPECIAL CONCERNS FOR PHYSICIAN CODING AND BILLING IN TRAUMA CARE: SURGICAL GLOBAL PACKAGE

In the 1980s, Medicare adopted the concept of a global surgical period to enable a single payment for all services inherent to a procedure. Thus, when a surgeon sees a patient preoperatively on the day of surgery, operates on the patient, and provides routine postoperative wound and other care in the hospital and in the clinic, there are no separate payments for the admission history and physical examination, daily hospital visits, and postoperative clinic visits, nor are there separate payments for dressing changes, suture removal, or other routine postoperative care. The global package applies not only to the surgeon who did the procedure, but also to his or her colleagues in the same specialty from the same billing group, because CMS considers them to be the same individual. Physicians from other specialties or physicians from the same specialty but other billing groups are not considered to be the same individual.

Initially, there was a great deal of variability among Medicare carriers (private health care insurers contracted by Medicare to process claims) with respect to what services were included in the global surgical package and the duration of the global surgical period for each procedure. On January 1, 1992, HCFA established a national global surgery policy, standardizing the included services and durations of each procedural CPT code's global package. E&M services that are included in the global package are considered to have been paid by the global package payment and, therefore, cannot be billed for separately (Table 6-1), although there is evidence that such payments lack equivalence to actual E&M payments for similar services.[7] However, there are many other professional services and procedures performed on surgical patients for which separate billing and payment is appropriate (Table 6-2).

These billable encounters during global periods are common in the practice of trauma surgery as well as in surgical critical care. Unfortunately, many surgeons fail to realize this and consequently lose significant amounts of revenue. The reality is that trauma surgeons currently operate on a small percentage of the patients they manage, so there are often no global package concerns.[8-11] If a trauma surgeon is providing overall management for a multiply-injured patient on whom an orthopedist has operated, the global surgical package for the orthopedic surgeon does not apply to the general (trauma) surgeon because these individuals have two different specialty codes (specialty codes identify the physician's specialty in the bills submitted to third parties).[12] Thus, a general trauma surgeon can bill a daily visit (ie, CPT code 99231, 99232, or 99233, depending on the extensiveness of the note) for nonoperatively managing four rib fractures (ICD-10 S22.4), a grade II splenic injury (ICD-10 S36.0), and acute anemia (ICD-10 D62) on a patient for whom an orthopedic surgeon has performed an open reduction and internal fixation (CPT 27506) on a mid-shaft femur fracture (ICD-10 S72.3).

TABLE 6-1: Components of the Global Surgical Package (cannot be separately billed)[15]

1. Preoperative visits after the decision is made to operate beginning with the day before the day of surgery for major procedures and the day of surgery for minor procedures *(Note that this usually applies to elective surgical cases where the decision to operate was made in the clinic days or weeks prior to the scheduled operative date. Emergency surgical cases are handled differently because the initial evaluation and management [E&M] service in the emergency department can be separately paid for if the -57 modifier is applied, thereby identifying the emergency E&M service itself as the source for the decision to operate. Similarly, a daily E&M progress note can serve as a decision to operate service for an existing inpatient whose condition has changed and now requires an operation; the -57 modifier on that daily progress note will ensure the note is paid.)*
2. Intraoperative services that are normally a usual and necessary part of a surgical procedure (eg, dissection services, exposure services, wound closure, dressing applications, local anesthesia provided by the surgeon)
3. Complications following surgery that do not require additional trips to the operating room (eg, opening a wound at bedside)
4. Postoperative visits related to recovery from the surgery
5. Postsurgical pain management by the surgeon
6. Supplies
7. Miscellaneous services (dressing changes; local incisional care; removal of operative pack; removal of cutaneous sutures and staples, lines, wires, tubes, drains, casts, and splints; insertion, irrigation, and removal of urinary catheters, routine peripheral intravenous lines, nasogastric and rectal tubes; and changes and removal of tracheostomy tubes)

Moreover, many trauma patients have multiple conditions that require the trauma surgeon's attention, over and above those conditions for which this individual has operated, and this management is also reimbursable. It is in these situations where use of a modifier is critical. As previously stated, modifiers indicate that the fundamental assumption about a CPT code is altered for the CPT code being submitted. If, in the previous example, the trauma surgeon performed a splenectomy (CPT 38101), the fundamental assumption about the CPT code is that the daily visit E&M service is bundled into the payment for the splenectomy. Yet, the management of the rib fractures and the anemia is not a usual, customary, and reasonable experience in postsplenectomy care. Applying the -24 modifier to the 99231/99232/99233 daily visit code during the postoperative visit indicates that the E&M service is separately payable because of the management of conditions unrelated to the splenectomy. The modifiers relevant to procedures and services performed during global surgical packages are listed in Table 6-3.

TABLE 6-2: Services and Procedures *Not* Included in the Global Surgical Package (ie, separate billing and payment are appropriate)[15]

1. The initial consultation or evaluation of the problem by the surgeon to determine the need for major (ie, 90-day global package period) surgery. This is a separate evaluation and management (E&M) service (with a -57 modifier). The initial evaluation is always included in the allowance for a minor surgical procedure.
2. Services of other physicians except where the surgeon and the other physician(s) agree on the transfer of care.
3. Visits unrelated to the diagnosis for which the surgical procedure is performed, unless the visits occur due to complications of the surgery.
4. Treatment for the underlying condition for which the surgery was performed or an added course of treatment that is not part of normal recovery from the procedure.
5. Diagnostic tests and procedures, including diagnostic radiologic procedures.
6. Clearly distinct surgical procedures during the postoperative period that are not reoperations or treatment for complications (except for those complications that require a return trip to the operating room [OR]; see 7 below). Note that a new postoperative global period begins with the subsequent procedure.
7. Treatment for postoperative complications that require a return trip to the OR.
8. If a less extensive procedure fails and a more extensive procedure is required, the second procedure is payable separately.
9. Splints and casting supplies are payable separately.
10. Immunosuppressive therapy for organ transplants.
11. Critical care services (codes 99291 and 99292) unrelated to the surgery where a seriously injured or burned patient is critically ill and requires constant attendance of the physician.

TABLE 6-3: Modifiers That Enable Appropriate Billing During Surgical Global Periods

Time point of the CPT code being modified	Type of CPT code that is modified: Evaluation & management (E&M)	Type of CPT code that is modified: Procedure
Same day as global procedure	-25: E&M service unrelated to the procedure performed on the same day. This is used when the procedure carries a 0- or 10-day global period. -57: E&M service involves the decision to operate. This is used when the procedure carries a 90-day global period.	-51: Applied to procedures that are performed on the same day through the same incision as the primary (ie, highest RVU) procedure. -59: Applied to procedures performed on the same day as the primary (ie, highest RVU) procedure but performed at a different time or through a separate incision.
Any day during global period after the day of the procedure	-24: Postoperative E&M service managing conditions unrelated to the procedure whose global period is still present.	-79: Applied to procedures performed for unrelated conditions after the procedure whose global period is still present. -78: Applied to unplanned procedures performed for conditions related to and after a procedure whose global period is still present. -58: Applied to staged or planned procedures performed after a procedure whose global period is still present.

CPT, *Current Procedural Terminology*; RVU, relative value unit.

SPECIAL CONCERNS FOR PHYSICIAN CODING AND BILLING IN TRAUMA CARE: SURGICAL CRITICAL CARE

Critical care E&M services for adult patients are time-based codes, with 99291 being applied for the first 30 to 74 minutes (the first "hour") and 99292 being applied for any subsequent 15- to 30-minute periods during a calendar day. The following two documentation requirements justify the higher E&M payments provided for critical care services: (1) the clinical documentation must support the premise that the patient meets the definition of critical illness; and (2) the physician (and his or her partners within the same specialty) must document the total time spent that day in the critical care of the patient, exclusive of the time spent performing procedures (as they are billed separately). Critical illness or injury is defined by CMS as an acute impairment of "one or more vital organ systems such that there is a high probability of imminent or life threatening deterioration in the patient's condition."[13] Therefore, in addition to documenting the total time spent, the physician's note should also focus on the nature of the organ system impairment(s), the potential consequences (eg, organ failure or death) if untreated, and what the physician has done to manage those impairments.

Despite what many physicians believe to the contrary, the provision of critical care services is specifically excluded from a global surgical package in the setting of trauma and burns[14]; in other words, critical care is not considered part of an operative procedure for trauma patients. Nevertheless, if a trauma surgeon has performed an operative procedure on a critically

ill patient, it is best to identify the clinical condition(s) requiring critical care for the ICD-10 code(s), instead of the diagnosis used for the operation. This would be essential anyway, because a ruptured spleen, for example, does not routinely justify critical care. Trauma patients who are truly critically ill, however, usually have multiple conditions that must be assessed and managed. These all have ICD-10-CM codes, such as respiratory failure (ICD-10 J96.00), traumatic shock (ICD-10 T79.4XXA), post-hemorrhagic anemia (ICD-10 D62), oliguria (ICD-10 R34), and many others. A postoperative (-24) or same-day (-25) modifier should be applied to the critical care E&M code as appropriate. The critical care note should also attest that the time spent performing critical care E&M was independent of time spent on any procedures on the patient.

EXAMPLE: TRAUMA ACTIVATION RESPONSE, OPERATIVE, AND CRITICAL CARE BILLING

You respond to a trauma activation, identifying a 26-year-old male ejected during a high-speed motor vehicle collision. His breathing is shallow, and he is hypoxic on pulse oximetry. The anesthesiologist intubates the patient, and you note decreased breath sounds on the left. A chest radiograph confirms a left hemothorax. You place a left chest tube, which drains 750 mL of gross blood. He is hypotensive and tachycardic, and you perform a focused assessment with sonography in trauma (FAST) exam, which is positive. You take him emergently to the operating room, removing a ruptured spleen. You also debride, cauterize, suture, and pack a grade IV liver laceration. The anesthesiologist places a central venous catheter and an arterial line. The patient begins to stabilize, and you perform a temporary abdominal closure.

You complete your examination of the patient and perform computed tomographic imaging of his head, neck, and pelvis. A small subarachnoid hemorrhage is identified, and you consult neurosurgery. You take him back to the intensive care unit, continue the resuscitation, and note that his vital signs have normalized. You adjust his ventilator settings, order a number of laboratory studies, and discuss his situation and your plans with his family. You have spent a total of 115 minutes in his critical care management (including writing the note).

The next day, you assess his respiratory, cardiovascular, renal, coagulation, and nutritional status, noting some oliguria and anemia. You order infusions of crystalloids and transfuse red blood cells. Also, you pass a gastric feeding tube and trickle in some enteral feedings slowly. You speak again with his family. After writing the daily note, you have spent a total of 75 minutes performing critical care.

On the third day, you perform critical care services as on the previous day, although it only takes you 45 minutes. The patient's oliguria, hypovolemia, and anemia have improved, but they require continued monitoring. You also take him back to the operating room, remove the temporary abdominal closure, and close his abdominal midline incision.

A billing summary for these 3 days of this patient's case is provided in Table 6-4. In the setting of a trauma activation, the comprehensive history, comprehensive physical examination, and high-complexity medical decision making usually qualify for a high-level E&M service, such as an admission note (CPT 99223) if the note appropriately documents those components. If the patient meets the definition of critical illness, as in this case, however, billing for critical care services will pay more. The injuries should be identified and coded as the diagnoses being managed if you are billing a 99223; however, if you are billing a 99291 with or without additional 99292 codes, the abnormal physiologic conditions making the patient critically ill should also be coded as the diagnoses being managed. If a decision is made to take the patient to the operating room, a -57 modifier should be applied to the E&M service on the first day. Other minor bedside procedures, such as the chest tube insertion, can also be billed, but will require that a -25 modifier be applied to the E&M service also. Without the -57 and/or the -25 modifiers being applied to the E&M CPT code (either 99223 or 99291/99292), you will not be paid for the E&M service. The underlying assumption is that the patient came to have the procedures performed, and all procedures are considered to have some E&M care included in their payments. Given that there are conditions requiring E&M other than those E&M services related specifically to the procedures performed, it is essential to provide appropriate modifiers to ensure that the additional E&M services are legitimately paid.

BACK-OFFICE ISSUES

Because of the complexity of the documentation, coding, and billing process for acute care surgeons, it is essential that physicians maintain a healthy and collegial relationship with their billing and coding personnel. Both groups share the common goal of ensuring that the organization receives the maximal legitimate reimbursement possible. Coding and billing personnel need to understand what conditions physicians are treating and how they are treating them so that the appropriate codes can be submitted for third-party payments. They should be able to capture this from the clinical documentation, but whenever doubt exists, they should feel absolutely free to contact the physicians for clarification. They should also feel comfortable in suggesting clarification of documentation by the physician when necessary should there be documentation deficiencies that could be challenged in an audit.

Physicians, in turn, should learn as much as they can about the coding and documentation issues pertinent to their field. There are several opportunities to acquire such knowledge, which can often include discussions with their coding and billing personnel regarding specific services. In addition, there are various books and subscriptions available, including online software and subscriptions. For example, software such as HCPro's EncoderPro Professional provides a comprehensive catalog of ICD-10, CPT, DRG, Ambulatory Payment Classification, and HCPCS codes, as well as a great deal of other information. Although modestly

TABLE 6-4: Summary of Billing Potential for Example Case

Day	Diagnoses (ICD-10-CM)	Description	Procedure or service (CPT)	Description	wRVUs
First day	J96.00	Respiratory failure	99291-57-25	2 hours of critical care with decision to operate modifier and same-day minor procedure for the E&M service	9.00
	T79.4XXA	Traumatic shock	99292-57-25x2		
	S06.6X9A	Subarachnoid hemorrhage (SAH) with loss of consciousness (LOC) of unspecified duration			
	S27.1XXA	Traumatic hemothorax (closed)	32551-59	Chest tube insertion with modifier for distinct procedure (from liver packing)	3.04
	S36.81XA	Hemoperitoneum	76700-59	Abdominal ultrasound with modifier for distinct procedure	0.81
	S36.116A	Major liver laceration	47361	Exploration of hepatic wound, extensive debridement, coagulation, suture, with packing of liver	52.60
	S36.032A	Major splenic laceration	38100-51	Splenectomy with modifier for multiple procedures: 50% payment[a]	9.78
	Total first day wRVUs				**75.23**
Second day	J96.00	Respiratory failure	99291-24	90 minutes of critical care with modifier for postoperative unrelated E&M services. (Note that gastric intubation is included in 99291.99292.)	6.75
	S06.6X9A	SAH with LOC of unspecified duration	99292-24		
	R34	Oliguria			
	E86.1	Hypovolemia			
	D62	Post-hemorrhagic anemia			
	Total second day wRVUs				**6.75**
Third day	J96.00	Respiratory failure	99291-24-57	60 minutes of critical care with modifier for postoperative unrelated E&M services as well as modifier for decision to operate on day of surgery	4.50
	S06.6X9A	SAH with LOC of unspecified duration			
	R34	Oliguria			
	E86.1	Hypovolemia			
	D62	Post-hemorrhagic anemia			
	S31.619A	Open wound of abdomen	49002-58	Relaparotomy for closure with modifier for staged procedure	17.63
	Total third day wRVUs				**22.13**

[a]Because of the E&M services purportedly included in operative procedures, the second procedure's RVU valuation is halved to avoid "overpayment" of duplicate included E&M services.

CPT, *Current Procedural Terminology*; E&M, evaluation and management; wRVU, work relative value unit.

expensive, these programs will pay for themselves if conscientiously used to improve the quality of documentation, coding, and billing, which will result in enhanced professional revenue.

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7

Critical Appraisal of Trauma Research

Angela Sauaia • Ernest E. Moore

KEY POINTS

- By randomly distributing patients to the study groups, the potential risk factors in randomized clinical trials are likely to be evenly dispersed.
- Bayesian statistics are an approach for learning from evidence as it accumulates; that is, the Bayes theorem is applied to combine prior information with current information on an outcome of interest and derive a probability.
- The propensity score is the probability of treatment assignment conditional on observed baseline characteristics; that is, each treated patient is matched to one or more control patients with similar propensity scores.
- Relative risk or risk ratio (RR) compares the outcome probability in two groups (ie, with and without an intervention, or with and without a risk factor). When the RR is equal to 1, there is no evidence of effect.
- Odds ratios are good estimates of the RR when the outcome is relatively rare (<20%); however, this is not true when the outcome is more common.
- *P* value is the probability of obtaining the observed effect (or larger) under the null hypothesis that there is no effect; that is, the *P* value can be interpreted colloquially as the probability that the finding was the result of chance.
- Bias is the deviation of results due to systematic errors in the research methods and includes selection bias (study groups differ systematically) or observer/information bias (systematic differences in the way information is collected).

Good doctors use both individual clinical expertise and the best available external evidence, and neither alone is enough. Without clinical expertise, practice risks becoming tyrannized by evidence, for even excellent external evidence may be inapplicable to or inappropriate for an individual patient. Without current best evidence, practice risks becoming rapidly out of date, to the detriment of patients.

Sacket et al, 1996

WHAT IS EVIDENCE-BASED TRAUMA CARE?

Evidence-based medicine (EBM) is "the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients."[1] The term *evidence-based medicine* was coined by a group of physicians at McMaster University in Hamilton, Ontario, in the early 1990s.[1,2] EBM is a combination of clinical expertise and best evidence, as eloquently stated by Sacket and colleagues in the quotation at the beginning of this chapter.[1] The definition of EBM has two basic components: the first is "the conscientious, explicit, and judicious use," which, in trauma, applies to split-second decisions in the face of an immense variety of unexpected clinical scenarios. The time pressure and the irreversibility of many surgical procedures enhance the anxiety of decision making. The second component of the EBM definition is "best evidence." What constitutes "best evidence"? Best evidence is "clinically relevant research,"[1] which can both invalidate previously accepted procedures or replace them with new methods that are more powerful, efficacious, safe, and cost containing. In simple words, it comes down to "How does the article I read today change (or not) how I treat my patients tomorrow?"[3]

Searching for evidence usually starts with the formulation of a searchable clinical question. For this purpose, experts recommend the use of the PICO framework,[4] which stands for population (P), interventions (I), comparators (C), and outcome(s) (O). When searching for evidence on a specific topic or simply determining whether the most recent article

applies to the patient currently being seen, one must define whether the PICO in the study(ies) is (are) similar enough to the individual patients in your practice to allow application of the evidence. For example, in the 1990s, randomized clinical trials showed the benefits of treating *Helicobacter pylori* in patients with peptic ulcer disease; thus, guidelines recommended this practice in all patients with peptic ulcer disease.[5] Yet subsequent evidence showed that, among elderly patients, *H pylori* treatment did not have as much of an impact as anticipated based on this evidence. Application of the PICO framework to the initial randomized clinical trials unveiled that many trials excluded elderly patients, who often have comorbidities and take nonsteroidal anti-inflammatory drugs (population); thus, their findings did not necessarily apply to elderly patients. Indeed, among this advanced age group, counseling for appropriate use of nonsteroidal anti-inflammatory drugs had a greater effect.[6]

Once you determine that a published report applies to your practice, critical appraisal is the next step. Critical appraisal ascertains the level of uncertainty of the study's findings through the assessment of its internal and external validity. This step is essential to filter the information that can improve individual patient care as well as contribute to an efficient, high-quality health care system. Critical appraisal of an article requires an inquisitive and skeptical mindset combined with a basic understanding of scientific and statistical methods.[7] Many would say that scientific and statistical methods are only for researchers, but actually they are essential in becoming effective consumers of research and EBM practitioners. This chapter will provide a basic review of methods for searching for, critically appraising, and applying evidence, including an introduction to statistical topics essential to a reliable interpretation of trauma literature.

DEFINING THE LEVEL OF EVIDENCE

Best evidence rarely comes from a single study; more often, it is the final step of a long scientific journey, in which experts collect, appraise, and summarize the findings of several individual studies using standard methods.[1] Systematic reviews, as detailed later in this chapter, are excellent starting points to distill the daunting amount of information available. Indeed, when first learning about a topic, a systematic review is a good start. These reviews are commonly assigned a level of evidence, which gauges the confidence of the summative collection of research on a specific topic.

When a systematic review is not available, how should busy health care providers manage the formidable volume of information that becomes available every day, much of which is contradictory? Appraisal is the answer. There are several systems to appraise and grade the level of evidence. For example, the Grading of Recommendations Assessment, Development and Evaluation (GRADE)[8] system follows a detailed stepwise process to rate evidence (http://www.gradeworkinggroup.org/; accessed September 9, 2018). The *Journal of Trauma and Acute Care Surgery* recently adapted the GRADE system to classify the level of evidence of individual articles (Table 7-1). The study design (ie, randomized study, observational prospective or retrospective, case-control, case series) is a major factor in the classification, because a randomized trial produces more certainty than a case series. Yet we must recognize that different clinical questions (ie, whether the question relates to a therapeutic procedure, diagnostic test, prognostic factors, epidemiologic description, etc) demand different types of study designs and tolerate different levels of uncertainty.[9,10] In other words, potentially harmful therapeutic approaches demand a higher level of certainty (thus a higher evidence level) than the validation of a new score to gauge injury severity.

The determination of the level of evidence of a study involves four steps.

Step 1: Define the Type of Clinical Topic Addressed by the Study

1. Therapeutic and care management studies evaluate a treatment's efficacy, effectiveness, and/or potential harm; also included in this category are comparative effectiveness research, investigations of adherence to standard protocols, clinical recommendations, guidelines, and/or algorithms.
2. Prognostic and epidemiologic studies[11] assess the influence of predictors or risk factors of a condition and/or its outcome. Epidemiologic investigations describe the incidence or prevalence of a disease and its risk factors, diagnosis, morbidity, and prognosis; also included are new or revised scores to gauge disease severity (eg, Injury Severity Score [ISS]).
3. Diagnostic test or criteria[12] studies describe the validity and applicability of diagnostic tests or procedures; also included are assessments of diagnostic criteria to define certain conditions (eg, definition of adult respiratory distress syndrome, multiple organ failure, trauma-induced coagulopathy).
4. Economic and value-based evaluations focus on the value, quality, and cost of specific care management strategies. They include cost-benefit, cost-effectiveness, and cost-utility analyses. More recently, Porter[13,14] proposed the concept of value-based health care evaluations, in which value was defined as the health outcomes achieved per dollar spent.
5. Systematic reviews and meta-analyses evaluate the body of evidence on a topic through comprehensive, systematic search and appraisal; meta-analyses specifically include the quantitative pooling of data across different studies addressing a single topic.
6. Guidelines are statements developed by reputable groups of experts to assist practitioners and patients with decisions about the appropriate health care for the specific clinical circumstances, based on the appraisal of existing evidence.[15]

Step 2: Define the Research Questions, the Hypothesis(es), and the Study Design Chosen to Address Them

The study design is the plan used by the investigators to test a hypothesis related to a research question as unambiguously as possible given practical and ethical constraints. Even

TABLE 7-1: Evidence Level of Individual Studies

Types of studies

Level	Therapeutic/care management	Prognostic and epidemiologic	Diagnostic tests or criteria	Economic and value-based evaluations	Systematic reviews and meta-analyses	Guidelines
Level I	• Randomized controlled trial (RCT) with no negative criteria[a] and with a significant difference • RCT with no negative criteria[a] without significant difference and adequate statistical power[b]	• Prospective[c] study with large effect[d] and no negative criteria[a]	Testing of previously developed diagnostic criteria in consecutive patients (all compared to "gold" standard)	Sensible costs and alternatives; values obtained from many sources; multiway sensitivity analyses	Systematic review (SR) or meta-analysis (MA) of predominantly level I studies and no SR/MA negative criteria[e]	GRADE system
Level II	• RCT with significant difference and only 1 negative criterion[a] • Prospective[c] comparative study with no negative criteria[a]	• Prospective[c] study with less than large effect[d] and no negative criteria[a] • Untreated controls from RCT • Prospective/retrospective[c] study with large effect[d] and only 1 negative criterion[a]	Development of diagnostic criteria on consecutive patients (all compared to "gold" standard)	Sensible costs and alternatives; values obtained from limited sources; multiway sensitivity analyses	SR/MA of predominantly level II studies with no SR/MA negative criteria[e]	
Level III	• Case-control study with no negative criteria[a] • Prospective[c] comparative study with only 1 negative criterion[a] • Retrospective[c] comparative study with no negative criteria[a]	• Case-control study with no negative criteria[a] • Prospective or retrospective[c] study with up to 2 negative criteria[a]	Nonconsecutive patients (without consistently applied "gold" standard)	Analyses based on limited alternatives and costs; poor estimates	SR/MA with up to 2 negative criteria[e]	
Level IV	Prospective or retrospective[c] study using historical controls or having more than 1 negative criterion[a]	• Prospective or retrospective[c] study with up to 3 negative criteria[a]	Case-control study with no negative criteria[a]	No sensitivity analyses	SR/MA with more than 2 negative criteria[e]	
Level V	• Case series • Studies with quality worse than level IV	• Case series • Studies with quality worse than level IV	No or poor "gold" standard			

[a]Negative criteria (decreases level of evidence):
1. <80% follow-up
2. >20% missing data or missing data not at random without proper use of missing data statistical techniques
3. Limited control of confounding (eg, mortality comparisons with inadequate risk adjustment)
4. More than minimal bias (selection bias, publication bias, report bias, etc)
5. Heterogeneous populations (eg, institutions with distinct protocols/patient volume; conditions caused by distinct pathogenic mechanisms)
6. For RCT only: no blinding or improper randomization

[b]Adequate statistical power: this only applies to studies *not* finding statistical differences, and it is defined as power >80% for declaring "failure to detect a significant difference" or power >90% for declaring "bio-equivalence or noninferiority or comparative effectiveness" of a *predefined* difference based on previous evidence.

[c]Prospective versus retrospective: studies with data collected to answer predefined questions are prospective; studies with data collected for questions unrelated to the original question for which the data were gathered are retrospective.

[d]Large effect:
- Study with large relative risk (RR; >5 or <0.2) about condition of low to moderate morbidity/mortality
- Study with moderate to large RR (2–5 or 0.2–0.5) about condition of high morbidity/mortality

[e]Negative criteria for systematic reviews and meta-analyses (decreases level of evidence):
1. No or inadequate standard search protocol
2. More than minor chance of publication bias or publication bias nonassessed
3. Moderate heterogeneity of included studies and/or populations (eg, elective surgery and acute surgery)
4. Predominance of level III or lower studies
5. No measures or inappropriate measures of pooled risk (for meta-analysis only)

Modified with permission from Sauaia A, Moore EE, Crebs JL, Maier RV, Hoyt DB, Shackford SR. Evidence level of individual studies: a proposed framework for surgical research. *J Trauma Acute Care Surg*. 2012;72(6):1484-1490.

descriptive and exploratory studies should have research questions and, most of the time, a testable hypothesis. This step is where you assess whether the research design was appropriate to address the research question and test the hypothesis. Certain designs are better than others; randomized clinical trials (RCTs) remain the paragon of biomedical research, followed by cohort comparative studies, case-control studies, and case series, in that order. In RCTs, by randomly distributing patients to the study groups, potential risk factors are likely to be evenly dispersed. When blinding of patients and investigators is added to the design, bias is further minimized. These processes diminish the risk of confounding factors influencing the results. Therefore, the findings generated by RCTs are likely to be closer to the true effect than the findings generated by other research designs.

Although RCTs provide the best evidence due to unparalleled control of confounding, they have weaknesses, such as limited generalizability/external validity due to strict enrollment criteria (ie, they may not reflect the intended population). In addition, study settings and procedures may not mirror real-life practices. Indeed, as Archie Cochrane, the creator of the Cochrane Collaboration (the most important collection of systematic reviews), himself noted, randomization does not, of itself, promote external validity; that is, the applicability of a trial's results to situations other than the precise one in which the trial was done. Recognizing that RCTs do not always have great generalizability, funding and regulatory agencies, such as the US Food and Drug Administration (FDA), have started to rely also on what is called real-world data (RWD; the data relating to patient health status and/or the delivery of health care routinely collected from a variety of sources) and real-world evidence (RWE; clinical evidence regarding the usage and potential benefits or risks of a medical product derived from analysis of RWD) in addition to RCTs (see https://www.fda.gov/scienceresearch/specialtopics/realworldevidence/default.htm; accessed October 18, 2018).

Also in response to these valid concerns, RCTs labeled as pragmatic have been designed, in which the context and the included subjects reflect more accurately the environment and population to which the findings should be applied.[16] For example, the COMBAT[17] and the PROPPR trials,[18] which assess blood transfusions in the resuscitation process, were both pragmatic trials, with limited exclusion criteria and conducted with minimal interference to the standard of care other than the tested intervention. More recently, stepped-wedge RCTs have been proposed.[19,20] The stepped-wedge pragmatic design is different than the usual parallel RCTs, in which the intervention and control groups run, as the name suggests, in parallel. The stepped-wedged approach involves a sequential rollout of an intervention to participants (individuals or clusters) over a number of time periods, such that at the end of the study, all participants will have received the intervention. The name of the design (stepped-wedged) comes from the schematic illustration of the design. The order in which participants receive the intervention is assigned at random. The design is particularly relevant when the intervention is likely to have a benefit, thus making a control group possibly unethical. For example, the 1987 Gambia hepatitis intervention study,[21] the likely pioneer stepped-wedge trial, tested the effectiveness of a hepatitis B vaccine. Because the vaccine had established effectiveness at preventing hepatitis B (although no definitive evidence showing protection for chronic liver disease existed), it would be unethical to have a simultaneous control group receiving placebo. Thus, in order to obtain evidence on long-term benefit, a sequential but random rollout of the hepatitis B vaccination was implemented; geographic areas were randomly allocated to incorporate the vaccine into the existing childhood vaccination schedule. A new region was randomly allocated at 10- to 12-week intervals, such that complete national coverage was obtained after a few years. The stepped-wedge design is also useful when it is difficult to deliver the intervention simultaneously to all participants (or hospitals) for logistical, practical, or financial reasons (eg, use of whole blood in postinjury resuscitation).

We should also mention Bayesian trials, which are starting to gain popularity with the availability of computational algorithms and computing speed making it possible to carry out calculations for the very complex Bayesian models.[22] Because these trials are much more complex than traditional frequentist approaches, a full description of this type of trial is beyond the scope of this chapter. In brief, Bayesian statistics are an approach for learning from evidence as it accumulates; it applies the Bayes theorem to combine prior information (the prior probability, ideally based on pilot data or literature) with current information on an outcome of interest and derive a posterior probability (revised/updated probability of an event occurring after taking into consideration new information). In a Bayesian trial, the inferences are updated each time new data become available. Bayesian trials can be advantageous to reduce sample size, make modifications to the randomization ratio mid-study, deal with missing data, and in trials with multiple outcomes. However, the complexities of Bayesian trials should not be underestimated.[22] For an example in trauma, Jansen et al[23] provided a didactic description of a Bayesian trial, the UK-REBOA trial, designed to compare standard major trauma center treatment plus resuscitative endovascular balloon occlusion of the aorta (REBOA) with standard-of-care treatment alone for trauma patients with life-threatening torso hemorrhage.

When controlled experimentation is not possible or ethical, other research models must be considered.[24] Cohort comparative and case-control studies include a comparison group (ie, a group of patients who received a different type of care, procedure, or test). Alternatively, the investigator can compare the same group of patients before and after an intervention, using historical controls. The fundamental difference between cohort and case-control studies resides on where we start (ie, the outcome or the intervention/risk factor). In case-control studies, investigators find a group of patients with a specific outcome (eg, patients with severe torso trauma and coagulopathy) and a comparable group of patients without the outcome (eg, patients with severe torso trauma but no coagulopathy); the investigator then compares the incidence

of a risk factor (eg, hemodynamic instability in the field) or whether a particular intervention was instituted (eg, plasma in the field). For example, Wu et al[25] used a case-control design to compare the bone mineral density (BMD) of 87 elderly patients with hip fractures to 87 elderly patients without hip fractures and found BMD to be significantly lower among patients with the outcome (ie, hip fracture). In contrast, for cohort studies, investigators find a group of patients with the risk factor or intervention (eg, severe torso trauma patients who received plasma in the field) and a comparable group without the risk factor or intervention (eg, severe torso trauma patients who did not receive plasma in the field); then the outcome (eg, coagulopathy) rates of these groups are compared.

To make things more confusing, a case-control study is sometimes a later offspring of a well-planned cohort study, as in the case-control study by Shaz et al[26] on postinjury coagulopathy, which was nested within the ETIC (Early Trauma-Induced Coagulopathy) Prospective Study. Of the 383 ETIC patients, the authors selected 38 patients with prolonged prothrombin time (study cohort) and compared them with 53 age- and sex-matched controls with normal prothrombin time (control cohort). The authors reported decreased factor activities without significant differences in thrombin and fibrin generation between these two groups. Although important medical discoveries have been made through case-control studies, such as the association between smoking and lung cancer,[27] this design has several limitations that place it lower on the evidence hierarchy. These include, but are not limited to, potential for bias in the selection of the control group and uncontrolled confounding in the assessment of the risk factor or intervention.

Case series rank the lowest in the evidence hierarchy. They describe a group of patients with a certain condition or patients who were submitted to a specific care, procedure, or test, but without a comparison group. Case series have a role in rare conditions and in describing the preliminary experience with a new intervention. These studies generate hypotheses and motivate larger studies with more powerful designs. The lack of a comparator, however, lowers the confidence in the findings; thus, this type of study is less likely to change current practice. Of course, if this is an innovative treatment for a currently incurable, lethal disease, we may adopt it despite the low confidence for lack of better options. The urgency to adopt the new treatment, however, does not change the fact that the confidence generated by this design is low and that further research is crucial to decrease the uncertainty.

Matching is a procedure employed to decrease confounding in nonrandomized studies. Specifically, propensity score matching (PSM) has gained increased popularity in clinical research as an alternative design when RCTs are not possible.[28-32] In brief, the propensity score is the probability of treatment assignment conditional on observed (emphasis on *observed*) baseline characteristics. Each treated patient is matched to one or more control patients with similar propensity scores (ie, similar propensity to receive the treatment) but who did not receive the intervention. For example, patients treated with REBOA can be matched to patients with similar clinical *propensity* to require REBOA (ie, similar propensity scores) but who did not receive the procedure for some nonclinical reason (eg, physicians not trained on the procedure); the outcomes of these two groups are then compared. In traditional matching, REBOA-treated patients are matched on specific variables (eg, injury mechanism, age, cardiac arrest), whereas in PSM, they are matched on a combination of these variables (the propensity score). PSM works well when the intervention is relatively new and there is variation in its adoption by health care professionals for patients with the same set of clinical indications. There is controversy regarding how well PSM studies reflect the results of correspondent RCTs. Lonjon et al[33] reported no significant differences in effect estimates between RCTs and PSM observational studies regarding surgical procedures. Conversely, Zhang et al[34,35] reported that PSM studies tended to report larger treatment effects than RCTs in the field of sepsis, but the opposite was true for studies in critical care medicine. It is likely that differences in studied populations and confounders play a role in the disparities between PSM and RCTs. We must always keep in mind that RCTs control for both measured and unmeasured confounders, whereas PSM is only as good as the measured confounders included in the propensity score model. While appraising a PSM study, we must make sure to inspect the model used to generate the propensity score: Were all important confounders included in the model? More on the PSM approach will be presented in subsequent sections of this chapter.

Step 3: Define Effect Size

Effect size represents the magnitude and the direction of the difference in the outcomes between studied groups.[36,37] There are several measures of effect size, and some of the most commonly reported are as follows:

1. *Relative risk or risk ratio (RR):* RR compares the outcome probability in two groups (eg, with and without an intervention, or with and without a risk factor). When equal to 1, there is no evidence of effect.
2. *Odds ratio (OR):* This index compares the odds of the outcome in two groups. Unless the reader is a gambler, the concept of odds is not intuitive, yet because the highly popular logistic regression (discussed later in this chapter) produces ORs, this measure is often reported in the literature. Thus, it is important we understand it. For example, in a group of pediatric severe trauma admissions:
    - Odds of death in the group receiving antifibrinolytic: number of deaths/number of survivors
    - Odds of death in the group *not* receiving antifibrinolytic: number of deaths/number of survivors
    - OR: odds of death in the antifibrinolytic group/odds of death in the non-antifibrinolytic group

ORs are good estimates of the RR when the outcome is relatively rare (<20%); however, this is not true when the

outcome is more common. In studies with high prevalence of the outcome, OR may exaggerate the effect and should be interpreted with caution.[38] To illustrate the dangers of misinterpretation of OR, consider the study by Schulman and colleagues[39] titled "The Effect of Race and Sex on Physicians' Recommendations for Cardiac Catheterization" published in the *New England Journal of Medicine* (*NEJM*) in 1999. In this experiment, physicians were asked to predict compliance and potential benefit from revascularization for cases portrayed by actors of different races and sex. A logistic regression model showed that black race was associated with a lower likelihood of receiving a referral to cardiac catheterization, with an OR of 0.4. The authors concluded that the "race and sex of a patient independently influence how physicians manage chest pain."[39] Their study received extensive coverage in the news media, including a feature story on ABC's *Nightline*, with Surgeon General David Satcher providing commentary.[40] For the most part, the media interpreted the findings as "blacks were 40% less likely to be referred for cardiac testing than whites." In a subsequent *NEJM* Sounding Board article, Schwartz and colleagues[40] called attention to the dangers of using the OR as an effect size measure when the outcome was very common; in the Schulman et al[39] study, greater than 80% of patients had the outcome (referral for cardiac catheterization). The OR, in this case, led to a gross exaggeration of the actual RR: the reported OR of 0.6 [(blacks referred/blacks not referred)/(white referred/whites not referred)] actually corresponded to an RR of 0.93. In other words, blacks were 7% less likely than whites to receive the referral for further cardiac testing. Quite a difference! When faced with the report of OR in studies with outcomes more frequent than 20%, one can use the formula proposed by Zhang and Yu[38] to obtain an estimate of the RR, as follows:

$$RR = OR/[(1 - Po) + (Po \times OR)]$$

where Po is the probability of the outcome in the control (or reference) group.

Although this formula has been criticized for still overestimating the RR (although less than the OR) and not accounting for confounders,[41] it is a simple calculation that can assist the reader in obtaining a more realistic estimate of the effect size when the OR was inappropriately used. One may ask why researchers are still using OR instead of RR; the answer is that logistic regression, one of the most popular statistical methods, deploys ORs. Until recently, models that derived RR were more difficult to use, and software was not as widely available as that for logistic regression. This has now changed with a myriad of user-friendly models that estimate RR; thus, there is little (no) excuse for reporting OR when the outcome is common.

Large RRs and ORs tend to strengthen the evidence. Although there is no strict definition of a large effect, effects greater than 5 (or smaller than 0.2, when the desired direction is to decrease the outcome) in a condition of low to moderate morbidity or mortality (eg, stable femur fracture in a young person) are considered large effects.[3] In highly morbid or lethal conditions (eg, penetrating chest wounds), an effect size greater than 2 (or <0.5) may be considered large enough.

Two other measures of effect size are also reported in publications:

1. *Cohen's d:* This measurement is the standardized mean difference; it is not commonly reported but can be easily calculated as: (mean 1 – mean 2)/(pooled standard deviation).[42] Cohen's d values around 0.2 are considered small; 0.5, medium; and 0.8 or greater, large.
2. *Correlation:* Pearson's correlation coefficient *r* (and its nonparametric equivalent, the Spearman's rho) measures the correlation between two continuous outcomes (eg, volume transfused and systolic blood pressure) and ranges from –1 to 1. Cohen (the same statistician who proposed the Cohen's d) suggested the following rule of thumb for correlations: small = |0.10|, medium = |0.30|, and large = |0.50|.[43]

Step 4: Assess the Limitations of the Study

The next step recognizes that all research designs, even RCTs, are more or less limited by confounding, bias (especially selection bias), inadequate sample size and statistical power, heterogeneity of included subjects, missing data, loss to follow-up, lack of generalizability, and other factors. These factors increase the uncertainty surrounding the research findings and, consequently, decrease its level of evidence. These factors will be explored in detail in the next sections of the chapter.

A FEW STATISTICAL CONCEPTS

Study Designs

In previous sections, we briefly defined study designs. The classic hierarchy ranks designs based on their ability to decrease bias and confounding. It is usually illustrated by a pyramid, with designs in the following order (from the top of the pyramid to the bottom): (1) systematic reviews and meta-analyses (of high-quality RCTs with little heterogeneity); (2) RCTs with confidence intervals (CIs) that do not overlap the threshold of clinically significant effect; (3) RCTs with point estimates that suggest clinically significant effects but with overlapping CIs; (4) cohort studies; (5) case-control studies; (6) cross-sectional surveys; and (7) case reports.[44] More recently, it has been proposed that systematic reviews are not at the top of the pyramid; rather, they correspond to the lens through which we examine the available evidence.[45] Currently, however, several investigators and organizations recognize that most clinical trials, despite the best designs, fail to provide the evidence needed to inform medical decision making.[46] Thus, such strict ordering has been criticized; a more flexible rule is that we must use the best research design available to define the best evidence.[47]

RCTs are considered the best design because the randomization provides an unbiased group allocation and often (but not always) results in similar distribution of both observed and nonobserved confounders across study arms. Ultimately, the goal of randomization is "to ensure that all patients have the same opportunity or equal probability of being allocated to a treatment group."[48] In random allocation, the assignment cannot be predicted for any individual patient.[49] If effective, randomization creates at the start of the study (before the intervention) groups of patients with similar prognoses; therefore, the trial results can be attributed to the interventions being evaluated.[50]

In emergency research trials, the patients are not recruited; instead, they are enrolled as they suffer an injury, in a completely random fashion. This is an advantage, as the subjects enrolled may more closely resemble the target population. However, emergency trauma research imposes challenges to the randomization as the interventions must be made available without any delay.[51,52] Conventional randomization schemes (eg, sealed envelope with computer-generated random assignment, calling a randomization center) may impose unethical delays in providing treatment. Other major challenges of sophisticated randomization schemes are adherence to protocol and rapid, inclusive enrollment. Thus, alternative schemes (eg, prerandomization in alternate weeks) have been proposed.[53] In a recent trial, we randomized severely injured patients for whom a massive transfusion protocol was activated to two groups: (1) viscoelastic (thromboelastography)-guided, goal-directed massive transfusion or (2) conventional coagulation assays (eg, prothrombin time) and balanced blood product ratios on predefined alternating weeks.[54] The system was formidably successful in producing comparable groups at baseline and minimized interference in the resuscitation. Another example is the Prehospital Acute Neurological Treatment and Optimization of Medical Care in Stroke Study (PHANTOM-S), published in 2014, in which patients were randomly assigned weeks with and without availability of a stroke emergency mobile unit.[55] These alternative randomization approaches are recognized as appropriate in emergency research.[51]

Adaptive designs, including adaptive randomization, have been proposed to make trials more efficient.[56,57] A 2015 draft guidance document from the FDA defines an adaptive design clinical study as "a study that includes a prospectively planned opportunity for modification of one or more specified aspects of the study design and hypotheses based on analysis of data (usually interim data) from subjects in the study."[58] Just as an example, the 2015 PROPPR trial used an adaptive design to grant their Data and Safety Monitoring Board (DSMB) authority to increase the sample size and reach adequate power during interim analyses.[59] The trial assessed effectiveness and safety of transfusing severely injured patients with major bleeding using plasma, platelets, and red blood cells in a 1:1:1 ratio compared with a 1:1:2 ratio. Their initial sample size (n = 580) was planned to detect a clinically meaningful 10% difference in 24-hour mortality based on previous evidence. Based on the results of an interim analysis, their DSMB recommended increasing the sample size to 680, which would allow the trial to have 95% power to detect the prespecified 10% difference at 24 hours and 92% power to detect the prespecified 12% difference at 30 days, if such differences existed. Interestingly, however, the trial did not show a significant difference in mortality at 24 hours (12.7% in 1:1:1 group vs 17.0% in 1:1:2 group; $P = .12$) or 30 days (22.4% vs 26.1%; $P = .26$). The study was not powered to detect the observed differences of 4.3% and 3.7% (much smaller than the prespecified differences) in the two mortality outcomes.

In sum, the research design must be appropriate to the research question, ethical, and valid, both internally and externally. Internal validity refers to the extent to which the results of the study are biased or confounded. In the next sections, we will discuss bias and confounding in more detail, but basically this comes down to one question: Is the association between outcome and effect reported in the study real? How much of it may be due to bias and/or confounders? External validity, on the other hand, reflects the extent to which the study is generalizable.

Primary and Secondary Data

When researchers use data collected for the specific, preplanned purpose of addressing a research question, they are using primary data. However, in the era of "big data," it is common to see the use of secondary data, collected for purposes unrelated to the specific research question. Secondary data sets have advantages, as they are usually large and inexpensive. Yet, they have had mixed results when used for such tasks as risk adjustment.[60] Administrative data sets collected for billing purposes, for example, are often influenced as a result of financial reasons, which can favor over- or undercoding, and the number of diagnoses may be capped or deincentivized due to declining marginal returns in billing.

In addition, medical coding and clinical practices are subject to change over time due to a variety of reasons; thus, longitudinal studies need to make sure to account for changes. For example, the coding of "illegal drugs" upon trauma admission is likely to change in unpredictable ways in states where cannabis became a legal recreational drug. Another glaring example relates to collection and coding of the social construct variable race and ethnicity, which has changed dramatically over the past few decades.[61] In addition, comorbidities may be misdiagnosed as complications and vice versa. To address this problem, since 2008, most hospitals now report a "present on admission" (POA) for each diagnosis in its administrative data as a means to distinguish hospital-acquired conditions from comorbidities. Of course, using data from before and after modifications were implemented, such as the inclusion of a POA code, affects the quality of the data. Whenever longitudinal data are used in a study, especially if covering long periods of time, it is important to verify whether there were changes in data collection, health policies, or regulations that could potentially affect the data.

Sometimes the distinction between primary and secondary data becomes blurry, as happens in registries such as state-mandated and hospital-based trauma registries or the National Trauma Data Bank (NTDB), a voluntary national trauma data set maintained by the American College of Surgeons. These data sets were developed to provide a comprehensive epidemiologic characterization of trauma; thus, one can assume that when the research question is related to frequency, risk factors, treatments, and prognosis of trauma, these represent legitimate primary data. However, registries may lack the granularity to address study hypotheses (eg, analyzing the effects of early transfusions of blood components on coagulation-related deaths). In the end, the discussion of whether registries represent primary or secondary data is probably irrelevant; what matters is whether the data are appropriate to address the research question and whether bias and confounding, variability, and inconsistencies are taken into account during data analysis and research appraisal. For example, low-volume hospitals may not contribute enough to aggregate estimates, biasing mortality toward high-volume facilities. The Center for Surgical Trials and Outcomes Research (Department of Surgery, Johns Hopkins School of Medicine) in Baltimore has done a commendable job of documenting differences in risk adjustment and, more important, providing standardized analytic tools to improve risk adjustment and decrease low-volume bias in studies using the NTDB.[62-64]

Hypothesis, Significance, and Power

All studies that use a statistical test, even purely descriptive studies, have a hypothesis. That is because a statistical test is based on a hypothesis. Every hypothesis can be placed in the following format:

Variable X distribution in Group A
is different (or not different) from
Variable X distribution in Group B

Despite its simplicity, this is a widely applicable model for constructing hypotheses. It sets the stage for elements that must be included in the methods section. The authors must define what characterizes Group A and Group B and what makes them comparable (aside from Variable X). Variable X, which is the variable of interest, must be defined in a way that allows the reader to completely understand how the variable is measured. The hypotheses should be further defined using the previously mentioned PICO framework. For example, "We hypothesize that adult trauma patients (P) receiving pharmacoprophylaxis for venous thromboembolism (I) will have fewer venous thromboembolisms (O) than patients not receiving pharmacoprophylaxis (C)."

The commonly reported *P* value is the probability of obtaining the observed effect (or larger) under the null hypothesis that there is no effect. Colloquially, we can interpret the *P* value as the probability that the finding was the result of chance.[65] The *P* value is the chance of committing what is called a type I error, that is, wrongfully rejecting the null hypothesis (ie, accepting a difference when in reality there is none). More important, the *P* value is *not* how sure one can be that the difference found is the correct difference.

Significance is the level of the *P* value below which we consider a result statistically significant. It has become a worldwide convention to use the $P = .05$ level, although this value is completely arbitrary, not based on any objective data, and, in fact, inadequate in several instances. This level was suggested by the famous statistician Ronald Fisher, who rejected it later and proposed that researchers report the exact level of significance.[66]

In research articles, readers will often see that *P* values were "adjusted for multiple comparisons," with significance set at *P* values smaller than the traditional $P = .05$ threshold. This is a controversial issue in biostatistics, with experts debating the need for such adjustment.[67-69] Those who defend the use of multiple comparisons adjustments claim that multiple comparisons increase the chances of finding a $P < .05$ and inflate the likelihood of a type I error.[68] Those who criticize its use argue that this leads to type II errors (ie, the chance of not finding a difference when indeed there is one).[67,69] One of the authors of this chapter (A.S.) recalls her biostatistics professor claiming that if multiple comparisons indeed increased type I error, then biostatisticians should stop working at the age of 40, as any differences after then would be significant just by chance. Our recommendation is that if the hypotheses being tested are predefined (ie, before seeing the data), then multiple comparisons adjustment is probably unnecessary; however, if hypothesis testing was motivated by trends in the data, then it is possible that even the most restrictive multiple comparisons adjustment will not be able to account for the immense potential for investigator bias. In the end, the readers should check the exact *P* values and make their own judgment about a significance threshold based on the impact of the condition under study. Lethal conditions for which there are few or no treatment options may require looser significance cutoffs, whereas benign diseases with many treatment options may demand stricter significance values.

A special case of multiple comparisons is the interim analysis, which is a preplanned, sequential analysis conducted during a clinical trial. These analyses are almost obligatory in contemporary trials due to cost and ethical factors. The major rationale for interim analyses relies on the ethics of holding subjects hostage of a fixed sample size, when a new therapy is potentially harmful, overwhelmingly beneficial, or futile. Interim analyses allow investigators, upon the DSMB independent advice, to stop a trial early due to efficacy (the tested treatment has already proven to be of benefit), futility (the treatment-control difference is smaller than predicted, and continuing the trial is not likely to result in a significant difference), or harm (treatment resulted in some harmful effect).[70] For example, Bulger et al,[71] in their RCT testing the effect of prehospital hypertonic resuscitation after traumatic hypovolemic shock, reported that the DSMB stopped the study based on the potential harm in a preplanned subgroup analysis of nontransfused subjects. The COMBAT study,[17]

published in 2018, was stopped after the second interim analysis because the absence of differences in the primary outcome of mortality persisted during all analyses, thus strongly suggesting that continuing the trial was unlikely to result in a change in the observed lack of differences.

The 95% CI is a concept related to significance. Its interpretation is as follows: if we were to repeat the experiment infinite times and, at each time, calculate a 95% CI, 95% of these intervals would contain the true effect. A more informal interpretation is that the 95% CI represents the range within which we can be 95% certain that the true effect lies. Although the calculation of 95% CI is highly related to the process to obtain the *P* value, the CIs provide more information on the degree of uncertainty surrounding the study findings. There have been initiatives to replace the *P* value with 95% CI, but these have been met with much resistance. Most journals now require that both be reported. For example, in the CRASH-2 trial, a randomized controlled study on the effects of tranexamic acid (TXA) in bleeding trauma patients, the TXA group showed a lower death rate (14.5%) than the placebo group (16.0%), with $P = .0035$.[72] We can interpret this *P* value as follows: "There is 0.35% chance that the difference in mortality rates was found by chance." The CRASH-2 authors reported the effect size as an RR of 0.91, with a 95% CI of 0.85 to 0.97. This means, in a simplified interpretation, that we can be 95% certain that the true RR lies between 0.85 and 0.97.

The CRASH-2 example reminds us that it is important to keep in mind that statistical significance does not necessarily mean practical or clinical significance. Even very small effect sizes can be statistically significant if the sample size is very large. The CRASH-2 trial, for example, enrolled 20,211 patients to obtain a 1.5% absolute difference and a 9% relative difference in mortality.[72] *P* values are related to many factors other than chance, including the sample size (larger samples sizes often result in significant *P* values), the effect size (larger effect sizes usually produce smaller *P* values), and multiple unplanned comparisons.[73]

We must make sure that the study used appropriate statistical tests. Statistical tests are based on assumptions, and if these assumptions are violated, the tests may not produce reliable *P* values. Many parametric tests (eg, t-test, analysis of variance, Pearson correlation) rely on the normality assumption. The parametric test is a test that relies on a known distribution (eg, the normal distribution), whereas nonparametric tests (eg, Wilcoxon-Mann-Whitney test, Kruskal-Wallis test) do not rely on any distribution. One may ask: Why not always use nonparametric tests? The answer is because they are usually less powerful in detecting differences than parametric tests. When the sample is large (n >30), parametric tests are robust against violations of the normal distribution, because of the Central Limit Theorem (the distribution of the mean of a large number of independent, identically distributed variables will be approximately normal, regardless of the underlying distribution) and the law of large numbers (the sample mean converges to the distribution mean as the sample size increases). However, gross skewness and small sample sizes (n <30) will render parametric tests inappropriate. Thus, if the data are very skewed, as is often the case with number of blood units transfused, length of hospital stay, and viscoelastic measurements of fibrinolysis (eg, clot lysis in 30 minutes), or the sample size is small (as is often the case in basic science experiments), nonparametric tests (eg, Wilcoxon rank sum, Kruskal-Wallis, Spearman correlation) or appropriate transformations (eg, log, Box-Cox power transformation) to approximate normality are more appropriate. More on this topic is in the section on sample descriptors later in this chapter.

Statistical power is the counterpart to the *P* value. It is highly related to the sample size (ie, the larger the sample, the greater the statistical power). It relates to type II error, or the failure to reject a false null hypothesis (a false negative). Statistical power is the probability of accepting the null hypothesis (there is no difference) when there is actually a difference. The most basic formula to calculate power is shown below:

$$N = \frac{2\sigma(Z_{\beta} + Z_{\alpha/2})}{\text{Difference}^2}$$

where N is the sample size in each group (assuming equal sizes), σ is the standard deviation of the outcome variable, Z_{β} represents the desired power (0.84 for power = 80%), $Z_{\alpha/2}$ represents the desired level of statistical significance (1.96 for α = 5%), and "Difference" is the proposed, clinically meaningful difference between means.

Despite its importance, power is one of the most neglected aspects of research articles. In most studies, the researchers are searching for a significant difference; that is, they conducted superiority studies, where one group is hypothesized to have a condition (or intervention) that is superior compared to the other. When a difference is found to not be significant in a superiority study, there are two alternatives: to declare failure to find a significant difference or to produce a power analysis to determine how confident we are to declare that the interventions (or risk factors) under study are indeed similar. The latter alternative is more appealing when it is preplanned as a noninferiority trial rather than as an afterthought in a superiority study. Ideally, all studies should report a power analysis.

Whether statistical power is calculated beforehand (ideally) or afterward (better than not at all), it must always contain the following four essential components: (1) power, minimal of 0.80 or 80% (another arbitrary cutoff in the statistical arena); (2) confidence, usually 0.95 or 95%; (3) value of the outcome of interest in the control group/comparator; and (4) minimum difference to be detected. For example, in the RCT by Gonzalez et al[54] comparing goal-directed resuscitation guided by viscoelastic tests to standard resuscitation with preestablished blood product ratios and guided by conventional coagulation tests:

> A 30% death rate was estimated in the control group[71]; thus, a sample size of 122 patients would have 80% power to detect a minimum of 20% points difference in survival rate between the 2 groups with 95% confidence.

We call attention to how Gonzalez et al[54] proposed the control difference (30%) *based on data from a previous similar trial* by Bulger et al.[71] The minimum difference that a study is powered to detect is based on clinical significance. For example, a difference of 1% in mortality is not likely to be of clinical significance and would require a prohibitively large sample size. However, a sample that can only detect a difference of 50% or larger is too underpowered, because only a miraculous intervention could operate such dramatic difference.

Power analysis can become more complex when we need to take into account the following: (1) multiple confounders (covariates), in which case the correlation between the covariates needs to be taken into account; or (2) cluster effects (explained in more detail later), in which case an inflation factor, dependent on the level of intracluster correlation and number of clusters, is used in the sample size estimation. Not surprisingly, all of these increase the required sample size.

Some clever but complex designs can decrease the required sample size, such as factorial designs, in which two (or more) factors are tested at once. For example, in an elegant RCT[74] about the effect of erythropoietin (EPO) and transfusion threshold (hemoglobin [Hgb] <7 g/dL or <10 g/dL) on neurologic recovery after traumatic brain injury, patients were randomized to one of four groups (EPO + Hgb <7 g/dL; placebo + Hgb <7 g/dL; EPO + Hgb <10 g/dL; or placebo + Hgb <10 g/dL). This design approach basically runs two trials in the same group of patients. The analysis is a bit more complex and includes testing for an interaction between the two interventions.

Power analysis for noninferiority studies requires a couple of extra steps. Noninferiority studies are becoming much more common in the era of comparative effectiveness.[75,76] Their null hypothesis assumes that there is a difference between arms, whereas for the more common superiority trials, the null hypothesis assumes that there is no difference between groups. A noninferiority trial seeks to determine whether a new treatment is not worse than a standard treatment by more than a predefined margin of noninferiority for one or more outcomes (or side effects or complications). Noninferiority trials and equivalence trials are similar, but equivalence trials are two-sided studies, where the study is powered to detect whether the new treatment is not worse and not better than the existing one. Equivalence trials are not common in clinical medicine.

In noninferiority studies, the researchers must prespecify the difference they intend to detect, known as the noninferiority margin (also known as irrelevant difference or clinical acceptable amount based on literature).[75] Within this specified noninferiority margin, the researcher is willing to accept the new treatment as noninferior to the standard treatment. This margin is determined by clinical judgment combined with statistical factors and is used in the sample size and power calculations. For example, is a difference of 4% in infection rates between two groups large enough to sway your decision about antibiotics? Would a difference of 10% make you think they are different? Or do you need something smaller? These decisions are based on clinical factors, such as severity of the disease, and also on known variation in the outcomes.

The USA Multicenter Prehospital Hemoglobin-Based Oxygen Carrier Resuscitation Trial is an example of a dual superiority/noninferiority assessment trial.[77] The noninferiority hypothesis assumed that patients in the experimental group would have no more than a 7% higher mortality rate compared with control patients, based on available medical literature as well as the feasibility of the study. The implication of a noninferiority outcome in this study was that the blood substitute product would not be used interchangeably with available blood products; rather, it applied to scenarios in which blood products are needed but not available or permissible.

Reviews of the quality of noninferiority trials have shown major problems due to underreporting.[78,79] Essential elements to be included in the reports of this type of study include the following: (1) the noninferiority margin and its rationale; (2) whether the participants in the noninferiority trial were similar to those included in the previous trials that established the efficacy of the control treatment; (3) whether the control intervention is in fact similar to the intervention tested in efficacy trials; and (4) whether secondary outcomes are also being tested for noninferiority rather than superiority.[76]

We close this section with a final message about significance and power. We often hear the comment that a small study with a significant difference may lack power. This is incorrect. Once a study has a significant difference, questions about statistical power are irrelevant. The issue then is related to external validity or generalizability: Are the study findings applicable to the general population they are supposed to represent?

Association and Causation

Most studies describe associations between outcomes and effects of interest. Whether these associations represent a cause-effect relationship, or rather an association without causality, is often up for debate. To strengthen the case for causation, we can apply the set of nine criteria proposed by Sir Austin Bradford Hill in 1965 as a guideline.[80,81]

1. *Strength of the association:* Large effect sizes are more likely to be causal than noncausal associations (effect size, not *P* value). For example, in the initial studies on risk factors for multiple organ failure (MOF), there was a strong association between transfusions of red blood cells (RBCs) and postinjury MOF, which triggered further investigations on the specific causative, harmful role of RBCs.[82-84]
2. *Consistency:* The results have been replicated under different conditions by independent investigators.
3. *Specificity:* The effect of interest is associated with a specific outcome rather than a wide range of outcomes. Its presence can help the case for a causal effect, but its absence does not discard it, as most outcomes have multifactorial, interdependent causes.
4. *Temporality:* There is a temporal relationship in which the effect of interest clearly precedes the disease. Although

this may seem obvious, it is important that we take into account how the outcomes and the effect are measured. For example, although lung failure seems to precede liver and renal dysfunction in the development of postinjury MOF, lung function tests are more sensitive than clinically used liver and renal function tests. Pao_2/Fio_2 ratios may detect early, mild levels of pulmonary failure, whereas bilirubin and creatinine only rise after substantial organ derangement.[85,86] Thus, the temporal relationship may be unclear.

5. *Biological gradient or dose response:* Increasing/decreasing exposure is associated with increasing/decreasing risk of disease. This is a powerful criterion when measurements are accurate. In the previously mentioned studies on the association between RBC and MOF, a dose-response relationship was observed: larger numbers of RBC units transfused were associated with progressively higher MOF rates.[87] Moreover, we observed that judicious transfusion practices that drastically limited the amount of RBCs transfused were associated with a decrease in postinjury MOF.[84]
6. *Plausibility:* When there is a plausible scientific mechanism that can explain the association, the case for causation is strengthened. The lack of such mechanism does not eliminate causation, as new mechanisms may be discovered; however, if one already exists, it enforces the case for causation.
7. *Coherence:* The association is consistent with what is known about the disease. Of course, novel observations may not fit this criterion. Yet, there is no denying that if the reported finding does not fit with current knowledge, there is a tendency toward a healthy skepticism.
8. *Experimental evidence:* Hill[80] proposed that "causation is more likely if evidence is based on randomized experiments."
9. *Analogy:* In the presence of previous evidence of a causal effect by one class of agent (eg, RBC and MOF), we are more likely to accept causation when another agent from the same class (eg, plasma) is implicated as a risk factor for the same outcome. Again, as Rothman and Greenland[88] indicate, we must be careful in the application of this criterion: "Whatever insight might be derived from analogy is handicapped by the inventive imagination of scientists who can find analogies everywhere. At best, analogy provides a source of more elaborate hypothesis about the associations under study; absence of such analogies only reflects lack of imagination or lack of evidence."

These criteria are to be used as guidelines, as Sir Bradford Hill himself wrote: "None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a sine qua non."[80]

Bias

Physicians can appreciate the acronym CBC as a mnemonic for the three main reasons for a spurious association: **c**hance, **b**ias, and **c**onfounding. Chance is dealt with by statistical testing, whereas appropriate designs and analytic techniques can assist with eliminating or minimizing bias and confounding. We have dealt with the "chance" reason and statistical testing in previous sections. In this section, we will deal with bias and confounding.

Bias is the deviation of results due to systematic errors in the research methods. Although there are several different names for biases, two types seem to capture most biases presented in surgical literature: (1) selection bias, which occurs when the study groups differ systematically in some way, or when the study sample differs from the study population; and (2) observer/information bias, which occurs when there are systematic differences in the way information is collected for the groups being studied. The article by del Junco et al[89] on the seven deadly sins in trauma outcome research is an excellent review of some of the most common biases in trauma research.

There are several types of selection bias commonly found in the trauma literature. One of the most common is missing data not at random. Treatment of missing data has become the focus of much attention recently. Indeed, the FDA requested that the National Academy of Sciences create the Panel on Handling Missing Data in Clinical Trials to propose appropriate study designs and follow-up methods to reduce missing data and appropriate statistical methods to address missing data for analysis of results.[90] Although their focus was on RCTs, the report is very informative for other study designs as well.

The most important things to consider about missing data are (1) the proportion of patients with missing data and (2) whether the data are missing at random, thus not biasing the results in a significant way, or there is some pattern that can bias the results. Finding out that data are missing not at random (MNAR) does not mean that the study is automatically flawed; rather, appropriate statistical methods must be used to deal with them. However, if the proportion of missing data is high and MNAR, one cannot ignore it and proceed to analyze the complete data set without further consideration. Let us illustrate this with a little story: The father of one of the authors (A.S.) was a physician interested in congenital heart defects. He was conducting a population-based study about the incidence of such defects in school-age children and visited several schools, screening children for heart defects. Some children were, of course, absent that day and were not screened. At the end of the day, this young physician researcher considered the absent children (his missing data) for a moment and, wanting complete data, decided to visit them at home. Not surprisingly, some of the absent children did in fact have a heart defect. This made sense because these ill children were more likely to miss school because of symptoms or medical appointments.

Missing data not at random in trauma occurs for two radically different reasons: (1) patients are too sick to have the test (eg, died early, intravenous access not possible, chaotic trauma scene), in which case adverse outcomes are common; or (2) patients are not sick enough to justify the test (eg, early discharge, hemodynamically stable, not in shock, not

requiring mechanical ventilation), in which case adverse outcomes are rare. For example, in the late 1990s, we developed predictive models for postinjury MOF and observed that lactate was a significant independent predictor of MOF.[82,91] As expected, lactate measurement was available only for the group of severely injured patients. We addressed the missing data using two analyses: one included only patients for whom lactate was measured, and the second included all patients and used a "missing indicator" (ie, lactate levels were categorized into three possible categories: missing, normal, and abnormal). The results of the two analyses were remarkably similar and increased the strength of our findings. In a more recent example, Odom and colleagues[92] addressed this issue in their study on the value of lactate as a predictor of trauma mortality. They astutely observed that the selection bias created by the missing lactate values would bias their results toward the null hypothesis rather than the positive effect they found. Even existing national databases suffer from the bias created by missing data not at random. Thiels et al[93] showed that clinical data missingness was much greater in firearm incidents compared to other injuries in the NTDB. Lack of personnel prepared to gather information in the sensitive circumstances surrounding gun-related episodes is likely a major culprit.

Many methods are available to deal with missing data, such as last value carried forward. Albeit highly criticized, this technique has its place in longitudinal studies, when most variables are not missing and there is high predictability of missing values.[94] For example, we used this technique to input the values of daily liver function tests using the last obtained value until a subsequent result was available.[95]

More sophisticated techniques such as multiple imputation by chained equations (MICE) are becoming popular. Simply put, this method imputes missing values based on regression equations derived M times, followed by an analysis of each imputed dataset and finalized by a combination of the M analyses using standard methods. Although multiple imputation functions better for data missing at random, it has been shown to provide good estimates even in certain cases when data are MNAR. For example, Moore and colleagues[96,97] tested the use of MICE to impute values of the Glasgow Coma Scale in 2005, and again in 2015 for the Glasgow Coma Scale, respiratory rate, and systolic blood pressure for a model to evaluate quality of trauma care with good success.

Another important type of selection bias, especially in trauma and emergency care, is survivor bias. This occurs when the individual does not survive long enough to have the "opportunity" to receive the complete intervention. These early nonsurvivors contribute to increasing the mortality rate of the group not receiving the intervention, artificially inflating the effect of the intervention. The studies on fixed, balanced blood product ratios (1:1:1 RBC:plasma:platelets) are well-known examples of this problem.[89,98,99] Survival analysis, which analyzes time to event and allows for censoring patients who died before experiencing the intervention, is a helpful technique to deal with this problem. The best solution, however, to deal with survivor bias is an RCT. Indeed, the results of the PROPPR trial, an RCT testing the effect of a fixed, balanced blood product ratio failed to find a difference, despite excellent statistical power, contradicting the results of previous observational studies that found it beneficial.[18] Another problem with the blood product studies was the "catch-up" practice. For example, if a patient received 6 RBC units and 3 units of plasma in the first 3 hours and no blood products between hours 4 and 6, the RBC:plasma ratio at 6 hours is 2:1. This is exactly the same ratio at 6 hours as someone who receives 6 RBC units in the first 3 hours and 3 units of plasma from hour 4 to the end of hour 6 (the "catch-up" practice). The big difference here is that the first patient experienced the 2:1 ratio at all times, whereas the second patient had an initial ratio of 3:0 followed by a ratio of 0:3. It is quite possible that the outcomes are different for these two extremes. Using a time-varying covariate in a survival analysis (ie, a variable that changes over time), we can actually express the changes in RBC:plasma ratio hourly.

Some authors deal with survivor bias by excluding early deaths from the analysis. This may be a solution, but it limits the generalizability, because the study findings apply only to patients who survive the acute postinjury period. Other types of commonly encountered selection biases include loss to follow-up not at random (eg, patients failed to return to follow-up visits due to death or long-term injury-related complications), refusal to participate or withdrawal due to side effects or invasiveness of the intervention, and consent not obtained due to traumatic brain injury, among others. This is a good reminder to always read the inclusion and exclusion criteria to determine whether they resulted in selection bias. Common exclusion criteria that may limit the generalizability of the investigation are advanced age, comorbidities, early deaths, and incomplete data. It is important to realize that the findings apply only to the population that fits both the inclusion and exclusion criteria.

Confounding

Now we will address the final C in the CBC acronym: **c**onfounding (ie, a third variable responsible for all or part of the association between two other variables). An often-quoted example is the association between coffee drinking and lung cancer, which is confounded by the real culprit, smoking, a variable associated with both coffee drinking and lung cancer. To be considered a confounder, a variable must be associated with both the outcome and the effect of interest (ie, a risk factor or an intervention). In trauma, injury severity, age, and comorbidities are frequent confounders of the association between an intervention and the outcome.

There are two ways to deal with confounders: (1) research designs, including RCT or matching; and (2) analytic tools, such as stratified analysis (when there are few confounders) or multivariate analysis (in the case of multiple confounders). These techniques will be discussed in a subsequent section of this chapter. Usually, the "table 1" in RCT reports the distribution of potential confounders between the control and experimental groups.

Appropriately so, readers should not find *P* values in this table, as any differences in the distribution of these confounders are, by definition, a result of chance. Randomization increases the likelihood of similar distribution of confounders, which then are not associated with group membership, allowing us to assess the effect of the intervention (the main difference between the two groups) on the outcome. Single blinding (only the patient) or double blinding (the patient and the researcher) are powerful add-ins to improve the likelihood that the researchers' and patients' own biases do not interfere with the design. Surgical and emergency interventions, however, are often not amenable to blinding.[100]

Matching is an alternative option to deal with confounders. This can be accomplished via traditional matching when only a few known risk factors exist or via PSM, which we mentioned in a previous section. In PSM, a multivariate model including potential reasons for receiving the intervention is used to derive a propensity score (ie, the probability of receiving the intervention). In this model, the "outcome" or "dependent variable" is the intervention. Using this propensity score, we can choose matching control patients (ie, patients who did not receive the intervention despite having the same propensity score as the patients who received the intervention). The downside of this procedure is that the reason why patients did not receive the intervention despite having the same propensity score is often unknown. In addition, the PSM accounts for measured variables, not for unmeasured variables (which an RCT accounts for). Finally, matching limits the ability of the investigators to examine the effects of the matching variables; once used in the matching, a variable is no longer available for analysis. Therefore, it is important that the investigators are certain that the matching variable is of no interest in the analysis of the outcomes.

Interactions and Effect Modification

In confounding, variable A is responsible for part or all of the association between variables B and C, and we want to adjust for it (in other words, minimize its effect) to be able to assess the association between B and C. There is little or no interest in any effects mediated by variable A. Conversely, in effect modification or interaction, variable A modifies the association between B and C. This type of association must be described, not adjusted for, as it provides important information of the mechanism underlying the association of B and C. Thus, when appraising multivariate models, the reader should make sure pertinent interactions were tested and, if significant, described appropriately.

An example of an interaction can be found in our study on the effect of preinjury antiplatelet therapy on postinjury outcomes. We showed that antiplatelet therapy modified the relationship between blood product transfusions and mortality.[101] Specifically, as shown in Fig. 7-1, among patients who were taking antiplatelet therapy prior to the injury, the odds of mortality associated with RBCs transfused were lower than among patients not receiving this medication.

Descriptive Statistics

Descriptive statistics, such as mean and standard deviation, median and interquartile range, frequency and percentages, are used to provide the reader with the best possible description of the sample. Because the reader does not have access to the raw data, it is up to the authors to provide readers with a clear picture of what the sample looked like. More important, this picture should allow the readers to use the PICO framework and compare the study's sample to the population

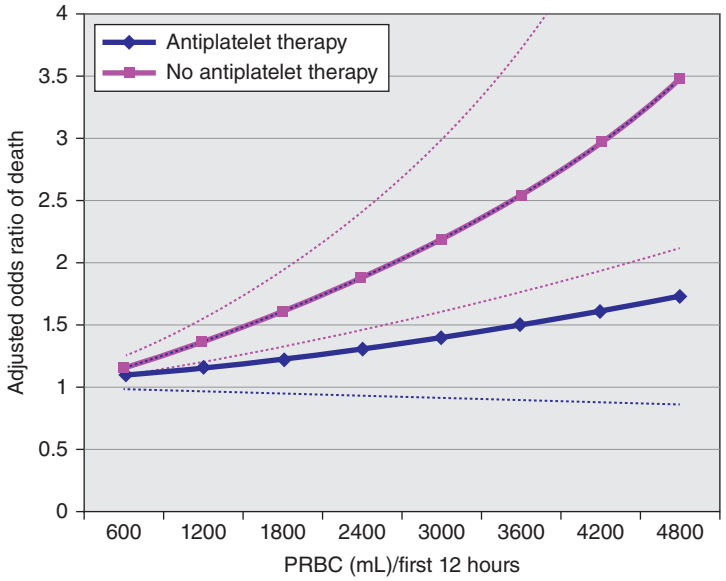

FIGURE 7-1 Example of an important effect modification (or interaction) in the study by Harr et al[101] investigating the effect of preinjury antiplatelet therapy on postinjury outcomes. These investigators showed that antiplatelet therapy modified the relationship between blood product transfusions and mortality, decreasing the risk associated with requirement for transfusions. PRBC, packed red blood cells.

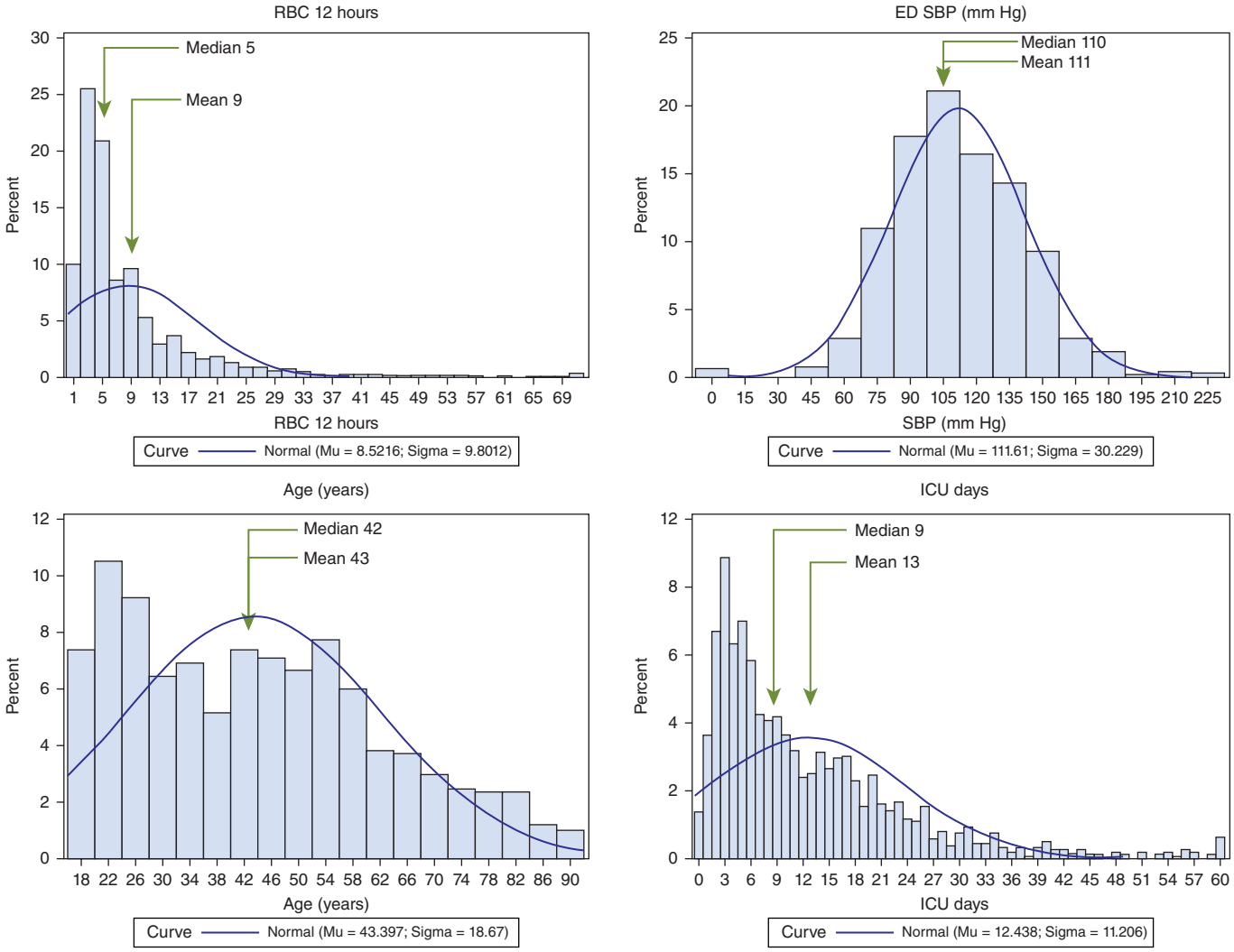

FIGURE 7-2 Distribution of variables commonly reported in trauma and surgery research. Note how means and medians differ in intensive care unit (ICU) days and RBC 12 hours (number of red blood cell units in the first 12 hours after injury), which are skewed, compared to age or emergency department (ED) systolic blood pressure (SBP), which are closer to normal. (Data from the Glue Grant; analysis by the authors.)

to whom they intend to apply the findings. Are they similar enough that one may directly apply the findings, or are they older, younger, more severely injured, etc?

Categorical variables such as sex and blunt versus penetrating mechanism are expressed as frequency (numbers) and percentages. Continuous, numeric variables, such as age, ISS, Glasgow Coma Scale, length of stay, ventilator time, and so on, are usually described by means or medians. The decision to use mean or medians can be tricky. When the variable distribution is normal and symmetric, medians and means are identical. However, when the variable is skewed or there are outliers, medians, rather than means, are better descriptors. As shown in Fig. 7-2, which illustrates data from a multicenter prospective study of severely injured patients (Glue Grant), the distributions of systolic blood pressure at the emergency department and age were approximately normal; thus, the median and mean both reflect the "typical patient." However, the mean does not describe well the typical number of RBC units transfused in the first 12 hours or the length of stay in the intensive care unit. Those are skewed variables, with most patients presenting low values and a few patients presenting with high values; thus, the median will provide a better description of the "typical patient."

Data dispersion for means is usually represented by the standard deviation (68% of the data should be contained within mean ± 1 standard deviation, 95% of the data within mean ± 2 standard deviations), whereas for medians, we most commonly use the interquartile range (the lower and upper quartile; 50% of the data are contained within this interval) or the range (maximum and minimum values). A box plot, shown in Fig. 7-3, is an excellent way to represent the data central tendency and dispersion. One may ask why not use median all the time. Especially in economics, one may be most interested in the outliers (eg, the few outlier patients

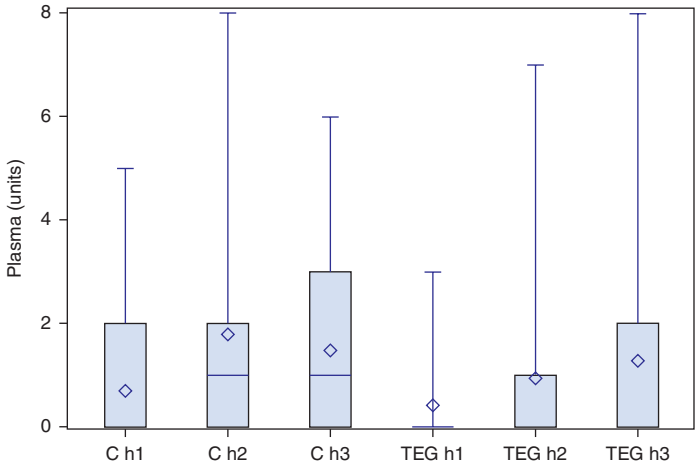

FIGURE 7-3 Example of a box plot showing the amount of plasma (fresh-frozen plasma) units given in the first (h1), second (h2), and third hour (h3) after injury in two study groups: resuscitation guided by conventional coagulation tests (C) and resuscitation guided by thromboelastography (TEG).[54] The length of the box represents the interquartile range (the distance between the 25th and 75th percentiles); the symbol in the box interior, the group mean; the horizontal line inside the box interior, the group median; and the vertical lines ("whiskers"), the minimum and maximum values (range). Note the difference in mean and median, suggesting skewness.

with longer hospital stay drive the cost; thus, the mean becomes a relevant measure).

Inferential Statistics

Studies will usually present initially bivariate analysis (sometimes called univariate). This consists of an unadjusted, crude comparison between two (or more) groups of subjects. In RCTs, this is the only comparison needed if randomization was successful (and usually it is) in distributing the confounders evenly across groups. In observational trials, the main point of this bivariate (or univariate) analysis is to detect confounders, which are variables with different distribution between the groups (thus associated with group membership) and also associated with the outcome. This difference does not need to be statistically significant, as the sample may be small and thus prone to a type II error. Deciding whether a variable may be playing a confounder role is a clinical, not a statistical, decision. After examining this information, the reader can decide which variables are potential confounders for which the researchers (hopefully) adjusted the subsequent analyses. An important differentiation here is between a confounder and a mediator variable. A mediator (eg, hyperfibrinolysis), different than a confounder, is in the pathway between the effect of interest (eg, traumatic hemorrhagic shock) and the outcome (ie, early mortality). If the mechanism by which hemorrhagic shock causes mortality involves hyperfibrinolysis, then hyperfibrinolysis is not a confounder; rather, it is a mediator. We do not adjust for mediators. As another example, in the hypothesis that the gunshot wound case-fatality rate has increased over the past decade, the association between the effect of interest (gunshot wounds) and the outcome (case-fatality rate over time) has a mediator (and not a confounder), which is injury severity (measured, for example, by the New ISS). Injury severity is how gunshot wounds cause death (ie, it is in the causal pathway). Potential confounders of this association are age (because older individuals would be more likely to die than younger individuals and age is increasing over time) and comorbidities (which also increase likelihood of death and become more common over time). Thus, we do not adjust the analysis for injury severity, but we adjust for age and comorbidities.

Analytic techniques that minimize confounding are stratification and multivariate analysis. Stratification may work when there are just a few risk factors and a large sample size. For example, in order to account for the confounding effect of smoking in the association between coffee drinking and lung cancer, one may stratify the analysis on smoking status and observe whether the association between coffee drinking and lung cancer differs by strata. We should observe that among smokers and nonsmokers alike, coffee drinking's association with lung cancer is not significant. Multivariate analyses are basically an advanced form of stratification done on multiple variables. There are several types of multivariate models depending on the distribution of the outcome. Binary outcomes (eg, death [yes or no]) are commonly addressed using logistic regression. Categorical outcomes with more than two strata can be analyzed with polytomous (or multinomial) logistic regression. When time to event is of interest or there is need to censor data (eg, patients who died or are discharged before experiencing the outcome of interest), survival analysis is an option.

Linear regression assumes that the outcome is continuous, has a distribution not too far from normal, and has, as the name says, a linear relationship with the covariates. When these assumptions do not hold (eg, outcome is categorical or too skewed), then we may apply a larger category of models, named *generalized* linear models. Please, note that there is a difference between *generalized* linear models and *general* linear models. The economic analysis by Schwartz and colleagues[102] on delays in laparoscopic cholecystectomy is an example of a study using a generalized linear model. The generalized linear

models are a broad class of models that include the logistic regression, the Poisson regression, log-linear models, gamma distribution models (as used in the previously mentioned article), and others.

The description of generalized linear models usually includes the word *link*, which is a function linking the actual outcome Y to the estimated Y in a model. Put simply, it is the transformation done to the outcome variable to convert it to continuous. In a linear regression model, the link is the identity (ie, the estimated and the actual outcome are expressed the same), and no transformation is needed. In a logistic regression, the transformation or link used is called the logit and the distribution is binomial (ie, yes or no type of variable). For the gamma model (used in the publication by Schwartz et al[102]), the link is the log, and the distribution is right-skewed with a variation that increases with the mean. This type of model is often used in econometrics because it fits the distribution of cost in health care (ie, care for most patients results in little costs, but a few patients require very costly treatments). In the log-linear and the Poisson regression, the link is a log and the distribution is the Poisson distribution. Poisson regression is usually applied to count data; for example, number of trauma deaths over a period of time, as seen in a 2013 article by Kahl et al[103] assessing time trends in annual trauma mortality rates.

Each multivariate model has its own set of assumptions, and although many are robust to small violations, this is an important consideration when appraising the article. For example, the Cox proportional regression model, a type of survival analysis, requires, as the name says, that the risks are proportional (ie, do not vary over time). This type of violation (ie, variation over time) is not, however, an insurmountable problem: when this model is used, we should verify whether the authors tested the proportionality assumption and, if violated, whether they reported remedying it (eg, by introducing time-varying covariates in the model).

POLYNOMIALS

Another assumption in regression models relates to the shape of the association between the outcome and the risk factor. Is it linear? If linear, is it a straight line or U-shaped? The computer software used for linear regression will draw a straight line, unless we add what is called a quadratic term (or second-order polynomial), in which case it will draw a U-shaped line (the U can face up or down). For example, in a recent study on fibrinolysis, we showed that the association of fibrinolysis and mortality was U-shaped, with higher mortality associated with very low (shutdown) and very high (hyperfibrinolysis) levels of fibrinolysis and a mortality nadir with moderate fibrinolysis levels.[104] Interestingly, we showed a similar U-shaped relationship between RBC:plasma ratios and mortality, with highest mortality peaking with both low and high ratios and lowest mortality associated with medium ratios.[105] More complex forms, such as those achieved by introducing third- or higher-order polynomials, are rarely found in the literature as they complicate the interpretation.

CLUSTER EFFECTS

Most multivariate models assume independence between observations. In other words, if one cannot predict a subject's outcome based on the outcome of other subjects, their outcomes are said to be independent. This is not completely true for patients within the same center or even treated by the same surgeon. Patients within a center tend to have similar outcomes, violating the independence assumption. This similarity between patients in the same center or treated by the same provider is called a cluster effect, and it should be accounted for in the statistical modeling and in sample size calculations. The larger the correlation between subjects within a cluster, the larger is the required sample size. This correlation can be assessed by the intraclass correlation coefficient, which measures how similar patients are within centers.[106]

MODEL PERFORMANCE

Another important issue in multivariate models is their performance (ie, how well they fit the data), which can be evaluated in several ways, depending on the model. Indeed, a model is only as good as its performance, and with rare exceptions, all multivariate models should be accompanied by performance measures in an article. The *R*-square, an often-reported measure of model performance of linear regression, gauges the percentage of the variation of the outcome that is explained by the independent variable(s) in the model. We must keep in mind that in clinical studies, given the multifactorial nature of most diseases and clinical scenarios, it is uncommon to see large *R*-squares (>0.30). The *R*-square should be accompanied by a *P* value that provides the probability that the observed *R*-square was due to chance. Together, the *R*-square and its correspondent *P* value compose an assessment of the model's performance. For example, Rourke et al[107] predicted admission fibrinogen levels with a multiple linear regression model including injury severity, base deficit, prehospital fluid resuscitation, and systolic blood pressure; the *R*-square for this model was 0.27, suggesting that 27% of the variation of the postinjury fibrinogen upon hospital admission was explained by the model variables. Although the *R*-square is a measure of performance for linear models, "pseudo" *R*-square exists for other models.

Model discrimination (ie, the ability of the model to discriminate individuals with and without the outcome; eg, survivors and nonsurvivors) is often evaluated for binary outcomes by the area under the receiver operating characteristic curve (AUROC; also known as the c-statistic). Receiver operating characteristic (ROC) curves were derived from studies on radar and sonar detection during World War II to ascertain the best radar setting to distinguish enemy airplanes from harmless targets (eg, flocks of birds). As you can see in Fig. 7-4, which shows the AUROC for thromboelastography values as predictors of massive transfusion, the *y*-axis depicts the sensitivity (eg, percentage of all deaths that were predicted), whereas the *x*-axis depicts [1 – specificity] (eg, [1 – percentage of survivors correctly predicted to survive]). The AUROC is a good assessment of the accuracy of the

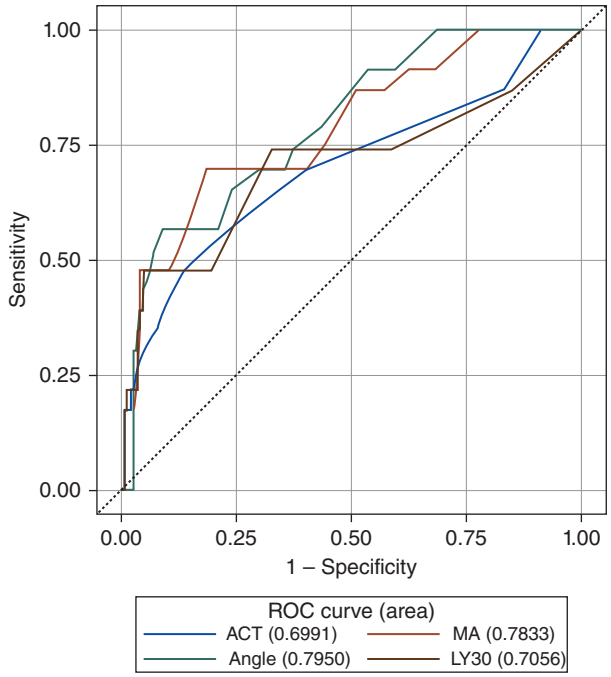

FIGURE 7-4 Example of a receiver operating characteristic (ROC) curve to compare the predictive power of the thromboelastography values (activated clotting time [ACT]; maximum amplitude [MA]; angle; and fibrinolysis [LY30]) for massive transfusion defined as ≥4 units of red blood cells in the first hour after injury (excludes deaths within 1 hour after injury). The areas under the ROC curve were as follows: angle, 0.79 (95% confidence interval [CI], 0.69–0.89); MA, 0.78 (95% CI, 0.68–0.89); LY30, 0.71 (95% CI, 0.56–0.85); and ACT, 0.70 (95% CI, 0.57–0.83).[108] Note that the 95% CIs overlap, suggesting no difference in prediction performance.

model, and it has been arbitrarily categorized as follows: 0.90 to 1 = excellent; 0.80 to 0.90 = good; 0.70 to 0.80 = fair; 0.60 to 0.70 = poor; and 0.50 to 0.60 = fail. AUROCs should be accompanied by 95% CIs to allow the reader to determine whether they are significantly different than the 0.50 no-value AUROC and to compare different AUROCs (if 95% of CIs overlap, the AUROCs are not significantly different). In another example, our group conducted a comparison of two MOF scoring systems (Denver MOF score and the Marshall Multiple Organ Dysfunction Score [MODS]) as predictors of death using AUROCs.[108] The Denver MOF score's AUROC was 0.88 (95% CI, 0.84–0.92), whereas the Marshall MODS's AUROC was 0.86 (95% CI, 0.82–0.89), suggesting that both scoring systems performed similarly well.

The ROC curve can also be used to determine the best threshold to discriminate between normal/abnormal values for a continuous variable. There are several methods to define the best cutoff, such as the Youden Index (also known as the Youden J statistic), which is defined as J = Sensitivity + Specificity – 1 and ranges from 0 (no discrimination) to 1 (complete discrimination). The Youden Index corresponds to the maximum vertical distance between the ROC curve and the diagonal, noninformative line. Another common measure is the upper leftmost point, which often (but not always) coincides with the Youden Index. Although these points represent an optimal combination of sensitivity and specificity, researchers may decide to privilege sensitivity or specificity depending on the condition. Certain conditions demand more sensitivity (eg, when one does not want to miss a case due to potential disastrous consequences), whereas others may require more specificity (eg, when the consequences of missing a case are minimal, but the cost of the test is high). In Fig. 7-4, the upper leftmost point in the ROC for angle was 65° and the Youden Index was 60°; thus, researchers may choose a cutoff between these two values to define a cutoff that is predictive of the need of massive transfusion. Tests or biomarkers with good sensitivity and specificity can disappoint clinicians when used in clinical practice, because they may present low predictive value. Predictive value is a function of prevalence; thus, predictors of relatively rare outcomes may show low predictive value, despite good sensitivity and specificity.

Calibration is another important assessment of model performance. It gauges the model's ability to correctly estimate the probability of an event. It is commonly assessed using the Hosmer-Lemeshow statistic, which compares the predicted and observed rates within deciles. In this statistic, the larger the *P* value (ie, the lower likelihood of disparity between predicted and observed rates), the better is the model's calibration. It is important to be careful, however, with sample sizes, when the *P* value can be small (ie, significant) for models that are not poorly calibrated.[109]

When assessing models, we should also contemplate whether there was overfitting (ie, the model has more variables than it can handle). This may seem counterintuitive: Is it not appropriate to adjust for all possible confounders? Yes, but only if you have the necessary sample size to do that. If one imagines a multivariate model as multiple stratifications by the various confounders, it is not difficult to see that in small samples some of these stratifications will end up having very small numbers or even zeros. It is important to note that the multivariate model needs subjects with and subjects without the outcomes. When we say we need large samples to model several confounders, we mean that we need a large enough number of subjects both with and without the outcome. A good rule of thumb, used even by expert statisticians, is 10 subjects with the outcome per variable in the model (not 10 subjects per variable, but 10 subjects with the outcome). When the number of confounders is higher than this threshold, an alternative is to use the previously mentioned propensity score, this time as an adjustment. Thus, instead of 20 variables, one has only one propensity score, representing the combined effect of the 20 variables. For example, Brown and colleagues[110] used this approach in a study on the scope of prehospital (PH) crystalloids in patients with and without PH hypotension. A propensity score was used to adjust the mortality comparison between the group receiving high versus low volumes of PH crystalloids. Instead of using five covariates (PH time, PH blood, PH systolic blood pressure, ISS, and initial base deficit), the authors had a single variable,

a propensity score ranging from 0 to 1. A final note on overfitting is that overfit models may have good performance for the data set from which they were derived, but because they address all the peculiarities of the derivation sample, they are unlikely to perform as well in other data sets.

There is controversy regarding whether multivariate adjustment for confounders is better than propensity score.[111] The answer seems to be that it depends. When the sample is large (>8–10 patients with the outcome per confounder), the multivariate method provides more information because one can assess the effect of individual confounders. Conversely, the propensity score is better when sample is limited and there are less than 8 to 10 patients with the outcome per confounder.

When using a multivariate model for confounder adjustment, it is important to compare the unadjusted effect and the adjusted (with confounders) effect. For example, if the unadjusted RR of a given variable is 2.0 (95% CI, 1.5–2.5) and the same variable, after adjustment of confounders, shows an adjusted RR of 1.2 (95% CI, 0.8–1.6), one can conclude that confounders were responsible for the effect seen in the unadjusted analysis.

Principal Components, Latent Class, and Cluster Analyses

So far, we have discussed how to analyze values of specific variables. Sometimes, however, researchers need to analyze the variables themselves. This is especially true when there are numerous variables and researchers may wish to reduce the number of variables, or when they want to examine patterns and combinations of variables using factor analysis. A special case of factor analysis is principal component analysis (PCA), in which the factors or components are combinations of variables that do not correlate, thus representing independent components. Two examples in the trauma literature may help illustrate the use of PCA. In the first example, the San Francisco group used PCA to define three uncorrelated groups of coagulation assays (prothrombin; factors V, VII, VIII, IX, and X; D-dimer; activated and native protein C; and antithrombin III levels): component 1 was defined as global clotting factor depletion, component 2 corresponded to the activation of protein C and fibrinolysis, and component 3 corresponded to factor VII elevation and factor VIII depletion.[112] The authors reported that component 1 predicted mortality and international normalized ratio/partial thromboplastin time elevation, whereas component 2 (fibrinolytic coagulopathy) predicted infection, end-organ failure, and mortality. A second example is seen in a similar study by our Denver group, this time using thromboelastography values.[113] As shown in Table 7-2, PCA generated a number of components, each of which contained a number of variables, each one associated with a factor loading (which can be interpreted much like a simple correlation coefficient, ranging from –1 to 1). Together these three components were responsible for 93% of the variation in the data. Component 1 included K (coagulation time), angle, maximum amplitude, maximal rate of thrombus generation, and total thrombus generation; component 2 included activated clotting time and time to maximal rate of thrombus generation; and component 3 reflected fibrinolysis. Taken together, both studies support the conclusion that trauma-induced coagulopathy has distinct, independent mechanisms, which may require tailored hemostatic therapeutic approaches.

TABLE 7-2: Principal Component Analysis (using thromboelastogram [TEG] values in trauma patients)[113]

Composition of principal components (PC)

	PC 1	CP 2	PC 3
% variance explained by component	63%	17%	13%
Activated clotting time	–30	90	6
K	–95	15	–4
Angle	92	–26	5
Maximum amplitude	95	–15	–10
% lysis at 30 min	–4	0	99
Time to maximal rate of thrombus generation	–13	95	–5
Maximal rate of thrombus generation	94	–23	3
Total thrombus generation	94	–14	–16

K, coagulation time.

Source: Modified with permission from Chin TL, Moore EE, Moore HB, et al. A principled component analysis of postinjury viscoelastic assays: clotting factor depletion versus fibrinolysis. *Surgery.* 2014;156(3):570-577.

PCA also has assumptions, including normal distribution and more subjects than variables. Therefore, this technique may not be applicable to many of the "omics" studies (eg, genomics, proteomics, and metabolomics), where the number of variables far exceeds the number of subjects. In this case, an option is the primary latent structures (PLS; formerly known as partial least squares) analysis, commonly followed by a discriminant analysis (PLS-DA). For example, in a metabolomics analysis of human plasma and lymph in deceased trauma patients, D'Alessandro et al[114] used this technique to group (ie, reduce) 215 metabolites with similar variance regarding the outcome (ie, lymph vs plasma). These groups of metabolites are named components or latent variables, which can then be used to predict outcomes. An examination of the combinations can reveal the commonalities between the members of a component; for example, a latent variable may well represent specific pathways (eg, glycolysis), or using specific measures, such as the variable importance in projection, can help identify the variables that contributed the most to groups' separation (eg, succinate), which can then be targeted for specific research. An excellent, very didactic review on the analytic approach for metabolomics was published in 2018 by Jayaraman and colleagues.[115]

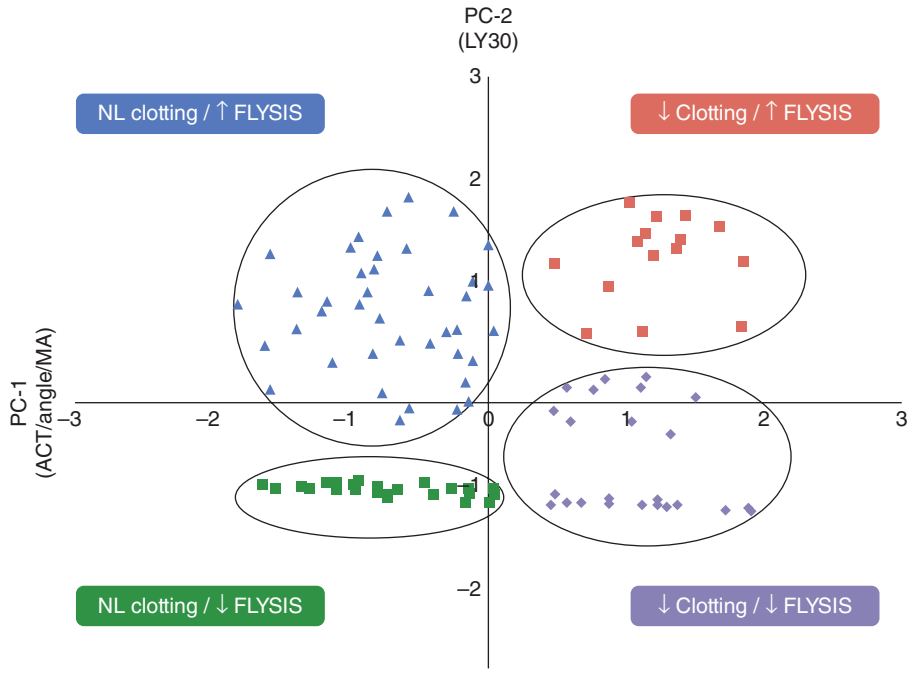

FIGURE 7-5 Cluster analysis: trauma patients plotted according at activated clotting time (ACT)/angle/maximum amplitude (MA) (*x*-axis) and fibrinolysis (FLYSIS; *y*-axis). NL, normal; PC, principal component.

Latent class analysis is a technique used to identify subgroups within a population. For example, Calfee et al[116] defined two subphenotypes within acute respiratory distress syndrome (ARDS) using this technique. The two subphenotypes within ARDS differed by magnitude of inflammation, shock, and metabolic acidosis and also by clinical outcomes. Moreover, the response to treatment also differed on the basis of subphenotype.

Cluster analysis groups subjects into groups or clusters based on their responses. One can use the factor or components derived from the PCA or the PLS to define the clusters. Cluster analysis is not really a statistical tool; rather, it is a mathematical model to group subjects based on their responses. In a recent example, the Denver group used cluster analysis to group patients according to PCA-derived thromboelastography components (Fig. 7-5). Patients in cluster 2 (hypocoagulable with no fibrinolysis) were more severely injured (higher ISS) but had a lesser degree of shock compared to patients in clusters 3 and 4. Patients in cluster 4 required significantly more blood products and had an increased hemorrhage-related mortality compared to the other groups.

SYNTHESIZING EVIDENCE

Systematic Reviews and Meta-Analysis

A systematic review (SR) collects, appraises, and collates all evidence addressing a specific research question by using explicit, reproducible, systematic methods. SRs were created to assist professionals involved in medical and health policy decisions in managing the daunting amount of information regarding heath care. If the reader is naïve about a topic, a good-quality SR is the essential first step to begin to understand that topic. The Cochrane Collaboration has led the field by establishing the Cochrane Library, one of the most important, useful, and regularly updated SR collections (available at http://www.cochranelibrary.com/; accessed July 6, 2018). Their use of rigorous methods assures the reader that available evidence has been collected in a manner that minimizes bias and informs medical decision making. In addition, the *Cochrane Handbook for Systematic Reviews of Interventions* contains methodologic guidance for anyone preparing or appraising SRs.[117]

Some SRs are accompanied by a meta-analysis (MA), in which the results of the SR independent studies are combined with the goal of deriving more precise estimates than each individual study.[117] Well-done SRs/MAs carefully examine the consistency of evidence and explore the differences across studies. As explained in more detail later in this chapter, an MA requires reasonable homogeneity between the studies. Many SRs/MAs are now registered in the PROSPERO, an international registry of SRs with a searchable database (available at http://www.crd.york.ac.uk/PROSPERO/; accessed July 7, 2018). As of September 7, 2018, a simple search using the term *injury* listed 3240 records of SRs completed or in progress.

To make this as practical and applied as possible, we will examine these concepts using a 2015 Cochrane Library SR/MA, "Blood-Clot Promoting Drugs for Acute Traumatic Injury," produced by the Cochrane Injuries Group based in the London School of Hygiene and Tropical Medicine.[118] This was the third update of this review of RCTs assessing the effects of antifibrinolytics in trauma patients; the first was published in 2004 and the second in 2012, all available from the same website. Although this review was restricted

to RCTs, there are SRs that include other types of studies. The search strategy included a variety of databases (PubMed, EMBASE, Cochrane Injuries Group Specialised Register & Cochrane Central Register of Controlled Trials, ClinicalTrials.gov, WHO International Clinical Trials Registry Platform, and others). In addition, as it is common, the authors checked all references in the identified trials and background papers and contacted study authors and pharmaceutical companies to identify relevant published and unpublished data.

The search for unpublished data is essential to minimize publication or reporting bias, which occurs when the dissemination of research findings is influenced by the nature and direction of results. In general, this affects negative (nonsignificant) studies, which are less likely to be published and, when published, are less likely to appear in a high-impact, English-language journal. In addition, publication bias should be further assessed by using funnel plots, which are scatterplots of the intervention effects from individual studies usually plotted on the horizontal axis and some measure of study size in the *y*-axis (eg, sample size, standard error, variance). The effects from small, less precise studies will spread widely at the bottom of the graph, whereas larger studies' effects will cluster together at the top. Unfortunately, our example did not contain a funnel plot, because there were too few studies. However, we refer the reader to the excellent examples in the *Cochrane Handbook for Systematic Reviews of Interventions*.[117] In the presence of publication bias (eg, unpublished small negative studies), the funnel plot will have an asymmetrical appearance, and a combined effect derived in the MA may overestimate the treatment's effect. It is important to mention that there are other reasons for asymmetry, such as trials of lower quality, which overestimate effects.[119]

Our example SR was not restricted by publication date or language, but some reviews limit searches to articles in the English language, which can create substantial bias. The terms used for the search are found in their Appendix 1 and can be useful to guide the readers' own searches. Our example's first figure shows that of 1371 records plus four trials already identified in previous versions of this SR, three RCTs were finally included in the MA. This information educates the reader about the amount of research available in the area, and it also provides information on trials still being conducted.

In an SR, each study's risk of bias (ie, quality) should be appraised; in the previous example,[118] the GRADE system, which is described in more detail later in this chapter, was used for this purpose. The risk of bias was considered low (high quality) for outcomes on mortality, need for further surgery, and blood transfusion, whereas the quality was considered moderate for the vascular occlusive outcomes (including heart attacks, deep vein thrombosis, stroke, and pulmonary embolism).

Many SRs will stop here and derive some qualitative conclusion from the summative body of evidence. Others, such as the previous example, will move forward to combine the results of the independent studies in an MA. As mentioned earlier, before combining studies, their statistical and clinical heterogeneity must be assessed. Statistical heterogeneity (ie, variability in intervention effects) is usually assessed using the I^2 statistic, which describes the percentage of total variation across studies due to heterogeneity rather than chance. The I^2 statistic should be accompanied by 95% CIs or a chi-square test, for which a larger *P* value indicates less heterogeneity. When statistical heterogeneity is high (I^2 >50%), an MA is usually inappropriate. In addition to statistical heterogeneity, clinical heterogeneity (variability in designs, populations, measurement of outcomes, etc) is also an important consideration.

The studies included in the postinjury antifibrinolytic SR/MA showed no evidence of statistical heterogeneity (I^2 = 13%, *P* = .32). Therefore, based on the pooled data, the authors concluded that these agents reduced all-cause mortality from 16% to 14.5% (RR, 0.90; 95% CI, 0.85–0.96; *P* = 0.002). However, we should note that one of the three included studies (CRASH-2[72]) was responsible for 98% of the data, thus likely exerting major influence in the final result; the second study[120] focused on traumatic brain injury only, and the third study assessed aprotinin[121] (the other two used TXA) in 77 participants with major skeletal trauma and shock. We wonder whether the clinical heterogeneity of these studies should not preclude pooling the data.

In another example of conflicting clinical and statistical heterogeneity, Natanson and colleagues[122] in an SR/MA on blood substitutes combined 16 trials and concluded that these products were associated with a statistically higher risk of death (RR, 1.30; 95% CI, 1.05–1.61) and of myocardial infarction (RR, 2.71; 95% CI, 1.67–4.40). However, the SR/MA encompassed different populations (elective surgery, trauma, acute care surgery, and ischemic stroke patients), different controls (fluids, blood products), and different blood substitutes. Although the statistical heterogeneity among these studies was not significant for either mortality or myocardial infarctions (for both, I^2 = 0, *P* ≥.60), conclusions based on a mix of studies differing at so many design and clinical levels must be taken with caution.[123]

A final note, heterogeneity is not a failure, rather it is an important finding that should be further explored and described. Understanding the differences between studies addressing the same topic can provide more information than inappropriately combining them to increase sample size.

When Studies Collide

It is not rare that we find articles, sometimes in the same issue, with conflicting results. How should we decide between them? Again, to make this practical and applicable, we can invoke two examples of these "collisions." In the May 2013 issue of the *Journal of Trauma and Acute Care Surgery*, two articles[29,124] explored the role of crystalloids in early trauma resuscitation. Both studies used data from the same database (Inflammation and the Host Response to Injury, also known as Glue Grant) and arrived at seemingly disparate conclusions. The accompanying editorial by Dr. David Hoyt was able to reconcile these differences, expertly navigating the reader through the two research studies and coming up with

a unified message.[125] Dr. Hoyt highlighted that the exclusion of patients who died within the first 48 hours in the study by Kasotakis et al[124] resulted in a dramatically different population than the study by Brown et al.[29] Basically, the message of both studies was that fluid resuscitation should be guided by blood pressure and oxygen delivery to avoid the harmful effects of excess fluids.

In a second example, in March 2014, we published a comprehensive review on the temporal trends of postinjury MOF using the previously mentioned Glue Grant database and concluded that the incidence of postinjury MOF decreased over time, while the MOF case-fatality rate remained stable.[95] Conversely, in April 2014, Fröhlich et al,[126] using the Trauma Register DGU of the German Trauma Society, reported a significant increase in MOF incidence but a decrease in case-fatality rate. We were left with two apparently disparate messages. How can they be reconciled so the messages can be appropriately translated to our clinical practice and/or advance our research agenda? As astutely done by Dr. Hoyt in the previous example, we invoked the PICO framework to determine to which population each study specifically applied and also to ensure that the outcomes (ie, postinjury MOF) were indeed similarly measured. Once this was done, the differences were glaring. The entry criteria for the two studies were different: the patients enrolled in the Glue Grant study were more severely injured (ie, blunt torso trauma with hemorrhagic shock; all required at least 1 unit of RBCs/12 hours) than the German patients (24% required 1 unit of RBCs between hospital arrival and intensive care unit admission). The German study population included a large proportion of traumatic brain injury victims (59%), whereas the Glue Grant study specifically excluded these patients. Second, the two studies used different definitions of the outcome (postinjury MOF). The German investigators employed the Sequential Organ Failure Assessment score, which assesses the dysfunction of six organ systems including the central nervous system (CNS). In contrast, the Glue Grant investigators used the Denver MOF score, which does not assess the CNS, and a modified version of the Marshall MODS without its CNS component. Thus, the two studies apply to different populations and to somewhat different outcomes.

Guidelines and Recommendations

According to the Institute of Medicine 2011 report, "clinical practice guidelines are statements that include recommendations intended to optimize patient care that are informed by a systematic review of evidence and an assessment of the benefits and harms of alternative care options."[127] This report established standards for the rigorous development of guidelines including the following:

- Based on systematic review of the evidence
- Led by a knowledgeable, multidisciplinary expert panel and representatives from key affected groups
- Considers relevant patient subgroups and patient preferences
- Based on an explicit and transparent process that minimizes distortions, biases, and conflicts of interest
- Explains the association between care options and health outcomes
- Rates both the quality of evidence and the strength of recommendations
- Reconsidered and revised as appropriate

The GRADE, mentioned earlier in this chapter, is widely adopted as a transparent, systematic process to appraise the quality of the summative body of evidence and define the strength of the recommendation.[8] We will use a recent example of a guideline presented at the Eastern Association for the Surgery of Trauma (EAST) meeting and subsequently published in the *Journal of Trauma and Acute Care Surgery*. Fox et al[128] addressed the evaluation and management of blunt traumatic aortic injury (BTAI). They formulated several pertinent questions using the PICO framework, of which we will follow the process for PICO question 2: In patients with BTAI (P), should endovascular (I) repair be performed versus open repair (C) to minimize mortality, stroke, paraplegia, and renal failure (O)? These four outcomes were deemed "critical for decision making" using the GRADE priority scale for outcomes, whereas other outcomes (cost, length of stay) were considered less important.

Using a standardized search covering the period from 1997 to 2013, the authors selected 37 studies; all 37 addressed the mortality outcome, 21 reported on paralysis, and 12 reported on stroke. No reliable evidence on the renal failure outcome was available. The selection process (ie, how many articles were initially found, how many were excluded and why) was well detailed in the article, reassuring the reader that no articles were excluded due to the researchers' bias and that all pertinent studies on this research question were considered. Often, this process is illustrated using a Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) diagram, which depicts the flow of information through the SR phases, mapping out the number of records identified, included and excluded, and the reasons for exclusions (available at http://www.prisma-statement.org/; accessed September 9, 2018).

The grading of the evidence was accomplished using the GRADE system, which begins with the research design of the included studies.[129] In intervention-related topics, RCTs start as high quality, whereas observational designs start as low quality. Any other design starts as very low quality. For diagnostic studies, the GRADE system allows observational studies to be rated initially as high quality if there was a direct comparison of the test results with an appropriate reference standard.[130] From this starting point (high- or low-quality research design), other factors may increase or decrease the quality of evidence.

FACTORS THAT DECREASE THE QUALITY OF THE EVIDENCE

The factors that can further decrease the quality of the evidence fall into five domains, as follows: risk of bias,

consistency, directness, precision, and publication bias. Incidentally, the Effective Health Care Program sponsored by the Agency of Healthcare Quality and Research uses similar domains.[131]

- *Risk of bias:* This domain refers to the execution of the studies. For RCTs, problems in the allocation concealment, lack of blinding, and loss to follow-up increase the risk of bias and decrease the quality. For other comparative studies, the researchers need to identify the potential for biases as described in previous sections of this chapter.
- *Consistency:* This refers to the degree to which included studies find either the same direction or similar magnitude of effect. In diagnostic studies, this involves looking at the sensitivity and specificity observed in the studies included in the review.
- *Directness:* This refers to the extent to which the population, interventions, diagnostic tests, and outcome measures are similar to those of interest. For example, if the patients in the selected studies were less injured or older than those to whom the intervention would be applied, directness would be low, and the quality would be downgraded. Lack of studies directly comparing two interventions or diagnostic tests of interest can also lower the quality.
- *Precision:* This refers to the degree of certainty surrounding an effect estimate with respect to a given outcome.
- *Publication bias:* As explained in previous sections of this chapter, this relates to the possibility of selective publication or reporting of research findings based on the favorability of direction or magnitude of effect.

FACTORS THAT CAN INCREASE THE QUALITY OF THE EVIDENCE

These factors include large effects (significant RR >2.0 or <0.5) and evidence of dose-response. In addition, if it is reasonable to assume that the observed effect would have been larger if potential confounders were accounted for, the quality may be upgraded.

In the Fox et al[128] SR on BTAI, there were no RCTs, only observational studies; thus, quality started low. They did not observe any serious risk of bias, inconsistency, indirectness, imprecision, or publication bias for any of the outcomes. Thus, the quality of the evidence remained at low (ie, it was not downgraded to very low). Heterogeneity was low for all outcomes; thus, the studies were combined into an MA. Endovascular repair was associated with reduced mortality rates compared to open repair (RR, 0.56; 95% CI, 0.44–0.73), which was not large enough to upgrade the quality of the evidence for the outcome mortality. However, endovascular repair was associated with a significantly lower rate of the outcome paraplegia (RR, 0.36; 95% CI, 0.19–0.71). Thus, for this outcome, the quality of the evidence was upgraded to moderate. Regarding stroke, there were no significant differences between the two procedures (RR, 1.48; 95% CI, 0.67–3.27); thus, the quality of the evidence remain unaltered.

Appropriately so, the EAST panel took into consideration the patients' perspective and deliberated that, despite a low to moderate quality of evidence, patients would likely place a high value on a less invasive procedure associated with lower mortality and risk of paraplegia. Based on these considerations as well as other logistical issues, the panel strongly recommended the use of endovascular repair in BTAI patients who did not have contraindications to endovascular repair. A limitation of this review was the lack of evaluation of long-term outcomes of endovascular repair.

TOOLS FOR APPRAISING THE QUALITY OF STUDIES

Critical appraisal is the systematic evaluation of research reports regarding the quality of several items, including:

- *The research question:* Is it well formulated? Does it address an existing gap in current knowledge?
- *Internal validity:* Is the research design appropriate to address the research question? Was sampling well conducted, bias and confounding minimized, effect modification explored, and an appropriate analytic technique used? Were the effects appropriately measured, and were their size, direction, and uncertainty well estimated?
- *Relevance:* Are these valid results relevant?
- *External validity:* To whom do these valid, relevant results apply?

There are several tools to conduct critical appraisal of evidence. Although originally created to guide peer review of manuscripts, the *Journal of Trauma and Acute Care Surgery*'s standardized research methods review can be a helpful tool to guide critical appraisal (Table 7-3). The Oxford Centre for Evidence-Based Medicine (CEBM) provides appraisal worksheets specific for SRs as well as for prognostic, diagnostic, and therapeutic studies (available at http://www.cebm.net/critical-appraisal/; accessed September 7, 2018), along with helpful examples.

Alternatively, the reporting standards, created to promote transparent and accurate reporting of research studies, may be used to appraise them. The first high-impact reporting guideline was the CONSORT (Consolidated Standards of Reporting Trials; available at http://www.consort-statement.org/; accessed July 7, 2018), mainly directed to RCTs. The CONSORT diagram, which describes the inclusion and exclusion criteria of patients in trials, is now considered an obligatory component by most of the important medical journals. The impact of the CONSORT was so dramatic that reporting standards quickly appeared for other types of studies, including STROBE for observational studies; PRISMA for SRs and MAs; CHEERS for economic evaluations; COREQ for qualitative research; GRADE for guidelines; and others. These reporting standards have checklists that may guide critical appraisal. They are available at the website of the Enhancing the Quality and Transparency of Health Research (EQUATOR) network, a group of renowned international experts that grew out of the work of CONSORT and other guideline development groups (http://www.equator-network.org/; accessed July 7, 2018).

TABLE 7-3: *Journal of Trauma and Acute Care Surgery* Standardized Biostatistical and Research Methods Review

Checklist for statistical assessment of general papers:

Appropriate study design used to achieve the objective(s).
Source of subjects/data appropriately described.
Sampling/sample size appropriately described.
Entry and exclusion criteria clearly defined.
Data exclusions are stated/explained, and impact on results is explored.
N reported at the start of the study, for each data set and for each analysis.
Discrepancies in value of *N* between analyses clearly explained/justified.
Missing data are explained, and impact on findings minimized/explained.
Satisfactory follow-up/response rate.
Appropriate measure(s) of center (eg, mean or median).
Appropriate measure(s) of variability (eg, standard deviation or range).
Adequate uni-/bivariate statistical analyses used/described.
Adequate multivariate statistical analyses used/described.
Confounding and bias explored and minimized.
Assumptions of tests applied met (particular attention paid to nonnormal data sets or small sample sizes).
Effects (odds ratios, relative risks, risk differences) in the "expected" direction, or if not, unexpected direction explained.
Adjustments made for multiple testing explained.
Unit of analysis given for all comparisons.
Alpha level given for all statistical tests.
Actual *P* values are given for primary analyses.
Unusual/complex statistical methods clearly explained.
Method of group assignment (eg, randomization) explained and justified.
Any data transformations clearly described and justified.
Confidence intervals given for the main results.
Conclusion drawn from the statistical analysis is justified.

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8

Acute Care Surgery

John E. Griepentrog • Anthony J. Lewis • Matthew R. Rosengart • Andrew B. Peitzman

KEY POINTS

- Acute care surgery includes trauma, emergency general surgery, surgical critical care, elective general surgery, and surgical rescue.
- The need for expeditious source control is imperative whether we are dealing with a trauma patient with a ruptured spleen or the emergency general surgery patient with a perforated viscus.
- Treat the patient empirically. Determining a specific diagnosis or injury is not the immediate goal.
- Secure the airway, initiate fluid/blood resuscitation, and administer early broad-spectrum antibiotics if sepsis is in the clinical picture.
- In any critically ill patient, resuscitate the patient according to Advanced Trauma Life Support guidelines.

Every important hospital should have on its resident staff of surgeons at least one who is well trained and able to deal with any emergency that may arise.

Dr. William S. Halsted

INTRODUCTION

Over the past decade, a new paradigm in the management of acutely ill surgical patients has emerged, emphasizing surgeons trained in trauma, emergency general surgery (EGS), and surgical critical care.[1] The need has been established for a specialized group of surgeons to care for patients with time-sensitive surgical disease of high acuity, at any time of the day or night.[2] This need comes at a time when we are experiencing an ever-increasing volume of EGS patients. Emergency conditions accounted for more than one-third of all general surgery admissions and over 2.6 million hospitalizations in 2010.[3,4]

As estimated by the Centers for Disease Control and Prevention, nearly 35 million hospital discharges occur annually in the United States.[5] Forty-one percent are emergent, 25% urgent, 24% elective, and 10% unknown. Furthermore, 10 of the most common diagnoses are within the scope of practice of the acute care surgeon (Table 8-1). An important diagnosis on the list is complication of medical or surgical care; this accounts for more than 1 million hospital discharges yearly. Patients who have sustained a major complication of medical or surgical care may be the most vulnerable of our patients. Time-dependent intervention is critical to ensuring an optimal outcome for these patients.

To address this need, the specialty of acute care surgery (ACS) was proposed by the American Association for the Surgery of Trauma (AAST). ACS, as initially presented, was composed of trauma, surgical critical care, and emergency surgery. We have added and redefined the components of ACS as to now encompass trauma, surgical critical care, emergency surgery, elective general surgery, and surgical rescue.

THE BIRTH OF ACUTE CARE SURGERY

The impetus to transform trauma surgery was twofold. First, as surgical specialty care has become more narrowly focused and tertiary and quaternary hospitals have proliferated, the general surgical patient has been left behind. Second, an identity crisis grew from the gradual erosion of the many defining aspects and characteristics of what was once considered a broad-based and fully trained general surgeon.[6] The rise of nonoperative management of many trauma diagnoses resulted in low operative volumes.[7,8] Surgical subspecialists had slowly subsumed many of the fundamental surgical diseases, a process that heralded the disappearance of the trauma surgeon as the master surgical technician.[9] Ironically, these same specialists did not wish to treat trauma patients. These factors, combined with long, unpredictable work hours and high levels of stress, including the high-stakes nature of the work, were not balanced by sufficient reward.[1,7,10]

TABLE 8-1: 2005 Hospital Discharge Diagnoses

Of the leading 29 first-listed diagnoses, 10 were within the practice of general surgery:

1. Complications of surgical and medical care
2. Cellulitis and abscess
3. Septicemia
4. Cholelithiasis
5. Appendicitis
6. Noninfectious colitis and enteritis
7. Diverticula of the intestine
8. Intestinal obstruction
9. Acute pancreatitis
10. Injury

Source: Data from Centers for Disease Control and Prevention. National Center for Health Statistics data. http://www.cdc.gov/nchs/data. Accessed November 21, 2019.

In addition, this high prevalence of specialization meant that only a minority of general surgery graduates would remain in the true general surgery workforce, available to address the most common surgical emergencies.[11] Overall, the number of general surgeons in the United States decreased by 25% from 1981 to 2005, with losses noted in both rural and urban areas.[12] Thus, a need clearly existed to encourage these trainees to remain in the business of caring for general surgical emergencies.

This need was equally visible to those who stood outside the field of surgery. A survey of emergency departments (EDs) documents that 74% of departments experienced problems with specialist coverage, 37% had incomplete or completely lacked general surgery coverage, and 55% reported inadequate trauma staffing[13]; in addition, 23% of facilities either lost or were downgraded in their trauma center level designation.[13] The problem extends beyond the ED to encompass the realm of intensive care; data from the United States underscore a shortage of critical care providers despite an increasing need.[14]

In response to these developments, a task force of the AAST, including members of the American College of Surgeons Committee on Trauma, the Eastern Association for the Surgery of Trauma, the Western Trauma Association, and invitees from additional surgical and critical care organizations convened to discuss the future of trauma surgery. A new field emerged, known as ACS in the United States and emergency surgery or visceral surgery in Europe. This newly defined specialty was to encompass trauma surgery, EGS, and surgical critical care.[1,8,15–17] As mentioned previously, we have redefined and expanded the field to encompass elective surgery and surgical rescue.

THE FIVE PILLARS OF ACUTE CARE SURGERY

The triad of trauma, EGS, and surgical critical care provided the initial foundation for ACS. The two additional pillars of ACS are elective general surgery (fourth pillar) and surgical rescue (fifth pillar) (Fig. 8-1).

Elective General Surgery

Although not universally agreed upon, we and others have always considered elective general surgery a fundamental component of ACS (the fourth pillar). Two reasons drive this approach. First, as a busy operative surgeon, you maintain your surgical skills and the operating room (OR) becomes your default in the management of a critically ill patient. Second, to become a master general surgeon requires commitment of time and effort to develop surgical skills to this high level. The depth and breadth of technical skillsets for emergency operative procedures are attained when you have the time to fully commit to managing the complicated elective general surgical patients. As an example, competence with laparoscopic elective sigmoid colectomy enables you to perform a sigmoid colon resection with or without anastomosis in the patient with perforated diverticulitis.

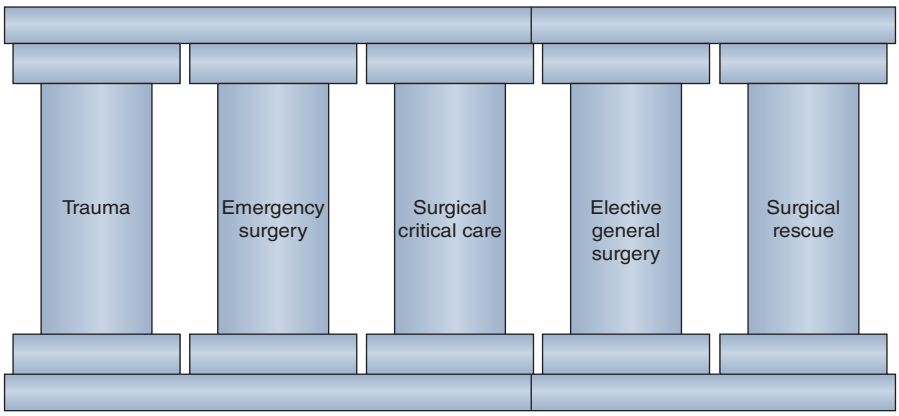

FIGURE 8-1 The five pillars of acute care surgery.

Let's discuss further how involvement in elective general surgery expands your abilities while on call for trauma. The common etiologies of mortality on our trauma patients are central nervous system injury and hemorrhage. These exsanguinating injuries are uncommon—major hepatic or juxtahepatic injury, major vascular injury, and thoracic injury. Even at busy trauma centers, these injuries are infrequent enough that simply taking additional trauma calls does not provide sufficient clinical volume to learn access and management of these injuries. In both our ACS fellowships and early practices as attending acute care surgeons, we should learn these techniques from our specialty partners. Scrub or observe your hepatobiliary surgeons on elective major operations; they resect the liver or repair the inferior vena cava or hepatic veins routinely. Do this as often as possible. The same approach applies for thoracic and vascular surgery; scrub or observe the major operations routinely. Additionally, performing spine exposures for our neurosurgical or orthopedic colleagues augments our ability to expose the vasculature from the clavicles to the inguinal ligaments. Lastly, teach or attend the Advanced Surgical Skills for Exposure in Trauma course as often as possible.

What distinguishes the practice of ACS from purely elective general surgery? The practice of elective general surgery focuses on the anatomy and pathology of the disease (eg, colon cancer). This skillset and knowledge base are also essential to the acute care surgeon. However, should the patient with colon cancer present critically ill with perforation or obstruction, then a different scenario evolves that necessitates a shift in priorities and a more complex delivery of care. It is this additional perturbation in physiology, superimposed upon a primary surgical pathology, with which the acute care surgeon must contend. Thus, ACS surgeons can be considered to be *physiologic surgeons*, compared to their more anatomically oriented colleagues.

Surgical Rescue

Surgical rescue is the fifth pillar of ACS, with the goal of early management of a surgical complication.[18] *Surgical rescue* is successful management of this postoperative or postprocedural complication, whereas *failure to rescue* is a death after either a medical or surgical complication. Thirteen to 20% of general surgery patients in our service are admitted with a primary diagnosis of complication, the vast majority from prior operations by other services or from other hospitals. Most of these patients require an operation, and over 50% require multiple operations to be surgically rescued. Other major components of surgical rescue include intensive care unit (ICU) care, ventilator support, nutritional support, management of sepsis, interventional radiologic procedures, and care by other surgical subspecialties[19] (Fig. 8-2A and B). As mentioned earlier, understanding of the anatomy and principles of the initial elective operation is vital to rescue the patient from the postoperative complication. The complex skill set required to manage the deranged physiology to achieve surgical rescue raises the bar for ACS. The concept of *failure to rescue* was introduced in 1992 by Silber et al[20] in their seminal paper in which they defined failure to rescue as death after an adverse event. Silber and colleagues[20] reported that deaths were related to hospital attributes (eg, nurse staffing, nursing education, hospital size, hospital technology, number of ICU beds) and patient factors (eg, age, comorbidity), whereas complications were due primarily to patient characteristics, and importantly, failure to rescue was related to hospital resources and staffing, as listed earlier. Interestingly, multiple papers have observed that what differentiates high-performing hospitals (lower mortality) from low-performing hospitals (high mortality) is failure to rescue, rather than frequency of the complication in the first place.[21–24]

Another important variable in failure to rescue is inadequate escalation of care. For successful rescue of a patient with a major complication, three steps must occur.[25] First, the bedside provider of care (nurse, respiratory therapist) must recognize the event in a timely fashion. Second, the adverse occurrence must be communicated quickly to the medical staff. Third, a physician/surgeon must be on site and intervene promptly—emphasizing the importance of the in-house ACS surgeon. This escalation of care fails in 21% to 47% of failure-to-rescue deaths.

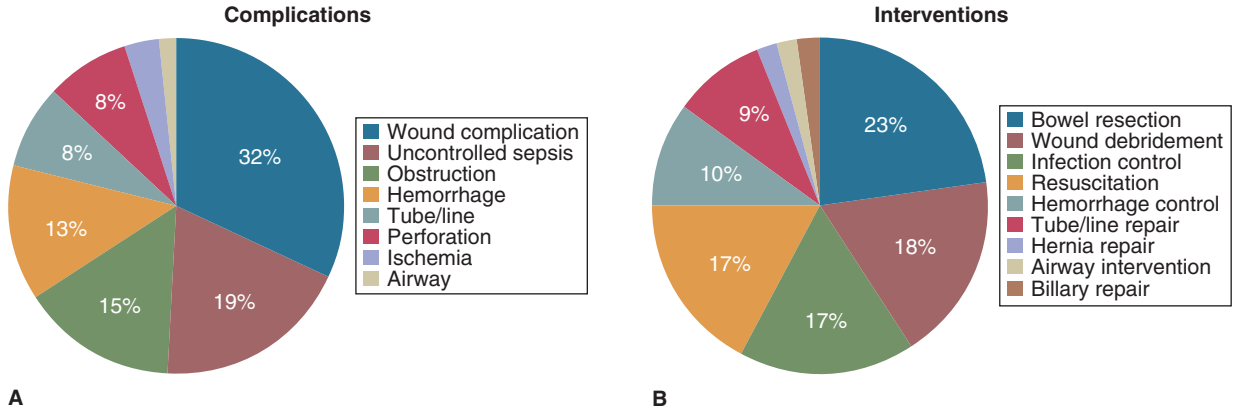

FIGURE 8-2 (**A**) Complications in emergency general surgery (EGS) patients requiring surgical rescue. (**B**) Interventions in EGS patients requiring surgical rescue. (Reproduced with permission from Kutcher ME, Sperry JL, Rosengart MR, et al. Surgical rescue: the next pillar of acute care surgery. *J Trauma Acute Care Surg.* 2017;82:280-286.)

Even a single postoperative complication significantly affects a patient's prognosis; 60-day mortality increases 3.4-fold in patients with complications.[26] Recognition and expeditious management of the first complication are crucial. The risk of secondary complications after the occurrence of five primary complications (pneumonia, myocardial infarction, deep space surgical site infection, bleeding or transfusion event, and acute renal failure) increases significantly.[27] Prompt recognition and treatment of the first complication (rescue) abort the cascade that may proceed to subsequent complications and death (failure to rescue).

EGS is burdened by significant morbidity and mortality, more frequently than in nonemergency surgical patients. EGS composes only 14.6% of general surgery procedures in the National Surgery Quality Improvement Program (NSQIP), but accounts for 53.5% of deaths in the general surgical population.[28] Bleeding, stroke, myocardial infarction, and pneumonia carry a high risk of postoperative death compared to surgical site infection or urinary tract infection.[29] Havens et al[30] reported mortality of 12.5% in EGS patients versus 2.7% in elective operation patients, and the corresponding morbidity rates were 32.8% versus 12.7%.[30] The increased complication rate is further supported by a 17% readmission rate for EGS patients.[30] Furthermore, seven EGS procedures account for 80% of deaths, morbidity, and costs of EGS—partial colectomy, small bowel resection, operations for peptic ulcer disease, cholecystectomy, lysis of adhesions, laparotomy, and appendectomy.[31]

FAILURE TO RESCUE IN THE TRAUMA PATIENT

Failure to rescue in the trauma patient is less common than in the EGS population. The majority of trauma deaths do not have a precedent event (complication) and are dictated by injury burden. Most trauma deaths occur within 24 to 48 hours of admission. Deaths from hemorrhage occur at a median of 1.6 hours after admission, and central nervous system deaths peak at 24 to 48 hours. A review of 35,000 admissions reported preventable deaths in 2.5% of fatalities, with bleeding being the most common etiology of preventable death, generally from delay in treatment.[32] Other causes of preventable deaths were judgment errors and missed injuries. This is in contrast to the EGS patient, in whom complications and failure to rescue generally occur later postoperatively. Holena et al[33,34] have contributed several important papers on failure to rescue in the trauma patient. In a detailed review, they observed that a minority of trauma deaths (6%) were truly preventable. However, the majority of failure-to-rescue cases were deemed preventable or potentially preventable.[33,34]

SURGICAL RESCUE IN THE MEDICAL ICU PATIENT

Although not strictly defined as surgical rescue, management of a major surgical complication in the ICU patient is an important function of the acute care surgeon. In addition, abdominal complications occur in 1% to 4% of medical ICU (MICU) patients. We have recently reviewed our experience of EGS consults on 911 MICU patients. One-third of the consults were for a nonabdominal issue, with 66% of patients undergoing intervention (eg, tracheostomy, soft tissue procedures, feeding tubes, vascular access). Sixty-three percent of the MICU consults were to evaluate an abdominal process; operative intervention was required in one-third (186 of 578 patients). Thus, abdominal operations were required in 1.7% of all MICU admissions (186 of 1192 admissions). The abdominal procedures, in declining frequency, were bowel resection, procedures for *Clostridium difficile* colitis, nontherapeutic laparotomy/laparoscopy, operation for peptic ulcer disease, cholecystectomy, and decompressive laparotomy, and the mortality rate was 37%.

ACUTE CARE SURGERY AND CLINICAL OUTCOMES

The rapid adoption of an ACS paradigm has provided the opportunity to analyze the ramifications of this new specialty on several important operational and clinically relevant outcomes. In a single-center study, implementation of an ACS service was associated with reduced surgical decision time by 15%; the authors hypothesized that this was due to the more immediate availability of an attending surgeon. This correlated with shortened average time to stretcher from the waiting area for all patients in the ED by 20%, thereby reducing ED overcrowding.[35] Another study showed reduced time to the OR for patients (192 vs 221 minutes, $P = .015$) and proportionally fewer after-hours operative cases (60% vs 72.6%, $P < .0001$).[36] Additionally, time in the ER, hospital length of stay (LOS), and complications were reduced after the establishment of an emergency surgical service.[3]

The associations between development of an ACS service and specific disease outcome have been reported. For patients with appendicitis, care provided by an ACS team compared to a traditional general surgery call practice had reduced time from consultation to the OR (3.5 vs 7.6 hours, $P < .05$), decreased time from presentation to the ED to the OR (10.1 vs 14.0 hours, $P < .05$), decreased rate of appendiceal rupture (12.3% vs 23.3%, $P < .05$), decreased complication rate (7.7% vs 17.4%, $P < .05$), and decreased hospital LOS (2.3 vs 3.5 days, $P < .001$).[37] Similar results were reported in other studies comparing ACS to traditional general surgery call, with reduction in patient time to surgical evaluation, shorter time to OR, shorter LOS, and increased cost savings.[36-40] This was due to around-the-clock presence of the ACS attending; in addition, cases that presented in the night hours were not delayed until the following morning, as was the previous practice with the traditional on-call model.[40]

The influence of an ACS service on the surgical management of biliary pathology has also undergone study with supportive observations. Mean time to surgical evaluation was reduced by nearly 6 hours, operative intervention occurred more than 24 hours earlier, fewer complications were incurred, there was a 2-day reduction in hospital LOS and cost savings of approximately $3000 per case.[38] Nearly identical observations have been reported in several independent single-center

studies with the additional benefits of decreased after-hours cases (5.6% vs 21.0%, $P = .004$)[40,41] and fewer conversions from laparoscopic to open cholecystectomy (4.2% vs 11.6%, $P = .013$).[42] An initial concern that combining EGS with a trauma practice would negatively impact trauma outcomes has been dispelled. Multiple studies have shown that despite the increase in workload that occurs with development of an ACS service, times to OR for the injured patient are not untowardly affected, and morbidity and mortality rates do not increase.[15,43,44] In addition, the impact an ACS group has on the practice and productivity of general surgeons has been shown to be symbiotic—both ACS surgeons and elective general surgeons thrive under the new model.

Finally, implementing an ACS service at a nontrauma hospital again demonstrated beneficial effects, with fewer complications, shorter hospital LOS, and monetary savings. After formation of an ACS service, appendectomy complications fell from 13% to 3.7% ($P < .0001$), and hospital LOS decreased from 3.0 days to 2.3 days ($P < .0001$). Cholecystectomy patients experienced fewer complications as well (9% vs 21%, $P = .012$) and also were noted to have shorter LOS (3.8 vs 5.3 days, $P = .0004$).[45] This study highlights the flexibility and variation of ACS groups between different practice locations and settings.

FELLOWSHIP TRAINING

The AAST Committee on Acute Care Surgery has established guidelines for a 2-year curriculum for fellowship training in ACS.[1] Nine months of critical care, the standard of the Accreditation Council for Graduate Medical Education–accredited fellowship in surgical critical care, are paired with an additional 15 months of exposure to various emergency and elective surgical disciplines. Some flexibility and variation are encouraged to capitalize upon the strengths of the local training environment and the perceived individual needs and talents of the fellow in training.[1,16,46] The first year is primarily designed to fully train the fellow in surgical critical care. The focus of the second year is threefold: (1) It provides a period of focused training in those areas most challenging to all trauma surgeons: major hepatic, thoracic, and vascular injuries. (2) Voids that may exist in operative experience and training during the fellow's residency are identified and addressed. (3) The fellow matures in both form and function as an attending through supportive participation on a busy trauma or general surgery service with help nearby. The fellow can also learn about trauma systems, performance improvement, and prehospital care and engage in the research opportunities in the program.

Case Log

Analysis of ACS fellow case logs from the first year of organized reporting revealed that fellows were performing approximately 200 cases in a year, with a 41% to 59% split between elective and urgent/emergent, respectively.[47] Up to half of these cases were in the abdomen, with cases of the head and neck underrepresented. Case requirements for the ACS fellowship have been revised since their initial release in 2007, with cases now divided into anatomic subsections with a minimum number of cases required in each section.[1,46] The goal is to train surgeons to be comfortable in numerous anatomic regions.

FUTURE DIRECTIONS

ACS will continue to evolve and grow. As interest and experience with ACS and the EGS patient cohort expand, so will initiatives to benchmark outcomes and quality improvement tracking, similar to what has been applied to trauma. Analyses of NSQIP data reveal significantly worse outcomes in operations on EGS patients versus nonemergent operations of the same type, with significantly higher prevalence of systemic inflammation and other alterations in physiology in the emergent group.[48,49] These data have led many to propose that different outcome metrics should be applied to emergent versus nonemergent cases of the same operation.[48] Efforts have been made to determine what preoperative characteristics are predictive of a deviation in outcome in EGS.

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9

Genomics and Acute Care Surgery

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KEY POINTS

- Variability in clinical phenotypes results from the following: differences in DNA sequence (genomics), RNA transcription (transcriptomics), and protein translation and structure (posttranscriptional regulation and posttranslational modification).
- It is estimated that only 2% of total DNA includes the code for the approximately 20,000 protein-coding genes of the human genome.
- Exons are the regions of DNA that are transcribed to mRNA and then translated into the amino acid structure of proteins.
- Histone proteins play a key role in the functional regulation of the genome.
- The most common type of DNA sequence variations are single base substitutions termed *single nucleotide polymorphisms* (SNPs).
- SNPs relevant to acute care surgery are found within the promotor regions of genes for Toll-like receptor 1 and tumor necrosis factor-α, both associated with increased mortality after trauma and sepsis.
- MicroRNAs are short single-stranded RNA fragments that downregulate gene expression when bound to messenger RNA.
- Postinjury systemic inflammatory response syndrome–induced early multiple organ failure is related to a "genomic storm" at the level of circulating leukocyte gene transcription and occurs simultaneously with a compensatory anti-inflammatory and immunosuppressive response.
- Chronic critical illness is characterized by an underlying pathophysiology of persistent inflammation, immunosuppression, and catabolism.
- A 63-gene transcriptomic metric score within 24 hours after injury has been shown to be associated with subsequent adverse outcomes including multiple organ dysfunction, length of mechanical ventilation and hospital stay, and infection rates.

INTRODUCTION

Perhaps more than in any other surgical disease, outcomes after traumatic injury have improved through evidenced-based standardization of care.[1] Nevertheless, despite seemingly optimized care, unexpected and complicated outcomes continue to occur in critically ill surgical patients. The question then becomes: Why do patients with similar injuries or severity of acute illness, despite receiving comparable and appropriate treatment, often follow different clinical trajectories? Although one patient recovers uneventfully from hemorrhagic shock after a motor vehicle collision, another follows a prolonged intensive care unit course complicated by nosocomial infections and persistent organ dysfunction. Similarly, despite our understanding of the biology of coagulation and the widespread implementation of prophylactic pharmacologic strategies, venous thromboses and pulmonary emboli still occur, often with fatal consequences.

Numerous clinical, environmental, and genetic factors contribute to variability in the innate inflammatory response, variable rates of drug metabolism, and risk for venous thromboembolic disease. The completion of the Human Genome Project and technologic advances in nucleic acid sequencing and small-molecule identification have provided the foundation on which to build knowledge regarding genetic variation in both humans and animal models.[2] This, in turn, has been used to establish genotype-phenotype associations for common polygenic diseases, such as diabetes mellitus, cancer, and hypertension. Using this genetic variability to understand disease biology, predict risk of disease onset, and calculate an individual's response to a given therapy (eg, drug selection and dosing) are the goals of the field of genomic medicine.[3]

At the molecular level, differences in DNA sequence (genomics), RNA transcription (transcriptomics), and protein translation and structure (posttranscriptional regulation

and posttranslational modification) all result in variability in clinical phenotypes. For instance, drug absorption, metabolism, and excretion are all affected by genetic variation and are the focus of the field of pharmacogenomics. Therefore, understanding the information contained in an individual's genetic fingerprint has the potential for further enhancing the prediction of disease risk, clinical course, and response to any given therapy and facilitating an individualized approach to the severely injured or critically ill patient. Proteomics, metabolomics, and epigenetics are additional aspects of systems biology that are rapidly evolving and promise to deliver new discoveries that are likely to improve patient care. Together, these concepts, techniques, and applications form the foundation of precision medicine. This chapter focuses on select aspects of these fields and their potential application to the care of severely injured and critically ill surgical patients.

STRUCTURE AND FUNCTION OF THE HUMAN GENOME

Since the discovery of the molecular structure of nucleic acids by Watson and Crick in 1954, the genetic basis for many conditions has been determined; however, many misconceptions exist regarding both the past and current role of genomics in clinical medicine.[4] Despite the commonly held notion that knowledge of genetics has had little influence on clinical medicine, it has in fact played an important role in understanding disease, but only for a small number of conditions and patients. Due to rapid advancements in knowledge and technology, however, we have entered a period of tremendous growth in the application of clinical genomics that will eventually influence the care of nearly all patients.[2] In order for clinicians to understand and apply these advances, we must become literate in the "language" of genomic medicine. Table 9-1 lists some

TABLE 9-1: Genomics Nomenclature and Definitions

Allele: One of two or more alternative versions of a genetic sequence at a particular location in the genome.
Epigenetics: The study of changes in organisms caused by modification of gene expression rather than alteration of the genetic DNA sequence.
Exome: Part of the genome composed of exons, the sequences of which remain within messenger RNA after introns are removed by RNA splicing and contribute to the final protein product encoded by that gene.
Gene: An ordered sequence of nucleotides located in a particular position on a particular chromosome that encodes a specific functional product (ie, a protein or an RNA molecule).
Genome: The complete set of DNA found in a cell. In humans, this consists of 23 pairs of chromosomes (~20,000 genes) in the nucleus, as well as a small chromosome found in the cells' mitochondria.
Genomics: The utilization of genetics and molecular biology techniques to analyze the structure, function, and mapping of either sets of genes or the complete genome.
Genome-wide association study (GWAS): An approach used to look for associations between many (typically hundreds of thousands) specific genetic variations (most commonly single nucleotide polymorphisms) and particular diseases or clinical conditions.
Genotype: A person's complete collection of genes. The term can also refer to the two alleles inherited for a particular gene.
Metabolomics: The systematic identification and quantification of the small-molecule metabolic products (the metabolome) of a biological system (cell, tissue, organ, biological fluid, or organism).
Microarray: An analytic technology where thousands of gene sequences are placed in known locations on a glass slide. A sample containing DNA or RNA is deposited on the slide (a gene chip), and the binding of complementary base pairs from the sample and the gene sequences on the chip can be measured with the use of fluorescence to detect the presence and quantity of specific sequences in the sample.
MicroRNA (miRNA): A small noncoding RNA molecule (~22 nucleotides) that selectively turns genes off and on within a cell via posttranscriptional regulation of gene expression (RNA silencing).
Next-generation sequencing: High-throughput technologies that allow nucleic acid sequencing without the physical separation of individual reactions into separate tubes, capillaries, or lanes. Sequencing reactions occur in parallel, allowing millions of sequencing reactions to occur and be analyzed simultaneously.
Pharmacogenomics: The study of the influence of genetic variation on drug response in patients by correlating gene expression or single nucleotide polymorphisms with a drug's efficacy or toxicity.
Phenotype: The set of observable characteristics or traits (eg, morphology, behavior, biochemical or physiologic properties) of an individual resulting from the interaction of its genotype with the environment.
Precision medicine: An approach for disease treatment and prevention that takes into account individual variability and interaction of genes, environment, and lifestyle for each person.
Proteomics: The utilization of molecular biology, biochemistry, and genetic biotechnology to analyze the structure, function, and interactions of proteins produced by the genes of a particular biological system or organism.
Single nucleotide polymorphism (SNP): A single nucleotide variation in a genetic sequence; a common form of variation in the human genome.
Systems biology: Interdisciplinary field of study that focuses on complex interactions within biological systems, using a holistic approach (holism instead of the more traditional reductionism) to biological research.
Transcriptomics: The study of the complete set of RNA transcripts that are produced by the genome, under specific circumstances or in a specific cell type, using high-throughput methods, such as microarray analysis.

important definitions of genetic concepts that clinicians should be able to recognize and understand and that will be discussed throughout this chapter.

Although much is known about the structure and function of the human genome, our knowledge continues to evolve. We now know that the minority of the three gigabases of DNA in the human genome actually code for proteins. It is estimated that only 2% of total DNA includes the code for the approximately 20,000 protein-coding genes of the human genome. The function of the remaining 98% of DNA (specifically, DNA-protein interactions, macrostructure changes, and molecular modification) is perhaps the most evolving and fascinating aspect of genomics. The structure of protein-coding genes is well known. The 5′ control region, often termed the *promoter*, includes DNA sequences that recognize and bind to proteins called transcription factors, whose function is to modify gene expression by modulating transcription. The "start codon" is a series of nucleotides that are recognized by the transcriptional machinery, which initiates the generation of messenger RNA (mRNA) from DNA. Not all of either the DNA or transcribed RNA sequence within an overall "gene" encodes for protein. Exons are the regions of DNA that are transcribed into RNA to make amino acids. In most genes, multiple exons are separated by introns, which are removed from mRNA prior to translation into protein. The end of transcription is signaled by a series of nucleotides at the end of the coding sequence, referred to as a *stop codon*. The DNA sequence after this stop codon, termed the *3′ control region*, can also influence the rate of gene transcription and may affect the stability of the mRNA sequence and its subsequent translation into protein.

Although the mechanisms of transcription and translation are well established, the macrostructure of the human genome is also now emerging as an important regulator of overall genomic function. Genomic DNA is intimately associated with histone proteins that ultimately determine chromosome structure. Although previously thought to be important primarily for chromosomal organization and cell division, it is now known that histones play a key role in the functional regulation of the genome. Through the rapidly advancing field of *epigenetics*, we now understand that histone organization, DNA-histone binding, and molecular DNA modification (eg, DNA methylation) have a strong influence on gene activation and expression (Fig. 9–1). Many epigenetic factors (eg, environmental, drug, nutritional, DNA methylation) have been identified that modulate the DNA-histone interaction and have a direct effect on accessibility of transcriptional factors to individual protein coding genes. This then directly affects gene activation, which can contribute to or directly cause many known disease processes.[5]

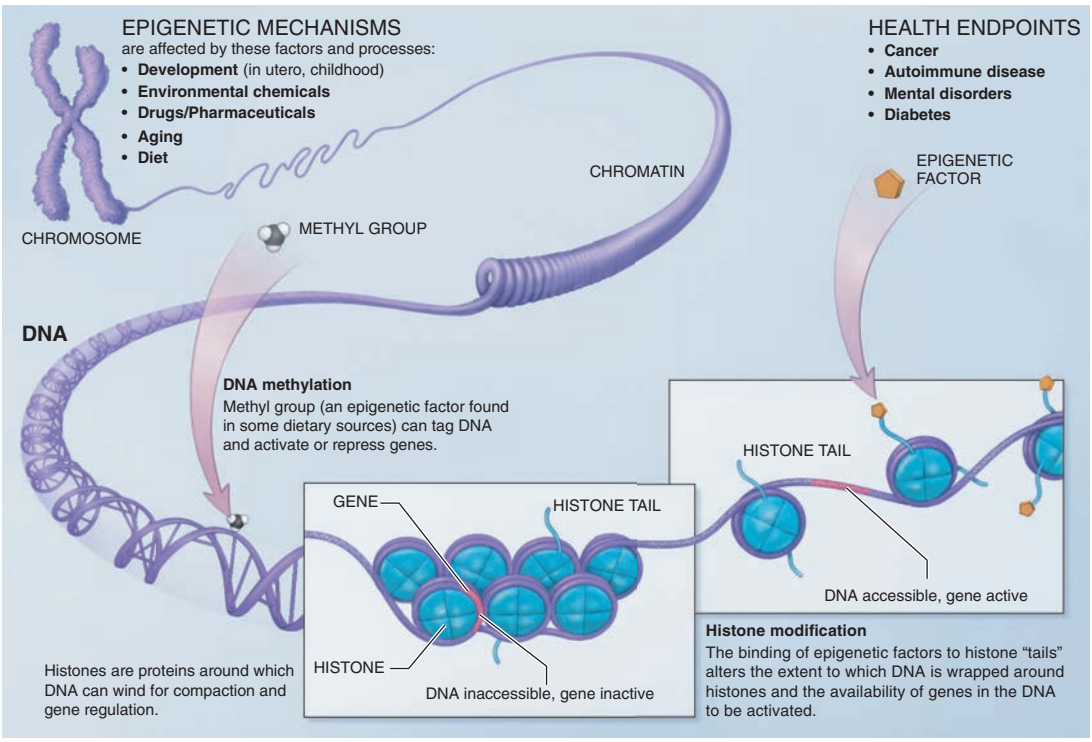

FIGURE 9-1 Epigenetic mechanisms. Epigenetic mechanisms are affected by several factors and processes including development in utero and in childhood, environmental chemicals, drugs and pharmaceuticals, aging, and diet. DNA methylation is what occurs when methyl groups can tag DNA and activate or repress genes. Histones are proteins around which DNA can wind for compaction and gene regulation. Histone modification occurs when the binding of epigenetic factors to histone "tails" alters the extent to which DNA is wrapped around histones and the availability of genes in the DNA to be activated. (Public domain: National Institutes of Health; retrieved from http://commonfund.nih.gov/epigenomics/figure.aspx.)

THE GENETIC BASIS OF DISEASE

Despite the current limited information regarding the function of a large (noncoding) portion of the human genome, our knowledge of the genetic basis for disease is extensive. Basic research and clinical observations have elucidated the inheritance of single-gene Mendelian disorders. Most of these diseases are uncommon, and taken together, the most prevalent (eg, cystic fibrosis and hemochromatosis) affect no more than one in several hundred people. When a genetic variant is present, however, its phenotypic effect on individual patients can be substantial. Understanding the mechanisms underlying many monogenic disorders has provided pathophysiologic information about related, more common disorders. For example, we have learned a great deal about the pathophysiology of cardiovascular disease from discoveries related to familial hypercholesterolemia, a rare genetic disorder leading to premature atherosclerosis.

The most common type of DNA sequence variations are single base substitutions termed *single nucleotide polymorphisms* (SNPs). These SNPs have emerged as important genetic markers for studying multifactorial diseases.[6] Genetic polymorphisms occur in all individuals, but by definition, SNPs exist with a population frequency of greater than 1%.[7] Therefore, the term *SNP* does not include numerous other single base substitutions in which the least common allele is present at a frequency of less than 1% (eg, "personal mutations" that may be limited to one family) or other variations such as insertion/deletion polymorphisms. Importantly, SNPs should be distinguished from disease-"causing" mutations, which are generally much less common but have higher penetrance (eg, sickle cell anemia). SNPs typically do not cause disease but, in aggregate, may be associated with the risk for developing a disease (eg, diabetes mellitus, asthma) or may predict the outcome from a disease or condition. SNPs that exist in mRNA-coding regions (exons) of DNA can lead to amino acid substitutions (missense mutations; eg, sickle cell disease) and, therefore, may change the structure and function of the resultant protein. Although this type of SNP variant potentially has the most profound impact on protein function, it is the least common.[8] SNPs in the regulatory (promoter) region of a gene may influence transcription of that gene and, therefore, may influence the amount of protein available (eg, tumor necrosis factor-α [TNF-α] polymorphism in sepsis).[9,10] Yet, other SNPs may not directly alter protein abundance or function but may be important markers for other (unidentified) functional variants—a concept known as linkage.[8] The analysis of SNPs has been facilitated by two related developments: (1) the establishment of large genomic data repositories, and (2) the increased availability and rapidly declining costs of high-throughput methods for genotyping. These analytical techniques have identified at least 10 million SNPs interspersed throughout the human genome, at a frequency of one SNP per approximately 300 base pairs.[11] Examples of SNPs relevant to acute care surgery are ones found within the coding or promotor regions of genes for Toll-like receptor 1 and TNF-α that are associated with increased mortality after trauma and sepsis.[10,12]

Microsatellite and insertion/deletion polymorphisms represent other genetic variations that may be used to characterize an individual's risk for disease and response to therapy. Microsatellite polymorphisms are tracts of repetitive DNA in which certain DNA motifs (ranging in length from 1–6 or more base pairs) are repeated, typically between 5 and 50 times. Microsatellites occur at thousands of locations within an organism's genome and have a higher mutation rate than other areas of DNA, leading to high genetic diversity. Insertion/deletion polymorphisms are characterized by the presence or absence of a single base in some cases or a longer fragment in others. Microsatellite and insertion/deletion polymorphisms are generally considered to be markers for other functional variants and are not themselves functional, but in some cases they may directly alter gene activity.[13,14] Typically, they occur in nonfunctional regions or in gene regulatory regions and not in coding regions because they would likely lead to a nonfunctional protein. Although translating knowledge obtained from SNPs and microsatellite data directly to the patient's bedside may seem an abstract concept, it is hoped that these studies will continue to contribute to the understanding of the biology of disease and facilitate characterizing individual patient risk, treatment response, and clinical outcome.

Many important advances in genomic analysis have been made using genome-wide association studies (GWAS) and, more recently, next-generation sequencing (NGS), which have revolutionized genomic research.[15] In a GWAS, hundreds of thousands of SNPs are genotyped and analyzed for association with a particular clinical phenotype. NGS can be used to sequence entire genomes or be constrained to specific areas of interest, including all 22,000 coding genes (whole exome) or small numbers of individual genes. Usually, this is accomplished by fragmenting the genome into small pieces, randomly sampling for a fragment, and sequencing it using one of a variety of technologies. Sequencing of an entire genome is often possible within a day because multiple fragments are sequenced at once (giving it the name *massively parallel sequencing*) in a highly automated process. This methodology has been used recently in injured patients to identify candidate SNPs, as well as gene expression patterns of peripheral leukocytes associated with persistent organ dysfunction and mortality.[16-18] The sheer amount of data gathered from a GWAS, however, can present significant statistical problems.[19] Whole-exome sequencing, as the term implies, involves sequencing the protein-coding genes of the genome.[20,21] Exome sequencing will identify rare variants directly. These rare variants are typically missed by GWAS, which rely on relatively common haplotype-tagging SNPs to capture variability across the genome. Therefore, NGS exome sequencing holds promise for digging more deeply into the genome of critically ill injured and surgical patients.

An emerging area of genome-based pathology is the role of microRNAs (miRNAs) in modulating gene expression. miRNAs are short, single-stranded RNA fragments that downregulate gene expression when bound to mRNAs by base pairing with the 3′ untranslated region of target gene

mRNAs to inhibit protein translation. Gene expression modulation via circulating miRNAs has been identified as a potential biomarker and possibly a compensatory mechanism, but also likely a direct contributor to several disease processes including many cancers, cardiovascular disease, and spinal cord injury.[22-24] Particularly relevant to acute care surgery, up to 69 unique and differentially expressed miRNAs have been identified in the plasma of severely injured trauma patients.[25] Further investigation is needed because it is possible that miRNAs play a key role in the modulation of shock-related genes and pathways, including cell surface signaling receptors (Toll-like receptors), inflammatory intracellular signaling (MyD88), and endothelial cell function (VCAM-1).[25,26]

THE "GENOMIC STORM," MULTIPLE ORGAN FAILURE, AND CHRONIC CRITICAL ILLNESS

The advent of intensive care units (ICUs) in the early 1970s facilitated the survival of patients with single organ failure; concurrently, multiple organ failure (MOF) emerged as a highly lethal syndrome (with mortality >80%). Since then, MOF has plagued ICUs for more than four decades, and its epidemiology has evolved as advances in critical care have allowed patients to survive previously lethal insults.[27] Through the years, various predominant clinical presentations of MOF have come and gone (eg, abdominal compartment syndrome, acute respiratory distress syndrome), all having consumed tremendous health care resources with associated prolonged ICU stays and high mortality. Investigators then recognized that both infectious and noninfectious insults could induce a similar, overwhelming, destructive systemic inflammatory response syndrome (SIRS). Epidemiologic studies revealed that MOF was subsequently evolving into a bimodal phenomenon with decreasing early and increasing late mortality. Early MOF occurred after either an initial severe insult (one-hit model) or sequential amplifying insults (two-hit model), whereas late MOF was precipitated by secondary nosocomial infections. The compensatory anti-inflammatory response syndrome (CARS) was proposed to follow SIRS and seemed to explain this increased susceptibility to infection and bimodal distribution of MOF. SIRS-induced early MOF was thought to occur because of exaggerated innate immune and inflammatory responses (ie, "cytokine storm"), whereas CARS was viewed as progressive depression in adaptive immunity resulting in secondary infections. At this point, our understanding of the mechanisms of this dysfunctional postshock immune response remained limited. Research thus shifted to determining the underlying mechanisms of this phenomenon of a postinjury dysfunctional inflammatory response to severe injury and shock.

Significant advancements in our understanding of the genomic basis for the dysfunctional inflammatory response driving MOF after hemorrhagic shock were made by the Inflammation and Host Response to Injury Collaborative Program (Trauma "Glue Grant"). Analysis of total circulating leukocyte gene expression of severely injured blunt trauma patients illustrated that a so-called "genomic storm" at the level of circulating leukocyte gene transcription occurred after injury. In addition, it was found that SIRS and CARS occurred simultaneously rather than sequentially, as previously thought.[28] Patients who exhibited a complicated clinical trajectory (defined as >14 days of persistent organ dysfunction or death) had exacerbation and prolongation of their dysfunctional transcriptomic response and failure to return to baseline expression patterns. Other acute proinflammatory insults such as burns and sepsis have been shown to have a similar pattern of early genomic inflammatory storm and persistent organ dysfunction followed by prolonged low-grade inflammation and immunosuppression.[29]

Due to ongoing advances in surgical critical care, a substantial portion of high-acuity patients with MOF now survive their initial insult and progress into a chronic critical illness (CCI) characterized by an underlying pathophysiology of persistent inflammation, immunosuppression, and catabolism (Fig. 9–2). Contrary to the initially hypothesized bimodal SIRS/CARS paradigm, inflammation co-occurs with immunosuppression and anti-inflammation after major acute events, such as severe trauma, burns, pancreatitis, and sepsis.[28,30,31] For a relatively few patients, an initial overly robust and dysfunctional inflammatory response leads to a trajectory of early MOF and fulminant death. Patients who do not succumb to early MOF follow one of two pathways as follows: (1) the patient is rapidly restored to genomic/immunologic homeostasis, or (2) immunologic dysfunction persists and leads to CCI, characterized by persistent organ dysfunction requiring critical care resources for 14 days or longer. These patients who develop CCI after trauma or sepsis have been shown to have longer hospital and ICU lengths of stay and increased utilization of health care resources, are discharged to care facilities associated with poor long-term outcomes (long-term acute care and skilled nursing facilities), and have a 1-year mortality in excess of 50%.[30,32,33] These dismal outcomes have clearly shown us that we need to think beyond initial shock resuscitation and have a deeper understanding of, and ability to predict and modify interventions for, the host response to injury and sepsis.

DIAGNOSTIC GENOMICS AND PRECISION MEDICINE

NGS expression technology with "big data" computational analyses of genomic data and data from the clinical electronic medical record continues to reveal novel insights into the biology of the response to injury and surgical sepsis.[34] For example, network-based analysis of gene expression from circulating white blood cells revealed that acute systemic inflammation causes transient alteration in numerous pathways and networks, including those involved in immunity and modulation of leukocyte translational machinery.[28,35,36] Additionally, genomic analyses have given us insight into differing host response factors to these insults, including sex and

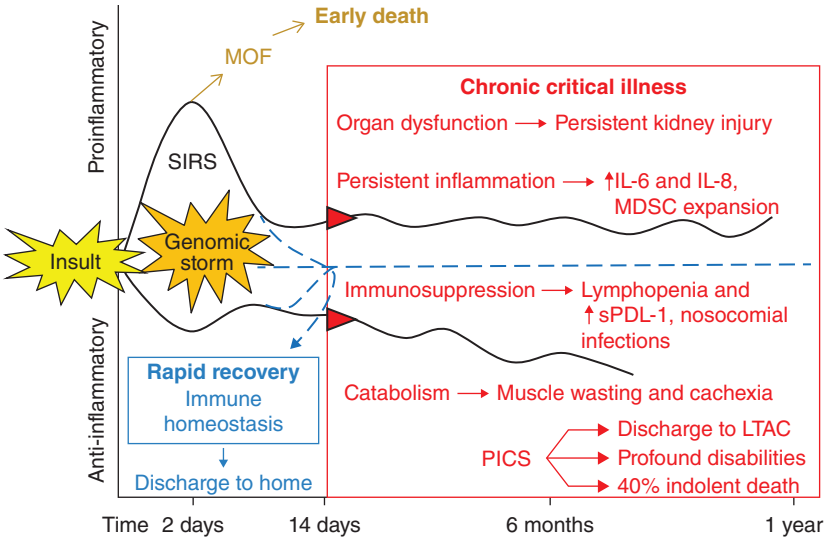

FIGURE 9-2 The "genomic storm" and its persistent downstream effects after trauma, burns, or sepsis. IL, interleukin; LTAC, long-term acute care facility; MDSC, myeloid-derived supressor cells; MOF, multiple organ failure; PICS, persistent inflammation, immunosuppression and catabolism syndrome; SIRS, systemic inflammatory response syndrome; sPDL-1, soluble programmed death ligand 1.

age, which are associated with differences in both genomic response and clinical outcomes.[37-39]

In addition to improving our understanding of pathophysiology, genomic analyses may help improve disease classification and prognostication for severely injured or septic patients.[40,41] The transcriptomic signature after severe injury and hemorrhagic shock has been shown to be highly predictive of onset, severity, and duration of MOF.[28,42] One methodology to develop a genomic metric for trauma uses a baseline or "reference" status for the average leukocyte gene expression profiles of healthy human volunteers. This is then compared to gene expression profiles from patients early after injury to compute a difference-from-reference (DFR) score for each individual.[43] A 63-gene DFR metric score within 24 hours after injury has been shown to be associated with subsequent adverse outcomes, including multiple organ dysfunction, longer length of mechanical ventilation and hospital stay, and increased infection rates. This genomic metric is highly predictive of persistent organ dysfunction (ie, CCI) or death after severe injury and hemorrhagic shock.[17] Clinical genomic metrics such as this may allow us to intervene earlier and select those likely to respond to various immunomodulatory interventions in a precision medicine diagnostic, prognostic, and therapeutic approach.

SUMMARY AND IMPLICATIONS FOR CLINICAL PRACTICE

A growing body of scientific knowledge strongly indicates that genomic data will continue to improve our understanding of acute disease processes and affect clinical care for conditions that are commonly encountered within the scope of acute care surgery, including severe trauma, burns, and surgical sepsis. These proinflammatory insults trigger a "genomic storm" within the innate immune system, leading to MOF, persistent immune dysfunction, nosocomial infections, and recurrent complications. These patients are at high risk of developing CCI and an underlying pathophysiology of persistent inflammation, immunosuppression, and catabolism, which ultimately results in dismal clinical outcomes. Rapidly evolving analytical technologies and our growing understanding of the genomic response to injury and infection are now beginning to successfully translate genomic analysis from the bench to the bedside. In the near future, this will contribute to clinically important advances in diagnosis, risk stratification, and prediction of treatment response for a precision medicine approach to severely injured and septic surgical patients.[44,45]

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10

Prehospital Care

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KEY POINTS

- In the United States, the Emergency Medical Services (EMS) Systems Act of 1973 (PL 93-154) resulted in federal funding for the establishment of emergency medical systems.
- The four levels of prehospital care providers described in the National EMS Education Standards of 2009 are emergency medical responder (formerly, first responder), emergency medical technician (EMT; formerly, EMT-Basic), advanced EMT (replaces both prior versions of the EMT-Intermediate), and paramedic.
- The Stop the Bleed campaign developed under the direction of Lenworth M. Jacobs, Jr., MD, at Hartford Hospital trains laypersons to evaluate for life-threatening bleeding, compress bleeding, and apply tourniquets effectively.
- Basic life support (BLS) in the field includes basic airway management, supplemental oxygen and rescue breathing, cardiopulmonary resuscitation, control of external hemorrhage, splinting, spinal immobilization, and uncomplicated childbirth.
- Advanced life support (ALS) in the field includes advanced airway management, cardiac monitoring, cardioversion and defibrillation, insertion of intravenous lines, and administration of numerous medications.
- In areas primarily covered by BLS units, a tiered response arrangement should be in place so that ALS backup is available when needed.
- By 2015, 96% of the US population lived in an area covered by an enhanced 911 phone system, but this only represents about 50% of the counties in the country.
- When caring for a critically injured patient, EMS personnel should limit on-scene time to 10 minutes or less.

INTRODUCTION

Critically injured patients must receive high-quality care from the earliest postinjury moment to have the best chance of survival. Most trauma victims first receive health care from the emergency medical services (EMS) system, which is responsible for rendering aid and transporting the trauma patient to an appropriate facility.

The practice of medicine in the prehospital setting presents numerous challenges not encountered in the hospital. Hazardous materials along with environmental and climatic conditions may pose dangers to rescuers as well as to patients. If the patient is entrapped in a mangled vehicle or a collapsed building, there must be meticulous coordination of medical and rescue teams. Communication with police may be required in the setting of an active shooter or when concerns exist regarding potential explosive devices. Providers of prehospital care are expected to deliver high-quality medical care in situations that are dynamic, dangerous, austere, and unforgiving, and, often, for prolonged periods.

The role of the EMS system is far more complex than simply transporting the trauma victim to a medical facility. In most EMS systems in the developed world, specially trained health care professionals are responsible for the initial assessment and management of the injured patient. Experience from the past several decades has shown that these paraprofessionals can safely perform many of the interventions that were previously performed only by physicians or nurses in the emergency department.

Prehospital research has helped direct best practices, influence policies, and improve outcomes. As EMS systems mature and additional prehospital care research is conducted, the question is no longer, "What *can* the emergency medical technician (EMT) do for the trauma patient in the prehospital setting?" but rather, "What *should* the EMT do?" The answer to this question can be dynamic depending on such

factors as scene safety, resources available, medical direction, transport times, and experience.

HISTORICAL PERSPECTIVE

While the roots of prehospital trauma care can be traced back to military physicians, modern civilian prehospital trauma care began several decades ago. J.D. "Deke" Farrington and Sam Banks instituted the first trauma course for ambulance personnel in 1960.[1] This course, initiated with the Chicago Committee on Trauma and the Chicago Fire Academy, marked the beginning of formal training in prehospital care of injured patients. Farrington is generally acknowledged as the father of modern EMS.[2]

In September 1966, the National Academy of Sciences and National Research Council published the landmark monograph, *Accidental Death and Disability: The Neglected Disease of Modern Society*.[3] This document argued that there were no standards for ambulances with respect to design, equipment, or training of personnel. As a direct result of this monograph, the Department of Transportation funded the development of the Emergency Medical Technician–Ambulance (EMT-A) curriculum, which was published in 1969. Continued public pressure resulted in the passage of the Emergency Medical Services (EMS) Systems Act of 1973 (PL 93-154). This act revolutionized EMS in the United States and resulted in federal funding for the establishment of EMS systems.

In the late 1950s, J. F. Pantridge, an Irish physician practicing in Belfast, developed a mobile coronary care unit that was staffed by physicians.[4] He conceived of a system in which the victim of an acute myocardial infarction was stabilized at the scene by bringing advanced life support (ALS) to the patient. The physicians worked to restore normal cardiac rhythm through medications and defibrillation at the location where the victim was stricken. This practice brought into focus the debate regarding whether to bring advanced practice to the patient versus transporting the patient as quickly as possible to higher levels of care.

It was soon recognized that physicians were not always available or necessary to provide more advanced care. In the United States, the concept of advanced prehospital care involved training EMTs to perform these lifesaving skills. The original "paramedic" programs began in Los Angeles, California; Houston, Texas; Jacksonville, Florida; and Columbus, Ohio, and were often associated with fire departments. Paramedics were trained to serve as the "eyes and ears" of the physicians in their base hospitals and provide care under their direction.

Although prehospital ALS proved beneficial for victims of cardiac emergencies, it was not until the 1980s that it became obvious that definitive care for trauma patients was fundamentally different than that for the cardiac patient. Efforts to restore circulating blood volume proved to be unsuccessful in the face of ongoing internal hemorrhage. The exsanguinating trauma patient requires operative intervention, and any action that delays the trauma patient's arrival in the operating room is ultimately detrimental to survival. During this period, significant controversy surrounded prehospital ALS for trauma patients as expert panels and editorialists debated the positive and negative impacts.[5,6] Several studies documented the detrimental effect of prolonged attempts at field stabilization on seriously injured trauma patients,[7-9] while others showed that paramedics could employ ALS measures in an expeditious manner.[10-13]

EMERGENCY MEDICAL SERVICES SYSTEM

The modern EMS system involves the integration of a number of complex components. Essential elements include the following: personnel, medications, equipment, communications, transport modalities, medical control, and an ongoing quality improvement process. Different configurations of EMS systems result when these components are integrated in varying combinations. The EMS system represents a significant component of the trauma system, described elsewhere (see Chapter 4).

The Department of Transportation, through the EMS office of the National Highway Traffic Safety Administration (NHTSA), provides federal leadership for the EMS system. With input from national stakeholder organizations, NHTSA developed the *EMS Agenda for the Future*, published in 1996.[14] This document detailed a vision for improving 14 aspects of EMS, including the following: integration of health services, EMS research, legislation and regulation, system finance, human resources, medical direction, education systems, public education, prevention, public access, communication systems, clinical care, information systems, and evaluation. Two related documents that expand on concepts addressed in the original *Agenda* are the *EMS Education Agenda for the Future: A Systems Approach* (2000) and the *National EMS Research Agenda* (2001).[15,16]

EMS Personnel

EMTs compose the vast majority of prehospital care providers employed in the United States, and only a small number of nurses and physicians deliver care in the out-of-hospital setting.

Emergency Medical Technicians

For more than a decade, the *National Emergency Medical Services Education and Practice Blueprint*, published by NHTSA in 1993, provided the basis for the levels and training of EMTs used in the United States.[17] The four levels of EMTs described in the document are the First Responder, EMT-Basic, EMT-Intermediate (EMT-I), and EMT-Paramedic. An enhanced EMT-I level was introduced in 1999, but many states retained the original 1985 curriculum. The Blueprint divided the major areas of prehospital instruction into 16 "core elements." For each core element, there are progressively increasing knowledge and skill objectives, representing

a continuum of education and practice. A National Standard Curriculum (NSC) provided lesson plans for each level.

With the publication of the *EMS Education Agenda for the Future*, the foundation was laid to replace the NSC with a system that it was hoped would standardize EMS training and certification across the country. This system is based on a medical model that includes a defined scope of practice, accredited education programs, certifying exams that ensure baseline competency, and licensure to permit one to practice. Three of the five components of this system focus on the levels and education of EMS providers, and each had input from national stakeholder organizations and the public during its development.

NATIONAL EMS CORE CONTENT

Published in 2005, this document describes the domain of prehospital care, identifying the universal body of knowledge and skills that could potentially be used by EMS providers who do not function as independent practitioners.[18]

NATIONAL EMS SCOPE OF PRACTICE MODEL

Published in 2007, this document identifies four new levels of prehospital care practitioners[19] (Table 10-1). The knowledge and skills described in the Core Content are divided among the four levels. During the development of the Scope of Practice Model, there was insufficient support in the EMS and medical communities to support the development of a fifth level of EMS provider with a scope of practice greater than that of the paramedic.

Regular updates of the National EMS Scope of Practice allow for best practices to be related to prehospital providers. The scope of practices has direct impact on trauma care delivered in the prehospital setting. In 2017, tourniquet use and wound packing were examples of practices highlighted to improve trauma care that impact the trauma care patients receive in the prehospital setting.[20]

NATIONAL EMS EDUCATION STANDARDS

Published in 2009, these standards describe the minimal, entry-level competencies that EMS personnel must achieve for each of the levels described in the Scope of Practice.[21]

TABLE 10-1: EMS Provider Levels

National EMS standard curricula	National EMS scope of practice (2007)
First Responder (FR)	Emergency Medical Responder (EMR)
Emergency Medical Technician-Basic (EMT-B)	Emergency Medical Technician (EMT)
Emergency Medical Technician-Intermediate (EMT-I)	Advanced Emergency Medical Technician (AEMT)
Emergency Medical Technician-Paramedic (EMT-Paramedic)	Paramedic

Compared to the NSC, the Education Standards allow for more diverse methods of implementation, more frequent updates of content, and some variation at the state or local level. Each level builds upon the knowledge and skills of the previous level.

The four levels of prehospital care providers described in this new system are discussed in the following sections.

Emergency Medical Responder. This level was previously termed *first responder*. After the terrorist attacks of September 11, 2001, this term now refers to those who are the initial responders to emergencies and can include law enforcement personnel or firefighters who may lack medical training. The emergency medical responder (EMR) uses a limited amount of equipment to perform initial assessment and rudimentary intervention until EMS providers with a higher level of training arrive at the scene. Skills used by the EMR include oral airways, suctioning, automated external defibrillators, cardiopulmonary resuscitation (CPR), oxygen therapy, hemorrhage control, and manual stabilization of the spine and injured extremity. New skills included at the EMR level that were not taught to the first responder include measurement of blood pressure, eye irrigation, and use of a bag-valve-mask (BVM) device and autoinjectors for self or peers.

Emergency Medical Technician. Previously termed *EMT-Basic*, the EMT has greater knowledge and skills than the first responder and holds the minimum qualifications to staff an ambulance. The EMT possesses expanded assessment skills and is trained to perform spinal immobilization and splinting, assist with uncomplicated childbirth, and use limited medications (oral glucose, sublingual nitroglycerine, and subcutaneous epinephrine). Compared to the EMT-Basic, the new EMT is trained to use more types of oxygen masks, automated transport ventilators, autoinjectors, and oral administration of aspirin.

Advanced EMT. In the current system, the EMT-I is the least well defined of all the levels of EMS providers, as training requirements and skills vary widely from state to state. The Advanced EMT (AEMT) replaces both versions of the EMT-I, although the AEMT is closer to the scope of practice of the 1999 EMT-I than to the 1985 version. Additional time is devoted to acquiring a more in-depth knowledge of pathophysiology, advanced techniques of patient assessment, and advanced skills for airway management, but not endotracheal intubation. The AEMT is trained in intravenous access and can perform fluid resuscitation with crystalloid solutions. Medications an AEMT may administer include epinephrine, glucagon, 50% dextrose, naloxone, and inhaled β-agonists and nitrous oxide.

Paramedic. In addition to the knowledge and skills of the previous levels, the paramedic is trained in the use of a wider range of medications and the performance of a greater number of advanced skills. The scope of practice of the EMT-Paramedic includes endotracheal intubation, needle

decompression of the pleural cavity, cardiac monitoring and interpretation of arrhythmias, and administration of numerous medications. The paramedic has had a major impact on the resuscitation of patients with cardiac or major medical problems and is very effective in urban areas in which response times are short. Compared to the EMT-Paramedic, the new paramedic is trained to administer continuous positive airway pressure, monitor and manage chest tubes, access indwelling venous devices, perform eye irrigation using a Morgan lens, initiate and monitor thrombolytic agents, and perform analysis of limited blood chemistry using portable devices.

EDUCATION AND CERTIFICATION OF EMS PERSONNEL

In addition to the three components of the *EMS Education Agenda for the Future* described earlier, the two additional elements that complete this system of EMS education are national certification and national accreditation of paramedic training programs.

National EMS Certification. In medical practice, certification exams serve to protect the public by ensuring that practitioners have minimal, entry-level competency on entering the workforce. Traditionally, individual states have offered certification exams for their EMS personnel, resulting in issues related to cost for test development, legal challenges, and reciprocity as EMS providers move from one state to another. The National Registry of EMTs (NREMT), a nonprofit organization founded in 1970, has emerged as the only national entity that offers certification exams for all recognized EMS levels. Through its careful test development, NREMT offers psychometrically sound, legally defensible examinations that states may use for licensure of their EMS personnel. Currently, states use the NREMT examination process.[22]

National EMS Education Program Accreditation. The Committee on Accreditation of EMS Programs (CoAEMSP), also a nonprofit corporation, is the only organization that offers accreditation of paramedic training programs on a national basis. CoAEMSP itself is accredited by the Commission on Accreditation of Allied Health Education Programs (CAAHEP). Established in 1994, this group had its origins as the Council on Medical Education of the American Medical Association. CoAEMSP uses a combination of self-assessment and peer assessment to a set of defined standards that ensure a quality educational experience for the student. One recent study demonstrated that graduates of accredited paramedic programs are more likely to successfully achieve certification by the NREMT.[23] The NREMT now requires applicants for the paramedic certification exam to have graduated from an accredited education program.

Nurses

Nurses occupy a unique position in the EMS system. They serve as prehospital providers, instructors, and proctors of quality improvement. Although nursing education imparts an excellent understanding of patient assessment, the pathophysiology of disease processes, and administration of medications, most nursing programs do not teach many of the skills necessary for prehospital care. These include splinting, spinal immobilization, and advanced airway management, so dual training is often required to function in the EMS setting. Nurses may also be employed by EMS services as on-site instructors for continuing education and may be used as field observers for quality improvement. They can provide insight to the EMTs on the smooth integration of patient care from the field to the emergency department.

GROUND NURSES

When dual trained as a nurse and an EMT, the individual can function in the field as a prehospital provider under the auspices of EMT certification. Nurses are used by many critical care transport services to assist in the care of special patients (eg, neonatal and cardiac). In this context, nurses can function to the extent of their training, abilities, and license restrictions. Most states have not developed standards for the prehospital role of nurses. Because of the paucity of trained EMTs, nurses often serve as ambulance attendants in foreign countries.

FLIGHT NURSES

Almost all air medical services in the United States use nurses in the delivery of prehospital care and transport. The composition of the flight crews varies widely, and common configurations are two nurses, a nurse and a paramedic or EMT, or a physician with either a nurse or paramedic, whereas some services fly a nurse with a respiratory therapist. The scope of practice of both the critical care nurses and the paramedics during critical care air or ground transports varies widely from state to state based on nursing law and state EMS regulations, as well as delegation of skills by authority of medical direction. Approximately two-thirds of helicopter medical services in the United States use a combination of a critical care nurse paired with a paramedic. This combination of disciplines provides a wide range of complementary knowledge and skills between the two providers in caring for both out-of-hospital scene flights, which are predominantly for victims of trauma, and the complex patients transported from one hospital to another.

Physicians

In the United States, it is unusual for physicians to directly participate in the provision of care to the injured patient in the field, although about 5% of air medical services use physicians as members of their primary flight crew. The physicians assigned to such crews are usually emergency medicine residents who rotate onto the aircraft as a formal part of their residency. Another use of physicians in the prehospital setting involves neonatologists, pediatric residents, or fellows who staff units used for interfacility transport of critically ill infants.

In Europe, Australia, New Zealand, Japan, and Central and South America, it is common for physicians to function as primary members of the EMS team. Physicians may work for an EMS service, either staffing an ambulance or responding in a separate vehicle. The standards of EMT training in the United States suggest that little is gained by employing physicians on EMS units, and this use of a valuable resource in the field is a challenging one to defend.

Physicians who happen upon the scene of a motor vehicle crash may be tempted to assume control of the patient despite the fact that they possess little experience caring for patients in the prehospital setting. In such situations, the physician should realize that the vast majority of EMTs are well trained, better equipped, and capable of performing their job and that they work under the medical direction of a licensed physician. Additionally, should the physician begin to direct care for a patient, he or she must remain with the patient until care is formally transferred over to an accepting physician, either by radio communication or by face-to-face turnover in the emergency department. Failure to do so may constitute abandonment of the patient and leave the physician exposed to serious legal repercussions.

In unique environments, physicians have been used to improve outcomes in patient transport. Longer-range air transports of critical trauma and medical patients by physicians and nursing staff has been refined by the US Air Force Critical Care Air Transport Teams (CCATT) with positive effect on morbidity and mortality.[24,25] In the setting of complex injuries, prolonged transports, and concurrent resuscitation, the presence of a critical care specialist has been deemed highly valuable.

Bystanders

Just as rapid time to defibrillation for cardiac arrest warranted placing automated defibrillators in public venues and training laypeople in their use, so too has trauma care followed a similar path in the management of hemorrhage. The Stop the Bleed campaign has been a national campaign championed by such organizations as the Department of Homeland Security and the American College of Surgeons in an effort to train laypersons to evaluate for life-threatening bleeding, compress bleeding, and effectively apply tourniquets. The campaign has been an effective opportunity for community engagement for prehospital providers, trauma surgeons, and community members.

EMS SYSTEM DESIGN

Prior to the early 1970s, EMS services in the United States were very rudimentary and focused primarily on transportation of patients. Actual medical care began only after the patient's arrival at the hospital. Today, numerous models of EMS systems exist, as the various elements of the system are combined in different ways. Community leaders design their system around the available resources in the community. An EMS service may be operated by a private company, a hospital, a fire department, a police department, or an agency funded by the government that is solely responsible for emergency medical care (a public "third service"). Regardless of which agency provides EMS, prehospital care generally fits into one of two distinct categories—basic life support (BLS) and ALS.[26]

Basic Life Support

BLS is a term used to describe a level of care that provides noninvasive emergency care and includes care rendered by personnel trained at the EMR and EMT levels. Although EMRs may drive an ambulance, the minimum level for providing patient care during transportation should be the EMT. BLS involves providing basic airway management, supplemental oxygen, and rescue breathing; CPR; control of external hemorrhage; splinting; spinal immobilization; and uncomplicated childbirth. The goal of BLS care is to maintain breathing and circulation and transport the patient without causing further harm. Many BLS services use semi-automatic external defibrillators that identify ventricular fibrillation and deliver electrical countershocks. Because of the limited equipment and training, BLS systems are less costly to establish and maintain than are more advanced levels of care.

Advanced Life Support

ALS describes care that involves the use of more advanced, invasive procedures such as those performed by personnel in an emergency department. EMS providers at the ALS level are capable of advanced airway management, cardiac monitoring, cardioversion and defibrillation, insertion of intravenous lines, and administration of numerous medications. ALS systems use individuals trained at the AEMT or paramedic level.

In contrast to BLS systems, ALS systems provide advanced therapy to the patient at the scene, rather than waiting until arrival at a hospital to institute care. ALS systems have had impressive results in the care of cardiac patients, especially when CPR is started within 4 minutes of a cardiopulmonary arrest and ALS can be initiated within 8 minutes of the arrest. These types of systems, however, are very expensive to establish and maintain, primarily because of the equipment and amount of training required. ALS systems also must invest more in continuing education for their personnel in order to maintain their skills.

Tiered Response Systems

An EMS system that is not purely BLS or ALS but a combination of both is called a *tiered response system*.[24] The goal of a tiered EMS system is to match the training level of the provider with the needs of the patient. The first level of care is typically BLS with the providers being from a public safety agency (eg, fire or police) or EMS units staffed by EMTs. In this model, BLS personnel would initiate transport if the patient did not require ALS procedures. If ALS interventions are needed, the BLS crews initiate basic care and attempt to

stabilize the patient until the ALS unit arrives. This allows ALS units to respond only when needed.

Proponents of this system argue that it functions in a more cost-effective manner, providing ALS-level care only to those patients who require it. In many communities, especially those in rural settings, a third tier composed of air medical transport may be used. This tier usually provides a slightly higher level of training and expertise, combined with the more rapid transport capabilities of the aircraft. The structure of the system will vary widely based on local geography, population density, and resources, but must be set up by local consensus in the region in conjunction with the state(s) involved.

EQUIPMENT FOR EMS UNITS

The American College of Surgeons Committee on Trauma (ACSCOT) joined with the American College of Emergency Physicians (ACEP), the National Association of EMS Physicians (NAEMSP), and other organizations to publish a document delineating the necessary equipment that should be stocked on an EMS unit.[27] This document includes separate recommendations for both BLS and ALS ambulances. More recent revisions require EMS units to include sufficient sizes of equipment to adequately care for infants and children in addition to adults. In most jurisdictions, state law mandates the equipment carried by EMS units, and administrative agencies periodically inspect ambulances to ensure that necessary equipment is present. Medical directors may also require that certain equipment or medications be added to units under their direction.

COMMUNICATIONS

Communications compose an essential component of the EMS system. The EMS dispatch center must be able to readily locate the unit closest to the incident and provide an exact location and description of the call. EMS units must also be able to communicate with other agencies that provide first responder care (ie, law enforcement and fire department) and those that serve an adjunctive role such as extrication and control of hazardous materials. EMS units must also have two-way communication with receiving facilities and with the physicians who provide medical oversight. EMS personnel may request specific orders from a physician when a patient's condition falls outside established treatment protocols. The increased availability of public safety answering points (PSAPs) and EMS dispatch centers to automatic notification of a crash information may enhance their ability to send an appropriate response to a traumatic event.[28,29]

TRANSPORT MODALITIES

Ground Units

EMS units operating on the ground may possess transport capabilities (ie, an ambulance), or they may be a "quick response" unit that contains only equipment and personnel, and a separate ambulance may be required for transport. Such quick response vehicles are common in rural areas or in tiered EMS systems. Ambulances should conform to size and performance specifications as outlined by governmental agencies and authoritative organizations and possess required equipment as described earlier.

In areas primarily covered by BLS units, a tiered response arrangement should be in place so that ALS backup by intercept is available when needed.[26] To qualify as an ALS unit, at least one member of the team must possess training beyond the EMT level. Most commonly, ALS units now are staffed by at least one paramedic, although many ALS services use units staffed by two paramedics. Additional equipment and supplies must be available on the ALS unit to support the defined scope of practice.

Rotor-Wing Aircraft

Helicopter evacuation of military casualties began during World War II, expanded in the Korean War, and matured during the Vietnam War.[30] A mandate made in 2009 by Secretary of Defense Robert Gates reduced the time between combat injury and arrival to definitive care, leading to improved survival.[31] The improvement noted in survival was largely attributed to the speed of evacuation to facilities capable of providing initial trauma care. Civilian air medical services were established in the United States as a result of the success during wartime and have proliferated throughout the industrialized world.

In the United States, helicopter EMS (HEMS) programs are most commonly operated by a private EMS service or are hospital based; however, the Coast Guard, military, law enforcement agencies, or park services may also provide helicopter transport. Crew configurations vary from service to service. The two most common combinations are two flight nurses or a flight nurse and a paramedic. Helicopters are equipped as ALS units and often function as compact intensive care units. HEMS personnel generally have an expanded scope of practice compared to ground EMS providers, including a greater variety of medications and additional skills (eg, management of an intra-aortic balloon pump, extracorporeal membrane oxygenation, complex ventilators, tube thoracostomies, rapid sequence intubation [RSI]), but only a small portion may be applicable to the care of trauma patients. Many advanced HEMS services are now carrying packed red blood cells and plasma to use with patients who have uncontrolled hemorrhage. The usual transport radius for a helicopter is 150 miles, depending on the helicopter model.

Helicopter transport appears to be beneficial for wilderness rescue and for the transport of critically injured patients from a rural facility with limited resources to a major trauma center.[32,33] Analysis in a study from the Centers for Disease Control and Prevention (CDC) of nearly 55,000 HEMS transported patients enrolled in the National Trauma Data Bank (NTDB) showed that the odds of death were 39% lower in patients transported by air versus ground.[34] An even larger study of 256,387 patients based on data from the NTDB

showed that, even though HEMS-transported patients had more severe injuries, they had lower mortality and were more likely to be discharged home.[35] A study from Nova Scotia, where HEMS and ground transport were under the same dispatch center and had the same medical oversight and the same destination medical facilities, showed that blunt trauma patients with an Injury Severity Score of greater than 12 transported by air showed significantly better outcomes than those transported by ground ambulance.[36] Loss of HEMS in one rural area was shown in one study to decrease interfacility transfers and increase transfer times and led to a fourfold increase in mortality of transferred patients.[37] A similar study in a different region of the United States showed no difference in transport times or mortality after loss of HEMS transport capability.[38] The introduction of a second helicopter to the eastern end of Long Island significantly reduced trauma mortality.[39] Unfortunately, many trauma patients transported by HEMS are not critical, and in many trauma centers, it is not unusual for up to one-third of the patients transported by HEMS to be discharged from the emergency department. It is essential that HEMS transports have strong utilization review and medical oversight. HEMS programs need to be fully integrated into the EMS and trauma systems to minimize patient discharge from the emergency department.

The benefits of on-scene HEMS response are also debatable in an urban or suburban setting when a well-trained ground EMS service is present and transport times are brief.[40,41] This is a systems issue because extenuating circumstances such as severe traffic congestion or a mass casualty incident may change HEMS utility in urban areas. A recent study by Diaz et al[42] found that ground transport is always faster than air medical transport when the distance from the scene to the trauma center is 10 miles or less, whereas helicopter transport is always faster when the scene is more than 45 miles from the trauma center. These findings are not surprising considering the time it takes to power up and power down a helicopter. Many EMS systems have the PSAP autolaunch the helicopter based on preset criteria to accelerate dispatch. This is especially true for scenes with potential serious injuries based on information obtained from callers into 911. The helicopter may launch and even land to assess the situation. The helicopter may not necessarily transport the patient, and no charge to the patient is made in that event.

There is a noteworthy element of risk associated with air medical transport, and a study by Bledsoe and Smith[43] documented a steady and marked increase in the number of crashes of medical helicopters over the decade of 1993 to 2002. This trend has continued at an alarming rate since that study was published, resulting in significant loss of life of both patients and the air medical crews. This serious problem is in the process of mitigation since HEMS safety hearings were held by the National Transportation Safety Board (NTSB) in 2009 involving all of the HEMS interested parties. Recommendations were made by the NTSB to the Federal Aviation Administration (FAA) regarding rulemaking pertaining to HEMS safety. The initial draft rules were published in 2014 and finalized in 2015[44] and include such safety items as radar altimeters, helicopter terrain and avoidance warning systems, requiring the pilot in command to maintain current instrument flight rating status, and requiring flight data monitoring systems on HEMS aircraft. All HEMS flights will be flown under the more stringent parameters of FAA Part 135. The various aspects of these rules will be phased in over several years. In addition, over 90% of programs have instituted night vision goggle usage. There has also been a marked increase in simulator training by HEMS programs. The FAA has been mandated to collect data on HEMS flights to determine if these measures increase HEMS safety or if additional measures are needed in rules such as mandating night vision goggles, actual flight data recorders, simulator training for all HEMS pilots, or autopilots for all HEMS aircraft. A recent study from the United Kingdom analyzed all HEMS accidents over the past 26 years and compared their numbers to those of Australia, Germany, and the United States. The authors considered the accident rates per 10,000 missions and the fatal accident rates per 10,000 missions to be comparable.[45] A position paper/policy statement ("Appropriate and Safe Utilization of Helicopter Emergency Medical Services")[46] was jointly developed by several organizations involved with HEMS along with a background paper.[47] Another joint paper by ACSCOT and NAEMSP on HEMS guidelines for appropriateness and safety has also been published.[48]

Fixed-Wing Aircraft

Fixed-wing aircraft are constrained by the need for a runway and, therefore, lack the versatility of rotor-wing units that can land at an accident scene or at a trauma center. With the additional time required to transport a patient to and from a local airport, fixed-wing aircraft only become more time efficient when a patient requires transfer over a distance greater than about 150 miles. Aircraft equipped for air medical transport often serve to transfer patients to regional specialized facilities such as those for burns or spinal cord injuries or to transplant centers. The equipment and supply requirements for fixed-wing aircraft are not as well defined as for ground or rotor-wing units. Fixed-wing aircraft have been used successfully by the US military with such systems as the Critical Care Air Transport Teams during Operation Iraqi Freedom and Operation Enduring Freedom.

PERFORMANCE IMPROVEMENT

Quality medical care is a vital issue in all areas of the health care system. This is attained by developing a performance improvement (PI) process. PI is an ongoing cycle of evaluation, data collection, interpretation, and modification of the system to improve patient care.[49]

An EMS service should have its own internal PI program, with strong oversight by the medical director of the service. Key aspects of this program include evaluation of the care rendered and monitoring the efficiency of the EMS system. A variety of methods are used in order to determine if care is rendered in a timely, efficient, and medically sound fashion.

Equipment must be reliable and durable in order to withstand the sometimes austere and difficult conditions associated with the delivery of prehospital care and not contribute to injury. Trauma centers should also evaluate the care of the patients transported to their facilities and provide appropriate feedback to EMS system administrators, medical directors, and field personnel.

System Efficiency

The evaluation process for any EMS system must determine the efficiency of all components involved in providing care to the patient. One method of evaluating efficiency of the system is to review notification time, response time, on-scene time, and transport time.

NOTIFICATION TIME

This represents the time interval between the injury and notification of the EMS dispatch center. In the United States, most requests for EMS arrive via the 911 phone system. By 2015, 96% of the US population lived in an area covered by enhanced 911 (E911), although that only represents about 50% of the counties in the country.[50] E911 is capable of delivering a wireless caller's number and location to the appropriate PSAP. The PSAP may still have to pass information along to the EMS dispatch center.

RESPONSE TIME

This is defined as the period that starts when an emergency call is received by the EMS dispatch center and ends with the arrival of the ambulance at the patient's side. This time frame encompasses several actions as follows: (1) the call must be physically received; (2) the dispatcher must analyze the call and decide on the appropriate response; (3) the ambulance must be contacted and dispatched; and (4) the ambulance must leave its current location and travel to the scene. The final factor, ambulance travel time, is a function of location and availability of the ambulance, weather, and traffic conditions.

The desired response time for any system directly impacts the number of ambulances that the system requires. To meet the target response times, sufficient EMS units must be available to meet the expected number of emergency calls in the coverage area. Although many urban systems have set a response time standard of 8 minutes that must be met 90% of the time, the ideal response time for trauma is unknown. A retrospective study failed to identify an association between shorter EMS response times and improved outcome in trauma patients.[51]

ON-SCENE TIME (SCENE TIME)

This is the interval from the arrival of EMS at the scene until their departure en route to the receiving facility. This time will vary according to environmental conditions, geography of the scene and location, accessibility of the patient, entrapment, injuries present, and requirements for packaging of the patient. When caring for a critically injured patient, the EMS personnel should strive to limit their on-scene time to 10 minutes or less.[52] Approximately 85% to 90% of trauma patients encountered by EMS are not critically injured and, thus, do not require rapid packaging and immediate transport. Continuous monitoring of on-scene times should be performed to ensure that time is not being lost in the performance of unnecessary procedures on patients with severe injuries.

TRANSPORT TIME

This is the length of time required to transport the patient from the scene to an appropriate facility. The factors that affect this time are distance from the facility, weather, transport modality (air vs ground), and traffic conditions, if transported via ground. The choice of a destination facility is an important decision in the care of the critical patient. The local or regional EMS system in conjunction with the trauma system must have criteria in place for appropriate transport to the appropriate facility.[53] A patient who requires emergent operative intervention to control hemorrhage should be taken to a hospital staffed and equipped to move the patient to the operating room immediately, if such a facility is available. PI reviews should address these issues in an ongoing manner.

Care Rendered

The medical director and leadership of an EMS service must be able to objectively review the care rendered by the personnel they supervise. Evaluation of medical care can be separated into prospective, concurrent, and retrospective phases.

PROSPECTIVE EVALUATION

This form of evaluation attempts to improve the level of care rendered prior to the actual delivery of the care. Evaluating continuing education programs and periodic assessment of skills are examples of prospective evaluation tools.

CONCURRENT EVALUATION

Concurrent evaluation involves direct observation of the EMS personnel during the delivery of care. The medical director or a designated member of the staff of the EMS system (eg, field training officer) accompanies the crew in order to observe the delivery of care in the field. Trauma surgeons may also evaluate the EMS personnel's assessment and care as they deliver patients to their facility. Steps can be taken immediately to correct deficiencies and improve patient care.

RETROSPECTIVE EVALUATION

Retrospective evaluation occurs after care has been delivered. This form of review is the easiest and least costly of the methods and comprises chart audits, case reviews, and debriefings to review the events of any particular EMS call. Trauma surgeons should participate in the retrospective evaluation of

TABLE 10-2: Audit Filters for Prehospital Trauma Care

Lack of adequate airway
Misplaced endotracheal tube
Hypoxia (Spo_2 <90%)
Inability to control external hemorrhage (ie, no tourniquet for extremity)
Spinal immobilization for penetrating torso trauma
Scene time <10 min for critical patient
Appropriateness of needle decompression of pleural cavity
Failure to transport critical patient to closest appropriate facility

EMS services that transport patients to their facility. Such involvement helps EMS providers gain perspective into the entire spectrum of trauma care. A noninclusive list of audit filters that may be used to evaluate prehospital care are shown in Table 10-2. Customized audit filters for specific local/regional PI initiatives should be incorporated as the PI process matures.

MEDICAL CONTROL

One of the most important relationships in EMS is that between the prehospital providers and the physician. Although state EMS offices issue licenses to prehospital care providers, EMS personnel do not function independently, and function under the auspices of their medical director. Thus, EMS personnel perform under delegated practice, which is typically described in the Medical Practice Act in state law.

Numerous terms have been applied to the association between EMS providers and the supervising or responsible physicians, including *medical control*, *medical direction*, and *medical oversight*. Medical direction of an EMS system is provided in a variety of fashions that differ from region to region. In some systems, a single physician provides medical direction, and in other circumstances, medical direction is carried out by a group of physicians acting collectively through a consensus process.

In 1986, Holroyd et al[22] recommended that the EMS medical director be a physician with the following qualifications: (1) knowledge and demonstrated ability in planning and operation of prehospital EMS systems; (2) experience in the prehospital provision of emergency care for acutely ill or injured patients; (3) experience in the training and ongoing evaluation of all levels of participants in the prehospital care system; (4) knowledge and experience in the application of medical control to an EMS system; and (5) knowledge of the administrative and legislative processes affecting regional and/or state prehospital EMS systems.

No specialty of medicine is required for being a medical director. Often, emergency medicine physicians or nontrauma physicians assume the role of medical director. The rise of EMS fellowship training programs has focused medical directorship into a trained subset of physician specialists. In more rural settings, often non-fellowship trained emergency medicine physicians fill the role of medical director. Because nontrauma surgeons may be directing much of the prehospital medical care, it is important for trauma surgeons to work closely with medical directors to ensure trauma care is cutting edge and results in best outcomes.

The medical director must be interested in and committed to the day-to-day activities of the EMS service. The role of medical director for an EMS service is not limited to emergency physicians, and trauma/critical care surgeons are well suited to function in this role once they have gained the prerequisite knowledge of how EMS systems function. The National Association of EMS Physicians has developed a workshop for EMS medical directors. EMS has now been established as a board certifiable subspecialty, with the first subspecialty board exams having been given in 2013. Further information about this process is obtainable from the American Board of Emergency Medicine.

Medical control is divided into two categories: indirect (offline/protocols) and direct (online).[54]

Indirect (Offline/Protocols)

This form of medical direction involves the development of written protocols and the review of EMT performance. The amount of time required to accomplish these administrative duties varies with the size and complexity of the particular EMS system. The medical director's review of care is a PI function and has been discussed previously.

Protocols are the overall steps in patient management that are to be followed by the prehospital provider at every patient contact. Because an accurate diagnosis is often not possible in the field, protocols are usually developed based on the patient's complaints or condition. For ease of memorization and integration with those of other conditions, many protocols are designed in an algorithmic fashion. Trauma surgeons should participate in the development of EMS protocols regarding care for injured patients in their region.

Direct (Online)

Direct or online medical control by the medical director or his or her designee is clinical in nature.[52] This form of direction involves providing radio or telephone instructions to prehospital providers for conditions that are not covered in their protocols and direct observation of individual performance.

Early in the development of EMS systems, a great deal of emphasis was given to direct medical control. Many authorities believed that direct communication between the physician and the prehospital providers would be the mainstay of good prehospital care. Despite this, several studies have demonstrated that there is no difference in survival with and without online medical control and less time is spent in the field when there is no requirement to call the hospital.[52] In cases where medical direction has developed robust guidelines or protocols, online medical control is infrequently used except in unusual circumstances. It is a duty of the medical

director to keep protocol knowledge up to date with the EMS providers.

TRAUMA EDUCATION FOR EMS PERSONNEL

Two continuing education courses have been developed to provide EMS personnel with the essential knowledge and skills to manage critically injured patients. Both courses have been promulgated nationally and internationally.

Prehospital Trauma Life Support

The Prehospital Trauma Life Support (PHTLS) program was developed by the National Association of EMTs in cooperation with ACSCOT.[52] The PHTLS course is based on the tenets of the Advanced Trauma Life Support course developed by ACSCOT, but has been modified to meet the needs of the patient in the prehospital setting.[55] The central philosophy of the PHTLS course is that EMS providers, when given an appropriate fund of knowledge, can make appropriate decisions regarding patient care. Thus, the course emphasizes "principles" of management, rather than focusing on individual preferences or protocols.

A new edition of the course is produced every 4 years, 1 year after the revised ATLS course has been released. This strategy guarantees that PHTLS continues to disseminate any changes in treatment or philosophy that have been introduced in ATLS and ensures a seamless interface between the prehospital providers and personnel in the emergency department in the initial management of the trauma patient. PHTLS is currently taught throughout the United States and in 64 foreign countries.[56]

International Trauma Life Support

About the same time that PHTLS was developed by the National Association of EMTs, the Alabama Chapter of the ACEP developed the Basic Trauma Life Support course.[57] The course was subsequently transitioned to BTLS International, a not-for-profit organization, and in 2005, the name was changed to International Trauma Life Support (ITLS). Like PHTLS, ITLS is also based on the philosophies of ATLS and is taught both in the United States and internationally.

ASSESSMENT AND MANAGEMENT

Assessment and management of the injured patient in the prehospital setting should proceed in an orderly manner, despite the fact that the EMT must frequently make rapid decisions about patient care under adverse conditions. Although the general approach is based on that taught in the ATLS course, one important modification is that the EMT first performs a "scene assessment" prior to evaluating an individual patient. Next, a "primary survey" is conducted to identify life-threatening conditions and initiate immediate therapy.

At the end of the primary survey, the EMT considers whether life-threatening or potentially life-threatening injuries have been identified. If so, the patient is expeditiously packaged and transported to the closest *appropriate* facility. Definitive care for severe, uncontrolled internal hemorrhage cannot be provided in the field, and surgery is usually required. Interventions such as direct pressure on a bleeding wound and infusion of intravenous fluids are not substitutes for rapid transportation to an appropriate facility with immediate surgical capabilities.

Assessment of the Scene

In the prehospital setting, assessment of the patient actually begins before reaching the patient's side. As an EMS crew is dispatched to a scene, they begin to consider numerous factors that may play a role in caring for the patient, as well as ensuring their safety and that of the patient. These factors include such things as mechanism of injury, environmental conditions, and hazards present at the scene. The important aspects of this assessment can be divided into the following two key categories: safety/standard precaution and situation.

SAFETY/STANDARD PRECAUTION

EMS personnel must first evaluate the safety of the scene; that is, they must not enter into a situation that puts their health and well-being at risk as this puts them in jeopardy of becoming patients as well. EMS workers are dependent on law enforcement personnel to ensure that the scene has been cleared of violent assailants and their weapons.[58] In addition to their personal safety, the EMS providers need to consider concerns that threaten the safety of the patient. The scene of a traumatic incident may include dangers such as traffic, downed power lines, hazardous materials, and harsh environmental conditions. In light of incidents of terrorism, there is heightened concern of chemical, biological, or nuclear contamination of a scene, or secondary devices planted with the intent of killing rescuers. Providers should assess for need for at-scene decontamination prior to transport.

Standard Precautions. One hazard ubiquitous to virtually all trauma scenes is blood. Blood and other body fluids may contain communicable diseases including hepatitis and human immunodeficiency viruses. In any patient encounter, health care workers are encouraged to employ measures to decrease the risk of contracting these pathogens. Standard precautions involve the use of impermeable gloves, gowns, masks, and goggles. In addition to wearing this protective gear, EMS providers must also exercise caution when handling sharp devices, such as needles that are contaminated with a patient's blood or body fluid.

SITUATION

The second component of the scene assessment is evaluation of the situation. The EMS providers should consider the

following issues: the number of patients and their ages; the need for specialized personnel or equipment (power company, heavy rescue); the need for additional EMS units, including summoning an air medical helicopter; the need for a physician at the scene to assist with triage; and the possibility that the traumatic event was triggered by a medical emergency (acute myocardial infarction or a cerebrovascular accident). Because the EMS personnel are essentially the "eyes and ears" of the emergency physician and trauma surgeon at the scene, they are in a position to observe key data about the mechanism of injury.

Kinematics. An understanding of the mechanism of injury assists in evaluating the patient for potential injuries (see Chapter 1). Certain mechanisms frequently result in specific injury patterns. Recognition of the mechanism may guide providers in the assessment of the patient. If the incident involves a motor vehicle crash, the EMS crew should evaluate the type of collision (eg, frontal, rear, or lateral impact) and note the degree of damage to the vehicles. The location of the patient at the time of the crash and the use of restraints or protective gear is also valuable information.

Mass Shooting and Terrorist Events. With the advent of frequent mass shootings as well as bombings in the civilian environment, special consideration should be considered for mass shootings. Recent years have brought about consideration for extra emphasis on the safety of the environment in which prehospital providers may find themselves, which had previously been a consideration expected more frequently in military conflicts. Training should be undertaken with police units. Communication is paramount in the dynamic setting of a mass shooting or terrorist attack, because the number of casualties and environment entered can change dramatically over time. Mass shootings may require close communication with law enforcement personnel who may be searching for active shooters or secondary explosive devices. In the case of blasts in workplace environments or bombing devices, prehospital personnel must consider their safety and the potential for further patients. Prehospital providers must be prepared to distribute large volumes of patients to different hospitals so as not to overwhelm a single hospital. Communication is paramount to ensure balance between patient stability and the ability of the closest hospital to continue to manage acute patients.

Primary Survey

After assessing the scene, EMS personnel perform a primary survey of the patient (see Chapter 13). As taught in ATLS, this survey serves to identify life-threatening or potentially life-threatening conditions. Although it is taught in a stepwise A–B–C–D–E approach, one must remember that many aspects of this evaluation can be done simultaneously. In the event of external massive hemorrhage, bleeding should be addressed first. In the event of significant danger to prehospital personnel, such as the presence of an active shooter, a consensus group has recommended a THREAT approach. This procedure consists of steps as follows: (1) threat suppression, (2) hemorrhage control, (3) rapid extrication to safety, (4) assessment by medical providers, and (5) transport to definitive care.[58] EMS personnel employ a "treat as you go" philosophy, wherein care is initiated for life-threatening conditions as they are identified. Thus, the primary survey establishes a framework for setting priorities for management.

AIRWAY MANAGEMENT

Management of the airway is given highest priority, but care must be taken not to aggravate a potential injury to the cervical spine (see Chapter 14). One EMS provider applies manual in-line stabilization to the head and neck while a coworker begins assessment and management of the airway. This stabilization of the cervical spine is continued either until the patient is completely immobilized on a long backboard or until it is determined that the patient does not require spinal immobilization.

All EMS providers, regardless of their level of training, must master the essential skills of airway management.[46] These skills include the following: manually clearing the patient's airway of foreign material; manually opening the airway using the trauma jaw thrust or trauma chin lift; suctioning the oropharynx; and inserting basic oral or nasal airways. An algorithm for prehospital management of the airway is provided in Fig. 10-1.[52]

Endotracheal Intubation. Although this has long been the gold standard for securing an airway in the hospital, its role in prehospital care has become increasingly controversial. This skill is typically limited to advanced providers, although all levels of EMTs have now been taught to safely insert endotracheal tubes. The use of this technique is almost universally accepted at the EMT-Paramedic level throughout the United States. A limited number of communities have allowed EMT-Basics to be trained in endotracheal intubation, as well. In most EMS systems, the success rate for endotracheal intubation exceeds 90%. With good medical direction and field preceptors, training in endotracheal intubation can be successfully accomplished.[59]

Because of the concern of potential fractures of the cervical spine, endotracheal intubation should be performed concurrently with in-line stabilization of the cervical spine.[60,61] Although intubation is most commonly accomplished via the orotracheal route using a laryngoscope, other techniques include digital intubation and retrograde intubation, although these are rarely used in the field.[62-64] Video laryngoscopy is becoming increasingly used in the field by ALS and HEMS personnel. It is not clear yet if this will improve the success rate with less time expended with the procedure. Success of RSI is certainly tied to concentrated experience. A study in San Diego showed that intubation of patients with traumatic brain injuries (TBI) by ground ALS crews led to increased mortality, whereas intubation by air medical crewmembers led to better survival.[65]

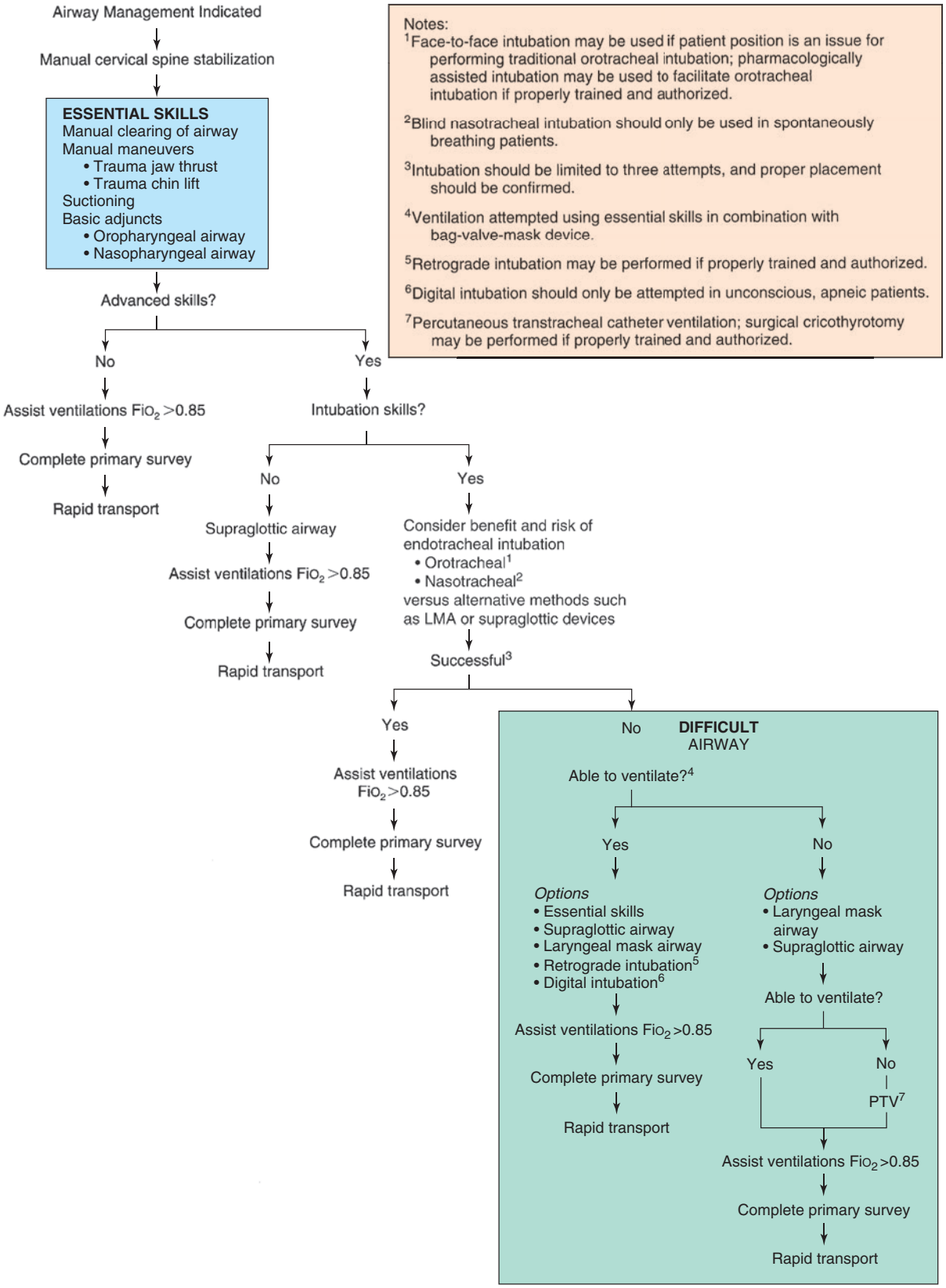


FIGURE 10-1 Airway management. LMA, laryngeal mask airway. (Reproduced with permission from Salomone JP, Pons PT, McSwain NE, eds. *PHTLS: Prehospital Trauma Life Support.* 7th ed. St. Louis, MO: Mosby; 2011:140. Copyright © Elsevier.)

Indications for endotracheal intubation in the field include the following:

- Inability of patient to maintain an airway due to altered level of consciousness (Glasgow Coma Scale [GCS] score <8)
- Need for assisted ventilations
- Threatened airway (eg, respiratory burns, expanding hematoma of the neck)

Concern has arisen that endotracheal tubes placed in the prehospital setting may be misplaced or may become dislodged more commonly than previously believed.[66] Once endotracheal intubation has been performed, care should be taken to confirm proper placement using a combination of clinical assessments and adjunctive devices. The clinical assessments include presence of bilateral breath sounds and the absence of ventilatory sounds over the epigastrium, chest rise with ventilation, and the provider watching the tube pass through the vocal cords. Adjuncts that help confirm a successful intubation include colorimetric carbon dioxide detectors, capnography, and the esophageal detector device.[67] Following intubation, the tube is carefully secured and its position checked each time the patient is moved. Continuous capnography in the prehospital setting may significantly reduce the incidence of misplaced or dislodged endotracheal tubes.[68]

A number of EMS services, especially air medical programs, permit their providers to perform RSI. This involves the administration of both a sedating agent and a neuromuscular blocking agent prior to endotracheal intubation. In skilled hands, this technique can facilitate effective airway control in patients when other methods fail or are otherwise unacceptable (eg, the patient with trismus). The role of RSI in the prehospital setting is controversial, primarily because of concerns related to the risks of losing a partially patent airway by administration of a paralytic agent and data suggesting that patient outcomes may be compromised when RSI is performed by EMS personnel.

With adequate medical control, several studies have documented that EMS personnel can safely perform this procedure.[69-71] Data from one case-control study, however, demonstrated an interesting paradox. Although paramedics using RSI had a higher success rate at performing endotracheal intubation, patients with a suspected severe TBI who were intubated with RSI had a higher mortality than did those in the control group.[72] Davis et al[73] published the findings of an expert panel on the role of prehospital RSI.

Wang and Yealy[74] reviewed the data on prehospital intubation and concluded that there is little literature to support maintaining endotracheal intubation as the standard airway of choice. More studies have documented worsened outcomes than improved outcomes. If intubation is used, the EMS systems must carefully review each intubation attempt and ensure that it is being performed safely. Most children can maintain adequate oxygenation/ventilation with BVM assistance and do not necessarily require endotracheal intubation.[75]

Supraglottic Airways. These are devices that are inserted without a laryngoscope (ie, blindly) into the hypopharynx. Although various models differ in design, properly positioned devices have openings that allow for passage of air from the device into the adjacent glottic opening to ventilate the lungs. Some devices have two ports and ventilations are then administered through the port that results in chest excursion and breath sounds (Combitube, Nellcor, Typo Healthcare, Pleasanton, CA). A similar alternative has a single ventilation port, making it even easier to use (King LT airway, King Systems, Noblesville, IN).

Another supraglottic device is the laryngeal mask airway (LMA) (LMA North America, San Diego, CA), consisting of an inflatable silicone ring attached to a silicone tube. This device is blindly inserted into the hypopharynx so that the ring seals around the glottic opening. Ventilation is then provided through the tube. This device has replaced endotracheal intubation for general anesthesia in a significant percentage of shorter operations, especially in Great Britain. LMAs have been popular in the prehospital setting in Europe and with some air medical services in the United States.[76]

The primary advantage of supraglottic airways is that minimal training is necessary to achieve competency because of their design and the blind insertion. A potential disadvantage of these devices is that the risk of aspiration is believed to be greater than with endotracheal intubation. Supraglottic devices are valuable backup ("rescue") airways when endotracheal intubation cannot be accomplished. Because of the controversies with endotracheal intubation, these airways are increasingly used as the initial airway of choice, although they can be problematic because of potential risks.[77] This is especially true in the urban setting where transport times are generally brief. It may be best to fall back on BVM if ventilation appears difficult. Use of either endotracheal intubation or a supraglottic airway is a low-volume, high-risk procedure and requires rigorous medical oversight.

Needle Jet Insufflation. This involves the insertion of a large-bore needle through the cricothyroid membrane and connecting it to high-pressure oxygen. The lungs are then insufflated periodically. This technique possesses the following advantages: it does not require paralysis, is less invasive than surgical cricothyroidotomy, affords easy access and insertion, and requires minimal education and very basic equipment. The technique has been demonstrated experimentally to be safe and effective even in the presence of complete obstruction of the airway. Although oxygenation is adequate, studies have shown that the patient may become hypercarbic.[78] Percutaneous transtracheal ventilation is indicated when an injured patient is unable to be intubated and cannot be ventilated using a BVM device or an alternative airway.

Surgical Cricothyroidotomy. This involves incising the skin and the cricothyroid membrane, followed by the insertion of a small endotracheal or tracheostomy tube. Because it is highly invasive, complications have included significant hemorrhage and injury to adjacent nerves, blood vessels, and

the larynx. Air medical crews have used surgical cricothyroidotomy in the prehospital setting for several decades with good success. Use of this procedure by out-of-hospital personnel requires strong medical oversight and intense training. It should be reserved for a situation of "can't intubate–can't ventilate."[77]

VENTILATORY SUPPORT

The patient's ventilatory status (breathing) is next examined. If the patient's ventilatory rate is 10 or lower, ventilations should be assisted with a BVM device connected to 100% oxygen. Rapid, shallow breaths indicate inadequate minute ventilation and require assistance with a BVM. Auscultation of breath sounds should be performed during the primary survey if the patient has an abnormal ventilatory rate or evidence of respiratory distress. Patients who have suffered an injury may benefit from supplemental oxygen. Pulse oximetry should be monitored and oxygen administered to maintain an Spo_2 of at least 90% if not more.

Prehospital care providers must exercise caution while providing ventilatory support, as deleterious effects may ensue. Hyperventilation by EMS personnel in one study was associated with increased mortality in patients with a suspected TBI.[79] Additionally, data from animal models suggest that hyperventilation resulted in auto-positive end-expiratory pressure that further compromised the hemodynamic status of a hypovolemic swine.[78] For an adult patient, a reasonable tidal volume of 350 to 500 mL delivered at a rate of 10 breaths/min is probably sufficient to maintain a satisfactory oxygen saturation while minimizing the risk of hyperventilation. Continuous pulse oximetry and capnography can help guide the ventilatory support. Continuous positive airway pressure ventilation and bilevel positive-pressure ventilation are increasingly frequently used in the prehospital environment with proposed benefit in thoracic trauma.[80]

CIRCULATION

Assessment of a patient's circulatory status involves examining for external hemorrhage and evaluating the adequacy of perfusion. Most life-threatening external hemorrhage can be controlled with direct pressure. If manpower is limited, a pressure dressing with gauze pads and an elastic bandage can be placed around an extremity. Should direct pressure alone not control bleeding in an extremity, a tourniquet should be applied just proximal to the site of hemorrhage and tightened until bleeding ceases. A second tourniquet may be required to obliterate the distal pulse and control bleeding. Improvised, noncommercial tourniquets have not been shown to be effective. No published data document any significant decrease in hemorrhage when a bleeding extremity is elevated, and such manipulation may result in the conversion of a closed fracture to an open one. The efficacy of applying pressure over "pressure points" in the axilla and groin has also not been studied in the prehospital setting and is labor intensive. In the operating room, arterial tourniquets have been used safely for periods of 114 to 150 minutes. Options for a tourniquet include a blood pressure cuff and the use of a readily available manufactured tourniquet.[81] If a manufactured tourniquet is used, it should be one that has been tested and recommended by the Committee on Tactical Combat Casualty Care (CoTCCC).[82]

1. Attempt to control hemorrhage with direct pressure or pressure dressing must fail.
2. A commercially manufactured tourniquet, blood pressure cuff, or "Spanish windlass" is applied to the extremity just proximal to the bleeding wound.
3. The tourniquet is tightened until hemorrhage ceases, and then it is secured in place.
4. The time of tourniquet application is written on a piece of tape and secured to the tourniquet ("TK 21:45" indicates that the tourniquet was applied at 9:45 pm).
5. The tourniquet should be left uncovered so that the site can be seen and monitored for recurrent hemorrhage. If bleeding continues after application and tightening of the initial tourniquet, a second tourniquet can be applied just above the first.
6. Pain management should be considered unless the patient is in Class III or IV shock.
7. The patient should ideally be transported to a facility that has surgical capability.

FIGURE 10-2 Protocol for tourniquet application. (Reproduced with permission from Salomone JP, Pons PT, McSwain NE, eds. *PHTLS: Prehospital Trauma Life Support*. 7th ed. St. Louis, MO: Mosby; 2011: 201. Copyright © Elsevier.)

A sample protocol for application of a tourniquet is described as follows and is shown in Fig. 10-2.

Multiple studies have looked at the outcomes of casualties who had tourniquets applied for their extremity wounds.[83-86] The data demonstrated that prehospital use of tourniquets was lifesaving and that complications were low. Less than 2% of the patients suffered transient nerve palsy at the level where the tourniquet was applied, and no limbs were sacrificed because of use of a tourniquet. In patients who had a tourniquet on for 2 hours or less, 28% required fasciotomy, whereas a slightly higher percentage (36%) required fasciotomy if the tourniquet was in place more than 2 hours.

Perfusion is assessed primarily by evaluating pulse rate and quality, skin color, temperature, and moisture. Time should not be taken in the primary survey to measure blood pressure. Even mild tachycardia (heart rate >100 bpm) should always make one consider that the injured patient is hypovolemic. Significant tachycardia (>114 bpm), weak peripheral pulses, and anxiety are associated with loss of 30% to 40% of the blood volume of an adult.[55] A new patient monitoring modality using noninvasive tissue oxygen saturation (Sto_2) shows promise as a way to determine early hemorrhagic shock.[87,88] It must be noted that even one drop in systolic blood pressure (SBP) while monitoring the patient may be enough to indicate early hemorrhagic shock.[89]

A topical hemostatic agent should be considered for significant external hemorrhage from body areas not amenable to placement of a tourniquet (neck, torso, axilla, and groin). The CoTCCC currently recommends Combat Gauze that is

impregnated with kaolin clay as the topical hemostatic agent of choice.[90] Civilian and military experience have found this agent to be safe and effective.[91,92] Other devices that have been proposed to assist with hemorrhage control include the iTClamp (Innovative Trauma Care, San Antonio, TX) and junctional tourniquets. The iTClamp has been suggested to be a fast and reliable device to stop external bleeding by closing the skin to create a temporary, contained hematoma until surgical repair.[93] Junctional tourniquets have been proposed as a means to compress arteries at the junction of the trunk and extremities. Current clinical evidence is not robust in support of the use of such junctional tourniquets; however. there is evidence to support the use based on laboratory work.[94]

Military to Civilian Translation of Trauma Care. As is the case with conflicts throughout history, advances in medical and trauma care are often made during war. An example is the resurgence of tourniquet use that was brought about through demonstrated benefit in outcomes during the wartime data of Operation Iraqi Freedom and Operation Enduring Freedom.[95] Survivability was noted to improve with the use of tourniquets on the battlefield, and despite liberal use of tourniquets, morbidity from the use of the tourniquets was very low.[85] Tourniquet use became more prominent in the civilian environment as a result of data and trauma practitioners returning from deployments.[96] Studies in civilian environments have demonstrated benefit,[97] whereas others have proposed that larger scale civilian studies ought to be conducted for a better understanding of safety and effectiveness of tourniquet use in civilian trauma settings.[98] Civilian and even bystander use of tourniquets has been championed.[99,100] The use of tourniquets has reached national attention with the launch of the Stop the Bleed campaign, a national campaign championing hemorrhage control including the use of tourniquets.[101]

Early hemostatic products, often used by the military, included powders or granules such as QuickClot and newer impregnated gauzes. Different products were found to be more appropriate for different types of wounds in the combat environment. Although much of the data regarding hemostatic agents come from animal models, human studies in the combat setting have demonstrated improvements with hemorrhage control.[102,103] Multiple police and EMS groups have since noted the benefits of hemostatic gauze and have instituted its use as a result of lessons learned in the combat environment.[104]

Traumatic Cardiopulmonary Arrest. Trauma patients who are found to be in cardiopulmonary arrest require special consideration. Unlike cardiopulmonary arrest associated with an acute myocardial infarction, most patients who suffer cessation of their vital signs prior to the arrival of EMS have exsanguinated. CPR, defibrillation, antidysrhythmic medications, and crystalloid resuscitation will not reverse this. Attempts at resuscitation are typically futile and place the EMS personnel at unnecessary risk from automobile crashes during emergency transport and exposure to blood. NAEMSP and ACSCOT have collaborated on a position paper that endorses the following guidelines[105]:

- For victims of blunt trauma, resuscitation efforts may be withheld if the patient is pulseless and apneic on the arrival of EMS.
- For victims of penetrating trauma, resuscitation efforts may be withheld if there are no signs of life (papillary reflexes, spontaneous movement, or organized cardiac rhythm on the electrocardiogram >40 bpm).
- Resuscitation efforts are not indicated when the patient has sustained an obviously fatal injury (eg, decapitation) or when evidence exists of dependent lividity, rigor mortis, or decomposition.
- Termination of resuscitation should be considered in trauma patients with an EMS-witnessed cardiopulmonary arrest and 15 minutes of unsuccessful resuscitation including CPR.
- Termination of resuscitation should be considered for a patient with traumatic cardiopulmonary arrest who would require transport of greater than 15 minutes to reach an emergency department or trauma center.
- Victims of drowning, lightning strike, or hypothermia or those in whom the mechanism of injury does not correlate with the clinical situation (suggesting a nontraumatic cause) deserve special consideration before a decision is made to withhold or terminate resuscitation.

Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA). REBOA has risen as a stabilization technique for noncompressible torso hemorrhage. Proposals from the austere combat surgery environment have proposed the use of REOBA as a lifesaving technique for patients with hemodynamic instability and noncompressible torso hemorrhage.[106] Feasibility studies have demonstrated the potential use of REBOA in both the field and en-route prehospital care.[107] Although the positive effects of REBOA have been noted, the results of current studies deserve further prospective investigation. A joint statement by the American College of Surgeons and ACEP states that an acute care surgeon must be immediately available if REBOA is to be used and also states that no current, high-grade evidence demonstrates REBOA improves outcomes of survival compared to standard treatment of severe hemorrhage.[108]

DISABILITY

During the primary survey, the EMS provider assesses neurologic function by evaluating the patient's GCS score and pupillary response. The GCS score comprises three components including eyes, verbal, and motor.[109] If a painful stimulus is required to complete the assessment, the EMT can either apply pressure to the nail bed or squeeze the axillary tissue. If the patient has an altered level of consciousness (GCS <15), pupillary response to light is assessed. Any belligerent, combative, or uncooperative patient should be considered to be

hypoxic or have a TBI until proven otherwise. In a trauma patient, a GCS score of less than or equal to 13, seizure activity, and a motor or sensory deficit are all reasons for concern. An important corollary to this assessment is to try to ascertain if the patient is anticoagulated, if possible. In some systems, AVPU, a simplification of the GCS, allows for rapid categorization using the categories of Alert, Verbal, Pain, and Unresponsive. An AVPU of anything less than A is considered an indication to get further help.

EXPOSURE AND ENVIRONMENTAL CONTROL

The final part of the primary survey involves a quick scan of the patient's body to note any other potentially life-threatening injuries. In general, this requires removal of the patient's clothes, but environmental conditions and the presence of bystanders may make this impractical. Hypothermia from failure to preserve body heat can contribute to a serious coagulopathy in the trauma patient.

Heavy, dark-colored, woolen clothing may absorb significant amounts of blood. On occasion, patients may have more than one mechanism of injury; that is, blunt trauma from a motor vehicle crash that occurred while trying to flee the assailant who had shot them. Injuries cannot be treated unless they are identified. Other easily missed features include those involving lightning strikes, snake envenomations, and blunt trauma associated with drowning.

Resuscitation

On completion of the primary survey, the EMS provider determines whether or not the patient is critical (Fig. 10-3). Because the primary survey involves a "treat as you go" philosophy, airway management, ventilatory support, and control of external hemorrhage are initiated as the problems are identified. Massive external hemorrhage takes precedence over other assessments.

When a critically injured patient is identified (Table 10-3), scene time should ideally be less than 10 minutes, unless extenuating circumstances such as entrapment or an unsafe scene preclude this. A large multicenter prospective study demonstrated no association between EMS intervals and mortality of injured patients with physiologic abnormalities in the field.[110] On-scene times is one of the most important factors to be scrutinized by medical direction in the quality control process.

If indicated, spinal immobilization should be performed expeditiously and the patient moved to the ambulance. Time is not taken to splint each individual fracture. For the critically injured patient, immobilization to the long backboard provides satisfactory immobilization of potential musculoskeletal injuries. A recent position statement from NAEMSP and ACSCOT discusses the appropriate use of spinal immobilization.[111,112]

Because definitive care cannot be provided to the critically injured patient in the field, EMS personnel must realize that initiation of transport to the closest appropriate facility demonstrates good judgment. Originally developed by ACSCOT, the Field Triage Decision Scheme was recently revised by a national expert panel convened by the CDC (Fig. 10-4).[113] The revision has been endorsed by more than 35 national EMS or trauma organizations. According to this algorithm, patients who meet specific anatomic or physiologic criteria should be transported to the highest level of care in the system, typically a Level I or II trauma center. Patients who meet mechanism of injury criteria should be transported to the closest trauma center, which need not be a Level I or II center. Protocols should be written so that EMS personnel may bypass a closer hospital in order to take a patient with life-threatening injuries to a trauma center based on state or regional guidelines.

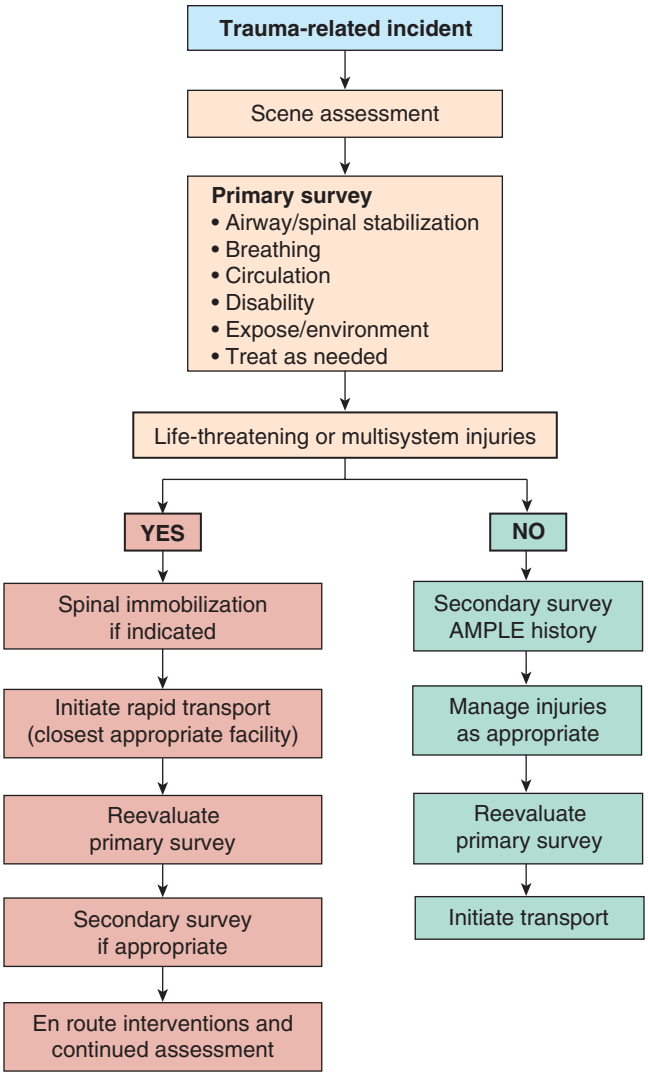


FIGURE 10-3 Prehospital care overview. AMPLE, allergies, medications, past pertinent medical history, last oral intake, events leading up to present injury. (Reproduced with permission from Salomone JP, Pons PT, McSwain NE, eds. *PHTLS: Prehospital Trauma Life Support*. 7th ed. St. Louis, MO: Mosby; 2011:429. Copyright © Elsevier.)

TABLE 10-3: Critical Trauma Patient

- Limit scene time to 10 min or less when any of the following life-threatening conditions are present:
  - Inadequate or threatened airway
  - Impaired ventilation as demonstrated by the following:
    - Abnormally fast or slow ventilatory rate
    - Hypoxia (SpO_2 <95% even with supplemental oxygen)
    - Dyspnea
    - Open pneumothorax or flail chest
    - Suspected pneumothorax
  - Significant external hemorrhage or suspected internal hemorrhage
  - Shock, even if compensated
  - Abnormal neurologic status
    - GCS score ≤13
    - Seizure activity
    - Sensory or motor deficit
  - Penetrating trauma to the head, neck, or torso, or proximal to the elbow and knee in the extremities
  - Amputation or near amputation proximal to the fingers or toes
  - Any trauma in the presence of the following:
    - History of serious medical conditions (eg, coronary artery disease, chronic obstructive pulmonary disease, bleeding disorder)
    - Age >55
    - Hypothermia
    - Burns
    - Pregnancy

GCS, Glasgow Coma Scale.

Source: Reproduced with permission from Salomone JP, Pons PT, McSwain NE, eds. *PHTLS: Prehospital Trauma Life Support.* 7th ed. St. Louis, MO: Mosby; 2011:424. Copyright © Elsevier.

FLUID THERAPY AND BLOOD TRANSFUSION

Infusions of crystalloid solutions and blood transfusion are the mainstays of therapy for the in-hospital treatment of severe hypovolemic shock (see Chapter 15). Because it requires refrigeration and typing, blood is not typically available in the prehospital environment; however, packed red blood cells and plasma are becoming increasingly used by critical care air and ground services. Isotonic crystalloid solutions, such as lactated Ringer's or normal saline (0.9% sodium chloride), can be used for volume resuscitation. Although hypertonic saline (7.5% sodium chloride) initially showed promise, a meta-analysis of several studies, as well as a more recent randomized controlled trial, failed to demonstrate an improvement in survival rates compared to patients treated with isotonic solutions.[114]

En route to the receiving facility, the EMS providers should insert two large-bore (14- or 16-gauge) intravenous catheters in veins of the forearm or antecubital area or use intraosseous access. If possible, lactated Ringer's solution (or normal saline) should be warmed (102°F/38.8°C) prior to administration. Fluid resuscitation in the prehospital setting must be based on the clinical scenario.[55] If the patient has suspected uncontrolled hemorrhage in the thorax, abdomen, or retroperitoneum, fluid infusions should be titrated to maintain an SBP in the range of 80 to 90 mm Hg (mean arterial pressure of 58–65 mm Hg) in the hope of perfusing vital organs while limiting the risk of increased, uncontrollable internal hemorrhage. If the patient has a suspected injury to the central nervous system injury (TBI or injury to spinal cord), intravenous fluids should be administered at a rate sufficient to maintain the SBP at 90 mm Hg (Fig. 10-5). If the patient has identifiable shock that resulted from external hemorrhage that has been controlled, fluids are titrated to maintain a normal pulse rate and blood pressure. If the patient again becomes hypotensive, further intravenous fluids should be titrated to maintain SBP in the range of 80 to 90 mm Hg.

Controversy exists regarding the role of therapy with intravenous fluids in the prehospital setting. No published study has ever demonstrated an improvement in survival resulting from the prehospital administration of fluids. An analysis of almost 777,000 patients from the NTDB showed that administration of intravenous crystalloids by EMS personnel increased mortality.[115]

Although further studies will be needed to clarify this issue, EMS providers should never delay transport simply to initiate intravenous therapy. In one sense, the most important fluid in the prehospital care of critically injured patients is *fuel*—to transport patients rapidly to the closest appropriate facility.

Blood transfusion in the prehospital environment is a source of debate as to benefit compared to the logistical obstacles of storing and administering blood products. Given the logistical challenges involved in prehospital blood product transport and administration, some have argued that standard use is not an effective use of resources.[116] Others have demonstrated effective application of prehospital, low-titer, cold-stored whole blood.[117] In a retrospective massive transfusion protocol analysis, the risk of isoimmunization was very low and the benefit of increased resources for the early administration of balanced resuscitation high, and it was found that the utilization of low-titer, cold-stored, O RhD-positive whole blood is safe and beneficial.

In cases where obstacles exist to blood transfusion, some have proposed the use of prothrombin complex concentrate (PCC).[118] The concept of remote damage control resuscitation begins with a prehospital evaluation for potential use for PCC or tranexamic acid (TXA). Of note, some have suggested using prehospital coagulation or thromboelastogram monitoring of resuscitation with point-of-care devices to guide prehospital or guide preparation for in-hospital resuscitation with more data upon arrival.[119]

TRANEXAMIC ACID

TXA has been proposed as an inexpensive and useful adjunct to patients with hemorrhagic shock.[120] Early prehospital administration of TXA has been suggested to improve clot stabilization and reduce fibrinolytic activity.[121] TXA has been recognized as offering potential benefits in civilian trauma

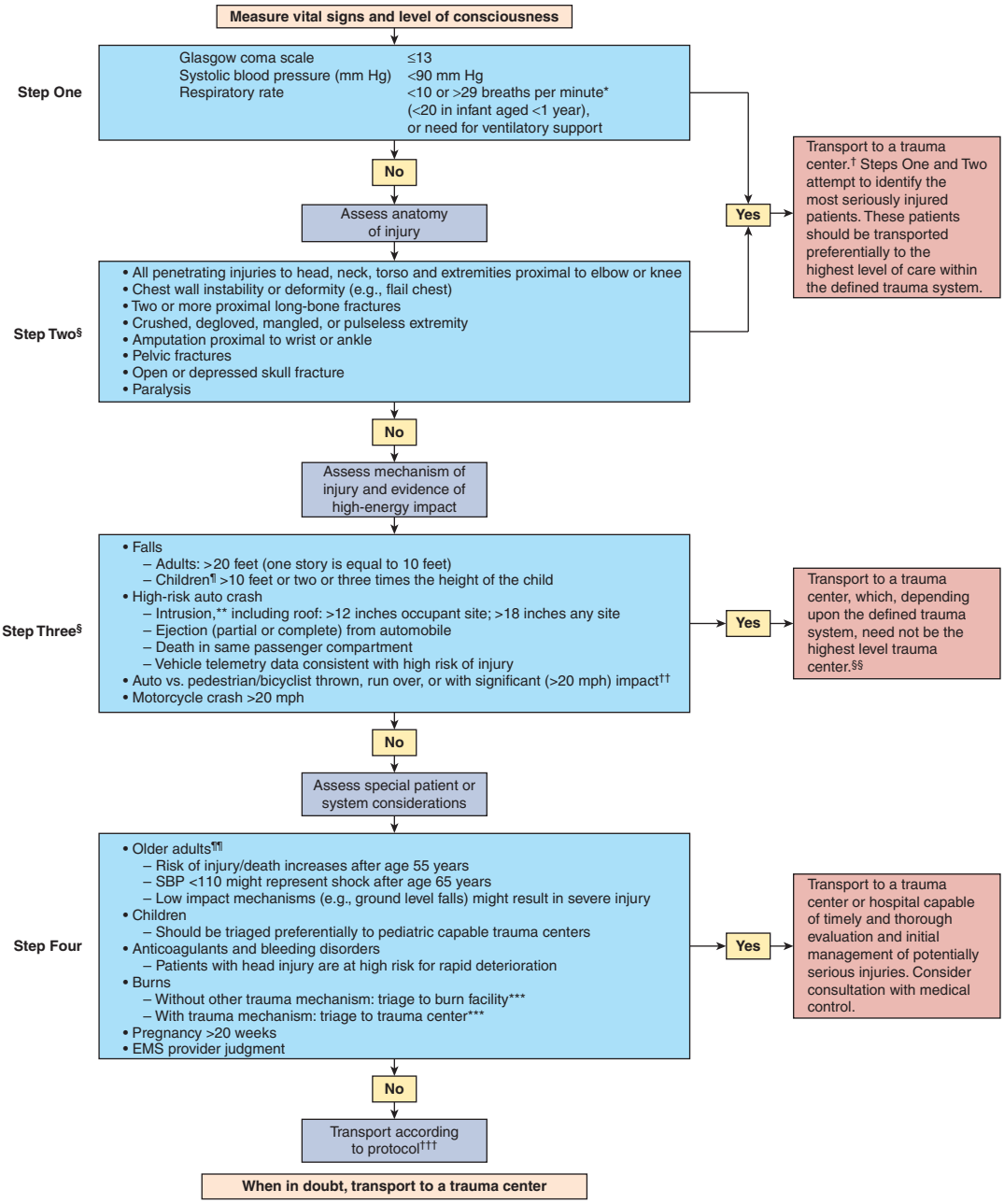


FIGURE 10-4 Field Triage Decision Scheme. EMS, emergency medical services. (Reproduced from Sasser SM, Hunt RC, Sullivent EE, et al. Guidelines for field triage of injured patients: recommendations of the national expert panel on field triage. *MMWR Recomm Rep*. 2009;58[RR-1]:1-35. Adapted from American College of Surgeons. *Resources for the Optimal Care of the Injured Patient*. Chicago, IL: American College of Surgeons; 2006.)

*The upper limit of respiratory rate in infants is 0.29 breaths per minute to maintain a higher level of overtriage for infants.

†Trauma centers are designated Level I to IV. A Level I center has the greatest amount of resources and personnel for care of the injured patient and provides regional leadership in education, research, and prevention programs. A Level II facility offers similar resources to a Level I facility, possibly differing only in continuous availability of certain subspecialties or sufficient prevention, education, and research activities for Level I designation; Level II facilities are not required to be resident or fellow education centers. A Level III center is capable of assessment, resuscitation, and emergency surgery, with severely injured patients being transferred to a Level I or II facility. A Level IV trauma center is capable of providing 24-hour physician coverage, resuscitation, and stabilization to injured patients before transfer to a facility that provides a higher level of trauma care.

§Any injury noted in step two or mechanism identified in step three triggers a "yes" response.

¶Age <15 years.

**Intrusion refers to interior compartment intrusion, as opposed to deformation, which refers to exterior damage.

††Includes pedestrians or bicyclists thrown or run over by a motor vehicle or those with estimated impact of 0.20 mph with a motor vehicle.

§§Local or regional protocols should be used to determine the most appropriate level of trauma center within the defined trauma system; need not be the highest level trauma center.

¶¶Age >55 years.

***Patients with both burns and concomitant trauma for whom the burn injury poses the greatest risk for morbidity and mortality should be transferred to a burn center. If the nonburn trauma presents a greater immediate risk, the patient may be stabilized in a trauma center and then transferred to a burn center.

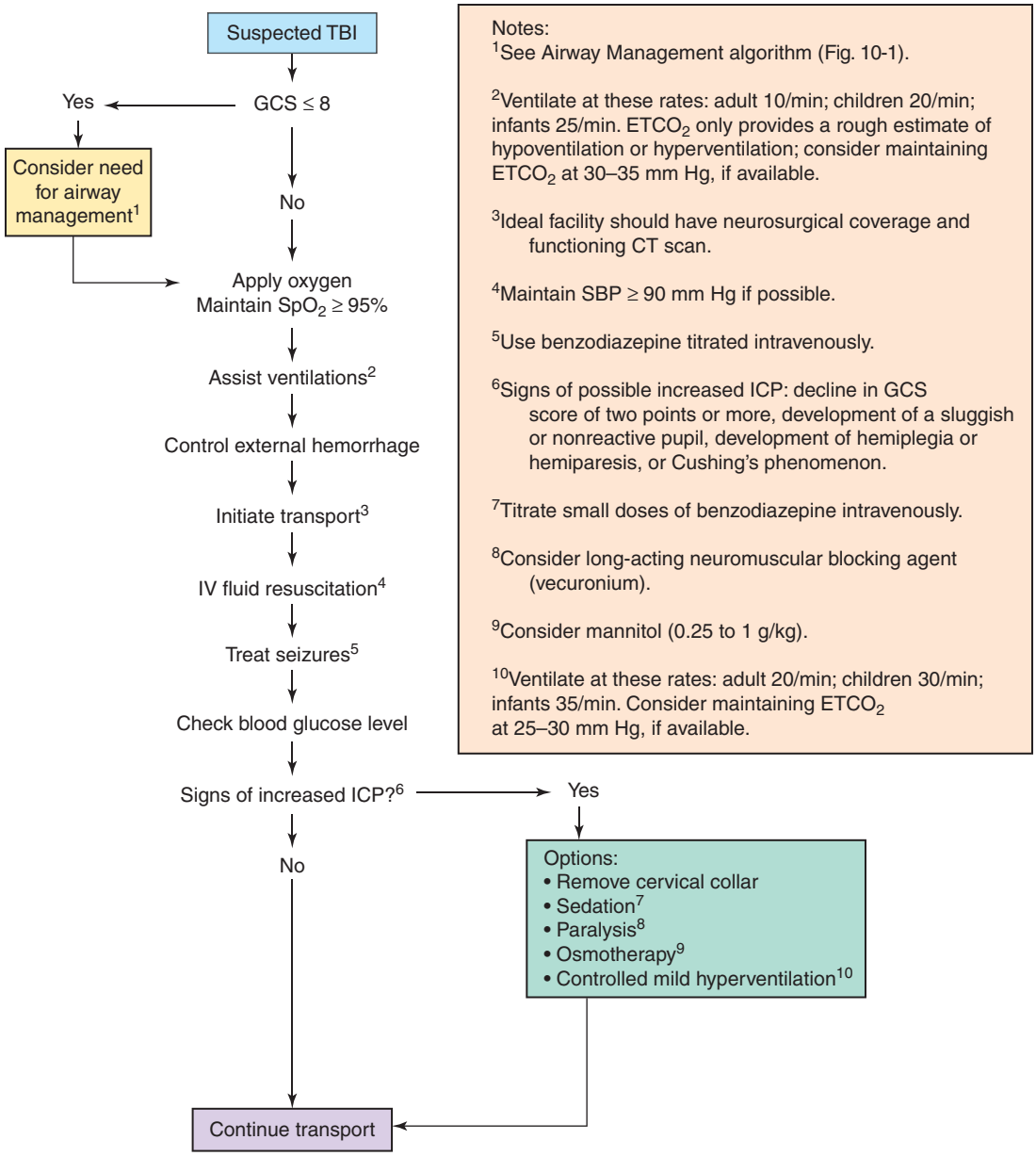


FIGURE 10-5 Algorithm for the prehospital management of traumatic brain injury (TBI). CT, computed tomography; $ETCO_2$, end-tidal carbon dioxide; GCS, Glasgow Coma Scale; ICP, intracranial pressure; SBP, systolic blood pressure. (Reproduced with permission from Salomone JP, Pons PT, McSwain NE, eds. *PHTLS: Prehospital Trauma Life Support.* 7th ed. St. Louis, MO: Mosby; 2011:240. Copyright © Elsevier.)

systems for severely injured shock patients.[122] Data from larger civilian studies found no significant adverse events and an associated decreased use of blood products. Of note, it appeared to be feasible for paramedics both to identify appropriate patients to receive TXA and to safely administer TXA.[123] The current data have caused the US military to advance TXA, as it has been added to the Joint Theater Trauma System Damage Control Resuscitation Clinical Practice Guideline. However, preliminary data demonstrate a risk of TXA use in patients with thrombolytic shutdown resulting in venous thromboembolism.[124] Further research will be necessary before definite recommendations on TXA can be made.

Secondary Survey

Secondary survey refers to a more thorough history and physical examination. For the patient with life-threatening conditions identified in the primary survey, the EMS provider performs the secondary survey when those conditions have been addressed and are stable or improving and the patient is being transported. If the primary survey fails to indicate that

the injured patient is critical, then the provider proceeds on to the secondary survey.

AMPLE HISTORY

This brief history from the patient or family includes the following:

- Allergies to medications
- Prescription or over-the-counter medications
- Pertinent past medical history
- Last eaten
- Recall of events leading up to the injury

HEAD-TO-TOE SURVEY

This complete physical examination begins with obtaining a complete set of vital signs. Injuries to the head, neck, chest, abdomen, pelvis, and extremities are noted. The patient is then turned using the log roll maneuver if a spinal injury is suspected, and the patient's back is examined. Finally, a neurologic examination that involves reassessing the GCS score, pupillary reaction, and motor and sensory functions in the extremities is completed.

Specific Conditions

SCALP WOUNDS

Because of the high concentration of blood vessels in the skin and soft tissues of the scalp, face, and neck, even a small wound can result in serious external hemorrhage. EMS providers and other health care workers often fail to appreciate that patients with a complex scalp wound may bleed sufficiently to develop shock. A compression dressing created with gauze pads and an elastic bandage often provides satisfactory control of hemorrhage. Hemostatic dressings can also be useful for uncontrolled scalp bleeding.

TRAUMATIC BRAIN INJURY

TBI remains one of the leading causes of mortality in injured patients. Secondary brain injury refers to the extension of the original injury and may result from numerous causes. These include hypoxia, hypocapnia and hypercapnia, anemia, hypotension, hypoglycemia and hyperglycemia, seizures, and intracranial hypertension as the result of edema or mass effect. Optimal prehospital care of the patient with a TBI involves preventing secondary brain injury, maintaining cerebral perfusion pressure (mean arterial pressure minus intracranial pressure), and expeditious transfer to a facility capable of caring for the injury.

A patient with a severe TBI may be unable to control his or her airway, and endotracheal intubation should be considered for patients with a GCS score of 8 or less, although an alternative airway device may provide a satisfactory airway. Ventilatory support should be administered, and the patient maintained eucapnic as prophylactic hyperventilation is no longer indicated.[125,126] Data from patients with TBI indicate that those who arrive in the emergency department with either hypocapnia (arterial Pco_2 <30 mm Hg) or hypercapnia (Pco_2 >45 mm Hg) have poorer outcomes compared to those who arrive in a eucapnic condition.[127] As indicated earlier, providers with greater experience and the availability of continuous end-tidal capnography led to better survival of patients with a TBI who had RSI performed by HEMS providers than by ALS paramedics in San Diego.[65] Targeted oxygen therapy has been noted to be of importance, as hyperoxia has been found to have deleterious effects in the critically ill, including traumatic head injured patients.[128]

Blood loss should be minimized by controlling external sources and splinting fractures as appropriate. Because of the risk of an associated injury to the spine, patients with a suspected TBI should undergo spinal immobilization. Intravenous fluids should be initiated en route to the receiving facility with a goal of maintaining the SBP at 90 mm Hg. During prolonged transport, blood glucose can be monitored and dextrose administered if the patient is hypoglycemic. Benzodiazepines are appropriate for control of seizures, but they should be carefully titrated intravenously because of the risk of hypotension and respiratory depression.

Intracranial hypertension may cause cerebral herniation and brain death, but it cannot be measured in the prehospital setting. Signs of possible intracranial hypertension include the following: a decline in the GCS score of 2 points or more, development of a sluggish or nonreactive pupil, development of hemiplegia or hemiparesis, or Cushing phenomena (bradycardia associated with arterial hypertension).

THORACIC TRAUMA

Flail Chest and Pulmonary Contusion. In the prehospital setting, the administration of oxygen and ventilatory support are the primary therapies for a flail chest and suspected pulmonary contusion (see Chapters 27–30). Oxygen saturation should be kept at 95% or higher by applying supplemental oxygen. If these measures fail to provide adequate oxygenation, ventilations should be assisted and endotracheal intubation considered if the patient's tidal volume appears inadequate.

Tension Pneumothorax. Tension pneumothorax should be suspected whenever the following three criteria are identified: increasing respiratory distress or difficulty ventilating with a BVM device, decreased or absent breath sounds, and hemodynamic compromise. Needle decompression of the pleural space can be lifesaving.[52,129] An intravenous catheter at least 12 gauge in diameter should be used and should be left in place. There is no need to create a one-way ("flutter") valve because any air exchange through the catheter is clinically insignificant. Recent data suggest that a catheter length of at least 8 cm is necessary to reach and decompress the pleural space.[130]

Open Pneumothorax. An open pneumothorax should be sealed with an occlusive dressing. One of the four sides of the dressing may be left untaped so that air can decompress

from the pleural space as needed. After an occlusive dressing has been applied to an open pneumothorax, any signs of a developing tension pneumothorax should prompt the EMS worker to remove the dressing. If this does not result in improvement of the patient's status, needle decompression should be considered.

Pericardial Tamponade. Pericardial tamponade is generally encountered following penetrating trauma to the heart; however, it may be a complication of a blunt cardiac rupture. In the prehospital setting, the classic symptoms of Beck triad (elevated venous pressure, muffled heart tones, and hemodynamic compromise) may be difficult to identify. Although some EMS systems permit ALS personnel to perform pericardiocentesis if pericardial tamponade is suspected, the emphasis should be placed on transporting that patient with a suspected tamponade to a facility that has immediate surgical capabilities.

ABDOMINAL TRAUMA

Intra-Abdominal Hemorrhage. In the absence of an obvious sign such as a bullet wound, intra-abdominal hemorrhage is difficult to identify in the prehospital setting, especially in the unconscious trauma patient (see Chapters 31–38). Unexplained hypovolemic shock should lead the EMS provider to suspect this condition. Management involves rapid transport to a facility that offers immediate operative intervention. A FAST (focused assessment for the sonographic evaluation of the trauma patient) examination is becoming increasingly available in air and ground EMS units and can help with triage.

Pelvic Fractures. The presence of a severe pelvic fracture may be suspected if the EMS provider finds instability on examination of the pelvis, especially if the patient has evidence of hypovolemic shock (see Chapter 39). Pelvic binders, which are often placed on hypotensive trauma patients with proven pelvic fractures in the hospital, have limited utility in the field. EMS providers may not be able to identify a fractured pelvis on physical examination alone, and the pelvic binders are costly. These binders may be useful in the setting of a hypotensive trauma patient with a known pelvic fracture who requires interfacility transport.

Pregnancy. Prehospital management of the injured pregnant patient focuses on adequately resuscitating the mother, especially if shock is present (see Chapter 41). In the third trimester, pregnant individuals may exhibit hypotension while lying supine due to compression of the inferior vena cava by the uterus. Supine hypotension is treated by gently rolling the mother into the left lateral decubitus position or, if immobilized on a long backboard, placing sufficient padding under the right side of the board to elevate it 30° or so. If hypotension does not correct with this measure, hemorrhagic shock should be suspected. Oxygen should be administered and the patient transported to a facility that has both trauma and obstetrical capabilities.

SPINAL TRAUMA

An algorithm has been developed that details the indications for spinal immobilization in the prehospital setting (see Chapter 26) (Fig. 10-6).[52,130] Patients with penetrating trauma to the torso almost never have an unstable vertebral column.[131] A recent analysis of data from the NTDB showed that victims of penetrating trauma who received prehospital spinal immobilization had a higher risk of death compared to those who did not.[132] Therefore, spinal immobilization is indicated in the setting of penetrating trauma only when the patient has a neurologic complaint or finding. In patients with blunt trauma, spinal immobilization should be performed if the patient has an altered level of consciousness (GCS score <15) or if spinal pain or tenderness, a neurologic deficit or complaint, or an anatomic deformity of the spine is present. In the absence of these findings, the mechanism of injury should be evaluated. If the mechanism is considered to be concerning, the patient should be evaluated for evidence of alcohol or drug intoxication, presence of a distracting injury, or the inability to communicate. If any of these are present, spinal immobilization should be performed. In their absence, spinal immobilization is not indicated.

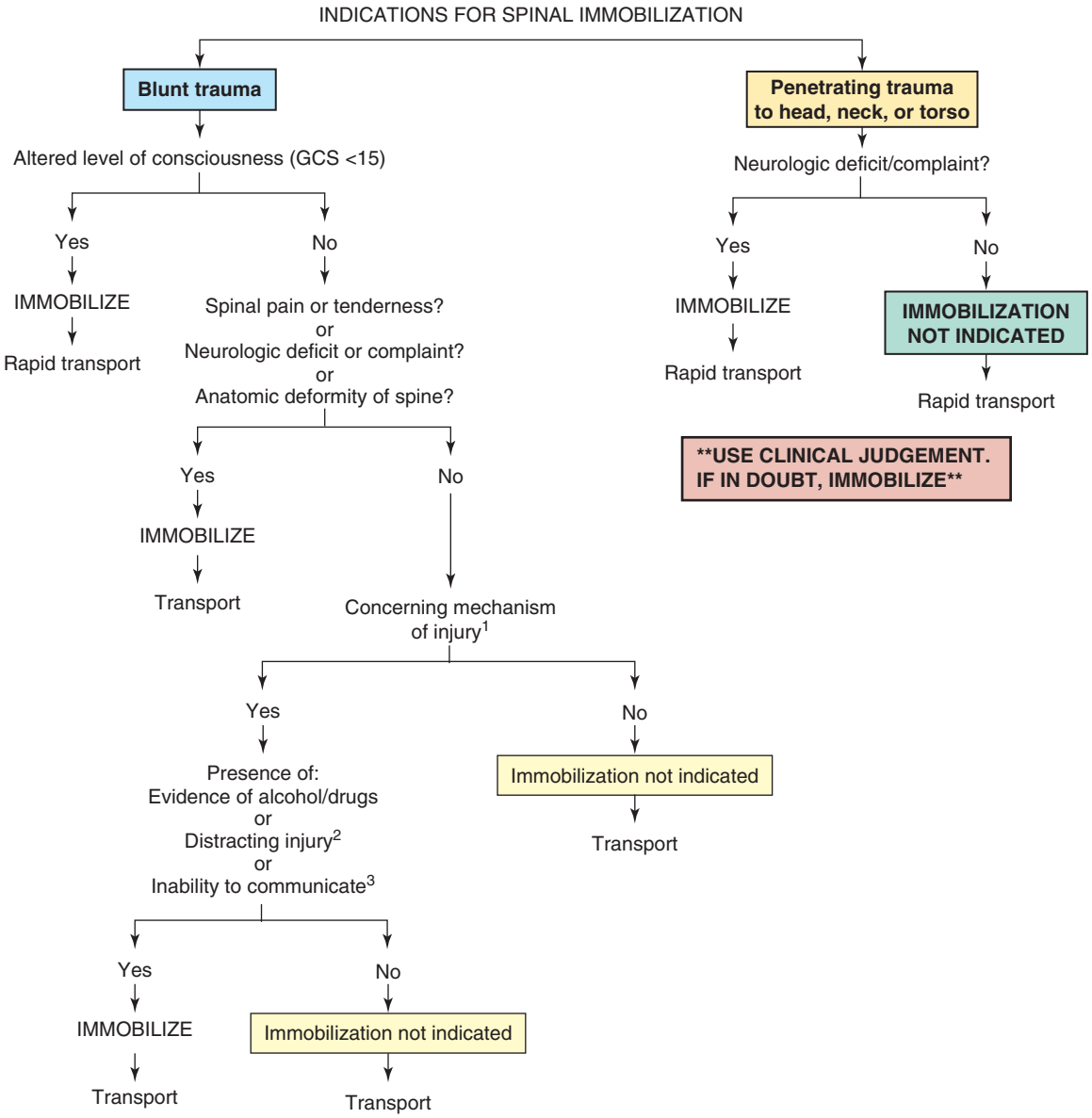
MUSCULOSKELETAL TRAUMA

Splinting. Hemorrhage is the only immediately life-threatening condition associated with trauma to an extremity (see Chapters 43–45). External hemorrhage should be controlled with direct pressure or a pressure dressing, followed by a tourniquet if these measures fail. Internal hemorrhage is best managed in the field by immobilization of the extremity. In the critically injured patient, immobilization to a long backboard is sufficient stabilization. If the patient does not have life-threatening injuries, time can be taken to splint each suspected fracture individually. A traction splint provides reasonable pain control and will stabilize a suspected fracture of the femur.

Amputation. ACSCOT has published guidelines for the management of amputated parts.[133] These include the following:

- Cleansing the amputated part by gentle rinsing with lactated Ringer's solution.
- Wrapping the part in sterile gauze moistened with lactated Ringer's solution and placing it in a plastic bag.
- Labeling the bag or container and placing it in an outer container filled with crushed ice.
- The part should not be allowed to freeze, and it should be transported along with the patient to the closest appropriate facility.

Pain Control. In the prehospital setting, analgesics are indicated for an isolated injury to an extremity, but not in a patient with multisystem trauma.[52,134] After appropriately splinting



Notes:
[1]Concerning mechanisms of injury
- Any mechanism that produced a violent impact to the head, neck, torso, or pelvis (e.g., assault, entrapment in structural collapse, etc.)
- Incidents producing sudden acceleration, deceleration, or lateral bending forces to the neck or torso (e.g., moderate- to high-speed MVC, pedestrian struck, involvement in an explosion, etc.)
- Any fall, especially in elderly persons
- Ejection or fall from any motorized or otherwise-powered transportation device (e.g., scooters, skateboards, bicycles, motor vehicles, motorcycles, or recreational vehicles)
- Victim of shallow-water diving incident

[2]Distracting injury
Any injury that may have the potential to impair the patient's ability to appreciate other injuries. Examples of distracting injuries include a) long bone fracture; b) a visceral injury requiring surgical consultation; c) a large laceration, degloving injury, or crush injury; d) large burns, or e) any other injury producing acute functional impairment.
(Adapted from Hoffman JR, Wolfson AB, Todd K, Mower WR: Selective cervical spine radiography in blunt trauma: methodology of the National Emergency X-Radiography Utilization Study [NEXUS], *Ann Emerg Med*. 1998 Oct;32(4):461–9.)

[3]Inability to communicate. Any patient who, for reasons not specified above, cannot clearly communicate so as to actively participate in their assessment. Examples: speech or hearing impaired, those who only speak a foreign language, and small children.

FIGURE 10-6 Indications for spinal immobilization. GCS, Glasgow Coma Scale; MVC, motor vehicle crash. (Reproduced with permission from Salomone JP, Pons PT, McSwain NE, eds. *PHTLS: Prehospital Trauma Life Support*. 7th ed. St. Louis, MO: Mosby; 2011:257. Copyright © Elsevier.)

the extremity, small doses of narcotics titrated intravenously may help relieve pain. The patient should be observed for side effects including hypotension and respiratory depression. Narcotics should not be administered in the trauma patient who exhibits signs of shock or when the patient appears to be under the influence of drugs and alcohol.

Medications other than narcotics have become commonplace for pain control in the prehospital environment. Ketamine has achieved a higher level of use as a result of benefits of sedation, pain control, and decreased posttraumatic stress disorder (PTSD) after traumatic events. Ketamine as a pain medication has been appreciated for its ease of use, efficacy, and multiple routes of administration, including intranasally.[135-137] Proposals that ketamine reduces rates of PTSD highlight a future area of study for its use in prehospital traumatic injuries.[138]

TRIAGE

Disasters may be the result of natural phenomena, such as tornadoes, hurricanes, and earthquakes, or manmade in the case of a building collapse or terrorist event. When situations such as these occur, the EMS and health care systems must try to match their resources with the needs of the community. A *multiple-patient incident* refers to a situation where numerous individuals may be injured but the EMS system possesses the ability to provide adequate care. In a *mass casualty incident*, the number of injured patients overwhelms the resources of the community, and additional aid from other locations is required. The number of patients involved in each of these circumstances may vary, depending on the size of the EMS system and the resources of the community.

In either situation, EMS personnel and health care workers must employ the principles of triage so that the most seriously injured patients receive care before those with minor injuries. Simple Triage and Rapid Treatment (START) is an easily taught, simple triage system that is widely used by EMS providers when faced with numerous injured patients.[52,129,139] A national expert panel, assembled by the CDC, reviewed all available triage schemes ("SALT" [*s*ort, *a*ssess, *l*ifesaving interventions, *t*reatment/*t*ransport]) and proposed a new method composed of the best components of the others.[140] A more comprehensive discussion of disaster care is presented in Chapter 12.

HAND-OFF AND TRANSITION OF CARE TO HOSPITAL

It is imperative for EMS personnel to give a clearly communicated, concise, and thorough report to personnel at the trauma center. The hand-off or report about the patient from EMS to the hospital is a potentially dangerous situation with implications for mistreatment of the patient if the prehospital interventions are not understood at the receiving facility. Every effort should be made to transmit what previous care was rendered. A system-wide standardized hand-off method is encouraged to promote exchange of vital information. This should include, but not be limited to, any abnormal vital signs, mechanisms of injury, care provided, and, especially, medications that may alter the patient's sensorium.

PREHOSPITAL RESEARCH

Over the past decade, the entire health care field has seen a growing trend toward evidence-based medicine. EMS, in general, and prehospital trauma care, specifically, require high-quality research to separate sound medical practice from conjecture. Management of the airway and fluid therapy are two areas that deserve intensive investigation in the prehospital setting. Far too few treatment protocols in EMS are based on high-quality data. More commonly, protocols are written that include the biases of the authors and extrapolations from in-hospital care.

Several major obstacles to the development of quality EMS research exist.[14] Funding is woefully inadequate, and integrated information systems are needed to link data on patient care with information on outcome. Few academic institutions possess a long-term commitment to EMS research. Governmental regulations regarding informed consent inhibit the conduct of research in the emergency situation, as well. As these challenges are overcome, new research will lead us to better care for our patients. NHTSA has published a document detailing numerous issues in prehospital care that are awaiting investigation.[16]

SUMMARY AND KEY POINTS

Prehospital management of the injured patient is in a state of transition. Few experts recommend a return to a mere "scoop and run" approach in which EMS personnel would leave the patient without support of vital functions during the transport to the hospital. Conversely, delays on the scene to perform unnecessary interventions are associated with increased mortality.[141] A recent publication compared trauma mortality before and after the implementation of a province-wide ALS program and found no significant change in survival between the two periods.[142] Although these findings could be the result of inexperienced ALS providers or inappropriate ALS protocols, it remains unclear which, if any, ALS procedures improve survival.

A more moderate approach that focuses on limited, key field interventions as taught in the PHTLS program is as follows:

- Rapid assessment to identify life-threatening injuries is critical.
- Key field interventions include management of the airway, ventilatory support with administration of oxygen, and control of external hemorrhage, among others.
- Rapid transport to the closest appropriate facility is essential.
- Prehospital trauma care must be part of an integrated regional trauma and EMS system (Table 10-4).
- High-quality research is desperately required in the field of prehospital trauma care.

TABLE 10-4: Golden Principles of Prehospital Trauma Care

1. Ensure the safety of the prehospital care providers and the patient.
2. Assess the scene situation to determine the need for additional resources.
3. Recognize the kinematics that produced the injuries.
4. Use the primary survey approach to identify life-threatening conditions.
5. Provide appropriate airway management while maintaining cervical spine stabilization.
6. Support ventilation and deliver oxygen to maintain an Spo_2 ≥95%.
7. Control any significant external hemorrhage.
8. Provide basic shock therapy, including appropriately splinting musculoskeletal injuries and restoring and maintaining normal body temperature.
9. Maintain manual spine stabilization until the patient is immobilized on a long backboard.
10. For critically injured trauma patients, initiate transport to the closest appropriate facility within 10 min of arrival on scene.
11. Initiate warmed, intravenous fluid replacement en route to the receiving facility.
12. Ascertain the patient's medical history and perform a secondary survey when life-threatening conditions have been satisfactorily managed or have been ruled out.
13. Provide thorough and accurate communication regarding the patient and the circumstances of the injury to the receiving facility.
14. Above all, do no further harm.

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11

Rural Trauma

Juan C. Duchesne • John P. Hunt • Alan B. Marr

KEY POINTS

- By the World Bank's calculation, 45.7% of the world's population still lives in a rural area.
- In the United States, at least 31% of rural residents live more than an hour from a Level I to III trauma center.
- In one study in a rural area, patients transported directly to the Level I trauma center from the field arrived 1.6 hours after injury, compared to 5.3 hours when patients were first sent to a local hospital that was not a trauma center.
- Recruiting and retaining emergency medical personnel in rural areas is difficult because of low wages, lack of training opportunities, and the need to cover a broad geographic region.
- The Rural Trauma Team Development Course of the American College of Surgeons is aimed at small hospitals without surgical capability and only nonsurgical personnel to treat the occasional injured patient.
- According to the National Highway Traffic Safety Administration, rural fatal crashes account for 61% of all traffic fatalities but only 39% of vehicle miles traveled.
- Many of the most dangerous occupations are found in rural areas, most notably mining and agriculture.
- The percentage of homes that experience a fire and have a working smoke alarm is 41.8% in urban areas but only 20.8% in rural areas.

HISTORICAL PERSPECTIVE

Injury continues to be a leading cause of death and disability throughout the United States; however, there are significant differences between injuries that occur in rural versus urban settings. Rural environments account for over 85% of the North American land mass and roughly 62.5 million people live in rural areas. Although less than one-fourth of the population lives in rural areas, the population in these regions accounts for a disproportionate number of trauma-related deaths.[1-4]

There are some key factor differences to consider when dealing with trauma patients in rural locations. They are typically older, less severely injured, and more likely to die at the scene than urban patients. The majority of trauma in rural settings is blunt injury, with fewer penetrating injuries, whereas the reverse is true in some urban locations. Similarly, the fatal crash rate is more than two times higher in rural than urban areas, with a rural rate of approximately 2.4 deaths per 100 million vehicle miles traveled.[5-7] The factors leading to these higher injury mortality rates include a lower rate of use of protective devices, greater severity of crashes due to higher speeds, and prolonged discovery time.[8-11]

Another important geographic disparity in these vulnerable populations is access to rural trauma care.[12] Rural may be defined in accordance with census data based on metropolitan areas, in terms of geography and distance, or by virtue of limited resources. In an analysis of the general surgery workforce, Thompson et al[13] identified significant differences between communities with a population between 10,000 and 50,000 (large rural) and those with 2500 to 10,000 residents. Large rural towns are far more likely to have the necessary resources such as general surgery, medical and surgical subspecialties, advanced life support ambulance services, and essential equipment to provide prompt and sophisticated trauma care.

In addition to longer distances to appropriate health care, health services in rural areas differ from those in urban areas, with fewer physicians and specialists per capita to furnish comprehensive trauma care. As a result, implementation of a systematic inclusive trauma system with outreach trauma education that focuses on injury prevention can potentially lower the incidence of rural trauma and improve outcomes in rural trauma hospitals.[14-18]

Although efforts in injury prevention might reduce the frequency or severity of injury in rural environments, it is

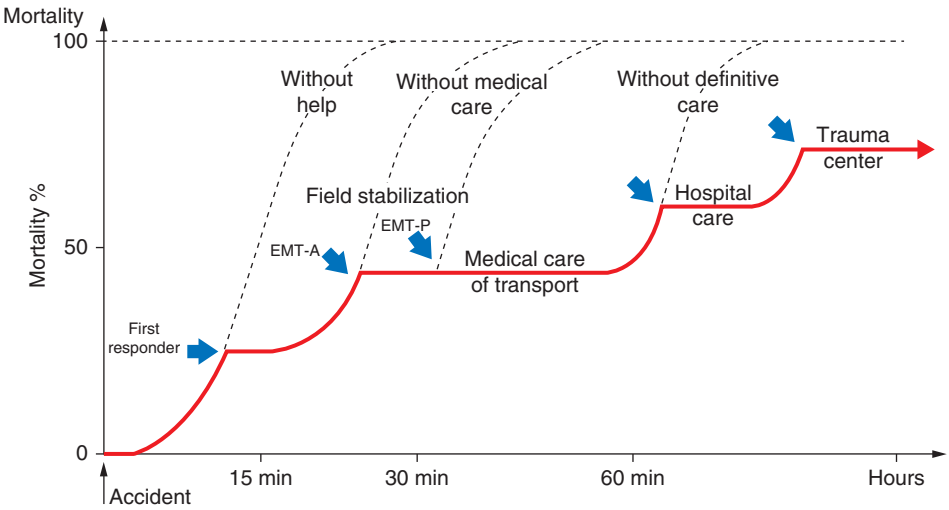

FIGURE 11-1 Mortality of trauma against time with field resuscitation and definitive care. EMT-A, emergency medical technician-advanced; EMT-P, emergency medical technician-paramedic. (Reproduced with permission from Boyd DR. Trauma: a controllable disease in the 1980s [Fourth Annual Stone Lecture, American Trauma Society]. *J Trauma.* 1980;20:14-24.)

evident that hospital-based interventions directed to improve injury care among rural trauma patients play a significant role in reducing mortality. As a result, the American College of Surgeons Committee on Trauma has designated trauma centers into Levels I to IV, with Level I or II trauma centers having the most comprehensive resources required to provide high-level trauma care in addition to trauma education and outreach. Trauma care at a Level I center has been shown to reduce risk of mortality in the severely injured by 25%.[8] Unfortunately, most advanced trauma centers are not distributed evenly across the nation, and approximately 45 million Americans do not live within 1 hour of the highest level of trauma care. This creates a marked rural-urban time-dependent disparity in access, with at least 31% of rural residents living more than an hour from a Level I to III trauma center compared to only 12% of urban residents.[19,20]

Rural trauma outcomes are time dependent. With approximately 500 verified or designated Level I and Level II trauma centers created for definitive trauma care and 1045 rotary-wing helicopters currently stationed at 879 bases in hospitals or airports to transport scene patients to trauma centers,[21,22] the key and still immediate determinant in rural trauma care survival is place of injury (Fig. 11-1).[23] In addition, there needs to be an established mechanism for fast patient transport with interfacility agreements developed by a regional inclusive trauma system.

The biggest benefit of an inclusive regional trauma system is to ease the transfer of injured patients from non–trauma center hospitals to higher echelon levels of care. Statewide inclusive regional trauma systems have demonstrated improvement in injury-related mortality as compared to states with less developed trauma systems. Currently, 9 states and districts lack a statewide trauma system and, of the remaining 42 states, only 24 have state-funded trauma systems. In this chapter, we will provide a current description of today's rural trauma care with suggested improvements, evidence of persistent disparities between urban and rural systems, and future improvements in education in an inclusive trauma system.[24,25]

DEFINING RURAL

Assessing the needs of rural populations is exceedingly difficult because of the lack of a uniform and internationally accepted definition of the term *rural*. Most international organizations rely on definitions set by nation states. By the World Bank's calculation, using United Nations data available in 2017, 45.7% of the world's population still lives in a rural area[26]; however, as demonstrated globally in Fig. 11-2,[27] enormous amounts of land still have very low population densities. In 2005, one study demonstrated that only 3% of the earth's land mass would be considered urban, although it contained nearly half of the planet's population.[28] By any definition, there are large numbers of people who live in rural areas worldwide, and these areas cover vast expanses.

There are numerous ways to determine whether a place is urban or rural. The most rudimentary is probably the simple calculation of population density. This requires determining a population for a given area of land. In the United States, the typical unit of area is the square mile, and the population for each area is counted every 10 years in the national census. Areas of similar population densities are aggregated to produce a map reflective of population densities of larger areas within the country (Fig. 11-3).[29] This representation clearly shows that there are still large areas of the country that are sparsely populated. Although most are in the Midwest and West, there are still large regions of the South and even areas of the Northeast that are rural.

Although useful, the crude population density mapping technique lacks some detail that may be useful in planning for delivery of medical and other services vital to a modern community. Although the Census Bureau and its

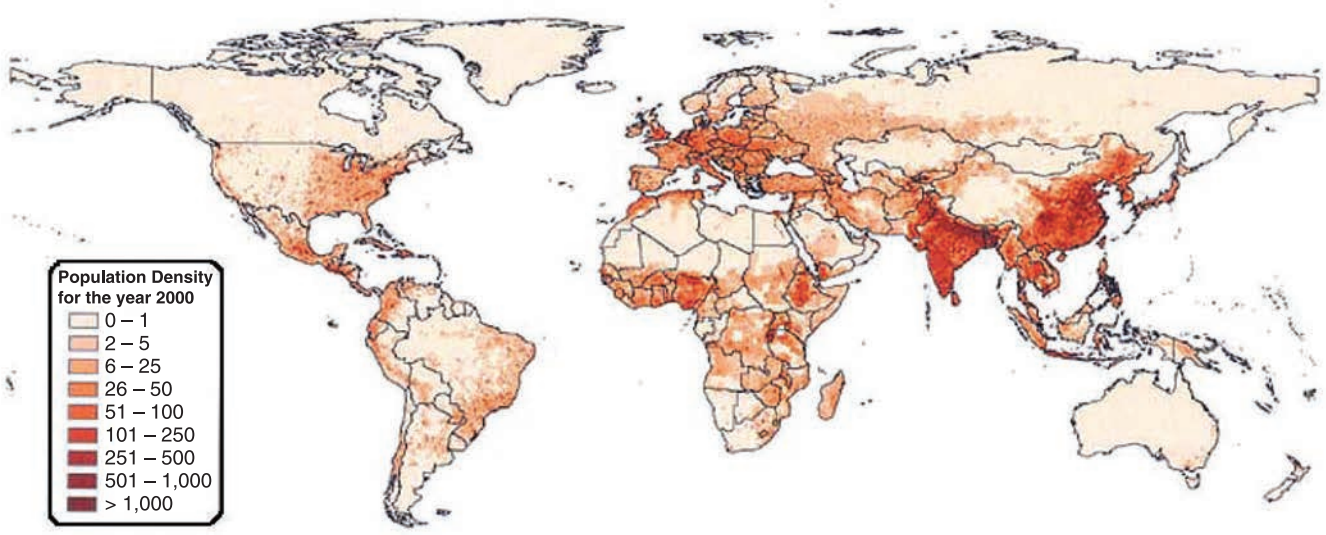

FIGURE 11-2 Map demonstrating population density for the year 2000. (Reproduced with permission from Food and Agriculture Organization of the United Nations [FAO]. Salvatore M, Pozzi F, Ataman E et al. *Mapping Global Urban and Rural Population Distributions.* Rome: FAO; 2005.)

forerunners were originally charged with the population count for the purposes of assigning the number of representatives to Congress,[30] over the course of 225 years, their data collecting has been expanded and used for other governmental functions. Historically, the US Census Bureau has never actually defined what "rural" is, other than all the land that is not classified as urban. Although the definition of urban has changed over time to include more people and land, the definition of rural remains all "territory, persons, and housing units not defined as urban."[31]

In 2000, the Census Bureau expanded the classification of urban regions. This included "urbanized areas," which contain over 50,000 persons, and "urban clusters," which are areas with at least 2500 but fewer than 50,000 people.

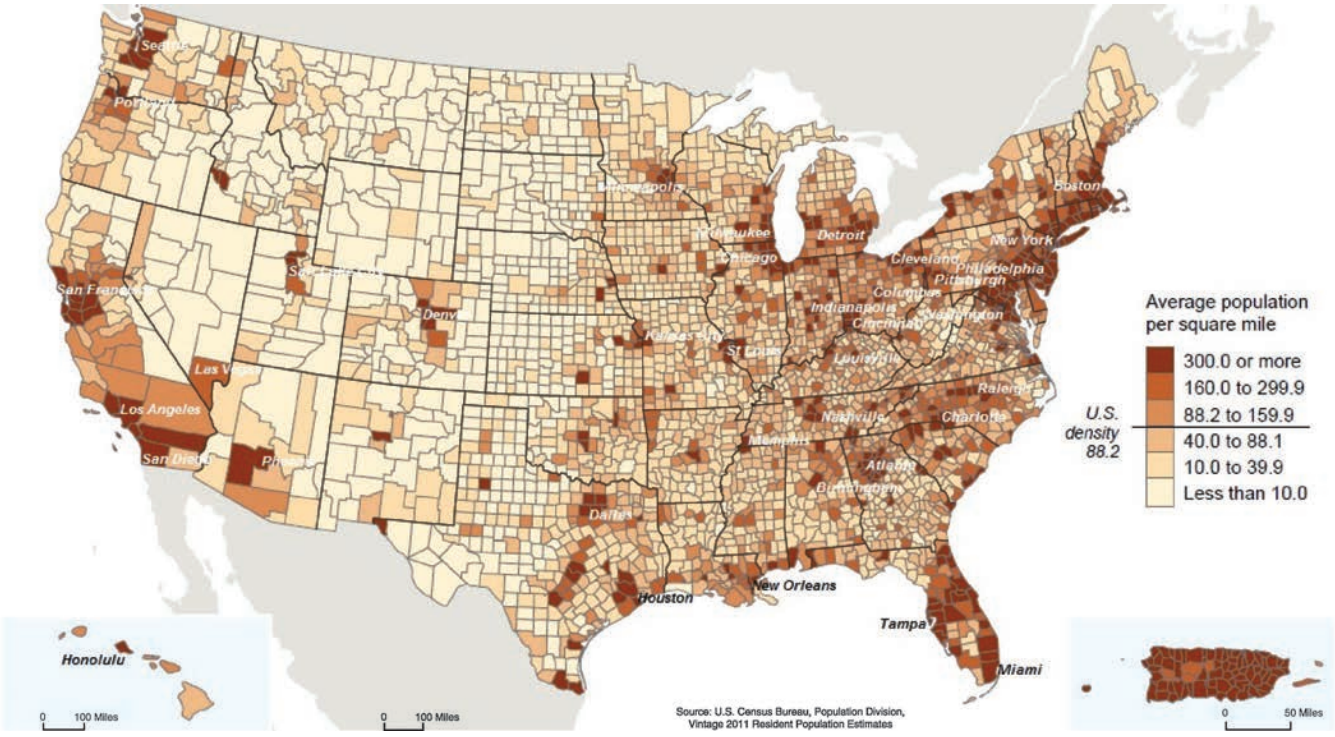

FIGURE 11-3 Map describing average population per square mile. Large sparsely populated areas remain in the Midwest and West, as well as the South and even areas of the Northeast. (US Census Bureau. Maps. Retrieved from https://www.census.gov/history/www/reference/maps/.)

Other factors such as population density, land use, and distance between urban clusters are also used to define the new urban area classifications.[32] In 2010, there were 486 urbanized areas and 3087 urban clusters in the United States.[33] This representation (Fig. 11-4)[34] provides more detail and may be useful in the planning of parts of trauma systems that may be integral to providing trauma care to rural areas.

Urban clusters are uniquely placed to be points of care to the sparsely populated regions of the country. They may not have the population to support larger tertiary care centers able to provide definitive care. They will, however, be indispensable in providing the initial phases of the continuum of trauma care. They could include a population center that can support a cadre of prehospital providers, a nontrauma hospital that can provide stabilizing care prior to transport to a trauma center, or in some cases, even Level III trauma centers that can provide definitive care to certain trauma patients. All of these elements are essential in bringing state-of-the-art trauma care to rural populations.

The challenges to bringing the best trauma care to rural citizens are not just related to the great distances between patients and definitive care. Rural areas are frequently starved of resources. Due to economies of scale, it is often difficult to bring more expensive care and technology to a smaller number of people. This is compounded by the fact that rural households, on average, earn almost 5% less than their urban counterparts.[35] Rural areas are frequently sites of inhospitable climates, which interfere with effective retrieval and transport. In addition, they often have geographic features such as mountains, which hamper radio communications and air travel, or bodies of water that will require special equipment for rescue and marine transport.

The challenge will be varied, and the solutions will depend on the environment and the resources available. It is imperative that attention and funds be applied to trauma care for the

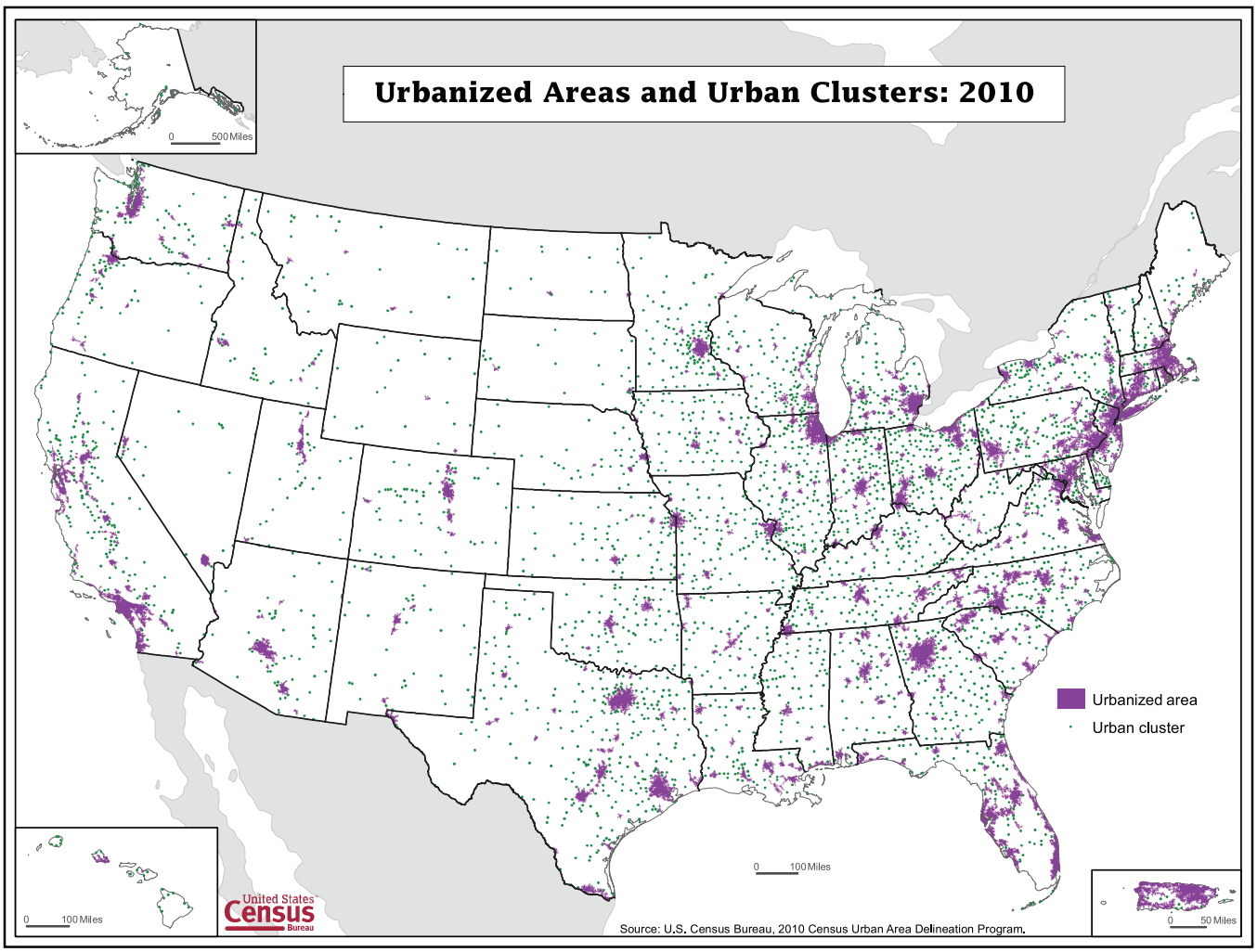

FIGURE 11-4 Understanding urbanized areas and urban clusters can be useful in the planning of trauma systems in rural areas. (US Census Bureau. Defining rural at the U.S. Census Bureau. Retrieved from https://www2.census.gov/geo/pdfs/reference/ua/Defining_Rural.pdf.)

rural population. In 2016, John H. Thompson, the Census Bureau Director, stated, "Rural areas cover 97 percent of the nation's land area and contain nearly 20 percent of the population."[36] That is approximately 60 million Americans.

THE NEED FOR REGIONAL INCLUSIVE TRAUMA SYSTEMS

As noted earlier, numerous challenges confront officials trying to render state-of-the-art trauma care to rural populations.[37] All of these factors can contribute to worse outcomes in the rural trauma patient. In one study in Ontario, Canada, deaths in the field were twice as frequent in rural areas of the province, and there was a threefold increase in the mortality rate for patients who had limited access to trauma center care.[38] This is notable in some particularly at-risk patient populations. For example, geriatric trauma patients admitted to rural hospitals have a significantly increased mortality when compared to their urban counterparts.[39]

In addition, it has been effectively demonstrated that trauma patients with an increased transport time have a higher rate of complications and length of stay.[40] Rural transport times are by definition longer when compared to those of urban cohorts. One study of Scottish trauma patients demonstrated increases in prehospital times in rural regions when compared to urban areas. This is true of response time, scene time, and transport time.[41] In one statewide study in the United States, the mean emergency medical services (EMS) response time was 13.9 minutes in the rural areas compared to 6.8 minutes in urban settings. This, in turn, corresponds to an average 10.5-mile response distance in rural settings versus a 7.7-mile distance in urban settings. This creates a twofold increase in mortality for rural trauma patients when compared to the urban trauma patient population and has been observed in other rural and urban comparisons.[42]

In addition to distance, the environment itself contributes to less favorable outcomes. In one rural Level I trauma center over a 5-year period, 1490 (15.7%) of 9482 adult patients admitted were hypothermic. Hypothermia was associated with a nearly twofold risk for mortality and significantly increased hospital days.[43] The only means to bring effective care to patients in rural regions in the face of the burden imposed by the frequently austere environments, geographic obstacles, distances involved, and a relative lack of resources is a well-functioning and orderly trauma system. The American College of Surgeons defines a *trauma system* as a trauma care delivery structure that addresses all aspects along the continuum of trauma care.[44] This includes all components of EMS notification, dispatch, prehospital instruction, and transport. It also involves initial hospital care and interfacility transport and then trauma center care and rehabilitation.

In the severely injured, definitive care will often be carried out in a trauma center away from the rural environment. It is the support of the rural medical community, through education, provision of resources, and a seamless integration with centers that can provide a higher level of care, that makes the trauma system essential for improved outcomes in the rural trauma patient. In numerous rural communities across the globe, the implementation of a trauma system has yielded clinical benefits. The introduction of a trauma system in rural Victoria, Australia, resulted in a marked decrease in preventable and potentially preventable deaths.[45] A similar implementation of the Iowa Trauma System noted a significant decrease in the mortality of patients with traumatic brain injuries treated at receiving Level I or II trauma centers.[46]

Benefits are realized through a variety of mechanisms that improve quality of care and streamline access to definitive care in the initial care of the critically injured. At the prehospital and first-responder levels, education is frequently provided through the trauma system. Emergency medical technician (EMT) and paramedic courses are often provided in urban centers and attended by providers from more rural areas. The effectiveness of trained prehospital providers in the United States is often overlooked and taken for granted because they have been in place for a long period. In similar fashion, new prehospital efforts in developing countries yield results from prehospital provider education that are impressive. In the developing regions of Kurdistan, Iraq, and rural Cambodia, a program that enlisted lay persons trained in basic first aid and paramedics trained in Advanced Trauma Life Support (ATLS) made appreciable strides in outcomes. They were able to reduce the mortality from landmine injuries from 23.9% to 8.8% over a 5-year period.[47]

In a similar environment in Iraq, a low-tech prehospital system was developed that decreased time to first treatment from 2.4 hours to 0.6 hour, and the system was durable, as demonstrated by continued proficiency as the mechanism of injury changed from largely penetrating to mixed penetrating and blunt.[48] Another prehospital system consisting of 135 paramedics and 7000 layperson first responders was set up in the mine and war zones in Iraq, where prehospital evacuation times are long. The mortality rate significantly dropped from 15.6% to 9.8% among patients initially managed in the field by first responders compared to patients without first-responder support.[49] In addition, prehospital personnel in the United States trained in the use of tourniquets and application of hemostatic combat gauze have been noted to use the technique and product with demonstrable effectiveness.[50]

Another mechanism by which trauma systems help prehospital personnel is with the coordination of care. The institution of standard and simplified triage tools, developed by the military, that rely on easily collected variables such as respiratory rate and abbreviated mental status evaluations, has been shown to be reliable in identifying critically injured patients.[51] One advantage of the trauma system is that there is an accurate accounting of what services can be provided by which hospitals. Prehospital providers can then be directed toward the right receiving center for the injuries noted at the scene. In one study of rural trauma patients, half of whom were transported directly to the Level I trauma center and half of whom were taken to a local hospital, prehospital transport costs were significantly greater for patients transported to a rural hospital first. In addition, many severely injured patients were initially transported to a rural hospital rather

than directly to the trauma center. At both the scene and rural hospital, consistent use of triage criteria appeared to be lacking in determining the severity of injury, appropriate destination, and mode of transport for trauma patients.[52]

The system provides an infrastructure for standardized field triage protocols and direction of patients to appropriate hospitals. Louisiana was able to demonstrate a sixfold reduction in the secondary transfer rate when a statewide call center implemented a system with standard field triage criteria and real-time data on resources available at the state's hospitals.[53] With this information, a central command center could appropriately assign ground, helicopter, or fixed-wing units to respond. Using global positioning and mapping technology to compare one-way road transfer times with two-way helicopter retrievals in 171 helicopter fights in rural Australia, air transport was significantly faster beyond 100 km, with a mean difference of 48 minutes. The average calculated distance flown was 160.4 nautical miles.[54] Fixed-wing transports are typically used for distances greater than 250 miles. With on-the-ground knowledge of patient status and real-time information on hospital resources, the system can better assign the mode of transport to avoid delay. A 4-year retrospective review of pediatric trauma helicopter transports to area trauma centers, including 130 rural and 419 urban pediatric patients, showed greater total mileage, flight times, and scene times for rural flights. The rural times, however, would have been extraordinary if ground transport was used.[55]

The system can also provide assistance at the physician level. Academic centers are frequently the teachers of physician-level courses, such as the American College of Surgeons ATLS course, which educates physicians in the rudiments of initial trauma care. In a 5-year study of nearly 5000 patients initially treated at rural Level III trauma centers in a single state, several components of the ATLS program were found to confer a mortality advantage. These included patients needing blood products in the emergency department for a systolic blood pressure less than 90 mm Hg, patients needing transfer to higher level of care with an Injury Severity Score (ISS) greater than 20, and patients with traumatic brain injuries.[56] Seriously injured patients treated at nine rural Level III and Level IV hospitals were studied before and after implementation of Oregon's trauma system. The system mandated ATLS training for emergency physicians. Twenty diagnostic and therapeutic interventions, taught in the ATLS course, were evaluated before and after ATLS was mandated. Processes of care for seriously injured patients significantly improved after categorization of rural trauma centers in Oregon.[57]

The uniform training of these emergency physicians also translates into fewer delays in care and a more efficient and rapid movement to definitive care. In one study, nearly half of the referring physicians obtained unwarranted imaging because they felt it was a requirement or because of fear of liability. Physicians trained in ATLS are significantly less likely to obtain unneeded imaging that delays transfer to definitive care.[58] In another study of rural trauma patients requiring transfer, 75% of trauma patients had computed tomography (CT) scans at outside referring hospitals. Of those, 58% transferred to a higher level of care had to have repeat imaging.[59] Undertriage can also create serious delays to definitive care. In one study at a Level I trauma center, all patients who were treated at two hospitals prior to arriving at the trauma center were evaluated. Patients were mostly transferred to the Level I trauma center for nonspine orthopedic injuries (28%), followed by spine injuries (14%) and traumatic brain injuries (13%). These patients had serious or complex injuries and took longer to evaluate. A system-directed refining of transfer protocols concerning specialty care and a central command aware of where these services are available have the potential to decrease secondary transfers and shorten the time to definitive care.[60] When Oregon initiated the state trauma system and mandated ATLS for all emergency physicians, transfers from Level IV rural trauma centers to a higher level of care increased by fourfold.[61]

The balance that must be maintained for the rural hospital is to recognize limitations and when transfer to a higher level of care is needed. Considerations always revolve around appropriate care, but also must take into account inappropriate transfers. These can take patients a distance from their families and overtax the trauma center with patients who could have been taken care of at a local facility. In one study, injury-adjusted mortality rates were significantly lower in patients treated at rural hospital emergency departments and transferred to Level I and II trauma centers than those who were admitted and treated at rural hospitals.[62] In another study, direct transport to higher level trauma centers had a beneficial impact on mortality when compared to patients who were treated at lower level facilities and transferred to a higher level of care.[63]

Transfers after initial assessment can be time consuming. In one study in rural Pennsylvania, patients transported directly to the Level I trauma center from the field arrived 1.6 hours after injury, compared to 5.3 hours after injury when patients were first sent to a local hospital that was not a trauma center.[64] Another study in a rural Vermont Level I trauma center also compared trauma patients admitted directly with those initially stabilized at an outlying hospital. Transfer patients spent an average of just over 3 hours at the outlying hospital and 72 minutes in transport to the trauma center. The transfer population was more severely injured than the trauma patients admitted directly and may have been better served by being brought directly to the trauma center.[65] These delays may be responsible for poorer outcomes and should always be minimized when possible. Most systems will have organized triage protocols and preexisting destination protocols to limit the variability that leads to these delays.

Another important system function is trauma center development and the promulgation of an inclusive system. Level I and II centers often naturally develop in cities and in population centers that will support tertiary care in general. It is frequently in the smaller rural cities, towns, and even villages where it is most difficult to get appropriate hospital resources marshalled toward the smaller volume of trauma patients. Community size, hospital size, and resources and

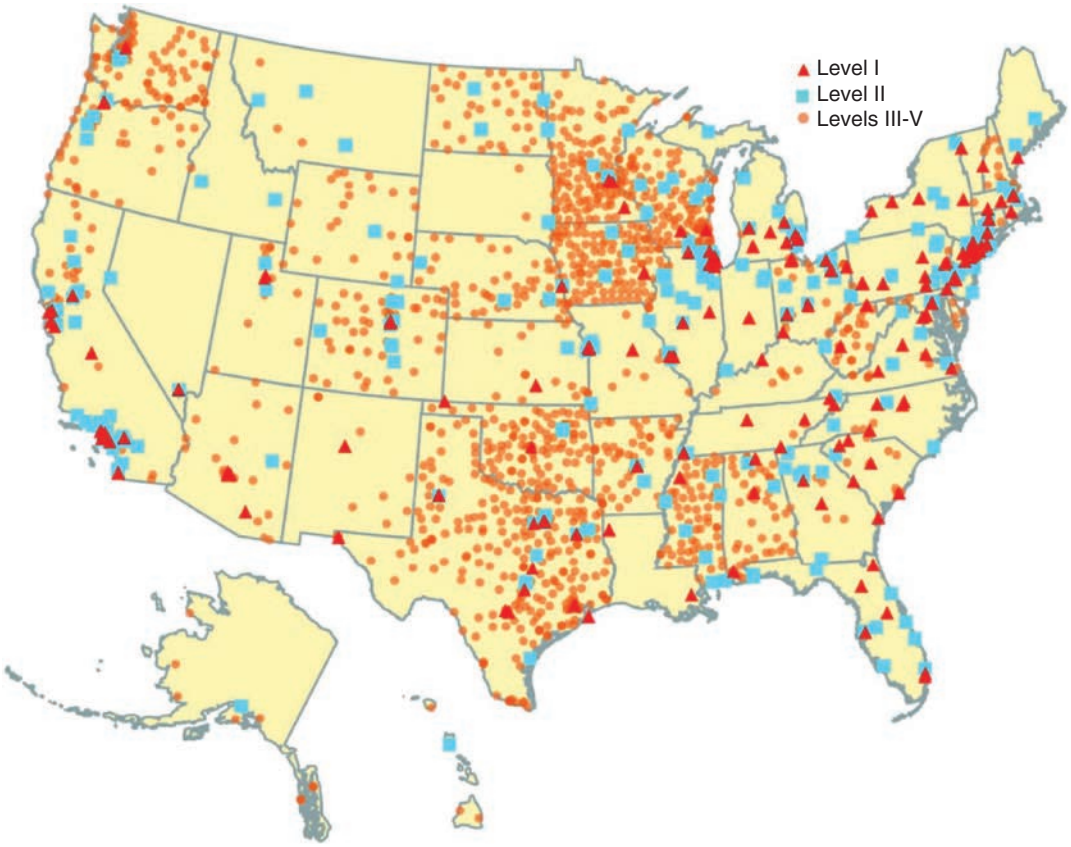

FIGURE 11-5 Distribution of recognized trauma centers is not uniform and clearly follows the population density. (Reproduced with permission from Eastman AB, MacKenzie EJ, Nathens AB. Sustaining a coordinated, regional approach to trauma and emergency care is critical to patient health care needs. *Health Aff (Millwood)*. 2013;32(12):2091-2098. Permission from Project HOPE conveyed through Copyright Clearance Center, Inc.)

personnel available will dictate the role of each acute care hospital.

The current distribution of recognized trauma centers is not uniform and clearly follows the population density discussed previously (Fig. 11-5). Most Level I and II centers are in the major metropolitan areas and large towns. Level III through V centers are much more randomly dispersed but clearly clustered in certain states. The systems in these states have been active in development and verification of lower level trauma centers; however, this may not be necessary at the Level III to V hospitals. In a study comparing two western states, Washington and Oregon, there was no improvement in outcomes between Level III, IV, and V trauma centers that were formally categorized or verified and those that were not.[66] Inclusion and active participation may be all that is required and may be attractive to hospitals that do not want to have the cost of verification. Preexisting structures, whether they are at the hospital corporation level or at a higher level such as the critical access hospital system (Fig. 11-6), may also be used to develop a trauma system that is inclusive of even the smallest hospitals and helpful to rural communities. The state of Kansas, a largely rural state, has used the existing Medicare-supported critical access hospital system and its referral pattern to support a trauma system that covers the entire state. Ten in- and out-of-state support hospitals and six designated trauma centers support the 83 critical access hospitals.[67] There may, however, be limitations to this system. In a nationwide study over 5 years of patients hospitalized with moderate to major traumatic injury in nonfederal, short-stay rural hospitals with annual discharges of 1500 or fewer patients, in-hospital death was more likely among patients treated at the nondesignated hospitals with fewer than 500 discharges per year than among patients treated at similar trauma-designated hospitals.[68]

The role any given hospital will play is individualized to personnel and resources available and is likely to be variable across the region. In the 14 rural hospitals in New South Wales, Australia, general and orthopedic surgeons provided trauma care. Resident subspecialty surgeons were available in 43%, and half of hospitals had a formal hospital trauma team. In hospitals without a team, 86% stated that there was a lack of personnel to provide 24-hour coverage. In 71% of the hospitals, there was only a single doctor available after hours. The majority of hospitals had plain x-ray equipment, blood products, ultrasound, and CT, although always on an on-call basis after hours. All hospitals had the capability to maintain ventilated patients, averaging two ventilated beds per hospital. An average of two trauma patients per hospital per month were transferred to a larger institution.[69] The variability of this largely rural area could lead to gaps in care for the trauma

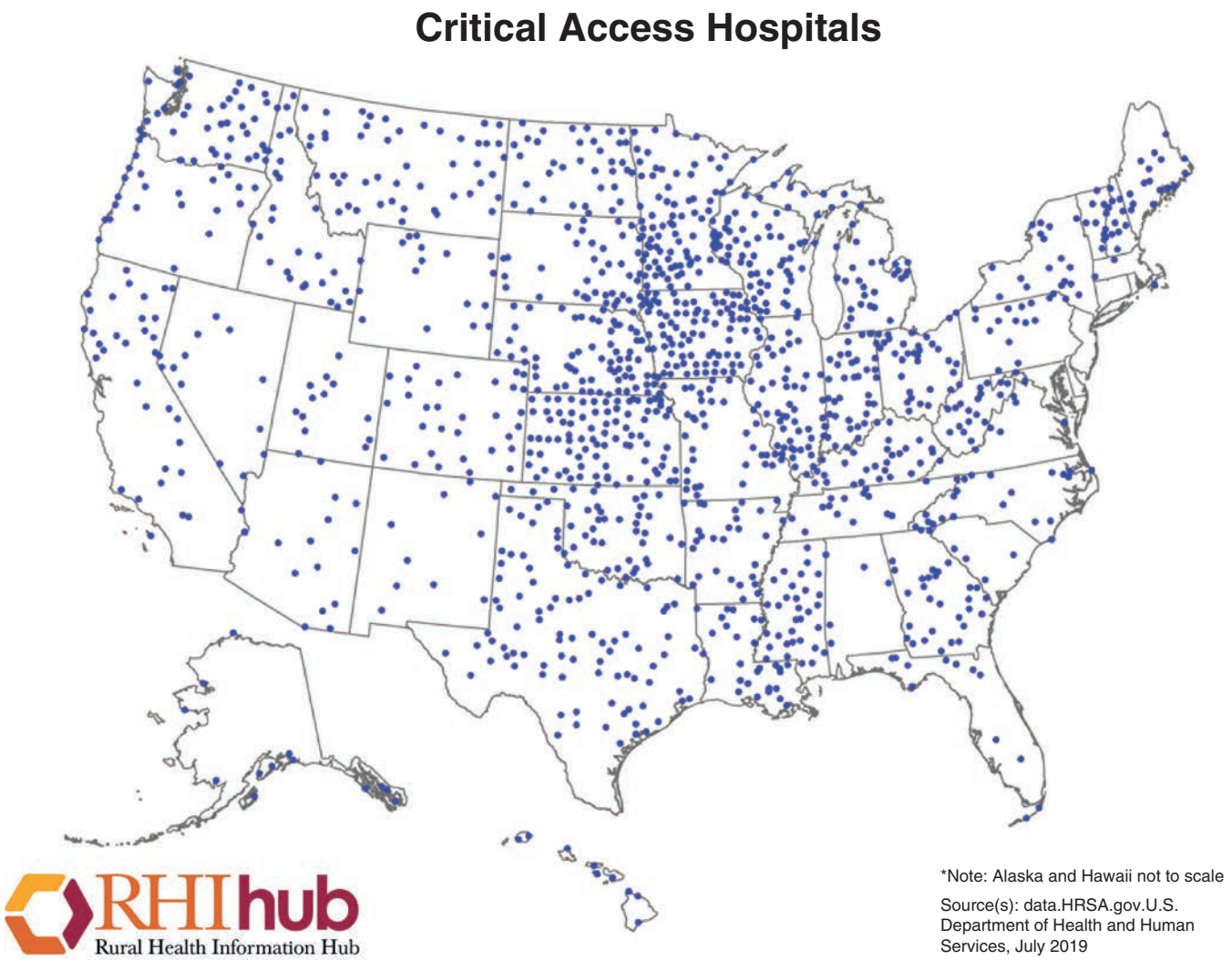

FIGURE 11-6 Critical access hospital system may also be employed to develop a trauma system that is inclusive of even the smallest hospitals. (HRSA Data Warehouse, US Department of Health and Human Services, November 2017. Reproduced with permission from the Rural Health Information Hub.)

patient. This is best managed by a system that has extensive knowledge of the resources across the system and can facilitate communication across the spectrum of prehospital and hospital care.

The spectrum of care that can be provided at lower level centers is subject to variation from hospital to hospital as well as within any given hospital. Whereas most operative procedures at rural Level III trauma centers will be orthopedic, the general surgeon may be called on to carry out lifesaving damage control procedures in patients who will be transferred on to a higher level of care. A study of the Missouri Trauma System showed that this could be performed safely.[70] Some hospitals with appropriate staffing and monitoring capabilities may also be employed to decrease unwarranted and costly transfers of patients who may be served better closer to home and family. In one study, patients with traumatic brain injuries admitted to a rural hospital without neurosurgical capability over a 2-year period had the scan reviewed via teleradiology at the regional neurosurgical referral center. The neurosurgeon on call and the trauma surgeon in the rural hospital then decided whether to transfer the patient to the neurosurgical referral center. Of 209 trauma victims with neurosurgical pathology in need of hospitalization, 126 (60.2%) were immediately transferred, whereas 83 of the patients (39.7%) were hospitalized in the rural Level II trauma center for observation. Only two patients (2.4%) failed the intent to treat locally and required transfer to the regional center later.[71]

Another function of the lower level center in the rural community could be to prevent overutilization of a single Level I or II center by avoiding the unnecessary transfer of trauma patients with relatively minor injuries. Rural patients can also suffer from a poorly integrated system because of an increased reliance on the rural trauma center. One such center in rural Iowa saw a twofold increase (11.8% to 21.2%) in the operative care of routine fractures in patients with Medicaid that could have been taken care of at a closer hospital. Of interest, the average distance traveled by patients with

simple injuries increased over time from 35.2 to 51 miles, and the distance was greater for Medicaid patients (59.7 vs 42.6 miles).[72]

One of the difficulties for personnel in lower trauma volume hospitals is the maintenance of proficiency. There have been several new efforts at addressing this problem. In one study, trauma providers from the same hospital always trained together as a team, consisting of one to three surgeons, two to three operating room nurses, an anesthesiologist, and one to two nurse anesthetists. The 19-hour course consisted of two interactive scenario-based lecture modules integrated with a hands-on operative experience on a live porcine model. Participants in the course reported improvement in proficiency with damage control procedures.[73] This method seems applicable to the Level III center, but the same issue exists at the lower level hospitals that do not even provide operative services.

The American College of Surgeons developed the Rural Trauma Team Development Course (RTTDC) for these institutions. This course is aimed at small hospitals without surgical capability and only nonsurgical personnel to treat the occasional trauma patient. It focuses on stabilization and transfer to definitive care. In one study, over 100 nurses trained in the RTTDC course found the course helpful. Over 95% felt the course increased their trauma knowledge, and there was a significant improvement in 13 of the 19 knowledge items tested. The RTTDC education intervention was an independent predictor in reducing time to transfer in the emergency department by 28 minutes. This was sustained at 6 months, with the emergency department time being 29 minutes less than the baseline.[74]

In another cohort, rural Level III and IV centers that used the RTTDC course were able to decrease the time from arrival to decision to transfer from 114 minutes to 95 minutes and the decision time to transfer to squad arrival time from 75 minutes to 65 minutes. This was further improved with the addition of training in effective communication techniques, trimming the arrival to decision time to 77 minutes and the decision to squad arrival time to 31 minutes.[75]

The RTTDC course can be taught by anyone with ATLS training and is a useful bridge for Level I and II centers to build ties with their rural counterparts. In one nonprofit hospital system in Colorado, a hub trauma center developed a trauma outreach team consisting of a systemwide medical director, a service line director, and a trauma nurse coordinator. They worked with a rural trauma program manager to facilitate integration of the rural center into the system. These efforts included consultation and support, education, connectivity, and expanded services and referrals. A total of 18 rural hospitals were initiated after 62 interventions/meetings.[76] This educationally based spoke-and-hub relationship has also been shown to improve communication within the system. A Level I trauma center in middle Tennessee taught the RTTDC course to six surrounding rural hospitals. The RTTDC group experienced an overall 61-minute reduction in length of stay in the referring hospital emergency department compared with the control group. The RTTDC group also showed a 41-minute reduction in time to call for transfer compared with control hospitals that did not participate in the course.[77]

As rural areas expand, distances lengthen, and resources become sparser, it becomes more difficult to maintain integration of a system. This is where the central command structure becomes essential. Knowing the strengths and weaknesses of the components of the system serving the rural population and integrating this with knowledge of weather, terrain, and limitations of available equipment are tested most in the rural environment. These are not constants across regions and systems. In one study over 2 years in adults with an ISS of 12 or greater who were transported to a Level I trauma center, air ambulance was faster than ground transport, with helicopters overall superior to fixed-wing aircraft, but limited by a 225-km (140-mile) range.[78] This may not be true, however, in another region with another set of circumstances. Over a 6-year period in Western Australia, a total of 1275 major trauma patients were treated at the Royal Perth Hospital, of whom 566 (44%) were from rural areas. For rural patients, the Royal Flying Doctor Service was responsible for 440 transfers (79%), of which 83% had a doctor and a nurse escort. A total of 450 rural patients (93%) were transferred within 24 hours of trauma, although the mean transfer time was over 9 hours.[79] However, what is possible in this fairly flat, arid region of a wealthy country may not be applicable or useful in a mountainous, poor region like Nepal.

An example of complicated and multifaceted trauma system coordination and patient flow would be the state of Hawaii. The only island state in the United States presents a challenge that the state system planners have met successfully. A patient with multisystem trauma, injured on the mountainous north coast of Kauai, would be met by local EMS personnel trained in mountain and water rescue and transported by ground to a predetermined rendezvous point. The patient would then be transferred to a helicopter transport team. Helicopter transport is necessary to get to the island's major city in a timely fashion. The single coast road is circuitous because it must traverse the island's perimeter and is frequently congested, whereas the island's interior is impassable due to its mountainous character. Once there, the patient would receive definitive or stabilizing treatment at the island's Level III trauma center depending on specific injuries. If the patient required specialty care such as neurosurgical intervention or consultation, he or she would be flown to the state's only Level II trauma center in Honolulu, Oahu, by a fixed-wing aircraft (Fig. 11-7).[80]

EPIDEMIOLOGY

Rural Disparities in Injury Rates

The National Center for Health Statistics published county-level injury rates in the 2001 *Urban and Rural Health Chartbook*, in which counties were classified according to their urbanization level (Fig. 11-8). The unintentional injury mortality rate for the most rural counties was 54.1 per 100,000 population, a rate almost two times higher than that observed in large metropolitan counties.

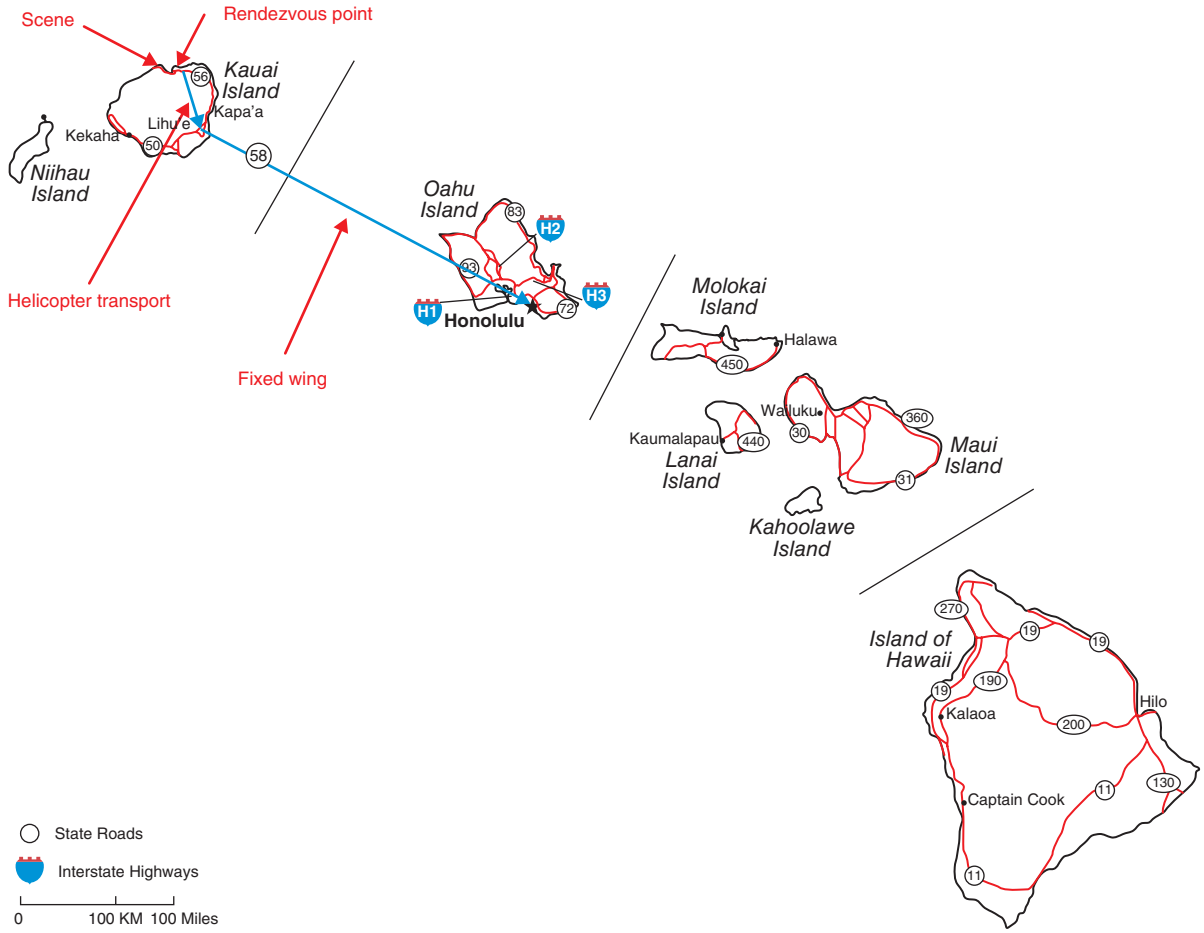

FIGURE 11-7 Hawaii islands trauma system.

The *Urban and Rural Health Chartbook* revealed similar trends in suicide deaths (Fig. 11-9). Relative to large fringe metropolitan counties, suicide death rates were 31% and 43% higher, respectively, in nonmetropolitan counties adjacent and not adjacent to small cities. Disparities were greatest among male residents of the western United States, whose suicide rate was 80% higher in rural areas than in metropolitan areas.[81]

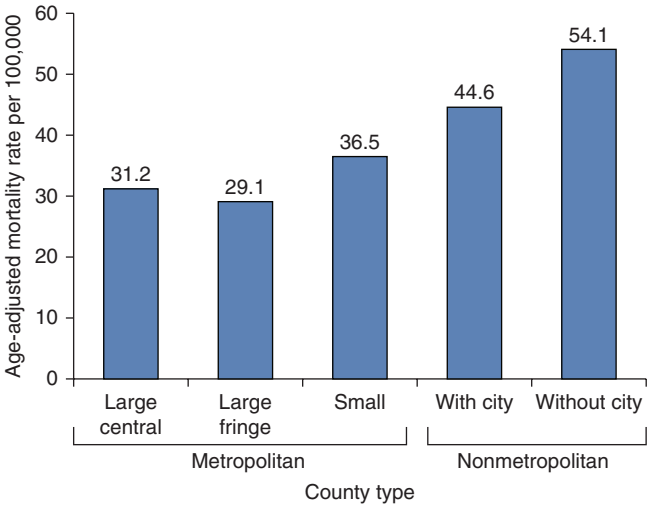

FIGURE 11-8 Unintentional traumatic injury death rates by urbanization level, 1996 to 1998.

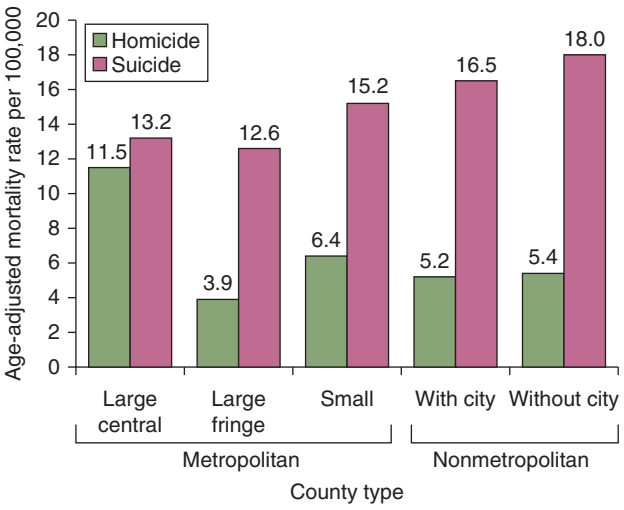

FIGURE 11-9 Homicide and suicide death rates by urbanization level, 1996 to 1998.

Although rates of both violent and property crime decreased from 1993 to 1998, this decrease occurred at a greater rate in urban and suburban areas than in rural areas.[82] This trend could indicate that prevention programs designed for urban populations have not been applied or have not been effective in rural areas.

Individual studies examining different types and mechanisms of nonfatal injury provide some information regarding rural disparities. An analysis of data from the Colorado Behavioral Risk Factor Surveillance System showed that the odds of a self-reported nonfatal injury were 30% higher among rural than among urban residents.[83] In the case of rural residents living in remote areas, the odds increased to 64%. Rates of traumatic brain injuries were shown to be higher in rural than in urban populations.[84] However, other studies that examined types of injuries, including overall fractures and hip fractures, showed rates to be lower in rural than in urban populations.[85,86] Studies have indicated that overall rates of serious firearm injuries are consistently higher in urban areas[87,88] but that unintentional firearm injury rates are higher among rural populations, perhaps driven by higher proportions of gun ownership.[89] At least one study has shown that safe storage of firearms in the home does not differ according to urban/rural status.[90]

Rural Disparities in Selected Mechanism of Injury

MOTOR VEHICLE INJURIES

Much of the increase in rural injury death rates is related to motor vehicle crashes. According to the National Highway Traffic Safety Administration, rural fatal crashes account for 61% of all traffic fatalities but only 39% of vehicle miles traveled, and the rural-urban difference is increasing over time. Crash-related mortality is inversely associated with population density and per-capita income. Analyses of motor vehicle crashes in several states have shown that fatality rates in the most rural counties are almost double those observed in urban counties.[91-97]

Certain types of crashes, such as those involving motor vehicle collisions with farm machinery, are unique to rural environments.[98] These crashes most frequently involve slow-moving tractors and are exacerbated by the high speeds at which vehicles often travel on rural roadways. In addition, other types of crashes, such as those involving all-terrain vehicles and snowmobiles, are more common in rural areas.

Roadmap Anatomy. Many factors may contribute to the increased fatality rates after motor vehicle crashes. Unlike interstate roads, rural highways are typically composed of two narrow lanes and do not have crash reduction features such as divided traffic streams, controlled vehicle entrances and exits, graded curves, skid-reducing surfaces, large lane and median widths, and lighted traffic signs. Head-on collisions, which most frequently occur when traffic streams are not separated and which are the most likely of all crash types to cause fatality and severe injury, account for 17% of fatal rural crashes and 9% of fatal urban crashes.[99]

Need for More Injury Prevention. Behavioral factors also differ according to population density. Rural residents are less likely than urban residents to wear seat belts or to use child safety seats,[91,94,95] and they are more likely to consume alcohol.[100] In addition, enforcement of traffic safety laws, such as laws against drunk driving and speeding, may be limited in some rural areas because of the reduced density of traffic enforcement officials.

WORK-RELATED INJURIES

Occupational fatality rates after trauma are higher among rural than urban populations. For example, in comparison with the national average, injury mortality rates in the construction industry are 40% higher in predominantly rural states.[101] Many of the most dangerous occupations are found in rural areas, most notably mining and agriculture.[102-105] In 2002, the mining industry had the highest occupational fatality rate, 29.1 per 100,000 workers, followed by agriculture, at 21.0.[106] Mining workers suffered approximately 10,000 disabling injuries, and agricultural workers experienced approximately 150,000 such injuries. Each year, approximately 10% of farmers are injured while working.[107,108]

Agricultural Injuries. Major initiatives established by the National Institute for Occupational Safety and Health have identified agricultural risks and potential approaches to prevention.[109,110] Research on agricultural injuries indicates that major injury risks are linked to working with animals and machinery, especially tractors.[111-114] Injuries are often multiple and severe, resulting in substantial disability, and compensation is less available for farm injuries than for non–farm-related occupational injuries.[115-117]

In addition to agricultural work, farms involve hazards for the entire family, because work areas and work tasks are so closely tied to living and play areas. Farm injury risks are similar among men and women when number of hours of exposure to farm tasks is controlled, and children and elderly farm residents are at especially high risk for farm-related injuries.[118-122] Occupational hazards on farms, such as animals and machinery, pose risks to children whether they are working or playing. In 2003, the National Children's Center for Agricultural Health and Safety led an effort to establish consensus development initiatives and generate work guidelines.

FIRE INJURIES

Fire death rates per capita are 36% higher in rural than in urban areas. Residential fires may be more common in rural homes because of older home construction and use of more risky heating sources. Heating is the leading cause (36%) of rural home fires, followed by cooking (13%). In urban areas, the situation is reversed, with cooking the leading cause of home fires (25%) and heating the second leading cause (16%).[123]

Residents of rural areas may be less likely to escape from a fire once it has started because of poor home fire protection. Smoke alarms reduce the risk of dying in a fire by half and reduce the risk of having a reportable fire by 75%.[124,125] Unfortunately, 73% of rural home fires occur in homes without operational smoke detectors. In the United States, the percentage of urban homes with smoke detectors is 92.9%, whereas the corresponding percentage of rural homes is 85.8%. Furthermore, the percentage of homes that experience a fire and have a working smoke alarm is 41.8% in urban areas, but only 20.8% in rural areas. The isolation of rural homes may also cause delayed detection and longer response times on the part of fire and emergency service personnel.[126]

TREATMENT INTERVENTIONS IN RURAL TRAUMA CENTERS

Definitive Surgery

Under most circumstances, a stabilizing procedure can be conducted and completed in conventional fashion such that no further treatment will be required for that particular problem.

Hemorrhage Damage Control

Key components during this phase of care must focus on hemorrhage control and preservation of blood volume. This involves a balance between effective low-volume hemostatic resuscitation with use of blood or plasma in combination with maneuvers that will preserve blood volume using hemorrhage control for compressible and noncompressible hemorrhage. For volume preservation in compressible hemorrhage, tourniquets, hemostatic dressings, pelvic binders, and junctional tourniquets have a great impact on survival when applied early before transfer of the patient. For patients with noncompressible hemorrhage, data are currently lacking for use of retrograde endovascular balloon occlusion of the aorta catheters, experimental ResQ foam (Arsenal Medical, Watertown, MA), preperitoneal packing, and Abdominal Aortic and Junctional Tourniquets. Because of these limitations, a potential solution in rural noncompressible hemorrhage control is the adaptation of an echelon system of care. This is where rural damage control interventions, as described earlier, can be incorporated as a bridge in care.

Damage control (see Chapter 42) is a strategy for the staged management of severe trauma using a modified operative sequence of rapid lifesaving maneuvers followed by physiologic stabilization and planned reoperation for definitive repair. Introduced in the early 1980s, damage control has gained wide acceptance in the past two decades as the "great equalizer" of trauma surgery, allowing surgeons with limited experience or resources to tackle severe truncal trauma in both military and rural settings. The wide applicability of the damage control approach has led to the development of several operative trauma courses, such as the Definitive Surgical Trauma Care Course (International Association for Trauma Surgery and Intensive Care) and Advanced Trauma Operative Management (American College of Surgeons). Even though a damage control operation involves the entire operating room team, the previously mentioned courses focus on operative techniques for the individual surgeon that can make a difference in rural trauma care.[127]

Damage control operations followed by temporary closure of the abdomen have been described for a variety of truncal injuries with an emphasis on limiting time in the operating room and limiting the triad of acidosis, hypothermia, and coagulopathy.[128-130] Candidates for these desperate measures typically have an overwhelming constellation of injuries. Rural surgeons caring for such patients can apply the same principles. Occasional patients will have such complex injuries that definitive management exceeds the technical abilities or resources of the local surgeon and hospital, but may be amenable to temporizing maneuvers, followed by rapid transfer in an aircraft equipped as an airborne intensive care unit.[131] Examples include packing of the liver for complex hepatic injuries; peritoneal cleansing, hemorrhage control, and stapling or rapid suture of multiple bowel perforations; temporary abdominal closure; and abbreviated thoracotomy, hemorrhage control, and temporary chest closure for patients with extensive pulmonary and thoracic vascular injuries.[132] If interventional radiology is not available locally, patients with unstable pelvic fractures may benefit from application of external fixators prior to transfer.

Neurosurgical Damage Control

Injury to the brain remains the single greatest source of morbidity and mortality for trauma victims. Many patients sustain their closed head injuries far from the nearest neurosurgeon, and time becomes the enemy. Under most circumstances, the only option is to minimize secondary brain injury with appropriate ventilatory and pharmacologic maneuvers and expedite transfer.[133,134] A small proportion of these patients, however, will have lesions amenable to surgical drainage. While rural hospitals generally lack neurosurgical services, most hospitals in the United States now have CT scanners. General surgeons can be trained to perform burr holes and/or limited craniotomy for decompression and hemorrhage control in patients with epidural or subdural hematomas noted on a CT scan.[135,136] When a patient with an injury to the brain shows signs of rapid deterioration and a delay of more than 90 minutes to definitive neurosurgical care is anticipated, consideration should be given to emergency decompression of the hematoma.[137] Refer to Tables 11-1 and 11-2 for appropriate operative skills for general surgeons and indications and performance of limited craniotomy by rural surgeon.

Damage Control Resuscitation

Hemorrhage is responsible for one-third of all traumatic deaths. Recent research has identified the coagulopathy of trauma as a significant component and risk factor for

TABLE 11-1: Appropriate Operative Skills for General Surgeons[a]

Establish surgical airway
Closure of evisceration
Splenectomy or splenorrhaphy
Hepatic resection/debridement; insertion of perihepatic packs
Closure of injuries to gastrointestinal tract
Shunting of truncal and extremity vascular injuries or repair
Control of hemorrhage from open extermity fractures with or without mangling
Reduction of dislocation of joint

[a]Options may be limited depending on anesthesia, blood bank, availability of surgical assistant, and availability of intensive care unit.

hemorrhagic death. Current treatment priorities include minimization of the use of crystalloid-based resuscitation in favor of early blood component–based resuscitation that includes plasma and platelets in equal ratios with packed red cells. These in-hospital practices, termed *damage control resuscitation*, are widely used for both battlefield and civilian resuscitation after traumatic injury. Initiation of the tenets of damage control resuscitation in the rural environment has the potential to reduce downstream complications attributable to hemorrhage by intervening close to the time of injury, before the development of coagulopathy, irreversible shock, and the ensuing inflammatory response.[138-141]

Recent studies have evaluated the use of tranexamic acid (TXA) in the trauma patient to treat hyperfibrinolysis. The CRASH-2 study was an international, randomized, placebo-controlled study investigating the effects of TXA on civilian trauma patients. All-cause mortality was reduced by 1.5% within the TXA group, as was the risk of bleeding-specific deaths. Follow-up studies found that this mortality effect was only present when TXA was administered within 3 hours of injury. Patients receiving TXA beyond 3 hours from injury had an increase in mortality. Additional support for the use of TXA was provided by the Military Application of Tranexamic Acid in Trauma Emergency Resuscitation (MATTERs) study. From this study, TXA was contraindicated if the time since injury was greater than 3 hours or in the case of an isolated traumatic brain injury.[142-147]

When analyzing its use in rural trauma, Bardes et al[148] demonstrated no benefit of TXA use mostly due to prolonged transport times. Of the 400 patients who were evaluated, 54% met indications for TXA, and 14% of these received TXA. In those with an indication for TXA, 30.4% were ineligible due to arrival beyond 3 hours from time of injury. There was no difference in TXA indications between the 265 patients arriving from the scene and the 135 patients who were transfers (73 vs 144, $P = 1$). As expected, transfers were more likely to arrive beyond the 3-hour window (59 vs 7, $P = .001$). Mortality for patients treated with TXA was 12.5%, and this was not significantly different from the 19% mortality rate in patients not treated with TXA.[148]

TABLE 11-2: Indications and Performance of Limited Craniotomy by Rural Surgeon

A general surgeon trained in indications for and technique of burr holes with limited craniotomy
Traumatic brain injury with lateralization signs and *threatened herniation*
Surgical lesion present on CT scan
Consultation with, and patient acceptance by, a neurosurgeon at the receiving trauma center
Limited craniotomy for decompression and hemorrhage control
Immediate transfer to definitive care

CT, computed tomography.

LIMITATIONS OF RURAL RESOURCES AND BENEFIT OF EVOLVING TECHNOLOGIES

Prehospital Limitations

An effective EMS program is vital for proper trauma care. In rural areas, the configuration of such systems varies and may include fire department–based, hospital-based, or freestanding entities. Personnel may be volunteers, salaried, or partially subsidized. Most are trained to the EMT-Basic level, which permits noninvasive interventions to reduce the morbidity and mortality associated with acute, out-of-hospital medical and trauma emergencies. Fewer emergency medical personnel have advanced training (prehospital trauma life support, paramedic), which is often difficult to maintain because individual EMS personnel see relatively few trauma patients per year. Additionally, recruiting and retaining emergency medical personnel in rural areas are difficult because of low wages, lack of training opportunities, and the need to cover a broad geographic region.[149–154]

The difficult terrain in rural areas combined with a scarcity of emergency medical personnel contributes to higher mortality rates and longer transport times to a care facility.[149–152] In addition, response times, scene times, and transport times have been shown to be significantly longer in rural areas.[150,151,153,155] Numerous studies have confirmed that rural death rates after motor vehicle collisions are twice as high as those occurring in urban environments.[150,156,157]

Communications

A number of improvements in communication between prehospital providers and health care facilities have been made in recent years. Upgrades to UHF and VHF radios and the addition of the 800-MHz bandwidth have allowed communications between multiple first-responder agencies. Despite improvements in radio communications, EMS providers often find themselves out of contact with the medical director, emergency department personnel, or the dispatcher because of challenging terrain rendering the radios unusable.

Cellular technology has improved prehospital provider communication in many areas; however, this, too, is limited in mountainous terrain.[149]

CONCLUSION

Many of the problems discussed in this chapter can be diminished with proper training, triage, and the development of trauma systems.[158] Esposito et al[159] evaluated preventable death rates and the nature and degree of inappropriate care before and after the implementation of a voluntary trauma system in a rural state. Preventable death rates decreased from 13% to 8% after implementation of a trauma system.

Having a regionalized trauma system potentiates appropriate transfers from the nontertiary hospital to the trauma centers. A study of 42 rural nontertiary hospitals in Oregon demonstrated that early transfer of patients to a tertiary center was associated with a lower mortality. The authors concluded that recognition and early transfer of at-risk rural trauma patients improve survival in a regionalized trauma system.[160] Developing a regionalized system with coordinated care and predetermined referral patterns facilitates interfacility transfers, contributes to improved outcomes, and improves efficiency. Developing these relationships promotes bidirectional communication that can be augmented with newer technology such as shared health information systems, back-transfer agreements, and telemedicine.[161]

The relationships developed in a regionalized trauma system also become a foundation for educating the providers in a rural hospital or prehospital environment. Some states have mandated that all prehospital providers will meet the standards of paramedics and be trained in Prehospital Trauma Life Support (National Association of Emergency Medical Technicians/American College of Surgeons).[149] In-hospital providers who are often family physicians, internists, or advanced practice providers can also benefit from these educational relationships.

New technology has the potential to increase education and improve communication between the rural hospitals and their tertiary referral centers. Telemedicine has the potential to aid in the decision to transfer critically ill patients and allow the rural provider to obtain advice on patient management.

In one study of six rural hospitals in upstate New York and Vermont, 80% of referring emergency physicians found direct telemedicine communications with a trauma surgeon at a distant Level I trauma center to be helpful. It was thought to facilitate care and transfer and, in almost 10% of cases, felt to be lifesaving.[162] The implementation of a telemedicine system with multiple rural hospitals feeding into a Level I trauma center decreased transfer time to definitive care from 13 hours to 1.7 hours.[163]

Telemedicine can be beneficial for prehospital providers, as well. In a simulated scenario, telemedicine to a moving ambulance was associated with better process measurements. Recognition rates for key signs, processes, and interventions were significantly higher in ambulances guided by physicians via telemedicine versus those that previously were not.[164]

Telemedicine has the ability to enhance the care of trauma patients in austere environments by bringing advanced skill and experience to injured patients who will have delays in transport to definitive care.[165] After the institution of a telemedicine program with a Level I trauma center, both providers at the rural hospitals and at the Level I trauma center receiving patients thought the quality of care was improved. Of the 41 consults over 30 months, three were felt to be lifesaving.[166]

Because of the extensive distances patients often must travel, follow-up can be difficult for trauma patients. In one study of 11 rural counties surrounding a Level I trauma center in Kentucky, patients were examined via telemedicine using an electronic stethoscope and a close-up imaging instrument. They were interviewed with the assistance of a nurse and x-rays performed at the site and viewed via a secure high-resolution internet connection. Patient and physician surveys indicated a high degree of satisfaction with the telemedicine clinic visit. In 15 of 22 patients, no further clinical follow-up was arranged. The differences in travel distances and times for an appointment at the tele-trauma clinic versus an appointment at a Level I trauma center were significant. The average and median durations of the appointments were both 14 minutes.[167]

As cellular technology has become more ubiquitous, expensive telemedicine systems may no longer be needed to communicate between facilities. In a study from Arizona, trauma surgeons were able to increase the number of tele-trauma consultations by providing secure smart phones to rural emergency departments. They concluded that smart phones may be an answer to meeting both the educational and clinical needs of rural facilities. Smart phones may replace the older stationary telemedicine technology.[168]

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II

GENERALIZED APPROACHES TO THE TRAUMATIZED PATIENT

12

Disaster and Multiple/ Mass Casualties

Susan M. Briggs

KEY POINTS

- The objective of disaster medical care is the "greatest good for the greatest number of victims."
- The Incident Command System/Incident Management System is the accepted disaster management system for all disasters, regardless of size or etiology.
- Disaster management teams are based on functional capabilities, not titles.
- The goal of disaster response is to reduce the number of survivors from the disaster who subsequently die (critical mortality rate).

INTRODUCTION

The prehospital and hospital management of the medical effects of contemporary disasters, whether natural or human-made, is one of the most significant challenges facing trauma providers today. No one can predict the time, location, or complexity of the next disaster. The demands of disaster care have changed over the past decade in the types of threats, the scope of care, and the field of operations (Fig. 12-1). Many of today's disasters occur or result in austere environments. Access, transport, resources, and other aspects of the physical, social, political, or economic environments may impose severe constraints on the adequacy of immediate care to victims of the disaster.[1-3]

Trauma providers must be prepared to respond to both mass casualty incidents and multiple casualty events.[1,4] **Mass casualty incidents (MCIs)** are events causing numbers of casualties large enough to disrupt the health care services in the affected community or region. *Demand* for resources always *exceeds the supply* of available resources in an MCI. This is in contrast to **multiple casualty events (MCEs)** in which medical resources are strained (prehospital and/or hospital resources) but not overwhelmed. Disasters involving weapons of mass destruction (WMD; biological, chemical, and radioactive), accidental or intentional (terrorism), are increasing in frequency and present unique challenges in the delivery of care to victims by trauma providers.[1,5,6] Active shooter incidents resulting in multiple casualties have been on the rise throughout the world, significantly increasing the challenges for trauma providers and trauma centers.[7-10]

Disaster trauma care is *not* the same as conventional trauma care. Disaster care requires a fundamental change (**crisis management care**) in the care of disaster victims to achieve the objective of providing the "**greatest good for the greatest number of victims**."[1-3]

EPIDEMIOLOGY OF DISASTERS

Natural disasters may be classified as sudden-impact (acute) disasters or chronic-onset (slow) disasters.[1,11] Sudden-impact natural disasters generally cause significant mortality and morbidity immediately as a direct result of the primary event (eg, earthquakes). Traumatic injuries such as crush injuries and burns are common injuries in sudden-impact disasters. Chronic-onset disasters (eg, droughts) cause mortality and morbidity through prolonged secondary effects such as infectious disease outbreaks, dehydration, and malnutrition.

Disasters involving WMD, whether accidental or intentional (terrorism), are a significant challenge for medical providers for several reasons[1,5,6]:

- WMD have the potential to produce casualties in numbers large enough to overwhelm health care systems, including surgical capabilities.
- WMD may produce large numbers of **expectant** victims (victims not expected to survive).
- WMD may produce a **contaminated** environment. Trauma providers must be able to perform triage, initial stabilization, and possible definitive surgical care outside traditional health care facilities.

FIGURE 12-1 Haiti earthquake, 2011.

- WMD produce significant numbers of **psychogenic** casualties, greatly complicating rescue and triage efforts.

The spectrum of agents used by terrorists is limitless and includes conventional weapons, explosives, and WMD.[12-16] Over 70% of terrorist attacks involve the use of explosive weapons. Improvised explosive devices (IEDs) are a particular concern for trauma providers. Such incidents present a significant challenge due to the complexity of injuries (primary, secondary, tertiary, and quaternary blast injuries) (Fig. 12-2). Responders must also be aware of the potential for secondary strikes directed at harming medical personnel. Terrorists do not have to kill people to achieve their goals. They just have to create a climate of fear and panic to overwhelm the health care system (examples: Sarin/anthrax attacks).

DISASTER PREPAREDNESS

Disaster preparedness is the key to an effective disaster response. Preparedness is the responsibility of every health professional regardless of specialty and all health care facilities. A key concept in disaster management is the **all hazards** approach to disaster preparedness. The all hazards approach is based on a single plan for all disasters regardless of etiology. The plan then has branch points that address specific risks.[1,4,17]

FIGURE 12-2 Improvised explosive device.

Disaster planning, whether at the local, regional, national, or international level, involves a wide range of individuals and resources. All disaster plans should involve key medical and public health organizations as well as public safety officials (eg, fire, police). Disaster plans must take into account special needs populations (children, the elderly, long-term care facilities, the disabled [both physically and mentally], the poor, and the homeless). These groups are often neglected in disaster planning.

Disaster preparedness activities consist of making an organization aware of possible disaster situations, training personnel, and purchasing equipment to support their response activities. It is important to determine the balance between what is needed in disasters versus what is available in terms of personnel and equipment in a specific organization or facility before a disaster occurs. Disaster preparedness must include practical drills, not just tabletop exercises, to ascertain the true magnitude of system problems.

Hospital preparedness involves the activities a hospital undertakes to identify specific environmental and physical risks to their facility and the necessary resources and personnel to respond to both internal or external disasters. It is important for hospitals to determine their own thresholds, recognizing that their disaster plans must address both MCIs and MCEs. Hospitals should be able to care for an increase in patient volume higher than normal (estimated to be 10%–20%). It is important to distinguish *surge capacity* (resources such as beds, ventilators) from *surge capability* (qualified personnel to staff the additional resources). All disaster plans must include training in disaster response appropriate to the skills of the individuals being trained and to the specific functions they will perform in a disaster. Cross-training of functional capabilities is important in hospital disaster preparedness. Hospital preparedness should include the following[1,4,17]:

- Security plans, including hospital lockdown if necessary
- Designation of triage and decontamination sites outside the hospital
- Designation of a site for the Incident Command Center separate from the emergency department
- Procedure for initiating hospital Incident Command System
- Procedure for activating the hospital disaster plan, including procedures for notifying off-duty personnel
- Establishment of redundant communication systems in the event of loss of telephone landlines and cellular circuits
- Storage for equipment, supplies, or special resources that may be necessary based on local hazards (sufficient to sustain hospital operations for a minimum of 72 hours)
- Plan to ensure unidirectional flow of patients from the emergency department to the operating room, intensive care unit (ICU), and inpatient units
- Development of predisaster agreements for transporting victims or inpatients to other facilities if necessary

- Training in nonmedical and medical aspects of disaster management for medical providers
- Designation of a site for patients with minor or no medical conditions and equipped with appropriate supplies/equipment
- Designation of a public information center to provide timely briefings to press, hospital personnel, and families

KEY PRINCIPLES OF DISASTER RESPONSE

Lessons from previous disasters are important in distinguishing myths (misconceptions) from facts (evidence-based data). Key principles of disaster response are the same regardless of the location of the disaster (prehospital and hospital).[18-22]

Principle 1

Medical providers cannot use traditional command structures when participating in disaster response. The Incident Command System (ICS)/Incident Management System is a modular/adaptable system for all prehospital and hospital incidents and facilities and is the accepted standard for all disaster response.[1,4,17] The Hospital Incident Command System (HICS) is an adaptation of the ICS for hospital use, allowing effective coordination in disaster preparedness and response activities with prehospital, public safety, and other response organizations. The trauma system and trauma centers are important components of the ICS.

Functional requirements, not titles, determine the ICS hierarchy (Fig. 12-3). The organizational structure of the ICS is built around five major management activities (incident command, operations, planning, logistics, and finance/administration).[1,4,17] Key activities of the five categories are listed in Table 12-1.

The structure of the ICS is the same regardless of the nature of the disaster. The difference is in the particular expertise of key personnel depending on the etiology of the disaster. An important part of disaster planning is the identification of the incident commander and other key positions *before* a disaster occurs (24 hours a day, 7 days a week). Each person within the command structure should supervise *only* three to seven persons. This is quite different from conventional prehospital/hospital command structures. All medical providers must adhere to the structure of the ICS in order to integrate successfully into the disaster response team and avoid many negative consequences including:

- Death of medical personnel due to lack of safety and training
- Lack of adequate medical supplies to provide care
- Staff working beyond their training or certification
- Lack of coordination

FIGURE 12-3 Incident Command System.

TABLE 12-1: Organizational Structure of the Incident Command System

- **Incident Command (IC):** Maintains overall responsibility for the disaster response and sets objectives and priorities for the disaster response.
- **Operations Section:** Directs the activities of all organizations responding to the disaster, including medical personnel.
- **Logistics Section:** Provides resources and logistical support to meet disaster needs.
- **Planning Section:** Develops the incident action plan (plan to carry out the objectives of the disaster response), monitors progress, and makes recommendations for further operational response.
- **Finance/Administration Section:** Monitors costs, manages legal affairs, and maintains personnel records.
- The incident commander is assisted by **liaison officer**, **safety officer**, and **public information officer**.

Principle 2

All disasters are *not* different. Disaster response includes basic concerns (similar to the ABCs [airway, breathing, circulation] of trauma care) that are the same in all disasters (Table 12-2). The difference in disasters is the degree of disruption of medical and public health infrastructures and the amount of outside assistance (regional, national, international) that is needed to meet the needs of disaster victims. Rapid assessment by experienced disaster personnel will determine which functional capabilities (public health and medical) are needed to meet the demands of the *acute* phase of the disasters.

Principle 3

Effective surge capability is not based on well-intentioned and readily available volunteers. Disaster responders must understand the basic principles of disaster response (ICS, disaster triage, gross decontamination) to be effective members of the disaster teams. All providers involved in disaster response must ensure their personal safety by obtaining vaccinations

TABLE 12-2: Rapid Needs Assessment in Disasters

Public health concerns
- Water
- Food
- Shelter
- Sanitation
- Security/safety
- Transportation
- Communication
- Endemic/epidemic diseases

Medical concerns
- Search and rescue
- Triage
- Definitive care
- Evacuation

appropriate to their roles in disaster response (local, national, international) before a disaster occurs.

Principle 4

Trauma care in disasters is not the same as conventional trauma care.[1,3,4] Disaster care of traumatic injuries requires a fundamental change in the approach to the care of victims (crisis management care). The objective of conventional trauma care is the **greatest good for the individual patient**. *Severity of injury/disease* is the major determinant of medical care. The objective of disaster trauma care is the **greatest good for the greatest number of victims**. Determinants of care are *severity of injuries, likelihood of survival, and available resources* (personnel, logistics, evacuation assets).

MEDICAL RESPONSE TO DISASTERS

Trauma providers are uniquely qualified to participate in all four aspects of disaster medical response (search and rescue, triage, definitive care, and evacuation) given their expertise in rapid decision making, resuscitation, damage control surgery, and critical care.

Search and Rescue

Many countries, including the United States, have developed specialized search and rescue teams as an integral part of their national disaster plans (Fig. 12-4).[1,3,4] Members of these teams, who receive specialized training in confined-space environments, include the following:

- A cadre of trauma specialists
- Technical specialists knowledgeable in hazardous materials, structural engineering, heavy equipment operation, and technical search and rescue methodology
- Trained canines and their handlers

FIGURE 12-4 Search and rescue teams.

Trauma centers must be prepared to deploy additional personnel as needed to the scene of a disaster with appropriate equipment (eg, amputation kits, tourniquets, hemostatic agents).

Triage

Triage is a *dynamic* decision-making process of matching patients' needs with available resources. Triage is the most important and psychologically challenging aspect of disaster medical response, both in the prehospital and hospital phases of disaster response. Disaster triage is significantly different from conventional trauma triage.[1,3,4] The major objective and challenge of disaster triage is to identify the *small minority* of critically injured patients who require urgent lifesaving treatments, including damage control surgery, from the *larger majority* of noncritical casualties. **Critical patients who have the greatest chance of survival with the least expenditure of time and resources are prioritized to be treated first.** Review of the literature from major disasters estimates that 15% to 25% of victims are critically injured, and the remainder of victims are noncritical casualties.

TRIAGE ERRORS

Triage errors, in the form of undertriage and overtriage, are always present in the chaos of mass casualty events. *Undertriage* is the assignment of critically injured casualties requiring immediate care to a "delayed" category. Undertriage leads to treatment delays with increased mortality and morbidity. *Overtriage* is the assignment of noncritical survivors with no life-threatening injuries to immediate urgent care. The higher the incidence of overtriage, the more the medical system is overwhelmed with increased mortality and morbidity. The level of acceptable over- and undertriage in an MCI and the best method for evaluation of triage effectiveness in an MCI are controversial.[1,4,22]

LEVELS OF TRIAGE

Three levels of disaster medical triage have been defined. **The level of disaster triage used at any phase of the disaster will**

FIGURE 12-5 Crush injury to face.

depend on the ratio of casualties to capabilities.[1,3,4] Many MCIs/MCEs will have multiple levels of triage as trauma patients move from the disaster scene to definitive medical care.

Field triage (level 1) is the rapid categorization of victims potentially needing immediate medical care "where they are lying" or at a casualty collection center. Victims are designated as acute or nonacute. Color coding may be used.

Medical triage (level 2) is the rapid categorization of victims by experienced medical providers at a casualty collection site or fixed/mobile medical facility.[15,17] Victims are classified into the following categories:

- **Red (urgent)**—Lifesaving interventions (airway, breathing, circulation) are required.
- **Yellow (delayed)**—Immediate lifesaving interventions not required (Fig. 12-5).
- **Green (minor)**—Minimal or no medical care or psychogenic casualties.
- **Black**—Deceased victims.

Initial triage should be conducted outside of hospitals or casualty collection sites. Green category of victims (minor or no medical care) should be triaged to a separate designated site. At the hospital, this site should be separate from the emergency department. Many institutions perform additional triage by surgeons in the emergency department to prioritize individuals needing operative interventions.

EXPECTANT (PALLIATIVE) CATEGORY OF TRIAGE VICTIMS

The expectant category of victims is unique to MCIs.[1,3,4] Victims are classified as expectant if they are not expected to survive due to the severity of injuries (massive crush injuries or burns) or underlying diseases and/or limited resources. The expectant category of triage was first developed given the threat of WMD (biological, chemical, radioactive) during military conflicts but is now used in all disasters. Traditionally, this category of disaster casualties has been classified as yellow or delayed category. Currently, many triage systems classify expectant victims as a separate category with a different color designation. Classification of the expectant category of disaster victims remains controversial. Many models have been proposed based on severity of injury, age, underlying diseases, and hemodynamic stability of victims at time of rescue. Criteria that are currently used as guidelines for the expectant category are as follows:

- Cardiac arrest on scene
- Severity of comorbid diseases
- Requirement for intubation and ventilation on scene in victims with multisystem traumatic injuries and severe hemodynamic instability
- Severe head injuries
- Massive burns (>80% total body surface area)

Evacuation triage (level 3) is often a neglected area of disaster preparedness. Priorities for transfer to medical facilities are assigned to disaster victims using the same color classification as medical triage.

Definitive Medical Care

Definitive medical care refers to care that will improve, rather than simply stabilize, a casualty's condition.[1,3,4,23,24] Maximally acceptable care for all trauma patients is not possible in the early stages of the disaster given the large number of victims in an MCI. **In the initial stages of the disaster, only minimally acceptable trauma care to provide lifesaving interventions is possible.**

Damage control surgery is an important component of crisis management care. Damage control surgery limits trauma interventions to control of hemorrhage and contamination. Tourniquets and hemostatic agents have been important adjuncts in hemorrhage control. Damage control surgery was initially developed for abdominal trauma with uncontrolled hemorrhage but has expanded to all other trauma specialties in disasters. Spinal and regional anesthesia and intravenous sedation and intraosseous infusions are important adjuvants to trauma care in disasters.

Evacuation

Evacuation may be useful in a disaster to decompress the disaster area and provide specialized trauma care for specific casualties, such as those with burns and crush injuries. Trauma providers with expertise in critical care are increasingly valuable resources in disasters. Special considerations during evacuation include the following[1]:

- A decrease in cabin pressure occurs as altitude increases. Trapped gas in any body cavity can cause serious complications as it expands on ascent. Special attention must be paid to trapped gas within the thorax, cranium, eye, and gut in the presence of an ileus. Patient care appliances, such as endotracheal tube cuffs, are also susceptible.
- The partial pressure of oxygen in the ambient air decreases with increasing altitude. Monitoring with pulse oximetry is important.

- Takeoffs and landings present unique challenges, especially with head injury patients.
- Young children, burn patients, and postsurgical casualties are particularly susceptible to temperature changes during evacuation.

WEAPONS OF MASS DESTRUCTION

Biological Agents

Biological terrorism is the intentional use of microorganisms or toxins to kill or injure humans.[1,4] Exposure to biological agents may be accidental or intentional (terrorism). Agents believed to have the greatest potential as bioterrorism weapons are listed in Table 12-3.

ROUTES OF EXPOSURE

The route of exposure of most concern with biological agents is inhalation of the agent. Oral exposure to biological agents may occur directly or secondarily after an aerosol attack. Agents with the highest potential for person-to-person transmission (pneumonic plague, smallpox, and viral hemorrhagic fevers) constitute the greatest hazard. **The most effective and important protection against biological agents is physical protection.** Removal of clothing will eliminate greater than 85% of the agents. Any dermal exposure should be treated immediately by gross decontamination with soap and water.

PROPHYLAXIS AND THERAPY

Medical defenses against some biological agents are limited. Vaccines are available to protect against some biological agents (anthrax, smallpox), and antibiotics may be effective against bacterial agents such as anthrax, plague, and tularemia if given early enough. Disasters involving biological agents have a significant impact on the health care system for the following reasons:

- Terror in affected populations and medical care systems
- Overwhelming casualties and significant ICU/special medication needs
- Problems with handling dead victims

Chemical Agents

Chemical agent release may be unintentional (industrial accidents) or intentional (terrorism).[1] Many chemical agents, especially warfare agents, are liquids and must be dispersed to be maximally effective. There are three general methods of dispersion:

- Aerosolizing with an aerial sprayer
- Aerosolizing the liquid with an explosion (IED and chlorine tanker)
- Allowing the liquid to evaporate (Tokyo Sarin attacks)

Time is of the essence in the decontamination and treatment of chemical agent casualties. Treatment areas should be upwind and uphill from the contamination site. It is important that decontamination facilities be *separate* from the emergency department.

TABLE 12-3: Bioterrorism Agents

- *Bacillus anthracis* (anthrax) – Bacteria
- *Yersinia pestis* (plague) – Bacteria
- *Francisella tularensis* (tularemia) – Bacteria
- Botulism toxins (botulism) – Toxins
- *Variola major* (smallpox) – Virus
- Hemorrhagic fever viruses such as Ebola and Marburg

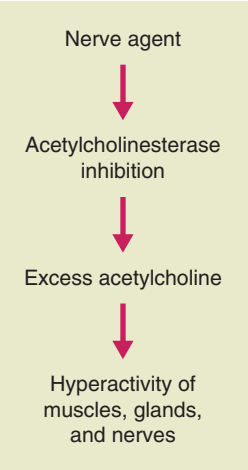

FIGURE 12-6 Mechanism of action of nerve agents.

SPECIFIC CHEMICAL AGENTS

Nerve Agents. Nerve agents are toxic relatives of organophosphate insecticides.[1,3,4] They cause effects by disrupting the normal mechanism by which nerves communicate with muscles, glands, and other nerves (Fig. 12-6). Nerve agents enter the body either percutaneously (through the skin) or by inhalation (through the lungs). Clinical effects of nerve agents are summarized in Table 12-4. The most important

TABLE 12-4: Clinical Effects of Nerve Agents

- **Eyes:** Miosis (small, pinpoint pupils)
- **Nose:** Rhinorrhea (nasal discharge)
- **Mouth:** Secretions
- **Skin:** Sweating
- **Airway:** Bronchoconstriction and bronchorrhea
- **Gastrointestinal:** Hypermotility of gastrointestinal tract (cramps, nausea, vomiting, diarrhea)
- **Genitourinary:** Involuntary urination
- **Skeletal muscles:** Fasciculations, twitching, fatigue, and flaccid paralysis
- **Cardiovascular:** Variable heart rate
- **Central nervous system:** Dose dependent (large vapor or liquid exposure: loss of consciousness, convulsions, apnea)

nerve agents are GA (Rabun), GB (Sarin), GD (Osman), GF, and VX. Treatment to counteract these agents includes the following[1,4]:

- **Atropine 2 to 6 mg intravenously (IV):** Antidote for smooth muscles and exocrine glands
- **Pralidoxime (2-PAM) 600 mg intramuscularly (IM):** Antidote for skeletal muscle sites
  - Atropine-sparing effect
  - Timing of 2-PAM administration is critical; binding of nerve agents to cholinesterase can become irreversible with time
- **Valium (diazepam):** Used as an anticonvulsant as needed (5 mg IV or 10 mg IM)

The traditional Mark 1 Kit contains two spring-loaded injectors of atropine (2 mg) and 2-PAM (600 mg). A new product, DuoDote, contains atropine (2.1 mg) and 2-PAM (600 mg) in a single autoinjector. Antidotes may be given by medical personnel in appropriate protective gear prior to decontamination.

Vesicants. Vesicants are agents that cause erythema and vesicles on the skin as well as injury to the eyes, airways, and other organs.[1,4] Key to treatment of these agents is thorough decontamination as soon as possible. Sulfur mustard has no specific antidote. Dimercaprol, also called British anti-Lewisite (BAL; 2.5–4 mg/kg IM), is the specific antidote for Lewisite.

Hydrogen Cyanide. Hydrogen cyanide has a long history as a deadly poison as it causes death within minutes of exposure.[1,4] Antidote is hydrocobalamin 5 g IV (preferred) or Cyanide Antidote Kit.

Pulmonary Agents. Pulmonary agents cause pulmonary edema, which can be exacerbated by exertion. Phosgene and chlorine are the most common agents. The pulmonary edema caused by phosgene and chlorine causes dry-land drowning to the point that the casualty can become hypoxic and apneic.

Riot Control Agents (Tear Gases or Lachrymators). Treatment is symptomatic with copious irrigation of eyes and skin with water or normal saline.

Radioactive Agents

Release of radioactive material would most likely involve the following scenarios[1,3,4]:

- Detonation of a nuclear device
- Meltdown of a nuclear reactor or melting of the nuclear fuel within a reactor with release of radioactive materials into the environment (Fukushima nuclear accident)
- Dispersal of material through use of a conventional explosive (radiologic dispersal device or "dirty bomb")
- Nonexplosive dispersal of radioactive material

Radiation types include nonionizing radiation (no tissue damage) and ionizing radiation (tissue damage). Electromagnetic radiation and particle radiation (radiation dust) are the two types of ionizing radiation seen in disasters. Radiation exposure may be external irradiation (whole body or localized) and/or contamination (radiation debris), including internal and external contamination. **Responders must assume both external and internal contamination when responding to disasters involving radiation agents.**[1,3,4]

MEDICAL EFFECTS OF IONIZING RADIATION

- Focal tissue damage and necrosis
- Acute radiation syndrome (result of whole-body exposure)
- Long-term effects (thyroid cancer, leukemia)

TREATMENT OF RADIATION CASUALTIES

- Removal of clothing in victims with external contamination eliminates more than 90% of the contamination.
- Radiation effects are delayed; trauma triage is done according to conventional trauma protocols.
- Decontamination should occur before, during, or after initial stabilization, depending on severity of injury.
- Emergency surgery, as well as closure of surgical wounds, should be performed early.
- Know the limitations of your radiation detection devices. Protect yourself until victim is free of all radiation contamination.

DECONTAMINATION

Decontamination is the removal of hazardous materials from contaminated persons or equipment without further contaminating the casualty, rescuers, or medical facilities (mobile or fixed hospitals). The basic principles in response to any hazardous material incident are the same regardless of agents involved. Removal of clothing and jewelry may reduce contamination by up to 85%. It is important for medical providers to protect themselves during decontamination with the appropriate level of personal protective equipment (PPE).

It is important to ensure that the hospital, particularly the emergency department, does not become secondarily contaminated. Failure to do so can result in contamination and quarantine of the entire facility. Local police and/or hospital security may be required to lock down a facility to prevent contaminated victims from entering the facility. The choice of decontamination technique (full decontamination vs gross decontamination) depends on the number of casualties, severity of contamination, severity of injuries, and available resources.[1,3,4]

- **Gross decontamination:** Consists of removing the patient's clothing and any jewelry and irrigating the patient's entire body with water hoses or sprays. This is the type of decontamination usually used in MCIs and MCEs.

- **Full decontamination:** Portable tents or fixed decontamination facilities.

The decontamination site is arranged in three zones:

- **Hot zone:** Area of contamination.
- **Warm zone:** Decontamination zone. The warm zone should be upwind and uphill from the hot zone. Intramuscular antidotes and simple lifesaving procedures such as control of hemorrhage can be administered to victims before decontamination by medical personnel wearing appropriate level of protective gear.
- **Cold zone:** Area where decontaminated victims receive emergency care if needed and transfer to other facilities.

SPECIFIC DISASTER INJURIES

Blast Injuries

Blast injuries are multisystem, life-threatening injuries that are caused by many types of disasters. Blast injury victims tend to sustain more traumatic injuries, varied anatomic locations of injury, and greater Injury Severity Scores (ISS) than other trauma victims. The predominant postblast injuries among survivors involve conventional penetrating and blunt trauma injuries.

MECHANISM OF INJURY

Explosives may be classified as high order or high energy or low order or low energy explosives and cause different patterns of injury. High-energy explosives (eg, C-4, Semtex, ammonium nitrite-fuel oil, TNT) **detonate** to produce a defining overpressurization shock wave. Low-energy explosives (eg, pipe bombs, Molotov cocktails) **deflagrate** to produce a subsonic explosion and lack the overpressurization shock wave.[1,4,5] An IED attack is the use of a homemade bomb and/or destructive device designed to cause death or injury. The survivability, mortality, and morbidity from a blast injury correlate significantly with whether victims are in open- or closed-space explosions. Underwater blasts are significantly more devastating because the lethal radius of an underwater explosion is about three times that of a similar explosion in air.

PRIMARY BLAST INJURY

Primary blast injuries result from the direct effect of the blast wave (traveling at supersonic speeds) on the body. Primary blast injuries affect the gas-containing organs: the lung, the ear, and the gastrointestinal tract.[1,4] The most common lung injury is a pulmonary contusion. Other pulmonary injuries range from pneumothoraces/hemothoraces to arteriovenous fistulas (source of air emboli). Abdominal blast injuries are a significant cause of mortality and morbidity. Abdominal blast injuries may be occult and difficult to diagnose and range from hemorrhage to mucosal ischemia, bowel necrosis, and perforations. The middle ear is particularly sensitive to blast injuries, and tympanic membrane (eardrum) rupture may be a useful marker for blast injury. However, isolated rupture of tympanic membranes without other symptoms is *not* a marker for high risk of associated blast injuries.

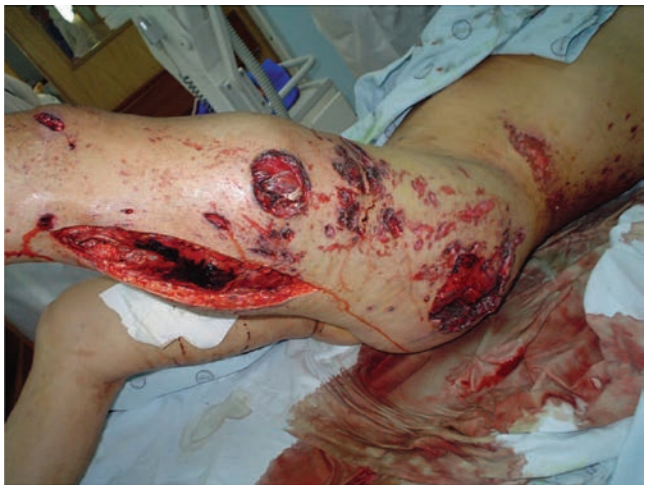

FIGURE 12-7 Secondary blast injury of extremity.

SECONDARY BLAST INJURY

Secondary blast injuries are caused by flying debris generated by the explosion. (Fig. 12-7) A favorite practice for terrorists is packing an IED with screws, bolts, nuts, and other sharp small objects. Significant soft tissue, internal, and orthopedic trauma occurs often from propelled missiles.[1,4]

TERTIARY BLAST INJURY

Tertiary blast injuries are caused by propulsion of the body by the shock wave into solid objects (eg, walls).[1,4] Tertiary blast injury casualties sustain major blunt trauma injuries, such as traumatic brain injuries, solid organ injuries, and complex orthopedic injuries. Penetrating injuries are not uncommon when the victims are impaled on objects in the environment.

QUATERNARY BLAST INJURY

Miscellaneous blast injuries encompass all other injuries caused by the blast, including burns, crush injuries, compartment syndromes, and toxic inhalations (carbon monoxide, dust, hot gases).

Crush Injury

Crush injury is defined as compression of body parts causing localized muscle and nerve damage and is seen in both natural and manmade disasters. The lower extremities are the most common site of crush injuries. Crush syndrome, first recorded in the bombings in London during World War II, is localized crush injury with systemic manifestations. The systemic manifestations are caused by traumatic rhabdomyolysis (muscle breakdown and the release of toxic muscle cell components and electrolytes into the circulatory system).

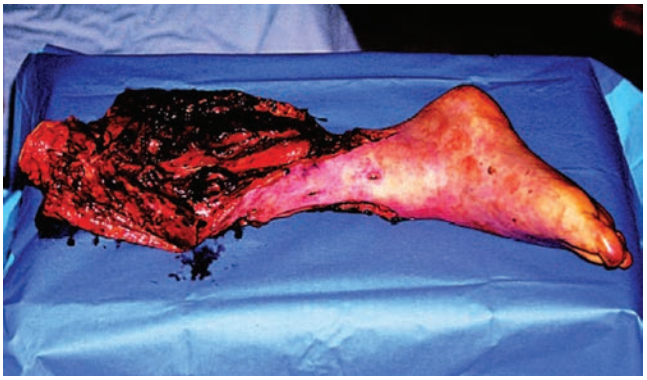

FIGURE 12-8 Field amputation.

Metabolic abnormalities include acidosis (low blood pH levels), hyperkalemia, and hypocalcemia.

Important guidelines for prehospital management of victims with crush injuries have now been established and implemented with search and rescue teams.[1,4,21,25]

- Pretreat casualties with prolonged crush (>4 hours) as well as those with abnormal neurologic or vascular exams with 1 to 2 L of normal saline *before* releasing the crushing object whenever possible.
- If not possible to pretreat, consider applying a tourniquet to crushed limbs and maintain until IV fluid administration is initiated. Risk of acute deterioration and death with sudden release of pressure on the involved extremity (reperfusion syndrome) is a significant risk. Reperfusion syndrome is manifest by acute hypovolemia and metabolic abnormalities.
- Monitor casualties for symptoms of compartment syndrome (fracture not mandatory for compartment syndrome to develop).
- Field amputation of a crushed extremity is considered as a last resort and should be as distal as possible[26-28] (Fig. 12-8).

DISASTER MANAGEMENT TEAMS

Clinical competencies, not titles, determine the roles of medical providers in disaster response. Disaster management teams are designed and trained to provide specific functional capabilities such as trauma, burns, critical care, pediatrics, and obstetrics. When the casualty load is unknown, acute care and trauma surgeons should be the first responders whether on scene or in the hospital. The complexity of today's disasters often demands civilian and military partnerships as key to effective disaster responses.

SUMMARY

A consistent approach to disasters, based on an understanding of their common features and the response expertise they require, is becoming the accepted practice throughout the world. The goal of all disaster response is to reduce the critical mortality associated with a disaster. **Critical mortality is defined as the percentage of critically injured survivors who subsequently die.**[1,4] Numerous factors influence the critical mortality including:

- Implementation of the ICS by prehospital and hospital organizations
- Triage accuracy, particularly the incidence of overtriage of victims
- Rapid movement of patients to definitive care
- Implementation of damage control procedures

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13

Initial Assessment

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KEY POINTS

- The goal of the initial assessment is the rapid identification and treatment of life-threatening injuries.
- Structured handoffs from EMS improve communication to the trauma team.
- The resuscitation bay should contain all instruments and equipment required in a major trauma.
- Trauma team composition should have sufficient personnel to both assess and treat the severely injured.
- The team leader should be the most senior person on the team with an understanding of injury.
- Coordination of care by the team leader is crucial to successful trauma assessments and resuscitation.
- The initial assessment should follow the standard primary and secondary survey algorithms as outlined in Advanced Trauma Life Support (ATLS).
- Patients who arrive in shock require even more rapid assessment and treatment by the most senior personnel.
- Initiating the resuscitation with intraosseous access can be lifesaving in patients in shock.
- Early arterial access for subsequent resuscitative endovascular balloon occlusion of the aorta (REBOA) deployment should be considered for patients presenting with subdiaphragmatic life-threatening hemorrhage.

INTRODUCTION

The importance of the initial trauma assessment cannot be overstated. Missed or delayed identification of injuries during the initial assessment can lead to significant morbidity, complications, and even death. Although there have been multiple studies demonstrating the effectiveness of trauma center care on outcome, in reality, the optimal approach to a trauma patient should be universal regardless of the center. Prompt evaluation and treatment are cornerstones of modern trauma systems, which all starts with the initial assessment.

The initial assessment begins with an understanding of which patients need an evaluation by a trauma team. After successful field triage (see Chapter 4), the injured patient should be seen in a setting that can accommodate the necessary equipment and personnel to provide high-quality trauma care. The basic principles of Advanced Trauma Life Support (ATLS) are used with the primary goal of rapidly identifying and treating life-threatening injury. Adjunctive diagnostic modalities such as a chest radiograph or focused assessment of sonography in trauma (FAST) are often employed to help with the assessment. Finally, ongoing evaluation with feedback and training of the trauma team is needed to maintain readiness.

TRIAGE

Although trauma can be easily defined as "injury," the patient with an injury requiring the resources and expertise of a trauma team is less clear. The ability to triage or "sort" trauma patients to the appropriate level care can be examined from several different aspects. These include (1) prehospital triage (ie, does this patient require a trauma center?), (2) trauma center designation (ie, in a region where there are several levels of trauma centers, which one is most appropriate?), and finally (3) in-hospital triage tiered response (ie, who is going to see and treat the patient upon arrival?). The best outcomes will be generated by a system that is seamlessly connected.

The most recent edition of *Resources for Optimal Care of the Injured Patient* recognizes that inclusive regional trauma systems should place the right patient in the right place at the right time.[1] Ideally, the only difference between hospitals should be related to their resources and not their commitment to provide the best care for the injured patient. There are many field triage variables that have been examined to identify patients who require trauma center resources. These include anatomy, physiology, mechanism of injury, comorbidities, and field triage scores. Of these, physiologic

parameters provide the most accurate single criteria for triage, but ideally a combination of physiologic factors, anatomic factors, mechanism, and comorbidities provides better triage than any criterion taken in isolation.[2,3] Levels of trauma center designation range from I to IV, with Level I centers providing the most comprehensive trauma care and Level IV centers the least (see Chapter 4). Decisions to transport an injured patient to a specific trauma center will be based on multiple factors that are regionally dependent and ultimately depend on the severity of the patient's injuries. Finally, using a tiered response system within the hospital has been shown to be safe and cost effective and can even potentially reduce overtriage.[4-6]

ROOM AND EQUIPMENT

Although, there is no "official" standard for the amount of space needed for the evaluation and treatment of the trauma patient, understanding of the needs and composition of the team will help dictate the space required. The room and equipment should be designed to facilitate trauma team performance. A space large enough to accommodate the trauma team (see later in chapter) and ancillary staff is absolutely necessary. When considering size, one must also consider the need for monitoring and resuscitation equipment, bedside radiographic studies and ultrasound, and crowd control. In ideal situations, the room or rooms should be solely dedicated to the evaluation and initial management of the trauma patient. This dedicated room will allow the staff to become acquainted with the dynamics of the space. Having a readily available resuscitation room allows for rapid assessment and the ability to keep the appropriate equipment stocked in one easily accessible space. The room should also have telephone access for communication with the operating room, blood bank, and other important locations.

The room should have a standard set of equipment that can be used by the trauma team for all trauma patients. Personal protective equipment in the form of fluid-resistant gowns, head covering, face shield, and gloves (both sterile and nonsterile) should be readily available. Depending on how x-rays will be performed, protective lead shielding should be available. A radiographically compatible stretcher to obtain x-rays without having to move the patient is mandatory. Cardiac and vital sign monitoring equipment that includes dynamic electrocardiogram, oxygen saturation, and blood pressure should be permanently available in the room. Quantitative end-tidal carbon dioxide monitoring is a nice adjunct to have as part of the monitoring system. A manual blood pressure cuff should also be immediately available.[7] Flat panel monitors placed within the resuscitation room, which can display radiographic images and laboratory data, are an excellent adjunct that allow the team to remain at the bedside of critically injured patients. Creation of a "universal" instrument set that includes standard equipment that can be used for multiple procedures (eg, controlling hemorrhage from complex lacerations and placing tube thoracostomies) improves trauma resuscitation bay functionality. Other equipment that should be readily available in the trauma bay, presented by body region or specific need, is provided in Table 13-1.

TABLE 13-1: Necessary Ancillary Equipment for a Complete Initial Assessment

Universal tray: hemostats/clamps, retractors (ie, self-retaining, army/navy), pick-ups, needle drivers.

Head and neck: suture material for scalp wounds, intracranial pressure catheters, scopes and light sources for eye and naso-/oropharynx exam, cervical collars.

Airway: preassembled airway kit/box. These can be constructed with input from the airway team but should include endotracheal tubes (various sizes), stylets, capnography, and traditional laryngoscopes. Video laryngoscopes, difficult airway devices (bougies, intubating laryngeal mask airway) tracheostomy tubes, ventilator availability, arterial blood gas kits.

Chest: chest tubes of various sizes (pediatric through adult), chest drainage system, chest tube and thoracotomy instrument tray (see below).

Abdominal and pelvis: ultrasound, diagnostic peritoneal lavage kit, nasogastric tube, urethral catheter (temperature sensing if available), pelvic binder (commercial or homemade device).

Extremity: pneumatic tourniquet, Doppler, cast material.

Shock: intravenous pressure bags, blood and fluid warming systems, large-bore central access catheters, arterial access catheters, intraosseous devices, scalpels, thoracotomy equipment, resuscitative endovascular balloon occlusion of the aorta (REBOA) equipment.

TRAUMA TEAM

Although there are many variations in trauma team composition, there are no hard recommendations as to the optimal number and formulation of the team involved in the initial assessment. Because the primary goal of the initial assessment is to identify significant injuries and initiate lifesaving measures, the team needs to have requisite number of personnel with the appropriate skill set to accomplish this task. The initial assessment can be further divided into the immediate evaluation and management of life-threatening injuries and subsequent identification and management of non–life-threatening issues. These concepts are commonly referred to as the primary and secondary surveys. Again, the composition of the team to evaluate a trauma patient needs to include the personnel necessary to perform these tasks. Based on industrial and psychological research, team efficiency has been divided into horizontal and vertical organizations, which can be translated into horizontal and vertical resuscitations for trauma patients. A horizontal approach is preferred and is one in which each team member is carrying out his or her individual tasks simultaneously. Studies have demonstrated the effectiveness of this approach in improving resuscitation times.[8] Conversely, the less efficient approach would be a vertical resuscitation where each task is performed sequentially.

Frequently seen as the "ideal" environment, the Level I trauma center has a dedicated team for the initial evaluation of the trauma patient. This will often include a senior surgeon, residents and/or advanced practice providers, emergency medicine physicians, dedicated trauma nursing, medical students, anesthesia staff, and respiratory and radiology staff. This team is clearly not attainable in all institutions and should only be used as a template. Trauma center designation should not factor into the proper evaluation of the trauma patient. With the goal to efficiently perform evaluation and management simultaneously, the *minimum* team numbers should include personnel to perform the primary and secondary survey (one or two team members), procedures (one or two members), and a team leader (one member, usually the most experienced surgeon). The nursing staff will be working to help with intravenous access and providing medications, transfusions, and monitoring. Although not well studied, the importance of experienced and dedicated trauma nurses as part of the trauma team cannot be stressed enough.

Trauma team leader: The leader of the trauma team should be the most experienced provider in the room with an understanding of the priorities in the evaluation and care of the injured patient. In a trauma center, this should be the trauma surgeon.[1] Prenotification of a patient arrival allows the team leader to provide provide direction and assign tasks to individual team members and to focus the team *prior* to arrival of the patient. With the goal of conducting the "trauma team orchestra," they should situate themselves with a view of the entire proceedings. Although this is often best accomplished at the foot of the bed, the geography of the resuscitation room will dictate the optimal position that affords the team leader the "big view." Once the patient arrives, the team leader should keep the team on track and offer prompts as needed to bring the initial evaluation and management to an appropriate conclusion. Decisions as to the next steps in care should be made by the team leader. If trainees are present, opportunities should be made for them to be team leader under the close supervision of the attending.

Primary surveyor: This individual is responsible for the actual evaluation of the trauma patient. Based on ATLS algorithms, this individual will work through the ABCD (airway, breathing, circulation, disability)/primary survey of the evaluation and report these findings to the team. Depending on available personnel, this individual can perform the secondary survey as well.

Procedures: Depending on the severity of the trauma, the patient may require multiple simultaneous procedures or none at all. However, it is imperative for the team leader to preassign who is going to perform whatever procedures should be needed. At the very minimum, someone should be designated to provide airway care. Although in some institutions this has been traditionally performed by anesthesia personnel, no difference exists in endotracheal intubation success rates between emergency medicine and anesthesia in Level I trauma centers.[9,10] Local protocols with appropriate quality assurance should dictate control of the trauma patient's airway. All other procedures can be addressed using the most appropriate member of the team.

Ancillary team members: Because the extent and severity of a patient's injuries are often not clear at the beginning of the initial assessment, a mechanism needs to be in place for bringing ancillary staff into the team. These may include extra nursing personnel, respiratory therapists, pharmacists, and x-ray technologists. Additionally, there needs to be a method for activating operating room or interventional radiology personnel if you are in a center that will be providing this level of care. Appropriate and up-to-date contact information for relevant consultants (ie, neurosurgeons, orthopedic surgeons) will also need to be readily available.

EMERGENCY MEDICAL SERVICES HANDOFF

The communication between prehospital providers and the trauma team provides the initial information that helps report observable and potential injuries the patient has sustained. Because communication errors have been shown to be a common cause of preventable disability or death, transmission of information between emergency medical services (EMS) and the trauma team should be afforded dedicated time. The EMS report ideally begins in the prehospital environment. Mature EMS systems will have a mechanism through which the prehospital providers can notify receiving teams about the incoming patient using radio, cell phone, or landlines. Creating a centralized center for dissemination of this information is an ideal way to minimize the need for multiple steps in communication, which could otherwise lead to misinformation. The report need only be brief, containing information as it relates to age, mechanism, hemodynamics, injuries, and estimated time of arrival (ETA). Prenotification allows the trauma team to make the appropriate preparations prior to patient arrival. This can include mobilizing personnel and obtaining equipment necessary for the particular type of injuries that may be presenting.

Once in the hospital, EMS providers should be afforded dedicated time for their report. There have been multiple studies demonstrating difficulties in the handoff between prehospital providers and a receiving team.[11] The reasons for this have been shown to be multifactorial but include the different perspective between EMS and the trauma team, the real or perceived need by the trauma team to rapidly assess the patient, and multiple providers relaying fragmented bits of information.[12]

Additionally, educating EMS providers to impart concise information deemed important to the trauma team has not been a priority. Because of this disconnect, trauma teams often do not demonstrate a willingness to listen, and EMS providers either fail to give an appropriate report or continue to provide information that may not be beneficial to the trauma team. Several studies demonstrate the effectiveness of educating all the involved parties in a more "formal" handover using specific data points. At the current time, several of these exist, one of the most common being MIST (mechanism, injuries suspected, vital signs and symptoms, treatments). MIST

has been adopted by many EMS systems internationally and is also being used by the military for their handovers.[11,13] Other studies have shown that better and more timely data are transmitted when a formal handover is performed.[14] Unless the patient is in extremis, the prehospital handover should take place before or as the patient is transferred to the hospital stretcher. This allows the entire team to focus on the EMS provider without jumping into an assessment that precludes listening. The report should be prompted to start and finish by the team leader, who should be cognizant of the information being given and limit extraneous stories. Ultimately, the prehospital report should not take more than 45 seconds to 1 minute.

PRIMARY ASSESSMENT

Because the overarching goal of trauma care is to minimize mortality and improve outcomes, the primary assessment is designed to systematically allow the team to achieve this objective (Fig. 13-1). In fact, the term *primary assessment* or *survey* is somewhat a misnomer in that the goal is not only to identify (using the ABCDE moniker) potentially life-threatening injuries, but also to intervene and correct these problems as soon as they are diagnosed. With the exception of patients presenting with external or compressible life-threatening hemorrhage or those presenting in extremis for whom the circulation evaluation and treatment will come before airway (ie, CAB [circulation, airway, breathing]), the provider should follow the airway, breathing, circulation, disability, and exposure/environment (ABCDE) approach. One can rapidly obtain a general picture of the patient's condition by merely asking the patient one or two questions. Appropriate answers to "What is your name?" and "Can you tell me what happened?" will provide the team with information regarding the first four elements (ie, ABCD) of the primary survey. If the patient is not awake and alert, tactile stimulation (ie, painful stimuli) in addition to questioning may result in the patient verbalizing answers to questions. A more complete description of assessing disability and altered metal status is outlined later.

Although the importance of the physical examination is stressed continuously throughout training, what data exist following injury demonstrate that it has significant limitations and needs to occur with the appropriate adjuncts to the primary and secondary surveys. However, although the negative predictive value of physical examination may be low, positive findings are almost always secondary to serious injuries and should be acted upon.[15-18]

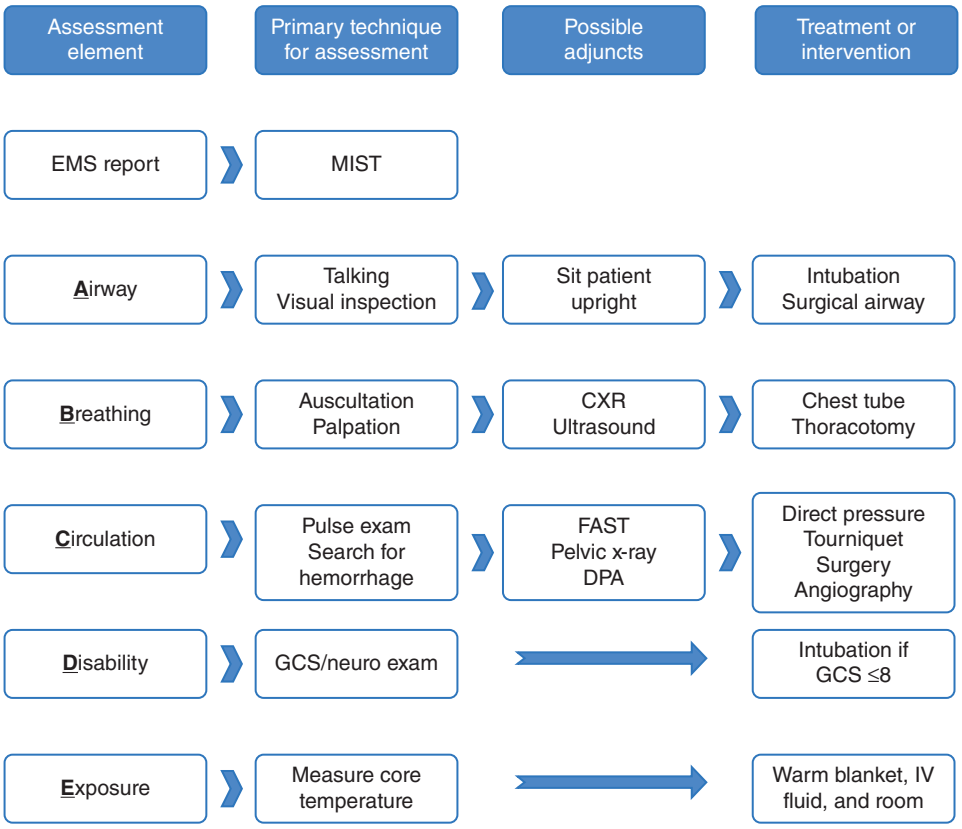


FIGURE 13-1 Overview of the elements of the primary assessment, assessment techniques, and possible treatments. CXR, chest x-ray; DPA, diagnostic peritoneal aspiration; EMS, emergency medical services; FAST, focused assessment with sonography for trauma; GCS, Glasgow Coma Scale; IV, intravenous; MIST, mechanism, injuries suspected, vital signs and symptoms, treatments.

The airway exam begins with assessment of patency. In general, a patient who is able to speak in his or her normal voice does not have airway obstruction. Significant maxillofacial bleeding into the airway or destruction in the area of the oropharynx or neck should prompt urgent airway stabilization. In the setting of penetrating trauma, where cervical spine precautions are not warranted, merely placing patients into an upright sitting position and allowing them to spit or suction the blood can turn an airway emergency into a situation that can be treated in a more controlled fashion. Airway stabilization should also be performed in the patient with a significant alteration in mental status (Glasgow Coma Scale [GCS] score ≤8). On occasion, patients who are combative either due to intoxication and/or head injury may require intubation in order to facilitate or complete their trauma assessment.[19] Once the decision is made to intubate a trauma patient, a brief but thorough neurologic exam should be performed prior to the administration of paralytics and sedation. Securing an airway will generally involve endotracheal intubation via rapid sequence intubation, but the team should always be prepared for a potentially difficult airway with other methods such as video laryngoscopy, flexible fiberoptic intubation, or surgical airway.

The assessment of a patient's breathing will depend on the severity of the injury and presentation. The rapid auscultation of each hemithorax should focus on the absence or presence of breath sounds. Although the presence of breath sounds does not rule out clinically significant pathology, the absence of breath sounds usually indicates a hemo- or pneumothorax that requires urgent treatment. A prolonged multilocation exam is never indicated at this stage in the assessment because the only goal should be to identify the presence of potentially life-threatening injuries. Continuous pulse oximetry can disclose occult hypoxemia but provides no information on the status of ventilation. In the intubated patient, absence of breath sounds, especially on the left, should prompt assessment of the depth of the endotracheal tube and the possible withdrawal of the tube a centimeter or two before other interventions. Other physical findings of chest trauma, such as significant deformity, bony crepitus, subcutaneous emphysema, tachypnea, or desaturations, should prompt further study (ie, chest radiograph) or intervention, most commonly tube thoracostomy. Expanding the FAST (extended FAST [eFAST]) exam from the traditional abdominal views to examine and screen the chest for significant pneumothoraces or pleural blood can be a useful bedside adjunct to traditional chest radiography. In the patient who presents in extremis, triage of the chest cavity needs to be more rapid and invasive. Presumptive placement of bilateral chest tubes will not only diagnose but also treat most life-threatening chest injury. Lastly, in the patient who presents in cardiac arrest, the chest can be definitively assessed via a thoracotomy (see Chapter 17).

Circulation can be rapidly assessed by looking for active hemorrhage and palpation of central and distal pulses. Although not well studied, each location is associated with a minimal threshold systolic pressure: carotid (60–70 mm Hg), femoral (70–80 mm Hg), radial (90–100 mm Hg), and pedal (>100 mm Hg) pulses. Traditionally, this is also the time the patient would be placed on a cardiac monitor. Historically, blood pressure measurement has not been part of this portion of the assessment, and as long as there is evidence of palpable radial or femoral pulses, an immediate blood pressure is not needed. If a blood pressure is to be taken, it should be done manually because the use of noninvasive blood pressure devices may be erroneous in patients in shock.[7] Simultaneously, intravenous access with large-bore catheters should be initiated. In the situation where intravenous access is not immediately obtainable, consideration should be made to perform intraosseous access (further discussed later). Treatment for active external bleeding identified through these exams should begin with direct pressure. Bleeding from wounds that is not able to be controlled by direct pressure should be treated by packing, whip-stitching, or urgent wound exploration. For extremity trauma, placement of a tourniquet can be lifesaving. Blind clamping of deep wounds is discouraged because of the proximity of nerves or the possibility of creating further trauma and hemorrhage. Adjuncts to the circulatory exam include a rapid search for intracavitary bleeding using plain chest and pelvic radiography, FAST, and diagnostic peritoneal aspiration or lavage (DPA or DPL). Identification of intrathoracic or intra-abdominal injury in unstable patients demands urgent intervention most commonly in the operating room. The treatment for pelvic fracture bleeding is beyond the scope this chapter, but a pelvic binder should be immediately placed in high-risk patients (see Chapter 39).

The disability exam focuses on the patient's neurologic status. The GCS score should be assessed immediately upon presentation and sequentially during the resuscitation. As outlined earlier, a persistent GCS less than or equal to 8 should prompt endotracheal intubation. Pupillary response and overall neurologic status (movement and sensation) should be examined and abnormalities noted for early intervention. In patients with an adequate blood pressure, presumptive osmotherapy (ie, mannitol) can be considered for lateralizing signs (see Chapter 22). It should be also remembered that generalized agitation and combativeness are also classic symptoms of class III and IV shock. Patients who present with signs of spinal cord injury should have rapid assessment of their likely injury level. A more detailed neurologic exam, including assigning an American Spinal Cord Injury Association (ASIA) level, should be performed in the secondary assessment (see Chapter 26).

The key to a full examination is appropriate exposure. All clothing should be removed to facilitate a complete 360-degree exam. This is recommended for all trauma cases as it contributes to standardization. Equally important, concern for keeping the patient warm should be paramount once the full exam has been completed. This includes the use of warm blankets, forced hot air blankets, and warm intravenous fluids.

INITIAL EVALUATION OF THE TRAUMA PATIENT IN EXTREMIS

The ideal trauma evaluation, as outlined earlier, relies on speed and efficiency to identify and treat significant injuries. The concept of the "golden hour" recognizes that time to definitive care is important to prevent unnecessary morbidity and mortality. Although this remains a useful concept and aphorism, experienced trauma surgeons realize that there is a wide spectrum of severity of injury. In reality, it is the alteration in a patient's physiology that is the greatest determinant of outcome. Even in patients with a high anatomic injury burden (ie, high Injury Severity Score), the time to evaluation and treatment (unless excessive) will play little role in outcome in those without physiologic derangement. Conversely, there are patients who present in class IV hemorrhagic shock who do not have a "platinum minute" prior to their cardiac arrest from exsanguination. Recent data have confirmed that despite improvements in trauma systems and resuscitation strategies, the mortality rate of patients presenting with a systolic blood pressure of less than 90 mm Hg who require an emergent laparotomy is greater than 40%.[20] Similar high mortality has been demonstrated in hypotensive patients requiring thoracotomy[21] or patients with pelvic fractures.[22] A recent single-center retrospective study demonstrated that patients who sustained torso gunshot wounds who arrived to the operating room in less than 10 minutes had improved survival.[23] Although the basic ABCDEs of the trauma evaluation and resuscitation should be identical from one patient to another, these data demand that patients who arrive in extremis must be considered a specific subgroup, where circulation takes precedence (ie, CABDE).

TEAM CONSIDERATION

Given the excessive mortality of these patients and the relatively thin margin to achieve a successful outcome, it is necessary to ensure the most experienced and capable providers are part of the trauma resuscitation. In addition, pulling in additional personnel may be beneficial. These are the cases where the senior surgeon/team leader will likely need to be an active participant and "lead from within" the resuscitation. Even in academic Level I trauma centers, these providers are most often not "teaching cases" during the resuscitation. Team debriefing and interactive teaching can take place following the completion of the case.

AIRWAY

Although there are no data to suggest an ideal time to secure the airway for the trauma patient in extremis, one must be thoughtful as to when endotracheal intubation (ETI) should occur. There are advantages and disadvantages to performing ETI in the trauma bay as opposed to the operating room. The clear advantages to ETI are the ability to secure an unstable airway, eliminate patient interference, and reduce metabolic demand. However, ETI takes time that the patient may not have, and it is not uncommon for ETI to precipitate physiologic collapse. In that scenario, the trauma bay is not an ideal location to begin necessary operative interventions. The team leader should be cognizant of these issues and make informed decisions as to the timing of ETI based on presumed injury, current hemodynamic status, patient level of consciousness, distance from resuscitation area to operating room, and composition of team.

INTRAOSSEOUS ACCESS

Resuscitation is not possible without access to the circulation. Although ATLS rightly stresses the importance of upper extremity large-bore intravenous accesses, the ability to find and cannulate a peripheral vein in a profoundly hypotensive patient is both challenging and time consuming. Large-bore catheters (6–8.5F) inserted in the femoral or subclavian veins have also been used to resuscitate this group of patients. Lack of circulating volume also makes placing these catheters a challenge with increased time to placement, failure of placement, or complications. For these reasons, alternative access needs to be considered to provide initial resuscitation until more traditional access can be established. Although intraosseous (IO) access was traditionally thought of as a technique restricted to field providers and pediatric patients, recent military experience has clearly demonstrated that this should not be the case,[24] especially because it has been shown that it is possible to provide all types of fluid resuscitation through an IO line. Several studies have demonstrated that the efficacy of the IO catheter and its speed and success of placement make it a possible superior initial option in patients who need rapid infusions without immediate intravenous access. Although there are several brands of IO systems and catheters on the market, we believe that trauma surgeons and emergency physicians need to be facile with the technique.[25-28]

The anatomic location for IO placement is ideally as close to the heart as possible. Although sternal access may be closest, it is often problematic in the patient in whom a possible thoracotomy or cardiopulmonary resuscitation is needed. Therefore, barring any contraindications, the humeral IO may be superior to other sites for the trauma patient. The tibia is often used as an alternative site of access (Fig. 13-2).

RESUSCITATIVE ENDOVASCULAR BALLOON OCCLUSION OF THE AORTA

The traditional approach to the trauma patient in extremis or with recent cardiac arrest has been to consider emergency thoracotomy with aortic cross-clamping (see Chapter 17). Although there remain controversies regarding the risk-benefit ratio of emergency thoracotomy, for patients with suspected injuries above the diaphragm, this approach continues to remain the only option.[29-31] Although present in some form since the 1950s, catheter-based hemorrhage control using resuscitative endovascular balloon occlusion of the

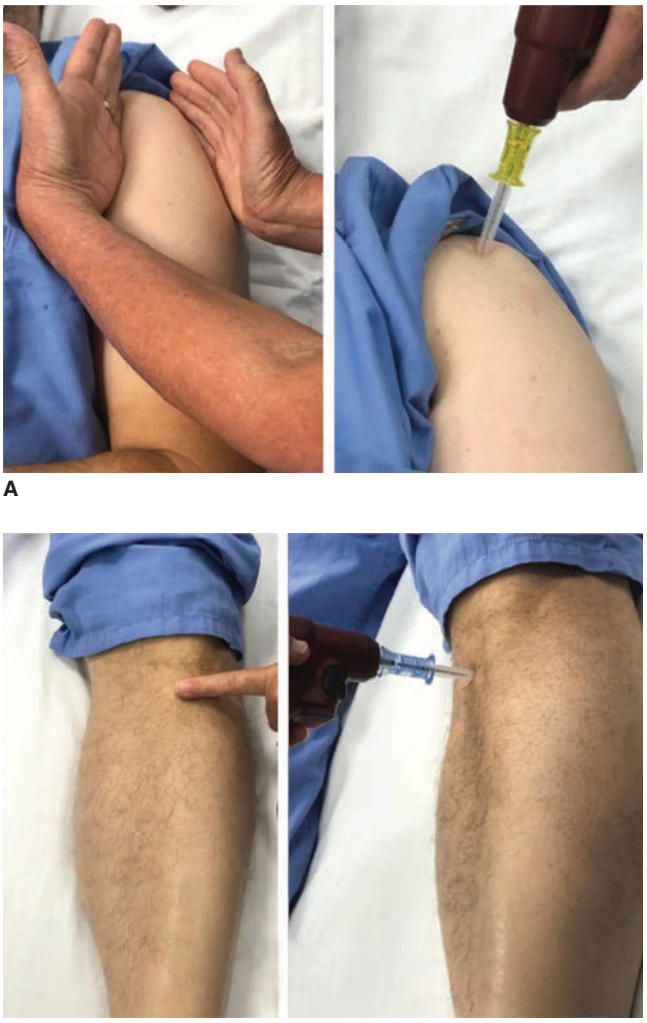


FIGURE 13-2 Landmarks for intraosseous infusion device insertion.

aorta (REBOA) has recently become a viable alternative to emergency department (ED) thoracotomy for the exsanguinating trauma patient with injuries below the diaphragm. The one clear advantage of this approach is that it does not create a wound (thoracotomy) in a previously uninjured body cavity with its attendant blood and heat loss. Preliminary results from the American Association for the Surgery of Trauma Aortic Occlusion Study Group suggested that in patients without penetrating thoracic injury undergoing aortic occlusion, mortality with REBOA was lower than in those undergoing emergency thoracotomy.[32] Conversely, a recent propensity analysis of the American College of Surgeons TQIP database found that REBOA placement was associated with increased mortality and a higher rate of acute kidney injury and amputation compared to a similar cohort of patients with no placement of REBOA.[33]

With the introduction of a small-bore (7F) wireless system, REBOA has become easier to place and its use more widespread. Although there is still a paucity of randomized prospective data and agreed upon indications, REBOA has been definitively added to the trauma surgeon's armamentarium.[34-37]

As with the adoption of all new technologies, the salient issues with its use can be broken down into the following: (1) indications (Which patient populations are candidates for REBOA?); (2) training (What, if any, training is required, and who requires it?)[38]; (3) technical considerations in usage (pitfalls in insertion and deployment); and 4) complications.[39] Until sufficient high-quality data exist, it is incumbent upon each institution to create a guideline for the use of REBOA based on their review of the literature. Once instituted, all cases should undergo robust peer review to ensure patient safety and identify areas for improvement.

Given the high mortality in patients with noncompressible torso hemorrhage, early arterial access for potential REBOA deployment should be considered in trauma patients presenting with hypotension secondary to blood loss from abdominal and pelvic trauma. Patients with concomitant thoracic trauma must be approached with caution because deploying the balloon in a patient with a great vessel injury could worsen and precipitate hemorrhage. What degree of hypotension makes a patient a candidate for REBOA remains unknown. Because the definition and breakpoint for hypotension is a systolic pressure of less than 90 mm Hg, this would seem a logical place to begin. Although many patients with that degree of hypotension will respond to early blood and plasma transfusion, some will not. Thus, it would make sense for the team to consider placing a REBOA in this population. The first hurdle to successful REBOA placement is femoral artery catheterization. The ability to palpate the femoral artery diminishes greatly at systolic blood pressures below 80 mm Hg. We strongly recommend using ultrasound for femoral arterial access because difficulties in achieving arterial access will delay deployment.[40] In patients in cardiac arrest or with profoundly low blood pressure, an arterial cutdown should be used. It would then follow that a more liberal use of femoral artery catheters for patients presenting in shock should be part of the approach to the patient in extremis. Continuous arterial blood pressure monitoring is superior to intermittent noninvasive modalities, and the indwelling arterial line can be used to follow chemical markers of shock, base excess, and lactate. Failure to respond to initial resuscitation efforts or worsening base excess identifies patients who require rapid hemorrhage control and could be seen as indications for REBOA deployment. The use of REBOA in patients presenting in cardiac arrest is also controversial. Without additional data, the indications for deploying a REBOA in this population should be identical to those in patients with nonthoracic injuries who would be candidates for ED thoracotomy.

Patient safety is paramount whenever a new technology or procedure is introduced, especially if that technique crosses specialty lines or is seen by some as just a small adjunct to a preexisting scope of practice. REBOA clearly falls into that category. Femoral arterial access is performed by multiple specialties; surgeons as well as many other practitioners have been deploying arterial lines and other intravascular catheters for decades. In an attempt to get ahead of the debate, a recent

joint statement from the American College of Surgeons Committee on Trauma and the American College of Emergency Physicians outlined their opinion as to best practice in the use of REBOA, including training in established and recognized courses.[38] Not surprisingly, this statement was followed by detractors taking issue with one aspect or another of the statement, including training. One argument for keeping REBOA primarily under the control of surgeons is that acute care or vascular surgeons are required to treat the complications that arise from REBOA. However, vascular surgeons already do so for specialists performing other catheter-based procedures. Other recent series of adoption of REBOA without specialized training have demonstrated good results and low complications.[41] These issues are complicated and clearly beyond the scope of this chapter, but the arguments should remind the astute reader of those that took place with ultrasound. Although one cannot fault additional specialized training taught by leading academic societies, local hospital credentialing dictates what a practitioner may or may not be allowed to do. However, most importantly, and to reiterate, patient safety is paramount, and we believe that no one should be performing any invasive procedure without appropriate competency.

As outlined earlier in the chapter, all necessary equipment for placement and deployment of REBOA needs to be readily accessible in the trauma bay. At the present time, there is only one commercial US Food and Drug Administration–approved small-bore REBOA catheter available. Thus, unless providers are using the older, large-bore catheters, from a technical standpoint, there is only one "system" to learn. Recent improvements have also made this catheter wireless, which eliminates a step in placement (Table 13-2). As stated earlier, the first technical consideration is femoral artery access. Once this is achieved and the decision is made that the REBOA catheter will be used, it should be deployed based on the specific location of injury. The balloon catheter may be inflated at the distal thoracic aorta (zone 1) for control of life-threatening intra-abdominal or retroperitoneal hemorrhage or those with traumatic cardiac arrest. The balloon may be also inflated at the distal abdominal aorta (zone 3) for patients with severe pelvic fracture or junctional or proximal lower extremity hemorrhage not easily amenable to external compression. What is just as important as the indication for REBOA is the time from balloon inflation to hemorrhage control and restoration of distal perfusion. This is the identical consideration that is observed with aortic cross-clamping. With the balloon inflated in zone 1, there is total visceral ischemia. The inability to restore perfusion within 30 minutes of inflation will likely result in a lethal ischemia reperfusion injury. Thus, at the present time, zone 1 inflation is only practical in those institutions and situations where the inflation to operating room time can meet that time limit. In contrast, balloon inflation in zone 3 allows a somewhat longer period of time where ischemia may be tolerated. Nonetheless, as recent military reports of deploying the balloon in a prehospital far-forward setting demonstrate, the ability to transfer to definitive care and control of hemorrhage need to be accomplished in a short time frame.[42] In addition, if REBOA is to be deployed outside of the trauma center, experienced personnel versed in REBOA insertion who can monitor the catheter in transport are mandatory to accompany the patient to definitive care.

TABLE 13-2: Steps for Resuscitative Endovascular Balloon Occlusion of the Aorta Insertion

- Obtain arterial vascular access (preferentially with ultrasound).
- Place compatible 7F sheath.
- Measure catheter using external landmarks.
- Ensure catheter balloon is deflated and pressure tubing is connected.
- Advance peel-away sheath over end of catheter.
- Flush catheter.
- Insert catheter and advance to desired depth.
- X-ray confirmation of placement is recommended.
- Inflate balloon to appropriate size but do not overinflate.
- Monitor hemodynamic response.
- Secure device.

The complications of REBOA are predominantly vascular and perfusion related and can occur anywhere along the path of the catheter. These include arterial laceration; disruption; dissection; pseudoaneurysms; hematomas; thromboemboli; visceral, renal, and extremity ischemia; perforations of the arterial system; intimal injury; and rupture of the aorta from overinflation of the balloon. These injuries may result in the need for operative repairs, organ failure, amputations, and even death. In addition to visceral ischemia, spinal cord ischemia may occur from prolonged zone 1 inflation. As with any new and emerging technology, the initial complication rates reported by those perfecting the technique will be lower than those observed over time as REBOA is used by a broad cross-section of providers. In addition to local performance improvement reviews of the use of REBOA, regional and even national anonymous registries open to all users should be created to fully identify the breadth and prevalence of complications to capture these events.

It is likely that the next decade will see further high-quality data that answer many of the questions posed earlier. It also will likely see refinements in catheter technology to allow more prolonged times between inflation and definitive control of hemorrhage. Experimental studies in porcine models have demonstrated superiority of partial occlusion of the aorta to prevent or reduce distal ischemia with pressure monitoring.[43] This concept has also been discussed in the clinical setting.[44]

SECONDARY ASSESSMENT

Once the primary assessment for life-threatening injury is completed, the team should begin a more thorough and detailed examination of each body region. This "secondary assessment" is actually a complete history and physical

examination whose aim is to identify all other injuries. After obtaining a full set of vital signs that should include pulse, respiratory rate, blood pressure, pulse oximetry, and temperature, the patient's medical history should be obtained. ATLS suggests using AMPLE (*a*llergies, *m*edications, *p*ast illnesses, *l*ast meal, *e*vents/environment/mechanism) to accomplish this. Providers should then perform and document a complete head to toe examination that should follow the standard inspection and palpation model. Specific details on the exam of each body region can be found in ATLS, and many of the issues and controversies in physical examination pertaining to specific body regions can be found in their respective chapters. The need to accurately document the findings on physical examination cannot be overemphasized. All too often later in a patient's hospitalization it is impossible to ascertain a real change in the physical examination from baseline due to poor documentation. This can lead to unnecessary testing or even delays in care.

ADJUNCTS TO INITIAL SURVEY

Although the physical examination is an important component in the initial assessment, its limitations make it necessary to use adjunctive tests to fully identify serious and potentially life-threatening injuries. Adjuncts to the primary survey should be quick and reliable, be able to be performed in the trauma resuscitation bay, and provide a high degree of specificity that positive test results are of unequivocal importance that demands attention. The primary adjuncts remain: chest and pelvic radiography, FAST or eFAST, and DPA (Fig. 13-3). Although computed tomography (CT) scanning revolutionized trauma care and is of undisputed importance in the complete identification of injuries, physiologically unstable patients should not undergo CT, and therefore, we consider it an adjunct to the secondary survey. REBOA placement with improvement in blood pressure, however, may allow for a quick CT scan. Although a complete review of all of these primary adjunctive modalities is beyond the scope of this chapter, the following outlines the major issues.

Chest Radiography

Although not yet relegated to the historical scrap heap, the role of routine chest x-ray (CXR) has been questioned.[45-47] Although chest CT is superior for the complete identification of thoracic injuries, lost in the debate is the true purpose of the plain CXR,[48] which is a screening tool to quickly evaluate a patient for life-threatening conditions. In the patient with an abnormal chest physical examination (eg, tachypnea, low oxygen saturation, significant chest deformity) or hemodynamic instability, the CXR provides a rapid means of identifying hemo- or pneumothoraces, a ruptured diaphragm, pulmonary contusions, or the suggestion of aortic injury. Additionally, pulmonary contusions or multiple rib fractures that can be seen on the screening CXR may predict later respiratory failure and the need for admission to a monitored bed for more comprehensive care.[49] Lastly, the CXR confirms placement of both endotracheal and chest tubes. Thus, although routine CXR may not be warranted in every hemodynamically stable patient with a normal physical examination, it remains an integral and invaluable part of the primary survey in patients with physiologic derangements or positive physical examination findings.

Pelvic X-Ray

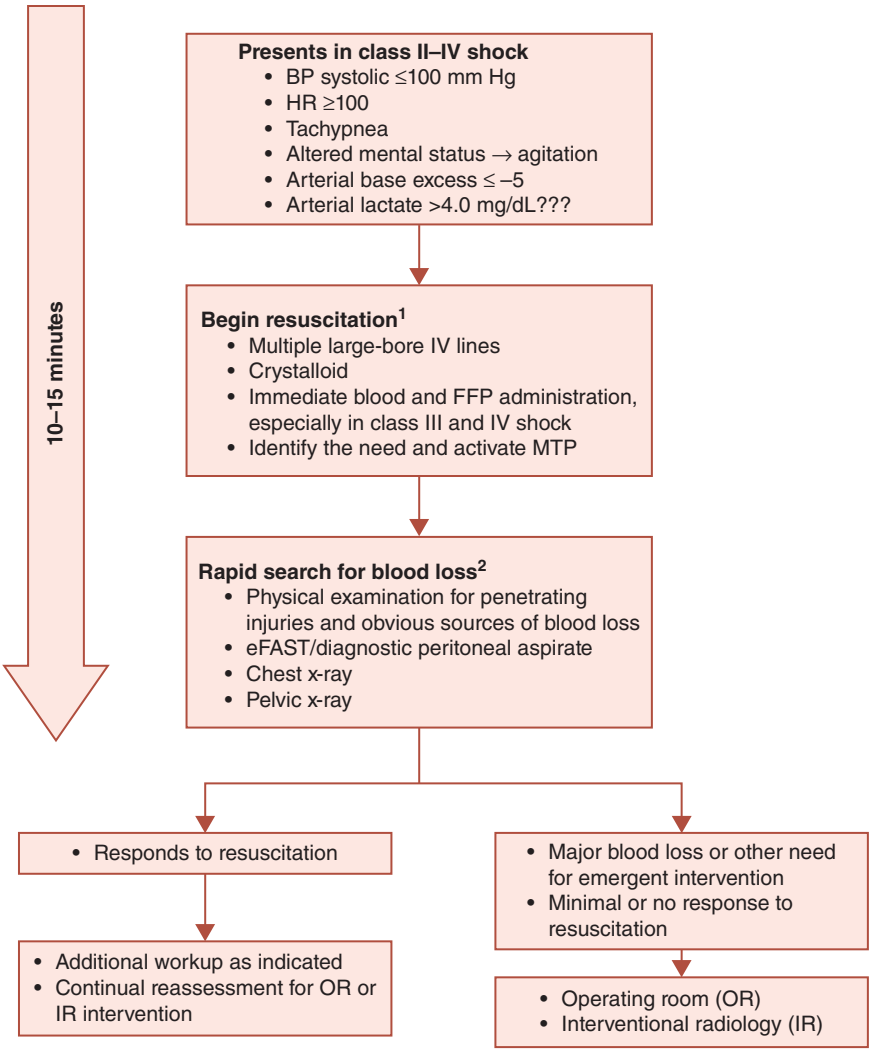
Similar to and possibly to a greater extent than the discussion on screening CXR, there are multiple studies demonstrating lack of utility for routine pelvic x-ray (PXR) as a standard adjunct to the primary survey.[50-52] This conclusion was reflected in current ATLS education, where PXR is no longer considered a mandatory adjunct to the primary survey. In our own practice, we have now focused our efforts in patients with unexplained hypotension in whom a simple intervention such as pelvic binding might be beneficial, or patients with obvious skeletal injuries in whom a hip dislocation may be considered.

Focused Assessment of Sonography in Trauma

The standard FAST exam (four views examining the pericardium, right and left upper quadrants, and the bladder) has been shown to be a useful adjunct in the hemodynamically unstable blunt trauma patient (see Chapter 18).[53-55] Its use has essentially replaced DPA/DPL as the prime adjunct for triage of the abdomen in the unstable patient. Recently, however, there have been questions as to the role this modality plays in certain subgroups such as those with penetrating trauma or the hemodynamically stable patient. Multiple studies have demonstrated that sensitivity and specificity decrease significantly in the patient who is stable or may have other injuries.[56-58] Unlike plain radiography, the reliability of the FAST exam is related to operator experience, patient's body habitus, underlying bowel gas, and the machine itself. The inability to get the critical views makes the scan unreliable, and the examination should not be considered "negative." Thus, in the hemodynamically unstable patient without external signs of bleeding and a clean CXR and PXR, a negative FAST should be interpreted with caution.

The past decade has seen an expansion in the use of ultrasound to include evaluation of the thorax for pneumothorax.[59] The eFAST has been shown in multiple studies to be equal or more reliable in detection of pneumothoraces than CXR.[60-62] Although operator experience always comes into play with ultrasound, the eFAST has the advantage of rapidity and decreased ionizing radiation. However, the true role for eFAST in the trauma patient has not yet been clearly defined. Because the eFAST can detect occult pneumothoraces that might not be found on CXR, caution should be used in making decisions about a tube thoracostomy in those patients.

In those centers with the capability, contrast-enhanced ultrasound has become a potentially viable alternative to evaluate the trauma patient with low-energy mechanisms,



[1] While standard ATLS practices begin with 2 L of crystalloid, in patients with obvious shock and the potential need for massive transfusion, early blood and plasma administration should begin prior to and even in lieu of crystalloid.

[2] While blood loss is the primary reason for shock following trauma in the vast majority of patients, it needs to be emphasized that nonhemorrhage causes of hypotension, such as neurologic, septic, and cardiogenic causes, pericardial tamponade, and tension pneumothorax, need to be identified and treated accordingly.

FIGURE 13-3 Algorithm showing a stepwise approach to a patient presenting in shock. Because time is of the essence, ideally the overall assessment and investigation should take no more than 10 to 15 minutes and should be monitored though performance improvement. Although much has been written about responders and nonresponders to resuscitation, we contend that rapid identification of patients who require emergent intervention is more meaningful. Although nonresponders fit into this group, so do patients who have responded to blood and fluid but still need emergent surgery or interventional radiology without further radiologic or investigatory delay (eg, those with pericardial tamponade, exsanguinating extremity trauma, or severe pelvic fractures). Patients who respond without obvious need for intervention may undergo complete trauma workup as needed. BP, blood pressure; eFAST, extended focused assessment of sonography in trauma; FFP, fresh frozen plasma; HR, heart rate; IV, intravenous; MTP, massive transfusion protocol.

especially in the pediatric population where it is best to avoid ionizing radiation.[63,64] Several studies have demonstrated superior sensitivity, specificity, and positive and negative predictive values as compared to standard ultrasound.

Diagnostic Peritoneal Lavage

Although DPL has been used for over 50 years in evaluation of the trauma patient with suspected intra-abdominal trauma, the adoption of FAST as a noninvasive means of evaluation has caused some to question whether it still has any role in trauma care.[65] However, as the use of FAST has become widespread, potential limitations due to patient, machine, and operator factors have become more apparent, and the role of the DPL in the hemodynamically unstable patient has reemerged.[66] We would submit that there is no longer a need for the actual lavage portion of the procedure, and as currently practiced, it is a direct aspirate of the peritoneal cavity designed to look for

gross blood in the face of hemodynamic instability. Eliminating the lavage mitigates some of the reported disadvantages to DPL, including lack of ability to repeat the study, possibility of altering/skewing CT or ultrasound results with installation of fluid, and high negative laparotomy rate due to its inherent sensitivity. We strongly believe that DPA should not be eliminated from the trauma surgeon's armamentarium. In those instances where the FAST is "negative" in a patient with unexplained hypotension, a DPA should be strongly considered as part of abdominal triage.

CT Scanning

CT scanning of the trauma patient has become a ubiquitous part of the trauma workup. As an adjunct to the secondary survey for the hemodynamically stable patient, there is no doubt that modern CT scanning provides the most accurate means of injury identification over a broad spectrum of possibilities. There is some evidence that the liberal use whole-body scanning identifies clinically significant abnormalities.[67-69] An additional advantage of the "pan-scan" is the ability to discharge patients from the ED safely when the scan is negative.[18] However, there should be some caution in liberally and routinely prescribing total-body CT scans for all trauma patients. In addition to the cumulative radiation over a patient's lifetime, there remains a debate regarding contrast-induced nephropathy, especially in the geriatric trauma patient. The provider should consider all factors, such as mechanism of injury/suspected injury, cooperation of the patient, clinical exam findings, and patient flow, when making decisions as to what scans are needed.

Laboratory

Although there are multiple studies that demonstrate a low utility for certain standard screening laboratory panels in trauma patients, there may be some tests of value depending on the nature of the trauma.[70,71] Although decisions as to appropriate testing can be made based on the severity or perceived severity of the patient, it is often useful to have a "trauma panel" that includes a complete blood count, coagulation profile, and alcohol level. Point-of-care testing should be considered because the results are more rapidly available. All patients should have a type and crossmatch sent. After that, there may be some utility in obtaining an arterial blood gas in patients meeting the highest level of trauma activation.[72,73] This should ideally be able to provide not only the standard values like pH and base deficit but lactate as well. A venous lactate may also provide similar information.[74] For those in shock, thromboelastography (TEG) and thromboelastometry have shown some promise in helping guide resuscitation.[75-77] Although a recent study demonstrated decreased mortality when TEG was used to guide a massive transfusion protocol, along with a decreased use of components, when compared with traditional means of resuscitation, there is a paucity of randomized prospective data as to its true benefit.[78,79]

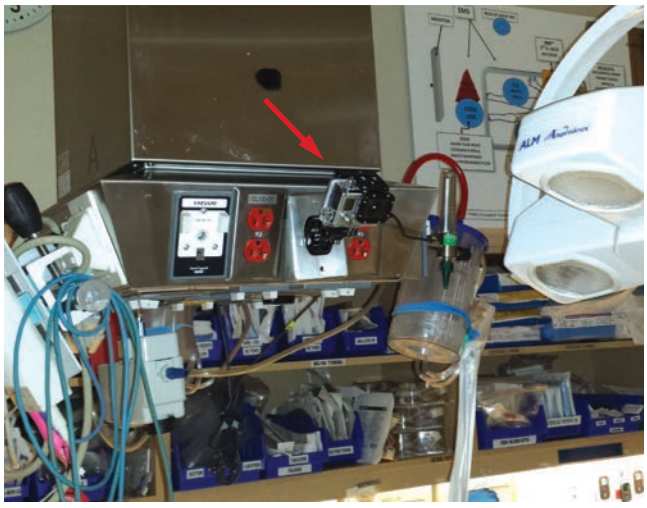


FIGURE 13-4 Small, wide-angled cameras can provide a full field of view during trauma resuscitation for later video review. These devices can be mounted unobtrusively and operated remotely such that the trauma team is unaware of their presence or operations and performs their tasks in a routine manner.

Performance Improvement

With increasing emphasis on short- and long-term outcomes, clinicians taking care of trauma patients should create performance improvement methodology that allows for a thoughtful analysis of each trauma encounter. It should include review of the team's performance during the initial assessment and management of the trauma patient and can begin with identification of appropriate triage of the trauma patient. Flow in the trauma bay, specifically evaluating times to accomplish certain tasks (eg, movement to the operating room or interventional radiology) and timely response of consultants, can also be measured. Although there is no perfect system of recording the events in the trauma bay, the backbone of most performance improvement programs is the trauma flow sheet. These should be completed in sufficient detail that allows the trauma center staff to do a thorough review of any case. Because the primary individuals responsible for charting are the trauma nursing staff, seeking their input in the creation of an institution's trauma flow sheet is invaluable.

First described in 1988, trauma video review has been used in many institutions (Fig. 13-4) and found to be an effective performance improvement tool.[80-82] Although there are medicolegal considerations to its use, trauma video review provides an opportunity to improve performance by highlighting team cooperation, error identification, and educating the entire trauma team.

SUMMARY

The importance of the initial assessment cannot be overstated because it sets the tone for the entire encounter. Recognition and treatment of life-threatening injury form the basis of the initial assessment of the trauma patient. Understanding the

multiple elements that go into the successful performance of the initial encounter with the trauma patient will result in optimal outcomes.

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14

Management of the Airway

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KEY POINTS

- Airway obstruction can occur from excessive bleeding, expanding hematomas, direct anatomic disruption, traumatic swelling, and edema.
- Prehospital airway interventions can range from BLS airway devices, alternative or blind inserted airway devices, to endotracheal intubation.
- Capnography is the measurement of end-tidal carbon dioxide, which is typically 2 to 5 mm Hg lower than the patient's $Paco_2$.
- In the technique of RSI, laryngoscopy and intubation are facilitated by use of sedating induction agents and short-acting neuromuscular blockade.
- Etomidate and ketamine are the most appropriate induction agents in most RSI protocols.
- Adequate preoxygenation along with passive oxygenation during intubation attempts are vital in preventing hypoxia.
- Factors contributing to difficult airways include anatomic features, abnormal vital signs, and airway contamination.
- General guidelines for appropriate depth of an endotracheal tube are 21 cm for adult women and 23 cm for adult men.
- Surgical cricothyroidotomy is contraindicated in children less than 12 years old.
- A commonly used formula in children to select the appropriate size of an uncuffed endotracheal tube (ET) is ET size in mm = 4 + (age in years)/4.

INTRODUCTION

Airway is the first priority for all civilian trauma patients in the prehospital setting, emergency department, and throughout their hospitalization. In all situations, failure to oxygenate and/or ventilate due to an inadequate airway will lead to death within minutes. The airway in a trauma patient may be adequately managed with either noninvasive maneuvers or a definitive airway, most commonly orotracheal intubation. Clinicians charged with caring for trauma patients must be able to quickly recognize a trauma patient in need of an airway intervention as well as develop and sustain the skills necessary required to perform the vast array of lifesaving maneuvers designed to establish and maintain a patent airway. This chapter will present a detailed discussion regarding the assessment and management of the airway for trauma patients in the prehospital setting and emergency department.

AIRWAY EVALUATION

Airway management in trauma patients is most often an emergency intervention complicated by required cervical spine immobilization for blunt trauma patients. The emergent nature of the procedure does not allow time for a detailed and thorough airway evaluation, so every trauma patient should be considered to have a difficult airway to ensure appropriate preparation. However, a brief evaluation of the airway prior to intervention may provide insight to the possibility of a particularly challenging situation.[1,2]

If the trauma patient is conscious and able to cooperate, a brief history may elicit additional risk factors for a difficult airway including obstructive sleep apnea, arthritis, head and neck cancer or radiation, or any difficulty with previous airway interventions. In addition, history of difficult prehospital airway interventions should be a cue that the current emergency department airway management will be challenging.

Although traditional difficult airway scoring systems such as the Mallampati and LEMON (**l**ook, **e**valuate the 3-3-2 rule, **M**allampati score, **o**bstruction, and **n**eck mobility) scores are not applicable in trauma patients due to the emergent nature of the airway intervention, several physical exam findings may be useful to determine increasing difficulty with airway management. Keeping in mind that the vast majority of trauma patients will have limited neck mobility due to cervical spine immobilization and cervical collar, additional physical exam findings that portend a difficult airway include presence of a beard or facial hair, obesity, and evidence of direct injuries to the head, face, and neck.

Trauma patients may present with blunt or penetrating injuries to the head, neck, or face, which ultimately may result in airway obstruction. This obstruction can be immediate or delayed. It is imperative to manage any airway that cannot by protected by the patient or any patient unable to be adequately oxygenated or ventilated. Additionally, the provider needs to anticipate the potential for obstruction and intervene prior to ventilatory failure. Common causes of direct airway trauma include blunt or penetrating maxillofacial or neck injuries, burns, and smoke inhalation. Airway obstruction can occur from excessive bleeding, expanding hematomas (Fig. 14-1), direct anatomic disruption, and secondary traumatic swelling or edema. Smoke inhalation can directly injure the airway mucosa resulting in severe swelling and edema. Providers need to be aware that this is a dynamic process, with the potential for rapid deterioration in the patient's ability to ventilate.

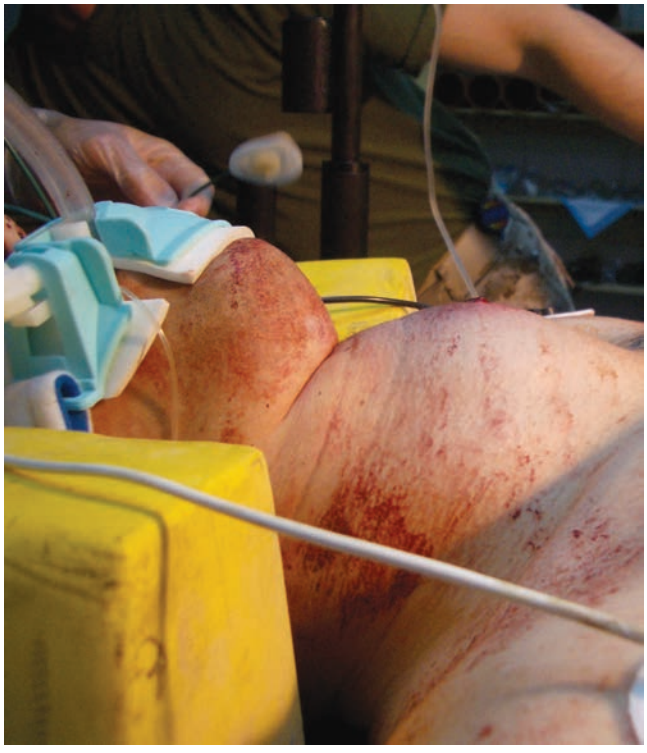

FIGURE 14-1 Large neck hematoma from a gunshot wound causing potential airway obstruction.

Many patients will have obvious signs of airway compromise and need for intervention such as marked decreased level of consciousness, dyspnea, stridor, hypoventilation, or apnea. Other indirect signs include trismus, drooling, odynophagia, or evidence of direct trauma and resulting anatomic abnormalities to the head, oropharynx, or neck.

Providers need to be familiar with signs for potential impending airway compromise including severe bleeding in the oropharynx from mouth or nose, alteration in voice phonation such as hoarseness, subjective sensation of dyspnea despite supplemental oxygen, hematoma in the neck or lower face, and subcutaneous air in the neck or upper chest. Burn patients should be examined closely for evidence of inhalation injury, including singed facial hair, edema, erythema, soot, or worsening upper airway swelling. In addition, initially stable patients may benefit from early airway intervention prior to progression to a difficult airway. This includes the burn patient with signs of inhalation injury, unstable facial fractures, uncontrolled bleeding into the oropharynx, and worsening or fluctuating level of consciousness.

Patients with penetrating trauma to the face or neck may not need cervical spine immobilization unless they present with an obvious neurologic deficit attributable to a cervical spinal cord injury. If the patient is conscious and there is no evidence of cervical spinal cord injury on presentation, a patient with a penetrating injury to the face or neck should be allowed to sit up and lean forward to facilitate spontaneous respirations and removal of damaged soft tissues, blood, and secretions from the airway. The patient may receive medications for intubation while sitting up and may only be laid flat immediately prior to establishing a definitive airway. Despite the gruesome appearance of these injuries at presentation, the majority of patients will be able to have their airway controlled with rapid sequence orotracheal intubation (Fig. 14-2).

PREHOSPITAL AIRWAY MANAGEMENT

Airway management is one the most essential interventions in care of the critically ill or injured patient in the prehospital environment. It is important to recognize the differences in managing airways in the challenging environment outside the hospital versus the reasonably controlled setting of the emergency department or operating room. In-hospital care allows for a well-lit room, proper patient positioning, and access to multiple trained personnel and advanced airway equipment and interventions. Outside the hospital, patients often present with limited access to medical history or known diagnoses and have the additional challenges of the physical setting in which they are being cared. Emergency medical services (EMS) providers may have to treat patients in the dark, in the rain, or in confined environments and may have limited tools with which to work.

Prehospital Priorities

With the evolution of prehospital care, airway management of trauma patients has continued to change. Endotracheal

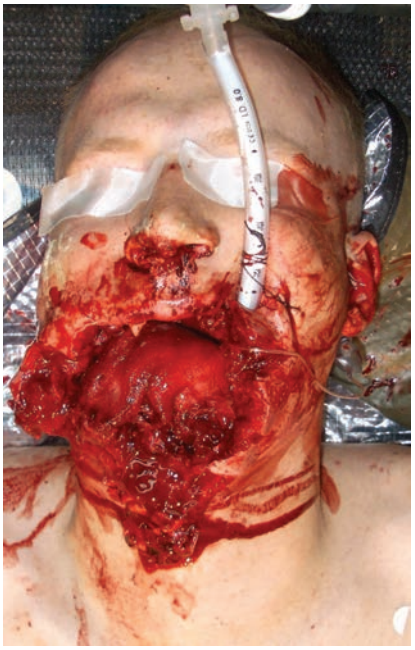

FIGURE 14-2 High-velocity gunshot wound to the face. Patient was managed sitting up and leaning forward and was able to maintain his own airway until immediately prior to successful rapid sequence induction and intubation.

intubation was long considered to be an essential skill for EMS personnel. Severely injured patients were considered in need of a definitive, or "secure" airway, which suggested endotracheal intubation as early as possible in the sequence of the patient's care. However, a large number of studies have demonstrated the difficulty in satisfactorily completing this procedure outside the hospital, with strong suggestions of worse patient outcomes as well as difficulties in developing and maintaining proficiency in the skill.[3,4] Patient environment, competency of the provider, access to induction and paralytic medications, and variable postintubation management have all been postulated as contributing factors to worse or adverse outcomes. Subsequently, there has been a slow shift away from endotracheal intubation as the procedure of choice in addition to the development of numerous alternative airways that may be placed more quickly and reliably by EMS providers. It is becoming apparent that maintaining appropriate oxygenation and ventilation for these patients likely results in improved outcomes rather than establishing a more definitive "protected" airway through endotracheal intubation.

Prehospital Personnel

In addition to a variety of devices, there is significant variability in the skill level of EMS providers. The extent to which they can provide airway interventions is based on their training, licensure, and their state or medical director delegated practice. In the United States, there are essentially three levels of training or licensure in paramedicine. There are first responders, often including firefighters or occasionally law enforcement personnel, who maintain basic life support (BLS) skills including minimally invasive techniques such as bag-valve-mask (BVM) ventilation, and placement of nasopharyngeal or oropharyngeal adjuncts. Some BLS providers (EMT) also have been delegated to use alternative airway devices that can be placed blindly and generally sit in the supraglottic space. Intermediate life support medics, also known as advanced emergency medical technicians (AEMTs), also are typically allowed to place these blind insertion airways and may have the benefit of giving some intravenous (IV) medications. Paramedics, or advanced life support (ALS) providers, generally maintain a skill set that includes endotracheal intubation and, in some systems, are allowed to perform drug-assisted intubations with induction and paralytic medications. Air medical providers typically maintain ALS skills at a minimum and often include respiratory therapists and flight nurses. These personnel are usually highly trained and may have access to more advanced equipment and medications, including video laryngoscopes and transport ventilators.

Prehospital Airway Options

BLS AIRWAYS

Many trauma patients can be managed initially in the prehospital setting with simple BLS techniques (Table 14-1). Oxygenation can be improved with supplemental oxygen provided via nasal cannula or simple facemask. Patients with need for a higher fraction of inspired oxygen or higher flow oxygen can be treated with a non-rebreather mask. In the event of poor respiratory effort, more significant respiratory compromise, or apnea, providers must be competent in providing active ventilatory support via BVM ventilation. Key components for BVM include a self-inflating bag, oxygen reservoir, and conforming facemask. The technique can be performed by one or two providers and includes maintaining a tight seal with the mask and the patient in a jaw-thrust position. Although a single provider can execute the procedure, it is certainly more reliable with two due to the need for good patient positioning and maintaining a firm seal while ventilating. This may be a limiting factor, especially with longer patient transports or with units that have access to only two providers. Other adjuncts used to facilitate ventilation include the oropharyngeal airway (OPA) and nasopharyngeal airway (NPA). The OPA is a rigid, curved plastic device placed into the patient's mouth. It is designed to improve

TABLE 14-1: Prehospital Airway Basic Life Support Options

Device	Oxygen flow	Fio_2
Nasal cannula	2–5 L/min	20%–40%
Oxygen mask	6–10 L/min	40%–60%
Non-rebreather mask	10–15 L/min	Near 100%

airway patency by displacing the tongue away from the posterior pharynx. The NPA is a soft, pliable plastic tube inserted into the patient's nose. One or two can be used, as it helps to lift the soft palate to facilitate air passage. During BVM, either or both of these adjuncts is recommended for use to improve both oxygenation and ventilation and can be used with simple or non-rebreather face masks as well. It should be noted, however, that placement of an OPA may induce gagging or vomiting in less obtunded patients.

ALTERNATIVE AIRWAYS

There has been significant progress in the development of alternative airways for use both in and out of the hospital. There are a number of logistical issues that arise with continued BLS management, most importantly the difficulties of performing adequate ventilation by a single provider over long transports. In addition, there are considerations with developing and maintaining competency with endotracheal intubation skills and the association with worse outcomes. Subsequently, there has been a new emphasis on airway management using devices that can be placed rapidly and reliably by EMS crews.[5] There are a number of terms used for these devices including rescue airway, failed airway device, supraglottic airway, or backup airway. However, these devices are increasingly being used as a primary device in order to minimize potential hypoxic time or other adverse events associated with repeated intubation attempts. Considering that these devices are placed without direct or indirect visualization of the glottic opening, the most appropriate descriptive term is a *blind-insertion airway device* (BIAD). The most commonly used BIADs include the King Laryngotracheal (King LT) airway, the I-gel supraglottic airway, and the laryngeal mask airway (LMA).

The **King LT** consists of a single lumen tube that is placed blindly into the patient's oropharynx and intended to enter the esophagus. A single insufflation port inflates two balloons simultaneously with the distal balloon intended to obstruct any gastric secretions and the proximal balloon intended to protect the airway from above. A ventilation port lies between the two balloons with the intention of being placed directly over the glottic opening. Often the tube will need to be adjusted by advancing or pulling back slightly to improve lung aeration once the tube is placed, and this adjustment may be necessary throughout transport or upon arrival to the emergency department. The King LT comes in multiple sizes for both adult and pediatric use, and some versions include an esophageal port allowing placement of an orogastric tube for gastric decompression. There are a number of studies demonstrating the King LT's effectiveness and ease of reliable placement; however, there are limited data suggesting trauma patients have improved outcomes with the device[6] (Fig. 14-3A and B).

The **I-gel supraglottic airway** looks similar to an LMA but has a soft, gel-like, noninflatable cuff designed to fit snugly over the laryngeal inlet (Fig. 14-4). It has a wider, oblong-shaped tube designed for stability in the patient's mouth and also functions as a bite block. Similar to the King LT, the I-gel

A

B

FIGURE 14-3 King LT airway.

FIGURE 14-4 Intersurgical I-gel airway.

has a port for an orogastric tube and comes in multiple sizes. Both the King LT and I-gel promote the ability to float an endotracheal tube introducer (gum elastic bougie) through the lumen and into the trachea, facilitating the exchange of the device with an endotracheal tube. There are a number of studies promoting the ease, reliability, and speed of the I-gel placement.[7-9] The lack of an inflatable cuff may have the advantage of less time required for placement of the device; in addition, some cadaver studies suggest it may actually provide a better seal and protect the airway better than an LMA. This may also be an advantage in air medical transports at higher altitudes where cuff pressure may become an issue.

The **LMA** was originally designed for use in the operating room. It includes a tube blindly inserted into the oropharynx with a distal inflatable cuff shaped to fit around the laryngeal structures. There are limited studies evaluating its use by EMS providers, and it has not been widely adopted, likely due to concerns regarding inadequate airway protection or risk of becoming dislodged.

ENDOTRACHEAL INTUBATION

Paramedics have maintained skills in endotracheal intubation (ETI) for greater than 25 years, and it continues to be included in their training curriculum. Generally, higher level providers such as paramedics have performed intubation; however, select basic-level EMTs are now being trained in the technique, including both the use of alternate airways as well as multiple intubation methods. ETI has long been considered a vital skill for paramedics to maintain, but recently there has been a large body of evidence linking prehospital intubation with worse outcomes or adverse events. A number of these studies have demonstrated patients arriving to emergency departments with tube misplacements, or they are being hyper- or hypoventilated. Other adverse events that have been noted include prolonged hypoxic times and bradycardia during intubation attempts.[3,4] Additionally, there is wide variability in access to adjuncts for intubation including medications to assist the procedure or advanced equipment such as video laryngoscopes.

In general, there are three main approaches to oral ETI by prehospital providers: direct oral intubation (nonmedicated), sedation-assisted intubation, and full drug-assisted intubation (rapid sequence induction [RSI]). There are still a number of programs that perform nasal tracheal intubation; however, this seems to be rapidly falling out of favor with the development of new techniques and airway devices.

Direct, nonmedicated oral ETI is typically reserved for extremely obtunded or cardiac arrest patients and is associated with a high failure rate due to poor visualization and often lack of confirmatory data such as end-tidal capnography. Sedation-assisted intubation has been promoted to be potentially safer than RSI since the patient theoretically maintains some respiratory drive and native airway reflexes. The concern with this approach is that it still does not provide ideal intubating conditions due to lack of muscle relaxation, and many of the same adverse events occur as in RSI. Success rates are considered borderline, although there is some anecdotal evidence suggesting this approach may ultimately require more attempts with the potential for prolonged hypoxia. There are a number of programs that continue to support drug-assisted intubation using the RSI method in which an IV sedative is given in combination with a neuromuscular blocker. Although the literature suggests that ETI in general may result in worse outcomes, there are select groups to which this may not apply. For example, a study of air medical providers actually demonstrated improved outcomes with this technique. It is postulated that this is likely due to the highly trained nature of the crews, both in the procedure and in postintubation management, and access to a larger relative proportion of high-acuity patients. Outcomes of patients are not based on establishing the airway alone, but rather on overall patient management including access to appropriate medications and airway tools, confirmation of airway placement, and postintubation management. This is evident when comparing performance of groups with varying experience and resources. Ultimately, it is clear that prehospital programs that feel there is a regional need to perform drug-assisted intubation must develop and maintain resources for adequate training and maintaining competency in the skill and postintubation management, must provide access to appropriate tools and medications, and must provide a rigorous quality control program to evaluate and continually improve performance.[4]

One final approach that is gaining popularity in EMS is the idea of a "rapid sequence airway" in which the patient is given IV sedation with or without a paralytic in order to facilitate placement of a device. An alternative airway (BIAD) then is placed as the primary device or if one to two intubation attempts are unsuccessful.

SURGICAL AIRWAYS

Most advanced-level EMS providers will receive training in the procedure of establishing a surgical airway when the situation of "can't intubate, can't ventilate" arises. This typically will be in the setting of severe maxillofacial trauma or facial/neck burns that preclude the ability to place an airway device or perform even BLS airway maneuvers. These procedures are potentially lifesaving, but clearly providers need explicit instruction in the appropriate use of the technique. Similar to ETI, there are a number of different methods that are used. Although a standard surgical approach is still used by some, many groups have adopted prepackaged cricothyrotomy kits that use a Seldinger-type approach with a needle and wire. Due to the infrequency of the procedure, there are few data on success rates or associated complications.

Management of Prehospital-Placed Airways

Upon arrival to the emergency department, it is vital to determine the efficacy and placement of any prehospital-placed airway device. For patients who have undergone ETI, confirmation of appropriate placement can be accomplished with the use of end-tidal capnography and/or direct visualization. Patients presenting with an alternative airway in place present

different challenges to the trauma team, including evaluation of its effectiveness and the need for longer-term assisted ventilation. Questions that need to be addressed upon arrival of the patient with these devices include the following:

- Is the patient being adequately oxygenated and ventilated?
- If so, what is the status of patient, and what other trauma priorities need to be addressed?
- What logistical factors determined the use of the alternative airway? For example, does the patient have a difficult airway, and was it placed after multiple ETI attempts?

General considerations for management include immediately exchanging the device in favor of ETI if the alternative airway is not adequately oxygenating and/or ventilating the patient. If the device seems to be functioning adequately, it is likely prudent to delay any exchange until the primary survey is completed at a minimum. Attempting to exchange the device immediately is often unnecessary and can be associated with complications or delays in getting the patient completely assessed. These devices can often provide adequate airway protection and ventilation long enough for not only the primary and secondary surveys to be completed, but also completion of further ancillary studies determining the need for other surgical interventions or the likelihood for longer-term ventilation support. In the event the alternative device is to be exchanged, preparation is vital and anticipation should be made for a difficult airway. Use of the device may have been secondary to multiple intubation attempts with secondary iatrogenic trauma, or the device itself can create significant oropharyngeal edema or bleeding. Both I-gel and King LT airways promote the use of an endotracheal tube introducer to be floated directly through the lumen of the device into the trachea, facilitating the exchange with an endotracheal tube and potentially reducing interruptions in assisted ventilations and hypoxic time.

EMERGENCY DEPARTMENT AIRWAY MANAGEMENT

Preparation, Monitoring, and Equipment

Successful airway management starts with planning and preparation. Planning begins by assembling an airway kit or cart containing the necessary equipment for intubation and rescue devices. The practitioner should take the time to inventory the equipment prior to the intubation, ensuring function and availability. The implementation of checklists has been demonstrated to be very effective in minimizing medical errors in both the operating room and intensive care settings. An airway checklist can be used to ensure all the appropriate personnel, equipment, and medications are in place before proceeding with any interventions (Table 14-2).

EQUIPMENT

The airway cart should consist of drugs, endotracheal tubes, airways, laryngoscopes, airway adjuncts, a variety of syringes and needles, and equipment to establish a surgical airway.

The primary components of the airway kit are the endotracheal tube and laryngoscope. The airway cart should have a variety of tubes in both cuffed and uncuffed types, including tubes down to a 2.0 internal diameter size for pediatrics, and a variety of stylet sizes. Both Miller and Macintosh laryngoscope blades should be included. In addition, the airway cart should be stocked with both the OPA and NPA in all sizes. In the event of failed intubation, several airway adjuncts should be included in the standard airway cart. An array of devices is now available to assist with difficult intubation and failed airway, including alternative airways, lighted wand stylets, retrograde intubation kits, a variety of video laryngoscopic devices, and the endotracheal tube introducer (gum elastic bougie).

The goal of airway management in the trauma admitting area is to closely simulate the control of the operating room. Key personnel should work as a team to assure a successful intubation. The optimal intubating team should consist of a minimum of three to four members including the intubator, a respiratory therapist, and assistants to assist with the procedure including maintaining cervical spine alignment.

MONITORING AND TROUBLESHOOTING

The intubating team should have the patient attached to a cardiac monitor, blood pressure cuff, and pulse oximeter. IV access should be established. Intraosseous devices can be used as well in the event of delayed or difficult IV access. The patient should receive supplemental oxygen via a nonrebreather mask or a BVM depending on the patient's inherent respiratory drive. The correct size of endotracheal tube should be brought to the bedside, and a correctly sized stylet should be inserted. The endotracheal balloon should be checked for leaks. Once the balloon has been checked, a 10-cm^3 syringe is left attached to the pilot balloon. A suction device with a large catheter tip should be readily available and be placed near the right side of the patient's head. The laryngoscope handle/blade connection should be checked for a functional light source. Video laryngoscopes should also be checked for functionality and appropriate power source.

A means to secure the endotracheal tube should be available. Dentures should be removed just before intubation, particularly if the patient is being bagged and his or her dentures allow for a tight mask seal. If cervical spine injury has been excluded, the patient should be positioned with the neck slightly flexed and the head slightly extended on an imaginary axis through the patient's ears. Placing a pillow or towel under the patient's occipital region and elevating the head approximately 10 cm may facilitate this position. Due diligence with respect to preparation of both personnel and equipment makes for a less stressful intubation and improves the practitioner's chances of successfully intubating the patient.

Pulse Oximetry. The pulse oximeter is a portable, noninvasive, and reliable device that measures Spo_2. Although very accurate in analysis of peripheral oxygen delivery, the pulse oximeter may display inaccurate readings in the case of carbon monoxide poisoning, high-intensity lighting, hemoglobin abnormalities, poor perfusion or pulseless extremity, or severe anemia.

Capnography. End-tidal carbon dioxide ($Etco_2$) detectors measure the partial pressure of carbon dioxide in a sample

TABLE 14-2: An Airway Checklist

Date:_____
Time:_____

Emergency Department
Intubation Checklist

Applies to ALL emergent intubations in the ED outside of the cardiac arrest patient

Equipment Necessary:

☐ Intubation cart – includes DL equipment, multiple ET tubes, endotracheal tube introducers (bougie – standard and malleable), stylets, syringes, Magill forceps, nasal trumpet, oral airway, and rescue airways (LMA, BIAD)
☐ Video laryngoscope (GlideScope, McGrath) including rigid stylet and replaceable blade
☐ Difficult airway cart (in room, or immediately accessible outside room)
☐ Monitor – minimum noninvasive BP, HR, Spo_2
☐ $Etco_2$ monitor
☐ Presence of RSI medications and immediate post-intubation medications
☐ Suction
☐ Supplemental oxygen

☐ Lidocaine	☐ Succinylcholine
☐ Fentanyl	☐ Rocuronium
☐ Etomidate	☐ Versed
☐ Ketamine	☐ Propofol

Personnel Necessary to Be Present:

☐ ED attending physician *(all resident intubations need to have an attending ED physician present)*
☐ Respiratory therapist
☐ (2) RNs
☐ Radiology technicians notified and on standby

Procedure:

☐ Patient on monitor, suction on, and accessible
☐ Preoxygenation per RT protocol
☐ Administration of premedications as indicated by condition (lidocaine, fentanyl, etc.)
☐ Administration of rapid sequence induction medications
☐ Application of continuous passive oxygenation per RT protocol
☐ Intubation procedure
☐ Immediate confirmation of tube placement via visualization and capnometry
☐ Tube secured by RT
☐ Patient placed on vent per RT
☐ Patient placed on continuous $Etco_2$ capnography
☐ Chest x-ray obtained and ETT adjusted as indicated
☐ Initiation of postintubation sedation *and* pain management

Signatures:
Recording RN:_____
Attending Physician:_____

BIAD, blind-insertion airway device; BP, blood pressure; DL, direct laryngoscopy; ED, emergency department; ET, endotracheal; ETT, endotracheal tube; HR, heart rate; LMA, laryngeal mask airway; RN, registered nurse; RSI, rapid sequence intubation; RT, respiratory therapy.

gas. The patient's $Paco_2$ is typically 2 to 5 mm Hg higher than the $Etco_2$, and a normal reading in a trauma patient is approximately 30 to 40 mm Hg. $Etco_2$ reading may be used to confirm placement of an endotracheal tube. The presence of carbon dioxide in the exhaled air strongly suggests correct placement of the endotracheal tube in the trachea in a perfusing patient. The disposable capnometer indicates the presence of carbon dioxide with a color change. The electronic capnometer provides the health care provider with a numerical $Etco_2$ and plots the carbon dioxide concentration against time. Although the use of capnometry as an adjunct to monitor the patient's exhaled carbon dioxide has met some success, conditions such as hypotension, increased intrathoracic pressure, and pulmonary embolus resulting in an increased dead space ventilation may decrease the accuracy of the capnometer.

NONINVASIVE AIRWAY MANAGEMENT

Before any airway maneuver is undertaken, a quick visual inspection of the oropharyngeal cavity should be done. Any foreign or loose material should be swept clear with a gloved finger or removed with suction. Blood may be present in the mouth of a trauma patient, and adequate suctioning is essential to maintaining an open airway. Administration of oxygen prior to suctioning may help prevent secondary hypoxia during the procedure. The tongue can cause airway obstruction in the unresponsive patient as it often lacks tone and falls into the oropharynx. Manual airway maneuvers serve to elevate the tongue out of the hypopharynx.

Jaw Thrust. The cervical spine should be kept in normal alignment. The provider should grasp the sides of the patient's face with fingers 3 to 5 along the ramus portion of the mandible. The provider's thumb is on the patient's cheek and the index finger on the chin and lower lip. These two fingers can open the patient's lips or serve to seal the mask on a BVM. The provider's fingers should form an "E" with the three lower fingers and a "C" with the thumb and index finger. Force is applied to the angle of the mandible forcing the mandible forward and anteriorly, while simultaneously opening the mouth with the index finger on the chin.

Chin Lift. With either a free, gloved hand or another provider's gloved hand, the provider's thumb is placed into the patient's mouth, the patient's lower incisors and chin are grasped, and the patient's mandible is lifted anteriorly. This maneuver supplements the jaw thrust and works to lift the mandible anteriorly, elevating the tongue out of the oropharynx.

Basic Mechanical Airway Devices

OROPHARYNGEAL AIRWAY

Oropharyngeal and nasopharyngeal devices can be inserted into either the mouth or nose of the patient, serving to elevate the tongue out of the oropharynx. The OPA is a curved, plastic or hard rubber device that comes in various sizes and has channeling for suction catheters. The device is sized by placing the OPA in the space between the patient's ear and corner of the mouth. A correctly sized OPA will extend from the patient's mouth to the angle of the jaw.

Indications for the use of the OPA include a patient who is unable to maintain his or her airway or to prevent an intubated patient from biting the endotracheal tube. Advantages for use of the OPA include the following:

1. Prevention of obstruction by the patient's teeth and lips
2. Maintenance of the airway in a spontaneously breathing unconscious patient
3. Ease of suctioning
4. Use as a bite block in a patient who is having a seizure

The OPA is contraindicated in a conscious patient as it may stimulate a gag reflex. In addition, it does not isolate the trachea, nor can it be inserted through clenched teeth. It may obstruct the airway if it is improperly placed and can be dislodged easily. To place the OPA, the mouth is opened and the OPA is inserted with the curve reversed and the tip pointing toward the roof of the patient's mouth. Using a twisting motion, the OPA is rotated into position behind the base of the patient's tongue. Alternatively, a tongue blade can be used to depress the tongue with the OPA placed directly into the oropharynx.

NASOPHARYNGEAL AIRWAY

The NPA is a soft rubber or latex uncuffed tube that is designed to conform to the patient's natural nasopharyngeal curvature. It is designed to lift the posterior tongue out of the oropharynx. Like the OPA, it is indicated for patients who cannot maintain their airway. The advantages of the NPA include ease and speed of insertion, patient tolerance and comfort, and effectiveness when the patient's teeth are clenched. Disadvantages of the NPA include its smaller size, the risk of nasal bleeding during insertion, and lack of utility when a basilar skull fracture is suspected.[2]

The provider should first size the NPA by selecting an NPA that is slightly smaller than the patient's nostril. The distance from the patient's nose to earlobe determines the length. The NPA should be liberally lubricated with lidocaine gel prior to insertion. The right nare is preferentially chosen, as it is typically larger. Gentle pressure should be applied until the flange rests against the patient's nostril. After a basic mechanical airway has been inserted, the patient should be oxygenated with supplemental oxygen or a BVM.

BAG-VALVE-MASK

The BVM assists the provider with oxygenation and ventilation in the apneic or hypoventilating patient. With an effective mask seal and an open airway, the BVM can deliver tidal volumes approaching 1.5 L and nearly 100% inspired oxygen with an attached oxygen reservoir. The BVM consists of a bag with a volume of 1.6 L and a standard facemask attached via a one-way, nonrebreathing valve. A reservoir bag and oxygen source are attached to the opposite end of the bag. Multiple sizes are available to treat neonatal, infant, children, and adult patients. To effectively use the BVM, a first provider should establish a properly fitted mask seal while a second

provider squeezes the bag. Using a basic mechanical airway and proper jaw thrust and chin lift techniques while "bagging" the patient can improve maintenance of oxygenation and ventilation.

ENDOTRACHEAL INTUBATION

The majority of injured patients are able to undergo successful orotracheal intubation. In compliance with the Advanced Trauma Life Support (ATLS) course, the preferred definitive airway is tracheal intubation through the mouth using direct laryngoscopy.

Guidelines for Emergency Tracheal Intubation

INDICATIONS

Indications for intubation relate to the following three simple questions:

- First, is the patient able to oxygenate and ventilate?
- Second, is the patient able to maintain an airway?
- Third, will the underlying injury and physiology of the patient lead to a failure to maintain the airway, oxygenate, or ventilate?

The practice management guidelines of the Eastern Association for the Surgery of Trauma (EAST) published in 2012 calls for emergency tracheal intubation in trauma patients exhibiting the following characteristics[10]:

Trauma patients with any of the following traits:

- Acute airway obstruction
- Hypoventilation
- Severe persistent hypoxemia (Spo_2 ≤90%) despite supplemental oxygen
- Severe cognitive impairment (Glasgow Coma Scale [GCS] score ≤8)
- Cardiac arrest
- Severe hemorrhagic shock

Smoke inhalation patients with any of the following traits:

- Airway obstruction
- Severe cognitive impairment (GCS score ≤8)
- Major cutaneous burn (>40% total body surface area)
- Major burns and/or smoke inhalation with an anticipated prolonged transport time to definitive care
- Impending airway obstruction as follows:
  - Moderate to severe facial burn
  - Moderate to severe oropharyngeal burn
  - Moderate to severe burn as seen on endoscopy

Other relative considerations for intubation include:

- Facial or neck injury with the potential for airway obstruction
- Moderate cognitive impairment (GCS score of 9–12)
- Persistent combativeness refractory to pharmacologic agents
- Respiratory distress (without hypoxia or hypoventilation)

In concordance with ATLS, the EAST guidelines promulgate the concept that orotracheal intubation using direct laryngoscopy is the procedure of choice for airway control following trauma.

RAPID SEQUENCE INDUCTION AND INTUBATION

Overview

Intubation by RSI has become the gold standard for management of the airway in trauma and critical illness. The technique of RSI has been demonstrated to increase intubation success rates and reduce complications compared to pre-RSI techniques in a variety of emergent settings.[11] RSI benefits include provision of optimal intubating conditions for injured patients, rapid airway control, high success rates, and reduction of pulmonary aspiration. The adaptability of RSI to individual patient conditions renders the technique optimal for airway control in the injured.

First developed to facilitate operating room intubations in patients with full stomachs, thereby minimizing risk of aspiration, the technique is now widely used by prehospital paramedics, emergency medicine physicians, and trauma surgeons, with a high reported intubation success rate by nonanesthesiologists. Approximately 80% of intubations performed in North American emergency departments use RSI, with a 90% success rate by the first intubator. In the technique of RSI, laryngoscopy and intubation are facilitated by use of sedating induction agents and short-acting neuromuscular blockade (NMB). RSI can be divided into nine components, the nine P's of RSI, as shown in Fig. 14-5.

It is imperative that providers caring for the injured be familiar with all pharmacologic agents used for RSI, including sedative induction agents, NMB agents, benzodiazepines, dissociative agents, and opiates. A "one method fits all" approach is not always applicable, and each patient should be individualized based on type and mechanism of injury,

The Nine P's of Rapid Sequence Induction and Intubation

- ❑ **Preparation**
- ❑ **Preoxygenation**
- ❑ **Pretreatment**
- ❑ **Preservation of blood pressure**
- ❑ **Paralysis with induction**
- ❑ **Passive oxygenation**
- ❑ **Protection and positioning**
- ❑ **Placement with proof**
- ❑ **Postintubation management**

FIGURE 14-5 The nine P's of rapid sequence induction and intubation.

comorbidities, and potential for adverse events. However, the majority of trauma patients can be effectively intubated using a generalized pharmacologic regimen.

Preparation

Although significant injury with physiologic instability may preclude prolonged preparation for RSI, all efforts should be made to allow for individualized assessment of comorbid conditions, airway status, predictors of difficult intubation, and anticipated pharmacologic regimen. The selection and sequence of pharmacologic agents should be determined, with all agents ready and available in clearly labeled syringes. A *horizontal approach* to the preparatory phase of RSI is optimal, during which multiple personnel with predetermined responsibilities and positions work simultaneously. It should be anticipated that *every* trauma patient requiring ETI will be a difficult airway. There is generally no time or limited ability to perform extensive anatomic evaluations such as Mallampati class or LEMON scores; in addition, these patients often have full stomachs and may have experienced significant maxillofacial or neck trauma. Subsequently it is vital to be prepared with the appropriate personnel and equipment, including the presence of advanced airway backup equipment, devices, and surgical airways. Medications should be chosen based on the physiologic presentation, with thought given to the need for additional sedation, chemical paralysis, analgesia, and blood pressure support in the immediate postintubation phase. All equipment should be prepared and checked as outlined earlier, and the provider should ensure that oxygen access has been established and suction is readily available.

Preoxygenation

Although not originally considered an essential component of RSI, preoxygenation is now considered optimal if the oxygenation, ventilation, and hemodynamic status of the patient permit. The purpose of preoxygenation is to replace the nitrogen-dominant room air occupying the pulmonary functional residual capacity with a 100% oxygen reservoir, prolonging the duration of time before hemoglobin desaturation. Current recommendations for preoxygenation include using a standard reservoir, well-fitting facemask with the oxygen flow rate set as high as possible. Noninvasive, positive-pressure ventilations can significantly improve preoxygenation by using a continuous positive airway pressure mask in the spontaneously breathing patient or gently assisting the hypoventilating or apneic patient with a BVM device. Care must be taken because gastric insufflation may occur, increasing the risk of complications. Recommendations for the duration of preoxygenation necessary for optimal hemoglobin saturation range from 3 to 5 minutes; however, the effectiveness of preoxygenation is dependent on the physiologic status of the patient as well as age, size, and comorbid conditions.[11] For example, an optimally preoxygenated, healthy, 70-kg adult will maintain Sao_2 over 90% for approximately 8 minutes, an obese adult less than 3 minutes, and a 10-kg child less than 4 minutes. More importantly, the desaturation from 90% Sao_2 to 0% occurs much more rapidly than the fall from 100% to 90%. The approximate Pao_2 at 90% Sao_2 is 60 mm Hg, falling to 27 mm Hg at an Sao_2 of 50%. An injured patient with little compensatory reserve can decline from 90% to 0% literally in seconds. Finally, the optimal patient position for preoxygenation will be dependent on the patient's condition. It is known that supine positioning is not ideal as the posterior lung is prone to more atelectatic collapse and it is often more difficult to take full breaths. The ability to sit the patient up in a head-elevated position has been demonstrated to significantly improve preoxygenation ability and prolong times to desaturation; however, this maneuver is limited in trauma patients who may be immobilized for possible spinal injuries. Reverse Trendelenburg positioning can also be used for these supine patients to help facilitate oxygenation.

The concept of a **delayed sequence intubation** (DSI) has gained favor over recent years. It is not uncommon for trauma patients to be significantly agitated secondary to traumatic pathophysiology, hypoventilation, or other intoxicants, inhibiting the ability to adequately preoxygenate. This technique may also be useful in patients who cannot be preoxygenated by other means. DSI involves administration of a sedative, usually ketamine, at procedural sedation doses. The medication is given slowly enough to minimize effects on respiratory drive. This allows for disassociation of the patient to tolerate preoxygenation efforts, including more aggressive maneuvers such as noninvasive positive-pressure ventilation or temporarily placed blind insertion airways. Observational studies suggest that this technique can be used safely and effectively in the hands of experienced providers; however, there needs to be increased awareness of the potential for loss of airway reflexes and the need for immediate intervention.[12]

Pretreatment

Airway stimulation, including laryngoscopy and endotracheal tube placement, results in a *pressor response*, an intense autonomic sympathetic discharge producing tachycardia, hypertension, and increased intracranial pressure. The degree of airway stimulation is proportional to the magnitude of the pressor response. Correspondingly, increased intragastric, intrathoracic, and intracranial pressures may result from Valsalva, bronchospasm, or coughing. As the foundation of RSI relates to abrogating the untoward effects of airway stimulation, preinduction agents can be considered to blunt the physiologic response to laryngoscopy and endotracheal tube placement.

Preintubation medications have both benefit and risk. In order to be effective, these medications should be given at least 3 minutes prior to the intubation attempt. Consideration needs to be given to the potential benefit of the medication and whether the delay created by its administration offsets the need for emergent intubation. Subsequently, these medications should be considered supplementary and not mandatory, and there is no definitive evidence that any of these medications improves outcomes.

Lidocaine. Despite a great deal of controversy regarding the potential benefits of lidocaine during RSI, it is a preinduction agent common to many RSI protocols[13] and is advocated in many emergency airway courses. Lidocaine has a number of theoretical beneficial preintubation effects, including decreasing airway reactivity, the tachycardic response to intubation, and succinylcholine-induced myalgia and fasciculations. It is also proposed that lidocaine reduces the rise in intracranial pressure caused by laryngoscopy, although there has been no high-quality evidence demonstrating this effect. The only absolute contraindications to lidocaine include known allergy or high-grade heart blocks such as third-degree or Mobitz type II. Considering its relative safety, its use should be considered for preintubation, in particular with head-injured patients. Current recommended dosage is 1.5 mg/kg IV, given 3 minutes prior to intubation.

Opioids. Depth of sedation may correlate with speed of intubation in RSI.[14] The sedative and analgesic effects of opioids may provide benefit to the injured patient prior to induction. A commonly used opioid for RSI in the prehospital and emergency department settings is **fentanyl**, which has been shown attenuate the rise in blood pressure and heart rate during RSI. Fentanyl effectively blunts airway reactivity and confers the significant added benefit of analgesia in the injured patient. Fentanyl and other opioids can be associated with respiratory depression and hypotension, which should be taken into consideration with the critically ill or head-injured patient. Current recommended dosage is 3 mcg/kg IV, given over 30 to 60 seconds and 3 minutes prior to intubation.

Atropine. Children, young infants in particular, may experience a pronounced vagal response to intubation attempts. This may be exacerbated by the use of succinylcholine. Current recommendations include the use of atropine for intubation in all children less than 1 year of age and consideration for its use in children under age 5 if using succinylcholine.[15] Dosage should be calculated at 0.02 mg/kg IV with a minimum dose of 0.1 mg and a maximum dose of 0.5 mg. In addition, adults may experience a secondary bradycardia if a second dose of succinylcholine is required. Although shown not to prevent this bradycardia, atropine should be readily available for treatment should bradycardia occur.

Defasciculating Paralytic Agents. **Succinylcholine**, the standard NMB agent used for RSI, produces significant myoclonal fasciculations, prompting a rise in intracranial pressure in patients at risk for intracranial hypertension and the potential for increased oxygen demand. Subsequently, a *defasciculating* dose of a competitive NMB has been previously considered during RSI. Common defasciculating agents include **vecuronium** and **rocuronium** and, less commonly, **pancuronium**. Defasciculating doses are administered as 10% of the paralyzing dose, given 3 to 5 minutes prior to administration of succinylcholine. There is no current high-quality evidence to support the use of a defasciculating dose for patients with elevated intracranial or intraocular pressures, and it is no longer recommended.

Preservation of Blood Pressure

It is common for trauma patients to be hemodynamically unstable, most often caused by hypovolemia due to hemorrhage. Transitioning to positive-pressure ventilation will increase intrathoracic pressure, further decreasing venous return and preload, which can exacerbate this hypotension. In addition, induction medications can directly cause hypotension and the loss of sympathetic drive. The importance of this cannot be understated as even transient drops in blood pressure are associated with adverse outcomes, particularly with head-injury patients. Patients with a **shock index** (heart rate divided by systolic blood pressure) of greater than 0.8 are at increased risk for developing peri-intubation hypotension.[16,17]

Prevention of intubation-induced hypotension begins with appropriate fluid resuscitation. Blood products should be initiated as soon as possible in the hypotensive patient secondary to presumed hemorrhage. RSI medication doses may need to be adjusted, as current dosing recommendations are based on patients without hemodynamic instability. The hypotensive effect of induction medications may be exaggerated when a full dose is administered to a volume-constricted patient. Evolving data recommend reducing their dose by up to 50% in patients with a shock index greater than 0.8.[18,19]

It is unlikely that paralytics have a direct effect on patient's blood pressure; however, consideration should be given to increasing their dose in hypotensive patients. Too small a dose in low-flow conditions may result in inadequate intubating conditions, and a higher dose may be necessary.

Traditionally, vasopressors have been avoided in trauma patients due to concerns for worsening bleeding or unrecognized patient deterioration. However, their use should be considered in high-risk patients such as the severe head-injured patient as they may be beneficial in preventing peri-intubation–associated hypotension. The concept of small "push-dose" or "bolus-dose" pressors has gained popularity in mitigating this hypotensive response, and these agents have demonstrated to be effective in improving hemodynamics in the peri-intubation period. The two most commonly used pressors include epinephrine and phenylephrine; however, epinephrine seems to have some advantages because it has both α and β effects. Consideration can also be given to peripheral IV vasopressor infusions; however, these commonly require more time to establish and administer.

Current recommendations for epinephrine push dose are as follows:

- In 10-mL syringe, draw up 1 mL of epinephrine 1:10,000 and 9 mL of normal saline (equals 10 mL of epinephrine 10 mcg/mL).
- Administer 0.5 to 2 mL every 2 to 5 minutes (5–20 mcg).

Paralysis and Induction Agents

INDUCTION

The perfect **induction** agent would possess rapid onset and elimination, render the patient unconscious but also amnestic, possess analgesic properties, and have negligible side

effects. In injured and critically ill patients, the ideal agent would produce little cardiovascular effects and maintain cerebral perfusion pressure. Regrettably, such an agent does not yet exist. Because many agents produce side effects, including myocardial depression with the potential for hypoperfusion, careful attention should be dedicated to the selection of individual agents. Understanding that all of these medications may create some hypotensive effects, the provider should consider reducing the recommended dose by up to 50% in preintubation hypotensive patients.

Etomidate. Etomidate is a short-acting carboxylated imidazole hypnotic agent frequently used for RSI. It possesses ideal characteristics for urgent and emergent RSI in trauma patients, including rapid onset and clearance, reduction in cerebral metabolic rate, and negligible effects on hemodynamics.[20] This favorable pharmacokinetic profile has led to the widespread use of etomidate for RSI in head injury and hemodynamically labile patients.[21] Accordingly, the American College of Surgeons Committee on Trauma added etomidate to the ATLS course as an induction agent for hypotensive trauma patients. Etomidate decreases cerebral blow flow and cerebral metabolic demand while preserving cerebral perfusion pressure. The most significant side effect of etomidate relates to adrenal insufficiency, as it produces a reversible blockade of adrenal 11-β-hydroxylase. In patients at risk for adrenal insufficiency, including head-injured, mechanically ventilated, and septic populations, etomidate has been independently correlated with reductions in serum cortisol. The controversial question relates to whether transient adrenal suppression produces lasting effects on outcome.[22-24] The majority of research has focused on its use with septic patients. In a systemic review of 18 studies and over 5500 patients, etomidate was not found to increase mortality in septic patients.[24] Further studies are warranted to determine the long-term safety of etomidate for RSI, especially in the setting of trauma. However, given the multiple favorable characteristics of the drug, etomidate remains the standard induction agent in most RSI protocols. Recommended dosage is 0.3 mg/kg IV, onset in 15 to 45 seconds, and duration of action is 3 to 12 minutes.

Ketamine. Ketamine, a rapid-onset dissociative sedative and anesthetic agent, is frequently used for RSI in the pediatric population and in adults with chronic obstructive pulmonary disease. In addition to its sedative effects, ketamine exhibits the beneficial properties of potent analgesia and a partial amnesia and preserves respiratory drive. As a sympathomimetic agent, ketamine may induce tachycardia and increased blood pressure. Secondarily, it is believed to cause bronchodilation, presumably from the release of catecholamines. Controversy has persisted regarding the use of ketamine in patients with head or eye injuries due to suspected elevations in intracranial or intraocular pressures. In actuality, a number of studies demonstrate that ketamine helps to maintain cerebral perfusion pressure and reduce ICP. A number of other studies demonstrated no elevation in intraocular pressures. Ultimately, the evidence to suggest ketamine creates harm in head-injured patients is extremely weak, and considering its associated beneficial effects, hemodynamic stability, and lack of adrenal suppression, its use in RSI in rapidly increasing in popularity.[23] Current recommended dose is 1 to 2 mg/kg IV, with an onset of action in 45 to 60 seconds and a 10- to 20-minute duration of action.

Propofol. Propofol is a nonbarbiturate hypnotic agent that rapidly induces deep sedation and significant relaxation of laryngeal musculature. When used for induction, propofol produces intubation conditions equal to thiopental and equal to or superior to etomidate and, likewise, does not provide any analgesia. Propofol should be used with caution in head-injured or hemodynamically labile patients due to a consistent hypotensive effect and potential reduction of cerebral blood flow. Induction dose is 1.5 to 3 mg/kg IV, with an onset of 15 to 45 seconds and a duration of action 5 to 10 minutes.

Benzodiazepines. Benzodiazepines cause sedation and amnesia through their effects on GABA receptors. **Midazolam** is the most rapidly acting and most commonly used. It does not provide any analgesia but does have anticonvulsant effects that should be taken into consideration in the seizing patient. It usually is associated with hypotension with an average drop of 10% to 25% in the average healthy patient's mean arterial pressure. Subsequently, midazolam should be avoided in the hypotensive or head-injured patient. The usual dose for induction with midazolam is 0.2 mg/kg IV. Other benzodiazepines such as **diazepam** and **lorazepam** are not recommended for RSI.

Barbiturates. **Thiopental** is the most commonly used barbiturate for RSI. Like other induction agents, thiopental has rapid onset and clearance. Thiopental reduces cerebral oxygen consumption and exhibits anticonvulsant effects, rendering it useful in closed-head injury. However, the significant limitation of thiopental use in trauma relates to inhibition of central nervous system sympathetic response. Consequently, thiopental produces reduced myocardial contractility and systemic vascular resistance, inducing hypotension. Therefore, it is best reserved for patients who are euvolemic and normotensive, limiting its application in critically ill patients. **Methohexital** is an additional barbiturate that has been used for RSI. It exhibits similar effects to thiopental, although it is significantly more potent and has shorter onset and duration than thiopental.

PARALYSIS

Pharmacologic paralysis by NMB agents represents an integral component of RSI, facilitating emergent intubation. Paralysis of the musculature improves visualization during laryngoscopy, confers total control of the patient, and reduces complications during intubation. Prehospital NMB has been demonstrated to be safe and to improve intubation success in injured patients undergoing RSI. Potential adverse effects should be diligently assessed, particularly with use of succinylcholine. In addition, preparations for rescue techniques must be made prior to administration of a paralytic agent, in the event of intubation failure. Because paralytic agents provide

no sedative, analgesic, or amnestic effect, it is imperative to combine paralytic use with an appropriate induction agent.

Succinylcholine. Succinylcholine, a depolarizing acetylcholine dimer, acts noncompetitively at the acetylcholine receptor in a biphasic manner to produce muscular paralysis at the motor end plate. Succinylcholine stimulates all muscarinic and nicotinic cholinergic receptors of both parasympathetic and sympathetic systems. Initial brief depolarization results in clinically notable muscular fasciculations, followed by sustained myocyte depolarization. Succinylcholine degradation is dependent on hydrolysis by pseudocholinesterase and is resistant to acetylcholinesterase. Due to rapid onset of action and short half-life, succinylcholine remains the gold standard for RSI in patients not at risk for adverse events. The standard dose of succinylcholine for RSI is 1.5 mg/kg, although recent data suggest a smaller dose of 0.5 to 0.6 mg/kg is sufficient for RSI, facilitating more rapid resumption of spontaneous respiration. Intramuscular injection of succinylcholine has been described, although the required dose, 4 mg/kg, is higher, and onset is slower, than IV injection.

A clear understanding of the potential adverse effects of succinylcholine is critical to its appropriate use in RSI. Contraindications are primarily related to conditions that accentuate the hyperkalemic effects of succinylcholine, as it normally produces a 0.5- to 1.0-mEq/L elevation of serum potassium. Contraindications related to hyperkalemia include thermal injury of age exceeding 24 hours, although receptor upregulation likely does not become clinically relevant until postburn day 5. Therefore, it is safe to use succinylcholine for RSI in most acute burns. Contraindications include crush injury or rhabdomyolysis with hyperkalemia, congenital or acquired myopathies, and conditions of subacute and chronic upper and motor neuron denervation including paralysis and polyneuropathy of critical illness. Contraindications related to known comorbid conditions include history of malignant hyperthermia and pseudocholinesterase deficiency. In addition, succinylcholine is reported to raise intragastric and intracranial pressure due to muscle fasciculations and may contribute to increased intraocular pressure. It should be used with caution in head injury and penetrating globe injury, although the evidence that succinylcholine raises intraocular pressure is anecdotal at best.

Nondepolarizing Agents. Nondepolarizing NMB agents, through competitive blockade of acetylcholine transmission at postjunctional, cholinergic nicotinic receptors, provide an alternative for patients not appropriate for succinylcholine. The aminosteroid compounds, including rocuronium, pancuronium, and vecuronium, represent the commonly used NMB agents for RSI. Nondepolarizing agents for RSI are selected based on the ability to best approximate the rapid onset and elimination of succinylcholine. The most intensively studied nondepolarizing agent used for RSI is **rocuronium**, which exhibits short onset and intermediate duration of action. When contraindications to succinylcholine exist, rocuronium produces acceptable intubating conditions and should remain in the RSI armamentarium as an alternative to succinylcholine. A number of studies have demonstrated no significant differences in intubating conditions with either medication. Proponents against nondepolarizing agents point to their long duration of action in the event the patient cannot be successfully intubated. However, with the addition of newer rescue airway devices and the lack of potential hyperkalemic effect, it has become the preferred paralytic to many providers. As mentioned, consideration should be given to increasing the recommended dose from 1.0 mg/kg up to 1.2 to 1.6 mg/kg in the hypotensive patient to improve intubating conditions.

Sugammadex is an agent developed to rapidly reverse the effects of nondepolarizing agents by binding and encapsulating them, including both rocuronium and vecuronium. Demonstrated to be very effective in reversing the paralytic effect of these drugs, it is approved for use in Europe; however, it has not been approved for use in the United States. In the eventuality that it does become available, nondepolarizing agents likely will become the paralytic medication of choice (Table 14-3).

Passive Oxygenation

Passive, or apneic, oxygenation refers to continuously exposing the oropharynx to high-flow oxygen despite no intrinsic respiratory effort. Oxygenation is a passive process; alveoli will continue to diffuse oxygen into the pulmonary capillary vasculature regardless of diaphragmatic movement. Respiratory movement simply helps expose the alveoli to the oxygen in the environment and force out the accumulated carbon dioxide. By maintaining a high enough partial pressure of oxygen, it will continue to diffuse into the bloodstream. The technique is performed by a number of other specialties, including during bronchoscopies and otolaryngeal procedures.[25] A large number of studies have demonstrated the Pao_2 can be maintained for markedly prolonged periods of time using this method. Eventually patients will become acidotic secondary to carbon dioxide accumulation; however, this technique can significantly help maintain oxygenation during the intubation process and is vital to those patients with poor reserve or at risk for the secondary insult of hypoxemia, such as traumatic brain injuries.[11] Current recommendations are to apply oxygen via nasal cannula to the patient at 15 L/min after preoxygenating the patient and during the intubation procedure. A patent passage to the lungs is vital and can be augmented by use of a nasal pharyngeal airway.

Protection and Positioning

Protection refers to maneuvers used to minimize the risk of aspiration as well as steps to decrease the risk of exacerbating any unstable cervical spine injuries. The Sellick maneuver is the process of an intubation assistant applying direct, downward pressure on the cricoid cartilage using his or her thumb and index or middle finger during BVM ventilations and during the intubation attempt. It was felt that this pressure would compress the esophagus between the cricoid cartilage

TABLE 14-3: Pharmacology of Rapid Sequence Induction and Intubation

Agent	Dose	Onset	Duration	Precautions
Premedication agents				
Fentanyl	1.0–5.0 mcg/kg	30–45 s	0.5–1.0 h	Chest wall rigidity
Midazolam	0.1–0.3 mg/kg	2–3 min	2–3 h	Slow onset
Lidocaine	1.0–1.5 mg/kg	30–90 s	10–20 min	Hypotension
Esmolol	2.0 mg/kg	2–5 min	9–30 min	Hypotension
Vecuronium	0.01 mg/kg	90–120 s	60–75 min	Extended duration
Induction agents				
Etomidate	0.05–0.15 mg/kg	10–60 s	2–5 min	Adrenal suppression
Propofol	0.5–2.0 mg/kg	10–50 s	2–10 min	Hypotension
Thiopental	1.0–3.0 mg/kg	10–60 s	5–30 min	Hypotension
Methohexital	1.0–3.0 mg/kg	30 s	5–10 min	Hypotension
Ketamine	0.5–2.0 mg/kg	30–120 s	5–15 min	ICP[a]
Neuromuscular blockade				
Succinylcholine	0.6–1.0 mg/kg	30–60 s	5–15 min	Hyperkalemia
Rocuronium	1.0 mg/kg	45–60 s	30–60 min	Long duration

[a]Concerns for elevated ICP with ketamine have been refuted in multiple studies.

and the vertebral bodies. There are some studies suggesting this maneuver may help reduce gastric insufflation during BVM ventilations; however, there is minimal evidence demonstrating a reduced incidence of aspiration. In addition, some studies indicate it may contribute to airway obstruction and difficult intubations.[26] Currently, most expert opinions consider the use of the Sellick maneuver to be optional. However, if it is used, the provider performing the intubation needs to control the direction and amount of force applied.

Unless the patient's cervical spine is considered clear of potential injury, immobilization must be maintained. Direct laryngoscopy in particular can impart a significant degree of force to the patient's oropharynx with the potential for lifting, rotating, or flexing or extending the neck. To minimize this and the potential impact on an unstable cervical spine injury, in-line immobilization should be maintained by an assistant, with the provider maintaining pressure on both of the patient's mastoid processes. This allows for a well-controlled head with minimal movement at the cervical spine and the ability to remove or open the cervical collar to minimize its limitations of accessing the airway and neck.[27]

Positioning refers to the position of the patient. The patient's bed should be at an appropriate height for the intubator. In addition, in the rare instance the cervical spine can be cleared prior to intubation attempts, placing a towel, blanket, or sheets directly under the patient's occiput will lift and slightly extend the head in order to optimize viewing the glottis by direct laryngoscopy by optimizing the alignment of the oral, pharyngeal, and laryngeal axes. A functioning large-bore suction catheter should be within arm's reach of the intubator at all times.

Placement with Proof

Success in intubating the injured patient is dependent on thorough knowledge of the anatomy of the upper airway and a meticulous adherence to proper technique. The vocal cords lie posterior and inferior to the pliable epiglottis, which should be visualized as a constant reference point during laryngoscopy. The posterior-most esophagus may be lifted into view with sufficient elevation of the epiglottis. Following preparation of equipment and personnel, the laryngoscope is grasped firmly with the left hand. It should be emphasized that the right hand should be kept free, for suctioning, manipulation of oral structures, and placement of the endotracheal tube. Selection of a straight versus curved blade has less to do with proven efficacy in a particular scenario than the comfort and proficiency of the intubator with a particular blade. In general, the straight blade is used to pass beneath, and directly elevate, the epiglottis. The straight blade is inserted into the esophagus, with the blade withdrawn slowly under direct visualization to expose the glottic opening. The same technique can be applied with a curved blade of sufficient size, although the curved blade technique typically uses insertion of the tip of the blade into the vallecula, with anterior traction of the epiglottis, exposing the glottic opening. The tongue should be displaced by the blade to the patient's left for best visualization of the epiglottis. The motion and direction of the laryngoscope in the left hand during laryngoscopy are of critical importance to safe and successful intubation of the trachea. The proper technique of laryngoscopy employs upward motion of the laryngoscope in the parallel plane of the handle. A "rocking" motion, during which the handle is rotated counterclockwise and posterior, should never be used. This posterior circular motion can impart dangerous extension on the cervical spine or fracture or dislodge teeth. In addition, the left elbow should not be placed on the bed or spine board for stabilization. As the blade is positioned to visualize the glottic opening, the right hand can be used to employ the "BURP" maneuver if the glottic opening is not readily visible. This technique includes **b**ackward-**u**pward-**r**ightward **p**ressure on the thyroid cartilage and is distinct from the Sellick maneuver.

Once the glottic opening is visualized, the tube and stylet should be maneuvered through the cords. If the view is limited, an endotracheal tube introducer (gum elastic bougie) can be placed through the cords first, with confirmation of its placement noted by the distinctive "clicks" felt as the distal portion of the introducer rubs against the tracheal rings. The endotracheal tube without a stylet can then be placed over the top of the introducer directly into the trachea. Once the tube is placed, the cuff should be inflated and the stylet or introducer withdrawn.

Using the centimeter markings on the external surface of the tube can make an estimation of appropriate tube depth. General guidelines for appropriate depth are **21 cm for adult women** and **23 cm for adult men** when measured at the corner of the mouth. The most accurate equation for estimating endotracheal tube depth in children is **depth = age/2 + 13**.[28] Tube placement should always be confirmed by x-ray and adjusted as necessary.

Confirmation of proper endotracheal tube placement is crucial as unrecognized esophageal intubations can lead to devastating complications. The most accurate method of determining this is via end-tidal carbon dioxide detection.[28] This can be done initially with a colorimetric device that reacts with the acidic component of the exhaled carbon dioxide. It is reliable with minimal false positives; however, it is only useful for the first few breaths of the patient. It is now recommended that intubated patients be continuously monitored on a quantitative device (capnometry) or with capnography, in which the exhaled carbon dioxide air is both graphically and quantitatively displaced. These devices are extremely useful not only for recognizing missed or dislodged intubations, but also to significantly improve noninvasive management of the ventilated patient.

Clinical indicators of intubation including tube misting, lung auscultation, and tube visualization through cords cannot be relied on to confirm appropriate tube placement. A chest x-ray should be obtained immediately after intubation; however, it is most useful for endotracheal tube depth and not necessarily endotracheal placement confirmation. Regardless, esophageal intubations need to be recognized as soon as possible and cannot wait on the time necessary to acquire an x-ray.

Postintubation Management

Once endotracheal tube placement is confirmed, the tube needs to be properly secured to the patient to minimize risk of displacement. Tape can be used, although it is not as well tolerated as some of the newer proprietary tube holders. The chosen method needs to be readily available and secure.

As mentioned, a chest x-ray should be obtained to determine appropriate endotracheal tube depth. Close attention should be paid toward any radiographic signs of pneumothorax as this can quickly convert to a tension pneumothorax now that the patient is under positive-pressure ventilation.

Hypotension is a common event immediately after intubation, occurring in approximately 25% of emergent intubations, despite appropriate medication selection. Roughly 2% of critically ill patients will experience cardiac arrest after intubation.[29-31] Proposed causes include decreased venous return due to positive-pressure ventilation, loss of autonomic vascular tone, and lingering effects of the induction medications. Treatment consists of maintaining adequate preload with appropriate fluid and blood resuscitation. Close attention should be paid to airway pressures and the potential development of a pneumothorax. Consideration can also be given to a titratable vasoconstrictor, such as phenylephrine or epinephrine, to be readily available and administered to maintain vascular tone and augment venous return in those patients hypotensive either prior to the procedure or immediately after intubation. Finally, providers need to be cognizant of the potential acid-base status of the patient. Many hypotensive trauma patients will be experiencing a metabolic acidosis secondary to poor perfusion. An increased ventilatory rate will be needed to help compensate for this and can be easily overlooked.

Analgesia and additional sedation also need to be addressed. Despite the induction medications, intubation is an uncomfortable procedure, and many patients will begin to start "fighting" the tube. Fentanyl is an easily titratable medication and more hemodynamically stable than the other opiates. The induction medications are metabolized fairly quickly, and the patient will likely require additional sedation and occasionally paralytics. The choice of sedation is determined by the need for neurologic reassessments and the hemodynamic status of the patient. Propofol is an effective medication and easy to titrate, and its quick "on/off" action allows the patient to be easily reassessed. Benzodiazepines such as lorazepam and midazolam are also used and have a more prolonged effect. All of these can be associated with induced hypotension, particularly with higher doses, and benzodiazepines have also been correlated with intensive care unit delirium. Dexmedetomidine (Precedex) is a newer medication gaining popularity that offers a variety of advantages, including analgesic effects, anxiolysis, hypnosis, and no respiratory depression. The two major downsides of dexmedetomidine are bradycardia, which is dose dependent and can be avoided, and cost. Finally, ketamine has anecdotally been shown to be effective in bolus dosing or as an infusion, in particular with the hypotensive trauma patient; however, prospective studies on its safety and efficacy for this purpose are still lacking.

VIDEO LARYNGOSCOPY

The morbidity and mortality associated with the difficult airway, compounded by potential litigation and the burgeoning epidemic of obesity, have fostered rapid growth in video laryngoscopy (Fig. 14-6). Many devices are now available, including the Verathon GlideScope, Truphatek Truview EVO2, Pentax Airway Scope, McGrath MAC Enhanced Digital Laryngoscope, Storz C-MAC Video Laryngoscope, and AirTraq Laryngoscope, among others. All devices have a rigid blade for laryngoscopy combined with videoscopic visualization of the anatomy in proximity to the end of the blade. The visual field is located through an eyepiece in proximity to the blade handle or on a separate portable monitor. The technology provides direct visualization of the glottic opening,

FIGURE 14-6 Video laryngoscope.

facilitating intubation. The specific features and characteristics of the video laryngoscopes vary, but they each provide an indirect view of the upper airway and a theoretical advantage in minimizing cervical spine motion during intubation.[32-35] Video laryngoscopes are being used in essentially any scenario in which a difficult airway may be encountered, including the emergency department, operating room, and intensive care unit. Application in the prehospital environment has been limited by financial constraints, lack of comparative data, and need for provider training. In comparisons of the effectiveness of video and conventional laryngoscopy, visualization of the glottis has been shown to be superior using video devices, but data regarding speed and efficacy of intubation are conflicting. As noted in the most recent EAST trauma guidelines, video laryngoscopy may offer significant advantages over direct laryngoscopy, including the following[10]:

- Superior views of the glottis (Cormack-Lehane I/II)
- Higher intubation success rates in patients with anatomically difficult airways, in obese patients, and in those with the cervical spine held in line
- Higher intubation success rates by inexperienced airway providers

Direct laryngoscopy (DL) by trained physicians, with the use of in-line stabilization for cervical spine protection, has been shown to be a safe and effective means of intubation in trauma patients. Despite the good success rates with DL, the use of video laryngoscopy (VL) and the studies evaluating its use have been increasing. In a review of current literature, the EAST Trauma guidelines summarize that VL provides an improved Cormack-Lehane view grade, cervical spine motion is lessened, force and pressures exerted on the airway are decreased, hemodynamic responses are minimized, and time to intubation is similar with VL as compared with DL.[10,36,37] One study specifically looking at patients intubated with cervical spine immobilization by a semirigid collar showed improved views with the GlideScope compared with a Macintosh blade. A large retrospective study of 822 emergency department intubations (of which >60% were trauma patients) showed that overall success rates for VL were similar to DL, but first-attempt success rates were higher using the GlideScope.[36] There are a number of challenges in studying the effectiveness of VL versus DL due to the variety of devices available, familiarity with these devices, and experience level of the providers with the use of either direct or video laryngoscopes. A Cochrane review published in 2017 concluded that video laryngoscopes reduced the incidence of intubation failure, particularly in the context of a difficult airway.[38] The importance of first-pass success rates cannot be understated considering the morbidity associated with multiple attempts and missed intubations.[39] Video-assisted devices may have a greater role for patients with potentially difficult airways in whom DL may not provide good visualization, especially in the setting of trauma and the limitations imposed by patient positioning and maintenance of cervical spine precautions.

Additional studies will undoubtedly be forthcoming. It is clear that VL demonstrates some advantages over DL, especially in the trauma patient or the patient with a difficult airway. In consideration of these advantages, gradually decreasing costs, and increased provider training in its use, VL will likely become the preferred primary airway modality.

ADJUNCTS TO ENDOTRACHEAL INTUBATION

The inability to establish a definitive airway or provide effective oxygenation and ventilation to a trauma patient in a timely manner can have devastating effects on their outcome. A number of devices have already been mentioned to help facilitate intubation including the use of video laryngoscopes and endotracheal tube introducers. If using DL, it is imperative to have an endotracheal tube introducer (bougie) immediately available, and some popular airway courses recommend it to be used routinely. Visualization of the patient's vocal cords can often be limited. The endotracheal introducer can often be placed significantly more easily than the endotracheal tube itself and can even be placed blindly using the "feel" of the device as it passes along the tracheal rings. The endotracheal tube can then be advanced directly over the introducer. They are inexpensive and come in multiple types, including malleable introducers that can be formed to maintain the same shape and curve of a video laryngoscope. This can prove quite valuable in the event it is difficult to pass the tube past the placed VL handle and blade.

Although previously mentioned, a stylet is very useful to "shape" the endotracheal tube to provide stability, improved control of the tube, and assist placement. Most video laryngoscope manufacturers recommend the use of proprietary rigid stylets that duplicate the same severe angle of the VL blade. Since the cords are indirectly visualized, the tube is forced to travel around the bend of the oral pharynx into the laryngeal and tracheal space, which the rigid stylets facilitate.

Two other types of stylets include the lighted and optical versions. Lighted stylets have a bright light source at the distal tip, allowing visualization of the glottis through the soft tissues of the anterior neck to confirm placement. Studies demonstrate it does take some time to acquire the skill in using the device with secondary longer times to intubation; however, it has been demonstrated to be useful in anteriorly located airways.[40,41] Optical stylets maintain a video viewing component at the distal end of the stylet and come in different forms including rigid and directable options. The endotracheal tube is loaded onto the stylet prior to the procedure, and then the stylet is advanced until visualized passing through the cords, usually through an attached eyepiece. Few data exist evaluating their comparative effectiveness, and it is generally believed their utility would be limited in airways with significant contamination, such as blood or emesis.[42]

It is vital to remember the importance of maintaining adequate ventilation and oxygenation. Allowing head-injured patients to become hypoxic or hypotensive has been well established to lead to secondary insult and significantly worse outcomes. In the event that an endotracheal tube cannot be easily introduced, alternative devices should be readily available and placed before a patient is allowed to become hypoxic. These devices are well described in the prehospital portion of this chapter but can also be very effective in the emergency department or operating room. Considered to be "extraglottic" because they do not enter the trachea, these airways are typically placed blindly. Examples include the LMA and the I-gel. The LMA is designed to form a seal with an inflatable cuff over the glottic opening and comes in multiple models. These include an intubating LMA, which allows passage of an endotracheal tube directly through the device into the trachea. The I-gel looks very similar to the LMA but has a noninflatable cuff made of a gel-like flexible polymer. There are several studies demonstrating the effectiveness of all these devices, including ease of reliable placement, effective temporary ventilation and oxygenation, and use in managing difficult airways.[7,8,43]

Flexible endoscopes are expensive, long, fiberoptic stylets that can be directed into the patient's trachea. They can be used both for orotracheal and nasotracheal intubation and typically take significant time to place an endotracheal tube. Subsequently, they are not well suited for emergent management of airways in the critically injured trauma patient.

UNIQUE CHALLENGES IN TRAUMA AIRWAY MANAGEMENT

Physiologic Challenges

There a number of nonanatomic patient factors that can influence the outcome of air management. These factors can dramatically worsen patient outcomes and thus should be considered in all patients, in addition to anatomic components, when preparing and planning for intubation. Whenever possible, these physiologic factors need to be prepared for and corrected prior to the procedure.

Considering that the majority of trauma patients requiring intubation are head injured, it is imperative to minimize the risk of a secondary brain injury. It is well documented that allowing these patients to become hypoxic and/or hypotensive leads to significantly worse clinical outcomes, with increases in both morbidity and mortality. The procedure of pharmacologically assisted intubation can create either of these conditions, and diligence must be maintained to minimize the extent. The process requires patients to become apneic, occasionally for several minutes, during the procedure. Combined with a patient who may have decreased pulmonary reserve, this may lead to rapid oxygen desaturation. Appropriate preoxygenation and continuous passive oxygenation are vital to minimize this risk. As previously described, the concept of DSI can be used to facilitate the preoxygenation process. An IV sedative is given at a dose to provide procedural sedation levels. Ketamine is often selected due to its hemodynamic stability and minimal effect on respiratory drive. This delayed approach can assist in improving preoxygenation levels in the combative or obtunded patient. Once sedated, supplemental oxygen can be provided by non-rebreather, BVM with or without a positive end-expiratory pressure valve, or even noninvasive positive-pressure ventilation to maximize alveolar nitrogen washout and oxygen saturation. The goal is maintaining an oxygen saturation greater than 94% for at least 3 minutes prior to making the intubation attempt.

Trauma patients are also at greater risk for developing hypotension during intubation. In addition to the direct effects of traumatic injuries with potential hypovolemia or neurogenic shock, patients may become hypotensive secondary to direct effect of the induction medications or loss of autonomic tone, in addition to converting to positive-pressure ventilation, which can further decrease venous return. In additional to worse outcomes in head-injured patients, preintubation hypotension has been identified as an independent risk factor for peri-intubation cardiac arrest.[30,31,44]

There are several proposed procedures intended to minimize the extent of this hypotensive response. The first is adequate volume resuscitation; in trauma patients with suspected significant hypovolemia secondary to blood loss, this implies early and aggressive resuscitation with blood products. As mentioned, there is also a proposed benefit to decreasing the dose of induction sedation medications and increasing the paralytic dosing. Finally, "push-dose" or "bolus-dose" vasopressors can be used to help sustain blood pressure and mitigate the effects of the induction medications and positive-pressure ventilation. Both phenylephrine and epinephrine have been proposed as agents for this procedure; however, most experts in resuscitation seem to prefer epinephrine due to its effects as an α- and β-agonist, which can increase vascular resistance and blood pressure and also augment cardiac output.

Contaminated Airway

Airways in trauma patients are frequently contaminated by blood, secretions, injuries from maxillofacial trauma, or emesis, leading to increased difficulty in successful intubation.[45] The operator must be prepared for dealing with these circumstances and anticipate this difficulty. James Decanto, MD, an anesthesiologist specializing in airway management training,

has promoted the use of the "SALAD" technique, referring to **s**uction-**a**ssisted **l**aryngoscopy **a**nd **d**econtamination. He promotes using a significantly larger bore suction catheter to minimize obstruction of the device by any debris in the oropharynx. He then promotes aggressive suctioning of the mouth and oropharynx prior to introducing the laryngoscope blade and then using the suction device as a type of tongue depressor to facilitate ideal placement of the blade. Once the blade is in the mouth, the suction catheter can be advanced into the proximal esophagus to provide continuous drainage. The catheter is then pushed to the left side of the patient's mouth and held in place by the laryngoscope blade during the intubation procedure. He further recommends suctioning of the endotracheal tube prior to any ventilations to minimize spread of any aspirated material. One option of this technique is to use the larger bore suction catheter in a similar fashion to the endotracheal tube, using it to intubate the trachea, and then disconnecting it from suction. It can then be used as a conduit for an endotracheal tube introducer (bougie). The catheter is then removed over the introducer and replaced with an endotracheal tube.

Suspected Cervical Spine Injuries

Trauma care providers typically proceed under the assumption that the patient has an unstable cervical spine injury, and patients are traditionally placed in cervical collars and often transported on a rigid backboard, despite little evidence to support that these measures prevent secondary spinal cord injury. These patients are typically supine, limiting the ability to optimally position the patient, and cervical collars or manual in-line stabilization can limit visualization. It is vital that providers tasked with maintaining in-line stabilization immobilize the head and neck without limiting mouth opening or mandibular movement. Evidence suggests these limitations are more pronounced with DL, resulting in higher occurrence of poor views, longer intubation times, and more frequent failed intubation attempts.[46,47] Video laryngoscopes have demonstrated improved glottic views in immobilized patients; however, this does not always translate to improved success rates because there are still challenges in delivering the tube via the hyperangulated approach.

Providers need to be prepared for difficult visualization of the cords or challenges in passing the endotracheal tube and should choose the device in which they have the most experience. Rigid cervical collars should be opened and replaced by manual in-line stabilization. Finally, endotracheal tube introducers may be beneficial is assisting tube placement.

An algorithm for airway management in trauma patients is presented in Fig. 14-7.

MANAGEMENT OF PATIENTS WITH DIRECT AIRWAY TRAUMA

Trauma patients often can present with blunt or penetrating injuries to the head, neck, or chest, which ultimately may result in airway compromise. This compromise can be immediate or delayed. It is imperative to manage any airway that cannot by protected by the patient or in any patient who cannot be adequately oxygenated or ventilated. Additionally, the provider needs to anticipate the potential for delayed obstruction and intervene prior to respiratory failure.[48-50]

Management of patients with direct airway trauma will depend on the severity of the injury and criticality of the patients at that particular time. A subset of patients may be candidates for close observation if they maintain normal mental status and normal vital signs, including pulse oximetry, and do not demonstrate any evidence of impending airway compromise. The majority of patients needing intervention with direct airway trauma can be managed with the described RSI technique; however, it is imperative to recognize that these patients are at significantly higher risk of becoming a difficult intubation. Preparation is vital including immediate access to airway adjuncts and a surgical airway kit. This is especially true in in injuries resulting in severe oropharyngeal trauma or a hematoma within the anterior neck. In patients with direct penetrating trauma or clothesline-type mechanism resulting in an exposed trachea, direct insertion of either an endotracheal tube or tracheostomy tube through a tracheal defect is an alternative option (Fig. 14-8).

In certain patients, there will be a high level of concern for a difficult intubation due to factors such as the extent of injury, excessive bleeding or swelling, facial deformities, or body habitus. In these patients, it is best to prepared with a "double setup" in which the neck is simultaneously prepped and equipment accessed for a surgical airway placement during the intubation preparation phase. Thus, in the event of unsuccessful intubation or failed airway, there is minimal time delay to secure access to a definitive airway. In the event of an associated upper airway obstruction that cannot be immediately cleared, providers should move directly to a surgical airway.

Another approach gaining popularity is the concept of an "awake" intubation, or utilization of the delayed sequence technique in patients with a high suspicion for difficulty placing the endotracheal tube successfully. Ketamine is often selected due to its hemodynamic stability and minimal effect on respiratory drive and can be supplemented with a topical anesthetic, such as atomized or nebulized lidocaine. Once sedated, supplemental oxygen can be provided by nonrebreather, BVM, or even noninvasive positive-pressure ventilation to maximize alveolar nitrogen washout and oxygen saturation. This is especially helpful for preoxygenation of the delirious or combative patient. The provider can then attempt to "look" at the airway using DL or VL techniques. If the cords are adequately visualized, the provider can then either attempt the intubation or subsequently have a chemical paralytic administered to facilitate placement of the tube. Current literature surrounding DSI is limited. One study including mostly nontrauma patients noted improvements in oxygen saturation with preoxygenation interventions and did not observe any complications.[12] It is important to note that this concept has not been extensively studied in the trauma population, and its application should be used with caution.

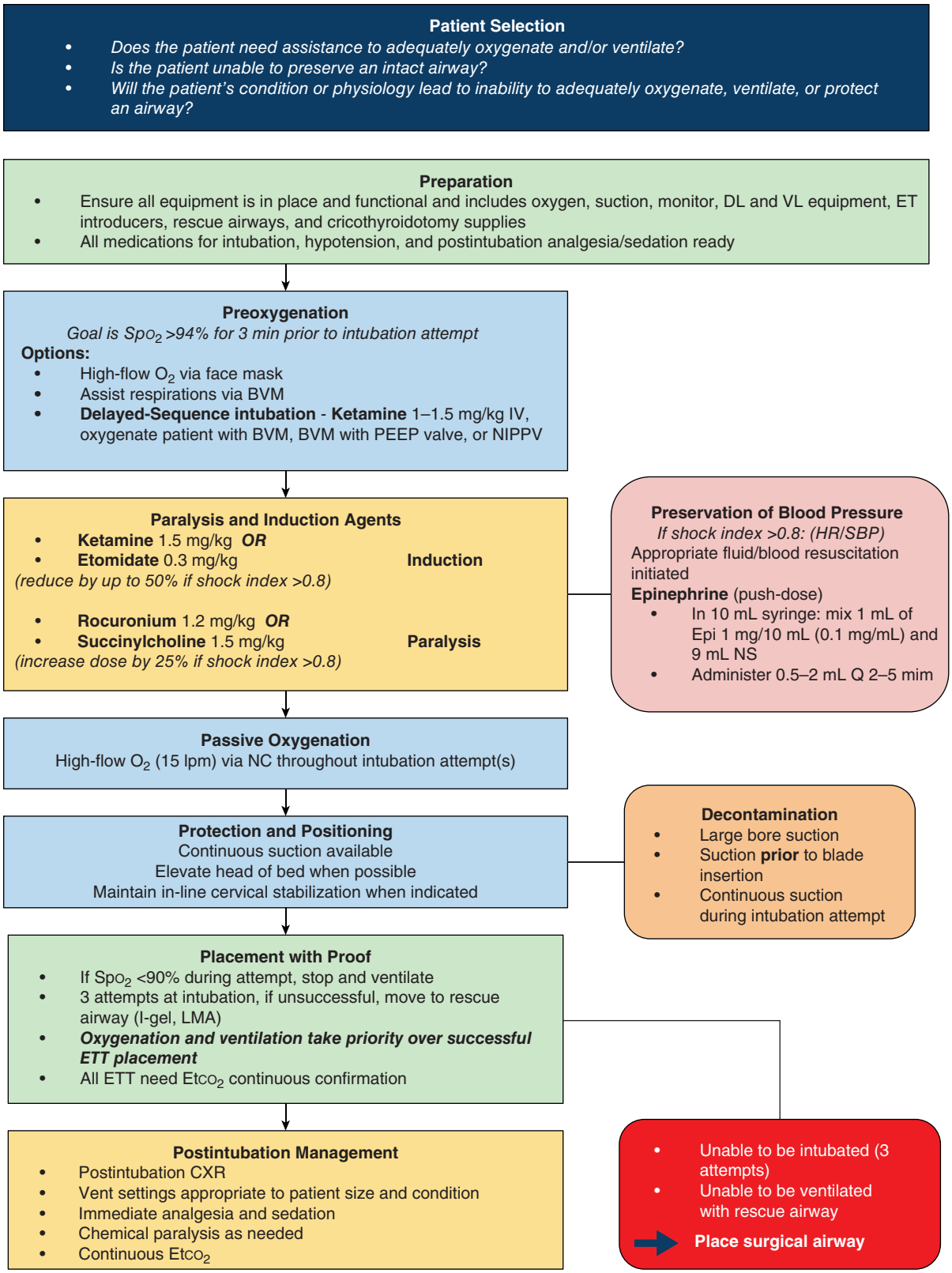

FIGURE 14-7 Airway trauma algorithm. BVM, bag-valve-mask; CXR, chest x-ray; DL, direct laryngoscopy; ET, endotracheal; ETT, endotracheal tube; HR, heart rate; LMA, laryngeal mask airway; NIPPV, noninvasive positive-pressure ventilation; PEEP, positive end-expiratory pressure; SBP, systolic blood pressure; VL, video laryngoscopy.

FIGURE 14-8 Direct airway trauma.

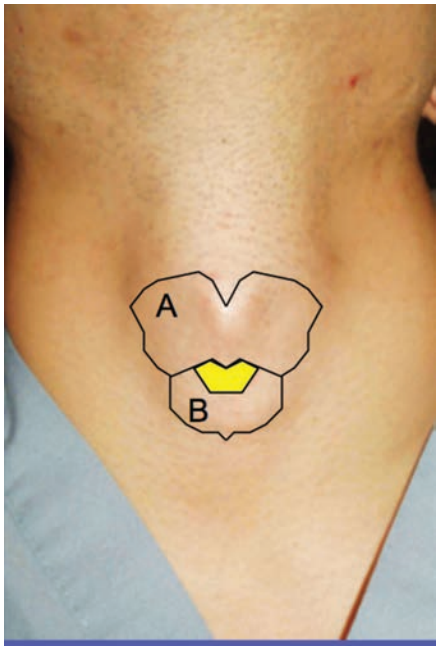

FIGURE 14-9 Cricothyroid anatomy, anterior view. (**A**) Thyroid cartilage. (**B**) Cricoid cartilage. Yellow = cricothyroid membrane.

SURGICAL AIRWAY

A surgical airway is the definitive rescue maneuver to manage patients with a difficult airway that cannot be secured by conventional techniques and results in a failed airway. Three options exist for a surgical airway: open cricothyroidotomy, Seldinger cricothyroidotomy, and needle cricothyroidotomy. The open and Seldinger cricothyroidotomy can be performed in an adult with a failed airway. Needle cricothyroidotomy is the rescue airway of choice in children because surgical cricothyroidotomy is contraindicated in children younger than 12 years old. In addition, a needle cricothyroidotomy may be performed in an adult if no equipment or expertise is available to perform an open or Seldinger cricothyroidotomy.

The key to any surgical airway is preprocedure preparation and a complete understanding of the relevant anatomy and surgical technique, as this uncommon procedure is typically performed in an emergent, time-sensitive manner. Anyone charged with managing a surgical airway for trauma patients should have thorough knowledge of the laryngeal anatomy and become familiar with the instruments available in their practice environment. Regardless of technique or instrumentation, the critical first step of performing a surgical airway is identification of the anatomic landmarks in the anterior neck. The cricothyroid membrane is located between the thyroid cartilage superiorly and the cricoid cartilage inferiorly (Fig. 14-9). It is more easily palpated in men due to a more prominent thyroid cartilage. Although fairly easy to identify in nonobese patients, the cricothyroid membrane can be obscured in trauma patients due to subcutaneous emphysema, hematoma, or direct laryngeal trauma.

Open cricothyroidotomy is performed with standard surgical instruments. Basic instruments required for an open cricothyroidotomy include a scalpel, hemostat, and either a smaller (size 6) tracheostomy or endotracheal tube. A trach hook can also be critically important, as this will provide stability to the tracheal cartilage during tube insertion. Although most surgical procedures require sterile technique, the lifesaving and emergent nature of a surgical airway does not allow time to perform a sterile procedure. Splashing iodine onto the neck prior to incision provides no anti-infective efficacy and merely makes the instruments wet and slippery, making an already challenging and stressful procedure even more difficult. The nondominant hand is used to grasp the laryngeal complex and the same index finger is used to identify the cricothyroid membrane. For this reason, a right-handed surgeon should stand on the patient's right side and a left-handed surgeon should stand on the patient's left side while performing an open cricothyroidotomy. Once the laryngeal complex has been stabilized and cricothyroid membrane identified, the nondominant hand should not be released until the airway is in place. With the dominant hand, the surgeon should make a vertical incision directly over the cricothyroid membrane. Although a horizontal skin incision may be used as well, a vertical incision avoids lacerating an anterior jugular vein and allows superior or inferior extension of the incision if necessary. The first incision should open all subcutaneous tissue between the skin and the cricothyroid membrane. This usually leads to bleeding, and the remainder of the procedure is typically performed with little or no visibility of anatomy. Instead, the next steps are guided by tactile identification of the cricothyroid membrane with the index finger of the nondominant hand. Next, a transverse incision is made in the cricothyroid membrane. If the cricothyroidotomy is not large enough to accept an airway, it may be dilated with the index finger, hemostat, or the end of a scalpel handle opposite the blade. Next, if available, a trach hook can be placed on the midline of the thyroid cartilage, and anterior-superior traction is applied to help prevent the anterior portion of the trachea from collapsing during tube insertion. If a

trach hook is not available, the nondominant hand is used to grasp the laryngeal complex to apply counterpressure. Finally, the tracheostomy or endotracheal tube is placed into the airway, the balloon on the tube is inflated, and confirmation of appropriate tube placement is performed, as discussed previously, and the tube is then sutured in place. If difficulty is encountered while placing the tracheal tube, a gum elastic bougie can be placed into the trachea and used in a Seldinger fashion to guide the tube into the appropriate location.

Seldinger cricothyroidotomy is performed with a prepackaged commercially available kit. As each kit is slightly different, clinicians should familiarize themselves with the kit used at their facility. Regardless of which commercial Seldinger kit is used, there are several basics steps common to all Seldinger cricothyroidotomies. Place the needle with overlying catheter onto a syringe with saline. While aspirating, puncture the cricothyroid membrane pointing the tip of the needle at a 45-degree angle toward the feet. Once bubbles are aspirated, advance the catheter over the needle and into the airway and remove the needle and syringe. Pass the guidewire through the catheter in the usual Seldinger manner and remove the catheter. Next, make a vertical skin incision around the wire, and advance the airway/dilator over the guidewire though the cricothyroid membrane and into the airway. Remove wire and dilator, inflate the cuff, and suture the airway in place.

The technique of needle cricothyroidotomy is the same as the first few steps of Seldinger cricothyroidotomy until the catheter is in the airway. For a needle cricothyroidotomy, once the catheter is in the airway, it is attached to a jet ventilation system using a Luer lock. Alternatively, a 5-mL syringe can be cut and attached directly to oxygen tubing, or the tubing can be wedged into the open end of the syringe and connected to high-flow 100% oxygen. An alternative method is to attach a 3-mL syringe to a 14-gauge catheter with an endotracheal tube adapter inserted into the open end of the syringe (Fig. 14-10). This technique allows direct attachment of the BVM to the syringe. Whatever system is used, an aperture should be created such that when occluded, jet insufflation occurs, and when open, flow may escape. Jet insufflation should proceed at approximately 1 second of flow (inspiration) for every 3 seconds of release (expiration). Because ventilation is not actually occurring, alveolar carbon dioxide rises. A point of emphasis is the temporizing nature of the procedure; life may be sustained for approximately 30 minutes while a definitive airway is established.

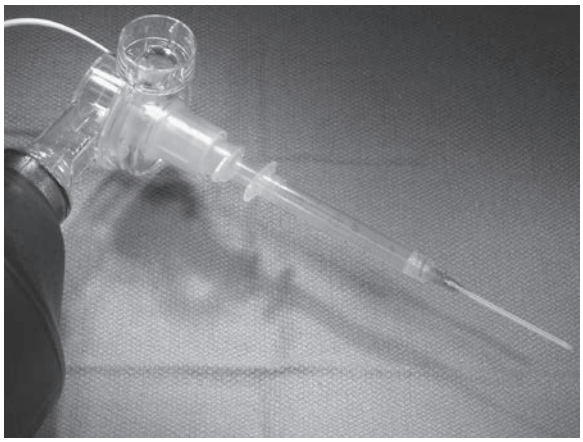

FIGURE 14-10 A photograph of a useful setup for needle cricothyroidotomy, consisting of a standard 8-mm diameter endotracheal tube adapter, a 3-cm^3 syringe, and a 14-gauge angiocatheter. The endotracheal tube adapter is removed and inserted into the open end of the 3-cm^3 syringe (plunger removed). The angiocatheter is affixed to the syringe. A bag valve mask is then able to be attached to the adapter, allowing easy ventilation of the patient.

PEDIATRIC AIRWAY MANAGEMENT

Children and infants have unique anatomic and physiologic features that may impact airway management and intubation. These need to be taken into consideration in the planning and management of pediatric intubations.[51-53]

Anatomy

PROMINENT OCCIPUT

The occiput in infants and younger children is proportionally larger, causing varying degrees of neck flexion in the supine position. This can result in anatomic airway obstruction or potentially interfere with attempts to visualize the glottic opening, particularly during DL. Placing a towel roll under the shoulders can improve airway alignment. This approach is in contrast to placing a pad under the occiput in adults.

LARGE TONGUE

Infants and young children also have larger tongues relative to the size of the oral cavity. Appropriate control and displacement of the tongue are vital to maintain adequate visualization of the larynx and trachea. In addition, the tongue becomes a common source of upper airway obstruction, particularly in patients with depressed level of consciousness and secondary loss of intrinsic airway tone.

LARGER TONSILS AND ADENOIDS

Children more commonly have larger tonsils and adenoids than adults. This may result in more difficulty in ventilating children by standard basic noninvasive measures. The tonsils are also more susceptible to injury and bleeding during intubation attempts.

SUPERIOR LARYNGEAL POSITION

The position of the larynx in infants and children is more superior, or cephalad, compared to adults. It is located opposite the C3 to C4 vertebrae, whereas an adult larynx is typically in line with C4 to C5. This creates a more acute angle between the glottic opening and the base of the tongue. This can make direct visualization more challenging and may favor the use of VL.

LARGER EPIGLOTTIS

The epiglottis is relatively large and floppy in children, particularly those younger than 3 years of age, and is angled more acutely with respect to the axis of the trachea. Subsequently,

more of the glottic opening can be obstructed. During DL, the use of a straight blade to directly lift the epiglottis can improve visualization.

SHORTER TRACHEA

The trachea length is approximately 5 cm in neonates and grows to about 12 cm in adults. Given the shorter segment within which an endotracheal tube can be correctly placed, there is a predisposition to right mainstem bronchus intubation and also inadvertent extubation.

NARROW TRACHEA

In addition to being shorter, the trachea in younger children is also narrower. Airway resistance is inversely proportional to the lumen radius to the fourth power, so even small decreases in the airway size from secretions or edema will have disproportionate effects on airflow. In addition, the space between tracheal rings is very narrow and the cricothyroid membrane is very small, making needle or surgical cricothyroidotomy technically challenging.

ANATOMIC SUBGLOTTIC NARROWING

The cricoid ring has been identified as the narrowest portion of the pediatric airway. Historically it was felt that this narrowing can create an effective anatomic seal without the need for a cuffed endotracheal tube and that the pressure associated with cuffed tubes may result in further subglottic stenosis. However, use of newer cuffed endotracheal tubes in children is associated with a reduced need for endotracheal tube exchanges and no increase in postextubation morbidity when compared to uncuffed tubes. Newer endotracheal tubes maintain a smaller profile and lower pressure cuffs, and the American Heart Association now has approved these for all pediatric patients outside the newborn period, provided the cuff pressure can be maintained at less than 20 cm H_2O.

COMPLIANT CHEST WALL

The thorax is more compliant in children due to the primarily cartilaginous nature of skeletal structure compared to the more rigid bony structures in adults. However, greater intrinsic muscle tone is required to maintain lung volumes and prevent chest wall distortion. Subsequently, infants and young children are more likely to experience respiratory muscle fatigue, atelectasis, and respiratory failure.

Physiology

Airway management can be affected by pediatric-specific physiologic factors as well.

AGE-RELATED RESPIRATORY RATE

Ranges for vital signs will vary by age, including heart rate, blood pressure, and respiratory rate. Variation in respiratory patterns may also occur, such as newborn periodic breathing. It is important to recognize significant variance from normal vital sign values in assessing disease and injury severity as well as response to therapy.

PREFERENTIAL NASAL BREATHING

A larger portion of infants are believed to be obligate nasal breathers. For this portion of children, the nares account for nearly half the total airway resistance. Therefore, obstruction by secretions, edema, or external compression can lead to significant increase in the work of breathing.

SMALLER TIDAL VOLUMES

Infants and young children have small, relatively fixed tidal volumes relative to body size (6–8 mL/kg). Subsequently, they are susceptible to iatrogenic barotrauma from aggressive positive-pressure ventilation. In addition, there is limited ability to increase minute ventilation with deeper breathing. Any compensatory response to physiologic demands for increased minute volume is dependent primarily on an increased respiratory rate. Children have limited ability to maintain a significant tachypnea, predisposing young infants in particular to respiratory failure.

LOWER FUNCTIONAL RESIDUAL CAPACITY

Due to a smaller functional residual capacity, young children have little intrapulmonary oxygen stores to use during hypoventilatory or apneic periods. In particular, apneic infants will have a precipitous decline in oxygen saturation. For this reason, children have a heightened need for preoxygenation and likely require bag-mask ventilation during RSI for intubation.

HIGHER OXYGEN METABOLISM

Infants have a higher metabolic rate and consume oxygen at a rate twice that of adults (6 vs 3 mL/kg/min). A higher oxygen consumption combined with a lower functional residual capacity in children results in a shorter safe apnea time. In a study of preoxygenated children with an American Society of Anesthesiologists physical status of class I (ie, healthy, no medical problems), the mean time to oxygen desaturation to 90% ranged from 1.5 minutes in children less than 6 months old to more than 6 minutes in children greater than 11 years of age.

HIGHER VAGAL TONE

Infants and young children may have a pronounced vagal response to laryngoscopy or airway suctioning. Because hypoxia potentiates the risk for bradycardia, efforts to maintain oxygenation before and during ETI should be maximized. The role of atropine has been discussed to mitigate this risk.

Equipment

It is imperative that providers have immediate access to a variety of sizes of airway interventional devices and equipment.

Both invasive and supportive airway equipment needs to be selected according to the size of the child, and selection criteria can be based on age, height, weight, or length. Access to rapid reference materials such as pediatric resuscitation cards, mobile phone applications, or length-based resuscitation systems (Broselow-Luten) is extremely valuable in determining appropriate-size equipment and reducing reliance on mathematical formulas.

It was a commonly held belief that children should be intubated with an uncuffed tube due to concerns for mucosal injury and secondary subglottic stenosis. However, recent studies have demonstrated safety with the newer high-volume, lower pressure cuffs. These are now recommended, particularly with patients at high risk for aspiration, burn injuries, or requiring higher airway pressures. Endotracheal tubes are measured according to their internal diameter in millimeters and vary in size from 2.5 mm to greater than 8 mm. Tube selection should be based on maximizing tube diameter to minimize air flow resistance, yet the tube should be small enough to fit through the patient's vocal cords comfortably. A commonly used formula to select the appropriate-size endotracheal tube (uncuffed) is **endotracheal tube size (mm) = 4 + (age in years)/4**. For cuffed tubes, the size should be decreased by 0.5 to 1.0 mm. If a cuffed endotracheal tube is used, current recommendations include the use of a manometer to measure cuff pressures, considering pressures in excess of 20 cm H_2O may be associated with mucosal ischemia.

A stylet should be used to facilitate passage of the endotracheal tube by providing additional rigidity. The largest size stylet the tube can accommodate should be used. Tubes greater than 5.5 mm will generally accept an adult-size stylet.

There is also some variety in laryngoscope blade sizing. Direct laryngoscope handles come in adult and pediatric sizes, and there are a variety of different shapes and sizes of laryngoscope blades. For DL, a straight (Miller) blade is usually recommended in order to directly lift the proportionally larger epiglottis out of the way. Blade sizes include size 0 for preterm and newborns, size 1 for infants, and size 2 for children greater than 2 years of age. Adult-size blades range from 3 to 5.

As in adults, alternative airways should be readily accessible and available in multiple sizes, including devices such as LMAs or I-gel airways. Sizing is dependent on the device and manufacturer and is typically based on patient's age, weight, or length.

Pediatric airway interventions will require the same monitoring equipment as adults including cardiac telemetry, noninvasive blood pressure monitoring, pulse oximetry, and capnography. Suction needs to be readily accessible, including access to both rigid and flexible suction catheters in multiple sizes. Oxygen supplementation should be provided and multiple size facemasks available. The appropriate-size mask should fit comfortably over the patient's mouth and nose. OPAs are sized by holding the device along the side of the patient's face with the external flanged end at the edge of the patient's mouth. The tip of the inserted portion should reach the angle of the mandible. NPAs should be selected based on a device that reaches from the nostril to the tragus of the ear.

Multiple video laryngoscope manufacturers also provide pediatric-sized equipment. The providers should prepare this equipment with the manufacturer-recommended size device based on patient age or size.

Technique

The intubation procedural steps are followed in the same format as those described for the adult procedure with some minor differences.[15,51,53] Preoxygenation is extremely important due to children's limited reserve. Their higher oxygen consumption rate and lower functional residual capacity will result in oxygen desaturation much more quickly than in adults. Pretreatment atropine is recommended for all children under age 1 year undergoing intubation and under 5 years of age receiving succinylcholine. Dosing is 0.02 mg/kg IV, with a minimum dose of 0.1 mg and maximum dose of 0.5 mg. Both etomidate and ketamine are considered acceptable agents for induction in pediatric trauma patients. Etomidate dosing is 0.3 mg/kg and ketamine 1.5 mg/kg. Rocuronium (1 mg/kg) and succinylcholine have been used extensively for chemical paralysis and are considered equally efficacious. Current recommended dosing for succinylcholine is 2 mg/kg for infants and 1 to 1.5 mg/kg for older children. In addition, providers should also be aware of the propensity for children to become bradycardic with succinylcholine. Tube placement can be significantly more challenging due to the anatomic variants mentioned. DL should employ the use of a straight (Miller) blade, particularly in infants, in order to lift the epiglottis and provide better cord visualization, whereas older children can be intubated with either a curved or straight blade. VL in children is promising due to some of the anatomical variants encountered; however, early evidence suggests thorough training with the device is necessary to achieve clinically significant success rates.

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15

Management of Shock

Barclay T. Stewart • Ronald V. Maier

KEY POINTS

- In shock, there is an imbalance between substrate delivery (supply) and substrate requirements (demand).
- In the neuroendocrine response to shock, the hypothalamus releases corticotrophin-releasing hormone, causing pituitary release of adrenocorticotropin hormone followed by adrenal cortex release of cortisol.
- Shock-activated neutrophils and their products may produce bystander cell injury and organ dysfunction.
- Because it is difficult to measure oxygen debt during resuscitation of trauma patients, surrogate parameters such as base deficit and serum lactate are measured.
- Crystalloid solutions pass relatively freely across the vascular endothelium and damaged endothelial glycocalyx layer in shock, and this can result in pronounced expansion of the extracellular fluid compartment.
- Lyophilized plasma is compatible with all blood types and can be stored at room temperature for up to 2 years, and its reconstitution requires less than 6 minutes.
- The principles of damage control resuscitation are permissive hypotension, restriction of crystalloid resuscitation, earlier blood and component resuscitation in appropriate ratios, and goal-directed correction of any coagulopathy.
- Exceptions to damage control resuscitation include elderly patients and those with traumatic brain injuries.
- Viscoelastic assays of coagulation such as thromboelastography and rotational thromboelastometry are now commonly used to correct any coagulopathies during resuscitation.
- Forms of shock include hypovolemic, neurogenic, cardiogenic, septic, obstructive, and traumatic.

A MODERN HISTORY OF SHOCK

In 1872, Dr. Samuel Gross, the American trauma surgeon immortalized in the Thomas Eakins painting, *The Gross Clinic*, described shock as a "rude unhinging of the machinery of life."[1] Although imprecise, this rendering of the shock state elegantly characterizes the manifestation of the physiologic derangements of decompensated shock. More precisely, shock is the inadequate delivery of oxygen to and removal of potentially toxic metabolites from tissues, which leads to cellular dysfunction and injury. Significant tissue hypoperfusion and cellular injury may occur despite normal systemic blood pressure; equating shock with hypotension and cardiovascular collapse is a vast oversimplification and results in delayed recognition of early shock, when intervention is most effective at preventing end-organ dysfunction. Therefore, understanding oxygen kinetics, the principle physiologic derangements that occur during shock, host responses to malperfusion, and ways to diagnose and reverse shock states can improve the outcome of injured and critically ill patients.

The initial cellular injury that occurs as a result of tissue malperfusion and dysoxia is reversible. However, cellular injury will become irreversible if tissue hypoperfusion is prolonged or severe enough such that compensation is no longer possible. Rapid recognition of the patient in shock and the prompt institution of steps to correct shock are critical skills for the trauma surgeon and team.

The management of the patient in shock has been integral to the surgeon's expertise for centuries. In 1854, the French physiologist Claude Bernard suggested that an organism attempts to maintain constancy in the internal environment despite external forces that attempt to disrupt the *milieu intérieur*[2]; this idea was later refined into the concept of homeostasis by Dr. Walter Bradford Cannon.[3] In the intact animal, the failure of physiologic systems to buffer the organism against these external forces and maintain homeostasis results in the *shock state*. Proof in point, serial analyses of the genomic response to severe injury identified that the patients able to most rapidly restore homeostasis have the best outcomes.[4]

In addition to the concept of homeostasis, Cannon described the "fight or flight response" generated by elevated levels of catecholamines in the bloodstream. After providing battlefield care for soldiers injured during World War I in the European theater, he published his classic monograph, *Traumatic Shock*, in 1923.[3] He proposed that shock was due to a disturbance of the nervous system that resulted in vasodilatation and hypotension and that secondary shock with its attendant capillary permeability was caused by a "toxic factor" released by injured tissues. His observations led to the early proposition of deliberate or permissive hypotension in patients with penetrating wounds of the torso to minimize intracavitary bleeding: "if the pressure is raised before the surgeon is ready to check the bleeding that may take place, blood that is sorely needed may be lost."[5]

Dr. Alfred Blalock described an association between shock after hemorrhage and reduced cardiac output and declared that hemorrhagic shock was the result of volume loss, not "toxic factors" alone.[6] The concept that hemorrhagic shock was due, in part, to "toxic factors" was abandoned and not considered again for several decades. In 1934, Blalock described four categories of shock: hypovolemic, vasogenic, neurogenic, and cardiogenic (Table 15-1).

- Hypovolemic shock, the most common type, results from loss of circulating blood or its components.
- Distributive or vasogenic shock, as seen in sepsis or anaphylaxis, results from decreased resistance to blood flow within capacitance vessels of the circulatory system, causing an effective functional decrease in circulating volume.
- Neurogenic shock is a form of vasogenic shock in which spinal cord injury (or spinal anesthesia) causes vasodilatation.
- Cardiogenic shock results from failure of the heart's pump function, as might occur with dysrhythmias or heart failure.

Two additional categories of shock have been added to those originally proposed by Blalock:

- Obstructive shock occurs when circulatory flow is mechanically impeded (eg, pericardial tamponade, tension pneumothorax, pulmonary embolism).

TABLE 15-1: Forms of Shock

Forms of shock	Examples
Hypovolemic	Hemorrhage, profound dehydration, pancreatitis
Neurogenic	Spinal cord injury, anesthesia
Cardiogenic	Dysrhythmia, myocardial infarction, heart failure, valve rupture
Distributive	Septic shock, anaphylaxis
Obstructive	Tension pneumothorax, pericardial tamponade, pulmonary embolism
Traumatic	Hemorrhage, tissue damage and activation of proinflammatory cascades

- Traumatic shock is often seen in multisystem trauma with significant soft tissue and bony injuries with or without significant hemorrhage and occurs secondary to the release of "toxins" from injured tissues that are normally excluded molecules, termed damage-associated molecular patterns (DAMPs) or "danger signals," and secondary upregulation of proinflammatory mediators that can create a state of shock that is much more complex than simple hemorrhagic shock.[7] The discovery and role of DAMPs and proinflammatory mediators in traumatic shock confirmed Cannon's early observations.

In addition to seminal observations on the clinical syndrome of shock on the battlefield, the early and mid-20th century witnessed important basic scientific contributions to the understanding of shock. In 1947, Dr. Carl Wiggers developed a model of graded hemorrhagic shock based on the uptake of shed blood into a reservoir to maintain a prescribed level of hypotension.[8] Dr. G. Tom Shires and coworkers performed a series of classical laboratory studies in the 1960s and 1970s that demonstrated that a large extracellular fluid (ECF) deficit occurred in severe hemorrhagic shock that was greater than could be attributed to vascular refilling alone.[9] A triple isotope technique in dogs revealed that this ECF deficit persisted when shed blood or shed blood plus plasma was used in resuscitation. Only the infusion of both shed blood and lactated Ringer's solution (an ECF mimic) repleted the red blood cell mass, plasma volume, and ECF.[10] Differences in mortality after hemorrhage and resuscitation dramatically illustrated the importance of this observation: The mortality rate after resuscitation with blood alone is 80%; with blood plus plasma, 70%; and with blood plus lactated Ringer's solution, 30%. The existence of this ECF deficit was subsequently confirmed in human patients.

Additional studies by Shires' group demonstrated significant dysfunction of the cellular membrane in prolonged hemorrhagic shock.[11] Depolarization of the cell membrane resulted in an uncontrolled uptake of water and sodium by the cell and loss of potassium in association with the loss of membrane integrity. The depolarization of the cell membrane was proportional to the degree and duration of hypotension. Ultimately, the study of red blood cells, hepatocytes, and skeletal muscle during hemorrhagic shock suggested that an abnormality in membrane active transport (Na^+/K^+-ATPase pump) was the basis of the cellular membrane dysfunction. In addition, the uptake of fluid by the intracellular compartment was a major site of fluid sequestration following prolonged hemorrhagic shock. These changes were reversible with appropriate resuscitation, further supporting the importance of prompt fluid resuscitation of severe hemorrhagic shock with balanced salt solution in addition to red blood cells.

With advances in our understanding of the pathophysiology and treatment of shock, new clinical problems soon became apparent. The Vietnam War provided a lurid clinical laboratory for shock research. Aggressive fluid resuscitation with red blood cells, plasma, and crystalloid solutions provided a survival advantage for the war-wounded who previously would have

succumbed to the effects of hemorrhagic shock, including a high rate of acute renal failure (ARF). More aggressive resuscitation made ARF a less frequent problem but, in its place, fulminant pulmonary failure appeared as an early cause of death after severe hemorrhage. Initially labeled "shock lung" or "Đà Nẵng lung," the clinical problem soon became characterized as acute respiratory distress syndrome (ARDS).[12] Resuscitation with excessive volumes of crystalloid solution was initially proposed as the primary mechanism of ARDS. However, ARDS was eventually recognized to be a component of the multiple organ dysfunction syndrome (MODS) or multiple organ failure syndrome (MOF), a result of complex upregulation of proinflammatory mediators greatly compounded by excessive volume resuscitation. The concepts of ARDS and MODS/MOF will be discussed in subsequent chapters (see Chapters 59 and 63, respectively).

Stemming from additional research during the Vietnam War, the role of the endothelium in shock has been clarified. For the better part of the 20th century, it had been assumed that fluid physiology, volume resuscitation, and clinical edema formation were governed solely by Starling's principle of microvascular fluid exchange.[13] Starling's principle states that capillary hydrostatic pressure and interstitial protein oncotic pressure drives transendothelial filtration, and a counteracting absorptive force is exerted by plasma protein oncotic pressure and interstitial hydrostatic pressure. However, fluid resuscitation does not result in the ECF expansion expected based on Starling's model's assumptions and is affected by the endothelial glycocalyx layer (EGL).[14] The EGL is a web of membrane-bound glycoproteins and proteoglycans on the luminal side of endothelial cells that regulate the passage of fluid, electrolytes, and proteins (eg, albumin, immunoglobulins).[15] When the EGL is damaged, fluid, electrolytes, and proteins are able to pass into the interstitial fluid space more easily, resulting in interstitial edema and fluid sequestration.[14] Disruption of the EGL, termed *compaction* or *shedding*, results in an increase in the plasma concentrations of the glycosaminoglycans that compose EGL. Thus, high plasma concentrations of these molecules are thought to represent EGL injury, which occurs during rapid fluid infusion, sepsis, shock, and surgery, and after injury.[16]

Several decades of research using the principles of the Wiggers models of hemorrhagic shock emphasized the importance of early control of hemorrhage in conjunction with restoration of intravascular volume with red blood cells and crystalloid solutions. Studies have extended the observations initially made by Cannon in 1918 on the futility of vigorously resuscitating patients with ongoing bleeding and have challenged traditional thinking on the appropriate end points of resuscitation from uncontrolled hemorrhage. The concepts of delayed fluid resuscitation and limited or controlled resuscitation are now advocated.[17]

Several essential concepts in the management of shock in the trauma patient have withstood the tests of time: (1) early definitive control of the airway should be achieved prior to circulatory collapse; (2) delay in hemorrhage control increases mortality; (3) inadequate resuscitation results in avoidable deaths and disabilities; and (4) excessive or uncontrolled fluid resuscitation is harmful.[18]

PATHOPHYSIOLOGY

Pathophysiology of Shock

Shock exists when the delivery of oxygen and metabolic substrates to tissues and cells and removal of metabolites are insufficient to maintain normal aerobic metabolism. This concept of mal- or hypoperfusion highlights the imbalance between substrate delivery (supply) and substrate requirements (demand) that occurs at the cellular level in shock states. Tissue hypoperfusion activates a cascade of cardiovascular and neuroendocrine responses designed to compensate for inadequate oxygen delivery and metabolite removal. The pathophysiologic sequelae of shock may be due to the direct effects of inadequate tissue perfusion on cellular and tissue function and/or the host's overzealous responses to the shock state.

The magnitude of both the shock insult and response varies depending on the degree and duration of shock. The consequences of shock may also vary from minimal physiologic disturbance with complete recovery to profound circulatory disturbance, end-organ dysfunction, and death. Accumulating evidence suggests that, although the quantitative nature of the host response to shock may differ between the various etiologies of shock, the qualitative nature of the host response to shock is similar regardless of the cause of the insult. This response consists of profound changes in cardiovascular, neuroendocrine, and immunologic functions. Furthermore, these responses vary with host genetics, time, and response to resuscitation. For example, in hemorrhagic shock, the initial compensation for blood loss occurs primarily through neuroendocrine responses to maintain tissue perfusion. This represents the compensated phase of shock (Fig. 15-1). With ongoing hypoperfusion, cellular injury worsens and the decompensated phase

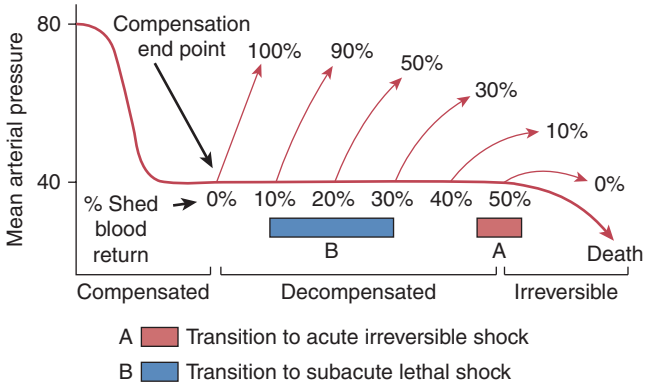

FIGURE 15-1 A rodent model of hemorrhagic shock depicting the relation between volume loss, duration of shock (represented by length of time in compensated, decompensated, and irreversible shock), and transition from reversible to irreversible shock. The percentages shown above the curve represent survival rates. (Reproduced with permission from Peitzman AB, Harbrecht BG, Udekwu AO, et al. Hemorrhagic shock. *Curr Probl Surg*. 1995;32:974, Copyright © Elsevier.)

of shock ensues. Microcirculatory dysfunction, cellular injury and death, and inflammatory cells activation perpetuate the hypoperfusion and may exacerbate tissue injury. If resuscitation is achieved, ischemia/reperfusion injury may further exacerbate the initial insult. Persistent hypoperfusion results in further physiologic and hemodynamic derangements and cardiovascular collapse, which has been termed the irreversible phase of shock. At this phase, extensive parenchymal and microvascular injury has occurred such that further volume resuscitation fails to reverse the process, leading to death of the patient.

Afferent Signals

Afferent impulses transmitted from tissue beds are processed within the central nervous system (CNS) and activate reflexive effector responses or efferent impulses designed to expand plasma volume, maintain peripheral perfusion and tissue oxygen delivery, and reestablish homeostasis (Fig. 15-2). The afferent impulses that initiate the body's intrinsic adaptive responses converge in the CNS and originate from a variety of sources.

The initial inciting event is often loss of actual or relative circulating blood volume. Other stimuli that can produce afferent impulses include tissue trauma, pain, hypoxemia, hypercarbia, acidosis, infection, change in temperature, emotional arousal, or hypoglycemia. As an example, the sensation of pain from injured tissue is transmitted via spinothalamic tracts and activates the hypothalamic–pituitary–adrenal axis. The sensation of pain can also activate the autonomic nervous system (ANS) and increase direct sympathetic stimulation of the adrenal medulla to release catecholamines.

Baroreceptors represent an important afferent pathway in initiating adaptive or corrective responses to shock.[19] Volume receptors within the atria of the heart are sensitive to changes in both chamber pressure and wall stretch. These receptors become activated with low-volume hemorrhage or mild reductions in right atrial pressure. Receptors in the aortic arch and carotid bodies respond to alterations in pressure or stretch of the arterial wall and respond to greater reductions in intravascular volume or changes in pressure. These receptors normally inhibit activation of the ANS. When these baroreceptors are activated, their output is diminished, resulting in greater ANS output principally via sympathetic activation at the vasomotor centers of the brainstem; this produces centrally mediated constriction of peripheral vessels that attempts to maintain blood pressure and tissue perfusion.

Chemoreceptors in the aorta and carotid bodies are sensitive to changes in oxygen tension, H^+ ion concentration, and carbon dioxide level.[20] These receptors also provide afferent

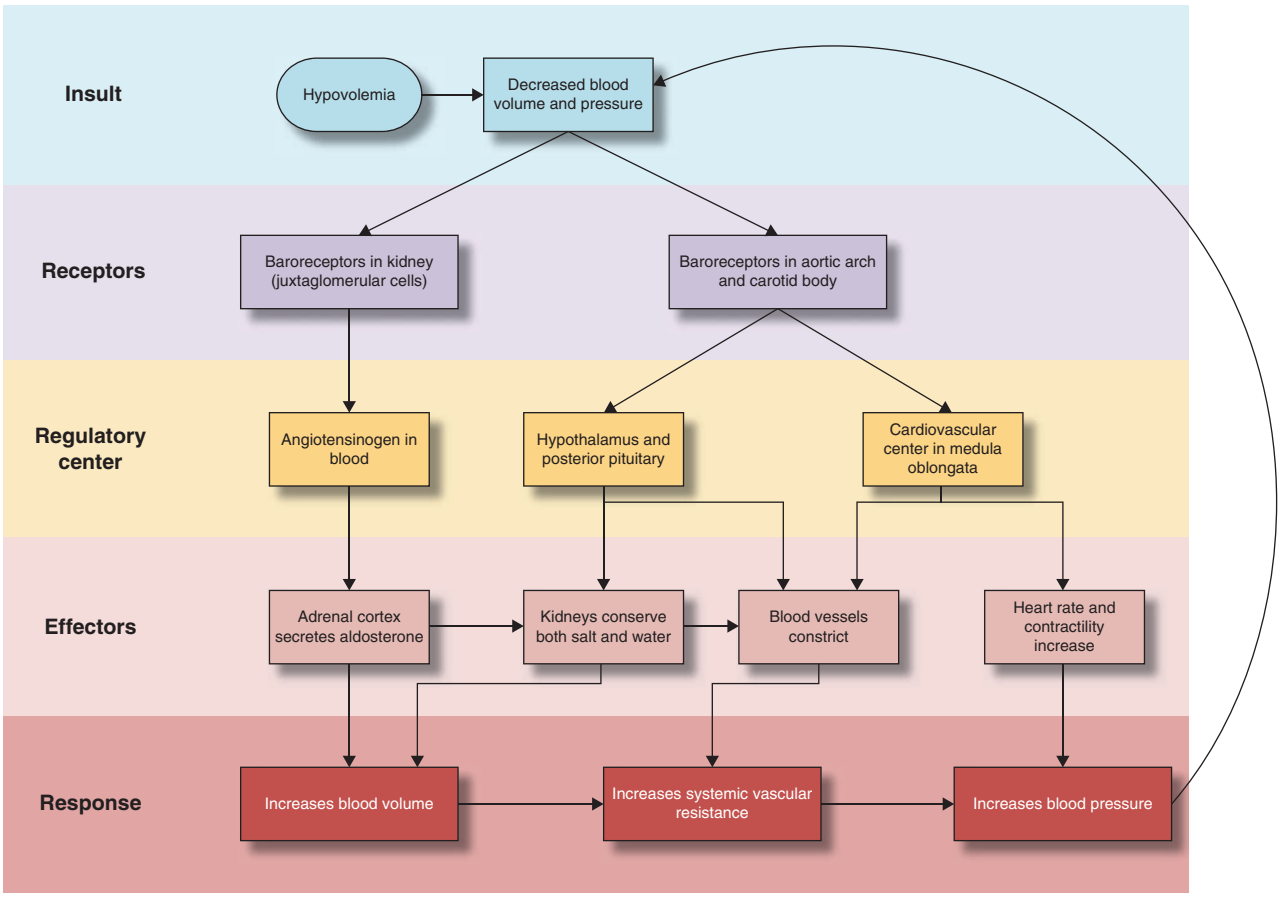

FIGURE 15-2 Afferent signals and efferent responses in hypovolemia and hemorrhagic shock.

stimulation when the circulatory system is disturbed and activate effector response mechanisms. In addition, a variety of protein and nonprotein mediators produced at the site of injury and inflammation act as afferent impulses and induce both central and peripheral responses to trauma and shock. Some of these compounds are components of the host immunologic response to shock and include histamine, cytokines, eicosanoids, endothelins, and others that will be discussed in greater detail in this and subsequent chapters.

Efferent Signals

CARDIOVASCULAR RESPONSE

The neuroendocrine and ANS responses to shock result in changes in cardiovascular physiology, which constitute a prominent feature in the host's adaptive reactions and clinical presentation of the patient in shock (see Fig. 15-2). Stimulation of sympathetic fibers innervating the heart activates β_1-adrenergic receptors that increase both heart rate (chronotropy) and contractility (inotropy) in an attempt to increase cardiac output. However, this response carries a cost: increased myocardial oxygen consumption occurs as a result of the increased workload. Adequate myocardial oxygen supply must be achieved and maintained or myocardial ischemia and dysfunction will develop and exacerbate the shock state.

Direct sympathetic stimulation of the peripheral circulation via activation of α_1-adrenergic receptors on arterioles increases vasoconstriction and causes a compensatory increase in systemic vascular resistance and blood pressure. Selective perfusion of tissues due to regional variations in arteriolar resistance from these compensatory mechanisms occurs in shock. The majority of afterload increase to maintain mean arterial pressure (MAP) is provided by splanchnic vasoconstriction. Blood is shunted away from organs such as the intestine, kidney, liver, and skin that are less essential to the host's immediate needs during shock.[21] Organs such as the brain and heart have autoregulatory mechanisms that attempt to preserve their blood flow despite a global decrease in cardiac output. Direct sympathetic stimulation also induces constriction of veins, akin to Trendelenburg positioning, which decreases the capacitance of the circulatory system and augments blood return to the central circulation.

Increased sympathetic output stimulates catecholamine release from the adrenal medulla. Catecholamine levels increase and peak within 24 to 48 hours of injury before returning to baseline; persistence of elevation in catecholamine levels is consistent with ongoing hypoperfusion and poorer prognosis after injury.[22] Most of the epinephrine that circulates systemically is produced by the adrenal medulla, while norepinephrine is also derived from synapses of the sympathetic nervous system. Catecholamines also have profound effects on peripheral tissues in ways that support the host's ability to respond to shock and hypovolemia. For example, catecholamines stimulate hepatic glycogenolysis and gluconeogenesis to increase the availability of circulating glucose to peripheral tissues, increase glycogenolysis in skeletal muscle, suppress the release of insulin, and trigger the release of glucagon. The genomic response is one of decreased intracellular signaling in response to insulin and decreased expression of cellular membrane proteins required for cellular uptake of glucose.[23] Together, these responses increase the availability of glucose to tissues that require it for maintenance of essential metabolic activity. In addition, hyperglycemia functions as an endogenous osmotic load to further retain and increase intravascular volume.

NEUROENDOCRINE RESPONSE

As earlier described, a variety of afferent stimuli lead to activation of the hypothalamic–pituitary–adrenal axis, an integral component of the adaptive response of the host following shock (see Fig. 15-2). Shock stimulates the hypothalamus to release corticotrophin-releasing hormone, which results in the release of adrenocorticotropin hormone (ACTH) by the pituitary. ACTH subsequently stimulates the adrenal cortex to release cortisol. Cortisol acts synergistically with epinephrine and glucagon to induce a catabolic state.[24] Cortisol stimulates gluconeogenesis and insulin resistance, resulting in hyperglycemia; it also induces protein breakdown in muscle cells and lipolysis, which provide substrates for hepatic gluconeogenesis. Cortisol causes retention of sodium and water by the kidney that aids in restoration of circulating volume. In the setting of severe hypovolemia, ACTH secretion occurs independently of negative feedback inhibition by cortisol. Absence of appropriate cortisol secretion during critical illness or after injury has been postulated as a contributor of ongoing circulatory instability in critically ill patients.[25] In addition, absence of immune cell glucocorticoid receptors prevents the normal downregulation of the immunoinflammatory response to stress and greatly increases mortality, particularly after septic shock.[26]

The pituitary also releases vasopressin or antidiuretic hormone (ADH) in response to hypovolemia, changes in circulating blood volume sensed by baroreceptors and stretch receptors in the left atrium, and increased plasma osmolality detected by hypothalamic osmoreceptors. Epinephrine, angiotensin II, pain, and hyperglycemia further stimulate the production and release of ADH. ADH levels remain elevated for about 1 week after the initial insult, depending on the severity and persistence of the hemodynamic perturbation. ADH acts on the distal tubule and collecting duct of the nephron to increase water permeability and sodium reabsorption, thereby decreasing loss of water and preserving intravascular volume. Vasopressin acts as a potent mesenteric vasoconstrictor, shunting circulating blood away from the splanchnic organs during hypovolemia. The intense mesenteric vasoconstriction produced by vasopressin may contribute to intestinal ischemia and predispose to dysfunction of the intestinal mucosal barrier in shock states.[27] Vasopressin also regulates hepatocellular function in concert with epinephrine and cortisol by increasing hepatic gluconeogenesis and hepatic glycolysis.

The renin–angiotensin system is likewise activated in shock states. Decreased perfusion of the renal artery, β-adrenergic stimulation, and increased sodium concentration in the renal

tubules cause the release of renin from the juxtaglomerular cells. Renin catalyzes the conversion of angiotensinogen (produced by the liver) to angiotensin I, which is then converted to angiotensin II by angiotensin-converting enzyme (ACE; produced in the lung). Although angiotensin I has no significant functional activity, angiotensin II is a potent vasoconstrictor of both splanchnic and peripheral vascular beds and stimulates the secretion of aldosterone, ACTH, and ADH. Aldosterone, a mineralocorticoid, acts on the nephron to promote sodium and water resorption in exchange for potassium and hydrogen ions that are lost in the urine. In the short run, oliguria in the patient with hypovolemic shock is acute renal success, not ARF. However, prolonged renal hypoperfusion results in depletion of renal ATP stores with subsequent acute renal injury, renal failure, and at times, paradoxical urine production despite systemic hypovolemia.

IMMUNOLOGIC AND INFLAMMATORY RESPONSE

Inflammatory and innate immune responses are a complex set of interactions between circulating soluble factors and cells that respond to injury, infection, ischemia, and toxic or autoimmune stimuli.[28] The function of the host's immune system after shock is intimately related to alterations in the production of mediators generally considered part of the body's innate response to localized inflammation, tissue injury, and infection (Fig. 15-3). However, when these mediators gain access to the systemic circulation, they induce changes in a number of tissues and organs. Although proinflammatory activation is a central feature of septic shock, proinflammatory cytokine production and mediator release also occur in other forms of shock, particularly traumatic shock, during which DAMPs and other endogenous stimulatory molecules are released from damaged tissue.[29] Severe trauma and shock have been shown

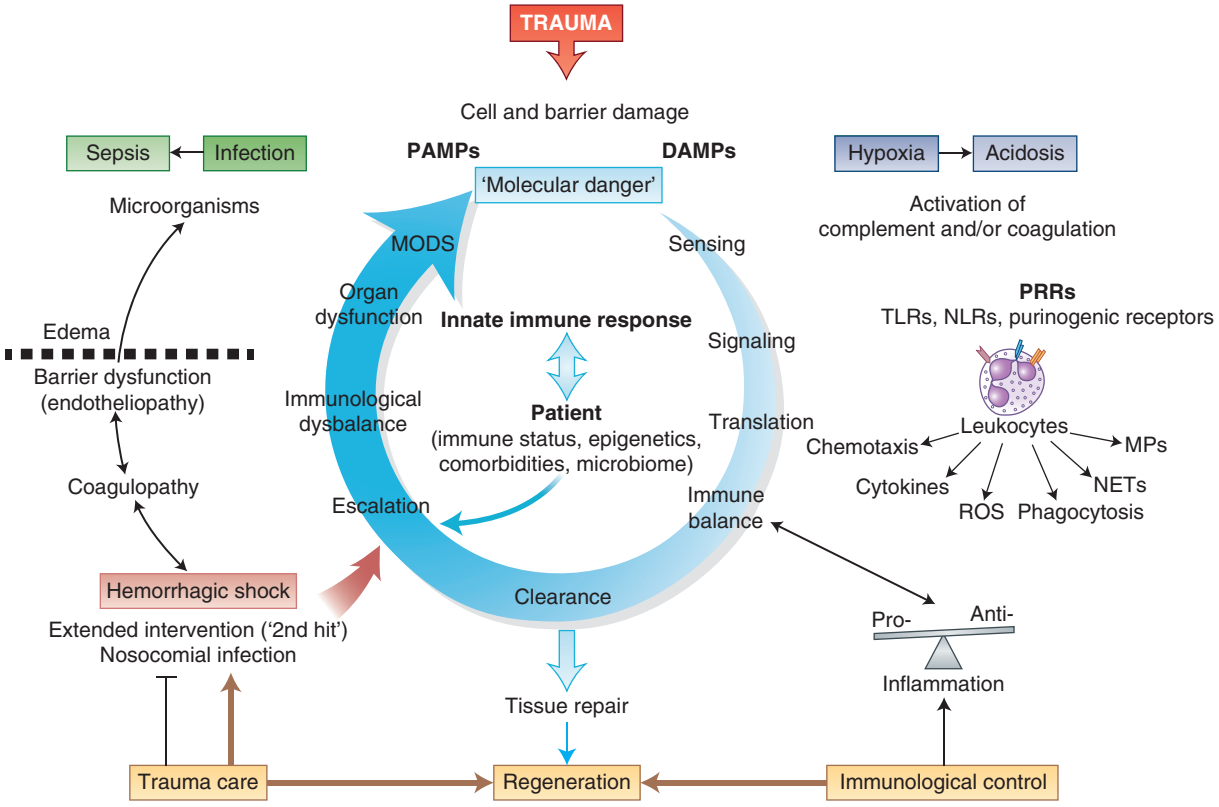

FIGURE 15-3 Innate immune responses to trauma. Trauma leads to the damage of external and internal barriers and thus exposes the immune system to damage-associated molecular patterns (DAMPs) and pathogen-associated molecular patterns (PAMPs). Molecular danger signals and the destruction of local barriers are sensed by the complement and the coagulation systems and induce intracellular signaling in leukocytes via pattern recognition receptors (PRRs), which leads to translation into an instantaneous cellular immune response. Ideally, a balanced proinflammatory and anti-inflammatory reaction leads to rapid clearance of debris and the induction of effective tissue repair and regeneration; adverse events can be caused by individual factors of the patient or aggravated tissue damage after hemorrhage, nosocomial infection, or extended surgical intervention. Escalation of the innate immune response in the form of coagulopathy and excessive inflammation leads to barrier disturbance, edema formation, and compromised innate defense against invading microorganisms. Such changes can aggravate hypoxic conditions, the accumulation of metabolites, and bacterial invasion, all of which can "feed in" more DAMPs and PAMPs and thus generate a vicious cycle of the innate immune response. This eventually results in organ dysfunction and systemic infection, which emphasizes the importance of damage-adjusted trauma care principles as well as control of the balance of the immune system, particularly in the acute phase after injury. MODS, multiorgan dysfunction syndrome; MPs, microparticles; NETs, neutrophil extracellular traps; NLRs, NOD-like receptors; ROS, reactive oxygen species; TLR, toll-like receptor. (Reproduced with permission from Huber-Lang M, Lambris JD, Ward PA. Innate immune responses to trauma. *Nat Immunol*. 2018;19:327-341. Copyright © Springer.)

to alter the gene response in up to 80% of the entire human genome, leading to a total body "genomic storm."[4] Thus, as initially proposed by Cannon in the early part of the 20th century, inflammatory "toxic" mediators can be a cause of shock as well as a by-product of the host response to shock.

Most mediators have a variety of effects due to the redundant and overlapping nature of the host response to injury. Therefore, in addition to regulating immune function in the host, many of these mediators have effects on the cardiovascular system, coagulation system, cellular metabolism, and cellular gene expression. Many compounds already discussed have substantial effects on the cardiovascular and/or neuroendocrine responses to shock. As an example, catecholamines have effects on immune function and activate proinflammatory cytokines.[30] Cytokines are small polypeptides and glycoproteins that exert most of their actions in a paracrine fashion and are responsible for fever, leukocytosis, tachycardia, tachypnea, and the upregulation of other cytokines. Their levels are elevated in hemorrhagic, septic, and traumatic shock.[28] The overexpression of certain cytokines is associated with the metabolic and hemodynamic derangements often seen in septic shock or decompensated hypovolemic shock, and certain patterns of cytokine production after shock correlate with the development of MODS.[29] The immune response to injury and infection is discussed in greater detail in Chapters 21 and 63. A brief review of several of the key components of the immune response is provided in the following paragraphs.

Tumor necrosis factor-α (TNF-α) is one of the earliest proinflammatory cytokines released by monocytes, macrophages, and T cells in response to injurious stimuli.[31] The classic model of TNF-α production is the injection of bacterial endotoxin in an animal subject. Under these controlled conditions, TNF-α levels peak within 90 minutes of the insult and return to baseline within 4 hours. Endotoxin stimulates TNF-α release and may be a primary inducer of cytokines, as in the case of septic shock. TNF-α release may also be a secondary event following the release of DAMPs and other mediators from injured tissue.[32] TNF-α levels are increased after hemorrhagic shock, and TNF-α levels correlate with mortality in animal models of hemorrhage.[33,34] In humans, TNF-α, interleukin-6 (IL-6), and IL-8 levels increase during hemorrhagic shock, although the magnitude of the increase is less than that seen in septic patients.[35] Once released, TNF-α can cause peripheral vasodilation, activate the release of other cytokines such as IL-1β and IL-6, induce procoagulant activity and increased consumption of coagulation factors, and stimulate a wide array of cellular metabolic changes. TNF-α enhances mechanisms of host defense against infection by promoting activation of macrophages and neutrophil intracellular killing of pathogens. During the stress response, TNF-α contributes to the breakdown of muscle protein and cachexia. Despite being linked to tissue injury and dysfunction, TNF-α appears to be essential in combating bacterial infection since neutralizing TNF-α in live bacterial peritonitis and pneumonia models increases mortality.[36,37]

IL-1β has actions that are similar to TNF-α and can cause hemodynamic instability and vasodilation.[31] It has a very short half-life (6 minutes) and primarily acts in a paracrine fashion. IL-1β produces a febrile response by activating prostaglandins in the posterior hypothalamus and causes anorexia by activating the satiety center. This cytokine also augments the secretion of ACTH, glucocorticoids, and β-endorphins. In conjunction with TNF-α, IL-1β can induce the release of other cytokines such as IL-2, IL-4, IL-6, IL-8, granulocyte-macrophage colony-stimulating factor (GM-CSF), and interferon-γ (IFN-γ). IL-2 expression is important for the cell-mediated immune response, and its attenuated expression has been associated with transient immunosuppression in injured patients. IL-6 has consistently been shown to be elevated in animals subjected to hemorrhagic shock or trauma and in patients after major surgery. Elevated IL-6 levels correlate with mortality in some forms of shock.[38] IL-6 contributes to neutrophil-mediated injury to the lung after hemorrhagic shock and may play a role in the development of diffuse alveolar damage and ARDS.[39] IL-6 and IL-1β are mediators of the hepatic acute phase response to injury and enhance the expression and/or activity of complement, C-reactive protein, fibrinogen, haptoglobin, amyloid A, and α_1-antitrypsin. Activation of neutrophils is promoted by IL-6, IL-8, and GM-CSF, and IL-8 also serves as a potent chemoattractant to neutrophils.

The complement cascade is activated by injury and shock and contributes to proinflammatory activation in both animal models and human patients. The degree of complement activation is proportional to the magnitude of the traumatic injury and may serve as a marker for severity of injury in trauma patients. Patients in septic shock also demonstrate activation of the complement pathway with elevation of the activated complement proteins C3a and C5a.[40] Complement activation can occur after hemorrhagic shock and may contribute to the hypotension, coagulopathy, metabolic acidosis, and MODS observed following shock and resuscitation.[41] The development of coagulopathy, ARDS, and MODS in trauma patients correlates with the intensity of complement activation.[29,42]

Activation of neutrophils is one of the early changes induced by the inflammatory response, and neutrophils are the first cells to be recruited to sites of injury and inflammation. These cells are important in the clearance of infectious agents, foreign substances that have penetrated host barrier defenses, and toxin-generating nonviable tissue. However, activated neutrophils and their products may also produce bystander cell injury and organ dysfunction. Activated neutrophils generate and release a number of substances such as reactive oxygen species, lipid peroxidation compounds, proteolytic enzymes (eg, elastase, cathepsin G), vasoactive mediators (eg, leukotrienes, eicosanoids), and platelet-activating factor (PAF); the latter leads to diffuse and nondiscriminatory activation, adherence, and clearance of platelets from the circulation that results in microcirculatory thrombosis and regional hypoxia. Oxygen radicals such as superoxide anion, hydrogen peroxide, and the hydroxyl radical are potent inflammatory molecules that activate peroxidation of lipids, inactivate cellular enzymes, and consume cellular

antioxidants (eg, glutathione and tocopherol). Intestinal ischemia and reperfusion cause activation of neutrophils and induce neutrophil-mediated organ injury in experimental animal models.[43] In both animal models of hemorrhagic shock and human patients with septic shock, activation of neutrophils correlates with irreversibility of shock and mortality, and neutrophil depletion prevents many of the pathophysiologic sequelae of hemorrhagic and septic shock.[44,45] In humans, activation of neutrophils in trauma and shock also plays a role in the development of MODS after injury.[46] Plasma markers of neutrophil activation such as elastase and other enzymes correspond to phagocytic activity and correlate with severity of injury.[29]

Interactions between endothelial cells, leukocytes, and platelets are important in host defense and the initiation and perpetuation of the inflammatory response in the host. The vascular endothelium regulates blood flow, adherence of leukocytes, and activation of the coagulation cascade. Adhesion molecules such as intercellular adhesion molecules (ICAMs), vascular cell adhesion molecules (VCAMs), and the selectins (E-selectin, P-selectin) are expressed on the surface of endothelial cells and are responsible for the adhesion of leukocytes and platelets to the endothelium. The interaction of surface proteins on leukocytes, platelets, and vascular endothelial cells allows activated neutrophils to marginate into the tissues in order to engulf invading organisms. However, the migration of activated neutrophils into tissues and adherence of platelets also lead to neutrophil-mediated cytotoxicity, microvascular damage, and tissue injury; this tissue damage contributes to microvascular thrombosis, coagulopathy, and organ dysfunction after shock.[47] It is important to note that the activation of platelets can lead to opposing hypo- and hypercoagulopathic states, further compounding the care of patients in shock.

Cellular Effects

Depending on the magnitude of the insult and the intrinsic compensatory mechanisms present in different cells, the response at the cellular level may be one of adaptation, dysfunction and injury, or death. The aerobic respiration of the cell (ie, oxidative phosphorylation by mitochondria) is the pathway most susceptible to inadequate oxygen delivery and toxic oxygen radicals. As oxygen tension within cells decreases, oxidative phosphorylation decreases and the generation of adenosine triphosphate (ATP) slows or stops. The loss of ATP, the cellular "energy currency," has widespread effects on cellular function, physiology, and morphology. As oxidative phosphorylation slows, the cells shift to anaerobic glycolysis that generates ATP from the rapid breakdown of cellular glycogen. However, anaerobic glycolysis is much less efficient than oxygen-dependent mitochondrial pathways. Under aerobic conditions, pyruvate, the end product of glycolysis, is fed into the Krebs cycle for further oxidative metabolism. Under hypoxic conditions, the mitochondrial pathways of oxidative catabolism are impaired and pyruvate is instead converted to lactate. The accumulation of lactic acid and inorganic phosphates is accompanied by a reduction in pH, resulting in intracellular metabolic acidosis. As cells become hypoxic and ATP depleted, other ATP-dependent cell processes are affected: synthesis of enzymes and structural proteins, repair of deoxyribonucleic acid (DNA) damage, and intracellular signal transduction. Tissue hypoperfusion also results in decreased availability of metabolic substrates and the accumulation of metabolic by-products such as oxygen radicals and organic ions that may be toxic to cells.

The consequences of intracellular acidosis on cell function can be quite profound. Decreased intracellular pH can alter the activity of cellular enzymes, lead to changes in cellular gene expression, impair cellular metabolic pathways, and interfere with ion exchange in the cell membrane.[48] Acidosis can also lead to changes in cellular calcium metabolism and calcium-mediated cellular signaling that can interfere with the activity of specific enzymes and alter cell function. These changes in normal cell function can produce cellular injury or cell death.[49] Changes in both cardiovascular and immune function in the host can be induced by intracellular acidosis.[50,51]

As cellular ATP is depleted under hypoxic conditions, the activity of the membrane Na^+/K^+-ATPase slows and thus the regulation of cellular membrane potential and volume is impaired. Na^+ accumulates intracellularly, whereas K^+ leaks into the extracellular space. The net gain of intracellular sodium is accompanied by an increase in intracellular water and the development of cellular swelling and tissue edema. This cellular influx of water is associated with a corresponding reduction in ECF volume.[52] Swelling of the endoplasmic reticulum is the first ultrastructural change seen in hypoxic cell injury. Eventually, swelling of the mitochondria and cells is observed. The changes in cellular membrane potential impair a number of cellular physiologic processes such as myocyte contractility, cell signaling, and the regulation of intracellular Ca^{2+} concentrations. Once intracellular organelles such as mitochondria, lysosomes, or cell membranes rupture, the cell will undergo death by either apoptosis or necrosis.[53]

Hypoperfusion and hypoxia cause widespread cell death by apoptosis. Apoptosis has also been detected in trauma patients with ischemia and reperfusion injury. Apoptosis of lymphocytes and intestinal epithelial cells occurs within the first 3 hours of injury.[53] Apoptosis in intestinal mucosal cells may compromise barrier function of the intestine and lead to proinflammatory activation of submucosal immune cells with release of mediators into the portal circulation during shock. Also, lymphocyte and neutrophil apoptosis has been hypothesized to contribute to the immune suppression that is observed in trauma, as well as increased risk of nosocomial infections and MODS.[54]

Tissue hypoperfusion and cellular hypoxia may also result in systemic metabolic acidosis as metabolic by-products of anaerobic glycolysis exit the cells and gain access to the circulation. In the setting of acidosis, oxygen delivery to the tissues is improved as the oxyhemoglobin dissociation curve is shifted toward the right. The decreased affinity of hemoglobin for oxygen in erythrocytes results in increased tissue oxygen release and increased tissue extraction of oxygen. In addition, hypoxia stimulates the production of erythrocyte 2,3-diphosphoglycerate (2,3-DPG), which also contributes to

the shift to the right of the oxyhemoglobin dissociation curve and increases oxygen availability to the tissues during shock.

Epinephrine and norepinephrine released during shock have a profound impact on cellular metabolism beyond their effects on vascular tone. Hepatic glycogenolysis, gluconeogenesis, ketogenesis, breakdown of skeletal muscle protein, and lipolysis of adipose tissue are all increased by these catecholamines. Cortisol, glucagon, and ADH also participate in the regulation of catabolism during shock. Epinephrine induces the release of glucagon while inhibiting the release of insulin by pancreatic β-cells. In addition, shock blocks genomic and cellular responses necessary for insulin recognition and uptake and utilization of glucose. The result is a catabolic state with glucose mobilization, hyperglycemia, protein breakdown, negative nitrogen balance, lipolysis, and insulin resistance during shock and injury.[55] The relative underutilization of glucose by peripheral tissues preserves it for the glucose-dependent organs such as the heart and brain.

In addition to inducing changes in cellular metabolic pathways, shock also induces changes in cellular gene expression. The DNA-binding activity of a number of nuclear transcription factors is altered by the production of oxygen radicals, nitrogen radicals, and hypoxia that occurs at the cellular level in shock. The expression of other gene products including heat shock proteins, vascular endothelial growth factor (VEGF), inducible nitric oxide synthase (iNOS), and cytokines is also increased in shock.[56,57] Many of these shock-induced gene products, such as cytokines, have the ability to subsequently alter gene expression in specific target cells and tissues.

Shock induces profound changes in tissue microcirculation that may contribute to organ dysfunction and the systemic sequelae of severe hypoperfusion. These changes have been studied most extensively in the microcirculation of skeletal muscle in models of sepsis and hemorrhage. Whether microcirculatory changes are primarily a result of the development of shock or a pathophysiologic response that promotes tissue injury and organ dysfunction has been difficult to determine. After hemorrhage, larger arterioles vasoconstrict, most likely due to sympathetic stimulation, whereas smaller distal arterioles dilate, presumably due to local metabolic mechanisms.[58] Flow at the capillary level is heterogeneous with swelling of endothelial cells and aggregation and activation of leukocytes and platelets, which produces microvascular thrombosis and diminished capillary perfusion in some vessels both during shock and following resuscitation.[59]

Platelets play a pivotal role in traumatic shock, hemostasis, and coagulopathy.[60] Recent evidence suggests that there is an inverse relationship between platelet count on admission and early mortality and transfusion in critically injured patients. In addition to platelet loss and consumption, platelet adhesion, degranulation, and dysfunction all occur and cause both microcirculatory thrombosis and generalized coagulopathy. After injury, platelets demonstrate significantly decreased adenosine diphosphate (ADP)–mediated and thrombin receptor agonist peptide (TRAP)–mediated platelet aggregation and ADP-mediated P-selectin expression.[60] Such dysfunction is particularly encountered after severe traumatic brain injury. Platelet dysfunction measured by light transmission aggregometry has been associated with a more than 10-fold higher early mortality.[60,61] In the massively bleeding patient, early platelet transfusion along with transfusion of a ratio of 1:1 packed red blood cells (PRBCs) and fresh frozen plasma (FFP) is associated with higher rates of hemostasis, fewer hemorrhagic deaths, and shorter duration of mechanical ventilation (see Chapter 16).

Damaged EGL and endothelial cells express procoagulant mediators and cause consumption of coagulation factors (Fig. 15-4). Hemorrhage-induced microcirculatory dysfunction also occurs in vascular beds besides skeletal muscle (eg, lungs, kidneys, liver) and contributes to tissue injury and organ dysfunction.[62,63] In sepsis, similar changes in microcirculatory function occur. Aggregation and sludging of neutrophils and platelets in the microcirculation with thrombosis can aggravate shock-induced hypoperfusion, induce direct cellular injury via toxic neutrophil-dependent processes such as production of oxygen radicals or release of proteolytic enzymes, and impair cellular metabolism.[64] Further, microcirculatory changes in patients with traumatic shock have been shown to portend MODS.[65]

The decreases in microcirculatory blood flow and capillary perfusion result in decreased capillary hydrostatic pressure. The changes in hydrostatic pressure promote an influx of fluid from the extravascular space into capillaries in an attempt to increase circulating volume. These changes are associated with additional decrements in the volume of ECF due to simultaneous increased cellular swelling. These basic cellular and microcirculatory changes have significant physiologic importance in the ability of the organism to recover from circulatory shock. Resuscitation with volumes of fluid sufficient to restore the ECF deficit is associated with improved outcome after shock.

Quantifying Cellular Hypoperfusion

Hypoperfused tissues and cells experience what has been called oxygen debt, a concept first proposed by Drs. Jack Crowell and Elvin Smith in 1964.[66] Oxygen debt is the deficit in tissue oxygenation over time that occurs during shock. When oxygen delivery (DO_2) is limited, oxygen consumption (VO_2) may be inadequate to meet the metabolic needs of cellular respiration due to a deficiency in oxygen at the cellular level. The measurement of oxygen deficit is calculated by taking the difference between the estimated oxygen demand and oxygen consumption (VO_2). Under normal circumstances, cells can "repay" the oxygen debt during reperfusion. The magnitude of oxygen debt correlates with the severity and duration of hypoperfusion. In a canine model of hemorrhagic shock, Crowell and Smith demonstrated a direct relation between survival and degree of shock.[66] They determined that a marker of mortality was the inability to recover from the oxygen debt. The median lethal dose (LD_{50}) occurred at 120 mL/kg of oxygen debt. In a canine model of hemorrhagic shock, mortality was directly correlated to the calculated oxygen debt and similar to the LD_{50} reported by Crowell and Smith (113.5 mL/kg).[67] A relation between oxygen debt and survival in human patients has also been shown. In over 250 high-risk surgical patients, the calculated

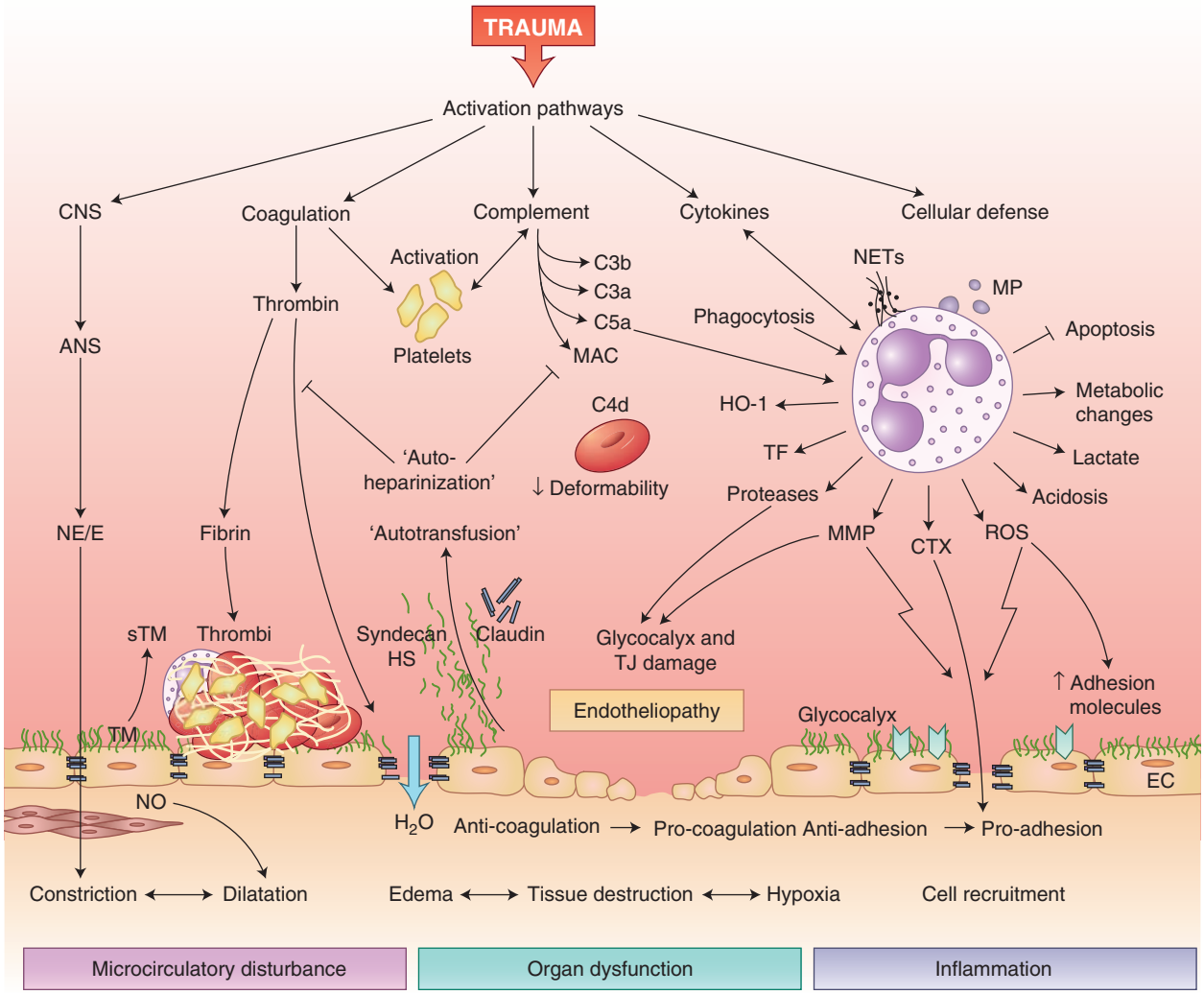

FIGURE 15-4 Activation of innate immune responses and endothelial dysfunction after trauma. After trauma, various pathways of innate immunity can induce posttraumatic endotheliopathy and further tissue damage. Activation of the autonomic nervous system (ANS) and its systemic release of norepinephrine and/or epinephrine (NE/E) leads to instant vasoconstriction (centralization), activates the endothelium, and induces the release of thrombomodulin (TM), which thereby diminishes the anticoagulant features of the endothelium. The dilation of subendothelial smooth muscle cells through stimulation with nitric oxide (NO) potentiates microcirculatory disturbances and hypoxia. Cleavage of thrombin during activation of the clotting cascade leads to the formation of a microthrombus on the endothelial surface and loosening of intercellular barriers, with efflux of water (H_2O) into the interstitial tissues. Activated platelets, in concert with products of the activation of coagulation and/or complement and leukocytes, form the thromboinflammatory response. The activation of complement on red blood cells compromises their deformability. The activation of innate leukocytes, particularly neutrophil granulocytes, by complement and proinflammatory cytokines creates an overall proinflammatory microenvironment with released neutrophil extracellular traps (NETs) and microparticles (MPs), reduced apoptosis, and metabolic changes that lead to local generation of lactate. The generation of reactive oxygen species (ROS) and matrix metalloproteinase (MMP) increases endothelial expression of adhesion molecules and widens cell–cell junctions, which facilitate the migration of leukocytes into inflamed tissue. Proteases secreted from leukocytes can damage the glycocalyx layer and tight junctions (TJs), which leads to the intravascular release of glycosaminoglycans that exhibit colloidal–osmotic and heparin-like effects. CNS, central nervous system; CTX, chemotaxis; EC, endothelial cell; HO-1, heme oxygenase-1; HS, heparan sulfate; MAC, membrane attack complex; NLR, NOD-like receptor; sTM, soluble thrombomodulin. (Reproduced with permission from Huber-Lang M, Lambris JD, Ward PA. Innate immune responses to trauma. *Nat Immunol*. 2018;19:327-341. Copyright © Springer.)

oxygen debt correlated directly with organ failure and mortality.[68] The maximum oxygen debt in nonsurvivors (33.2 L/m^2) was greater than that of survivors with organ failure (21.6 L/m^2) and survivors without organ failure (9.2 L/m^2). In addition, the total duration of oxygen debt and the time required to restore perfusion correlated with outcome. Survivors were able to reverse the oxygen debt–induced injury, whereas nonsurvivors were unable to recover from the oxygen debt. Thus, the magnitude of the oxygen debt, its rate of accumulation, and the time required to correct it all correlate with survival.

It is difficult to directly measure the oxygen debt during resuscitation of trauma patients. Easily obtainable clinical parameters such as arterial blood pressure, heart rate, urine output, central venous pressure (CVP), and pulmonary artery occlusion pressure are poor proxies of tissue perfusion. Therefore, surrogate parameters have been sought to estimate the

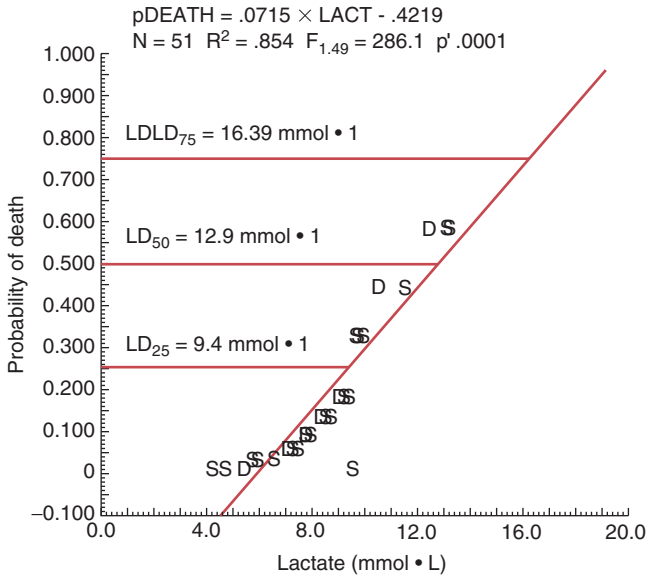

FIGURE 15-5 The relation between mortality and serum lactate levels is described by data generated in a canine hemorrhagic shock model. LD, lethal dose. (Reproduced with permission from Dunham CM, Siegel JH, Weireter L, et al. Oxygen debt and metabolic acidemia or quantitative predictors of mortality and the severity of the ischemic insult in hemorrhagic shock. *Crit Care Med*. 1991;19:231.)

oxygen debt. Experimental studies have shown that serum lactate and base deficit (BD) correlate with oxygen debt.[67,69] Cardiac output, blood pressure, and shed blood volume were all inferior to the BD and lactate in estimating the oxygen debt and in predicting mortality in hemorrhaged animals. Dunham et al[67] demonstrated a direct correlation between arterial lactate and probability of survival in a model of canine hemorrhage (Fig. 15-5). The LD_{50} for lactate was 12.9 mmol/L in hemorrhaged dogs.

BD is the amount of base in millimoles that is required to titrate 1 L of whole blood to a pH of 7.40 with the blood fully saturated with oxygen at 37°C (98.6°F) and a $Paco_2$ of 40 mm Hg. It is usually measured by arterial blood gas analysis using automated devices and has a rapid turnaround time. Good correlation between the BD and survival has been shown in patients with shock[70]; in blunt trauma patients with a BD of 0 mmol/L at presentation, mortality was 8% compared with 95% mortality among patients with a BD of 26 mmol/L. In the canine hemorrhage model previously mentioned, the LD_{50} occurred at a BD of 11.8 mmol/L (Fig. 15-6).

Other clinical parameters such as blood pressure, heart rate, hemoglobin, plasma lactate, and oxygen transport variables were not as accurate as the BD in determining the probability of death in trauma patients. However, neither BD nor serum lactate is precise for measuring physiologic stress as the oxygen debt. In Crowell's model of hemorrhage and resuscitation, lactate level decreased too slowly and tended to estimate higher residual oxygen debt, whereas the BD decreased more rapidly and tended to estimate lower values of oxygen debt. However, in the absence of better proxies, both lactate and BD are useful in the assessment of trauma patients and in the evaluation of the patient's response to resuscitation.

EVALUATION OF THE TRAUMA PATIENT IN SHOCK

General Overview

The manifestations of shock may be dramatic, as in the patient with profound hypotension. However, the signs of shock can be subtle and require careful assessment and interpretation by the trauma team. The evaluation, diagnosis, and treatment of the trauma patient in shock begin with the ABCs (airway, breathing, circulation) of the primary survey.[71]

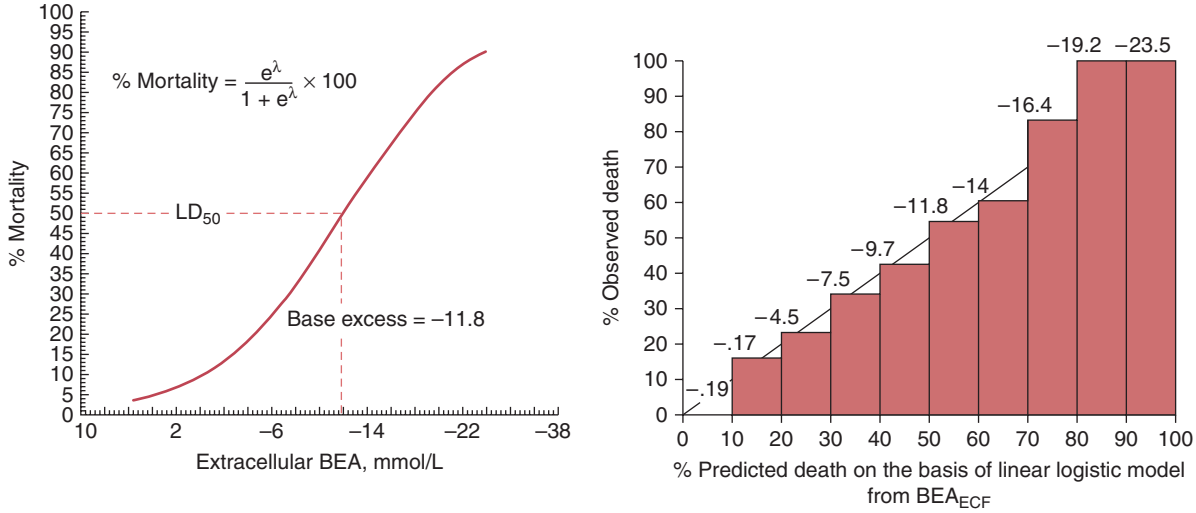

FIGURE 15-6 The relation between base deficit (negative base excess) and mortality is depicted for patients who suffered blunt hepatic injury. BEA, arterial base excess; ECF, extracellular fluid; LD_{50}, medial lethal dose. (Reproduced with permission from Siegel JH, Rivkind AI, Dalal S, et al. Early physiologic predictors of injury severity and death in blunt multiple trauma. *Arch Surg*. 1990;125:498, Copyright © 1990 American Medical Association. All rights reserved.)

Advanced shock may produce coma with loss of the ability to maintain and protect the airway, so that endotracheal intubation is necessary. Marked tachypnea may be present as a symptom of a primarily respiratory homeostatic response as the respiratory system attempts to compensate for metabolic acidosis or in response to generalized anxiety from hypoperfusion of the CNS.

During the primary survey, the circulation can be rapidly assessed by evaluation of the presence and location of the pulse (central vs peripheral), its rate, and its character. A rough estimation of systolic blood pressure (SBP) is possible by palpation of the radial pulse (SBP ≥80 mm Hg), both carotid and femoral pulses (SBP ≥70 mm Hg), or only carotid pulse (SBP ≥60 mm Hg). Absent peripheral pulses (ie, radial, pedal) associated with weak, rapid central pulses (ie, femoral, carotid) denote a profound circulatory disturbance that requires prompt intervention. Pulse pressure (systolic pressure minus diastolic pressure) is determined by stroke volume and vascular capacitance. Stroke volume is reduced in hypovolemic shock, which leads to a narrowed pulse pressure (normal range is 40–60 mm Hg). A narrowed pulse pressure is a sensitive indicator of impending severe shock for patients with less than 30% of blood volume lost; however, pulse pressure may be unreliable in older patients with less aortic and peripheral vascular compliance. Further, a normal SBP is markedly different in the extremes of age, which should be considered when attempting to evaluate a patient in shock or impending shock. For example, an SBP of 120 mm Hg in a baseline hypertensive adult may be less than adequate to maintain CNS perfusion. Associated findings that may be manifestations of abnormal tissue perfusion include cool clammy skin, altered sensorium (eg, confusion, lethargy, coma), and tachycardia. Low urine output, often considered an indicator of hypovolemia, is unlikely to be a useful adjunct in the initial assessment of the patient in the trauma resuscitation area. Further, measurement of blood pressure may be misleading because SBP may not be diminished in the early stages of shock. Compensatory mechanisms to maintain cerebral and coronary perfusion may maintain relatively normal systemic arterial pressure despite hypovolemia and significant hypoperfusion of splanchnic and peripheral tissues. Up to 30% of a patient's blood volume may be lost before significant changes in blood pressure occur. When present, hypotension represents an abject circulatory derangement and failure of compensatory mechanisms that portends imminent cardiovascular collapse and requires immediate intervention(s).

The correction of shock should begin immediately once recognized. Treatment generally begins before an etiology for shock is identified. The forms of shock are listed in Table 15-1; the most common etiology for shock in the injured patient is hypovolemia from loss of circulating blood volume (Fig. 15-7). Two large-bore intravenous lines (ie, at least 16-gauge peripheral or 7F central resuscitation catheters) should be inserted, and volume resuscitation ensues. The availability of rapid infusion systems facilitates prompt volume expansion with the delivery rate limited predominantly by the size and length of the intravenous cannulae (ie, catheter resistance). Fluid warmers to heat the infusate are essential to prevent hypothermia and mitigate the effects of concomitant coagulopathy and acidosis. For patients in profound shock, immediate blood replacement is necessary. As soon as possible, PRBCs should be given alongside both FFP and platelets to prevent the start or worsening of coexistent coagulopathy.

Hospitals should have a massive transfusion protocol (MTP) in place to ensure immediate access to blood components when necessary. Massive transfusion has traditionally been defined as administration of 10 or more units of PRBCs within 24 hours. However, this definition does not allow for prospective use or promote balanced blood product transfusion practices. A more recent definition for massive transfusion is the critical administration threshold, which is defined as the transfusion of at least 3 PRBC units within any 1-hour period in the first 24 hours of admission.[72] This new definition accounts for the intensity of ongoing resuscitation and need for early intervention to produce optimal results, avoids survivor bias implicit in the historical definition of massive transfusion, and has been prospectively validated as a sensitive predictor of mortality.[73]

As correction of the shock state is underway, the etiology for shock is systematically sought. Physical examination may indicate potential etiologies (eg, obvious external hemorrhage, flaccid warm extremities from spinal cord injury, penetrating precordial wounds). Rapidly performed adjunct examinations (ie, x-rays of chest and pelvis, focused assessment with sonography for trauma [FAST], diagnostic peritoneal aspiration [DPA]) can provide additional information while the initial resuscitation is being conducted and the response to resuscitation is being evaluated. Diagnostic maneuvers that do not directly contribute to the identification and treatment of shock should be deferred until shock has been corrected. As an example, traumatic brain injury is not considered a cause of shock until all other potential sources of hemorrhage or spinal cord injury are eliminated; therefore, head computed tomography is deferred until the source of shock is identified and managed.

Trauma patients can be categorized into three general groups with respect to their response to resuscitative maneuvers: responders, transient responders, and nonresponders (see Fig. 15-7).

- Responders are patients who rapidly correct their shock state with minimal replacement of intravascular volume. These patients often have had an intravascular volume loss but do not have ongoing bleeding; bleeding has stopped or been tamponaded (eg, multiple extremity fractures), or the patient has an etiology for hypoperfusion other than hypovolemia such as neurogenic shock or obstructive shock. Aggressive resuscitation should cease with return of normal clinical parameters to avoid fluid overload.
- Transient responders are patients who initially improve with resuscitative efforts but subsequently deteriorate. These patients frequently have intracavitary bleeding that will require surgical control or interventional radiology intervention (eg, high-grade liver or spleen laceration).

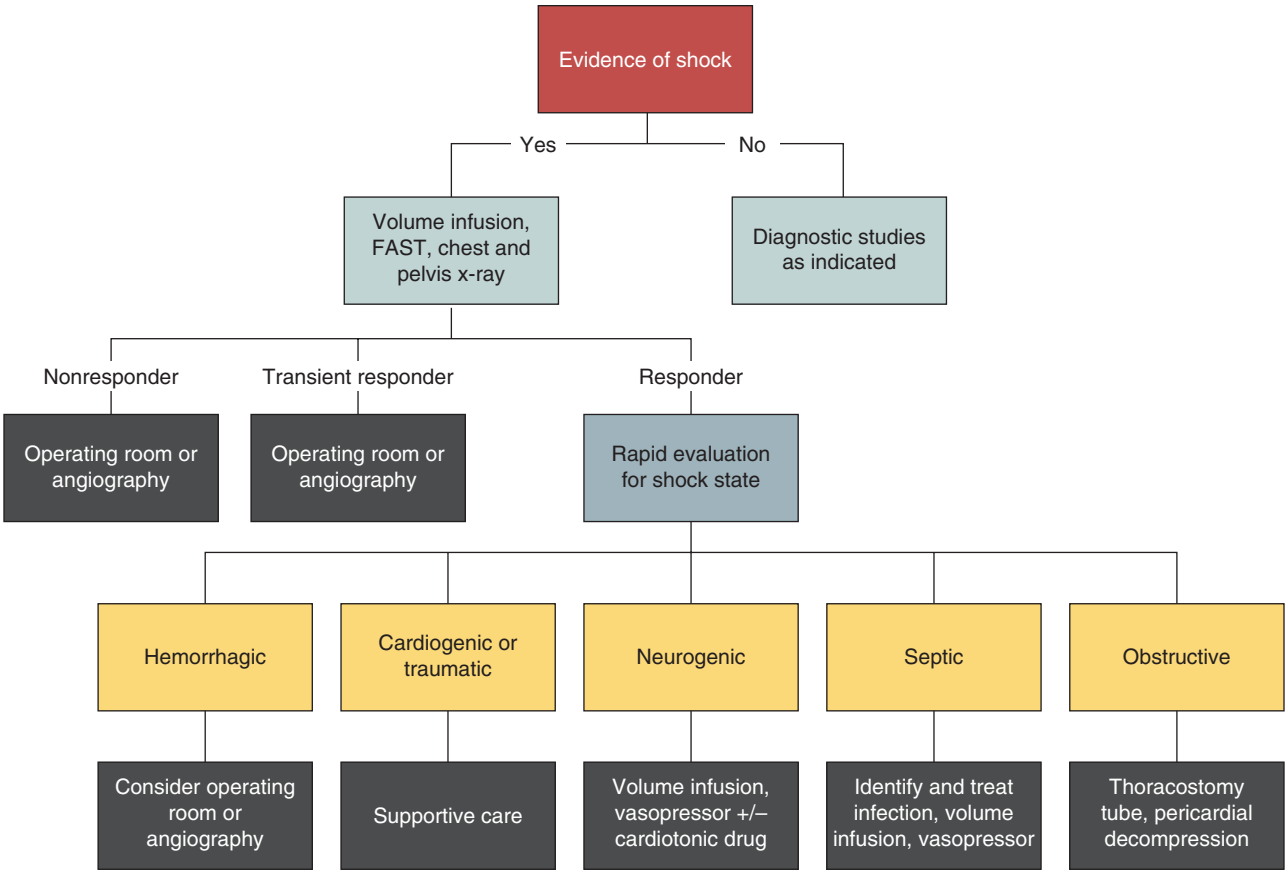

FIGURE 15-7 Tissue hypoperfusion algorithm. Trauma patients can be categorized into three general groups with respect to their response to resuscitative maneuvers. FAST, focused assessment with sonography for trauma.

- Nonresponders are patients who have persistent manifestations of shock despite vigorous resuscitative efforts. These patients are gravely ill and often present in extremis. These patients typically have high-volume bleeding from injuries to major vessels or severe injuries to solid organs that require immediate operative intervention or other invasive control maneuvers (eg, emergency resuscitative thoracotomy or endovascular balloon occlusion of the aorta [REBOA]). They will rapidly die from circulatory collapse or develop the progressive lethal triad of acidosis, hypothermia, and coagulopathy, and irreversible shock unless bleeding is rapidly controlled. Patients who have active, ongoing hemorrhage cannot be successfully resuscitated until hemorrhage has been controlled; survival depends on rapid identification of conditions that require operative or other invasive interventions.

VASCULAR ACCESS AND CONTROL FOR PATIENTS WITH SEVERE HEMORRHAGE

In the trauma patient presenting with multiple serious injuries and/or hemorrhagic shock, vascular access is necessary to restore intravascular volume rapidly. The most important factor in considering the procedure and route for vascular access is the anatomic location and magnitude of hemorrhagic injuries and the trauma team's expertise.

Venous access should never be initiated in an injured limb. In patients with injuries below the diaphragm, at least one intravenous (IV) line should be placed in a tributary of the superior vena cava, as there may be vascular disruption or obstruction of the inferior vena cava. In patients with severe multiple trauma in whom occult thoracoabdominal damage is suspected, it is ideal to have one IV access site above the diaphragm and one below the diaphragm, thus accessing both the superior vena cava and inferior vena cava, respectively. For rapid administration of large amounts of IV fluids, short large-bore catheters should be used. Recalling Poiseuille's law, doubling the internal diameter of the venous cannula increases the flow through the cannula 16-fold. When using 8.5F pulmonary vascular catheter introducers, the side port should be removed, as this increases the resistance roughly fourfold.

Advanced Trauma Life Support (ATLS) guidelines recommend rapid placement of two large-bore (ie, 16-gauge or larger) IV catheters in the patient with serious injuries and/or hemorrhagic shock.[71] The first choice for IV insertion should be a peripheral extremity vein. The most suitable veins are at the wrist, the dorsum of the hand, the antecubital fossa in the arm,

and the saphenous in the leg. The complication rate of properly placed IV catheters is low. Intravascular placement of a large-bore IV should be verified by assessing backflow. An IV site should infuse easily without added pressure. IV fluids can leak into soft tissues when pumped under pressure through an infiltrated IV line and may create a compartment syndrome.

Subclavian, internal jugular, and femoral catheterization should be used with caution in hypovolemic trauma patients as the venous collapse associated with their hypovolemia may increase the risk of inadvertent arterial puncture and cannulation. In part, for this reason, the rate of success is lower and complications are more frequent in these situations. Rapid peripheral percutaneous IV access may be difficult to achieve in patients with hypovolemia and venous collapse, edema, obesity, scar tissue, history of IV drug abuse, or burns. In such cases, subclavian or femoral vein catheterization usually provides rapid and safe venous access in experienced hands. The most frequent complication of subclavian vein catheterization is pneumothorax. Pneumothorax is more likely to occur on the left side because the left pleural dome is anatomically higher.

Subclavian and internal jugular catheters should be inserted on the side of injury in patients with chest wounds, reducing the chances of collapse of the uninjured lung. A simple pneumothorax may result in respiratory compromise in individuals with pulmonary contusions or a pneumothorax in the contralateral hemithorax. Regardless of the site of insertion, it is extremely important not to force the wire, dilator, or introducer if resistance is encountered. Forcing the introducer could result in perforation of large central veins or arteries and bleeding. Venous air embolism is another complication of central line insertion. All central venous access lines should be placed with complete aseptic technique.

Although percutaneous placement of internal jugular catheters is an excellent means of attaining rapid large-bore catheter access, this is a rather unusual site for intravenous insertion in trauma patients because of the possibility of cervical trauma and the need for cervical collar immobilization.

Thrombophlebitis or line infection is more common with femoral catheters; however, this is most common with prolonged use. Any lines placed during resuscitation of a trauma patient without strict aseptic technique should be removed as soon as the patient's condition permits.

In patients with hemodynamic shock and in situations where percutaneous peripheral or central venous access is either contraindicated or impossible to achieve, intraosseous (IO) placement is a feasible alternative for both volume replacement and infusion of therapeutic agents; however, massive transfusions may not be achieved using the IO route. Preferred locations are the proximal or medial tibia or lateral aspect of the greater tubercle of the humerus.

In addition to IV access for volume resuscitation, REBOA is a tool useful in select patients with noncompressible torso hemorrhage.[74] Although there is no high-grade evidence that unequivocally demonstrates improved survival compared to standard care in patients with severe hemorrhage, REBOA is less invasive and, in well-trained hands, may be more rapidly applied than emergency resuscitative thoracotomy. Arterial access can be achieved percutaneously by ultrasound guidance, palpation, or direct cutdown; the latter is often considered after traumatic arrest or when circulating volume is extremely low.[75] Current indications for use of REBOA in trauma include[74]:

1. Life-threatening hemorrhage below the diaphragm in patients who are unresponsive or transiently responsive to resuscitation
2. Life-threatening hemorrhage below the diaphragm and traumatic arrest (no evidence exists for the duration of arrest and use of REBOA, but it should be used within the same time period as resuscitative thoracotomy would be used)
3. Severe intra-abdominal or retroperitoneal hemorrhage, or those with traumatic arrest and inflation of the balloon catheter in the distal thoracic aorta (zone 1)
4. Severe pelvic, junctional, or proximal lower extremity hemorrhage and inflation of the balloon catheter in the distal abdominal aorta (zone 3)

There are a number of potential complications related to REBOA, namely those related to arterial access (eg, arterial disruption, dissection, pseudoaneurysms, hematoma, extremity ischemia), aortic and iliac injuries, balloon rupture and loss of hemorrhage control, spinal cord ischemia, stroke and other thromboembolic events, and complications of visceral malperfusion.[76] REBOA should only be used in situations where a surgeon is immediately available to definitively address the hemorrhage to avert complications related to spinal cord, visceral, and limb ischemia. Specifically, placement of REBOA in zone 1 should only be performed if the anticipated time to the start of the operation is less than 15 minutes. REBOA in zone 3 may be tolerated for a longer period of time and may be use for temporary hemorrhage control prior to angioembolization, preperitoneal packing, or exploration; however, once aortic occlusion is achieved, hemostasis should occur immediately and the balloon deflated as soon as possible. Although not well studied, intermittent deflation and/or partial aortic occlusion may prolong the safe interval for REBOA use; when this is performed, care should be taken to avoid catheter migration and inadvertent balloon inflation in an unintended zone or the iliac vessels. Lastly, removal of the sheath should be done as soon as possible. Vigilant assessment of extremity perfusion should occur before, during, and after REBOA use and sheath removal. Consultation with vascular surgeons when using the REBOA and during sheath removal is recommended.

In addition to intravascular access and hemorrhage control and application of manual pressure, early tourniquet application can be used to arrest life-threatening extremity hemorrhage.[77] Certain tourniquets have proven efficacy in achieving broad and appropriately high arterial occlusion pressure (eg, combat application tourniquet, special operations forces tactical tourniquet, emergency and military tourniquet) and should be used when possible. Tourniquet time should be documented,

and the tourniquet should be removed as soon as possible in a facility with definitive hemorrhage control capacity.[78]

RESUSCITATION FLUIDS

Systemic reviews have consistently shown that there is little evidence that resuscitation with one type of fluid compared with another reduces mortality or that one solution is more effective or safer than another.[79,80] However, there is mounting evidence that the types and dose of resuscitation fluid administered may affect patient outcomes in specific populations. Therefore, the selection and administration of fluid should be based on the patient's degree and type of shock, comorbidities, and potential toxic effects of excessive fluid to capitalize on the benefits of appropriate resuscitation while minimizing adverse effects, namely those related to fluid overload.

Crystalloid Solutions

Crystalloids are solutions of ions, which determine the fluid's tonicity (eg, lactated Ringer's, normal saline, PlasmaLyte, hypertonic saline). These solutions are the mainstay of resuscitation given their relatively physiologic pH and electrolyte compositions. In general, crystalloids are inexpensive, easy to ship and store, and fairly innocuous in small doses. However, crystalloid solutions pass relatively freely across the vascular endothelium and damaged EGL, often resulting in pronounced ECF expansion during resuscitation with negative consequences on organ systems when given indiscriminately.

LACTATED RINGER'S

Ringer's solution was developed in 1885 to aid the development of an extracorporeal beating heart model.[81] As such, the original solution was an isotonic salt solution that contained calcium required for perpetuation of the cardiac cycle. In the 1930s, Hartmann added lactate to the Ringer's solution to act as a buffer, which is metabolized to bicarbonate and carbon dioxide. Lactated Ringer's is a relatively balanced solution that mimics normal human electrolyte composition and has a pH of 6.5. No human study has unequivocally demonstrated superiority of lactated Ringer's versus other crystalloid solutions. However, resuscitation with large volumes of lactated Ringer's was less likely to potentiate acidosis and cause hyperchloremia, hyperkalemia, and/or dilutional coagulopathy in animal models of hemorrhagic shock.[82,83] Data have also shown that the *R*-isomer of lactate is capable of activating the innate proinflammatory response and aggravating tissue and organ injury, reinforcing the use of *L*-lactate isomer in lactated Ringer's solutions.[84]

NORMAL SALINE

Saline solution was developed during the Indian blue cholera epidemic in the 1830s.[85] The solution was designed to be "normal" or isotonic, measured by its tendency to not lyse red blood cells. However, normal saline (0.9% sodium chloride) has supraphysiologic amounts of sodium and chloride (154 mEq of each) and is markedly more acidotic than human plasma (pH 5.0). Thus, resuscitation with significant volumes of normal saline causes hyperchloremic metabolic acidosis that compounds the acidemia characteristic of shock states.[82] Renal dysfunction has been attributed to hyperchloremic metabolic acidosis from normal saline resuscitation.[86,87] Plasma chloride content regulates renal blood flow and may contribute to acute kidney injury.[88] Delivery of chloride to the macula densa drives mesangial contraction and decreases glomerular filtration.[89] A meta-analysis of high- versus low-chloride fluid resuscitation in critically ill patients found an increased incidence of acute kidney injury but no difference in mortality.[90]

PLASMALYTE

PlasmaLyte is a family of balanced crystalloid solutions with formulations developed to mimic the pH, osmolality, and electrolyte compositions of normal human plasma.[91] PlasmaLyte has a significant buffer capacity from the addition of lactate, acetate, and gluconate. Use of PlasmaLyte for resuscitation has been shown to result in a lower base deficit and more physiologic chemistry profile compared to use of normal saline.[91] In general surgical patients, use of PlasmaLyte has been associated with a lower rate of major complications (eg, postoperative infection, renal replacement therapy, blood transfusion, acidosis-related investigations) compared to normal saline.[92,93] Although cost constraints have been reported as barriers to the use of PlasmaLyte, a cost-minimization analysis of PlasmaLyte suggested that use of it is overall more cost effective due to less need for electrolyte repletion and lower incidence of ongoing acidemia compared to normal saline.[94]

HYPERTONIC SALINE

Concerns about the effect of large-volume isotonic crystalloid resuscitation led to experimentation with small-volume resuscitation using hypertonic saline (eg, 3%, 5%, and 7.5% sodium solutions). There is no high-quality evidence that the use of hypertonic saline for resuscitation provides short- or long-term mortality benefits to the critically ill.[95] However, use of low-volume hypertonic saline among patients who underwent damage control surgery resulted in a lower incidence of ARDS, sepsis, MODS, and mortality.[96] Further, there is some evidence that patients who had hypertonic saline used as a maintenance fluid after damage control laparotomy were more likely to achieve primary fascial closure and were closed more quickly than those who did not receive hypertonic saline.[97]

However, Resuscitation Outcomes Consortium (ROC) trials for both shock and traumatic brain injury were halted after preliminary data showed no beneficial effect of hypertonic saline for either one of these groups studied in the clinical trials.[98,99] In addition, use of hypertonic saline appears to increase the coagulopathy seen in severe hemorrhagic shock.[100] Better evidence is needed before recommending the routine use of hypertonic saline in this population.

Colloid Solutions

Colloid solutions (eg, albumin hetastarch, gelatins, dextran) contain macromolecules suspended in salt solutions that were designed to remain within the intravascular fluid compartment under Starling's original assumptions regarding the role of plasma oncotic pressure in fluid physiology. However, as described earlier, the disrupted EGL does not retain large molecules, limiting the theoretical effectiveness of these solutions. Given that there is no significant absorption of interstitial fluid to the plasma in the setting of damaged EGL and increased interstitial oncotic pressure, one can understand why colloid-based resuscitation (eg, albumin, dextran) does not prevent or improve tissue edema or result in a sustained expansion of intravascular volume.

ALBUMIN

Albumin was made available with the advent of blood fractionation in 1941 and was used for the first time in large quantities as a resuscitation fluid for patients burned during the attack on Pearl Harbor.[101] Human recombinant albumin is commonly available in 5% and 25% solutions suspended in an isotonic salt solution. Albumin is produced by the fractionation of blood and is heat treated to reduce the risk of transmission of bloodborne infections; thus, albumin is relatively expensive to produce and distribute compared to other crystalloid and colloid solutions.

A meta-analysis compared albumin with crystalloid solutions in patients with hypovolemic shock, burns, or hypoalbuminemia. Pooled analysis found that use of albumin-based resuscitation was of no benefit and was potentially associated with an increased rate of death compared to crystalloid solutions.[102] Subsequently, a randomized trial examined the safety of 4% albumin for use in critically ill adults.[103] The Saline versus Albumin Fluid Evaluation (SAFE) study of 7000 critically ill patients randomized to 4% albumin or normal saline reported no difference between albumin and saline with regard to 28-day mortality or development of new organ failure. Secondary analyses of results from the SAFE study suggested an association between albumin and death at 2 years among patients with traumatic brain injury.[104] However, albumin resuscitation was associated with a decreased risk of death at 28 days in patients with severe sepsis and septic shock.[105]

In contrast to the SAFE study, the Albumin Italian Outcome Sepsis (ALBIOS) study randomized 1818 patients with severe sepsis to daily administration of 20% albumin targeting a serum albumin level of 3 g/L or crystalloid resuscitation.[105] There were no differences in either 28- or 90-day mortality rates between the two groups. Similarly, the multicenter Colloids Versus Crystalloids for the Resuscitation of the Critically Ill (CRISTAL) trial did not show a mortality difference at 28 days between patients with hypovolemic shock randomized to albumin or saline.[106]

Given the additional cost of albumin and a recent meta-analysis reporting no impact of albumin on sepsis-related mortality, the Surviving Sepsis Campaign (SSC) continues to recommend crystalloids as the initial sepsis resuscitation fluid.[107] However, the SSC has advised consideration of albumin when patients require substantial amounts of crystalloids.[108]

SEMISYNTHETIC COLLOIDS

The donor pool required and relative cost of albumin prompted the development of semisynthetic colloid solutions (eg, hydroxyethyl starch [HES], succinylated gelatin, dextran). Globally, HES solutions are the most commonly used semisynthetic colloids and have gained popularity in the recent past due to use in military medicine.[109] HES solutions are produced by hydroxyethyl substitution of amylopectin from starches (eg, sorghum, maize, potatoes)[110]; this substitution protects against hydrolysis by nonspecific amylases in the blood, which has been proposed to prolong the intravascular expansion associated with the volume and increased oncotic pressure provided by the larger molecules. However, when the vascular endothelium and EGL are damaged and leaky, HES accumulates in tissues, such as skin, lung, liver, and kidney, and has been shown to cause significant adverse effects.[111] Further, large-volume HES resuscitation has been associated with risks of renal injury and coagulopathy.[112] Studies of the use of HES in blunt trauma and burns and among critically ill patients requiring volume resuscitation suggest a potential correlation with increased risk of renal replacement therapy and mortality.[109,113,114] Therefore, the potential harms and cost of HES likely outweigh the benefits of its administration when other solutions are available and suggest that semisynthetic colloids should not be used for volume resuscitation in critically ill patients.

PLASMA

Although plasma is typically considered for patients at risk of or with clinically significant coagulopathy, it has other potential uses in resuscitation. For example, plasma has extreme buffering capacity that can be useful in profound acidemia and may better maintain and potentially restore damaged vascular endothelium and EGL compared with other solutions.[115,116] The use of plasma as a resuscitation fluid is limited by its cost and limited donor pool, as well as risks of transfusion reactions (eg, transfusion-related acute lung injury [TRALI], transfusion-associated circulatory overload, ABO incompatibility), immunosuppression, and transmission of bloodborne infections. Further, the use of plasma for resuscitation in patients without coagulopathy has been associated with development of sepsis, MODS, and ARDS.[117-119]

Given the aforementioned limitations and risks, solvent/detergent-treated plasma has been introduced that minimizes risk of the transmission of enveloped viruses (eg, HIV, hepatitis B and C) and is hypothesized to reduce the risk of TRALI.[120] However, solvent/detergent-treated plasma is prohibitively expensive in most settings. Freeze-dried plasma and lyophilized plasma were developed to lengthen the storage life of plasma and make its transport easy for use in combat settings.[121,122] These products have demonstrated the same hemostatic properties as fresh plasma after reconstitution.

Further, lyophilized plasma is compatible with all blood types and can be stored at room temperature for up to 2 years, and its reconstitution requires less than 6 minutes.[122] Further research is needed to determine specific indications for such products in the management of civilian patients with severe hemorrhage.[123,124]

EXCESSIVE RESUSCITATION

Early landmark work in the physiology of shock states identified several perturbations of normal homeostasis, including abnormal capillary permeability, decreased oncotic pressure, and a loss of intravascular volume into the interstitial fluid compartment.[14] Therefore, pioneers in shock resuscitation advocated for aggressive replacement of the fluid sequestered from the intravascular space in addition to maintenance fluid. This led to the legacy of supraphysiologic resuscitation and excessive resuscitation, which has profound functional consequences on multiple organ systems.[125]

Excessive resuscitation in trauma patients is associated with impaired oxygen delivery, preventable cardiopulmonary morbidity, prolonged intensive care unit and hospital stay, prolonged mechanical ventilation, intestinal anastomotic failure, compartment syndromes, increased intracranial pressure in the setting of traumatic brain injury, MODS, and death.[125-132]

Conversely, excessive fluid restriction and fixed, unmonitored fluid resuscitation are associated with hypovolemia, poor oxygen delivery, secondary brain injury in patients with traumatic brain injury, and overall worsened organ dysfunction.[133] Therefore, it has been inferred that a restrictive but goal-directed fluid resuscitation approach to maintain adequate perfusion while avoiding excessive intravascular pressure that enhances blood and volume loss during shock states may lead to better outcomes.[134]

DAMAGE CONTROL RESUSCITATION

There is significant evidence that the best resuscitative fluid for a person in persistent or severe hemorrhagic shock is not crystalloid or colloid but, in fact, a balanced infusion of blood components to mimic, as possible, replacement of lost whole blood.[135] This includes early administration of PRBCs, which have been shown to improve outcomes when given early in resuscitation of hemorrhagic shock. A balanced transfusion strategy of PRBCs, FFP, and platelets has been shown to enhance hemostasis, reduce the incidence of MODS, and decrease death due to exsanguination.[136,137]

Major advances in the resuscitation of hemorrhagic shock have come from the experiences and research of military medicine. Most recently, damage control resuscitation (DCR) was developed by the Tactical Combat Casualty Care Committee of the US military and used for combat casualties in Iraq and Afghanistan.[138] The principles of DCR include the following:

- Permissive hypotension
- Restriction of crystalloid resuscitation
- Earlier blood transfusion with balanced plasma and platelet to red blood cell transfusion ratios
- Goal-directed correction of coagulopathy

Traditionally, the management of patients in shock has been focused on providing aggressive fluid resuscitation with crystalloid or colloid solutions to rapidly restore circulating blood volume and thus maintain "optimal" vital organ perfusion. This approach can potentially increase bleeding by elevating the blood pressure and dislodging established blood clots. Other unwanted effects of aggressive fluid resuscitation include worsening coagulopathy and increased tissue edema, which play a role in the occurrence of abdominal compartment syndrome and MODS/MOF.

Hypotensive resuscitation is not a new concept. As described earlier, in 1918, Cannon described the deleterious effects of injecting fluids before the surgeon could achieve vascular control of the injury.[5] Cannon suggested an end point of resuscitation prior to definitive hemorrhage control of a systolic pressure of 70 to 80 mm Hg using a crystalloid/colloid mixture as his fluid of choice. In 1994, Bickell et al[139] published a prospective analysis comparing immediate and delayed fluid resuscitation in hypotensive adult trauma patients with penetrating torso injuries. In the delayed group, fluid administration was withheld until the time of operative intervention. Improved survival was seen in this population, with a trend toward fewer complications. These data corroborated the concept that delaying fluid resuscitation until hemorrhage is controlled improves outcome in this select group of penetrating trauma patients.

In subsequent publications, investigators have shown that increased prehospital time associated with attempts to place an intravascular access as well as prehospital use of rapid infusion were correlated with increased mortality.[140] Importantly, outcomes were not significantly different when patients were randomized to either a blood pressure of 70 mm Hg or SBP of 100 mm Hg. Multiple studies have since confirmed these findings.[141] Such findings support a resuscitation approach that mimics the management of patients with another form of shock, those with a ruptured abdominal aortic aneurysm, in whom aggressive volume resuscitation during their preoperative management has been found to increase the perioperative risk of death regardless of their SBP measurements.[142]

Management protocols currently adapted from military experience recommend the use of a palpable radial pulse (ie, SBP of 80–90 mm Hg) and/or the presence of a normal mental status as the most appropriate indicators of adequate perfusion. Combat casualties found at the scene with a palpable radial pulse and normal mentation are given intravenous access, but no intravenous fluids are infused until arriving to a far-forward facility where initial surgical management can be simultaneously instituted.

In civilian trauma, a goal-directed approach in the prehospital setting demonstrated that while a 2-L bolus of crystalloid in the normotensive patient was associated with an increased mortality, the same bolus in the significantly hypotensive (SBP <90 mm Hg) patient led to an improvement in survival, supporting the need for a controlled volume in

the severely hypotensive patient with a goal SBP of 90 mm Hg. Overall, preservation of native hemostatic mechanisms appears best achieved by allowing for a lower than normal blood pressure, thus reducing the rate of bleeding while still ensuring adequate tissue perfusion pressure for optimal survival. The characteristics of the local environment, the type of injury, and whether fast and adequate hemostasis can be promptly achieved are the current determinants for the use of limited resuscitation.

Exceptions to this resuscitative strategy include two main groups: elderly patients and those with traumatic brain injury. For elderly patients, many of whom are on antihypertensive medications at baseline, even an SBP of 110 to 120 mm Hg may be indicative of relative hypotension and cerebral hypoperfusion. Therefore, resuscitation in these individuals must take their expected baseline perfusion pressures into account. Second, in civilian trauma centers, patients with multiple blunt injuries are the most common, and the most critical injury is a traumatic brain injury. In the setting of potential traumatic brain injury, hypotension remains the best-defined risk factor known to produce a secondary insult to the injured brain tissue at risk and a significant worsening of functional outcome. Hypotension in these individuals has been associated with an increased mortality.[143] A controlled but less hypotensive approach (ie, goal SBP of 110 mm Hg) to resuscitation appears most appropriate in these settings.

Restricting crystalloid infusion resuscitation is another contrasting concept to traditional overly aggressive volume resuscitation that aimed to prevent occult hypoperfusion from splanchnic vasoconstriction and subsequent MODS. Studies have demonstrated that large-volume crystalloid-based resuscitation in the absence of physiologic targets is associated with an increased risk of ARDS, abdominal compartment syndrome, and MODS and higher mortality.[125,144-146] In patients with hemorrhagic shock, administration of large volumes of crystalloid solutions promotes dilutional and consumptive coagulopathy, promotes hypothermia, and may worsen metabolic acidosis with high doses of chloride; these three states have been termed *the lethal triad* and, when present, are associated with a marked increase in the risk of death.[147] Therefore, modern strategies for early fluid resuscitation focus on immediate whole blood or balanced blood component transfusions in massively bleeding patients.

Transfusion ratios of plasma to platelet to PRBC of nearly 1:1:1 closely approximate the whole blood that has been lost leading to hemorrhagic shock. The Prospective, Observational, Multicenter, Major Trauma Transfusion (PROMMTT) Study evaluated the association between in-hospital mortality and the timing and amount of blood products.[136] Among patients dying from hemorrhage, there was a protective association of higher blood product ratios (>1:2 plasma or platelet to PRBC) and decreased mortality that was concentrated to the first 24 hours for plasma and the first 6 hours for platelets. The results from the PROMMTT Study prompted the first multicenter randomized trial to compare blood component transfusion ratios, the Pragmatic, Randomized Optimal Platelet and Plasma Ratios (PROPPR) trial.[137] PROPPR was conducted to assess the effectiveness and safety of a 1:1:1 transfusion ratio (plasma to platelets to PRBCs) compared to a 1:1:2 transfusion ratio for patients requiring a massive transfusion. There was no difference in overall mortality at 24 hours or 30 days between the two transfusion ratio groups. However, fewer patients died of exsanguination in the 1:1:1 group. Rates of transfusion-related complications (eg, ARDS, MODS, sepsis, venous thromboembolism) were not different between the two treatment groups. From these studies, the American College of Surgeons Committee on Trauma recommends a plasma to red blood cell (RBC) transfusion ratio of 1:1 to 1:2. Additionally, the recommended ratio of platelets to RBCs is 1:6, with one platelet pack as either a single donor apheresis or random donor platelet pool equal to 6 units of PRBCs.[148]

Recently, military experience has demonstrated that early, far-forward transfusion of fresh whole blood is associated with less total volume transfusion, less coagulopathy, limited inflammatory response, and improved survival compared to standard blood component transfusion.[149-151] Application of whole blood transfusion in civilian trauma centers has been limited by concerns surrounding the theoretical reduction in the hemostatic efficacy of cold-stored platelets, risk of hemolytic transfusion reactions if group O whole blood is used in non–group O recipients, and logistical issues of providing whole blood through a civilian hospital blood bank.[152] In response to these concerns, multiple studies have demonstrated that the hemostatic effects of platelets in cold-stored whole blood are well preserved and bacterial growth is retarded by cold-stored platelets compared to room temperature–stored platelets due to decelerating lactate metabolism.[153,154] Additionally, use of strict donor criteria (eg, group O positive, male to mitigate risk of TRALI, titer of anti-A and anti-B antibodies <50), platelet-sparing leukoreduction, whole blood storage and component salvage protocols, and careful selection of recipients have made transfusion of fresh whole blood possible in the civilian setting.[155] There has been only one randomized controlled trial of whole blood transfusion versus component blood transfusion in the civilian setting.[156] Although this trial did not show a difference in the primary outcome of volume of blood transfused between the two arms, the study had several limitations. The whole blood used in the study was platelet depleted and supplemented with transfusion of apheresis or pooled random-donor platelets once 6 units of whole blood were transfused. Additionally, the study included severe and nonsurvivable traumatic brain injuries that likely confounded analysis of the primary outcome measure. A sensitivity analysis that excluded patients with traumatic brain injuries demonstrated that use of whole blood significantly reduced transfused volume compared to blood component therapy. Trials comparing transfusion of whole blood leukoreduced with a platelet-sparing filter versus balanced transfusion of blood components in the civilian setting are underway.

Conventional coagulation assays (ie, the international normalized ratio, prothrombin time, partial thromboplastin time, platelet count, fibrinogen concentration) and newer

viscoelastic assays of coagulation (ie, thromboelastography and rotational thromboelastometry) should be used to further guide correction of coagulopathy during ongoing resuscitation (see Chapter 16).[157]

FORMS OF SHOCK

Hypovolemic Shock

Hypovolemic shock occurs when rapid loss of fluids results in inadequate intravascular volume and subsequent inadequate perfusion. As previously noted, the most common cause of shock in the trauma patient is loss of circulating volume from hemorrhage. Acute blood loss causes decreased stimulation of baroreceptors (ie, stretch receptors) in the large arteries, resulting in decreased inhibition of vasoconstrictor centers in the brainstem, increased stimulation of chemoreceptors in vasomotor centers, and diminished output from atrial stretch receptors. These changes increase vasoconstriction and peripheral arterial resistance. Hypovolemia also induces sympathetic stimulation, leading to the release of epinephrine and norepinephrine, activation of the renin–angiotensin cascade, and increased release of vasopressin. Peripheral and splanchnic vasoconstriction is prominent in the maintenance of MAP, whereas lack of sympathetic effects on cerebral and coronary vessels and local autoregulation promote maintenance of blood flow to the heart and brain.

DIAGNOSIS

Shock in a trauma patient should be presumed to be due to hemorrhage until proven otherwise. Treatment is instituted as soon as shock is identified, typically before a source of hemorrhage is located.

The clinical and physiologic response to hemorrhage has been classified according to the magnitude of volume loss. Loss of up to 15% of the circulating volume (ie, 700–750 mL for a 70-kg patient) may produce little in terms of obvious symptoms, whereas loss of up to 30% of the circulating volume (ie, 1.5 L) may result in mild tachycardia, tachypnea, and anxiety. Hypotension, marked tachycardia (ie, adult pulse >110 bpm), and confusion may not be evident until more than 30% of the blood volume has been lost, whereas loss of 40% of circulating volume (ie, 2 L) is immediately life threatening. Symptoms of the degrees of hypovolemic shock are summarized in Table 15-2. Thus, there is a fine line between the development of mild symptoms of shock and the presence of life-threatening blood loss. Young, healthy patients with vigorous compensatory mechanisms may tolerate larger volumes of blood loss while manifesting fewer clinical signs. These patients may maintain a near-normal blood pressure until a precipitous cardiovascular collapse occurs. Elderly patients may be taking medications that either promote bleeding (eg, warfarin, aspirin, novel oral anticoagulants) or mask the compensatory response to hypovolemia (eg, β-blockers, calcium channel blockers, ACE inhibitors). In addition, atherosclerotic vascular disease, diminished cardiac compliance with age, inability to elevate heart rate or cardiac contractility in response to hemorrhage, and overall decline in physiologic reserve decrease the ability of the elderly patient to tolerate hemorrhage.[158]

Understanding the mechanism of injury of the patient in shock will help direct the evaluation and management. Identifying the source of blood loss in patients with penetrating wounds is relatively simple since potential bleeding sources will be located along the known or suspected path of the wounding agent. Patients with penetrating injuries who are in shock usually require operative intervention. Occasionally, patients in shock from penetrating injuries may have problems in addition to hemorrhage that are readily treated by simple maneuvers outside the operating room. Treatment of a tension pneumothorax (ie, obstructive shock) with insertion of a thoracostomy tube in the emergency department (ED) is one example. Generally speaking, shock from penetrating wounds is typically due to ongoing hemorrhage that mandates operative control.

Patients who suffer multisystem injuries from blunt trauma often have multiple sources of potential hemorrhage. However, there is a limited number of sites that can harbor sufficient extravascular blood volume to induce hypoperfusion or hypotension. Prehospital medical reports may confirm a significant blood loss at the scene of an accident, history of massive blood loss from wounds, visible brisk bleeding, or presence of an open wound in proximity to a major vessel. Injuries to major vessels should be suspected when there is ongoing hemorrhage from an open pelvic fracture. Although persistent bleeding from uncontrolled small vessels can, over time, precipitate shock if left untreated, attributing profound blood loss to these wounds (eg, scalp lacerations) should be

TABLE 15-2: Symptoms Associated with Blood Loss and Hypovolemic or Hemorrhagic Shock

0%–15% loss	15%–30% loss	30%–40% loss	>40% loss
Normal or increased pulse pressure	Tachycardia	Marked tachycardia	Extreme tachycardia
Mildly anxious	Decreased pulse pressure	Decreased systolic blood pressure	Hypotension
	Tachypnea	Marked tachypnea	Extreme tachypnea or respiratory failure
	Delayed capillary refill	Oliguria	Negligible urine output
	Anxious	Cool, clammy skin	Cold, pale skin
		Confusion or agitation	Lethargic or loss of consciousness

done only after major intracavitary bleeding or other etiologies have been excluded.

When major blood loss is not immediately visible, intracavitary blood loss should be suspected. Intraperitoneal hemorrhage is a common source of blood loss inducing shock. Its presence may be suspected based on physical examination (eg, distended abdomen, abdominal tenderness, visible abdominal wounds), although the sensitivity of the physical examination for detecting substantial abdominal injuries after blunt trauma is notoriously unreliable. A large volume of intraperitoneal blood from abdominal injuries may be present before the physical examination is abnormal. Therefore, FAST or DPA is used frequently in the resuscitation area to rapidly identify potential significant intraperitoneal blood in the unstable patient who may require emergency laparotomy. In selected patients, diagnostic laparotomy may be indicated.

The chest can be a significant source of hemorrhage as each pleural cavity has the capacity to hold 2 to 3 L of blood. Diagnostic and therapeutic tube thoracostomy may be indicated in patients based on clinical findings or evidence of a hemopneumothorax on a chest x-ray or extended FAST (eFAST; includes the FAST scan and sonographic examination of bilateral lungs and thoracic spaces).[159] In addition, with the immediate removal of greater than 1000 mL of blood, a second tube thoracostomy should be placed to ensure patency and accurate monitoring of ongoing blood loss.

Major retroperitoneal hemorrhage occurring in association with a pelvic fracture can be diagnosed by urgent pelvic radiography. The pattern of the pelvic fracture may provide clues as to the risk of massive blood loss, but none is adequately predictive in the individual patient to exclude need for further evaluation.

TREATMENT

Hemorrhage control is a central component of resuscitation of the patient in shock and is part of the primary survey. Treatment of hemorrhagic shock is instituted concurrently with diagnostic evaluation to identify a source. As mentioned earlier, all trauma patients in shock should be presumed to have hemorrhage until proven otherwise. The method of treatment will depend on the patient's response to resuscitation, the specific injury or injuries responsible for the blood loss, and consideration of factors such as mechanism of injury, age of the patient, associated injuries, and institutional resources. Patients who fail to respond to initial resuscitative efforts should be assumed to have ongoing active hemorrhage (eg, external bleeding, pleural cavity, peritoneal cavity, retroperitoneum, femur fracture[s]) and require prompt intervention. Identification of the body cavity harboring active hemorrhage will help focus operative efforts, but since time is of the essence, rapid treatment is essential and diagnostic laparotomy or thoracotomy may be indicated. Alternatively, temporary or definitive endovascular control of distal or central hemorrhage can be attempted using angiographic intervention with embolization, stenting, or REBOA, where appropriate.[160] The actively bleeding patient cannot be resuscitated until control of ongoing hemorrhage is achieved.

Patients who respond to initial resuscitative efforts but then deteriorate hemodynamically frequently have injuries that require operative intervention. The duration of their response will dictate whether and which diagnostic maneuvers can be performed safely to identify the site of bleeding. Hemodynamic deterioration typically denotes ongoing bleeding for which some form of surgical or angiography-based intervention is required. As noted earlier, with cessation of hemorrhage, even patients who have lost significant intravascular volume will often respond to resuscitative efforts if the degree and duration of shock have been limited. In patients with hemorrhagic shock, survival is improved if the time between injury and control of bleeding is reduced. Clarke et al[161] demonstrated that trauma patients with major abdominal injuries requiring emergency laparotomy had an increased probability of death with increasing length of time in the ED. This probability increased approximately 1% for every 3 minutes in the ED up to 90 minutes.

A subset of patients fails to respond to resuscitative efforts despite adequate control of ongoing hemorrhage. These patients may present in one or more ways: have ongoing fluid requirements despite adequate control of hemorrhage; have persistent hypotension despite restoration of intravascular volume; require vasopressor support to maintain their systemic blood pressure; and/or exhibit a futile cycle of uncorrectable hypothermia, hypoperfusion, acidosis, and coagulopathy that cannot be interrupted despite maximum therapy. These patients have classically been described to be in decompensated or irreversible shock, and mortality may be inevitable. The hemodynamic decompensation and paradoxical peripheral vasodilation responsible for irreversible shock and the clinical factors that predict its onset have not been elucidated.

Neurogenic Shock

Neurogenic shock refers to diminished MAP and tissue perfusion as a result of loss of vasomotor tone to peripheral vascular beds. Loss of vasoconstricting impulses results in increased vascular capacitance, decreased venous return, and decreased cardiac output. Neurogenic shock is typically due to injuries to the spinal cord from fractures of the cervical or high thoracic vertebrae that disrupt sympathetic regulation of peripheral vascular tone (see Chapter 26). Occasionally, an injury such as an epidural hematoma impinging on the spinal cord can produce neurogenic shock without an associated vertebral fracture. Penetrating wounds to the spinal cord can also produce neurogenic shock.

Sympathetic input to the heart that normally increases heart rate and cardiac contractility and input to the adrenal medulla that increases the release of catecholamines can be disrupted by a high injury to the spinal cord, preventing the typical reflex tachycardia that occurs with the relative hypovolemia from increased venous capacitance and loss of vasomotor tone.

In addition, acute spinal cord injury results in activation of multiple secondary injury mechanisms: (1) vascular

compromise to the spinal cord with loss of autoregulation, vasospasm, and thrombosis; (2) loss of cellular membrane integrity and impaired energy metabolism; and (3) neurotransmitter accumulation and release of free radicals. Importantly, hypotension contributes to the worsening of acute spinal cord injury as a result of further reduction in blood flow to the injured spinal cord.

DIAGNOSIS

The classic description of neurogenic shock consists of the following: decreased blood pressure associated with bradycardia or normal heart rate (indicating the absence of reflexive tachycardia due to disrupted sympathetic discharge); warm extremities due to loss of peripheral vasoconstriction; motor and sensory deficits indicative of an injury to the spinal cord; and radiographic evidence of a fracture or injury in the vertebral column or spinal cord (see Chapter 26). However, determining the presence of neurogenic shock may be difficult since patients with multisystem trauma may have confounding injuries such as a traumatic brain injury, tension pneumothorax, and/or uncontrolled intracavitary hemorrhage. In a study of patients with injuries to the spinal cord from penetrating wounds, most patients with hypotension had blood loss as the etiology (74%) and not a neurogenic cause, and few (7%) had all the classic findings of neurogenic shock.[162] Hypovolemia from hemorrhage should be sought and excluded before the diagnosis of neurogenic shock is made. To assume that the cause of hypotension in a multiply injured patient is due to neurogenic shock without first evaluating and treating potential hemorrhage is often a costly mistake.

In patients who have neurogenic shock, the severity of the spinal cord injury seems to correlate with the magnitude of the cardiovascular dysfunction. Patients with complete motor deficits from spinal cord injury are over five times more likely to require vasopressors for neurogenic shock compared to those with incomplete lesions.[163] Similarly, patients with high cervical spine injuries (ie, C1–C5) are more likely to require cardiovascular intervention compared to those with lower cervical or high thoracic spine injuries.[164]

TREATMENT

After the airway is secured and ventilation is adequate, fluid resuscitation and restoration of functional intravascular volume will often improve systemic blood pressure and perfusion in neurogenic shock. Most patients with neurogenic shock will respond to volume resuscitation alone, with adequate improvement in perfusion and resolution of hypotension. Administration of vasoconstrictors (eg, norepinephrine, phenylephrine) can improve peripheral vascular tone, decrease vascular capacitance, and increase venous return, but should only be considered once hypovolemia is excluded and the diagnosis of neurogenic shock is established. Some studies suggest that norepinephrine promotes better restoration of blood flow and oxygenation compared to phenylephrine, particularly in cervical and upper thoracic injuries.[165] There are no high-quality data regarding optimal MAP goals and duration in the management of acute spinal cord injury. However, MAP goals of 85 to 90 mm Hg for 5 to 7 days should be considered.[166] The duration of the need for vasopressor support for neurogenic shock may correlate with the overall prognosis for improvement in neurologic function.[163]

Appropriate and rapid restoration of blood pressure may also improve perfusion to the spinal cord, prevent progressive ischemia of the spinal cord, and minimize secondary injury to the spinal cord.[167] Restoration of normal hemodynamics should precede any operative attempts to stabilize the vertebral fracture. Patients who are hypotensive from spinal cord injury are best monitored in an intensive care unit and should be carefully followed for evidence of cardiac or respiratory dysfunction.

Cardiogenic Shock

Cardiogenic shock refers to a failure of the circulatory pump leading to inadequate forward flow and subsequent tissue hypoperfusion and ischemia, in the setting of adequate intravascular volume. Hemodynamic criteria for cardiogenic shock include sustained hypotension (ie, SBP ≤90 mm Hg for at least 30 minutes in a young adult), reduced cardiac index (<2.2 L/min/m^2), and elevated pulmonary artery occlusion pressure (>15 mm Hg) if left heart failure is present, or elevated CVP (>8 mm Hg) if isolated right heart failure is present. Acute myocardial infarction is the most common cause of cardiogenic shock. In this population, mortality for cardiogenic shock ranges between 50% and 80%.

In the trauma patient, inadequate cardiac function after blunt thoracic trauma can be due to blunt myocardial injury, cardiac arrhythmia, myocardial infarction, or direct injury to a cardiac valve. As the average age of the population increases, the prevalence of comorbid medical conditions in trauma patients will also increase. In addition, a large proportion of the elderly population is on β-blockers and calcium channel blockers, which confound expected hemodynamic responses to hemorrhage. Elderly patients with preexisting intrinsic cardiac disease will be more susceptible to suffering an acute myocardial infarction or significant arrhythmia associated with the stress of injury that can also induce cardiac failure and cardiogenic shock (see Chapter 58).

Diminished cardiac output or contractility in the face of adequate intravascular volume (ie, preload) may lead to hypoperfused vascular beds and reflexive sympathetic discharge. Increased sympathetic stimulation of the heart, either through direct neural input or from circulating catecholamines, increases heart rate, myocardial contraction, and myocardial oxygen consumption. Patients with fixed, flow-limiting stenoses of the coronary arteries may not be able to increase coronary perfusion to meet the increased myocardial oxygen demands; thus, these lesions further increase the risk for myocardial damage. Diminished cardiac output decreases coronary artery blood flow, resulting in a scenario of increased myocardial oxygen demand at a time when myocardial oxygen supply may be limited and insufficient.

Acute heart failure can also result in fluid accumulation in the pulmonary microcirculatory bed, impairing the diffusion of oxygen from the alveolar space and decreasing myocardial oxygen delivery even further.

DIAGNOSIS

Rapid identification of the patient with pump failure and initiation of treatment are essential in preventing further decreases in cardiac output. If increased myocardial oxygen needs cannot be met, there will be progressive and unremitting cardiac dysfunction. Blunt injury to the heart is rarely severe enough to induce pump failure, but manifestations of shock in the setting of a patient at risk should raise one's index of suspicion (see Chapter 58). Elderly patients with known preexisting cardiac disease are at increased risk of suffering injury-related cardiac complications including cardiac failure. Furthermore, elderly patients with intrinsic cardiac disease are at risk of suffering a primary cardiac event that induces syncope, a fall, or loss of control of one's vehicle that then leads to presentation to a trauma center.

Making the diagnosis of cardiogenic shock involves the identification of cardiac dysfunction or acute heart failure in a susceptible patient. Since patients with blunt cardiac injury typically have multisystem trauma, hemorrhagic shock from intra-abdominal bleeding, intrathoracic bleeding, and bleeding from fractures must be excluded. In most instances of blunt cardiac injury, the symptoms are self-limited with no long-term cardiac sequelae. Relatively few patients with blunt cardiac injury will develop dysfunction of the cardiac pump, and those who do generally exhibit cardiogenic shock early in their evaluation.[168] Therefore, establishing the diagnosis of blunt cardiac injury is secondary to excluding other etiologies for shock and establishing that significant cardiac dysfunction is present.

Invasive cardiac hemodynamic monitoring, which generally is not necessary, may be useful in the complex patient with the combination of hemorrhagic shock and cardiogenic shock, when it is necessary to exclude right ventricular infarction or mechanical cardiac complications, or in the patient with known preexisting myocardial disease (see Chapter 58). This typically involves continuous monitoring of cardiac output and other hemodynamic variables using the pulmonary artery catheter (PAC). Transesophageal echocardiography (TEE) provides excellent views of the myocardium that are not hindered by subcutaneous air, bandages covering chest wounds, thoracostomy tubes, or unfavorable body habitus that may limit evaluation of cardiac function by transthoracic echocardiography. However, the rapid evaluation of cardiac function by TEE may be problematic in the presence of severe maxillofacial trauma or unstable injuries to the cervical spine that can interfere with probe placement. There is evidence that evaluation of cardiac function using bedside ultrasound by surgeons, emergency medicine practitioners, and intensivists demonstrates adequate accuracy to direct treatment.[169] In addition to these tools, serum troponin assays are both sensitive and specific for myocardial damage and can be used to assess for ischemic, demand-associated, or trauma-related injury. It is important to note that all of these tools are only point-in-time assessments and serial evaluation is routinely needed.

TREATMENT

Patients with blunt cardiac injury will often have associated injuries that produce hypovolemia, and expansion of intravascular volume as an initial maneuver can improve perfusion significantly. However, hypervolemia can magnify the physiologic derangements produced by cardiac dysfunction and should be avoided. In addition, under conditions of limited flow, anemia becomes a potential limiting factor and requires transfusion to higher levels as a buffer to help ensure adequate oxygen delivery. In the face of acute myocardial infarction or acute myocardial ischemia, maintaining a hematocrit of 30% may be indicated.[170] When profound cardiac dysfunction exists, ionotropic support may be indicated to improve cardiac contractility and cardiac performance. Several agents are available, and selection is dependent on the overall cardiovascular pathophysiology (see Chapter 58).

Patients whose cardiac dysfunction is refractory to cardiotonics may require mechanical circulatory support with an intra-aortic balloon pump.[171] This can be inserted at the bedside in the intensive care unit via the femoral artery through either a cutdown or percutaneous approach. In addition, recent data support use of venoarterial extracorporeal membrane oxygenation for acute cardiac failure due to trauma.[172] Aggressive circulatory support of patients with cardiac dysfunction from intrinsic cardiac disease has led to more widespread application of these devices and more familiarity with their operation by both physicians and critical care nurses (see Chapter 58).

Patients who have suffered an acute myocardial infarction around the time of their injury should have preservation of existing myocardium and cardiac function as priorities of therapy. The use of anticoagulation or thrombolytic therapy for the management of acute coronary syndromes will depend on associated injuries and the risk of secondary bleeding. Patients in cardiac failure from an acute myocardial infarction may benefit from pharmacologic or mechanical circulatory support, similar to that of patients with cardiac failure related to blunt cardiac injury. Additional pharmacologic tools include the use of β-blockers to control heart rate and myocardial oxygen consumption, nitrates to promote coronary blood flow through vasodilation, and ACE inhibitors to reduce ACE-mediated vasoconstriction that increases myocardial workload and oxygen consumption.[173] Selected patients who do not have significant associated injuries may be candidates for coronary angiography and subsequent procedures to improve coronary blood flow such as transluminal angioplasty, coronary artery stents, or urgent coronary artery bypass grafting.

Septic Shock

Sepsis is an inflammatory response to severe infection characterized by hypovolemia, vasodilation, and capillary leak treated by early antibiotics and fluid resuscitation. The multidisciplinary consensus consortium, the Surviving Sepsis

Campaign, has established useful standardized definitions for the patient with an inflammatory response with or without sepsis.[174] First, the *systemic inflammatory response syndrome* (SIRS) defines the clinical innate immune clinical manifestation that occurs in response to a wide variety of physiologic insults and is defined as the presence of two or more of the following conditions:

- Temperature greater than 38°C or less than 36°C
- Pulse rate greater than 90 bpm
- Respiratory rate greater than 20 breaths/min or $Paco_2$ less than 32 mm Hg
- White blood cell count greater than 12,000/mm^3 or less than 4000/mm^3 or greater than 10% immature (band) forms

Because of the lack of specificity of SIRS, the third International Sepsis Task Force defined *sepsis* as life-threatening organ dysfunction caused by a dysregulated host response to infection and *septic shock* as a subset of sepsis with circulatory and cellular/metabolic abnormalities associated with higher risk of mortality.[175] The new definition emphasizes the pathologic response to infection, the increased risk of mortality, and the need for urgent recognition of sepsis. In addition to the new definition of sepsis, the task force published recommendations for the use of clinical criteria to identify patients with sepsis and septic shock. The Sequential Organ Failure Assessment (SOFA) score is used to identify organ dysfunction in patients with suspected infection.[176,177] Baseline SOFA score in a patient without preexisting organ dysfunction (prior to infection) is assumed to be 0. Patients with a presumed infection and an increase in SOFA score of 2 or greater have an overall mortality risk of 10%.[178] For patients in the ED or on the hospital wards (outside of the intensive care unit), the Quick SOFA (qSOFA) score is a bedside tool that does not require laboratory tests to screen patients for possible infection. qSOFA uses three criteria, assigning one point each for low SBP (≤100 mm Hg), high respiratory rate (≥22 breaths/min), and altered mental status (Glasgow Coma Scale score <15). Identification of patients with two or more qSOFA criteria should prompt further workup for organ dysfunction and initiation of therapy as indicated. Use of SOFA and qSOFA scores is primarily intended to facilitate earlier recognition and initiation of treatment for patients with sepsis and septic shock. Without such scores and systematic screening tools, sepsis and septic shock are often underdiagnosed, and treatment delays and poor outcomes are common.

These definitions reinforce that septic shock is a clinical syndrome that occurs as a result of the body's immune and inflammatory responses to invasive or severe localized infection, typically from bacterial or fungal pathogens. In its attempt to eradicate the pathogens, the innate immune system elaborates a wide array of proinflammatory mediators (eg, cytokines, chemokines). These mediators enhance effector mechanisms for macrophage and neutrophil killing, increase procoagulant-induced microcirculatory thrombosis and fibroblast activity to localize the invaders, and increase surrounding microvascular blood flow to enhance delivery of bactericidal mediators to the area of invasion. When this response is overly exuberant or becomes systemic rather than localized, manifestations of sepsis become evident. These findings include peripheral vasodilation, fever, leukocytosis, and tachycardia.

Sepsis is an uncommon etiology for shock in the acute presentation of a trauma patient unless there has been a substantial delay between injury and presentation to the ED. Typically, invasive infection occurs days to weeks after injury and is prevalent in the severely injured and immunosuppressed patient who develops a nosocomial infection in the intensive care unit.[179]

DIAGNOSIS

Attempts to standardize terminology have led to the establishment of criteria for the diagnosis of sepsis in the hospitalized adult.[175] These criteria include manifestations of the host response to infection (eg, fever, leukocytosis, mental contusion, tachypnea, tachycardia, hypotension, oliguria), as well as identification of an offending organism. Septic shock requires the presence of these conditions associated with hypotension resistant to volume resuscitation and evidence of organ dysfunction due to tissue hypoperfusion.

Recognizing septic shock in the trauma patient begins with defining high-risk groups: critically ill patients in the intensive care unit with organ dysfunction requiring invasive support and who are immunosuppressed from their injuries; patients who have suffered injuries associated with significant contamination (eg, colorectal wounds with fecal spillage, soft tissue wounds embedded with soil or dirt); patients with injuries that may be associated with persistent devitalized tissue (eg, crush or electrical injury); patients whose wounds put them at risk for complications (eg, anastomotic disruption, pancreatic leak); or patients with missed injuries.

The clinical manifestations of septic shock should prompt the empiric initiation of treatment after obtaining appropriate cultures and before bacteriologic confirmation of an organism or source of active infection is identified. An aggressive search for the source of the infection includes a thorough physical examination, inspection of all wounds, evaluation of intravascular catheters or other foreign bodies (eg, urinary catheter, thoracostomy tube, surgical drain), sampling of appropriate body fluids for culture, and imaging studies as indicated by the patient's condition.

The hemodynamic parameters characteristic of septic shock include peripheral vasodilatation with resultant decrease in systemic vascular resistance. Initially, there is a hyperdynamic cardiac state, and after volume resuscitation, the cardiac output may be significantly elevated. Diagnosis and treatment do not usually require placement of a PAC to guide therapy in patients with septic shock. Most of these patients can be resuscitated using CVP, central venous oxygen saturation ($Scvo_2$), and serum lactate.

TREATMENT

Obtunded patients may require intubation to protect their airway while patients whose work of breathing is excessive may require intubation and mechanical ventilation to prevent

respiratory collapse. Empiric antibiotics that cover the most likely pathogens based on institution-specific antibiogram should be chosen while culture results are pending (see Chapter 21).[108] Antibiotics should be tailored to cover the responsible organisms once culture data are available and, if appropriate, the spectrum of coverage narrowed. Long-term empiric use of broad-spectrum antibiotics should be minimized to reduce the development of resistant organisms and avoid the potential complications of fungal overgrowth and antibiotic-associated colitis from *Clostridium difficile*.

Patients with sepsis are typically hypovolemic from decreased intake, large-volume intravascular volume losses into the interstitium from EGL disruption, and increased insensible losses. In addition, the inflammation related to the immune response to infection alters systematic vascular resistance, venous capacitance, and myocardial function, further exacerbating hypoperfusion. Resultant decreases in stroke volume and cardiac output imbalance oxygen delivery and demand, precipitating tissue hypoxia, shock, anaerobic metabolism, and lactic acidosis. The rationale for fluid resuscitation in sepsis is to restore intravascular volume, cardiac output, and oxygen delivery before irreversible injury occurs. As sepsis progresses, pathogen and host mediators impede mitochondrial and endoplasmic reticular function and cellular uptake and utilization of the oxygen delivered.[180]

The landmark study by Rivers et al[181] on the use of early goal-directed therapy (EGDT) in the treatment of severe sepsis and septic shock highlighted most importantly the early prompt recognition and treatment of sepsis to prevent ongoing organ insults. The study randomized 263 patients with sepsis and hypoperfusion to either standard therapy or EGDT. Standard therapy involved arterial and central venous catheterization and a protocol targeting CVP of 8 to 12 mm Hg, MAP of at least 65 mm Hg, and urine output of at least 0.5 mL/kg/h. EGDT included all elements of standard therapy in addition to a catheter measuring $Scvo_2$; 6 hours of treatment in the ED before admission; and protocolized administration of 500 mL of IV crystalloid every 30 minutes to achieve CVP goals, vasopressors and vasodilators to maintain MAP goals, and blood transfusion or dobutamine to achieve $Scvo_2$ of 70% or greater. During the 6 hours of intervention, EGDT patients received more IV fluids, red cell transfusions, and dobutamine. In-hospital mortality was 16% lower with EGDT compared to standard therapy. The mortality benefit demonstrated by the study by Rivers et al[181] made early, protocol-driven, goal-directed fluid resuscitation the preferred strategy for septic shock globally.

Over the past decade, three large, multicenter trials (ProCESS, ARISE, and ProMISe trials) compared EGDT to usual care in which invasive management was optional (eg, central venous access in ProCESS) or not permitted (eg, $Scvo_2$ measurement in ARISE).[182-184] These trials reported no differences in any clinical outcome between EGDT and usual care among the 4201 patients in these trials, calling into question the need for invasive hemodynamic monitoring in the current era of sepsis awareness, aggressive early treatment, and earlier and better access to critical care services.[88]

After initial resuscitation, the potential benefits of fluid must be balanced against the risks of excessive resuscitation. Observational studies have associated positive fluid balance with additional morbidity and mortality. Multiple trials have reported increased odds of mortality for patients with higher fluid balance after resuscitation compared to those who were maintained more euvolemic.[185,186] Although these studies were inherently limited by indication bias (ie, that patients with higher severity of illness may have been more likely to both have fluid administered and die compared to those with a less severe illness), the association calls into question unchecked resuscitation and the potential role of deresuscitation or diuresis. The Fluid and Catheter Treatment Trial (FACTT) controlled postresuscitation fluid management for 1000 ARDS patients, of whom 70% had underlying sepsis.[128] Once adequate resuscitation was achieved, the fluid strategy that emphasized diuresis and limiting fluid boluses reduced the number of ventilator and intensive care unit days without precipitating cardiovascular or renal dysfunction.

Vasopressor therapy may be required as a supportive measure when hypotension is refractory to volume infusion in patients in septic shock. α-Adrenergic agents promote peripheral vasoconstriction, improve systemic blood pressure, and can be titrated by continuous infusion to target an adequate MAP to maintain core organ perfusion (see Chapter 57).

For patients in septic shock, current recommendations advise immediate initiation of resuscitation with a minimum of 30 mL/kg of IV crystalloid fluid administered within the first 3 hours.[108] During this resuscitation, serial evaluation with diagnostics and reassessment of responsiveness to fluid should take place. Patients with circulatory failure may require an additional fluid bolus to increase stroke volume. However, the risk of repeated fluid challenges is fluid overload, which has potentially deleterious effects on the function of the heart, lungs, and kidneys, leading to increased mortality in patients with septic shock. A study of postresuscitation fluid boluses in severe sepsis and septic shock demonstrated that ongoing and repeated fluid boluses did not have a sustained effect on parameters typically associated with improved outcomes (eg, urine output, $Scvo_2$, lactate). While resuscitation commences, efforts should be made to obtain cultures prior to initiation of antibiotic therapy, promptly perform imaging to identify the source of infection as appropriate, initiate broad-spectrum antibiotic therapy within 1 hour of diagnosis of septic shock, achieve source control, and subsequently narrow antibiotic coverage once microbiologic and susceptibility data allow.

In the trauma patient, IV antibiotics alone will frequently be insufficient to adequately treat a severe surgical infection. Source control (ie, drainage of infected fluid collections, removal of infected foreign bodies, and debridement of devitalized tissue) is essential to eradicate the infection. This process may require multiple operations. For patients who manifest symptoms of septic shock early in their hospitalization, consideration of the possibility of a missed injury to a hollow viscus should be entertained. Missed abdominal injuries represent a significant source of sepsis and the septic response leading to MODS.

Stress-dose glucocorticoid therapy should be given primarily to patients with septic shock based on clinical parameters and only if hypotension is poorly responsive to fluid and vasopressor therapy, so-called "refractory shock." Multiple strategies for immune modulation have been developed and tested for the treatment of sepsis and septic shock. Use of antiendotoxin antibodies, anticytokine antibodies, cytokine receptor antagonists, immune enhancers, anti–nitric oxide compounds, and oxygen radical scavengers has been tried.[187,188] Each of these compounds is designed to alter a specific aspect of the host immune response to shock. To date, each of these strategies has failed to demonstrate clinical efficacy in improving patient outcome despite utility in well-controlled experiments. For example, previous trials demonstrated efficacy of activated protein C in improving mortality from sepsis, but subgroup analysis of patients with sepsis at low risk of death documented an increased risk of bleeding complications associated with therapy without a substantial improvement in survival.[189,190] It is unclear whether the failure of these interventions is due to trial design, inadequate understanding of the interactions of the complex immune response to injury and infection, or use of animal models of shock that poorly represent human disease.[191]

Sepsis and nosocomial infections in critically ill patients continue to represent significant sources of morbidity and consume substantial health care resources. Despite advances in critical care, the mortality rate for severe sepsis and shock remains at 30% to 50%.

Obstructive Shock

Hypoperfusion can be due to mechanical or anatomic obstruction impeding venous return to the heart or preventing cardiac filling. The end result of either of these two events is decreased cardiac output leading to decreased peripheral perfusion. Most commonly, obstruction is due to the presence of a tension pneumothorax, massive pulmonary embolism, or cardiac tamponade (see Chapters 28 and 30). With any of these conditions, there is decreased cardiac output associated with increased CVP.

DIAGNOSIS AND TREATMENT

The manifestations of a tension pneumothorax are the presence of shock in the context of diminished breath sounds over one hemithorax, hyperresonance to percussion, jugular venous distension, and shift of mediastinal structures to the unaffected side. Unfortunately, not all of the clinical manifestations of tension pneumothorax may be evident on physical examination. Hyperresonance may be difficult to appreciate in a noisy resuscitation area. Jugular venous distension or tracheal deviation may be obscured by a cervical collar and not seen unless specifically sought. Furthermore, hypovolemia from concurrent bleeding may diminish CVP and prevent jugular venous distension even when increased pleural, pulmonary artery, or pericardial pressure restricts outflow.

For the multiply injured patient with life-threatening hypotension, the placement of bilateral chest tubes may be both diagnostic and therapeutic. In these circumstances, a chest x-ray is unnecessary and potentially a dangerous waste of time. Due to the immediate threat to life, the diagnosis of tension pneumothorax should be a clinical one. If a chest x-ray is obtained due to missing the diagnosis on clinical examination, the typical findings include deviation of mediastinal structures, depression of the hemidiaphragm (ie, deep sulcus sign), and hypo-opacification with absent lung markings.

Cardiac tamponade results from the accumulation of blood within the pericardial sac and most commonly occurs from penetrating trauma. While precordial wounds are most likely to injure the heart and produce tamponade, any projectile or wounding agent that passes in proximity to the mediastinum can potentially produce tamponade. Blunt rupture of the heart is rare but has been reported. The diagnosis is aided by the FAST examination, which can be performed immediately on all injured patients presenting with signs of shock at risk of cardiac injury. The manifestations of cardiac tamponade may be as catastrophic as total circulatory collapse and cardiac arrest, or they may be more subtle. A high index of suspicion is warranted to make a rapid diagnosis. Patients who present with circulatory arrest due to cardiac tamponade from a precordial penetrating wound require emergency pericardial decompression through a sternotomy or left anterolateral thoracotomy, and the indications for these maneuvers are reviewed in Chapters 27 and 30. Cardiac tamponade may also be associated with hypotension, muffled heart tones, jugular venous distension (Beck triad), and elevated CVP with tachycardia.[192] Absence of these clinical findings may not be sufficient to exclude cardiac injury and cardiac tamponade. As described earlier, muffled heart tones may be difficult to appreciate in a busy trauma center, and jugular venous distension may be diminished by coexistent bleeding and hypovolemia. Therefore, patients at risk for cardiac tamponade whose hemodynamic status permits should undergo diagnostic testing.

Invasive hemodynamic monitoring may support the diagnosis of cardiac tamponade if elevated CVP, pulsus paradoxus (ie, decreased systemic arterial pressure with inspiration), or elevated right atrial and right ventricular pressure by PAC is present. These hemodynamic profiles suffer from lack of specificity, the time required to obtain them, and their inability to exclude cardiac injury in the absence of tamponade. Chest radiographs may provide information on the possible trajectory of a projectile but are rarely diagnostic since the acutely filled pericardium distends poorly. Pericardial ultrasound as part of a FAST examination through either the subxiphoid or transthoracic approach has excellent sensitivity in detecting pericardial fluid.[193] The yield in identifying pericardial fluid obviously depends on the skill and experience of the ultrasonographer, body habitus of the patient, and absence of wounds that preclude visualization of the pericardium. Standard two-dimensional transthoracic echocardiography or TEE to evaluate the pericardium for fluid is typically performed by cardiologists or anesthesiologists skilled at evaluating ventricular function, valvular abnormalities, and integrity

of the proximal thoracic aorta. These skilled examiners may not be immediately available, and waiting for this test may result in inappropriate delays and poor outcomes. In addition, although both ultrasound techniques may demonstrate the presence of fluid or characteristic findings of tamponade (ie, large volume of pericardial fluid, right atrial collapse, poor distensibility of the right ventricle), negative tests do not exclude cardiac injury.

Pericardiocentesis to diagnose pericardial blood and potentially relieve tamponade has a long history in the evaluation of the trauma patient. However, its inability to evacuate clotted blood and potential to produce cardiac injury make it a poor alternative. Diagnostic pericardial window represents the most direct method to determine the presence of blood within the pericardium (see Chapter 30). It can be performed through either the subxiphoid or transdiaphragmatic approach. Some authors report performing this technique using local infiltrative anesthesia. However, the ability to achieve satisfactory safety and visualization in the trauma victim who may be intoxicated, in pain, or anxious from hypoperfusion usually mandates the use of general anesthesia. Once the pericardium is opened and tamponade relieved, hemodynamics will usually improve dramatically, and formal pericardial exploration can be performed. Exposure of the heart can be achieved by extending the incision to a sternotomy, a left anterolateral thoracotomy, or bilateral anterior thoracotomies ("clamshell") depending on the injury, as reviewed in Chapters 27 and 30.

Traumatic Shock

Traumatic shock is defined by a combination of several insults after injury that, alone, may be insufficient to induce shock, but together produce profound hypoperfusion. Hypoperfusion from relatively modest loss of volume can be magnified by the proinflammatory activation that occurs following direct injury and/or shock-induced tissue damage. The systemic response after trauma, combining the effects of soft tissue injury, long bone fractures, and blood loss, is clearly a distinct physiologic response compared to hemorrhagic shock alone. In addition to ischemia or ischemia/reperfusion, hemorrhage alone can induce qualitatively proinflammatory activation and cause many of the cellular changes typically attributed previously only to septic shock.[42] Examples of traumatic shock include small-volume hemorrhage accompanied by significant injury to tissue (eg, femur fracture, crush injury) or combination of hypovolemic with neurogenic, cardiogenic, or obstructive shock that induces rapidly progressive activation of proinflammatory innate immunity and potential diffuse tissue and bystander organ injury. As a consequence, ARDS and MODS develop relatively often in the blunt trauma patient, but rarely after pure hemorrhagic shock alone. The hypoperfusion in traumatic shock is magnified by the proinflammatory activation that occurs following the induction of shock and the release of "danger" stimuli following tissue damage.

At a cellular level, the pathophysiology of traumatic shock may be attributable to the release of cellular products termed *damage-associated molecular patterns* (DAMPs; eg, mitochondrial DNA, ribonucleic acid, uric acid, high mobility group box 1 [HMGB1]) that activate the same set of cell surface receptors as bacterial products, initiating similar cell signaling.[7,32] The receptors are termed *pattern recognition receptors* (PRPs) and include the toll-like receptor (TLR) family of proteins. In models of traumatic shock, the addition of a soft tissue or long bone injury to the hemorrhage produces lethality with significantly less blood loss than when animals are stressed by hemorrhage alone.

Therapy for this form of shock is focused on correction of the individual derangements to diminish the cascade of proinflammatory activation contributing to its progression. Therapeutic maneuvers include prompt control of hemorrhage, adequate volume resuscitation to correct oxygen debt, stabilization of bony injuries, early debridement of nonviable tissue (including amputation as necessary), and appropriate treatment of soft tissue wounds.

END POINTS IN RESUSCITATION OF THE TRAUMA PATIENT

Most clinicians would agree that heart rate, systemic arterial blood pressure, skin temperature, and urine flow provide relatively little information about the adequacy of oxygen delivery to tissues. Physiologically, shock begins when DO_2 falls below the tissue VO_2 requirements. A persistent mismatch between the DO_2 and VO_2 has been associated with progressive MODS. Unfortunately, there are major limitations in our ability to quantify this mismatch and assess perfusion status. Due to these limitations, it is necessary to use surrogates of tissue hypoxia. During anaerobic metabolism, large quantities of pyruvate are converted to lactate rather than being recycled by entering the tricarboxylic acid cycle, and substrate-level phosphorylation of ADP to ATP causes a net accumulation of protons. Increases in blood lactate concentration are often evidence of an increase in the rate of anaerobic metabolism. Numerous studies have documented that high blood lactate levels (≥2 mmol/L) portend an unfavorable outcome in patients with shock, but it has not been proven that survival is improved when therapy is titrated using blood lactate concentration as an end point. Serum lactate levels are also affected by hepatic and renal clearance, both of which can be depressed due to preexisting comorbidities or prolonged tissue dysoxia. BD has been shown to have prognostic value in patients with shock.[194] However, it remains to be proven that titrating therapy to a BD end point improves survival.

There are numerous parameters that have been used as surrogates to measure intravascular volume as a surrogate for adequate resuscitation, including CVP. Similarly, the inferior vena cava diameter measured by ultrasound has been shown to correlate with the CVP and to predict fluid responsiveness in critically ill patients.[195] One study found that patients with an inferior vena cava diameter of less than 2 cm were almost uniformly volume responsive compared to those with inferior vena cava diameters of 2 cm or greater.[196] However, titration

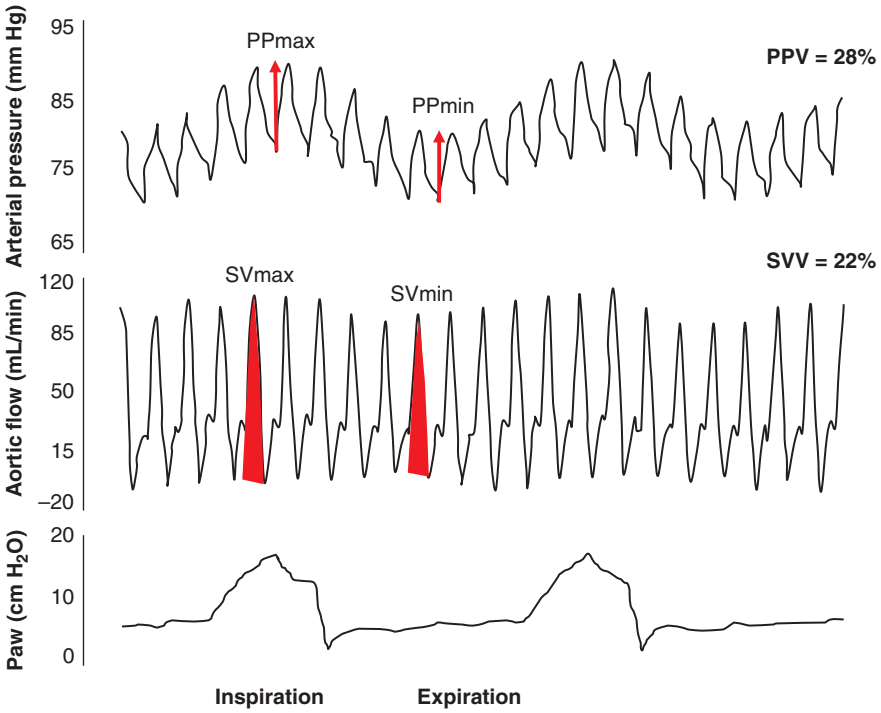

FIGURE 15-8 Pulse contour analysis. Paw, airway pressure; PPmax, maximum pulse pressure; PPmin, minimum pulse pressure; PPV, pulse pressure variation; SVmax, maximum stroke volume; SVmin, minimum stroke volume; SVV, stroke volume variation.

of resuscitation to CVP or inferior vena cava diameter has not been shown to improve survival.

Cardiopulmonary interactions are responsible for pulse pressure variation (PPV), which is a surrogate for stroke volume variation (SVV) (Fig. 15-8). Pulse pressure is the difference between the systolic and diastolic blood pressures at any given point in time as measured in mm Hg. PPV is the difference in pulse pressure that occurs over a respiratory cycle and can be monitored by examining the alterations of the arterial waveform in patients who are mechanically ventilated, not spontaneous breathing, and in sinus rhythm. In mechanically ventilated patients with controlled respiratory effort (ie, not overbreathing the ventilator, synchronous respirations), the positive pressure delivered with each breath forces blood from the lungs to the left heart, effectively increasing preload and stroke volume. Simultaneously, the positive intrathoracic pressure decreases venous return to the right heart, lowering right heart preload and stroke volume. The pulmonary transit time of the smaller right heart stroke volume accounts for the short delay in decreased left heart stroke volume that is manifest during exhalation. PPV increases in states of hypovolemia where decreased intrathoracic blood volume and ventricular filling pressures are more sensitive to changes in intrathoracic pressure that occur during a respiratory cycle with positive-pressure ventilation.[197] A PPV of 13% has been shown to discriminate between patients in whom additional volume will increase their cardiac index (responders) and those that will not (nonresponders) with a sensitivity and specificity of 94% and 96%, respectively.[198] A recent meta-analysis of PPV in septic shock reported a pooled sensitivity of 0.72 (95% confidence interval [CI], 0.61–0.81) and a specificity of 0.91 (95% CI, 0.83–0.95) in predicting fluid responsiveness.[199] However, resuscitation using an end point of a PPV of less than 13% has not been shown to improve meaningful outcomes in critical illness.

Arterial waveform analysis is a noninvasive way to measure cardiac output (CO) and further characterize PPV and SVV in critically ill patients. CO, PPV, SVV, and other hemodynamic parameters can be measured continuously with a minimally invasive lithium dilution CO (LiDCO) or pulse contour analysis CO (PiCCO or FloTrac). These systems use a pulse contour CO algorithm to estimate CO and systemic vascular resistance in the absence of invasive techniques. There is some evidence that the information garnered from such devices is most useful at a single point in time; when used for continuous assessment, the serial values show limited agreement or reproducibility.[200] In small clinical trials, devices that use arterial waveform analysis have shown some promise, but large multicenter trials looking at patient outcomes have yet to be reported.[201]

Near-infrared spectroscopy (NIRS) offers continuous, noninvasive bedside monitoring of tissue oxygenation. It measures oxygenation in the tissue's microvasculature and, thus, not only examines the adequacy of tissue perfusion, but also provides a window to noninvasively study tissue metabolism. In the clinical setting, NIRS has been used for continuous monitoring of metabolic variables including tissue oxygen availability, tissue oxygen consumption, tissue oxygen saturation (StO_2), and changes in StO_2 in diverse populations of patients (eg, injury, sepsis, heart failure).[194,202] However, the exquisite sensitivity of the technique and the rapid and

labile nature of peripheral perfusion in critically ill patients result in poor reproducibility; thus, the clinical utility of the technology has not been realized.

Recent reviews suggest that our lack of understanding of the effects of shock and resuscitation stem from a discrepancy between the need to identify effective strategies aimed at restoring normal oxygen delivery and the fact that most resuscitation research is aimed at controlling inflammation and coagulopathy. Although the degree of activation of the inflammatory mechanisms and coagulation derangements are directly related to the magnitude of the hypoxia-induced tissue injury, it is naïve to believe that by simply correcting oxygen delivery or modulating a specific pathway we could prevent the already established physiologic derangements and ensuing multiple organ injury.

Until more practical and quantitative methods are introduced to measure the cumulative effect of the oxygen deficit and subsequent host response, it is likely that we will continue to use a combination of surrogates of tissue perfusion to diagnose shock and guide resuscitation.

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16

Trauma-Induced Coagulopathy

Hunter B. Moore • Ernest E. Moore

KEY POINTS

- The pathogenesis of trauma-induced coagulopathy is multifactorial and includes impaired thrombin generation, defective fibrinogen, platelet dysfunction, and dysregulated fibrinolysis.
- Metabolic acidosis has a more significant effect on trauma-induced coagulopathy than hypothermia at clinically relevant levels.
- Fibrinolysis is the active degradation of polymerized fibrin through the co-localization of tissue plasminogen activator or urine-type plasminogen activator with the lysine avid binding plasminogen and subsequent conversion to plasmin.
- Systemic hyperfibrinolysis occurs in 10% to 15% of those requiring a massive transfusion.
- Impaired fibrinolysis (shutdown) is present in the majority of severely injured patients and may result in microvascular occlusion and organ dysfunction.
- Tranexamic acid should be used selectively and only in patients with active bleeding and severe shock (systolic blood pressure <75 mm Hg or base deficit >6) and optimally with documented hyperfibrinolysis.
- Prehospital plasma improves survival in patients with transport times that are anticipated to exceed 20 minutes.
- Goal-directed hemostatic strategies employing viscoelastic assays improve survival.
- Cryoprecipitate contains fibrinogen, factor VIII, von Willebrand factor, fibronectin, factor XIII, and platelet microparticles.
- Weight-based low molecular weight heparin and antifactor Xa–guided dosing are advantageous in the prevention of venous thromboembolism.

INTRODUCTION

Despite advances in emergency medical systems (EMS) and trauma care, deaths from injury have increased in the United States over the past decade.[1] In both the civilian[2] and military[3] settings, uncontrolled hemorrhage is the leading cause of preventable death after injury. In civilian studies, more than 95% of deaths from hemorrhage occur within the first 24 hours at a median time of less than 3 hours.[2] Consequently, there is intense interest worldwide in the pathogenesis of trauma-induced coagulopathy (TIC) and its early management. Although there have been substantial insights, the words of Mario Stefanini in his address to the New York Academy of Medicine in 1954[4] remain applicable today:

> *The ponderous literature on the subject of hemostasis could perhaps be considered a classical example of the infinite ability of the human mind for abstract speculation. For several years, the number of working theories of the hemostatic mechanism greatly exceeded and not always respected the confirmed experimental facts. In recent years, however, the revived interest in this field has led to an accumulation of new findings which has been almost too rapid for their orderly incorporation into a logical working pattern. As a result, we have rapidly gone from a state of orderly ignorance to one of confused enlightenment.*

Although transfusion medicine has undergone enormous development over the past 60 years since the challenge issued by Stefanini, important gaps in scientific knowledge persist, and several fundamental issues involving the diagnosis and management of TIC remain unclear. The more we learn about TIC, the more we appreciate the contributions of surgical scientists from the past century who had the insight to form the basis of our current understanding of trauma-related bleeding. This chapter will review important historical landmarks in our evolution of understanding TIC, synthesize recent investigations into the pathophysiology underlying these processes, and provide a rationale for current diagnostic and resuscitation strategies in these patients.

HISTORICAL PERSPECTIVE

The evolution of our understanding of the complexities of TIC has been, in large part, the result of collaboration between civilian and military teams. The early reports of TIC were generated from military research teams, often including civilian consultants, during major wars. These novel observations intensified hemostasis research in civilian centers. Ultimately, the resulting findings improved coagulopathy management in subsequent conflicts and primed the civilian environment for making new observations. The specific contributions to our understanding of TIC, however, are somewhat difficult to ascertain from World War I through Vietnam because the primary focus was on optimizing shock resuscitation at a time when plasma or whole blood was employed to replace acute blood loss.[5] Nonetheless, several landmark contributions are well recognized.

In 1916, the US National Research Council formed a subcommittee on traumatic shock that collaborated with the British Medical Research Committee to study wounded soldiers in the front lines of France. Among them was Walter B. Cannon, MD, who was perplexed by the inconsistencies of the prevailing toxin theory of shock. Cannon documented experimentally that stress (ie, epinephrine infusion into animals) provoked hypercoagulability followed by hypocoagulability. Cannon stated that "shock is a loss of homeostasis, and without homeostasis the patient does not survive."[6] During World War II, experts recognized that bottled whole blood would be logistically impractical and enlisted the expertise of Edwin J. Cohen, a biochemist, to deconstruct blood to enable components to be used on the battlefield.[7] Cohen was successful in purifying albumin as well as preparing plasma. At the onset of World War II, the National Research Council's Committee on Transfusion recommended that dried plasma—not blood—would be used if combat occurred outside the continental United States because it was easy to prepare and transport, whereas whole blood had to be typed, cross-matched, and refrigerated. Based on the legendary work of consultant Edward D. Churchill, MD,[8] who concluded that "wound shock is blood volume loss," the policy was changed to whole blood administration and implemented in 1943.

During the 1950s, civilian investigators recognized that impaired coagulation was associated with severe injury; the culprits were believed to be fibrinolysis,[9] loss of labile clotting factors,[10] disseminated intravascular coagulopathy (DIC),[11] and platelet dysfunction.[12] The labile factor depletion hypothesis was supported during the Korean War. Scott and Crosby[13] reported that the prothrombin time (PT) was doubled in combat casualties, while platelet count and fibrinogen were increased. Artz and Fitts[14] observed that severely injured soldiers in the Korean conflict required both return of shed blood and crystalloid for optimal survival. The recognition of the high incidence of acute renal failure further stimulated the classical experimental work of Shires et al,[15] who demonstrated the intracellular accumulation of sodium in hypoxic cells. These studies ultimately led to the routine practice of initial crystalloid loading for shock resuscitation.[16] During the Vietnam War, crystalloid resuscitation was used to such excess that it led to acute lung dysfunction, similar to the civilian description of acute respiratory distress syndrome.[16]

The factor depletion component of coagulopathy was proposed by Simmons et al[17] and attributed to a DIC-like phenomena. During the 1970s, coagulation research in civilian centers began to accelerate; the recurring theme was trauma patients with liver injury bleeding to death despite control of mechanical hemorrhage. From these clinical observations, the concept of preemptive plasma resuscitation was introduced[18,19]: "If the patients remain hypotensive after the second unit of blood—FFP [fresh frozen plasma] should be administered then and with every fourth unit thereafter" and "fresh whole blood should be given if bleeding persists despite normal PT, PTT [partial thromboplastin time], and bleeding times."[19] The bloody viscous cycle, later known as the lethal triad, was introduced in 1981 as it was observed that refractory bleeding was associated with hypothermia, acidosis, and coagulopathy, and experimental work supported this construct.[20] The frequent recognition of this scenario led to the concept of damage control resuscitation in which definitive repair of injuries was delayed in favor of optimizing the patient's physiologic status.[21] Coagulation research during this period was further complicated by the practice of aggressive crystalloid resuscitation targeting supraphysiologic oxygen delivery. This resulted in an epidemic of compartment syndromes, and in retrospect, much of the coagulopathies were generated by clotting factor dilution with overzealous infusion of crystalloid.[22]

The first decade of the 21st century provided perhaps the most significant insights into TIC in modern history. Progress was unquestionably inspired by the revolutionary concept of the cell-based model of coagulation proposed by Hoffman and Monroe,[23] who emphasized the fundamental role of platelets as a platform for clotting factor assembly and thrombin generation on damaged endothelium. In 2003, MacLeod et al[24] from the University of Miami and Brohi et al[25] from the Royal London Hospital independently reported that 25% of severely injured patients had prolonged plasma clotting times, and this abnormality was independent of fluid administration. Consequently, the London group termed the syndrome the *acute coagulopathy of trauma* (ACOT).[25] Stimulated by his observations of ACOT, Brohi pursued a trauma research fellowship with Cohen, Pittet, and colleagues in San Francisco. Together, in 2007, this civilian research team provided evidence suggesting that activation of protein C was an integral component of ACOT.[25] Simultaneous with these provocative civilian findings, the US military analyzed combat casualties in Iraq and concluded that uncontrolled hemorrhage was the predominant source of preventable death.[3] Armand and Hess[26] suggested the solution was to replace the lost blood with component therapy that replicated the composition of whole blood, initiating the concept of 1:1:1 (fresh frozen plasma [FFP]:red blood cells [RBCs]:platelets). Remarkably, in the same year as the proposed protein C hypothesis, Borgman et al[27] reported that resuscitation with FFP:RBC approaching 1:1 improved combat survival. These

landmark civilian and military reports prompted a literal explosion of coagulation research. Recognizing the ongoing controversies, the National Institutes of Health responded with a workshop on postinjury hemostasis in 2010 and achieved consensus in renaming this challenge *trauma-induced coagulopathy* (TIC).

Cell-Based Model of Hemostasis

Hoffman and Monroe[23] enhanced our understanding of the mechanisms of coagulation by shifting from a simple enzymatic cascade to a cell-based paradigm with tightly regulated events (Fig. 16-1). The key concept underlying the paradigm of "cell-mediated hemostasis" is that cells play active roles in regulating and localizing the coagulation reactions. Many cells participate in hemostasis and thrombosis, but two critical players are platelets and endothelial cells. This model consists of three overlapping phases: initiation, amplification, and propagation. A breach of the endothelium promotes clot formation via exposed collagen and tissue factor. Collagen localizes platelets via their glycoprotein (GP)-VI receptor. Circulating von Willebrand factor also binds collagen, and this complex further promotes platelet adherence through their GP-Ib-V-IX receptors, particularly important under high-stress flow. Tissue factor (TF) binds circulating factor VII; the resulting TF/factor VIIa complex activates factors IX and X. The initial amount of thrombin generated is insufficient to cleave fibrin, but activates platelets through their PAR-1 and PAR-4 receptors and also activates factors V and VIII. In the ensuing amplification phase, these activated factors form the prothrombinase (Va/Xa) and tenase (VIIIa/IXa) complexes on the surface of platelets. Tenase (VIIIa/IXa) generates sufficient amounts of factor Xa to support thrombin generation through prothrombinase (Va/Xa). These coagulation complexes require phospholipid and calcium for their activity. Prothrombinase and tenase then potentiate the "thrombin burst" in the propagation phase, which cleaves fibrinogen. The resulting fibrin polymerizes and integrates platelets by binding via their GP-IIb–IIIa receptors. This thrombin further generates Va and VIIIa that promote prothrombinase and tenase assembly and activate XIII, which cross-links fibrin to stabilize the evolving clot. Activated platelets release adenosine diphosphate (ADP) and thromboxane A2 that further recruit platelets to form an outer shell to stabilize the clot. The various proteins known to be active in the clotting system are summarized in Table 16-1.

DRIVERS OF TRAUMA-INDUCED COAGULOPATHY

Impaired Clot Formation (Part I)

ACTIVATED PROTEIN C

The activated protein C (APC) pathway attenuates coagulation activation, serving as an anticoagulant restraint on the multiple physiologic processes that promote clot formation. Independent of these anticoagulant properties, APC has been shown to have multiple cytoprotective effects as well, acting as an anti-inflammatory agent and preventing endothelial barrier leakage.[28] By these two mechanisms, the APC pathway serves to maintain vascular flow by preventing excessive thrombosis and also protects cells from damage associated with inflammatory insults, such as sepsis and trauma. Once activated, APC achieves its primary anticoagulant function through proteolytic cleavage of activated factors Va and VIIIa, the major drivers of thrombin formation.[29] Activation of protein C is achieved via thrombin-mediated cleavage.[30] Following injury, thrombin binds to upregulated thrombomodulin, activating the endothelial receptor for protein C, culminating in the release of protein C (Fig. 16-2). Thrombomodulin can increase this activity by 20,000-fold.[31] Thrombomodulin is a transmembrane protein that is found predominantly on the surface of endothelial cells and is heavy concentrated in the microvasculature.[32] Protein S also increases APC complex adherence to the phospholipid membrane, further enabling the inactivation of factor Va.[33] Krishnaswamy et al[34] have shown that meizothrombin released from stored RBCs can also bind to thrombomodulin and activate protein C.

As previously mentioned, data from the San Francisco general group suggested that APC has a central role in pathogenesis of TIC. The hypothesis is that this event is related to an evolved but maladaptive response to severe injury. In the setting of severe trauma and tissue hypoperfusion, an excess of protective anti-inflammatory APC is released in an attempt to prevent local microvascular thrombosis and mitigate cellular dysfunction. This hypothesis is supported by data from a trauma cohort demonstrating that poor outcomes associated with increased levels of inflammatory histones are abrogated by simultaneous increases in endogenous APC, implying a protective effect of APC in the setting of widespread inflammation.[35] The role of APC in the pathogenesis of TIC, however, remains controversial.[36]

AUTOHEPARINIZATION FROM GLYCOCALYX DEGRADATION

Shortly after the proposed role of APC in TIC, Johansson et al[37] from Copenhagen added evidence implicating endothelial glycocalyx degradation, emphasizing the endotheliopathy component of TIC. The vascular endothelium comprises a single layer of cells that line blood vessels and lymphatics in every organ, covering a surface area of 4000 to 7000 m^2 with a weight of 1 kg.[38] The luminal surface of the endothelial cells is covered by a 0.2- to 1.0-μm-thick, negatively charged, carbohydrate-rich surface layer, the endothelial glycocalyx, which represents a large component of the vascular system by containing a fixed noncirculating plasma volume of approximately 1 L in adults, corresponding to one-third of the vascular plasma volume.[38] The glycocalyx is an antiadhesive and anticoagulant structure that protects endothelial cells and maintains vascular barrier function.[39] It is bound to the underlying endothelial cells through various backbone

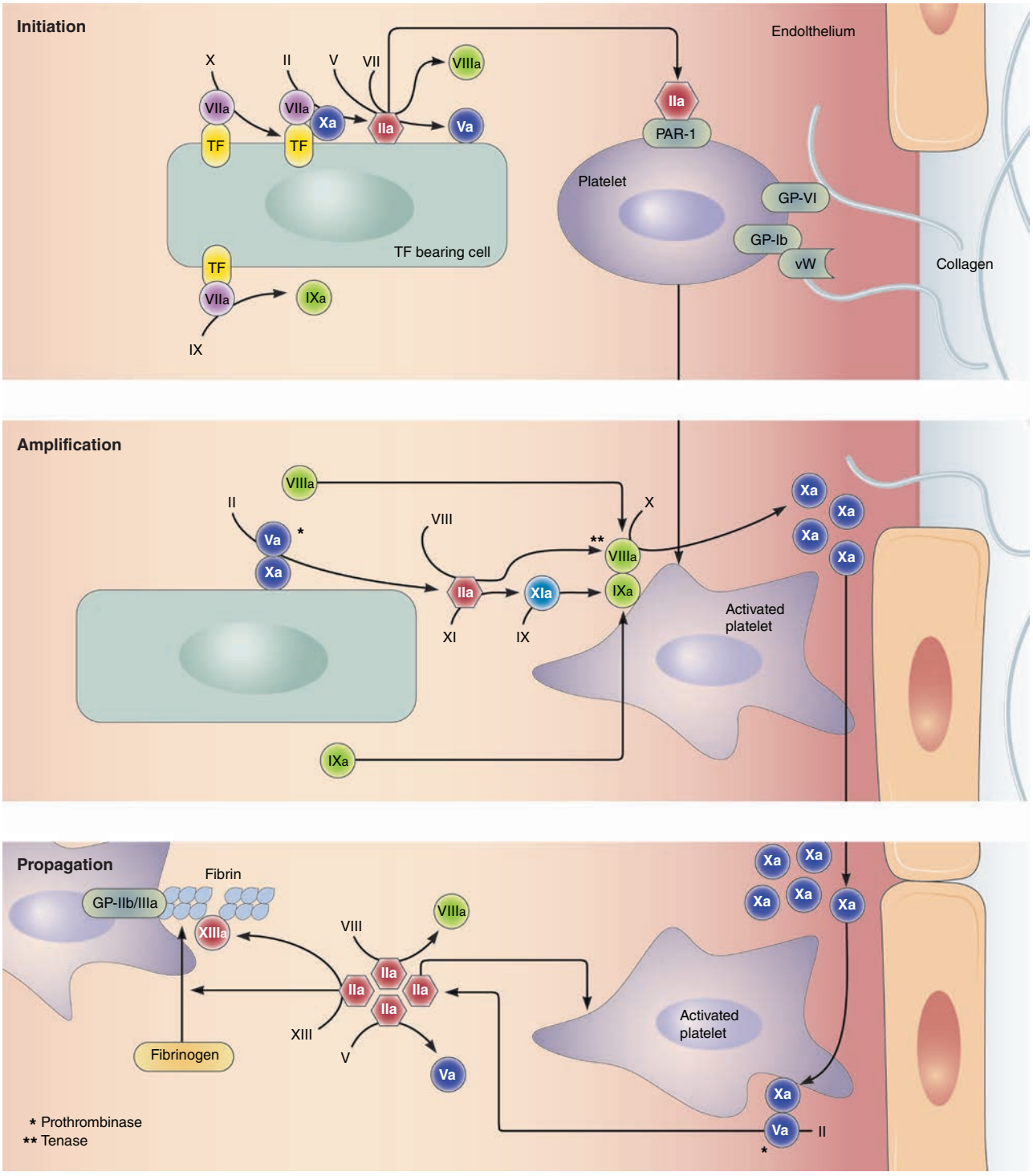


FIGURE 16-1 Cell-based model of hemostasis. Initiation is triggered by subendothelial tissue factor (TF) that binds circulating factor VII; the TF/factor VIIa complex activates factor X, generating thrombin. This initial thrombin activates platelets and factors V and VIII. During amplification, the prothrombinase (Va/Xa) complex generates additional thrombin, which activates the tenase (VIIIa/IXa) complex on the platelet surface. The tenase complex generates sufficient Xa to maintain prothrombinase-mediated thrombin, which cleanses fibrinogen into fibrin and activates factor XIII, which then cross-links fibrin to form a stable clot. (Reproduced with permission from Gonzalez E, Moore E, Moore H, et al. Trauma-induced coagulopathy: an institution's 35 year perspective on practice and research. *Scand J Surg.* 2014;103[2]:89-103.)

TABLE 16-1: Proteins Involved in Coagulation, Anticoagulation, and Fibrinolysis

Protein	Source	Activated by	Function
Procoagulant			
Tissue factor (TF)	Subendothelium, monocytes	Binding to circulating platelets	Complexes with VII to initiate clot formation
Fibrinogen (factor I)	Activated platelets	Thrombin	Clot formation
Factor V	Activated platelets	Thrombin	Cofactor that accelerates conversion of prothrombin to thrombin
Factor VII	Liver	Thrombin, Xa, XIa, XIIa	Complexes with TF to convert X to Xa Activates TAFI
Factor VIII	Endothelial cell, liver	Thrombin	Cofactor, activates factor X
Factor IX	Liver	Factor XI	Cofactor, activates factor X
Factor X	Liver	Factors VIII and IX	Converts ~ prothrombin to thrombin
Factor XI	Liver	Thrombin	Activates IX
Factor XIII	Liver	Thrombin	Crosslinks fibrin
Kallikrein	Liver		Activates contact system
Anticoagulants			
Heparan sulfate	Endothelial cells	Ischemia	Activation of ATIII
Antithrombin III	Liver	Thrombin	Inhibition of thrombin, Xa, XIa, XIIa
Protein C	Liver	Thrombin, TM, EPCR	Irreversibly inactivates Va and VIIIa
Protein S	Liver		Cofactor for protein C
Thrombomodulin (TM)	Endothelial cell	Ischemia	Complexes with thrombin to activate protein C; inhibits TAFI
Endothelial protein C receptor (EPCR)	Endothelial cell		Complexes with thrombin and TM to activate protein C
Tissue factor pathway inhibitor (TFPI)	Liver	Thrombin	Inhibits TF–VII complex from converting X → Xa
Meizothrombin	Red blood cells		Precursor of thrombin
Fibrinolytic system			
Plasminogen	Liver	t-PA	Converted to plasmin
Tissue plasminogen activator (t-PA)	Endothelial cell	Ischemia, thrombin	Converts plasminogen to plasmin
Urine-type plasminogen activator (u-PA)	Epithelium, monocytes	Kallikrein	Converts plasminogen to plasmin
Plasminogen activator inhibitor 1 (PAI-1)	Endothelium, platelets, adipocytes		Binds t-PA and u-PA
Plasminogen activator inhibitor 2 (PAI-2)	Placenta, adipocytes, monocytes		Binds t-PA and u-PA
Thrombin-activatable fibrinolysis inhibitor (TAFI)	Liver, platelets	Thrombin–thrombomodulin complex	Inhibits plasminogen binding to fibrin
α-2 antiplasmin	Liver, platelets		Binds plasmin and inactivates
α-2 macroglobulin	Endothelium, platelets		Binds plasmin and inactivates
C1-esterase inhibitor	Liver		Binds t-PA and inactivates

Platelet activation = adhesion/aggregation/degranulation/shape change

Agonists	Platelet receptors
Thrombin	PAR-1 and PAR-4
Collagen	GP1a-IIa and GPVI
von Willebrand factor	GP1ba-IX-V
Adenosine diphosphate	P2Y1 and P2Y12
Thromboxane A2	TRα and TRβ
Fibrinogen	GPIIb-IIIa
Epinephrine	
Serotonin	

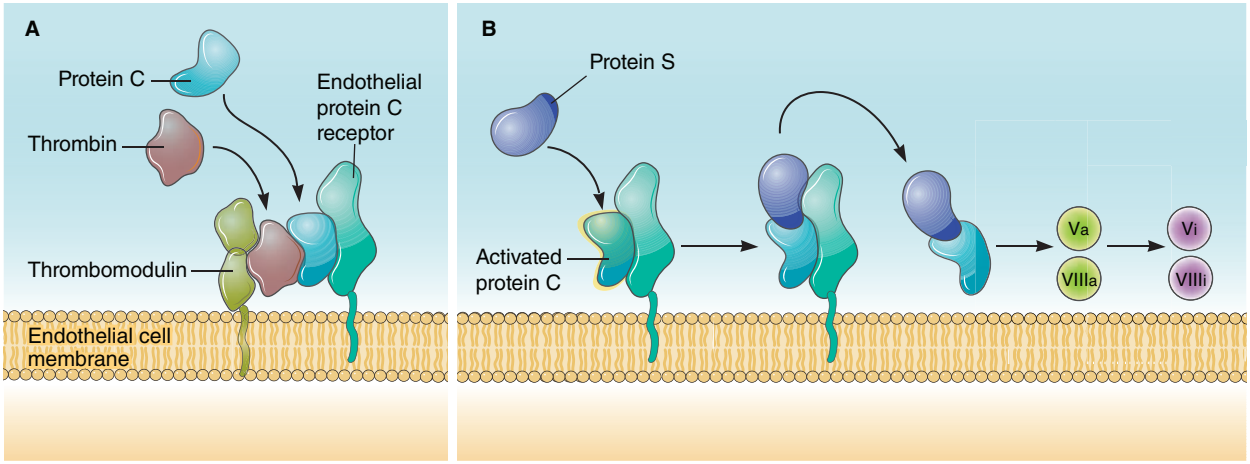


FIGURE 16-2 Protein C activation. Increased expression of the transmembrane endothelial protein thrombomodulation binds thrombin, which activates protein C (**A**). The endothelial protein C receptor localizes protein, facilitating this activation (**B**). Activated protein C cleaves peptide bonds of factor Va and factor VIIIa, preventing their assembly into the prothombinase (Va/Xa) and tenase (VIIIa/IXa) complexes.

molecules of proteoglycans (the most abundant is syndecan-1) and glycoprotein. The proteoglycans have long glycosaminoglycan side chains composed mainly of heparan sulfate that has heparin-like functions.[39] Glycocalyx damage can range from discrete disturbances in the composition of the most luminal layer to degradation with loss of the entire glycocalyx. Upon shedding, the glycocalyx glycosaminoglycans retain their anticoagulant (heparin-like) activity and promote measurable hypocoagulability in the circulating blood through endogenous autoheparinization.[39] Rehm et al[40] provided the first evidence of acute destruction of the endothelial glycocalyx in patients undergoing vascular surgery associated with ischemia/reperfusion injury. This study reported that syndecan-1 and heparan sulphate levels increased after ischemia/reperfusion injury during vascular surgery and that this increase correlated to shedding of glycocalyx as detected by electron microscopy. Despite 40- to 60-fold increases in syndecan-1 postischemia, the levels of the classical endothelial adhesion molecules ICAM-1 and VCAM-1 did not change, emphasizing the occurrence of selective glycocalyx disruption. Ostrowski and Johansson[41] translated these observations to trauma when their group demonstrated reversibility of prolonged clotting time by heparinase-thromboelastography in critically injured trauma patients. These patients had syndecan-1 levels fourfold higher than noncoagulopathic patients in this study, in addition to a higher injury severity and transfusion requirements. However, the role of glycocalyx degradation in the pathogenesis of vascular barrier function remains unclear.[42] Furthermore, the Copenhagen group has failed to translate a clinical strategy to reduce bleeding by blocking circulating glycocalyx products. The use of protamine or antiheparin in the resuscitation of trauma patients cannot be advocated at this time. Although this group continues to publish multiple papers on syndecan-1 levels and poor outcomes, they ultimately have identified a biomarker of endothelial activation/injury after trauma that is associated with poor outcomes.[43]

DIC AND CONSUMPTIVE COAGULOPATHY

The role of DIC in depleting circulating clotting factors resulting in impaired thrombin generation remains debated.[44] The distinction is that DIC occurs systemically within the vessel lumen, whereas factor consumption from disrupted endothelium exposing tissue factor and collagen is a localized process at the site of injury. Most investigators do not believe DIC is a dominant mechanism in TIC but acknowledge its potential contribution. The innate immune response to injury has been implicated in the genesis of DIC, particularly with the release of damage-associated molecule patterns (DAMPs). However, beyond increased tissue factor expression on monocytes and microparticles, the mechanism remains unclear. Among those with procoagulant implications are HMGB1, histone DNA complexes,[45] polyphosphates,[46] and myosin.[47] In addition, neutrophils are believed to contribute to a procoagulant state via degranulation of elastase, which can act as an indiscriminate protease.[48] Human neutrophil elastase mediates fibrinolysis shutdown through competitive degradation of plasminogen and generation of angiostatin.[49] With extensive tissue disruption, there is undoubtedly some element of coagulation factor consumption at the site of injury to achieve hemostasis.

HYPOTHERMIA AND ACIDOSIS

Hypothermia and acidosis were initially considered the primary drivers of TIC. Although hypothermia is usually not a major factor, acidosis may play a more important role than recognized. In vitro[50] and in vivo[51] work indicates that hypothermia affects hemostasis when the temperature is below 33°C. Below this temperature, hypothermia inhibits the initiation phase of clotting. This would be anticipated as most of the coagulation enzymes are slowed by hypothermia. However, although moderate hypothermia delays the onset of thrombin generation, the total amount of thrombin generation is unaffected.[51] The effect of hypothermia on platelet function

is contradictory due to limitation in the ability to replicate platelet function under physiologic conditions.[52] Some investigators have indicated enhanced platelet activation (priming) and aggregation,[53] whereas others indicate decreased adhesion and aggregations.[51] Profound hypothermia has long been recognized for promoting hepatic sequestration of platelets, but the clinical relevance outside of cardiac surgery is minimal, as a temperature below 30°C is needed for this phenomenon.

Metabolic acidosis has a more significant effect on TIC than hypothermia at clinically relevant levels.[54] A reduction of pH from 7.4 to 7.0 has been shown in vitro to reduce factor VIIa activity by 90%, prothrombin complex (Xa/Va) activity by 70%, and factor VIIa/TF complex activity by 55%.[55] In vitro work has confirmed a pronounced inhibition of the propagation phase of thrombin generation at a pH of 7.1, resulting in a marked reduction in clot strength.[50] These studies also suggest this degree of acidosis increases fibrinogen degradation twofold. Although impaired platelet aggregation and adhesion at a pH of less than 7 has been appreciated for decades, more recently the mechanism has been linked to the store-operated calcium entry (SOCE) channels. Perhaps more worrisome, the correction of pH with bicarbonate[50] or trishydroxymethylaminomethane (THAM)[56] does not restore platelet function. This may have implications for the optimal timing of platelet transfusion in the critically injured patient.

HEMODILUTION

Hemodilution is no longer the dominant mechanism for TIC, but overzealous crystalloid resuscitation can exaggerate an existing coagulopathy. Reduced levels of coagulation proteins have been documented in healthy volunteers after administration of crystalloids, colloids, and stored RBCs[57] and remain a fundamental rationale for permissive hypotensive resuscitation (see Chapter 15). Interestingly, acute hemodilution to 50% in vitro does not impair clot formation,[58] but this magnitude of hemodilution enhances the sensitivity to tissue plasminogen activator (t-PA) due to the dilution of endogenous antifibrinolytics.[59]

In sum, impaired clot formation is central in the pathogenesis of TIC, but the mechanism is complex and time dependent and can be exacerbated by acidosis, factor consumption, and hemodilution (Fig. 16-3).

PHENOTYPES OF TIC

With the multiple mechanisms and time dependence that drive TIC, it is not surprising that patients manifest coagulopathy, with unique phenotypes of coagulopathy warranting patient-specific management. Trauma is a combination of tissue injury and shock, and no two patients have an identical injury pattern and magnitude of shock. These variables in combination with genetic variability, preexisting medical conditions, and age-related changes in coagulation make every trauma patient unique when they arrive at the emergency department (Fig. 16-4). In the early description of TIC, two components were proposed: (1) impaired blood clot formation (hypocoagulation) and (2) increased clot degradation (hyperfibrinolysis).[60] These two processes appear to be mechanistically unique based on principal component analyses, which also implicate a third mechanistic variant of coagulopathy.[61,62] To make it more complicated, the extremes of the phenotypes can be pathologic. Recently it has been identified that trauma patients can present with a spectrum of fibrinolysis within 12 hours of injury and those patients on either extreme with excessive or impaired fibrinolysis have increased mortality.[63]

Dysregulation of Fibrinolysis (Part II)

Stafford's review on fibrinolysis and hemostasis in 1964[64] established a logical understanding for why coagulation becomes pathologic: "a general assumption has developed that clotting is not episodic but a continuous process which is normally never allowed to progress to a physical end point." In the previous section, we stressed that APC and the glycocalyx serve physiologic functions to localize thrombin-generated clot and that fibrinolysis serves a physiologic role to control clot burden. The recurring theme of biologic systems is germane to TIC; coagulation is a balance, and preventing the extremes should be the resuscitation goal (Fig. 16-5).

HYPERFIBRINOLYSIS

Although proposed in 1948,[9] during the 1960s, a number of investigators emphasized that fibrinolysis was a physiologic process to counterbalance thrombosis and was occurring perpetually.[64,65] Fibrinolysis is the active degradation of polymerized fibrin through the lysine avid binding protease plasmin.[66] The conversion of plasminogen to plasmin is accomplished by proteolytic cleavage by one of its activators (t-PA or urine-type plasminogen activator [u-PA]). Plasmin activity is highly dependent on its local environment, and its proenzyme plasminogen binds numerous receptors, indicating the process occurs on cell surfaces and not in circulation.[67] The regional distribution of specialized endothelial cells contributory to the fibrinolytic system is reflective of the importance of localizing this process.[68] The primary driver of intravascular fibrinolysis, t-PA, is released from precapillary arteriole and postvenular endothelial cells; u-PA is considered a secondary source derived from epithelial cells, but its relative importance is likely tissue specific, including the brain.[69] Release of t-PA into the circulation results in complex formation with its most abundant serine protease inhibitor, plasminogen activator inhibitor 1 (PAI-1), and secondary inhibitors such as C-1 inhibitor. These t-PA complexes are cleared by the liver.[70] There are a number of additional proteins present in plasma that either inhibit t-PA directly, inhibit plasmin, or impair plasmin access to fibrin (see Fig. 16-5). These proteins are normally in relatively high abundance in the plasma, including α-2 antiplasmin (α-2AP), α-2 macroglobulin, α-1 antitrypsin, and histidine-rich glycoprotein.[71] In addition, there are potent inhibitors integrated into the fibrin clot, including α-2AP and thrombin-activatable fibrinolysis inhibitor (TAFI), which are directly released from platelets to ensure a tight regulation of the system. TAFI cleaves lysine binding

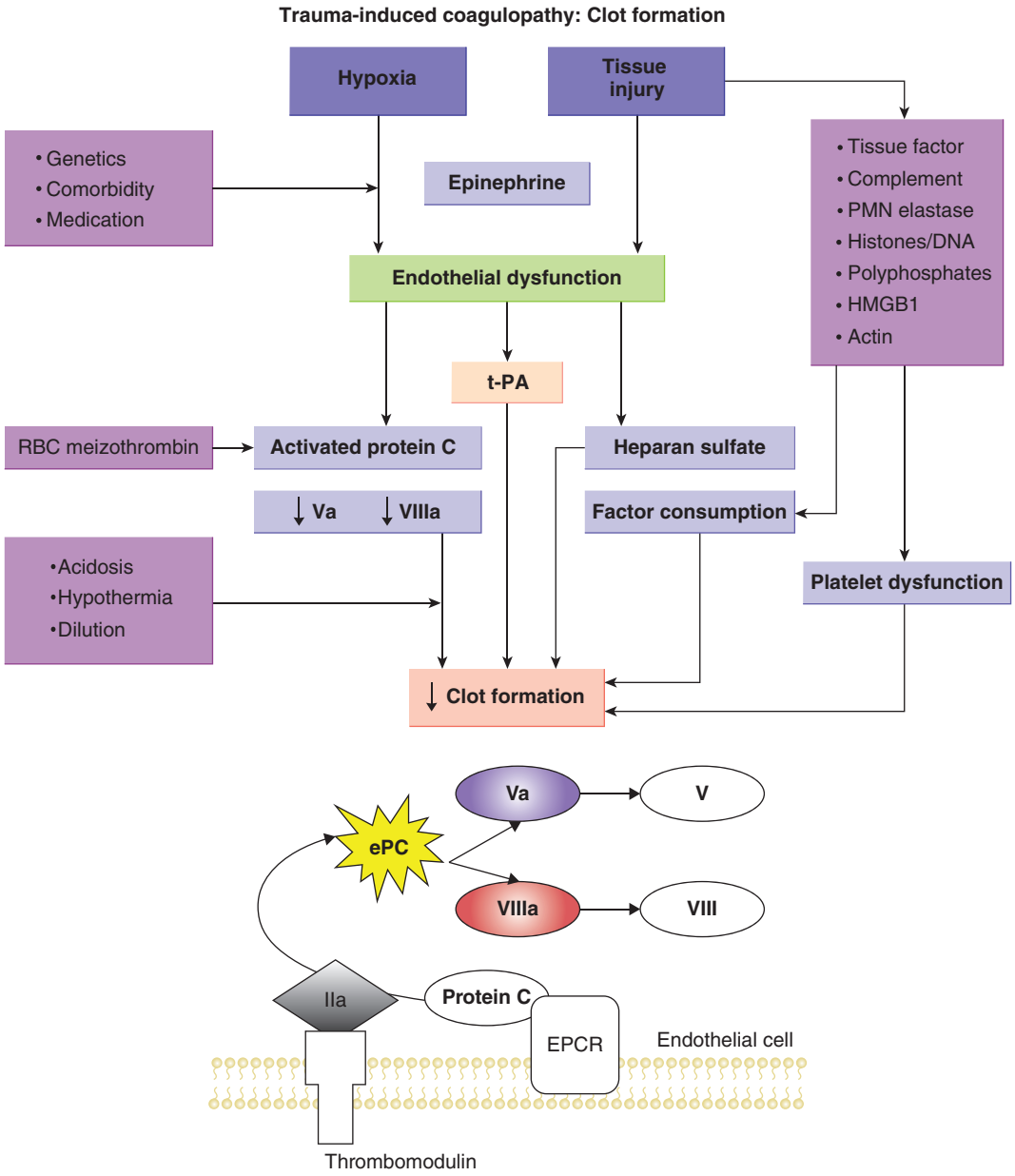


FIGURE 16-3 Ineffective clot formation is the dominant manifestation of trauma-induced coagulopathy. Hypoxia and tissue injury provoke endothelial dysfunction, which activates protein C and releases anticoagulants from the glycocalyx. Platelet dysfunction, factor consumption, and acidosis further impair thrombin generations and clot formation. EPCR, endothelial cell protein C receptor; PMN, neutrophil; t-PA, tissue plasminogen activator.

sites on fibrinogen, inhibiting plasminogen and t-PA binding. Plasma can buffer the effects of t-PA in vivo,[72] and in vitro exogenous t-PA mixed in whole blood of healthy volunteers requires supraphysiologic concentrations to reproduce hyperfibrinolysis. While fibrinolysis is constitutively occurring at a microvascular level to maintain vascular patency, pathologic systemic hyperfibrinolysis is the result of both an increase in circulating t-PA and a reduction of its circulating inhibitors. Fibrinolysis shutdown is associated with a fivefold increase in mortality in trauma patients lacking hypersensitivity to tissue t-PA.[73]

Interest in fibrinolysis as a component of TIC was stimulated by the proposal of Brohi et al,[60] who suggested APC degraded PAI-1. However, recognition of systemic fibrinolysis following injury did not occur until the widespread use of viscoelastic assays. Recent clinical studies indicate that systemic hyperfibrinolysis occurs in 2% to 5% of critically injured patients and 10% to 15% of those requiring a massive transfusion.[74,75] In our experience, the highest rate of hyperfibrinolysis in trauma occurs in those undergoing emergency department resuscitative thoracotomy. Currently, the only established mechanistic factor for hyperfibrinolysis is inadequate tissue perfusion,

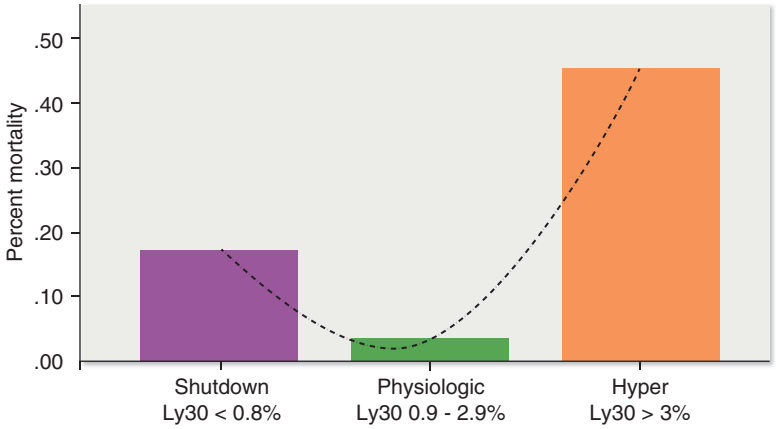


FIGURE 16-4 Systemic fibrinolysis phenotypes. The *y*-axis represents mortality, the *x*-axis is the fibrinolysis phenotype based on the LY30 measured by thromboelastography. There is a U-shaped distribution of mortality, with a nadir evident in the physiologic group. LY30 above (hyperfibrinolysis) and below (fibrinolysis shutdown) this range had increased mortality. (Reproduced with permission from Moore H, Moore E, Gonzalez E, et al. Hyperfibrinolysis, physiologic fibrinolysis, and fibrinolysis shutdown: the spectrum of postinjury fibrinolysis and relevance to antifibrinolytic therapy. *J Trauma Acute Care Surg.* 2014;77:811-817.)

suggested by a number of retrospective studies[63,75] and further strengthened by the fact that the only patients in the CRASH II trial who benefited from antifibrinolytics had a systolic blood pressure of less than 75 mm Hg.[76] Supporting this observation is that blood taken from patients who have nontraumatic cardiac arrest has a high prevalence of hyperfibrinolysis.[77] Shock may also generate metabolic disturbances that enhance fibrinolysis. For example, taurocholic acid increases fibrinolysis in vitro and is markedly elevated following shock.[78] Recent animal work indicates that t-PA levels increase substantially in animals undergoing hemorrhagic shock, whereas tissue injury does not increase systemic t-PA levels.[79]

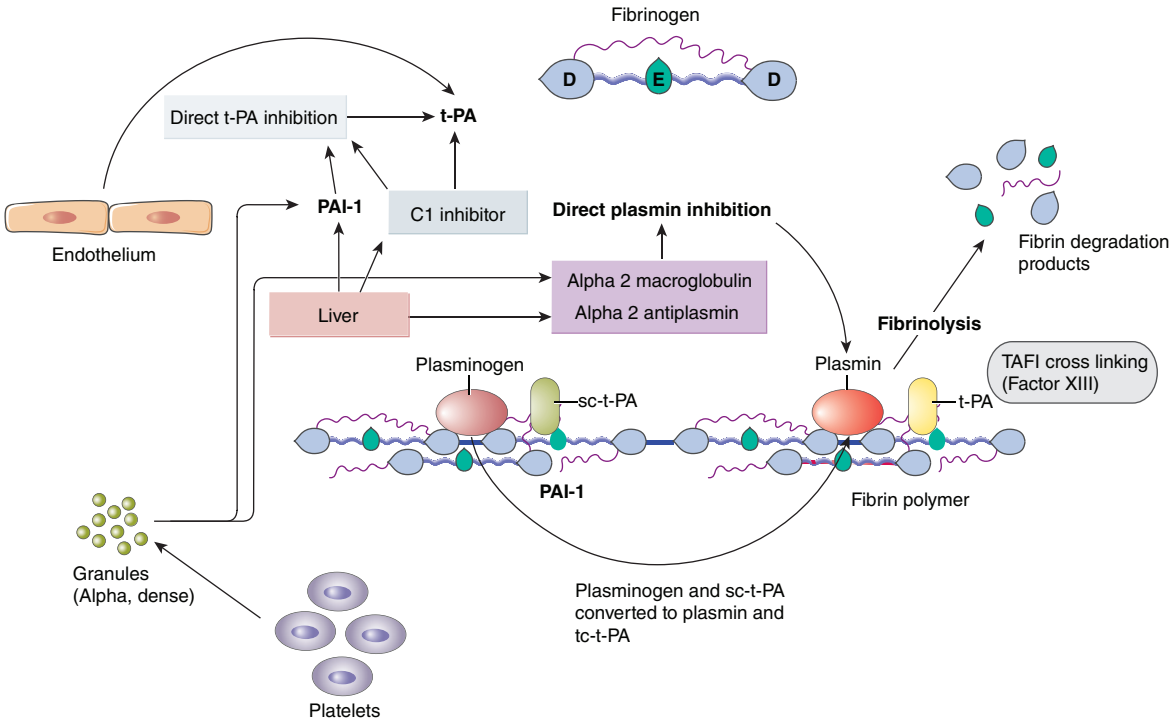


FIGURE 16-5 Fibrinolysis regulation. Systemic fibrinolysis is controlled conceptually at three levels. The first is direct inhibition of the predominant plasminogen activator, tissue plasminogen activator (t-PA). The major inhibitor is the serine protease plasminogen activator inhibitor 1 (PAI-1). The next is direct inhibition of plasmin, with antiplasmin the major circulating factor. The third is access to fibrin for degradation via factors that stabilize the clot (factor XIIIa) or prevent plasmin binding to fibrin (thrombin-activatable fibrinolysis inhibitor [TAFI]). (Reproduced with permission from Moore H, Moore E, Gonzalez E, et al. Postinjury fibrinolysis shutdown: rationale for selective tranexamic acid. *J Trauma Acute Care Surg.* 2015;78:S65-S69.)

FIBRINOLYSIS SHUTDOWN

The mortality rate of injured patients with impaired fibrinolysis (shutdown) has been reported to be nearly four times greater than that of patients with a physiologic level of fibrinolysis.[63] Death is primarily from organ failure, attributed to microvascular fibrin deposition. The term *fibrinolysis shutdown* was first used in 1969[80] in a review describing the effects of electroplexy, myocardial infarction, and elective surgery on fibrinolysis, but was originally described by Innes and Sevitt in 1964.[81] Animal work prior to this time suggested microemboli in visceral small vessels led to irreversible shock that was later found to be survivable with a profibrinolytic.[82] Hardaway et al[83] further translated these findings to humans and implicated trauma and shock in producing microvascular occlusion. Confusion arises as the nomenclature of describing impaired removal of vascular thrombi is often referred to as either the syndrome of DIC or pathologic impairment of fibrinolysis (shutdown). As mentioned previously, some investigators consider DIC to have two distinct phenotypes; that is, hyperfibrinolysis and fibrinolysis shutdown,[44] which is somewhat difficult to reconcile because diffuse intravascular thrombus should not exist in the presence of systemic hyperfibrinolysis.

It is well known that trauma patients are prone to thrombotic events. The prevalence has been reported to approach 60% with Duplex screening for postinjury venous thrombosis.[84] There is evidence for thrombosis in the pulmonary vasculature in nearly one in four seriously injured patients within 48 hours of their injury.[85] Furthermore, microvascular clot has been implicated in organ dysfunction.[86] Therefore, it is intuitive that maintaining adequate fibrinolysis to clear the microvasculature of excessive fibrin deposition would be beneficial.

In 1991, Enderson et al[87] described that the majority of multisystem trauma patients in their study had elevated D-dimers but low fibrinolytic activity. Subsequently, Raza et al[88] showed minimal fibrinolysis activity measured by rotational thromboelastometry (ROTEM) and high plasmin-antiplasmin (PAP) complexes in 57% of their patients. It is important to recognize the circulating half-life of PAP and D-dimer exceed 12 hours.[89] Raza et al[88] misinterpreted their data to question the validity of viscoelastic test for fibrinolysis, which the London group appears to remain confused about,[90] as they propose elevated D-dimer levels as evidence of active "occult fibrinolysis." Although D-dimer and PAP levels after injury correlate to poor outcomes, it is important to remember that these are measurements of what has occurred over the past 12 hours in the patient, and at one point the fibrinolytic system was activated, but a one-time plasma level does not accurately reflect the patient's current systemic fibrinolytic activity.

The seemingly paradoxical condition of elevated PAP or D-dimer levels has also been described in the United States[91] and Canada.[92] These patients have elevated mortality that occurs days after injury,[90,91] but they require early blood product resuscitation,[91] indicating that fibrinolysis shutdown is not a universally hypercoagulable state. In contrast, patients who manifest hyperfibrinolysis require significantly more blood products, and the majority of deaths occur within 24 hours of admissions.[90] Recent evidence supports that trauma patients with low fibrinolytic activity on presentation to the hospital classified as fibrinolysis shutdown can be stratified into subphenotypes.[73] One subphenotype has elevated D-dimer levels and can harbor additional coagulation derangements that can cause bleeding, whereas the other subphenotype has minimal bleeding and delayed mortality from head injury. Regardless of the subphenotype of shutdown, antifibrinolytic use has not been shown to improve clot strength when tested ex vivo.[93]

The transition from a wide range of fibrinolysis activity to fibrinolysis resistance within hours of injury has been observed for several decades[81,94] and further verified within the past 5 years.[95] These transitions make research and treatment challenging, as patients can shift between different states of fibrinolytic activity from injury through resuscitation. Although single blood draws can capture ephemeral fibrinolytic states, delays from injury to blood sampling increase the probability of detecting low fibrinolytic activity by viscoelastic hemostatic assay. This is evident in several studies where prevalence of shutdown ranges from 22% with field draws[73] to 58% with samples from the first 2 hours[90,96] to as high as 65% when the first sample is obtained up to 12 hours from injury.[63] Comparing similar patient populations at similar times from injury is essential for advancing our understanding of fibrinolysis following injury. It is likely that a significant number of patients in the reports by Moore et al,[63] Meizoso et al,[96] and Gall et al[90] were misclassified as being in acute shutdown due to the timing from injury to blood draw as samples obtained within an hour of injury indicate that the prevalence is 20% to 40%.[92,97-99]

Decades of research have demonstrated a transition to fibrinolysis resistance (not necessarily shutdown) several hours after injury or elective surgery,[80,81,94,100,101] which is thought to be an acute phase response via PAI-1 upregulation.[94] In a study of hyperfibrinolytic trauma patients who did not receive antifibrinolytics, fibrinolysis was significantly inhibited within 4 hours of injury, correlating to a several thousand–fold PAI-1 upregulation during this time frame.[95] With an endogenous transition to increased fibrinolysis resistance with PAI-1 production following resuscitation, differentiating pathologic early fibrinolysis shutdown from a physiologic acquired shutdown becomes difficult. Tissue injury appears to play a role in impairing fibrinolysis in animal models,[79,102,103] which is consistent with the association of blunt injuries with acute shutdown.[98] This endogenous impairment of fibrinolysis followed by a PAI-1 surge after resuscitation could be responsible for persistent shutdown.

The duration of shutdown following resuscitation may be the critical insult leading to organ injury and thrombotic complications. Specifically, fibrinolysis activation followed by a delayed upregulation of inhibitors would be an expected physiologic response to prevent excessive bleeding but at the cost of increased thrombotic risks. Although upregulation of inhibitors after activation is appreciated in thrombolytic

therapy, PAI-1 levels become upregulated in the post-lytic period.[104] This elevation of PAI-1 after reperfusion is presumably a physiologic response, since the prevalence of low fibrinolytic activity between 4 and 6 hours after injury exceeds 80%, yet only patients with persistently low fibrinolysis at 24 hours from injury have adverse outcomes.[95] A study from Miami suggests an eightfold mortality increase in trauma patients who retain low fibrinolytic activity for 7 days following injury compared to patients who recover from shutdown.[97] Similarly, persistent fibrinolysis shutdown following resuscitation has also been associated with adverse outcomes in pediatric trauma patients.[105] The mechanism for persistent fibrinolysis shutdown is presumed to be related to PAI-1. Prospective observation data in patients with sepsis show improved survival with recovery of fibrinolytic activity associated with decreases in PAI-1 levels.[106]

In sum, enhanced clot degradation (hyperfibrinolysis) (Fig. 16-6) can be a major component of bleeding in TIC, but the incidence is relatively low. On the other hand, impaired fibrinolysis (shutdown) is present in the majority of severely injured patients and may result in microvascular occlusion and organ dysfunction.

Platelet Dysfunction (Part III)

PLATELET ROLE IN HEMOSTASIS

Platelets are anucleated cell fragments from megakaryocytes (MKs). Maturation of platelets occurs in two phases.[107] The first phase is MK maturation in the bone marrow that takes several days. In the second phase, mature MKs grow pedicles that project into the sinusoids of bone marrow blood vessels and fragment into circulation as platelets, a process that only requires several hours. The circulating count of platelets in normal subjects is around 250,000/mcL with a half-life of 9 days, representing a turnover rate of 35,000 platelets per day.

Successful hemostasis requires the rapid accumulation of platelets to occlude the site of blood extravasation (Fig. 16-7). In animal models, platelets can seal a small wound in approximately 30 seconds and can form a fibrin-rich core within minutes.[108] Contracted platelets free of fibrin have decreased permeability. This work suggests that activated platelets serve as the primary sealant, with fibrin acting as a secondary stabilization of the platelet plug. In addition, platelets form a complex around fibrin clots that provides a unique microenvironment that is both prothrombotic and antifibrinolytic.[109] Furthermore, platelets have multiple roles in initiating and maintaining hemostasis due to their myriad of receptors and complex granule contents. The interplay of platelet auto and paracrine signaling from release of granules and the local environment optimize clot formation and resist degradation.

Effects of Trauma on Platelet Dysfunction

Endogenous activators of platelets have been well characterized in both the arterial and venous systems, with a focus on collagen and thrombin in the inner core of a platelet hemostatic plug and platelet-released ADP and thromboxane for

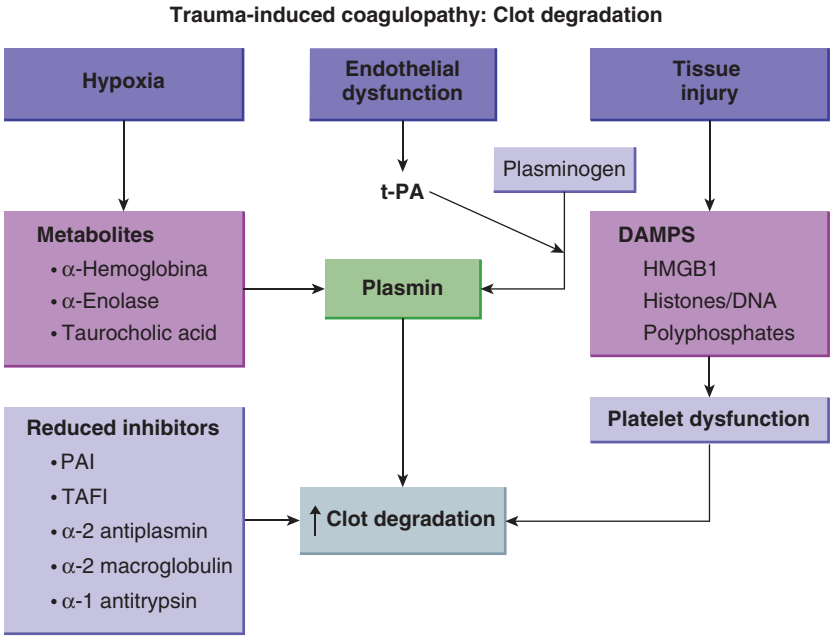
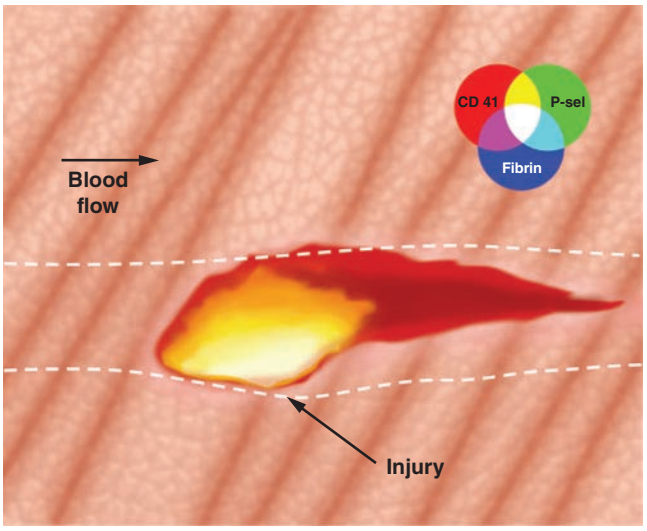
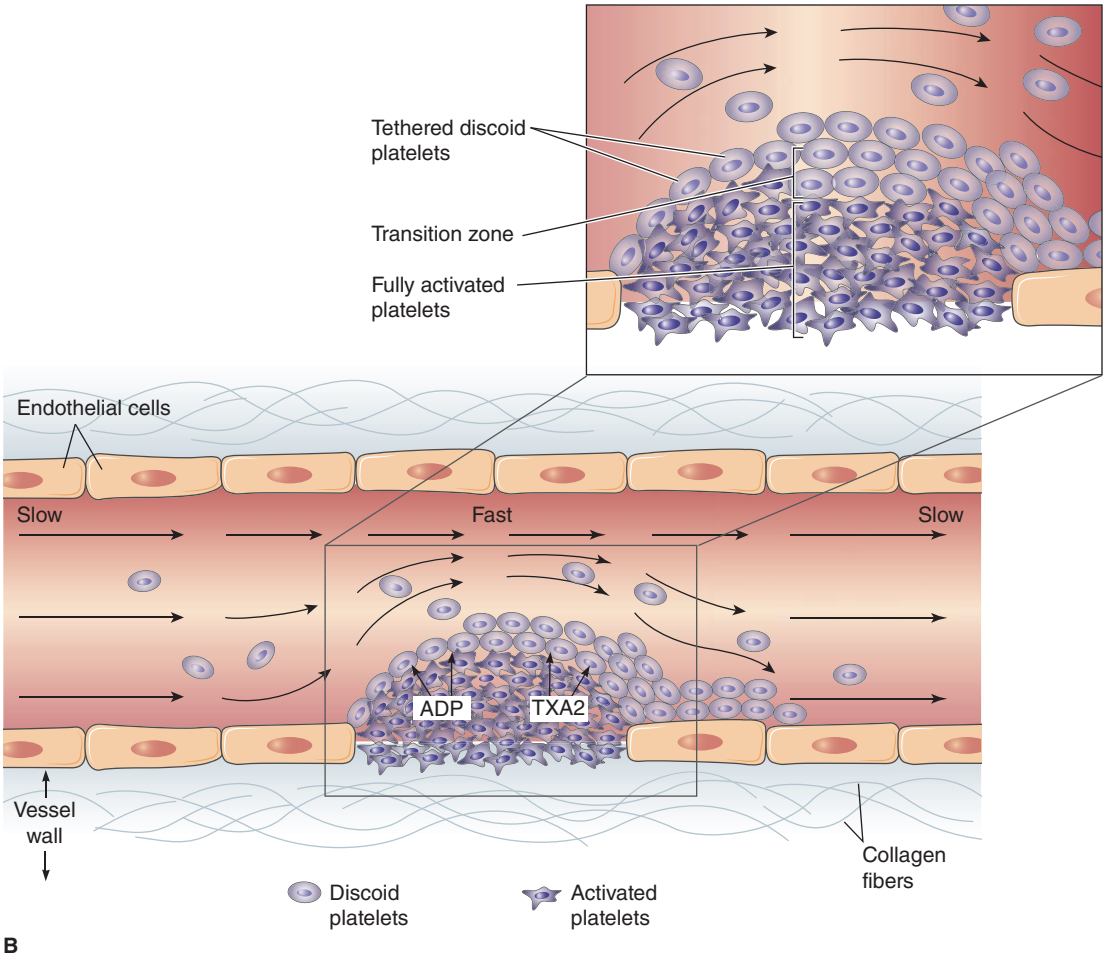


FIGURE 16-6 Clot degradation. Degradation of the clot is an important component of trauma-induced coagulopathy in some patients, particularly those with hypoperfusion. Degradation is primarily via the action of plasmin on fibrin, although platelet dysfunction can contribute to clot instability. The mechanisms responsible for postinjury fibrinolysis are complex and include the depletion of circulating antifibrinolytic agents. DAMPs, damage-associated molecule patterns; PAI-1, plasminogen activator inhibitor 1; TAFI, thrombin-activatable fibrinolysis inhibitor; t-PA, tissue plasminogen activator.



A



B

FIGURE 16-7 Role of platelets in hemostatic thrombus formation. (**A**) Illustration of a laser-induced injury in a mouse cremasteric arteriole, showing platelet and fibrin accumulation. The injury site is indicated, and dashed white lines outline the vessel wall. Platelets are labeled with an anti-CD41 antibody (red), and platelets that have released their alpha granules are labeled with anti-P selection (P-sel) antibody. Fibrin is labeled with an antifibrin antibody (blue). White represents overlap. (Image used with permission of Timothy J. Stalker, PhD, University of Pennsylvania.) (**B**) The drawing illustrates platelet incorporation into an assembled hemostatic clot. ADP, adenosine diphosphate; TXA2, thromboxane A2.

the outer core. However, the precise roles of these platelet activation pathways during TIC are not well defined. Interestingly, the TIC patient is more likely to display platelet dysfunction as opposed to platelet consumption.[110,111] Platelet dysfunction occurs early after injury and has been documented with isolated traumatic brain injury.[112] In the context of TIC, the molecular definitions of the desensitized, stunned, inactive, postactivated, dysfunctional, degranulated, exhausted, or zombie platelets are still emerging. For example, DAMPs have been shown to bind the toll-like 4 (TL4) receptor of platelets, leading to platelet dysfunction–related coagulopathy.[113] Circulating platelets may experience combinations or sequential exposures of agonists to trigger modes of activation as indicated by shape change, degranulation, and receptor expression, activation, or shedding.[114] As mentioned earlier, the optimal timing of platelet transfusion in the severely injured patient remains unclear as platelets may be rendered dysfunctional in a metabolically suboptimal environment. Furthermore, unnecessary platelet transfusion may result in adverse consequences.

COAGULATION ASSESSMENT

Traditional Laboratory Tests

Lack of an accurate tool to identify and track coagulopathy remains a major limitation surrounding both postinjury hemostatic derangements and empiric blood component replacement therapy. Traditional laboratory tests of coagulation function, such as PT and partial thromboplastin time (PTT), are plasma based and designed originally for the assessment of coagulation function with isolated factor deficiencies in hemophiliacs and, later, for the effects of warfarin. To date, the performance characteristics of these tests in the trauma patient are questionable, and they do not appear to represent clotting factor deficiency.[115]

Furthermore, because both the PT and PTT are performed on platelet-poor plasma, they are sensitive only to the earliest initiation of clot formation (estimated to represent 5% of the thrombin generation during clot formation) and do not incorporate cellular contributions, including platelets, red cells, and neutrophils. Finally, these tests are performed in an artificial environment, irrespective of the patient's core body temperature and pH. Measurements of individual pro- and anticoagulant factors, such as protein C, are both costly and time consuming. Diagnosis of fibrinolysis is also problematic. The euglobulin lysis time is a complex and time-consuming procedure that requires more than 180 minutes, reduces some of the inhibitors, and does not include platelets that regulate fibrinolysis. Other techniques suggested to identify hyperfibrinolysis, such as plasmin–antiplasmin complex, fibrin monomers, and D-dimers, are only reflective of the footprint of fibrinolysis; that is, due to their prolonged half-lives, they do not represent the patient's current systemic fibrinolytic activity.[89] Thus, partitioning the components of a patient's blood and testing them independently in an artificial environment that requires a lengthy assay time is not clinically optimal to manage coagulopathy in the critically injured patient.

Viscoelastic Hemostatic Assays

In response to the shortcomings of conventional measurements of coagulopathy, point-of-care, viscoelastic, hemostatic assays are emerging as the standard of care for both the diagnosis and treatment of postinjury coagulopathy in many trauma centers throughout the world. Viscoelastic assays quantify whole blood clot formation and degradation based on the resistance created by liquid blood solidifying and degrading after reaching maximum strength. The time and rate to achieve output of the device correlate with coagulation factor status, fibrinogen function, and platelet function and quantify fibrinolysis. Interpretation of the two most clinically employed viscoelastic assays is described in the following discussion.

THROMBOELASTOGRAPHY

The various components of the thromboelastography (TEG) tracing are depicted in Fig. 16-8, and the viscoelastic output measured by TEG is summarized in Table 16-2. The rapid TEG is designed to provide the quickest results by using both extrinsic (tissue factor) and intrinsic (kaolin) pathway activators. The downside of maximizing clot initiation is the resulting amplification of the coagulation system, which may mask

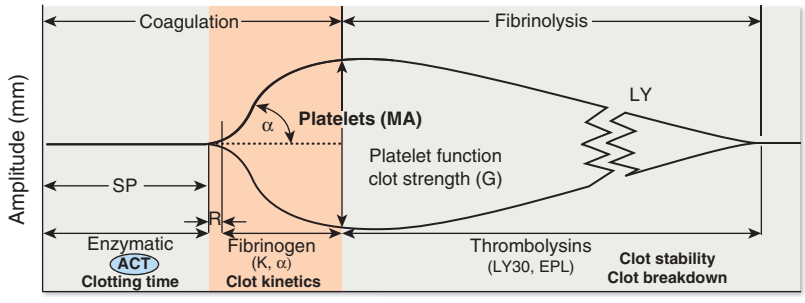


FIGURE 16-8 Thromboelastography (TEG). The TEG tracing depicts the temporal phases of clot assembly: ACT (activated clotting) represents the enzymatic component (initiation), α (angle) reflects fibrin crosslinking (propagation), and MA (maximum amplitude) is the final clot strength resulting from fibrin and platelet interaction (amplifications). Clot dissolution is quantified by LY30, which is percent lysis at 30 minutes following time of MA. EPL, estimated percent lysis.

TABLE 16-2: TEG Parameters

Parameter	Significance
SP time	Earliest activity of enzymatic factors, causing tracing to split
R time	Initiation phase of enzymatic factor activity
K time	Potentiation phase of enzymatic factors yielding clot strengthening
Alpha-angle	Rate of clot strengthening through polymerization of available fibrinogen
MA	Platelet functional contribution to clot strength through IIb–IIIa receptor interaction with fibrin
G	Overall total clot strength resulting from all coagulation interactions, calculated from amplitude (*A*), $G = (5000 \times A)/(100 \times A)$
EPL	Fibrinolytic activity, derived from percent decrease in clot strength after MA is reached

Alpha-angle, degrees; EPL, estimated percent lysis, %; *G*, clot strength, dynes/cm^2; *K*, coagulation time, minutes; MA, maximum amplitude, millimeters; *R*, reaction time, minutes; SP, split point, minutes.

more subtle changes in coagulation, particularly in the hypercoagulable setting or when quantifying fibrinolysis. Kaolin activation is used for the later indications. A native TEG is performed without activators for subtle changes in the coagulation system and is primarily used for research. The reaction time (*R*, minutes) is defined as the time elapsed from the initiation of the test to the point where the onset of clotting provides enough resistance to produce a 2-mm amplitude reading on the TEG tracing.[116] Of note, in the R-TEG assay (discussed later), due to the acceleration of clotting initiation, the *R* time is represented by a TEG-derived activated clotting time (TEG-ACT). The *R* time and TEG-ACT are most representative of the initiation phase of clotting due to enzymatic factors. A prolonged *R* time or TEG-ACT is diagnostic of hypocoagulability, whereas shortened times suggest hypercoagulability. The alpha-angle (α, degrees) is the angle formed by the slope of a tangent line traced from the *R* to the time required to reach 20-mm amplitude (*K* time) measured in degrees. Alpha-angle represents the rate at which the clot strengthens and is most representative of thrombin's cleavage of available fibrinogen into fibrin. The maximum amplitude (MA, mm) is the point at which clot strength reaches its maximum on the TEG tracing, reflects the end result of maximal platelet–fibrin interaction, and is often used to guide platelet transfusions. The LY30 variable represents the percentage of clot degradation 30 minutes after the clot reaches MA and is used to quantify fibrinolysis. The functional fibrinogen (FF-TEG) assay is a more specific method to quantify fibrinogen contribution to clot strength and is performed by using a monoclonal antibody to glycoprotein IIb–IIIa to eliminate the contribution of platelets to clot formation.[117] One of the limitations of the viscoelastic assay is the generation of abundant thrombin, which masks the effects of antiplatelet therapy or potential platelet dysfunction by activating platelets via the robust protease activating receptors (PAR) 1 and 4. To quantify platelet dysfunction, a platelet mapping assay (PM-TEG) relies on heparin to eliminate thrombin activation and employs reptilase and factor XIII to cross-link fibrin and, thus, eliminate the fibrin contribution to clot strength. The contribution of the P2Y12 or thromboxane receptors to clot formation is measured by the addition of ADP or arachidonic acid, respectively. There are additional TEG variables that are not routinely used clinically that also provide information on clot strength and fibrinolysis, but at this time, these are mostly used in experimental work.

ROTEM

ROTEM is the other commonly employed viscoelastic assay[118] and is based on the same fundamental principles as TEG. Consequently, the graphical output appears similar to that obtained with TEG (Fig. 16-9). In the TEG system,

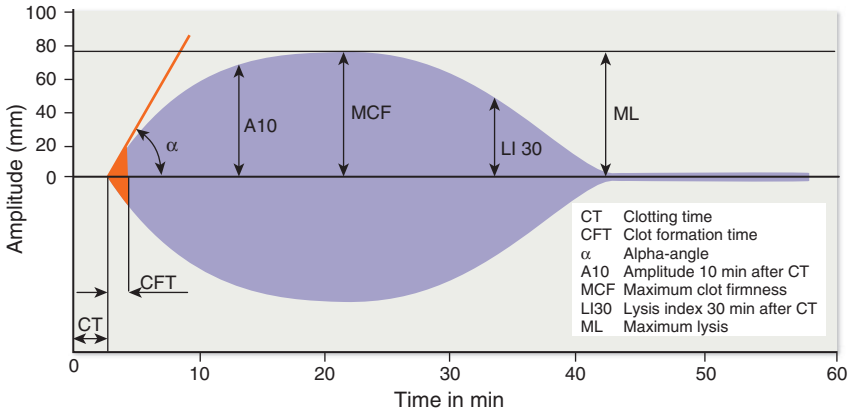


FIGURE 16-9 Rotational thromboelastometry (ROTEM). The ROTEM tracing depicts the temporal phases of clot assembly: CT (clotting time) represents the enzymatic component (initiation), α (angle) reflects fibrin crosslinking (propagation), MCF (maximum clot firmness) is the final clot strength resulting from fibrin and platelet interaction (amplification). Clot dissolution is quantified by CL30, which is percent clot lysis at 30 minutes following initiation of clot formation.

the sample cup oscillates and a pin on a torsion wire is suspended in the blood sample. In the ROTEM system, the sample cup remains fixed while the pin oscillates. ROTEM and TEG provide essentially the same information on clot formation kinetics and strength (Table 16-2). EXTEM is activated with tissue factor (external) and INTEM with ellagic acid (internal). FIBTEM is a modified EXTEM with the added platelet inhibitor cytoclasin D to provide an estimate of functional fibrinogen,[119] and APTEM is a modified EXTEM with the addition of tranexamic acid (TXA) to guide antifibrinolytic therapy. The NATEM is recalcified without activators. Another assay, approved in Europe, is ECATEM using the viper venom ecarin (prothrombin activator) to measure the effect of direct thrombin inhibitors. Because of differences in operating characteristics, the results of ROTEM and TEG are not interchangeable. Furthermore, the two tests use different nomenclature to describe the same variables, although the sensitivity of TEG and ROTEM variables to detect coagulopathy is comparable.[120] Limits of agreement between TEG and ROTEM variables from multiple institutions have been reported. However, newer generation TEG and ROTEM devices, currently undergoing clinical validation for US Food and Drug Administration (FDA) approval, are operator-independent, vibration-resistant, and smaller devices. Thus, these devices facilitate point-of-care capabilities, including prehospital use.

Clinical Implementation of Viscoelastic Hemostatic Assays

A recent prospective randomized trial has shown that viscoelastic assays are superior compared to PT/international normalized ratio (INR), activated PTT (aPTT), platelet count, fibrinogen level, and D-dimer for the management of TIC.[121] Furthermore, a retrospective cohort study indicated that viscoelastic assays are better than a fixed ratio (1:1:1) strategy.[122] TEG is also capable of detecting hypercoagulability (increased MA), whereas the PT and aPTT are not.[123] Perhaps the greatest advantage of viscoelastic assays is to individualize management of patients based on their phenotype of TIC. Considering the diverse mechanisms driving TIC (Figs. 16-3 and 16-6), severely injured patients manifest unique patterns of TIC (Fig. 16-4).[61,62] Patients with predominately impaired thrombin generation have a prolonged *R* and low MA. Depressed MA has been associated with an increased risk of bleeding and mortality.[124,125] The ROTEM A5 (5 minutes after clot initiation) and A10 have also been accurate in early identification of patients with advanced TIC.[126]

MANAGEMENT OF TIC

Blood Components

RBC TRANSFUSION

RBC transfusion is lifesaving in the setting of critical anemia associated with hemorrhagic shock. However, the optimal target hematocrit during resuscitation remains unknown (see Chapter 15). Elimination of oxygen debt involves optimization of oxygen delivery, which is the product of cardiac output and arterial oxygen content. The arterial oxygen content, in turn, is dependent primarily on the hemoglobin concentration and oxygen saturation. During resuscitation, a balance must occur between the competing goals of maximal oxygen content (hematocrit = 100%) and minimal blood viscosity (hematocrit = 0%). Furthermore, beyond a role in oxygen delivery, erythrocytes are integral to hemostasis via their involvement in platelet function as well as thrombin generation. The hematocrit is thus relevant to hemorrhagic shock as it relates to both oxygen availability and hemostatic integrity. As the hematocrit rises, platelets are displaced laterally toward the vessel wall, placing them in contact with the injured endothelium; this phenomenon is referred to as margination. Platelet adhesion via margination appears optimal at a hematocrit of 40%.[127] Erythrocytes are also involved in the biochemical and functional responsiveness of activated platelets. Furthermore, RBCs participate in thrombin generation through exposure of procoagulant phospholipids and generation of meizothrombin.[34] Interestingly, animal studies suggest that a decrease of the platelet count of 50,000 is compensated for by a 10% increase in hematocrit.[128] At this point, we continue to target a hemoglobin of greater than 10 g during hemorrhage control.[129]

PLASMA TRANSFUSION

As discussed previously, the role of preemptive plasma transfusion was introduced in the late 1970s to manage coagulopathy associated with major liver injuries.[19,130] Several decades later, Borgman et al[27] evaluated the military data and identified that a high ratio of plasma to RBCs has a survival advantage. This report prompted ongoing debate in the civilian setting in regard to the optimal empiric ratios of plasma to RBC.[131] Plasma is obtained via whole blood centrifugation or apheresis. Despite plasma's acellular composition, this blood product contains hundreds of biologically active proteins that have considerable variability between individuals.[132] Although blood banks have minimal requirements for concentrations of certain coagulation factors, it is becoming clear that not all plasma is the same. AB plasma is the universal donor, but it is limited in supply because less than 2% of the population qualify as donors. Appreciation of the calcium binding capacity due to citrate in stored plasma is essential to prevent iatrogenic hypocalcemia, which exacerbates coagulopathy. Pooled plasma with pathogen reduction, such as Octaplas and Uniplas, may have a role in early plasma resuscitation in trauma patients because they have a more standardized composition and do not require the calcium-chelating anticoagulant citrate.[133]

Plasma containing the highest concentration of coagulation factors is currently FFP that represents plasma frozen at –18°C within 8 hours of blood draw. Alternatively, plasma can be frozen within 24 hours, termed FP24, which has a small reduction in coagulation factors. Frozen plasma

requires 20 to 40 minutes to thaw prior to transfusion by conventional devices. For this reason, many trauma centers have prethawed plasma available in the emergency department. Thawed FFP is required to be transfused within 24 hours or becomes relabeled as thawed plasma. Thawed plasma can be stored at 4°C for up to 5 days and can be useful in the setting of trauma resuscitations. Thawed plasma retains many functioning proteins but is depleted of the more labile factors V, VII, and VIII.[134] In plasma resuscitation, it is important to appreciate that patients are receiving more than just coagulation factors. Plasma contains abundant proteins with diverse regulatory functions related to inflammation, coagulation, and fibrinolysis, in addition to being an effective colloid. The levels of these proteins have high variability person to person and between sexes.[132] Plasma-first resuscitation in animal models has been shown to repair the endothelium,[135] improve platelet function,[136] and attenuate fibrinolysis.[72]

Animal, in vitro, and retrospective clinical data have been supportive of using plasma for prehospital resuscitation, which has been confirmed by the recently completed Prehospital Air Medical Plasma (PAMPer) trial.[137] This randomized clinical study allocated patients to standard of care (crystalloid) prehospital helicopter resuscitation versus 2 units of plasma. The overall mortality was reduced by nearly 10% in the plasma arm of the study. Although these results were encouraging for adopting plasma's role in early prehospital resuscitation, the same benefit in mortality was not appreciated in an urban ambulance study, the Control of Major Bleeding After Trauma (COMBAT) trial.[138] Major differences in these trials included a higher rate of penetrating trauma in the COMBAT study and much shorter transport times compared to PAMPer. Thus, the patients who appear to benefit the most from prehospital plasma resuscitation are patients with blunt injury and greater than 40 minutes of transport time. Both of these studies provide critical data on the evolving role of plasma in the resuscitation of trauma patients; we originally believed that every patient benefited from early plasma, but in reality, clotting factor deficiency may not be the critical factor driving TIC. Perhaps plasma is more important for volume restoration, minimizing the duration of shock during transport. Ongoing research is needed to identify a more defined role of plasma in the prehospital setting, in addition to cost and potential complications. The prohibitive logistics of thawing plasma for ground transportation, however, have now been resolved with the availability of lyophilized plasma.

CRYOPRECIPITATE/FIBRINOGEN REPLACEMENT

Fibrinogen levels are decreased early after injury in coagulopathic trauma patients (<1.5 g/L in 15%, <1.0 g/L in 5%, and <0.5 g/L in 2%). Although plasma units contain fibrinogen, the low concentration requires a large volume of transfusion; hence, cryoprecipitate has remained the main source of fibrinogen supplementation in the United States. Cryoprecipitate also contains factor VIII, von Willebrand factor, fibronectin, factor XIII, and platelet microparticles, which may all be important in hemostasis. The European experience suggests recombinant fibrinogen concentrate is effective in the management of TIC. Each 10-unit pool of fibrinogen contains approximately 5 g of fibrinogen or a median 500 mg per unit. Most trauma centers administer fibrinogen dosed by 10-unit pools (one fibrinogen "pack" = 10 pooled units). A 10-pooled unit of fibrinogen transfusion is expected to increase fibrinogen by 0.5 to 0.9 g/L; however, dose response varies in the setting of ongoing bleeding and may require redosing. Several retrospective studies in trauma patients, civilian and military, suggest that more aggressive use of fibrinogen replacement improves outcome.[139,140] Consensus guidelines recommend a fibrinogen threshold of greater than 1.5 to 2.0 g/L for transfusing for hemostasis.[141] Recent studies using a viscoelastic assay to administer cryoprecipitate as part of a goal-directed transfusion strategy use the alpha-angle or FF assay of TEG or FIBTEM of ROTEM as a trigger for transfusion. Randomized clinical trials are underway to evaluate the impact of empiric cryoprecipitate administration on outcomes in patients requiring massive transfusion, as well as prehospital administration of cryoprecipitate. Label indications for recombinant fibrinogen concentrate use in Europe allow for its use in both congenital and acquired coagulopathies (eg, TIC); however, in the United States, its use is restricted to congenital coagulopathies. On the other hand, cryoprecipitate has been removed from most blood banks in Europe due to transfusion-related infectious concerns given that transfusion of one dose (or "pack") of cryoprecipitate exposes the patient to 10 random donors, hence the surge in use of fibrinogen concentrate in Europe. To date, retrospective reviews of the use of fibrinogen concentrate in trauma patients suggest it has been associated with reduced blood product requirement, but no conclusions can be drawn for the impact on mortality.

PLATELET TRANSFUSION

Before the development of platelet concentrates in the second half of the 20th century, platelets were administered through whole blood transfusion. When blood component therapy became the standard, transfusion of platelets focused on treating thrombocytopenia; in trauma, this was often the result of dilution from crystalloid and RBC transfusion. But platelet dysfunction has been shown to occur following injury despite platelet numbers exceeding these traditional threshold levels.[110-112] The strategy of early transfusion of hemostatic blood components has improved outcomes after injury; however, whether this is due to the early administration of platelets or of plasma is difficult to distinguish. Perkins et al[142] reported that platelet administration was independently associated with survival in combat casualties. In a review of civilian trauma patients requiring transfusion of 5 or more RBC units in 24 hours, Brasel et al[143] reported that a higher ratio of platelets to RBC (≥1:2) units was independently associated with improved 30-day survival among patients with traumatic brain injury, whereas a higher ratio of plasma to RBC (≥1:2) units was independently associated with improved 30-day survival only in non–traumatic brain injury patients. On the other hand, more recent data suggest

that presumptive platelet administration does not improve outcome for patients with blunt traumatic brain injury.[144] Holcomb et al[145] reported that in civilian trauma patients transfused 10 or more RBC units in 24 hours, using a time-dependent analysis of survival, a higher plasma:platelet:RBC unit ratio (≥1:1:2) was associated with improved survival, compared to lower ratios. However, survival was not significantly different between patients with lower plasma but higher platelet to RBC unit ratio and higher plasma but lower platelet to RBC unit ratio. A drawback of these retrospective studies is that the collinearity between plasma and platelet ratios obscures the relative contribution of each component to patient outcomes. This is further compounded by the fact that all platelet concentrates are suspended in plasma; for apheresis platelets, each unit contains 1 unit of plasma. The recent multicenter, randomized, controlled PROPPR (Pragmatic, Randomized, Optimal Platelet and Plasma Ratios) trial to assess the impact on survival of 1:1 versus 1:2 FFP:RBC did not show a survival benefit for a higher ratio of platelets to RBCs.[146] Perhaps one of the reasons for conflicting results is the timing of platelet administration. Previous experimental work indicates that platelets given to acidotic animals are irreversibly impaired.[56] Furthermore, a recent multicenter trial of patients with cerebral hemorrhage indicated increased mortality with preemptive platelet transfusion (ie, the Platelet Transfusion Versus Standard Care After Stroke due to Spontaneous Cerebral Haemorrhage Associated With Antiplatelet Therapy [PATCH] trial, a recently completed multinational study). Finally, to add to the confusion, there is evidence that cold stored platelets may be superior for reversing coagulopathy.[147]

Warm Fresh Whole Blood

After the introduction of whole blood fractionation into blood components for clinical use in the late 1970s, several civilian trauma centers continued to transfuse warm fresh whole blood for severely injured patients with a refractory coagulopathy.[19] This concept was resurrected in the recent military conflict in Iraq, where plasma and platelets were not immediately available.[148] The concept of fresh whole blood transfusion is becoming the international standard for far-forward care,[149] and the reported experience has prompted its reconsideration in the civilian setting.

Low Titer O-Negative Whole Blood

The success with fresh whole blood in the military setting has rejuvenated interest in the administration of stored low titer O-negative whole blood (LTOWB) for civilian use. The American Association for Blood Banking approved LTOWB for civilian trauma use in 2018, and a number of trials are underway to determine its optimal application.[150]

PROTHROMBIN COMPLEX CONCENTRATE

Prothrombin complex concentrate (PCC) contains the vitamin K–dependent coagulation factors II, VII, IX, and X, as well as the anticoagulants protein C and protein S. As such, its main indication is for rapid reversal of the anticoagulant effects of warfarin in bleeding patients. Importantly, PCC provides these factors in a concentrated form as compared to FFP (approximately 50 mL vs 300 mL, respectively). The efficacy of PCC for urgent reversal of warfarin in the perioperative period has been well documented,[151] and use in this setting has been recommended by expert panels in the United States and Europe. The role of PCC in the bleeding patient not on anticoagulants continues to be studied. Bruce and Nokes[152] reported favorable outcomes following PCC administration for uncontrolled hemorrhage after cardiac surgery, exclusive of warfarin use. Several animal studies of hemodilutional coagulopathy have reported superior achievement of hemostatic resuscitation using PCC as compared to FFP.[153] Although the reduced time and volume necessary to achieve coagulation factor replacement are appealing, transfusion of PCC represents only a limited number of coagulation proteins and likely carries the same immunomodulatory risks as those of FFP. Controlled trials in injured patients are necessary to define recommendations.[154]

RECOMBINANT FACTOR VIIA

Treatment of postinjury coagulopathy with supraphysiologic doses of recombinant factor VIIa (rVIIa) was proposed to amplify coagulation through generation of a thrombin burst in the presence of both tissue factor and functional platelets and in the absence of either hypothermia or acidosis. Due to continued concerns over efficacy, thrombotic risk, and cost, two parallel randomized controlled trials, one of penetrating and one of blunt trauma patients, were conducted among 32 international institutions (exclusive of the United States) from 2003 to 2004.[155] Eligibility criterion was transfusion of 6 or more units of RBCs in 4 hours. Subjects were randomized to receive either placebo or three doses of rVIIa (200, 100, and 100 mcg/kg), with the first dose given after the eighth unit of RBC was transfused. A reduction in RBC transfusion requirement for both blunt (median 7.0 vs 7.5) and penetrating (3.9 vs 4.2) groups was observed among those alive 48 hours after the first dose of the study drug. However, these relatively small differences were eliminated when the analysis was expanded to include patients who had died within the first 48 hours of injury. In another multicenter trial of rVIIa in trauma, including the United States, there was also no evidence of significant clinical benefit.[156] Thus, the role for rVIIa in trauma care is considered limited. If rVIIa therapy is contemplated, several pharmacokinetic properties of the drug warrant consideration. The activity of rVIIa is both pH and temperature dependent, with a 60% decrement at a pH of 7.20 and a 20% decrement at a temperature less than 34°C.[55] Furthermore, physiologic concentrations of calcium, fibrinogen, and platelets are required, such that replacement of these clotting factors is essential prior to rVIIa administration. Dosages range from 50 to 200 mcg/kg, with redosing recommended for continued coagulopathy beyond the half-life of 2 hours.

FACTOR XIIIA

Factor XIII acts to stabilize the fibrin clot once formed, and a relationship between XIII concentration and maximal clot firmness has been demonstrated experimentally.[157] Early depletion of the limited endogenous XIII stores has been documented following major hemorrhage and is believed to contribute to coagulopathy.[158] Although prospective data are lacking, recombinant factor XIIIa should be considered for postinjury coagulopathy that is refractory to conventional factor replacement therapy. As discussed previously, factor XIII is abundant in cryoprecipitate.

ANTIFIBRINOLYTICS

The role of antifibrinolytics in trauma remains controversial and has been fueled by the recognition that the majority of severely injured patients manifest fibrinolysis shutdown.[98] Antifibrinolytic agents include the lysine analogue inhibitors of plasminogen binding to fibrin, aminocaproic acid (Amicar) (dosing, 150 mg/kg, followed by 15 mg/kg/h) and TXA (dosing, 10 mg/kg, followed by 1 mg/kg/h), as well as the direct plasmin inhibitor aprotinin (dosing, 280 mg, followed by 70 mg/h). In addition to its effect on plasmin, aprotinin also inhibits kallikrein, factor XIIa, and trypsin. Aprotinin, however, is not currently approved in the United States due to increased mortality in a randomized trial during cardiac surgery.[159] The CRASH II trial suggested improvement in survival with TXA,[76] but this trial has many limitations,[160] particularly when applied to care at US Level I trauma centers. Furthermore, there is compelling investigative work suggesting that indiscriminate use of antifibrinolytics can have adverse effects.[161] Retrospective studies in civilian centers in the United States have failed to identify a survival advantage,[162] and several studies have suggested increased mortality.[163,164] On the other hand, inhibiting plasminogen activation may have benefits beyond coagulation considering the diverse cellular receptors for plasminogen.[67] There are a number of ongoing randomized trials evaluating the efficacy of TXA in trauma patients that will be completed over the next several years that will better define the role of antifibrinolytics in trauma. The most recent randomized trial evaluating 180 patients with traumatic brain injury identified no survival advantage and a fivefold increased rate of pulmonary embolism in patients who received TXA compared to controls.[165] The same association of no benefit in mortality with TXA and increased thrombotic complications was also appreciated in the most recent military experience.[166]

Resuscitation Strategies for TIC

PREDICTING MASSIVE TRANSFUSION

Massive transfusions require a predetermined plan to rapidly deliver blood products to the exsanguinating patients. One of the major challenges in trauma resuscitation is correctly identifying patients at risk of massive transfusion. Several scoring strategies have been proposed to rank patients at risk of life-threatening hemorrhage. Many of these scores use a combination of physiologic measurements, laboratory values, and physical findings. The Trauma-Associated Severe Hemorrhage (TASH) score was developed from a regression analysis that identified seven independent predictors of massive transfusion in trauma patients based on retrospective data.[167] The score takes into consideration the patient's systolic blood pressure, hemoglobin, focused assessment with sonography in trauma (FAST) examination, pelvic/long-bone fracture, gender, and base deficit. The TASH score has a range from 0 to 28, making it somewhat complex to calculate. The Assessment of Blood Consumption (ABC) score was proposed and was developed as a minimalist score not requiring laboratory values to predict massive transfusions.[168] The ABC score incorporates four variables that are simplified by dichotomous point scoring, rather than ordinal grouping as used in TASH. These include a positive FAST, penetrating mechanism, heart rate >120 bpm, and systolic blood pressure less than 90 mm Hg. In consideration of these scoring systems, the timing of when a decision can be made to activate a massive transfusion is critical. The optimal goal is early communication to the blood bank of the urgent need of a large volume of blood products. The majority of the scoring systems have a high negative predictive value but universally lack a strong positive predictive value, which is likely best determined at this time by experience.[169] Using clinical variables will continue to be problematic due to their dynamic nature, and an objective measure to identify those at risk of massive transfusion is needed. We are optimistic that the t-PA challenge TEG assay may prove to have clinical utility, as results to risk stratify patients for massive transfusion can be obtained within 15 minutes[170] while providing additional information on the patients' coagulation status. Our current massive transfusion protocol using rapid TEG emphasizing goal-directed approach is shown in Fig. 16-10.

HYPOTENSIVE CRYSTALLOID RESUSCITATION

Permissive hypotension was proposed as a deliberate tolerance of lower arterial pressures in the face of uncontrolled hemorrhagic shock in order to minimize further bleeding. This strategy is based on the notion that decreasing perfusion pressure will maximize the body's natural mechanisms for hemostasis, such as arteriolar vasoconstriction, increased blood viscosity, and in situ thrombus formation (see Chapter 15). In the first large-scale trial, Bickell et al[171] randomized patients in hemorrhagic shock (systolic blood pressure [SBP] <90 mm Hg) who had sustained penetrating torso trauma to either crystalloid resuscitation or no resuscitation prior to operative intervention. Mean SBP was significantly decreased on arrival for the delayed resuscitation group as compared to the immediate resuscitation group (72 mm Hg vs 79 mm Hg, respectively, $P = .02$), with a corresponding increase in survival (70% vs 62%, respectively, $P = .04$). A subsequent subgroup analysis, however, indicated that these benefits were limited to patients who had cardiac injury with tamponade. Furthermore, prehospital crystalloid resuscitation in severely injured patients with sustained hypotension appears to be

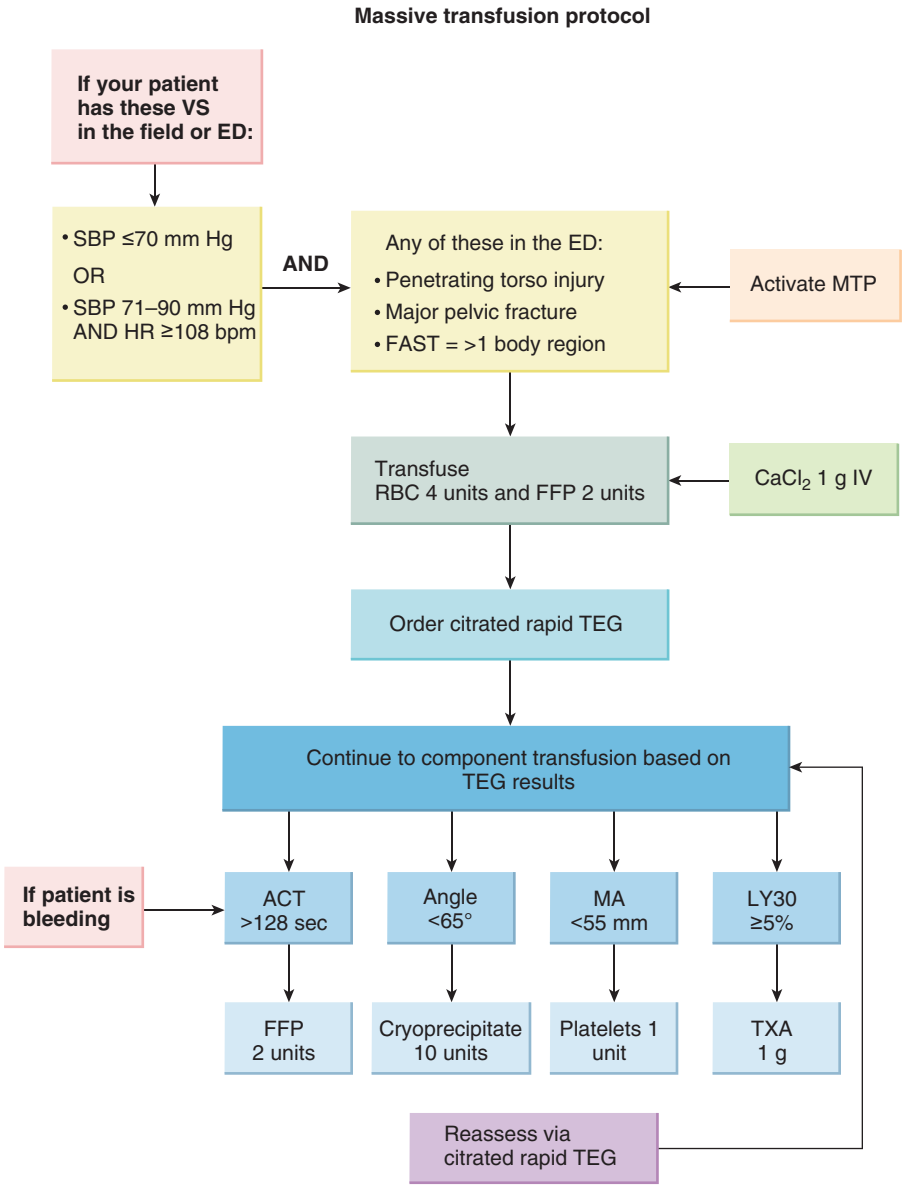


FIGURE 16-10 Massive transfusion protocol (MTP). The Denver Health MTP is primed by the Research Outcomes Consortium (ROC) prehospital physiologic criteria (systolic blood pressure [SBP] ≤70 mm Hg or SBP 71–90 mm Hg and heart rate [HR] >108 bpm) and activated at emergency department (ED) arrival with the anatomic findings of a penetrating torso injury, major pelvic fracture, or a focused assessment with sonography in trauma (FAST) = >1 body region. Upon activation, 1 g of calcium chloride ($CaCl_2$) is administered, and resuscitation is initiated with a fresh frozen plasma (FFP) to red blood cell (RBC) ratio of 1:2. Subsequent blood components are delivered based on thromboelastography measurements. ACT, activated clotting time; IV, intravenous; LY30, percent lysis at 30 minutes; MA, maximum amplitude; TXA, tranexamic acid; VS, vital signs.

beneficial. Brown et al[172] observed a survival benefit with increased crystalloid use in blunt injured trauma patients with prehospital hypotension. Schreiber et al[173] reported a survival advantage using limited crystalloid resuscitation in the prehospital setting in a prospective phase II trial. In this study, patients with an SBP of less than 70 mm Hg were randomized either to resuscitation with a 250-mL bolus of crystalloid with repeated doses to maintain an SBP of greater than 70 mm Hg or palpable radial pulse or to standard care of 2 L. In blunt injury, mortality was reduced from 18% in the standard-of-care group to 3% in the controlled resuscitation group, whereas in penetrating trauma, there was no difference in survival. In considering the dominant factors for TIC, hypoperfusion and associated metabolic acidosis are associated with impaired thrombin generation and hyperfibrinolysis. Overzealous crystalloid dilution dilutes whole blood and results in the depletion of the critical balance of regulatory proteins.[72]

Goal-Directed Management of TIC

Although there is no debate in the United States that early blood component resuscitation is warranted for the severely injured patient at risk, there is substantial debate as how to best deliver the optimal quantity of plasma and platelets; that is, fixed ratios or point-of-care guided resuscitation with clinical assays. The large Prospective, Observational, Multicenter, Major Trauma Transfusion (PROMMTT) trial suggested the beneficial effects of early plasma administration during trauma resuscitation and indicated platelets were given late.[2] These findings were then translated into the multicenter randomized controlled PROPPR trial to assess the impact on survival of 1:1 versus 1:2.[146] The PROPPR study also included a different platelet:RBC transfusion target, which confounded the results. The important finding from this study was that the ratios did not make a difference in the primary study end points of 24-hour or 30-day mortality. However, there have been no randomized trials to address the important issue of fixed-ratio versus goal-directed management, although a retrospective cohort evaluation suggested that TEG-driven resuscitation is superior.[122] Our current TEG-driven protocol[174] is outlined in Fig. 16-10. In the injured patient considered at risk of TIC, a citrated rapid TEG is performed and 1 g of calcium chloride is administered, followed by transfusion of 2 units of FP24 and 4 units of RBCs. Subsequent blood products are delivered based on the initial rapid TEG output, and serial TEG-directed transfusions are continued until hemostasis is achieved. If TEG results are not available after 4 units of RBCs with the first hour, we administer empiric platelets and cryoprecipitate.[175]

Novel Resuscitation Strategies to Mitigate TIC

New resuscitation strategies are continually being developed and tested.[176] The most recent to fail was hypertonic saline, which was ultimately believed to enhance coagulopathy. The most recent promising solution is adenosine-lidocaine-magnesium (ALM). Originally designed for cardioplegia in cardiothoracic surgery based on an induced hibernation state for myocytes, ALM has now been shown to attenuate TIC in experimental models of postinjury shock.[177]ALM fluid therapy attenuates systemic inflammation, platelet dysfunction, and coagulopathy after noncompressible truncal hemorrhage.[178]

MANAGEMENT OF ANTICOAGULATION/ANTIPLATELET THERAPY

Antiplatelet Therapy

Antiplatelet therapy, which consists predominantly of acetylsalicylic acid (ASA) and clopidogrel (P2Y12 inhibitors), has revolutionized the care of patients with atherosclerotic cardiovascular disease. Although via independent mechanisms, both ASA and clopidogrel irreversibly inhibit platelet aggregation. The average lifespan of a platelet is roughly 9 days, and platelet function recovers gradually following cessation of antiplatelet agents, as the bone marrow generates approximately 30,000 platelets per day. Data comparing the incidence of intracerebral hemorrhage (ICH) between patients using antiplatelet therapy and controls are sparse. However, several matched case series of head-injured patients have indicated an increased severity of hemorrhage, as well as mortality, among patients using antiplatelet therapy as compared to controls.[179,180] These reports are limited primarily by confounding comorbidity, as patients using antiplatelet therapies tend to be sicker. Despite an apparent increased morbidity associated with preinjury antiplatelet agents among head-injured patients, little data exist to support routine platelet transfusion in this setting.[144] Prospective randomized data are warranted, preferably incorporating serial platelet functional assessment. These studies are particularly timely as both the elderly demographic and prevalence of antiplatelet therapy are increasing exponentially. Interestingly, the deleterious effects of antiplatelet therapy do not appear to extend to the non–head-injured trauma patient. In a recent report, no mortality difference was noted for patients using antiplatelet therapies as compared to controls among a cohort of trauma patients with no evidence of ICH.[181]

Warfarin

Warfarin is an oral anticoagulant that inhibits synthesis of the vitamin K–dependent clotting factors II, VII, IX, and X, as well as the anticoagulant protein C and protein S. It is the most common drug prescribed to achieve chronic anticoagulation. Several studies have documented an increased likelihood of traumatic ICH, as well as severity of injury and mortality, among trauma patients using warfarin as compared to nonanticoagulated patients. Importantly, the increased likelihood of adverse outcomes is observed only among patients who have achieved therapeutic anticoagulation (INR >2).[182] Rapid identification of patients on therapeutic warfarin and at risk for traumatic ICH is paramount to minimize hemorrhage progression with resultant irreversible neurologic consequences. Most authors advocate empiric transfusion of FFP prior to laboratory documentation of therapeutic anticoagulation. In such patients, it is argued that the morbidity of a delay in reversal of patients with a supratherapeutic INR outweighs that of unnecessary factor administration to patients with a subtherapeutic INR. Although up to one-half of trauma patients on warfarin present with a subtherapeutic INR,[183] the morbidity of an expanding ICH renders this argument reasonable. However, a striking amount of variability exists among trauma surgeons as to the INR above which reversal of anticoagulation should be implemented, the rapidity with which the PT is normalized, or the target INR following reversal. As mentioned previously, reversal of warfarin-induced anticoagulation using PCC may be particularly beneficial in patients at high risk for cardiopulmonary complications secondary to the large-volume plasma administration to correct the INR.[151,153,154] The risk of warfarin anticoagulation in the

non–head-injured trauma patient is less clear. A recent study of blunt injured patients age 60 years or older who sustained trauma to the abdomen or thorax, in the absence of intracranial injury, demonstrated no difference in outcome.[182]

Direct Thrombin and Factor Xa Inhibitors

Chronic anticoagulation has changed substantially in the United States since the introduction of the oral direct thrombin inhibitor dabigatran (Pradaxa) in 2010.[184] Subsequently, the FDA has approved two oral factors Xa (FXa) inhibitors, rivaroxaban (Xarelto) and apixaban (Eliquis), and no doubt others are on the horizon. Dabigatran binds to free and clot-bound thrombin, preventing the conversion of fibrinogen to fibrin and platelet aggregation via the PAR receptors. Peak plasma levels occur in 1 to 2 hours. Clearance is predominantly renal, with a half-life of 14 to 17 hours. In addition, 20% is metabolized in the liver. Laboratory assessment of the anticoagulation effects are problematic. PT, PTT, and thrombin time are flat dose response (ie, useful for screening only). Rapid TEG results are erratic, but trends appear to correlate with circulating drug levels.[185] Ecarin clotting time has a dose response to dabigatran in therapeutic ranges but is not currently available in most hospitals. ROTEM is anticipated to have a channel to accomplish this testing soon. Reversal has been challenging until recently, and hemodialysis was recommended for emergent settings. The only promising universal reversal agent has been factor eight inhibitor bypass (FEIBA).[186] The antibody fragment idarucizumab appears to be clinically effective in reversing dibigitran,[187] but results have been variable.[188] Rivaroxaban reversibly binds the active site of FXa, preventing the formation of thrombin and subsequent fibrinogen cleavage.[189] The plasma half-life is 2 to 4 hours. Clearance is 66% renal and 33% hepatic; the half-life is 5 to 9 hours.[184] As expected, the most accurate assay for circulating levels is the anti-FXa assay. PT is prolonged in a linear fashion but is variable depending on the test reagents. The response to PTT is nonlinear, and the reliability of TEG remains unclear.[190] Dialysis is not useful for reversal because rivaroxaban is highly protein bound. A new reversal agent for FXa inhibitors, andexanet alfa, is now available.[191] Apixaban is another competitive inhibitor of FXa.[184] Peak circulating levels are achieved in 3 to 4 hours; clearance is 50% renal and 50% hepatic. At this point, there are insufficient data to recommend testing and reversal agents, but similarities to rivaroxaban are anticipated.

Hypercoagulability

VENOUS THROMBOEMBOLISM

The incidence of proximal lower extremity deep venous thrombosis (DVT) in injured patients without preventive measures has been reported at 18% to 32%, and the risk of significant pulmonary embolism (PE) ranges from 0.3% to 2%.[192] These figures are confounded by the fact that surveillance duplex will identify a higher incidence of asymptomatic DVT, but postinjury PE occurs in the absence of DVT in the majority of patients.[192] Furthermore, asymptomatic PE has been identified in more than 25% of severely injured patients on admission chest computed tomography scanning,[85] yet early symptomatic PE has been associated with extremity trauma but not thoracic injuries.[193] Consequently, it is not surprising that the prevention of postinjury venous thromboembolism (VTE) remains controversial, underscoring the critical need to understand the pathophysiology and fundamental mechanisms to design effective preventive strategies.

Virchow's Triad and Beyond

A century ago, Cannon recognized that the initial response to severe trauma is hypercoagulability, quickly followed by hypocoagulability. Severely injured patients who survive their injury subsequently transition to a hypercoagulable state as early as 24 hours following injury.[194] In fact, patients presenting with advanced TIC requiring a massive transfusion are at the greatest risk for VTE in the surgical intensive care unit. The etiology of this hypercoagulability is likely multifactorial, involving endothelial injury, circulatory stasis, platelet activation, hyperfibrinogenemia, decreased levels of endogenous anticoagulants, and impaired fibrinolysis. Furie and Furie[195] emphasize the role of the endothelium in maintaining blood flow via the generation of anticoagulants, nitric oxide, prostacyclin, and receptor expression. But even intact endothelium, when activated, can provoke thrombus formation. Furthermore, circulating leukocytes and microparticles can express tissue factor. Obesity[196] and traumatic brain injury[197] are independent risk factors for postinjury VTE, although the responsible mechanisms remain speculative.

Diagnosis of Hypercoagulability

The diagnosis and, thus, treatment of hypercoagulability following injury have been limited by the lack of accurate laboratory testing addressing the pathogenesis. Degradation products of fibrin (eg, D-dimer; Fig. 16-11) are not reliable in the acutely injured patient because these products merely indicate that a clot has been formed, since the half-life is greater than 12 hours.[89]

Conventional coagulation tests (ie, aPTT and PT/INR) are not able to diagnose hypercoagulability or delineate the relative contributions of enzymatic and platelet components. Previous trials investigating the benefit of various mechanical and pharmacoprophylactic regimens among injured patients have neither documented hypercoagulability nor monitored the efficacy of prophylaxis. Furthermore, current guidelines do not address the contribution of platelets or fibrinogen to hypercoagulability. Severely injured patients develop inflammation-driven hyperfibrinogenemia within days of injury,[198] and there is a growing body of evidence implicating platelet activation in the development and propagation of VTE.[199] Our work indicates that platelet hyperactivity contributes substantially to hypercoagulability beyond 48 hours of injury.[200] Antiplatelet therapy may have additional effects on attenuating hypercoagulability.[201] Finally, there is recent

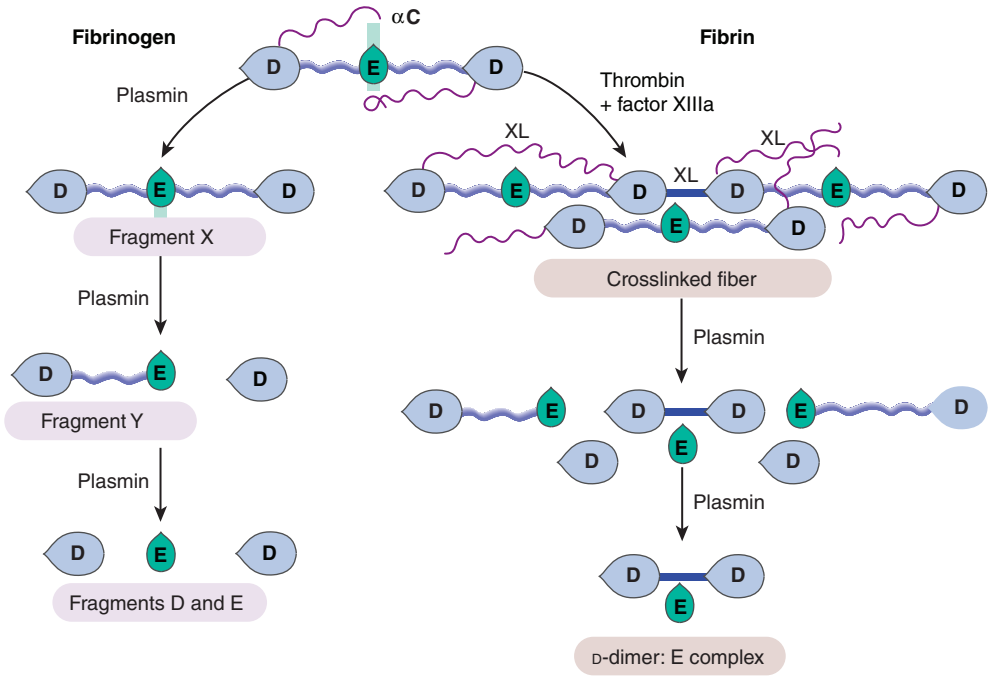


FIGURE 16-11 Fibrin degradation products. Plasmin initially cleaves the AC domains in fibrinogen generating fragment X. The subsequent cleavage step is at the central D and terminal D domains, resulting in fragment Y. Ultimately plasmin can cleave D from E. Plasmin cleaves fibrin protofibrils by removing the crosslinked α chains, followed by release of a number of fibrin degradation products, the smallest being DD/D, which is D-dimer. The persistence of the DD link is due to crosslinking of fibrin by thrombin activated factor XIII.

evidence documenting fibrinolysis shutdown in severely injured patients.[202] In light of these limitations, it is not surprising that most VTEs among trauma patients occur because of prophylaxis failure rather than failure to provide prophylaxis. In a recent comprehensive international analysis of medical and surgical intensive care units, the rate of DVT was 7.7% and the PE rate was 1.3%.[203]

Viscoelastic hemostatic assays may provide a breakthrough in managing hypercoagulability. A TEG-shortened *R* time, increased alpha-angle, and enhanced MA are indicative of hypercoagulability (Fig. 16-8). Our group documented a strong correlation between hypercoagulability indicted by TEG measurements and subsequent VTE. In the face of standard chemoprophylaxis, 60% of patients displayed evidence of hypercoagulability. Furthermore, TEG and ROTEM can distinguish platelet versus fibrinogen contribution to clot formation as well as calibrate fibrinolysis shutdown.[182] We have recently documented that persistent fibrinolysis shutdown (>24 hours) is a risk factor for postinjury VTE and have developed a t-PA sensitivity test (Fig. 16-12) to further

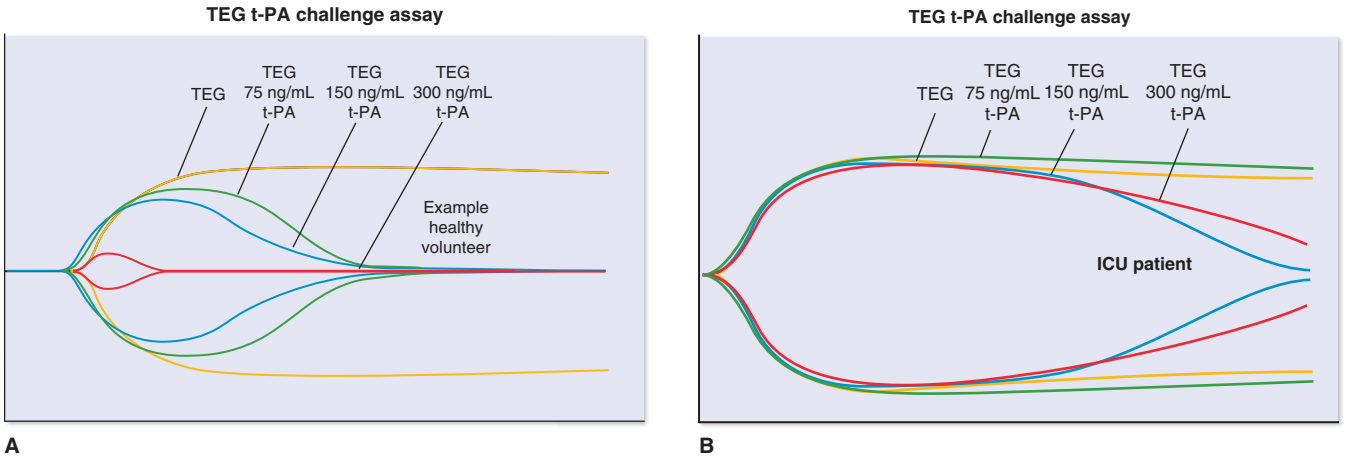


FIGURE 16-12 The tissue plasminogen activator (t-PA) challenge assay. (**A**) The objective of the t-PA assay is to determine sensitivity to exogenous t-PA added to a native thromboelastography (TEG). (**B**) Patients in fibrinolysis shutdown demonstrate reduced LY30 with high concentrations of exogenous t-PA. ICU, intensive care unit.

risk stratify and manage patients at risk. Despite the theoretical advantages of TEG-driven chemoprophylaxis protocols, prospective data remain sparse.

Prevention of Venous Thromboembolism

Early use of preventive measures is critical to prevent postinjury VTE, but identifying the high-risk population has been difficult. The two most widely used scores, Risk Assessment Profile (RAP) and Trauma Embolic Scoring System (TESS), miss a third of the patients who develop VTE.[204] Paradoxically, patients presenting with advanced TIC requiring a massive transfusion are at the greatest risk for subsequent VTE. Mechanical compression devices should be applied as soon as possible in all seriously injured patients. Heparin remains the cornerstone of pharmacologic VTE prophylaxis, and low molecular weight heparin (LMWH) is superior to unfractionated heparin.[201] Furthermore, weight-based LMWH or anti-factor Xa-guided dosing appears to be advantageous.[205] The use of novel oral anticoagulants is now being evaluated as an alternative to LMWH.[206]

The most controversial issue is timing of preventive agents with central nervous system injury.[205] The current debate is 48 versus 72 hours in patients without progressive intracranial hemorrhage. We currently believe aspirin should be routinely added at 72 hours in patients at high risk of VTE and should be dosed according to platelet mapping targeting arachidonic acid receptor inhibition at greater than 80%. Statin therapy may also become routine in the surgical intensive care unit for VTE prophylaxis,[207] via reducing PAI-1 to attenuate t-PA resistance. The role of inferior vena cava filters remains controversial, and at best, these devices have a limited role.[208]

In sum, the prevention of postinjury VTE remains a challenge because we do not currently understand the responsible biologic mechanisms.[209] A number of prospective trials are underway including the Department of Defense–supported Consortium of Leaders in the Study of Traumatic Thromboembolism (CLOTT) program. We currently believe TEG- or ROTEM-driven protocols should be employed, and the combination of a LMWH, aspirin, and a statin appears to be the optimal benefit-to-risk strategy at this moment.

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17

Resuscitative Thoracotomy

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KEY POINTS

- Resuscitative thoracotomy (RT) refers to an emergent thoracotomy, most commonly performed in the emergency department for patients arriving in extremis; RT may also be performed in the operating room or intensive care unit within hours after injury for physiologic deterioration.
- The primary objectives of RT are to release pericardial tamponade, control cardiac or intrathoracic hemorrhage, evacuate bronchovenous air embolism, perform open cardiac massage, and temporarily occlude the descending thoracic aorta.
- The critical determinants of survival following RT include the injury mechanism, anatomic location of injury, and the patient's physiologic condition at the time of thoracotomy.
- The highest survival rate following RT is in patients with penetrating cardiac wounds, especially when associated with pericardial tamponade.
- Based on the literature to date, RT should be performed for (1) penetrating nontorso trauma with cardiopulmonary resuscitation (CPR) of less than 5 minutes, (2) blunt trauma with CPR of less than 10 minutes, and (3) penetrating torso trauma with CPR of less than 15 minutes.
- RT is unlikely to yield productive survival when patients (1) sustain blunt trauma and require more than 10 minutes of prehospital CPR, (2) have penetrating wounds and undergo more than 15 minutes of prehospital CPR, (3) have isolated extremity trauma with more than 5 minutes of prehospital CPR, or (4) manifest asystole without pericardial tamponade.
- Outcome following RT in the adolescent population is largely determined by injury mechanism and physiologic status on presentation to the emergency department; for patients under 15 years of age, there are only isolated cases of survival following penetrating trauma and no apparent survival benefit following blunt trauma.

INTRODUCTION

The number of patients arriving at hospitals in extremis, rather than dying in the prehospital setting, has increased due to the maturation of regionalized trauma systems (see Chapter 4). Salvage of individuals with imminent cardiac arrest or those already undergoing cardiopulmonary resuscitation (CPR) often requires immediate thoracotomy as an integral component of their initial resuscitation in the emergency department (ED). The optimal application of a resuscitative thoracotomy (RT) requires a thorough understanding of its physiologic objectives, technical maneuvers, and the cardiovascular and metabolic consequences. Although resuscitative endovascular balloon occlusion of the aorta (REBOA) has been advocated as a resuscitation maneuver that should replace RT, there is a paucity of objective data to clarify the precise role of REBOA versus RT. In fact, aortic occlusion can be achieved quicker with RT than REBOA in patients arriving with CPR in progress.[1] Furthermore, RT is indicated for life-threatening thoracic injuries. However, we believe REBOA is ideal for unstable pelvic fractures with advanced hemorrhagic shock (see Chapter 39), and we have placed a REBOA in patients who have recovered perfusion following RT when it is evident that a pelvic fracture is the source of acute blood loss. This chapter reviews the features of RT and highlights the specific clinical indications, all of which are essential for the appropriate use of this potentially lifesaving yet costly procedure.

HISTORICAL PERSPECTIVE

Emergent thoracotomy came into use for the treatment of heart wounds and anesthesia-induced cardiac arrest in the late 1800s and early 1900s.[2] The concept of a thoracotomy

as a resuscitative measure began with Schiff's promulgation of open cardiac massage in 1874.[2] Block first suggested the potential application of this technique for penetrating chest wounds and heart lacerations in 1882.[3] Following use of the technique in animal models, the first successful suture repair of a cardiac wound in a human was performed at the turn of the century.[4] Subsequently, Igelsbrud described the successful resuscitation of a patient sustaining cardiac arrest during a surgical procedure using emergent thoracotomy with open cardiac massage.[2] The utility of the emergent thoracotomy was beginning to be tested in a wide range of clinical scenarios in the early 1900s.

With improvement in patient resuscitation and an ongoing evaluation of patient outcomes, the indications for emergent thoracotomy shifted. Initially, cardiovascular collapse from medical causes was the most common reason for thoracotomy in the early 1900s. The demonstrated efficacy of closed-chest compression by Kouwenhoven et al[5] in 1960 and the introduction of external defibrillation in 1965 by Zoll et al[6] virtually eliminated the practice of open-chest resuscitation for medical cardiac arrest. Indications for emergent thoracotomy following trauma also became more limited. In 1943, Blalock and Ravitch[7] advocated the use of pericardiocentesis rather than thoracotomy as the preferred treatment for postinjury cardiac tamponade. In the late 1960s, however, refinements in cardiothoracic surgical techniques reestablished the role of immediate thoracotomy for salvaging patients with life-threatening chest wounds.[8] The use of temporary thoracic aortic occlusion in patients with exsanguinating abdominal hemorrhage further expanded the indications for emergent thoracotomy.[9,10] In the past two decades, critical analyses of patient outcomes following postinjury RT has tempered the unbridled enthusiasm for this technique, allowing a more selective approach with clearly defined indications.[11-16]

DEFINITIONS

The literature addressing RT appears confusing, likely due to widely varying terminology. As a result, there is a lack of agreement among physicians regarding the specific indications for RT as well as the definition of "signs of life."[17] In this chapter, RT refers to an emergent thoracotomy, most commonly performed in the ED for patients arriving in extremis. At times, an RT is performed in the operating room (OR) or intensive care unit (ICU) within hours after injury for delayed physiologic deterioration or an acute anatomic disruption or physiologic change. The value of RT for acute resuscitation may also be confusing because of the variety of indices used to characterize the patient's physiologic status prior to thoracotomy. Because there have been a wide range of indications for which RT has been performed in different trauma centers, comparisons in the literature are difficult. The authors define "no signs of life" as no detectable blood pressure, respiratory or motor effort, cardiac electrical activity, or pupillary activity (ie, clinical death). Patients with "no vital signs" have no palpable blood pressure but demonstrate electrical activity, respiratory effort, or pupillary reactivity.

PHYSIOLOGIC RATIONALE FOR RESUSCITATIVE THORACOTOMY

The primary objectives of RT are to (1) release pericardial tamponade, (2) control cardiac hemorrhage, (3) control intrathoracic bleeding, (4) evacuate bronchovenous air embolism, (5) perform open cardiac massage, and (6) temporarily occlude the descending thoracic aorta. Combined, these objectives attempt to address the primary issue of cardiovascular collapse from mechanical sources or extreme hypovolemia.

Release Pericardial Tamponade and Control Cardiac Hemorrhage (See Atlas Figure 25)

The highest survival rate following RT is in patients with penetrating cardiac wounds, especially when associated with pericardial tamponade.[8] Early recognition of cardiac tamponade, prompt pericardial decompression, and control of cardiac hemorrhage are the key components to successful RT and patient survival following penetrating wounds to the heart (see Chapter 30).[18] The egress of blood from the injured heart, regardless of mechanism, results in tamponade physiology. In acute conditions with a nondistendable pericardium, only 100 to 150 mL of pericardial blood results in tamponade. Rising intrapericardial pressure produces abnormalities in hemodynamic and cardiac perfusion that can be divided into three phases.[19] Initially, increased pericardial pressure restricts ventricular diastolic filling and reduces subendocardial blood flow.[20] Cardiac output under these conditions is maintained by compensatory tachycardia, increased systemic vascular resistance, and elevated central pressure (ie, ventricular filling pressure). In the intermediate phase of tamponade, rising pericardial pressure further compromises diastolic filling, stroke volume, and coronary perfusion, resulting in diminished cardiac output. Although blood pressure may be maintained deceptively well, subtle signs of shock (eg, anxiety, diaphoresis, and pallor) become evident. During the final phase of tamponade, compensatory mechanisms fail as the intrapericardial pressure approaches the ventricular filling pressure. Cardiac arrest ensues as profound coronary hypoperfusion occurs.

The classic description of clinical findings, Beck's triad (muffled heart sounds, distended neck veins, and hypotension), is rarely observed in the ED; therefore, a high index of suspicion in the at-risk patient sustaining penetrating torso trauma is crucial, with prompt intervention essential. In the first two phases of cardiac tamponade, patients may be aggressively managed with volume resuscitation to increase preload and pericardial drainage (via pericardiocentesis, pericardial window, or median sternotomy). The patient in the third phase of tamponade, with profound hypotension (systolic blood pressure [SBP] <60 mm Hg), should undergo RT rather than pericardiocentesis as the management for evacuation of pericardial blood. Following release of tamponade, the source of tamponade can be directly controlled with appropriate interventions based on the underlying injury

(see later section titled Technical Details of Resuscitative Thoracotomy).[21]

Control Intrathoracic Hemorrhage

Life-threatening intrathoracic hemorrhage occurs in less than 5% of patients following penetrating injury presenting to the ED, and in an even lower percentage of patients sustaining blunt trauma.[22] The most common injuries include penetrating wounds to the pulmonary hilum and great vessels; less commonly seen are torn descending thoracic aortic injuries with frank rupture or penetrating cardiac wounds exsanguinating into the thorax through a traumatic pericardial window. There is a high mortality rate for injuries to the pulmonary hilar vasculature or thoracic great vessels due to the lack of hemorrhage containment by adjacent tissue tamponade or by lack of vessel spasm (see Chapters 29 and 30). Either hemithorax can rapidly accommodate more than half of a patient's total blood volume before overt physical signs of hemorrhagic shock occur. Therefore, a high clinical suspicion is warranted in patients with penetrating torso trauma, particularly in those with hemodynamic decompensation. Patients with exsanguinating wounds require RT with rapid control of the source of hemorrhage if they are to be salvaged.

Perform Open Cardiac Massage

External chest compression provides approximately 20% to 25% of baseline cardiac output, with 10% to 20% of normal cerebral perfusion.[23] This degree of vital organ perfusion can provide reasonable salvage rates for 15 minutes, but few normothermic patients survive 30 minutes of closed-chest compression. Moreover, in models of inadequate intravascular volume (hypovolemic shock) or restricted ventricular filling (pericardial tamponade), external chest compression fails to augment arterial pressure or provide adequate systemic perfusion; the associated low diastolic volume and pressure result in inadequate coronary perfusion.[24] Therefore, closed cardiac massage is ineffective for postinjury cardiopulmonary arrest. The only potential to salvage the injured patient with ineffective circulatory status is immediate RT with subsequent open cardiac massage.

Achieve Thoracic Aortic Cross-Clamping (See Atlas Figure 24)

The rationale for temporary thoracic aortic occlusion in the patient with massive hemorrhage is twofold. First, in patients with hemorrhagic shock, aortic cross-clamping redistributes the patient's limited blood volume to the myocardium and brain.[10] Second, patients sustaining intra-abdominal injury may benefit from aortic cross-clamping due to reduction in subdiaphragmatic blood loss.[9] Temporary thoracic aortic occlusion augments aortic diastolic and carotid SBP, enhancing coronary as well as cerebral perfusion.[25] Experimental work has shown that the left ventricular stroke-work index and myocardial contractility increase in response to thoracic aortic occlusion during hypovolemic shock.[26] These improvements in myocardial function occur without an increase in the pulmonary capillary wedge pressure or a significant change in systemic vascular resistance. Thus, improved coronary perfusion resulting from an increased aortic diastolic pressure presumably accounts for the observed enhancement in contractility.[27]

These experimental observations suggest that temporary aortic occlusion is valuable in patients with either shock due to nonthoracic trauma or continued shock following the repair of cardiac injuries or other thoracic exsanguinating wounds. Indeed, occlusion of the descending thoracic aorta appears to increase the return of spontaneous circulation following CPR.[28] Reports of successful resuscitation using RT in patients in hemorrhagic shock exist, including patients sustaining cardiac arrest due to exsanguinating extremity or cervical injuries.[29] In these situations, RT may be a temporizing measure until the patient's circulating blood volume can be replaced by blood product transfusion. However, once the patient's blood volume has been restored, the aortic cross-clamp should be removed. Thoracic cross-clamping in the normovolemic patient may be deleterious because of increased myocardial oxygen demands resulting from the increased systemic vascular resistance.[30] Careful application of this technique is warranted as there is substantial metabolic cost and a finite risk of paraplegia associated with the procedure.[31-33] However, in carefully selected patients, aortic cross-clamping may effectively redistribute the patient's blood volume until external replacement and control of the hemorrhagic source are possible. Complete removal of the aortic cross-clamp or replacement of the clamp below the renal vessel should be performed within 30 minutes; the viscera's tolerance of normothermic ischemia is 30 to 45 minutes. As mentioned previously, there may be a role for REBOA in the patient with ongoing pelvic bleeding from either pelvic fracture or gunshot wound.

Evacuate Bronchovenous Air Embolism

Bronchovenous air embolism can be a subtle entity following thoracic trauma and may be much more common than is recognized.[34-36] The clinical scenario typically involves a patient sustaining penetrating chest injury who precipitously develops profound hypotension or cardiac arrest following endotracheal intubation and positive-pressure ventilation. Traumatic bronchovenous communications produce air emboli that migrate to the coronary arterial systems; any impedance in coronary blood flow causes myocardial ischemia and resultant shock. The production of air emboli is enhanced by the underlying physiology—there is relatively low intrinsic pulmonary venous pressure due to associated intrathoracic blood loss and high bronchoalveolar pressure from assisted positive-pressure ventilation. This combination increases the gradient for air transfer across bronchovenous channels.[37] Although more often observed in penetrating

trauma, a similar process may occur in patients with blunt lacerations of the lung parenchyma (see Chapter 28).

Immediate thoracotomy with pulmonary hilar cross-clamping prevents further propagation of pulmonary venous air embolism. Pericardotomy provides access to the coronaries and cardiac ventricles; with the patient in the Trendelenburg position (done to trap to air in the apex of the left ventricle), needle aspiration is performed to remove air from the cardiac chamber. Additionally, vigorous cardiac massage, done by running a finger along the coronary vessel, may promote dissolution of air already present in the coronary arteries.[36] Aspiration of the aortic root is done to alleviate any accumulated air pocket, and direct needle aspiration of the right coronary artery may be effective.

CLINICAL RESULTS FOLLOWING RESUSCITATIVE THORACOTOMY

The value of RT in resuscitation of the patient in profound shock who is not yet dead is unquestionable. Its indiscriminate use, however, renders it a low-yield and high-cost procedure.[15,38-40] In the past three decades, there has been a significant clinical shift in the performance of RT, from a nearly obligatory procedure before declaring any trauma patient dead to very few patients undergoing RT. During this swing of the pendulum, several groups have attempted to elucidate the clinical guidelines for RT. In 1979, we conducted a critical analysis of 146 consecutive patients undergoing RT and suggested a selective approach to its use in the moribund trauma patient, based on consideration of the following variables: (1) location and mechanism of injury, (2) signs of life at the scene and on presentation to the ED, (3) cardiac electrical activity at thoracotomy, and (4) SBP response to thoracic aortic cross-clamping.[40]

To further delineate these clinical parameters, we established a prospective study in which these data were carefully documented in all patients at the time of thoracotomy. In 1982, the first 400 patients were analyzed.[39] In 1998, the second analysis of 868 patients was reported.[41] The most recent review has summarized the data on 1708 patients who have undergone RT at the Denver Health Medical Center.[42] Of these, 1289 (78%) were dead in the ED, 272 (16%) died in the OR, and 41 (2%) succumbed to multiple organ failure in the surgical ICU. Ultimately, 106 patients (6%) survived. Although, this yield may seem low, it is important to emphasize that thoracotomy was done on virtually every trauma patient delivered to the ED in the early years of the study period. Additionally, 79% were without vital signs in the field and underwent prehospital CPR. In contrast, it is equally important to stress that patients without signs of life at the scene but who responded favorably to resuscitation were excluded from this analysis because they did not require RT; these patients remind the practitioner that prehospital clinical assessments may not always be reliable in triaging these severely injured patients. Indeed, the authors have salvaged a number of individuals sustaining blunt and penetrating trauma who were assessed to have no signs of life at the scene of injury. The importance of guidelines for RT is evident in the improved survival rate in recent years: in 1974 to 1979, survival was only 5%, compared to 14% survival in 2010 to 2014.[42]

The survival rate and percentage of neurologic impairment following RT vary considerably, due to the heterogeneity of patient populations reported in the literature. As previously discussed, critical determinants of survival include the mechanism and anatomic location of injury and the patient's physiologic condition at the time of thoracotomy.[43-46] We have attempted to elucidate the impact of these factors in ascertaining the success rate of RT by collating data from a number of clinical series reporting on 50 or more patients (Table 17-1). Unfortunately, inconsistencies in patient stratification and a paucity of clinical details limit objective analysis of these data. Although some reviews provide a specific breakdown of the injury mechanism and clinical status of patients presenting to the ED, others combine all injury mechanisms. We believe it is crucial to stratify patients according to the anatomic location (cervical/extremity vs torso) and mechanism of injury (penetrating vs blunt), as well as the presence of signs of life (ie, blood pressure, respiratory effort, cardiac electrical activity, and pupillary activity).

The data summarized to date confirm that RT has the highest survival rate following isolated cardiac injury (Table 17-1). An average of 35% of adult patients presenting in shock, defined as an SBP less than 70 mm Hg, and 20% without vital signs were salvaged after isolated penetrating injury to the heart if RT was performed. In contrast, 1% to 15% of blunt trauma patients undergoing RT survive. Following penetrating torso injuries, 14% of patients requiring RT are salvaged if they are hypotensive with detectable vital signs, whereas 8% of those who have no vital signs but have signs of life at presentation and 1% of those without signs of life are salvaged. These findings are reiterated by a recent report incorporating all patients undergoing RT for either blunt or penetrating mechanism from 24 separate studies[44]; survival rates for patients undergoing RT for penetrating injuries and blunt mechanisms were 8.8% and 1.4%, respectively. Additionally, more patients survive RT for isolated cardiac wounds (19.4%), followed by stab wounds (16.8%) and gunshot wounds (4.3%).

Although there is a clear role for RT in the patient presenting in shock but with measurable vital signs, there is disagreement about its use in the patient population undergoing CPR prior to arrival in the ED. Although there have been multiple reports with low survival rates and dismal outcomes following prehospital CPR, termination of resuscitation in the field should not be performed in all patients.[47] Our most recent evaluation, spanning 40 years of experience, indicates that RT does play a significant role in the critically injured patient undergoing prehospital CPR.[42] The majority of patients arriving in extremis who survived to discharge sustained a stab wound to the torso, consistent with previous reports. Additionally, the majority of patients were neurologically intact at discharge.

TABLE 17-1: Outcome Following Emergency Department Thoracotomy in Adults

Injury pattern	Shock	No vital signs	No signs of life	Total
Cardiac				
Denver[91]	3/9 (33%)	0/7 (0%)	1/53 (2%)	4/69 (6%)
Detroit[92]	9/42 (21%)	3/110 (3%)		12/152 (8%)
Johannesburg[93]				13/108 (12%)
Los Angeles[94]	2/5 (40%)	6/11 (55%)	2/55 (4%)	10/71 (14%)
New York[95]	7/20 (35%)	18/53 (32%)	0/18 (0%)	24/91 (26%)
San Francisco[45]	18/37 (49%)	0/25 (0%)		18/63 (29%)
Seattle[96]	4/11 (36%)	11/47 (23%)		15/58 (26%)
Overall	43/124 (35%)	47/254 (19%)	4/126 (3%)	96/612 (16%)
Penetrating				
Denver[41]	19/78 (24%)	14/399 (4%)		33/477 (7%)
Detroit[92]	9/42 (21%)	3/110 (3%)		12/152 (8%)
Houston[97]	14/156 (9%)	18/162 (11%)		32/318 (10%)
Indianapolis[98]	3/7 (43%)	1/50 (2%)	0/80 (0%)	4/137 (3%)
Johannesburg[93]	31/413 (8%)	10/149 (7%)	1/108 (1%)	42/670 (6%)
Los Angeles[94]	2/5 (40%)	6/11 (55%)	2/55 (4%)	10/71 (14%)
New York[99]	8/32 (25%)	8/77 (10%)	0/25 (0%)	16/134 (12%)
Oakland[100]	8/24 (33%)		2/228 (1%)	10/252 (4%)
San Francisco[45]				32/198 (30%)
Seattle[96]	4/11 (36%)	11/47 (23%)		15/58 (25%)
Washington[101]	7/13 (54%)	3/47 (6%)		10/60 (17%)
Toronto[15]				3/96 (3%)
Denver[42]				11/88 (12.5%)
Overall	145/1007 (14%)	100/1252 (8%)	6/615 (1%)	297/3170 (9%)
Blunt				
Denver[41]	4/86 (5%)	4/311 (1%)		8/397 (2%)
Houston[97]	0/42 (0%)	0/27 (0%)		0/69 (0%)
Johannesburg[93]	1/109 (1%)	0/39 (0%)	0/28 (0%)	1/176 (1%)
San Francisco[45]				1/60 (2%)
Seattle[102]				1/88 (1%)
Denver[42]				13/89 (15%)
Overall	5/237 (2%)	4/377 (1%)	0/28 (0%)	24/879 (2.7%)

To further define the limits of RT, a prospective, multicenter trial was performed by the Western Trauma Association (WTA).[13] The WTA data substantiate that injury mechanism alone is not a discriminator of futility. Specifically, with the exception of an overtly devastating head injury, blunt trauma does not prohibit meaningful survival, even with requirements for CPR. This multicenter experience, combined with prior reports, suggests current indications for RT (Table 17-2). RT should be performed for penetrating nontorso trauma with CPR of less than 5 minutes, blunt trauma with CPR of less than 10 minutes, and penetrating torso trauma with CPR of less than 15 minutes. RT is unlikely to yield productive survival when patients (1) sustain blunt trauma and require more than 10 minutes of prehospital CPR, (2) have penetrating wounds and undergo more than 15 minutes of prehospital CPR, (3) have isolated extremity trauma with more than 5 minutes of prehospital CPR, or (4) manifest asystole without pericardial tamponade. We recognize, however, that there will invariably be exceptions to the recorded literature.[29,43,48,49]

Emerging data indicate the clinical results in the pediatric population mirror those of the adult experience for penetrating injury (Table 17-3). One might anticipate that children would have a more favorable outcome compared to adults; however, this has not been borne out in multiple studies.[50-56] A recent review of RT in the pediatric population encompassed eight published reports with 2336 pediatric trauma patients[57]; 269 RTs were performed in this study population. Overall survival rate was 1.7% in blunt trauma and 14% following RT for penetrating trauma. According to this review, in the past 25 years, no published series of RT has demonstrated a survivor following blunt trauma in the pediatric age group. In the published literature over the past four decades, only two blunt trauma patients are reported to have undergone

TABLE 17-2: Current Indications and Contraindications for Emergency Department Thoracotomy

Indications:

Salvageable postinjury cardiac arrest
- Penetrating thoracic trauma patients with <15 min of prehospital CPR
- Penetrating nonthoracic trauma patients with <5 min of prehospital CPR
- Blunt trauma patients with <10 min of prehospital CPR

Persistent severe postinjury hypotension (SBP <60 mm Hg) due to:
- Cardiac tamponade
- Hemorrhage: intrathoracic, intra-abdominal, extremity, cervical
- Air embolism

Contraindications:
- CPR >15 min following penetrating injury and no signs of life (pupillary response, respiratory effort, or motor activity)
- CPR >10 min following blunt injury and no signs of life
- Asystole is the presenting rhythm and there is no pericardial tamponade

CPR, cardiopulmonary resuscitation; SBP, systolic blood pressure.

RT and survived; each of these patients was over the age of 15. Beaver et al[52] reported no survivors among 27 patients, from 15 months to 14 years of age, undergoing postinjury RT at Johns Hopkins Hospital. Powell et al,[55] at the South Alabama Medical Center, described an overall survival of 20% (3 of 15 patients) in patients ranging from 4 to 18 years old. A review using the National Trauma Data Bank of all pediatric blunt trauma patients with no sign of life in the field identified a 1.3% survival rate for those undergoing RT.[56] In a study at Denver Health Medical Center, encompassing an 11-year experience with 689 consecutive RTs, we identified 83 patients (12%) who were under 18 years old.[53] Survival by injury mechanism was 9% (1 of 11 patients) for stab wounds, 4% (1 of 25 patients) for gunshot wounds, and 2% (1 of 47 patients) for blunt trauma. Among 69 patients presenting to the ED without vital signs, only 1 patient (1%) survived (with a stab wound). This contrasted to a salvage of 2 (14%) of 14 patients with vital signs. The outcome in blunt trauma, the predominant mechanism of lethal injury in children, was disappointing, with only 2% salvage, and there were no survivors when vital signs were absent. A more recent analysis spanning 40 years identified a similar rate of RT in the pediatric population[58]; of 179 trauma patients undergoing RT, there were 6 survivors (3.4%). In the adolescent age group (15–18 years old), survival rate was 4.8%, whereas in the group younger than age 15 years, there were no survivors. Thus, as in adults, outcome following RT in the adolescent population is largely determined by injury mechanism and physiologic status on presentation to the ED; for patients under 15 years of age, there are only isolated cases of survival following penetrating trauma and no apparent survival benefit following blunt trauma.

In sum, overall analysis of the available literature indicates that the success of RT approximates 35% in the patient arriving in shock with a penetrating cardiac wound and 15% for all penetrating wounds. Patients undergoing CPR upon arrival to the ED should be stratified based on injury and transport time to determine the utility of RT. Conversely, patient outcome is relatively poor when RT is done for blunt trauma: 2% survival in patients in shock and less than 1% survival with no vital signs. A clear understanding of the indications for RT combined with available expertise will hopefully improve compliance with commonly accepted clinical practice and result in reduced mortality rates.[59]

TABLE 17-3: Outcome Following Emergency Department Thoracotomy in Children

Injury pattern	Shock	No vital signs	No signs of life	Total
Penetrating				
Baltimore[52]		0/2 (0%)		0/2 (0%)
Denver[53]	1/3 (33%)	1/5 (20%)	0/28 (0%)	2/36 (6%)
Mobile[55]	0/1 (0%)	3/9 (33%)		3/10 (30%)
Sacramento[54]	1/4 (25%)	0/4 (0%)		1/8 (13%)
Overall	2/8 (25%)	4/20 (20%)	0/28 (0%)	6/56 (11%)
Blunt				
Baltimore[52]		0/15 (0%)		0/15 (0%)
Denver[53]	1/11 (9%)	0/6 (0%)	0/30 (0%)	1/47 (2%)
Mobile[55]		0/5 (0%)		0/5 (0%)
Sacramento[54]	0/6 (0%)	0/9 (0%)		0/15 (0%)
NTDB[56]			6/499 (1.3%)	6/499 (1.3%)
Europe[50]	1/17 (6%)	0/10 (0%)		0/10 (0%)
Overall		0/35 (0%)	6/529 (1%)	7/591 (1.2%)

NTDB, National Trauma Data Bank.

INDICATIONS FOR RESUSCITATIVE THORACOTOMY

Based on our successive years of RT prospective analysis[42] and the recent WTA multicenter trial,[12] we propose current indications for RT (Table 17-2). Clearly, the specific application of these guidelines must include signs of life, mechanism of injury, and logistic issues such as qualified personnel. Our current decision algorithm for resuscitation of the moribund trauma patient and use of RT was formulated based on these analyses (Fig. 17-1). At the scene, patients in extremis without electrical cardiac activity are declared dead.[60] Patients in extremis but with electrical cardiac activity are intubated, supported with cardiac compression, and rapidly transported to the ED.

On arrival to the ED, the time from initiation of CPR is reported by the prehospital personnel to the trauma surgeon; blunt trauma patients with greater than 10 minutes of prehospital CPR and no signs of life are declared, whereas penetrating torso trauma patients with greater than 15 minutes or isolated extremity/cervical trauma patients with greater than 5 minutes of prehospital CPR and no signs of life are pronounced. Patients within the time guidelines or those with signs of life trigger ongoing resuscitation and RT. After performing a generous left anterolateral thoracotomy and subsequent pericardotomy, the patient's intrinsic cardiac activity is evaluated. Patients in asystole without associated cardiac tamponade are declared. Patients with a cardiac wound, tamponade, and associated asystole are aggressively treated; the cardiac wound is repaired first, followed by manual cardiac compressions, intracardiac injection of epinephrine, and defibrillation. Following treatment and blood product resuscitation, one should reassess salvageability, typically defined as the patient's ability to generate an SBP greater than 70 mm Hg.

Patients with an intrinsic rhythm following RT should be treated according to underlying pathology. Those with tamponade should undergo cardiac repair, either in the trauma bay or in the OR (see Chapter 30). Control of intrathoracic hemorrhage may entail hilar cross-clamping, digital occlusion of the direct injury, or even packing of the apices for subclavian injuries. Treatment of bronchovenous air embolism includes cross-clamping of the hilum, putting the patient in Trendelenberg position, aspirating the left ventricle and aortic root, and massaging the coronaries. Finally, cross-clamping of the descending aorta is performed to decrease the required effective circulating volume, increase cerebral and coronary blood flow, reduce infradiaphragmatic blood loss, and facilitate resuscitation. In all of these scenarios, reassessment of the patient following intervention and aggressive resuscitation efforts is performed, with the goal SBP of 70 mm Hg used to define salvageability.

TECHNICAL DETAILS OF RESUSCITATIVE THORACOTOMY

The optimal benefit of RT is achieved by a surgeon experienced in the management of intrathoracic injuries. The emergency physician, however, should not hesitate to perform the

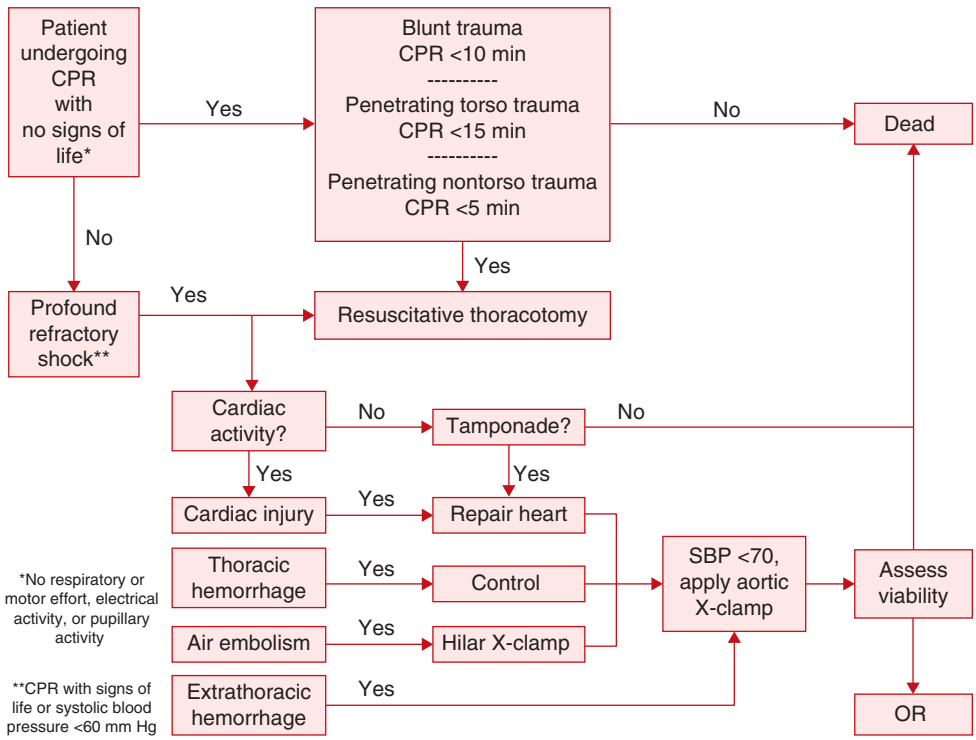


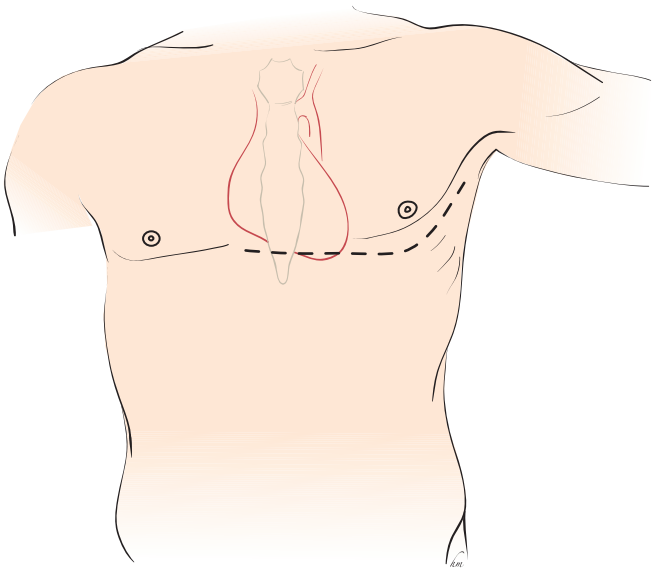
FIGURE 17-1 Algorithm directing the use of resuscitative thoracotomy in the multiply injured trauma patient. CPR, cardiopulmonary resuscitation; OR, operating room; X-clamp, cross-clamp.

procedure in the moribund patient with a penetrating chest wound when thoracotomy is the only means of salvage. The technical skills needed to perform the procedure include the ability to perform a rapid thoracotomy, pericardiotomy, cardiorrhaphy, and thoracic aortic cross-clamping; familiarity with vascular repair techniques and control of the pulmonary hilum are advantageous. Once life-threatening intrathoracic injuries are controlled or temporized, the major challenge is restoring the patient's hemodynamic integrity and minimizing vital organ reperfusion injury.

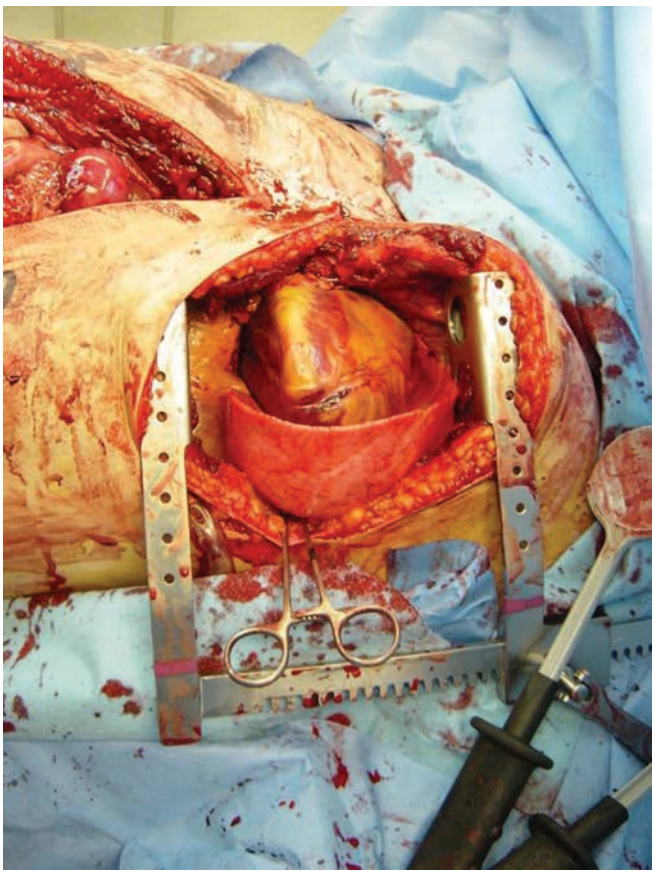
Thoracic Incision

A left anterolateral thoracotomy incision is preferred for RT. Advantages of this incision in the critically injured patient include (1) rapid access with simple instruments, (2) the ability to perform this procedure on a patient in the supine position, and (3) easy extension into the right hemithorax, a clamshell thoracotomy, for exposure of both pleural spaces and anterior and posterior mediastinal structures. The key resuscitative maneuvers of RT, namely, pericardiotomy, open cardiac massage, and thoracic aortic cross-clamping, are readily accomplished via this approach. The initial execution of a clamshell thoracotomy should be done in hypotensive patients with penetrating wounds to the right chest. This provides immediate, direct access to a right-sided pulmonary or vascular injury while still allowing access to the pericardium from the left side for open cardiac massage. Clamshell thoracotomy may also be considered in patients with presumed air embolism, providing access to the cardiac chambers for aspiration, coronary vessels for massage, and bilateral lungs for obliteration of the source.

Preparation for RT should be performed well ahead of the patient's arrival. Set-up should include a 10-blade scalpel, Finochietto chest retractor, toothed forceps, curved Mayo scissors, Satinsky vascular clamps (large and small), long needle holder, Lebsche knife and mallet, and internal defibrillator paddles. Sterile suction, skin stapler, and access to a variety of sutures should be available (specifically 3-0 Prolene on a CT-1 needle, 3-0 silk ties, and Teflon pledgets). Upon patient arrival and determination of the need for RT, the patient's left arm should be placed above the head to provide unimpeded access to the left chest. The anterolateral thoracotomy is initiated with an incision at the level of the fifth intercostal space (Fig. 17-2). Clinically, this level for incision corresponds to the inferior border of the pectoralis major muscle, just below the patient's nipple. In women, the breast should be retracted superiorly to gain access to this interspace, and the incision is made at the inframammary fold. The incision should start on the right side of the sternum; if sternal transection is required, this saves the time-consuming step of performing an additional skin incision. As the initial incision is carried transversely across the chest and one passes beneath the nipple, a gentle curve in the incision toward the patient's axilla rather than direct extension to the bed should be performed; this curvature in the skin correlates with the natural curvature of the rib cage. The skin, subcutaneous fat, and

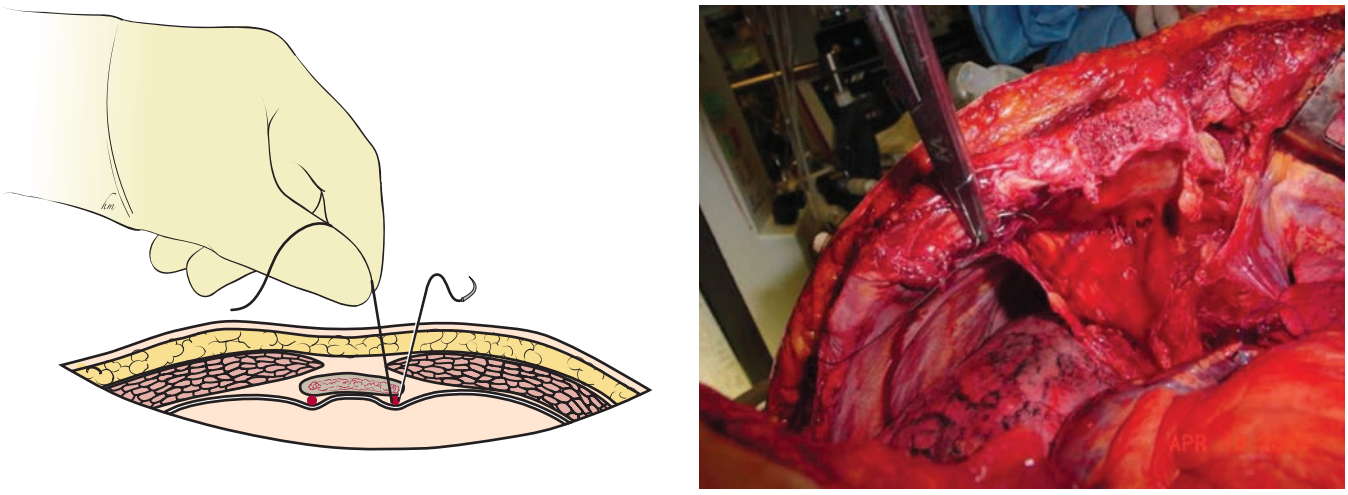


A



B

FIGURE 17-2 (**A, B**) The thoracotomy incision is performed through the fourth or fifth intercostal space; the incision should start to the right of the sternum, and begin curving into the axilla at the level of the left nipple. The Finochietto rib retractor is placed with the handle directed inferiorly toward the bed.



A B

FIGURE 17-3 (**A, B**) Transverse division of the sternum requires individual ligation of the internal mammary arteries.

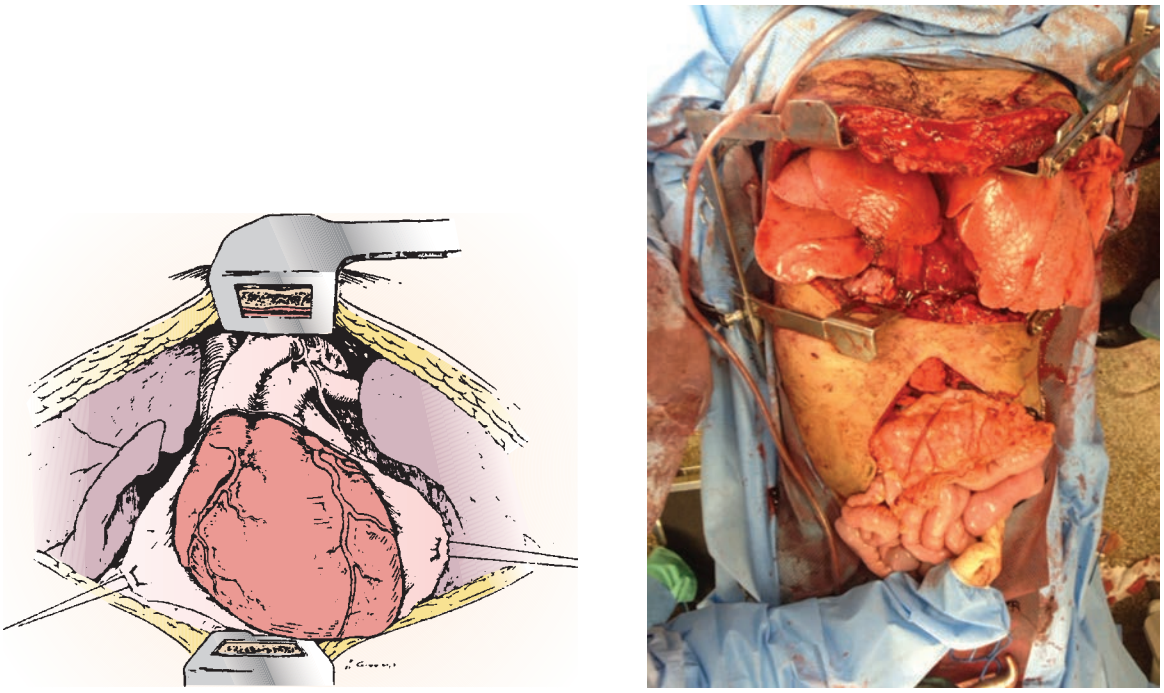
chest wall musculature are incised with a knife to expose the ribs and associated intercostal space. Intercostal muscles and the parietal pleura are then divided in one layer with curved Mayo scissors; the intercostal muscle should be divided along the superior margin of the rib to avoid the intercostal neurovascular bundle. Chest wall bleeding is minimal in these patients and should not be a concern at this point in the resuscitation. Once the incision is completed and the chest entered, a standard Finochietto rib retractor is inserted, with the handle directed inferiorly toward the axilla (Fig. 17-2) (see Atlas Figures 22 and 23). Placement of the handle toward the bed rather than the sternum allows extension of the left thoracotomy into a clamshell thoracotomy with crossing of the sternum without replacing the rib retractor.

If the left anterolateral thoracotomy does not provide adequate exposure, several techniques may be employed. The costal cartilages can be readily divided with heavy scissors. Alternatively, the sternum can be transected for additional exposure with a Lebsche knife; care must be taken to hold the Lebsche knife firmly against the underside of the sternum when using the mallet to forcefully transect the sternum, or the tip of the instrument may deviate and result in an iatrogenic cardiac injury. If the sternum is divided transversely, the internal mammary vessels must be ligated when perfusion is restored; this may be performed using either a figure of eight suture with 3-0 silk or by clamping the vessel with a tonsil and individually ligating it with a 3-0 silk tie (Fig. 17-3). A concomitant right anterolateral thoracotomy produces a "clamshell" or "butterfly" incision and achieves wide exposure to both pleural cavities and anterior and posterior mediastinal structures (Fig. 17-4). Once the right pleural space is opened, the rib retractor should be moved to more of a midline position to enhance separation of the chest wall for maximal exposure. When visualization of penetrating wounds in the aortic arch or major branches is needed, the superior sternum is additionally split in the midline.

Pericardiotomy and Cardiac Hemorrhage Control

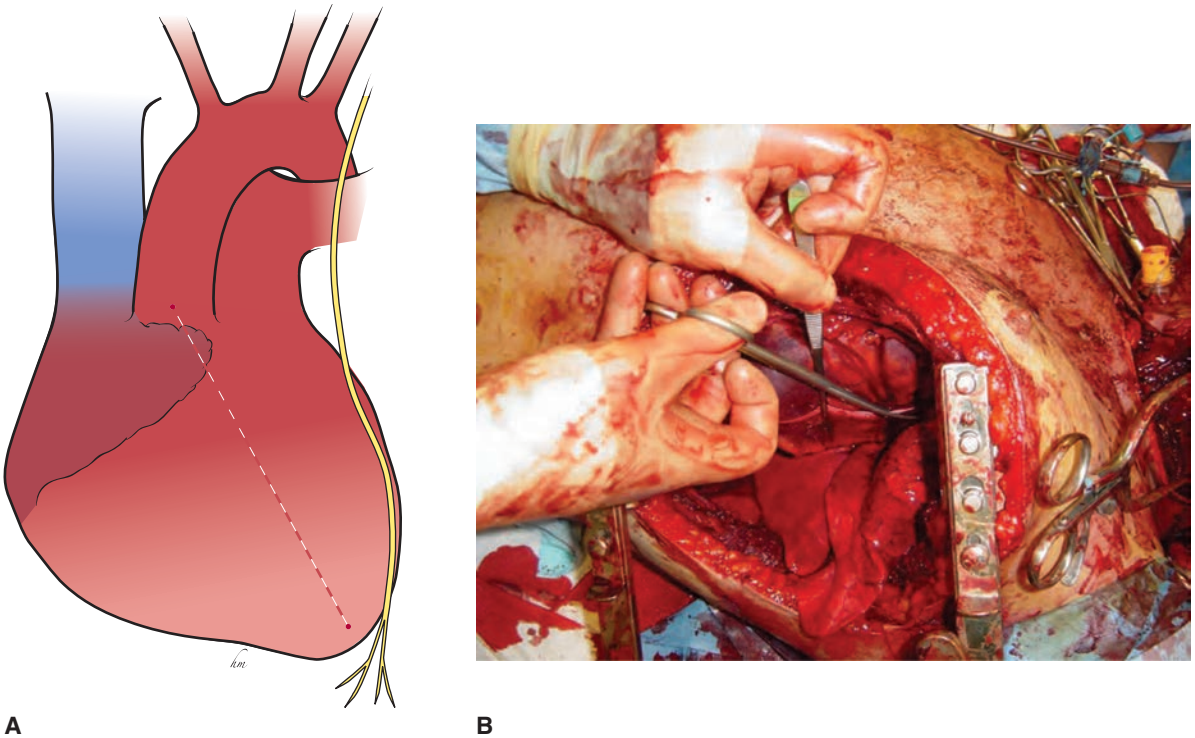
The pericardium is incised widely, starting at the cardiac apex and extending toward the sternal notch, anterior and parallel to the phrenic nerve (Fig. 17-5). If the pericardium is not tense with blood, it may be picked up at the apex with toothed forceps and sharply opened with scissors. If tense pericardial tamponade exists, a knife or the sharp point of a scissors is often required to initiate the pericardiotomy incision. Blood and blood clots should be completely evacuated from the pericardium. The heart should be delivered from the pericardium by placing the right hand through the opening in the pericardium posterior to the heart, encircling the right side of the heart and pulling it into the left chest. This effectively places the left side of the pericardium behind the heart allowing access to the cardiac chambers for repair of cardiac wounds and access for effective open cardiac massage.

Prompt hemorrhage control is paramount for a cardiac injury. In the beating heart, cardiac bleeding sites should be controlled immediately with digital pressure on the surface of the ventricle and partially occluding vascular clamps on the atrium or great vessels. Efforts at definitive cardiorrhaphy may be delayed until initial resuscitative measures have been completed. In the nonbeating heart, cardiac repair is done prior to defibrillation and cardiac massage. Cardiac wounds in the thin-walled right ventricle are best repaired with 3-0 nonabsorbable running or horizontal mattress sutures. Buttressing the suture repair with Teflon pledgets is ideal for the thinner right ventricle. When suturing a ventricular laceration, care must be taken not to incorporate a coronary vessel into the repair. In these instances, vertical mattress sutures should be used to exclude the coronary and prevent cardiac ischemia. In the more muscular left ventricle, control of bleeding can occasionally be achieved with a skin-stapling device if the wound is a linear stab wound whose edges coapt in diastole.



A B

FIGURE 17-4 (**A, B**) A bilateral anterolateral ("clamshell") thoracotomy provides access to both thoracic cavities including the pulmonary hila, heart, and proximal great vessels.



A B

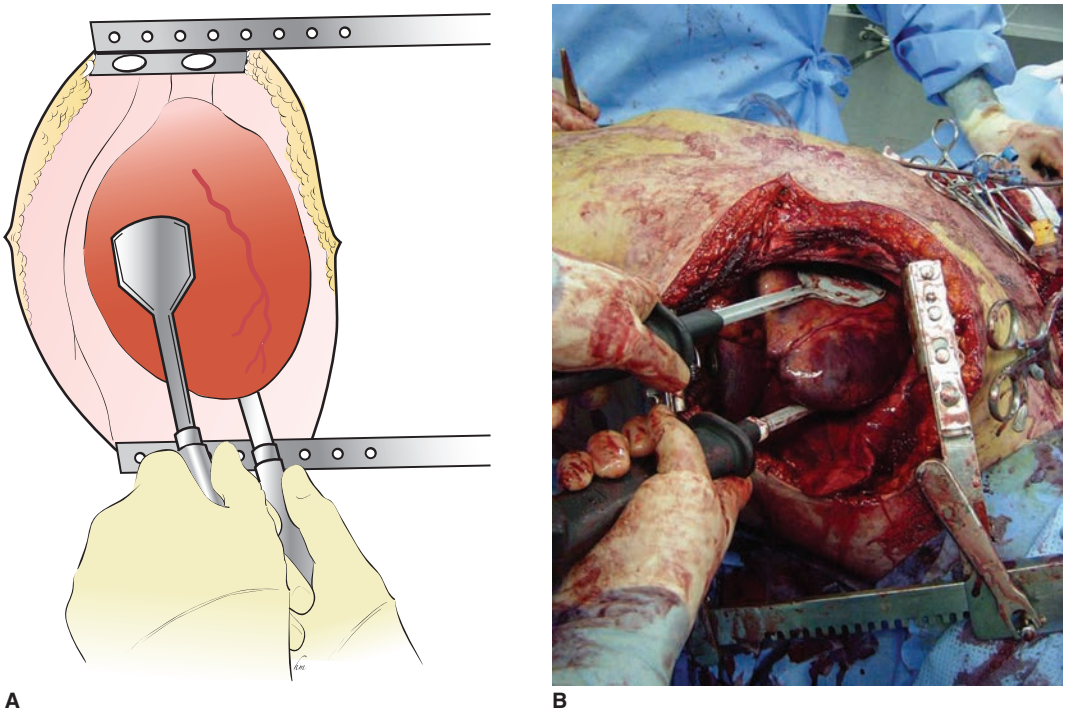
FIGURE 17-5 (**A, B**) Pericardiotomy is done with toothed pickups and curved Mayo scissors; the incision begins at the cardiac apex, anterior to the phrenic nerve, and extends on the anterior surface of the heart toward the great vessels.

Low-pressure venous, atrial, and atrial appendage lacerations can be repaired with simple running or purse string sutures of 3-0 Prolene. Posterior cardiac wounds may be particularly treacherous when they require elevation of the heart for their exposure; closure of these wounds is best accomplished in the OR with optimal lighting and equipment. For a destructive wound of the ventricle or for inaccessible posterior wounds, temporary inflow occlusion of the superior and inferior vena cava may be employed to facilitate repair (see Chapter 30). BioGlue may be used as a hemostatic agent in such cases. Use of a Foley catheter for temporary occlusion of cardiac injuries has been suggested; in our experience, this may inadvertently extend the injury due to traction forces.

Advanced Cardiac Life Support Interventions Including Cardiac Massage

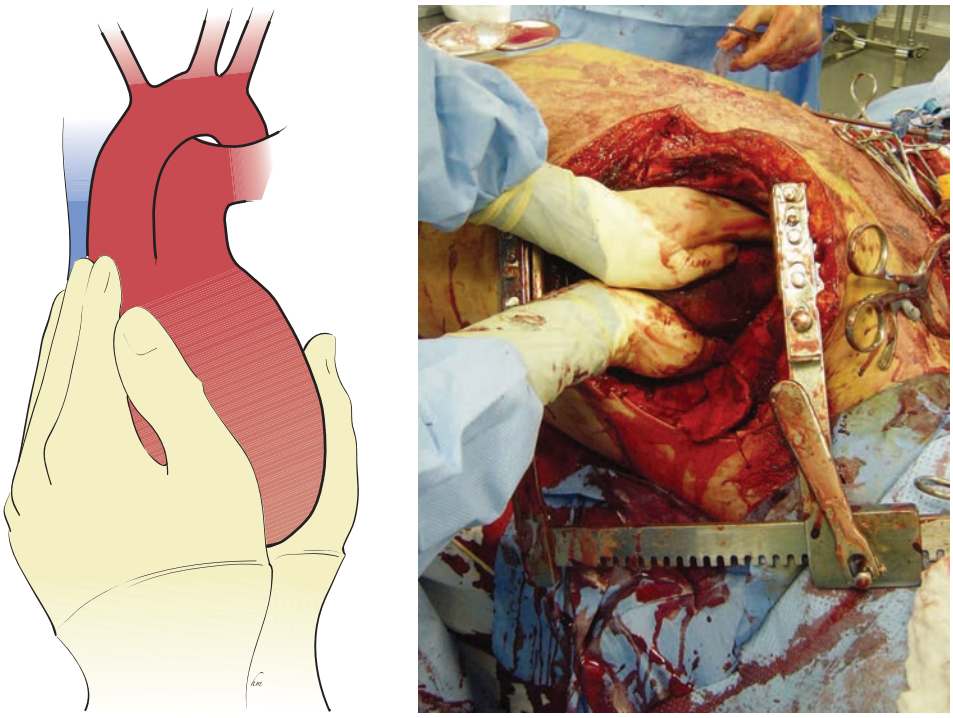
The restoration of organ and tissue perfusion may be facilitated by a number of interventions.[61] First, a perfusing cardiac rhythm must be established. Early defibrillation for ventricular fibrillation or pulseless ventricular tachycardia has proven benefit, and evidence supports the use of amiodarone (with lidocaine as an alternative) following epinephrine in patients refractory to defibrillation. Magnesium may be beneficial for torsades de pointes; other dysrhythmias should be treated according to current guidelines.[61] Familiarity with the internal cardiac paddles and appropriate charging dosages in joules are required (Fig. 17-6). In the event of cardiac arrest, bimanual internal massage of the heart should be instituted promptly (Fig. 17-7). We prefer to do this with a hinged clapping motion of the hands, with the wrists apposed, sequentially closing from palms to fingers. The ventricular compression should proceed from the cardiac apex to the base of the heart. The two-handed technique is strongly recommended, as the one-handed massage technique poses the risk of myocardial perforation with the thumb.

Pharmacologic adjuncts to increase coronary and cerebral perfusion pressure may be needed. The first agent in resuscitation at this juncture is intracardiac epinephrine. Epinephrine should be administered using a specialized syringe, which resembles a spinal needle, directly into the left ventricle. Typically, the heart is lifted up slightly to expose the more posterior left ventricle, and care is taken to avoid the circumflex coronary during injection. Although epinephrine continues to be advocated during resuscitation, there is a growing body of data suggesting that vasopressin may be superior to epinephrine in augmenting cerebral perfusion and other vital organ blood flow.[62] Administration of calcium, while theoretically deleterious during reperfusion injury, increases cardiac contractility and may be helpful in the setting of hypocalcemia produced by blood component transfusion (see Chapter 16). Although metabolic acidosis is common following RT and resuscitation,[63] the mainstay of therapy is provision of adequate alveolar ventilation and restoration of tissue perfusion. Sodium bicarbonate therapy has not been proven beneficial in facilitating defibrillation, restoring spontaneous circulation, or improving survival. However, it may be warranted following protracted arrest or resuscitation because catecholamine receptors may be desensitized with advanced metabolic acidosis.



A B

FIGURE 17-6 (**A, B**) Internal paddles for defibrillation are positioned on the anterior and posterior aspects of the heart.



A B

FIGURE 17-7 (**A, B**) Open cardiac massage is performed with a two-handed hinged technique; the clapping motion sequentially closes the hands from palms to fingers.

Thoracic Aortic Occlusion and Pulmonary Hilar Control

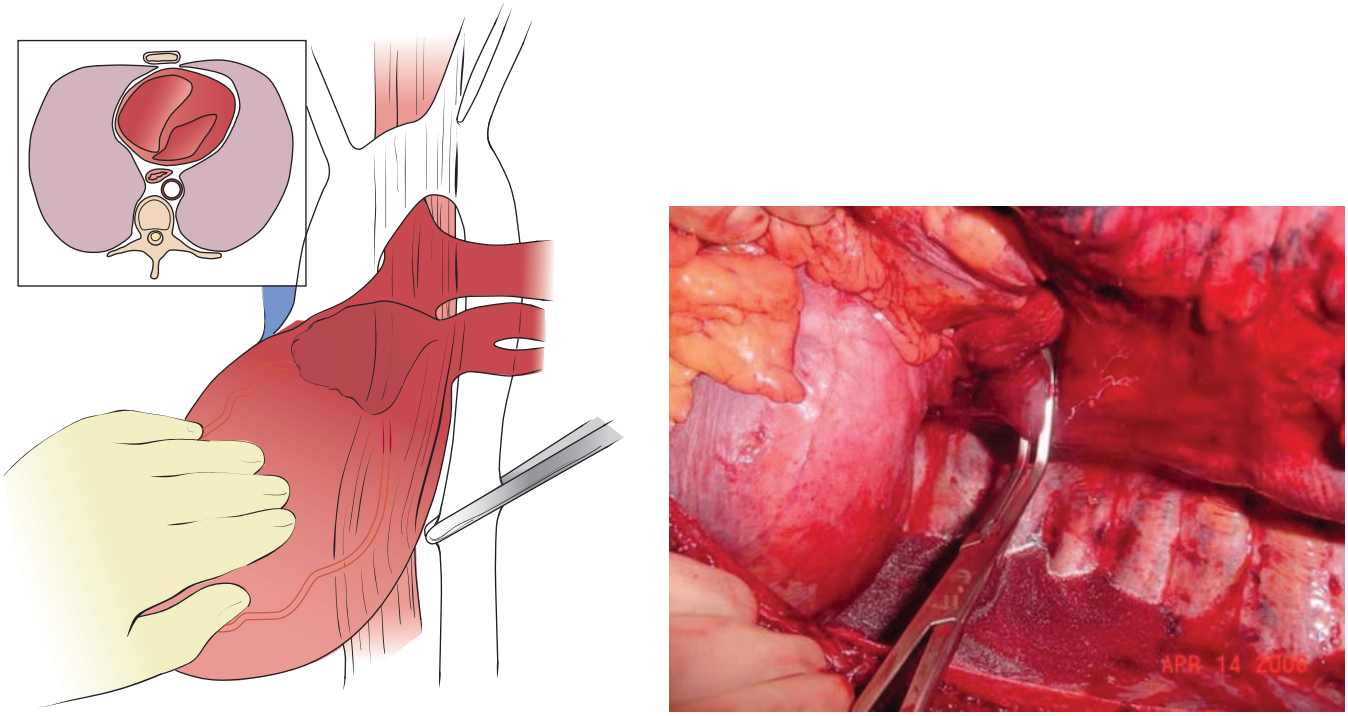
Following thoracotomy and pericardiotomy with evaluation of the heart, the descending thoracic aorta should be occluded to maximize coronary perfusion if hypotension (SBP <70 mm Hg) persists. We prefer to cross-clamp the thoracic aorta inferior to the left pulmonary hilum (Fig. 17-8). Exposure of this area is best provided by elevating the left lung anteriorly and superiorly. Although some advocate taking down the inferior pulmonary ligament to better mobilize the lung, this is unnecessary and risks injury to the inferior pulmonary vein. Dissection of the thoracic aorta is optimally performed under direct vision by incising the mediastinal pleura and bluntly separating the aorta from the esophagus anteriorly and from the prevertebral fascia posteriorly. Care should be taken in dissecting the aorta, and completely encircling it may avulse thoracic and other small vascular branches. Alternatively, if excessive hemorrhage limits direct visualization, which is the more realistic clinical scenario, blunt dissection with one's thumb and fingertips can be done to isolate the descending aorta. Once identified and isolated, the thoracic aorta is occluded with a large Satinsky or DeBakey vascular clamp. If the aorta cannot be easily isolated from the surrounding tissue, digitally occlude the aorta against the spine to effect aortic occlusion. Although occlusion of the thoracic aorta is typically performed after pericardiotomy, this may be the first maneuver upon entry into the chest in patients sustaining extrathoracic injury and associated major blood loss.

Control of the pulmonary hilum has two indications. First, if coronary or systemic air embolism is present, further embolism is prevented by placing a vascular clamp across the pulmonary hilum (Fig. 17-9). Associated maneuvers such as vigorous cardiac massage to move air through the coronary arteries and needle aspiration of air from the left ventricular apex and the aortic root are also performed (Fig. 17-10). Second, if the patient has a pulmonary hilar injury or marked hemorrhage from the lung parenchyma, control of the hilum may prevent exsanguination. Hilar control is best performed with a large Satinsky clamp or temporarily with digital control (see Chapter 30).

COMPLICATIONS AND CONSEQUENCES OF RESUSCITATIVE THORACOTOMY

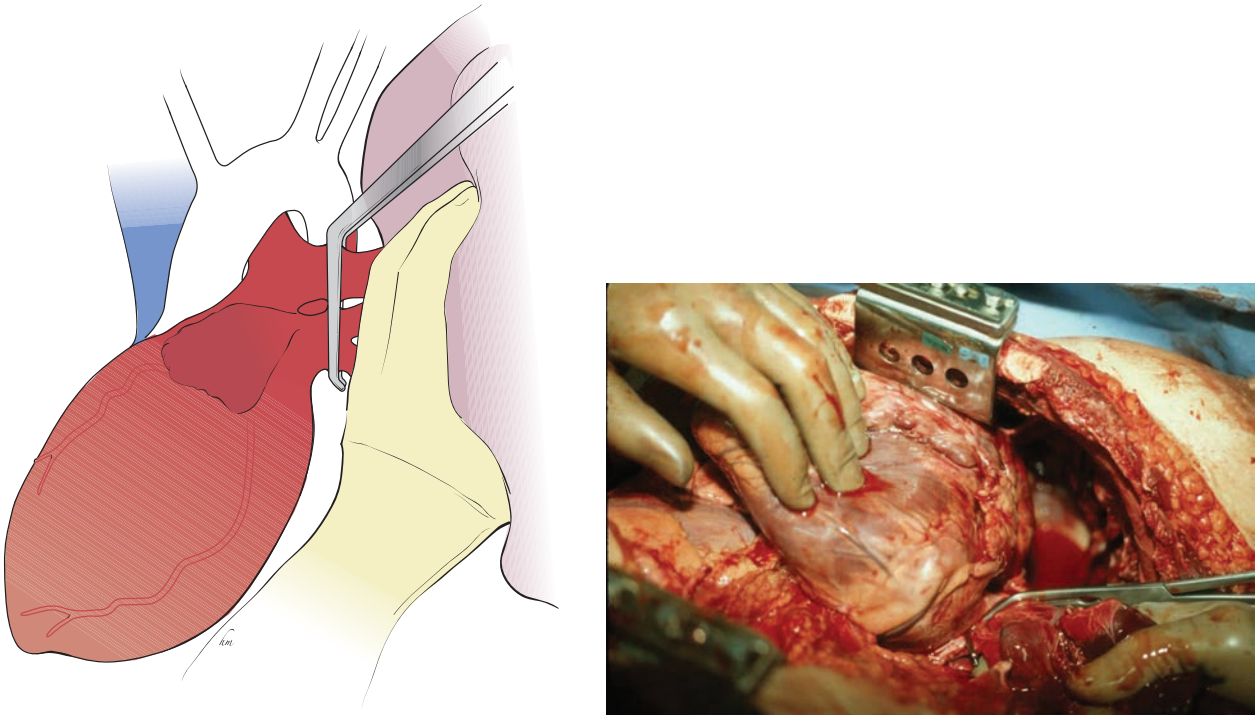
Procedural Complications

Technical complications of RT involve virtually every intrathoracic structure. The list of such misadventures includes lacerations of the heart, coronary arteries, aorta, phrenic nerves, esophagus, and lungs, as well as avulsion of aortic branches to components of the mediastinum. Previous thoracotomy virtually assures technical problems from the presence of dense pleural adhesions and is therefore a relative contraindication to RT. Additional postoperative morbidity among ultimate survivors of RT includes recurrent chest bleeding; infection



A B

FIGURE 17-8 (**A, B**) Aortic cross-clamp is applied with the left lung retracted superiorly, below the inferior pulmonary ligament, just above the diaphragm. The flaccid aorta is identified as the first structure encountered on top of the spine when approached from the left chest.



A B

FIGURE 17-9 (**A, B**) A Satinsky clamp is used to clamp the pulmonary hilum for hemorrhage control or to prevent further bronchovenous air embolism.

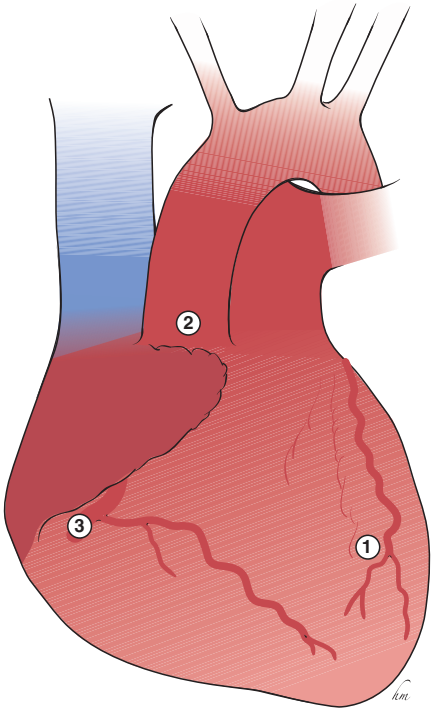


FIGURE 17-10 In cases of bronchovenous air embolism, sequential sites of aspiration include the left ventricle (1), the aortic root (2), and the right coronary artery (3).

of the pericardium, pleural spaces, sternum, and chest wall; and postpericardiotomy syndrome.

Importantly, there is a finite risk to the health care providers and trauma team performing an RT.[64] The use of RT by necessity involves the rapid use of sharp surgical instruments and exposure to the patient's blood. Even during elective procedures in the OR, the contact rate of patient's blood with the surgeon's skin can be as high as 50%, and the contact rate of patients' blood with health care workers' blood is as high as 60%. The overall seroprevalence rate of HIV among patients admitted to the ED for trauma is around 4%, but is much higher among the subgroup of patients most likely to require an RT, for example, 14% of penetrating trauma victims and nearly 30% of intravenous drug abusers. Caplan et al[65] found that 26% of acutely injured patients had evidence of exposure to HIV (4%), hepatitis B (20%), or hepatitis C virus (14%); there was no difference in the incidence comparing blunt to penetrating trauma. Thus, the likelihood of a health care worker sustaining exposure to HIV or hepatitis in the ED is substantial. The risk of contagion from exposures to HIV and other bloodborne pathogens can be minimized by the use of appropriate barrier precautions, minimizing the use of needles and scalpels, and selective use of RT.

Hemodynamic and Metabolic Consequences of Aortic Cross-Clamping

Aortic cross-clamping may be lifesaving during acute resuscitation, but there is a finite cost to the patient. Occlusion of the aorta results in an increase in blood pressure, but there is an associated 90% reduction in femoral artery SBP; in addition, abdominal visceral blood flow decreases to 2% to 8% of baseline values.[32,33] Therefore, cross-clamping magnifies the metabolic cost of shock by reducing local blood flow to abdominal viscera even further. This results in tissue acidosis and increased oxygen debt, and may ultimately contribute to postischemic multiple organ failure.[33] Additionally, return of aortic flow may not result in normalization of flow to vital organs; in animal models, blood flow to the kidneys remained at 50% of baseline despite a normal cardiac output. The metabolic penalty of aortic cross-clamping becomes exponential when the normothermic occlusion time exceeds 30 minutes, both in trauma and in elective thoracic aortic procedures.[66,67] Hypoxia of distal organs, white blood cells, and endothelium induces the elaboration, expression, and activation of inflammatory cell adhesion molecules and inflammatory mediators; this systemic inflammatory response syndrome has been linked to impaired pulmonary function and multiple organ failure[68] (see Chapter 63). Consequently, the aortic clamp should be removed as soon as effective cardiac function and adequate systemic arterial pressure have been achieved.

Removal of aortic occlusion may result in further hemodynamic sequelae.[69] Besides the abrupt reperfusion of the ischemic distal torso and washout of metabolic products and inflammatory mediators associated with aortic declamping, there are direct effects on the cardiopulmonary system. The return of large volumes of blood from the ischemic extremities, with its lower pH, elevated lactate, and other mediators, may exert a cardiodepressant activity on myocardial contractility.[70] Overzealous volume loading during aortic occlusion may also result in ventricular strain, acute atrial and ventricular dilatation, and consequently, precipitous cardiac failure.[34] Following release of aortic occlusion, there is impaired left ventricular function, systemic oxygen utilization, and coronary perfusion pressure in the postresuscitation period.[27,68] The transient fall in coronary perfusion may not be clinically relevant in patients with efficient coronary autoregulation; however, in patients with coronary disease or underlying myocardial hypertrophy, this increase in cardiac work may result in clinically critical ischemia.[27]

OPTIMIZING OXYGEN TRANSPORT FOLLOWING RT

Following RT, patients are frequently in a tenuous physiologic state. The combination of direct cardiac injury, ischemic myocardial insult, myocardial depressants, and pulmonary hypertension adversely impact postinjury cardiac function (see Chapter 58). Additionally, aortic occlusion induces profound anaerobic metabolism, secondary lactic academia, and release of other reperfusion-induced mediators. Consequently, once vital signs return, the resuscitation priorities shift to optimizing cardiac function and maximizing oxygen delivery to the tissues. The ultimate goal of resuscitation is adequate tissue oxygen delivery and cellular oxygen consumption (see Chapter 57). Circulating blood volume status is maintained

at the optimal level of cardiac filling in order to optimize cardiac contractility, and the oxygen-carrying capacity of the blood is maximized by keeping the hemoglobin above 7 to 10 g/dL. If these measures fail to meet resuscitative goals (eg, resolution of base deficit or clearance of serum lactate), inotropic agents are added to enhance myocardial function.

FUTURE CONSIDERATIONS

Defining Nonsalvageability

As clinicians faced with increasing scrutiny over appropriation of resources, it is critical to identify the patient who has permanent neurologic disability or death. Although there may be a widespread belief that survivors of RT have long-term functional or neurologic impairment, a recent analysis reports the majority have no long-term sequelae.[71] Resuscitative efforts should not be abandoned prematurely in the potentially salvageable patient, but field assessment of salvageability is unreliable.[43,72-76] Our clinical pathway attempts to optimize resource utilization, but outcomes must continue to be evaluated, searching for more definitive predictors of neurologic outcome. For example, markers of brain metabolic activity such as increased serum neuron-specific enolase activity appear to have prognostic significance for irreversible brain damage.[77] Although the optimal outcome of RT is undoubtedly the neurologically intact survivor, some have recognized the impact of RT on the potential salvageability of organs for donation.[78] The use of more advanced monitoring devices in the ED, together with further elucidation of the characteristics of irreversible shock, may permit a more physiologic prediction of outcome for these critically injured patients in the future.

Temporary Physiologic Hibernation

A potential adjunct in the care of traumatic arrest is the timely application of hypothermia. Recent randomized studies suggest the use of hypothermia for central nervous system protection after nontraumatic cardiac arrest.[79,80] In these studies, patients randomized to a period of mild-to-moderate hypothermia (32–34°C) after cardiac arrest had improved neurologic outcomes compared to those kept normothermic. This favorable effect was presumably due to a decrease in cerebral metabolic demand during hypothermia. In addition, hypothermia may reduce oxygen radical generation and inflammatory mediator production. By extension, then, if an injured patient in transport could be cooled to a minimal metabolic rate (ie, suspended animation), one can posit that transfer to definitive care might be possible.[81,82] Application of this principle to the multiply injured or bleeding patient, however, is problematic. Rapid cooling is not currently practical in the field, and there are legitimate concerns about the adverse effects of hypothermia on immune function and effective clot formation. Overall, although some[83] preclinical work supports the application of hypothermia after resuscitation from hemorrhage, other investigators have reached the opposite conclusion.[84]

Temporary Mechanical Cardiac Support

The concept of temporary mechanical cardiac support for the failing heart following injury is intuitively attractive. Unfortunately, experience with the intra-aortic balloon pump in this scenario has been unrewarding. The advent of centrifugal pumps (Bio-Medicus Pump; Bio-Medicus, Inc., Minneapolis, MN), which allow partial cardiac bypass without systemic anticoagulation, offers another potential means for increasing salvage of the moribund patient. Centrifugal pumps have become the standard approach for open repair of a torn descending thoracic aorta (see Chapter 30).[85,86] The adjunctive use of extracorporeal membrane oxygenation may also play a critical role in supporting the patient with massive injuries or early multiple organ failure.[87] Finally, hypothermia circulatory arrest may find utility in a broad spectrum of patients with injuries that are considered "irreparable."[88,89] Although a political issue, restoring perfusion in a patient with unsurvivable injuries is a potential source of organs for donation,[90] particularly with kidneys that can tolerate prolonged periods of warm ischemia.

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18

Surgeon-Performed Ultrasound in Acute Care Surgery

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KEY POINTS

- Medical diagnostic ultrasound imaging most often uses transducer frequencies between 2.5 and 10 MHz (1 megahertz = 1 million cycles per second).
- For the focused assessment with sonography for trauma (FAST) examination, either a sector- or convex-shaped transducer (low frequency: 2.5–5.0 MHz) is used to visualize the pericardial space and relatively deep regions in the abdominal cavity (hepatorenal fossa, splenorenal recess, pelvis).
- A negative FAST examination in the hypotensive patient with blunt trauma, particularly one with a pelvic fracture, should be followed by a second FAST examination in 15 to 30 minutes or a diagnostic peritoneal aspiration.
- The primary goal in the thoracic component of the extended FAST examination is to detect a pneumothorax or hemothorax in the pleural cavity.
- The assessment of intravascular volume status during resuscitation can be estimated by measuring the diameter and collapsibility of the inferior vena cava using ultrasound.
- The ultrasound diagnosis of acute calculous cholecystitis is made by visualizing cholelithiasis, thickening of the wall of the gallbladder, and the presence of pericholecystic fluid.
- The ultrasound diagnosis of acute appendicitis is made by visualizing an enlarged appendix with an appendicolith, lack of compressibility and diameter under compression of greater than 6 mm, and increased blood flow in the appendiceal wall.
- A high-frequency linear transducer is used to confirm the presence of a soft tissue infection or abscess and allow for percutaneous drainage.
- Cardiac goal-directed ultrasound includes the subxiphoid, parasternal long-axis, parasternal short-axis, and apical four-chamber views.
- During ultrasound-guided catheterization of the internal jugular vein, the transverse view is used to identify puncture of the vessel, whereas the longitudinal view is beneficial to localize the needle tip and wire.

INTRODUCTION

Ultrasound is routinely incorporated into the workup of patients with a variety of trauma, critical care, or emergency surgical disorders.[1-6] Despite advances in other imaging modalities such as multidetector-row computed tomography (MDCT), the ability to acquire real-time and repeatable data with ultrasound allows the surgeon to make critical decisions expeditiously and with minimal risk of complications. Surgeons are also capable of obtaining good-quality images and interpreting ultrasound examinations independently.[7-9] Given that medical students, surgical residents, and fellows have frequent opportunities to learn the technique of bedside ultrasound examination in patients with trauma and emergency general surgery problems, broad familiarity is typical.[10] Similarly, the use of ultrasound in critically ill patients for resuscitation, hemodynamic monitoring, and procedural guidance is now commonly performed.[11-18] These critical care ultrasound studies can also be completed at the bedside and, therefore, can avoid transportation of patients out of the intensive care unit (ICU). This chapter reviews the indications, techniques, and currently available data for ultrasound examinations in trauma, emergency general surgery, and critical care patients.

PHYSICS OF ULTRASOUND

Ultrasonography is operator dependent. Therefore, an understanding of select principles of ultrasound physics is necessary so that images may be acquired rapidly and interpreted correctly. Knowledge of these basic principles enables the acute care surgeon to select the appropriate transducer, optimize resolution of the image, and recognize artifacts. Basic terms and principles of physics relative to ultrasound imaging in the acute setting are defined in Tables 18-1, 18-2, and 18-3.

In general, an ultrasound system includes the following components: (1) a transmitter that controls electrical signals sent to the transducer; (2) a receiver or image processor that admits the electrical signal; (3) a transducer containing piezoelectric crystals to interconvert electrical and acoustic energy; (4) a monitor to display the ultrasound image; and (5) an image recorder or printer.[19,20] The ultrasound images themselves depend on the orientation of the transducer or probe relative to the structure or organ being imaged, with each transducer having an indicator that directs its orientation to the screen. The indicators on most probes are oriented such that placing the indicator cephalad in a coronal or sagittal plane or to the left in the transverse plane will orient the image correctly. This left-to-right convention is typically reversed in cardiac imaging presets that are employed in focused echocardiography examinations. As the image may be purposefully reversed by a machine adjustment, the surgeon should confirm the orientation of the probe by adding ultrasound gel to the probe's footprint and gently rubbing it to visualize where the motion is detected on the screen (Fig. 18-1). This will allow the surgeon to determine how the indicator orients the image. The orientations or scanning planes are described in Table 18-4.[21]

Although diagnostic ultrasound uses transducer frequencies ranging from 1 to 30 MHz (1 megahertz = 1 million cycles per second), medical diagnostic imaging most often uses frequencies between 2.5 and 10 MHz (Table 18-5). Accordingly, transducers are chosen on the basis of the depth of the structure or organ to be imaged. High-frequency transducers (≥5 MHz) provide excellent resolution for imaging superficial structures such as a thyroid nodule or an abscess in the soft tissue of an extremity. Lower-frequency transducers emit waves that penetrate deeply into tissue and are preferred for visualizing organs such as the liver or spleen.[19,22,23]

ULTRASOUND IN TRAUMA

Since the 1980s, ultrasound has been used to detect free fluid such as blood in the pericardial and intraperitoneal spaces in acutely injured patients.[24,25] This simple, rapid, and accurate ultrasound technique was adopted in the United States in the 1990s and subsequently named the focused assessment with sonography for trauma (FAST).[26] The application of ultrasound has expanded to other areas of the body to identify injuries and help to direct patient management in the prehospital setting and within the emergency room.

Technique of FAST: General Principles

The FAST examination is performed by a trauma team member (trauma surgeon, emergency medicine physician, trainee, or advanced practice provider) (Fig. 18-2). Although a designated examiner typically performs the FAST examination, the care provider who conducts the primary and secondary survey may also complete the FAST sequentially. The patient's chest and abdomen are exposed during the primary survey. Trauma patients are normally placed in the supine position during the primary and secondary survey of the Advanced Trauma Life Support (ATLS) algorithm.[27] Therefore, dependent portions in the abdominal cavity should be scanned to identify abnormal hypoechoic areas (ie, free fluid). In many institutions, a small-size portable ultrasound machine is attached to the bedside tower in the emergency department (Fig. 18-3). For the FAST examination, either a sector- or convex-shaped transducer (low frequency: 2.5–5.0 MHz) is used to visualize the pericardial space and relatively deep regions in the abdominal cavity. A linear-shaped transducer (high frequency: ≥7.5 MHz) is not suitable for this reason (Fig. 18-4). Before starting the examination, the image settings, including depth or gain, should be adjusted. Recording of real-time images is required for review to ensure quality of the FAST examination. The entire examination should not require more than 5 minutes, even in the hands of trainees. The goal of the FAST examination is to detect or exclude fluid in the pericardial space and abdominal cavity.

Pericardial Examination

The FAST examination typically begins with visualizing the heart so that the image settings, such as gain or depth, can be optimized (with a known fluid-filled structure). It is also the component of the examination that may illicit immediate surgical response (eg, thoracotomy). The intracardiac blood should be anechoic, and the posterior heart should be visualized on the screen. First, a transducer is placed in the subxiphoid region for a sagittal or transverse view of the heart. The transducer should be directed toward the head of the patient under the xiphoid process. In this position, the heart will be visualized beneath the left lobe of the liver. This sagittal view enables the examiner to view simultaneously the inferior vena cava and was prescribed in the original description of the FAST examination. A transverse view allows the provider to visualize more pericardial surface area, although more pressure is applied with consequent pain. A pericardial effusion is observed as an anechoic area around the heart (Fig. 18-5). Both the anterior and posterior walls of the heart should be visualized because a small effusion can otherwise be missed. For patients with severe injuries to the chest and/or abdominal wall, subcutaneous emphysema, a narrow costal angle, or a thick thoracoabdominal wall, appropriate views may not be obtainable through this window. Alternate views include the apical view or parasternal view in which the transducer is placed adjacent to the left nipple or along the sternum in the second intercostal space, respectively.

TABLE 18-1: Ultrasound Physics Terminology Relevant to Ultrasound Imaging

Term	Definition	Significance
Ultrasound	High-frequency (>20 kHz) mechanical radiant energy transmitted through a medium	Diagnostic ultrasound: 1–30 MHz Medical diagnostic ultrasound: 2.5–10 MHz
Frequency	Number of cycles per second (10^6 cycles/s = 1 MHz)	Increasing frequency improves resolution Higher-frequency transducers (eg, 7.5 MHz) provide better resolution of tissues.
Propagation speed	Speed with which wave travels through soft tissue (1540 m/s); propagation speed (determined by density and stiffness of medium) is greater in solids than in liquids and greater in liquids than in gases	To image an organ, the ultrasound wave must be emitted from the transducer, travel through a medium (soft tissue or liquid), strike the organ, and bounce back to the transducer. It is the reflected wave that forms the ultrasound image. Ultrasound waves travel better through solids and liquids (molecules are more compact, less interference) than through gas. Therefore, ultrasound waves do not travel well through air-filled structures (eg, lungs or bowel). These organs are visualized, however, when they are surrounded by fluid that acts as an acoustic window and allows the through transmission of waves. The formation of a good ultrasound image depends on two principles of physics: (1) how well sound waves are transmitted through the tissue (acoustic impedance, which equals the density of the material times the speed of sound through the material) and (2) the amount of sound waves reflected once they hit a target organ. A good ultrasound image, therefore, is formed when sound waves travel well through tissues of higher and similar density, such as the liver and kidney. In the presence of subcutaneous emphysema, a large impedance mismatch exists because of the difference in densities between the air-filled tissue and the soft tissue (liver). As a result, the waves travel poorly through the air-filled tissue, and not enough of them are reflected back to the transducer to form a good image. The air-filled lung is not normally visualized because the air within the lung reflects the sound waves too strongly, and therefore, no image is formed. When a hemothorax or pleural effusion is present, the differences in tissue acoustic impedance between fluid and the lung allow the lung to be visualized.
Acoustic impedance		Acoustic impedance = density of tissue × the speed of sound in tissue (sound velocity). The strength of the returning wave depends on difference in density between two organs imaged. Structures of different acoustic impedance (eg, bile and gallstone) are relatively easy to distinguish from one another. Those of similar acoustic impedance (eg, spleen and kidney) are more difficult to distinguish, although Gerota's fascia has a higher tissue acoustic impedance (more dense) and, therefore, allows the spleen and kidney to be visualized as two distinct organs.
Amplitude	Strength or height of wave	Amplitude and intensity are reduced (attenuated) as waves travel through tissue. The higher the frequency, the more the wave is attenuated. Therefore, higher-frequency transducers cannot visualize deep structures well. Increasing the gain setting on the machine enhances the amplitude of the returning or reflected ultrasound waves. If the gain setting is too high, echo amplification is too strong and the image appears too bright.
Attenuation	Decrease in amplitude and intensity of wave as it travels through a medium; attenuation is affected by absorption, scattering, and reflection	
Absorption	Conversion of sound energy into heat	
Scattering	Redirection of wave as it strikes a rough or small boundary	

(*continued*)

TABLE 18-1: Ultrasound Physics Terminology Relevant to Ultrasound Imaging (*Continued*)

Term	Definition	Significance
Reflection	Return of wave toward transducer	
Artifact	Error in imaging; features on the ultrasound image that do not have precise correspondence to the image being scanned	Examples include shadowing (gallstones), reverberation (comet tail, metallic fragment), and mirror image (diaphragm as strong reflector). In the normal sagittal ultrasound image of the lower thoracic cavity, the supradiaphragmatic area (lung) appears to have a similar echogenicity to the liver because of a mirror-image artifact. This artifact occurs because the diaphragm acts as a strong reflector of the ultrasound waves, sending them back to the transducer and then re-reflecting them as they return to the interface of the diaphragm and the liver. (It should be recalled that it is the *returning* or *reflected* wave that forms the ultrasound image on the screen.) These re-reflected or smaller waves return to the transducer *after* the original reflected waves and, therefore, "create" an image that appears to be deeper than the liver and diaphragm, hence the "mirror image."[76]

kHz. kilohertz; MHz, megahertz.

Abdominal Examination

There are three dependent spaces that are scanned in the abdominal portion of the FAST examination: the hepatorenal recess (Morison's pouch), the splenorenal recess, and the pelvis around the bladder. To obtain a longitudinal view in the hepatorenal recess, the transducer is placed in the right upper quadrant (RUQ) of the abdomen. Although this view is often called the RUQ view, optimal views typically are obtained in the right mid-to-posterior axillary line at the level of the 9th to 12th ribs. The right lobe of the liver and the right kidney should be visualized in the same view (Fig. 18-6). Fanning the transducer to visualize the right kidney from one side to the other is key to preventing a false-negative examination, and free fluid can be identified between the liver and kidney. For patients with a significant amount of intraperitoneal fluid, this fluid may also be visualized above the anterior surface of the liver. The longitudinal view of the splenorenal recess is obtained next in the left upper quadrant (LUQ). The transducer should be oriented more superiorly and posteriorly than in the RUQ (Fig. 18-7). Given that trauma patients often present with a full stomach that can be misread as free fluid, it is imperative to visualize the spleen and the left kidney in the same image (Fig. 18-8). Finally, sagittal and transverse views are obtained in the pelvis. The transducer is placed a few centimeters above the pubic symphysis. The FAST examination is ideally performed before an indwelling urinary catheter is placed. Fluid inside (urine) and outside (blood) of the bladder is visualized as an anechoic area separated by the bladder wall (Fig. 18-9).

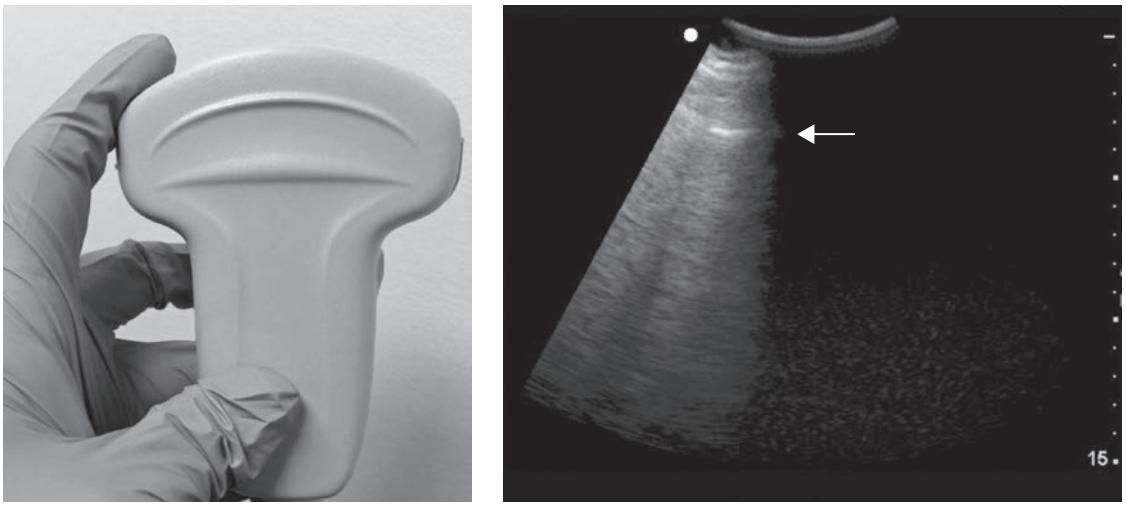
TABLE 18-2: Essential Principles of Ultrasound

Principle	Explanation
Piezoelectric effect	Piezoelectric crystals expand and contract to interconvert electrical and mechanical energy.
Pulse-echo principle	When ultrasound waves contact an organ, some are reflected and some are transmitted through the organ or tissue. Reflected waves return to the transducer and generate electrical impulses that are converted to the image seen on the monitor.

TABLE 18-3: Terminology Used in Interpretation of Ultrasound Images

Term	Definition
Echogenicity	Degree to which tissue echoes the ultrasonic waves (generally reflected in ultrasound image as degree of brightness)
Anechoic	Showing no internal echoes; appearing dark or black
Isoechoic	Having appearance similar to that of surrounding tissue
Hypoechoic	Less echoic or darker than surrounding tissue
Hyperechoic	More echoic or brighter than surrounding tissue

Source: Adapted from Hedrick WR, Hykes L, Starchman DE. Glossary. In: *Ultrasound Physics and Instrumentation*. 3rd ed. St. Louis, MO: Mosby; 1995:355.



A B

FIGURE 18-1 Orientation of the transducer should be confirmed before the examination commences. (**A**) After adding ultrasound gel, a finger is placed on one side of the footprint. (**B**) The motion of the finger (hyperechoic) is detected on the screen (arrow).

Data in Blunt Trauma Using the FAST Examination

The utility of the FAST examination has been examined most extensively in patients with blunt torso injury.[2,7,28-35] Since the 1990s, many prospective and retrospective studies have been conducted in various patient populations. A significant disparity in data regarding the FAST examination is due to different inclusion criteria and outcomes of interest (intraperitoneal fluid vs any intra-abdominal injury).

TABLE 18-4: Scanning Planes Used in Ultrasound Imaging

Scanning plane (Fig. 18-2)	Definition	Transducer orientation
Sagittal	Divides body into right and left sections parallel to long axis	Transducer indicator points toward patient's head
Transverse	Divides body into superior and inferior sections perpendicular to long axis	Transducer indicator points toward patient's right side
Coronal	Divides body into anterior and posterior sections perpendicular to sagittal and parallel to long axis	Transducer indicator points toward patient's head when imaging exteriorly

Source: Adapted from Tempkin BB. Scanning planes and methods. In: Tempkin BB, ed. *Ultrasound Scanning: Principles and Protocols*. Philadelphia, PA: WB Saunders Company; 1993:7.

Accuracy of the FAST examination can be affected by several factors, including hemodynamic stability (a likely surrogate for amount of fluid) or associated injuries.[36,37] Currently, the dominant indication for a FAST examination remains hypotensive patients with blunt torso injuries. A large prospective study conducted by Rozycki et al[38] demonstrated that surgeon-performed FAST was 100% sensitive and 100% specific for detecting hemoperitoneum in hypotensive patients with blunt abdominal trauma. Patients with a positive FAST examination (showing intra-abdominal free fluid) and persistent hypotension should be taken to the operating room for an emergent exploratory laparotomy. In contrast, a negative FAST examination should not be used to rule out a serious intra-abdominal injury in patients with unstable vital signs. Diagnostic options in these cases are to (1) repeat the FAST examination in 15 to 30 minutes or (2) perform a bedside diagnostic peritoneal aspiration to determine the presence of intraperitoneal hemorrhage.[39,40] Furthermore, it remains

TABLE 18-5: Clinical Applications of Selected Transducer Frequencies

Frequency (MHz)	Applications
2.5–3.5	General abdominal FAST Transvaginal
5.0	Pediatric abdominal Testicular Vascular Soft tissue
7.5	Thyroid Pneumothorax

MHz, megahertz; FAST, focused assessment with sonography for trauma.

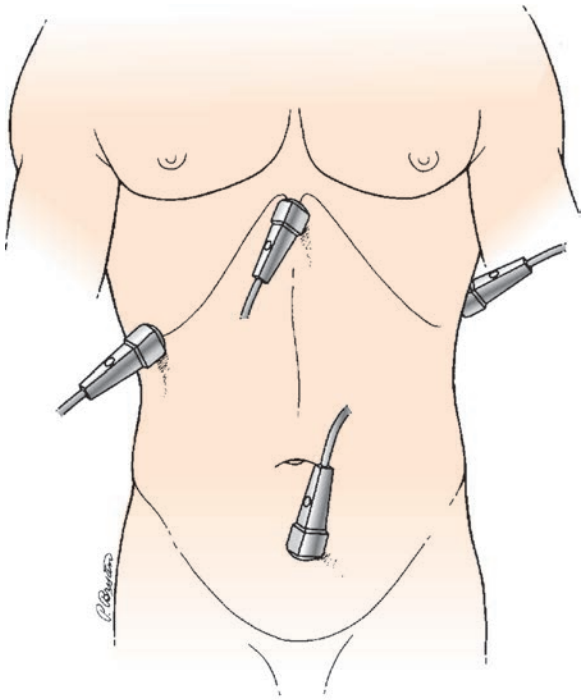


FIGURE 18-2 Transducer positions for the focused assessment with sonography for trauma (FAST) examination: subxiphoid, right upper quadrant, left upper quadrant, and pelvis.

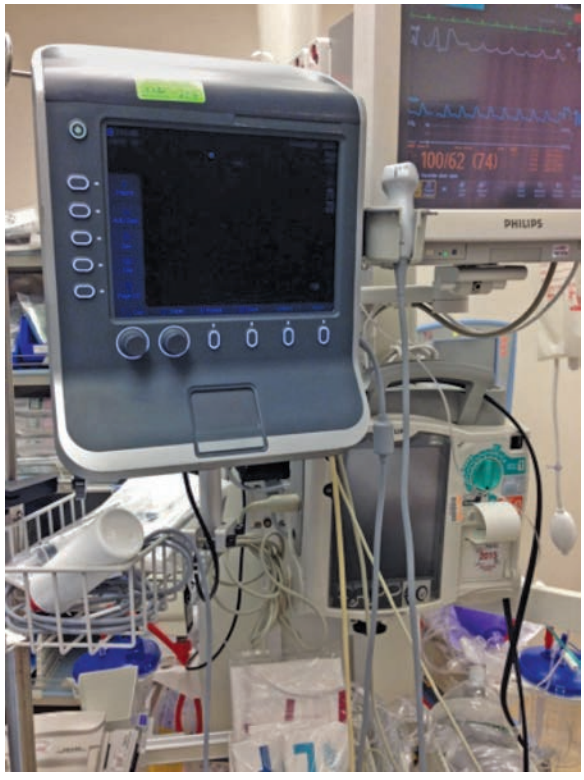
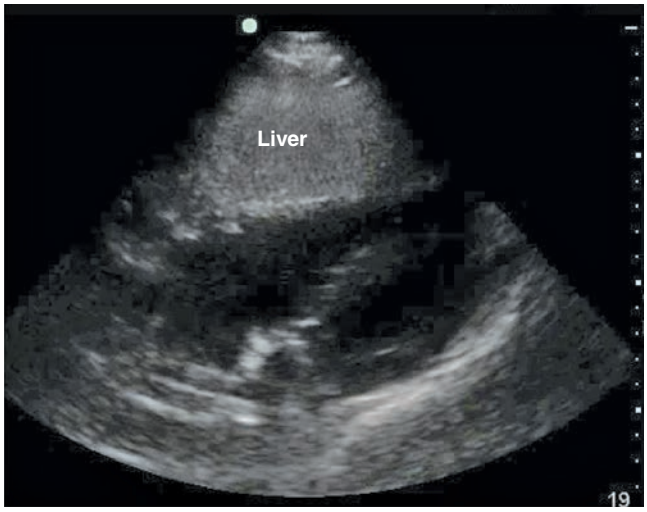
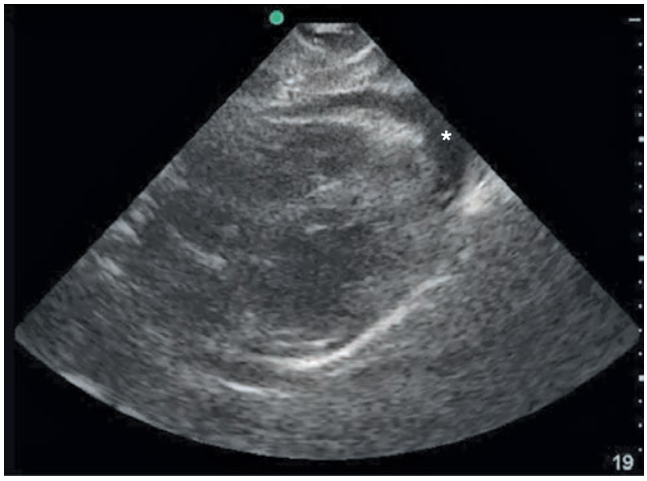


FIGURE 18-3 Ultrasound machine used for the focused assessment with sonography for trauma (FAST) examination.

FIGURE 18-4 Various types of transducers; from left to right: low-frequency convex, sector, and high-frequency linear.

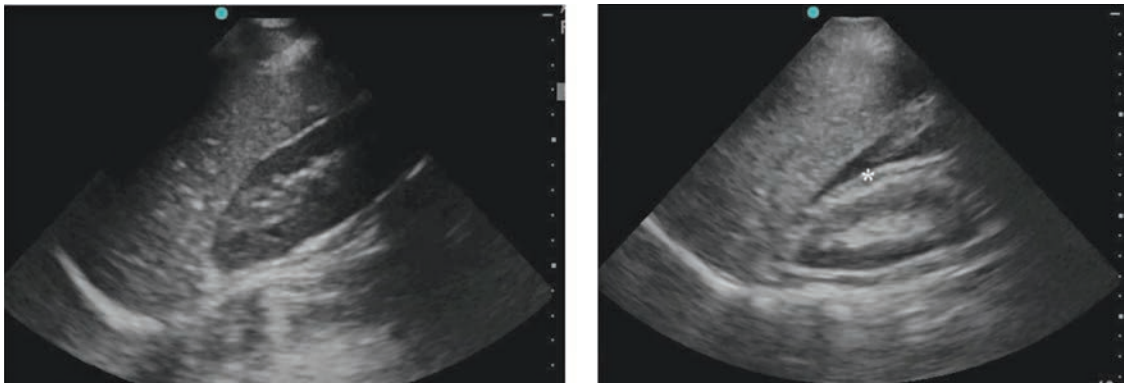


A



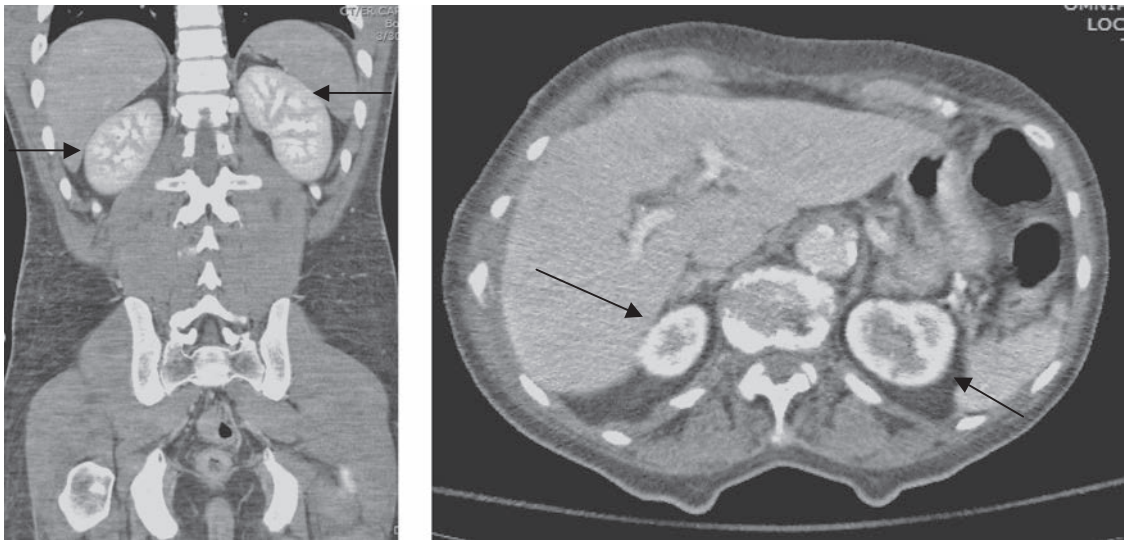
B

FIGURE 18-5 Normal (**A**) and abnormal (**B**) views of pericardial examination. Asterisk indicates pericardial effusion.



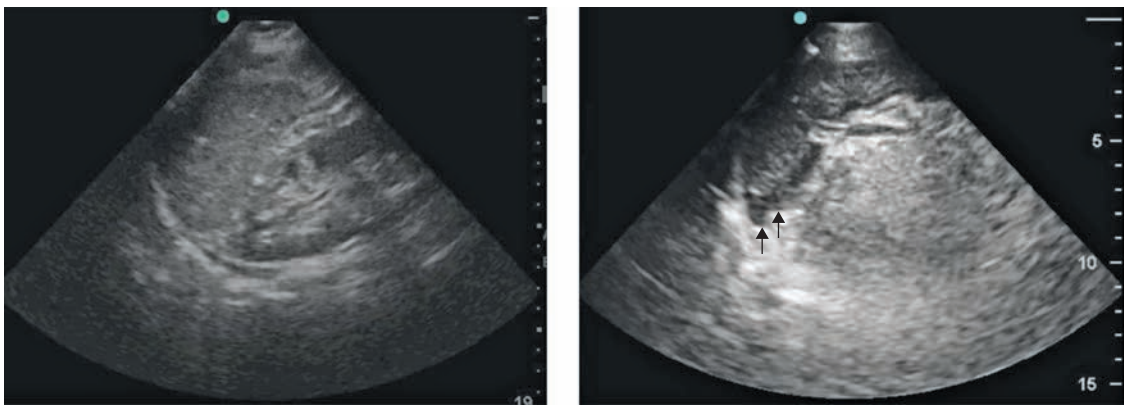
A B

FIGURE 18-6 Normal (**A**) and abnormal (**B**) views of Morison's pouch. Asterisk indicates intraperitoneal free fluid.



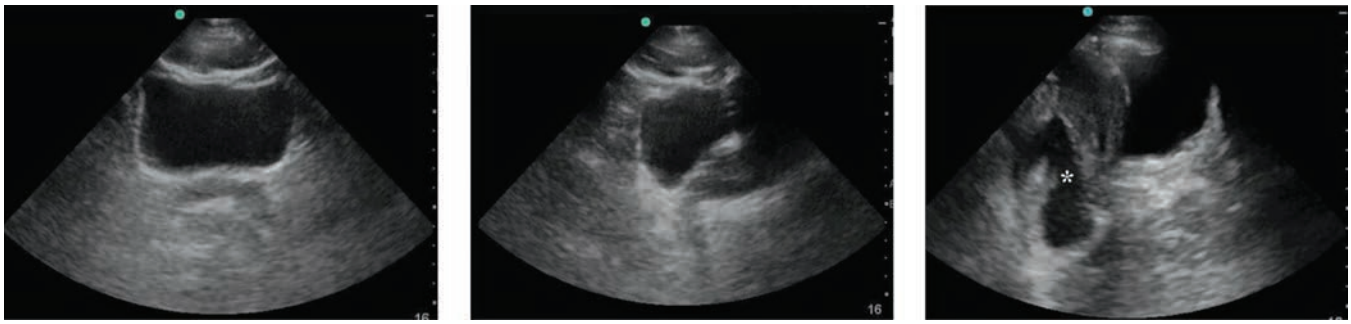
A B

FIGURE 18-7 Coronal (**A**) and axial (**B**) views of the abdominal cavity. Note that the splenorenal recess is located more superiorly and posteriorly (arrows) than the hepatorenal recess.



A B

FIGURE 18-8 Normal (**A**) and abnormal (**B**) views of the splenorenal recess. Free fluid is seen around the spleen (arrows).



A B C

FIGURE 18-9 Normal (**A**: transverse view, **B**: longitudinal view) and abnormal (**C**) views of the pelvic region. Asterisk indicates intraperitoneal free fluid.

unclear how sensitive the FAST examination can be for intraperitoneal fluid in patients with pelvic fractures. More specifically, Friese et al[36] reported that the sensitivity of the FAST examination performed by surgical residents for high-risk patients with a pelvic fracture (age >55 years, systolic blood pressure [SBP] <100 mm Hg, or unstable pattern fracture) was only 26.1% for detecting intraperitoneal fluid. Even for patients with an SBP of less than 100 mm Hg, the FAST examination was not reliable to rule out a hemoperitoneum (sensitivity, 36.4%). Another recent retrospective study, in contrast, concluded that the FAST is sensitive enough (96%) to rule out clinically significant hemoperitoneum associated with pelvic fracture in hemodynamically unstable patients.[41] Although several studies have been reported showing the utility of the FAST examination in the detection of intraparenchymal injuries involving solid organs, the FAST examination carries a low sensitivity in detecting intra-abdominal injuries that are not associated with intra-abdominal fluid.[8,42,43] In the series by Chiu et al,[8] 29% of patients with blunt abdominal injury had no hemoperitoneum. In these patients, the FAST examination was interpreted as negative, although computed tomography (CT) imaging identified grade 2 to 3 splenic and hepatic injuries.

Pericardial fluid secondary to a cardiac injury is rarely observed in patients with blunt trauma. As a result, data reflecting the accuracy of the FAST examination in detecting this fluid are scarce. A series early in the 1990s reported no patients with pericardial fluid after blunt trauma.[38] Press and Miller[44] focused on the cardiac component of the FAST examination in blunt trauma using institutional databases. Out of 29,236 patients with blunt trauma over 7 years, only 14 patients were identified with a hemopericardium and 3 patients with cardiac rupture, respectively. Further, the incidence of hemopericardium confirmed during the emergency cardiac ultrasound was only 0.13%.

Data in Penetrating Trauma Using the FAST Examination

Patients with penetrating injury to the anterior chest can potentially sustain life-threatening injuries. A cardiac injury with an associated hemopericardium should be identified rapidly and accurately regardless of a patient's hemodynamic stability, as early detection of these injuries improves the prognosis. Thus, using ultrasound for patients with penetrating wounds in the so-called "cardiac box" is another critical indication for the FAST examination. A prospective multicenter study by Rozycki et al[45] examining patients with precordial or transthoracic wounds at five Level I trauma centers suggested that a hemopericardium could be identified in all patients without any false-negative examinations (sensitivity 100%). Therefore, patients with a positive pericardial view should have an emergent sternotomy or thoracotomy based on the very high specificity (96.9%). These data were consistent with results from other series of the precordial FAST examination.[1,38] A pitfall in interpreting the pericardial FAST examination for patients with a suspected penetrating cardiac injury, however, is that pericardial blood can decompress into the thoracic cavity if there is an associated laceration of the pericardial sac.[46] In these cases, a patient could present with a hemothorax without evidence of hemopericardium on the FAST examination (Fig. 18-10).

The utility of the FAST examination is limited in patients with penetrating abdominal trauma as compared to blunt injuries. Hemodynamically unstable patients with a penetrating injury require operative intervention regardless of the result of a FAST examination. Similarly, patients with peritonitis should be explored independent of FAST examination results. Therefore, ultrasound examination is not included in the algorithm suggested by the Western Trauma Association to guide the management of patients with anterior abdominal stab wounds.[47] This is supported by multiple studies showing a low sensitivity of the FAST examination for intra-abdominal injuries.[48,49] Although Rozycki et al[7] showed comparable sensitivity and specificity for the FAST examination in patients with penetrating wounds (sensitivity, 83.8%; specificity, 97.4%), subsequent studies consistently failed to replicate this accuracy.[7] In the aforementioned multicenter study, the FAST examination was performed in half of hemodynamically stable and asymptomatic patients with abdominal stab wounds.[47] Of these, only 21% of patients who required therapeutic laparotomy had a positive FAST examination. Similarly, Soffer and Cohn[50] conducted a prospective cohort study to include patients with both stab and

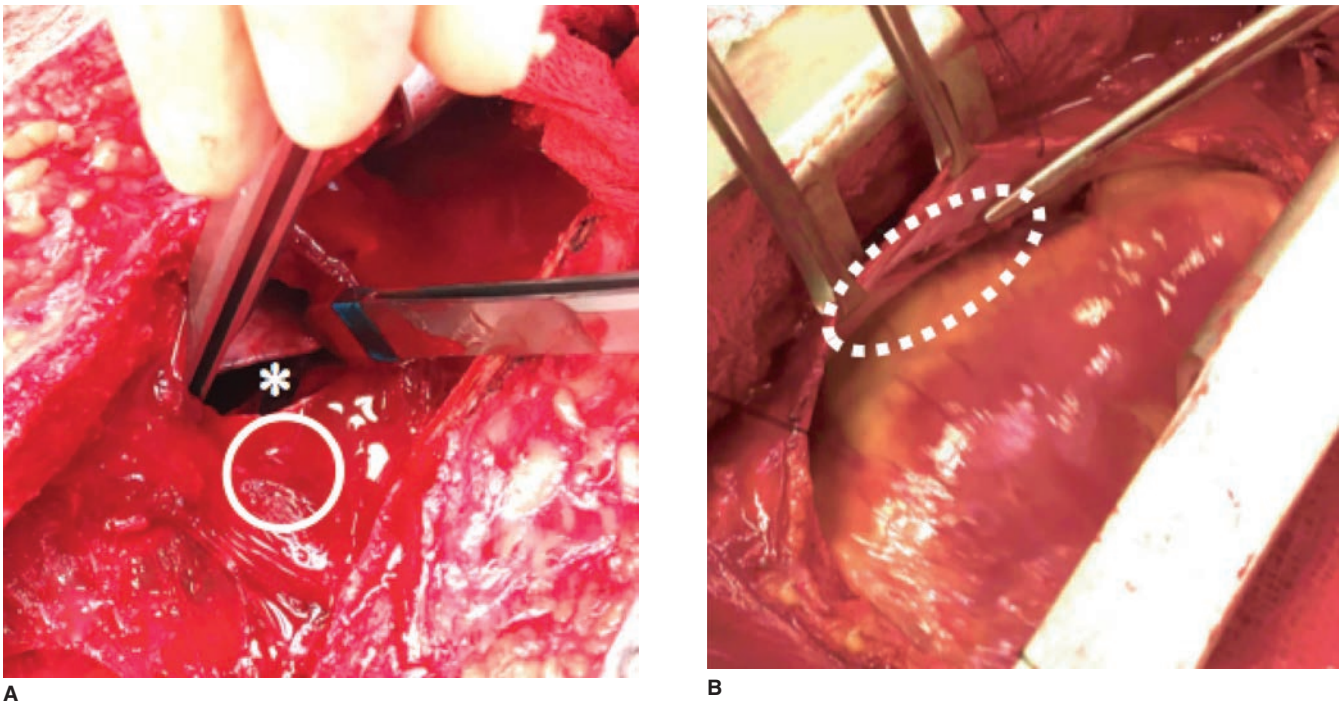


FIGURE 18-10 A case with false-negative pericardial-focused assessment with sonography for trauma (FAST). (**A**) Stab wound was penetrated through the right thorax into the heart (circle). Asterisk indicates lung. (**B**) A hole in the pericardium (dotted area) and injury to the right atrial appendage are identified.

gunshot wounds. A majority of the FAST examinations were performed by trauma surgeons or surgical trainees. Sensitivities of the FAST examination for therapeutic laparotomy in patients with stab and gunshot wounds were 47% and 49%, respectively. Approximately 40% of injuries to a hollow viscus, the most common injury identified at exploratory laparotomy, were missed by the FAST examination. Furthermore, authors have indicated that initial FAST findings rarely alter their management (3 of 177 cases). To date, there are scarce data to determine the role of the FAST examination in hemodynamically unstable patients with penetrating injury. For unstable patients with multicavitary penetrating injury (eg, thoracoabdominal injury), the FAST examination can be a useful tool to guide surgeons in their decision as to which cavity to explore first.[51]

Technique of Extended FAST

Thoracic injuries are common in multisystem blunt or penetrating trauma to the torso.[52] The ATLS protocol emphasizes the importance of identifying life-threatening thoracic injuries, including pneumothorax and hemothorax, during the primary and secondary survey.[27] Frequently, these injuries are missed on a physical examination as patients do not often present with classic signs. Although a chest x-ray (CXR) is considered the gold screening standard for these serious thoracic injuries, equipment or personnel may not be readily available in certain circumstances. Further, because of the patient's position (supine) and other interfering factors such as a backboard, foreign bodies, and/or associated injuries, the quality of the CXR is often inadequate for use as a screening tool (ie, 50% sensitive for detection of a pneumothorax).[53] The application of thoracic ultrasound in trauma patients was first reported as a part of the ultrasound examination in the 1990s.[54] Although inertia was evident, an extended FAST (E-FAST) examination is now typically performed in the trauma bay to detect thoracic injuries.

The E-FAST is normally performed as a continuation of the FAST examination. It is not necessary to change the position of the patient (supine) or the type of transducer (low-frequency sector or convex type). The primary goal in the thoracic component of the E-FAST examination is to detect air (pneumothorax) and/or fluid (hemothorax) in the thoracic cavity. After the pericardial and abdominal examination, the transducer is placed longitudinally in the mid-axillary line, a few costal spaces more cephalad than the area for the RUQ FAST examination. This view should include the right lobe of the liver and diaphragm, which has respiratory excursion. A hemothorax is observed above the diaphragm as an anechoic area (Fig. 18-11). In patients with a moderate to large hemothorax, passively collapsed lung parenchyma is detected as a hypoechoic structure. This maneuver is repeated on the left side to scan the entire thoracic cavity. The transducer should be placed more posteriorly and cephalad to visualize the left hemidiaphragm. The remainder of the E-FAST examination can be performed with either the same low-frequency transducer or a high-frequency (7.5–10 MHz) linear transducer. When a low-frequency transducer is used, the depth of image

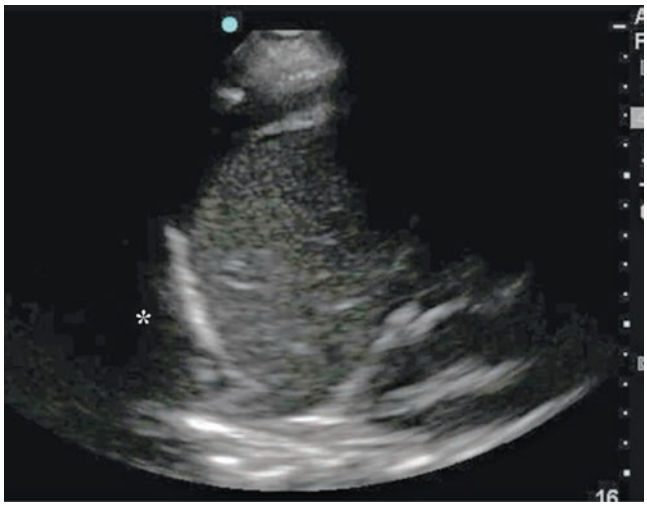


FIGURE 18-11 Hemothorax (asterisk) is seen above the right diaphragm.

requires adjustment to focus on more superficial structures of the anterior thoracic wall. Patients should be maintained in the supine position as the goal of this maneuver is to identify a pneumothorax located anteriorly. The transducer is placed in the midclavicular or parasternal line longitudinally. In order to obtain an optimal view, the transducer should be placed in the intercostal spaces (superior, mid, and lower levels). In normal individuals, a "lung sliding" sign can be observed. This is the finding whereby the parietal and visceral pleurae (both hyperechoic linear structures) are moving in relation to each other. Similarly, "comet tails" are visualized in normal individuals due to artifacts generated by reverberation of the ultrasound waves. This sign is named for vertical, hyperechoic lines arising from the pleural line (Fig. 18-12).These two signs (sliding and comet tails) are *not* observed in patients

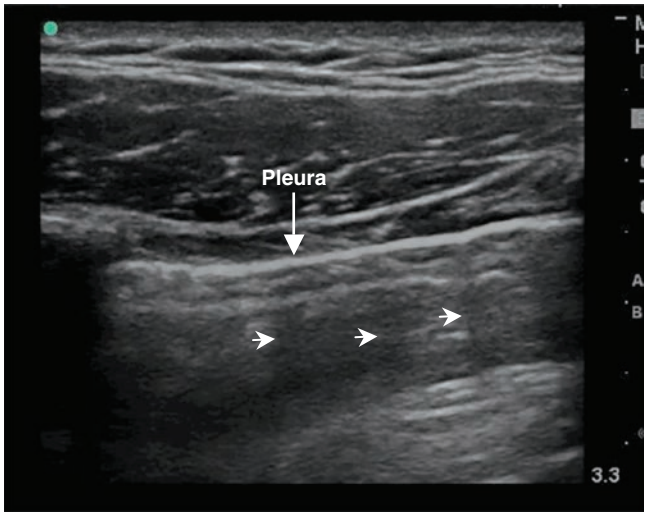
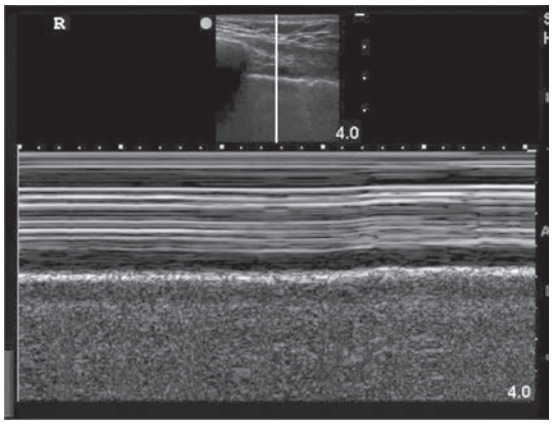
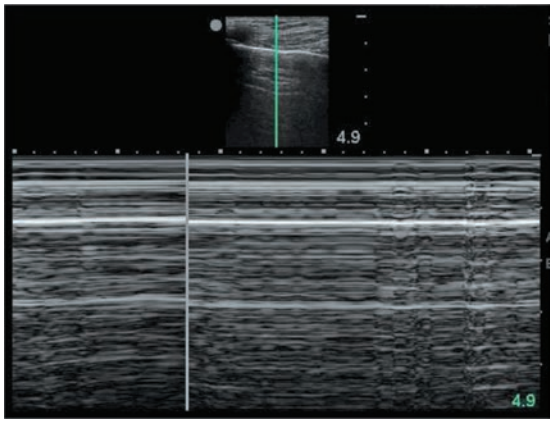


FIGURE 18-12 The "lung sliding" artifact is seen about the pleural line (vertical arrow) in dynamic imaging of the normal lung. Comet tail artifacts (horizontal arrows) can be observed in the normal lung as well.



A



B

FIGURE 18-13 M-mode images show a "seashore" sign in the normal lung (**A**) and "barcode sign" in the lung with a pneumothorax (**B**) (see text).

with a pneumothorax, who have air between the parietal and visceral pleurae. If an ultrasound machine with an M-mode (motion mode) function is employed, the cursor is placed perpendicular to the pleural line. While linear and granular patterns are seen above and below the pleural line, respectively, in normal lung ("seashore sign"), a linear pattern both above and below the pleural line, the so-called "barcode sign," is appreciated in patients with a pneumothorax (Fig. 18-13). Finally, an even more specific ultrasonographic sign for a pneumothorax is called the "lung point." The transition zone between normal lung and the pneumothorax is apparent by the seashore (normal) and barcode signs (abnormal) aligning on M-mode or the sliding lung (normal) adjacent to the nonsliding lung (abnormal) in B-mode imaging (Fig. 18-14).

Data in Trauma Patients Using the E-FAST

Application of thoracic ultrasound in the trauma setting has been described since the 1990s.[54,55] In an early series reported by Ma et al,[54] thoracic ultrasound performed by emergency medicine physicians on 240 patients with both blunt and

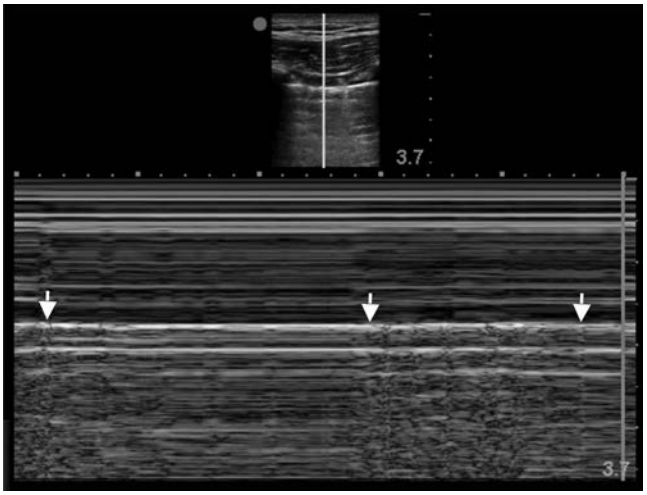


FIGURE 18-14 The lung point is best visualized on M-mode imaging representing the transition between normal lung and a pneumothorax.

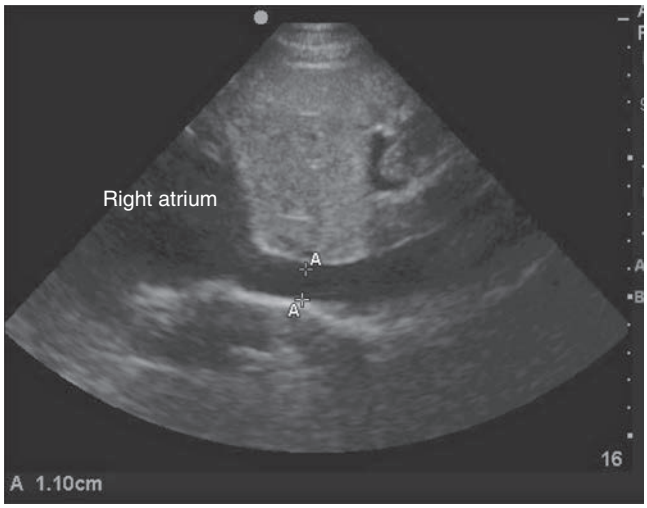


FIGURE 18-15 The inferior vena cava is measured within a few centimeters of entry into the right atrium.

penetrating trauma had a sensitivity of 96.2%, a specificity of 100%, a positive predictive value of 100%, a negative predictive value of 99.5%, and an accuracy of 99.6% to detect a hemothorax confirmed by either tube thoracostomy or CT. Surgeon-performed ultrasound to detect a hemothorax displayed similar results (sensitivity, 97.5%; specificity, 99.7%) to that of the CXR but with a significantly shorter examination time (1.30 ± 0.08 vs 14.18 ± 0.91 minutes, P <.0001).[56] Other early studies that examined the sensitivity and specificity of the ultrasound for a hemothorax also showed promising results comparable to the CXR. The utility of ultrasound to detect a pneumothorax was demonstrated in the 1990s for patients in the ICU.[57] Bedside ultrasound can be helpful to expedite care in patients with unstable vital signs. In trauma patients, Knudtson et al[58] conducted a prospective study to evaluate surgeon-performed ultrasound to detect a pneumothorax. As previously noted, the diagnosis of a pneumothorax was made based on the absence of the lung sliding sign and/or the comet tail artifact. Of note, a low-frequency (2.5–4 MHz) transducer was used in this study. In comparison with the CXR, ultrasound displayed a sensitivity of 92.3%, a specificity of 99.6%, a positive predictive value of 92.3%, a negative predictive value of 99.7%, and an accuracy of 99.3%.

Ultrasound-Guided Resuscitation in the Emergency Department

The resuscitation of severely injured patients should be initiated immediately in the trauma bay. Although damage control resuscitation has been proposed for over a decade, invasive hemodynamic monitoring devices to guide the resuscitation are not always available until the patient is transferred to the ICU. Two components of information that should be obtained using ultrasound are the intravascular volume status (preload) and contractility of the heart. The assessment of intravascular volume status can be estimated by measuring the diameter and collapsibility of the inferior vena cava (IVC) using ultrasound (Fig. 18-15).[59] In one study, noncardiologists successfully visualized the IVC to measure the diameter in 89% of trauma and surgical patients.[60] The assessment of the intravascular volume depletion by a small IVC diameter (<1 cm) with significant collapsibility (>50% with respiration) was comparable to the central venous pressure measured with an intravenous catheter with regard to the examination of cardiac function. Murthi et al[61] compared the cardiac index (CI) obtained by the focused rapid echocardiography evaluation (FREE) to other relevant invasive hemodynamic monitoring methods (pulmonary artery catheter and arterial pressure waveform–based device). Their data suggested that patient management was frequently changed based on the data derived from FREE. Furthermore, there was good agreement between the CIs obtained in FREE and a pulmonary artery catheter. Of interest, the role of point-of-care bedside ultrasound for unstable trauma patients has been evaluated in a randomized control trial.[62] In this study, the limited transthoracic echocardiogram (LTTE) was performed by trained noncardiologists (trauma surgeon, emergency medicine physician, surgical and emergency medicine residents) in the trauma bay. In the LTTE, fluid status, contractility, and pericardial effusion were reported. The use of the LTTE was associated with significantly less volume of intravenous fluid administered, faster time to the operating room, and a higher rate of admission to the ICU. In a subgroup of patients with a traumatic brain injury, a significantly lower mortality rate was also noted in the LTTE group (14.7% vs 39.5%; P = .03).

Ultrasound in Prehospital, Mass Casualty, and Military Settings

With recent advances in technology, ultrasound machines have now become incredibly small and portable. In special environments such as prehospital settings, mass casualty incidents, or military care, a smaller hand-carried ultrasound machine can be used to make rapid diagnoses in injured patients.[63-67]

One of the goals of the ultrasound examination in the out-of-hospital setting is to assist in triaging patients. Although a smaller machine is used, the quality of images should be equivalent to those obtained in the conventional FAST/E-FAST examination within the emergency department. This will facilitate detection of free fluid in the pericardial and intra-abdominal spaces, as well as a hemothorax and/or pneumothorax. In addition, ultrasound has been used for the evaluation of other types of injuries to the eye and extremities.[68,69]

The impact of the prehospital FAST (PFAST) exam on the management of patients with blunt abdominal trauma was evaluated in a multicenter prospective study from Germany.[70] In 95% of patients, the prehospital time was not prolonged because of the PFAST examination (mean time, 2.4 minutes), and good or acceptable quality images could be obtained to establish a diagnosis in 93% of patients. Compared with the FAST examination or CT performed in the emergency department, the PFAST examination displayed a sensitivity of 93%, specificity of 99%, and accuracy of 99%. A mass casualty incident is another unique situation that requires rapid and accurate triage of a large number of trauma victims. The use of ultrasound in a mass casualty incident was initially reported by Sarkisian et al[64] in the early 1990s. Following the Armenian earthquake in 1988, ultrasound examinations identified trauma-related pathology in 51 of 400 patients screened. The average study time was 4 minutes, with no false-positive and four false-negative cases. Zhou et al[67] reported a sensitivity of 91.9%, specificity of 96.9%, and accuracy of 96.6% in detecting any intra-abdominal injury from a more recent experience of surgeon-performed ultrasound in a Chinese earthquake.

There are limited studies that confirm the feasibility of ultrasound for the triage of wounded soldiers and civilians in the combat setting.[71,72] The FAST examination was performed in 281 patients during the 1-month period of the Second Lebanese War.[72] Sensitivity, specificity, and accuracy of the FAST examination to detect intraperitoneal free fluid were 76.5%, 98.4%, and 94.0% in soldiers and 67.6%, 95.2%, and 91.6% in civilians, respectively. As further developments in telemedicine are expected to improve the accuracy of the ultrasound examination in the field, images obtained there can be reviewed by physicians at a support hospital.[73]

The utility of ultrasound in detecting different types of injury has also been studied in weightlessness (eg, the International Space Station [ISS]).[74-76] A preliminary report by Sargsyan et al[74] showed the feasibility of an ISS crew member–performed FAST examination in space. Remote guidance of the examination can be provided by the on-ground ultrasound experts, and images are transmitted via a satellite link for real-time interpretation.

ULTRASOUND IN EMERGENCY GENERAL SURGERY

In addition to a thorough history and physical examination, ultrasound is frequently used in the management of patients with acute abdominal pain. It is particularly helpful when the consulting surgeon personally performs ultrasound imaging to expedite the diagnosis.

TABLE 18-6: Ultrasound Findings of Acute Cholecystitis

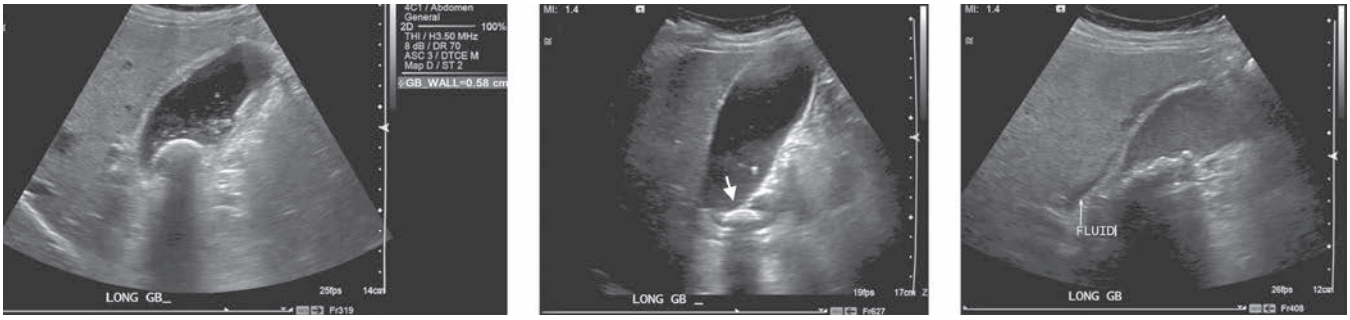
Sonographic Murphy sign (tenderness elicited by compressing the gallbladder with the transducer)
Thickened gallbladder wall (>4 mm)
Noted in patients without chronic liver disease with ascites or right heart failure
Enlarged gallbladder (long axis >8 cm, short axis >4 cm)
Impacted gallstone, debris echo, pericholecystic fluid collection
Sonolucent layer in the gallbladder wall, striated intramural lucencies, and Doppler signals

Acute Biliary Disease

In patients with RUQ abdominal pain, acute biliary disease is always listed within the differential diagnosis. Ultrasound examination of the RUQ area can visualize the majority of biliary structures including the gallbladder and bile duct. Although MDCT is frequently obtained for patients with acute abdominal pain, ultrasound is still considered the imaging modality of choice for patients with suspected acute biliary disorders.[77,78] The diagnostic criteria for acute cholecystitis consist of clinical signs, physical findings, and laboratory results in a guideline based on an international consensus meeting.[79] Ultrasound or other imaging modalities are used to confirm the diagnosis (Table 18-6 and Fig. 18-16). The sensitivity and specificity of ultrasound for acute cholecystitis have been reported previously in multiple studies.[80-82] In 1994, a systematic review by Shea et al[83] documented an 88% sensitivity (95% confidence interval, 0.74–1.00) and 80% specificity (95% confidence interval, 0.62–0.98) in evaluating patients with suspected acute cholecystitis. Nearly two decades later, Kiewiet et al[84] included cholescintigraphy and magnetic resonance imaging to compare the sensitivity and specificity of ultrasound with other imaging modalities. In a head-to-head comparison, the sensitivity of ultrasound (80% vs 94%; P <.001) and the specificity (75% vs 89%; P <.001) were significantly lower than with cholescintigraphy. Nonetheless, because of its availability, low cost, and low risk profile, ultrasound remains the practical diagnostic modality of choice.

Acute Appendicitis

The diagnosis of appendicitis has improved with increased and appropriate use of imaging.[85] Recent data suggest that the negative appendectomy rate has significantly decreased from 23.0% to 1.7%, whereas the preoperative CT rate has increased from 1% to 97.5%.[86] Previous studies comparing ultrasound and MDCT for adult patients with suspected appendicitis showed comparable sensitivities and specificities.[87,88] Limitations of ultrasound examination include the following:



A B C

FIGURE 18-16 Ultrasound findings of acute cholecystitis. (**A**) Gallbladder wall thickness >0.4 cm. (**B**) Enlarged gallbladder and impacted stone (arrow). (**C**) Pericholecystic fluid collection.

(1) operator dependence; (2) decreased accuracy in patients with a thick abdominal wall or overlying bowel gas; and (3) inability to identify other associated pathology. Although MDCT is currently the most commonly obtained study for adult patients with suspected appendicitis, the use of ultrasound is still indicated for pediatric or pregnant patients to avoid radiation exposure or intravenous contrast dye. The sonographic findings to suggest acute appendicitis are listed in Table 18-7 and in Fig. 18-17.[89]

Pyloric Stenosis

Ultrasound is more frequently employed to make a diagnosis of surgical disorders in the pediatric population. Nausea and vomiting in infants can be caused by serious intra-abdominal conditions requiring surgical intervention such as pyloric stenosis. In addition to a history of nonbilious projectile vomiting and a palpable "olive" on a physical examination, ultrasound is considered the dominant imaging test to confirm the diagnosis.[90,91] A target sign is observed in the transverse view, while the length and thickness of the pyloric muscle are measured in a longitudinal view of the upper abdomen (Fig. 18-18). A muscle thickness greater than 3 mm and length greater than 15 mm are considered abnormal, although there are variations in diagnostic criteria.[90] Malcom et al[91] demonstrated the feasibility of the point-of-care ultrasound by nonradiologists to diagnose pyloric stenosis in their case series.

TABLE 18-7: Sonographic Findings of Acute Appendicitis

Enlarged appendix (outer anteroposterior diameter under compression ≥6 mm)
Fluid in the appendiceal lumen
Lack of compressibility of the appendix
Color in the appendiceal wall on color Doppler ultrasound images
Inflammatory changes in perienteric fat in the right lower quadrant
Cecal wall thickening
Right lower quadrant lymph nodes
Peritoneal fluid

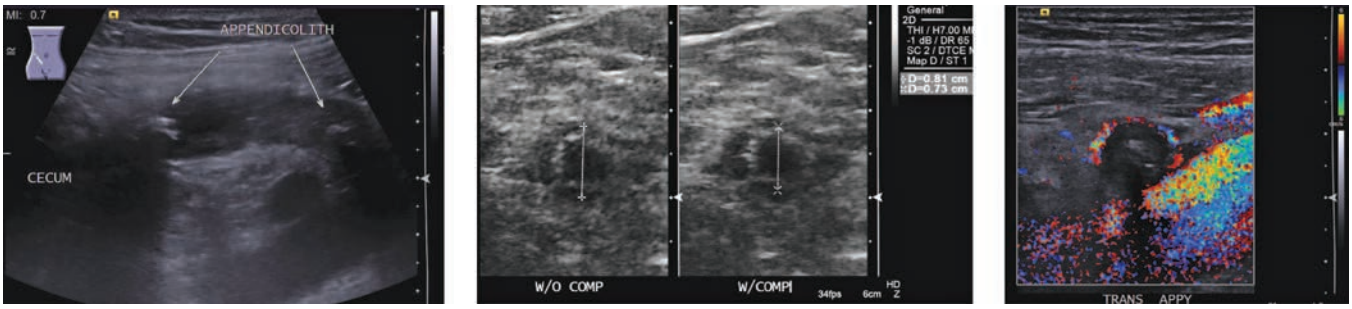
Soft Tissue Infection and Abscess

A superficial soft tissue infection can be easily detected on clinical examination. In contrast, an ultrasound examination can be engaged as an adjunct to visualize a deep space infection, particularly an abscess involving the deep subcutaneous tissue (breast, gluteal area) or muscle (Fig. 18-19). A high-frequency linear transducer is placed over the suspected area. The depth of imaging should be adjusted based on the location (eg, forearm vs thigh), so that a fluid collection (anechoic to hypoechoic area) will not be missed. Furthermore, percutaneous aspiration or incision and drainage can also be guided by ultrasound imaging.[92]

ULTRASOUND IN SURGICAL CRITICAL CARE

As previously noted, the use of ultrasound in the ICU allows surgeons to supplement findings on physical examination to make diagnoses without the need to transport a critically ill patient to another area of the hospital. Furthermore, diagnostic and therapeutic ultrasound examinations are noninvasive, rapid, and readily repeatable. Once a diagnosis has been achieved, ultrasound may allow the surgeon to use invasive techniques under image guidance to provide a lifesaving treatment. Ultrasound guidance and direct visualization during procedures performed by surgeons can lead to decreased complications, shorter hospital length of stay, and decreased cost.[93]

Training programs that teach ultrasound skills in critical care to surgeons should be rigorous and include competence-based testing. Didactic training, hands-on experience, and a partnership with the echocardiography department are essential to develop advanced ultrasound skills. Surgeons should also have a continuing medical education program and formal certification and credentialing process when developing ultrasound skills.[94-97] A certification examination will be administered by the proprietary National Board of Echocardiography beginning in January 2019 and will assess competence in critical care echocardiography and sonography.



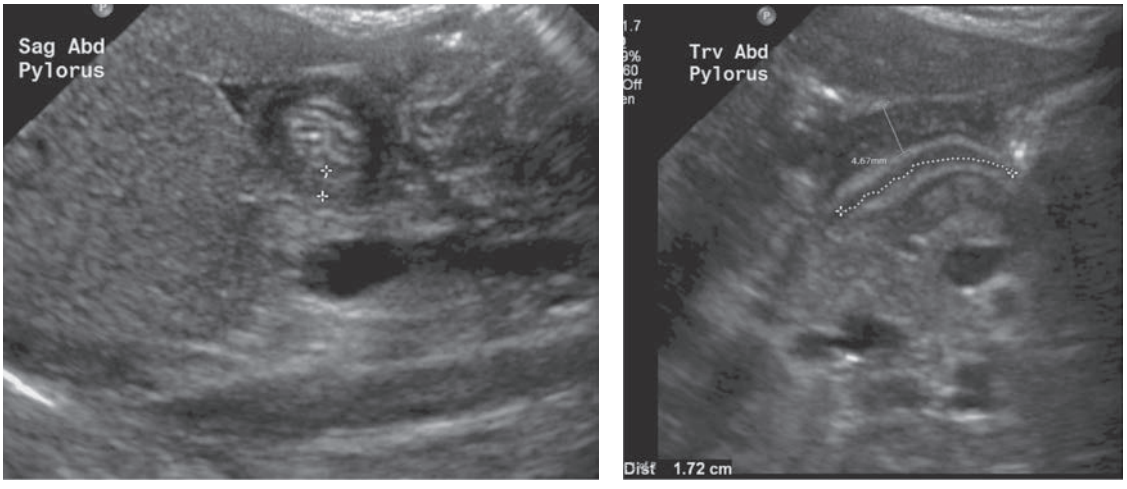
A B C

FIGURE 18-17 Ultrasound findings of acute appendicitis. (**A**) Enlarged appendix with appendicolith. (**B**) Lack of compressibility, diameter under compression ≥6 mm. (**C**) Increased blood flow in the appendiceal wall.

Hemodynamic Monitoring

Focused echocardiography performed by bedside clinicians can be useful for the assessment and management of hemodynamically unstable patients. Training surgeons with previously limited ultrasound skills in focused echocardiography is feasible because this exam is goal directed, problem oriented, limited in scope, and simple compared to traditional echocardiography. In addition, it allows for time-sensitive and repeated assessments. Focused echocardiography can be used to rapidly diagnose causes of hemodynamic instability and/or acute respiratory failure. A surgeon's goal in performing cardiac ultrasound in a hemodynamically unstable patient is to assess global left ventricular (LV) and right ventricular (RV) systolic function, intravascular volume status and preload responsiveness, and the presence of pericardial effusion/tamponade physiology. Additionally, LV diastolic measurements/assessments are possible, unlike with other common monitoring devices (eg, a pulmonary artery flotation catheter). Typically, focused echocardiographic measurements are either qualitative (ie, good vs poor) or semiquantitative based on measuring techniques with substantial extrapolations in computation.

Several variations of the focused echocardiography technique have been described over the years. Their utility has also been examined in critically ill surgical patients.[98] The first, referred to as the focus-assessed transthoracic echocardiography examination, is a two-dimensional and M-mode imaging exam consisting of the most basic cardiac and pleural views. Two derivative examinations have been described in the surgical literature. Gunst et al[59] developed a bedside echocardiography protocol (the bedside echocardiographic assessment in trauma/critical care [BEAT] examination) to be used for the hemodynamic assessment of patients in the surgical ICU. Each letter of BEAT corresponds to a component of the examination (B: beat—CI; E: effusion—pericardial effusion; A: area—subjective cardiac function; T: tank—volume status). In the study, the BEAT examination could be completed rapidly. Of 85 BEAT examinations performed by either surgical trainees or trauma surgeons, the images obtained for the CI calculation were considered of good quality in approximately 60%. In the IVC images for assessment of preload, 97% were



A B

FIGURE 18-18 Ultrasound findings of pyloric stenosis. (**A**) Transverse view showing a target sign. (**B**) Longitudinal view showing thickened muscle >15 mm.

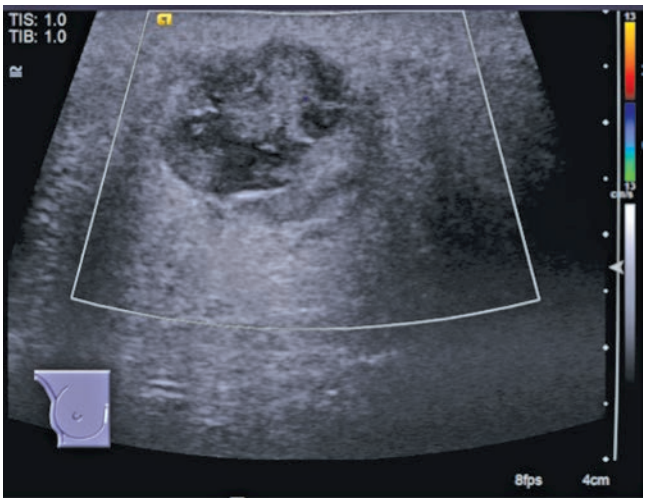


FIGURE 18-19 Abscess cavity is visualized as a hypoechoic lesion (arrow).

of good quality. The CI in the BEAT examination was significantly correlated with that obtained from a pulmonary artery catheter.[16]

Similarly, the focused FREE examination has been used to monitor the hemodynamic status of patients in the surgical ICU. The examiner is able to obtain or calculate information regarding cardiac function, volume status, afterload, and anatomy. Despite the prevalence of thoracoabdominal surgery and mechanical ventilation in their cohort, FREE examiners were successful in completing the study.[61]

TECHNIQUE

Focused echocardiographic examinations are performed using a low-frequency cardiac transducer (small footprint) with the depth set to 14 to 16 cm for adequate visualization of the heart. If available, the cardiac software package and presets should be used as opposed to the abdominal software package that is commonly used in the FAST examination. Cardiac goal-directed ultrasound requires four main views: (1) the subcostal view allows for visualization of IVC size/collapse, RV and LV size, and pericardial effusions; (2) the parasternal long-axis view evaluates LV systolic function and presence of effusions; (3) the parasternal short-axis view allows visualization of LV function at the mid-left ventricle; and (4) the apical four-chamber view can demonstrate a pericardial effusion and RV size and function. In most cases, these four views can be obtained for patients in the supine position. More favorable images can be also be acquired by placing the patient in the left lateral decubitus position as the heart moves more closely to the chest wall. In some patients, only a single view may be possible, but even in these instances, valuable information is available.

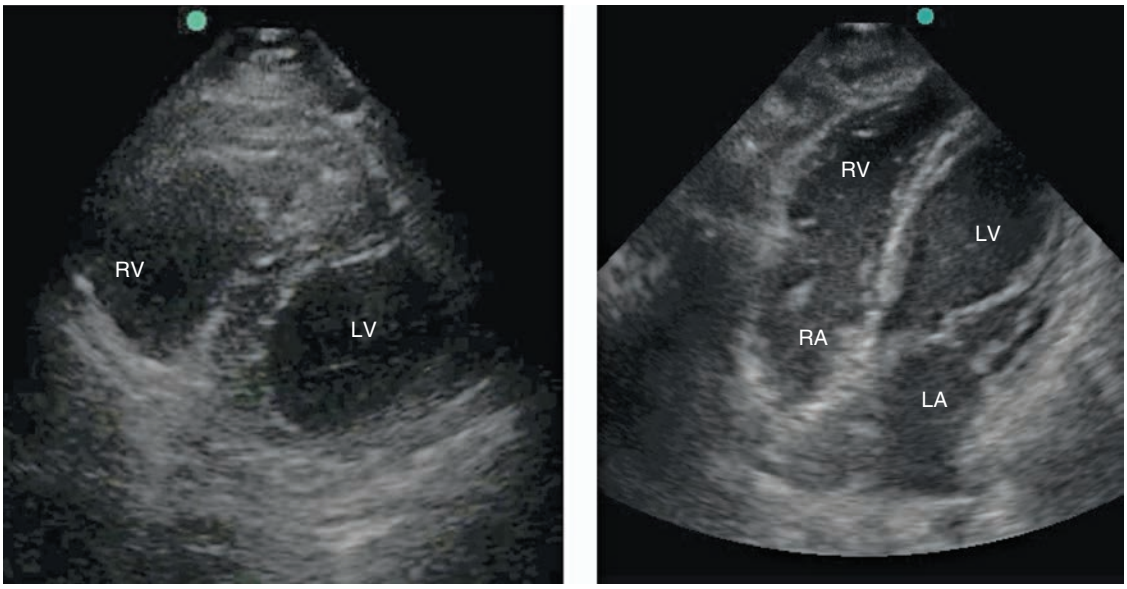
The assessment of volume status and preload responsiveness is performed as a part of the cardiac examination. The latter is perhaps the more relevant parameter and represents whether a patient would benefit from volume administration (by increasing stroke volume and cardiac output). Assessment of volume status via echocardiography is typically performed by obtaining a static image of the structure of interest. Determination of preload responsiveness requires the use of dynamic imaging that entails a before and after assessment (relative to administration of fluid or inspiration or another maneuver) of the structure or parameter of interest. The most commonly used structure to determine volume status and preload responsiveness is the IVC. The diameter of the IVC is measured at the end of both the inspiration and expiration phase in the subcostal view. For better representation of the relevant anatomy, a longitudinal view at the junction of the right atrium and the IVC is visualized first. The measurement is taken within a few centimeters of the cavoatrial junction. The volume status and preload responsiveness are estimated based on the static IVC diameter as well as the collapsibility during the respiratory cycle (Table 18-8). Other echocardiographic techniques of preload responsiveness exist and are beyond the scope of this chapter. Nonetheless, it is important to state that static visualization of the IVC is a crude measure of volume status and is most useful at extreme values, (hypo- or hypervolemia) over time or after therapeutic intervention (ie, fluid resuscitation).

Cardiac function can be determined by both qualitative and quantitative means. Qualitative assessment implies visualizing the beating heart and ascribing its function as normal or abnormal (more specifically, as increased, decreased, or normal contractility). This skill is learned quickly and provides information on the cardiac ejection fraction that is not possible with more traditional methods of hemodynamic assessment, including the use of pulmonary artery catheters. Most often, the parasternal short-axis view is employed to acquire qualitative information regarding contractility, morphology, size, and wall motion abnormality of the LV. The LV and RV are visualized simultaneously in the apical four-chamber views for the assessment of the right-sided cardiac function (Fig. 18-20). Normally, the RV size is two-thirds that of the LV. An enlarged RV can be the result of pressure or volume overload. Quantitative and semiqualitative assessment of left-sided heart function provides actual values for either stroke volume (SV), ejection fraction, or both using relatively simple methodologies.[59,61,98] Semiquantitative methods use computational assumptions. Of three popular

TABLE 18-8: Estimated Central Venous Pressure Related to IVC Imaging

IVC diameter	Collapsibility	Estimated central venous pressure (mm H_2O)
<20 mm	>50%	5
<20 mm	<50%	10
>20 mm	<50%	15
>20 mm	Minimal	20

IVC, inferior vena cava.



A B

FIGURE 18-20 Parasternal short-axis view (**A**) and four-chamber view (**B**) of cardiac ultrasound. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

semi- or fully quantitative methods (fractional shortening, Simpson, and LV outflow tract [LVOT]/velocity time integral [VTI] method) to estimate SV, the fractional shortening method was most significantly correlated with the data obtained from a pulmonary artery catheter in the aforementioned study of patients in the surgical ICU.[99] In the fractional shortening method, a parasternal long-axis view is used for assessment of cardiac function. After an adequate view is obtained in two-dimensional mode, the M-mode cursor is placed to visualize the LV at the level of the distal mitral valve (Fig. 18-21). End-diastolic and end-systolic diameters of the LV are measured in the M-mode image. SV can be calculated by applying these values to a formula stored in the cardiology presets of the ultrasound machine (Table 18-9). Similarly, CI and ejection fraction are calculated. The fully quantitative methodology using a measurement of LVOT and assessment of VTI is thought by others to be the most accurate; however, it requires acquisition of two cardiac views.[100]

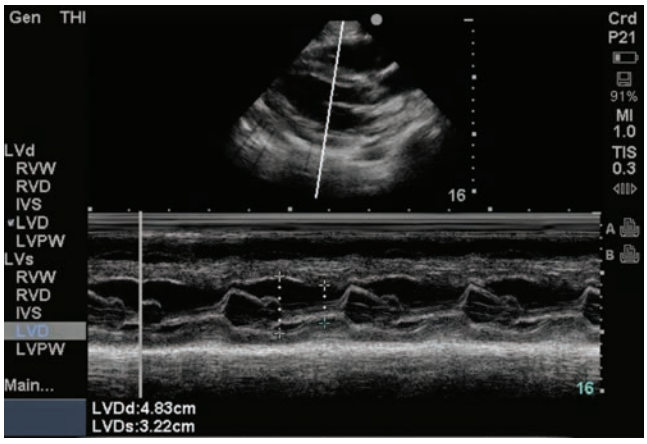


FIGURE 18-21 Parasternal long-axis view is used to calculate the stroke volume in the fractional shortening method.

Pleural Ultrasound

The use of ultrasound for the detection of pleural-based diseases has some advantages over traditional radiographic imaging. Ultrasound eliminates radiation exposure, is easily portable, and provides real-time imaging. Pleural effusions are easily diagnosed and treated by surgeons using bedside ultrasound. As discussed previously in the trauma

TABLE 18-9: Formulas Used in the Fractional Shortening Method

Parameter	Calculation
Left ventricular end-systolic volume	$(7/[2.4 + LVs]) \times LVs^3$
Left ventricular end-diastolic volume	$(7/[2.4 + LVd]) \times LVd^3$
Stroke volume	LVEDV – LVESV
Cardiac output	SV × HR
Cardiac index	CO/BSA
Ejection fraction	SV/LVEDV

BSA, body surface area; CO, cardiac output; HR, heart rate; LVd, left ventricular diastolic diameter; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; LVs, left ventricular systolic diameter; SV, stroke volume.

section, bedside thoracic ultrasound is more sensitive than a plain CXR in detecting the presence of pleural fluid and distinguishing fluid from consolidation of the lung. During bedside thoracentesis to drain a pleural effusion in the ICU, ultrasound guidance increases the likelihood of a successful tap and reduces the risk of a pneumothorax.[93] A pneumothorax may occur in the ICU during mechanical ventilation or after invasive procedures such as a thoracentesis or insertion of a central venous catheter via the internal jugular or subclavian vein. A surgeon can use portable ultrasound to quickly diagnose the pneumothorax in the ICU in the same manner as described in the trauma section.[101,102] In supine and semierect views, an ultrasound is more sensitive than a CXR (91% vs 50%), and both modalities provide similar specificities in the diagnosis of a pneumothorax.[103]

TECHNIQUE

Diagnosis and drainage of pleural effusions can be performed with the patient in the upright position, with the head of the bed elevated to at least a 45° to 60° angle. Patients who cannot tolerate sitting up can be imaged and/or drained in the supine position with the ipsilateral arm positioned across the chest or in lateral decubitus position with the side of the suspected pleural effusion up. A 3.5- to 5.0-MHz convex array transducer is held perpendicular to the skin with the transducer marker pointed cephalad. The transducer is oriented to allow visualization between the ribs to identify pleural fluid. Once the targeted pleural fluid is imaged, a sterile field is prepared and a cover is placed on the ultrasound probe. The pleural space is then entered with a large-gauge needle while aspirating until pleural fluid is obtained. A guidewire is passed through the needle into the pleural cavity using the Seldinger technique. A small skin incision is made around the guidewire, and a pigtail catheter is placed over the wire to allow drainage of the pleural fluid.

Pulmonary Ultrasound

Acute respiratory failure and insufficiency are critical conditions commonly encountered in the ICU. Making a rapid and accurate assessment of the underlying etiology is crucial because patient outcomes can be time dependent. In the past, it was believed that ultrasound was not a useful tool to examine the lung because the air within the normal lung parenchyma prevents transmission of sound waves (Table 18-1). Recently, more data regarding the efficacy of ultrasound in the diagnosis of acute respiratory failure have been published.[104-106]

Lichtenstein and Meziere[106] incorporated several ultrasound findings into a decision tree to identify the cause of acute respiratory failure (bedside lung ultrasound in emergency [BLUE] protocol). Using the algorithm, pulmonary etiologies of acute respiratory failure, including parenchymal (eg, pneumonia, pulmonary embolism) or pleural lesions (eg, pneumothorax), were accurately identified in 90.5% of patients.

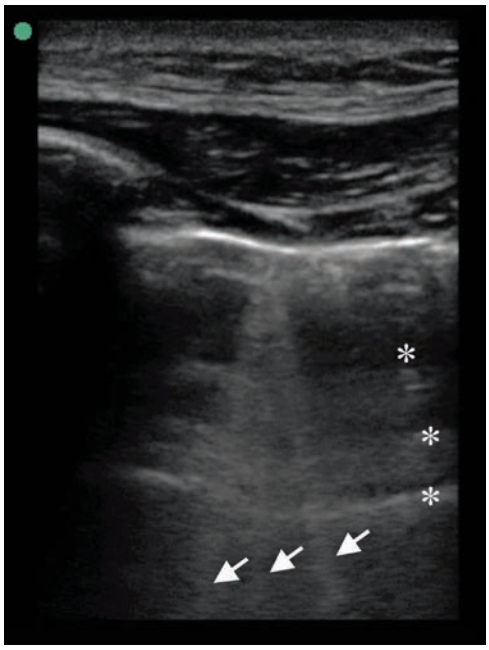


FIGURE 18-22 A lines and B lines on lung ultrasound. A lines (asterisks) are seen as repetitive horizontal lines in normal lung. "Lung rocket" is defined as at least three B lines identified in the intercostal space (arrows).

The BLUE protocol starts with eliciting the aforementioned lung sliding sign (Fig. 18-12), which excludes a pneumothorax. In addition, A lines (repetitive horizontal artifacts) can be identified in normal lung (A profile). A pulmonary embolus should be considered as a cause of acute respiratory failure if venous thrombosis is associated with an A profile. The B line is another artifact characterized as a hyperechoic comet tail arising from the pleural line. Lung rockets are defined as at least three B lines in the intercostal space (Fig. 18-22). The combination of lung sliding and lung rockets (B profile) favors conditions associated with pulmonary edema. Pneumonia can be diagnosed in several patterns of ultrasound findings, including the following: (1) A profile without associated venous thrombosis or positive posterolateral alveolar and pleural syndrome (PLAPS); (2) anterior lung consolidation (C profile); and (3) lung rockets without lung sliding (B profile). An A profile without associated venous thrombosis or PLAPS suggests other pulmonary disorders including chronic obstructive pulmonary disease (COPD) or asthma. The utility of a lung ultrasound protocol has been prospectively evaluated in 189 patients on mechanical ventilation.[105] Sensitivities and specificities of the ultrasound examination for each pulmonary disease were as follows: cardiogenic pulmonary edema, 97% and 95%; COPD or asthma, 89% and 97%; pulmonary embolism, 81% and 99%; pneumothorax, 88% and 100%; and pneumonia, 89% and 94%. Of 253 examinations, patient management was significantly changed in 119 patients (47%). Of note, a majority of these patients (81 of 119) required invasive interventions based on the diagnoses made by the ultrasound of the lung.

Diaphragm Ultrasound

A high incidence of diaphragmatic dysfunction has been reported in critically ill patients, particularly in those receiving prolonged mechanical ventilation (ventilation-induced diaphragmatic dysfunction).[107] Diaphragm dysfunction following mechanical ventilation is significantly associated with extubation failure.[108] Therefore, a bedside ultrasound assessment of diaphragm function can be a useful tool in determining whether the patient can be successfully extubated. Two predictors commonly measured in diaphragmatic ultrasound are the diaphragm excursion and the diaphragm thickening fraction. The diaphragm excursion is the distance that the diaphragm moves during the respiratory cycle. The diaphragm thickening fraction is calculated using a difference in thickness of the diaphragm between end-inspiration and end-expiration. Kim et al[109] defined ultrasonographic diaphragm dysfunction as an excursion of the diaphragm during a spontaneous breathing trial less than 10 mm.[109] In their study, patients with diaphragm dysfunction had a significantly longer total ventilation time, and the rate of weaning failure within 48 hours was significantly higher compared to patients with no diaphragm dysfunction (83% vs 59%, P <.01). The utility of the diaphragm thickening fraction was evaluated in a prospective study by DiNino et al.[110] In patients who were weaned with a spontaneous breathing trial, the positive and negative predictive values of diaphragm thickening fraction of 30% or greater for extubation success were 100% and 67%, respectively, whereas they were 91% and 25%, respectively, with a cutoff of 105 min/L for rapid shallow breathing trial.

TECHNIQUE

The diaphragmatic ultrasound is normally performed while the patient is participating in a spontaneous breathing trial. A low-frequently transducer (3.5–5 MHz) is longitudinally placed in the right midclavicular line below the costal margin or in the right anterior axillary line between the lower ribs to visualize the posterior diaphragm and liver (Fig. 18-23).

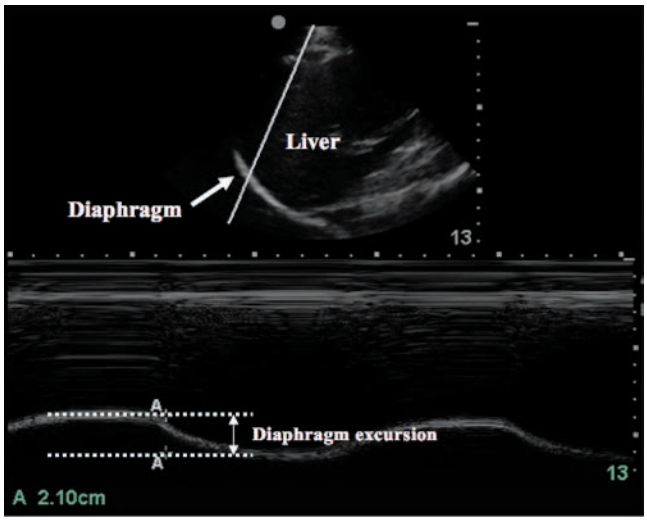


FIGURE 18-23 Measurement of diaphragm excursion.

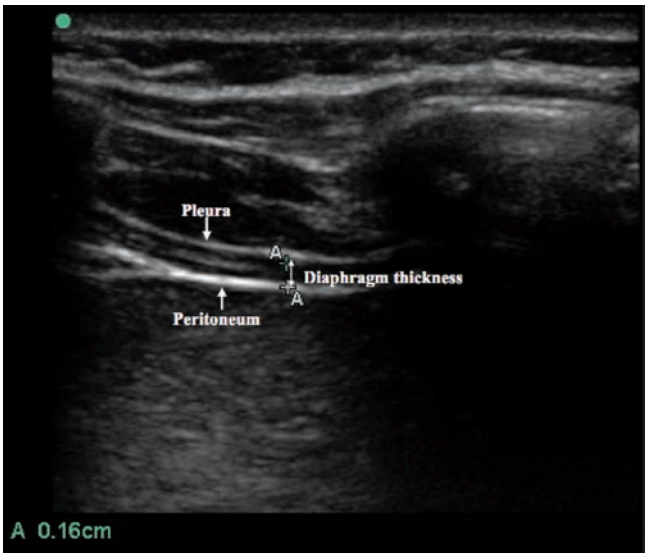


FIGURE 18-24 Measurement of diaphragm thickness.

Then, in the M-mode view, an interrogation line is placed to be perpendicular to the posterior third of the diaphragm. The diaphragm excursion is measured by calculating the depth (centimeters) between the beginning and end of an inspiratory phase. To measure the diaphragm thickening fraction, a high-frequency linear transducer (≥7–10 MHz) is positioned in the midaxillary line between the 8th and 10th intercostal spaces (zone of apposition of the diaphragm to the rib cage). The diaphragm is visualized using the B-mode between the pleura and peritoneum (Fig. 18-24). The M-mode is used to measure diaphragm thickness (millimeters) at end-expiration and end-inspiration. The diaphragm thickening fracture is calculated using the following formula: thickness at end-inspiration – thickness at end-expiration/thickness at end-expiration. Newer measures of diaphragm area (vs. thickness and excursion) have been demonstrated to be more predictive of extubation success in small trials; however, they are beyond the scope of this chapter.

Insertion of a Central Venous Catheter

Insertion of a central venous catheter is performed frequently in critically ill patients. Central venous catheters are required for hemodynamic monitoring, volume resuscitation, administration of blood products, parenteral nutrition and medications, acquisition of lab specimens, and provision of renal replacement therapy.[111] Insertion can be associated with complications such as injury to surrounding vessels causing a hematoma and to the lung resulting in a hemothorax and/or pneumothorax. Ultrasound-guided insertion of a central venous catheter has been clearly shown to decrease the number of attempts at venous cannulation, time required to successful insertion, and the overall complication rate in randomized control studies.[112-114] A meta-analysis including 18 studies concluded that real-time ultrasound-guided cannulation of the internal jugular vein in adult patients was

associated with a lower risk of overall cannulation failure (relative risk [RR], 0.14; 95% confidence interval, 0.06–0.33), fewer complications (RR, 0.43; 95% confidence interval, 0.22–0.87), reduced first attempt failure (RR, 0.59; 95% confidence interval, 0.39–0.88), and reduced first attempt failure rate (1.5 fewer attempts; 95% confidence interval, 0.39–0.88) compared to the landmark technique.[115] Currently, the insertion of a central venous catheter into the internal jugular vein using ultrasound guidance has become considered as best practice and is strongly advised.[114] In contrast, until recently, there were scarce data to support the use of ultrasound to guide cannulation of the subclavian vein.[115] Fragou et al[116] conducted a prospective randomized study that showed significantly improved outcomes associated with real-time ultrasound-guided cannulation compared to the landmark technique. The success rate in the ultrasound group was 100% with a significantly shorter average access time (26.8 vs 44.8 minutes, $P < .05$) and lower complication rates. Because the subclavian vein is located posterior to the clavicle, the penetration of the ultrasound beam is more challenging. In this study, physicians rated the ultrasound-guided subclavian cannulation as a complex task (8 ± 0.2/10). Furthermore, ultrasound-guided cannulation of the subclavian vein is more accurately referred to as an axillary approach.

TECHNIQUE

The internal jugular vein in the cervical and upper thoracic region is imaged with a high-frequency linear transducer. Prior to preparation of the insertion site, the targeted vein should be visualized with ultrasound. The transducer probe is placed transversely on the neck to obtain a cross-sectional image of the internal jugular vein and common carotid artery. The probe is then rotated 90° to obtain a longitudinal image. Patency of the vein should also be assessed by its ability to be easily compressed with the ultrasound transducer. During skin preparation and sterile draping of the insertion site, the ultrasound probe is covered with a telescopically folded sterile sheath. The ultrasound transducer should be held in the nondominant hand perpendicular to the skin. The cannulating needle is positioned in the dominant hand and directed at the target vessel in real time. The tip of needle should be visualized in the image while the needle is being advanced. Once the vein is cannulated by the needle, the remainder of the procedure is completed using the Seldinger technique. The wire should be identified inside the jugular vein before dilating the tract (Fig. 18-25). Typically, the transverse view is used to identify puncture of the vessel, whereas the longitudinal view is beneficial to localize the needle tip and wire.

As the subclavian vein is close to bony structures, it is often challenging to visualize the anatomic relationship to the subclavian artery and pleural line. The axillary vein is the distal continuation of the subclavian vein, and its cannulation under ultrasound guidance can be more easily performed. Similar to imaging of the internal jugular vein, transverse and longitudinal images of the subclavian vein and artery are obtained before draping the area. The subclavian artery is normally located cephalad to the vein. Another key point in cannulation of the subclavian vein is to appreciate the proximity of the pleural line, which can be within a few centimeters below the vein (Fig. 18-26).

Paracentesis

Abdominal paracentesis is a procedure that surgeons use in the ICU for diagnosis and/or therapy. Diagnostic paracentesis consists of obtaining a small amount of peritoneal fluid for culture to rule out infection and to characterize the fluid as a transudate or exudate (or occasionally for lipase/amylase levels to rule out pancreatic ascites). Therapeutic paracentesis is a technique that removes a large volume of ascites (typically in excess of 2 L) to reduce intra-abdominal pressure and treat the resulting abdominal discomfort and dyspnea. Therapeutic paracentesis is a procedure that is commonly performed by surgeons caring for patients with hepatic failure in the ICU setting who are at a high risk of postprocedure bleeding. Ultrasound guidance for paracentesis in patients with ascites results in greater success in acquiring fluid compared to traditional techniques (95% vs 61%).[117] Patients undergoing ultrasound-guided paracentesis also have a decreased risk of bleeding (0.27% vs 1.25% without ultrasound), leading to decreased hospital length of stay and costs.[93]

TECHNIQUE

Paracentesis is typically performed with the patient supine with the needle inserted into the left lower quadrant. The general area of the puncture site is localized 2 to 3 cm medial and 2 to 3 cm cephalad to the anterior iliac spine in the left lower quadrant. A convex ultrasound of 3.5 to 5.0 MHz is then used to locate peritoneal fluid and to visualize the inferior epigastric artery to avoid injury and hemorrhage. Skin preparation and sterile draping of the area and use of the ultrasound probe are performed, followed by insertion of the paracentesis needle. The paracentesis catheter is left in place to achieve removal of the desired diagnostic sample or to reduce intra-abdominal pressure.

SUMMARY

It has been approximately 25 years since surgeons began using ultrasound in the management of trauma patients. Currently, the FAST examination is most useful for patients with blunt trauma who have unstable vital signs. In patients with penetrating trauma to the chest, the pericardial component of the FAST examination is mandatory to rapidly diagnose a cardiac injury.

In recent years, thoracic ultrasound has been more frequently engaged to detect pneumothoraces, hemothoraces/pleural effusions, and lung lesions both in the trauma bay and in the ICU. In the emergency department, focused ultrasound examination is more frequently performed to evaluate patients with suspected intra-abdominal inflammatory processes.

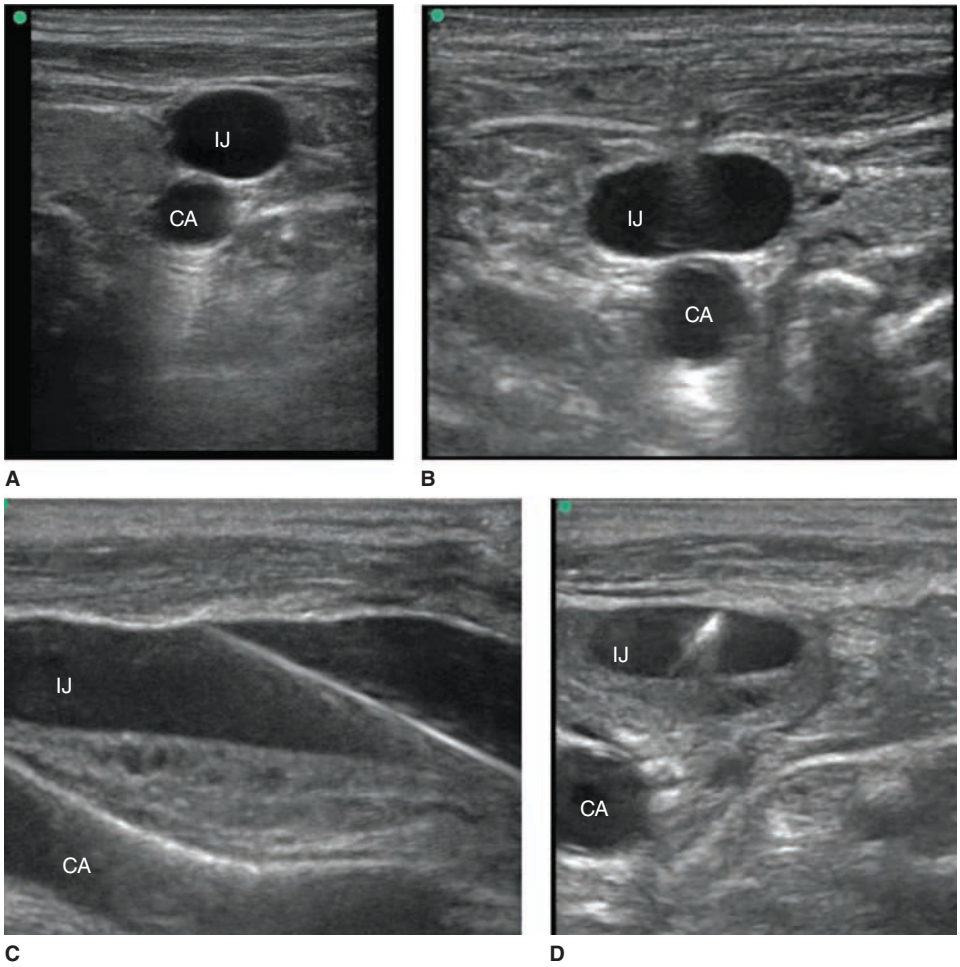


FIGURE 18-25 Ultrasound-guided central venous catheter placement in the internal jugular (IJ) vein. (**A**) Transverse view of right-side neck. (**B**) Tip of needle is visualized on the anterior wall of the IJ. (**C, D**) Guidewire is seen inside the IJ. CA, carotid artery.

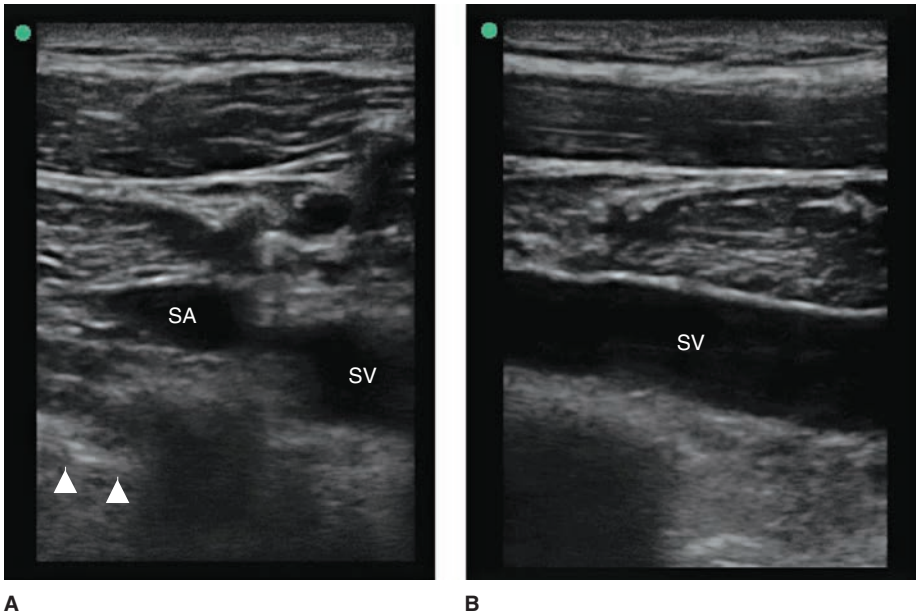


FIGURE 18-26 Transverse (**A**) and longitudinal views (**B**) of subclavian vein (SV). Note that the subclavian artery (SA) is located cephalad to the SV. The pleural line is visualized inferiorly (arrowheads).

Similarly, more advanced ultrasound techniques, including echocardiography, are now guiding the resuscitation of critically ill patients. Of note, these surgeon-performed ultrasound examinations are currently considered the standard care in many clinical settings and hospital systems.

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19

Diagnostic and Interventional Radiology

Scott D. Steenburg

KEY POINTS

- Imaging is a critical adjunct to the comprehensive evaluation of the trauma patient.
- Imaging protocols specific to blunt and penetrating trauma victims should be clearly defined and operationalized to enhance rapid imaging and patient throughput.
- Modern computed tomography (CT) technology and imaging protocols allow for high-resolution imaging of any body part.
- Advanced imaging exams, such as extremity and neck CT angiography, can now be integrated as a part of the initial imaging evaluation of the trauma patient.
- Multiphase CT can provide important information regarding the presence and character of vascular lesions, including active bleeding and solid organ pseudoaneurysms.

INTRODUCTION

Obtaining reliable clinical history and a physical examination may be challenging in the acute trauma patient. Thus imaging can provide timely and helpful information about these patients and identify injuries that otherwise may not be readily recognized. Imaging can be used to assist in injury prioritization and patient triage, as well as guiding the trauma surgeon toward any number of management choices. Despite the advanced imaging technology available in a modern trauma center, it is important to recognize that imaging alone cannot be used to make management decisions in isolation; that is, the surgeon must treat the patient based on an experienced integration of their clinical assessment and diagnostic images.

Practical variables that affect imaging strategies include the proximity of available imaging technology to the resuscitation area, the technical capabilities of the imaging equipment, the experience and availability of radiology technologists performing these imaging procedures, and timely access to expert interpretation and reporting.

Imaging is typically initiated in the trauma bay and integrated as a part of the clinical survey. However, it should reflect the needs of each individual patient. Hemodynamically unstable patients should be resuscitated prior to imaging according to accepted guidelines and recommendations, with some exceptions for image-guided endovascular hemostasis techniques in select scenarios (eg, resuscitative endovascular balloon occlusion of the aorta [REBOA]). To enhance efficiency, imaging should be obtained based on the acute needs for accurate information that can be used to direct treatment of the patient. Close cooperation and open communication between all major stakeholders, including emergency medicine physicians, traumatologists, consultants, nurses, imaging technologists, and radiologists, are essential to optimize any imaging assessment and provide superior patient care.

A single chapter alone cannot reasonably teach interpretation of diagnostic images. Instead, a general overview of trauma imaging strategies is provided. In addition, reflecting current trends in the modern trauma center, an emphasis on advanced computed tomography (CT) technology and capabilities, focusing on select high-yield and common clinical scenarios, is presented.

INITIAL IMAGING FOR THE ASSESSMENT OF BLUNT TRAUMA

Trauma Series

Imaging of the trauma patient is integrated as a part of the secondary clinical survey in the resuscitation suite. The goal of the initial imaging studies is to identify life-threatening, but clinically occult, injuries that require emergent intervention prior to any further imaging, such as an unstable pelvic fracture, hemopneumothorax, or malpositioned/misplaced

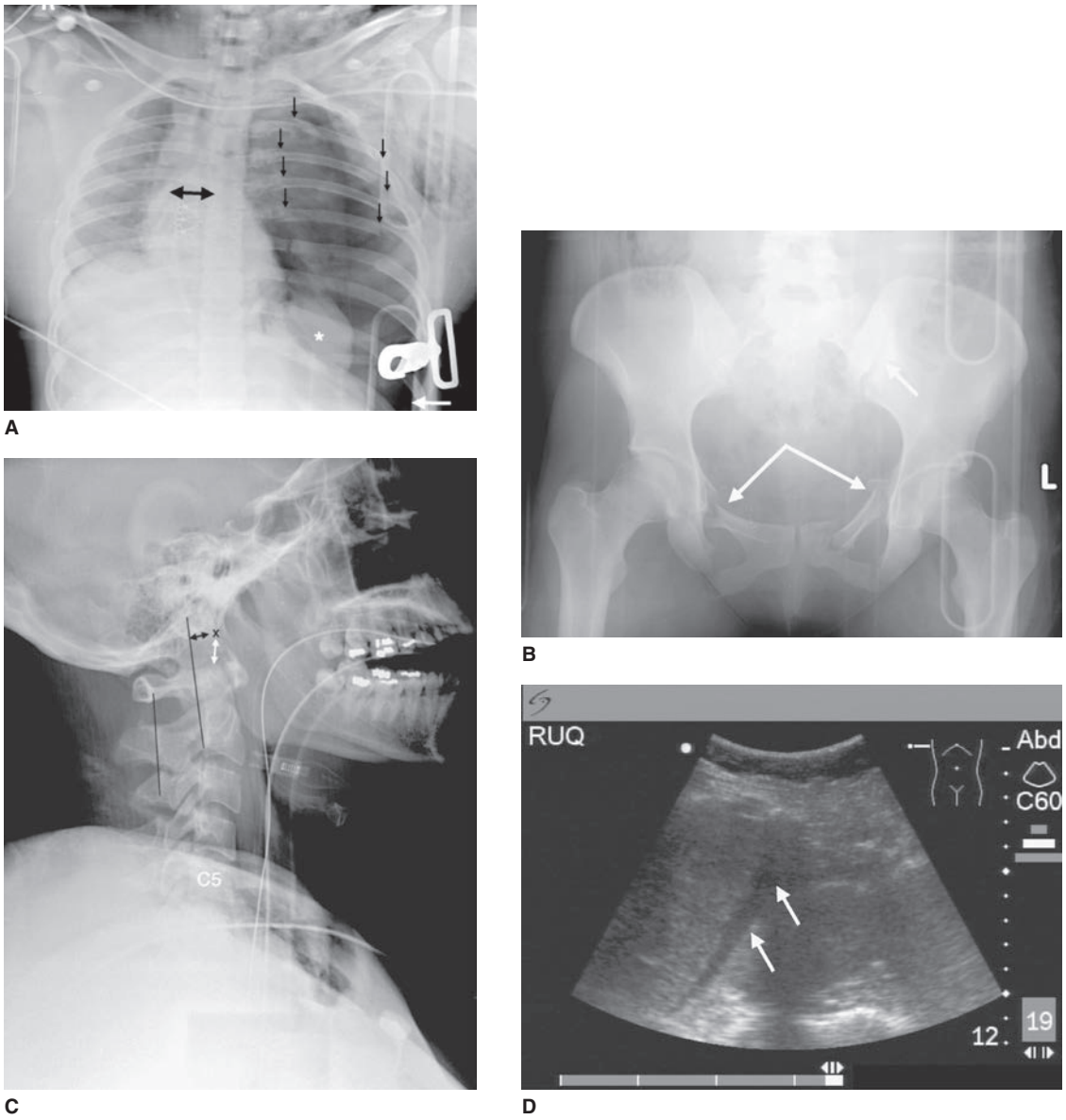


FIGURE 19-1 Trauma series. This 27-year-old unrestrained left rear seat passenger sustained multiple injuries in a high-speed side-impact crash. (**A**) Anteroposterior (AP) recumbent chest radiograph shows hyperexpanded and hyperlucent left hemithorax with deep sulcus sign (short white arrow) and rightward mediastinal shift (double-ended arrow) due to left tension pneumothorax. Short black arrows show multiple displaced rib fractures. Asterisk shows irregularity of left hemidiaphragm, which strongly suggests herniation of abdominal contents through left diaphragmatic laceration. (**B**) AP pelvis radiograph shows lateral-compression–type pelvic ring disruption consisting of bilateral iliopubic and ischiopubic ramus and left sacral fractures (long arrows) with sacroiliac joint disruptions (short arrow). (**C**) Cross-table lateral cervical spine radiograph is grossly normal to C5. Therefore, this constitutes a nondiagnostic study. Craniocervical alignment should be assessed and may be easily overlooked. Dens–basion distance (white double-ended arrow) is normally no greater than 12 mm. Posterior axial line represents cephalad extension of posterior cortex of C2 body (and is normally no more than 12 mm posterior or 4 mm anterior to basion) (black double-ended arrow). Anterior atlantodens interval is normally no greater than 3 mm in adults and 5 mm in children (8 years and younger). Laminar point of C2 (laminar points are most anterior extent of neural canal margin of lamina) should be within 1.5 mm of line connecting laminar points of C1 and C3. (**D**) Coronal image of right upper quadrant (RUQ) from focused abdominal sonography for trauma (FAST) shows free intraperitoneal fluid in anterior subhepatic (Morison's) space (arrows), compatible with hemoperitoneum.

support lines and tubes.[1] Anteroposterior (AP) supine chest and AP supine pelvis radiographs are typically performed as a part of this initial assessment, if clinical evaluation alone is deemed insufficient (Fig. 19-1). A single-view, cross-table, horizontal beam cervical spine radiograph can be obtained to evaluate for gross cervical malalignment but should not be used to exclude all fractures of the cervical spine (see later section).

The trauma resuscitation ABCD strategy may be extended to this trauma series. Verification of the integrity of the

airway (and other tubes and lines) should occur on the chest radiograph. Radiographic pulmonary opacities associated with hypoxemia include pulmonary contusions, aspiration pneumonitis, and atelectasis (including collapse due to aspirated dental or foreign debris). Tension pneumothorax and hemothorax are typically detected on clinical examination, whereas clinically occult pneumothoraces or hemothoraces are commonly shown by chest x-rays as a deep sulcus sign and generalized hemithoracic opacification, respectively. Other injuries, such as rupture of the hemidiaphragm, flail chest, pneumopericardium, and pneumomediastinum and hemomediastinum, may be diagnosed or at least suggested by initial x-ray findings. Hemodynamic instability may arise from any number of causes, including extraperitoneal hemorrhage from pelvic ring disruption or hemoperitoneum from solid organ injuries. Biomechanically unstable disruptions of the pelvic ring are almost always shown on AP radiographs and may be associated with injuries to the bladder and urethra. In addition, pelvic x-rays may show hip dislocations and fractures of the acetabulum and proximal femur.

Although used less commonly, a technically adequate (C1–T1) lateral x-ray of the cervical spine is a limited examination but may provide helpful information in the setting of a cervical spine fracture/dislocation or other gross malalignment and may confirm that spinal shock from a vertebral fracture is the cause of unexplained hypotension.

Focused Assessment with Sonography in Trauma

Focused assessment with sonography in trauma (FAST) is performed as part of the secondary survey in victims of torso trauma to identify fluid accumulations in the chest and abdomen. These abnormal fluid collections serve as a proxy for significant internal injury. The ultrasound probe is used to search for fluid in the pericardial sac, both upper abdominal quadrants, and the intraperitoneal recesses in the pelvis adjacent to the bladder. Scanning is optimally performed in two orthogonal planes (eg, longitudinal and transverse) and may also be used to detect a hemothorax or a pneumothorax immediately superior to the perihepatic and perisplenic views. In patients who have severe hemodynamic compromise or obvious hemorrhagic shock, FAST can establish the abdomen as a source of hemorrhage within a few seconds. It is important to recognize that FAST is a limited anatomic examination and, as such, the identification of intraparenchymal and retroperitoneal injuries is not a goal, but certainly occurs. FAST is widely available, inexpensive, and noninvasive; uses no ionizing radiation; is portable; and can be repeated serially with little additional time and effort. Commercially available handheld real-time imaging devices are technically adequate to perform FAST.

FAST is uniformly accurate for the detection of intraperitoneal fluid with moderately large volumes greater than 400 cm^3 (at smaller volumes, accuracy varies with user experience).[2-4] Unfortunately, isolated hepatosplenic injuries with minimal or no hemoperitoneum represent as many as one-third of solid organ injuries.[5,6] Fortunately, small isolated intraparenchymal lesions with less than 250 mL of intraperitoneal blood often do not require endovascular or surgical intervention (liver <1%, spleen <5%).[7] False-positive interpretation of FAST images can result from improper machine settings (gain), sonolucent perinephric fat (which is rarely sonolucent in both axial and coronal scanning planes), fluid-filled bowel and bladder, various types of fluid-filled intra-abdominal cysts, physiologic fluid (especially in females), or preexisting nontraumatic free fluid (ascites). A final false-negative scenario outlines a negative pericardial window despite the underlying presence of a cardiac injury secondary to ejection of the blood out of the pericardial sac via an adjacent rent in the pleura or diaphragm.

The FAST does, however, require operator training and experience for reliable performance and interpretation, which may limit its value. In addition, FAST lacks value in patients with preexisting nontraumatic ascites, bowel and mesenteric injuries, injuries to the retroperitoneum, subcutaneous emphysema, and hemoperitoneum due to pelvic fractures. Although a systematic review of the literature would not support the use of FAST as a replacement for diagnostic peritoneal lavage (DPL) and CT in blunt abdominal trauma, many trained surgeon-sonographers use it on a daily basis with great accuracy.[8]

Hemodynamically stable patients who have suffered trauma with a clinical presentation suspicious for injuries, even in the setting of a positive FAST scan, are candidates for intravenous contrast-enhanced CT of the abdomen and pelvis to identify or exclude internal injuries.

COMPREHENSIVE IMAGING FOR BLUNT POLYTRAUMA

Blunt polytrauma victims are triaged based on the physical examination, initial imaging assessment, and hemodynamic status to the operating room, intensive care unit, or angiography suite for ongoing resuscitation. Some remain in the trauma center for completion of secondary and tertiary clinical surveys and advanced targeted imaging. Typically, this consists of CT for clinically appropriate imaging (eg, head, neck, chest, abdomen, pelvis, and lower extremities) for hemodynamically stable patients. Subsequently, conventional x-rays of the extremities may be obtained. Less severely injured individuals may take a slightly different route with conventional x-rays preceding CT and directed at abnormalities found by clinical examination or prior imaging.

There has been recent increased utilization of the "pan scan" or other variations of whole-body CT imaging techniques as a method of screening patients for clinically unsuspected injuries or to further define injuries identified or suspected on physical examination and initial imaging. This paradigm advocates rapid acquisition of high-resolution thin collimation images to include CT of any or all body parts, including the extremities in some instances. Some literature suggests that integrating whole-body CT as a part of the comprehensive evaluation of the trauma patient decreases

morbidity and mortality.[9,10] Employing this strategy provides excellent anatomic coverage with high accuracy for the detection and exclusion of injuries and is most frequently used in high-volume facilities with considerable imaging resources and expertise.

Multidetector Computed Tomography

The widespread availability of multidetector CT (MDCT) scanners has revolutionized the way that trauma patients are imaged.[11-13] Modern MDCT scanners, particularly those with 16 or more detector rows, acquire imaging data at submillimeter slice thickness. Because of this isotropic spatial resolution, two-dimensional (multiplanar) images in any arbitrary plane can be reconstructed with the same resolution as the axial images. Three-dimensional images with surface shading and cinematic rendering approximate that of anatomic specimens. Some of the newest software packages can reconstruct CT raw data sets to generate "virtual" radiographs (Fig. 19-2).

This, coupled with rapid intravenous contrast injection and increased table speed, allows for even shorter scan times as well as imaging large portions of the body during multiple phases of vascular and organ enhancement. As a result, patients can be scanned from the cranial vertex to feet in only a few seconds.[14,15] Reconstructions of any scanned body part, including the face, thoracolumbar spine, pelvis, and extremities, can be generated in multiple planes, thus reducing unnecessary repeat scanning. The previous practice of focused, specialized imaging of the face and spine can be abandoned. As such, individual repeat thin-section imaging of parts of the body in addition to the CT survey is superfluous, increases ionizing radiation unnecessarily, and increases time away from the resuscitation area.

Instead of sending all of the submillimeter thin-section data to a picture archiving and communication system (PACS) for review (which can be quite time consuming, despite improved image transfer time), the thinner slices are stacked together so that maximum useful information can be extracted without overwhelming the reviewer with high image volume. It is typical for images though the chest, abdomen, and pelvis to be reconstructed at 2- to 5-mm slice thickness in axial, sagittal, and coronal planes, although any slice thickness can be prescribed and tailored to local preferences. It is common practice for the axial images to be reconstructed using thicker slices (eg, 3–5 mm), with sagittal and coronal images reconstructed using thinner slices (eg, 1.5–3 mm) to provide finer spatial resolution of the pelvis and spine. Specific soft tissue and/or bone reconstruction algorithms can be applied to the CT raw data, again supplanting redundant focused imaging. Open communication with the radiology team is strongly encouraged so that optimal image quality and the imaging needs of the trauma surgeon and patient can be achieved. Consistency and familiarity with the scanning process and the image reconstructions can help improve patient throughput once a standard trauma CT survey is agreed upon, thus decreasing patient time away from the trauma care area.

In the era of "as low as reasonably achievable" (ALARA), there is concern regarding the radiation doses that trauma patients receive given the younger demographic of trauma patients, as well as the need for additional or repeat imaging for monitoring. Although dose reduction in CT is achievable, multiple studies have demonstrated cutoffs for minimum dose and noise levels for maintaining image and diagnostic quality in a variety of clinical settings. In the setting of trauma, it is essential to balance dose reduction with diagnostic quality given the potential for significant morbidity and mortality resulting from missed diagnoses in this patient population.

There are a number of radiation dose reduction techniques available. Aside from only scanning what is deemed necessary, there have been many technologic advances that have resulted in substantial radiation dose reduction delivery to the patient while maintaining diagnostic image quality. Inherently high-contrast structures, such as the bones and lungs, lend themselves well to dose reduction. Z-axis dose modulation can be employed to automatically reduce delivered dose through less dense parts of the body (eg, through the lungs), thus only using the dose necessary to maintain diagnostic image quality. Back-end computational methods for image reconstruction from the raw data can be used to reduce image noise, thus providing the potential to reduce radiation dose while maintaining desired image quality.

Computed Tomography of the Head

Axial noncontrast CT scanning remains the reference standard in patients with acute craniocerebral trauma, guiding initial decisions regarding clinical management.[16,17] Indications for CT of the head include the following: (1) objective evidence of closed injury to the brain, including decreased level of consciousness; (2) cranial or facial deformity; (3) hemotympanum; or (4) evidence for leakage of cerebrospinal fluid (Figs. 19-3 to 19-5). Clinical criteria reliably predict significant intracranial injury and help determine which patients will require CT scanning of the head. In children, significant intracranial injury is extremely unlikely in any child who does not exhibit at least one of the following high-risk criteria: (1) evidence of significant skull fracture; (2) altered level of alertness; (3) neurologic deficit; (4) persisting vomiting; (5) scalp hematoma; (6) abnormal behavior; or (7) coagulopathy.[18] More generically, minor trauma to the head may lead to surgically important injuries to the brain, and liberal utilization of CT is appropriate among individuals who have sustained "high-risk" mechanisms. These clinical criteria are not as reliable in the elderly patient.[19]

Head CT images are traditionally acquired using a conventional "step and shoot" axial technique. Most modern MDCT scanners, however, have the capability of scanning the head using helical acquisition, as used for other body parts, and can thus reconstruct head CT images in both sagittal and coronal planes. Traditionally, axial images are reconstructed at 5-mm slice thickness using both bone and soft tissue algorithms and are viewed using bone windows and at least two different soft tissue windows (including "brain" and "blood" window settings).

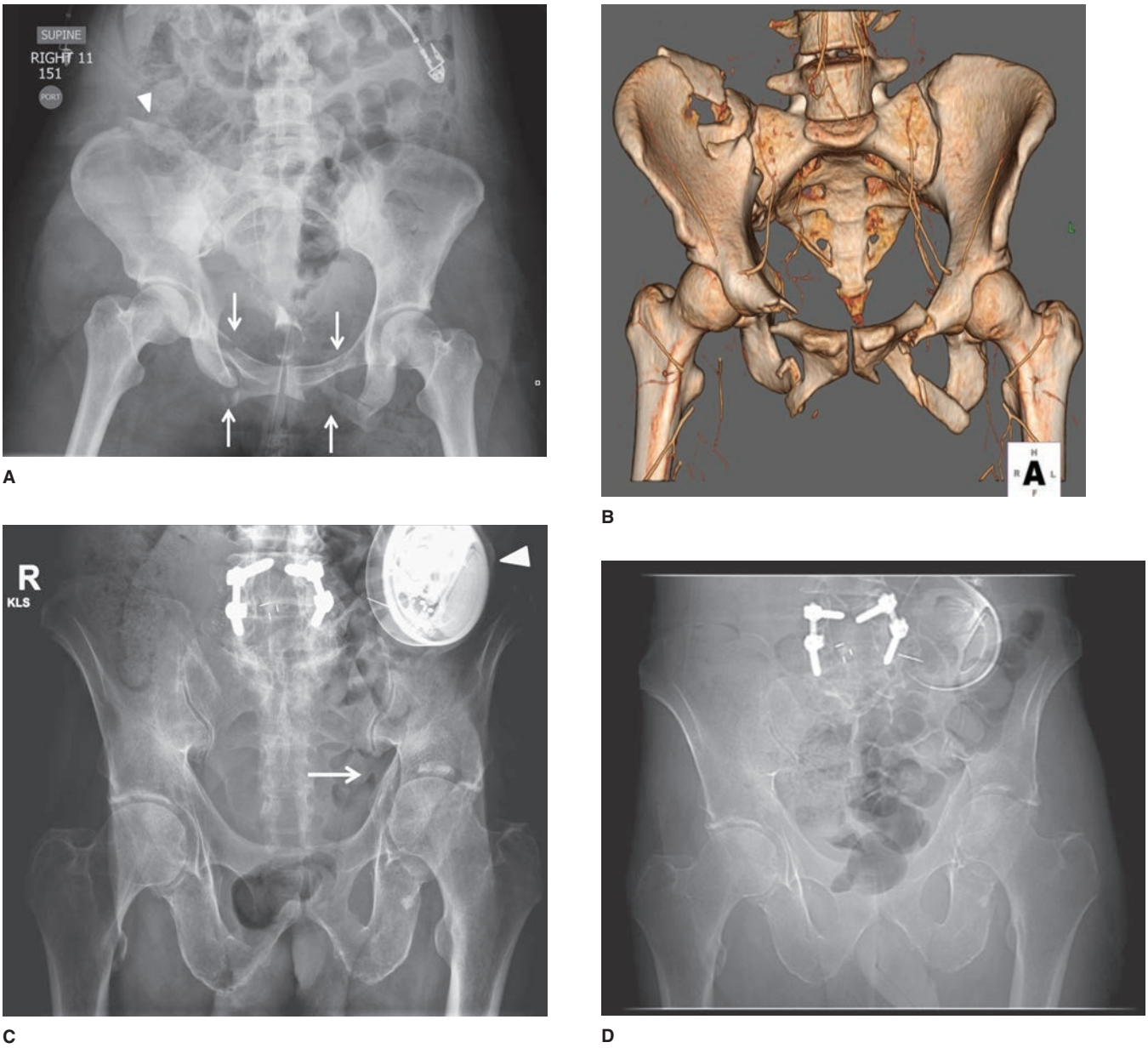
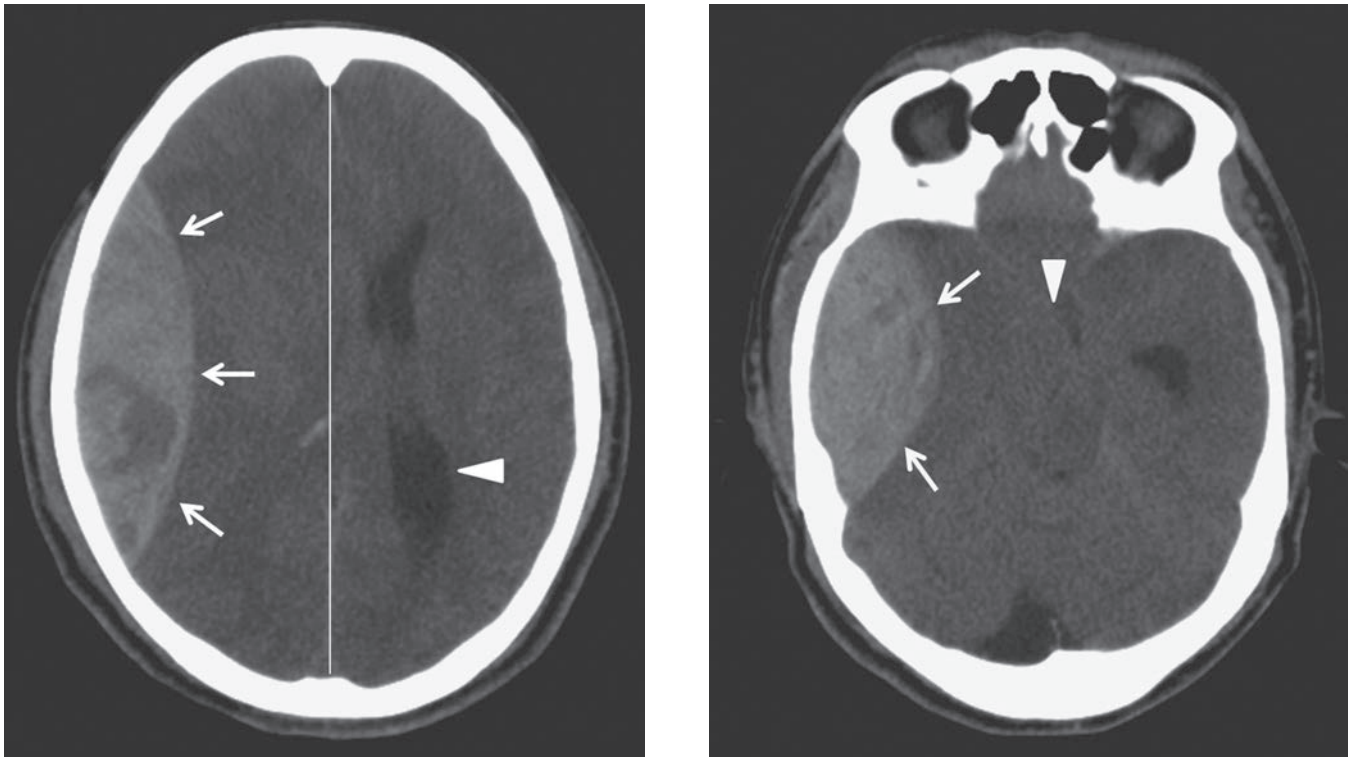


FIGURE 19-2 Advanced computed tomography (CT) imaging depicting pelvic and acetabular fractures. (**A**) Anteroposterior (AP) frontal radiograph of a 34-year-old female following a motorcycle crash. There is a highly disorganized pelvic ring disruption with fractures of the superior and inferior pubic rami (arrows) indicating a lateral compression force vector. There is also a comminuted fracture of the right iliac crest (arrow head). Notice the vertical height difference indicating a vertical shear mechanism of injury in addition to lateral compression. (**B**) Three-dimensional image with surface detail from the same patient nicely displays the bony fractures and their relationships to one another, with overlying soft tissue, bowel gas, and stool no longer a limiting factor. (**C**) AP frontal radiograph of a 74-year-old male with a left acetabular fracture (arrow) following a fall from standing height. The patient has L4–L5 posterior fixation hardware as well as a pain pump reservoir (arrowhead) overlying the left ilium. (**D**) A "virtual" pelvic radiograph on the same patient as in **C**, generated using the raw data from a subsequently performed pelvic CT. Notice that the pain pump reservoir has been partially removed from the image.

CT scanning is highly sensitive and specific for the detection of extra- and intra-axial hemorrhage, mass effect and midline shift, soft tissue injuries to the scalp and globes, and fractures to the calvarium, skull base, bony orbits, and paranasal sinuses. In patients with diffuse axonal injury, however, CT may even be normal and discordant between the severity of the clinical brain injury and radiographic findings (Fig. 19-4). On a cautionary note, skull fractures that are aligned in the plane of scanning may easily elude detection, and review of multiplanar reconstruction (MPR) images or the scout views used to plan the CT study of the head may alert the clinician to such fractures. Except for medicolegal imaging of pediatric nonaccidental trauma (child abuse), conventional x-rays of the skull are usually not necessary.[20]



A B

FIGURE 19-3 Epidural hematoma. (**A**) Axial computed tomography (CT) using brain windows shows epidural hematoma (arrows), associated midline shift (white line), and leftward deviation of the left lateral ventricle (arrowhead). (**B**) Axial CT at level of the suprasellar cistern and brain windows shows epidural hematoma (arrow) and obliteration of the basal cisterns due to uncal herniation (arrowhead).

Computed Tomography of the Maxillofacial Skeleton

Indications for specific facial CT scans include the following: (1) deformity or instability of the maxillofacial structures found by physical examination; (2) deformity, opacification, or fracture of the periorbital or paranasal sinus shown on head CT; and (3) clinical evidence for leakage of cerebrospinal fluid (Fig. 19-6). The mnemonic LIPS-N (lip lacerations, intraoral lacerations, periorbital contusions, subconjunctival hemorrhage, or nasal lacerations) provides a helpful tool during clinical examination of trauma patients given the high association between LIPS-N lesions and facial fractures.[21] Fortunately, physical examination is highly sensitive for selecting which patients need maxillofacial CT imaging.[22] Absence of paranasal or periorbital sinus fluid on CT (the "clean sinus" sign) generally excludes surgically important injury to the maxillofacial skeleton, with only very rare exceptions.[23]

Images of the face are acquired using helical scanning technique and are reconstructed in the axial, sagittal, and coronal planes at 1- to 2-mm slice thickness, typically using both soft tissue and bone reconstruction algorithms. Helical acquisition with MDCT technology can be used to generate three-dimensional (3D) surface-rendered images of the bones, which can be quite helpful in determining the type of facial fracture patterns, particularly for patients with highly comminuted and disorganized high-energy midface smash fracture patterns. This technique provides valuable information on spatial relationships and is particularly useful in planning operative treatment.[24,25] However, axial and two-dimensional reformations best portray defects in soft tissue, interposition of osseous fragments, and herniations of soft tissue (eg, orbital floor blowout fractures) and are best for quantifying size of fractures.[26,27] It is important to recognize that some institutions do not routinely include the mandible as a part of the maxillofacial CT scan protocol; thus, open communication with the radiology team is important in order to include the relevant anatomy for evaluation.

Radiation reduction techniques for CT continue to evolve; however, maxillofacial CT can still deliver significant radiation to the orbits and to the soft tissues of the neck (eg, thyroid), which is an important consideration when imaging children in particular.[28]

Imaging for Soft Tissue Injuries of the Neck

Clinical findings for blunt injury to the aerodigestive tract are nonspecific but include subcutaneous crepitus, hemoptysis, hoarseness, neck pain, and abrasions or hematomas (eg, from the shoulder harness of a three-point restraint; Figs. 19-7 and 19-8). X-ray findings include parapharyngeal or precervical

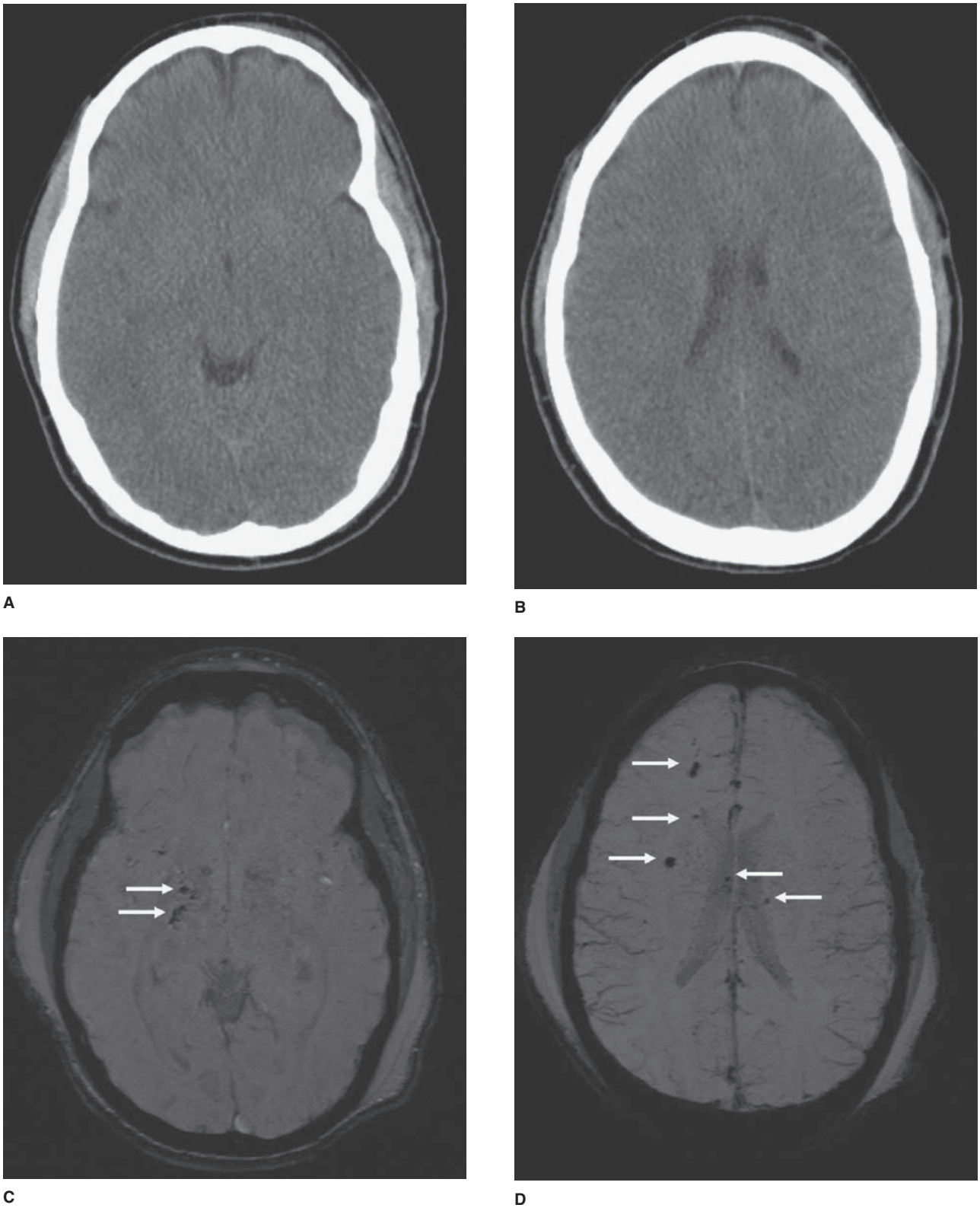
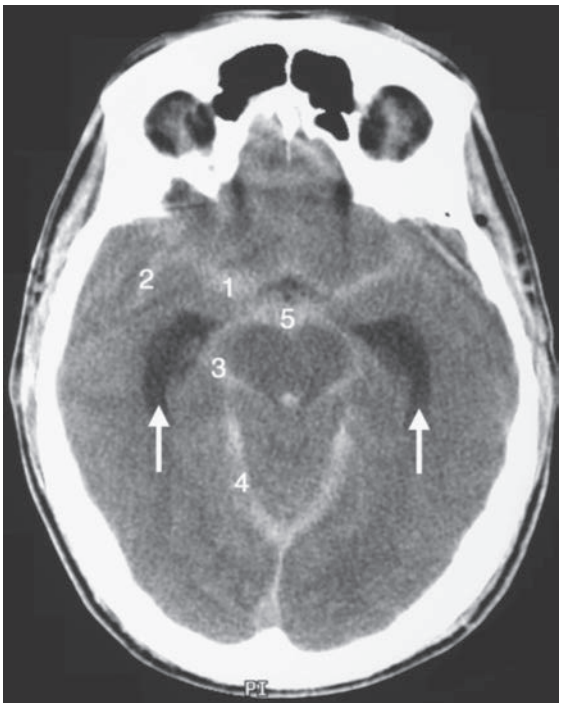


FIGURE 19-4 Blunt head injury: axonal shear injury. A 24-year-old helmeted male following a high-speed motorcycle collision. Admission axial noncontrast computed tomography (CT) at level of the third ventricle (**A**) and lateral ventricles (**B**) demonstrates no significant abnormality. The patient's neurologic status did not improve; therefore, brain magnetic resonance imaging (MRI) was performed 25 hours after admission (**C, D**). Axial MRIs at the same levels as **A** and **B** using gradient echo (GRE) sequences (which are highly sensitive for blood products) demonstrated numerous hemorrhagic foci throughout the brain, concentrated in the right basal ganglia (**C**) and the gray-white junction (**D**) (arrows).



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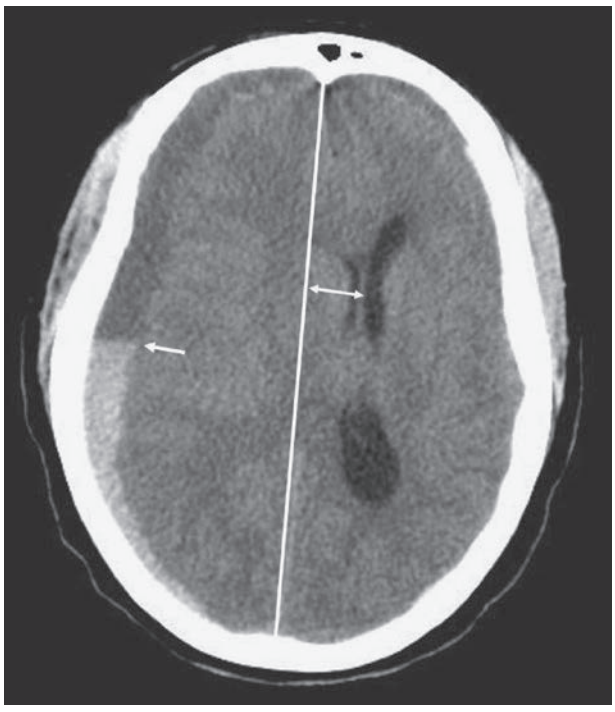

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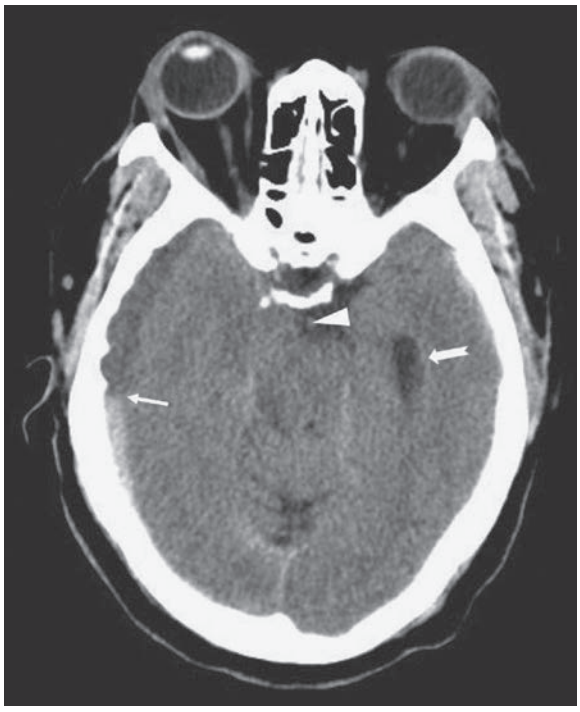


C

FIGURE 19-5 Patterns of herniation. (**A**) Axial computed tomography (CT) at level of suprasellar cistern shows extensive subarachnoid hemorrhage extending from lateral aspect of suprasellar cistern (1), into the Sylvian fissure (2), circumferentially about brainstem and perimesencephalic cistern (3), along tentorium (4), and interpeduncular cistern (5). Entrapment of lateral ventricles is shown as dilatation of temporal horns (white arrows). Brainstem appears relatively lucent and heart shaped, with pointed inferior portion of heart due to "beaking" of mesencephalon due to upward herniation. (**B**) Subfalcine shift. Axial CT of another patient at level of lateral ventricles shows marked leftward subfalcine shift (white double arrow), quantified as distance from midline (vertical white line) connecting the anterior and posterior portions of the sagittal sinus (which tend not to shift due to their fixed relation to calvarium) to the interventricular septum. Note a right-sided holohemispheric subdural hematoma with hematocrit level (white arrow). (**C**) Uncal herniation. Axial CT of the same patient as in **B** at the level of middle cranial fossa again shows right-sided subdural hematoma (white arrow) with associated right uncal herniation (arrowhead). Enlargement of the left temporal horn (notched white arrow) is compatible with obstruction of the cisternal system.

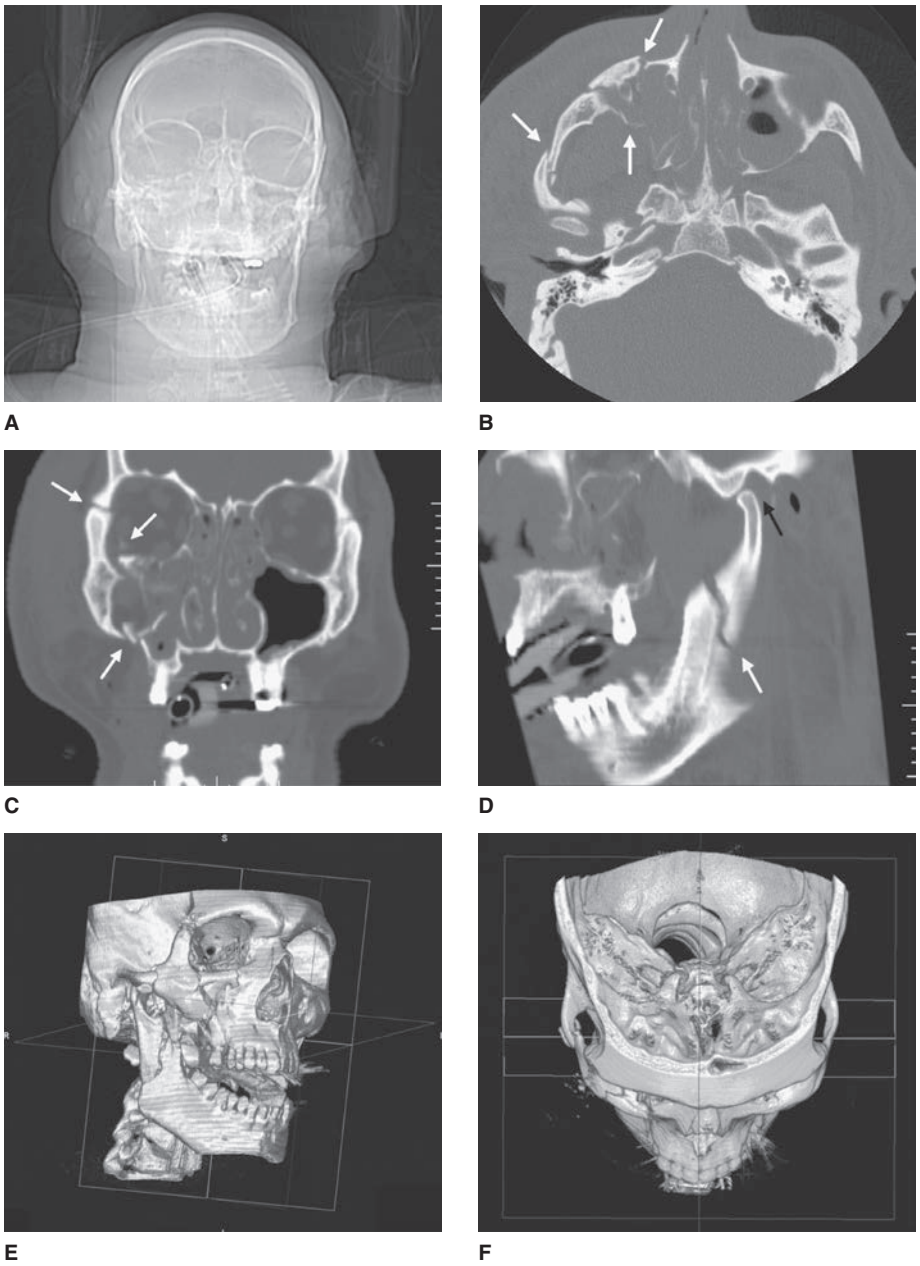
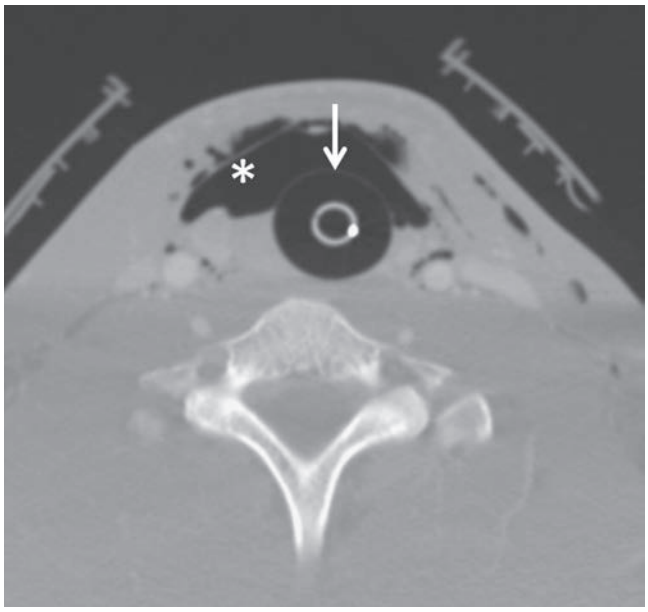


FIGURE 19-6 Facial fracture: zygomaticomaxillary complex (ZMC) fracture with associated naso-orbito-ethmoid (NOE) complex fracture. This 54-year-old male sustained a blow to the face in a motorcycle crash. (**A**) Anteroposterior (AP) scanogram shows loss of symmetry to orbital volumes, with elliptoid enlargement of right orbit. Associated indistinctness of orbital floor and lateral maxillary sinus walls is also present. Opacification of right maxillary sinus is shown. (**B**) Axial computed tomography (CT) image at the level of zygomatic arches shows depression and overriding apposition of impacted zygomatic arch fracture (lateral white arrow), posterolateral maxillary sinus wall disruption (posterior arrow), and segmental comminuted fracture of anterior maxillary wall (anterior arrow). Medial to this anterior arrow is the base of the nasofrontal process of the maxilla with a fractured nasolacrimal duct just posterior to it. Internal rotation of the nasofrontal process of the maxilla and associated fracture of the nasolacrimal duct are not portions of ZMC fracture and represent an associated NOE complex fracture. (**C**) Coronal CT reformation shows separation of right frontozygomatic suture (lateral and superior arrow), disruption of orbital floor (white arrow projected over orbit), and lateral maxillary wall (inferior white arrow). (**D**) Sagittal CT reformation shows associated vertical fracture of right ascending ramus of mandible, with anterior subluxation at temporomandibular joint (white and black arrows, respectively). (**E**) Three-dimensional CT reformation gives an overview of complex fracture of zygomaticomaxillary region and right mandibular fracture. It is important to note that spatial resolution is lost with three-dimensional reformations, although spatial comprehension is often improved. (**F**) Three-dimensional CT reformation shows depression of right zygomatic arch and loss of projection of the right zygoma (flat cheek).



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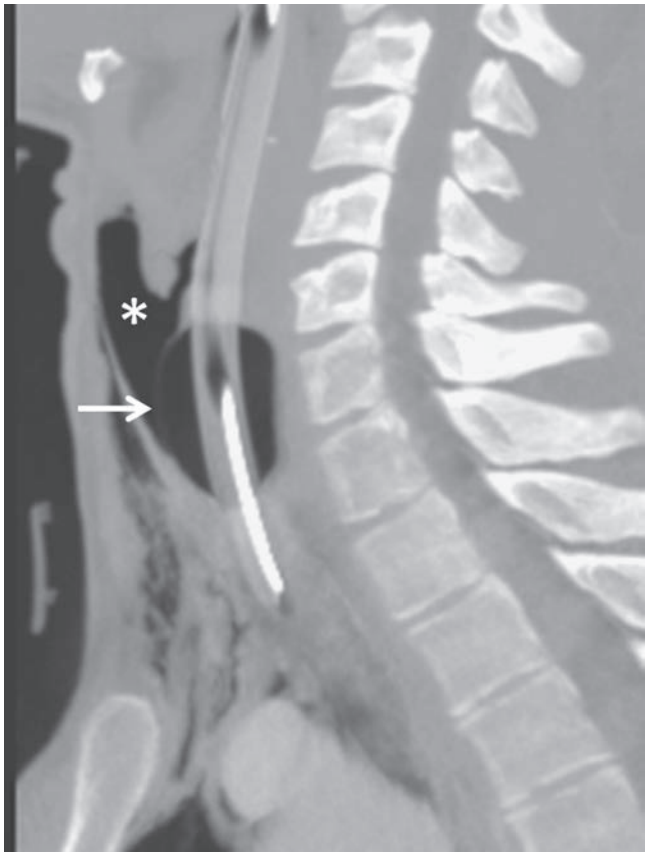

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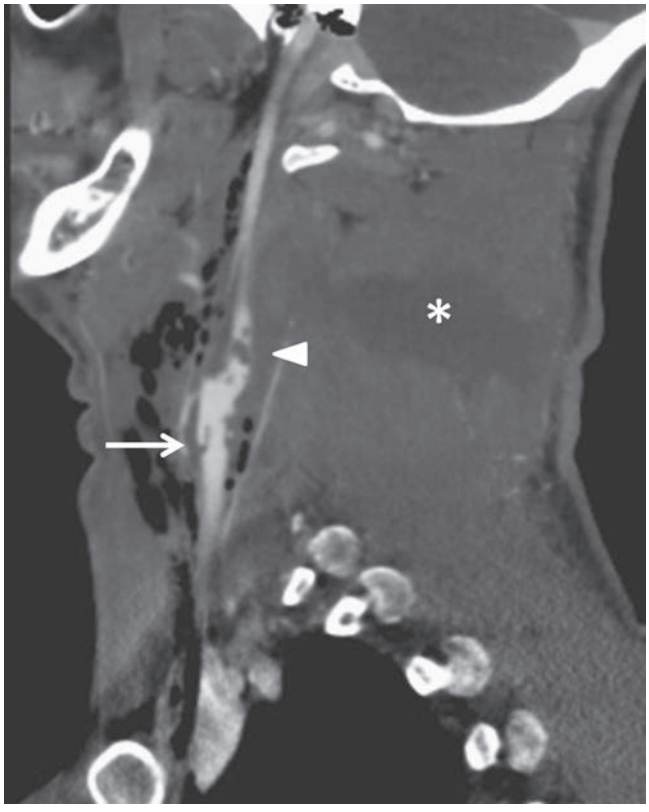

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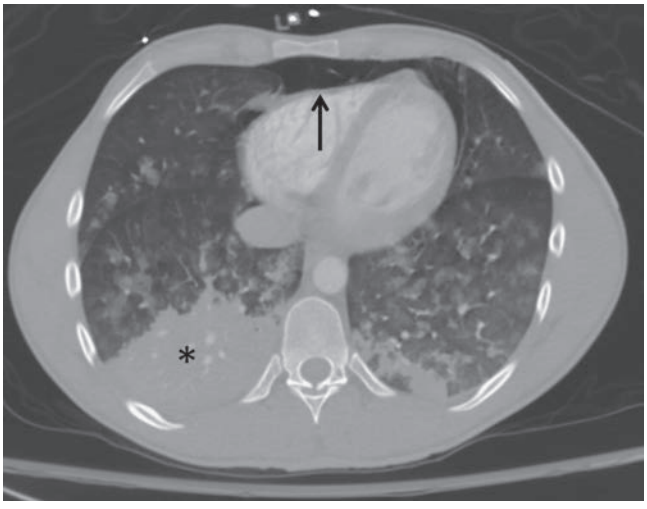
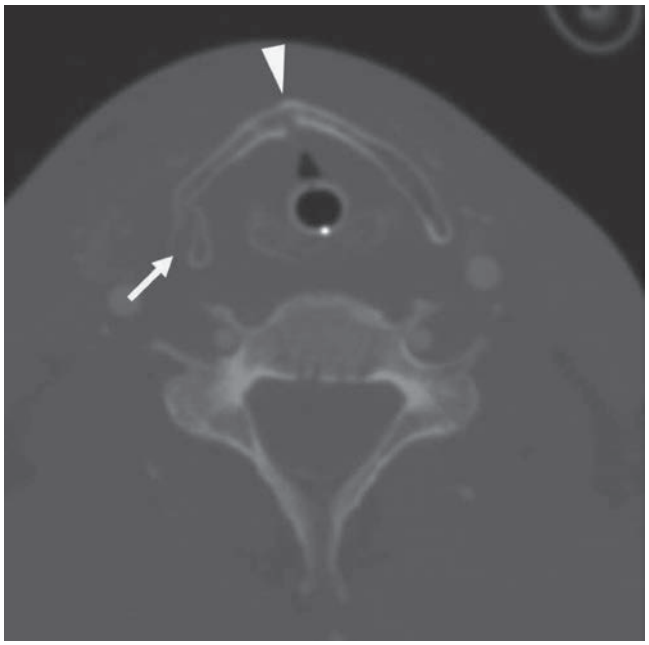


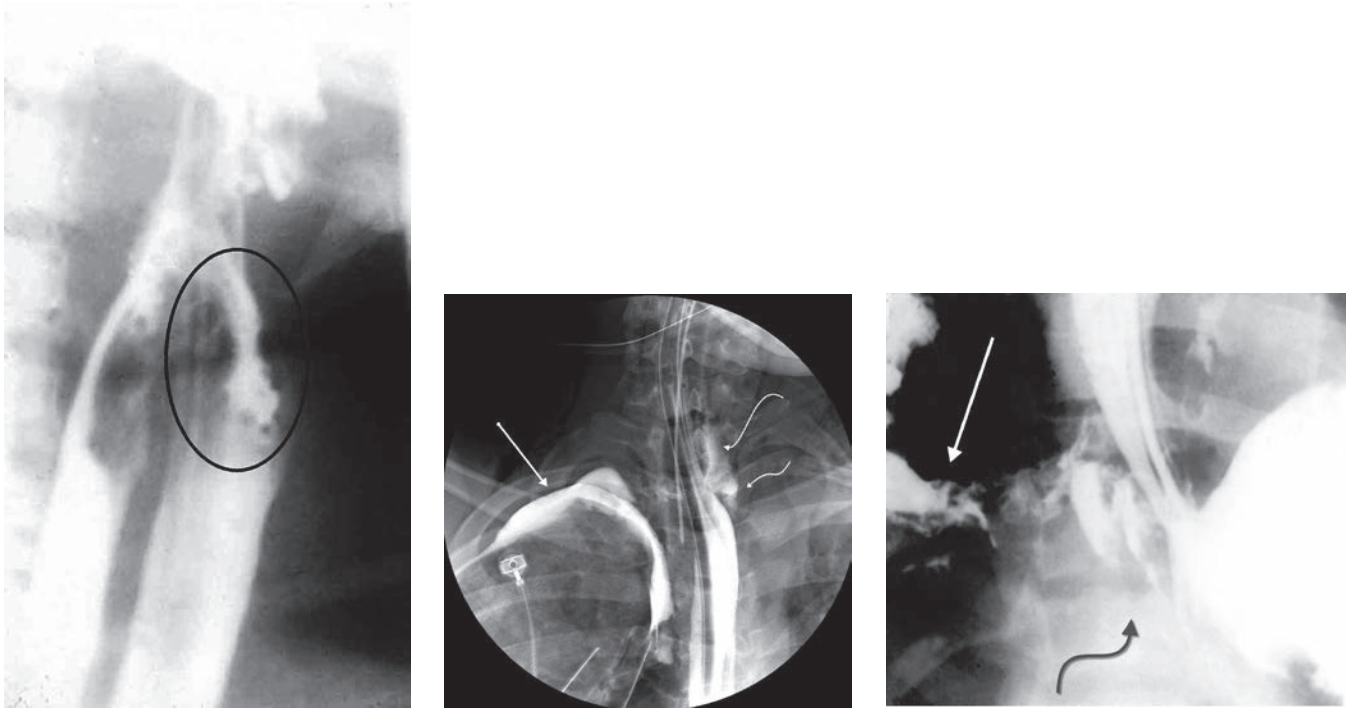
D

FIGURE 19-7 Tracheal, soft tissue, and vascular neck injuries. (**A–D**) This is a 21-year-old male motorcyclist with a "clothesline injury," resulting in tracheal transection, as well as a complex cervical vascular injury and neck muscle injuries. (**A**) Axial and (**B**) sagittal computed tomography (CT) images using lung windows reveal an endotracheal balloon inflated in a tracheal defect (arrows). There is surrounding cervical emphysema (asterisk). (**C**) Sagittal CT image using soft tissue windows reveals a complex injury of the carotid bulb (arrow) and proximal internal carotid artery (arrowhead). There is an associated neck soft tissue injury manifested by fluid in the muscular defect (asterisk). (**D**) Axial CT image through the chest using lung windows reveals pneumomediastinum (black arrow) resulting from inferior extension of cervical emphysema. There is extensive right greater than left bilateral aspiration (black asterisk).



E

FIGURE 19-7 (*Continued*) (**E**) Axial CT from a 36-year-old male who was hit in the neck with a hockey puck reveals a fracture of the midline (triangle) and right side (arrow) of the thyroid cartilage. The patient was intubated due to immediate voice changes and to protect the airway.



A B C

FIGURE 19-8 Esophageal injuries. (**A**) Gunshot wound in zone II of the neck of a teenager. Barium extravasation directly enters the airway from a high laryngoesophageal fistula (circled). (**B**) Gunshot wound traversing zone I into the right chest. Barium swallow shows leak on both the left side of the cervical esophagus (curved arrows) and the right thoracic esophagus (straight arrow). (**C**) A 27-year-old male sustained a low transverse mediastinal gunshot wound entering left and exiting right. Gastrografin esophagram shows leak into the right chest (straight right arrow) and into the peritoneal cavity (curved black arrow).

emphysema, soft tissue swelling, or fracture of the larynx or hyoid bone on the lateral image of the cervical spine. Soft tissue injuries are inadequately evaluated using radiography; hence CT is the accepted imaging standard for imaging this area.

MDCT of the neck with intravenous contrast can not only be used to evaluate for upper aerodigestive tract injuries, but can also be used to simultaneously evaluate the cervical vasculature and spine (see later). Images are routinely reconstructed in three planes using both soft tissue and bone algorithms.

CT is often most helpful in the evaluation of laryngotracheal injuries when physical examination and endoscopy are technically difficult (Fig. 19-7E).[29] A careful search for direct signs of a laryngotracheal injury, manifested by focal defects, guides the need for intervention for debridement and mucosal closure. Soft tissue gas may be focal adjacent to the site of injury, but larger defects, particularly if the patient is under positive-pressure ventilation, can result in massive soft tissue emphysema extending into the skull base and head superiorly or into the mediastinum and beyond inferiorly.[30] CT can assist in the grading of laryngotracheal injuries but tends to understage injuries compared to endoscopy or open exploration. The most common injuries are those to the thyroid cartilage, which typically occur within 2 to 3 mm of the anterior crest of the two lateral laminae. Comminuted fractures of the laminae generally result from higher energy and direct impact injuries to the larynx and are more commonly associated with thyrocricoid dislocation. In addition, CT can demonstrate subluxations and dislocations of the arytenoid cartilage. Most tracheal disruptions are in the membranous portion of the trachea and can heal with conservative therapy. Soft tissue emphysema immediately adjacent to the trachea suggests an injury in this location but, again, could be massive in volume and anatomic distribution. A search for a fracture of a tracheal ring is often fruitless unless the fracture is displaced. Coronal reformations through the trachea may show separation (vertical diastasis between tracheal rings) of the trachea compatible with more serious grades of injury.

Blunt esophageal injuries are very uncommon, but when they do occur, they are most often observed in the proximal third of the esophagus. However, a penetrating injury to the esophagus is more common and may involve any portion of the esophagus. Any focal gas adjacent to the esophagus, without an obvious airway source, should raise the possibility of an injury, particularly if a wound tract extends up to or through the region of the esophagus.

If there is a concern for an esophageal injury, contrast esophagography, endoscopy, or both should be performed. Both tests have a sensitivity of 80% to 90%, but this increases to approximately 95% when both examinations are performed.[31,32] As a result, clinicians must use their level of suspicion to determine which of these tests (or both) to employ. Iso-osmolar water-soluble contrast material is the preferred *initial* contrast agent of choice for suspected esophageal injuries because it is less toxic to the lungs if aspirated and is better tolerated if leaked into the mediastinum or peritoneum. Unfortunately, iso-osmolar contrast material is less dense on fluoroscopy, with the potential to miss subtle injuries. Barium sulfate is easier to see but can be irritating to the soft tissues if an esophageal injury is present and should be used after initial water-soluble esophagography is negative. Gastrografin should be avoided because of its hyperosmolarity and because it can cause life-threatening pulmonary edema if aspirated.

Esophagography performed on patients who are awake and able to cooperate with the examination is performed as any other esophagram, but with the contrast agent modifications as described earlier. Performing esophagography in intubated and/or sedated patients is technically challenging and often limited despite maximal efforts on the part of the radiology team. A nasogastric tube is necessary to perform this examination on obtunded, intubated, and/or sedated patients. The patient is placed in reverse Trendelenburg position (10–20° as tolerated) with the fluoroscopy table floor board in place. The radiologist manipulates the nasogastric tube to the distal esophagus and, under real-time fluoroscopic surveillance, injects the contrast agent in an attempt to achieve full distention of the esophagus. The contrast is then immediately aspirated, and the nasogastric tube is repositioned more proximally. This process is repeated to evaluate all segments of the esophagus. Care should be taken to avoid overdistention of the proximal esophagus, which can result in reflux and aspiration, even in intubated patients. Rapid imaging is necessary, with fluoroscopic cineradiographs obtained at 2 to 3 frames per second.

IMAGING OF CERVICAL VASCULAR INJURIES

Neck CT angiography (CTA) with MDCT has a sensitivity of 90% to 100% for blunt cervical vascular injuries and is, therefore, the imaging test of choice in patients without indications for immediate surgery or catheter angiography. Neck CTA can be performed as its own stand-alone examination (as in the setting of penetrating neck injuries), added on to the standard trauma CT protocol, or integrated as a part of a single-pass whole-body CT trauma protocol.[33-37] Neck CTA protocols vary widely based on local preferences and mechanism of injury (ie, blunt vs penetrating). Positioning of the patient's arms is an important consideration when integrating neck CTA into the imaging evaluation of these patients, as increased image noise and decreased image quality will result when the arms are included in the scanned region.[37] Optimal cervical arterial opacification can be obtained following either a timing bolus or using a fixed scan delay. These images are reconstructed at 1- to 3-mm slice thickness from the aortic arch to the skull base (or to the cranial vertex if needed), with MPR reconstructed in axial, sagittal, and coronal planes. Coronal maximum-intensity projections (MIPs) are particularly helpful for visualizing the cervical vasculature but should not be relied upon for evaluation of the vertebral arteries due to volume averaging effects and the potential for a missed injury. Liberal use of a dedicated 3D volume viewing station

for manipulation of the data set is strongly encouraged, as it is often the case that a cervical vascular injury may be best visualized and displayed using nontraditional planes.

Catheter angiography is indicated emergently in patients with an expanding cervical hematoma; active extravasation from the nose, mouth, or ears; or a cervical bruit in individuals younger than 50 years old.[38] Angiography is not only a diagnostic test in symptomatic blunt carotid and vertebral artery injuries, but is also a therapeutic tool as it allows for rapid utilization of endovascular therapy for bleeding and identifies patients at risk for embolic stroke.[39,40] The sensitivity of duplex Doppler US is inadequately low (38.5%) for directly depicting vascular injuries in the neck and is, therefore, not a primary tool for evaluation of the cervical vasculature.[41] Existing literature for the use of magnetic resonance angiography (MRA) demonstrates limited sensitivity for cervical vascular injuries compared to CTA and catheter angiography.[42] This, in addition to the technical and practical limitations of MRI in the setting of trauma, limits the utility of magnetic resonance imaging (MRI)/MRA in the acute setting.

BLUNT CERVICAL VASCULAR INJURIES

Blunt carotid and vertebral injuries (Fig. 19-9A–C) are now known to be much more common than previously appreciated, occurring in 1% to 3% of screened asymptomatic patients.[35] Vascular imaging of the neck is warranted in injury patterns associated with a high risk of blunt injury to the carotid and vertebral arteries, including the following: fracture of the cervical spine (especially involving C1–C3, involving the transverse foramina, or extension into the foramen magnum), neurologic deficits not explained by findings at brain imaging, new-onset Horner syndrome, high-energy facial fractures (Le Fort II or III), fracture of the skull base involving the foramen lacerum, or soft tissue injury in the neck (neck belt sign and "hangings" sufficient to cause central anoxia).[39,43] It should be noted, however, that approximately one-fifth of patients with blunt cervical vascular injuries do not have these classically described risk factors and thus may elude detection if selective screening is employed.[44]

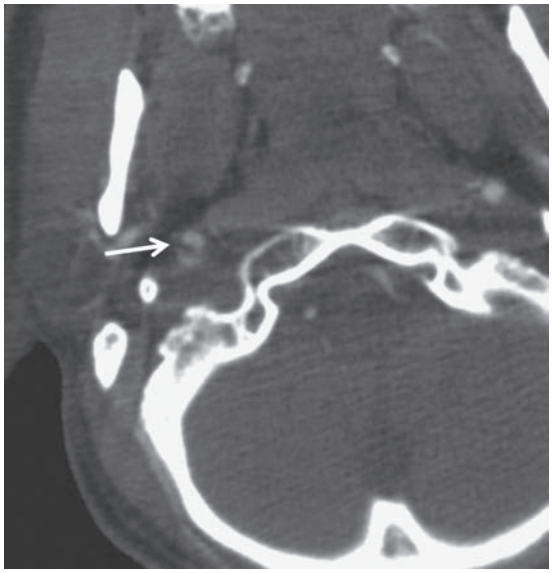
PENETRATING NECK INJURIES

Penetrating injuries to the neck are associated with high morbidity and mortality, primarily due to injuries of the cervical vasculature, upper aerodigestive tract, and spine. As such, any patient with "hard" signs of a cervical vascular injury (eg, expanding pulsatile hematoma, active bleeding from the wound, hemorrhagic shock) or compromise or injury of the upper aerodigestive tract or airway (eg, stridor, hoarseness) requires urgent intervention by individuals with experience managing such injuries. Neck CTA has proven highly useful in identifying and excluding injuries that require intervention and can significantly reduce the rate of nontherapeutic cervical explorations for patients without indications for immediate surgery or intervention[45-48] (Fig. 19-9D–E). In addition, it can be used to determine trajectory and identify direct signs of vascular injury. When patients are stable or asymptomatic, immediate CTA has great value in excluding injury, in detecting vascular injuries that can be treated nonoperatively by embolization or insertion of a stent or stent graft, and in facilitating surgical exploration.

There has been a shift away from mandatory evaluation of the neck with surgery and other invasive tests based on the zonal wound location toward a "no zone" selective approach with CTA evaluation of the neck.[49] Many factors must be considered when approaching the patient with a penetrating wound to the neck, including the mechanism of injury (high vs low energy), the number and location of surface wounds, hemodynamic and neurologic status, and the likelihood of injury. Gunshot wounds can result in significant soft tissue cavitation that may result in direct or indirect vascular injuries. In addition, fragments of bone and metal frequently cause artifacts in the area, which make interpreting a CT difficult. Catheter angiography (particularly digital subtraction angiography) is extremely useful for the evaluation of the carotid and vertebral arteries when they are partially obscured by beam hardening artifact from metallic bullet fragments.

Neck zonal anatomy is an important consideration for patients with "hard" clinical signs of significant internal injury; however, it should be noted that clinically significant internal injuries may be present in a zone differing from that of the physical examination and that penetrating wound tracts may extend beyond the neck into the skull base, cranial vault, mediastinum, or upper thorax. Zone III, above the angle of the mandible extending to the skull base, is difficult to assess clinically and to explore operatively. There are numerous vessels at risk, including the internal carotid artery, the external carotid artery and branches, the vertebral artery, and the accompanying veins. Thus, imaging plays a vital role in these patients. Neck CTA can be used to evaluate these vessels, but a low threshold for catheter angiography should be maintained for patients with possible injuries in zone III. Selective internal and external carotid arteriography, vertebral arteriography, and four-vessel intracranial arteriography all play important roles in detecting and evaluating zone III vascular injuries. Indeed, arteriography is so valuable for the evaluation and treatment of bleeding in this zone that aggressive steps to resuscitate the unstable patient and control bleeding by packing are warranted to allow angiography to proceed. Most vascular injuries in zone III are best managed by an experienced interventional radiology service.

Zone II, between the inferior margin of the angle of the mandible and cricoid cartilage, is more easily and reliably evaluated by physical examination. If the common carotid artery or the internal jugular vein requires repair, operative exposure is relatively simple and unobstructed. Thus, little imaging is necessary after penetration when "hard" signs of a vascular injury are present. The detection and treatment of vascular injuries in asymptomatic patients are more controversial.



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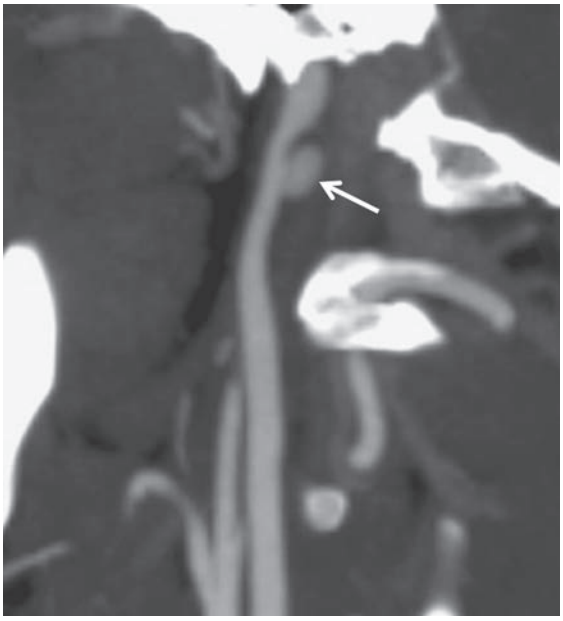

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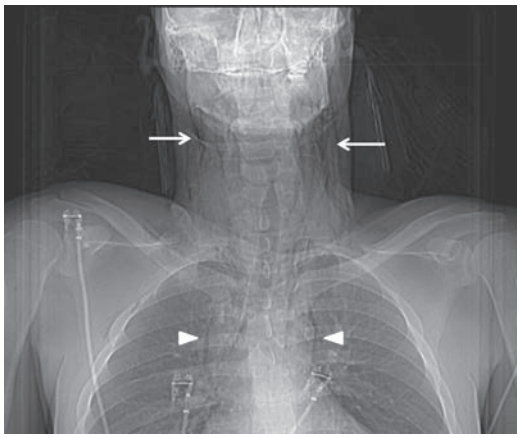

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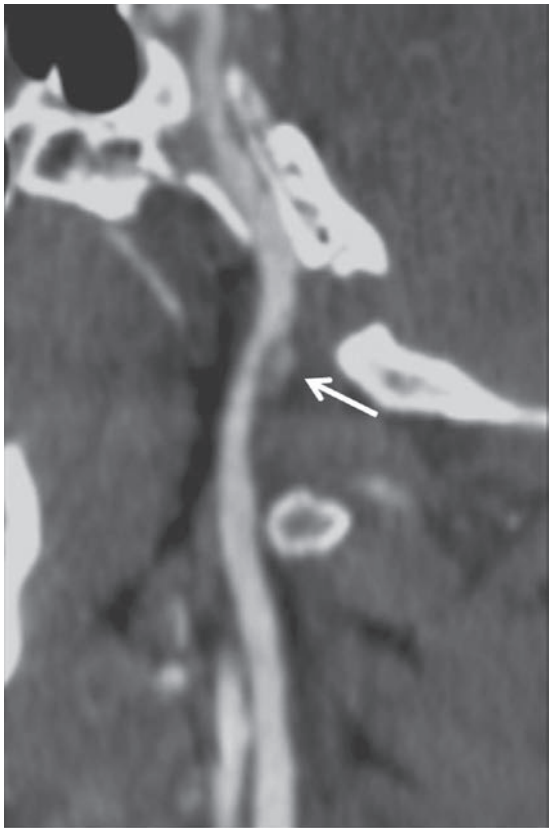

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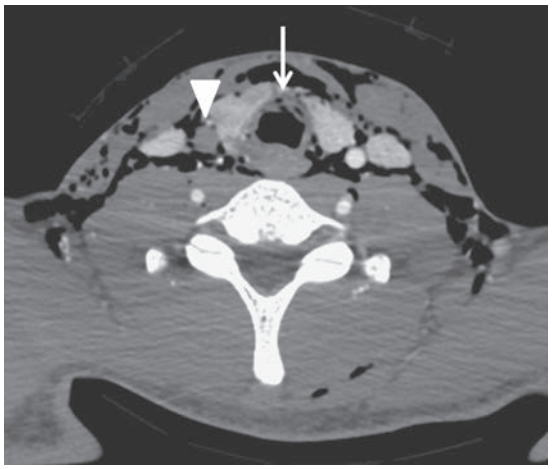

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FIGURE 19-9 Cervical vascular injuries. (**A**) A 24-year-old female involved in a high-speed motor vehicle collision. Admission axial computed tomography (CT) image demonstrates an intimal flap in the distal internal carotid artery (arrow) that is actually a pseudoaneurysm seen best on the sagittal image (**B**). The patient was treated conservatively. (**C**) Repeat neck CT angiography 20 days after injury reveals an enlarging pseudoaneurysm (arrow). (**D**) A 24-year-old male with common carotid artery and tracheal injuries following a dog attack. The right common carotid artery is not opacified (arrow) due to injury and subsequent thrombosis. A tracheal defect (arrowhead) is present, along with extensive cervical emphysema. (**E**) The extent of the cervical emphysema (arrows) is best seen on the scout image with gas extending inferiorly into the mediastinum (arrowheads).

Zone I is the area between the cricoid cartilage and the sternal notch. Structures here include the brachiocephalic vessels, trachea, and esophagus. Vascular injuries in this zone may result in rapid exsanguination, and as a result, symptomatic patients may undergo urgent exploration without imaging.

IMAGING OF THE SPINE

Imaging of spinal trauma often begins with simple radiography to evaluate for biomechanically unstable injuries, such as burst fractures and dislocations. It is critical to note that adequate radiographs of the spine may be technically difficult to obtain, may have limited quality in obtunded or immobilized patients, and may not detect all fractures. A meta-analysis of the sensitivity of radiography for cervical spine fractures by Holmes and Akkinepalli[50] documented a pooled sensitivity of 52%, compared to 98% for cervical spine CT. Thus, the decision to image with radiography or CT must be made based on the mechanism of injury and the likelihood of a spinal fracture and must be balanced with the possibility of other more life-threatening injuries. If an abnormality is identified on conventional x-rays, CT and/or MRI can be used to further characterize the injury for planning of treatment and provide information on the patient's prognosis.

Conventional Radiographs of the Spine

Validated clinical prediction rules (ie, National Emergency X-Radiography Utilization Study Group [NEXUS] and the Canadian Cervical Spine Rule) for adults and older children (≥10 years old) can reliably identify trauma victims who need imaging of the cervical spine.[51,52] In essence, oriented asymptomatic individuals without findings on a physical examination following trauma do not require subsequent imaging. Imaging of the thoracic and lumbar spine following blunt trauma is indicated when patients present with one or more of the following: (1) signs or symptoms of local injury (pain, tenderness, interspinous step-off); (2) depressed level of consciousness, including intoxication; (3) acute myelopathy or radiculopathy referable to the thoracolumbar spine; and (4) major distracting injury, including concomitant injuries to the cervical spine.[53,54]

A cross-table lateral cervical spine radiograph is often obtained in the setting of acute trauma, although its use is declining due to the easy availability of MDCT and relative lack of sensitivity compared to CT. Single-view lateral cervical spine films may not reveal a significant percentage of fractures. In fact, a study by Brohi et al[55] in 2005 reported a sensitivity of 39.3% and a sensitivity of 51.7% for single lateral view radiography evaluating unstable cervical injuries. This same study reported the sensitivity, specificity, and negative predictive value of CT to be 98.1%, 98.8%, and 99.7%, respectively.[55] The standard three-view cervical spine series includes an AP open-mouth odontoid view (an AP image of the craniocervical junction), an AP view of the subdental cervical spine, and a lateral view of the cervical spine that extends down to the C7–T1 interspace. To supplement these three views, a swimmer's lateral image (one arm elevated above the head and the other arm in caudal traction) is often obtained to better visualize the cervicothoracic junction, and bilateral oblique views are obtained to evaluate the facet joints. The addition of these supplemental images adds time and radiation exposure and may still be technically limited, despite maximum effort on the part of the x-ray technologist and the patient.

Given the differences in the incidence of injury to the cervical spine in children and the differences in their distribution (far more common in the upper cervical spine) relative to adults, an examination limited to frontal and lateral views is generally acceptable.[56] In infants 0 to 4 years of age and in children 5 to 9 years of age, AP, lateral, and open-mouth views are satisfactory. All patients age 10 and over require a minimum of three views to as many as five or six views to adequately survey the cervical spine. Clinical decision rules developed for adults cannot be reliably applied to children.

Films of the thoracic and lumbar spine are usually obtained as separate sets of frontal and lateral projections. The upper thoracic spine may require the addition of a swimmer's lateral view, if one has not been previously obtained as part of a cervical spine series. In general, if pathology is identified, then further evaluation with CT is indicated to further define the fracture pattern and to identify potential adjacent spinal injuries.

Technically inadequate examinations of the cervical or thoracic spine may be due to lack of visualization of the cervicothoracic junction or overlying arms due to upper extremity injuries. It is necessary to see the top of T1 on the cervical spine and the bottom of C7 on radiographs of the thoracic spine. A careful count of the vertebral bodies on the frontal examination to establish the correct levels is recommended to avoid wrong level surgery and is based on the number of rib-bearing (thoracic) and non–rib-bearing (lumbar) vertebrae. On the lateral images, it is important to look at the corners of the vertebral bodies, especially the anterior superior corner, which is affected in approximately 90% of all vertebral body fractures. On the frontal images, it is most important to evaluate the adjacent end plates for continuity, the lateral margins of the vertebrae, the posterior elements for pathologic interspinous and interpedicular widening, and horizontal lucencies that would suggest horizontal soft tissue and/or osseous disruption of a flexion-distraction–type injury.

One of the common errors made in evaluating the cervical spine is mistaking developmental variations for pathology. Common variations at the craniocervical junction include the following: (1) fusion of C1 to the occiput (which may be partial or complete); (2) failure of fusion or development of the posterior elements of C1; (3) pseudospreading of C1 relative to C2, which may mimic Jefferson burst fractures (most common in the 0- to 4-year age range, but may be seen up through puberty); (4) pseudosubluxation of C2 on C3 in pediatric patients, which can be recognized as normal by a normal C1–C3 spinolaminar line; and (5) os odontoideum

(an anomalous bone that replaces all or part of the dens but is not attached to the C2 body).

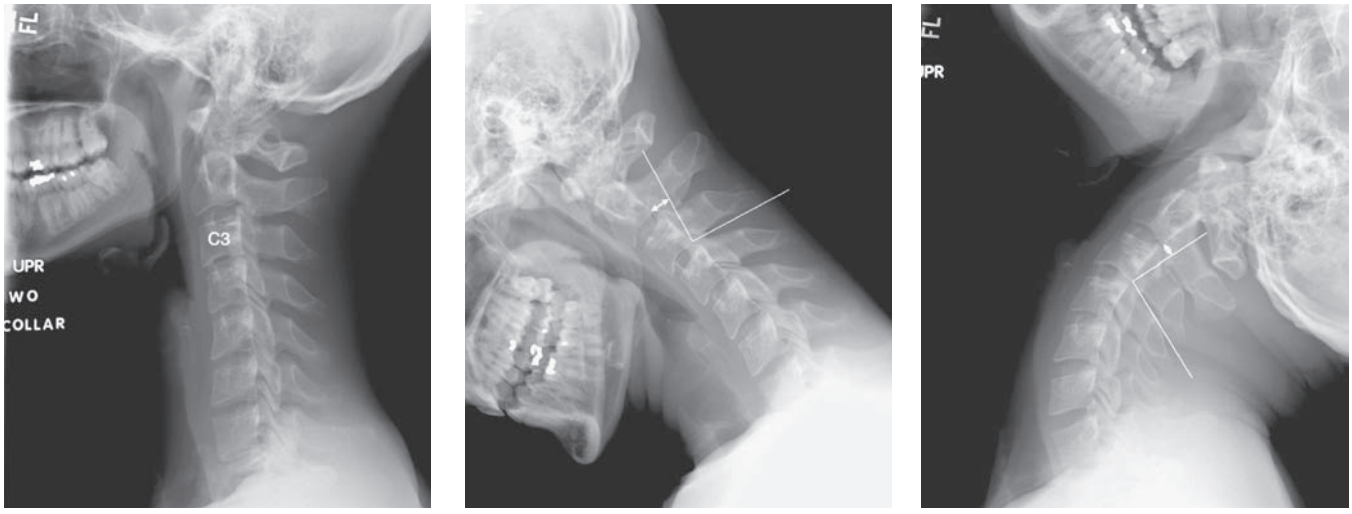
Flexion and Extension Cervical Spine Radiographs

Flexion and extension radiographs may be used to assess for potential ligamentous instability in patients who are completely alert and who have normal x-rays but exhibit posterior midline tenderness (Fig. 19-10). Some centers have recommended the use of passive (guided by the physician) flexion and extension studies using fluoroscopy, and although this may be appropriate in very limited circumstances in the hands of physicians with considerable experience, the published data are not sufficiently strong to warrant generalization.

A qualified physician should be in attendance if the examination is performed shortly after injury (hours to days), and the patient needs to be completely alert and able to assume an upright posture and precisely follow commands. An initial lateral radiograph is obtained with the patient upright and with the cervical spine in a neutral position. This image is reviewed by the physician overseeing the examination, and if normal, the patient is asked to actively extend the neck. An additional lateral image in maximum extension is obtained. The patient's cervical spine is then returned to a neutral position. This extension x-ray is evaluated in a manner similar to that taken with the cervical spine in a neutral position. If normal, the examination is repeated with maximum effort at flexion, and an x-ray is obtained. Standards for an adequate examination vary from a range of motion of 30° to 90°. The test is intended to study the capacity of the spine to resist physiologic stresses, however, and the mean normal range of motion in adults is approximately 90°. If the flexion-extension x-rays are abnormal in the acute or subacute setting, MRI is pursued. If the examination does not show an adequate range of motion, the patient's spine is immobilized, and the examination is repeated in 2 weeks if the patient remains symptomatic. It should be noted that approximately one-third of flexion-extension x-rays exhibit suboptimal flexion and/or extension, thus limiting its utility.[57] As a result, some centers with available resources omit flexion-extension series and move straight to MRI.

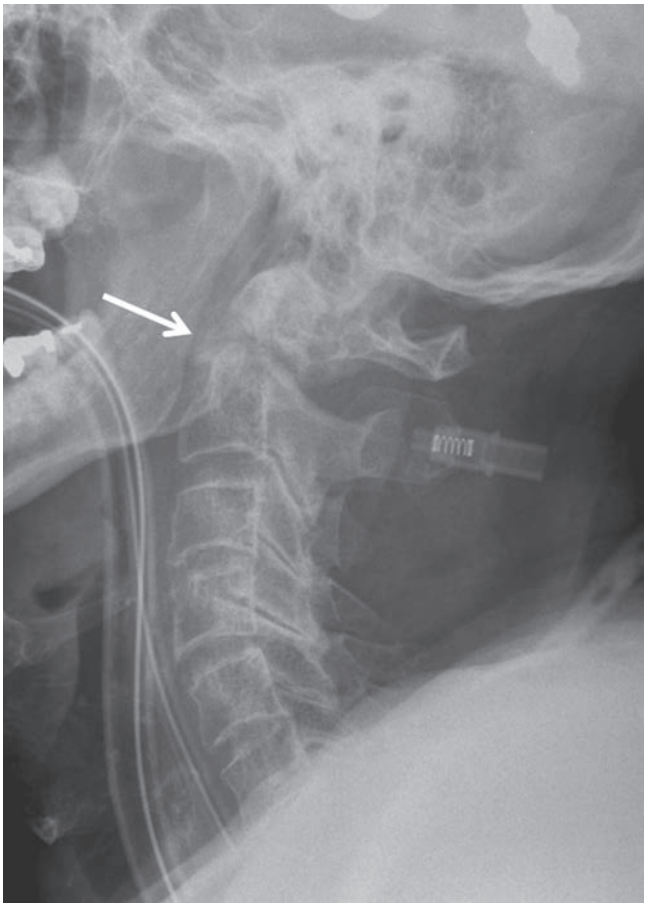
Computed Tomography of the Cervical Spine

Blackmore et al[58] developed a clinical prediction rule to determine preimaging risk of fracture in select patients about to undergo a helical CT to survey the cervical spine for fracture and soft tissue injuries (Figs. 19-11 and 19-12). For the application of this rule, it is assumed that the patient will already be undergoing CT of the cranium. Any one of three mechanisms of injury or any one of three clinical findings puts the patient at a pretest risk of greater than 5% of harboring an injury in the cervical spine. High-risk mechanisms of injury include a high-speed motor vehicle crash greater than 35 mph (or greater than 50 km/h), combined impact, motor vehicle crash with a death at the scene, or a fall from a height



A B C

FIGURE 19-10 Flexion-extension radiograph: cervical spine showing subtle instability at C2–C3. This 24-year-old male bicyclist was struck by a car from behind, and posterior midline tenderness of upper cervical spine was palpated. (**A**) Upright lateral out-of-collar radiograph shows loss of usual cervical lordosis without focal kyphosis or translation. Precervical soft tissues are normal. (**B**) Upright lateral flexion radiograph of cervical spine shows no gross interspinous widening or loss of parallelism of facet joint. Reference lines are drawn from posteroinferior corner of C3 to most inferior aspect of C3 spinous process. Perpendicular to that line from posteroinferior corner, a line is used as a reference for translation of C2 relative of C3, as demonstrated by double-arrowed line. (**C**) Upright lateral extension radiograph of cervical spine again shows no gross widening of anterior disk space. Using same reference for translation of C2 on C3, 2.5 mm of difference at C2–C3 disk space is demonstrated between flexion and extension, compatible with partial, dynamic instability.



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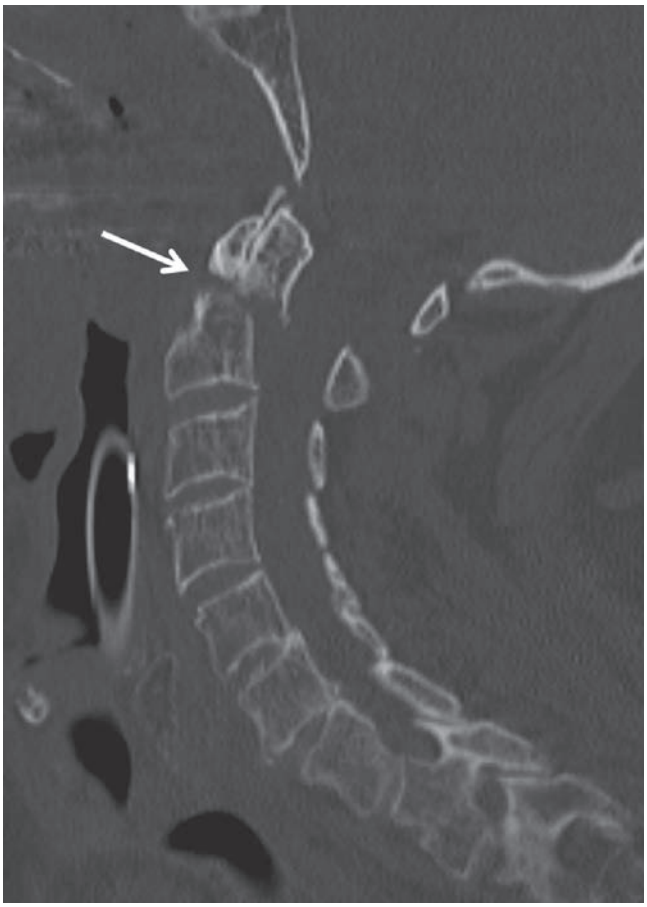

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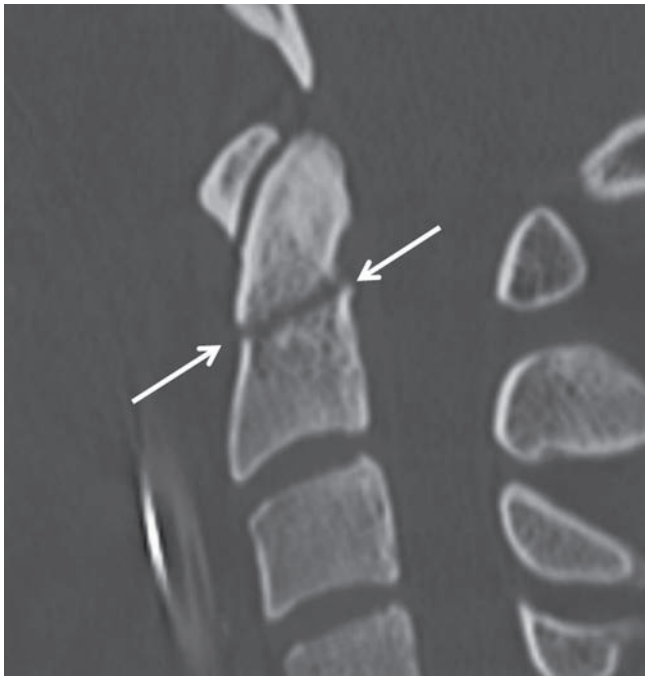

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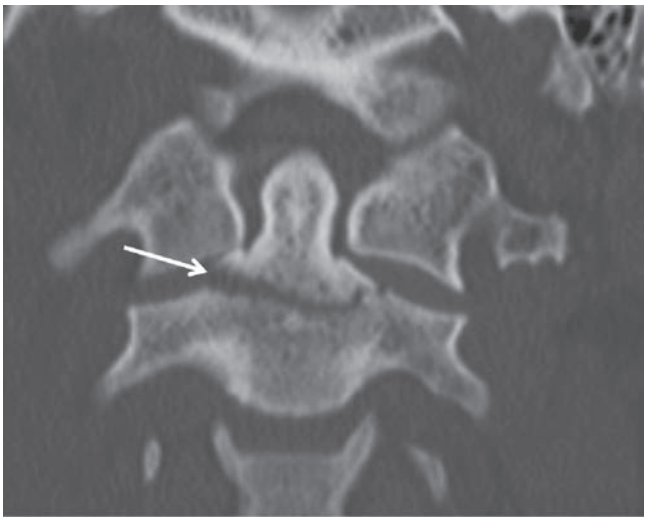

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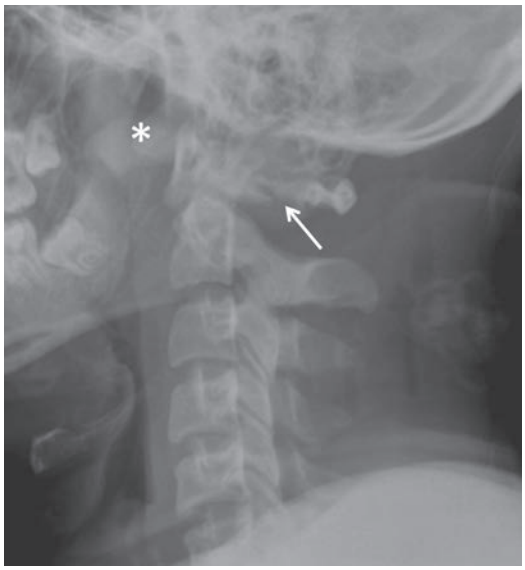
FIGURE 19-11 Upper cervical spine injuries. (**A, B**) Elderly patient status post fall. Lateral cervical spine image (**A**) demonstrates a fracture at the base of the odontoid process (arrow) compatible with a type II odontoid fracture. Sagittal (**B**) reformation nicely demonstrates the odontoid base fracture, which is displaced posteriorly with respect to the C2 body proper. (**C, D**) Another patient with a type III odontoid fracture. Sagittal (**C**) and coronal (**D**) reformations demonstrate a fracture of the base of the odontoid (arrows), which extends into the C2 body. (**E–G**) Another patient with a Jefferson burst fracture of C1 after falling while waterskiing.



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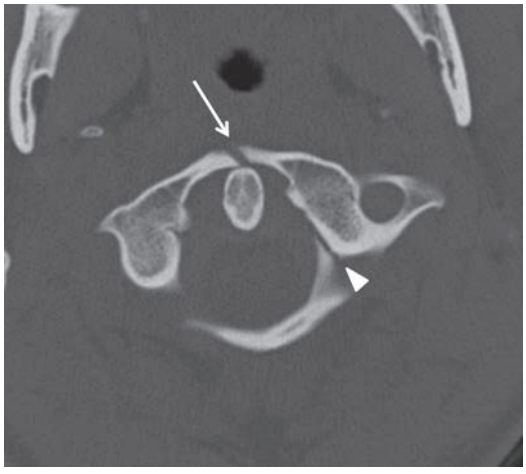

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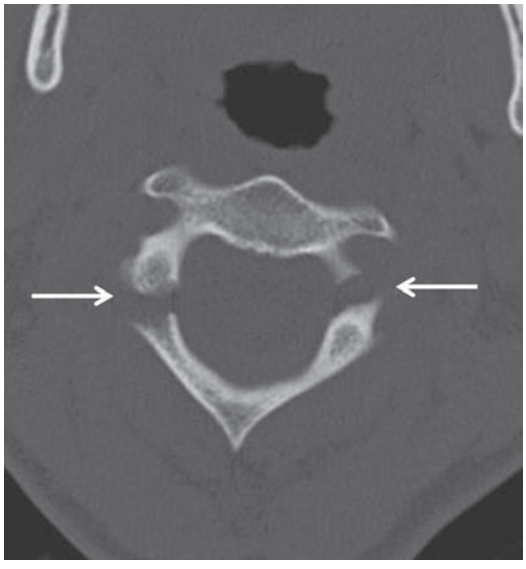

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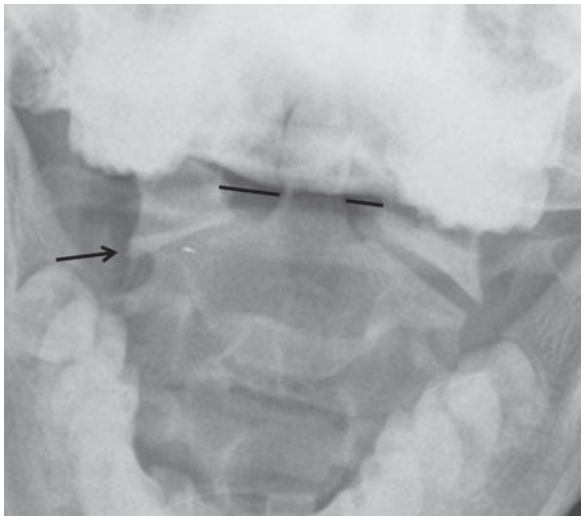

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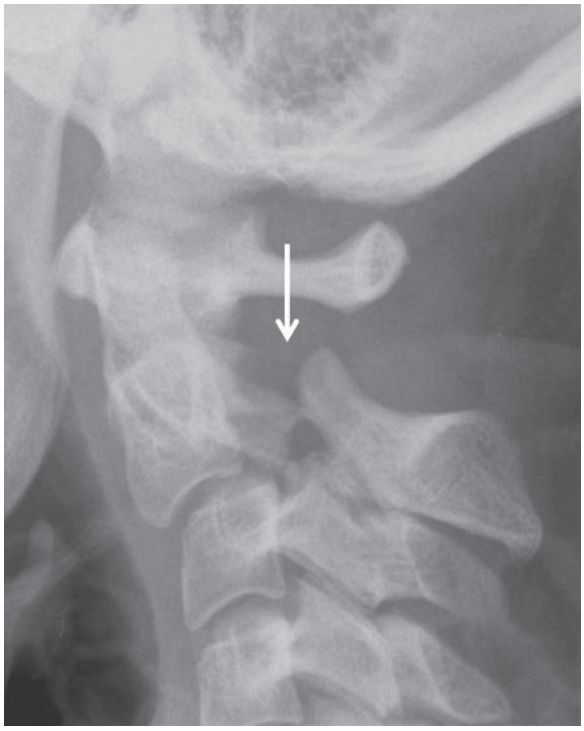

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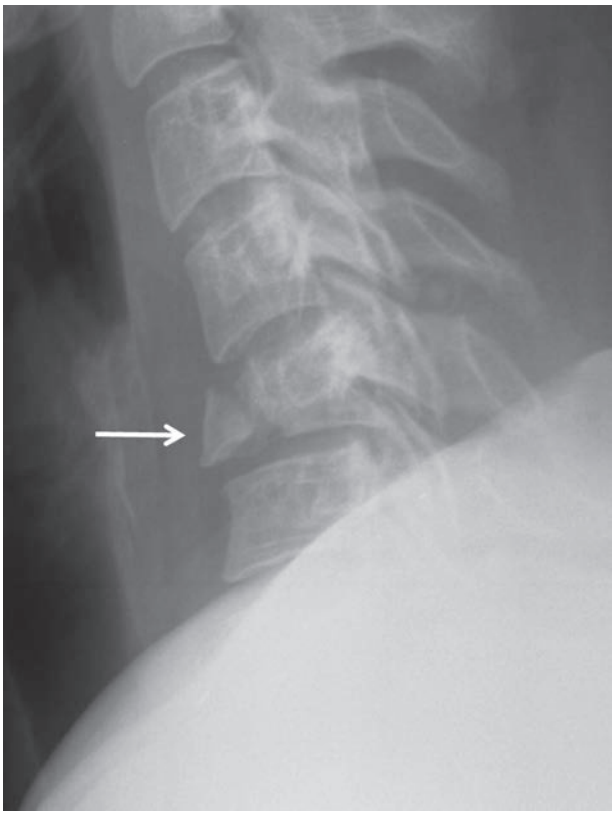
FIGURE 19-11 (*Continued*) Lateral radiograph (**E**) of the upper cervical spine demonstrates prevertebral soft tissue swelling (asterisk) at the skull base and a fracture of the C1 posterior ring (arrow). Open mouth radiograph (**F**) demonstrates asymmetry of the lateral dental interval (black lines) and a white band (black arrow) due to abnormal overlap of the C1 ring and the lateral mass of C2. Axial computed tomography (CT) image (**G**) demonstrates fractures of the anterior (arrow) and posterior (arrowhead) arches of C1 compatible with a Jefferson fracture. (**H, I**) Traumatic spondylolysis (Hangman fracture) following a high-speed motor vehicle collision. (**H**) Lateral cervical spine radiograph demonstrates bony defects (arrow) in the C2 pars. (**I**) Corresponding axial CT image demonstrates bilateral C2 pars fractures (white arrows).



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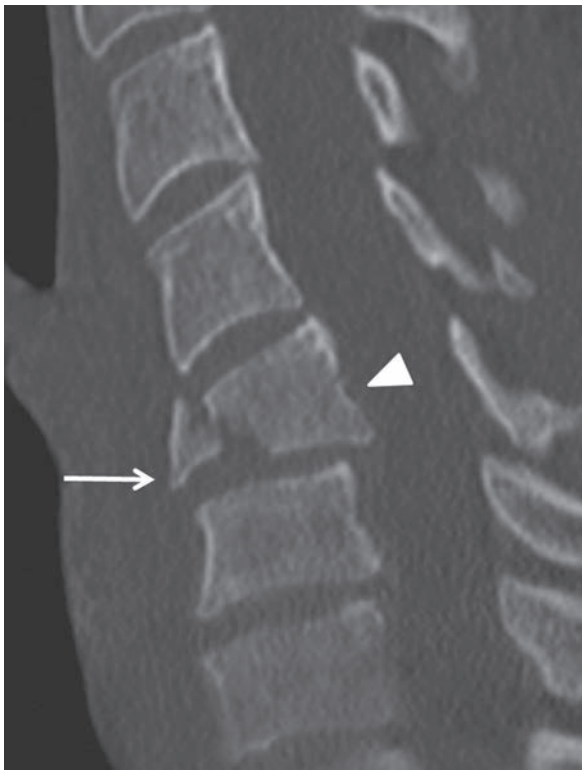

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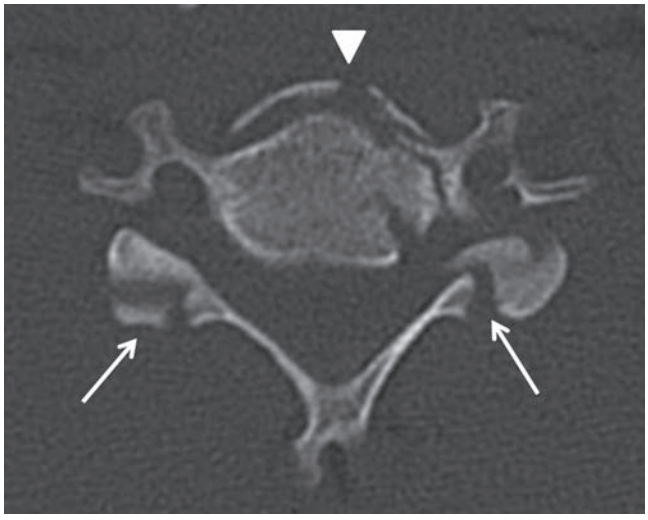

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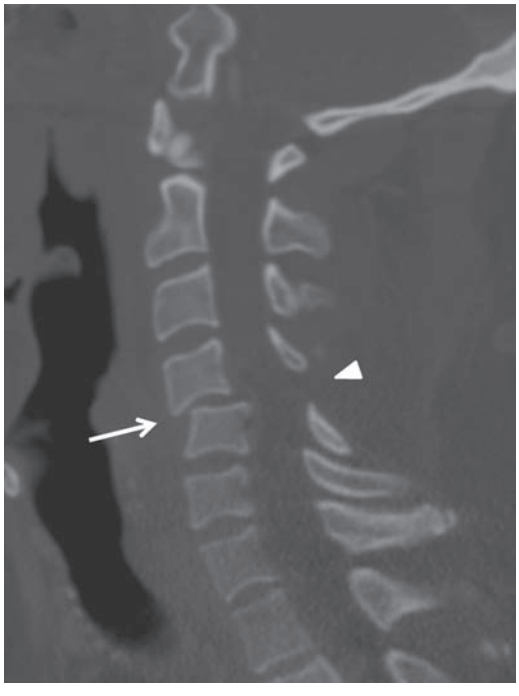

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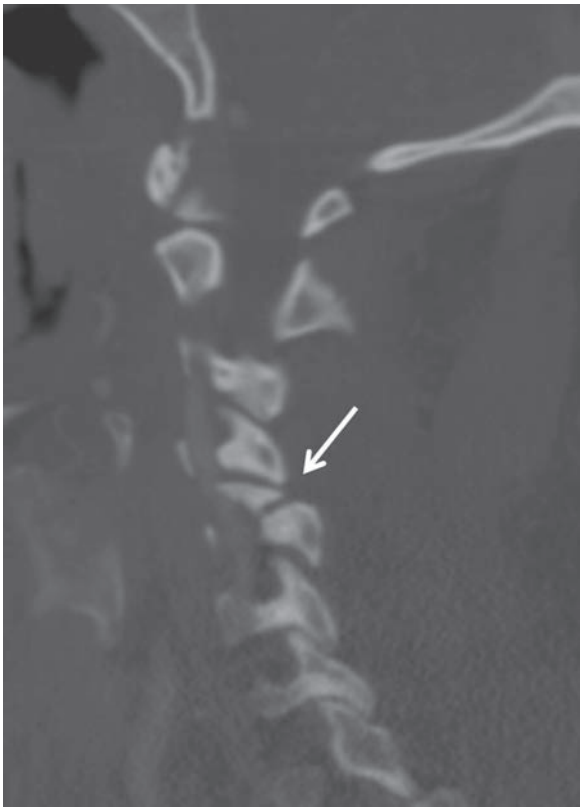
FIGURE 19-12 Mid and lower cervical spine fractures. (**A–C**) Cervical burst fracture. (**A**) Lateral cervical spine radiograph demonstrating the characteristic, triangular-shaped flexion "teardrop" fracture fragment (arrow) following a hyperflexion and axial load injury. (**B**) Midline sagittal computed tomography (CT) image of the same patient redemonstrates the teardrop fracture (arrow) and better illustrates retropulsion of bone into the spinal canal (arrowhead). (**C**) Axial CT image demonstrates a highly disorganized fracture, typical of this type of burst fracture. Bilateral lamina fractures (arrows) and comminuted vertebral body fracture (arrowhead) are better visualized on CT. (**D–G**) A 45-year-old male with neck pain and cerebellar symptoms following a fall from a mechanical bull. (**D**) Midline sagittal CT image view using bone windows demonstrates malalignment of C4 with respect to C5 (arrow) with corresponding widening of the interspinous distance (arrowhead).



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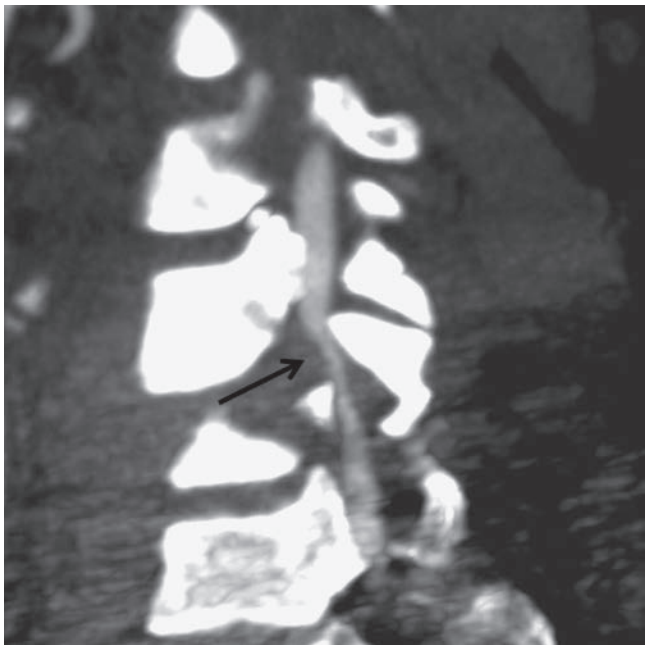

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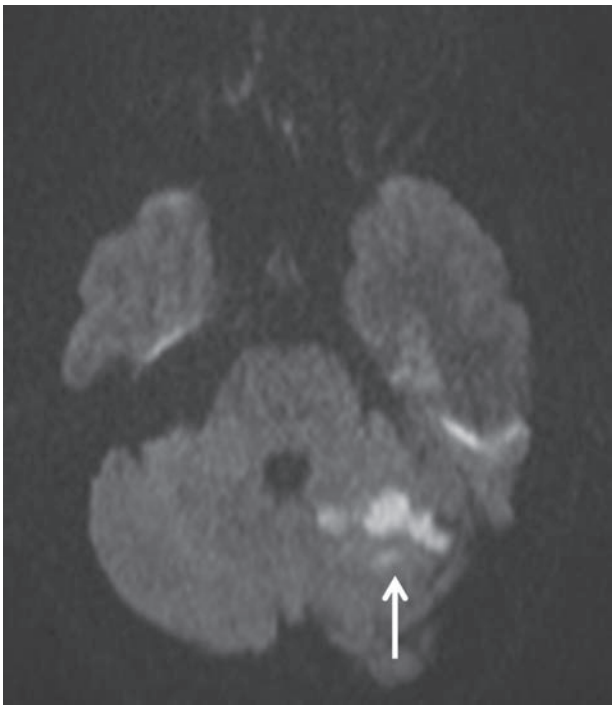

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FIGURE 19-12 (*Continued*) (**E**) Parasagittal CT image (bone windows) at the level of the facet joints reveals a C5 articular facet fracture (arrow). (**F**) Coronal oblique CT angiography image viewed using soft tissue windows demonstrates marked focal narrowing of the vertebral artery (black arrow) at the level of the facet fracture. (**G**) Axial diffusion-weighted magnetic resonance image demonstrates restricted diffusion (arrow) in the left cerebellar hemisphere compatible with acute cerebellar infarction.

of greater than 10 ft or greater than 3 m. Clinical parameters suggesting an increased risk for injury to the cervical spine that are associated with a high-risk mechanism include a significant closed injury to the brain (or intracranial hemorrhage shown on head CT), acute neurologic deficits referable to the cervical spine (acute myelopathy or radiculopathy), or either pelvic fracture or multiple extremity fractures. Hanson et al[59] validated the clinical prediction rule prospectively and showed its application by separating victims of blunt trauma into a high-risk group (12% prevalence of acute cervical spine injury) and a low-risk group (0.2% prevalence of cervical spine injury). No validated clinical decision rules exist for infants (<1 year old) and younger children (<9 years old). In general, patients with greater severities of injury (Injury Severity Score >25) have an elevated risk of injury to the cervical spine. For patients age 9 years or younger, conventional x-rays will depict essentially all clinically important fractures and dislocations. CT is generally not indicated in younger children and infants to screen the cervical spine or to search for other occult injuries causing neurologic deficits.[60] CT should be reserved as a staging/treatment planning procedure among patients with a known bony abnormality.

Modern helical MDCT scanners allow for high-resolution imaging of the cervical spine, with the quality of sagittal and coronal images equating to that of the axial images. Axial slices of the cervical spine are obtained at 1.0- to 3.0-mm slice thickness from the skull base through the upper thoracic spine (T2–T4 vertebral body level). Coronal and sagittal reformations are typically 1.0- to 3.0-mm-thick images and reconstructed using bone and soft tissue algorithms. Generally, the information gathered from these reformations is sufficient and makes plain x-rays unnecessary. PACS workstations can facilitate the rapid review of these large image sets, and the use of cross-referencing tools assists with identification of specific vertebral levels.

Careful review of the entire cervical spine in all three planes is necessary for comprehensive evaluation. Some of the more common fracture patterns (eg, type II fractures of the base of the odontoid process or horizontally oriented fractures of the spinous process) have "in plane" axial fracture lines and thus may be difficult to identify when reviewing the axial images alone. The coronal reformations may be viewed using the same approach as standard AP x-rays, whereas viewing of the sagittal reformations can be performed with guidelines used for the lateral cervical spine. Nonetheless, careful attention to axial images is necessary to detect fractures involving the craniocervical junction, transverse processes (which increase the likelihood of a vertebral artery injury), margins of vertebral bodies, pedicles, lateral mass, or lamina and spinous processes.

The sensitivity and specificity of cervical spine CT for acute bony injuries are routinely in excess of 95%. Although CT does not directly show soft tissue injuries to the spine, focal kyphosis, focal lordosis, and widening of the disk space can be used as with conventional x-rays to suggest associated injuries to soft tissue. Occasionally, acute disk herniations can be identified on axial or sagittal soft tissue images. Some authors feel that clinically important injuries to soft tissue causing biomechanical instability are almost always evident on technically adequate CTs of the cervical spine (especially on the sagittal and parasagittal reformations).[61]

Computed Tomography of the Thoracolumbar Spine

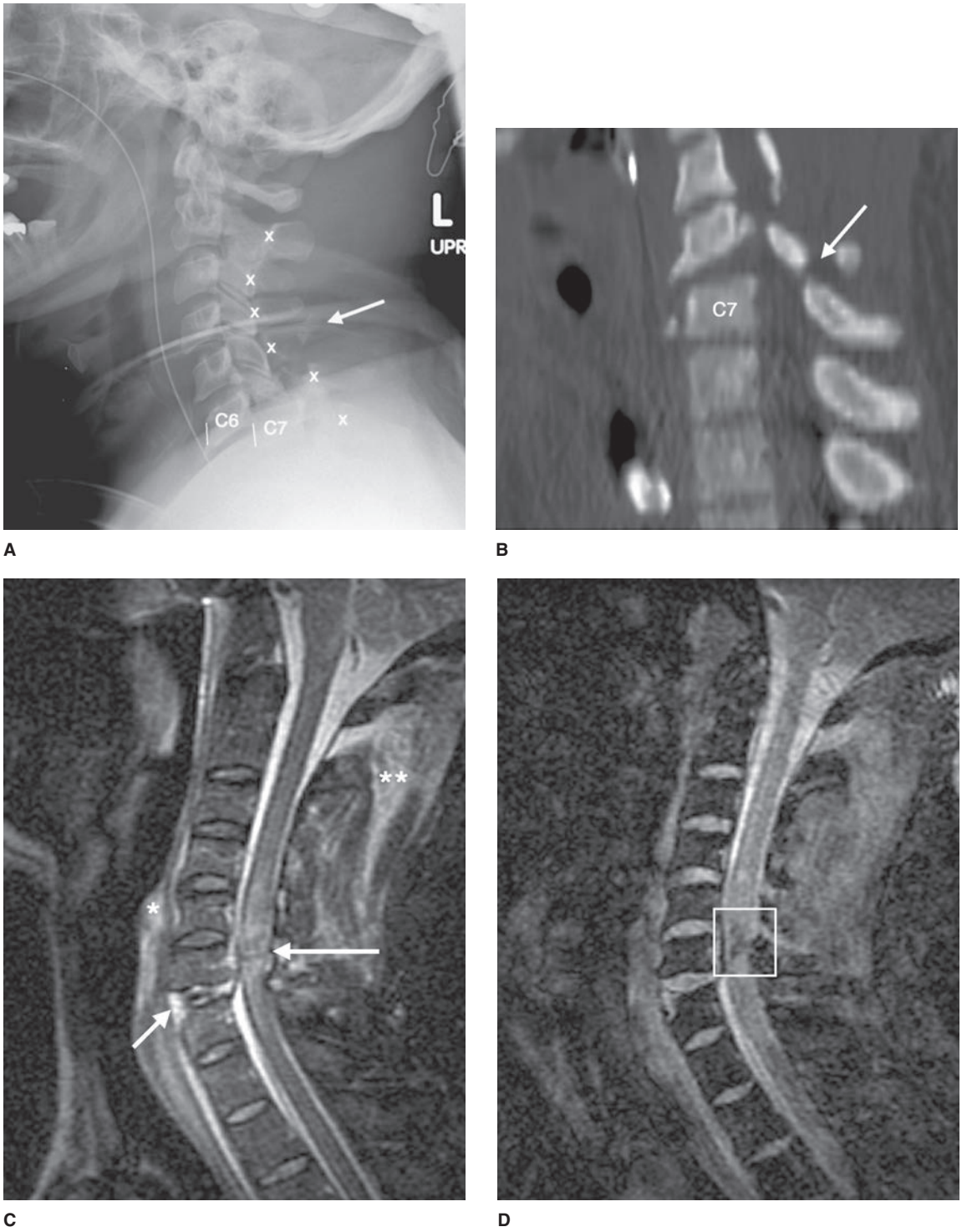
A low threshold should be maintained for the use of CT to further evaluate vertebral body deformities identified on conventional x-rays. In addition, patients with high-risk mechanisms and impressive signs or symptoms without abnormal x-rays should undergo a thoracolumbar CT to detect occult minimal burst fractures or Chance or flexion-distraction–type injuries. Dedicated radiographs of the thoracolumbar spine can be omitted if a CT of the chest and/or abdomen is obtained. The CT raw data of the chest and/or abdomen can be used to generate dedicated coned down images of the thoracolumbar spine, reconstructed in axial, sagittal, and coronal planes using a bone algorithm, without the need to reimage the patient. Limited imaging of specific vertebral body levels based on radiographic findings is being replaced by more comprehensive spinal imaging.

Typically, 2.0- to 3.0-mm-thick axial slices are acquired and are reconstructed using both bone and soft tissue algorithms. Imaging of the entire thoracic or lumbar spine is advised as it allows more accurate determination of the location of injury. Sagittal reformations are made in both algorithms and viewed at bone and soft tissue windows, respectively. Careful attention to the paraspinal structures, such as the ribs, mediastinum, and retroperitoneum, is strongly recommended if a flexion-distraction or Chance fracture is detected on dedicated thoracolumbar CT imaging. This diagnostic pitfall is becoming less of a factor due to the increased use of contrast-enhanced whole-body CT imaging in trauma.

Magnetic Resonance Imaging of the Spine

The principle indications for MRI are to characterize soft tissue injuries and potential spinal cord injuries in patients with associated fractures and/or malalignment of vertebrae (Fig. 19-13). An MRI is indicated in individuals who have no conventional x-rays or CT abnormality but who have an acute myelopathy or radiculopathy (spinal cord injury without radiographic abnormality [SCIWORA]). The use of MRI is frequently controversial in the setting of dislocation of bilateral or unilateral facets. Some advocate initial reduction followed by MRI, but this is a practice that varies from institution to institution. In general, urgent MRI is appropriate when there is an evolving neurologic deficit or neurologic deficits without explanation.

Edema of the spinal cord has a much better prognosis than cord hemorrhage. MRI is helpful in making this distinction, as well as detecting epidural hematomas that may require decompression. Evaluations of the disk spaces, ligaments of



A B C D

FIGURE 19-13 Multimodality correlations: cervical spine. A patient with C6–C7 fracture dislocation underwent prereduction lateral conventional radiograph (**A**), postreduction sagittal reformation (**B**) from axial computed tomography (CT), and magnetic resonance imaging (MRI) of the spine and cord (**C**) and (**D**). (**A**) White Xs mark laminar points, and connecting them shows disruption of spinolaminar line at C6–C7. Arrow points to fractures of inferior articular process of C6. Sagittal CT reformation (**B**) shows improved alignment of vertebral bodies but persistent encroachment of neural canal from bony fragments from body and posterior elements (arrow). (**C, D**) Sagittal MRI performed using short tau inversion recovery (STIR) and gradient recalled echo (GRE) sequences following reduction of C6–C7 fracture dislocation. Single asterisk shows precervical edema, and double asterisk shows edema in posterior spinal musculature. Long white arrow shows a region of cord swelling with heterogeneous signal, suggesting cord transection at C6 level. Short arrow shows abnormal signal within C6–C7 disk space. GRE sequences (**D**) show decreased signal at C6 level within the center of the cord that is compatible with hemorrhage (white square), which portends poorer neurologic prognosis than edema alone.

the spinal column and facet joints, and the craniocervical junction are best made with MRI.

Spine MRI imaging protocols in trauma vary among institutions, but sagittal and axial T1-weighted and fluid-sensitive sequences (eg, T2-weighted short tau inversion recovery [STIR]) are standard. Gradient echo (GRE) sequences are useful in detecting magnetic susceptibility artifact due to blood products. When assessing the transverse atlantal ligament, images should be obtained in the axial plane parallel to Ranawat's line (a line from the anteriormost portion of the anterior tubercle of C1 to the most posterior aspect of the posterior arch of C1).

MRIs have been used to "clear" the cervical spine in obtunded or unexaminable patients with otherwise normal imaging (CT or high-quality conventional radiographs).[62,63] The absence of abnormal high signal intensity in ligaments and disks effectively excludes biomechanically significant injuries; however, an abnormal signal does not necessarily confirm instability.

Computed Tomography of the Chest

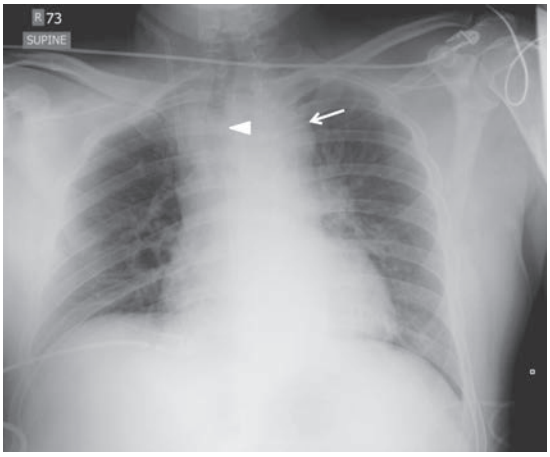
Chest CT has high sensitivity, specificity, and negative predictive value in the setting of trauma and can provide significant information about the lungs, pleural cavities, thoracic vasculature, and chest wall.[64] Chest CT is generally performed to evaluate adult victims of high-energy blunt trauma (especially those with chest pain, deformity, or hypoxia), with particular attention to the mediastinal structures (Figs. 19-14 to 19-18). Children presenting with hypotension, elevated respiratory rate, abnormal physical examination, depressed consciousness, and femur fractures after blunt trauma are at a substantially increased risk for an intrathoracic injury.[65]

Chest CT is performed with intravenous contrast, preferably with the patient's arms abducted above the head, from the thoracic inlet to the mid abdomen to include the entire thoracic cavity. Continuing the scan through the abdomen and pelvis can be easily performed. Images are acquired during the systemic arterial phase to maximize aortic enhancement and are reconstructed in the axial, sagittal, and coronal planes at 2.0- to 4.0-mm slice thickness. MPRs are used to evaluate not only the thoracic aorta and mediastinum, but also the thoracic spine (see earlier discussion). Tailored MPRs, such as sagittal oblique ("candy cane" view of the aorta), may be helpful in visualization of an aortic injury. Noncontrast chest imaging has limited utility in the setting of trauma, although mediastinal blood, hemopneumothoraces, and fractures to the spine and ribs may be detected on noncontrast imaging. In patients unable to receive contrast due to a known severe allergy, noncontrast CT can be effective in detecting a mediastinal hematoma and guiding the patient to evaluations such as transesophageal echocardiography and MRA. In patients with renal insufficiency, the risk of contrast-induced nephropathy or worsening renal function should be weighed against the risk of missing a major thoracic injury.

Chest CT is believed to be most cost-effective when patients: (1) are already undergoing another CT examination (eg, CT of the head, abdomen, and pelvis); (2) are at risk for injury to the thoracic aorta because of high-energy mechanism, associated injuries, or age (>50 years old); or (3) have a previously abnormal chest radiograph. A clinical decision rule was proposed by Blackmore et al,[66] in which individuals with two or more of the following are at high risk for aortic injury: age greater than 50, unrestrained occupant in motor vehicle crash, hypotension, thoracic injury (rib fracture, pneumothorax, pulmonary contusion, or laceration), abdominopelvic injury (fracture of lumbar spine or pelvic ring, injury requiring laparotomy), fractures of appendicular skeleton, or injury to the brain.

The role of MDCT in the diagnosis of acute traumatic aortic injury has undergone significant evolution over the past 25 years, with sensitivity and negative predictive value of contrast-enhanced chest CT routinely in excess of 98% for modern scanners. In fact, patients who have direct signs of a thoracic aortic injury on MDCT no longer require catheter angiography confirmation, as was often the case in the past, and a normal thoracic aorta on chest CTA reliably excludes a traumatic aortic injury.[67] CT imaging features of a traumatic aortic injury can be divided into direct and indirect findings. Direct findings of traumatic aortic injuries include pseudoaneurysms, intimal flaps, pseudocoarctation (due to subadventitial dissection), and active bleeding. A mediastinal hematoma, however, is an indirect finding and can be present in the absence of an aortic injury. To suggest an aortic injury, a mediastinal hematoma should be contiguous with the aortic wall and should not be separated from the aorta by a rim of fat (Fig. 19-17D). Determination of whether or not a mediastinal hematoma has obliterated juxta-aortic fat can be difficult in thin patients or in patients with extensive soft tissue edema. Complex atheromatous disease can make interpretation of the examination difficult, particularly for subtler injuries. An emerging form of an acute aortic injury, called the "minimal" or "minor" aortic injury, is an injury pattern thought to be secondary to advancing imaging technology (Fig. 19-19).[68,69]

There is significant debate regarding what lesion is defined as a "minimal" aortic injury. More specifically, they may include an intimal injury with thrombus, intimal flap less than 1 cm in length, and pseudoaneurysm less than 1 cm in diameter. There is often very little or no mediastinal hemorrhage with this type of injury.[68,69] There are no universally accepted imaging follow-up recommendations for patients with minimal aortic injuries, although serial short interval follow-up to resolution has been advocated.[69] Finally, aortic pulsation artifact (particularly involving the aortic root and ascending aorta) and beam hardening artifact due to dense contrast in adjacent venous structures may make interpretation difficult. Further evaluation with catheter angiography can be performed in equivocal or suboptimal examinations but is of low diagnostic value in the setting of a high-quality diagnostic chest CT angiogram. Multiplanar and 3D reformations of the injured thoracic aorta are particularly helpful for planning treatment. It is important to delineate the anatomy of interest to the trauma surgeon, such as the distance



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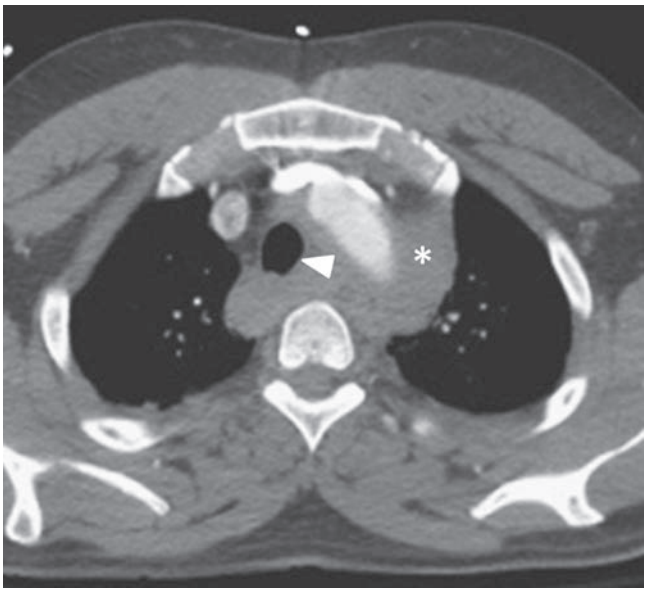

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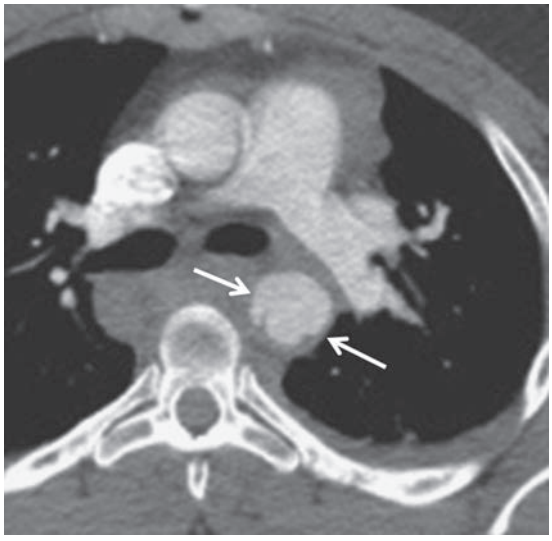

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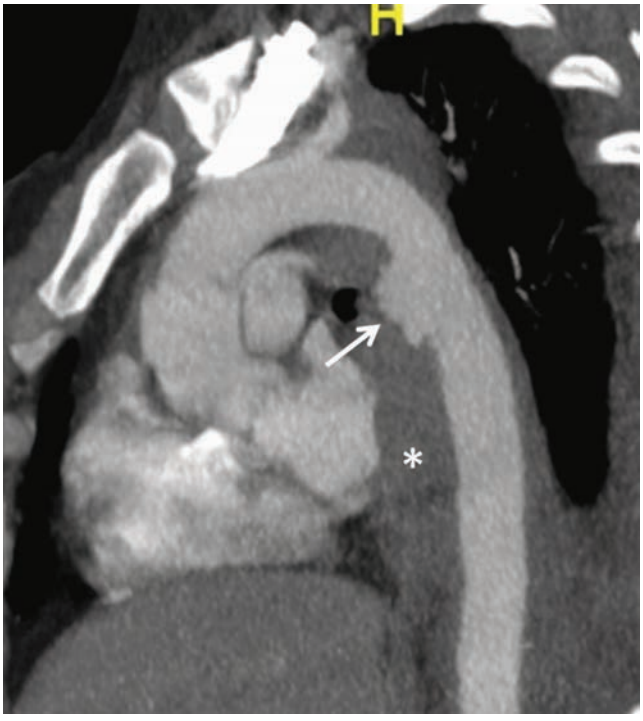

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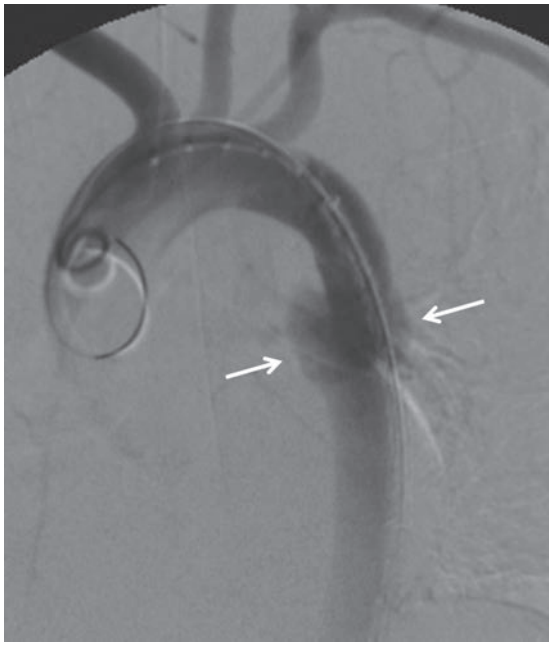

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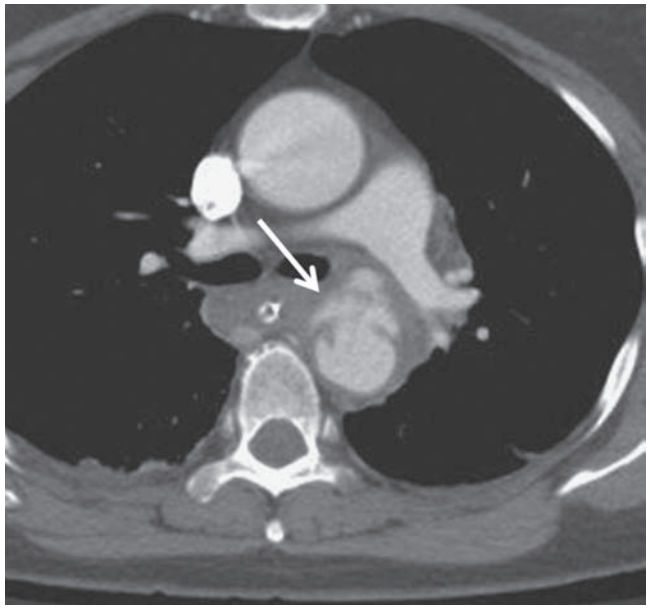
FIGURE 19-14 Blunt traumatic rupture of the thoracic aorta. (**A**) Anteroposterior supine chest radiograph demonstrates a bulbous aortic arch contour (arrow) and rightward deviation of the trachea (arrowhead), concerning for a mediastinal hematoma. (**B**) Axial computed tomography angiography (CTA) of the upper mediastinum demonstrates a superior mediastinal hematoma (asterisk) and rightward tracheal deviation (arrowhead). (**C, D**) Axial and sagittal CTA of the mid thorax demonstrates intimal flaps and abnormal outpouching of contrast along the anterior surface of the proximal descending thoracic aorta due to a traumatic pseudoaneurysm (arrows). There is adjacent mediastinal hematoma (asterisk). (**E**) Catheter aortography adds little information in this instance. Focal outpouching of the proximal descending thoracic aorta due to a traumatic pseudoaneurysm (arrows) is again identified.



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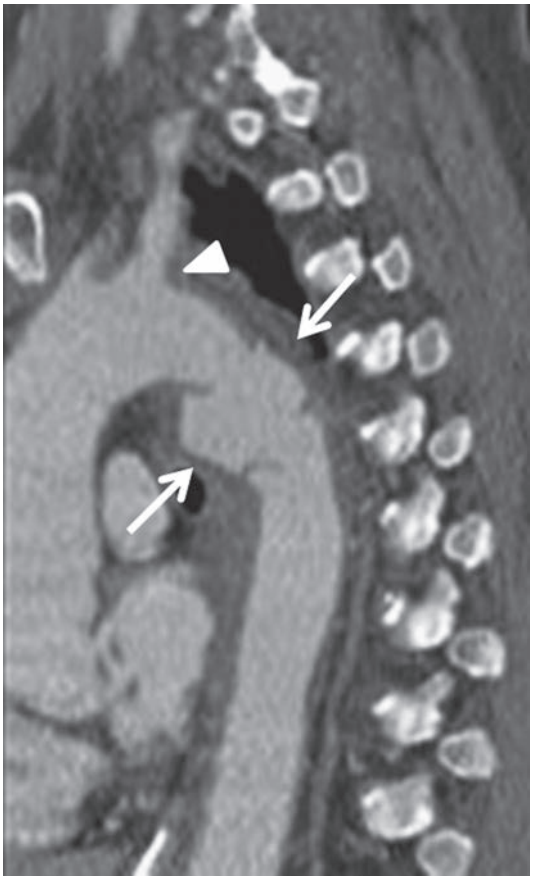

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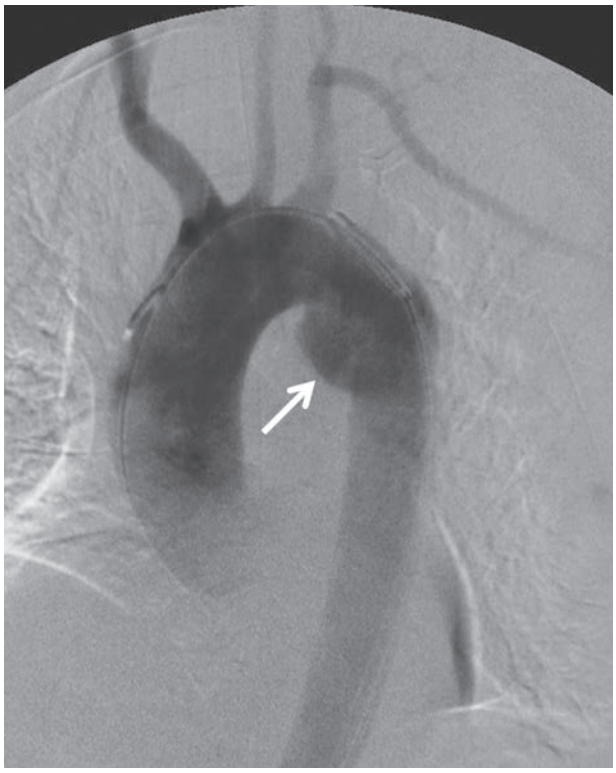

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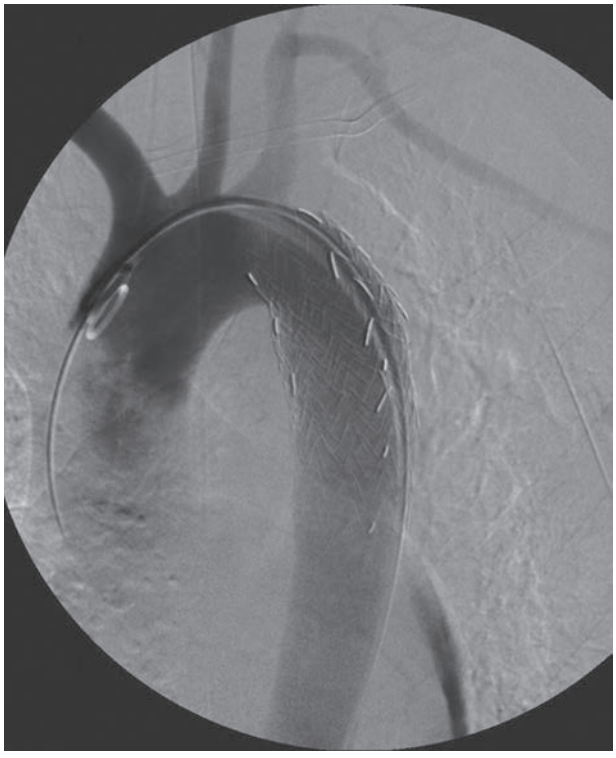

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FIGURE 19-15 Acute thoracic aortic injury following blunt chest trauma. Axial (**A**) and sagittal (**B**) chest computed tomography angiography shows a transection of the proximal descending thoracic aorta (arrows) just distal to the origin of the left subclavian artery (arrow head). (**C**) Catheter angiogram of the thoracic aorta showing the aortic injury (arrow), which was successfully treated with an endovascular stent graft (**D**).

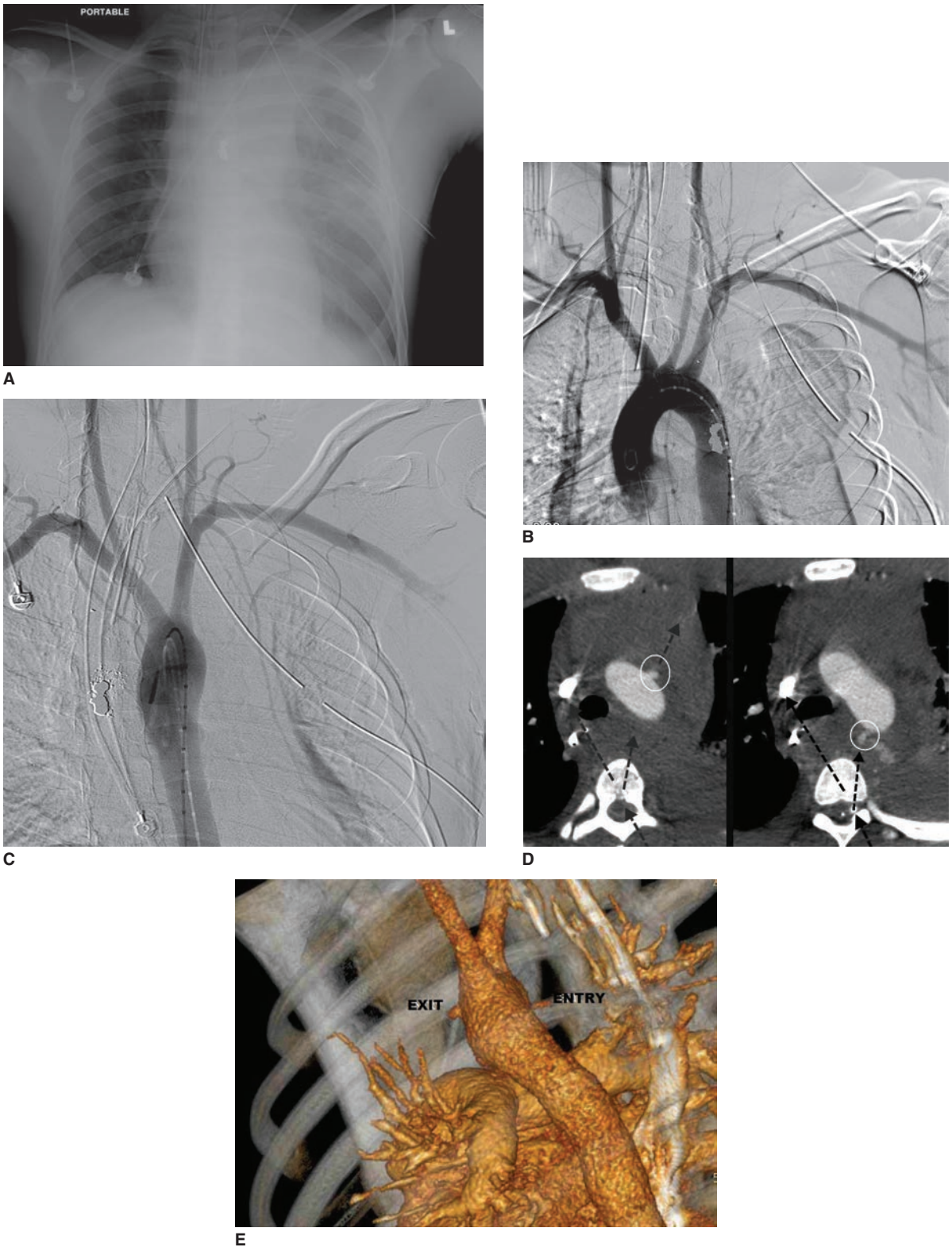
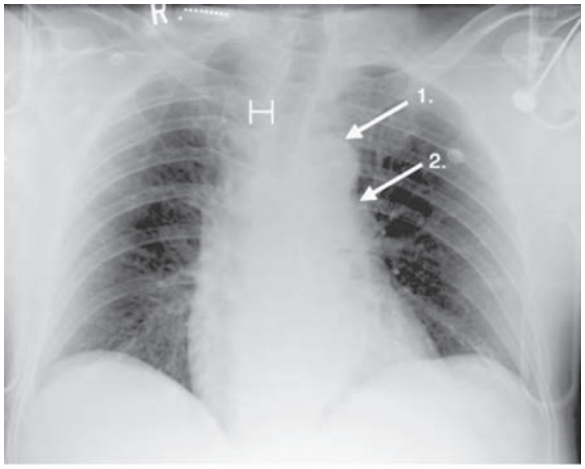


FIGURE 19-16 Gunshot injury of the aortic arch not detected on catheter aortography. A 24-year-old male sustained a gunshot wound of the back with mediastinal traverse. He was hemodynamically stable but paraplegic. (**A**) Portable chest radiography showed hemothorax and a very wide indistinct mediastinum with a bullet in the mediastinum. (**B, C**) Right and left anteroposterior catheter aortograms did not show any evidence of an injury. (**D**) However, suspicion of an aortic injury persisted. Therefore, a computed tomography arteriogram was performed. This shows that the bullet had traversed the spine and fragmented into parts that went to the right and the left (dashed arrows). The left fragments penetrated the posterior arch and exited the anterior arch of the aorta (arrows). (**E**) Surface-rendered reformations clearly show the path of the bullet.



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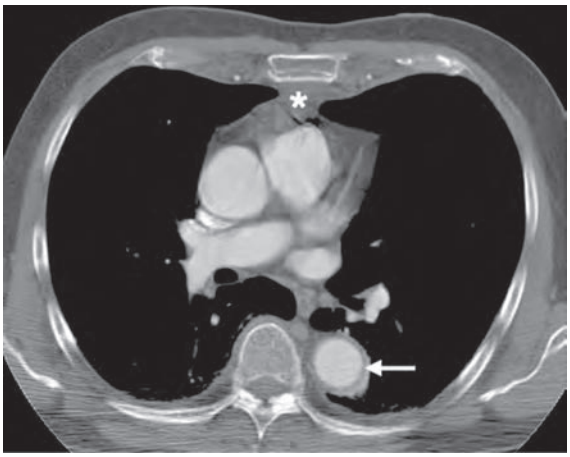

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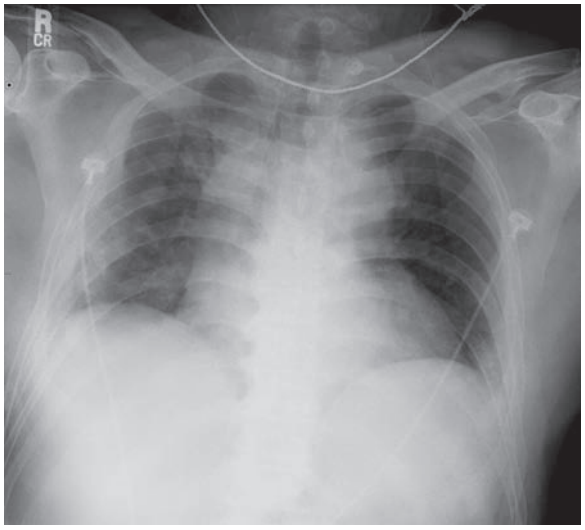

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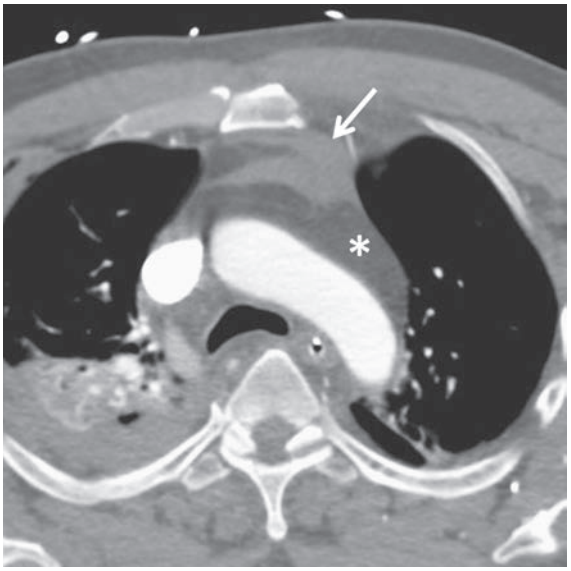

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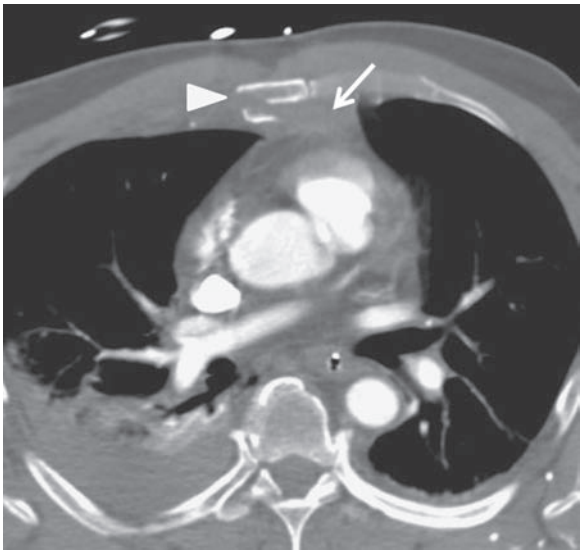

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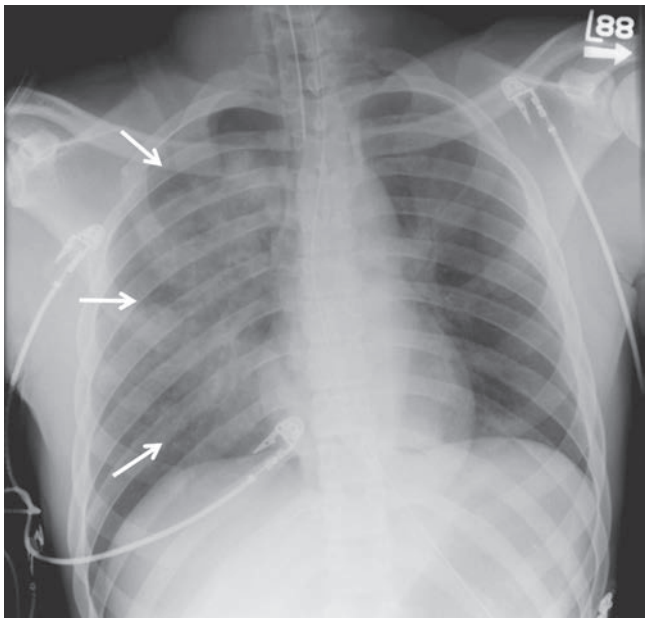
FIGURE 19-17 Mediastinal hematoma caused by nonaortic injury. (**A**) Anteroposterior (AP) chest radiograph performed on 19-year-old unrestrained driver in head-on motor vehicle crash shows multiple injuries, including T6 and left shoulder fractures. Widening of right paratracheal stripe (H), obscuration of aortic arch (black arrow), and abnormal right paraspinal line (white arrows) suggest mediastinal hematoma. In absence of osteophytes, right paratracheal stripes are not typically seen in young adults, and their presence locally should direct search for underlying pathology. Left paraspinal line is typically seen due to descending aorta and should not be seen as continuous line between the lower chest and the apex of lung. Continuous left paraspinal line from apex to diaphragm is pathognomonic for mediastinal collection, such as hematoma in setting of trauma. (**B**) Same patient as in **A**; axial computed tomography (CT) following intravenous contrast shows extensive posterior mediastinal hematoma (asterisks). (**C–E**) This is a 56-year-old male involved in a motor vehicle collision. (**C**) AP supine chest radiograph demonstrates a widened mediastinum with abnormal and ill-defined aortic contours, concerning for a mediastinal hematoma. (**D**) Axial postcontrast CT through the upper mediastinum demonstrates retrosternal hematoma (arrow) with a clean fat plane between the hematoma and aorta (asterisk) and no periaortic hemorrhage. (**E**) Axial postcontrast CT through the mid chest demonstrates a fracture of the sternal body (arrowhead) as a source of the mediastinal hematoma (arrow). The aorta was not injured.



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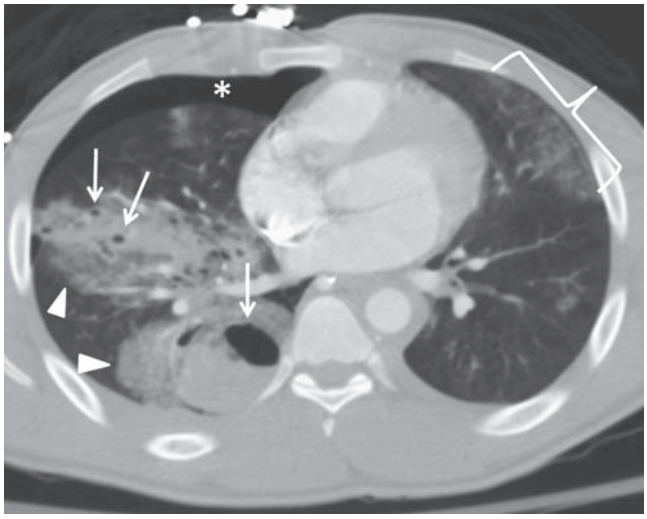

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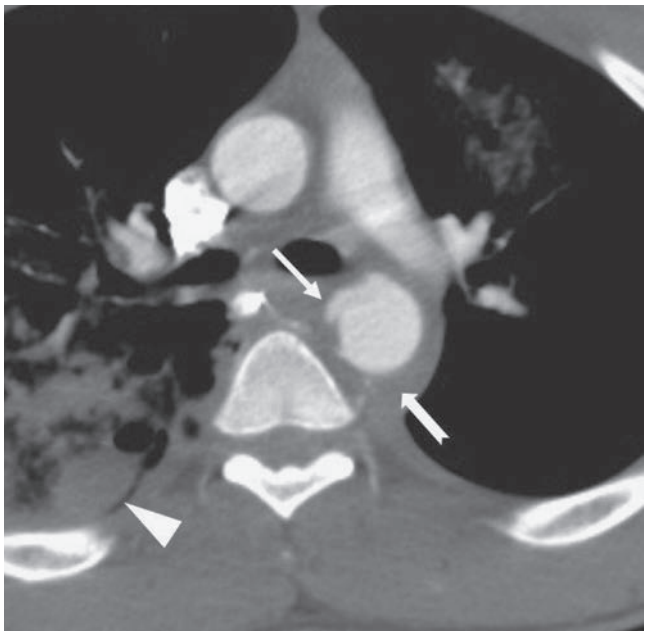

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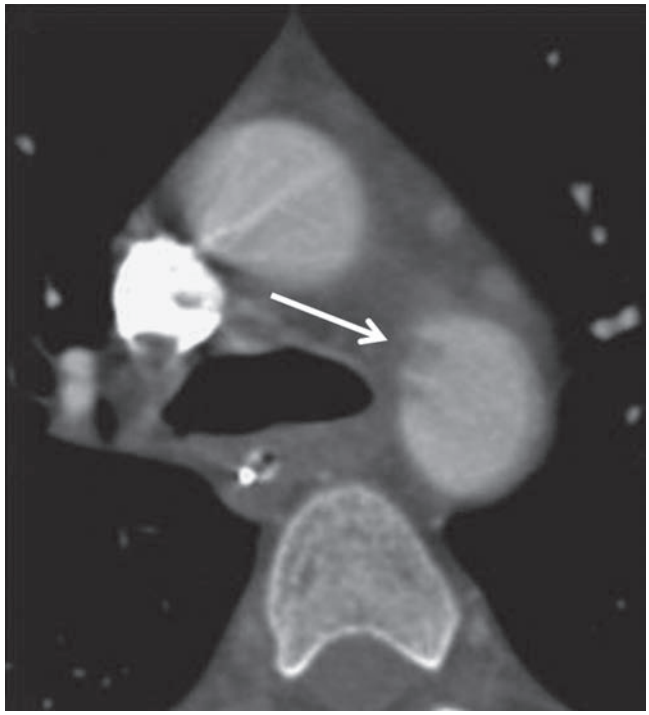
FIGURE 19-18 Pulmonary contusions and lacerations due to blunt-force injury. (**A**) Anteroposterior (AP) supine chest radiograph of a patient involved in a T-bone motor vehicle collision shows extensive right perihilar distribution airspace opacities (arrows). The left lung is clear. (**B**) Axial postcontrast computed tomography (CT) image reveals a right pneumothorax (asterisk, which was radiographically occult) and multiple pulmonary parenchymal lacerations (arrows) of various sizes, the larger of which demonstrates a layering blood fluid level. Ground glass opacities surrounding these lacerations are due to pulmonary contusions (arrowheads). There are patchy peripheral ground glass opacities in the left upper lobe compatible with pulmonary contusions (bracket), which are better appreciated on CT. Pulmonary contusions are typically present by the time the patient presents to the hospital and may evolve for 48 to 72 hours. Progression thereafter should be considered a complication, such as pneumonia or adult respiratory distress syndrome. Typically, pulmonary contusions resolve within 1 week. This example nicely illustrates that a supine chest radiograph often underestimates the severity of a chest injury. (**C**) Axial CT through the upper chest shows an aortic injury (arrow) and pulmonary contusions/lacerations (triangle) not as well seen on this soft tissue image.

from the most proximal point of injury to the origin of the left subclavian artery or any anomalous branches. These capabilities have significantly altered the role of catheter angiography from its traditional role of diagnosis, staging, and pretreatment planning, to one more often used for resolving diagnostic conundrums raised by CT or transesophageal echocardiography or as part of treatment using an endovascular stent graft.

CT is the most sensitive diagnostic method for detection of acute blood in the pericardium.[70] It is also among the most sensitive methods for detection of injuries to the chest wall, pleural cavities, and lungs.[64] Older CT technology exhibited relatively low sensitivity for the detection of injuries to the hemidiaphragm (sensitivity of 65%–70%) but has vastly improved with helical MDCT technology, now with sensitivities of 71% to 90% and specificities of 98% to 100%.[71] For suspected diaphragmatic injuries, especially herniations through a diaphragmatic tear, coronal and sagittal multiplanar reformations are useful because they better display characteristic findings (Fig. 19-20). Direct CT signs of diaphragmatic rupture include focal diaphragmatic defect, the "dangling" diaphragm sign (from a free-floating diaphragm flap), and



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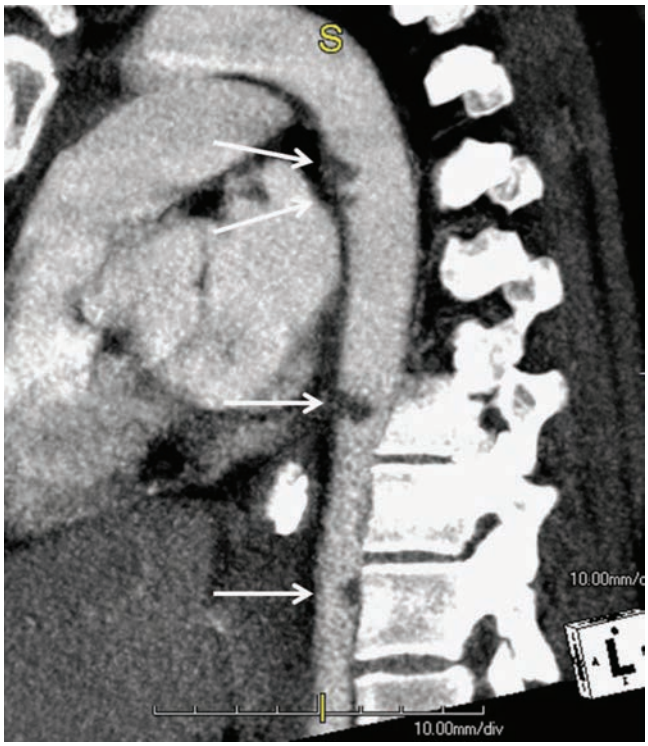

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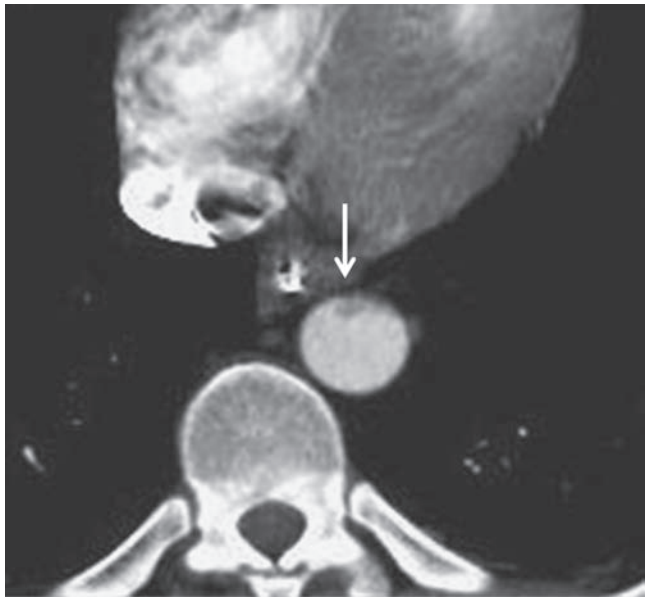

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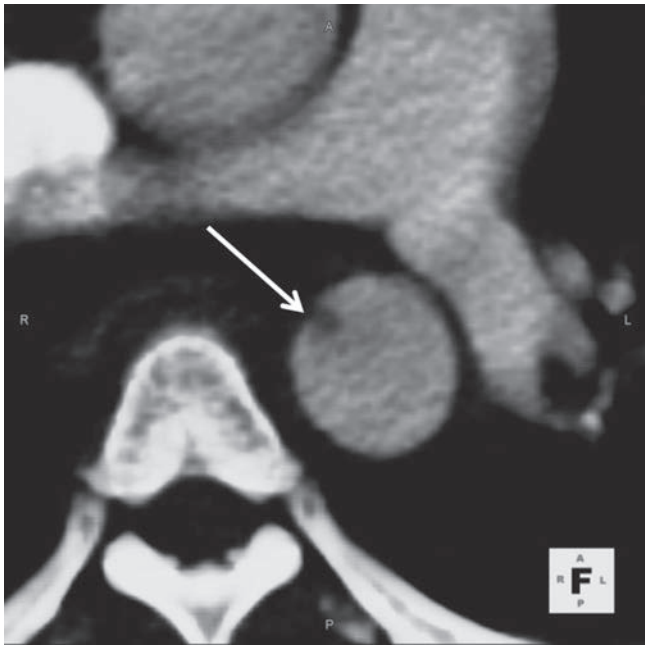

D

FIGURE 19-19 Minimal aortic injuries following blunt thoracic trauma. (**A–B**) Axial postcontrast images from two different patients each demonstrates intimal injuries with focal intraluminal thrombi (arrows) involving the aortic arch (**A**) and mid descending thoracic aorta (**B**). There is trace adjacent periaortic hemorrhage in **A** and no periaortic hemorrhage in **B**. (**C**) Sagittal postcontrast computed tomography (CT) of another patient with multiple intimal injuries of throughout the descending thoracic aorta (arrows) compatible with multifocal minimal aortic injuries. There is no periaortic hemorrhage. (**D, E**) A 42-year-old female with a minimal aortic injury of the mid descending thoracic aorta. Admission axial postcontrast image (**D**) demonstrates a focal intimal injury and thrombus of the mid descending thoracic aorta (arrow).

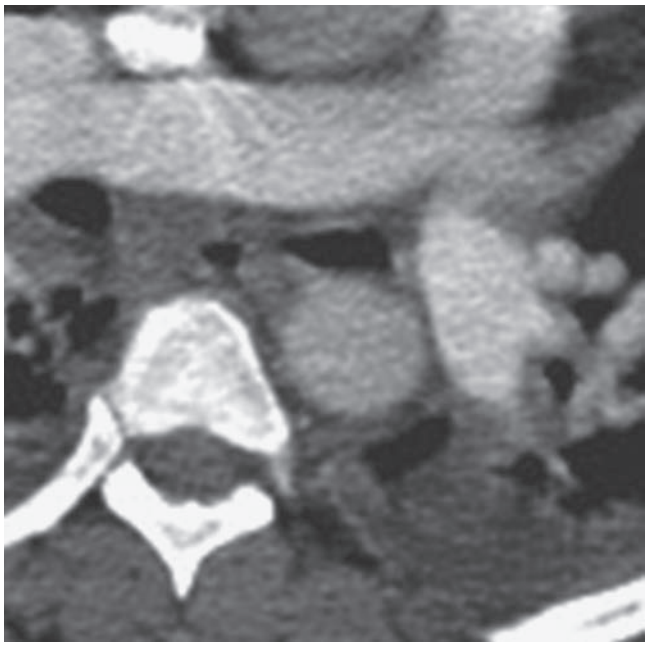

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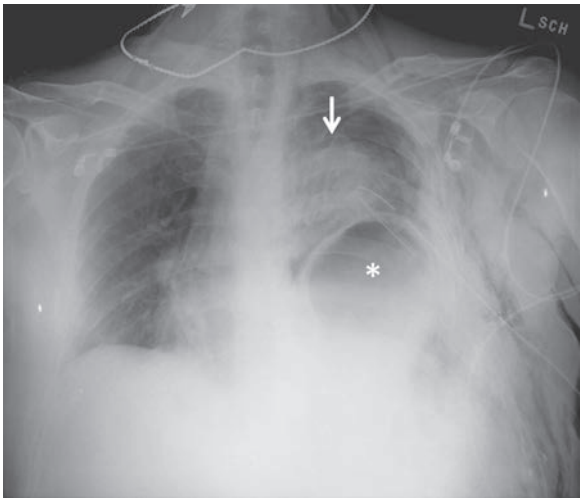
FIGURE 19-19 (*Continued*) (**E**) One-week follow-up chest CT reveals resolution of intimal injury and thrombus.



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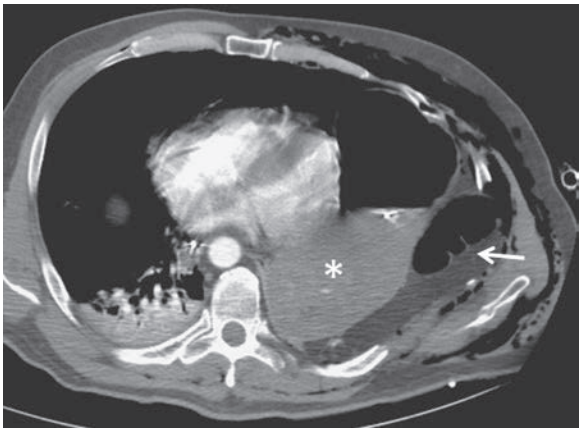

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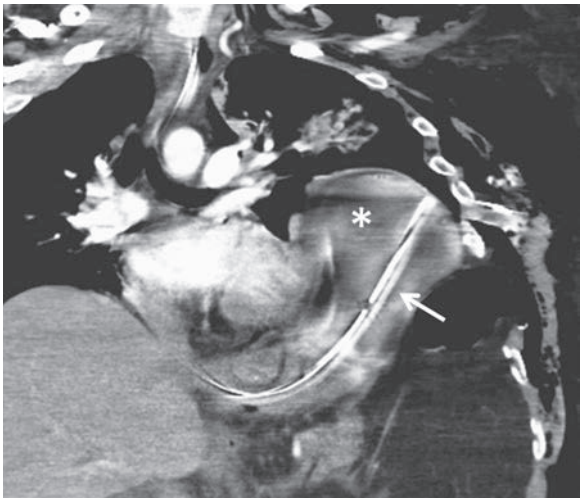

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FIGURE 19-20 A 60-year-old-male with a left diaphragm rupture following a high-speed motor vehicle collision. (**A**) Portable chest film on admission demonstrates superior displacement of the stomach (asterisk). There is a left upper lobe opacity (arrow) due to pulmonary contusion. (**B**) Axial computed tomography (CT) image through the lower chest demonstrates the stomach (asterisk) above the diaphragm next to the heart. There is herniation of abdominal fat and colon (arrow) into the left thoracic cavity. Notice that the stomach and abdominal fat are displaced posteriorly and are layering dependently against the posterior chest wall, referred to as the "dependent viscera sign," a specific sign of a diaphragm rupture. (**C**) Coronal CT image demonstrates the stomach (asterisk) herniated into the left thoracic cavity. There is a nasogastric tube in place (arrow).

herniation of abdominal contents through a diaphragmatic defect. A normal contour of the diaphragm and no pleural collections or adjacent airspace disease effectively exclude diaphragmatic injury.

CT is helpful for diagnosing injuries to the tracheobronchial tree and has a sensitivity of 85% for tracheal rupture.[30] Nearly all patients with tracheobronchial injuries exhibit a massive pneumomediastinum or soft tissue emphysema (Fig. 19-7A and B; Fig. 19-9D and E).

Computed Tomography of Abdomen and Pelvis

Abdominopelvic CT is one of many adjunctive tests to assist the trauma surgeon in the evaluation of hemodynamically stable patients with suspected occult intra-abdominal injuries or to aid in more definitive characterization of injuries previously detected by other diagnostic tests (eg, DPL or FAST; Figs. 19-21 to 19-27). Usual indications include abdominal signs (eg, lap belt sign) or symptoms (eg, pain and tenderness) following high-energy blunt trauma. The combination of pleuritic chest pain at the left costal margin and left lower rib fractures is an independent predictor of splenic injury and warrants diagnostic evaluation.[72] Abdominopelvic CT is extremely helpful in guiding management and has been shown to significantly decrease the number of negative or nontherapeutic laparotomies. There has been a recent increase in the utilization of "triple-contrast" abdominopelvic CT in the setting of penetrating trauma, in the absence of indications for immediate laparotomy (eg, peritonitis or hemodynamic instability).[73] This is particularly true for back and flank penetrating wounds.

It is estimated that clinical signs and symptoms of intra-abdominal injuries may be misleading in 20% to 50% of patients, that physical examination has a 55% to 65% sensitivity for the detection of internal injuries, and that up to 19% of patients with blunt abdominal trauma have unsuspected injuries.[9,74,75] This, coupled with a negative predictive value of greater than 97% for abdominopelvic CT, has resulted in overall increased utilization of CT in the setting of blunt abdominal trauma. In addition, abdominopelvic CT is the principle means of both detection and characterization of renal injuries among adults with gross hematuria, children with microscopic hematuria (>50 red blood cells per high-power field), or microscopic hematuria among adults who have had one or more episodes of systolic hypotension.

CT of the abdomen and pelvis acquired during the portal venous phase (60- to 75-second scan delay) has been shown to have a high sensitivity, specificity, and negative predictive value for the diagnosis, grading, and exclusion of solid organ injuries. The addition of an arterial phase aids not only in the detection of arterial injuries, but also in the detection of pseudoaneurysms in solid organs, which may not be visible on portal venous phase images (Fig. 19-26).[76,77]

Portal venous or delayed phase imaging can also assist in the detection of active bleeding from the organs or pelvis. CT is accurate in the detection of urinary leaks from the upper genitourinary tract, and delayed excretory phase (>5 minutes) imaging is recommended in the setting of renal lacerations to evaluate for urinary leaks (Fig. 19-25). Isolated fluid around the ureters on initial scanning should raise the possibility of an injury, and 5-minute delayed scanning is indicated (Fig. 19-27).

Portal venous or delayed phase scanning through the pelvis is helpful for the detection of pelvic active bleeding. Images of all acquired phases should be reconstructed at 2- to 5-mm slice thickness in the axial, sagittal, and coronal planes.

The arterial-weighted phase images are particularly useful for the detection of brisk arterial bleeding and may detect an intraparenchymal pseudoaneurysm in a solid organ or an arteriovenous fistula, which may otherwise go undetected if portal venous phase imaging alone is acquired (Fig. 19-26). Recent literature suggests that a comprehensive evaluation to assess for the presence of a splenic injury requires both arterial and portal venous phases. The arterial phase performs better for the detection of splenic pseudoaneurysms, and portal venous phase has improved detection for parenchymal lacerations and active bleeding over arterial phase alone.[76] Extravasation of venous contrast is observed in 5% to 10% of victims of high-energy blunt trauma and may be difficult to differentiate from slow arterial bleeding on CT. The spleen is the most common region of isolated active extravasation of contrast; however, fractures of the pelvic ring are most commonly associated with multiple sites of extravasation.[78] The amount of hematoma associated with disruptions of the pelvic ring directly correlates with the likelihood of an angiographically demonstrable arterial injury (200 cm^3, 5% arterial injury; >500 cm^3, approximately 50% arterial injury).[79] Nonetheless, otherwise unexplained continued hemodynamic instability in patients with blunt pelvic fractures warrants angiographic evaluation, even in the absence of a pelvic hematoma or active pelvic bleeding on the initial CT (Fig. 19-28).[80]

Active bleeding identified on a CT is an independent predictor for the failure of nonoperative management, particularly when bleeding is arising from the spleen, liver, mesentery, or pelvis. Active bleeding from the spleen or liver is a stronger indicator for the need for intervention than the organ injury grade alone (Fig. 19-21). The detection of lacerations that extend to the hepatic veins is of particular importance in the liver, as these have a strong predictive value for failure of nonoperative management when associated with large (>10 cm) hypoperfused regions.[81] Adrenal hemorrhage is relatively common, particularly on the right, and is not of clinical importance unless bilateral. Even then, posttraumatic hypoadrenalism is rare. It should be noted that CT grading scales for the solid organs do exist, and although there is significant overlap with the American Association for the Surgery of Trauma (AAST) solid organ grading scales, they are not identical. Thus, open communication with the interpreting radiologist is recommended to avoid any miscommunication. Evaluation of the lower genitourinary tract can be performed with urethrography or cystography (Figs. 19-29 and 19-30). CT cystography typically is performed after the

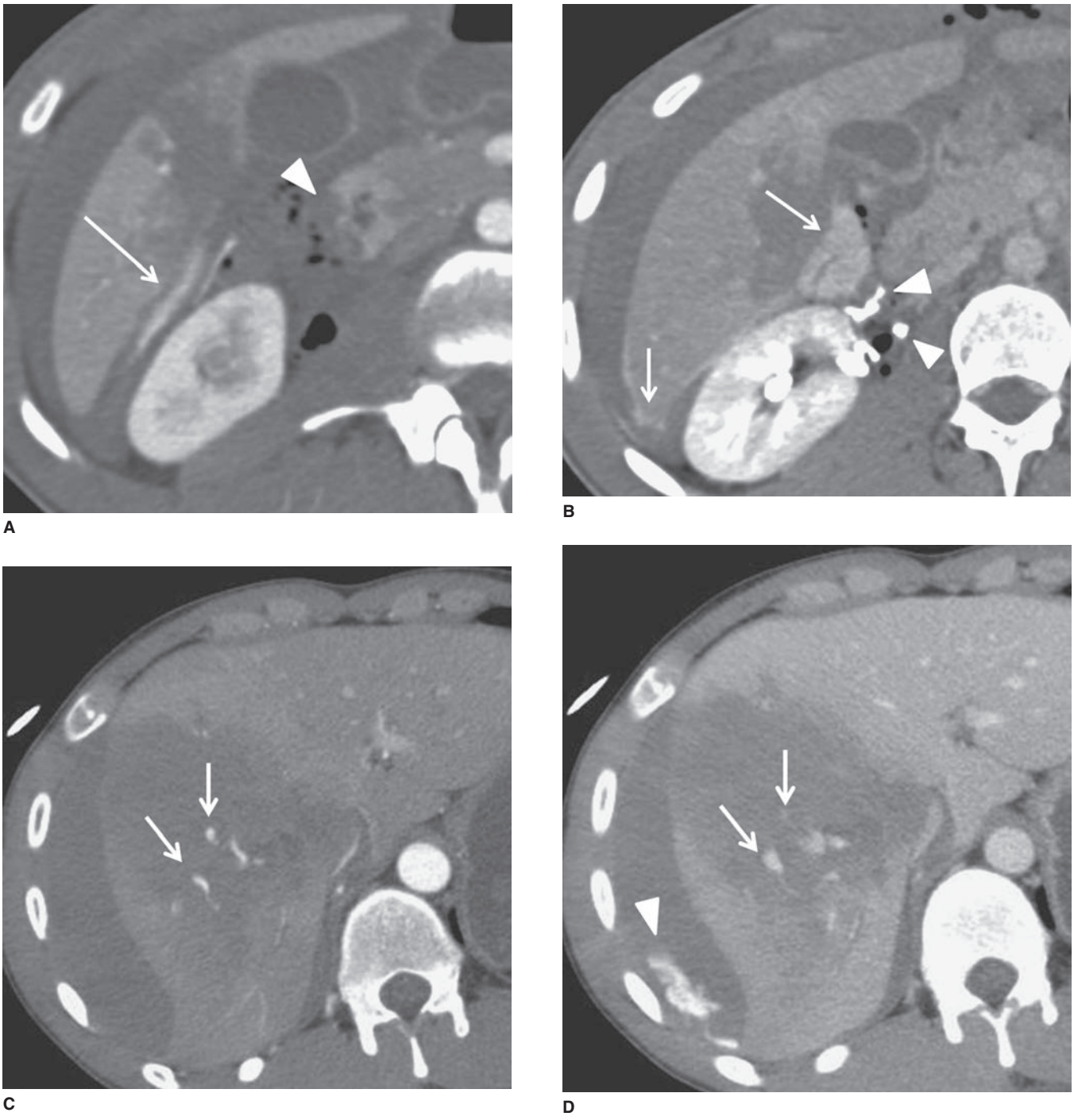
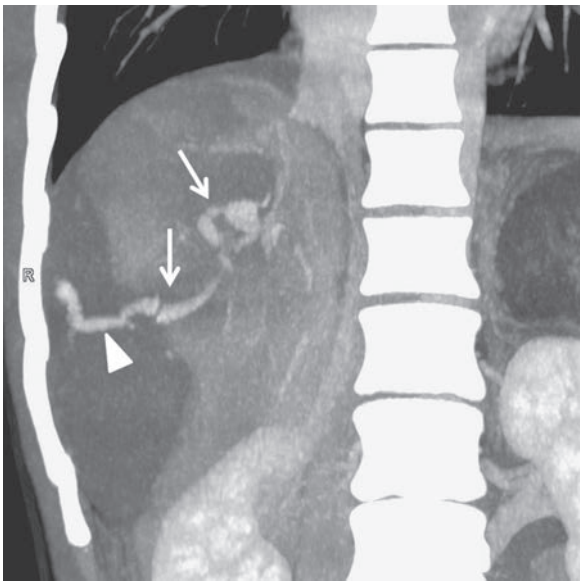


FIGURE 19-21 Examples of contrast extravasation in the abdomen. (**A, B**) Laceration of the right hepatic lobe following a gunshot wound. There is active contrast extravasation seen on arterial phase (**A**, arrow), which enlarges on 5-minute delayed phase (**B**, arrows) compatible with active bleeding. There is retroperitoneal gas due to a full-thickness duodenal injury (**A**, arrowhead). Five-minute delayed scan (**B**) also reveals extravasated urinary contrast from a renal pelvis laceration (**B**, arrowhead). (**C–E**) Active bleeding from a large liver laceration. Axial arterial phase (**C**), axial portal venous (PV) phase (**D**), and coronal maximum-intensity projection PV phase.



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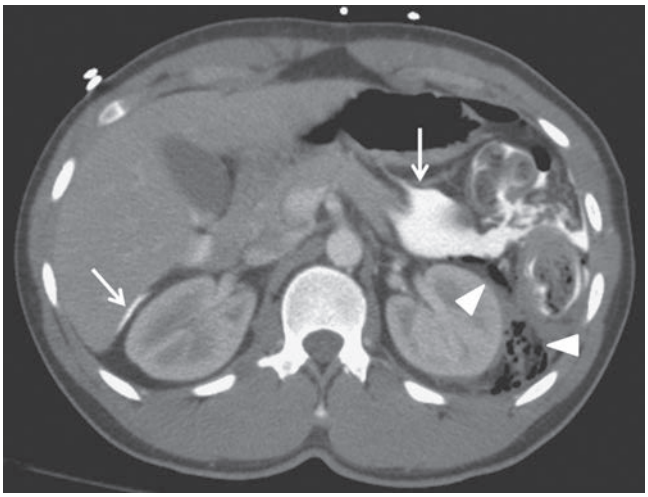

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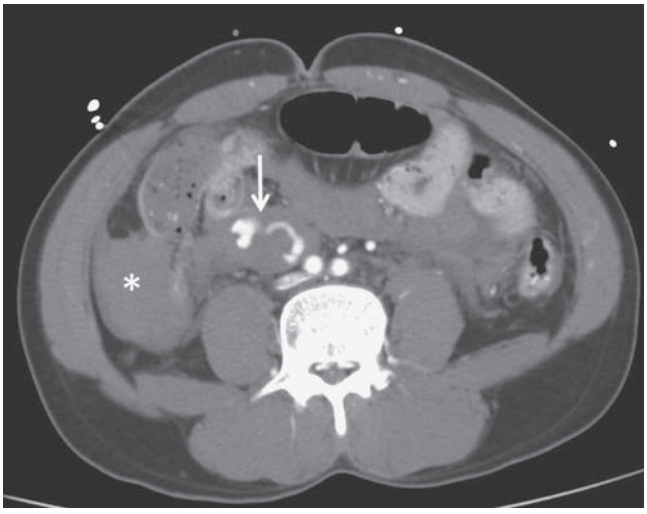

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FIGURE 19-21 (*Continued*) (**E**) Image demonstrates active bleeding (arrows), which enlarges on the PV phase, accumulating in a large subcapsular hematoma (arrowhead). (**F**) Bowel contrast leak. A 23-year-old male following a left flank gunshot wound demonstrates leakage of a large amount of rectally administered contrast (arrows) from a full-thickness colon injury. Bubbles of extraluminal gas are seen in the left retroperitoneum (arrowheads). (**G**) Active contrast extravasation (arrow) from small bowel mesenteric root in a 28-year-old male following a motor vehicle collision. A large amount of free fluid and hemoperitoneum (asterisk) are also present. Active bleeding from the mesentery and an associated small bowel perforation were identified at surgery.

administration of intravenous contrast, as a large amount of leaked extraperitoneal contrast may obscure active arterial extravasation in the pelvis. CT cystography can be easily included in the initial evaluation of the trauma patient after the traditional trauma CT survey and is highly accurate for the diagnosis, exclusion, and comprehensive characterization of bladder injuries.[82] Passive physiologic filling of the bladder (eg, a 10-minute delay with a clamped Foley catheter) may detect injuries to the bladder but has been shown to be insufficient for the reliable exclusion of all injuries and should not be relied on as the sole examination for suspected bladder trauma (Fig. 19-30).[82,83]

To perform CT cystography, the bladder is first emptied of unopacified urine to diminish the effects of contrast dilution. Scanning through the pelvis is then performed. Administration of dilute water-soluble contrast via low gravity drip can proceed only after the positioning of the Foley catheter within the bladder is confirmed. It is not uncommon for a Foley catheter to be inserted through a laceration of the bladder dome in a patient with an intraperitoneal bladder

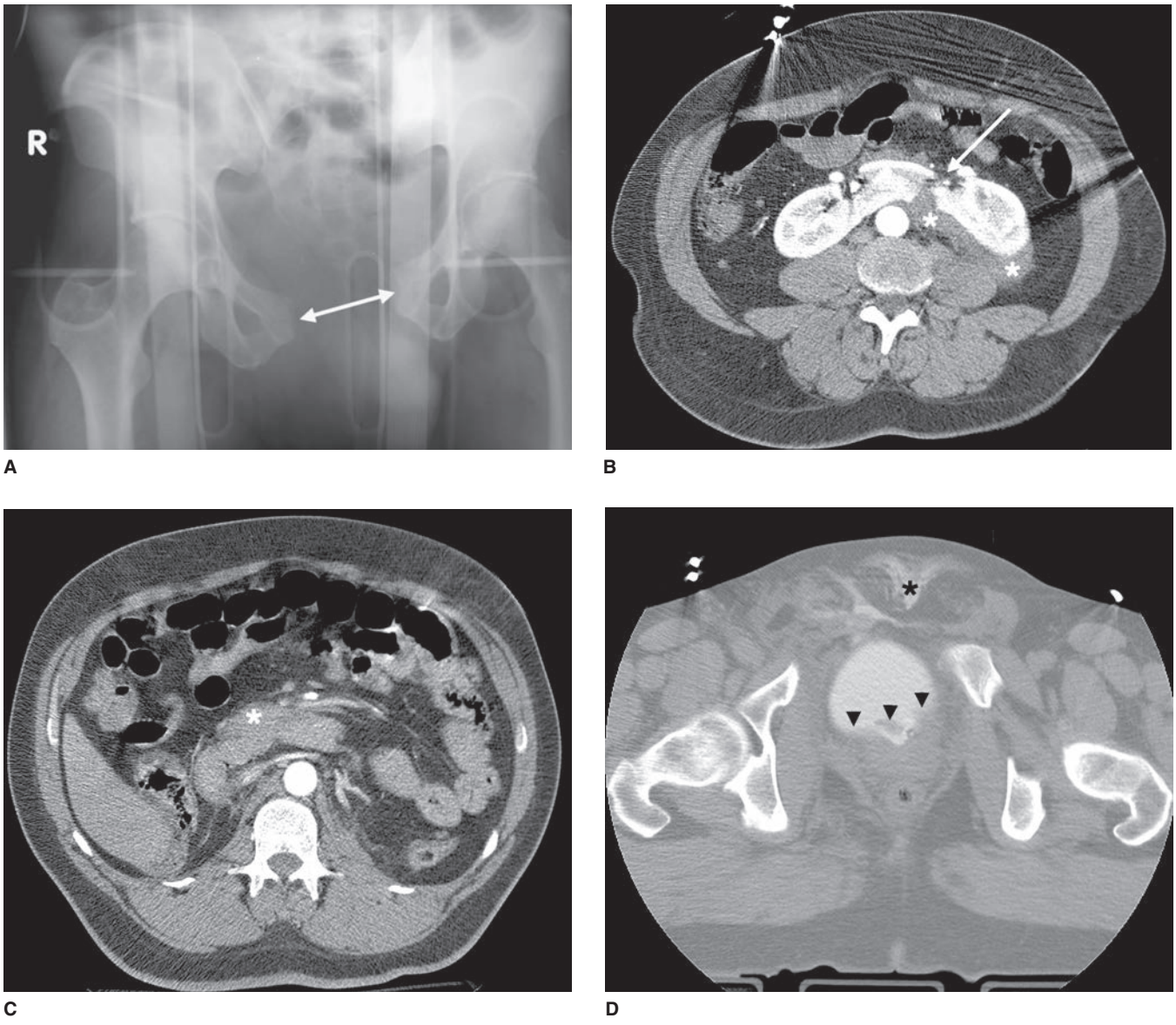

FIGURE 19-22 Patterns of injury: "the central package." This 54-year-old male motorcyclist sustained multiple injuries, including laceration of horseshoe kidney, duodenal contusion, bladder rupture, and anteroposterior (AP) compression fracture of pelvic ring. (**A**) AP radiograph of pelvis. Greater than 2.5-cm diastasis of pubic symphysis is compatible with disruption of sacrospinous, sacrotuberous, and anterior capsular ligaments of sacroiliac joints. Appearance supports AP compression mechanism and is associated with increased risk for intra-abdominal, intrathoracic, and head injuries. (**B**) Axial computed tomography (CT) of abdomen at L3–L4 level in the arterial phase. White arrow shows median fracture of horseshoe kidney with posterior perinephric hematoma (asterisks). This is an arterial phase image because there is dense opacification of aorta directly posterior to neck of horseshoe kidney without opacification of the inferior vena cava immediately to its right. Arterial phase images best demonstrate active extravasation and pseudoaneurysms. (**C**) Axial CT at level of third portion of duodenum shows paraduodenal hematoma (asterisk), suggestive of duodenal injury. (**D**) Axial CT at level of right acetabulum shows widening of symphysis and extraperitoneal bladder laceration as contrast in anterior abdominal wall (asterisk). Posterior wall of bladder is irregular with double densities within urine contrast compatible with hematoma (arrowheads).

rupture. The bladder should then be filled with at least 250 to 300 mL of contrast, or until the patient can no longer tolerate the degree of bladder distention or until the contrast stops passively flowing into the bladder. It should be noted that contrast may continue to freely flow in patients with intraperitoneal bladder injuries, and infusing over 350 to 400 mL without scanning is not advised. Scanning through the pelvis with maximum bladder distention is then performed. All scan series are reconstructed in axial, sagittal, and coronal planes at 1- to 3-mm slice thickness. Review of the images using "bone" windows may make the identification of the source of urinary leak more apparent, depending on the density of the contrast material in the bladder. Postvoid scanning may identify small bladder leaks, but this is rare.

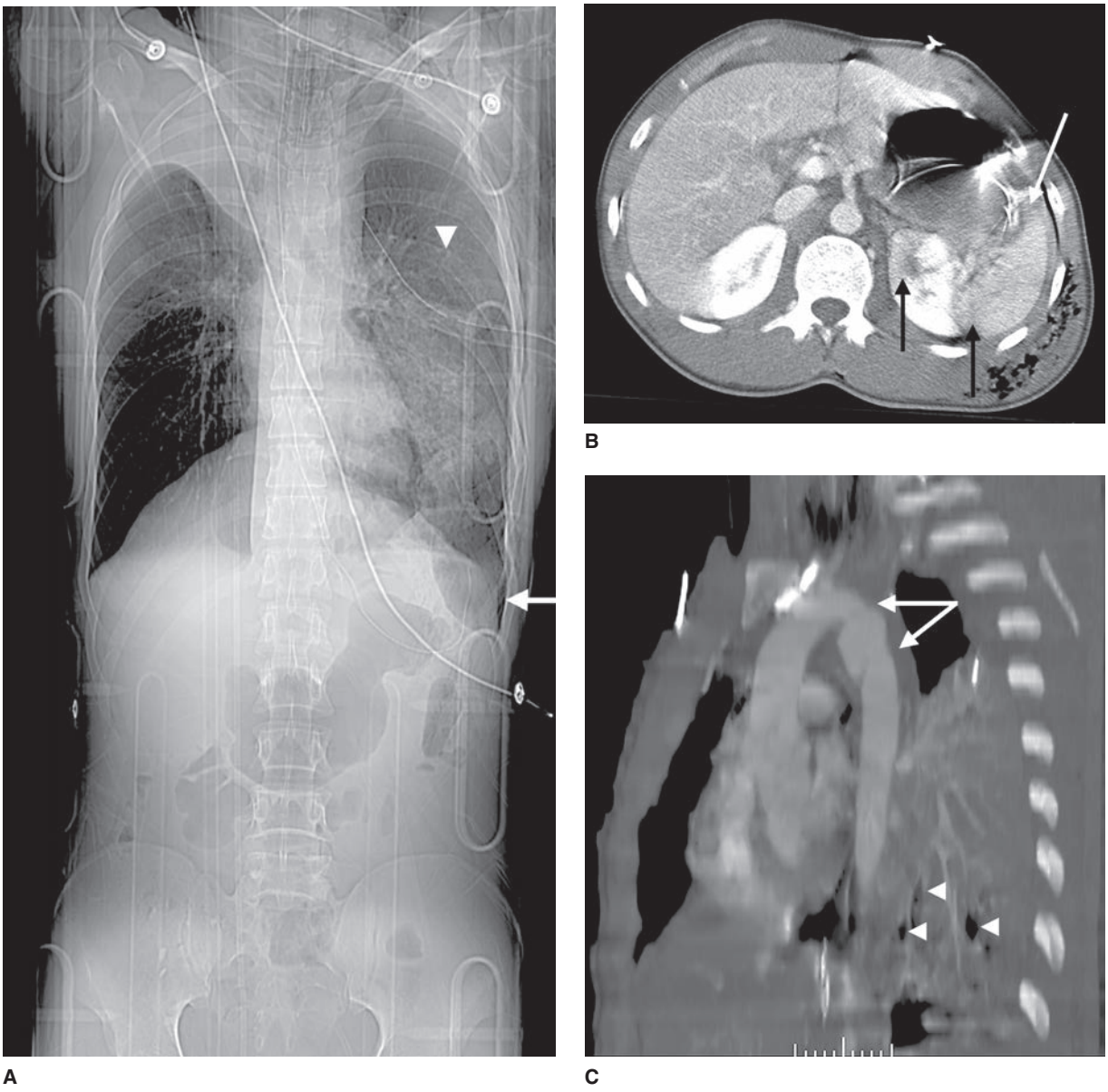

FIGURE 19-23 Patterns of injury: "the left package." This 22-year-old male driver was injured in a side-impact crash with substantial intrusion to driver's side of car. Multiple injuries were sustained. (**A**) Anteroposterior (AP) scanogram from computed tomography (CT) of chest, abdomen, and pelvis shows extensive opacity of left mid and lower lung fields compatible with contusion, a deep sulcus (arrow) at left costophrenic angle compatible with left pneumothorax, and multiple left-sided rib fractures (arrowhead). Patient is intubated, and there is right upper lobe collapse. (**B**) Axial CT of upper abdomen during parenchymal phase shows injury of the anterior portion of left kidney (medial arrow) and splenic laceration with sentinel clot (black and white arrows, respectively). Although rib fractures are not shown on current image, subcutaneous emphysema in left chest wall and distal extent of small pneumothorax are shown. (**C**) Oblique sagittal reformation from CT aortography shows complex segmental intimal injury to proximal descending aorta in the typical location (arrows) and pseudoaneurysm formation due to acute traumatic aortic injury. Air-fluid levels (arrowheads) are compatible with pulmonary lacerations.

Indications for CT cystography include hematuria and fracture of either the pelvic ring or acetabulum or hematuria and free intraperitoneal fluid in the absence of a clearly identifiable source. Cystography is low yield in the absence of hematuria and pelvic fractures.

Injuries to the bowel and mesentery are thought to occur in 1% to 5% of patients with blunt trauma but, when present, are associated with increased morbidity and mortality primarily due to a delayed diagnosis.[84,85] The physical examination alone lacks sensitivity and specificity, particularly in patients with an unreliable physical examination.[85] The sensitivity and specificity of CT for bowel injuries are modest, ranging from 69% to 95% and 94% to 100%, respectively.[85] Specific CT findings of bowel injuries include thickened bowel

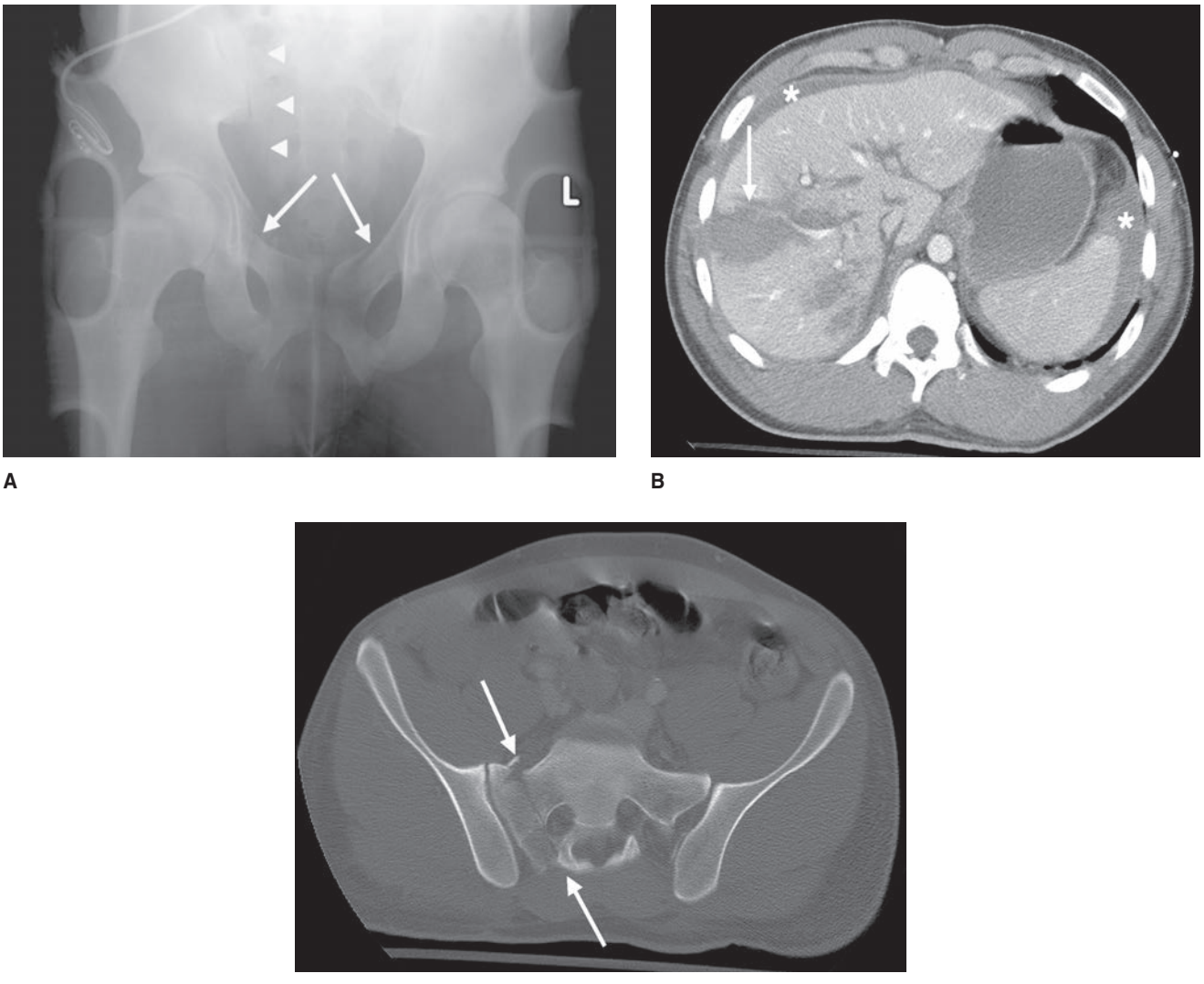

FIGURE 19-24 Patterns of injury: "the right package." This 22-year-old unrestrained passenger was ejected from a car in side-impact high-speed crash. (**A**) Anteroposterior (AP) view of pelvis shows bilateral iliopubic and ischiopubic ramus fractures (white arrows) and disruption of right sacral arcuate lines (arrowheads); findings are compatible with lateral compression fracture due to right lateral impact. (**B**) Axial contrast-enhanced abdominal computed tomography (CT) shows free intraperitoneal fluid (asterisks) due to complex collection of liver lacerations (arrow) extending to the intrahepatic inferior vena cava. The relatively uniform enhancement of hepatic parenchyma suggests that the hepatic veins are not occluded. (**C**) Axial CT through S1, bone windows, shows through-and-through fracture of S1 ala, which traverses S1 neuroforamina (white arrows). Such through-and-through fractures are typically associated with biomechanical instability.

wall, asymmetric mural enhancement, pneumoperitoneum, leak of oral contrast, focal defect in the bowel wall, and free fluid not explained by other injuries (Fig. 19-31). Unfortunately, the specific signs of a bowel perforation are relatively uncommon and are not sensitive.[85,86] Active bleeding from the bowel mesentery, as with active bleeding elsewhere in the body, is an indication for intervention and is associated with a high likelihood of injury to the bowel or delayed necrosis of the bowel.[87] Fluid present in the mesentery or between loops of bowel is very suspicious for a transmural injury to the bowel, even in the presence of injury to a solid organ and even immediately following DPL (Fig. 19-31). One note of caution regarding free intraperitoneal fluid is that women of childbearing age may have small amounts of fluid (±50 mL) in their pelvis. Recent observations also suggest that a small volume of isolated free fluid in the pelvis in male patients with blunt abdominal trauma, which can be seen in up to 4.9%, is unlikely to be due to a bowel injury.[88] Patients who have been vigorously resuscitated (especially if they are >24 hours from their injury) may have ascites and interloop fluid present due to a capillary leak syndrome (Fig. 19-32). Acute and subacute hemorrhage typically measures 40 to 70 Hounsfield units (HU), whereas urine, bowel contents, and ascites measure closer to water (eg, 0 +/–20 HU). If a patient

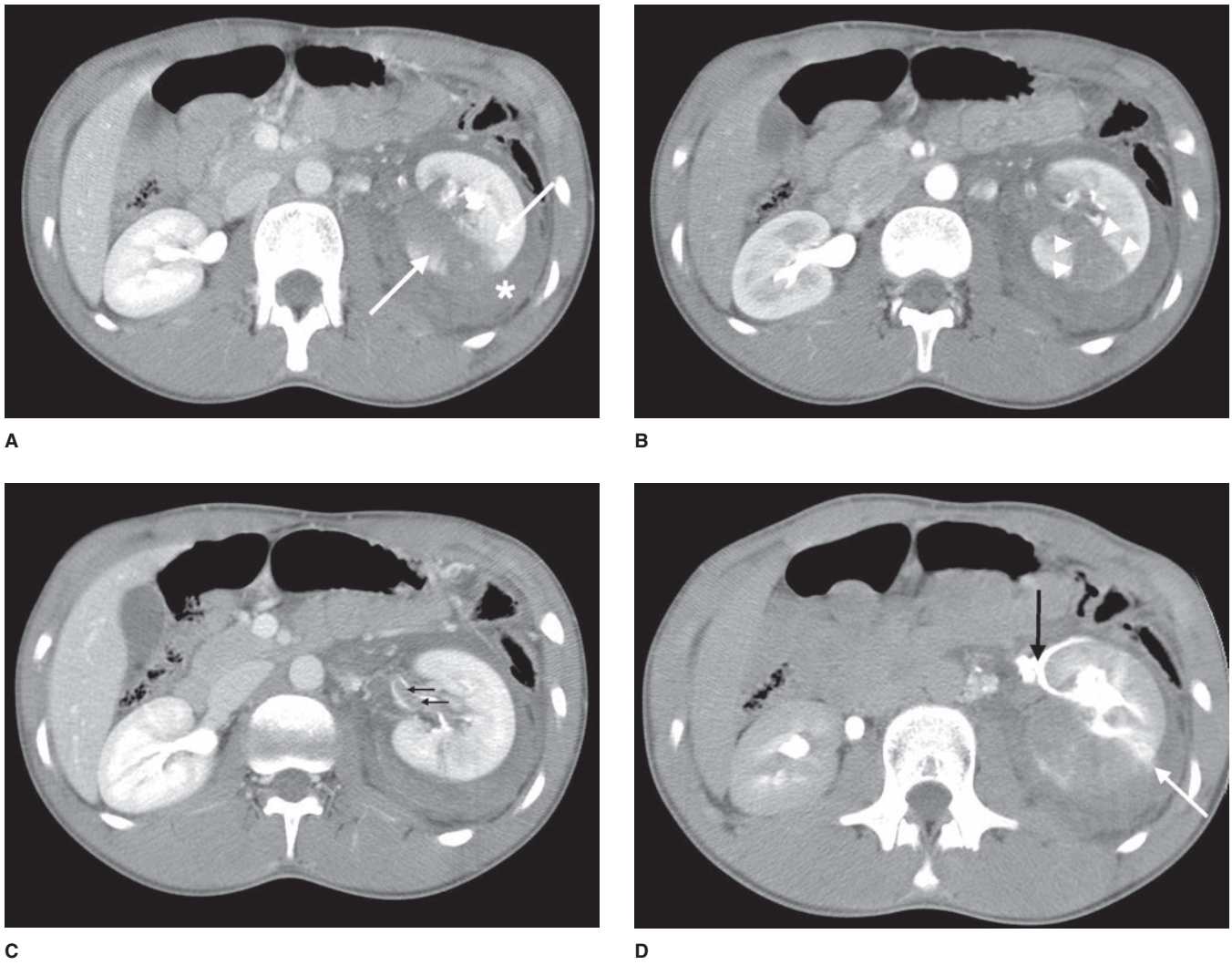

FIGURE 19-25 Grade IV renal laceration. This 14-year-old sustained an injury in a fall from a dirt bicycle while jumping. (**A**) Contrast-enhanced axial computed tomography (CT) scan, soft tissue windows, performed in parenchymal phase shows perinephric hematoma on left (asterisk) adjacent to laceration that extends into renal hilum (arrows). Right kidney shows normal pyelographic phase. Free intra-abdominal fluid is due to grade III splenic laceration (not shown). (**B**) Contrast-enhanced axial CT obtained in arterial phase shows wedge-shaped defect in left kidney (arrowheads) compatible with laceration and infarct extending to capsule secondary to segmental arterial occlusion. (**C**) Contrast-enhanced axial CT at level of kidneys shows thrombus within collecting system (black arrows), perinephric hematoma, and contusion of posterior aspect of kidney, just below laceration seen on image **A**. Perinephric hematoma surrounds kidney. (**D**) In such complicated cases, delayed images (10 minutes) are highly valuable in assessing associated urinary leakage. Ten-minute delayed images show a type III extravasation of contrast-enhanced urine from anterior and medial pole of kidney into the perinephric space (black arrow). Striated nephrogram is present posteriorly (white arrow), compatible with contusion adjacent to laceration.

with blunt trauma has free fluid in the peritoneal cavity of uncertain source and lacks signs of peritonitis on physical examination, a repeat short-interval abdominopelvic CT in 4 to 6 hours may be warranted.[89]

CT performs well in the diagnosis of peritoneal violation and intra-abdominal injuries in the setting of penetrating abdominal trauma.[73] The diagnosis of injuries to the bowel following penetrating trauma is aided by the use of the triple-contrast protocol using intravenous, oral, and rectal contrast material. Leakage of enteric contrast is highly specific for bowel injury (Fig. 19-21F). A wound tract that extends to the bowel surface is the most sensitive sign of an associated bowel injury, in the absence of other definitive findings.

Computed Tomography of the Pelvis and Acetabulum

CT is extremely valuable in the evaluation of the pelvic girdle, including the pelvic bones, acetabulum, and sacrum, following both blunt and penetrating trauma. There are several indications for pelvic CT, including the following: (1) to evaluate

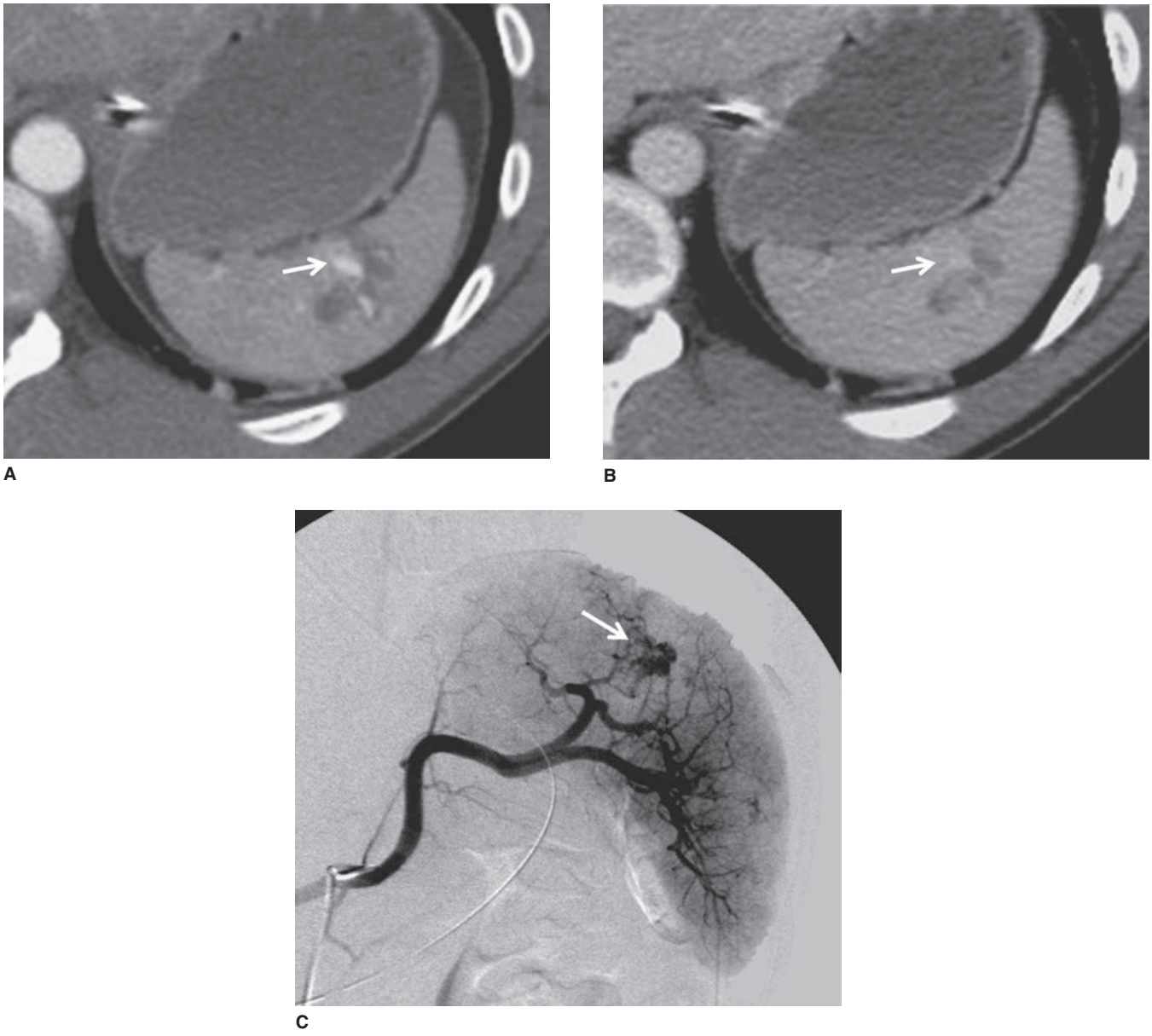

FIGURE 19-26 Spleen laceration with associated pseudoaneurysm. Axial postcontrast computed tomography during arterial (**A**) and portal venous (**B**) phases demonstrate a laceration of the splenic hilum. There is a focal hyperdensity within the laceration on arterial phase (**A**, arrow), which becomes isodense to adjacent parenchyma on portal venous phase (**B**, arrow) consistent with a traumatic pseudoaneurysm. (**C**) Splenic angiogram confirms the presence of a splenic pseudoaneurysm (arrow).

unstable fractures of the pelvic ring as determined by physical examination or appearances on conventional x-rays; (2) to evaluate for radiographically occult fractures in the setting of high clinical suspicion; (3) to detect entrapped intra-articular debris/bone fragments following hip dislocation; and (4) for preoperative surgical planning (Figs. 19-33 to 19-37). Pelvic CT can be performed on its own or be reconstructed from the raw CT data of the abdomen and pelvis performed as a part of a whole-body trauma CT series. Three-dimensional volume-rendered images can be generated from the CT data set, and dedicated evaluation of acetabular fractures can be performed with femoral head subtraction. In fact, some of the most recent CT scanner software packages can generate "virtual pelvis" images from the CT raw data that approximate images of conventional pelvic radiographs (Fig. 19-2D).

The primary goal of CT evaluation of disruption of the pelvic ring and acetabular fractures is to aid in surgical planning.[90] CT scans of acetabular fractures are performed for the following: (1) assessment of fracture types; (2) secondary congruence of the hip (eg, are the fracture fragments symmetrically oriented about the intact femoral head?); (3) evidence for marginal impaction (eg, subarticular bone depressed or

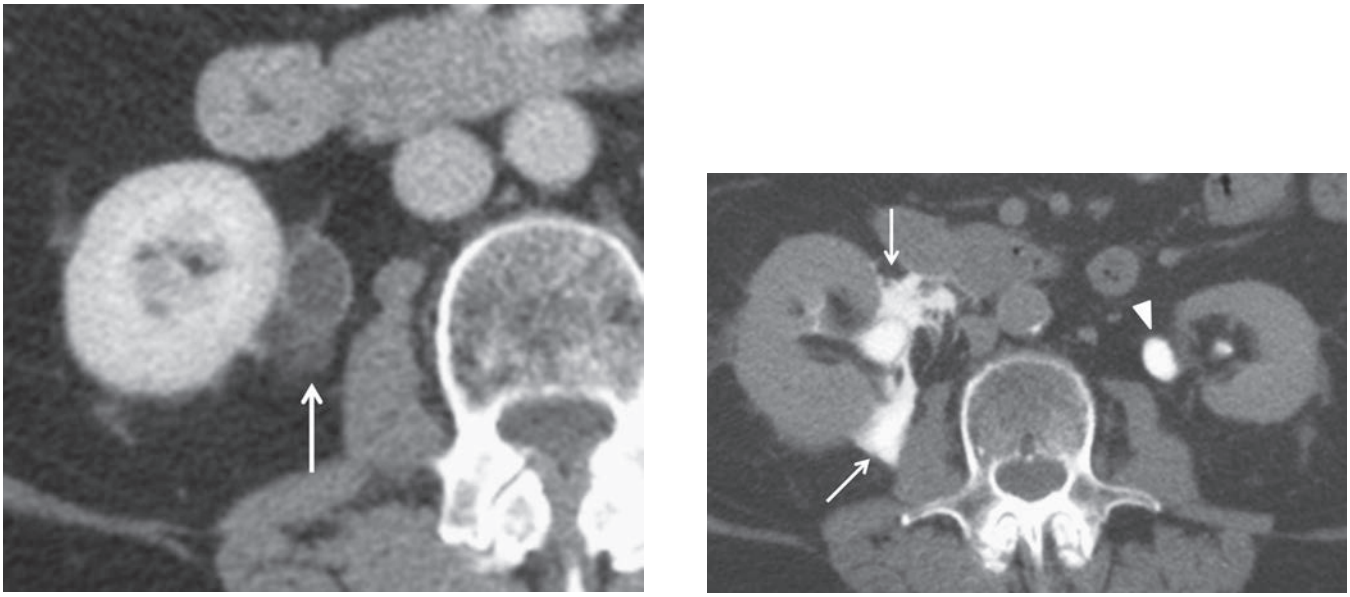

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FIGURE 19-27 Renal pelvis laceration following blunt abdominal trauma. (**A**) Admission portal venous phase image reveals a small amount of fluid (arrow) surrounding the right renal pelvis. No other injuries or other abnormalities were seen. (**B**) Delayed excretory phase image reveals a large amount of extravasated urinary contrast (arrows) in the retroperitoneal space compatible with a renal pelvis laceration. The contralateral normal renal pelvis (arrowhead) is included for comparison.

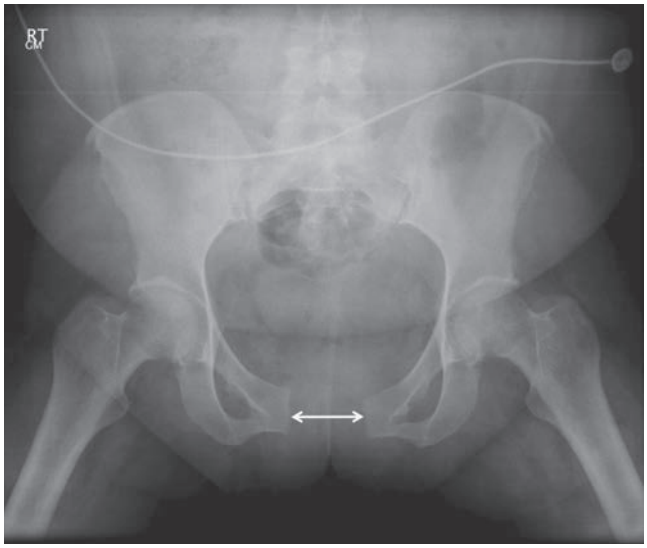
impacted and not showing secondary congruence); (4) detection of a fracture of the femoral head; and (5) detection of intra-articular debris. There is approximately a 15% concurrent rate for fractures of the pelvic ring and acetabulum, so any fracture of the pelvis or acetabulum should initiate a search for other fractures in the pelvic girdle.

CT of the pelvis and acetabulum typically uses a slice thickness of 1 to 3 mm in the axial plane, and images are reconstructed using a bone algorithm. Sagittal and coronal reformatted images can be helpful in fully characterizing a fracture of the pelvic ring or acetabulum.

A systematic approach should be employed when reviewing pelvic CT images, with special attention paid to the fractures that one would expect to see in the setting of AP compression, lateral compression, and vertical shear force. Correlation with at least an AP pelvic radiograph is recommended to provide an overview of the type of pelvic ring disruption. Fractures of the iliopubic and ischiopubic rami, as well as the sacral wings, are typical of a lateral compression-type injury. The anterior surface of the sacrum is carefully evaluated for "buckle" fractures due to internal rotation of the hemipelvis due to a lateral impact. Fractures of the iliopubic and ischiopubic rami are assessed for their orientation (lateral compression fractures typically show orientation in the axial plane or coronal plane, whereas AP compression and vertical shear fractures will show orientation in the sagittal plane). Diastasis of the pubic symphysis and sacroiliac joints (eg, "open book" pelvis) is typical of an AP-type injury. The normal pubic symphysis should not be more than 1 cm in width in normal subjects, regardless of age. When the pubic symphysis is traumatically wider than 2.5 cm, both sacrospinous and sacrotuberous ligaments are presumed to be injured. In the posterior ilium, it is important to look for avulsion fractures of the posterior superior iliac spine, so-called crescent fractures, as these are strongly associated with biomechanically unstable fractures in the presence of disruption of the anterior pelvic ring. The axial images give good evaluation of the amount of internal or external rotation but underestimate the amount of flexion or extension of a hemipelvis relative to the intact pelvis. Furthermore, evaluation of the amount of pelvic hematoma may be helpful in determining the need for angiography for embolization. Localization of these hematomas is a good predictor of associated pelvic vascular injuries. Indeed, as discussed earlier, a search for active bleeding in the pelvis should be conducted if the patient undergoes the contrast-enhanced trauma CT series.

It should be noted that CT is not a dynamic examination and provides information for a single snapshot in time. As such, the assessment of potential biomechanical instability of the pelvis may be difficult. Certainly, the combination of a crescent fracture from the posterior superior iliac spine and displaced anterior pelvic ring fractures will be unstable under anesthesia. The stability of other patterns, however, is not so predictable, even though CT images provide a great deal of information about the injury pattern. Therefore, conventional x-rays are necessary as guides to intraoperative reduction even for the most experienced pelvic orthopedic surgeon.

If an acetabular fracture is present, the goal is to determine what remains attached to the intact hemipelvis and to describe and characterize the major fracture fragments and



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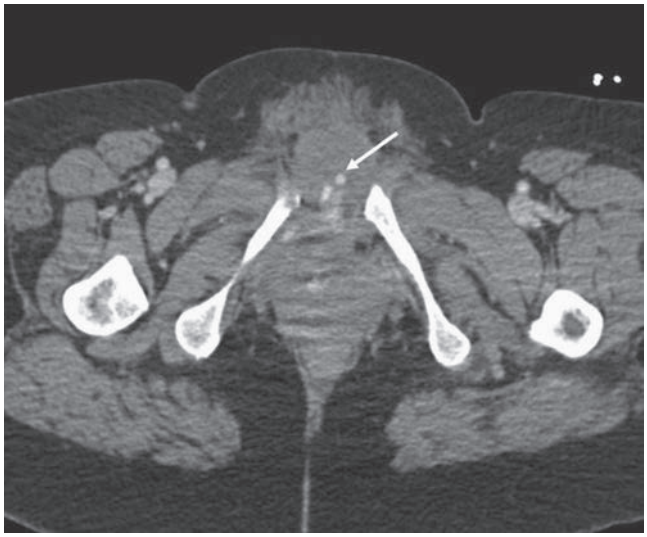

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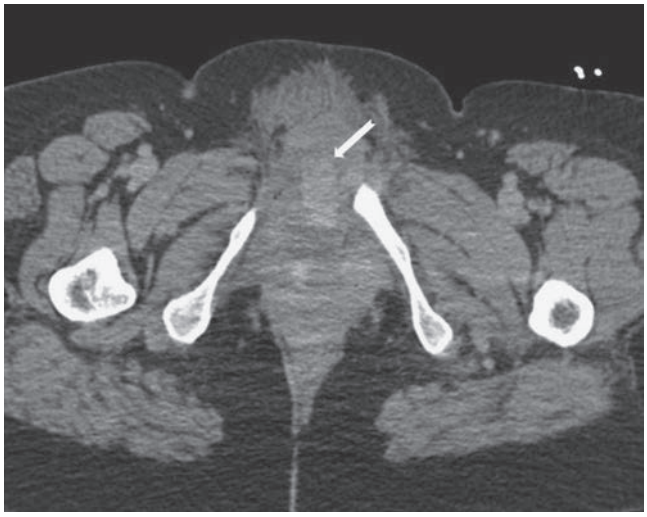

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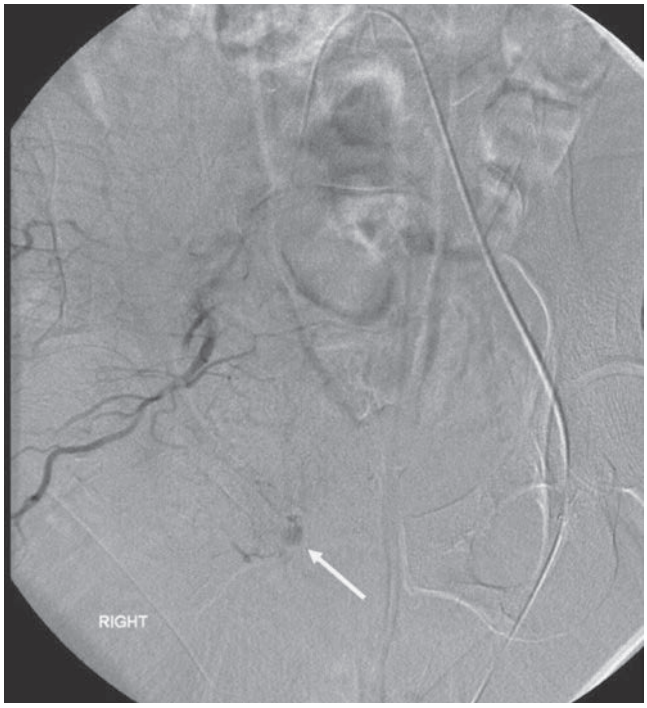

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FIGURE 19-28 Pelvic ring disruption and active bleeding in a young female following a motorcycle collision. (**A**) Anteroposterior radiograph demonstrates marked diastasis of the pubic symphysis (double arrow). Axial arterial phase (**B**) and delayed phase (**C**) computed tomography images at the level of the widened pubic symphysis reveal active bleeding (**B**, arrow), which increases in size (**C**, notched arrow) and diffuses into the existing blood pool. (**D**) Right posterior oblique pelvic digital subtraction angiogram shows the focus of active bleeding (arrow) next to the right pubic root.

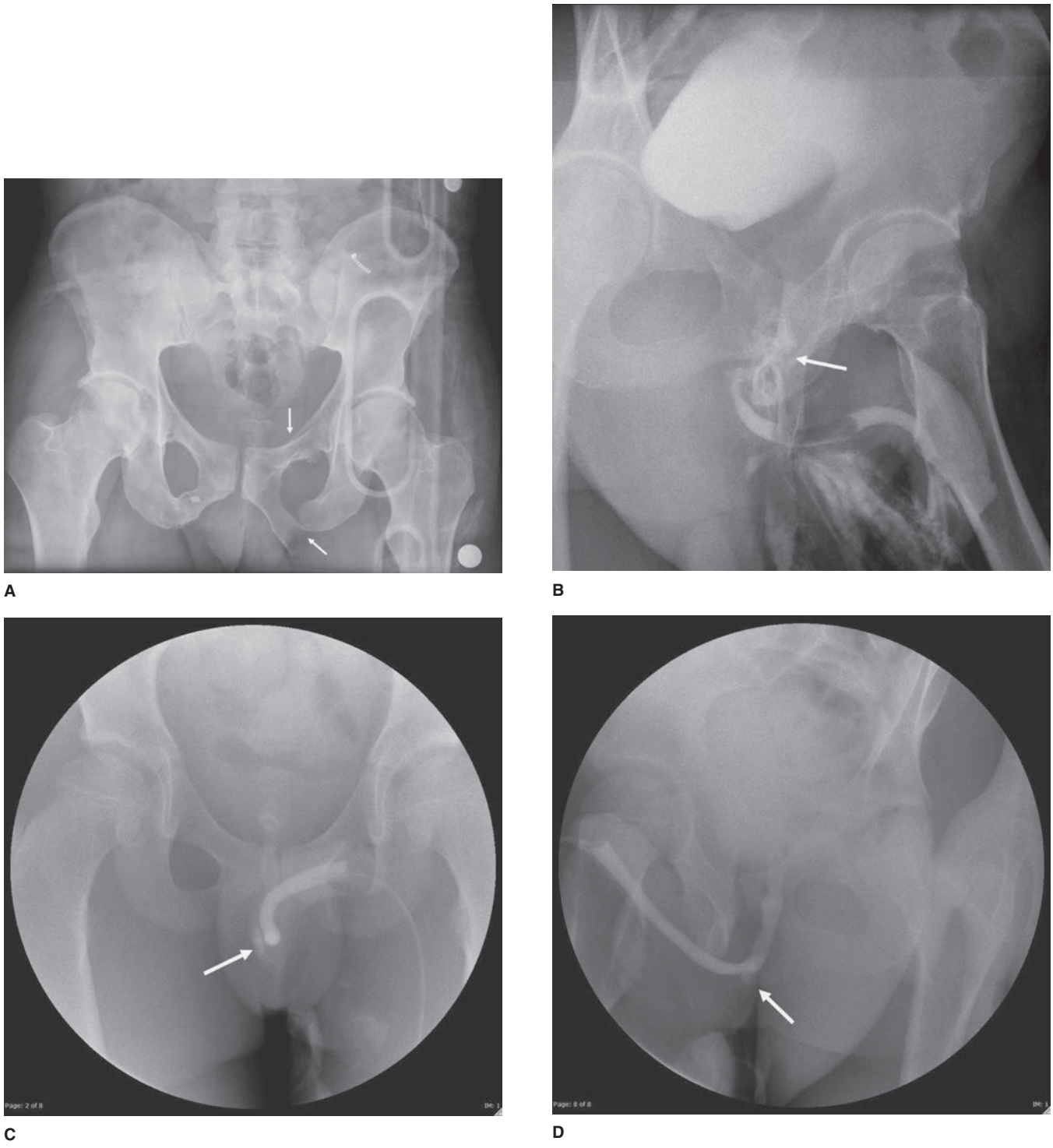
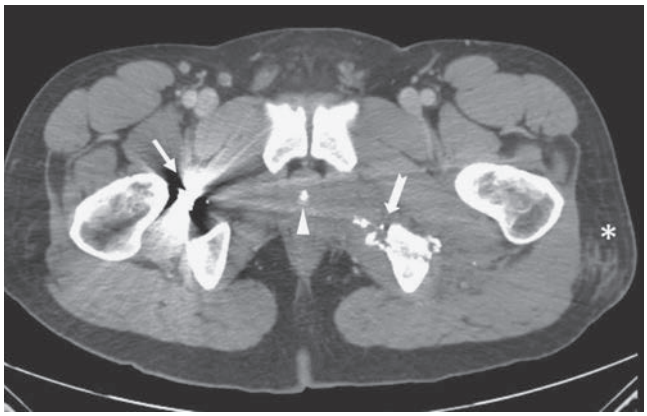


FIGURE 19-29 Urethral injuries. (**A, B**) Young male status post blunt pelvic trauma. (**A**) Anteroposterior (AP) pelvic radiograph demonstrates fractures of the left superior and inferior pubic rami (arrows). The patient was unable to void, and there was blood at the penile meatus. (**B**) Left posterior oblique image from a retrograde urethrogram (RUG) demonstrates leak of contrast from the membranous portion of the urethra (arrow), with contrast extending inferior to the urogenital diaphragm. (**C, D**) An 11-year-old male with a straddle injury and blood at the meatus. AP (**C**) and right posterior oblique (RPO) (**D**) RUG images demonstrate focal contrast outpouching along the right inferior aspect of the bulbous anterior urethra (arrows) indicating a urethral injury. (**E, F**) A 27-year-old male status post transpelvic gunshot wound.



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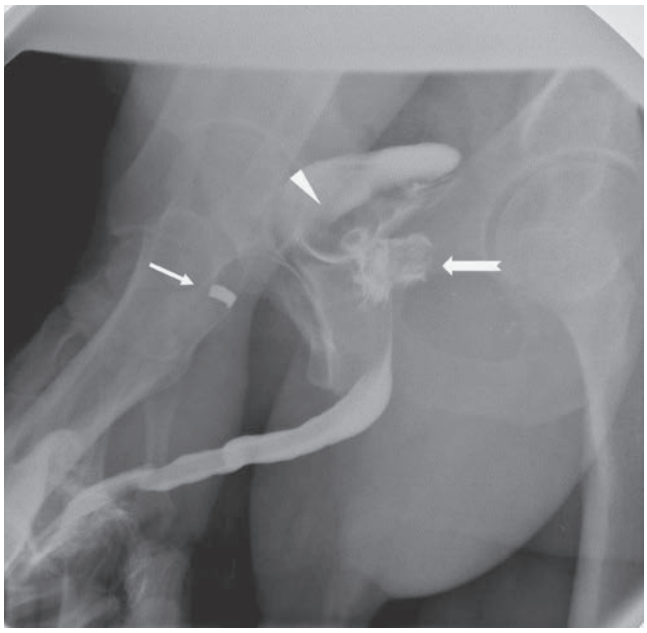

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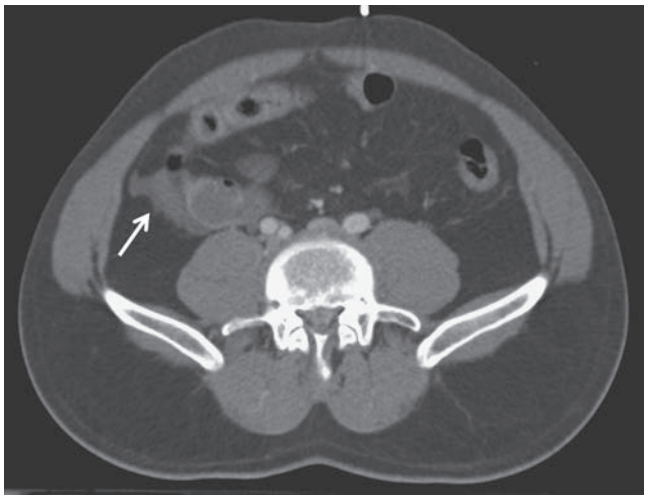
FIGURE 19-29 (*Continued*) (**E**) Axial computed tomography image through the lower pelvis shows the bullet trajectory extending through the region of the urethra. The wound tract extends from the left lateral gluteal region (asterisk), through the left ischiopubic ramus (notched arrow), with bullet resting just medial to the right proximal femur (arrow). A Foley catheter (triangle) was able to be passed in the trauma bay without incident. (**F**) RPO RUG image, performed by inserting a small-bore pediatric feeding tube adjacent to the existing Foley catheter, shows leak of contrast from the prostatic portion of the urethra (notched arrow). The Foley balloon is appropriately positioned in the bladder (arrowhead). Bullet fragment (arrow) is again seen adjacent to the right proximal femur.



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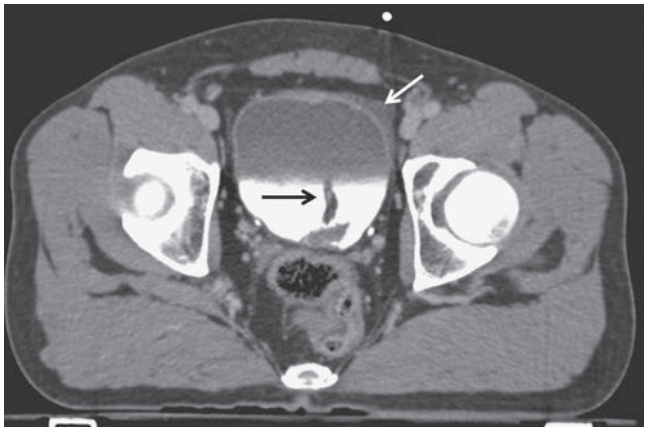

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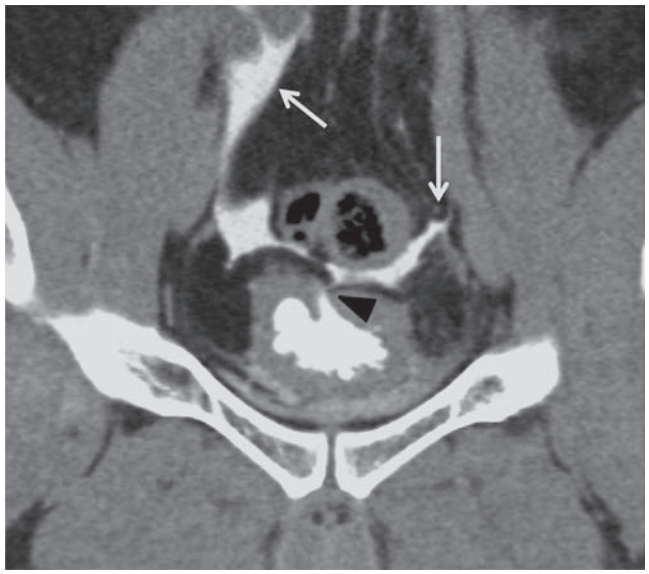
FIGURE 19-30 Blunt traumatic bladder rupture. (**A–C**) Intraperitoneal bladder rupture in a patient following blunt trauma. (**A**) There is free fluid in the right paracolic gutter (arrow) without other computed tomography (CT) signs of solid organ injury or bowel perforation. (**B**) Five-minute delayed excretory phase image through the pelvis demonstrates filling defects (black arrow), due to blood clot, in a partially opacified bladder. There is no leak of urinary contrast via passive physiologic filling of the bladder. There is trace free fluid adjacent to the bladder (white arrow).



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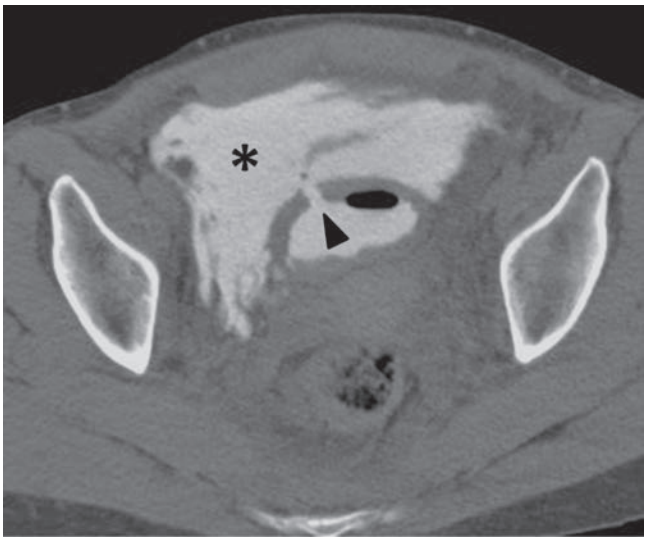

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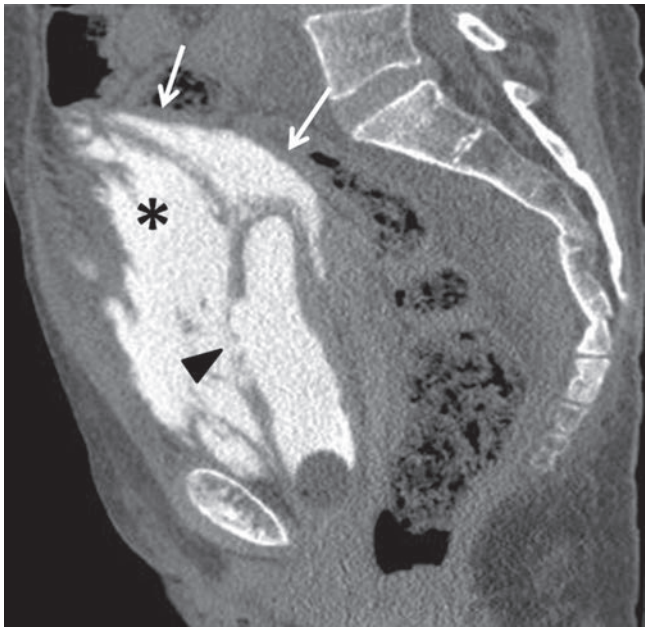

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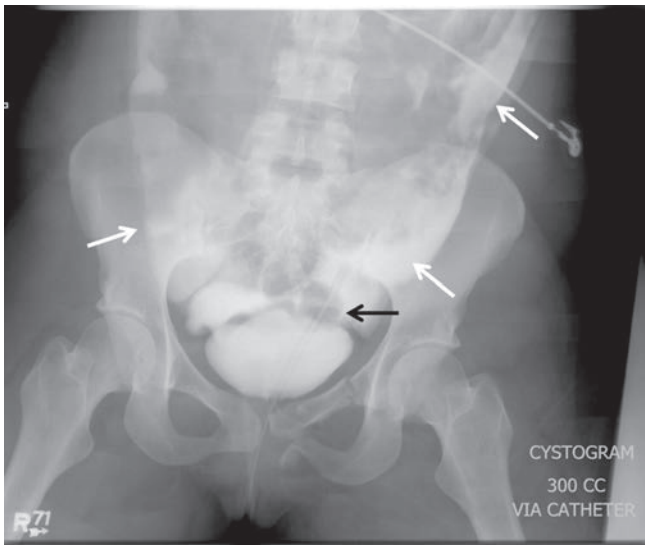

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FIGURE 19-30 (*Continued*) (**C**) Coronal CT image from a CT cystogram reveals a focal defect in the bladder dome (black arrowhead) and free flow of contrast into the peritoneal cavity (white arrows) consistent with an intraperitoneal bladder injury. (**D, E**) Extraperitoneal bladder rupture in another patient following blunt trauma. Axial (**D**) and sagittal (**E**) CT cystogram images demonstrate a focal defect in the anterior bladder wall (black arrowhead) and leak of urinary contrast into the perivesicular space (asterisk). The leaked contrast is confined to the extraperitoneal space by the peritoneal reflection superiorly (white arrows). (**F**) Conventional cystogram from another patient with an intraperitoneal bladder injury. Radiograph demonstrates a large amount of intraperitoneal contrast outlining loops of bowel (white arrows). Close inspection of the image demonstrates the bladder catheter balloon (black arrow) outside of the bladder, traversing the bladder wall through a dome injury. Careful inspection of all images, both CT and film, is necessary during the cystogram planning process to ensure proper bladder catheter positioning prior to the initiation of the examination.

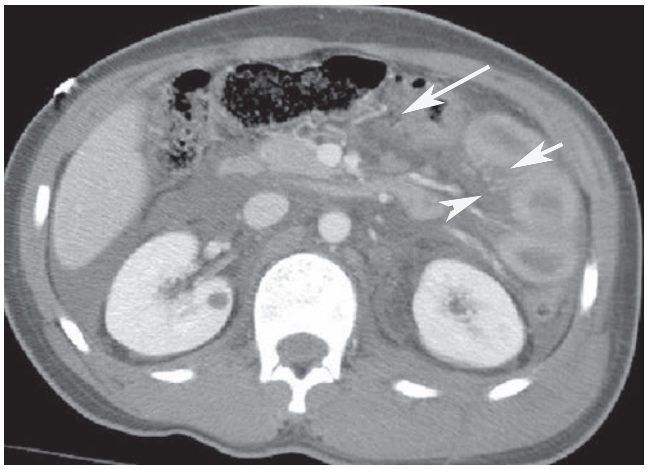
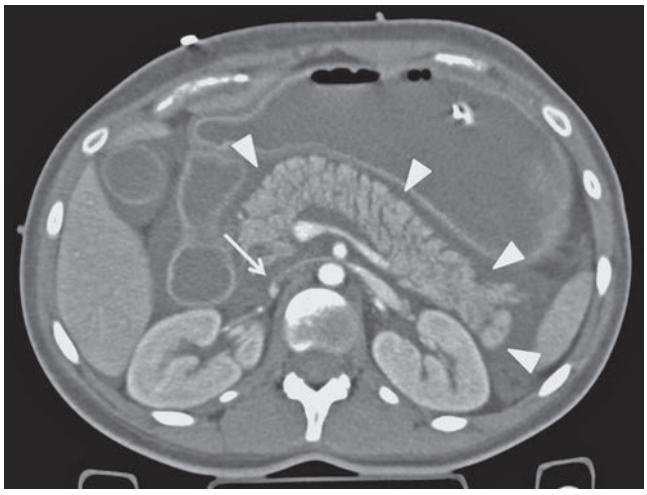


FIGURE 19-31 Small bowel perforation: A 14-year-old male, unhelmeted bicycle rider hit by a car sustained a small bowel perforation. Intravenous contrast-enhanced axial computed tomography shows three findings consistent with small bowel (jejunal) injury: (1) diffusely enhancing and thickened jejunum loops within the left side of the abdomen, with a focal hypoenhancing segment compatible with at least partial transmural injury (short arrow); (2) high-density interloop fluid within the mesentery adjacent to abnormal bowel (arrowhead) strongly suggesting transmural bowel laceration; and (3) small amount of pneumoperitoneum (long arrow) collecting within mesentery. Extra-alimentary air almost always correlates with transmural laceration of bowel.



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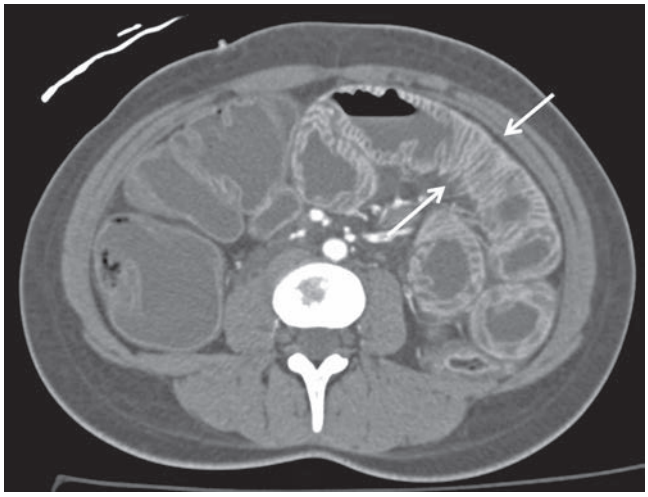

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FIGURE 19-32 Hypoperfusion shock complex/shock bowel syndrome. (**A**) Axial postcontrast computed tomography (CT) demonstrates a thin, slit-like inferior vena cava (arrow) and pancreatic edema (the pseudo-pancreatitis sign) with peripancreatic fluid (arrowheads). (**B**) Axial postcontrast CT from the same patient also demonstrates dilated bowel with thickened and intensely enhancing bowel wall (arrows). There is also diffuse mesenteric edema. Reduced splanchnic blood flow due to underresuscitation results in capillary leak and prolonged transit time for intravenous contrast. This constellation of findings is consistent with hypoperfusion shock complex/shock bowel syndrome.

their relation to each other and the femoral head. Assessment of the posterior through anterior walls, the ischium for the posterior column, the pubis and symphysis for evaluation of the anterior column, the iliac wing for superior extension, and congruence between the femoral head and acetabular cup allows for a more complete recognition of fracture fragments. There are 10 types of acetabular fractures based on the Judet and Letournel classification scheme. Fortunately, approximately 90% fall into one of five fracture patterns, divided into two groups based on the presence (both-column and T-shaped) or absence (transverse, transverse with posterior wall, and isolated posterior wall) of involvement of the obturator ring.[91]

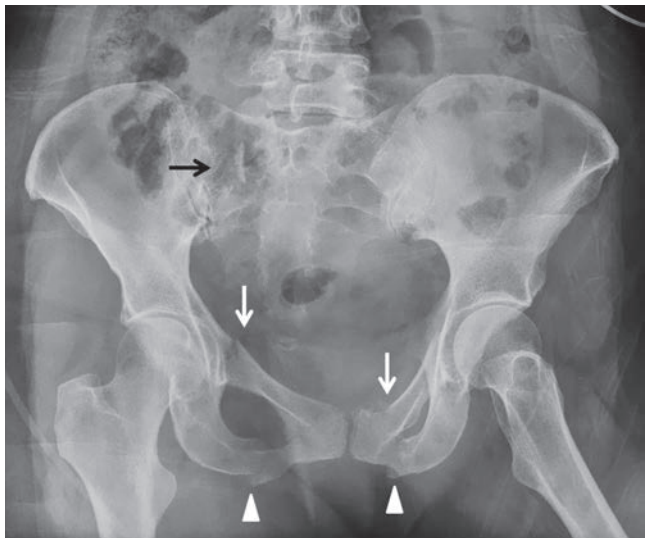
APPENDICULAR SKELETON

Conventional Radiography

Conventional radiography remains the imaging standard for the evaluation of long bones showing obvious deformity, instability, palpable crepitus, pain, and swelling. For periarticular regions, conventional x-rays are indicated for deformity, instability, decreased range of motion, pain, and swelling. The Ottawa ankle and knee clinical prediction rules add considerable precision to specificity.[92-94]

For long bones (Fig. 19-38), two orthogonal views are obtained, including an AP view and lateral projection centered at the midshaft. Projections should include both the proximal and distal joints. Joints should be imaged with two orthogonal views at a minimum, with additional optional one or two oblique views, centered at the midportion of the articulation (Figs. 19-38 to 19-43).

Analysis of the long bone should allow assessment of the direction of the force that created the fracture pattern (eg, twisting injuries result in spiral fractures; bending injuries result in wedge fractures). In general, higher-energy injuries



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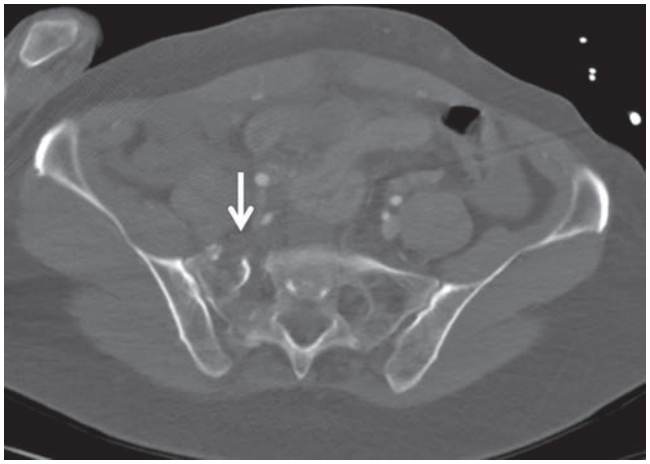

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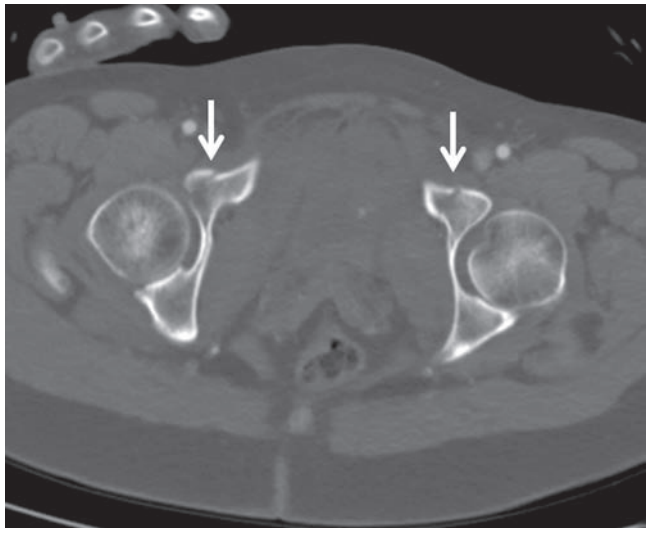

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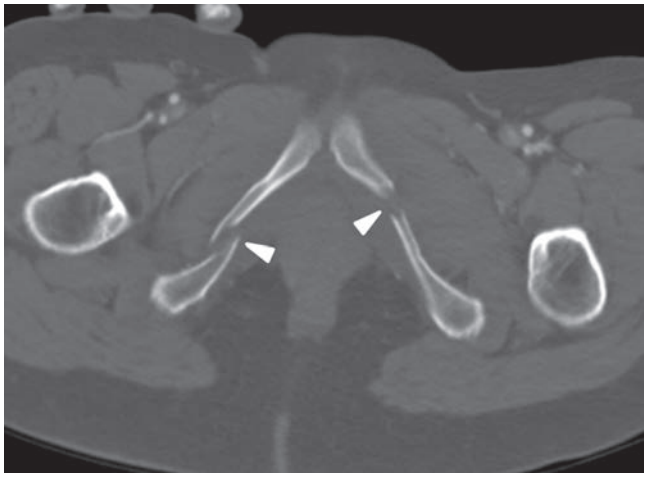

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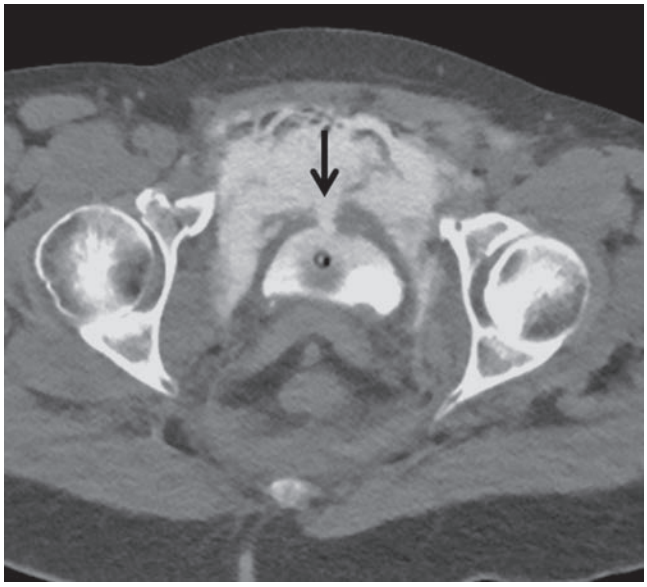

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FIGURE 19-33 Pelvic ring fracture: lateral compression type. A 54-year-old female passenger involved in a T-bone motor vehicle collision. (**A**) Anteroposterior (AP) radiograph of pelvis shows disruption of right sacral wing (black arrows), bilateral iliopubic rami (white arrows), and bilateral ischiopubic rami (arrowheads). (**B**) Axial computed tomography (CT) shows a comminuted fracture of the right sacral ala through the neural arches (arrow). Frequency of injury to sacral nerve roots is greatest when fractures involve medial aspect of the neural canal (Denis zone 3), lowest when lateral to the neuroforamen (Denis zone 1), and intermediate when involving neural foramina (Denis zone 2). (**C**) Axial CT image at the level of the femoral heads again demonstrates fractures of the superior iliopubic rami (arrows). (**D**) Axial CT image demonstrates fractures of the bilateral ischiopubic rami (arrowheads). (**E**) Subsequently performed CT cystogram demonstrates a defect in the anterior bladder wall (black arrow) with leak of contrast into the perivesicular space consistent with an extraperitoneal bladder rupture, an injury often seen in association with midline pelvic ring fractures.

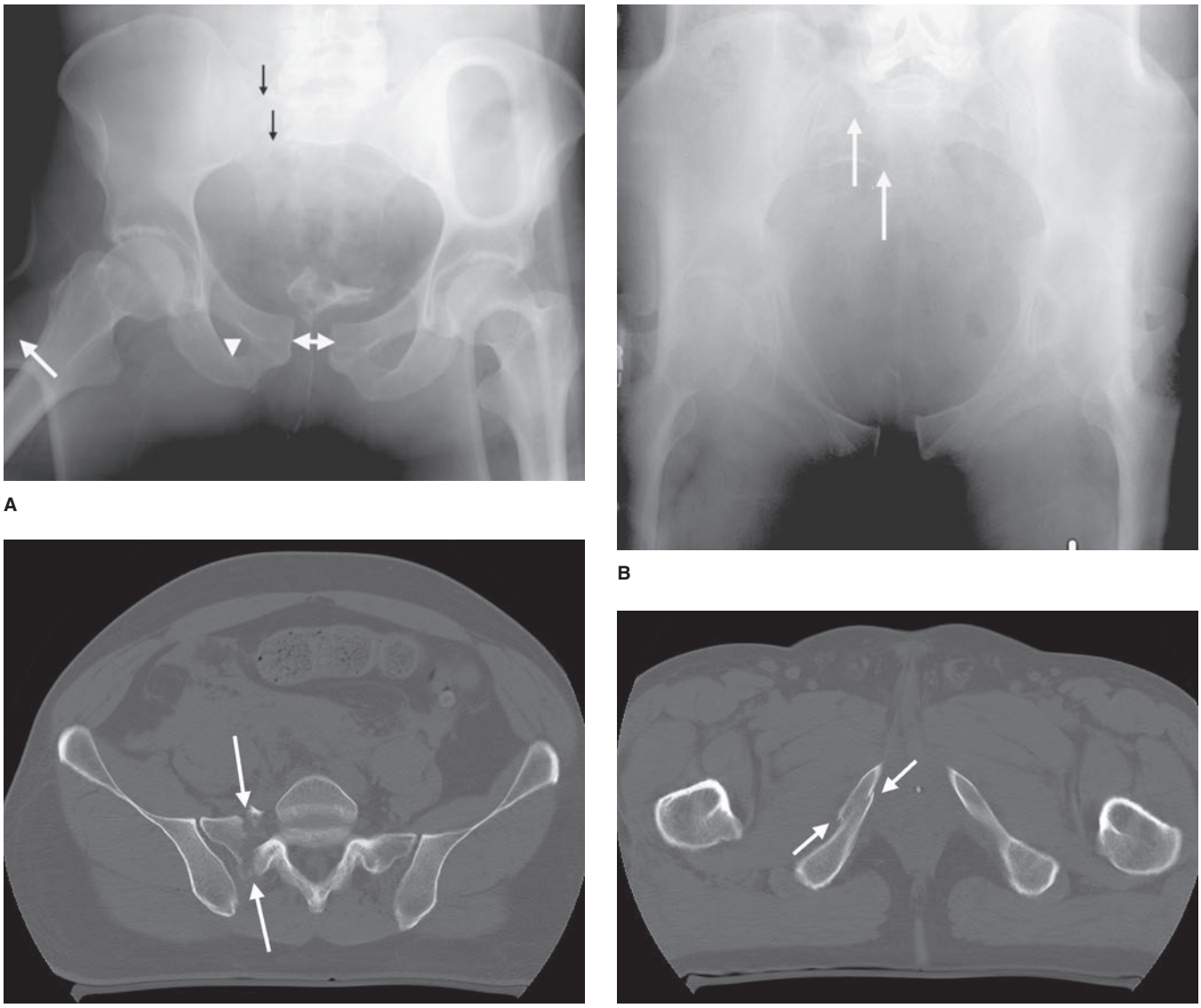


FIGURE 19-34 Pelvic ring fracture: anteroposterior (AP) compression type. This 55-year-old male sustained an injury during a 7-m fall onto concrete. (**A**) AP radiograph of pelvis shows symphyseal diastasis (double-ended arrow); right ischiopubic ramus fracture, which is minimally displaced (arrowhead); and disruption of right sacral arcuate lines (black arrows). Right femur is abducted (white arrow), a finding that is common with fractures of femoral shaft that this patient also sustained. (**B**) Inlet view of pelvis (obtained with 45° angulation caudally) better shows disruption of arcuate lines (white arrows) and again shows pubic symphyseal diastasis. (**C**) Axial computed tomography (CT) at the lumbosacral junction shows through-and-through fracture (arrows) of right lateral mass of S1 with 6 mm of lateral and 8 mm of anterior translation. (**D**) Axial CT image at ischial tuberosities shows oblique sagittal fracture through right ischial pubic ramus (white arrows). Orientation of ischial fractures often reflects mechanism injury (sagittal plane fractures due to AP compression or vertical shear; transverse or axial plane fractures due to lateral compression).

tend to be more comminuted and displaced. If there is a mismatch between the apparent amount of comminution and the reported energy of the injury, osteoporosis or otherwise pathologic bone should be suspected. The degree of displacement and angulation of the predominant fracture fragments, as well as fracture line involvement of an articular surface, must be noted.

Periarticular regions should be evaluated for fracture involvement as well as for partial or complete loss of congruity of the joint. The appearance of a superimposed white line due to overlapping of bones may indicate a dislocation and disruption of expected alignment of adjacent articulating structures (Fig. 19-38E).

Careful attention to soft tissues (eg, focal swelling, obliteration of normal fat pads, joint effusions) is helpful for subtle or otherwise occult fractures (eg, elbow, knee, and wrist). A search for foreign bodies in soft tissue in the setting of an open fracture or soft tissue injury should be performed.

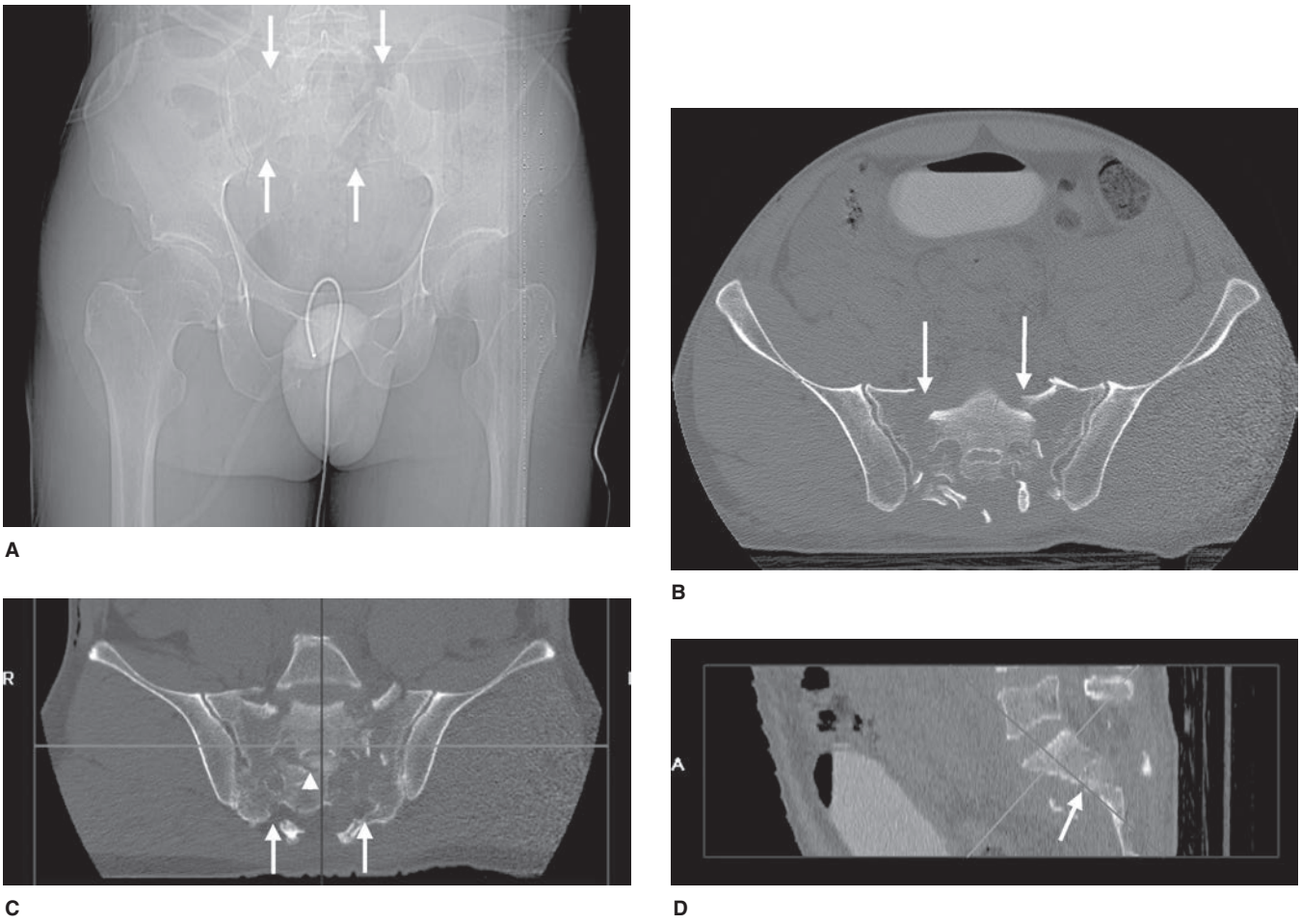


FIGURE 19-35 Vertical shear injury with unstable sacral fracture. An H-shaped sacral fracture was sustained in 40-ft fall. This was associated with right calcaneus and T12 compression fractures. (**A**) Anteroposterior (AP) pelvis computed tomography (CT) scout shows disruption of arcuate lines bilaterally (arrows). Such a finding requires excellent lateral view of sacrum to exclude transverse components of the fracture to create either H- or U-shaped sacral fractures, which are typically biomechanically unstable. This can rarely be obtained. CT better evaluates this area as coronal reformations of thin-section axial CT. (**B**) Axial CT shows bilateral through-and-through sacral fractures (arrow) that are transforaminal in their course. At this level, no transverse fracture is appreciated. (**C**) Coronal oblique CT reformation shows bilateral lateral mass fractures (arrow), as well as a portion of transverse fracture (arrowhead). (**D**) Sagittal CT reformation clearly shows transverse fracture (arrow).

Computed Tomography of Appendicular Joints

CT of appendicular joints, particularly the shoulder, tibial plateau, pilon, midfoot, and calcaneus, is indicated for "displaced" intra-articular fractures (eg, 1–2 mm at the wrist, scapula, or glenoid; 5–10 mm at the tibial plateau) or unstable fracture patterns (Figs. 19-39 and 19-41 to 19-43). CT is a valuable surgical planning tool and is very helpful in the detection of otherwise occult fractures, particularly of the midfoot.

In most patients, CT should be performed after provisional placement of traction or reduction, if feasible. Use of traction prior to imaging allows ligamentotaxis to indirectly reduce fracture fragments and support indirect assessment of the integrity of soft tissue attachments to major bony fragments. Specifically, bone fragments that do not move or reduce on stretch are presumed to be no longer attached to soft tissue and may require debridement or direct repositioning.

Because acquisition of isotropic image data of modern MDCT scanners allows for reconstruction of images in any conceivable plane, the joint in question no longer needs to be precisely positioned in the CT scanner gantry. Thus, "axial" images of a joint are not necessarily oriented in the axial plane of the body, but instead are oriented in the "axial" plane of the joint. Orthogonal images in both the sagittal and coronal planes are reconstructed from the joint-specific axial plane, usually at 1- to 2-mm slice thickness using a bone algorithm.

Peripheral Vascular Injuries

Vascular injuries in the extremities compose between 40% and 75% of vascular injuries observed in civilian trauma

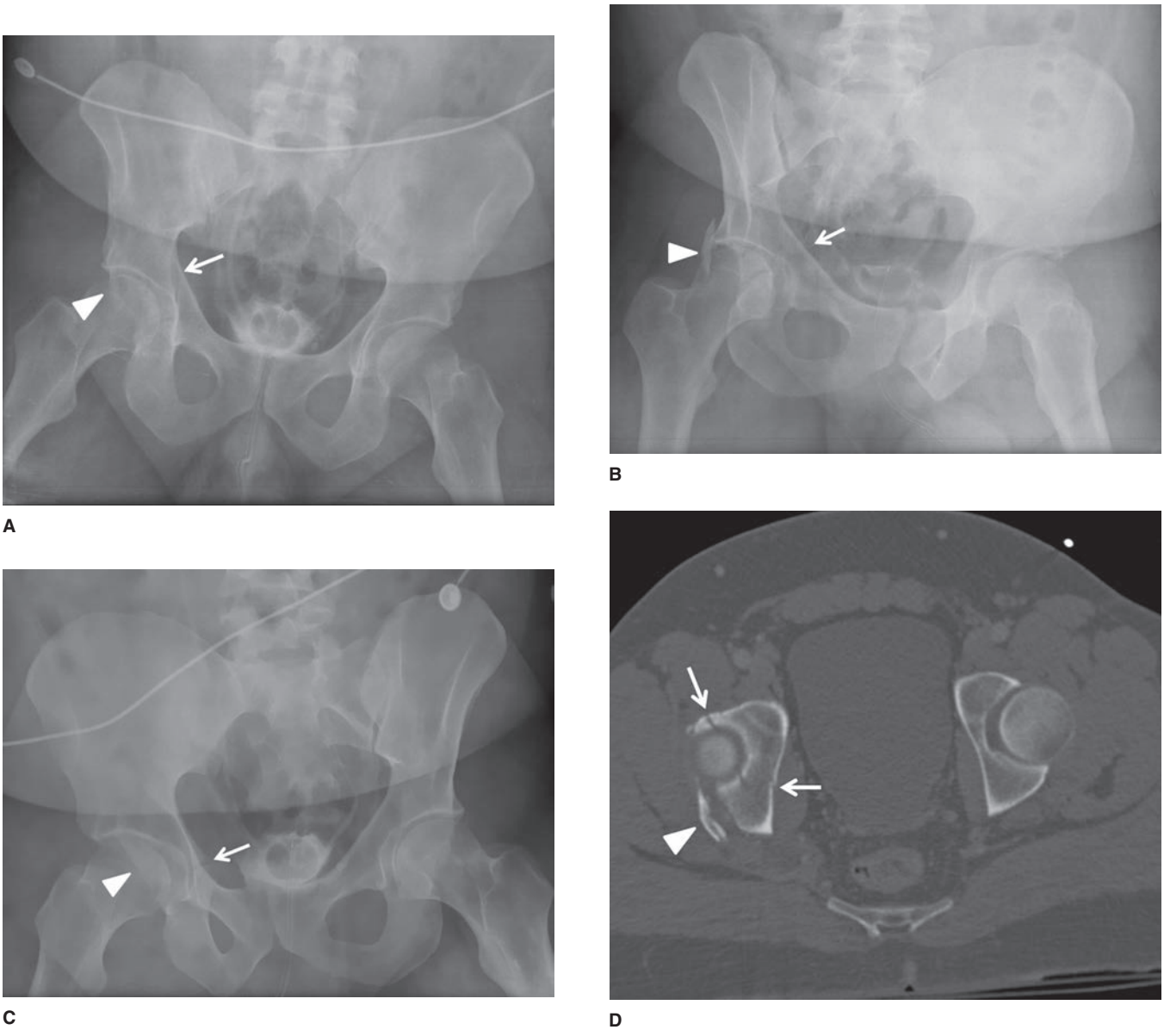
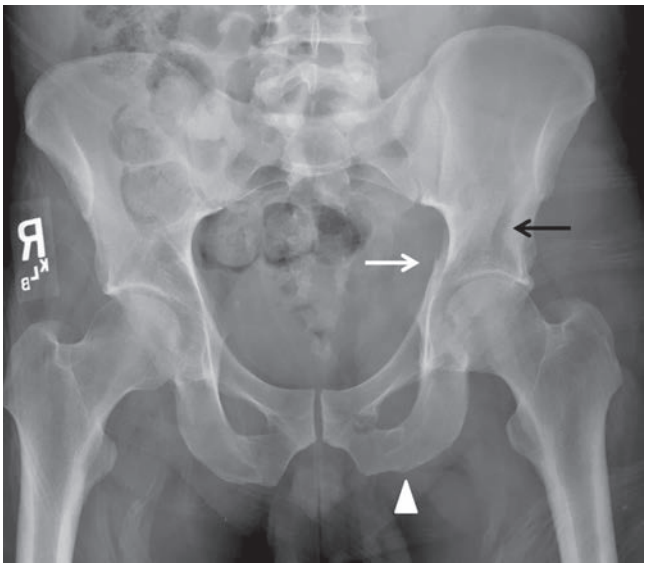


FIGURE 19-36 Transverse acetabular fracture with associated posterior wall fracture. (**A**) Anteroposterior (AP) view of pelvis shows disruption of iliopectineal line adjacent to acetabulum (arrow), with an eyebrow-shaped radiodensity projecting over the femoral head (arrowhead), which is a displaced posterior wall fragment. Bilateral Judet (**B, C**) oblique radiographs again show transverse fracture (arrow) and a posterior wall fragment (arrowhead). (**D**) Axial computed tomography (CT) image at the level of the acetabular roof shows a sagittal plane fracture (arrows) characteristic of transverse fractures of acetabulum. The posterior wall fracture fragment is again seen (arrowhead).

centers, with a majority of these (approximately two-thirds) involving the femoral or popliteal arteries. Approximately three-fourths of peripheral vascular injuries are the result of penetrating trauma, with 70% to 80% secondary to projectiles such as gunshot wounds and shrapnel. Fatal exsanguination, multiorgan failure secondary to hemorrhagic shock, and limb loss are catastrophic consequences of peripheral vascular injuries; thus, rapid identification and appropriate triage are of paramount importance. Vigorous or pulsatile external active hemorrhage, a rapidly expanding hematoma, and loss of distal pulses mandate emergent operative exploration.[95]

The imaging strategy for patients with suspected peripheral vascular injuries depends on a variety of factors that are primarily related to clinical presentation, hospital course, associated injuries, mechanism of injury, and signs of circulatory shock. Imaging modalities available include catheter angiography, CTA, Doppler ultrasound, and MR angiography (MRA).



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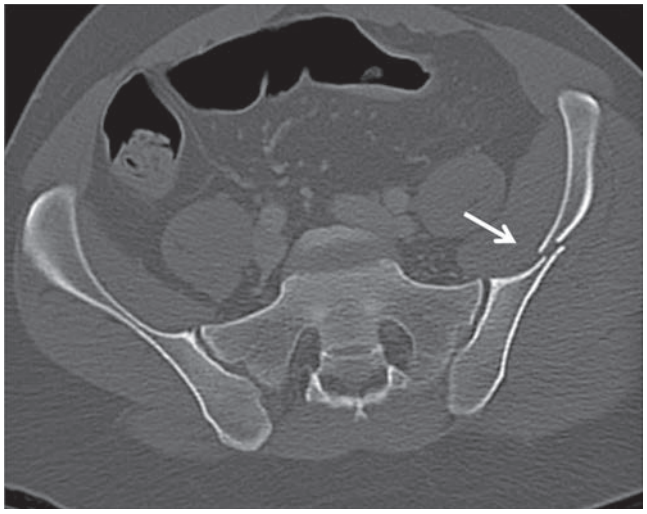

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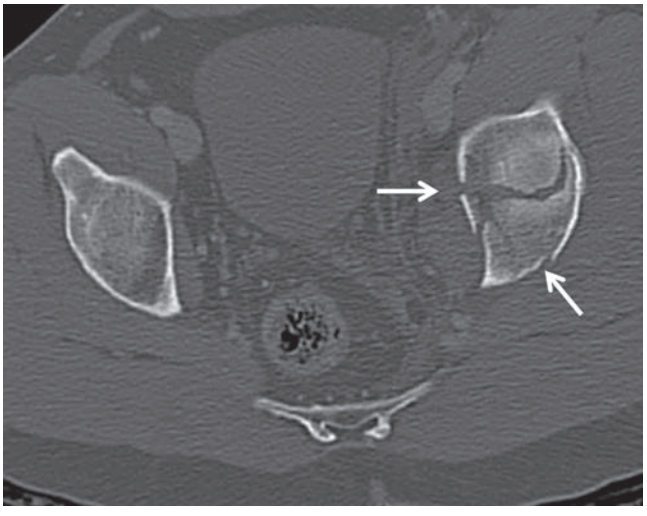

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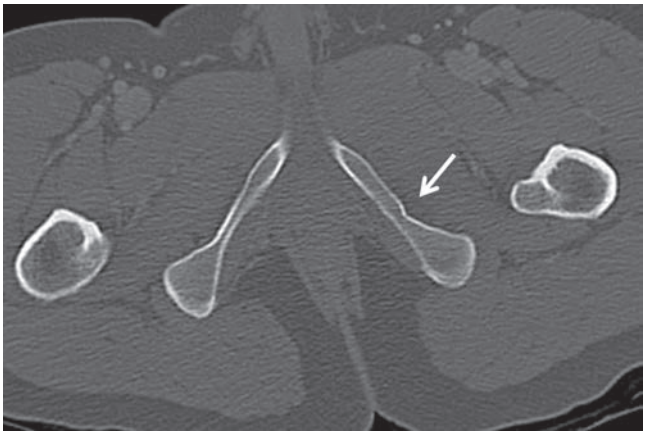

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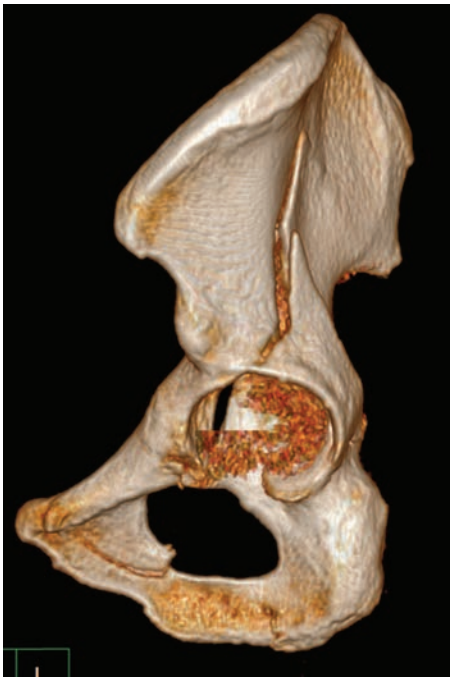

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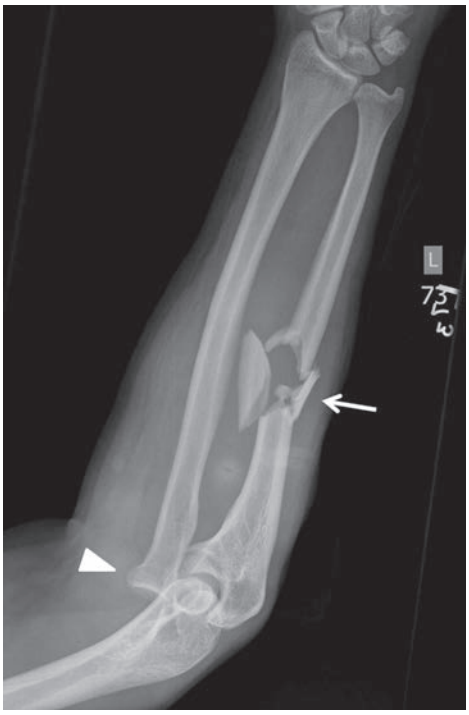
FIGURE 19-37 Both-column acetabular fracture. A 34-year-old male with left acetabular fractures following a motor vehicle collision. (**A**) Anteroposterior (AP) conventional radiograph of pelvis shows disruption of both iliopectineal and ischiopubic lines (arrow) and disruption of left ischiopubic ramus (arrowhead). There is a vertical lucency of the left iliac wing just superior to the acetabulum compatible with a fracture (black arrow). Axial computed tomography of the pelvis viewed using bone windows (**B–D**) demonstrates a fracture of the left iliac wing (**B**, arrow), a comminuted fracture of the left acetabulum (**C**, arrows), and a fracture of the left ischiopubic ramus (**D**, arrow). These fractures together are characteristic of a both-column acetabular fracture. Three-dimensional surface-rendered image (**E**) with femoral head subtraction nicely demonstrates the fracture fragments with respect to the acetabular cup.



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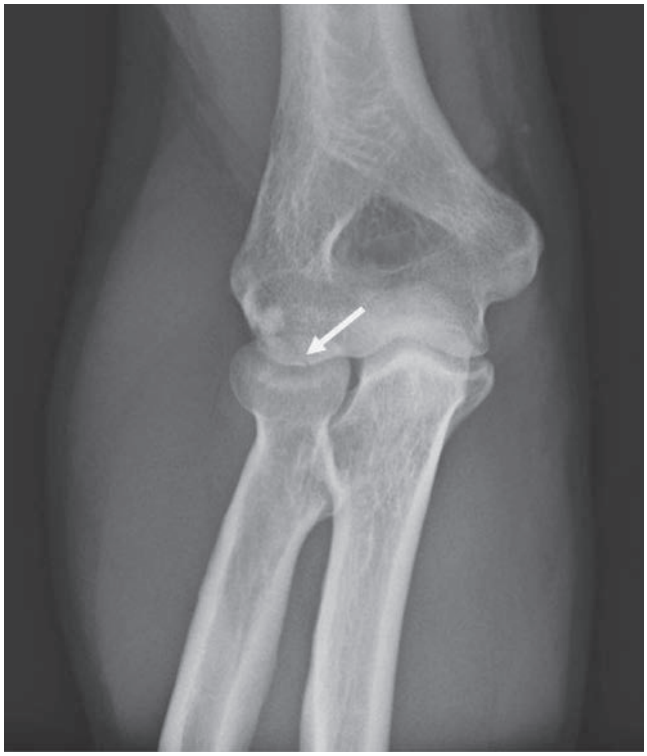

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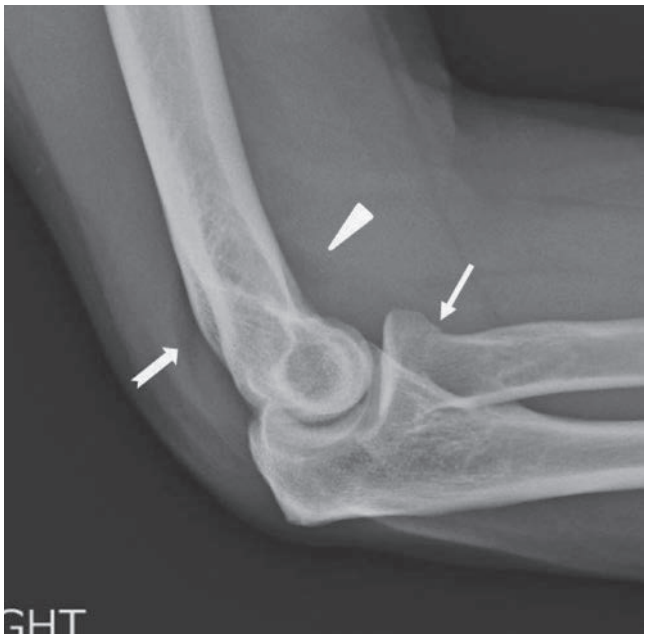

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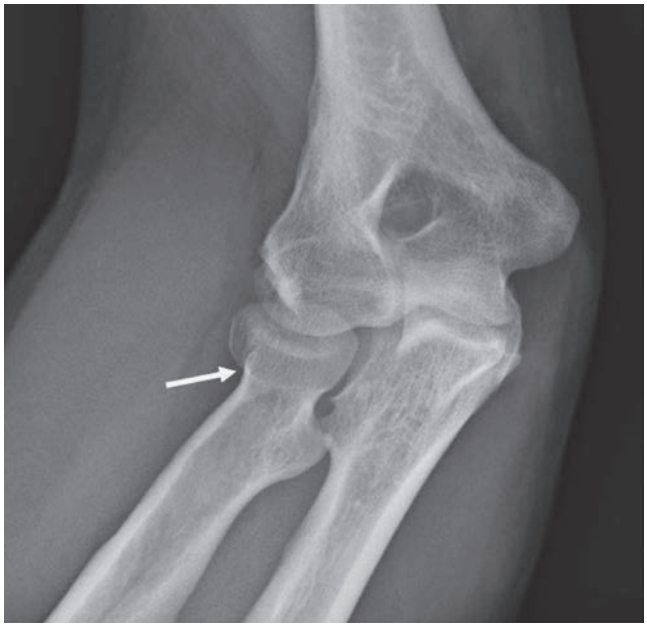

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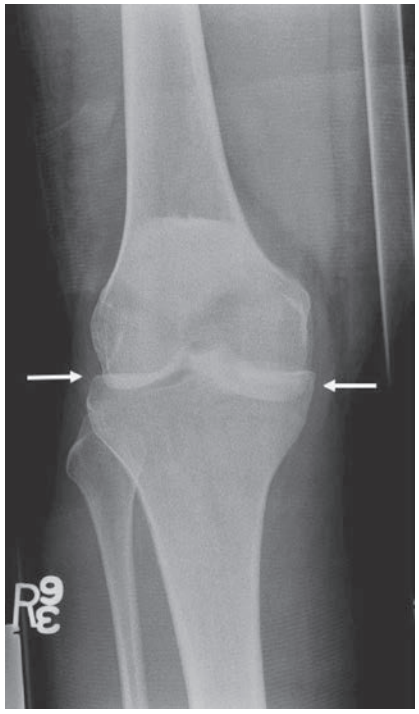
FIGURE 19-38 Extremity injuries. (**A**) A 33-year-old man with a Monteggia fracture. Lateral radiograph of the forearm demonstrates a comminuted fracture of the mid ulnar diaphysis (arrow) with associated anterior dislocation of radius at radiocapitellar articulation (triangle). (**B–D**) A 38-year-old man with elbow pain status post fall. Anteroposterior (AP) (**B**), lateral (**C**), and oblique (**D**) images demonstrate a fracture line through the radial head (arrow). There is elevation of the anterior fat pad (triangle) and posterior fat pad (notched arrow) indicating hemarthrosis.



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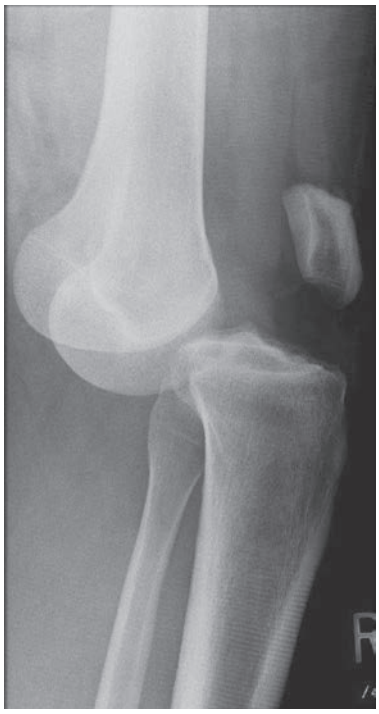

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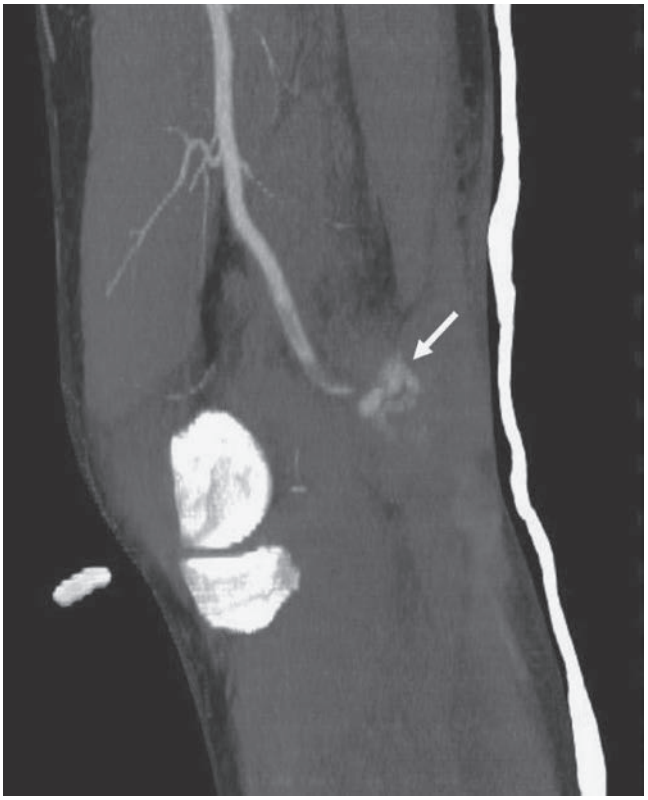


G

FIGURE 19-38 (*Continued*) (**E–G**) A 25-year-old man with knee dislocation after falling 25 ft. AP (**E**) knee radiograph demonstrates overlap and increased density of the femoral condyles and tibial plateau (arrows). Lateral (**F**) knee radiograph demonstrates anterior dislocation of the tibia-fibula with respect to the distal femur. Postreduction sagittal computed tomography angiogram image (**G**) demonstrates popliteal artery transection with active bleeding (arrow).

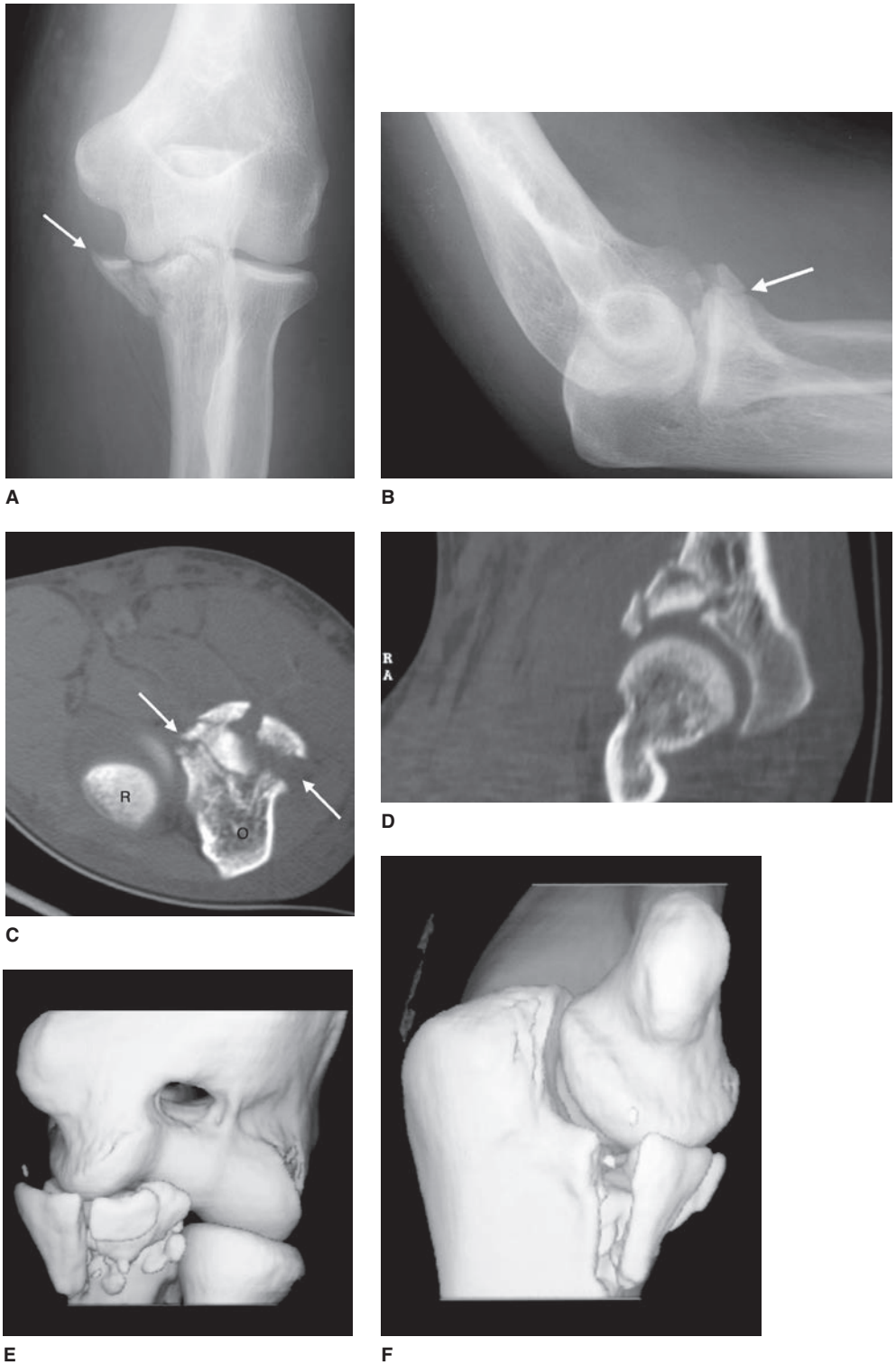


FIGURE 19-39 Periarticular fracture: coronoid process fracture of elbow. (**A**) Anteroposterior (AP) radiograph of left elbow shows displaced coronoid process (arrow). Elsewhere, joint appears congruent. (**B**) Lateral view of left elbow shows tip of coronoid process fracture (arrow). Coronoid process fractures can be graded by amount of coronoid process involved, such that larger coronoid process fracture fragments are more likely to result in elbow instability. (**C**) Axial computed tomography (CT) obtained because mismatch between radiographic and clinical findings of instability shows highly comminuted fracture of coronoid process (arrows). Radial head (R) and olecranon process (O) appear normal. (**D**) Sagittal CT reformation shows nearly all the coronoid process is involved in fracture. Secondary congruence between trochlea and olecranon-coronoid process is fair. (**E, F**) Three-dimensional surface-rendered CT reformations more graphically demonstrate transverse and distal extent and displacement of coronoid process fracture.

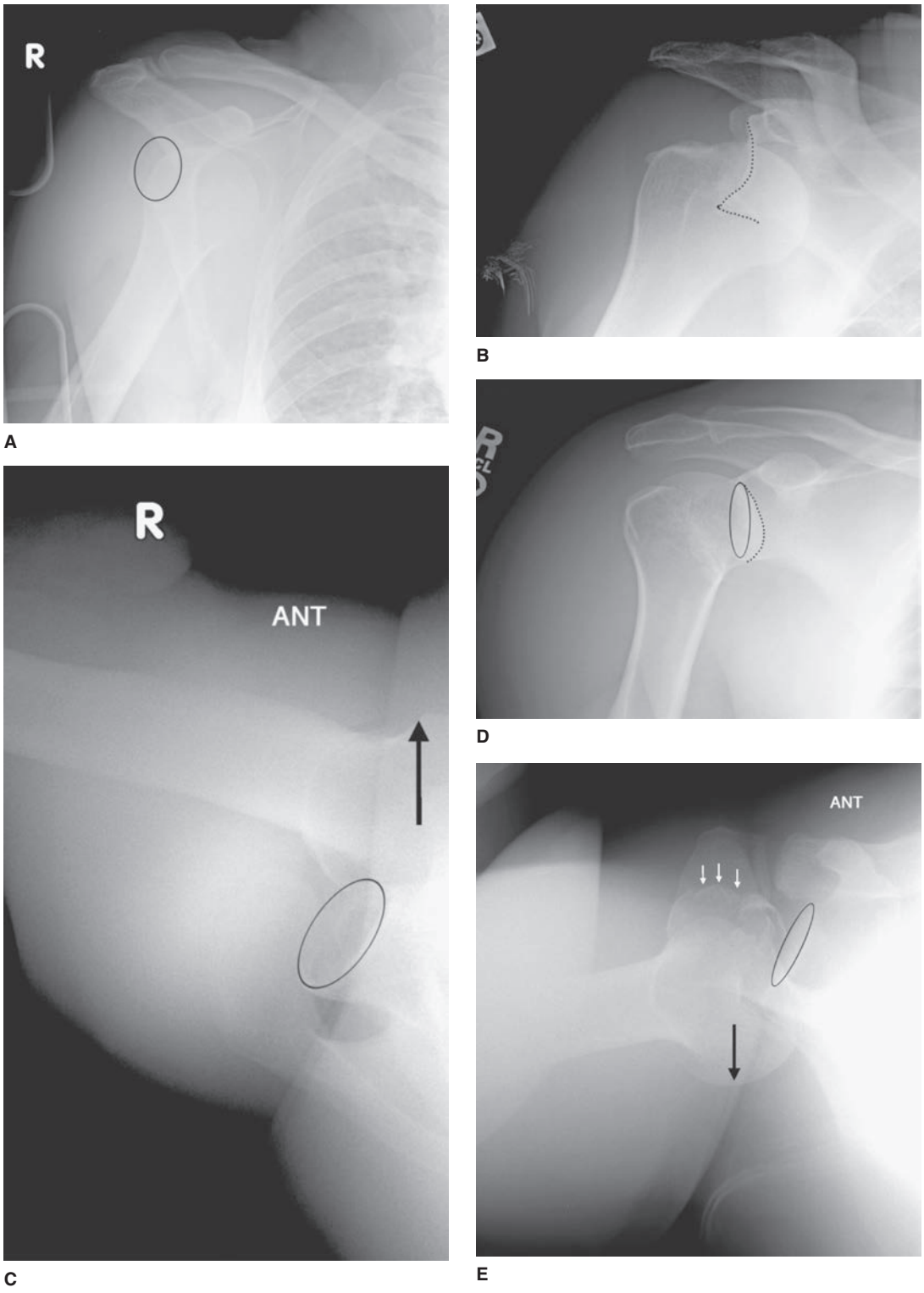


FIGURE 19-40 Periarticular injury: shoulder dislocation. (**A–C**) Anterior dislocation sustained by a 52-year-old struck by a falling tree on his back. (**A**) An anteroposterior (AP) radiograph with medial location of humeral head relative to glenoid (circle). (**B**) Postero-oblique radiograph of humeral head and glenoid (dotted line). Note that amount of overlap of scapula is less on this posterior oblique view than it is on anterior view (**A**), characteristic of anterior dislocation. (**C**) An axillary view; shows anterior location of humeral head relative to glenoid (upward pointing arrow and circle, respectively) on axillary lateral view. (**D, E**) Posterior dislocation sustained in a 42-year-old man who fell during a seizure. Posterior oblique radiograph (**D**) shows overlap of glenoid (circle) and medial aspect of humeral head (dotted line). (**E**) An axillary projection that shows the location of the humeral head posterior to glenoid (oval). Posterior margin of the head is denoted by downward pointing black arrow. Three downward pointing white arrows show impaction on anterior margin of humeral head, so-called trough fracture or reverse Hill-Sachs deformity.

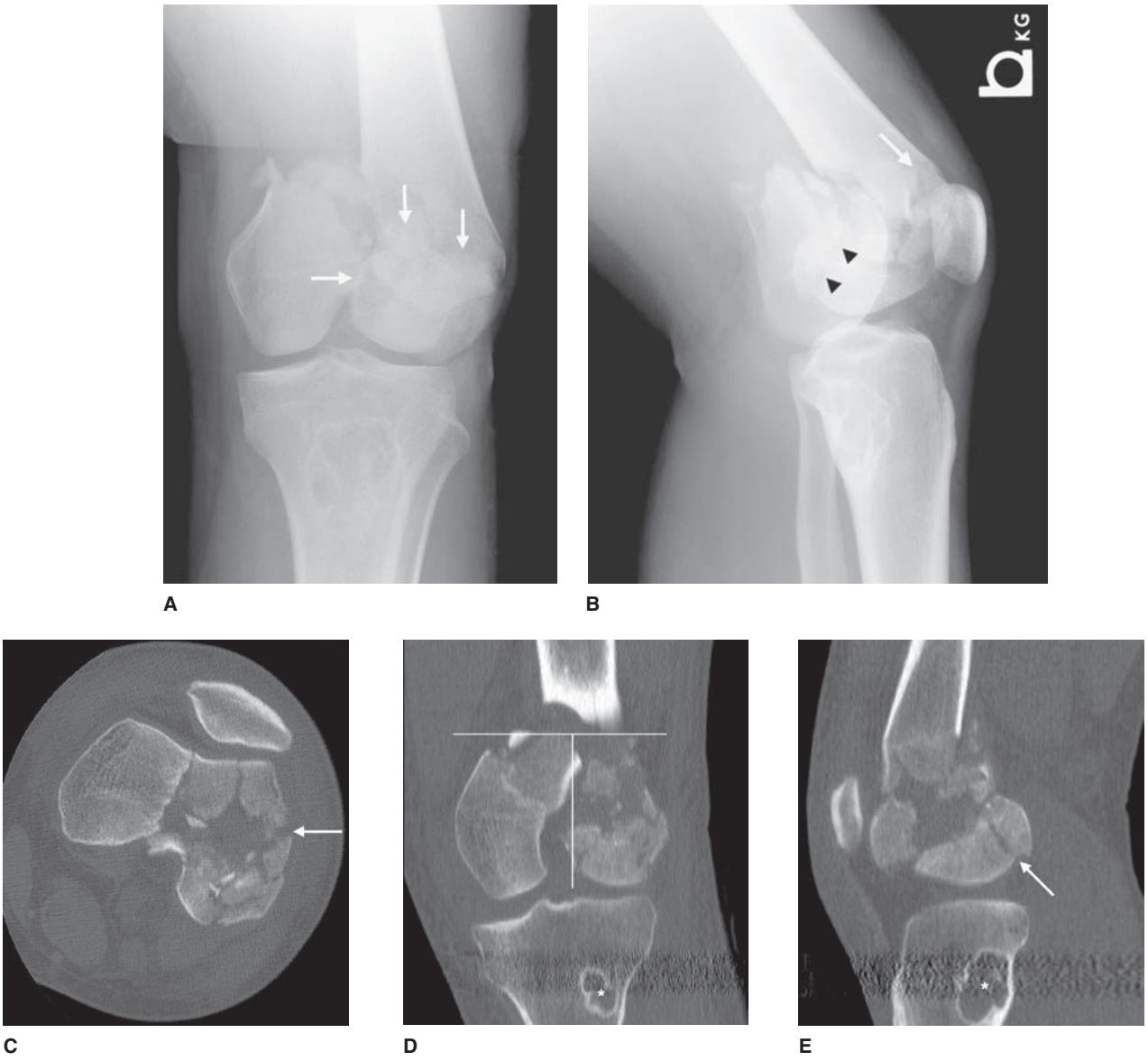
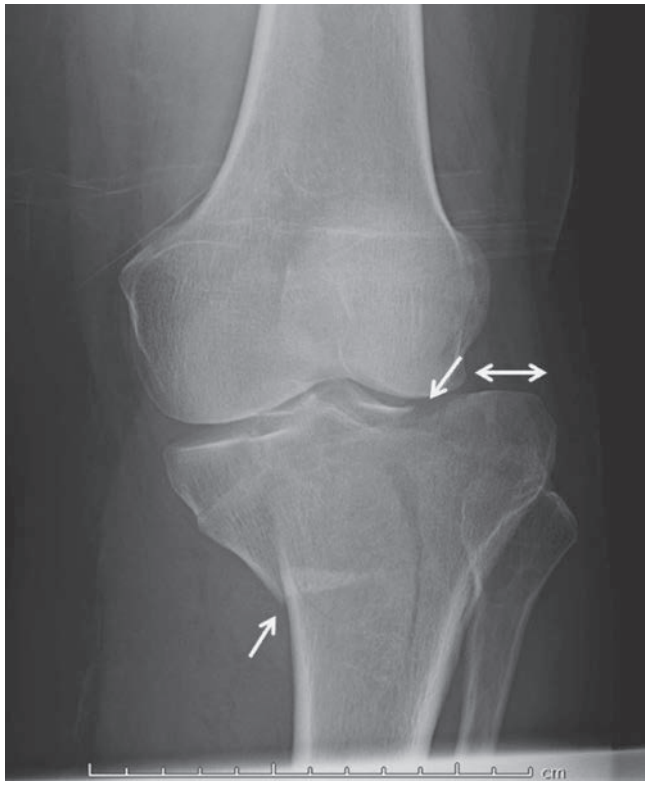


FIGURE 19-41 Periarticular fractures: intra-articular intercondylar distal femur fracture. This 20-year-old man was involved in a high-speed motor vehicle crash as a belted driver. (**A**) Anteroposterior (AP) radiograph of knee shows transverse T-type fracture of distal supracondylar femur, with intra-articular extension into intercondylar notch (arrows). (**B**) Lateral radiograph of knee shows transverse supracondylar component (arrow), from which femoral condyles have dissociated. In addition, lateral femoral condyle shows coronal plane, comminuted fracture of posterior aspect of condyle (arrowheads). In up to 40% of intra-articular intracondylar fractures caused by high-energy mechanisms, such coronal plane fractures (Hoffa fracture) may be overlooked. (**C**) Axial computed tomography (CT) shows a sagittal plane fracture extending into midportion of trochlea of the patellofemoral joint and a comminuted coronal plane fracture of posterior aspect of lateral femoral condyle (arrow). (**D**) Coronal plane reformation from axial CT shows T-type intra-articular fracture with dissociation of medial and lateral femoral condyles (white lines). Asterisk marks developmental variant, nonossifying fibroma. (**E**) Sagittal reformation from axial CT in central portion of lateral knee joint compartment shows coronal plane fracture of posterior femoral condyle (Hoffa fracture), as marked by arrow. Asterisk notes nonossifying fibroma, a benign developmental variant.

Doppler ultrasound may be a useful adjunct but is not widely used, primarily because of its decreased sensitivity and accuracy for peripheral vascular injuries when compared to catheter angiography. This, coupled with the operator-dependent nature of the examination and limited visualization of the entire vascular system for a variety of reasons (eg, external fixator devices, overlying split or cast material, and associated soft tissue injuries and gas), makes the utility of Doppler ultrasound for peripheral vascular injuries quite limited.

MRA is useful for the assessment of chronic peripheral vascular disease but is not widely used in the acute setting for the following reasons: (1) it may not be available at all centers at all times of the day; (2) it is technically challenging



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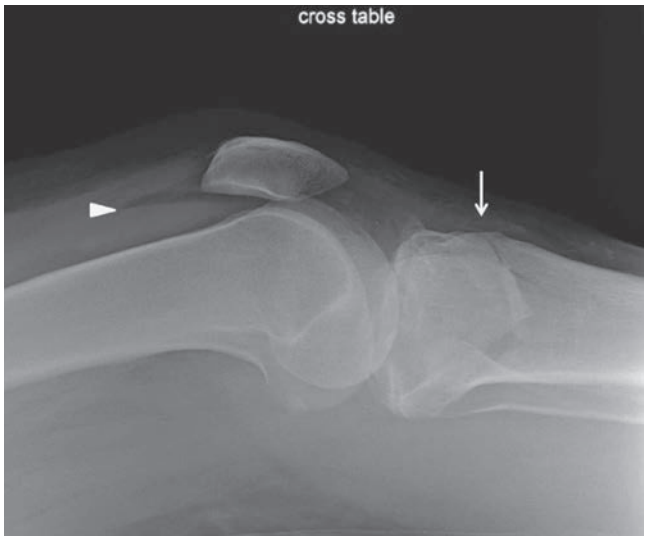

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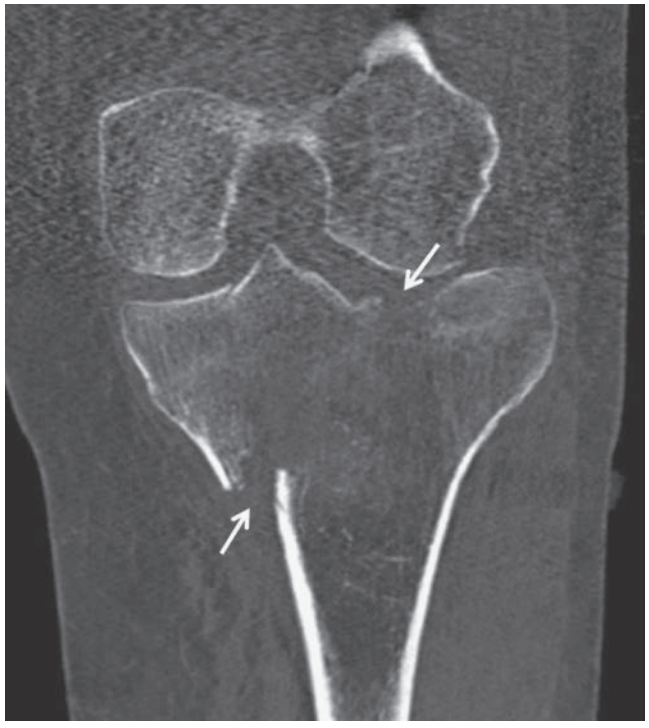

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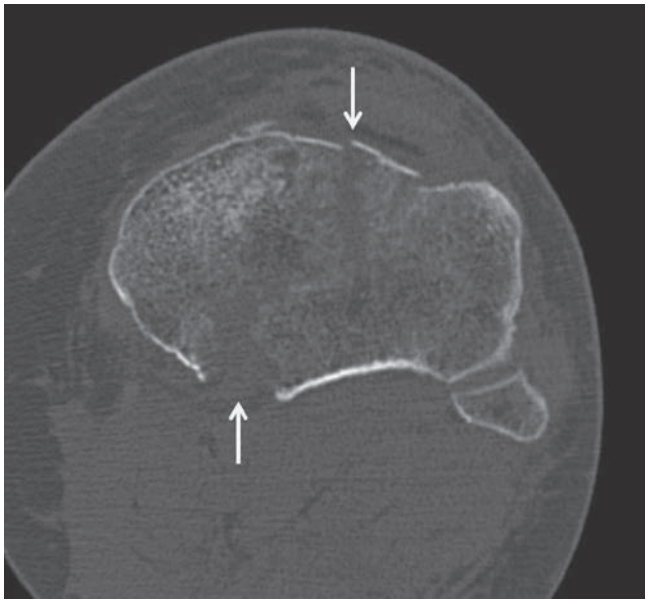
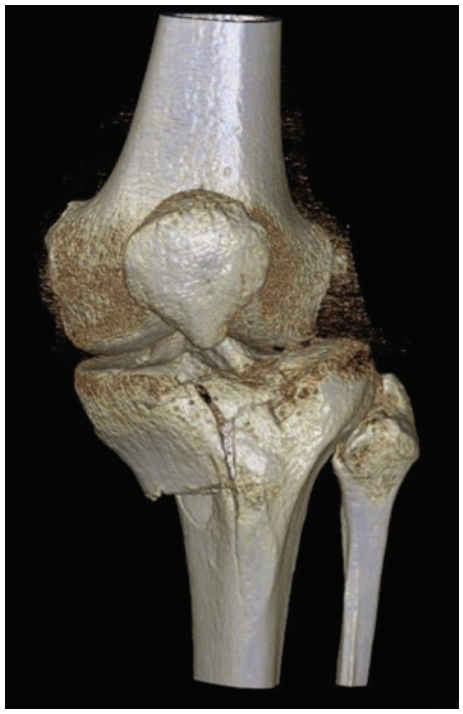


D

FIGURE 19-42 Periarticular fractures: tibial plateau fracture (Schatzker 4). This is a 57-year-old woman who was hit by a car. (**A**) Anteroposterior (AP) radiograph of the knee shows a comminuted oblique fracture through the tibial plateau extending into the medial cortex (arrows). There is lateral subluxation (double arrow) of the tibial plateau with respect to the femoral condyles. (**B**) Cross-table lateral radiograph demonstrates the tibial plateau fracture (arrow) and lipohemarthrosis (triangle). The presence of a fat fluid level in the suprapatellar joint space indicates that the fracture line extends to the tibial plateau surface, thus liberating the fatty elements of the bone marrow into the joint space. Coronal (**C**) and axial (**D**) computed tomography (CT) images demonstrate the extent of intra-articular comminution and persistent fracture fragment subluxation despite closed reduction.



E

FIGURE 19-42 (*Continued*) (**E**) Three-dimensional surface-rendered CT image nicely demonstrates the fracture lines and relationships between the primary fracture fragments.

to effectively manage severely traumatized patients while they are in the magnetic resonance magnet; (3) the presence of metallic foreign bodies may limit patient and operator safety; and (4) even if not ferromagnetic, foreign bodies may still cause extensive artifact, obscuring the adjacent soft tissues. Thus, CT or catheter angiography is the primary imaging choice for patients with extremity injuries and suspected peripheral vascular injuries.

CTA FOR SUSPECTED PERIPHERAL VASCULAR INJURIES

CTA is an extremely useful adjunct for suspected peripheral vascular injuries in patients without indications for immediate surgical exploration. It is readily available, fast, convenient, noninvasive, and reliable. In addition, CTA can be easily integrated into the imaging workup of a traumatized patient, performed either as a stand-alone examination or integrated as part of a whole-body CT for polytrauma. CTA has high sensitivity, specificity, and accuracy when compared to catheter angiography (which remains the reference gold standard). In addition, CT capabilities allow for global evaluation of the adjacent structures and can thus be used to detect and characterize associated nonvascular injuries. The inability to immediately perform interventions on an injured vessel is a major limitation of CTA compared to catheter angiography.

The overall performance of CTA for peripheral vascular injuries compared to catheter angiography is certainly a concern for the trauma surgeon, as there is very little tolerance for missed or mischaracterized vascular injuries. A meta-analysis by Jens et al[96] pooling data from 11 studies from both the radiology and surgical literature and using a variety of scanner types documented that peripheral CTA for extremity vascular injuries had a sensitivity of 96.2% and specificity of 99.2% compared to catheter angiography. The rate of nondiagnostic CTA examinations was 4.2% in this meta-analysis. Thus, in patients without indications for immediate surgical exploration, CTA is useful to exclude peripheral vascular injuries and to characterize injuries that are present.

The CTA signs of a vascular injury include pseudoaneurysm, active bleeding, occlusion, intimal injury, dissection, and arteriovenous fistula (Fig. 19-38G). Imaging findings that raise the likelihood of an injury include a perivascular hematoma, a vessel within a projectile wound tract, and projectile fragments within 5 mm of a vessel. The latter two findings should typically prompt further investigation with catheter angiography.

Extremity CTA should be performed using MDCT with thin-section acquisition, peripheral IV access or a power injectable central line, and a high contrast injection rate (4–5 mL/s) and should be acquired during the systemic arterial phase. Images are reconstructed in the true axial plane to the long axis of the extremity at 1- to 3-mm slice thickness, with additional sagittal and coronal images reconstructed with respect to the axial plane. Coronal MIPs (or thick slab images) at 5- to 10-mm slice thickness are extremely valuable in displaying vessels along their long axis using a relatively small number of slices. Thin images should be reviewed as well, because small intimal vascular injuries may not be apparent on MIP images due to volume averaging. Liberal use of a 3D volume viewing station is encouraged, as some vascular injuries may be best displayed on nontraditional planes.

It should be noted that vascular injuries near highly mobile joints, such as the knee, are poorly treated with endovascular techniques. Thus, the decision to image with CTA over catheter angiography must be weighed against patient stability and the need for urgent surgical vascular repair. Despite this, the wide availability of stent grafts has also increased the utilization of angiography as a prelude to nonoperative management of some clinically significant vascular injuries.

CATHETER ANGIOGRAPHY

Catheter angiography (whether based in the radiology suite, operating room, or a hybrid operating theater) remains the reference gold standard for the definitive evaluation of arterial blood vessels for injury, as well as identifying active arterial hemorrhage, pseudoaneurysms, and arteriovenous fistulas. Although CTA is frequently favored for the initial imaging of many traumatic vascular lesions in stable patients, it is limited

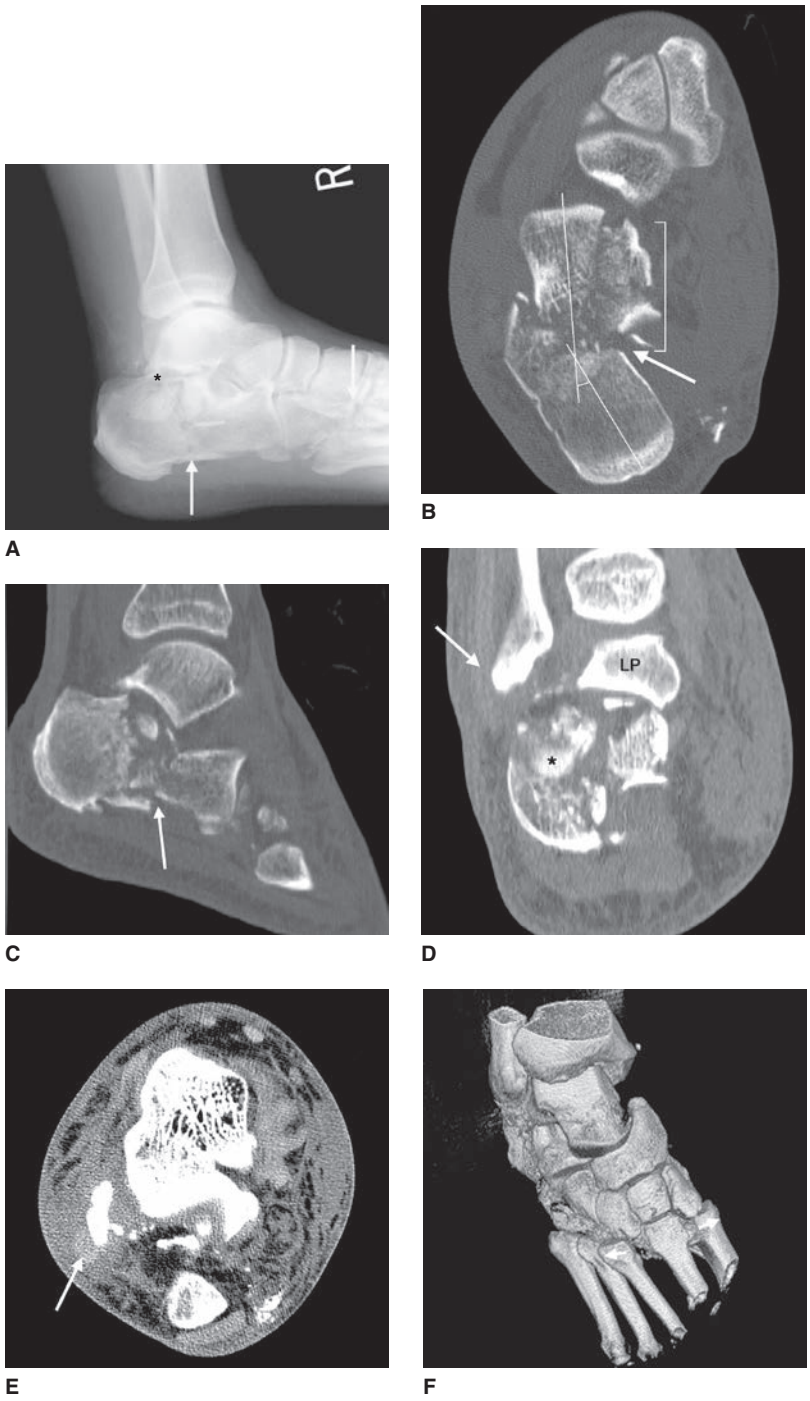


FIGURE 19-43 Periarticular fractures: calcaneus and Lisfranc fractures of midfoot. This 51-year-old restrained driver in a high-speed motor vehicle crash sustained multiple extremity and torso injuries. (**A**) Lateral conventional radiograph shows intra-articular fracture of calcaneus (upward pointing arrow denotes primary fracture plane; asterisk shows double density of central lateral fragment of posterior subtalar joint of calcaneus). Downward pointing arrow shows displacement of one of the metatarsal bases with an adjacent cuneiform fracture. (**B**) Axial computed tomography (CT) image at level of base of sustentaculum tali shows varus deformity through primary fracture (arrow). Secondary fracture plane extends toward anterior process (bracket). It is important to note continuity of cortex of medial wall of anterior process, as it influences distal extent of necessary fixation. (**C**) Sagittal reformation from axial CT shows primary fracture plane (upward arrow) with centrolateral fragment rotated into its superior extent. (**D**) Coronal reformation shows comminuted fracture of posterior facet of calcaneus due to bursting of body by lateral process (LP) of the talus. Centrolateral fragment is shown by asterisk. White arrow denotes lateral dislocation of peroneal tendons from peroneal groove in posterior fibula. (**E**) Axial CT at level of sinus tarsi, soft tissue window, shows lateral and anterior dislocation of peroneal tendons surrounded by hemorrhage and edema (white arrow). (**F**) Three-dimensional reformation from axial CT, medial oblique projection, shows divergent dislocations of great toe and third to fifth metatarsal bases (arrows).

by the inability to immediately intervene in the event of a positive study. CTA performance is very high, but not yet as accurate as catheter angiography. Catheter angiography is used as a problem-solving tool for equivocal CT examinations and for definitive therapy of some acute vascular conditions.

There are many advantages of catheter angiography. It allows simultaneous detection and treatment of a wide variety of traumatic vascular injuries. It is a very specific method of identifying bleeding at the submillimeter diameter of vessel. It can evaluate many sites of bleeding simultaneously. It has an excellent safety record, especially when using iso-osmolar nonionic contrast agents, coaxial micropuncture access, digital subtraction techniques, coaxial microcatheters, and steerable guidewires.

The disadvantages of catheter angiography are cost, the delay necessary to assemble the interventional radiology team (composed of radiologists, technologists, and nurses), the lack of suitability as a screening test for most traumatic conditions, and the risks of radiation exposure. Technical expertise is limited to predominantly subspecialty-trained interventional radiologists. These disadvantages are magnified when the likelihood of injury is low. Thus, noninvasive vascular techniques such as CTA should be entertained when considering the patient's overall management.

Transcatheter Endovascular Therapies

Endovascular techniques have become a broadly accepted way of controlling traumatic hemorrhage for a variety of reasons. Catheter-based hemostasis allows precise control from a remote site that avoids exacerbation of venous hemorrhage, introduction of pathogens, and hypothermia that may result from open exposure. It is especially valuable for hemorrhage that is remote or hidden from view and requires laborious time-consuming exposures or that is the result of multiple small bleeding sites that are not easily detected or controlled during operative exploration.

Endovascular techniques include embolization, stenting, stent grafting, and temporary balloon occlusion and may be definitive in nature or an adjunct to operative exposure. The methods of embolization include particulate or microcoil embolization of small vessels, proximal and distal control of a bleeding vessel, and coil occlusion to cause selective temporary hypotension of the bleeding zone.

Stenting, which facilitates blood flow beyond an injury, has largely been replaced by covered stent grafts that exclude lacerations, transections, and arteriovenous fistulae while maintaining flow through the conduit. Endografts are made of a variety of porous materials such as expanded polytetrafluoroethylene and are reinforced by a metallic skeleton that apposes the stent graft to the native artery. Reports of midterm patency, while limited at this time, are beginning to show that these are durable options to vascular repairs.

Contraindications to endovascular techniques are highly dependent on skills, teamwork, and hemodynamics; however, there are some injuries that are difficult for rapid surgical control and for which endovascular techniques have a role, even in the unstable patient.

Arch Angiography for Acute Blunt-Force Traumatic Aortic Injury

Screening and diagnostic arch angiography for blunt traumatic aortic injuries has largely been replaced by chest CTA, particularly with the widespread availability of MDCT technology.[67] Arch angiography, however, does have a role in the evaluation of equivocal findings on chest CTA and in potential endovascular stent graft therapy. If patients are going directly to angiography for evaluation of disruptions of the pelvic ring and the mediastinum is not normal on a chest x-ray, catheter arch angiography is the preferred "screening" modality; otherwise, CT is preferred. Modern CTA techniques are quite exquisite in demonstrating aortic injuries as well as providing coronal and sagittal reformations that can illustrate the important relationships and variants necessary for surgeons to create a treatment plan. Among many patients sustaining aortic injury, endovascular stent grafts have been advocated as definitive therapy.

Typically, a 5F pigtail catheter is guided to the ascending aorta via a femoral arterial approach. Patients are positioned and imaged in both 35° right anterior oblique and left anterior oblique projections, using injection rates of approximately 25 to 30 mL/s for a 40- to 60-mL volume (depending on hemodynamic status) and positioning to include the great vessels and diaphragm.

The angiographic appearance is classical. Linear filling defects indicate torn and ruffled intimal lining, with expansion of the lumen (typically at the ligamentum arteriosum) indicating the presence of a pseudoaneurysm (Fig. 19-44). The tear of the aortic wall may be segmental or circumferential and is sometimes associated with distal narrowing of the contrast column (pseudocoarctation). Minimal aortic injuries, as discussed earlier in this chapter, may be very difficult to visualize angiographically, and a "negative" aortogram following a positive chest CTA for a minimal aortic injury does not exclude the diagnosis.

Associated mediastinal vascular injuries should also be identified. Injuries of the arch arteries may occur instead of, or in association with, an aortic injury. Bleeding from the internal mammary or the intercostal arteries may be easily overlooked without diligence.

Hepatic Angiography for Blunt-Force Lacerations

Visceral catheter angiography is appropriate to evaluate hepatic lacerations (Fig. 19-45), particularly in patients with a labile hemodynamic status or those with active extravasation or vascular abnormalities as seen on a contrast-enhanced CT. Visceral catheter angiography may have a role after a "damage control" operation. Gross hemodynamic instability and profound shock, however, usually mandate urgent celiotomy.

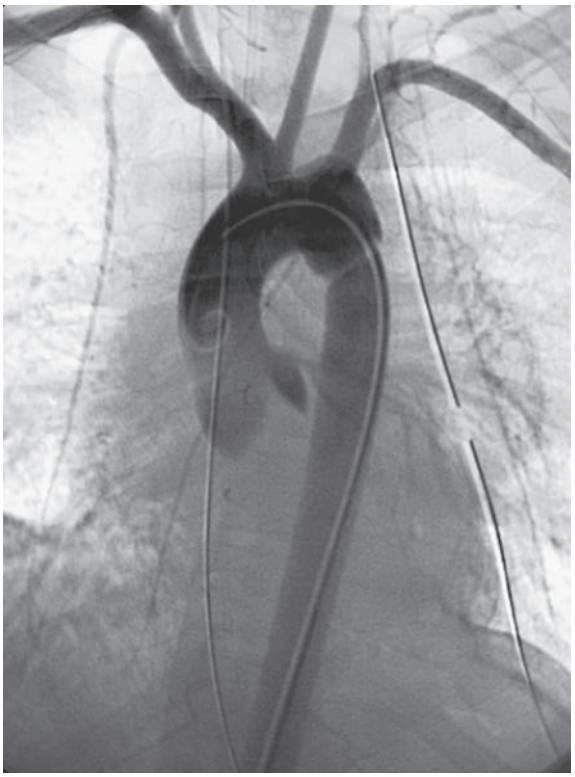
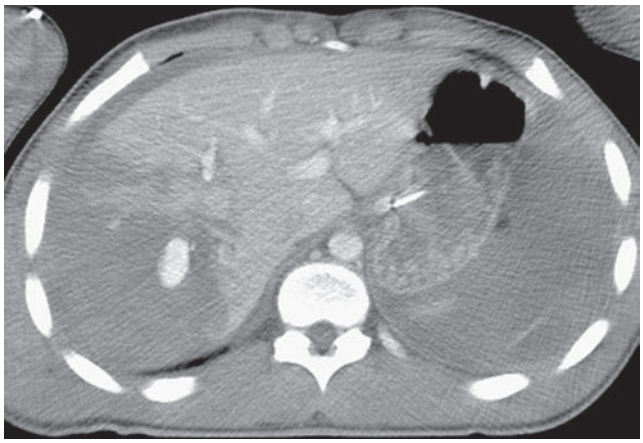


FIGURE 19-44 Traumatic aortic pseudoaneurysm. A 30-year-old man following a high-speed motor vehicle accident. Left anterior oblique digital subtraction arch aortogram shows traumatic aortic pseudoaneurysm extending proximal to the left subclavian artery. Of note, the aortic diameter and the distance from the left subclavian artery are important when considering endovascular therapy.

On CT, hepatic fracture lines that traverse the hepatic triad result in bleeding more often than those that run parallel to the triad. Contrast extravasation on CT tends to be associated with a positive arteriogram, but the decision to use angiography should primarily be based on clinical status rather than the CT appearance. Lack of enhancement of liver segments on CT is a very important finding, as these regions represent an intraparenchymal hematoma, occlusion of the portal triad, or injury of the hepatic outflow from that segment. It is vital to distinguish nonenhancement from a hematoma, which can be challenging. A large hepatic hematoma pushes the hepatic fragments away from each other and lacks hepatic vessels. If the area of nonenhancement has vessels running through it, it suggests an occlusion of the portal vein and hepatic artery or injury to a hepatic vein. Therefore, CT nonenhancement of the liver is an indication for angiography to confirm such injuries and to control ongoing arterial hemorrhage. As surgical exploration of damaged hepatic veins may be quite difficult, hepatic embolization and observation of a nonbleeding hepatic venous injury can be lifesaving. Hepatic angiography can also be helpful in the management of penetrating liver injuries that are isolated to the liver.



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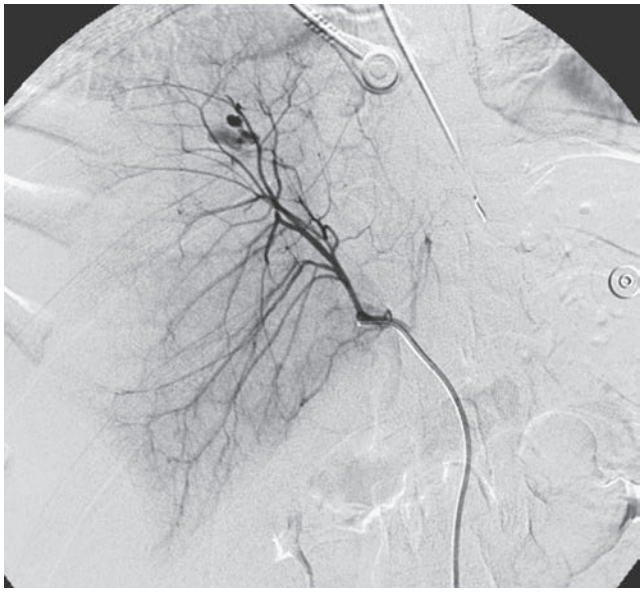

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FIGURE 19-45 Liver laceration. (**A**) Computed tomography of the upper abdomen reveals a grade V liver laceration with pseudoaneurysm of the right hepatic lobe in this 18-year-old man status post high-speed motor vehicle accident. (**B**) Right hepatic angiogram identified the pseudoaneurysm. Note the size of the feeding vessel in relation to the 5F diagnostic catheter. Selective coil embolization was performed through a microcatheter. When selective catheterization is not possible, the liver is quite tolerant of wide arterial embolization due to the dual blood supply, provided the portal veins are patent.

Selective catheterization of both the celiac trunk and the superior mesenteric artery (SMA) is essential due to the high rate of hepatic vascular variants, particularly the aberrant replaced right hepatic artery from the SMA (15%–20%). Imaging should be continued through the late portal venous phase to evaluate for slowly bleeding vessels. There should be careful inspection for abnormal parenchymal enhancement and intrahepatic portal venous filling, as these are features of hepatic arterial-portal venous fistulae, which can easily be overlooked.[97]

Critical findings include arterial extravasation, spasm and occlusion, or shunting and fistula to portal or hepatic venous structures. Embolization of discretely abnormal vessels can be performed using a number of methods. A diffusely abnormal parenchymal injury with arterial bleeding may be safely embolized with Gelfoam due to the dual blood supply of the liver (hepatic arterial and portal venous). Embolization of hepatic arteries in the absence of portal flow increases the risk of developing an infarction or abscess. Depending on the location of bleeding and on the difficulty with catheterization, particulate embolization is the fastest technique; however, single microcoil embolization is preferred if time and circumstances allow. While formation of a postprocedure abscess is a complication of embolization, outcomes are favorable by integrating percutaneous image-guided drainage into the scheme.

Splenic Angiography for Blunt-Force Lacerations

Patients with splenic injuries diagnosed on CT are candidates for nonoperative management with overall good salvage rates, particularly when combined with splenic angiography and embolization.[77,98] When CT demonstrates active arterial extravasation or a parenchymal vascular abnormality (eg, an intraparenchymal pseudoaneurysm), one should consider angiography, as these are independent predictors of failure of nonoperative management.[99] It is thought that there was poor correlation between the CT grading system and outcome of treatment, as many grade IV injuries can be observed, and some grade I injuries become worse, rebleed, and require definitive procedural therapy. However, there is emerging evidence that a CT-based grading scheme, in combination with clinical parameters (eg, Abbreviated Injury Scale score), may be helpful for patient triage.[100,101] The treatment algorithms for patients with splenic injuries in the absence of active bleeding or other vascular lesions vary greatly by institution. The absence of arteriographic extravasation is a highly reliable predictor of successful nonoperative therapy regardless of injury grade. Identification of active arterial extravasation is the standard indication for endovascular treatment.

Diagnostic angiography of the celiac trunk is followed by selective splenic artery catheterization with a 5F catheter. If splenic artery anatomy permits and a solitary pseudoaneurysm or focus of extravasation is seen, distal coil embolization at the site of injury can be attempted. This is especially true in a patient in whom the extravasation extends beyond the splenic capsule into the peritoneal cavity. One should note that distal superselective embolization is associated with the development of more postprocedure splenic infarctions and abscess, although these complications are uncommon. Finally, most patients have tortuous splenic arteries, and most extravasations are multiple.

Diffuse intrasplenic extravasation is far more common, and superselective occlusion of these multiple sites would be very time consuming and less effective. In addition, the splenic tortuosity that results from medial displacement of the spleen by the perisplenic hematoma often prevents rapid catheterization (Fig. 19-46). In such cases, embolization of the proximal splenic artery by coils or other occlusion devices placed distal to the dorsal pancreatic branch and proximal to the pancreatic magna branches is advocated to reduce the arterial pressure head at the injury site while allowing perfusion through collateral vessels. Such collaterals prevent splenic infarction by maintaining splenic perfusion through connections between the left gastric and the short gastric arteries, between the dorsal pancreatic artery and the greater pancreatic artery branches, between the right and left gastroepiploic vessels, and others.

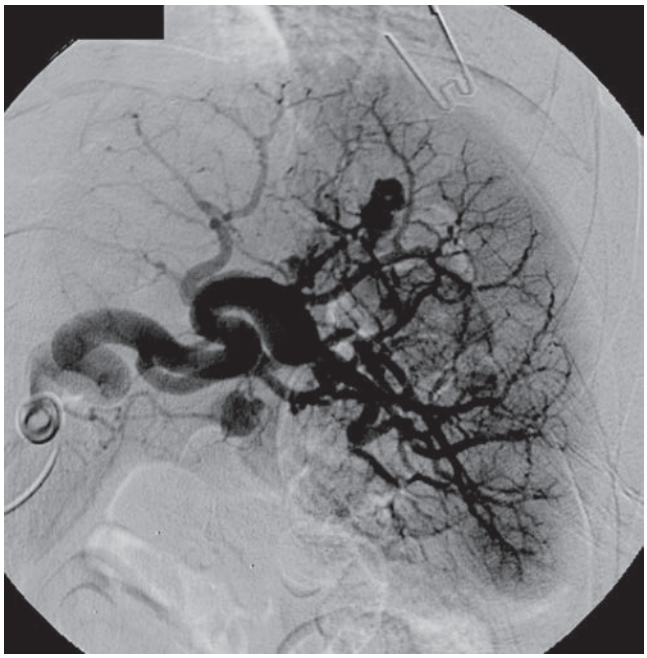


FIGURE 19-46 Splenic intraparenchymal false aneurysms. Digital subtraction angiogram of the splenic artery reveals multiple focal extravasations in this 56-year-old man status post motor vehicle accident. Selective embolization is not desirable because so many vessels are injured and selective catheterization would be difficult due to splenic artery tortuosity. In such cases, proximal splenic artery coil embolization proximal to the pancreatic magna branch is usually successful in controlling this hemorrhage.

Complications are uncommon when proximal splenic artery embolization is performed. A poorly selected coil size may result in hilar occlusion if the selected coil is too small and migrates distally. Coils that are too large may migrate proximally to occlude the celiac axis or embolize into the aorta. As noted earlier, distal microembolization bypasses the collateral circulation and results in more loss of immune function. It should be noted, however, that splenic blood flow is maintained in most patients via a combination of intact left gastroepiploic and short gastric arteries. This preserves immune function of the spleen, as well as distal flow. Occlusion of the pancreatic branches may result in pancreatic necrosis and

pancreatitis, although these complications are rare and can be avoided by careful review of the angiographic images if main splenic artery embolization is considered.

Interventions for Renal Trauma

Many renal injuries are usually well tolerated and do not require angiography, especially when caused by blunt trauma. Initial nonoperative management of blunt renal injuries with an intact pedicle is the current accepted management standard. High-grade injuries that result in massive hemorrhage may necessitate nephrectomy. Angiography for embolization of active bleeding is appropriate for hemodynamically stable patients. Angiography is recommended for patients with CT evidence of a major renal injury and ongoing blood loss or persistent gross hematuria. Peripheral wedge-shaped regions of nonenhancement on CT suggest a segmental or distal renal artery injury, often due to avulsion injury or intimal stretch resulting in distal platelet embolization. Penetrating renal injuries are more aggressively approached by angiography if nonoperative management is undertaken. Large perinephric hematomas, areas of nonenhancement, and active bleeding on CT warrant angiography following penetrating trauma.

Aortography is helpful to assess injury of the origin of the renal artery, to exclude renal parenchymal injury perfused by accessory renal arteries, and to look for associated bleeding sites. A selective renal arteriogram using a 5F catheter is then performed. Most injuries will require use of a coaxial microcatheter and embolization of small branches. Coils are preferred as they can be carefully placed to prevent infarction of adjacent noninjured renal tissue, but surgical gelatin pledgets can be used as well. Because renal branches are end vessels with little collateralization, infarction is likely, and the goal is to reduce these infarctions to a minimum.

The treatment of vascular injury in the renal pedicle continues to be a vexing problem, especially because delays in revascularization usually result in a renal infarction or renovascular hypertension. Partial wall injuries that result in a pseudoaneurysm or segmental infarction often went unrecognized prior to the widespread use of CT. Such injuries are routinely detected before complete arterial thrombosis and renal infarction occur. Therefore, arteriography is indicated when an injury in the renal artery is suspected. When such injuries are detected, treatment options are many, including operative revascularization, antiplatelet therapy and observation, and the application of covered stent grafts. Stent grafts can effectively seal full-thickness injuries and cover exposed media that results in embolic infarctions. Although long-term follow-up of series of these patients is lacking, the mid-term (1–5 years) patency of stent grafts throughout the body remains high (Fig. 19-47).

Angiography for Pelvic Hemorrhage

Pelvic fractures are potentially life-threatening injuries that are caused by high-energy impact trauma and are the third most common lethal injury following motor vehicle crashes. The majority of patients with pelvic fractures do not require massive transfusion as bleeding in most cases is likely to be venous or osseous in nature and often self-limited. Radiologic intervention is not commonly required in patients with routine pelvic fractures. Severe hemorrhage, however, occurs in 3% to 10% of patients, and mortality rates may be as high as 40% in patients with unstable pelvic fractures.[102] Thus, the use of angiography in patients with pelvic fractures is highly dependent on the hemodynamic status, the type of pelvic fracture pattern, the transfusion requirements, and the presence or absence of pelvic hematoma.

BLUNT PELVIC FRACTURES

Blunt pelvic fractures with crushing or shearing tear the small branches of the internal iliac artery that accompany ligaments, muscles, and tendons. Injuries tend to be multiple and bilateral and from several branches. In addition, bony fragments can penetrate or perforate vascular structures. Examples include a fracture of the superior pubic ramus injuring the internal pudendal or obturator artery, a fracture of the iliac wing through the sciatic notch injuring the superior gluteal artery, and disruption of the sacroiliac joints injuring the lateral sacral arteries.

Most of the indications for angiography in blunt pelvic trauma have remained the same for many decades and include the following:

1. Hemodynamic instability in a patient with a pelvic fracture with no or little hemoperitoneum detected by FAST or DPL.
2. Pelvic fracture and transfusion requirement of greater than 4 units in 24 hours or 6 units in 48 hours.
3. Pelvic fracture and a large or expanding hematoma identified during celiotomy.
4. CT evidence of large retroperitoneal hematoma with extravasation of contrast.
5. Need for detection and treatment of other injuries during angiography.

The presence of contrast extravasation on MDCT has been used as an indication for follow-up pelvic angiography. Although it should not delay angiography that is already indicated for pelvic hemorrhage, CT is helpful in localizing the vessels likely to be bleeding not only in the pelvis, but also from the solid organs and thoracic cavity. Correlations of location of the hematoma and site of vascular injury include obturator space and obturator artery, presacral space and lateral sacral artery, space of Retzius and internal pudendal artery, and buttock and gluteal artery.

Femoral access is the preferred approach; however, catheterization may be difficult because of hypotension, tachycardia, and difficulty in palpating the vessels as the pelvic hematoma expands. Ultrasound or fluoroscopic guidance is very helpful in these situations. A 5F aortic flush catheter is used for flush abdominopelvic aortography. This is valuable

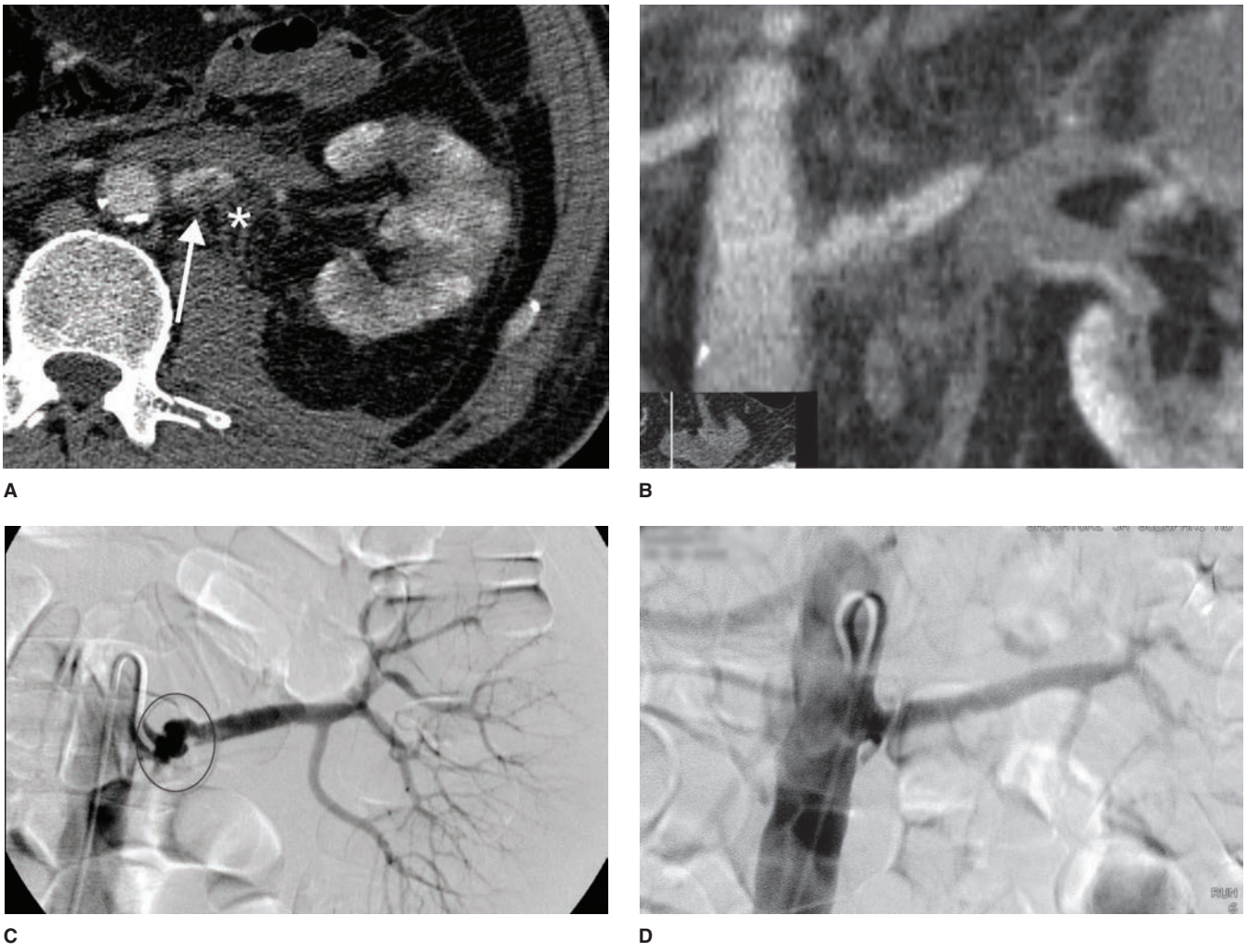


FIGURE 19-47 Renal artery injury. A 56-year-old man fell from a height of about 10 m. (**A**) During computed tomography evaluation, inhomogeneous enhancement of the spleen was detected. Central perinephric hemorrhage (asterisk) and irregularity of the renal artery (arrow) were seen. (**B**) Coronal reformation shows irregularity of the renal artery and thickening of its wall. (**C**) Aortography shows irregular enlargement of the proximal renal artery near the ostium (circled). Slight extravasation was seen on the later images. (**D**) Therefore, a stent graft was placed over the area of injury. The vessel wall was then smooth, and no extravasation was seen. Two-year follow-up arteriography showed continued patency and no stenosis.

to screen the abdominal viscera and mesentery, to exclude aortoiliac and other retroperitoneal bleeding sources, and as a road map of the pelvic vessels. Selective bilateral internal iliac arteriography is mandatory to exclude bleeding sites since aortography may not identify all bleeding. From one access, both internal iliac arteries are sequentially catheterized and opacified. Then, external iliac arteriography is used to evaluate the external pudendal and external obturator vessels.

It is common to identify multiple areas of extravasation during pelvic angiography. These may be bilateral and may involve multiple vascular beds. Extravasation is often punctate but can be large and coarse, and the size of such extravasations may not correlate with the degree of blood loss. Vascular occlusions due to transection or dissection with subsequent occlusion can be present as well. It can be difficult to differentiate between arterial injury due to thrombosis and vasospasm. Failure to treat these occlusions may result in recurrent hemorrhage when vasospasm resolves. Arteriovenous fistulas can occur but are more common in penetrating trauma.

Because bleeding is usually multifocal and originates from multiple small blood vessels, embolization requires small particulate embolization. Large coil occlusion is as ineffective as surgical ligation of the internal iliac artery because bleeding soon resumes through numerous collateral circuits. Surgical gelatin pledgets are ideal because they are inexpensive, readily available, and often temporary, lasting only a few weeks and allowing reestablishment of normal blood flow after the

tissue has healed (Fig. 19-48). Permanent particulate emboli, however, are often used because of their ease of use through a microcatheter (Fig. 19-49). Embolization is technically successful in more than 90% of patients, and hemorrhage control is highly effective. Survival depends on many other factors including associated injuries, the presence of an open fracture, transfusion requirements, and delays to embolization.

PENETRATING PELVIC TRAUMA

Penetrating trauma is an uncommon indication for pelvic angiography, as many patients are hemodynamically unstable or have clear indications for immediate exploratory celiotomy, often due to injury to a large vessel. Because the extraperitoneal space has been exposed by a penetrating wound, intraperitoneal bleeding is likely, and direct exploration is typically warranted. Occasionally, angiography is valuable when operative control cannot be initially accomplished and damage control has been performed. Angiography and embolization prior to unpacking can aid in decreasing blood loss at a reoperation.

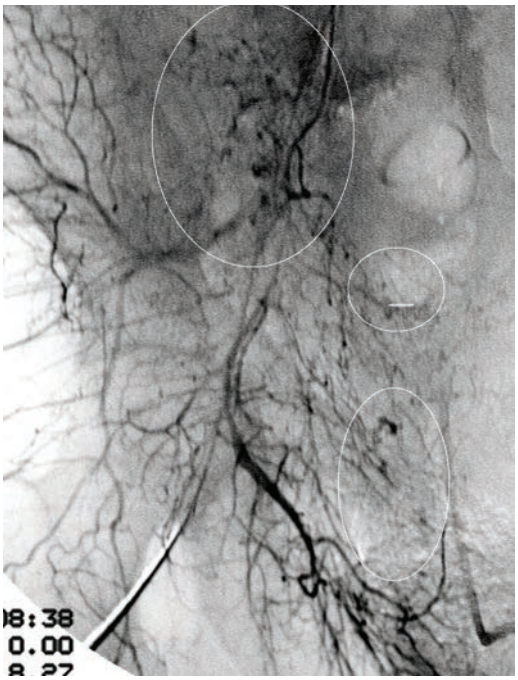
Injuries to large vessels require a very different endovascular strategy than small vessels or end organ vessels. When an injury to a noncritical internal iliac artery or branch has been missed at operation but detected on postoperative angiography, coil occlusion of both the proximal and distal end of the vessel (whenever possible) is the standard treatment.

Catheter Angiography for Peripheral Vascular Injuries

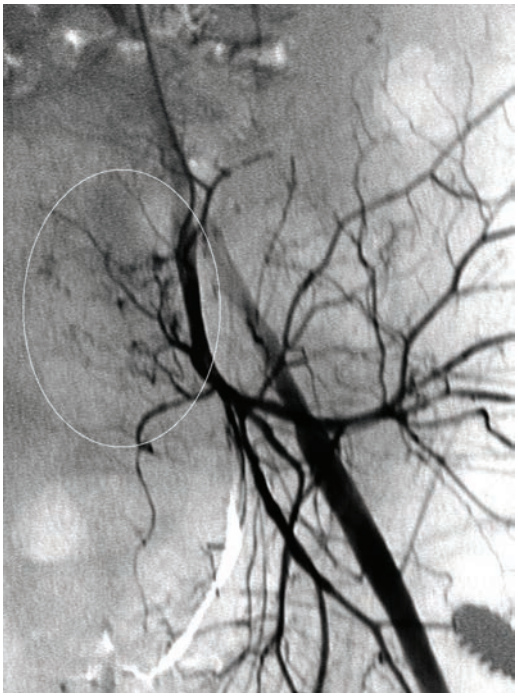
Almost all peripheral vascular injuries can be reached using a 5F catheter from femoral access provided a long enough catheter is available. Angiography should be done in multiple projections with opaque marking of surface wounds demonstrating that the entire course of the wounding agent is within the field of view. Iso-osmolar nonionic contrast medium is the optimal agent for visualization. Multiple images in the arterial, capillary, and venous phases are necessary.

There is a body of literature demonstrating that the combination of physical exam findings and noninvasive tests (eg, ankle-brachial index and Doppler ultrasound) is reliable in the exclusion of significant peripheral vascular injuries for patients with low likelihood of vascular injury. The overall incidence of extremity vascular injuries in the setting of penetrating trauma for asymptomatic patients is relatively low (3%–4%). Thus, screening CTA or angiography for "proximity" injuries is likely to be low yield in the absence of "hard" clinical signs.

Vascular injuries resulting from fractures and dislocations are uncommon. Clinical evaluation is often difficult because the hematoma from a fracture may be quite large and indistinguishable from one associated with a vascular injury. Crush wounds, angulation deformities, and fracture hematomas may cause a pulse deficit by kinking, entrapping the vessel, or inducing spasm without an intrinsic vascular injury.

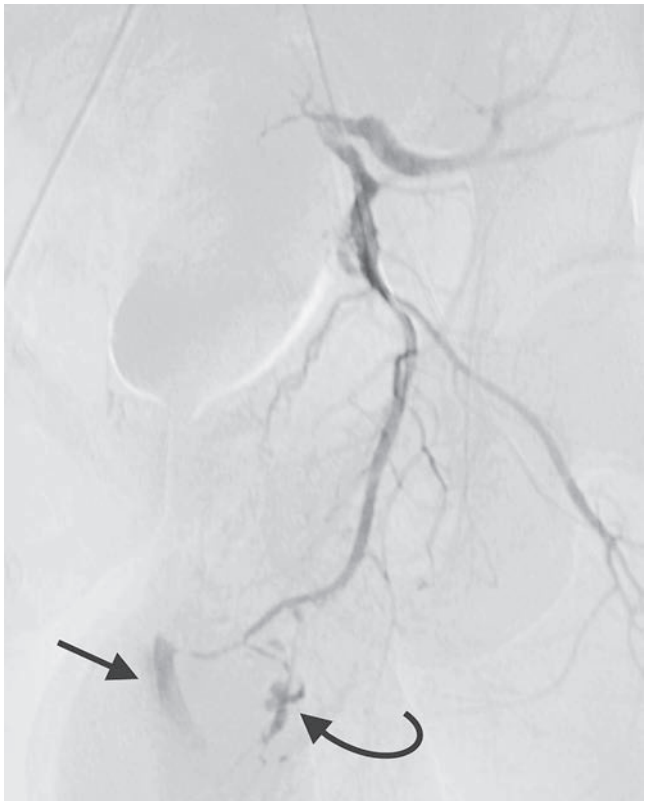


A



B

FIGURE 19-48 Multiple bleeds from pelvic fractures: 48-year-old male driver in a motor crash sustained pelvic fractures requiring transfusions. (**A**) Circles surround multiple bleeding sites from the region of the sacroiliac joint; from the pelvic side wall on the right hemipelvis emanate anterior and posterior branches of the right internal iliac artery and in the region (**B**) multiple points of extravasation were detected (circle). They are emanating from the left lateral sacral artery. Such diffuse hemorrhage is not amenable to superselective embolization because it would be too time consuming. Pledgets of surgical gelatin, 2 to 3 mm in size, can occlude these vessels effectively.



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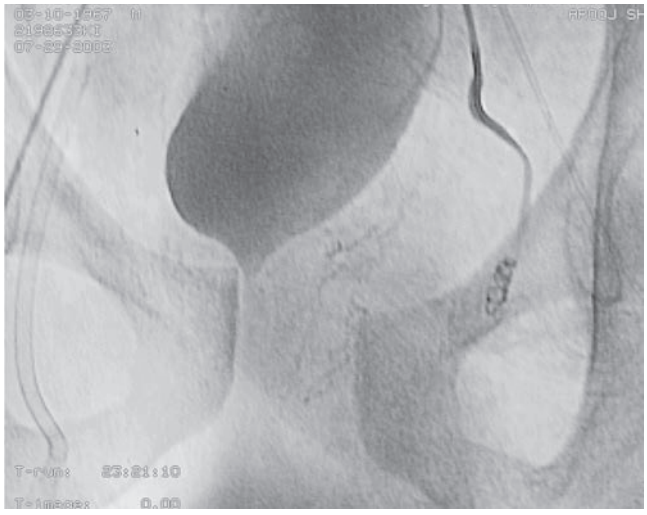


B

FIGURE 19-49 A 26-year-old motorcyclist sustained unstable pelvic fractures during a crash. He developed expanding perineal and scrotal hematomas requiring red cell transfusion. (**A**) Left internal iliac arteriogram reveals a source of bleeding from the left internal pudendal artery (curved arrow). The more medial contrast stain (straight arrow) is a normal finding. It represents the blush of the perineal body and root of the ischiocavernosa muscle that is frequently seen on internal iliac arteriography of males. (**B**) Because this was focal hemorrhage, selective embolization via 2.8F catheter placed coaxially through the 5F catheter was attempted and successfully achieved hemostasis.

A laceration into muscle may result in external blood loss without major vascular injury. The natural history of many injuries cannot be predicted by the angiographic appearance alone. Therefore, observation of some injuries is warranted. Equivocal findings such as luminal narrowing can be assessed by repeating angiography, after infusion of an intra-arterial vasodilator, on a subsequent day. Small irregularities and intimal tears that do not restrict flow may be treated by antiplatelet therapy and will generally heal (Fig. 19-50).

Treatment of angiographically diagnosed vascular injuries is based on the anatomic role of the bleeding vessel; its size, location, and accessibility; the hemodynamic condition of the patient; and the specific type of lesion. Small vessels that are not essential for tissue perfusion can be treated by small-particle embolization, using surgical gelatin pledgets or more permanent smaller agents. Permanent agents have no advantage but, in some instances, are more easily administered through microcatheters than surgical gelatin. These agents are delivered by flow direction toward the path of least resistance, which is usually toward the bleeding site. Microcoils can be used for injury to a small vessel provided they can be delivered near enough to the injury site to avoid collateral recruitment that permits continued bleeding. Examples of vessels that can be treated by embolization of small particles include hemorrhage from a pelvic fracture, multifocal hepatic arterial hemorrhage, and injuries to muscular branches such as those of the profunda femoris artery in the lower extremity.

Injury to larger vessels such as those greater than 3 mm in diameter requires two techniques, one for essential vessels and one for expendable vessels. The treatment of essential vessels mandates repair of the bleeding site while allowing continued blood flow. Thus, stent grafts can be deployed to cover the injured segment while allowing antegrade flow (Fig. 19-51).

Nonessential conduits, such as branches of the profunda femoris artery or the brachial artery, or one of the arteries in the shank, can be safely embolized. Particulate embolization will flow past the injury and penetrate deep into the vascular bed. When conduits are injured, this insult to the vascular bed is unnecessary. Therefore, large-vessel agents are used to occlude the damaged segment of the conduit while the vascular bed is perfused through collaterals (Fig. 19-52).

Coils in various sizes, some containing threads or fibers to accelerate thrombosis, are the most common devices used to occlude a large vessel. A coil is sized to have a diameter large enough to prevent distal migration, but not too large to end up recoiling into a parent, nontarget vessel.

The technique of arterial isolation attempts to occlude both the proximal and distal vessels around the area of injury by coiling (Fig. 19-52). The goal is to exclude the vascular defect and prevent rebleeding through collateral vessels. This is highly desirable in most circumstances, but mandatory when treating arteriovenous fistulas. The guidewire is carefully maneuvered distal to the injured segment but proximal to any branches, and coils are delivered. The catheter is then withdrawn, and coils are placed in the proximal segment.

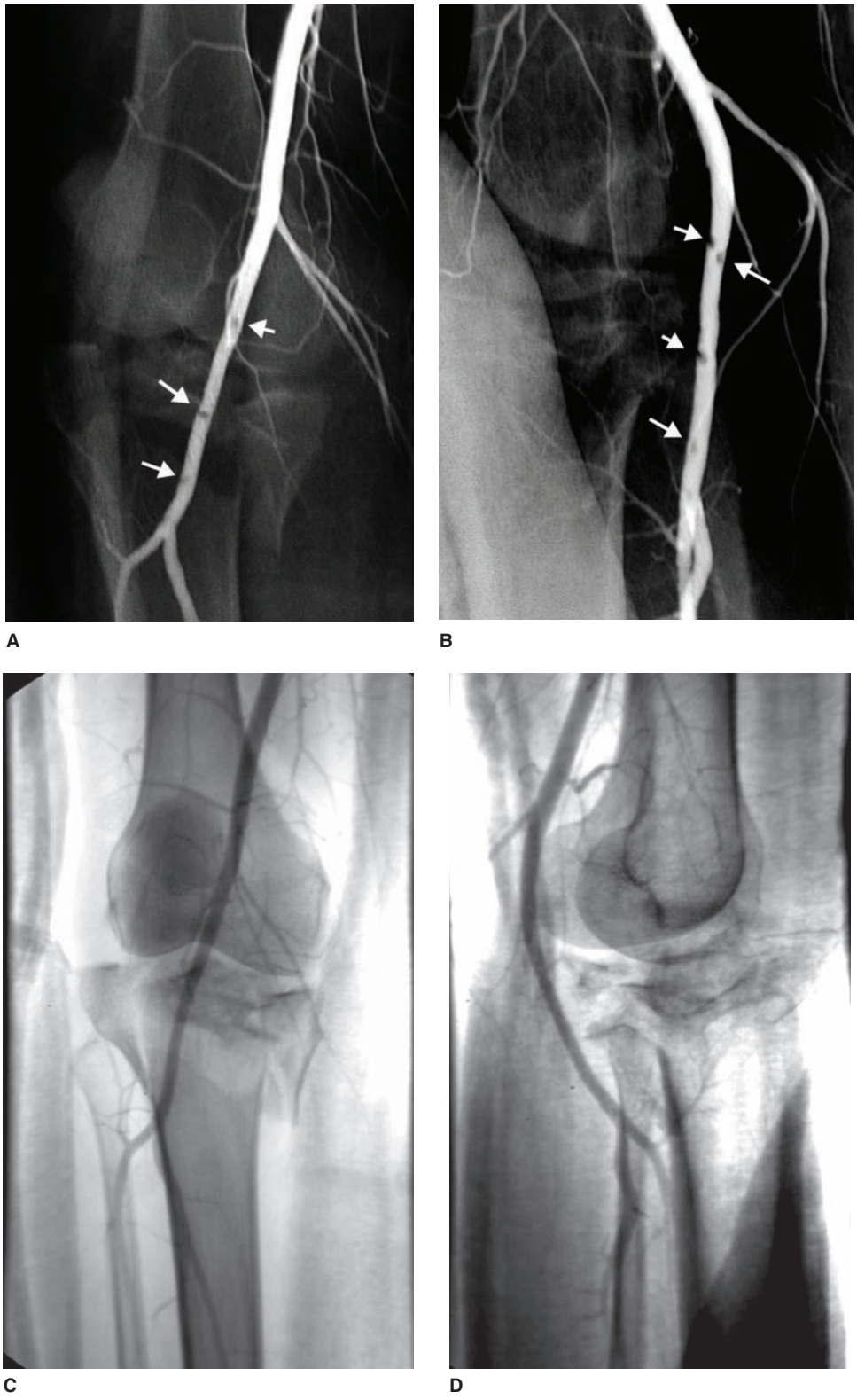
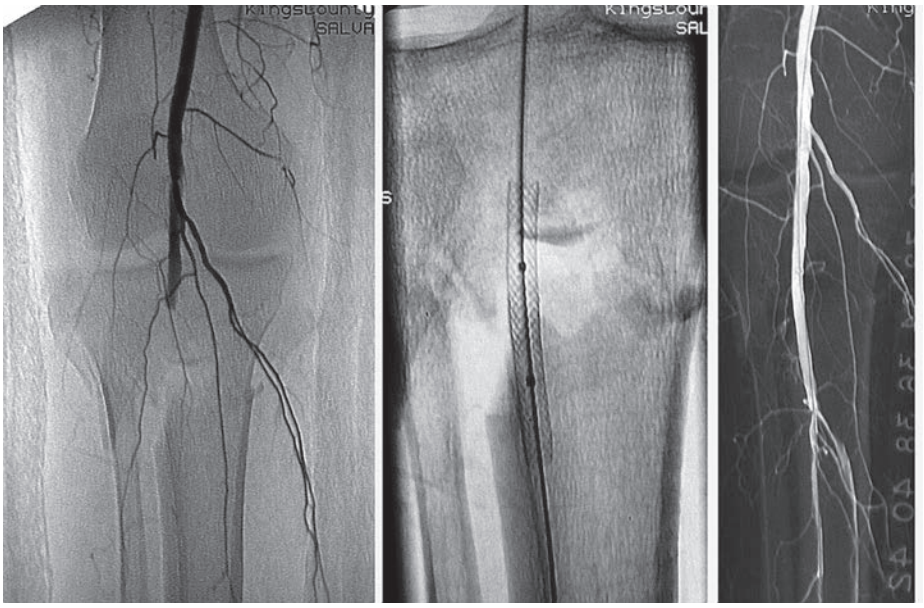
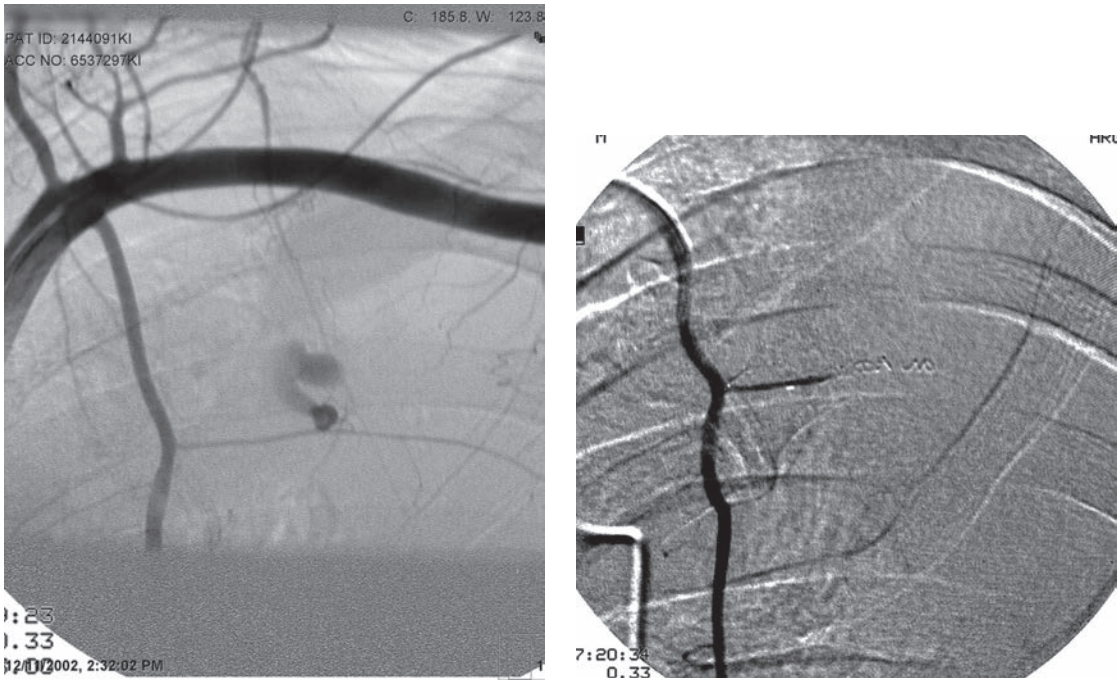


FIGURE 19-50 "Minimal injury" of the popliteal artery. Pedestrian who was struck by a motor vehicle sustained comminuted tibial plateau fracture of the left knee. Pulses were diminished and angiography was sought after incomplete reduction. (**A, B**) Initial popliteal arteriogram showed numerous filling defects consistent with intimal tears (white arrows). Patient was treated with aspirin. (**C, D**) Arteriogram 1 week later showed healing of the intimal tears.



A B C

FIGURE 19-51 Thrombosis of popliteal artery with endovascular repair. A 46-year-old morbidly obese woman sustained comminuted tibial plateau fractures after a fall from curb. Pulses were absent. (**A**) Popliteal arteriogram shows complete occlusion of the mid-popliteal artery. (**B**) The catheter was quickly advanced to a location just above the occlusion, and a guidewire was advanced easily into the posterior tibial artery. An expanded polytetrafluoroethylene–reinforced stent graft was deployed between proximal and distal extent of the occlusion. (**C**) Follow-up popliteal arteriogram showed restoration of direct line flow. The entire procedure took less than 1.5 hours.



A B

FIGURE 19-52 Example of vascular isolation by proximal and distal coil occlusion. A 22-year-old man sustained a single stab wound of the upper left chest resulting in a very large hemothorax. (**A**) Subclavian arteriogram shows that there is active arterial hemorrhage from a lacerated fourth anterior intercostal branch of the left internal mammary artery. (**B**) Because there was continuity between anterior and posterior intercostals, it was necessary to advance a 2.8F microcatheter across the laceration into the distal segment to deliver a coil distally before withdrawing the catheter and delivering a coil proximally.

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20

Principles of Anesthesia and Pain Management in Trauma Patients

Ron E. Samet • Justin E. Richards • Ian R. Slade

KEY POINTS

- Anesthesia in trauma care is the practice of perioperative resuscitation.
- Anesthesiologists, surgeons, and emergency medicine and critical care physicians must communicate and collaborate effectively to provide comprehensive continuous trauma care.
- Anesthesiologists blend clinical pharmacology expertise and advanced physiologic monitoring to optimize resuscitative outcomes in trauma patients.
- Standard anesthetic objectives of anxiolysis, analgesia, hypnosis/amnesia, and immobility often require creative modification to accommodate trauma physiology.
- Regional anesthesia techniques should be incorporated earlier in trauma care to facilitate patient comfort, assessment, procedural care, and transport without impairing cognitive function.
- Multimodal analgesia techniques are proving increasingly valuable in reducing the harmful impacts of opioid use, both in the acute hospitalized trauma patient and in the immediate postdischarge phase.

Anesthesiologists are perioperative physicians who are responsible for preoperative evaluation and preparation, intraoperative anesthetic and critical care medicine, and acute postoperative care.[1] Whereas elective surgical patients benefit from comprehensive preoperative evaluation, medical optimization, and a multitude of safe and comfortable anesthetic options, management of the acute trauma patient can be remarkably different. Limited planning time, unknown patient variables, and rapidly changing patient and surgical conditions demand a more adaptive approach. The trauma patient's physiologic instability from ongoing hemorrhage and multiorgan injury demands the prioritization of resuscitative medicine and may require significant adaptations of traditional anesthetic approaches. These patients require a dynamic balance of perioperative goals with consideration of type and depth of anesthesia, medication selection and dosing adjustments, airway management techniques, continuous resuscitative assessment and treatment, and postoperative disposition and pain management. With unique challenges in trauma anesthesia care, this chapter highlights the key considerations and modifications to standard anesthetic practice for each phase of care in the acute trauma patient.

PREOPERATIVE ASSESSMENT AND MANAGEMENT

Anesthetic planning begins preoperatively and must consider preoperative, intraoperative, and postoperative objectives. Information about a patient's current medical and surgical condition is gathered to appraise risk at the organ-system level, predict likely changes from evolving injury, and formulate a dynamic anesthetic prescription (and contingency plans) to manage perioperative risks. In busy trauma centers, anesthesiology presence at the initial phases of care allows for direct participation in early resuscitation and helps allocate resources (eg, staff, space, time, equipment, blood availability) to facilitate emergent surgical interventions; this is particularly crucial when triaging multiple-casualty incidents.[2] For trauma patients without immediate surgical needs, early anesthesiology assessment remains a valuable opportunity to document future anesthetic options and discuss medical optimization with other providers. Finally, trauma patients with complex pain management requirements can also be identified at the onset and offered regional anesthesia techniques to ameliorate the need for large-dose opioids or other sedative medications.[3]

General Principles of Preoperative Evaluation

Standard preoperative anesthesia evaluation involves a focused history, physical examination, and review of laboratory and radiology tests, if available. Particular attention is given to planned surgical procedure and urgency; comorbidities; previous medical and surgical histories; previous anesthetics and their complications; the airway, cardiovascular, and lung exam; and whether the patient has been optimized for surgical fitness. In the conscious trauma patient, gathering this information may be straightforward. Otherwise, the anesthesiologist must rely on reports from first responders, family members, medical personnel, or other available records. Special attention to emergency medical services reports as to the mechanism of injury, blood loss at the scene, airway, and resuscitative interventions are of utmost value because prehospital details can dramatically alter in-hospital interventions. Some use the AMPLE mnemonic: *a*llergies, *m*edications, *p*ast medical/surgical history, *l*ast meal, and *e*vents/*e*nvironment leading to injury. Physical examination may provide some important clues, such as scars from previous traumatic injury or repair, medic alert bracelets, sternotomy scars, palpable pacemakers, insulin pumps, and general body habitus (obesity, pregnancy).

Because many drugs routinely used in anesthetic care induce vasodilation and hypotension, careful attention is given to baseline cardiovascular disease and perturbations in volume status, especially in trauma patients who may be hypovolemic due to ongoing hemorrhage. Preoperative evaluation enables close consideration of factors influencing choice of anesthetic induction technique, airway management, vascular access, hemodynamic monitoring, and fluid management. These interests for anesthesiologists align closely with the Advanced Trauma Life Support primary survey, with special attention to airway status, examination for impediments to airway management such as potential coexisting cervical spine injury with the presence of a cervical collar, blood in the airway, and general functional capacity. The presence of the anesthesiology team on the patient's arrival and during the primary survey, therefore, expedites this portion of early assessment.

Shared Role in Early Resuscitation

In conjunction with the entire trauma team, anesthesiologists often have a key role in joint resuscitation of patients in the preanesthetic phase. Particular contributions of the anesthesia team can include airway securement; establishment of arterial and large-bore venous access; planning of ongoing resuscitative needs, such as obtaining a blood sample for type and crossmatch; assessment of coagulation parameters; blood gas analysis and ventilator management; coordinating vasoactive infusions; and transfusion management. Effective early resuscitation aimed at restoring circulating volume, organ perfusion, and oxygen delivery can reduce the risk for profound hypotension upon induction of general anesthesia and prolonged circulatory shock. Volume and blood resuscitation, in combination with early prevention and treatment of hypothermia and coagulopathy, helps prepare patients for the risk of further physiologic disruption as their injuries evolve and in the course of surgical care. Patients needing massive transfusion can be quickly identified, and blood bank resources can be mobilized and initiated promptly. In some circumstances, anesthetic care may even commence in the emergency department when operative procedures need to be conducted in the trauma bay. By having already participated in assessment and early resuscitation, anesthesiologists are particularly well positioned to assume the resuscitative responsibilities of the trauma team as the patient transfers to the operating room.

INTRAOPERATIVE ASSESSMENT AND MANAGEMENT

Intraoperative anesthesia management is often triphasic with induction, maintenance, and emergence phases. Induction involves administration of induction drugs and doses, techniques to secure the airway, and management of hemodynamic perturbations from the preceding two interventions. Maintenance focuses on maintaining the anesthetized state and homeostasis, whereas emergence defines the task of smoothly transitioning patients to an awake and comfortable state. The anesthesiologist is responsible to provide modulation of physiologic functions, monitoring, and when applicable, anxiolysis, analgesia, hypnosis, amnesia, and immobility. Given that nearly all analgesic and anesthetic medications risk inducing further hypotension via vasodilation, negative cardiac inotropy, or obliteration of the catecholamine response, it is often said that a patient in traumatic shock must "earn" their anesthetic medications as resuscitative efforts restore adequate perfusion. In severe injuries, the anesthesiologist may not administer any classical "anesthetics" and functions more as a traumatologist practicing resuscitative medicine.[4] A review of intraoperative anesthetic goals and commonly used anesthetic agents follows, with particular attention to their benefits and risks in the acute trauma patient.

Intraoperative Resuscitation

In acute trauma patients, the primary responsibility of a trauma anesthesiologist is to resuscitate and restore macro- and microcirculatory perfusion while simultaneously preparing the patient for surgical hemorrhage control and repair. Constant communication with the trauma surgeon, observation of operative progress, and inquiry of identified and repaired injuries help anticipate further resuscitative needs. Institution-specific massive transfusion events (Fig. 20-1A), often activated preoperatively, should be continued intraoperatively, such that protocol-driven balanced volumes of red blood cells, plasma, and platelets are rapidly delivered to the patient when needed. Viscoelastic laboratory testing (Figs. 20-1B and C) and markers of anemia, coagulation, and

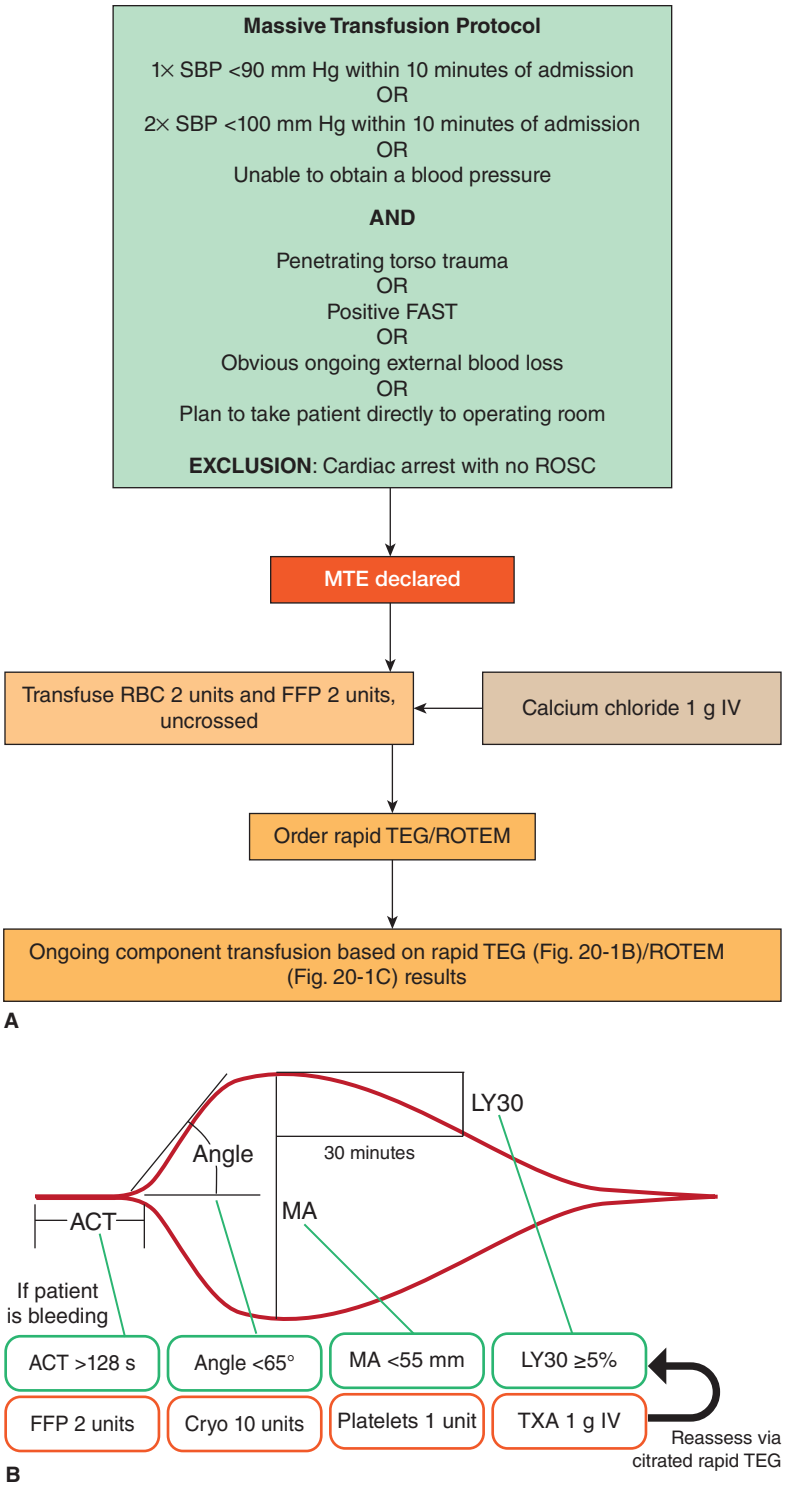
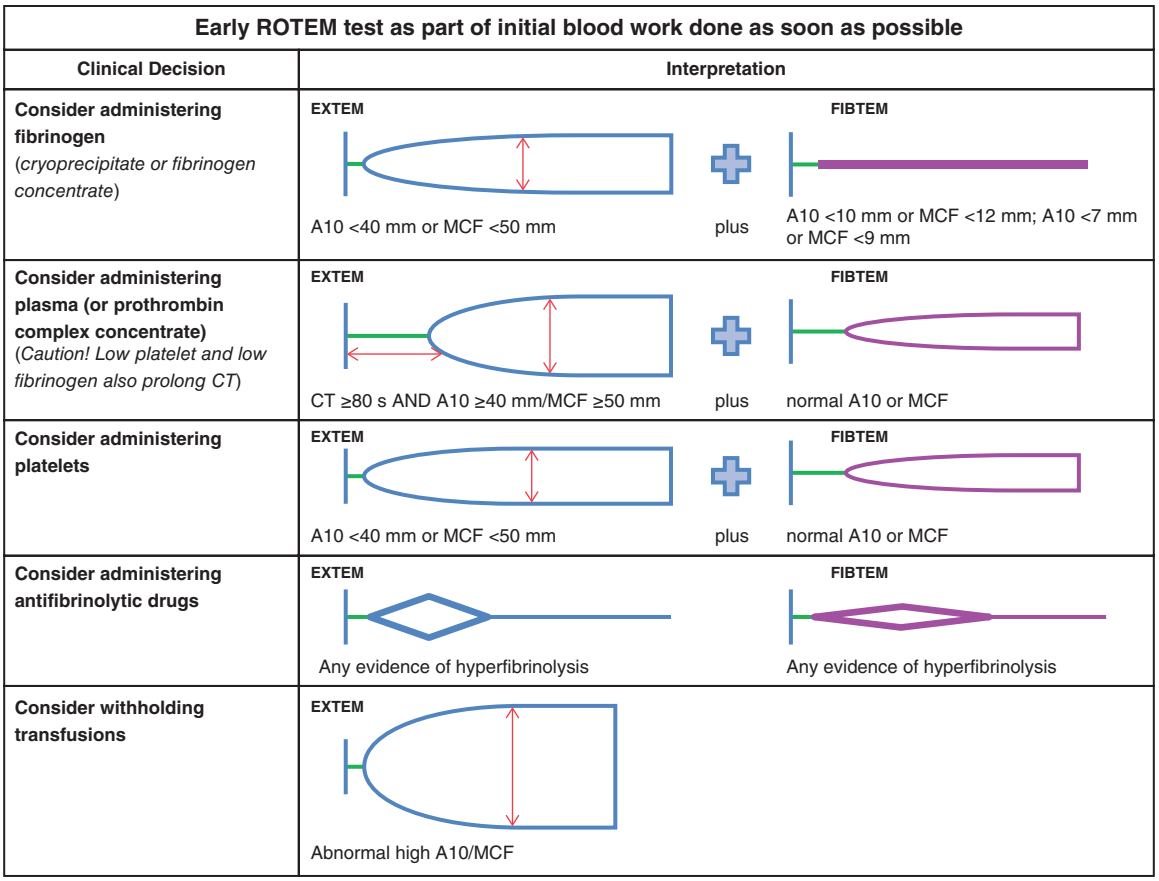


FIGURE 20-1 (**A**) Massive transfusion protocol. (**B**) Rapid TEG. (**C**) ROTEM. ACT, activated clotting time; CT, clotting time; Cryo, cryoprecipitate; EXTEM, extrinsically activated test with tissue factor; FAST, focused assessment with sonography in trauma; FIBTEM, fibrin-based extrinsically activated test with tissue factor and the platelet inhibitor cytochalasin D; FFP, fresh frozen plasma; IV, intravenous; LY30, clot lysis after 30 minutes; MA, maximum amplitude; MCF, maximum clot firmness; MTE, massive transfusion event; RBC, red blood cell; ROSC, return of spontaneous circulation; ROTEM, rotational thromboelastometry; SBP, systolic blood pressure; TEG, thromboelastography; TXA, tranexamic acid. (Reproduced, with permission, from Inaba K, Rizoli S, Veigas PV, et al. 2014 Consensus conference on viscoelastic test-based transfusion guidelines for early trauma resuscitation: report of the panel. *J Trauma Acute Care Surg.* 2015; 78:1220-1229.)



C

FIGURE 20-1 (*Continued*)

end-organ perfusion should direct appropriate intraoperative blood product transfusion, factor concentrates, and antifibrinolytics.[5] Although excessive crystalloid administration (ie, >2 L) should be avoided in an acutely injured trauma patient,[6] balanced crystalloid solutions are acceptable when necessary.[7,8]

Large-bore intravenous access is critical for successful resuscitation. When two peripheral intravenous lines (18-gauge or greater) cannot be obtained, an interosseous line can be placed until more definitive central venous access is secured. In the patient with multiple injuries, the insertion site for intravenous access should be in a location that will effectively restore intravascular volume and deliver medications to the systemic circulation without interfering with the operative procedure. It is routine in our institutions to insert large-bore intravenous access above the diaphragm in patients with intra-abdominal or pelvic injuries due to the risk of possible extravasation of much needed resuscitation fluid and supportive medications infused via femoral lines. Similarly, in patients with severe extremity injuries, intravenous access should be obtained at sites other than the injured extremity. Rapid infusion systems should be used when available to restore intravascular volume quickly. Furthermore, invasive hemodynamic monitoring with an arterial catheter is essential for the hypotensive trauma patient to provide continuous hemodynamic monitoring and frequent arterial blood gas measurements to assess metabolic status and resuscitative success. Invasive arterial access must also consider whether resuscitative endovascular balloon occlusion of the aorta (REBOA) (see Chapter 13) or eventual angiographic intervention will be necessary, because this will require arterial cannulation of specific vessels. The use of ultrasound frequently facilitates insertion of intravascular monitors and can be critical in severely hypovolemic patients, burn patients, or those with a history of intravenous drug use.[9]

True resuscitative goals and optimal hemodynamic and laboratory targets are hard to substantiate due to limited data. However, Dutton[10] has described early (prior to hemorrhagic control) and late (after surgical hemostasis) resuscitative objectives as a useful way of organizing intraoperative goals and treatment priorities (Table 20-1). More recent expert consensus opinion recommends a systolic blood pressure of 100 to 110 mm Hg until hemorrhage control is achieved.[11] The rate of volume resuscitation should be such that intravascular volume is administered in order to adequately support cardiac filling pressures, allow for sufficient end-organ perfusion, and support a mean arterial blood pressure (MAP) of at least 65 mm Hg.[12] Once hemostasis is obtained, it is reasonable to target a MAP of greater than 65 mm Hg because prolonged hypotension and hypoperfusion are associated with

TABLE 20-1: Early and Late Goals of Resuscitation

Resuscitation goals *prior to* hemorrhagic control	Resuscitation goals *after* surgical hemostasis
• Systolic blood pressure 90 mm Hg	• Systolic blood pressure >100 mm Hg
• Heart rate <120 bpm	• Heart rate <90 bpm
• Pulse oximeter functioning, Sao_2 >95%	• Pulse oximeter functioning, Sao_2 >97%
• Urine output present	• Urine output >0.5 mL/kg/h
• $Paco_2$ <50 torr	• $Paco_2$ <40 torr
• pH >7.25	• pH >7.35
• Hematocrit >25%	• Hematocrit >20%
• Lactate stable and decreasing	• Lactate normal
• Ionized calcium >1.0 mmol/L	• Ionized calcium >1.0 mmol/L
• International normalized ratio <1.6	• International normalized ratio <2
• Platelets >50,000/mm^3	• Platelets >50,000/mm^3
• Normothermia	• Normothermia
• Deep anesthesia	• Cardiac output normal or high
	• Light sedation (comfortable, able to initiate spontaneous ventilation)

$Paco_2$, partial pressure of carbon dioxide; Sao_2, oxygen saturation.

Source: Adapted with permission from Dutton RP. Damage control anesthesia. *International Trauma Care (ITACCS)*. Fall 2005;197-201. http://emcrit.org/wp-content/uploads/damage_controanesthesial.pdf. Accessed January 6, 2020.

postoperative organ dysfunction.[13] Metabolic indices must be routinely monitored (ie, every 30–60 minutes), and a successful resuscitation should include a pH approaching normal values (ie, pH >7.3),[14] decreasing serum lactate (ie, lactate clearance of 10% per hour),[15] base deficit greater than –2.0, partial pressure of carbon dioxide ($Paco_2$) of 35 to 40 torr, and a core body temperature of greater than 35°C.[16] Temperature management and avoidance of hypothermia are critical in an acutely injured trauma patient and may be achieved by active means such as fluid warmers or intravascular warming devices or with passive convection warming devices. Extracorporeal devices, such as extracorporeal membrane oxygenation or renal replacement therapy, may be used to rewarm patients with critical hypothermia (ie, temperature <32°C).

If cardiovascular physiology deteriorates as evidenced by continued bleeding in the operative field and/or acute hemodynamic decompensation, the anesthesia team should request for temporary surgical hemorrhage control with pressure packing, vascular clamps, or REBOA inflation until adequate intravascular volume is restored. Newer techniques, such as REBOA[17] (see Chapter 13) or emergency preservation and resuscitation trials,[18] carry associated physiologic impacts that require additional anesthesia education and collaborative management protocols.[19] Hypotension and bradycardia in an acutely hemorrhaging patient should alert the resuscitation team to imminent cardiovascular collapse. At times, intraoperative damage control techniques should be expedited, followed by resuscitation to reverse the shock in an intensive care unit (ICU), prior to surgical reexploration and definitive repair. When blood replacement needs exceed surgical blood loss, consideration of other sites of hemorrhage should be discussed and explored. For patients with a tourniquet in place, a pelvic binder, or inflated REBOA, the planned complete or partial release of hemorrhage control devices must be communicated to the anesthesiologist to allow for monitoring and treatment of sudden hypotension and negative inotropy from decreased preload and reperfusion of acidic metabolites. At times, vasopressor administration may be indicated. Although retrospective evidence has suggested that early vasopressor use following traumatic hemorrhagic shock is associated with increased mortality,[20] recent investigations suggest that norepinephrine administration does not adversely affect mortality.[21] Single-center, prospective, randomized data also demonstrated that continuous infusion of vasopressin to patients in hemorrhagic shock was associated with decreased transfusion requirements.[22] However, large doses of vasopressors should be critically evaluated and every effort made to restore intravascular volume prior to vasopressor use (Table 20-2).

While injury leading to hemorrhage is of primary focus, many other alterations in normal physiology can occur in the injured patient and must be rapidly diagnosed and treated in the operating room. For example, brain injury with consequential increases in intracranial pressure (ICP),[23] coagulopathy and arrhythmias, airway and thoracic injuries with subsequent difficulty oxygenating and ventilating, and spinal cord injuries with resultant hypotension unresponsive to volume replacement[24] must all be concurrently considered by the anesthesiologist and should dictate therapeutic interventions. Advanced Trauma Life Support training, education in management of nonhemorrhagic injury and morbidity (found in other chapters in this book), and simulation are vital in preparing the trauma anesthesiologist to manage the entire patient.

Finally, disposition from the operating room must also be formulated (ie, Does the patient require ICU admission? Is further imaging required? Does hemorrhagic control require damage control management via interventional angiography?). These decisions and communication should all occur in conjunction with the anesthesiologist, trauma surgeon, and critical care intensivist. The emphasis on perioperative vigilance as the core of anesthetic practice cannot be more accurate when caring for the multisystem severely injured trauma patient.

ANESTHETIC MONITORING AND PLANNING

Monitors and Machines

The American Society of Anesthesiologists (ASA) has established standards to ensure appropriate and safe physiologic monitoring during all anesthetics.[25] These include monitoring of oxygenation, ventilation, circulation and hemodynamics, and temperature. During general endotracheal anesthesia,

TABLE 20-2: Vasopressor Activity, Risks, and Benefits

Vasopressor	Mechanism of action	Risk	Benefit
Phenylephrine	Pure α_1-receptor agonist	Peripheral and splanchnic vasoconstriction, reflex bradycardia	Augment venous return and increase central systemic vascular compartment volume No proven benefit in hemorrhagic shock
Norepinephrine	α_1-Receptor agonist with modest β_1-agonist activity	Increased oxygen consumption, decreased regional perfusion due to excessive vasoconstrictive effects, immunosuppression	Augment venous return and increase central systemic vascular compartment volume, increase coronary artery perfusion, support cardiac contractility through β_1 activity
Epinephrine	β_1, β_2 activity at doses <5 µg/min, additional α_1 activity with higher doses	Arrhythmia, increased lactate, cardiac ischemia	Improve cardiac contractility, augment venous return, and increase central systemic vascular compartment volume
Dopamine	D_1 and D_2 receptors at doses of 0.5-3 µg/kg/min, β_1-receptors at doses of 3-10 µg/kg/min, α_1-receptor at doses >10 µg/kg/min	Arrhythmia, cardiac ischemia, vasoconstriction, and increased LV afterload at high doses	Theoretical improved coronary and renal blood flow at low doses No proven benefit in hemorrhagic shock
Vasopressin	0.03-0.04 U/min	Increased visceral ischemia due to splanchnic vasoconstriction and translocation of intestinal bacteria, exacerbation of cerebral edema, coronary ischemia due to vasoconstriction	Activation of V_1 receptors and counteracting nitric oxide synthesis, thereby increasing vascular tone, immunomodulation, regulation of intravascular volume resorption via V_2 receptors in collecting tubules, decreased transfusion requirement (blood product and overall volume)

LV, left ventricular.

oxygenation should be determined with continuous pulse oximetry and the fraction of inspired concentration of gases. Ventilation is monitored with continuous end-tidal carbon dioxide measurements and exhaled tidal volume. Systemic blood pressure is determined either via a continuous invasive arterial catheter or noninvasive blood pressure cuff measured at least every 5 minutes. Heart rate and rhythm are monitored with continuous electrocardiographic telemetry. Last, all patients should have temperature measurements obtained when significant changes in body temperature are expected to occur. This is especially crucial for trauma patients who may experience prolonged exposure to the elements prior to hospital arrival, burn patients, or those who undergo sizable open operations with large insensible losses of fluid and heat. Furthermore, exposure to large volumes of resuscitative fluids may cause profound hypothermia and dilutional coagulopathy. In summary, every anesthetist must maintain constant and vigilant monitoring of the previously mentioned essential vital functions.

The basic anesthesia machine is designed to safely and efficiently deliver a mixture of oxygen, air, and inhalational anesthetic. Principles of redundancy, both in machine function and monitors/alarms, are incorporated to aid in early detection, and user alerts indicate particular risks (eg, delivery of a hypoxic gas mixture or the loss of tidal volume resulting from a breathing circuit disconnection). Despite the many safety systems inherent to the modern operating room anesthesia machine, there remain circumstances when anesthetics must be administered in austere environments or outside of a designated operating room setting, without access to the usual anesthetic apparatus and its safety features. In these clinical situations, the ASA standards of anesthetic monitoring must still be considered, but in dire emergencies, the anesthesiologist may initially rely on tools such as clinical observation, palpation of pulse, and auscultation of heart and lung sounds before there is time for application of routine monitors. Capnography can provide timely insight into states of hypoperfusion because end-tidal carbon dioxide falls with profound decreases in cardiac output. Portable monitors that display electrocardiographic telemetry, pulse oximetry, invasive and/or noninvasive blood pressure, and end-tidal carbon dioxide are routinely used for transport of critically injured trauma patients and may substitute for the anesthetic machine monitor when providing remote anesthesia service. In addition, backup safety equipment, such as manual ventilation bag-valve mask and circuit, airway and intubating devices, vasoactive medications, and intravenous sedation medications, must be readily available in all anesthetic locations.

There is no consensus guideline for intraoperative hemodynamic monitoring and resuscitation in trauma patients. Although continuous cardiac output monitors appear attractive, there is little comparative data to demonstrate improved clinical outcomes in the trauma setting. As previously described, serum markers of resuscitation, such as lactate, base deficit, and venous oxygenation, are valuable tools to guide resuscitation.[14,26] However, each has certain limitations, as discussed in other chapters of this text. Last, the anesthesiologist may be specially trained and certified to

perform transesophageal echocardiography (TEE). Although this modality has not been formally validated for intraoperative care of trauma patients, TEE appears to be gaining prominence as a valuable tool to aid in hemodynamic resuscitation.[27]

Anesthetic Types

Anesthetics can be categorized into three types: general, regional, and local anesthesia. Each one can be administered independently or in combination with the others. When regional or local anesthesia is provided in the absence of general anesthesia, the patient may require supplemental light or deep sedation. The decision as to which anesthetic to provide the trauma patient is dependent upon numerous variables. Most critically is the urgency of the operation, potential likelihood of clinical deterioration due to severity of injury, and underlying patient factors, such as risk of aspiration and patient mental status. Severe traumatic brain injury (TBI), intoxication, and the stress of a traumatic incident often render patients unable to cooperate such that they pose a danger to themselves and surrounding providers. Rapid induction and intubation followed by general anesthesia with an inhalational or intravenous anesthetic is often the most effective and safe option in these patients with careful consideration of hemodynamics, oxygenation, and ventilation to minimize secondary brain injury. Finally, in the severely injured patient, definitive airway control and general anesthesia allow for a smooth transition to the ICU or further surgical interventions if required.

General anesthesia is not without certain side effects and disadvantages. Nearly all sedative medications and inhalational anesthetic agents are sympatholytic and directly produce vasodilation and hypotension (Table 20-3), further exacerbated by hypovolemia. Therefore, continued and aggressive resuscitation prior to induction and maintenance of a general anesthetic is critical to intraoperative stability and tissue oxygen delivery. In addition, the use of vasopressors to counteract anesthetic vasoplegia may be used, as described previously.

Trauma patients are also at increased risk of intraoperative awareness (recall) when general anesthetics are used sparingly due to hypotensive instability. Recognition of this risk is crucial to minimizing potential adverse neuropsychological effects. Appropriate supplementation of a general anesthetic with a benzodiazepine (ie, midazolam 0.02 mg/kg) to prevent memory formation may assist in limiting intraoperative recall. Furthermore, a high index of suspicion and close postoperative evaluation with appropriate neuropsychological follow-up are also recommended.[28]

In the absence of an indication for general anesthesia with a definitive airway, regional anesthesia techniques, combined with minimal or no additional sedation, may be a valuable approach, especially for patients whose injuries are primarily localized to the extremities. This can be particularly useful for fracture reduction and splinting, application of traction, pain relief before or after surgical repair, and occasionally to facilitate definitive repair of injuries. The profound analgesia that can be provided by peripheral nerve blockade can greatly reduce the need for systemic opioids and their side effects and alleviates some of the patient distress that might otherwise be addressed with sedative-hypnotic drugs.[29] Caution must still be exercised when formulating plans for any sedation accompanying regional anesthetic techniques, recognizing that offering concurrent sedation may impair airway reflexes and could warrant airway securement and conversion to a general anesthetic.

Anesthetic Considerations

In addition to the focus on maintaining safe physiology and resuscitative medicine, anesthesiologists consider a number of other factors, including the importance of anxiolysis, analgesia, hypnosis and amnesia, and immobility to facilitate safe and comfortable care. An overview of these anesthetic goals is presented in Table 20-4 and discussed in the following four sections. The accompanying Table 20-3 summarizes properties of several classes of anesthetic medications commonly used in trauma that are referenced across this chapter.

ANXIOLYSIS

In the setting of traumatic injury, patients often experience increased anxiety due to the psychological impact of circumstances that led to the injury, acute trauma pain, fear from impaired situational awareness, and simultaneous stimulation by multiple providers and procedures involved in their care. Unfortunately, for trauma patients who require prolonged stays in the hospital or ICU, increased anxiety is prevalent.[30]

Before resorting to anxiolytic drugs, the side effects of which may be undesirable in early resuscitation, basic principles of bedside manner go a long way in easing patient fear. Reducing anxiety by speaking in a calm voice, listening, providing clear explanations and reassurance about the care being rendered, providing positive imagery, having respectful interactions with other members of the care team, and attending to comfort can reduce drug administration.[31] When necessary, various medications are also used to aid in anxiolysis, with the benzodiazepine class most commonly prescribed.

Benzodiazepines stimulate inhibitory γ-aminobutyric acid (GABA) receptors in the central nervous system. Clinically, they produce a dose-dependent effect of anxiolysis, hypnosis, and antegrade amnesia.[32] Commonly prescribed benzodiazepine medications and their properties in the acute care and ICU setting are listed in Table 20-3. Midazolam is the most frequently used intravenous benzodiazepine for preoperative anxiolysis due to its short onset of action (1–2 minutes), relatively short duration of action, and minimal adverse cardiovascular effects when administered at a premedication anxiolytic dose. Of considerable importance in the trauma patient with hemorrhagic shock and hypovolemia is that rather modest doses of benzodiazepines may result in marked cardiovascular depression. Therefore, appropriate dose adjustment and reduction are often necessary when administering these medications to under-resuscitated patients.[33]

Other medications used to minimize anxiety and agitation include α_2-adrenergic agonists (eg, dexmedetomidine

TABLE 20-3: Anesthetic Medications in Trauma

Intravenous benzodiazepines

Drug	Onset (min)	Duration of action (min)	Common clinical uses in trauma patients	Drug-specific side effects and considerations	Drug class side effects and considerations
Midazolam	0.5–1.5	10–30	Anxiolysis, antegrade amnesia, sedation, component of induction, seizure cessation, infusion for ICU sedation	↓MAP and ↓CBF when used at induction doses	All benzodiazepines can cause respiratory depression (hypoventilation, hypoxia, apnea) and potentiate other CNS and respiratory depressants; recommend avoidance in early pregnancy
Lorazepam	1–2	60–120	Anxiolysis	Pain on injection, disorientation, abuse and addictive potential	
Diazepam	1–1.5	15–30	Anxiolysis, skeletal muscle relaxant, part of multimodal analgesia	↓MAP, pain on injection	

Intravenous opioids

Drug	Onset (min)	Duration of action	Common clinical uses in trauma patients	Drug-specific side effects and considerations	Drug class side effects and considerations
Morphine	5–10	3–5 h	Analgesia, sedation	Asthma exacerbation, histamine release, drug accumulation in renal dysfunction	All opioids can cause respiratory depression (hypoventilation, hypoxia, apnea), potentiate other CNS and respiratory depressants, and result in ↓MAP, ileus, nausea and vomiting, abuse and addictive behavior, and pruritus
Fentanyl	3–5	30–60 min	Analgesia, sedation, infusion	Long context-sensitive half-time; primarily metabolized by liver so useful in renal failure; can cause chest wall rigidity	
Hydromorphone	5–15	1–3 h	Analgesia (often postoperative)		
Remifentanil	1–1.5	5–10 min	Analgesia, sedation; usually given by infusion	Very short context-sensitive half-time	
Sufentanil	1–3	60–90 min	Analgesia	Greater respiratory depression than fentanyl at equianalgesic levels	
Alfentanil	0.5–2	15–45 min	Analgesia, especially for briefly painful procedures (eg, fracture reduction, dressing change)		
Methadone	3–5	24–36 h	Analgesia	Histamine release, prolonged QTc; also acts via NMDA receptor antagonism	

(*continued*)

TABLE 20-3: Anesthetic Medications in Trauma (*Continued*)

Local anesthetics

Drug	Duration of action (h)	Maximum safe dose	Common clinical uses in trauma patients	Drug-specific side effects and considerations	General side effects and considerations
Lidocaine	3–4	5 mg/kg plain 7 mg/kg with epinephrine	Single injection, intravenous infusions for multimodal analgesia, part of anesthetic induction	Least cardiotoxic; used for intravenous regional anesthesia (Bier block)	All local anesthetics can cause local anesthetic systemic toxicity: can lead to mental status changes, arrhythmias, and cardiac arrest; reversed with intralipid
Mepivacaine	3–4	4.5 mg/kg plain 7 mg/kg with epinephrine	Single injection		
Ropivacaine	6–10	3 mg/kg	Single injection, continuous neuraxial or PNB infusion		
Bupivacaine	8–12	2.5 mg/kg	Single injection, continuous neuraxial or PNB infusion	Most cardiotoxic	
Levobupivacaine	8–12	2.5 mg/kg	Single injection, continuous neuraxial or PNB infusion		

Intravenous hypnotics/sedatives

Drug	Onset (min)	Duration of action (min)	Common clinical uses in trauma patients	Drug-specific side effects and considerations
Propofol	<0.5	5–10	Anesthetic induction and maintenance, procedural and ICU sedation	Pain on injection; respiratory depression; ↓SVR, ↓CVP with induction doses causes hypotension; ↓ICP; risk of propofol infusion syndrome with prolonged ICU infusion
Etomidate	<0.5	3–12	Anesthetic induction	Pain on injection; respiratory depression; 11β-hydroxylase inhibition may result in cortisol insufficiency
Ketamine	0.5–1	10–20	Anesthetic induction (alone or in combination with another agent), procedural sedation, analgesia	Increases sympathetic nervous system output; increases oral and tracheobronchial secretions; bronchodilator; analgesic properties via NMDA receptor antagonism; direct myocardial depressant effects may become evident in catecholamine-depleted patients; disturbing hallucinogenic effects
Dexmedetomidine	5–15	Dose dependent; usually given by infusion	Sedation for awake intubation, controlled emergence, ICU sedation	No respiratory depression; no increase in ICP; biphasic BP response (possible brief hypertension followed by hypotension); bradycardia; risk for sinus arrest in patients with high vagal tone

(*continued*)

TABLE 20-3: Anesthetic Medications in Trauma (*Continued*)

Inhaled anesthetics

Drug	Used for inhalation induction?	Potency[a]	Cardiovascular effects	Bronchodilatory effect	General side effects and considerations
Isoflurane	No	Greatest	Decrease BP, prolong QTc, increase HR	Yes	No analgesic properties; all promote immobility; all can cause hypotension, nausea and vomiting, hyperkalemia, and malignant hyperthermia and potentiate nondepolarizing muscle relaxants
Sevoflurane	Yes	Intermediate	Decrease BP, prolong QTc	Yes	
Desflurane	No	Low to intermediate	Decrease BP, prolong QTc, increase HR	No	
Nitrous oxide	Yes, but only in combination with other inhaled agents	Least	Decrease BP, increase HR	No	Analgesia even at subanesthetic doses; increases CBF and ICP; causes nausea and vomiting; does not trigger malignant hyperthermia; lowest potency—must be combined with other agents to achieve appropriate anesthetic depth

Neuromuscular blocking drugs

Drug	Class	Onset (s)	Duration of action (min)	Drug-specific side effects and considerations
Succinylcholine	Depolarizing	30–60	4–6	Risk of hyperkalemia, malignant hyperthermia
Rocuronium	Nondepolarizing, aminosteroid	60–120	30–60	Suitable for rapid sequence intubation with doses of 1.2 mg/kg; reversible with acetylcholinesterase inhibitors or sugammadex; can induce allergic reaction
Vecuronium	Nondepolarizing, aminosteroid	120–180	25–40	Reversible with acetylcholinesterase inhibitors or sugammadex
Cisatracurium	Nondepolarizing, benzylisoquinolinium	120–180	30–60	Elimination independent of renal or hepatic function; reversible with acetylcholinesterase inhibitors, but not with sugammadex

[a]Potency is a function of blood solubility of inhaled anesthetics and is inversely related to onset and elimination time.

BP, blood pressure; CBF, cerebral blood flow; CNS, central nervous system; CVP, central venous pressure; HR, heart rate; ICP, intracranial pressure; ICU, intensive care unit; MAP, mean arterial pressure; NMDA, *N*-methyl-D-aspartate; PNB, peripheral nerve block; QTc, corrected QT interval; SVR, systemic vascular resistance.

and clonidine), antihistamines (eg, diphenhydramine), and antidopaminergic medications (eg, haloperidol). However, their pharmacokinetic and pharmacodynamic properties differ from benzodiazepines and are less commonly used as anxiolytics in the preoperative setting.

ANALGESIA

The International Association for the Study of Pain defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."[34] Naturally, trauma patients often present with significant pain from tissue injury and inflammation, which is accompanied by increased sympathetic nervous system activity, reflexive movement, and emotional distress. Additional discomfort may result from comprehensive physical examination and invasive procedures (eg, supine positioning on hard backboard, vascular access, tube thoracostomy, injury assessment, fracture reduction, traction pin

TABLE 20-4: Overview of Anesthetic Goals

Goal	Nonpharmacologic tools or coexisting conditions affecting goal	Pharmacotherapy
Anxiolysis	• Calm demeanor • Empathetic listening • Explanations • Set expectations • Reassurance • Distraction, positive imagery • Attend to comfort (warm blankets, positioning, cover exposed areas)	• Benzodiazepines: midazolam, diazepam, lorazepam (and antagonism with flumazenil) • α_2-Agonists: dexmedetomidine, clonidine • Antihistamines: diphenhydramine • Antidopaminergics: droperidol, haloperidol • Anesthetic induction agents at low dose (eg, low-dose propofol infusion, low-dose ketamine) • Nitrous oxide
Analgesia	• Attend to comfort (especially positioning) • Set expectations; acknowledge expected degree of pain from injury or planned intervention • Homeopathic techniques • Ice • Distracting stimulus • Coexisting nerve injury or spinal cord injury may alter patient's perception of tissue injury	*Systemically administered drugs* • Acetaminophen • Nonsteroidal anti-inflammatory drugs (NSAIDs) • Steroids • Mu-opioid agonists (morphine, hydromorphone, fentanyl, alfentanil, sufentanil, remifentanil, meperidine, methadone, oxycodone, hydrocodone) (and antagonism with naloxone) • NMDA antagonists (ketamine, methadone) • Nitrous oxide *Local anesthetics* • Ester class (eg, procaine, chloroprocaine, tetracaine, benzocaine) • Amide class (eg, lidocaine, bupivacaine, mepivacaine, ropivacaine, levobupivacaine) *Location of local anesthetic deposition:* • Topical (mucosa or skin) • Infiltration/field block • Regional block (particular nerve or plexus) • Neuraxial block (epidural or spinal [subarachnoid])
Amnesia/ hypnosis	• Distraction • Traumatic brain injury may affect doses of agents needed to attain amnesia/hypnosis	Amnesia, potentially without hypnosis: benzodiazepines (especially midazolam), propofol (especially low dose) Sedation, potentially without amnesia: α_2-agonists (eg, dexmedetomidine) Hypnosis (impairment/loss of consciousness, generally accompanied by amnesia at anesthetic doses): • IV anesthetic agents: propofol, thiopental, methohexital, etomidate, ketamine, benzodiazepines (high dose) • Inhaled anesthetics: nitrous oxide, halothane, isoflurane, sevoflurane, desflurane

Immobility	• Verbal cues: "hold still"
	• Physical restraint
	• Attend to comfort (see above)
	• Coexisting nerve injury or spinal cord injury may impair neuromuscular function distal to injury
		• Deep anesthesia from any agent or combination (intravenous or inhaled anesthetics)
		• Opioids: sufficient doses suppress movement in response to noxious stimulus
		• Regional anesthesia with motor block
		• Neuromuscular blocking drugs
		  • Depolarizing class: succinylcholine
		  • Nondepolarizing class: vecuronium, rocuronium, atracurium, cisatracurium, pancuronium
		  • Reversal agents:
		  • For depolarizing class: none
		  • For nondepolarizing class: acetylcholinesterase inhibitors (neostigmine, edrophonium)
		  • For rocuronium and vecuronium: sugammadex

(*continued*)

TABLE 20-4: Overview of Anesthetic Goals (*Continued*)

Goal	Nonpharmacologic tools or coexisting conditions affecting goal	Pharmacotherapy
Resuscitation: monitor and maintain safe physiology and mitigate drug side effects	*Monitors* Noninvasive: visual inspection, palpation of pulse, auscultation of heart/lung sounds, blood pressure cuff, pulse oximetry, electrocardiogram, temperature, airway pressure, end-tidal gas (oxygen, carbon dioxide, anesthetic vapor), muscle paralysis, bispectral depth of anesthesia monitoring, point-of-care ultrasound (eg, color flow Doppler, lung scanning, transthoracic echocardiography), estimated blood loss Invasive: oral/nasal airways, tracheal intubation, arterial blood pressure, central venous pressure, cardiac output monitoring, transesophageal echocardiography, intracranial pressure, cerebrospinal fluid drains, urinary catheters *Therapies* Suction, active warming devices (air and fluid based), position changes (eg, reverse Trendelenburg for high intracranial pressure), DC cardioversion or pacing, pump oxygenators, vascular occlusion balloons, one-lung ventilation	*Resuscitation and optimization of oxygen delivery to end organs* • Oxygen • Fluid therapy: crystalloids, colloids, blood components • Vasoactive and inotropic drugs: direct- and indirect-acting agonists and antagonists of α_1, α_2, β_1, β_2, vasopressin receptors • Bronchodilators: antimuscarinics, β_2-agonists, steroids, epinephrine, inhaled anesthetics (except nitrous oxide) • Pulmonary vascular resistance: inhaled nitric oxide, prostaglandins *Optimization of cerebral oxygen delivery (especially with presence of intracranial hypertension)* • Hemodynamic regulation as above • Osmotic agents, loop diuretics (controversial; see Chapter 22) (in addition to nonpharmacologic approaches) • Anticonvulsants *Infection prophylaxis* • Euglycemia (insulin and dextrose) • Prophylactic and therapeutic antibiotics *Side effect treatment and prophylaxis* • Allergy: antihistamines (H_1 and H_2), epinephrine, steroids • Nausea: antidopaminergics, 5-HT_3 antagonists, antimuscarinic (transdermal scopolamine), steroids • Antiarrhythmics *Management of physiology with resuscitative endovascular balloon occlusion of the aorta (REBOA)* *Advanced Cardiac Life Support management* *Participation in emergency preservation and resuscitation (EPR) protocols*

DC, direct current; NMDA, *N*-methyl-D-aspartate.

applications, endotracheal intubation, surgery). Beyond the obvious benefits of improving patient comfort and distress, early treatment of pain may facilitate physical examination and therapeutic interventions and diminish the need for levels of sedation that could further impair physiology.

However, not all pain is physiologic. Complex psychological and behavioral factors, as well as past experiences and tolerances of prescribed or illicit analgesics, will alter a patient's reaction to painful stimuli, thereby requiring strategic assessment and treatment plans. Analgesic approaches should not only interrupt the transmission of noxious stimuli to the central nervous system, but also address the patient's physiologic and emotional experience of that nociception. Similar to anxiolysis, nonpharmacologic means of addressing pain are paramount, especially in the pediatric trauma population.[35] Allowing parents or other close allies of young children at the bedside can reduce stress and decrease painful experiences. Distraction techniques including cognitive interventions (eg, non–procedure-related conversation, music listening, video watching or playing, meditation) and behavioral techniques (eg, breathing exercises, squeezing an object) have all shown benefit.[36-38]

Outside of the operating room and postanesthesia care unit, pain management is generally provided by the surgical service or intensivist, while anesthesiologists manage all intraoperative and immediate postoperative pain needs. To provide continuity of care, information about patient analgesic needs should be shared among providers, and unusual tolerance or sensitivity to traditional medications and doses must be communicated.

Pharmacologically, opioids remain the cornerstone of analgesic treatment for acute trauma patients. They bind to mu, kappa, and delta opioid receptors in the peripheral and central nervous systems, resulting in a decreased response to noxious stimuli. Analgesic efficacy is often rapid and profound. Morphine, fentanyl, remifentanil, alfentanil, sufentanil, hydromorphone, and methadone are among the most common of the opioid class to be administered, and their specific properties, common uses, and considerations for trauma patients are listed in Table 20-3. When prescribing opioids, patients who are chronically dependent on opioids should be provided their prehospital daily doses with additional short- and long-acting analgesics required to treat the added acute pain burden. For acute trauma and surgical pain, a reverse

analgesic ladder, whereby diminishing doses of strong analgesics are prescribed as recovery occurs, is recommended.[39]

However, opioids alone are inadequate to treat pain and are relatively inefficient at managing neuropathic symptoms. They are associated with a long list of common deleterious side effects (eg, nausea, ileus, itching, sedation, dysphoria, respiratory depression), have a ceiling effect, and ironically can lead to the development of hyperalgesia. In the hypovolemic bleeding patient or a patient at high risk of respiratory failure (eg, rib fractures, pulmonary contusion, hemo- or pneumothorax), even small doses can lead to cardiovascular collapse or severe respiratory compromise.

Naloxone produces reliable antagonism of the narcosis and respiratory depression produced by opiate agonists. However, its short duration of action dictates close monitoring should re-narcotization occur due to longer-acting opioids. Furthermore, opioid antagonism requires slow dilute boluses of naloxone because rapid and full reversal of the opioid-induced analgesia can lead to extreme pain and massive sympathetic discharge sufficient to produce cardiac arrest.

More concerning perhaps is the large percentage of patients who survive their injuries but misuse or develop long-term dependence on opioid medications. Attempts to diminish side effects and curb the global opioid epidemic has led to innovative methods to treat pain. Multimodal analgesia by combining differing analgesic drugs and techniques to target a variety of pain receptors is encouraged to synergistically diminish pain scores and side effects at lower drug doses. Nonopioid analgesics, such as acetaminophen, nonsteroidal anti-inflammatory drugs, gabapentinoids, anticonvulsants, serotonin-norepinephrine reuptake inhibitors, ketamine infusions, and local anesthetics for neuraxial or peripheral nerve blockade, for intravenous infusion, or in transdermal patches (lidocaine), are increasingly being used to complement or, at times, replace opioid analgesics. Commonly used local anesthetics are summarized in Table 20-3. Although emerging data demonstrate superior pain relief, decreased opioid consumption, and shorter hospital stays when multimodal analgesia is implemented, more research is needed to determine improvement in long-term functional outcomes and differences in chronic opioid dependence and addiction.[40,41]

HYPNOSIS AND AMNESIA

Primary objectives of sedation or general anesthesia are to provide psychological comfort for the patient via hypnosis (depressed consciousness or sleep) and amnesia. It is physiologically possible to have amnesia without hypnosis (as is observed with the antegrade amnesia midazolam produces in conscious patients) and, conversely, to have hypnosis without amnesia (as can unfortunately happen with light anesthesia resulting in patient awareness). Patients may also present under the impairment of various substances that alter normal neurologic and psychologic function, and these factors are considered when selecting, dosing, and monitoring the effects of hypnotic drugs. Sedation may be achieved via multiple possible agents. The previously discussed classes of benzodiazepines and opioids produce sedative effects at higher than amnestic and analgesic doses, respectively, but are rarely used in isolation for these purposes in the operating room.

The most commonly used intravenous agents for sedation or induction of general anesthesia in trauma patients are propofol, etomidate, and ketamine (Table 20-3). Propofol is a lipid-soluble compound that, when administered as a bolus dose, is short lived. Significant dose reduction is critical when propofol is administered to patients with hypovolemia or shock or in patients of advanced age or frailty.[33] Etomidate is an imidazole-containing intravenous anesthetic that, unlike propofol, will induce sedation with relatively minimal hemodynamic side effects. Due to the favorable hemodynamic profile, etomidate would appear to be the ideal drug to provide safe and effective sedation in critically ill trauma patients. However, etomidate possesses intrinsic activity that inhibits 11β-hydroxylase in the synthesis of cortisol and therefore results in cortisol insufficiency. Previous retrospective investigations have noted that administration of etomidate to critically injured trauma patients is associated with acute respiratory distress syndrome.[42] A small, single-center, prospective study also demonstrated that etomidate caused an immediate decrease in cortisol levels in trauma patients and that patients who received etomidate required longer ICU and hospital lengths of stay.[43] Ketamine is a water-soluble compound with a mechanism of action that is related to its antagonism of the *N*-methyl-D-aspartate (NMDA) receptor and, as such, can also provide analgesia. Due to its neuropsychological stimulant property, ketamine was historically contraindicated in patients with TBI. However, evidence suggests that ketamine may be safely used with propofol as a total intravenous anesthetic in patients with TBI.[44] Although ketamine possesses direct negative inotropic effects, these are usually superseded by its sympathomimetic activity, often resulting in tachycardia and hypertension. This property of ketamine makes it an attractive option for administration in the hypovolemic, hypotensive trauma patient who requires immediate sedation and control of cardiorespiratory function. However, caution is advised not to ignore ongoing hemorrhage and hypovolemia in the setting of a pharmacologically induced elevation in blood pressure. In addition to the generally favorable hemodynamic profile, ketamine is a potent bronchodilator and is useful in patients with reactive airway disease.

Most trauma patients are anesthetically induced with intravenous medications, as described earlier. However, in pediatric trauma patients or patients with difficult airways, anesthetic gases may be administered to anesthetize a spontaneously breathing patient prior to advanced airway management techniques. Following induction, maintenance of general anesthesia is often sustained with inhaled gases, administration of intravenous drugs, or a combination thereof. Modern inhaled anesthetic agents, summarized in Table 20-3, exert a dose-dependent suppression of consciousness, memory formation, and movement. Apart from nitrous oxide (N_2O), this class consists of nonflammable halogenated ethers delivered by agent-specific vaporizers. Despite decades of research and over 150 years of clinical use,

the exact mechanism of inhaled anesthetics' action remains unknown.[45] Advantages of this drug class include a long history of safety, familiarity with the pharmacodynamics, and the convenience of rapid titration and monitoring of inspired and expired gas concentrations. The primary disadvantage is the degree of vasodilation that accompanies clinical doses of inhaled anesthetics. Hypotension and end-organ hypoperfusion are significant risks and can exacerbate shock in hypovolemic trauma patients. Dosing should be adjusted to promote optimal organ perfusion and oxygen delivery and often requires concurrent administration of vasoactive infusions, intravenous fluids, blood products, and supplemental intravenous anesthetic and analgesic agents.

IMMOBILITY

Most tracheal intubations and surgical operations require a patient to be immobile, both for safety and to facilitate operative progress. Multiple methods prevent movement, with neuromuscular blocking drugs being the most frequent drug class employed. Movement is also suppressed by deep anesthesia with any anesthetic agent and by use of systemic analgesics and local anesthetics to blunt the physiologic response to noxious stimuli. Regional anesthesia techniques that block motor nerves can also be of utility in preventing movement in particular muscle groups.

Neuromuscular blocking agents are classified into two broad categories: depolarizing (eg, succinylcholine) and nondepolarizing (eg, rocuronium). These are summarized in Table 20-3. Neither class produces any analgesic or sedative effects. Succinylcholine structurally consists of two linked acetylcholine molecules and binds to nicotinic acetylcholine receptors to cause rapid muscle depolarization. Generalized muscle fasciculations manifest within 30 to 60 seconds after an intubating dose, with subsequent flaccid paralysis lasting about 5 minutes. Succinylcholine transiently increases intraocular pressure and is contraindicated in patients with an open globe injury. Similarly, because succinylcholine may briefly increase ICP, questions can be raised about its safety in acute TBI. However, inadequately paralyzed patients may cough or be difficult to intubate, potentially leading to hypoxia, with either condition causing significant increases in ICP. Given these considerations, succinylcholine remains a reasonable option in rapid sequence intubation induction for TBI patients.

Nondepolarizing agents, such as rocuronium, vecuronium, and cisatracurium, competitively bind the acetylcholine receptors at the motor endplate and inhibit depolarization. Thus, no muscle fasciculations occur. Rocuronium achieves rapid and effective intubating conditions within 60 to 90 seconds, but given its slow metabolism, intubating doses may last as long as 1 hour. Vecuronium and cisatracurium have a slower onset, not suitable for rapid intubation. Cisatracurium, due to its elimination being independent of renal or hepatic dysfunction, is most commonly selected in patients with renal failure or as a continuous infusion in the ICU to achieve chemical paralysis in some patients with acute respiratory distress syndrome.[46]

Prior to extubation, patients must demonstrate appropriate neuromuscular function for adequate ventilation and airway protection. Whereas succinylcholine is rapidly metabolized and diaphragm strength returns within minutes, nondepolarizing neuromuscular blockers often require pharmacologic antagonism with acetylcholinesterase inhibitors (eg, neostigmine) or selective relaxant binding agents (eg, sugammadex) to hasten their spontaneous reversal. Acetylcholinesterase inhibitors increase the amount of acetylcholine available at the motor endplate to overcome the nondepolarizing neuromuscular blockers' competitive inhibition of the acetylcholine receptor. Adverse effects relate to parasympathetic activity of acetylcholine at muscarinic receptors, with potential for bradycardia, diarrhea, and bronchorrhea, typically counteracted by coadministration of a muscarinic anticholinergic drug (eg, glycopyrrolate or atropine).

Sugammadex is the first drug in a new class of selective relaxant binding agents; it binds to and irreversibly encapsulates rocuronium and, to a lesser extent, vecuronium, preventing their action at the neuromuscular junction. Sugammadex does not reverse cisatracurium. Sugammadex is especially useful in clinical scenarios where a deep neuromuscular blockade has been induced by rocuronium and rapid antagonism is necessary for prompt recovery of neuromuscular function.[47]

SPECIAL CONSIDERATIONS

Aside from resuscitation and standard anesthesia provisions, there are several unique considerations that may demand added attention and anesthetic modifications in the acute trauma patient. Detailed management of these concerns is found in other chapters of this text; however, given the anesthesiologist's role in leading the critical care efforts in the operating room, the surgical practitioner should be aware of several circumstances that may impact surgical planning and care.

Acute trauma patients are considered "full stomach," and therefore, rapid sequence induction and intubation is necessary when definitive control of the airway is indicated.[48] In the event of a traumatic or difficult airway or cervical spine injury with developing prevertebral edema, anesthesiology should request for surgical airway standby at the bedside and have the appropriate equipment and resources to secure the airway quickly and safely. Brief discussion should ensue between the anesthesiologist and surgeon to review the expected roles and action necessary should an immediate surgical airway be needed. Thoracic injuries that require a thoracotomy for hemothorax or repair of lung injury or intercostal vessels may benefit from one-lung ventilation to improve surgical exposure and definitive hemorrhage control. Decision to insert a large double-lumen endotracheal tube (ETT), to insert a single-lumen ETT and place a bronchial blocker, or to advance a single-lumen ETT to a particular bronchus will depend on the patient's airway assessment, the skill set and experience of the anesthesiologist, and the ability of the patient to tolerate one-lung ventilation. With various factors increasing ventilation-perfusion mismatch in these patients, modifications of one-lung ventilation may be intermittently required to avoid significant hypoxia and hypoventilation. Modifications include increasing the fraction of inspired

oxygen, endotracheal suctioning, confirmation of tube position with fiberoptic bronchoscopy, increasing positive end-expiratory pressure to the ventilated lung, adding continuous positive airway pressure to the nonventilated lung, and occasionally reinstitution of double-lung ventilation. Communication between the operative surgeon and anesthesiologist is critical to balance the need to visualize and address bleeding and injury with the requirement to maintain adequate oxygen saturation and carbon dioxide levels.

In patients who incur TBI, intraoperative management of elevated ICP may necessitate elevation of the head of the bed, hyperosmotic infusions, coagulopathy treatment, and a targeted cerebral perfusion pressure between 60 and 70 mm Hg. Indications of clinically intractable increases in ICP should be communicated to the surgeon with consideration of additional measures (eg, craniectomy, decompressive laparotomy) and termination of non–life-threatening procedures.[49] Primary brain surgery or other head and neck surgery may be best performed with the patient's head and airway positioned away from the anesthesiologist and anesthesia machine. Taking the necessary time to organize and secure connections of an extended breathing circuit, ensure access to intravenous lines, and be able to administer vasoactive and blood products is critical and can be lifesaving. Similarly, prone positioning for spinal surgery requires a coordinated effort for securing the airway (suturing the ETT to the teeth or lip) and intravascular lines, flipping the patient without dislodging or malpositioning life support devices, reapplying monitors quickly, and having the ability throughout the operation to rapidly flip back supine onto a nearby bed in situations of a lost airway or significant cardiovascular instability or arrest. Prone position is also associated with postoperative visual loss, and the anesthesiologist must be vigilant of factors that are associated with increased risk, such as large blood loss, hypotension, large-volume crystalloid resuscitation, and prolonged anesthetic time.[50]

Spinal surgeries that call for particular neurologic monitoring may indicate specific anesthetics. Motor evoked potentials require that a patient not receive intermediate- or long-acting neuromuscular blockade to evaluate central nervous system motor function intraoperatively. The maintenance anesthetic should rely primarily on an intravenous technique that does not alter the amplitude and latency of neurologic monitoring signals. Somatosensory evoked potentials that measure activity in the cerebral cortex will afford the administration of full neuromuscular blockade and volatile anesthetics.

Pediatric and geriatric trauma patients possess substantial deviations from what is considered "normal adult" cardiovascular, pulmonary, hepatic, and renal physiology. They may respond to volume depletion with precipitate hypotension, apnea is poorly tolerated, and drug clearance may be unpredictable (it is generally reduced) in the presence of immature or senescent hepatorenal function. They share an inability to maintain normal body temperature under conditions of stress, and an impressive coagulopathy may develop in the presence of hypothermia and bleeding. With profound differences in physiology, anesthetic tolerance, and equipment needed for pediatric trauma patients, anesthesiologists who routinely care for adults should seek assistance from their pediatric anesthesiology–trained colleagues when caring for injured children.

POSTOPERATIVE MANAGEMENT

The acute postoperative management of the trauma patient is extremely variable due to many factors. Analogous to successful discharge planning beginning on admission, anticipating postoperative needs should begin in the preoperative and intraoperative phases. In fact, consideration of postoperative care may dictate modifications to preoperative and intraoperative management. For example, intraoperative management of spinal injuries may necessitate placement of arterial and central venous catheters to ensure adequate MAPs during surgery and postoperatively.[24] Multiple rib fractures or limb salvage surgery should suggest consideration of a regional anesthesia technique to potentially favor early extubation and decrease opioid requirements. In austere environments, valuable resource utilization both intra- and postoperatively may champion nerve blocks as the surgical anesthetic of choice.

When transitioning out of the operating room, the evolutionary nature of traumatic injury and changing patient needs warrant frequent discussion between trauma and anesthesia providers. Dynamic modifications are required to enable unimpeded flow and optimized management for the next phase of care.[51] Factors considered include the severity of injury, expected resources needed for continued fluid/blood management and pharmacotherapy to meet target resuscitative end points, the nature of the surgery performed, monitoring needs for early detection and immediate treatment of potential complications or decompensation, cardiovascular and pulmonary support, plan for repeat surgical procedure, ability to protect airway, postoperative pain management, and, at times, availability of beds.

Planning disposition from the operating room requires differentiating patients who are suitable for a controlled extubation and emergence with transfer to the postanesthesia care unit from those who need further intervention in an angiography suite, and still others who need to complete the formal trauma evaluation and receive ongoing aggressive resuscitation in a multitrauma ICU or transfer to a higher care facility. Maintaining resuscitative activities during transport to the next point of care is a key responsibility. In addition, as for any postanesthesia patient, the anesthesiologist manages any immediate postoperative complications and leads the formulation of pain management plans. Strong coordination and protocol-driven hand-off communication during transfer of care are essential to ensure the safe and effective transition from the intraoperative to postoperative phase.[52]

Acute Pain Management

Postinjury and postsurgical pain management can vary significantly from no analgesic requirements to indications for a complex multimodal pain management regimen with

continuous neuraxial or peripheral nerve blockade. Adequate pain management following trauma is an important component of healing and recovery. Undertreated pain is a source of unnecessary human suffering and anxiety and can cause acute alterations in the immune response, coagulation cascade, and patient vital signs with potentially damaging consequences in an already compromised trauma patient.[53] If left untreated, acute trauma pain can lead to the development of debilitating posttraumatic pain syndromes.[54] While opioids remain a large component of acute pain management, there has been a concerted effort to decrease reliance on these drugs due to the global opioid epidemic and its deleterious side effects.[55] Moreover, many of these drugs have limited effectiveness due to preinjury substance dependence and tolerance even at high doses. Multimodal analgesic plans with limited opioids are favored especially in high-dose opioid-tolerant patients and in those who experience profound clinical side effects from narcotics. Intravenous ketamine and lidocaine infusions often infused intraoperatively can be continued in analgesic doses postoperatively. Combining acetaminophen, nonsteroidal anti-inflammatories, gabapentinoids, muscle relaxants, antidepressants, anxiolytics, and regional anesthesia techniques, when appropriate, provides the best pain relief and decreases the risk of side effects and addictive-forming behavior.[41] Enhanced recovery after surgery (ERAS) protocols have assisted in standardizing these multimodal methodologies.[56]

Over the past few decades, dedicated regional anesthesia and acute pain medicine (RAAPM) services have proliferated and should be sought to provide an increasing array of analgesic options. Whereas most anesthesiologists are trained in spinal and epidural blocks, RAAPM physicians are skilled in many additional single-shot and continuous catheter techniques that allow for significantly improved pain scores and possibly improved functional outcomes. Mild analgesia to dense anesthesia can be accomplished with the administration of local anesthetics (Table 20-3) on spinal, epidural, and peripheral nerves. Amino amides such as lidocaine, mepivacaine, ropivacaine, levobupivacaine, and bupivacaine all provide sodium channel blockade and inhibit action potentials of afferent nerves with varied clinical results due to their different physiochemical and pharmacodynamic properties. Caution must be taken to limit cumulative local anesthetic administration to below toxic doses because local anesthetic systemic toxicity (LAST) can lead to profound neurologic symptoms and fatal cardiovascular collapse. In fact, expert opinion advocates for incorporating the maximum allowed dose of local anesthetic during the surgical time-out to prevent overdose by multiple provider administration.[57] Accessibility to intravenous intralipid to treat LAST is necessary wherever local anesthetics are routinely used.

The implementation of safe and effective regional anesthesia techniques in trauma patients has gained momentum due to the growing knowledge and training of ultrasound-guided techniques.[29] For various approaches to the brachial plexus for upper extremity blocks, blockade of branches of the lumbosacral plexus (most notably the femoral and sciatic nerves) for the lower extremity, and many recently described interfascial plane blocks (pectoralis I, pectoralis II, serratus anterior muscle plane, erector spinae, rectus abdominis, transversus abdominis plane, quadratus lumborum) that target thoracic, abdominal, and pelvic pain, several reports of these procedures show significant benefits in the trauma population.[58,59] Aside from superior analgesia and improved patient satisfaction scores, regional techniques in the acute trauma patient have shown benefits in reducing the incidence and severity of chronic pain syndromes, decreasing side effects from other modalities, and possibly improving surgical outcomes in vascular injuries and repair via sympathetic blockade, and earlier discharge and facilitation of transport.[60-62] Although a full discussion of the risks, benefits, indications, and contraindications of regional anesthesia procedures is beyond the scope of this chapter, there are some unique conditions in trauma patients worth mentioning.

Multiple Rib Fractures

Multiple rib fractures can induce significant pain and impair pulmonary function with resultant risks of pneumonia, acute respiratory failure, chronic pain, and disability. Thoracic epidural analgesia, once considered the gold standard, has not demonstrated definitive mortality benefit, decreased length of stay in the ICU or hospital, or a decrease in ventilator days in large meta-analyses.[63] Nonetheless, it remains commonly practiced to decrease pain scores and provide improved patient satisfaction. Treatment plans for patients with epidurals require attention to hypotension from the sympathectomy-induced vasodilation and modification in thromboprophylaxis schedules to decrease the risk of an iatrogenic epidural hematoma. Alternative techniques include thoracic paravertebral, intrapleural, or intercostal blocks as well as novel fascial plane blocks (serratus anterior plane and erector spinae plane blocks), which may offer comparable benefits with fewer side effects. However, large prospective studies are still lacking. For best results, multidisciplinary protocols with strategical clinical pathways and a multimodal approach are strongly advised to improve outcomes and limit complications.[64]

Compartment Syndrome

Some injury patterns (high-energy musculoskeletal or vascular injuries) and orthopedic repairs (eg, tibial plateau or radial fracture repairs) can cause immense pain but also risk development of an acute compartment syndrome (ACS) with potentially devastating loss of function or limb if not detected early and treated emergently. Management of pain in these patients can be challenging as dense analgesia via peripheral or neuraxial blocks or via intravenous opioids carries the potential to mask the first signs of ACS—disproportionate pain and changes in neurologic function. With conflicting arguments as to the value of a comfortable patient with an impaired motor and sensory exam versus a more uncomfortable patient who can rapidly report an increase in pain and/or diminished function, direct communication between

surgeons and anesthesiology should occur with attention to vigilant serial neurologic exams, dilute and short-acting local anesthetic infusions that when paused allow for rapid return of nerve function, and the ability to perform emergent fasciotomies for high-risk patients.

Other reasons to consult the RAAPM service include their expertise with difficult-to-manage pain patients, high-dose opioid titration, transition to oral doses, discharge planning, and weaning schedules. RAAPM specialists can help achieve decreased pain scores and set reasonable functional expectations. Consideration of alternative therapies such as acupressure, acupuncture, music and art therapy, mirror therapy for acute amputees, and relaxation techniques is advisable and can decrease pharmacologic requirements. Unfortunately, posttraumatic and postsurgical pain can continue and sometimes worsen upon discharge. Appropriate counseling and short-interval follow-up will allow for serial evaluation of a patient's pain management needs. If patients require more than 90 mg of oral morphine equivalents a day upon discharge, follow-up with an outpatient pain specialist or perioperative surgical home clinic is recommended and should be scheduled prior to discharge.[65,66]

SUMMARY

The role of the anesthesiologist in trauma care has evolved significantly to function as a resuscitationist with the expertise to tailor a safe anesthetic and analgesic plan in the setting of an altered physiology due to trauma. Comprehensive and often rapid pre-, intra-, and postoperative planning and adaptive care are true hallmarks of trauma anesthesia practice and are paving the way for subspecialty training in trauma and acute care anesthesiology.[67] Direct and ongoing collaborative efforts between anesthesiologists, trauma surgeons, emergency medicine physicians, critical care physicians, and allied members of the trauma team remain the optimal mechanism to provide best practice and improve patient outcomes.

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21

Infections

Heather L. Evans • Eileen Bulger

KEY POINTS

- *Staphylococcus aureus*, the most common pathogen associated with surgical site infections, has numerous virulence factors that facilitate invasion and thwart host defenses.
- Bacterial counts in the proximal gastrointestinal tract are in the range of 10^4 to 10^5 colony-forming units (CFU)/mL, whereas numbers in the terminal ileum (10^8–10^{10} CFU/mL) approach those in the colon (10^{11}–10^{12} CFU/mL).
- When childhood immunizations have not been given and there is a tetanus-prone wound, tetanus toxoid as well as tetanus immune globulin at a separate site should be administered.
- Approximately 25% of soft tissue infections from *Vibrio vulnificus* are caused by direct exposure of an open wound to warm seawater containing the organism.
- Class IV wounds are characterized by pus or extensive and prolonged contamination and had a historic infection rate of 30% if closed primarily.
- When using alcohol-based skin disinfectants in the operating room, it is imperative that the solution be dry prior to incision to lower the risk of an intraoperative fire.
- Acute infections in soft tissue still present with calor (heat), dolor (pain), rubor (redness), tumor (swelling), and loss of function (functio laesa).
- Intra-abdominal infections identified later in the hospital course (>4 days after injury) are more likely to be caused by hospital-associated rather than community-associated organisms.

INTRODUCTION

Death after traumatic injury has been described in terms of a bimodal distribution. Immediate and acute (<24 hours) deaths usually result from uncontrolled hemorrhage, but infections and multiple organ dysfunction syndrome, which often arise from infection, are responsible for a significant proportion of late deaths. Indeed, infection is responsible for most deaths in patients who survive longer than 48 hours after trauma.[1] Trauma-related infections can be divided into those that result directly from the injury (eg, due to contamination that occurs in conjunction with the traumatic injury) and nosocomial infections that arise in the health care setting in conjunction with treatment of the injury. The pathogens involved can be exogenous or endogenous bacteria, depending on the mechanism of injury and/or the iatrogenic cause.

Most posttraumatic infections are polymicrobial, involving a mixture of aerobic and anaerobic organisms.[2] In one series, 37% to 45% of all trauma patients experienced infectious complications during their initial hospitalization.[3] In that same study, 80% of trauma patients who were in the intensive care unit (ICU) at least 7 days met the criteria for the systemic inflammatory response syndrome (SIRS). All caregivers need to understand the principles of surgical infections in the context of traumatic injury. This chapter will review the following: factors that normally prevent infection, how trauma disrupts or overwhelms normal host defenses, recognition and treatment of the most common infectious complications after traumatic injury, principles of infection prevention, and how prophylaxis and prevention principles can be applied chronologically during the treatment of trauma patients.

PATHOGENESIS OF INFECTION

Humans have evolved mechanisms to avoid infection despite the ubiquitous presence of bacteria in our environment and throughout our bodies. A recent review documented the magnitude and diversity of the human microbiota.[4] Under normal circumstances, there is a balance between our microbiota,

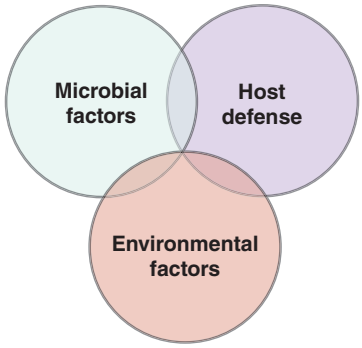


FIGURE 21-1 Under normal circumstances, the determinants of infection, microbial factors, environmental factors, and host defenses interact such that there is no infection. (Adapted with permission from Meakins JL, et al. Host defenses. In: Howard RJ, Simmons RL, eds. *Surgical Infectious Diseases.* 2nd ed. Norwalk, CT: Appleton & Lange; 1988. Copyright © The McGraw-Hill Companies, Inc.)

intact environmental barriers, and host defenses (Fig. 21-1). Invasive surgical procedures or traumatic injury disrupt this balance and significantly increase the probability of developing an infection (Fig. 21-2). Microbes are abundant on the surface of the skin and within the oral cavity and increase in number down the length of the gastrointestinal tract. Bacterial numbers differ at various locations, and the pathogenic species and their respective numbers at different anatomic sites are summarized in Table 21-1. Once inoculation of bacteria into normally sterile sites occurs, infection will ensue if bacteria proliferate faster than the host defense mechanisms eradicate them. Trauma generally results in a much greater disruption of normal barriers than occurs with elective surgery. Trauma may also have concomitant hypoperfusion (shock), devitalized tissue, and retained foreign bodies.

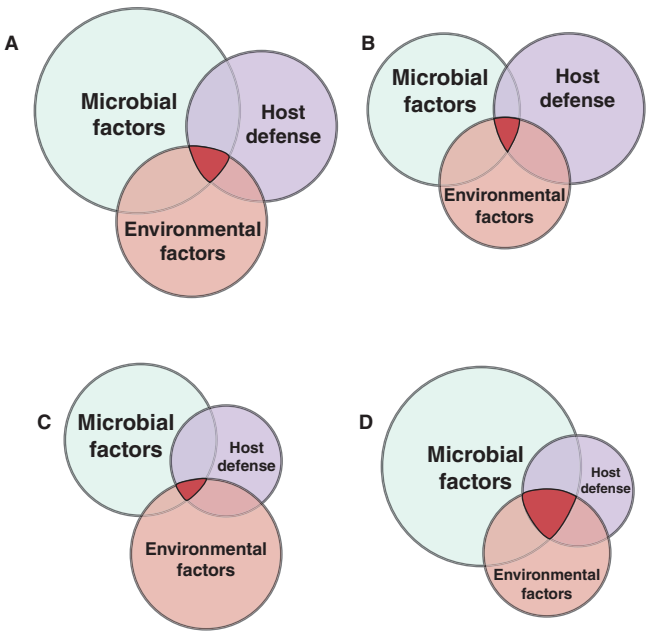


FIGURE 21-2 (**A**) Under circumstances in which there is excessive microbial contamination, (**B**) serious disruption of environmental barrier, (**C**) impaired host defenses, or (**D**) all factors that ensure there will be an increased likelihood of developing infection (shaded intersection of determinants of infection).

TABLE 21-1: Pathogenic Microorganisms Present at Various Anatomic Sites

Region	Microbes	Quantity
Skin (all areas)	*Staphylococcus aureus* and epidermidis *Streptococcus* (nonenterococcal)	10^{2-3}
Skin (infraumbilical)	*Streptococcus faecalis* and *S faecium* *Escherichia coli* *S aureus* and *S epidermidis* *Streptococcus* (nonenterococcal)	10^{2-5}
Oropharynx	*Bacteroides* (nonfragilis) *Fusobacterium* *Haemophilus* *Peptostreptococcus* *S aureus* and *S epidermidis* *Streptococcus* (nonenterococcal)	10^{9-11}
Stomach	*Streptococcus* (nonenterococcal) *Candida*	10^{2-3}
Proximal small intestine	*Bacteroides fragilis* and other *Escherichia coli* and other Enterobacteriaceae	10^{3-7}
Distal ileum and Colon	*Bacteroides fragilis* and other spp. *E coli* and other Enterobacteriaceae *Streptococcus faecalis* and *S faecium*	10^{5-10}

Source: Adapted from Dunn DL. Diagnosis and treatment of infection. In: Norton JA, Barie PS, Bollinger RR, et al, eds. *Surgery: Basic Science and Clinical Evidence*. 2nd ed. New York, NY: Springer-Verlag; 2008:212. With kind permission from Springer + Science Business Media.

Environmental Barriers

Entry of microbes is normally limited by the integrity of environmental barriers, such as intact skin and the mucosa of the respiratory, gastrointestinal, and genitourinary tracts. The importance of intact skin is clearly evident when one considers the potential for microbial infection seen in burn patients or in patients with toxic epidermal necrolysis.[2] Many traumatic injuries are associated with an alteration in the integrity of the skin. Even minor lacerations and abrasions have the potential to disrupt crucial environmental barriers. Interventions made in the process of caring for trauma patients, such as insertion of intravenous or urinary catheters or tube thoracostomy, likewise disrupt the integument and may permit skin bacteria to access sterile sites. The quantitative number of microbes required to produce clinical infection is significantly decreased in the presence of foreign bodies, blood, or devitalized tissue, which are often present with traumatic wounds.[2]

Microbial Factors

The bacteria species responsible for clinical infections in surgical or trauma patients constitute a minority of the skin or gastrointestinal flora. These pathogenic bacterial species generally possess one or more virulence factors that facilitate infection and increase their pathogenicity. In contrast, the vast majority of endogenous and environmental bacteria are relatively nonpathogenic. For example, more than 99% of the colonic microbiota are nonpathogenic anaerobes that never cause clinical infections. Lactobacilli, which do not cause clinical infection, are the most abundant skin bacteria. In contrast, the most common pathogen associated with surgical site infections (SSI), *Staphylococcus aureus*, has numerous virulence factors that facilitate invasion and thwart host defenses. In the abdominal cavity, *Escherichia coli* and *Bacteroides fragilis* are the prototypical organisms associated with intra-abdominal infection, yet they account for only 0.01% and 1% of colonic bacteria, respectively. Under normal circumstances, the overwhelming numbers of nonpathogenic bacteria constitute a robust "defense" against infection, because infection is proportionately less likely if more than 99% of the inoculum is incapable of producing infection. This concept of adherent resident bacteria preventing invasion has been termed *colonization resistance*.[5] This is an important point because the skin and gastrointestinal microbial flora change considerably when trauma patients are hospitalized, both in terms of number and proportion of virulent bacteria and in terms of susceptibility to antibiotics, should an infection develop.

The skin microflora is relatively homogeneous, although bacterial numbers are higher in the axilla and groin areas. The endogenous skin bacteria are predominately gram-positive aerobic *Staphylococcus* and *Streptococcus* species, along with *Corynebacterium* and *Propionibacterium*.[5] As noted earlier, *S aureus* is the most common pathogen present on the skin, but increasingly isolates from trauma patients and those with community-acquired infections are methicillin-resistant *S aureus* (MRSA).[6,7] This fact needs to be taken into account in terms of appropriate empiric or prophylactic antibiotic selection for these patients.

The mouth and nasopharynx harbor large numbers of bacteria, most frequently streptococcal species. Much smaller numbers of bacteria, typically 10^2 to 10^3 colony-forming units (CFU)/mL, are present in the normal stomach because the normal acid pH of the stomach inhibits bacterial growth. Gastric bacterial numbers increase in the absence of gastric acid, as in patients receiving proton pump inhibitors. Bacterial numbers are much higher in the small intestine, and the density of bacteria increases from the duodenum to the terminal ileum. Bacterial counts in the proximal gastrointestinal tract are in the range of 10^4 to 10^5 CFU/mL, whereas numbers in the terminal ileum are close to colonic densities (10^8–10^{10} CFU/mL). Bacterial numbers in the colon are even higher (10^{11}–10^{12} CFU/mL), although most colonic bacteria are nonpathogenic. The presence of such large numbers is associated with very low oxygen tension, and 99.9% of bacteria present are anaerobes. The urogenital, biliary, pancreatic ductal, and distal respiratory tracts are "sterile" in healthy individuals.

Host Defense Mechanisms

Host defense refers to endogenous factors that counteract microbial invasion. In addition to the environmental factors and colonization resistance described earlier, crucial humoral and cellular host defense mechanisms exist that eliminate bacteria within sterile spaces. Initially, several primitive and relatively nonspecific host defenses, including proteins such as lactoferrin, fibrinogen, and complement, begin to act against invading microbes. Lactoferrin sequesters the critical microbial growth factor iron, thereby limiting microbial growth. Fibrinogen within the inflammatory fluid has the ability to trap large numbers of microbes during polymerization into fibrin.[5] Complement is activated on contact with bacteria and viruses, from tissue damage, or when IgG/IgM antibodies recognize microbial agents. Activation of complement releases C3a and C5a, which are potent chemotaxins that recruit neutrophils and macrophages. These components enhance endothelial adhesiveness and increase vascular permeability. Complement activation can directly destroy microbial agents via formation of a membrane attack complex (composed of complement proteins C5–C9) and enhance microbial phagocytosis by way of C1q and C3bi subunits. In vitro studies have shown that 50% to 70% of a moderate inoculum is eliminated *prior to* the influx of phagocytic host cells.

Many different tissues also contain resident innate immune cells. These include macrophages, dendritic cells, Kupffer cells, glial cells, mesangial cells, and alveolar macrophages.[8] These innate immune cells express a wide variety of pathogen-associated molecular pattern (PAMP) receptors on their surface.[9-11] The best known examples of PAMPs are the toll-like receptors (TLRs), of which there are now many well-described receptor molecules.[11] TLRs bind to ligands on bacteria (or damaged host tissue), and TLR binding results in activation of these cells. Activated macrophages secrete an array of substances in response, leading to amplification and regulation of the acute proinflammatory response (Fig. 21-3). Sequential release of protein cytokines, including tumor necrosis factor-α (TNF-α), interleukin (IL)-1, IL-6, IL-8, and interferon-γ (INF-γ), follows. These mediators produce the signs and symptoms that are associated with infection (eg, fever, tachycardia, tachypnea, leukocytosis). IL-8 is a very potent chemoattractant for neutrophils, the innate immune cells primarily responsible for ongoing microbial phagocytosis and intracellular microbial killing. Unfortunately, the same process that recruits neutrophils and stimulates phagocytosis and oxidative killing may also contribute to damage of host tissues. Simultaneous with this proinflammatory response, there is also production of anti-inflammatory mediators, such as IL-10 and transforming growth factor-β (TGFβ).[12] Some of these mediators may contribute to the immune "hyporesponsiveness of trauma" over the ensuing days (Table 21-2).[13]

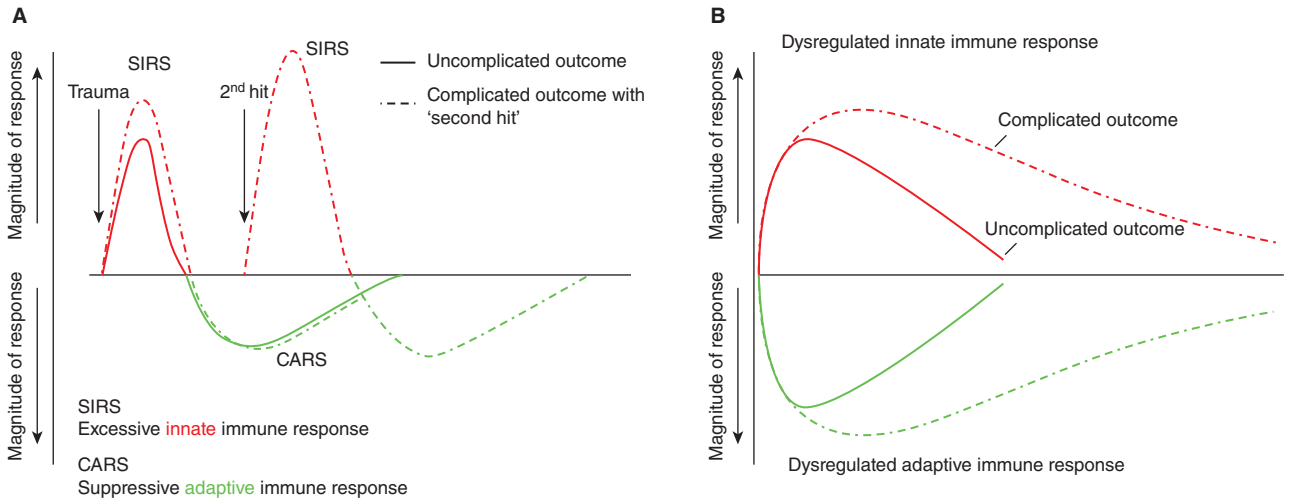


FIGURE 21-3 Schematic depiction of a refined paradigm for pro- and anti-inflammatory responses after trauma or inflammation based on empirically observed alterations in gene expression after injury. (**A**) The previous paradigm had invoked an initial proinflammatory response followed by an anti-inflammatory response. (**B**) The updated paradigm demonstrates that both pro- and anti-inflammatory responses take place simultaneously, beginning at the time of the initial injury or insult. Patients who go on to develop complications and multiple organ dysfunction syndrome (MODS) have a different temporal response, with prolongation of both responses, but they do not have qualitative or fundamental differences in the nature of the inflammatory responses. CARS, compensatory anti-inflammatory response syndrome; SIRS, systemic inflammatory response syndrome. (Reproduced, with permission, from Xiao W, Mindrinos MN, Seok J, et al. Genomic storm. *J Exp Med.* 2011;208(13):2581-2590.)

TABLE 21-2: Immunologic Defects Associated with Traumatic Injuries

Type of cell and action	Change from normal
T and B lymphocytes	
CD4+/CD8+ ratio	Decreased
TGF-β production	Increased
Th17 phenotype	Increased
Immunosuppressive T_{reg} phenotype	Increased
Monocytes/macrophages	
Immunocompetent activation	Decreased
HLA-DR expression	Decreased
Antigen-presenting capability	Decreased
PGE_2 production	Increased
IL-12 production	Decreased
IFN-γ release	Decreased
Common characteristics of T cells and monocytes	
Proportion of Th2 phenotype	Increased
Trauma-induced apoptosis	Increased
Polymorphonuclear neutrophils (PMN)	
Chemotaxis	Decreased
Phagocytic capacity	Decreased
Release of elastase	Increased
O_2 radical release	Increased
β-Integrin expression	Decreased
Leukotriene B_4 production	Decreased
Apoptosis	Delayed

IFN, interferon; IL, interleukin; TGF, transforming growth factor.

Source: Adapted with permission from Tschoeke SK, Ertel W. Immunoparalysis after multiple trauma. *Injury.* 2007;38(12):1346-1357. © Elsevier.

Specific anatomic locations have additional unique factors that defend against infection.[14] For example, the peritoneal cavity has lymphatic channels on the undersurface of the diaphragm that facilitate removal of bacteria.[15] The subdiaphragmatic surface is a lower-pressure area due to the effect of respiratory excursion, and this serves to move free fluid within the peritoneal cavity to this location. Movement of the diaphragm "pumps" this fluid into the thoracic duct, and from there, it gains rapid access to the systemic circulation. Experimental studies show that labeled bacteria inoculated into the peritoneal cavity appear in the thoracic duct within 6 minutes and in the bloodstream within 12 minutes.[14] The respiratory tract has unique host defenses that help to ensure the sterility of the lung parenchyma, as well. Goblet cells within the respiratory mucosa secrete mucin that helps to trap bacteria. Ciliated respiratory epithelial cells move the mucus centrally, where it, and the bacteria trapped within it, can be expectorated by coughing. The presence of endotracheal tubes, smoking, inhaled toxins, and some anesthetic agents interfere with mucociliary clearance mechanisms, and this may predispose to pneumonia. Bacteria or other microbes that gain access to the alveoli are normally phagocytized by alveolar macrophages, although the macrophage activation that may accompany this process has been proposed as one possible pathogenic mechanism for acute lung injury or adult respiratory distress syndrome (ARDS).[16-18]

MICROBIOLOGY

To a very large extent, the microbial agents responsible for infections or infectious complications after trauma are the same agents that cause most other surgical or ICU-associated

TABLE 21-3: Intensive Care Unit Pathogens Isolated from Patients with Ventilator-Associated Pneumonia

Organism/Class	% Total
Lactose-fermenting gram-negative bacillus	
Escherichia coli	8.1%
Klebsiella spp.	11.1%
Enterobacter spp.	7.6%
Morganella spp.	0.5%
Citrobacter spp.	1.0%
Serratia spp.	1.5%
Lactose-non-fermenting gram-negative bacillus	
Stenotrophomonas spp.	2.0%
Acinetobacter spp.	8.6%
Pseudomonas spp.	13.6%
Staphylococcus aureus	31.8% total
Methicillin-sensitive	22.2%
Methicillin-resistant	9.6%
Community pathogens	
Streptococcus pneumoniae	2.0%
Haemophilus spp.	3.0%
Other pathogens	
Polymicrobial species	3.0%
Fungus	1.5%

Source: Adapted from Eachempati SR, Hydo LJ, Shou J, Barie PS. The pathogen of ventilator-associated pneumonia does not influence the mortality rate of surgical intensive care unit patients treated with a rotational antibiotic system. *Surg Infect (Larchmt)*. 2010;11(1):13-20.

infections. Table 21-1 shows the most common infectious agents that cause trauma-associated infections at various anatomic sites. Generally, *Staphylococcus* spp. and *Streptococcus* spp. are the most common pathogens responsible for infections in which the traumatic injury or operative intervention needed to treat the injury did not transgress a mucosal surface. For traumatic injuries that involve the aerodigestive tract, the most common isolates are *E coli* (43.4%), *S aureus* (18.9%), *Klebsiella pneumoniae* (14.4%), and *Enterococcus faecalis* (5.6%).[1] Hospitalized trauma patients develop nosocomial bacterial infections from the usual ICU-associated pathogens (Table 21-3). A few infectious agents that can be associated with trauma are seldom encountered in other settings including rabies virus, *Clostridium tetani*, and *Vibrio*.

Rabies

Rabies is a rare, potentially fatal, clinical disease caused by the rabies virus. Rabies is an RNA virus present in the saliva of mammals, and transmission to humans generally occurs following a bite from a rabid animal. Prior to the development of a vaccine by Louis Pasteur, bites from a rabid animal were uniformly fatal. In North America, raccoons, skunks, bats, foxes, coyotes, and bobcats are the primary reservoirs. Most patients with rabies have no documented exposure to a rabid animal, and the majority of these are associated with bat bites. Many victims underestimate the importance of a bat bite, and a substantial portion do not even recall being bitten. Bats (Carnivora and Chiroptera) represent the ultimate zoonotic reservoir for the virus. The rabies virus is highly labile and can be inactivated readily by ultraviolet radiation, heat, desiccation, and other environmental factors.

The word *rabies* derives from the Latin *rabere*, meaning "to rage," and refers to the clinical manifestations of the disease that include hyperactivity, disorientation, hallucinations, and bizarre behavior. The rabies virus is neurotropic and causes an acute encephalitis. Other hallmarks of the disease include an intense fear of suffocation (eg, hydrophobia and aerophobia) secondary to intense laryngeal and pharyngeal spasm. Once the patient begins manifesting symptoms, death is nearly certain. With increased vaccination and postexposure prophylaxis (PEP) over the past 50 years, the clinical disease is becoming increasingly uncommon. According to the Centers for Disease Control and Prevention (CDC), 49 cases of human rabies were reported in the United States between 1995 and 2011, with only 3 survivors. That said, it is important for the practitioner of emergency medicine/surgery to be knowledgeable about rabies since animal bites are encountered frequently in clinical practice.

Humans are not routinely vaccinated against rabies, while domestic animals receive routine rabies vaccinations. If a human is bitten by a rabid animal, rabies can be *prevented* by PEP before the virus enters the central nervous system during the incubation period. The diagnosis of rabies can be made rapidly by identification of rabies virus in the brain of a potentially infected animal. This procedure can be performed most expeditiously by euthanizing the suspected animal. If the rabies test is negative, then no postexposure vaccination or prophylaxis is needed. The incidence of positive rabies tests ranges from as high as 6% to 10% in wild animals down to levels of approximately 1% in domestic pets. An acceptable alternative approach, if the suspected source is a domestic pet (eg, dog, cat, ferret), is for the offending animal to be quarantined and observed for 10 days. If the animal exhibits signs of rabies, the exposed person should begin PEP immediately and the animal should be euthanized and its brain tissue tested for rabies. If the animal is confirmed to have rabies, PEP must be completed; however, if the test results are negative, PEP can cease.

Immediate measures that should be taken to decrease the risk of rabies transmission include thorough washing of bite and scratch wounds with soap and water, followed by application of povidone–iodine or alcohol. Human rabies immune globulin (HRIG) and rabies vaccine should be given in all cases except in persons who have been immunized previously.[19] Immune globulin should never be delivered in the same syringe as the vaccine, as this will cause precipitation. The Advisory Committee on Immunization Practices (ACIP) of the CDC and the American Academy of Pediatrics recommend a single dose (20 IU/kg) of HRIG be given to provide protection for the first 2 weeks until the vaccine elicits an antibody response. Detailed and up-to-date information for rabies exposure is available on the CDC's website

(http://www.cdc.gov/rabies/index.html), and this site should be consulted for the latest information. The ACIP recommends a regimen of human diploid cell vaccine (Imovax) for PEP on days 0, 3, 7, 14, and 28, along with a single dose of HRIG on day 0. Once initiated, rabies prophylaxis should not be interrupted or discontinued because of local or mild systemic reactions to the vaccine.

Tetanus

Tetanus is a rare, life-threatening condition that is caused by toxins produced by *Clostridium tetani*, a spore-forming, gram-positive bacillus.[20] Clostridial spores can survive indefinitely, and they are ubiquitous in soil and feces. Under anaerobic conditions, the spores can germinate into mature bacilli that elaborate the neurotoxins tetanospasmin and tetanolysin. Tetanospasmin produces most prominent clinical symptoms by interfering with motor neuron release of the inhibitory neurotransmitters γ-aminobutyric acid (GABA) and glycine. The resulting loss of inhibition produces muscle spasm (usually spasm of the masseter muscle) and severe autonomic overactivity manifested by high fever, tachycardia, and hypertension. Historically, tetanus was highly fatal, but intensive medical therapy with neuromuscular blockade, mechanical ventilation, and ICU monitoring has lowered the case fatality rate to 11% to 28%. According to the CDC, there were a total of 233 tetanus cases reported in the United States between 2001 and 2008, with 71.7% arising from acute trauma. Clinicians and trauma surgeons must remain alert for the potential of clostridial contamination and provide appropriate, timely tetanus prophylaxis.[20]

The diagnosis of tetanus is made on clinical grounds alone, as there are no laboratory tests that can diagnose the condition or rule it out. Tetanus immunization is accomplished as a component of standard early childhood immunizations (diphtheria–pertussis–tetanus), with administration of tetanus toxoid (TT) every 5 to 10 years to maintain immune memory (see http://www.cdc.gov/tetanus/vaccination.html). No deaths have been reported in individuals who have been fully immunized. The CDC recommendations for tetanus prophylaxis depend on the wound characteristics and the prior immunization status of the patient. A wound with extensive contamination, one that is poorly vascularized, or one with extensive soft tissue trauma is considered to be a "tetanus-prone wound." A tetanus booster should be administered to patients who have received primary immunization but who have not received TT during the past 10 years, or the past 5 years for tetanus-prone wounds.[19] In patients who have never undergone primary immunization, human tetanus immune globulin (TIG) should be administered along with TT at a different site. Antitetanus antibody binds to exotoxins and neutralizes their toxicity. High-risk groups such as the elderly, individuals with HIV, and intravenous drug users who had received primary vaccination may not have tetanus antibodies, and more liberal use of TIG should be considered in these groups.[19] If TIG is not available, intravenous immune globulin (IVIG) may be substituted.

Infections Associated with Marine Trauma

Vibrio vulnificus is a gram-negative rod present in seawater that can result in atypical, necrotizing soft tissue infections when traumatic injuries occur in the ocean.[21-23] *V vulnificus* is common in warm seawater and thrives in water temperatures greater than 68°F (20°C). The organism is not associated with pollution or fecal waste. Approximately 25% of *V vulnificus* infections are caused by direct exposure of an open wound to warm seawater containing the organism. Exposure typically occurs when the patient is participating in water activities such as boating, fishing, or swimming. Infections are occasionally attributed to contact with raw seafood or marine wildlife. The risk of developing *Vibrio* infection is much higher in immunocompromised patients or patients with preexisting hepatic disease or diabetes mellitus.[22] Established infection with *V vulnificus* can be highly invasive, with mortality rates of 30% to 40% and a mortality of greater than 50% in immunocompromised patients. A published report from 2009 documented a 37% mortality rate even after implementation of a specific treatment guideline for necrotizing *Vibrio* infections.[21]

Patients with wound infections caused by *V vulnificus* develop painful cellulitis that progresses rapidly.[22-24] Physical examination will often reveal marked swelling and painful, hemorrhagic bullae surrounding traumatic wounds. In some cases, there can be rapid progression and associated systemic symptoms. Marked local tissue swelling with hemorrhagic bullae are characteristic. Systemic symptoms include fever and chills, and bacteria are present in the bloodstream in more than 50% of patients. Hypotension or septic shock may be an early symptom, and alterations in mental status occur in approximately one-third of patients. Table 21-4 summarizes clinical symptoms present in patients with *Vibrio* infection.[23]

TABLE 21-4: Clinical Characteristics Associated with *Vibrio vulnificus* Wound Infections

Characteristic	% of Patients
Cellulitis at wound site	88
Skin bullae	88
Fever (>37.8°C)	65
Chills	29
Ecchymosis	18
Obtundation, disorientation, or lethargy	18
Hypotension (<90 mm Hg)	12
Vomiting	6
Diarrhea	6

Source: Adapted from Klontz KC, Lieb S, Schreiber M, Janowski HT, Baldy LM, Gunn RA. Syndromes of *Vibrio vulnificus* infections. Clinical and epidemiologic features in Florida cases, 1981-1987. *Ann Intern Med.* 1988;109(4):318-323.

It is important for trauma surgeons to be aware of the potential for *Vibrio* infections in the appropriate clinical setting, because antibiotic treatment is distinctly different from the agents typically employed for trauma patients. Aggressive surgical debridement, incision and drainage of purulent collections, and even amputation may be crucial adjuncts for management of these often severe soft tissue infections.[22] Recent experience in 30 patients with documented *Vibrio* infection found that fasciotomy was needed in all patients, and 17% required amputation.[21] Recommended antibiotics include doxycycline (100 mg intravenously or orally twice a day), ceftazidime (2 g every 8 hours), cefotaxime (2 g every 8 hours), or ciprofloxacin (750 mg orally twice a day or 400 mg intravenously every 12 hours).[22,25]

Traumatic injuries that occur in freshwater conditions may develop infections from *Aeromonas hydrophila*.[24] *A hydrophila* is a gram-negative anaerobic rod that is a common pathogen of fish and amphibians. Cutaneous inoculation of the organism can result in cellulitis, abscesses, and, occasionally, necrotizing soft tissue infections. Like the situation with *Vibrio* infections, patients with hepatic disease and immunocompromise have a greater risk of developing generalized disease. *A hydrophila* can be recovered from the bloodstream in a significant proportion of patients, and this fact, along with a history of injury in fresh water, will aid in alerting clinicians to the correct diagnosis. Antibiotic agents active against *A hydrophila* include third-generation cephalosporins, fluoroquinolones, doxycycline, or trimethoprim-sulfamethoxazole.[24]

Ebola Virus Disease

On September 30, 2014, in the midst of the worst Ebola virus disease (EVD) outbreak on record, the CDC confirmed the first documented case of EVD in the United States in Dallas, Texas.[26] Within 2 weeks, a nurse who cared for that initial patient was also diagnosed with the disease. While EVD is not a surgical disease per se, knowledge and skills in resuscitation, critical care, and disaster management may be invaluable in the treatment of EVD. Therefore, all surgeons should be familiar with the fundamentals of EVD management.

TRANSMISSION

Ebola virus is a zoonosis belonging to the *Filoviridae* family. Five species have been identified as follows: *Zaire, Sudan, Ivory Coast, Bundibugyo*, and *Reston*.[27] Ebola is a single-stranded RNA virus that sporadically spreads to humans from a presumptive animal or bat reservoir in the wild. Human spread only occurs through *direct contact with infected bodily fluid*, and the most infectious sources are blood, feces, and vomit.[28] Ebola virus can remain viable on surfaces for 1 to 6 days, although the risk of transmission from a surface contact is considered very low.[29,30] Humans are only contagious while they are symptomatic. Blood from patients with untreated late EVD harbor 10^9 virus particles/mL, making it highly infectious. This value can be compared to the much lower viral loads in patients with untreated HIV (10^5/mL blood) or hepatitis C (5–20 × 10^6/mL blood), both of which are recognized as being highly transmissible.[31] EVD is not an airborne pathogen, but since it can be transmitted via large droplets, health care providers in close contact with symptomatic patients should take necessary droplet precautions (see Prevention).[28]

PATHOPHYSIOLOGY

Ebola virus enters the body through breaks in the skin, mucus membranes, or ingestion and then infiltrates cells, especially lymph tissues, liver, and spleen. The virus continues to replicate until cells become necrotic and lyse, spilling more viral particles into the circulation. Ebola virus elicits a profound proinflammatory cytokine and chemokine response, producing a vigorous SIRS reaction.[32,33] Significant endothelial injury ensues, with loss of vascular tone and increased vascular permeability leading to hypotension and shock.[34,35] At the end stages of disease, the virus is found in all body fluids and skin, making handling of bodies of EVD victims extremely hazardous. In contrast to prevailing biases, bleeding does not occur in all patients and only manifests late in the disease process as bleeding from skin, gums, and the gastrointestinal tract.[36] Mortality rate remains high at 50% to 90% overall, with a poorer prognosis seen in those with older age, diarrhea, hemorrhagic conjunctivitis, shortness of breath, confusion/coma, and hemorrhage.[37]

SYMPTOMS

Patients in early stages of the disease present with the acute onset of flu-like symptoms. Fever is the most common symptom (present in 87%), with fatigue (76%), abdominal pain (44%), and nausea and vomiting (65%) also frequently seen.[37,38] Late symptoms are representative of a fulminant infection and SIRS with symptoms of severe vomiting and diarrhea, both of which may be bloody. Other late signs include a coagulopathy identified by diffuse oozing from mucosal surfaces and sites of intravenous lines. Death is often the result of recalcitrant multiorgan system failure.[26]

TREATMENT

Currently, there is no vaccine against or pharmacologic treatment for EVD, and the mainstay of therapy is supportive care. Many patients will have severe diarrhea, losing up to 15 L/d, and require aggressive resuscitation efforts focused on replenishing circulatory volume. Significant electrolyte abnormalities are common, and correction of these must be done in order to prevent cardiac dysrhythmias. In the early stages of the disease, patients can often tolerate oral fluids, antiemetics, and antidiarrheals. Patients with protracted vomiting or in those later stages of the disease should have intravenous fluids administered. Blood transfusions may be necessary, especially for those with hemorrhagic symptoms, and should follow current recommendations for transfusion

triggers. Respiratory support in the form of mechanical ventilation may be necessary, but caution should be exercised with noninvasive support, as it may lead to aerosolization.[39] Some novel antivirals known to have in vitro or animal anti-Ebola activity were used during the 2014 outbreak, but data regarding their effectiveness are inconclusive at this time. Experimental therapies used during the 2014 EVD outbreak included plasma from convalescent or immune patients and experimental monoclonal antibodies, but it is not clear that either method provided a survival benefit.[40,41] Several vaccines are currently undergoing accelerated human trials, but none as yet are commercially available.[42]

PREVENTION

The 2014 EVD outbreak clearly demonstrated how globalization has enhanced the capacity for a previously isolated tropical disease to rapidly spread. Prevention relies on maintaining a level of preparedness of providers and facilities that can receive patients with EVD, provide quality critical care, protect the providers, and assuage the fears of the community. Preventing EVD transmission is divided into mitigating transmission of the primary disease and screening for early identification of the disease as part of planning for a disaster.

With a new outbreak in the Congo in 2018, the recommendations issued by the CDC for use in health care facilities are still relevant.[43] Strict adherence to protocols for use of personal protective equipment (PPE) and adoption of practices to limit exposure to body fluids constitute the mainstays of primary prevention, as well as environmental controls.[44] Practiced use of full PPE, as described by the CDC (http://www.cdc.gov/vhf/ebola/healthcare-us/ppe/guidance.html), that is donned and doffed in the presence of a trained observer is paramount when treating a patient with EVD.[45] All practitioners who will come into contact with a patient with EVD should be trained and certified in their ability to properly wear full PPE prior to their first encounter with the patient. The body should be covered from head to toe with single-use hood, gown (or coveralls), double gloves, impermeable boot covers, and a respirator or N95 mask. Ideally, extended cuff gloves, gowns with thumb hooks, and/or tape securing the gowns and gloves should be used. During contact with the patient, the practitioner should limit contact with surfaces with body fluid, keep their hands away from their face, and employ frequent alcohol-based scrubs on their gloved hands. There should be no rush or shortcuts taken while placing the PPE, so time consideration should be given if plans for procedures or intubation are required. Total time spent in the care of the patient while wearing PPE should be monitored because providers can become fatigued and dehydrated. Elective surgical procedures should be postponed in a patient with EVD, and nonoperative alternatives should be strongly considered for urgent conditions (eg, appendicitis, perforated ulcer). A risk-benefit analysis considering the risk of exposure to the operative team must be considered prior to operating for an emergent condition bearing in mind that, in late EVD disease, the patient is unlikely to survive.

DISASTER MANAGEMENT

Health care preparedness for a real or potential Ebola outbreak mandates the presence of an effective screening procedure that will capture any potential carrier of EVD. A robust program that contains multiple contingency plans and has been rehearsed by all the team members is essential, as disaster leaders should be prepared to operate without significant assistance for at least 72 hours. A simple screening method that can be used at any first encounter with a patient (including clerical areas) should focus on the following: (1) recent travel (last 21 days) to an affected area; and/or (2) recent (21 days) direct contact with a patient infected with Ebola.

Positive responses to these simple questions lead to an escalation of care with an immediate separation and isolation of the patient. After proper isolation, a more focused history identifying exposure risk can be performed. Once a diagnosis of EVD is confirmed, the CDC recommends that plans should be in place to ensure immediate isolation is available, which includes a private room with a door, a private bathroom, and separate areas for donning and doffing PPE. Other pragmatic plans for handling of lab specimens, environmental waste management, and restriction of nonessential personnel must be in place.[46] The institutional management of threatened or actual Ebola outbreak requires trained, professional emergency managers and disaster management experts to be involved in incident command. Incident control should designate a team of site managers who have a constant presence to oversee implementation of safety precautions, monitor adequacy of necessary supplies, and evaluate care in isolation areas.

The high infectious risk and mortality rate of EVD evoke strong emotions of fear, both in health care workers and the general public. The incident command should remain the voice of calm and reason during the disaster and encourage constant communication between the front-line providers, the incident commander, and the community.

PREVENTION OF INFECTIONS

General Principles

As in all other aspects of surgical care, it is preferable to try to prevent infections wherever possible. A number of interventions and practices have been demonstrated to be highly effective in preventing infections after elective operations, and many of those techniques have specific application in the care of injured patients. In this section, current evidence-based interventions that are applicable to trauma patients to prevent infection will be reviewed.

Prophylactic Antibiotics

Prophylactic antibiotics are intended to *prevent* development of infection. The concept of prophylaxis presupposes that infection is *not* present. The decision to use prophylactic antibiotics and the choice of agents are based on the risk of developing SSI. There are very good data regarding SSI

TABLE 21-5: Classification of Surgical Wounds

Wound class	Characteristics	Examples	SSI rate (%)
Clean (I)	Uninfected, no inflammation, no mucosal surface transected	Mastectomy Thyroidectomy Vascular bypass CABG	<2%
Clean-contaminated (II)	Uninfected, no inflammation, no mucosal surface transected	Colectomy Cholecystectomy Laryngectomy Urologic procedures Pulmonary lobectomy	5%–15%
Contaminated (III)	Open accidental wounds, break in sterile conditions, spillage, stomas	Appendicitis Diverticulitis Small bowel GSW	15%–30%
Dirty-infected (IV)[a]	Infection, perforation, devitalized tissue	Incise and drain abscess Peritonitis Enteric fistulas Remove infected implant	>30%

[a]Dirty wounds = infection antibiotics indicated as therapy.

CABG, coronary artery bypass graft; GSW, gunshot wound.

Source: Adapted from Nichols RL. Surgical infections: prevention and treatment—1965 to 1995. *Am J Surg*. 1996;172(1):68-74.

rates for elective surgery, and the incidence of SSI by wound class for elective operations is shown in Table 21-5.[47] Traditionally, class I, or clean wounds, are those that do not violate the respiratory, alimentary, or genitourinary tracts. The wound infection rate is approximately 2%. Class II, or clean-contaminated wounds, refers to elective operations on potentially contaminated organs, such as the gastrointestinal tract, biliary tract, genitourinary tract, and respiratory tree (the procedure will violate a mucosal surface, which can never be completely sterile). The usual incidence of infection for these types of wounds is 5% to 10%. Contaminated wounds (class III) differ from class II wounds by the degree of spillage, with an incidence of infection of 15% to 30%. Finally, class IV, or dirty-infected wounds, are characterized by frank pus or extensive and prolonged contamination. In one series of patients from the 1970s, these wounds were characterized by an infection rate of greater than 30% if primary closure is attempted.[48] A recent single-center, randomized controlled trial of primary versus delayed closure of wounds in complicated appendicitis reported nonsignificantly lower SSI rates in the primary closure group, with rates ranging from 7% to 10%.[49] Overall, it appears that hospitalized SSI rates were considerably lower in 2015 compared to 2011 in a National Healthcare Safety Network (NHSN) point prevalence study in 199 hospitals across 10 states, with the number of *Clostridium difficile* infections surpassing SSI for the first time.[50]

In trauma surgery, the majority of wounds encountered will be class III or IV, and the luxury of a preinoculation dose of antibiotics, as recommended by the Surgical Care Improvement Project, is usually unavailable.[7] These recommendations were reinforced in 2017 in updated guidelines for prevention of SSI from the CDC.[51] A single-center retrospective cohort study of laparotomy following mostly penetrating trauma demonstrated a very high rate of SSI (39%) and increased risk of SSI if intraoperative temperatures dipped below 35°C.[52] Interestingly, failure to administer preoperative antimicrobials was not an independent predictor of SSI, whereas hypothermia was. With this in mind, it is prudent to administer a single dose of an agent with activity against community-acquired aerobic and anaerobic pathogens as soon as possible for all patients requiring operation in the thorax or abdomen. Evidence-based guidelines for antibiotic prophylaxis of other surgical interventions or different anatomic sites are summarized in Table 21-6.

The issue of postoperative continuation of prophylactic antibiotics in penetrating abdominal trauma has been investigated extensively. The Eastern Association for the Surgery of Trauma (EAST) has published guidelines derived from an evidence-based review.[53] A single preoperative dose of a prophylactic antibiotic with broad-spectrum aerobic and anaerobic coverage is recommended for trauma patients sustaining penetrating abdominal wounds. Absence of an injury to a hollow viscus requires no further administration. Even in the presence of a hollow viscus injury, class I evidence supports limiting prophylactic antibiotics to only 24 hours.[54] Timely discontinuation of prophylactic antibiotics is important because the practice of prolonged administration has been linked to increased rates of subsequent nosocomial infections with resistant organisms.[55] To maintain adequate tissue and serum levels of antibiotics in the face of ongoing hemorrhage and vasoconstriction, the administered dose may be increased two- or threefold and repeated after every 10 units of transfusion of blood products, although there is no evidence to support this practice.

TABLE 21-6: Evidence-Based Recommendations for Antibiotic Prophylaxis for Specific Interventions or Injuries

Intervention/Injury	Agent/Route/Duration	Reference(s)
Chest tube	May use first-generation cephalosporin prior to chest tube insertion, ≤24-h duration, but not enough evidence to support this recommendation (EAST guidelines)	118
Mandibular fractures	Perioperative antibiotics only, 24-h duration at most	223
Open globe injuries	Ceftazidime and vancomycin IV for 48 h to reduce risk of endophthalmitis	224
Basilar skull fractures	Evidence does not support prophylactic antibiotics to decrease risk of meningitis in presence of rhinorrhea or otorrhea	225
Penetrating brain injury	First-generation cephalosporin for 5 days after injury	226
Intraventricular pressure monitor	Antimicrobial based on local antibiogram administered prior to insertion only; use antimicrobial-impregnated catheters	227
Burns	Prophylactic systemic parenteral antibiotics are strongly discouraged	228
Hand/tendon repairs	Single dose of first-generation cephalosporin may be beneficial; no evidence to support >24-h duration	229, 230
Closed fractures	Perioperative first-generation cephalosporin at time of ORIF; no longer than 24 h	231
Open fractures	First-generation cephalosporin for 48 or 24 h after wound closed Gram negative antimicrobial coverage for grade III fractures Add anaerobic coverage for soil, fecal, or standing water contamination No benefit to routine use of antibiotic beads	174, 232, 233
Chest trauma	Cephalosporin	116
Penetrating abdominal trauma	Agent(s) with activity against aerobic and anaerobic bacteria	54, 141
Combat injuries	Point of injury/delayed evacuation: moxifloxacin 400 mg PO × 1 dose; ertapenem 1 g IV or IM if penetrating abdominal injury, shock, or unable to tolerate PO medications Extremity injury: cefazolin 2 g IV q 6–8 h for 24–72 h Abdominal injury: cefazolin 2 g IV q 6–8 h plus metronidazole 500 mg IV q 8–12 h for 24 h after definitive washout	234

EAST, Eastern Association for the Surgery of Trauma; IM, intramuscular; IV, intravenous; ORIF, open reduction internal fixation; PO, oral; q, every.

Surgical Scrub

For many years, povidone–iodine scrub was the standard disinfectant used for preparation of surgical sites and preoperative scrubbing by the surgical team. This dominance was challenged by a randomized controlled trial performed in elective surgery showing significantly lower SSI rates with the use of chlorhexidine–alcohol compared with iodine (9.5% vs 16.1%).[56] On the contrary, a prospective cohort analysis of over 7500 patients across the state of Washington demonstrated no clear benefit to any one skin preparation agent,[57] and a randomized controlled trial of povidone–iodine versus chlorhexidine gluconate in clean-contaminated upper gastrointestinal and hepatobiliary operations detected no difference in SSI rates between the two arms.[58] There are no published trials of the use of skin preparations in trauma or other emergency surgery, however, but the fact that chlorhexidine–alcohol begins bacterial killing immediately on contact and does not require drying for antimicrobial effectiveness makes it potentially attractive for use in these cases. One caveat regarding use of alcohol-based disinfectants is that it is imperative that the solutions dry prior to incision to lower risk of intraoperative fires.

Double Gloving

Glove perforation is an underappreciated phenomenon that may adversely impact the sterility of an operative procedure. Microperforation rates as high as 16% have been reported.[59] When two pairs of gloves are used, inner glove perforation rate is substantially lower. In addition to patient outcome, the surgeon must also consider personal safety (see Occupational Exposure section).

Temperature Control

Hypothermia has been shown to be a strong independent predictor of poor outcome.[60] Hypothermia slows enzymatic activity needed for blood clotting and can induce an acquired coagulopathy. Hypothermia also modulates the inflammatory response, resulting in immune suppression associated with higher rates of all infectious complications, including SSI.

In elective colorectal surgery, a prospective, randomized study compared a group in whom intraoperative normothermia (36.6°C) was maintained to a control group with mild hypothermia (34.7°C). The normothermic group had a significantly lower rate of SSI (6% vs 19%) and a 20% shorter hospital stay.[61] The precise mechanism for the beneficial effects of normothermia remains unclear but may relate to tissue perfusion and improved host defense. Therefore, every reasonable effort should be made to maintain normothermia in all surgical patients.

Supplemental Oxygen

Perioperative supplemental oxygen is now strongly recommended for the reduction of SSI risk. In another prospective randomized study in patients undergoing elective colorectal operations, the authors observed a significantly lower SSI rate when 80% versus 30% inspired oxygen was delivered during the operation.[62] This was recently replicated in a study of emergency laparotomy for perforated peptic ulcer, demonstrating a 50% decrease in SSI with 80% fraction of inspired oxygen delivered during and for 6 hours following surgery.[63]

Suture Material

Sutures decrease the inoculum of bacteria needed to establish infection and can serve as a foreign body within potentially infected wounds, so there is a sound physiologic basis for a potential benefit in using antimicrobial impregnated suture material. There have now been several randomized controlled trials demonstrating a decreased incidence of SSI when antibiotic-coated sutures were employed for facial closure, and a 30% decrease in SSI was seen in a large meta-analysis.[64] Subsequent trials, however, have failed to demonstrate benefit.[65]

Blood Transfusion

Blood transfusion can be lifesaving for an exsanguinating patient, but numerous authors have reported worse infectious complications with increased blood utilization both in the immediate resuscitation[66-69] and when used in a delayed fashion.[70-72] Transfusion results in a multitude of immunosuppressive effects, including the following: (1) decreased CD3+, CD4+, and CD8+ cells; (2) overall reduced T-cell proliferation to mitogenic stimuli; (3) decreased natural killer cell activity; (4) defective antigen presentation; and (5) impaired cell-mediated immunity.[73] The increased risk of infection associated with blood transfusion appears to be dose dependent,[72,74] and logistic regression analyses report that the risk of infection in patients sustaining burn injury increases 13% per unit transfused.[75] Taylor et al[76] reported that for each unit of packed red blood cells (PRBCs) transfused, the odds of developing a nosocomial infection were increased by a factor of 1.5 in critically ill surgical patients. The age of the transfused blood is an additional risk factor for infectious complications.[77,78] As blood ages in the blood bank, it undergoes predictable changes that affect its ability to deliver oxygen. This "storage lesion" includes the following: (1) an increased affinity of hemoglobin for oxygen and reduced oxygen release to tissues; (2) depletion of 2,3-diphosphoglyerate (2,3-DPG) with resultant inadequacy of oxygen transport by red blood cells (RBCs); (3) reduction in deformability, altered adhesiveness, and aggregability; and (4) accumulation of bioactive compounds with proinflammatory effects. In trauma patients, Zallen et al[79] estimated that each transfused unit of RBCs older than 14 days increased the risk of organ failure by 13%.

To minimize infection risk, blood transfusion in nonbleeding patients should be avoided when at all possible. A large multicenter randomized study reported the safety of a restrictive transfusion strategy (trigger of 7.0 g/dL hemoglobin) compared to a liberal strategy (trigger of 10.0 g/dL). In fact, mortality rates in patients who were younger (age <55) and less sick (Acute Physiology and Chronic Health Evaluation [APACHE] score <20) were more than double with liberal transfusion.[80] When the subgroup of trauma patients (n = 203) was reviewed in a secondary analysis, McIntyre et al[81] confirmed the safety of the restrictive strategy. Additionally, practices to be avoided include using blood as a volume expander and transfusing blood preemptively in anticipation of future operative blood loss. Advanced age should not be used as a sole criterion to transfuse a patient. Further guidelines for the transfusion of RBCs in trauma patients in the postresuscitation period are available.[82,83]

Nutritional Support

The timing, adequacy, and route of administration of nutrition to trauma patients have definite implications for infectious complications. Adequate nutrition is essential for patient recovery, healing of traumatic wounds, and posttrauma rehabilitation. This is because trauma causes increased metabolism and protein turnover that results in a catabolic state characterized by breakdown of skeletal muscle, impaired healing, and immunosuppression. Once acute resuscitation is complete, nutritional support should be instituted, and the enteral route is preferred. Numerous trials have compared enteral nutrition (EN) with parenteral nutrition (PN). Advantages of the enteral route include lower cost, maintenance of function of the gut mucosal barrier, and more physiologic delivery of nutrients; PN may be administered as soon as intravenous access is available and in the setting of ileus or gastrointestinal tract discontinuity commonly seen in critically ill trauma patients. While past evidence strongly favors the use of EN over PN in trauma patients in regard to infectious complications,[84,85] newer studies support the use of early PN with decreased risk of infection, hyperglycemia, and overfeeding.[86,87]

An additional factor that has been implicated in infectious complications is poor glycemic control. Current recommendations relating to glycemic control are in flux, but it appears clear that results are improved when hyper- and hypoglycemia are avoided. Enthusiasm for "very tight" glucose control has waned after several studies showed no

benefit and increased complications with attempts to maintain blood glucose less than 110 mg/dL.[88-91] Newer consensus documents favor continuous glucose monitoring and dynamic insulin protocols to optimize glucose control, although one randomized controlled trial demonstrated that computerized decision support for tight glucose control was associated with more frequent severe hypoglycemic episodes.[92,93]

Tracheostomy

Although several studies comparing early and late tracheostomy have been performed, there is still no consensus regarding whether earlier tracheostomy impacts development of ventilator-associated pneumonia.[94-96] The decision to perform tracheostomy is often institution and surgeon specific, but increasing evidence is mounting that in acutely brain-injured patients, early tracheostomy is favorable to prolonged intubation. The most recent Cochrane Database meta-analysis showed that mortality was decreased slightly (relative risk [RR], 0.83) and that the likelihood of discharge from the ICU was higher at day 28 (RR, 1.29) with early versus late tracheostomy.[97] A systematic review of 10 randomized controlled trials of early versus late tracheostomy following acute brain injury demonstrated not only lower ICU and long-term mortality, but also decreased ICU ventilator days, suggesting that early tracheostomy (defined in this review as within the first 10 days of intubation) may improve access to intensive care beds by enabling earlier ICU discharge and transfer to long-term care facilities.[98,99]

TRAUMA-RELATED INFECTIONS

Diagnosis of Infection

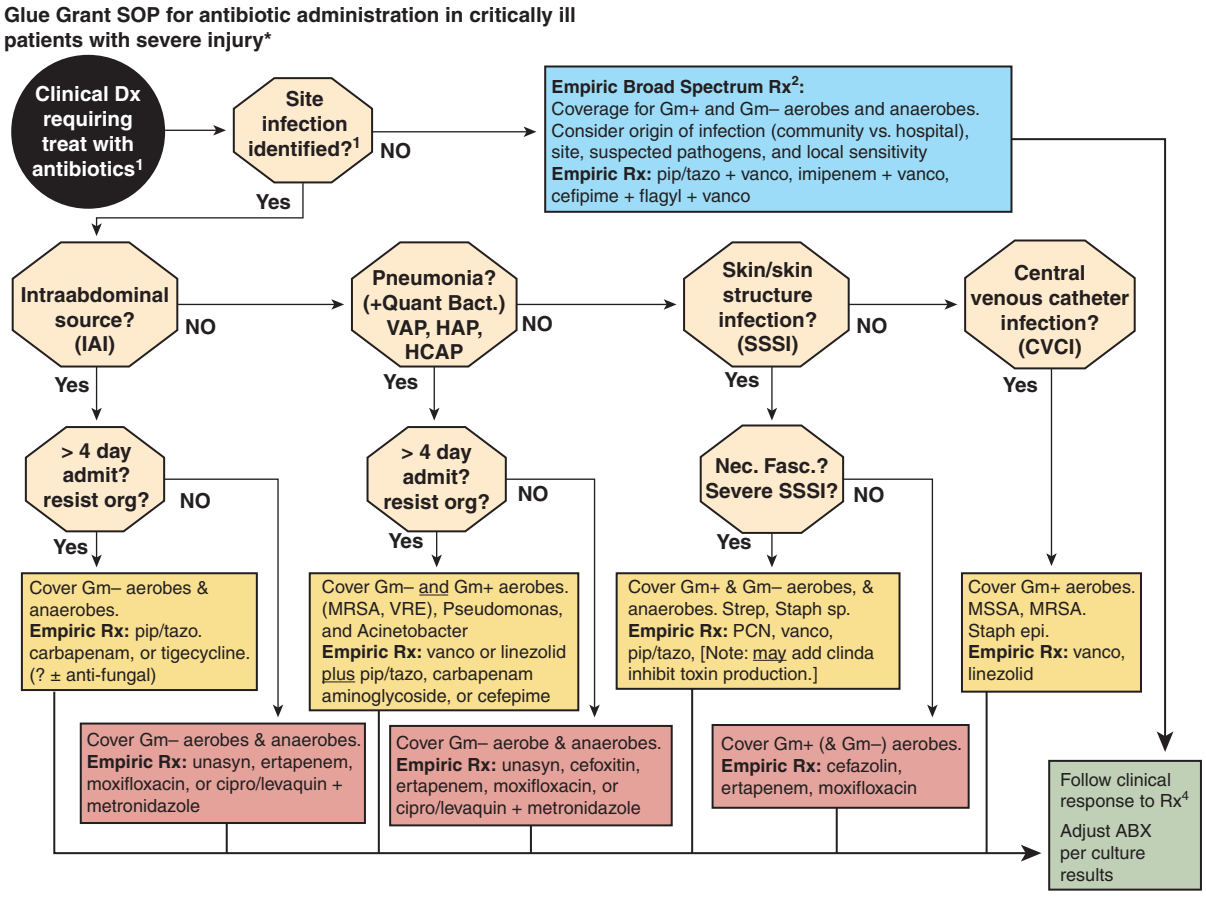
Almost by definition, infectious problems are rarely the presenting complaint of a patient who has sustained an acute traumatic injury. Recognition of an infection in a patient recovering from traumatic injuries, however, continues to be a common, sometimes challenging, clinical problem. Frequently, many of the signs and symptoms commonly associated with infections are also present in trauma patients. The immediate physiologic and immunologic response to tissue injury is initiation of the inflammatory response. Acute traumatic injuries cause the cardinal signs of inflammation including pain (*dolor*), edema (*turgor*), heat (*calor*), redness (*rubor*), and loss of function (*functio laesa*). Trauma victims will often manifest several of the SIRS criteria (eg, tachycardia, elevated temperature, elevated white blood cells [WBCs]) in a clinical context where they are at increased risk for infection.[100]

The diagnosis of infection requires a high clinical suspicion tempered by knowledge of the most likely infectious complications at various time points after injury and filtered by experience with caring for patients with similar injuries. When prevention measures have been ineffective, the diagnosis of infection is based on clinical, laboratory, and imaging methods. Most trauma patients, especially those requiring operative interventions, those with open fractures, or those who have sustained penetrating trauma, will be treated with a course of empiric antibiotics (Table 21-6). The choice of specific antimicrobial agents is determined by the endogenous pathogens or likely exogenous contamination at the anatomic site of injury (eg, exposure to *Vibrio* spp. with marine trauma, exposure to *Clostridium* spp. with farm injury).

Hospitalized patients are at risk for development of *nosocomial* infections, and the diagnosis is made based on a high suspicion coupled with laboratory and/or radiologic confirmation. In most cases, bacterial culture constitutes the gold standard for diagnosis of infection, although at times it can be impractical or impossible to obtain adequate samples. The most specific culture information, if available, is obtained with quantitative or semiquantitative methods (eg, burn wound biopsy, bronchoscopic alveolar lavage [BAL], or mini-BAL). In cases where cultures cannot be obtained, empiric treatment is initiated based on the most likely pathogenic organisms and adjusted based on clinical response. Evidence-based recommendations have been developed for diagnosis and antimicrobial treatment of most hospital-acquired infections,[51,101-104] albeit not specifically addressing trauma patients. The trauma Glue Grant (www.gluegrant.org) proposed a standard operating procedure (SOP) to identify the source of infection in critically ill trauma patients.[105] An updated adaptation of the SOP is reproduced in Fig. 21-4. This approach emphasizes and prioritizes the most likely infections and suggests acceptable antimicrobial agents that are otherwise consistent with evidence-based guidelines from infectious disease societies for a wide range of different infections. Diagnostic imaging, particularly cross-sectional imaging, and/or ultrasound can be helpful to identify and access potentially infected fluid collections in deep locations. Percutaneous aspiration and/or drainage has largely replaced the need for operative management of deep infections and abscesses.[106,107]

Intra-Abdominal Infections

Intra-abdominal infections may complicate both blunt and penetrating abdominal injury. The presence of very high numbers of bacteria within the gut, coupled with the impaired perfusion present in shock states and the immune alterations associated with trauma, result in a 10% to 25% incidence of intra-abdominal infection. Appropriate resuscitation and empiric antibiotics, along with sound intraoperative decision making, minimize the risk for infectious complications. Early source control is crucial to limit the likelihood for postoperative infections. Clinical examination may provide sufficient information to warrant abdominal exploration, and the presence of peritoneal signs or other signs of an acute abdomen should not be ignored in trauma patients. However, hollow viscous injury may be difficult to recognize upon initial presentation, especially if patients arrive after field intubation or if there are distracting injuries that belie a good examination of the abdomen. Peritonitis and acute abdominal signs may develop if bacterial contamination has been present for more than 12 hours. The presence of unexplained free fluid or free air on initial imaging studies is highly suspicious for



1 Important to try, whenever possible, to obtain cultures from suspected sites of infection prior to initiating antimicrobial therapy.

2 Empiric choice should include ≥ 1 antibiotic with activity directed against the likely pathogens.

3 Adjust antibiotics with eye toward appropriate "de-escalation" of therapy based on culture results, response to therapy and clinical condition.

4 Always reassess antimicrobials after 48–73 hr based on micro and clinical data. There is no evidence that combination Rx is more effective than mono Rx if microorganisms are sensitive to agent. Duration of Rx should typically be 7–10 days.

* Individual antibiotic choices will be heavily influenced by local ID restrictions and/or formulary choices. This guideline is intended to highlight parameters that need to be considered in choice of agents.

FIGURE 21-4 Diagnostic approach to identify and treat postinjury infections in critically ill trauma patients. Note that oral metronidazole is no longer recommended as first-line treatment for *Clostridium difficile* colitis (enteral vancomycin is first line, with intravenous [IV] Flagyl and vancomycin enemas added in the setting of septic shock). ABX, antibiotics; Dx, diagnosis; Gm, Gram stain; HAP, hospital-acquired pneumonia; HCAP, health care–associated pneumonia; IAI, intra-abdominal infection; MRSA, methicillin-resistant *Staphylococcus aureus*; MSSA, methicillin-sensitive *S aureus*; PCN, penicillin; Rx, treatment; SOP, standard operating procedure; VAP, ventilator-associated pneumonia; VRE, vancomycin-resistant *Enterococcus*. (Reproduced, with permission, from West MA, Moore EE, Shapiro MB, et al. Inflammation and the host response to injury, a large-scale collaborative project: patient-oriented research core–standard operating procedures for clinical care VII: guidelines for antibiotic administration in severely injured patients. *J Trauma.* 2008;65(6):1511-1519.)

injury to a hollow viscus, but other signs such as mesenteric hematoma or bowel thickening are also suggestive and may warrant exploration.[108]

Intra-abdominal infections identified later in the hospital course (>4 days after injury) are much more likely to be caused by hospital-associated, rather than community-associated, organisms, as most patients will have received one or several doses of relatively broad-spectrum antibiotics that alter the bacterial flora.[101] Late intra-abdominal infections may grow *Pseudomonas* spp., *Serratia* spp., or *Candida* spp. Percutaneous aspiration and/or drainage has become the mainstay for treatment of late intra-abdominal abscesses and fluid collections. Whenever possible, material from intra-abdominal collections should be sent for Gram stain, culture, and sensitivity determination to guide therapy. The Surgical Infection Society (SIS) and the Infectious Diseases Society of American (IDSA) published updated evidence-based guidelines to inform antibiotic choices for intra-abdominal infections.[109] These guidelines differentiate between community- and hospital-acquired infections (Tables 21-7 and 21-8). As a general rule, antimicrobial coverage directed against community abdominal pathogens may be given within the first 3 days after injury, whereas antibiotic choices 4 or more days after injury should anticipate hospital organisms. A recent multicenter, randomized controlled trial of intra-abdominal infection demonstrated that short duration (4 days) of antimicrobial therapy after definitive source control of infection is as effective as treatment extended twice as long.[110] To avoid antibiotic-associated complications, such as *C difficile* colitis and infection and/or colonization with antibiotic-resistant organisms, antibiotic exposure should be limited to the shortest duration possible.

Empyema

Under normal circumstances, the pleural cavity has a net negative pressure and a very small (<20 mL) volume of fluid.[111] Current understanding of fluid flux within the pleural cavity implicates parietal pleural lymphatics as the main route through which fluid is removed.[112] Pleural fluid normally turns over at a rate of approximately 0.15 mL/kg/h (10–12 mL/h for a 70-kg individual), and the maximum capacity for lymphatic drainage is estimated to be 700 mL/d (~30 mL/h). When there is increased production or decreased clearance of pleural fluid, a pleural effusion develops. Pleural effusions are frequently associated with fluid overload, but they may also arise in the setting of acute inflammatory processes of the lung including pneumonia (parapneumonic effusion), acute lung injury, or ARDS. Blunt and penetrating chest trauma can induce alterations within the pleural cavity, with loss of negative pressure (traumatic pneumothorax), accumulation of blood (hemothorax), or a combination of both (hemopneumothorax).

TABLE 21-7: SIS/IDSA Evidence-Based Guidelines for Antibiotic Choices for Community-Acquired Intra-Abdominal Infections

	Adults		Children
	Mild-moderate severity	**Severe or high-risk patients**	**Severe or high-risk patients**
Monotherapy (single agent)	Cefoxitin Ertapenem Moxifloxacin[a] Tigecycline Ticarcillin-clavulanate	Imipenem-cilastatin Meropenem Doripenem Piperacillin-tazobactam	Ertapenem Meropenem Imipenem-cilastatin Ticarcillin-clavulanate Piperacillin-tazobactam
Combination therapy[b]	Cefazolin Cefuroxime Ceftriaxone Cefotaxime, ciprofloxacin[a] Levofloxacin[a] **plus** Metronidazole	Cefepime Ceftazidime Ciprofloxacin[a] Levofloxacin[a] **plus** Metronidazole	Ceftriaxone Cefotaxime Cefepime Ceftazidime **plus** Metronidazole Gentamicin Tobramycin **plus** Metronidazole Or clindamycin ± Ampicillin

[a]*E coli* resistance to fluroquinolones is increasingly reported, so local sensitivity information should be consulted.
[b]With combination regimens, acceptable agents with activity against aerobic gram-negative bacteria are listed first, followed by agents directed against anaerobes. A single agent with aerobic activity should be paired (combined) with an antianaerobic drug.

IDSA, Infectious Diseases Society of America; SIS, Surgical Infection Society.

Source: Adapted from Solomkin JS, Mazuski JE, Bradley JS, et al. Diagnosis and management of complicated intra-abdominal infection in adults and children: guidelines by the Surgical Infection Society and the Infectious Diseases Society of America. *Surg Infect (Larchmt).* 2010;11(1):79-109.

TABLE 21-8: SIS/IDSA Evidence-Based Guidelines for Initial Adult Antibiotic Dosing in Intra-Abdominal Infections

Antibiotic	Adult dosage
β-Lactam/β-lactamase inhibitor combination	
Piperacillin-tazobactam	3.375 g q 6 h (Note: increase dose to 3.375 g q 4 h or 4.5 g q 6 h for *Pseudomonas* infections)
Ticarcillin-clavulanic acid	3.1 g q 6 h (FDA labeling indicates 200 mg/kg/d in divided doses q 6 h for moderate infection and 300 mg/kg/d in divided doses q 4 h for severe infection)
Carbapenems	
Doripenem	500 mg q 8 h
Ertapenem	1 g q 24 h
Imipenem-cilastatin	500 mg q 6 h or 1 g q 8 h
Meropenem	1 g q 8 h
Cephalosporins	
Cefazolin	1–2 g q 8 h
Cefepime	2 g q 8–12 h
Cefotaxime	1–2 g q 6–8 h
Cefoxitin	2 g q 6 h
Ceftazidime	2 g q 8 h
Ceftriaxone	1–2 g q 12–24 h
Cefuroxime	1.5 g q 8 h
Tigecycline	100 mg initial dose, then 50 mg q 12 h
Fluoroquinolones	
Ciprofloxacin	400 mg q 12 h
Levofloxacin	750 mg q 24 h
Moxifloxacin	400 mg q 24 h
Metronidazole	500 mg q 8–12 h or 1500 mg q 24 h
Aminoglycosides	
Gentamicin or tobramycin	5–7 mg/kg q 24 h (based on adjusted body weight)
Amikacin	15–20 mg/kg q 24 h (based on adjusted body weight)
Aztreonam	1–2 g q 6–8 h
Vancomycin	15–20 mg/kg q 8–12 h (based on total body weight)

FDA, Food and Drug Administration; IDSA, Infectious Diseases Society of America; SIS, Surgical Infection Society; q, every.

Source: Adapted from Solomkin JS, Mazuski JE, Bradley JS, et al. Diagnosis and management of complicated intra-abdominal infection in adults and children: guidelines by the Surgical Infection Society and the Infectious Diseases Society of America. *Surg Infect (Larchmt).* 2010;11(1):79-109.

Treatment of either condition generally requires insertion of a thoracostomy tube, and this can introduce bacterial contamination of the fluid or blood present within the pleural cavity, particularly when chest tubes are placed emergently under less than sterile conditions. Because lymphatic stomata of the parietal pleural are the means by which blood and/or fluid is normally reabsorbed, the presence of a fibrin clot can obstruct this route of egress and contribute to persistence of fluid within the chest cavity.

TABLE 21-9: Bacteriology of Posttraumatic Empyema (N = 37)

Organism	No. (%)
Staphylococcus aureus	20 (54%)
Methicillin-resistant *S aureus*	7 (19%)
Pseudomonas aeruginosa	2 (6%)
Streptococcus pneumoniae	1 (3%)
Mixed	4 (11%)
No growth	10 (27%)

Source: Adapted from Hoth JJ, Burch PT, Bullock TK, Cheadle WG, Richardson JD. Pathogenesis of post-traumatic empyema: the impact of pneumonia on pleural space infections. *Surg Infect (Larchmt).* 2003;4(1):29-35.

Diagnosis of empyema requires sampling of fluid or tissue from the pleural space.[113] In the setting of empyema, analysis of pleural fluid will demonstrate a pH of less than 7.20, glucose of less than 40, the presence of bacteria on Gram stain, or a positive culture.[114] Table 21-9 shows the most common bacterial isolates from posttraumatic empyema (although some purulent collections may not grow bacteria). It is now generally understood that posttraumatic empyema arises from exogenous contamination of the pleural cavity. Hoth and colleagues[115] obtained simultaneous BAL and pleural cultures and noted that there was minimal correlation between pleural cultures and BAL samples. Recognition that skin flora is associated with empyema underscores the importance of using sterile technique during insertion of a thoracostomy tube. A meta-analysis of five trials that included patients with blunt or penetrating thoracic trauma found a significant reduction in the risk of empyema (RR, 0.19; 95% confidence interval [CI], 0.04–0.70) and pneumonia (RR, 0.44; 95% CI, 0.15–0.87) for patients who received antibiotic prophylaxis compared with placebo.[116] However, because of conflicting accumulated evidence, in 2012, the EAST Practice Management Guidelines Work Group revised an earlier guideline[117] and declined to make a definitive recommendation for or against the routine use of presumptive antibiotics in trauma thoracostomy placement for traumatic hemopneumothorax to reduce the incidence of empyema and pneumonia.[118]

Complete evacuation of a hemothorax and reexpansion of the lung at the time of initial thoracostomy tube placement are important to prevent empyema. Retained hemothorax and empyema complicate about 4% of patients with a hemothorax treated with a thoracostomy tube.[119] In a prospective, multicenter, observational study of patients with posttraumatic retained hemothorax, the incidence of empyema was 26.8%.[120] Rib fractures, severe injury, and need for additional interventions beyond chest tube thoracostomy to evacuate hemothorax were found to be independent predictors of empyema. Instillation of fibrinolytic agents has been demonstrated to aid in the evacuation of a retained hemothorax; while presence of an intrathoracic injury would seem to increase risk of bleeding complications, this has not been borne out in several case series.[121] Video-assisted

thoracoscopy (VATS) is more widely employed for the treatment of retained hemothorax, but the ideal timing of VATS is unclear, and multiple procedures including conversion to thoracotomy may be required if the hemothorax is large or diaphragm injury is concurrent, or if empyema develops.[118] For patients with flail chest physiology, rib fixation is increasingly used for chest wall stabilization and may prevent the subsequent development of pneumonia or empyema.[122-124]

Osteomyelitis/Septic Arthritis

Trauma-related osteomyelitis and septic arthritis may complicate musculoskeletal injury.[125] Posttraumatic musculoskeletal infections generally arise from bacteria introduced exogenously, either at the time of injury or during operative repair. This is in contrast with hematogenous dissemination of bacteria with most nontraumatic bone and joint infections. Although a variety of bacterial species have been isolated from posttraumatic osteomyelitis and septic arthritis (Table 21-10), *Staphylococcus* species are far and away the most common isolates.[126,127] *S aureus* and *Staphylococcus epidermidis* have a number of virulence factors that provide a particular predilection to bone tissue.[127] These virulence factors, summarized in Table 21-11, include adhesive properties and exotoxins and enzymes that facilitate invasion. Another property important to the development and persistence of osteomyelitis is a small colony variant (SCV) phenotype that grows more slowly with increased resistance to aminoglycosides and decreased hemolytic activity.[126] Clinical use of aminoglycoside beads and broader-spectrum antibiotics may select for these more resilient SCV phenotypes in vivo.

TABLE 21-10: Bacteria Isolated from Septic Joints and Osteomyelitic Bone Infections

Bacteria isolated from joint infections

- *Staphylococcus aureus*
- *Streptococcus pyogenes*
- *Streptococcus pneumoniae*
- *Escherichia coli*
- *Pseudomonas aeruginosa*
- *Serratia marcescens*
- *Salmonella* species
- *Neisseria* species
- *Aerobacter* species
- *Bacteroides* species

Bacteria isolated from bone infections

- *Staphylococcus aureus*
- *Staphylococcus epidermidis*
- *Streptococcus* species
- *Haemophilus influenzae*
- *Escherichia coli*
- *Pseudomonas aeruginosa*
- *Salmonella* species
- *Mycobacterium* species

Source: Adapted from Wright JA, Nair SP. Interaction of staphylococci with bone. *Int J Med Microbiol.* 2010;300(2-3):193-204.

TABLE 21-11: Selected Virulence Determinants for *Staphylococcus aureus* Bone and Joint Infections

Bacterial determinant	Putative function
Collagen-binding protein	Adhesin for collagen
Protein A	Interferes with opsonization and phagocytosis
Polysaccharide capsule	Resists phagocytosis and bacterial killing
Enterotoxins A, B, C, and D	Superantigens
TSST-1	Superantigen
Alpha-, beta-, and gamma-toxin	Cytolytic toxins
Agr	Gene regulator
Sar	Gene regulator
CpG motifs	DNA, TLR9-dependent release proinflammatory cytokines

Source: Adapted from Mandal S, Berendt AR, Peacock SJ. *Staphylococcus aureus* bone and joint infection. *J Infect.* 2002;44(3):143-151.

The presence of contaminating bacteria at the site of a bone or joint injury incites a vigorous local inflammatory response. The local source of the inflammatory cytokines TNF, IL-1, and IL-6 is not entirely clear, but osteoblasts may contribute. In any case, high local levels of proinflammatory cytokines have dramatic effects on bone turnover and new bone formation. TNF and IL-1 induce increased maturation of osteoclasts and enhance osteoclastic activity.[128] At the same time, these mediators inhibit mesenchymal cell differentiation into osteoblasts. Similar processes contribute to destruction of cartilage and bone in chronic arthritic conditions. In such settings, anti-TNF and anti–IL-1 therapies have been very successful in preventing inflammation.[129] The net impact of increased osteoclast and decreased osteoblast activity is either bone destruction or inhibition of bone healing. Furthermore, contact between osteoblasts and *S aureus* can induce TNF-related apoptosis-inducing ligand (TRAIL), which, in the presence of Fas-associated death domain (FADD), commits cells to apoptotic cell death.

Several unique factors associated with bone infection underscore the importance of evidence-based recommendations that emphasize early and continuing aggressive debridement of orthopedic injuries, particularly those associated with open or contaminated wounds.[130] During the initial evaluation of the patient in the emergency room, it is imperative to promptly begin antibiotic prophylaxis for open fractures, with antibiotic coverage increasing with degree of injury, tissue devitalization, and contamination with soil or standing water.[131,132] At sites of bone or joint injury, local blood supply to fragments may be interrupted, predisposing to bone necrosis. Lack of blood supply also precludes delivery of systemic antibiotics, host inflammatory cells, and the molecular oxygen needed for oxygen-dependent bactericidal activity of such cells. It is axiomatic that all dead bone must be removed to minimize the risk of bone infection. In addition, many

orthopedic injuries require the presence of foreign bodies, such as plates or rods, to stabilize fractures. Experimentally only very low levels of bacteria are needed to produce infection in the presence of foreign material. In the presence of extensive local bony destruction and/or contamination, many orthopedic surgeons use external fixation devices to minimize, but not eliminate, the risk of infection and nonunion.

Occupational Exposure to Environmental Pathogens

It is an unfortunate reality that health care workers in general, and surgeons in particular, are exposed to bloodborne occupational hazards, including hepatitis B virus (HBV), hepatitis C virus (HCV), and HIV. Body fluids considered potentially infectious include blood, cerebrospinal fluid, synovial fluid, pleural fluid, peritoneal fluid, pericardial fluid, and amniotic fluid. Conversely, exposures to feces, nasal secretions, saliva, sputum, sweat, tears, urine, and vomitus are not considered potentially infectious, as the risk for transmission of HBV, HCV, and HIV infection from these fluids is extremely low.[133] PPE demonstrated to be effective include double gloving, using blunt needles for fascial closure, protective eye shields, impervious surgical gowns, and routine implementation of universal precautions.

The CDC estimates that more than 2.7 million US citizens (~1%) have chronic HCV (http://www.cdc.gov/hepatitis/HCV/HCVfaq.htm) and that 1.2 million Americans are living with HIV (http://www.cdc.gov/hiv/basics/livingwithhiv/index.html). One epidemiologic study has reported the prevalence of HIV and HCV to be as high as 20% to 65% and 10% to 45%, respectively, in an urban university hospital population for patients undergoing lymph node biopsy or drainage of a soft tissue infection.[134] A 2-year prospective study of penetrating trauma patients at an urban trauma center reported a 9% rate of HIV, HBV, or HCV, with 75% of these patients unaware of their seropositive status prior to admission.[135] In addition to reducing the potential inoculum by sixfold, double gloving affords the benefit of detecting glove failure, if indicator gloves are used.[136]

While prevention is the best course of action, certain measures taken after an exposure can decrease the risk of seroconversion. The first step involves treatment of the exposure site. Wounds and skin sites should be washed with soap and water, and mucous membranes flushed with water. There is no evidence to support applying antiseptics to the wound or expressing fluid to reduce the risk of transmitting a bloodborne pathogen. The CDC strongly discourages the application of caustic agents (eg, bleach) or the injection of antiseptics or disinfectants into the wound. Second, the exposure source (ie, the patient) should be evaluated for their HBV, HCV, and HIV status. If the status is unknown, the patient should be informed of the incident. The health care practitioner must be aware of applicable state and local laws regarding informed consent for serologic testing. Testing of needles or other sharp instruments involved in the exposure is not acceptable as a replacement or complement to testing the source patient. Additional up-to-date resources and recommendations are available via the National Clinician's Postexposure Prophylaxis (PEP) Hotline (PEPline, 888-448-4911) or via http://nccc.ucsf.edu/clinical-resources/pep-resources/pep-guidelines/.

When an exposure to HBV occurs, the risk of seroconversion is dependent on the degree of contact (ie, size of the inoculum) and the hepatitis B e antigen (HBeAg) status of the source. For example, if the patient is HBeAg positive, the risk of developing HBV infection is about 50%, compared to 25% if the patient is HBeAg negative. PEP includes hepatitis B immune globulin (HBIG) and, possibly, the hepatitis B vaccination series, depending on the hepatitis B antigen status of the patient and the antibody status of the at-risk health care worker. If indicated, HBIG should be given as soon as possible, since early administration after exposure to hepatitis B surface antigen–positive blood can provide an estimated 75% protection from HBV infection.[133]

In contrast to HBV, HCV is not efficiently transmitted via occupational exposure. It is estimated that HCV seroconversion after accidental percutaneous exposure from an HCV-positive source occurs 1.8% of the time, and some have suggested that transmission occurs only from puncture by hollow-bore needles. Transmission has never been reported after intact or nonintact skin exposure to blood and only rarely occurs after exposure of mucous membranes to blood. Currently, IVIG is not recommended after occupational HCV exposure. The rationale is based on several clinical observations, including the following: (1) HCV infection does not incite a protective antibody response; (2) studies of IVIG use for PEP in virus and HBV cannot be extrapolated to HCV; and (3) HCV IVIG use in chimpanzees has failed to prevent HCV seroconversion after exposure.[133] There is no evidence that the administration of interferon-α or antiviral agents prevents HCV infection after occupational exposure, and their use is not currently recommended. The exposed health care worker should be tested for baseline HCV viral status and should have continued close follow-up for 12 months for the purpose of early identification should seroconversion occur. Transient suppression of viremia can occur with acute HCV infection, even among those who progress to chronic HCV, so a single negative test is not enough to rule out infection.[137] Additionally, the health care worker should contact the CDC Hepatitis Information Line (888-443-7232) or see http://www.cdc.gov/hepatitis/hcv/index.htm.

Like HCV, transmission of HIV occurs rarely after occupational exposure. The CDC estimates the risk of seroconversion at approximately 0.3% following a percutaneous exposure to HIV-infected blood and 0.09% after exposure of a mucous membrane. For exposure to fluids or tissues other than HIV-infected blood, the risk of transmission has not been quantified but is probably much lower. In 2013, the US Public Health Service updated the recommendations for HIV PEP to include three agents: raltegravir and Truvada (tenofovir plus emtricitabine).[138] To maximize the possibility of protection, the health care worker exposed to HIV should be evaluated within hours, and PEP should be initiated

as soon as possible. A baseline HIV test should be performed, and HIV antibody testing should be performed for at least 6 months after exposure (at 6 weeks, 12 weeks, and 6 months). A 4-week regimen is advised for most HIV exposures. If the source person's virus is known to be resistant to the routine PEP regimen, selection of an alternate regimen is highly recommended.

CHRONOLOGIC APPROACH TO PREVENTION, RECOGNITION, AND TREATMENT OF INFECTIONS IN TRAUMA PATIENTS

Resuscitation Bay

Efforts to minimize infection must be initiated as soon as the patient arrives in the trauma bay. Although the initial focus will appropriately center on control of hemorrhage and initiation of resuscitation, these efforts will reduce the risk of infection as well. Restoration of adequate blood flow and oxygen delivery is the first step in reducing the incidence of infection. It has been clearly shown that the incidence of infection from invasive procedures in the ICU, such as insertion of a central venous line, is dramatically decreased by employing handwashing, full-barrier precautions, and chlorhexidine skin preparation as part of an evidence-based procedural bundle to prevent infection.[139] Full-barrier precautions may not always be practical in severely injured victims, but suspension of proven infection control measures and sterile technique for invasive procedures should be the rare exception. Maintenance of normothermia likewise represents optimal treatment for the trauma patient and has the additional benefit that it will decrease the likelihood for development of infectious complications.[61] Contaminated wounds should be cleaned and/or formally debrided in an expeditious time frame, although more recent reviews of experience in high-energy orthopedic injuries have downplayed the importance of time to debridement as the most important factor to prevent osteomyelitis.[140]

Prophylactic or empiric antibiotics, if indicated, should be initiated in the trauma bay. Antibiotics should be started in patients with open fractures or penetrating abdominal injuries and in any patient in whom there is a high likelihood of injury to a hollow viscus.[131,141] Due to the hostile environment in which military injuries occur, current recommendations are that injured soldiers start oral or parenteral antibiotics as soon as possible if there is any break in the skin.[142] Patients with blunt mechanisms who require operative interventions should receive perioperative antibiotic prophylaxis according to established evidence-based guidelines. Antibiotic choices for different anatomic regions are compiled in Table 21-6.

Operating Room

The conduct of operative interventions can significantly impact the likelihood of postoperative infectious complications. The primary factors determining the risk for infection are the nature and magnitude of the traumatic injury requiring surgical intervention. The surgeon must remember that the highest initial priority during an exploratory laparotomy for hemorrhagic shock is bleeding control and not control of enteric content. After acute hemorrhage control, it is appropriate to stop ongoing leakage from the bowel and to evacuate gross contamination. In addition to the bacterial contamination arising from enteric leakage, disruption of gastrointestinal integrity also releases foreign bodies (eg, undigested food), mucin, and bile. These adjuvant substances significantly decrease the bacterial inoculum needed for clinical infection via two mechanisms. First, some adjuvant substances augment bacterial growth or stimulate bacteria to express virulence factors.[143] Second, they may interfere with host defense mechanisms such as the function of innate immune cells.[15] For example, the detergent activity of bile can lyse polymorphonuclear leukocytes and macrophages. Blood and devitalized tissue are two additional adjuvant factors frequently present. Blood (specifically hemoglobin) can be converted to a leukotoxin by some enteric bacteria.[144] Fibrin clots sequester bacteria and make them inaccessible to host phagocytes, and this may predispose to late development of intra-abdominal abscesses.[145,146] Devitalized, ischemic, or necrotic tissues are also potent sources of damage/danger signals that activate host immune cells and exacerbate acute inflammation. At the same time, these substances can interfere with phagocytosis and oxidative killing mechanisms of the host defense cells.[9,147] Thus, to the extent possible, blood and blood clots should be removed from the peritoneal cavity. This can be accomplished by irrigating the peritoneal cavity with a goal of removing the obvious contamination, foreign material, and blood. It is worth noting that a prospective randomized study showed no benefit to formal meticulous debridement to remove fibrinous debris from the peritoneal cavity in established peritonitis.[148] Additionally, neither a meta-analysis of a rather heterogeneous set of studies of intraoperative irrigation nor a recent single-center, randomized controlled trial of different volumes of intra-abdominal irrigation in emergency trauma laparotomy demonstrated benefit in preventing SSI.[149,150] Increased deep SSI in the large-volume (20 L) irrigation group compared to 5 L of irrigation suggests that the least amount of irrigation sufficient to remove gross contamination and adjuvant material should be used, avoiding leaving exogenous fluid in the abdominal cavity.

Evidence-based guidelines for antimicrobial prophylaxis of trauma recommend broad-spectrum agents with activity against the anticipated pathogens that are likely to be encountered at the anatomic area of injury.[141] With respect to the infectious risk after blunt or penetrating abdominal injury, this requires coverage against colonic bacterial flora. In the case of abdominal trauma, agents with activity against aerobic and anaerobic bacteria are recommended.[151-153] However, extended courses of antibiotics are not indicated, even in the setting of hollow viscus injury, and there is no current evidence to support continued antibiotic administration following damage control operations prior to abdominal fascial closure.[54,141] With injuries to the extremities, the most likely

pathogens will be aerobic gram-positive bacteria, particularly *Staphylococcus* species. Injury to maxillofacial structures requires antibiotic prophylaxis with activity against normal oral flora, and neurosurgical procedures most often employ agents similar to those used for the extremities.

Little is known about the pharmacology of antibiotics in the acute resuscitative phase of trauma. Most available pharmacologic data derive from healthy individuals and, therefore, cannot be applied directly to injured patients, but there are a few studies in critically ill patients. Buijk et al[154] described a cohort of 89 septic patients who received aminoglycosides. The volume of distribution was significantly higher than in those without septic shock, and the maximum antibiotic concentration achieved was significantly lower. In a study of patients who required significant resuscitation with fluids and blood during a laparotomy, the volume of distribution was expanded and correlated with the degree of fluid resuscitation.[155] Antibiotic elimination was also more rapid in these injured patients compared with normal controls.

With massive blood loss, antibiotic prophylaxis requires frequent redosing to maintain plasma and tissue levels above the mean inhibitory concentration (MIC). Animal models of experimental infection after hemorrhagic shock report better prophylaxis with increasing doses of appropriate intraoperative antibiotics.[156] Large-volume resuscitation and altered endothelial permeability with trauma or burns result in an expanded volume of distribution. Renal dysfunction from hypovolemia, myoglobinuria, or radiologic contrast often accompanies severe injury, but the potential risk of a nephropathy has no impact on acute antibiotic dosing. The greatest risk for subsequent infectious complications arises from underdosing rather than overdosing in acute trauma.

The conduct of the operation itself significantly impacts the risk of infection. Damage control resuscitation has the collateral benefit of positively impacting the incidence of postoperative infectious complications.[157,158] In most cases, vascular reconstruction and bowel anastomoses, if needed, should be delayed until the patient is warm, adequately resuscitated, and hemodynamically stable. If the patient is hemodynamically stable, euvolemic, and normothermic, then there is no adverse impact to definitively managing abdominal, vascular, neurosurgical, or orthopedic injuries during the first operation. In a grossly contaminated wound, primary closure is associated with an unacceptably high wound infection rate. Delayed primary closure (DPC), a practice dating back to Ambrose Pare, was advocated by surgical pioneer John Hunter in the 1700s and popularized during World War I.[159] It is based on development of fine granulations within the wound prior to definitive closure. Thus, DPC combines the infective resistance of healing by secondary intention with the cosmetic benefits of primary closure. Randomized prospective trials have reported significantly lower rates of wound infections compared to primary closure of grossly contaminated wounds.[160-162]

The management of penetrating wounds to the colon has evolved since World War II, when the diverting ostomy reduced mortality rates to about 30% in the preantibiotic era. With improvements in trauma resuscitation and accumulating experience with antibiotics, investigators began to question whether fecal diversion was necessary after colonic repair. Initially, Stone and Fabian[163] published the first prospective randomized trial of colostomy versus primary repair. They excluded patients with "high-risk" criteria such as shock, hemorrhage, greater than two organs injured, gross contamination, operative delay greater than 8 hours, injury requiring resection, and loss of the abdominal wall. Subsequent investigations have reported that primary colon repair, even in the face of "high-risk" criteria, is associated with a decreased incidence of infectious complications compared to diverting ostomy.[164-166] In a more recent American Association for the Surgery of Trauma multicenter cohort study of predominantly penetrating rectal injuries, for patients with intraperitoneal injury, diversion was not associated with better outcomes; distal rectal washout and presacral drainage at the time of the index operation for extraperitoneal injuries were independent risk factors for developing subsequent abdominal complications.[167]

Open pelvic fractures are associated with extremely high morbidity and mortality, including from septic complications. Diverting colostomies are often placed in such patients, but the evidence supporting this approach is weak and generally derived from small retrospective studies. One systematic review found no difference in the overall infection rate with or without colostomy, with the exception of a lower complication rate when colostomy was used for perineal/rectal wounds.[168] Subsequent single-center retrospective studies have demonstrated a higher rate of mortality with rectal laceration in the setting of shock and absence of diversion.[169,170] More studies are required to provide definitive recommendations, but diversion is still the preference for many surgeons in the setting of extraperitoneal rectal injury.[171]

Recommendations for management of extremity fractures continue to evolve. A large study found that the most important factor in outcome was early transfer to a trauma center for definitive management.[140] Specifically, this study called into question the benefit of early operative debridement of open fractures, inasmuch as time to operative debridement did not confer a statistically significant benefit. The concept of damage control has also been applied to orthopedic injuries.[172] The military experience in Iraq and Afghanistan also underscores the utility of this concept, coupled with more liberal use of external fixation devices to minimize infectious complications in this hostile environment.[173] With proper debridement and acute management of the fracture, bacteria inoculated into fractures at the time of injury are rarely isolated from postoperative infections. Rather, hospital-acquired flora are almost universally responsible for infections in fractures.[174]

Intensive Care Unit and Early Postoperative Period

Infectious complications are commonly encountered in the early postoperative period and are even more likely to be seen

TABLE 21-12: Health Care–Associated Infections (HAI) in the Surgical/Trauma Intensive Care Unit

Pneumonia
Ventilator-associated events (VAE)
Ventilator-associated pneumonia (VAP)
Central line–associated bloodstream infections (CLABSI)
Intra-abdominal infections (IAI)
Intra-abdominal abscess
Secondary peritonitis
Tertiary peritonitis
Catheter-associated urinary tract infections (CAUTI)
Skin and soft tissue infections
Superficial surgical site infections (SSI)
Decubitus ulcers
Burn wound sepsis
Clostridium difficile infection (CDI)
Empyema
Sinusitis

in patients who require ongoing critical care (Table 21-12). Clinicians should have a high index of suspicion and consider potential sources of infection in the context of the injuries, characteristics of the patient, likely offending organisms, and length of hospital stay. The immediate postoperative course of a severely injured patient typically involves a vigorous systemic inflammatory response.[100] Clinical signs such as mild to moderate fever and tachycardia are almost universal. Furthermore, it is now known that danger signals released from injured tissue activate innate immune responses via the same TLR pathways stimulated by bacterial infection. Leukocytosis is a common manifestation, so monitoring the WBC count immediately after injury has little utility. It is useful to keep the overall trajectory of the patient in mind before automatically initiating a series of expensive and low-yield investigations (eg, blood cultures, urinalysis, x-rays). Identifying infection is particularly difficult in the ICU setting.[175] Injured patients are at increased risk for development of nosocomial infections such as pneumonia, central line–associated bloodstream infections (CLABSIs), catheter-associated urinary tract infections (CAUTIs), *C difficile* infection (CDI), and SSI; however, critically ill trauma victims are susceptible to severe sepsis and septic shock as well.

The most recent update of the Surviving Sepsis Campaign guidelines restates the fundamental definition of sepsis as a "life-threatening organ dysfunction caused by a dysregulated host response to infection." Specifically, the diagnosis of sepsis now requires an increase in the Sequential Organ Dysfunction Score (SOFA) from baseline by 2 points or greater in the setting of suspected or confirmed infection. This change was initiated in order to provide more objective clinical criteria that were demonstrated to be more predictive than the SIRS criteria intensivists have relied on to date.[176] The 2018 update of the Surviving Sepsis Campaign includes a directed guideline for the first hour of care after sepsis is recognized

TABLE 21-13: Hour 1 Surviving Sepsis Campaign Bundle of Care

Measure lactate level. Remeasure if initial lactate is >2 mmol/L.
Obtain blood cultures prior to the administration of antibiotics.
Administer broad spectrum antibiotics.
Begin rapid administration of 30 mL/kg crystalloid for hypotension or lactate ≥4 mmol/L.
Apply vasopressors if patient is hypotensive during or after fluid resuscitation to maintain MAP ≥65 mm Hg.
Time of presentation is defined as the earliest time of triage or from the earliest chart annotation consistent with all elements of sepsis (formerly called severe sepsis) or septic shock ascertained through chart review.

Source: Adapted, with permission, from Levy MM, Evans LE, Rhodes A. The Surviving Sepsis Campaign Bundle: 2018 update. *Crit Care Med.* 2018;46(6): 997-1000.

(Table 21-13). It is unclear how the Sepsis 3 definitions will differently affect the care of the critically injured trauma patient with suspected infection, as clinicians adjust to applying the new evaluative strategy. While the intent behind these changes is to allow for more prompt recognition and discrimination of sepsis from other physiologic responses, prospective evaluation of this new strategy and the impact on timely, appropriate resuscitation and early institution of broad-spectrum antimicrobial agents is essential.

HEALTH CARE–ASSOCIATED INFECTIONS

Pneumonia

As in sepsis, the definition of pneumonia has undergone reevaluation and the result is controversial. Currently, pneumonia can still be divided into community-acquired pneumonia (CAP) and hospital- or health care–associated pneumonia (HCAP), with the former being present on admission and the latter manifesting later in the hospital course. HCAP can further be divided into early (<4 days) and late (≥4 days). These distinctions are more than merely pedantic, as pneumonia that develops later is more likely caused by multidrug-resistant organisms. Patients with CAP or early HCAP who have no known risk factors should be treated with ceftriaxone or levofloxacin (or equivalent), ampicillin/sulbactam, or ertapenem in accordance with local prevalence patterns.[177]

Within the broader category of HCAP, an important subgroup are patients on mechanical ventilation who develop pneumonia. Ventilator-associated pneumonia (VAP) is a leading cause of morbidity and mortality in the injured population and remains a daunting diagnostic challenge. The definition of VAP is very nonspecific, does not correlate well with histopathologic findings of pneumonia, and is a poor-quality measure in the ICU. In 2013, in an effort to improve the surveillance of noninfectious etiologies of pulmonary dysfunction in mechanically ventilated patients, the NHSN

released the guidelines for detecting ventilator-associated events (VAEs).[178] This new guideline replaces the older definition of VAP; it is intentionally broad in order to encompass all pulmonary complications affecting critically patients, including ARDS, atelectasis, and cardiogenic sources of pulmonary edema. The new definition consists of a three-tiered progression from ventilator-associated condition (VAC) to infection-related ventilator-associated condition (IVAC) to probable ventilator-associated pneumonia (PrVAP). This is a shift away from reporting only on the infectious complications that affect mechanically ventilated patients and is thought to improve comparability, facilitate automation, and minimize gaming in the reporting of pulmonary dysfunction in ICU patients.

The first tier of VAE is a VAC. This occurs when a patient (ventilated for at least 2 days) has more than 2 days of worsening oxygenation (defined by >0.2 increase in the minimum daily fraction of inspired oxygen or positive end-expiratory pressure increase of >3 cm H_2O). The second-tier VAE is the IVAC that occurs when the criteria for VAC have been met in the presence of (1) fever, (2) abnormal WBC (>12 or <4 × $10^3/mm^3$), and (3) the initiation of antibiotics for >4 days. The third VAC tier consists of possible VAP (PoVAP) and PrVAP. PoVAP is considered when there is the presence of purulent secretions and/or positive cultures. PrVAP must meet the specific diagnostic requirements for culture growth based on the method in which the samples were obtained. The first two VAE tiers of VAE are intended as quality metrics, whereas the third tier is meant to be used for internal quality improvement only.

Despite the intentions of the improved surveillance and reporting structure of VAEs, there have been little data to support its effectiveness in improving diagnostic discrimination. In a retrospective analysis of over 1300 patients from 11 North American ICUs, Muscedere et al[178] found a 10.5% VAC rate and 4.9% IVAC rate, but an 11.2% VAP rate (based on traditional definitions). Further, interrater reliability was very low (VAP and VAC $\kappa = 0.18$ and VAP and IVAC $\kappa = 0.19$), and the VAC rates did not change with the increase in preventative measures. However, VAC and IVAC were both associated with a significantly higher morbidity and mortality than VAP. Additional studies have demonstrated that the new criteria are identifying a fundamentally different group of patients, in some cases failing to detect up to 75% of VAP.[179]

While the criteria for VAE were designed to be objective and reproducible by electronic medical record review, the clinical diagnostic criteria for VAP are more subjective. Diagnosing pneumonia in critically ill trauma patients is particularly difficult because physical examination is often limited in the obtunded or sedated patient. In addition, lung trauma, including contusions and lacerations, as well as hemopneumothorax particularly complicate the interpretation of chest x-rays in trauma patients. Bacterial colonization of the endotracheal tube and trachea is universal after a few days of mechanical ventilation, so distinguishing between this and a clinically significant lung infection becomes challenging as well. The clinical pulmonary infection score (CPIS) initially described by Pugin to compare invasive and noninvasive pulmonary sampling techniques for VAP has been used to assist in the diagnosis of VAP, but subsequent trials have shown that CPIS alone is not sufficiently accurate to diagnose or rule out VAP.[180,181] It is difficult to make the diagnosis, as a gold standard really does not exist.

Diagnostic sampling of the respiratory tract can assist in differentiating tracheal colonization from true infection and in guiding antibiotic therapy. Three modalities of diagnostic sampling of the lower respiratory tree—BAL, bronchoscopic protected specimen brushing, and blind mini-BAL—are currently employed, and the specimens are sent for quantitative or semiquantitative microbiologic culture. A positive confirmatory culture is defined as a BAL or mini-BAL culture greater than 10^4 CFU/mL or protected specimen brush greater than 10^3 CFU/mL. Some controversy remains as to the optimal mode for sampling, and the quantitative criteria differ depending on the diagnostic method of diagnosis. To make matters more confusing, in 2016, the American Thoracic Society guidelines advise that noninvasive sampling with semiquantitative cultures should be used to diagnose VAP, rather than invasive (BAL or mini-BAL) sampling with quantitative cultures or noninvasive sampling with quantitative cultures.[182]

The impact of all of these diagnostic criteria changes on VAP rates and outcomes are not yet clear. However, like all other infections in the critically ill, when pneumonia is identified, it is important to institute therapy as soon as possible. Current recommendations for antimicrobial coverage for pneumonia, and especially VAP, emphasize beginning with broad-spectrum coverage and then de-escalating or narrowing the coverage once culture results are obtained. Inadequate initial antibiotic therapy has repeatedly been shown to be associated with worse outcomes and increased mortality.[183-185] Again, knowledge of local antibiograms should be used when choosing initial therapy. Patients with late HCAP or VAP are at risk of having multidrug-resistant bacteria including *Pseudomonas*, *Acinetobacter*, and extended-spectrum β-lactamase producers such as *Klebsiella* and *E coli*, especially if previously exposed to antibiotics. Gram-negative coverage should include antipseudomonal agents such as piperacillin/tazobactam, cefepime, imipenem, or meropenem. The addition of an aminoglycoside for "synergistic effect" against *Pseudomonas* is not supported by current guidelines. With the increasing prevalence of MRSA, vancomycin should always be included as initial empiric coverage. Once pneumonia has been diagnosed and therapy initiated, the next question is the appropriate duration of therapy. A study by Chastre and colleagues[186] reported that an 8-day antibiotic course was equivalent to 15-day therapy for VAP. The foremost consideration should be the clinical status of the patient, with a good response consisting of resolution of elevated temperature, resolving leukocytosis, decreased respiratory secretions, and improved oxygenation. Radiographic resolution often lags behind clinical improvement.

In the ICU, there are abundant data focusing on the utility of process measures designed to prevent VAP.[187] All

postoperative patients are at risk for pulmonary complications, and, therefore, aggressive mobilization and pulmonary toilet should be employed when possible. Unfortunately, injuries such as rib fractures that interfere with coughing and deep breathing, orthopedic injuries that limit mobility, and traumatic brain injuries resulting in an altered mental status may complicate therapies employed to combat altered mucociliary clearance and pulmonary physiology. Measures such as routine hand hygiene and elevation (30–45°) of the head of bed to decrease the likelihood of reflux have resulted in dramatic decreases in VAP rates as part of care bundles. Reverse Trendelenburg positioning is an acceptable alternative in patients who must remain supine due to spinal precautions or other restrictions. The duration of mechanical ventilation is the greatest risk factor for development of VAP, with an estimated risk of developing pneumonia of 1.2% to 3.5% per day of mechanical ventilation.[188] Every effort should be made to achieve liberation from the ventilator as soon as possible. Patients should be assessed daily for a trial of spontaneous breathing, and extubation should occur if the patient successfully passes the trial.[189-191]

Catheter-Associated Urinary Tract Infection

CAUTI is the most common hospital-acquired infection and is almost universally associated with an indwelling urinary catheter.[192-194] Most injured patients in the ICU require urinary drainage, and differentiation between urinary colonization (asymptomatic bacteriuria) and infection can be difficult. The diagnosis of CAUTI requires a quantitative urine culture with greater than 10^5 CFU/mL along with at least one of the following clinical criteria: (1) temperature greater than 38.5°C; (2) WBC greater than 10,000 or less than 3000; and (3) urinary urgency, dysuria, or suprapubic tenderness.[195] Diagnosis can be made with greater than 10^3 CFU/mL if there is also pyuria (>3 WBC per high-power field) or a positive leukocyte esterase. Clinically significant CAUTI usually occurs in the setting of urinary trauma and/or repair. Responsible organisms in hospitalized patients include *E coli*, *Pseudomonas*, *Proteus*, *Enterobacter*, *Serratia*, and *Citrobacter*.[196] Gram-negative coverage is indicated, but local resistance patterns should be taken into consideration when empiric antimicrobials are selected prior to confirmatory culture results. Fortunately, many systemic antibiotics are excreted via the kidney and will achieve urinary levels that far exceed the MICs for most of these pathogens.

Central Line–Associated Bloodstream Infection

CLABSIs have decreased in incidence in recent years, but they remain a source of serious morbidity, increased hospital and ICU stay, increased costs, and potential death.[197] The decision to insert a central line should not be taken lightly. Indications for central access include poor peripheral access, administration of total PN, and administration of high-dose vasoactive medications. Large-bore introducers are also commonly inserted to assist in the acute resuscitation of unstable patients. Often, the clinician is faced with the difficult decision to remove necessary central access or maintain the catheter in the face of potential line sepsis.

In trauma patients in whom catheters were placed emergently using nonsterile technique, the lines should be removed and replaced, if needed, with aseptically inserted catheter(s) at a new site(s). Femoral venous catheters are associated with unacceptably high rates of both infection and deep vein thrombosis, and catheters at these sites should always be removed as soon as possible. The use of peripherally inserted central catheters (PICCs) is associated with a lower incidence of CLABSI. Routine guidewire exchange at predefined intervals does not decrease the rate of CLABSI and may even increase the incidence. Using an evidence-based approach, Pronovost et al[198] reported a 66% sustained reduction in the incidence of CLABSI by removing unnecessary catheters, avoiding the femoral site, using full-barrier precautions during insertion, hand washing, and cleaning the skin with chlorhexidine. The data regarding routine antibiotic-impregnated catheter use is not clear; however, if the local CLABSI rate exceeds the NHSN surveillance data, then consideration should be given to using these types of catheters.[199]

The diagnosis of CLABSI is one of exclusion and requires the presence of bacteremia or fungemia in a patient in whom there is no alternate source of infection. Unless there are local signs of infection (ie, redness and purulence), a search for other infectious etiologies should always be performed first. Infected catheters are frequently thrombogenic, and the first sign of infection may be an inability to aspirate blood through the port. It is important to acknowledge that CLABSI is a surveillance definition instituted by the CDC for ease in identification and tracking of bacteremia, and there is a significant risk of overestimation of bacteremia in surgical patients in particular, due to the likelihood of bacterial translocation from the gut as a source.[200] The older clinical definition of catheter-related bloodstream infection (CRBSI) favored by infectious disease societies includes more specified criteria: (1) positive semiquantitative culture (>15 CFU/cm catheter segment); (2) quantitative (>10^3 CFU/cm catheter segment) culture with the same organism isolated from blood cultures; and (3) simultaneous quantitative blood cultures with greater than 3:1 central venous catheter to blood ratio of the same bacterial species.

Surgical Site Infections

Surgical site infections (SSIs) arise at the site of a previous surgical procedure, defined as a location in which an incision had been made or a procedure performed.[201,202] Classification of SSI is based on the anatomic depth of the infection and whether the infection is present in the wound or within an organ space. Superficial and deep incisional SSIs are differentiated based on whether the infection extends below the fascial layers (deep SSI). By convention, SSIs are infections identified within 30 days of the initial surgical procedure.

Superficial SSIs are not infrequent in trauma patients based on the wound classification, disruption of environmental barriers, bacterial inoculation at the time of injury, and dysfunction of host defenses seen with injury. Diagnosis of a superficial SSI is determined by the presence of at least one of the following clinical criteria at the site of a surgical procedure: (1) purulent drainage from the surgical incision; (2) culture of organisms from an aseptically obtained fluid or tissue sample from the incision; (3) clinical signs or symptoms of infection; or (4) clinical diagnosis of infection by the surgeon (eg, the need to open wound). Conditions such as stitch abscesses and erythema or serous drainage at external fixator pin sites do not constitute superficial SSI. In contrast to superficial SSI, a deep SSI involves the deeper soft tissues (eg, fascial and muscle layers) at the site of the surgical incision and at least one of the following: (1) purulent drainage deep to the fascia or muscle layers; (2) spontaneous fascial dehiscence; or (3) identification of a deep abscess on direct examination, during reoperation, or on radiologic examination. Finally, an organ space SSI involves anatomic structures (eg, organs or spaces) that were manipulated during the surgical procedure. In addition, diagnosis of an organ space SSI is based on at least one of the following: (1) purulent drainage from a drain placed into the organ or space (either incisional or percutaneously drained); (2) culture-positive fluid or tissue; or (3) the presence of an abscess during reoperation or radiologic evaluation.

Treatment of SSI depends on the location and depth of infection.[203,204] Superficial SSIs are treated by opening of the surgical incision followed by local wound care. There is no demonstrated benefit to systemic antibiotic treatment for superficial SSIs in the absence of systemic symptoms. For deep SSIs and organ space infections, it is advisable to try to obtain cultures of any purulent drainage, since prior antibiotic selection pressure and the increasing incidence of resistant strains within hospitals make it difficult to predict antimicrobial responses.[105] In the face of negative cultures, clinicians should base antibiotic choices on the anticipated pathogens at the anatomic site. It is well to keep in mind that infections that arise after treatment with a longer course of antibiotics will likely be resistant to, or perhaps only partially sensitive to, the prior agent used. Thus, while awaiting culture and sensitivity results, it is wise to employ a different antibiotic agent from a different class of antibiotics.

Clostridium difficile Infection

C difficile is a gram-positive anaerobic bacterium that is a frequent cause of infectious colitis.[205] It is a part of the normal colonic flora in 2% to 5% of the healthy population and is normally nonpathogenic. The incidence in the trauma population has been reported to be 3%.[206] Disruption of the colonic microbiota by antibiotics causes relative overgrowth of this organism. Although the occurrence has been reported with all antibiotics, the association is highest with clindamycin and third-generation cephalosporins. The organism reproduces via spore formation and should be considered highly contagious, as the spores are heat and stomach acid resistant. Additionally, alcohol-based hand sanitizers are ineffective in eradicating the spores. Soap-and-water hand washing is mandatory after contact with a contagious patient. Since 2001, there has been a significant increase in the incidence of CDI to approximately 84 per 100,000, and this has coincided with an increased number of serious or fatal infections.

Increasing failure rates with metronidazole therapy have led to the issuance of new guidelines that no longer recommend metronidazole as first-line therapy in adults.[207] Oral vancomycin (125 mg four times a day) or fidaxomicin (200 mg twice daily) should be administered for 10 days in both nonsevere and severe CDI. This new recommendation was made based on evidence that use of vancomycin or fidaxomicin provides patients with the highest likelihood of sustained symptom resolution 1 month after treatment. In patients with nonsevere cases of CDI, metronidazole may still be administered when patients are unable to obtain or be treated with vancomycin or fidaxomicin. In patients with severe, complicated CDI with shock, hypotension, ileus, or megacolon, vancomycin 500 mg four times a day is recommended in combination with parenteral metronidazole. Patients with ileus can be administered vancomycin via retention enema.

Surgical therapy for CDIs was historically limited to performing a subtotal colectomy in patients who developed severe complicated infection unresponsive to medical therapy. The result was often associated with a high mortality. More recently, there has been considerable interest in a different surgical approach using laparoscopic placement of a loop ileostomy for antegrade flushing of the colon. Intraoperatively, the colon is lavaged with warm polyethylene glycol, and postoperatively, vancomycin is instilled via the ileostomy antegrade through the colon. In a small trial, the mortality of this approach was 19% versus 50% in historic controls of subtotal colectomy. Furthermore, in the loop ileostomy group, 83% of the operations were done laparoscopically, and there was a greater than 90% salvage rate of the colon.[208] There are still no clear indications for determining which patients should undergo this type of surgical intervention; however, contraindications include colonic necrosis or perforation as well as the presence of abdominal compartment syndrome.[205]

Another therapeutic modality for recurrent CDI that is gaining popularity is fecal microbiota transplantation (FMT) to repopulate the normal colonic flora. While the first report of administration of a fecal enema for severe sepsis associated with CDI was in 1958,[209] the first prospective clinical trial of FMT via nasoenteric feeding tube did not occur until 2013.[210] The investigators in this trial compared three groups as follows: (1) vancomycin followed by colonic lavage and then FMT delivered via a nasoduodenal tube; (2) vancomycin followed by colonic lavage; and (3) vancomycin alone. The trial was stopped early because almost all of the patients in the control groups had CDI recurrence, whereas the success rate in the FMT group was 81%. Although FMT is still considered an investigational therapy by the US Food and Drug Administration, current guidelines strongly recommend FMT as therapy for multiply recurrent CDI (at least

two prior episodes).[207] The dramatic growth in microbiome science and supportive laboratories for the screening, processing, and banking of stool specimens and the increasing acceptance of oral encapsulation of processed donor fecal material suggest that this therapy will only continue to expand in indication.

LATE INFECTIOUS COMPLICATIONS

Overwhelming Postsplenectomy Infection

Historically, the spleen was considered expendable and the prevailing opinion was that it could be removed with relative impunity. Our current understanding is that the spleen plays an important role in the production of immune mediators that aid in the clearance of bacteria and viruses. Splenic mediators (opsonins) coat circulating bacteria and viruses and convert them into immune complexes, facilitating clearance.[211,212] *Streptococcus pneumoniae*, *Neisseria meningitidis*, *Haemophilus influenzae* type b, and influenza virus are important vaccination targets. It is difficult to estimate the current incidence of overwhelming postsplenectomy infection, as most of the published data on OPSI antedate the widespread availability of the pneumococcal and *H influenzae* vaccines. The time since splenectomy is an important risk factor because 50% to 70% of admissions to the hospital for serious infections occur within the first 2 years. A recent retrospective cohort study of 20,132 Swedish patients who underwent splenectomy for a variety of indications between 1979 and 2009 found an overall incidence of hospital admission for sepsis of 5.7% (95% CI, 5.6%–6.0%).[213] These investigators found that the incidence of sepsis and mortality was lower if splenectomy occurred secondary to trauma versus a hematologic malignancy. Interestingly, they did not observe a major impact following initiation of routine vaccination, except in the subjects undergoing splenectomy for hematologic conditions. In a more recent series from 173 ICUs across Germany, pneumococcal infections accounted for the majority of cases likely due to the lower overall vaccination rate than in the Swedish study.[214]

Postsplenectomy Vaccinations

The optimal timing of administration of vaccines after traumatic splenectomy is unknown, but data suggest an increasing trend for elevated functional antibody activity with a delay in vaccination and improved immune antibody response to vaccination at 14 days after surgery.[215] Most trauma patients will be discharged from the hospital within this interval; given that the population of trauma patients is characterized by poor follow-up, it is generally advised to vaccinate the patient prior to discharge (Table 21-14). ACIP recommends the use of the 23-valent polysaccharide pneumococcal vaccine for persons age 2 to 64 years who have functional or anatomic asplenia. The CDC suggests a single booster dose for those older than 2 years of age who are at high risk for serious pneumococcal infection and those most likely to have a rapid decline in antibody titers. A single revaccination should be given at least 5 years after the first dose, with further dosing not recommended routinely. *H influenzae* vaccination with ActHIB conjugate vaccine is recommended. There currently is no recommendation to revaccinate for *H influenzae* type b (Table 21-14). There are two types of meningococcal vaccinations recommended for patients with impaired splenic function in the United States: a quadrivalent meningococcal conjugate vaccine that protects against meningococcal

TABLE 21-14: Recommendations for Postsplenectomy Vaccinations in Adults Undergoing Emergent Splenectomy*

Vaccine	Primary series	Revaccination (booster)
Pneumococcal vaccine • PCV13 (Prevnar) plus PPSV23 (Pneumovax)	1 dose of PCV13 followed by 1 dose of PPSV23 ≥8 weeks later¶	PPSV23 every 5 years[Δ◊]
***Haemophilus influenzae* type b vaccine** • Hib	1 dose§	Not applicable
Meningococcal serotype ACWY vaccine • MenACWY (Menactra or Menveo)	2 doses at least 8 weeks apart¥	Every 5 years
Meningococcal serotype B • MenB-FHbp (Trumemba) or MenB-4C (Bexsero)	2 doses of MenB-4C at least 1 month apart or 3 doses of MenB-FHbp at 0, 1 to 2, and 6 months‡	Not applicable
Seasonal influenza virus	1 dose annually at the start of influenza season	Repeat annually at start of influenza season

*Available vaccine formulations and recommendations may differ outside of the United States.
¶If primary series already received (eg, due to concurrent immunocompromising condition or age >65 years), revaccinate with PPSV23 every five years. If only a single vaccine in the series has been received, refer to the UpToDate topic on prevention of sepsis in patients with impaired splenic function.
ΔSome experts prefer to give the second dose of PPSV23 three years after the first dose of PPSV in patients with sickle cell disease.
◊This recommendation differs from the Advisory Committee on Immunization Practices, which recommends a single revaccination dose of PPSV23 five years after the first PPSV23 dose.
§Hib vaccination is recommended for all adults who have not been previously vaccinated or if vaccination status is unknown.
¥If primary series already received, continue revaccinating every five years.
‡If not already received.

Source: Reproduced with permission from Pasternack MS. Prevention of infection in patients with impaired splenic function. In: UpToDate, Post TW, ed. UpToDate, Waltham, MA. Last updated September 2019. Copyright © 2019 UpToDate, Inc. For more information visit www.uptodate.com. Retrieved from https://www.uptodate.com/contents/prevention-of-infection-in-patients-with-impaired-splenic-function?search=infection%20after%20splenectomy&topicRef=1404&source=see_link. Accessed February 3, 2020.

serotypes A, C, W, and Y (MenACWY [Menactra or Menveo]) and a univalent serogroup B vaccine (MenB-4C [Bexsero] or MenB-FHbp [Trumenba]). Both vaccines are given as a primary series. Only the quadrivalent vaccines require revaccination.

Surgical Site Infection After Discharge

In the modern health care environment, there is increased pressure for earlier hospital discharge. However, it must be recognized that with shorter hospital stays, the relative rates of postdischarge SSI have increased substantially, and the ability to detect postdischarge SSIs is limited by resources available to prospectively collect these data.[216] In a prospective study of 268 trauma patients, 33% of all patients with SSI were diagnosed after hospital discharge.[217] Trauma caregivers should educate patients about signs and symptoms of SSI at the time of discharge and request that they be notified if infections are identified. However, even with these systematic measures in place, patients describe feeling unprepared for the challenges of self-monitoring postoperative wounds and tend to overtriage.[218,219] Telehealth platforms, and mobile health apps in particular, provide an innovative solution for ongoing education and communication with patients at their most vulnerable time; evidence is growing to support the feasibility of user adoption and substitution of virtual care for in-person follow-up visits, even for wound assessment.[220-222] Smart-enabled devices for vital sign, wound temperature, and activity monitoring are increasingly available and used by consumers, but widespread integration of patient-centered mobile health apps within health system–based telemedicine platforms awaits reimbursement for such encounters and better interoperability for documentation and reporting.

KEY SUMMARY POINTS

1. Trauma patients are susceptible to infection because of their unique exposure to tissue trauma and common need for surgical interventions with breach of host defenses and subsequent inflammatory response.
2. Trauma surgeons require training in infection control and prevention including knowledge of vaccinations and antimicrobial prophylaxis, as well as management of relatively rare infectious disease exposures in the context of disaster management and critical care support.
3. Comprehensive care of patients who sustain traumatic injury requires a commitment to health care–associated infection triage and prompt treatment of infectious complications to ensure optimal outcomes.

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22

Brain

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KEY POINTS

- Of the approximately 1.4 million individuals who are evaluated for traumatic brain injuries in emergency departments in the United States each year, 1.1 million are treated and released, 240,000 are hospitalized, and 50,000 die.
- In patients with gunshot wounds to the head, the mortality rate is greater than 50% among those who are alive upon presentation to a hospital.
- When calculating a Glasgow Coma Scale score, confounding factors include alcohol and other drugs, hypotension, hypoxia, sepsis, hypothermia, and other systemic factors.
- Magnetic resonance imaging (MRI) scans provide higher resolution images of the brain, spinal cord, and other soft tissues than computed tomography scans.
- In patients with diffuse axonal injury, T2-weighted MRI scans may show multifocal hyperintense lesions at gray matter/white matter interfaces.
- As many as 90% of patients with a concussion do not exhibit loss of consciousness; however, when this does occur, it is usually brief.
- The modified Monro-Kellie hypothesis states that an increase in the volume of one component inside the skull (brain, intravascular blood, cerebrospinal fluid) or addition of a new component (hemorrhage, tumor) mandates a compensatory decrease in other components to maintain constant intracranial pressure.
- Relative indications for surgical elevation of a depressed skull fracture include depression of greater than 8 to 10 mm or greater than the thickness of the adjacent skull, a focal neurologic deficit due to compression of underlying brain, significant inward intrusion of bone fragments, and persistence of cosmetic deformity after overlying scalp swelling has subsided.
- An acute subdural hematoma with a thickness of greater than 1 cm or a midline shift of greater than 5 mm should usually be evacuated regardless of Glasgow Coma Scale score.
- When intracranial hypertension cannot be controlled by routine measures, hypothermia, barbiturate coma, and unilateral hemispheric decompressive craniectomy should be considered.

INTRODUCTION

Traumatic brain injury (TBI) remains frustratingly resistant to treatment. Several decades ago, progress in resuscitation, hemostasis, imaging, noninvasive management, critical care, rehabilitation, and emergency medical services organization brought about a decrease in mortality rates, but the failure to develop interventions targeted specifically to the injured brain has inhibited further improvements in outcome. New and developing insights into our classification and approach to TBI may soon pave the way for meaningful advances. Until then, clinicians who care for these patients must rely upon therapies that still await validation in appropriately constructed clinical trials.

TBI has been defined in many different ways. A good working definition is that it is a disruption or alteration of brain structure or function caused by external mechanical forces. The disruption may be variable in severity and may be transient or permanent. The causative external forces are diverse and include rapid acceleration or deceleration, direct compression, penetration and physical disruption of brain tissue, blast and other complex mechanisms, and various combinations of these and other etiologies. Mild TBI may be present despite absence of abnormalities on imaging studies. At the other end of the spectrum, severe injuries may be associated with large contusions, traumatic hematomas, or other immediately life-threatening structural lesions.

EPIDEMIOLOGY

The exact number of people who sustain TBI is unknown since some patients with severe systemic injuries do not survive long enough to undergo a thorough evaluation that would uncover the presence of brain injury. At the other end of the spectrum, many individuals suffering mild or moderate TBI choose not to seek medical care.

Many epidemiologic studies include only TBI patients who receive medical care in an emergency department. By this criterion, approximately 1.4 million people per year suffer TBI in the United States. Approximately 1.1 million are treated and released, 240,000 are hospitalized, and 50,000 die.[1]

Common causes of TBI include falls, motor vehicle collisions (MVCs), pedestrian impact, and assault. TBI has a bimodal age distribution, with the greatest risk in 0- to 4-year-olds and 15- to 19-year-olds. Falls predominate at the extremes of age, whereas MVCs are most common in teenagers and young adults. The incidence in males is 1.5-fold higher than in females. The youngest patients are often victims of abuse. Military personnel compose a statistically small number of the overall volume of TBI patients, but combat operations cause them to have a higher incidence of penetrating and blast injuries.

TYPES OF INJURY

Classification and Management

PRIMARY VERSUS SECONDARY INJURY

TBI is a dynamic process. Knowledge of the natural history of the various types of TBI is essential for optimal care because management priorities for a given patient may change rapidly as his or her underlying pathophysiology changes.

Primary injuries result directly from the forces imparted at the time of the accident, or primary insult. These may include both focal disruption of tissue, such as contusions and hematomas, and diffusely distributed damage.

Secondary insults are defined as those occurring subsequent to the initial impact. These injuries may include hypoxemia, ischemia, seizures, fever, hypoglycemia, and other systemic (nonneurologic) events that can directly impact the brain's ability to respond to a primary insult. Thus, secondary insults may lead to secondary injuries. A brief period of mild hypotension or hypoxia may be tolerated quite well by an uninjured brain, but the same minimal secondary insult can be devastating if it occurs soon after a brain injury. A neurosurgeon may spend several hours operating on an acutely injured patient, but the biggest determinant of outcome may be the management that the patient receives in the intensive care unit (ICU) in the days and weeks after admission.

CLOSED VERSUS PENETRATING INJURY

Closed injuries are those in which the overlying scalp remains intact.

Penetrating Nonmissile Injury. Penetrating injuries to the central nervous system are generally considered to be of low velocity when caused by such objects as knives, arrows, lawn darts, or ice picks. High-velocity injuries are caused by missile-type projectiles. Physical examination should describe any visible wounds, but labeling these as "entry" or "exit" wounds is best avoided until all information is available, including results of detailed imaging studies. Clipping or shaving hair may be required for a complete assessment. Control of vigorous scalp hemorrhage may require suturing or tight wrapping of scalp wounds, which can bleed profusely.

Assessment and resuscitation proceed as in other types of TBI. Computed tomography (CT) scanning should be performed to localize any intracranial projectiles or indriven bone fragments and to characterize the extent of injury. If a foreign body has passed near the location of any major vessels, catheter angiography should be considered once more urgent evaluation and stabilization have been performed. Antibiotics and prophylactic anticonvulsants are usually administered.

If an object is still embedded and protruding from the skull, great care should be taken to stabilize it during transport and evaluation. Evaluation, planning, and operative setup should be undertaken with the foreign body still in place, and removal should proceed only in the operating room (Fig. 22-1). To help plan the removal, a similar or identical object may be useful to have on hand as a reference.

Penetrating Missile Injury. Gunshot wounds to the head (GSWHs) make up the majority of penetrating cranial injuries. In some parts of the United States, GSWHs are the most

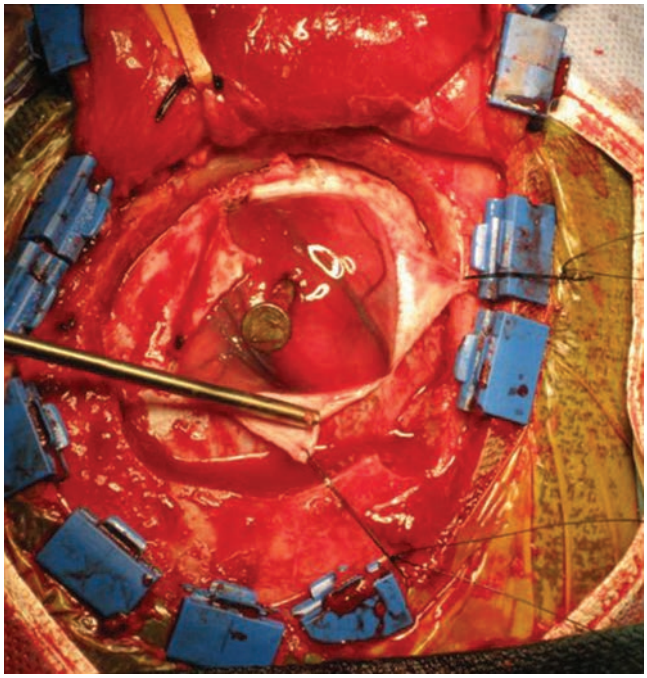

FIGURE 22-1 Intraoperative photograph of a penetrating intracerebral nail gun injury. The bone flap has been removed and the dura has been opened, showing the nail entering the brain parenchyma.

common form of TBI. Some studies report an overall mortality rate of greater than 90% if on-scene deaths are included, with a mortality rate exceeding 50% among those who are alive upon presentation to a hospital.

Traditionally, GSWHs have been divided into those from high-muzzle-velocity rounds (approximately 750–1000 m/s, such as hunting rifles and military weapons) and those from low-muzzle-velocity rounds (approximately 200–500 m/s, as seen in most handguns). With escalating civilian firepower, however, these distinctions are becoming blurred.

Primary injuries from gunshot wounds include direct injuries to face and scalp, pressure waves of gaseous combustion from the weapon if it is touching the target or fired at close range, coup and contrecoup injuries from missile impact, and destruction of brain or bone along the primary path of the original bullet or secondary paths created by fragments of bullets and bone. High-velocity projectiles also create secondary cavitation that pushes tissue away from the bullet in a cone of injury many times wider than the projectile itself. The vacuum that follows the cavitation may pull surface debris into the wound, potentially creating a nidus for infection. Some specially designed bullets mushroom, fragment, or tumble in order to increase the width of the destructive path. Projectiles can also ricochet off the inner table of the skull.

Secondary injuries from GSWHs mimic those seen in other types of TBI and include edema, growth of contusions, disseminated intravascular coagulation (DIC), hemorrhage from vessel disruption, ischemia, infarction, and herniation. Late complications can include abscess or traumatic aneurysm formation, seizures, and migration of fragments of bone, bullet, or other debris.

The decision about whether to take a GSWH patient to the operating room may be difficult since many of these patients have devastating and irrecoverable injuries that will not benefit from surgery. The Glasgow Coma Scale (GCS) score is the most useful piece of information for triaging a GSWH patient.[2] Patients with GCS scores of 3 to 4 generally have a poor outcome regardless of CT findings, and for that reason some clinicians do not even obtain CT scans on these patients. Limited treatment is a reasonable course in such cases. Those with a GCS score of 5 are in an indeterminate category. If the GCS score is 6 or higher, most experts would opt for aggressive treatment in the absence of additional significant findings to suggest that a less aggressive course would be preferable. Poor prognostic factors include self-infliction of injuries, the presence of bilaterally fixed and dilated pupils, and the development of coagulopathy. Poor prognosis and high mortality have also been associated with bullet passage across the midline, through the geographic center of the brain, through the ventricles, or across more than one lobe of the brain.[3]

Basic principles of surgery for GSWH include evacuation of hematomas causing mass effect, meticulous hemostasis, thorough debridement of devitalized tissue and foreign debris, and watertight layered closure to prevent cerebrospinal fluid (CSF) leaks. A "chain of evidence" for forensic purpose should be preserved when bullet fragments are removed.

Diffuse Injury

DIFFUSE AXONAL INJURY

Diffuse axonal injury (DAI) refers to axonal damage that is caused largely by rotational and other mechanical forces. Mild cases result in axonal stretching and transient neuronal dysfunction. More severe cases may set into motion a complex series of cellular events that cause focal impairment of axoplasmic transport, culminating in axonal disconnection at the site of impairment. In humans, this sequence of events may occur over the course of roughly 6 to 12 hours, suggesting that a yet-to-be-developed therapeutic intervention might be administered within this time frame. More severe traumatic forces may cause direct mechanical disruption of tissue and immediate disconnection of axons. Subsequent axonal sprouting and attempts to reestablish connectivity may result in the creation of aberrant neural pathways, potentially contributing to the morbidity of severe brain injury. The development of DAI does not require direct impact to the head. It may result from rapid noncontact acceleration or deceleration in a linear or rotational fashion.[4]

Histologically, DAI-associated lesions are usually microscopic in size and nonhemorrhagic in composition. CT scans may be normal or may show hyperdense petechial hemorrhages. T2-weighted magnetic resonance imaging (MRI) scans may show multifocal hyperintense lesions at gray matter/white matter interfaces, especially in the frontal lobes, corpus callosum, and brainstem. When hemorrhagic DAI is visible on CT or MRI scanning, one must assume that the radiologically visible lesions are just the tip of the iceberg and that more widespread DAI exists.

Injury severity parallels the amount of force required to cause DAI. Prognosis worsens with increasing number of lesions or as lesion depth progresses from the cortex to corpus callosum to brainstem.

BLAST INJURY

Military personnel are at much higher risk than civilians for blast TBI (bTBI). Brain injury accounts for approximately 20% of all combat-related injuries in modern wars,[5,6] including Operation Enduring Freedom in Afghanistan and Operation Iraqi Freedom. In these two conflicts, bTBI was frequently caused by improvised explosive devices (IEDs). Modern helmets, body armor, rapid transport of injured personnel, and forward-based field hospitals have brought about unprecedented rates of warfighter survival and have allowed for a better understanding of the effects of bTBI.

bTBI may be composed of four distinct types of injuries that often overlap. Primary blast injury occurs from overpressure. This has long been known to contribute to injuries in air-filled organs, such as the lungs, bowel, and middle ear. The potential contribution of overpressure to brain injury is currently the focus of much study. Secondary blast injury results from penetration of objects that are set into motion by the explosion. Tertiary blast injury results from a patient being thrown and striking the ground or other object. Quaternary

blast injury refers to additional factors not included earlier, such as heat, toxic fumes, and inhalational injury, or hypoxia. Blast injuries in an enclosed space produce an enhanced and complex pattern of forces as energy reflects off walls and objects to impact the head and body at multiple angles and to multiple degrees.

bTBI often includes closed and penetrating TBI components, and many of these patients also have additional serious injuries, such as traumatic limb amputations or hemorrhagic shock. This combination of factors makes it difficult to assess the true contribution of primary or quaternary blast effects on patients who present with TBI after an explosion. In milder cases, combatants may not recognize that they suffer delayed effects of bTBI, or they may knowingly hide their deficits in hopes of remaining with their combat unit.

bTBI can cause headache, confusion, amnesia, altered mental status, and other symptoms associated with concussions. Postconcussive symptoms may be difficult to differentiate from posttraumatic stress disorder, which often coexists with TBI.

Severe bTBI may cause hyperemia and severe cerebral edema early after injury, often requiring decompressive craniotomy. Higher rates of traumatic pseudoaneurysm formation and vasospasm are seen in comparison to civilian closed and penetrating TBI. These vascular pathologies may require more frequent endovascular or open vascular repair.[7]

CONCUSSION

Concussion is the mildest form of TBI. It may be defined as a usually transient alteration of neurologic function caused by nonpenetrating injury to the brain and characterized by normal imaging studies. Classic symptoms of concussion include headache, irritability, confusion, amnesia, nausea, vomiting, memory problems, difficulty concentrating, vertigo, alterations of balance, anorexia, insomnia, hypersomnolence, impaired coordination, anxiety, and depression. As many as 90% of concussed patients exhibit no loss of consciousness, and when this does occur, it is usually brief. Grading the severity of concussion is currently not recommended because the grading systems that have been described to date have not been shown to correlate with outcome, treatment recommendations, or duration of symptoms.

By definition, CT scanning is unremarkable in these patients. MRI may reveal abnormalities in up to 25% of cases in which CT scans are normal.[8] Pathologic specimens generally show no gross or microscopic parenchymal abnormalities in patients who have suffered a single concussion.

Second impact syndrome is a rare but potentially catastrophic sequela of concussion. It is seen most commonly in children and teenagers. These patients are still recovering from the effects of a concussion when they suffer a second one. Within minutes, the patient develops cerebral vascular engorgement, possibly from impairment of cerebral autoregulation caused by the first concussion. Neurologic deterioration quickly follows, usually culminating in cerebral herniation and death.

Most important for the treatment of concussion is the recognition of injury. Signs of concussion might be so subtle that they do not prompt the patient or family to seek medical attention. However, returning while still symptomatic to an activity that carries a risk of another concussion, such as contact sports, can be disastrous. All experts agree that a symptomatic player should not return to play until he or she has fully recovered.

ABUSIVE HEAD TRAUMA

Nonaccidental trauma (NAT) is traumatic injury that is deliberately inflicted on infants and children. The concept was first described in infants as an injury triad consisting of long bone metaphyseal fractures, subdural hematomas (SDHs), and retinal hemorrhages[9] and has become known in common parlance as "whiplash shaken infant syndrome" or "shaken baby syndrome."[10] Despite the fact that it is almost certainly underreported, it is nevertheless recognized as the primary etiology of brain injury–associated death in children less than 2 years of age.

Some medical professionals find management of these cases to be difficult due to awkwardness of discussions with the child's parents, emotional attachment to the children, frequent lack of accurate information, rarity of admissions of guilt from perpetrators, and the medicolegal implications of child abuse accusations. However, a heightened level of suspicion must be maintained since missed recognition of NAT returns the child to a harmful environment, almost always results in continuation or escalation of the abuse, and may result in the patient's death.

The two most commonly provided histories are trivial blunt trauma, such as a short-height fall from bed or low surface, or complete denial of any history of trauma. Except for the rare epidural hematoma (EDH) with middle meningeal arterial bleeding, low-height falls (head-to-impact distance <3 ft) do not result in life-threatening brain injuries.[11,12] In the absence of a history of trauma, the existence of NAT may be indicated by feeding difficulty, emesis, lethargy, irritability, abnormal movements, seizures, unresponsiveness, or apnea.

On imaging studies, NAT may appear as multiple brain injuries that are more severe than expected given the reported history. Injuries may appear to vary in age (some recent, some remote). Impact injuries include skull fractures and superficial scalp lacerations or swelling in addition to injuries to the underlying brain. There is a high association with other organ injuries. Historically, some fracture patterns have been incorrectly considered suspicious for child abuse. Fractures that are multiple, compound, diastatic, midline, or nonparietal or that cross suture lines may denote a greater degree of imparted force, but they are not pathognomonic for NAT.

A shaking mechanism can result in diffusely distributed SDH. The most frequent hemorrhagic finding is a combination of convexity and interhemispheric SDH, often located posteriorly. Some experts believe interhemispheric SDH has the highest degree of specificity for abuse of any intracranial injury. SDH, subarachnoid hemorrhage (SAH), and retinal

hemorrhages are far more commonly seen in abused than nonabused children. EDHs can occur, but they are much more commonly associated with accidental injury. Retinal hemorrhages are seen in 65% to 95% of children with inflicted head injuries and may be unilateral or bilateral. However, severe bilateral retinal hemorrhages are occasionally seen in accidental trauma, usually after major application of force via a well-defined mechanism, such as MVC.

In the appropriate circumstances, practically any pattern of hemorrhage or fracture can result from either accidental or inflicted trauma. However, inflicted injury is the only known illness or condition associated with the combination of acute SDH, skeletal fractures, and severe bilateral retinal hemorrhages. The severity of injury is staggering, with 15% to 38% overall mortality and 60% mortality if the patient is comatose on presentation. Survivors face a 60% to 70% likelihood of significant neurologic handicap.

Focal Injuries and Their Management

TBI varies widely in both severity and subtype. During a single traumatic event, a patient may be subjected to multiple forces that differ in magnitude, direction, and duration. Although the following injuries are discussed individually, it must be remembered that multiple types of injury may, and often do, occur in the same patient. Furthermore, focal and diffuse injuries often coexist. For example, initial treatment efforts may focus on immediate evacuation of a patient's large SDH, but associated DAI may have an even greater influence on ultimate neurologic outcome.

SKULL FRACTURE

Skull fractures can be categorized by the state of the overlying scalp (closed or open), the number of bone fragments (simple or comminuted), the relationship of bone fragments to each other (depressed or nondepressed), the widening or entry of a fracture into an existing cranial suture (diastatic), and involvement of the cranial vault or skull base. In general, lower-force impacts like falls from standing will create fractures that tend to be linear, closed, and without dural laceration. Higher-force impacts like motor vehicle accidents, falls from heights, or penetrating trauma are more likely to produce comminuted, open fractures with a greater likelihood of underlying dural or cerebral injury. "Ping-pong" fractures are greenstick-type fractures usually seen in newborns due to the plasticity of the skull (Fig. 22-2). They show a local concavity of the skull, without sharp edges, and usually do not require intervention because the skull remodels significantly during growth, with gradual improvement or even resolution of the cosmetic deformity.[13]

Clinical signs suggestive of the presence of calvarial fractures include gross deformity and palpable step-offs. Basilar skull fractures may show postauricular or periorbital ecchymosis, hemotympanum or laceration of the external auditory canal, and CSF rhinorrhea or otorrhea. Cranial nerve (CN) injuries may be seen with fractures of the cribriform plate (CN I, anosmia), optic canal (CN II, visual deficit), and temporal bone (CN VII, facial weakness; or CN VIII, hearing loss). Severe basilar skull fractures may result in pituitary gland or stalk injury and resultant endocrinopathies. Direct injury to vasculature that penetrates the skull base may result in arterial dissection, traumatic aneurysm formation, or traumatic carotid-cavernous sinus fistula formation with resulting cranial neuropathies, chemosis, bruit, and stroke. Fractures of air sinuses or mastoid air cells may result in meningitis, even years after the initial event.

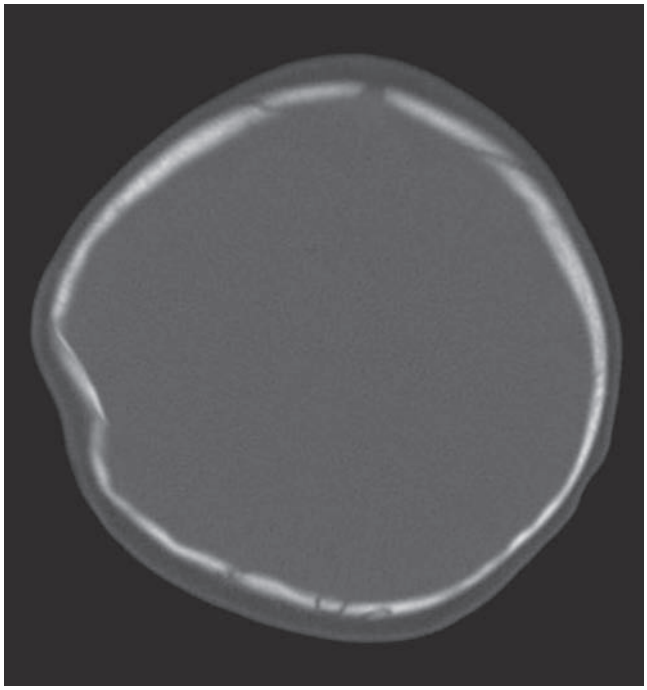

FIGURE 22-2 Computed tomography scan showing ping-pong skull fracture. The multiple nondisplaced linear lucencies are normal calvarial sutures.

Most skull fractures can be seen on CT scans. Plain films may be superior to CT for discovering linear calvarial fractures parallel to the skull base (in the plane of CT slice acquisition). They can be differentiated from vascular grooves or normal cranial sutures by the characteristics listed in Table 22-1.[14] CT scans provide better visualization of facial and orbital

TABLE 22-1: Differential Diagnosis of Fractures on Skull X-Rays

	Linear skull fracture	Vascular groove	Suture line
Density	Black	Gray	Gray
Course	Straight	Curving	Follows known suture course
Branches	Usually none	Often branching	Joins other suture lines
Width	Very thin	Thicker than fracture	Jagged, wide

fractures, temporal bone fractures, and pneumocephalus, as well as better detection of air-fluid levels and varying degrees of opacification that may indicate injury to air sinuses and mastoid air cells. Thin-cut bone windows can be reconstructed in coronal and sagittal planes and via three-dimensional surface modeling to aid in fracture characterization and surgical planning. CT angiograms and venograms are useful for assessing fractures involving skull base foramina containing important vasculature, such as the carotid canal and foramen magnum, or fractures that cross major venous sinuses.

Fractures must be assessed and treated in concert with management of any underlying brain injury. The following discussion of skull fracture treatment assumes that evaluation has been conducted and appropriate treatment instituted for SDHs or EDHs, parenchymal hemorrhages, contusions, and/or cerebral edema, and that clinical criteria do not mandate separate operative intervention for these lesions.

Closed, nondisplaced fractures do not normally require intervention. Open skull fractures may require debridement and careful exploration in addition to prophylactic antibiotics. Those with obvious underlying dural laceration or CSF leakage require layered surgical repair to reduce the risks of meningitis or brain herniation through a dural defect. In the pediatric population, laceration of the underlying dura may rarely lead to development of a growing skull fracture (or leptomeningeal cyst) in 0.05% to 0.6% of skull fractures.[15] Over time, pulsations of the underlying rapidly growing brain widen the dural laceration and fracture line (Fig. 22-3). These are most common in children under 1 year of age, and over 90% occur in children less than 3 years old. Surgical repair includes wide bony exposure to repair dural edges that often retract beyond the limits of the visible fracture.

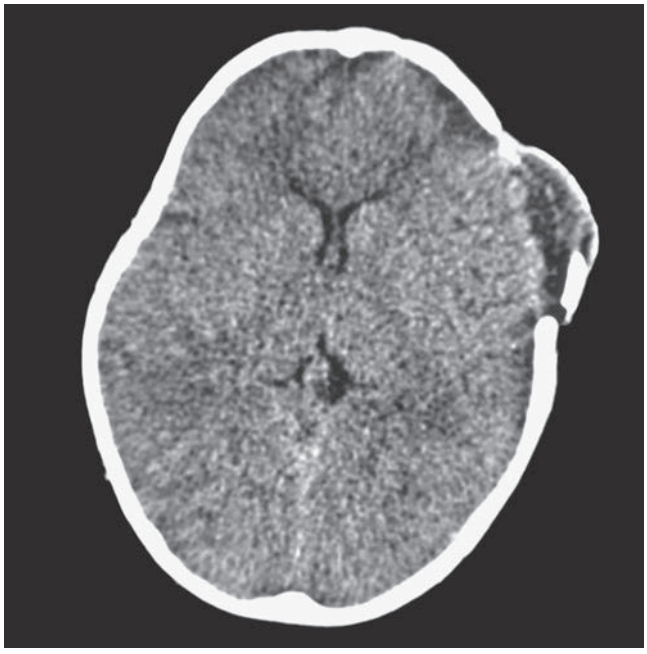

FIGURE 22-3 Computed tomography scan showing growing skull fracture from leptomeningeal cyst in child. Note displaced bone and expansion of cerebrospinal fluid–filled soft tissue.

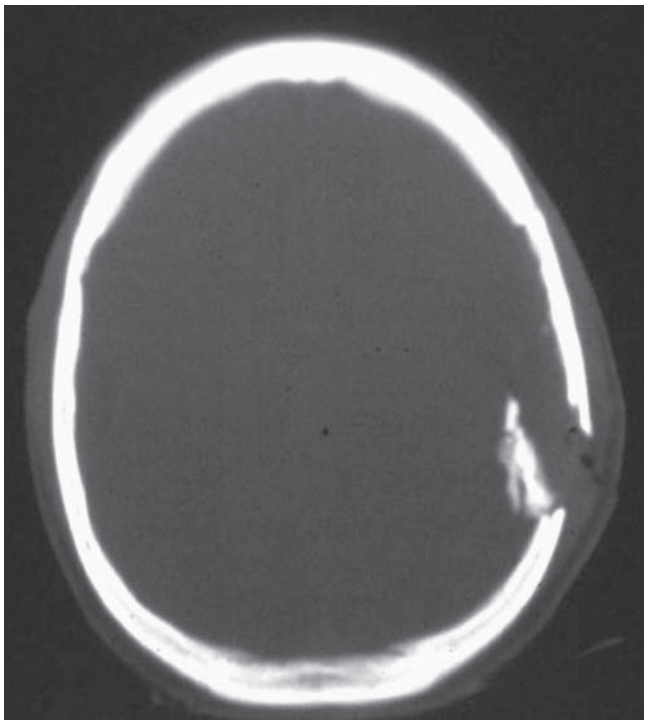

FIGURE 22-4 Computed tomography scan showing a depressed skull fracture that required surgical elevation and dural repair. The patient also had an underlying brain contusion and presented with a receptive aphasia.

Relative indications for surgical elevation of a depressed skull fracture include depression greater than 8 to 10 mm or greater than the thickness of the adjacent skull (Fig. 22-4), a focal neurologic deficit clearly attributable to compression of underlying brain, significant inward intrusion of bone fragments (implying possible dural laceration), and persistence of cosmetic deformity after overlying scalp swelling has subsided. For simple depressed fractures without dural violation, there is no evidence that posttraumatic seizure risk[16] or neurologic outcome[17] is improved by surgical elevation of the fracture. At least in very young patients, cosmesis may not be improved by surgery because, over time, the growth of the child's brain induces remodeling of the overlying skull.[17] Fractures that cross a major dural venous sinus may warrant a more conservative approach given the increased risks of bleeding and air embolism that may occur during surgical repair.

Current recommendations support surgical repair of open fractures depressed greater than the thickness of the cranium. Surgery is also supported in the presence of significant intracranial hematoma, depression greater than 1 cm, significant frontal sinus or nasofrontal duct involvement, gross cosmetic deformity, wound infection, pneumocephalus, or gross wound contamination. In the absence of gross contamination, primary bone fragments may be replaced without excessive risk of infection.[18] Nonoperative management may be preferred

for management of open depressed cranial fractures if there is no evidence of dural penetration.

EPIDURAL HEMATOMA

EDH occurs when blood collects in the potential space between the dura and inner table of the skull. Although classically described EDHs arise from arterial bleeding caused by a temporal bone fracture that lacerates the middle meningeal artery, the advent of CT scanning has revealed that many EDHs originate from bleeding of the bony edges of skull fractures. Another common cause is injury to a venous sinus.

On CT scanning, an EDH usually appears as a hyperdense, biconvex (lenticular) mass adjacent to the inner table of the skull (Fig. 22-5). Unless sutural diastasis is present, the EDH is often externally bounded by cranial sutures. Additional associated findings may include SDHs and cerebral contusions. A significant percentage of EDHs managed nonsurgically will demonstrate an increase in size, usually in the first few hours after injury. Some of these lesions may appear in a delayed manner after an initial CT scan failed to reveal their presence.

The classic clinical presentation of an EDH describes a brief posttraumatic loss of consciousness (LOC) followed by a lucid interval of varying duration, which progresses to obtundation, contralateral hemiparesis, and ipsilateral pupillary dilatation. In reality, however, this sequence of events occurs only rarely. LOC is seen in a small minority of cases. Overall mortality from a unilateral EDH has been reported as 5% to 12%,[19] with rates increasing in cases of bilateral EDHs, posterior fossa location (25% mortality), and concurrent acute SDH.

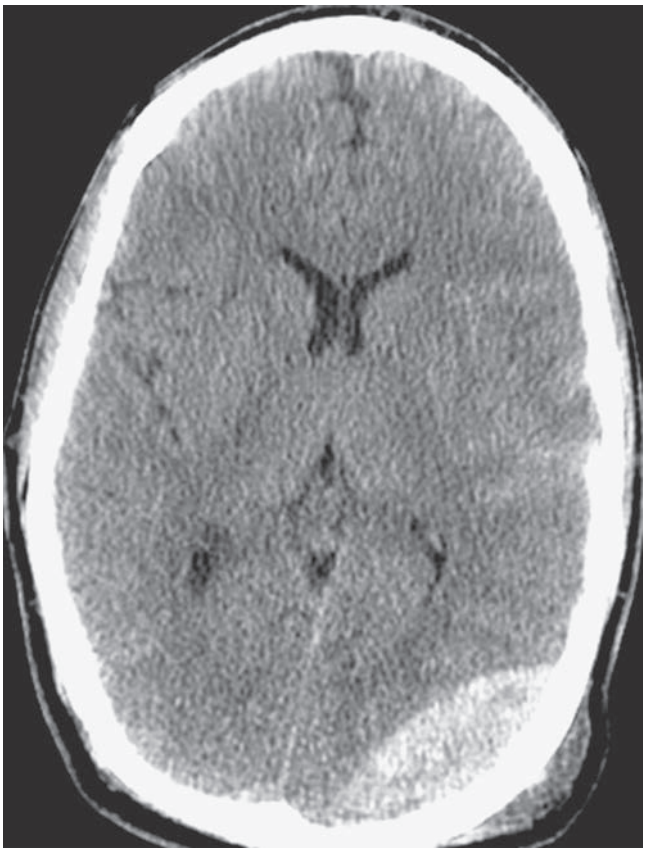

FIGURE 22-5 Computed tomography scan of acute epidural hematoma. Note the biconvex- or lenticular-shaped hemorrhage. This hematoma was subjacent to a diastatic left lambdoid suture.

Rapid diagnosis and timely intervention are essential for optimal outcome (see Atlas Figures 2 and 3). Surgical guidelines suggest that an EDH greater than 30 cm^3 in volume should be evacuated regardless of GCS score. An EDH less than 30 cm^3 in volume and less than 15 mm of thickness and less than 5-mm midline shift may be treated conservatively at a neurosurgical center with frequent neurologic examinations and serial CT scanning.[18] Relative indications for evacuation of EDHs include the presence of neurologic symptoms or maximal hematoma thickness greater than 1 cm. Acute EDH patients in a coma (GCS score ≤8) and with anisocoria should undergo surgical evacuation as soon as possible.[18]

Posterior fossa injury is rare, composing less than 3% of head injuries. However, a disproportionately large proportion of these lesions are EDHs. The limited volume of the posterior fossa and the potential compromise of the brainstem and CSF pathways underscore the importance of rapid evacuation via suboccipital craniectomy.[18] Patients without signs of mass effect or neurologic deterioration may be watched conservatively, with serial neurologic examinations, CT scans, and a low threshold for surgical intervention.

SUBDURAL HEMATOMA

SDH occurs when blood collects between the arachnoid and inner dural layers of the meninges. Thus, from a strict anatomic point of view, these are really intradural hematomas. A common cause is traumatic stretching and tearing of cortical bridging veins that cross the subdural space and drain into the dura or into a dural sinus. The force may be applied by direct impact or by indirect linear or rotational motion. Less common etiologies include coagulopathy, subdural dissection of parenchymal hematomas, and rupture of a vascular anomaly into the subdural space. Patients with cerebral atrophy, cranial CSF shunts, and large middle fossa arachnoid cysts are predisposed to SDH because of increased traction on cortical veins. Many patients with acute SDHs also have other significant intracranial lesions.

SDHs are commonly located over the cerebral convexities and may cover part or all of a hemisphere. Classically, they are crescent shaped, may cross calvarial suture lines, and layer along the falx or tentorium (Fig. 22-6). Patients may present with symptoms of mass effect or of underlying diffuse brain injury. Chronic SDH may present more insidiously, with progressively worsening headaches and/or focal deficits.

The appearance of SDHs evolves over time. On CT scanning, acute SDHs are usually hyperdense, but some may show areas of mixed density if ongoing active bleeding causes

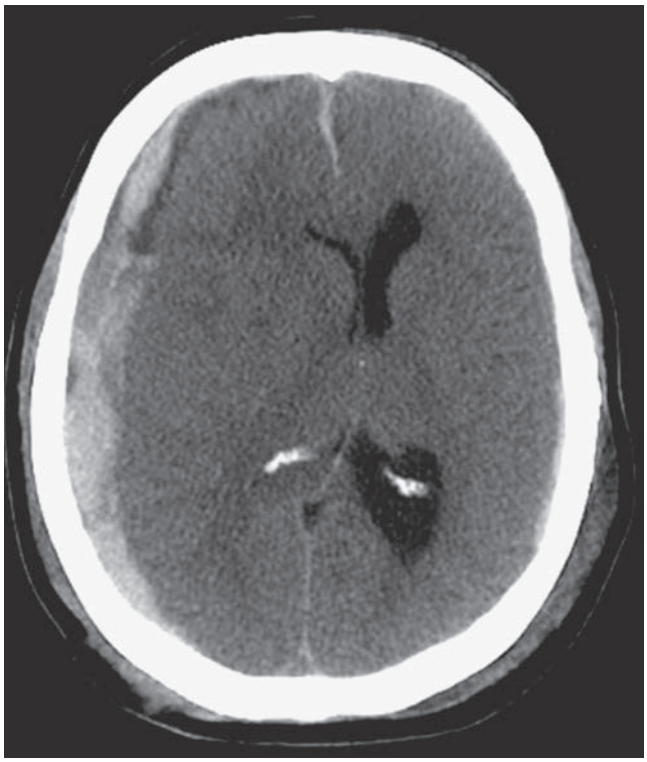

FIGURE 22-6 Computed tomography scan of acute subdural hematoma. Note that the crescentic hemorrhage crosses under the right coronal suture.

the not-yet-clotted blood to continue to accumulate in the subdural space. Some acute SDHs may be isodense in the presence of coagulopathy, significant anemia, or admixing of blood and CSF. Subacute SDHs are isodense to brain, and chronic SDHs become hypodense. Inward displacement of the gray/white cortical ribbon and cortical vessels may be evident on contrast-enhanced CT scanning. The subdural membranes that often exist with chronic SDHs may appear within 4 days and enhance with contrast administration.[20] MRI may show evolving signal intensities that vary with the age of the SDH as blood breakdown products are converted to oxyhemoglobin, deoxyhemoglobin, methemoglobin, and hemosiderin (Table 22-2).

SDHs often have worse outcomes than EDHs of similar size. EDHs are frequently of arterial origin and thus present with symptoms quickly, facilitating prompt diagnosis and treatment. In contrast to EDHs, it has been postulated that many SDHs result from higher-magnitude forces, which cause greater damage to the brain and cortical vessels. Because of the venous origins of the bleeding, an SDH may require more time to grow and become symptomatic. The attribution of symptoms to diffuse brain injury has also been postulated to delay diagnosis in some cases until the signs of midline shift and brainstem compression become evident. Mortality associated with SDH can be high and may be related more to the underlying brain injury than to the SDH itself. Mortality rates increase in the elderly and in patients on anticoagulants.[21]

TABLE 22-2: Appearance of Subdural Hematoma on Computed Tomography (CT) and Magnetic Resonance Imaging (MRI)

	CT density	MRI T1	MRI T2
Hyperacute (minutes)	↓	—	↑
Acute (hours to days)	↑/swirl/iso if coagulopathy	—	↓
Early subacute (4–7 days)	—	↑	↓
Late subacute (1–3 weeks)	—	↑	↑
Chronic (3 weeks to 3 months)	↓	↓	↓

↑/—/↓, hyperdense/isodense/hypodense to brain (CT) or hyperintense/isointense/hypointense (MRI).

Guidelines propose that an acute SDH with thickness greater than 1 cm or a midline shift greater than 5 mm should be evacuated regardless of GCS score. Comatose patients (GCS score ≤8) with an acute SDH less than 1 cm in thickness and midline shift less than 5 mm should undergo SDH evacuation if the GCS decreases by 2 points between the time of injury and hospital admission, if the patients present with pupils that are asymmetric or fixed and dilated or if intracranial pressure (ICP) reaches or exceeds 20 mm Hg.[18] A craniotomy to evacuate an acute clot should be performed as soon as possible. Over time, as solid blood clots pass through subacute to chronic stages, they liquefy and may be amenable to drainage via burr holes.

INTRACEREBRAL HEMATOMA AND CONTUSION

Traumatic intracerebral hematomas (TICHs) and contusions may expand rapidly. Worsening perilesional edema and rapid hematoma growth may produce increasing mass effect and neurologic deterioration. If patients demonstrate neurologic decline referable to an expanding TICH, surgical intervention may be warranted. Surgical decompression is often necessary in patients when TICH volume exceeds 50 cm^3 or when patients present with GCS scores of 6 to 8 with frontal or temporal contusions greater than 20 cm^3 in volume with midline shift of 5 mm or greater and/or cisternal compression on CT scan.[18] Surgical procedures range from localized frontal or temporal craniotomy with resection of underlying focal clot to more extensive craniectomy with duraplasty, evacuation of severely contused brain, and temporal lobectomy.

SUBARACHNOID HEMORRHAGE

Subarachnoid blood occupies the space between the pial and arachnoid membranes. Traumatic SAH (tSAH) results from venous tears in the subarachnoid space. The injured vessels are often quite small, and the resulting hemorrhage frequently appears as a thin, diffuse hyperdensity over the sulci and gyri of the cerebral convexity (Fig. 22-7) and as a

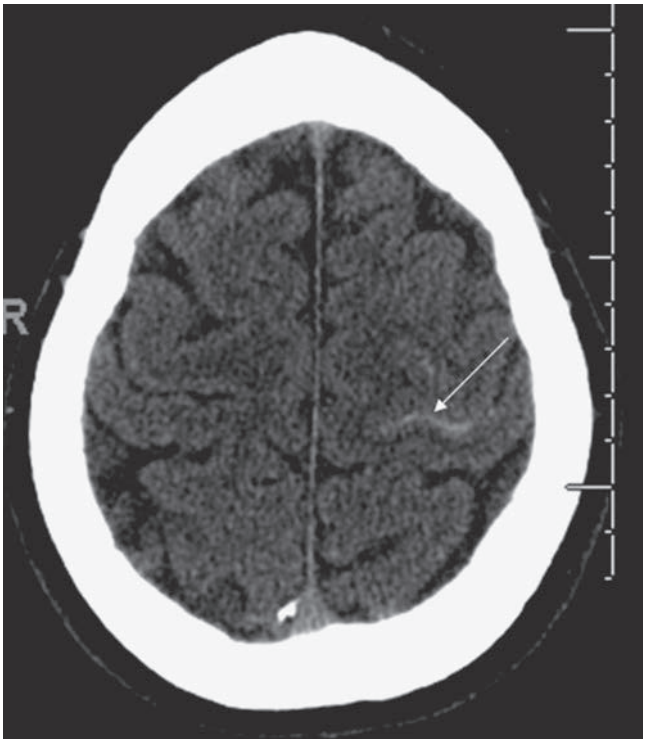

FIGURE 22-7 Computed tomography scan with arrow pointing to a small traumatic subarachnoid hemorrhage in left central sulcus.

thin hyperintensity on fluid-attenuated inversion recovery (FLAIR) MRI. The usual convexity location of tSAH distinguishes it from SAH caused by spontaneous rupture of a cerebral aneurysm, in which the blood is usually most prominent in the basilar cisterns and appears thicker on CT scanning. Occasionally, tSAH can have a CT appearance similar to that of aneurysmal SAH, and if a detailed history cannot help distinguish between spontaneous and traumatic SAH, imaging of the vasculature via CT, magnetic resonance, or catheter angiography may be required. In general, no specific treatment is needed for tSAH, and patient management is dictated by associated injuries.

Vasospasm refers to narrowing or even closure of a vessel. Severe cases may lead to subsequent ischemia or infarction in the associated vascular territory. Although much more common after rupture of a cerebral aneurysm and associated spontaneous SAH, it has also been reported after tSAH, especially in association with blast injury. The classic time course of vasospasm after spontaneous SAH is onset as early as 2 or 3 days after injury, peak incidence from days 3 to 14, and duration of up to 3 weeks.

VASCULAR INJURY

Posttraumatic stroke may result from injuries to the internal carotid artery (ICA), common carotid artery (CCA), or vertebral artery (VA) in the neck. Rarely, injury may be due to penetrating trauma with direct ICA, CCA, or VA injury. More often, vessel dissection is sustained in motor vehicle accidents, falls, extreme neck rotation, spine fracture, or through iatrogenic injury, such as surgery or chiropractic maneuvers.

Traumatic dissection is more common in the ICA and can occur anywhere from a few centimeters above the carotid bifurcation to the skull base. Less frequent is VA dissection, which is seen most commonly at the C1–C2 level. Vessel dissection allows blood to collect between the adventitia and media (pseudoaneurysm formation) or between the intima and media of the vessel wall (luminal stenosis). This intramural hematoma may expand or propagate, with distal propagation occurring more commonly than proximal.

Spontaneous dissections occur in younger to middle-aged adults; 70% occur between the ages of 35 and 50 years. A much lower proportion occurs in adolescents, and spontaneous dissection is rare in children. Most patients present with headache and neck pain that is often unrelenting. Symptoms and subsequent diagnosis may occur hours to weeks after the initial trauma and may include transient ischemic attack or stroke, Horner syndrome, and, less often, carotid bruits, pulsatile tinnitus, and lower cranial nerve palsies (CN IX–XII).

On CT scanning, dissection is seen as a linear lucency within an enhancing vessel. This represents the flap separating the true and false vessel lumens. MRI shows a crescentic band surrounding the native flow void.

Twenty percent of cases have an associated injury, such as cervical spine injury or silent dissection of another vessel. Treatment often consists of heparin anticoagulation (if not contraindicated by other injuries) followed by longer-term anticoagulation. Balloon angioplasty may be employed in select cases.

CLINICAL ASSESSMENT

Glasgow Coma Scale

The GCS has become the standard for clinical grading of TBI severity.[2] A patient is scored in three parameters: best motor function (M), best verbalization (V), and best eye opening (E). The sum of these individual scores represents the overall GCS score (Table 22-3). A neurologically intact patient can achieve a maximum score of 15. The most severely injured patients have a total score of 3. If a patient is intubated, the verbal score cannot be assessed. Instead of assigning an arbitrary verbal

TABLE 22-3: Glasgow Coma Scale (Recommended for Age ≥4)

Best motor		Best verbal	Best eye opening
6	Obeys		
5	Localizes	Oriented	
4	Withdraws	Confused (fluent speech)	Spontaneously
3	Flexes	Inappropriate (words)	To speech
2	Extends	Incomprehensible (sounds/grunts)	To pain
1	None	None	None

score in such cases, it is best to note that the verbal score cannot be determined reliably. Because the same GCS score can be derived from different combinations of motor, verbal, and eye opening scores, it is important that practitioners record each of the subscores and not just the overall number.

The GCS allows practitioners to communicate quickly and reliably about a patient's neurologic status. The postresuscitation GCS score is also effective for stratifying patients by injury severity. Patients with GCS scores between 13 and 15 are traditionally considered to have mild TBI. They are usually awake and have no focal deficits. Patients with GCS scores of 9 to 12 are often described as having moderate TBI. In general, they usually have alterations of sensorium and may have focal deficits. Those with the lowest GCS scores (3–8) have severe TBI. They usually will not follow commands and are often described as comatose.

Confounding factors that may alter the neurologic examination must not be present if the examiner expects a GCS evaluation to be accurate. Alcohol and other drugs may depress neurologic function, as can hypotension, hypoxia, sepsis, hypothermia, and other systemic factors. Sedating agents and paralytics may also interfere with accurate assessment. Spinal cord injury and long bone fractures can affect the motor component score of the GCS and should be sought if suggested by the mechanism of injury. Hearing deficits, lack of hearing aids, or a language barrier should also be taken into consideration. The pediatric population represents a special group for whom a modified version of the GCS is often used (Table 22-4).[22]

Pupils

The pupillary light reflex can be easily and rapidly assessed in the unconscious patient. Damage or hypoperfusion affecting the Edinger-Westphal nucleus in the midbrain, or compression of the third cranial nerve as the uncus (located in the medial temporal lobe) herniates into the tentorial notch from displacement by a space-occupying mass lesion, may result in pupillary dilatation. If these pathologic processes are sufficiently severe, the pupil will be fixed in the dilated position, with no constriction in response to bright light. Direct orbital trauma can also cause pupillary dilation and fixation in a sizable minority of cases. This possibility should be considered (quickly) before automatically assuming that a dilated pupil is caused by intracranial hypertension.

To facilitate performance of fundoscopy in patients in coma or with orbital or periorbital trauma, facial fractures, abnormal eye movement, or other pathologies, it may be necessary to instill mydriatics. To avoid confusion, this practice should only be permitted after concerns regarding elevated ICP have been addressed. Clear notation should be made, in the chart and at the bedside, as to when and which mydriatics have been used and for how long their effect will persist.

TABLE 22-4: Glasgow Coma Scale for Children (Recommended for Age <4)

Best motor		Best verbal		Best eye opening
6	Obeys			
5	Localizes	Smiles, oriented to sound, follows objects, interacts		
		Crying	**Interaction**	
4	Withdraws	Consolable	Inappropriate	Spontaneously
3	Flexes	Inconsistently consolable	Moaning	To speech
2	Extends	Inconsolable	Restless	To pain
1	None	None		None

Neurologic Examination

An accurate neurologic examination is essential to determine diagnosis, treatment, and prognosis in TBI patients. The examination may be limited due to a patient's age, level of education, native language, presence of sedating or paralytic medication, illicit drugs, hypotension, hypoxia, hypothermia, hypoglycemia, or other factors. Examination of the pediatric patient may be further limited by other considerations, including inability to visualize the pupils or fundi of the premature or newborn infant, limited cooperation, inability to understand language, and so on.

Monitoring the overall trend of a neurologic examination over time is critical. It must be understood that these examinations can and will fluctuate based on a patient's improving or declining condition, the evolution of disease processes, and the ability of medical personnel to minimize or eliminate sedation and other factors that confound an accurate neurologic assessment.

In the uncooperative patient or unconscious patient with severe TBI, the examination may be limited to the GCS, pupillary reactivity, and testing of various reflex actions (Table 22-5). As the patient becomes more alert and cooperative, a more complete neurologic examination will provide greater sensitivity for assessment of neurologic injury. The extent of the examination must be tailored to the patient's ability to interact with an examiner.

Systemic Evaluation

Assessment and treatment of TBI patients begin in the prehospital setting, often with input from family or bystanders. Care continues with the primary care physician or emergency medical technician, transfers to the physician in the emergency department, and eventually involves the neurosurgeon and other members of the trauma team. Treatments may be started at any point along a patient's journey as dictated by recognition of signs of neurologic injury and by availability of appropriate medication, equipment, and personnel.

The nervous system does not function in isolation. Patients with TBI often have additional injuries. Treatment specific to TBI is often complementary or adjunctive to treatment

TABLE 22-5: Neurologic Examination for Trauma

	Uncooperative patient	Cooperative patient
Level of consciousness	Glasgow Coma Scale	Orientation Comprehension, verbalization Fluency of language
Cranial nerves	Pupil reactivity (ambient and bright light) Afferent pupillary defect Facial asymmetry	Visual acuity/visual fields Funduscopic examination (papilledema, retinal hemorrhage) Complete cranial nerve examination
Motor	Movement to central and peripheral noxious stimuli (differentiate from flexion-withdrawal spinal reflex) Resting tone of anal sphincter	Strength assessment of muscle groups in four extremities Tone of voluntary anal sphincter contraction
Sensory	Grimace/grunt/withdrawal to noxious stimulus	Detailed examination to light touch and pinprick in major dermatomes (C5, C6, C7, C8, T4, T10, L2, L4, L5, S1) Joint position sense (posterior column function)
Reflexes	Deep tendon reflex Plantar reflex (Babinski), clonus Anal wink, bulbocavernosus reflex	Same as in uncooperative patient

of the trauma patient without neurologic injury. The basic principles of trauma resuscitation should be followed. These include rapid assessment and maintenance of airway, breathing, and circulation.

A detailed medical and surgical history should be obtained, including the events preceding a trauma, a description of the accident scene, accurate description of the patient's neurologic baseline, and any subsequent changes in neurologic status. Long-term medications as well as medications administered in the prehospital setting should be identified. Special attention should be paid to medications with the ability to alter the neurologic examination, including sedatives or psychopharmacologics (to restrain a combative patient), paralytics (for intubation or transportation), atropine (for cardiac resuscitation), and other mydriatics (for evaluation of ocular trauma), in addition to the possible presence of alcohol and other recreational drugs.

Primary and secondary surveys should be undertaken to evaluate for systemic injuries. Open scalp lacerations with vigorous hemorrhage can lead to fatal hemorrhagic shock. In the newborn or premature infant, cephalohematoma may allow enough displacement of blood to produce hemodynamic instability. Raccoon eyes (periorbital ecchymosis), Battle's sign (postauricular ecchymosis), and otorrhea or rhinorrhea suggest the presence of a basilar skull fracture. Palpable fractures or depressions may indicate bony injury and a higher likelihood of underlying hemorrhage or parenchymal injury, but care must be taken not to confuse significant soft tissue edema for a bony step-off. Periorbital edema or proptosis may suggest local ocular or orbital trauma. Puncture wounds may indicate the existence of serious penetrating injury to the brain, spinal cord, sympathetic plexus, or vasculature. Bruits of the carotid artery or globe of the eye may reveal the presence of carotid dissection or carotid-cavernous fistula, respectively. Multiple areas of swelling or bruising may indicate prior seizure activity or repeated syncopal episodes, as well as serially inflicted injuries.

IMAGING

Computed Tomographic Scan and Skull Radiography

Advances in imaging technology have relegated plain x-rays to a very limited role in the contemporary evaluation of trauma patients with potential head or spine injuries. Some clinicians still use skull films to identify pneumocephalus, skull fractures, and the tracks of penetrating objects. In the vast majority of cases, however, CT scanning provides more information. Also, it must be remembered that intracranial ricochets of projectiles may occur, and CT is far superior to plain films for characterizing such complex events.

Overwhelmingly, CT scanning has become the preferred initial imaging modality for patients presenting after head trauma. In a single rapid scanning session, and without patient repositioning, scans of the head, neck, chest, abdomen, and pelvis can be performed. From these, cervical, thoracic, and lumbar spine images can be reconstructed without additional radiation exposure. Administration of contrast allows for CT angiography and three-dimensional reconstruction to evaluate the vasculature of the head and neck.

Magnetic Resonance Imaging

MRI scans provide higher-resolution images of the brain, spinal cord, and other soft tissues than CT scans. This superior visualization of neural parenchymal tissue is ideal for the evaluation of infarction, ischemia, edema, and DAI. MRI is

also helpful for detecting ligamentous injury of the spine as well as spinal cord injury. However, disadvantages of MRI compared to CT scanning include slower image acquisition time, increased cost, restricted access to patients during image acquisition, and the magnet's incompatibility with ferromagnetic monitoring and supportive equipment that is commonly used in trauma patients. Use of MRI for the initial assessment of trauma is not routinely recommended since CT scanning permits detection of acute hemorrhage and fractures, which are the pathologies of greatest immediate concern in TBI patients.[23] In addition, once patients have stabilized in the ICU, MRI usually contributes little to prognostication beyond what has already been learned from other imaging studies and from the patient's clinical course.

Angiography

When the track of a penetrating injury passes near known locations of major vessels or when a delayed intracranial hemorrhage occurs, angiography may be used to look for pseudoaneurysms or other direct injuries to vessels. In prior decades or even nowadays when CT or MRI scanning is unavailable, angiography could reveal midline shift or other types of vascular displacement indicative of compressive mass lesions that might require immediate intervention.

MANAGEMENT

General Measures

Numerous guidelines based on critical evaluation of the pertinent literature have been created and disseminated. The best known among these is the *Guidelines for the Management of Severe Traumatic Brain Injury.*[24] Subsequent companion guidelines have been created for prehospital management of TBI,[25] pediatric TBI,[26] surgical management of TBI,[18] penetrating TBI,[27] and field management of combat-related head trauma.[28] The reader is referred to these documents for further background on creation of treatment recommendations. See Fig. 22-8 for a management algorithm.

Interventions for TBI may be initiated in series or in parallel. Some treatments can be started in the field. Others may be added as more advanced equipment or qualified personnel become available in the emergency department, operating room, or ICU.

Basic measures should be implemented in all TBI patients undergoing monitoring. These patients often require a setting with the capability for careful assessment of vital signs, fluid balance, and neurologic status. Initial goals are normothermia and euvolemia, with administration of isotonic fluids (ie, 0.9% NaCl + 20 mEq KCl/L). Many practitioners avoid dextrose-containing intravenous fluids because of concerns about potential deleterious effects of anaerobic metabolism of glucose in injured or ischemic brain. Patients usually receive prophylaxis against Cushing (stress) gastric ulcers, which are frequently seen in patients with severe TBI and elevated ICP. The head of bed is generally elevated to 30°. The neck should be kept midline, and the fit of the patient's cervical collar and endotracheal tube stabilizer should be assessed to prevent compression of the jugular veins. Most important is frequent assessment of the patient's condition, evaluation of responses to therapies, and willingness of providers to modify care strategies in a timely manner to help ensure optimal patient outcome.

Another basic measure is maintenance of hemoglobin (Hgb) concentration above a specified minimum level. Historically, this level has been commonly set at 10 g/dL, which was felt to represent the optimal trade-off between blood viscosity and oxygen-carrying capacity. Numerous reports have questioned the benefit of such a high threshold, and most practitioners came to accept a Hgb as low as 7 g/dL. But the question remained as to whether the exquisite sensitivity of the injured brain to hypoxia made TBI patients a special subgroup in which the traditional higher Hgb level was still required. A prospective randomized trial failed to confirm this hypothesis,[29] leading many clinicians to accept the lower Hgb of 7 g/dL as the minimum acceptable value even in TBI patients—assuming, of course, that there exists no other indication for targeting a higher goal.

Over the past two decades, it has become common to advocate for systematic, evidence-based treatment of TBI patients by dedicated neurosurgeons, neurologists, intensivists, surgical trauma and critical care teams, nurses, and allied health care workers. Several publications claim improved outcomes as a direct result of the arrival of an intensivist to a specific ICU. More likely, however, is that the very process of standardizing care and eliminating unwarranted excessive variation in care was the real driver behind improvement in outcomes compared to historical controls. The exact background or specialty of the clinician driving that standardization is less important.

BLOOD PRESSURE AND OXYGENATION

Medical practitioners cannot undo the events surrounding a primary brain injury. However, every effort must be made to mitigate or eliminate secondary insults, which may be even more devastating than the primary insult. Prior to, or during, transport to a hospital setting, a significant portion of patients may experience periods of hypoxemia or hypotension.[30] These are among the most common and most deleterious secondary insults. Occurrence of a single episode of hypoxemia or hypotension is an independent predictor of worse outcome after TBI.[31,32]

Oxygen saturation and blood pressure monitoring should start in the field and continue in the hospital setting. The goal is to prevent hypoxemia and hypotension or, if they occur, to identify and treat them as rapidly as possible. Crystalloid, colloid, blood products, or even intravenous pressors may be required to avoid hypotension. Empiric oxygen administration should start as early as possible. Endotracheal intubation may be required. Of interest, several studies have demonstrated worse outcomes in TBI patients who are

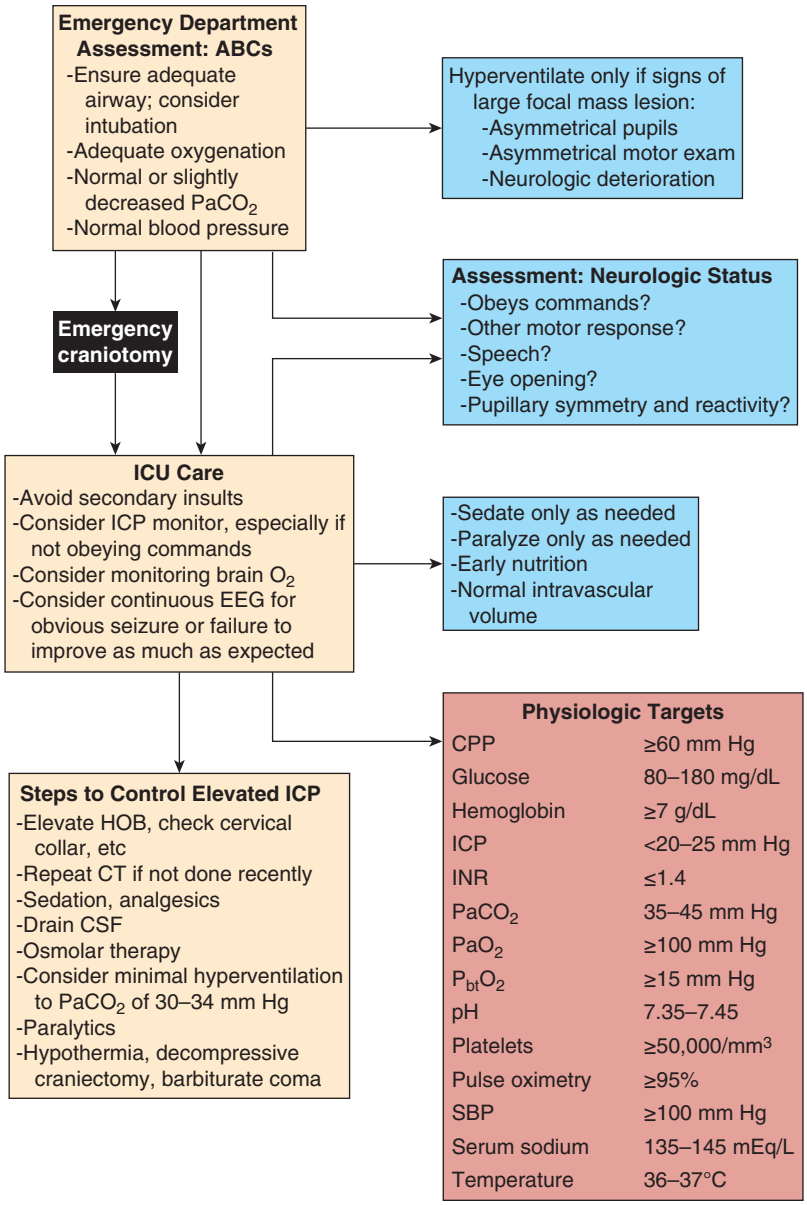

FIGURE 22-8 Algorithm for management of traumatic brain injury. CPP, cerebral perfusion pressure; CSF, cerebrospinal fluid; CT, computed tomography; EEG, electroencephalogram; HOB, head of bed; ICP, intracranial pressure; ICU, intensive care unit; INR, international normalized ratio; O_2, oxygen; SBP, systolic blood pressure.

intubated in the field. One possible reason is that the frequently chaotic prehospital environments surrounding TBI patients and the relative inexperience of some first responders in performing difficult intubations may combine to cause prolonged hypoxemia during attempts at endotracheal intubation. Another factor may be unintentional hyperventilation of intubated patients (and consequent cerebral vasoconstriction and reduction in cerebral blood flow [CBF]) from excessively rapid and forceful squeezing of the bag/valve system if patients are manually ventilated. Consequently, for many TBI patients, careful oxygenation and ventilation via a facemask may be preferred over attempts at prehospital endotracheal intubation.

NUTRITION

TBI patients usually exhibit an increase in basal energy expenditure (BEE). Patients who are sedated and paralyzed may show BEE increases up to 120% to 130% of baseline. Comatose patients (GCS score ≤8) with isolated TBI may have BEE approximately 140% of baseline (range, 120%–250%).[33] Mortality is reduced in patients who receive full caloric replacement by 1 week postinjury.[34] At least 15% of calories should be supplied as protein. Since it may take 2 or 3 days to ramp up feedings to desired levels, nutritional replacement should begin as soon after injury as possible.

Enteral feeding is preferred over parenteral nutrition because it enhances immunocompetence and reduces risk

profile.[35] If a patient has diminished gastric motility, a jejunal feeding tube can be placed since patients with severe TBI can tolerate early jejunal feeding even in the presence of gastric dysfunction and limited small bowel activity. Total parenteral nutrition can be considered if enteral feeding is not possible or if higher nitrogen intake is required.

PROPHYLAXIS

Seizures. Posttraumatic seizures (PTS) are potentially harmful in TBI patients for many reasons, including subsequent increased ICP and elevated metabolic demand that may exacerbate the effects of ischemia. TBI patients at increased risk for PTS include those with GCS score below 10 and those with depressed skull fractures, cortical contusions or intracranial hemorrhage, and penetrating injury or seizure within 24 hours of brain injury. Phenytoin has been shown to effectively reduce the risk of PTS during the first 8 days after injury, but not later.[24,36] Despite the absence of high-quality studies showing equivalent efficacy of levetiracetam for early PTS prevention, many centers are now using it because of ease of administration and a lower rate of adverse reactions.

Infection. Trauma patients may develop infections from grossly contaminated wounds, from immunosuppression following the stress of severe trauma or other causes, or from such necessary interventions as open surgical procedures, intubation for mechanical ventilation, and use of invasive monitoring equipment. In general, antibiotic coverage should be targeted toward specific organisms and stopped as soon as possible to minimize the risk of development of drug-resistant strains of bacteria or alterations in normal floral patterns.

Perioperative antibiotics are generally recommended only for a single dose preoperatively or at most for the first 24 hours after surgery. This same principle applies to insertion of external ventricular drains (EVDs). Maintaining patients on prophylactic antibiotics for the entire period that an EVD is in place is not recommended. Routine flushing or exchange of ventricular catheters is also not recommended. Use of ventriculostomy catheters impregnated or coated with antibiotics may result in an overall decrease in infection rate.[37]

Hemorrhage. Most critically ill trauma patients have decreased levels of plasma antithrombin (AT) activity. TBI patients, however, tend to have increased rates of coagulopathy, with supranormal AT activity that can progress to disseminated intravascular coagulation and fibrinolysis (DICF), as well as expansion of existing cerebral contusions and delayed development of additional hemorrhages.[38] Coagulopathy may be especially severe after penetrating brain injury.

For all trauma patients, medical history and review of systems should specifically inquire about history of excessive bleeding or clot formation, use of specific antiplatelet or anticoagulant medication (eg, aspirin, warfarin, or novel oral anticoagulants), medications that have antiplatelet compounds as a component, and medical disorders that pose an increased risk of bleeding. Measurements of prothrombin time, activated partial thromboplastin time, international normalized ratio, platelet count, and occasionally bleeding time, platelet function assay, or thromboelastography are often helpful in guiding the management of hemorrhage.

Effects of warfarin anticoagulation may be reversed by administration of vitamin K, fresh frozen plasma, or prothrombin complex concentrate; effects of heparin may be reversed with protamine sulfate; and thrombocytopenia or platelet dysfunction may be treated with donor platelet transfusion. Unfortunately, these interventions require time for preparation and administration, which may limit their usefulness in patients with life-threatening intracranial hemorrhage and rising ICP. Recombinant activated coagulation factor VII (rFVIIa) rapidly forms a complex with tissue factor to produce thrombin and, separately, converts factor X to its active form, factor Xa, resulting in a "thrombin burst" at the site of tissue damage. It is US Food and Drug Administration–approved for treating or preventing bleeding in hemophiliacs and in those with congenital factor VII deficiency. Studies of off-label uses for intracerebral hemorrhage and TBI have not demonstrated improved survival and also suggest an increased risk of thromboembolism. The novel oral anticoagulant (NOAC) dabigatran can be counteracted by a direct inhibitor, idarucizumab, but no specific reversal agent is yet available for other NOACs.

Deep Venous Thrombosis. Trauma patients in general, and TBI patients in particular, are at risk for venous thromboembolic complications such as deep venous thrombosis (DVT) and pulmonary embolus (PE). Risk factors for DVT and PE include stroke or spinal cord injury, prolonged surgery or prolonged bed rest, SAH or TBI causing altered coagulation or dehydration, and increased blood viscosity from cerebral salt wasting and treatment of cerebral edema.[39] Low-risk, prophylactic measures against DVT include passive range of motion, early ambulation, rotating beds, and electrical stimulation of calf muscles. If DVTs are not already present, pneumatic compression boots (PCBs) and sequential compression devices may be safely used and can reduce the incidence of DVT and PE.

Pharmacologic anticoagulation can increase the effectiveness of DVT prophylaxis but with the risk of additional hemorrhagic complications. Low-molecular-weight heparins have a higher ratio of anti–factor Xa to anti–factor IIa activity versus unfractionated heparin, have greater bioavailability after subcutaneous injection, and have more predictability in terms of plasma levels. They can be added to use of PCBs without significantly increased risk of hemorrhage,[40] and their use has been recommended in postoperative neurosurgery patients. Because there are no universally accepted recommendations for the method and timing of postoperative anticoagulation, this decision should be tailored individually to each patient. Many centers now initiate pharmacologic DVT prophylaxis within 24 hours of surgery or, in the case of intracranial hemorrhage, within 24 hours of verifying stability of hematoma size on repeat CT scan.

Cerebral Metabolism and Pathophysiology

INTRACRANIAL PRESSURE

To understand the rationale behind ICP management, one must first recognize that pressure in the intracranial space differs from that in other body compartments. If a patient sustains an injury to an arm or leg, the surrounding soft tissue may expand outward from the humerus or femur. By contrast, in cases of TBI, the brain is unable to expand because it is confined within the rigid skull.

The modified Monro-Kellie hypothesis assumes that the skull is completely inelastic, that the ventricular space is confluent, and that pressures are equally and readily transmitted throughout the intracranial space. This hypothesis states that there is a balance between the brain, intravascular blood, and CSF contained in the intracranial space. Increases in the volume of one constituent (eg, cerebral edema, hyperemia) or addition of new components (eg, tumor, hemorrhage) mandate compensatory decreases in other constituents in order to maintain constant ICP.

Mildly increased localized pressure in part of the brain may cause neurologic dysfunction confined to the affected area. More severe increases in pressure may cause local tissue compression as well as shift of intracranial structures, subfalcine and transtentorial herniation, and both local and distant neurologic dysfunction. In the most severe cases, transtentorial herniation produces compression at the level of the brainstem, potentially causing direct tissue damage to the midbrain, occlusion of arteries, infarction, and death.

Normal ICP varies by age. Normal values probably lie below 10 to 15 mm Hg in adults and older children and in the range of 3 to 7 mm Hg in younger children, 1.5 to 6 mm Hg in infants, and possibly even at subatmospheric levels in neonates. Intracranial hypertension (IC-HTN) has been reported in 13% of trauma patients with a normal head CT, 60% of patients with an abnormal head CT (demonstrating hemorrhage, contusion, edema, herniation, or compressed basal cisterns), and approximately 60% of patients with a normal head CT plus two or more of the following criteria: age greater than 40 years, systolic blood pressure greater than 90 mm Hg, and unilateral or bilateral abnormal motor posturing (flexing or extending to a noxious stimulus).[41] Therefore, ICP monitoring is often recommended in patients with severe TBI (GCS score of 3–8) and an abnormal CT scan or in those with severe TBI, a normal CT scan, and two or more of the select criteria listed earlier. ICP monitoring may also be considered in patients in whom a reliable neurologic examination cannot be performed because of sedatives or paralytics required for other reasons, such as difficult ventilator management or extreme agitation, in addition to those who require general anesthesia for nonneurologic surgery.

Higher mortality and worse outcomes have been described in patients with ICP persistently above 20 mm Hg.[42] Therefore, most centers consider ICP to be elevated when it exceeds 20 to 25 mm Hg, and ICP reduction measures are often recommended to bring values below this range.

MONITORING OF ICP

The most accurate, reliable, and lowest-cost ICP monitoring technology is the fluid-coupled ventriculostomy catheter, or EVD, connected to an external strain gauge. Another advantage of EVDs is that they permit CSF drainage as a therapeutic measure to control ICP. Parenchymal ICP monitors based on fiberoptic or miniaturized strain gauge transduction require less tissue penetration and do not require the ability to cannulate the cerebral ventricles (see Atlas Figure 1). They are roughly as accurate as EVDs but carry a higher cost. They cannot be recalibrated in situ and may be subject to measurement drift, although this is currently not as significant a concern as it was with earlier versions of these monitors. Parenchymal monitors do not allow for therapeutic drainage of CSF. Although some neurosurgeons place these devices in the subdural or epidural spaces, such positioning renders their data less accurate.

Of interest, a prospective trial found no difference in outcome between severe TBI patients managed with ICP monitoring and patients in whom elevated ICP was inferred and treated based on clinical and imaging findings.[43] This trial is often incorrectly interpreted as indicating that ICP monitoring is of no benefit in severe TBI patients. A more appropriate interpretation is that ICP was monitored and elevated ICP was treated in both groups, with the difference being invasive monitoring versus noninvasive monitoring based on examination and imaging. This trial did not compare a monitored to an unmonitored group.

CEREBRAL PERFUSION PRESSURE

Neurologic dysfunction may result from direct disruption of tissue or from impaired metabolism in a structurally intact brain. The recently injured brain is highly vulnerable to ischemia both regionally, near contused or otherwise damaged tissue, and also globally, from more diffuse impairment of CBF regulation. In addition, brief periods of hypotension or hypoxia that might be well tolerated by an uninjured brain may have significant deleterious consequences in a brain that has just received a traumatic insult.

For neural tissue to function normally, CBF must be adequate to meet metabolic demand. CBF depends on cerebral perfusion pressure (CPP), which is calculated by subtracting ICP from mean arterial pressure (MAP – ICP). Studies have shown that the injured adult brain is more susceptible to ischemia if the CPP trends below 50 mm Hg.[44] Children have been demonstrated to show improved survival when CPP is sustained above 40 mm Hg.[45] In adults, artificially maintaining CPP above 70 mm Hg results in unacceptably higher rates of adult respiratory distress syndrome without significant improvement in functional outcome.[46,47] Collectively, these data suggest that there exists a "floor" below which CPP should not drop (the exact value varies with age), but above this floor, there is no benefit from driving CPP to higher levels. There is likely an age-dependent continuum of optimal CPP values. Common practice is to avoid CPP less than 40 mm Hg in children, less than 60 mm Hg in adults,

and artificial elevation greater than 70 mm Hg in either population.

MONITORING OF CEREBRAL BLOOD FLOW AND METABOLISM

CT perfusion scanning can measure relative cerebral blood volume, CBF, and mean transit time after injection of iodinated contrast. It has been used extensively in stroke patients and has been investigated in TBI patients to determine the potential viability of contusional and pericontusional tissue and also to help guide judicious use of other therapeutic strategies, such as hyperventilation (HV).

Measurement of cerebral arteriovenous difference in oxygen content or of oxygen saturation in the jugular bulb ($S_{jv}O_2$) can also be used to assess global cerebral perfusion. Normal venous saturation of oxygen is approximately 50% to 69%. The occurrence of multiple episodes of venous desaturation (<50%) or of a sustained and profound single such episode is associated with poor outcome.[48] In addition, excessively high $S_{jv}O_2$ (>75%) is associated with poor outcome, perhaps because such measurements reflect cerebral hyperemia or possibly the existence of significant areas of infarcted tissue that will not extract oxygen.

More focal techniques to measure CBF include transcranial Doppler ultrasonography and parenchymal CBF monitoring. Thermal diffusion probes provide local CBF measurements based on the temperature difference between two points on a probe, the relative conductive properties of cerebral tissue, and convective properties of blood flow.

Cerebral microdialysis requires intraparenchymal placement of a very thin microdialysis catheter. Specific biochemicals diffuse into dialysate through a semipermeable membrane at or near the tip of these catheters. The dialysate is collected and subsequently analyzed. Neurochemical changes indicative of primary and secondary brain injury may be detected. TBI patients with poor clinical outcome have been shown to have elevated levels of specific neurotransmitters, as well as elevated lactate/pyruvate ratios and abnormal lactate and glutamate levels.

Brain tissue oxygen tension ($P_{bt}O_2$) monitoring allows direct measurement of oxygen level in a specific region of the brain. In most cases, $P_{bt}O_2$ mirrors oxygen delivery, but it may also increase if the tissue surrounding the catheter is infarcted and incapable of metabolizing oxygen. Normal $P_{bt}O_2$ has been reported as approximately 32 mm Hg. Patients with multiple or prolonged episodes of $P_{bt}O_2$ below 10 to 15 mm Hg have been shown to have increased morbidity and mortality.[49] Therapies are commonly targeted to keep $S_{jv}O_2$ above 50% and $P_{bt}O_2$ above 15 mm Hg or higher.[24]

Probes are often placed in penumbral tissue that is thought to be "at risk" but still salvageable. Others prefer to place such probes in brain that appears "normal," with the thought that monitors in this area provide data about global cerebral metabolism, as opposed to the local metabolic environment near a contusion or other visible lesion. Favorable results in a phase II study of $P_{bt}O_2$-based management in severe TBI patients[50] have led to the organization, funding, and imminent launch of a definitive phase III trial. At the present time, however, $S_{jv}O_2$ and $P_{bt}O_2$ monitoring are best viewed as adjuncts to ICP and CPP monitoring.

Management of Elevated Intracranial Pressure

ANALGESICS AND SEDATIVES

TBI patients will likely have increased levels of stress, agitation, and discomfort. These may cause increases in sympathetic tone, temperature, and blood pressure and lead to increased ICP, metabolic demand, and resistance to controlled ventilation.

The medications used to treat pain and agitation and their doses must be carefully monitored to achieve a balance between their beneficial effects in reducing pain and anxiety and their potentially adverse effects of hypotension, alteration or masking of the neurologic examination, and rebound ICP elevation when they are discontinued. If heavy sedation or pharmacologic paralysis is needed and a neurologic examination becomes unobtainable, consideration should be given to placement of an ICP monitor.

Short-acting agents are preferred in order to facilitate frequent neurologic examinations. Continuous infusion is often used instead of bolus administration to avoid potential transient ICP increases between doses. Fentanyl and its derivatives (remifentanil and sufentanil) are used increasingly for both acute and longer-term analgesia. They are short acting, reversible, and suitable for administration by continuous infusion.

Midazolam and propofol are two commonly used sedatives. Midazolam is a short-acting benzodiazepine that is effective for sedation of the ventilated TBI patient. Propofol is a hypnotic anesthetic with rapid onset and a very short half-life that facilitates rapid neurologic assessment. It should be limited in both concentration and duration to avoid propofol infusion syndrome.[51] First described in children and later in adults, this syndrome has generally been reported after use of excessively high doses or extensive durations of use of propofol. Features can include hyperkalemia, hepatomegaly, metabolic acidosis, rhabdomyolysis, renal failure, and death. Caution should be exercised if doses exceed 5 mg/kg/h or if duration of treatment exceeds 48 hours.

HYPEROSMOLAR THERAPY

While the exact mechanism of mannitol's beneficial effects is unclear, two primary actions have been postulated. In the first few minutes, it produces immediate plasma expansion with reduced hematocrit and blood viscosity, improved rheology, and increased CBF and oxygen delivery. This reduces ICP and is most notable in patients with CPP below 70 mm Hg.[52,53] Over the next 15 to 30 minutes, and perhaps lasting 90 minutes to 6 hours, mannitol creates an osmotic gradient, with increased serum tonicity and withdrawal of edema fluid from the cerebral parenchyma.

When given as a bolus, mannitol begins to reduce ICP after 1 to 5 minutes. This effect peaks at 20 to 60 minutes.

For acute ICP reduction in cases of neurologic worsening or herniation, an initial bolus of mannitol is often given at 1 g/kg, with subsequent administration at smaller doses and longer intervals, such as 0.25 to 0.5 g/kg every 6 hours. Mannitol opens the blood–brain barrier (BBB). It may cross the BBB itself, drawing water into the brain and transiently exacerbating vasogenic cerebral edema. In the past, furosemide was sometimes used synergistically with mannitol to reduce cerebral edema through increased serum tonicity and reduced production of CSF, although this intervention seems to have fallen out of favor.

There has been concern that continuous mannitol infusions lead to elevated serum levels of mannitol, with sequestering of mannitol within brain tissue, rebound shifts of water back into the brain, and worsening outcomes. It was thought that bolus administration reduced this effect and was more effective than continuous mannitol infusion for ICP reduction, with the added benefit of maximized rheologic increase in CBF. More recent investigations suggest that there are no good data to support this.[54] The significant water shifts caused by mannitol suggest that it may be wise to replace intravenous fluids to maintain euvolemia.

Acute renal tubular necrosis may be seen when mannitol is used in high doses in patients with preexisting renal disease or when other potentially nephrotoxic drugs are administered. Use of mannitol is often stopped when serum osmolality exceeds 320 mOsm/L.[55] Urine output should be followed to help minimize the likelihood of hypotension and hypovolemia.

Although TBI patients usually receive mannitol in conjunction with ICP monitoring, some clinicians employ high-level empiric dosing.[25] No strong evidence supports empiric prehospital administration of mannitol to TBI patients,[56] but mannitol may be of benefit in patients with signs of rapidly expanding mass lesions, such as decreasing level of consciousness with unilateral pupillary dilatation and contralateral hemiparesis. In such cases, mannitol may be used as a bridge toward definitive therapy, such as operative evacuation of mass lesions.

As with mannitol, hypertonic saline (HTS) is thought to lower ICP through two mechanisms. First, hypernatremia produces an oncotic pressure gradient across the BBB and mobilizes water from brain tissue. Second, rapid plasma dilution and volume expansion, endothelial cell and erythrocyte dehydration, and increased erythrocyte deformability lead to improvements in rheology, CBF, and oxygen delivery. HTS is often administered as a continuous infusion of 25 to 50 mL/h of 3% saline (replacing the patient's isotonic intravenous fluid) or bolus infusions of 10 to 30 mL of 7.2%, 10%, or 23.4% saline solution. Clinical response can begin within minutes and may last for hours, making HTS a potentially useful intervention in cases of severe ICP elevation or acute herniation syndrome.

A common target for serum sodium concentration is 145 to 160 mEq/L, and even higher serum sodium levels are sometimes chosen. However, it is unclear if deliberately driving up the serum sodium concentration to supranormal levels can prevent IC-HTN. Instead, high sodium levels are better viewed as a reason to stop administering HTS, rather than as a desired goal. Serum sodium and osmolality levels are often followed serially because excessively rapid increases in sodium, which may occur during HTS administration, may result in central pontine myelinolysis. This occurs most commonly in patients with preexisting chronic hyponatremia and is rarely seen in the patient with baseline normonatremia who receives HTS. HTS may also induce or exacerbate pulmonary edema in patients with underlying cardiac or pulmonary deficits.

Considerable debate attends the question of whether mannitol or HTS is the "better" osmolar agent. No high-quality data are available to provide guidance. In most cases, either agent would be acceptable. Obvious exceptions might include patients with fluid overload or congestive heart failure, in whom the diuretic effects of mannitol might be beneficial and the extra sodium load of HTS could be harmful, and hyponatremic patients, for whom an increase in sodium concentration may be desirable.

HYPERVENTILATION

By lowering PCO_2, HV induces vasoconstriction, reduction of cerebral blood volume, and reduction in ICP. Time of onset of effect ranges from 30 seconds to 1 hour. Peak effect may be seen at 8 minutes and may last up to 15 to 20 minutes. This rapid onset of action makes HV particularly effective in the treatment of an IC-HTN crisis and as a bridge to more definitive therapy, such as surgical decompression, or as a temporizing measure while other ICP-lowering therapies take effect.

Although HV was once used as a first-line therapy, concerns subsequently grew regarding prophylactic or prolonged use in TBI patients. During the first 24 hours after injury, perhaps as many as 50% of patients with severe TBI exhibit cerebral ischemia.[57] Induced vasoconstriction from HV may lower CBF even further. Depending on the degree of functional autoregulation, the brain may undergo increases in the oxygen extraction fraction or shunting of blood to ischemic areas, with the net result being an increase in the total ischemic volume.

Severe TBI patients should be kept normocarbic (PCO_2 = 35–45 mm Hg). If HV becomes necessary, brief periods of mild HV (PCO_2 = 30–35 mm Hg) may be effective for bringing ICP down while other treatment strategies are initiated. Prophylactic HV is contraindicated because it has been associated with worse outcomes.[58] If HV is used, consideration should be given to tracking cerebral oxygenation via monitoring of $S_{jv}O_2$ and/or $P_{bt}O_2$.

DECOMPRESSIVE CRANIECTOMY

If the above therapies fail to provide adequate control of ICP, additional interventions that may be considered include decompressive craniectomy (DC), barbiturate coma, and hypothermia.

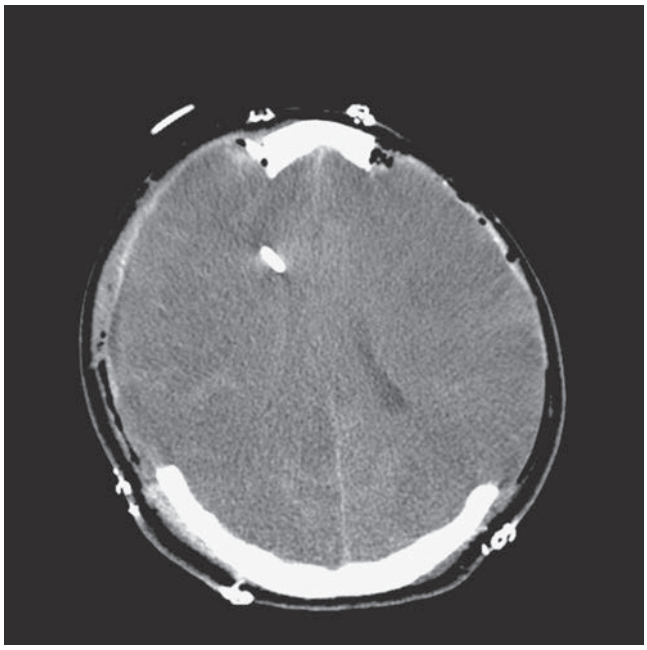

FIGURE 22-9 Computed tomography scan showing bilateral hemispheric decompressive craniectomies performed in a patient with severe cerebral edema.

Some patients with TBI require emergency craniotomies to evacuate focal hemorrhagic lesions. The bone is removed, the lesion resected, dura closed, and bone replaced. More severe cases of TBI may develop diffuse cerebral edema, contusions of large size in adjacent areas, or multiple, coalesced contusions. In these cases, it may be preferable to leave the bone flap off. Additionally, when ICP is refractory to the previously mentioned management techniques, DC effectively expands the intracranial space to lower the ICP.

The most common DC technique is a unilateral hemispheric decompression. Bifrontal and bilateral hemispheric craniectomies (Fig. 22-9) have also been described and may be more appropriate in some cases. The dura is opened widely, and areas of noneloquent contused or devitalized brain can be removed if required. The larger the size of the decompression, the better. The brain is then contained only by the augmented dural covering and the more compliant scalp.

DC is most commonly used in patients with IC-HTN refractory to maximal medical management. Ideally, operative intervention should occur before ICP has been dangerously elevated for sustained periods of time. Success depends largely on patient selection. Regardless of the preoperative indications or patient profile, continuing postoperative IC-HTN greater than 35 mm Hg has been associated with very high mortality rates.[59]

Early surgical intervention may be an option for potentially salvageable patients presenting with severe unilateral or bilateral cerebral edema, parenchymal lesions resistant to initial medical management of ICP, or other injuries whose management conflicts with standard ICP control measures (eg, patients with acute respiratory distress syndrome requiring elevated ventilatory pressures).

DC can be effective for reducing ICP, but improvement in outcome has been difficult to demonstrate because most reports describing this practice consist only of case series, lack appropriate control subjects, and/or do not achieve statistical significance with regard to all end points.[60] The prospective, randomized, controlled DECRA trial of bifrontotemporoparietal DC in patients with diffuse TBI who exhibited increasing ICP was essentially an investigation of "prophylactic" DC intended to prevent IC-HTN and its sequelae. The results demonstrated that DC not only failed to improve outcome, but was actually associated with worse outcomes. However, post hoc statistical adjustment for imbalance of rates of baseline pupillary reactivity between groups caused differences in outcome to disappear.[61] Thus, very early (essentially prophylactic) DC in patients with diffuse TBI does not improve outcome and may even make things worse.

RESCUE-ICP was another trial investigating the role of DC not as an up-front prophylactic intervention, but as a salvage procedure after other interventions have failed to control elevated ICP.[62] The investigators found that DC was effective at lowering mortality, but at the price of increased numbers of vegetative and severely disabled patients; the likelihood of favorable outcome was not improved by DC.

DC is not a benign procedure. Complications are common. These include infection, subdural hygroma, hydrocephalus, syndrome of the trephined, CPP breakthrough, and cerebral infarction.

BARBITURATES

Barbiturates benefit TBI patients by decreasing the cerebral metabolic rate of oxygen ($CMRO_2$), decreasing formation of free radicals and intracellular calcium influx, and lowering ICP. Side effects such as immunosuppression and hypotension from reduced sympathetic tone and mild myocardial depression often limit the use of these agents. TBI patients in coma (GCS score ≤8) receiving barbiturate therapy demonstrate infectious and respiratory complication rates in excess of 50%,[59] and significant systemic hypotension may occur in 25% of patients[63] despite adequate intravascular volume and pressor therapy. Barbiturates clearly reduce ICP, but studies have shown both worsened and improved outcomes for TBI patients receiving barbiturate therapy.

Patients with hemodynamic instability, sepsis, respiratory infection, or cardiac risk factors are excluded from barbiturate therapy. Those receiving barbiturates should be closely monitored for signs of cardiac compromise or infection, with cessation of therapy if systemic effects of the treatment become significant and unmanageable. A pretherapy echocardiogram and intratherapy use of a pulmonary artery catheter should be considered.

There is no role for prophylactic barbiturate therapy in TBI patients because it increases hypotension without significantly improving outcome.[64] It should be reserved for use in controlling ICP only after other treatments have failed.

A typical pentobarbital regimen is a loading dose of 10 mg/kg over 30 minutes, followed by a 5 mg/kg/h infusion

for 3 hours. A maintenance dose of 1 mg/kg/h should then be started. Serum barbiturate levels of 3 to 4 mg/dL should be maintained, although poor correlation exists between serum level, therapeutic benefit, and systemic complications. Titration to clinical effect is commonly done. Continuous electroencephalographic evaluation is preferred by some. Dosing to the point of burst suppression produces near-maximal reductions of $CMRO_2$ and CBF.

HYPOTHERMIA

Induced therapeutic hypothermia in TBI patients may reduce cerebral metabolism, ICP, inflammation, lipid peroxidation, excitotoxicity, cell death, and seizures. Adverse effects of hypothermia include decreased cardiac function, thrombocytopenia, elevated creatinine clearance, pancreatitis, and shivering with associated elevations in ICP. Interest in induced hypothermia was fueled by a large body of preclinical evidence, anecdotal observations (such as children trapped under the ice in frozen lakes), single-center clinical trials, and several meta-analyses.[65-69] Although its use in TBI patients has been adopted by some trauma centers, and level 1 evidence supports its use in patients with cardiac arrest from ventricular fibrillation or ventricular tachycardia and in neonatal hypoxic-ischemic encephalopathy, clinical trials to date have not shown significant improvements in outcome from induction of hypothermia in TBI patients. This lack of effect includes both immediate prophylactic use and later use as a salvage therapy after other measures have failed to control IC-HTN.[70-72]

Meta-analyses of more recent data and subsequent guidelines[24] note a nonsignificant trend toward mortality reduction (compared to normothermic controls) when target temperatures are maintained for greater than 48 hours. Hypothermia-treated patients have been reported to reach significantly better outcomes. Similarly, retrospective analyses suggest that perhaps patients with traumatic intracranial hematomas that require surgical intervention fare better if their temperature is lowered to or below 35°C within 90 minutes of the start of surgery.[73] Additionally, patients who are hypothermic on admission seem to have improved outcomes if hypothermia is maintained, as opposed to rapidly warming them to normothermia. Interpretation of these results is limited, however, by small sample sizes and potential confounding factors in each study.

Many practitioners aim for a target temperature of 33°C and, if possible, maintain that degree of hypothermia for 48 hours or longer. This temperature may be carefully titrated upward to 34°C or 35°C while ICP is monitored because some patients may still demonstrate the ICP-lowering benefit of hypothermia at these temperatures, with perhaps less risk of adverse systemic events from lower temperatures. Monitoring for untoward effects of hypothermia should include attention to potential electrolyte abnormalities and cardiac rhythm disturbances. Rewarming of these patients should take place very slowly, not exceeding 1°C per 4 to 6 hours, or more slowly (or even returning to a lower temperature) if ICP begins to rise precipitously.

STEROIDS

Glucocorticoids are not recommended for treatment of TBI.[24,74,75] Increased mortality has been reported in trials investigating the effect of steroids on outcome after TBI. Of course, this restriction does not apply in cases when patients require steroids for treatment of other medical problems.

Progesterone is another steroid that has been investigated as a potential therapy for TBI. Despite supportive preclinical and early-phase clinical data, progesterone failed to confer benefit to TBI patients in two large multicenter trials.[76,77]

OUTCOME

Prognostic Data

To interpret and compare the effectiveness of various treatments, common end points are necessary for communication between practitioners or comparison of studies. The Glasgow Outcome Scale (GOS)[78] (Table 22-6) is a widely used outcome grading scale, with many studies separating patients into those with good outcome (GOS = 4 or 5) and those with poor outcome (GOS = 1–3). Although its separation of patient categories is relatively coarse and may not identify the subtleties of recovery in many TBI patients, it remains a useful tool for describing patient outcome, just as the GCS is a useful tool for tracking a patient's neurologic examination. The subsequent GOS-Extended permits slightly more refined discrimination by expanding the original five categories into eight.[79] Importantly, a patient's ultimate neurologic outcome may not be fully evident until weeks or months of treatment have taken place in hospitals, rehabilitation centers, and at home.

The medical practitioner is often called upon early in a patient's course to make predictions of outcome based on

TABLE 22-6: Glasgow Outcome Scale

Score	Meaning	Notes
5—good recovery	Resumption of normal life	May have minor deficits, ± return to work
4—moderate disability	Disabled but independent	Can use public transportation and work in sheltered environment
3—severe disability	Conscious but disabled	Dependent for daily support, ± needs institutionalization
2—persistent vegetative state	Unresponsive, speechless	May open eyes spontaneously after several weeks and achieve sleep/wake cycles
1—death	Dead	

limited information. Various studies and meta-analyses[80-82] show that worse prognosis is seen in patients with bilaterally dilated pupils or absent pupillary light reflexes, absent oculocephalic or oculovestibular reflexes, increased Injury Severity Score (>40), extremes of age (>60 years and possibly <2 years), hypotension (systolic blood pressure <90 mm Hg, worse with concomitant hypoxemia), abnormal CT scan (extensive tSAH, compression or obliteration of basal cisterns), persistent ICP greater than 20 mm Hg, elevated ICP during the first 24 hours, or presence of apolipoprotein E4 allele. Probability of poor outcome increases with worsening initial total GCS score (especially GCS <9), and some studies show worse prognosis based on lower GCS subscores (motor ≤3, eye opening ≤2, verbal response ≤2).

Brain Death

"Brain death" refers to the irreversible absence of any brain function. Ancillary tests are sometimes used to support the clinical diagnosis, but they should not supplant the clinical examination as the primary means of making this diagnosis. Many people, including some in health care fields, think that brain death is somehow not "real" death. Uninformed discussion of this condition by popular media often contributes to this misunderstanding. It must be emphasized that brain death is a legally binding death, with the same degree of finality and certitude as cessation of cardiopulmonary function. The required components of diagnosing brain death may vary between states and between different hospitals, but the core elements are comparable.

No confounding factors may be present if brain death is to be diagnosed. Examples include hypothermia, hypotension, sedatives, paralytics, alcohol or other drugs of abuse, hepatic encephalopathy, hyperosmolar coma, atropine, recent cardiopulmonary resuscitation/shock/anoxia, and similar factors. Patients must have nonreactive and usually dilated pupils and no observable corneal, oculocephalic, oculovestibular, gag, cough, or other brainstem-mediated reflexes. No spontaneous breathing is present. Patients demonstrate no movement or other response to pain.

If the clinician chooses to forgo apnea testing because of a patient's inability to tolerate it or because of institutional protocols, other types of ancillary testing may be used if desired. These include cerebral radionuclide testing to confirm lack of uptake in brain parenchyma, cerebral angiography to verify absence of intracranial blood flow, and specialized electroencephalography techniques to verify electrocerebral silence, among other methods.

TBI patients who progress to brain death may be candidates for organ donation. Specialized organ procurement organizations are present in most states and represent a party separate from the treating team, with no conflict of interest regarding the patient's care. In many of these tragic cases, a patient's family members may receive a small measure of comfort from the knowledge that their decision to donate has helped other suffering patients (see Chapter 54).

FUTURE DIRECTIONS

Although much has been learned about the pathophysiology and treatment of TBI, translation of this knowledge to effective treatments has been challenging. One major change in the conceptualization of TBI has been the realization that TBI is actually a heterogeneous category composed of different subtypes of diseases, such as nonoperative diffuse injury versus large focal hematoma requiring immediate surgery. Future trials will likely be more focused in terms of the subtypes of TBI patients that are enrolled. Clinical studies are already moving beyond simple classification of outcome as "good" or "bad" by incorporating more sophisticated statistical techniques.

Another change in approach to clinical research is the growing recognition of the value of high-quality patient registries. There are far too many questions and far too few patients, investigators, and funds to subject every research question to a randomized, prospective, blinded, controlled trial. Conducting such trials for surgical interventions in emergency and critical care patients is especially problematic. Although the traditional approach to clinical trials will continue to predominate for the foreseeable future, the awareness that registries can supplement such trials and even represent a better method of investigation in some cases will allow many important questions to be addressed in a more expeditious manner than in the past.

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23

Eye

Hampton Addis • Joshua Brozek • F. Lawson Grumbine

KEY POINTS

- Mechanical trauma to the eye is divided into open globe injury, where the sclera and/or cornea (eyewall) have a full-thickness wound, and closed globe injury, where these structures do not have a full thickness-injury.
- The six extraocular muscles are attached directly to the sclera, which is covered by the thin clear conjunctiva, which itself terminates at the cornea.
- In the patient's history, posttrauma floaters and a visual field defect are highly suggestive of a retinal detachment.
- When trying to measure vision between 1/200 and 20/20, the patient should be wearing his or her eyeglasses.
- The pupil may be peaked if the iris is sealing (plugging) a corneal or anterior scleral laceration.
- Computed tomography imaging is used to evaluate orbital fractures, orbital foreign bodies, and intraocular foreign bodies.
- After the application of appropriate medication, corneal abrasions heal faster without patching.
- Prior to consultation with an ophthalmologist, routine treatment for a hyphema can be initiated with topical steroid eye drops four times a day and a topical cycloplegic agent.
- Intraocular foreign bodies are associated with an increased incidence of endophthalmitis that results in poorer visual outcomes after penetrating injury.
- One of the signs of an orbital floor fracture is decreased skin sensation on the cheek of the affected side.

EPIDEMIOLOGY OF EYE TRAUMA

Worldwide 1.6 million people are estimated to be blind from ocular trauma, and another 19 million people suffer from severely impaired vision in one eye due to trauma.[1] Published literature from England looking at more than 39,000 patients treated for major trauma over 15 years found that 2.3% of patients had associated ocular injuries. Given that the eyes represent only 0.27% of the total body area, it is a curious phenomenon that the eyes are affected so often. In this series, the most common injuries involved the cornea, optic nerve, conjunctiva, and sclera.[2]

Men are reported to be four times more likely to suffer from ocular trauma compared to women, and in the same series from England, 75.1% of major trauma patients with ocular injuries were men. While ocular trauma most commonly results from motor vehicle accidents, workplace injuries and recreational injuries are also very commonly seen. Most injuries were due to sharp objects (54.1%), followed by blunt objects (34.4%); chemical injuries accounted for 11.5% of ocular injuries.[3]

EYE TRAUMA: TERMINOLOGY AND CLASSIFICATION

Eye trauma is divided first by etiology into mechanical, chemical, thermal, and electric. Thermal (eg, corneal burn from curling iron) and electric (eg, lightning) eye traumas are very uncommon, and treatment of complications will be by an ophthalmologist in an outpatient setting after discharge from the emergency department/urgent care setting. Chemical injury (alkali and acid burns) is not uncommon, and its management will be discussed in detail because immediate intervention by first responders and emergency department physicians can be sight-saving.

Mechanical eye trauma is the most common form of eye injury. It is divided into open globe injury, where the sclera and/or cornea (eyewall) have a full-thickness wound, and closed globe injury, where the eyewall does not have a full-thickness wound (Fig. 23-1).[4-6] Closed globe injuries are further subdivided into contusion injuries, lamellar laceration (ie, partial-thickness laceration), and superficial foreign body (ie, foreign body lodged on cornea, conjunctiva, or under

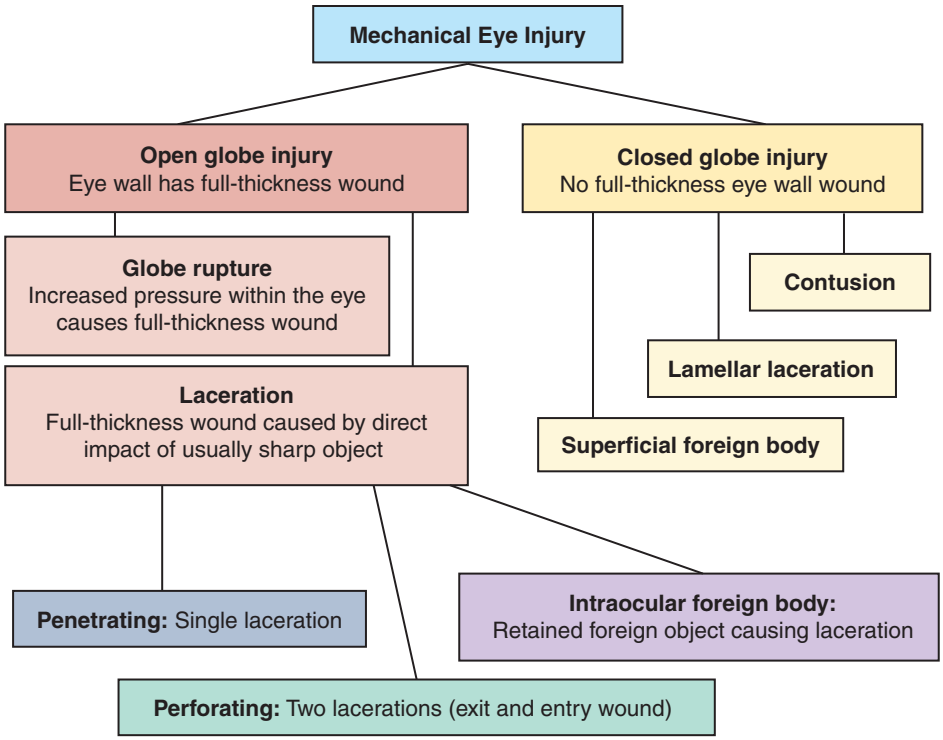

FIGURE 23-1 Injury classification.

the conjunctiva but *without* full-thickness wound of the eyewall).[6] Open globe injuries are further divided into ruptured globes and globe lacerations.[4,5] Ruptured globes result from blunt trauma, due to an extreme elevation of intraocular pressure at the moment of impact causing a rupture of the eyewall at the weakest site of the globe (force from inside out), usually away from the site of impact and frequently with significant herniation of intraocular contents.[4-6] Globe lacerations result from sharp trauma (usually) due to the direct impact on the eyewall (force from outside inward).[4,5] Perforating injury is a specific type of open globe injury in which the projectile or sharp object has caused an entry as well as an exit full-thickness eyewall wound.[4,5] In a laceration (also sometimes referred to as a *penetrating injury*), only a single full-thickness eyewall wound is present per projectile/object (there is no exit wound).[4,5] Finally, an intraocular foreign body (IOFB) is a type of laceration in which the offending foreign object is retained within the globe.[4-6]

The above classification is not simply an academic exercise. It provides an effective means of communication between treating physicians, and the type of injury has critical implications to management and visual prognosis.[4-6] For example, an open globe injury needs urgent operative repair, whereas a closed globe injury typically does not. Among open globes, globe rupture portends a poorer prognosis for final visual outcome than globe laceration.[7,8] Finally, an IOFB is usually best removed by a vitreoretinal surgeon and may require vitrectomy (sometimes not available in general ophthalmology operating rooms), whereas a simple laceration or perforating injury can be managed by any ophthalmologist in an operating room with an ophthalmic operating microscope. Certainly, in needed circumstances, primary closure can be achieved and the patient can be referred to a retina specialist for removal of an IOFB at a second procedure.

CLINICAL APPROACH TO EYE TRAUMA

It is imperative that concomitant nonocular injuries be evaluated and assessed on presentation to the emergency department. Involvement of the ophthalmologist in a timely manner and in the absence of life-threatening injury before transfer to the operating room is important. Sight-threatening injury needs to be recognized and treated within an appropriate time interval.

History

Every effort should be made to take a focused history—if not from the patient (if he or she is unconscious, distracted by other severe injuries, or under the influence of alcohol or drugs), then from relatives, bystanders, or first responders. In the setting of trauma, being time efficient is obviously of the utmost importance.

The most important aspect of the history is the mechanism of injury, as specific mechanisms suggest specific injuries that must be assessed for and treated. For example, hammering is associated with intraocular metallic foreign bodies, whereas fireworks injury is commonly associated with chemical

injury that must be treated emergently (as well as contusion injury—rarely open globe). Injury to the forehead as a result of a bicycle accident followed by loss of consciousness is a common scenario in which traumatic optic neuropathy may develop, whereas injuries from BB guns are associated with globe lacerations with particularly poor prognosis. Additionally, it is important to elucidate the setting of the injury: lacerations in a rural setting are associated with higher rates of endophthalmitis. Documenting whether protective eyewear was worn at the time of the injury is important for medicolegal reasons.

Patient symptoms are also important: floaters and a visual field defect are highly suggestive of a retinal detachment, whereas pain with sensitivity to light without compromise in the vision suggests a traumatic iritis (although a globe laceration and even an IOFB remain a possibility).

Ocular history is important for two reasons. First, it may modify the effects of trauma, for example, in the case of a patient who has previously had a corneal transplant, an open globe due to graft dehiscence will occur with much less force than normally expected. Similarly, in patients with previous cataract, glaucoma, or radial keratotomy surgery, the globe ruptures at the site of the previous wound. Second, ocular history is important because preexisting pathology may dictate different treatment decisions following trauma. For example, the threshold for surgical evacuation of a hyphema would be much lower in a patient with advanced glaucomatous optic neuropathy than in a patient with healthy optic nerves.

Past medical history is similarly important because it can modify treatment decisions. For example, hyphema is managed differently in patients with sickle cell disease. Another example would be patients with pseudoxanthoma elasticum who invariably have angioid streaks and have a much higher risk of choroidal rupture. There are also several systemic conditions that result in eye conditions unrelated to trauma, an obvious example being diabetes mellitus causing diabetic retinopathy that can cause nontraumatic vitreous hemorrhage.

An important aspect of the drug history is whether the patient is on anticoagulants or antiplatelet agents as this will complicate operative repair. Additionally, determining allergies to medications is critical.

Review of systems must assess for the patient's ability to survive anesthesia and surgical repair. Patients who cannot undergo surgery safely may be better managed medically even though the risk of losing their sight in one eye is inevitable rather than dying from complications of anesthesia.

Ocular Anatomy

Understanding the anatomy of the eye is critical in diagnosing ocular trauma. The eye sits inside the bony orbit with six attached extraocular muscles. The extraocular muscles are attached directly to the sclera. Overlying the sclera is a thin clear membrane called the conjunctiva, which terminates at the cornea. The cornea is the clear, most anterior surface of the eye that overlies the iris and, more posteriorly, the lens. The anterior chamber exists between the cornea and iris and is filled with aqueous fluid. The vitreous cavity separates the anterior segment from the retina. The retina contains light-sensitive photoreceptors, which transmit signal to the optic nerve. See Figure 23-2.

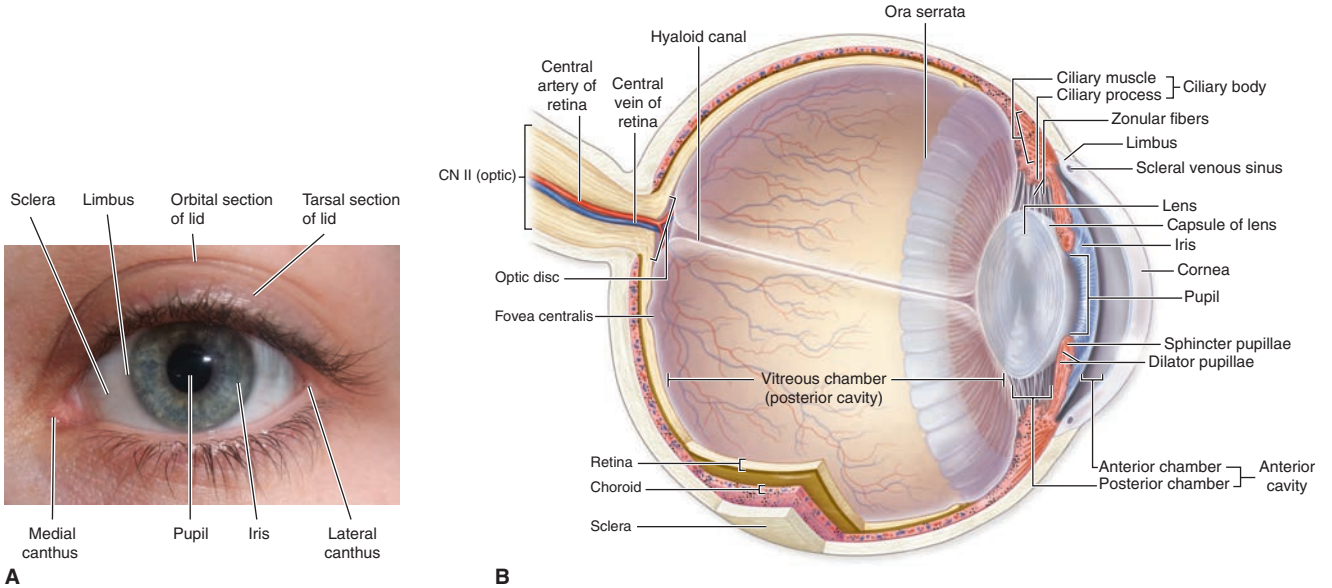

FIGURE 23-2 Anatomy of the eye. (***A:*** Reproduced with permission from Riordan-Eva P. Anatomy & embryology of the eye. In: Riordan-Eva P, Augsburger JJ, eds. *Vaughan & Asbury's General Ophthalmology.* 19th ed. New York, NY: McGraw Hill; 2018:15. ***B:*** Reproduced with permission from McKinley MP, O'Loughlin VD. *Human Anatomy*. 3rd ed. New York, NY: McGraw Hill; 2012.)

Clinical Examination

In the blunt multisystem trauma patient, the presence of periocular facial injuries or, when able to be provided, complaints related to vision should prompt a screening ophthalmic evaluation. This will help to determine if an ophthalmology consultation is needed and, if so, how urgently. A screening ophthalmic examination should include visual acuity (each eye tested separately), pupillary examination, motility, measurement of intraocular pressure, and a penlight exam of the external eye. Clinical examination can be challenging due to pain or poor patient cooperation due to the influence of alcohol, drugs, or severe eyelid swelling, yet it is essential for proper diagnosis and management of ophthalmic trauma. The basic tool kit needed for rudimentary eye examination includes penlight, near vision card, topical anesthetic, fluorescein strip, and eye wash.

VISUAL ACUITY

Every ophthalmologic exam should begin with an assessment of visual acuity if the patient's clinical status allows. It is no exaggeration to state that failure to document the visual acuity is inexcusable and akin to failure to document the pulse! Measuring visual acuity is relatively easy.

Each eye is tested separately by having the patient read from a near card from one eye while the other eye is covered. The best means of covering the nontested eye is with an occluder, but the patient's hand or a 4×4 will suffice. The smallest line that the patient can read is what should be documented. The goal is to determine whether the patient has no light perception (NLP—cannot even see the light from a strong pen torch right in front of the injured eye with the room darkened), light perception only (LPO—can see the light but no hand movements), hand motions only (HM—can see hand movements but cannot count fingers), or vision between 1/200 and 20/20. When trying to measure vision between 1/200 and 20/20, the patient should be wearing his or her spectacles (if these are available). If a formal vision testing card is not available, documentation of ability to read the newspaper title (approximately 20/200) or normal magazine print (approximately 20/40) is still extremely helpful. In a blunt multisystem trauma patient, documentation of visual acuity is not always possible. In this scenario, it should be noted in the chart why the vision could not be assessed.

When possible, measuring visual acuity is crucial for three reasons. First, a specific level of vision prompts the examining physician to search for a diagnosis explaining it. For example, HM vision is not explained by a subconjunctival hemorrhage and requires the examiner to carefully examine for the other signs of a scleral rupture. Another example would be the patient who had trauma 3 days previously and presents to the emergency department with photosensitivity, mild lid edema, and vision of 20/400: before knowing this level of vision, traumatic iritis could have been contemplated, but with vision of 20/400, endophthalmitis with a self-sealed corneal or scleral laceration becomes a strong possibility.

The second reason it is important to measure the visual acuity is to document a baseline so that later in the course it can be established whether there is improvement or deterioration. For example, a patient with a vitreous hemorrhage and vision of HM is seen by a vitreoretinal surgeon for examination to rule out retinal tears and detachment; if a week later the vision is 20/200 (and there are no retinal tears or detachment), further observation is reasonable as it appears that the vitreous hemorrhage is spontaneously resolving. In contrast, if a week later the vision is light perception, this suggests that a retinal detachment has occurred due to an undetected retinal tear.

The final reason why it is important to measure visual acuity is that visual acuity at presentation is a strong predictor for final visual outcome.[7,8] Therefore, having an initial visual acuity is essential if discussing the prognosis of the injury with the patient.

PUPILLARY EXAMINATION (SHAPE, REACTION, AND RELATIVE AFFERENT PUPILLARY DEFECT)

After visual acuity, the pupillary exam is a critical component of the physical examination in ocular trauma. A bright, handheld light can be used to examine the pupils as the patient looks off into the distance. The size in millimeters and shape of each pupil should be noted. The pupils should constrict briskly and equally after application of bright light. Any discrepancy between the size and shape of the pupils between the two eyes should alert the clinician for possible ocular injury. The pupil may be peaked if the iris is sealing (plugging) a corneal or anterior scleral laceration. The pupil may also be irregular if there has been injury to the iris sphincter muscle (typically a result of blunt trauma, commonly associated with hyphema).

The pupil will be dilated and not react to light if there is compression or damage to the third cranial nerve intracranially (following head trauma); if this is suspected, urgent neurosurgical consultation and computed tomography (CT) imaging is required. Additionally, an orbital compartment syndrome (due to retrobulbar hemorrhage or any cause for swelling within the orbit) may cause compression of the third nerve (and all the other nerves) and result in a fixed dilated pupil. Finally, if there has been damage or ischemia of the iris sphincter (very elevated intraocular pressure or torn iris sphincter), the pupil will not react to light.

A relative afferent pupillary defect (RAPD) is important to document for two reasons. First, its presence means that there is injury to the retina or optic nerve. This is important as it prompts the examiner to carefully consider the diagnoses that may be affecting the retina and optic nerve and not satisfy himself or herself with a diagnosis involving the anterior segment only. Second, the absence of an RAPD is a strong predictor of visual survival, with only 97% of eyes without an RAPD maintaining some vision.[9]

RAPD is measured by alternately shining a strong light into each eye. At least 2 seconds should be spent shining each eye with 1 second in transit. When the pupils dilate when the

light is shone into one of the eyes, it is said that an RAPD is present in that eye. It is important to note that it is the first movement of the pupil when the light is shone into it that matters. From the above, it should be obvious that, even in a patient with a pupil that is immobile in the injured eye, determination of the presence or absence of an RAPD in that eye is possible since the contralateral pupil movement can be observed while shining the light into the injured eye.

MOTILITY

Examination of motility should be performed in all patients in need of a screening ophthalmic examination. In a normal state, the two eyes will move in unison fully in all directions. If the motility of one eye is limited, the patient will experience double vision when looking in the direction of the limitation. This is because the eyes will become misaligned.

Examination of motility is performed by asking the patient to follow an extended second digit or pen in all directions of gaze. It is important to ascertain whether the patient has diplopia when looking at any of these directions, as this indicates that the two eyes are not moving in synchrony.

EXTERNAL AND OCULAR ADNEXAL EXAMINATION

Examination of the ocular adnexa involves looking at the eyelid position with eyes both open and closed, contour, and evidence of laceration. It is important also to evaluate for proptosis, and in patients with proptosis, testing for resistance to retropulsion may point toward elevated intraocular pressure and congested orbital compartment. After the integrity of the globe has been established, the clinician may apply gentle digital pressure to the closed eyelids. Any resistance with digital pressure and asymmetry between the two eyes may indicate a retrobulbar hemorrhage. As part of the routine external examination, the examiner should palpate the orbital rim for "step-off" in cases of suspected orbital fractures. In cases of suspected orbital floor fractures, testing sensation along the distribution of cranial nerve V on either cheek can be an early sign.

ANTERIOR SEGMENT EXAMINATION

The slit lamp biomicroscope is the ideal instrument to examine the anterior segment. Portable versions exist for patients who cannot sit up to be examined with the regular slit lamp. If not even a portable slit lamp is available, a direct ophthalmoscope offers high magnification and can be used, and if even this is unavailable, a penlight with a blue filter and a magnifying lens can be used.

Throughout the examination of the anterior segment, it is important to remember that pressure should not be exerted on the globe (as it may be open and it is uncomfortable to the patient); rather, the lids should be lifted and held up by applying pressure against the orbital rim.

Examination of the anterior segment starts with inspection of the conjunctiva and sclera. Subconjunctival hemorrhage is a common finding sometimes even after trivial trauma but can be a sign of an open globe; therefore, the other signs of an open globe should be sought. Additionally, a subconjunctival hemorrhage can be a sign of a retrobulbar hemorrhage, especially if its posterior margin cannot be defined; therefore, the other signs of this condition should also be sought. Uveal tissue, vitreous gel, or even retina is sometimes evident on or under the conjunctiva in cases of scleral rupture or laceration.

Inspection of the cornea should be performed, actively searching for a corneal laceration, a corneal foreign body, a corneal abrasion, and a corneal concussive injury to the endothelium (appears as opacity on the endothelium). A corneal abrasion may be more easily seen by applying fluorescein drops or a fluorescein strip in the tear lake and using the cobalt blue filter.

Examination of the anterior chamber should be performed looking for hyphema, hypopyon (layering of white cells inferiorly diagnostic of endophthalmitis in the setting of trauma), a shallow anterior chamber suggestive of open globe, an anterior chamber foreign body, and anterior chamber cell (white cells in the anterior chamber—are seen in endophthalmitis or traumatic iritis). Examination of the iris should be performed looking for iris tears or iris dialyses.

Finally, examination of the lens should be performed to determine whether it is present or not (it may have been lost in the case of a corneal laceration with extrusion of ocular contents or in the case of rupture at the site of prior cataract surgery with extrusion of the intraocular implant), whether it is subluxed, whether there is an intralenticular foreign body, or whether cataractous changes have developed.

INTRAOCULAR PRESSURE

There is no need to check the intraocular pressure if the globe is obviously open, but if not, measurement of the intraocular pressure is mandatory. Several methods are available for measuring intraocular pressure. The gold standard is the Goldmann applanation device used with the slit lamp, but a Tono-Pen is a convenient device for use in the emergency department setting. To measure the intraocular pressure, a drop of topical anesthetic is administered to each eye. The handheld tonometer can then be applied to the corneal surface repeatedly to obtain a pressure reading. Take care not to press on the eye when performing this exam element, as it will yield a falsely elevated reading.

A high pressure can be seen with hyphema or with a retrobulbar hemorrhage (due to transmission of the elevated intraorbital pressure), whereas a low pressure is seen with an open globe or severe intraocular inflammation. It should be noted, however, that the intraocular pressure may, on occasion, be normal (rarely high) with an open globe.

DILATED FUNDOSCOPY

Dilated fundoscopy is best performed using indirect ophthalmoscopy, a skill beyond the remit of a trauma surgeon. This will be performed by your consulting ophthalmologist.

Ancillary Studies

B-mode ultrasonography is very useful for examination of the posterior segment in the presence of media opacities not allowing ophthalmoscopy. Retinal tears, detachments, and IOFBs can be detected. It should be noted that the investigation is strongly operator dependent and that, even in experienced hands, severe vitreous hemorrhage cannot be reliably distinguished from a retinal detachment.[10]

CT imaging is important in evaluating for orbital fractures, orbital foreign bodies, and IOFBs, especially metallic. An orbital CT scan with thin slices should be ordered. Note that the dimensions of foreign bodies are commonly exaggerated on CT images.[10] It should also be noted that vegetable matter (eg, wood) in the orbit is not well imaged by CT.

Initial Management of the Patient with Ocular Trauma

After the patient is stabilized (ie, life-threatening injuries have been stabilized), other organ-threatening injuries need to be managed in parallel to evaluating the injured eye. The following are priorities when managing the injured eye:

1. Rule out a chemical injury by history (splash of liquid into the eye, explosion at chemical facility, firework injury). If there is suspicion of a chemical injury, a pH strip should be checked (from the fornix) and irrigation should be started at once (see Chemical Injury section).
2. Rule out an open globe if possible: look for the specific signs (corneal/scleral laceration, prolapse of uveal tissue, hemorrhagic chemosis of the conjunctiva, low intraocular pressure, asymmetry in anterior chamber depth, vitreous hemorrhage). If there is reasonable suspicion of an open globe, exploration in the operating room should still be carried out (eg, appendectomy; while one endeavors to reduce the rate of negative exploration, it is better to have a negative exploration than to miss the diagnosis). If there is an open globe or an open globe is suspected:
   a. The patient needs urgent (as soon as possible and certainly within 12 hours) repair in the operating room by an ophthalmologist, and the necessary arrangements need to be made (this may include transfer to a center with an operating microscope and available ophthalmologist, ophthalmology consult, etc). The patient should be made NPO (nothing by mouth), and appropriate fluids for their systemic status should be given.
   b. If there is any suspicion that the globe could be ruptured or a rupture is confirmed, the eye should be shielded. If there is ever any doubt, the eye should be shielded until a formal ophthalmic evaluation can occur. Proper placement of the shield is such that the edges of the shield contact the bony orbital rim while the shield vaults over the soft tissues of the eyelids and eye. Patients should be instructed not to squeeze their lids or strain because this may cause further extrusion of intraocular contents. If a metal shield is not available, a cut Styrofoam cup may be taped over the eye.
   c. Order a CT scan of the orbits to rule out an IOFB. This should be performed for all open globe injuries.
   d. Administer tetanus toxoid.
   e. An intravenous fluoroquinolone antibiotic (moxifloxacin, levofloxacin, or ciprofloxacin) should be administered.
   f. Repair of lid lacerations or orbital fractures should *never* be undertaken before an open globe has been ruled out or repaired.
3. Identify other orbital or ocular injuries and treat accordingly.

OCULAR IMAGING

Various imaging modalities can be used to provide further information to supplement the clinical examination. Typically, B-scan (brightness-amplitude scan) ultrasonography is performed when there is no clear view to the posterior segment of the eye on dilated examination. This typically occurs as a result of a media opacity located anywhere along the visual axis. For example, if corneal scarring, significant hyphema or hypopyon, lens opacity, or vitreous hemorrhage occurs, it may be impossible to adequately assess the status of the retina. B-scan ultrasonography uses 10 MHz to provide high-resolution images with low penetration to determine if lens dislocation is present or retinal detachment has occurred. If a retinal detachment has occurred, it will be seen as a thick, hyperechoic, billowing sheet just anterior to a fluid-filled hypoechoic space. Typically, this specialized form of ultrasound will not be available to the trauma surgeon, and the decision about whether or not B-scan is necessary will be made by your consultant ophthalmologist.

In general, CT is the imaging modality of choice in the acute setting of ocular trauma. As mentioned earlier, a CT of the orbits should obtained in any open globe injury to rule out an IOFB. Since this modality provides excellent visualization of osseous structures, it is exceedingly useful in evaluating fractures around the orbit. As a reminder, entrapment in the setting of orbital fracture is a clinical diagnosis based on multiple exam findings including extraocular motility restriction, diplopia, or observation of oculocardiac reflex.

Magnetic resonance imaging (MRI) is not routinely used for the initial evaluation of the trauma patient. Orbital fractures, IOFBs, and retrobulbar hemorrhage can be identified quickly and effectively with CT scan. If a patient requires an MRI, it is important to first rule out an intraocular metallic foreign body with CT scan. If optic neuropathy persists and is suspected secondary to trauma, an MRI may be ordered to further evaluate the optic nerve after the initial injuries are stabilized.

WHEN TO CONSULT OPHTHALMOLOGY

The decision to consult ophthalmology will be provider dependent, depending on the comfort level and knowledge of the trauma physician in dealing with the current ocular

issue. The following list of ophthalmic issues, while not all-encompassing, should prompt a consult to ophthalmology:

1. Suspected penetrating injury to the globe necessitating exploration
2. Orbital fracture with suspicion for extraocular muscle entrapment
3. Eyelid laceration, especially when involving the margin or when medial to the puncta
4. Suspicion for retrobulbar hemorrhage resulting in orbital compartment syndrome
5. Suspected retinal detachment
6. Marked decrease in vision from patient's baseline despite use of corrective lenses

PROGNOSIS OF EYE TRAUMA

Prognosis of eye trauma involves discussion of three entities: whether the patient is going to retain his or her globe, what the patient may expect his or her vision to be in the long term, and finally, whether this will affect the uninjured eye (see later discussion on sympathetic ophthalmia).

Whether a patient is going to retain his or her globe depends on the specifics of the traumatic injury. It is rare that enucleation will be required for an eye sustaining an injury other than an open globe. Primary enucleation is rare (0.17% of open globes) and reserved for eyes where the sclera and cornea have been injured so severely that they cannot be sutured back together (usually due to a blast injury where the eye has been blown away or a gunshot injury directly to the eye).[11] Secondary enucleation (reported in 6%–20% of open globes) is much more common for ruptures than lacerations and is usually performed for a blind (NLP), painful eye.[7,9,11,12] An RAPD, NLP or LPO, visible uveal tissue, and concomitant eyelid laceration at presentation are risk factors for enucleation.[7,11,12] Enucleation to prevent sympathetic ophthalmia is also sometimes performed, although it is controversial (see later).

The best system that predicts long-term visual outcome (after appropriate management including surgical treatment) is the ocular trauma score (OTS).[8,13] In the OTS, a functional outcome (initial visual acuity) and five signs or diagnoses (rupture, endophthalmitis, RAPD, retinal detachment, and perforating injury) are used to estimate the likely visual outcome (Table 23-1).[8]

Many patients worry that a poorly seeing eye will cause "straining" of the other eye—this is unequivocally not true. However, the uninjured eye may develop sympathetic ophthalmia, a rare (incidence 0.03/100,000 per year),[14] bilateral uveitis that may occur 2 weeks to 50 years usually following eye trauma or surgery.[15,16] While originally described as a consequence of trauma, currently it is more common following eye surgery.[14] This is a consequence of improved management of ocular trauma, including prompt primary repair. Indeed, most cases with sympathetic ophthalmia following eye trauma present to the ophthalmologist several weeks after the initial trauma when the vision in the second eye is affected.[17] Tellingly, since World War II, there had been no cases of sympathetic ophthalmia reported in any military conflict until a single case was reported in the recent war in Iraq.[15] With current treatments, eyes affected with sympathetic ophthalmia commonly maintain functional vision, with the majority maintaining reading vision.[18] While removing the injured eye (when the vision is NLP) may decrease the rates of sympathetic ophthalmia, this is quite controversial given that sympathetic ophthalmia is rare (especially with appropriate management of the injured eye) and treatable.

TABLE 23-1: Determination of Ocular Trauma Score (OTS)

Determination of OTS	Variable	Raw score
Vision	No light perception (NLP)	60
	Light perception only (LPO) or hand motions only (HM)	70
	1/200 to 19/200	80
	20/200 to 20/50	90
	≥20/40	100
Diagnoses	Globe rupture	–23
	Endophthalmitis	–17
	Perforating injury	–14
	Retinal detachment	–11
Sign	Relative afferent pupillary defect	–10

Probability of a Given Visual Outcome Given OTS Score

Score	OTS	NLP (%)	LPO/HM (%)	1/200 to 19/200 (%)	20/200 to 20/50 (%)	≥20/40 (%)
0–44	1	74	15	7	3	1
45–65	2	27	26	18	15	15
66–80	3	2	11	15	31	41
81–91	4	1	2	3	22	73

CHEMICAL INJURY

Chemical injuries to the eye are true ocular emergencies, and time is of essence when treating acute chemical exposure. They represent 7.7% to 18% of ocular trauma.[19-21] Immediate and copious irrigation is vital to limiting the extent of damage to the ocular surface. Alkaline agents tend to penetrate the eye more rapidly due to saponification of cell membranes and lead to liquefactive necrosis. Acidic agents cause coagulative necrosis with protein precipitation within the tissue; thus, acidic injuries tend to cause less severe injury compared to alkali agents due to less penetrative damaging effects.[22] The nature of the toxic agent should be identified and brought into the emergency center if possible so that pH can be tested.

Following toxic chemical exposure to the ocular surface, irrigation should begin immediately with water, saline, or any commercially available eyewash with a neutral pH, and continued if possible while en route to the nearest emergency center. On arrival to the emergency center, an initial pH should be taken by placing pH testing paper in the inferior fornix. Irrigation should continue until the measured pH is neutral (7.2–7.4) for at least 5 minutes after irrigation has stopped. It is important to note that irrigation can last up to an hour or more depending on the severity of the splash injury in order for the eye's pH to normalize.

Irrigation can be performed by directly pouring saline from intravenous tubing to the surface of the eye. Placing one drop of topical ophthalmic anesthetic such as proparacaine may help the patient to keep the affected eye open. Caution should be exercised when placing irrigation lenses such as a Morgan lens since retained particulate matter or foreign body can be trapped in the fornices of the eye. If an irrigation lens is to be used, the superior eyelid should be everted to look for embedded foreign body, and both the superior and inferior fornices should be swept clean with a moist cotton swab to remove any particulate matter.

Chemical injuries are classified using the Dua classification system (Table 23-2), which reflects the amount of limbal involvement and the percentage of conjunctival involvement. Corneal epithelial defects can be easily detected using topical fluorescein staining, such as a moistened fluorescein strip or manufactured combination of fluorescein and topical anesthetic eye drops. Limbal ischemia appears as blanching of normal conjunctival and limbus blood vessels. Hyperemia in the setting of chemical injury presents better prognosis than a white eye.

The goals of treatment for chemical ocular injury are to stop ongoing tissue degradation, promote reepithelialization of the surface, minimize inflammation, and prevent infection. For grade I damage, the patient can be treated with an antibiotic (eg, erythromycin) or antibiotic/steroid mixed combination eye ointment (eg, dexamethasone/polymyxin/neomycin) four times a day to the affected eye and a topical cycloplegic agent (eg, atropine) to decrease ciliary spasm and decrease formation of posterior synechiae.[22] For grade II, topical steroid eye drops may need to be added to the regimen to decrease the inflammatory response for the first 1 to 2 weeks after injury. In grades III and IV, high-dose vitamin C, 10 ascorbate eye drops, and 10% citrate eye drops have been associated with more rapid recovery and better vision.[23] Oral doxycycline is a collagenase inhibitor and may reduce the risk of corneal thinning and perforation in severely burned eyes.[24,25]

Consultation with an ophthalmologist is necessary for follow-up and ensuring that the treatment regimen is leading to clinical improvement. Rarely is immediate surgical intervention needed in chemical injury patients.

MECHANICAL INJURY

Subconjunctival Hemorrhage

Subconjunctival hemorrhage is a very common condition that presents as an ocular emergency. Clinically, subconjunctival hemorrhages appear as flat, bright red blood noted under the bulbar conjunctiva (Fig. 23-3). It can be alarming in appearance, and although severity can be variable, in general it is rather benign and poses no threat to vision.

TABLE 23-2: Dua Classification for Ocular Surface Burns

Grade	Prognosis	Clock hours of limbal involvement	Conjunctival involvement	Analog scale*
I	Very good	0	0%	0/0%
II	Good	≤3	<30%	0.1–3/1–29.9%
III	Good	>3–6	>30–50%	3.1–6%/31–50%
IV	Good to guarded	>6–9	>50–75%	6.1–9/51–75%
V	Guarded to poor	>9–<12	75–<100%	9.1–11.9/75.1–99.9%
VI	Very poor	12	100%	12/100%

*The analog scale records accurately the limbal involvement in clock hours of affected limbus/percentage of conjunctival involvement. While calculating percentage of conjunctival involvement, only involvement of bulbar conjunctiva, up to and including the conjunctival fornices, is considered.

Source: Reproduced from Dua HS, Kin AJ, Joseph A. A new classification of ocular surface burns. *Br J Ophthalmol.* 2001;85(11):1379-1383. With permission from BMJ Publishing Group Ltd.

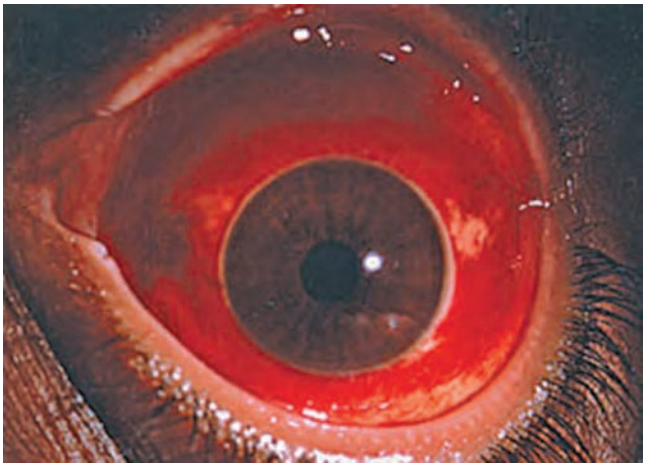

FIGURE 23-3 Subconjunctival hemorrhage.

Spontaneous subconjunctival hemorrhage can be due to Valsalva maneuvers, coughing, sneezing, vomiting, or heavy lifting. Minor trauma such as excessive eye rubbing can also cause subconjunctival hemorrhages. Often, no specific etiology can be found. When subconjunctival hemorrhage is noted with other signs of facial or ocular trauma, one must rule out occult globe injury. Obtaining a good history is vitally important to determining if further workup is needed. A history of blunt trauma may present with subconjunctival hemorrhage, but the patient may also have orbital fractures that need to be evaluated.

Patients who present with complete 360° of subconjunctival hemorrhage from blunt trauma should be examined by an ophthalmologist to rule out open globe injury. Clues that may indicate occult open globe injury include peaked pupil, asymmetric anterior chamber depth, asymmetrically low intraocular pressure, and subconjunctival pigment.

In cases of isolated subconjunctival hemorrhage, no treatment is needed. The hemorrhage will usually resolve spontaneously in a few weeks. Patients need to be informed that the hemorrhage will change color over the next few days and may expand as the bruising process evolves. These patients typically do not require ophthalmic follow-up.

Conjunctival Lacerations

Conjunctival lacerations may present in isolation or in combination with damage to deeper layers of the eyewall and the sclera. Isolated conjunctival lacerations do not require surgical repair unless they are large (eg, >2 cm) or lie over an extraocular muscle insertion. Often it is difficult to assess if the sclera is involved without manipulation using a cotton tip swab to gently push away the conjunctiva exploring the scleral wall beneath. For large conjunctival lacerations or those that may involve the sclera, ophthalmic consultation is warranted.

Scleral penetration can be associated with vitreous hemorrhage, and if the scleral defect is large enough, vitreous prolapse can be seen as well. If there is vitreous hemorrhage, the patient's vision may be compromised. It is important to not engage the vitreous prolapsed through a scleral defect since traction on the vitreous strands can lead to retinal tears, leading to rhegmatogenous retinal detachments.

CORNEAL ABRASION

Patients with corneal abrasions typically present with intense pain and photophobia. Trauma to the cornea from a fingernail, paper cut, thrown objects, and contusive injury (eg, air bag) can result in the superficial corneal epithelium being stripped away from the underlying stroma. Simple corneal abrasions can be one of the most painful injuries that patients experience because beneath the corneal epithelium lies an extensive plexus of sensory nerves from the ophthalmic division of the trigeminal nerve, and when they become exposed, severe pain results.

Corneal abrasions can be diagnosed clinically when topical fluorescein dye is taken up by the area devoid of epithelium and turns bright green viewed with a cobalt blue light. The size and location of the abrasion can be documented using a circle to represent the cornea. Traditional teaching advocated patching for corneal abrasions in the past. Currently, evidence shows that patients heal faster without patching, and also with patching there is no benefit with regard to pain reduction. Small corneal abrasions without concomitant ocular injury can be managed and treated with antibiotic ophthalmic ointment, topical cycloplegic agent, and topical ophthalmic nonsteroidal anti-inflammatory drugs (eg, ketorolac or diclofenac). Although there is no good evidence that topical ophthalmic antibiotic is indicated in cases where there was no recent history of contact lens wear or injury with organic material, use of a topical ophthalmic antibiotic is not unreasonable given the devastating sequelae of corneal infection and scarring.[26-33]

History of contact lens wear or injury with organic material raises the risk of infection. These patients should never be patched given the elevated risk of infection and should all be referred for prompt ophthalmic consultation. Patients with large corneal abrasions may benefit from bandage soft contact lens placed by an eye care professional for comfort. If the affected eye is patched, the patient should follow up the next day with an eye care professional to monitor healing and assess for early signs of infection.

CORNEAL FOREIGN BODIES

Corneal foreign bodies are one of the most common forms of ocular trauma, presenting second in frequency only to corneal abrasions in emergency centers.[34] Most patients present with small superficial corneal foreign body with good or mildly affected vision. Individuals can have debris blown into the eye while walking outdoors or while performing high-risk activities such as grinding, drilling, hammering, and using a leaf blower. There are many causes for corneal foreign body, but lack of protective eyewear contributes to increased risk.

Specific questions regarding hammering metal on metal or grinding metal need to be asked, and a detailed history

regarding the exact mechanism of injury helps to highlight patients at increased risk for penetrating ocular injury or IOFB. An initial examination should include looking at the corneal surface with magnification; if slit lamp is not readily available, surgical loupes can offer a better examination than the naked eye. Often a superficial corneal foreign body will be obvious, but it is important to evert and inspect under the upper eyelid and to look in the inferior fornix as well.

After a penetrating injury has been ruled out, superficial corneal foreign bodies can sometimes be easily removed with a moist cotton swab. Instill a topical ophthalmic anesthetic and moisten a sterile cotton swab with anesthetic, and then gently roll across the surface of the cornea and the foreign body may stick to the cotton tip. Irrigation with eyewash can also be used to loosen and remove the foreign body. If these maneuvers fail, ophthalmic consultation should be considered. Those foreign bodies that are moderately embedded in the anterior one-third of the cornea can be removed at the slit lamp with a tuberculin syringe or 25-gauge needle. Care must be exercised to not go too deep into the corneal tissue. The average central corneal thickness measures only 550 µm.

After removal of the corneal foreign body, start treatment as a corneal abrasion with ophthalmic ointment, cycloplegic agent, and topical antibiotic. Follow up with eye care provider to assess if further debridement is indicated and to look for early signs of infection.

CORNEAL LACERATIONS

Corneal rupture is unusual unless the patient has had previous penetrating keratoplasty (corneal graft) or radial keratotomy. In the former case, dehiscence at the junction of the graft with the host cornea is common. In the latter, rupture occurs along the keratotomy as the cornea is very thin in that location. Loss of the crystalline lens or intraocular implant through the rupture site is sometimes observed. Management is by emergent operative repair, by closing the rupture site with 10-0 nylon sutures.

Corneal and/or scleral lacerations have better prognosis than ruptures, with about 50% retaining vision of 20/40 or better (driving vision).[35,36] Scleral/corneal lacerations limited in location to being anterior to the insertion of the recti muscles and those having a length of greater than 5 mm are associated with a better prognosis.[37] Endophthalmitis is a major concern following penetrating injuries. An IOFB also needs to be ruled out.

A corneal laceration is evident on examination with a slit lamp biomicroscope, although it can be usually seen by oblique illumination with a penlight. It is important to realize that smaller corneal lacerations can self-seal or be plugged by the iris (which can result in a peaked pupil), allowing normal vision and a formed anterior chamber with normal or near-normal intraocular pressure. It is critically important to realize that eyes that have sustained a self-sealing laceration are still at risk of endophthalmitis, particularly if there is an IOFB, and are at risk for further extrusion of intraocular contents or low intraocular pressure if the patient applies pressure immediately behind the laceration (eg, by rubbing the eye). Sadly, we have seen several eyes with undiagnosed self-sealed lacerations that developed endophthalmitis that was erroneously treated as traumatic iritis with steroids by unsuspecting emergency department physicians or pediatricians (with disastrous effects for the eye).

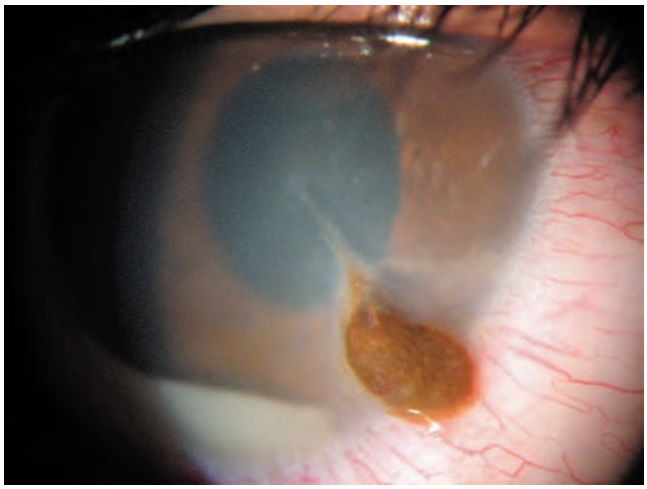

FIGURE 23-4 Corneal laceration with protrusion of iris.

Corneal lacerations can be full thickness or partial thickness (lamellar laceration). A full-thickness laceration is classified under the general diagnosis of open globe. With lamellar or partial-thickness lacerations, the globe is considered closed. The diagnosis of a full-thickness corneal laceration is often easy to recognize when there is protruding iris or uveal tissue (Fig. 23-4). In these cases, the anterior chamber may also be shallow or flat, which can be seen when shining a penlight from the lateral or temporal side of the eye.

Some corneal lacerations may be combined and encompass the limbus and extend into sclera (ie, corneal–scleral laceration). As with all open globe injuries, extreme care must be taken to prevent any direct pressure on the eye that may result in extrusion of intraocular contents. If corneal laceration is suspected, a metal shield (ie, Fox shield) should be placed over the affected eye. The goal is to prevent any direct pressure or contact on the globe; soft eye patches should be avoided.

For partial-thickness lacerations, depending on the size and depth of the laceration, simple observation with topical antibiotic prophylaxis up to suturing the partial-thickness flap may be warranted. Due to the expertise needed to evaluate the depth of the laceration with a slit lamp, ophthalmic consultation should be sought in corneal lacerations.

SCLERA LACERATION OR RUPTURE

Scleral rupture occurs in about 3.5% of eyes with severe blunt trauma.[38] It most commonly involves the sclera immediately posterior to the recti muscle insertion (about 6–7 mm posterior to the limbus) as the sclera is thinnest at that location; in patients who have had previous surgery involving the sclera

(most commonly, a glaucoma filtration surgery), rupture at the site of the previous scleral wound may also occur.

Scleral rupture is invariably accompanied by rupture of the highly vascular choroid or ciliary body. As a result, hemorrhagic chemosis, hyphema, vitreous hemorrhage, or a combination of these is invariably present.[38] Prolapsed uveal tissue appears dark brown or black, whereas prolapsed vitreous gel appears as a transparent or blood-tinged blob of gel; since the mechanism of scleral rupture is due to an extreme elevation of intraocular pressure at the moment of blunt impact, the force causing the rupture is from the inside. Prolapse of intraocular contents is therefore not uncommon, although it can be difficult to discern clinically because it may be covered by the hemorrhagic chemosis of the conjunctiva. Signs with the greatest specificity are a low intraocular pressure (<6 mm Hg), anterior chamber depth asymmetry (can be abnormally shallow or deep compared to fellow eye), and vision poorer than HM.[35,38]

Scleral rupture generally has poor outcomes, with only 30% to 35% of eyes regaining ambulatory vision (ie, vision that allows getting around without a guide).[36] Moreover, scleral rupture is a risk factor for enucleation, with up to 40% of eyes with scleral rupture being enucleated.[7,12,35,36] Particularly poor predictors are scleral rupture greater than 11 mm, golfball injury, and presenting vision of HM or worse.[12,35]

Treatment is by operative repair for scleral ruptures anterior to the equator that consists of excising necrotic uveal tissue, repositing viable uveal tissue and retina into the globe, and approximating the scleral edges using 8-0 nylon sutures. Posterior scleral ruptures are not accessible for repair (to access the posterior sclera, one would have to disinsert one or more of the recti muscles and pull to turn the eye, which would cause further extrusion of intraocular contents, making matters worse) and are allowed to heal by secondary intention.

SPECIAL SITUATION: TRAUMA IN LASIK PATIENTS

In 2007, more than 800,000 Americans underwent laser-assisted in situ keratomileusis (LASIK) vision correction surgery.[39] The first clinical trial for LASIK refractive surgery was performed in 1995, and it has been a growing surgical procedure for the past 10 years. To perform LASIK, a thin corneal flap is first made and folded back to allow for laser remodeling of the corneal stroma. The flap is most commonly hinged either superiorly or nasally and floated back into position once laser remodeling is completed without any need for suturing. Since this is commonly done as an outpatient procedure in an ophthalmologist's office or laser center, most patients do not consider this a significant part of their medical history.

It is important to recognize that both early and late traumatic flap dislocation and amputation have been reported in the literature. There are case reports of LASIK flap complications up to 7 years after surgery. Minor blunt traumas with fingernail or sports-related injuries have been the reported cause of flap dislocation and amputation.[40-42] Patients with LASIK flap dislocation and amputation will typically present with decreased vision and pain and a very similar history to simple traumatic corneal abrasion patients. In fact, a complete amputation of the LASIK flap can look very much like a large corneal abrasion with fluorescein staining since the corneal epithelium is lost with the flap.

LASIK flap dislocation and amputations are treated very differently than a simple traumatic corneal abrasion, and patients will need consultation with an ophthalmologist, preferably a corneal-refractive surgeon. Prognosis for early flap dislocation has generally been good, and with later flap dislocation, vision recovery is generally acceptable. For patients with flap amputation, prognosis is poor due to development of irregular astigmatism and corneal haze and scarring. Given the large number of patients who have undergone this very popular procedure over the past 15 years, it is crucial to always ask specifically regarding prior history of refractive or LASIK surgery.

Traumatic Hyphema

Hyphema refers to blood accumulating in the anterior chamber (Fig. 23-5). It can present even after minor trauma in patients with impaired coagulation either idiopathic or medically induced. The presentation includes pain, photophobia, and decreased vision, and on further examination with a penlight, a reddish pool of blood can be seen layered toward the inferior half of the anterior chamber due to gravity. If a patient has been recumbent due to gravity, the hyphema may be layered over the pupil precluding a clear view to the posterior segment.

The estimated incidence of hyphema is 17 per 100,000. The mean age of presentation is 25 years old with a higher prevalence in men than women. A hyphema does not rule out an open globe injury, and up to one-third of patients with traumatic hyphema can have concomitant open globe injury.[43] Patients can also present with eyelid injuries or orbital fractures concurrently with hyphema (Fig. 23-6).

Concussive injury causes equatorial expansion of the eyeball with resulting damage to the iris, ciliary body, and major

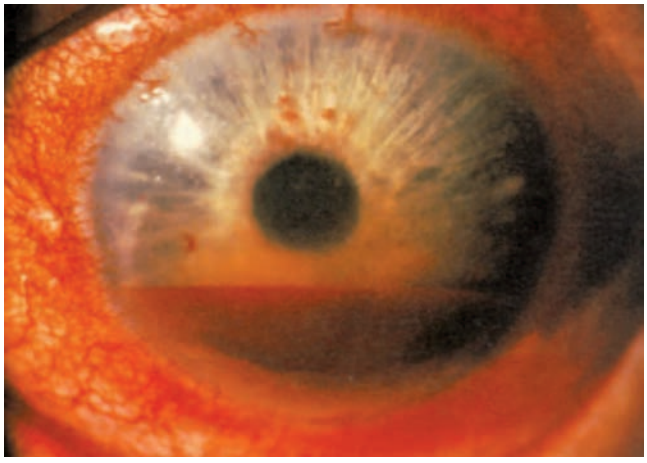

FIGURE 23-5 Hyphema.

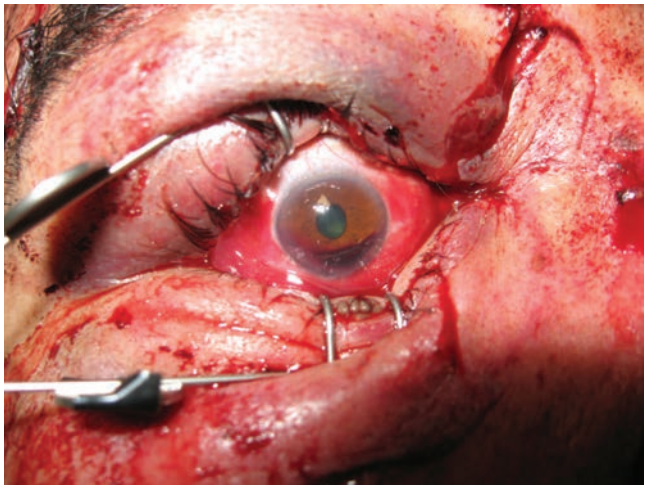

FIGURE 23-6 Hyphema and lid laceration.

arterial circle of the anterior segment. Depending on the level of force exerted, differing levels of bleeding can be seen. Some patients may only have a microhyphema, where a very small amount of red blood cells circulates in the anterior chamber but not enough blood is present to collect in the inferior part of the anterior chamber. These patients generally have very mild symptoms and can be managed similarly to traumatic iritis patients.

For patients who suffer more severe bleeding, the hyphema may completely fill the anterior chamber, also known as a total hyphema when filled with bright red blood. If the anterior chamber is filled with dark blood, this is referred to as an "eightball" hyphema. An eightball hyphema indicates lower oxygen concentration secondary to decreased aqueous circulation, and the patient is at greater risk of secondary angle closure glaucoma. Typically, the grading scale for hyphema is described by using an estimated percent layered in the anterior chamber. For example, a hyphema reaching mid-pupil level could be described as a 50% layered hyphema.

At the initial encounter, visual acuity, intraocular pressure, and grading of the hyphema severity should be documented. For patients of African American descent and others with known risk factors for hemoglobinopathies, a sickle cell prep or hemoglobin electrophoresis should be ordered along with other initial laboratory workup. This information is important for guiding targets for intraocular pressure management. When considering topical ophthalmic drops, β-blockers such as timolol are commonly used as first-line treatment when intraocular pressure is elevated. For individuals with sickling hemoglobinopathies, carbonic anhydrase inhibitors are contraindicated because the resultant metabolic acidosis can precipitate sickling. If those individuals have a sustained increase in intraocular pressure, other interventions, including surgery with anterior chamber washout, may be necessary.

For any patient with a layered hyphema regardless of percentage, an ophthalmic consultation is warranted since these patients will need close follow-up for the first 5 days after initial injury when the risk of rebleeding is highest. In addition, patients who suffer concussive injury great enough to cause a layered hyphema are likely to have concomitant injury to other intraocular structures and have an increased risk of traumatic glaucoma, resulting in permanent blindness in the future.

For patients who will need to be transferred for ophthalmic consultation, a clear or metal eye shield should be placed on the affected eye to protect from further injury. Depending on the interval until the patient is seen by an ophthalmologist, routine treatment for hyphema can be initiated with topical steroid eye drops (ie, prednisolone acetate 1% four times a day) and a topical cycloplegic agent (eg, cyclopentolate 1% or 2%, two or three times a day, or atropine 1% daily).

Traumatic Iritis, Mydriasis, Iris Sphincter Tears, and Iridodialysis

With minimal blunt trauma to the eye, patients may suffer a mild inflammatory reaction known as traumatic anterior uveitis or traumatic iritis. Patients generally present with mild blurry vision, eye pain, and light sensitivity or photophobia. The symptoms can be immediate or delayed for 24 to 48 hours, and examination often will reveal nothing more than mildly red eyes or conjunctival injection. However, if a slit lamp examination is done, an inflammatory reaction with circulating cells and flare can be seen in the anterior chamber.

Most cases will resolve within 1 to 2 weeks with topical steroid eye drop (1% prednisolone acetate, four times a day) and topical cycloplegic agent (1% or 2% cyclopentolate, twice a day, or 1% atropine, once a day). When prescribing cycloplegic agents, it is important to inform the patient that near objects will be blurry in the treated eye due to the loss of accommodation from pharmacologic dilation. In patients who have longstanding anterior uveitis from any etiology, posterior synechiae (iris-lens adhesions) can form. The use of a topical cycloplegic agent is not only for patient comfort, but also as a preventative measure against formation of posterior synechiae (Fig. 23-7).

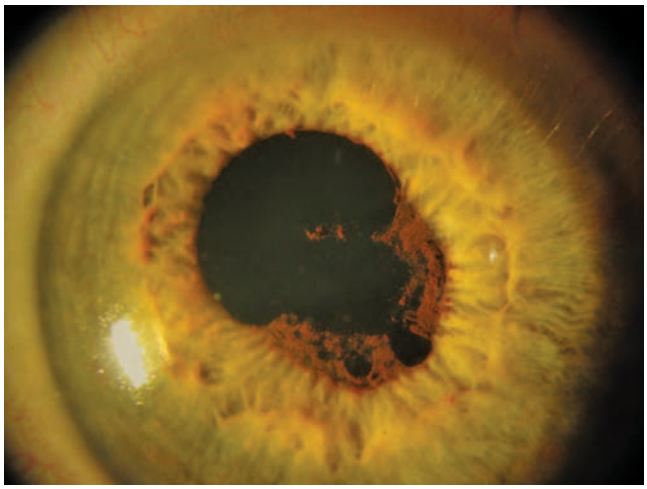

FIGURE 23-7 Posterior synechiae.

With more forceful blunt trauma, patients may suffer direct damage to the iris sphincter causing tears or traumatic mydriasis (seen near the pupil) or to the iris base causing iridodialysis (seen near the limbus). On initial examination, patients with traumatic mydriasis (dilation) or iris sphincter tears will have unequal pupil size and irregularity to the pupillary border. Most patients may not complain acutely of symptoms, but some will note the difference between the affected and unaffected eye in a mirror and question the etiology. There is no acute treatment for sphincter tears and traumatic mydriasis other than to treat the associated traumatic iritis.

With significant blunt force trauma, the longitudinal and radial fibers of the iris root can be torn apart at the ciliary body causing an iridodialysis. The diagnosis can easily be made with a penlight exam. The separation can occur at any clock hour and often will look as though a second pupil has been formed near the limbus, the area where the white sclera and clear cornea meet. Sometimes patients with large iridodialysis will complain of diplopia (seeing double) in the affected eye due to images being projected through the pupil and also simultaneously, through the new iris defect.

Patients who suffer iris trauma need proper follow-up with an ophthalmologist to monitor resolution of the inflammatory reaction and intraocular pressure and assess potential damage to other anterior segment structures. For some patients with extensive iris damage, reconstructive anterior segment surgery may be warranted.

Traumatic Lens injury

LENS DISLOCATION/SUBLUXATION

The lens is suspended like a hammock behind the iris by fibers called zonules that attach to the ciliary body and to the equator of the lens capsule. The lens is part of the total visual system and helps with vision and accommodation. Without the natural lens in position, patients would need a contact lens or intraocular lens placed to help visual rehabilitation. Thus, patients who suffer from lens dislocation will complain of decreased vision as well.

Three major traumatic injuries to the lens are lens dislocation/subluxation, traumatic cataract, and intralenticular foreign body. With blunt trauma, the zonules may break and cause the lens to dislocate either partially or completely depending on the amount of support remaining. A sign of zonular fiber loss may be seen at the slit lamp as iridodonesis, where the iris "jiggles" with rapid eye movements, or phacodonesis, where the lens shakes or moves with rapid eye movements. Typically, emergent intervention is not needed for lens subluxation or dislocation unless the lens is dislocated into the anterior chamber causing pupillary block, where the lens is obstructing aqueous flow from behind the pupil, or corneal endothelial decompensation, where the lens is in contact with the corneal endothelium.

Patients who have a history of prior cataract surgery with artificial intraocular lens placement can also experience artificial lens dislocation. They will also experience a decrease in vision since the artificial lens acts as a substitute for the natural lens and is part of the total visual system. Surgical intervention can usually be done to reposition or replace the intraocular lens as an outpatient on an elective basis.

TRAUMATIC CATARACT

Traumatic cataracts are generally seen months to years after the acute episode. They can result from blunt trauma, penetrating trauma, electrical shock, and ionizing radiation. Unilateral vision loss in young adults is most commonly due to unilateral traumatic cataract, most likely due to not only accidental trauma, but also sports-related activities that routinely have associated blunt trauma, such as boxing, soccer, and martial arts. Although traumatic cataracts may pose additional intraoperative risks, modern techniques for cataract removal are quite successful and yield excellent vision provided there are no retinal or optic nerve abnormalities.

LENTICULAR FOREIGN BODY

Lenticular foreign body refers to the unusual occurrence of a small foreign body, usually metallic, being retained within the crystalline lens.[44-46] On many occasions, these have been well tolerated for many years without adverse effect as the anterior capsule heals over the site of entry into the crystalline lens. A potential consequence of this type of injury would be cataract formation, requiring surgical removal by phacoemulsification.

Vitreous Hemorrhage

Vitreous hemorrhage occurs in 30% of eyes with serious trauma. Its presence attests to the severity of the injury and is a marker for concomitant injury that is harder to detect due to its presence. Vitreous hemorrhage is invariably present in eyes with scleral rupture and may be present in penetrating injuries. In closed globe injury, it may be associated with iris sphincter tears, hyphema, and lens dislocation, while in the posterior segment, associated findings include retina tear/detachment, traumatic macular hole, choroidal rupture, and traumatic optic neuropathy.[47]

Eyes with vitreous hemorrhage need to be examined carefully to rule out occult scleral rupture or laceration and concurrent retinal tears or detachment. If the severity of the vitreous hemorrhage does not allow sufficient visualization of the retina, B-mode ocular echography to detect retinal tears or detachment can be used as an acceptable alternative to direct visualization. It should be noted that when the vitreous hemorrhage is very severe, it is impossible to reliably detect a retinal detachment.

In the case of closed globe injury, management includes observation for 4 to 6 weeks, as spontaneous resolution is common, with vitrectomy if faster visual rehabilitation is desired or at a later date for nonclearing vitreous hemorrhage. Recently it has been suggested that early vitrectomy can prevent retinal complications, although convincing evidence for this is lacking thus far.

Intraocular Foreign Body

IOFBs are present in 30% to 40% of open globe injuries.[48-50] In the vast majority (>85%), the patient is male.[48,50-52] The IOFB is typically metallic (90%), usually iron, and results from hammering (60%–70%) or use of a high-speed rotary tool.[49,51,52] Glass foreign bodies can be found after car accidents, explosive blast injuries typically associated with terrorism, or assault with beer bottle.[53] Stone and concrete represent less than 2% of IOFBs, except in combat trauma, where they are common after eye injury following explosion of roadside improvised explosive devices (IEDs).[49-51] The majority of IOFBs are in the posterior segment (75%).[52]

Patient symptoms are dictated by the possible concomitant injury or complication (corneal laceration, scleral laceration, cataract, vitreous hemorrhage, endophthalmitis, retinal detachment). In every case of corneal or scleral laceration, even when self-sealing (ie, not requiring operative repair), it is critically important to rule out IOFB by helical CT of the orbits. Additionally, any time a patient presents with ocular pain after hammering and subconjunctival hemorrhage, CT is essential to rule out IOFB as a scleral laceration may be present under the hemorrhage.

IOFBs are associated with an increased incidence of endophthalmitis that results in poorer visual outcomes after penetrating injury.[54,55] Additionally, copper foreign bodies may incite severe inflammation, and in the long term, retained IOFBs may lead to retinal toxicity (eg, iron IOFBs lead to ocular siderosis, which among other effects leads to loss of vision due to retinal toxicity).[56]

Although there is agreement that primary repair of open globe injury should be undertaken emergently, the timing of IOFB removal is controversial.[52,54,57-59] IOFB removal from the posterior segment is usually performed by pars plana vitrectomy, although for ferromagnetic IOFBs, an external magnet can be used for smaller IOFBs if the requisite expertise for performing pars plana vitrectomy is not available.[58-60]

Retinal Contusion

Commotio retinae, also known as Berlin edema when it involves the posterior pole, is the term used to describe the opacified retina observed as a result of closed globe contusion injury to the retina. If the macula is involved, the vision will be affected. Spontaneous resolution is the rule, with the long-term prognosis determined by concomitant retinal pigment epithelium injury.

Contusion of the retinal pigment epithelium is infrequently described, yet it is a common sequela of closed globe injury characterized by atrophic changes and mottling of the retinal pigment epithelium in the long term. It is an important cause of limited vision following such injury.[61] Patients with this injury require serial dilated examinations with an ophthalmologist as an outpatient to determine if late sequelae (days to weeks) develop such as retinal holes, tears, or detachments.

Retinal Detachment

Retinal tears/dialyses and detachment may arise following closed or open globe injury. The difference between retinal tears and dialyses is beyond the scope of this text but is significant when considering surgical repair of a detachment.

Following closed globe injury, the sudden expansion of the equatorial region of the eye results in the forced separation of the vitreous from the retina; in young individuals, the vitreous may adhere strongly to the retina, and its forced separation may result in tears in the retina or retinal dialysis. Usually vitreous hemorrhage (which can be very minor) occurs at the same time due to bleeding from the vascular retina. In about 85% of patients with retinal tears or dialyses, fluid passes under the tear/dialysis, causing the retina to separate from the choroid, which is a retinal detachment.[62]

Patients with retinal tears may report floaters (black dots or "spider"-like opacities in their vision that move as the eye moves), whereas patients with a retinal detachment report a visual field defect ("dark area" or "curtain" in the vision, "like seeing underwater"). If the retinal detachment advances to include the fovea, patients will also report blurred vision. Timely treatment of retinal tears with laser retinopexy (or cryopexy) to reinforce the adhesion between retina and retinal pigment epithelium will prevent retinal detachment. Given that most traumas involve young individuals whose vitreous is more gel than fluid, only 12% to 30% of traumatic retinal detachments present immediately after trauma, whereas 20% present more than 1 year after trauma.[62-64] Therefore, there is ample opportunity to prevent retinal detachment after closed globe injury, and it is imperative that a thorough examination of the fundus by indirect ophthalmoscopy by an experienced ophthalmologist is performed within a few days of such injury.

If a retinal detachment occurs, timely treatment (before the fovea is involved) can lead to preservation of excellent vision; therefore, a retinal detachment not involving the fovea needs to be repaired within 24 hours. Once the fovea is involved, the vision will never be normal; patients presenting with fovea-involving retinal detachments need to undergo repair within 1 week of presentation. When retinal detachments are not treated promptly, proliferative vitreoretinopathy (a process of scar formation in the vitreous cavity) supervenes and surgical outcomes are poorer (both anatomic and visual outcomes), and there is a greater risk of phthisis bulbi (globe becoming shrunk with opacification of the cornea). Treatment of retinal detachments is by scleral buckling if the cause is a dialysis, pars plana vitrectomy if there is a giant retinal tear, and pars plana vitrectomy or scleral buckling if the cause is a retinal tear.[65,66]

In open globe injury, retinal tears commonly arise by the same mechanisms as in closed globe injury but may also be the direct result of the penetrating or perforating injury (eg, a sharp projectile penetrating the eyewall and the choroid and retina causing a retinal tear); IOFBs may cause further retinal tears at the site of internal impact on the retina. Moreover, a hemorrhagic retinal detachment may arise from bleeding under the retina. Retinal detachment occurs in approximately

20% to 29% of open globe injuries.[67,68] Treatment is by pars plana vitrectomy following primary repair.

Traumatic Macular Hole

Traumatic macular holes usually arise as a consequence of blunt ocular trauma, typically from a fist or finger, a champagne cork, a ball (baseball, softball, soccer ball, tennis ball usually), or rubber band.[69-71] The typical symptom is a blurring of the central vision. It should be noted that development of a traumatic macular hole may be delayed by a few days or weeks following trauma.

Anatomic closure occurs spontaneously in 44% to 64% of cases within the first 4 to 6 months.[72,73] Pars plana vitrectomy with lifting of posterior hyaloid face and gas endotamponade successfully closes macular holes in up to 96% of patients and is indicated after a period of observation for spontaneous closure, typically about 3 to 4 months.[69-71] Improvement in vision usually accompanies anatomic closure but may be limited by retinal pigment epithelium mottling or atrophic changes due to retinal pigment epithelium concussive injury.

Chorioretinitis Sclopetaria

Chorioretinitis sclopetaria is a rare type of closed globe contusion injury due to a high-velocity projectile passing adjacent to the globe without penetrating it. On dilated eye examination of the retina, a white area seen as sclera is visible surrounded by hyperpigmentation adjacent to the path of the projectile. Remote to the path of the projectile is an area of hyperpigmentation, and retinal pigment epithelium atrophic changes are present with a characteristic severe epiretinal membrane, usually at the macular area.[74,75] These appearances become apparent several weeks after the injury, whereas immediately, they are typically obscured by vitreous hemorrhage.[74,75]

Endophthalmitis

Endophthalmitis is a devastating complication of ocular trauma occurring in 1% to 11% of open globes,[52,55,76-79] with a higher incidence (4%–30%) when IOFBs are present.[52,54,77] Delayed primary closure, presence of IOFB, disruption of the lens capsule, rural setting of injury, and possibly posterior segment involvement increase the risk of endophthalmitis.[54,55,76,79] Common microorganisms are streptococci, staphylococci (especially with IOFBs), and *Bacillus cereus*, whereas gram-negative organisms occur in about 10% and fungi in fewer than 5% of injuries.[54,78-80]

Symptoms include severe to extreme pain, sensitivity to light, and decreased vision. Hypopyon (white blood cells/pus collection in the anterior chamber), fibrin in the anterior chamber (Fig. 23-8), vitreous inflammation, and sheathing of vessels are characteristic.[79] Other signs commonly present are chemosis and erythema of the conjunctiva (which can be severe), severe tenderness, and lid edema. Sadly, this may be misdiagnosed as the patient having only traumatic iritis, which rarely would present with hypopyon or severe tenderness.

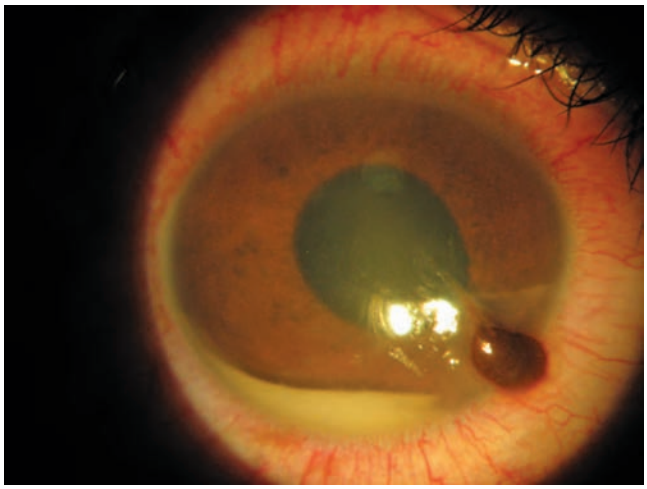

FIGURE 23-8 Hypopyon in an eye with corneal laceration and iris protrusion.

Endophthalmitis is a true ophthalmic emergency, and in the case of virulent microorganisms such as *Bacillus*, a few hours can make the difference between retaining the globe and losing it to phthisis. Suspicion of endophthalmitis should prompt an emergent ophthalmology consult and institution of systemic treatment with a fluoroquinolone (ideally fourth generation) because fluoroquinolones have excellent penetration into the vitreous and they are effective against *B cereus*.[81] Definitive management is by injection of intravitreous antibiotics, with vitrectomy for selected cases.

Choroidal Rupture

Rupture of the choroid accompanies closed globe trauma more often than open globe trauma and is usually a consequence of injury with a ball (soccer ball), other large projectile (eg, rock, shoe), or fist.[82,83] The choroidal rupture appears as a white crescent, typically concentric to the optic nerve and within the macula (>70%).[82,83] Vision immediately following injury may be limited if the choroidal rupture is through or adjacent to the fovea or from associated subretinal hemorrhage (which resolves).

A treatable, long-term complication of choroidal ruptures in the macula, especially in older individuals, is the formation of choroidal neovascular membranes (CNVMs; growth of new vessels from the choroid under the retina, which tend to leak fluid or cause hemorrhage). Anti-vascular endothelial growth factor inhibitors injected intravitreally or photodynamic therapy for CNVM due to choroidal rupture may lead to excellent visual outcomes if the patient presents early.[84,85] Therefore, it is critically important to inform patients with choroidal rupture at risk of developing CNVM that if they develop a sudden change in vision or metamorphopsia (distortion in vision so that straight lines appear curved), they should see an ophthalmologist at once.

Traumatic Optic Neuropathy

Sharp objects, projectiles, or bone fragments may directly damage the optic nerve (direct traumatic optic neuropathy). Direct optic neuropathy is associated with severe loss of vision with little prospect of improvement. If suspected, imaging of the optic nerve (orbital CT) is recommended to detect the rare case where surgical relief of impingement of the optic nerve may improve vision.

More commonly, however, traumatic optic neuropathy results from concussive head injury, especially involving impact to the forehead (indirect traumatic optic neuropathy). In indirect optic neuropathy, impact forces are transmitted from the frontal bone to the orbital bones and are concentrated in the area of the optic canal, causing a shearing injury to neuronal axons of the intracanalicular portion of the optic nerve.[86,87]

Patients with indirect traumatic optic neuropathy are usually young, male (85%), have commonly lost consciousness, and have typically sustained their injury as a result of involvement in a motor vehicle or bicycle accident (about 50%), fall, or assault.[88,89] Patients notice a typically unilateral decrease in vision immediately or after regaining consciousness.[88,89] The severity of visual loss is often dramatic (40% have NLP and a further 23% only have light perception or HM vision). In cases with less dramatic visual loss, symptoms may include visual field defects and abnormalities of color vision.[88,89] On examination, a RAPD is present, but the fundoscopic examination is normal until several weeks after the injury when optic nerve pallor or atrophy becomes apparent.

Spontaneous improvement has been reported in up to 57% of patients with indirect traumatic optic neuropathy.[88,89] The regimen employed in the Second National Acute Spinal Cord Injury Study (NASCIS) of an initial dose of 30 mg/kg of methylprednisolone followed by continuous infusion of 5.4 mg/kg/h for 24 to 48 hours has been used for the treatment of traumatic optic neuropathy despite lack of evidence for efficacy in this condition.[88-93] Indeed, several comparative studies have failed to show benefit from the use of megadose or high-dose corticosteroids over placebo for indirect traumatic optic neuropathy.[88,89,94] Moreover, recently the CRASH study demonstrated excess mortality for patients with significant closed-head injury who were given corticosteroids compared to placebo.[95] Therefore, the role, if any, of megadose steroids in the treatment of this condition is controversial. Finally, surgical decompression has been performed with encouraging results in several case series but with no evidence that results are superior to observation.[96]

OPTIC NERVE AVULSION

Optic nerve avulsion is a specific type of indirect traumatic optic neuropathy with a distinct pathogenesis. In optic nerve avulsion, a blunt object (typically a finger, unusually a snooker cue or golf club) is inserted between the globe and orbit and causes abrupt rotation of the globe as well as a sudden increase in intraocular pressure resulting in retraction of the optic nerve within its dural sheath. The clinical appearance is striking once the accompanying hyphema or vitreous/subhyaloid hemorrhage clears: there is a hole or cavity where the optic disc has retracted within its dural sheath.[97,98] Prognosis is guarded, with limited potential for spontaneous improvement and no effective treatment.

Eyelid Lacerations

The approach to a patient with eyelid trauma must be systematic and take into account a detailed history to rule out open globe injuries that may preclude repair of the eyelid until the globe can be surgically closed. There are multiple types of eyelid injury, and a patient can have more than one type depending on the mechanism of injury. These include the following:

1. Contusion: blunt impact injury with superficial soft tissue swelling and ecchymoses
2. Abrasion: scraping causing superficial epithelial skin loss
3. Avulsion: shearing or tearing away of tissue
4. Puncture: defect through multiple tissue planes caused usually by sharp objects
5. Laceration: cut tissue can be superficial or deep, usually caused by sharp objects

For patients with isolated eyelid contusion and abrasion, conservative medical management with ice packs and antibiotic ointment is usually all that is required. Given time, the healing and reepithelialization of the skin give good results.

For eyelid avulsion, puncture, and laceration, surgical repair is generally needed. Depending on the patient's age, mental status, and size of injury, repair may be done at the bedside with local anesthesia, preferably with 2% lidocaine with epinephrine for better hemostasis. Deeper tissue involvement, full-thickness, marginal, or lacrimal duct involvement will generally require involvement of an ophthalmologist or oculoplastic surgeon to repair. Signs that may clue into deeper tissue involvement include orbital fat prolapse, exposed sclera under the laceration, and medial canthal involvement. Of note, lacerations of the eyelid medial to the puncta should raise concern for potential injury to the lacrimal drainage system, requiring further exploration and possible probing and irrigation prior to repair.

Once an eyelid laceration is suspected, a plan to explore the extent of the injury needs to be formulated. Since soft tissue swelling can distort the natural anatomy and create the false impression of missing tissue due to excessive tension when approximating tissue margins, ice packs applied to the wound can help to decrease swelling before manipulation. It is important to be sure that an occult open globe injury has been ruled out before exploration of the eyelid laceration (Fig. 23-9).

When exploring an eyelid laceration, anesthetize the tissues prior to cleansing and gently pull lacerated tissues apart during your inspection, as fibrin tends to hold these lacerated edges together giving an inaccurate impression on the level of deep tissue involved. It is important to reapproximate the deep tissue layers to avoid undue excess tension on the

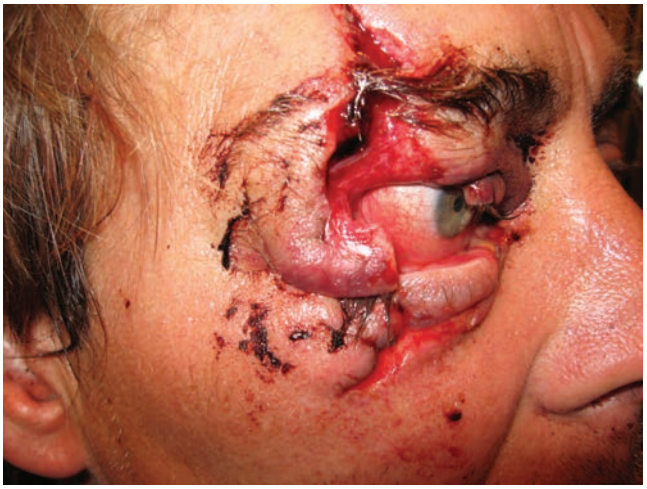

FIGURE 23-9 Lid laceration.

superficial layers. The timing of eyelid laceration repair is more forgiving than most other ocular injuries due to the well-vascularized tissues of the eyelid. In some cases of extensive swelling, waiting 24 to 72 hours for eyelid repair can give a better anatomic and cosmetic result.

For subcutaneous closure, an absorbable suture is preferred, such as 5-0 polyglactic acid (Vicryl, Ethicon, Somerville, NJ) on a spatulated needle, to close deeper tissues. This same suture size can be used above and below the brow or to secure tissue to the periosteum. For skin closure, nylon suture is preferred because it creates the least amount of inflammation, but if follow-up for removal of the sutures in 7 to 10 days cannot be assured, it is better to use an absorbable suture. For closure above and below the brow, 6-0 sutures can be used; some oculoplastic surgeons advocate using a smaller suture size such as 7-0 for closure below the brow.

Orbital Fractures

Patients with orbital injuries such as orbital wall and floor fractures typically will have concomitantly significant facial trauma. These patients will need to be simultaneously managed with head and neck, oral maxillofacial, or plastic surgeons. An orbital CT scan should be obtained to optimally diagnose and manage orbital injuries. Many times, specific orbital cuts can be added to a standard face CT scan protocol when initially ordered to evaluate the patient with facial trauma.

Neurosurgery consultation may be necessary for patients with orbital roof fractures, which can be sustained as a result of significant trauma, usually from motor vehicle accidents or falls from heights. These fractures can often include complications of cerebrospinal fluid leaks due to dural tears, intracranial hemorrhage, traumatic encephalocele, and brain abscesses or meningitis.

The orbit is composed of many bony structures, with the purpose of protecting the globe. The orbital roof and lateral wall have the thickest walls. The thinnest wall is the medial wall, composed of the ethmoidal bone, also known as the lamina papyracea, and the orbital floor medial to the infraorbital groove. Contusive orbital injuries can lead to a "blowout" fracture of these thin areas.

The quality of the thin bones in orbital blowout fractures actually provides a protective feature, where a large area of the orbital floor and medial wall has given way allowing for decompression of the orbital contents. This expanded volume into the sinuses allows for decreased congestion in the orbital space. A pure blowout fracture does not include fracturing of the orbital rim.

When evaluating a patient with possible orbital fracture, one of the signs of an orbital floor fracture is decreased skin sensation on the cheek of the affected side. The infraorbital nerve, a branch of the trigeminal nerve, travels through the infraorbital canal and within the floor to exit just under the inferior orbital rim at the infraorbital foramen. This nerve is often affected when traumatized by orbital floor fractures and presenting with hypoesthesia of the cheek. Another clue can be subcutaneous emphysema that often results from the patient blowing his or her nose, forcing air into the tissues.

Since up to one-third of patients with orbital blowout fractures will have other accompanying ocular injuries, such as corneal abrasion, iritis, hyphema, ruptured globe, retinal detachment, and retinal hemorrhage, it is important to examine these patients systematically with the eight-point eye examination to diagnose correctly and treat more sight-threatening conditions first.

Once an orbital wall or floor fracture has been diagnosed, patients must be given instructions to not blow their nose since this can cause further expansion of air into the orbital tissue and can lead to a tight orbit and elevated intraocular pressure. Patients can use ice packs during the first 48 hours to help with reduction of the soft tissue swelling, and for patients who need surgical repair, this is usually done in the next 1 to 2 weeks.

Management and treatment of orbital floor fractures are discussed in Chapter 24.

Intraorbital Foreign Bodies

Intraorbital foreign bodies can result from both blunt and sharp objects, usually as a result of assault, industrial accidents, accidents at home, or recreational activities. They can cause vision loss if the globe is involved, or in the case of neurologic damage from intracranial extensions. A high index of suspicion is important in evaluating patients with recent or remote history with persistent periocular inflammation. Signs and symptoms of retained intraorbital foreign bodies include the following:

1. Orbital mass
2. Proptosis
3. Painful or restricted eye movements
4. Diplopia
5. Ptosis
6. Lagophthalmos
7. Orbital cellulitis

8. Draining sinus tract
9. Gaze-evoked amaurosis (transient loss of vision)

An orbital CT scan is the preferred imaging modality since metallic foreign bodies, which are the most common type of intraorbital foreign body,[99] are a contraindication for MRI scan; once a metallic foreign body is ruled out, an MRI scan may be better at detecting organic matter such as wood. Depending on the size and extension of the foreign body, neurosurgery and/or otorhinolaryngology consultation may be necessary to safely remove the foreign body.

Not all orbital foreign bodies need to be removed. Certain inert metals, glass, plastic, and silicone can be left in place as long as there is no optic nerve impingement. Foreign bodies made from iron should be removed since long-term toxicity can occur leading to vision loss and retinal damage from siderosis. A detailed history of the mechanism of injury will be crucial in deciding the most likely composition of the foreign body.

Orbital Compartment Syndrome

The orbit is susceptible to compartment syndrome due to its small size and the bony walls of the orbit lacking the ability to stretch or flex. The normal orbital volume is 30 cm^3. Trauma directly to the orbit or to other regions of the face resulting in fractures can cause bleeding into retrobulbar, subperiosteal, extraconal, and/or intraconal spaces of the orbit with rapid expansion in orbital distention. Rapid escalation in orbital compartmental pressure can cause ischemia of the orbital tissues, and increased intraocular pressure can lead to damage and permanent vision loss. Patients with large orbital floor and wall fractures have less risk for developing orbital compartment syndrome since the orbital contents can be decompressed into the sinuses.

Proptosis and taut orbital content or increased resistance to retropulsion on examination are always seen in orbital compartment syndrome. Depending on the extent of orbital congestion, patients with mild compartment syndrome have only mildly elevated intraocular pressures without visual compromise and can be treated with glaucoma agents topically or orally. Once intraocular pressures exceed 40 mm Hg despite antiglaucoma therapy in an orbital compartment syndrome patient, associated with vision loss and likely with an associated afferent pupillary defect, a lateral canthotomy and cantholysis would be indicated (see Atlas Figure 5).

On rare occasions, a patient may have bleeding into the optic nerve sheath, causing direct impingement and compression of the optic nerve, or a bony fragment from posterior fractures can compress on the optic nerve, causing an RAPD. Thus, reviewing the orbital imaging is important to rule out such cases that will need to be referred to an oculoplastic surgeon for urgent optic nerve sheath fenestration.

Patients with mild compartment syndrome should not have any signs of optic nerve compromise, and a patient with an RAPD and decreased vision may need urgent decompression with emergent canthotomy and cantholysis of the lateral canthal tendon performed at the bedside. This works to allow the taut orbital content to prolapse anteriorly out of the orbit. Success is measured by improved vision, lower intraocular pressure, and reversal of optic nerve compromise or RAPD. If the orbital compartment syndrome is not decompressed adequately with a lateral canthotomy and cantholysis, consultation with an oculoplastic surgeon is warranted to decompress the orbit via bony decompression.

Steps to perform a lateral canthotomy and inferior cantholysis are as follows:

1. Obtain informed consent if patient is able to cooperate.
2. Prep and drape the affected eye.
3. Anesthetize with 2% lidocaine with epinephrine in the lateral canthal region. Make sure to infiltrate subcutaneously and subconjunctivally.
4. Allow the anesthetic to take effect, and clamp a hemostat over the lateral canthus vertically; this will help direct the cut in the next step.
5. Using Steven scissors, place one blade on the conjunctival side of the lateral canthus and the other blade on the skin surface, and then cut the lateral corner of the eyelid while applying lateral pressure.
6. The inferior crus of the lateral canthal tendon will need to be cut to release the lower eyelid from the lateral orbital wall. Taking the scissors with blades closed, strum the inferior tendon inside the cut canthotomy wound. The attachment should feel like a firm, tense cord. Now, open the blades of the scissors and cut the cordlike structure until the lower eyelid becomes freely mobile.
7. Hemostasis can be achieved with pressure or the use of handheld cautery if available.

CONCLUSIONS

Although in some cases need of a consultation with ophthalmology will be grossly evident (Fig. 23-10), others may be more subtle, such as an occult scleral laceration or open globe injury from a full-thickness eyelid laceration (Fig. 23-11). The key in successfully managing patients with ocular injury

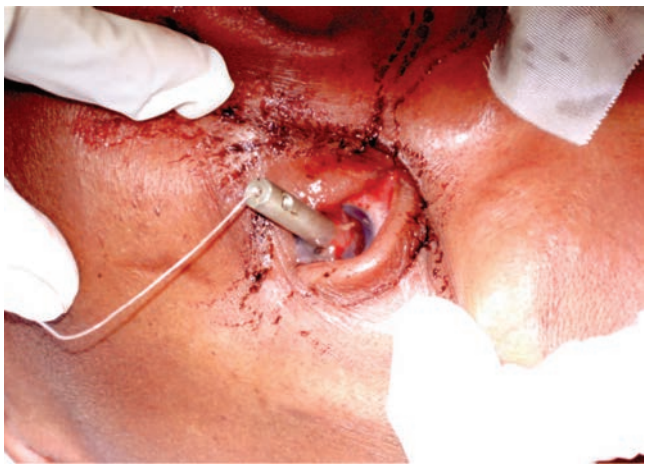

FIGURE 23-10 Intraocular foreign body.

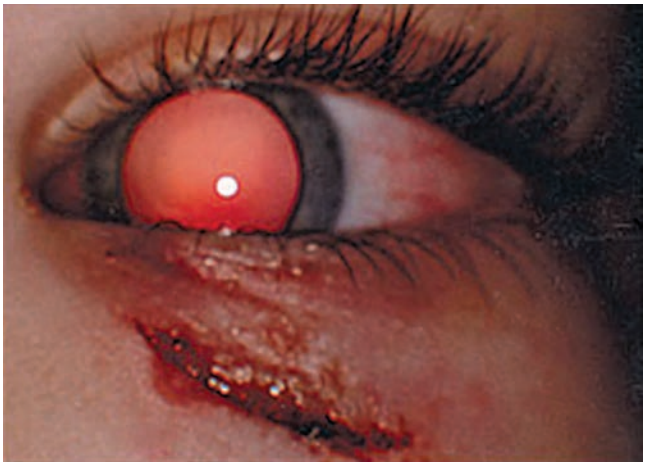

FIGURE 23-11 Open globe.

is to perform a systematic exam and use a common vocabulary to communicate those findings with the consultant.

The role of the emergency care provider or trauma surgeon with regard to ocular trauma is to recognize common traumatic eye injuries that can be managed immediately and be able to refer sight-threatening conditions appropriately for ophthalmic follow-up. While there are a few true ophthalmic emergencies—open globe, chemical injury, endophthalmitis, and orbital compartment syndrome—many isolated ocular injuries in an otherwise healthy individual can be managed outside of the emergency department setting. Like injury to any other organ system, potential long-term complications require follow-up with specialists.

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24

Face

Raj D. Dedhia • D. Gregory Farwell

KEY POINTS

- A thorough understanding of head and neck anatomy is critical in identifying the true extent of injuries to soft tissue and underlying viscera.
- Early documentation of facial nerve function is critical in guiding management of facial nerve injuries, particularly in critically ill patients in whom sedation may confound later examination.
- The goal of bony reconstruction in maxillofacial trauma is to restore function, structure, and symmetry.
- Long-term follow-up and postoperative imaging are often required to confirm correct skeletal repair and to watch for skeletal or soft tissue complications that would benefit from revision surgery.

INTRODUCTION

The face is the centerpiece of human interaction and expression. Facial structures are also integral in essential functions such as breathing, vision, eating, and communication. Patients with facial deformity pay a significant social penalty[1] and suffer functional impairment. Therefore, restoring facial form and function can greatly improve a patient's quality of life.

The evaluation of any trauma patient should begin with a primary survey that is focused on airway, breathing, circulation, disability, and exposure, or the "ABCDEs." After the appropriate Advanced Trauma Life Support (ATLS) algorithms are instituted and the patient is acutely stabilized, secondary survey begins, including a thorough evaluation of facial injuries (Fig. 24-1). The anatomy, evaluation, and management of soft tissue and bony trauma of the face are reviewed.

MANAGEMENT OF FACIAL SOFT TISSUE TRAUMA

Scalp and Forehead

ANATOMY

The scalp is a multilayered soft tissue structure that envelopes the calvarium, extending from the external occipital protuberance to the supraorbital rim. The forehead aesthetic subunit of the scalp includes the arc from the supraorbital rims to the hairline, or trichion. The soft tissue of the scalp and forehead consists of five layers and can be remembered using the mnemonic SCALP: *s*kin, sub*c*utaneous tissue, *a*poneurosis galea, *l*oose areolar tissue, and *p*eriosteum (Fig. 24-2).

The arterial supply to the scalp comes from five named vessels off of the internal and external carotid arteries.[2] The external carotid branches include the occipital artery, superficial temporal artery, and the posterior auricular artery. Internal carotid artery branches include the supraorbital and supratrochlear arteries. These ultimately form extensive anastomoses in the subcutaneous tissue layer of the scalp, providing a robust blood supply and vascular redundancy that limits watershed areas of compromised blood supply. Injury to vessels in this layer can result in significant blood loss as the vessels remain tethered open from their dense connective tissue attachments.

The layers of the lateral scalp overlying the temporal regions are more complex (Fig. 24-3). The skin and subcutaneous layers in this region are continuous with the remainder of the scalp. The galea is continuous with temporoparietal fascia (TPF) and contains the superficial temporal artery and temporal branch of the facial nerve.[3] Deep to the TPF lies the deep temporal fascia, which splits into superficial and deep layers and envelops the intermediate (or middle) temporal fat pad above the level of the zygomatic arch.[3] The deep temporal fat pad, temporalis muscle, periosteum, and temporal bone lie deep to the deep temporal fascia.[4]

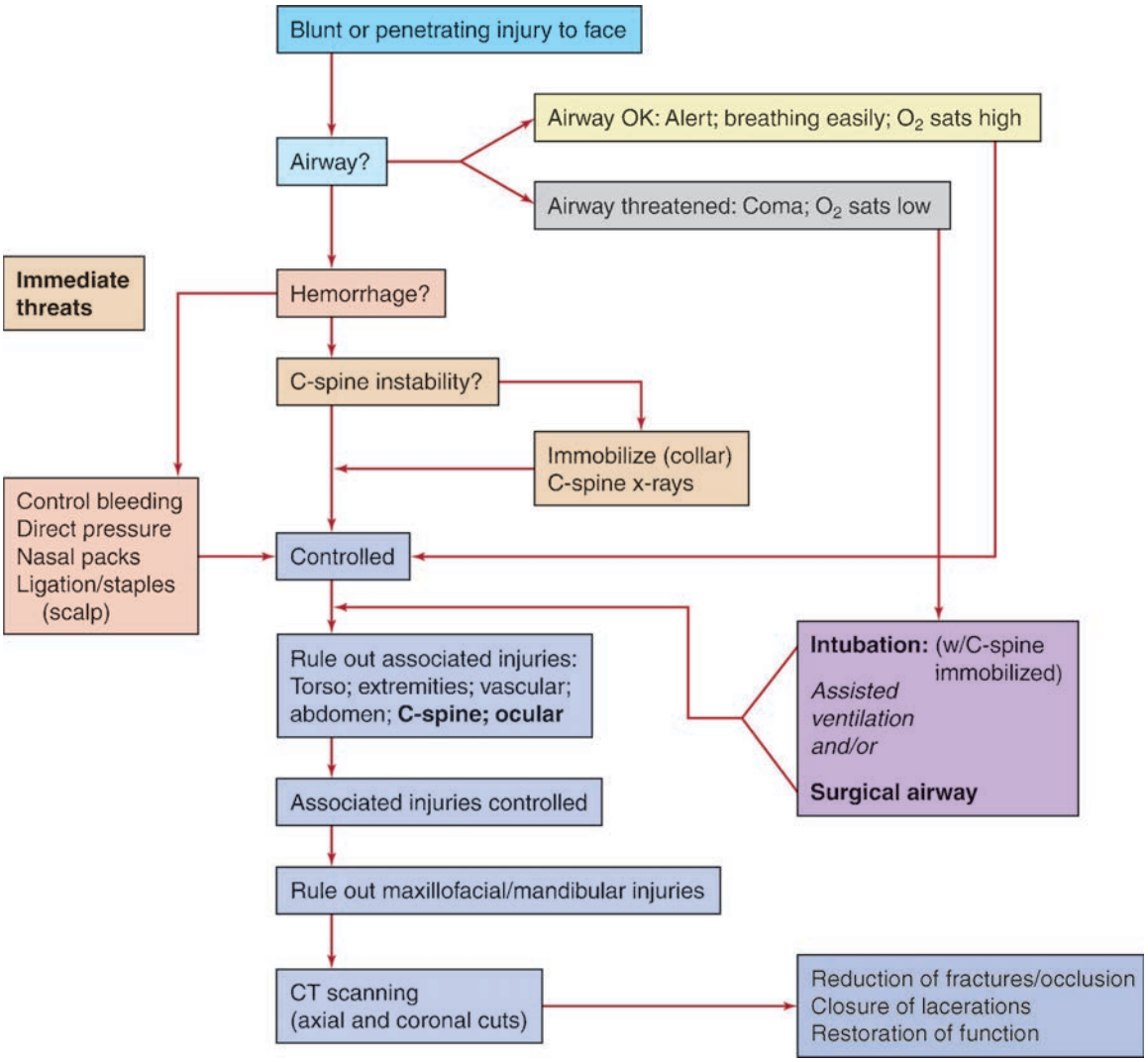


FIGURE 24-1 An approach to maxillofacial trauma. C-spine, cervical spine; CT, computed tomography; O_2 sats, oxygen saturation.

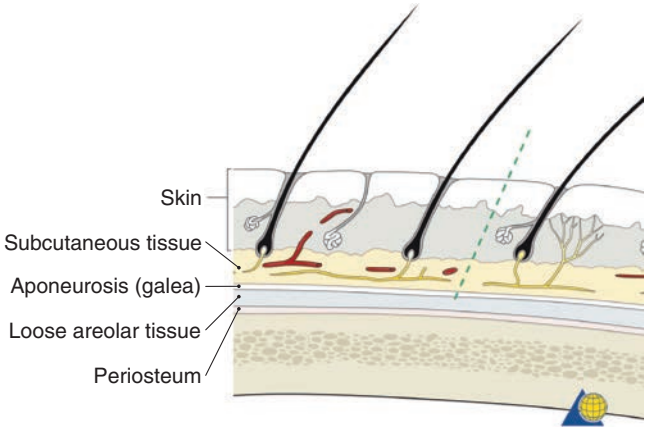


FIGURE 24-2 Anatomic layers of the scalp. (Reproduced with permission from AO Surgery Reference. www.aosurgery.org. Copyright by AO Foundation, Switzerland.)

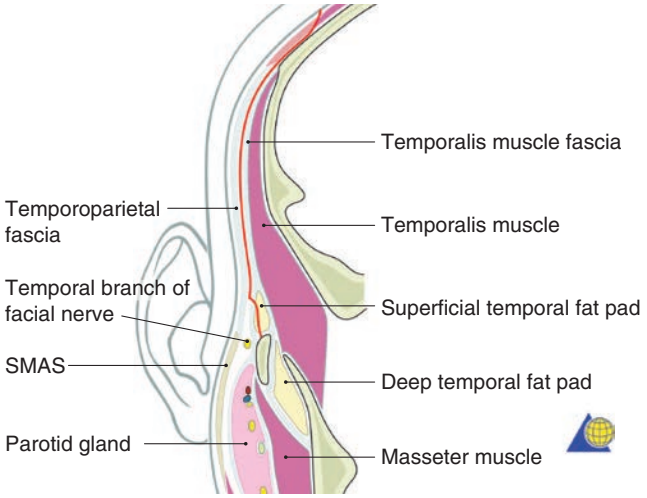


FIGURE 24-3 Temporal scalp anatomy. The red line represents the plane of dissection in raising a coronal flap. SMAS, superficial musculoaponeurotic system. (Reproduced with permission from AO Surgery Reference. www.aosurgery.org. Copyright by AO Foundation, Switzerland.)

EVALUATION

Scalp injuries, even when small, can present with extensive bleeding. Manual pressure or pressure dressings should be the initial measures to stop bleeding. If the bleeding does not tamponade, the wound edges should be examined for vessels that can be controlled with vessel clips, scalp hemostatic clips such as Raney clips, suture ligature, or cautery. Care should be taken to limit prolonged scalp hemostatic clips or cautery as damage to hair follicles can worsen alopecia along the wound.[5] Mild bleeding is frequently controlled by wound closure.

Once hemostasis is achieved, the scalp wound should be thoroughly irrigated and examined for size, depth, tissue loss, and involvement of the hairline. Open calvarial and dural injuries in severe traumas require earlier intervention and combined reconstructive efforts with a neurosurgeon.

MANAGEMENT

Lacerations without soft tissue loss are closed in a multilayer fashion. The galea and subcutaneous tissue are reapproximated with 3-0 sized braided suture for strength. The skin can be closed with absorbable or nonabsorbable 4-0 monofilament suture. Stainless steel skin staples are an alternative for skin closure and have similar aesthetic outcomes in hair-bearing scalp closures in the pediatric population, with the added benefit of decreased procedure time.[6]

When avulsion injuries result in a defect, wound management involves more complex reconstruction. The reconstruction method should take into consideration patient factors (eg, health and healing potential), size of the wound, relation to the hairline, and need for underlying cranioplasty.[4]

Critically ill patients who cannot tolerate an anesthetic may require healing by secondary intention with local wound care. This is ideal in bald patients, areas of concavity and light skin, and vascular wound beds that will promote granulation (ie, at least pericranium).[2] In addition, split-thickness skin grafting can be a quick reconstructive option used for wounds of any size. This can be done in one stage when there is a vascularized wound bed. Single- or two-staged techniques have been described for skin grafting denuded calvarium where a more vascular wound bed is created by drilling down to the diploic space.[7]

Scalp defects can be classified as small (<9 cm^2), medium (10–30 cm^2) and large (>30 cm^2).[2] Small defects can be closed by primary intention with the help of galeotomies to allow advancement and decrease tension. Medium and large wounds require skin grafting, tissue expanders, locoregional flaps, or free tissue transfer. Flaps should be designed in a manner that maintains the hairline when possible. When there is a calvarial or dural defect with cerebrospinal fluid (CSF) leak, large locoregional flaps or free tissue transfer is beneficial to protect the cranioplasty site and dural repair.

Cheek

ANATOMY

Understanding the intricate anatomy of the cheek is critical to the management of soft tissue injuries of the face. Beyond addressing lacerations and defects of the soft tissue, potential injuries to the parotid gland, parotid duct, and facial nerve must also be considered.

The soft tissue envelope overlying the cheek demonstrates continuity with the layers of the scalp and has several unique considerations. Below the skin and subcutaneous layers, the cheek contains the superficial musculoaponeurotic system (SMAS),[8] which is continuous with the TPF and galeal layers of the scalp as well as the platysma of the neck. In the lateral cheek, the parotidomasseteric fascia lies deep to the SMAS and covers the parotid gland and masseter muscle.

The parotid duct exits the anterior portion of the parotid gland and runs parallel and inferior to the line running from the tragus to the midportion of the upper lip.[9] Van Sickels[10] subdivided the parotid duct into three anatomic segments: (1) duct within parotid parenchyma, (2) duct overlying the masseter muscle, and (3) duct anterior to the masseter muscle as it pierces the buccinator and buccal fat prior to entering the oral cavity. These subdivisions predict risk of duct injury and guide approaches to repair.

An understanding of the course of the facial nerve branches within the parotid gland is critical to recognizing potential injury and instituting appropriate intervention. The facial nerve enters the parotid gland in very close proximity to the nerve's exit from the temporal bone through the stylomastoid foramen. As it traverses the parotid gland, the facial nerve branches into five main divisions: (1) temporal, (2) zygomatic, (3) buccal, (4) marginal mandibular, and (5) cervical.

The temporal branch exits the gland early, and its course can be projected by a line described by Pitanguy and Ramos[11] that begins 0.5 cm below the tragus and passes 1.5 cm above the lateral extent of the eyebrow. The nerve is at risk in penetrating trauma in the vicinity of that line and during surgical approaches to the zygomatic arch (Fig. 24-4).

The marginal mandibular nerve courses deep to the platysma and is superficial to the deep cervical fascia overlying

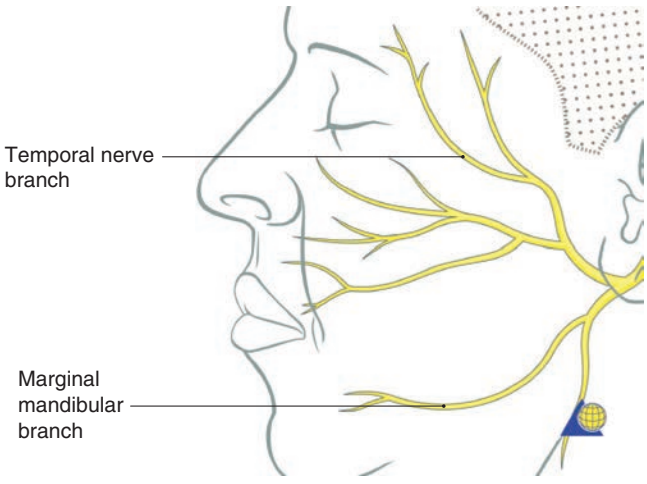


FIGURE 24-4 Orientation of facial nerve branches. Branching patterns can be variable. (Reproduced with permission from AO Surgery Reference. www.aosurgery.org. Copyright by AO Foundation, Switzerland.)

the submandibular gland. The nerve has a variable course inferior to the angle and body of the mandible and must be considered during transcervical approaches to the mandible.

EVALUATION

Evaluation of facial nerve function at the initial trauma evaluation is critical in determining need for surgical intervention and predicting degree of recovery. Facial nerve paralysis can lead to significant functional and social handicap from impairment in eye closure, oral competence, and facial expression.

Facial movement in the distribution of each of the five major branches of the facial nerve should be promptly assessed and documented because this will anatomically guide any surgical intervention. Assessing patients with severe trauma who are intubated and sedated can be difficult. Painful stimuli can elicit facial movement in these patients. Several facial nerve grading systems have been developed to describe facial nerve paresis and recovery, including the House-Brackmann[12] and Sunnybrook[13] facial grading systems. However, these can be difficult to interpret and do not substitute for a thorough descriptive visual exam.

Wounds should be evaluated for depth of injury. Penetrating injuries that violate the SMAS or platysma increase the risk for facial nerve, parotid gland, and parotid duct injury. Massage of the cheek with expression of saliva in the wound would be indicative of parotid gland or duct injury. The oral cavity should be examined for full-thickness injuries and the integrity of the parotid duct papilla. The parotid duct integrity can be assessed by cannulating the duct with a lacrimal probe intraorally via the papilla. Exposure of the probe in the wound would indicate injury to the duct. Others have advocated retrograde injection of methylene blue via the papilla and assessing for blue discoloration in the wound.[14] This has been used less frequently due to discoloration of the surgical field and obscuration of critical structures.[15] Diagnostic sialography is not routinely performed due to cost, challenge of cannulation in setting of edema and poor patient cooperation, and procedural time.[16]

MANAGEMENT

Parotid Gland Injury. Early identification of and intervention for parotid glandular injuries can avoid late complications that are difficult to manage, such as sialoceles and salivary-cutaneous fistulae. Lacerations to the gland without ductal injury can be managed with multilayer closure of the wound, including the parotid capsule and SMAS, and application of a pressure dressing for at least 48 hours.[17] Concomitant medical management with antisialagogues (eg, glycopyrrolate) helps decrease saliva excretion during the healing process. Some advocate for placement of a parotid duct stent to help maintain patency and flow in the setting of edema and pressure dressing.[15,17]

Most investigators believe that parotid duct injuries should be surgically managed and preferably early.[15] There are three general methods to manage duct injury depending on location and severity of injury. These include (1) primary repair with microsurgical reanastomosis, (2) salivary diversion by creation of oral fistula, and (3) ductal ligation with suppression of salivary gland function.[14] The method of repair is often dictated by the location of the injury, which Van Sickels[10] divided anatomically into three regions:

- Region A: duct injuries occurring in the glandular area
- Region B: duct injuries over the masseter muscle
- Region C: duct injuries anterior to the masseter muscle as the duct enters the oral cavity

Region B injuries often have adequate proximal and distal duct length to facilitate primary reanastomosis. In this case, the duct is stented with a silicone catheter or angiocatheter, and the duct is repaired with 8-0 or 9-0 nylon (Figs. 24-5A and B). The stent is then sutured in place to the buccal mucosa and kept in place for 10 to 14 days[14] (Fig. 24-5C).

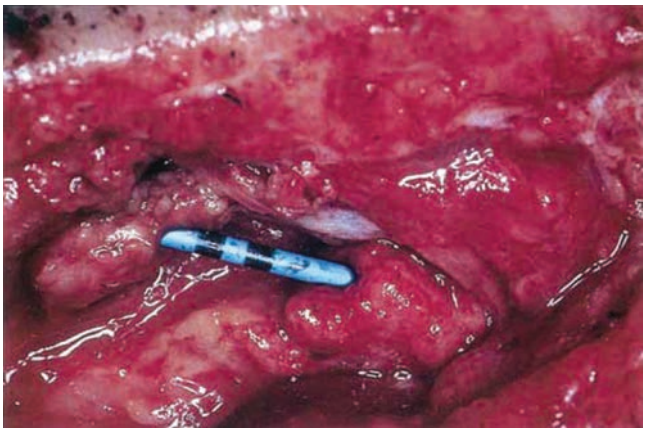
Region A and C injuries can also be repaired with primary microsurgical reanastomosis if there is adequate identifiable proximal and distal stump. When this is not the case, region A injuries can be managed as a parenchymal injury with multilayer closure and pressure dressing or ligation of the proximal duct with subsequent edema and atrophy of the gland.[17] In region C injuries where the distal stump is too proximal to the papilla, the proximal stump can be diverted to a separate location in the buccal mucosa and stented to keep the new papilla patent during the healing process.[14]

Late complications of sialocele and salivary-cutaneous fistula can be difficult to manage. Mainstay therapy for these complications includes aspiration of the fluid collection and salivary gland suppression with antisialagogues. Botulinum toxin has been shown to effectively treat sialoceles and salivary-cutaneous fistulae in traumatic and postparotidectomy cases.[18] Persistent fistula could require parotidectomy and excision of the tract if medical management fails; however, parotidectomy in a posttraumatic setting can pose a higher risk of facial nerve injury due to scar and obscuration of tissue planes.

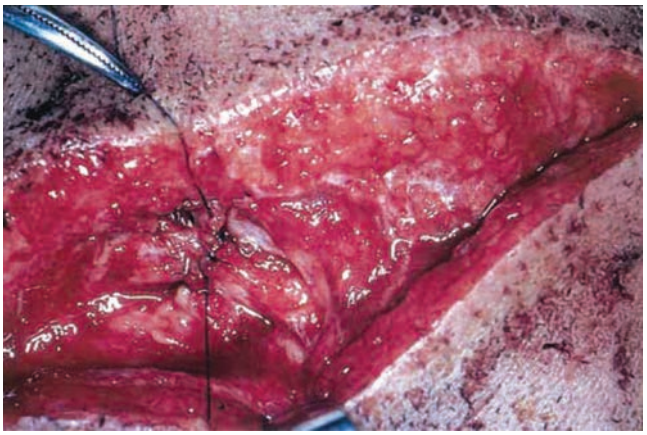
Facial Nerve Injury. Incomplete eye closure from facial nerve injury and orbicularis oculi muscle impairment can result in poor ocular protection, exposure keratopathy, and even blindness. Therefore, initial therapy includes ocular lubrication, moisture chamber, and eyelid taping if the patient has incomplete eye closure on exam.

For penetrating facial nerve injuries, early wound exploration and repair should be performed. If the patient is medically stable, facial nerve exploration should be done immediately. Electromyographic monitoring can be used to identify distal ends of transected nerve branches if exploration is performed within 72 hours of the injury (ie, prior to Wallerian degeneration).[19]

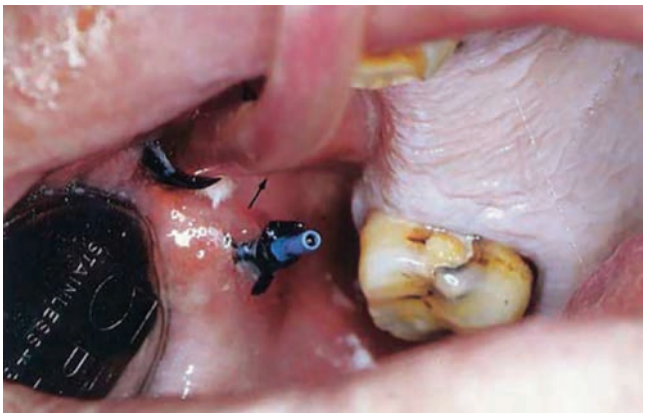
Primary neurorrhaphy has been shown to provide the best chance for recovery.[20-24] Nerve cooptation is performed with three interrupted sutures using 8-0 to 10-0 monofilament suture[19,20,22,25] in the epineurial layer (Fig. 24-6). Perineurial sutures have been supported by some to help reduce synkinesis but are technically challenging and more time consuming.[25]



A



B



C

FIGURE 24-5 Images of the parotid gland. (**A**) Stent cannulating distal and proximal ends of parotid duct. (**B**) Suture repair of parotid duct over stent. (**C**) Suture fixation of stent to buccal mucosa. (From Lewkowicz AA, Hasson O, Nahlieli O. Traumatic injuries to the parotid gland and duct. *J Oral Maxillofac Surg.* 2002;60(6):676-680. Used with permission. Copyright © American Association of Oral Maxillofacial Surgeons. Published by Elsevier Inc.)

When primary neurorrhaphy in a tension-free manner cannot be performed, cable grafting is the next preferred method of repair.[19,22,25] Options for donor nerves include great auricular, sural, and medial antebrachial cutaneous nerves.[25] When primary neurorrhaphy or cable grafting is not possible or recovery is not obtained, facial reanimation procedures can be performed secondarily at a later date.

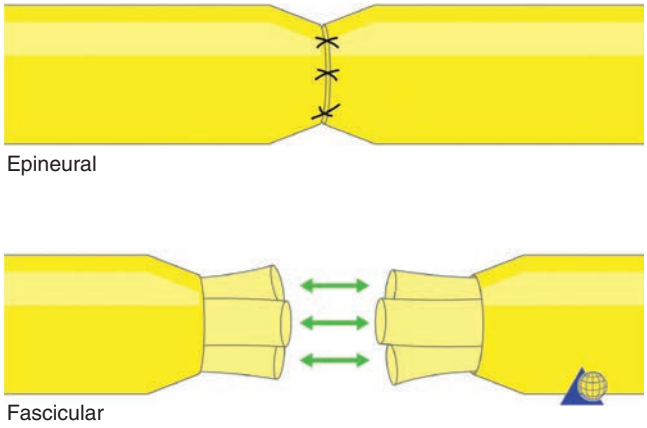


FIGURE 24-6 Schematic of epineurial neurorrhaphy. Three interrupted sutures are placed circumferentially. (Reproduced with permission from AO Surgery Reference. www.aosurgery.org. Copyright by AO Foundation, Switzerland.)

Eyelid

ANATOMY

The upper and lower eyelids are complex, multilayered structures (Fig. 24-7). The anterior lamella consists of the skin and orbicularis oculi muscle, and the posterior lamella consists of the eyelid retractors, tarsal plate, and palpebral conjunctiva. The orbital septum that separates the preseptal space from the postseptal orbital contents is referred to as the middle lamella, although this terminology is not consistently used in the literature.[26]

The medial and lateral canthal tendons serve as a fibrous, aponeurotic sling for the upper and lower eyelids. The medial canthal tendon has bony attachments to the anterior and posterior lacrimal crest, surrounding the lacrimal sac. The lateral canthal tendon has bony attachments to Whitnall's tubercle, a bony prominence of the lateral orbital wall posterior to the orbital rim. The lateral canthal tendon and medial canthal tendon receive attachments from the tarsal plates, orbicularis oculi muscle, levator aponeurosis, and Whitnall's ligament superiorly and the capsulopalpebral fascia and Lockwood's ligament inferiorly.[26]

The lacrimal drainage system is intimately associated with the eyelids. The lacrimal puncta are located 6 and 8 mm from the medial canthus in the upper and lower eyelids, respectively.[27] These vertically oriented puncta then transition into a horizontally oriented canaliculus of the upper and lower eyelids, which merge into a common canaliculus and drain into the lacrimal sac, medial to the medial canthus. The orbicularis oculi muscle attachments surrounding the lacrimal sac play an important role in the lacrimal pump and drainage. The lacrimal sac drains into the nasolacrimal duct, which extends into the frontal process of the maxilla and ultimately drains at Hasner's valve in the inferior meatus of the medial maxilla.

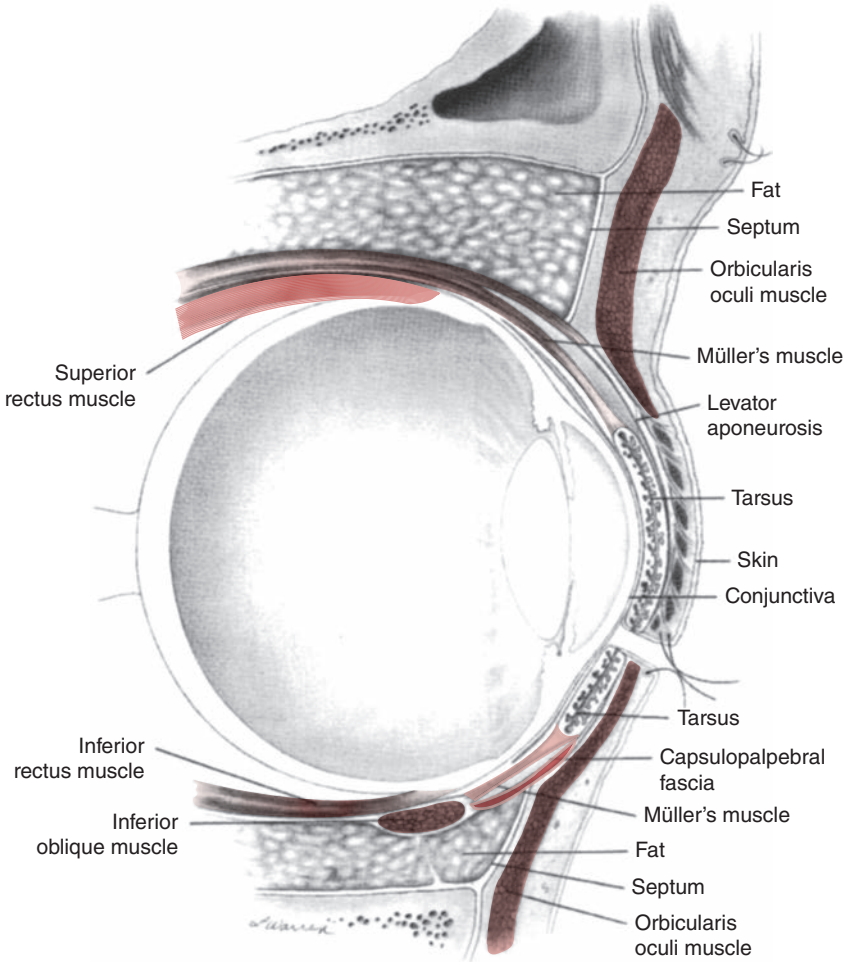


FIGURE 24-7 Complex, multilayer anatomy of the eyelids and surrounding structures. (Reproduced with permission from Wobig J. Eyelid anatomy. In: Putterman AM, ed. *Cosmetic Oculoplastic Surgery*. 2nd ed. Philadelphia, PA: WB Saunders; 1993:73. Copyright © Elsevier.)

EVALUATION

Evaluation of periorbital trauma should include a thorough ocular history and examination of the globe, eyelids with their adnexa, and surrounding bony structures. Diplopia should elevate concern for globe displacement or extraocular muscle entrapment. Visual impairment or abnormalities on examination should prompt early ophthalmologic consultation.

Visual acuity testing should be performed using a standard Snellen chart. In patients who have significant visual impairment, finger counting, hand motion, and light perception should also be tested. Pupils should be examined for asymmetry (anisocoria), shape irregularity, and poor reactivity. Abnormalities in the pupillary exam can suggest an afferent pupillary defect, oculomotor nerve injury, intraocular injury, or Horner syndrome. Red color perception should be performed as monocular red desaturation can indicate optic nerve injury.[28]

Extraocular muscle function evaluation for limited movement can indicate direct muscle injury, muscle entrapment, cranial nerve injury, or severe edema. In a sedated patient who cannot participate in examination or when exam findings are equivocal, forced duction testing can be performed with topical anesthetic and toothed forceps.

Intraocular pressure (IOP) can be tested at the bedside with a tonometer. IOPs greater than 20 mm Hg are considered elevated. Significant ocular injury, periorbital edema, or retrobulbar hemorrhage can elevate ocular pressure, resulting in compression of the optic nerve and its vasculature, ultimately leading to visual compromise and even blindness.[29]

The eyelids and their adnexa should be thoroughly examined for lacerations, tissue loss, and deformity. Traumatic telecanthus can manifest as blunting of the medial canthal angle and widening of the intercanthal distance. Laxity on a bowstring test, where the eyelid is pulled laterally and the medial canthal tendon is palpated, can be suggestive of medial canthal tendon disruption. Integrity of the lacrimal system should be assessed for any soft tissue injury medial to the lacrimal puncta. Punctal dilation with subsequent cannulation with a lacrimal probe can assess the integrity of the canalicular system. Obstruction or exposure of the probe is indicative of injury.

MANAGEMENT

Any suspicion for globe injury should prompt emergent ophthalmology evaluation, and further examination of the eye should avoid significant pressure on the globe.

Intraocular injury, periorbital edema, or retrobulbar hematoma can result in orbital compartment syndrome and require emergent lateral canthotomy and cantholysis to relieve that pressure. Decompression of the orbit is indicated to relieve pressure on the optic nerve; however, the exact indications have been debated. Some have suggested IOP greater than 40 mm Hg in addition to optic nerve compression or central retinal artery occlusion as indications for lateral canthotomy and cantholysis.[30] Others have suggested an IOP of greater than 30 mm Hg.[31]

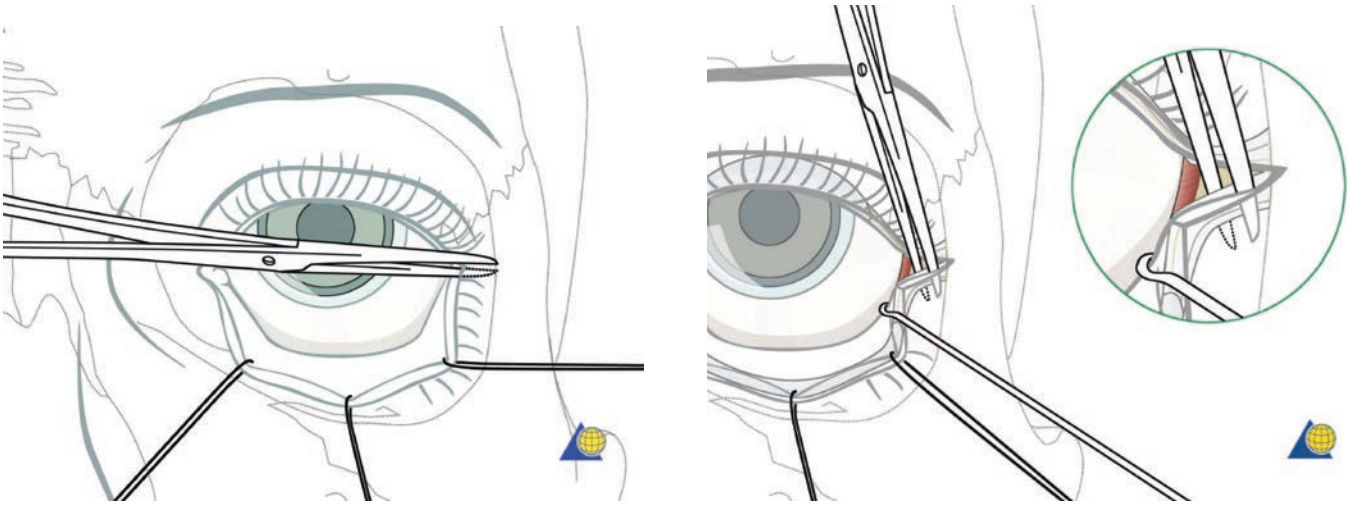
The lateral canthotomy and cantholysis can be performed rapidly at the bedside.[32] The region between the lateral canthus and lateral orbital rim is anesthetized with 1% lidocaine with 1:100,000 epinephrine. The lower eyelid is retracted outward with toothed forceps, and a sharp dissecting scissors is used to make a horizontal incision across the lateral canthus to the lateral orbital rim. Next, the lower eyelid is retracted inferiorly, and the inferior crus of the lateral canthal tendon is incised with the scissors oriented inferiorly (Fig. 24-8).

Partial-thickness lacerations of the eyelids should be explored for potential injury to deeper tissues. In the upper eyelid, injury to the levator aponeurosis necessitates repair to prevent blepharoptosis. This can be done with a 6-0 braided suture in horizontal mattress fashion between the levator aponeurosis and partial thickness of the superior edge of the tarsal plate.[27] The skin can be closed with 6-0 nylon or fast-absorbing gut suture. Care must be taken not to incorporate the septum orbitale, as this may result in eyelid retraction and lagophthalmos of the upper eyelid or ectropion of the lower eyelid.

Full-thickness lacerations and those with margin involvement require a multilayer closure. The conjunctiva is often not closed, particularly if the edges are well approximated, as sutures can cause corneal irritation. The eyelid margin requires meticulous reapproximation and eversion to prevent notching and deformity.[33] Murchison and Bilyk recommend closure beginning with a traction suture of 6-0 silk or polypropylene that is placed at the eyelid margin along the meibomian gland line.[33] The tarsus is then reapproximated with 5-0 or 6-0 polyglactin suture. The eyelid retraction suture is tied down with tails left long. An additional eyelid margin suture is placed at the ciliary line. Furthermore, some advocate yet an additional eyelid margin suture at the gray line, but this risks corneal irritation and is often not necessary.[33] The skin is then closed with 6-0 nylon or fast-absorbing gut suture, incorporating the tails of the eyelid margin sutures[27] (Fig. 24-9).

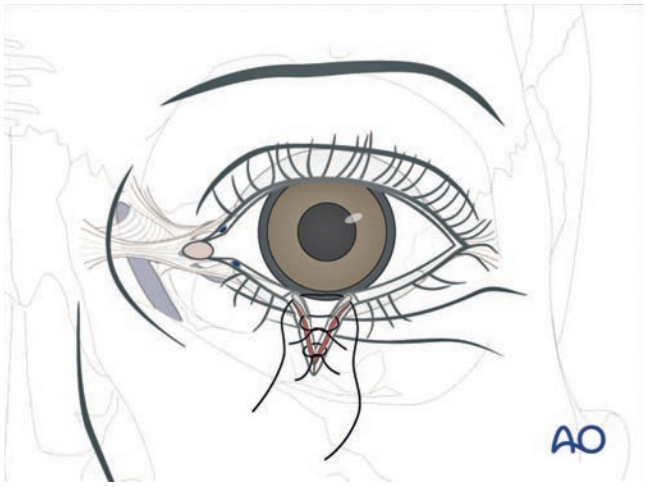
Avulsion injuries can be addressed with primary closure, local advancement flaps, or skin grafts for partial-thickness wounds depending on amount of tissue loss.[27] Full-thickness tissue loss can be primarily closed when less than 25% of the eyelid margin is lost. Large defects require a combination of skin grafting, local rotation/advancement techniques, Cutler-Beard bridge flaps, and Hughes tarsoconjunctival flaps.[27]

The degree of impairment in lacrimal drainage from a monocanalicular injury is controversial, as there are patients with known obstruction who are asymptomatic.[34] Repair is recommended for all patients with canalicular injury given there is no method to predict those who will develop symptomatic epiphora (ie, persistent or excessive tearing). Studies have demonstrated the best outcomes are achieved when repairs are performed by subspecialty trained surgeons and in the operating room setting.[35] The landmark study by Kersten and Kulwin[36] demonstrated that 96% of patients were asymptomatic after reapproximation of the



A B

FIGURE 24-8 Lateral canthotomy. (**A**) Lateral canthotomy is a full-thickness cut made from the lateral canthal margin to the bony lateral orbital rim. (**B**) Inferior cantholysis is made by everting the eyelid and cutting the inferior canthal tendon. (Reproduced with permission from AO Surgery Reference. www.aosurgery.org. Copyright by AO Foundation, Switzerland.)



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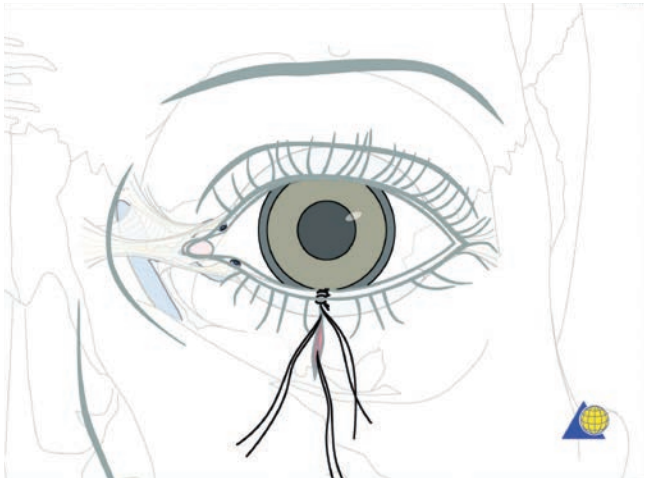

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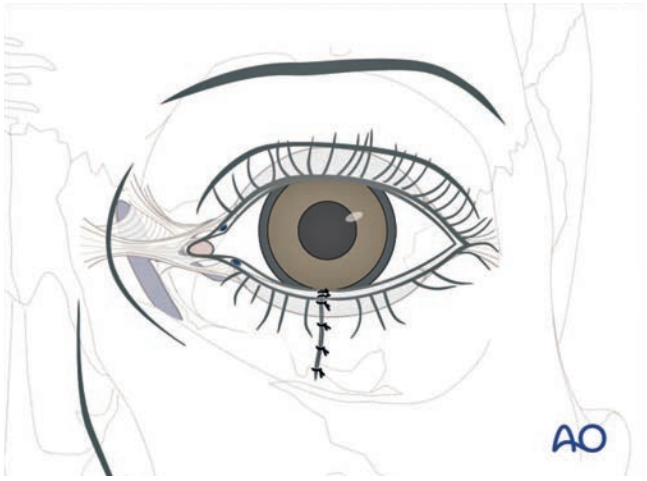
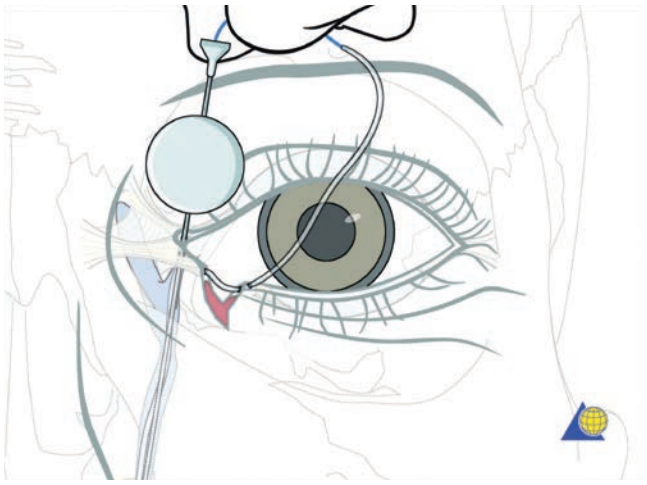


C

FIGURE 24-9 Full-thickness laceration repair of the eyelid. (**A**) Placement of eyelid margin traction suture allows for alignment while tarsal sutures are made. (**B**) Eyelid margin sutures are made at the gray line. (**C**) Skin closure is performed with incorporation of the tails of the eyelid margin sutures to prevent irritation. (Reproduced with permission from AO Surgery Reference. www.aosurgery.org. Copyright by AO Foundation, Switzerland.)



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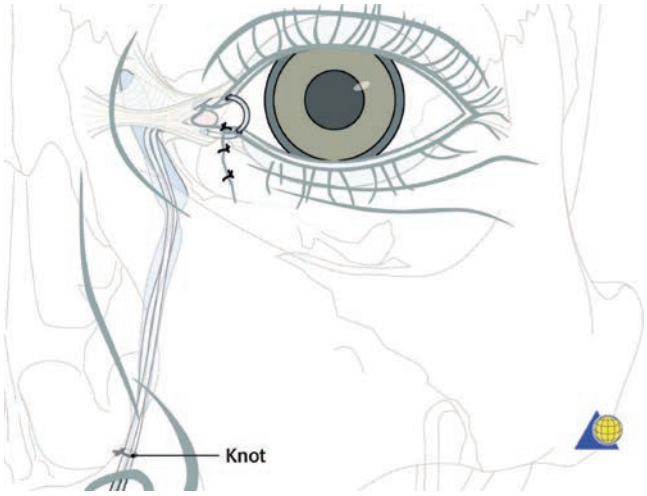


B

FIGURE 24-10 Complex eyelid laceration with canalicular involvement. (**A**) Bicanalicular stents are placed through the lacrimal punctum spanning the laceration into the nasolacrimal duct. (**B**) Resting position of bicanalicular stents. (Reproduced with permission from AO Surgery Reference. www.aosurgery.org. Copyright by AO Foundation, Switzerland.)

pericanalicular orbicular oculi muscle with a 7-0 polyglactin suture and bicanalicular stenting (Fig. 24-10). Studies have demonstrated no difference in outcomes when monocanalicular stents versus bicanalicular stents are used for monocanalicular injuries.[35] The most common monocanalicular stent is a Mini Monoka (FCI Ophthalmics, Marshfield Hills, MA) stent. Bicanalicular stent options include the Guibor (Guibor Canalicular Intubation Set; Xomed Surgical Products Inc, Jacksonville, FL) and Crawford (JedMed, St. Louis, MO) tubes.

Nose

ANATOMY

The nose is frequently injured in trauma to the face given its prominent projection on the facial contour. The nose

consists of a bony and cartilaginous framework that contains a complex external soft tissue envelope and internal mucosal lining. The nasal bones have a pyramidal shape and are bordered by the nasal process of the frontal bone superiorly, frontal process of maxilla laterally, upper lateral cartilage inferiorly, and septum on its deep surface. All of these structures together with the lower lateral cartilages and sesamoid cartilages play an important role in the strength and structure of the nose.

The external nose can be subdivided into nine aesthetic subunits: the midline nasal dorsum, tip, and columella, as well as the paired lateral sidewalls, ala, and soft tissue triangles. The septum plays a critical function in nasal support, appearance, and function. Anteriorly, the septum consists of the quadrangular cartilage. Posteriorly, the septum has bony contributions from the perpendicular plate of the ethmoid superiorly and the vomer, maxilla, and palatine bones inferiorly.

EVALUATION

The nose should be evaluated for any aesthetic and functional changes. Evaluating the frontal and base views of the nose can give insight into any asymmetry or deviation. Palpation of step-offs or bony mobility suggests an underlying fracture. Anterior rhinoscopy evaluates for septal injury, mucosal disruption, and CSF leak. The septum should also be palpated for underlying septal hematoma. Any laceration should be assessed for cartilage exposure or full-thickness injury. Avulsion injuries should be characterized based on nasal aesthetic subunits involved and layers of tissue involved.

MANAGEMENT (SEE ATLAS FIGURE 4)

Lacerations without significant tissue loss should be closed in a multilayer fashion. For full-thickness injuries, the internal mucosal lining of the nose should be reapproximated with 4-0 to 6-0 absorbable sutures.[37] Silastic stents can be placed for 2 to 4 weeks to limit stenosis when the internal lining is violated.[37] Cartilage lacerations are reapproximated with 4-0 polydioxanone, clear nylon, or poliglecaprone suture in horizontal mattress fashion. Deep dermal sutures may be required to decrease tension. The skin layer is then closed with judicious nonreactive suture to limit foreign body reaction and strangulation.[37] Tissue eversion is critical, particularly at the alar rim, and can limit notching.

Avulsion injuries with missing tissues pose a reconstructive challenge. Timing of reconstruction is balanced between delaying reconstruction for adequate debridement in heavily contaminated or bite wounds and early intervention to prevent soft tissue contracture and distortion. Ideal reconstructive method depends on location of defect, need for free cartilage graft coverage, and patient comorbidities. Cartilage grafting is required for missing cartilage, reestablishing nasal support, and preventing contracture and notching at edges such as the alar rim.

For small avulsion injuries, full-thickness skin grafting, composite chondrocutaneous grafts, and rotational advancement techniques (eg, single-lobed, bilobed, and dorsal nasal flaps) can be used.[38] For larger defects or if free cartilage grafts need vascularized tissue coverage, locoregional flaps (eg, nasolabial or paramedian forehead flaps) are preferred.

Lips

ANATOMY

Even minor abnormalities to the lips can be quite noticeable. Additionally, the lip participates in facial expression, speech, and alimentation.[39] Therefore, management of lip trauma must address restoring function and aesthetic units.

The lip consists of three major layers: cutaneous, orbicularis oris muscle, and mucosa. Reapproximating each of these layers is critical in managing lip lacerations and defects. The surface anatomy of the upper lip consists of a cutaneous lip and vermilion lip (Fig. 24-11). The junction of these subunits forms the vermilion-cutaneous junction (VCJ). The cutaneous roll is an area of convexity of the cutaneous lip at the VCJ. Reflecting light at the cutaneous roll makes it a prominent feature of the lip. Even minor disruption of the cutaneous roll or VCJ can be aesthetically unpleasing. The contour of the VCJ is often referred to as the Cupid's bow, with the peaks correlating to the position of the philtral columns and the trough correlating to the philtral dimple. The contour of the lower lip VCJ is curvilinear with simpler surface anatomy. The lower lip junctions with the chin subunit at the mentolabial sulcus.

EVALUATION

Lip trauma can be associated with intraoral and dental injury. Lacerations should be probed to assess full-thickness and intraoral involvement. A thorough dental evaluation for loose, avulsed, or chipped teeth should be performed. Dental consultation and management of dental injury are best performed prior to closure of lacerations to limit traction on soft tissue repair.[40] Extension of wounds into the floor of the mouth or buccal mucosa should be evaluated for salivary duct injury.

MANAGEMENT

Local anesthesia with nerve blocks is critical in lip laceration repair as direct infiltration into the wound can distort landmarks. Infraorbital and mental nerve blocks are used

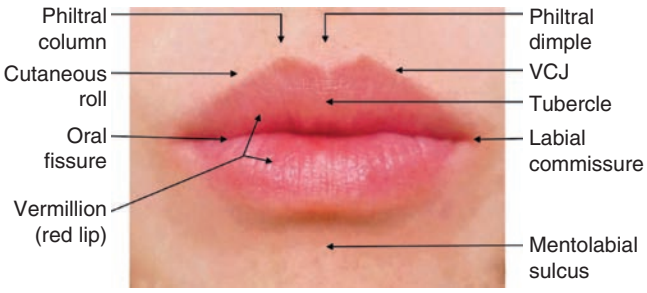


FIGURE 24-11 Surface landmarks of the lip. VCJ, vermilion-cutaneous junction.

for upper and lower lip lacerations, respectively. The superior labial and inferior labial arteries form the major blood supply for the upper and lower lips and can result in significant bleeding. Pressure on the lip with a full-thickness grasp between the thumb and index finger can help with immediate hemostasis. Local anesthetic infiltration with epinephrine in the region of the artery lateral to the oral commissure can help with vasoconstriction.

Primary closure of full-thickness lip defects should be done in a layered fashion, with attention to the mucosa, orbicularis oris muscle, and skin. Muscle closure is performed with 4-0 polyglactin or polydioxanone suture. Dehiscence in the muscle layer can result in an irregular depression, and failure to close the marginal aspect of the muscle can result in notching or a "whistle" deformity. After the muscle is reapproximated, the key step is realignment of the vermilion border, as even a 1-mm error can be aesthetically displeasing.[40] A 6-0 nylon or polypropylene suture is used to reapproximate the vermilion border. A 5-0 chromic suture is used for the remainder of the vermilion and mucosal closure, and skin is closed with 6-0 nylon or polypropylene suture.

Reconstruction of avulsion injuries requires consideration of percentage of tissue loss, philtral or commissure involvement, and degree of resulting microstomia. Generally, defects less than 30% can be closed primarily. Larger defects require complex tissue rearrangement, including bilateral advancement, Abbe or Estlander lip switch, or Karapandzic, Bernard-Burrow, or Gilles fan flaps.[40]

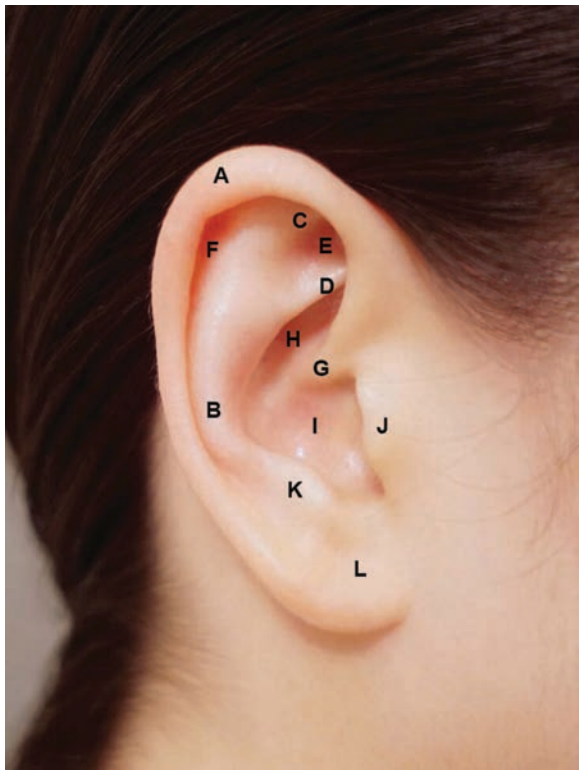


FIGURE 24-12 Surface anatomy of auricle. A, helix; B, antihelix; C, superior crus of antihelix; D, inferior crus of antihelix; E, triangular fossa; F, scaphoid fossa, G, root of helix; H, concha cavum; I, cymba concha; J, tragus; K, antitragus; L, lobule.

Auricles

ANATOMY

The auricle is derived embryologically from the six hillocks of His, with the first three hillocks giving rise to the tragus, helix, and helical crus and the latter giving rise to the antihelix, antitragus, and lobule.[41] The auricle protrudes from the skull base at an angle of 25° to 30° and angles 15° away from the vertical axis.[42] The external anatomy of the auricle is shown in Fig. 24-12.

EVALUATION

Auricular wounds should be examined for depth of laceration, cartilage involvement, and avulsed tissue. Bruising over the mastoid (Battle sign) could be indicative of a skull base fracture and should raise suspicion for underlying temporal bone fracture. Otoscopic examination should asses the external auditory canal for lacerations and tympanic membrane for perforations or hemotympanum.

MANAGEMENT

After local wound care, lacerations can be managed with reapproximation of the skin and perichondrium with the goal of reapproximating the underlying lacerated cartilage.[42] Cartilage sutures are not necessarily required. Significant extension into the external auditory canal warrants consideration for stenting.[42]

Avulsion injuries can be divided into complete and partial avulsions. Avulsion injuries with a wide base or those with a narrow base with small size of avulsed tissue can be primarily repaired with reapproximation of the perichondrium and skin. Avulsion injuries with a narrow base and large size can have a compromised blood supply, and the patient and surgeon should monitor for impending tissue loss with selective debridement and reconstruction of any demarcated tissue.

Completely avulsed tissue can be considered for microvascular anastomosis and reimplantation if tissues are appropriately handled.[43] The avulsed tissue should be wrapped in gauze, placed in a plastic bag, and submerged in a 4°C ice water bath.[42] Postauricular banking techniques have been abandoned by some due to high resorption rates and disruption of the postauricular soft tissue, if needed, for more complex auricular reconstruction.[43] Auricular prostheses are an option for those who do not desire autologous rib cartilage reconstruction or in patients with significant medical comorbidities precluding prolonged anesthetic.

Any area where the perichondrium has been lifted away from the cartilage should be bolstered to prevent blood collection and cauliflower ear deformity. Similarly, auricular hematomas should be drained and overlying soft tissue reinforced with a bolster. Various bolster techniques, including dental rolls or ointment-impregnated gauze with horizontal mattress suture, can be used.[41]

MANAGEMENT OF MAXILLOFACIAL SKELETAL TRAUMA

The facial skeleton can be categorized into horizontal thirds: cranial vault, midface, and mandible. The cranial vault includes the frontal sinus. Midface fractures include orbital, nasal bone, naso-orbito-ethmoid (NOE), zygomaticomaxillary complex (ZMC), and Le Fort fractures. Fractures involving two or more of the facial thirds are considered panfacial fractures.

Radiography

Multiplanar thin-cut (0.6–1.5 mm) facial computed tomography (CT) imaging is the gold standard for maxillofacial trauma. Axial, coronal, and sagittal reconstructions should be available for accurate diagnosis. Three-dimensional CT images improve visualization of the shape, size, and orientation of individual bone fragments in complex fractures and ultimately help with surgical planning and patient counseling.

Occlusion and Intermaxillary Fixation

In dentistry, occlusion represents the static contact between teeth of the maxilla and mandible. In 1899, Edward Angle was the first to classify malocclusion, or the malalignment of teeth when the jaws close.[44] He defined normal occlusion as the dental relationship where the mesiobuccal cusp of the maxillary first molar lies within the buccal groove of the mandibular first molar. Class I malocclusion has the same molar relationship as in normal occlusion with irregularities in spacing or rotation. Class II malocclusion is the dental relationship where the mesiobuccal cusp of the maxillary first molar is anterior to the mesiobuccal groove of the mandibular first molar (retrognathia). Class III malocclusion is defined by the mesiobuccal cusp of the maxillary first molar lying posterior to the mesiobuccal groove of the mandibular first molar (prognathia).

Occlusion plays a critical role in maxillofacial reconstruction. Dental relationships can provide a template for aligning fractures and are the basis of maxillomandibular, or intermaxillary, fixation to ensure that fracture repair is performed in an orientation where the patient's functional occlusion is restored. It is important to note that patients may not have normal occlusion prior to their injuries. Therefore, photographs and dental records play a key role in establishing the patient's premorbid occlusion. Dental ware facets point to areas of prior dental contact and help maxillofacial surgeons reestablish premorbid occlusion.

Options for maxillomandibular fixation include Erich arch bars (gold standard), Ivy loops, intermaxillary fixation screws, embrasure wires, hybrid arch bars, gunning splints, and, more historically, circummandibular-transpyriform wires. In patients with seizure disorders, those suffering from alcohol abuse, and patients who could not protect their airway in the event of vomiting, long-term use of maxillomandibular fixation is contraindicated.

Intraoperative Computed Tomography

The use of intraoperative CT in maxillofacial reconstruction has evolved with the advent of portable CT scanners. Intraoperative CT allows for assessment of reduction in areas where direct visualization is difficult, such as the orbit and zygoma.[45] Intraoperative assessment allows for immediate modification, obviating the need for often more expensive and difficult delayed revisions.[46] Additionally, immediate assessment can eliminate the morbidity of additional fracture exposure sites that would otherwise be required to confirm reduction and, in the case of ZMC fractures with orbital floor involvement, determine the need of orbital floor reconstruction. Shaye et al[45] demonstrated a revision rate of 24% with an average intraoperative imaging time of 14.5 minutes, suggesting that in select cases, intraoperative imaging can be efficient and prevent delayed revision. The rate of revision is biased by the lower threshold to make adjustments while still in the operating room and the bias against additional revision surgery once the patient has left the operating room. Additionally, the risks of radiation must be balanced with the benefit of immediate revision.

Rigid Fixation

Reconstitution of skeletal support is the foundation of maxillofacial reconstruction. Reduction and fixation of fractured skeletal elements restores proper functioning and allows optimal bony healing.[47] The evolution of internal fixation hardware technologies has allowed for optimal rigid fixation of fractures. Rigid fixation prevents micromovements of the fractured segments, allowing formation of a delicate blood supply and promoting primary bone formation without callus formation.[48] Lack of adequate fixation increased the chance of nonunion and wound complications.[48] The added benefit of rigid fixation is to avoid postoperative maxillomandibular fixation and sequelae such as temporomandibular joint ankylosis.[49]

Frontal Sinus Fractures

ANATOMY AND ETIOLOGY

The frontal sinus is the last of the paranasal sinuses to develop and is absent at birth. Pneumatization of the frontal sinus begins around age 2 years and reaches adult size at age 15 years.[50] The frontal sinus is bounded by the anterior table, which contributes to the bone underlying the forehead, glabella, and brow. The posterior table of the frontal sinus separates the sinus from the anterior cranial fossa, and the floor of the frontal sinus is the orbital roof.

The frontal sinus requires significant force to fracture.[51] Given the high-energy mechanism required to generate that force, it is not surprising that 75% of patients with these fractures have serious concomitant injuries.[52] Thirty-three percent of frontal sinus fractures are isolated anterior table fractures, and 67% involve a combination of the anterior table, posterior table, and frontal recess.

EVALUATION

Soft tissue injury overlying the forehead, particularly in the context of high-energy trauma, should raise suspicion for underlying frontal sinus fracture. Palpation of any bony step-offs on the forehead or visible bony depression suggests a fracture, although edema in the acute setting can obscure these findings. The patient should be asked about signs and symptoms of CSF leak, including clear rhinorrhea and salty-tasting drainage. Clear rhinorrhea noted on examination can be collected for a bedside "halo" test, where fluid is placed on filter paper and CSF diffuses faster than blood, leaving a clear halo around the blood. Beta-2 transferrin analysis can also be performed but has limited acute utility given it is generally a send-out laboratory test that can take up to a week to obtain results. A thorough ophthalmologic exam should be performed given the shared wall of the frontal sinus floor and orbital roof.

MANAGEMENT

The goals of frontal sinus fracture management include restoring normal frontal sinus function or obliterating an unsafe sinus, treating CSF leaks, and restoring frontal bone contour. Strong[50] outlined five major considerations that guide management of frontal sinus fractures: anterior table fracture, posterior table fracture, frontal recess injury, dural injury/CSF leak, and fracture comminution (Fig. 24-13).

FRONTAL RECESS INJURY

Patients with frontal recess injury as part of their frontal sinus fracture are at risk of frontal sinus outflow obstruction and late complications of mucocele or complicated sinusitis. Nondisplaced or minimally displaced fractures at the frontal recess can be observed with repeat imaging in 3 months. If there is aeration of the frontal sinus suggestive of restoration of frontal sinus function, the patient can be observed without surgical intervention. If the frontal sinus is opacified, then endoscopic sinus surgery with frontal sinusotomy can be pursued to restore sinus outflow. There has been a trend toward observation and frontal sinus preservation.[53] Studies have demonstrated spontaneous ventilation of the frontal sinus[54,55] in fractures involving the frontal recess. Smith et al[54] demonstrated that five of seven patients with anterior table and frontal recess fractures had spontaneous ventilation. The other two patients were successfully managed with endoscopic sinus surgery after failed medical therapy with antibiotics, systemic steroids, and topical nasal steroids. Jafari et al[55] demonstrated that seven of eight patients with frontal recess fractures had spontaneous ventilation.

For patients with moderate or severe frontal recess fractures and frontal sinus outflow impairment, frontal sinus obliteration or cranialization should be performed depending on the status of the anterior and posterior tables.

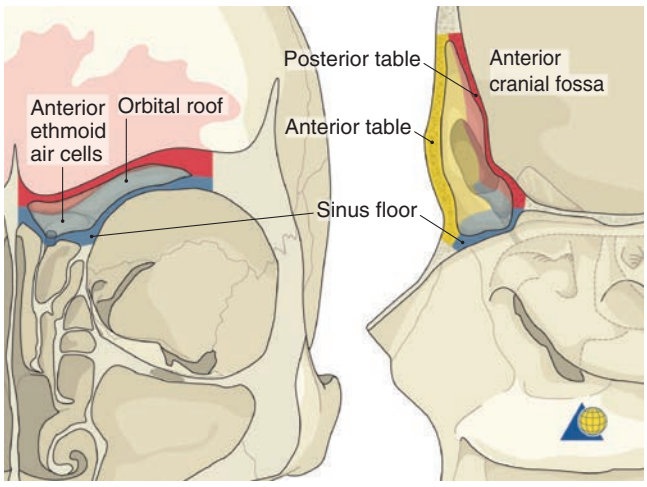


FIGURE 24-13 Anatomy of the frontal sinus. Yellow = anterior table, red = posterior table, blue = sinus floor and frontal recess. (Reproduced with permission from AO Surgery Reference. www.aosurgery.org. Copyright by AO Foundation, Switzerland.)

ANTERIOR TABLE

Anterior table fractures that have less than 1 to 2 mm of displacement can be observed and are unlikely to cause significant frontal contour deformity.[50] Fractures with greater displacement, while posing little risk of mucocele formation, are more likely to have aesthetic deformities. After waiting 2 to 4 months for resolution of postinjury edema, patients can undergo endoscopic repair with porous polyethylene implant to camouflage the frontal contour deformity. By waiting for the edema to resolve, patients can make an educated decision for surgical intervention. Often the deformity is less noticeable than anticipated, and the patient may desire no intervention.[50] Endoscopic repair is performed similar to an endoscopic brow lift with a medial 1- to 2-cm subperiosteal endoscope incision in the hairline and 3- to 5-cm parasagittal working incision. The porous polyethylene is contoured to the defect size and sometimes layered. The implant is then fixated with a percutaneous 1.7-mm self-drilling screw (4–7 mm in length). A prominent forehead or receding hairline may require exposed incisions or preclude the endoscopic approach due to limited visualization around forehead curvature.

Open reduction, internal fixation via a bicoronal approach is an alternative approach. The coronal incision can be designed in a zigzag fashion 4 to 6 cm behind the hairline for patients who wear their hair long and in a straight line, or widow's peak fashion for patients who wear their hair short. The incision is carried down from temporal line to temporal line in the subgaleal plane, protecting the pericranium as a potential flap. Lateral to the temporal line, the dissection plane is carried below the TPF, thereby protecting the temporal branch of the facial nerve. The incision can be carried in a pre- or postauricular position laterally. The pericranial flap is incised from temporal line laterally to 2 cm posterior to the coronal incision and elevated in a subperiosteal plane to the superior orbital rims (Fig. 24-14). Further exposure can be obtained by releasing the supraorbital neurovascular bundle from the foramen with osteotomies. The anterior table fragments can then be reduced with a combination of

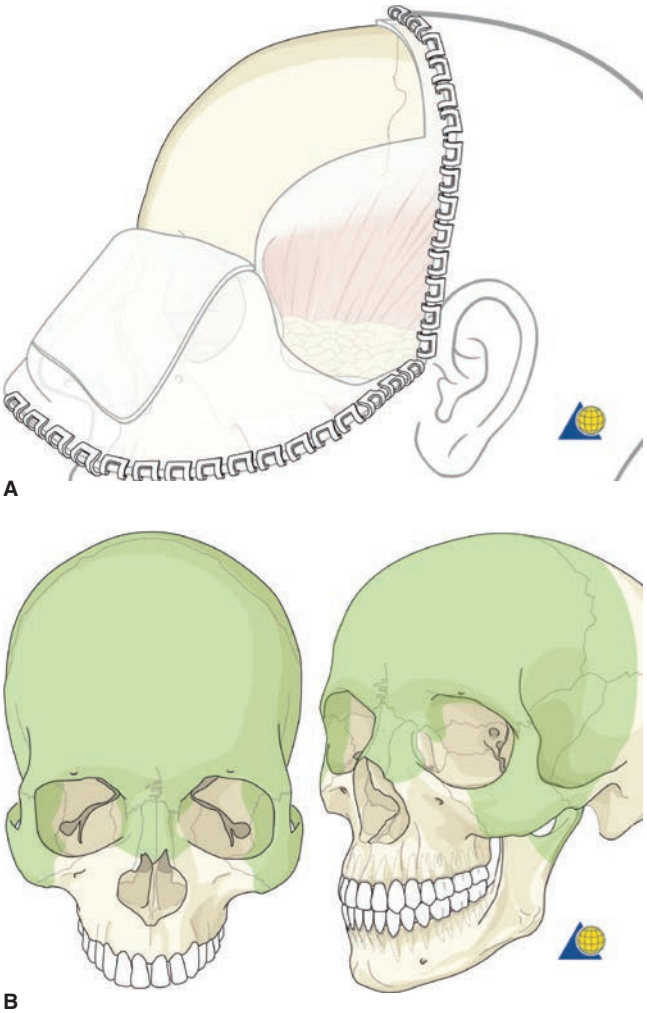


FIGURE 24-14 Exposure of the anterior table of the frontal sinus. (**A**) Coronal flap. (**B**) Access provided by a coronal approach. (Reproduced with permission from AO Surgery Reference. www.aosurgery.org. Copyright by AO Foundation, Switzerland.)

bone hooks or 1.5- to 2.0-mm screws placed in the depressed segment and retracted with a heavy hemostat. Fragments can be plated with 1.0 to 1.3 microplates, with small gaps of 4 to 10 mm being reconstructed with titanium mesh.[50] Mucosa in the fracture lines should be removed thoroughly. If reduction cannot be performed, sinus obliteration may be required.

POSTERIOR TABLE

Posterior table fractures with minimal displacement (less than one table width) can be observed. In the presence of CSF leak, these fractures can be observed with the patient on strict skull base precautions for 7 days. These precautions include bedrest, no nose blowing, no open mouth sneezing, no Valsalva maneuvers, and no noninvasive positive-pressure ventilation. Persistent CSF leak after a period of observation generally requires frontal sinus obliteration or cranialization. Similarly, significantly displaced fractures (greater than one table width) benefit from frontal sinus obliteration or, in the presence of significant comminution or CSF leak, cranialization for repair of dural injury.[50]

Obliteration begins with exposure of the anterior table with a coronal approach. Multiple methods for identifying the boundaries of the anterior table include transillumination, 6-foot penny Caldwell x-ray, surgical navigation, or use of bayonet forceps with one tine inside the sinus palpating the edges and the exterior tine serving as the marker for the outline of the sinus. The anterior table is drilled out, and the mucosa is meticulously removed from the undersurface and the remainder of the sinus cavity. The residual mucosa of the frontal sinus infundibulum is inverted toward the nasal cavity and plugged with temporalis muscle. Fat harvested from the abdomen is used to fill the sinus cavity, and the anterior table bone set aside previously is plated using microplates.

Cranialization is usually performed in consultation with a neurosurgeon. The procedure begins similarly to the obliteration. After the mucosa is removed from the sinus, the posterior table bone is removed and any dural injury is repaired. The frontal recess is plugged with temporalis muscle fascia, and if further reconstruction is required, the pericranial flap is placed along the skull base. The anterior table bone is then replaced, with a small inferior gap to prevent strangulation of the pericranial flap.

Nasal Bone

ANATOMY

See section on nose in Management of Facial Soft Tissue Trauma.

EVALUATION

History should be obtained about the mechanism of trauma, and necessary trauma workup should be initiated for suspected concomitant injuries. Patients should be asked about perceived changes to the appearance of their nose. History of prior trauma and nasal surgeries should be obtained. Pictures can help differentiate prior deformities from acute changes. Clear rhinorrhea or salty taste should elevate concern of associated skull base injury and CSF leak.

Physical examination includes palpating for any bony step-offs or mobile bony segments. Nasal examination with anterior rhinoscopy or endoscopy can identify septal deviation, mucosal lacerations, and septal hematoma. Palpation of the nasal septum is a critical component in diagnosing septal hematomas.

MANAGEMENT

The goal of treating nasal bone fractures is to restore premorbid appearance and nasal airway. Patient factors play a large role in the decision making for intervention, including age, aesthetic expectations, and medical comorbidities. Mild or minimally displaced nasal bone fractures may not result in nasal obstruction or noticeable deformity. If the patient has significant edema at the time of presentation, reevaluation after 3 to 5 days (but before 2 weeks) to allow the edema to subside may unmask deformities.[56] Options for patients

with nasal deformity and anatomic nasal airway compromise include closed reduction or open fracture treatment via septorhinoplasty approach.

Closed reduction techniques can be successful in select patients and fractures. The procedure can be performed under local or general anesthesia[57]; 1% lidocaine with 1:100,000 epinephrine is injected along the lateral aspect of the nasal bones, premaxilla, and septum. Additional nerve blocks are performed, including the infraorbital, external nasal, and infratrochlear nerves.[56] Pledgets soaked in 4% cocaine provide additional anesthesia and vasoconstriction. A Boies elevator is placed in the nasal cavity and against the nasal bone. The instrument should be marked or gripped at the distance between the external nares and medial canthus so as to protect the skull base (Fig. 24-15). The surgeon's opposite thumb and index finger should be placed externally on the nasal bones to palpate and control the reduction. Reduction begins on the side of the concavity for deviated fractures.[56] The bony pyramid is pulled anteriorly and toward the side of concavity to restore anatomic alignment. Ash forceps can be used to reduce septal fractures and are often required to obtain adequate nasal bone reduction due to the relationship between the nasal bones and septum. After reduction is complete, the bony pyramid should be splinted with a Denver or thermoplastic splint and the septum with a silastic splint.

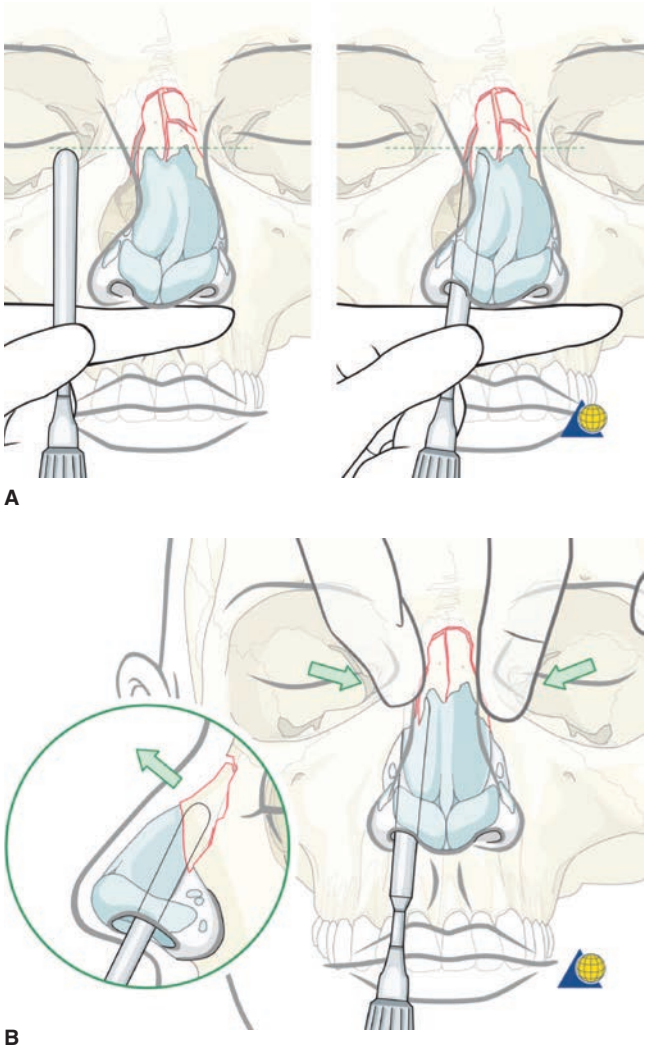


FIGURE 24-15 Closed reduction of nasal bone fractures. (**A**) The Boies elevator should be placed below the level of the skull base to prevent iatrogenic injury. (**B**) The surgeon's opposite thumb and index finger should be placed externally on the nasal bones to palpate and control the reduction. (Reproduced with permission from AO Surgery Reference. www.aosurgery.org. Copyright by AO Foundation, Switzerland.)

In complex fractures with septal involvement, severe skeletal comminution, and pyramidal instability, closed reduction techniques may result in poor outcomes and high revision rates.[58] In a detailed literature review, Staffel[59] found that surgeons were dissatisfied 37% of the time with closed reduction techniques alone. Multiple series have also demonstrated revision rates as high as 50%.[58] With the evolution of contemporary rhinoplasty techniques, there has been a shift toward early open fracture treatment. Staffel[59] demonstrated improved satisfaction from 40% with closed reduction techniques to 71% with rhinoplasty techniques including septoplasty, osteotomies, upper lateral cartilage release, anterior perpendicular plate fracture, and camouflage grafting.

Septal hematomas should be drained to prevent septal abscess, cartilage necrosis, and saddle nose deformity. After local anesthesia is injected into the septum, the mucoperichondrium overlying the hematoma should be incised bilaterally, but in nonopposing locations to prevent perforation. Once the hematoma is drained, nasal packing should be placed for 7 days to prevent reaccumulation of blood. Prophylactic antibiotics are suggested to prevent toxic shock syndrome while packing is in place.

Midface Fractures

ANATOMY

The structure of the midface is tightly related to its adaptation to masticatory forces. The shape of the midface is also related to paranasal sinus pneumatization, which is involved in vocal resonance, lightening the weight of the skull, and energy absorption.[60] The resulting structure of the midface can be thought of as a lattice-like structure of vertical and horizontal buttresses,[61] where the buttresses represent areas of stronger bone.

The four vertical buttresses are as follows: (1) nasomaxillary (also known as the medial or frontonasomaxillary) buttress, (2) zygomaticomaxillary (also known as the lateral or frontozygomaticomaxillary) buttress, (3) pterygomaxillary buttress, and (4) ethmoid-vomerian (or bony septal) buttress. The horizontal buttresses include the following: (1) superior orbital rim, (2) inferior orbital rim with extension to the zygomatic arch, and (3) maxillary alveolus/palate.

Common fracture patterns of the midface include nasal bone, NOE, ZMC, orbital, and Le Fort fractures.

EVALUATION

A thorough head and neck examination is required for every facial fracture. For the midface, examination of any

asymmetry, decreased malar projection, or gross deformities may indicate an underlying fracture. Patient pictures prior to the injury can help provide a baseline for assessment. The face should be palpated for bony step-offs or mobile segments. Bimanual examination with stabilization of the head at various levels can assess for palatal or midface mobility suggestive of dentoalveolar, palatal, or Le Fort fractures. Trismus may indicate fracture of the zygomatic arch and impingement or spasm of the underlying temporalis muscle. Oral examination should include quality of dentition (which has implications on method of intermaxillary fixation), loose dentition, and malocclusion. History of prior dental or orthognathic procedures and records should be obtained, particularly when the fracture pattern does not match the degree of malocclusion. A thorough cranial nerve and ophthalmologic examination should be performed.

ORBIT MANAGEMENT

Orbital fractures can be classified as trap door (greenstick), floor, medial wall, two wall, roof, or complex fractures. These fractures can occur in isolation or in the setting of concomitant midface fractures. Orbital "blowout" fractures occur from a combination of increased orbital pressure ("hydraulic" mechanism) and direct impact to the infraorbital rim ("buckling" theory).[62]

Although most orbital fractures can be treated within the first 2 weeks, there are certain circumstances under which emergent intervention is indicated. The oculocardiac reflex is a vagally mediated reflex with afferent signaling from the ophthalmic division of the trigeminal nerve, resulting in nausea, bradycardia, and potentially asystole. The stimulus is thought to be secondary to orbital pressure or entrapment of orbital contents.[63]

Extraocular muscle entrapment is another indication for immediate repair and is most commonly described in a greenstick or "trap door" fracture of the orbital floor. Orbital contents, including the inferior rectus muscle, displace into the maxillary sinus at the time of impact and are then entrapped as the hinged bone segment retracts into its original position. Prolonged entrapment can lead to ischemia and fibrosis of the muscle and therefore requires emergent intervention.

The goal of routine orbital fracture management is restoration of orbital volume to prevent delayed enophthalmos, hypoglobus, and diplopia. Not all blowout fractures require intervention. Some have suggested that 2 cm^2 or 50% of the area of the orbital floor is predictive of delayed enophthalmos and diplopia.[63] Distortion of the shape of the inferior rectus from a horizontally flat structure to a more rounded or vertically elongated structure is predictive of enophthalmos.[63]

The orbit can be accessed via a transconjunctival, subciliary, or subtarsal approach. Because the subciliary approach has a higher risk of cicatricial ectropion and the subtarsal approach results in visible scarring, the transconjunctival approach is favored[64] (Fig. 24-16). While autologous bone is the gold standard for orbital reconstruction, titanium and porous polyethylene are now the most commonly used materials.[62]

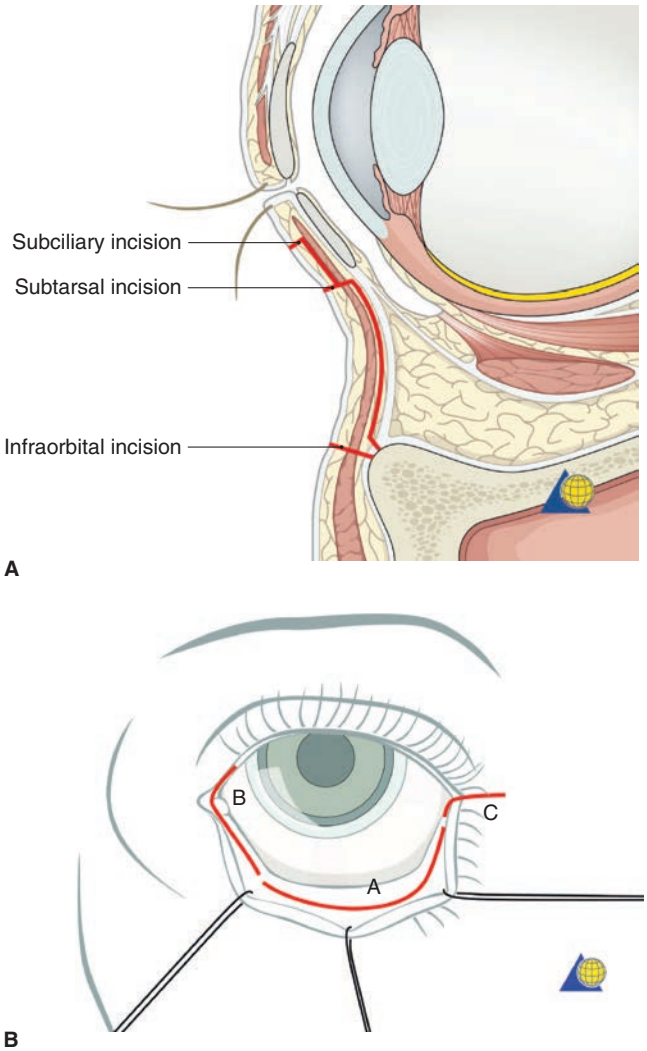


FIGURE 24-16 Incisions to approach the orbit. (**A**) Cutaneous orbital approaches via subciliary, subtarsal, and infraorbital incisions. (**B**) Transconjunctival approaches with medial and lateral extension. Incision A demonstrates the standard transconjunctival approach, with incision B representing a transcaruncular approach for medial extension and incision C representing a lateral canthotomy for lateral extension. (Reproduced with permission from AO Surgery Reference. www.aosurgery.org. Copyright by AO Foundation, Switzerland.)

ZMC MANAGEMENT

The ZMC describes fractures of the zygoma and its surrounding articulations. The zygoma has four articulation sites: zygomaticomaxillary suture, frontozygomatic suture, zygomaticosphenoid suture, and temporal process of the zygoma. The temporal process of the zygoma forms the anterior portion of the zygomatic arch. The zygoma contributes to the lateral orbital wall, orbital rim, and orbital floor, and therefore concomitant orbital fractures also need to be taken into consideration in the management of ZMC fractures.

The goals in repair of ZMC fractures include reestablishing facial width, malar projection, and orbital volume. Exposure

of a given articulation of the ZMC allows for confirmation of reduction and opportunity for internal fixation. This must be balanced with the morbidity of the exposure. Preexisting lacerations can provide direct exposure with minimal extension.

The best site for evaluation of reduction is the zygomaticosphenoid suture given the long segment and multiplanar articulation with the sphenoid bone.[65] The best sites for internal fixation include the (1) zygomaticomaxillary fracture line due to thicker bone and vector of fixation counteracting the pull of the masseter (2) and frontozygomatic suture due to thicker bone.

Management of ZMC fractures can range from observation with soft diet to four-point fixation, with fixation sites including the zygomaticomaxillary, frontozygomatic, zygomaticotemporal, and infraorbital rim. Minimally displaced, noncomminuted, and stable fractures with low risk for malar depression are appropriately managed with observation and a soft diet.

Manipulation of the ZMC for reduction can be performed via a percutaneous Carroll-Girard screw, sublabial Keen approach, or temporal Gilles approach. Closed reduction alone can be performed for mildly displaced fractures without comminution. Areas of comminution or significant displacement require open reduction with internal fixation.

The sublabial approach is performed with an incision in the gingivobuccal sulcus. A mucosal incision is made with care to leave an adequate cuff of mucosa for closure with care to protect the papilla of the parotid duct. Cautery is used to enter a subperiosteal plane. A periosteal elevator is used to elevate over the face of the maxilla, laterally over the zygoma, medially toward the pyriform aperture, and superiorly to the orbital rim, with care to dissect around the infraorbital neurovascular pedicle. This approach would allow plating at the zygomaticomaxillary buttress with a midface L-plate (0.7 mm thick, or 1.5/2.0 mm plates using older nomenclature). Infraorbital rim plates with curved midface plates can be accessed using this approach,[66] although orbital approaches may provide easier access to this area (as described in the orbit section).

The frontozygomatic suture can be accessed via an upper blepharoplasty approach. The incision is planned in the lateral aspect of the natural supratarsal skin crease. An incision is made through the skin, and dissection is carried through the orbital part of the orbicular oculi muscle. Dissection continues laterally to the superolateral orbital rim, where subperiosteal dissection reveals the frontozygomatic suture. Curved midface plates (0.5 mm thickness, or 1.3 mm by older nomenclature) can be used for internal fixation.

Exposure of the zygomatic arch for internal fixation requires a coronal approach and carries the morbidity of a large incision, alopecia, scarring, and risk of damage to the temporal branch of the facial nerve. Generally, this approach is reserved for significant comminution, displacement, and telescoping of the zygomatic arch.[65] The approach begins as described in the frontal sinus fracture section. Laterally, as the TPF is elevated, the intermediate temporal fat pad is identified, and the superficial layer of the deep temporal fascia is incised to enter the fat pad above the level of the zygoma. Dissection is performed through the fat pad onto the superficial aspect of the arch, followed by subperiosteal dissection from the superficial to lateral aspect of the zygomatic arch. This dissection plane protects the temporal branch of the facial nerve as it crosses over the lateral surface of the zygomatic arch. The introduction of intraoperative CT has allowed for confirmation of reduction without requiring the coronal incision and arch approaches in many patients.

NOE MANAGEMENT

The NOE region is a confluence of the nose, frontal process of the maxilla, orbit, ethmoid, lacrimal bone, and anterior skull base.[52] The hallmark clinical finding of an NOE fracture is telecanthus due to displacement of the bony segment attached to the medial canthal tendon, termed the *central fragment*, or disruption of the medial canthal tendon itself.

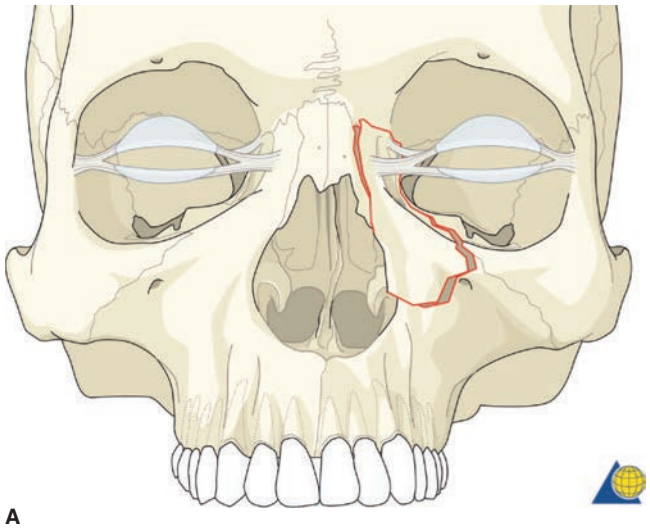
Markowitz et al[67] classified NOE injuries into three subtypes depending on the degree of comminution around the central fragment and integrity of the medical canthal tendon (Fig. 24-17).[67] Type I fractures have a single, intact central segment. Type II fractures have comminution of the central fragment with intact medial canthal tendon attachment. Type III fractures have comminution of the central fragment with detachment or tear in the medial canthal tendon. CT imaging can help distinguish between type I and type II/III fractures. Physical examination for integrity of the medial canthal tendon (eg, bowstring test) can help distinguish between a type II and III fracture.

Nondisplaced or minimally displaced type I fractures may not require surgical intervention if the fragment is stable and not resulting in any deformity. Displaced or unstable type I fractures typically require miniplate fixation that can be achieved via a sublabial, gingivobuccal sulcus approach combined with transconjunctival approach or existing laceration.[52]

Type II and III fractures typically require a coronal approach for adequate reduction of comminuted segments, transnasal wiring, or other canthopexy techniques to restore medial canthal position.[52] Another consideration is the degree of comminution of the nasal root and dorsum, as cantilevered bone or cartilage grafts may be required to restore dorsal projection.

MAXILLARY (LE FORT) MANAGEMENT

Rene Le Fort described lines of weakness along the midface where fractures formed from high-energy impact[68,69] (Fig. 24-18). A Le Fort I fracture results from trauma to the lower midface and results in separation of the alveolar ridge and palate unit from the upper midface. Trauma to the upper midface can result in a Le Fort II fracture, which crosses from the nasal dorsum, frontal process of the maxilla, and lacrimal bones into the orbit and ultimately posteriorly through the pterygoid plates. A downward impact on the upper midface can result in craniofacial separation, or a Le Fort III fracture, where the fracture spans the nasofrontal suture, lacrimal and ethmoid bones, and orbital floor and then across the zygomaticofrontal suture, zygomatic arch, and pterygoid plates. A uniform finding in these fractures is involvement of the pterygoid plates in the fracture line.



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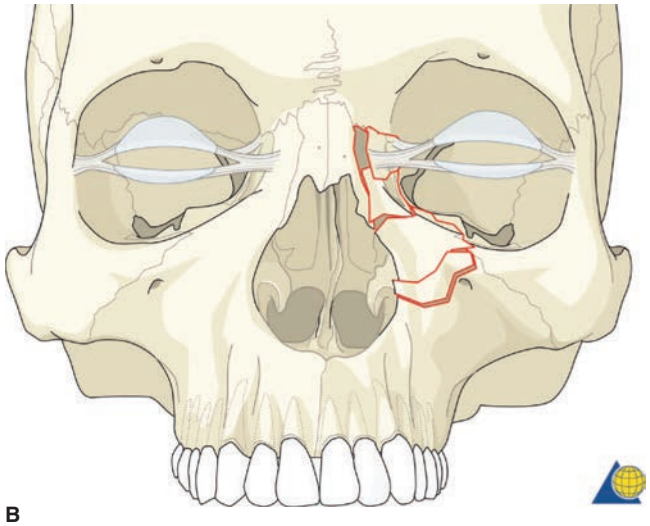

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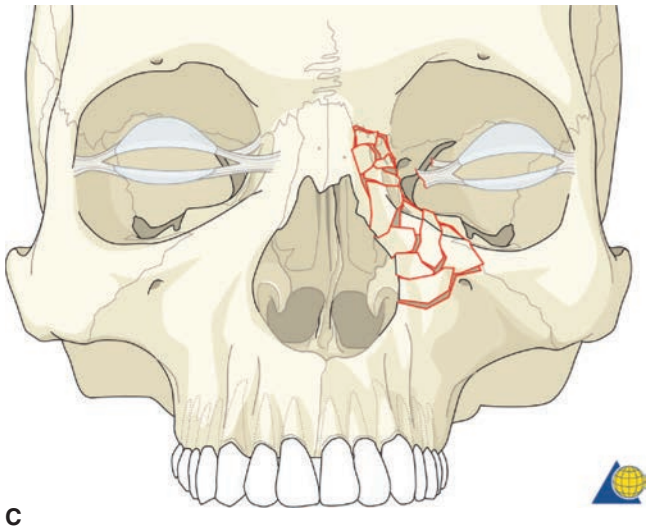

C

FIGURE 24-17 Markowitz-Manson classification of naso-orbito-ethmoid fractures. (**A**) Type I. (**B**) Type II. (**C**) Type III. (Reproduced with permission from AO Surgery Reference. www.aosurgery.org. Copyright by AO Foundation, Switzerland.)

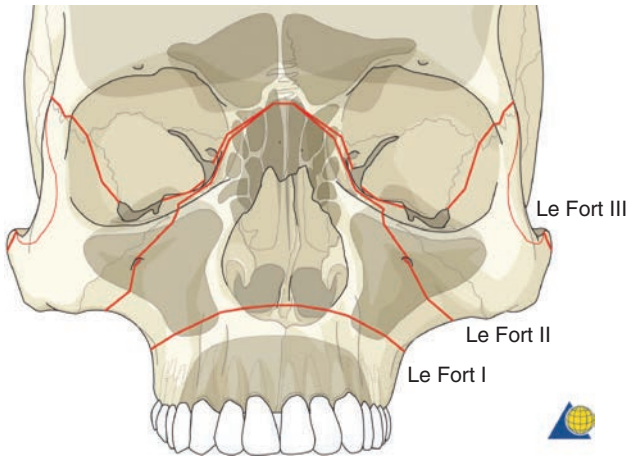

FIGURE 24-18 Le Fort fracture patterns. (Reproduced with permission from AO Surgery Reference. www.aosurgery.org. Copyright by AO Foundation, Switzerland.)

For displaced or unstable Le Fort fractures, open reduction with internal fixation is required. Establishing occlusion with maxillomandibular fixation in patients with dentition is critical to restore function. Reduction of the midface can be performed with Rowe disimpaction forceps (Fig. 24-19). For Le Fort I fractures, miniplate fixation is performed along the medial and lateral buttresses via a gingivobuccal sulcus approach. For Le Fort II and III fractures, additional orbital (eg, transconjunctival, upper blepharoplasty) or even coronal approaches are required for access to the frontal process of maxilla, orbital rim, and frontozygomatic suture.

Mandible

The mandible can be divided into its subsites: symphysis and parasymphysis, body, angle and ramus, condylar

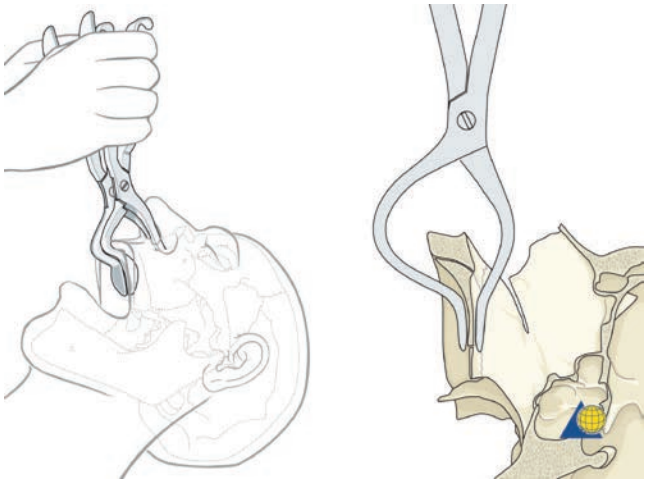

FIGURE 24-19 Use of Rowe disimpaction forceps in reduction of Le Fort fractures. (Reproduced with permission from AO Surgery Reference. www.aosurgery.org. Copyright by AO Foundation, Switzerland.)

process and head, and coronoid process. Fracture management for the mandible is dependent on the patient's dentition, fracture subsite, degree of comminution, and medical comorbidities. There are two major categories of fixation for mandible fractures: load-bearing and load-sharing. Load-bearing osteosynthesis involves fixation such that the plate construct bears 100% of the load placed at the fracture site.[70] This is accomplished with heavier reconstruction plates and bicortical screws. Load-sharing osteosynthesis denotes a construct where the plate and bone share the forces placed at the fracture site, requiring adequate bone stability to buttress to load. Load-bearing fixation is indicated for comminuted fractures, atrophic mandibles, and reconstruction of mandibular defects.[70]

Maxillomandibular fixation is performed if the patient has adequate dentition to restore occlusion. Once rigid fixation is achieved, maxillomandibular fixation can be removed prior to awakening the patient.

Mandibular body fractures can be approached through a transoral gingivobuccal sulcus approach or externally via a transcervical or Risdon incision. Internal fixation with a load-bearing inferior border plate (with or without miniplate tension band just below the tooth roots) is favored over two-miniplate internal fixation due to the higher risk of wound infections and hardware failure in the latter technique.[71]

Parasymphyseal and symphyseal fractures are treated similarly, with the preferred internal fixation approach for unstable and displaced fractures being a load-bearing inferior border plate with or without miniplate or arch bar tension band superiorly.[70] The key to reduction of fractures in this region is to avoid splay of the fracture along the lingual cortex. Lag screw techniques can be used in this region but are technically challenging.[72] These fractures can be exposed via a gingivobuccal sulcus incision, with care to preserve the mental nerve. Existing submental lacerations can be used, but care must be taken if extending these, as the marginal mandibular branch of the facial nerve lies in close proximity.

Angle fractures are the most common isolated mandible fracture. Repair can be challenging and is associated with a high complication rate.[73] Although nondisplaced and stable fractures can be observed with soft diet or short course of antibiotics, most fractures require open reduction, internal fixation due to displacement.[70] For simple fractures, multiple techniques have been described including open reduction, internal fixation with single miniplate along external oblique ridge (Champy technique), single miniplate along the lateral surface of the mandible, two miniplates, or geometric plates. Ellis[73] found that a single miniplate had fewer complications than two-miniplate techniques and that a lateral border miniplate had fewer complications than one placed on the external oblique ridge. Moreover, geometric box miniplates were associated with fewer postoperative complications than conventional miniplates. Comminuted fractures of the angle require load-bearing fixation.

The management of subcondylar fractures is perhaps the most controversial of the subtypes of the mandible. A variety of approaches are used and take into consideration the patient's dentition, age, and comorbid conditions precluding maxillomandibular fixation. In edentulous patients or in minimally displaced fractures with no malocclusion, soft diet and close observation can be performed. Closed treatment with a short course of maxillomandibular fixation with early mobilization was the mainstay of therapy for most fractures, with few absolute and relative indications for open reduction, internal fixation as described by Zide and Kent.[74] Since that time, there has been a paradigm shift toward open reduction, internal fixation, with increasing reports of improved outcomes, including pain, chin deviation, protrusion, and mobility.[75]

Panfacial Trauma

Panfacial fractures involve at least two of the three horizontal thirds of the facial skeleton: cranial vault, midface, and mandible. The principles of management remain the same for each individual fracture. However, panfacial fractures are more complex than a collection of independent fractures. The unique challenge to panfacial trauma is establishing the ideal sequence of repair. Although multiple sequencing strategies have been described, including "bottom-up," "top-down," "outside-in," and "inside-out,"[76,77] the general method of starting from stable areas and proceeding to less stable areas remains key.[78]

SUMMARY

The management of facial trauma requires a comprehensive patient and injury evaluation including careful physical examination and imaging. Careful attention to the extent and location of soft tissue injuries can optimize functional and aesthetic outcomes. Special care should be taken in critical areas such as the facial nerve, orbit, eyelid, parotid duct, nose, and lips. Skeletal deformities should be managed to restore premorbid form and function with care to protect the central nervous system, orbit, and critical adjacent soft tissues. Meticulous attention to scar placement and wound care postoperatively can minimize the aesthetic impacts of surgical repair. Long-term follow-up and postoperative imaging are often required to confirm correct skeletal repair and to watch for skeletal or soft tissue complications that would benefit from revision surgery. These patients are best managed by well-trained experts who are skilled in the nuances of the management of craniofacial soft tissue and skeletal injuries.

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III

MANAGEMENT OF SPECIFIC INJURIES

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25

Neck and Larynx

Jason L. Sperry • Elizabeth Guardiani • Grace Snow • Kirsten Meenan • David V. Feliciano

KEY POINTS

- Penetrating wounds to the neck, particularly those that might involve cervical vascular structures, are grouped into three separate vertical zones with different operative exposures.
- In zone I, a vascular injury from a penetrating wound or blunt trauma may cause a mediastinal or extrapleural hematoma seen on a chest x-ray or computed tomography (CT) scan of the chest, external hemorrhage from the thoracic outlet, or intrapleural exsanguination.
- In zone II, a large lateral hematoma compressing or deviating the trachea mandates endotracheal intubation over a fiberoptic bronchoscope; failure of intubation mandates a cricothyroidotomy.
- In zone III, exsanguinating hemorrhage from the base of the skull after a penetrating wound mandates insertion of a Fogarty balloon catheter for rapid tamponade.
- When all patients with penetrating wounds through the platysma muscle in zone II of the neck are managed with a mandatory operative exploration, nearly 50% will have an unnecessary operation.
- Diagnostic options in patients with vascular "soft" signs after a penetrating wound of the neck are CT arteriography or duplex ultrasonography/color flow Doppler in the modern era.
- A fracture-dislocation of the cervical spine has a 30% or greater incidence of an associated blunt cerebrovascular injury.
- Thirty to 40% of patients with blunt injury to the carotid artery and 10% to 15% of those with blunt injury to the vertebral artery will suffer a stroke without treatment.
- Hyoid bone fractures are rare, and most can be managed conservatively.
- Mild laryngeal trauma can be managed with supportive care, whereas more severe trauma will require surgical intervention.
- Large laryngeal lacerations with exposed cartilage, disruption of the anterior commissure, cartilage fracture, injury to the recurrent laryngeal nerve, and cricotracheal separation are indications for operative repair.
- Laryngeal stents are used in the presence of significant mucosal injuries, injury to the anterior commissure, or when there are multiple cartilaginous fracture lines that cannot be adequately stabilized.

INTRODUCTION

One of the first reported cases of cervical trauma was in Homer's *Iliad* when Achilles delivered a fatal lance blow to Hector's neck, "where the clavicle marks the boundary between the neck and thorax." This was portrayed by Peter Paul Rubens in about 1631 and hangs in the Museum Boymans-van Beuningen in Rotterdam, the Netherlands.[1] Treatment was first described by Ambrose Pare in the mid-16th century when he ligated the right carotid artery and jugular vein of a soldier who had suffered a bayonet wound.[2] The patient survived, but was aphasic and developed a dense, left-sided hemiplegia.[3] The first successful treatment of a major cervical vascular injury did not occur until 1803 when Fleming, aboard the *HMS Tonnant*, ligated the common carotid artery of a sailor after a suicide attempt while at sea. The sailor made a prolonged but complete recovery.[4,5] A similar case was reported by Eves of Cheltenham, England, in 1849.[6]

ANATOMY (SEE ATLAS FIGURES 6 AND 7)

Knowledge of the surface landmarks of the neck is essential for optimal evaluation and management of cervical injuries.[7] The defining borders of the neck encompass the area between

the lower margin of the mandible and the superior nuchal line of the occipital bone and the suprasternal notch and the upper borders of the clavicles.

The first palpable structure from the upper to lower border of the neck is the symphysis menti, which is where the two halves of the body of the mandible unite in the midline. The submental triangle, located between the symphysis menti and the body of the hyoid bone, is bounded anteriorly by the midline of the neck. Laterally, it is bounded by the anterior belly of the digastric muscle, and the mylohyoid muscle forms the floor. The body of the hyoid bone lies opposite the third cervical vertebra. The area between the hyoid bone and the thyroid cartilage is the thyrohyoid membrane, while the notched upper border of the thyroid cartilage is at the level of the fourth cervical vertebra. The cricothyroid ligament or membrane occupies the space between the thyroid cartilage and the cricoid cartilage, which lies at the level of the sixth cervical vertebra and the junction of the pharynx with the esophagus. The interval between the cricoid cartilage and the first tracheal ring is filled by the cricotracheal ligament. Moving inferiorly, the isthmus of the thyroid gland is at the level of the second, third, and fourth tracheal rings. The suprasternal notch can be palpated between the clavicular heads and lies opposite the lower border of the body of the second thoracic vertebra. The sternocleidomastoid muscles, which divide the sides of the neck into anterior and posterior triangles, can be palpated from sternum and clavicle to the mastoid process. The borders of the posterior triangle are the body of the mandible, the sternocleidomastoid muscle anteriorly, and the border of the trapezius muscle posteriorly along with the clavicle inferiorly.

Posteriorly, the structures of the neck that can be palpated in the midline are the external occipital protuberance, the nuchal groove, and the spinous process of the seventh cervical vertebra (cervical spines 1–6 are covered by the ligamentum nuchae).

The platysma, a thin muscular sheet, is enclosed by the superficial fascia. Its origin is from the deep fascia that covers the upper part of the pectoralis major and deltoid muscles, and it inserts into the lower margin of the body of the mandible. It is the anatomic landmark that is often cited when determining whether a penetrating wound of the neck is superficial or deep. The potential for injury to a vital structure exists when this structure is penetrated.

Because it is the crossroads of the brain and corpus, the neck is dense in structure. Beneath the superficial sternocleidomastoid, strap, and trapezius muscles that envelop much of the neck, there are eight body systems that lie within or pass through the neck. Included among these are the following: (1) skeletal system (cervical vertebrae, hyoid bone); (2) nervous system (spinal cord and the facial [VII], glossopharyngeal [IX], vagus [X], spinal accessory [XI], and hypoglossal [XII] cranial nerves); (3) respiratory system (oropharynx, larynx, cervical trachea); (4) gastrointestinal system (oropharynx, cervical esophagus); (5) vascular system (common, internal, and external carotid arteries, vertebral arteries, internal and external jugular veins); (6) lymphatic system (thoracic duct); (7) endocrine system (thyroid and parathyroid glands); and (8) immune system (cervical extensions of the thymus).

ZONES

Penetrating wounds to the neck, particularly those that might involve cervical vascular structures, have been grouped into three separate zones since the original description by Monson et al[8] in 1969 (Fig. 25-1). A minor modification suggested by Roon and Christensen in 1979 is not of clinical significance.[9] Zone I is inferior to the clavicles and manubrium sterni and encompasses all structures in the thoracic outlet. Structures in this zone include the proximal common carotid arteries, vertebral arteries, right and left extrapleural subclavian arteries, jugulo-subclavian venous junctions, crossover left innominate vein, thoracic duct, trachea, esophagus, spinal cord, proximal brachial plexus, and the vagus nerve. Operative exposure for injuries in zone I mandates a median sternotomy with cervical extension, high anterolateral thoracotomy, or a supraclavicular incision with claviculotomy or partial excision of the clavicle, so strong clinical evidence of vascular or visceral injury must be present prior to operation. Zone II is between the thoracic outlet and the angle of the mandible. Structures in this zone include the common carotid arteries and bifurcations, vertebral arteries, internal jugular veins, larynx and cervical trachea, cervical esophagus, spinal cord, and the vagus, spinal accessory, and hypoglossal nerves. Operative exposure for injuries in zone II mandates an ipsilateral oblique incision along the anterior border of the sternocleidomastoid muscle or a high anterior cervical (collar) incision with oblique extensions for possible bilateral injuries. Zone III is between the angle of the mandible and the base of the skull. Structures in this zone include the internal carotid arteries, vertebral arteries, internal jugular veins, pharynx, spinal cord, and the facial, glossopharyngeal, vagus, spinal accessory, and hypoglossal nerves. Operative exposure for injuries in zone III mandates subluxation of the temporomandibular joint with interdental wiring or a vertical ramus mandibulotomy. Again, as exposure in this zone is difficult, strong clinical evidence of hemorrhage not responsive to catheter-based therapy should be present prior to operation.

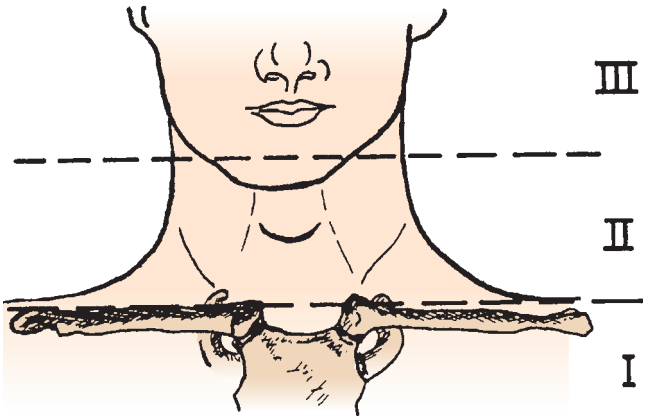

FIGURE 25-1 Zones of the neck. (From Monson DO, Saletta JD, Freeark RJ. Carotid vertebral trauma. *J Trauma.* 1969;9:987-999. Used with permission.)

PRESENTATION

Patients with penetrating or blunt trauma to the neck present with *overt* symptoms and/or signs, present with *modest* or *moderate* symptoms and/or signs, or are *asymptomatic* without signs of aerodigestive or vascular injury. The presentation of a patient with *overt* symptoms or signs will vary depending on the zone of the neck involved. In zone I, a vascular injury from a penetrating wound may cause a mediastinal or extrapleural hematoma on a chest x-ray or computed tomography (CT) scan of the chest, external hemorrhage from the thoracic outlet, or intrapleural exsanguination. A penetrating wound in zone II or blunt disruption of the cricotracheal junction secondary to a "clothesline" injury may lead to loss of the airway and early asphyxiation. Loss of the airway can occur secondary to the presence of a large hematoma from an injury to the carotid artery as well. Active hemorrhage from either the carotid artery or internal jugular vein in zone II can be external and lead to exsanguination or internal into an associated injury to the trachea, leading to aspiration and asphyxiation. Although injures in zone III are uncommon, exsanguination can occur from an injury to the internal carotid artery at the base of the skull.

Patients with *modest* or *moderate* symptoms or signs may present with complaints of hoarseness, dysphagia, or odynophagia and palpable crepitus suggestive of injury to the larynx, trachea, or esophagus. The other presentations in this group are proximity of penetrating wound or blunt contusion to the carotid sheath and/or a stable hematoma suggestive of injury to the carotid artery or internal jugular vein.

An *asymptomatic* patient will have penetration of the platysma muscle by a gunshot or knife wound or bruising or contusion after blunt trauma but have no symptoms or signs of injury to the aerodigestive tracts, cervical vessels, the spine, or the spinal cord.

Management of the patient depends on presentation. Overtly symptomatic patients have "A, B, or C" problems on the primary survey as taught in the American College of Surgeons' Advanced Trauma Life Support (ATLS) course, and immediate resuscitation is performed in the emergency center or operating room. Patients with modest or moderate symptoms or signs undergo a diagnostic evaluation referable to the suspected system injured or one that encompasses the aerodigestive and arterial systems. Asymptomatic patients can be admitted for observation, can undergo radiologic evaluation such as cervical and/or chest CT or CT arteriography (CTA), or can be discharged in some instances.

MANAGEMENT OF PATIENTS WITH OVERT SYMPTOMS OR SIGNS

Zone I and Inferior Zone II

In patients with exsanguinating external hemorrhage from the thoracic outlet or the lower anterior neck, usually from a penetrating wound, blind finger compression of the bleeding vessel through the skin defect is appropriate in the trauma bay (Fig. 25-2). Another trauma bay option is to insert a Foley balloon catheter and see if inflation of the balloon will temporarily tamponade the bleeding. If this is unsuccessful, rapid enlargement of the skin defect with or without local anesthesia is performed. Once again, an attempt is made to compress the bleeding vessel with two or three fingers inserted through the enlarged skin defect. An unsuccessful attempt is followed by tight packing of the area using 3- or 4-in gauze and rapid transport to the operating room. Of interest, a wound compression device has been tested and has been shown to control cervical hemorrhage in a perfused cadaver model.[10] The operative incision will depend on the track of the wound, whether or not manual compression or packing has controlled the bleeding, the patient's hemodynamic status, and the experience of the surgeon. For example, a wound thought to involve the proximal common carotid artery at the base of the neck rather than more proximally in the mediastinum is exposed with the standard anterior oblique incision on the side of injury. An unstable patient with continuing hemorrhage from the outlet or presumed intrapleural exsanguination should undergo a high anterolateral thoracotomy (third or fourth intercostal space above the male nipple) on the side of the injury to allow for direct proximal clamping or pack compression of the injured vessel in the thoracic outlet. If the wound is on the right side of the thoracic outlet and the patient is profoundly hypotensive, the high right anterolateral thoracotomy is performed, the sternum is divided transversely, and a standard (below the nipple) left anterolateral thoracotomy is performed, as well. This will allow for cross-clamping of the descending thoracic aorta to increase perfusion to the coronary and carotid arteries as resuscitation and vascular repair or ligation are accomplished on the right side.

Zone II

In zone II, a penetrating wound may cause impending asphyxiation from a major injury to the trachea, which is suggested by a continuing air leak from the entrance site. The skin defect is rapidly enlarged with or without local anesthesia, and a tracheostomy tube or an endotracheal tube is inserted through the enlarged track following the air bubbles into the distal end of the trachea once it is visualized. The aforementioned "clothesline" injury from blunt trauma may cause cricotracheal separation. Even with impending asphyxiation, there should only be one attempt at standard rapid sequence endotracheal intubation.[11,12] If this is unsuccessful, a rapid standard tracheostomy using a midline (vertical) incision to avoid unnecessary bleeding is performed between the second and third tracheal rings below the area of injury. Should there be bruising and palpable crepitus over the thyroid cartilage suggestive of an injury to the larynx itself, once again there should only be one attempt at standard endotracheal intubation. Failure to complete this is followed by a rapid tracheostomy much as with cricotracheal separation.

Loss of the airway in zone II may occur secondary to tracheal deviation or compression from a hematoma resulting from an injury to the carotid artery or internal jugular vein,

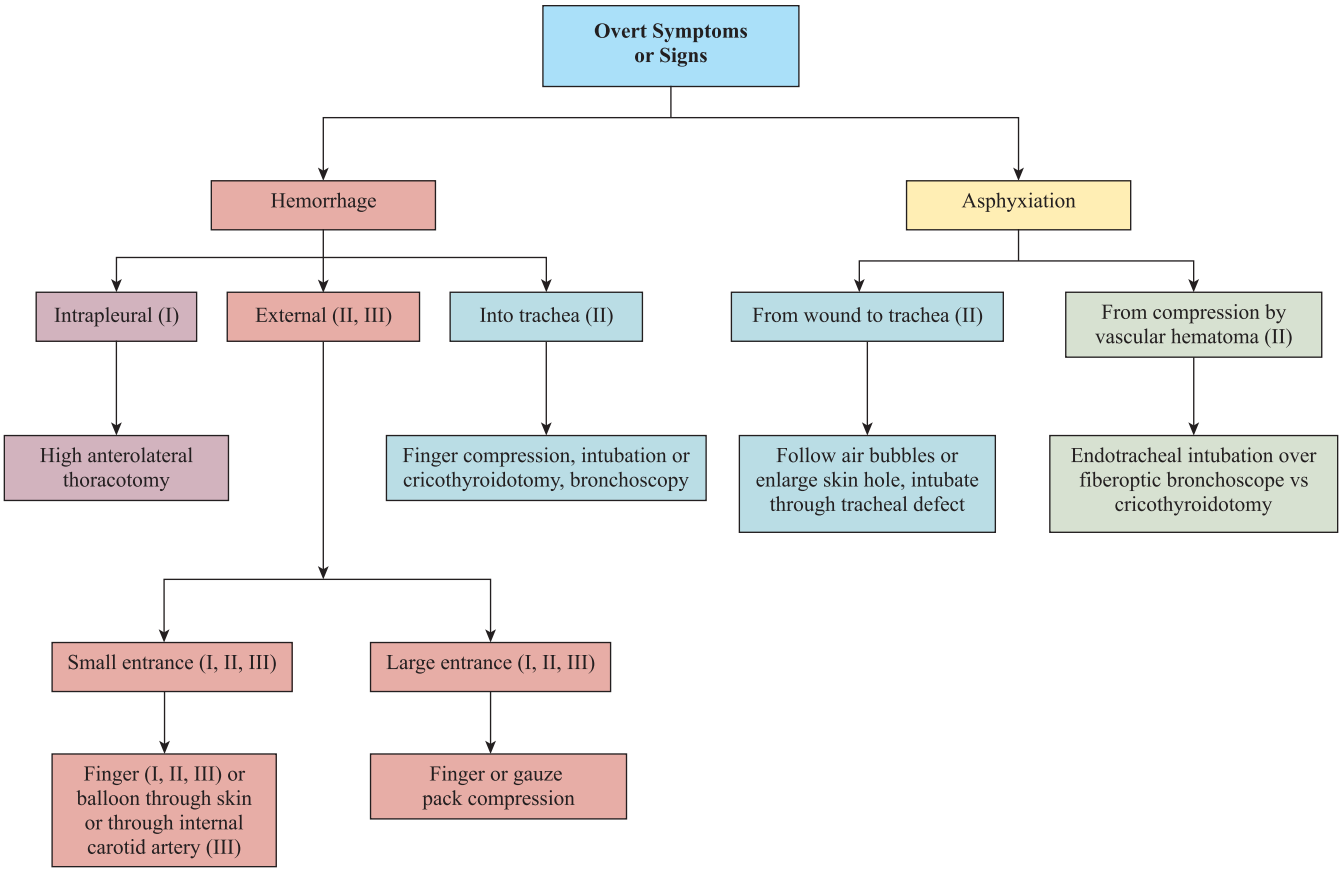

FIGURE 25-2 Algorithm for the management of patients with overt symptoms or signs.

also (Fig. 25-3). With impending asphyxiation, the patient is rapidly moved to the operating room for an attempt at endotracheal intubation over a fiberoptic bronchoscope. If this fails or if the patient is unable to move air when first seen, a cricothyroidotomy is performed rapidly because its high limited central incision (as compared to a standard tracheostomy) avoids the lateral hematoma from the vascular injury.

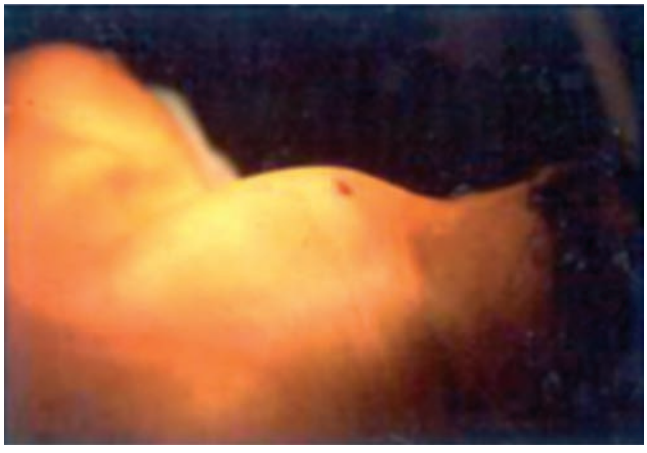

FIGURE 25-3 Significant zone II hematoma secondary to gunshot wound of carotid artery. (From Brown MF, Graham JM, Feliciano DV, et al. Carotid artery injuries. *Am J Surg*. 1982;144:748-753. Used with permission. Copyright © Elsevier.)

With external hemorrhage from a penetrating wound in zone II, direct compression with a finger or fist on the entrance site is performed in the emergency department or trauma bay and en route to the operating room. The decision on where to make the incision can be made in the operating room after endotracheal intubation has been performed.

On rare occasions, there may be internal hemorrhage into the airway when there are adjacent injuries to the carotid artery and trachea. If a single attempt at endotracheal intubation is successful, temporary but relatively tight inflation of the endotracheal balloon may prevent further aspiration of blood while exposure and repair of the artery are accomplished. If a single attempt at intubation is unsuccessful, compression is placed on the carotid artery at the entrance site or at the base of the neck, and a cricothyroidotomy is performed. Because aspiration will have occurred, fiberoptic bronchoscopy is performed once the injury to the carotid artery has been repaired. Tracheal repair should then follow.

Zone III

There are two options when exsanguinating hemorrhage occurs from a penetrating wound to the internal carotid artery at the base of the skull in zone III.[13] Finger compression is

often only partially successful in this location as the internal carotid artery is deep to the mandible. The quickest option is to maintain manual compression as the patient is moved rapidly to the operating room. Once the patient is intubated and the neck is draped, a #3 or #4 Fogarty balloon catheter is inserted into the wound and advanced 2 cm, and the balloon is inflated.[14,15] If hemorrhage continues, the balloon is deflated and advanced 1 cm at a time and inflated until balloon tamponade controls the hemorrhage. The catheter is then sutured to the skin, and endovascular control of hemorrhage can be sought. When external passage of the Fogarty balloon catheter is only partially successful or unsuccessful, a Foley balloon catheter is passed because it has a larger balloon. If this is unsuccessful as well, the catheter is removed and manual compression is applied once again. A standard oblique cervical incision on the anterior border of the sternocleidomastoid muscle is made, the internal carotid artery is exposed, and a small arteriotomy is made in the middle of a 6-0 polypropylene purse-string suture. Once again, a #3 or #4 Fogarty balloon catheter is passed through the arteriotomy and inflated sequentially until balloon tamponade is successful. On rare occasions, it may be necessary to pass balloon catheters through the entrance site and through the internal carotid artery simultaneously (Fig. 25-4).

In addition to operative techniques including balloon tamponade and temporary manual compression, endovascular/angiography techniques when hemodynamic stability allows can be beneficial. These typically can be performed at most Level I and II trauma centers in the operating room, in a hybrid operating room, or in an angiographic suite and can provide information on contralateral carotid flow and allow for ipsilateral proximal and distal control. This type of management assumes that endovascular expertise is readily available, and the patient is temporized, allowing possible transport if needed. Every attempt should be made to keep the patient normotensive with a 100% oxygen saturation level during the case. Intracranial pressure monitoring may be appropriate so that treatment can be initiated when ischemic edema occurs. If the contralateral CT carotid arteriogram documents inadequate crossover flow and a baseline electroencephalogram is abnormal, cerebral ischemia is occurring. This is expected in patients who have had a period of significant hypotension secondary to exsanguination prior to inflation of the balloon. In the past, it was recommended that a patient with ipsilateral cerebral ischemia undergo a saphenous vein bypass from the cervical internal carotid artery to the petrous portion through a small temporal craniotomy.[16] Currently, deflation of temporary occlusive balloon(s) and rapid insertion of an endovascular stent into the high internal carotid artery would be the procedure of choice.[17,18]

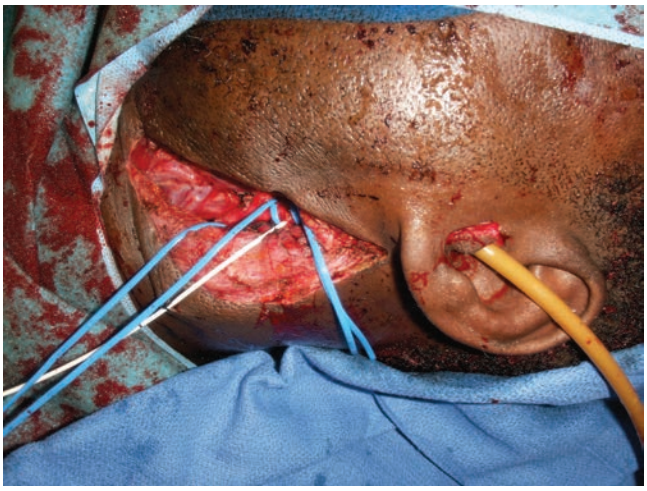

FIGURE 25-4 Patient with stab wound through left eardrum into left internal carotid artery behind left mandible. Hemorrhage was controlled by passing Foley balloon catheter through left ear and #3 Fogarty balloon catheter through the left internal carotid artery. (Reproduced with permission from Ball CG, Wyrzykoski AD, Nicholas JM, et al. A decade's experience with balloon catheter tamponade for the emergency control of hemorrhage. *J Trauma*. 2011;70:330.)

EVALUATION OF PATIENTS WITH MODEST OR MODERATE SYMPTOMS OR SIGNS OR ASYMPTOMATIC PATIENTS (FIG. 25-5)

Zone I

Hemodynamically stable patients with penetrating wounds in proximity to the thoracic outlet should undergo surgeon-performed ultrasound and a chest x-ray. The ultrasound (looking for hemopericardium) will rule out an associated cardiac injury and document the presence of a hemothorax or pneumothorax. The chest x-ray will aid in tracking the course of the missile and in documenting the presence of a hematoma in the superior mediastinum, base of the neck, or supraclavicular area. There have been several retrospective studies documenting that a normal physical examination and chest x-ray virtually exclude a vascular injury at the thoracic outlet.[19,20] Even so, certain trauma centers will use a screening CT as an added study to determine the track of a penetrating wound in this area.[21] When the track of a missile or knife wound is in proximity to vessels at the thoracic outlet and there is an adjacent hematoma on the chest x-ray, a CTA of the carotid, vertebral, and subclavian arteries is performed. This will document the presence and location of a vascular injury and allow for the choice of an appropriate operative incision or endovascular management plan depending on the hemodynamic status and the symptoms demonstrated by the patient. A CTA that is inadequate to make a diagnosis should be followed by a standard arteriogram with the potential for endovascular management/intervention.

There are symptoms (hoarseness, dysphagia, odynophagia), signs (palpable crepitus, continuing air leak through the wound), or findings on imaging (cervical or mediastinal air) that suggest a possible injury to the trachea or esophagus. The diagnostic workup is described in the following "Zone II" section and can then be followed by appropriate operative management if required.

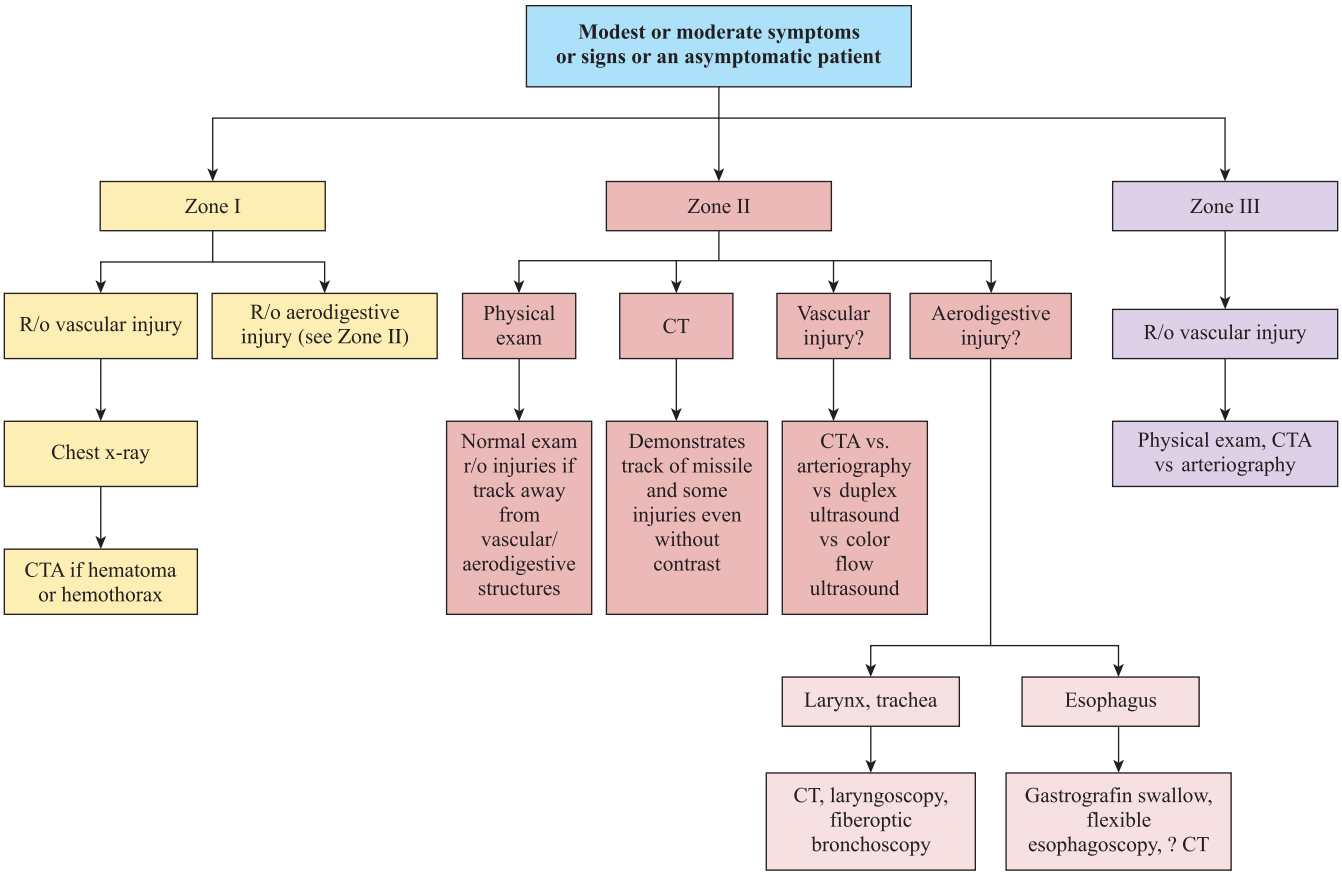

FIGURE 25-5 Algorithm for evaluation of patients with modest or moderate symptoms or signs or an asymptomatic patient. CT, computed tomography; CTA, computed tomography arteriography; r/o, rule out.

Zone II

The approach to possible injuries in this zone has varied considerably over the past 60 years. Based on the report by Fogelman and Stewart[22] at Parkland Memorial Hospital in 1956, mandatory exploration for wounds penetrating the platysma muscle was recommended. This historic recommendation was based on a mortality rate of 6% in patients undergoing early operation versus 35% in those undergoing delayed operation.

It quickly became obvious, however, that cervical explorations in all patients (overtly symptomatic, modestly or moderately symptomatic, or asymptomatic) with penetration of the platysma muscle in zone II resulted in a "negative" exploration rate of approximately 50%.[23] A more selective approach to operation based on symptoms and signs, as described earlier, was then adopted by many centers. One review article in 1991 comparing the two approaches noted that mandatory cervical exploration for platysma penetration had a mortality rate of 5.8% versus 3.7% for a selective approach.[24] Of interest, a negative or nontherapeutic cervical operation occurred in 46.2% of patients treated with mandatory exploration. When patients with modest or moderate symptoms or signs or those who are asymptomatic are managed with a selective approach, only 55% to 65% eventually come to operation. Numerous large studies subsequently verified the safety of a selective approach in the 1980s and 1990s.[25-35]

PHYSICAL EXAMINATION

Physical examination alone is highly accurate in evaluating an asymptomatic patient with a penetrating (through the platysma muscle) stab wound in zone II. This is true for patients with gunshot wounds in zone II as well, as long as the track is tangential or away from the major vessels (lateral) or aerodigestive tract (central).[35-37] With platysma penetration, but without further evaluation by CT, CTA, duplex ultrasonography, or endoscopy, serial examinations of the patient's neck every 6 to 8 hours for 24 to 36 hours are appropriate.

COMPUTED TOMOGRAPHY

Over the past two decades, the evaluation and management of penetrating neck injuries have significantly evolved. Practice has moved from mandatory neck exploration of zone II injuries and the high negative exploration rate toward expectant and selective operative management

with assessment based on increased utilization of CT imaging.[21,22,38-44] When patients have no "hard signs" of vascular injury in zone II, but are "at risk for injury to vital structures within the neck," CT can demonstrate a trajectory away from these structures.[21] With such a trajectory, "invasive studies can often be eliminated from the diagnostic algorithm."[20] Studies using multislice helical CT angiography in the past 15 years have documented a 100% sensitivity and 95.5% specificity in detecting all cervical vascular and aerodigestive injuries.[44,45]

ARTERIOGRAPHY, DUPLEX ULTRASONOGRAPHY, COLOR FLOW DOPPLER, AND CTA

Patients with "hard" signs of a vascular injury in zone II present with external bleeding, bleeding into the trachea or esophagus, an expanding or stable large hematoma, and/or an audible bruit/palpable thrill. Patients with bleeding or an expanding or large stable hematoma undergo an emergency cervical exploration. A patient with a likely arteriovenous fistula should have some type of vascular diagnostic study performed for localization. Should a fistula between the internal carotid artery and jugular vein be present, an endovascular stent rather than cervical exploration may be chosen.

In patients with "soft" signs (modest or moderate signs) of a vascular injury in zone II, such as a history of bleeding at the scene; proximity of a stab, missile, or pellet track; or a small nonexpanding hematoma, the role of arterial diagnostic studies remains controversial. As noted earlier, physical examination alone is highly accurate in ruling out an arterial injury in the asymptomatic patient. Much as in evaluating possible peripheral arterial injuries, however, there is at least a 3% to 5% chance of a surgically reparable arterial lesion in a patient who presents with a cervical vascular "soft sign." In addition, it is likely that a combination of "soft signs" (ie, proximity of wound and small hematoma) will increase the need for surgical intervention. Therefore, some type of diagnostic study is performed in patients with "soft signs" in the majority of centers (Fig. 25-6).

Four-vessel cerebral arteriography was the longtime gold standard of care for evaluating the carotid and vertebral arteries. The technique is highly accurate in diagnosing arterial injuries, eliminating nontherapeutic explorations, and allowing for transcatheter embolization when indicated.[46,47] The disadvantages include the time required to allow the interventional radiology team to return to the hospital at night, the dye load required, the potential for an iatrogenic pseudoaneurysm, and the low yield when all asymptomatic or modestly symptomatic patients are studied.[48]

Duplex ultrasonography, a combination of real-time brightness (B)–mode imaging and pulsed Doppler velocimetry, has been used in the diagnosis of atherosclerotic occlusive decrease of the carotid artery for 40 years.[49,50] Basically, the technology produces images that define anatomy and a spectral profile that documents flow through the vessel. Numerous reports during the 1990s documented the ease and accuracy of the technique when applied to patients with penetrating

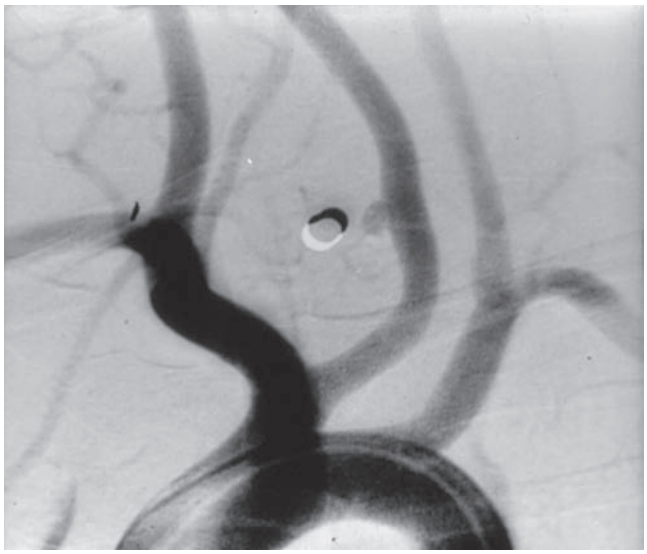

FIGURE 25-6 Pellet wound in an asymptomatic child caused 30% transection of left common carotid artery in zone II. Arteriogram performed secondary to traverse of missile through zone II.

wounds in zone II.[51-54] It was suggested that duplex replace conventional arteriography because of ease of performance and the significant cost savings that would result.[53-55] This did not happen over time in most trauma centers because the technique can be performed only by a registered vascular technologist or experienced vascular surgeon trained in duplex.

A related technique of color flow Doppler has also been used to evaluate the carotid arteries after penetrating trauma to zone II.[56,57] In this technique, flow to and from the point of the Doppler examination is represented on a color scale. Several studies in the 1990s documented that the combination of a careful physical examination and color flow Doppler was a safe alternative to routine contrast angiography.[56,57] It should be noted that Doppler imaging should not be used for the diagnosis of suspected vascular injuries in zone I or zone III or at their borders because imaging will be obscured beneath the clavicle or the angle of the mandible and may lead to a false-negative study.

For the past two decades, there have been ongoing studies to determine the accuracy of CTA, particularly in patients with possible blunt cerebrovascular injuries (BCVIs). Penetrating cervical injuries have been studied as well, with reports demonstrating high sensitivity and specificity.[44,58-62] Based on the ease and speed of obtaining accurate images reconstructed at 1-mm intervals, helical CT can replace conventional angiography in the initial screening setting in most cases.[53] CTA is able to significantly decrease the number of conventional arteriograms required and negative cervical explorations performed.[60-63] The early enthusiasm for using CTA was tempered by early concerns about its accuracy in evaluating possible BCVIs.[64] Subsequent prospective multicenter studies in evaluating patients with penetrating cervical wounds were reassuring.[44,45] Based on currently available data, it appears that multidetector helical CTA has replaced

conventional arteriography as a rapid and noninvasive screening modality to evaluate possible arterial injuries in all zones after *penetrating* trauma. An equivocal screening study or one in which the anatomic area of interest is obscured by scatter or artifact created by adjacent metallic fragments should still, however, be followed by a conventional arteriogram.

ESOPHAGOGRAM, ESOPHAGOSCOPY, AND CT

Patients with modest or moderate symptoms of an esophageal injury present with complaints of deep cervical pain, dysphagia, odynophagia, or hematemesis. On examination, palpable crepitus and deep cervical tenderness may be present. Imaging of the neck will usually demonstrate retropharyngeal or retroesophageal air in the soft tissues, whereas a pneumomediastinum will be present on a chest x-ray if there has been a delay in the patient's arrival to the trauma center.

Historically, the time-honored "sip test" was performed in such patients in centers with limited resources.[32] A patient who was able to swallow a mouthful of water without severe discomfort was felt to have at most only a small injury or no injury of the cervical esophagus and was admitted for observation. The patient who had severe pain with swallowing would then undergo standard diagnostic testing to evaluate for the presence of an esophageal injury.

Although CT has now been widely applied in the diagnostic evaluation of patients with penetrating and blunt cervical trauma as previously noted, its accuracy in detecting an injury of the cervical esophagus has not been studied extensively. This is because several of the reports in which CT has been evaluated do not include any patients with esophageal injuries.[38,44,45] For this reason, asymptomatic patients with air in the soft tissues of the neck after trauma, or a concerning trajectory or proximity, or a combination of modest/moderate symptoms and signs of an esophageal injury should undergo the standard diagnostic evaluation using a contrast esophagogram and endoscopy.[65-67]

Although there is a risk of secondary necrotizing pneumonitis and pulmonary edema if the contrast agent Gastrografin (meglumine sodium) is aspirated, it remains the initial contrast agent of choice for esophagograms in most centers.[66] The accuracy of detecting an injury to the cervical esophagus with this agent is 57% to 80%.[67-70] A "thin" barium study follows a negative Gastrografin swallow or has been used as the primary contrast agent in some centers.[66,67] There should be no concern for mediastinitis after contrast esophagogram because patients with positive esophagograms will go to surgery immediately for washout, drainage, and/or repair.

Because contrast esophagograms with either Gastrografin or thin barium have a less than 100% sensitivity in diagnosis, flexible esophagoscopy is next performed in the at-risk patient with a negative contrast study. It has long been known that the combination of a contrast study and esophagoscopy has an accuracy of nearly 100% in patients with esophageal injuries in zone II.[71,72] In two studies describing the results of flexible esophagoscopy specifically over 16 years, sensitivity was 98.5% to 100%, specificity was 96% to 100%, and accuracy was 97% to 99.3%.[73,74]

LARYNGOSCOPY, FIBEROPTIC BRONCHOSCOPY, AND CT

Patients with modest or moderate symptoms of an injury to the larynx or cervical trachea present with hoarseness, stridor, or hemoptysis.[75] On examination, contusions over the larynx or cervical trachea, palpable crepitus, deep cervical tenderness and bubbling, or an ongoing leak of air from a penetrating wound may be present. As with injuries of the cervical esophagus, paratracheal air or a pneumomediastinum will usually be present on cervical plain films, a chest x-ray, or CT imaging if performed.

In asymptomatic patients with air in the soft tissues of the neck or those with a combination of modest/moderate symptoms and signs of a tracheal injury, the traditional diagnostic evaluation includes laryngoscopy, fiberoptic tracheoscopy, and bronchoscopy. Laryngoscopy will diagnose and localize an injury to the supraglottic, glottic, or subglottic larynx[7,76] (see section on injuries to the larynx).

Fiberoptic tracheoscopy and bronchoscopy continue to be used to evaluate stable patients with suspected injuries to the trachea and major bronchi as well.[77-79]

Zone III

Historically, conventional arteriography was recommended for all stable patients with penetrating wounds in zone III.[80] Much as with mandatory diagnostic studies and/or operation in all patients with penetrating wounds of the neck, the approach to wounds in zone III is now more selective.[72,81] As with the other zones of the neck, patients with hard signs of an arterial injury in zone III have a greater than 90% chance of having a positive cervical exploration. Patients without hard signs, however, rarely have an arterial injury on CTA or conventional arteriography that will require surgical intervention.[81-84] Of course, CTA or arteriography will be of value in a select number of stable patients with hard signs not including bleeding or a combination of soft signs such as a history of bleeding, proximity of wound, and/or a nonexpanding hematoma in this location. When a limited arterial injury is diagnosed on one of these studies, observation or endovascular therapy would be appropriate.

EVALUATION AND TREATMENT OF PATIENTS WITH POSSIBLE BLUNT CEREBROVASCULAR INJURIES

Although BCVIs were first described in 1872, knowledge about pathophysiology, screening, diagnosis, and treatment has mainly accumulated over the past 35 years.[85-88]

The unique relationship of the carotid and vertebral arteries and the skull and cervical vertebrae is a causative factor for these injuries. The common carotid artery bifurcates into the internal and external carotid arteries at the level of the fourth cervical vertebra. The internal carotid artery passes upward and is then fixated in the carotid canal of the petrous portion of the temporal bone until it reaches the foramen lacerum.

Most blunt injuries occur in the internal carotid artery below this fixed area in the skull. In a similar fashion, the second portion of the vertebral artery passes through the transverse foramina (foramina transversaria) of the cervical vertebrae C2–C6 before curving behind the lateral mass of the atlas.

Blunt injury to the neck that results in an intimal tear in the carotid or vertebral artery may lead to stenosis, occlusion, pseudoaneurysm, or embolization. One would suspect that the circle of Willis would compensate for most lesions. However, because 80% of individuals have an anatomic variation (hypoplastic segments or missing components) leading functionally to an incomplete circle, there is less capacity for collateral flow around a lesion, leading to an increased incidence of stroke.[89]

The mechanism of blunt injury to the internal carotid artery can be a direct cervical blow, a basilar skull fracture involving the carotid canal, or a fracture of the petrous portion of the temporal bone. Most authors, however, feel that the most common mechanism is a cervical hyperflexion/hyperextension injury with stretching of the vessel over the bodies of cervical vertebrae C1–C3.[90] The hyperflexion/hyperextension injury is a presumed sequela of the use of shoulder harness restraints in the modern car. Prior to the use of these, many front-seat victims of head-on motor vehicle crashes sustained hyperextension injuries only on impact with the windshield. The hyperflexion/hyperextension injury to the internal carotid artery may be compounded by lateral rotation of the neck away from the side of the shoulder restraint.[90,91]

The mechanism of blunt injury to the vertebral artery may be a fracture of one of the foramina transversaria or a subluxation-type injury in the cervical spine with a locked facet, destroyed facet, or dislocation with instability. Depending on the cause, direct trauma to the vertebral artery (ie, a fracture) or distraction of the foramina with secondary stretching of the artery (ie, dislocated facet) will result.

Incidence and Screening

Most recent series place the incidence of BCVI at approximately 1% to 2% of all admissions for blunt trauma, which is a significant rise from earlier reports with incidences of 0.58% and 1.03%.[89,92] As such, there has been long-term interest in developing and analyzing screening criteria to increase the yield of diagnostic studies.[64,93-100] The group at Denver Health Medical Center/University of Colorado has described liberal screening criteria that include symptoms and signs of BCVI and associated injuries from a high-energy mechanism[93] (Table 25-1). The revised screening criteria added to their screening triggers include mandible fractures, complex skull fractures or cranial injuries, and upper thoracic injuries with traumatic brain injury. These additions have resulted in a significant increase in identification of BCVI at screening.[101,102]

When evaluating the yield of individual criteria, the Memphis group noted that the incidence of BCVI when only one criterion was present ranged from 6% to 17%.[64] When similar criteria were analyzed at the University of Cincinnati in 2009, the respective incidences of BCVI varied widely. For example, skull base fractures through or near the carotid canal had a BCVI incidence of 16.9%; midface fracture or fracture-dislocation of the cervical spine had an incidence of 38.8% and 30.7%, respectively; and Raeder or Horner syndrome had an incidence of 80%.[98] Of interest, a 1987 report based on an analysis of 1,398,310 patients with blunt trauma in the National Trauma Data Bank of the American College of Surgeons (BCVI incidence, 0.15%) noted that BCVI was "poorly predicted by modeling with other injuries."[100]

TABLE 25-1: Denver Screening Criteria for Blunt Cerebrovascular Injuries

Neurologic deficit
Ischemic stroke
Glasgow Coma Scale score <6
Basilar skull fracture
LeFort II/III
Cervical hematoma
Cervical bruit
Cervical fracture
Hanging/anoxia

Source: Adapted from Burlew CC, Biffl WL, Moore EE, et al. Blunt cerebrovascular injuries: redefining screening criteria in the era of noninvasive diagnosis. *J Trauma Acute Care Surg*. 2012;72:330-335. Used with permission.

Imaging for Screening

With the availability of CTA over the past 20 years, there has been increasing enthusiasm for using this modality rather than digital subtraction cerebral arteriography to screen for BCVI. CTA screening has become significantly more common around the globe due to the extent of the screening literature and attention paid to this injury complex.[103,104]

There have been numerous studies suggesting that 16-slice and higher multidetector CTA is a very accurate modality when screening for BCVI. In one study by Berne et al[105] in which four-vessel cerebral arteriography was performed if a 16-slice CTA was positive or equivocal for BCVI, no patient with a negative CTA subsequently developed neurologic symptoms. Eastman et al[106] described 146 patients who had both a 16-slice CTA and cerebral arteriography and noted a 97.7% sensitivity, 100% specificity, and 99.3% accuracy for CTA. In the study by Biffl et al[107] in which 331 patients had a 16-slice CTA and abnormal studies were followed by a conventional arteriogram, no patient with a negative CTA developed a BCVI.

In the not too distant past, much as with the lack of consensus regarding screening criteria, there were numerous studies that questioned the accuracy of CTA in detecting BCVI.[108,109] In 2014, data from the University of Tennessee, Memphis, documented a 68% sensitivity using 64-channel CTA versus 51% sensitivity with older 32-slice scanners, and 62% of

the false-negative studies occurred in low-grade injuries that would likely remain clinically silent.[110] There remains significant variation in the reported sensitivity of CTA despite 64-channel technology, with some series missing up to 20% of carotid injuries.[111] In an attempt to increase the accuracy of diagnosis of BCVI, the Memphis group added CTA to the workup of any patient with potential head, neck, or facial injuries. By adding these injuries to their screening criteria, they were able to find a significant number of carotid and vertebral injuries that were missed by screening criteria alone.[112] When used to help screen patients with a history of blunt head, face, or cervical trauma, 64-slice or higher multidetector CTA has the sensitivity to detect clinically relevant BCVI and has supplanted conventional angiography as an accurate and noninvasive test in the majority of circumstances.

Only a limited number of patients with possible BCVI have been appropriately characterized and studied with magnetic resonance angiography.[113] The most recent literature would suggest that magnetic resonance angiography can "potentially accurately identify and evaluate BCVI."[114] Improvements in technology and vessel wall imaging, however, hold promise to supplement current MRI practices and improve diagnostic specificity for these injuries.[115]

Types of Injuries

Most centers use the grading scale for blunt carotid arterial injury developed at Denver Health Medical Center by Biffl et al[116] in 1999. This grading scale is described as follows: grade I, luminal irregularity or dissection with less than 25% narrowing; grade II, dissection or intraluminal hematoma with 25% or greater luminal narrowing, intraluminal thrombus, or raised intimal flap; grade III, pseudoaneurysm; grade IV, occlusion; and grade V, transection with free extravasation.

Management

As a group, 30% to 40% of patients with blunt injury to the carotid artery and 10% to 15% of those with blunt injury to the vertebral artery will suffer a stroke without treatment.[117-119] The anatomy of the circle of Willis may be an important factor in determining which patients do have a stroke.[120] In the current era of screening, evidence demonstrates that 37% of strokes attributable to BCVI will be apparent upon admission, with the majority of the remainder being diagnosed in the initial 72 hours and only a small percentage (11%) beyond 72 hours.[121] It is evident that, if detected, there is a therapeutic window for treatment with anticoagulation or antiplatelet agents in the majority of patients. Studies have shown that they are both safe and equivalent in asymptomatic patients with carotid or vertebral lesions.[122] The goals of anticoagulation and antiplatelet therapy are to prevent thrombosis, stop embolization, and prevent the propagation of a thrombus that has formed prior to the initiation of treatment.

Early heparinization with a continuous dosage of 10 U/kg/h to a target partial thromboplastin time of 40 to 50 seconds is appropriate for patients with BCVI grade III or IV injuries.[123,124] Antiplatelet therapy may be substituted when heparin is contraindicated.[122] Depending on the rate of hemorrhage, a grade V injury can be treated with insertion of an endovascular stent with insertion of extraluminal coils as needed or, on the rarest of occasions, surgery after subluxation of the temporomandibular joint with interdental wiring or vertical ramus osteotomy as previously noted.

Observation only without the administration of anticoagulants is appropriate temporarily in patients with a grade I to IV injury and an associated traumatic brain or solid organ injury, although the risk of an early stroke is significant with the grade IV injuries.[125] The timing of initiation of anticoagulant therapy in the described patient groups remains controversial at this time, but an early therapeutic window exists to reduce the risk of stroke.[126,127] Treatment should be based on the presence or absence of neurologic findings, the magnitude of the BCVI, and the magnitude of the associated injuries and associated risks of anticoagulation.

The early enthusiasm for endovascular stenting for primarily grade III injuries in the internal carotid artery[128-131] was tempered somewhat by the follow-up report from the Denver group in 2005 in which carotid stents had an occlusion rate of 45%.[132] Subsequent reports, however, have demonstrated a significant decrease in the occlusion rate.[133-137] This difference may be due to strict adherence to the use of antiplatelet agents during the time in which it takes endothelialization of the stent to occur. When endovascular and operative therapies were compared in 842 patients with blunt injuries of the carotid arteries in the National Trauma Data Bank, there was no functional or survival advantage for either group.[138] The development of a postinjury stroke and significant injury severity originally result in the worse outcomes after BCVI.[139]

OPERATIVE MANAGEMENT

General Principles, Incisions, and Emergency Control of Hemorrhage

Once an airway has been established using the emergency techniques previously described or with standard endotracheal intubation, a rolled sheet is placed transversely under the shoulders to hyperextend the neck. The patient is then placed in a sitting position to bring the operative field closer to the surgical team.

With a unilateral track of a penetrating wound or with any aerodigestive injury below the larynx in zone II, an ipsilateral oblique incision is made along the anterior border of the sternocleidomastoid muscle. The length depends on the location of the known or presumed injury, the likelihood of a significant vascular injury, and the experience of the surgeon. For example, a patient with a large unilateral hematoma in zone II overlying a presumed injury to the carotid artery or internal jugular vein should have an incision extending from the sternum inferiorly to the mastoid process superiorly. Such an extensive incision will allow for proximal and distal vascular control around the injury before entering the hematoma. In contrast, a patient without symptoms and signs of

a vascular injury despite a deep stab wound in zone II may be explored through a more limited oblique incision by an experienced surgeon.

When the track of a missile is through zone II bilaterally, a high anterior collar incision at the level of the track of the missile is appropriate. Depending on the patient's hemodynamic status, superior and inferior subplatysma flaps are raised, the midline raphe of the sternohyoid muscles is opened longitudinally, and the sternohyoid muscles are separated from the sternocleidomastoid muscles laterally. An injury on either side of the neck that is higher than expected can be exposed through an oblique extension of one side of the collar incision.

The appropriate incision for probable or documented injury in zone I will depend on the likely injury, the patient's hemodynamic status, and the experience of the surgeon (see Atlas Figures 15 and 16). A profoundly hypotensive patient with active intrapleural hemorrhage from an injury to a great vessel in the superior mediastinum or a subclavian vessel behind the clavicle should have an ipsilateral high (above the nipple) anterolateral thoracotomy. If the injury is on the right side, a trans-sternal extension and left anterolateral thoracotomy (below the nipple) is added to allow for cross-clamping of the descending thoracic aorta as part of resuscitation as previously noted. Vascular control is then obtained with pack compression or direct clamping. When the patient is modestly hypotensive (systolic blood pressure 90–120 mm Hg) and there is a hematoma in the superior mediastinum, a median sternotomy with a cervical or supraclavicular extension is appropriate. In the stable patient with a localized vascular injury on a CTA or conventional arteriogram, a median sternotomy or supraclavicular incision is chosen depending on which vessel is injured.

In the rare patient with hemorrhage from a penetrating wound to the internal carotid artery at the base of the skull in zone III, a balloon catheter is passed through the entrance site on the skin, as previously noted. Inadequate vascular control with the balloon inflated should prompt an ipsilateral oblique cervical incision to allow for exposure of the internal carotid artery and transarterial passage of a Fogarty balloon catheter for internal tamponade. This would then be followed by endovascular stenting, grafting, or angiographic embolization as appropriate.[140]

Injury to the Carotid Artery (See Atlas Figure 8)

Patients with "hard" signs of an arterial injury such as external hemorrhage from zone II, internal hemorrhage into the trachea or esophagus, or the presence of a pulsating/expanding hematoma in the anterior triangle of the neck should undergo immediate cervical exploration. In patients with loss of the carotid pulse but no neurologic deficit, many centers choose to perform a CTA to verify thrombosis of the internal carotid artery. Management of a documented thrombosis from a penetrating wound in the asymptomatic patient (observation vs revascularization) is controversial. The presence of a suspected carotid artery–internal jugular vein fistula, particularly in high zone II or in zone III, should prompt a CTA as well. Depending on local expertise, a documented arteriovenous fistula may be treated with an endovascular stent graft or an open repair.

As previously noted, patients with "soft" signs of an arterial injury still undergo a diagnostic workup in many centers. Included would be patients with a history of bleeding at the scene or in transit, a gunshot wound passing through zone II, a stable hematoma, or a neurologic deficit such as hoarseness from an injury to the vagus nerve proximal to the origin of the recurrent laryngeal nerve. Management will vary depending on the magnitude of any injury to the carotid artery documented on a CTA, conventional arteriogram, duplex ultrasonography, or color flow Doppler examination. In general, extravasation, the presence of an early, pulsatile pseudoaneurysm, significant disruption of the intima, or significant disruption of flow to the brain mandates ipsilateral cervical exploration and repair of the common or internal carotid artery.

PATIENT WITH AN ASSOCIATED NEUROLOGIC DEFICIT

A neurologic deficit in a patient with a penetrating wound to the common or internal carotid artery may be due to cerebral ischemia from the injury itself, hypotension from hemorrhage, acute alcoholic intoxication, or the use of illicit drugs. In patients with any neurologic deficit short of coma (Glasgow Coma Scale score <8), immediate repair of the carotid artery is indicated as the etiology of the deficit is unknown.[141-146] The often-quoted review article by Liekweg and Greenfield[147] in 1978 documented that a "favorable outcome" occurred in only 27% of patients undergoing carotid revascularization versus 25% undergoing ligation when "coma" was the presentation.

In the patient with a carotid thrombosis documented on CTA with a neurologic deficit and a significant (several hours) delay in diagnosis, repair should not be attempted due to the significant possibility of converting an ischemic to a hemorrhagic stroke.

EXPOSURE OF INJURIES IN ZONE III

When an endovascular repair is not appropriate for an injury to the internal carotid artery in zone III (ie, active hemorrhage, pseudoaneurysm has failed stenting with trans-stent coil, the internal carotid artery is very small), an operative approach is indicated. Exposure of the distal internal carotid artery at the base of the skull is obtained by a "stepladder" mandibulotomy,[148] subluxation of the temporomandibular joint with interdental wiring[149-151] or with monocortical screws and steel wiring,[152] or a vertical ramus osteotomy.[153]

REPAIR

In the absence of other significant injuries, systematic heparinization (100 U/kg) is used when any repair more complex

TABLE 25-2: Principles of Repair of the Carotid Artery

- Systemic heparinization (100 U/kg) if complex repair (resection with end-to-end anastomosis or interposition graft) or repair at base of skull will be necessary
- No intraluminal shunt unless inadequate back-bleeding or prolonged repair at base of skull will be necessary
- Interrupted 6-0 polypropylene suture repair in children or in internal carotid artery in all patients
- Flushing sequence after verifying back-bleeding is externally, and then into external carotid artery, and, finally, flow is reestablished into internal carotid artery

than lateral arteriorrhaphy is needed (Table 25-2). Repairs of the carotid artery are accomplished using standard techniques including the following: (1) minimal debridement and lateral arteriorrhaphy with interrupted 6-0 polypropylene sutures for a lateral defect; (2) patch angioplasty with saphenous vein, thin-walled polytetrafluoroethylene, or bovine pericardium for loss of one wall; (3) segmental resection and end-to-end anastomosis for through-and-through injuries or segmental disruption; and (4) segmental resection and insertion of a saphenous vein or polytetrafluoroethylene interposition graft[13,142-144] (see Atlas Figure 9). On rare occasions, an injury to the proximal internal carotid artery may be repaired by ligating and dividing the distal external carotid artery and using the proximal segment as a transposition graft (see Atlas Figure 10).

In a young patient with excellent back-bleeding from the internal carotid or common carotid artery after vascular control has been attained, an intraluminal shunt is *not* indicated as the repair is completed. With a rare distal injury in the internal carotid artery in upper zone II or in zone III, insertion of a graft may take longer than 30 minutes. In this situation, a temporary intraluminal shunt should be considered as the repair is completed.

Because cross-clamping of the common or internal carotid artery after a period of hypotension and during the period of repair may result in ipsilateral cerebral ischemia, postoperative care is critical.[154] The possibility of an ischemia–reperfusion injury with secondary ipsilateral cerebral edema mandates avoiding hypotension and hypoxemia as well as performing serial careful postoperative neurologic examinations.

Should the patient have no improvement of preoperative neurologic symptoms or develop neurologic deterioration in the early postoperative period, emergent CT of the brain is performed. Ipsilateral cerebral edema is treated with the insertion of an intracranial pressure monitor as well as standard drainage and medications such as hypertonic saline and/or mannitol.

In one older series, the survival rate for all 129 patients undergoing operation on an injured carotid artery was 75%. When patients failing resuscitation were excluded, the survival rate was 85%.[142]

Injury to the Vertebral Artery (See Atlas Figure 12)

An injury to the vertebral artery such as dissection from blunt trauma or intimal disruption, a pseudoaneurysm, arteriovenous fistula, or active hemorrhage tamponaded by packs or bone wax at operation is often diagnosed or confirmed on a CTA or other imaging study in the hemodynamically stable patient. Appropriate treatment is the placement of an endovascular stent for a pseudoaneurysm or intimal lesion and acute balloon occlusion, if needed, followed by coil embolization of an arteriovenous fistula or active hemorrhage.[155,156]

In patients undergoing a cervical exploration for hemorrhage or a suspected injury to the aerodigestive systems, active hemorrhage originating from the posterolateral neck adjacent to the spinal transverse processes is likely from an injured vertebral artery. Although detailed descriptions of operative approaches to the different levels of the vertebral artery are available, they are used infrequently in the modern era.[157-159] As proximal ligation of the ipsilateral vertebral artery originating from the second portion of the subclavian artery is unlikely to stop the hemorrhage, packing with bone wax or gauze is commonly used and is nearly always successful (see Atlas Figure 13). Many surgeons leave the bone wax in place, whereas the gauze pack will need to be removed at a reoperation. After packing, an ipsilateral vertebral CTA followed by proximal embolization is appropriate.

With occlusion of the vertebral artery by the trauma itself or by operative ligation or coil embolization, antegrade thrombosis is a risk in the postoperative/postprocedure period. For this reason, anticoagulation with heparin and/or antiplatelet therapy is appropriate before discharge. Whether long-term anticoagulation is necessary is still unclear at this time.

When unilateral vertebral artery ligation, packing, or coil occlusion is performed, a mortality rate of 5% to 15% is expected.[157,160] Deaths are invariably due to prehospital exsanguination or an associated traumatic brain injury.

Injury to the Internal Jugular Vein

Selective nonoperative management is now used in several centers around the world.[161,162] When an operation is necessary, lateral venorrhaphy is appropriate for wall defects, whereas more extensive injuries are treated with ligation. Because bilateral ligation of the internal jugular veins may result in pseudotumor cerebri, every effort should be made to repair one internal jugular vein when bilateral injuries are present.

Injury to the Esophagus

The simplest technique to expose the cervical esophagus is to dissect down to the cervical vertebral bodies and then lift the posterior wall of the esophagus off them by stripping with a finger (Table 25-3). Exposure of the anterior esophagus requires some care, as the recurrent laryngeal nerves are

TABLE 25-3: Principles of Repair of the Esophagus

- Either one- or two-layer repair with absorbable sutures is acceptable, preferably in a transverse direction.
- Loss of a portion of the wall in zone II and some patients with a delayed diagnosis of perforation should be treated with a loop esophagostomy over a rod rather than an acute tenuous repair that is likely to dehisce.
- Combined injuries with the trachea or carotid artery mandate a vascularized muscle buttress/separator such as the sternocleidomastoid muscle.

located in the tracheoesophageal groove at the lower cervical level. Once the esophagus has been dissected circumferentially, it can be looped with a finger or Penrose drain and carefully inspected by pulling it toward the operating surgeon. Any area of hematoma staining should be gently explored with a scissor to see if the mucosa underneath has been perforated. If the mucosa is intact, the esophageal muscle is reapproximated with several simple interrupted sutures of 3-0 absorbable material.

On occasion, it may be necessary to have the anesthesiologist help make the diagnosis of a small occult perforation in the cervical esophagus. One technique is to compress the distal esophagus at the thoracic inlet with a finger or noncrushing clamp and to fill the proximal esophagus with 30 to 50 mL of methylene blue dye in saline (one ampule in 200 mL). Full-thickness staining of dye at any location suggests that a perforation is present. Another technique is to place the tip of a nasogastric tube in the midcervical esophagus, compress the distal esophagus, and have the anesthesiologist inject 30 to 50 mL of air into the proximal esophagus through the nasogastric tube. By filling the operative field with saline solution, any air leak from an occult perforation would be seen as bubbling into the saline. On-table endoscopy can similarly provide air and result in bubbling at the site of injury.

With a limited injury from a stab or gunshot wound, minimal debridement is performed. A two-layer repair starts with a continuous 3-0 absorbable suture closure of the mucosa, preferably in a transverse direction. The repair is completed by placing interrupted 3-0 absorbable sutures through the muscularis layer of the esophagus. Because there is a 5% to 25% leak from repairs of the cervical esophagus historically, a small Penrose drain or closed suction drain is placed adjacent to the repair before closure of the incision.[163-166] This drain is brought anteriorly so as not to cause erosion of the carotid artery laterally.

When there has been a loss of tissue from one wall or the diagnosis of a perforated cervical esophagus has been delayed, a simple lateral suture repair or end-to-end anastomosis is not appropriate. A lateral loop esophagostomy at the site of the defect should be placed over a red Robinson catheter (much like a rod under a loop colostomy) located in the incision or lateral to it.[167,168] Whether a tie of absorbable suture material should be placed around the distal side of the elevated loop remains controversial.[168] Keeping the esophagus in continuity, even with a large defect, will preserve esophageal length and avoid the need for a colon interposition or free jejunal graft in the future. Conversion to a loop esophagostomy rather than performing a tenuous repair avoids the complication of a large esophagocutaneous fistula with secondary problems such as tracheoesophageal fistula, carotid artery blowout, or wound infection in the postoperative period. The esophagostomy has a tendency to shrink and to pull to the posterior midline over time, and delayed closure is often much easier than expected.

Although primary repair when feasible is preferred, newer options also include esophageal stenting. This can be performed safely as an adjunct to appropriate drainage when minimal contamination at the time of diagnosis exists or when primary repair would be less than adequate. The temporary stent reduces contamination and spillage from a documented esophageal injury and avoids complications from a "forced" or tenuous repair.[169-171]

Injury to the Trachea

Anterior or lateral perforations are not debrided and are closed with interrupted full-thickness 3-0 absorbable sutures to create an airtight seal (Table 25-4).[78,172] When there is tissue loss in the anterior or lateral trachea, a tracheostomy tube can be placed into the defect until a decision is reached on use of a vascularized muscle patch or formal reconstruction. Should the large defect be in the proximal trachea, the sternal head of the sternocleidomastoid muscle is detached, rotated medially, and sewn directly to the defect to create an airtight seal after removal of the tracheostomy tube. Resection of a large defect, mobilization of both ends of the trachea, and an end-to-end anastomosis can be performed electively but have never been performed at a first operation after trauma in the senior author's experience.

When there is a large defect in the membranous portion of the cervical trachea, a three-sided longitudinal anterior pericardial flap based superiorly is created after a median sternotomy is performed. The pericardial flap is then sewn to the defect in the membranous trachea to create an airtight seal.

TABLE 25-4: Principles of Repair of the Trachea

- No debridement is necessary.
- One-layer repair with absorbable suture if small or moderate-sized hole.
- When there is loss of a portion of the anterior or lateral wall, a tracheostomy tube is inserted into the defect. The sternocleidomastoid muscle is then detached inferiorly, mobilized, and sewn in an airtight fashion to the defect after the tracheostomy tube is removed.
- When there is loss of a portion of the membranous trachea, a three-sided rectangular longitudinal flap of pericardium based superiorly is sewn to the defect to create an airtight seal.

Late reconstruction of a previously injured trachea with a segmental partial loss of tissue is best performed by a thoracic surgeon with experience in tracheal resection and reconstruction. Dissection should be limited at the 3 and 9 o'clock areas of the trachea to avoid devascularizing the ends. Both laryngeal lowering and bilateral lung elevating procedures may be necessary with gaps in the trachea exceeding 5 to 6 cm.[173] Repair is accomplished with interrupted 3-0 absorbable sutures, no protective tracheostomy is performed, and a sternocleidomastoid muscle flap may be used to buttress the suture line.

Combined Injuries to the Trachea–Esophagus, Trachea–Carotid Artery, or Esophagus–Carotid Artery

A postoperative complication rate of 74% was reported in one older series of 23 combined tracheoesophageal injuries.[174] Analysis of the complications documented that the majority were due to leaks from the esophageal repair. This led to wound infections, tracheoesophageal fistulas, secondary pneumonias, and blowouts of adjacent repairs of the carotid artery. With adjacent repairs of the trachea and esophagus, trachea and carotid artery, or esophagus and carotid artery, a vascularized sternocleidomastoid muscle flap should be used to cover the visceral repair[175] (see Atlas Figure 11). This should lower the incidence of a leak from the visceral repair and, if a leak occurs, protect the adjacent arterial repair.

The sternocleidomastoid muscle has a tripartite blood supply that includes the thyrocervical trunk, superior thyroid artery, and occipital artery. Therefore, it can be detached from the sternum and clavicle inferiorly or the mastoid process superiorly and rotated to cover the repair of the trachea or esophagus and act as a vascularized buttress. With combined injuries of the trachea, esophagus, and/or carotid artery at the upper or mid-area of zone II, the first step is detaching the sternal head of the sternocleidomastoid muscle from the sternum if the muscle is bulky. If it is not, both the sternal and clavicular attachments are divided. Either the detached sternal end or the entire muscle is then mobilized and rotated medially to buttress the tracheal or esophageal repair and separate it from the repair in the carotid artery. The mobilized muscle is sewn in place with multiple interrupted sutures of 3-0 absorbable material. Any esophageal repair is drained anteriorly with the drain track away from the repair in the carotid artery.

SUMMARY OF TRAUMA TO THE NECK (NOT INCLUDING INJURIES TO THE HYOID BONE AND LARYNX)

Patients with penetrating or blunt injuries to the three zones of the neck present with overt symptoms or signs, present with modest and moderate symptoms or signs, or are asymptomatic without signs of aerodigestive or vascular injury. When overt symptoms and/or signs are present, standard "ABC" resuscitation as described in the ATLS manual is performed and early intervention should occur. With moderate or modest symptoms or signs, a variety of diagnostic tests including cervical CT, conventional arteriography, duplex ultrasonography, color flow Doppler, CTA, esophagography, and fiberoptic esophagoscopy, tracheoscopy, and bronchoscopy are used to determine whether an injury to the carotid artery system, vertebral arteries, esophagus, or trachea is present.

When a BCVI is diagnosed, heparinization or antiplatelet treatment for grade I to IV injuries and endovascular stenting, balloon occlusion, or operation for grade V injuries are indicated. Prior to repeat imaging (CTA) in patients with grade I to III injuries, aspirin and clopidogrel bisulfate are administered in anticipation of the need for an endovascular stent.

Basic principles and techniques of arterial repair are used when a penetrating injury to the carotid artery is present. Temporary intraluminal shunts are only indicated if distal backflow is poor or if a prolonged complex repair is anticipated in high zone II or in zone III. Penetrating unilateral vertebral artery injuries with hemorrhage are managed with packing, temporary balloon occlusion, proximal and distal ligation, or embolization.

Simple esophageal perforations are repaired with one or two layers of absorbable sutures. When there is a loss of esophageal tissue from one wall or a delay in diagnosis of an esophageal injury, a cervical loop esophagostomy is occasionally necessary.

Tracheal repairs for routine perforations are performed with one layer of absorbable sutures. A more significant anterior defect is managed with a tracheostomy at the first operation. When there is loss of the membranous trachea, a three-sided pericardial flap that is rotated superiorly will be necessary.

Diagnostic approaches and operative techniques have been refined significantly over the past 70 years, and this has resulted in a significant decrease in unnecessary operations and better outcomes after indicated operations.

INJURY TO THE HYOID BONE

The hyoid bone is a U-shaped bone in the anterior neck. Given its relative anatomic protection by the mandible anteriorly as well as its mobility in all directions, fractures of the hyoid bone are rare occurrences.[176] These typically occur as a result of blunt trauma to the anterior neck and are particularly associated with asphyxiation-type injuries, although motor vehicle accidents are the most common cause of hyoid fractures currently.[177,178] Hyoid fractures may occur in isolation, but often occur simultaneously with fractures to the midface and mandible, injuries to the cervical spine, and laryngotracheal injuries.[177,178] Because of its position relative to the pharynx, fracture of the hyoid bone can result in significant airway edema, which can be delayed in presentation. There may also be concurrent pharyngeal lacerations, which can result in bleeding and contribute to airway obstruction.[179]

Diagnosis

Patients may complain of anterior neck pain, dysphagia, dysphonia, painful coughing, gagging, and/or dyspnea.[178] In more severe cases, patients may present with hemoptysis and respiratory distress; however, patients may be also be asymptomatic initially, and there may be other more obvious injuries.[179] Physical examination may reveal swelling or ecchymoses of the anterior neck, crepitus, and tenderness of the anterior neck on palpation.[177] A fracture of the hyoid bone may be detected by plain x-ray; however, CT of the neck has largely become the diagnostic modality of choice due to its ability to provide better detail of the fracture as well as injury to any surrounding structures. Flexible laryngoscopy is also critical in the evaluation of patients with a suspected hyoid fracture to evaluate for the presence of airway edema and pharyngeal lacerations. In addition, the performance of a nasal Valsalva maneuver (asking the patient to forcibly exhale with mouth closed) during flexible laryngoscopy may reveal instability of the hyoid bone and detect fractures that are not recognized on CT.[180]

Management

Because of the rare nature of hyoid bone fractures, no definitive management algorithm exists. The majority of these injuries can be managed conservatively with steroids, rest, analgesics, and a soft diet.[177] Because delayed airway edema can occur, inpatient observation for a period of 24 to 72 hours is recommended.[176] In patients in whom there are significant pharyngeal lacerations or severe dysphagia, nasogastric or orogastric feeding should be considered. Tracheotomy is sometimes required in the case of severe airway edema. Surgical exploration may be considered in patients in whom there are extensive pharyngeal lacerations or bone fragments entering the pharynx or impinging on other critical structures. Open reduction and internal fixation (ORIF) of the hyoid bone appears to be of limited utility. In a systematic review of 41 patients with hyoid bone fractures that were conservatively managed, all reported resolution of symptoms.[177]

INJURY TO THE LARYNX

The larynx is protected by the sternum inferiorly, the mandible superiorly, and the sternocleidomastoid muscles laterally. It is also supported by numerous muscles and tendons, which can help disperse the forces of trauma. Its protected location makes external laryngeal trauma rare. In the 1990s, the incidence was reported to be between 1 in 5000 and 1 in 30,000 emergency department visits in the United States.[181-183] A recent study in Canadian centers reported an incidence of 1 in 2478 emergency presentations, suggesting that laryngeal trauma might be more prevalent than previously described.[184] Major blunt laryngeal trauma has declined in recent years due to improvements in motor vehicle safety, but the proportion of penetrating injuries has increased secondary to an increase in violent crimes.[185]

TABLE 25-5: Classification Scheme, Characteristics, and Management of Laryngeal Trauma

Group	Characteristics of injury	Management
Group 1	Minor endolaryngeal hematoma/laceration No fracture	Observe at least 24 hours, repeat flexible laryngoscopy Conservative management
Group 2	Edema, hematoma, minor mucosal disruption No exposed cartilage May have nondisplaced fracture on computed tomography	Direct laryngoscopy and esophagoscopy to rule out more severe injury Serial exams Conservative management May require intubation or tracheotomy
Group 3	Massive edema, large mucosal lacerations Exposed cartilage Displaced fractures Vocal cord immobility	Direct laryngoscopy and esophagoscopy Often require tracheostomy Generally require surgical repair
Group 4	Same as group 3 plus: • Massive mucosal damage • Disruption of anterior commissure • Two or more fracture lines	Direct laryngoscopy and esophagoscopy Tracheostomy Surgical repair with laryngeal stent
Group 5	Complete laryngotracheal separation	Awake tracheostomy or ventilating bronchoscopy to secure airway followed by tracheotomy Laryngotracheal reconstruction

The most common cause of blunt laryngeal trauma is motor vehicle collisions, during which the neck is hyperextended, leaving the larynx exposed to injury from the dashboard or steering wheel. Other causes include violence, strangulation, and sports injuries.[181] Penetrating laryngeal injuries are often secondary to gunshot or knife wounds and projectiles from machinery and motor vehicle collisions.

The most commonly used classification system for laryngeal trauma is the Schaefer-Fuhrman system (Table 25-5).[182,186] This system was first described for blunt laryngeal injuries but can be extrapolated for penetrating injuries.

Diagnosis

The mechanism, trajectory, and force of the injury can help predict the subsite of injury in the larynx, extent of injury, and involvement of the recurrent laryngeal nerve. A thorough physical examination the is key to diagnosing laryngeal trauma. The signs and symptoms of laryngeal injury after blunt trauma to the neck may be minimal and overlooked.

The skin and muscle may be unharmed, and symptoms may progress over hours as edema, hematoma, or instability of the larynx evolves.[186] The most common symptoms are dysphonia and respiratory distress as well as neck pain, dysphagia, odynophagia, and hemoptysis.[181,185-187] Signs of laryngeal trauma include subcutaneous emphysema, ecchymoses or edema of the anterior neck, stridor, tenderness, and inability to tolerate supine positioning.[185,188,189] Patients may also present with cardiopulmonary arrest, shock/hemorrhage, and neurologic deficits, particularly after penetrating trauma.[189]

The most critical part of the initial examination is recognizing and treating an impending airway obstruction. If the patient has a stable airway, nasolaryngoscopy should be performed to assess patency of the airway, mobility of the vocal folds, and mucosal injuries. A topical anesthetic should be avoided to prevent aspiration.[186] Some authors advocate for obtaining a CT scan in all patients with laryngeal trauma after they are stabilized.[183,190,191] Others recommend not performing a CT scan if the injuries are very mild or if they are severe enough that the patient will undergo neck exploration regardless of imaging.[192-194] In intermediate cases, CT can help determine if there are displaced fractures that would benefit from repair (Fig. 25-7). In patients with type 2 to 5 laryngeal injuries, direct laryngoscopy should be performed to assess the degree of injury provided there is no injury to the cervical spine that would preclude safely extending the neck. Bronchoscopy and esophagoscopy can be performed at the same setting to evaluate for any tracheal or esophageal injury. If there is concern for a concomitant esophageal injury that cannot be assessed by esophagoscopy, an esophagogram should be obtained.[181]

Management

Maintaining a safe airway is the primary goal in the management of laryngeal trauma. Tracheostomy is the preferred method for securing the airway. Unless laryngotracheal separation has been ruled out, orotracheal intubation and cricothyrotomy should be avoided. Awake fiberoptic intubation may be a safe option in select patients when a physician experienced in this technique is available. Ideally, the airway should be evaluated by fiberoptic laryngoscopy prior to intubation to ensure that fiberoptic intubation is feasible.[183,185] A smaller endotracheal tube with a high-volume, low-pressure cuff should be used.[181] If a cricothyrotomy is performed, it should then be converted to a tracheostomy in the operating room.[185] Secondary goals of management include preservation of voice and avoidance of a long-term tracheostomy. Depending on the severity of injury, laryngeal trauma can be managed medically or surgically.[195]

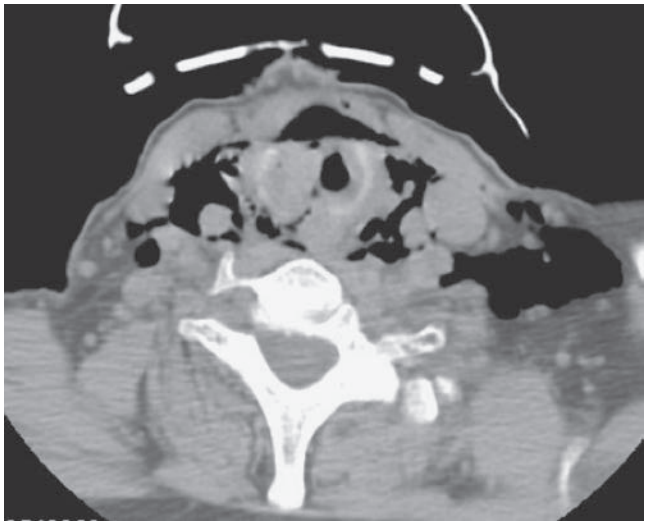

FIGURE 25-7 Computed tomography of the neck revealing a severely displaced fracture of the cricoid cartilage and extensive subcutaneous air.

MEDICAL TREATMENT

Patients in group 1 and many patients in group 2 (Table 25-5) who have injuries consisting of edema, small hematomas, minor mucosal lacerations without exposed cartilage, or a single nondisplaced fracture of the thyroid cartilage are likely to recover without surgical intervention. Patients should be observed with continuous pulse oximetry for at least 24 hours and should undergo repeat flexible laryngoscopy in order to assess for worsening of edema.[185] Supportive care measures should be employed (Table 25-6).

SURGICAL TREATMENT

Patients in groups 3, 4, and 5 typically require surgical intervention. Specific indications for surgery include large mucosal lacerations, lacerations involving the vibratory edge of the vocal fold, exposed cartilage, multiple or displaced cartilage fractures, avulsed or dislocated arytenoids, and transection of the recurrent laryngeal nerve. In general, any patient undergoing surgical management should have a tracheostomy placed at the same setting if not already performed. Fig. 25-8 illustrates the standard management algorithm for laryngeal trauma.

ARYTENOID DISLOCATION

The arytenoids are two small paired laryngeal cartilages that articulate with the cricoid cartilage and are attached to the vocal cords. Arytenoid dislocation is a rare finding associated with blunt laryngeal trauma and can be differentiated from vocal fold paralysis with a CT scan or with laryngeal

TABLE 25-6: Conservative Management of Laryngeal Trauma

Systemic corticosteroids
Cool humidified air
Antireflux treatment
Head of bed elevation
Voice rest
Consider antibiotics

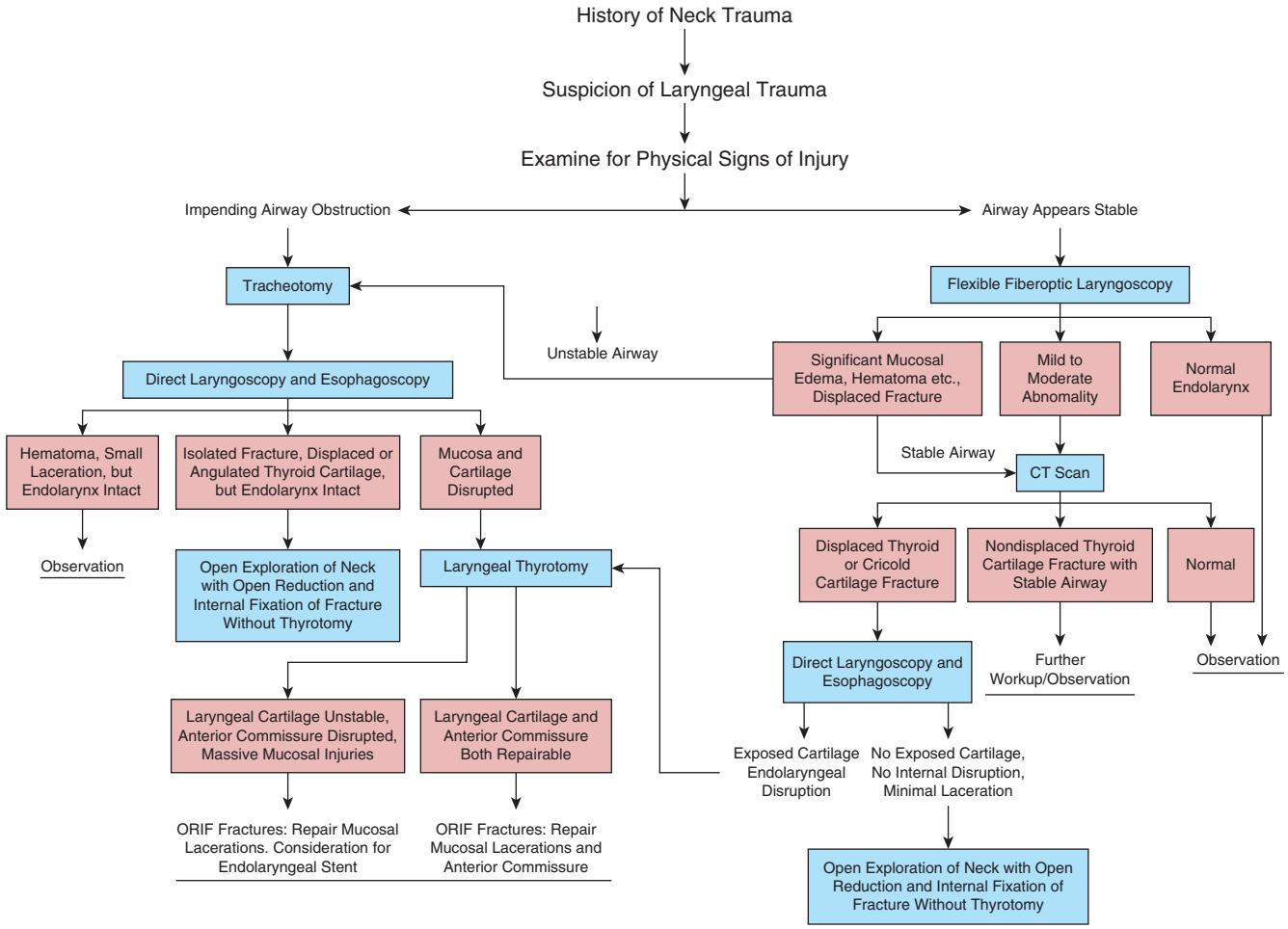

FIGURE 25-8 Algorithm for management of laryngeal trauma. CT, computed tomography; ORIF, open reduction and internal fixation. (Reproduced with permission from Jordan R, Norris BK, Stringer SP. Laryngeal trauma. In: Johnson J, Rosen CA, eds. *Bailey's Head and Neck Surgery: Otolaryngology*. Philadelphia, PA: Lippincott Williams & Wilkins; 2014:1144.)

electromyography (EMG). A CT scan will show obliteration of the cricoarytenoid joint, whereas a laryngeal EMG typically shows normal recruitment in arytenoid dislocation but signs of denervation when paralysis of a vocal cord is present.[196]

Arytenoid dislocation is treated by performing endoscopic reduction to restore the proper height of the vocal process. To reduce an anterior dislocation, an anterior commissure laryngoscope is placed against the anterior surface of the arytenoid and is rotated in a posterior-lateral direction while applying external counterpressure to the larynx. To reduce a posterior dislocation, a Miller laryngoscope blade is placed against the posterior surface of the arytenoid and cephalic pressure is applied, followed by anteromedial rotation.[197]

MUCOSAL TEARS OR EXPOSED CARTILAGE

Endolaryngeal mucosal tears or exposed cartilage can be addressed using a median thyrotomy approach. A horizontal incision is made in a skin crease at the level of the cricothyroid membrane. Subplatysmal flaps are raised superiorly to the level of the hyoid and inferiorly to the cricoid. The strap muscles are separated in the midline and retracted laterally. The cricothyroid membrane is incised horizontally. The larynx is then entered by making a vertical incision through the thyroid cartilage extending superiorly through the anterior commissure under direct visualization of the true vocal folds. If a median or paramedian vertical thyroid cartilage fracture is present within 3 mm of the anterior commissure, it can be used to gain access to the larynx. The endolarynx is examined thoroughly, and mucosal lacerations are repaired with 5-0 or 6-0 absorbable suture. Exposed cartilage should be covered primarily or with local mucosal advancement flaps. If the anterior commissure is disrupted, the anterior margin of the true vocal cord should be sutured to the external perichondrium of the thyroid cartilage to resuspend it.[198] After repair of endolaryngeal injuries, ORIF is performed of the median thyrotomy incision through the thyroid cartilage.

FRACTURE OF THE THYROID OR CRICOID CARTILAGE

Except for a single nondisplaced fracture, all thyroid or cricoid cartilage fractures should be repaired by ORIF. If there is associated injury to endolaryngeal soft tissue, the cartilage

fractures should be repaired first to provide a stable framework for the soft tissue repair. If there is no associated injury to endolaryngeal soft tissue, the cartilage fractures can be repaired without entering the larynx.

Wire, nonabsorbable suture, resorbable miniplates, or permanent miniplates can be used. Miniplate fixation is preferred because it is more likely to result in cartilaginous union rather than fibrous union and maintains better anatomic reduction of the fractures.[187,199] Resorbable plates are made of polylactic acid copolymer and absorb within 1 to 2 years.[200] If there is loss of cartilage in the anterior cricoid ring, the infrahyoid strap muscles can be sutured into the defect to help maintain patency of the airway.

LARYNGEAL STENTING

Laryngeal stents are indicated in the setting of extensive mucosal injury and lacerations involving the anterior commissure in order to prevent web formation and laryngeal stenosis. They are also indicated in the setting of multiple cartilaginous fractures that cannot be stabilized by ORIF such as in a gunshot wound to the larynx. If a laryngeal stent is placed, a tracheostomy must also be performed as the stent will obstruct the airway at the level of the larynx. It is also critical that the stent is secured such that it cannot migrate. Montgomery and Eliachar stents are two commercially available laryngeal stents that are commonly used in the setting of laryngeal trauma.[201] If preformed stents are not available, a stent can be fashioned from an endotracheal tube, or a finger can be cut from a surgical glove and filled with soft material such as Gelfoam. Stents are left in place for approximately 2 weeks and are then removed endoscopically. If left in place for a prolonged period of time, they can promote the formation of granulation tissue.

INJURY TO THE RECURRENT LARYNGEAL NERVE

Vocal cord immobility in the setting of laryngeal trauma may indicate injury to the recurrent laryngeal nerve. If this occurs as a result of blunt trauma, these injuries can typically be observed because it is unlikely that the nerve has been transected and spontaneous recovery may occur. In the case of penetrating trauma, the neck should be explored, and if the recurrent laryngeal nerve is found to be severed, it should be repaired primarily. The recurrent laryngeal nerve contains fibers that adduct and abduct the vocal cord, so normal mobility of the vocal cord will not be restored if the recurrent laryngeal nerve is repaired; however, bulk and tone of the vocal cord can be preserved. This can improve voice and swallowing.[202] If primary reanastomosis of the recurrent laryngeal nerve is not possible, the ansa hypoglossi, which innervates the infrahyoid strap muscles, can be redirected and sutured to the distal stump of the recurrent laryngeal nerve.

LARYNGOTRACHEAL SEPARATION

Laryngotracheal separation is the most severe form of laryngeal trauma (Table 25-5). Patients typically present with airway obstruction but may be less symptomatic than expected if fascial tissue anterior to the airway remains intact. The trachea will retract inferiorly, so a low tracheotomy should be performed. If the cricoid is intact, nonabsorbable suture should be placed from the superior aspect of the cricoid to the inferior aspect of the second tracheal ring, tying the knots extraluminally. If this is not possible due to the extent of injury, then thyrotracheal anastomosis can be performed.

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Endovascular Commentary to Chapter 25: Neck and Larynx

Joseph J. DuBose

In this edition, Dr. Sperry and colleagues have constructed an excellent treatise on the optimal evaluation and management of injuries to the neck. In review of their work, I am struck by the emerging importance of imaging and therapeutic technologies and their evolving role in optimal care of these injuries.

As an example, while a comprehensive understanding of the neck zones remains integral to decision making in the unstable or overtly injured patient, the value of computed tomographic (CT) imaging has evolved as an increasingly valuable element of evaluation for stable patients. In the not so distant past, the zone of injury of the neck dictated the initial operative approach along with the consideration of possible extension to the other incisions. For the stable patient, however, CT and other imaging adjuncts now provide the surgeon an opportunity to more precisely guide an operative plan, a particularly valuable resource at the extremes of zones I and III, where operative intervention may require extension into the chest or advanced skull-based exposures.

The authors also outline briefly the limited data available on the utilization of endovascular adjuncts in the care of vascular injuries to the neck. Endovascular skill sets afford additional options for diagnosis, proximal control of injury with intraluminal balloons to mitigate blood loss during subsequent operative exposure, and even definitive management options. These capabilities are emerging quickly, particularly as many trauma centers incorporate endovascular practitioners and hybrid operating rooms into their trauma teams.

Among stable patients without overt signs of injury, the selection for and timing of potential endovascular therapy remain areas of needed investigation. As the authors outline, data from Denver Health Medical Center and others have blunted the initial enthusiasm for early, aggressive coverage of blunt cerebrovascular injuries (BCVIs) with endovascular stent grafts. Although traumatic pathologies are very different from those of atherosclerotic disease, the concern for higher stroke rates with endovascular versus open treatment is a concern in the wake of multicenter studies.[1]

Other endovascular adjuncts, however, would seem to represent valuable options for use. In my opinion, the hybrid pairing of endovascular capabilities with open surgical management has significant potential application for vascular injuries to the neck. I envision that there will come a time when endovascular adjuncts will more effectively supplement optimal management in ways that are just now being better appreciated. For stable patients, it would make sense that proximal balloon occlusion used for hemorrhage control should serve as a natural fit in this fashion. If successful, descriptions of Foley catheters through wounds and balloon catheters placed into vessels through open incisions that are welling with blood may become relics of the pre-endovascular era.

In particular, it is intriguing to observe the changes in management with regard to BCVIs. In my practice, most blunt injuries can be observed and treated with anticoagulation/antiplatelet therapy and medical management. Repeat imaging is then obtained at 24 to 48 hours. For BCVI injuries that improve, antiplatelet therapy is continued, and additional imaging is obtained at 1 week and monthly thereafter. During long-term follow-up, the risks and benefits of antiplatelet and anticoagulation therapy must be weighed against the potential risk of stroke that might occur without treatment. If the injury heals, medical therapy can be discontinued.

Recognizing that these data are not well established, I advise transitioning to a simple 81-mg aspirin daily after the first month for a grade I or II BCVI that does not progress, recognizing that the greatest risk of cerebrovascular accident from these injuries is likely early in the postinjury course.

For grade III BCVI/pseudoaneurysm, initial medical management is almost universally appropriate, but careful attention must be paid to the 24-/48-hour and 1-week interval imaging. If progression or increase in size occurs at this interval or in a more delayed fashion, coverage of the region with an endovascular stent graft is warranted. It must be appreciated, however, that the placement of these stents represents an inherent thromboembolic risk that requires lifelong antiplatelet therapy (often dual therapy in this regard) possibly paired with anticoagulation. The optimal regimen is not well established, because long-term data are not available. The issue is further confused by the availability of more oral anticoagulation agents with little to no data supporting their optimal use universally. Regardless of the pharmacologic agents employed, repeat imaging for these patients should probably be employed at 1-, 3-, and 6-month intervals initially. Thereafter, 6-month or annual imaging or clinical exam is advisable.

Grade IV BCVI injuries (occlusion) represent a clinical challenge and are most commonly observed. The role of antiplatelet or anticoagulation therapy for asymptomatic patients is unclear, while the potential benefit is thought to be the mitigation of propagation of clot proximal and distal to the occlusion. I do not routinely use any medical therapy for asymptomatic occlusions but do conduct 1-week and 1-month follow-ups to look for evidence of propagation of clot.

Grade V BCVI transections, particularly those that cannot be easily repaired with open intervention, are amenable to embolization for proximal control of ongoing hemorrhage. For distal injuries, such as those at high carotid or vertebral locations, sound principles often involve both proximal and distal embolization if the region cannot be covered by a stent graft to restore in-line flow. When required, distal embolization is a technically demanding procedure requiring traverse of the circle of Willis and specialized catheter/device capabilities most commonly found in the skill sets of neuro-interventional providers—a key partner in these situations.

High carotid–jugular fistulas, although uncommon, represent another unique endovascular challenge. As with other high injuries, a specific subset of skills and devices inherent to neuro-interventional training (and much less commonly to vascular surgeons) is typically required. Although some of these fistulas can be observed and followed with repeat imaging, many require treatment. Stent graft coverage of the arterial segment, if it can be achieved, is typically curative. As an alternative, if embolization does not compromise the distribution of blood flow through the circle of Willis, this approach is well tolerated.

The lack of long-term data and study in this particular area of trauma remains a dilemma. Data are sorely needed to define optimal utilization of endovascular capabilities (or any treatment modality for that matter) and study the natural history of specific lesions, particularly focal dissections of traumatic origin or small pseudoaneurysms. It is this author's hope that ongoing efforts to study vascular injury in a longitudinal way, including through the American Association for the Surgery of Trauma's PROspective Observational Vascular Injury Treatment (PROOVIT) registry,[2,3] will provide some useful answers in this regard.

The authors are to be congratulated on an excellent contribution. As one would expect from their cumulative reputations and known experience, this chapter is certainly a "must read" for any surgical provider who might be called upon to care for trauma victims with injuries to this body region.

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26

Spinal Cord and Spine

Philip F. Stahel • Benjamin C. Dorenkamp • Michael E. Janssen

KEY POINTS

- The presence of an unstable spinal injury is presumed in all trauma patients until proven otherwise.
- Complete and thorough spinal evaluation and neurologic examination are mandatory in all critically injured patients.
- Strict log-roll precautions and cervical rigid-collar immobilization should be continued until unstable injuries are ruled out or identified and managed by early proactive surgical treatment protocols.
- Computed tomography is the standard initial diagnostic trauma workup in patients requiring imaging. Magnetic resonance imaging is indicated on a case-by-case basis after formal spine surgery consultation.
- An accurate classification of spine injuries using validated classification systems facilitates surgical decision making and serves as a basis to guide treatment.
- Early mobilization of critically injured patients with spinal injuries is essential. This requires either spinal clearance or spinal stabilization by surgical means.
- Spinal clearance should be provided within 24 hours of admission to minimize the risk of preventable immobilization-related complications.
- A standardized *spine damage control* protocol allows stabilization of unstable thoracic and lumbar spine fractures within 24 hours and subsequent mobilization of patients without restrictions.
- Unstable cervical spine injuries benefit from halo-vest application or Gardner-Wells tong traction until definitive surgical fixation is performed.
- The use of steroids is considered obsolete in the management of acute traumatic spinal cord injury.

INTRODUCTION

The scope of this chapter is to provide the trauma surgeon a pragmatic approach on how to coordinate the care of the multiply injured patient who presents with associated spinal injuries. The chapter focuses on current strategies for initial assessment and management of spinal injuries relevant to trauma surgeons with respect to the decision making for the integrated care of the critically injured patient. Pertinent issues include the conundrum of how to recognize an unstable spine injury, when and how to clear the cervical spine, the role of steroids in the management of spinal cord injury, the optimal timing of tracheostomy in spinal cord–injured patients, and the coordination of care for associated spine injuries in the multiply injured patient. Understanding these critical aspects related to the care of spinal injuries will allow optimization of the coordination of care for these highly vulnerable patients at risk of sustaining delayed "second-hit" insults and adverse outcomes.

INITIAL ASSESSMENT AND DIAGNOSTIC WORKUP

The presence of an associated spinal injury must be assumed in any multiply injured patient until proven otherwise.[1] Of note, approximately 10% to 15% of all trauma patients with severe head injuries have an associated cervical spine injury.[2] It is important to understand that most spine injuries do not present with a neurologic impairment. Pain or tenderness anywhere along the spine, from the occiput to the sacrum, should raise the concern for a spinal injury. The key imperative in the acute management of a trauma patient with a suspected spine injury consists of application of a cervical collar, exact documentation and timing of the findings, and immediate evacuation to a designated trauma center.[3] A cervical collar must be applied in all trauma patients until cervical spine injuries are either confirmed as absent (see spinal clearance protocols provided later) or identified and treated appropriately. The unstable spine is at risk for injury from

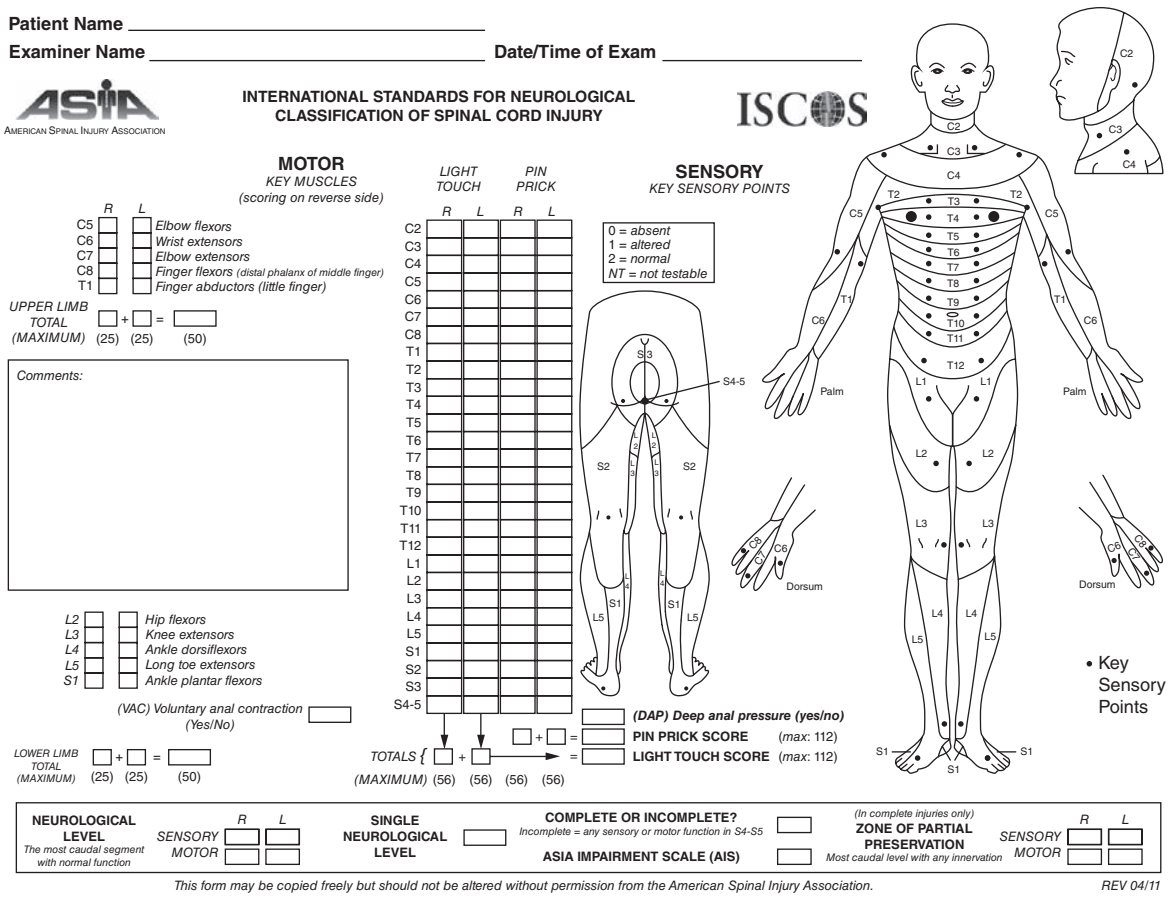
careless manipulation. Therefore, strict log-roll precautions should be maintained until spinal injuries are excluded or spinal stability is restored.[4] If a long spine board has been applied for transport, this must be removed on arrival in the emergency department in order to avoid pressure sores from prolonged immobilization.[3] The entire posterior spine should be inspected and palpated for local tenderness and deformities while adhering to log-roll precautions. This requires a team of four to five health care personnel to log-roll a patient with simultaneous in-line cervical stabilization. The paraspinal soft tissues should be inspected for evidence of swelling, malalignment, or bruising. Systematic palpation of the spinous processes of the entire spinal column can help to identify and localize a spinal injury because significant gapping between processes can occur in flexion–distraction injuries and spinal fracture–dislocations.[4] During inspection of the face and trunk, it is important to keep in mind that certain injuries can be associated with significant visceral and axial skeletal injuries. Facial trauma and head injuries should alert the examining physician to the possibility of an injury to the cervical spine.[2] An abrasion caused by a shoulder restraint in a motor vehicle occupant can be associated with significant injuries to the cervical spine and cervicothoracic junction. Similarly, lap belt contusions should heighten suspicion for flexion–distraction injuries to the thoracolumbar spine. These can also be associated with visceral injury. Calcaneal fractures from significant decelerations, including falls from heights, are associated with axial loading injuries of the thoracic and lumbar spine.[5] Subsequent to a systematic inspection, a complete neurologic examination is performed (see later). The goal of repeated assessments is to identify and provide initial treatment of potentially unstable spinal fractures from both a mechanical and a neurologic basis. This allows for a longitudinal comparison of the neurologic status and early determination of a potentially progressive neurologic deficit. Spinal injuries are identified and worked up during the secondary survey, after successful lifesaving measures. Most spine injuries do not present with a neurologic impairment. If a patient has signs of numbness, tingling sensation, or paralysis to any extremity, a serious injury to the spinal cord must be suspected. Impairment of bladder and bowel function is frequently missed on initial evaluation, which speaks to the absolute necessity of a thorough neurologic exam, as discussed later.[6]

The diagnostic workup of spinal injuries includes computed tomography (CT) scan and magnetic resonance imaging (MRI) for visualization of soft tissue injuries to ligaments and intervertebral disks, epidural bleeding, dural tears, spinal cord contusions and lacerations, and intramedullary lesion expansion over time. Importantly, an MRI should be exclusively obtained only in patients who are hemodynamically stable and adequately resuscitated. The initial workup of multiply injured patients by multislice whole-body CT scans provides thin-section images of the entire spine, along with two- and three-dimensional reconstructions. In this regard, the general availability of multislice CT scanners has eliminated the historic necessity of obtaining conventional radiographs of the entire spine to prevent missing additional vertebral fractures at a different level, which occur in approximately 10% of all cases.[7] Of note, the exclusive finding of transverse process fractures in the initial CT scan does not warrant a spine surgical consultation per se, unless there is a suspicion for associated unstable spinal column injuries based on radiographic findings or presence of neurologic symptoms.

NEUROLOGIC EVALUATION

A thorough neurologic exam is mandatory in any trauma patient with suspected spinal injury. Cranial nerve abnormalities can be suggestive of occipitocervical junction injuries. Four-extremity motor examination should focus on the C5–T1 and L2–S1 myotomes. Truncal and four-extremity sensory exams should document light-touch and pinprick sensation throughout all dermatomes. Subjective numbness or tingling sensation or paralysis to any extremity implies presence of a serious injury to the spinal cord. The level and severity of a spinal cord injury can be determined using a systematic chart published by the American Spinal Injury Association (ASIA).[8] This chart illustrates the key sensory points that correspond to specific neurologic levels in the spinal cord (Fig. 26-1).

When assessing neurologic impairment in an individual who sustained a spine injury, it is crucial to document the exact time of assessment because neurologic deterioration may occur over time.[6] The extent of neurologic injury is stratified into complete (ASIA grade A) or incomplete (ASIA grades B–D), with ASIA grade E reflecting a normal neurologic status.[9] Spinal cord injury is further stratified into paraplegia (paralysis of the lower extremities), resulting from thoracic and lumbar spine injuries, and quadriplegia (paralysis of all four extremities), which originates from cervical spine injuries. With incomplete injuries, the patient has some extent of preserved neurologic function below the level of injury, which is associated with a better outcome prediction compared to complete injuries, where the prognosis is dismal. The neurologic exam should include reflex examination with a focus on triceps, brachioradialis, and patellar and Achilles tendons. Hyperreflexivity may be indicative of a spinal cord injury, whereas areflexia is reflective of spinal shock with temporary absence of all reflexes. Testing for pathologic reflexes includes the Hoffman sign, Babinski sign, and presence of clonus in the case of impaired upper motor neuron downregulation. A rectal examination is part of the mandatory exam and should document anal resting and voluntary tone, anal and perianal sensation (light touch and pinprick), and spinal reflexes (anal wink and bulbocavernosus reflex; the latter requires Foley catheter placement in females). Obtunded patients should be observed for voluntary movements and withdrawal activity with objective assessments taken from reflex and rectal examinations. Return of the bulbocavernosus reflex is reflective of resolution of spinal shock. In contrast to spinal shock, neurogenic shock represents a hemodynamic entity characterized by hypotension and bradycardia resulting from impaired sympathetic outflow tracts in the injured spinal cord.



Muscle function grading

0 = Total paralysis

1 = Palpable or visible contraction

2 = Active movement, full range of motion (ROM) with gravity eliminated

3 = Active movement, full ROM against gravity

4 = Active movement, full ROM against gravity and moderate resistance in a muscle specific position.

5 = (Normal) active movement, full ROM against gravity and full resistance in a muscle specific position expected from an otherwise unimpaired peson.

5* = (Normal) active movement, full ROM against gravity and sufficient resistance to be considered normal if identified inhibiting factors (ie, pain, disuse) were not present.

NT = Not testable (ie, due to immobilization, severe pain such that patient cannot be graded, amputation of limb, or contracture of >50% of the range of motion).

ASIA impairment (AIS) scale

- ❑ **A = Complete.** No sensory or motor function is preserved in the sacral segments S4-S5.
- ❑ **B = Sensory in complete.** Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5 (light touch, pin prick at S4-S5: or deep anal pressure [DAP]), AND no motor function is preserved more than three levels below the motor level on either side of the body.
- ❑ **C = Motor incomplete.** Motor function is preserved below the neurological level**, and more than half of key muscle functions below the single neurological level of injury (NLI) have a muscle grade less than 3 (Grades 0–2).
- ❑ **D = Motor incomplete.** Motor function is preserved below the neurological level**, and at least half (half or more) of key muscle functions below the NLI have a muscle grade ≥ 3.
- ❑ **E = Normal.** If sensation and motor function as tested with the ISNCSCI are graded as normal in all segments, and the patient had prior deficits, then the AIS grade is E. Someone without an initial SCI does not receive an AIS grade.

**For an individual to receive a grade of C or D, ie, motor incomplete status, they must have either (1) voluntary anal sphincter contraction or (2) sacral sensory sparing with sparing of motor function more than three levels below the motor level for that side of the body. The standards at this time allows even non-key muscle function more than 3 levels below the motor level to be used in determining motor incomplete status (AIS B vs C).

NOTE: When assessing the extent of motor sparing below the level for distinguishing between AIS B and C, the *motor level* on each side is used; whereas to differentiate between AIS C and D (based on proportion of key muscle functions with strength grade 3 or greater) the *single neurological level* is used.

Steps in classification

The following order is recommended in determining the classification of individuals with SCI.

1. Determine sensory levels for right and left sides.
2. Determine motor levels for right and left sides.
   Note: in regions where there is no myotone to test the motor level is presumed to be the same as the sensory level, if testable motor function above that level is also normal.
3. Determine the single neurological level.
   This is the lowest segment where motor and sensory function is normal on both sides, and is the most cephalad of the sensory and motor levels determined in steps 1 and 2.
4. Determine whether the injury is Complete or Incomplete. (ie, absence or presence of sacral sparing)
   If voluntary anal contraction = **No** *AND all S4-5 sensory scores* = 0 *AND deep and pressure* = **No**, *then injury is COMPLETE. Otherwise, injury is incomplete.*
5. Determine ASIA impairment scale (AIS) grade:

Is injury Complete?
If **YES**, AIS=A and can record ZPP (lowest dermatome or myotome on each side with some preservation)

NO ↓

Is injury motor Incomplete?
If **NO**, AIS=B

YES ↓

(YES=voluntary anal contraction OR motor function more than three levels below the motor level on a given side, if the patient has sensory incomplete classification)

Are at least half of the key muscles below the single neurological level graded 3 or better?

NO ↓ AIS=C

YES ↓ AIS=D

If sensation and motor function is normal in all segments, AIS=E
Note: AIS E is used in follow-up testing when an individual with a documented SCI has recovered normal function. If at initial testing no deficits are found, the individual is neurologically intact; the ASIA Impairment Scale does not apply.

FIGURE 26-1 International standards for neurological classification of spinal cord injury. (Reproduced with permission from the American Spinal Injury Association.[8])

NEUROLOGIC SYNDROMES

Anterior Cord Syndrome

Anterior cord syndrome represents an entity of *incomplete* spinal cord injury resulting from vascular compromise in the anterior spinal artery distribution and subsequent ischemic injury to the anterior two-thirds of the cord, which can occur after blunt trauma mechanisms or ischemic injuries (Fig. 26-2). Clinically, patients present with loss of motor function and pain and temperature sensation below the level of injury from involvement of the ventrally located lateral corticospinal and spinothalamic tracts. Patients typically retain proprioception and the ability to sense vibration and deep pressure from preservation of the posterior columns of the spinal cord. The chance of a relevant clinical recovery in anterior cord syndromes is poor due to the irreversible ischemic neuronal tissue damage.

Central Cord Syndrome

Traumatic central cord syndrome is associated with a contusion, ischemia, or hemorrhage in the central portions of the spinal cord due to traumatic injury sustained in the cervical or upper thoracic spine. The syndrome is characterized by weakness in the arms with "burning hands" and relative sparing of lower extremity motor function, associated with variable sensory loss. Traumatic central cord syndrome typically results from a cervical hyperextension injury in patients with preexisting degenerative changes and narrowing of the spinal canal. Clinically, the upper extremities are more involved than the lower extremities due to the more central location of the upper extremity axons within the spinal cord tracts. Patients typically regain the ability to walk but have more limited return of function to the upper extremities.

Brown-Séquard Syndrome

This entity represents an *incomplete* spinal cord syndrome resulting from a hemitransection of the spinal cord frequently resulting from penetrating injuries. The syndrome is characterized by unilateral damage to the corticospinal tract, spinothalamic tract, and dorsal columns. The clinical presentation consists of loss of ipsilateral light touch sensation, proprioception, and motor function and contralateral loss of pain and temperature sensation.

Posterior Cord Syndrome

This syndrome is rare and results from involvement of the dorsal columns with subsequent loss of proprioception and vibration and preserved motor function. The prognosis is variable, with many patients experiencing difficulty walking due to the deficit in proprioceptive sensation.

Cervical Root Syndrome

This injury pattern is reflective of an isolated nerve root injury that causes a deficit in sensation and motor function secondary to an acute disk herniation or facet fracture, subluxation, or dislocation.

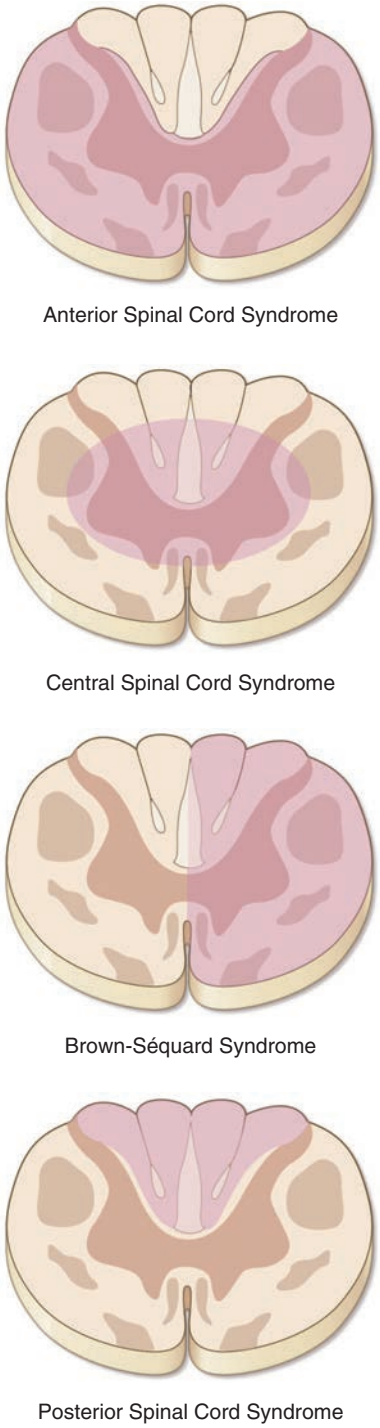


FIGURE 26-2 The most common patterns of incomplete spinal cord injury.

Conus Medullaris Syndrome

Injury to the conus medullaris, which is typically located at the level of the L1–L2 intervertebral space, can produce

mixed upper and lower motor neuron findings. Isolated injury to the conus may result in loss of bowel and bladder control without sacral sparing. Nerve roots that "escape" the injury can allow preserved lower extremity motor and sensory function despite injury to the distal tip of the spinal cord.

Cauda Equina Syndrome

The cauda equina extends distal to the conus and is composed of the lumbar and sacral nerve roots. Injury to the cauda equina results in lower motor neuron findings with sensory loss and motor dysfunction. Involvement of the lower sacral roots can result in bladder and bowel dysfunction.

ACUTE MANAGEMENT STRATEGIES

The initial management principles for trauma patients with associated spinal injuries consist of protection of airway and breathing/ventilation, ensuring adequate oxygenation, control of blood loss, and maintenance of adequate blood pressure according to the established guidelines by the Advanced Trauma Life Support (ATLS) protocol.[3,10] Presence of an unstable spinal injury must be suspected in any patient who sustains a high-energy trauma mechanism, independent of a neurologic impairment. A cervical collar is kept in place until formal spine clearance (see protocols later), which usually requires additional radiographic workup. When securing the airway, care is required during intubation to prevent hyperextension of the neck that might cause an iatrogenic injury to the cervical spine or spinal cord. Endotracheal intubation must be performed with in-line cervical traction or by fiberoptic assistance. Maintenance of oxygenation and avoiding hypotension are key to minimizing the potential for second-hit insults to the vulnerable spinal cord. In a selected subset of patients with spinal cord injuries, hypotension may result from neurogenic shock due to disruption of sympathetic output to the cardiovascular system.[11] Neurogenic shock is characterized by bradycardia in the presence of hypotension. These patients typically require the use of a vasopressor (eg, Neo-Synephrine or Levophed) to maintain systolic blood pressure. Occasionally in some patients with profound bradycardia, chronotropic support (eg, dopamine) or even temporary pacing is necessary to maintain an acceptable heart rate. Patients with spinal cord injuries above the level of C5 are more likely to require intubation and mechanical ventilation.[11]

HOW TO RECOGNIZE AN UNSTABLE SPINE INJURY

One of the most important tasks for the treating spine surgeon is to determine the presence or absence of spinal stability. Most spine surgeons agree with the general definition that spinal stability refers to the ability of the spine to maintain its alignment and protect the neural structures during normal physiologic loads.[12] Biomechanically, spinal instability refers to an abnormal response to applied loads and can be characterized by motion in spinal segments beyond the normal constraints. In a clinical scenario, defining *spinal stability* remains challenging and a topic of ongoing debate. The working definition of spinal stability takes into consideration that under physiologic loads (the influence of gravity on body mass) the spine will not experience increasing deformity, onset of neurologic impairment, or a drastic increase in patients' subjective level of pain.[12] Reciprocally, unstable spine injuries are at risk for progressive deformity and neurologic compromise (or both), which may represent the basis for considering surgical spine stabilization and decompression of the spinal cord. If spinal stability can be confirmed without the need for surgical intervention, patients should be cleared from log-roll precautions followed by early mobilization with or without adjunctive bracing. If spinal injuries are deemed unstable, early surgical treatment should be considered to prevent complications related to prolonged bed rest and immobilization (eg, pressure sores, pulmonary and thromboembolic complications).

Spinal fractures, traumatic dislocations, and fracture–dislocations are best classified by the comprehensive AO Foundation/Orthopaedic Trauma Association (AO/OTA) classification system, which is based on the alphanumeric classification published by Magerl et al[13] in 1994 (Fig. 26-3).

The AO/OTA classification codes the anatomic spine region by a number (cervical: 51; thoracic: 52; lumbar: 53) and the injury pattern/severity by an alphanumeric combination (A, B, C and 1, 2, 3).[14] The clinical implication of classifying spinal injuries by the AO/OTA system is that the alphanumeric code reflects injury severity and guides treatment (operative vs nonoperative) based on spinal stability. In brief, A-type spinal injuries represent *axial loading* trauma mechanisms leading to compression fractures (A1), vertebral split fractures (A2), or burst fractures (A3). In contrast, B-type injuries reflect unstable fractures and fracture–dislocations originating from *flexion/distraction* (B1, B2) or *hyperextension* (B3) trauma mechanisms. Finally, C-type injuries are by definition *rotationally unstable* and include rotational wedge/split/burst fractures (C1), flexion–distraction or hyperextension injuries with rotation (C2), or complete rotational shearing injuries (C3). The incidence of associated neurologic (spinal cord) injury increases with the alphanumeric fracture classification, from nearly 0% in stable A1-type compression fractures to close to 100% in C3-type injuries (Fig. 26-3).[6] The C3-type injury is representative of the worst spinal injury pattern (so-called Holdsworth injury, slice fracture, or traumatic spondyloptosis) and is associated with an almost 100% risk of spinal cord injury or spinal cord transection.[15]

The extent of vertebral displacement (spondylolisthesis) is classified by the Meyerding grading system (Fig. 26-4).[16] This classification is based on radiographic measurements on lateral views of the according spine segment. The Meyerding classification describes four grades of severity of spondylolisthesis, based on the calculated percentage of vertebral slip: 0% to 25% (grade I), 25% to 50% (grade II), 50% to 75% (grade III), and 75% to 100% (grade IV).

A traumatic dislocation of the spine (traumatic spondyloptosis) is almost exclusively related to high-energy

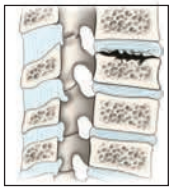
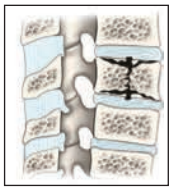
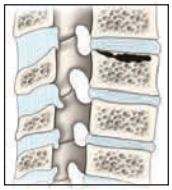
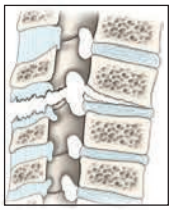
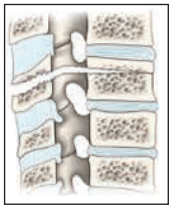
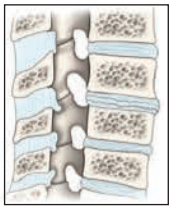
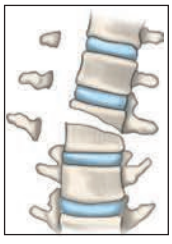
Fracture type (AO/OTA)	Stability	Risk of SCI	Management
A-type: Axial compression		0%	
 A1: Impaction/ compression	Stable		Nonoperative
 A2: Split	Stable		Nonoperative
 A3: Burst	Stable/unstable*		Nonoperative/operative*
B-type: Flexion/distraction or hyperextension			
 B1: Flexion/ distraction	Unstable		Operative
 B2: Chance fracture	Unstable		Operative
 B3: Hyperextension	Unstable		Operative
C-type: A or B type with rotation			
 C1: Rotational wedge	Unstable		Operative
C2: Rotational flexion/ extension	Unstable		Operative
C3: Rotational shear (Holdsworth slice fracture)	Unstable		Operative
		100%	

FIGURE 26-3 Spinal fractures, traumatic dislocations, and fracture–dislocations are best classified by the comprehensive AO Foundation/ Orthopaedic Trauma Association (AO/OTA) classification system, which is based on the alphanumeric classification published by Magerl et al[13] in 1994.

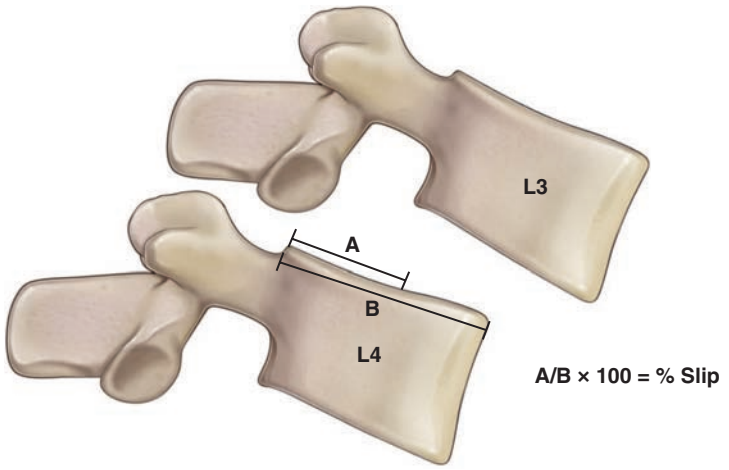


FIGURE 26-4 The extent of vertebral displacement (spondylolisthesis) is classified by the Meyerding grading system.

acceleration/deceleration trauma mechanisms. The cervical spine is particularly vulnerable due to its flexible fixation between the head and the thorax. Rigidity at these two points (the occipitocervical junction and the cervicothoracic junction) causes vulnerability to occur at the intervening cervical segment between. For the same reason, the thoracolumbar junction is particularly vulnerable to traumatic fractures and fracture–dislocations (Fig. 26-5).[16]

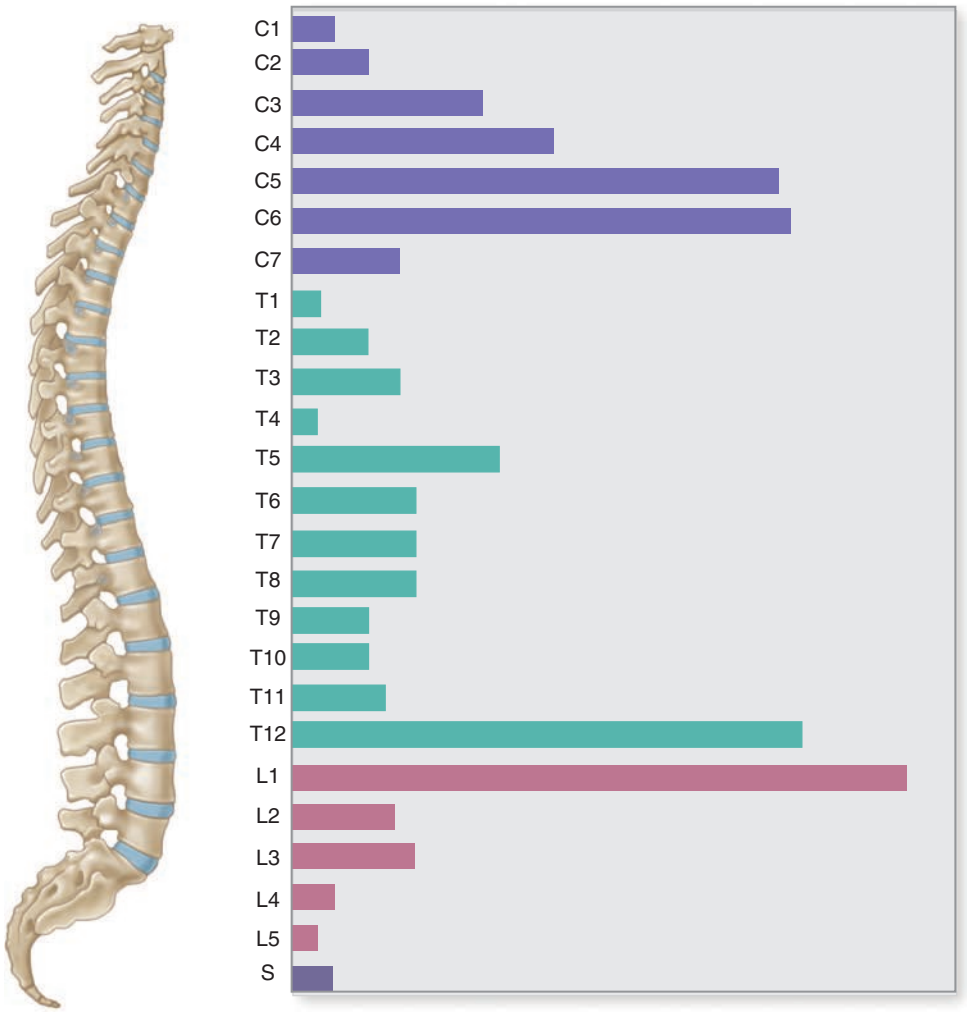


FIGURE 26-5 The thoracolumbar junction is particularly vulnerable to traumatic fractures and fracture–dislocations.

TABLE 26-1: Subaxial Injury Classification and Severity Scale (SLIC)

Injury characteristic	Points
Injury morphology	
No abnormality	0
Compression	1
Burst	+1 (=2)
Distraction (eg, facet perch, hyperextension)	3
Rotation/translation (eg, facet dislocation, unstable "tear-drop" or advanced stage flexion compression injury)	4
Discoligamentous complex (MRI)	
Intact	0
Indeterminate (eg, isolated interspinous widening, MRI signal change only)	1
Disrupted (eg, widening of anterior disk space, perched facet/dislocation, kyphotic deformity	2
Neurologic status	
Intact	0
Root injury	1
Complete cord injury	2
Incomplete cord injury	3
Ongoing cord compression in setting of neurodeficit	+1

A summarized score <4 points suggests nonoperative management as an adequate treatment strategy, whereas a score ≥4 points implies consideration of surgical management.

MRI, magnetic resonance imaging.

TABLE 26-2: Thoracolumbar Injury Classification and Severity Score (TLICS)

Injury characteristic	Qualifier	Points
Injury morphology		
Compression		1
Burst		+1 (=2)
Rotation/translation		3
Distraction		4
Neurologic status		
Intact		0
Nerve root		2
Spinal cord/conus medullaris	(incomplete)	3
Spinal cord/conus medullaris	(complete)	2
Cauda equina		3
Posterior ligamentous complex (MRI)		
Intact		0
Suspected/indeterminate		2
Disrupted		3

A summarized score <4 points suggests nonoperative management as an adequate treatment strategy, whereas a score ≥4 points implies consideration of surgical management.

MRI, magnetic resonance imaging.

Additional classification systems for vertebral fractures include the classic three-column classification by Denis, the Allen/Ferguson fracture classification, the White and Panjabi system, and the load sharing classification system for assessment of stable versus unstable burst fractures.[17-19] In addition, the Subaxial Injury Classification (SLIC) system for cervical injuries (Table 26-1) and the Thoracolumbar Injury Classification and Severity Score (TLICS) for thoracic and lumbar fractures (Table 26-2) offer guidance in the decision making for operative versus nonoperative management of spinal injuries.[20,21] Although these two widely used classification systems are helpful for guiding treatment, the respective scoring relies on availability of MRI for prediction of discoligamentous complex integrity.

WHEN TO CLEAR THE CERVICAL SPINE

The incidence of cervical spine injury is reported to be around 4% in the trauma patient.[22,23] Given the potential for catastrophic complications following a missed cervical spine injury, it is essential that patients be properly immobilized in rigid cervical collars at the time of rescue with continued immobilization until safe and protocolized cervical spine clearance.

During patient rescue, transport, and initial assessment and management, every attempt should be made to minimize the movement of the spinal column. Spinal precautions are aimed at avoiding secondary damage to the spinal cord by excessive bending or twisting in presence of potentially unstable injury patterns. The presence of neurologic symptoms must be assumed to represent a spinal cord injury with associated spinal column instability until proven otherwise.

Clearance of the cervical spine has represented a conundrum for many decades, due to the potential medical-legal implications related to missed spinal injuries and the risk of complications associated with prolonged spinal immobilization.[24] An observational cohort study from a Level I trauma center in the Netherlands revealed that more than half of all adult trauma patients admitted through the emergency department had indentation marks from their cervical collars and that the risk of developing a pressure ulcer increased by 66% per additional day of immobilization.[25] In particular, patients admitted to the intensive care unit (ICU), patients on mechanical ventilation, and patients with traumatic brain injury being monitored for intracranial pressure were noted to have increased complications associated with their cervical collars.[25] Additional risks of prolonged cervical spine immobilization in rigid collars include the following:

- Technical challenges for endotracheal intubation and airway management
- Technical challenges for central line placement

- Time delay for tracheostomies in spinal cord–injured patients
- Increased risk of aspiration and pulmonary infections
- Increased risk of skin breakdown and pressure ulcers
- Increased pain and discomfort in awake patients
- Impaired nursing care and patient experience

The published literature provides a multiplicity of protocols for clearance of the cervical spine in the trauma patient. In essence, the relevant decision-making algorithms are stratified into three main categories based on the underlying patient condition. These include (1) awake and alert patients without neurologic symptoms[26]; (2) awake and alert patients with presence of neurologic symptoms[27]; and (3) obtunded, intoxicated, or otherwise nonexaminable patients.[28]

For patients who are awake, alert, and hemodynamically stable, without any neurologic symptoms, there are standardized guidelines for cervical spine clearance guided exclusively by clinical findings. These include the National Emergency X-Radiography Utilization Study (NEXUS) low-risk criteria[29] (Fig. 26-6) and the Canadian Cervical Spine Rule (CCR) guideline[30] (Fig. 26-7).

The application of these clinical prediction tools allows for a rapid clinical clearance of the cervical spine without the need for radiographic imaging. Multiple validation studies and meta-analyses of the literature revealed a high sensitivity (98.1%) and negative predictive value (99.8%) for the NEXUS low-risk criteria and the CCR guideline to safely clear the cervical spine without radiographic imaging in alert, asymptomatic patients who are hemodynamically stable and are able to complete a functional range-of-motion examination in absence of distracting injuries or neurologic deficits.[26,27,31-33]

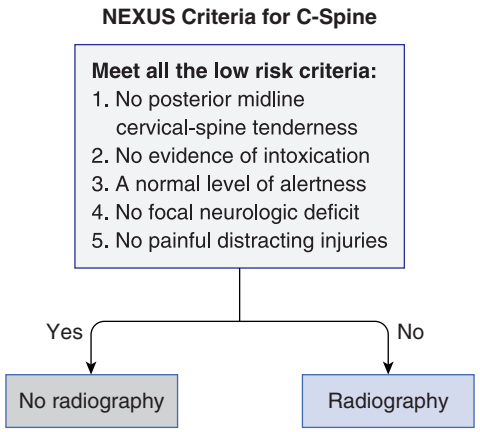


FIGURE 26-6 NEXUS (National Emergency X-Radiography Utilization Study) low-risk criteria for clearance of the cervical spine. (Reproduced with permission from Allen B, Ganti L, Desai B. *Quick Hits in Emergency Medicine.* New York, NY: Springer; 2013:59. Copyright © 2013. Springer Science + Business Media New York. Adapted from Hoffman JR, Wolfson AB, Todd K, Mower WR. Selective cervical spine radiography in blunt trauma: methodology of the National Emergency X-Radiography Utilization Study (NEXUS). *Ann Emerg Med.* 1998;32(4):461.)

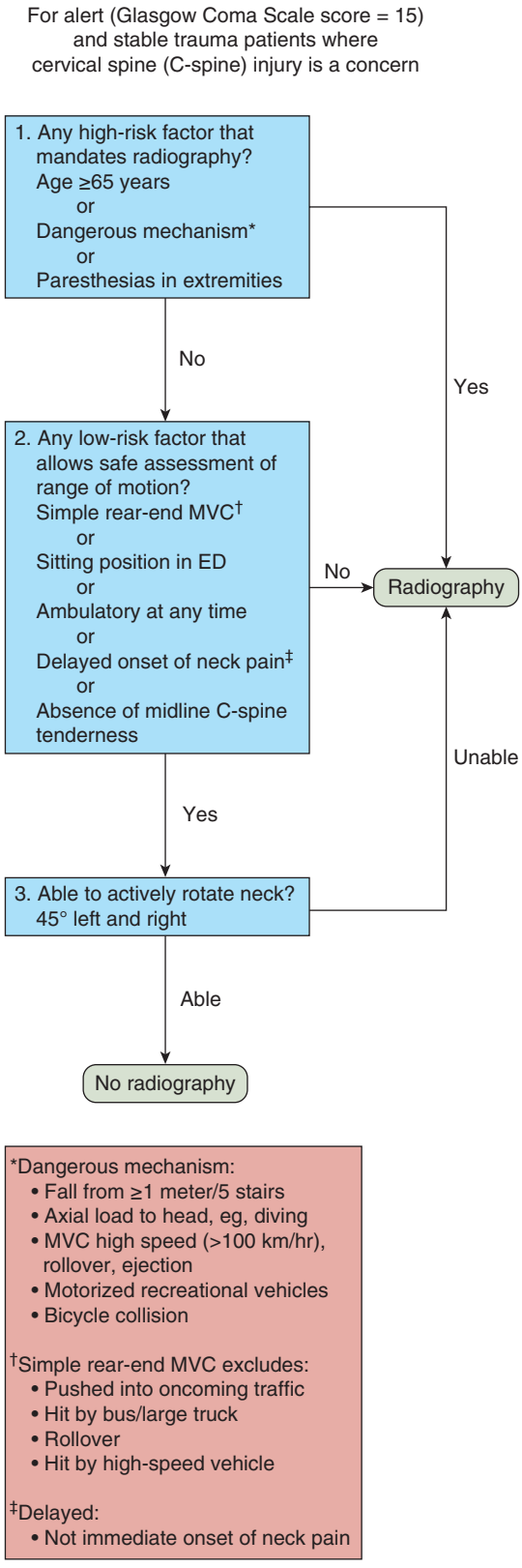


FIGURE 26-7 The Canadian Cervical Spine Rule for radiography in alert and stable trauma patients. ED, emergency department; MVC, motor vehicle collision. (Used with permission from Dr. Ian Stiell, Ottawa Hospital Research Institute. Copyright © Ian Stiell.)

Symptomatic patients with neurologic symptoms and obtunded or intoxicated patients who are not amenable to a clinical evaluation require advanced imaging studies. The use of upright plain radiographs of the lateral cervical spine is outdated and obsolete, based on a low reported sensitivity of just 45.5% in detecting presence of a spinal injury.[34] In contrast, current available evidence has unequivocally demonstrated that a normal high-quality CT scan alone is sufficient to allow safe clearance of the cervical spine in the nonexaminable patient.[28] In this regard, the 2015 guidelines by the Eastern Association for the Surgery of Trauma (EAST) recommend cervical collar removal after a negative high-quality cervical spine CT scan results in obtunded adult blunt trauma patients.[35] These recommendations by the EAST study group are based on a systematic review of the literature that revealed a negative predictive value of 100% for spinal clearance by a normal high-quality CT scan alone.[35] These insights were confirmed by the 2017 Western Trauma guidelines, which are based on a prospective multicenter observational study confirming that CT alone was effective for ruling out a clinically relevant cervical spine injury with a negative predictive value of 100%.[36] Based on these data, the escalation to further advanced imaging by MRI appears exclusively indicated in patients with an abnormal neurologic examination.[36]

Despite the unequivocal available evidence from the pertinent literature, it appears that there is an ongoing unjustified overutilization of MRI studies in the current clinical practice. For example, a large-scale survey of 22 trauma centers in England reported that 52% of the respondents were using MRI for clearance of the cervical spine in obtunded patients despite the presence of a negative high-quality CT scan.[37] The authors raised a call for greater awareness about the evidence from the EAST guidelines and the British Orthopaedic Association Standards for Trauma (BOAST) recommendations regarding the reliability of a normal CT examination in clearing the cervical spine in obtunded patients in the absence of focal neurology.[37] This recommendation is supported by a recent meta-analysis that reported an extremely low rate of positive findings by MRI in obtunded patients with blunt cervical spine trauma of only 0.12% in a pooled cohort of 5286 patients.[38] In addition to the low diagnostic yield, obtaining an MRI in obtunded patients has multiple technical ramifications and potential risks. These include unnecessary transports of critically injured patients, difficult patient positioning, suboptimal monitoring and access during a prolonged examination, the unjustified delay to discontinuation of cervical spine immobilization pending MRI clearance, and the unnecessary utilization of time and resources.[39,40]

In summary, the current peer-reviewed literature and international consensus-based guidelines support the discontinuation of cervical collar immobilization in the obtunded, intoxicated, or otherwise nonexaminable patient subsequent to a negative high-quality CT scan.

Figure 26-8 provides a summarized algorithm of the current evidence-based recommendations for cervical spine clearance after blunt trauma mechanisms in adult patients.

THE ROLE OF STEROIDS REVISITED

Despite current science unequivocally demonstrating a lack of effectiveness of high-dose methylprednisolone in the acute management of patients with spinal cord injuries, the issue remains a topic of daily debate and uncertainty among the involved care providers in the trauma bay. From a historic perspective, the consideration of high-dose steroids originated from presumed benefits in patients with brain tumors and head injuries in the 1960s and 1970s. After publication of the second National Acute Spinal Cord Injury Study (NASCIS-2) in 1990, the application of high-dose methylprednisolone for patients with acute spinal cord injury became a globally accepted standard of care for more than a decade.[41,42] A critical analysis of the NASCIS data, however, placed the use of high-dose steroids under scrutiny due to questionable benefits and the potential for inflicting unintentional harm, such as placing the patients at increased risk for pulmonary infections.[43] The large-scale, prospective, randomized, multicenter trial Corticosteroid Randomization After Significant Head Injury (CRASH) confirmed the notion of the unjustified experimental nature of high-dose steroids in the acute management of neurologic injuries. The CRASH trial was unexpectedly aborted after enrollment of about 10,000 patients, based on the unexpected finding of a drastically increased mortality in patients treated with methylprednisolone, compared to the placebo control group.[44] The extrapolation of the negative results from this large-scale trial implied that the uncritical administration of corticosteroids in the 1980s and earlier may have been the cause of preventable postinjury mortality.[45] In absence of new prospective randomized trials on the role of steroids in spinal cord injury, current guidelines and clinical recommendations consider the routine use of steroids for patients with acute spinal cord injury obsolete.[3] Selected neurologic injury patterns, including spinal contusions or traumatic central cord syndrome, allow consideration for a short course of steroids at the individual treating spine surgeon's discretion.

THROMBOEMBOLISM PROPHYLAXIS

Prophylaxis against venous thromboembolism (VTE) is a crucial consideration in patients with unstable spine fractures and spinal cord injuries. The most recent and updated American College of Chest Physicians CHEST guidelines for VTE prophylaxis were published in 2016.[46] All trauma patients with associated spinal injuries should have mechanical prophylaxis instituted as soon as possible with graduated compression stockings and/or sequential compression devices. Pharmacologic prophylaxis with unfractionated heparin or low-molecular-weight heparin (LMWH) should be initiated within 24 hours of admission or as soon as the acute risk for additional bleeding in the trauma patient is adequately controlled (ie, pending adequate response to resuscitation and reversal of postinjury coagulopathy).[47] LMWH appears to be more effective for VTE prevention and is associated with fewer bleeding complications than unfractionated heparin in patients with spine injuries.

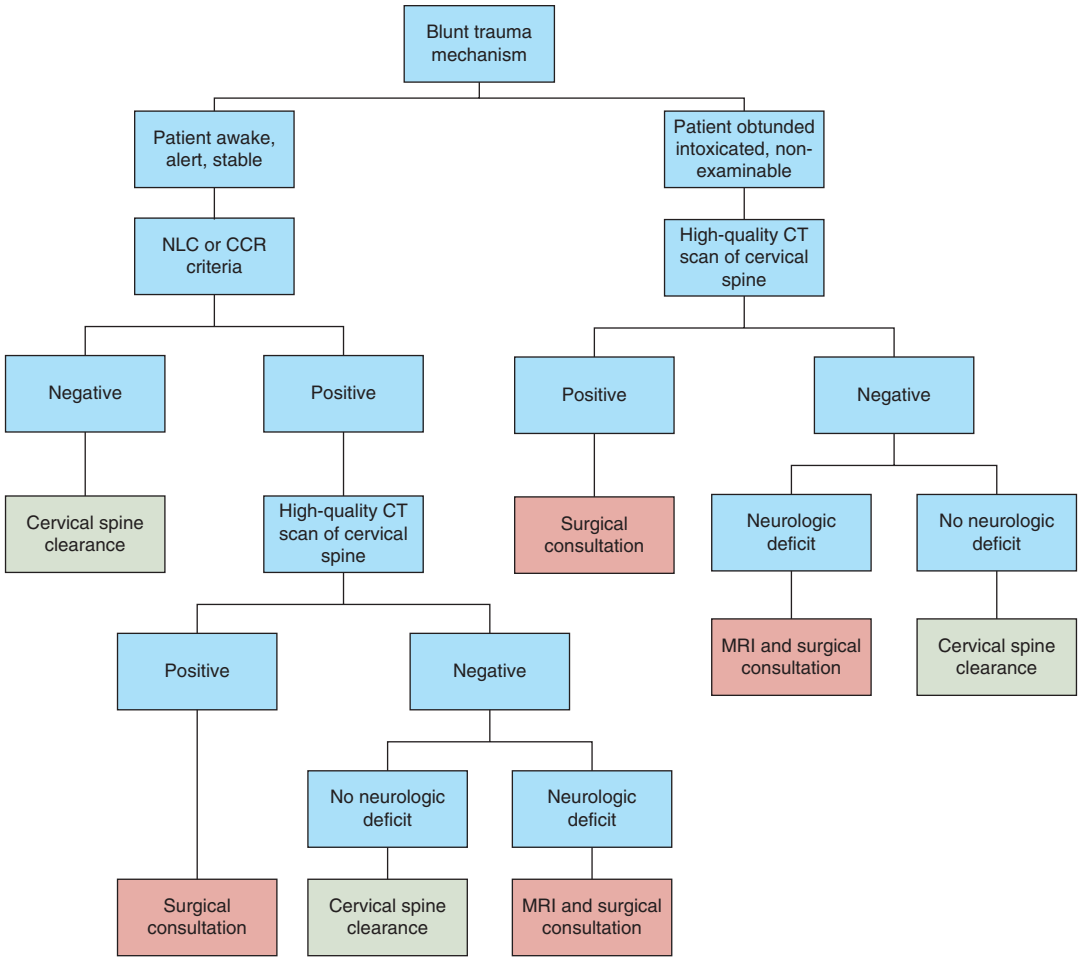


FIGURE 26-8 Algorithm for clearing the cervical spine in both awake and alert and obtunded or intoxicated patients. CCR, Canadian Cervical Spine Rule; CT, computed tomography; MRI, magnetic resonance imaging; NLC, NEXUS (National Emergency X-Radiography Utilization Study) low-risk criteria.

The duration of pharmacologic VTE prophylaxis is determined by the patient's mobility status and should be typically continued for 2 weeks in patients without neurologic injuries and for 6 to 12 weeks in the presence of spinal cord injury. If patients are poor candidates or have a contraindication to pharmacologic VTE prophylaxis due to increased risk of bleeding complications, then the early placement of a removable inferior vena cava filter should be considered as an alternative option.[47] Pharmacologic VTE prophylaxis is not indicated in low-risk situations (eg, for ambulatory patients with isolated stable A1-type thoracolumbar compression fractures).

TIMING OF TRACHEOSTOMY

One of the unresolved challenges in the management of ventilator-dependent trauma patients with associated unstable spine fractures and spinal cord injuries consists in defining the ideal timing of conversion to a tracheostomy.[48] The conundrum consists of coordinating the timing of early tracheostomy (the intent of which is to reduce the inherent risk of ventilator-associated pneumonia) and the timing of cervical spine fixation (if indicated for the management of unstable cervical injuries). Spine surgeons are generally worried that a preceding tracheostomy may increase the risk of a surgical site infection for a delayed anterior cervical discectomy and fusion (ACDF). This concern appears unjustified based on the existing literature, which demonstrates that ACDF surgery is safely performed in the presence of a prior tracheostomy, without an increased risk of a postoperative infection.[49] Because the dual incisions are typically located several centimeters apart, the tracheostomy wound can be safely draped off during the preparation for the ACDF procedure and does not appear to pose a risk of cross-contamination. In fact, these two procedures can be safely performed during the same operating room visit under the same anesthetic. At our own institution, the requirement for prolonged mechanical ventilation in patients is discussed as part of the overall surgical plan in the management of patients with spine fractures and spinal cord injuries. Our protocol attempts to coordinate the early timing of tracheostomy with the timing for the spinal fusion.

This proactive approach, tailored at decreasing the risk of preventable pulmonary infections and adverse outcomes in a highly vulnerable patient population, requires close cooperation between spine surgeons and the general surgery trauma team.[50]

TIMING OF SPINAL SURGERY

Unstable spine fractures, dislocations, and fracture-dislocations must be recognized early and treated in a timely fashion. This entails early closed reduction and application of Halo fixation for unstable cervical spine injuries and early open reduction and spinal fixation/fusion for unstable thoracic and lumbar injuries and for irreducible cervical spine dislocations. Examples of institutional protocols for the surgical management of thoracic and lumbar fractures/fracture–dislocations have been published elsewhere.[16] The widely disseminated practice of internally fixating unstable thoracic and lumbar fractures in polytrauma patients consists of either (1) a conservative approach of delayed spine fixation (after full resuscitation), or (2) a more proactive approach of early total care. Early total care often includes invasive anterior approaches, vertebral corpectomy, spinal canal decompression, and anterior spinal column stabilization/fusion. Many spine surgeons are discouraged from early spinal surgery based on the notion that multiply injured patients are frequently "too sick" to safely undergo surgical procedures within the first few days after major trauma.[51] The problem associated with this conservative philosophy is that these vulnerable patients remain bedridden on log-roll precautions, which precludes from a coherent and proactive management of severe associated injuries to the head, chest, abdomen, and pelvis. In a landmark article, Croce and colleagues[52] performed a retrospective analysis of a prospective database on 291 consecutive patients with unstable spine fractures requiring surgical fixation. Patients were matched for injury severity and stratified by level of spine injury into two distinct cohorts, depending on the timing of fracture fixation: early fixation (within 3 days, n = 142) versus late fixation (>3 days, n = 149).[52] The authors found that the early fixation of thoracic spine fractures resulted in a lower incidence of pneumonia, fewer ventilator-dependent days, a shorter ICU stay, and reduced hospital charges.[52] This notion was confirmed by a systematic review of the published literature by Rutges et al,[53] who provided a comparison between different time points of surgical stabilization of thoracic or lumbar spine fractures. The authors concluded that early intervention for fracture stabilization in the thoracolumbar spine is safe, advantageous, and associated with a significantly decreased incidence of postoperative complications.[53]

However, due to the lack of unequivocal scientific evidence from prospective randomized trials, there remains a lack of consensus regarding the "optimal" timing of spine fracture fixation in multiply injured patients.[54] Intuitive advantages of early spine fixation relate to preventing complications associated with prolonged bed rest and the inability to adequately position and mobilize severely injured patients in the ICU. Unequivocally, multiply injured patients require unrestricted options for mobilization and positioning in the ICU, such as the upright seated position for treatment of head injuries and prone positioning for the management of acute respiratory distress syndrome. Finally, unstable and unreduced spinal fractures contribute to adverse sequelae of major trauma related to stress, pain, ongoing bleeding, and sustained systemic inflammation.[55] These concepts provide a strong argument for the necessity of early spine clearance and discontinuing bed rest and log-roll precautions in multiply injured patients. Current evidence suggests that any unstable thoracic or lumbar spine fracture or dislocation should be reduced and fixated within 24 hours of admission.[54,56-58] This approach is particularly applicable in the care of multiply injured patients at risk of sustaining second-hit insults and postinjury complications, including pressure sores, pulmonary infections, and thromboembolic complications.[59]

A proactive approach of spine damage control has been described and validated in the recent literature with the intent of mitigating the risk of adverse outcomes in polytrauma patients with associated unstable thoracic or lumbar spine fractures at risk of adverse outcomes.[60-64] The concept of spine damage control entails a staged procedure of immediate posterior fracture reduction and instrumentation within 24 hours (day 1 surgery), followed by scheduled 360° completion corpectomy and fusion during a physiologic time window of opportunity (>3 days after trauma).[51] Proceeding with the second stage is done if an adjunctive anterior decompression and fusion is indicated for neurologic or biomechanical reasons. This concept differs from the more common elective strategy of staged spine fixation by initial posterior fixation and delayed anterior completion in two ways: first, by its timeliness (posterior fixation within 24 hours), and second, by its expanded applicability to all unstable thoracolumbar fractures, including exclusive anterior column burst fractures.

The analogy of management strategies for femur shaft fractures and unstable thoracolumbar spine fractures in multiply injured patients is schematically depicted in Fig. 26-9.

In a prospective validation study of the spine damage control protocol, 112 consecutive patients with unstable thoracic or lumbar spine fractures and Injury Severity Score (ISS) of greater than 15 were prospectively enrolled during a 3-year time period.[65] Early spine damage control within 24 hours was performed in 42 patients, whereas 70 matched patients in the control group underwent definitive operative spine fixation at a delayed time point. The mean time to initial spine fixation was significantly decreased in the spine damage control group (8.9 ± 1.7 hours vs 98.7 ± 22.4 hours, $P < .01$). The early spine fixation cohort also showed a reduced length of operative time (2.4 ± 0.7 hours vs 3.9 ± 1.3 hours), length of hospital stay (14.1 ± 2.9 days vs 32.6 ± 7.8 days), and number of ventilator-dependent days (2.2 ± 1.5 days vs 9.1 ± 2.4 days) compared to the delayed spine fixation control group. Most importantly, the postinjury and postoperative complication rates were significantly decreased after spine damage control, including a reduced incidence of wound complications and surgical site infections (2.4% vs 7.1%), urinary tract infections (4.8% vs 21.4%), pulmonary complications (14.3% vs 25.7%),

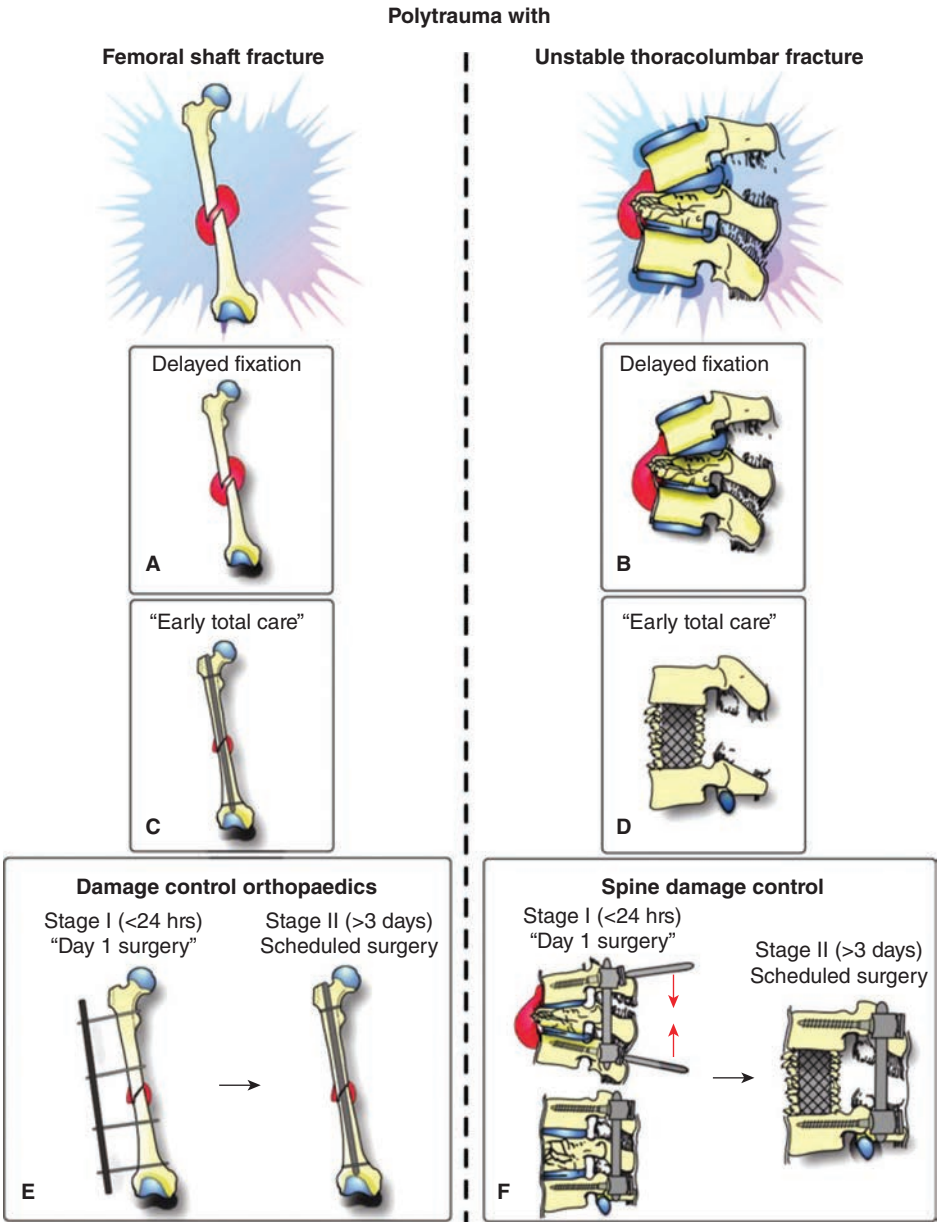


FIGURE 26-9 The analogy of management strategies for femur shaft fractures and unstable thoracolumbar spine fractures in multiply injured patients. (Reproduced with permission from Stahel PF, Flierl MA, Moore EE, et al. Advocating "spine damage control" as a safe and effective treatment modality for unstable thoracolumbar fractures in polytrauma patients: a hypothesis. *J Trauma Manag Outcomes*. 2009;3:6.)

and pressure sores (2.4% vs 8.6%). Our early experience implies that a proactive concept of early stabilization of unstable thoracic and lumbar fractures in multiply injured patients represents a safe and effective treatment strategy that should be considered for implementation in other trauma centers.[65]

SURGICAL CONSIDERATIONS

Surgery for unstable spinal injuries attempts to accomplish three main goals: (1) to decompress neurologic structures (spinal cord, spinal nerve roots, cauda equina, conus medullaris), as indicated; (2) to restore and maintain the sagittal alignment of the spine; and (3) to restore and maintain stability of the spine.

Cervical Injuries

The preferred treatment modality for unstable cervical spine fractures consists of an anterior decompression (including corpectomy) and fusion with anterior plating (ACDF) in conjunction with a bone graft substitute or cage. Unstable three-column fractures and fracture–dislocations with posterior facet dislocations may require a posterior approach

or combined posterior/anterior approach with 360° fusion. Posterior cervical spine fixation is typically accomplished by placement of multilevel lateral mass screws with or without adjunctive bone grafting for spinal fusion.

Thoracic and Lumbar Injuries

Unstable vertebral fractures with or without neurologic injuries are generally managed by anterior decompression with corpectomy and fusion using expandable cages and graft constructs, with or without adjunctive anterior column instrumentation with plates and screws or rods and screws. As described earlier, a standardized spine damage control approach may be considered in multiply injured patients by initial posterior fracture reduction, fixation, and decompression by laminectomy. This modality allows early mobilization and positioning of multiply injured patients as needed for intensive care. Definitive treatment consists of a delayed, staged anterior corpectomy and anterior column fusion (360°), if indicated. Chance fractures of the B2 type (Fig. 26-3) are managed exclusively by a posterior approach with definitive internal fixation using pedicle screws, with instrumentation two levels above and two levels below the fracture site. Three-column injuries frequently require a combined posterior/anterior 360° fusion, as outlined earlier. Of note, the placement of surgical drains is rarely indicated in the acute surgical management of spinal injuries. Exceptions where drains are beneficial include placement of retroperitoneal drains after extensive anterior approaches and chest tubes for transthoracic approaches. In contrast, the prophylactic placement of drains around the cervical spine for anterior or posterior approaches and for posterior thoracic or lumbar approaches does not appear to convey any benefit. While the perceived benefits include the theoretical decreased risk of postoperative hematoma formation, this must be weighed against the risk that keeping drains increases the risk of surgical site infections. Furthermore, side effects from prolonged antibiotic therapy can occur if the surgeon requests antibiotics be continued while surgical drains remain in place. Thus, on balance, the use of drains does not appear to be justified from a quality of care or patient safety perspective.

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Trauma Thoracotomy: Principles and Techniques

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KEY POINTS

- Despite frequent emphasis on devastating injuries, the most common thoracic injury is a chest wall contusion or rib fracture.
- History, physical examination, and chest x-ray remain fundamental to the diagnosis of thoracic injury. Additional imaging, including extended focused ultrasonography assessment for trauma, computed tomography (CT) scanning, and CT angiography are frequently used.
- A thoracostomy tube output of 1500 mL of blood on insertion or 200 mL of blood per hour usually warrants an urgent thoracotomy.
- A left anterolateral thoracotomy through the third or fourth interspace allows exposure for opening the pericardium, open cardiac massage, clamping of the descending thoracic aorta, and treatment of a large percentage of cardiac and left lung injuries.
- A retained hemothorax should be evacuated by video-assisted thoracoscopy or thoracotomy as soon as diagnosed.
- New technologies, such as retrograde endovascular balloon occlusion of the aorta and extracorporeal cardiopulmonary resuscitation, have the potential to further change the philosophy of prehospital management for thoracic injuries.

INTRODUCTION

Thoracic injury occurs in 20% to 25% of trauma patients, resulting in 16,000 deaths annually in the United States.[1,2] Because the chest accounts for 25% of the total body mass, it is susceptible to injury from virtually any etiology or mechanism. Injury to the chest and its organs may be caused by penetration (missiles, fragments, knives, needles, and other objects), blunt forces, sudden deceleration, iatrogenic misadventure, blasts, and ingestion of toxic substances. Each of these etiologies has differing initial manifestations as well as evaluation and treatment approaches.[3-5] These differences are more specifically discussed in other chapters of this textbook.

Despite frequent emphasis on devastating injuries, such as cardiac or great vessel injury, the most common thoracic injury is a chest wall contusion or rib fracture.[2] Injuries to the heart or lungs frequently present with obvious signs that require prompt intervention. Some injuries, however, are less apparent and present with delayed manifestations requiring ongoing clinical suspicion and management. Consequently, patients with thoracic trauma require logical and sequential evaluation, followed by focused therapy, which involves an operation less than 20% of the time. Notably, the most common operation in a patient with a chest injury is an exploratory laparotomy, with formal thoracotomy a rare intervention. The objective of this chapter is to review the diagnostic process and judgment decisions leading to performing a thoracotomy in the trauma patient.

INITIAL EVALUATION

Initial evaluation follows the standard Advanced Trauma Life Support (ATLS) protocols. Physical examination should carefully evaluate for presence or absence of breath sounds or diminished breath sounds, presence of chest wall contusions, penetrating injuries, flail chest segments, and evidence of prior thoracic surgery (ie, sternotomy or thoracotomy incision scars). Upper and lower extremity pulse examination is essential and may be a marker of injury to the thoracic aorta or subclavian artery. Finally, the presence of cardiac murmurs or diminished heart sounds may be indicative of a traumatic cardiac injury.

The chest x-ray is a fundamental diagnostic tool in evaluating chest trauma. It is used to determine the position of the trachea and evaluate the lung parenchyma, mediastinal silhouette, aortic knob, and pleural cavities. In addition, it can

diagnose a hemopneumothorax, pneumomediastinum, rib fractures, spinal or other skeletal fractures, and foreign bodies. Ideally, the chest x-ray is obtained in an upright position to minimize mediastinal widening that accompanies supine positioning and to accentuate air-fluid levels.[6] An electrocardiogram (ECG) should be performed on all trauma patients, especially those with blunt thoracic injury.[7] The presence of an arrhythmia, frequent premature ventricular contractions, bundle branch blocks, or ST-segment depressions may be indicative of a blunt cardiac injury.

Focused Ultrasonography Assessment for Trauma (FAST) and Extended FAST

Focused ultrasonography assessment for trauma (FAST) allows for a rapid bedside evaluation of the pericardial and abdominal cavities that can be performed by trauma surgeons and emergency department physicians. FAST allows for identification of fluid or blood in the pericardial space via a subxiphoid view. Although operator dependent, focused echocardiography can be used to confirm tamponade physiology, cardiac filling status, and cardiac function. The presence of hemopericardium, however, is exceedingly rare in blunt trauma and often diagnosed from other acute clinical signs.[8] FAST has been extended (so-called E-FAST) to evaluate the thoracic cavity for the presence of a traumatic hemothorax and pneumothorax. In one recent large series including 756 patients, the sensitivity of E-FAST for identifying pneumothorax (69% vs 37%) and hemothorax (48% vs 29%) was favorable compared to chest x-ray.[9] As with FAST, however, a learning curve and operator expertise must be developed, as other studies have shown poor sensitivity (43%) when performed by some practitioners.[10] The decision to pursue E-FAST in lieu of a chest-x-ray should be approached with caution, taking into account the expertise of the operators at each trauma center.

Additional Diagnostic Maneuvers

A variety of diagnostic studies may be required to delineate intrathoracic injury. Additional imaging includes computed tomography (CT), CT angiography, angiography, cardiac catheterization, and echocardiogram, as mentioned earlier. Endoscopic evaluation of the trachea and esophagus, as well as contrast esophagrams, may be required. In deciding which diagnostic evaluation to perform, the first consideration is what the test is expected to demonstrate, whereas the second consideration is how the results will alter decision making or treatment. Frequently, additional tests, when needed, are initiated based on findings from the physical examination, chest x-ray, and/or FAST. Although CT scanning has become ubiquitous in the evaluation of trauma patients, it may be overused and result in a delay of necessary surgical therapy and in excessive radiation exposure.[11-13] In general, especially for suspected vascular injury, CT scanner detectors with greater than 64 slices are recommended. Older scanners with fewer slices produce inconsistent results and often create unnecessary confusion. Using these newer CT scanners with multidetectors, ECG gating, and multiplanar reconstruction, CT aortography (CTA) for aortic injury can have a sensitivity of 98% and specificity of 100%. In addition, it can provide valuable insight in surgical planning, particularly if endovascular treatment is planned.[14] Although CTA has largely supplanted angiography in clinical practice, angiography remains valuable in the diagnostic and potential therapeutic management of thoracic trauma, especially at centers without access to ECG-gated CT scanning. With the advent of hybrid operating rooms, intraoperative angiography may lead to more rapid diagnosis and treatment of thoracic vascular injuries in polytrauma patients.[15]

TUBE THORACOSTOMY

Tube thoracostomy is the most common therapeutic procedure performed following thoracic trauma, and it is also one of the most misunderstood and underrated procedures in medicine. Of patients with thoracic injuries who do need surgical intervention, tube thoracostomy is the only invasive procedure that 85% will require. Unfortunately, more than 25% of patients with thoracostomy tubes will encounter some difficulty with malposition, drainage problems, and various complications. One of the most common problems encountered is incomplete evacuation of a hemothorax, resulting in a clotted hemothorax or an empyema. Second and multiple thoracostomy tubes are then frequently inserted as a result of misunderstanding the function of the chest tube and/or improper technique for insertion.

Tube thoracostomy following trauma should be accomplished with adequate anesthesia and analgesia. Trocar-tipped chest tubes should be avoided. Chest tubes are best inserted in the area of the auscultatory triangle in the mid-axillary line in the fourth or fifth intercostal space. Using clamps or dissecting scissors, subcutaneous tissue and muscular dissection is performed to create a track directed posteriorly. The pleura is anesthetized with local anesthetic injection, and the pleural cavity is entered with an exploring finger and not a sharp instrument. Care is taken to avoid injury to the intercostal vessels and nerve on the undersurface of each rib, as this can lead to iatrogenic bleeding and significant pain. Following a gentle, digital exploratory thoracotomy, an appropriately sized thoracostomy tube (32–36F) is directed toward the back and apex of the pleural space and attached to an appropriate collection device. Because this insertion site overlies the major pulmonary fissure, care must be taken to ensure that the tube is not placed in this fissure. On occasion, the location can be determined by preinsertion digital exploration. Autotransfusion of fresh blood from a hemothorax may be considered if preprocedural chest x-ray demonstrates a complete whiteout suggestive of a massive hemothorax—800 to 1500 mL of volume can be recycled this way. Up to 25% of the population has some element of visceral and parietal pleural symphysis, and this can contribute to subcutaneous emphysema in the absence of a pneumothorax. In such

TABLE 27-1: Acute Indications for Thoracotomy

Acute hemodynamic deterioration and cardiac arrest in the trauma center
Penetrating truncal trauma and need for proximal vascular control
Cardiac tamponade
Ultrasound demonstration of hemopericardium
Vascular injury at the thoracic outlet
Massive air leak from chest tube
Endoscopic or radiographic demonstration of tracheal or bronchial injury
Endoscopic or radiographic evidence of esophageal injury
Radiographic evidence of great vessel injury
Missile embolism to heart or pulmonary artery
Traumatic thoracotomy (loss of chest wall substance)

instances, the thoracostomy tube may be inserted into the substance of the lung, rather than the pleural space. Should a pneumothorax or hemothorax actually exist, care must be taken to insert the chest tube into the space containing the blood or air, rather than at a point of pleural symphysis.

INDICATIONS FOR THORACOTOMY FOLLOWING TRAUMA

Approximately 15% of patients with thoracic trauma will eventually require a formal thoracotomy. The classic indications for emergency department or operating thoracotomy after trauma are listed in Table 27-1.[16-25]

TIMING OF THORACOTOMY

Timing of an acute thoracotomy is a function of the immediacy of the life-threatening condition.[26,27] Potentially life-threatening conditions include acute pericardial tamponade, acute and massive blood loss, disruption of ventilation, and decreased cardiac output. Infection, sepsis, pulmonary insufficiency, and other functional impairments may occur secondarily, and any or all contribute to the timing and decision to operate.

Immediate and Emergent

For an acute injury to the heart and immediate loss of cardiac output from reversible conditions such as pericardial tamponade or cardiac perforation or rupture, immediate thoracotomy is indicated. Intubated patients with posttraumatic prehospital external cardiac massage for more than 5 to 10 minutes are unlikely to be resuscitated in the hospital, even with emergency department (ED) thoracotomy (see Chapter 17). Most trauma surgeons and trauma societies agree, however, that resuscitative thoracotomy by qualified personnel is indicated for patients with penetrating thoracic trauma who have less than 15 minutes of prehospital cardiopulmonary resuscitation (CPR).[28,29] ED thoracotomy for patients with blunt injury, especially those with greater than 10 minutes of prehospital CPR and without signs of life upon arrival, has had very limited success. One recent study from the National Trauma Data Bank revealed a 100% mortality rate in 2519 patients over the age of 57 years who underwent ED thoracotomy from 2008 to 2012 for penetrating or blunt trauma.[30] When the team determines a need for an ED thoracotomy, however, such procedures can be individualized and tracked by the hospital's trauma quality review process.

Urgent

An urgent or operating room thoracotomy is performed within 6 hours after injury to control and manage a newly developed life-threatening condition or prevent the development of further deterioration, injury, or infection.

Delayed

In a patient with multisystem trauma, delayed repair of an injured hemidiaphragm or stable injury of the thoracic aorta is appropriate to allow time for stabilization or treatment of a severe injury to the brain and intra-abdominal organs or pelvis.

PATIENT POSITIONS/INCISIONS (SEE ATLAS FIGURE 27)

Most patients undergoing an emergency thoracotomy are positioned supine. This allows for a median sternotomy, right and/or left anterolateral thoracotomy, trans-sternal bilateral anterolateral thoracotomy (clamshell for a wound to the right side of the heart), and partial anterior incisions (see Atlas Figure 28). Furthermore, this positioning allows for concomitant endovascular intervention, laparotomy, and access to the extremities. As previously noted, a left anterolateral thoracotomy through the third or fourth interspace allows exposure for opening the pericardium, open cardiac massage, clamping of the descending thoracic aorta, and treatment of a large percentage of injuries to the heart and left lung. Pitfalls include making the incision too small, injury to the intercostal arteries, and injury to the esophagus or aorta during aortic cross-clamping (see Atlas Figure 35).

For an urgent or delayed thoracotomy, a more targeted thoracotomy may be considered. Approaches to the posterior mediastinum and, at times, the hilum of the lung are via either a right or left posterolateral thoracotomy through the fifth intercostal space. This position and these incisions are best suited for injury to the descending thoracic aorta, esophagus, azygous vein, and the mediastinal trachea and bronchi. If, for whatever reason, the initial approach was via an anterior incision but a predominately posterior injury is found, the anterior incision should be closed. The patient is then reopened in a lateral decubitus position and posterolateral

incision that optimizes exposure and management of the injury, provided that patient is hemodynamically stable. In a hemodynamically unstable patient who is already in a supine position, extension to a clamshell incision will still allow for limited access to the previously mentioned structures.

In the past, one indication for a thoracotomy was the presence of a thoracoabdominal injury, in which a laparotomy incision was extended across the costal margin. In the modern era, thoracoabdominal incisions are rarely used. It is more appropriate to approach injuries in multiple cavities by entering the cavity with the most apparent life-threatening injury first.

THORACIC DAMAGE CONTROL

Damage control operations in the abdomen were among the most important advances in trauma management during the 1990s. These techniques have been applied subsequently to thoracic trauma, although packing an area inside the chest does not have the same damage control utility as when used in the abdomen.[26] Thoracic damage control techniques are, philosophically, a simple approach to a complex problem. Damage control tactics for the patient with thoracic trauma are cited in other chapters of this book and include the following:

1. ED thoracotomy and resuscitation[18-21,23]
2. Pulmonary tractotomy[31]
3. Pulmonary hilar twist[32]
4. Temporary damage control thoracic closure[26]
5. Endovascular treatment of injury to thoracic aortic or great vessel[33]

COMPLICATIONS OF THORACIC TRAUMA

Many of the complications after thoracic trauma will be discussed in the organ-specific injury chapters. For completeness, a few of these complications are briefly discussed here.

Retained Hemothorax and/or Empyema

When a clotted hemothorax persists, video-assisted thoracoscopic surgery or thoracotomy should be used to evacuate it as soon as possible, because this reduces the incidence of posttraumatic empyema.

Fluid Overload and Adult Respiratory Distress Syndrome

Recognized for decades but specifically codified during the Vietnam War, fluid overload and the resultant adult respiratory distress syndrome (ARDS) have become better understood over the past 20 years. The fact that various crystalloids activate inflammatory mediators, as well as contribute to ARDS, is now well described. In addition, it is well recognized that the contused lung is more prone to barotrauma, pneumonia, and fluid overload than the uninjured lung.

Barotrauma

Although higher positive end-expiratory pressure was used for patients with posttraumatic respiratory insufficiency during the 1970s, it is now recognized that high inspiratory pressures and other ventilator forces cause volutrauma/barotrauma to both the bronchial lining and the interstitium of the lung.

Systemic Air Embolism

Vascular air embolism can affect venous return to the heart and the right cardiac circulation. Trauma with injuries to bronchi or bronchioles and pulmonary vein branches can also cause pulmonary venous air embolism, which becomes systemic air embolism. Both are most often iatrogenic, with systemic air embolism being secondary to a pulmonary injury under conditions of increased endobronchial pressures greater than 40 torr. Air embolizing into pulmonary venules moves to the left atrium, producing systemic air embolism, seizures, and ventricular fibrillation secondary to air in the coronary (mostly right coronary given its anterior location when patient lays supine) and cerebral arteries. When it does occur, it is almost always fatal.

Aspiration

Resuscitative efforts in injured patients may contribute to aspiration and some of its more undesirable effects. The insertion of a nasogastric or feeding tube into the pharynx of a fully awake patient with a stomach full of food is conducive to aspiration of vomited gastric contents. Aspiration of water-soluble contrast material (eg, diatrizoate meglumine) into the lungs produces a chemical pneumonitis much more severe than that produced by aspiration of a barium-based contrast material. The insertion of a feeding tube into the bronchus, lung substance, or even the pleural cavity and then subsequent introduction of enteral feedings obviously produces devastating results. Aspiration is best treated by immediate bronchoscopy and lavage to remove solid particles.

Radiation Exposure

Overutilization of chest x-rays, CT scanning, and injections of vascular contrast material has resulted in significant doses of radiation to providers. Controversy persists on the short- and long-term radiologic damage from the many radiologic studies, often unnecessary and duplicative, during the initial evaluation of a trauma patient. Unfortunately, quality review of medical records rarely reveals a preimaging progress note indicating why the test was ordered, what the clinician wanted to learn from the study, or how results might alter decision making. Such progress notes undoubtedly would be beneficial in defending excessive radiation that might be associated with development of lymphoma or leukemia many years later.[11-13]

CONTROVERSIES IN THORACIC TRAUMA

In evaluating and treating patients with thoracic trauma, it is important to recognize that some of the historic approaches are both controversial and lacking in scientific evidence, despite wide popular use.

Resuscitative Endovascular Balloon Occlusion of the Aorta

Resuscitative endovascular balloon occlusion of the aorta (REBOA) is an emerging tool for management of noncompressible torso hemorrhage in patients with traumatic injury below the diaphragm.[34] REBOA is less invasive than resuscitative thoracotomy, may be performed rapidly, and may be performed by a wider variety of trained trauma physicians. Although high-grade evidence is limited for this newer technique, REBOA in skilled hands of trained personnel was shown to offer survival benefit as compared to resuscitative thoracotomy in one recent study of a REBOA registry.[35] The use of REBOA in patients with penetrating thoracic trauma is highly controversial, but some centers are expanding REBOA in this patient population.[36] As with all emerging technologies, high-grade data are limited and deployment should be approached cautiously by appropriately trained personnel, along with rigorous hospital quality review.

Prehospital Resuscitative Thoracotomy

Prehospital resuscitative thoracotomy for patients with penetrating thoracic trauma in extremis can be lifesaving.[37] In some countries, emergency medical services (EMS) have trained personnel to perform resuscitative thoracotomy in patients with penetrating and blunt trauma. In a 15-year database review of the London EMS system, 18% of 71 patients who underwent prehospital thoracotomy survived to discharge.[38] Because many EMS systems do not integrate physicians, such as in the United States, performance of this procedure requires significant resources and training. In addition, the benefit of in-the-field treatment versus rapid transfer to a trauma center must be weighed. New technologies such as REBOA and extracorporeal CPR have the potential to further change the philosophy of prehospital management.[39]

Pericardiocentesis

Resuscitation courses have historically recommended the technique of pericardiocentesis to relieve hemopericardium and cardiac tamponade following cardiac injury. However, trauma surgeons have routinely described clotted blood between the pericardium and heart at emergency thoracotomy for hemopericardium for over a century, and an acutely clotted hemopericardium is not amenable to acute removal via pericardiocentesis. Additionally, surgeons often describe iatrogenic cardiac perforation following an attempted pericardiocentesis for acute trauma. Therefore, pericardiocentesis for acute hemopericardium after trauma is rarely indicated, and emergency thoracotomy, pericardiotomy, and cardiorrhaphy are indicated.

Subxiphoid Pericardiotomy

A subxiphoid pericardiotomy, often performed in the operating room (and sometimes performed in the emergency department) to detect hemopericardium, was introduced as a technique prior to the wide adoption of the FAST examination. The small upper abdominal incision allows for direct drainage of hemopericardium but does not allow for focused cardiorrhaphy. With more precise diagnostic techniques for pericardial fluid, a directed thoracic incision should be used to relieve pericardial tamponade and treat any cardiac injury.

Needle Decompression of Pleural Cavity

Historically, a tension pneumothorax following thoracic trauma was believed to account for significant numbers of deaths in the prehospital and emergency department phases. Insertion of a "decompressing" needle into the pleural cavity has been recommended in many resuscitation courses, despite controlled studies to demonstrate the exact frequency of tension pneumothorax or the specific benefit or utility of needle decompression. Furthermore, tension pneumothorax is undoubtedly more difficult to determine than has been presumed, particularly in a moving ambulance. In agonal or arrested trauma patients, blind thoracentesis is acceptable; unfortunately, actual entrance of available needles into the pleural cavity occurs less than 5% to 10% of the time.

Pledgets in Cardiorrhaphy

Cardiorrhaphy is routinely accomplished during cardiac surgery without the use of adjunctive pledgets in the suture line. Although often used during posttraumatic cardiorrhaphy, this practice is not supported by experience and introduces an unnecessary added step for the surgeon and operating room nurse. Pledgets are only recommended on the rare occasion of significantly bruised myocardium that will not hold sutures or when pressors have created significant intraoperative hypertension during cardiac repair. In elderly patients or those with frail tissue, a tissue reinforcement or patch repair with autologous tissue, such as pericardium, may be beneficial.

Trapdoor Thoracotomy

This combined anterolateral thoracotomy, partial sternotomy, and supraclavicular ("trapdoor" or "book") incision that was popular in the 1970s for injuries to the left thoracic outlet offers little exposure advantage. In addition, it can cause causalgia in the left upper extremity secondary to stretching of the cervical nerve roots. In the current endovascular era, proximal vascular control of the left subclavian artery can be obtained with an intravascular balloon, followed by either endovascular or open repair via a supraclavicular incision.

CONCLUSIONS

1. Improvements in imaging for patients with thoracic trauma have included the E-FAST examination, contrast CT, and CT angiography.
2. The indications for and techniques of thoracotomy have not changed significantly over the past three decades.
3. Early video-assisted thoracoscopy has replaced delayed thoracotomy for the evacuation of a retained hemothorax and has significantly lowered the incidence of posttraumatic empyema.
4. The role of REBOA and extracorporeal CPR in patients with thoracic injuries remains to be defined.

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28

Chest Wall and Lung

Jamie J. Coleman • Fredric M. Pieracci • Joseph J. DuBose • Thomas M. Scalea • James V. O'Connor

KEY POINTS

- Only 7% (blunt trauma) to 15% (penetrating trauma) of patients with thoracic trauma will require a thoracotomy.
- The ribs are flexible, the sharpest angle of the rib occurs posteriorly at the tubercle, and the ribs are furthest apart from one another anteriorly.
- Currently available classification systems for rib fractures include the Organ Injury Scale–Chest, Rib Fracture Score, Chest Trauma Score, and RibScore.
- Both acetaminophen and nonsteroidal anti-inflammatory drugs have been shown to provide comparable analgesia to oral narcotics in trauma patients and should be given routinely to patients with injury to the chest wall.
- Surgical stabilization of rib fractures is currently recommended for patients with a flail segment and a poor response to nonoperative therapy or associated respiratory failure, patients with multiple displaced rib fractures, and patients with multiple nondisplaced fractures that undergo interval displacement and worsening clinical status.
- Prolonged (ie, 7–10 days) trials of nonoperative management in patients with multiple displaced rib fractures increase the risk of pulmonary compromise.
- A large pneumothorax seen on computed tomography, but not detected on a chest x-ray, is most often anterior.
- Indications for a thoracotomy for a hemothorax include initial chest tube drainage (15–30 minutes) of 1200 to 1500 mL or 200 mL/h for 2 to 4 hours.
- Significant injuries to the lung, particularly those from gunshot wounds and away from the hilum, are often best treated with pulmonotomy and selective vascular ligation.
- A "persistent" air leak after 5 to 7 days of drainage of the pleural cavity can be treated with autologous blood pleurodesis, a commercially available sealant, an endobronchial one-way valve, a Heimlich valve, or a thoracotomy.

INTRODUCTION

Injuries to the chest wall and lung are common following both blunt and penetrating trauma. Blunt thoracic injuries are responsible for approximately 8% of all trauma admissions and contribute to 25% of trauma deaths in the United States, with motor vehicle crashes as the most common mechanism.[1-4] The most common injury following blunt chest trauma is rib fractures.[5] In comparison, penetrating chest trauma accounts for 7% of all trauma admissions and 16% of penetrating trauma admissions overall.[6]

The first task in managing patients with injuries to the chest wall and lung is determining the need for emergency intervention. Conditions typically needing emergency intervention are related to either bleeding or a pneumothorax. The majority of patients continue to be successfully managed nonoperatively or with minor procedures. Between 18% and 40% of patients sustaining thoracic trauma can be effectively managed with tube thoracostomy alone, and thoracotomy will be required for only 7% to 15% of patients. Even among those with penetrating chest trauma, only 14% of patients with stab wounds and 20% of patients with gunshot wounds to the chest require emergent thoracotomy.[6]

When emergent operation is required, approximately 30% of patients undergoing thoracotomy require a pulmonary resection. Mortality varies between 5% and 45%.[3] This wide variability is related to differences in mechanism of injury, the inclusion of cardiac and major thoracic vascular injury in some of the datasets, the extent of pulmonary resection performed, and the contribution of concomitant extrathoracic injuries to outcome.[3,7-9]

Although the likelihood of emergency intervention following trauma to the chest wall and lung is relatively low, prompt recognition is imperative to achieve optimal outcomes. In an

era of nonoperative management for many injuries, clinicians caring for trauma patients must appreciate the indications for operation and understand the treatment options in the emergency department as well as in the operating room. Sophisticated judgment is essential when evaluating these injuries in the context of a trauma victim with multiple injuries.

When emergent interventions are not necessary, management is focused on supportive care, specifically in the form of both multimodal analgesia and pulmonary toilet. Fractures of both the ribs and sternum are unique because their involvement in respiration renders them unable to be effectively immobilized following fracture in any way other than surgically. Moreover, painful motion at sternal and rib fracture sites impairs respiration, increasing the risk of splinting, accumulation of secretions, pneumonia, and respiratory failure. Beyond these acute complications, patients with severe chest wall injuries are at high risk of chronic pain, dyspnea, and narcotic dependence.[10]

The traditional treatment of both rib and sternal fractures has been limited to pain control with narcotics and internal pneumatic stabilization with mechanical ventilation in selected patients.[11] The shortcomings of these strategies have been well documented, and contemporary management of chest wall injuries now ideally involves a multidisciplinary, protocolized approach, with selective use of locoregional anesthesia, nonnarcotic oral and intravenous adjuncts, and surgical stabilization.

The field of chest wall injury, and surgical stabilization of rib fractures (SSRF) specifically, has received much interest over the past two decades. At the turn of the century, most trauma surgeons did not recommend SSRF for any rib fracture pattern, nor had they ever been involved in either a rib or sternal repair.[12] More recent data suggest that approximately 7% of patients with three or more rib fractures undergo SSRF[13] and that the incidence of this operation is increasing exponentially.[14] This increase has been accompanied by, and is partially due to, the introduction of several rib-specific fixation systems, as well as SSRF-specific current procedural terminology codes. Finally, multidisciplinary surgical societies devoted specifically to the treatment of chest wall injuries have emerged.[15]

Although the increased interest in chest wall injuries is ultimately beneficial to patients, the trauma community needs to objectively delineate which patients derive the most benefit from each therapy. The vast majority of injuries to the chest wall and lung do not require surgery, underscoring the continuing need for expertise in nonoperative management.

INJURY TO THE CHEST WALL

Anatomy and Taxonomy

The ribs are 12 paired, curved bones that articulate with both the transverse and costal facets of the thoracic vertebral bodies posteriorly. Anteriorly, the first six ribs (true ribs) articulate directly with the sternum via independent costal cartilages, ribs 6 through 10 (false ribs) fuse costal cartilages to articulate with the lower sternum, and ribs 11 and 12 (floating ribs) do not articulate with the sternum. The ribs are bicortical, cancellous bone with a medullary canal, and the thickness of a human rib typically measures 6 to 12 mm.

As opposed to other cancellous bone, ribs possess several unique properties that must be taken into consideration when selecting implants to stabilize fractures, as well as exposing rib fractures surgically.[16] The rib surface is twisted and conical, the extent of which varies from rib to rib. The sharpest angle of the rib occurs posteriorly at the tubercle, and the ribs are furthest apart from one another anteriorly. The ribs are uniquely flexible, a property that is essential to their role in chest wall expansion and retraction during respiration. Rib flexibility is maximal during childhood, and rib fractures in children signify a large amount of force transferred during injury. The ribs are also subject to osteoporosis and fracture most easily in elderly patients. Finally, many chest wall muscles, including the intercostal, scalene, subclavius, and serratus, and the abdominal muscle, the rectus abdominus, either originate from or insert onto the ribs.

The sternum consists of the upper manubrium, manubriosternal joint (a secondary cartilaginous as opposed to a synovial joint), body, and xiphoid process. In addition to the ribs, the sternum articulates with the clavicle at the sternoclavicular joint, an atypical synovial joint. Due to the unique properties of both the manubriosternal and sternoclavicular joints (as opposed to true synovial joints), dislocations have been treated successfully via reduction and fixation.[17,18]

Finally, the scapula represents an oblong bone that articulates with both the clavicle at the acromioclavicular joint and the humerus at the glenohumeral joint. The thin scapular body covers, and is intimately associated with, the first seven ribs posteriorly. The migration of the scapula from lateral to posterior throughout evolution is believed to reflect the importance of the use of the upper limb to wield a spear for hunting.[19] Displaced upper, posterior rib fractures, particularly in conjunction with a scapular body fracture, may cause painful grinding that substantially impairs shoulder mobility (Fig. 28-1).

Organization of the complexity of rib fractures into a unified taxonomy, as has been developed for most other organs, has only recently been undertaken. Most chest wall scoring systems are limited to the number of ribs fractured and the presence of bilaterality. Recently, the Chest Wall Injury Society conducted a Delphi consensus exercise that addressed multiple rib taxonomic issues, including fracture location, degree of displacement, associated fractures in neighboring ribs, and the definition of flail chest.[20] Displacement was divided into three categories based on the degree visualized on axial computed tomography (CT) imaging (Table 28-1). Furthermore, three anatomic sectors were selected to describe the location of rib fractures including anterior, lateral, and posterior. Fractures themselves were further categorized as simple, wedge, or complex. Finally, fractures on consecutive ribs were termed a series of fractures. Although future validation is warranted, this framework represents a starting point to assist in communication among providers regarding chest

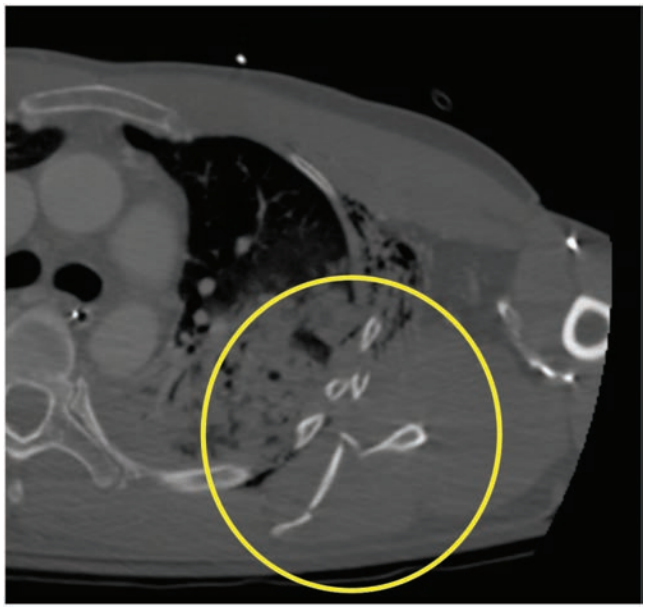
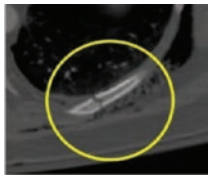
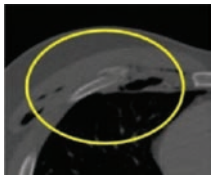
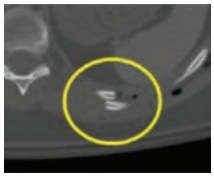


FIGURE 28-1 Displaced, subscapular rib fractures in conjunction with a comminuted scapular body fracture. This injury pattern is particularly morbid with respect to shoulder mobility.

wall injuries, as well as select patients for both invasive therapies and research.

The term *flail chest* was used originally to describe paradoxical motion of a portion of the chest wall with respiration due to multiple, consecutive ribs fractured in more than one location.[21] Respiratory compromise was due to the flail segment moving in opposition to the remainder of the chest wall. When diagnosed clinically, flail chest has had a particularly high mortality historically.[22] More recently, the diagnosis of flail chest has evolved into one that is used most commonly based upon a CT finding of two or more consecutive ribs fractured in two or more places. This ambiguity in definition has hindered research into both the prognosis and management of flail chest, as patients grouped together as having flail chest may in reality range from having clinically insignificant to devastating fractures. To address this dilemma, Edwards et al[20] suggested use of the term *flail segment* to describe the radiologic findings of three or more ribs fractured in two or more places, and the term *flail chest* to describe the paradoxical motion seen on clinical examination.

TABLE 28-1: Standardized Definitions of Rib Fracture Displacement

Category	Definition	Example
Nondisplaced	Both cortices are fractured, and the two fracture fragments are displaced by 0%–9% of the width of the rib	
Offset	Both cortices are fractured, and the two fracture fragments are displaced by 10%–99% of the width of the rib	
Displaced	Both cortices are fractured, and the two fracture fragments are displaced by ≥100% of the width of the rib	

Source: Data from Edwards JG, Clarke PB, Pieracci FM, et al. Taxonomy of multiple rib fractures: results of the Chest Wall Injury Society international consensus survey. *J Trauma Acute Care Surg.* doi: 10.1097/TA.0000000000002282. [Epub ahead of print April 2, 2019].

Initial Evaluation and Stratification of Risk

The initial evaluation of the patient with a suspected injury to the chest wall involves the primary and secondary surveys. Findings on physical examination concerning for rib fractures include tenderness over the chest wall, crepitus, bruising, and deformity, typically with a depressed segment of the chest wall. Rib and sternal fractures are commonly associated with pulmonary and cardiac contusions, respectively, as well as pneumothorax and hemothorax.

The initial imaging modality in patients with suspected chest wall injury is the chest x-ray. Chest x-ray is useful in terms of ruling out life-threatening injuries that require immediate attention in the trauma bay, such as a pneumothorax or hemothorax. Unfortunately, the sensitivity of chest x-ray for detecting both rib and sternal fractures is low when compared to CT.[23] Multiple studies have documented that CT identifies significantly more rib fractures, as well as both pleural and mediastinal pathology, as compared to a chest x-ray[24-26]; however, the clinical significance of these injuries remains uncertain. Many of these studies were performed prior to widespread use of both locoregional anesthesia and SSRF. Chapman et al[27] found that over half of patients with rib fractures had at least three additional rib fractures discovered on CT, as compared to a chest x-ray. Furthermore, these patients were more likely to undergo admission to an intensive care unit, locoregional anesthesia, or SSRF. Currently, it seems reasonable to perform CT of the chest in patients with three or more rib fractures visualized on chest x-ray after blunt trauma, those who require a CT chest for another reason, or those with physical exam findings discordant with the chest x-ray. Three-dimensional reconstructions of the CT chest are now routinely available; however, their routine use has not been shown to alter decision making or improve outcomes.

Even when radiologic data are made available, risk stratification of patients with rib fractures by clinicians is highly variable, as previously noted.[28] Several injury scoring systems for the chest wall have been developed in order to assist in clinical decision making. Challenges in the development of a scoring system for chest wall injury involve capture of the

TABLE 28-2: Comparison of Various Chest Wall Injury Scoring Systems

	OIS Chest	RFS	CTS	RibScore
Rib fracture variables				
No. of ribs fractured	•	•	•	•
Flail chest	•			•
Bilateral fractures	•	•	•	•
Degree of displacement				•
Fracture location				•
First rib fractured				•
Non-rib fracture variables				
Age	•	•	•	
Pulmonary contusion	•		•	
Clavicle/scapula/sternal fractures	•			

CTS, Chest Trauma Score; OIS, Organ Injury Scale; RFS, Rib Fracture Score.

large number of injury permutations, as well as the patient's pulmonary physiologic response to them, in a single, reproducible score. Scoring of patients with rib fracture at the most basic level involves summing the number of ribs fractured. A direct relationship has been observed by multiple authors between the total number of ribs fractured and adverse pulmonary outcomes, with a specific number of six ribs fractured suggested to serve as a threshold above which mortality is significantly increased.[29-32] More recently, the degree of displacement of rib fracture has also been correlated with adverse outcomes.[33]

A number of scoring systems for chest wall injury, summarized in Table 28-2, have been published, each incorporating a unique set of demographic, physiologic, and radiographic parameters.[34-39] Of these scores, the RibScore[38] includes the most detailed information on rib fracture pattern and has been validated most extensively.[40] In practice, these static scores are limited by subjectivity in data abstraction, as well as failure to incorporate the patient's physiologic response to injuries. Because of these limitations, their utility in guiding invasive therapies is relatively limited. These scores do, however, serve as a common language for communication among providers as to the degree of anatomic injury.

Dynamic assessments of a patient's physiologic response to a chest wall injury are required to guide invasive therapies, as well as address the common observation that two patients with similar demographic and radiographic findings may demonstrate vastly different phenotypic responses to injury. One commonly used dynamic measurement is the pulmonary vital capacity, which is relatively easy to perform at the patient's bedside and has been found to be associated with pulmonary complications.[41,42] Recently, the vital capacity, in addition to the numeric pain score, cough quality, and respiratory rare, were combined to create a Sequential Clinical Assessment of Respiratory Function (SCARF) score, a dynamic score that, when measured daily, correlates with adverse pulmonary outcomes.[43] Additional dynamic, pulmonary physiologic scores have since been reported and represent an important next step in real-time risk stratification of patients with injuries to the chest wall.[44]

Regardless of the scoring system used, an objective, reproducible metric should be employed consistently when assessing the patient with chest wall injury. Furthermore, it is recommended that trauma centers caring for such patients protocolize management. One such admission protocol is shown in Fig. 28-2. In general, admission algorithms have been shown to minimize variability in practice, decrease length of stay in the emergency department, and improve outcomes of patients with chest wall injuries.[39,45] Protocol details will be dictated by both the institution's patient population and available resources. One common aspect, however, should be that the protocol is reevaluated at regular intervals and modified to include contemporary evidence.

Nonoperative Management

The cornerstones of nonoperative management of patients with chest wall injury are analgesia and pulmonary toilet. Pain may be secondary to irritation of the pleura and pericardium with blood; fractures of the ribs, sternum, clavicle, and scapula; contusion of the chest wall muscles; or cutaneous lacerations, burns, and road rash. The chest wall cannot be effectively immobilized due to respiration, so nonoperative analgesia is thus centered on interrupting its sensory innervation.

Analgesics may be categorized broadly into systemic and locoregional. The term *multimodal analgesia* refers to the use of different classes of analgesics simultaneously to manage pain. Multimodal analgesia should be employed routinely in the management of patients with chest wall injuries in order to target multiple pathways involved in pain sensation. In general, a tiered approach, in which potency is escalated sequentially and based on objective measurements of response to therapy (eg, the vital capacity), should be employed. It is also important to standardize analgesia to minimize variability and allow for optimal data collection and outcomes analyses. One such example of a standardized, tiered analgesic practice management guideline is shown in Fig. 28-3.

Unfortunately, narcotics continue to be used indiscriminately for analgesia in patients with chest wall injuries. This is due to a perceived lack of effective alternatives and a well-intentioned but misguided focus on pain as the "fifth vital sign." The United States consumes a disproportionate amount of opioids, and in many centers, opioid overdose has now surpassed trauma as a reason for visits to the emergency department.[46] In addition, narcotics have unfavorable side effects such as somnolence, constipation, and respiratory depression, which are particularly disadvantageous in patients with injury to the chest wall. Although many patients with rib fractures will require narcotics to control pain, these medications should be used in a protocolized fashion and only after nonnarcotic alternatives have been exhausted.

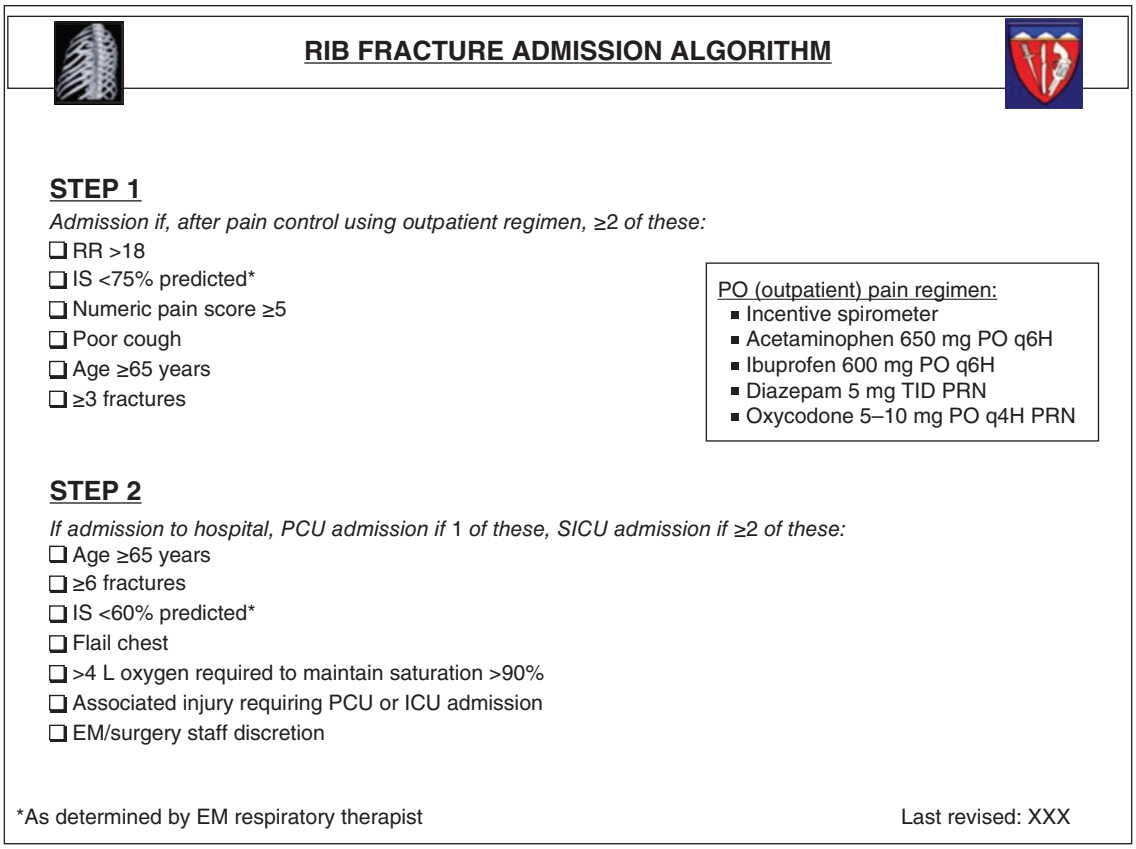


FIGURE 28-2 Denver Health Medical Center admission algorithm for patients with rib fractures. EM, emergency medicine; ICU, intensive care unit; IS, incentive spirometry; PCU, progressive care unit; PO, oral; PRN, as needed; q4H, every 4 hours; q6H, every 6 hours; RR, respiratory rate; SICU, surgical intensive care unit; TID, three times a day.

Both acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs) have been shown to provide comparable analgesia to oral narcotics in trauma patients and should be given routinely to patients with chest wall injury.[47] The contention that NSAIDs either increase bleeding risk or impair bone healing has been disproven.[48,49] Muscle relaxants, such as diazepam and cyclobenzaprine, may provide additional relief, although their efficacy in patients with rib fractures is largely unproven. Recently, novel narcotic alternatives such as oral gabapentin,[50] ketamine infusion,[51] and lidocaine infusion[52] have been evaluated specifically in patients with rib fractures. Although initial results have been underwhelming, their use has continued to increase in this patient population, and current studies are underway employing increased dosing regimens.

Locoregional anesthesia for rib fractures refers to therapy targeted to the intercostal innervation specifically and is applicable anywhere from the spinal cord to the terminal rami of the intercostal nerves. A thoracic epidural catheter is the most extensively studied locoregional anesthetic modality. Despite only being used in 3% of patients hospitalized with rib fractures, it is associated with decreased morbidity and mortality as compared to no locoregional anesthesia.[13,53,54] Alternatives to epidural anesthesia, including ultrasound-guided erector spinae and paravertebral catheters, eliminate the risk of an epidural hematoma and may be placed in the setting of a coagulopathy and irrespective of ongoing anticoagulation.[55] Surgeon-driven locoregional modalities, including continuous intercostal nerve block and video-assisted thoracoscopic surgery (VATS) intercostal nerve blocks with liposomal bupivacaine, carry the added advantage of intraoperative placement under direct visualization.[55-57] In practice, the choice of a locoregional modality will depend on the patient's injury pattern and local expertise. Some form of locoregional therapy, however, should be employed in all patients with rib fracture who do not respond to initial tiered therapy with multimodal agents. A recent practice management guideline from the Eastern Association for the Surgery of Trauma for pain management conditionally recommended routine use of both multimodal analgesia and thoracic epidural analgesia specifically in patients hospitalized with blunt thoracic trauma. The authors recognized, however, that the use of locoregional anesthesia itself was more important than the specific type selected.[58]

Indications for Surgery

SSRF is now performed routinely at most high-volume trauma centers.[14] Although familiarity with the technique of SSRF has improved markedly among trauma surgeons, ambiguity

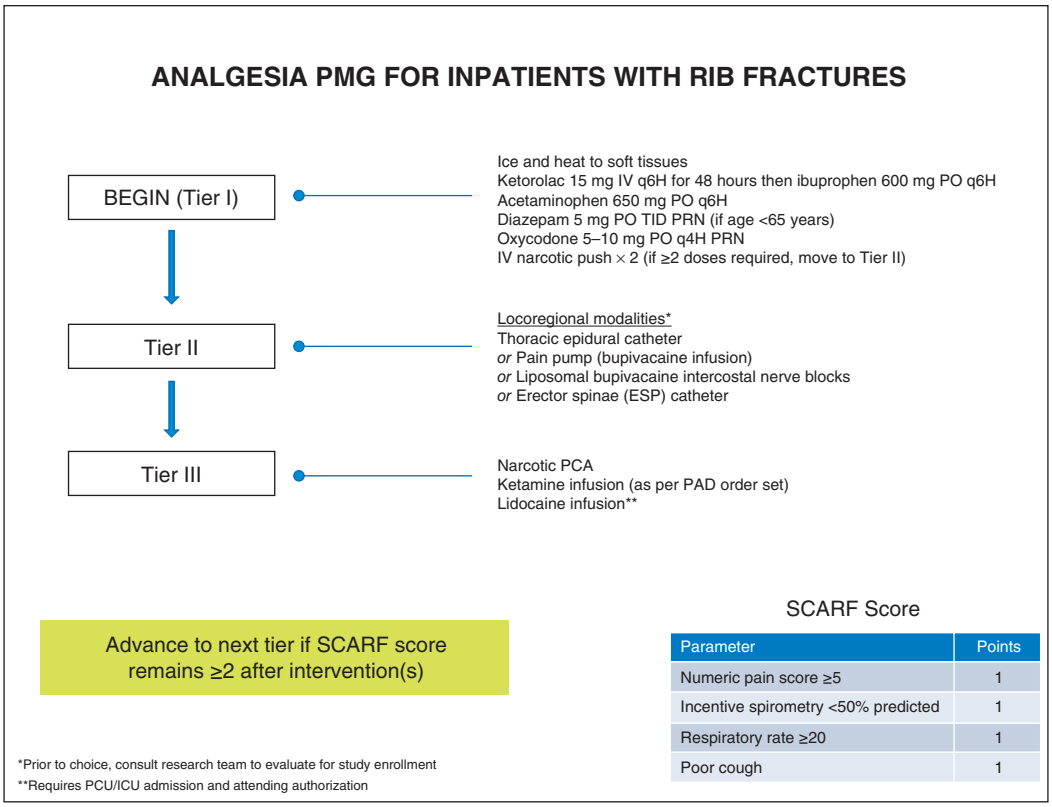


FIGURE 28-3 Denver Health Medical Center practice management guideline (PMG) for analgesia in patients with rib fractures. ICU, intensive care unit; IV, intravenous; PAD, pain, agitation, and delirium; PCA, patient-controlled analgesia; PCU, progressive care unit; PO, oral; PRN, as needed; q4H, every 4 hours; q6H, every 6 hours; SCARF, Sequential Clinical Assessment of Respiratory Function; TID, three times a day.

remains regarding the indications for surgery. Randomized controlled trials,[59-62] prospective nonrandomized trials,[63] and meta-analyses[64-67] have all shown improved outcomes with SSRF in terms of respiratory morbidity, length of stay, resource utilization, and, in some cases, mortality. Although sparse, data addressing long-term outcomes in patients with a flail chest managed operatively have also been favorable.[12,68,69] Accordingly, consensus documents conditionally recommend consideration of SSRF in all patients with flail chest.[52,70] It is important to note that the trials on which these recommendations are based vary widely in terms of the definition of flail chest, injury severity, time from injury to surgery, and surgical technique. In practice, SSRF should not be undertaken based on a radiographic finding of a flail segment alone. Rather, patients with a flail segment in conjunction with either a poor response to nonoperative therapy or respiratory failure due to rib fractures will likely derive the most benefit from surgery. The importance of patient selection is underscored by recent retrospective data suggesting little or no benefit of SSRF in certain cohorts of patients with a radiographic flail segment.[71-73]

Patients with multiple displaced rib fractures, in the absence of a flail segment, may also benefit from SSRF, although there are fewer data to support this recommendation. Many of the same pathophysiologic principles of flail chest such as painful motion at the fracture site causing splinting, displacement increasing the risk of nonunion, and risk of bony bridging to neighboring fracture fragments over time apply (Fig. 28-4). Several prospective studies have included patients with three or more bicortically displaced fractures, along with patients with a flail segment, and found benefit to SSRF.[74-78] One survey found that, in addition to the presence of three or more displaced fractures, at least two deranged pulmonary physiologic variables were required in order for respondents to recommend SSRF.[79] Multiple prospective trials that include patients with displaced fractures in the absence of a flail segment are currently underway. Pending the results of these studies, SSRF in this situation should be considered on a case-by-case basis and in the setting of a poor response to multimodal analgesia.

There is currently no evidence to support SSRF for nondisplaced fractures. Nondisplaced rib fractures, however, may undergo interval displacement due to patient motion, static and dynamic forces of chest wall muscles, and changes in intrathoracic pressure (Fig. 28-5). Patients with multiple nondisplaced fractures, and particularly those who worsen clinically, may warrant repeat CT imaging 48 to 72 hours after injury to assess for interval displacement, in which case SSRF may be reconsidered.

Additional potential indications for surgery include the following: (1) one or two painful, clicking, mobile rib

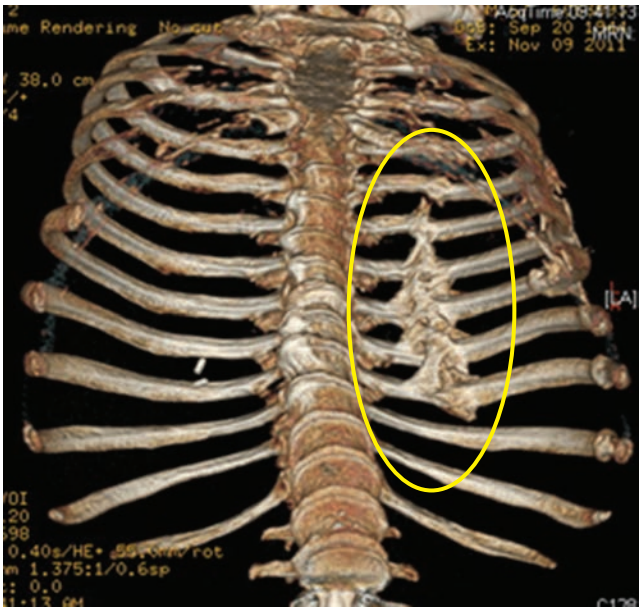


FIGURE 28-4 Bony bridging caused by multiple displaced posterior left-sided rib fractures. The consequences of the bridging for this patient were manifest as chronic dyspnea and a restrictive pattern on pulmonary function testing. (Reproduced with permission from Marasco S, Liew S, Edwards E, Varma D, Summerhayes R. Analysis of bone healing in flail chest injury: do we need to fix both fractures per rib? *J Trauma Acute Care Surg.* 2014;77(3):452-458.)

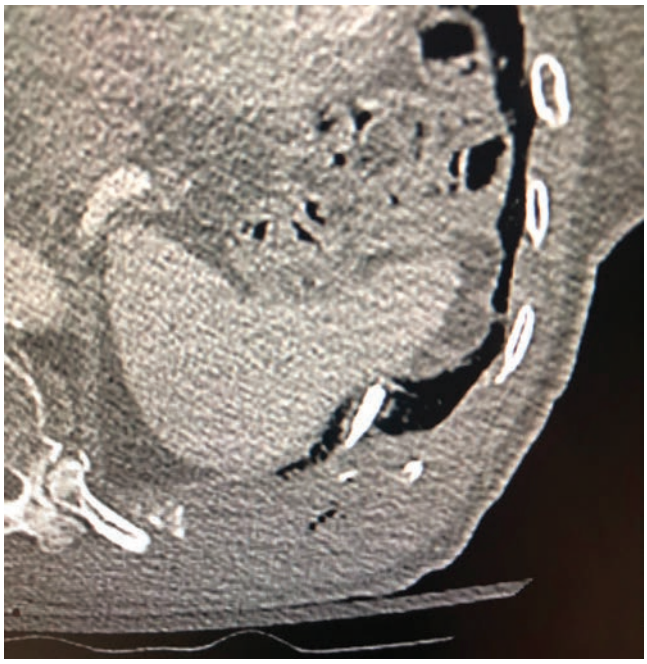


FIGURE 28-6 Impalement of the diaphragm and left lower lobe by a tenth rib fracture.

fractures in a highly functional patient (eg, professional athlete); (2) rib fracture impalement of internal organs, such as the lung, diaphragm, and spleen (Fig. 28-6); (3) "on the way out" of the thoracic cavity for other indications; and (4) following elective rib fracture or removal, as in the case of thoracic spine exposure. These indications have not been studied specifically, and recommendations are based on expert opinion as opposed to outcome data. Of these indications, painful clicking and impalement are most consistent with the benefits seen after SSRF in more severe injury patterns.

Contraindications to SSRF fall into three broad categories as follows: (1) patients who will not benefit from surgery; (2) patients who will likely benefit from surgery, but it must be delayed secondary to higher priority injuries; and (3) patients who may benefit from surgery and are undergoing a trial of nonoperative management in order to determine if it is needed. In general, patients who will require long-term mechanical ventilation for reasons unrelated to their chest wall injury will not benefit from SSRF, regardless of the pattern of rib fracture. Common examples of this clinical scenario include an associated severe traumatic brain injury, high spinal cord injury, and anoxic brain injury following cardiac arrest with some period of cardiopulmonary resuscitation (and a resultant anterior flail segment). Although there may be rare exceptions to this recommendation (eg, a patient with mild to moderate traumatic brain injury with a severe flail chest unable to wean from mechanical ventilation), SSRF in these patients should not be undertaken.

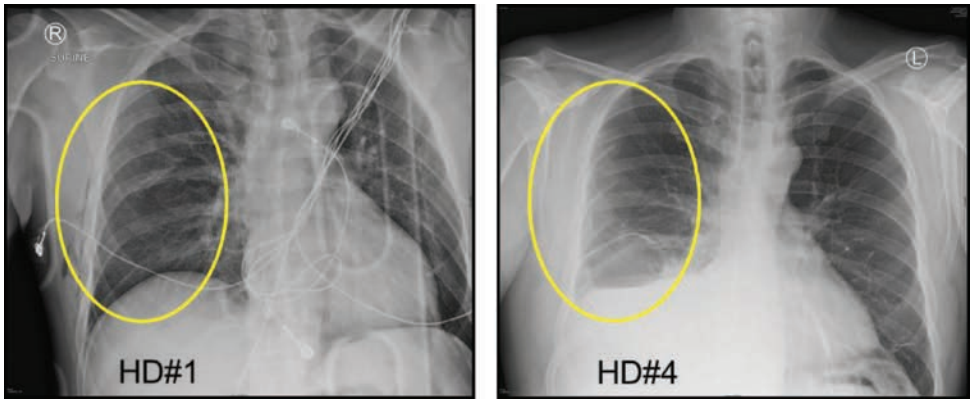


FIGURE 28-5 Interval displacement of right posterolateral rib fractures from hospital day 1 (HD#1) to hospital day 4 (HD#4). Note also the development of a right hemothorax.

One particular associated diagnosis that has received scrutiny in the literature is pulmonary contusion. An early report of SSRF in patients with severe pulmonary contusions concluded that surgery should not be undertaken, arguing that the main reason for pulmonary morbidity is the contusion itself, as opposed to the chest wall injury.[80] This study was limited by a small sample size, a relatively long interval from injury to surgery, and confounding by associated injuries. More recent studies have reported favorable outcomes after SSRF in patients with mild to moderate pulmonary contusions, and this diagnosis in and of itself should not be considered an absolute contraindication to surgery.[81] In practice, clinical judgment must be exercised regarding stability for transport to the operating room, positioning, and potential single-lung ventilation.

Pleural space infection has been considered a contraindication to SSRF due to the risk of postoperative infection in implanted hardware. The overall risk of hardware infection after SSRF is, however, less than 5%, and successful rib fixation with permanent hardware has been reported in the setting of severe pleural space infection.[82,83] The efficacy of SSRF in the setting of empyema remains to be determined.

Preinjury anticoagulation is not associated with worsened outcomes following either rib fractures or SSRF and is not considered a contraindication to surgery.[84] Open rib fractures are rarely encountered but may be considered for repair with permanent hardware after irrigation of the wound bed and in the setting of adequate soft tissue coverage of the hardware.

Elderly patients have traditionally been excluded from studies of SSRF due to a presumption that surgery would be poorly tolerated.[59] More recent data have suggested that the geriatric population may benefit even more from SSRF as compared to younger patients.[76,85] This benefit may be derived from the relative inability of elderly patients to tolerate the pain and respiratory depression from unstable fractures. Pending further data, SSRF should remain a treatment option for elderly patients with severe injuries to the chest wall.

Certain associated injuries preclude early SSRF, including shock with active resuscitation, intracranial hypertension, unstable spine injury, rupture of the thoracic aorta, and inability to position patients either prone or decubitus due to physiologic instability or implanted devices (eg, external fixator on pelvis or extremity). In these cases, patients require stabilization prior to SSRF. In the absence of these conditions and when indicated, SSRF should be performed as soon as possible following injury and ideally within 48 hours. Delays in surgery expose the patient to additional pulmonary morbidity from unstable fractures, prolong the time to definitive drainage of the pleural space (and potential provision of guided locoregional anesthesia), and increase the amount of tissue edema and inflammation. A multicenter study of time to SSRF found that, after adjustment for injury severity, outcomes were improved when surgery was performed within 48 hours.[86] These findings have been replicated in a subsequent single-institution analysis.[87] In addition to the aforementioned factors, time to surgery is commonly delayed due to lack of availability of surgeon and operating room. Therefore, trauma centers that perform SSRF with regularity are encouraged to develop a pathway to ensure operating room access for patients with severe injuries to the chest wall within 48 hours, similar to recommendations for other orthopedic injuries.[88]

In certain patients, the benefit of SSRF may not be certain at the time of initial assessment and imaging, so a trial of nonoperative management is warranted. One example of this situation might be a patient with three displaced fractures and marginal pain control after locoregional anesthesia. In general, trials of nonoperative management should be both standardized and brief, such that patients who fail do so early and before respiratory consequences occur. In general, patients who require locoregional anesthesia for three or more displaced rib fractures should be assessed for response within 12 hours, at which time the decision for surgery is made. Prolonged (ie, 7–10 days) trials of nonoperative management expose patients to the risks of pulmonary compromise, while delaying stabilization.

Indications for surgery in sternal fractures are less established as compared to rib fractures. Most surgeons who perform sternal fracture repair with regularity cite visible deformity, loss of sternal continuity, complete displacement, sternomanubrial joint dislocation/fracture, and persistent sternal mobility/clicking as indications for repair (Fig. 28-7).[89]

Surgery to repair chronic nonunions of rib and sternal fractures differs in many ways from acute SSRF, including indications, surgical technique, and likelihood of success.[90-92] In general, delayed fixation should be undertaken by surgeons with expertise in the management of bony nonunion, as these fractures routinely require reaming, gap bridging, and some form of bone grafting. Although some success in terms of pain relief has been reported in cases of painful clicking at the fracture sites,[93,94] the success of delayed SSRF is far lower than in the acute setting. When indicated, the surgery should be performed early during the index hospitalization.

Operative Technique

The surgical technique of rib repair has undergone refinement from the relatively morbid posterolateral thoracotomy to muscle-sparing, minimally invasive approaches. Although videoscopic extrathoracic[95,96] and intrathoracic[97,98] SSRF approaches have been described, most operations are performed via open incisions and using permanent plates and screws.

Prior to surgery, the patient's chest CT should be reviewed systematically in order to determine which fractures will be repaired. A data collection tool on which rib fractures can be checked off serially as they are repaired can be helpful to display in the operating room. (Fig. 28-8). In general, fractures of ribs 3 to 10 should be considered for repair. Fractures of ribs 1 and 2 are relatively difficult to access surgically and contribute minimally to respiratory function. Similarly, fractures of the floating ribs typically do not cause significant pain or pulmonary embarrassment. One exception may

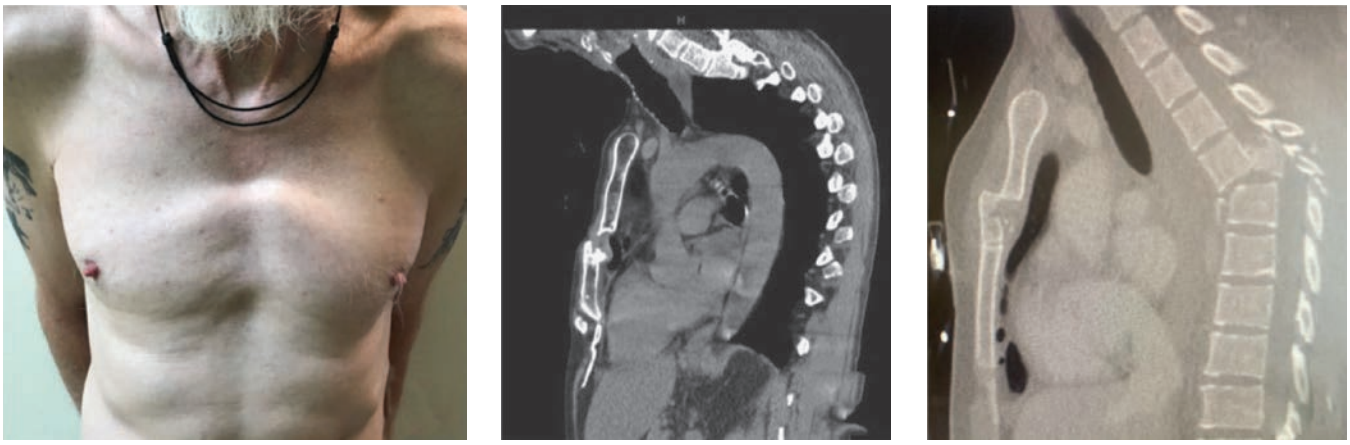


FIGURE 28-7 Examples of sternal fractures that underwent operative repair: (**A**) visible deformity; (**B**) loss of sternal continuity; (**C**) sternomanubrial joint fracture/dislocation with associated T6 vertebral body fracture.

be impalement of the liver, spleen, diaphragm, or kidney, in which case the fracture fragment may simply be resected. In general, at least 3 cm of distance is required between a posterior rib fracture and the transverse process to successfully place a plate. Practically, fractures within 5 cm of the transverse process should not be repaired, because the exposure required and the stability afforded by overlying muscle and ligaments in this area likely mitigate any additional benefit from surgical fixation. Anterior fractures may be stabilized to either the costal cartilage and/or the sternum.

Multiple fractures, as seen in a flail segment, should all be repaired. Although repair of single fractures (usually anteriorly) will theoretically eliminate the pathophysiology of flail chest, instability in the other fractures remains and places the patient at risk of both persistent pain and bony bridging.[99] Finally, there is no evidence to support an "every other" approach of rib fracture repair. Rather, it is recommended to repair each rib because the morbidity is relatively low and the untreated fractures may remain symptomatic, although this is not the practice in many centers.

Conceptually, both positioning and incision placement are dependent upon the location of fractures. Furthermore, incisions should be based on the borders of chest wall muscles as opposed to directly overlying the fractures such that muscles may be retracted as opposed to divided. Lateral rib fractures may be approached with the patient in the lateral decubitus position and through a 7- to 9-cm longitudinal incision, placed along the anterior border of the latissimus dorsi muscle and coursing obliquely (Fig. 28-9). The anterior border is then incised, and a flap is raised underneath the muscle

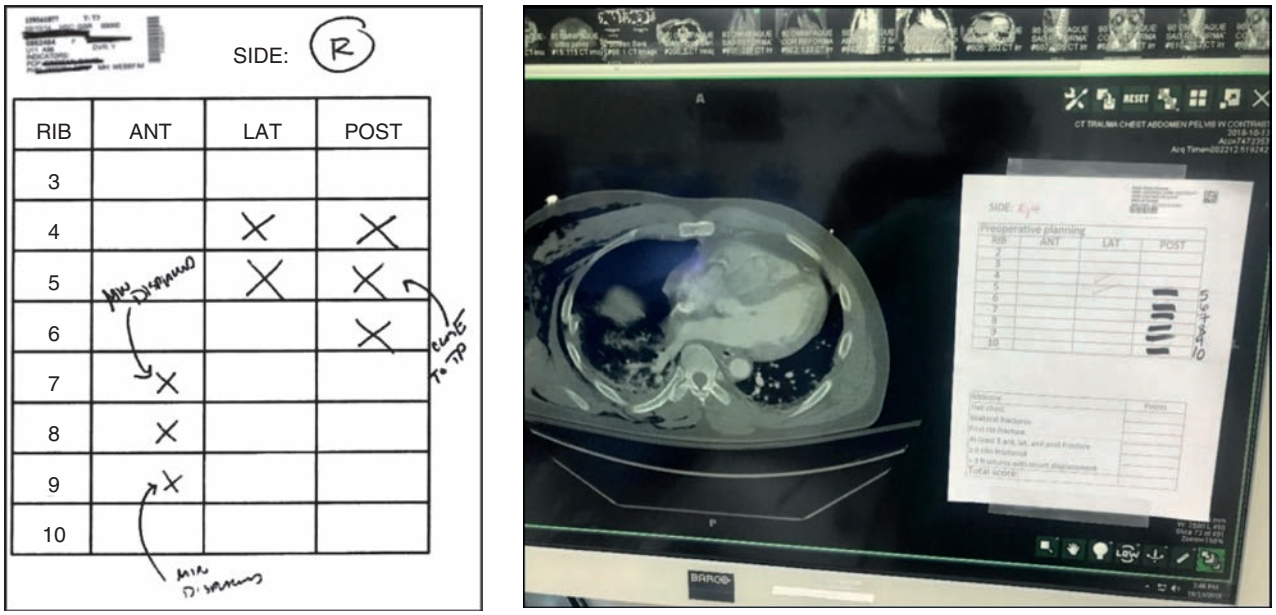


FIGURE 28-8 Example of a standardized data collection tool for preoperative planning of surgical stabilization of rib fractures. The tool is displayed alongside the patient's imaging so that fractures are checked off intraoperatively after they are repaired.

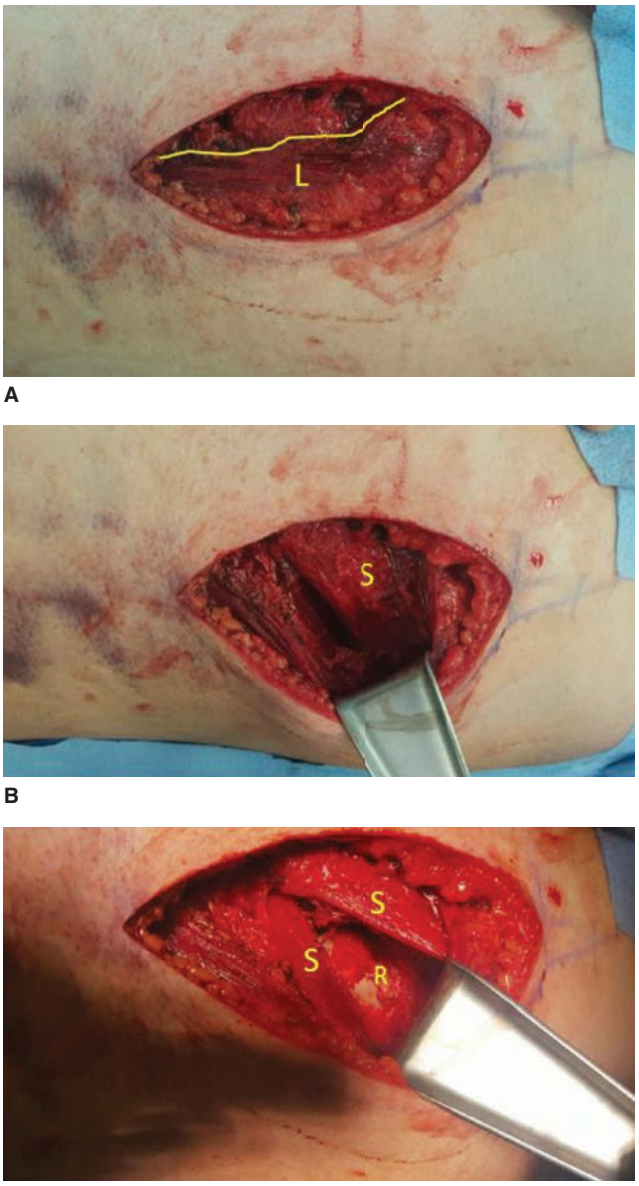


FIGURE 28-9 Surgical approach to lateral rib fractures. (**A**) The medial border of the latissimus dorsi muscle (L) is incised and a sublatissimus flap is raised. (**B**) The serratus anterior muscle (S) is split along the direction of its fibers. (**C**) The rib fracture (R) is encountered under the serratus anterior muscle (S).

such that it may be retracted posteriorly. This exposes the muscle branches of the serratus anterior, which may be split to expose the rib fractures. Care must be taken at this point to avoid injury to the long thoracic nerve, which courses down the anterior axillary line and superior to the serratus anterior muscle. Typically, three to five sequential fractures may be exposed through a single split of the serratus anterior muscle.

Anterior fractures may be exposed with the patient in the supine position and through an oblique incision along the inframammary fold. A subpectoral flap is then developed, which provides excellent exposure to anterior ribs 4 to 6. Anterior fractures of the third rib may be approached through a small horizontal incision directly over the fracture, with splitting of the fibers of the pectoralis major and minor muscles.

Posterior fractures are typically the most difficult to repair due to proximity to the transverse processes of the spine, angulation of the ribs, and subscapular location. These fractures may be approached with the patient in the prone position and the ipsilateral arm supported on a table that is lowered approximately 8 in relative to the operating table. This maneuver allows for lateralization of the scapula and facilitates exposure of posterior, subscapular fractures. A longitudinal incision is then made just medial to the scapular tip, and the triangle of auscultation is developed, raising both subtrapezial and sublatissimal flaps. This exposure also affords the opportunity to simultaneously address thoracic spine fractures (Fig. 28-10).

Patients with a flail segment typically have a combination of either anterior and lateral fractures or lateral and posterior fractures. Either fracture pattern may be approached through two of the incisions described earlier. Alternatively, a traditional posterolateral thoracotomy may be used. Another common fracture pattern is an anterior flail chest with bilateral anterior fractures. This fracture pattern may be exposed via bilateral inframammary incisions.

In addition to rib repair, SSRF affords an opportunity to perform pulmonary toilet, evaluate and manage intrapleural pathology, and provide guided locoregional anesthesia. The ideal approach involves routine use of both bronchoscopy and VATS during SSRF. Routine pleural irrigation at the time of SSRF is associated with a decreased likelihood of subsequent retained hemothorax, and resection/repair of lung parenchymal injury at the time of VATS decreases both duration of postoperative chest tubes and risk of infection.[100,101] Additional advantages of routine VATS include diagnosis and repair of occult injuries to the hemidiaphragm, instillation of directed locoregional anesthesia, directed placement of chest tubes, and education of surgical trainees.

Sternal repair was first undertaken in 1943 using a Kirschner wire.[102] Since then, permanent plates have become the standard modality of fixation, although absorbable plates have also been used.[103,104] The surgical technique consists of a midline, longitudinal incision exposing the fracture, evacuation of the surrounding hematoma, and debridement of nonviable tissue. Reduction forceps are then introduced laterally into the surrounding intercostal spaces, being careful to avoid injury to the internal thoracic vessels. Bicortical fixation is recommended, with screw length estimated by CT and intraoperative calibration.

Complications of Surgery

The incidence of complications following SSRF appears to be less than 5%,[82,105-107] although long-term follow-up has not been reported in most series. Intraoperative complications can involve inadvertent injury to chest wall nerves, intercostal vessels, and lung parenchyma. The nerves most commonly

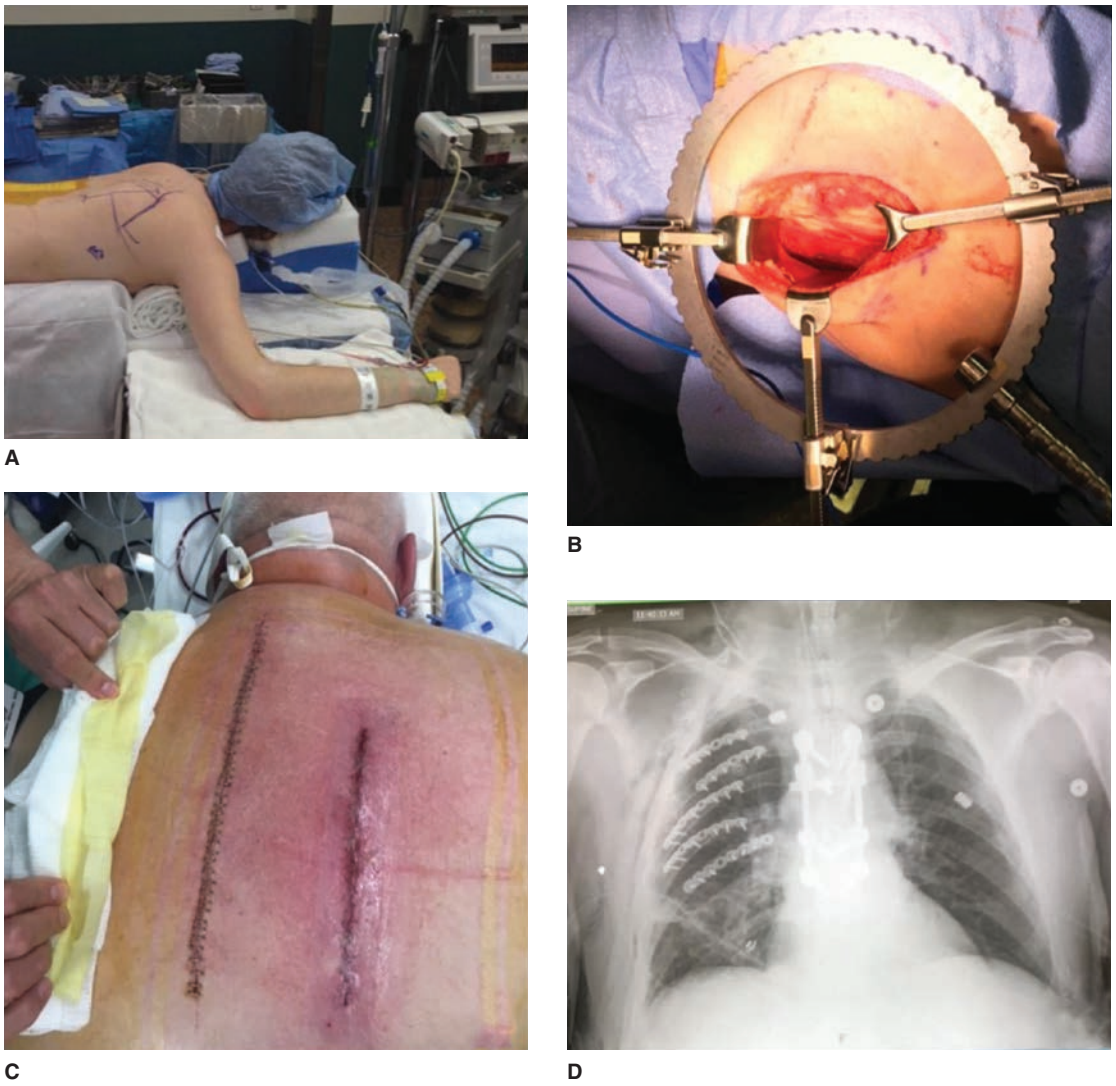


FIGURE 28-10 Positioning and exposure for posterior rib fractures. (**A**) The patient is positioned prone with the ipsilateral arm lowered in order to retract the scapula laterally. (**B**) A longitudinal incision is made over the triangle of auscultation. Note the erector spinae fibers at the base of the incision and the use of a self-retaining retraction system. (**C, D**) Simultaneous thoracic spine and posterior rib fractures.

encountered during SSRF are the long thoracic nerve during high lateral and axillary exposures, spinal accessory nerve during high posterior exposures, and the lateral cutaneous branches of the intercostal nerves in both. Although these nerves course in a usual location, injury leading to scapular and abdominal muscular dysfunction has been reported.[108,109]

Immediate postoperative complications are mostly due to bleeding, which may arise from injury to intercostal vessels or muscle bleeding from raising flaps. Surgical drains have not been shown to mitigate this risk and do not substitute for meticulous hemostasis at the time of surgery. Deep surgical site infection requiring hardware removal occurs in approximately 1% of cases of SSRF and is associated with an increased body mass index and hemorrhagic shock on presentation. Junker et al[110] recently reported an algorithm for management of suspected hardware infection, as well as a favorable salvage rate from hardware using antibiotic beads.

Late dislodgement and migration of hardware also appear to be relatively rare following SSRF.[107] The most common location for hardware to fail is posteriorly, likely due to both the angulation of the rib and shear forces exerted on the plate by the serratus muscles.[106] Most cases of hardware dislodgement are asymptomatic and occur long after the fracture has healed. Certain cases will result in uncomfortable, palpable subcutaneous hardware that may require removal. Absorbable plates, although not widely developed to date, may eliminate this risk.

Intercostal Muscles, Costal Cartilage, and Costal Margin

The costal cartilages, interchondral joints, and costal margin–diaphragm interface represent an interrelated group of structures, the injuries to which are largely unappreciated.[111] Fractures of the costal cartilages have been reported predominantly in

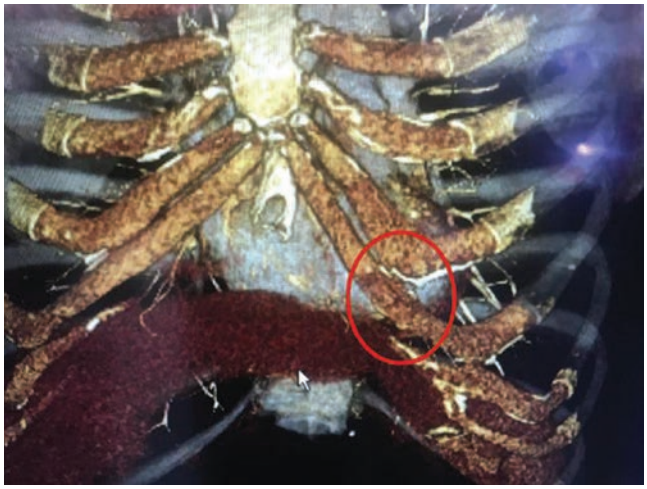
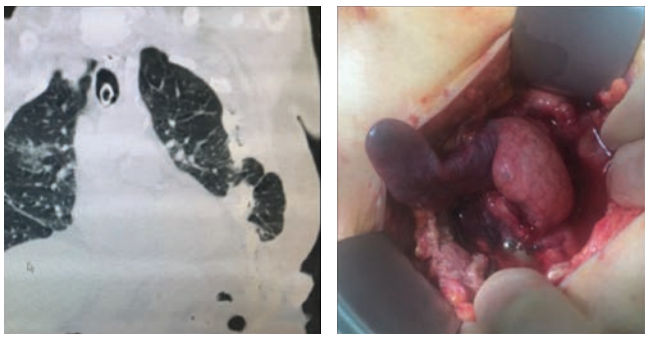


FIGURE 28-11 Computed tomography chest reconstructions formatted to costal cartilage and demonstrating an acute fracture of the left seventh costal cartilage.

the setting of professional contact sports.[112,113] Because diagnosis of these injuries is often delayed due to normal imaging, persistent pain and point tenderness should prompt three-dimensional reconstruction of the chest CT formatted specifically to cartilage[114] (Fig. 28-11). Although the results are favorable, data supporting the efficacy of surgical repair of costal cartilage fractures are limited to case reports.[115]

Costal margin rupture, with or without involvement of the diaphragm and intercostal muscles, represents a rare, elusive injury. Gooseman et al[116] recently proposed a unifying classification of such injuries to guide treatment. This system recognizes the spectrum of pathology, which may involve a tear of the diaphragm, through the costal margin, and finally one or more intercostal muscles and ribs. Injury to both intercostal muscle and costal margin should be suspected in the presence of posterior rib fractures and splaying of the ribs anteriorly on a CT of the chest. In extreme cases, complete loss of intercostal muscle may result in pulmonary herniation and necrosis (Fig. 28-12).[117] Management of these complex injuries may require a combination of suture cerclage of the ribs and costal cartilage/margin, intrathoracic mesh or extrathoracic mesh placement, and suture repair of the diaphragm.



A B

FIGURE 28-12 Acute intercostal margin rupture from blunt trauma with herniation of the lingula through the fifth intercostal space. (**A**) Coronal series of chest computed tomography. (**B**) Intraoperative photograph.

INJURY TO THE LUNGS

Although physiologically complicated, the lungs are anatomically simple, consisting primarily of alveoli and blood vessels. The paired large pulmonary artery and vein are high-volume, low-pressure circuits (see Atlas Figures 32 and 33). The bronchial vascular bed is characterized by a higher systemic pressure but relatively small-caliber vessels. The anatomic simplicity of the lungs lends itself to a limited parenchymal response to trauma regardless of the severity and mechanism of injury. The alveoli can rupture, causing a pneumothorax. Compressed, contused, or otherwise mechanically disrupted pulmonary parenchyma can also lead to pulmonary contusion, compromising the ability of the injured lung to perform its basic functions. Hemothoraces are common, with up to 300,000 cases each year, and can result from bleeding of the injured parenchyma itself or the vasculature of the chest wall, particularly the intercostal and mammary arteries.[118,119] Associated bony injuries to the chest wall can increase respiratory compromise and distress.

Very large pneumothoraces produce tension by shifting the mediastinal structures toward the contralateral side with resulting anatomic distortion. This increase in intrathoracic pressure leads to decreased venous return, decreased cardiac output, and, if untreated, cardiac arrest. In contrast, large hemothoraces generally produce symptoms through the effects of hypovolemia, although a massive hemothorax may also produce tension physiology.

Presentation and Evaluation

Any patient with blunt or penetrating chest trauma is at risk for injury to the lung. The mechanism of injury, time from injury, vital signs, and neurologic status at the scene and any changes during transport are critical components of an adequate history, which can guide both rapid evaluation and treatment.

Physical examination alone may confirm the diagnosis of an injury to the lung. The presence of distended neck veins, tracheal deviation, subcutaneous emphysema, chest wall instability, absent breath sounds, or muffled heart sounds can provide crucial information allowing for both prompt diagnosis and treatment of a tension pneumothorax. Vital signs should be frequently monitored, with careful attention to the work of breathing and arterial saturation. Hypoxia and increased work of breathing may be manifested by anxiety, confusion, combative behavior, dyspnea, or the use of accessory muscles. Findings of subcutaneous air or decreased breath sounds should alert the clinician to the possibility of a closed or tension pneumothorax and/or hemothorax. Prompt placement of a 36F to 38F thoracostomy tube or 8.3F to 14F pigtail catheter if only a pneumothorax is present in an unstable patient is imperative because radiographic confirmation

may delay treatment and lead to further physiologic derangement (see Atlas Figure 21).

Hemodynamic instability in a patient with penetrating thoracic trauma is virtually always an indication for operative exploration. Conversely, hemodynamically stable patients with penetrating or blunt thoracic injury may benefit from additional imaging, including chest x-ray, ultrasound, and/or CT. A single anteroposterior image of the chest is commonly used as the initial imaging modality in injured patients. Injuries that can be identified using a chest x-ray include a pneumothorax, hemothorax, pulmonary contusion, rib fractures, mediastinal widening, or a suspected rupture of the hemidiaphragm. It is important to note that a routine chest x-ray is associated with a relatively low sensitivity (27%–83%) in the diagnosis of a pneumothorax, missing up to 50%[120-124]. Conversely, bedside ultrasound, as part of the extended focused assessment with sonography for trauma (E-FAST), has been shown to have sensitivity rates of 86% to 100% in the diagnosis of a pneumothorax but is limited in that it is unable to assess for injuries apart from a pneumothorax, hemothorax, or pericardial fluid.[121,125,126] Chest x-ray and bedside ultrasound have the advantages of being quick and portable and not requiring transport of patients. Although it does expose patients to considerably higher doses of radiation as compared to plain films, CT is the gold standard for imaging of the injured patient.[25,121,127,128] Over the past several years, this modality has become faster, more informative with higher resolutions and reconstruction capabilities, and safer with lower radiation doses.[129,130] Overall, studies have shown CT to identify occult injuries not identified on a chest x-ray in 18% to 71% of patients.[25,131,132] In addition to the imaging modalities mentioned earlier, stable patients may require esophagoscopy, bronchoscopy, and echocardiography.

Pneumothorax

Pneumothoraces not visualized on chest x-ray are often seen on CT. If they are small and the patient is asymptomatic, observation is typically all that is required. For patients undergoing operation for an associated injury or intubation for positive-pressure ventilation, the chance that a small pneumothorax will become clinically significant is relatively low, but all patients should be monitored for this possibility in the early phases after injury.[133]

In general, a large pneumothorax seen on CT, but not detected on a chest x-ray, is most often anterior. Evacuation of a larger collection can very effectively be undertaken by insertion of a thoracostomy tube or pigtail catheter, as noted earlier.

Hemothorax

Current practice management guidelines from the Eastern Association for the Surgery of Trauma recommend all hemothoraces be considered for drainage, regardless of size.[119] Although all hemothoraces should be considered for drainage, tube thoracostomies are associated with complication rates of 20% to 40%, and there is literature that suggests small-volume hemothoraces (<300 mL) are able to be successfully managed without tube thoracostomy.[118,134-136] If management without tube thoracostomy is elected, monitoring should be performed with serial chest x-rays to document resolution. Any moderate or large hemothorax, however, should be drained with a thoracostomy tube or pigtail catheter. The tube or catheter should be placed posteriorly to allow for dependent drainage. Although traditional teaching has included the use of a size 36F to 38F thoracostomy tube, more recent literature has shown no difference in outcomes with smaller-sized tubes, ranging from 20F to 32F, or pigtail catheters.[137,138]

In hemodynamically stable patients, indications for surgical intervention for hemothoraces in the acute setting include initial drainage of 1200 to 1500 mL or 200 mL/h of output for 2 to 4 hours after placement.[119,139] Blood left within the pleural cavity has a tendency to form mature clot resistant to subsequent drainage via a thoracostomy tube. Evaluation for effective drainage by repeat imaging is advised because undrained blood, or "retained hemothorax," may progress to a fibrothorax with lung entrapment or become infected, resulting in an empyema.

Pulmonary Contusion

Pulmonary contusions are the most common injuries of the lung from a blunt mechanism of trauma[140,141] (Fig. 28-13). In one series, pulmonary contusions were found in 27% of patients with an Injury Severity Score (ISS) of 15 or higher.[141] Although they can occur with any blunt mechanism or chest injury, they are highly associated with motor vehicle collisions, particularly front and lateral impacts, and rib fractures, especially a flail chest.[141] Traditionally the diagnosis of a pulmonary contusion was associated with high morbidity. The severity of these injuries ranges from clinically silent to more severe cases causing severe respiratory distress. With increased use of CT scans in the evaluation of trauma patients, pulmonary contusions, particularly minor ones, are now diagnosed

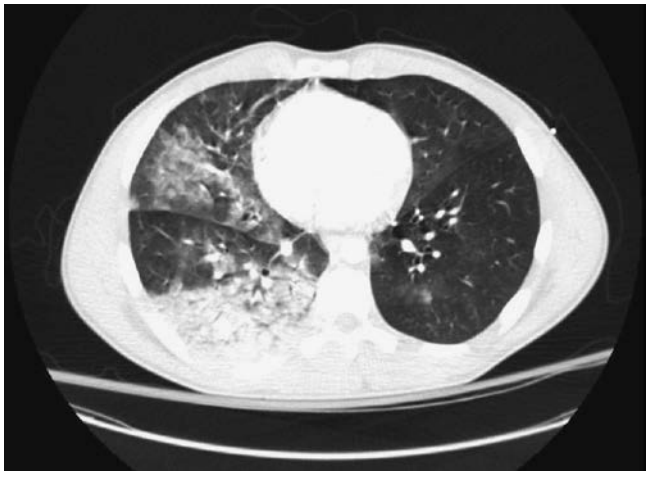


FIGURE 28-13 Pulmonary contusion.

more frequently.[142] In one series, 73% of pulmonary contusions were able to be visualized on a CT scan.[142] The clinical symptoms of severe pulmonary contusion include respiratory distress, increased work of breathing, hypoxia, and, less commonly, hypercarbia. One of the hallmarks of this pathology is that clinical symptoms and radiographic findings increase over time, appearing within 4 to 6 hours of injury and evolving over 3 days, and most resolve in 1 week.[143] Supportive treatment with judicious volume administration, pulmonary toilet, and supplemental oxygen is typically all that is necessary. For patients with concomitant rib fractures, adequate pain control is essential to allow for adequate pulmonary toilet. Noninvasive positive-pressure ventilation may be used in patients with significant hypoxemia who otherwise are not in respiratory distress and has been shown to avoid the need for intubation in approximately 10% of patients with acute lung injury.[144] Mechanical ventilation is indicated for respiratory distress or failure refractory to less invasive therapies.[141]

Lung Laceration

Patients with significant lung lacerations will often have large air leaks or, less commonly, hemoptysis. Bronchoscopy is the modality of choice to diagnose a tracheobronchial injury, which may also be the cause (see Chapter 29). Blood and secretions must be suctioned clear, allowing unimpaired visualization of the entire airway. When encountered, large air leaks from either a bronchus or lung resulting in respiratory compromise generally require thoracotomy. While also rare, significant hemoptysis can result in profound respiratory compromise, and bronchoscopy may permit localization of the bleeding to a specific pulmonary lobe or segment. Control of the airway is by a double-lumen endotracheal tube, selective mainstem intubation, or a bronchial blocker. Other adjuncts that may prove useful in optimal management include lateral decubitus positioning, catheter-based therapy, and surgery.

Indications for Operation

In addition to the volumes of blood from a thoracostomy tube mentioned earlier, thoracic trauma resulting in persistent hemodynamic instability, without another obvious source, should also prompt emergent thoracic exploration. Delaying emergent thoracic exploration may result in increases in morbidity and/or mortality.[145]

Care must be exercised when evaluating output from a thoracostomy tube. Although a dramatic decrease in output may signify a cessation of intrathoracic bleeding, it may also be the result of clotting of the thoracostomy tube. An increasing hemothorax in these instances may not be appreciated until visualized on subsequent chest x-ray or chest CT. Although a second chest tube may be helpful, patients with a large retained hemothorax should generally be explored and drained. A thoracoscopic approach is often successful, particularly if performed early within the first few days following injury, before the clot becomes organized and loculations and adhesions form.

Delayed operative intervention may be indicated for a variety of traumatic complications, including retained hemothorax, persistent air leak, missed injury, and empyema. Posttraumatic empyema is almost always best treated with operation. Many of these other nonemergent procedures can be performed using noninvasive methods, such as VATS.[146,147]

Surgical Exposure

There are a number of operative approaches to the thorax, each with advantages and disadvantages. Unlike an elective thoracotomy, in which the posterolateral approach is most commonly used, several important factors influence the choice of the incision for a traumatic injury. The overall clinical condition, hemodynamic instability, results of the imaging studies, and presence of concomitant injuries will influence the operative approach.

Commonly employed operative approaches include anterolateral, bilateral anterior ("clamshell"), and posterolateral thoracotomies and median sternotomy. As a general rule, an anterolateral thoracotomy, which can be extended as a clamshell, is the preferred incision for exploring the hemodynamically unstable patient. It affords excellent exposure to both pleural spaces and the anterior mediastinum. The main disadvantage of the anterolateral approach is the inability to provide adequate exposure of posterior structures. By extending the ipsilateral arm and placing a bump to elevate the thorax approximately 20°, the incision can be carried to the axilla, improving posterior exposure (Fig. 28-14).

A median sternotomy provides excellent access to the heart, great vessels, and anterior mediastinum. It is versatile and can be extended as an abdominal, periclavicular, or neck incision. Widely incising the pleura provides access to either hemithorax, whereas exposure of posterior structures is limited. The "trapdoor" incision is rarely used since left-sided

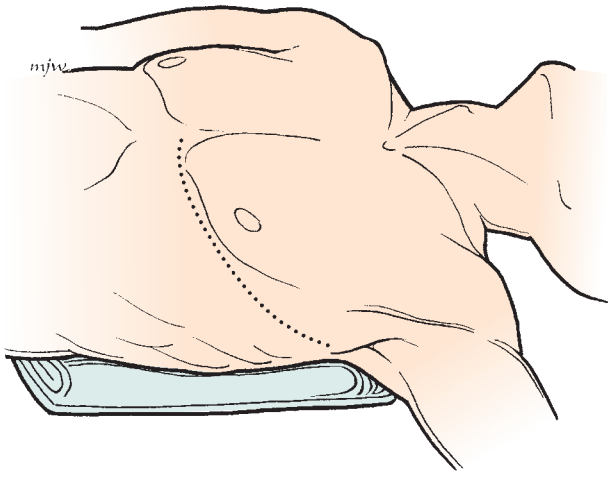


FIGURE 28-14 Illustration of incision for bilateral anterolateral thoracotomy.

thoracic vessels can be approached via sternotomy with extension.[148,149]

The posterolateral thoracotomy affords optimal exposure of the hemithorax, especially the posterior structures, and is the standard incision for most elective operations, as noted earlier. Its lack of versatility limits the usefulness in trauma, but it is the preferred approach to repair intrathoracic tracheal and esophageal injuries (see Chapter 29).

Operative Techniques

The use of a double-lumen endotracheal tube, as commonly used in elective thoracic operations, dramatically improves operative exposure. This adjunct is not, however, frequently used in trauma and, in particular, is not employed for emergency thoracotomies. Lung isolation in hemodynamically compromised trauma patients is not likely to be tolerated. In addition, the time typically required to ensure proper tube placement to facilitate single-lung ventilation is not warranted in an emergency. One exception may be in the setting of massive hemoptysis, where lung isolation may indeed be lifesaving. In contrast to other emergent thoracotomies, the placement of a double-lumen tube should be considered because it improves exposure and facilitates pulmonary resection. If a single-lumen tube is used, intermittently holding ventilation is advantageous during the critical steps of pulmonary repair or resection. If the patient's respiratory status is tenuous, however, any extended interruption of ventilation and oxygenation may precipitate decompensation. In this case, manual compression of the adjacent lung tissue may provide sufficient exposure to facilitate operative repair or resection.

Upon entering the chest, blood and clot should be evacuated, allowing a thorough examination and exploration of the hemithorax. The lung is mobilized by incising the inferior pulmonary ligament and lysing any adhesions. Exsanguinating hemorrhage demands immediate attention, and initial control is achieved with digital pressure. This allows time for ongoing volume resuscitation and an improved assessment of the injury. Hilar bleeding is a particularly significant challenge. Bleeding from the low-pressure pulmonary artery is more similar to a major systemic vein hemorrhage than arterial bleeding sources at other locations in the vasculature. There are several techniques for hilar compression. Finger occlusion or double looping placement of a Penrose drain around the hilum in a tourniquet fashion are both techniques that can be rapidly performed. With the latter technique, tightening the elastic Penrose around the hilum will effectively provide temporary vascular control. More definitive and secure control of the hilum can be achieved by placing a vascular clamp. Finally, the lung can be twisted on itself at the level of the hilum, which occludes the pulmonary artery and vein, as well as the mainstem bronchus. Clamping the hilar vessels or performing the "hilar twist" may, however, result in further decompensation in hemodynamically compromised patients. The rapid increase in pulmonary artery pressure can cause acute right heart dysfunction or failure, with catastrophic consequences.

There are a number of techniques for repair of the lung. The decision regarding the technique chosen will be influenced by the type and severity of the parenchymal injury, concomitant injuries, and the patient's physiologic status. Pneumorrhaphy is the simplest technique and is generally used to treat superficial pulmonary lacerations. The laceration is closed using an absorbable suture in either a running simple or mattress fashion. More extensive injuries require resection including simple wedge resection, pulmonotomy with selective vascular ligation, and nonanatomic and formal anatomic resections. Peripheral lacerations not amenable to simple repair can be treated by wedge resection using any of the commercially available cutting staplers. It is crucial, however, to determine the location of major pulmonary artery branches prior to firing the stapler when injuries are located closer to the hilum.

Significant lung injuries, particularly those from gunshot wounds, are often best treated with pulmonotomy[150-152] (Fig. 28-15) (see Atlas Figure 34). This is performed by placing the jaws of the stapler through the injury tract itself and firing it, similar to the technique used to expose and repair intraparenchymal hepatic injuries. This technique results in the exposure of bleeding vessels and injured airways for individual ligation. The staple line can be oversewn with a running absorbable suture to achieve adequate hemostasis and an airtight seal. In general, this method is most appropriate for peripheral injuries because this method is not typically appropriate for long central missile tracts.

Significant lobar injuries not amenable to pulmonotomy can be treated by nonanatomic resection or formal lobectomy. For the latter, the arterial and venous lobar branches must be dissected in the hilum and either ligated or stapled. Similarly, the lobar bronchus is identified and divided using a stapler. Prior to firing the stapler, and with the bronchus occluded by the stapling device, the lung is inflated. The lobe to be resected will not inflate, ensuring the appropriate bronchus is transected.

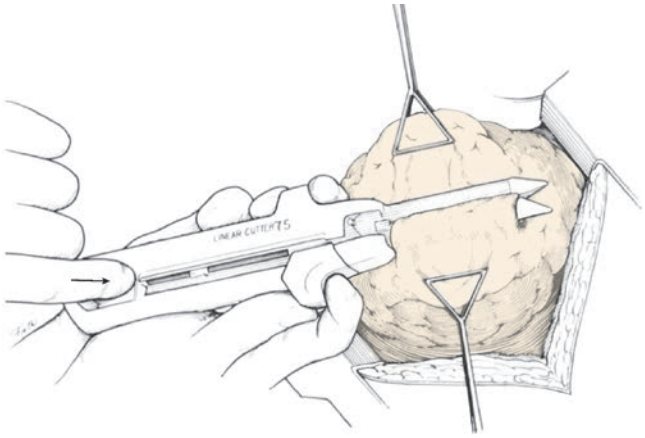


FIGURE 28-15 Illustration of a stapled pulmonotomy. (Reproduced with permission from Asensio JA, Demetriades D, Berne JD, et al. Stapled pulmonary tractotomy: a rapid way to control hemorrhage in penetrating pulmonary injuries. *J Am Coll Surg.* 1997 Nov;185(5):486-487. Copyright © 1997 American College of Surgeons. Published by Elsevier Inc. All rights reserved.)

Hilar injuries pose special challenges because hemorrhagic shock is almost always present and the anatomic challenges are significant. In very proximal hilar injuries, inflow occlusion is virtually always necessary in order to assess the extent of injury. Opening the pericardium and controlling the intrapericardial pulmonary artery and vein is a useful maneuver in this regard. Hilar injuries are rarely amenable to direct repair and may require pneumonectomy. Unfortunately, mortality after pneumonectomy for patients in shock approaches 100%, with patients dying from either uncontrolled hemorrhage or acute right heart failure.[153-155] If pneumonectomy is considered, it should be performed early, and rapid treatment of right heart dysfunction and support with extracorporeal membrane oxygenation may improve survival in these devastating injuries.[156]

The concept of damage control, originally described for penetrating abdominal trauma, has been expanded to include thoracic injuries as well. The well-established principles of hemorrhage control, resuscitation in the intensive care unit, and a planned, delayed definitive repair are applicable to patients with thoracic trauma with severely impaired physiology as well. Hemorrhage from named vessels and structures is controlled, the pleural cavity or cavities are packed, and the chest is left open. Experience has demonstrated that, when appropriately used, thoracic packing does not interfere with cardiac or pulmonary function. Once normal physiology is restored, the packs are removed, and the chest closed at a reoperation. In a series of 44 patients with a mean pH of 7.07 and a median ISS of 29 on admission, the mortality following the utilization of this technique was 23%. All patients surviving to chest closure were physiologically normal at the time of definitive operation, which on average was 2 to 3 days after the original emergency operation.[156]

Video-Assisted Thoracoscopic Surgery

Increasing experience with minimally invasive techniques has contributed to enthusiasm for VATS for a variety of sequelae of trauma.[146,147] As a diagnostic tool, VATS remains an acceptable alternative to laparoscopy to identify and repair penetrating diaphragmatic injuries. Persistent air leak, retained hemothorax, and, in selected patients, decortication for empyema are recognized indications for VATS. VATS is not, however, an emergent modality; emergent exploration for hemorrhage or severe parenchymal trauma should be performed using an open operative approach.

VATS is performed in the operating room under general anesthesia, with lung isolation achieved with a double-lumen endotracheal tube. Lung isolation provides superior exposure and an operative field with good visualization. The procedure is performed with the patient in the full lateral decubitus position with the affected side up. The operative field should be widely prepped and draped to facilitate conversion to a thoracotomy if indicated.

On single-lung ventilation, the first port is placed in the fourth or fifth intercostal space in the mid- or anterior axillary line. The tip of the scapula serves as a convenient landmark to facilitate appropriate positioning (Fig. 28-16). An angled thoracoscope is preferred for initial use, as it improves visualization of the pleural space recesses. An aspiration catheter can be placed coaxially to the optical port to facilitate the lavage and evacuation required for initial visualization. Additional ports can then be developed under direct visualization to address the pathology encountered.

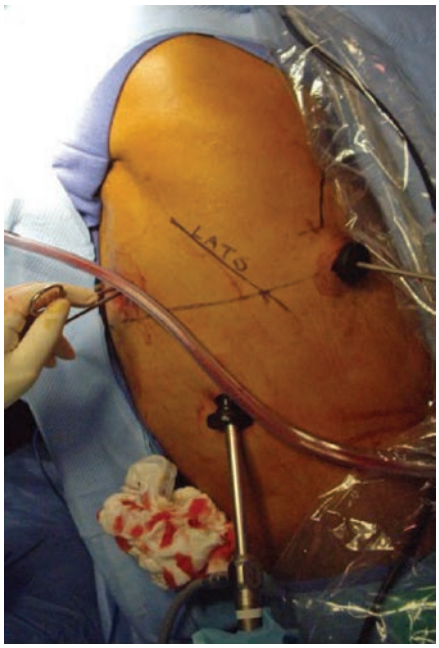


FIGURE 28-16 Landmarks for video-assisted thoracoscopic surgery port placement.

The instruments used for VATS are similar to those for laparoscopic procedures. Conventional open surgery forceps can also be used. Cautery, however, should be used cautiously and in close coordination with anesthesia, as oxygen-rich air leaks and cautery may interact to create a fire hazard with catastrophic results. On completion, the chest is irrigated with normal saline or sterile water. Using the existing port sites, chest tubes are positioned under direct visualization, and the lung reexpanded prior to closure. Following the procedure, a chest x-ray should be obtained and the thoracostomy tubes managed as they would be after a thoracotomy.

Outcomes

There is wide variability in the reported mortality after thoracic injury. With major blunt trauma, a mortality as high as 68% has been reported.[1,2] This is probably related to higher ISS, lower Glasgow Coma Scale score, and more associated nonthoracic injuries than in patients with penetrating injuries.[3,157] There is also variation in the reported mortality with penetrating trauma. Cardiac and major thoracic vascular injuries and the percentage of major pulmonary resections all contribute to poorer outcomes[3] (see Chapter 30). A relationship between the magnitude of the pulmonary resection and subsequent mortality has been well demonstrated.[3]

Complications of Injury to the Lung

PNEUMONIA

Pneumonia is the most common significant complication following pulmonary injury, and the relative risk is closely associated with the need for mechanical ventilation. Following thoracic injury, patients requiring intubation are approximately seven times more likely to develop pneumonia than those who do not.[1] Of all patients admitted with the diagnosis of pulmonary contusion (Fig. 25-13), nearly 50% will develop pneumonia, barotrauma, and/or major atelectasis, and 25% will go on to develop acute respiratory distress syndrome.[1]

RETAINED HEMOTHORAX

It has been estimated that tube thoracostomy fails to completely evacuate a hemothorax in over 5% of patients.[158] Typically, small hemothoraces will be reabsorbed; however, posttraumatic empyema and, less commonly, fibrothorax with an entrapped lung are known sequelae of a retained hemothorax, as previously noted.[134,158,159]

The diagnosis of retained hemothorax requires a chest CT because plain chest x-ray has been shown to be inadequate in effectively characterizing this entity.[160] There is wide variability, even among trauma centers, regarding the treatment of retained hemothoraces. Many patients require more than one procedure to evacuate the pleural space. In general, CT-estimated retained hemothorax volumes less than 300 mL can be safely observed in the absence of infection, whereas those greater than 300 mL will likely benefit from evacuation.[118] The timing of VATS has been somewhat controversial, with early studies reporting optimal results when the retained hemothorax was evacuated within the first few days following injury.[147,161] A more recent multicenter study found no significant impact of the timing of VATS and successful evacuation of the pleural space.[48]

EMPYEMA

Empyema, an infection of the pleural space, is diagnosed by positive pleural cultures or frank purulence. Overwhelmingly, the most common cause of posttraumatic empyema is a retained hemothorax, with both postpulmonary resection and postpneumonic etiologies much less common. A recent large multicenter study reported that empyema developed in 26.8% of patients with a retained traumatic hemothorax.[159]

Empyema has been characterized by three often overlapping stages, including exudative, fibrinopurulent, and organizing. Although most early-stage postpneumonic empyemas can be successfully treated by thoracostomy tube drainage and antibiotics, this is not the case for posttraumatic empyema. Delay in evacuating a retained hemothorax and an appreciable inflammatory response with resultant loculations make simple tube thoracostomy inadequate treatment. Both VATS and thoracotomy are acceptable modalities, with VATS more successful in the earlier stages and thoracotomy best for later-stage empyema or failed initial therapy. In a large, single-institution series of 125 consecutive patients with posttraumatic empyema, VATS and thoracotomy were performed in 20% and 80% of patients, respectively. Mortality was 4% and associated with ruptured lung abscesses.[161] It is important to note that failure of the first intervention to treat empyema is an independent predictor of mortality.[162] Therefore, it is essential that thoughtful judgment is exercised when considering treatment options.

PERSISTENT AIR LEAK AND BRONCHOPLEURAL FISTULA

A true bronchopleural fistula is a centrally located communication between a lobar or segmental bronchus and the pleural cavity. This specific complication is uncommon following trauma but may occur with an injury to a major bronchus or following pulmonary resection for a lung injury. Most posttraumatic air leaks are actually communications from the lung parenchyma to the pleural space and are more accurately termed *parenchymal–pleural* or *alveolar–pleural fistula*. Traditionally, however, bronchopleural fistula refers to any air leak from the lung to the thoracic cavity.

Although there is no agreed upon definition of what constitutes a "persistent" air leak, it is generally a leak that continues beyond 5 to 7 days.[163] This entity can be challenging to manage, especially in the ventilated patient in whom large leaks may result in loss of effective tidal volume and, consequently, hypoxia and/or hypercarbia. The diagnosis is usually not subtle, with persistent, vigorous air bubbling through the water-seal chamber of the collection system of the thoracostomy tube. Bronchoscopy should be performed if there is concern for an associated major airway injury.

Although the vast majority of air leaks will resolve within 7 days, those that persist will require treatment. The management of the air leak is complicated if the patient is on mechanical ventilation, in which case safely minimizing the mean and end-inspiratory plateau pressure is a useful strategy. Autologous blood pleurodesis, various commercially available sealants, endobronchial one-way valves, Heimlich valves, and operative therapy are among the management options to treat a persistent air leak.[164]

CHYLOTHORAX

A primary traumatic chylothorax, characterized by milky chest tube output, is uncommon after traumatic injury or surgery. The diagnosis is established by analyzing the content of the effusion and documenting the presence of fat (triglyceride levels >110 mg/dL) with or without predominant lymphocytes in the effusion. The primary complications of chylothorax are nutritional depletion, electrolyte abnormalities, and compromised immune function. Nonoperative management includes lung expansion to promote tamponade, total parenteral nutrition, enteral medium-chain triglycerides, and octreotide. Persistent chylous drainage for 5 to 7 days is a failure of nonoperative management. Although there are a few reports of successful embolization of the thoracic duct, direct ligation following lymphangiographic localization is the preferred approach.[165,166]

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29

Trachea, Bronchi, and Esophagus

Joseph J. DuBose • Thomas M. Scalea • James V. O'Connor

KEY POINTS

Injury to Trachea and Bronchi

- Direct laryngoscopy, video laryngoscopy, bronchoscopic-guided intubation, and a surgical airway are all methods to establish a secure airway in a patient with a tracheal injury.
- The classic presentation of a distal tracheal or proximal bronchial injury is a large pneumothorax and a significant air leak and/or persistent pneumothorax following insertion of a thoracostomy tube.
- The distal half of the trachea, the right mainstem bronchus, and the proximal left mainstem bronchus are best approached through a right posterolateral thoracotomy.
- During repair of a mainstem bronchus, a single-lumen endotracheal tube in the contralateral mainstem bronchus allows for ventilation and an easier repair.
- Tracheal repair is accomplished with interrupted absorbable sutures.

Injury to Esophagus

- The accuracy of combining a contrast esophagram and esophagoscopy approaches 100%.
- As opposed to the trachea, the blood supply to the esophagus courses longitudinally in the submucosa, allowing for full mobilization.
- The extent of the mucosal defect is exposed by incising the muscular layer until both ends of the mucosal tear are visualized.
- A tension-free esophageal repair is performed in two layers, with absorbable sutures on the mucosa and permanent interrupted sutures on the muscular layer.
- A vascularized intercostal muscle pedicle is ideal to buttress a repair of the thoracic esophagus.

INTRODUCTION

Thoracic trauma can result in a variety of clinical entities that demand thoughtful management in order to achieve optimal outcome. The bony thorax, major vascular structures, esophagus, trachea, and lung are all vital structures at risk following injury to the chest. Even potential spaces between these structures can manifest serious acute pathologies, such as a pneumothorax or hemothorax, that require knowledge of both emergent and less acute management considerations. In an effort to optimally address each of these traumatic pathologies most effectively, the editors of *Trauma* have thoughtfully distributed their discussion across several chapters constructed by experts. The focus of this chapter will be injuries specifically to the trachea, bronchi, and esophagus. The other aforementioned entities, including the thoracic wall, pneumothorax, and hemothorax, are discussed elsewhere (see Chapter 28).

INJURIES TO THE TRACHEA AND BRONCHI

Tracheobronchial injuries are infrequent but potentially life threatening. The incidence of blunt and penetrating injury is 0.4% and 4.5%, respectively.[1] Although these injuries occur more commonly in the cervical trachea and may be the result of either mechanism, thoracic tracheobronchial trauma generally is related to blunt injuries from high-speed motor vehicle crashes.[2-6] Most penetrating injuries to the cervical trachea, although straightforward to repair, may be associated with significant injuries to blood vessels, esophagus, and thoracic duct.[7]

Presentation and Evaluation

Cervical tracheal injuries are often obvious on physical examination.[8] A large volume of subcutaneous air and/or air

exiting from the missile tract may be observed. Patients often present in respiratory distress requiring urgent airway control. Direct laryngoscopy, video laryngoscopy, bronchoscopic guided intubation, and a surgical airway are all methods to establish a secure airway. The choice of technique depends on the specific injury, impending loss of airway, and the skill of the providers. Extreme care must be taken when intubating, because a partial tracheal injury can be converted to total circumferential disruption. The resultant loss of airway can lead to a respiratory arrest and, if the airway cannot be secured, death. Similarly, blindly placing an airway through a cervical wound with air escaping through it is treacherous. The airway may inadvertently be inserted into the mediastinum with catastrophic results. To avoid this, the trachea must be visualized and controlled and an endotracheal or tracheostomy tube inserted under direct vison. The classic presentation of a distal tracheal or proximal bronchial injury is a very large pneumothorax, a large air leak through a thoracostomy tube, and/or persistent pneumothorax despite the presence of a tube.

Patients with a suspected tracheobronchial injury must undergo emergent bronchoscopy (Fig. 29-1). Flexible fiberoptic bronchoscopy is the technique most often used. To achieve an unobstructed view of the mucosa and perform a thorough bronchoscopic examination, it is essential to clear the airway of blood and secretions. More than three-quarters of blunt tracheobronchial injuries occur within 2 cm of the carina.[6]

The entire circumference of the trachea must be examined. If the cervical trachea is at risk and the patient is intubated, the endotracheal tube may have been inserted distal to the suspected injury. In these cases, the endotracheal tube should be carefully withdrawn, keeping the bronchoscope distal to the endotracheal tube to ensure control of the airway. When the examination is complete, the endotracheal tube can be advanced distally over the bronchoscope and secured.

As computed tomography (CT) scanning has evolved, it is now used in the evaluation of potential tracheal injuries.[8] In the case of penetrating injury, a trajectory that is clearly remote from the trachea should effectively rule out the possibility of a tracheal injury. Three-dimensional reformatting of the images may demonstrate a tracheal injury, although a negative study does not effectively rule out the presence of an injury.[9-11] Bronchoscopy remains the diagnostic modality of choice. It defines the location and extent of the injury and should be performed by the operative surgeon.

Operative Exposure and Repair

As noted earlier, the management of a tracheobronchial injury begins with establishing a secure airway using the techniques listed.[9,12] The location and extent of the airway injury must then be completely characterized and associated injuries evaluated.

Intraoperative airway management necessitates close cooperation between the anesthesiologist and the surgeon. An injury to the cervical trachea can be managed with a single-lumen endotracheal tube. If necessary, the airway can be intubated across the operative field (Fig. 29-2). The surgical repair is facilitated by maintaining the mean airway pressure as low as possible, consistent with adequate oxygenation and ventilation. Brief periods of apnea afford improved visualization of the operative site. Intraoperative airway

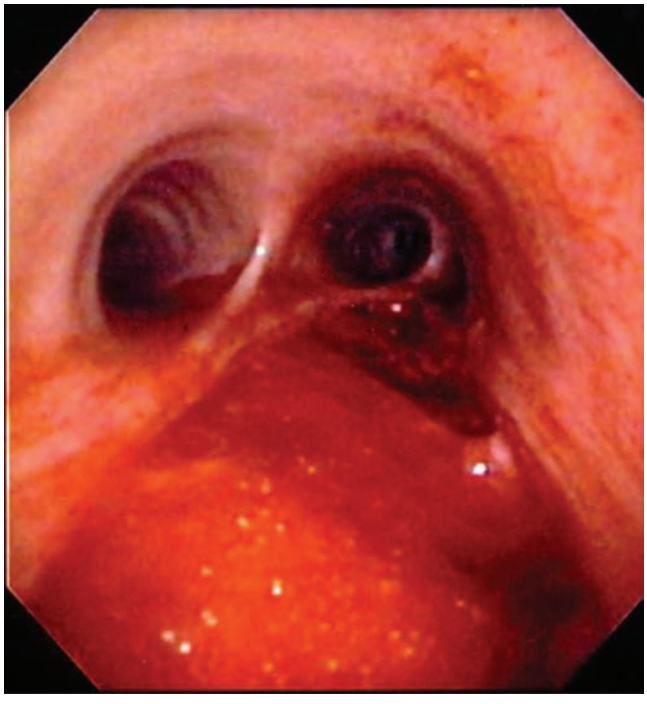

FIGURE 29-1 Tracheal injury visualized with preoperative bronchoscopy.

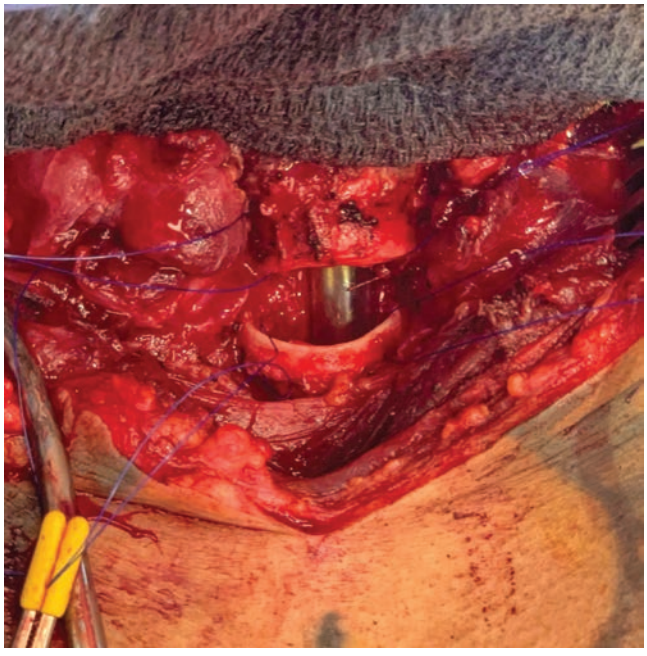

FIGURE 29-2 Tracheal injury with stay sutures placed to control edges and endotracheal tube distally advanced.

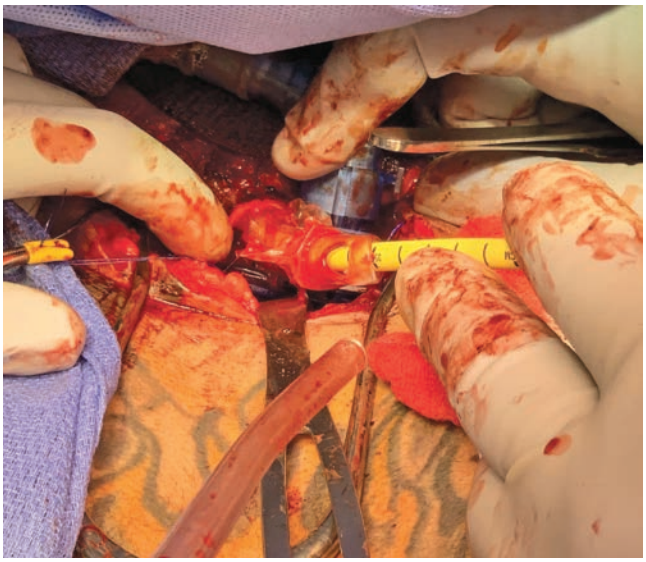

FIGURE 29-3 Retrograde placement of bougie cannula through oropharynx, facilitating advancement of endotracheal tube.

management is further complicated when an emergent surgical airway was placed through the tracheal injury and oral or nasal intubation is not possible. In this rare circumstance, a bougie is passed retrograde through the tracheal defect into the oral cavity, and an endotracheal tube is inserted over the bougie and guided into the distal trachea (Fig. 29-3)

Although operative repair is the mainstay of treatment for tracheobronchial injuries, there is a limited role for both stent placement and nonoperative management.[12-14] Conservative management is reserved for highly selected patients with small (<2 cm), linear, nontransmural tears.[15]

Appropriate tracheal exposure is determined by the anatomic location of the airway injury. The arterial blood supply to the trachea is segmental arising from the inferior thyroid and bronchial vessels, and the main vessels are at 3 and 9 o'clock. The proximal half of the trachea can be exposed through a collar incision, blunt dissection in the pretracheal plane will facilitate mobilization, and a partial sternotomy is needed rarely (Fig. 29-4).

The distal half of the trachea, the right mainstem bronchus, and the proximal left mainstem bronchus are best approached through a right posterolateral thoracotomy. A double-lumen endotracheal tube will greatly enhance operative exposure with repair of a mainstem bronchus. Another option is a single-lumen endotracheal tube in the contralateral mainstem bronchus (Fig. 29-5). Widely opening the mediastinal pleura and doubly ligating and dividing the azygous vein will provide the desired exposure. If an esophageal injury has been excluded, placing an esophageal bougie or nasogastric tube will facilitate dissection in the plane between the esophagus and trachea. The distal left mainstem bronchus is approached through a left posterolateral thoracotomy. This approach, while necessary, is complicated by the aortic arch.

Tracheal repair is accomplished with interrupted absorbable sutures. Simple lacerations are closed primarily, whereas

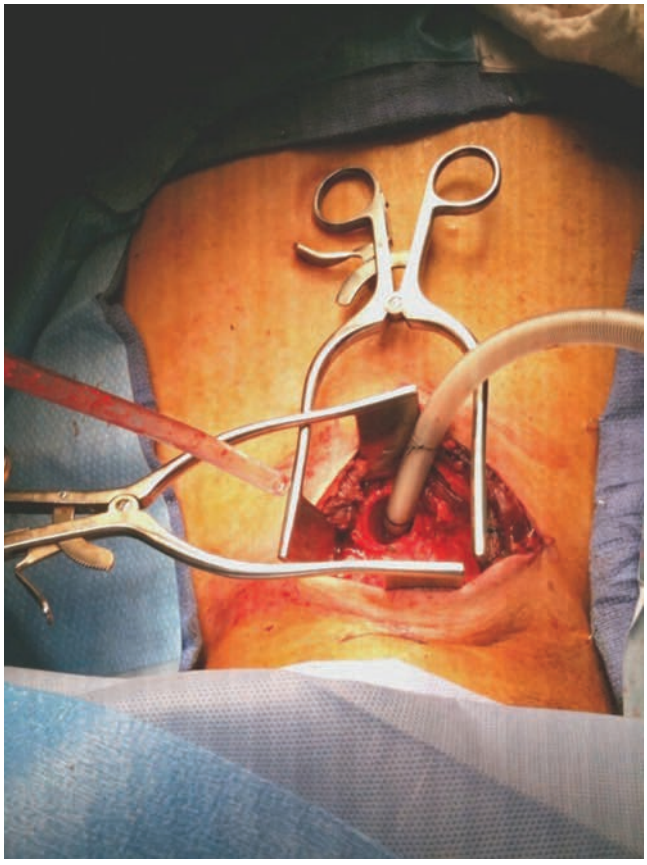

FIGURE 29-4 Tracheal intubation on the operative field. Partial sternotomy was chosen to obtain control of the great vessels.

more extensive injuries may require some minimal debridement to healthy tissue prior to repair. Extensive circumferential injures necessitate an end-to-end anastomosis, while preserving the blood supply to avoid suture line ischemia. This is always performed at a reoperation. If necessary, additional length can be achieved by mobilizing the trachea by blunt dissection in the avascular pretracheal plane. Other maneuvers such as a laryngeal release are rarely needed. In extreme circumstances, approximately half of the length of the trachea can be resected and a primary end-to-end anastomosis performed. Care must be exercised when placing the sutures, avoiding the endotracheal tube and its cuff. Lower tidal volume ventilation and brief periods of apnea will allow precise suture placement. After the tracheal repair is completed and before the thorax is closed, it is wise to have the anesthesiologist ensure the endotracheal tube is movable, confirming the endotracheal tube has not been inadvertently caught by a suture. Ideally, patients are extubated in the operating room to highly humidified supplemental oxygen.

A concomitant vascular or esophageal injury should be primarily repaired. The presence of suture lines in apposition increases the possibility of a fistula. The risk of this dreaded complication is reduced by interposing a vascularized muscle between the suture lines. In the neck, the sternal head

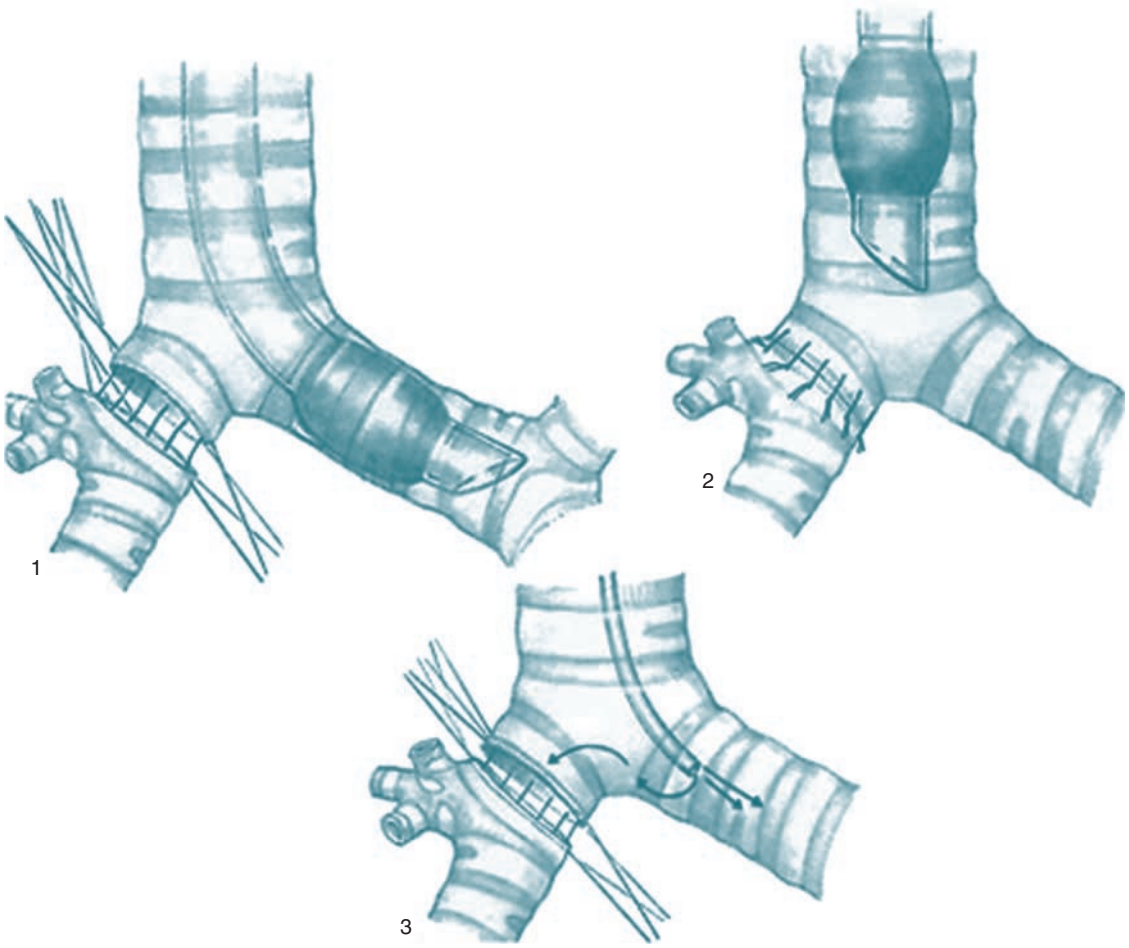

FIGURE 29-5 Intraoperative airway management using a single-lumen endotracheal tube during repair of a ruptured mainstem bronchus. (Reproduced with permission from Hood RM. *Thoracic Surgery: Techniques in General Thoracic Surgery*. Philadelphia, PA: WB Saunders Company; 1985. Copyright © Elsevier.)

or detached sternocleidomastoid muscle is ideal, whereas an intercostal muscle pedicle is ideal in the chest.

Infrequently, tracheobronchial injures are complicated by severe respiratory failure on presentation, which may be refractory to aggressive, conventional ventilator management. In these instances, immediate operation on a patient with profound hypoxia and/or hypercarbia would be fatal. Venovenous extracorporeal membrane oxygenation can provide optimal support during surgical repair.[16]

Outcomes

Mortality ranges from 0% to over 40%, with most deaths the result of associated injuries.[7,9,12,17] Complications are common, occurring in approximately 25% of patients, and are divided into short and long term.[9,12,17] Sepsis, pneumonia, and respiratory failure are among the common short-term complications encountered.

The major long-term complications are tracheal stenosis, abnormal phonation, and vocal cord paralysis. Laryngeal injuries and injury to the recurrent laryngeal nerve, from the initial injury or during the operative repair, commonly result in dysphonia. Tracheobronchial stenosis is an uncommon but potentially devastating complication, and initial symptoms may be underappreciated. Wheezing from air moving through the stenotic airway may be attributed to reactive airway diseases such as asthma or chronic obstructive pulmonary disease. Any patient presenting with new respiratory symptoms following tracheobronchial repair must be evaluated for airway stenosis. Although a detailed history, physical examination, pulmonary function tests, flow-volume loops, CT imaging, and bronchoscopy are each important in establishing the diagnosis, bronchoscopy is essential. It identifies the severity, location, and extent of the stenosis. Once the diagnosis is confirmed, initial management consists of controlling the airway. This is crucial in patients with high-grade lesions. Subsequent therapy is rigid or balloon dilation, which should be performed under direct visualization. Surgical treatment for postrepair airway obstruction is typically reserved for severe and relatively short-segment lesions.

The surgical treatment for posttraumatic stenosis varies by location of the stenosis but most commonly consists of resection and end-to-end anastomosis or tracheal sleeve resection.

INJURIES TO THE ESOPHAGUS

Esophageal injuries, although rare, are nonetheless serious, especially when they involve the thoracic esophagus. Blunt esophageal injuries are extremely rare but may occur as the result of a direct blow against a hyperextended neck or overpressure of the esophageal lumen.[18] Due to the protection of the bony thorax, esophageal injuries are much more common in the neck than the thorax. Overwhelmingly, esophageal injuries are the result of penetrating trauma.[19-22] A large, multicenter study of penetrating esophageal injuries was conducted by the American Association for the Surgery of Trauma (AAST) and involved 34 trauma centers in the United States. Penetrating esophageal injuries were identified in 405 patients over a span of 10.5 years, and 98% had associated injuries.[19] This high percentage of associated injuries is similar to other series.[22]

Presentation and Evaluation

Patients with an injury pattern or trajectory adjacent to the esophagus require evaluation. Although physical examination may be helpful in diagnosing an injury to the cervical esophagus, it is rarely useful for an intrathoracic injury. Saliva exiting a neck wound confirms the diagnosis of an injury to the cervical esophagus. Unfortunately, physical findings are often nonspecific, occurring in less than 25% of patients.[13] A patient with an injury to the thoracic esophagus may present with a concomitant pneumothorax or hemothorax from an associated injury to the lung and/or vascular structures. Drainage from a thoracostomy tube with obvious saliva or food particles warrants urgent esophageal evaluation.[19,20,23,24]

CT scanning has become more widely used in the evaluation of penetrating thoracic injuries, because it will define the missile trajectory to determine if the esophagus is at risk. If oral contrast has been administered, CT may identify extraluminal extravasation. The ability to obtain three-dimensional mediastinal imaging makes CT more attractive than a simple contrast study. CT is a static examination, however, and cannot assess the passage of contrast moving through the esophagus, as with an esophagram.

A contrast esophagram has traditionally been used to evaluate both the cervical and the thoracic esophagus. If correctly performed, the entire esophagus can be evaluated. In intubated patients, contrast can be instilled via a nasogastric tube that is pulled back so the tip rests in the proximal esophagus. Contrast studies may, however, miss esophageal injuries, and obtaining high-quality contrast studies may be difficult. Esophagoscopy is an excellent study that allows direct visualization of the location and extent of an esophageal injury. It has a negative predictive value of 100% but a positive predictive value of only approximately 33%.[24-26] The accuracy of combining an esophagoscopy and a contrast study approaches 100%.[24] Performing esophagoscopy first avoids contrast obscuring the esophageal mucosa.

Operative Exposure and Repair

Because esophageal injury is uncommon, most single-institution studies include only a small numbers of trauma patients for analysis.[25-33] Larger published series are either multicenter studies or use large data banks.[20-27] Iatrogenic esophageal injuries are quite different than those due to external trauma, are typically located in the cervical esophagus, and are commonly successfully managed nonoperatively. Conversely, surgical repair remains the mainstay for noniatrogenic esophageal injuries.

The cervical esophagus is exposed through an incision along the anterior border of the left sternocleidomastoid muscle. Lateral retraction of this muscle and blunt dissection in the avascular prevertebral plane will allow a Penrose drain to encircle the esophagus, achieving 360° exposure. As opposed to the trachea, the blood supply to the esophagus courses longitudinally in the submucosa, allowing for full mobilization. During dissection, care must be exercised to avoid injuring the recurrent laryngeal nerves that lie in the tracheoesophageal grove.

It is important to remember that the esophagus lacks a serosal layer and, therefore, is more prone to a postoperative leak. If this occurs in the cervical esophagus, it is easily managed without serious complications. A leak from a repair of the thoracic esophagus in the mediastinum, however, can cause significant morbidity and mortality. A fundamental principle in repairing esophageal injuries is visualizing the entire extent of the mucosal injury. The defect in the muscular layer is almost always less extensive than that in the mucosa. The extent of the mucosal defect is exposed by incising the muscular layer until both ends of the mucosal tear are visualized. A tension-free esophageal repair is performed in two layers—the mucosa is approximated with interrupted sutures, either absorbable or nonabsorbable, and the muscular layer is closed with interrupted nonabsorbable sutures (Fig. 29-6). The repair can be facilitated by placing a nasogastric tube, and the area is widely drained.

Intrathoracic esophageal injuries require more extensive exposure. The operative principles are preservation of esophageal length, primary repair, buttressing the repair, wide drainage, antibiotics, and enteral feeding access. Preoperative localization of the site of injury is imperative, because it influences the surgical approach. With the exception of its most distal segment, the intrathoracic esophagus is approached through a right posterolateral thoracotomy. The azygous vein is doubly ligated and divided, and the lung is retracted. The mediastinal pleura is widely opened to expose the esophagus, which is mobilized by blunt dissection. Devitalized mediastinal and esophageal tissues are debrided. A vascularized intercostal muscle pedicle is ideal to buttress the repair because it is robust and easily harvested[24] (Fig. 29-7).

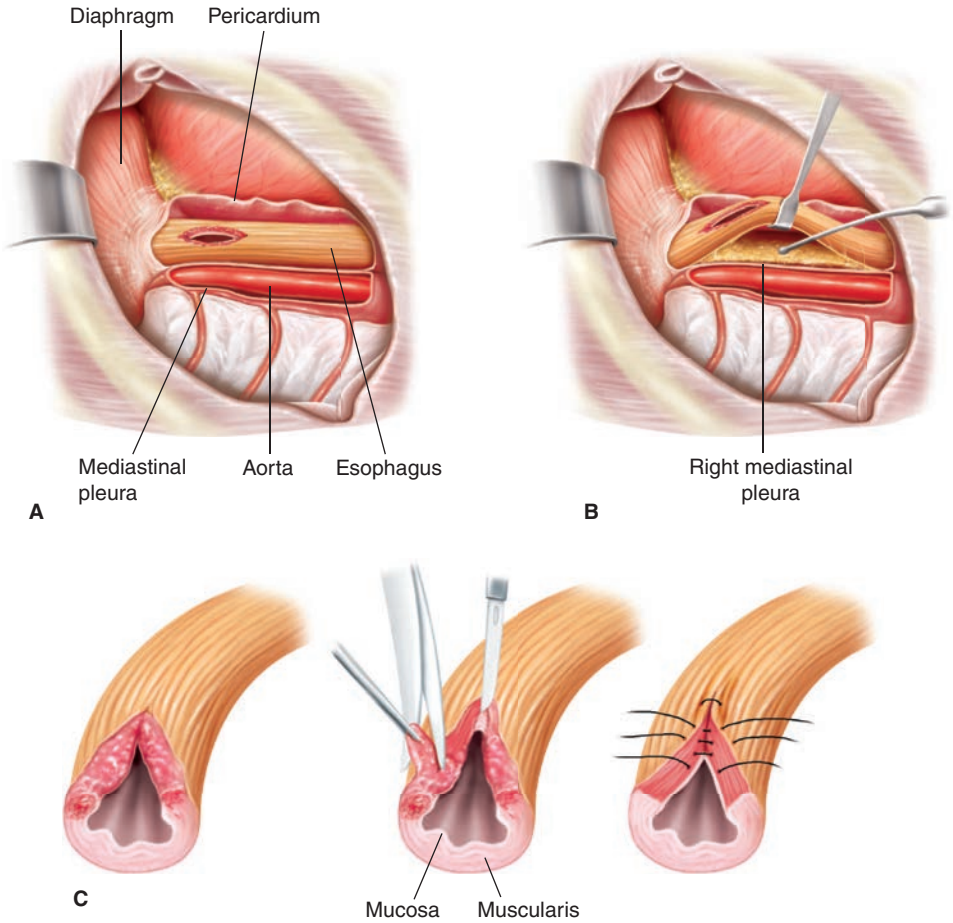

FIGURE 29-6 (**A**) Necrotic mediastinal pleura has been excised, and esophageal tear has been debrided. (**B**) Elevation of the esophagus on a rubber drain allows for debridement of the right mediastinal pleura if indicated. (**C**) Debridement of the esophageal rupture. Muscularis is incised superiorly and inferiorly to allow visualization of the extent of mucosal defect before two-layer closure of the perforation if possible. (Reproduced with permission from Patterson GA, ed. *Pearson's Thoracic and Esophageal Surgery*. Vol. 2. 3rd ed. Philadelphia, PA: Elsevier; 2008. Copyright © Elsevier.)

Wide mediastinal drainage is mandatory, and distal enteral feeding access is placed. A contrast study is performed approximately 1 week postoperatively, and if no leak is noted, oral feedings are started.

There are several surgical approaches to the distal esophagus and esophagogastric junction including a left sixth or seventh interspace posterolateral thoracotomy, a laparotomy, or a thoracoabdominal approach. Injuries to the distal intrathoracic esophagus are best approached via thoracotomy. The choice of incision to expose the esophagogastric junction is influenced by the exact location of the injury and associated injuries. If there are concomitant abdominal injuries, laparotomy alone may be sufficient, whereas associated intrathoracic injuries may require thoracotomy or thoracoabdominal incision. Distal esophageal injuries lend themselves to reinforcement with a fundal wrap.

Regardless of the location, a primary buttressed repair and adequate mediastinal drainage are the optimal treatment. Esophageal excision and resection with diversion should be avoided, and every effort should be made to preserve esophageal length. In rare circumstances, the magnitude of the esophageal injury or the patient's clinical condition precludes definitive repair. In these instances, a damage control procedure may be lifesaving. Creating a controlled esophageal fistula by using a surgeon-created large T-tube is an effective option.[34] This technique, combined with wide drainage, controls mediastinal contamination and preserves esophageal length. A devastating injury to the stomach and esophagogastric junction presents a unique challenge. These are not amenable to T-tube drainage; however, retrograde esophageal drainage after resection may prove useful. Continuity is reestablished by an esophagojejunostomy performed several months later.[34,35]

Endoluminal Stents

The use of endoluminal esophageal stents for the management of benign esophageal perforation has increased significantly over the past several years. Several investigators have examined their limited experience using stents for esophageal perforations for both iatrogenic and trauma indications.[36-40]

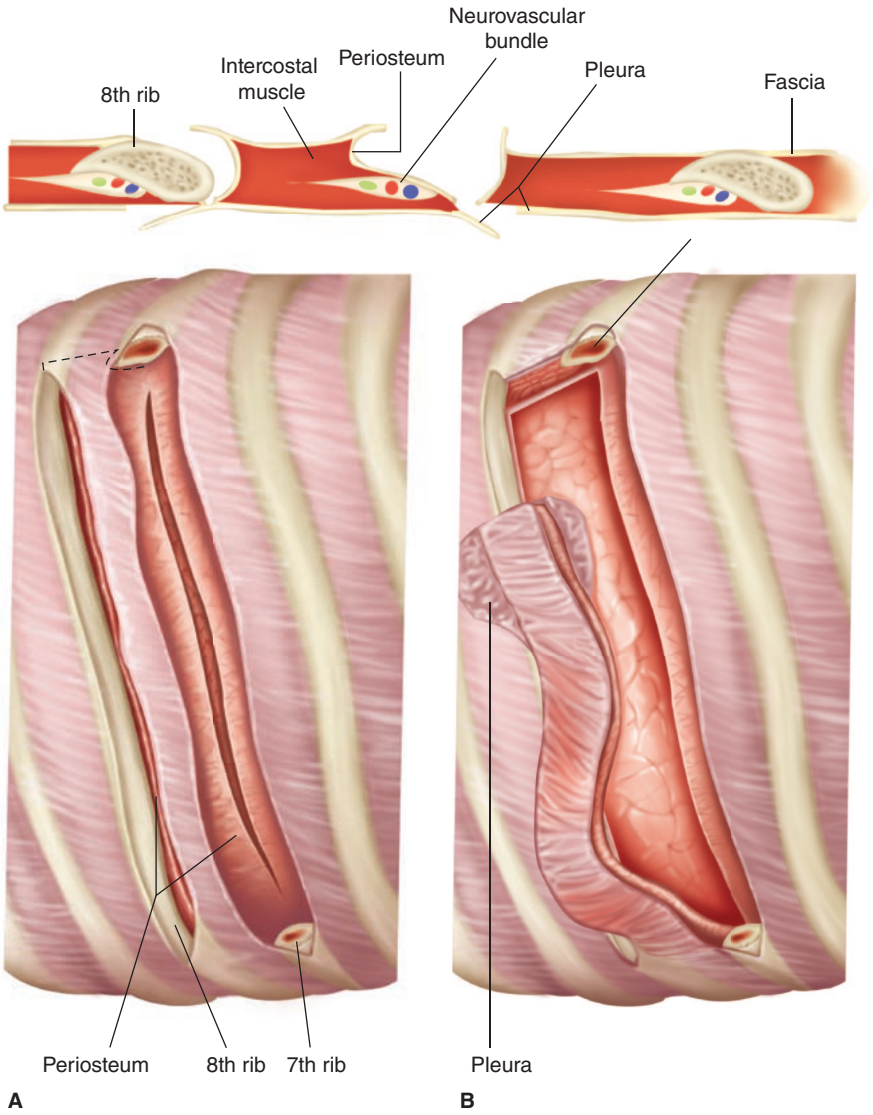

FIGURE 29-7 Construction of intercostal musculopleural flap. (**A**) Periosteum of the rib inferior to thoracotomy incision is incised, and the subjacent pleura is mobilized. (**B**) The neurovascular bundle is divided anteriorly, and the flap is created. (Reproduced with permission from Patterson GA, ed. *Pearson's Thoracic and Esophageal Surgery.* Vol. 2. 3rd ed. Philadelphia, PA: Elsevier; 2008. Copyright © Elsevier.)

Data for iatrogenic esophageal injuries suggest that covered stents are associated with similar effectiveness, but have lower morbidity, shorter hospital stay, and lower costs when compared to surgery.[36] An appropriate study of the use of this modality among trauma patients has yet to be conducted. However, a stent may represent a suitable alternative for damage control because it will limit ongoing contamination due to a persistent esophageal leak.

Outcomes

Esophageal trauma results in significant morbidity and mortality. Mortality ranges from 0% to 44% and is related to delays in treatment, associated injuries, and esophageal resection rather than primary repair.[27,41,42] In the large AAST series, the mortality was 19%.[19] Complications are common, with esophageal-related complications ranging between 38% and 66%.[31,41,42] Independent risk factors for these complications include delays in operative treatment, higher grade esophageal injury, and resection and diversion.[41,42] Prompt diagnosis and operation, a precise technical repair, and consideration of a damage control procedure in dire circumstances are essential for a satisfactory outcome.

In the previously mentioned largest study to date of penetrating esophageal injuries, Asensio et al[19] found that patients subjected to lengthy evaluations had higher complication rates overall and higher esophageal-related complications specifically, compared to counterparts who proceeded directly to operation for diagnosis and received operative treatment in an expedited fashion. This finding has been confirmed by

other investigators and emphasizes the need for expeditious evaluation and definitive management.[21,22]

NONMALIGNANT TRACHEOESOPHAGEAL FISTULA

An acquired, nonmalignant tracheoesophageal fistula is a dreaded, but rare, complication. Contemporary endotracheal tubes with high-volume, low-pressure cuffs have virtually eliminated this dreaded complication among ventilated patients. Combined airway and esophageal injuries repaired without muscle interposition, however, are at risk to develop a tracheoesophageal fistula. The clinical presentation initially may be subtle, with symptoms ascribed to a respiratory problem. Recurrent pneumonia and a persistent cough with an unidentified cause mandate an evaluation to rule out this complication. Once diagnosed, treatment commonly requires surgical intervention, with division of the fistula, repair of the respective tracheal and esophageal defects, and interposition with a well-vascularized muscle pedicle. Ideally, this is accomplished once the patient is liberated from mechanical ventilation, as the operative repair is more complex on mechanical support. The use of covered tracheal or esophageal stents has been described for patients who are not operative candidates, as has operative diversion.[36-40] Avoiding rather than treating this complication is the preferred strategy. As described earlier, with a combined airway and esophageal injury, a precise technical repair of each individual structure and muscle interposition will greatly reduce the chance of this complication.[41] The same strategy is used with a combined esophageal and vascular injury. Each undergoes primary repair, and a muscle buttress is placed between the suture lines. Unlike a tracheoesophageal fistula, the presentation of a carotid artery–esophageal fistula with acute hemorrhage can lead to death in the unattended patient.

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Heart and Thoracic Vessels

Matthew J. Wall Jr • Ravi K. Ghanta • Kenneth L. Mattox

KEY POINTS

Injury to the Heart

- Avoid overaggressive resuscitation with crystalloid solutions in patients with suspected cardiac/vascular injuries.
- The pericardial view of the focused assessment with sonography for trauma examination provides a rapid and accurate diagnosis of a hemopericardium.
- The majority of penetrating wounds to the heart can be managed with suture repair of the myocardium.
- An injury adjacent to a coronary artery is repaired with deep mattress sutures passing underneath the vessel.
- Complex cardiac injuries involving the cardiac valves or the atrial/ventricular septa are extremely uncommon and are usually repaired with cardiopulmonary bypass in a subacute manner.
- Consider pericardial tears and possible cardiac herniation with high-energy lateral blunt trauma and posttrauma positional hypotension.
- Screening for blunt cardiac injury involves history and physical examination, electrocardiogram, and in some centers, measurement of troponin I.

Injury to the Thoracic Vessels

- Contrast-enhanced computed tomography (CT) of the chest can be a useful screening test for stable patients with the following injuries: penetrating wound with mediastinal traverse; any thoracic trauma and mediastinal or supraclavicular hematoma; and blunt trauma with symptoms or signs or x-ray findings of an injury to the descending thoracic aorta.
- Anatomic and physiologic classification of blunt injury to the thoracic aorta guides therapy, particularly recognizing that small, stable injuries can be observed.
- The management of blunt injury to the descending thoracic aorta has evolved, with CT of the chest the most common screening test, endovascular repair the most common technique of repair, and delayed repair often employed.
- For endovascular repair of blunt injury to the descending thoracic aorta, it is important to understand issues of room setup, vascular access, device sizing, seal zones, injury configuration, and vascular anomalies.
- Many patients with penetrating thoracic vascular injuries are unstable and have the diagnosis made during an emergent exploration, usually via a left anterolateral thoracotomy, bilateral anterolateral thoracotomy, or less commonly, a median sternotomy.
- Endovascular repair of thoracic outlet vascular injuries performed in an operating room with imaging capability permits the application of this technology to complex and unstable patients. The endovascular snare technique allows through-and-through access for even transected vessels permitting nonsurgical repair of these complex injuries.

INTRODUCTION

The heart, the thoracic aorta, and its tributaries are enclosed in the thoracic skeleton composed of the manubrium, sternum, clavicles, rib cage, and vertebral bodies. This rigid cage protects the heart, lungs, and great vessels. The bony structures can also create unique forms of injuries as they cause deflection of bullets, altering vectors of the original direction of penetration or by secondary fragments.

Blunt forces can lead to crushing, traction, or torsion injuries to the heart and great vessels from deceleration. Penetrating trauma to the heart and great vessels usually leads to immediate tamponade or exsanguination but may follow a

pattern of injury similar to blunt trauma, including partial transection with formation of a traumatic false aneurysm, an intimal flap, thrombosis, or a rare arteriovenous fistula.

There has been a profound shift in the management of thoracic vascular injuries over the past two decades. Improvements in imaging technology have resulted in a shift from plain films and catheter angiography to computed tomography (CT) imaging for screening and diagnosis. In addition, advances in endovascular therapy have changed the way these injuries are managed.

INJURY TO THE HEART

Incidence

Penetrating cardiac trauma is a highly lethal injury, with only 25% to 50% of victims surviving long enough to reach the hospital in reviews from medical examiners; however, in one extraordinary series of 1198 patients with penetrating cardiac injuries in South Africa, only 6% of patients reached the hospital with any signs of life.[1]

With improvements in organized emergency medical transport systems, more patients with a significant cardiac injury reach the emergency department with signs of life in the modern era. It is somewhat frustrating, however, to note the overall mortality for penetrating trauma has not changed significantly, even in the major trauma centers.[2]

The actual incidence of blunt cardiac injury (BCI) is unknown. Because thoracic trauma has historically been responsible for 25% of deaths from motor vehicle crashes, it is possible that 10% to 70% of this subgroup may have been the result of blunt cardiac rupture.[3] There has been confusion historically, as the term *cardiac contusion* has been applied to a wide spectrum of pathology, making epidemiology of BCI difficult to define.

Mechanism

PENETRATING CARDIAC INJURY

Penetrating trauma is a common mechanism for cardiac injury, with the predominant etiologies being from knives and firearms (Table 30-1). In a review of 711 patients with penetrating cardiac trauma, 54% sustained stab wounds and 42% had gunshot wounds. Because of an anterior and left lateral location, the cardiac chambers at greatest risk for injury are the right and left ventricles. The right ventricle was injured in 40% of patients, the left ventricle in 40%, and the right atrium in 24%. With its posterior location, the left atrium was injured in only 3% of patients. The overall mortality was 47%, and one-third of injuries involved multiple cardiac structures.[4] More complicated intracardiac injuries involving the coronary arteries, the cardiac valves, and septae were noted as well. Septal injuries result in intracardiac fistulas such as atrial or ventricular septal defects, but only 2% of patients surviving the initial cardiac repair required reoperation for a residual defect in a delayed fashion in one review.[4] Thus, the majority of injuries are simple lacerations to the myocardium and are readily managed by the general, trauma, or acute care surgeon.

TABLE 30-1: Etiology of Traumatic and Iatrogenic Injuries of the Heart

1. Penetrating
   a. Low energy
      - Stab wounds—knives, ice picks, fence posts, wire, sports injuries
   b. High energy
      - Gunshot wounds—handguns, rifles, nail guns
      - Shotgun wounds—close range versus distant
      - Blast—fragments
2. Nonpenetrating (blunt)
   a. Motor vehicle collision
   b. Vehicle–pedestrian collision
   c. Falls from height
   d. Crush—industrial accident
   e. Blast—explosives, fragments, improvised explosive devices
   f. Assault
   g. Sternal or rib fractures
   h. Recreational—sporting events, rodeo, baseball
3. Iatrogenic
   a. Percutaneous interventions
   b. Pericardiocentesis
4. Others
   a. Electrical
   b. Embolic—missiles/fragments
   c. Factitious—needles, foreign bodies

Retained intrapericardial foreign bodies after penetrating wounds can sometimes cause complications of acute suppurative pericarditis, chronic constrictive pericarditis, foreign body reaction, and a hemopericardium.[5] On occasion, needles and other foreign bodies have been noted after deliberate insertion by patients with psychiatric diagnoses. A report by LeMaire et al[5] recommended removal of intrapericardial foreign bodies that are greater than 1 cm in size, are contaminated, or produce symptoms.

Intracardiac missiles can be embedded in the myocardium, retained in the trabeculations of the endocardial surface, or free in a cardiac chamber. These result from direct cardiac injury or injury to a peripheral venous structure with embolization to the heart. Observation might be considered when the missile is small, right-sided, embedded completely in the wall, contained within a fibrous covering, not contaminated, and producing no symptoms. Right-sided missiles, however, can embolize to the pulmonary artery, where they can be removed with catheter-based techniques, if large. In rare cases, they can embolize through a patent foramen ovale or atrial septal defect. Any left-sided missile can embolize systemically shortly after the initial injury, causing an arterial occlusion.

BLUNT CARDIAC INJURY

As previously mentioned, blunt cardiac injury, or BCI, has replaced the term *cardiac contusion* and describes injuries

ranging from insignificant bruises of the myocardium to cardiac rupture. These injuries can be caused by direct energy transfer to the heart, by a mechanism of compression of the heart between the sternum and the vertebral column, or by a sternal or rib fracture. Cardiac rupture during external cardiac massage as part of cardiopulmonary resuscitation (CPR) can occur as well, particularly in patients with a prosthetic heart valve.

The spectrum of BCI includes septal rupture, wall rupture, coronary artery thrombosis, cardiac failure, rupture of chordae tendinae/papillary muscles, and simple and complex dysrhythmias. Mechanisms with significant energy transfer are motor vehicle collisions, vehicular–pedestrian collisions, falls, crush injuries, blasts/explosions, and assaults. In one report, a fatal cardiac dysrhythmia occurred when the sternum was struck by a baseball, which may be a form of commotio cordis.[6]

The biomechanics of blunt cardiac rupture include the following: (1) direct transmission of increased intrathoracic pressure to the chambers of the heart; (2) a hydraulic effect from a large force applied to the abdominal or extremity veins, causing the force to be transmitted to the right atrium; (3) a decelerating force between fixed and mobile areas, explaining atriocaval tears; (4) a direct force causing myocardial contusion, necrosis, and delayed rupture; and, as noted earlier, (5) penetration from a broken rib or fractured sternum.[7] From autopsy data, BCI with chamber rupture occurs most often to the left ventricle. In contrast, in patients who arrive alive to the hospital, right atrial disruption (superior vena cava–atrial junction, inferior vena cava–atrial junction, or right atrial appendage) is more common. Blunt rupture of the cardiac septum occurs most frequently near the apex of the heart, whereas injury to only the membranous portion of the septum is the least common cause of a blunt ventricular septal defect. Multiple cardiac ruptures and disruption of the conduction system have been reported as well. Traumatic rupture of the thoracic aorta is also associated with lethal cardiac rupture in almost 25% of patients.

IATROGENIC CARDIAC INJURY

Iatrogenic cardiac injury can occur with insertion of a central venous catheter, cardiac catheterization procedures, endovascular interventions, and pericardiocentesis. Cardiac injuries caused by insertion of a central venous catheter usually occur from either the left subclavian or the left internal jugular vein.[8] Insertion of left-sided central lines or introducer sheaths, particularly during dilation of the tract, can lead to perforation of the innominate vein, superior vena cava, or right atrium. These small perforations sometimes lead to a compensated cardiac tamponade. Drainage by pericardiocentesis is often unsuccessful, and evacuation via a subxiphoid pericardial window or full median sternotomy is sometimes required. At operation, when the pericardium is opened, the site of injury has sometimes sealed and may be difficult to find. Perforation causing tamponade has also been reported with insertion of a right internal jugular introducer sheath for vascular interventions such as a transjugular intrahepatic portacaval shunt.

Complications from coronary catheterization, including perforation of the coronary arteries or a cardiac chamber and dissection of the thoracic aorta, are rare but can be catastrophic and require emergency surgical intervention.[9]

Other iatrogenic potential causes of cardiac injury include the aforementioned external and internal cardiac massage, transthoracic percutaneous interventions, and during intracardiac injections.[10]

ELECTRICAL INJURY

Cardiac complications after electrical injury include the following: immediate cardiac arrest; acute myocardial necrosis with or without ventricular failure; myocardial ischemia; dysrhythmias; conduction abnormalities; acute hypertension with peripheral vasospasm; and asymptomatic, nonspecific abnormalities evident on an electrocardiogram (ECG). Damage from electrical injury is due to direct effects on the excitable tissues, heat generated from the electrical current, and associated injuries from falls, explosions, or fires.[11]

Clinical Presentation

PENETRATING CARDIAC INJURY

Wounds involving the entire chest, especially the precordium and epigastrium, can cause a cardiac injury. Patients with cardiac injury can present with a clinical spectrum from asymptomatic with normal vital signs to a cardiac arrest. Up to 80% of stab wounds that injure the heart, however, will eventually cause tamponade.

Rapid bleeding into the pericardium favors clotting rather than defibrination. As pericardial blood accumulates, a decrease in ventricular filling occurs, leading to a decrease in stroke volume. A compensatory rise in catecholamines leads to tachycardia and increased right heart filling pressures. The limits of right-sided distensibility are reached as the pericardium fills with blood and the septum shifts toward the left side, further compromising left ventricular function. As little as 60 to 100 mL of blood in the pericardial sac can produce the clinical picture of tamponade.[12] The rate of accumulation depends on the location of the wound. Because it has a thicker wall, wounds to the ventricle seal themselves more readily than wounds to the atrium. Patients with freely bleeding injuries to the coronary arteries can present with rapid onset of tamponade combined with cardiac ischemia.

The classic findings of the Beck triad (muffled heart sounds, hypotension, and distended neck veins) are seen in a minority of acute trauma patients. Pulsus paradoxus (a substantial decrease in systolic blood pressure during inspiration) and Kussmaul sign (increase in jugular venous distention on inspiration) may be present but are also not reliable signs. A more valuable and reproducible sign of pericardial tamponade is narrowing of the pulse pressure. An elevation of the central venous pressure often accompanies overaggressive hyperresuscitation with crystalloid solutions, but in such instances, a widening of the pulse pressure occurs.

Gunshot wounds to the heart with a larger injury and a larger pericardial defect are more frequently associated with hemorrhage than with tamponade. Thus, these patients often present with exsanguination into a pleural cavity.

BLUNT CARDIAC INJURY

Clinically significant BCI includes the injuries listed earlier. These injuries present with tamponade, hemorrhage, or severe cardiac dysfunction. Septal rupture and valvular dysfunction (leaflet tear, papillary muscle or chordal rupture) can initially appear without symptoms but then cause delayed heart failure.

BCI can also present as a dysrhythmia for unknown reasons. Premature ventricular contractions are common, but ventricular tachycardia, ventricular fibrillation, and supraventricular tachycardia can occur usually within the first 24 to 48 hours after injury.

As previously noted, a major difficulty in managing BCI relates to old nonspecific terms such as *cardiac contusion*. It is best to describe these injuries as BCI followed by the clinical manifestation, such as dysrhythmia or heart failure.[13]

PERICARDIAL INJURY

Traumatic pericardial rupture is rare, and most patients with pericardial rupture do not survive transport to the hospital due to other associated injuries. Pericardial tears secondary to increased intra-abdominal pressure or lateral deceleration forces can occur on either side of the pericardium, to the diaphragmatic surface of the pericardium, or to the mediastinum. Cardiac herniation with cardiac dysfunction can occur as the heart is displaced into either pleural cavity or even into the abdomen depending on the tear. With a right pericardial rupture, the heart can become twisted, preventing venous return, leading to the surprising discovery of an "empty" pericardial cavity at resuscitative left anterolateral thoracotomy. With a left-sided cardiac herniation through a pericardial tear, a trapped apex of the heart prevents the heart from returning to the pericardium, and the term *strangulated heart* has been applied.[14,15]

Unless the heart is returned to its normal position, hypotension and cardiac arrest can occur. One clue to the presence of cardiac herniation in a patient with blunt thoracic injury is sudden loss of pulse when the patient is repositioned, such as when moved or placed on a stretcher.[14]

The overall mortality of patients treated at trauma centers with such an injury remains as high as 64%.[16] An overwhelming majority of these cases are diagnosed either intraoperatively or at autopsy.[14] The clinical presentation of pericardial rupture with cardiac herniation can mimic that of pericardial tamponade with low cardiac output due to impaired venous return; however, when the heart returns to its normal position in the pericardium, venous return resumes. As noted, positional hypotension is the hallmark of cardiac herniation due to pericardial rupture, whereas pericardial tamponade is associated with persistent hypotension until the pericardium is decompressed.[14] Therefore, a high index of suspicion is helpful when evaluating polytrauma patients with unexplained positional hypotension.

Evaluation

PHYSICAL EXAMINATION

The diagnosis of a cardiac injury requires a high index of suspicion. On initial presentation to the emergency center, airway, breathing, and circulation under the Advanced Trauma Life Support protocol are evaluated and treated as needed.[17] Intravenous access is obtained, and a blood specimen is sent for type and crossmatch. The patient is examined for the Beck triad as well as for pulsus paradoxus and Kussmaul sign. These findings suggest cardiac injury but, as noted previously, are present in only 10% of patients with cardiac tamponade. The patient undergoes simultaneously a focused assessment with sonography for trauma (FAST) (see later). If the FAST demonstrates pericardial fluid in an unstable patient (systemic blood pressure <90 mm Hg), transfer to the operating room is immediate if the patient has signs of life.

Patients in extremis from suspected or confirmed tamponade or intrapleural hemorrhage require emergency department thoracotomy for resuscitation. The current indications for this procedure by surgical personnel are divided into two categories and include the following[18] (see Chapter 17):

Salvageable postinjury cardiac arrest

- Patients with penetrating thoracic trauma and less than 15 minutes of prehospital CPR
- Patients with penetrating nonthoracic trauma and less than 5 minutes of prehospital CPR
- Patients with blunt trauma and less than 10 minutes of prehospital CPR

Persistent severe postinjury hypotension (systolic blood pressure <60 mm Hg) due to

- Cardiac tamponade
- Hemorrhage: intrathoracic, intra-abdominal, extremity, cervical
- Air embolism

If vital signs are regained after resuscitative thoracotomy, the patient is transferred to the operating room for definitive repair.

IMAGING

A surgeon-performed ultrasound (FAST) examination of the pericardial sac is highly accurate in determining the presence of an acute hemopericardium, hemothorax, or pneumothorax (see later). The routine chest x-ray is most valuable in diagnosing a hemothorax, pneumothorax, or track of a missile; confirming the presence of a missile in the mediastinum; or documenting the presence of a mediastinal or extrapleural hematoma. Thoracic CT will do all the same, as well as document trajectory and precisely locate a missile and confirm injuries to other structures in the mediastinum.

ECG

In cases of BCI, conduction disturbances can occur, with sinus tachycardia being the most common. Other common disturbances include T-wave and ST-segment changes, sinus bradycardia, first- and second-degree atrioventricular block, right bundle branch block, right bundle branch block with hemiblock, third-degree block, atrial fibrillation, premature ventricular contractions, ventricular tachycardia, and ventricular fibrillation. Thus, a screening 12-lead ECG is in the BCI guideline from the Eastern Association for the Surgery of Trauma.[19]

CARDIAC ENZYMES

The relationship between troponin levels and prognosis of BCI has been controversial.[20] Therefore, troponin measurements are not performed in all centers unless one is evaluating concomitant coronary artery disease.[20] Recent data, however, suggest that a normal ECG and troponin I effectively rule out BCI.[21,22]

FAST

The FAST examination evaluates four anatomic windows for the presence of pericardial or intra-abdominal fluid. Ultrasonography in this setting is not intended to reach the precision of studies performed in the radiology or cardiology suite but is intended to determine the presence of abnormal fluid collections.[23] Ultrasonography is safe, portable, and expeditious and can be repeated as indicated. If performed by a trained surgeon in patients with truncal trauma, primarily penetrating wounds to the chest and blunt trauma to the abdomen, the overall sensitivity and specificity were 83.3% and 99.7%, respectively, in one study.[24] In the same study, the sensitivity was 100% and specificity was 99.3% in patients with possible cardiac injuries from precordial or transthoracic wounds.[24] Two other ultrasound studies in patients with possible penetrating cardiac wounds from the same surgeon authors documented accuracies of 100% and 97.3%, respectively.[25,26] In urban environments with rapid transport times, the initial FAST to determine a hemopericardium may be falsely negative on rare occasions, and all negative FAST examinations are repeated in 15 minutes. As previously noted, the extended FAST looking at the pleural space has been adopted by many.[27] Finally, limited cardiac ultrasound is being used by the intensive care unit (ICU) to evaluate cardiac function and volume status (see Chapter 18).

FORMAL ECHOCARDIOGRAPHY

To evaluate more subtle findings of BCI such as wall motion or valvular or septal abnormalities in the stable patient, formal transthoracic echocardiography or transesophageal echocardiography is obtained. Transthoracic echocardiography is helpful but can have limited use in evaluating BCI because many patients have significant injury to the chest wall as well.

Formal echocardiography is most useful in the following situations: (1) diagnosing intrapericardial blood and tamponade physiology; (2) intraoperative evaluation of the pericardial cavity and heart during a nonthoracic procedure; (3) diagnosis of cardiac septal defects and valvular insufficiency; and (4) differentiating between ventricular dysfunction and cardiac tamponade, particularly in older patients. Of interest, most BCIs identified by echocardiography rarely require acute treatment.

SUBXIPHOID PERICARDIAL WINDOW

A subxiphoid pericardial window can be performed in the emergency department or in the operating room with the patient having either general or local anesthesia. In a prospective study, Meyer et al[28] compared the subxiphoid pericardial window with echocardiography in cases of penetrating cardiac injury and reported that the sensitivity and specificity of subxiphoid pericardial window were 100% and 92%, respectively, compared with 56% and 93%, respectively, with echocardiography. The authors suggested that the difference in sensitivity may have been due to the presence of a hemothorax, which can be confused with pericardial blood, or due to the fact that pericardial blood had drained into the pleural cavity.[28] The availability of the surgeon-performed FAST examination has almost eliminated the role of subxiphoid pericardial window in the evaluation of cardiac trauma. If necessary, pericardial exploration performed via a transdiaphragmatic route during laparotomy is more useful to diagnose a hemopericardium than pericardiocentesis (Fig. 30-1).

PERICARDIOCENTESIS

Pericardiocentesis has had significant historical support, especially years ago when the majority of penetrating cardiac wounds were produced by ice picks and the (surviving)

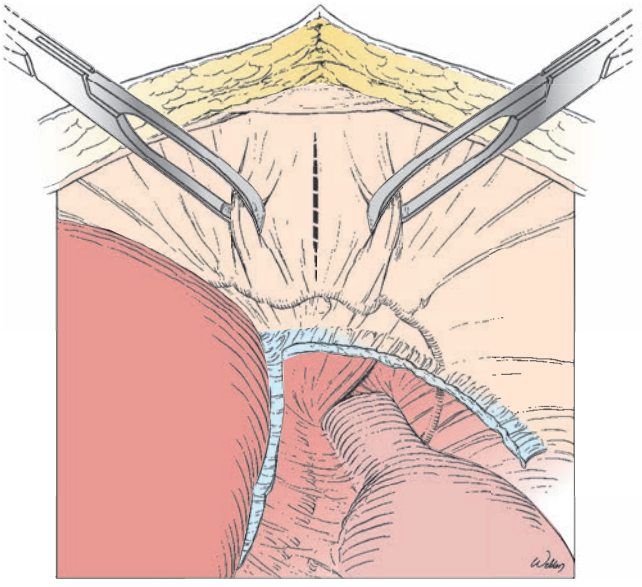


FIGURE 30-1 Transdiaphragmatic exploration of the pericardium during laparotomy. (Copyright © Baylor College of Medicine.)

patients arrived several hours and/or days after injury. In such instances, there was a natural triage of the more severe cardiac injuries, and the intrapericardial blood had become defibrinated and was easy to remove. As there are now probably more injuries from pericardiocentesis than accurate diagnoses, almost all trauma surgeons discourage the use of pericardiocentesis to diagnose acute cardiac trauma.[10] One indication for its use would be an iatrogenic injury caused by cardiac catheterization, at which time immediate decompression of the tamponade may be lifesaving, especially in the setting when a surgeon is not available.

Treatment

PENETRATING INJURY (SEE ATLAS FIGURE 29)

Because of the lethality of penetrating cardiac injuries, expeditious transport to an appropriate facility is important to survival. Rapid transport times and successful endotracheal intubation are positive factors for survival when the patient suffers a pulseless cardiac injury; however, recent data on hypovolemic patients suggest that positive-pressure ventilation may have an adverse effect on cardiac output.[29,30]

In the arrested or moribund patient with a positive pericardial FAST examination, exposure of the heart is accomplished via a left anterolateral thoracotomy as previously noted (Fig. 30-2).

This allows access to the pericardium and heart and exposure of the descending thoracic aorta for cross-clamping, if necessary. This incision can be extended across the sternum to gain access to the right side of the chest and for better exposure of the right atrium. Manual access to the right hemithorax from the left side of the chest can be achieved via the anterior mediastinum by blunt dissection as well. This allows rapid evaluation of the right side of the chest for major injuries without transecting the sternum or placing a separate chest tube. Once the left pleural space is entered, the lung can be retracted superiorly to allow clamping of the descending thoracic aorta distal to the left subclavian artery. The amount of blood present in the left chest suggests whether hemorrhage or tamponade is the primary issue. The pericardium anterior to the phrenic nerve is opened, injuries are identified, and control of hemorrhage or repair is performed.

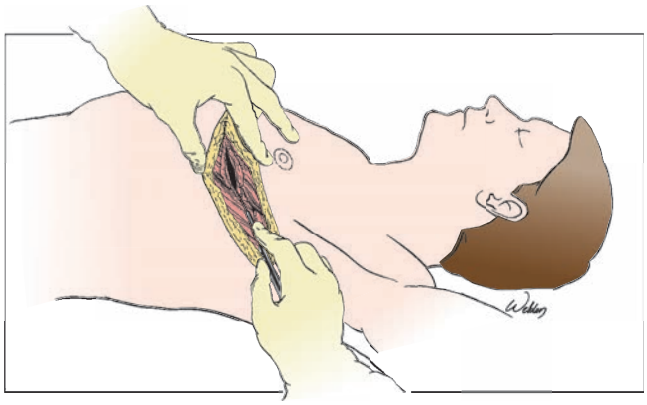


FIGURE 30-2 Left anterior thoracotomy (extension across the sternum if required). (Copyright © Baylor College of Medicine, 2005.)

In hemodynamically stable patients, particularly those with stab wounds to the precordium, a median sternotomy can be used. This allows exposure of the anterior structures of the heart but limits access to the posterior mediastinal structures and descending thoracic aorta for cross-clamping. Although posterior injuries can be addressed, these require skillful retraction and visualization during repair.

Poor technique during cardiac repair can result in enlargement of the injury or injury to the coronary arteries. If the initial treating physician is uncomfortable with suturing the injured heart, a Satinsky vascular clamp can be applied to the atrium or digital pressure to the ventricle until an experienced surgeon arrives. A large hole in a ventricle may require temporary insertion of a Foley balloon catheter, the use of crossed horizontal mattress sutures on either side of the injury, temporary occlusion of the superior and inferior vena cavae (inflow occlusion), or the intravenous injection of 3 mg of adenosine. Because cardiorrhaphy in the emergency center carries a 30% incidence of needlestick to the operator, a special skin stapler allows for rapid cardiac repair without the use of a needle[31] (Fig. 30-3). Repairs are definitive, although they can be buttressed in the operating room.

Injuries adjacent to coronary arteries can be managed by placing mattress sutures deep to the artery, avoiding it (Fig. 30-4). Mechanical support or cardiopulmonary bypass is required in less than 2% to 3% of patients in the acute setting.[4]

For stable patients with multiple thoracic gunshot wounds, diagnosis in the past was accomplished with chest x-rays in two projections, fluoroscopy, angiography, or echocardiography. Recently, multidetector CT scan has been used to determine missile tracks and localize any fragments. As previously noted, treatment of retained intracardiac missiles is individualized.[32-34] Removal is recommended for intracardiac missiles that are left-sided, larger than 1 to 2 cm, or rough in shape, or that produce symptoms. Although a direct approach, either with or without cardiopulmonary bypass, has been advocated, a large percentage of right-sided foreign bodies can now be removed by endovascular techniques, as mentioned earlier. Alternatively, those in the periphery of the atrium or right ventricle can be trapped with encircling sutures against the thinner chamber walls, the muscle incised, the missile removed, and then cardiorrhaphy performed.

Balloon occlusion of the aorta was described in the Korean War by the late Carl Hughes.[35] Resuscitative endovascular balloon occlusion of the aorta (REBOA) via femoral artery access is now being used to avoid the need to open the chest to clamp the descending thoracic aorta. Of interest, it has become common in the treatment of ruptured abdominal aortic aneurysms.[36,37] For trauma, REBOA is primarily recommended for hemodynamically unstable patients with an intact cardiac rhythm and a subdiaphragmatic injury or pelvic fracture. In such a patient, the balloon is inflated at the diaphragm (zone) or at the bifurcation of the abdominal aorta (zone 3), respectively.[38,39] This technique is less

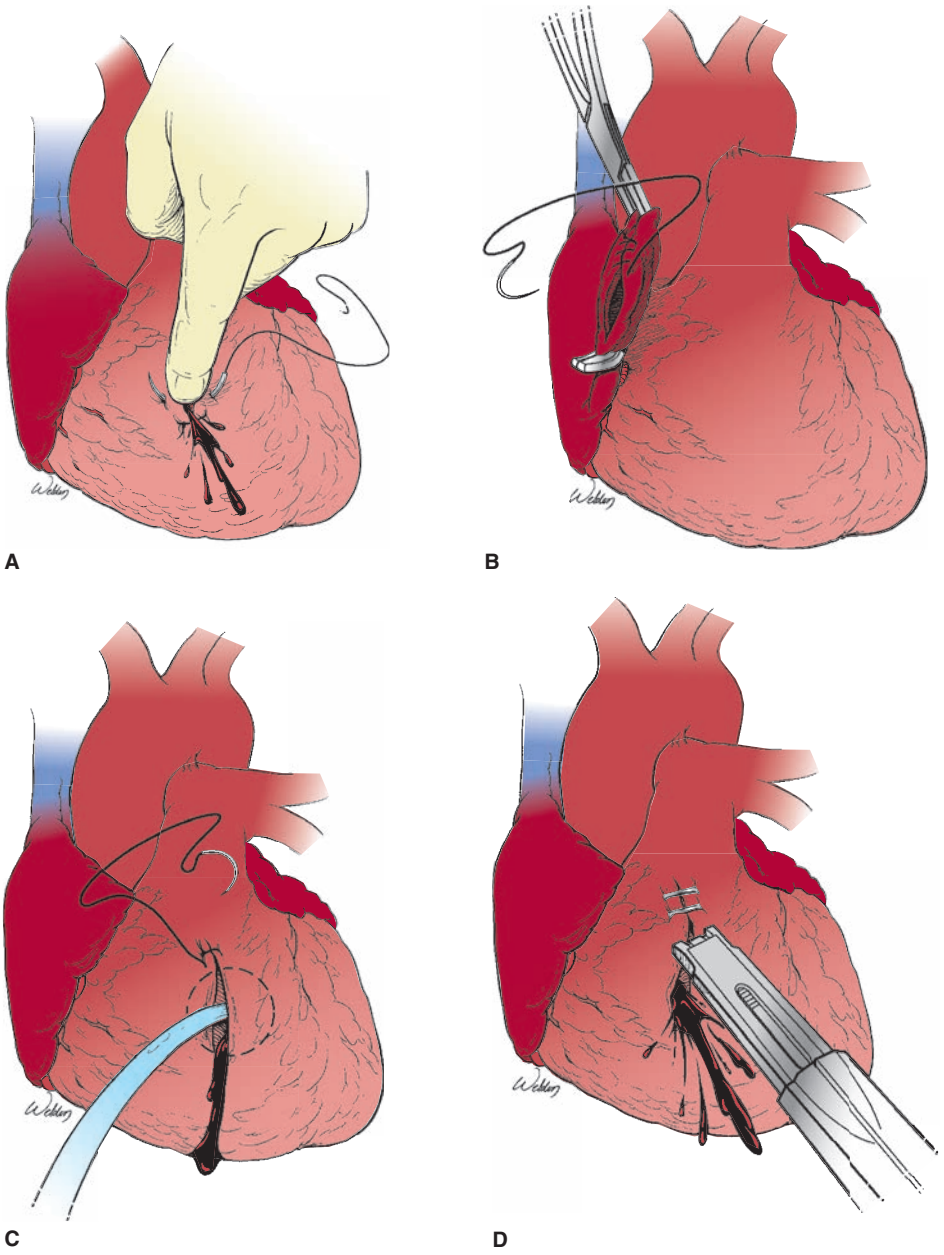


FIGURE 30-3 Temporary techniques to control bleeding. (**A**) Finger occlusion; (**B**) partial occluding clamp; (**C**) Foley balloon catheter; (**D**) skin staples. (Copyright © Baylor College of Medicine, 2005.)

useful for thoracic vascular injuries, as inflation of the balloon would occlude the aorta distal to potential injuries resulting in increased hemorrhage. It has its own complication profile, with reports of femoral access issues leading to amputation[40] (see Chapter 13).

BLUNT CARDIAC INJURY

Patients with new-onset cardiac arrhythmias without hypotension after blunt thoracic trauma are presumed to have a BCI and are admitted to a monitored bed.[13] Other patients with blunt thoracic injury and unexplained hypotension (rule out cardiogenic shock), presumed new-onset cardiac arrhythmias, and hypotension or a prior history of cardiac disease or surgery are admitted to an ICU.[13]

Results with Routine Cardiac Injuries

Many factors determine survival in the patient with a traumatic cardiac injury, including the following: mechanism of injury; location of injury; associated injuries; injury to a coronary artery or cardiac valve; presence of tamponade; length of prehospital transport; requirement for resuscitative

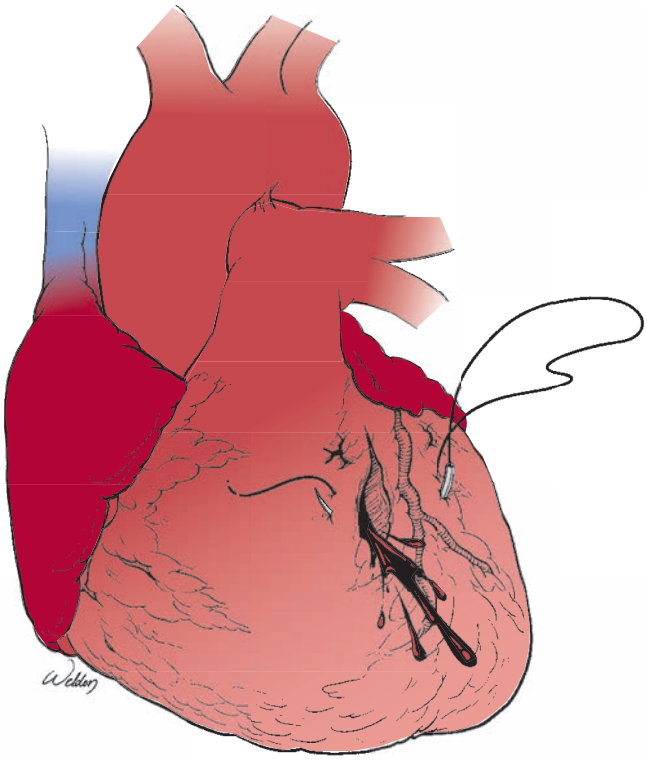


FIGURE 30-4 Injuries adjacent to coronary arteries can be addressed by placing sutures deep, avoiding injury to the artery. (Copyright © Baylor College of Medicine, 2005.)

thoracotomy; and experience of the trauma team. The overall hospital survival rate for patients with penetrating heart injuries who arrive with signs of life ranges from 30% to 85%, with rates of approximately 15% for those requiring emergency center thoracotomy and 74% to 86% for those stable enough to undergo operating room thoracotomy.

The survival rate for patients with stab wounds is 70% to 80%, whereas survival after gunshot wounds ranges between 30% and 40%. Blunt cardiac rupture has a worse prognosis than penetrating injuries to the heart, with a survival rate of approximately 20%.

Complex Cardiac Injuries

Complex cardiac injuries include those to the coronary artery or valvular apparatus (annulus, leaflet, papillary muscles, and chordae tendineae), intracardiac fistula, and delayed tamponade. These injuries have been reported to have a broad incidence (4%–56%), depending on the definition. Injury to the coronary artery is rare, occurring in 5%–9% of patients with cardiac injuries, and has a 69% mortality rate.[4] An injury to a distal coronary artery is most often controlled by simple ligation, but bypass grafting using a saphenous vein may be required for an injury to the proximal left anterior descending or right coronary artery.[4] Off-pump bypass can be used for such an injury in the unlikely event that the patient is hemodynamically stable.

Injury to the valvular apparatus is rare (0.2%–9%) and can occur after either blunt or penetrating trauma.[4] The aortic valve is most frequently injured, followed by the mitral and tricuspid valves. Unfortunately, many patients with injury to the aortic valve usually die in the prehospital period.

If severe cardiac dysfunction exists at the time of the initial operation and valvular injury is identified, immediate valve repair or replacement may be required; otherwise, delayed repair is more commonly advised. These injuries are usually identified postoperatively after the initial cardiorrhaphy and resuscitation have been performed in a patient with a new-onset postoperative murmur and/or hemodynamic deterioration. The timing of repair depends on the patient's condition.[4]

Intracardiac fistulas including ventricular septal defects, atrial septal defects, and atrioventricular fistulas have an incidence of 1.9% among all cardiac injuries. Management depends on symptoms and degree of cardiac dysfunction, with only a minority of these patients requiring repair.[4] These injuries are also usually identified after primary repair is accomplished, and they can be repaired after the patient has recovered from the original and associated injuries. Echocardiography should be obtained before repair so that specific anatomic sites of injury and operative planning can be accomplished.

Follow-Up

As discussed earlier, secondary sequelae in survivors of cardiac trauma include valvular abnormalities and intracardiac fistulas. Early postoperative clinical examination and ECG findings to diagnose these entities are unreliable. Thus, echocardiography is recommended during the initial hospitalization in all patients to identify an occult injury and establish a baseline study.[4,28,41] Because the incidence of late sequelae can be as high as 56%, follow-up echocardiography 3 to 4 weeks after injury has been recommended by some.[28,41]

INJURY TO THE THORACIC GREAT VESSELS

Injury to the thoracic great vessels—the aorta and its brachiocephalic branches, the pulmonary arteries and veins, the superior and intrathoracic inferior vena cava, and the innominate and azygos veins—occurs following blunt or penetrating trauma. Exsanguinating hemorrhage, the primary acute manifestation, also occurs in the chronic setting when a posttraumatic false aneurysm ruptures.

The operative treatment of great vessel injuries, primarily late traumatic false aneurysms and arteriovenous fistulas, was well described during World War II.[42] Current knowledge regarding the treatment of injured thoracic great vessels has been derived primarily from experience with civilian injuries related to improvements in emergency medical services and increased experience with elective surgery of the thoracic aorta and its major branches.[43]

TABLE 30-2: Common Anomalies of the Thoracic Aorta

Common origin of innominate and left common carotid arteries ("bovine arch")
Ductus diverticulum
Persistent left ductus arteriosus
Aberrant takeoff of the right subclavian artery from the descending thoracic aorta
Dextroposition of the thoracic aorta
Coarctation of the thoracic aorta
Origin of left vertebral artery off the aortic arch
Pseudocoarctation of the thoracic aorta ("kinked aorta")
Double aortic arch
Right ductus arteriosus

A detailed understanding of normal and variant anatomy and structural relationships is important in the evaluation of imaging studies. Venous anomalies occur, with the most common being absence of the left innominate vein and a persistent left superior vena cava. Anomalies of the aortic arch are relatively common and can mimic injuries or have implications for repair (Table 30-2). Knowledge of such anomalies is essential for both open and catheter-based therapies. For open cases, knowledge of aortic anomalies is helpful when dissecting to achieve vascular control around the often large hematoma that significantly distorts the anatomy. For endograft repair, vascular anomalies can affect the ability to achieve adequate seal zones. Aberrancies in the site/origins/outflow of the vertebral arteries can also significantly alter strategies and potential need for additional revascularization.

Etiology and Pathophysiology

More than 90% of thoracic great vessel injuries are due to penetrating trauma or therapeutic misadventures.[43] Iatrogenic lacerations of various thoracic great vessels, including the arch of the aorta, have been reported as complications of placement of percutaneous central venous catheters. The placement of chest tubes or pigtail catheters has caused injuries to the intercostal arteries and major pulmonary and mediastinal vessels as well. Finally, intra-aortic cardiac assist or occlusion balloons can produce injury to the thoracic aorta.

During emergency center resuscitative thoracotomy, the aorta may be injured during clamping if a crushing (nonvascular) clamp is used. Overinflation or migration of the balloon of a Swan-Ganz catheter has produced iatrogenic injuries to pulmonary artery branches with resultant fatal hemoptysis. Thus, once a linear relationship has been established between the pulmonary artery diastolic pressure and the pulmonary capillary wedge pressure, further "wedging" is unnecessary. Covered stent grafts being used for endovascular repairs of vascular injuries can cause iatrogenic injury through access, fixation, puncture, rupture, or occlusion. Self-expanding metal stents have recently produced perforations of the aorta and innominate artery following placement into the esophagus and trachea, respectively.[44]

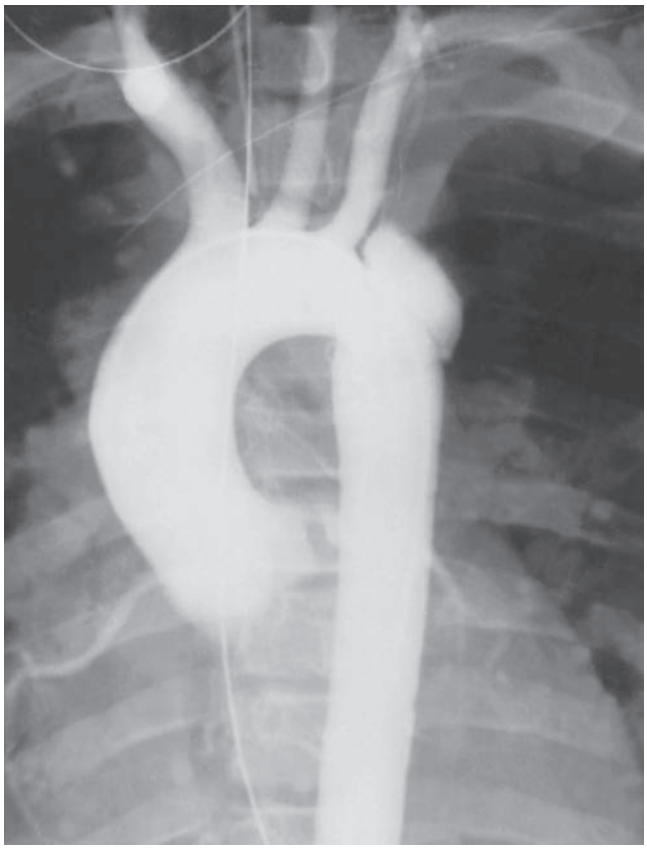


FIGURE 30-5 Aortogram demonstrating the classic intimal tear and traumatic pseudoaneurysm of the descending thoracic aorta.

The great vessels susceptible to injury from blunt trauma commonly include the origin of the innominate artery, pulmonary veins, vena cava, ascending aorta, and, most commonly, the descending thoracic aorta.[45] Aortic injuries have caused or contributed to 10% to 15% of deaths following motor vehicle accidents in the past 50 years before the introduction of airbags. These injuries commonly involve the proximal descending aorta (54%–65% of cases) (Fig. 30-5), but can involve other segments, such as the ascending aorta or transverse aortic arch (10%–14%), the mid- or distal descending thoracic aorta (12%) (Fig. 30-6), or multiple sites (13%–18%).

The postulated mechanisms of blunt great vessel injuries include the following: (1) shear forces caused by the relative mobility of a portion of the vessel adjacent to a fixed portion; (2) compression of the vessel between bony structures; and (3) abrupt, profound intraluminal hypertension during the traumatic event. The pericardial attachments of the pulmonary veins and vena cavae and the fixation of the descending thoracic aorta at the ligamentum arteriosum and diaphragm enhance their susceptibility to blunt rupture by the first mechanism. At its origin, the innominate artery may be "pinched" between the sternum and the vertebrae during sternal impact and cause what is actually an aortic arch injury.

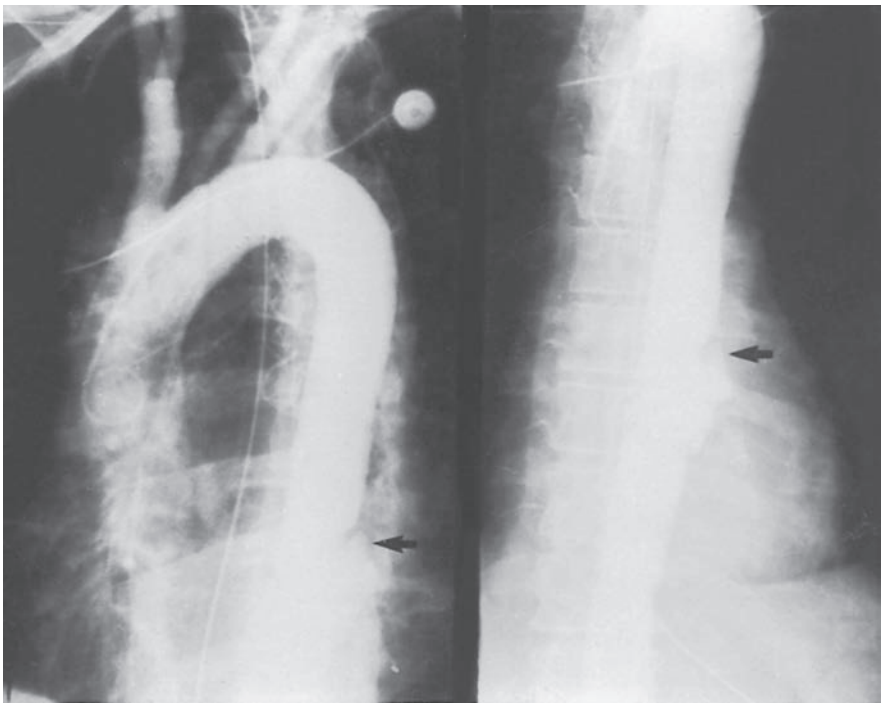


FIGURE 30-6 Aortogram in a patient with blunt chest trauma demonstrating an intimal tear of the descending thoracic aorta at the diaphragm.

Blunt aortic injuries may be partial thickness (traumatic true aneurysm)—histologically similar to an intimal tear—but most commonly are full thickness (traumatic false aneurysm) and, therefore, equivalent to a ruptured aortic aneurysm that is contained by surrounding tissues.

The histopathologic similarities between aortic injuries and nontraumatic aortic catastrophes suggest that similar therapeutic approaches be employed. Therefore, in hemodynamically stable patients, the concepts of permissive hypotension and minimization of arterial pressure impulse (dP/dT), which are widely accepted in the treatment of aortic dissection and aneurysm rupture, should be considered in the initial treatment of patients with blunt aortic injuries.

In contrast to patients with aortic medial disease where the adventitia is the restraining barrier, it is the intact parietal pleura that contains the hematoma and prevents a massive hemothorax around a full-thickness blunt injury to the descending thoracic aorta.

True traumatic aortic dissection, with a longitudinal separation of the media extending along the length of the aorta, is extremely rare.[46] The use of the term *dissection* in the setting of aortic trauma should thus be equally rare. Similarly, the terms *aortic transection* and *blunt aortic rupture* should be used only when describing specific injuries (ie, full-thickness lacerations involving either the entire or partial circumference, respectively). The term *blunt aortic injury* is probably most accurate and descriptive.

Patients with injury to a thoracic great vessel almost always have associated head, abdominal, and extremity injuries. Often, preexisting medical conditions are present, such as diabetes mellitus, hypertension, coronary artery disease, or cirrhosis. In addition, these patients may be taking a variety of medications, including aspirin, warfarin, platelet inhibitors, or other anticoagulants. These interfere with the clotting mechanism, and adaptations in treatment may be required.

Classification of Blunt Aortic Injury

ANATOMIC

Several anatomic classification systems of aortic injury have been proposed.[47,48] Azizzadeh et al[47] proposed a classification describing intimal tears, intramural hematoma, pseudoaneurysm, and rupture. Starnes et al[48] suggested a classification based on the presence of an abnormality in the aortic external contour (Table 30-3). Those without a contour abnormality were subdivided into intimal tears and large intimal flaps. Those with aortic contour abnormalities were subclassified into pseudoaneurysm and rupture. This is a functional

TABLE 30-3: Classification of Blunt Injuries to Thoracic Aorta

External aortic contour abnormality	Type
Absent	Intimal tear <10 mm
	Large intimal flap >10 mm
Present	Pseudoaneurysm
	Rupture

Source: Data from Starnes BW, Lundgren, RS, Gunn M, et al. A new classification scheme for treating blunt aortic injury. *J Vasc Surg.* 2012;55:47.

classification, and they noted that no patient with normal external aortic contour died of aortic injury in their experience. Eight of nine patients with free rupture died, raising the question of futility in these patients. The authors recommended the following: use of anti-impulse therapy and observation for initial intimal tears less than 10 mm; selective management of intimal flaps greater than 10 mm; semielective treatment of stable patients with pseudoaneurysms; and consideration of earlier treatment of patients with an abnormality of the external aortic contour and a traumatic brain injury because such patients might benefit from an increased blood pressure. In addition, they noted that coverage of the orifice of the left subclavian artery orifice was well tolerated in most patients.

PHYSIOLOGIC

Three distinctly different groups of patients with thoracic aortic trauma exist (Table 30-4). The epidemiology of aortic injury is changing, perhaps due to restraints and airbags in vehicles, road design, rapid accident notification, and emergency medical service (EMS) transport. The mortality statistics reveal that patients with exsanguinating hemorrhage almost all die within the first 0 to 2 hours of injury. Those who die in the emergency department, operating room, or ICU within 2 to 4 hours of injury often have extensive multisystem injury with hemorrhage, often from sites other than the thoracic aorta.

Patients who died several hours after injury from a traumatic brain injury but were found to have an unoperated thoracic aortic injury were subsequently analyzed by Mattox et al.[49] Cohorts of this group who remained moderately hypotensive did *not* die from an aortic rupture. This led to a policy of using drugs to alter the aortic shear force in patients with known aortic injuries who also had severe injuries to the brain or to multiple organs. The continuing lack of aortic-related mortality in this cohort led to a purposeful delay in aortic repair. The timing of such later repair and the technique of the repair are matters of local judgment. Hemodynamically stable patients who die and are subsequently found to have an aortic injury most often have injury to the brain as the cause of death.[49]

Prehospital Management

In patients with significant penetrating or blunt thoracic trauma, interventions performed by paramedics during transport can include judicious administration of intravenous fluids and endotracheal intubation when indicated.[50] In patients with acute injuries to a thoracic great vessel, excessive fluid resuscitation with the goal of increasing blood pressure to normal or supernormal levels increases mortality and other postoperative complications.[51]

Evaluation in the Emergency Center

HISTORY

In a patient with penetrating thoracic trauma, information regarding the length of a knife, or the firearm type, number of rounds fired, and the patient's distance from the firearm should be sought from the patient or witnesses.

Although the head-on motor vehicle collision is often considered the typical mechanism for blunt aortic injury, recent epidemiologic data reveal that up to 50% of cases occur following side-impact collisions. These injuries have also been reported following equestrian accidents, blast injuries, auto–pedestrian crashes, crush injuries, and falls from heights of 30 feet or more.[52] EMS personnel should be queried about the amount of hemorrhage at the scene, any history of intermittent paralysis following the accident, and hemodynamic instability during transport. Employing patterns of injury, the extent and location of damage to the vehicle parallel the potential injuries to the patient and may suggest the possibility of injury to a great vessel.[53]

PHYSICAL EXAMINATION

Upon arrival in the emergency center, each patient is given a rapid, thorough primary and secondary survey. External signs of penetrating or blunt trauma are noted. With an intrapericardial vascular injury, the previously described classic signs of pericardial tamponade (distended neck veins, pulsus paradoxus, muffled heart sounds, and elevated central venous pressure) may be present but occur infrequently. Clinical findings associated with injury to a thoracic great vessel include the following: hypotension; upper extremity hypertension; unequal blood pressures or pulses in the extremities (upper extremity from injury to the innominate or subclavian artery, or lower extremity from pseudocoarctation syndrome); external evidence of major chest trauma (eg, steering wheel imprint, hoofprint on chest); expanding hematoma at the thoracic outlet; intrascapular murmur; palpable fracture of the sternum; palpable fracture of the thoracic spine; and left flail chest.

TABLE 30-4: Groups of Patients with Injury to Thoracic Aorta

Group	Description	Time to diagnosis	Location of death	Mortality	Cause of death
1	Dead/dying at scene	<60 min	Scene/EMS	100%	Bleeding
2	Unstable during transport	1–6 h	EMS/EC	>96%	Multisystem trauma
3	Stable	14–18 h	ICU	5%–30%	CNS injury

CNS, central nervous system; EC, emergency center; EMS, emergency medical services; ICU, intensive care unit.

IMAGING

Upon arrival, a supine anteroposterior chest x-ray should be performed, ideally in the emergency center and not in a distant radiologic suite. Emergency physicians, radiologists, and surgeons should develop diagnostic experience viewing supine portable chest x-rays because many trauma patients are hemodynamically unstable or have suspected spinal injuries, making an "upright" chest x-ray unsafe to obtain. In many patients with injury to a great vessel, the radiologic findings are sufficient to warrant further investigation or direct transport to the operating room.

For penetrating injuries, it is helpful to place radiopaque markers to identify the surface wounds. Radiographic findings that suggest a penetrating wound to a thoracic great vessel include the following: large hemothorax; foreign bodies (bullets or fragments) or their trajectories in proximity to the great vessels; a foreign body out of focus with respect to the remaining x-ray, which may indicate an intracardiac location; a trajectory with a confusing course, which may indicate a migrating intravascular bullet (Fig. 30-7); and a "missing" missile in a patient with a gunshot wound to the chest, suggesting distal embolization in the arterial tree.

Several x-ray findings have been associated with blunt injuries of the descending thoracic aorta. Although widening of the mediastinum is nonspecific, the most reliable of these signs is the "double shadowing" of the aortic knob contour (Table 30-5). Mediastinal widening at the thoracic outlet and leftward tracheal deviation are suggestive of an injury to the innominate artery. These signs are secondary to a mediastinal hematoma, which is an indirect sign of injury to a thoracic great vessel. The presence of any of these signs is a positive screening test and warrants further investigation. Currently, screening contrast CT of the chest is commonly obtained in these patients and has replaced the screening chest x-ray.

Due to the density of important structures, missile wounds that appear to traverse the mediastinum create concern regarding injury to the heart, esophagus, trachea, spinal cord, or major vessels. Mediastinal transverse with paraplegia suggests a posterior trajectory, with injures to the heart and vascular structures less likely. Should a cardiac or vascular injury be present, tamponade or major hemorrhage is usually obvious and precludes the need for a CT. Therefore, multidetector CT scanners are often used in stable patients to demonstrate missile trajectory.

Initial Treatment and Screening

TUBE THORACOSTOMY

When the chest x-ray indicates a significant hemothorax, a chest tube can be placed and connected to a collection system

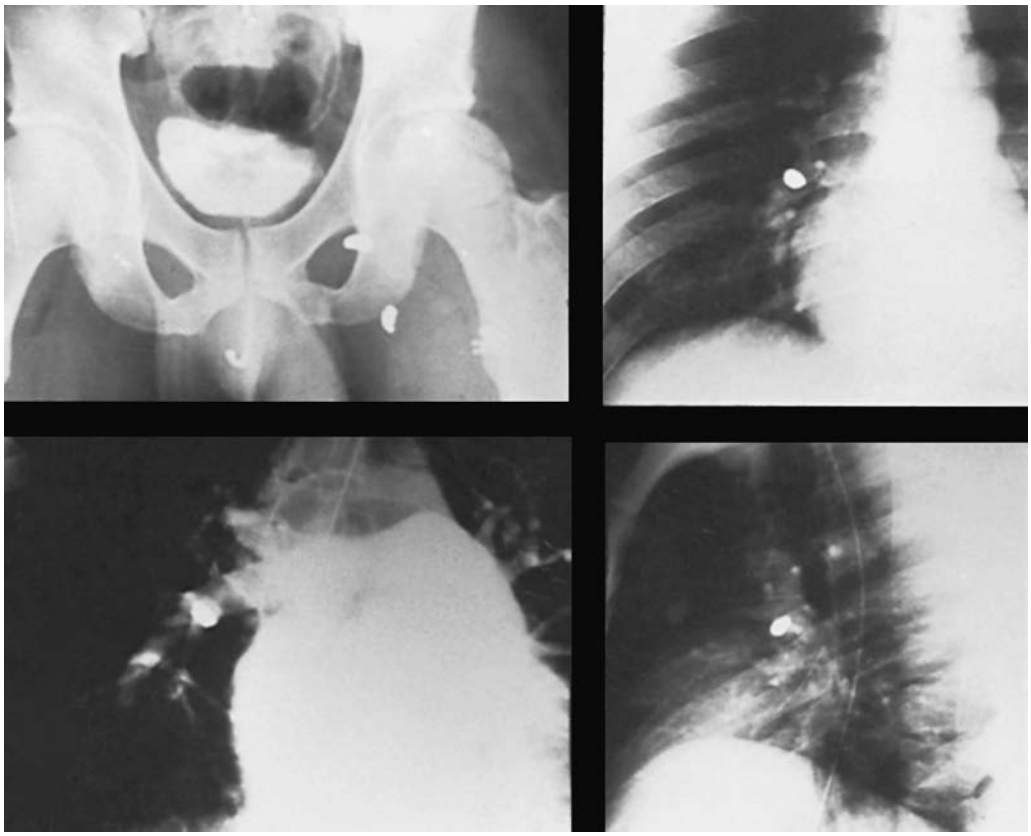


FIGURE 30-7 Series of x-rays demonstrating the entrance site of a bullet in the left groin. The bullet embolized to the right pulmonary artery, as confirmed by arteriography.

TABLE 30-5: Radiographic Clues That Should Prompt Suspicion of an Injury to a Thoracic Great Vessel

Fractures	Sternum
	Scapula
	Multiple left ribs
	Clavicle in multisystem injured patient
Mediastinal clues	Obliteration/double shadow of aortic knob contour
	Widening of the superior mediastinum >8 cm
	Depression of the left mainstem bronchus >140° from trachea
	Loss of paravertebral pleural stripe
	Calcium layering at aortic knob
	Deviation of nasogastric tube in the esophagus
	Lateral displacement of the trachea
	"Funny looking" mediastinum
Lateral chest x-ray	Anterior displacement of the trachea
	Loss of the aorta/pulmonary artery window
Other findings	Apical pleural hematoma
	Massive left hemothorax
	Obvious blunt injury to the diaphragm

that allows for autotransfusion. As previously noted, an output of 1500 mL over the first 15 minutes or significant ongoing hemorrhage (>200–250 mL/h) is an indication for an emergent or urgent thoracotomy.[54]

EMERGENCY CENTER THORACOTOMY

Emergency center thoracotomy in patients presenting with signs of life and hemodynamic collapse may reveal injuries to major thoracic vessels. These injuries require temporizing maneuvers to gain rapid control of bleeding and allow resuscitation and then subsequent transfer to the operating room for definitive repair.[55] Subclavian vessel injuries, for example, can be controlled by intrapleural packing, clamping at the thoracic apex, or inserting intravascular balloon catheters. Aortic injuries can be controlled with aortic or partial occluding clamps. Major hemorrhage from the pulmonary hilum can be temporarily managed by cross-clamping the entire hilum proximally or twisting the lung 180° after releasing the inferior pulmonary ligament.[56]

INTRAVENOUS ACCESS AND FLUID ADMINISTRATION

Currently, unless a patient is in extremis, large-bore intravenous catheters are inserted, but high-volume resuscitation is avoided until the time of vascular control at operation. If a subclavian venous catheter is required in a patient with an upper thoracic injury, the contralateral side should be used for cannulation. The treatment of severe shock should start with blood transfusion; however, rapid infusions of excessive volumes of either blood or crystalloid solutions prior to operation may increase the blood pressure to a point that a protective soft perivascular clot is dislodged and fatal exsanguinating hemorrhage ensues.

The principles of permitting moderate hypotension (systolic blood pressure of 60–90 mm Hg) and limiting fluid administration until achieving operative control of bleeding are cornerstones in the management of ruptured abdominal aortic aneurysms and equally apply to acute injuries to thoracic great vessels. Following guidelines developed with the military, a conscious patient with a radial pulse is not resuscitated until the time of operative vascular control.[57] Aggressive preoperative fluid resuscitation also increases postoperative respiratory complications and may contribute to an increased mortality when compared to fluid restriction.[51] With both penetrating and blunt chest trauma, associated pulmonary contusions are common and provide an additional rationale for limiting the infusion of preoperative crystalloid solutions.

IMPULSE REDUCTION THERAPY/β-BLOCKADE FOR SUSPECTED BLUNT AORTIC INJURY

The pharmacologic reduction of arterial impulse (dP/dT) has remained a critical component of the treatment of aortic dissection since its original description by Wheat et al[58] in 1965. Based on the similarity between aortic dissection and blunt aortic injury, this principle was first applied in 1970 using β-blockade to reduce aortic impulse for patients with suspected or confirmed blunt injury to the thoracic aorta. Subsequent reports have described using this therapy in hemodynamically stable patients who had proven blunt aortic injuries but required a delay in definitive operative treatment.[59] Some centers routinely begin impulse reduction therapy as soon as an injury to the thoracic aorta is suspected—prior to obtaining diagnostic studies—in an attempt to reduce the risk of fatal rupture during the interval between presentation and confirmation of the diagnosis. Although retrospective studies suggest that this policy is safe, no prospective studies have demonstrated either the safety or efficacy of such treatment.

SCREENING/PLANNING CT SCAN FOR THORACIC VASCULAR INJURY

Following blunt trauma, the potential for thoracic great vessel injury, and, therefore, the need to proceed with further investigation, are determined based on the following: (1) the mechanism of injury; (2) physical examination; (3) the standard chest x-ray; or (4) a screening CT scan.

All of these factors must be considered in concert. Traumatic ruptures of the thoracic aorta have occurred following seemingly innocuous mechanisms, including low-speed automobile crashes (<10 mph) with airbag deployment and intrascapular back blows used to dislodge an esophageal foreign body. Additionally, 50% of patients with thoracic vascular injuries from blunt trauma present without any external physical signs of injury. In addition, 7% of patients with

blunt injury to the aorta and brachiocephalic arteries have a normal-appearing mediastinum on admission chest x-ray.

As previously noted, multidetector CT scan of the chest is recommended as a screening test for blunt injury to the thoracic aorta.[60-62] As resolution and experience in using CT to plan operations increase, it is important to ensure that the appropriate information regarding extent of injury, anatomy, and aberrant branches is obtained. Because it is the most common location for blunt aortic injuries, CT scan as a diagnostic test is most helpful to diagnose an injury of the proximal descending thoracic aorta. Motion artifact in the proximal ascending aorta, however, can be difficult to interpret on CT. A CT scan gated to cardiac motion may better delineate the ascending aorta and provide increased resolution.[63]

The diagnostic controversy regarding CT for thoracic injuries lies in the technology and technique, which has evolved at a very rapid rate. It is important to understand that a 4-channel, 16-detector machine has different capabilities than a 64-channel/detector machine. The protocols for obtaining the CT examination, such as number and spacing of detectors, channels, pitch, slice thickness, contrast injection, timing, and reconstruction, can significantly alter the information obtained. Thus, it is important to be familiar with the appropriate protocols needed.

The raw CT data are manipulated in a "postprocessing" function to deliver the final images. The previous static CT film images are now read on digital displays where a knowledgeable observer can further manipulate the image. Three-dimensional reconstructions, although impressive to view, consume processing resources and have not been necessary in the acute evaluation of all blunt aortic injuries (Fig. 30-8).

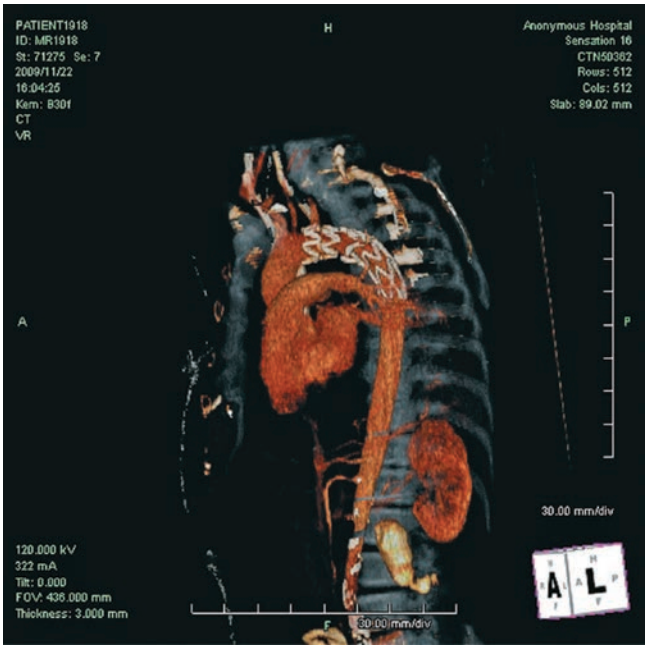


FIGURE 30-8 A three-dimensional reconstruction of the computed tomography in a patient with an injury to the aortic isthmus showing the thoracic endograft deployed.

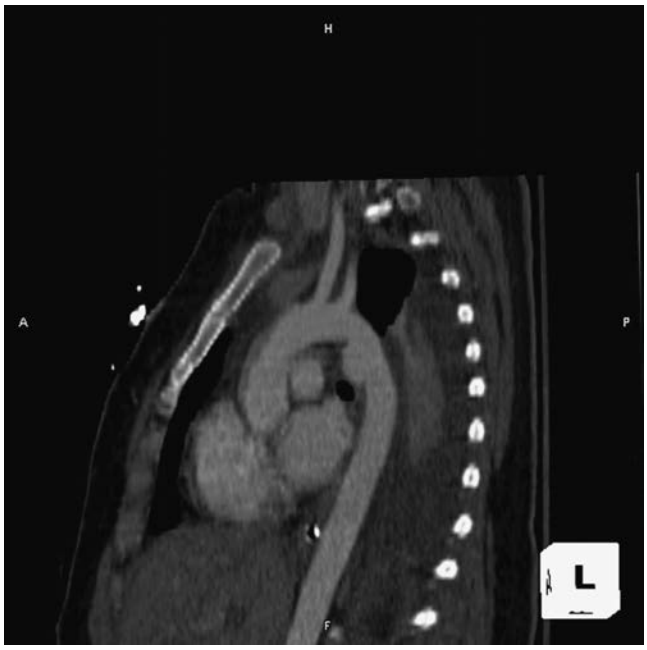


FIGURE 30-9 Multiplanar reformatting display of a typical injury through the descending thoracic aorta distal to the left subclavian artery. This allows the viewer to align the slice along the axis of the aorta. The small cube in the lower right corner represents the orientation.

Multiplanar reformatting is a postprocessing mode where the CT slice can be angled and positioned to best display the pathology. This is most useful for the evaluation of blunt aortic injury when the CT slice/virtual gantry is aligned with the curvature of the ascending/arch/descending thoracic aorta, and the slices can traverse through the aorta (Fig. 30-9). This is helpful not only for diagnosis, but also for planning, selecting endovascular devices, and evaluating seal zones for the device. Center-line flow analysis displays the aorta as a straight line along its center allowing precise measurements of diameter and accurate measurements of seal zones for planning (Fig. 30-10). If the clinician directly caring for the patient cannot manipulate and interpret the images themselves, much useful information as well as artifacts may not be appreciated. This may explain some of the conflicting reports and opinions on the utility of CT for screening or diagnosis. With appropriate scanners, protocols, processing, display, and experience, CT potentially yields more information than catheter angiography.

If a mediastinal hematoma is visualized on CT, formal aortography was obtained in the past to specifically determine the site of the injury and to identify any vascular anomalies that required modifications in the operative approach. With improved resolution of the screening CT of the chest, this study is no longer needed for screening. In current practice, this now occurs just prior to placing an endograft.

Algorithms can be constructed to aid the surgeon in reaching a diagnosis and treating a patient with an injury to the thoracic aorta (Fig. 30-11). As experience has developed with endovascular repair, the CT scan has been extremely helpful

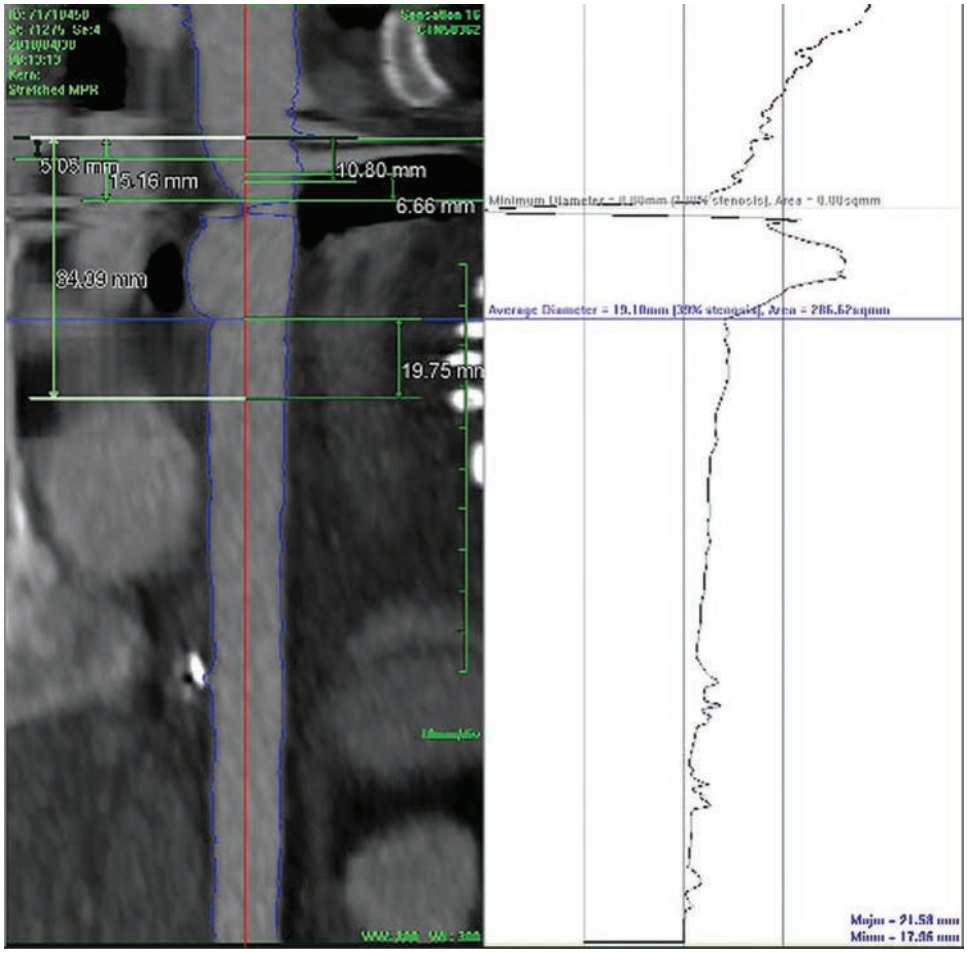


FIGURE 30-10 Center-line flow analysis of a patient with injury at the aortic isthmus. This view electronically straightens the aorta along the center-line axis of flow, allowing accurate measurements regarding landing zones/seal areas and the device length to be determined. This analysis shows that by covering the left subclavian artery, a 15- to 16-mm proximal seal area is available and a 35-mm area will need to be covered. This display also shows the average aortic diameter to be 19 mm. This can be useful to plan difficult cases for which the landing zones/seal area are difficult to precisely determine.

for preoperative planning for evaluation for access and stent graft repair. The CT protocol for thoracic trauma, however, has much less information than the protocol for planning elective stent grafting. Initial CT scans are often performed on underresuscitated patients, with highly compliant/dynamic aortas. Intraoperative planning with intravascular ultrasound is frequently performed and very useful. Transesophageal echocardiography has added little in the screening or diagnosis of thoracic aortic injury, except perhaps intraoperatively when operating on another cavity. Magnetic resonance angiography can generate similarly detailed information; however, its application in these potentially unstable trauma patients is not currently practical. In patients who are treated in a delayed fashion with a very complex injury, it can be helpful to get a stent graft protocol CT after resuscitation.

CATHETER ARTERIOGRAPHY

In the past, catheter angiography was recommended to diagnose suspected penetrating injuries to the thoracic aorta or to the innominate, carotid, or subclavian arteries in stable patients. As noted earlier, however, experience with chest CT arteriography (CTA) has replaced catheter angiography as an initial screening test or to plan procedures. Any questionable findings on the CTA can be further evaluated by catheter arteriography or intravascular ultrasound if needed. Also, catheter arteriography or CTA is helpful for localizing the injury and planning the appropriate incision. Proximity of a missile trajectory to the brachiocephalic vessels, even without any physical findings of vascular injury, can be an indication for imaging, recognizing also that thoracic outlet vascular injuries may involve multiple vessels.[64] Catheter arteriography, however, has recognized limitations. A "negative" aortogram may convey a false sense of security if the laceration has temporarily "sealed off" or if the column of aortic contrast overlies a small area of extravasation (Fig. 30-12). Therefore, an effort must be made to obtain views tangential to possible injuries (Figs. 30-13 and 30-14). In addition, CTA has the limitations of scatter adjacent to a missile, which may obscure a possible injury.

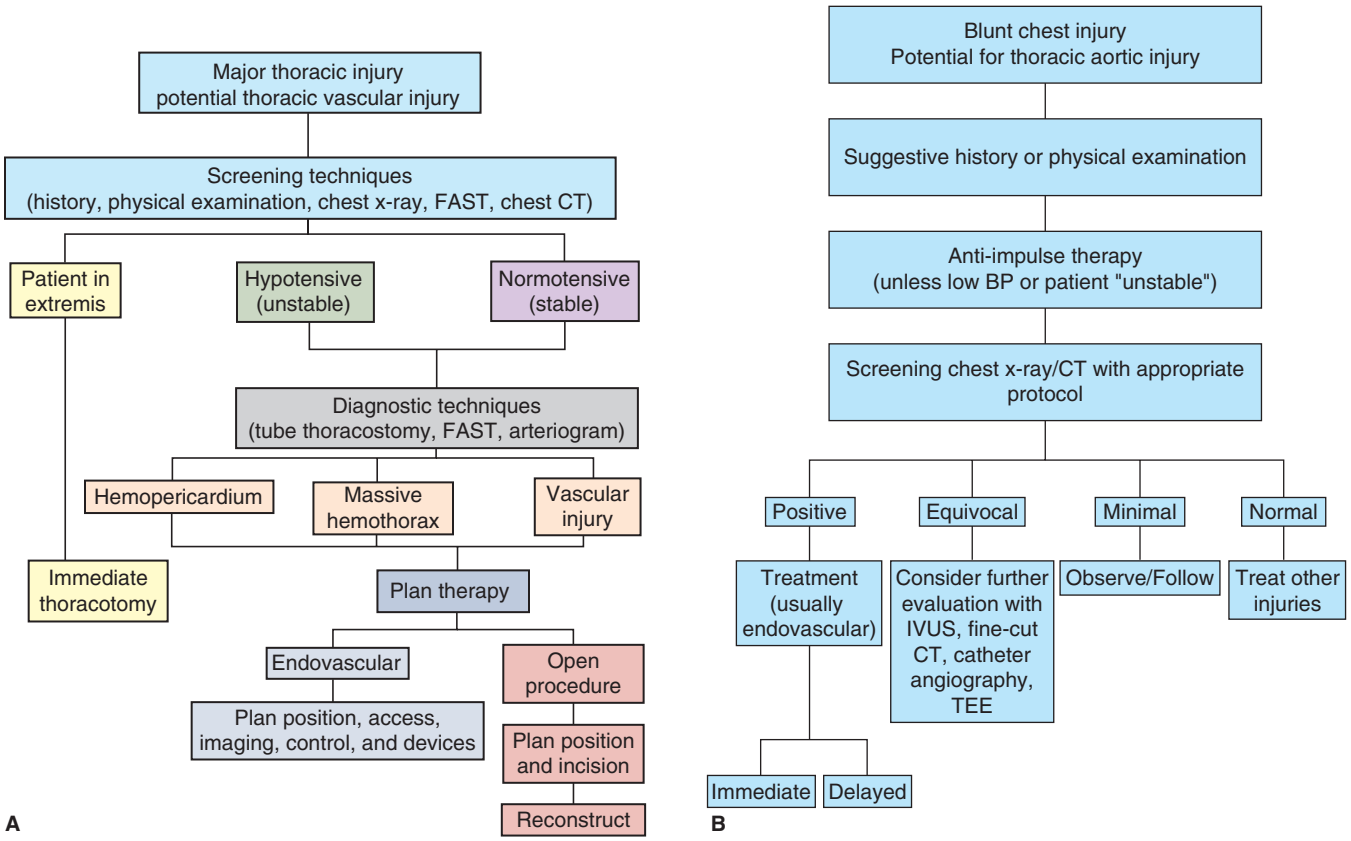


FIGURE 30-11 (**A**) Algorithm for an approach to patients with suspected thoracic vascular injury. (**B**) Algorithm for the evaluation and treatment of a patient suspected of having a blunt injury to the thoracic aorta. BP, blood pressure; CT, computed tomography; FAST, focused assessment with sonography for trauma; IVUS, intravenous ultrasound; TEE, transesophageal echocardiography.

Treatment Options

NONOPERATIVE MANAGEMENT

Nonoperative management of blunt injuries to the thoracic aorta should be considered in patients who have potentially lethal associated injuries or contraindications to early repair. Examples would include the following: traumatic brain injury; risk factors for infection (major burns, sepsis, heavily contaminated wounds); severe multisystem trauma with hemodynamic instability; poor physiologic reserve; and a minimal aortic injury.

In such instances, nonoperative management is actually a purposeful delay in operation while attempts are made to achieve physiologic optimization and improve the potential outcome of repair. Nonoperative management has also been used successfully in patients with "nonthreatening" aortic lesions, such as minor intimal defects and small traumatic false aneurysms. Close observation without operation is similarly reasonable for small intimal flaps involving the brachiocephalic arteries in asymptomatic patients, as many such lesions will heal spontaneously.

With the increased use of endograft repair as well as patients with an increased number of associated injuries, blunt injuries to the thoracic aorta are often definitively repaired more than 24 hours after presentation when the patient is optimized. In the multicenter report by Demetriades et al,[59] delayed repair (>24 hours) of stable patients with blunt injury to the thoracic aorta was associated with improved survival, but also a longer stay in the ICU and a higher complication rate.

Although apparent minor vascular injuries may resolve or stabilize, their long-term natural history remains uncertain. Life-threatening complications of great vessel injuries—including rupture and fistulization with severe hemorrhage—occurring more than 20 years after injury have been reported.[52] Therefore, careful follow-up, including serial imaging studies, is a critical component of nonoperative management. Avoiding hypertension and the use of impulse control agents are recommended as well when patients with blunt injuries to the thoracic aorta are managed nonoperatively.

ENDOVASCULAR REPAIR: GENERAL PRINCIPLES

There are many new techniques and devices for the endovascular approach to injuries to the thoracic aorta and entries in the thoracic outlet. With better understanding of the endovascular tools, this approach has increasingly been adopted by

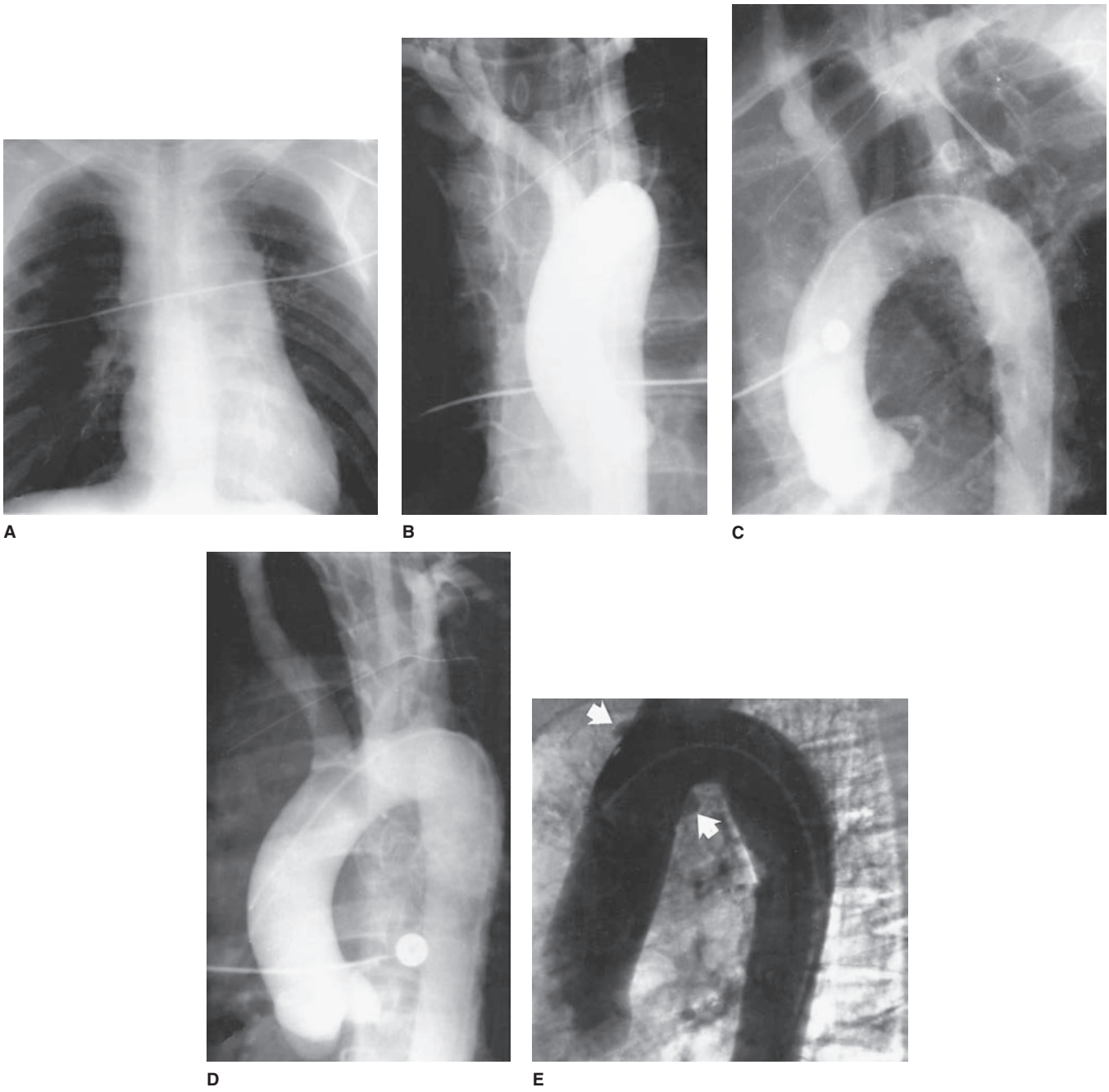


FIGURE 30-12 Misdiagnosis by aortography. (**A**) Chest radiograph of a patient with a tiny puncture wound from a Philips screwdriver at the left sternal border in the second intercostal space. The patient arrived in the emergency room 30 minutes after being wounded and had stable vital signs for the following 48 hours. (**B**) Anteroposterior projection of the aortogram was interpreted as showing no injury. (**C**) Left anterior oblique projection of the aortogram was also interpreted as showing no injury. (**D**) Near-lateral projection of the aortogram was also read as normal by staff radiologist. (**E**) Subtraction aortography in the lateral projection demonstrates tiny outpouching of the thoracic aorta anteriorly at the base of the innominate artery and posteriorly on the undersurface of the transverse aortic arch (arrows). Penetrating injury of the transverse aortic arch was confirmed intraoperatively. (Reproduced with permission from Mattox KL. Approaches to trauma involving the major vessels of the thorax. *Surg Clin North Am*. 1989;69:83. Copyright © Elsevier.)

vascular and cardiothoracic surgeons. One limit has been the perception that a full endovascular suite is required. Although this multimillion-dollar investment may be helpful, the majority of these endovascular repairs can be performed with a small inventory of devices including a portable vascular bed and a portable vascular C-arm.

Analogies between the open and endovascular approach can be easily drawn. For an open approach, an incision is made to access the area of concern. For the endovascular approach, vascular access in an appropriate site is obtained. To access the injury via the open approach, a retractor is placed and dissection occurs from the outside to identify target vessels.

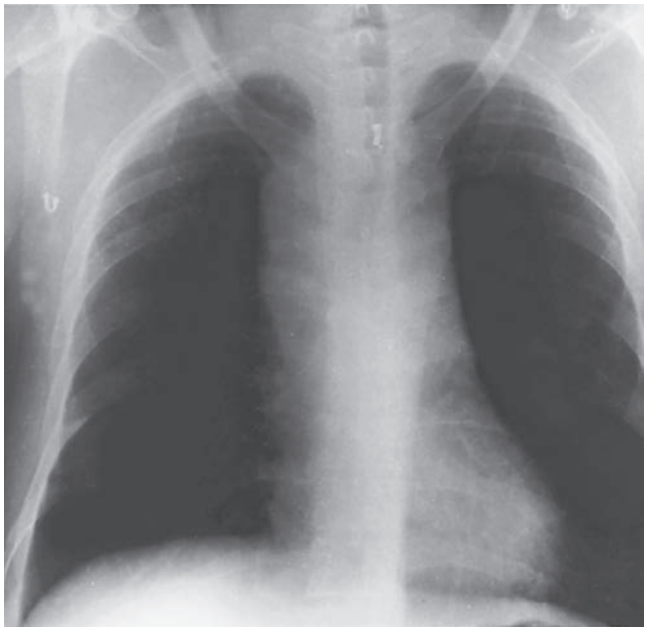


FIGURE 30-13 Plain chest x-ray of a patient with a penetrating wound of the ascending aorta.

In the endovascular approach, access with a wire sheath and a catheter is used to approach the injury from inside the vessel. To achieve vascular control via the open approach, vascular clamps are used from the outside. To achieve control via the endovascular approach, a balloon is used. To repair an injury via the open approach, a vascular suture or vascular graft is used. For the endovascular approach, a covered stent is used. To ligate branches directly, suture or clips are used. For the endovascular approach, coils or glue are used. When one becomes familiar with the endovascular tools and parallels to open instruments, these repairs becomes less daunting.

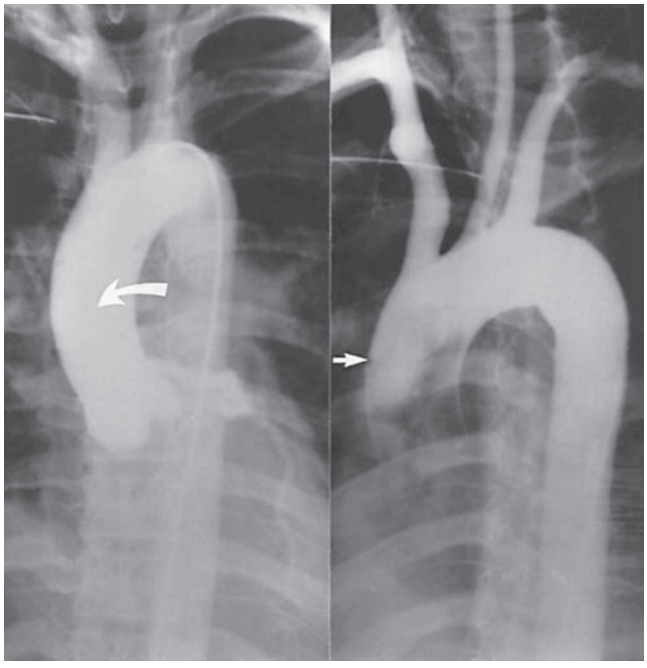


FIGURE 30-14 Aortogram of the patient in Fig. 30-13 demonstrating no apparent injury in the anteroposterior projection, but revealing a defect in the anterior aortic wall on the left anterior oblique projection (*arrows*).

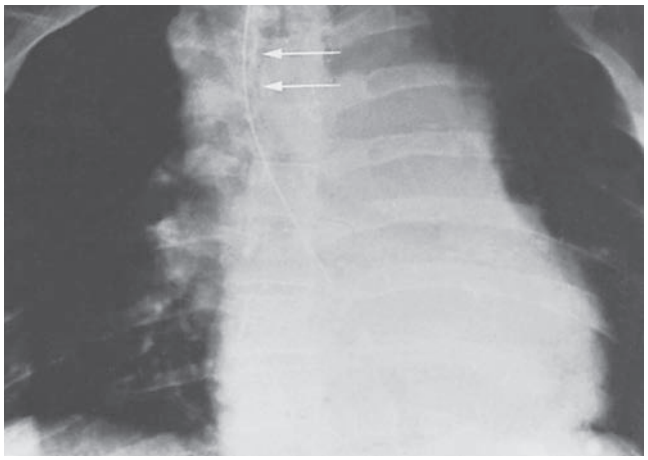


FIGURE 30-15 Plain chest x-ray in a patient with a blunt injury to the descending thoracic aorta. Note the rightward deviation of both the trachea and nasogastric tube in the esophagus.

OPEN SURGICAL REPAIR: GENERAL PRINCIPLES

As previously noted, indications for urgent transfer to the operating room for thoracotomy include hemodynamic instability, significant hemorrhage from chest tubes, and radiographic evidence of a rapidly expanding mediastinal hematoma (Fig. 30-15).

DAMAGE CONTROL

Patients with "physiologic exhaustion" often require damage control management for survival. The two approaches to thoracic damage control are as follows: (1) definitive repair of injuries using quick and simple techniques that control hemorrhage during a single operation; and (2) abbreviated thoracotomy; reversal of hypothermia, acidosis, and coagulopathy; and a planned reoperation for definitive repairs.[55]

Pulmonary tractotomy allows rapid management of associated penetrating injuries to the lung.[65] Severe vascular injuries in the pulmonary hila can be quickly controlled by performing a pneumonectomy using stapling devices. Temporary vessel ligation or placement of intravascular shunts in the common carotid or subclavian arteries can control bleeding and maintain arterial flow until the reoperation. En masse suture closure of the chest wall muscles is more hemostatic than towel-clip closure. A Bogotá bag or vacuum pack may be used as a temporary closure of a median sternotomy in cases with associated cardiac dysfunction.

Endovascular Repair

DESCENDING THORACIC AORTA

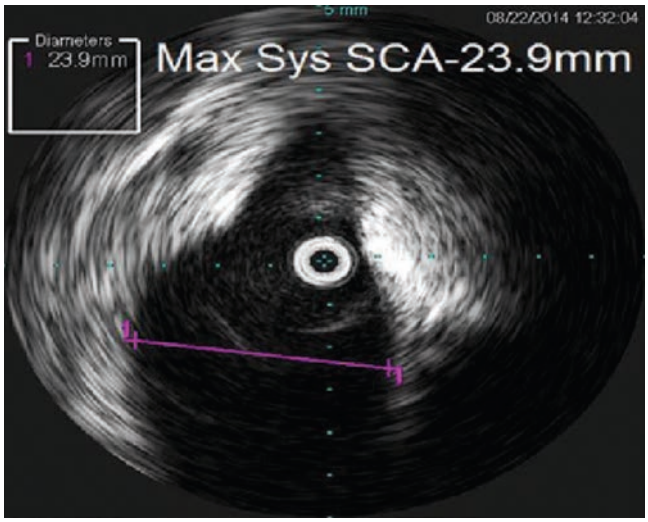
Beginning in the late 1990s, case reports and small series of thoracic endografting for acute transections of the proximal

descending thoracic aorta were reported.[66-69] These were often custom devices using aortic or iliac artery extenders.[70] Not infrequently, the left subclavian artery was occluded by the endograft, with the need for a subsequent left carotid–subclavian bypass in some patients. Iatrogenic injury to the access site of the femoral or iliac artery was occasionally reported. Although the majority of reports have focused on the proximal descending thoracic aorta, isolated reports exist for repair of injury to the ascending aorta and transverse arch.[71]

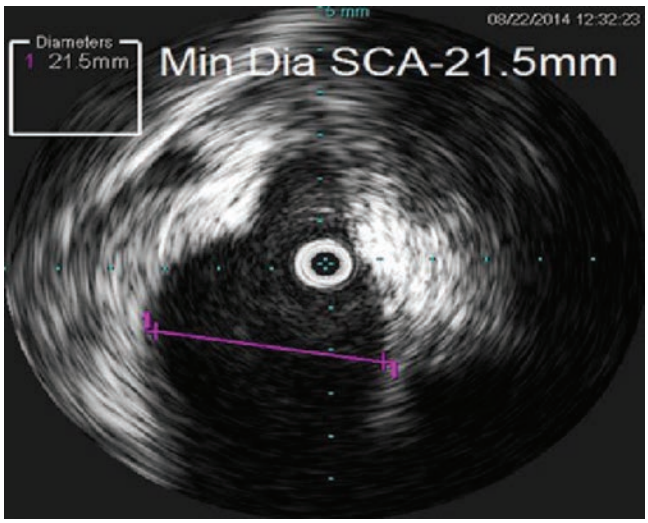
In the United States, several commercial devices have been approved by the US Food and Drug Administration for thoracic aortic aneurysms and are used off label in patients with traumatic injuries to the descending thoracic aorta. The average diameter of the thoracic aorta among patients with aortic tears is 19.3 mm. Because manufacturers recommend 15% to 20% oversizing, thoracic endovascular devices need an aortic diameter of greater than 18 mm. Smaller aortas treated with endografts require custom or off-label abdominal devices, and smaller aortic devices are being developed. With greater oversizing, compression and infolding have been reported, and infolding can result in thrombosis of the aorta. Over 85% of blunt injuries in the descending thoracic aortic are less than 1 cm from the orifice of the left subclavian artery. An endovascular seal zone length on either side of the pathology of 1 to 2 cm is recommended. Additionally, the young patient's aorta has significant angulation in the potential proximal seal zone, which can cause leading edge "beaking" and infolding. This has implications for where to land in the space between the stent on the endograft in relation to this angulation. Thus, consideration for covering the orifice of the left subclavian artery 40% to 80% of the time can be influenced by the intracerebral and spinal circulation as well as a history of a prior coronary artery bypass using the left internal mammary artery. Dominant left vertebral arteries or those with blind posterior intracommunicating arteries may require revascularization with the aforementioned bypass from the left common carotid artery to the left subclavian artery. More recently, endovascular techniques with fenestration at the orifice of the left subclavian artery or chimney grafts have been used.[71]

As previously noted, preoperative planning includes vascular, cardiothoracic, and acute care surgeons and a protocol-driven CTA of the chest, abdomen, and pelvis. Goals of the CT are to define the following: (1) size and extent of the aortic injury; (2) angulation of the arch at its junction with the descending thoracic aorta; (3) diameter and length of aorta proximal and distal to the injury to help determine seal zones; (4) location of the injury in relation to the orifice of the left subclavian artery; (5) size of the vertebral arteries; and (6) angulation of aortic branches. All of this information helps to determine which introducer sheaths and devices are appropriate to cover the aortic injury.

The patient with a blunt injury to the descending thoracic aorta is often young and has a very compliant aorta. As noted, these patients are often underresuscitated, and their aortic diameter is often very small. It has been noted that the diameter of the descending thoracic aorta on thoracic CT varies, perhaps with the cardiac cycle. Intravascular ultrasound (IVUS) can be very helpful to interrogate aortic size in systole, as well as the location of branch vessels and appropriate seal zones. Following experience with the use of IVUS for ruptured abdominal aneurysms, many endovascular surgeons have begun the routine use of IVUS during endograft repair of blunt injury to the descending thoracic aorta.[72,73] The IVUS probe is passed on the working wire, and aortic diameter and seal zones are assessed. The IVUS catheter can then be used as the exchange catheter for the stiff wire used to deploy the endograft. The dynamic nature of the aortic diameter in these patients is striking (Fig. 30-16). Because the screening chest CT for trauma is often not adequate for planning, our service routinely uses IVUS to do final planning of seal zones and selection of appropriate stent graft.



A



B

FIGURE 30-16 Intravascular ultrasound of the descending thoracic aorta at the left subclavian artery during the cardiac cycle. Note the marked difference in diameter between end systole (**A**) and end diastole (**B**). This can have a significant effect on endograft sizing.

A recent report comparing sizing based on CT with intraoperative IVUS noted that the size of the planned endograft was changed based on IVUS over 50% of the time, particularly for more proximal aortic repairs covering the left subclavian artery.[72]

Access can often be a problem in young patients, especially females, with small iliac/femoral arteries that preclude safe placement of the introducer sheath. Currently, the smallest commercially available thoracic endografts require a 7- to 8-mm diameter of the external iliac artery. Direct introduction or sewing of an extra-anatomic graft to the common iliac artery or abdominal aorta to allow deployment of an endovascular stent graft may be necessary in an occasional patient. It should be noted that the majority of morbidity/mortality from thoracic endograft repair is from disruption of iliac vessels during placement of the endograft.

In a composite report using a variety of approved and customized endografts, 239 patients were treated for blunt injury to the proximal descending thoracic aorta (Table 30-6).[74] Among the 239 patients, there were nine deaths (3.8%) and one paraplegia (<0.5%). Even with potential selection bias, the lower mortality and almost nonexistent paraplegia rate makes consideration for endovascular repair very compelling, particularly in a delayed fashion.[59,61,75,76] Yet to be answered are the engineering challenges of graft compression and infolding, as well as the need for smaller sizes, conformation of the endograft to the curvature of the arch, tailored/branched grafts, and improved delivery systems. The short- and mid-term follow-up have favored endovascular stenting, and this has been enthusiastically adopted by most centers.[77] The long-term fate of endovascular stent grafts, however, is currently being assessed.[78]

With changes in the presenting patient population, as well as endograft-related diagnosis, imaging, and engineering technology, the timing, diagnosis, and management of blunt aortic injuries have been evolving rapidly. The report by Demetriades et al[59] of the American Association for the Surgery of Trauma (AAST) multicenter study, with its two follow-up manuscripts, documented significant shifts in diagnosis and management. In his Scudder Oration, "Blunt Thoracic Aortic Injuries: Crossing the Rubicon," comparing the early AAST report (Fabian et al[79]) with the latter AAST reports (Demetriades),[59,61,80] Demetriades noted the following: cross-sectional imaging is now routinely used for screening; purposeful delay in definitive treatment is commonly performed; endovascular repair is now the most common repair performed, practically displacing open repair; endovascular repair has a 60% decrease in mortality and paraplegia; and endovascular repair results in an increase in access and device-related complications and concern for long-term sequale.[79,80] These reports have been the most comprehensive to date and are a template to track results as the technology evolves. Studies documenting the rate of aortic dilation after endograft repair are being reported and will be important for assessing the long-term durability of endograft repair.[78] With better imaging, there is recognition that minimal blunt aortic injuries often heal spontaneously and do not require intervention. With improvements in vascular access and devices that are able to accommodate the smaller patient, endovascular repair can be applied to more patients. Multiple follow-up studies continue to support endovascular repair in blunt injury to the descending thoracic aorta.[81] It is clear, however, that the treatment for a specific patient will continue to be individualized, and multiple approaches (nonoperative/delayed/open/endograft) will continue to be needed.[59,61,76,81]

TABLE 30-6: Comparison of Open versus Endovascular Treatment of Blunt Injury to Thoracic Aorta

	Open operations	Endograft repair
Mortality	0%–55%	0%–12%
Average mortality	13%	3.8%
Paraplegia	0%–20%	<1%
Average paraplegia	10%	1 out of 239 cases
Complications	ARDS	LSCA occlusion
	CNS problems	Graft compression
	Neurologic	Access site problems

ARDS, adult respiratory distress syndrome; CNS, central nervous system; LSCA, left subclavian artery.

The Endovascular Procedure The patient is placed supine on a vascular table with the imager and display on the patient's left. Extraneous monitoring devices such as ECG leads are removed so as not to impede imaging of the left chest, and the patient is prepared so access to both groins is available. In addition, with consideration for fenestration of the orifice of the left subclavian artery, access to the left arm is made available. When preoperative imaging has documented a steep angulation of the arch that may lead to difficult tracking of the endograft, access to the right arm is needed so that through-and-through access can be obtained to assist in tracking the endograft around the arch. It has been found that tracking of the device works best with access from the right groin. In patients who can be heparinized, percutaneous access through both groins with a pre-close technique is often used. Otherwise, percutaneous diagnostic access is obtained from the left groin with device access obtained via cutdown of the right groin.

For patients with small vessels and difficult access, a right lower quadrant incision can be made to expose the retroperitoneal right common iliac artery or the distal abdominal aorta. The distal abdominal aorta can be cannulated directly using a pursestring suture. For access to the right common iliac artery, a Dacron graft sewn to the vessel over a partially occluding vascular clamp is appropriate.

Cross-sectional imaging is carefully examined to note which, if any, portion of the aortic wall is intact so that the wire can be directed to that area to traverse the site of injury. The injury is traversed using a directable catheter and a floppy wire. This is exchanged for a stiffer wire and a

pigtail catheter with radiographic markings. The right common femoral artery is accessed with an 8F sheath, and the site of injury traversed in a similar manner. Catheter exchange occurs, and placement of the IVUS catheter is performed. The aorta is interrogated, and the anatomy of the injury, anticipated seal zones, and position of the left subclavian artery are then assessed. Final graft selection is confirmed or modified based on these findings. Wire exchange to the stiff device delivery wire is performed using the IVUS catheter. Using the radiographic markers on the pigtail catheter and the previously measured angles on the CT scan, the gantry of the imaging device is placed orthogonal to the arch to prevent parallax error. Arteriography is then performed to confirm the anatomy. The right-sided access is dilated and the device delivered. After magnifying down to the proximal seal zone, arteriography is performed again, and the area of the proximal sealed zone is confirmed. The proximal seal zone of the device is then delivered. Without moving the imaging head, the view can be unmagnified to show the distal graft as it is deployed. The delivery device can be removed with replacement of an equivalent size sheath maintaining wire access. A balloon can be used to dilate the seal zones if needed. A confirmatory aortogram is obtained to evaluate the repair after recapturing and repositioning the flush catheter.

Future Investigation Although the short-term results of endografts have been quite favorable, there is concern about the long-term results in young patients. As described earlier, studies note that the aorta dilates over time and the behavior of the endografts in the aging patient is being tracked. There have been reports investigating this issue, and a national registry to track these repairs has been suggested.[77,78,80]

Even with potential selection bias in favor of endografts, the low mortality and almost nonexistent paraplegia rate make the use of endografting very compelling. The reported complications of graft migration, infolding, compression, occlusion of the left subclavian artery, and problems at the entry site are all technical and engineering challenges that may potentially be solved by new commercial devices.

THORACIC OUTLET/SUBCLAVIAN ARTERY

Early endovascular approaches to thoracic outlet injuries had variable results.[82] Some early repairs were attempted in the radiology suite where there was limited ability to monitor and treat unstable patients. As surgeons have become more involved in the use of these devices and they are performed in the operating room, they are used more frequently. As previously noted, there has been recognition that a hybrid suite is not always required and that these grafts can be placed with a portable C-arm. This creates flexibility because potentially any well-equipped operating room could be a site for these procedures with a vascular table and a portable vascular C-arm, as described earlier. Thus, an unstable patient can be brought to the operating room with all its benefits and in the care of the trauma team, anesthesiologist, and surgeon. Even unstable patients with complete transections or patients with large hematomas can be treated. In an effort to eliminate the thoracic incisions for proximal control of injuries to the subclavian artery, surgeons have developed the skills to place an endovascular balloon via femoral access for proximal control. The injury can then be managed with a supraclavicular incision.

The development of small, self-expanding, covered stents has facilitated this endovascular approach. A wire placed via femoral access can traverse a partial lateral injury. For transected vessels or thrombosed injuries, through-and-through snare techniques allow complex repairs in unstable patients to occur.[83,84] For thoracic outlet injuries, a CT scan of the chest with vascular contrast in the stable patient defines the injury and allows planning. The patient is positioned supine on a vascular bed, with the appropriate arm available to allow for access. In the operating room, access to the femoral artery is obtained. A wire and catheter are directed into the vessel that is injured, and a balloon can be placed for proximal control. Arteriography can verify the injury and the position of the vertebral artery if the subclavian artery is injured. For the subclavian artery, a cutdown on the ipsilateral brachial artery is performed so that brachial access for a wire snare from the arm can be achieved. The wire from the groin is introduced into the injury or hematoma and grasped by the snare from the arm establishing through-and-through access traversing the injury. A self-expanding covered stent can then be placed. These techniques have been used for transected vessels and for hypotensive patients, as well as for patients with multiple injuries including combined injuries to the carotid and subclavian arteries. These approaches are also extremely helpful for patients who have had previous thoracic operations. In the report by Gilani et al[84] of endovascular management of eight unstable patients or those with transected subclavian arteries, there was 100% survival and only one graft thrombosis at 14 months. Others have reported similar results with endovascular repair, noting improved mortality and lower complication rates.[85,86]

Open Surgical Repair

In the preoperative phase, whenever possible, patients and their families should be made aware of the potential for neurologic complications such as paraplegia, stroke, or injury to the recurrent laryngeal nerve or brachial plexus following surgical reconstruction of thoracic great vessels. Therefore, careful documentation of preoperative neurologic status is helpful. With any suspicion of vascular injury, prophylactic antibiotics are administered preoperatively. In hemodynamically stable patients, fluid administration is limited until vascular control is achieved in the operating room, as noted earlier. An autotransfusion device should be available. During the induction of anesthesia, wide swings in blood pressure should be avoided. Although profound hypotension is clearly undesirable, hypertensive episodes can have equally catastrophic consequences.

The operative approach to great vessel injury depends on the hemodynamic status of the patient and the specific

TABLE 30-7: Recommended Incisions for Injuries to Thoracic Great Vessel

Injured vessel	Incision
Uncertain injury (hemodynamically unstable)	Left anterolateral thoracotomy Transverse sternotomy ± Right anterolateral thoracotomy (clamshell)
Ascending aorta	Median sternotomy
Transverse aortic arch	Median sternotomy ± Cervical extension
Descending thoracic aorta	Left posterolateral thoracotomy (fourth intercostal space)
Innominate artery	Median sternotomy with right cervical extension
Right subclavian artery or vein	Median sternotomy with right cervical extension
Left common carotid artery	Median sternotomy with left cervical extension
Left subclavian artery or vein	Left anterolateral thoracotomy (third or fourth intercostal space) with separate left supraclavicular incision Endovascular balloon occlusion may eliminate need for thoracotomy for proximal control
Pulmonary artery	
Main/intrapericardial	Median sternotomy
Right or left hilar	Ipsilateral thoracotomy
Pulmonary vein	Ipsilateral thoracotomy
Innominate vein	Median sternotomy
Intrathoracic vena cava	Median sternotomy
Azygos vein	Ipsilateral thoracotomy

injury. Patient positioning and selection of incision are particularly important in surgery for great vessel injuries because adequate exposure is needed for proximal and distal control (Table 30-7). Prepping and draping of the patient should provide access from the neck to the knees. For the patient in extremis with an undiagnosed injury, the mainstay of thoracic trauma surgery is the left anterolateral thoracotomy, with the patient in the supine position. In stable patients, CT/CTA may dictate an operative approach by another incision.

Appropriate graft materials should be available, recognizing known limitations. The failure mode of an infected prosthetic graft is a pseudoaneurysm, whereas an infected saphenous vein graft, a devitalized collagen tube susceptible to bacterial collagenase, will dissolve and cause uncontrolled hemorrhage. Therefore, for vessels larger than 5 mm, a prosthetic graft is the conduit of choice, especially in potentially contaminated wounds.

Due to patency considerations, a saphenous vein graft may need to be used when smaller grafts are required. For fragile vessels, such as the subclavian artery and the aorta in young patients, a soft knitted Dacron graft is preferred.

ASCENDING AORTA

Patients with blunt injuries to the ascending aorta rarely survive transport to the hospital. Operative repair requires total cardiopulmonary bypass and insertion of a Dacron graft. If the sinus of Valsalva or the aortic valve is involved, aortic root replacement with reimplantation of the coronary ostia may be required.[63]

Penetrating injuries involving the ascending aorta are uncommon (see Figs. 30-11 and 30-12). Survival rates approach 50% for patients having stable vital signs on arrival at a trauma center.[87] When in extremis, these injuries are found during an exploration through a left anterolateral or clamshell incision. For urgent explorations, these injuries can be approached via a median sternotomy. Although primary repair of anterior lacerations can be accomplished without adjuncts, cardiopulmonary bypass may be required if there is an additional posterior injury. The possibility of a peripheral bullet embolus must be considered in patients with a single gunshot wound to the thoracic aorta.

TRANSVERSE AORTIC ARCH

When approaching an injury to the transverse aortic arch, extension of the median sternotomy to the neck is necessary to obtain exposure of the arch and brachiocephalic branches. If necessary, exposure can be further enhanced by division of the left innominate vein. When hemorrhage limits exposure, balloon tamponade is useful as a temporary measure. Simple lacerations may be repaired by lateral aortorrhaphy using a 4-0 polypropylene suture. With difficult lesions, such as posterior lacerations or those with concomitant injuries to the pulmonary artery, cardiopulmonary bypass may be required. As with injuries to the ascending thoracic aorta, survival rates approaching 50% have been reported.[87] In stable patients, isolated reports of endograft repair with arch vessel debranching have been described.

DESCENDING THORACIC AORTA

Injury to the descending thoracic aorta is often accompanied by other organ injuries. If the patient has a stable thoracic hematoma and concomitant abdominal injury, laparotomy should be the initial procedure. For the patient with a rapidly expanding hematoma, however, repair of the thoracic injury should be the primary therapeutic goal. Sequencing is driven by the lesion that is most likely to cause exsanguination.

Although uncommonly performed now, the standard technique of repair involves *clamping and direct reconstruction* (Table 30-8). The three commonly employed adjuncts to this approach are as follows: (1) pharmacologic agents; (2) temporary, passive bypass shunts; and (3) pump-assisted atriofemoral bypass or cardiopulmonary bypass. In the latter approach, the following two options exist: (1) traditional pump bypass, which requires heparin; and (2) use of a centrifugal (heparinless) pump circuit (see Atlas Figures 36 and 37). The surgeon must choose the approach most appropriate to the specific clinical situation and one that works the best in his or her system.

TABLE 30-8: Current Therapeutic Approaches to the Management of Blunt Injuries to the Thoracic Aorta

1. Endograft repair
   a. With/without coverage of left subclavian artery
   b. With/without restoration of flow to left subclavian artery
2. Surgical (clamp and direct reconstruction with or without an interposition graft)
   a. Pharmacologic control of proximal hypertension
   b. Passive bypass shunts
   c. Pump-assisted bypass
      (1) Traditional cardiopulmonary bypass (with total-body heparinization)
      (2) Left heart bypass using centrifugal pump (with/without heparinization)
3. Nonoperative and/or purposeful delay of operation (with pharmacologic treatment and close radiologic surveillance)

The initial objective is proximal control. Therefore, the transverse aortic arch is exposed, and umbilical tapes are passed around the arch between the left common carotid and left subclavian arteries. Similarly, the left subclavian artery is encircled with an umbilical tape. Care should be taken to avoid injuring the left recurrent laryngeal nerve at this location, although it is often difficult to visualize in the hematoma. If it is suspected that the tear extends to the aortic arch or ascending aorta, cardiopulmonary bypass should be available in the operating room. If the patient has had previous coronary artery bypass surgery with use of the left internal mammary artery as a conduit, repair may require full cardiopulmonary bypass to protect the heart.

Vascular clamps are applied to the transverse arch proximal to the left subclavian artery, distal descending thoracic aorta, and left subclavian artery. Close communication between anesthesiologist and surgeon is essential to maintain stability of hemodynamic parameters before, during, and after clamping, particularly with use of vasodilators to prevent cardiac strain. The hematoma is entered, and back-bleeding from intercostal arteries is controlled with oversewing. Care is taken to avoid indiscriminate ligation of intercostal vessels, and only those required for adequate repair of the aorta should be ligated. After defining the injury, the clamps are moved closer, allowing maximal collateral flow. The proximal and distal ends of the aorta are completely transected and dissected away from the esophagus, allowing full-thickness suturing while minimizing the risk of a secondary aortoesophageal fistula. The injury is then repaired by either end-to-end anastomosis or insertion of an interposition graft, which is performed in more than 85% of reported cases. Prior to clamp removal, blood transfusions and infusion of crystalloid solutions are often needed to avoid hypotension.

For patients undergoing repair of a blunt injury to the descending thoracic aorta, the historic reported mortality has ranged from 0% to 55% (average 13%).[74,88,89] As expected in these victims of major blunt trauma, the mortality is primarily associated with other injuries and is often due to a traumatic brain injury, infection, or multiple organ failure.

The most feared complication of open repair of the descending thoracic aorta is paraplegia. Utilization of protective adjuncts during repair remains a topic of considerable debate. There have been proponents of the use of passive shunts and cardiopulmonary bypass, with and without heparinization. The mortality rate with the use of routine cardiopulmonary bypass is probably secondary to hemorrhage in the brain, abdomen, or at fracture sites. Over the past 35 years, experience using centrifugal pumps for left heart bypass without heparinization has provided an attractive alternative for surgeons who wish to use controlled flow bypass without systemic anticoagulation. This also allows unloading of the left heart during clamping, which can be helpful in patients with cardiac disease. The use of bypass systems, however, is not without complications. In the trauma patient, difficulty inserting cannulae may occur due to patient position, the presence of a periaortic hematoma, and time constraints imposed by an expanding, pulsatile, uncontrolled hematoma. Intraoperative and postoperative complications include bleeding at the cannulation sites and formation of a false aneurysm.

Use of simple clamp-and-repair for injuries to the descending thoracic aorta (without the use of systemic anticoagulation or shunts) is a technique that continues to be used with excellent results. In 1992, Sweeney et al[90] reported using simple clamp-and-repair in 75 patients, only one of whom developed postoperative paraplegia.

Ultimately, the determinants of postoperative paraplegia are multifactorial; therefore, the exact causes cannot be precisely identified in an individual patient (Table 30-9). Paraplegia has been associated with perioperative hypotension, injury or ligation of the intercostal arteries, and duration of clamp occlusion during repair.[90] There are, however, reports of patients surviving surgery without paraplegia, despite having long segments of aorta replaced as well as ligation of multiple intercostal arteries.

The length of aortic cross-clamp time does not *directly* correlate with postrepair paraplegia. A cross-clamp time less than 30 minutes has been argued to provide a safe margin against paraplegia, and shunting techniques have been recommended when longer cross-clamp times are necessary.[91] The use of a shunt, however, does not offer protection for the area of the spinal cord supplied by the arteries between the clamps. Furthermore, patients requiring longer clamp times or interposition grafts have more extensive injuries than those requiring shorter clamp times or end-to-end anastomoses. Thus, it is likely that an increased incidence of paraplegia associated with longer clamp times is secondary to more extensive disruption of intercostal arteries and other flow to the anterior spinal artery caused by the original injury.

Various monitoring techniques are available to assess the effect of occlusion of the descending thoracic aorta on the spinal cord, including the measurement of somatosensory and motor-evoked potentials. Although correlation appears to exist with loss of somatosensory-evoked potentials, duration

TABLE 30-9: Possible Contributing Factors to the Development of Paraplegia Following Operations for Injuries to the Thoracic Great Vessel

Injury factors	Direct segmental/radicular/spinal artery injury
	Spinal cord contusion/concussion
	Spinal canal compartment syndrome
	Severity of aortic injury
	Specific anatomic location of aortic injury
Patient factors	Location of arteria radicularis magna (artery of Adamkiewicz)
	Continuity of anterior spinal artery
	Caliber of individual segmental radicular arteries
	Congenital narrowing of spinal canal
	(?) Increased blood alcohol levels
	Total perispinal collateral blood supply
Operative factors	Required occlusion of segmental arteries
	Pharmacologic agents required
	(?) Declamping hypotension
	(?) Required cross-clamp times (in combination with anatomic and injury factors cited in this table); length of required interposition grafting or required exclusion
	(?) Level of systolic (or mean) proximal aortic blood pressure
	(?) Level of distal aortic mean blood pressure
	(?) "Flow" in the aorta distal to clamp
Postoperative factors	Progressive swelling of the spinal cord
	Spinal canal compartment syndrome
	Delayed or secondary occlusion of injured or contused segmental, radicular, or spinal arteries
	Pharmacologic-induced spasm of spinal cord nutrient arteries

of loss of conduction, and postoperative paraplegia, the use of this modality is not common to all trauma centers. The interpretation of results is still being debated, and actual applicability requires further study. Regardless of the technique used, paraplegia occurs in approximately 10% of these patients (range, 0%–22%).[74,89] No prospective, randomized trial has identified the superiority of any single method. Therefore, the choice of operative technique does not infer legal liability when paraplegia occurs.

INNOMINATE ARTERY (SEE ATLAS FIGURES 30 AND 31)

Median sternotomy is employed for access to injuries to the innominate artery. A right cervical extension can be used when necessary. As previously described, blunt injuries typically involve the proximal innominate artery and, therefore, actually represent aortic injuries and require obtaining proximal control at the transverse aortic arch (Fig. 30-17). In contrast, penetrating injuries of the innominate artery may occur

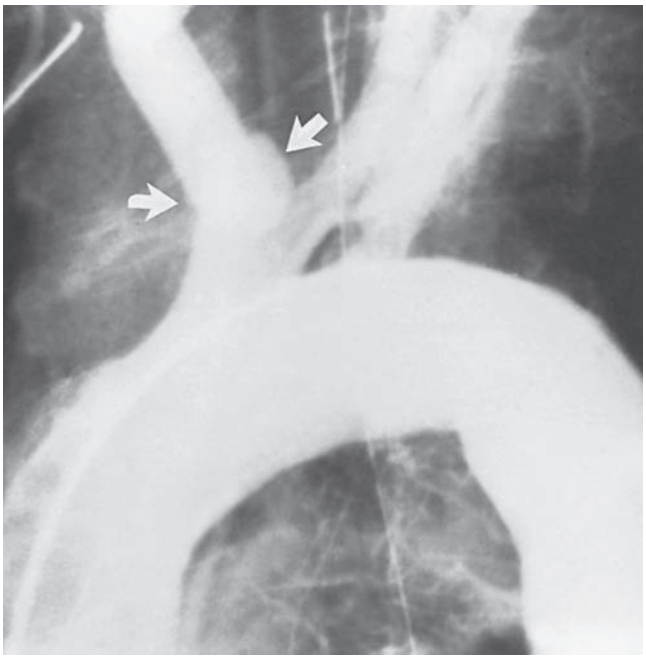


FIGURE 30-17 Aortogram of a patient with blunt innominate injury demonstrating the tear involving the proximal innominate artery.

throughout its course. Once again, exposure is enhanced by division of the crossover left innominate vein.

In selected patients with penetrating injuries, a running lateral arteriorrhaphy using 4-0 polypropylene suture is occasionally possible. More often, injuries to the innominate artery require repair via the bypass exclusion technique developed by Mattox and described in Johnston et al[92] (Fig. 30-18). Bypass grafting is performed from the ascending aorta to the distal innominate artery (immediately proximal to the bifurcation into the subclavian and right common carotid arteries) using a Dacron tube graft. The area of injury is avoided until the sites for insertion of a bypass graft are exposed. A vascular clamp is placed proximal to the bifurcation of the innominate artery to allow collateral flow to the brain via the right subclavian into the right common carotid artery. Hypothermia, systemic anticoagulation, and shunting are not required. After the bypass is completed, the area of hematoma is entered, and the injury controlled with a partial occluding clamp (usually at the origin of the innominate artery) and oversewn. If the right innominate vein is injured as well, it may be ligated if the azygous system is intact.

The treatment of a tracheostomy-induced late tracheal–innominate artery fistula deserves special consideration. These fistulas are usually caused by the concave surface of a low-riding tracheostomy tube eroding into the innominate artery. Arteriography during a "stable interval" is generally not helpful in making a precise diagnosis. Therefore, the possibility of a tracheal–innominate fistula should be evaluated via bronchoscopy through a partially withdrawn endotracheal tube. A rapid chest CT, looking for a violated plane between the trachea and innominate artery, can be obtained if the

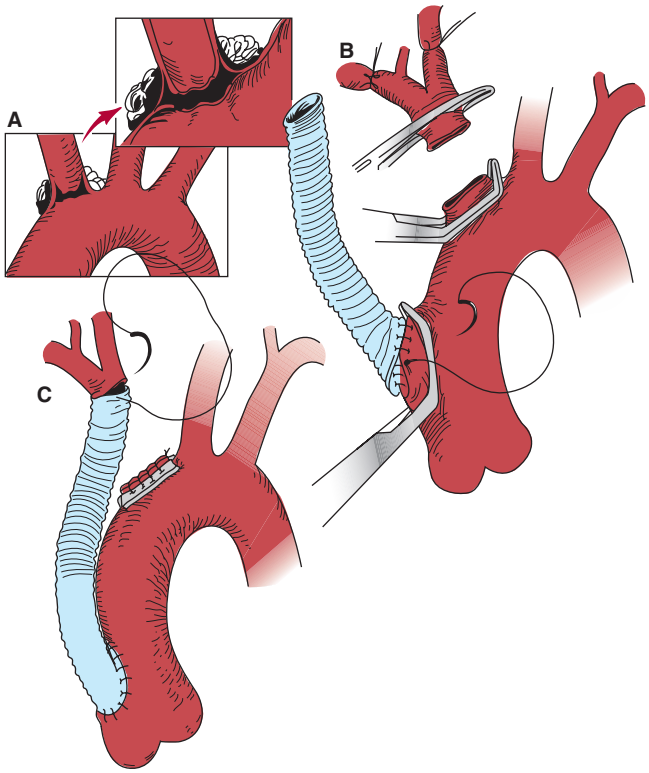


FIGURE 30-18 Drawing depicting the bypass exclusion technique employed in patients with innominate artery injuries. (Copyright © Baylor College of Medicine, 1981.)

bleeding has stopped or been controlled. With massive bleeding, control is achieved by performing orotracheal intubation, removing the tracheostomy tube, and directly tamponading the bleeding digitally through the transverse cervical incision during transport to the operating room. Through a median sternotomy with a right neck extension, the innominate artery is ligated at its origin from the aorta and distally just before the division into the right common carotid and right subclavian arteries. Despite a greater than 25% chance of neurologic complications, no attempt should be made at revascularization, since a delayed graft infection with its dreaded associated complications inevitably occurs. There are sporadic descriptions of the use of endografts to temporize these critical patients. This is occurring with increasing frequency as a damage control approach, accepting a likely infected graft.

SUBCLAVIAN ARTERY (SEE ATLAS FIGURES 17 AND 20)

Subclavian vascular injuries can involve any combination of the intrathoracic, thoracic outlet, cervical (zone I), and axillary/upper extremity areas. In stable patients, a thoracic CTA allows for planning appropriate incision(s) to obtain adequate exposure and control.

For open repair, cervical extension of the median sternotomy is employed for exposure of right-sided subclavian injuries. For injury to the first portion of the left subclavian artery, proximal control is obtained through a left anterolateral thoracotomy above the nipple in the third or fourth intercostal space, whereas a separate supraclavicular incision provides distal control (see Atlas Figure 19). Although these incisions can be connected to create a formal "book" thoracotomy, this results in a high incidence of postoperative "causalgia"-type neurologic complications and its use should be limited. As previously noted, the endovascular placement of a balloon for proximal control is now commonly used to obviate the need for a thoracic incision.

In obtaining exposure, it is important to avoid injuring the phrenic nerve (anterior to the scalenus anticus muscle). This is particularly true because there is also a high associated rate of injury to the brachial plexus. Thus, documentation of preoperative neurologic status is important, as noted.

In most instances, repair consists of either lateral arteriorrhaphy or insertion of an interposition graft. It is unusual that an end-to-end anastomosis can be employed. With an intrathoracic associated injury to the left lung, this can be managed with stapled wedge resection or pulmonary tractotomy.[65] One pitfall in subclavian injuries is failure to anticipate the exposure necessary for proximal control. The guiding principle is to achieve vascular control, either endovascular or open, at a level proximal to the injury hematoma or site of bleeding. As noted earlier, an endovascular balloon may permit proximal control closer to an injury, decreasing collateral bleeding. When approaching the subclavian/axillary artery via the supraclavicular or deltopectoral groove approach without proximal control, exsanguination may occur. Division or resection of the mid-clavicle may aid in proximal control, whereas combination supra- and infraclavicular incisions may be used to avoid clavicular resection. A mortality rate of 4.7% for patients with injuries to the subclavian artery who have vital signs on admission has been reported, but death is often due to associated injuries.

COMMON CAROTID ARTERY

The open operative approach for injuries of either common carotid artery is a median sternotomy with a right or left cervical extension when necessary. With transection at the origin of the left common carotid artery, a bypass graft repair is preferred over end-to-end anastomosis. Intraoperatively, a temporary intraluminal carotid shunt can be inserted by the initial surgeon if needed until resources or assistants can be gathered in the operating room.

PULMONARY ARTERY

The intrapericardial pulmonary arteries are approached via a median sternotomy. Minimal dissection is needed to expose the main and proximal left pulmonary arteries.[93] Exposure of the intrapericardial right pulmonary artery is achieved by dissecting between the superior vena cava and ascending aorta. Although anterior injuries can be repaired primarily without adjuncts, repair of a posterior injury usually requires cardiopulmonary bypass. Mortality rates for injury to the central pulmonary arteries or veins are greater than 70%.[43]

Injuries to the distal main pulmonary artery present with a massive hemothorax. Because these are part of a major hilar injury, a rapid pneumonectomy using en bloc stapling may be a lifesaving maneuver. The use of a hilar clamp can control hemorrhage as well as prevent air embolism while assessment occurs.

INTERNAL MAMMARY ARTERY

The internal mammary artery in a young patient is capable of flows in excess of 300 mL/min. Injuries to this artery can produce an extensive hemothorax or pericardial tamponade, simulating a cardiac or major vascular injury. Such injuries are usually serendipitously discovered at the time of thoracotomy for a suspected injury to the heart or a great vessel.

INTERCOSTAL ARTERIES

Persistent bleeding through a thoracostomy tube can be caused by laceration of an intercostal artery. Because of difficulty in exposure, precise ligation can be difficult. At times, control can only be achieved by circumferential ligatures around the rib on either side of the injury to the intercostal vessel. This is sometimes accomplished via separate stab wounds and a suture passer to loop the suture around the rib. Exposure can sometimes be obtained by extension of the thoracotomy into that interspace.

THORACIC VENA CAVAE

Isolated injury to the intrapericardial inferior or superior vena cava is infrequently reported. Injury at either location has a high incidence of associated organ injuries and a mortality rate greater than 60%. Injury to the intrapericardial inferior vena cava causes a hemopericardium and cardiac tamponade. Exposure of the posterior intrapericardial inferior vena cava is extremely difficult unless the patient is placed on total cardiopulmonary bypass with the inferior cannula inserted via the groin into the abdominal inferior vena cava. The injury is exposed by a right atriotomy and intracaval balloon occlusion to prevent air entering the cannula. Repair is achieved from inside the cava via the right atrium. Injuries to the superior vena cava are repaired by lateral venorrhaphy. At times, an intracaval shunt is necessary before repair can be accomplished. Occasional patients survive following blunt partial transection at the junction of the right atrium and the superior or inferior vena cava. Primary repair can be accomplished after control of the atrium and cava.[94] For complex injuries, a polytetrafluoroethylene (PTFE) patch or Dacron interposition tube graft can be used and is more expedient than the time-consuming construction of panel grafts from a segment of the greater saphenous vein.

PULMONARY VEIN

Injury to a pulmonary vein is difficult to manage through an anterior incision. With major hemorrhage, temporary clamp occlusion of the entire hilum may be necessary. If a lobar pulmonary vein must be ligated, the appropriate lobe needs to be resected. Injury to a pulmonary vein is often associated with concomitant injuries to the heart, pulmonary artery, thoracic aorta, and esophagus.

SUBCLAVIAN VEINS

The operative exposure of the subclavian veins is the same as that described for injuries to the subclavian artery (ie, median sternotomy with cervical extension for right-sided injuries, and left anterolateral thoracotomy with a separate supraclavicular incision for left-sided injuries). In most instances, control of bleeding requires either lateral venorrhaphy or ligation.

AZYGOUS VEIN

The azygous vein is not usually classified as a thoracic great vessel; however, because of its size and high flow, injury to the azygous vein must be considered potentially fatal. Due to its posterior location, an injury is difficult to diagnose and expose through an emergent anterolateral thoracotomy. The key to diagnosis is recognizing ongoing hemorrhage of dark venous blood from a posterior location in the right chest. A penetrating wound of the chest or thoracic outlet causing a combination of injuries involving the azygous vein, innominate artery, trachea, bronchus, and superior vena cava is difficult to control and obviously has a high mortality rate. Combined incisions are frequently needed for successful repair. When injured, the azygous vein is best managed by suture ligature of both sides of the injury (Fig. 30-19). Concomitant injury to the esophagus and bronchus should be considered and ruled out.[95]

Special Problems

MEDIASTINAL TRAVERSE INJURIES

Stab and gunshot wounds that traverse the mediastinum were in the past thought to have a high probability of injury to the heart, a thoracic great vessel, the tracheobronchial tree, or esophagus, and a mandatory exploration was performed.

The evaluation of stable patients using less invasive means such as surgeon-performed FAST and contrast-enhanced CT is now the standard of care. The CT documents the bullet trajectory and guides the need for surgery or additional diagnostic tests.

INJURY TO THE THORACIC DUCT (SEE ATLAS FIGURE 18)

Injury to a thoracic great vessel may be complicated by a concomitant injury to the thoracic duct. If unrecognized, this may produce significant morbidity due to a chyle leak with marked nutritional depletion, especially in children.[96] Diagnosed by chylous material with high triglycerides draining from a thoracostomy tube, this condition is usually treated medically. Continued drainage through a thoracostomy tube coupled with a diet devoid of long-chain fatty acids usually result in spontaneous closure over several weeks. Prolonged

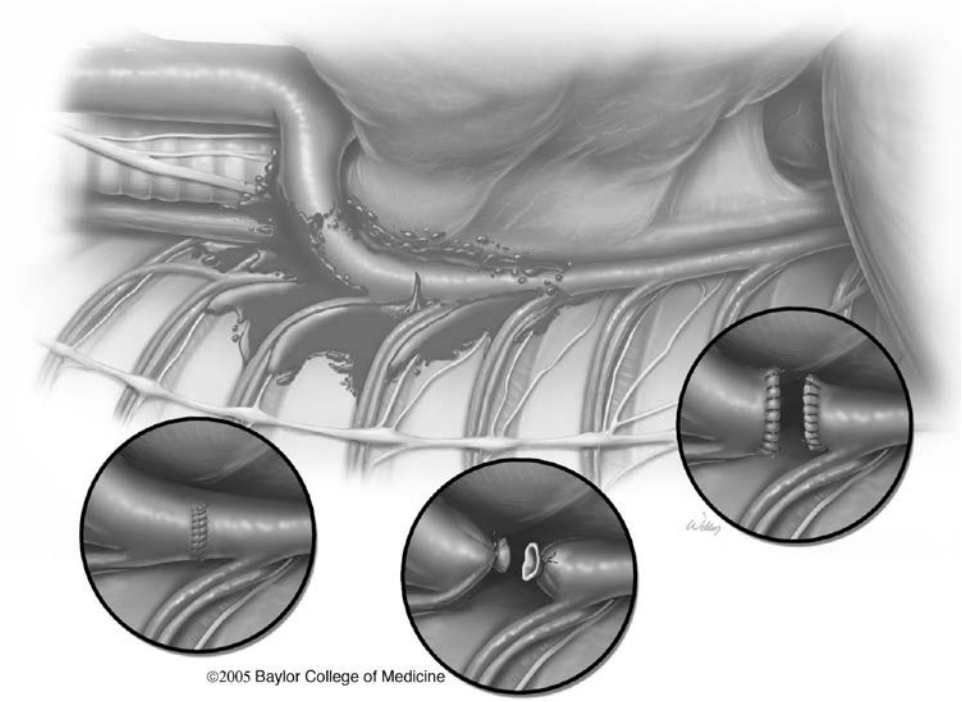


FIGURE 30-19 Injury to the azygous vein with control with lateral repair, ligation and division, and oversewing. (Copyright © Baylor College of Medicine, 2005.)

hyperalimentation beyond 3 weeks, however, has not consistently resulted in spontaneous closure of a fistula from an injured thoracic duct. If a thoracotomy for ligation of duct at the area of injury is required, a fatty meal or heavy cream to increase the chylous flow and facilitate identification of the fistula is given to the patient prior to surgery. The fistula is ligated with 6-0 monofilament suture. Fistulas do recur after ligation, and a reoperation with mass ligation of the thoracic duct at the diaphragm will be necessary. Pleurodesis has also been used as definitive treatment.

SYSTEMIC AIR EMBOLISM

A fistula between a bronchiole and pulmonary vein due to a penetrating wound to the lung may result in a systemic air embolism. The fistula allows air bubbles to enter the left heart and embolize to the coronary arteries, cerebral arteries, and systemic circulation (Fig. 30-20). An intrabronchial pressure above 60 torr increases the incidence of this complication.[97]

Manifestations include seizures and cardiac arrest. Resuscitation requires a left anterolateral thoracotomy, temporary clamping of the hilum of the injured lung to prevent further air embolization, and needle aspiration of air from the left ventricle and ascending aorta. Cardiopulmonary bypass can be considered, but few survivors have been reported.

FOREIGN BODY EMBOLISM

Because of their central location, the thoracic great vessels may serve as both an entry site and final resting place for an intravascular bullet embolus.[98] These migratory foreign bodies present a diagnostic and therapeutic dilemma. As a result of intravascular embolization, bullets may produce infection, ischemia, or injury to organs distant from the site of trauma.

Approximately 25% of migratory bullets finally lodge in the pulmonary arteries (see Fig. 30-5).[98] Although small fragments such as those the size of a BB can be left in place without causing problems, significant catheter emboli and larger bullet emboli should be removed to prevent thrombosis of a pulmonary branch artery, sepsis, or other complications. Percutaneous retrieval of the foreign body using transvenous catheters and fluoroscopic guidance has virtually eliminated the need for thoracotomy.

Postoperative Management

Careful hemodynamic monitoring, with avoidance of both hypertension and hypotension, is critical. Although urinary output is a generally a good indicator of cardiac function, hemodynamic monitoring (surgeon-performed ultrasound of heart and inferior vena cava; Flo Trac system, Edwards Lifesciences, Irvine, CA) is often necessary to optimize hemodynamic parameters and manage fluids, pressors, and vasodilators for the patient with massive injuries (see Chapter 58).

Various pulmonary problems such as atelectasis, respiratory insufficiency, pulmonary contusion, pneumonia, and acute lung injury syndrome represent the primary postoperative complications in this group of patients. The presence

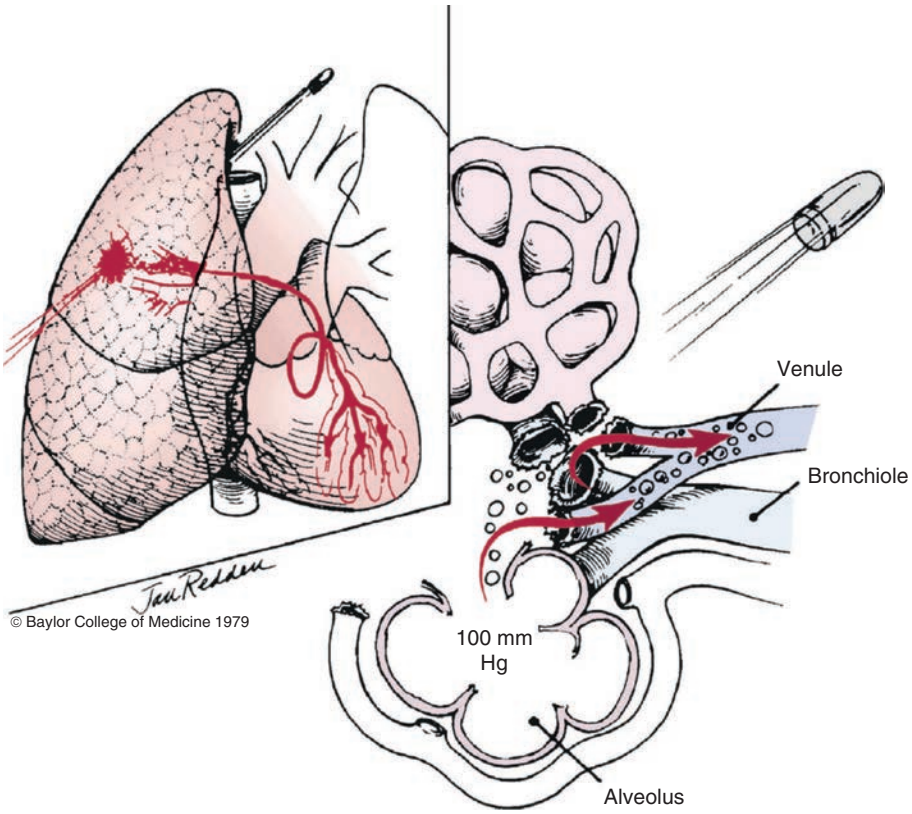


FIGURE 30-20 Drawing depicting the mechanism of systemic air embolism following a penetrating lung injury. (Copyright © Baylor College of Medicine, 1979.)

of pulmonary contusions and the potential for development of adult respiratory distress syndrome mandate that fluid administration be carefully monitored. Special ventilatory strategies to address potential complications of these pulmonary injuries and syndromes can be used (see Chapter 59). Patient mobility is important, and adequate medication for pain relief may decrease atelectasis and pneumonia. For the management of pain related to a thoracotomy or multiple rib fractures, postoperative thoracic epidural anesthesia can be considered in stable patients without spinal injuries. With the advent of long-acting local anesthetic agents, intercostal nerve blocks can be performed shortly after admission or at the completion of a thoracotomy. Multimodal nonopioid pain management strategies are increasingly being employed as well.

Postoperative hemorrhage is often due to a technical problem but may be the result of a coagulopathy related to hypothermia, acidosis, and massive blood transfusion. Coagulation studies, especially thromboelastography or rotational thromboelastometry, can be carefully monitored and corrected with administration of appropriate blood products[99] (see Chapter 16). Blood draining via a thoracostomy tube can be collected and autotransfused. During the initial resuscitation of these critically injured patients, various intravascular lines are often rapidly placed at the expense of strict sterile technique. All such lines should be replaced after the patient has been stabilized in the ICU. Prophylactic antibiotic therapy should be given perioperatively. Patients are counseled regarding the necessity of antibiotic prophylaxis during invasive future procedures, including dental manipulations.

Most late complications are related to infections or sequelae from other injuries. Long-term complications specifically related to the vascular repair include stenosis, thrombosis, arteriovenous fistula, graft infection, and formation of a pseudoaneurysm.

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31

Trauma Laparotomy: Principles and Techniques

Jessica A. Bowman • Gregory J. Jurkovich

KEY POINTS

- Wide prepping and draping allow the surgeon access to the chest and the groin to complete all necessary procedures.
- The trauma laparotomy should be performed in a routine, systematic manner, minimizing the likelihood of missed injuries.
- Damage control laparotomy focuses on immediate control of hemorrhage and contamination, followed by goal-directed resuscitation, reversal of coagulopathy, rewarming, and delayed definitive repair.
- Preperitoneal packing is proving to be a quick, efficient method to control zone III hematomas.
- A quality fascial closure helps prevent future morbidity associated with dehiscence, evisceration, and hernias.
- Surgeons should use laparotomy pads with a radiofrequency detection system or obtain abdominal and pelvic x-rays prior to definitive closure to decrease the risk of retaining foreign bodies.

INTRODUCTION

Performing a complete, efficient emergency exploration of the abdominal cavity is a defining and essential skill of the trauma surgeon. Following penetrating or blunt injury, a laparotomy is indicated for hemodynamic instability, peritonitis, evisceration, positive or questionable radiographic findings of organ injury, a positive diagnostic peritoneal tap (or lavage), and in some cases, a persistent drop in hematocrit. The objectives of a trauma laparotomy include control of hemorrhage, control of contamination from the gastrointestinal tract, and identification of all injuries. The patient's physiology determines whether definitive repair of injuries is accomplished at the initial operation or deferred in a damage control approach. It is the trauma surgeon's responsibility to devise a plan to address all injuries in a comprehensive and time-sensitive manner.

The word *laparotomy* comes from Greek origin, with *lapara* signifying the flank or waist and the suffix "-tomy" from the Greek word *tomoz* meaning to cut. In modern trauma surgery, the word *laparotomy* is used interchangeably with *celiotomy*, which stems from the Greek word *koilia,* meaning belly or bowels. Both words imply opening the peritoneal cavity, although perhaps a laparotomy would more accurately relate to a flank incision.[1,2]

This chapter provides an overview of the trauma laparotomy, including principles of the trauma laparotomy, preparation and team effort, a description of the technical steps and key maneuvers, considerations for the damage control approach, the practical aspects of temporary abdominal closure, and complications.

PRINCIPLES

The Core Mission

In a trauma laparotomy, the core mission is to identify and treat the greatest threat to the patient's life as quickly as possible. Most commonly, this threat is exsanguination, and the primary mission is hemorrhage control. The success of the operation depends on the team's ability to identify, expose, and control hemorrhage, while simultaneously resuscitating the patient with appropriate blood products, fluids, and electrolytes to maintain intravascular volume, correct coagulopathy, and counterbalance physiologic insult. Following hemorrhage control, the mission becomes identifying and addressing contamination from bowel injuries. Lastly, but no less important, is identification and treatment of all other injuries, including injuries to the abdominal wall, the mesentery, the genitourinary tract, and the solid organs of the kidney, pancreas, spleen, and liver.

Indications and Preparation

The patient's mechanism of injury, vital signs, physical exam, and imaging studies all play a role in the decision regarding

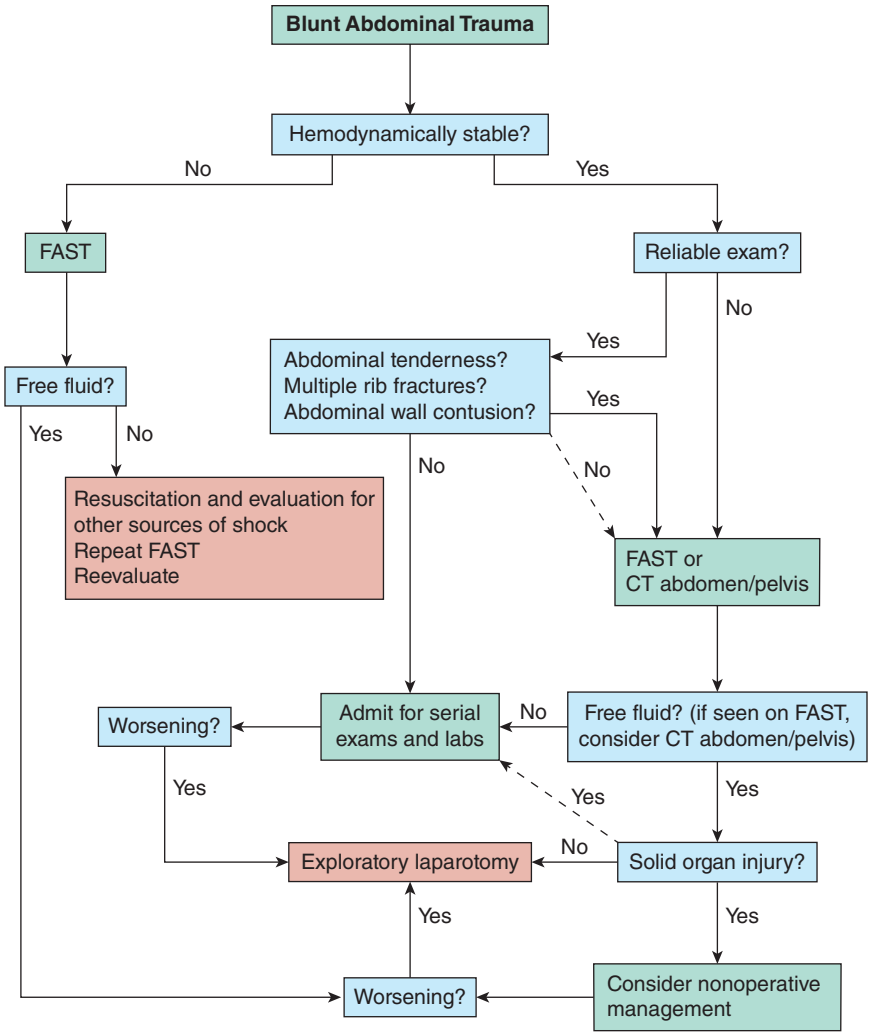


FIGURE 31-1 Indications for trauma laparotomy in a patient with blunt abdominal trauma. The first step is to determine if a patient is hemodynamically normal and stable. If not, suspicion for intra-abdominal injury should be high. These patients should undergo focused assessment with sonography in trauma (FAST) exam and, if positive, laparotomy. A negative FAST in this population is a worrisome and challenging situation. It is difficult to decide when to proceed to the operating room versus continuing resuscitation while evaluating for other sources of blood loss and shock. The surgeon must maintain a high level of suspicion, knowing that a false negative on initial FAST exam is possible.

Among hemodynamically normal and stable patients, assess the reliability of the abdominal exam. If reliable and there are no other high-risk signs of intra-abdominal injury, the patient may be monitored with serial exams and labs. Alternatively (dashed arrow), they may undergo computed tomography (CT) imaging and, if negative, there is assurance there is no intraabdominal injury. Patients without a reliable exam should undergo further imaging. For patients with free fluid and no solid organ injury, we advocate exploratory laparotomy or laparoscopy to evaluate for bowel, bladder, or mesenteric injury, particularly for those who also had high-risk signs of intra-abdominal injury (eg, lap belt sign). If there are no high-risk signs (dashed arrow), patients may be monitored with serial exams and labs, knowing there is a possibility of missed or delayed bowel injury.

whether a patient requires an urgent or emergent laparotomy. Algorithms are a convenient way to organize one's thinking about a clinical scenario, and Figs. 31-1 and 31-2 are presented as standard approaches to determine if a patient requires a trauma laparotomy. The astute clinician needs to be aware of those patients who are not "following the rules," but it is impossible to define an algorithm for every unique patient and situation. Each of the decision branches in these algorithms demands surgical judgment and a careful understanding of the pitfalls and traps of going down the wrong pathway. Continual reassessment of the patient's physiology and the results from any imaging or response to resuscitation are necessary to avoid making a wrong decision.[3,4]

The two management algorithms presented in Figs. 31-1 and 31-2 are simplified guidelines for the many complex decisions that must be made at each of the branching nodes. Assessment of blunt trauma to the abdomen has been greatly aided by the rapid and widespread availability of computed tomography (CT) and the bedside ultrasonography. The benefits and disadvantages of both techniques should always be considered carefully. The penetrating abdominal trauma algorithm includes both gunshot and stab mechanisms and

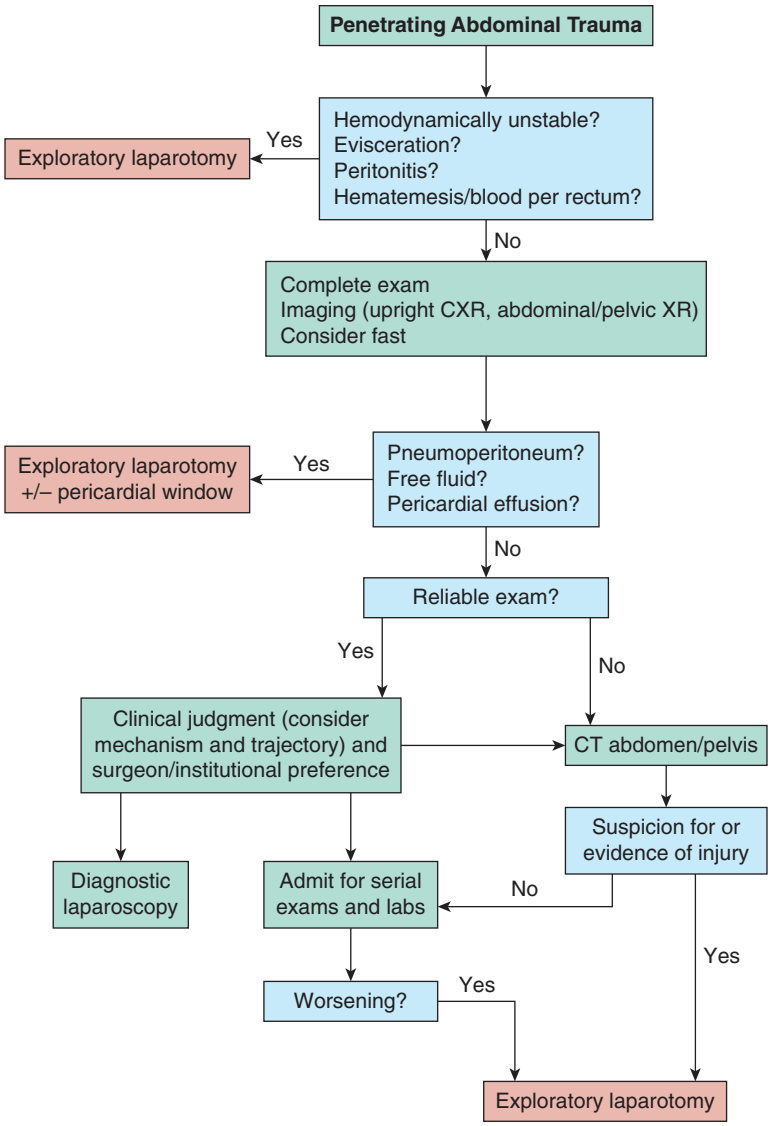


FIGURE 31-2 Indications for trauma laparotomy in a patient with penetrating abdominal trauma. Patients who are hemodynamically unstable and have evidence of peritoneal penetrating injury on physical exam should proceed to laparotomy, as should patients with evidence of intra-abdominal (or cardiac) injury on imaging. Imaging is helpful to evaluate thoracic pathology or bullet trajectory based on residual fragments. The pericardial component of the focused assessment with sonography in trauma (FAST) may identify excessive pericardial fluid and should be carefully performed for patients with penetrating injury to the cardiac box, bounded by the clavicle superiorly and the xiphoid inferiorly, and extending to the midclavicular line laterally. For patients with a reliable exam and no clear evidence of intra-abdominal injury, the surgeon should consider mechanism of injury, trajectory, and institutional/personal preference to determine whether serial exams and labs, computed tomography (CT) imaging, or diagnostic laparoscopy is most appropriate. For example, a patient with a skiving bullet trajectory may be appropriate for observation. This figure is similar to the more detailed Western Trauma Association algorithms for gunshot and stab wounds presented at the 48th Annual Meeting in 2018 (westerntrauma.org/algorithms/AbdominalStabWounds/WTAAbdStabWounds.pdf and westerntrauma.org/algorithms/AbdominalGSW/AbdominalGSWAlgorithmFinal.pdf). CXR, chest x-ray; XR, x-ray.

is perhaps even more fraught with institutional and individual preferences for management. Again, each of the decision branches requires considerable judgment and knowledge of contemporary literature.

For hemodynamically unstable trauma patients, activities in the trauma bay should be limited to interventions that are immediately lifesaving (see also Chapter 13). The surgeon should consider airway control, chest decompression for significant hemopneumothorax, obtaining intravenous access and initiating blood transfusion, placing direct pressure on sites of external hemorrhage, pelvic sheeting (if mechanism dictates), and obtaining a blood specimen for type and cross. In patients who require emergent laparotomy, it is the time from presentation to hemorrhage control that impacts mortality, and obtaining or examining studies should not delay time to incision.

In the operating room (OR), priorities before incision include positioning and prepping the patient, ensuring large-bore intravenous access and Foley catheter placement, obtaining blood products, temperature control of the room and the patient, and the administration of perioperative antibiotics. This requires an organized effort between the surgeon, anesthesiologist, and the nursing team. Communication with the entire OR team regarding objectives, operative plan, anticipated pitfalls, and necessary equipment is essential. Dedicated trauma ORs with ready access to all necessary surgical (eg, staplers) and resuscitation (eg, rapid transfusion devices) equipment should be a standard part of advanced preparation. Ideally, emergency trauma laparotomy trays containing instruments for both abdominal and major vascular procedures are opened prior to the patient arriving in the OR. Chest instruments should also be easily accessible.

Trauma patients should initially be positioned in the supine position with arms fully abducted at a 90° angle (Fig. 31-3). If the patient is unstable, prepping and draping may occur before or simultaneously with intubation. Meanwhile, other members of the team can work on venous and arterial access and the other issues noted earlier. This allows for immediate commencement of the operation should the patient decompensate further with induction of anesthesia.

Antimicrobial skin preparation for trauma surgery remains problematic. Historically, povidone-iodine was the agent of choice, but more recent literature shows povidone-iodine is associated with increased risk of surgical site infections compared to chlorhexidine-based products.[5] On the other hand, chlorhexidine-based products commonly contain alcohol, which increases the risk of an OR fire. In an ex vivo model, Jones et al[6] demonstrated that after alcohol-based skin preparation, immediate use of electrocautery resulted in flash fires 22% of the time. Even after waiting the manufacturer's suggested 3 minutes, electrocautery use still caused fires 10% of the time.[6] In addition to fire risk, critically injured patients do not have 3 minutes to spare.

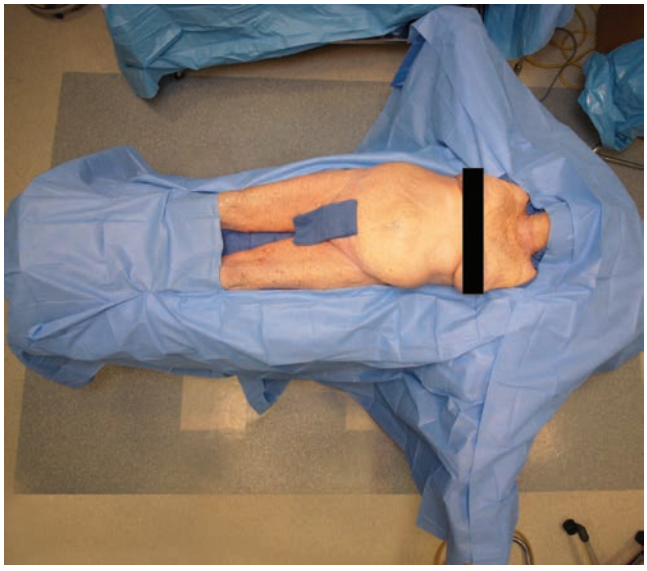


FIGURE 31-3 Prepping from chin to knees: the sterile operative field for trauma provides access to the neck, chest, abdomen, and groins. It allows the surgeon to plan for the unexpected during the procedure, providing access into an adjacent cavity and access to the groins for vein harvest or vascular control.

Typical draping for trauma laparotomy should be performed with exposure from chin to knees, extending to the posterior axillary line laterally. The groin is isolated with a sterile towel, leaving room to access the femoral vessels if needed. Anesthesia is allowed access to the head and arms while the surgeon maintains access to the neck, chest, abdomen, bilateral groins, and saphenous veins. A wide sterile field allows the surgeon to access multiple body cavities and be prepared for any eventuality, such as the gunshot wound to the abdomen that tracks superiorly into the chest or inferiorly to the groins.

In the exsanguinating patient, sterility remains desirable but is not essential. In instances where the patient has been rolled to the OR with a finger holding pressure on a bleeding wound, the assistant's hand may be prepped into the field until surgical control is achieved. In the stable patient, prophylactic antibiotics should be given prior to incision, most commonly in the trauma bay on the way to the OR. Even in unstable patients, antibiotics should be administered as soon as feasible.

Role of the Surgeon

During a trauma laparotomy, the surgeon plays several roles that must be seamlessly integrated. The surgeon must perform the operation in a technically proficient manner while supervising the other members of the team. The surgeon must focus on the specific details of the case while staying attuned to the patient's physiology. Based on the assimilation of these details, the surgeon must decide whether to perform a standard trauma laparotomy with definitive repair or use the damage control approach. Specific considerations will be discussed later in the chapter. The surgeon must anticipate subsequent steps of the operation and communicate effectively with the anesthesiologist and scrub nurse, keeping in mind the end point of the operation (eg, "Next we are going to perform splenectomy, staple off the bowel injuries, and be in the SICU [surgical intensive care unit] in under an hour with a temporary abdominal closure in place. Please let them know we will be coming and have a rapid transfusion device ready.").

The trauma laparotomy is a dynamic process that cannot be performed by the surgeon alone. Continued dialogue among the entire team regarding the patient's condition is of paramount importance. The anesthesiologist should verbally communicate on the patient's hemodynamic and volume status, any dysrhythmias, need for vasopressors, and number of blood transfusions, as well as on the presence of acidosis, coagulopathy, and hypothermia. These variables, reflecting physiologic reserve, guide critical surgical decision making. In patients with thoracoabdominal injuries, anesthesia should ensure that the chest tubes are on suction and that output is

monitored and communicated to the surgeon. In turn, the surgeon must communicate with anesthesia as to the status of the bleeding, the potential release of tamponade, and clamping and unclamping of major vessels, which may alter hemodynamics and arterial pH. Communication with the nursing staff is also vital to ensure availability of needed equipment.

An additional responsibility of the operating surgeon is to acknowledge the need for help. Even the most seasoned surgeons will encounter cases that require an additional experienced trauma surgeon or more specialized assistance. The decision to stop and call for additional help reflects sound judgment and humility and emphasizes patient safety.

TRAUMA LAPAROTOMY

The patient's hemodynamic status dictates the course and urgency of the laparotomy. The standard trauma laparotomy should follow a goal-directed, reproducible sequence of steps that provides a thorough and efficient evaluation of the entire abdomen. These steps include entering the peritoneum, prompt control of bleeding and contamination, complete exploration of the abdominal cavity, and repair of identified injuries. In cases of severe physiologic insult and/or hemodynamic instability, the surgeon may elect an abbreviated version of the standard laparotomy: the damage control approach.

Gaining Access to the Peritoneum

The trauma laparotomy is most commonly performed through a midline incision. In general, the "sicker" the patient (eg, class III/IV shock, multiple torso wounds), the larger the incision should be in order to gain rapid control of the hemorrhage and intestinal spillage. Thus, a classic xiphoid-to-pubis incision is indicated. This incision affords wide exposure of intraperitoneal and retroperitoneal organs and may easily be extended into a sternotomy as needed. In contrast, in patients who are hemodynamically stable from penetrating trauma or those who have undergone preoperative CT scanning, a more directed or limited incision can be used to start, and the surgeon can rapidly extend the incision superiorly or inferiorly based on need and operative findings. Occasionally, subcostal incisions may be appropriate for known isolated injuries of the spleen or liver, and a "transplant" incision can be particularly advantageous for known major liver injuries that require laparotomy. Most general and trauma surgeons are comfortable with this incision and can use the midline to enter the abdomen quickly. Using the scalpel with a size 10 (standard) or 20 or 21 (larger) blade to cut through the skin to the fascia and then again through the linea alba superiorly is more efficient than using electrocautery, but it is also a more difficult skill.

Incising through skin and soft tissue can be done with a few strokes of the knife. In obese patients, a useful technique to get through the pannus in the midline is to apply a significant amount of lateral traction on the skin. The white decussating fibers of the anterior rectus sheath denote the midline. The linea alba from the xiphoid to the umbilicus should be divided sharply, revealing preperitoneal fat and then peritoneum. In patients with significant hemoperitoneum, opening the fascia widely prior to getting into the abdomen prevents the blood from obscuring the rest of the incision and aids in fully opening the incision. The peritoneum may be entered bluntly just cephalad to the umbilicus or entered sharply with a pair of Metzenbaum scissors while lifting the peritoneum to avoid injuring the bowel. This is best performed near the xiphoid with the lateral segments of the liver providing some protection beneath. Once the peritoneum is deemed free of adhesions, it may be freely opened with the scalpel, Mayo scissors, or electrocautery. Of note, in patients with a prior abdominal incision, it is safest to enter the peritoneum away from the preexisting scar in order to avoid underlying adhesions.

Initial Maneuvers

Once the peritoneum is opened, the surgeon should rapidly evacuate any pooled or clotted blood by scooping it out with both hands and laparotomy pads. Note that we do not advocate blind four-quadrant packing as the first step because it is inadequate to tamponade bleeding, may injure delicate structures (splenic ligaments, friable or injured mesentery), and masks ongoing bleeding. The next step is small bowel evisceration, which is accomplished by sweeping the hand around the small bowel from the left upper quadrant toward the pelvis, then up the right side, lifting the entire small bowel up onto the right upper abdominal wall, allowing the mesentery to remain in line and not twisted. Likely sources of bleeding include solid organs, bowel mesentery, and the great blood vessels. The solid organs are quickly inspected and palpated for injury. These maneuvers can be followed by directed packing under vision into the pelvis and other quadrants. These packs will also absorb residual blood and begin to tamponade ongoing bleeding. Liver hemorrhage can be treated with packing, while splenic hemorrhage is typically managed with immediate splenectomy. Mesenteric bleeding is managed by clamps and ligature as the first step, with later evaluation of bowel viability. Retroperitoneal and great vessel injuries are more complex (also see Chapter 38).

If directed packing controls hemorrhage, a pause in the operation can occur, allowing the team to regroup, restore the patient's blood volume, obtain equipment, and mentally prepare for the next portion of the operation. The surgeon's next immediate priorities include locating the injured vessel or organ, exposing the injury, and then deciding on temporary or definitive repair.

If bleeding is not controlled with packing, the surgeon should gain control of the supraceliac aorta with a trained finger or sponge stick. The supraceliac aorta is exposed by retracting the lateral segments (II and III) of the liver toward the patient's right and incising the gastrohepatic ligament. The esophagus can then be laterally displaced, exposing the aorta. The aorta should be dissected anteriorly and laterally to allow for passage of a vascular clamp. Division of the left

crus of the diaphragm will also help to expose the proximal aorta at the hiatus. While suture ligation of most bleeding intra-abdominal vessels is accepted, primary repair of the aorta, vena cava, and very proximal superior mesenteric artery (and rarely the portal vein) should be part of the hemorrhage control plan. Once initial hemorrhage has been controlled, the surgeon should address gastrointestinal spillage to limit contamination.

Hemorrhage Control Techniques

Hemorrhage from the liver is initially treated with manual compression followed by tight packing lateral, superior, and inferior to the liver (see Chapter 33 for further details). If packing controls hemorrhage, packs should be left in place. If bleeding is not controlled, a Pringle maneuver can be performed by placing a finger through the foramen of Winslow and pinching the portal triad (occluding the portal vein and the hepatic artery). A vascular clamp or Rommel occlusive loop can then replace the surgeon's hand. Failure to control bleeding with the Pringle maneuver indicates hepatic vein or retrohepatic vena cava injury.[7]

Bleeding from the spleen is managed by first mobilizing the spleen up into the midline for better exposure, obtaining vascular pedicle control, and then resection (see Chapter 34). For kidney injury, primary vascular control versus initial mobilization of the kidney from the Gerota fascia is more controversial[8] (see Chapter 40).

The decision whether to explore a retroperitoneal hematoma depends on the mechanism of injury, location of the hematoma, and evidence of ongoing bleeding. Zone I is the central zone, extending from the diaphragmatic hiatus to the bifurcation of the vena cava and aorta and bounded by the kidneys laterally. This zone can be further divided by a supramesocolic or inframesocolic location. It is inspected by lifting the transverse colon and gently retracting it either caudally or cranially. Zone I retroperitoneal hematomas require surgical exploration, as they signify great vessel injury, including aorta, vena cava, celiac axis vessel, superior mesenteric artery, superior mesenteric vein, or portal vein injury. Zone II is located laterally from the kidneys to the paracolic gutters, and hematoma in this area usually signifies injury to the kidney, renal artery, or vein. Penetrating injuries to zone II are generally explored, following the trajectory of the bullet or knife to exclude all injuries. Blunt injuries are only explored if the hematoma is expanding, although this decision can be difficult, knowing exploration increases the rate of nephrectomy. Immediate postoperative CT and possible angioembolization of a bleeding renal vessel are options, but not at the expense of hemorrhagic shock and death. Maneuvers to expose zone I and II structures are described in the following section. Zone III includes the pelvis, encompassing the iliac arteries and veins, and should generally only be explored in the case of penetrating injury.

In blunt trauma, management of zone III pelvic retroperitoneal bleeding, which is most commonly caused by venous bleeding, depends on institutional resources, including the availability of interventional radiology for angioembolization, the capability for urgent external fixation, and surgeon comfort with preperitoneal packing (PPP). Opening these hematomas is reserved for cases of exsanguination or critical limb ischemia where a named pelvic arterial injury is expected, as packing the pelvis from the abdominal cavity rarely controls hemorrhage. When confronted by a previously unsuspected expanding zone III hematoma during a laparotomy, the surgeon must decide whether to move to angiography or proceed with PPP. It cannot be overemphasized that this is not simply packing the intraperitoneal part of the pelvis from the celiotomy incision (see Chapter 39). Although routine pelvic x-rays are no longer usually obtained, they still remain indicated in patients with hypotension and/or signs of obvious pelvic instability. In these patients who may be at greater risk of pelvic hemorrhage (eg, known pelvic fracture), we advocate limiting the inferior portion of the initial trauma laparotomy incision sufficiently above the pubis to allow a separate incision for PPP if a major pelvic hematoma is identified.

PPP has come back into favor as a quick, efficient method to control pelvic hemorrhage, especially given that interventional radiology may not be immediately available and prolonged time to hemorrhage control is associated with increased mortality. Additionally, because most pelvic bleeding is due to venous injury, it cannot be controlled with angioembolization.[9] PPP is accomplished by making a separate lower midline incision (10-cm midline incision just above pubis) and dissecting down through the anterior fascia to the preperitoneum without entering or disrupting the anterior peritoneum.

From the lower midline PPP incision, the surgeon can retract the bladder to one side, palpate the pelvic brim, and proceed posteriorly to the sacroiliac joint. Three laparotomy pads are then placed on each side: one posteriorly at the sacroiliac joint, the next just anterior at the middle of the pelvic brim, and the last just posterior and lateral to the pubis. These steps are then repeated on the contralateral side.[10] Ideally, PPP takes place after external fixation, which decreases the volume of the pelvis and also facilitates sterility by removing the sheet/binder.[11] Laparotomy can follow once this cavity is closed, or alternatively, the two procedures can occur simultaneously if necessary. Postoperative angiography can be obtained if suspicion remains high for an arterial injury.

Exposing Retroperitoneal Structures

The medial visceral rotations are maneuvers used to expose key retroperitoneal structures. A right medial visceral rotation, also known as the Cattell-Braasch maneuver, is used to expose the intra-abdominal inferior vena cava, the right renal pedicle, and the right iliac artery and vein (Fig. 31-4). The ureter, head of the pancreas, duodenum, and posterior aspect of the right colon will also be exposed. The Cattell-Braasch begins with mobilization of the hepatic flexure of the right colon and a full Kocher maneuver to mobilize the duodenum and pancreatic head along the peritoneal reflection. This is

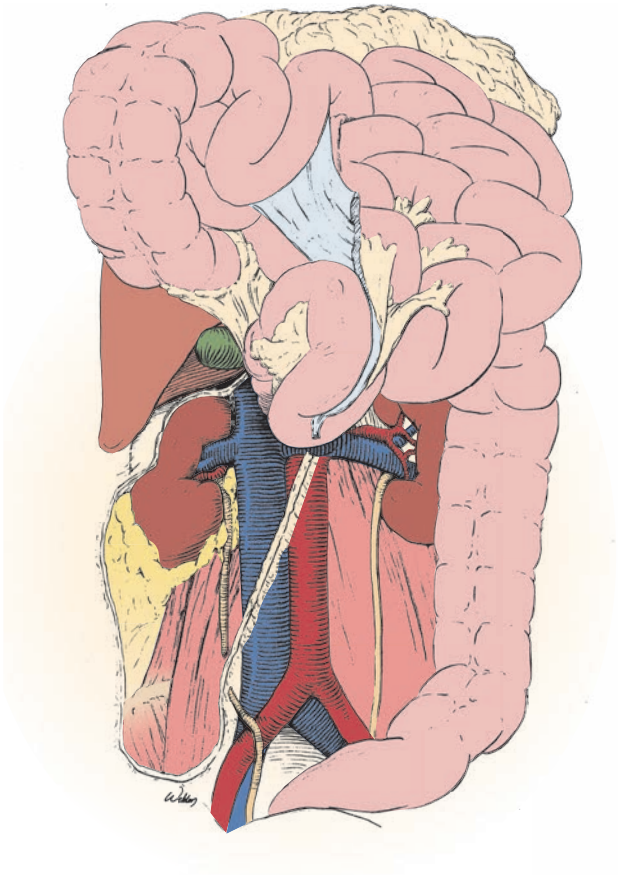


FIGURE 31-4 The full Cattell-Braasch maneuver provides broad exposure of the retroperitoneum. The only two areas of the retroperitoneum that remain inaccessible are the retrohepatic vena cava and the suprarenal aorta. The latter is accessible with the Mattox maneuver.

further carried down the right colon along the paracolic gutter by dividing the white line of Toldt. The exposure ends by dividing the avascular plane, which exists between the root of the mesentery and the peritoneum. The small bowel and right colon are then retracted medially, allowing visualization of the inferior vena cava.

To improve exposure of the retrohepatic vena cava, the surgeon can extend the midline incision across the costal margin into an intercostal space of the right chest. The diaphragm is incised to expose the vena cava and hepatic veins, allowing proximal control of the inferior vena cava from inside the chest.

A left medial visceral rotation, the Mattox maneuver, is used to explore a zone I or a zone II retroperitoneal hematoma on the left (Fig. 31-5). The left-sided organs are mobilized off the aorta, which allows for broad exposure of the aorta from the diaphragmatic hiatus to the iliac vessels, including the celiac axis, superior mesenteric artery and vein, left renal artery and vein, and left iliac artery and vein. Before opening the retroperitoneum, proximal control at the supraceliac aorta should be obtained. A left medial visceral rotation is initiated by dividing the splenorenal ligament. The white line of Toldt is then divided from the splenocolic flexure down the paracolic gutter to the distal sigmoid colon. The left colon, spleen, stomach, and pancreas are mobilized to the midline, just anterior to the Gerota fascia. In the classic Mattox maneuver, the kidney is included in the mobilization, which allows for access to the posterior aspect of the kidney and the aorta below the renal pedicle. Otherwise, the left renal vein restricts access to the anterior aorta. Care must be taken to avoid iatrogenic injury to the spleen when placing traction on the descending colon.

Exploring the Peritoneal Cavity

The peritoneal cavity is explored in a similar manner each time, so as not to overlook any injuries. As previously described, zones I, II, and III of the retroperitoneum are examined for hematoma early in the procedure. The anterior aspect of the stomach is examined in its entirety from the gastroesophageal junction to the pylorus. The lesser sac is then opened by dividing the gastrocolic ligament, and the posterior aspect of the stomach and the anterior aspect of the pancreas are inspected.

From the pylorus, the gastrointestinal tract can be examined from proximal to distal extent. If the duodenum has been mobilized with a Kocher maneuver, the anterior and posterior aspects of the duodenum can be visualized. The small bowel is run in a methodical fashion, examining the circumference of the bowel and identifying any abnormalities. As a segment of small bowel is lifted for examination, the corresponding mesentery is also inspected for hematoma, rents, or other abnormalities.

Serosal injuries or full-thickness perforations of the small bowel may be contained with a Babcock clamp or oversewn with a rapid whipstitch as they are encountered for temporary control of contamination. Definitive management should wait until a complete exploration is accomplished. Once the terminal ileum is encountered, the appendix and ascending, transverse, descending, and sigmoid colon are inspected. It may be necessary to mobilize the colon along the white line of Toldt to examine the retroperitoneal aspect, particularly in cases of penetrating trauma where the trajectory is suspicious. The liver, spleen, kidneys, and gallbladder are palpated for injury. In the pelvis, the genitourinary organs are inspected for injury. Finally, the diaphragm is inspected carefully as a site of potential missed injury. Structures appearing bruised, visceral hematomas, or those located close to a missile trajectory should be fully mobilized and explored carefully to identify all injuries.

Vascular Repair

Treatment options for intra-abdominal vascular injury include vessel ligation, primary repair, vein patch, interposition grafting, and temporary intravascular shunting (see Chapter 38). Shunting is described in further detail later. The decision to ligate or repair a vessel depends on the nature of the vessel and the risk of ischemia with ligation. Ligation of the celiac

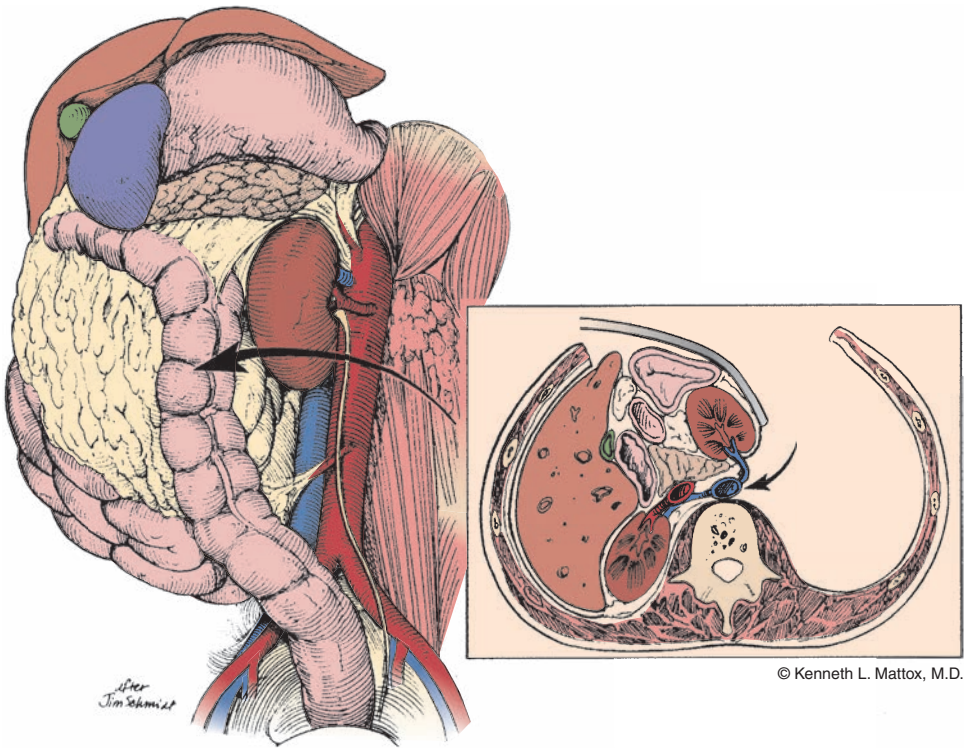


FIGURE 31-5 A full left-sided medial visceral rotation for trauma (Mattox maneuver) provides access to the abdominal aorta and is the only way to rapidly expose the suprarenal aortic segment in the presence of a central retroperitoneal hematoma. The inset shows the correct plane of dissection that is immediately on the muscles of the posterior abdominal wall. The presence of a large retroperitoneal hematoma greatly facilitates the maneuver. (Copyright © Kenneth L. Mattox, MD.)

artery and the internal iliac artery may be done with low risk of morbidity in most patients, as good collateral circulation exists. On the contrary, ligation of the common iliac artery and the external iliac artery carries a high risk of amputation. Primary repair of the vessel should be performed whenever time (and the nature of the wound) permits. Debridement to healthy tissue is performed prior to any repair.

Whenever possible, closure of a vascular injury should be performed in a transverse fashion with a running permanent monofilament suture in a lateral arteriorrhaphy or in an end-to-end anastomosis. Stenosis of the vessel is often accepted in favor of hemorrhage control, but when time permits, stenosis may be avoided with vein patch. In all cases, the tenets of vascular surgery, including proximal and distal control, adequate inflow and outflow, and a tension-free anastomosis, should be followed.

Injuries to the aorta and the suprarenal inferior vena cava mandate repair. Any vein below the renal vein may be ligated if repair is felt to be unsafe. Tying off the external iliac vein, common iliac vein, or even the infrarenal inferior cava is acceptable in critical situations, although lower extremity edema leading to a compartment syndrome may result. In these cases, four-compartment fasciotomy should be performed on the ipsilateral limb. Injuries to the iliac veins may be amenable to a primary repair after proximal control at the bifurcation and distal control in the femoral canal are obtained.

Repair of the portal vein with carefully placed monofilament suture should be attempted when possible, although portal triad vascular injuries remain one of the most lethal of intra-abdominal injuries, typically due to exsanguination.[12] Portal vein ligation should be used as a last resort, as ligation leads to massive intestinal edema. When employed, this strategy should be used in conjunction with an open abdomen and a planned second-look procedure.

The proximal superior mesenteric artery or vein must be repaired to preserve blood flow to the small bowel. Injuries to the superior mesenteric artery beyond the pancreas can be addressed with vessel ligation in conjunction with bypass grafting from the distal aorta, as would be performed for superior mesenteric artery thrombosis. Caution should be used when placing interposition grafts near a major pancreatic injury, as a pancreatic leak could lead to a complete disruption of the anastomosis.[13] Injury to the mesenteric vessels necessitating vascular repair and large mesenteric hematomas may compromise blood flow to the intestine. In these circumstances, a planned second-look laparotomy is prudent.

When segments of critical vessels are destroyed or resected, interposition grafts of polytetrafluoroethylene (PTFE; Gore-Tex, Newark, DE) or Dacron can be used to bridge the deficit in clean or minimally contaminated cases. Tissue coverage with omentum or muscle may be used to protect the graft. Autogenous great saphenous vein can also be harvested as

conduit, particularly in instances of bowel perforation and contamination.

Damage Control Considerations

Over the past few decades, principles of the damage control approach have been widely adopted and applied to both the military and civilian approach to the critically injured trauma patient (see also Chapter 42). The damage control approach refers to an abbreviated laparotomy in an unstable trauma patient with goals of quickly controlling hemorrhage and gastrointestinal spillage with a planned return to the OR for definitive repair of injuries following resuscitation. There are signals in the literature that the techniques of damage control and delayed definitive repair are perhaps overused, recognizing that considerable judgment is needed in this circumstance.[14,15]

The concept of damage control was first introduced to the trauma community in 1908, when Dr. J. Hogarth Pringle described the technique of using suture over gauze to control portal venous bleeding in the trauma patient.[16] In the early 1990s, Dr. Michael F. Rotondo and colleagues at the University of Pennsylvania described the damage control approach in three stages: the first stage of laparotomy with rapid hemostasis and control of contamination with a temporary closure of the abdomen, the second stage of resuscitation and restoration of normal physiology in the intensive care unit, and the third stage of reexploration, definitive repair, and closure. A retrospective review showed the damage control approach improved survival among patients with combined major vascular injury and two or more visceral injuries.[17]

The primary objective of the damage control approach is avoidance of an irrevocable physiologic insult, the "bloody viscous cycle" of trauma—acidosis, coagulopathy, and hypothermia—from which a patient cannot recover. The bloody viscous cycle, also known as the lethal triad, was first described in 1981 by Dr. Gene Moore and the Denver General (now Denver Health) group.[18] Severely injured patients displaying such physiology are at heightened risk of mortality.

The decision to proceed with a damage control approach should be made before the patient develops the lethal triad of physiologic abnormality. Rapid restoration of normal physiology takes precedence over definitive restoration of normal anatomy. For a patient in profound shock or with multisystem/multicavity trauma, the decision to use the damage control approach may be made before going to the OR. Parameters such as pH of <7.20, temperature of 93°F, and blood loss of 10 units or more predict the need for damage control approach.[19] Wound patterns such as combined major vascular injury and gastrointestinal injury may warrant damage control. Intraoperative signs of the bloody viscous cycle, including diffuse oozing from all surfaces, edematous bowel, or dusky-appearing viscera, should guide the surgeon toward the damage control approach.

In the first stage of a damage control approach, the surgeon should (1) pack injuries to solid organs such as the liver and kidney, (2) perform splenectomy as needed, (3) ligate or shunt major vascular injuries, (4) oversew or staple off injuries to the intestines, (5) drain suspected biliary or pancreatic injuries, and (6) place a temporary abdominal closure.

Shunting is a method to temporarily bridge injured vessels with a prosthetic conduit, rapidly controlling hemorrhage and restoring blood flow, while deferring definitive repair. In patients with injury to the common or external iliac arteries, shunting has been shown to reduce the rate of amputations and fasciotomies.[20] Before shunting, thrombectomy with proximal and distal embolectomy should be performed with Fogarty catheters to ensure adequate inflow and outflow. Commercial shunts exist, but surgeons can also employ pediatric feeding tubes, nasogastric tubing, or thoracostomy tubes, depending on the vessel size. The choice of shunt depends mainly on the size of vessel injured, as size match between shunt and vessel is a primary factor in shunt success. Patency and flow through the shunt can be demonstrated with on-table angiography. It is not necessary to systemically anticoagulate patients with temporary intravascular shunts. Complications of shunting include shunt thrombosis, distal embolization, shunt dislodgement, and infection.

If there is concern for ongoing bleeding from solid organs, the surgeon should consider angiography and embolization. Hybrid ORs, which have all the features of a traditional OR plus additional capabilities for angiography, endovascular interventions, and fluoroscopy, are becoming more popular in large centers and allow multiple teams to intervene on a patient at once or to proceed sequentially with endovascular techniques without moving the patient.

The patient should be promptly transported to the surgical intensive care unit for the second stage of resuscitation. Hemodynamics, urine output, and serial lab parameters such as lactate and base deficit are used as quantifiable markers of shock to guide fluid resuscitation. Rewarming with forced-air warmers, radiant heat, and heated fluids is also performed. Coagulation labs and/or thromboelastography should guide transfusions with the goal of correcting underlying coagulopathy. In preparation for the third stage, a thorough head-to-toe tertiary exam should be performed to identify any injuries that may have been overlooked during the primary survey.

Once the patient is fully resuscitated, reexploration with definitive repair can occur. The exact timing is based largely on the patient's response to resuscitation, but a shorter time (<2 days) to the first attempt at definitive closure is associated with greater odds of successful fascial closure.[21] After removing the temporary abdominal closure, copious irrigation is used to soak and rehydrate the packs so they can be removed without disturbing underlying clot. The abdomen is completely explored for other injuries, and then vascular and gastrointestinal injuries can be definitively repaired. If at any point the patient becomes hemodynamically unstable, the damage control sequence can be reinitiated.

When possible, a tension-free fascial closure is performed. In cases of ongoing contamination, bleeding requiring repacking, or concern for bowel viability, a temporary closure may be replaced for subsequent reexploration. Efforts at progressive fascial closure on a daily basis are warranted, since the overall success rate of definitive closure falls dramatically after

TABLE 31-1: Indications for Temporary Abdominal Closure in the Trauma Patient

Damage control approach
Acidosis
Hypothermia
Coagulopathy
Large-volume blood loss
Combination of major vascular and gastrointestinal injury
Planned second-look operation
Intra-abdominal packing in place
Unable to achieve tension-free fascial closure
Risk of abdominal compartment syndrome
Treatment of abdominal compartment syndrome

1 week. When the abdomen cannot be fully closed due to loss of domain or unrelenting bowel edema, a sequential abdominal closure with retention sutures may be employed. When all attempts to close the fascia have failed, complex closure methods with component separation or mesh may be used.

Temporary Abdominal Closure

Temporary abdominal closure allows the surgeon a quick and easy reentry into the abdomen without damaging the fascia, and it can be reapplied as needed before definitive closure. Indications for temporary abdominal closure are listed in Table 31-1. The temporary abdominal closure aims to contain the viscera, protect the bowel, provide early identification of intra-abdominal complications, and preserve healthy fascia for eventual closure. Note that abdominal compartment syndrome (ACS) has been reported with a temporary abdominal closure in place.

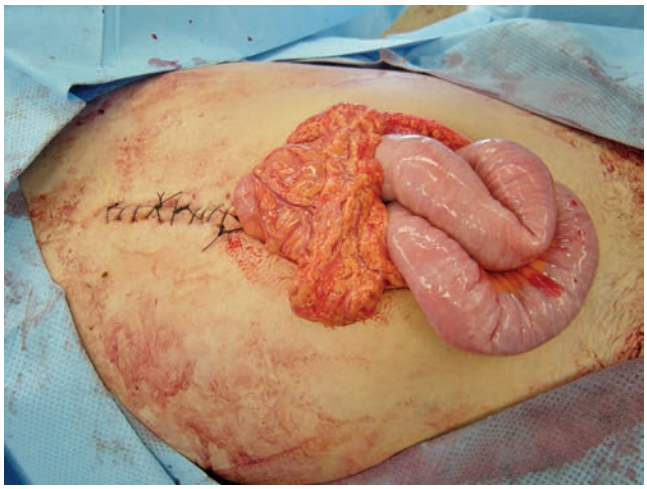
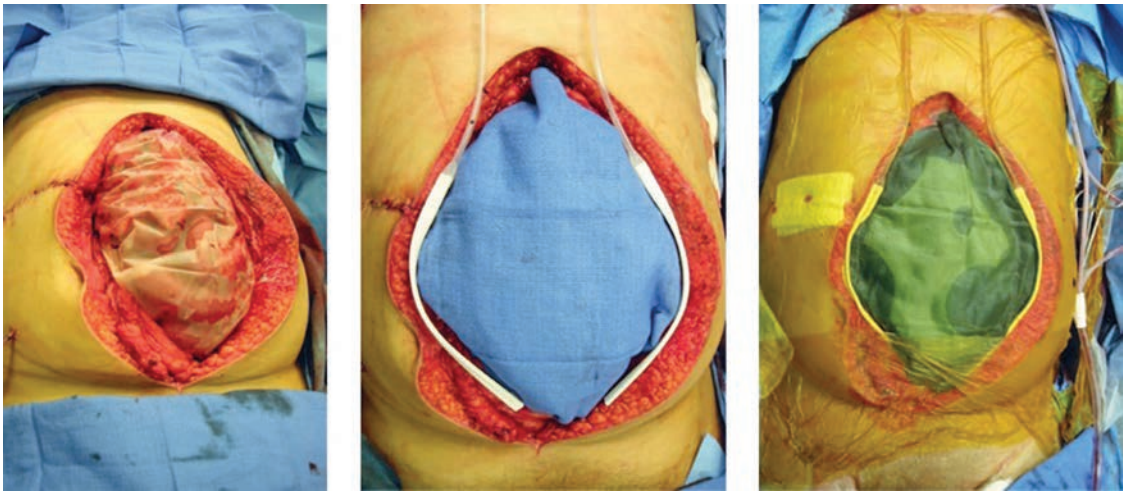


FIGURE 31-6 The abdomen has been partially closed with a skin-only closure. (Used by permission from Clay Cothren Burlew, MD.)

There are a variety of techniques used to perform temporary abdominal closure. A running suture in the skin is an easy method to bring the skin together, but drawbacks include injury to the skin and possible evisceration. Skin-only closures do not allow for fascial expansion and have a higher rate of secondary ACS and thus have fallen out of favor (Fig. 31-6). The two most widely accepted techniques for temporary abdominal closure employ either the ABThera Open Abdomen Negative Pressure Therapy (Acelity, San Antonio, TX) device or a homemade vacuum pack device.

Barker et al[22] described their success with the homemade vacuum pack. A similar method uses perforated subfascial 1010 Steri-Drapes (3M Health Care, St. Paul, MN), placed as laterally as possible to prevent adhesions, with two overlying Jackson-Pratt drains, a sterile towel to protect the bowel, and an Ioban drape to close the open abdomen (Fig. 31-7).



A **B** **C**

FIGURE 31-7 One method of temporary abdominal closure. (**A**) The bowel is covered with a sterile 1010 drape. (**B**) A sterile towel is placed to protect the bowel, and two Jackson-Pratt sump drains are placed to remove effluent from the peritoneum. These are placed to bulb or wall suction. (**C**) The closure is completed with a large Ioban drape. (Used by permission from Walter Biffl, MD.)

With any method, the suctioned effluent should be monitored for bloody or bilious drainage. The open abdomen may drain several liters over 1 day, and control of effluent with a vacuum device helps to keep the patient dry. Note that blood clot may collect below the closure, and a patient may have significant blood loss despite minimal effluent.

An open abdomen with a temporary closure does not mandate paralysis or sedation. Rather, patients may be extubated, enterally fed, and mobilized. In patients without bowel injury, enteral feeding has been associated with greater fascial closure rates, decreased complications, and decreased mortality.[23]

Once all packs and foreign bodies have been removed from the abdomen, vascular reconstruction is complete, and the gastrointestinal tract is in continuity, fascial closure can be attempted. Note that the need for enteral access should be addressed at this time.[24] Whenever possible, omentum should be placed between the bowel and the abdominal wall before closure. Several studies have shown that definitive fascial closure should be performed as early as possible to avoid morbidity and mortality. Early abdominal closure shortens hospital and intensive care unit length of stay and diminishes cost. Patients whose abdomens are closed within a week report higher quality of life and improved emotional health and are more likely to return to work than those closed after a week.[25] Factors associated with failure to achieve delayed primary closure include greater number of reexplorations, the development of intra-abdominal infections, bloodstream infections, acute renal failure, enteric fistula, and injury severity score greater than 15.[26]

When the fascia cannot be closed all at once, sequential fascial closure can be employed. Sequential closure may be performed with a sponge "sandwich" (Fig. 31-8). This technique consists of multiple white sponges (Acelity, San Antonio, TX) covering the bowel with the fascia held under tension with full-thickness number 1 polydioxanone (or other similar) sutures. A black sponge is placed over the white sponge with an occlusive dressing, and the vacuum is placed to suction. This addresses the need to keep the fascia under tension to prevent fascial retraction and loss of domain. The patient is returned to the OR at least every other day to perform sequential fascial closure with interrupted sutures. During this period, the surgeon must pay close attention to the patient's fluid status, as forceful diuresis is often required to get the laparotomy incision closed, and wound care, as clean fascial edges and progressive tightening are mandatory.

Despite all efforts, the fascia cannot always be closed. These scenarios present a technical challenge to the surgeon, and the decision and options are complex and not uniformly advocated. When a primary fascial closure is not possible, options include a component separation, direct skin grafting onto slightly granulated bowel, or closure with a variety of meshes (biologic, permanent, absorbable). A component separation, releasing the rectus sheath laterally, may provide up to 10 additional centimeters to bring the fascia together primarily. Biologic meshes, such as a porcine intestinal submucosa (Surgisis; Cook, Bloomington, IN) or a human acellular dermis such as Alloderm (Lifecell, Branchburg, NJ), are often recommended for contaminated wounds as they have no risk of infection, but they are very expensive and have a significant rate of hernia recurrence.

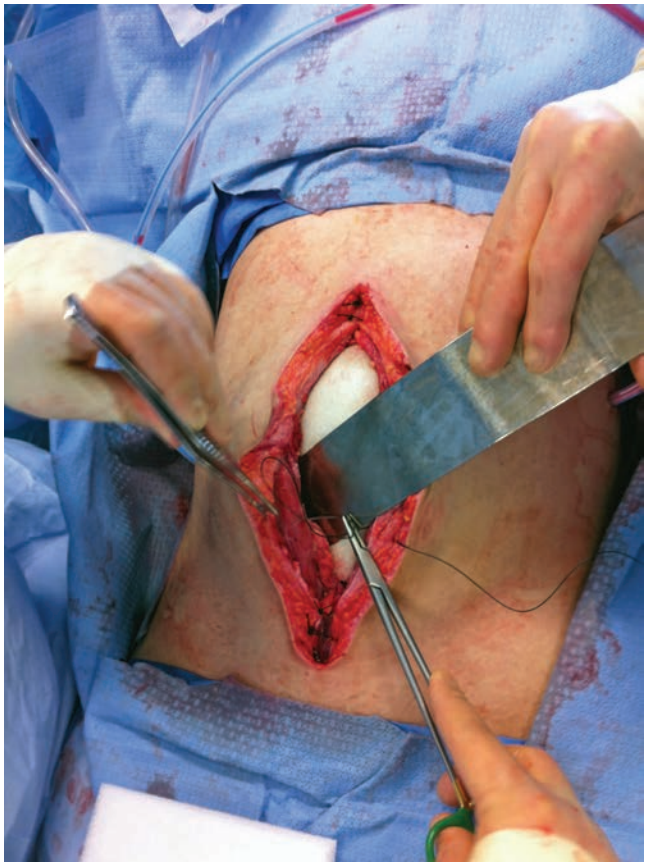


FIGURE 31-8 A sequential closure is performed on the second takeback operation. The lower fascia has been closed in a tension-free manner. A white vacuum-assisted closure sponge is placed over the bowel, and fascial retention sutures are placed. A black sponge will be placed over top and an Ioban drape will seal the closure. (Used by permission from Clay Cothren Burlew, MD.)

In cases of contamination, an absorbable mesh, such as a Vicryl mesh (Ethicon, Somerville, NJ), may be employed in a bridging fashion with an overlying skin graft. Vicryl has a high tensile strength and a very low rate of infection. Negative-pressure dressings can be placed on top of exposed mesh to promote growth of granulation tissue, allowing earlier skin grafting, although there are reports of this being associated with bowel fistula formation. Until grafted, these wounds are similar to full-thickness burns and represent a major catabolic drain for the patient, and the unprotected viscera are susceptible to injury and fistula formation. These patients can eventually undergo planned ventral hernia repair.[27]

A permanent prosthetic, such as a Marlex mesh (Davol, Cranston, RI) or a Prolene mesh (Ethicon, Somerville, NJ), may be employed to bridge a fascial defect, although they are much more commonly used for definitive repair of a long-standing hernia and not the closure of an open

abdomen. They become well-incorporated into the body but carry a risk of infection, adhesion formation, seroma, and fistula formation. Permanent prosthetics should not be used in a contaminated abdomen, as they will stay infected and eventually require removal. Composite meshes use a combination of materials in order to take advantage of their various properties. They have improved tissue incorporation and create less inflammatory reaction, but they are more expensive.

In complex cases where there is a lack of intact skin to cover the defect, the surgeon should consider autologous tissue transfer in the form of either pedicled or free flaps.

COMPLICATIONS OF TRAUMA LAPAROTOMY

This section will focus on four potentially devastating complications after trauma laparotomy: fascial dehiscence and evisceration, enterocutaneous fistula, missed injury, and retained foreign body.

Fascial Dehiscence and Evisceration

Dehiscence occurs when previously closed fascia pulls apart. Evisceration occurs when intra-abdominal contents leave the peritoneal cavity through an opening in the fascia. Among trauma patients, predictors of dehiscence include technical error, wound infection, and intra-abdominal infection.[28] Additional risk factors for dehiscence include obesity, pulmonary disease, hemodynamic instability, sepsis, poor nutritional status, malignancy, ascites, and steroid use. Mortality among patients with dehiscence reaches nearly 30%.[29] The best treatment of dehiscence is prevention—namely, a quality fascial closure. The evidence for running versus interrupted sutures remains equivocal. In a retrospective review of trauma patients, there was no difference in the rate of dehiscence, incisional hernia, or surgical site infection based on closure with interrupted or running suture.[28,30] A recent prospective study demonstrated decreased dehiscence and mortality in an emergency laparotomy cohort with the use of slowly absorbable monofilament suture, the "small step" technique, and a suture-to-wound ratio of 4:1. This means the length of the suture used is at least four times the length of the incision. Exact placement of sutures is 5 to 6 mm from the edge of the fascia, with 4 to 5 mm between sutures.[31]

Unfortunately, dehiscence may complicate any exploratory laparotomy. Dehiscence with evisceration mandates urgent return to the OR. Principles of operative management include debridement to healthy fascia, control of any intra-abdominal sepsis, and a tension-free fascial closure. More complex methods of abdominal closure, as described earlier, may be employed when tension-free closure cannot be obtained. Prophylactic abdominal retention sutures may be useful in decreasing evisceration rates, but ultimately, their benefit remains debatable within the literature.

Enterocutaneous and Enteroatmospheric Fistulae

A fistula is an abnormal connection between two epithelialized organs. Factors contributing to the formation of enterocutaneous fistulas (ECFs) include damage to the serosal lining, erosion of the bowel against a prosthetic mesh, missed traumatic or iatrogenic injury to the bowel, and anastomotic breakdown. In the patient with an open abdomen, bowel ischemia and inflammation, bowel obstruction, increased manipulation of the bowel, desiccation of the bowel, and the presence of intra-abdominal infection can also contribute to fistula formation, known as enteroatmospheric fistula (EAF) in this setting. Prevention of a fistula can be attempted by placing omentum over the bowel, minimizing injury to the serosal layer of the bowel, keeping the bowel moist when the abdomen is open, and obtaining fascial closure as early as possible.

Unlike an ECF, an EAF has no vascularized tissue over the fistula tract. Although the majority of ECFs heal spontaneously, 50% to 80% of EAFs require surgical repair.[32] ECFs and EAFs require patience, during which time nutrition and wound care are of the utmost importance. When the patient has an open abdomen, early intervention has been recommended. If the surgeon misses the critical window before the abdomen becomes "frozen" with dense adhesions, operative intervention is delayed several months, which can lead to persistent electrolyte imbalance, malnutrition, prolonged intensive care unit and hospital stays, and increased mortality.

Missed Injury and Retained Foreign Bodies

Missed injuries and retained foreign bodies contribute to increased morbidity and mortality. While injuries may be missed during the primary or secondary survey, the majority of missed injuries are caused by incomplete surgical exploration.

Injuries to the colon, diaphragm, and genitourinary tract are the most commonly missed intra-abdominal injuries. Errors in technique and judgment can contribute to an imperfect exploration, as may hemodynamic instability and distracting injuries. Typically, missed injuries occur in proximity to another identified injury, potentially even in the same organ. Surgeons must be vigilant with regard to complete exposure of an injured area, including visceral rotations to explore retroperitoneal structures as needed. Additionally, surgeons should be aware of injury trajectory and number of bullet holes through the digestive tract.[33]

After trauma laparotomy, there are various factors that may contribute to a retained foreign body. Although quite rare, estimated at 0.14% by Teixeira et al[34] in a retrospective review of their 7-year experience at University of California, Los Angeles, retained foreign body can have devastating effects. The emergent nature of the procedure precludes the routine counting of instruments. During the initial entry and packing of the abdomen, it is easy and common to lose track

of the number of laparotomy pads placed in the abdomen when the focus is on lifesaving hemorrhage control. The presence of multiple teams operating in multiple body cavities may also contribute to the potential for instruments to be left behind. In one retrospective analysis, emergency surgery, unplanned change in procedure, and body mass index of the patient were found to be significantly associated with risk of foreign body retention.[35]

When a patient's abdomen is left open and packed, it is helpful to include the location, and ideally the number, of packs left within the abdomen in the operative note. This is especially important when a different team completes the subsequent case, as is common in trauma. Before preparing to close fascia, the surgeon should perform a thorough search of the peritoneum for remaining laparotomy pads and instruments. Prior to definitive closure of the abdomen, even when the instrument and sponge counts are correct, plain films should be performed and assessed by both the surgeon and a radiologist to look for retained foreign bodies.[34] Another option is the use of laparotomy pads embedded with a radiofrequency detection system, which has been shown to detect retained laparotomy pads even when x-rays were normal.[36]

Paramount to the management of any missed injury or retained foreign body is prompt identification, notification, and intervention. Once the missed injury has become evident to the team, full disclosure to the patient and family must occur. A hospital's risk management team may help to manage and mitigate this difficult situation.

NEGATIVE AND NONTHERAPEUTIC LAPAROTOMIES

A negative laparotomy is a laparotomy in which there is no evidence of injury. A nontherapeutic laparotomy is a laparotomy in which there is an injury that does not require further surgical intervention. A retrospective review of over 1800 trauma laparotomies at an urban, Level I trauma center demonstrated a negative laparotomy rate of 4%. Peritonitis, hypotension, and suspicious CT findings were the most common indications cited for laparotomy.[37] Another population-based study focusing on splenic and hepatic injuries identified a negative laparotomy rate of 5% and a nontherapeutic laparotomy rate of 9%.[38]

Despite the highly accurate radiographic imaging available, the decision to perform laparotomy for trauma is not always straightforward. On occasions where imaging or physical examination is equivocal or when the imaging does not correlate with the patient's clinical state, there is not always a clear decision algorithm. In these instances, the risk of a nontherapeutic laparotomy must be weighed against the risk of a delay in diagnosis.

There is controversy about the relative morbidity of a nontherapeutic laparotomy. Potential complications include, but are not limited to, cellulitis, wound infection, dehiscence, ileus, deep vein thrombosis, and myocardial infarction. Among patients with splenic and hepatic injuries who had a laparotomy, 6% developed subsequent small bowel obstruction (80% of those resolving with nonoperative management) and 9% developed an incisional hernia. In contrast, less than 1% of patients managed nonoperatively had a small bowel obstruction.[38]

The greater acceptance of minimally invasive techniques for trauma has reduced the negative laparotomy rate. Thoracoscopy or laparoscopy for penetrating trauma can be used in select hemodynamically stable patients with thoracoabdominal stab wounds to rule out diaphragm injury. Laparoscopy is a useful tool in the evaluation of left-sided thoracoabdominal gunshot wounds, gunshot wounds with suspected extraperitoneal trajectory, and for assessment of peritoneal violation after stab wounds to the flank and anterior abdomen.[39]

RELAPAROTOMY

Relaparotomy for Hemorrhage

The decision to return to the OR for postoperative bleeding, especially when using the damage control approach, is complex. The surgeon must weigh the risks of worsening coagulopathy and hypothermia associated with return to the OR against the likelihood of identifying surgically correctable bleeding. There is minimal benefit to returning a patient to the OR for coagulopathic, nonsurgical bleeding. The decision is often an interplay of factors including what was found and done during the original laparotomy, the patient's coagulopathic profile, physiologic status including core temperature, and transfusion requirements. In patients with a temporary abdominal closure, large volumes of sanguineous drainage may be used as a trigger to return to the OR.

Intraoperatively, hemostasis should focus on surgically correctable causes followed by appropriate packing to control coagulopathic bleeding. These patients almost always require temporary closure because they have a high rate of ACS after massive transfusion.

Relaparotomy for Intra-Abdominal Infection

In a patient who underwent a trauma laparotomy in which no injury to the gastrointestinal tract was identified and who then develops an intra-abdominal infection, the surgeon must consider a missed injury. As mentioned before, missed injuries require prompt patient notification and intervention. In a patient who had repair of an injury to the gastrointestinal tract and then develops an intra-abdominal infection, the surgeon must consider a leak. A septic clinical picture warrants further intervention—either return to the OR or urgent cross-sectional imaging to identify fluid collections that might be amenable to percutaneous drain placement. Infected biloma, hematoma, or abscess cavities that are multiloculated or not amenable to percutaneous drainage necessitate return to the OR.

If returning to the OR, the surgeon should be prepared for a challenging reoperation. After a few days, the abdominal cavity becomes entrapped and immobile secondary to

TABLE 31-2: Technical Options for Source Control During Relaparotomy for Uncontrolled Abdominal Infection

Technical option	Comment
Resection and anastomosis	Rarely feasible for early leaks
Resection and exteriorization	Almost never feasible due to foreshortened mesentery
Proximal diversion	Occasionally lifesaving (edematous abdominal wall is obstacle)
Tube drainage	Contraindicated
Vacuum-assisted management	Useful option for exposed fistula (caution if unprotected suture lines)

adhesions and contamination. The bowel is hard to free and is often highly edematous and easily injured. A variety of treatment options exist, but repairs are fraught with potential complications, and often the best strategy is wide drainage of the infected fluid collections and controlled drainage of a bowel fistula to air. Simple suture closure of a bowel leak or perforation often puts part of the bowel under tension. Secondary to the inflamed nature of the bowel surrounding the leak, this simple method has a high likelihood of failure. Resection and anastomosis is a better option when enough bowel can be mobilized, although any new anastomosis remains at high risk for failure in this situation. Tube drainage directly into a fistula site is controversial, and although it may help control the drainage, one can also expect continued leak around the tube, and the tube can serve to enlarge the enterotomy and maintain patency.

Proximal diversion is another treatment option. Injuries to the duodenum and proximal jejunum may be addressed with pyloric exclusion and a gastrojejunostomy. A loop ileostomy would permit more distal repairs in the ileum and large bowel to heal while diverting enteric contents. The technical options for reoperation for sepsis are outlined in Table 31-2.

BEDSIDE LAPAROTOMY

Resuscitative laparotomy may be performed at the bedside in the intensive care unit for patients requiring high levels of ventilatory support or escalating vasopressor requirements that preclude safe transport to the OR. One of the most common indications is decompressive laparotomy for ACS.

ACS is usually defined as intra-abdominal pressures greater than 20 mm Hg in the presence of elevated airway pressures or renal impairment. Primary ACS is often the result of injury, typically due to hemorrhage. Secondary ACS is caused by increased bowel edema and ascites from resuscitation in a closed abdomen; there is no primary intra-abdominal injury. Tertiary ACS, also called open abdomen ACS, occurs after treatment of primary or secondary ACS.

On physical examination, the abdomen will be distended and tight. The increased intra-abdominal pressure can directly compress of the inferior vena cava and, as a result, increase central venous pressure, increase pulmonary wedge pressure, decrease cardiac preload, and increase afterload. Lung expansion is difficult against a tense abdomen, elevating peak airway. Compression of the renal veins leads to an oliguria that is unresponsive to fluid resuscitation.

Diagnosis should be based on clinical findings in conjunction with a measurement of a bladder pressure. The trend of the bladder pressure may be more helpful than the absolute number. More importantly, the patient's physiologic profile and degree of organ dysfunction should hold more value in assessing ACS than the absolute bladder pressure measurement. It is commonly accepted that bladder pressures greater than 26 to 30 mm Hg should prompt decompression, but the absolute intra-abdominal pressure leading to a compartment syndrome is not definitively known and may vary by patient. Treatment for ACS is emergent decompression of the abdomen via midline laparotomy incision with placement of a temporary abdominal closure.

Cooperation and coordination between the surgical team, anesthesia, and nursing staff are essential for success of the bedside laparotomy. Essentially you are transporting the OR to the patient instead of vice versa. All potentially necessary personnel (eg, OR staff) and instruments and equipment (eg, sutures, staplers, electrocautery) need to be readily available. Most intensive care units have insufficient lighting, and one or more headlights are an absolute necessity. Despite these disadvantages and high associated mortality, laparotomy at the bedside can be lifesaving and the only option for a critically ill trauma patient.

Acknowledgments

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32

Diaphragm

Walter L. Biffl • William G. Cioffi

KEY POINTS

- Traumatic diaphragm injury is rare (<1%) following blunt trauma, whereas penetrating thoracoabdominal trauma has an incidence of 24% to 38%.
- Most traumatic diaphragm injuries are identified at surgical exploration.
- The overall accuracy of chest x-ray is quite poor; the only direct sign of diaphragmatic injury is the visualization of herniated abdominal viscera into the chest.
- Laparoscopy is recommended over computed tomography for the diagnosis of left-sided thoracoabdominal stab wounds.
- The two principles of repairing acute diaphragmatic hernias are complete reduction of the herniated organs back into the abdominal cavity and watertight closure of the defect.
- Repair of the acutely blunt-injured diaphragm is best performed via laparotomy, although laparoscopic or thoracoscopic repair is feasible.

INTRODUCTION

The diaphragm, the most important muscle of respiration, separates the thoracic and abdominal cavities. It can be injured in isolation or involved with injury in either body cavity. The most challenging aspect of management is the identification of injury. Penetrating injuries in particular may be initially asymptomatic but later develop herniation and strangulation of the stomach or other abdominal viscera.

HISTORY

Traumatic diaphragmatic rupture was first reported by Sennertus in 1541, and Ambroise Paré was the first to report a series of diaphragmatic perforations found at autopsy.[1] Paré also described the consequences of gastric and colonic incarceration in a ruptured diaphragm.[2] The first documented antemortem diagnosis was by Bowditch in 1853,[3] and it was not until 1886 that Riolfi was credited with the first successful repair.[4] The first acute repair was by Walker in 1899, in a patient who had been struck by a falling tree.[5] The largest early review of 378 diaphragmatic hernias was by Hedblom in 1925.[4]

ANATOMY

The diaphragm is a dome-shaped musculofibrous septum separating the abdomen and thorax. It is bounded above by both pleural spaces and the pericardium, which is attached to the central tendon. Structures immediately adjacent to the inferior side of the diaphragm include the liver, spleen, stomach, and to varying degrees the colon, omentum, and small bowel. The origin of the diaphragm includes the lower sternum, lower six costal cartilages and adjacent ribs, and medial and lateral lumbocostal arches. The crura, two tendinous pillars, arise from the lumbar vertebrae. The insertion of the diaphragm is into the central tendon, an aponeurosis, located at the top of the dome, oriented transversely and separated into three segments. At rest, the diaphragm rises to the level of the fourth intercostal space on the right and the fifth intercostal space on the left. At maximal contraction, the diaphragm descends two rib spaces bilaterally. The aorta passes behind the diaphragm and between the crura, where it has no attachments. Along with the aorta, the thoracic duct and azygous vein pass through this opening. The esophagus traverses the esophageal hiatus, which is mostly composed of the right crus along with the vagus nerves. The inferior vena cava passes through its hiatus at the junction of the right and middle leaflets of the central tendon to which it may be adherent (Fig. 32-1).

The blood supply to the diaphragm is redundant, making necrosis rare.[6] The major blood supply to the abdominal aspect of the diaphragm arises from the inferior phrenic arteries, which are branches of the abdominal aorta or celiac trunk. Additional blood supply is from the superior phrenic, pericardiophrenic, musculophrenic, and intercostal arteries. Lymphatic drainage is rich on both sides of the diaphragm

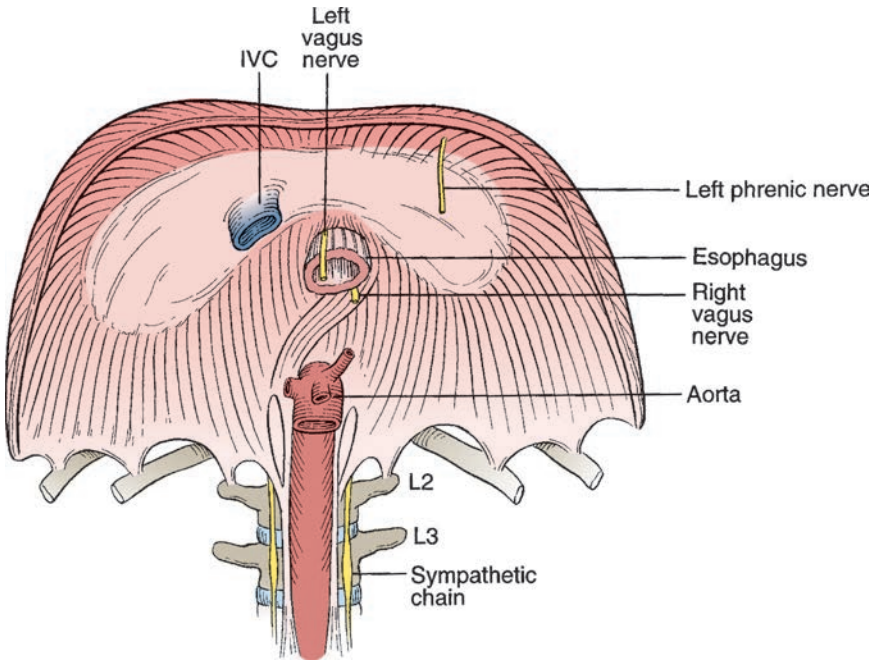


FIGURE 32-1 View of the diaphragm from the abdomen including the aortic, esophageal, and caval hiatuses. IVC, inferior vena cava.

with the peritoneal surface the major contributor to peritoneal lymphatic drainage. Innervation is principally through the phrenic nerves, with additional contribution of the sixth or seventh intercostal nerves to the costal region of the diaphragm. Both phrenic nerves enter the diaphragm near the anterior border of the central tendon. These nerves branch along the thoracic surface of the diaphragm before penetrating it and spreading branches in anterior, posterior, and lateral directions. The nerves are often buried deep in the muscle, and one should not rely on visualizing the nerves in order to choose incisions in the diaphragm. Safe diaphragmatic incisions that protect the phrenic nerves are depicted in Fig. 32-2.

PHYSIOLOGY

The diaphragm is a vital muscle involved in the function of both the digestive and respiratory systems. It participates in breathing, swallowing, coughing, defecation, emesis, micturition, parturition, sneezing, and vocalization.[7] In humans, it is the most important muscle of inspiration and can independently generate negative intrapleural pressure sufficient for respiration.[8] The innervation of the diaphragm is centrally unified in the spinal cord, and the entire diaphragm from the crura to the lateral margins has motor neuron origins intermingled within the spinal cord.[9] The crural components, however, may have their function overridden through peripheral or central mechanisms related to function of the gastrointestinal tract.

Perforation of the diaphragm can lead to acute changes in physiology. A normal pressure gradient between the abdomen and the chest leads to displacement of abdominal viscera into the chest, which may compromise both cardiac and respiratory function. Cardiac dysfunction due to reduced ventricular filling can lead to decreased cardiac output. Significant compression of the pulmonary parenchyma can lead to impaired ventilation on the ipsilateral side and, if more severe, mediastinal shift and compression of the contralateral lung. As with any herniation of portions of the gastrointestinal tract, sequelae such as ischemia, necrosis, and perforation may develop. There are no long-term outcome data that describe diaphragmatic function after repair for trauma, but it appears that normal physiologic pulmonary and gastrointestinal function is generally attained and is durable.

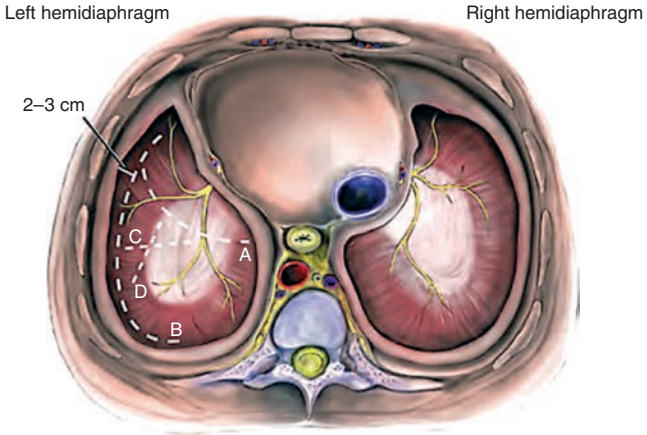


FIGURE 32-2 Surgical incisions on the diaphragm. (**A**) An incision with a risk of total paralysis of the diaphragm. (**B**) A preferred incision with minimal risk of nerve injury. (**C**, **D**) Incisions in safe areas, but with small risk of nerve injury. (Reprinted with permission from Anraku M, Shargall Y. Surgical conditions of the diaphragm: anatomy and physiology. *Thorac Surg Clin.* 2009;29:419-429. Copyright © 2009 Elsevier.)

INCIDENCE

The incidence of traumatic diaphragm injury (TDI) has been reported to range from less than 1% to 7% after blunt trauma and from 10% to 15% after penetrating trauma.[10-12] This wide range stems from an inability to identify injuries treated at nontrauma centers, deaths prior to hospital admission, and missed injuries after admission to a trauma center. The largest source of variability is the denominator in question. An analysis of the National Trauma Data Bank (NTDB) for the admission year 2012 identified 3873 TDIs among 833,309 patients, for an overall incidence of 0.46%.[13] Based on 2016 NTDB data, TDIs are rare among overall blunt trauma patients, with an incidence of 0.17%.[12] In contrast, the highest reported rate of injury occurs in patients sustaining thoracoabdominal gunshot wounds. Berg et al[14] reported TDI in 63% of such patients. Among patients with penetrating thoracoabdominal trauma, 24% to 38% have been found to have TDI when laparoscopy is performed.[15-18]

MECHANISM OF INJURY AND PATHOPHYSIOLOGY

A recent 16-year review from three regional trauma centers in Wisconsin found that 75% of TDIs were caused by penetrating mechanisms.[19] A 15-year review from the R. Adams Cowley Shock Trauma Center in Baltimore similarly reported 73% of injuries resulting from penetrating mechanisms.[20] These centers' data are consistent with the 2012 NTDB data, in which 67% of TDIs resulted from penetrating mechanisms of injury.[13] Most blunt TDIs are reported on the left side: 67% in the Wisconsin series[19] and 69% in the Baltimore series.[20] It is hypothesized that the left posterolateral diaphragm is the weakest area and thus predisposed to injury.[11] It is likely, however, that the liver affords protection to the right hemidiaphragm by mitigating the effects of blunt kinetic energy applied to the abdomen.[21] Similarly, penetrating injury to the diaphragm has had a left-sided predilection. This has been suggested, but not proven, to be related to the prevalence of right-handed assailants. In fact, recent data indicate a more even distribution. In the Baltimore series,[20] 45% of penetrating TDIs were on the right. Interestingly, in the Wisconsin series,[19] in the first half of the study period, the right:left distribution was 27%:70%; however, in the second half, it was 49%:49%.

Penetrating wounds typically result in a smaller injury, but the defects may enlarge over time and eventually lead to acute incarceration and/or strangulation of abdominal viscera if not promptly recognized and repaired.

PRESENTATION

Grimes[22] classified diaphragmatic injuries into the following three phases of presentation: acute, or during the period of recovery from injury; latent, following an asymptomatic period; and obstructive, during which time herniation leads to cardiovascular compromise or gastrointestinal obstruction or perforation. In the acute phase, patients may have minimal signs of external injury or be experiencing severe shock and respiratory compromise that may or may not be directly related to the diaphragmatic injury. In the less severely injured, possible signs and symptoms include shoulder pain, epigastric pain, vomiting, dyspnea, absent breath sounds, or bowel sounds heard during auscultation of the chest.[23] It is difficult to glean from the literature how many patients present with symptoms directly related to a TDI. Some are identified on imaging, and most are identified at surgical exploration.[19,20]

In the latent phase, TDI or visceral herniation is often asymptomatic and may be discovered incidentally on radiographic studies performed for other reasons. Although the majority of injuries are diagnosed acutely, case series have reported 3% to 15% of cases presenting in the late or obstructive phase.[24,25] Patients presenting in the obstructive phase often experience nausea, vomiting, early satiety, pain, dyspnea, postprandial pain, or generalized chest and abdominal pain. Patients may also present in extremis with signs and symptoms of septic shock due to ischemia or perforation related to strangulation or gangrene of incarcerated viscera or with cardiovascular collapse due to compression.[26] In the series by Murray et al,[27] 14 (50%) of 28 patients with posttraumatic diaphragmatic hernia presented as surgical emergencies, with an associated mortality of 11%.

ASSOCIATED INJURIES

Blunt diaphragmatic injury typically involves high-energy compression-type mechanisms, and associated injuries are common (Table 32-1). Not surprisingly, liver and lung injuries are most commonly associated with TDI. As seen in the

TABLE 32-1: Injuries Associated with Diaphragmatic Injury and the Mortality Rate Among Patients with Those Injuries in a Large Single-Center Series[20]

Associated injury	Number (%)	Mortality rate
Total patients	773	21%
Lung	592 (77%)	23%
Hemothorax	414 (54%)	24%
Liver	402 (52%)	25%
Pneumothorax	372 (48%)	15%
Rib fractures	257 (33%)	23%
Spleen	248 (32%)	13%
Bowel	153 (20%)	27%
Stomach	144 (19%)	19%
Kidney	133 (17%)	18%
Head	123 (16%)	35%
Pancreas	57 (7%)	23%
Heart	54 (7%)	63%
Pericardium	37 (5%)	60%

large series from Maryland,[20] traumatic brain injury and injuries to the heart and pericardium are less common, but their presence indicates a higher risk of death.

DIAGNOSIS AND DIAGNOSTIC TESTS

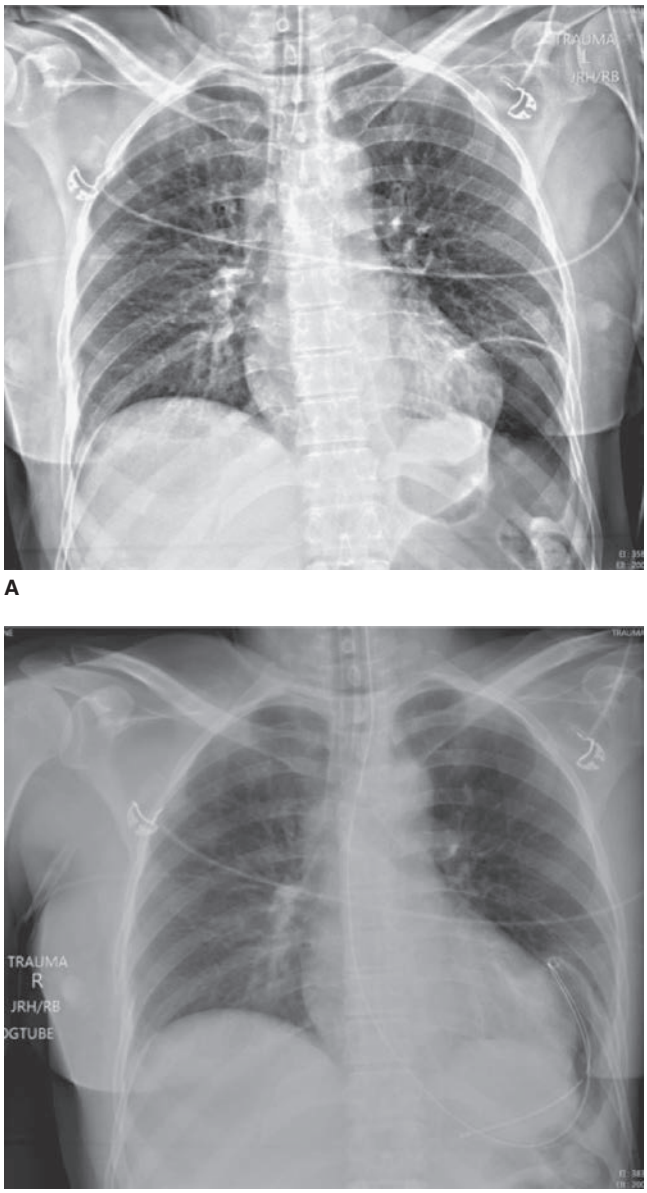
Diaphragmatic injury is often asymptomatic, or the presentation is dominated by other injuries. Consequently, the clinical diagnosis is very challenging. Information about the mechanism of injury should always be obtained from prehospital personnel, as it may direct the diagnostic evaluation. Blunt diaphragmatic injuries generally result from a sudden increase in abdominal pressure, with motor vehicle crashes being the most common blunt injury mechanism.[13] In penetrating trauma, any injury to the thoracoabdominal area should raise suspicion for a potential diaphragmatic injury.[14,18] Although most stab wounds that result in diaphragmatic injury will be in this region, gunshot wounds that injure the diaphragm may occur anywhere on the trunk.[26] Injuries are graded according to the American Association for the Surgery of Trauma Organ Injury Scale for Diaphragmatic Injuries (Table 32-2).[28]

Plain chest x-ray (CXR) is regarded as a key initial diagnostic study in any patient in whom there is a possibility of significant torso injury and especially those with thoracic or thoracoabdominal penetrating injury. Unfortunately, the overall accuracy of CXR is quite poor; the sensitivity has been reported to be in the range of 27% to 62% for left-sided injuries and 18% to 33% for right-sided injuries.[29] Most patients with TDI have a normal CXR or only nonspecific findings, such as loss of a smooth, diaphragmatic contour, ipsilateral pleural effusion, ipsilateral elevated hemidiaphragm, or mediastinal shift.[30] The only direct sign of diaphragmatic injury is the visualization of herniated abdominal viscera into the chest. A nasogastric or orogastric tube may help determine that a hyperlucency is the herniated stomach rather than a pneumothorax (Fig. 32-3A and B). In the Wisconsin series,[19] only 50 (11%) of 454 patients had TDI diagnosed based on CXR, and the authors report that none of them were identified by nasogastric tube position. Contrast swallow studies and enemas, as well as fluoroscopy, have been used in the past and may be potentially helpful but are rarely employed today (Fig. 32-4).

TABLE 32-2: Grading of Diaphragmatic Injuries

Grade	Description of injury
I	Contusion
II	Laceration ≤2 cm
III	Laceration 2–10 cm
IV	Laceration >10 cm with tissue loss ≤25 cm^2
V	Laceration with tissue loss >25 cm^2

Source: Reproduced with permission from Moore EE, Malangoni MA, Cogbill TH, et al. Organ injury scaling IV: thoracic vascular, lung, cardiac and diaphragm. *J Trauma*. 1994;36(3):299-300.



A

B

FIGURE 32-3 Chest x-ray demonstrating a left diaphragmatic injury. (**A**) Intestinal herniation seen above the diaphragm. (**B**) Verified by placement of a gastric catheter.

Ultrasonography is employed frequently during trauma resuscitation, and evaluation for hemoperitoneum, hemopericardium, pneumothorax, and hemothorax is routine in trauma centers. The diaphragm can be recognized as a curved hyperechoic structure, and its movement can be observed with respiration. Signs of TDI include the presence of floating diaphragm edges, intrathoracic visceral herniation, and absence of diaphragmatic excursion.[31-34] Rip's absent organ sign, referring to the nonvisualization of the spleen or heart, has also been identified as a sign of TDI,[34] and in patients with right-sided ruptures, a finding of liver sliding against the chest wall has been reported.[33] There are inadequate data to evaluate the sensitivity or specificity of these findings.

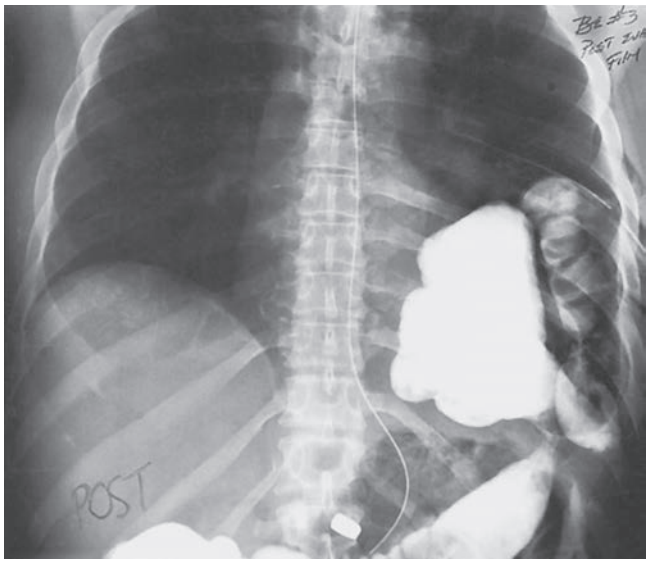
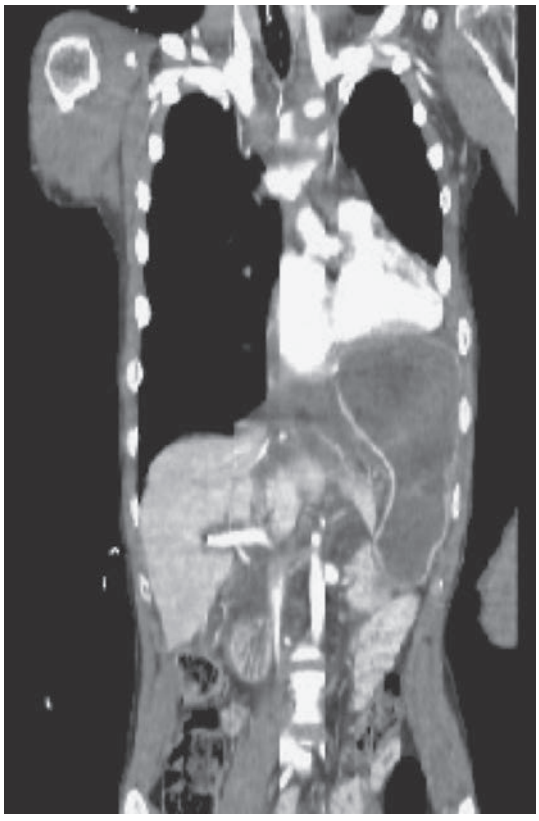


FIGURE 32-4 Contrast enema with splenic flexure identified above the diaphragm.

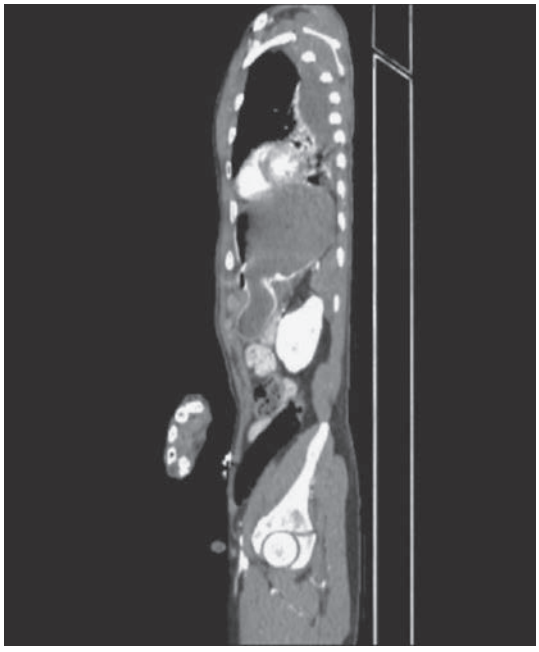
It is likely that the advent of routine helical computed tomography (CT) scanning has decreased the incidence of missed diaphragmatic injuries after blunt trauma.[35] Helical CT for the detection of blunt TDI has a reported sensitivity of 71% to 100% and specificity of 75% to 100%; sensitivity improves to 78% to 100% if only left-sided injuries are included.[36] Findings consistent with diaphragmatic disruption include the following: direct visualization of the defect; segmental nonvisualization of the diaphragm; herniation of viscera; constriction of a herniated viscus or a "collar sign"; dependent viscera sign or contact of intra-abdominal organs with the posterior chest wall; thickening of the diaphragm; and active extravasation of contrast at the level of the diaphragm (Fig. 32-5A and B). Multidetector CT scanning has been evaluated in patients with penetrating trauma with a reported sensitivity and specificity of 87% and 72%, respectively.[37] In addition to the signs listed earlier, contiguous visceral injuries on both sides of the diaphragm are an important potential finding in penetrating trauma.

Magnetic resonance imaging (MRI) provides excellent resolution of the diaphragm and separates it from surrounding structures including the liver and atelectatic lung.[36] In general, the signs seen on MRI are the same as those seen on CT (Fig. 32-6). The utility of MRI is limited due to availability, time, and other issues. In the hemodynamically normal patient with equivocal CT findings, MRI may have a role, although there have not been any large studies examining its accuracy to date.

Despite the imaging options, most patients are diagnosed intraoperatively. Over the 16-year study period in Wisconsin trauma centers,[19] 29% of blunt and 71% of penetrating TDIs were diagnosed intraoperatively. This is in part due to patients being taken directly to the operating room due to physiologic condition, but it has also been recognized that the diagnosis of penetrating injuries in particular can be very



A



B

FIGURE 32-5 Computed tomography scan demonstrating herniated viscus in coronal plane (**A**) and in the sagittal plane (**B**).

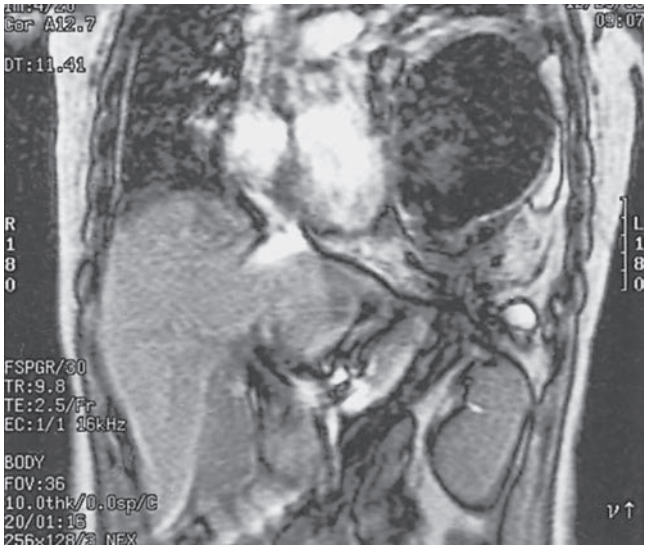


FIGURE 32-6 Magnetic resonance imaging with gastric herniation through diaphragmatic defect.

difficult. Diagnostic peritoneal lavage was employed liberally in the 1970s to 1990s and is currently advocated as an option in a few specific circumstances,[38] but it has all but disappeared from the trauma surgeon's armamentarium. Currently, laparoscopy and thoracoscopy are considered the best diagnostic tests for TDI.

Uribe et al[15] performed routine thoracoscopy in patients with penetrating thoracoabdominal injuries and found diaphragmatic injuries in 32%. Of note, the authors performed laparotomy on all patients with diaphragmatic injuries and found that 89% of them had intra-abdominal injuries that required surgical repair. In the 1990s, a number of investigators attempted to clarify the role of laparoscopy. Murray et al[16] prospectively studied 110 patients with penetrating injuries to the left lower chest and found occult diaphragmatic injuries in 26 (24%) of them. Friese et al[17] confirmed these results, also finding diaphragmatic injuries in 24% (8 of 34) of patients with penetrating thoracoabdominal injuries. They further evaluated the accuracy of laparoscopy by following it with laparotomy and found one (11%) missed injury. In more contemporary experience, among 595 patients with thoracoabdominal stab wounds who arrived to the hospital alive, 38% were found to have TDI.[18] Of note, the authors reported that CT scan did not show evidence of diaphragmatic injury in 32% of patients. The same group, analyzing their experience with 984 thoracoabdominal gunshot victims, found that 63% had diaphragmatic injuries.[14] The authors cautioned that preoperative diagnosis of TDI is inaccurate and that there is limited role for nonoperative management in this subgroup of patients.

A recent practice management guideline from the Eastern Association for the Surgery of Trauma conditionally recommends laparoscopy over CT for the diagnosis of left-sided thoracoabdominal stab wounds.[39] The superior accuracy of laparoscopy is weighed against procedural risks, which are few. A diagnostic algorithm for identifying diaphragmatic injury is presented in Fig. 32-7.

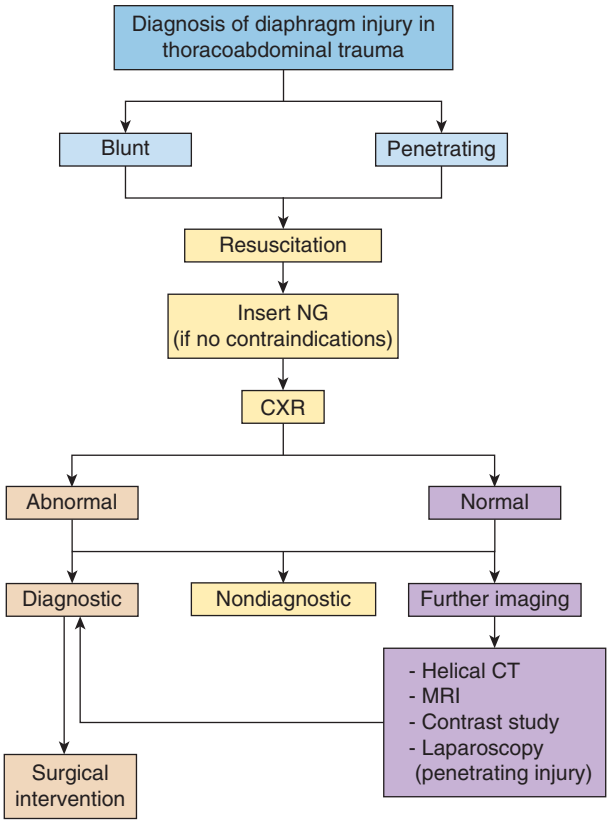


FIGURE 32-7 Diagnostic algorithm for identification of diaphragmatic injury. CT, computed tomography; CXR, chest x-ray; MRI, magnetic resonance imaging; NG, nasogastric.

REPAIR OF ACUTE DIAPHRAGMATIC INJURIES

The two principles of repairing acute diaphragmatic hernias are complete reduction of the herniated organs back into the abdominal cavity and watertight closure of the defect to prevent recurrence. Given the high rate of associated abdominal injuries following blunt TDI, on average 1.6 intra-abdominal injuries per patient, repair of the acutely blunt-injured diaphragm is best performed via an exploratory laparotomy. Similarly, unstable patients with penetrating injuries require laparotomy. Stable patients with penetrating injuries may undergo diagnostic laparoscopy, with laparoscopic repair of injuries or conversion to laparotomy depending on the laparoscopic skills of the surgeon. Although there are limited data, it appears that nonoperative management of stable patients with right-sided penetrating wounds is safe.[39] The ligamentous attachments of the liver prevent its herniation through relatively small penetrating wounds, and the liver itself prevents intestinal herniation into the right chest. Only in the setting of large defects or leakage of bile into the chest is repair required.

The right hemidiaphragm is best inspected after transection of the falciform ligament and downward traction of the liver. The left hemidiaphragm can be inspected by applying gentle downward retraction of the spleen and greater curvature of the stomach. The central tendon of the diaphragm should also be examined, along with the esophageal hiatus. Reduction of the intra-abdominal contents is generally not difficult in the period immediately following injury. If the herniated contents are difficult to reduce, the phrenotomy can be partially extended to facilitate reduction, with care taken to avoid injury to the phrenic nerve.

It may be necessary to carefully debride the edges of a laceration if devitalized tissue is found, as with a high-velocity missile or close-range shotgun wound. The edges of the diaphragmatic laceration should then be grasped with Allis clamps and the laceration spread apart to inspect the ipsilateral pleural cavity. This allows for evaluation of ongoing hemorrhage from within the thoracic cavity, as well as the determination of the degree of contamination between the abdominal and thoracic cavities. Small diaphragmatic disruptions, commonly seen after penetrating trauma, can generally be repaired using interrupted nonabsorbable sutures. Larger defects, more likely associated with blunt trauma, may be repaired in a number of different ways, including interrupted figure of eight, horizontal mattress sutures, a running hemostatic suture line, or a two-layer repair (Fig. 32-8). Generally, a #0 or #1 monofilament or braided nonabsorbable suture is used. The authors prefer a #1 nonabsorbable monofilament running suture for the repair of traumatic diaphragmatic defects. In patients in whom a laceration through the central tendon exposes the inferior aspect of the heart, meticulous attention is given to the placement of the sutures to prevent inadvertent puncture or laceration of the myocardium. At the completion of the repair, the integrity of the suture line may be tested by increasing intrathoracic pressure with the administration of large tidal volumes and assessment of diaphragmatic motion. This maneuver is repeated with the field flooded with sterile saline to determine if there is escape of air through the suture line.

In cases where there is concomitant injury to a hollow viscus in the abdomen, contamination of the chest will have occurred due to the pressure gradient between the positive pressure in the abdomen and the negative pressure in the thoracic cavity. In this event, careful irrigation of the thoracic cavity through the diaphragmatic disruption is advised prior to diaphragmatic repair, as empyema is three times more prevalent when there is an associated injury to the bowel.[25,40] Zellweger et al[41] studied the management of patients with

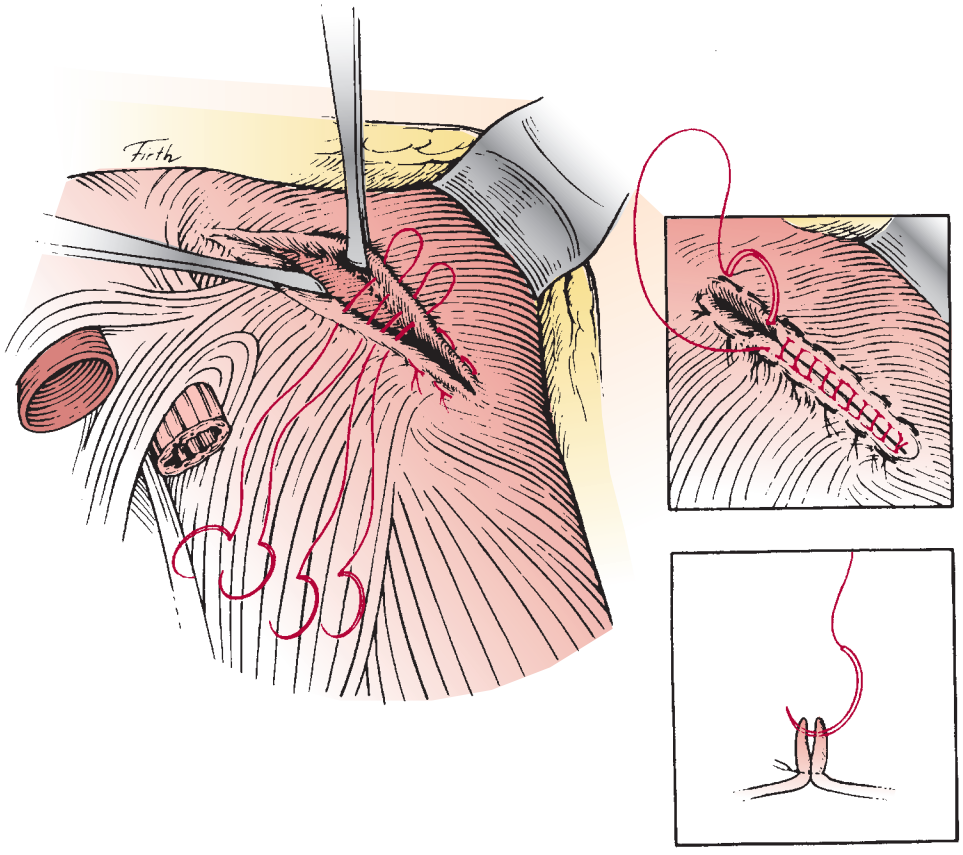


FIGURE 32-8 Technique for two-layer repair of diaphragmatic defect. (Reproduced with permission from Juan A. Asensio, MD, FACS, FCCM, and Demetrios Demetriades, MD, PhD, FACS.)

penetrating thoracoabdominal wounds that injured the diaphragm and gastrointestinal tract and/or liver. They demonstrated that a transdiaphragmatic washout of the pleural cavity was an effective strategy to decrease thoracic contamination.

Laparoscopic repair of diaphragmatic injuries is feasible.[39] A diaphragmatic injury diagnosed by laparoscopy in the absence of other injuries mandating laparotomy or thoracotomy can be repaired with this approach.[42] Laparoscopic repairs of diaphragmatic injuries can be performed with sutures or staples.[13] The decision to proceed laparoscopically should be solely dependent on the skill of the operating surgeon. For laparoscopic explorations to rule out a left-sided diaphragmatic injury, port placement is important. The initial port should be at the level of the umbilicus or just above depending on the patient's body habitus. A subxiphoid port is placed for retraction of the liver, and a port is placed laterally on the left side just below the costal margin. This port is used to maintain traction on the stomach or other cranially herniated abdominal organs. The umbilical port is used for the 30° laparoscopic camera. Two additional ports in the bilateral mid-clavicular lines are used as working ports. These two ports are also placed in a subcostal location. After placing the patient in steep reverse Trendelenburg position and careful reduction of the abdominal contents back into the abdomen, the defect is repaired. Techniques of repair are identical to the open methods. For right-sided injuries, port placement is similar, moving the left lateral subcostal port to the right. The right lobe of the liver is retracted medially or caudally. The falciform ligament and the right triangular ligament may require division for adequate retraction and exposure of the injury. During repair, air will be introduced into the pleural cavity. Tube thoracostomy is recommended if there was a significant hemo- or pneumothorax preceding repair. In the absence of either, air may be removed from the pleural space via a catheter just as the diaphragmatic repair sutures are tied down; this may be assisted by a Valsalva maneuver and may obviate the need for tube thoracostomy. A postoperative CXR is recommended.

At times, a thoracotomy is required for the management of a massive hemothorax, generally defined as greater than 1500 mL of blood on insertion of a chest tube or in the first 15 to 30 minutes or more than 200 mL of blood per hour for the first 4 hours after trauma.[43] A laceration of the right hemidiaphragm with an associated laceration of the liver may present as a massive hemothorax, with the diagnosis made at the time of thoracotomy. In this scenario, the diaphragm may be repaired through the chest, but a formal laparotomy will be necessary for the operative management of the hepatic injury and to rule out other associated intra-abdominal injuries.

For thoracoscopic management of a known diaphragmatic injury, the patient is placed in the lateral decubitus position with the arm abducted to allow maximal superior displacement of the scapula. The initial 2-cm incision should be placed just below the tip of the scapula. Two further incisions are then placed to complete a triangle based on the patient's intrathoracic anatomy. The principles of visceral reduction and the technique of repair remain similar to those used in open surgery.

Disruption of the diaphragm following high-energy crushing injuries or major deceleration can result in avulsion of the diaphragm from its attachments to the chest wall. Repair of this injury may require an ipsilateral thoracotomy, which allows horizontal mattress sutures to be placed around the ribs and secures the diaphragm into its normal anatomic position. In the presence of a flail segment of the ipsilateral chest wall, formal fixation of the ribs may be required to facilitate this complex repair of the diaphragm.[23] Prosthetic material for diaphragmatic reconstruction in the acute setting is rarely indicated, because tissue retraction and loss have not occurred and concomitant gastrointestinal injuries may lead to an increased rate of postoperative infection.

Massive diaphragmatic destruction such as that caused by thoracoabdominal shotgun injuries merits special mention. Bender and Lucas[44] described the immediate reconstruction of the chest wall following this type of injury by first detaching the affected hemidiaphragm anteriorly, laterally, and posteriorly. The diaphragm was then translocated to a position above the full-thickness chest wall defect, which converted the defect functionally into an abdominal wall defect. This is performed by suturing the ribs at a higher intercostal space, while the abdominal wall defect is managed with local wound care in anticipation of reconstruction with either split-thickness skin grafts or myocutaneous flaps at a later date.

REPAIR OF CHRONIC DIAPHRAGMATIC INJURIES

Patients who initially sustain small, undetected diaphragmatic lacerations may remain asymptomatic or may experience a progressive increase in visceral herniation of the omentum or of a hollow viscus.[45,46] The diaphragm as a muscle is quick to retract and atrophy. Therefore, tissue that could be approximated easily early after injury retracts in the latent or obstructive phase to the point where approximation is impossible at a late reoperation.

Chronic diaphragmatic hernias can be repaired either transabdominally or transthoracically, with the choice generally determined by the surgeon's preference.[47,48] The classical teaching, however, is that large chronic posttraumatic diaphragmatic hernias should be approached using a thoracotomy to allow for lysis of intrathoracic adhesions. On occasion, a combined approach may be indicated to complete the procedure safely and effectively. The transthoracic approach offers several benefits including the direct visualization of intrathoracic adhesions, which may extend all the way to the apex of the pleural cavity, and avoidance of abdominal adhesions from the prior trauma.[27,49-51] The thoracic approach has been performed successfully using both open and thoracoscopic techniques.[52-54] The open procedure is generally performed through the seventh or eighth intercostal space using a posterolateral approach. On rare occasions, an extension along the costal arch into a thoracoabdominal incision may be needed to allow access to the abdominal cavity.

As noted earlier, the transabdominal approach is often considered less attractive due to the inherent difficulty of visualizing adhesions to the lung and other intrathoracic structures and dealing with potential dense intra-abdominal adhesions from prior trauma and/or surgery. Additionally, if the procedure cannot be completed at laparotomy, the incision will need to be closed and the patient repositioned for a posterolateral thoracotomy. This is because it is difficult to visualize the posterior diaphragm from an anterolateral approach. Laparoscopy has been described for the reduction of chronic diaphragmatic hernias as well, although problems with an iatrogenic pneumothorax have mandated insertion of a thoracostomy tube.[27,55-58] No matter which approach is used, current recommendations support the role of open surgery for patients with defects greater than 10 cm and for those whose hernia extends to or through the esophageal hiatus.[39]

Careful dissection is required to free the entire diaphragm from its adhesions to surrounding tissues. Once mobilized, the edges should be brought together to evaluate for tension. If the edges can be approximated easily, the repair should be performed primarily as described in the section on acute diaphragmatic injuries. Generally, defects up to 8 cm can be closed primarily.[59] Primary repair of larger defects in the diaphragm will cause unacceptable flattening of the diaphragm, and prosthetic material will be needed to reconstruct the diaphragm. Many different prostheses have been used, including Mersilene, polytetrafluoroethylene (PTFE), polypropylene, and polydioxanone meshes.[50,55,60-63] Recently, a 2-mm-thick PTFE patch has been advocated as an excellent material for reconstructing the diaphragm because it provides the necessary strength and is watertight.[59] Generally, the patch is sutured using a running 0 nonabsorbable suture around the edges of the defect, often starting medially (Fig. 32-9). The patch must be tailored to minimize laxity in the diaphragm. Full-thickness bites in the diaphragm are necessary, with care taken to avoid injury to underlying structures. Other alternatives to synthetic mesh include a bovine pericardial patch, which in case series has proven to be an effective alternative to prosthetic materials.[64]

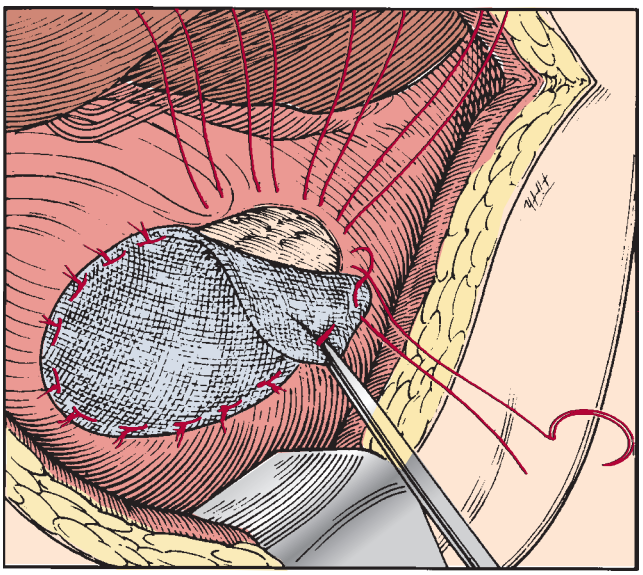


FIGURE 32-9 Technique for repair of the diaphragm using a prosthetic. (Reproduced with permission from Juan A. Asensio, MD, FACS, FCCM, and Demetrios Demetriades, MD, PhD, FACS.)

For injuries extending laterally to the chest wall without adequate tissue for fixation, the prosthetic can be secured with interrupted sutures placed around the ribs, following the natural course of the native diaphragm. Medially, the mesh can be secured either to pericardium or to the posterior crus if inadequate native diaphragmatic tissue exists.[59] In contaminated fields, autologous tissue may be used instead of PTFE to reconstruct large diaphragmatic defects. Latissimus dorsi, rectus abdominis, external oblique, and transversus abdominis flaps have been described, mainly in pediatric populations.[65-69] The benefit of autologous tissue in the pediatric population is that it allows for growth of the child. A surgical algorithm to approach diaphragmatic injuries is presented in Fig. 32-10.

OUTCOMES

Mortality following diaphragmatic injury is dependent on the severity of the associated injuries.[20] Mortality rates vary between 4% and 9% in penetrating injuries and 15% and 24% after blunt diaphragm injuries.[19,20] Recurrence rates of diaphragmatic hernias following repair are difficult to ascertain; however, it appears that the recurrence rate is higher when absorbable suture is used for the initial repair.[25]

Postoperative morbidity directly related to the acute surgical repair includes suture line dehiscence or failure of the diaphragmatic repair, paralysis of the hemidiaphragm secondary to an iatrogenic injury to the phrenic nerve, respiratory insufficiency, empyema, and subphrenic abscess. The complication rate after repair of a diaphragmatic injury has ranged from 30% to 68%.[70,71] Atelectasis has been documented in 11% to 68% of patients, with pneumonia and pleural effusions reported in another 10% to 23%. Sepsis, multiorgan system failure, hepatic abscess, and empyema have been reported in 2% to 10% of patients.[70,71]

When complication rates were compared in blunt versus penetrating injuries of the diaphragm in one review, patients with blunt trauma had a 60% complication rate versus a 40% complication rate in those with penetrating trauma.[72] Most of the morbidity reported in these studies is clearly the result of the large number of associated injuries present in association with diaphragmatic injuries.[70,72] Mortality following repair of a chronic diaphragmatic hernia depends entirely on the presence or absence of obstructive symptoms at the time of presentation. A mortality of less than 10% is expected in patients with asymptomatic diaphragmatic hernias, and this is related to the patient's comorbidities and not to the repair.[26,73] Patients presenting with gastrointestinal obstruction have a significantly higher morbidity (>60%) and mortality (25%–80%). This reinforces the need to pursue the diagnosis and surgical

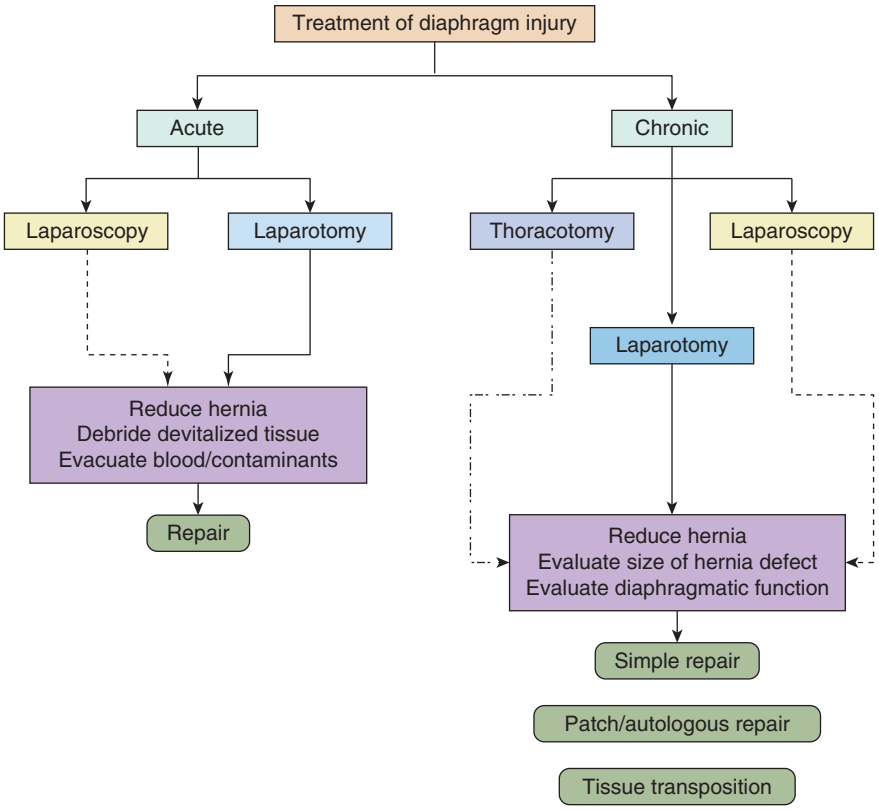


FIGURE 32-10 Algorithm for repair of an acute or chronic diaphragmatic defect.

management of diaphragmatic injuries prior to the onset of obstructive symptoms with chronic hernias.

PERICARDIODIAPHRAGMATIC RUPTURE

Blunt ruptures of the central tendon of the diaphragm involving the pericardium are rare and may present as acute injuries or as chronic hernias following missed injuries. Most are caused by combined blunt trauma to the chest and abdomen; however, isolated trauma to either one of the cavities can also cause this entity. Simultaneous rupture of the pericardium into the left and the right pleural spaces has also been described, as has herniation of the heart inferiorly into the peritoneal cavity.[74] There is a high incidence of associated injuries including musculoskeletal injuries that occur predominantly on the left side of the body. The organs most frequently involved in pericardial herniation are the transverse colon, stomach, omentum, liver, and small bowel. Exploratory laparotomy is recommended as the preferred approach for the acute repair of these injuries.[74]

CONCLUSION

Diaphragmatic injuries may be associated with other severe life-threatening injuries as after blunt trauma or may be subtle in their presentation in following penetrating trauma. In either situation, a high index of suspicion is necessary to make the diagnosis. As modern imaging techniques improve, diagnostic accuracy similarly improves but can still miss injuries, thus the reliance on diagnostic laparoscopy in patients with a high suspicion for injury. Once the diagnosis is made, repair in the acute phase can usually be accomplished using the surgical techniques described. In the latent or obstructive phases of presentation, repair or reconstruction of the diaphragm can be a surgical challenge. If gastrointestinal obstruction, perforation, or ischemia occurs with a chronic posttraumatic diaphragmatic hernia, postoperative morbidity and mortality are significant.

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33

Liver and Biliary Tract

Brandon Bruns • Rosemary Kozar

KEY POINTS

- Sixty to ninety percent of hepatic injuries are treated nonoperatively.
- A contrast-enhanced computed tomography scan documenting extravasation in the liver may require angioembolization or operation.
- Organ Injury Scale grade I or II hepatic injuries do not require drains at laparotomy; for higher grade injuries with evidence of biliary leak at laparotomy, drainage should be considered.
- Partial disruption of hepatic segments II/III or VI/VII are managed with resectional debridement at the time of laparotomy.
- Perihepatic packing is useful in selected patients with intraoperative coagulopathies or ongoing bleeding.

INTRODUCTION

Due to its large size, the liver is one of the most commonly injured organs after trauma.[1] The size of the organ and its position under the right costal margin make the liver susceptible to both blunt and penetrating trauma to the right thoracoabdomen and right upper quadrant of the abdomen. The management of hepatic injuries continues to evolve with improved modes of diagnosis and nonoperative and operative management. The most severe hepatic parenchymal and retrohepatic venous injuries, as well as those involving the portal triad, continue to have a high mortality; therefore, despite progress, the opportunity for further improvements in management exists.

ANATOMY (SEE ATLAS FIGURE 41)

A comprehensive knowledge of hepatic anatomy is essential to the proper treatment of trauma to the liver. An understanding of the ligamentous attachments, parenchyma, and intraparenchymal and extraparenchymal vascularity of the liver is key to the effective application of methods for control and repair of hepatic injuries (Fig. 33-1).

Lobes

The liver is divided into two lobes by a 75° angle line traversing from the gallbladder fossa inferiorly to the left side of the inferior vena cava superiorly. The left lobe includes the hepatic tissue to the left of the falciform ligament along with the quadrate and caudate lobes, whereas the right lobe consists of the remaining parenchyma.

Functional Anatomy (See Atlas Figure 42)

The functional anatomy of the liver separates the liver into segments pertinent to resection. Couinaud provided the basis of modern resection planes by dividing the liver into eight segments (I–VIII) based on the distribution of the portal vein (horizontal plane) and hepatic veins (vertical planes).[2] The horizontal plane divides the sections of the liver into superior and inferior segments, as follows:

- Right anterior (or medial) section: Segment V below the portal plane and segment VIII above
- Right posterior or lateral section: Segment VI below the portal plane and segment VII above
- Left medial section: Segment IVb below the portal plane and segment IVa above
- Left lateral section: Segment III below the portal plane and segment II above

The right hepatic vein traverses between the right posterolateral (segments VI and VII) and right anteromedial (segments V and VIII) segments. On the left, the left hepatic vein delineates the anterior (segments III and IV) and posterior

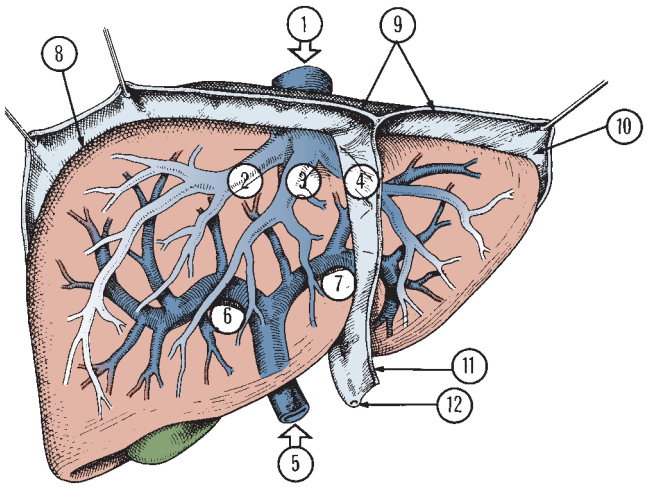


FIGURE 33-1 Surgical anatomy of the liver: (1) inferior vena cava; (2) right hepatic vein; (3) middle hepatic vein; (4) left hepatic vein; (5) portal vein; (6) right branch portal vein; (7) left branch portal vein; (8) right triangular ligament; (9) coronary ligament; (10) left triangular ligament; (11) falciform ligament; (12) ligamentum teres.

(segment II) segments. The caudate lobe (segment I) drains directly into the inferior vena cava (Fig. 33-2).

Common Hepatic Artery

The common hepatic artery is one of the three main branches of the celiac axis in the visceral portion of the abdominal aorta. It becomes the proper hepatic artery beyond the gastroduodenal artery and is found in the porta hepatis, typically to the left of the common bile duct and anterior to the portal vein. Transversely incising the peritoneum overlying the hepatoduodenal ligament exposes the proper hepatic artery, a maneuver facilitated by mobilization of the hepatic flexure

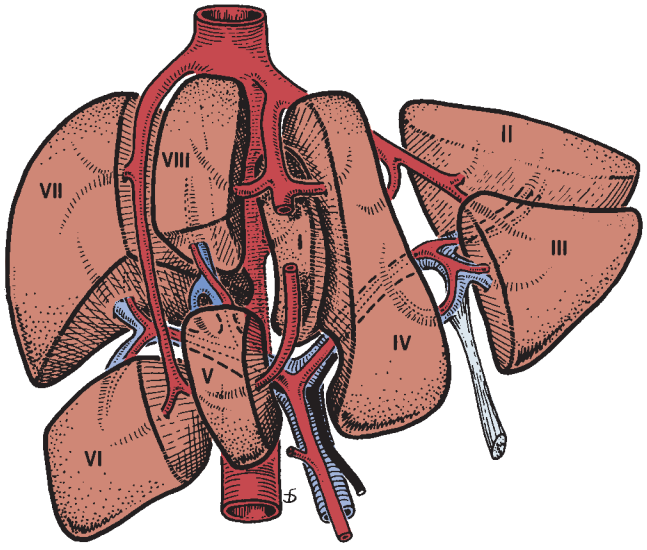


FIGURE 33-2 Functional division of the liver, according to Couinaud's nomenclature. (Reproduced with permission from Blumgart LH, ed. *Surgery of the Liver and Biliary Tract.* New York, NY: Churchill Livingstone; 1988. © Elsevier.)

of the colon toward the midline. At the hilum of the liver, the proper hepatic artery bifurcates into a right (the longer branch) and a left branch. These furnish approximately 25% to 30% of all hepatic blood flow (overall, 1.5 L/min) and 50% of hepatic oxygenation.

There are a number of anatomic variants. The most frequent (11%) is the aberrant origin of the right hepatic artery from the superior mesenteric artery traversing behind the duodenum. Other variants include a left hepatic artery origin from the left gastric artery (8%) and the left and right hepatic arteries arising from the superior mesenteric artery (9%). Given these multiple variants, great care is taken when controlling traumatic hemorrhage in this region.

Hepatic Veins

The hepatic veins develop from the hepatocytes' central lobar veins. The superior, middle, and inferior vein branches originating from the right lobe form the right hepatic vein. The two veins arising from segments IV and V combine to form the middle hepatic vein, frequently including a branch from the posterior portion of segment VIII. In 90% of patients, the middle hepatic vein joins the left hepatic vein just before draining into the inferior vena cava. The left hepatic vein is more variable in its segmental origin. Most important is the posterior positioning of the left hepatic vein when dissecting the left coronary ligament, so care must be taken in this area to avoid inadvertent injury. The liver must be fully mobilized for visualization of the hepatic veins, which are notoriously fragile and can be easily torn. The retrohepatic vena cava is about 8 to 10 cm in length and receives the blood of the hepatic veins in addition to multiple small direct hepatic vessels.

Portal Vein

The portal vein is formed from the confluence of the splenic and superior mesenteric veins directly behind the head of the pancreas. It provides about 75% of hepatic blood flow and 50% of hepatic oxygen. The portal vein lies posterior to the hepatic artery and common bile duct as it ascends toward the liver. At the parenchyma, the portal vein divides into a short right and a longer left extrahepatic branch. Surgically, the portal vein can be approached by division of the pancreas at its neck or with a generous Kocher maneuver of the duodenum toward the midline.

Ligaments

When operating on the liver, it is crucial to understand the orientation of its ligamentous attachments. The coronary ligaments attach the diaphragm to the superior parietal surface of the liver. The triangular ligaments are at the lateral extensions of the right and left coronary ligaments. The falciform ligament, with the underlying ligamentum teres, attaches to the anterior peritoneal cavity. The medial portion of the coronary ligaments is where the hepatic veins traverse; therefore,

TABLE 33-1: Liver Injury Scale (2018 Revision)

AAST grade	AIS severity	Imaging criteria (CT findings)	Operative criteria	Pathologic criteria
I	1	• Subcapsular hematoma <10% surface area • Parenchymal laceration <1 cm in depth	• Subcapsular hematoma <10% surface area • Parenchymal laceration <1 cm in depth • Capsular tear	• Subcapsular hematoma <10% surface area • Parenchymal laceration <1 cm • Capsular tear
II	2	• Subcapsular hematoma 10%–50% surface area; intraparenchymal hematoma <10 cm in diameter • Laceration 1–3 cm in depth and ≤10 cm length	• Subcapsular hematoma 10%–50% surface area; intraparenchymal hematoma <10 cm in diameter • Laceration 1–3 cm in depth and ≤10 cm length	• Subcapsular hematoma 10%–50% surface area; intraparenchymal hematoma <10 cm in diameter • Laceration 1–3 cm depth and ≤10 cm length
III	3	• Subcapsular hematoma >50% surface area; ruptured subcapsular or parenchymal hematoma • Intraparenchymal hematoma >10 cm • Laceration >3 cm in depth • Any injury in the presence of a liver vascular injury or active bleeding contained within liver parenchyma	• Subcapsular hematoma >50% surface area or expanding; ruptured subcapsular or parenchymal hematoma • Intraparenchymal hematoma >10 cm • Laceration >3 cm in depth	• Subcapsular hematoma >50% surface area; ruptured subcapsular or intraparenchymal hematoma • Intraparenchymal hematoma >10 cm • Laceration >3 cm in depth
IV	4	• Parenchymal disruption involving 25%–75% of a hepatic lobe • Active bleeding extending beyond the liver parenchyma into the peritoneum	• Parenchymal disruption involving 25%–75% of a hepatic lobe • Intraparenchymal hematoma	• Parenchymal disruption involving 25%–75% of a hepatic lobe
V	5	• Parenchymal disruption >75% of hepatic lobe • Juxtahepatic venous injury to include retrohepatic vena cava and central major hepatic veins	• Parenchymal disruption >75% of hepatic lobe • Juxtahepatic venous injury to include retrohepatic vena cava and central major hepatic veins	• Parenchymal disruption >75% of hepatic lobe • Juxtahepatic venous injury to include retrohepatic vena cava and central major hepatic veins

- Vascular injury is defined as a pseudoaneurysm or arteriovenous fistula and appears as a focal collection of vascular contrast that decreases in attenuation with delayed imaging. Active bleeding from a vascular injury presents as vascular contrast, focal or diffuse, that increases in size or attenuation in delayed phase. Vascular thrombosis can lead to organ infarction.
- Grade based on highest grade assessment made on imaging, at operation, or on pathologic specimen.
- More than one grade of liver injury may be present and should be classified by the higher grade of injury.
- Advance one grade for multiple injuries up to a grade III.

Source: Adapted with permission from Kozar RK, Crandall M, Shanmuganathan K, et al. Organ Injury Scaling 2018 update: spleen, liver, and kidney. *J Trauma Acute Care Surg*. 2018;85:1119-1122.

dissection in this area is done cautiously. In order to effectively visualize the liver during operative therapy, these ligaments must be divided and the liver fully mobilized into the field of view.

ORGAN INJURY SCALE

The American Association for the Surgery of Trauma recently revised the Organ Injury Scale for the liver.[3] The new scale includes the following three sets of criteria to assign grade: imaging, operative, and pathologic. For the first time, the scale incorporates findings from computed tomography (CT) scans (Table 33-1). Additionally, vascular injuries, defined as pseudoaneurysms or arteriovenous fistulae, are included. This classification provides for uniform comparisons of hepatic injuries managed both operatively and nonoperatively. An example of a grade IV hepatic laceration with involvement of 25% to 75% of a hepatic lobe is shown in Fig. 33-3.

INITIAL MANAGEMENT

Care for the patient with a possible hepatic injury should proceed by the tenets of Advanced Trauma Life Support (ATLS). After performing a primary survey, the search for ongoing hemorrhage ensues. Given its size, the liver is a likely source of active bleeding in the hypotensive patient after truncal trauma. Resuscitation strategies continue to focus on early transfusion of blood products and the establishment of a ratio of packed red cells to plasma to platelets that is more similar to whole blood and is reliant on a viscoelastic assay.

Plain radiographs and surgeon-performed ultrasound obtained in the trauma resuscitation unit may give clues

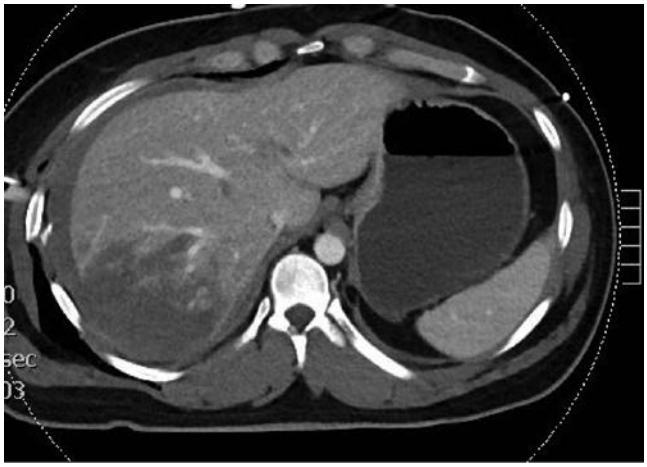


FIGURE 33-3 Computed tomography showing a grade IV liver laceration in a patient sustaining blunt injury to the right thoracoabdominal region.

to a possible hepatic injury. The presence of right-sided rib fractures and/or a hemothorax on chest radiograph or hemoperitoneum on ultrasound may raise suspicion. Although nonoperative management of hepatic injuries has become routine, a patient exhibiting clear-cut peritoneal signs and/or hemodynamic instability likely requires operative exploration. Often overlooked is the detrimental effect that hypothermia can have on coagulation, particularly for the patient with a significant hepatic injury. Type and crossmatch and measurement of hemoglobin levels, coagulation parameters, and a viscoelastic assay (thromboelastography/rotational thromboelastometry) should all be performed early in the resuscitation and repeated as necessary.

DIAGNOSIS OF HEPATIC INJURY

Hemodynamically Unstable Patient

If after the primary survey and initial resuscitation the patient remains hemodynamically unstable, determining the possible causes of the continued shock state is necessary. This can be difficult in patients with injuries involving multiple organ systems, but the correct body cavity that harbors the ongoing hemorrhage must be identified and treated. An intra-abdominal injury can be an obvious cause of instability, especially if the physical exam reveals peritoneal signs or a penetrating injury or if fluid is noted on the focused abdominal sonography for trauma (FAST). Similarly, chest and pelvic x-rays are mandatory to ensure the ongoing blood loss is not from a hemothorax or a rapidly expanding pelvic hematoma.

Focused Abdominal Sonography for Trauma

The FAST exam is the primary modality for the determination of hemoperitoneum in the unstable patient. Richards et al[4] reported a 98% sensitivity of ultrasound for hemoperitoneum in grades III and higher hepatic injury; however, they were not able to identify the anatomic location of the hepatic parenchymal injury in 67% of these severely damaged livers. A multi-institutional study by Rozycki et al[5] concluded that the right upper quadrant is the most common site where a hemoperitoneum accumulates in blunt abdominal trauma. Diagnostic peritoneal aspirate is also a method for determining the presence of intraperitoneal blood, although it has been largely replaced by ultrasound for the diagnosis of hemoperitoneum.

Hemodynamically Normal Patient

The FAST examination has proven to be a very good diagnostic tool in the diagnosis of hemoperitoneum in the blunt trauma patient, but a negative FAST does not preclude the presence of a hepatic injury.[6]

Since the first use of CT to diagnose intra-abdominal injuries in the early 1980s, it has become a routine part of the management of trauma patients.[7,8] In recent years, the availability of the helical CT scan has improved resolution and speed of scanning. Being able to grade the extent of visceral injury and to follow the evolution of an existing injury can determine if nonoperative management is possible and successful. A contrast-enhanced helical CT scan provides information on injury grade, amount of hemoperitoneum, active extravasation of contrast (Fig. 33-4), and the presence of vascular injuries.

Additionally, triple-contrast CT has long been used in penetrating wounds to the back and flank with excellent sensitivity; however, the sensitivity for injuries to the diaphragm and small bowel is less.[9] Because of the risk of hollow viscus injury, minor hepatic lacerations can be evaluated and nonoperatively managed with CT guidance, but continued frequent abdominal exams must also be performed.

Laparoscopy has been successfully used to diagnose peritoneal penetration in penetrating trauma, thus saving the patient from a nontherapeutic exploratory laparotomy.[10] Repair of a hepatic injury found at laparoscopy has been reported in hemodynamically stable patients as well.[11]

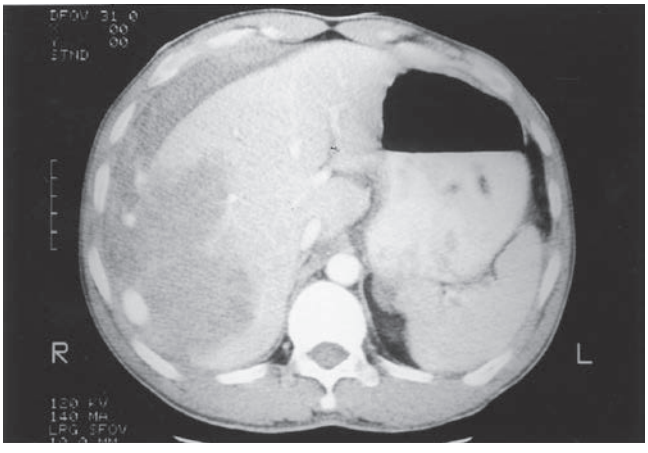


FIGURE 33-4 Computed tomography scan demonstrating a "contrast blush," indicative of active arterial bleeding in a patient with a grade IV blunt hepatic injury.

MANAGEMENT OF HEPATIC TRAUMA

Blunt hepatic injury traverses almost exclusively along the lines dividing the segments of the liver. This most likely occurs due to the strength of the fibrous covering around the portal triad preventing injury from transecting these structures. The hepatic veins do not have a similar fibrous structure and, therefore, are the primary vascular structures injured in blunt trauma. On the other hand, penetrating trauma involves both venous and arterial injury with direct transection of any structure in the trajectory. These anatomic principles are key to understanding the rationale for making decisions in the management of liver trauma.

Hemodynamically Normal Patient with Blunt Injury

The Western Trauma Association has recently proposed an algorithm for the management of nonoperative blunt hepatic trauma (Fig. 33-5).[12] As noted earlier, most blunt hepatic trauma causes venous injuries that are low pressure (3–5 cm H_2O); therefore, hemorrhage usually stops once a clot forms on the area of disruption. Such an injury can be managed nonoperatively, which leads to lower transfusion requirements, decreased abdominal infections, and decreased hospital lengths of stay. Hurtuk et al[13] found no effect on mortality after solid organ injury with increased use of nonoperative management. With grade III and IV hepatic injuries, Coimbra et al[14] documented a reduced mortality with nonoperative management. Richardson[15] has recommended that hemodynamically stable patients who have received less than 4 units of blood can be safely managed nonoperatively. Unlike the spleen, which can be easily removed, liver-related bleeding can be made worse by operative intervention.[16]

A study by Tinkoff et al[17] showed that the need for operative intervention increases with grade of hepatic injury, but grade alone is not an indication for operation. These data from the National Trauma Data Bank demonstrated that 73% of grade IV and 63% of grade V liver injuries could be successfully managed nonoperatively.[17] A more recent retrospective study by the research consortium of New England Centers for Trauma examined nonoperative management of only grade IV and V injuries and reported an 8.8% failure rate for nonoperative management. Risk factors for failure were a presenting systolic blood pressure of less than 100 mm Hg and the presence of associated abdominal injuries.[18] Importantly, they

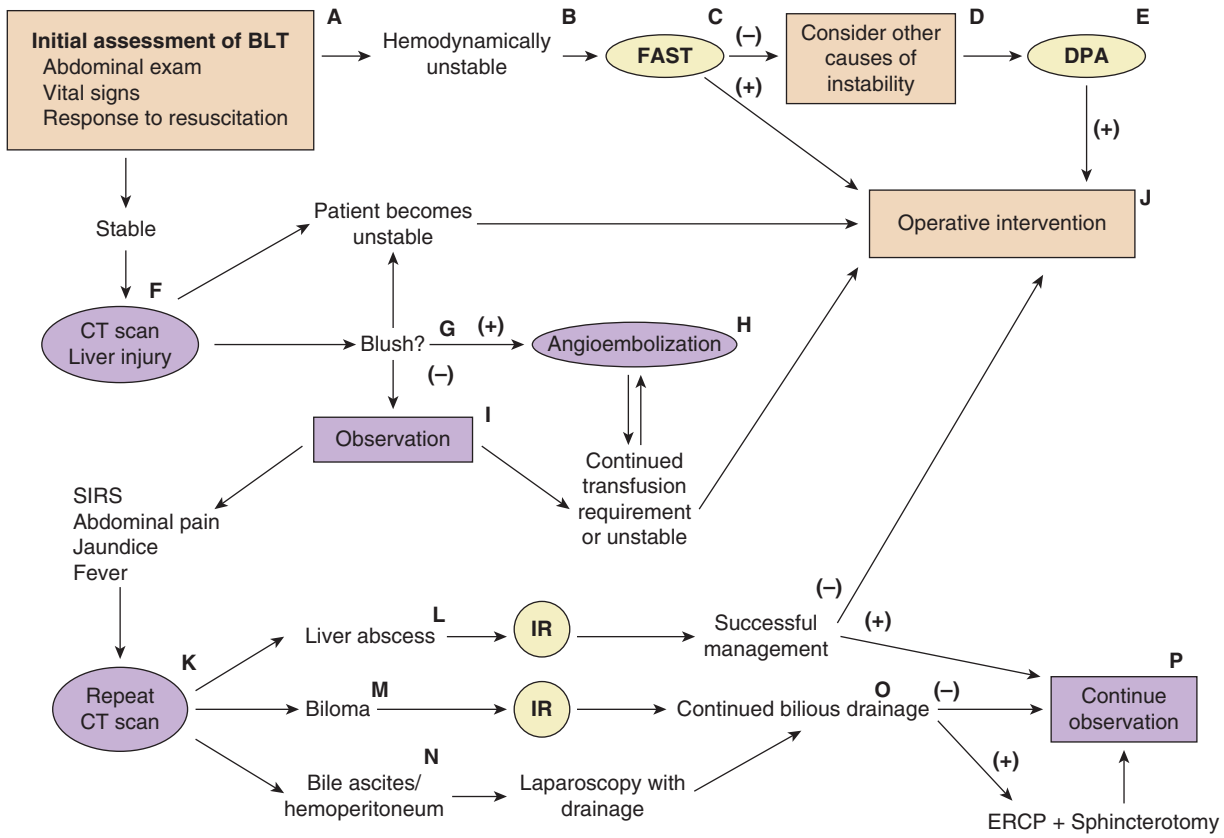


FIGURE 33-5 Western Trauma Association algorithm for nonoperative management of adult blunt hepatic trauma. BLT, blunt liver trauma; CT, computed tomography; DPA, diagnostic peritoneal aspirate; ERCP, endoscopic retrograde cholangiopancreatography; FAST, focused abdominal sonogram for trauma; IR, interventional radiology; SIRS, systemic inflammatory response syndrome. (Reproduced with permission from Kozar RA, Moore FA, Moore EE, et al. Western Trauma Association critical decisions in trauma: nonoperative management of adult blunt hepatic trauma. *J Trauma.* 2009;67(6):1144.)

found no increase in mortality in patients who failed initial nonoperative management.

In contrast, Polanco et al[19] used the National Trauma Data Bank data to examine patients with liver Abbreviated Injury Scale scores of 4 or higher and asked the question, "Has the pendulum swung too far?" In this study, there was a rather alarming trend in the percentage of hypotensive patients who underwent attempted nonoperative management.[19] Although only 7% of the patients failed, those patients had a statistically significant increase in mortality. Age, sex, Injury Severity Score, Glasgow Coma Scale, and hypotension were predictors of unsuccessful nonoperative management. An additional factor to consider in review of nonoperative management of high-grade hepatic injuries is the type of resuscitation strategy that was implemented. As shown by Shrestha et al,[20] damage control resuscitation strategies increased successful nonoperative management and decreased mortality in a retrospective review of over 200 patients with grade IV and V hepatic injuries who received blood products. In addition to more nonoperative management of hepatic injuries, there was no increase in liver-related complications.

The extent of hemoperitoneum and the presence of contrast extravasation and/or a pseudoaneurysm are not contraindications for nonoperative management; however, patients with these entities are at higher risk for failure. A patient with a CT finding of a contrast blush or extravasation (active hemorrhage) may benefit from catheter-directed intravascular angioembolization. Misselbeck et al[21] reviewed their 8-year experience with hepatic angioembolization and found that hemodynamically stable patients with contrast extravasation on a CT scan were 20 times more likely to require embolization than those without extravasation. Sivrikoz et al[22] have shown that angiography in severe blunt hepatic injury is associated with improved survival in both operatively and nonoperatively managed patients; however, patients managed with angiography did have more complications. Finally, Letoublon et al[23] employed angioembolization for either contrast extravasation seen on a CT scan in hemodynamically stable patients or as an adjunctive technique to control arterial bleeding despite laparotomy. They report a complication rate of 70% in their retrospective review.[23]

Complications of Nonoperative Management of Blunt Hepatic Injury

Most patients undergoing nonoperative management of blunt hepatic injuries heal without complication.[24] In a retrospective multi-institutional study of 553 patients with grade III to V hepatic injury, 12.6% developed hepatic complications. Significant coagulopathy and grade V injury were found to be predictors of a complication.[25]

BILE LEAKS

Bilomas from a bile leak have occurred in 3% to 36% of patients managed nonoperatively.[26] Either a hepatobiliary hydroxyl-iminodiacetic acid (HIDA) radioisotope scan or magnetic resonance cholangiopancreatography has been used to localize bile leaks.[27] Hyperbilirubinemia, abdominal distention, and intolerance to feeding may all indicate a bile leak. Percutaneous drainage directed by ultrasound or CT is the primary treatment used for symptomatic biliary leaks.

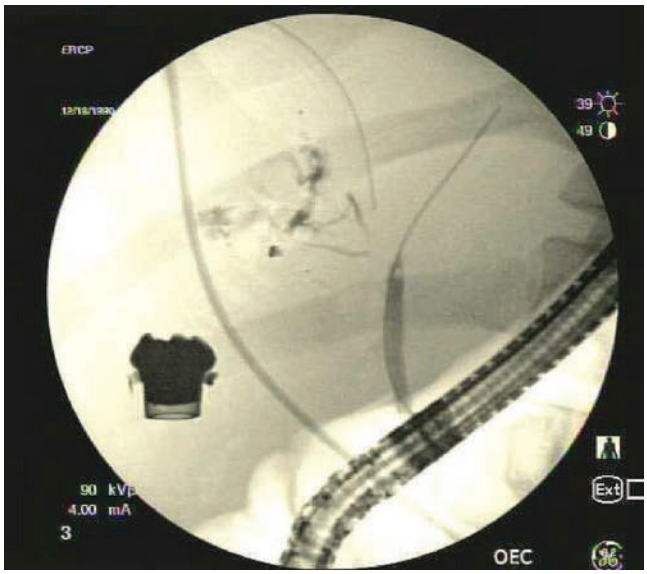


FIGURE 33-6 Endoscopic retrograde cholangiogram demonstrating leak and placement of stent with large bullet fragment visible.

The majority of bile leaks, however, occur after operative management. Anand et al[28] found that 8% of high-grade hepatic injuries managed nonoperatively developed a bile leak. For patients presenting with bile peritonitis and/or with large leaks not responsive to percutaneous drainage alone, the addition of endoscopic retrograde cholangiography (ERC) (Fig. 33-6) with sphincterotomy and biliary stent placement can be effective. Griffen et al[29] have reported success with a combined laparoscopic and ERC approach. They described patients with biliary ascites taken to the operating room for laparoscopic bile drainage and drain placement near the site of injury with postoperative ERC and bile duct stenting. They reported no septic complications and healing of the substantial biliary leaks.[29] In their prospective observational study, Hommes et al[30] classified bile leaks as minor or major, with major defined as greater than 400 mL/d or leaks lasting greater than 14 days. Patients with major leaks underwent ERC and stenting, whereas minor bile leaks nearly always resolved without ERC or other decompressive maneuvers.[30]

PERIHEPATIC ABSCESS

Although rare after nonoperative management, a patient with a perihepatic abscess may exhibit signs of sepsis, abnormal liver function tests, abdominal pain, or food intolerance (Fig. 33-7). Similar to biliary collections, an abscess can often be managed by CT-guided drainage catheters; however, if the patient fails to improve with drainage and antibiotics, wide surgical drainage should be performed. Operative therapy may involve merely incision and adequate drainage of the cavity, or it may involve extensive debridement of necrotic hepatic parenchyma.

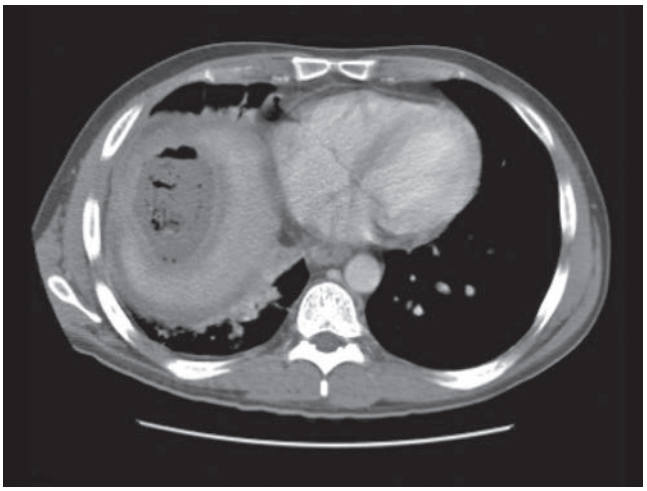


FIGURE 33-7 Computed tomography scan demonstrating a hepatic abscess from a patient who had sustained a blunt hepatic injury.

HEMORRHAGE

Delayed hemorrhage after nonoperative management is rare. Kozar et al[25] reported a 13% overall incidence of liver-related complications in patients managed nonoperatively after grade III to V injuries, with bleeding accounting for 8% of the complications. Bleeding occurred almost equally between early (<24 hours) and late (>24 hours) time periods after injury. Late bleeds occurred only in patients with grade IV and V injuries, the majority of which were managed with angioembolization.[25]

DEVASCULARIZATION AND HEPATIC NECROSIS

Disruption of vascular inflow to a hepatic segment following trauma or after angioembolization can lead to ischemia and subsequent necrosis of that segment of the liver. The consequences of necrosis may include elevation of liver transaminases, coagulopathy, bile leaks, abdominal pain, feeding intolerance, respiratory compromise, renal failure, and sepsis. Devascularization can be differentiated from intraparenchymal hemorrhage when follow-up CT scans reveal segments of liver that remain hypoperfused or have foci of air within the devascularized area.[31]

HEMOBILIA

Hemobilia can occur after blunt hepatic injury when abnormal connections between the vasculature and biliary tree form. The classic triad of right upper quadrant pain, jaundice, and upper gastrointestinal bleeding is present in most, but not all, trauma patients with hemobilia.[32] Selective arterial embolization is the initial treatment of choice with a substantial rate of success and a low incidence of serious complications.[33]

SYSTEMIC INFLAMMATORY RESPONSE

Patients with inadequately drained bile collections or hematomas may be susceptible to the development of the systemic inflammatory response syndrome. Franklin et al[34] and Letoublon et al[35] have recommended laparoscopic evacuation of undrained bile or hemoperitoneum at postinjury days 3 to 5. Early evacuation of these collections can markedly decrease this inflammatory response.

UNUSUAL COMPLICATIONS

A subcapsular hematoma may increase hepatic intraparenchymal pressure high enough to cause segmental portal hypertension and hepatofugal flow. This "compartment syndrome of the liver" was described in a patient managed nonoperatively who had a decreasing hematocrit and abnormal liver function tests. These findings prompted an angiographic examination documenting hepatofugal flow in the right portal vein. After operative drainage of the tense hematoma, the patient did well with return of hepatopetal flow and viability of the right lobe.[36] This type of compressive complication has also caused Budd-Chiari syndrome when the hematoma led to compression of the inferior vena cava or hepatic veins.[37]

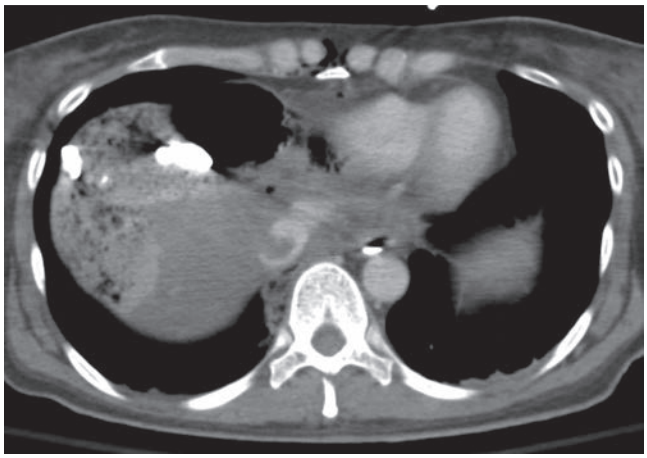
With the frequent use of CT scanning, previously unidentified complications of a hepatic injury may be seen. Fig. 33-8 shows a series of CT images of a patient who underwent operative control of parenchymal bleeding with packing followed immediately by CT scanning. Thrombosis of the retrohepatic vena cava was seen, with clot extending up to the right atrium. Approximately 24 hours after packs were removed, therapeutic anticoagulation was started.

Follow-Up CT Scanning After Blunt Hepatic Injury

Definitive data on the value of follow-up CT scanning for blunt hepatic injury are not available. Some published reports suggest utility of postobservation CT scans for patients with more severe (grade III–V) injuries. Cuff et al[38] reported that of the 31 patients who had follow-up CT scans 3 to 8 days after injury, only three scans changed management. Additionally, the three scans that affected management were obtained due to a change in clinical picture and were not merely routine.[38] Cox et al[39] concluded from their follow-up of 530 patients, including 89 with grade IV or V injuries, that follow-up CT scans are not indicated as part of the nonoperative management of blunt hepatic injuries. Unfortunately, the time to onset of biliary complications can range from days to weeks, making a recommendation difficult.[12] Currently, follow-up CT scans are generally indicated only for patients who develop signs or symptoms suggestive of a hepatic abnormality.

Length of Observation, Prophylaxis for Venous Thromboembolism, and Resumption of Activity

Bed rest and prolonged periods of in-hospital observation are no longer advocated after nonoperative management of hepatic trauma. In their retrospective study of 591 patients with blunt hepatic injuries, Parks et al[40] concluded that the



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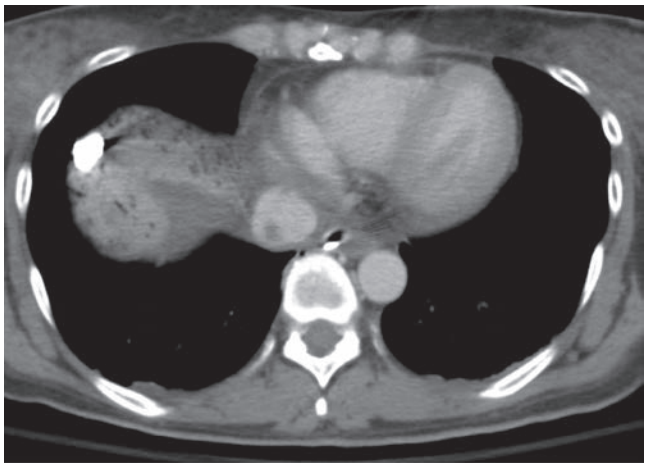

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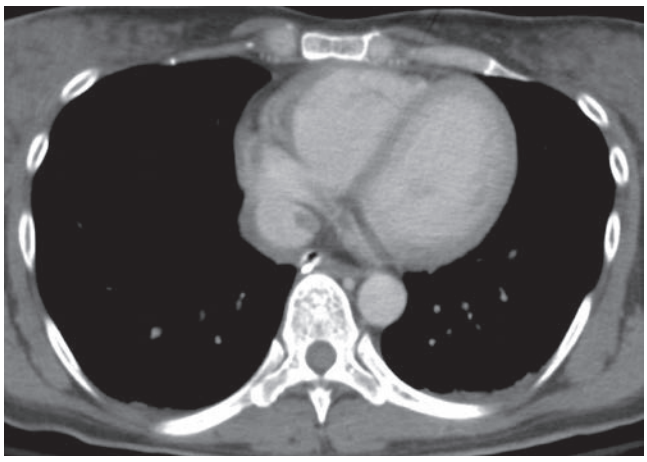

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FIGURE 33-8 Computed tomography scans from a patient who underwent perihepatic packing then immediate postoperative scanning. Images reveal a retrohepatic caval thrombus that extends proximally to the atrial caval junction.

length of in-hospital observation should be based solely on clinical criteria. The authors recommended discharge of patients with a normal abdominal examination and stable hemoglobin.[40]

Although data are retrospective and studies are small, existing data suggest that early (defined as ≤48 hours) institution of chemical prophylaxis against thromboembolism is safe.[41,42] Resumption of normal activity also seems safe, but the period of time needed to refrain from high-risk activities such as contact sports remains unclear. Most hepatic injuries resolve by CT in 4 months, but whether this period of time is optimal is unknown. Dulchavsky et al[43] found, in animal studies, that hepatic wound burst strength at 3 weeks was as great or greater than uninjured hepatic parenchyma. This is most likely a result of fibrosis throughout the injured parenchyma and Glisson capsule. This study suggests that activity can be resumed about 1 month after injury, although human studies have not been performed.

Hemodynamically Normal Patient with Penetrating Injury

NONOPERATIVE MANAGEMENT OF PENETRATING INJURY

Peritoneal penetration had traditionally mandated operative exploration; however, selective nonoperative management of stab wounds is common.[44] Reports of successful nonoperative management of gunshot wounds (GSWs) to the liver have also been published. Renz and Feliciano[45] prospectively treated 13 patients with right thoracoabdominal GSWs nonoperatively. The rationale behind this management is that the wounds caused by small-caliber weapons may cause modest injury to diaphragm and liver only, sparing any hollow viscus injury. The authors stressed the importance of serial abdominal exams and contrast CT scanning in their successful nonoperative management of penetrating injury.[45] Other centers concur with this selective nonoperative management.[46,47] Demetriades et al[48] even reported successful nonoperative management of penetrating grade III to IV hepatic injuries that required angioembolization. The criteria for nonoperative management include the following: (1) patient is hemodynamically normal; (2) patient does not have peritoneal signs; and (3) patient is able to cooperate with serial abdominal examinations. Before nonoperative management is decided upon, the patient has a contrast-enhanced CT scan to rule out other abdominal visceral or vascular injury. As reported by Shanmuganathan et al,[49] triple-contrast CT of 86 abdominal GSWs had a sensitivity and specificity of 97% and 98%, respectively, in the detection of organ injury and peritoneal violation. Velmahos et al[50] used a single intravenous dose of contrast in lieu of triple contrast and reported a sensitivity and specificity of 90.5% and 96%, respectively, in diagnosing intra-abdominal organ injuries requiring surgical intervention.

Missed or deliberate nonrepair of small penetrating diaphragmatic injuries may lead to long-term adverse sequelae,

not only of diaphragmatic herniation, but also of possible biliopleural fistula.[51] Late intervention for other missed injury (eg, duodenal injury) may also lead to substantial morbidity.

Operative Management of Patients with Minor Hepatic Injury

The decision to operate on a patient with hepatic injury may be made due to a penetrating mechanism with secondary peritonitis, hemodynamic instability, or evidence of a concomitant organ injury. The incision of choice remains the midline incision from the xiphoid to the pubis with full surgical preparation and draping of the chest, abdomen, and thighs. On opening the peritoneal cavity, initial attention is on the rapid evacuation of old blood and halting ongoing hemorrhage. The peritoneal cavity is cleared of clot using hands or laparotomy pads. When a minor hepatic injury is present, bleeding is managed with packing and pressure applied to the injured area. While packs are in place, inspection of the remainder of the abdomen for other injuries is performed. Nonbleeding injuries should not be probed or otherwise manipulated, as this may cause dislodgement of clot. Small wounds of the hepatic parenchyma with minimal bleeding can be controlled with electrocautery, argon beam coagulation, or topical hemostatic agents. Small to moderate-sized bleeding cavities are first inspected for any obvious bleeding vessels that can be ligated. In 1975, Stone and Lamb[52] first described the technique of packing a tongue of vascularized omentum into the wound in an effort to stop venous bleeding. Wrapping a column of absorbable gelatin sponge with oxidized regenerated cellulose that can be plugged into deeper bleeding cavities is another option.

Operative Management of Patients with Major Liver Injury

In 2009, Lucas and Ledgerwood[53] reported that a surgical resident would perform a hemostatic technique on the liver only 1.2 times during his or her training. With the increasing utilization of nonoperative management, gaining and maintaining the necessary skills to control major hepatic bleeding is a challenge. The Western Trauma Association has published a useful algorithm for operative management of adult blunt hepatic trauma (Fig. 33-9).[54]

INITIAL MANAGEMENT

Once the peritoneum is entered in these patients, the natural tamponade provided by hemoperitoneum is released, and

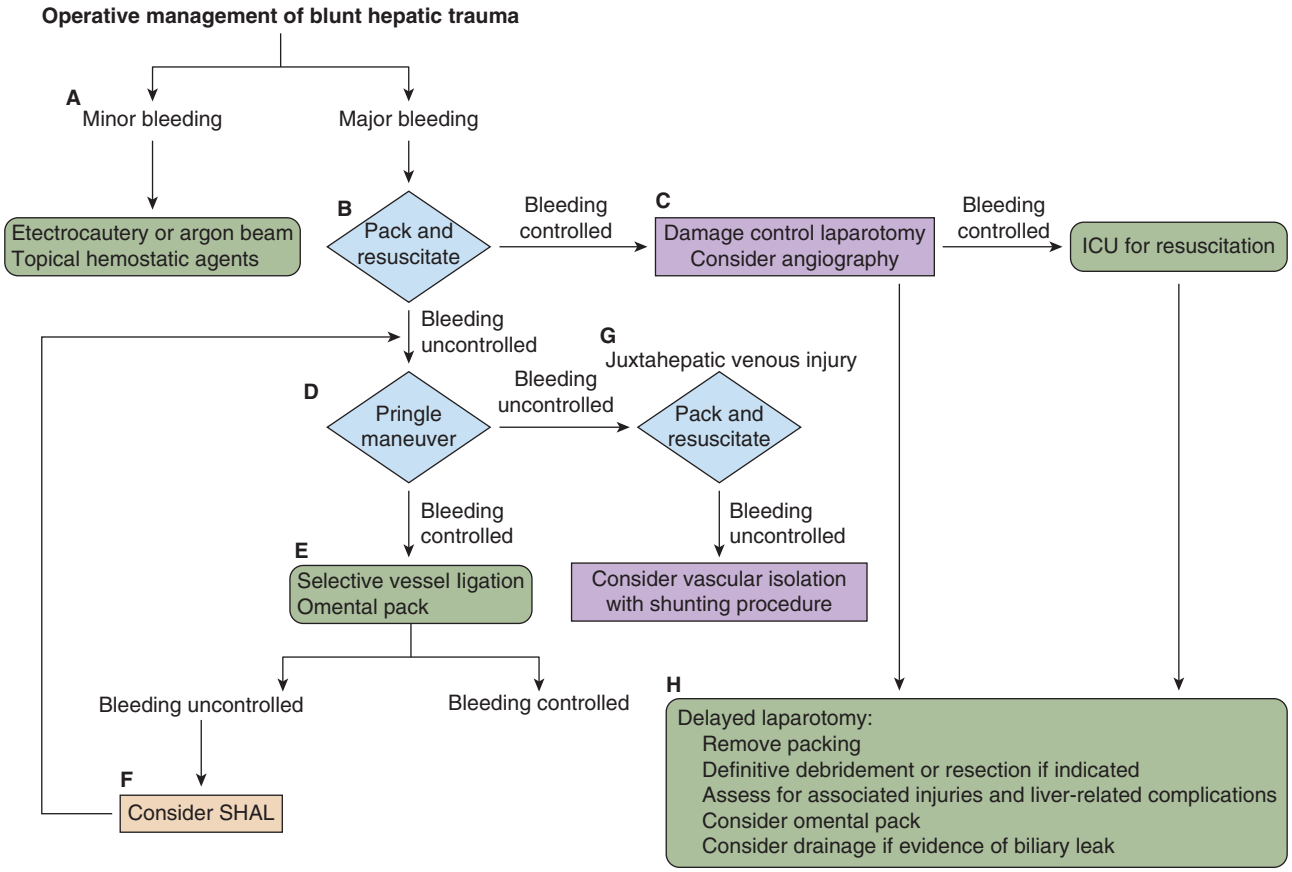

FIGURE 33-9 Western Trauma Association algorithm for operative management of adult blunt hepatic trauma. ICU, intensive care unit; SHAL, selective hepatic arterial ligation. (Reproduced with permission from Kozar RA, Feliciano DV, Moore EE, et al. Western Trauma Association critical decisions in trauma: operative management of adult hepatic trauma. *J Trauma*. 2011;71(1):1-5.)

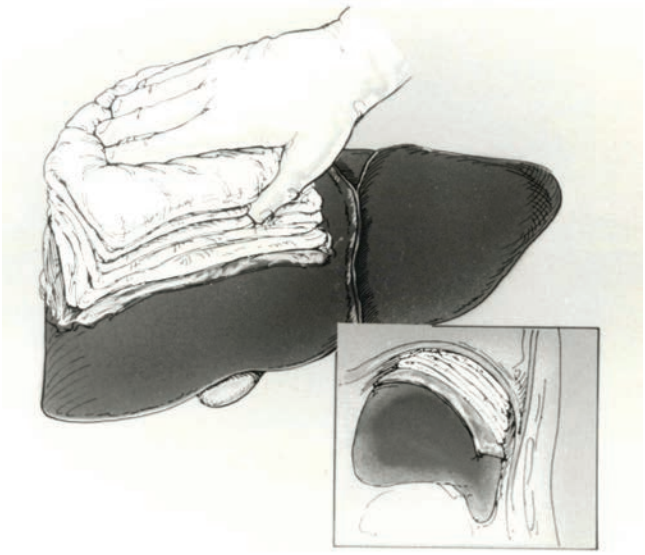

FIGURE 33-10 Manual compression of a hepatic injury. Sagittal view (inset) of packs placed above the liver to assist with compression of hepatic bleeding. (Reproduced with permission from Feliciano DV, Pachter HL. Hepatic trauma revisited. *Curr Probl Surg*. 1989;26:453.)

this may exacerbate bleeding. Manual compression of obvious injury will decrease bleeding (Fig. 33-10) and allow the anesthesia team some time to initiate a massive transfusion protocol and "catch up" with blood loss prior to proceeding. Any resuscitative products should be warmed and coagulopathy corrected, keeping in mind current recommendations for blood products in ratios approaching 1:1:1 (packed red cells to fresh frozen plasma to platelets) or viscoelastic assay-directed resuscitation while minimizing the administration of crystalloids. Once the patient has been adequately resuscitated, a more thorough examination of the peritoneal cavity is completed. If the bleeding source is localized to the liver and bleeding continues after manual compression is released, then the portal triad should be identified and a Pringle maneuver performed (Fig. 33-11) (see Atlas Figure 43).

Much controversy has centered on the ischemic time produced by the use of the Pringle maneuver. Multiple studies have documented that long duration (60 minutes) portal triad occlusion is well tolerated in the absence of cirrhosis.[55,56] In one study describing the operative management of 1000 patients with hepatic trauma, the Pringle maneuver was used routinely for between 30 and 60 minutes without adverse sequelae.[57] Pachter et al[58] managed 81 patients with the assistance of the Pringle maneuver for up to 75 minutes without any apparent morbidity. Given these data, it seems that longer normothermic, or hypothermic, ischemic times can be used without added morbidity. The Pringle maneuver often does not control all bleeding. It will control the inflow bleeding from the hepatic artery and portal vein, but not retrograde bleeding from the inferior vena cava and hepatic veins; therefore, if bleeding persists with the portal triad clamped, injury to the retrohepatic vena cava and/or hepatic veins should be suspected.

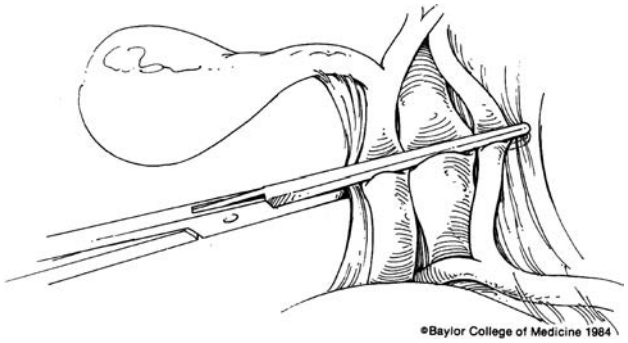

FIGURE 33-11 The Pringle maneuver places a vascular clamp across the common bile duct, portal vein, and common hepatic artery. Any residual hemorrhage is likely the result of bleeding hepatic veins. (Reproduced with permission from Feliciano DV, Pachter HL. Hepatic trauma revisited. *Curr Probl Surg*. 1989;26:453.)

Clinical experience and animal data suggest that resuscitative endovascular balloon occlusion of the aorta (REBOA) is useful for partially controlling bleeding from intra-abdominal visceral and vascular injuries.[59,60] It is becoming increasingly common to place femoral arterial access in patients presenting in shock and, if needed, a REBOA catheter if the patient does not respond to resuscitation (Fig. 33-12). Matsurmura et al[61] demonstrated that quicker femoral access was significantly associated with a decrease in mortality in patients undergoing REBOA placement with occlusion. A new smaller diameter sheath (7F) is now available and reduces the time to balloon inflation as well as frequently obviating the need for repair of the puncture site in the femoral artery.[62]

Hemostatic Maneuvers for Severe Parenchymal Injury

PACKING (SEE ATLAS FIGURE 44)

Perihepatic packing has become the most widely used "bailout" method for damage control of a severe hepatic injury. Packing laparotomy pads around the liver compresses the bleeding wound between the anterior chest wall, diaphragm, and retroperitoneum and stops venous bleeding. Beal[63] reported an 86% survival rate in 35 patients in whom perihepatic packing was used. In order to provide the tamponade necessary for effective packing, it is sometimes necessary to mobilize the liver by taking down the falciform ligament and right and left coronary and triangular ligaments. If, however, there is obvious hematoma in a ligament, this mobilization should be avoided, as it may indicate an injury to the hepatic veins or retrohepatic vena cava and mobilization may lead to rapid exsanguination. The decision to pack should be made early in the operation in order to provide the best chances for patient survival.[64,65] Also, it is important to remember that packing will not control major arterial hemorrhage. The major refinement in the technique of perihepatic packing has been the placement of separate posterior paracaval, lateral, anterior, and posteroinferior packs as described by Baldoni et al.[66]

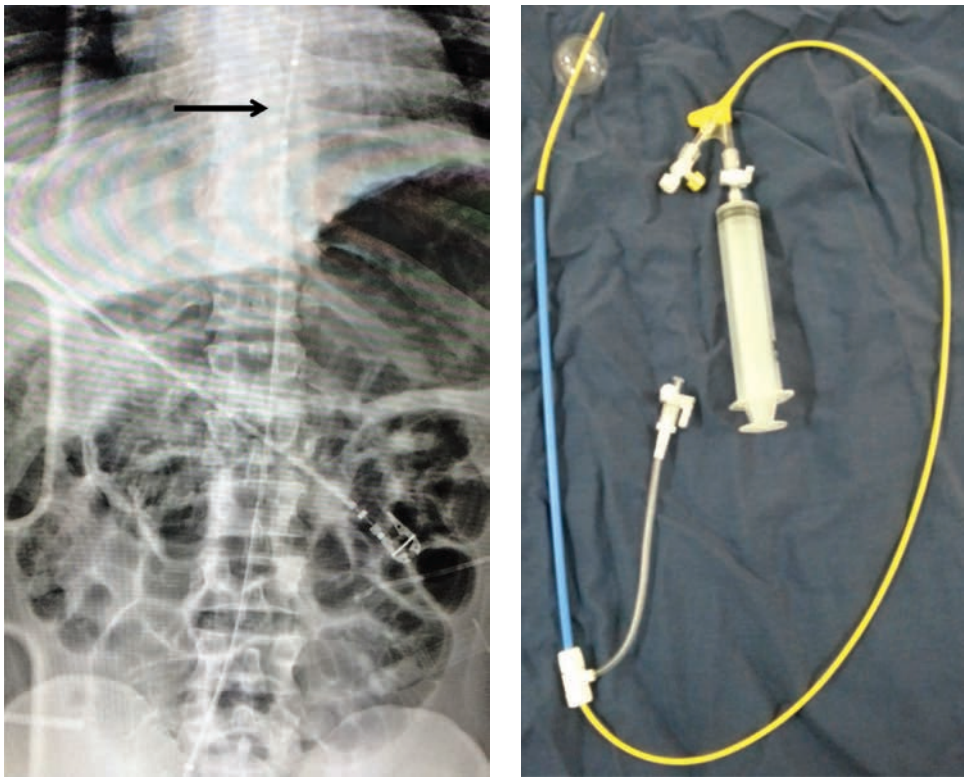

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FIGURE 33-12 (**A**) Plain abdominal radiograph from a patient with a grade V blunt hepatic injury that had a resuscitative endovascular balloon occlusion of the aorta (REBOA) catheter deployed in zone I (arrow) in the emergency room prior to operaexploration. (**B**) Photo of a REBOA catheter.

One of the challenges with perihepatic packing is the removal of the pads. Often, the bare liver area that has become hemostatic is now adherent to the packs, and bleeding will occur with removal. Different solutions to this problem have been described, from wetting the gauze with saline on removal to more innovative techniques. Feliciano and Pachter[67] suggest placing a nonadherent plastic drape directly on top of the hepatic surface, followed by laparotomy pads above this plastic interface, thus preventing adherence of the packs to the parenchyma.

Abdominal hypertension can be minimized in packed patients by leaving the fascia and skin edges open over the inferior two-thirds of the midline incision and placing a temporary closure device over the open abdomen, remembering that abdominal compartment syndrome can still arise.

The timing of pack removal continues to be the subject of debate. With damage control resuscitation, correction of perioperative hypothermia, acidosis, and coagulopathy can almost always be accomplished within 24 hours of packing. Intraabdominal sepsis is a theoretical risk of prolonged packing, but an obvious risk of continued bleeding exists with premature removal. Krige et al[68] found that packs that remained for more than 3 days had an 83% incidence of developing perihepatic sepsis, whereas those left less than 3 days had a 27% chance of sepsis. Caruso et al[64] advocate the removal of packs at 36 to 72 hours, because they have experienced a higher rate of repacking for recurrent hemorrhage in the group of patients who had their packs removed earlier. Nicol et al[69] reported a significantly higher repacking rate in hemodynamically stable patients whose packs were removed at 24 hours, compared to patients whose packs were removed after 48 hours.

DIRECT SUTURE (SEE ATLAS FIGURE 45)

Grade III and IV hepatic lacerations often do not respond to the superficial procedures described for minor injury control. One of the oldest reported techniques to control deep parenchymal bleeding is direct suturing of the tissue with a large, blunt-tipped 0 chromic suture. The sutures can be continuous, or if a deeper laceration is encountered, a mattress configuration is useful. This technique is most appropriate for lacerations less than 3 cm in depth. It is best to avoid deep suture placement as blind passage of these large blunt needles may injure bile ducts and vascular structures, leading to possible intrahepatic hematomas or hemobilia. Of note, hepatic necrosis can occur with the use of liver sutures, especially if tied too tightly.

FINGER FRACTURE OR HEPATOTOMY (SEE ATLAS FIGURE 46)

More severe parenchymal lacerations may involve larger branches of the hepatic artery or portal vein and will not

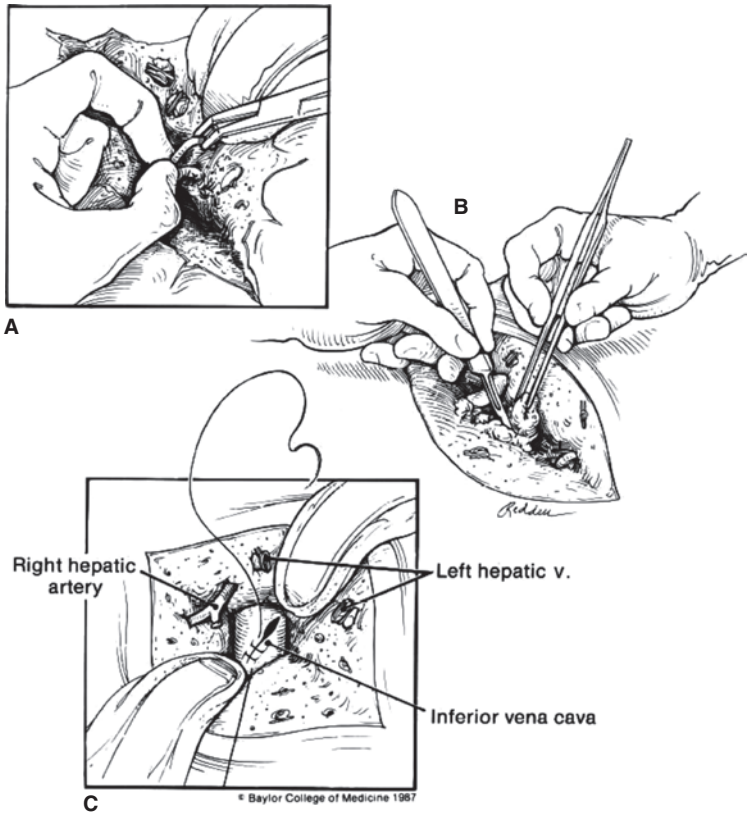


FIGURE 33-13 Hepatotomy with selective ligation is an important technique for controlling hemorrhage from deep (typically penetrating) lacerations. This technique includes finger fracture to extend the length and depth of the wound (**A**), clipping of visible ducts and vessels after fracture through the hepatic parenchyma (**B**), utilization of sponge stick and knife to fracture the parenchyma (**C**), and repair of any injuries to major veins and/or arteries. (Reproduced with permission from Feliciano DV, Pachter HL. Hepatic trauma revisited. *Curr Probl Surg*. 1989;26:453.)

respond to packing or parenchymal suturing. In these cases, finger fracture has been described (Fig. 33-13). The utilization of this technique involves careful extension of the laceration using finger fracture or clamping of the hepatic parenchyma as dissection proceeds through the liver. The fracture continues until bleeding vessels are identified and controlled with clips, ligation, or direct repair.[70] Ordoñez et al[71] published a series of penetrating hepatic injuries with 20.8% mortality for grade IV injuries and 70% for grade V. The authors concluded that intraparenchymal exploration and selective vessel ligation are useful in patients who fail packing and the Pringle maneuver. More recently, the use of stapling devices has been advocated.[72]

OMENTAL PACKING (SEE ATLAS FIGURE 47)

Omental packing can be used as the primary technique or in conjunction with other methods of hemorrhage control. Omental packing can fill a hepatic laceration or hepatotomy site with viable tissue that also is a source of macrophage activity. Stone and Lamb's original work[52] was reinforced by Fabian and Stone[73] when they managed to stop venous hemorrhage in severe parenchymal lacerations in 95% of patients with an 8% mortality. The greater omentum is first mobilized from the transverse mesocolon in the avascular plane and then freed from the greater curvature of the stomach, preserving a gastroepiploic (usually the right) vascular pedicle. The tongue of omentum is then placed into the injury defect (Fig. 33-14). It is then fixated in place by placing 0 chromic sutures from one side of the laceration or hepatotomy site to the other over the omentum and tying the knots firmly.

TRACT OF A PENETRATING WOUND (SEE ATLAS FIGURE 48)

Penetrating tracts through the hepatic parenchyma may be of great depth and length, making visualization of the entire injury impossible. Management of these injuries has included packing of the tract with an omental pedicle or a self-assembled device such as the rolled cellulose-covered gelatin sponge. Poggetti et al[74] advocate the use of balloon tamponade of the tract using a Penrose drain placed over a red Robinson catheter and tied on both ends. The "balloon" over Robinson catheter is then placed into the tract and inflated with a contrast agent (Fig. 33-15). If successful tamponade is achieved, the balloon is left in the abdomen and removed 24 to 48 hours later at a second laparotomy. A similar technique using a Foley catheter has been described after inflation of the balloon to obtain tamponade. If there is continued active bleeding, the catheter is moved back or forward and inflated again.

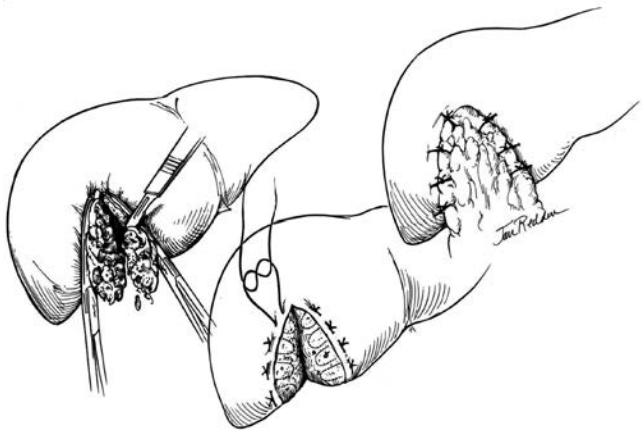


FIGURE 33-14 Major hepatic laceration controlled with clamps and sharply debrided. The edges of the laceration are oversewn with heavy silk or chromic suture to obtain some degree of hemostasis. A well-vascularized pedicle of omentum is mobilized and secured to the denuded section of liver. (Reproduced with permission from Feliciano DV, Pachter HL. Hepatic trauma revisited. *Curr Probl Surg*. 1989;26:453.)

If bleeding continues through the catheter but not out of the tract, the balloon is proximal to the bleeder and needs to be repositioned deeper. If the bleeding continues from the tract orifice, then the balloon must be repositioned further out of the tract.[75] Once the catheter is positioned, drains are placed in the area and brought out through the skin along with the end of the catheter. The Foley can be removed after deflation produces no further signs of bleeding or at the time of the next planned reexploration. Sengstaken-Blakemore tubes have been used in these situations as well.[76]

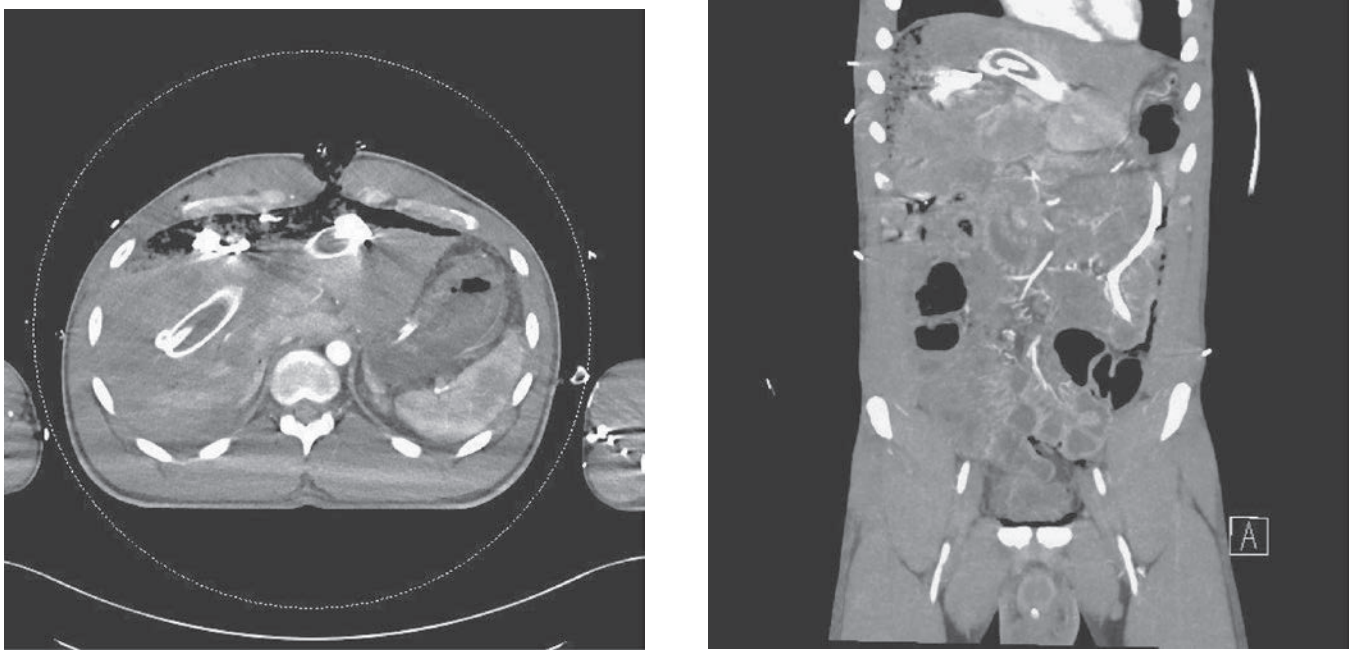
When a hepatotomy with selective vascular ligation, insertion of an omental pack, or balloon catheter tamponade is ineffective, angioembolization is another option for these lesions, if the patient can be stabilized for the procedure.

RESECTION

Anatomic resections for severe hepatic trauma were often performed in the late 1960s and early 1970s. In most series, mortality was high[77]; thus, resection fell out of favor until recently, when several groups reported improved outcomes. Polanco et al[78] reported on their experience of patients who underwent hepatic resection during their initial operation, with a morbidity of 30% and a mortality of 17.8%. Based on these data, the authors recommended hepatic resection in patients with massive bleeding related to a hepatic venous injury that must be repaired directly, in patients with massive destruction of hepatic tissue, and in patients with a major bile leak from a proximal main intrahepatic bile duct. Of the 56 patients undergoing resection, 42 had some type of resection performed during the initial surgical procedure.[78]

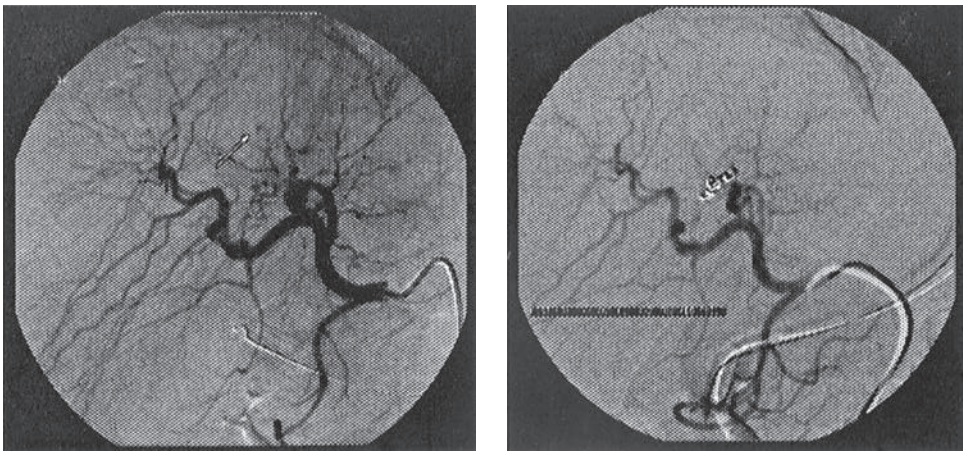
SELECTIVE HEPATIC ARTERY LIGATION OR ANGIOEMBOLIZATION

Although hepatic artery ligation can be a useful maneuver, it has largely been supplanted by angioembolization. In the



A B

FIGURE 33-15 Computed tomography of a patient with a transhepatic gunshot wound trajectory managed with a handmade balloon fashioned from a Penrose drain to control hemorrhage. (**A**) Axial views show the patient with an open abdomen and packing anterior to the liver. (**B**) Coronal views.



A B

FIGURE 33-16 (**A**) Hepatic pseudoaneurysm. (**B**) Coiled hepatic pseudoaneurysm.

operating room, if a patient has a noticeable decrease in bleeding after the right or left hepatic artery is clamped, hepatic artery ligation can be considered. This would usually be after attempts at direct vessel ligation or control, and bleeding cannot be controlled sufficiently to permit time for angioembolization. When the portal vein remains patent, the chance for severe hepatic dysfunction after hepatic artery ligation is minimal[79,80]; however, with patients in traumatic shock, hepatic artery ligation may lead to further ischemia with later development of necrosis or sepsis.[81]

Currently, most centers are advocating a multimodality approach to hepatic arterial bleeding, with a special emphasis on angioembolization. In 1984, Sclafani et al[82] reported successful selective arterial embolization to control bleeding from a severely injured liver after packing. Angiography has since become an important step in the management algorithm for a severe hepatic injury (Fig. 33-16). In a recent systematic review of angiography for hepatic trauma, angioembolization was successful in 77% to 100% of cases. The most common indication for angioembolization was a contrast blush seen on CT, followed by failure of nonoperative therapy and continued bleeding after damage control laparotomy.[83] Although this review was unable to draw a definitive conclusion regarding mortality, Matsumoto et al,[84] using data from the National Trauma Data Bank, reported that the use of postoperative hepatic angiography was associated with decreased mortality.

HEPATIC TRANSPLANTATION

Hepatic transplantation after trauma has been successfully reported. The patient must have an overall excellent chance of survival with minimal other injuries, especially in the abdomen or to the brain. In a review of over 1500 liver transplants by Heuer et al,[85] six were done following trauma with a 33% survival rate. In a 2013 report from Kaltenborn et al,[86] 12 patients undergoing liver transplantation for trauma had an overall mortality rate of 42%. In the survivors, retransplantation was necessary in 25%.

Juxtahepatic Liver Injuries

In an article by Buckman et al,[87] the authors divide injuries to the retrohepatic vena cava or hepatic veins into two categories: intraparenchymal and extraparenchymal. *Intraparenchymal* injuries include those to hepatic veins within the body of the liver; therefore, bleeding occurs through the injured liver. *Extraparenchymal* injuries include those to the hepatic veins outside the liver and to the retrohepatic vena cava. Life-threatening bleeding from these injuries occurs if the supporting structures, mainly the suspensory ligaments and diaphragm, are disrupted. Thus, inadvertent exposure of a major venous injury will release the tamponade and result in free bleeding and possible exsanguination. As Buckman et al[87] outlined, there are three main strategies to deal with these mortal injuries. The first is to directly repair the venous injury with or without vascular isolation. The second is to perform a formal lobectomy. The third is by using a strategy of tamponade and containment of the venous bleeding.[87] Once a juxtahepatic injury is identified, manual compression should be maintained on the liver while plans for repair are being carried out. These should include activation of a massive transfusion protocol, active warming, a call for additional help to the operating room, and on rare occasions, a call to a perfusion team if the need for venovenous bypass is likely.

DIRECT VENOUS REPAIR

Direct venous repair without shunting has been advocated by Pachter and Feliciano.[88] They described occlusion of the portal triad for a significant time, mobilization of the liver with medial rotation, and efficient finger fracture to the site of injury. With these methods, they reported a 43% survival rate (6 of 14 patients). Chen et al[89] have published similar results with a 50% survival rate.

Various shunting maneuvers of the retrohepatic cava have been described. Schrock et al[90] first introduced the atriocaval shunt in 1968 (Fig. 33-17) (see Atlas Figure 49). The goal is to shunt all venous blood from the infrahepatic vena cava

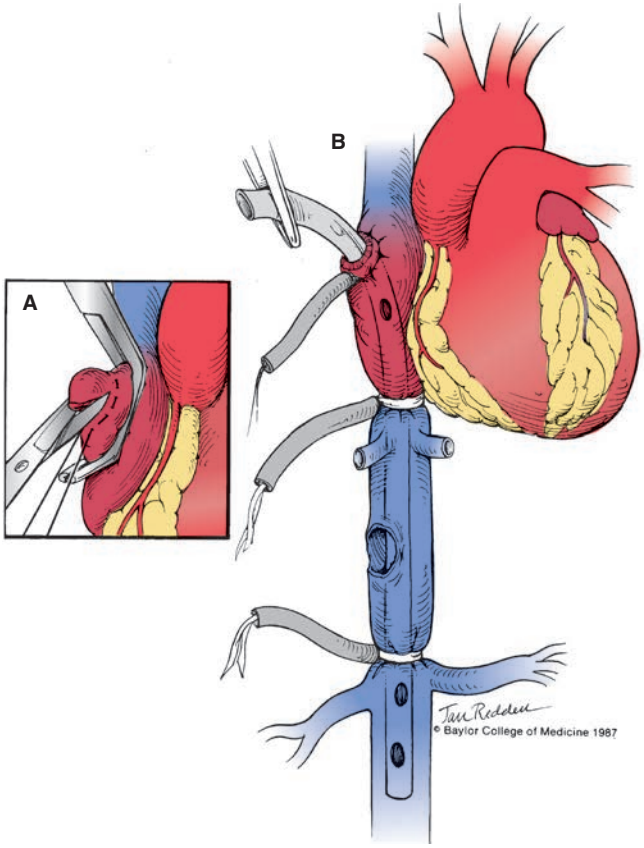


FIGURE 33-17 (**A**) A hole is cut in the right atrial appendage above a 2-0 silk pursestring suture. A Satinsky clamp maintains vascular control. (**B**) Final position of No. 36 chest tube acting as an atriocaval shunt. Note the extra hole cut in the chest tube at the level of the right atrium. All holes in the chest tube are outside the umbilical tapes, thereby forcing blood from the lower half of the body and the kidneys through the shunt. (Reproduced with permission from Feliciano DV, Pachter HL. Hepatic trauma revisited. *Curr Probl Surg*. 1989;26:499.)

thorough the shunt in the retrohepatic vena cava and into the right atrium.[90] Unfortunately, of the approximately 200 cases published using atriocaval shunting, at best 10% to 30% of patients have survived their injury.[15] For the Shrock atriocaval shunt to be performed in a timely manner, all required equipment must be ready, and the addition of a median sternotomy is necessary. Shunting a patient will not be successful if the patient has arrested, had major blood loss, or has become coagulopathic.

Additional shunting techniques exist. In 1977, Pilcher et al[91] reported on a balloon shunt introduced through the saphenofemoral junction. This method has had some anecdotal success and avoids an emergent median sternotomy without destruction of the surrounding ligamentous tamponade.[92] A 1988 multi-institutional trial, however, did not show any survival benefit of the balloon shunt versus the atriocaval shunt.[93]

Total vascular occlusion has been successfully employed in a small number of patients.[94,95] Vascular clamps are placed on the porta hepatis, suprarenal inferior vena cava, and suprahepatic inferior vena cava usually via a sternotomy. If clamping is tolerated, a direct vessel repair can be accomplished; however, hypovolemic patients will not tolerate total vascular occlusion. If this technique is to be used, it is best performed at the time of a delayed laparotomy for those patients who responded to initial packing.

Venovenous bypass has been used successfully in some institutions as well[96,97] (see Atlas Figure 50). Again, this method requires considerable planning and equipment but obviates the hemodynamic instability of occlusion of the inferior vena cava. It allows for active rewarming of the patient and may be a better option for hemodynamically unstable patients who do not respond to packing. In general, direct approaches to vein repair are difficult and can result in uncontrollable hemorrhage.

ANATOMIC RESECTION

Anatomic resection of the overlying injured hepatic lobe was described as a way to access injuries to the retrohepatic vena cava in the past. In certain circumstances, when the dissection has already been done by the injury itself, further resectional debridement for exposure may still be indicated.

ADJUNCTIVE TECHNIQUES

In a swine model of retrohepatic caval injury, Reynolds et al[98] used a Coda balloon to gain rapid control of the suprahepatic inferior vena cava. The resuscitative balloon occlusion of the inferior vena cava (REBOVC) reduced blood loss and decreased time to death.[98] The Coda balloon, however, is not intended for use in veins. There is a balloon catheter specifically designed for the venous system that was developed to provide temporary control to the superior vena cava due to hemorrhage occurring during extraction of cardiac leads. The system has a low-pressure compliant balloon that is 8 cm in length and covers the length of the superior vena cava.[99] Although there are no reports using this balloon for trauma, it seems suited to provide rapid control of the suprahepatic inferior vena cava.

Reports of using endoluminal fenestrated stent grafts in juxtahepatic caval injuries have been published.[100] These grafts have been placed both after damage control laparotomy and prior to laparotomy when the lesion is seen on CT.[101,102] Hommes et al[103] report the survival of a patient with intraoperative placement of an endovascular stent graft into the inferior vena cava for a juxtahepatic caval injury with perihepatic packing. Although these stenting techniques are not yet the norm, increased use may occur as technologies advance.

TAMPONADE WITH CONTAINMENT

At this time, it seems that the most successful method of managing injuries to the retrohepatic vena cava or hepatic veins is by using packs to tamponade the bleeding. Direct repair of damaged veins and use of the atriocaval shunt have had few survivors, and these have primarily been in the hands of experienced hepatobiliary surgeons in somewhat stable patients. In Memphis, the mortality of patients with juxtahepatic venous injuries treated with omental packing was

20.5%.[104] Cue et al[105] described four patients with injuries to the retrohepatic vena cava, hepatic veins, or both, who underwent initial packing. On planned reexploration and unpacking, the injuries were repaired, and three of the four patients survived.[105]

Drains After Hepatic Repair

In 1985, a review of 161 significant hepatic injuries demonstrated that the injury grade, blood loss, shock, and associated injuries were similar in patients who underwent postoperative closed-suction drainage and those who did not.[106] The study concluded that drainage should be done only in injuries with obvious bile leaks noted at the time of laparotomy. A more recent Cochrane review of elective (nontrauma) hepatic resections also concluded that there were no data to support routine postoperative drainage.[107] If a postoperative hematoma, biloma, or perihepatic abscess develops, almost all can be dealt with by percutaneous drain placement using radiographic guidance.

Complications of Operative Management

BLEEDING

Most reviews quote a 2% to 7% rate of postoperative bleeding.[67,93] Decreases in serial hematocrits, increasing abdominal distention, and episodes of hypotension or tachycardia signal continued bleeding. Hemodynamically unstable patients need a reoperation with reexploration of previously packed areas that have failed to stop bleeding. Kutcher et al[108] have recently examined the sensitivity and specificity of early postoperative CT scanning after laparotomy for hepatic trauma and concluded that it identifies clinically relevant ongoing bleeding and is sufficiently sensitive and specific to guide triage to angiography.

ABDOMINAL COMPARTMENT SYNDROME

As noted, an abdominal compartment syndrome may develop with perihepatic packing. Packs should be carefully placed around the liver, taking care to tamponade the bleeding, but not create excessive compression on the retrohepatic inferior vena cava. Crystalloid infusions should be minimized and bladder pressures serially measured in patients with ongoing resuscitation needs in the postoperative period.

HEMOBILIA (SEE PRIOR DISCUSSION AFTER NONOPERATIVE MANAGEMENT)

In a patient who develops significant upper gastrointestinal bleeding following repair of the injured liver, hemobilia should be considered as an etiology. As previously noted, the common signs and symptoms of hemobilia include jaundice, right upper quadrant pain, and a decrease in hematocrit. These findings are common in many patients after operation for severe hepatic trauma; therefore, making the diagnosis of hemobilia, with its reported 3% incidence,[109] can be difficult. The presentation may be days to weeks after operation. Classically, blood is seen emanating from the ampulla of Vater on upper gastrointestinal endoscopy, although bleeding may not always be present at the time the procedure is performed. Angiography will frequently identify the arterial bleeding and allow for embolization of the damaged vessel.[110,111] Operative debridement and drainage may be necessary if a large cavity has formed or sepsis is apparent, but is rarely necessary.

HYPERBILIRUBINEMIA

Biliovenous fistulae have been described but are quite rare.[112] This entity occurs as the bile in venous blood dissolves in the bloodstream and is carried directly to the right heart. This leads to a dramatic rise in bilirubin with relatively normal liver function tests. Series are small and management strategies have varied, but endoscopic retrograde cholangiopancreatography (ERCP) with stenting has been reported to be curative.[113]

BILE LEAKS

As with hepatic injuries managed nonoperatively, bile leaks are a common complication of operative management. In a study by Bala et al,[114] 40% of patients with high-grade hepatic injuries who required operative intervention developed a biliary complication. In a study by Asensio et al,[115] biliary fistulae (defined as >50 mL of biliary drainage per day for 14 days) were found in 22.5% of similar patients. Although the diagnosis can be confirmed by a HIDA scan, an ERCP affords the opportunity for diagnosis and treatment with sphincterotomy and stenting. A major injury to a left or right bile duct with a continued leak and/or a biliary stricture may require a late reoperation with a Roux-en-Y hepaticodochojejunostomy.

HEPATIC NECROSIS

Major hepatic necrosis can be a complication of the multimodality management of severe hepatic trauma. At the University of Maryland Shock Trauma Center, Dabbs et al[116] found that 29 of 30 patients developed major hepatic necrosis after initial operative intervention and 87% had undergone a damage control laparotomy. Many of the patients then had arterial embolization performed, making their risk of major hepatic necrosis around 70%. A large number of these patients then underwent resection of their necrotic hepatic parenchyma. Both serial debridement and formal lobectomy were performed, but lobectomy was associated with fewer procedures overall and a lower complication rate.[117]

OTHER FISTULAS

A pleurobiliary fistula is a rare complication. Early identification and management can prevent progression to a bronchobiliary fistula, with its potential for serious bile damage to the right lower lobe of the lung. After a penetrating thoracoabdominal injury, the patient may do well initially with resolution of a hemothorax and no evidence of jaundice,

with symptoms presenting later in the postoperative course. Definitive treatment has included pleural and/or abdominal drainage, ERC with sphincterotomy and stenting, or operative intervention.[118] Rothberg et al[119] recommended operative intervention in order to evaluate for significant diaphragmatic injury, hepatic necrosis, or lung necrosis with possible bronchial involvement.

A pleurocaval fistula may result from a penetrating thoracoabdominal wound as well. This fistula may be the source of a life-threatening air embolism.[120] An intraparenchymal arterioportal fistula is associated with initial hemorrhage and subsequent portal hypertension. In one case report in which the authors described a GSW that caused a left hepatic artery to portal vein fistula, the fistula was successfully managed by arterial embolization.[121] Portosystemic venous shunts have also been reported after severe blunt hepatic injuries.[122]

HEPATIC FAILURE

When hepatic failure occurs following severe trauma, options are limited. The Molecular Adsorbent Recirculating System (MARS; Baxter International, Deerfield, IL) is an extracorporeal liver replacement device that can be used to treat acute liver failure, although experience is limited in injured patients. In a study of 27 patients with acute liver failure, five of whom had hepatic trauma, survival was 60% in the trauma patients with recovery of both liver and renal function.[123]

INJURY TO EXTRAHEPATIC BILIARY DUCTS

Extrahepatic biliary and portal triad injuries make up only about 0.07% to 0.21% of all trauma admissions at Level I trauma centers, with roughly 30% to 40% caused by a blunt mechanism. At operation, technical problems include hemorrhage, adjacent organ injury, and small duct size. Injury to this area carries an overall 50% mortality, especially if a vascular injury is present. If injury to both the portal vein and hepatic artery are present, the mortality is 99%.[124,125]

Injury Types and Diagnosis

GALLBLADDER

Injury to the gallbladder accounts for up to 66% of injuries to the extrahepatic biliary tract.[126] Blunt injury often involves avulsion, contusion, or perforation. Patients with either blunt or penetrating rupture may also present with bile peritonitis and right upper quadrant pain. If an isolated injury to the gallbladder is seen on a CT scan, additional intra-abdominal injuries should be suspected (Fig. 33-18). The findings of an ill-defined contour of the wall, collapse of the lumen, or intraluminal hemorrhage highly suggest blunt injury to the gallbladder.[127] Blood in the gallbladder can cause stasis and blockage of the cystic duct, which may present later as acute cholecystitis.[128] With any magnitude of injury to the gallbladder, cholecystectomy is recommended.

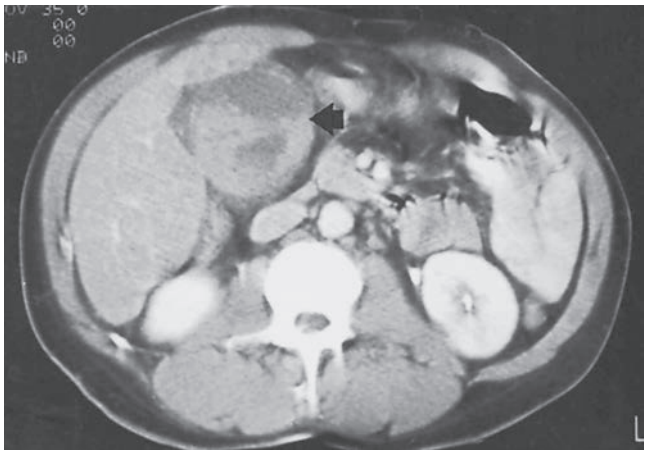


FIGURE 33-18 Computed tomography scan revealing a distended gallbladder filled with blood (dark arrow) in a patient with blunt abdominal trauma and virtually no peritoneal signs.

BILE DUCT

Most injuries to the bile duct are caused by penetrating trauma.[124] Blunt ductal injury occurs where the bile duct is fixed to its surroundings, such as near the pancreaticoduodenal junction.[129] In one multi-institutional trial, it was found that blunt injuries usually led to complete transections, whereas penetrating injuries led to partial transections the majority of the time.[124]

Injuries to the extrahepatic bile ducts are typically discovered at the first laparotomy. A much smaller group of patients have presented 24 hours and up to 6 weeks after trauma if the injury was not diagnosed during the original evaluation or operation. Patients with late presentation develop jaundice, abdominal distention and pain, intolerance to enteral feedings, a fever, or worsening base deficit due to bilious ascites or infection.[125]

Evaluation of the stable patient with a CT scan or ultrasound in the acute setting will not be able to differentiate abdominal blood from bile. There may be fullness of the head of the pancreas, duodenal thickening, or portal edema on the admission CT, but these are nonspecific findings. In the presence of bile staining noted at operation and no obvious injury, a cholangiogram through the gallbladder can be helpful.[130] A diagnostic peritoneal lavage would have lack of specificity for a ductal injury because injuries to the duodenum, small bowel, and liver may also produce bile.[130] Late presentation of a bile duct injury may not be recognized until symptoms are apparent. At that time, CT, ultrasound, or ERC can be used to visualize bile collections and localize the injury.[131]

Management of Injury to Extrahepatic Biliary Ducts

GENERAL CONSIDERATIONS

Extrahepatic biliary injuries remain a rare entity. A 12-year experience from the Royal Melbourne Hospital in Australia noted a 0.1% incidence of biliary tract injuries among all

trauma admissions. Blunt trauma caused 77% of the injuries, whereas 23% were from penetrating wounds. Concomitant injuries were present in 97% of patients, thus illustrating the importance of modern imaging and abdominal exploration as needed.[132]

General operative principles described for a major hepatic injury apply to a ductal injury as well. A hematoma or bleeding around, or within, the hepatoduodenal ligament or a severe hepatic parenchymal injury leading to the porta hepatis should raise suspicion of a portal triad injury. Bile staining should be fully investigated, as 12% of bile duct injuries may be missed at the initial operation.[129] The Pringle maneuver is helpful in decreasing the inflow to a vascular injury in the portal triad. In order to obtain adequate examination and exposure for repair, a wide right medial visceral rotation may need to be performed. This maneuver includes mobilizing the ascending colon and hepatic flexure, thus exposing the duodenum. Similarly, a full Kocher maneuver may be necessary to improve exposure of the entire common bile duct.

GALLBLADDER

As noted earlier, an isolated injury to the gallbladder is most often managed with open cholecystectomy; however, there have been reports of laparoscopic cholecystectomy in highly selected patients after penetrating trauma.[133] If chosen, laparoscopic cholecystectomy should include evaluation of adjacent structures, as most injuries to the gallbladder are associated with other intra-abdominal injuries. Although the laparoscope can give a good superficial exam of the peritoneal cavity, visualization of the duodenum, pancreas, and porta hepatis is difficult and requires some expertise. A minor contusion to the gallbladder could be managed nonoperatively,[134] but cholecystectomy is recommended to avoid delayed rupture.[135] Cholecystectomy should also be performed on all patients with injury to the cystic duct or right hepatic artery that would eliminate the blood supply to the gallbladder.

BILE DUCT

In the unstable patient, packing and placement of drains in the area of a presumed ductal injury are adequate until reexploration is performed. In the somewhat more stable patient who is becoming coagulopathic, a small T-tube placed in the injured duct will provide adequate drainage until a formal repair can be accomplished.[136] With a partial transection of a right or left hepatic duct, insertion of a small T-tube into the common hepatic duct with a long limb traversing the partially transected area, even without suturing, may provide enough support for full healing.

For the stable patient, definitive repair is preferred at the first operation. Four broad categories of injuries to the extrahepatic biliary ducts are as follows: (1) avulsion of cystic duct or small laceration; (2) transection without loss of tissue; (3) extensive defect in the wall; and (4) segmental loss of ductal tissue.[137]

Avulsions and small lacerations in the duct can have a small T-tube inserted into the defect or can be repaired primarily with 6-0 polyglycolic suture, making sure not to narrow the lumen. A T-tube with a limb under the repair can be used; however, this may be difficult to insert in a patient with a normal-sized duct. The techniques used to place a T-tube may also devascularize an already compromised duct. For avulsions in which primary repair may narrow the lumen, a piece of the cystic duct or proximal gallbladder wall can be rotated down for the repair as a cholecystectomy is performed.[138]

A rare stab wound that results in a clean transection of the bile duct without significant tissue loss can be repaired with an end-to-end anastomosis. Only minimal dissection around the duct or the lacerated ends is performed in order to avoid damage to the fragile ductal arteries at the 3 and 9 o'clock positions. Tension and/or ischemia of the anastomosis will certainly lead to stricture. End-to-end anastomoses are contraindicated after GSWs, especially since Ivatury et al[139] reported a 55% stricture rate after these repairs. Stewart and Way[140] had success in 67% of patients initially managed with Roux-en-Y reconstruction for complete duct laceration following laparoscopic cholecystectomy, with failure in all lacerations treated with an end-to-end anastomosis.

Extensive wall defects and segmental tissue loss require a biliary-enteric anastomosis as well (Fig. 33-19). In the past, many methods of "patching" were attempted. Saphenous vein grafts have had issues with shrinking and fibrosis, which then required stenting.[141] Prosthetic patches and jejunal mucosal patches have also been tried with anecdotal success only.[142]

Deciding which type of biliary-enteric anastomosis to perform depends on the injury location, access, and size of the duct. Roux-en-Y choledocho- or hepatodochojejunostomy with cholecystectomy is the most utilized approach to a complex injury. Healthy bleeding from the proximal end of the transected duct(s) is the main criterion for success (ie, avoidance of late stricture). A retrocolic Roux limb at least 40 cm long is created and brought up to the common bile or hepatic duct, or even to the hilar plate. An avulsion of the hepatic ducts at the bifurcation can be managed by suturing the ducts together medially before the end-to-side hepatodochojejunostomy.[143] If the distal common duct is not found due to its retraction behind the pancreas, drainage of the area is necessary.[130] Cholecystojejunostomy and ligation of the very distal common bile duct are a possibility if intraoperative cholangiography reveals a patent cystic duct. This is a viable option, especially in patients with small-caliber ducts or instability.

Placing a stent through a biliary-enteric anastomosis is a controversial topic. Surgeons in favor of stenting report that stenting allows for decompression and may allow for cholangiography if a T-tube is the stent. T-tubes must exit the duct outside of the repair area or a stricture will result. Some surgeons feel comfortable without stents, stating that a foreign body in an already small duct may promote stricture or obstruction.[144]

When an injury to the distal common bile duct is discovered, a pancreaticoduodenectomy may be appropriate if combined duodenal and pancreatic injuries are present as well. Primary repair or reimplantation of an avulsed ampulla should only be performed by an experienced hepatopancreatobiliary surgeon.

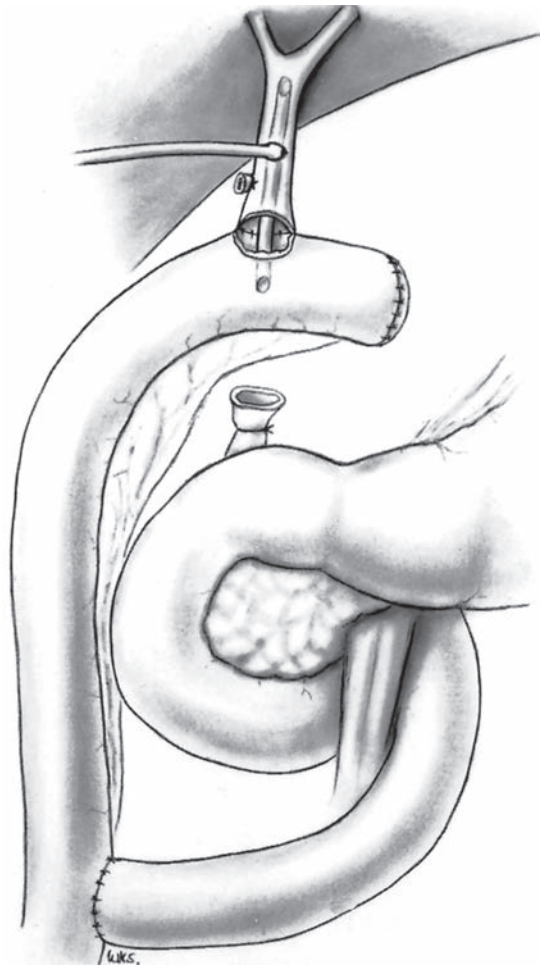


FIGURE 33-19 Roux-en-Y choledochojejunostomy. Anastomosis is performed in a one-layer fashion. The T-tube is brought out through a separate proximal stab wound. The gallbladder has been removed.

The major complications after repairs of injuries to extrahepatic biliary ducts are fistulas and strictures. A fistula may be managed nonoperatively with drainage alone, whereas a persistent fistula occasionally requires reoperation with a new reconstruction. A delayed stricture will present with recurrent episodes of cholangitis. A recent publication describes an aggressive technique of placing an increasing number of stents through a stricture until it resolves. Although the authors did have a complication rate of 9%, their mean duration of treatment was 12 months with a 48.8-month stricture-free interval after treatment.[145] Conversely, Johns Hopkins reported their experience with operative management of all postoperative bile duct strictures and had a 98% success rate.[146]

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34

Spleen

Thomas H. Shoultz • Joseph P. Minei

KEY POINTS

- A high suspicion for splenic injury should exist when patients present with signs or symptoms of trauma to the left upper quadrant or thoracoabdominal region.
- In hemodynamically stable patients, dual-phase, multidetector computed tomography scan of the abdomen with intravenous contrast allows grading of splenic injuries and aids in clinical decision making.
- The revised Organ Injury Scale from the American Association for the Surgery of Trauma incorporates vascular injury and active bleeding as grade IV and grade V injuries.
- For patients with splenic vascular injury or active bleeding, angiography and embolization can significantly increase the success rate of nonoperative management over observation alone.
- If nonoperative management for splenic injury is undertaken, serial physical exams are imperative for avoiding missed injuries to the hollow viscera.
- For patients requiring surgical intervention, mobilization of the body and tail of the pancreas with the splenic hilum allows full mobilization of the spleen for examination and appropriate surgical treatment.
- The incidence of overwhelming postsplenectomy infection is exceptionally rare, but vaccination in the postoperative period remains the current standard of care.

Splenic injuries demonstrate themselves clinically more often than do hepatic injuries, making it the most commonly injured solid viscus requiring laparotomy. During the past 50 years, there has been increasing interest in the notion that not all splenic injuries require splenectomy. Nonoperative management with close observation is safe in appropriately identified patients. There is also increasing evidence supporting the safety of selective angioembolization; however, optimal patient selection is still critical. Although the paradigm has shifted toward frequent consideration of nonoperative management, it is important to always keep in mind that patients with splenic injury can succumb to hemorrhage.

HISTORICAL PERSPECTIVE

The spleen has been subject to injury for as long as man has suffered trauma. In ancient India, where malaria was endemic and large, fragile spleens were commonplace; intentional injury of the spleen was a method of assassination. Paid assassins called *thuggee* carried out their mission by delivering a blow to the left upper quadrant of the intended victim. They hoped to cause splenic rupture, and if this were severe enough, the targeted would bleed to death.

Ancient Greeks and Romans felt the spleen to play a significant role in human physiology. Aristotle thought that the spleen was on the left side of the body as a counterweight to the right-sided liver.[1] He believed that the spleen was important in drawing off "residual humors" from the stomach. The close relation of the stomach and spleen and the presence of the short gastric vessels so important in present-day splenic mobilization likely encouraged this belief. The spleen was also felt to "hinder a man's running," and Pliny reportedly claimed that "professed runners in the race that bee troubled with the splene, have a devise to burne and waste it with a hot yron."[2] The exceptional speed of giraffes was felt to be related to the erroneous belief that giraffes were asplenic. Early references to removal of the spleen to increase speed make it apparent that it has long been known that the spleen is not absolutely necessary to sustain life. Paracelsus believed that the spleen could be removed, and rejected the notion that it was important for the storage of "black bile."[3]

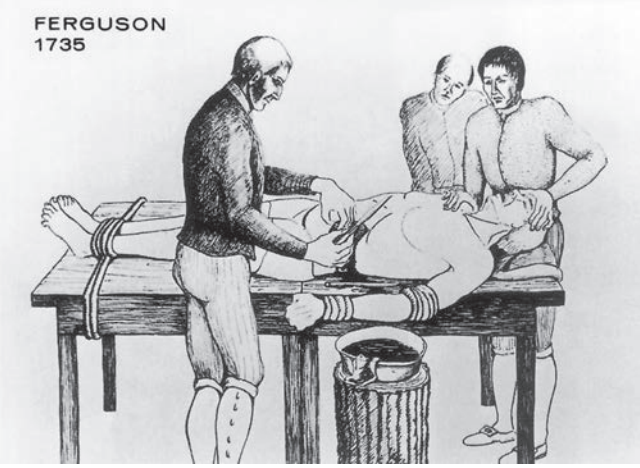


FIGURE 34-1 A depiction of the partial splenectomy done by John Ferguson of Scotland and reported in 1738. The operation actually had been done some years earlier. (Reproduced with permission from Hiatt JR, Phillips EH, Morgenstern L, eds. *Surgical Diseases of the Spleen*. Berlin Heidelberg, Germany: Springer-Verlag; 1997:6. Copyright © Springer-Verlag, Berlin Heidelberg 1997.)

In 1738, John Ferguson of Scotland removed a portion of the spleen through an open wound in the left side (Fig. 34-1). Once the era of abdominal surgery had begun, it was discovered that the spleen could be removed with what seemed like relative impunity. Mayo reported in 1910 that "the internal secretion of the spleen is not important, as splenectomy does not produce serious results."[4] Although some suggested that the spleen was important in some way for immune function and for the removal of senescent red blood cells, it was not felt that these functions were of great importance. Until several decades ago, this resulted in a philosophy in which any traumatic splenic injury, no matter how trivial, was treated with splenectomy.[3,4]

There were some early thoughts that the spleen played a role in combating infection, but it only has been in the last century that our understanding of the role of the spleen in immune function has developed.[5] The initial clinical impetus to more closely examine the immunologic role of the spleen was based on the observation that neonates and infants who required splenectomy for hematologic disease suffered otherwise inexplicably high rates of postoperative morbidity and mortality from overwhelming infection. Pneumonia and meningitis secondary to pneumococcus species and other encapsulated organisms were particularly common. The dramatic consequences of splenectomy in this very specific group of patients led to the investigation of the effects of splenectomy in pediatric trauma patients. Although the evidence for severe immunologic consequences of splenectomy in this group was less convincing than in pediatric patients with hematologic disease, there was a strong signal that splenectomy for trauma would lead to an increased rate of overwhelming sepsis, just as occurred after splenectomy for hematologic disease. Cases of overwhelming postsplenectomy sepsis in adults who had undergone splenectomy for trauma were also reported.[6,7]

The development of improved abdominal imaging and increasing questions about the safety of transfused blood paralleled our increased understanding of the importance of the spleen for immune function. The advent of computed tomography (CT) scanning of the abdomen and its continued improvement in speed and resolution markedly increased our ability to diagnose splenic injury nonoperatively, and it became apparent that clinically silent splenic injuries could occur. Concerns about the safety of stored blood transfusion with respect to hepatitis and human immunodeficiency virus, however, led to pressing questions about transfusions for patients with splenic injury.

Most splenic injuries are due to the same blunt and penetrating mechanisms that cause other traumatic injuries. Increased use of percutaneous procedures and abdominal ultrasound has revealed more obscure mechanisms such as colonoscopic manipulation and placement of a nephrostomy tube as potential causes of splenic injury.[8,9]

SPLENIC FUNCTION

Histologically, the spleen is divided into what has been termed *red pulp* and *white pulp*. The red pulp is a series of large passageways that filter old red blood cells and trap bacteria. Filtering is important in removing poorly functioning senescent red blood cells from the bloodstream and in keeping the hematocrit and blood viscosity within a normal range. The trapping of bacteria in the filters of the red pulp allows the antigens of the bacterial walls to be presented to the lymphocytes in the adjacent white pulp. The white pulp is filled largely with lymphocytes located such that they can be exposed to antigens either on microorganisms or freely circulating. Lymphocyte exposure to antigens results in the production of immunoglobulins. Other potentially important functions of the white pulp are the production of opsonins and activation of complement in response to appropriate stimuli.

All these functions of the spleen are, of course, lost after splenectomy. Collections of lymph tissue are also found in the liver, thymus, intestinal tract, and skin, and these areas may take over some of the functions of the spleen after splenectomy. In addition, some of the necessary functions of the spleen could conceivably be carried out by accessory spleens, but the removal of the spleen results in loss of most filtering and immune functions. How serious these losses are to normal function is a matter of debate. The loss of the filtering function of senescent red blood cells seems to be tolerated reasonably well. Although certain kinds of senescent red blood cells in the bloodstream are more pronounced after splenectomy, the homeostatic genesis and removal of red blood cells seems to remain intact. The loss of splenic function has been the subject of a great deal of investigation. There is evidence of an increased incidence of overwhelming sepsis after splenectomy for trauma; however, the precise incidence, especially in adults, is so low that it is difficult to quantify.

The possibility that small accessory spleens might provide residual splenic function raises the question of how much

splenic mass is necessary for the filtering and immune functions of the spleen. This is a question of more than academic importance, in that a variety of surgical techniques have been described for partial splenectomy or autotransplantation of the spleen after splenectomy.[10,11] The exact amount of spleen to reimplant after splenectomy or leave behind after partial splenectomy is dependent on the minimum amount of splenic tissue necessary for normal function. How much spleen is necessary for normal function is not precisely known but is thought to be between 30% and 50%.[12]

SPLENIC ANATOMY

The spleen develops initially as a bulge on the left side of the dorsal mesogastrium and begins a gradual leftward migration to the left upper quadrant. It changes in relative size during maturation. In children, it is relatively large because it is necessary for both reticuloendothelial function and production of erythrocytes. As the child's bone marrow matures, the spleen becomes less important and diminishes in size relative to the rest of the body. There are also differences between pediatric and adult spleens with respect to the splenic capsule and the consistency of the splenic parenchyma. The capsule in children is relatively thicker than it is in adults, and the parenchyma is firmer in consistency in children than it is in adults. These two differences have implications for the success of nonoperative management. A thicker capsule and tougher parenchymal consistency imply that pediatric spleens are more likely to survive an insult without major bleeding and the need for operative intervention. This is part of the explanation for why children are more often candidates for nonoperative management than are adults, and why nonoperative management tends to be somewhat more successful in children than it is in adults.

The normal adult spleen ranges in size from 100 to 250 g. A number of disease processes, however, can change both the size and consistency of the spleen. Malaria and its effects on the spleen with respect to enlargement and changes in consistency have been referred to earlier. Hematologic diseases such as lymphoma and leukemia can also change both the size and consistency of the spleen and make it more susceptible to damage. Other more common diseases such as mononucleosis make the spleen more vulnerable to injury. An equally important and prevalent pathology that can increase splenic vulnerability is portal venous hypertension. Usually such portal hypertension is secondary to cirrhosis of the liver, and when it is present, the spleen can become both enlarged and less firm in consistency.

It is perhaps not intuitive from the anteroposterior views depicted in anatomy textbooks, but the spleen is normally located quite posteriorly in the upper abdomen (Fig. 34-2). It is covered by the peritoneum except at the hilum. Posteriorly

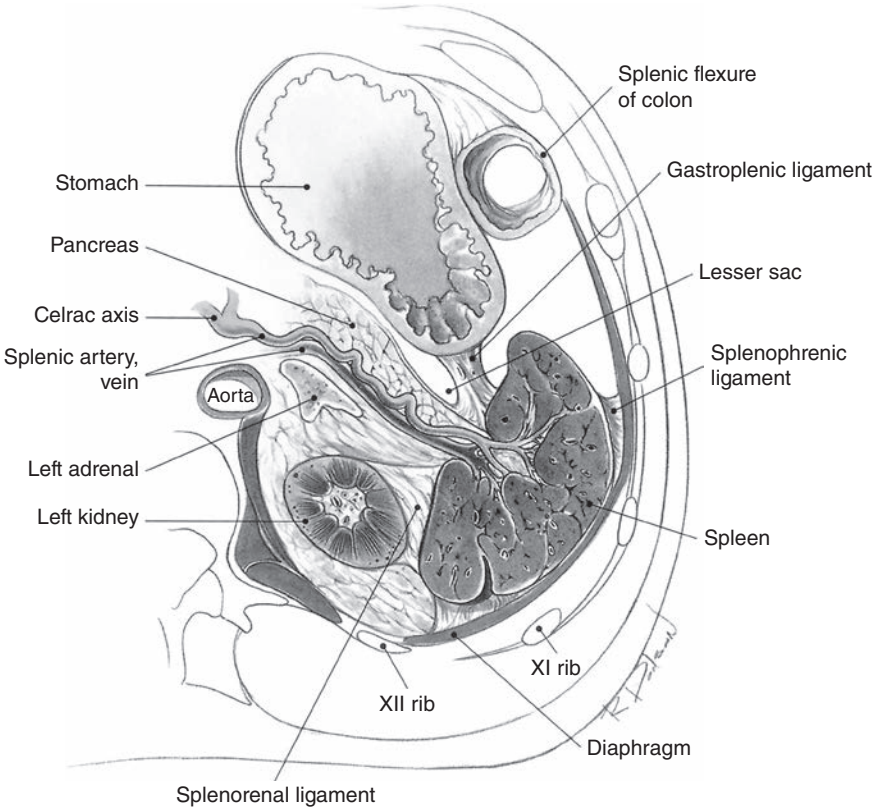


FIGURE 34-2 The spleen is located quite posteriorly in the left upper quadrant and is attached to surrounding structures by a variety of ligaments. (Reproduced with permission from Carrico CJ, Thal ER, Weigelt JA, eds. *Operative Trauma Management: An Atlas*. Norwalk, CT: Appleton & Lange; 1998. Copyright The McGraw-Hill Companies, Inc.)

and laterally the spleen is related to the left hemidiaphragm and the left lower ribs. The lateral aspect of the spleen is attached to the posterior and lateral abdominal wall and the left hemidiaphragm (splenophrenic ligament) with a variable number of attachments that require division during mobilization of the spleen. The extent of these attachments is quite variable. Minimal attachments will result in a fairly mobile spleen. Thick attachments, when present, will require sharp dissection. The lateral attachments tend to be smaller and less extensive in children than in adults. As the spleen lies adjacent to the posterior ribs on the left side, left posterior rib fractures should increase the index of suspicion for an underlying splenic injury. Because of the close relation of the spleen to the diaphragm, simultaneous injuries to the two structures are not uncommon. After penetrating trauma, a knife or bullet can injure both the left hemidiaphragm and the spleen. The diaphragm can also be injured in blunt trauma, and the spleen, either injured or uninjured, can herniate through a diaphragmatic defect into the left pleural space. The diaphragm should always be closely inspected during surgery for splenic injury.

Posteriorly, the spleen is related to the left iliopsoas muscle and the left adrenal gland. The left adrenal gland is usually fairly small and has a characteristic yellow-gold color. It tends to be related to the posterior aspect of the superior portion of the spleen and should be protected when seen during splenic mobilization.

Posteriorly and medially, the spleen is related to the body and tail of the pancreas. Therefore, mobilizing the tail and body of the pancreas along with the spleen increases the extent to which the spleen can be elevated out of the left upper quadrant.

Medially and to some extent anteriorly, the spleen is related to the greater curvature of the stomach. This relation is important in that the spleen can receive a variable amount of blood supply from short gastric branches of the left gastroepiploic artery. The short gastric vessels require division during full mobilization of the spleen.

Posteriorly and inferiorly, the spleen is related to the left kidney. There are attachments between the spleen and left kidney (splenorenal ligament) that require division during mobilization of the spleen. The left kidney should be left in place while mobilizing the spleen and tail of the pancreas from lateral to medial. There are exceptions to leaving the kidney in place, most notably if the kidney also has been injured or if mobilization of the spleen is being done to provide exposure to the aorta from the left side.

Finally, the spleen is related inferiorly to the distal transverse colon and splenic flexure. The lower pole of the spleen is attached to the colon (splenocolic ligament), and these attachments require division during splenic mobilization.

The splenic artery, one of the major branches of the celiac axis, takes a tortuous course along the superior aspect of the body and tail of the pancreas toward the splenic hilum. It divides at its terminus into a variable number of branches that provide a segmental blood supply to the spleen. Both the number of branches and the site at which the branching occurs are quite variable (Fig. 34-3). This variability is of surgical significance in that there is no absolute and dependable number of splenic artery branches that require division during splenectomy or segmental resection of the spleen. Most commonly, a number of separate splenic artery branches are ligated during splenectomy rather than a single ligation of the main splenic artery. It is possible to find the splenic artery along the superior margin of the body and tail of the pancreas if necessary, and sometimes it is helpful to ligate the artery at that location even after hilar branches have been ligated if there is evidence of continued bleeding.

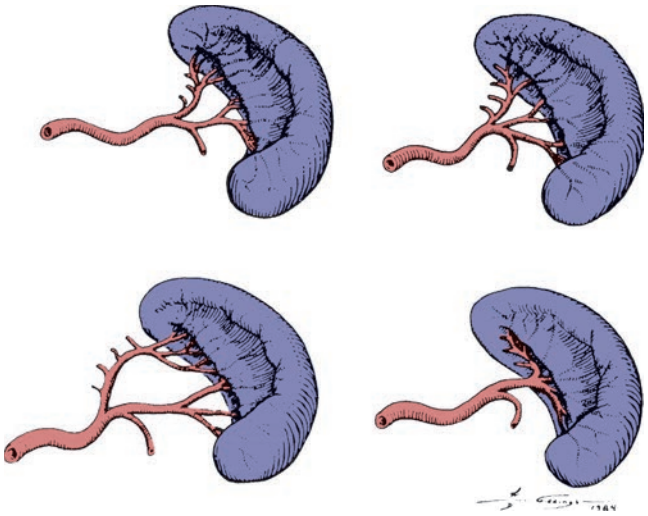


FIGURE 34-3 The arterial blood supply to the spleen can be quite variable. The most common configuration consists of two extraparenchymal divisions of the splenic artery (upper left figure).

The other sources of arterial blood supply for the spleen are the short gastric vessels that connect the left gastroepiploic artery and the splenic circulation along the greater curvature of the stomach. There is an average of four to six short gastric arteries. As implied by their name, these branches off the greater curvature are generally fairly short and are easily injured during mobilization of the spleen.

The venous drainage of the spleen, like the arterial inflow, is via two routes. The splenic vein drains the spleen via a number of tributaries that coalesce to form a single large vein that courses along the posterior aspect of the body and tail of the pancreas to its confluence with the superior mesenteric vein. Like venous anatomy elsewhere in the body, the location, size, and branches of the splenic vein can be quite variable. The other route of splenic venous drainage is via short gastric veins that course adjacent to the short gastric arteries. They drain into the left gastroepiploic vein during its course along the greater curvature of the stomach.

PATHOPHYSIOLOGY OF INJURY

Although nonoperative management is often appropriate after splenic injury, many patients with splenic injury still need emergency surgery to control the hemorrhage. In a large

multi-institutional survey, approximately 45% of patients with splenic injury required emergency surgery.[13] A study using rigid predefined criteria for nonoperative management revealed that 33% of isolated blunt splenic injuries require immediate operation and a further 23% treated with initial nonoperative management required operation, for an overall 56% operative rate.[14] Overall operative rates vary depending on setting, with higher operative rates for rural and nonteaching hospitals.[15] The higher operative rate in smaller hospitals may in part be due to different management strategies. The differential operative rates may be related to the fact that large referral hospitals see a higher percentage of transferred patients who have already withstood the test of time and have proven themselves good candidates for nonoperative management. The rate also varies when comparing large multi-institutional series with single-institutional series. Regardless of the setting, however, it is clear that rapid operative intervention is sometimes necessary. This is particularly true when patients have a coagulopathy either from preinjury anticoagulation or as a consequence of their injury.

Hemorrhage can also be a problem on a delayed basis. The concept of "delayed rupture" of the spleen is actually a misconception. The initial notion that the spleen could rupture on a delayed basis dates back to an era before abdominal CT scanning. In that era, it was observed that some patients who had suffered a traumatic injury did not manifest overt bleeding from their spleen for a number of days, or sometimes even weeks or months, after the traumatic event. With the advent of abdominal CT scanning, it became apparent that these were probably cases of "delayed bleeding" rather than delayed rupture. The distinction between these two entities is more than academic. If "delayed rupture" of the spleen can occur without much evidence of preexisting injury, CT scanning of the abdomen shortly after injury would be negative. In this case, there would be no good way to screen patients and make sure that they were not at risk for delayed splenic bleeding. In contrast, if what used to be called *delayed rupture* is actually just *delayed bleeding*, early diagnosis of the presence of the splenic injury should allow us to tailor our management such that the risk of the delayed bleeding is minimized.

Penetrating injuries to the spleen are most commonly managed operatively, often because of concerns about associated intraperitoneal injuries. Concerns about injury to the stomach, colon, or diaphragm from the knife or bullet are a common rationale for operative intervention in patients with penetrating injury to the spleen. However, some retrospective series have shown that selective nonoperative management of abdominal gunshot wounds in hemodynamically stable patients without peritonitis is as safe as immediate operation and may even be associated with lower rates of complication and hospital length of stay. From a practical standpoint, it is still unknown which patients on presentation are most likely to succeed with this nonoperative approach.[16,17] Operative intervention, of course, does not mandate splenectomy after penetrating injury any more than it does after blunt injury, although the risk of major arterial disruption after penetrating trauma is somewhat higher than after blunt trauma. Attempts at splenic salvage are reasonable after blunt or penetrating splenic injury, especially if the grade of injury is low and associated injuries are not particularly severe.

INITIAL EVALUATION AND MANAGEMENT

As with any other trauma patient, the initial management of the patient with splenic injury should follow the ABCs (airway, breathing, and circulation) of trauma evaluation and resuscitation. A particularly important general comment relative to initial resuscitation is that it is important to recognize refractory shock early and treat it with an appropriate operative response. There are some aspects of the initial evaluation, with respect to the spleen, that deserve special mention:

1. The possibility of an additional intra-abdominal injury in patients with splenic injury seen on CT scanning should be kept in mind. Injury to the gastrointestinal tract is of particular concern.
2. While operating on patients with splenic injury, it is important to look for associated injuries, particularly to the left hemidiaphragm and the pancreas.
3. When mobilizing the spleen, always mobilize the tail of the pancreas medially with the spleen to optimally expose the splenic hilum and minimize risk to the spleen and pancreas.
4. Despite the fact that nonoperative management of splenic injury is a commonly successful strategy, patients can still bleed to death from splenic injury. Therefore, a significant percentage of patients still require surgical intervention and splenectomy.

Elements of the history may be helpful in the diagnosis of splenic injury, and mechanism of injury is important. In patients injured in a motor vehicle crash, the position of the patient in the car can be of some importance in diagnosing splenic injury. Victims located on the left side of the car (drivers and left rear passengers) are perhaps slightly more susceptible to splenic injury because the left side of their torso abuts the left side of the car. This does not mean, however, that victims in other locations in a vehicle are not at risk. For patients who have suffered penetrating injury, the type and nature of the weapon may be important.

In the initial history taking, it is important to note any previous operations the patient has undergone. Of particular importance are any operations that may have resulted in splenectomy (ie, previous operations for hematologic disease or abdominal trauma). Any preexisting conditions that might predispose the spleen to enlargement or other abnormality should be asked about. The patient or significant others should be asked about the presence of hepatic disease, ongoing anticoagulation, or recent usage of aspirin or nonsteroidal anti-inflammatory drugs.

On physical examination, it is important to determine if the patient has left rib pain or tenderness. Left lower ribs are particularly important in that they overlie the spleen,

especially posteriorly. Even with tenderness over the left lower ribs as their only indication of possible abdominal injury, 3% of patients will have a splenic injury.[18] In children, the plasticity of the chest wall allows for severe underlying injury to the spleen without the presence of overlying rib fractures. Such a phenomenon is also possible in adults but is less common than it is in children.

The absence of tenderness over the left lower ribs does not preclude the presence of an underlying splenic injury and, in some cases, may be related to an altered level of consciousness from an associated traumatic brain injury (TBI) or intoxication. In elderly patients, rib fractures may not manifest in a fashion similar to that seen in younger patients. Patients over the age of 55 may not describe lower rib pain and may not have particularly noteworthy findings on physical examination despite severe trauma to the chest wall and an underlying splenic injury.

Another finding on physical examination that is occasionally helpful in the presence of a splenic injury is the presence of Kehr's sign. Kehr's sign is the symptom of pain near the tip of the left shoulder secondary to pathology below the left hemidiaphragm. There is minimal shoulder tenderness, and the patient typically does not have pain on range of motion of the left arm and shoulder unless there is an associated musculoskeletal injury. Kehr's sign after splenic injury is the result of irritation of the diaphragm by subphrenic blood. The sensory innervation of the left hemidiaphragm comes from cervical roots 3, 4, and 5, which are redundant for the ipsilateral shoulder, and referred pain from the diaphragmatic irritation causes the left shoulder pain. Although it is relatively uncommon, the presence of Kehr's sign shortly after trauma should increase the index of suspicion for splenic injury.

The physical examination of the abdomen sometimes demonstrates localized tenderness in the left upper quadrant or generalized abdominal tenderness, but not all patients with splenic injury will reliably manifest peritoneal or other findings on physical examination. Ecchymoses or abrasions in the left upper quadrant or left lower chest may also be present. The unreliability of the physical examination of the abdomen is obvious in patients with an altered mental status and may be absent in patients with normal mentation as well. As a consequence, imaging of the abdomen in hemodynamically stable patients has become an important element of diagnosis and management.

There are no laboratory studies specific to patients with splenic injury, although a hematocrit and type and crossmatching of blood are useful initial laboratory tests. Coagulation studies may be warranted if there is reason to believe that the patient is coagulopathic. As with all other early posttraumatic bleeding, splenic bleeding will not always cause a drop in initial hematocrit. An extremely low hematocrit on arrival of the patient in the resuscitation room, however, especially if the transport has been short and prehospital fluid resuscitation has been minimal, should alert the surgeon to the possibility of severe ongoing hemorrhage.

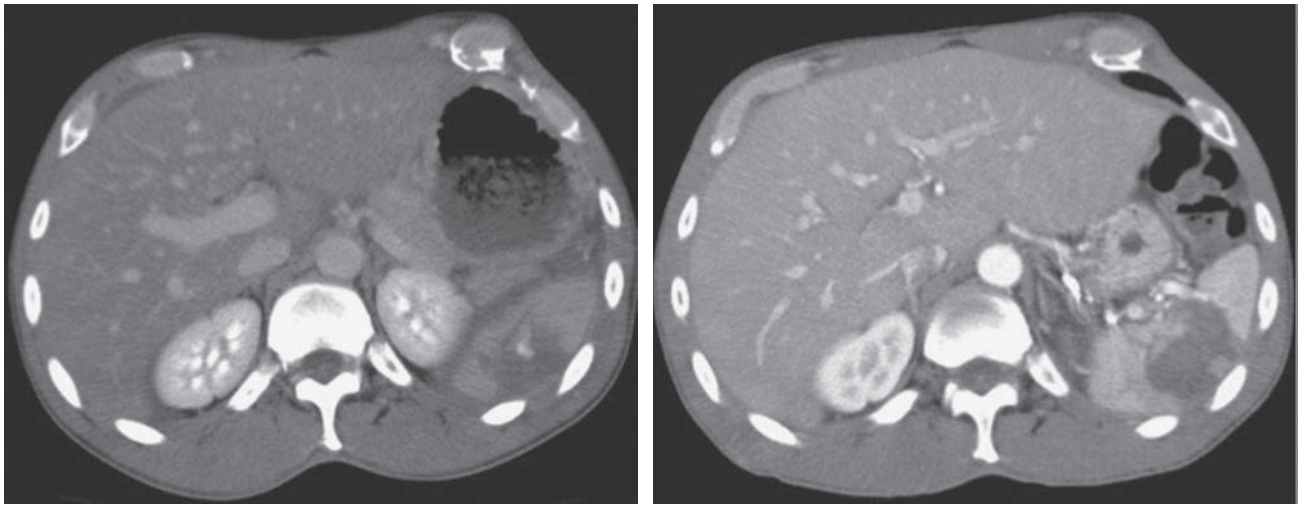
Plain x-rays generally are not helpful in the diagnosis of splenic injury. Rupture of the left hemidiaphragm is sometimes apparent on an initial chest x-ray, however, and can suggest an associated splenic injury. A severe pelvic fracture on an anteroposterior pelvic film can sometimes be of importance in subsequent decision making about how to manage a splenic injury, as the presence of simultaneous splenic and severe pelvic injuries may dictate the removal of the spleen. When penetrating trauma is the mechanism of injury, an initial chest x-ray is important in assessing for associated thoracic injury and, in the case of gunshot wounds, helping to determine the path of a bullet and the location of a retained bullet or bullet fragments.

IMAGING AND DIAGNOSTIC PERITONEAL LAVAGE

Diagnostic peritoneal lavage (DPL) is used much less frequently now. Its role as an initial diagnostic maneuver to dictate subsequent testing or operative intervention has been supplanted in many institutions by both ultrasonography and CT scanning of the abdomen. Peritoneal lavage maintains some utility when ultrasonography is not available in that it is a quick way of determining if a hemodynamically unstable patient has intraperitoneal bleeding. Although DPL is not specific for splenic injury, ongoing intraperitoneal hemorrhage results in a positive peritoneal lavage that should prompt timely operative intervention. DPL may not yield positive results when there is an associated diaphragmatic injury because the instilled fluid may be decompressed and retained in the pleural space. If the DPL yields little or no return of fluid, a diaphragmatic injury should be considered and extraperitoneal sources of hemorrhage assessed.

Ultrasound of the abdomen for free fluid, the so-called focused assessment with sonography in trauma (FAST) exam, is now an essential imaging modality in diagnosing hemoperitoneum in patients with blunt trauma, and in many institutions it has become an additional adjunct to the primary survey. Like DPL, it is most useful for guiding management in unstable patients, but it may also determine the need for further imaging in stable patients. As with peritoneal lavage, the ability of ultrasound to localize bleeding to specific organs in the peritoneal cavity is limited. Small injuries and subcapsular hematomas of the spleen can also be missed by ultrasonography if they do not result in a significant hemoperitoneum. There have been attempts to use ultrasound not only to diagnose intraperitoneal fluid, but also to diagnose specific injuries such as splenic injuries. Such attempts have met with limited success, and the most common reason to perform FAST exams is for detection of intraperitoneal fluid as a determinant of the need for either further imaging of the abdomen or emergency surgery.

CT scanning of the abdomen is the most common imaging study that may allow for nonoperative management of a splenic injury. When performed, it should be done with intravenous contrast and using dual-phase imaging. Oral contrast is much less helpful and does not increase the sensitivity of CT for detecting a splenic injury. The importance of



A B

FIGURE 34-4 Computed tomography (CT) images of the abdomen from a 43-year-old male who was assaulted with a bat. (**A**) Representative delayed-phase CT image obtained the day of injury demonstrates a grade IV injury with active extravasation. (**B**) CT image after angioembolization shows partial splenic infarct and no extravasation.

dual-phase imaging was demonstrated by Bosack et al[19] in a retrospective review of 120 blunt abdominal trauma patients. Dual-phase CT has better overall diagnostic performance than single-phase CT for detection of splenic trauma. The arterial phase was important for the diagnosis of splenic pseudoaneurysms, while the portal venous phase was better for the detection of active bleeding and parenchymal injuries.

Radiation exposure from CT, especially in children, has been raised as a potential concern, and some selection should be used with respect to which patients with abdominal trauma should undergo scanning.[20] Undue concern about radiation, however, should not put a patient at risk for a missed splenic injury and occult bleeding.

The findings of splenic injury on CT scan are variable. Hematomas and parenchymal disruption generally show up as hypodense areas. Free fluid can be seen either around the spleen or throughout the peritoneal and pelvic spaces. Locations where fluid frequently accumulates after splenic injury are Morrison's pouch, the paracolic gutters, and the pelvis. When a large amount of fluid is present in the peritoneal cavity, it can sometimes be seen between loops of small bowel as well as in the subphrenic spaces.

When looking at CT scans of patients with splenic injury, it is important to also look at the adjacent left kidney and the distal pancreas. Injury to the spleen implies a blow to the left upper quadrant that can injure adjacent organs. The diagnosis of a pancreatic injury is particularly important in that this can significantly affect the patient's subsequent course and prognosis. Also, it is important to remember that the presence of free fluid is not solely related to bleeding from a visible splenic injury in all cases. One of the pitfalls of CT diagnosis is that free fluid in the peritoneal cavity or in the pelvis may be attributed to a splenic injury when in fact the fluid is secondary to both a splenic injury and an associated injury to the mesentery or bowel.

Other than an obvious injury, the most important CT finding in the spleen is the presence in the disrupted splenic parenchyma of a "blush," which represents active bleeding from a vascular injury and appears as a hyperdense area of contrast that becomes brighter in subsequent phases (Fig. 34-4). Other vascular injuries are pseudoaneurysms and arteriovenous fistulae, which are radiographically indistinguishable from each other and appear as areas of contrast that fade with subsequent phases. There is evidence that the presence of a vascular injury correlates with an increased likelihood that continued or delayed bleeding will occur, resulting in failure of nonoperative management. These arterial injuries need further assessment with either angiography or repeat CT scanning.[21,22]

It is important to remember that, historically, the CT grade of splenic injury and a patient's subsequent clinical course have only been only roughly correlated. Bleeding and nonbleeding vascular injuries are associated with higher-grade parenchymal injuries on CT scan, but CT scan does not capture all vascular injuries. Therefore, high-grade splenic injuries may warrant empiric splenic angiography.[23]

Magnetic resonance imaging (MRI) has been used sporadically in the diagnosis of splenic injury. The images obtained are sometimes quite impressive but, given that CT scanning has both a very high sensitivity and specificity for the presence of splenic injury (especially when newer generation multidetector scanners are used), MRI so far has not proven to hold an obvious advantage. Furthermore, MRI is usually less available than is CT scanning, especially after hours. The logistical difficulties inherent in trying to obtain MRIs in a badly injured patient who requires close monitoring and, possibly, even mechanical ventilation make MRI even less helpful as a diagnostic modality. Continued improvements in MRI and our increasing ability to use it even for critically ill patients could conceivably increase the role of MRI in the characterization of splenic injury in the future.

Radioisotope scintigraphy and conventional angiography were each used for diagnosis of splenic injury in the past but are largely of historical interest at this point, as they have been replaced by CT, as described earlier. Angiography with embolization for bleeding does, however, have an important therapeutic role in the management of splenic injury.

GRADING SYSTEMS FOR SPLENIC INJURY

Based primarily on the CT imaging characteristics of the spleen, a number of different grading systems have been devised to quantify the degree of injury in patients with injured spleens.[24-26] Widespread use of an Organ Injury Scale (OIS) allows for objective standardization of terminology and ensures that individual injuries are described in precise terms understandable to others. Standardized organ injury scaling is useful in clinical research, in describing populations of splenic injury patients, and in risk stratifying patients for treatment algorithms (Fig. 34-5).

Based on available published literature and a consensus of experts in the field, in 2018, the American Association for the Surgery of Trauma (AAST) developed a revised OIS for splenic injuries (Table 34-1).[27] The Solid Organ Injury Scale includes three sets of criteria to assign grade: imaging, operative, and pathologic.

The updated spleen OIS is structured, as with the prior scale, by grades I through V. The most significant change is the incorporation of CT-diagnosed vascular injury, defined as pseudoaneurysm, arteriovenous fistula, or active bleeding. Presence of vascular injury regardless of parenchymal destruction results in a grade IV or V, with the latter defined by active bleeding beyond the splenic capsule. The consideration of these high-risk imaging features in the revised scale acknowledges the increased risk of failure of nonoperative management in their presence and thus provides a potential branch point for the decision to pursue angiography or other splenic intervention.[28]

NONOPERATIVE MANAGEMENT

Nonoperative management of splenic injury has become more common over time. Although approximately 40% of patients with splenic injury will require immediate operative intervention, nonoperative management is reasonable for hemodynamically stable patients.[29]

Patient Selection

Appropriate patient selection is the most important element of nonoperative management. Determining which patients require emergency surgery and which can be initially managed nonoperatively is a multifactorial decision, although hemodynamic status, age, grade of splenic injury, quantity of hemoperitoneum, and associated injuries have been shown to roughly correlate with the success or failure of nonoperative management. The decision for nonoperative management must also consider the institutional resources available.

Of paramount importance in the determination of the appropriateness of nonoperative management is the hemodynamic stability of the patient. Hemodynamic stability can be a somewhat illusory concept and one for which there is no consensus definition. Stability in itself is a beguiling term when describing an acute trauma patient, because patients in shock may maintain stable vital signs until they abruptly decompensate, requiring rapid treatment and further diagnostic maneuvers. Hypotension is generally considered to be worthy of concern. Historically, a prehospital systolic blood pressure (SBP) less than 90 mm Hg has mandated triage to a trauma center. In fact, that criterion is too low, because a prehospital SBP of less than 110 mm Hg has been shown to be associated with poor outcomes.[30,31] Hypotension in the prehospital period or emergency department is worrisome, and a high index of suspicion for ongoing hemorrhage should be maintained when either is present. Patients who have been hemodynamically unstable in the prehospital phase and remain hemodynamically unstable during their initial emergency department stay are, in most instances, inappropriate candidates for abdominal CT scanning. They require either a direct trip to the operating room (OR) or at least a FAST exam to help guide the initial decision-making process.

Assuming hemodynamic stability, the other important prerequisite for consideration of nonoperative management is the patient's abdominal examination. In patients who are awake and alert and can cooperate with a physical examination and provide feedback, it is important that they not have diffuse peritonitis. Although patients with splenic injury will often have localized pain and tenderness in the left upper quadrant and abdominal findings secondary to intraperitoneal blood, obvious diffuse peritonitis can be a sign of intestinal injury and mandates an abdominal exploration. If a patient with a splenic injury is sent for CT scanning and subsequent nonoperative management, it is important to perform repeat physical examinations. If the examination worsens, the possibility of a blunt intestinal injury should be considered. The most common CT finding in patients with blunt intestinal injury is free fluid in the peritoneal cavity. As previously noted, the free fluid can be mistakenly attributed solely to the splenic injury, and the presence of an associated injury to the bowel can be missed.

The success rates of nonoperative management of splenic injuries are very high in many of the published series. Reported success rates for nonoperative management are 95% or higher for pediatric patients and 80% to 94% in adults.[32-34] These high success rates can be misleading, however, in that they apply only to the group of patients in whom nonoperative management was chosen rather than all patients with splenic injury. When patients undergoing immediate splenectomy are included, the overall splenic salvage rates tend to be 50% to 60% in adult patients. It is important to remember that these series generally do not include patients in whom the initial plan was for nonoperative management, but an emergency operation was necessary when the patient became

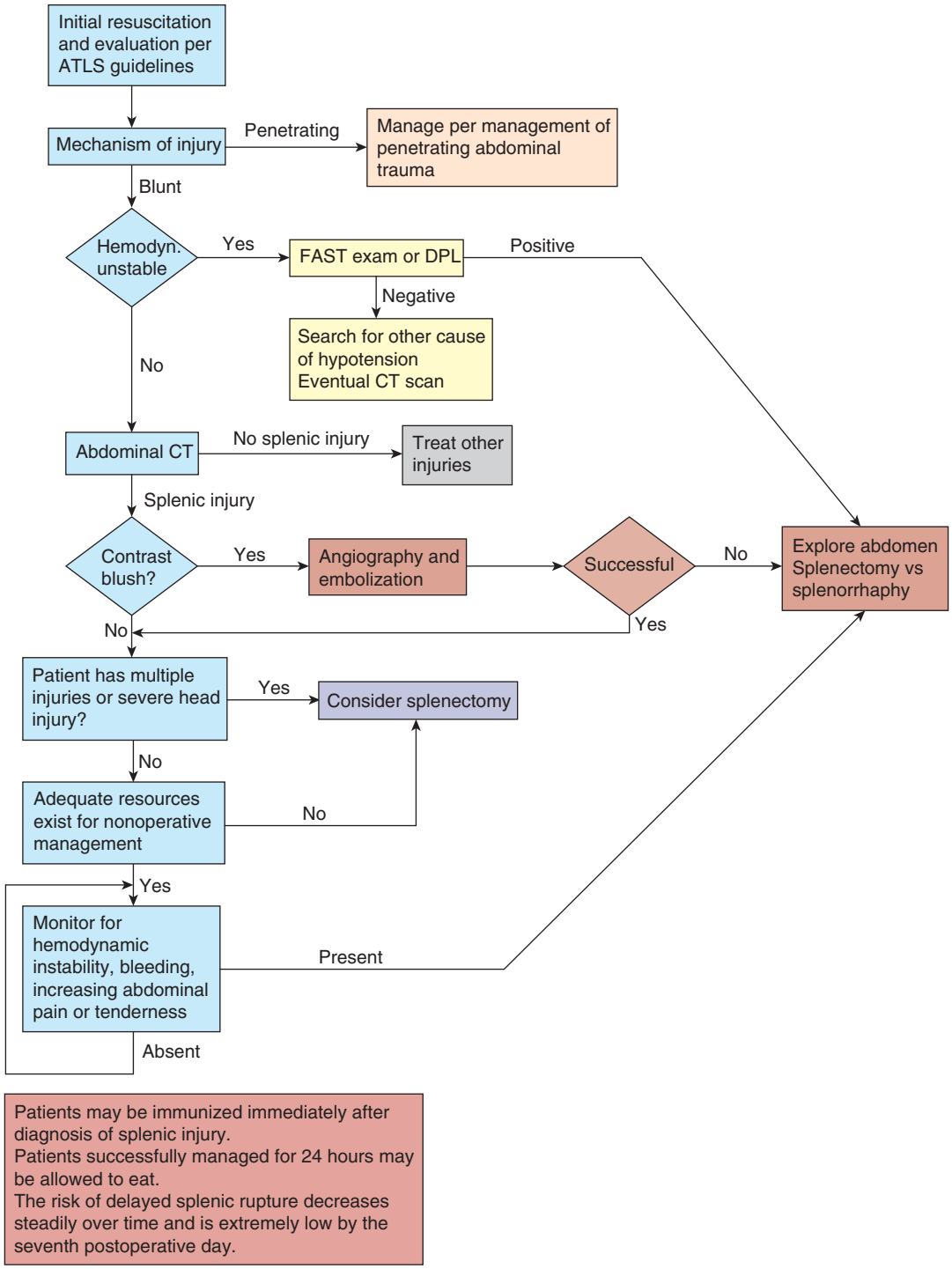


FIGURE 34-5 Algorithm for the diagnosis and management of splenic injury. ATLS, Advanced Trauma Life Support; CT, computed tomography; DPL, diagnostic peritoneal lavage; FAST, focused assessment with sonography in trauma; Hemodyn., hemodynamically.

hypotensive or developed peritonitis in the emergency department or in the CT scanner. The published series of nonoperatively managed spleens generally include only the selected patients who were stable enough to undergo CT scanning of the abdomen and in whom the CT scan showed an injured spleen. Patients who became unstable either before or during the scan and were taken emergently to surgery are usually not counted as patients who underwent "nonoperative" management. When these patients are reported at all, they are placed into the "operative" group rather than into the "failed nonoperative" group. Published series of splenic injuries, particularly in pediatric patients, are more likely to describe patients

TABLE 34-1: The Splenic Organ Injury Scaling System of The American Association for The Surgery of Trauma, 2018 Revision

AAST grade[a]	AIS severity	Imaging criteria	Operative criteria	Pathologic criteria
I	2	• Subcapsular hematoma <10% surface area • Parenchymal laceration <1 cm depth • Capsular tear	• Subcapsular hematoma <10% surface area • Parenchymal laceration <1 cm depth • Capsular tear	• Subcapsular hematoma <10% surface area • Parenchymal laceration <1 cm depth • Capsular tear
II	2	• Subcapsular hematoma 10%–50% surface area; intraparenchymal hematoma <5 cm • Parenchymal laceration 1–3 cm	• Subcapsular hematoma 10%–50% surface area; intraparenchymal hematoma <5 cm • Parenchymal laceration 1–3 cm	• Subcapsular hematoma 10%–50% surface area; intraparenchymal hematoma <5 cm • Parenchymal laceration 1–3 cm
III	3	• Subcapsular hematoma >50% surface area; ruptured subcapsular or intraparenchymal hematoma ≥5 cm • Parenchymal laceration >3 cm depth	• Subcapsular hematoma >50% surface area or expanding; ruptured subcapsular or intraparenchymal hematoma ≥5 cm • Parenchymal laceration >3 cm depth	• Subcapsular hematoma >50% surface area; ruptured subcapsular or intraparenchymal hematoma ≥5 cm • Parenchymal laceration >3 cm depth
IV	4	• Any injury in the presence of a splenic vascular injury[b] or active bleeding confined within splenic capsule • Parenchymal laceration involving segmental or hilar vessels producing >25% devascularization	• Parenchymal laceration involving segmental or hilar vessels producing >25% devascularization	• Parenchymal laceration involving segmental or hilar vessels producing >25% devascularization
V	5	• Any injury in the presence of splenic vascular injury with active bleeding extending beyond the spleen into the peritoneum • Shattered spleen	• Hilar vascular injury that devascularizes the spleen • Shattered spleen	• Hilar vascular injury that devascularizes the spleen • Shattered spleen

[a]Grade based on highest grade assessment made on imaging, at operation, or on pathologic specimen. More than one grade of splenic injury may be present and should be classified by the higher grade of injury. Advance one grade for multiple injuries up to a grade III.
[b]Vascular injury is defined as a pseudoaneurysm or arteriovenous fistula and appears as a focal collection of vascular contrast that decreases in attenuation with delayed imaging. Active bleeding from a vascular injury presents as vascular contrast, focal or diffuse, that increases in size or attenuation in delayed phase. Vascular thrombosis can lead to organ infarction.

AAST, American Association for the Surgery of Trauma; AIS, Abbreviated Injury Scale.

Source: Adapted with permission from Kozar RK, Crandall M, Shanmuganathan K, et al. Organ Injury Scaling 2018 update: spleen, liver, and kidney. *J Trauma Acute Care Surg.* 2018;85:1119-1122.

treated at referral centers where there are large numbers of transfer patients who have already been triaged for stability prior to their arrival at the referral center. Finally, the literature on the success of nonoperative management of splenic injury should be interpreted with the awareness that publication bias tends to favor series in which success rates are high.

Other important considerations beyond hemodynamic stability and abdominal findings in the determination of the appropriateness of nonoperative management have to do with the medical environment and some specific characteristics of the patient. Nonoperative management should generally be undertaken only if it will be possible to closely follow the patient. If close inpatient follow-up is simply not possible, abdominal exploration may be appropriate. Similarly, if rapid mobilization of the OR and quick operative intervention in the case of ongoing or delayed bleeding is not possible, early rather than emergent operative intervention may be appropriate. Finally, the patient's circumstances after discharge occasionally may be important in the decision-making process. Risk of delayed bleeding after 24 hours of successful nonoperative management for grade I splenic injuries is rare. Nonetheless, for patients who are to be discharged to a location remote from medical care, the consequences of delayed bleeding are greater in that they may not be close enough to a hospital that can perform an emergency operative procedure. In such circumstances, an otherwise reasonable candidate for nonoperative management may be considered for operative intervention.

For patients who are stable enough to undergo CT scanning and in whom an injured spleen is identified, nonoperative management is reasonable if they continue to remain

stable. In addition to vital signs, one of the other commonly followed parameters in such patients is the hematocrit. A common practice is to determine a cutoff value below which the hematocrit will no longer be simply monitored. If the hematocrit drops to that level or below, operative intervention is undertaken. Such an approach works best if there are no associated injuries; when associated injuries are present, it can be quite difficult to know if the spleen is continuing to bleed or if the fall in hematocrit is secondary to bleeding from other injuries.

In general, there is consensus that hemodynamically stable patients without peritonitis who can be followed closely are candidates for nonoperative management. Historically, there has been a debate about certain subgroups of patients and their appropriateness for nonoperative management.[35]

Pediatric patients are excellent candidates for nonoperative management because they have a low incidence of delayed bleeding after splenic injury.[36,37] As nonoperative management has become the standard of care in this population, there has been an increase in angioembolization with great success.[38,39] Because of the trauma mechanisms suffered by pediatric patients as opposed to adult patients, children are more likely to have isolated splenic injuries. As previously noted, the relative thickness of the splenic capsule is greater in children, which likely confers more structural integrity to the spleen. The spleen in children is also more likely to fracture parallel to the splenic arterial blood supply rather than transverse to it (Fig. 34-6).[40] This orientation of splenic injury tends to decrease the amount of blood loss from the splenic parenchyma. Children are more likely to have excellent physiologic reserve and minimal preexisting disease. Finally, the risks of splenectomy with respect to immunologic consequences are greater in young children than they are in adults.

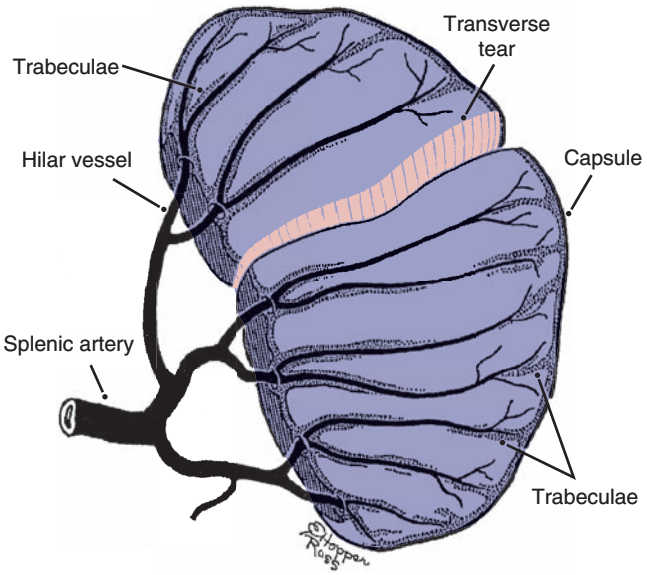


FIGURE 34-6 Diagrammatic representation of a transverse laceration relative to the splenic vasculature in a pediatric patient. (Reproduced with permission from Upadhyaya P. Splenic trauma in children. *Surg Gynecol Obstet*. 1968;126:781, Copyright © Elsevier.)

Historically, older patients were thought to have a worse prognosis with respect to nonoperative management than younger patients. Other series examining the question of the threshold at 55 years of age and nonoperative management suggest the success of nonoperative management is no different in this group than it is in younger patients. In fact, there is an increasing body of evidence that being elderly is not a contraindication for nonoperative management, although the evidence in this area is still somewhat conflicting.[41,42]

The presence of severe associated injuries, particularly a TBI, has been suggested as another relative contraindication to nonoperative management of a splenic injury. As previously mentioned, following the hematocrit in a patient with severe associated injuries can be difficult to interpret because the underlying hemorrhage may not be attributable to the spleen alone. Furthermore, the effects of ongoing or delayed splenic bleeding are felt to negatively impact the prognosis of a severe TBI if secondary injury from hypotension is incurred.

Although these factors do not mandate operative intervention in all patients who fall into these groups, they should lower the threshold for operative intervention on an individual basis.

There is little uniformity about what constitutes a "failed" attempt at nonoperative management. Different surgeons and different institutions have set different criteria for operative intervention, and much of the decision making is subjective. As has already been pointed out, there is no perfect relationship between the severity of injury seen on CT scanning and a patient's subsequent success or failure of nonoperative management. However, higher grade injuries are less likely to succeed with observation alone, and the revised AAST grading criteria take this into account. Some of this discrepancy is probably related to the imperfect nature of the scoring systems and a lack of sensitivity of CT scanning. Also, it is likely that some of the differences are in the approach and thresholds for operative intervention. In some instances, concern about a "bad-looking" spleen on a CT scan might prompt more aggressive and quicker surgical intervention and make failed nonoperative management of severe splenic injuries a self-fulfilling prophecy.

As previously noted, an objective finding on CT scan that has proven useful as a prognostic sign with respect to nonoperative management is that of a vascular injury (eg, pseudoaneurysm, arteriovenous fistula, or active bleeding) in the injured spleen (Fig. 34-4). In general, a vascular injury seen on initial CT scan should be evaluated with angiography and treated with embolization if ongoing bleeding is present and the patient is normotensive (Fig. 34-7). Active bleeding on CT scan is also associated with a higher need for operative intervention. This approach seems reasonable because angiography with splenic embolization has improved success rates in patients managed nonoperatively. The most dramatic improvement is seen in patients with higher-grade splenic injuries. A 2011 meta-analysis revealed a decrease in nonoperative failure rates from 44% to 17% in grade IV injuries and from 83% to 25% in grade V injuries.[43] To optimize therapeutic potential and resource utilization, only patients

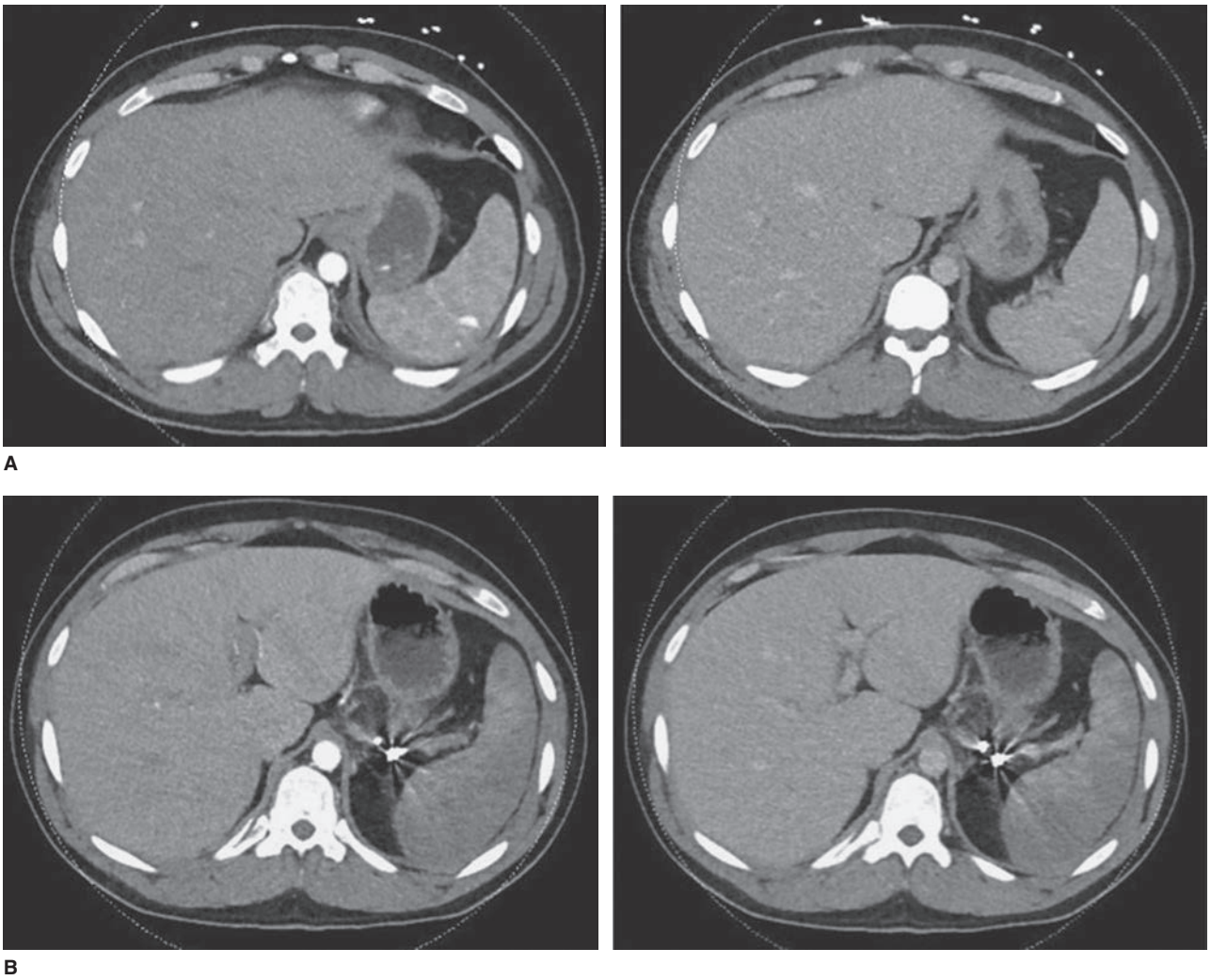


FIGURE 34-7 Computed tomography (CT) images of the abdomen from a 28-year-old male involved in a motor vehicle collision. Representative images with the arterial phase on the left and portal venous phase on the right. (**A**) CT scan reveals a pseudoaneurysm seen in the arterial phase. (**B**) CT images after angioembolization show no evidence of pseudoaneurysm.

with high-grade splenic injuries should undergo angiographic embolization. Prospective evaluation of a protocol to perform angiography and embolization on grade III to V splenic injuries revealed significantly decreased failure rates in the protocol group.[44]

Patient Management

After nonoperative management has been selected, the initial resuscitation should be continued and other diagnostic and therapeutic procedures carried out as necessary. There is little scientific evidence to dictate the specifics of how nonoperative management of splenic injury should be done. The most rigorous attempts to systematize recommendations for nonoperative management have been in children; however, those guidelines may overestimate the minimum amount of time needed for inpatient observation (Table 34-2).[45-47] Strong consideration should be given to admitting to a monitored unit for the patient's initial nonoperative management. This may include patients with at least grade III splenic injuries and patients with multiple associated injuries that make following serial hematocrit levels and physical examinations difficult, and in some centers, even lower-grade injuries. Most centers monitor such patients for 24 to 72 hours. Although there are some theoretical reasons why bed rest might be a good idea, there is little empirical evidence that it makes a difference and it is no longer commonly practiced. The patient's vital signs and urine output should be monitored closely, serial physical examinations performed, and serial hematocrits measured.

Vaccines against encapsulated organisms, *Streptococcus*, meningococcus (*Neisseria meningitidis*), and *Haemophilus influenzae* infections may be considered as part of nonoperative management in high-risk patients. There are some

TABLE 34-2: Proposed Guidelines for Resource Utilization in Children with Isolated Spleen or Liver Injury

	CT grade			
	I	II	III	IV
ICU stay (days)	None	None	None	1
Hospital stay (days)	2	3	4	5
Predischarge imaging	None	None	None	None
Postdischarge imaging	None	None	None	None
Activity restriction (weeks)[a]	3	4	5	6

[a]Return to full-contact, competitive sports (ie, football, wrestling, hockey, lacrosse, mountain climbing) should be at the discretion of the individual pediatric trauma surgeon. The proposed guidelines for return to unrestricted activity include "normal" age-appropriate activities.

Source: From Stylianos S, the APSA Trauma Committee. Evidence-based guidelines for resource utilization in children with isolated spleen or liver injury. *J Pediatr Surg.* 2000;35:164–169, with permission, Copyright © Elsevier.

theoretical reasons to believe that the vaccinations are more effective if given while the spleen is still in situ. The evidence to support such a practice, however, is contradictory, and it is very difficult to study the effectiveness of vaccination timing even in splenectomized patients because the incidence of overwhelming postsplenectomy infection is extremely low.

How long a patient should be kept in the hospital is poorly defined. There is no strong evidence supporting any particular approach, but a large multi-institutional study showed that most failures of nonoperative management occur within the first 6 to 8 days after injury.[48] Some institutions will keep patients in the hospital for an arbitrary length of time, which may range from several days to up to 1 week. Longer periods of observation have obvious financial and insurance implications but will pick up most of the delayed bleeds while the patient is still an inpatient. Our institutional approach is to consider the grade of injury, associated injuries, and social situation of the patient to determine the length of hospital stay. In many circumstances, associated injuries dictate the length of hospitalization more than does the splenic injury. Also, it is important to pay attention to where the patient lives and how close he or she will be to medical attention when deciding about timing of discharge. Patients without the ability to obtain immediate medical attention may need to be kept in the hospital longer.

Prophylaxis against deep venous thrombosis (DVT) is a continuing problem in patients undergoing nonoperative management for a splenic injury. Sequential compression devices on the lower extremities should be used routinely. Early mobilization or range-of-motion exercises are also important in minimizing thromboembolic complications. Pharmacologic prophylaxis is more problematic because of concerns about exacerbating bleeding from the injured spleen. After 24 to 48 hours of successful nonoperative management, it is reasonable to begin pharmacologic prophylaxis against DVT. If associated injuries require it, warfarin prophylaxis is also reasonable beginning approximately 1 week after injury. These recommendations are based primarily on physician gestalt rather than large series data; however, there are an increasing number of studies supporting the safe use of early DVT chemoprophylaxis in the nonoperative management of patients with blunt splenic injuries.[49,50] Both the rate of clinically significant thromboembolic events in patients with splenic injury and the rate of failure of nonoperative management in anticoagulated patients are quite low, making prospective study of the risks and benefits of anticoagulation prophylaxis in this patient population difficult.

The issue of follow-up CT scans in patients undergoing nonoperative management of splenic injuries is controversial.[51,52] Most series indicate that they are not necessary or that the frequency with which they alter management is extremely low. A variety of different suggestions have been made in the literature about follow-up CT scans, ranging from no follow-ups at all to routine follow-ups at frequent intervals. The rationale is that some patients have developed pseudoaneurysms of the spleen on follow-up imaging, even if the initial CT scan did not demonstrate a vascular injury. The author's institutional policy is to study only patients who have persistent abdominal signs and symptoms after a week or more of observation.

When patients are discharged, they should be counseled not to engage in contact sports or other activities during which they might suffer a blow to the torso. The best length of time to maintain this admonition is unknown, but typical recommendations range from 2 to 6 months. There is experimental evidence that most injured spleens have not recovered their normal integrity and strength until at least 6 to 8 weeks after injury, so the recommendation to avoid contact sports for 2 to 6 months seems reasonable. Other than with respect to contact sports, there are no major restrictions for patients who have undergone successful nonoperative management.

OPERATIVE MANAGEMENT (SEE ATLAS FIGURES 56, 57, AND 58)

In general, preoperative antibiotics should be given but do not need to be continued in the postoperative period unless dictated by associated injuries. A nasogastric tube is inserted

during the procedure to decrease the volume of the stomach and allow for easier visualization and mobilization of the spleen.

A midline incision is the best incision for splenic surgery as well as most trauma operations on the abdomen. It is versatile, can be extended easily both superiorly and inferiorly, and is the quickest incision if speed of intervention is important. For operations on an injured spleen, it may be necessary to extend the incision superiorly and to the left of the xiphoid process. This maneuver improves exposure of the left upper quadrant, particularly in obese patients and those with a narrow costal angle.

Transverse incisions in the left upper quadrant have occasionally been suggested for patients with a presumed isolated splenic injury. A midline incision is preferable because it is quicker and allows the surgeon to deal with a variety of different intra-abdominal findings. One situation in which a left subcostal approach may be the incision of choice is when the patient is morbidly obese and preoperative CT scanning has suggested that an isolated splenic injury is present or the patient has had a prior midline laparotomy concerning for a hostile abdomen.

As with all trauma laparotomies, it is important to rapidly examine all four quadrants of the abdomen in patients who are hemodynamically unstable. This initial investigation of the abdomen should not be definitive. It should only be used for rapid exploration and packing, especially of the upper quadrants. Definitive management of any injuries found should not be attempted until the entire abdomen has been inspected. While the quadrants are being inspected, it is helpful to look for clot. Clotting tends to localize to the site of injury, whereas defibrinated blood will spread diffusely in the abdomen. Clotted blood will often indicate the site of an injury and is helpful in determining where to direct definitive management after the abdomen has been packed.

In patients who are thought to have an isolated splenic injury based on initial imaging or failed nonoperative management, direct attention can be turned sooner to the left upper quadrant. If viscera other than the spleen seem to be more badly injured and are bleeding more profusely than the spleen, the spleen should necessarily take second priority and be left packed until it is appropriate to attend to it. In comparison, a quick splenectomy is often a wise early move in a patient with multiple serious injuries in that it rapidly eliminates the splenic hemorrhage as a source of ongoing shock.

Once attention has been directed to the left upper quadrant, all the structures in that quadrant should be inspected (Fig. 34-2). There should be an initial look at the greater curvature of the stomach and the left hemidiaphragm. If the spleen is mobilized, the left hemidiaphragm should be reinspected. If the left hemidiaphragm is injured in a patient with blunt trauma and the spleen is in the left side of the chest, it should be pulled down into the abdomen through the defect. The left lobe of the liver and left kidney should also be inspected, as should the tail of the pancreas. If the spleen is to be mobilized, inspection of the tail of the pancreas is easier after mobilization has been accomplished.

The anterior and anterolateral surfaces of the spleen can sometimes be seen fairly easily through the midline incision prior to any splenic mobilization, particularly if the patient is thin and there is a wide costal margin. If the patient is heavy and/or the costal margin is narrow, adequate inspection without some splenic mobilization may be very difficult. If the left upper quadrant is adequately inspected and there is no evidence of any bleeding or a splenic injury, the spleen does not require mobilization. If it is known that there is a small splenic injury, but it is not the primary reason for abdominal exploration or the spleen does not seem to be bleeding at the time of exploration, splenic mobilization is also not always necessary. If the surgeon is in doubt about the need for mobilization, the best thing to do is to mobilize the spleen so that the full extent of injury is elucidated and the spleen can be repaired or removed as necessary. It is important to be as gentle as possible during mobilization of the spleen so that the splenic injury is not worsened.

Splenic mobilization should be done in a stepwise fashion to provide adequate mobilization while minimizing the chance of furthering injury. Proper mobilization allows for better visualization of the left kidney, the left hemidiaphragm, and the posterior aspects of the body and tail of the pancreas. The sequence of splenic mobilization is important in that it allows for splenic salvage and splenorrhaphy up until the final step of hilar ligation.

In mobilizing the spleen, it is important to remember how posteriorly it is situated (Fig. 34-2). Also, it is important to remember that there is a great deal of variability in the length of the different ligaments around the spleen and in how mobile the spleen is before any dissection is done. If mobilization is done correctly, even spleens with fairly short surrounding ligaments and spleens in obese patients can be mobilized to a level at or above the anterior abdominal wall.

The first step in mobilization of the spleen is to cut the splenophrenic and splenorenal ligaments laterally (Fig. 34-8). This step should be started with sharp dissection and can then be continued with a combination of blunt dissection and further sharp dissection. The dissection should be taken up to near the level of the esophageal hiatus so that all the lateral and superior attachments are cut. Cutting the lateral attachments is sometimes facilitated by putting a finger or clamp underneath them and then sharply dividing the overlying plane. In obese patients and in those with a spleen that is very posterior, it may be necessary to do some of the dissection by feel.

After the lateral attachments have been divided, the next step is to mobilize the spleen and tail of the pancreas as a unit from lateral to medial. One of the easier ways to do this is to cup the nondominant hand over and around spleen while sliding the back of the fingertips against the underlying left kidney, thus elevating the spleen and pancreas from the retroperitoneal bed. The kidney can be palpated easily because it is quite firm and provides an excellent landmark for the proper plane of dissection. A common error is to try to mobilize the spleen alone without the adjacent pancreas. Not mobilizing the pancreas with the spleen is easy to do if the surgeon is not posterior enough and is not in the plane between the

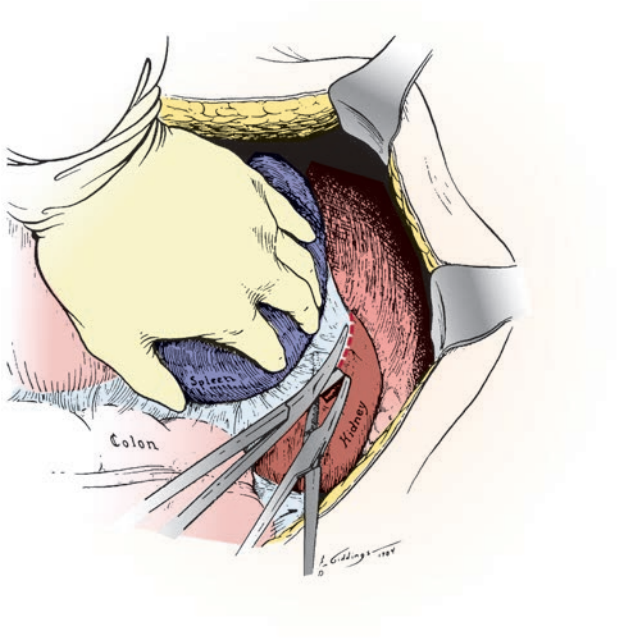


FIGURE 34-8 Mobilization of the spleen is begun by early division of its lateral attachments.

tail of pancreas and kidney. If the tail of the pancreas is not mobilized with the spleen, the degree of splenic mobilization possible is much more limited and it is more difficult to avoid injury to the spleen.

Injuries can occur during mobilization of the spleen. The splenic hilum can be damaged from behind as the surgeon's fingers attempt mobilization from lateral to medial. The pancreas is more difficult to see if it is not mobilized with the spleen and can be damaged during hilar clamping if the spleen is to be removed. The pancreas is quite variable in length and requires varying degrees of mobilization. If the pancreas is fairly long, a great deal of its body and tail will require mobilization in order to bring the spleen anteriorly and to the midline.

After the spleen and pancreas have been mobilized as a unit, it is generally apparent that the next constraining attachments of the spleen are the short gastric vessels. Because of the dual blood supply of the spleen through its hilum and through the short gastric vessels, it is possible to divide the short gastric vessels without compromising splenic viability. The best way to divide the short gastric vessels is to have an assistant elevate the spleen and tail of the pancreas into the operative field and then to securely clamp the vessels starting proximally on the greater curvature of the stomach. The short gastric vessels should always be clamped and tied. They can be small and difficult to see, and it is tempting to simply divide the loose tissue between the spleen and stomach with the scissors or electrocautery. This should not be done because the short gastric vessels can then bleed either immediately or on a delayed basis. It is not uncommon for a clamp to encroach on the gastric portion of a short gastric vessel, including a small portion of stomach. In such cases, the tie on the stomach can necrose the wall, leading to a delayed gastric leak. Care should be taken to avoid this situation, but this concern can be addressed by oversewing the short gastric tie on the stomach side with a series of Lembert sutures in the seromuscular layer of the stomach.

The final step necessary for full mobilization of the spleen is division of the splenocolic ligament between the lower pole of the spleen and the distal transverse colon and splenic flexure. Obvious vessels in this ligament should be divided between clamps. During division of both the short gastric vessels and the splenocolic ligament, bleeding from the spleen can be controlled using digital compression of the hilum. If the patient is exsanguinating and the bleeding is massive, occasionally a clamp can be placed on the hilum during the later steps of mobilization. Mass clamping should only be done in extreme circumstances because it increases the chances of injury to the tail of the pancreas.

After the spleen has been fully mobilized, it is possible to inspect it in its entirety and to examine the posterior aspect of the body and tail of the pancreas as well. It is helpful after mobilization to pack the splenic fossa to tamponade any minor bleeding and to help keep the spleen and distal pancreas elevated into the field. During this packing maneuver, the left adrenal gland can be inspected and the left hemidiaphragm reexamined.

Factors that figure into the decision about what to do with the injured spleen after mobilization include the degree of splenic injury, the overall condition of the patient, and the presence of any other intra-abdominal injuries. Obviously, if the spleen is not injured at all, it should be left in place. Similarly, if there is a trivial injury to the spleen and it is not bleeding, the spleen can be simply returned to the left upper quadrant and no further therapy is necessary. If there is a grade I injury of the spleen that is bleeding minimally or not bleeding at all, hemostatic agents can be used to stop the bleeding or prevent future bleeding. A variety of hemostatic agents are available, including microfibrillar collagen, gelatin sponge, and fibrin glue. Whichever agent is chosen, the bleeding from the spleen should have ceased by the time the patient is closed.

If the injury is more severe (grades II and III) and the patient's overall condition is not too serious, splenorrhaphy can be done.[53] Splenorrhaphy has become much less common with the increasing use of nonoperative management. Because we are no longer operating as much on the spleen, especially for lower grades of splenic injury, the number of splenic injuries found at surgical intervention that are amenable to splenorrhaphy has decreased along with experience with the techniques. The simplest version of splenorrhaphy has already been described earlier and is the placement of topical agents. Electrocautery of the spleen is only rarely helpful and has met with limited success, whereas argon beam coagulators may be helpful for hemostasis, especially of parenchyma that has been denuded of splenic capsule.[54,55] The spleen can also be sutured, especially when there is an intact capsule, but it does not hold sutures particularly well. Therefore, it is often necessary to use pledget materials to bolster

the repair. Several different methods for suturing the spleen have been described, and use of monofilament or chromic suture has some advantages in that either is less likely to cause injury while being placed through the splenic parenchyma. The splenic parenchyma is fairly soft even in the presence of an intact capsule, and it is easy to cinch sutures so tightly that the parenchyma is further disrupted.

Partial splenectomy also has been described and is possible because of the segmental nature of the splenic blood supply. A pole or even half of the spleen can be removed, and the remaining spleen will survive provided that its hilar blood supply is left intact. One method of performing partial splenectomy is to ligate the blood supply to the damaged portion of the spleen and then observe the spleen for its demarcation into viable and nonviable portions. The damaged nonviable portion is removed, and the resultant cut splenic parenchyma is made hemostatic with the use of either mattress sutures or mesh wrapping.

Wrapping of either all or part of an injured spleen with absorbable mesh has been used on occasion as well. This technique is moderately time consuming, but reported success rates are high because of careful patient selection. Such an approach should be reserved for highly selected cases of isolated splenic injury in stable patients.

Splenectomy should be performed in patients who are unstable, have serious associated injuries, or have the higher grades of injury (grade IV or V). Bleeding from the splenic parenchyma can be temporarily controlled with digital pressure on the hilum while the spleen is being mobilized. As previously noted, mass clamping of the hilum should be reserved for profoundly hypotensive patients because it increases the risk of damage to the adjacent tail of the pancreas. If the decision has been made to remove the spleen, this is best done with serial dissection and division of the hilar structures after mobilization. Suture ligation should be used for large vessels. As mentioned in the section "Splenic Anatomy," a number of different splenic arterial and venous branches must be divided before removal of the spleen (Fig. 34-3). During the course of this dissection, it is common to encounter accessory spleens in the hilum. If an accessory spleen is encountered, it should be left in place if possible.

A special circumstance is the patient who has failed nonoperative management. The majority of these patients undergo splenectomy rather than splenorrhaphy. One reason is that the spleen is somewhat softer after a period of nonoperative management than it was before injury, and both mobilization of the spleen and splenorrhaphy are more difficult. Also, it is likely that splenic injuries that have failed nonoperative management are worse than injuries that do not fail nonoperative management. Another important factor is that the surgeon operating on a spleen that has failed nonoperative management has already decided that the spleen is a problem and is psychologically prepared for splenectomy at the time of operation. The worst-case scenario for such a surgeon is to perform splenorrhaphy after nonoperative management and have it fail, in which case the patient would require yet another trip to the OR.

As has been mentioned earlier, it is helpful to pack the splenic bed during the latter stages of splenic mobilization and during splenectomy. After the spleen has been removed, the packs in the left upper quadrant should be removed and the splenic fossa reexamined. Inspection of the splenic fossa is facilitated by using a rolled-up laparotomy pad. The laparotomy pad is placed deep in the splenic fossa and then rolled by the surgeon's fingers up toward the cut vessels at the splenic hilum. During the course of this inspection, it is important to carefully visualize the splenic bed, stumps of the splenic vessels, and stumps of the short gastric vessels along the greater curvature of the stomach. This is because postoperative hemorrhage after splenectomy is most commonly related to bleeding from the cut ends of the short gastric vessels.

Splenic tissue has a remarkable ability to survive in ectopic locations even without a clearly identifiable blood supply. Greater or lesser degrees of spontaneous splenosis after splenectomy for trauma are quite common, and patients with splenosis demonstrate some degree of splenic function after splenectomy.[56,57] The observation that accidentally seeded pieces of splenic tissue could survive and function led to the logical suggestion that portions of the spleen could be intentionally autotransplanted to ectopic sites after splenectomy. Studies of autotransplantation in both animals and humans have demonstrated that some of the splenic tissue survives and has some level of function.[58,59] Whether or not enough of it survives without attachment to the splenic artery in an adequately functioning form to provide adequate protection against postsplenectomy sepsis is an open question.[60,61] Reports of overwhelming infection after autotransplantation suggest that autotransplantation is not universally successful in restoring normal immune function.[62] These observations, combined with the relatively time-intensive technique of autoimplantation and the exceedingly low rate of overwhelming postsplenectomy infection, have rendered this practice largely of historical interest.

Drains should not be routinely placed after either splenectomy or splenorrhaphy unless there is concern for an associated pancreatic injury or an associated renal injury if there is concern about a postoperative urine leak.

COMPLICATIONS

Nonoperative Management

The most common complication of nonoperative management of the spleen is continued bleeding. Many cases of the bleeding are probably just persistent bleeding that never stopped after the original injury. In these circumstances, there is hemodynamic instability or a progressive drop in hematocrit. Although about 60% to 70% of the failures of nonoperative management occur early after admission, some occur on a delayed basis, and approximately 10% occur more than 1 week after injury.[46] A recent prospective observational study by Zarzaur et al[63] of 383 patients reported a 0.27% incidence of outpatient bleeding and subsequent splenectomy.

Early failures of nonoperative management can be determined by closely following the patient's hemodynamic status,

hematocrit, and physical examination. In many patients, drop in hematocrit will be gradual and steady, but will ultimately dictate the need for surgical intervention. In other patients, especially those in whom the bleeding is delayed, bleeding can occur rather suddenly and be fairly dramatic. If an emergency operation is not performed in such cases, the patient is at risk for exsanguination. The pathophysiology of persistent bleeding after splenic injury and early failure of nonoperative management is fairly straightforward. The pathophysiology of the more delayed bleeds is less obvious, and there are several hypotheses on why it occurs. One hypothesis is that as the blood in a subcapsular hematoma breaks down, increased osmotic forces pull water into the hematoma and expand the capsule. A similar pathophysiology has been described as an explanation for the increase in size of subdural hematomas. Another hypothesis for delayed bleeding from a splenic injury is the concept of "remodeling" of the clot in the splenic parenchyma. This hypothesis is based on the observation that the clot undergoes revision and degradation over time. It is possible that as this remodeling process occurs, the initial hemostasis of the splenic injury is lost. The observation that splenic injury can result in intraparenchymal pseudoaneurysms raises the possibility that delayed bleeding could be the result of rupture of a pseudoaneurysm. Finally, it is simply possible that the damaged spleen, highly vulnerable to further injury, suffers what would otherwise be a minor second blow and starts to bleed again. The failure rate for nonoperative management varies due in part to the lack of a standardized definition of failure. Some surgeons and institutions have a low threshold for operative intervention after an attempt at nonoperative management, and some have a very high threshold. When nonoperative management has failed and the patient requires operative intervention, splenectomy is most often the appropriate operation unless there is minimal concern about subsequent bleeding.

Another potential complication of nonoperative management of splenic injuries is that an associated intra-abdominal injury that requires operative intervention will be missed.[64-66] This is most commonly a problem for missed injuries of the bowel and pancreas. Injury to the small bowel is particularly troublesome, as often free fluid is the only finding of blunt intestinal injury seen on CT of the abdomen. When splenic injury is present, it is easy to attribute the free fluid to bleeding from the spleen. If patients are good candidates for nonoperative management of their splenic injury, it is possible to miss the bowel injury and delay needed abdominal exploration. Pancreatic injuries are occasionally missed on initial CT scanning done shortly after injury and can result in serious morbidity or even mortality if not treated in an expeditious fashion. The proximity of the tail of the pancreas to the spleen makes the combination of injuries to the two organs a possibility. Repeated physical examinations, measurement of pancreatic enzymes, and repeat abdominal CT scanning are all helpful in minimizing the number of missed injuries to the small bowel and pancreas when signs or symptoms justify.

Failure of nonoperative management is not without negative consequences. In a multicenter study, 10 (13%) of 78 patients who failed nonoperative management died, with most of the deaths related to delayed treatment of intra-abdominal injuries. A significant number of the cases of failed nonoperative management could be traced to an inappropriate initial decision to proceed with nonoperative management in hemodynamically unstable patients and/or when there was misinterpretation of the diagnostic imaging studies.[47]

There are no other abdominal complications specific to nonoperative management of the spleen, but intrathoracic complications can occur. Associated pleural sympathetic effusions may result from blood and clot beneath the left hemidiaphragm, whereas a hemothorax may be caused by bleeding from associated fractured ribs.

DVT in the lower extremity is another potential complication after nonoperative management of a splenic injury because prophylaxis is sometimes delayed, as previously described. There is no firm evidence that the rate of thromboembolic complications is higher in patients with nonoperative management of a splenic injury. When a patient with a splenic injury develops a DVT or pulmonary embolism, it can be difficult to decide when and how to proceed with treatment. Anticoagulation of the patient puts the injured spleen at risk, while placement of a caval filter is invasive and expensive. Such patients should be managed on a case-by-case basis, particularly regarding whether the thromboembolic complication has clinical manifestations or was discovered incidentally. Fortunately, these cases are rare in that most clinically apparent thromboembolic problems will not manifest themselves until after the major risk of bleeding from the injured spleen has passed.

Although angioembolization has been used to diagnose and control splenic bleeds since the 1960s, the earliest report of angioembolization to control bleeding specifically for traumatic injury was in in 1981.[67] The increased use of selective angioembolization has led to recognition of a number of complications unique to this modality. Major complications include continued splenic hemorrhage requiring splenectomy and contrast-induced nephropathy. Minor complications include splenic infarction or abscess, groin hematoma or infection, coil migration, and reactive left pleural effusions. When comparing angioembolization proximal or distal to the main splenic trunk, distal embolizations have a higher incidence of minor complications. Although it appears that the overall rate is low, there is still much to be learned about complications from angioembolization.[68,69]

Patients who are managed nonoperatively often receive blood products secondary to the splenic injury or because of associated injuries. There are well-known risks associated with transfusion. These include the risk of blood incompatibility, transmission of bloodborne diseases such as hepatitis, and the significant immunologic effects of transfusion, especially in critically ill and injured patients.[70,71]

Operative Management

There is a risk of bleeding after splenectomy from the short gastric vessels or splenic bed and after splenorrhaphy from the splenic parenchyma. As after any operative procedure, it

is important to closely follow the patient and to reexplore if postoperative bleeding is suspected. Patients with multiple associated injuries and a coagulopathy generally should have undergone splenectomy rather than splenorrhaphy. In these patients, the coagulopathy will be treated, but the possibility of surgical bleeding in the postoperative period should always be entertained when the patient has hemodynamic instability. In patients who have undergone splenorrhaphy, the risk of continued bleeding from the repaired spleen is low.

Gastric distention is a risk, because theoretically when the short gastric vessels have been cut and ligated, gastric distention can result in loss of a tie on the gastric end of a vessel with resultant hemorrhage. Even though this danger may be more theoretical than real, a short period of gastric decompression is practiced by some surgeons.

As previously noted, necrosis of a portion of the greater curvature of the stomach has been described, most commonly related to inclusion of a portion of the gastric wall in the ties placed on the gastric side of the cut short gastric vessels. The resultant gastric leak contaminates the abdomen, in particular the left upper quadrant, and can lead to the formation of a subphrenic abscess or generalized peritonitis.

Pancreatic injuries can be related either to the original trauma or to an iatrogenic injury during mobilization or removal of the spleen. A pancreatic injury will cause an increase in pancreatic enzymes, an ileus, and a generalized inflammatory state. The diagnosis is made from a combination of clinical and CT findings.

The rare complication of an arteriovenous fistula in the ligated vessels in the hilum of the spleen has also been described as a risk of splenectomy. The best way to avoid such a complication is to avoid mass ligation of the hilar structures.

There is some evidence that the long-term risk of thrombotic events is increased after splenectomy for trauma.[72,73] Clinically significant thrombocytosis, however, is still less common after a splenectomy for trauma than it is after a splenectomy for other diseases. As previously noted, appropriately timed prophylaxis should be a standard measure in all injured patients and should cover the risks associated with the transient postsplenectomy thrombocytosis.

There is some evidence that early postoperative complications are more common after splenectomy than they are in patients who do not have their spleens removed.[74,75] Evidence is conflicting, however, and a difficulty in reviewing the literature on the subject is that it is hard to standardize the severity of injury in patients who have undergone splenectomy as compared to patients who have not undergone splenectomy. Some of the series that have suggested an increased risk of complications after splenectomy have noted that such patients were more severely injured than those who did not undergo a splenectomy.

OVERWHELMING POSTSPLENECTOMY INFECTION

The first experimental evidence supporting the possibility that the spleen is of immunologic importance dates to 1919; however, splenectomy remained the treatment of choice for both iatrogenic and traumatic splenic injuries until just several decades ago.[76] In the early 1950s, it was noticed that neonates and young children (up to 6 months of age) with hematologic diseases who required a splenectomy had a high subsequent risk of serious infection. It became clear that an asplenic state in neonates and young children with hematologic diseases was a risk factor for overwhelming infection. From this observation, it was a logical next step to investigate the risk of overwhelming infection in both children and adults who had undergone splenectomy for trauma.[77-79] Several studies suggested that the rate of overwhelming infection after splenectomy is increased when compared with a control population of patients who have not had their spleens removed. The actual rate at which overwhelming infection in asplenic patients occurs is unknown. One estimate is a 0.026 lifetime risk for adults and a 0.052 lifetime risk for children, but all the estimates of risk tend to be very low. Not all studies have documented an increased risk of overwhelming life-threatening infection after splenectomy. One single-institution study reviewed 18 years of splenectomy patients and identified no incidents of overwhelming postsplenectomy infection.[80] Therefore, the clinically significant risk is very low and probably does not merit much consideration when considering the most appropriate treatment of an adult patient with a splenic injury.

When infection does occur in the asplenic state, encapsulated organisms such as pneumococcus and meningococcus are the most common pathogens and pneumonia and meningitis are the most common infections. Because of the inference that overwhelming infection is more common after splenectomy, vaccines to prevent infection by pneumococcus, meningococcus, or *Haemophilus* organisms are recommended for splenectomized patients. There is empirical evidence in both animals and humans that the use of vaccines results in an antibody response; however, because the incidence of overwhelming infection after splenectomy is very low, it is difficult to prove that the vaccines actually have an impact on postsplenectomy infection and mortality. Nonetheless, they have become the standard of care in patients who have had a splenectomy. In patients who have undergone splenectomy, the exact timing of vaccination is somewhat controversial, but evidence from a randomized trial suggests that postsplenectomy trauma patients vaccinated in a delayed fashion (14 days postoperatively) have improved functional antibody responses compared to those vaccinated at 1 day or 7 days after splenectomy.[81-83] As with the question of the overall effectiveness of vaccines in preventing postsplenectomy infection, study of the optimal timing of vaccination is hampered by its low incidence. The most important principle of vaccination after splenectomy is to remember to perform it before discharge from the hospital in patients who are unlikely to return for postoperative follow-up. Whether or not patients should be revaccinated and when such revaccinations should be done remain open questions. One recommendation based on longitudinal antibody studies in a general group of patients (not just trauma postsplenectomy patients) is for revaccination every 6 years.

Another measure that has been suggested for postsplenectomy patients is the continuous administration of antibiotics or the provision of a supply of antibiotics to be taken at the first sign of infection. When such measures have been tried, studies of patients' compliance with the antibiotic regimen have been discouraging.[84] The exact role of antibiotics in postsplenectomy patients is difficult to ascertain for the same reason that the effectiveness of the vaccines is difficult to prove and is not routinely recommended in adults.

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35

Stomach and Small Bowel

Ronald M. Stewart • David H. Livingston

KEY POINTS

- The normal stomach is relatively free of bacteria and other microorganisms because of the low intraluminal pH.
- The thick-walled stomach is relatively resistant to blunt rupture except when it is full and the crash has high kinetic energy or there has been inappropriate placement of a lap seat belt.
- The presence of an anterior penetrating gastric wound requires a thorough exploration of the lesser sac to identify the posterior wound.
- A suspected "missing" gastric perforation from a gunshot wound mandates a provocative test by insufflating and distending the stomach with either air or saline down the nasogastric tube.
- Because the stomach has an excellent blood supply, two-layer suture closures are performed to close perforations and injuries.
- Hollow viscus injuries present in 1% to 3% of all blunt trauma patients, with small bowel injuries accounting for 90% of these injuries.
- A transverse seat belt contusion ("seat belt sign") across the abdominal wall carries an increased risk of injury to the small bowel and mesentery.
- Modern computed tomography (CT) scans have improved the ability to diagnose injuries to the small bowel but can still be normal in 4% of patients with documented blunt rupture.
- Free fluid on CT scanning without solid organ injury does not mandate laparotomy but does mandate close observation and serial examination.
- Blunt ruptures of the small bowel, most often related to lap seat belts, most commonly occur near the ligament of Treitz or ileocecal valve or at the site of adhesions.
- Delays in operation in patients with ruptures of the small bowel and stomach are associated with increased complications and death.
- In patients with multiple small bowel injuries, the overarching concept is to leave as much bowel as possible with the smallest number of repairs and anastomoses.
- The complication rate is similar for stapled and handsewn small bowel anastomosis.
- Closure of an enterocutaneous fistula from prior repair of the small bowel is best accomplished with resection and anastomosis.

INTRODUCTION

Injuries to the stomach and small bowel are common following penetrating abdominal trauma. The incidence of gastrointestinal injury following gunshot wounds and stab wounds that penetrate the peritoneal cavity is in excess of 80% and 30%, respectively. Blunt injuries to the stomach and small bowel are much less common than penetrating injuries, but collectively compose the third most common type of blunt abdominal hollow viscus injuries (HVIs). Given these data, the trauma surgeon must be able to diagnose and treat these potentially life-threatening injuries.

The operative repair of injuries to the stomach and small bowel is relatively straightforward. The key to the successful management of stomach and small bowel injuries is prompt recognition and treatment, thus decreasing the likelihood of abdominal septic complications, including anastomotic leaks, fistulas, and intra-abdominal abscesses.

HISTORICAL PERSPECTIVE

Intestinal injuries were reported early in the medical literature, and small bowel perforation from blunt trauma was first reported by Aristotle.[1] Hippocrates was the first to report

intestinal perforation from penetrating abdominal trauma. In 1275, Guillaume de Salicet described the successful suture repair of a tangential intestinal wound. Reports of attempted surgical repair of gastric and intestinal wounds appeared in the literature during the American Civil War, the Spanish-American War, the Russo-Japanese War, and other military conflicts. The dismal results of surgical intervention led to abandonment of laparotomy even with obvious intestinal injury during these military campaigns.[2]

By the late 19th century, improved anesthesia and surgical techniques led to renewed interest in laparotomy and repair of penetrating abdominal injuries. Theodore Kocher was the first surgeon to report successful repair of a gunshot wound of the stomach. Dr. George Goodfellow is credited with the first laparotomy to treat a gunshot wound.[3] Dr. Goodfellow was a follower of Lister and sterilized his instruments and his hands. The patient's intestines were covered with a large amount of "purulent stinking lymph" as the victim had six holes in his intestines.

A laparotomy for intestinal perforation at the start of World War I carried a mortality rate of 75% to 80%, almost equal to the mortality rate of nonoperative management. In the latter part of World War I, operative management was recognized as the preferred management for penetrating abdominal trauma.

In World War II, prompt evacuation, improvements in anesthesia, and better understanding and treatment of shock led to mortality rates of 13.9% for jejunal or ileal injuries and 36.3% if multiple injuries were present.[4] Further improvements in mortality were noted during the Korean and Vietnam Wars. Earlier evacuation and improvements in resuscitation combined with prompt recognition and repair of injuries are responsible for the current low morbidity and mortality following injuries to the stomach and small bowel.

ANATOMY

The nondistended stomach, especially in a supine individual, is located largely in the intrathoracic abdomen, where it is offered some protection by the lower chest wall. The position of the stomach can be quite variable and, in the erect individual, may extend into the lower abdomen, particularly when distended with food or liquid. The stomach is fixed along the lesser curvature by the gastrohepatic ligament, cephalad by the gastrophrenic ligament, and distally by the retroperitoneal duodenum. The greater curvature of the stomach is loosely bound to the transverse colon via the greater omentum and to the spleen by the gastrosplenic ligament. The stomach enjoys a rich blood supply from the left and right gastric arteries, the left and right gastroepiploic arteries, and the short gastric arteries (Fig. 35-1).

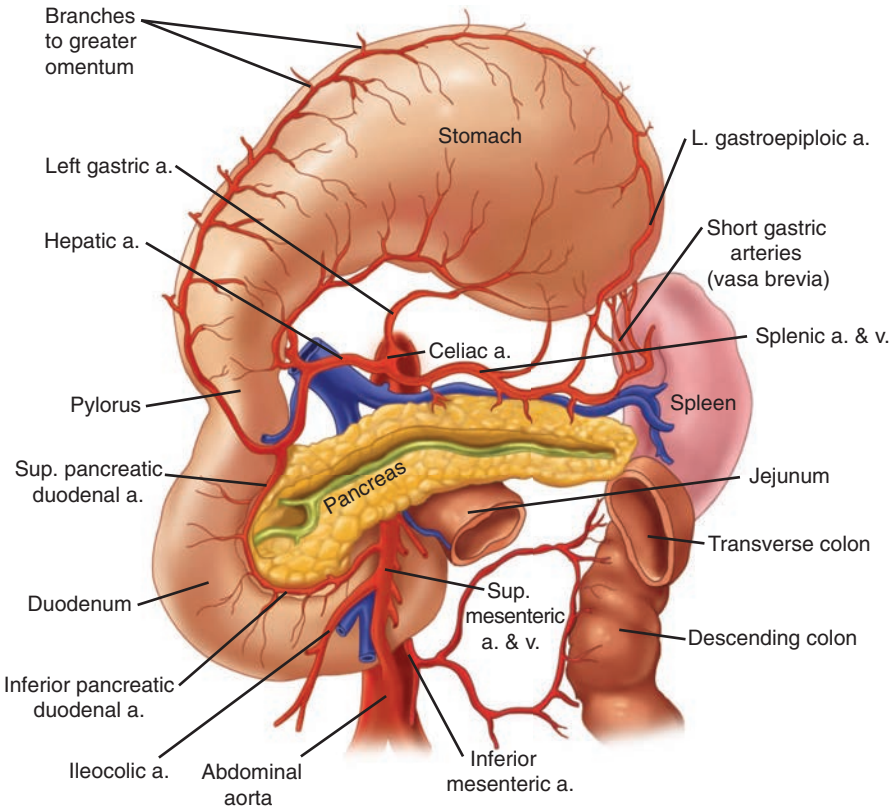


FIGURE 35-1 Blood supply to the stomach. An anomalous left hepatic artery can arise as a branch of the left gastric artery. This should be looked for when doing gastric resections. a., artery; v., vein. (Reproduced with permission from Mercer DW, Liu TH, Castaneda A. Anatomy and physiology of the stomach. In: Zuidema GD, Yeo CJ, eds. *Shackleford's Surgery of the Alimentary Tract*. 5th ed., Vol II. Philadelphia, PA: Saunders; 2002:3. Copyright © Elsevier.)

Venous drainage follows the arterial supply to the stomach for the most part.

The normal stomach is relatively free of bacteria and other microorganisms because of the low intraluminal pH; however, up to 10^3 organisms/mL, including lactobacilli, aerobic streptococci, and even *Candida*, may be isolated. Increasing gastric pH, due to chronic use of antacids, H_2-receptor blockade, or proton pump inhibitors, will lead to increased bacterial concentrations in the stomach and proximal gastrointestinal tract.[5] This increases the risk of peritoneal contamination with gastric perforation. Because trauma patients often have full stomachs, spillage of retained food will also increase the risk of infection following gastric injury.

The small bowel distal to the ligament of Treitz is approximately 5 to 6 m in length in the adult. Protected anteriorly only by the abdominal wall musculature and occupying most of the true abdominal cavity, the small intestine is anatomically vulnerable to injury. The small intestine is suspended from the retroperitoneum by its mesentery, the base of which extends from the duodenal-jejunal flexure, superior to inferior and left to right, to the level of the right sacroiliac joint. The arterial supply to the small bowel is provided by the superior mesenteric artery (SMA), which emerges from under the pancreas and then courses anterior to enter the root of the mesentery. The blood supply to the majority of the small bowel comes from the left side of the SMA via intestinal arteries (Fig. 35-2). The jejunal and ileal branches vary in number and lead to numerous intestinal arcades, which ensure excellent collateral blood supply. The ileocolic artery is the termination of the SMA. Venous return from the small

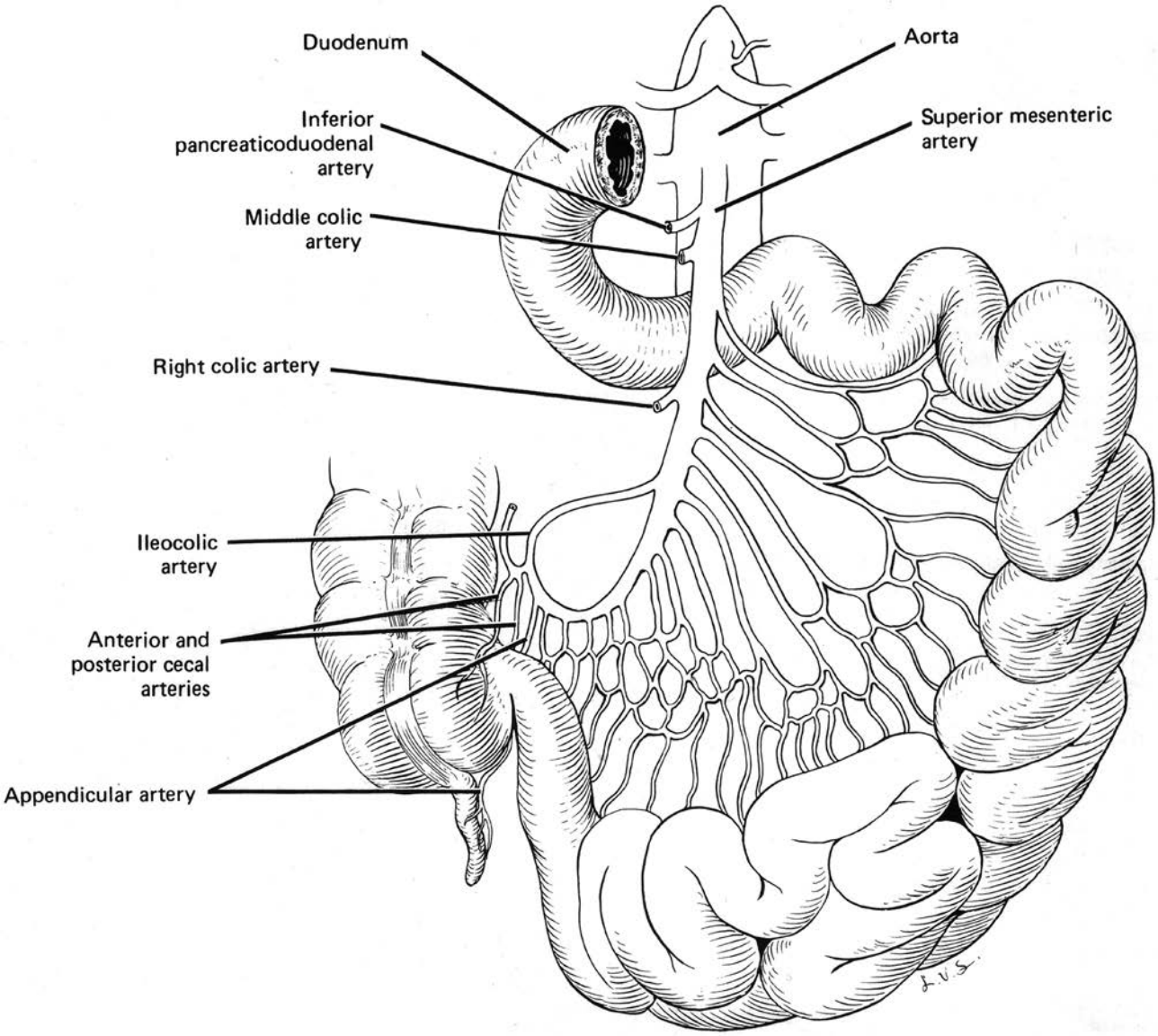


FIGURE 35-2 Blood supply to the small bowel. Multiple branches to the jejunum and ileum originate directly from the superior mesenteric artery. The distal ileum is supplied via the ileocolic artery. (Reproduced with permission from Lindner HH, ed. *Clinical Anatomy*. San Mateo, CA: Appleton & Lange; 1989. Copyright © The McGraw-Hill Companies, Inc.)

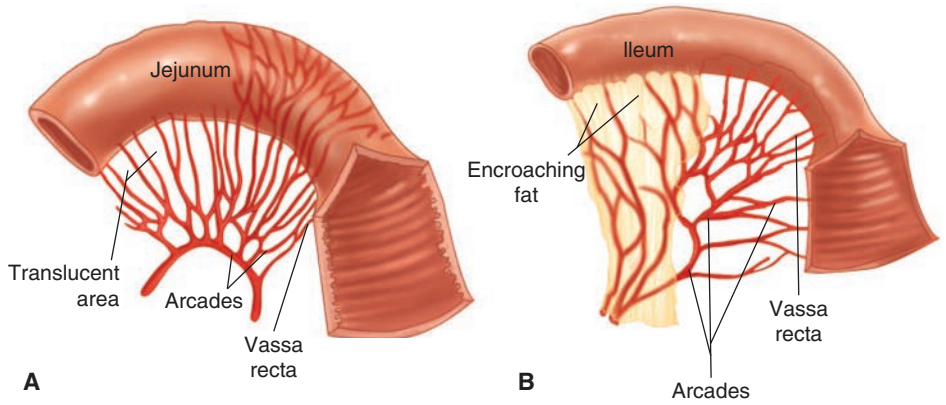


FIGURE 35-3 The jejunum and ileum can be distinguished from one another by differences in luminal diameter, number of arterial arcades, and the presence or absence of fat encroaching on the gut wall. (Modified with permission from Tavakkoli A, Ashley SW, Zinner MJ. Small intestine. In: Brunicardi FC, Andersen DK, Billiar TR, et al, eds. *Schwartz's Principles of Surgery,* 11th ed. New York, NY: McGraw Hill; 2019.)

intestine follows the arterial supply. The inferior mesenteric vein runs lateral to the ligament of Treitz to drain into the splenic vein, which combines with the superior mesenteric vein to form the portal vein.

FUNCTION

Although no clear distinction exists, the first 40% or so of the bowel is jejunum and the remainder is the ileum (Fig. 35-3). The jejunum has a larger diameter, more circular folds, and larger villi, but less lymphoid tissue than the ileum. The mesentery of the jejunum contains only a single arcade, whereas more than two or three sets of vascular arcades are present in the ileum. Mesenteric fat is also more prominent in the ileum than in the jejunum. The proximal jejunum is the primary site of absorption of carbohydrate, protein, and water-soluble vitamin absorption. Fat absorption occurs over a larger length of small bowel. The ileum is the primary site of carrier-mediated bile salt and vitamin B_{12} absorption. Distinctions between jejunum and ileum are of clinical importance only if a significant length of bowel is resected. The ileocecal valve is thought to act as a "brake" to the delivery of small bowel content into the cecum. It may also be a barrier for reflux of colonic content into the small bowel. Ileal peristalsis probably is the main factor in those functions.

The luminal content of the proximal small bowel is of neutral pH and is relatively sterile, containing few bacteria. Most studies of the small bowel microflora have demonstrated increasing bacterial counts with distance away from the pylorus.[6] The proximal small bowel flora resembles the gastric flora, and alterations in the bacterial load of the stomach will influence the bacteriology of the small bowel. The jejunum and proximal ileum contain gram-positive and gram-negative organisms at 10^4 to 10^5 colony-forming units (CFU)/mL. The bacterial concentration in the distal ileum rises to 10^5 to 10^8. There is also a higher number of anaerobic species in the ileum. This increase in bacterial load in the ileum is thought to contribute to an increased risk of infection with full-thickness injury in the distal small bowel versus the proximal small bowel. Again, because trauma patients have not been fasting, the risk of an increased volume of luminal contents resulting in a greater degree of intestinal spillage containing bacteria likely plays an important role.

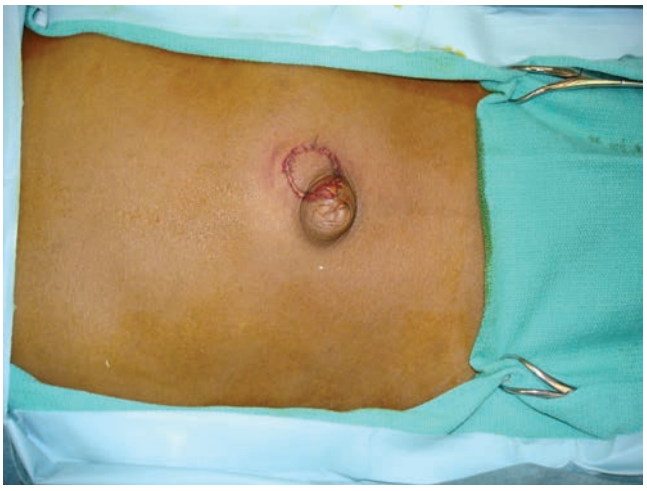
DIAGNOSIS

Following penetrating trauma, the diagnosis of injuries to the stomach and small bowel is almost always made during an exploratory laparotomy. The small bowel is at risk of perforation following virtually any penetrating injury that violates the peritoneum. Patients who present with hemodynamic instability or evisceration of abdominal contents after penetrating abdominal trauma obviously need an emergent laparotomy. Bloody nasogastric aspirate may be indicative of gastric injury, and free air demonstrated on an upright chest x-ray should be presumed to be from some HVI after blunt trauma.

Blunt HVIs are infrequently encountered. In the multi-institutional study from the Eastern Association for the Surgery of Trauma (EAST), HVIs were found in 1.2% to 3.1% (depending on the cohort denominator used) of admissions during the 2-year study period, and 90% of these were in the small bowel.[7]

Blunt gastric injuries are extremely rare and usually related to excessive force such as a pedestrian struck by a motor vehicle or a high-speed motor vehicle crash. The stomach has a thick wall and is relatively resistant to a blunt injury; however, contributing factors include a full stomach, trauma to the left side of the body, and the inappropriate use of a seat belt. Blunt gastric ruptures most frequently involve the anterior gastric wall.[8,9] Peritoneal signs and a bloody nasogastric tube aspirate are usually present and are diagnostic. Associated injuries to the liver, spleen, and pancreas, as well as injuries to the chest and head, are common due to the degree of force necessary to produce a gastric blowout.[10]

A small bowel perforation secondary to blunt abdominal trauma is uncommon, although 90% of all HVIs occur in the small bowel, as previously noted. Motor vehicle crashes are



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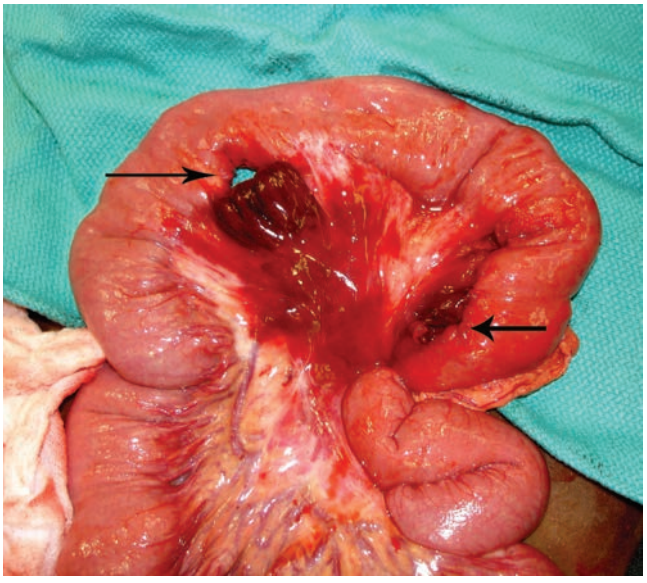


B

FIGURE 35-4 Direct blows to the abdominal wall following a bicycle crash are a classic mechanism of blunt small bowel injury in children. (**A**) Abdominal wall imprint of the handlebars. (**B**) Two full-thickness injuries (arrows) and the associated mesenteric hematoma. These injuries were treated by a single resection.

the most important mechanism for blunt intestinal trauma, followed by falls, bicycle accidents (Fig. 35-4), or a localized blow. Mechanisms postulated for injury to the small intestine to occur include the following: (1) crushing of bowel against the spine; (2) shearing of the bowel from its mesentery of a fixed point by sudden deceleration; and (3) bursting of a "pseudo-closed" loop of bowel due to a sudden increase in intraluminal pressure. Garret and Braunstein first referred to the seat belt mark as ecchymoses across the abdominal wall that corresponds to the lap belt (Fig. 35-5).[11] With the advent of the three-point restraint system, restraint injuries may also involve the neck and chest. Depending on the severity of the blunt force trauma, "seat belt syndrome" may include HVI, abdominal wall disruption, associated lumbar fractures, and a major abdominal vascular injury (see Chapter 38).[12]

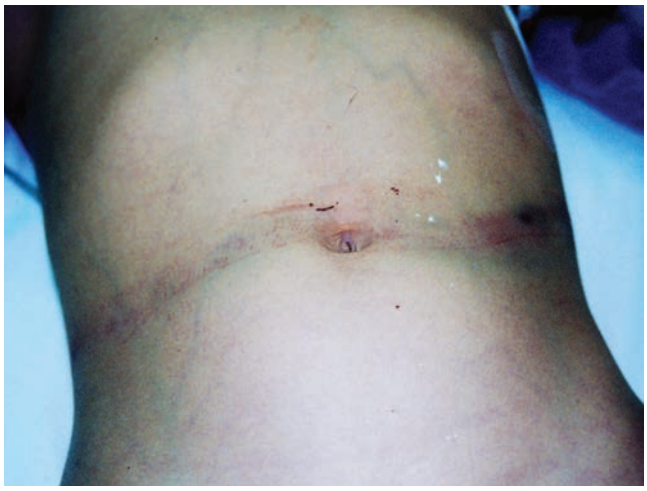
Anderson et al[13] reported a 4.38-fold increase in risk of small bowel injuries with use of a lap/shoulder restraint and a more than 10-fold increase in risk with lap belts alone, compared with no restraint use. In an EAST multi-institutional study, the seat belt sign (SBS) was associated with a 4.7-fold increase in relative risk of small bowel perforation in patients following motor vehicle crashes.[14] Because the degree of force transmitted against the torso is unknown in the vast majority of blunt trauma patients, the presence of an SBS or handlebar bruise is simply a clear indication that there has been a significant impact to the abdominal wall. Thus, it should be no surprise that the presence of an HVI is significantly higher in this subpopulation than in the population at large.

Children with an SBS also have a higher rate of gastrointestinal injury, especially given the poor fit of seat belts in children. Sokolove et al[15] demonstrated that children with an SBS had a significantly greater risk of intra-abdominal injury, including gastrointestinal and pancreatic injuries. The increased risk of injury was only apparent in patients with abdominal pain or tenderness. In a study by Chidester et al,[16] the SBS only had a sensitivity of 25% and a specificity of 85% for abdominal injury. Similar to the study by Sokolove et al, the presence of SBS with abdominal tenderness was more predictive of abdominal injury.

The contemporary view of the seat belt syndrome is a constellation of injuries, some of which may be subtle in presentation and appearance.[17] As noted earlier, the seat belt syndrome is more common in children and associated with the use of a lap belt only, most often because the child is not properly restrained in a booster seat. The full manifestations of the seat belt syndrome from anterior to posterior involve injury to the skin, subcutaneous tissue, abdominal wall musculature, small bowel, small bowel mesentery, sigmoid colon and mesentery, cecum, aorta, proximal iliac arteries, and spine (Fig. 35-5). When there is a seat belt contusion, particularly above the level of the iliac crests, an intentional and careful search for injuries to the sigmoid colon, aorta, and iliac vessels and spine should be undertaken.

The association of a Chance-type fracture of the lumbar spine as an independent predictor of HVI is variably reported in the literature (Fig. 35-5). In the EAST multi-institutional trial with small bowel perforations, there was no difference in the incidence of Chance-type fracture in patients with perforations of the small bowel compared with those without perforations.[14] The incidence of bowel perforations ranged from 2% to 3% of patients. Similar to SBS, the presence of severe fractures of the thoracolumbar spine is evidence that the torso has sustained a significant degree of blunt force trauma. Because almost all spine fractures are now diagnosed by computed tomography (CT) scanning, the identification of associated intra-abdominal and intrathoracic injuries should no longer be a question.

CT scanning is the primary diagnostic modality in hemodynamically stable patients following blunt trauma. Although a focused assessment with sonography in trauma (exam) may



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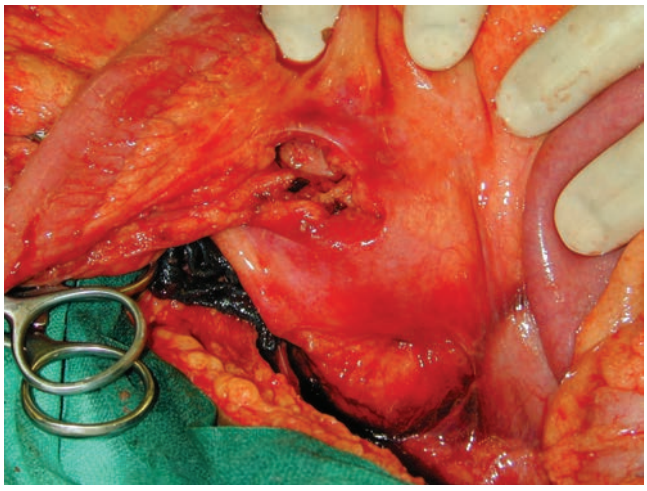

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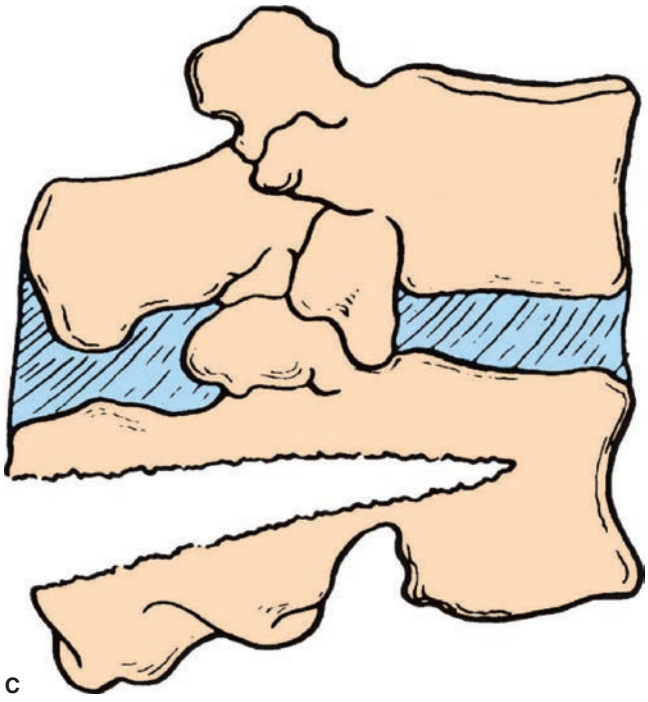

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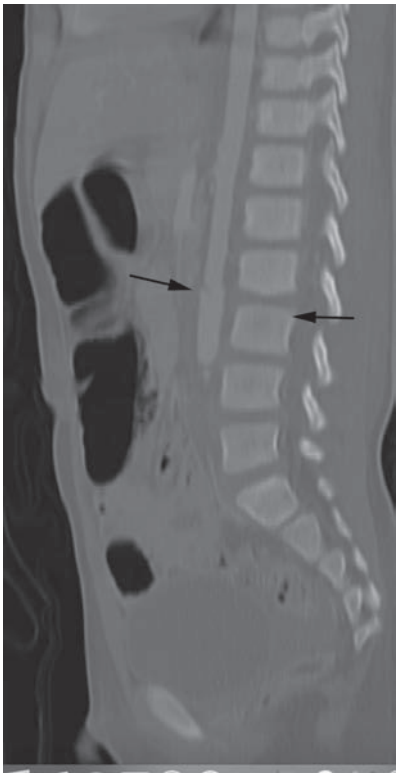
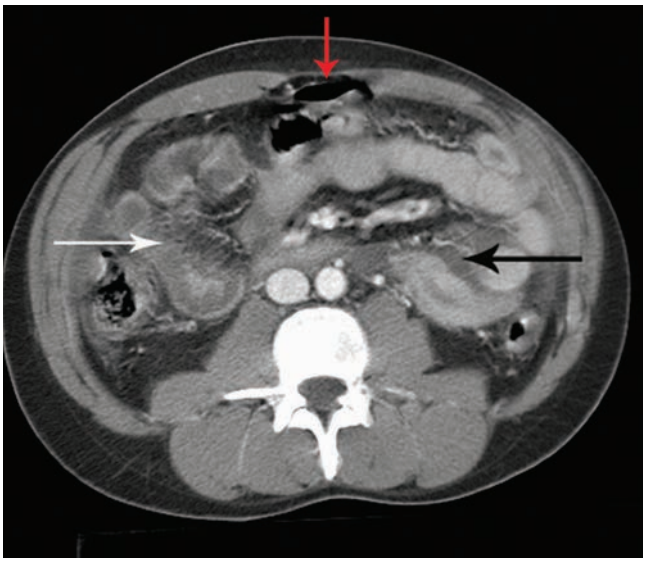


D

FIGURE 35-5 The "seat belt syndrome" is secondary to a direct blow to the mid abdomen from the lap portion of the seatbelt. It presents with ecchymosis of the abdominal wall (**A**) and is associated with mesenteric and small bowel injuries (**B**). (**C**) Increasing force can result in a transversely oriented fracture through bone, known as a Chance fracture. (**D**) Aortic or iliac vascular injuries require a high degree of suspicion and are often associated with seat belt signs and Chance fractures.

demonstrate free fluid, patients who are hemodynamically stable will still undergo CT scanning. Thus, the primary diagnostic modality for blunt HVI is CT. Initially, following introduction of CT scanning for trauma in the 1980s, the reliability to diagnose blunt HVI was poor. The advent of helical CT scanning in the early 1990s and many advances since have made CT extremely accurate. Although it may not identify overt HVI itself, it provides the astute trauma surgeon with findings that indicate a high likelihood of injury. Findings on CT associated with HVI include the following: fat stranding; hematoma of the mesentery; extraluminal air; thickened (>4–5 mm) wall; abnormal bowel contour; and free fluid without injury to a solid organ (Fig. 35-6). It is apparent that when a blunt small bowel perforation is



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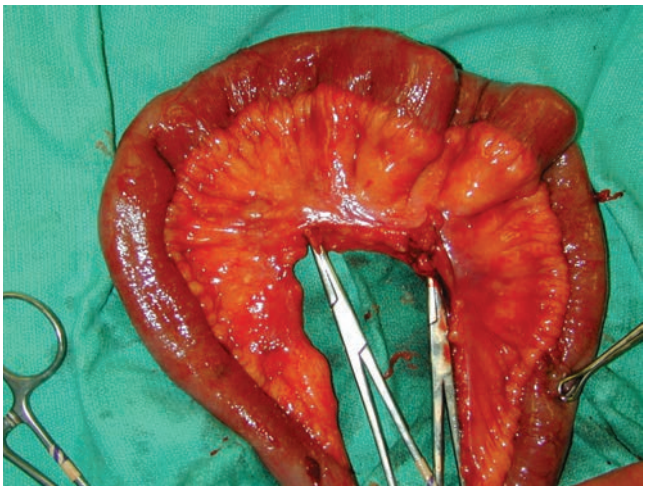


B

FIGURE 35-6 Computed tomography (CT) findings suggesting of blunt small bowel injury (**A**) include thickened loops of bowel (white arrow), free fluid within loops of bowel (black arrow), and free air (red arrow). (**B**) The associated small bowel injury from the findings on the CT included extensive mesenteric rent with two frank perforations (one identified by the Babcock clamp).

present, abdominal CT is usually abnormal.[18-21] CT findings specific for the rupture of the small bowel include extraluminal oral contrast and discontinuity of the wall of a hollow viscus, although oral contrast is rarely used for trauma CT scanning. Even when it is employed, extravasation is noted in less than 10% of documented cases of small bowel rupture. Active extravasation of contrast can be a specific sign of a mesenteric laceration, as is a mesenteric hematoma or fluid collection in the mesenteric folds.

Malhotra et al[21] reviewed the Presley Regional Trauma Center experience with screening helical CT evaluation of blunt injuries to the bowel and mesentery. In a group of 8112 scans, 100 were suspicious for injuries to the bowel or mesentery. There were 53 patients with bowel/mesentery injuries (true positive) and 47 without (false positive). The most common finding in both true-positive and false-positive groups was unexplained intraperitoneal fluid, which was present in 74% and 79% of scans, respectively. A pneumoperitoneum and bowel wall thickening were much more common in the true-positive scans. Multiple findings suspicious for bowel/mesenteric injury were seen in 57% of the true-positive scans but in only 17% of false-positive scans. The overall sensitivity and specificity of CT for bowel/mesenteric injury were 88.3% and 99.4%, respectively, whereas the positive and negative predictive values were 53.0% and 99.9%, respectively.

In an EAST multicenter study of HVI in 2019, sensitivity of CT scanning improved, but a significant number of patients with proven small bowel ruptures still had a negative CT scan. There was also a decrease in case mortality from 6% to 1.3%, associated with a shortened time to operation related to improved detection of findings indicative of HVI.[22]

The importance of free fluid on CT scan without overt solid organ injury has been vigorously debated. As outlined earlier, free fluid is common in patients without HVI. In a multicenter EAST study, free fluid without solid organ injury had a 38% incidence of a ruptured small bowel, but this means that 62% of patients did not.[21] In a multicenter prospective series of 2299 patients by Livingston et al,[23] 90 patients with free fluid and no solid organ injury were identified. In that group, only seven patients (9%) sustained an HVI.[15,23] Even with the use of multidetector CT scans, free intraperitoneal fluid is the most common finding of a blunt injury to the intestine or mesentery.[19,23] The amount and locations of the free fluid have been used to identify patients with HVI from those without HVI. Although patients with fluid in multiple areas are clearly at a higher risk from HVI than those with trace fluid in one location, the data are not sufficient to mandate laparotomy on that basis alone.[24] These patients do not have a "negative" CT scan, do require observation and serial abdominal examinations, and should never be discharged from the trauma receiving area.

Laboratory studies including hematocrit, white blood cell (WBC) count, and serum amylase are not useful in the initial evaluation of patients with blunt rupture of the stomach and small bowel.[15] In patients with a solid organ injury who are being managed nonoperatively or in patients with penetrating injuries undergoing serial clinical exams, unexplained tachycardia, hypotension, leukocytosis, an increase in serum amylase, or the development of a metabolic acidosis should arouse suspicion of a missed HVI. This is especially true in patients with an altered mental status, such as those with a concomitant traumatic brain injury or those who require intubation for other reasons. In this population, peritoneal lavage has been suggested as an adjunct with examination of the fluid for succus or WBCs. Although the presence of succus is a clear indication for laparotomy, an elevated WBC count is less predictive. Confounding issues include the presence of blood and the time between the injury and the lavage.[25]

Jacobs et al[26] found that a lavage WBC count of more than 500/mm^3 as the sole positive lavage criterion was a nonspecific indicator of intestinal perforation. In the presence of hemoperitoneum, Otomo et al[27] suggested using an adjusted WBC criterion (calculated as WBC ≥ red blood cells/150) as positive. These criterion had a sensitivity of 96.6% and a specificity of 99.4% for intestinal injury when performed more than 3 hours after injury. What is true is that a patient who is having unexplained signs of sepsis in the face of free fluid in the abdomen, especially one who cannot be easily examined, should be considered to have an HVI and undergo urgent operative exploration.

The diagnosis of a blunt intestinal injury is especially problematic in pediatric patients, in whom the findings on physical examination and x-rays may be less sensitive than in adults and may contribute to delays in operative treatment. Canty et al[28] suggested a delay of up to 24 hours after blunt intestinal trauma did not increase mortality or morbidity; however, a delay in definitive repair over 24 hours was directly associated with increased morbidity but not mortality. Additionally, a multi-institutional retrospective study of 214 patients failed to demonstrate a correlation between time to surgery, complication rate, and hospital length of stay.[29]

In contrast to that pediatric experience, Fakhry et al[30] published a multicenter experience in 198 patients with blunt injuries to the small bowel. There were 21 deaths (10.6% of total), with nine of these deaths attributable to a delay in operation. In patients in whom rupture of the small bowel injury was the major injury, the incidence of mortality increased with time to operative intervention. Mortality rates were 2% if the patient was operated on within 8 hours, 9.1% if operated on between 8 and 16 hours, 16.7% if operated on between 16 and 24 hours, and 30.8% if operated on more than 24 hours after injury (P = .009). The incidence of postoperative bowel-related complications, especially an intra-abdominal abscess, also increased significantly with time to operative intervention. In a retrospective review, Malinoski et al[31] noted that patients with delays of 5 or more hours had a 3.2 odds ratio of increased mortality. Clearly, these data would suggest that patients at high risk for HVI be carefully observed and monitored with early and aggressive intervention if their clinical condition deteriorates.[32]

LAPAROSCOPIC EVALUATION AND TREATMENT

The role of diagnostic laparoscopy for possible abdominal trauma has been controversial since its introduction to modern surgical practice. It has been considered by some to be helpful in avoiding laparotomy in hemodynamically stable patients with possible penetrating thoracoabdominal trauma and a suspected diaphragmatic injury.[33] With improved techniques and capabilities, many diaphragmatic tears and even gastric perforations may be repaired using laparoscopic techniques. Indications for diagnostic laparoscopy are less certain for patients with suspected blunt intestinal trauma. Early reports demonstrate an excessively high rate of missed injuries.[33] Because laparoscopic training and skills have markedly improved over time, it is possible that laparoscopic evaluation of a very select subset of patients, such as those with free fluid and worsening abdominal pain, but not frank peritonitis, may be an option.

OPERATIVE MANAGEMENT

Stomach

The surgeon should ensure that the incision is carried superiorly in the paraxiphoid area to facilitate exposure of proximal stomach and distal esophagus. The use of any one of the table-mounted self-retaining retractor systems can greatly facilitate this exposure. In the hemodynamically stable patient, the reverse Trendelenburg position can aid in exposure of this area by decreasing the pooling of blood to allow better visualization.

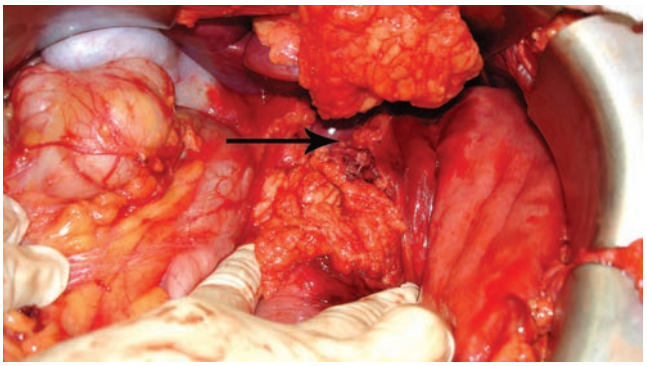
Mobilization of the stomach is essential for detection of gastric injuries. Exposure is generally easier if the stomach is decompressed first by a properly placed nasogastric tube. Unfortunately, most trauma patients appear to have eaten just prior to sustaining their injury, resulting in a stomach full of solid food. Almost all injuries can be managed despite a full stomach, albeit with some difficulty. Rarely, an injury may need to be extended into a gastrotomy to evacuate the stomach sufficiently to repair the injuries. A bloody nasogastric return should arouse suspicion for a gastric injury, as previously noted. Certain areas of the stomach, including the gastroesophageal junction, the proximal gastric fundus, the lesser curvature, and the proximal posterior area of the body and fundus, are difficult to assess (see Atlas Figure 62). Division of the left triangular ligament and mobilization of the lateral segment of the left lobe of the liver are helpful in exposing the anterior gastroesophageal junction. Encircling the esophagus at the gastroesophageal junction with a Penrose drain to maintain downward traction on the stomach can be useful to repair injuries in the proximal stomach.

If the gastrohepatic ligament is divided, care must be taken to avoid injury to the vagus nerve or its branches or the occasional anomalous left hepatic artery. To visualize the superior portion of the gastric fundus, the short gastric vessels should be divided and ligated. Overzealous traction in this area may cause tearing of these vessels or the splenic capsule, leading to troublesome bleeding. In patients with a concomitant splenic injury or those with extensive injuries to the body and fundus, mobilization of the spleen will facilitate exposure of the injuries to both organs.

The posterior wall of the stomach may be inspected several ways. The easiest and most expeditious is to enter the lesser sac through an avascular portion of the gastrocolic omentum along the body and/or antrum, leaving the gastroepiploic vessel intact. In obese patients with a thickened omentum and foreshortened mesentery, it is often better to enter this space in the upper or mid portion of the greater curvature of the stomach to avoid making a rent in the transverse mesocolon and possibly causing injury to the middle colic artery. For injuries that are close to the greater curve, it is better to expose

the surrounding area by ligating the branches of the epiploic vessel to the stomach. This dissection may be extended as necessary including up to the short gastric vessels to visualize an injury high in the fundus. Once the lesser sac is entered, the avascular gastropancreatic adhesions must be incised all the way up the lesser curve to ensure complete visualization of the posterior wall of the stomach. When there is an injury to the transverse colon or splenic flexure, mobilization of the left colon and splenic flexure will allow the lesser sac to be entered laterally through the space created by taking down the attachments from the spleen.

When an anterior hole in the stomach is found, a diligent search for a second hole *must* be undertaken. This is usually relatively straightforward, but there are several areas that can hide injuries and should be carefully inspected. These include the greater and lesser curvatures of the stomach, proximal posterior gastric wall, fundus, and cardia. Any hematoma, especially those on the greater and lesser curves where fat can easily obscure an injury, must be opened and explored (Fig. 35-7).



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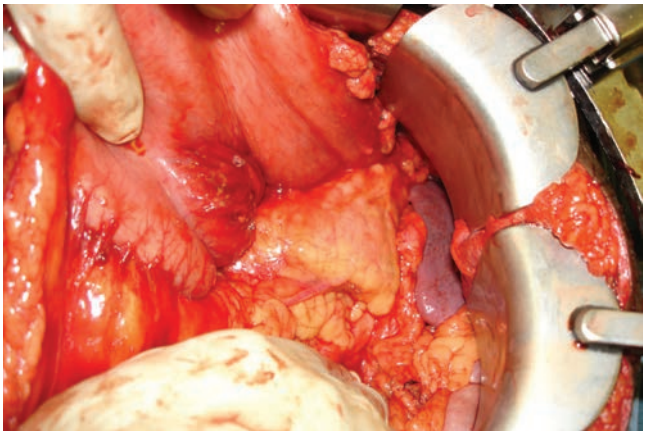


B

FIGURE 35-7 (**A**) Blunt injury to the stomach resulting in a full-thickness laceration to the lesser curve. The surgeon's hand is depressing the antrum of the stomach, and the extensive hematoma can be observed. The extent of laceration (black arrow) that is identified is also obscured by the fat on the lesser curve of the stomach, requiring full exploration for successful repair. (**B**) The posterior aspect of the stomach following full mobilization of the lesser sac demonstrating how benign the hematoma of the lesser curve can appear before exploration.

TABLE 35-1: Stomach Injury

Grade[a]	Description of injury	AIS-90
I	Contusion or hematoma	2
	Partial-thickness laceration	2
II	Laceration in GE junction or pylorus <2 cm	3
	In proximal one-third of stomach <5 cm	3
	In distal two-thirds of stomach <10 cm	3
III	Laceration >2 cm in GE junction or pylorus	3
	In proximal one-third of stomach ≥5 cm	3
	In distal two-thirds of stomach ≥10 cm	3
IV	Tissue loss or devascularization less than two-thirds of stomach	4
V	Tissue loss or devascularization greater than two-thirds of stomach	4

[a]Advance one grade for multiple lesions up to grade III.

AIS, Abbreviated Injury Scale; GE, gastroesophageal.

If a suspicion still exists after the search for a second hole is unsuccessful, a useful diagnostic adjunct is to have the anesthesiologist insufflate the stomach with air through a nasogastric tube. With the stomach submerged in saline, a telltale leakage of bubbles localizes any missed injury. Another option is to put 200 mL of saline containing an ampule of methylene blue down the nasogastric tube. An area of occult injury will be demarcated by blue staining. Very rarely, a tangential wound to the stomach and bowel can occur, but this is a diagnosis of exclusion. In the management of gastric injuries, it should be assumed that there should always be an even number of holes created.

Gastric injuries should be graded (Table 35-1), and the severity of the injures will often dictate treatment. Following blunt trauma, almost all grade I intramural hematomas require no treatment. Occasionally a hematoma that is expanding requires unroofing and control of hemorrhage. The entire area can then be imbricated with absorbable or nonabsorbable sutures. Following penetrating trauma, any hematoma must be explored because it may be hiding a full-thickness laceration. Gastric lacerations can be closed primarily in one or two layers using absorbable or nonabsorbable sutures. Many surgeons believe that there are several advantages to a two-layer repair. The first is that it quickly closes the hole and decreases any continued contamination. The second is that due to the vascularity of the stomach, ongoing bleeding is common and the first layer can help ensure hemostasis. The stomach is extremely difficult to narrow because it is capacious and will further expand over time; thus, the rule of thumb is to take large secure bites of the gastric wall with the sutures. Anterior and posterior wounds near the greater or lesser curve are often best treated by connecting the injuries into one discrete laceration. This allows the surgeons to ensure that the edges of the injury are well visualized circumferentially and will ensure complete closure. Staplers may also be used to close some gastric injuries. Because the stomach is thick, care must be taken to ensure that the proper sized staples are used.

For injuries near the gastroesophageal junction and the pylorus, care must be taken to avoid stenosis. The use of a bougie can help during closure of an injury near the gastroesophageal junction. Occasionally, a pyloric wound may be converted to a pyloroplasty to avoid possible stenosis in this area. Extensive wounds (grade IV) may be so destructive that either a proximal or a distal gastrectomy is required. Reconstruction with a gastroduodenostomy, gastrojejunostomy, or Roux-en-Y gastrojejunostomy is dictated by the anatomy and the presence or absence of associated duodenal and/or pancreatic injuries. Often these patients are in hemorrhagic shock due to other injuries and should be treated with damage control–type resections followed by later reconstructions. In rare cases, a total gastrectomy and a Roux-en-Y esophagojejunostomy are necessary for severe injuries (grade V).

Small Bowel

Examination of the small intestine for injury should be done systematically from the ligament of Treitz to the ileocecal valve. The small bowel should be completely eviscerated to the right. Any prior adhesions should be lysed to facilitate this maneuver. This also allows for the identification and control of mesenteric bleeding. The ligament of Treitz should be identified, and the small bowel examined loop by loop. Following blunt trauma, small bowel injuries are generally obvious and are often associated with extensive mesenteric bleeding and hematomas. Conversely, in penetrating trauma, especially stab wounds, small bowel injuries may be small and subtle. This is the reason that all hematomas on or adjacent to the bowel wall must be explored. Injuries should be marked with a suture or Babcock clamp to decrease spillage, but no attempt should be made to definitively repair any injury until the entire bowel is inspected. At that point, a plan of treatment can be devised as to how many bowel resections and repairs are needed. The overarching concept is to leave as much bowel as possible with the smallest number of repairs and resections. Thus, one might elect to resect an otherwise reparable injury if it is adjacent to an injury that requires resection. Small nonexpanding mesenteric hematomas away from the wall of the bowel should be reassessed at intervals throughout the operative procedure to assure their stability.

If significant bleeding from the mesentery is encountered, it should be controlled directly by either placement of clamps on the ends of the bleeding vessels followed by suture ligature or the accurate placement of sutures in a figure eight fashion. Mesenteric vessels often retract several millimeters from an injury, and the peritoneum over the mesentery may need to be opened to accurately identify the area of injury. Mesenteric defects are closed later to prevent internal hernias.

Bleeding at the root of the mesentery requires extra caution in obtaining hemostasis because of the concern for inadvertent damage to or ligation of the SMA or superior mesenteric vein. Clinical judgment about bowel viability has only a 65% predictive value under the best circumstances. Adjunctive techniques to access bowel viability such as intravenous fluorescein and bowel inspection using a Wood's lamp, Doppler flow studies, intraoperative laser angiography with indocyanine green, and bowel surface oximetry may be useful. If planned resection of small bowel will be so extensive that it may result in a short gut syndrome, an option is to terminate the procedure and provide temporary abdominal closure. Then a second-look procedure can be performed after the patient is rewarmed and perfusion deficits corrected before deciding to perform an extensive bowel resection. Performing resection at this later time may allow preservation of bowel that was of questionable viability at first operation. With massive bowel resections, it is important to note the location and length of the segment of the resected bowel. The most critical measurement is the length of the remaining small bowel. Preserving as much of the ileum as possible and the ileocecal value, if feasible, may obviate the complications related to extensive resections of the small bowel.

Exposure and repair of distal duodenal/proximal jejunal injuries are facilitated by taking down the ligament of Treitz. The entire small bowel in this area can be easily dissected back to the superior mesenteric vessels. There are often small vessels at the medial aspect that should be taken carefully using a right-angle clamp and fine ties. Following adequate mobilization, the injuries should be able to be clearly identified and either repaired or resected.

Treatment of an injury to the small bowel depends on its grade (Table 35-2). Serosal tears can be either closed with seromuscular sutures or left alone depending on size and if one is certain as to the depth of the wound. Similarly, intramural hematomas can either be left alone or be safely inverted with 3-0 or 4-0 seromuscular sutures. In general, the type of sutures used depends on surgeon preference.

Full-thickness perforations or ruptures of the small bowel usually less than 50% of the circumference (grade II) and not

TABLE 35-2: Small Bowel Injury Scale

Grade[a]	Type of injury	Description of injury	AIS-90
I	Hematoma	Contusion or hematoma without devascularization	2
	Laceration	Partial thickness, no perforation	2
II	Laceration	Laceration <50% of circumference	3
III	Laceration	Laceration ≥50% of circumference without transection	3
IV	Laceration	Transection of the small bowel	4
V	Laceration	Transection of the small bowel with segmental tissue loss	4
	Vascular	Devascularized segment	4

[a]Advance one grade for multiple injuries up to grade III.

AIS, Abbreviated Injury Scale.

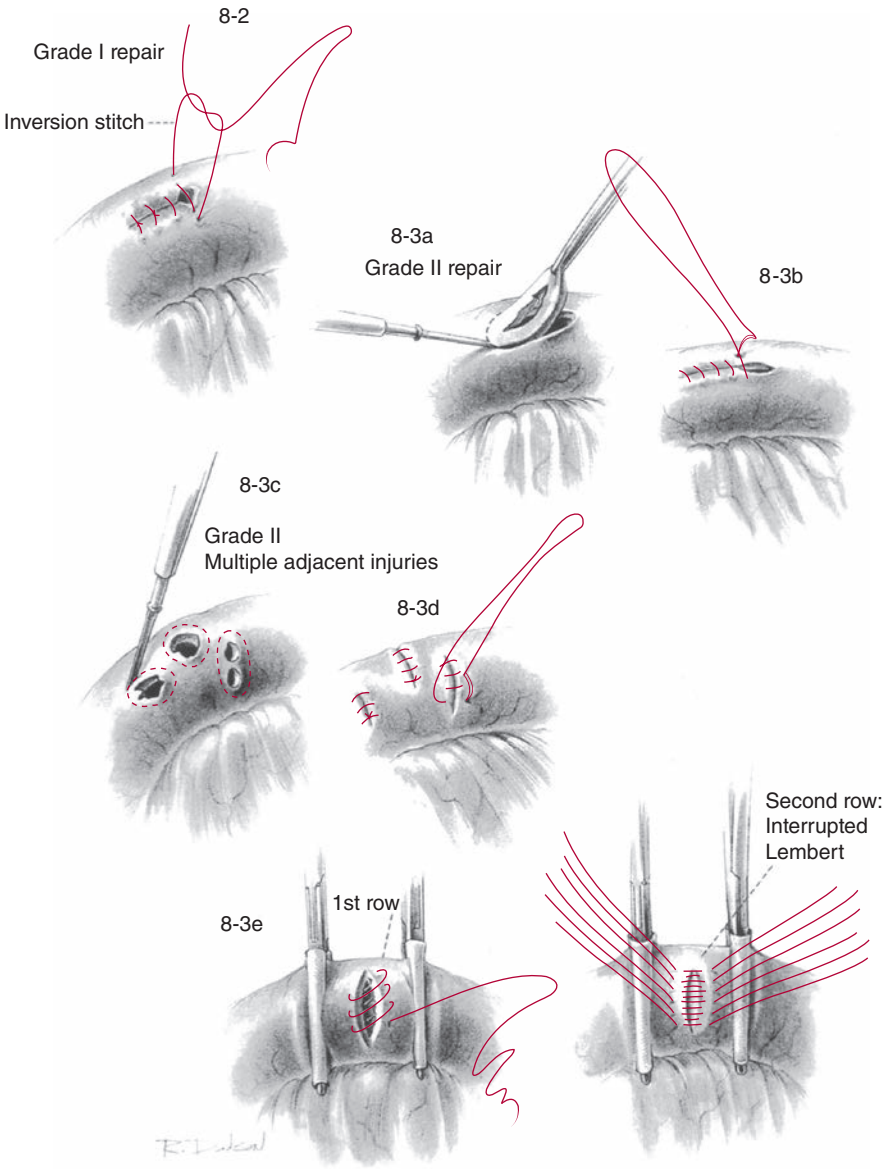


FIGURE 35-8 Treatment of grade I and II small bowel injuries. Grade I injuries are treated by inversion with seromuscular sutures. Grade II injuries are treated by careful debridement and primary closure. Either a one- or two-layer closure may be used. Adjacent through-and-through perforations are treated as a single defect by dividing the bridge of tissue separating them with electrocautery. (Reproduced with permission from Carrico CJ, Thal ER, Weigelt JA, eds. *Operative Trauma Management: An Atlas*. Norwalk, CT: Appleton & Lange; 1998. Copyright The McGraw-Hill Companies, Inc.)

involving the mesenteric border are most often amenable to suture repair (Fig. 35-8). The wound should be closed transversely by a single- or two-layer technique as it ensures the widest lumen. With lacerations or injuries along the antimesenteric border of bowel, the wound can be reoriented in a transverse fashion. If this is not possible, resection is often the best treatment. Adjacent through-and-through wounds of the bowel should be joined transversely using electrocautery and closed as a single defect. Multiple grade II injuries can usually be closed individually. Small bowel resection for multiple perforations may be favored if resection and anastomosis would take less time than closing the perforations individually and the amount of bowel sacrificed is minimal. An additional concern is the tendency to compromise the bowel lumen with closure of multiple perforations in a short segment.

Grade III and higher injuries usually require resection and anastomosis (Fig. 35-9). Occasionally, grade III wounds that are oriented transversely or in the relative large proximal to mid jejunum may be primarily repaired provided that an adequate lumen (at least 30% of the circumference) is maintained.

STAPLES VERSUS SUTURES

There continues to be controversy as to the risks of stapled versus handsewn anastomoses for traumatic bowel injuries.[34-41] Most of the available data are from retrospective studies,

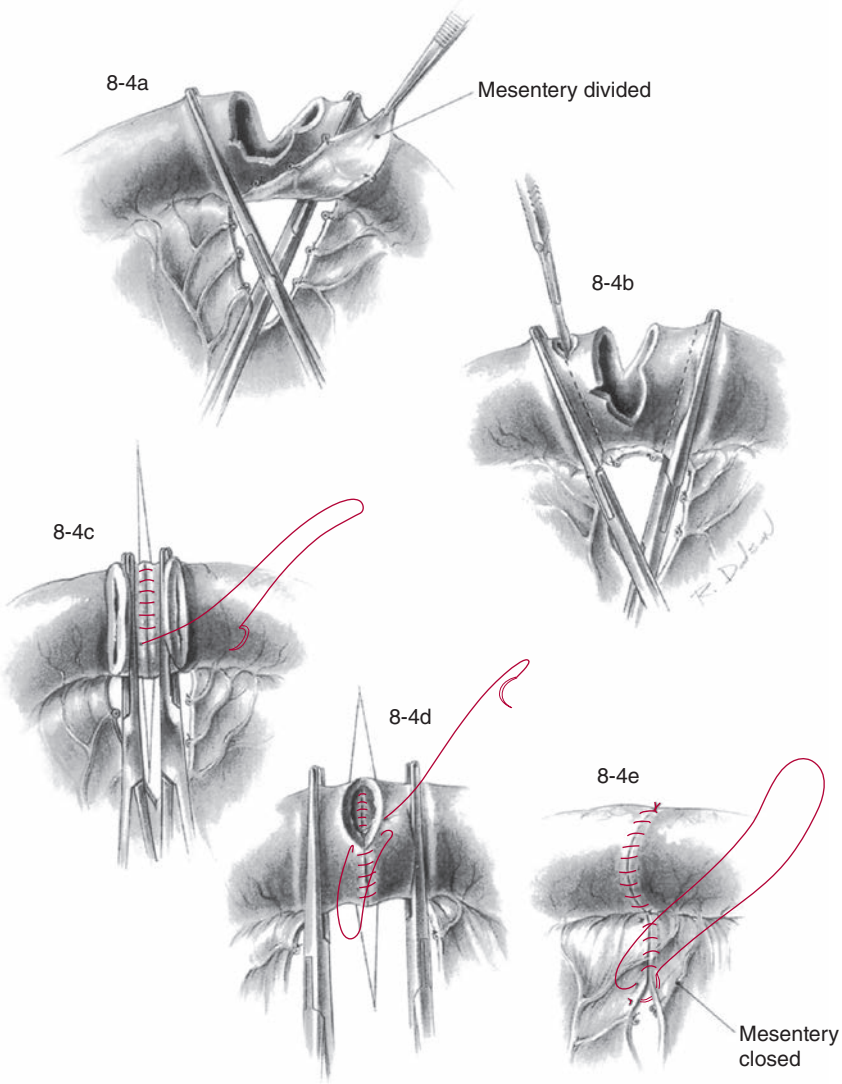


FIGURE 35-9 Grade III small bowel injuries are usually treated by resection and anastomoses. Proximal small bowel injuries or transversely oriented wounds may on occasion be primarily repaired. (Reproduced with permission from Carrico CJ, Thal ER, Weigelt JA, eds. *Operative Trauma Management: An Atlas*. Norwalk, CT: Appleton & Lange; 1998. Copyright The McGraw-Hill Companies, Inc.)

and there has only been one prospective study (Table 35-3). There are no controlled clinical trials comparing techniques for intestinal anastomosis following trauma. It is clear from over four decades of experience in thousands of patients undergoing elective resection that the complication rate is similar for stapled and handsewn anastomoses. Extrapolating this to trauma, where most patients are not in shock and not receiving aggressive fluid resuscitation, the lack of difference between handsewn and stapled anastomoses in the available series should not be surprising.

In contrast, patients who have significantly thickened bowel due to edema from massive resuscitation, damage control laparotomy, or sepsis must be considered separately. In a small retrospective series of emergency general surgery patients, Farrah et al[39] found that there was a higher rate of anastomotic failure with staples versus sutures (15% vs 6%; P <.003). Recently, in an American Association for the Surgery of Trauma multicenter trial, Bruns et al[40] prospectively studied 595 emergency general surgery patients undergoing 649 anastomoses (253 handsewn and 396 stapled) and documented a similar rate of anastomotic failure (12.5%). Of note, the handsewn group was more likely to be on vasopressors, have a higher lactate level, and have a lower albumin level. Thus, there remains a perception and treatment bias for the use of sutures in this group of patients.

In summary, the use of either staples or sutures to create an intestinal anastomosis is likely equivalent in almost all patients, and the trauma surgeon should use the technique that he or she is most comfortable using. In the severely injured patient, with a thick bowel wall and alterations in blood supply, it is also likely that the usual fixed staple height, which is perfectly acceptable for normal bowel, may not be optimal. In these cases, sutures may be advantageous. The other instance where sutures may be preferred is when the architecture of the anastomosis is unfavorable for stapling. This occurs at the ligament of Treitz or in the distal ileum near the ileocecal valve.

TABLE 35-3: Type of Small Bowel Anastomoses

Author	No. of patients	Site of injury	Handsewn	Stapled	Results and comments
Brundage et al[38]	84	Small bowel (101) and colon (17)	60	58	Retrospective single-institution study. Stapled anastomoses had significant increase in anastomotic leak requiring reoperation.
Witzke et al[34]	257	Small bowel only, but included primary repair (131) and resection/ anastomoses (144)	34	110	Retrospective single-institution study showed no difference in rate of intra-abdominal abscess, fistula, an anastomotic leak, or postoperative bowel obstruction.
Brundage et al[35]	199	Both small bowel (224) and colonic anastomoses (61)	84	140	Retrospective study from five Level I trauma centers. Higher leak rate and overall complication rate for stapled anastomoses.
Kirkpatrick et al[36]	227	Small bowel only; both primary repair (104) and resection with anastomoses (123)	24	52	Retrospective study from two institutions. No differences in anastomotic complication as related to technique used. Increased anastomotic complications associated with pancreaticoduodenal injury, or if done as part of a damage control procedure; 47 anastomoses were done using sutures/staples in a combined technique.
Farrah et al[39]	231	Small bowel to small bowel (125) Small bowel to colon (89) Colon to colon (15)	100	133	Retrospective series from single institution with large EGS population. Stapled anastomosis had over twice the leak rate of handsewn.
Bruns et al[40]	595	Small bowel to small bowel (466) Small bowel to colon (139) Colon to colon (44)	253	396	15-center AAST prospective observation trial of EGS patients undergoing emergency surgery. Overall leak rate was identical at 12.5%. The use of handsewn anastomosis in sicker patients reveals some treatment bias.

AAST, American Association for the Surgery of Trauma; EGS, emergency general surgery.

RESTORING INTESTINAL CONTINUITY FOLLOWING DAMAGE CONTROL

Trauma patients with intestinal injuries who require damage control laparotomy are often left with multiple disconnected segments of small and large bowel. At the time of reoperation, the surgeon needs to examine the remaining bowel and determine viability and how best to restore intestinal continuity. The concept is similar to that in patients with multiple bowel injuries who did not require damage control. Leave as much bowel as possible with the smallest number of anastomoses.

At the reoperation, segments resulting from injuries that initially did not appear in close proximity to each other on the index operation have contracted, resulting in insufficient length to adequately perform both a proximal and distal anastomosis. Shorter segments may be more likely to be engorged due to edema and intraluminal contents, which may compromise intestinal viability. In our experience, segments less than 15 cm are usually better resected. One specific difference in a bowel resection for damage control compared to that in a hemodynamically stable patient is that little thought is given to architecture of the initial resection as long as the remaining segment is viable. As the bowel fans out on its mesentery during the reoperation, the surgeon must be keenly aware that the transected ends of bowel may be ahead of their mesenteric blood supply. Although this blood supply may be sufficient to keep the segment viable, it may not be sufficient to support an anastomosis. If there is any question, one should not hesitate to re-resect the ends of the bowel more proximally. Although proximal bowel needs to be anastomosed, the risks and benefits of creating an ileostomy versus an anastomosis to the right colon must be carefully weighed. There is an old surgical adage that "the anastomosis never made, never leaks." Conversely, an ileostomy will require subsequent surgery.

The open abdomen does not appear to place the enteric anastomosis at risk of breakdown in the patient if the abdominal cavity can be closed "early."[41,42] In a Western Trauma Association multi-institutional trial that enrolled 244 patients with enteric injuries and an open abdomen, the leak rate for small bowel anastomoses was 3%.[43] In a recent series of emergency general surgery patients who underwent bowel resection, the need to treat with an open abdomen (odds ratio, 2.529; 95% confidence interval, 1.492–4.286) was independently associated with anastomotic failure.[40] In all patients who undergo a bowel anastomosis or repair, attempts should be made to protect the suture lines by using omentum, hiding the anastomosis laterally beneath the

abdominal wall or with other loops of bowel. In any reoperation to achieve abdominal wall closure, unless there is a specific indication, no attempt to dissect out and inspect the anastomosis or repair should be attempted. Achieving early fascial closure is the optimal modality to decrease anastomotic leaks.[44,45]

POSTOPERATIVE MANAGEMENT AND COMPLICATIONS

The postoperative care of patients after repair of injuries to the stomach and small bowel is usually relatively straightforward and is no different than that of elective surgical patients. Antibiotics such as cefoxitin or ampicillin-sulbactam are limited to no more than a 24-hour course, although appropriate dosing may be problematic in patients undergoing massive transfusion.[46] The use of routine nasogastric decompression, although still practiced by some, has been debunked by multiple prospective randomized controlled trials and a meta-analysis.[47] The potential impact of other clinically important variables, including the presence of multiple associated injuries, hemorrhagic shock, and postresuscitation bowel edema, as well as an impaired sensorium from a traumatic brain injury or drugs and alcohol, may make short-term nasogastric decompression the more prudent choice in select patients. Patients with extensive gastric repairs or resections may also benefit from a short course of nasogastric decompression, although this practice is also not generally supported by data. Nutritional support should be started as soon as feasible after the patient is resuscitated (see Chapter 62). Often, overall injury burden and shock rather than the gastric or small bowel injuries per se will dictate how well patients will tolerate gastric or enteral nutrition.

Complications that follow repair of injuries to the stomach and small bowel include intra-abdominal septic complications and anastomotic disruption. As previously noted, the most important etiologic factor in the development of an intra-abdominal abscess is delayed recognition and surgical treatment.[11,12] Anastomotic failures may present as peritonitis, as an abscess, or with the development of an external fistula.

Bleeding complications after repair of injuries to the stomach and small bowel trauma are rare but may present as bleeding into the peritoneal cavity or into the bowel lumen. Bleeding from an unrecognized short gastric vessel or from a torn splenic capsule is a common iatrogenic source of bleeding in patients with gastric injuries. Bleeding from the mesentery or lesser curvature of the stomach may not be apparent intraoperatively in the hypotensive patient and may only become clinically apparent when the patient's blood pressure normalizes. Continued bleeding postoperatively then manifests as hypotension and a falling hematocrit.[48] Bleeding from a gastric suture line presents as bloody nasogastric secretions, and endoscopic hemostatic techniques can be carefully employed in this setting. Finally, unexplained gastrointestinal bleeding from the small bowel may be the harbinger of an anastomotic disruption.

An anastomotic leak following repair of a gastric or small bowel injury can lead to significant morbidity and mortality. The definition of anastomotic leak is variable in both emergency and elective gastrointestinal reviews on the topic.[49] As noted earlier, anastomotic failures may present as a contained leak, diffuse peritonitis, or a gastrocutaneous or enterocutaneous fistula (ECF). Risk factors for breakdown of intestinal suture lines include resection and anastomosis rather than repair, massive transfusion and fluid administration, associated pancreatic injuries, and the development of the abdominal compartment syndrome. In patients with enteric injuries managed with an open abdomen, failure to obtain fascial closure after postinjury day 5 has been found to result in an increased leak rate.[41,43] Additional factors include evidence for ongoing hypoperfusion and the use of vasopressors during the initial resuscitation and early postoperative management in the intensive care unit.[50]

CT scanning obtained after day 4 to 5 is the best diagnostic imaging study to identify intra-abdominal pathology such as abscesses or anastomotic leaks that are not clinically obvious. Therapeutic options depend on the location of the initial injuries and pathology identified on CT. Percutaneous drainage is the primary intervention for fluid collections and intra-abdominal abscesses. A small area of radiographic inflammation in a stable patient may be treated as clinically insignificant leak with antibiotics. Anastomotic leaks diagnosed in the immediate postoperative period should be treated by reoperation with resection of the anastomosis back to healthy noninflamed bowel and, if appropriate, restoration of intestinal continuity. After 10 or 14 days, the inflammatory process may make the dissection and reoperation extremely difficult. In these cases, the goal is to control the effluent and provide adequate external drainage to create a controlled fistula. The use of latex or rubber drains and tubes will increase the inflammatory reaction and, provided there is no distal obstruction, will allow the fistula to close.

Enterocutaneous Fistula

An ECF is a dreaded complication following trauma laparotomy and may be the result of an anastomotic leak, missed injury, delayed perforation, or complications from an open abdomen following a damage control laparotomy. An ECF developing with an open abdomen is often referred to as an enteroatmospheric fistula (EAF). An EAF is actually a stoma without the benefit of the abdominal wall. As such, control of effluent from these fistulas can be exceedingly problematic. Factors associated with the development of an EAF include deserosalization and iatrogenic injury to the bowel in an open abdomen or adhesion of bowel loops to the fascial rim or adjacent bowel loops.[51] Excessive force on the bowel from coughing or even movement by the patient in this setting can lead to shearing of the bowel and bowel disruption. The goal in a patient with an open abdomen is to protect the bowel. Measures include keeping as much omentum as possible over the bowel or the use of nonadherent dressing materials. If negative-pressure therapy is to be used over the exposed small bowel, the white hydrophilic sponges should be used. Excessive use of negative-pressure therapy of abdominal wounds to promote granulation tissue may also contribute to the development of an EAF.[51] Dressing changes need to be performed

gently by experienced caregivers. Aggressive attempts should be made to obtain abdominal fascial or "skin only" closure as early as possible. It is our practice to aggressively attempt to close patients in the first week following damage control. Inability to make progress by that time results in immediate split-thickness skin grafting with planned late reconstruction of the abdominal wall. This can be done right over the intestines without waiting for any granulation tissue. A single-institution review of the development of an ECF in the era of open abdomen management was published by Fischer et al.[52] The overall incidence of ECF following trauma laparotomy was 1.9%. Patients with open abdomen had a higher ECF incidence (8% vs 0.5%) and a lower rate of spontaneous closure (37% vs 43%). The development of an ECF initiates the requirement for a prolonged stay in the intensive care unit and hospital as well as the need for a team of dedicated and experienced nurses, wound care therapists, and surgeons.

The three important concepts in the management of an ECF are as follows: (1) control of effluent and any associated infection; (2) nutritional support with fluid and electrolyte management; and (3) wound care with protection of the skin. In a posttraumatic ECF, there is no concern for radiation or neoplasia. Therefore, closure of the fistula will depend on location, size, nutritional status of the patient, and the absence of distal obstruction (ie, if the fistula tract has a higher pressure gradient than that of the normal alimentary tract, drainage will decline and the fistula will close). For example, a fistula emptying into an abscess cavity will never close because there is no pressure differential from the bowel until that abscess cavity is drained and collapsed. Fistulas that eventually form stomas to the skin or those in an open abdomen will never close based on lack of pressure differential and epithelization. These fistulas will eventually all require surgical intervention. Fistulas have been classified as high output (>500 mL/d), moderate output (200–500 mL/d), or low output (<200 mL/d). Yet, this distinction does not take into account the reason why a fistula actually stays open. The only reason to measure output is to help guide fluid management and nutritional support. In most patients, normal saline with 10 to 20 mEq of potassium per liter is a suitable fluid to use for the initial intravenous fluid replacement. Patients with an ECF may also develop significant calcium, magnesium, and phosphate deficits that should be measured and corrected. Careful attention should also be made to acid-base balance. Occasionally, measurement of the electrolyte composition of the fistula effluent is helpful for guiding fluid replacement (Table 35-4).

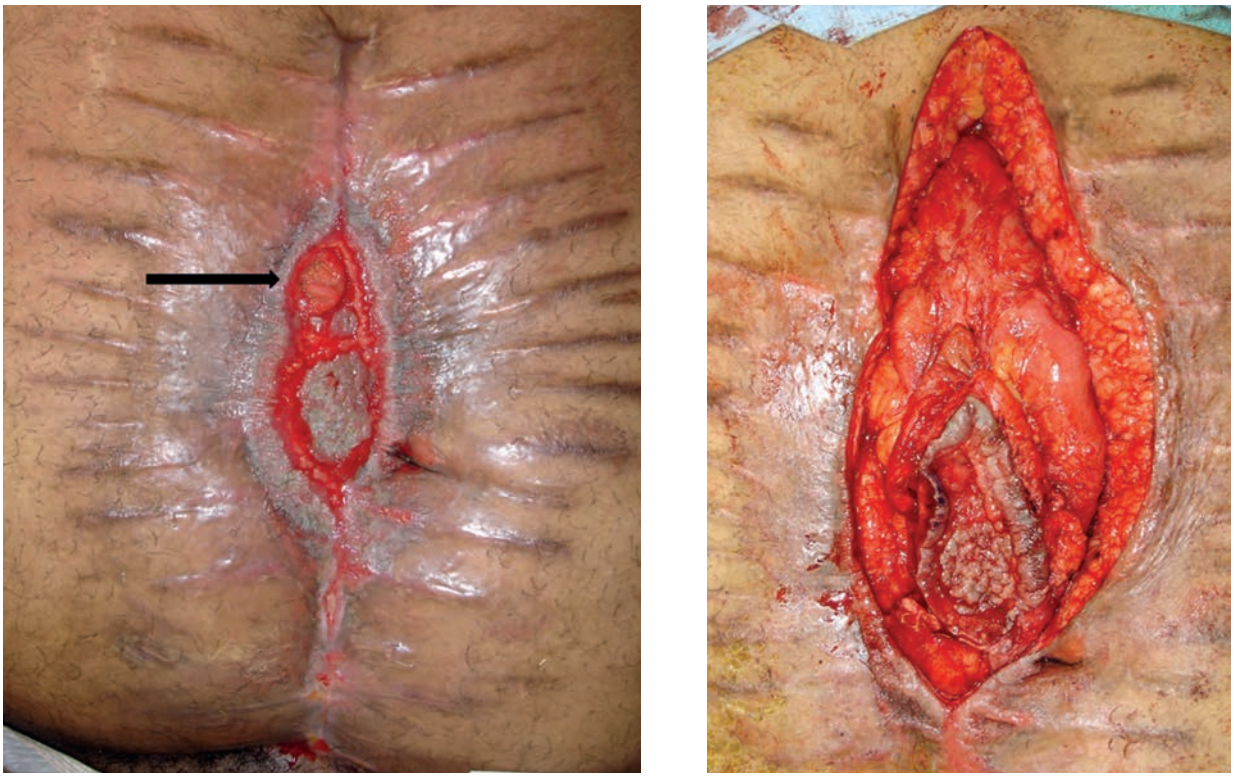
The delivery of adequate nutritional support is critical to help support the patient and hopefully eventually close an ECF (see Chapter 62). Enteral nutritional support is the preferred way of providing nutrition; however, with a high-output proximal small bowel fistula, this route may fail to provide adequate nutrition, aggravate fluid and electrolyte imbalances, and make wound care more difficult. In this situation, total parenteral nutrition (TPN) may be necessary. Adjuncts to control fistula drainage include a nasogastric tube and suppression with H_2-receptor antagonists or proton pump inhibitors. Somatostatin analog will decrease the volume of output but has not been shown to be beneficial to ECF closure.

Protecting the integrity of the skin surrounding the fistula is important and will improve patient comfort. Consultation with wound care nurses can be exceedingly helpful in devising systems to protect the skin and control effluent. In many cases, the surgeon must be resourceful to devise a system that works. It should also be realized that as the patient's wound heals, the architecture of the ECF and the abdominal wall will change, requiring the surgeon to alter the collection system sometimes daily. Negative-pressure therapy may be useful, but often ECF fluid is viscous and not amenable to a standard dressing. Creation of a negative-pressure system with opening for stomas has been devised; however, excessive negative pressure may prevent the ECF from closing.

Spontaneous closure of an ECF may occur in patients without epithelization (stomas) and distal obstruction who are free of sepsis and provided adequate nutritional support. Surgery will be required for patients whose ECF forms a stoma and those with longstanding fistulas. Definitive surgery is ideally delayed 3 to 6 months following the initial operation. Failure to obtain spontaneous closure should not be a primary factor in the timing of operative intervention but rather nutritional and wound status. Ideally, the patient should be in optimal shape, nutritionally replete, and free of ongoing inflammation before embarking on an often long and difficult surgical procedure. There are, however, patients whose tenuous nutritional balance mandates operative closure at an earlier time than ideal because these patients will never get better otherwise. Resection of a fistula requires complete lysis of adhesions to identify all small bowel limbs involved and eliminate the possibility of distal obstruction as a contributing factor for the failure of spontaneous closure (Fig. 35-10). Resection of the involved bowel segments back to healthy small intestine is the optimal management. In a

TABLE 35-4: Composition and Volume of Gastrointestinal Secretions

Type	Volume (mL/y)	Na (mEq/L)	K (mEq/L)	Cl (mEq/L)	HCO_3 (mEq/L)
Salivary	1500	10	26	15	50
Stomach	1500	60–100	10	100	0
Duodenum	2000	130	5	90	0–10
Small bowel	3000	140	5	100	15–30
Pancreas	800	140	5	75	70–115
Bile	800	145	5	100	15–35



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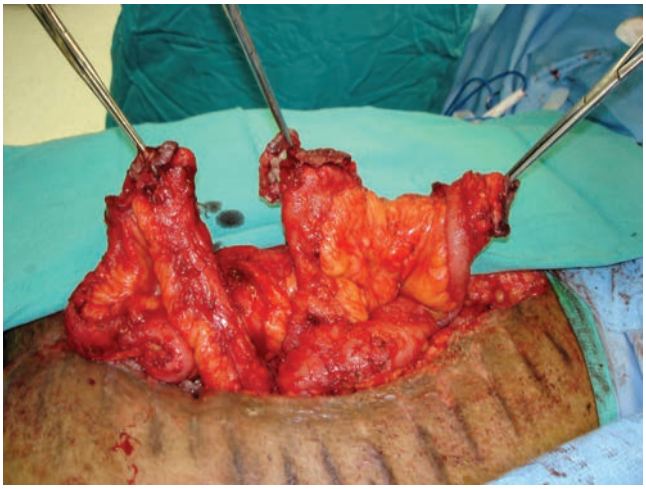

C

FIGURE 35-10 Morbidly obese (body mass index, 50 kg/m^2) patient who sustained a gunshot wound to the abdomen with an injury to the iliac artery. (**A**) Postoperative dehiscence resulted in an ECF (arrow) secondary to traction on a loop of bowel. The fistula was in the proximal jejunum and a feeding tube could never be passed distally for unclear reasons. Patient remained on total parenteral nutrition for 3 months until able to be explored. (**B**) The fistula complex is isolated with extension of the incision cephalad and extensive lysis of the adhesions to identify the anatomy. (**C**) Complete dissection disclosed three separate fistulas (clamps) with obstruction distal to the first fistula, which explains the inability to intubate the bowel.

study by Lynch et al,[53] simple oversewing or wedge resection of the ECF was associated with a 36% recurrence rate compared to 16% with resection and reanastomosis. After resection of the fistula and reestablishment of gastrointestinal continuity, attention is then directed to coverage of the bowels and closure of the abdominal wound. It is perfectly acceptable, and may be preferable, to leave the patient with a planned hernia following surgery to close an ECF. If the patient was closed with split-thickness skin grafting, operating through the skin graft is both possible and preferred.[54] Embarking on a complex abdominal wall reconstruction requiring unilateral or bilateral component separation after a long and complicated operation to close an ECF is not optimal. In these patients, the fistulas can be resected or closed and the overlying skin

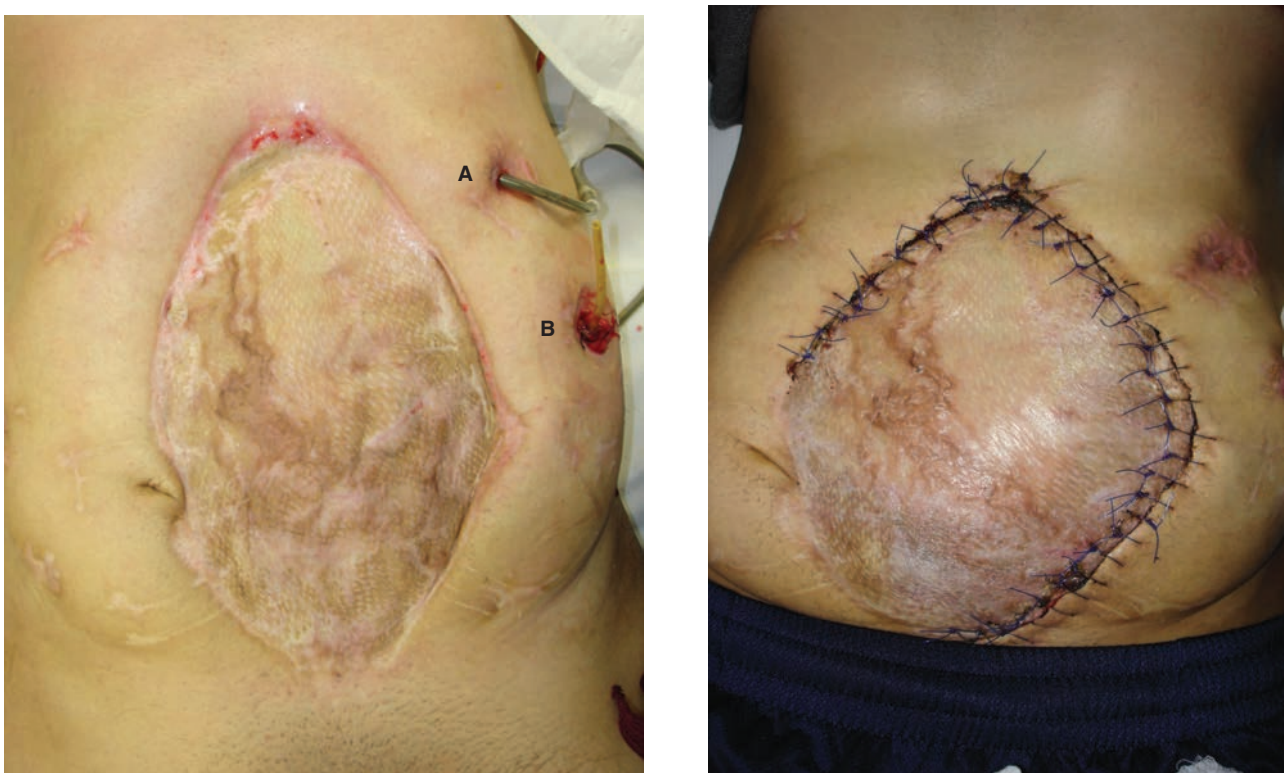

FIGURE 35-11 (**A**) Patient sustained severe duodenal injury requiring damage control, skin grafting of the abdomen, and placement of a decompressive gastrostomy tube (*A*) and an end feeding jejunostomy (*B*). Intestinal continuity was restored with a Roux-en-Y gastrojejunostomy through the skin graft, taking care not to devascularize it from the underlying viscera. (**B**) The abdomen was then closed using simple interrupted sutures from the skin graft to the native abdominal wall. The patient underwent abdominal wall reconstruction using mesh 8 months later.

simply reclosed (Fig. 35-11). The use of prosthetic materials, including cross-linked biological materials, is ill-advised due to concerns for the breakdown of intestinal anastomotic repair, the development of a de novo intestinal fistula, and serious wound complications and infection, as well as overall expense.[55,56]

Later reconstruction once intestinal continuity has been established and the patient has recovered from the ECF operation is a very reasonable and safe approach, and patients tolerate a hernia much better than they tolerate a fistula.

Small Bowel Obstruction

Small bowel obstruction (SBO) is a well-known complication following any abdominal operation. Patients with nontherapeutic laparotomies for trauma had a 2.4% incidence of SBO in a report by Renz and Feliciano.[57] The rate is higher if operative repair is required and may be up to 7.4% in patients with penetrating abdominal trauma and 10.8% in patients with injuries to the small or large bowel.[58] Trauma laparotomy does not appear to have added risk versus that reported following elective colorectal and general abdominal surgery on the development of an early SBO and need for operative management. The management of SBO following trauma laparotomy is similar to that following elective surgery.

Short Gut

Multiple intestinal injuries requiring extensive resection of significant amounts of small bowel may lead to problems with nutrition due to malabsorption. Removal of significant portions of the jejunum may lead to lactose intolerance; however, this is usually self-limited. Resection of the distal ileum often leads to vitamin B_{12} deficiency as well as bile salt deficiencies and subsequent fat malabsorption. Ileal resection including the ileocecal valve also removes the "ileal braking mechanism" that may cause decreased transit time throughout the gut. This may result in profuse diarrhea and significant fluid and electrolyte imbalances.

More often than direct injury to the bowel itself, a short bowel syndrome may result from an injury to its blood supply. In a series of 196 adult patients evaluated at a single institution over 23 years, 8% of the patients with a short bowel syndrome had been victims of abdominal trauma.[59] Historically, 80% of trauma-related short bowel syndrome has been due to mesenteric injuries. Clinical manifestations include malabsorption, diarrhea, steatorrhea, fluid and electrolyte disturbances, and malnutrition. Late complications include cholelithiasis and kidney (oxalate) stones. Short bowel syndrome is fully manifest when the remaining jejunum and ileum are less than 200 cm in length. Plasma citrulline may be a useful

biomarker to index the remaining small bowel enterocyte mass.[60]

Physiologic adaptation of patients with a short bowel syndrome follows a distinct trajectory.[61] The acute phase occurs during the immediate postoperative weeks and may last 1 to 3 months. This time period is marked by poor absorption of almost all macronutrients and micronutrients. Ostomies, if present, may have outputs exceeding 5 L/d during the first few days. Aggressive intravenous fluid and electrolyte replacement is necessary to prevent life-threatening dehydration and electrolyte imbalances. Gastric hypersecretion is frequent in this phase and may be treated with proton pump inhibitors. Loperamide, codeine, diphenoxylate, or even tincture of opium should be used to slow gastric and intestinal transit to control diarrhea. Careful monitoring of fluid and electrolytes, particularly potassium, magnesium, and calcium, is critical. Fluid needs can be monitored by urinary and fistula output, as well as by urinary sodium and osmolarity. Depending on the amount of bowel remaining, nutrition is often a combination of full TPN with supplemental elemental enteral feedings as tolerated. An adaptation phase will occur over time (months to years) with improvements in absorption of fluids and nutrients. Enteral feeding to provide intraluminal nutrients to maintain gut mass is necessary for the adaptation response to occur. Thus, enteral nutrition remains the primary therapy in maximizing luminal nutrient absorption in the intestinal remnant.

Patients who have survived their injury but have lost most of their small bowel may be candidates for intestinal transplantation. Trauma patients seem to have equivalent long-term survival rates as compared with nontrauma patients following intestinal transplantation. A multidisciplinary approach to the treatment of intestinal failure due to short bowel syndrome appears to be the best outcome for these patients.

CONCLUSION

An algorithm reviewing the operative management of injuries to the stomach and small bowel is presented in Fig. 35-12.

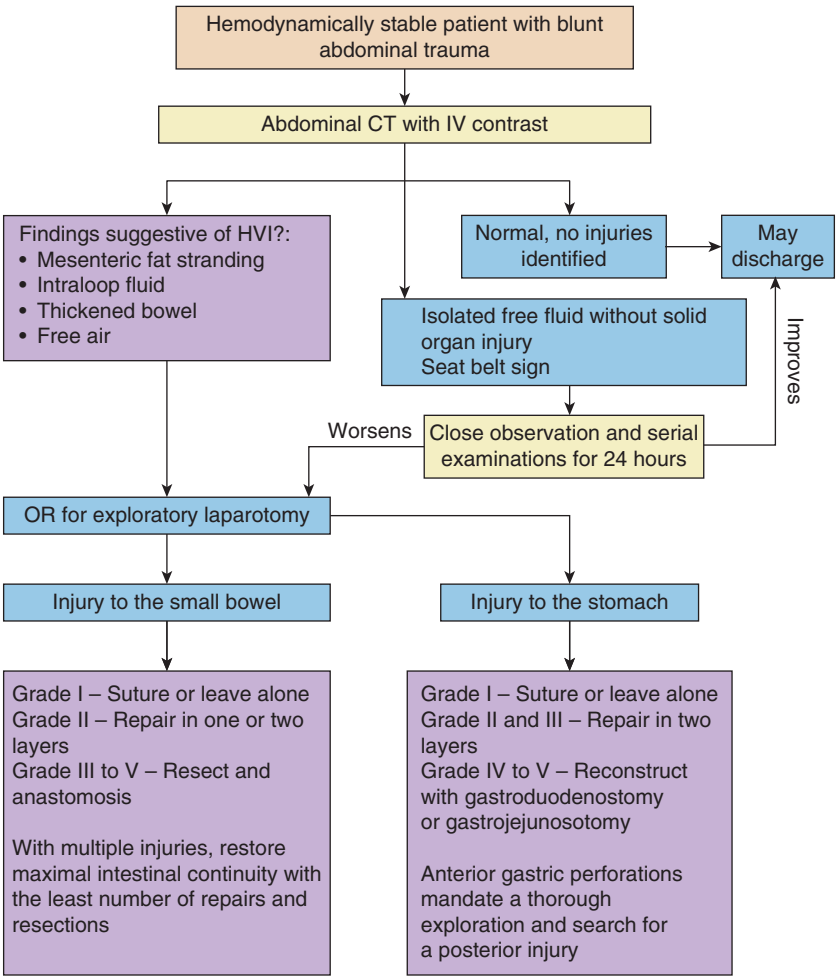

FIGURE 35-12 Algorithm reviewing the operative management of injuries to the stomach and small bowel. CT, computed tomography; HVI, hollow viscus injury; IV, intravenous.

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36

Duodenum and Pancreas

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KEY POINTS

- The incidence of injuries to the duodenum at laparotomy is <2% with stab wounds, 5% to 6% with blunt trauma, and 10% to 11% with gunshot wounds.
- A patient with a blunt rupture of the duodenum may have minimal symptoms initially due to its retroperitoneal location, low bacterial count, and neutral pH contents.
- A computed tomography (CT) diagnosis of a ruptured duodenum is suggested by air outside the duodenum in the right upper quadrant on the "scout" film and leakage of contrast on the formal study.
- A primary transverse or oblique suture repair of a blunt rupture or penetrating wound of the duodenum is appropriate if there is no loss of tissue and no injury to the major papilla.
- Pyloric exclusion with gastrojejunostomy should be considered as an adjunct to a duodenal repair when the repair narrows the duodenum, the duodenal wall around the repair is severely contused, there has been a delay in diagnosis of the duodenal injury, or there are adjacent, but not destructive, injuries to the C-loop and head of the pancreas.
- The incidence of injuries to the pancreas at laparotomy is approximately 6% with all forms of trauma.
- When a contrast-enhanced multidetector CT study is unclear about whether an injury to the duct of Wirsung is present after blunt trauma, a magnetic resonance cholangiopancreatography and/or an endoscopic retrograde cholangiopancreatography is performed.
- The Letton-Wilson Roux-en-Y distal pancreatojejunostomy is appropriate in a highly selected group of patients with Organ Injury Scale proximal grade III and grade IV injuries.
- A damage control approach (sequential operations) is now used in the majority of patients who need a pancreatoduodenectomy after trauma.
- Combined pancreatoduodenal injuries have significantly increased postoperative complications and mortality rates when compared to injury to either organ alone.

INTRODUCTION

The duodenum and pancreas are retroperitoneal structures that are protected somewhat from injury by the spine, retroperitoneal muscles, and overlying intra-abdominal viscera. Injuries to these structures are uncommon, and many trauma surgeons have limited experience in treating them. In the United States, injuries to the duodenum and pancreas are noted at only 1.5% to 11% and 5% to 6% of all laparotomies for trauma, respectively.[1] A recent review from the Trauma Audit and Research Network of the United Kingdom and Wales found that only 4.7% of patients with abdominal trauma had injuries to the duodenum or pancreas.[2]

Isolated injuries to the duodenum and pancreas are rare, and early deaths after injury are usually due to associated vascular injuries or multiple visceral and vascular injuries in the same patient. Other factors in management include delays in patient presentation after blunt trauma, the significant leak rate after pancreatic resections, and difficult decisions with combined injuries.

Outcomes for injuries to either organ have improved in recent years secondary to increased experience and to utilization of time-tested management algorithms.[3-7]

DUODENUM[8,9]

Anatomy

The duodenum is approximately 30 cm (12 fingerbreadths) in length and has four parts. The intraperitoneal short superior duodenum (D_1) starts beyond the pyloric muscle ring

of the stomach and goes to where it crosses over the common bile duct and gastroduodenal artery in the hepatoduodenal ligament. The retroperitoneal descending duodenum (D_2) extends from the hepatoduodenal ligament to the ampulla of Vater. The retroperitoneal transverse duodenum (D_3) is from the ampulla of Vater to the superior mesenteric vein and artery at the uncinate process of the pancreas. Finally, the retroperitoneal and distal intraperitoneal ascending duodenum (D_4) extends from the mesenteric vessels to the duodenojejunal junction at the ligament of Treitz.

The duct of Wirsung, or main pancreatic duct, enters the common bile duct to form the poorly named ampulla of Vater. This empties into the medial wall of the duodenum at the papilla (junction D_2–D_3), which can be palpated through a traumatic perforation or rupture of reasonable size in this area. The duct of Santorini or accessory pancreatic duct enters at the minor papilla in D_2, which lies 2 cm proximal to the major papilla described earlier.

The vascular anatomy of the upper abdomen complicates management of duodenal and pancreatic injuries, particularly the shared blood supply of the C-loop of the duodenum and head of pancreas. The superior pancreaticoduodenal artery, a branch of the gastroduodenal artery, is the arterial supply of the upper half of the duodenum. The inferior pancreaticoduodenal artery, a branch of the superior mesenteric artery, is the arterial supply of the lower half of the duodenum. On the venous side, the anterior branch of the superior pancreaticoduodenal vein drains into the right gastroepiploic vein, while the posterior branch drains into the portal vein. One or more branches of the inferior pancreaticoduodenal vein drain into the superior mesenteric vein.

Associated injuries in patients with duodenal trauma are most commonly to an upper abdominal vessel, liver, colon, and pancreas (approximately one-third of the time).[9] Major vessels adjacent to D_1 and D_2 include the hepatic artery, portal vein, inferior vena cava, the right renal vessels, and the left renal vein as it enters the inferior vena cava. Penetrating wounds to D_3 and D_4 may involve the underlying visceral abdominal aorta and the left renal vessels.

Mechanism of Injury

With blunt trauma, the duodenum can be compressed by a direct blow to the epigastrium such as from the handlebar of a child's bicycle. In an adult, a fist, the lower rim of the steering wheel, or a misplaced lap seat belt may be the cause. The injury that may result is a submucosal or subserosal hematoma, usually in D_2, although more extensive hematomas involving both D_2 and D_3 occur. A more extensive injury such as rupture is thought to be related to the anatomy of the duodenum (ie, the pyloroduodenal and duodenojejunal junctions, which are the two main points of fixation). It has long been hypothesized that sudden kinking of the duodenum at these points of fixation results in a sudden increase in intraluminal pressure during a deceleration-type motor vehicle crash and causes a rupture in D_2 or D_3.

In patients with penetrating thoracoabdominal, abdominal, flank, or back wounds mandating an emergency (peritonitis, hypotension, evisceration, bleeding from gastrointestinal or genitourinary tract) or urgent (delayed diagnosis, failure of nonoperative management) laparotomy, the duodenum is always exposed to see if an injury is present. Injuries to adjacent abdominal vessels and viscera, as described earlier, obviously increase the likelihood that a duodenal injury is present.

Clinical Presentation and Diagnosis

The clinical presentation of patients with *blunt* trauma to the duodenum will depend on whether a submucosal or subserosal hematoma versus rupture is present, the presence of associated injuries, and the time interval since injury.

A patient with a submucosal or subserosal hematoma of the duodenal wall will often have only minimal or moderate pain and epigastric tenderness on the initial physical examination in the emergency center. In the past, patients with minimal pain and tenderness were discharged from the emergency center without abdominal computed tomography (CT) being performed. As posttraumatic edema occurs, the patient develops a complete duodenal obstruction and persistent vomiting, usually within 6 to 12 hours. A 128-multidetector CT of the abdomen will confirm the presence of the hematoma in D_2 and/or D_3. A "coiled spring" sign or complete duodenal obstruction will be present if oral diatrizoate meglumine (Gastrografin) is administered before the CT (Fig. 36-1).

A blunt rupture of the duodenum as described earlier may also not be diagnosed on the first physical examination because the bacterial count of the duodenum is low (10^3–10^4) and pancreatic bicarbonate tends to neutralize gastric acid; hence, retroperitonitis may have a delayed presentation as compared to traditional peritonitis. When abdominal flat plate x-rays were performed routinely in patients with abdominal trauma in the past, surgeons would always look to see if there was a collection of air outlining the C-loop of the duodenum in the right upper quadrant. This finding suggested the diagnosis of a retroperitoneal duodenal rupture and would prompt an upper gastrointestinal contrast study to confirm the diagnosis. Currently, this abnormal collection of air would be noted on the preliminary scout CT or on the formal CT of the abdomen. The addition of oral Gastrografin would, once again, be used to confirm the diagnosis. An example of a CT diagnosis of a duodenal perforation is presented in Fig. 36-2.

An early diagnosis of a perforation or rupture of the duodenum is critical because a delay of 24 hours, a rare event in the modern era, led to a mortality rate of 40% in the past.[10] This is related to autodigestion of the retroperitoneum and the difficulty in repairing an edematous duodenum with everted mucosa.

In a patient undergoing an emergency laparotomy (no preoperative abdominal CT) after either blunt or penetrating

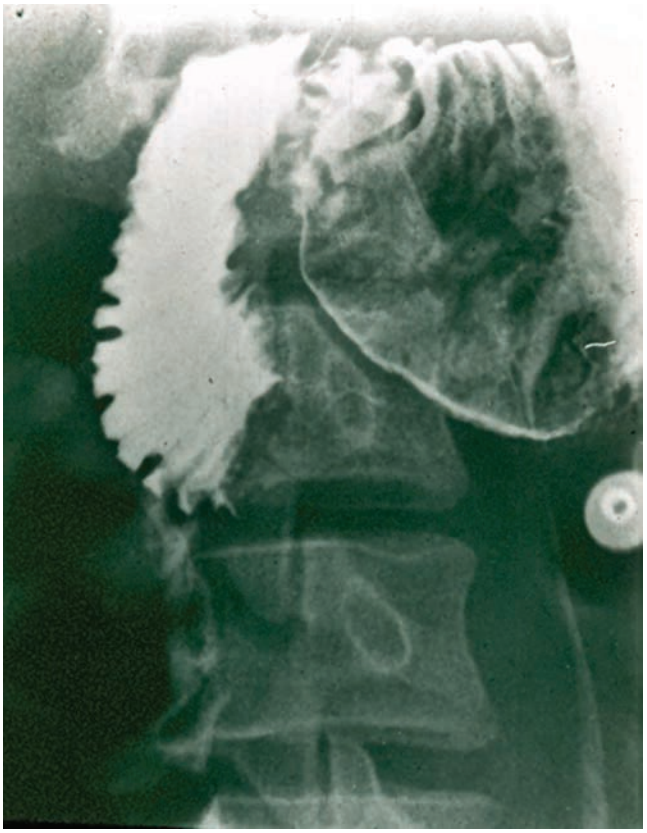

FIGURE 36-1 Duodenal hematoma at junction of D_2 and D_3.

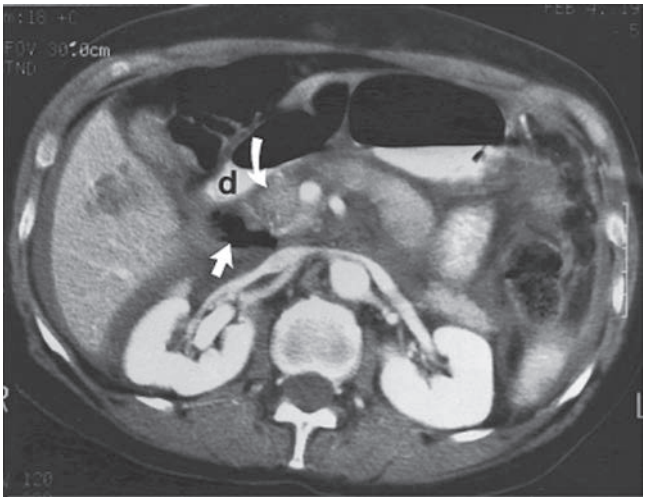

FIGURE 36-2 Computed tomography (CT) findings of retroperitoneal duodenal perforation. CT scan shows poor definition of the structures in the region of the head compared to the body. A collection of extraluminal, retroperitoneal gas (straight arrow) lies immediately posterior to the second portion of the duodenum (d), consistent with a duodenal perforation. (Reproduced with permission from Smith DR, Stanley RJ, Rue LW III. Delayed diagnosis of pancreatic transection after blunt abdominal trauma. *J Trauma*. 1996;40:1009.)

TABLE 36-1: American Association for the Surgery of Trauma Duodenum Organ Injury Scale

Grade		Injury description
I	Hematoma	Involving single portion of duodenum
	Laceration	Partial thickness, no perforation
II	Hematoma	Involving more than one portion
	Laceration	Disruption <50% of circumference
III	Laceration	Disruption 50%–75% circumference of D_2
		Disruption 50%–100% circumference of D_1, D_3, D_4
IV	Laceration	Disruption >75% circumference of D_2
		Involving ampulla or distal common bile duct
V	Laceration	Massive disruption of duodenopancreatic complex
	Vascular	Devascularization of duodenum

Source: Adapted with permission from Moore EE, Cogbill TH, Malangoni MA, et al. Organ injury scaling II: pancreas, duodenum, small bowel, colon, and rectum. *J Trauma.* 1990;30:1427-1429.

trauma, any area of injury in proximity to the duodenum is explored (see later in chapter).

Duodenum American Association for the Surgery of Trauma Organ Injury Scale (Table 36-1)

Management of the patient with a diagnosis of an injury to the duodenum is based on multiple factors, especially the magnitude of injury as determined by the American Association for the Surgery of Trauma (AAST) Organ Injury Scale (OIS).[11]

Nonoperative Management: Organ Injury Scale Grade I Duodenal Hematoma (Fig. 36-3)

An isolated blunt OIS grade I duodenal hematoma diagnosed on CT is managed nonoperatively. A nasogastric tube is inserted because of the total obstruction of the upper gastrointestinal tract, and the patient is maintained on total parenteral nutrition (TPN) through a central venous line. A repeat abdominal CT with upper gastrointestinal contrast is performed after 10 to 14 days to see if there has been any resolution of the hematoma. If the hematoma is still causing a complete obstruction of the duodenum, current options include another week of nasogastric suction and TPN, percutaneous drainage, or open or laparoscopic evacuation with insertion of a T-tube into the hematoma cavity.[12-15] After 21 days, failure of the hematoma to resolve or failure of the approaches mentioned earlier to drain or evacuate the hematoma should be followed by a laparoscopic or open

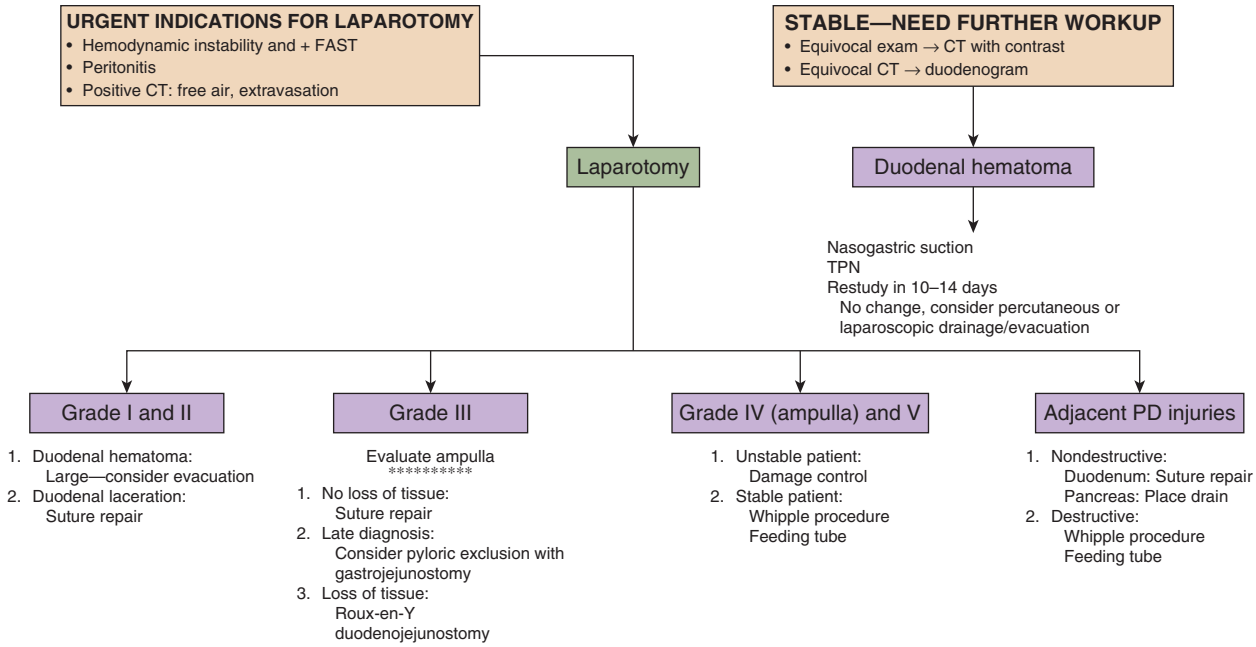

FIGURE 36-3 Algorithm for operative management of duodenal trauma. CT, computed tomography; FAST, focused assessment with sonography in trauma; PD, pancreatoduodenal; TPN, total parenteral nutrition.

gastrojejunostomy. The patient should be screened for the presence of *Helicobacter pylori* before discharge and treated if this is positive.

Operative Exposure

In patients with either blunt or penetrating trauma, areas of bleeding from viscera or vessels are controlled and gastrointestinal perforations or ruptures are repaired or resected. Exposure of the duodenum is then performed.[16] Ideally, the entire duodenum should be exposed at each laparotomy for trauma. In reality, blunt ruptures most commonly occur in D_2 and D_3 and are readily visualized by an extensive Kocher maneuver. As the track of a stab wound or civilian missile is usually straight, only the area of the duodenum in the track is usually exposed in patients with penetrating trauma.

The first step in complete exposure of the duodenum is medial mobilization of the distal ascending colon and hepatic flexure. An extensive Kocher maneuver is then performed, and this is facilitated by applying several Babcock clamps to D_2 and D_3 through the retroperitoneum and pulling these anteriorly. A scissors is used to divide the retroperitoneum just lateral to the antimesenteric edge of the C-loop of the duodenum. Gentle manual sweeping of the surgeon's hand under the elevated C-loop and head of the pancreas will allow for exposure of D_2 and a portion of D_3. In obese individuals, the middle colic vein can be used as a marker to where the superior mesenteric vein lies inferior to the pancreas.

The ligament of Treitz is then sharply divided from lateral to medial to mobilize the duodenojejunal junction until the fingers enter the space to the right where the Kocher maneuver was performed. This maneuver allows for complete visualization of D_4 and anterior D_3, whereas posterior D_3 can be palpated. The Cattell-Braasch maneuver described in 1960 can be performed to improve visualization of posterior D_3 if this is necessary to complete a duodenal repair.[17] First, the cecum, ascending colon, and hepatic flexure are mobilized medially by sharp division of the line of Toldt and traction on the colon. The retroperitoneal attachments of the mesentery of the small bowel are then sharply divided from the right lower quadrant to the duodenojejunal junction. With evisceration of the small bowel to the left, D_3 and D_4 can be visualized completely.

Operative Management: Factors in Repair

In critically injured patients, intraoperative hypotension and physiologic exhaustion secondary to associated injuries are the most important factors in operative management. Even so, an effort should be made to temporarily or permanently close the hole in the duodenum at the first damage control procedure. This is appropriate even if the temporary repair results in significant narrowing of the duodenal lumen. With near transection, both ends can be oversewn or stapled shut. Some type of closure decreases contamination and inflammation in an immunocompromised patient who will need more laparotomies.

When the patient is hemodynamically stable in the operating room, there are two important factors in choosing a technique for duodenal repair. The first is whether or not there has been a significant loss of tissue from the duodenal wall that will prevent a primary suture repair. The second is whether or not combined injuries of the duodenum and

pancreas in proximity are present, as the postoperative complication rate is always higher in such patients.[18,19]

Operative Management: Organ Injury Scale Grades II, III, and Selected Grade IV Injuries[1] (Fig. 36-3)

As previously noted, the duodenum has an excellent blood supply and reasonable mobility after an extensive Kocher maneuver is performed. Routine suture repairs of blunt ruptures (Fig. 36-4) or penetrating perforations without loss of tissue will, therefore, have a leak rate of only 1% to 3%. Repairs of such injuries away from the medial wall are performed in a transverse or oblique fashion using an inner full-thickness row of 3-0 absorbable suture and an outer seromuscular row of 3-0 silk sutures.[20] When the medial wall is part of the rupture or perforation, the hole is extended transversely toward the antimesenteric boarder. Through this hole, the major papilla is visualized and palpated to verify that it is not injured. If a hole in the medial wall is confirmed (pancreatic tissue visualized), closure is attempted using a single row of interrupted 3-0 absorbable sutures. The risk of a postoperative pancreatic fistula is significant with such an injury, and the hope would be that the fistula would drain internally into the duodenal lumen.

A repair of a longer rupture or perforation or one with contusions surrounding the area of duodenal repair is presumably at an increased risk for a postoperative leak. As a buttress to this repair, a viable pedicle of omentum can be mobilized off the right side of the transverse colon, placed on top of the repair, and held in place with interrupted 3-0 absorbable sutures.

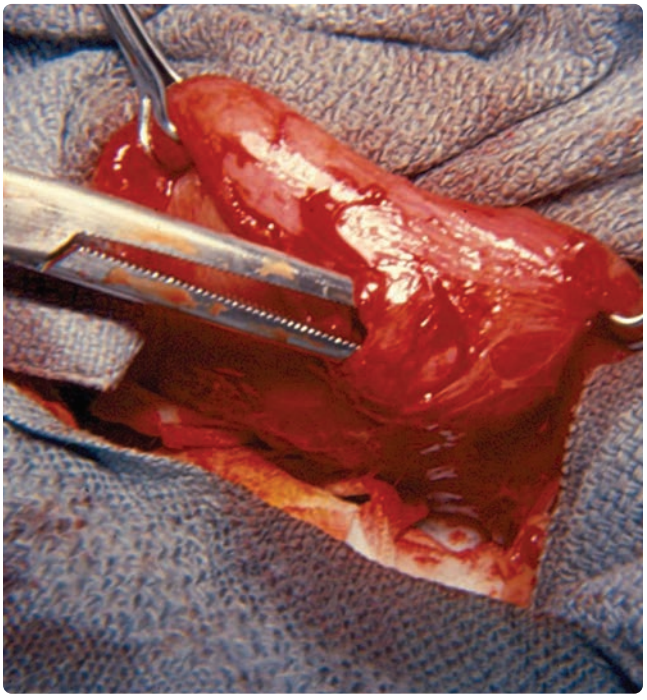

FIGURE 36-4 Blunt duodenal rupture in victim of pedestrian–motor vehicle crash.

When there is a loss of a portion of the anterior, antimesenteric, or posterior wall of the duodenum, a more complex repair is needed. The same is true in a patient with significant narrowing or an early leak after a rapid repair at the prior "damage control" laparotomy. The sutures in the duodenal wall are removed, and the edges of the open duodenal wall are debrided back to bleeding tissue. A standard jejunal Roux limb 40 cm in length is created and passed through the avascular area of the right transverse mesocolon. A side-to-end duodenojejunostomy is then performed at the site of the duodenal defect using the two-layer repair described earlier. As in general surgery procedures, the jejunal Roux limb is fixated to the window in the transverse mesocolon with circumferential 3-0 silk sutures. The final step in the procedure is to perform a sutured or stapled end-to-side or side-to-side jejunojejunostomy to restore gastrointestinal continuity.

There are few data on the use of a jejunal serosal patch to repair large defects in the wall of the duodenum as described in older publications.[21] A mucosa-to-mucosa repair is the procedure of choice in the modern era.

ADJUNCTIVE PROCEDURES: PYLORIC EXCLUSION WITH GASTROJEJUNOSTOMY

Several adjunctive operative procedures have been described over the past 113 years to lower the incidence of duodenal leaks after repair or to treat duodenal fistulas. In the modern era, these procedures are used occasionally in patients with one of the following, but in whom a Whipple procedure cannot be justified: (1) a narrowed duodenal repair; (2) a discolored or bruised duodenal repair; (3) a delayed duodenal repair in the previously mentioned presence of retroperitoneal digestion and duodenal mucosal eversion; or (4) a combined pancreatoduodenal injury.

The first adjunctive operative procedure described by Albert A. Berg, MD, at Mt. Sinai Hospital in New York City in 1907 was pyloric exclusion with gastrojejunostomy.[22] The original description was for treatment of postoperative duodenal fistulas. The procedure, however, was adopted by the trauma group at Ben Taub General Hospital (now Ben Taub Hospital)/Baylor College of Medicine in Houston, Texas, as an adjunct for the treatment of complex duodenal injuries or combined pancreatoduodenal injuries 70 years later.[18,23,24] The technique has been controversial in the trauma literature for the past 40+ years, with support from the Ben Taub group and others,[18,23-26] concerns about the addition of gastrojejunostomy,[27] and lack of improvement in overall results.[28,29] In the senior author's recent experience, the technique is used one to two times per year in a busy urban trauma center with 10% to 30% penetrating wounds. The indications have continued to be those described earlier, most commonly a delayed diagnosis of a duodenal rupture or perforation.

The procedure starts by skeletonizing approximately 10 cm of the dependent greater curve of the stomach within 6 to 10 cm of the pylorus. A dependent gastrotomy is then made through the skeletonized area. To facilitate an accurate and complete pyloric occlusion, Babcock clamps are placed

on the pyloric muscle ring, not the prepyloric antrum, at the 12 and 6 o'clock positions. By pulling these clamps superiorly and inferiorly, respectively, the pyloric outlet is converted to a vertical slit. Two rows of #1 polypropylene suture are then placed into the pyloric muscle ring to complete the exclusion. The procedure is completed by sewing or stapling an antecolic gastrojejunostomy.

Because this operative adjunct may increase the risk of a stomal ulcer over time, the patient is screened for the presence of *H pylori* before discharge. Standard treatment for *Helicobacter* is initiated if the screening is positive. In addition, an upper gastrointestinal contrast x-ray is performed to verify complete closure of the pylorus (Fig. 36-5). One older study documented that pyloric exclusions open between 14 and 21 days in 94% of patients.[24] Therefore, the exclusion will minimize output from a postrepair fistula that develops in the first 2 to 3 postoperative weeks.

Variations in the operative technique for pyloric exclusion have been reported. At the Hospital Universitario del Valle in Cali, Colombia, a feeding tube is first passed through the pylorus. Then the pyloric muscle ring is sewn closed tightly against the feeding tube through a gastrotomy, and a gastrojejunostomy is *not* performed. In Taiwan, the "controlled reopen suture technique for pyloric exclusion" was reported in 1998.[26] First, a slip knot is used at the end of the suture closing the pylorus. Then, the long suture end of the slip knot is brought out of the stomach and abdomen attached to a gastrostomy tube. If there is no evidence of a leak from the duodenal repair after 2 weeks, the slip knot is released by pulling on the long suture outside the abdomen.

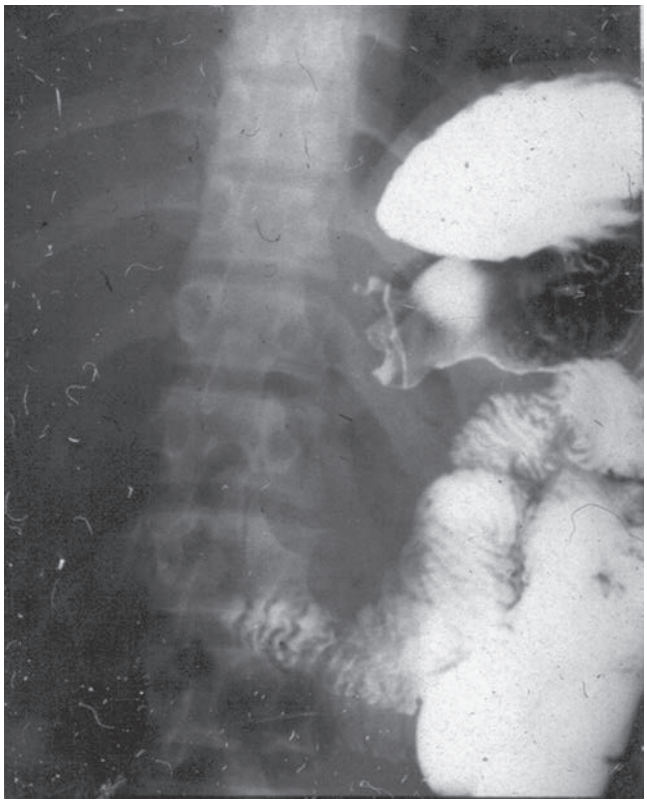

FIGURE 36-5 Postoperative upper gastrointestinal contrast x-ray documents complete pyloric exclusion.

ADJUNCTIVE PROCEDURES—DUODENAL DIVERTICULIZATION

In papers published in 1968 and 1974, the trauma group from Los Angeles County Hospital/University of Southern California described a diversion procedure for 50 patients with combined duodenal and pancreatic injuries.[30,31] The operation consisted of a truncal vagotomy, antrectomy, gastrojejunostomy, T-tube drainage of the common bile duct, and a tube duodenostomy or the insertion of drains adjacent to the duodenal repair. Although results were excellent, the technique has been used rarely in other centers over the past 40 to 50 years because of the following concerns: possible sequelae of the vagotomy; the sacrifice of normal antrum; and the difficulty in inserting a T-tube into a normal-sized common bile duct.

ADJUNCTIVE PROCEDURES: TUBE DECOMPRESSION

Originally described at Denver General Hospital (now Denver Health Medical Center) in 1978 in a small subset of patients, one to three tubes for decompression and/or feeding (gastrostomy, standard or retrograde duodenostomy, feeding jejunostomy) were inserted after the duodenal repair.[32] A subsequent report by H. Harlan Stone and Timothy C. Fabian at Grady Memorial Hospital in Atlanta, Georgia, in 1979 described 234 patients with duodenal injuries who had triple tube decompression after a duodenal repair.[33] Only one patient had a postoperative leak from the duodenal repair, and this patient had an adjacent periduodenal abscess. Although a tube duodenostomy is performed occasionally to decompress the C-loop after a tenuous repair in the modern era, the triple tube approach is used rarely. The major concern is the conversion of a solitary gastrointestinal injury, the hole in the duodenum, to four holes that must heal.

DRAINS AFTER REPAIR OF THE DUODENUM

The use of a closed suction drain adjacent to a repair of an OIS grade II laceration (<50% of circumference) of the duodenum is unnecessary. The exceptions would be when there has been a delay in diagnosis or when there are adjacent combined pancreatoduodenal injuries.[1] With OIS grade III, IV, or V lacerations, particularly those with a delay in diagnosis, contusions of the duodenal wall around the repair, or a complex repair, most, but not all, surgeons will insert a closed suction drain.

JEJUNAL FEEDING TUBE AFTER REPAIR OF THE DUODENUM

There is always a concern about passing a nasojejunal feeding tube through a duodenum that has just been repaired. Therefore, when a feeding tube is indicated (eg, multiple

intra-abdominal injuries, particularly after a first damage control laparotomy), a Witzel feeding jejunostomy away from an area of jejunal injury should be considered as an alternative. Unfortunately, with the distension of the midgut and the increased risk of intra-abdominal complications after a damage control laparotomy, some surgeons are wary of this approach as well. These concerns should not, however, prevent the insertion of a feeding tube by either approach when indicated.

Operative Management: Organ Injury Scale Grade IV Injury Involving the Ampulla or Distal Common Bile Duct and Grade V Injury (Fig. 36-3)

Destruction of the ampulla of Vater, massive disruption of the duodenopancreatic complex, and devascularization of the duodenum are all classic indications to perform a pancreatoduodenectomy. The timing of this operation will depend on whether the patient has one or more classic indications to perform a damage control laparotomy. If so, duodenal injuries should be sewn or stapled shut as previously noted, peripancreatic bleeders ligated, and closed suction drains placed adjacent to the duodenal and pancreatic injuries. The drains will remove any duodenal contents or pancreatic amylase and, should an early leak occur, act as a marker of the same should the first reoperation be delayed. In another scenario, the surgeon has been able to complete the Whipple resection, but the patient's physiologic status deteriorates. The operation should be terminated and the reconstruction completed at another operation. Reconstruction at a reoperation is usually by an end-to-end or end-to-side pancreatojejunostomy, end-to-side choledocho- or hepatodochojejunostomy, and an antecolic gastrojejunostomy. Two closed suction drains are placed adjacent to the pancreatojejunostomy.

There have been isolated reports of reimplantation of a transected ampulla back into the medial wall of the duodenum over the years. This avoids the pancreatoduodenectomy but should be performed with the assistance of an experienced hepatopancreatobiliary surgeon.

Complications After Operation for Duodenal Injuries

The incidence of postrepair duodenal fistulas with all grades of injury ranged from 4% to 16.6% with a mean of 6.6% in older reviews.[34] In a review of 29 patients with penetrating duodenal injuries (16 OIS grade IV injuries) in 2007, no patient developed a postoperative duodenal fistula.[28] A 19-year experience with 125 patients who survived longer than 24 hours after penetrating duodenal wounds (four OIS grade IV injuries) noted a fistula state of 8% in 2016.[3] In contrast, a review of 44 patients with "complex penetrating duodenal trauma" (nine OIS grade IV injuries) from four trauma centers in Colombia and the United States in 2014 described a fistula rate of 33.7%.[35]

In the comprehensive analysis of the experience at the University of Tennessee, Memphis, Schroeppel et al[3] noted that the incidence of a postoperative duodenal leak/fistula was not affected by demographics, admission variables, injury severity, location, grade of injury, or technique of repair. Some of these results seem counterintuitive, particularly when compared to the results in the Colombia–United States review. The Memphis group did note, however, that a patient was more likely to develop a duodenal fistula if a major vascular injury was present (60% vs 23%; $P = .02$), if a pancreatic injury was present (70% vs 31%; $P = .03$), or if a drain was present (90% vs 45%; $P = .008$).

The management of a suspected (no drain present but patient symptomatic or clinically deteriorates) or likely (bile in drainage) leak from the duodenal repair has remained the same for many years. An emergency abdominal CT with oral contrast is performed to confirm the leak and to see if a fluid collection is present in the periduodenal area or in Morison's pouch. A percutaneous drain is inserted into any fluid (bile) collection. The patient is then placed on NPO (nothing by mouth) status, TPN is administered, and subcutaneous injections of somatostatin analog at a dose of 200 mcg every 8 hours are considered. These latter injections are painful and expensive; however, they will lower the output of the fistula to minimize fluid and electrolyte losses and to protect the skin of the abdominal wall from leaks around a drain. If a pyloric exclusion had not been performed at the trauma laparotomy, a lateral duodenal fistula (in continuity with the gastrointestinal tract) in the absence of an associated intra-abdominal infection will take approximately 2 to 8 weeks to close. A persistent duodenal fistula beyond this time is extraordinarily rare. Should this occur, the patient is optimized nutritionally for 3 months before a difficult reoperation is considered. A repeat abdominal CT scan with oral contrast is performed again to verify that a duodenal fistula is present. At the reoperation, there are two options for repair. If the leak site is small, local debridement, suture closure, and an omental buttress are performed. A larger defect with extensive surrounding inflammation is debrided, and a Roux-en-Y side-to-end duodenojejunostomy to the defect is performed, as described previously.

Mortality

The mortality after duodenal (or pancreatic) trauma is always deceptive because most deaths are due to vascular injuries in the upper abdomen or to multiple intra-abdominal injuries, as previously noted. Overall mortality in patients with duodenal injuries ranged from 5.5% to 30%, and the mean mortality in 17 series from 1960 to 1990 was 17%.[34] Actual mortality related to the duodenal injury and its postoperative complication ranged from 0.09% to 3.9%, with a mean of 1.7%, in five series describing 942 patients from 1970 to 1985.[34]

In more recent series, the mortality in 2220 patients with isolated blunt duodenal injuries reported to the National Trauma Data Bank was 5.2%.[36] With penetrating injuries

TABLE 36-2: Mortality After Duodenal Injuries

Author	Mechanism	OIS	No. of patients	Mortality	Duodenum-related
Siboni et al,[36] 2015	Blunt	OIS ≥1; only duodenum	2220	5.2%	—
Seamon et al,[28] 2007	Penetrating	OIS ≥2	29	First repair 7% Exclusion 21%	—
DuBose et al,[29] 2008	Penetrating/blunt	OIS ≥3	147	First repair 15.1% Exclusion 10.7%	—
Schroeppel et al,[3] 2016	Penetrating	OIS ≥2	125	12%	1.6%
Phillips et al,[4] 2017	Penetrating	OIS ≥2	879	14.4%	—
Ordoñez et al,[35] 2014	Penetrating	OIS ≥2; "complex"	36	11.1%	2.8%

OIS, Organ Injury Scale.

(most OIS grade ≥II), the mortality rate in four reviews from 2007 to 2017 including 1069 patients ranged from 7% to 21% (Table 36-2).[3,28,35,37] In two reviews, duodenum-related mortality was 1.6% and 2.8%.[3,35]

PANCREAS[8,37]

Anatomy

The pancreas has a thin mesothelial capsule and is 15 to 20 cm (6+ inches) in length, 1.0 to 1.5 cm in thickness, and approximately 3.0 cm in width. It crosses the retroperitoneum at the level of the first and second lumbar vertebrae. The head of the pancreas is within the C-loop of the duodenum and encircles the distal common bile duct. The inferior part of the head or uncinate process partially wraps around the superior mesenteric vein to the right and the superior mesenteric artery arising from the visceral portion of the abdominal aorta. The thinnest part of the pancreas, the neck, lies between the head and body and overlies the mesenteric vessels and upper lumbar spine. The longest part of the pancreas is the body, which lies between the neck and tail and is posterior to the distal stomach. To the left of the body in the left upper quadrant is the tail of the pancreas attached to the spleen.

The main ventral pancreatic duct of Wirsung passes through the entire length of the pancreas just above a line halfway between the superior and inferior edges. As previously noted, it enters the left side of the common bile duct in the head of the pancreas to form the ampulla of Vater. The accessory dorsal duct of Santorini enters the duodenal lumen at the minor papilla. In 10% of individuals, the duct of Santorini is the main drainage of the pancreas.

There is a shared blood supply of the head of the pancreas and the C-loop of the duodenum through the anterior and posterior branches of the superior and inferior pancreaticoduodenal arteries as previously noted. Therefore, a major injury to the head of the pancreas or C-loop of the duodenum usually mandates a combined resection. On the venous side, veins around the head of the pancreas drain into the portal vein, while the remainder of the pancreas is drained by the splenic vein.

Associated injuries in patients with pancreatic trauma are most commonly to the liver, stomach, an upper abdominal vessel, and spleen.[9] Penetrating wounds to the head of the pancreas may involve the underlying inferior vena cava, the right renal vessels, and the left renal vein as it enters the cava. In similar fashion, injuries to the neck and uncinate process are in proximity to the inferior pancreaticoduodenal artery, superior mesenteric vein, and superior mesenteric artery. Finally, the body of the pancreas lies over the visceral portion of the abdominal aorta, whereas the body and tail encircle or are intimately adherent to the splenic artery and vein.

Mechanism of Injury

With a blunt blow to the epigastrium or a compressive force such as a misplaced lap seat belt in the obese adult, the pancreas can be partially or completely transected at the neck and over the mesenteric vessels and lumbar spine. This location is much closer to the C-loop of the duodenum than is commonly depicted in drawings in textbooks, with 70% to 90% of the pancreatic mass to the left of the transection.

As noted in the section on injuries to the duodenum, a penetrating abdominal wound with a track through the upper abdomen mandates exposure of the possibly involved portion of the pancreas. Once again, injuries to adjacent viscera and abdominal vessels increase the likelihood that a pancreatic injury is present.

Clinical Presentation and Diagnosis

The clinical presentation of patients with *blunt* trauma to the pancreas will often depend on the magnitude of associated injuries. Even with transection over the spine at the level of the neck of the pancreas, the patient may have only modest epigastric pain and tenderness when first evaluated because of its retroperitoneal location. In one older study from the University of Louisville in Louisville, Kentucky, 34% of patients with a

documented pancreatic injury had an equivocal or negative physical examination at the time of first evaluation.[38] Moderate epigastric pain and tenderness or persistent symptoms associated with nausea and vomiting during a period of observation mandate further evaluation with a multidetector CT.

It has long been recognized that the initial serum amylase is neither sensitive nor specific enough to confirm or rule out an injury to the pancreas.[39-41] For example, salivary amylase elevations related to acute alcohol intoxication account for a significant percentage of the hyperamylasemia seen on admission to trauma centers. An elevated serum amylase level, particularly one drawn 3 hours after trauma, or a level remaining elevated or rising during a period of observation mandates further evaluation as well.[42]

There have been concerns about the accuracy of 16- or 64-slice multidetector CT scans in diagnosing pancreatic injuries, particularly one to the duct.[43-45] The sensitivity and specificity of 4-, 16-, or 64-slice multidetector CT in detecting a ductal injury by visualizing a laceration and a laceration greater than 50% of the parenchymal width in one study in 2013 was 50% and 73.2% and 50% and 95.1%, respectively.[44] At this time, there are not sufficient data to evaluate the accuracy of 128-slice multidetector CT.

Recognizing these limitations, CT findings suggestive or diagnostic of an injury to the pancreas are listed in Table 36-3 and illustrated in Fig. 36-6. If a CT is not available or the result is equivocal (unclear if ductal injury is present, with or without associated hyperamylasemia), a magnetic resonance cholangiopancreatogram (MRCP) or endoscopic retrograde cholangiopancreatogram (ERCP) is appropriate.[46] Because MRCP is noninvasive, it is theoretically the next diagnostic test of choice; however, it is disappointing how little data on the use of MRCP in this situation are available.[47,48] With either MRCP or ERCP, the goal is to see if a ductal injury (OIS grade III) is present.[49,50]

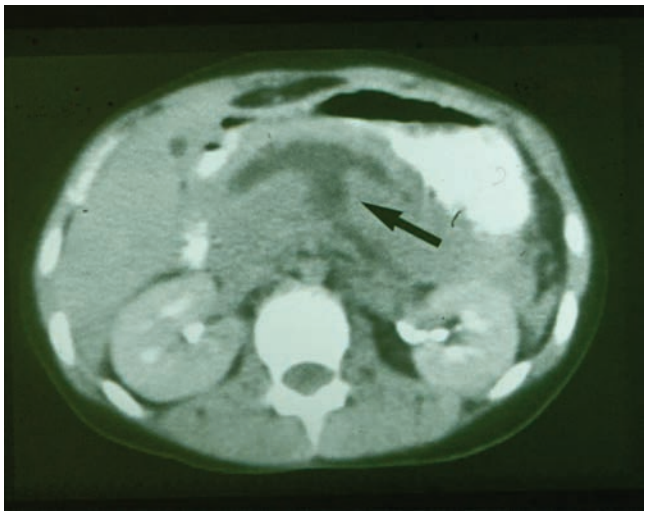

FIGURE 36-6 Preoperative computed tomography-documented transection of body of pancreas with fluid in lesser sac. (Reproduced with permission from Feliciano DV. Abdominal trauma. In: Schwartz SI, Ellis H, eds. *Maingot's Abdominal Operations*. 9th ed. Norwalk, CT: Appleton & Lange; 1989:457.)

Pancreas American Association for the Surgery of Trauma Organ Injury Scale (Table 36-4)

Management of the patient with a diagnosis of an injury to the pancreas is based on multiple factors, especially the magnitude of injury.[11]

TABLE 36-3: Computed Tomography Findings Suggestive or Diagnostic of a Pancreatic Injury

Suggestive of injury

Fluid in the lesser sac
Fluid between pancreas and splenic vein
Hematoma of transverse mesocolon
Thickening of left anterior renal fascia
Duodenal hematoma or laceration
Injury to spleen, left kidney, left adrenal gland
Chance (transverse) fracture of lumbar spine, especially in a child

Diagnostic of injury

Parenchymal hematoma or laceration
Obvious transection of the parenchyma/duct with fluid in the lesser sac
Disruption of the head of the pancreas
Diffuse swelling characteristic of posttraumatic pancreatitis

Source: Adapted with permission from Feliciano DV. Operative management of pancreatic trauma. In: Fischer JE, ed. *Fischer's Mastery of Surgery.* 7th ed. Philadelphia, PA: Wolters Kluwer; 2019:1573-1578.

TABLE 36-4: American Association for the Surgery of Trauma Pancreas Organ Injury Scale

Grade[a]		Injury description
I	Hematoma	Major contusion without duct injury or tissue loss
	Laceration	Major laceration without duct injury or tissue loss
II	Hematoma	Involving more than one portion
	Laceration	Disruption <50% of circumference
III	Laceration	Distal transection or parenchymal injury with duct injury
IV	Laceration	Proximal (to right of superior mesenteric vein) transection or parenchymal injury
V	Laceration	Massive disruption of pancreatic head

[a]Advance one grade for multiple injuries to the same organ.

Source: Adapted with permission from Moore EE, Cogbill TH, Malangoni MA, et al. Organ injury scaling II: pancreas, duodenum, small bowel, colon, and rectum. *J Trauma.* 1990;30:1427-1429.

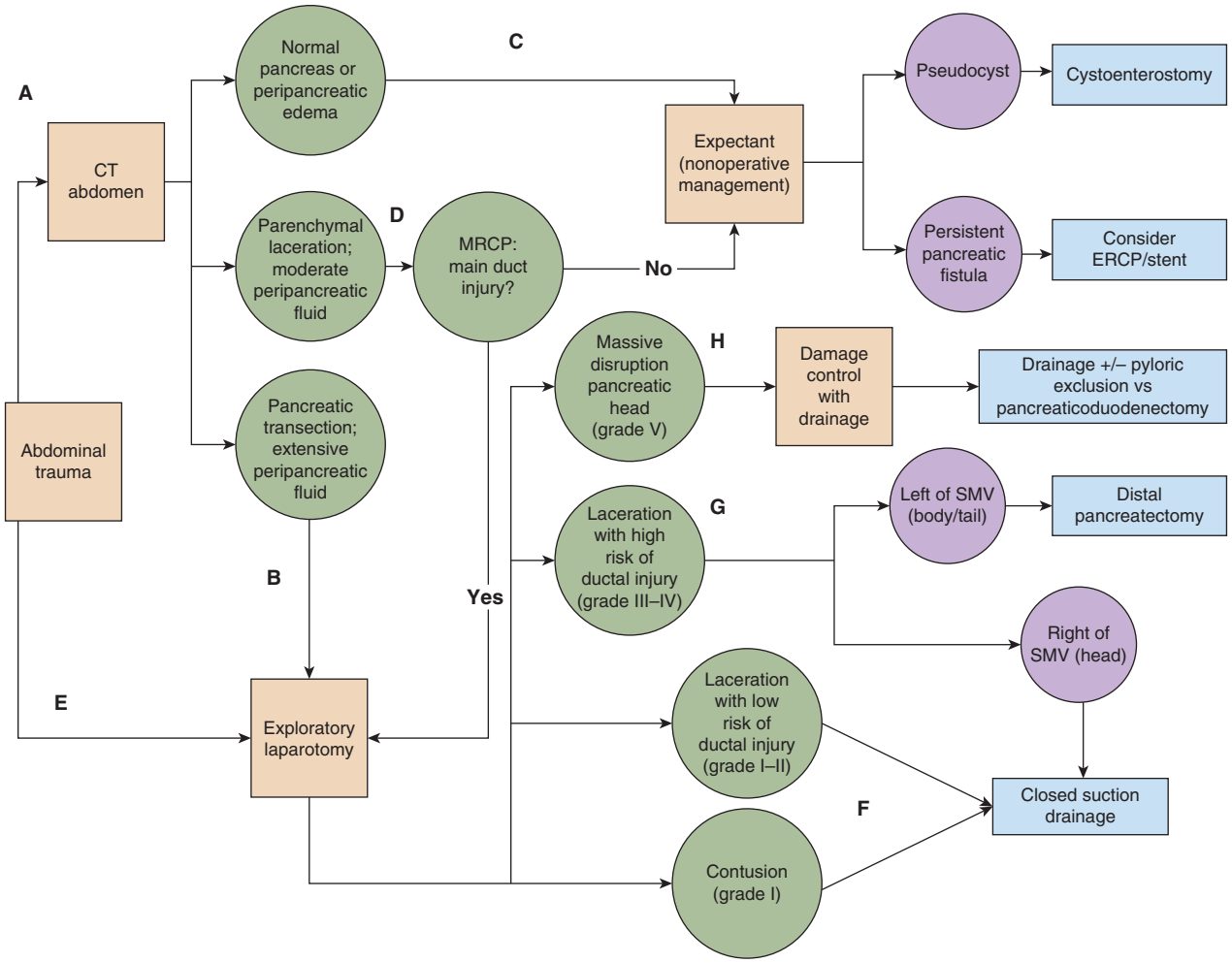

FIGURE 36-7 Western Trauma Association management algorithm for pancreatic injuries. CT, computed tomography; ERCP, endoscopic retrograde cholangiopancreatogram; MRCP, magnetic resonance cholangiopancreatogram; SMV, superior mesenteric vein. (Reproduced with permission from Biffl WL, Moore EE, Croce M, et al. Western Trauma Association critical decisions in trauma: management of pancreatic injuries. *J Trauma Acute Care Surg*. 2013;75:941.)

Nonoperative Management: Organ Injury Scale Grade I, II, and Select Grade III Injuries (Fig. 36-7)

In the absence of associated intra-abdominal injuries mandating an emergent or urgent laparotomy, a blunt OIS grade I or II pancreatic injury on CT is managed nonoperatively.[8] Such contusions or lacerations would be expected to heal, although an occasional patient may develop a peripancreatic fluid collection or pseudocyst. A follow-up abdominal CT scan is appropriate if the patient develops new-onset epigastric pain or hyperamylasemia while being observed.

Endoscopic stent placement into the main pancreatic duct for a variety of benign pancreatic diseases (stricture, leak) has been available for over three decades.[51] Stent placement across a partially or completely transected main duct after blunt trauma is appropriate under the following circumstances: (1) when the patient has no other intra-abdominal injury mandating an urgent laparotomy; (2) when the patient has an associated injury or medical condition (severe blunt cardiac injury, new-onset myocardial infarction, early-onset adult respiratory distress syndrome) that increases perioperative risk; and (3) when a surgeon or gastroenterologist with extensive experience in ERCP and stenting of the pancreatic duct is available. Endoscopic stenting is considered to be a success when the patient's symptoms resolve, hyperamylasemia clears, the stent can be removed at 3 months, and a follow-up CT and ERCP document a normal duct.[52,53]

Operative Exposure and Evaluation for an Injury to the Pancreatic Duct

Much as with injuries to the duodenum, areas of bleeding from viscera or vessels are controlled and gastrointestinal perforations or ruptures are repaired or resected. Ideally the entire pancreas should be exposed at each laparotomy for trauma because pancreatic contusions or hematomas can occur anywhere in the pancreas. Blunt partial or complete

transections, however, most commonly occur at the thin neck of the pancreas over the mesenteric vessels and spine, as previously noted. Much as with penetrating wounds of the duodenum, only the area of the pancreas in the track of a stab or missile wound is usually exposed.

The first step in complete exposure of the pancreas is medial mobilization of the distal ascending colon and hepatic flexure. This is followed by an extensive Kocher maneuver (to the superior mesenteric vein), which will allow for visualization of the anterior and posterior aspects of the head and the anterior aspect of the neck of the pancreas. The anterior aspect of the body is visualized after division of the gastrocolic omentum. Division of the retroperitoneal attachments to the inferior border of the pancreas while carefully avoiding the inferior mesenteric vein is then performed. Bimanual gentle elevation of the inferior border of the pancreas will allow for visualization of the posterior body. Finally, division of the lienorenal and splenocolic ligaments will allow for elevation and medial mobilization of the spleen. In the absence of a history of pancreatitis, the tail of the pancreas can be elevated as well, by sweeping the surgeon's right hand under the pancreas toward the midline.[8]

A hematoma under the thin mesothelial capsule of the pancreas should always be opened because a complete transection of the pancreas may be under this. In similar fashion, a penetrating wound of the pancreas should be exposed to determine its relationship to the likely area of the duct of Wirsung.

In the distant past, a surgeon who was still unsure about whether a ductal injury was present or not at this point had two options.[54] One was to perform a duodenotomy, cannulate the orifice of the pancreatic duct in the ampulla of Vater with a 5F pediatric feeding tube, and perform an operative pancreatogram. The other was to perform a resection of the tail of the pancreas and cannulate the duct in the open proximal end of the pancreas to perform the pancreatogram. In the modern era, the operative findings suggestive of an injury to the duct of Wirsung have been found to be very accurate in assessing its integrity[5,37] (Table 36-5 and Fig. 36-8).

A routine intraoperative cholangiopancreatogram or one using methylene blue dye is still an option to assess the integrity of the duct of Wirsung.[55] A #5 pediatric feeding tube is inserted into the cystic duct. After the anesthesiologist injects fentanyl to cause spasm of the sphincter of Oddi, standard cholangiogram contrast or one ampule of methylene blue solution in 200 mL normal saline is injected through the feeding tube. The spasm should cause reflux of the dye into the pancreatic duct and, when methylene blue is injected, staining and leakage at the site of a ductal injury. The obvious disadvantage of the approach is the need for a cholecystectomy after the cystic duct has been opened to perform the cholangiopancreatogram.

TABLE 36-5: Operative Findings Suggestive of Injury to the Duct of Wirsung

Fat necrosis in the lesser sac
Blunt laceration involving half or more of the width of the gland
Central missile wound
Significant disruption of the parenchyma
Leakage of clear fluid from area of injury during 5-minute period of observation

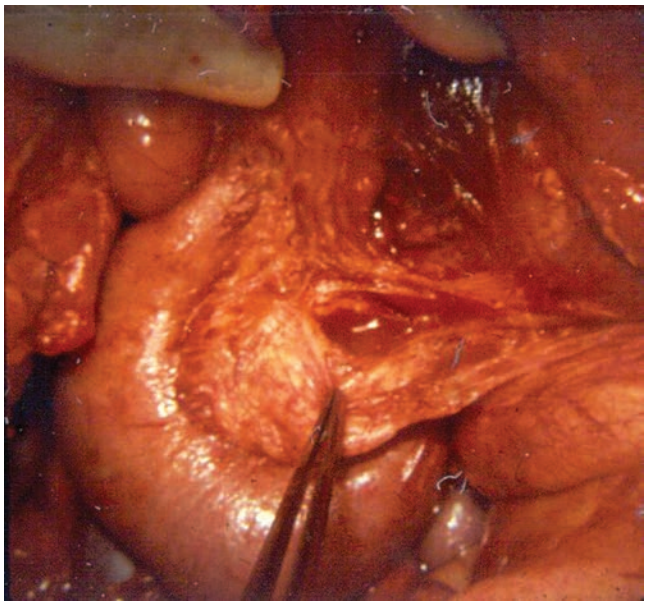

FIGURE 36-8 Stab wound of the head of the pancreas in which the surgeon believed there was no gross evidence of an injury to the main pancreatic duct. Drainage alone was performed. A pancreatic fistula developed but closed spontaneously. (Photo courtesy of Jon M. Burch, MD. Reproduced with permission from Feliciano DV. Abdominal trauma. In: Schwartz SI, Ellis H, eds. *Maingot's Abdominal Operations*. 9th ed. Norwalk, CT: Appleton & Lange; 1989:457.)

Operative Management: Factors in Repair

Much as with injuries to the duodenum, intraoperative hypotension and physiologic exhaustion are the most important factors in operative management. Once peripancreatic hemorrhage is controlled and a closed suction chain is inserted, no other operative procedure on the pancreas is necessary at a damage control operation.

A delay in diagnosis is another factor in choice of repair because autodigestion, pancreatic fistulas, and suture line leaks are more likely to occur in the postoperative period. After a delayed repair or resection, consideration should be given to inserting a feeding jejunostomy tube and peripancreatic drains as well as initiating empiric subcutaneous injections of octreotide. The value of octreotide is only that it may decrease the daily volume of any pancreatic fistula that might occur, although prospective clinical data are lacking.[56,57]

In hemodynamically stable patients with an early diagnosis and operation, the most important factor in choosing

drainage only, resection, Roux-en-Y reconstruction, and/or diversion is whether or not the duct of Wirsung has been injured.[1,8] Ductal transection mandates resection in most patients in the modern era (see later in chapter).

Finally, a combined pancreatoduodenal injury increases the complexity of management and, when the injuries are in proximity to one another, increases the fistula rate from either repair.

Operative Management: Organ Injury Scale Grade I to II Injury (Fig. 36-7)

An OIS grade I injury noted at laparotomy does not need to be drained. With an OIS grade II major laceration (Fig. 36-9), one option is to fill the laceration with a viable omental plug held in place with absorbable sutures. Although there are no data proving the value of this adjunct, theoretic benefits (based on its use in hepatic and splenic trauma) include the following: (1) tamponade of venous bleeding; (2) lower incidence of postoperative pancreatic fistulas; and (3) bringing mobile macrophages to the area of injury. Again, there are no clear-cut data regarding the need to drain OIS grade II pancreatic injuries.

Operative Management: Organ Injury Scale Grade III Injury (Fig. 36-7)

Transection of the duct of Wirsung over or to the left of the superior mesenteric vein is almost always treated with a distal pancreatectomy and splenectomy in adults.[58] Once again,

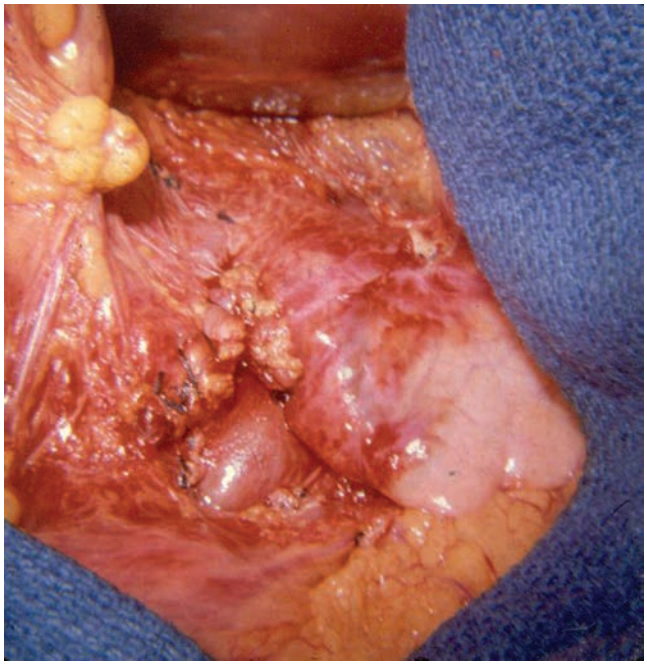

FIGURE 36-9 Organ Injury Scale grade II pancreatic laceration over superior mesenteric vein.

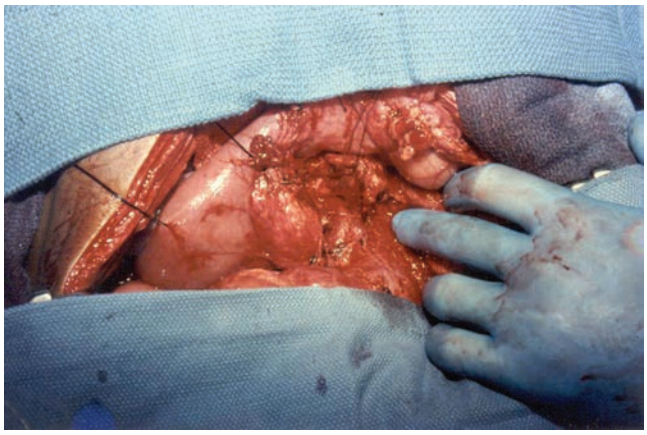

FIGURE 36-10 If this grade IV pancreatic injury is treated with distal resection, it will be a 90% pancreatectomy and splenectomy. (Reproduced with permission from Feliciano DV. Abdominal trauma. In: Schwartz SI, Ellis H, eds. *Maingot's Abdominal Operations*. 9th ed. Norwalk, CT: Appleton & Lange; 1989:457.)

this is often an 80% to 90% resection, as previously noted (Fig. 36-10). The splenic artery and vein are suture ligated separately with a 4-0 polypropylene suture 2 cm proximal to where the duct has been injured or where the pancreas is to be divided. Along with coverage of the stumps of the vessels with a viable omental pedicle, separate sites of division and ligation isolate the stumps from a distal pancreatic fistula occurring in the postoperative period.

A variety of surgical techniques have been used over the years by elective and trauma pancreatic surgeons to lower rates of postoperative pancreatic fistulas after distal resection. These have included the following: (1) fishmouth central beveled resection and horizontal mattress suture closure with or without ligation of the pancreatic duct; (2) use of transverse anastomosis or endovascular stapler; and (3) suture closure buttressed with fibrin glue, omental patch, or falciform mesothelial membrane.[59-65]

Large retrospective and prospective studies on elective and trauma pancreatectomies, some using the 2016 revised International Study Group on Pancreatic Fistula definition, have now been performed.[66] A 28% to 32% rate of fistulas has been consistent, no matter which type of closure is performed.[58-65] Therefore, a Jackson-Pratt or Blake drain is always inserted after distal pancreatectomy.[67,68]

LETTON-WILSON PROCEDURE

In highly selected patients who are hemodynamically stable and have no or few associated injuries, a Letton-Wilson procedure can be performed for an OIS grade III (or IV) injury[69] (Fig. 36-11). Originally performed in 1957 at Grady Memorial Hospital in Atlanta, Georgia, the first step in the procedure is ligation of the exposed duct of Wirsung in the open transected proximal pancreatic remnant (head or neck). This open end of the pancreas is then oversewn or stapled in the surgeon's usual fashion. The next step

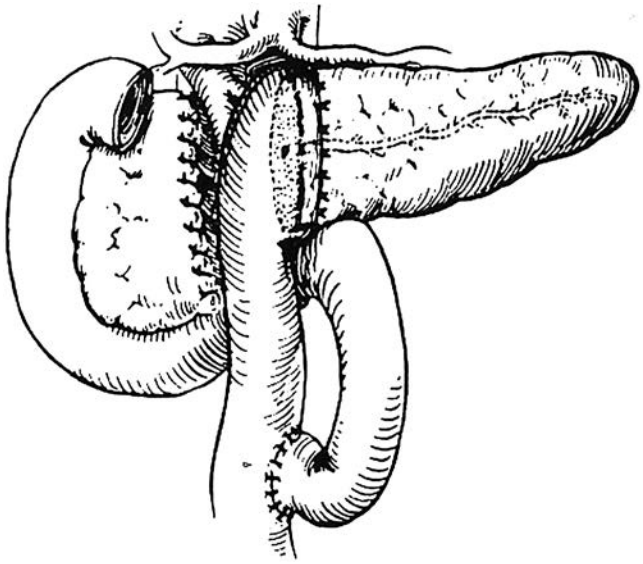

FIGURE 36-11 Letton-Wilson procedure for highly selected patients with proximal grade III or grade IV pancreatic injuries. (Reproduced with permission from Letton AH, Wilson JP. Traumatic severance of pancreas treated by Roux-Y anastomosis. *Surg Gynecol Obstet.* 1959;109:473. Copyright © Elsevier.)

is mobilization/elevation of 2 cm of the opened end of the distal pancreatic fragment (body and tail) off the splenic vessels. A 40-cm jejunal Roux limb is then created and passed through the right side of the transverse mesocolon, a two-layer invaginating end-to-end distal pancreatojejunostomy is performed,[70] and the Roux limb is fixated in the mesocolon with circumferential sutures. Finally, the standard end-to-side or side-to-side jejunojejunostomy is performed, and closed suction drains are inserted.

SPLEEN-PRESERVING DISTAL PANCREATECTOMY

Splenic immunity is critical in children, particularly those less than 2 years of age and probably in those up to 10 years of age.[71-73] For this reason, a spleen-preserving pancreatectomy for OIS grade III and IV injuries is preferred in young children.[74-76] In 2018, Schellenberg et al[76] reviewed data from 2007 to 2014 in the National Trauma Data Bank and noted that patients undergoing spleen preservation were younger, more likely to have blunt trauma, and had a lower Injury Severity Score.

Spleen-preserving distal pancreatectomy is easier in children than in adults. There is less fat obscuring pancreatic branches to the splenic vessels and less adherence of spleen to the pancreas. Using manual inferior traction on the pancreas, the small arterial and venous branches connecting the splenic vessels and pancreas are isolated over a small hemostat or right-angle clamp. A small metal clip is then placed on the pancreatic side of each vessel, while the end attached to a splenic vessel is ligated with a 4-0 silk tie. Once the pancreas is completely free from the splenic vessels, the distal pancreatectomy is completed.

LONG-TERM CONSIDERATIONS AFTER DISTAL PANCREATECTOMY

In the 1985 report by Ronald C. Jones from Parkland Memorial Hospital/University of Texas Southwestern in Dallas, Texas, 11 patients had 80% or more distal pancreatic resections for trauma.[77] Three patients developed diabetes mellitus requiring insulin, whereas three other patients had elevated blood glucose levels and abnormal glucose tolerance tests. Another patient who had a Whipple procedure for trauma developed diabetes mellitus as well.

It is worthwhile, therefore, to inform patients how much of the exocrine and islet cell masses of the pancreas are removed when a distal, hemi-, or subtotal pancreatectomy has been performed. In addition, they should be educated about the risks of alcoholism and obesity on the pancreatic remnant in the future. Finally, they should be advised to have a fasting blood sugar level checked every year in the future.

Operative Management: Organ Injury Scale Grade IV Injury

There are three options to manage the rare transection of the duct of Wirsung to the right of the superior mesenteric vein. First, a subtotal or 70% to 90% pancreatectomy and splenectomy in an adult is complicated by the need to close over the bulky proximal stump in the head-neck junction or head of the pancreas. Closure of this stump is usually with sutures because standard staplers may be too narrow to fit around it. The second option is the Letton-Wilson procedure, which is only appropriate in a highly selected group of patients, as previously described. Finally, the third option for blunt transections or destructive missile wounds through the head of the pancreas is a pancreatoduodenectomy (see next section).

Older trauma textbooks mention a fourth option, which is a Roux limb of jejunum sewn to the edges of the injury in this unusual location—a so-called "anterior" Roux-en-Y pancreatojejunostomy. This procedure should *not* be performed because it does not encompass the posterior aspect of any full-thickness transection or track of a missile and will lead to a high-output pancreatic fistula.

Operative Management: Organ Injury Scale Grade V Injury

Massive disruption of the head of the pancreas, devascularization of the C-loop of the duodenum, or destruction of the ampulla of Vater mandate a pancreatoduodenectomy. The timing will depend on the patient's hemodynamic status, the physiologic state, and the magnitude of associated injuries. In certain patients with blunt compression injuries, the trauma itself has "performed the Whipple" (ie, the head of the pancreas and C-loop of the duodenum have been devascularized and are already partially separated from surrounding structures).

The major change in operative approach in patients requiring a Whipple procedure for trauma over the past three

decades has been the use of staged procedures based on damage control principles.[78-82] If the Whipple resection cannot be performed at the first operation because of intraoperative hypothermia, metabolic acidosis, or a clinical coagulopathy, the management is as described previously in the duodenum section—the duodenal injury is closed, peripancreatic bleeders are ligated, and closed suction drains are placed. When the Whipple resection can be completed at the first operation, but reconstruction is contraindicated by "physiologic exhaustion," the following are indicated: (1) staple shut the open end of the common bile duct (prevents a bile leak and leads to dilation of the duct); and (2) staple shut the open end of the pancreas (prevents major leakage from duct, leads to dilation of the duct, and makes the gland more firm).[79] Reconstruction is performed when the patient's physiologic exhaustion has been reversed.

The mortality rate after Whipple procedures for trauma in 170 patients in 52 published reports from 1964 to 1990 was 33%.[34] In the most recent large civilian series from Harborview Medical Center/University of Washington, 12 of 15 patients (80%) undergoing Whipple procedures from trauma had a staged approach, and the mortality rate was only 13%.[82]

Summary of Operative Management for Pancreatic Injuries

In a review of 1000 patients undergoing operation for pancreatic injuries from 1980 to 1990, the following procedures were performed: (1) simple drainage, 72.6%; (2) distal resection, 16.9%; (3) Whipple procedure, 1.8%; (4) pyloric exclusions, 1.7%; and (5) other, 7.1%.[83]

Complications After Operation for Pancreatic Injuries

In older large reviews, the incidence of postoperative pancreatic fistulas with all grades of injury has ranged from 3% to 17% with a mean of 6%; however, trauma centers have used a variety of definitions of the complication over time.[66,83] In one recent series of distal pancreatectomies performed for trauma, pancreatic fistulas occurred in 29% of patients.[58] This is the exact same figure for the incidence of fistulas after elective distal pancreatectomies.

After elective pancreatic surgery, the International Study Group of Pancreatic Fistula now (as of 2016) uses the following definition: "a postoperative pancreatic fistula is now redefined as a drain output of any measurable volume of fluid with an amylase level >3 times the upper limit of institutional normal serum amylase activity, associated with a clinically relevant development/condition related directly to the postoperative pancreatic fistula."[66] Using this definition, pancreatic fistulas can be divided into clinical grades "biochemical leak" B and C. More commonly, trauma surgeons in the United States use the classic definition of intra-abdominal fistulas based on volume of output (high output, >500 mL/24 hours; moderate output, 200–500 mL/24 hours; and low output, <200 mL/24 hours).

The majority of pancreatic fistulas after a distal, hemi-, or subtotal pancreatectomy or a rare Whipple procedure for trauma are related to a soft pancreas encountered at the time of operation. They are usually self-limited in the absence of a history of preexisting benign pancreatic disease and will close within 2 to 6 weeks.

An elevated amylase level in an increased volume of fluid from a peripancreatic drain suggests that either a pancreatic or upper gastrointestinal fistula is present. A patient with a moderate- or high-output fistula associated with clinical deterioration (elevated temperature, onset of systemic inflammatory response syndrome, ileus) should undergo an abdominal CT with contrast. A fluid collection adjacent to the oversewn or stapled end of the pancreas or to a pancreatojejunostomy (or pancreatogastrostomy) can be treated with insertion of a percutaneous drain. This is controversial after distal resections because these collections are frequently sterile.[84] Appropriate antibiotic therapy is initiated should cultures of the fluid be positive.

Enteral nutrition beyond D_2 is appropriate after any left-sided resection when there is a moderate- or high-output fistula. Conversion to an oral diet is appropriate as the volume of fistula output decreases or when only a low-output fistula is present.

Much as with duodenal fistulas, the use of somatostatin analog in a patient with a postoperative pancreatic fistula remains controversial. After elective pancreatic resections, multiple reviews have failed to document a higher or earlier rate of closure of the fistula when somatostatin analog has been administered.[85,86] In a patient with a high-output fistula, however, it may be worthwhile to administer high-dose somatostatin analog for 5 to 7 days to see if the output decreases.

Much as with postoperative biliary fistulas, a persistent high-volume pancreatic fistula can be managed with ERCP, endoscopic sphincterotomy, and placement of a stent through the sphincterotomy into the pancreatic duct. One indication for an emergency reoperation is sudden catastrophic bleeding from the ligated stump of the splenic or gastroduodenal artery. An indication for a semi-elective reoperation is multiple recurrent abscesses in the left subphrenic area. Some of these patients will be found to have necrosis of the sewn or stapled stump of the pancreas and will require a re-resection.

The second most common complication after an operation for pancreatic trauma is a postoperative intra-abdominal abscess, with a range of 5% to 18% and mean of 5%. A patient with a left subphrenic abscess will usually develop an elevated temperature, onset of systemic inflammatory response syndrome, and a left pleural effusion on chest x-ray, and will have a positive abdominal CT. Percutaneous drainage is appropriate and usually effective for treatment. A rare patient has unsuccessful percutaneous drainage or recurrent abscesses, as described earlier. Rather than a formal reoperation through the midline incision, a local operative approach such as through the left flank or with resection of the left 12th rib posteriorly is appropriate.

TABLE 36-6: Forty-Year Comparison of Combined Pancreatoduodenal Injuries

Author/year	No. of patients	Penetrating mechanism	Damage control	Whipple	Mortality
Feliciano et al,[18] 1987	129	104/129 (80.6%)	0/129	13/129 (10.1%)	38/129[a] (29.4%)
Krige et al,[19] 2016	75	62/75 (82.6%)	29/75 (38.6%)	19/75 (25.3%)	21/75 (28%)

[a]One "pancreatoduodenal" death.

Mortality

Mortality after pancreatic trauma is obviously affected by the mechanism of injury, magnitude of the pancreatic injury, number and magnitude of associated injuries, and the patient's hemodynamic status on admission. Overall mortality is approximately 20%, with the following rates based on mechanism of injury: stab wound, 2.8% to 5%; gunshot wound, 15.4% to 22%; and blunt trauma, 16.9% to 19%.[31]

COMBINED DUODENAL AND PANCREATIC INJURIES[8]

Even in the busiest urban centers with penetrating wounds accounting for 25% to 30% of all trauma admissions, only 7 to 10 patients with combined injuries will be treated each year.

Operative Principles

The first principle of operative management is to treat geographically separate, but combined, injuries individually. An example would be a patient with multiple gunshot wounds and grade III injuries to the mid-pancreas and to D_2. This patient should have a distal pancreatectomy and splenectomy, a transverse repair of the duodenum, and insertion of a closed suction drain adjacent to the stump of the pancreas.

The second principle of operative management is to treat adjacent destructive injuries such as the aforementioned disruption of the head of the pancreas, devascularization of the C-loop of the duodenum, or destruction of the ampulla of Vater with a pancreatoduodenectomy.

The third principle of operative management is to consider adding a proximal diversion procedure such as a pyloric exclusion with gastrojejunostomy to treat adjacent nondestructive injuries described previously (see earlier section "Adjunctive Procedures: Pyloric Exclusion with Gastrojejunostomy").

Complications

The two largest civilian series of combined pancreatoduodenal injuries in the past 40 years are compared in Table 36-6.[18,19] In the older series (1987), 108 of 129 patients surviving longer than 48 hours had the following postoperative pancreatoduodenal-related complications: pancreatic fistula, 25.9%; intra-abdominal abscess, 16.6%; and duodenal fistula, 6.5%.[18] In the more recent series (2016), the following postoperative pancreatoduodenal-related complications were noted (number of patients surviving longer than 48 hours is unclear): 11 pancreatic fistulas and 3 cases of pancreatitis; 15 cases of "abdominal sepsis"; and 16 duodenal fistulas and 1 stenosis.[19]

Mortality

In the older series, the overall mortality rate was 29.4%, with 55% of these patients dying within 48 hours secondary to hypovolemic shock and transfusion-associated coagulopathies. Six or more intra-abdominal visceral or vascular injuries were present in 15 of the 21 patients with perioperative deaths. In the four patients who died in the perioperative period from 1981 to 1985, the average operative blood loss was 32 units.[18]

In the more recent series, the overall mortality rate was 28%, and the 21 deaths occurred at a median of 3 days after the injury (range, 1–42 days). Shock on presentation, increased composite grade of injury (see Table 1 in Krige et al[19]), associated vascular injuries overall, major visceral venous injuries, and the combination of vascular plus the total number of associated organs inured were all significantly related to mortality.[19]

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Colon and Rectum

Martin A. Croce • Timothy C. Fabian

KEY POINTS

- The presence of blood on digital examination of the anorectum is highly suggestive of injury, but its absence does not rule out injury to the colon or rectum.
- Primary repair of colonic injuries is safe and is the preferred option for nondestructive wounds.
- The management of patients with destructive colon wounds is controversial, but a prudent approach would be resection and anastomosis for stable patients without shock or other major comorbidities and diversion for unstable or high-risk patients.
- Degree of fecal contamination influences the probability of developing septic complications, including intra-abdominal abscess, but is not associated with an increased risk of suture line failure (anastomotic leaks).
- Patients with intraperitoneal penetrating rectal injuries should be managed the same as those with colon injuries. In patients with blunt injuries, proximal colostomy is usually the procedure of choice, as these injuries are frequently associated with pelvic fractures or complex perineal injuries.
- Extraperitoneal rectal injuries should be managed according to the anatomy of the injury. If the injury is accessible, it may safely be repaired primarily without diversion. If it cannot be repaired, then diversion is indicated.
- The anatomy of the rectal injury should dictate whether presacral drains be placed (selective use), but the practice of rectal washout cannot be supported by contemporary data.

INTRODUCTION

The management of injuries to the colon and rectum has changed dramatically through the years. Colon-related morbidity has decreased to approximately 15% to 20%, and mortality has decreased from approximately 90% during the Civil War to around 1% in the current literature.

Much of the early description of management for patients with colon and rectal injuries was from wartime experiences. These principles were then used for civilian patients initially. Over time, surgeons realized that civilian wounds were different from military ones, and new clinical approaches were devised. These evidence-based approaches have improved morbidity and mortality for civilian patients, as noted earlier.

Controversy still exists with respect to destructive wounds, location of colonic injury, blunt injury, abbreviated laparotomy, primary rectal repair, and presacral drainage. In this chapter, we will address these controversial issues and present a workable management scheme.

INJURIES TO THE COLON

Military and Civilian Experience

In order to understand the controversies that exist with management of colon wounds, it is important to review military and civilian experiences. The first report of a colon injury is recorded in the book of Judges when Ehud killed King Eglan. "And Ehud reached with his left hand, took the sword from his right side, and thrusted into his belly. And the hilt also went in after the blade, and the fat closed over the blade, for he did not pull the sword out of his belly; and the dung came out."[1]

Little was written about the management of colon injuries until the Civil War, when colonic wounds were almost uniformly fatal. Patients were typically managed without operations during that time. Unless a patient developed a fistula, death was likely.

As wars changed, so did the weaponry and injuries. World War I brought about more sophisticated guns with higher

velocity missiles, resulting in more destructive injuries. Management evolved from nonoperative to operative. Patients underwent either primary repair or diversion, but there was no universal policy for management of colon wounds. Despite the lack of uniformity in management, mortality rates fell to approximately 70%.[2]

Improvements in triage, resuscitation, blood banking, and antibiotics were evident in World War II. There was also standardization of the management of colon wounds. Major General William Heneage Ogilvie, a British surgeon who served in both World Wars, provided the basis for mandatory colostomy for all colon wounds: "The treatment of colon injuries is based on the known insecurity of suture and the dangers of leakage. Simple closure of a wound of the colon, however small, is unwarranted; men have survived such an operation, but others have died who would still be alive had they fallen into the hands of a surgeon with less optimism and more sense. Injured segments must either be exteriorized, or functionally excluded by a proximal colostomy."[3]

The concept of mandatory colostomy was then extended to civilian populations. Woodhall and Ochsner challenged that scheme in postwar New Orleans.[4] They realized the difference in weaponry and demonstrated that low-energy colonic wounds could safely be managed with primary repair. Despite their publication, most colon wounds continued to be managed with colostomy for the next 30 years. In 1979, Stone and Fabian[5] at Grady Memorial Hospital, Atlanta, Georgia, performed the first randomized trial comparing primary colon repair versus colostomy. They demonstrated the safety and superiority of primary repair for nondestructive injuries. Since then, debate has centered around the management of destructive colon wounds and injuries in those patients with "damage control" laparotomies.

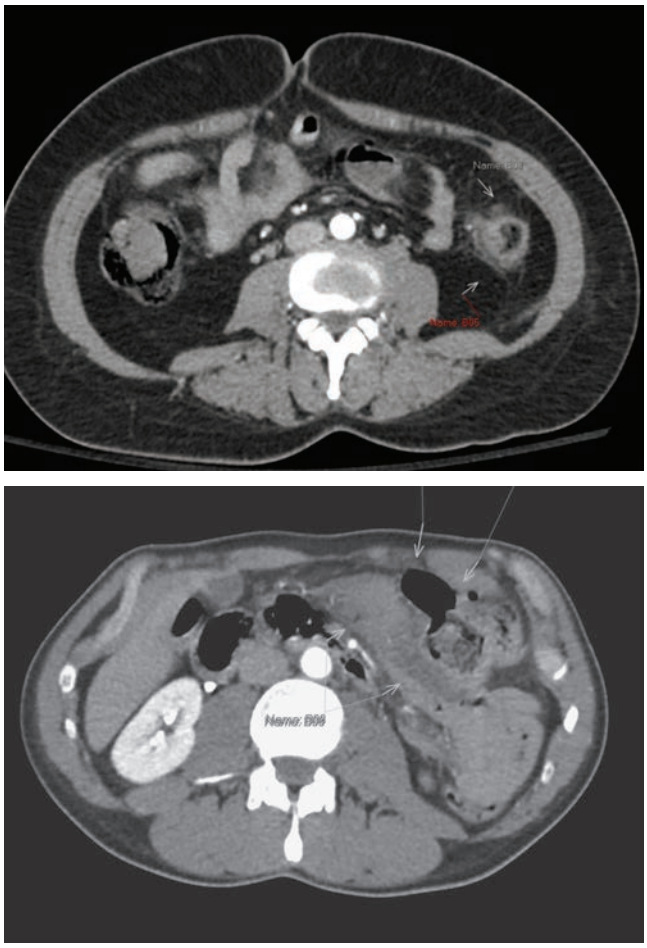

FIGURE 37-1 Thickened colon wall with small amount of surrounding fluid (top); thickened colon wall with pneumoperitoneum (bottom).

General Considerations

Following injury, the patient should undergo standard resuscitation with blood and blood products as necessary. For patients with penetrating abdominal wounds, extensive preoperative evaluation is not necessary. Selected patients with tangential wounds, wounds to the flank or back, or wounds to the right thoracoabdomen may be managed nonoperatively after appropriate imaging, but expedient operation is the best option for the majority of patients.[6]

The preoperative evaluation of patients with blunt abdominal trauma, however, is not as straightforward. Hemodynamically unstable patients with intra-abdominal fluid on a surgeon-performed ultrasound should undergo prompt operation. Stable patients are best evaluated by a contrast-enhanced computed tomography (CT) scan. An injury to the colon is suspected by a thickened colonic wall, usually with unexplained intraperitoneal fluid or even a pneumoperitoneum (Fig. 37-1). A digital rectal exam is imperative, as previously noted.[7]

The American Association for the Surgery of Trauma (AAST) has developed grading systems for various organ injuries. These Organ Injury Scales describe injury severity and allow for standardization of clinical research. In addition, management schemes may be designed based in part on the injury scales. The AAST colon injury and rectal injury scales are listed in Table 37-1.[8]

Operative Management: Nondestructive Colon Wounds

Nondestructive colon injuries include grade I and II and selected grade III injuries. Such injuries are typically low-energy wounds that do not require significant debridement. There is ample evidence that primary repair for nondestructive injuries is the optimal treatment. In the previously mentioned study by Stone and Fabian[5] in 1979, 139 patients with nondestructive colon wounds were randomized to either primary repair or colostomy. Improved outcomes with reduced intra-abdominal infection rates were documented in the primary repair group. In a follow-up study from the University of Tennessee–Memphis in 1989, George et al[9] evaluated 102 consecutive patients with penetrating colon injuries. All

TABLE 37-1: American Association for the Surgery of Trauma Organ Injury Scales for Colon and Rectum

Grade[a]	Type of injury	Description of injury
Colon Injury Scale		
I	Hematoma	Contusion or hematoma without devascularization
	Laceration	Partial thickness, no perforation
II	Laceration	Laceration <50% of circumference
III	Laceration	Laceration ≥50% of circumference without transection
IV	Laceration	Transection of the colon
V	Laceration	Transection of the colon with segmental tissue loss
	Vascular	Devascularized segment
Rectum Injury Scale		
I	Hematoma	Contusion or hematoma without devascularization
	Laceration	Partial-thickness laceration
II	Laceration	Laceration <50% of circumference
III	Laceration	Laceration ≥50% of circumference
IV	Laceration	Full-thickness laceration with extension into the perineum
V	Vascular	Devascularized segment

[a]Advance one grade for multiple injuries up to grade III.

Source: Adapted with permission from Moore EE, Cogbill TH, Malangoni MA, et al. Organ injury scaling II: pancreas, duodenum, small bowel, colon, and rectum. *J Trauma.* 1990;30:1427-1429.

were managed with either primary repair (83%) or resection and anastomosis (17%). There were no failures of primary repair and only one anastomotic leak. The authors identified transfusions, associated injuries, contamination, and the severity of the colon injury as independent predictors of abdominal infection, but none were risks for suture line leaks. In another randomized study, Chappuis et al[10] likewise demonstrated the safety of primary repair in 56 patients. Gonzales et al[11] randomized 109 patients to primary repair or diversion. They included patients regardless of shock, associated injuries, extent of fecal contamination, timing of surgery, and grade of colon injury. They found that primary repair was at least as successful as diversion and stated that all patients with penetrating colon injuries should undergo primary repair. A number of other prospective studies have also demonstrated that primary repair can be successfully performed in most patients with colon injuries.[12-25]

Thus, there is sufficient high-level evidence that primary repair of nondestructive colon wounds is the treatment of choice. Despite this, a survey of 342 surgeons in the United States demonstrated that colostomy was still preferred in 43% of patients with fecal contamination.[26] It appears that old habits die hard, despite the overwhelming evidence of safety for primary repair of nondestructive colon wounds.

Operative Management: Destructive Colon Wounds

As mentioned previously, destructive colon wounds include most grade III and all grade IV and V injuries.[27] Resection of injured tissue, taking care to ensure an adequate mesenteric blood supply, and anastomosis is the optimal treatment for these destructive injuries; however, whether to perform proximal diversion or even exteriorization of the injury as an end colostomy is the subject of debate. Stewart et al[27] evaluated 60 patients with destructive colon wounds managed by resection and anastomosis in 1994. They observed that patients who had significant medical comorbidities or those who received more than 6 units of blood had a suture line dehiscence rate of 42%, compared to 3% in patients who are otherwise healthy and received less than 6 units of blood. This was the first study to identify specific risk factors for anastomotic leak in patients with destructive colon injuries.

Subsequently, Cornwell et al[28] evaluated 25 patients with resection and anastomosis in 1998. They had an 8% leak rate, and these patients all had high transfusion requirements (>6 units). Murray et al[29] had similar results and conclusions in 140 patients.

In 2001, a prospective observational study sponsored by the AAST involving 297 patients from 19 centers was published.[30] All patients underwent resection; 66% had resection and anastomosis, and 34% had diversion. The authors stated that the method of colon management did not impact colon-related complications and suggested that all injuries requiring resection should be managed by anastomosis. While the concept has merit, there were differences in decision making among the 19 separate centers since there was no standardized protocol for management of the colon wound.

To evaluate a standard management protocol, the group from the University of Tennessee–Memphis performed a follow-up study 8 years after Stewart's original algorithm.[31] With adherence to the algorithm (Fig. 37-2), abdominal abscess rates decreased from 37% to 27%, suture line dehiscence dropped from 14% to 7%, and mortality decreased from 12% to 5%. An important observation was that the degree of fecal contamination was not associated with suture line failure, a finding confirming previous studies from the same institution.[9,32] Ten years later, the same group evaluated an additional 102 patients with destructive colon wounds.[30] Abscess rates further dropped to 18%, suture line failure to 5%, and mortality to 1%. The improvements over time seemed to be related to increased compliance with the algorithm, which was greater than 90% in the latest time period.

Thus, the evidence for management of patients with destructive colon wounds is somewhat less clear than for nondestructive wounds. It seems that prudent management would be resection and anastomosis for stable patients without shock or other major comorbidities and diversion for

unstable patients in shock or patients with chronic underlying illnesses.

Operative Management: Blunt Colon Wounds

Injuries to the colon following blunt abdominal trauma are uncommon, occurring in 0.1% to 0.5% of patients.[33,34] These injuries demand special consideration since they can be devastating. The degree of trauma imparted to the colon causing it to rupture is substantial, making these injuries particularly susceptible to ischemia. Approximately 50% of blunt colon injuries are grade III to V, thus necessitating resection.[35] Typical injuries include extensive serosal tears, mesenteric rents with devascularization, and perforations.

Sharpe et al[35] evaluated 151 patients with blunt colon injuries over a 13-year period. The management scheme was based on the original algorithm described by Stewart et al[27] (Fig. 37-2). Seventy-eight patients underwent primary repair, 44 had resection and anastomosis, and 29 underwent diversion. Compliance with the algorithm resulted in a suture line leak rate of 2%, compared to 13% in patients not managed according to the algorithm. These values are comparable to those following destructive penetrating wounds and compare favorably to other series of patients with blunt colon injuries.[36-38] Thus, even though the algorithm was developed for patients with penetrating injuries, it appears to be appropriate for patients with blunt injuries as well.

Operative Management: Abbreviated Laparotomy

In 1983, H. Harlan Stone described the concept of a limited laparotomy in unstable patients following initial control of hemorrhage and gastrointestinal contamination and then with temporary abdominal closure and transfer to the intensive care unit.[39] He termed the strategy *abbreviated laparotomy*. This was later referred to as *damage control laparotomy* by Rotondo et al.[40] This approach has been shown to be beneficial and to improve survival in patients with hypothermia, a metabolic acidosis, and/or a clinical coagulopathy.[39-45] Although this approach can be lifesaving, management of hollow organ wounds at a reoperation, especially the colon, can be challenging. Typically, these destructive wounds are resected, and the colon is left in discontinuity. After correction of "physiologic exhaustion," the surgeon must decide how to manage the colon at the time of a repeat laparotomy.

Miller et al[46] from Wake Forest described 11 patients with delayed anastomosis of the colon. None had suture line dehiscence, but almost half died during their hospitalization, making interpretation difficult. Weinberg et al[47] described 56 patients with abbreviated laparotomy over a 7-year period. A delayed anastomosis of the colon was performed in 33 and ostomy in the remaining 23, with a 12% leak rate. The authors cautioned against delayed anastomosis in the setting of abbreviated laparotomy. Another study from Denver Health described 25 patients with delayed anastomoses.[48] Their leak rate of 16% was higher than those reported earlier, but the authors concluded that delayed anastomosis was safe after a damage control laparotomy. In a larger series, Ott et al[49] reported 44 patients with delayed anastomosis with a failure rate of 27%. They concluded that the risk of leak was too high to justify delayed anastomosis. Other studies[50-53] have drawn fairly disparate conclusions following delayed anastomosis (Table 37-2).

The group from the University of Tennessee–Memphis reported a 17-year experience of 149 patients with colon wounds and abbreviated laparotomy.[54] Of these, 42 patients had a delayed anastomosis. No specific risk factors were identified in the 17% who developed leaks; however, the authors noted that 55% of the patients with delayed anastomoses were managed according to Stewart's protocol (Fig. 37-2). For patients not managed by the algorithm, the leak rate was 32%, compared to only 4% for those who were managed according to the algorithm (P = .03). Thus, a management

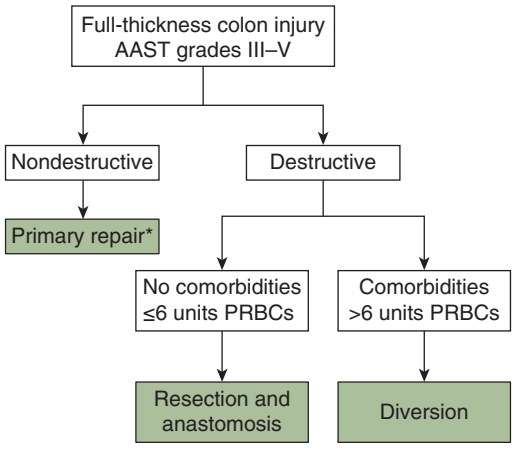

FIGURE 37-2 Management algorithm for full-thickness colon injuries. AAST, American Association for the Surgery of Trauma; PRBCs, packed red blood cells. *Primary repair for nondestructive wounds or destructive wounds in the absence of greater than 6 units of transfused blood or medical comorbidities.

TABLE 37-2: Suture Line Failure Rates for Patients with Delayed Colonic Anastomoses and Abbreviated Laparotomy

First author (reference)	Number of patients	% Leak
Miller[46]	11	0
Weinberg[47]	33	12
Kashuk[48]	29	16
Ott[49]	44	27
Vertrees[50]	10	10
Ordoñez[51]	27	7
Georgoff[52]	28	21
Burlew[53]	60	20
Sharpe[54]	42	17
Sharpe *without* algorithm	19	32
Sharpe *with* algorithm	23	4

scheme initially described for penetrating colon injuries in patients with a single laparotomy has been shown to be effective for patients with blunt colon injuries and those with delayed anastomoses following abbreviated laparotomy.

Risk Factors for Abdominal Complications

The overall incidence of abdominal septic complications in patients with colon injuries is approximately 20%. There are a number of risk factors for infection and suture line failure, and this distinction is critical.

LOCATION OF COLON WOUND

Historically, there has been concern that left-sided colon wounds carry an increased risk of colon-related complications, and suture lines should be avoided. In a multicenter study sponsored by the AAST, it was noted that over half of left-sided colon wounds were managed by diversion compared to only one-third of right-sided ones.[30] Military data have demonstrated a similar ratio in management of colon wounds; however, these data were not controlled.[53] A Western Trauma Association observational trial reported leak rates of 3% for right colon wounds, 20% for transverse colon wounds, and 45% for left colon wounds.[55] Similarly, these data were not controlled, making interpretation difficult, since it is unclear whether there is a true cause and effect or this is simply a measurement of surgeon preference. Dente et al[56] evaluated 217 patients with penetrating colon wounds and determined that injuries around the splenic flexure should be managed with caution due to the sometimes tenuous blood supply to this area. The group from Memphis evaluated 469 colon wounds and analyzed injury location and outcome.[57] They found that neither suture line dehiscence nor intra-abdominal abscess was associated with location of the colon injury. After adjusting for shock, injury severity, and operative management, location was still not associated with adverse outcomes (Table 37-3).

ASSOCIATED ABDOMINAL INJURIES

Despite the opinion that multiple associated injuries should mandate colostomy, there is sufficient evidence that these are associated with development of postoperative intra-abdominal abscesses, but *not* failure of the suture line in the colon.[5,9,11,27,28,32,35]

TRANSFUSIONS

Multiple blood transfusions are associated with the development of postoperative intra-abdominal abscesses[5,9,27,30,32,35] but are not associated with suture line failure for patients with primary repair of nondestructive colon wounds.[30,32,35] As mentioned previously, transfusions do influence the integrity of the suture line for patients with resection and anastomosis.[27,30,35,36,54]

FECAL CONTAMINATION

Peritoneal fecal contamination is associated with postoperative abdominal infection[5,9,11,27,28,32,35,36,58-60]; however, many surgeons have interpreted this fact to mean the contamination is also associated with dehiscence of the colon suture line. There are no data to suggest that fecal contamination is associated with failure of the colon suture line.[5,10,30,35,36] Thus, the practice of colostomy for fecal contamination alone following a penetrating injury to the colon should be abandoned. A summary of risk factors is shown in Table 37-4.

Technical Tips

As with all laparotomies for penetrating trauma, the initial primary goal is to control hemorrhage and limit contamination. After this is accomplished, injuries should be explored and prioritized. Colon injuries should be adequately mobilized, taking care to dissect the fat off the colon wall so that the full extent of the wound is visualized and debrided to viable tissue. For injuries amenable to primary repair, the authors prefer a two-layer closure with an inner layer of full-thickness 3-0 absorbable suture and an outer seromuscular closure with interrupted 3-0 silk Lembert sutures. For destructive wounds, the sites of resection should be chosen to ensure an adequate blood supply to the anastomosis; however, it is not necessary to adhere to oncologic principles. The exception to this principle is a destructive injury to the right colon. Segmental resection of the right colon is not advised due to its thin wall and marginal blood supply. Either a two-layer end-to-end or side-to-side handsewn anastomosis or a side-to-side stapled anastomosis may then be performed.

TABLE 37-3: Adjusted Odds Ratios and Confidence Intervals for Suture Line Failure and Abdominal Abscess Based on Colon Anatomy

	Suture line failure		Abdominal abscess	
Injury location	Adjusted OR	95% CI	Adjusted OR	95% CI
Right	0.96	0.25–3.69	1.39	0.78–2.48
Transverse	0.16	0.02–1.34	0.68	0.38–1.18
Left	3.36	0.91–12.42	1.31	0.71–2.42
Sigmoid	1.37	0.25–7.39	0.80	0.35–1.81

CI, confidence interval; OR, odds ratio.

TABLE 37-4: Factors Associated with Suture Line Failure and Abdominal Abscess

	Suture line failure	
	Primary repair	**Resection and anastomosis**
Location	—	—
Transfusions	—	↑↑
Contamination	—	—
Associated injuries	—	—
	Abdominal abscess	
Location	—	—
Transfusions	↑↑	↑↑
Contamination	↑↑	↑↑
Associated injuries	↑↑	↑↑

INJURIES TO THE RECTUM

Accepted management of penetrating rectal injuries evolved from the military experience in World War II and Vietnam.[3,61,62] These principles include routine fecal diversion, injury repair (if possible), distal rectal washout, and presacral drainage. As experience with civilian rectal injuries has increased, the utility of war-based principles has been challenged.

Anatomy

It is important to consider the anatomy of the rectum since it can influence management of the injury and outcome. The rectum is approximately 15 cm in length, and only the proximal two-thirds is intraperitoneal and covered with serosa. The lower one-third is extraperitoneal and technically difficult to expose, making primary repair difficult. The entire posterior aspect of the rectum is without a serosa to varying degrees.

General Considerations

The presence of blood on a digital anorectal examination is suggestive of an injury, as previously noted, and should trigger further investigation unless the patient is hemodynamically unstable and an immediate operation is indicated. Stable patients with blunt injury and selected patients with penetrating trauma should undergo CT scanning with contrast. If an injury is identified, prompt operation is indicated. If the CT clearly demonstrates no injury, a period of observation is appropriate in case of a false-negative study.

For stable patients with a penetrating injury and blood present on digital rectal exam, further investigation with sigmoidoscopy is indicated to possibly localize the injury.

Operative Management (Fig. 37-3)

Patients with intraperitoneal penetrating rectal injuries should be managed the same as those with colon injuries (ie, nondestructive wounds should be repaired primarily without proximal fecal diversion). As previously noted, proximal colostomy is usually the procedure of choice in patients with blunt injuries because these are often associated with pelvic fractures or complex perineal injuries.

Patients with extraperitoneal injuries should be managed according to the anatomy of the injury.[63,64] If the injury is accessible, it may safely be repaired primarily without diversion. If it cannot be repaired, then diversion is indicated.[63-69]

The literature suggests that the anatomy of the injury should dictate the use of presacral drains.[63,67] A large multicenter AAST

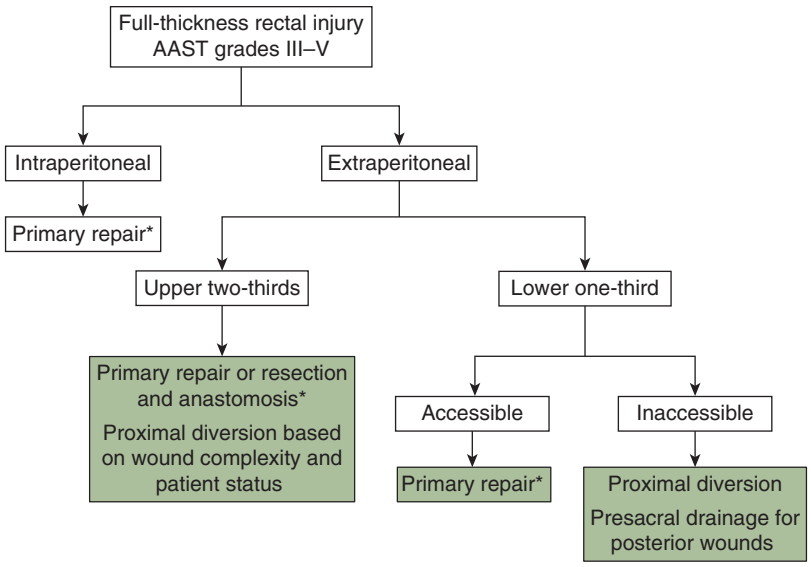

FIGURE 37-3 Management algorithm for full thickness rectal injuries. AAST, American Association for the Surgery of Trauma. *Primary repair for nondestructive wounds or destructive wounds in the absence of greater than 6 units of transfused blood or medical comorbidities.

study suggested that presacral drains were not indicated; however, the use of such drains was not controlled, making interpretation difficult.[65] Weinberg et al[64] also emphasized rectal anatomy as the primary determinant of presacral drain placement, following up a study by McGrath et al.[63] Wounds to the anterior part of the distal rectum will not benefit from posterior placement of presacral drains. Extraperitoneal wounds that are explored and repaired will effectively become intraperitoneal, thus precluding the need for presacral drainage. Extraperitoneal posterior wounds that are not explored are the injuries that will benefit from presacral (retrorectal) drainage.[64] This scheme is both intuitive and anatomically consistent, and the lack of benefit of presacral drainage from other studies is likely explained by this anatomic distinction.

The use of distal rectal washout was once part of the standard management of rectal injuries. There is no compelling evidence that it reduces morbidity, and this practice should be abandoned.[70,71]

Technical Tips

When performing preoperative sigmoidoscopy (either with a rigid or flexible scope), it is important to be prudent with insufflation. Too much insufflation makes subsequent abdominal exploration more difficult and potentially increases contamination from associated injuries, while too little will inhibit identification of the injury.

When the injury is located at the subsequent laparotomy, it is imperative to debride back to healthy tissue. All surrounding fat must be cleared to adequately evaluate the wound edges. A two-layer suture repair is appropriate, as previously described for colon wounds.

Placement of presacral drains, if anatomically indicated, should be done with the patient in the lithotomy position. A curvilinear incision between the anal sphincter and coccyx is made. Using a long clamp, the muscles of the pelvic floor and endopelvic fascia are penetrated until the sacral hollow is easily felt. Penrose drains are then placed in the presacral space and secured at the skin incision. These drains are gradually removed over several days beginning around postoperative day 3.

Ostomy

If a colostomy is deemed necessary, it is the authors' preference to perform a loop colostomy. There is no difference in function relative to an end colostomy if the loop is placed over a rod and the colotomy is made on the down side of the loop.[72] Creation of a loop allows for easier closure when the patient is ready. Timing of closure is debatable but typically occurs after 6 to 8 weeks.

Antibiotic Prophylaxis

As with all patients with penetrating abdominal trauma, preoperative antibiotics are mandatory. The choice of antibiotics must provide coverage against enteric aerobes and anaerobes including *Bacteroides fragilis*. Efficacy for single- or dual-agent therapy is equivalent as long as the regimen has coverage against enteric organisms. The duration of antibiotic prophylaxis is 24 hours.[32,73]

Management of the Incision

Primary skin closure following laparotomy in patients with colon or rectal injuries is not advised. Although tempting, primary skin closure is associated with significantly increased rates of wound infection and fascial dehiscence. All of these incisions are contaminated regardless of the amount of fecal spillage. In a prospective randomized trial, primary skin closure doubled the risk of infection when compared to delayed primary closure.[74] Newer agents and techniques, such as absorbable antimicrobials and negative-pressure wound dressings, may be promising, but controlled trials are necessary.

SUMMARY

The management of trauma to the colon and rectum can be quite challenging. Evidence-based practices have changed from mandatory colostomy to primary repair of nondestructive wounds and resection and anastomosis for destructive wounds in appropriate patient populations. Simplified management algorithms are effective and have been shown to improve patient outcomes.

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38

Abdominal Vessels

David V. Feliciano • Juan A. Asensio

KEY POINTS

- Patients with gunshot wounds to the abdomen will have injury to a named abdominal vessel 20% to 25% of the time.
- Abdominal vessel injuries will present with intraperitoneal hemorrhage; a contained mesenteric, retroperitoneal, or portal hematoma; thrombosis; or some combination of these.
- Options for management of abdominal vessel injuries include nonoperative (intimal flap on computed tomography angiogram), endovascular (hostile abdomen, delayed diagnosis, or failed prior repair), and operative.
- A midline supramesocolic hematoma is approached with a left-sided medial mobilization maneuver, whereas hemorrhage is approached directly through the lesser sac to the diaphragmatic aorta.
- The proximal superior mesenteric artery and common and external iliac arteries should always be shunted and *never* ligated during damage control operations.
- Ligation of the infrarenal inferior vena cava after a severe injury is more commonly practiced in the modern era and is usually well tolerated in young patients.
- Endovascular stents or stent grafts are commonly placed to cover intimal defects or restore arterial flow after blunt injury of the renal artery.
- An end-to-end anastomosis or interposition graft in the common or external iliac artery in the presence of significant enteric or colonic contamination has an increased rate of postoperative infection and subsequent arterial blowout.
- Survival rates after abdominal arterial injuries are as follows: suprarenal aorta, 8% to 24%; infrarenal aorta, 34% to 58%; superior mesenteric artery, 40% to 61%; and iliac artery, 60% to 80%.
- Survival rates after abdominal venous injuries are as follows: infrarenal inferior vena cava, 46% to 76%; superior mesenteric vein, 35% to 71%; iliac vein, 74% to 91%; and portal vein, 50%.

INTRODUCTION

Abdominal vessel injuries are among the most lethal injuries encountered by trauma surgeons because the vast majority of these patients arrive at trauma centers in profound hemorrhagic shock.[1-4] Patients sustaining abdominal vessel injuries best exemplify the lethal vicious cycle of shock, with secondary hypothermia, acidosis, and a coagulopathy.

The major sites of hemorrhage in patients sustaining blunt or penetrating abdominal trauma are the viscera, the mesentery, and the major abdominal vessels. The term *abdominal vascular or vessel injury* generally refers to injury to major intraperitoneal or retroperitoneal vessels and is classified into four zones described as follows and in Table 38-1:

- Zone 1: Midline retroperitoneum
  - Supramesocolic region
  - Inframesocolic region
- Zone 2: Upper lateral retroperitoneum
- Zone 3: Pelvic retroperitoneum
- Zone 4: Porta hepatis/retrohepatic inferior vena cava

Because most of the vessels in these areas are in the retroperitoneum, they are difficult to quickly access via a midline laparotomy incision. Therefore, a systematic operative approach is required to adequately diagnose and manage these potentially devastating injuries. A general discussion of epidemiology and methods of diagnosis, with subsequent descriptions of the operative management of abdominal vessel injuries within each region of the abdomen, follows.

EPIDEMIOLOGY

In reviews of vascular injuries sustained in older military conflicts, abdominal vascular injuries have been extraordinarily rare. For example, the classic article by DeBakey and Simeone

TABLE 38-1: Classification of Abdominal Vessel Injury

Zone	Major arterial branches	Major venous branches	Operative maneuvers[a]
1 (Supramesocolic)	Suprarenal aorta Celiac axis Superior mesenteric artery Proximal renal artery	Superior mesenteric vein	Left medial visceral rotation Midline suprarenal aortic exposure
1 (Inframesocolic)	Infrarenal aorta	Infrahepatic inferior vena cava	Midline infrarenal aortic exposure Right medial visceral rotation
2	Renal artery	Renal vein	Midline control of the renal hilum Lateral control of the renal hilum
3	Common, external, and internal iliac arteries	Common, external, and internal iliac veins	Midline control of iliac arteries and veins Isolation and control of common iliac vein/vena caval confluence Total pelvic isolation
4	Hepatic artery	Portal vein Retrohepatic vena cava	Portal exposure Exposure and control of retrohepatic inferior vena cava

[a]Discussed in sections titled "Exposure and Vascular Control."

on 2471 arterial injuries during World War II included only 49 that occurred in the abdomen, an incidence of 2%.[5] Reporting on 304 arterial injuries from the Korean conflict, Hughes[6] noted that only seven, or 2.3%, occurred in the iliac arteries. In the review by Rich et al[7] of 1000 arterial injuries in the Vietnam War, only 29, or 2.9%, involved abdominal vessels.

During Operation Iraqi Freedom and Operation Enduring Freedom (Afghanistan), the overall incidence of vascular injuries in American troops has been approximately 12%.[8] This figure is significantly higher than the 1% to 3% documented in the older military conflicts listed earlier. The incidence of abdominal vascular injuries increased significantly as well, accounting for 9.1% (143/1570) of all vascular injuries reported from both conflicts from January 2002 through September 2009.[9]

The data from civilian trauma centers are quite different. A review from Ben Taub Hospital in Houston, Texas, in 1982 revealed that 31.9% of all vascular injuries occurred in the abdomen, including 18.5% of all arterial injuries and 47.5% of all venous injuries.[10] A 30-year review (1958–1988) at the same hospital in 1989 documented that 33.8% of 5760 cardiovascular injuries occurred in the abdomen.[11] In the last 5 years of the period covered by the report (1984–1988), abdominal vascular injuries accounted for 27.3% of all cardiovascular injuries.

Over the past 30 years, many patients with abdominal vascular injuries have continued to be treated in civilian centers. Asensio et al[12] reported a series of 302 patients with 238 (47%) abdominal arterial and 266 (53%) abdominal venous injuries who underwent operative repair at the Los Angeles County Hospital (University of Southern California) from 1992 to 1997. Similarly, there were 300 patients with 205 abdominal arterial and 284 abdominal venous injuries who underwent operative repair at the Grady Memorial Hospital (Emory University) from 1989 to 1998 as reported by Davis et al.[13] Finally, there were 470 patients with 731 abdominal vascular injuries treated at Detroit Receiving Hospital (Wayne State University) from 1980 to 1997 as reported by Tyburski et al.[14]

The significantly higher number of abdominal vascular injuries treated in civilian as opposed to military practice likely reflects the modest wounding capacity of many handguns when compared with military ordnance, as well as the shorter prehospital transit times in most urban areas of the United States. Advances in torso-protecting military armor and the changing tactics of modern warfare have led to a shift in injuries to the extremities rather than the torso as well, although noncompressible (torso) hemorrhage remained the leading cause of combatant death from hemorrhage in one review.[15]

At present, the estimated incidence of injury to major abdominal vessels in patients sustaining blunt abdominal trauma is thought to be about 5% to 10%.[16,17] This is compared to patients with penetrating stab and gunshot wounds to the abdomen who will sustain a major abdominal vascular injury 10% and 20% to 25% of the time, respectively.[18]

PATHOPHYSIOLOGY

Blunt Trauma

Rapid deceleration in motor vehicle collisions may cause two different types of vascular injuries in the abdomen. The first is avulsion of small branches from major vessels, with subsequent *hemorrhage*. A common example of this is the avulsion of intestinal branches from either the proximal or distal superior mesenteric artery at sites of fixation. A second type

of vascular problem seen with deceleration injury is the development of an intimal tear with secondary *thrombosis* of the lumen, such as is seen in patients with renal artery thrombosis, or a full-thickness tear with a secondary traumatic false aneurysm of the renal artery.[19]

Crush injuries to the abdomen, such as by a lap seat belt, posterior blows to the spine, and any mechanism that causes significant anterior-to-posterior compression may cause two different types of vascular injury, also. The first is an intimal tear or flap with secondary *thrombosis* of a vessel, such as the superior mesenteric artery,[20-22] infrarenal abdominal aorta,[23,24] or iliac artery.[25-27] Direct blows can also completely disrupt exposed vessels, such as the left renal vein over the aorta[28] or the superior mesenteric artery or vein at the base of the mesentery,[29] leading to massive intraperitoneal *hemorrhage*, or they may even partly disrupt the infrarenal abdominal aorta, leading to the development of a traumatic false aneurysm.[23,24]

Penetrating Trauma

In contrast, penetrating injuries create the same kinds of abdominal vessel injuries as seen in the vessels of the extremities, producing blast effects with intimal flaps and secondary *thrombosis*, lateral wall defects with *hemorrhage* or pulsatile *hematomas* (early false aneurysms), or complete transection with either *free hemorrhage* or *thrombosis*.[30] On rare occasions, a penetrating injury may produce an arteriovenous *fistula* involving the portal vein and hepatic artery, renal vessels, iliac vessels, or superior mesenteric vessels.

Iatrogenic injuries to major abdominal vessels are an uncommon but persistent problem. Reported iatrogenic causes of abdominal vascular injury include diagnostic procedures (angiography, cardiac catheterization, laparoscopy), abdominal operations (pelvic and retroperitoneal procedures), spinal operations (removal of a herniated disk), and adjuncts to cardiac surgery (cardiopulmonary bypass, intraaortic balloon assist).[31-33]

DIAGNOSIS

History and Physical Examination

An abdominal vessel injury may present in one of four ways including free intraperitoneal *hemorrhage*; a contained mesenteric, retroperitoneal, or portal *hematoma*; *thrombosis* of the vessel; or some combination of these. As such, patients can be quickly divided into two major groups: those with ongoing hemorrhage and those without ongoing hemorrhage (contained hematoma or thrombosis). Thus, the presenting symptoms are variable based on both presentation and the involved vessel. After blunt trauma, for example, free intraperitoneal hemorrhage may be seen with avulsion of mesenteric vessels and lead to secondary hypovolemic shock. Conversely, when thrombosis of the renal artery is present, the patient will be hemodynamically stable but may complain of upper abdominal and flank pain and will commonly have hematuria (70%–80%).[34] Thrombosis of the infrarenal abdominal aorta will obviously cause pulseless lower extremities.

Penetrating truncal wounds between the nipples and the upper thighs remain the most common cause of abdominal vessel injuries. The exact vessel injured is generally related to the track of the missile or stab wound. For example, gunshot wounds directly on the midline most commonly involve the inferior vena cava or abdominal aorta. Gunshot wounds traversing the pelvis will often injure branches of the iliac artery or vein, while gunshot wounds in the right upper quadrant may involve the renovascular structures, vascular structures within the porta hepatis, or the infrahepatic or retrohepatic inferior vena cava.

On physical examination, the findings in patients with abdominal vessel injury will depend on whether a contained hematoma or active hemorrhage is present. Patients with contained hematomas at the base of the mesentery, in the retroperitoneum, or in the hepatoduodenal ligament, particularly those with injuries to abdominal veins, may be hypotensive in transit but often respond rapidly to the infusion of fluids. They may remain remarkably stable until the hematoma is opened at the time of laparotomy. These patients are potential candidates for the imaging studies mentioned in the next section. Conversely, patients with active hemorrhage, particularly those with injuries to abdominal arteries, generally have a rigid abdomen and unrelenting hypotension. Most of these patients will have an admission base deficit of –10 to –25 and will need 10 to 15 units of packed red blood cell transfusion in the perioperative period.[35] These patients should obviously undergo immediate laparotomy without further evaluation.

The other major physical finding that may be noted in patients with abdominal vascular injury is loss of the pulse in the femoral artery in one lower extremity when the ipsilateral common or external iliac artery has been transected or is thrombosed. In such patients, the presence of a transpelvic gunshot wound associated with a wavering or an absent pulse in a femoral artery is pathognomonic of injury to the ipsilateral iliac artery.

Imaging

In both stable and unstable patients, a rapid surgeon-performed ultrasound (focused assessment for the sonographic evaluation of the trauma patient [FAST]) is useful in ruling out an associated cardiac injury with secondary tamponade or an associated hemothorax mandating the insertion of a thoracostomy tube.[36-39] In a *stable* patient with an abdominal gunshot wound, a routine flat-plate x-ray of the abdomen is of diagnostic value, so that the track of the missile can be predicted from markers placed over the wounds or from the position of a retained missile.

In patients with blunt abdominal trauma, hematuria, modest hypotension, and peritoneal signs in the emergency department, computed tomography (CT) scanning of the abdomen has documented that the absence of renal enhancement and excretion and the presence of a cortical rim sign are

diagnostic of thrombosis of the renal artery; therefore, selective renal arteriography is no longer indicated for this diagnosis. Similarly, any stable patient with blunt trauma who does not require an immediate laparotomy and who has significant hematuria should undergo a CT cystogram as well.

In other patients with blunt trauma, CT angiography is used to diagnose and treat deep pelvic arterial bleeding associated with fractures and to diagnose unusual injuries such as the previously mentioned intimal tears with or without thrombosis in the infrarenal aorta, the superior mesenteric artery, the renal artery, or the iliac artery.

As the technology of CT scanning has advanced, many surgeons and radiologists are comfortable making therapeutic decisions based on data acquired from multiplanar scanning and formal CT angiography.[40] In one study in 2007, contrast-enhanced CT alone had a 94% sensitivity and 89% specificity for the diagnosis of active hemorrhage when compared with angiography.[41] Most of the positive scans involved branches of the internal iliac artery with a concomitant pelvic fracture or injuries to solid organs, and thus were not necessarily diagnostic of true *abdominal vessel injury.* Still, in the stable patient with blunt trauma, findings on CT that are suggestive of injury to the retroperitoneal great vessels mandate nonoperative management with CT angiographic follow-up, endovascular management, or operation.

INITIAL MANAGEMENT AND RESUSCITATION

Prehospital Resuscitation

Resuscitation in the field in patients with possible penetrating or blunt abdominal vessel injuries should be restricted to basic airway maneuvers such as intubation or cricothyroidotomy and decompression of a tension pneumothorax at the scene. Insertion of intravenous lines for infusing crystalloid solutions and blood products is best attempted during transport to the hospital. Restoration of blood pressure to reasonable levels is critical to neurologic recovery in patients with associated blunt intracranial injuries and possible abdominal injuries.[42] In contrast, there is no consistent evidence to support the aggressive administration of crystalloid solutions during the short prehospital times in urban environments. Although the data on permissive hypotension in injured patients with shock after penetrating trauma are encouraging, its role in multisystem blunt trauma and its effect on coagulation are unclear. Even so, a key component of "damage control resuscitation" as espoused by the US military and discussed later is minimization of early crystalloid resuscitation.[43-46]

Emergency Department Resuscitation

In the emergency department, the extent of resuscitation clearly depends on the patient's condition at the time of arrival. In the agonal patient with a rigid abdomen after penetrating or blunt (if the admission chest x-ray is not suggestive of an injury to the descending thoracic aorta) trauma, resuscitative endovascular balloon occlusion of the aorta (REBOA) is now performed in trauma centers around the world in preference to emergency department thoracotomy (see Chapter 13).

A REBOA device inserted through a 7F sheath mimics a cross-clamp on the descending thoracic aorta in the following ways: (1) preserves available blood supply to the coronary and carotid arteries; (2) decreases arterial bleeding from injuries to the abdomen, pelvis, and lower extremities; and (3) possibly decreases bleeding from abdominal venous injuries.[47-52]

In the patient arriving with blunt abdominal trauma, significant hypotension, and a positive surgeon-performed FAST or penetrating abdominal trauma and hypotension, peritonitis, or evisceration, a *time limit of less than 5 minutes in the emergency department is mandatory.* An identification bracelet is applied; an airway and thoracostomy tube are inserted if necessary to maintain vital signs, especially if the operating room is geographically distant; and a blood sample for type and cross-match is obtained with the insertion of the first intravenous catheter. Whether more intravenous lines should be inserted in the emergency department or after arrival in the operating room is much debated. The authors have always believed that patients needing an emergency laparotomy should be in the operating room as soon as the identification bracelet has been applied and a blood specimen has been sent to the blood bank.

With short, large-bore (10-gauge or number 8.5F) catheters in peripheral veins for resuscitation, flow rates of 1400 to 1600 mL/min of crystalloid solutions can be obtained when an external pressure device is exerting 300 mm Hg of pressure. Blood replacement during resuscitation is usually with type-specific blood, although universal donor type O negative blood may be used when there is no time for even a limited cross-match.

Measures in the emergency department that will diminish the hypothermia of shock in patients with abdominal vessel injuries include the following: a heated resuscitation room, the use of prewarmed (37–40°C or 98.6–104.0°F) crystalloid solutions if blood is not available, passage of all crystalloids and blood through high-flow warmers, and covering the patient's trunk and extremities with prewarmed blankets or heating units.

Damage Control Resuscitation and Massive Transfusion

In the past 15 years, based mostly on the military experience during the conflict in Iraq, there has been a dramatic change in the resuscitation philosophy of critically injured patients in many centers. The military resuscitation philosophy of damage control resuscitation is seen as an extension of the concepts of damage control surgery, a term first used in 1993 by Rotondo et al.[53] The cornerstone of damage control resuscitation is the initial use of either fresh whole blood or an idealized ratio of packed red blood cells to fresh frozen plasma to platelets of 1:1:1. With control of hemorrhage or the onset of an intraoperative coagulopathy, further infusion of blood and

components is guided by thromboelastography or rotational thromboelastometry (see Chapters 13, 15, and 16).

OPTIONS FOR MANAGEMENT OF INJURIES TO ABDOMINAL VESSELS

In patients with possible abdominal trauma, especially blunt, and no obvious indication for a laparotomy, a CT is usually performed in those with the following: (1) equivocal physical examination; (2) altered consciousness secondary to acute alcohol intoxication or use of illicit drugs; (3) associated injuries to the brain or spinal cord; and (4) associated injuries to lower ribs, thoracolumbar spine, or pelvis. Even in patients requiring continuous transfusion to maintain a "reasonable" blood pressure after multisystem blunt trauma, particularly when a pelvic fracture is present, a contrast-enhanced CT is performed.[54,55] Such a study will document an intimal injury or thrombosis of a major named artery, as mentioned previously.

An early diagnosis of an injury to a major named abdominal vessel in a hemodynamically stable or reasonably stable patient allows for several options in management.

Nonoperative

Much as in trauma to cervical, thoracic, and peripheral vessels, an intimal flap or mural hematoma that is not flow limiting may be observed in selected patients (ie, not every wall injury needs to be stented or undergo operation). The risk is progression of the wall injury and secondary thrombosis of the artery. Should observation be chosen, one option is to perform an early (48 hours) repeat CT angiogram to see if there has been further progression of the injury. Otherwise, a repeat CT angiogram should be performed in the asymptomatic patient before discharge. With either repeat CT angiogram, evidence of progression of the injury and a decrease in arterial flow mandate choosing an endovascular or operative approach.

Endovascular

There has been extensive experience with the insertion of endostents for intimal flaps, intramural hematomas, and luminal thromboses in major named abdominal vessels after trauma for over two decades.[56] In similar fashion, endostent grafts have been used to treat wall defects resulting in early pulsatile hematomas, traumatic false aneurysms, and arteriovenous fistulas. Finally, interventional vascular surgeons routinely embolize bleeding vessels in solid organs and the pelvis in the modern era.

Endovascular approaches for injuries to abdominal vessels are particularly appealing in patients without another indication for laparotomy in the following circumstances: (1) associated injury to the brain, associated extensive burns, or early organ failure; (2) "hostile" abdomen from prior laparotomies; (3) delayed diagnosis; or (4) patient returns with failed operative repair or chronic missed vessel injury.

Operative

Detailed descriptions of injuries to major abdominal vessels have been available for 100 years.[57] Operative exposures and/or options for management in civilians have been described and refined since the classic wartime papers by the following: (1) Michael E. DeBakey and Fiorindo Simeone in 1946 (World War II)[5]; (2) Carl W. Hughes, John M. Howard, and Frank C. Spencer from 1954 to 1958 (Korean War)[6,58-60]; and (3) Norman M. Rich in 1970 (Vietnam War).[7]

OPERATIVE PREPARATIONS

Draping and Incisions

In the operating room, the entire trunk from the chin to the knees is prepared and draped in the usual manner. Before making the midline incision for a laparotomy, the trauma surgeon should confirm that the following items are available: blood components for transfusion, blood salvaging device, autotransfusion apparatus, a thoracotomy tray, an aortic compressor, a complete tray of vascular instruments, intravascular shunts, sponge sticks with gauze sponges in place for venous compression, and appropriate vascular sutures.

Maneuvers to Prevent or Decrease Hypothermia

In addition to the maneuvers previously described for preventing hypothermia in the emergency department, operative maneuvers with the same purpose include the following: warming the operating room to more than 85°F (29.4°C); covering the patient's head; covering the upper and lower extremities with a heating unit (Bair Hugger Normothermia System, 3M Company, Maplewood, MN); irrigation of nasogastric tubes, thoracostomy tubes, and open body cavities with warm saline; and use of a heating cascade on the anesthesia machine.[61]

General Principles

Insertion of a REBOA device (in aortic zone 1 for abdominal bleeding or in aortic zone 3 for pelvic bleeding) should be considered in any injured patient with a systolic blood pressure of less than 90 mm Hg who is a partial responder or nonresponder to initial resuscitative attempts. A resuscitative thoracotomy would still be indicated in the following situations: (1) REBOA device is not available or cannot be passed into the thoracic aorta; (2) failure of the patient's blood pressure to respond to REBOA; or (3) ultrasound diagnosis of pericardial tamponade or exsanguination through a thoracostomy tube.

A midline abdominal incision from xiphoid to pubis is made, and all clots and free blood are manually evacuated or removed with suction. A rapid inspection is performed to visualize contained hematomas or areas of hemorrhage in the retroperitoneum, base of the mesentery, or porta hepatis. Active hemorrhage from solid organs is controlled by packing, while standard techniques of vascular control are used to control the active hemorrhage from major intra-abdominal vessels. Digital pressure, compression with laparotomy pads, grabbing the perforated artery with a hand (common or external iliac artery), or formal proximal and distal control is needed to control any actively hemorrhaging major artery. Similarly, options for control of bleeding from major veins such as the inferior vena cava, superior mesenteric vein, portal vein, renal veins, or iliac veins include digital pressure, compression with laparotomy pads or sponge sticks, grabbing the perforated vein with a hand, applying Judd-Allis clamps to the edges of the perforation,[62] and the application of vascular clamps. Once hemorrhage from the vascular injury is controlled in a patient with a penetrating wound, it may be worthwhile to rapidly apply Babcock clamps, Allis clamps, or noncrushing intestinal clamps or to rapidly use a surgical stapler to control as many gastrointestinal perforations as possible. This avoids further contamination of the abdomen during the period of vascular repair. The abdomen is irrigated with an antibiotic–saline solution, the vascular repair is then performed, a soft tissue cover is applied over the repair, and the remainder of the operation is directed toward repair of injuries to the bowel and solid organs.

Conversely, if the patient has a nonexpanding retroperitoneal, mesenteric, or portal hematoma at the time of laparotomy, the surgeon occasionally has time to first perform necessary gastrointestinal repairs, change gloves, and irrigate with an antibiotic–saline solution. The surgeon can then open the retroperitoneum, mesentery, or porta hepatis to expose the underlying abdominal vessel injury. The magnitude of injury is best described using the Organ Injury Scale (OIS) of the American Association for the Surgery of Trauma (AAST).[63]

MANAGEMENT OF INJURIES IN ZONE 1: SUPRAMESOCOLIC REGION

Exposure and Vascular Control

The midline retroperitoneum of zone 1 is divided by the transverse mesocolon into a *supramesocolic* region and an *inframesocolic* region. If a hematoma or hemorrhage is present in the midline supramesocolic area, injury to the *diaphragmatic aorta, suprarenal aorta, celiac axis, proximal superior mesenteric artery*, or *proximal renal artery* should be suspected. In such patients, there are several techniques for obtaining proximal vascular control of the aorta at the aortic hiatus of the diaphragm. When a contained *hematoma* is present, as it may be with wounds to the aorta in the aortic hiatus (diaphragmatic aorta), the surgeon usually has time to reflect all left-sided intra-abdominal viscera, including the colon, kidney, spleen, tail of the pancreas, and fundus of the stomach to the midline

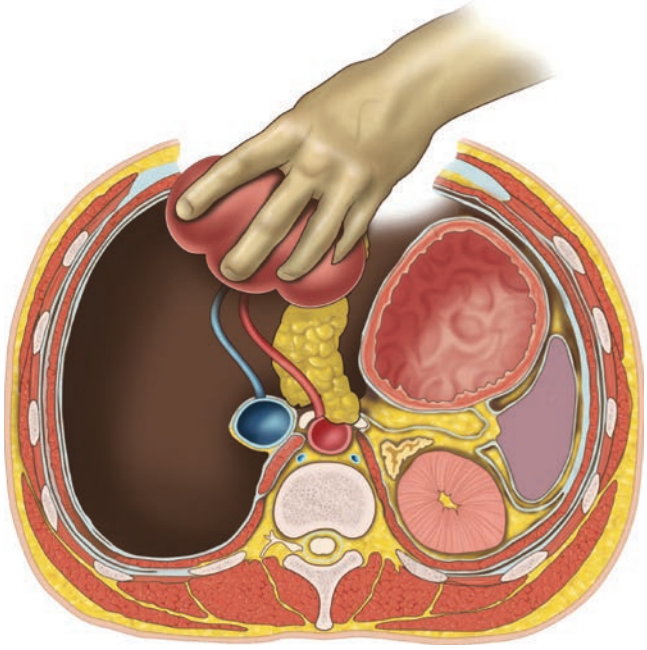

FIGURE 38-1 Completion of left medial visceral mobilization after elevation of left colon, left kidney, spleen, tail of the pancreas, and fundus of the stomach.

(left-sided medial visceral rotation). This maneuver was originally described by DeBakey et al,[64] applied by Elkins et al,[65] and modified by Mattox et al[66] (Fig. 38-1). The advantage of this technique is that it provides extensive visualization of the entire abdominal aorta from the aortic hiatus of the diaphragm to the aortic bifurcation. Disadvantages include the time required to complete the maneuver (5–7 minutes); risk of injury to the spleen, left kidney, or posterior left renal artery during the maneuver; and a fold in the aorta that results when the left kidney is rotated anteriorly.[67] One alternative is to leave the left kidney in its fossa, thereby eliminating potential damage to the kidney or decreasing renal blood flow resulting from rotation of this structure.

Because of the dense nature of the celiac plexus of nerves connecting the right and left celiac ganglia as well as the lymphatics that surround the supraceliac aorta, it is frequently helpful to transect the left crus of the aortic hiatus of the diaphragm and the adjacent diaphragm at the 2 o'clock position to allow for exposure of the distal descending thoracic aorta superior to the hiatus.[67,68] With the distal descending thoracic aorta in the hiatus exposed, a supraceliac aortic clamp such as a DeBakey or DeBakey-Crafoord can be applied. This allows for the extra few centimeters of exposure that are essential for complex repair of the vessel within this tightly confined anatomic area.

Conversely, if active *hemorrhage* is identified from this area, the surgeon may attempt to control it manually or with one of the aortic compression devices. An alternate and rapid approach is to divide the lesser omentum manually, retract the stomach and esophagus to the left, and digitally separate the muscle fibers of the aortic hiatus of the diaphragm from

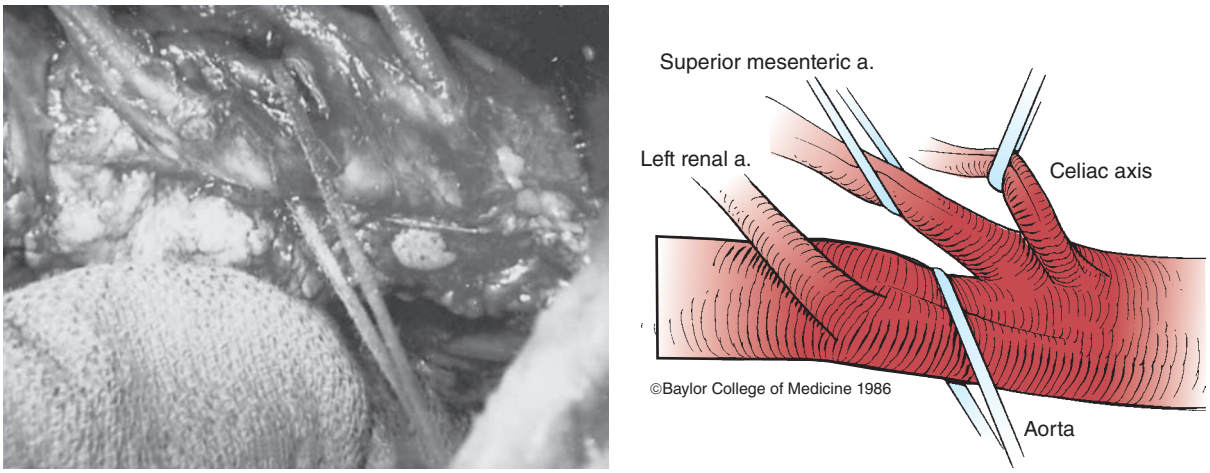

FIGURE 38-2 (**A**) View of suprarenal aorta and major branches after left-sided medial mobilization maneuver. (**B**) Diagrammatic representation of structures with labels. (Reproduced with permission from Baylor College of Medicine.)

the diaphragmatic aorta. This allows for anterior exposure for application of an aortic cross-clamp rather than the left-sided exposure from medial visceral rotation.[69] After either approach to the suprarenal abdominal aorta, cross-clamp time should be minimized to avoid the primary fibrinolytic state that occurs, presumably due to hepatic hypoperfusion.[70] Therefore, the time of placement of the clamp should be noted. Distal control of the aorta in this location is awkward because of the presence of the celiac axis and superior mesenteric artery (Fig. 38-2). In some patients with injury confined to the supraceliac aorta, the celiac axis may have to be divided and ligated to allow for more space for the distal aortic clamp and subsequent vascular repair. Necrosis of the gallbladder is a likely sequela, and cholecystectomy is generally warranted, although this may be performed at repeat exploration when damage control techniques are required.[71]

Suprarenal Aorta

With small perforating wounds to the aorta at this level, lateral aortorrhaphy with 3-0 or 4-0 polypropylene suture is preferred. If two small perforations are adjacent to one another, they should be connected by incising them with a Potts scissors and the defect closed in a transverse fashion, if possible, with polypropylene sutures. When closure of the perforations results in significant narrowing or if a portion of the aortic wall is missing, patch aortoplasty with polytetrafluoroethylene (PTFE) is indicated.

On occasion, a profoundly hypotensive patient with an extensive injury to the diaphragmatic or supraceliac aorta will require insertion of a thoracostomy tube as a temporary intraluminal shunt.[1] If the patient is hemodynamically stable at a reoperation, options for a synthetic interposition graft include Dacron, albumin-coated Dacron, or PTFE.[72,73] When there are concerns about placing a synthetic vascular graft in the presence of extensive gastrointestinal contamination, options described in the literature have included the following: (1) portion of infrarenal inferior vena cava[74]; (2) handmade spiral vein graft[75]; and (3) handmade bovine pericardium tubular graft. There is lack of follow-up data on the few patients who have survived insertion of a prosthetic graft into an injured suprarenal (or infrarenal) abdominal aorta, so the infection rate is unknown.

As previously noted, repairs of the intestine and the aorta with a significant injury should not be performed simultaneously. Once the perforated bowel has been packed away and the surgeon has changed gloves, the aortic prosthesis is sewn in place with 3-0 or 4-0 polypropylene suture. After appropriate flushing of both ends of the aorta and removal of the distal aortic clamp, the proximal aortic clamp should be removed very slowly as the anesthesiologist rapidly infuses fluids. If a long aortic clamp time has been necessary, the prophylactic administration of intravenous bicarbonate is indicated to reverse the "washout" acidosis and hyperkalemia from the previously ischemic lower extremities. The retroperitoneum is then copiously irrigated and closed in a watertight fashion with an absorbable suture.

Cross-clamping of the abdominal aorta at any level in a patient with hemorrhagic shock results in severe ischemia of the lower extremities. Restoration of arterial inflow will then cause a reperfusion injury with its physiologic consequences. In a patient who is hemodynamically stable after repair of the suprarenal (or infrarenal) abdominal aorta, measurement of compartment pressures below the knees should be performed before the patient is moved from the operating room. Pressures in the range of 30 to 35 mm Hg should be treated with below-knee, two-incision, four-compartment fasciotomies.[76] Measurement of the pressure in the anterior compartments of the thighs is worthwhile, as well.

The survival rate of patients with penetrating injuries to the suprarenal abdominal aorta in the distant past was 35%[77-83]; however, more recent reviews have documented a decline in survival (Table 38-2). In one series from 2001 in which injuries to the suprarenal and infrarenal abdominal aorta were

TABLE 38-2: Survival with Injuries to the Abdominal Aorta[a]

	Asensio et al[12]	Davis et al[13]	Tyburski et al[14]	Deere et al[84]
Abdominal aorta overall	36.1 (13/63)	39.1% (25/64)	21.9% (15/71)	33.8% (16/42)
Isolated	21.7% (10/46)	—	—	—
With other artery	17.6% (3/17)	—	—	—
Suprarenal	—	—	8.3% (3/36)	24% (6/25)
Infrarenal	—	—	34.2 (12/35)	58.8% (10/17)

[a]Excludes patients with exsanguination before repair or ligation.

separated, the survival rate in the suprarenal group was only 8.3% (3/36).[14] Another review from 2007 noted that the survival rate in 24 patients (80% penetrating wounds) with injuries to the "subdiaphragmatic" or "suprarenal" aorta was 24% if patients who were dead on arrival were excluded.[84] The reasons for this decrease in survival figures are not defined in the reviews described, although the most likely cause is the shorter prehospital times secondary to improvements in emergency medical services. This brings many patients who would otherwise not survive transit to the trauma center to die in the same.[85]

Celiac Axis

Injury to the celiac axis is rare. In the 2005 review by Asensio et al,[86] only 13 patients with this uncommon injury were treated. Penetrating injuries were the cause in 12 patients, and overall mortality was 62%. Eleven patients were treated with ligation and one with primary repair, with the final patient exsanguinating prior to treatment. Of the five survivors, four had undergone ligation, and all deaths occurred in the operating room. This group also performed an extensive literature review and could only document 33 previously reported cases, all the result of penetrating trauma. Furthermore, they could find no survivor treated with any sort of complex repair. Given these results, patients with injuries to the celiac axis that are not amenable to simple arteriorrhaphy should undergo ligation, which should not cause any short-term morbidity other than the aforementioned risk of gallbladder necrosis.[71] The collateral circulation between the celiac axis and the superior mesenteric artery will maintain viability of the viscera in the foregut. If in doubt, a second look laparotomy should be performed.

When branches of the celiac axis are injured, they are often difficult to repair because of the dense neural and lymphatic tissue in this area and the small size of the vessels in patients with profound shock and secondary vasoconstriction. There is clearly no good reason to repair major injuries to either the left gastric or proximal splenic artery in the patient with trauma to this area. In both instances, these vessels should be ligated. The common hepatic artery may have a larger diameter than the other two vessels, and an injury to this vessel may occasionally be amenable to lateral arteriorrhaphy, end-to-end anastomosis, or the insertion of a saphenous vein or prosthetic graft. In general, however, one should not worry about ligating the hepatic artery proximal to the origin of the gastroduodenal artery, since the extensive collateral flow from the midgut through this vessel will maintain the viability of the liver.

Superior Mesenteric Artery

Injuries to the superior mesenteric artery are managed based on the level of injury. In 1972, Fullen et al[87] described an anatomic classification of injuries to the superior mesenteric artery that has been used intermittently by subsequent authors in the trauma literature.[21,22] If the injury to the superior mesenteric artery is beneath the pancreas (Fullen zone 1), the pancreas may have to be transected between Glassman or Dennis intestinal clamps or GIA or TA staplers to locate and control the bleeding points. Because the superior mesenteric artery has few branches at this level, proximal and distal vascular control is relatively easy to obtain once the overlying pancreas has been divided. Another option is to perform medial rotation of the left-sided intra-abdominal viscera, as previously described, and apply a clamp directly from the left side of the aorta to the proximal superior mesenteric artery at its origin. In this instance, the left kidney may be left in the retroperitoneum as the medial rotation is performed. It is important to remember that the celiac axis and superior mesenteric artery have a "v" conformation when approached from the left side (Fig. 38-2).

Injuries to the superior mesenteric artery also occur beyond the pancreas at the base of the transverse mesocolon (Fullen zone 2, between the pancreaticoduodenal and middle colic branches of the artery). Although there is certainly more space in which to work in this area, the proximity of the pancreas and the potential for pancreatic leaks near the arterial repair make injuries in this location almost as difficult to handle as the more proximal injuries.[22,88] If the superior mesenteric artery has to be ligated at its origin from the aorta or beyond the pancreas (Fullen zone 1 or 2), collateral flow from both the foregut and hindgut should theoretically maintain the viability of the midgut in the distribution of this vessel.[89] Profound vasoconstriction of the visceral vessels, however, may compromise the viability of distal segments of the small bowel and the right colon. In the hemodynamically unstable patient with hypothermia, acidosis, and a coagulopathy, the

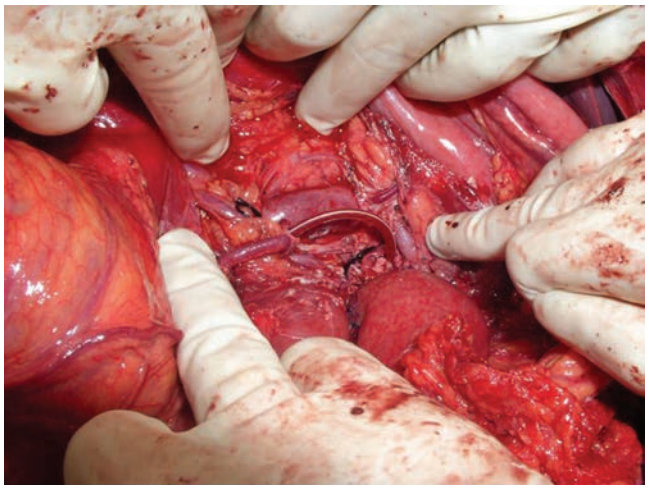

FIGURE 38-3 Argyle shunt in proximal superior mesenteric artery of patient with gunshot wound to upper abdomen.

insertion of a temporary intraluminal shunt into the debrided ends of the superior mesenteric artery is most appropriate and fits the definition of *damage control*[90] (Fig. 38-3). If replacement of the proximal superior mesenteric artery is necessary in a more stable patient or at a reoperation after a damage control procedure, it is safest to place the origin of the saphenous vein or prosthetic graft on the inframesocolic distal infrarenal aorta, away from the pancreas and other upper abdominal injuries (Fig. 38-4). A graft in this location should be tailored so that it will pass through the posterior aspect of the mesentery of the small bowel and then be sutured to the superior mesenteric artery in an end-to-side fashion without significant tension. It is mandatory to cover the aortic suture line with retroperitoneal fat or a viable omental pedicle to avoid an aortoduodenal or aortoenteric fistula at a later time. This is much easier to perform if the proximal origin of the graft is located on the distal aorta. Injuries to the more distal superior mesenteric artery (Fullen zone 3, beyond the middle colic branch, and zone 4, at the level of the enteric branches) should be considered for repair, since ligation in this area is distal to the connection to collateral vessels from the foregut and the hindgut.[91] Because this may require microsurgical techniques, however, it is essentially never performed, and ligation may mandate extensive resection of the ileum and right colon.[92]

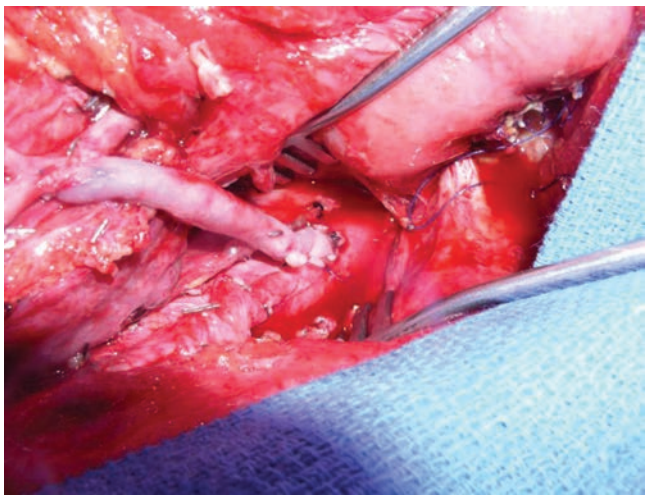

FIGURE 38-4 Same patient as in Fig. 38-3. Shunt has been removed, proximal superior mesenteric artery has been ligated, and view is of aorto-superior mesenteric artery (posterior aspect) bypass graft with autogenous saphenous vein.

The survival rate of patients with penetrating injuries to the superior mesenteric artery in the distant past was 57.7% (Table 38-3).[80,82,87,88,93-95] In four separate reviews from 2001, including a large multi-institutional study,[22] the mean survival was 58.7%.[12-14,22] In one of the older series, survival decreased to 22% when any form of repair more complex than lateral arteriorrhaphy was performed.[88] Independent risk factors for mortality in the multi-institutional study by Asensio et al[22] included injury to Fullen zone 1 or 2, transfusion of 10 units of packed red blood cells, intraoperative acidosis or dysrhythmias, and multisystem organ failure.

ENDOVASCULAR

Most injuries to the proximal superior mesenteric artery are perforations or blunt avulsions with hemorrhage or early pulsatile hematomas. Therefore, endovascular stent grafts have been used most commonly for treatment after late diagnosis of intimal flaps, stenoses, or superior mesenteric arteriovenous fistulas.[96-98]

Proximal Renal Arteries

Injuries to the proximal renal arteries may also present with a zone 1 supramesocolic hematoma or with hemorrhage in this area. The left medial visceral rotation maneuver described earlier allows visualization of much of the left renal artery from the aorta to the kidney. This maneuver does not, however, allow for visualization of the proximal right renal artery. The proximal vessel is best approached through the base of the mesocolon beneath the left renal vein and between the infrarenal abdominal aorta and inferior vena cava. Vascular control may be achieved with DeBakey or Henly clamps. Options for repair of either the proximal or distal renal artery are described later in this chapter (see section "Management of Injuries in Zone 2").

Superior Mesenteric Vein

One other major abdominal vessel, the proximal superior mesenteric vein, lying to the right of the superior mesenteric artery, may be injured at the base of the mesocolon. Injury to the most proximal aspect of this vessel near its junction with the splenic vein is difficult to manage. The overlying pancreas, proximity of the uncinate process, and close association with the superior mesenteric artery often preclude easy access to proximal and distal control of the vein. Therefore, as with injuries to the proximal superior mesenteric artery, the neck

TABLE 38-3: Survival with Injuries to the Superior Mesenteric Artery

Reference	Year	No. of patients	No. of survivors	Survival (%)
Six series[80,87,88,93-95]	1972–1986	116	67	57.7
Asensio et al[12] (Los Angeles County)	2000	27 (isolated injury)	11	40.7
		7 (with other artery)	2	28.6
Asensio et al[22] (multi-institutional)	2001	223	143	61
Davis et al[13]	2001	15	8	53.3
Tyburski et al[14]	2001	41	20	48.8

of the pancreas may have to be transected between noncrushing vascular or intestinal clamps or GIA and/or TA staplers to gain access to a distal injury to the vein. More commonly, the surgeon will find an injury to this vessel inferior to the lower border of the pancreas. Vascular control may be achieved with a small Cooley or Satinsky partial occlusion clamp. Often, the vein can be compressed manually or squeezed between the surgeon's fingers as an assistant places a continuous row of 5-0 polypropylene sutures into the edges of the perforation. If a posterior perforation is present, multiple collaterals entering the vein at this point will have to be ligated to roll the perforation into view. Occasionally, the vein will be nearly transected, and both ends will have to be controlled with vascular clamps. With an assistant pushing the small bowel and its mesentery back toward the pancreas, the surgeon can reapproximate the ends of the vein without tension.

When multiple vascular and visceral injuries are present in the upper abdomen and the superior mesenteric vein has been severely injured, ligation can be performed. After ligation of the superior mesenteric (or portal) vein, there is immediate swelling and discoloration of the midgut. Patients who have had rapid control of hemorrhage and stabilize quickly will usually not develop areas of necrosis of the small bowel in the postoperative period. In patients with a prolonged period of perioperative shock and need for pressor agents in the postoperative period, however, there is a risk of significant necrosis of segments of small bowel.

On occasion, there is prolonged splanchnic hypervolemia after ligation of the superior mesenteric vein. The massive distension of the midgut prevents closure of the midline abdominal incision, and the continuous leak of transudative fluid mandates vigorous daily fluid resuscitation. There are two reoperative options to decompress the splanchnic venous circulation in such patients. One is splenic vein turndown to the stump of the ligated superior mesenteric vein, whereas the other is use of a segment of the femoral vein as a conduit to the splenic vein or portal vein.[99,100]

In one 1988 review of injuries to the superior mesenteric vein, survival was 85% among 33 patients treated with ligation versus 64% in 77 patients who underwent repair.[101] Stone et al[102] have emphasized the need for vigorous postoperative fluid resuscitation in these patients as splanchnic hypervolemia leads to peripheral hypovolemia for at least 3 days after ligation of the superior mesenteric vein. The survival rate of patients with injuries to the superior mesenteric vein in four series in the distant past was 72.1% (Table 38-4).[80,95,102,103] Three more recent reviews in 2001 showed a mean survival rate of 58.3%.[12-14] One review in 2009 noted a survival rate of 55% in 11 patients.[104]

Asensio et al[105] reported the largest series in the literature, consisting of 51 injuries to the superior mesenteric vein. The mean Injury Severity Score was 25 ± 12, the mechanism of injury was penetrating for 38 patients (76%) and blunt for 13 patients (24%), and 4 patients (8%) required emergency department thoracotomy. Surgical management consisted of ligation in 30 patients (59%) and primary repair in 16 patients (31%), and 5 patients (10%) exsanguinated before repair. The overall survival rate excluding patients undergoing emergency department thoracotomy was 51%. The survival rate excluding patients who sustained greater than three to four

TABLE 38-4: Survival with Injuries to the Superior Mesenteric Vein

Reference	Year	No. of patients	No. of survivors	Survival (%)
Four series[80,95,102,103]	1978–1983	104	75	72.1
Asensio et al[12]	2001	19 (isolated injury)	9	47.4
		14 (with other vein)	5	35.7
Davis et al[13]	2001	21	15	71.4
Tyburski et al[14]	2001	32	18	56.3
Asensio et al[105]	2007	51	26	51
Fraga et al[104]	2009	11	6	55

associated injured vessels was 65%. The survival rates were 55% for patients with combined superior mesenteric vein and artery injuries, 40% for patients with injuries to the superior mesenteric vein and portal vein, and 55% for patients with isolated injuries to the superior mesenteric vein. When mortality was stratified to AAST-OIS grade, survival was 44% for grade III and 42% for grade IV. Survival rates stratified to method of management were 60% for primary repair versus 40% for ligation.

MANAGEMENT OF INJURIES IN ZONE 1: INFRAMESOCOLIC REGION

Exposure and Vascular Control

The second major area of hematoma or hemorrhage in the midline is the inframesocolic area. In this location, abdominal vascular injuries include those to the *infrarenal abdominal aorta* or *inferior vena cava.* Exposure of an inframesocolic injury to the aorta is obtained by duplicating the maneuvers used to gain proximal aortic control during the elective resection of an abdominal aortic aneurysm. The transverse mesocolon is displaced up toward the patient's head, the small bowel is eviscerated toward the right (surgeon's) side of the table, and the midline inframesocolic retroperitoneum is opened until the left renal vein is exposed. A proximal aortic clamp such as a DeBakey or Crafoord-DeBakey should then be placed immediately inferior to the left renal vein. When a large retroperitoneal hematoma is present and proximal inframesocolic control is difficult to obtain, it should always be remembered that the injury in the aorta is under the highest point of the hematoma ("Mount Everest phenomenon"). Therefore, rapid digital dissection of the hematoma will generally bring the surgeon directly to the area of injury. Exposure to allow for application of the distal vascular clamp is obtained by dividing the midline retroperitoneum down to the aortic bifurcation, carefully avoiding the left-sided origin of the inferior mesenteric artery. This vessel, however, may be sacrificed whenever necessary for exposure.

If the aorta is intact and an inframesocolic hematoma appears to be more extensive on the right side of the abdomen than on the left or if there is active hemorrhage coming through the base of the mesentery of the ascending colon or hepatic flexure of the colon, injury to the inferior vena cava caudad to the liver should be strongly suspected. Although it is possible to visualize the vena cava through the midline retroperitoneal incision previously described, most trauma surgeons are more comfortable in visualizing the inferior vena cava by performing an extensive right-sided medial visceral rotation. This consists of mobilizing the right half of the colon and C-loop of the duodenum via an extensive Kocher maneuver and leaving the right kidney in situ (Fig. 38-5). This permits the entire vena caval system from the confluence of the iliac veins to the suprarenal vena cava inferior to the liver to be visualized. This maneuver must be carefully performed to avoid an iatrogenic injury to the right gonadal vein. It is often difficult to define precisely where a hole is in a large vein of the abdomen, such as the inferior vena cava, until much of the loose retroperitoneal fatty tissue is stripped away from the wall of the vessel. Once this is done, the site of hemorrhage can be localized.

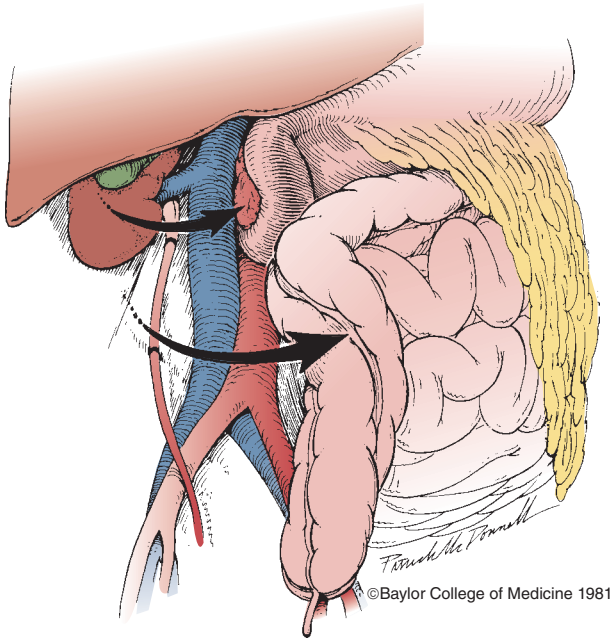

FIGURE 38-5 Medial mobilization of right-sided intra-abdominal viscera except the kidney allows for visualization of the entire infrahepatic inferior vena cava. (Reproduced with permission from Baylor College of Medicine.)

If active hemorrhage appears to be coming from the anterior surface of the inferior vena cava, a Satinsky-type vascular clamp should be applied directly to the perforation as it is elevated by a pair of vascular forceps or Allis clamps. When the inferior vena cava has been extensively lacerated and partial occlusion cannot be performed, it is often helpful to compress the proximal and distal vena cava around the partial transection or extensive laceration using gauze sponges placed in straight sponge sticks. Because of back-bleeding from lumbar veins, it may be necessary to use large DeBakey aortic clamps and completely occlude the vena cava above and below some injuries. This maneuver carries a risk in the already hypotensive patient since venous return to the right side of the heart is significantly impaired. For this reason, one should consider simultaneous clamping of the infrarenal abdominal aorta.

The two areas in which proximal and distal control of the inferior vena cava below the liver is especially difficult to obtain are at the confluence of the common iliac veins and at the caval junction with the renal veins. Although sponge-stick compression of the common iliac veins and the vena cava superiorly may control hemorrhage at the confluence, visualization of perforating wounds in this area is compromised by the overlying aortic bifurcation. In the case of difficult exposure, one technique is to divide and ligate the right internal iliac artery, which may allow for lateral and cephalad

retraction of the right common iliac artery to expose the venous injury. An alternate and interesting approach, but one that is rarely necessary, is the temporary division of the overlying right common iliac artery itself, with mobilization of the aortic bifurcation to the left.[106] This technique provides wide exposure of the confluence of the common iliac veins and the proximal vena cava, and the venous injury can be repaired or ligated in the usual fashion. The right common iliac artery is then reconstituted by an end-to-end anastomosis. Another option is to dissect the common iliac veins with Kittner dissectors and separate them or the external and internal iliac veins with Cushing vein retractors to locate the area of injury.

When the perforation occurs at the junction of the renal veins and the inferior vena cava, it should be directly compressed either digitally or with sponge sticks. An assistant then clamps the infrarenal vena cava and the suprarenal infrahepatic vena cava and loops both renal veins individually with vascular tapes or vessel loops to allow for the direct application of angled vascular clamps. When time does not allow for this dissection, medial mobilization of the right kidney may allow for the application of a partial occlusion clamp across the inferior vena cava at its junction with the right renal vein. This medial mobilization maneuver is useful for exposing posterior perforations in the suprarenal infrahepatic vena cava also.[107] Should this latter maneuver be performed, care must be taken to divide and ligate but not avulse the first lumbar vein on the right, as it frequently enters the junction of the right renal vein and inferior vena cava. One other useful technique for controlling hemorrhage from the inferior vena cava in all locations is to use a Foley balloon catheter for tamponade.[108,109] Either a 5-mL or a 30-mL balloon catheter can be inserted into a caval laceration, the balloon inflated in the lumen, and traction applied to the catheter. Once the bleeding is controlled, either a purse-string suture is inserted or a transverse venorrhaphy is performed, taking care not to rupture the underlying balloon with the needle. The balloon catheter is then deflated and removed just before completion of the suture line.

Over the past 15 years, balloon catheters have been inserted percutaneously in the preoperative or intraoperative period to control hemorrhage from the inferior vena cava and/or iliac veins as well.[110,111]

Infrarenal Aorta

As with injuries to the suprarenal aorta, penetrating or blunt injuries in the infrarenal abdominal aorta are repaired primarily with 3-0 or 4-0 polypropylene sutures or by patch aortoplasty, end-to-end anastomosis, or insertion of a woven Dacron graft, albumin-coated Dacron graft, or a PTFE graft, none of which require preclotting. Because of the small size of the aorta in young trauma patients, it is unusual to be able to place a tube graft larger than 12, 14, or 16 mm in diameter if one is required, as previously noted. The principles of completing the suture lines, sequence of clamp release, and flushing are exactly the same as for aortic repairs in the suprarenal area. Since the retroperitoneal tissue is often thin in young patients, it may be worthwhile to cover an extensive aortic repair or the suture line of a prosthesis with mobilized omentum before closure of the retroperitoneum.[112] After mobilization of the gastrocolic omentum off the transverse colon, it can be placed into the lesser sac superiorly and then brought down through an opening in the transverse mesocolon over the repair or graft in the infrarenal aorta. An alternate approach is to mobilize the gastrocolic omentum off the left side of the transverse colon and then bring the mobilized pedicle around the ligament of Treitz to once again cover the aortic repair or graft. This vascularized pedicle of omentum should prevent a postoperative aortoduodenal fistula.

Although the vast majority of injuries to the infrarenal aorta are penetrating in nature, a small number occur after blunt trauma. The largest review to date is the Western Trauma Association multicenter study by Shalhub et al[24] in 2014. There were 113 patients with a blunt abdominal aortic injury, 60% from motor vehicle collisions, and 44% of patients had associated spinal fractures. Injuries noted included intimal tears (18%), large intimal flaps (34%), pseudoaneurysms (16%), and free aortic rupture (32%). While 89% of intimal tears were managed nonoperatively, more serious lesions underwent open repair (43%) or endovascular repair (15%). Mortality was 39% overall, with 68% of deaths in the first 24 hours due to hemorrhage and shock.

The survival rate of patients with injuries to the infrarenal abdominal aorta in six series in the distant past was 46.2% (Table 38-2).[77,79-82,113] As previously noted in the discussion of recent decreases in survival figures for injuries to the suprarenal abdominal aorta, the same has been true for injuries to the infrarenal abdominal aorta. In the one series published in 2001 in which injuries to the suprarenal and infrarenal abdominal aorta were separated, the survival rate in the infrarenal group was only 34.2%.[14] Another review in 2007 in which 11 of 20 patients had shock on admission documented a survival of 27.3%.[84]

ENDOVASCULAR

Endovascular stent grafts have been successfully used to treat intimal flaps, wall injuries, traumatic false aneurysms, and contained transections of the infrarenal abdominal aorta since 1997.[114-117]

Infrahepatic Inferior Vena Cava

Anterior perforations of the inferior vena cava are best repaired in a transverse fashion using a continuous suture of 4-0 or 5-0 polypropylene. If vascular control is satisfactory and a posterior perforation can be visualized adequately by extending the anterior perforation, the posterior perforation can be repaired from inside the vena cava, with the first suture knot outside the lumen. When a significant longitudinal perforation is present, especially when adjacent perforations have been joined, the repaired vena cava will often take on the appearance of an hourglass. This narrowing may lead to slow postoperative occlusion of the inferior vena cava.

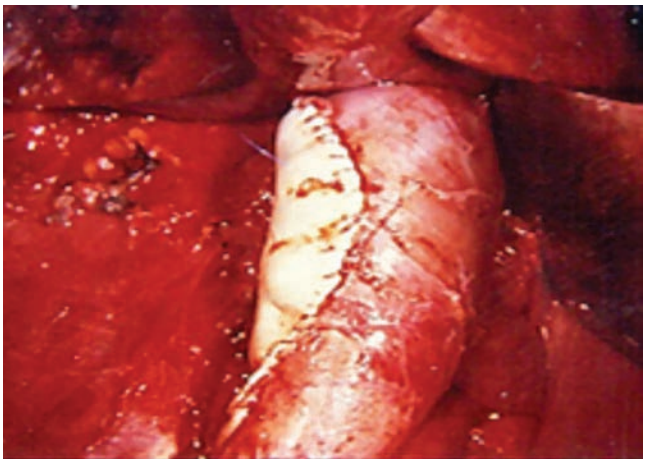

FIGURE 38-6 Polytetrafluoroethylene patch venoplasty of infrarenal inferior vena cava after mid-abdominal gunshot wound.

In the unstable patient who has developed a coagulopathy, no further attempt should be made to modify the repair. In the stable patient, there may be some justification for applying a large venous patch taken from either the resected inferior mesenteric vein or an ovarian vein, or applying a thin-walled PTFE patch (Fig. 38-6).

In a patient who is exsanguinating and in whom extensive repair of the infrarenal inferior vena cava appears to be necessary, ligation of this vessel is usually well tolerated as long as certain precautions are taken (Fig. 38-7). The first of these is to measure the pressures in the compartments of the legs and thighs at the first operation and to perform bilateral below-knee four-compartment fasciotomies if the pressure is 30 to 35 mm Hg, depending on the patient's hemodynamic status. Bilateral thigh fasciotomies may be necessary as well, within the first 48 hours after ligation. The second is to maintain circulating volume in the postoperative period through infusion of the appropriate fluids. The third is to apply

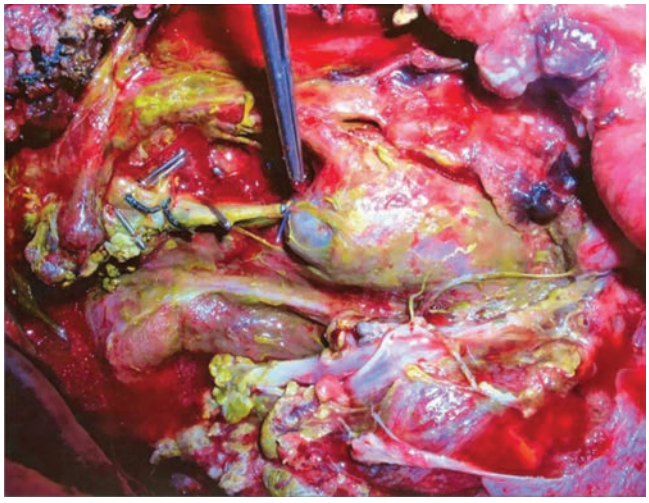

FIGURE 38-7 One day after ligation of the infrarenal inferior vena cava in patient with paraumbilical gunshot wound. Note extensive fat necrosis related to associated injury to the head of the pancreas.

elastic compression wraps to both lower extremities and keep them continuously elevated for approximately 5 to 7 days after operation. Patients should wear the wraps when they start to ambulate, as well. If there is some residual edema even with the wraps in place at the time of hospital discharge, the patient should be fitted with full-length, custom-made support hose.

A 2005 review from Groote Schuur Hospital (University of Cape Town, South Africa) emphasizes the significant increase in the use of ligation for injuries to the infrarenal inferior vena cava over the past two decades.[118] In 41 patients with such injuries, ligation was performed in 29 (70.7%).

In a 2010 review of 100 injuries to the inferior vena cava treated at Grady Memorial Hospital (Emory University) in Atlanta, 25 underwent ligation, including 22 of 54 (40.7%) with injuries to the infrarenal vena cava.[119] Survival to hospital discharge was 41%; 1-year follow-up was available in seven of nine survivors after ligation, and no patient had more than trace lower extremity edema. While the majority of patients, therefore, seem to have no or minimal long-term edema, there have been occasional reports of severe edema in the postoperative period that has required later interposition grafting.[120,121]

Ligation of the suprarenal inferior vena cava is performed only when the patient has an extensive injury at this location and appears to have terminal shock at operation.[122,123] In the previously mentioned series from Grady Memorial Hospital, three patients underwent ligation of the suprarenal inferior vena cava with only one long-term survivor. This patient required dialysis over his hospital course but gradually experienced a return of his renal function and was discharged without renal insufficiency. One-year follow-up in this patient revealed minimal lower extremity edema and normal renal function.[119] In order to avoid the risk of acute renal failure and massive edema of the lower half of the body that would ordinarily be associated with ligation at this level, several innovative approaches have been used. These include suprarenal insertion of a femoral vein graft, a saphenous vein composite interposition graft, insertion of a Dacron or ringed PTFE interposition graft, insertion of a cavo–right atrial Dacron or PTFE bypass graft, or a hybrid approach with transcaval insertion of an endostent graft.[120,121,124,125] Although long-term data are lacking for both Dacron and PTFE interposition or bypass grafts in the major veins of the abdomen, the use of an externally supported PTFE graft in combination with chronic anticoagulation would presumably offer the best long-term patency.

Survival rates for patients with injuries to the inferior vena cava obviously depend on the location of injury (Table 38-5). If one eliminates injuries to the suprahepatic and retrohepatic vena cava (to be discussed) from seven series published in the distant past, the average survival for 515 patients with injuries to the infrahepatic vena cava was 72.2%.[80,82,107,126-129] Further eliminating juxtarenal injuries, the average survival for 318 patients with true infrarenal caval injuries was 76.1%.[80,82,107,126-129] Two more recent series (2001 and 2010), in which injuries are stratified by location, show lower overall survival rates (46.1% and 59.2%) for injuries to the infrarenal

TABLE 38-5: Survival with Injuries to the Inferior Vena Cava

	Seven series, 1978–1994[80,82,107,126-129]	Tyburski et al[14]	Sullivan et al[119]
Infrahepatic inferior vena cava	72.2% (372/515)	40.3% (31/177)	57.3% (43/75)
Infrarenal inferior vena cava	76.1% (242/318)	46.1% (30/65)	59.2% (32/54)

inferior vena cava.[14,119] Finally, short-term patency of repair of the inferior vena cava was studied by Porter et al[130] in 1997. In 28 patients with prior lateral venorrhaphy of the inferior vena cava, patency of the cava was documented by sonography, CT scan, or both in 24 patients (86%).

ENDOVASCULAR

In the rare patient with a contained hematoma around a perforation or rupture of the inferior vena cava noted on a screening or preoperative CT of the abdomen, an endovascular stent graft can be inserted as definitive treatment.[131,132]

MANAGEMENT OF INJURIES IN ZONE 2

Exposure and Vascular Control

If a hematoma or hemorrhage is present in the upper lateral retroperitoneum, injury to the *renal artery, renal vein*, or *both*, as well as injury to the *kidney*, should be suspected. Most patients with penetrating trauma to the abdomen are explored prior to extensive radiologic workup; however, in selected patients who are hemodynamically stable after sustaining penetrating wounds to the flank, CT scanning has been used to document an isolated renal injury and operation has been avoided.[133] Conversely, patients found to have a perirenal hematoma at the time of exploration for a penetrating abdominal wound should have unroofing of the hematoma and exploration of the wound track. If the hematoma is not rapidly expanding and there is no free intra-abdominal bleeding, some surgeons will loop the ipsilateral renal artery with vessel loops or a vascular tape in the midline at the base of the mesocolon.[134] The left renal vein can be looped with vessel loops or a vascular tape in the same location; however, vascular control of the proximal right renal vein will have to wait for mobilization of the C-loop of the duodenum and unroofing of the inferior vena cava at its junction with the renal veins. It should be noted that obtaining proximal vascular control prior to exploration of a perirenal hematoma is controversial. Indeed, in one study, preliminary vascular control of the renal hilum had no impact on nephrectomy rate, transfusion requirements, or blood loss.[135]

Conversely, if there is active bleeding from the kidney through Gerota's fascia or from the retroperitoneum overlying the renal vessels, central renovascular control is not obtained. The surgeon should simply open the retroperitoneum lateral to the injured kidney and manually elevate the kidney directly into the wound. A large vascular clamp can be applied proximal to the hilum either at the midline on the left or just lateral to the inferior vena cava on the right to control any further bleeding.

Patients who present after blunt trauma may have either a renovascular or renal parenchymal injury, also. Patients in the former group, however, generally present with renovascular occlusion, which will be discussed later. In patients who have suffered blunt abdominal trauma and have undergone a duplex ultrasound of the renal artery or a CT of the abdomen that has demonstrated flow to the kidney and/or a low AAST-OIS grade of injury, there is no justification for exploring the perirenal hematoma should an emergency laparotomy be indicated for other reasons.

Renovascular Injuries: Renal Artery

Renovascular injuries are difficult to manage, especially when the renal artery is involved. It is a small vessel that is deeply embedded in the retroperitoneum. Occasionally, small perforations of the artery from penetrating wounds can be repaired by lateral arteriorrhaphy or resection with an end-to-end anastomosis. Interposition grafting using a saphenous vein graft for extensive injuries is indicated only when the injury leads to a solitary kidney. Alternatively, borrowed or substitute arteries, such as the splenic artery to replace the left renal artery and the hepatic artery to replace the right renal artery, have been used rarely but are not often indicated in hypotensive trauma patients with significant renovascular injuries from penetrating wounds. In these patients and in those with multiple intra-abdominal injuries or a long preoperative period of ischemia, nephrectomy may be a better choice, as long as intraoperative palpation has confirmed a normal contralateral kidney. The survival rate of patients with injuries to the renal arteries from penetrating trauma in two studies in the distant past was approximately 87%, with renal salvage in only 30% to 40%.[134,136] In the three large series from 2001, the survival rate was 65.1%.[12-14]

Intimal tears in the renal arteries may result from deceleration in motor vehicle crashes, automobile–pedestrian accidents, and falls from heights. These can lead to secondary thrombosis of the vessel and complaints of upper abdominal and flank pain in some patients, as previously noted. One older literature review noted that only 30% of patients with intimal tears in the renal arteries had gross hematuria, 43% had microscopic hematuria, and 27% had no blood in the urine.[137] Hence, the diagnosis may be missed because a duplex ultrasound or CT scan may not be performed expeditiously in a stable patient with a normal abdominal examination.

If either duplex ultrasound or a CT scan documents occlusion of a renal artery, the surgeon must decide on the need

for operation, endovascular intervention, or observation. Intuitively, the time interval from the episode of trauma to restoration of arterial flow would appear to be the most critical factor in saving the affected kidney. In one older study, there was an 80% chance of restoring some renal function at 12 hours, but this significantly decreased to 57% at 18 hours after the onset of occlusion.[137] A Western Trauma Association multicenter review in 2000[138] and a literature review in 2017,[19] however, did not demonstrate a clear relationship between time of occlusion and eventual function of the kidney after vascular repair.

A laparotomy to repair an isolated blunt stenosis or thrombosis of a renal artery is no longer performed in the modern era. Even in patients undergoing a needed laparotomy for other intra-abdominal injuries, a postoperative endovascular approach is used.[19]

It is of interest that some older case reports in the literature have documented either spontaneous recovery or the late successful revascularization of one or both kidneys after presumed blunt thrombosis of the renal artery.[139] The authors of the report suggest that attempts at late revascularization may be occasionally rewarding and advise that early nephrectomy is unnecessary because of the low incidence of chronic hypertension in cases of renal artery thrombosis.

In patients who are critically ill after multisystem trauma, isolated thrombosis of one renal artery may be observed if the contralateral artery and kidney are normal on imaging. Obviously, patients with bilateral renal artery injuries or those with injury to a solitary renal artery should be strongly considered for endovascular revascularization. In addition, prolonged follow-up should be arranged for all patients to see if hypertension develops.[19,34]

In the 2006 review of 517 patients with blunt renal artery injuries in the National Trauma Data Bank, only 13 patients had insertion of an endovascular stent.[140] The kidney was not explored in 376 patients (73%), 95 patients (18%) had nephrectomy, and only 45 patients (9%) had surgical revascularization. Patients who had surgical revascularization had longer stays in the intensive care unit and hospital on multiple regression analysis. The recommendation by the authors was that "nonoperative management should be considered as an acceptable therapeutic option."[140]

ENDOVASCULAR

As noted earlier, endovascular stents are commonly placed to cover intimal defects or restore arterial flow after blunt injury of the renal artery. The endovascular approach is one of the oldest and most commonly performed of such procedures in abdominal vessel trauma.[141,142]

Renovascular Injuries: Renal Vein

A patient with a penetrating wound to the renal vein may be quite stable as a result of the previously described retroperitoneal tamponade. Either digital compression or the direct application of a Satinsky or Henly vascular clamp can be used to control bleeding after exposure of the injured vein. Lateral venorrhaphy remains the preferred technique of repair. If ligation of the right renal vein is necessary to control hemorrhage, nephrectomy should be performed either at the initial operation or at the reoperation if damage control has been necessary. The medial left renal vein, however, can be ligated as long as the left adrenal and gonadal veins are intact. In one review of 36 patients followed more than 1 year after elective ligation of the left renal vein during complex surgery on the abdominal aorta, only two developed elevation of creatinine levels over 2 years later.[143] The survival rate for patients with penetrating injuries to the renal veins has ranged from 42% to 88% in the older literature, with the difference presumably due to the magnitude and number of associated visceral and vascular injuries.[135,144] In the three 2001 reviews, survival ranged from 44.2% to 70%, with a mean of 60.4%.[12-14]

Injuries to the renal parenchyma are discussed in Chapter 40.

MANAGEMENT OF INJURIES IN ZONE 3

Exposure and Vascular Control

The fourth major area of hematoma or hemorrhage is the pelvic retroperitoneum. In this location, the *iliac artery, iliac vein*, or *both* may be injured. Furthermore, the vascular injury may be in combination with an injury to the *ureter.* The majority of injuries reported in major series are the result of penetrating trauma. It is of interest, however, that major blunt abdominal trauma or pelvic fractures, particularly of the open type, have become a more frequent cause of occlusion or laceration of the iliac arteries in the past 30 years than previously noted.[26,27] In fact, a 2009 review from the National Trauma Data Bank noted a 3.5% rate of blunt iliac artery injury in 6377 patients with Abbreviated Injury Scale 3 or 4 pelvic fracture.[25] The presence of a blunt iliac arterial injury in combination with a pelvic fracture was associated with a significantly higher hospital mortality (40% vs 15%) and a 7.7% amputation rate.[25]

If a hematoma or hemorrhage is present after penetrating trauma, digital compression, compression with a laparotomy pad, or simply grabbing the bleeding vessel with a hand after opening the pelvic retroperitoneum should be performed until proximal and distal vascular control is attained. The proximal common iliac arteries are exposed by eviscerating the small bowel to the right and dividing the midline retroperitoneum over the aortic bifurcation. In young trauma patients, there is usually *no adherence* between the common iliac artery and vein in this location, and vessel loops or vascular tapes can be passed rapidly around the proximal arteries. Distal vascular control is obtained at the point at which the external iliac artery comes out of the pelvis proximal to the inguinal ligament. The artery is readily palpable under the retroperitoneum and can be rapidly elevated into the field of view with a vessel loop or a vascular tape. A Cushing vein retractor is useful in pulling the adjacent external iliac vein away during this maneuver. The major problem in this area is continued back-bleeding from the internal iliac artery. This artery can be exposed by further opening the retroperitoneum on the side of the pelvis, elevating the vessel loops or vascular tapes

on the proximal common iliac and distal external iliac arteries, and looking for the large branch of the iliac artery that descends into the pelvis.

When bilateral iliac vascular injuries are present, one of the former coauthors of this chapter (Jon M. Burch, MD) has used the technique of total pelvic vascular isolation. This includes proximal cross-clamping of the abdominal aorta and inferior vena cava just above their bifurcations and distal cross-clamping of both the external iliac artery and vein with one clamp on each side of the pelvis. Back-bleeding from the internal iliac vessels is minimal with this approach.

Injuries to the iliac veins are exposed through a technique similar to that described for injuries to the iliac arteries. It is not usually necessary to pass vessel loops or vascular tapes around these vessels, however, because they are readily compressible with either sponge sticks or fingers. As previously noted, the partially covered location of the right common iliac vein has led to the suggested temporary transection of the right common iliac artery in order to improve exposure at this location.[106] Similarly, transection and ligation of the internal iliac artery on the side of the pelvis will allow for improved exposure of an injured ipsilateral internal iliac vein.[145]

Common, External, and Internal Iliac Arteries

Injuries to the common or external iliac artery should be repaired or temporarily shunted. Ligation of either vessel in the hypotensive patient will lead to progressive ischemia of the lower extremity and the need for a high above-knee amputation or a hip disarticulation in the later postoperative course. In fact, in World War II, ligation of these vessels led to amputation rates of approximately 50%.[5] Furthermore, in a large review by Burch et al[146] in 1990, mortality associated with ligation was 90%. In patients with severe shock, insertion of a temporary intraluminal shunt is a better choice for damage control. Ball and Feliciano[147] compared the results of ligation (1 common and 14 external iliac arteries) versus temporary intraluminal shunts (2 common and 5 external iliac arteries). Patients treated with shunts had fewer amputations (0% vs 47%), less distal fasciotomy procedures (43% vs 93%), and lower mortality rates (43% vs 72%). In contrast, an injured internal iliac artery can be ligated with impunity even with injuries that occur bilaterally.

Options in the management of more stable patients with injuries to the common or external iliac artery include the following: lateral arteriorrhaphy; completion of a partial transection and end-to-end anastomosis; resection of the injured area and insertion of a saphenous vein or PTFE graft[148,149]; mobilization of the ipsilateral internal iliac artery to serve as a replacement for the external iliac artery; or transposition of one iliac artery to the side of the contralateral iliac artery for wounds at the bifurcation.[150]

Extensive injuries to the common or external iliac artery in the presence of *significant* enteric or colonic contamination in the pelvis remain a serious problem for the trauma surgeon. Both end-to-end repairs and vascular grafts in this location have suffered postoperative formation of postrepair false aneurysms and even blowouts secondary to pelvic infection from the original intestinal contamination. Therefore, the authors have occasionally avoided an end-to-end anastomosis or the insertion of a saphenous vein or PTFE graft in either the common or external iliac artery in such a situation. Rather, the artery is divided just proximal to the injury, closed with a double-running row of 4-0 or 5-0 polypropylene sutures, and covered with noninjured retroperitoneum or a vascularized pedicle of omentum. If the patient's lower extremity on the side of the ligation appears to be in jeopardy at the completion of the abdominal operation in the reasonably stable patient, an extra-anatomic femorofemoral crossover graft should be performed to return arterial inflow to the extremity.[72] If the surgeon chooses not to perform a femorofemoral crossover graft until the patient's condition has been stabilized in the surgical intensive care unit, an ipsilateral four-compartment below-knee fasciotomy should be performed, since ischemic edema below the knee will often lead to a compartment syndrome. The thigh compartments should be monitored for increased ischemic edema as well.

The survival rate among patients with injuries to the iliac arteries will vary with the number of associated injuries to the iliac vein, aorta, and vena cava, but was approximately 61% in 189 patients reviewed in four large series published in the distant past (Table 38-6).[95,146,151,152] When patients with other vascular injuries, especially to the iliac vein, were eliminated, the survival rate among 57 patients in three older series was 81%.[146,151,152] If the injury is large and free bleeding from the iliac artery into the peritoneal cavity has occurred during the preoperative period, the survival rate in one older series was only 45%.[151]

In one of the largest series in the literature, published in 2003, Asensio et al[153] reported on the predictors of outcome for 148 patients sustaining a total of 185 iliac vessel injuries. In this series, the authors reported a 95% incidence of penetrating injuries with a mean estimated blood loss of 6246 ± 6174 mL. Of the 185 injured vessels, 71 (99%) of 72 iliac arteries were repaired, 101 (89%) of the 113 iliac veins were ligated, and overall survival was 51% (76/148). Mortality was 82% in patients with exsanguination (49/72). Survival by vessel included the following: iliac artery, 57% (20/35); iliac vein, 55% (42/76); and iliac artery and vein combined, 38% (14/37). Significant predictors of outcome were thoracotomy in the emergency department, associated injury to the abdominal aorta or inferior vena cava, combined injuries to the iliac artery and vein, intraoperative arrhythmia, and intraoperative coagulopathy. On logistic regression, independent risk factors for survival were absence of thoracotomy in the emergency department, surgical management, and arrhythmias. When mortality was stratified according to the AAST abdominal vascular injury grade, grade III was 35% (33/95), grade IV was 71% (23/34), and grade V was 79% (15/19).

The survival rates in two of the 2001 reviews for patients with injuries to the common iliac artery (other vascular injuries not specified) ranged from 44.7% to 55.5%, with a mean of 46.8% (Table 38-6).[13,14] In the same reviews, the survival rate with injuries to the external iliac artery was a mean of 64.1%

TABLE 38-6: Historic Survival with Injuries to the Iliac Artery and Vein

		Iliac artery			Iliac vein		
Reference	Year	No. of patients	No. of survivors	Survival (%)	No. of patients	No. of survivors	Survival (%)
Millikan et al[151]	1981	19 (6)[a]	9 (5)[a]	47.4 (83.3)[a]	16 (8)[b]	11 (8)[b]	68.8 (100.0)[b]
Ryan et al[152]	1982	66 (17)[a]	41 (15)[a]	62.1 (88.2)[a]	97 (48)[b]	71 (45)[b]	73.2 (93.8)[b]
Sirinek et al[95]	1983	21	15	71.4	28	23	82.1
Burch et al[146]	1990	130 (34)[a]	80 (26)[a]	61.5 (76.5)[a]	214 (81)[b]	153 (70)[b]	71.5 (86.4)[b]
Wilson et al[155]	1990	—	—	—	49	24	48.9
Davis et al[13]	2001	55	35	63.6	76	58	76.3
Tyburski et al[14]	2001	70	37	52.9	73	40	54.8
Asensio et al[12]	2001	—	—	—	37 (22)[b]	23 (18)[b]	62.2 (81.8)[b]
Asensio et al[153]	2003	35	20	57	76	42	55
Overall		396 (57)[a]	237 (46)[a]	59.8 (80.7)[a]	666 (159)[b]	445 (141)[b]	66.8 (88.7)[b]

[a]Isolated injury to iliac artery.
[b]Isolated injury to iliac vein.

(Table 38-6).[13,14] In the comprehensive review of data from the National Trauma Data Bank in 2013 by Lauerman et al,[26] survival rates were as follows: penetrating iliac artery, 60.8%; and penetrating iliac artery and vein, 48.2% (Table 38-7).[26]

Blunt trauma to the iliac arteries is still less common as they are protected by the bony pelvis and lie deep in the retroperitoneum. Partial transections, avulsions, and intimal injuries with secondary thrombosis have all been reported in association with pelvic fractures, particularly in recent years, as previously noted. Despite the low amputation rates described previously,[25,26] the amputation rate in 24 patients with blunt injuries to the common or external iliac artery (pelvic fractures in 89% of all iliac artery injuries) was 50% in a 2014 review from the Shock Trauma Center/University of Maryland, Baltimore.[27]

ENDOVASCULAR

Endovascular stents and stent grafts have been widely used in patients with complications after repair of penetrating wounds to the common and external iliac arteries and to restore flow in patients with blunt thrombosis.

Common, External, and Internal Iliac Veins

Injuries to the common or external iliac vein are treated either with lateral repair using 4-0 or 5-0 polypropylene suture or with ligation. Ligation in young patients has been well tolerated in the authors' experience and that of others if the same precautions used after ligation of the inferior vena cava are applied.[154] When significant narrowing of the common or external iliac vein results from a lateral repair, postoperative anticoagulation is appropriate to lessen the risk of thrombosis and/or pulmonary embolism.

The survival rate of patients with injuries to the iliac veins is variable but was approximately 70% in 404 patients reviewed in five large series published in the distant past (Table 38-6).[95,146,151,152,155] When patients with other vascular injuries, especially to the iliac artery, were eliminated, the survival rate among 137 patients in three series was 95%.[146,151,152] The survival rate in the three 2001 series in patients with injuries to the iliac vein (not otherwise specified or common/external/internal combined) was a mean of 65.1%.[12-14]

TABLE 38-7: Survival with Injuries to the Iliac Artery and Vein—National Trauma Data Bank

Reference	Year	Penetrating artery	Penetrating vein	Penetrating combined
Lauerman et al[26]	2013	60.8% (124/204)	74.5% (190/255)	48.% (76/157)
Reference	**Year**	**Penetrating/blunt artery**	**Penetrating/blunt vein**	**Penetrating/blunt combined**
Magee et al[156]	2018	80.7%% (1562/1936)	83.5% (740/886)	51.3% (286/557)
Reference	**Year**	**Iliac vein repair**	**Iliac vein ligation**	
Magee et al[156]	2018	91.2% (206/226)	81.3% (156/192)	

In more recent reviews from the National Trauma Data Bank in 2013 and 2018, the survival rates after injury to the iliac vein (but not the artery) were 74.5% and 83.5%, respectively, whereas the survival rates after combined arterial and venous injuries were 48.4% and 51.3%[26,156] (Table 38-7). In the latter review, the survival rate was 91.2% after venous repair versus 81.3% after ligation.[156]

Pelvic Fractures and Injury to Pelvic Vessels

The management of pelvic fractures is comprehensively reviewed in Chapter 39. Trends in the management of hemorrhage from the iliac vessels and branches over the past 15 years have included the following: (1) REBOA positioned at zone 3 (infrarenal aorta)[157,158]; (2) preperitoneal packing[159]; (3) endovascular stent graft for major arterial injury[157]; (4) angioembolization with access through either common femoral or radial arteries[157]; and (5) return of operative ligation of bilateral internal iliac arteries.[160]

Transpelvic Gunshot Wounds and Presacral Bleeding

A number of techniques, including some from colorectal surgeons, are available to control presacral bleeding after transpelvic gunshot wounds. These include the following: (1) sterilized metallic thumbtacks[161,162]; (2) endoscopic mesh stapler[163]; (3) electrocautery passed through muscle fragments[164]; (4) tamponade with Sengstaken-Blakemore balloon[165]; (5) tamponade with breast implant sizer[166]; (6) free rectus muscle piece for tamponade[167]; and (7) external fixator to hold direct compression.[168]

MANAGEMENT OF INJURIES IN THE PORTA HEPATIS AND TO THE RETROHEPATIC VENA CAVA

Exposure and Vascular Control

If a hematoma or hemorrhage is present in the area of the portal triad in the right upper quadrant, there may be injury to the *portal vein, hepatic artery*, or *both.* Furthermore, the vascular injury may be in combination with an injury to the *common bile duct.* When a hematoma is present, the proximal hepatoduodenal ligament should be encircled with a vessel loop or vascular tape or a noncrushing vascular clamp should be applied (the Pringle maneuver) before the hematoma is entered. If hemorrhage is occurring, finger compression of the bleeding vessels will suffice until the vascular clamp is in place. The Pringle maneuver clamps the distal common bile duct as well as the bleeding vessels but led to only one stricture of the common bile duct in one older series of hepatic injuries from the Ben Taub Hospital in Houston, Texas, in 1986.[169] Because of the short length of the porta in many patients, it may be difficult to place a distal vascular clamp right at the edge of the liver. In such patients, manual compression with forceps may allow partial distal vascular control until the area of injury can be isolated. Because of the proximity of the common bile duct, no sutures should be placed into the porta until the vascular injury is precisely defined.

Injuries to the portal vein in the hepatoduodenal ligament are isolated in much the same fashion as injuries to the hepatic artery. The posterior position of the vein, however, makes the exposure of these injuries more difficult. Mobilization of the common bile duct to the left and of the cystic duct superiorly, coupled with an extensive Kocher maneuver, will usually allow for excellent visualization of any suprapancreatic injury after proximal (and, if possible, distal) vascular control has been obtained. As with proximal wounds to the superior mesenteric artery or vein, division of the neck of the pancreas is necessary on rare occasions to visualize perforations in the retropancreatic portion of the portal vein. With the assistant compressing the superior mesenteric vein below and a vascular clamp applied to the hepatoduodenal ligament above, the surgeon should open both ends of the retropancreatic tunnel over the anterior wall of the portal vein by gently spreading a clamp or scissors. This maneuver may be prevented above by the position of the gastroduodenal artery, which should then be divided and ligated. When the tips of the surgeon's index fingers touch under the neck of the pancreas, two straight noncrushing intestinal (Glassman or Dennis) or slightly angled vascular (Glover) clamps are placed across the entire neck of the pancreas. The pancreas is divided between the clamps and retracted away until the perforations in the portal vein or distal superior mesenteric or splenic veins are visualized. Alternatively, the neck of pancreas may be divided with a GIA or TA stapler as previously noted.

Hepatic Artery

Due to its short course, injury to any portion of the hepatic artery is rare. Replacement of the injured common hepatic artery with a substitute vascular conduit is rarely indicated, since most patients with a portal hematoma or hemorrhage have significant injuries to the liver, right kidney, or inferior vena cava also. As previously noted, ligation of the hepatic artery appears to be well tolerated in the young trauma patient. This is true even when performed beyond the origin of the gastroduodenal artery, owing to the extensive collateral arterial flow to the liver[170]; however, extensive necrosis of any repair of an associated injury to the liver may occur. Because of the small size of the right or left hepatic artery, lateral repairs are often difficult and will frequently be followed by occlusion of the vessel in the postoperative period.

Because of its rarity, few large studies have been performed on injuries to the hepatic artery. A relatively large multicenter experience was published in 1995 by Jurkovich et al,[171] which documented the course of 99 patients with injury to the portal triad. Of this group, 28 patients had 29 injuries to a segment of the hepatic artery. Nineteen patients underwent ligation, with eight survivors (mortality 42%). Only one patient developed hepatic necrosis requiring debridement, and this patient had an associated extensive injury to that lobe. Seven patients

TABLE 38-8: Survival with Injuries to the Portal Vein

Reference	Year	No. of patients	No. of survivors	Survival (%)
Graham et al[103]	1978	38	18	48.6
Petersen et al[174]	1979	28	17	60.7
Stone et al[102]	1982	41	22	53.7
Kashuk et al[80]	1982	9	3	33.3
Ivatury et al[175]	1987	14	7	50.0
Jurkovich et al[171]	1995	44[a]	20	45.4
Fraga et al[104]	2009	15	8	53.3
Overall		188	95	50.5

[a]Only patients who had repair or ligation; also, one patient had two injuries to portal vein.

had attempts at repair with only one survivor, and two other patients exsanguinated prior to therapy. It should be noted, again, that selective ligation of the right hepatic artery warrants a cholecystectomy.

Portal Vein

As noted earlier, injuries to any portion of the portal vein are more difficult to manage than are injuries to the hepatic artery, owing to the posterior location of the vein, the friability of its wall, and the greater blood flow through it. Techniques for repair of the vein are varied, but lateral venorrhaphy with a 4-0 or 5-0 polypropylene suture is preferred. More extensive maneuvers that have occasionally been used with success include the following: (1) resection with an end-to-end anastomosis; (2) interposition grafting; (3) transposition of the splenic vein down to the superior mesenteric vein to replace the proximal portal vein; (4) an end-to-side portacaval shunt; and (5) a venovenous shunt from the superior mesenteric vein to the distal portal vein or inferior vena cava.[80,100,172-174] Such vigorous attempts at restoration of blood flow have resulted from the concern about viability of the midgut if the portal vein is ligated. Unfortunately, any type of portal–systemic shunt may have the undesirable effect of causing hepatic encephalopathy because the direction of splanchnic venous flow with the shunt would mimic that in the patient with cirrhosis and hepatofugal flow in the obstructed portal vein. Ligation of the vein is compatible with survival, as Pachter et al,[173] Stone et al,[102] and Asensio[12] have emphasized. In the 1979 review of the literature on this subject by Pachter et al,[173] one of six survivors of ligation of the portal vein developed portal hypertension. The 1982 series by Stone et al[102] included 9 survivors among 18 patients who underwent ligation of the portal vein. In essence, ligation of the portal vein should be performed if an extensive injury is present and the patient is hypothermic, acidotic, and/or coagulopathic (damage control indicated). As previously noted, the surgeon must then be prepared to infuse significant amounts of fluids to reverse the transient peripheral hypovolemia secondary to splanchnic hypervolemia.[102]

To apply some perspective to the somewhat controversial area of injuries to the portal vein, a review of techniques for management is helpful. In the 1978 review by Graham et al[103] of 37 patients with injuries to the portal vein, 26 underwent lateral venorrhaphy, 5 had packing or clamping only, 4 had ligation (none of whom survived), 1 had an end-to-end anastomosis, and 1 had a portacaval shunt. In contrast, the aforementioned 1982 review by Stone et al[102] of 46 patients included 17 who had lateral venorrhaphy, 18 who had ligation (9 survived), 7 who had no repair, 3 who underwent an end-to-end anastomosis, and 1 who had a portacaval shunt.[102] Ivatury et al[175] reported on 14 patients with injuries to the portal vein in 1987, among whom exsanguination occurred in 3, venorrhaphy was performed in 10 (of whom 6 survived), and ligation was done in 1 (who survived). In 1995, Jurkovich et al[171] reported on 56 injuries to the portal vein with 33 patients undergoing primary repair (42% mortality), 1 undergoing complex repair (died), and 10 undergoing ligation (90% mortality). In 2009, Fraga et al[104] described primary suture repair in 15 patients with injury to the portal vein with an overall survival of 53%. Ten of these patients had stab wounds, and the survival rate was 60%.[104] An additional 11 patients died prior to definitive ligation and/or repair. In the seven series with greater than nine patients from 1978 to 2009, the mean survival rate was 50.5%[80,102-104,171,174,175] (Table 38-8).

Retrohepatic Vena Cava

The management of injuries to this vessel is described in Chapter 33. Newest approaches have included the following: (1) resuscitative balloon occlusion; (2) endovascular stent grafts for repair; (3) hybrid open and endovascular approaches; and (4) recognition of variability in length and diameter of suprarenal-to-suprahepatic caval segments as customized fenestrated endografts are developed.[176-179]

COMPLICATIONS

The complications of vascular repairs in the abdomen are much the same as those elsewhere in the body. They include postoperative thrombosis, dehiscence of a suture line, and infection.[180] Occlusion is not uncommon when small, vasoconstricted vessels, such as the superior mesenteric artery or renal artery, undergo lateral arteriorrhaphy. In such patients,

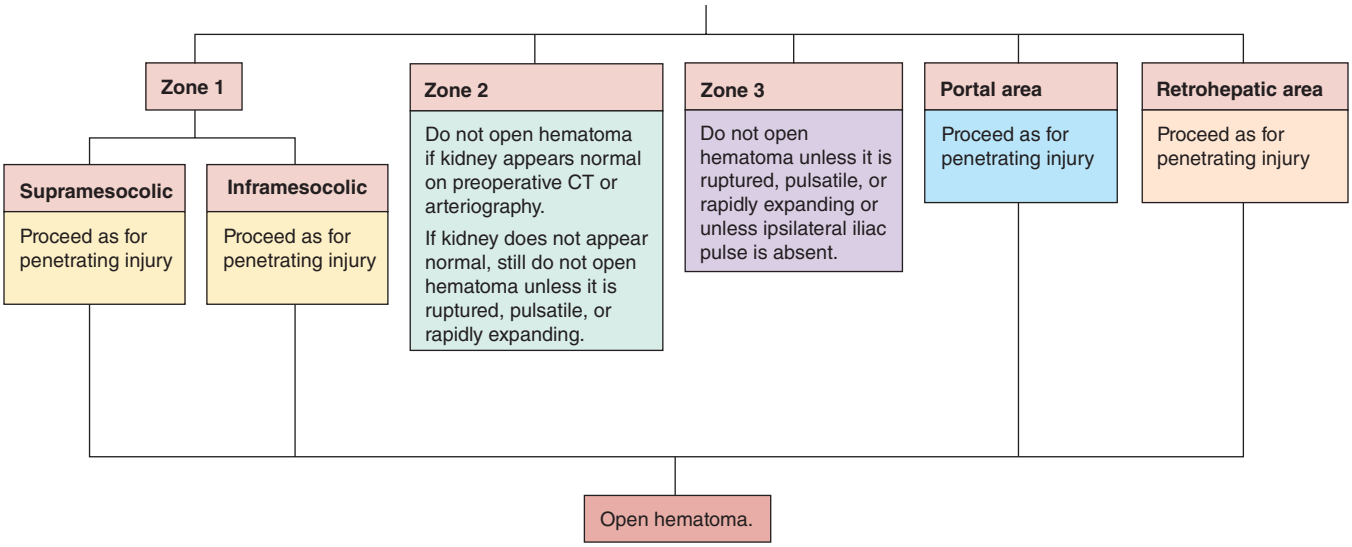

FIGURE 38-8 Blunt abdominal vascular injury algorithm. CT, computed tomography.

it may be valuable to perform a second-look operation within 12 to 24 hours after the patient's temperature, blood pressure, and coagulation abnormalities have returned to normal. When this is done, correction of an early vascular thrombosis may be successful.

Dehiscence of vascular suture lines in the abdomen has occurred in two locations in the authors' experience, and both have been previously discussed. First, a substitute vascular conduit inserted in the superior mesenteric artery near a pancreatic injury may be disrupted if a small pancreatic leak occurs in the postoperative period. For this reason, the proximal anastomosis of such a graft should be on the infrarenal abdominal aorta inferior to the transverse mesocolon and far away from the pancreas as previously noted. Second, the dehiscence of end-to-end anastomoses and graft-artery suture lines in the iliac arteries can be avoided by limiting the extent of repair if there is significant enteric or colonic contamination in the pelvis and considering an immediate or early femorofemoral bypass if the patient's limb is threatened, as described earlier.

Finally, a vascular complication unique to the abdomen is the postoperative development of vascular–enteric fistulas. This will occur most commonly in patients who have anterior aortic repairs, aortic interposition grafts, or grafts to the superior mesenteric artery from the aorta. Again, this problem can be avoided by proper coverage of suture lines on the aorta with retroperitoneal tissue or a viable omental pedicle and on the recipient vessel with mesentery. [112]

SUMMARY

Abdominal vessel injuries are most commonly seen in patients with penetrating wounds to the abdomen but occur after blunt abdominal trauma as well (Figs. 38-8 and 38-9). They

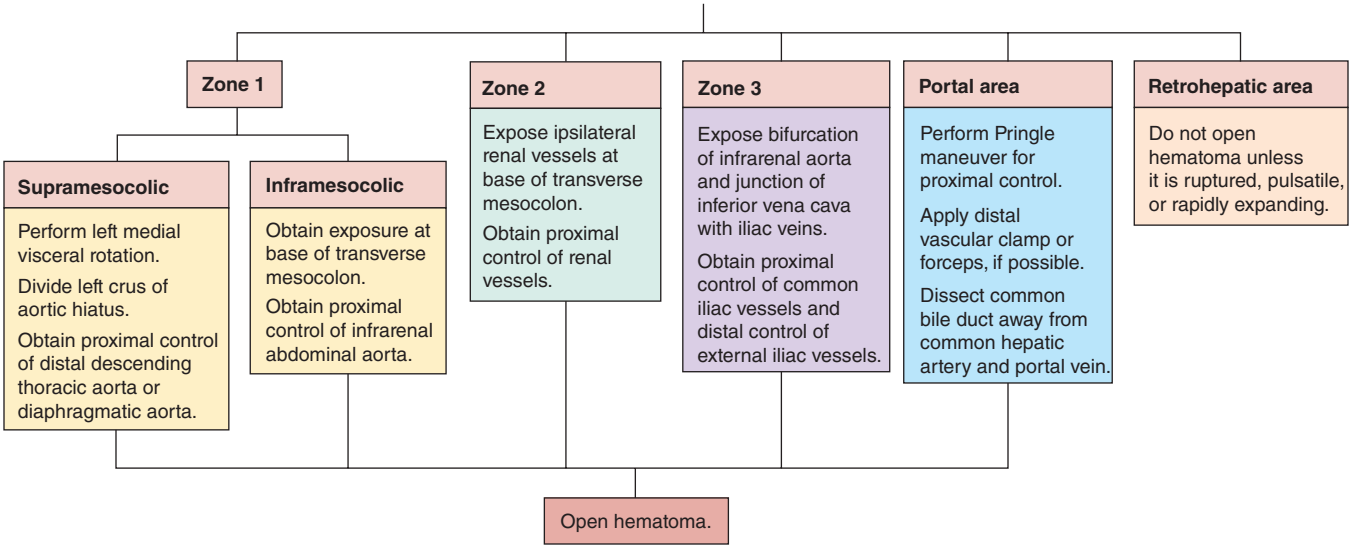

FIGURE 38-9 Penetrating abdominal vascular injury algorithm.

present either with a contained retroperitoneal, mesenteric, or portal hematoma; active hemorrhage; thrombosis; or a combination of these. When tamponade is present, proximal and distal vascular control should be obtained before opening the hematoma causing the tamponade. If active hemorrhage is present, direct compression of the bleeding vessels with a finger, hand, laparotomy pad, or sponge stick at the site of injury is necessary until proximal and distal vascular control can be obtained. Vascular repairs are generally performed with polypropylene sutures and can range from simple arteriorrhaphy or venorrhaphy to the insertion of substitute vascular conduits, much as in vascular injuries in the extremities. Also, in the occasional patient who presents with normal hemodynamics and angiographic evidence of a major vessel injury, thrombosis of a repair or other postoperative complication, or in a delayed fashion after an abdominal vessel injury, endovascular techniques have a role in management. Overall, if hemorrhage can be rapidly controlled and distal perfusion restored, many patients with major abdominal vessel injuries can be salvaged with the techniques described in this chapter.[73]

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Endovascular Commentary to Chapter 38: Abdominal Vessels

Joseph J. DuBose

The authors do an outstanding job of outlining the diagnosis of these injuries as well as their open management. Where does endovascular management fit in the contemporary care of these injuries? This is an important question that is made all the more important by the rapidly emerging capabilities of endovascular technologies. In order to adequately provide guidance on the potential utility of these approaches, one must present both the potential promise and the paradox of endovascular therapies in the current era.

First, I will touch on the potential of endovascular management for select patients. In a variety of emerging studies using multi-institutional data, patients managed with endovascular modalities versus traditional open approaches for vascular injury at noncompressible locations (including abdominal and iliac vessels) have been shown to have improved outcomes. It is also clear that these modalities are being employed more aggressively in this regard based on these findings.[1-3] The technologies are commonly available to modern vascular surgeons, who have become ever more facile with their expedient employment for a variety of vascular pathologies, including trauma.

While these findings are promising, the results of these efforts must be considered in the context of the significant limitations of present endovascular capabilities for trauma management. In outlining these limitations, it must first be acknowledged that the aforementioned promising results have been noted almost exclusively among patients with a major arterial injury confined to the aorta, iliac arteries, and the pelvic vasculature.[4] Additionally, these results are largely achieved only at high-volume centers with the right combination of both trauma volume and vascular expertise. Accordingly, the results of these efforts must be considered in context and

are not fair to extrapolate to a wider variety of environments. In addition, what are the most significant limitations of endovascular approaches?

First, a stable patient is almost universally required for endovascular management. Advanced imaging capabilities must be employed, appropriate vascular access achieved, specialized technicians to assist with device selection called in, and the appropriate providers summoned. The latter, at present, typically consist of formally trained vascular surgeons or interventionalists who are not present when the unstable patient demanding emergent hemorrhage control arrives. The delays required in mustering these capabilities are neither practical nor appropriate in the care of an unstable patient.

Second, endovascular technologies are not well suited to the care of patients with multiple pressing injuries that demand urgent attention. Open exposure within the abdomen allows for the rapid intervention for a variety of potentially life-threatening injuries. A facile trauma surgeon can pack a liver and remove a spleen in relatively short order. Endovascular procedures are confined to the intravascular space alone and require focus on one portion of the arterial tree at a time. The use of these technologies simply limits the ability to rapidly address multiple injuries in the same fashion as can be achieved by open means.

Third, there remains a need to define the long-term durability of endovascular approaches. Although embolization of the pelvis or solid organs is now a mature practice that has demonstrated its worth, the endovascular repair of a major vascular injury with a stent graft is a newer capability comparatively. Although efforts are underway to obtain longitudinal data on endovascular repair of traumatic arterial injuries after treatment, the known durability of these devices remains a matter of needed examination. This is especially true when suggesting they replace the "tried and true" open repair modalities (interposition repair with native or synthetic conduits) available for use.

Even among stable patients, the urge to overtreat certain injuries must be avoided. Among more minor aortic abdominal injuries, for example, it has been shown that the majority of these injuries will heal without sequalae when managed nonoperatively.[5] The aggressive employment of endovascular stent grafts for these injuries may, therefore, introduce unnecessary risk for the patient.

There are, however, areas where endovascular potential is possibly clearer. The stable patient with a "hostile" or reoperative abdomen and a lesion amenable to the existing capabilities of endovascular technologies would seem an ideal candidate. So too might the delayed diagnosis of specific injuries such as a traumatic false or true aneurysm or an arteriovenous fistula. Many delayed pseudoaneurysms of larger arteries likely behave clinically in a fashion similar to pathologies familiar to vascular surgeons facile with endovascular treatment.

Moving forward, there may even be a potential future for the adoption of "hybrid" approaches in select patients. In this fashion, the traditional "open versus endovascular" considerations give way to an evolved consideration of these technologies as complementary in specific situations. Resuscitative endovascular balloon occlusion of the aorta (REBOA) represents one such "hybrid" facilitator. Although still a matter of active investigation, this technology has already demonstrated its potential in affording emergent proximal aortic control prior to entry into the abdomen to surgically address injuries using traditional approaches.[6] Theoretically, among more stable patients, hybrid partnerships between endovascular and open providers might afford proximal arterial control in a similar fashion at a variety of intra-abdominal locations of arterial injury. These hybrid techniques are already used commonly at several high-volume academic centers. Further study is required, however, to guide optimal patient selection and occlusion practices using this modality.

And so, endovascular capabilities do demonstrate significant potential for improving outcomes in select patients after abdominal vascular injury. In stable patients with appropriate capabilities and expertise present, they can be employed in staged or hybrid approaches to good effect. In order to optimally define these capabilities (and limitations), however, significant additional study is required.

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39

Pelvis

Aussama Nassar • Lisa Knowlton • David A. Spain

KEY POINTS

- The anterior elements of the pelvis, including the pubic rami and pubic symphysis, only contribute to approximately 40% of pelvic stability.
- In the Tile classification of pelvic fractures, type A are stable, type B are vertically stable but rotationally unstable, and type C are vertically and rotationally unstable.
- In the Young-Burgess classification of pelvic fractures, the three types are anteroposterior compression (APC), lateral compression, and vertical shear.
- Pelvic binders are indicated for APC ("open book") fractures when first diagnosed, but not for lateral compression fractures.
- Early external fixation stabilizes the fractured elements, decreases the pelvic volume, and allows clot to form.
- For the patient who is exsanguinating from a closed pelvic fracture, embolization of the bilateral internal iliac arteries is considered to be the "damage control" procedure of choice.
- In the technique of preperitoneal pelvic packing for hemorrhage, three laparotomy pads are placed on either side of the bladder in the retroperitoneum.
- APC III, most lateral compression II, and vertical shear pelvic fractures typically require posterior stabilization by internal fixation.
- The mean Injury Severity Score (ISS) for patients with pelvic fractures is 18, reflecting the significant number of patients with associated injuries to the brain, thorax, abdomen, and long bones.
- Control of bleeding in open pelvic fractures includes packing through the laceration, application of a pelvic binder, angiographic embolization, and definitive bony fixation.

INTRODUCTION

Pelvic ring fractures (PRFs) are frequent, particularly after blunt trauma (9% of all blunt trauma patients), and range from clinically insignificant minor pelvic fractures to life-threatening injuries that can lead to exsanguination (0.5% of all blunt trauma patients). The overall mortality rate of patients with PRFs is approximately 8%.[1] Anterior-posterior compression and vertical shear injury mechanisms are associated with a higher incidence of pelvic vascular injury and hemorrhage. Pelvic fractures are one of the insidious, unrecognized sources of shock and death in polytrauma patients with other distracting injuries. This is largely due to the fact that an actively bleeding pelvis is not usually appreciated on clinical exam or focused abdominal sonography for trauma (FAST) and occasionally does not present with active extravasation of contrast on computed tomography (CT) of the pelvis. These complicating factors make the management of an exsanguinating pelvic fracture challenging, with lack of consensus on a single best management approach. The recent evolution of rapid pelvic stabilization by pelvic binding or external fixation during the trauma primary survey, combined with a selective approach to preperitoneal pelvic packing and angiographic embolization, has significantly decreased the mortality rates of devastating PRF. Resuscitative endovascular balloon occlusion of the aorta (REBOA) has been introduced as a promising hemorrhage control modality in the immediate management of exsanguinating PRF, which will be discussed later in this chapter. A multidisciplinary coordinated approach is crucial in managing patients with PRF, as there is no single treatment modality that has been shown to be the gold standard.

PELVIC ANATOMY

The pelvic ring comprises the sacrum and the two innominate bones, all attached with strong ligaments. The innominate bones join the sacrum at the sacroiliac joints and each other

anteriorly at the pubic symphysis. The anterior and posterior sacroiliac ligaments include shorter and longer elements that extend over the sacrum and to the iliac crests, and provide vertical stability across the sacroiliac joints. The pelvic floor is bridged by the sacrospinous and sacrotuberous ligaments that connect the sacrum to the ischial spine and the ischial tuberosity, respectively. The anterior elements, including the pubic rami and pubic symphysis, contribute to approximately 40% of pelvic stability, but the posterior elements are more important, as shown by biomechanical studies.[2]

The internal iliac (hypogastric) arteries provide blood supply to the organs, bones, and soft tissues of the pelvis. The anterior division includes the inferior gluteal, obturator, inferior vesicular, middle rectal, and internal pudendal arteries. The posterior division includes the iliolumbar, lateral sacral, and superior gluteal arteries. The largest branch is the superior gluteal artery, which is the most commonly injured major arterial branch after pelvic fractures. Pelvic veins run parallel to the arteries and form an extensive plexus that drains into the internal iliac veins. The sacral venous plexus is adhered to the anterior surface of the sacrum and is often shredded after major pelvic fractures. Thus, venous bleeding or bleeding from bony fragments accounts for approximately 85%, with the remainder stemming from an arterial source.[3]

The sciatic nerve is formed by the nerve roots of L4 to S3 and exits the pelvis under the piriformis muscle. The anterior roots of L4 and L5 cross the sacroiliac joints and can be injured in sacral ala fractures or sacroiliac joint dislocations.

All pelvic organs are at risk of injury following severe PRF, with the bladder and urethra being the most frequently injured. The extraperitoneal rectum and vagina are also at risk of injury from the sharp pelvic bone fragments and convert the type of fracture to an open pelvic fracture.

CLASSIFICATION OF PELVIC FRACTURES

Despite multiple classification systems described, the two most commonly used are those described by Tile[3] and Young and Burgess.[4,5]

The Tile classification categorizes pelvic fractures in three groups based on stability, as evaluated primarily by clinical examination and plain radiographs (Table 39-1):

- Type A fractures are stable, as the posterior ligaments are intact. These fractures include transverse sacral, iliac wing, pubic rami, pure acetabular, and chip and avulsion fractures.
- Type B fractures are caused by internal and external rotational forces and are "partially" stable (vertically stable but rotationally unstable). They include open-book and bucket-handle fractures (Figs. 39-1 to 39-3).
- Type C fractures are vertically and rotationally unstable, as they involve a complete disruption of the sacroiliac complex (Fig. 39-4).

The Young-Burgess classification divides pelvic fractures according to the vector of the force applied into anteroposterior compression (APC), lateral compression (LC), and vertical shear fractures (Table 39-2):

- APC injuries are produced by forces applied in the sagittal plane, as is usually the case with motor vehicle crashes. APC-I injuries may result in a small widening of the pubic symphysis (<2.5 cm), but the posterior ligaments are intact. APC-II injuries include tearing of the anterior sacroiliac ligaments as well as the sacrospinous and sacrotuberous ligaments, but the posterior sacroiliac ligaments are intact. The pubic symphysis diastasis may be more than 2.5 cm. Rotational instability is usually present and hemorrhage more likely. APC-III injuries are caused by high-energy transfer, and the posterior sacroiliac ligaments are disrupted, causing full instability of the hemipelvis with a high likelihood of bleeding, nerve damage, and organ injuries.
- LC injuries are produced from lateral impacts across the horizontal plane, also common with motor vehicle crashes. LC-I injuries include transverse fractures of the anterior ring or impacted sacral fractures, and are typically stable. LC-II injuries are caused by higher-energy forces that produce tearing of the posterior sacroiliac ligament and displacement of the sacroiliac joint or an oblique fracture of the ilium, the superior part of which remains attached to the sacrum, while the inferior is mobile (crescent fracture). Depending on the force applied, this fracture can be stable or unstable. LC-III injuries are severely unstable fractures, as the lateral force continues to compress and rotate the hemipelvis to the point of complete destruction of the sacroiliac joints, as well as the sacrospinous and sacrotuberous ligaments. Neurovascular and organ injuries are common.
- Vertical shear injuries are typically produced by a fall from a height and involve anterior (pubic rami, pubic symphysis) and posterior (sacroiliac complex) fractures. Typically, they are unstable.
- Combinations of the above types produce a variety of fracture patterns, most commonly involving LC and vertical shear injuries. Nearly one-third of PRF patients have combination injuries.

DIAGNOSIS

Physical examination occasionally establishes the diagnosis for a PRF. Before the availability of sophisticated imaging, the optimal bedside physical exam maneuver was by the examiner placing his or her hands on the anterior iliac spines of the patient, then exerting gentle compression toward the midline, followed by a mild divergent movement of the hands toward the outside; these combined maneuvers would assess for pelvic instability. This was done by an experienced physician who would interrupt the motion immediately if instability or a fracture was felt so as not to produce pain and aggravate bleeding. Careful, detailed inspection of the perineum and skin overlying the pelvic bones is critical to diagnose lacerations or hematomas that might aid in the diagnosis of severe PRF. In addition to assessing the perineum, there must be a

TABLE 39-1: Tile Classification of Pelvic Fractures

Type			Characteristics	Hemipelvis displacement	Stability
Type A, posterior arch intact	A1, pelvic ring fracture (avulsion)	A1.1	Anterior iliac spine avulsion	None	Stable
		A1.2	Iliac crest avulsion		
		A1.3	Ischial tuberosity avulsion		
	A2, pelvic ring fracture (direct blow)	A2.1	Iliac wing fracture	None	Stable
		A2.2	Unilateral pubic rami fracture		
		A2.3	Bilateral pubic rami fracture		
	A3, transverse sacral fracture	A3.1	Sacrococcygeal dislocation	None	Stable
		A3.2	Nondisplaced sacral fracture		
		A3.3	Displaced sacral fracture		
Type B, incomplete posterior arch disruption	B1, anteroposterior (AP) compression	B1.1	Pubic diastasis, anterior sacroiliac (SI) joint disruption	External rotation	Rotationally unstable, vertically stable
		B1.2	Pubic diastasis, sacral fracture		
	B2, lateral compression	B2.1	Anterior sacral buckle fracture	Internal rotation	Rotationally unstable, vertically stable
		B2.2	Partial SI joint fracture/subluxation		
		B2.3	Incomplete posterior iliac fracture		
	B3.1, AP compression	B3.1	Bilateral pubic diastasis, bilateral posterior SI joint disruption	External rotation	Rotationally unstable, vertically stable
	B3.2, AP and lateral compression	B3.2	Ipsilateral B2 injury, contralateral B1 injury	Ipsilateral internal rotation, contralateral external rotation	Rotationally unstable, vertically stable
	B3.3, bilateral lateral compression	B3.3	Bilateral B2 injury	Bilateral internal rotation	Rotationally unstable, vertically stable
Type C, complete posterior arch disruption	C1, vertical shear	C1.1	Displaced iliac fracture	Vertical (cranial)	Rotationally unstable, vertically unstable
		C1.2	SI joint dislocation or fracture/dislocation		
		C1.3	Displaced sacral fracture		
	C2, vertical shear and AP/lateral compression	C2	Ipsilateral C1 injury, contralateral B1 or B2 injury	Ipsilateral vertical (cranial), contralateral internal or external rotation	Rotationally unstable, vertically unstable
	C3, bilateral vertical shear	C3	Bilateral C1 injury	Bilateral vertical (cranial)	Rotationally unstable, vertically unstable

thorough exam of the genitalia for any evidence of urethral injury, especially in males. A careful vaginal exam should be performed in women to assess for possible lacerations. In a study of 66 patients with a Glasgow Coma Scale score over 12, a focused physical examination protocol, including posterior palpation of the sacrum and sacroiliac joint, anteroposterior and lateral iliac wing compression, active hip range of motion, and a digital rectal examination, resulted in 98% sensitivity and 94% specificity for the detection of posterior pelvic fractures.[6]

The plain anteroposterior pelvic film is an important adjunct to the primary survey. Some studies indicate that pelvic films are unnecessary in patients with a negative clinical exam for PRF. In a review of 743 blunt trauma patients with no pain or other clinical findings of PRF, only three patients (0.4%) had a pelvic fracture.[7] In all cases it was a single, nondisplaced pubic ramus fracture that required no treatment. In another study of 686 blunt trauma patients, 311 received a pelvic film, which carried a false-negative rate of 32%.[8] Although plain films of the pelvis have their limitations and patients often undergo more detailed evaluation by CT, they have a useful role in screening for pelvic disruption early in the assessment of a trauma patient.[9] This is of particular importance since bleeding from a pelvic source is often missed on FAST. In the hemodynamically stable patient with a negative FAST but abdominal or pelvic tenderness on exam, this film may be omitted because an abdominopelvic CT will be performed.

CT scan with reconstructions (and recently three-dimensional reconstructions) is routinely performed to accurately characterize pelvic fractures and associated pelvic hematomas, as well as identify associated pelvic organ injuries (Fig. 39-5). Intravenous contrast is routinely administered unless there is a contraindication. Oral and rectal contrast are not necessary for blunt trauma.

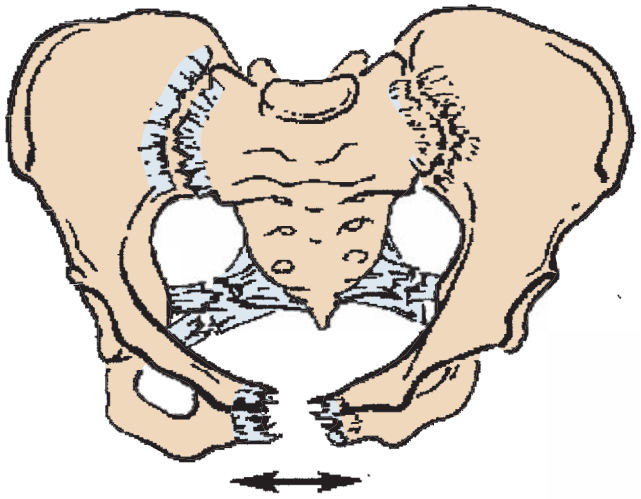


FIGURE 39-1 Type B1 open-book injury. Diastasis of the pubic symphysis and fracture of the superior or inferior pubic rami may occur.

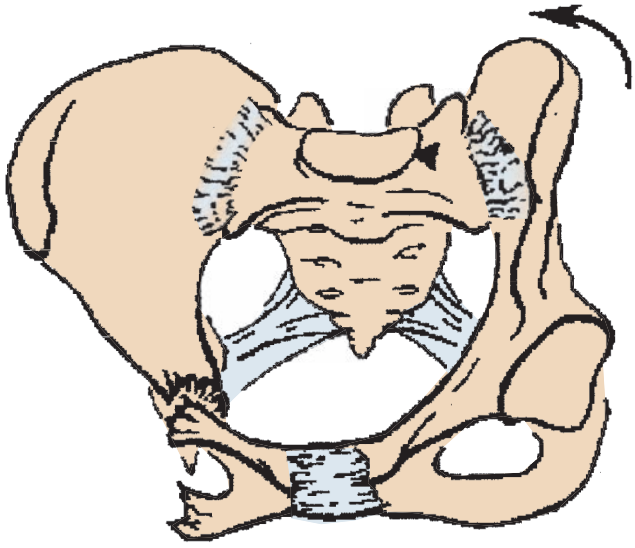


FIGURE 39-3 Type B3 lateral compression (contralateral) or bucket-handle injury. Note anterior rami fracture with contralateral posterior sacroiliac injury.

Magnetic resonance imaging (MRI) does not offer a distinct advantage over CT scan and is only rarely considered if radiation exposure becomes an issue, as it is with nonemergent cases of pediatric patients, pregnant patients, or repeat imaging. On occasion, the ligaments need to be evaluated in more detail, and this can be done with higher accuracy by magnetic resonance. It is important to remember, however, that MRI has limited usefulness in the acute diagnosis of pelvic trauma, as trauma patients would be at undue risk for hemorrhagic complications and shock during their evaluation in the MRI suite.

The FAST exam has become a routine part of the primary survey and initial assessment of trauma patients. FAST exam is only able to confirm the presence of intraperitoneal fluid with limited assessment of the retroperitoneal space and inability to qualify the nature of the intraperitoneal fluid. In PRF, a number of FAST exam findings can be useful: (1) the absence of intraperitoneal fluid in a hemodynamically unstable patient should raise the possibility of a major retroperitoneal hemorrhage from PRF (if no other obvious sites of bleeding and no nonhemorrhagic reasons for shock); (2) a distorted bladder contour indicates the presence of a compressing pelvic hematoma; and (3) the presence of intraperitoneal fluid indicates that intraperitoneal organ injury must be excluded by additional diagnostic methods or laparotomy. However, there can be false-negative or equivocal

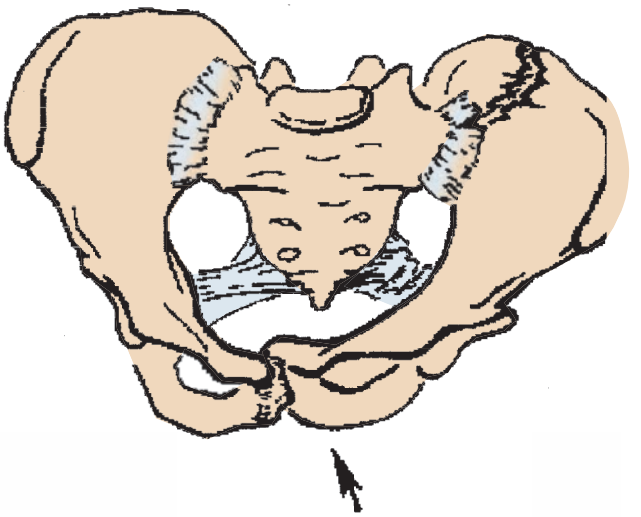


FIGURE 39-2 Type B2 lateral compression (ipsilateral) injury. Note overriding of the left hemipelvis and crush injury to ipsilateral sacrum and ipsilateral iliac fracture.

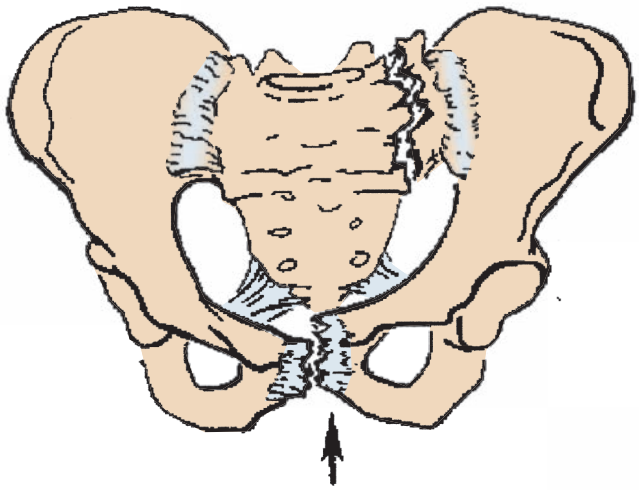


FIGURE 39-4 Type C1 unilateral injury with vertical instability. Involves symphysis disruption or rami fracture with ipsilateral sacroiliac joint or sacral injury.

TABLE 39-2: The Young-Burgess Classification of Pelvic Fractures

Mechanism and type	Characteristics	Hemipelvis displacement	Stability
Anteroposterior (AP) compression, type I	Pubic diastasis <2.5 cm	External rotation	Stable
AP compression, type II	Pubic diastasis >2.5 cm, anterior sacroiliac (SI) joint disruption	External rotation	Rotationally unstable, vertically stable
AP compression, type III	Type II plus posterior SI joint disruption	External rotation	Rotationally unstable, vertically unstable
Lateral compression, type I	Ipsilateral sacral buckle fractures, ipsilateral horizontal pubic rami fractures (or disruption of symphysis with overlapping pubic bones)	Internal rotation	Stable
Lateral compression, type II	Type I plus ipsilateral iliac wing fracture or posterior SI joint disruption	Internal rotation	Rotationally unstable, vertically stable
Vertical shear	Vertical pubic rami fractures, SI joint disruption ± adjacent fractures	Vertical (cranial)	Rotationally unstable, vertically unstable

FAST exams with PRF. Thus, although diagnostic peritoneal lavage has nearly completely disappeared from algorithms in most modern trauma centers, diagnostic peritoneal aspiration (DPA) does have a role on rare occasions with hemodynamically unstable PRF patients and negative or equivocal FAST. In addition, some use the aspiration portion of it only (DPA) to further characterize the nature of the free peritoneal fluid (blood, succus, or ascites).[10] In patients with PRF, the procedure should be performed above the umbilicus to avoid pelvic hematoma.

MANAGEMENT OF BLEEDING PELVIC RING FRACTURES

The management approach to acutely bleeding PRF focuses on reducing pelvic volume to secondarily reduce the amount of bleeding, stabilize the fractured segments, and prevent clot dislodgement. Hemodynamically unstable patients with suspected PRF should be assumed to be bleeding from a pelvic fracture source until proven otherwise. Patients should be resuscitated as per Advanced Trauma Life Support (ATLS) protocol. The concept of permissive hypotension (ie, accepting a lower than normal blood pressure during the early phases of resuscitation in order to prevent ongoing bleeding and other known sequelae of overzealous resuscitation) has been adequately established for penetrating trauma; however, it is not universally accepted for blunt trauma despite the encouraging reports.[11,12] The coexistence of neurologic injuries, which have been shown to produce worse outcomes in the presence of hypotension, is the main deterrent to allow a low blood pressure in a hemodynamically unstable blunt trauma patient. In a recent prospective randomized study from 19 emergency medical services of the Research Outcomes Consortium, 192 hypotensive trauma patients were assigned in the prehospital setting to receive crystalloid resuscitation to maintain a blood pressure above 70 mm Hg (controlled resuscitation group) or above 110 mm Hg (standard resuscitation group). There was no difference in most outcomes, but patients with blunt trauma in the controlled resuscitation group had a lower 24-hour mortality (3%) compared to the standard resuscitation group patients (18%). Although we still need phase III studies to evaluate the different resuscitation regimens, this study offers some evidence to suggest that at least there is no harm from limiting the initial crystalloid resuscitation and it may arguably be beneficial in hypotensive trauma patients regardless of the type of injury.[13] We advocate the principles of permissive hypotension (assuming no concern for traumatic brain injury), even in blunt trauma, with limited use of crystalloids and early use of blood products for resuscitation. There is no proven gold standard intervention for managing bleeding pelvic fractures. A multimodality approach with a combination of pelvic binders, interventional radiology, and operative management should be considered.

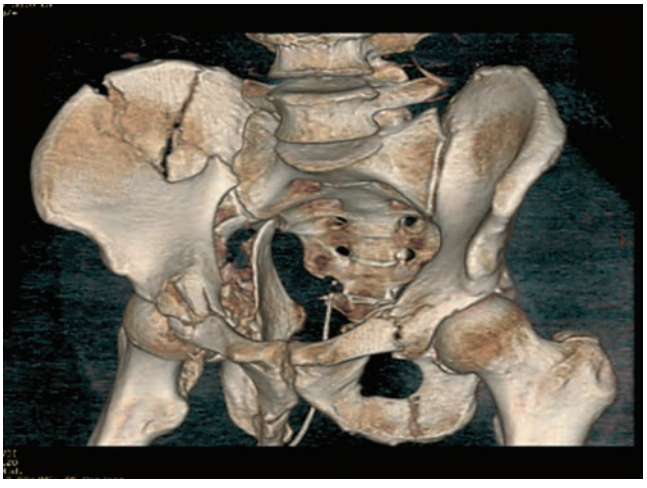


FIGURE 39-5 Three-dimensional computed tomographic reconstructions provide a realistic assessment of the anterior and posterior elements in pelvic fractures.

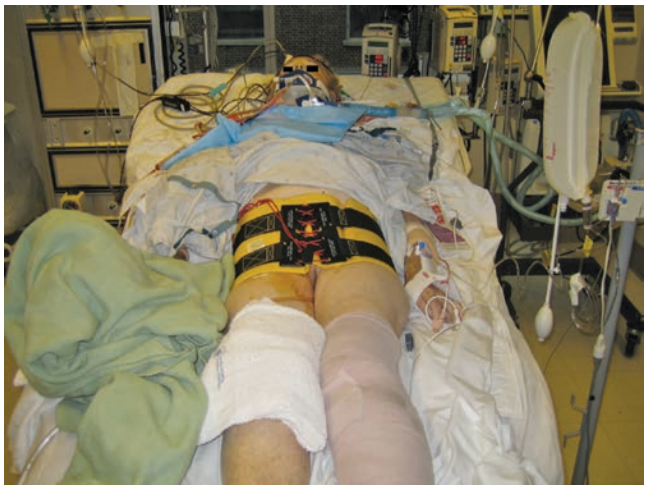


FIGURE 39-6 Pelvic binder.

Pelvic Binders

Pelvic binders have been traditionally used as the primary temporizing measure in bleeding control of PRF both in the prehospital setting and in the trauma bay.[14-17] A binder is primarily indicated to stabilize the pelvis and reduce the volume after a pelvic x-ray confirms an open-book fracture. The thought behind the wrap is to decrease the space of the unstable disrupted pelvis, which can contain several liters of blood. It is our practice to follow the pelvic binder with a second pelvic x-ray to confirm reduction of an open-book fracture. Pelvic binders also act by stabilizing bony fragments and helping to slow bleeding (Fig. 39-6). In contrast, binding a significant LC mechanism pelvic fracture can have the opposite effect and can increase bleeding (Fig. 39-7). Under these principles, simple stabilization by a pelvic device is desirable in all unstable fractures, but significant compression should only be used in the open-book variety.

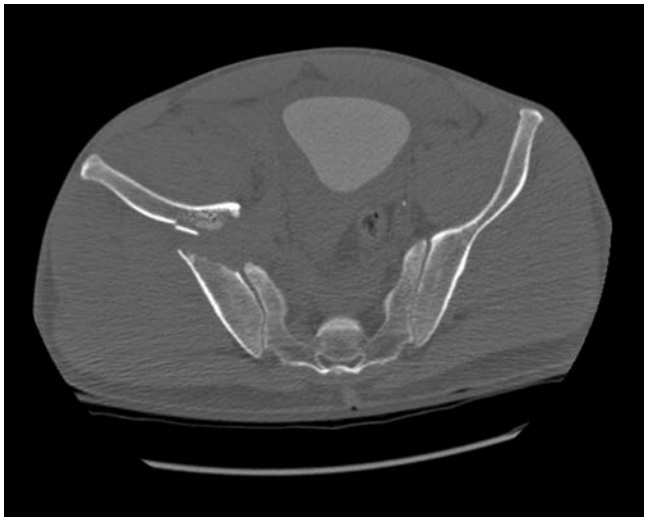
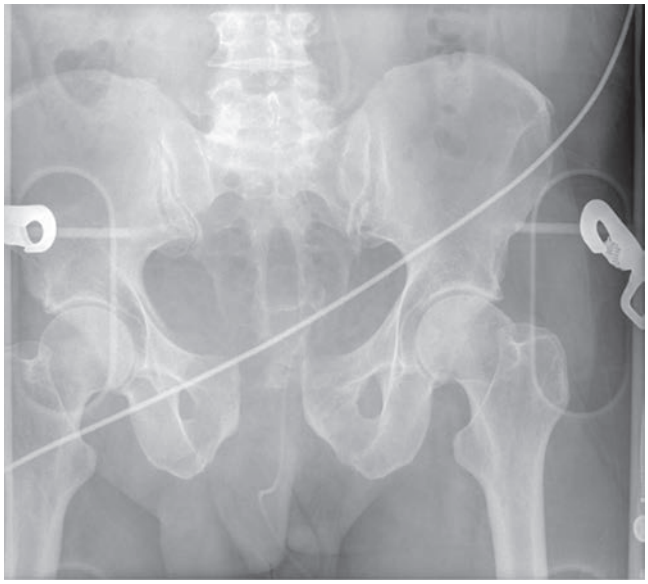


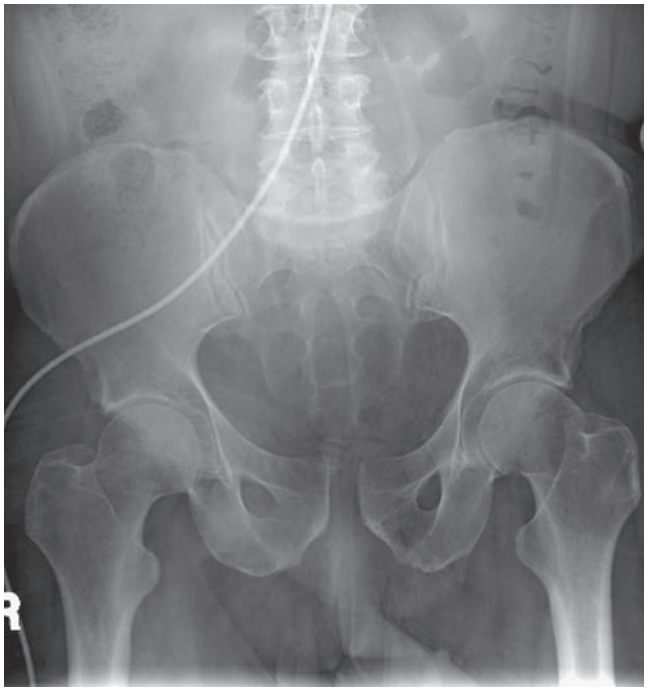
FIGURE 39-7 A fracture that would be inappropriate for a tight pelvic binder. Displacement with forceful lateral compression by the binder could be exacerbated and result in vascular injury.

A bed sheet is frequently used as an immediately available and inexpensive way to wrap the pelvis.[17] The edges of the sheet are tied together around a rod, which can be turned to tighten the sheet and apply the desired degree of compression (Fig. 39-8).

Pelvic binders have been commercialized by different companies along the same principles with only a few differences



A



B

FIGURE 39-8 A simple sheet wrapped around the pelvis produces inexpensive and adequate reduction of a pelvic fracture. A significant pubic diastasis, as shown in the first image (**A**), is reduced by the sheet, as shown in the following image (**B**).

among them. Typically, a binder consists of a wide belt with a Velcro that attaches the two ends of the binder (which can be cut to customize its length according to the patient's body habitus). On the belt, there is a "buckle pulley" mechanism. By pulling the strings, the binder tightens and compression increases. The device is radiolucent, which allows radiographic imaging with no artifacts. The evidence on the effectiveness of pelvic binders is poor.[14,15] There are three major pitfalls related to its use after x-ray confirmation of an open-book pelvic fracture. First, an inappropriately high placement of the binder can lead to excessive abdominal pressure and minimal pelvic stabilization. It is not uncommon to place the binder too high. Correctly, the binder must be centered around the greater trochanters and not over the iliac crests. Usually, it needs to be gently passed under the patient's back and then pulled slightly lower and over the buttocks for proper placement. Second, indiscriminate pulling of the strings can lead to greater compression than necessary. The initial step should involve only moderate tightening if a pelvic x-ray has not yet been performed. The pulley mechanism attached on the binder makes tightening very easy, and with minimal force, an enthusiastic operator can squeeze the binder tight, producing on occasion more harm than good. Third, the binder may compromise the viability of skin, subcutaneous tissue, or even muscle if left in place for too long. A general guideline of a maximum 24-hour placement exists, but obviously even this may be too long after a tight application of the binder.[16] The health care providers should understand that the binder is only an imperfect and temporary tool for bleeding control. Definitive pelvic reduction and cessation of hemorrhage should be planned immediately in order to minimize the need for a binder.

The military antishock trousers (MAST) garment became popular in the 1980s after initial reports of improved survival in patients with multiple injuries. However, in 1989, a definitive prospective randomized study found it to be associated with increased mortality, mostly related to respiratory compromise and compartment syndrome due to its interference with blood supply.[18] MAST was for the most part abandoned, although some emergency medical systems still use it on patients with pelvic or lower extremity fractures. *Based on the data, use of MAST should be abandoned.*

External Fixation

External fixation has been popularized as a rapid means of controlling bleeding. In a few institutions, this can be accomplished in the emergency department, but in most centers, the patients are transferred in the operating room. A number of clamps and devices have been used to provide external fixation. The C-clamp was designed for easy placement in the emergency department in the presence of a posterior pelvic fracture.[19] As opposed to other fixators, it is easy to assemble and apply. Its crossbar rotates around the fixation pins, which are anchored in the cancellous bone in both acetabula. The rotation of the clamp allows other procedures in the abdomen or pelvis to be offered without difficulty. The pins can be placed more anteriorly or posteriorly according to the location of the pelvic fracture and the need to reduce them. It is clearly a temporary method, which needs to be replaced later with either a proper pelvic frame or internal fixation. The C-clamp has been used more frequently in European than American trauma centers, which typically prefer a frame placed in the operating room.

Early external fixation stabilizes the fractured elements, decreases the pelvic volume, and allows clot to form. A variety of external fixators exist. The early systems used small pins and heavy bars, whereas the newer systems are more compact, are easy to adjust, and have larger pins. The standard placement of pins is in the superior iliac crest above the superior anterior iliac spine. Lower placement of the pins is also acceptable and can improve the access to the abdominal cavity. In certain designs, more than one pin is placed on each side. Pins can be placed by an open or percutaneous technique. All single-bar systems require two pins in each hemipelvis, whereas the frames require three pins on each side, except the Pittsburgh system, which requires two clusters of two pins in each hemipelvis. In most cases of a true unstable pelvis, external fixators remain a temporary device, which bridges the period to definitive internal fixation. In the supine position, external fixation provides adequate stability. In the standing position, the vertical load is usually greater than the capacity of the external fixator to resist these forces. Dislocation of fractured elements can happen, particularly at the sacroiliac complex.[20]

After placement of the frame, reduction of the pelvic fracture is done by applying opposite forces to the ones that created the fracture. Open-book fractures are corrected by internal rotation of the pins, whereas LC fractures are reduced by external rotation. Vertical shear fractures require skeletal traction by placement of a femoral pin, and are the ones least likely to be adequately reduced and stabilized by external fixation.[21]

If not converted to internal fixation, external fixators usually stay for 6 to 12 weeks. The most common complication is infection at the pin sites, ranging from mild to severe. Appropriate sterile technique during pin placement and proper care of the pin sites are essential to avoid infection. If the pins become infected or loose, they must be removed and replaced. Other complications are typically associated with placement and include injury to the lateral femoral cutaneous nerve or other neurovascular structures.

In general, it seems that external fixation should be considered in two stages, an early resuscitative and a later definitive stage. In the early stage, the fixator is placed to stabilize the fracture and help control the bleeding. At a later stage, a decision must be made about the long-term effectiveness of the external frame versus the need to convert to internal fixation. LC fractures are likely to respond to external fixation as the only method, if reduction is satisfactory. Vertical shear fractures are unlikely to be managed without definitive internal fixation.[22] Each patient must be carefully assessed to balance the therapeutic choices of fixing the fracture while maintaining hemodynamic stability and inflicting the

minimum physiologic insult during the initial critical hours after trauma.

Angiographic Embolization

PRF producing hemodynamic instability is one of the most common indications for angiographic embolization. Historically, surgical ligation of the internal iliac arteries was performed to control bleeding associated with pelvic fractures. This technique has proven to be ineffective given the rich collateral blood supply to the pelvis.[23] The ability to control the bleeding by minimally invasive techniques and without the need for an operation, which is routinely unsatisfactory, is very appealing. The appeal is hampered, however, by the unavailability of interventional radiology teams around the clock, the presence of minimal contrast extravasation on some CT scans, the poor monitoring available in an angiography suite, and the long time spent on the angiography table. All these reasons have been mitigated in modern trauma centers.

Interventional radiology teams are now readily available on short notice in most Level I trauma centers. Monitoring and resuscitation in the angiography suite should be no different in a Level I trauma center than it is in the operating room. High-rate fluid infusion devices, noninvasive hemodynamic monitoring, mechanical ventilatory support, arterial blood gas assessments, blood transfusions, and aggressive resuscitation efforts should take place during angiography.

The first challenge for the trauma and interventional radiology teams is to decide if the patient would benefit from angiographic embolization. Active arterial contrast extravasation (blush) on angiography and the presence of pelvic hematoma in a hemodynamically abnormal patient with PRF are accepted indications for embolization. CT scan has a sensitivity of 60% to 90% and a specificity of 92% to 100% in predicting the need for angioembolization. Unfortunately, approximately one-fourth of the angiographies performed for PRF find no direct or indirect evidence of ongoing bleeding (blush) on angiography.[24] There are no controlled studies in the present literature, however, and the precise indications are unknown. In 97 patients with pelvic fractures retrospectively reviewed, no factors predicted a positive angiogram with sufficient likelihood.[22] Mechanism of trauma, injury severity, hemodynamic presentation, and associated injuries were similar between patients with and without radiographic evidence of pelvic bleeding. In a later prospective study by the same group, 65 patients with pelvic fractures were included in the study total of 100 consecutive patients evaluated by angiography for bleeding.[25] Three independent predictors of bleeding were identified: age older than 55 years, absence of long-bone fractures (indicating that the pelvis was the main source for bleeding), and emergent angiography (indicating that semi-acute interventions had a lower likelihood of identifying bleeding). The predictive effect of age was confirmed by another prospective observational study.[26] Approximately 94% of patients older than 60 years of age had a positive angiogram, as opposed to 52% of younger patients. The authors recommended that angiographic embolization be offered liberally to pelvic fracture patients over 60 years old.

Pelvic fracture pattern is considered a major predictor of bleeding. Traditionally, three types of PRF are considered to be associated with hemorrhage: pubic symphysis diastasis of more than 2.5 cm, bilateral superior/inferior pubic rami fractures (butterfly), and posterior fractures (especially of the vertical shear variety).[27] There is evidence, however, that even anterior fractures can produce bleeding,[24] particularly in older patients or those receiving anticoagulants. The presence of contrast extravasation on pelvic CT scan has also been widely used as a predictor of a positive angiogram.[28,29] It is suggested that the sensitivity and specificity of a "contrast blush" on CT to identify bleeding that requires embolization are 84% and 85%, respectively, with an overall accuracy of 90%.[29] Our experience has been that the new-generation CT scanners are highly sensitive and—in combination with precise intravenous (IV) contrast infusion protocols—may pick up small bleeds that are potentially self-limited without further intervention. Therefore, the mere presence of contrast extravasation on CT is not an immediate indication for angiography in our institution. We consider contrast extravasation a crucial element of the constellation of symptoms, signs, and findings of PRF and consider it in the context of the entire clinical picture. A patient who is hemodynamically labile and has a contrast blush is emergently transferred to the angiography suite. A patient with a contrast blush, who is hemodynamically stable and has a small pelvic hematoma, does not usually receive a preemptive angiogram but rather is placed under close observation. In a study of 296 patients with pelvic fractures, 40% were found to have extravasation on CT, but only half eventually required some form of pelvic hemorrhage control.[30] Similarly, the size of pelvic hematoma cannot be used as an isolated indication for angiography.[31]

Interventional radiologists typically seek to identify the precise site of bleeding and control it with selective embolization via coils. This requires subselective intubation of internal iliac artery branches, time, and larger doses of IV contrast. For true trauma emergencies, the interventional radiologist should be in a different mindset. In alignment with surgical damage control principles, damage control angiography should be offered. The procedure should be rapid, effective, and temporary. Bilateral internal iliac artery embolization embraces these principles. The interventional radiologist does not consume time maneuvering small catheters into small arterial branches. The bleeding is controlled by truncating all the branches of the internal iliac arteries using a temporary agent, such as gelatin sponge particles (Fig. 39-9). There are at least three reasons for performing bilateral internal iliac artery embolization: (1) At the time of embolization, the patient is often in shock and profoundly vasoconstricted. This prevents IV contrast extravasation during angiography and offers a misleading impression of bleeding control. Once the patient is resuscitated and vasoconstriction is reversed, bleeding may ensue. Blockage of all the branches of the internal iliac arteries prevents this problem. (2) The pelvic vascular network is so extensive that a bleeder may be fed from the contralateral

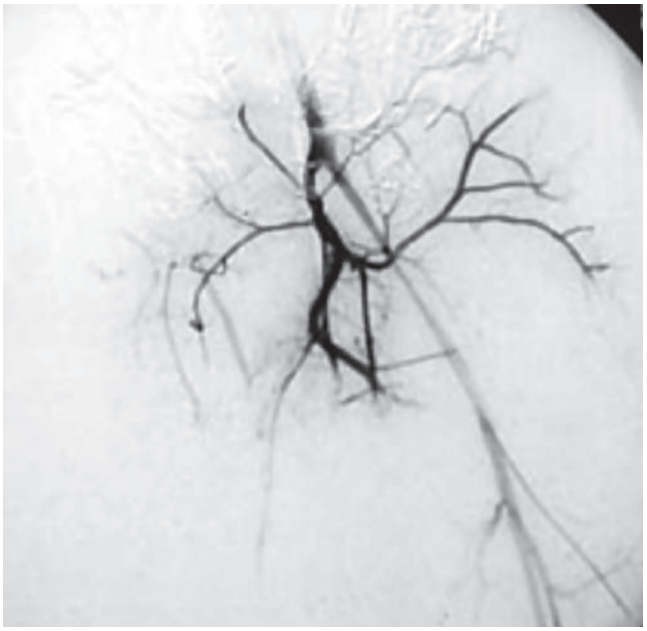


FIGURE 39-9 Truncation of all the branches of the internal iliac artery after injection of gelatin particles.

side. Embolizing only the unilateral internal iliac artery may not offer effective bleeding control (Fig. 39-10). (3) Some bleeders are right in the center, and it is difficult to discern if they are supplied by the right or the left arterial system.

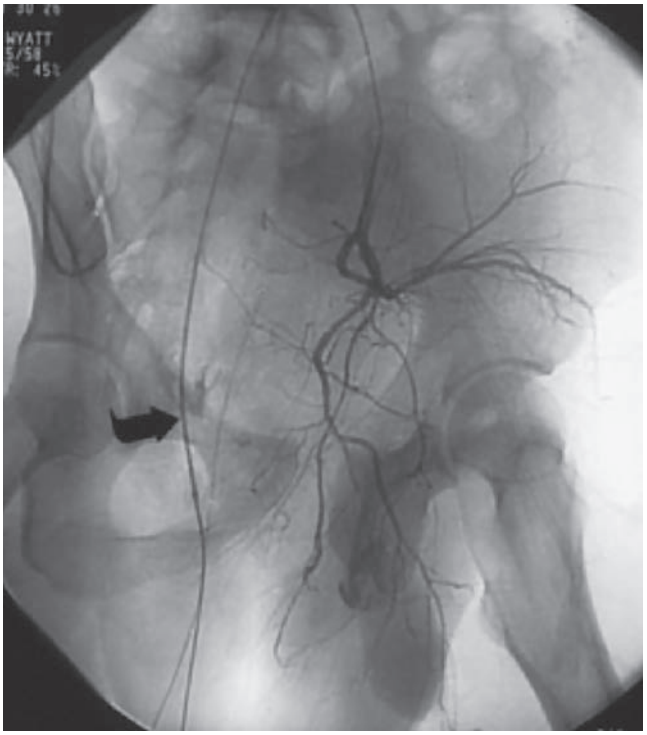


FIGURE 39-10 A right pelvic bleed is fed via the extensive pelvic network through the left arterial circulation. In such cases, bilateral embolization may be appropriate.

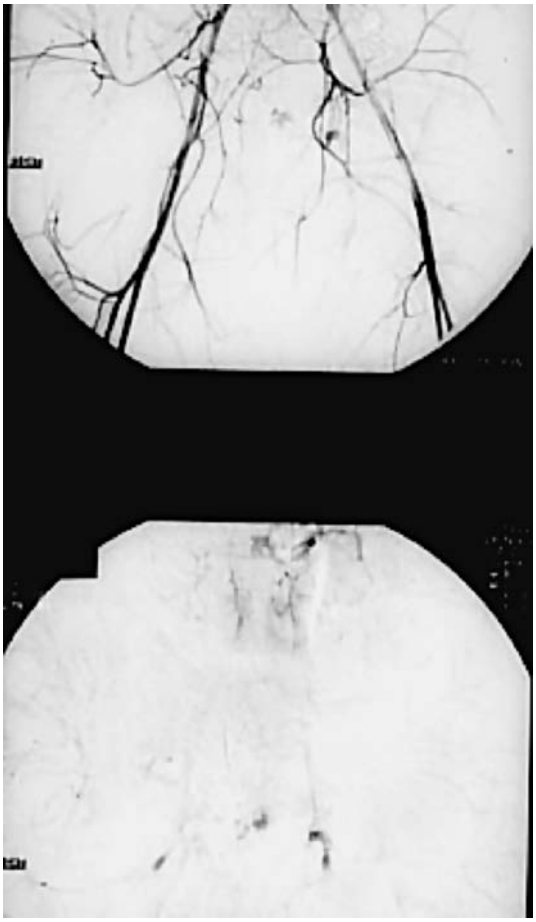


FIGURE 39-11 Midline bleeds can be hard to attribute to the left or the right circulation. Bilateral embolization may be appropriate.

Embolizing both sides is the more effective way to control the bleeding (Fig. 39-11).

The safety of bilateral internal iliac artery embolization has been shown in a study of 30 consecutive patients who received the procedure with no major complications.[32] It seems that the extensive vascular supply of the pelvis ensures survival of pelvic tissues and organs during the few days of Gelfoam embolization and until the arteries recanalize (Fig. 39-12). Gelfoam pledgets are usually cut to a size not smaller than 2 mm to prevent migration to smaller vessels and allow baseline collateral circulation. Despite isolated reports of serious complications with bilateral embolization,[33,34] such as colon necrosis, perineal wound sepsis, or avascular necrosis of the femoral head, our experience over the past 15 years has been very encouraging and without any significant complications. In a case-matched study of similar male pelvic fracture patients with and without bilateral iliac artery embolization, the incidence of sexual dysfunction 1 to 2 years after the injury was high but not different between the two groups.[35] Although major pelvic fractures affected sexual function, the addition of temporary embolization of both internal iliac arteries did not worsen the outcome. The authors assumed that, if this delicate function was not affected by embolization,

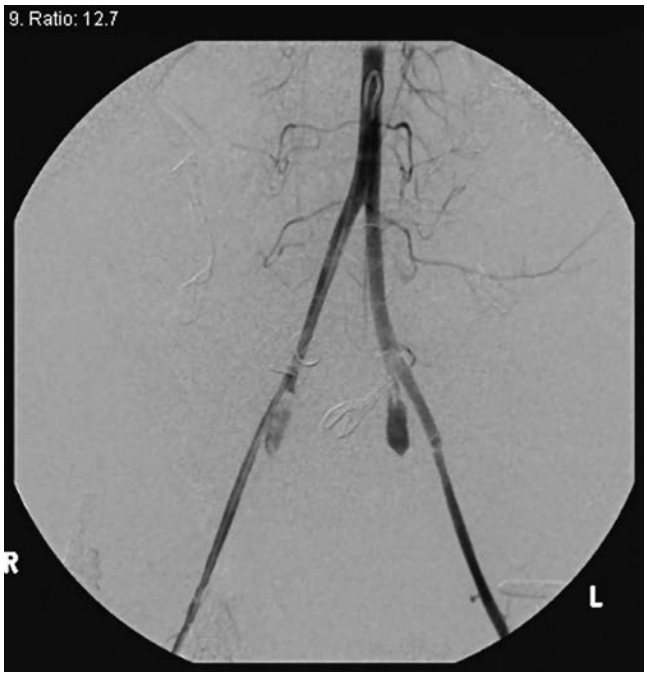


FIGURE 39-12 Heavy bilateral internal iliac artery embolization with near complete (but temporary) interruption of the pelvic circulation. The patient did well.

it was unlikely that any other pelvic organ would suffer major long-term consequences.

Failure of embolization occurs in approximately 15% of patients and is typically associated with coagulopathy.[36,37] In such patients, the thrombogenic potential of the injected gelatin material may not be fully realized and vessels may not be effectively blocked, showing near-full recanalization within only hours of seemingly effective initial embolization. Superselective embolization is associated with a higher risk of rebleeding. Patients who continue to require blood transfusions within 72 hours after embolization should be taken back to the angiography suite, because repeat embolization is typically successful.[24,38]

Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA) (See Chapter 13)

Central temporary aortic occlusion has been described for temporary control of exsanguinating truncal/pelvic bleeding.[39-42] This is done either via extraluminal aortic thoracic compression by resuscitative thoracotomy and aortic clamping or through a minimally invasive approach using a percutaneous endovascular balloon inserted through the femoral artery. The balloon is inflated in one of two zones: zone 1: descending aorta above the celiac artery; or zone 3: abdominal aorta between the lowest renal artery and aortic bifurcation. Zone 2 (abdominal aorta between the celiac artery and lowest renal artery) inflation is not used. The zone 1 surface landmark is the xiphoid process. The zone 3 surface landmark is the umbilicus. For exsanguinating pelvic injuries, the balloon is usually inflated in zone 3. There is no strong prospective evidence to support its efficacy. Retrospective studies have shown it to be effective in managing severe hemorrhagic shock from an intra-abdominal or pelvic source with a low complication rate, with the main barrier being expertise. The main complications are vascular events, acute renal failure, and rhabdomyolysis.

Preperitoneal Pelvic Packing

Packing is an important part of damage control operations for severe abdominal injuries. Its utility in the setting of severe pelvic fractures became apparent when European trauma surgeons Ertel et al[43] from Switzerland showed excellent bleeding control after major pelvic fractures using a combination of packing and external fixation by C-clamp. It is not clear which of the two techniques was primarily responsible for the outcomes. Recently, pelvic packing was reintroduced in the United States by the Denver trauma group and has become a useful adjunct in the management of bleeding PRF patients.[44] Unlike intra-abdominal packing, the preperitoneal pelvic packing technique involves placing laparotomy sponges within the preperitoneal space, in order to effectively tamponade venous and bony sources of bleeding and reduce the available volume of the retroperitoneal space.[45]

A small suprapubic incision is made, the fascia opened, and recti muscles retracted laterally. If intra-abdominal hemorrhage is also suspected, care should be taken to keep the suprapubic incision separate from the midline laparotomy. A minimum of three packs are placed on either side of the bladder deep into the pelvis, while ensuring that the peritoneum is not violated. If the peritoneum is incised and the abdominal cavity entered, tamponade is released and packing is rendered ineffective. Furthermore, the open funnel that the pelvis presents does not allow for the packs to remain in place and exercise a hemostatic effect by compression; rather, they float free back toward the abdomen.

If a separate laparotomy is required, the incision should be limited to the upper margin of the pelvic hematoma (Fig. 39-13). The preperitoneal packing technique can be lifesaving in patients who are hemodynamically unstable and cannot tolerate transport to the angiography suite. In these patients, the pelvic hematoma is typically very large, and more than six packs may be needed to effectively compress all the bleeding sites. One needs to remember that the bleeding typically originates in the posterior pelvis, and therefore the packs should be placed deep into the pelvis and pressed against the sacrum. Given that the preperitoneal pelvis is not an open cavity, it is not an unusual pitfall to put the packs in a relatively superficial plane, failing to produce adequate compression against the bleeding vessels.

Furthermore, pelvic packing is effective in controlling hemorrhage from the vast venous plexus surrounding the pelvis, in situations where angiography may not be effective or

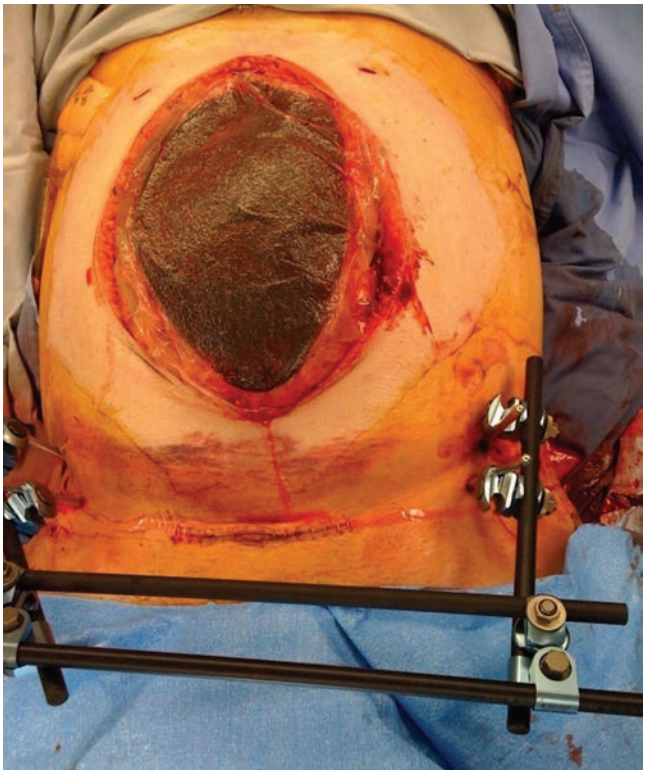


FIGURE 39-13 Note the Pfannenstiel incision, which is separate and distinct from the midline laparotomy, leading to the open abdomen. An external fixator is also placed to reduce the fracture. Very likely, such a patient will also receive angiographic embolization immediately following the operation.

may be unavailable. External fixation of the pelvis can also be performed at the time of pelvic packing.

In a study of 28 patients receiving preperitoneal pelvic packing by the Denver trauma group, 21 (75%) survived. Only 14% of the patients had postoperative angiographic embolization.[44] In a similar study by a Norwegian group, 13 (72%) of 18 patients with packing survived, but postoperative angiographic embolization was used in 80% of the patients.[46] In a follow-up study from Denver, 75 patients who received preperitoneal pelvic packing and external fixation were reviewed. Hemorrhage control was successful in most patients, and angiographic embolization was offered only in a minority who required additional interventions to control the bleeding.[47] One should consider these three procedures (preperitoneal pelvic packing, external fixation, and angiographic embolization) complementary rather than competing.[48] All of them can be offered on the same patient. While angiographic embolization is the preferred method of controlling pelvic bleeding for most patients, preperitoneal pelvic packing provides a useful management alternative in the following circumstances: (1) if there is no angiographic support, as may happen in non–Level I trauma centers; (2) if there is angiographic support but the team cannot assemble expeditiously; or (3) if there is profound hemodynamic instability, which makes even mild delays unacceptable and calls for rapid packing in a ready operating room. Following packing, angiographic embolization should still be strongly considered if the patient has ongoing transfusion requirements, and arrangements made accordingly for transfer to definitive care.

After resuscitation and correction of coagulopathy, packs should be ideally removed within 24 to 48 hours to minimize infection risk. Pelvic space infection can occur in up to 15% of patients. Risk factures include open pelvic fractures with associated bowel or bladder injuries, as well as multiple packing procedures.

DEFINITIVE FIXATION

Unstable and bleeding PRFs are usually managed by a staged repair, which includes a pelvic binder initially, external fixation shortly after that, and internal fixation as the final step to reconstruction. The timing and surgical approach for definitive internal fixation of the pelvis are patient specific and beyond the scope of this chapter. External frames via anterior external fixator or posterior pelvic C-clamps can be placed in the emergency department or operating room. Once placed, they do not interfere with intra-abdominal or preperitoneal packing access in the operating room and also allow operative access to the abdomen and pelvis and may serve as definitive therapy in certain clinical scenarios, particularly in anterior PRFs. They do not offer adequate stability in posterior fractures, particularly in the vertical axis. External fixation is appealing as a long-term solution because it avoids the risks of open operation, but the reduction needs to be precise. If a reduction to less than 1 cm of initial displacement is not maintained throughout the period of healing, then 80% of the patients require chronic analgesics compared with almost none with precise reduction.[49] The complications rise as time progresses, however, and pin infection develops in 50% of definitive fixators, as opposed to 13% of temporary fixators.[50] The most common reason for replacing an external fixator is aseptic pin loosening. Approximately 10% of patients require replacing external with internal fixation as definitive treatment. For APC-II injuries, a plate across the pubic symphysis may be enough, as the posterior pelvic stability is maintained by the unaffected strong posterior sacroiliac ligaments.

APC-III and most LC-II and vertical shear fractures typically require posterior stabilization by internal fixation. According to the specific type of fracture, sacroiliac screws (placed openly or percutaneously), special LC screws, or plates should be used. The complications of internal fixation are many and range from bleeding to injury of nerves, devascularization of muscle, infection, suboptimal reduction, and chronic pain.

ASSOCIATED INJURIES

Major pelvic fractures are high-energy injuries and are therefore commonly associated with intra- and extra-abdominal injuries. The mean Injury Severity Score (ISS) for patients

with pelvic fractures is 18.[51] Traumatic brain injuries, long-bone fractures, and thoracic injury are commonly associated with injuries to the pelvis. The incidences of associated solid and hollow intra-abdominal organ injuries have been reported as 11% and 4.5%, respectively, and diaphragmatic injuries occur in 2% of pelvic fractures.[52] All these associations point to the complexity of diagnosing all the injuries and choosing the correct therapy. A patient with a major pelvic fracture will usually have—at least lower—abdominal tenderness and hemodynamic fluctuations. The decision to explore or avoid the abdominal cavity is often hard as clinical examination, FAST, DPA, or even CT scan may give equivocal information. The presence of a central neurologic injury will only confuse the picture further. The suggested algorithm shown in Fig. 39-15 at the end of the chapter can serve as a guideline for the management of major pelvic fractures, although each patient is unique and requires individualized decisions.

The most commonly identified injuries directly associated with PRF include lower genitourinary tract injury, rectal injury, or iliac vessel injury. Bladder or urethral injury occurs in 6% to 10% of patients, which increases by nearly fivefold with male gender and severe fractures.[53] The majority of bladder ruptures (80%) are extraperitoneal and can be managed by simple Foley catheter drainage for 10 to 14 days. Intraperitoneal injuries require a laparotomy and direct repair. Straddle injuries, typically producing bilateral pubic rami fractures, are associated with urethral injuries. The presence of a perineal hematoma or blood at the urethral meatus should alert the clinician about a urethral injury. A retrograde urethrogram should be performed before inserting a Foley catheter. On selected occasions, in which time is of the essence, advancing a Foley catheter without first performing a retrograde urethrogram is an acceptable alternative. It should be done by a senior person and with extreme care to stop and withdraw in the presence of any resistance. If a Foley catheter cannot be inserted because of a urethral injury, a suprapubic catheter is appropriate. Urethral injuries are usually repaired at a later stage after the inflammation has subsided, but there are reports of successful aggressive early management. Obviously, these decisions will be made jointly by a urologist and the trauma surgeon.

The perineum should be examined for evidence of lacerations requiring repair. In females, a speculum-based vaginal exam should be performed to identify injury due to open fracture. Patients with a suspected rectal injury should undergo an exam under anesthesia in the operating room with sigmoidoscopy to exclude a rectal injury.

Finally, less common associated soft tissue injuries include closed internal degloving injuries termed Morel-Lavallee lesions. They represent a traumatic separation of the skin and subcutaneous tissue from the underlying fascia. Perforating lymphatics and blood vessels become disrupted, leading to seroma or hematoma formation in the subcutaneous tissues. The pressure of this fluid builds, and as the lesion matures, a fibrotic capsule forms and blood supply to the skin can become compromised, leading to skin necrosis. These lesions classically occur over the greater trochanter but may extend along the trunk and lower extremities. Treatment can include percutaneous aspiration if the hematoma is small, but recurrence rates are high. The Mayo Clinic published their recent series and developed practice management guidelines favoring operative incision and drainage. In some cases, formal debridement of skin and soft tissues is necessary, with vacuum-assisted closure and eventual skin grafting.[54]

OPEN PELVIC FRACTURES

A combination of open wounds with pelvic ring injuries produces an extremely challenging situation, as bleeding and ongoing contamination are typically profound and death rates are usually in excess of 20% and up to 50%. Lacerations of the perineum are much more difficult to manage than anterior lacerations. These two types of open pelvic fractures should not be described as the same entity because the management should be different according to the location and extent of the skin laceration.

The priorities in management of major open pelvic fractures are not much different than the management of any other devastating injury and include, in order of priority, control of bleeding, control of contamination, and definitive fixation. Control of bleeding in open pelvic fractures involves packing through the laceration, application of a pelvic binder, angiographic embolization, and external fixation. Preperitoneal pelvic packing, as noted earlier, may not be effective because the tamponade of the retroperitoneal pelvic space is already released to the external environment. There is a debate about the need for colostomy to control contamination. Many authors believe that a diverting colostomy should be routinely performed as an integral part of the surgical management of an open pelvic fracture (Fig. 39-14). In a study of 39 patients with open pelvic fractures, the mortality was 26% and predicted by fracture instability and rectal injury. The authors suggested that early colostomy is important to survival.[55] In another study of 44 patients, 23 were managed with a diverting colostomy and 21 without it. Even if the patients with colostomy were more severely injured, they had a lower 30-day mortality. Pelvic sepsis and anastomotic complications contributed to mortality in the no-colostomy group, and the authors recommended the liberal use of colostomy.[56] A systematic analysis of the literature, however, showed that there were no differences in outcome between patients with and those without a colostomy.[51] Nevertheless, the authors recognized that the evidence was of poor quality and that solid conclusions could not be made, and called for prospective randomized studies, a rather unlikely goal given the low incidence of these injuries. A midway solution was described on 18 patients, of whom 5 had a colostomy because of perineal wounds, whereas 13 with anterior wounds were not subjected to this procedure.[57] No patient without a colostomy developed pelvic sepsis, and the authors

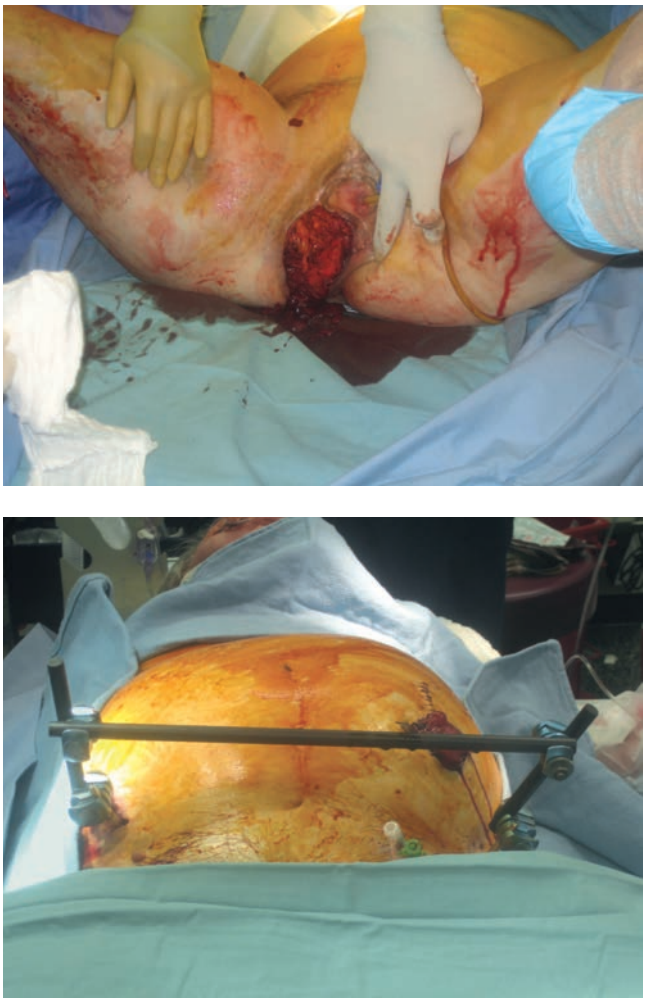


FIGURE 39-14 Colostomy is advisable for lacerations that are in immediate proximity or involve the perineum and perianal region. This patient received debridement and packing of the perineal wound, external fixation of the pelvic fracture, and a diverting colostomy.

recommended a selective approach. This is exactly our approach too. Patients with rectal wounds or with wounds in immediate proximity to the anus usually receive a diverting colostomy. Patients with more distal wounds are usually managed without fecal diversion. Debridement of ischemic tissue is an important part of the management. Despite all strategies, the mortality of open pelvic fractures remains very high, even in the modern era and even in expert hands. The Grady Memorial Hospital group described 44 such patients with a mortality of 45%.[58] The presence of bleeding and need for angiographic embolization were associated with a grim prognosis. Late pelvic sepsis developed in five patients, and three of them died. Therefore, it seems that primarily bleeding and secondarily sepsis continue to claim a heavy toll on the lives of patients with this devastating injury.

LONG-TERM OUTCOMES

Pelvic fractures, if severe, can be associated with significant morbidity and mortality. Mortality associated with severe pelvic fractures can reach up to 40%, depending on the degree of shock at presentation and identification of associated injuries.[59,60] In the early postinjury phase, patients with severe pelvic fractures requiring surgical fixation are at high risk for deep venous thromboembolism and pulmonary embolism.[61] Patients should receive prophylaxis (mechanical and pharmacologic) for deep vein thrombosis, or if contraindicated due to other injuries, a removable inferior vena cava filter should be considered.

Neurologic injury is also a characteristic complication after pelvic trauma with serious long-term implications. In an electrodiagnostic study of 78 patients with pelvic trauma and lower extremity neurologic symptoms, the incidence of gait instability and neuropathic pain was high.[62] As already discussed, sexual dysfunction remains a major problem in approximately two-thirds of male patients with major pelvic fractures.[35] Sensory impairments were noted in 91% of the patients with unstable sacral fractures 1 year after the injury.[58] Impaired gait was recorded in 63% and bladder, bowel, or sexual impairments in 59%. In a questionnaire study of 24 women with a Tile B or C pelvic fracture and a median age of 24 years, 16 reported de novo pelvic dysfunction.[63] Bladder symptoms were present in 12, bowel problems in 11, and sexual dysfunction in 7. Malunion of fractures can produce leg-length discrepancies, creating gait instability and pain. It is, therefore, obvious that even with optimal management, severe pelvic fractures are associated with long-term sequelae. Pain and neurologic impairments are the most common problems that can compromise the quality of life.

CONCLUSION

Major pelvic fractures are associated with significant bleeding, complications, and mortality. A multidisciplinary approach is important. The diagnosis of major pelvic bleed should be made in the trauma bay based on external clues of injury to the pelvis, physical examination indicating pelvic ring instability, pelvic x-ray, and focused abdominal sonography, as well as exclusion of other potential bleeding sources. CT scan is currently the most useful test to characterize fractures, detect hematomas and active contrast extravasation, and plan further treatment and operative approach. In the presence of bleeding, angiographic embolization is indicated and should be done in most cases along the principles of damage control angiography. Pelvic binding in the emergency department and external fixation are important interventions to reduce bleeding, pain, and ongoing injury. Internal fixation is best left for a later stage. Preperitoneal pelvic packing can be a life-saving maneuver for patients who are too unstable to travel to the angiography suite or in hospitals that do not have easy access to angiography. A general algorithm is provided (Fig. 39-15), but the exact sequence of interventions should be

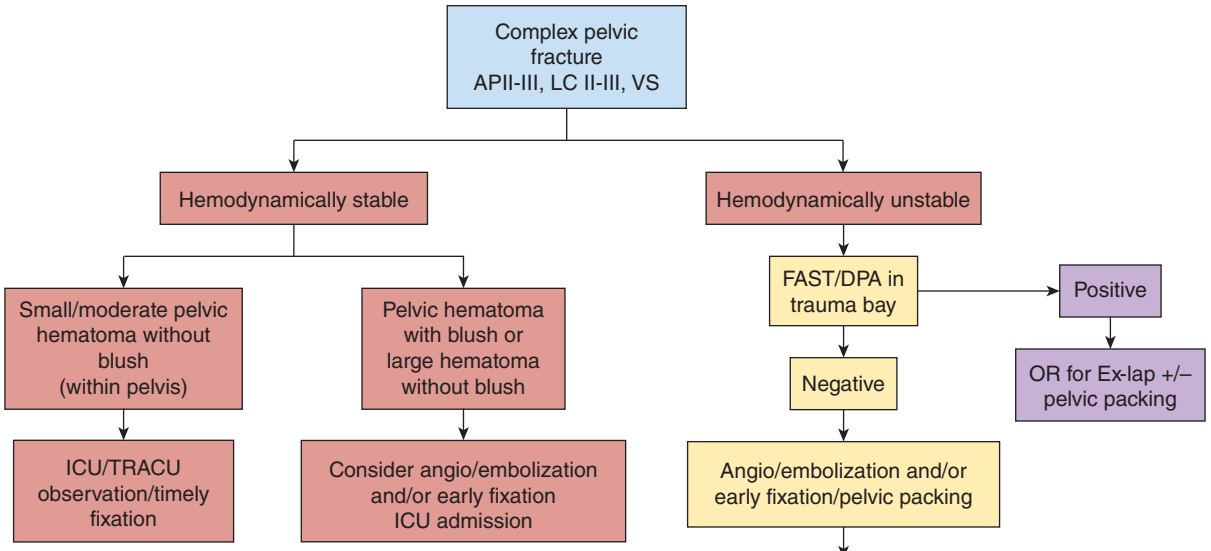


FIGURE 39-15 Algorithm for the management of major pelvic fractures. APC, anteroposterior compression; DPA, diagnostic peritoneal aspiration; FAST, focused abdominal sonography for trauma; ICU, intensive care unit; LC, lateral compression; OR, operating room; VS, vertical shear.

individualized to the particular complexities of these challenging patients.

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Genitourinary Tract

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KEY POINTS

- About 25% of kidneys receive accessory arterial branches directly from the aorta.
- Approximately 90% of significant renal injuries are due to blunt trauma in the United States.
- Injuries to the renal pedicle, including intimal disruption of the renal artery or renal devascularization, present with no hematuria in 20% to 33% of patients.
- Advantages of computed tomography (CT) over intravenous pyelogram (IVP) include identification of contusion and subcapsular hematoma, definition of the location and depth of parenchymal lacerations, more reliable demonstration of the extravasation of contrast, and identification of injuries to the pedicle and artery.
- Although hematuria is an important sign of ureteral injury, it may be absent 15% to 45% of the time.
- Indications for renal exploration include hemodynamic instability or ongoing hemorrhage presumably related to the kidney, pulsatile or expanding perirenal hematoma at laparotomy, and avulsion of the pedicle.
- Partial nephrectomy for polar lesions is performed using a "guillotine" technique with the transected vessels ligated and the collecting system closed.
- Ninety percent of grade V renal injuries require urgent nephrectomy.
- Delayed recognition of an injury to the ureter is managed with endoscopic or interventional radiology techniques due to local inflammation, edema, friability, and presence of a urinoma.
- Nonoperative management of an extraperitoneal injury to the bladder is with an indwelling catheter for 10 to 14 days followed by a cystogram.

INTRODUCTION

Genitourinary injury occurs in 2% to 5% of all trauma patients and in at least 10% of patients with abdominal trauma, emphasizing the need for a close collaboration between the general and urologic trauma surgeon. This unique relationship that the urologist and general trauma surgeon share in the management of urologic injuries requires common philosophies of management to be applied.

Controversies exist in the approach to urologic trauma, and recent efforts to achieve a broad consensus in the management of diverse urologic injuries have resulted in numerous publications. One such effort, sponsored by the World Health Organization and the Societe Internationale d'Urologie, involved a 25-year review of world literature focusing on levels of evidence and the development of evidence-based management recommendations.[1-4] Another effort through the European Association of Urology had a similar focus.[5] Both produced useful syntheses of a large body of literature. The current discussion will offer a broadly applicable approach to the management of urologic trauma based on current literature, local experience, and local perspective.

ANATOMY

The contemporary surgical approach to the injured kidney is through an anterior midline abdominal incision. Access to the kidneys and ureters is generally obtained by reflecting the colon medially on either side and exposing Gerota's fascial envelope. The exposure of an injured kidney may be achieved after obtaining vascular control of the renal vessels prior to entering the perirenal hematoma or by expeditious exploration of the retroperitoneum and manual renal control. Parenchymal compression is necessary in cases of active renal hemorrhage. The important step of either approach is to access the pedicle and apply atraumatic vascular clamping while damage is assessed and treated. Vascular control can be

accomplished through individual dissection and "looping" of the renal vessels through an incision in the posterior peritoneum over the aorta (which can allow access to either the left- or right-sided artery and the left-sided vein) or by first reflecting the colon on the side of the injury and then obtaining vascular control or access to the pedicle. This surgical step has been successful in experienced hands, but it may increase the exploration time. In cases of low suspicion of a renovascular injury and depending on the urologic trauma surgeons' comfort level, another successful approach to the kidney and renal hilum can be achieved by first reflecting the colon and then, by manual compression, the surgeon can achieve vascular control while the assistant can evaluate and apply an "en bloc" atraumatic vascular clamp if necessary.

The kidney is located high and posterior in the retroperitoneum. The midline incision may need to be extended to the xiphoid process, and additional upper abdominal retraction may need to be inserted for proper exposure. The kidney overlies the diaphragm, transversus abdominis aponeurosis, and quadratus lumborum muscle laterally and psoas major muscle medially. Significant bleeding from these muscles and the deep muscles of the back can occur following penetrating trauma and may be misinterpreted in the imaging as a kidney injury when it may be brisk bleeding occurring in the renal fossa. The kidney is enclosed in a thin but strong fibrous capsule, which should be left intact during renal dissection and mobilization. As the capsule is typically lifted off the parenchyma by an underlying hematoma, the entire capsule may inadvertently be stripped off the kidney by using a sweeping finger to quickly mobilize the kidney. Ideally, the kidney should be mobilized through sharp and blunt dissection working from a normal area toward the area of parenchymal injury to keep the capsule on the kidney. Stripping the capsule complicates the repair of the kidney and should be avoided.

Recognizing patterns of injury is important; the trauma surgeon should anticipate injuries to adjacent organs based on the relational anatomy of the kidney and ureter as well as the trajectory of a penetrating injury (Fig. 40-1).[6] The left kidney is crossed anteriorly in its upper portion by the tail of the pancreas and lies behind the lower portion of the spleen. On the right, the duodenum is immediately anterior to the hilar region. In the case of a renal injury on the right side, the right colon, liver, and duodenum are commonly injured in penetrating trauma. With blunt trauma, an associated hepatic laceration is most common. On the left side, injuries to the left colon, stomach, spleen, and pancreas are common in penetrating trauma, and lacerations of the spleen are particularly common with blunt trauma to the left upper quadrant. Injuries to the diaphragm are also common with penetrating renal injury and less common with blunt injury. The left adrenal gland is located medial to the upper pole of the left kidney, while the right adrenal gland is located in a more cephalad position relative to the right upper renal pole and may be in a retrocaval position.

At the level of the renal pedicle, there are commonly single renal arteries and veins present bilaterally. The renal vein, artery, and renal pelvis are organized in an anterior-to-posterior

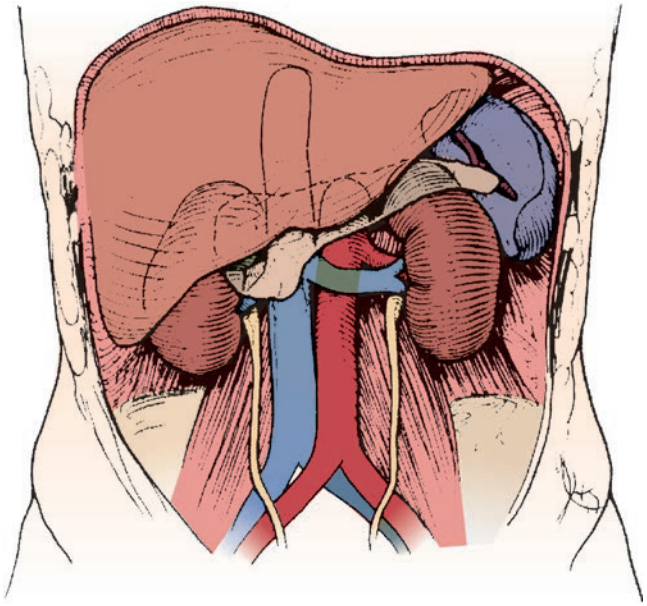


FIGURE 40-1 Renal anatomy: relational anatomy of the kidney. Note proximity of great vessels, duodenum, liver, spleen, pancreas, and colon, relevant to predicting patterns of injury and likely sites of concomitant organ injury in renal trauma.

orientation. On the right side, the gonadal vein arises from the vena cava at or slightly below the level of the renal pedicle. A lumbar vein, which may be quite large, often arises from the posterior aspect of the right renal vein near the connection with the inferior vena cava. The right adrenal vein enters directly into the vena cava, often on its posterolateral aspect. On the left, the main branches of the renal vein include the left gonadal, the adrenal, and one or more lumbar veins. This asymmetry of the collateral branches of the renal veins explains why the left renal vein can be safely ligated near the vena cava with an 85% chance of renal preservation. In contrast, the right kidney will most likely develop venous thrombosis and become nonviable if the right renal vein is ligated.

For the urologic trauma surgeon who engages in intrarenal surgery and renal reconstruction, knowledge of the intrarenal anatomy is important (Fig. 40-2). The renal arterial supply consists of the following five segments: apical, superior (anterosuperior), middle (anteroinferior), lower (inferior), and posterior. The posterior branch crosses cephalad to the renal pelvis to reach its segment. About 25% of kidneys receive accessory arterial branches directly from the aorta. These may enter through the renal sinus or at the upper or lower poles. Certain anomalies of the upper urinary tract, such as horseshoe kidney and congenital obstructive and duplicated systems, must be familiar to the trauma surgeon because they may impact management.

The blood supply to the ureter is particularly important in surgery for urologic trauma (Figs. 40-3 and 40-4). The main sources are the renal artery from above, the aorta or common iliac arteries, and the vesical arteries from below. Branches approach the upper and mid ureter primarily from the medial side, whereas in the lower pelvis, the blood supply

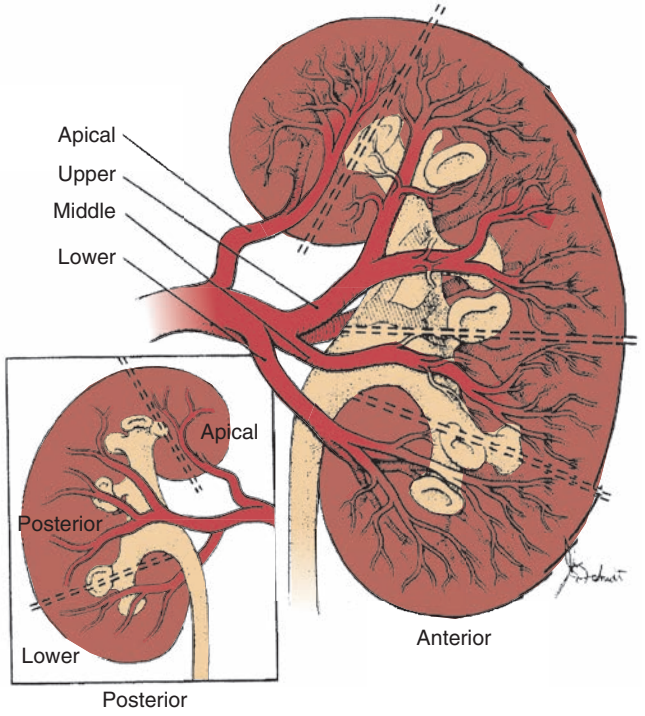


FIGURE 40-2 Intrarenal vascular anatomy: vascular branches supplying various arterial segments of the renal parenchyma. Knowledge of intrarenal anatomy is critical to successful reconstructive efforts.

to the ureter enters primarily from a lateral direction. These branches form a long, predictable anastomotic chain usually with a single longitudinal vessel that runs the length of the ureter in the plane between the ureteral adventitia and muscularis.

The anatomy of the urethra, perineum, and external genitalia may be less familiar to the general trauma surgeon. The gross anatomy and fascial layers of the genitalia and perineum are important in trauma because they largely determine the manner in which blood and urine extravasate following urethral or genital trauma (Fig. 40-5).

INJURY GRADING AND SCORING SYSTEMS FOR GENITOURINARY INJURIES

The American Association for the Surgery of Trauma (AAST) Injury Scaling Committee has devised a staging system for urologic injuries. The system, originally published in 1989 and since amended, addresses injuries to the kidney, ureter, bladder, urethra, testis, scrotum, and penis (Table 40-1).[7] For some organs, such as the kidney, the system has proven highly applicable and has come into common use. For other organs, such as bladder and ureter, the AAST system has been less commonly used for a variety of reasons, largely relating to lack of specificity of available imaging approaches to provide the necessary data for assignment of a grade. The grading systems for the urethra and external genitalia are becoming more

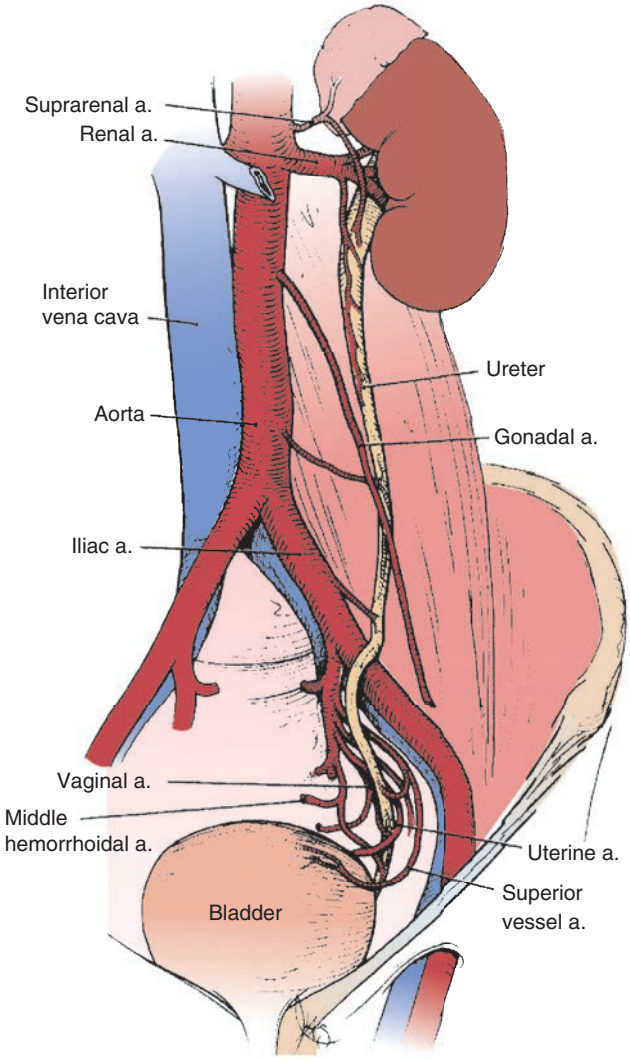


FIGURE 40-3 The ureteral blood supply originates from branches of the adrenal and renal arteries in the upper third, branches of the aorta and gonadal arteries in the middle third, and the pelvic vessels as shown in the lower third. Knowledge of the ureteral blood supply and derangements due to preexisting pathology or prior surgery is important in maintaining ureteral viability during surgical mobilization and reconstruction.

commonly used and are of value when addressing outcomes following such injuries. Several aspects of the staging system have received attention regarding their clinical significance and impact on decision making, complication rates, and patient outcomes.[8,9]

As noted in Table 40-1, the renal Organ Injury Scale uses five grades of injury, ranging from contusion or subcapsular hematoma (grade I) to shattered kidney or avulsion of the hilum (grade V) (Fig. 40-6). It is valuable to specifically distinguish the parenchymal lacerations from renovascular trauma in the group IV and V injuries when reporting experience because management and outcomes differ between these entities. The varying degrees of renal injury as described in the scaling system are depicted diagrammatically in Fig. 40-6.

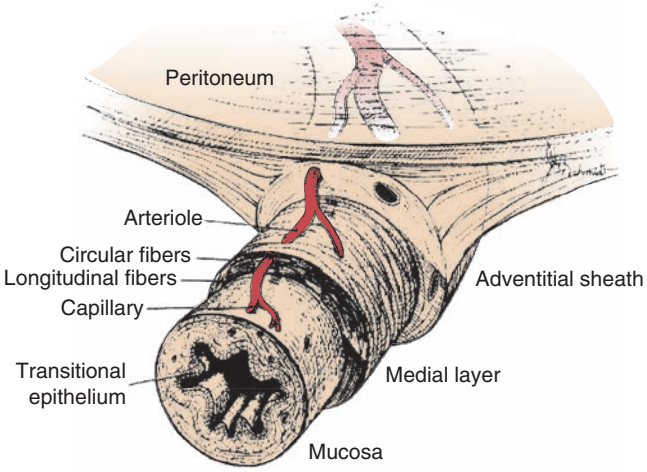


FIGURE 40-4 Ureteral anatomy: the longitudinal blood vessels run deep to the adventitial sheath; it is important to achieve a dissection plane superficial to this layer to avoid devascularization of the ureter during surgical mobilization.

Recent data have shown support for the clinical utility and validity of the renal injury scale, indicating that this system is predictive of morbidity in blunt and penetrating renal injury, of mortality in blunt injury,[9] and of the risk of nephrectomy with exploration for renal trauma.

The determination of ureteral injury type and classification can be challenging without surgical exploration, intraluminal endoscopic view, or use of radiopaque intraluminal contrast. Other indirect signs, such as the presence of ipsilateral hydronephrosis, can determine ureteral injury but will not determine the percentage of the ureteral circumference damaged. For the bladder, the distinction of intraperitoneal from extraperitoneal rupture is important and is addressed in the scaling system, but whether the length of the laceration in the bladder wall truly has clinical significance has not been demonstrated.

For urethral injuries, the scaling system addresses anatomic factors that can often be determined from retrograde urethrography (RUG) and provide advantages over the earlier system described by Colapinto and McCallum.[10] The current AAST system addresses urethral disruption based on whether the injury is complete or incomplete (eg, whether contrast enters the bladder), the length of the urethral defect, and the presence of extension into the prostate or vagina. Endoscopic assessment indicates cases where the RUG may suggest a complete disruption; however, partial circumference continuity may exist, which may allow for insertion of a catheter into the bladder. Despite some lack of specificity (anterior vs posterior injury), the AAST organ injury scaling system has substantial usefulness.

The scaling system for organ-specific injuries as applied to genitourinary trauma (Tables 19–22 and 29–31 from AAST website) has introduced a much-needed advance in the field.[7] The designations of the AAST system provide a universal language among clinicians to describe injuries and enact the right protocol and management of genitourinary trauma among different specialties.

CLINICAL PRESENTATION AND DIAGNOSIS OF RENAL TRAUMA

Incidence and Patterns of Injury

Renal injuries occur in approximately 1% to 3% of all trauma patients and up to 10% of patients with abdominal trauma. The percentage of blunt and penetrating trauma varies dramatically depending on the health care institution and the population served. In some urban trauma centers, penetrating

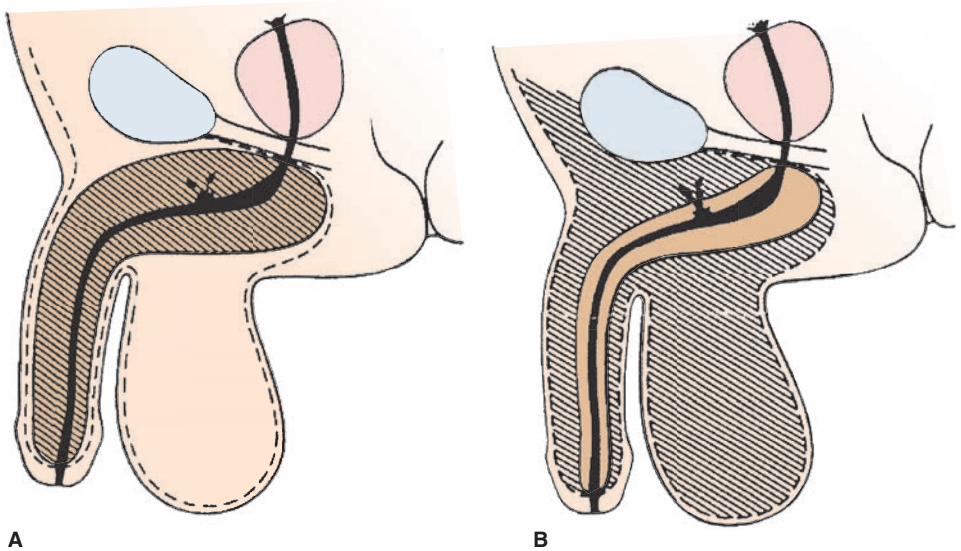


FIGURE 40-5 Diagram of sites of extravasation associated with urethral disruption. (**A**) With an intact Buck's fascia, extravasation of blood and/or urine is isolated to the penile shaft. (**B**) With Buck's facial defect, extravasation extends into the scrotal tissues and compartments.

TABLE 40-1: Urologic Injury Scale of the American Association for the Surgery of Trauma

Grade[a]		Injury description[b]
Renal injury scale		
I	Contusion	Microscopic or gross hematuria; urologic studies normal
	Hematoma	Subcapsular, nonexpanding without parenchymal laceration
II	Hematoma	Nonexpanding perirenal hematoma confined to the renal retroperitoneum
	Laceration	<1 cm parenchymal depth of renal cortex without urinary extravasation
III	Laceration	>1 cm parenchymal depth of renal cortex without collecting system rupture or urinary extravasation
IV	Laceration	Parenchymal laceration extending through the renal cortex, medulla, and collecting system
	Vascular	Main renal artery or vein injury with contained hemorrhage
V	Laceration	Completely shattered kidney
	Vascular	Avulsion of renal hilum that devascularizes kidney
Ureter injury scale		
I	Hematoma	Contusion of hematoma without devascularization
II	Laceration	≤50% transection
III	Laceration	>50% transection
IV	Laceration	Complete transection with 2 cm devascularization
V	Laceration	Avulsion of renal hilum that devascularizes kidney
Bladder injury scale		
I	Hematoma	Contusion, intramural hematoma
	Laceration	Partial thickness
II	Laceration	Extraperitoneal bladder wall laceration ≤2 cm
III	Laceration	Extraperitoneal (>2 cm) or intraperitoneal (≤2 cm) bladder wall lacerations
IV	Laceration	Intraperitoneal bladder wall laceration >2 cm
V	Laceration	Intraperitoneal or extraperitoneal bladder wall laceration extending into the bladder neck or ureteral orifice (trigone)
Urethral injury scale		
I	Contusion	Blood at urethral meatus; urethrography normal
II	Stretch injury	Elongation of urethra without extravasation on urethrography
III	Partial disruption	Extravasation of urethrographic contrast medium at injury site, with contrast visualized in the bladder
IV	Complete disruption	Extravasation of urethrographic contrast medium at injury site without visualization in the bladder; <2 cm of urethral separation
V	Complete disruption	Complete transection with >2 cm urethral separation, or extension into the prostate or vagina

[a]Advance one grade for multiple injuries to the same organ.
[b]Based on most accurate assessment at autopsy, laparotomy, or radiologic study.

Source: Reproduced with permission from Moore EE, Shackford SR, Pachter HL, et al. Organ injury scaling: spleen, liver, and kidney. *J Trauma*. 1989;29:1664.

injuries predominate.[6,11-13] Overall, approximately 90% of significant renal injuries in the United States are due to blunt trauma.[14]

For penetrating trauma, nearly all renal gunshot wounds are associated with injuries to other intra-abdominal organs; for renal stab wounds, approximately 60% of cases occur in combination with another intra-abdominal injury.

Interestingly, minor mechanisms of blunt abdominal trauma may cause significant renal injury and hematuria in kidneys with preexisting anatomic abnormalities (renal cysts, ureteral pelvic junction obstruction with hydronephrosis, renal neoplasm).[15,16]

Clinical Presentation and Evaluation

Any history of blunt and/or penetrating trauma to the chest, abdomen, or pelvis may increase the probability of a renal injury.

The physical examination of patients at risk for renal injury should include careful assessment of the abdomen, back, flank, and chest as well as a complete genitourinary examination. Findings suggestive of a renal injury include tenderness in the flank, costovertebral angle, or abdomen; palpable flank mass; or ecchymosis in the flank, back, or abdomen. Complete inspection of the torso for a penetrating injury is critical. Stab wounds posterior to the anterior axillary line carry a risk of renal injury, with only about 12% of such injuries being associated with an injury to another organ. Hematuria is the most common sign of renal trauma, although the magnitude of the hematuria correlates poorly with the magnitude of injury.[15,16]

Laboratory assessment should include urinalysis by dipstick, as well as a microscopic examination for blood or infection in the urine. The first specimen assessed in the emergency department should be analyzed for hematuria to optimize diagnostic accuracy. The determination of serum

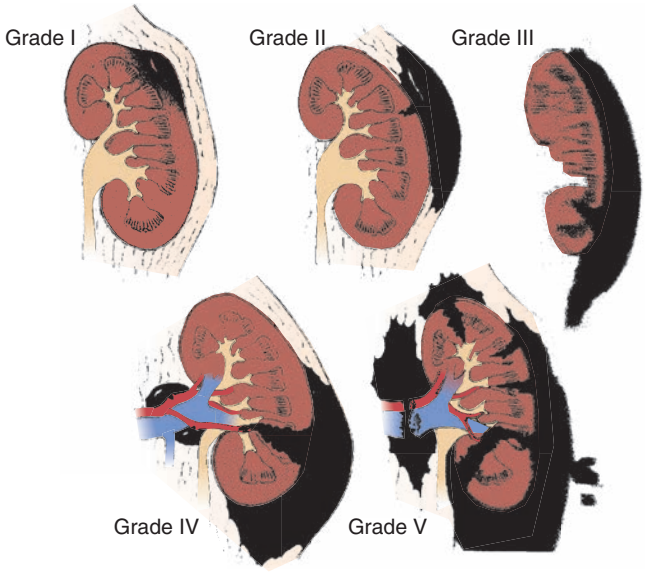


FIGURE 40-6 Organ injury scaling system for renal trauma.

electrolytes, blood urea nitrogen, serum creatinine, lactate level, and hemoglobin is important. A blood sample should be obtained to screen, determine blood type, and cross-match when clinically appropriate.

Radiographic Imaging for Renal Trauma

Traditionally, all patients with abdominal trauma and any degree of hematuria were imaged in the emergency department on presentation, yielding minor injuries in 90% of imaged patients without requiring intensive monitoring or intervention. Therefore, in order to be cost-effective and minimize potential morbidity of unnecessary imaging, more selective approaches to renal imaging in the trauma setting have been proposed without increasing the risk of missed injuries and delay in diagnosis.[17] In 1985, a group from San Francisco General Hospital analyzed their renal trauma experience and identified that the only findings that were predictive of significant renal injury were the presence of penetrating trauma, blunt trauma with gross hematuria, or blunt trauma with microhematuria and shock. Shock was defined as a systolic blood pressure less than 90 mm Hg at any time after injury, including during transport by emergency medical services. In a review of 812 patients with microhematuria without shock, no significant renal injuries were detected. All 44 injuries in this original series were found among the 195 patients with gross hematuria or microhematuria and shock. This series has been extended over the years, and in the expanded patient group of 2254 patients with renal trauma, it was found that approximately one-third were imaged and two-thirds were not. Within this group, no major renal injuries were missed when using the established criteria.[18-20]

Other investigators have modified imaging criteria according to their own experience and judgment. Some have suggested including standard imaging for patients with injury to the brain, loss of consciousness, or altered mental status, with the belief that the loss of information on a physical examination and the magnitude of trauma in such patients may create a higher risk of a missed injury. Some have suggested extending imaging indications to patients with mechanisms of injury consistent with deceleration trauma. This approach avoids missing injuries to the renal pedicle (eg, intimal disruption in the renal artery and renal devascularization), which may present with no hematuria in 20% to 33% of patients. The presence of fractures of long bones, lower ribs, or transverse spinous processes has also been suggested as an indication to modify the previous imaging restrictions, possibly predicting a higher risk of occult renal injury. In the pediatric population (addressed later in the section "Pediatric Renal Trauma"), imaging for patients with only microhematuria has been more liberally used.

Conversely, patients with penetrating trauma with any degree of hematuria, injury proximity, or suspicion are appropriate candidates for imaging of the urinary tract, regardless of the presence or magnitude of hematuria. Significant penetrating injuries can present without hematuria, particularly if trauma to the major collecting system causes all urine from the injured kidney to exit into the retroperitoneum, preventing ureteral peristalsis.

In penetrating trauma, imaging would generally be obtained while assessing a patient's candidacy for nonoperative management in the appropriate clinical setting. The concept of obtaining preoperative renal imaging solely to demonstrate the presence of two functioning renal units prior to surgical intervention has become less popular in recent years. Instead, careful intraoperative palpation of the kidneys and, on occasion, intraoperative intravenous pyelogram (IVP) may be used selectively during a trauma laparotomy to demonstrate renal presence or function.[21] The selection of imaging modalities has evolved greatly since the advent and availability of computed tomography (CT) scanning in emergency center evaluation.[17] Although bolus IVP with nephrotomography was previously the standard imaging approach, the CT scan has, over the years, become the gold standard for precise staging of renal injuries (Fig. 40-7) and has largely replaced IVP in most clinical settings.

Although the IVP was considered an accurate tool for clinical staging purposes in 60% to 85% of patients, CT scanning also offers a number of important advantages.[22] Nevertheless, trauma surgeons and urologists should maintain familiarity with the findings suggestive of renal injury on IVP because the use of CT for trauma assessment is not consistently available, especially when considering variations in international practice and infrastructure. These IVP findings that indicate renal trauma include the presence of a transverse process fracture on the scout film, presence of a mass effect in soft tissue, loss of the psoas margin on the involved side, and alteration of the longitudinal axis or vertical displacement of the kidney. Loss of a clear renal cortical outline, gross extravasation of contrast, ipsilateral decrease in renal excretory function, and loss of opacification in portions of the collecting

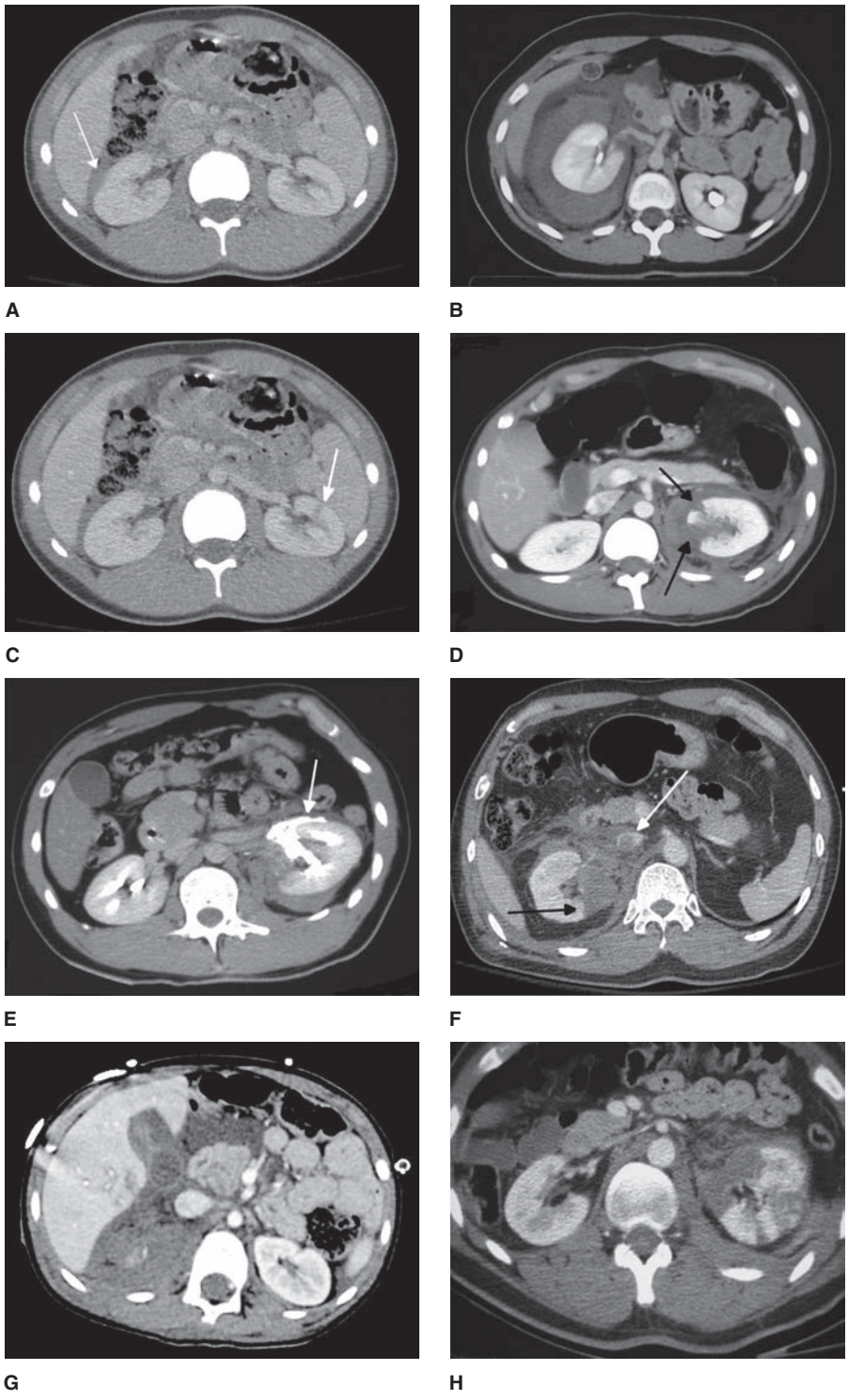


FIGURE 40-7 Staging computed tomography scans for renal injury. (**A**) Grade I hematoma (white arrow indicates right renal subcapsular hematoma); (**B**) grade II hematoma; (**C**) grade II laceration (white arrow indicates left renal parenchymal <1 cm laceration); (**D**) grade III laceration without urinary vascularization (black arrows indicate left renal parenchymal >1 cm laceration); (**E**) grade IV laceration with extravasation (white arrow indicates left renal parenchymal/collecting system laceration); (**F**) grade IV vascular with vena cava thrombus (white arrow indicates inferior vena cava thrombus and black arrow indicates right renal vascular compromise); (**G**) grade V shattered right kidney; (**H**) grade V vascular (left renal artery injury).

system should all be noted. The IVP allows confirmation of the presence of two renal units, gives general information of the extent of injury, and may show significant extravasation.

Estimates of the accuracy of IVP in detection of renal injury vary. In general, the IVP should be viewed as a crude means of detection rather than as a process to obtain precise staging. Some studies indicate that as many as 20% of patients with significant renal injuries may have a normal IVP. Renal arterial occlusion may not always be the cause of reduced function or nonfunction of a kidney on IVP. In up to 50% of patients, other factors, including contusion, hypotension, or hypoperfusion, may lead to a reduced function/nonfunctional kidney when viewed through IVP.

Advantages of CT over IVP include identification of contusion and subcapsular hematoma, definition of the location and depth of parenchymal lacerations, more reliable demonstration of extravasation of contrast, and identification of injuries to the pedicle and artery (eg, "rim sign," "cutoff sign") with or without three-dimensional reconstruction. There is also enhanced imaging of the perinephric space and other solid viscera (liver, spleen, pancreas), delineation of many cases of hollow viscus perforation, and identification of free intraperitoneal fluid. For these and other reasons, the contrast-enhanced CT scan has largely replaced IVP for trauma imaging. One should be extremely cautious to obtain correct CT scan sequences and obtain delayed, excretory images to avoid missing extravasation from the collecting system or ureter that may not be apparent from early nephrogenic and vascular phases.[23]

Often, interventional radiographic studies are obtained when vascular injury is diagnosed after CT scan and treatment is needed. Precise delineation of arterial anatomy, interventions to control hemorrhaging, or placement of a vascular Palmaz stent mandates the continued use of renal arteriography on a selective basis (Fig. 40-8). In several places, including the United States, the Focused Assessment for Sonography in Trauma (FAST) study is performed to assess for free intra-abdominal fluid rather than for the delineation

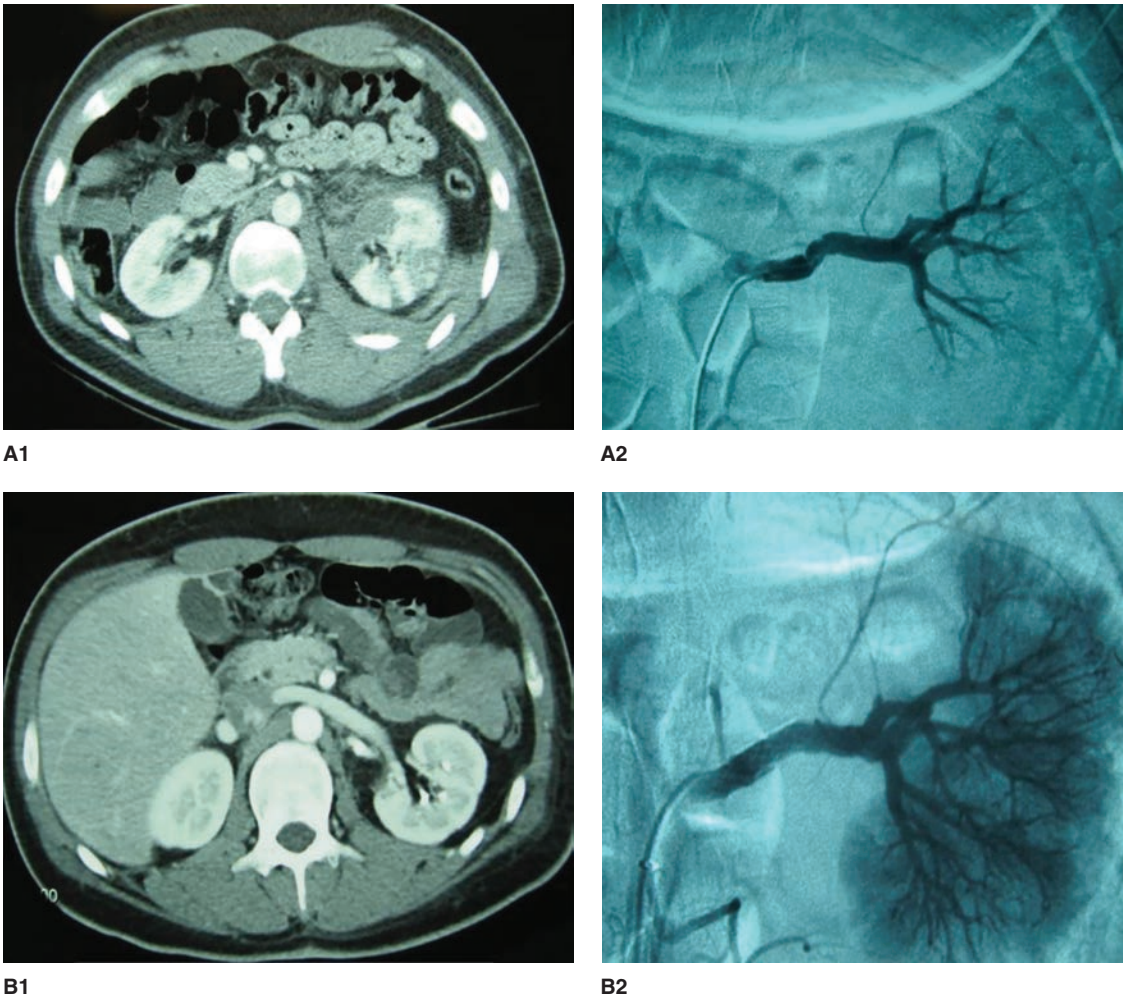


FIGURE 40-8 Left renal artery occlusion due to intimal disruption following injury (computed tomography [CT] and angiogram views of the same patient before and after vascular stent placement). (**A1**) Grade IV left renal vascular injury (CT scan vascular phase). (**A2**) Grade IV left renal vascular injury (angiogram). (**B1**) CT vascular phase/30 days after vascular stent placement. (**B2**) Vascular stent placement angiogram.

of an injury to parenchyma of solid organs. The evolvement of high-resolution ultrasound and Doppler techniques for the assessment of renal perfusion and vascular anatomy can be used intra- and perioperatively in cases of renal trauma.[24]

Currently, retrograde ureteropyelogram plays a limited role in defining renal injury but can be performed to evaluate and treat concomitant ureteral injuries when ureteral stents may decrease urinary extravasation and/or assist the passage of blood clots from the upper collecting system.

PEDIATRIC RENAL TRAUMA

The pediatric kidney may be more vulnerable to trauma than the adult kidney due to the relatively larger size of the kidneys in relation to the body, the relative lack of perinephric fat in the child, and the association of renal injuries with congenital renal abnormalities in pediatric patients. One recent review found that 8.3% of pediatric renal injuries occurred in patients with preexisting renal abnormalities,[15] with other estimates of preexisting renal abnormality described in up to 23% of major pediatric renal injuries due to blunt trauma. Some data suggest that the kidney is the most commonly injured intra-abdominal organ in children.

It is commonly agreed that the presence of gross hematuria after trauma in the pediatric patient warrants further investigation with imaging of the urinary tract. As in the adult, the CT scan plays a major role in staging such injuries for the same reasons as described earlier. Several studies suggest that only about 5% of pediatric patients with major renal injuries will develop signs of shock, further emphasizing the importance of an aggressive diagnostic approach. Pediatric patients can maintain normal blood pressure despite significant blood loss, and persistent tachycardia is a particularly important parameter to note in the pediatric patient as a potential sign of significant blood loss.

Many authors suggest that all pediatric patients with any degree of hematuria after significant trauma should undergo renal imaging, whereas some have suggested modified criteria. One study has suggested that microscopic hematuria with greater than 50 red blood cells per high-power field in the pediatric setting should be considered an imaging criterion, regardless of hemodynamic parameters.[25]

Certain types of renal injuries are more common in the pediatric patient. These include laceration of the renal pelvis, avulsion of the ureteropelvic junction, and forniceal avulsion. When extensive medial extravasation is noted and/or the ureter does not opacify with contrast despite adequate excretion into the renal collecting system, a disruption of the major collecting system should be considered. In such cases, retrograde pyelography with ureteral stent placement may be necessary to define the anatomy, diagnosis, and treatment.

Much like in the adult, the use of the rapid spiral CT scanner can lead to a pitfall in diagnosis if a delayed sequence is not requested. Limiting the study to a nephrographic or early excretory phase may fail to demonstrate extravasation or asymmetrical opacification of the ureters that would be readily visible on later images.

Overall, approximately 85% of pediatric renal injuries from blunt trauma are minor (contusions, superficial parenchymal lacerations) and are managed with bed rest and observation. Pedicle injuries compose about 5% of renal injuries, whereas major parenchymal injuries occur in 10% to 15% of patients. As in the adult, it is these latter groups for which management is somewhat controversial; however, it is largely agreed among pediatric urologists that operative decisions are based mainly on hemodynamic status rather than imaging criteria. The potential for successful management of kidneys that appear severely injured in imaging studies is remarkable in the pediatric population, and a nonoperative approach is the norm. Surgical treatment is generally reserved for patients with ongoing bleeding or hemodynamic instability, for those who have clearly failed an attempt at nonoperative management, and for penetrating injuries.[26]

CLINICAL PRESENTATION AND DIAGNOSIS OF TRAUMA TO THE URETER, BLADDER, URETHRA, AND EXTERNAL GENITALIA

Ureter

Ureteral injuries are relatively uncommon, occurring in approximately 4% of patients with penetrating abdominal injuries and in less than 1% of those with blunt abdominal trauma. Concomitant visceral injury occurs in the majority of patients with ureteral injuries from penetrating trauma. Although hematuria is an important sign of ureteral injury, it may be absent 15% to 45% of the time. As such, a high index for suspicion of ureteral injury is critical.[27-29] In fact, the ureter is one of the most common sites of missed injury during laparotomy, with one recent report noting a missed injury rate of 11%.[13] Although direct visualization of the ureter is the mainstay of detection of ureteral injury at the time of laparotomy, imaging modalities useful for detection of ureteral trauma also include an IVP and contrast-enhanced CT scan with excretory phase.[16] Failure of the distal ureter to opacify or the presence of ipsilateral hydronephrosis on a CT scan should raise concern for a potential injury.[30,31] When noninvasive imaging fails to provide sufficient detail regarding ureteral anatomy or the specific nature of an injury, cystoscopy with retrograde pyelography and possible ureteral stent placement may be indicated.

Bladder

Sudden compression of the full bladder, shear forces, or a pelvic fracture may result in a blunt rupture, especially when the bladder is full. Rupture may be accompanied by lower abdominal pain, an inability to void, and suprapubic or perineal ecchymosis. The cardinal sign of injury to the bladder is gross hematuria, present in greater than 95% of cases, whereas only about 5% of patients will only have microscopic hematuria.[32] Over 80% of patients with a bladder rupture

have an associated pelvic fracture in health care centers with a high percentage of blunt trauma. An association of bladder rupture with disruption of the posterior urethra, along with the occurrence of a pelvic fracture, may occur in 10% to 20% of patients.[33,34] Overall, recent data indicate that genitourinary injury occurs in approximately 15% of pelvic fractures in the pediatric setting[15] and that the incidence of injury to a pelvic organ is fairly comparable between adult and pediatric patients.[35,36]

Voiding cystogram with postvoid film is the standard method for diagnosis of injury to the bladder (Fig. 40-9).[37] It is important that the bladder be adequately filled to avoid false-negative studies. For the adult bladder, the standard volume of filling is 300 to 400 mL of iodinated contrast (30% iodine commonly used), which is infused through the indwelling Foley catheter by gravity. Alternatively, the bladder can be filled by gravity to a point at which the patient describes a sense of bladder fullness. If the patient is unable to indicate that there is a sense of fullness, using a standard filling volume is a useful methodology. A vertically oriented abdominal filling film image, designed to show the entire abdomen, should be obtained. Postdrainage films are necessary to avoid false-negative cystograms in which extravasated contrast may be missed if located only anterior or posterior to the distended bladder on an anteroposterior film. Patterns of contrast extravasation have been described for intraperitoneal, extraperitoneal, and combined ruptures (Fig. 40-10). Hematuria of the bladder without contrast extravasation on

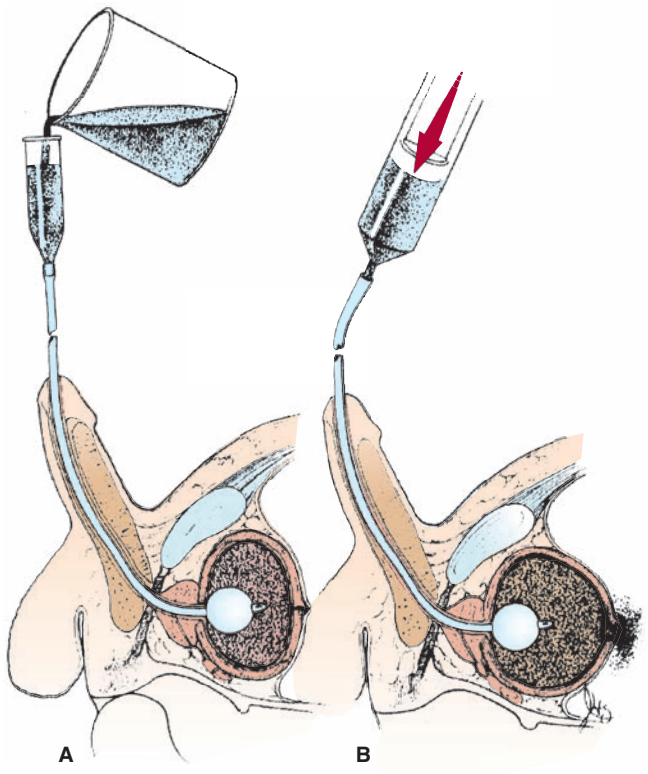


FIGURE 40-9 (**A** and **B**) Stress cystogram: through Foley catheter, the bladder is filled by gravity to a standard volume (300–400 mL typically in adult) or to the point of perceived fullness by patient. Plain radiograph is obtained to allow visualization of upper and lower abdomen, followed by washout film.

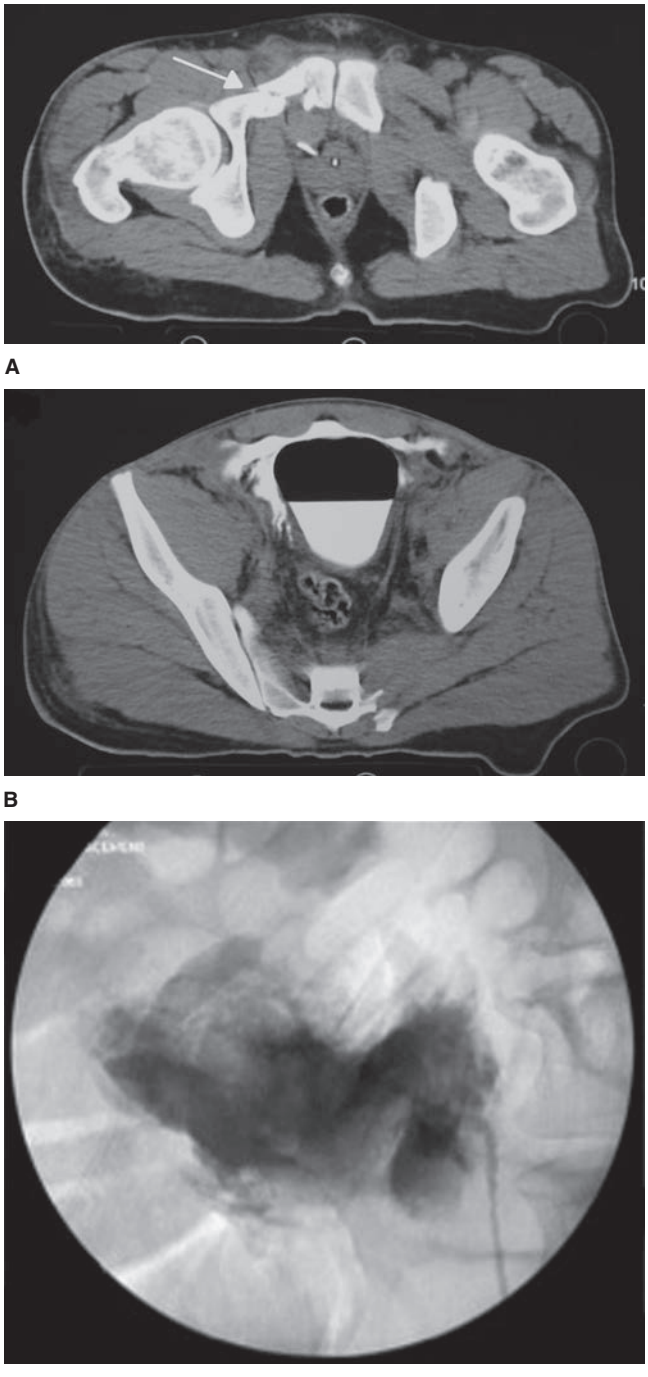
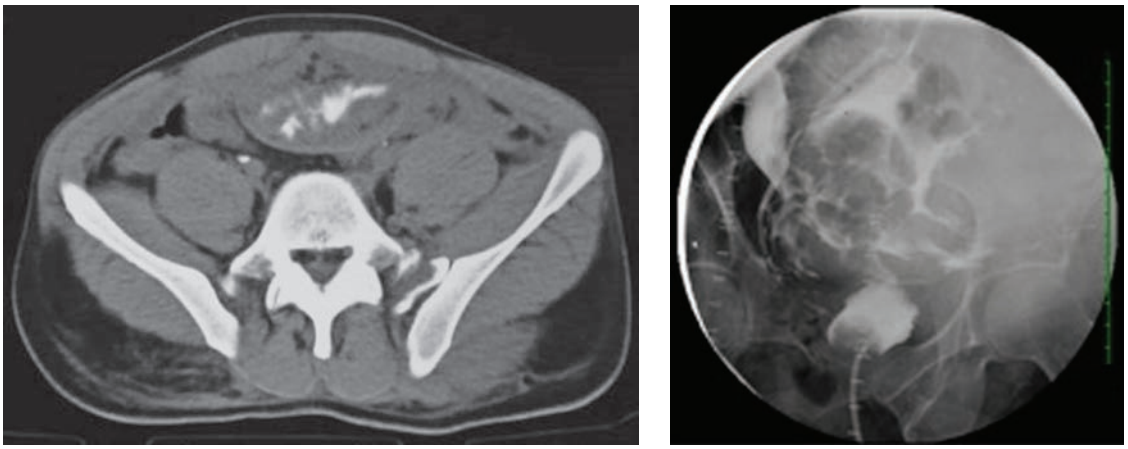


FIGURE 40-10 Bladder: stress cystograms for assessment of suspected bladder injury following blunt trauma to pelvis. (**A**) Pelvis computed tomography (CT) scan (white arrow indicates pelvic fracture). (**B**) CT cystogram reveals extraperitoneal bladder rupture with contrast extravasation confined to the retropubic space. (**C**) Voiding cystogram postdrainage film reveals extraperitoneal bladder rupture.



A B

FIGURE 40-11 (**A**) Computed tomography cystogram with intraperitoneal rupture (contrast delineating intraperitoneal small bowel). (**B**) Voiding cystourethrogram postdrainage film of intraperitoneal bladder rupture (contrast delineating intraperitoneal small bowel).

a properly performed voiding cystogram is consistent with a contusion or minimal mucosal injury, which is uniformly managed nonoperatively.

Currently, cystogram is most commonly obtained using a CT technique (Fig. 40-11A).[38] The advantages of CT cystograms are the speed to obtain the images, accuracy of extravasation detection, and the lack of need for voiding images (Fig. 40-11B). Studies comparing the accuracy of standard radiographic stress cystography versus CT cystography suggest equivalent capability in defining and staging bladder injuries. Simply clamping a bladder catheter following intravenous contrast administration, with the expectation that passive filling with contrast-opacified urine will suffice, is not adequate and will result in an unacceptably high percentage of false-negative examinations, with either the standard radiographic or the CT technique.[38] In selected cases, flexible cystoscopy may aid in the acute diagnosis of bladder injury and placement of a urinary Foley catheter.[39]

Urethra

Trauma to the anterior urethra may result from straddle injuries with sudden compression at the level of the mid urethra to deep bulbar urethra against the inferior pubic arch. Urethral distraction injuries or posterior urethral disruption may accompany pelvic fracture in 4% to 10% of patients. Bilateral fractures of the pubic rami, especially when accompanied by an open pelvic ring (abnormally distracted sacroiliac joint), may also be present in patients who have suffered posterior urethral disruption. The classification system used to further describe urethral trauma is discussed in the earlier section "Injury Grading and Scoring Systems for Genitourinary Injuries." It is important to determine from the urethrogram if an injury is partial (contrast passes proximal to the point of extravasation, filling the more proximal urethra or bladder) or complete (all contrast extravasates and none enters the urethra proximal to injury or bladder) because this factor has an impact on selection of management.[40]

Blood appearing at the urethral meatus, inability to void, presence of a perineal hematoma, and inability to clearly palpate the prostate on rectal examination should make one suspicious of urethral injury (Fig. 40-12). When urethral injury is suspected, a RUG should be performed (Fig. 40-13). Approximately 30 mL of iodinated contrast is instilled via a catheter inserted just within the urethral meatus, and then a plain radiograph is obtained. A normal RUG should demonstrate contrast filling an intact urethra and entering the bladder without extravasation. No attempt at insertion of a bladder catheter should be pursued until a negative RUG is obtained to avoid further complicating a urethral rupture (Fig. 40-14).

Following placement of either a urethral catheter (if the urethra proved normal or by a urologist using direct vision techniques in selected incomplete injuries) or a suprapubic catheter (if urethral disruption was revealed), a stress cystogram should still be performed if hematuria is present. This is because 10% to 15% of patients with urethral disruptions from a pelvic fracture will have a concomitant injury to the bladder.

External Genitalia

Genital injuries represent a diverse group of traumatic events.[41] These include the classic blunt penile fracture (which occurs from forceful bending of the erect penis, often during intercourse), crushing injuries with rupture of the testis, penetrating injuries, and industrial accidents. Amputation injuries of the penis or testicle can occur due to assaults, self-mutilation, or industrial trauma. After major blunt trauma to the scrotum, the risk of testicular rupture is approximately 50%. An ultrasound examination of the scrotum may be valuable to distinguish testicular rupture from a hematoma

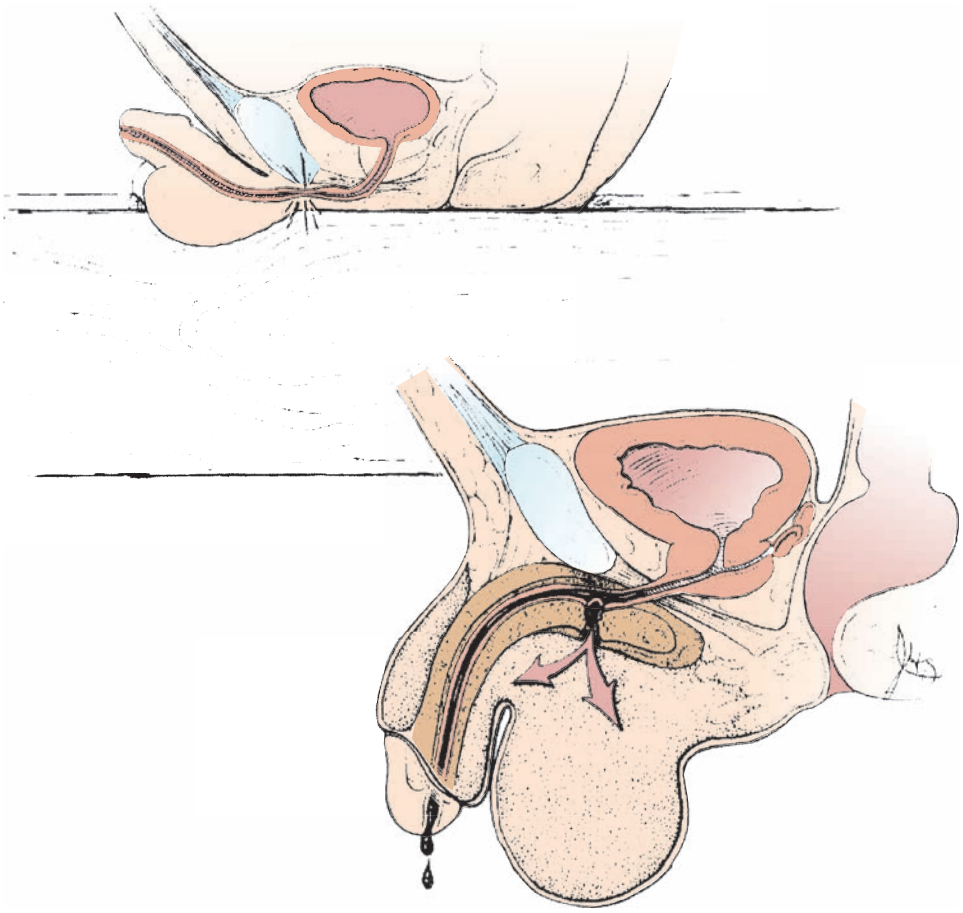


FIGURE 40-12 Mechanism of anterior urethral disruption due to straddle injury; extravasation pattern and hematoma limited in this case by Colles' fascia, due to rupture of Buck's fascia along with full thickness of urethral wall. Hematoma and urinoma may extend along shaft of penis and into scrotum and perineum.

of the scrotal wall or hematocele (blood within the tunica vaginalis compartment).

NONOPERATIVE MANAGEMENT OF GENITOURINARY INJURIES

Although nonoperative management for many urologic injuries has become well established, the selection of operative versus nonoperative management for certain genitourinary injuries remains controversial. Recent reviews of urologic management based on careful assessment of levels of evidence reveal a notable paucity of level 1 prospective management studies.[1-5] The relatively recent efforts to accurately and uniformly describe and stage the nature of injuries and the lack of long-term follow-up leave many questions regarding the best way to manage many forms of genitourinary trauma.

Kidney

It has long been accepted that low-grade renal injuries can be managed nonoperatively with a high success rate. Renal contusion and subcapsular hematomas are routinely managed expectantly and only rarely would require surgical or other interventions in such cases. These injuries heal spontaneously with few exceptions, as do low-grade parenchymal lacerations. Depending on the institutional bias and experience, some urologic trauma surgeons may limit operative management of renal injuries to those in which the patient is hemodynamically unstable, almost regardless of imaging findings. Alternatively, others would include high-grade injuries, presumably translating into a higher incidence of postinjury complications with nonoperative management. A number of indications for renal exploration following injury have been suggested by McAninch and Carroll.[42] These include hemodynamic instability, an ongoing hemorrhage requiring significant transfusion, pulsatile or expanding hematoma upon exploration, and avulsion of the pedicle. These strong indications for surgical or other procedural intervention remain widely accepted. Relative indications for surgical intervention have included high-grade injuries, large perirenal hematoma, presence of urinary extravasation on contrast studies, significant devitalized fragments of parenchyma, and findings in the operating room during laparotomy with an incompletely

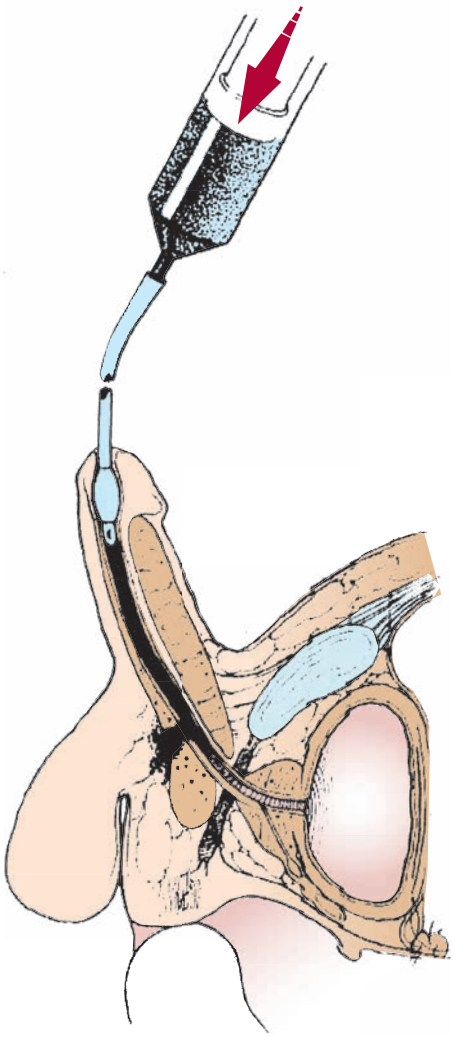
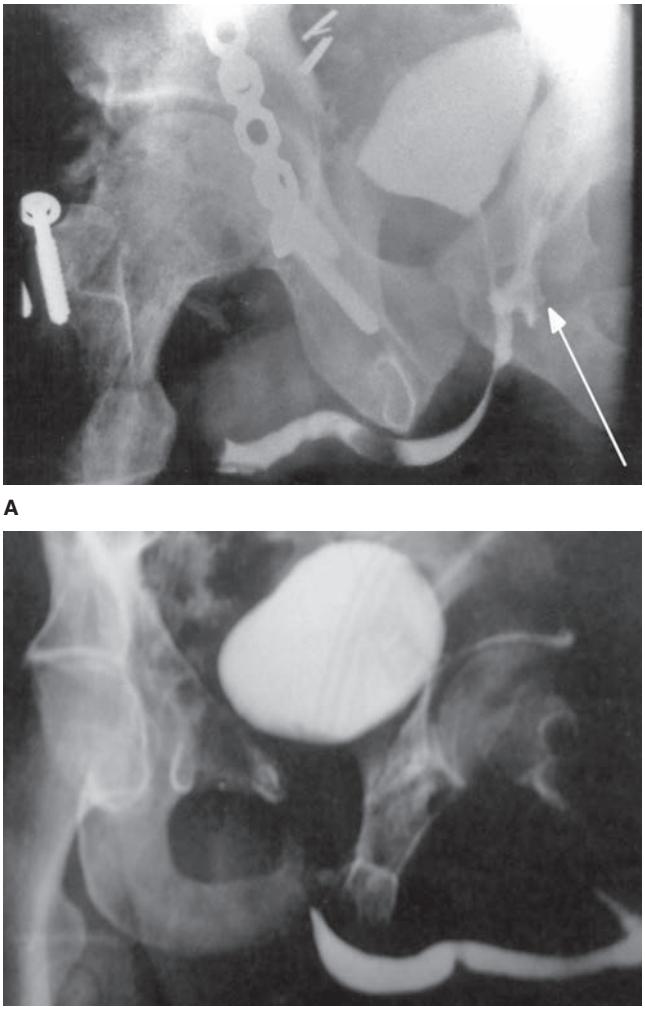


FIGURE 40-13 Technique of retrograde urethrogram. Retrograde urethrogram: catheter is inserted into urethral meatus, with minimal balloon inflation to maintain position and allow hands to be out of x-ray field. Contrast is instilled to distend urethra.



A

B

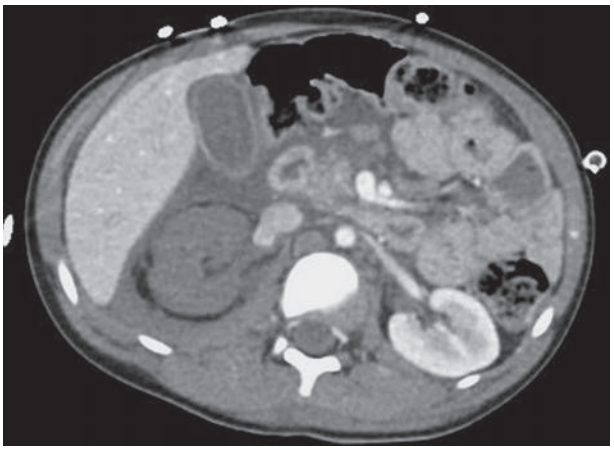
FIGURE 40-14 Urethra: posterior urethral disruption with pelvic fracture. (**A**) Retrograde urethrogram with incomplete posterior urethral injury (white arrow indicates contrast extravasation of posterior urethra injury). (**B**) Retrograde urethrogram with complete posterior urethral injury. Note the disconnected bladder, also known as "pie in the sky."

staged injury. Although there is lack of consensus regarding these relative surgical indications, there is a general trend toward nonoperative management in many of these situations as long as hemodynamic stability is maintained.[26]

Proponents of the nonoperative management approach suggest that many high-grade injuries will heal without surgery, complications can frequently be managed with nonsurgical techniques (percutaneous drainage, stenting, angiographic embolization), and renal salvage rates are better overall when renal exploration is avoided. This school of thought would maintain that, with few exceptions, it is only hemodynamic instability that should prompt surgical intervention for the injured kidney, not injury stage or other predetermined imaging criteria.

In contrast, proponents of a more aggressive surgical approach would suggest that higher grades of renal injury carry an unacceptably high complication rate and that such complications, when they occur, have a high likelihood of resulting in otherwise avoidable morbidity or nephrectomy (Fig. 40-15). Proponents would suggest that early exploration offers the advantages of early debridement of devitalized tissue, definitive hemostasis, repair of injuries to the collecting system, and early utilization of appropriate drainage. As such, postinjury infection, urinoma, and hemorrhage risk are minimized. The descriptions of "absolute" and "relative" indications for renal exploration of trauma have been suggested to attempt to provide assistance in this decision-making process.[42-44]

For certain injuries, operative management is commonly accepted. These include blunt avulsion or penetrating lesions of the renovascular pedicle, AAST grade V parenchymal injuries, and ureteropelvic avulsion or complete avulsion of the



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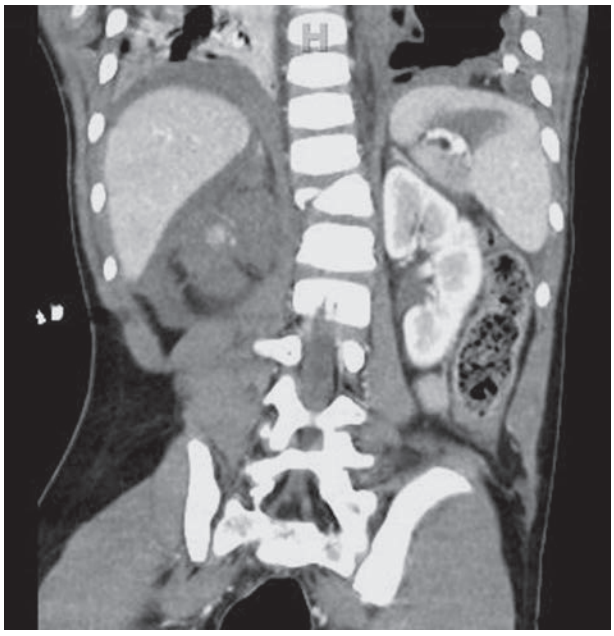

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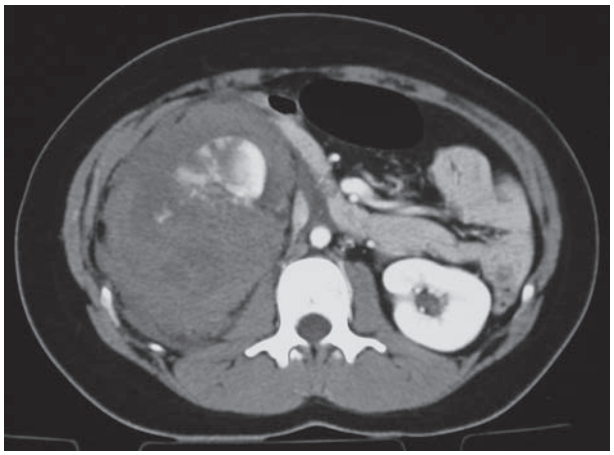

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FIGURE 40-15 Grade IV and V parenchymal injuries: (**A**) grade IV vascular; (**B**) grade IV right renal laceration and spinal fracture; and (**C**) grade V parenchymal laceration.

fornices. Although occasional case reports have suggested that grade V renal injuries can be managed nonoperatively, most studies demonstrate that 90% to 100% of such injuries require urgent nephrectomy.[45] In reviewing the literature on nonoperative management of grade V renal injuries, the accuracy of classification is questionable, and some reports of successful management of grade V injuries are likely describing grade IV parenchymal lacerations. In general, attempts at nonoperative management of true grade V renal injuries are not advised and may expose the patient to substantial risk, although some controversy remains in this area.[46]

Patients with significant ongoing bleeding from an injured kidney where angiographic control is not likely to correct the problem, is not available, or has failed will also require prompt operative attention. For penetrating renal injuries in cases where laparotomy will occur regardless, especially when preoperative radiologic staging has not been performed or is incomplete, operative management is widely recommended.

When moderate or high-grade renal injuries are selected for nonoperative management, certain general principles apply. Such patients are at risk for continued bleeding or significant delayed bleeding, and it is important that they be observed in the surgical intensive care unit. Serial abdominal examinations are essential, as are serial laboratory studies including hemoglobin level and electrolyte status. Typed and cross-matched blood should be available for the first 24 to 48 hours. The patient's hemoglobin should be maintained in such a range that a sudden drop from renewed bleeding would not be catastrophic. Particular attention should be paid to the size of the perirenal hematoma on initial imaging. Large hematomas suggest bleeding from larger intrarenal vessels and, presumably, indicate cases in which the risk of continued bleeding is greater. Elderly patients or patients with cardiovascular disease should be transfused more liberally, with a low threshold for intervention, as any sudden substantial blood loss may not be tolerated. When managing high-risk renal injuries nonoperatively, it is advisable to reimage such injuries at 48 to 96 hours to allow early diagnosis of complications such as enlargement of the perirenal hematoma, formation of a urinoma, or evolution of ischemic parenchyma. Early knowledge of such untoward events allows for treatment before the patient demonstrates complications such as sepsis, azotemia, or severe anemia.[47]

It is routine to impose a period of strict bed rest with nonoperative management of a major renal injury, although specific data to support this policy are lacking. Nevertheless, it seems reasonable to have the patient remain on bed rest for the first 24 to 72 hours or until significant gross hematuria resolves and then reinstitute ambulation cautiously in a monitored environment. If nonoperative management has been successful, patients should be instructed to avoid significant physical exertion until follow-up imaging reveals adequate healing.

In cases of emergent exploratory laparotomy and retroperitoneal bleeding, an intraoperative IVP with intravenous bolus injection of iodinated contrast (2 mL/kg body weight) and a 10-minute excretion film can offer better staging of

renal/ureteral injuries and presence of both kidneys. If significant anatomic distortion is observed, this is considered suggestive of major parenchymal disruption and/or injury to the collecting system for which exploration may be of benefit. If the kidney appears grossly intact, observation would be selected, often with postoperative CT scanning for more precise imaging. In general, current trends in the urologic literature favor nonoperative management of most blunt renal injuries in the absence of staged grade V lesions, active bleeding noted intraoperatively, or hemodynamic instability.

Injuries to branch renal arteries from blunt trauma, resulting in segmental devascularization without laceration, can be managed nonoperatively with a low complication rate and angiographic studies and vascular stent placement.

Penetrating injuries to the kidney are accompanied by injury to nonurologic organs in a large proportion of cases, and the majority of these patients will undergo laparotomy. These patients may or may not be imaged preoperatively. The issue of whether to explore the (suspected) renal injury in such cases is addressed in the later section "Operative Management of Specific Genitourinary Injuries." When the general trauma surgeon sees no clear operative indication and penetrating renal injury is possibly present, the urologist will have to decide on operative versus nonoperative management based on the clinical status of the patient and on the findings of a contrast-enhanced CT scan. In general, patients may be safely managed nonoperatively if they present with penetrating injuries (stab wound) to the kidney that involve the lateral and peripheral parenchyma with small perirenal hematomas, minimal if any extravasation of contrast, and in which the pedicle and renal sinus structures are not at risk (Fig. 40-16). Conversely, penetrating renal lesions that result in large perirenal hematomas; traverse the deep medial renal parenchyma, renal sinus, or hilar region; or cause major urinary or vascular extravasation carry higher risks for nonoperative management (Fig. 40-17). The risk of delayed bleeding from such injuries is significant, and some authors have suggested prophylactic arteriography with embolization of violated arterial branches prior to nonoperative management. In addition, the risk of a missed associated visceral injury must be considered with nonoperative management of penetrating renal trauma. In one retrospective review of the nonoperative management of penetrating renal trauma, 55% of renal stab wounds and 24% of renal gunshot wounds were managed nonoperatively with low complication rates.[48]

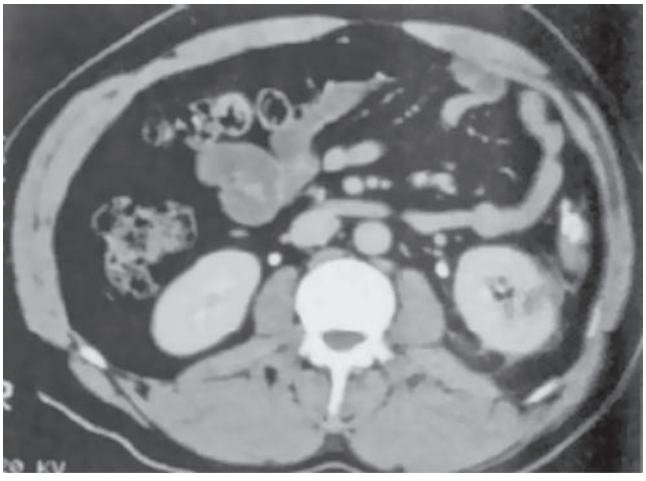


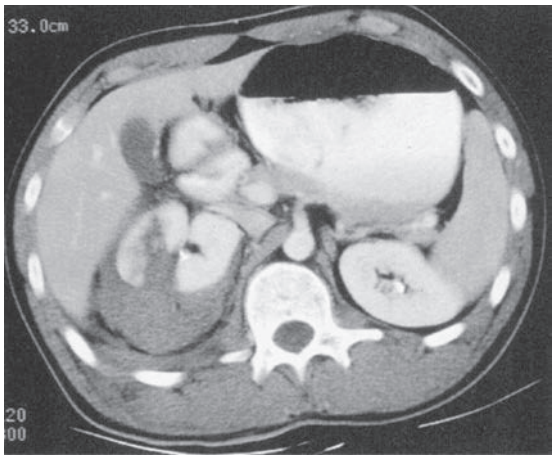
FIGURE 40-16 Penetrating renal injury, successful nonoperative management. Staging computed tomography scan demonstrating laceration to lateral left kidney. There is minimal perinephric hematoma, no urinary extravasation, and no devitalized parenchyma. Injury is lateral, and laceration does not extend into hilar region or renal sinus structures. Posterior descending colon is in proximity to injury, but general surgeons are prepared to manage nonoperatively. Ideal candidate for nonoperative management of a penetrating renal injury.

Although uncommon, blunt or penetrating trauma to the adrenal gland warrants brief mention. If an adrenal hematoma is not expansile, it is managed nonoperatively as with parenchymal injuries to other solid organs. If the adrenal gland is explored due to the path of a stab or bullet wound, suturing to achieve hemostasis and/or placement of biologic fibrin glue may prevent total adrenalectomy. Because each adrenal gland has several sources of arterial blood supply, devascularization from trauma is rare.

Ureter

Nonoperative management of ureteral trauma has limited applications. When a ureteral injury is recognized intraoperatively, surgical repair is favored.[49,50] Reviews of the outcomes of ureteral injuries indicate that most types of ureteral trauma fare better with early operative repair when compared to delayed repair or attempts at nonoperative management, with the exception of limited iatrogenic injuries from endoscopy. This is the case for stab and gunshot wounds, as well as avulsion injuries from blunt trauma (Fig. 40-18). Nonoperative management is performed in selected patients with missed ureteral injuries or other settings of delayed diagnosis or in patients in whom damage control strategies are being adopted. Traditional urologic teaching dictates that if ureteral trauma is recognized soon after injury, operative repair is performed. More significantly, delayed recognition is managed with utilization of endoscopic or interventional radiologic techniques (stenting or percutaneous nephrostomy diversion) followed by delayed operative reconstruction as indicated. This approach has developed due to the long-standing recognition of problems such as inflammation, edema, friability, presence of a urinoma, and increased risks and complications of reconstructive efforts encountered when operative intervention is pursued greater than 3 to 5 days after injury. Ureteral contusions recognized intraoperatively, due to either penetrating or blunt trauma, may be managed nonoperatively and observed; however, some reports suggest that the risk of late perforation and urinary extravasation may be reduced by intraoperative insertion of a ureteral stent.[51]

When nonoperative management is selected, retrograde ureteropyelography with attempted retrograde ureteral stent placement is often performed. Alternatively, percutaneous renal drainage may be the treatment of choice. The selection



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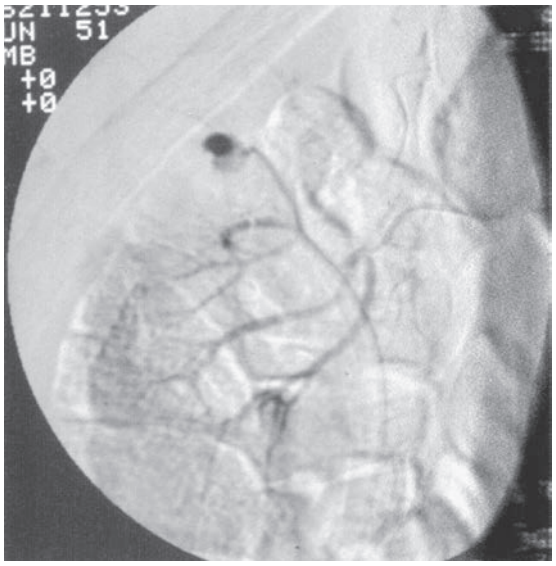

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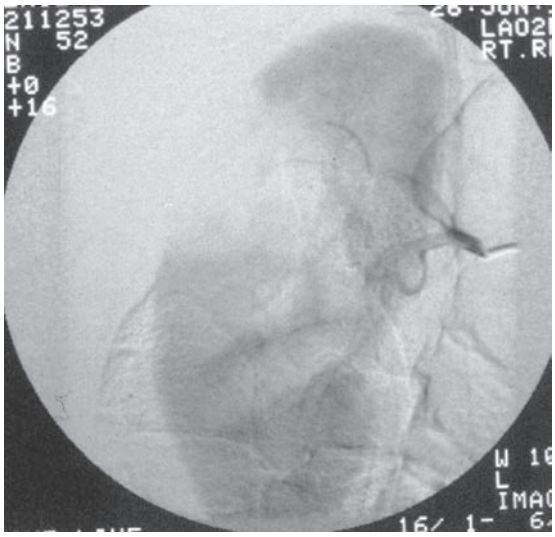

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FIGURE 40-17 Penetrating renal injury, complicated. (**A**) Staging computed tomography scan of abdomen following single stab wound to right posterior flank in patient presenting with gross hematuria. Deep laceration of right kidney with moderate-sized perinephric hematoma. Injury extends into renal sinus region, although no contrast extravasation is noted. After initial attempt at nonoperative management, patient develops major secondary hemorrhage manifested by profuse gross hematuria, resulting in hypotension, and requiring transfusion of 4 units of packed red blood cells. (**B**) Arteriogram reveals two areas of arteriocalyceal fistula, successfully managed with subselective embolization. (**C**) Delayed arteriogram image demonstrates wedge-shaped infarct defect due to embolization. Remainder of hospital course uneventful. Embolization is ideal means of managing this problem because the only indication for intervention is hemorrhage.

between these two approaches depends on the hemodynamic and metabolic stability of the patient as well as specific anatomic and logistical factors. These include the appropriateness of performing a procedure under general anesthesia, the ability of the patient to undergo a procedure in a prone position (generally necessary for obtaining percutaneous renal access), the skill and availability of interventional radiology, and the expected ease of percutaneous access. The latter depends largely on the anatomy and degree of distension of the collecting system and the presence of a perirenal hematoma. The finding of coagulopathy is often considered a relative contraindication to percutaneous renal drainage because renal bleeding is always a risk of such procedures. Achievement of percutaneous access can be followed by antegrade ureteral stenting if there is ureteral continuity and a guidewire can be placed across the injury into the bladder. Conversion from a nephrostomy tube to a percutaneous antegrade universal stent, which can be changed, manipulated, opened to external drainage, or

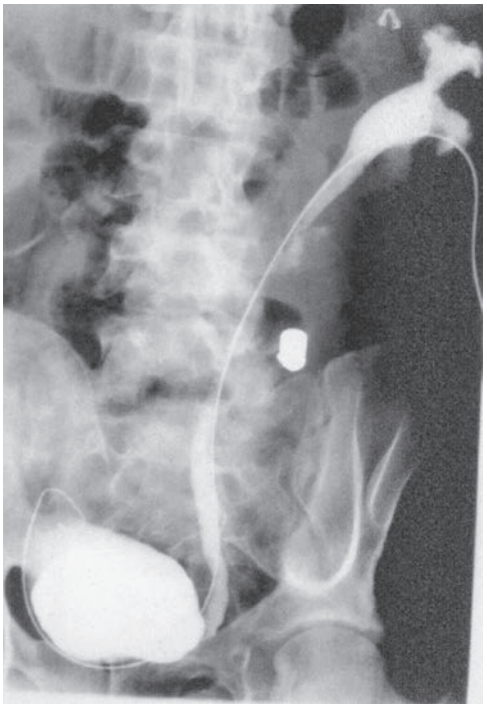


FIGURE 40-18 Ureter: gunshot wound to ureter with missed injury in a patient who had no hematuria on initial presentation. Patient developed abdominal fluid collection after laparotomy; intravenous pyelogram demonstrated missed ureteral injury 5 days postoperatively. Injury was initially managed with percutaneous nephrostomy and antegrade placement of universal stent. Long, densely fibrotic stricture of midureter developed, as shown here, ultimately requiring nephrectomy. A high index of suspicion is necessary to detect penetrating ureteral injuries at the time of initial laparotomy; outcomes are significantly improved with early recognition and prompt operative repair in such cases.

capped to allow internal drainage, may be attempted. Following an appropriate period, a pullback antegrade nephrostogram will determine if healing is complete and the patient is ready for stent removal with clamping of the nephrostomy tube.

When this type of management is used, ureteral strictures may be expected in approximately 50% of cases. A stricture may require an attempt at endourologic management, although delayed surgical reconstruction of the ureter is often necessary.

With blunt trauma, limited ureteral injuries with minimal urine extravasation may be treated with retrograde pyelography and retrograde ureteral stent placement. For penetrating injuries, small-gauge shotgun pellet wounds may create minute ureteral perforations that can be managed nonoperatively as well. Such injuries may be noted at laparotomy or may be seen on a contrast-enhanced CT or on intravenous or retrograde pyelography. Again, such cases represent the rare exception to the general principles that favor early operative exploration and repair when technically and medically feasible.

Bladder

Nonoperative management of extraperitoneal injury to the bladder has been the standard approach for over 10 years, largely as a result of the studies in which catheter drainage alone was consistently successful.[52,53] An 18F to 20F or larger bladder catheter should be used to allow free drainage for the adult. The catheter is left indwelling for 10 to 14 days followed by a cystogram to confirm cessation of extravasation prior to removal. After this period, more than 85% of bladder injuries will show absence of extravasation. If extravasation persists, another 7 to 10 days of catheter drainage followed by repeat cystography is appropriate. Rarely, persistent extravasation will occur after a prolonged period of catheter drainage. In such cases, CT scanning and/or cystoscopy is indicated to ensure that a foreign body such as a bony spicule from a pelvic fracture or some other anatomic cause is not resulting in failure of the laceration to properly heal. Indications for initial selection of operative management instead of catheter drainage alone include concomitant injury to the vagina or rectum, injury to the bladder neck in the female, avulsion of the bladder neck in any patient,[54] and the need for pelvic exploration for other surgical indications. If retropubic access is required for internal fixation of a pelvic fracture, surgical repair of the bladder is favored in order to prevent continued extravasation adjacent to orthopedic hardware. Open pelvic fractures may also require operative repair of the bladder. If catheter realignment is planned, the presence of combined extraperitoneal and intraperitoneal rupture or combined extraperitoneal bladder rupture and posterior urethral injury would be considered an appropriate situation to proceed with operative repair of the bladder as well. Finally, clot formation with troublesome occlusion of the drainage catheter may mandate operative repair.[55]

Intraperitoneal ruptures of the bladder are uniformly managed with operative repair. Such injuries typically result in large, stellate tears in the dome of the bladder due to the sudden rise in pressure within a full bladder as from a blow to the lower abdomen or compression by a seat belt. Rare exceptions to the routine application of operative repair for intraperitoneal bladder rupture include minimal intraperitoneal perforations. These usually occur during endoscopic procedures, mainly when a resectoscope is being used for resection of a bladder tumor or during biopsies of lesions of the dome and anterior wall, or other minimal iatrogenic injuries. Several reports have appeared in recent years describing laparoscopic techniques of repair for iatrogenic injuries.[56,57]

The application of laparoscopic repair techniques to manage intraperitoneal ruptures of the bladder from blunt trauma and other forms of bladder injury is being explored at several centers.

For penetrating injury to the bladder, nonoperative management is occasionally applicable in carefully selected extraperitoneal injuries.[58] Such patients may require proctoscopy and/or pelvic arteriography. Selectively, peritoneal lavage or laparoscopy may play a role in such cases to ensure that the peritoneal surface of the pelvis is intact. Cystoscopy and

upper tract imaging (IVP or retrograde pyelography) have been shown to be helpful in assuring that the magnitude of the defect in the bladder is minimal and is likely to heal with catheter drainage alone. The considerations for conversion to operative management, postinjury monitoring, postinjury imaging, and catheter management are comparable to those used with blunt extraperitoneal injuries.

Urethra

The nature (blunt or penetrating) and location of the injury (anterior vs posterior urethra), completeness (partial vs complete circumferential laceration), presence and severity of associated injuries, and the stability of the patient all impact the selection of management for urethral trauma.[59,60] When urethral trauma is suspected, RUG should be performed. If the RUG reveals minimal extravasation and flow of contrast past an anterior injury from blunt trauma into the proximal urethra and bladder, some authors have suggested that a single attempt at gentle passage of a bladder catheter should be performed. Other urologists believe that even minimal blind instrumentation of the injured urethra is ill advised, preferring an endoscopically guided approach. We believe that endoscopically guided instrumentation of the injured urethra is preferable to blind insertion of a catheter. The most conservative recommendation is to avoid any blind instrumentation of the injured urethra by the nonurologist. For incomplete anterior urethral injuries, urethral catheterization is a reasonable therapy. Early urethral realignment techniques for posterior urethral trauma fall within the realm of the experienced urologist and constitute operative procedure. Penetrating injuries to the anterior urethra are generally managed with operative exploration and repair.[61] Penetrating injuries to the posterior urethra may present complex challenges in management, may be complicated by adjacent rectal injuries or other intrapelvic or visceral injuries, and are also considered later.

Genital Injuries

Although penile fractures and testicular ruptures are best managed with early recognition as well as operative exploration and repair, certain genital injuries due to blunt trauma may be managed nonoperatively.[62] This would be the case when the injury is limited to the subcutaneous tissues, the tunica albuginea and urethra of the penis are intact, and the tunica albuginea of the testes is intact as well. For penile injuries, nonoperative management is appropriate for rupture of subcutaneous vessels, resulting in limited ecchymosis or a hematoma. Scrotal trauma may be managed nonoperatively when the testis is intact and there is a limited hematocele that is not bothersome to the patient. In most situations, however, significant genital trauma is best managed by operative exploration and repair. If physical findings are suspicious of a significant injury to deep tissue or the injury cannot be ruled out by imaging studies, operative exploration is prudent. This is because the outcomes of nonoperative management of such injuries like penile fracture or testicular rupture are poor as compared with the very high success rates of early operative repair.[63] Because the relative morbidity of surgical exploration of the external genitalia is minimal and the morbidity of missed injuries or delayed recognition is significant, one should err in the direction of operative management for such injuries.[41,64]

A scrotal ultrasound demonstrating heterogeneity of the testicular parenchyma is suggestive of testicular rupture, even if clear loss of continuity of the investing tunica albuginea cannot specifically be identified.[65] Certainly, if a clear defect in the continuity of the testicular tunic is noted on ultrasound, the diagnosis of testicular rupture should be suspected and operative repair undertaken. Patients with a significant hematocele (blood and/or clot within the tunica vaginalis compartment) with an intact testis may be observed, although they may often have a quicker recovery of activity and more rapid resolution of scrotal pain and swelling if this lesion is evacuated surgically. An intratesticular hematoma without testicular rupture is generally managed nonoperatively. At times, testicular ultrasound may demonstrate an abnormality in which a preexisting testicular lesion such as a germ cell neoplasm is suspected. Such may be the case when relatively minor trauma causes a significant intratesticular bleed or testicular rupture. When preexisting testicular pathology is suspected and nonoperative management is selected for the traumatic lesion, it is critical that the testis be reevaluated until the suspicious abnormality resolves or its continuing presence mandates further imaging and intervention.

For genital injuries involving significant loss of soft tissue or skin, nonoperative management may be appropriate as an initial approach, especially when more immediately life-threatening injuries demand priority. Wounds should be cleansed and a conservative approach should be adopted when determining whether to perform debridement of genital skin or soft tissues of marginal or questionable viability. Secondary operative management and delayed reconstruction with skin grafting or other tissue transfer techniques is often necessary when wounds are initially managed in this manner.[66]

OPERATIVE MANAGEMENT OF SPECIFIC GENITOURINARY INJURIES

Kidney

Renal exploration for trauma begins with prioritization of the injuries and determining that the initial operation is in fact the appropriate time to embark on the renal exploration (see the later section "Damage Control Principles in Genitourinary Trauma"). When contemplating exploration of an injured kidney in the absence of preoperative imaging, some assessment of the presence and normalcy of the contralateral kidney should be undertaken. Palpating the contralateral renal fossa for a grossly normal kidney is certainly appropriate and is often the only assessment necessary. In select cases, an intraoperative IVP may provide more precise information. This can be performed by administering 1 to 2 mL/kg of iodinated contrast intravenously and then obtaining a

10-minute excretion film. This can occur while other general surgical tasks are being accomplished to avoid wasting time. While an intraoperative IVP provides some additional reassurance that a functional contralateral kidney is present when exploring an injured kidney, it is generally advised to proceed with exploration of the injured kidney based on contralateral renal palpation alone.

If it is jointly determined by the urologist and the general surgeon that renal exploration should occur, exploration is carried out through an anterior vertical incision in Gerota's fascia. There has been some controversy regarding the importance of first obtaining vascular control of the renal pedicle prior to renal exploration, as previously described.[67,68] Some proponents claim a markedly reduced nephrectomy rate if the renal vessels are first dissected and controlled with vessel loops. Others claim that this maneuver is unnecessary for successful renal exploration and repair. This controversy is probably overstated, as even those who do not believe that individual dissection of the renal vessels is essential prior to renal mobilization tend to use some other approach to control the pedicle or limit renal bleeding during examination and repair of the kidney. The bulk of the literature would suggest that the rate of otherwise unnecessary nephrectomies is minimized by having exposure and control of the renal pedicle prior to renal exploration. This can be achieved by the traditional maneuver of incising the posterior peritoneum lateral to the aorta and individually dissecting and looping the renal vessels on the side of the injury (Fig. 40-19). This can also be achieved by reflecting the colon medially first and then clamping the pedicle if significant bleeding is encountered upon opening the Gerota's fascial envelope (Fig. 40-20). Alternatively, the pedicle or the renal parenchyma can be compressed digitally (most applicable to polar injuries) without having individual control over the renal vessels. Certainly, if there is an injury to the pedicle suggested by a large or expanding medial hematoma in the vicinity of the great vessels, there is broad agreement that central vascular control should be the initial maneuver.

Following pedicle control or access, the colon and mesocolon on the side of the injury are dissected medially following incision of the peritoneal reflection. When the anterior surface of Gerota's fascia is fully exposed, a generous vertical anterior incision is made through the fascia and the kidney is fully mobilized. As indicated earlier in the section "Anatomy," it is important to dissect in an extracapsular plane and avoid inadvertently dissecting the renal capsule away from the underlying cortex. Accomplishing this is facilitated by beginning the dissection in an area of intact parenchyma rather than directly within the laceration. Completely mobilizing the kidney is very helpful, as it allows the kidney to be lifted anteriorly into the wound for complete inspection. If significant bleeding results during this maneuver, a noncrushing vascular clamp is applied to the renal artery, renal vein, or entire renal pedicle. An initial decision must be made regarding renal salvageability and the magnitude of the reconstructive effort that would be required to repair the injury. This is based largely on the amount of devitalized parenchyma, the degree of injury to the central vasculature and central collecting system, and the condition of the patient. If the kidney is determined to be reconstructible in an unstable patient, any significant intrarenal vascular injury can be rapidly sutured and the kidney can be packed off with laparotomy pads as other surgical injuries are treated. After repair of other injuries or at the time of a secondary surgical procedure, a formal exploration and reconstruction of the kidney are performed.

If, based on the anatomy of the injury, the kidney is not considered reconstructible, a nephrectomy is performed. It is preferable to separately ligate the renal artery and vein to avoid the potential for arteriovenous fistula. A rapid search is made for accessory or polar vessels that must be ligated as well. Although urologists frequently suture or simply ligate the renal artery and a long stump of vein, vascular surgeons and some urologists prefer to oversew the short right renal vein with a continuous 3-0 or 4-0 Prolene suture. For trauma nephrectomies, the ureter and adjoining vessels are ligated near the kidney, whereas the gonadal vein is ligated and divided when necessary with no need for concern for adverse impact on the gonadal structures.

If renal reconstruction is planned, several steps are generally followed (Fig. 40-21). Following evacuation of the hematoma, the kidney is carefully examined to identify lacerated vessels, the open collecting system, and devitalized parenchyma. Large areas of lacerated, devitalized parenchyma are excised sharply, whereas smaller vessels are controlled with an absorbable 3-0 or 4-0 suture. In general, an absorbable suture is used for intrarenal suturing, as a permanent suture may create a nidus for stone formation if in contact with the collecting system. If adequate closure of the collecting system is achieved, there is no need for stenting or a nephrostomy. If repair of the collecting system is tenuous or incomplete, placement of an internal stent (complemented by a bladder catheter) or a nephrostomy tube may decrease the risk of postoperative urinary extravasation and the formation of a urinoma.

Partial nephrectomy for polar lesions is performed using a "guillotine" technique with the transected vessels and collecting system closed as noted earlier (Figs. 40-22 and 40-23). Topical hemostatic agents may be placed within a parenchymal defect to aid in hemostasis, with the capsule closed over the defect and the hemostatic material. If the capsule can be closed with mattress sutures or absorbable bolsters following debridement or partial nephrectomy, parenchymal hemostasis is aided considerably. If capsular closure is not feasible, either due to the shape and location of the parenchymal defect or due to loss of the capsule from the injury or dissection, using absorbable materials or native tissue as a patch may be helpful if hemostasis is still problematic. The argon beam coagulator has also been used successfully in the kidney to achieve hemostasis in the parenchyma after suturing larger vessels and closing the collecting system. Topical hemostatic agents and tissue adhesives may be used on the kidney, collecting system, ureter, and other urologic repairs to aid in hemostasis and minimize the risk of postoperative urinary

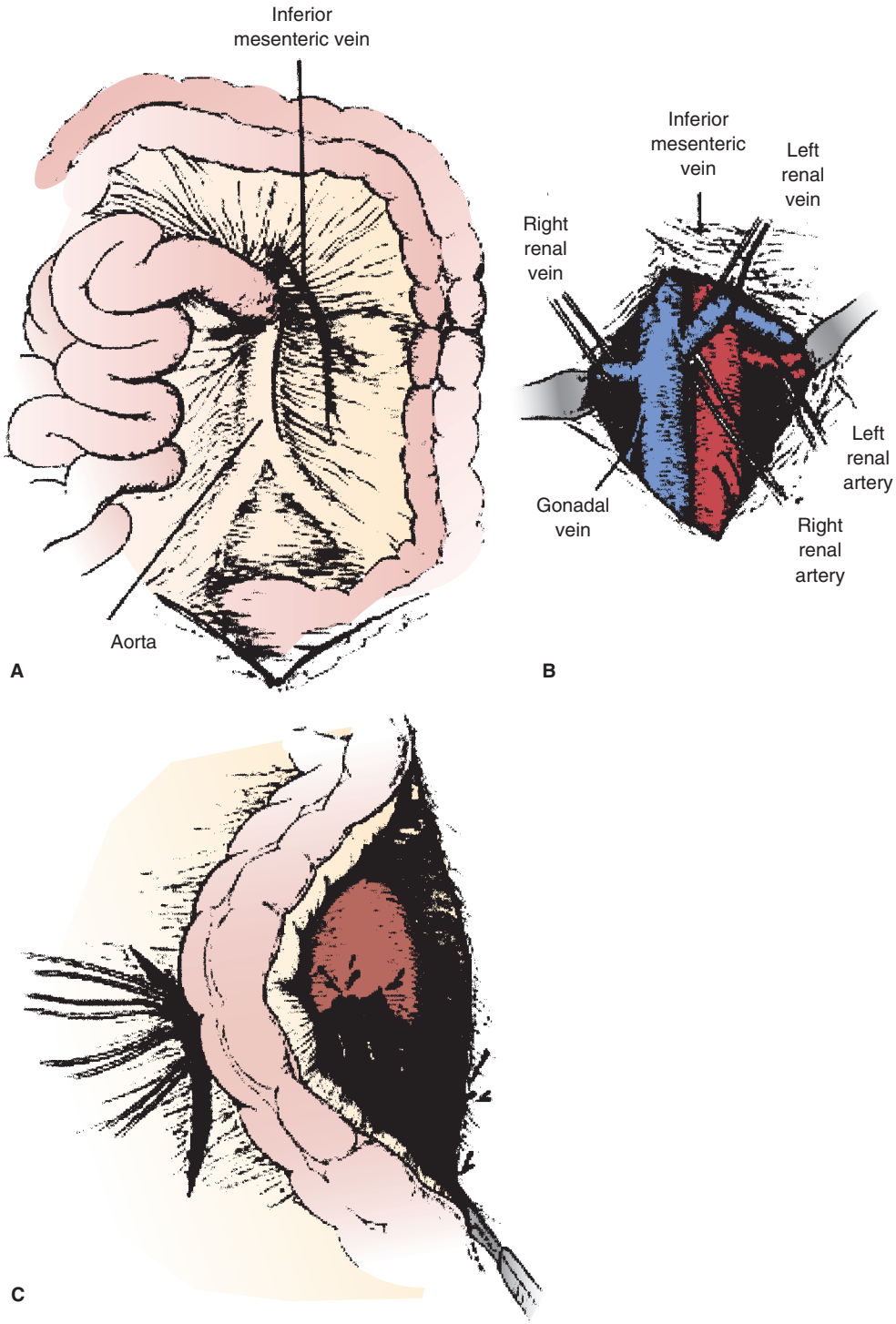


FIGURE 40-19 Surgical management of renal trauma: vascular control. Diagram demonstrating early vascular control prior to renal exploration. (**A**) The posterior peritoneum is opened over the aorta medial to the inferior mesenteric vein. (**B**) The renal vessels are individually dissected and surrounded with vessel loops. (**C**) The colon is reflected medially exposing the perinephric hematoma. Some clinicians believe preliminary control of the renal vessels is not necessary when performing renal exploration for trauma, although best renal salvage rates are reported when vascular access or control is obtained.

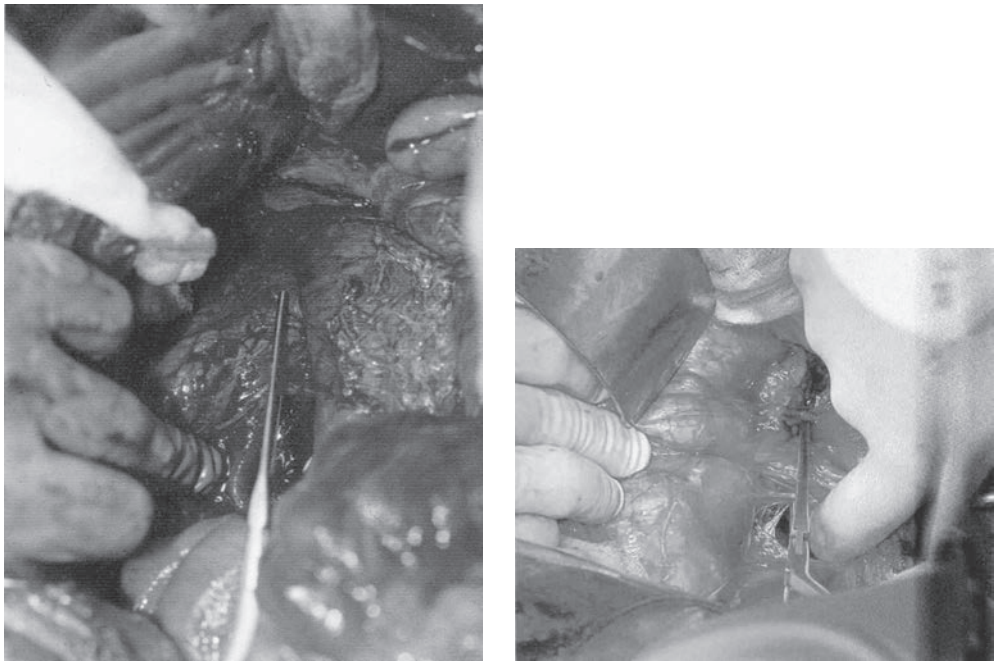


FIGURE 40-20 (**A**) Alternate means of obtaining vascular pedicle access prior to renal exploration. Colon is reflected medially initially. Blunt dissection lateral to vena cava allows creation of space anterior to psoas muscle for placement of pedicle clamp if necessary on renal exposure. (**B**) Comparable technique on the left side, creating space for pedicle clamp lateral to aorta. This approach has been used successfully in the author's center.

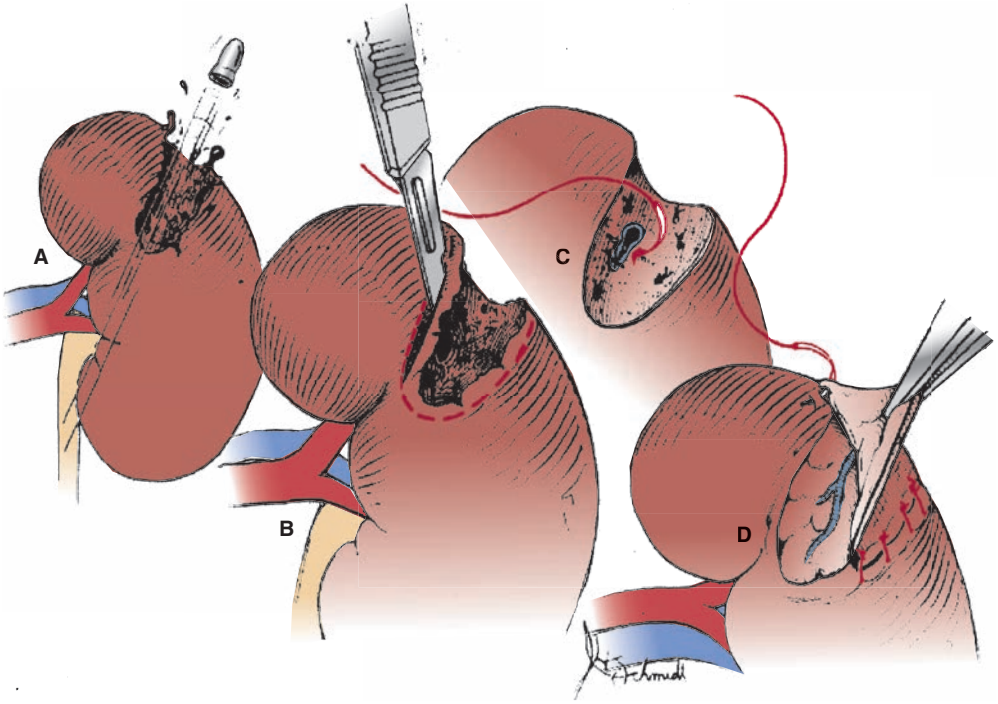


FIGURE 40-21 (**A** and **B**) Wedge resection of injured parenchyma. (**C**) Suturing of open collecting system and significant vessels with absorbable suture. (**D**) Capsule, if present, may be closed or reconstructed using peritoneal patch, with absorbable gelatin sponge or local fat pedicle to aid in hemostasis.

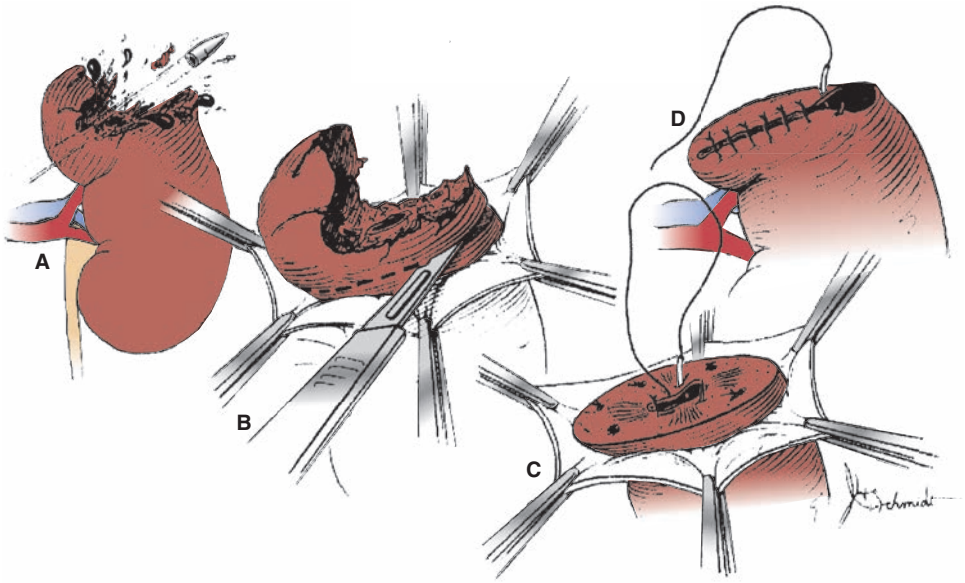


FIGURE 40-22 (**A** and **B**) Partial nephrectomy for major injury to upper pole. (**C**) Repair of collecting system and suturing of bleeding vascular branches. (**D**) Mattress sutures of 2-0 chromic gut to reconstruct parenchyma and aid in hemostasis.

extravasation.[69] Some data exist to suggest that the application of fibrin sealant over a urinary tract suture line may decrease the likelihood of postoperative urinary leakage.[70] At times, wrapping the decapsulated kidney in absorbable mesh material has been used to provide mild temporary parenchymal compression for continued venous bleeding from lacerated parenchyma (Fig. 40-24).

Injuries to adjacent organs such as the liver, pancreas, duodenum, and colon generally do not change the indications for renal salvage versus nephrectomy,[71,72] because good results have been described for renal repairs in the presence of injuries to these adjacent organs. It is desirable, however, to separate the renal injury from the adjacent visceral injury using available viable tissue. This can be accomplished by replacing the kidney within Gerota's fascia and closing the fascial layer over the kidney or by using omentum in the form of a pedicle flap. Drains for renal injury are used when the injury is complex, incompletely repaired injuries to the collecting system are present, or there is concern for the need to evacuate blood postoperatively. Closed suction drains are used because there is a lower risk of contributing to postoperative infection. When an injury to an adjacent organ exists, the organ sites should be drained separately.

Certain injuries are more common in the pediatric population and deserve specific mention. Avulsions of the fornices, ureteropelvic junction, and renal pedicle are more commonly seen in the pediatric population than in the adult.[73] Complete forniceal avulsion injuries are managed with nephrectomy because repair is nearly impossible. Avulsions of the ureteropelvic junction are amenable to repair through a direct anastomosis. Lacerations of the renal pelvis should also alert the trauma surgeon to the possibility of a preexisting obstruction of the ureteropelvic junction. Repair of the obstructing lesion may need to be performed with closure of the pelvis, or nephrectomy may be preferable if the kidney appears to have minimal parenchyma due to long-standing obstruction.

Renovascular injury from blunt or penetrating trauma presents certain challenges. As noted earlier, select patients are taken to laparotomy for revascularization surgery based solely on a CT scan demonstrating the classic findings of renal nonperfusion following deceleration trauma. If exploration is undertaken based on the CT findings or if arteriographic imaging has been performed, the approach is similar. The artery is dissected from its origin at the aorta toward the kidney and the arterial pulse is palpated or assessed with a Doppler instrument. The artery is clamped near the aorta and opened at the circular ring of hematoma, resected to the point of normal anatomy, and a direct end-to-end anastomosis is performed. When necessary, an autogenous vein graft or prosthetic graft is interposed. As in the pediatric population (in which the injury is more common), avulsion injuries involving the renovascular pedicle require urgent surgical intervention. Most of these patients are managed with nephrectomy, although isolated vascular repairs have been described depending on the level of the avulsion. Avulsion of multiple branches from within the renal sinus is virtually impossible to repair in the trauma setting and generally requires nephrectomy as well. Although current data suggest that the likelihood of achieving a favorable outcome with renal revascularization following renal injury is low,[74] patient selection is critical. In the appropriate clinical setting (brief warm ischemia time and a patient in suitable condition for surgery), the effort may be worthwhile in carefully selected patients. A collaborative approach involving the vascular surgeon and the urologist is highly applicable to cases in which renovascular reconstruction is planned. In selected

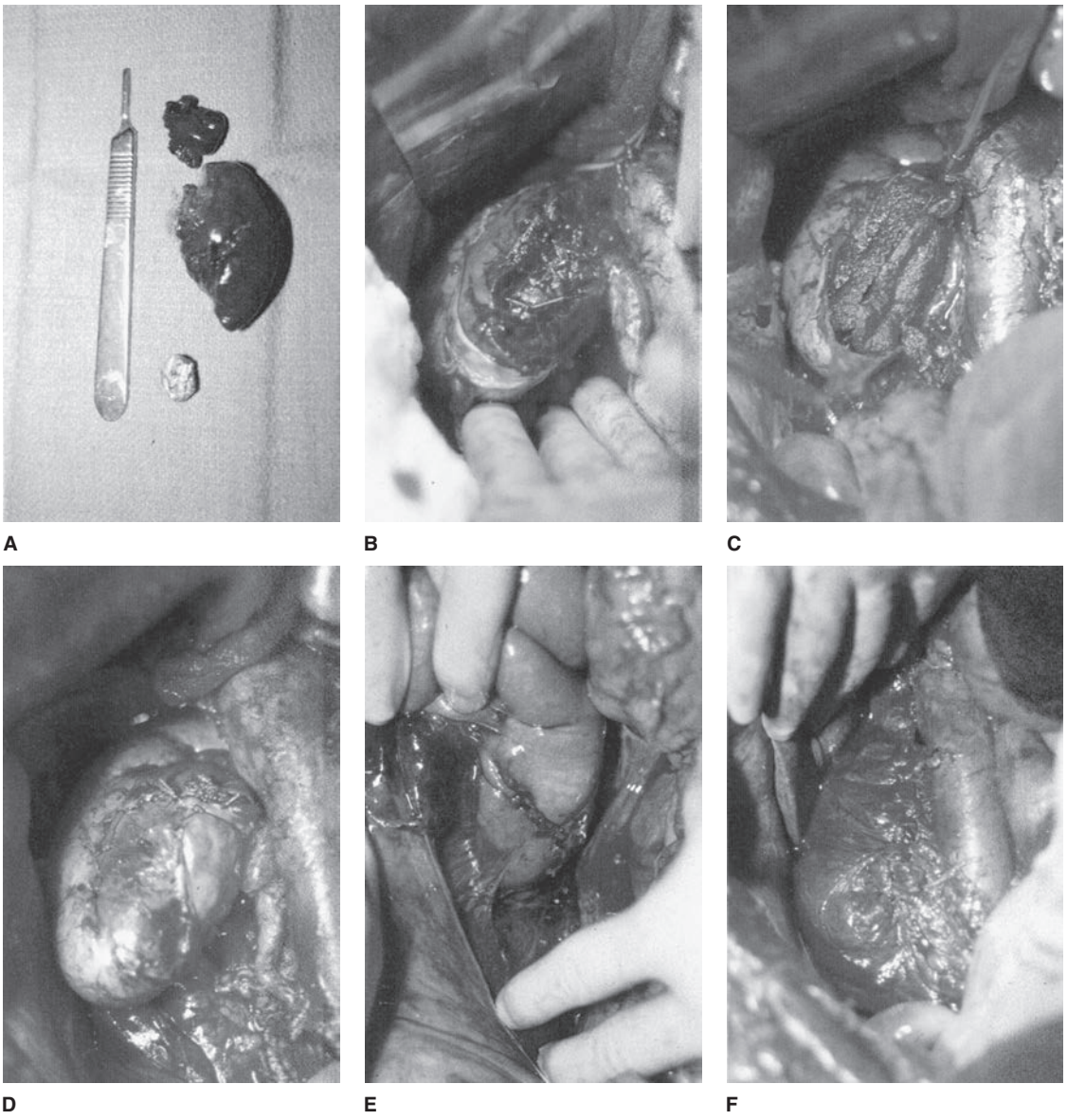
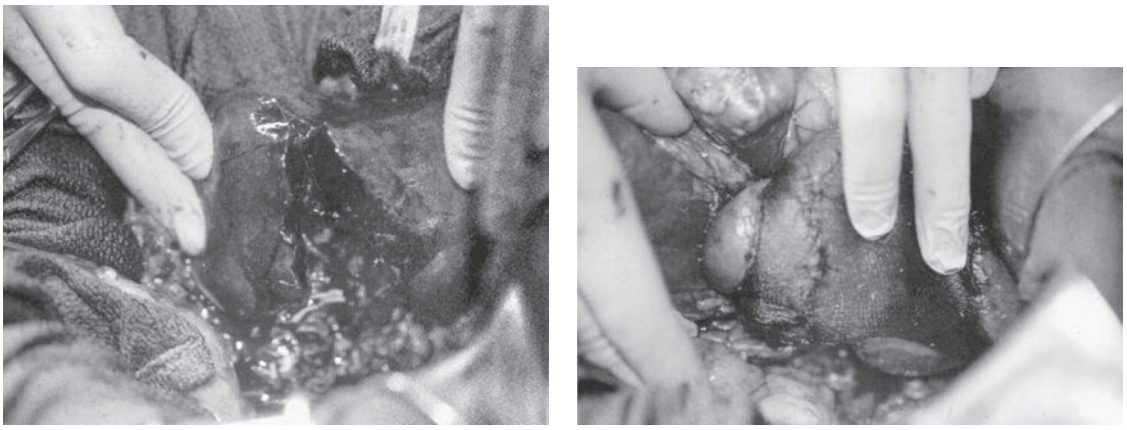


FIGURE 40-23 Surgical management of renal trauma. (**A**) Partial nephrectomy for lower pole laceration due to gunshot wound. Excised fragment of devascularized, lower pole parenchyma, debrided. Bullet removed, found immediately posterior to kidney. (**B**) Appearance of lower pole following suture repair of vessels and repair of collecting system. Capsule has been reflected back for completion of partial nephrectomy and will be used for coverage of defect. (**C**) Defect covered with absorbable gelatin sponge soaked in thrombin. Note vessel loops surrounding renal vessels. (**D**) Defect covered with adjacent capsule and peritoneal patch to aid in hemostasis. (**E**) Duodenal injury, repaired, immediately anterior to the renal injury. It is desirable to separate such injuries with viable tissue interposition, when possible, to minimize the risk of postoperative leak from either source affecting the other repair. (**F**) Gerota's fascia is closed over the kidney to separate the duodenal and renal injuries. Omental pedicle flaps are also very useful for this purpose. The renal repair was drained with an extraperitonealized closed suction drain.

cases in which an intimal disruption of the renal artery is documented arteriographically but perfusion is maintained, radiologic placement of a vascular stent may be applicable. Many limited penetrating injuries to the renal vein can be repaired, whereas arterial injuries have a high rate of nephrectomy. Injuries to branch vessels in a parenchymal laceration are ligated. When diagnosed on imaging studies in stable patients with intact parenchyma, nonoperative management is appropriate.

Bilateral renal injuries are rare and present particular problems.[75] Assuming neither kidney is bleeding briskly, the kidney that seems to be less seriously injured (eg, based on hematoma size and location, apparent orientation and location of entrance and exit wounds) is assessed to ensure that renal salvage is feasible. One kidney can also be packed off temporarily after obtaining gross hemostasis while the opposite kidney is assessed in an effort to avoid nephrectomy in these cases whenever possible.



A B

FIGURE 40-24 Surgical management of renal trauma: renal parenchymal injury due to blunt trauma. (**A**) Large, deep laceration through posterior parenchyma, left kidney. Bleeding sites are sutured and collecting system closed with absorbable suture. Venous bleeding continues from lacerated cortex. (**B**) Due to absence of renal capsule (dissected away from parenchyma by hematoma), absorbable surgical mesh is used to wrap renal parenchyma, providing gentle compression to assist in achieving hemostasis.

Although rarely indicated, ex vivo renal reconstructive surgery may be used in the trauma setting. This would be the case when a solitary (functionally or anatomically) kidney is injured and a complex reconstruction is needed for salvage.

Ureter

The approach to ureteral repair depends largely on the level of the injury, the amount of ureteral loss (if any), and the condition of the local tissues. A ureteral laceration along with extensive destruction of the kidney from blunt or penetrating trauma is generally managed with nephrectomy. If the kidney is uninjured or the renal injury is limited and can be observed or repaired, ureteral repair is best performed at the time of recognition.[76,77]

Injuries to the ureter from blunt trauma require a high index of suspicion for diagnosis. Hematuria may be absent in such cases, and a delayed presentation is not uncommon. As noted earlier, the spiral CT scanners complete the initial renal imaging survey so rapidly that, unless a delayed excretory phase is requested, the study may be completed before the contrast has opacified the collecting system or injured ureter.

Blunt avulsion of the proximal ureter or ureteropelvic junction is best managed with limited debridement to viable tissue and a spatulated end-to-end anastomosis using fine absorbable suture (3-0, 4-0, or 5-0). In general, ureteral repairs performed after trauma are often stented (see Atlas Figure 64). This can be performed with an internal double-J-type stent or an externalized single-J stent. The single-J stent is usually exteriorized through a small stab incision in the anterior bladder wall and secured with a purse-string suture. Some surgeons also secure the stent to the bladder mucosa just outside the ureteral orifice with a fine absorbable suture (4-0 or 5-0). For tenuous repairs of the proximal ureter, diversion using a nephrostomy tube may be considered but is generally unnecessary.

A blunt injury to the mid ureter is uncommon, but when diagnosed, it is managed with a primary anastomosis. In the distal ureter (below the internal iliac artery), ureteral reimplantation into the bladder is preferred.

Injuries to the ureter from penetrating trauma also require a high index of suspicion for diagnosis. The presence of urine in the operative field may be difficult to appreciate, and the ureters, when at risk, must be thoroughly assessed by intraoperative inspection. The proximal and mid ureters down to the internal iliac arteries are easy to visualize and examine. For very distal injuries, a vertical cystotomy with observation of efflux from the ureteral orifices and intraoperative retrograde pyelography may be a less morbid means of assessing the area of concern rather than embarking on a difficult dissection of the ureter all the way to the bladder in the setting of a pelvic hematoma. Alternatively, intraoperative flexible cystoscopy with retrograde pyelography may be performed, avoiding the cystotomy. For proximal ureteral and mid-ureteral injuries, limited debridement of damaged tissue and a tension-free, spatulated end-to-end anastomosis is the procedure of choice (Fig. 40-25). For very distal injuries (generally below the internal iliac artery), reimplantation into the bladder is preferred, as noted earlier, because the blood supply to the distal ureteral stump may be compromised. A direct anastomosis to the bladder avoids the potential ischemic complications of a very distal ureter-to-ureter anastomosis. Stenting of such repairs is routine, as described previously.

For injuries to the lower third of the ureter, it is not always possible to perform a direct anastomosis to the bladder without tension. In such cases, the bladder can be brought cephalad and lateral toward the injured side to achieve a tension-free anastomosis with the ureter by several techniques. The most commonly employed is the "psoas hitch" (Fig. 40-26). The bladder is opened anteriorly, lateral peritoneal attachments are divided as needed, and then the bladder body is displaced toward the side of the injury and sutured

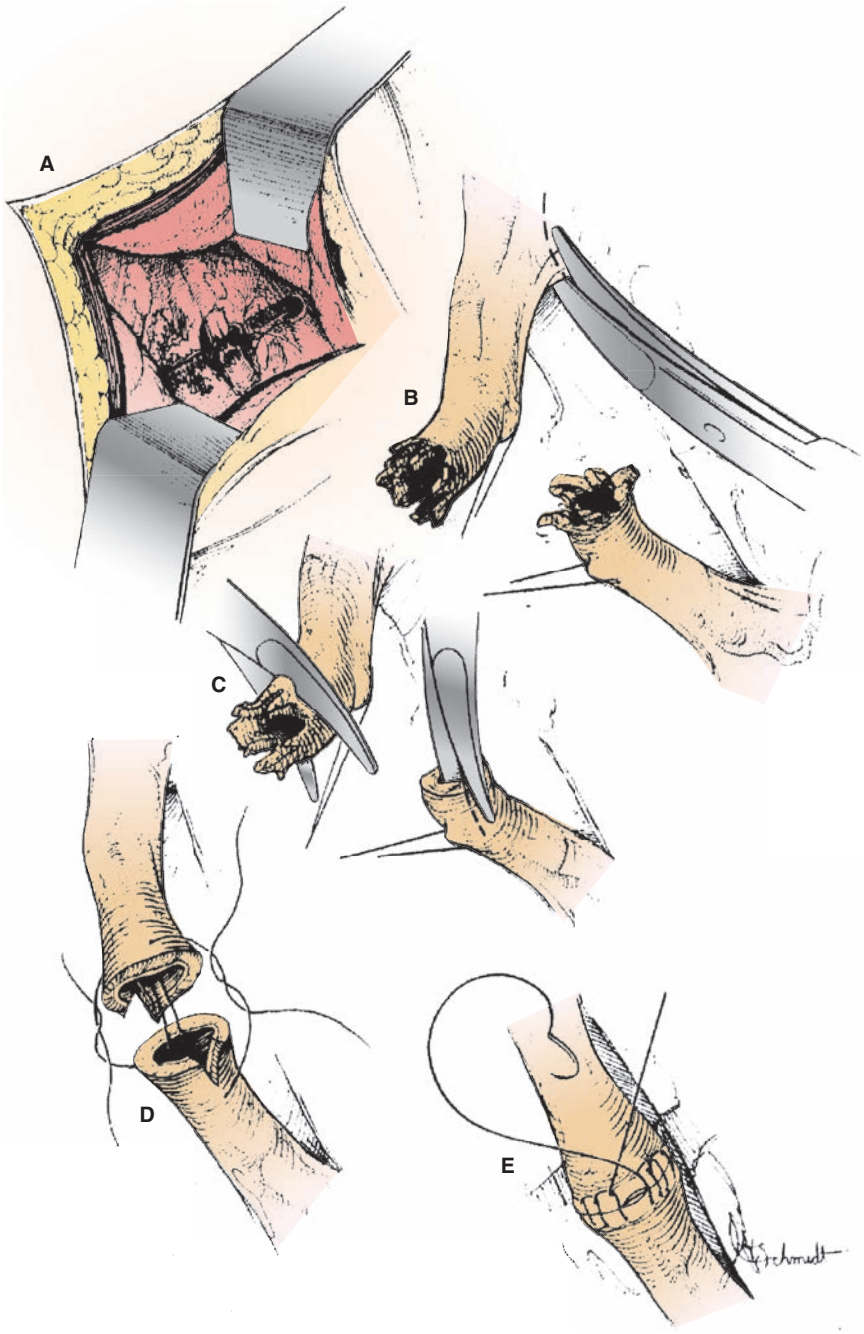


FIGURE 40-25 Techniques of ureteral reconstruction. Debridement and primary anastomosis for ureteral transection from gunshot wound. (**A** and **B**) Mobilization of ureter superficial to adventitial plane. (**C**) Limited debridement of lacerated ureter to viable tissue with spatulation for repair. (**D** and **E**) End-to-end anastomosis with fine absorbable suture over stent (not shown).

to the psoas muscle with 2-0 absorbable suture, taking care not to injure or entrap any major nerves. The ureter can then be reimplanted into the bladder using a tunneled antirefluxing anastomosis, or the tunnel can be omitted if length is still a problem. It is important to ensure that no obstruction or acute angulation exists at the vesical hiatus where the ureter enters. If a psoas hitch cannot achieve a tension-free connection to the ureter, a bladder flap (Boari flap) can be created. This procedure has a higher complication rate than a psoas hitch and is performed only if the psoas hitch does not accomplish the required objective. The bladder flap may be performed in conjunction with the psoas hitch to maintain the cephalad extension of the bladder wall posterior to the flap. Again, a nonrefluxing tunneled or a refluxing repair can then be performed. These procedures can also be performed laparoscopically.[78]

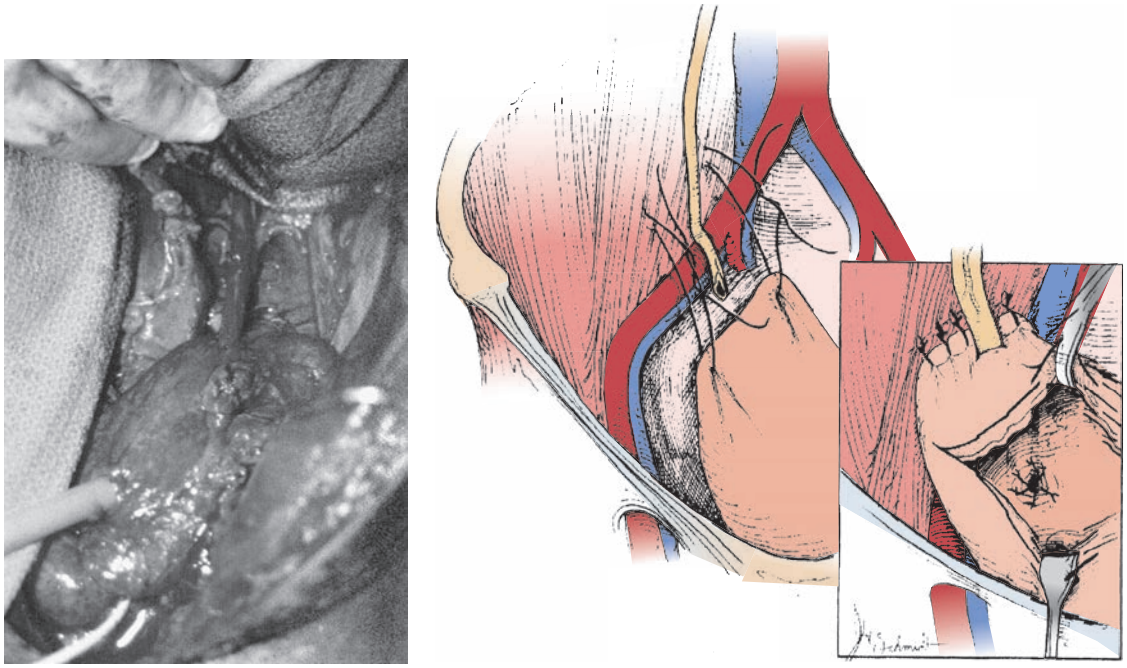


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FIGURE 40-26 (**A**) Ureteral reimplantation with psoas hitch for lower ureteral injury: the bladder is opened either transversely or vertically and obliquely toward the side of injury and then hitched to the ipsilateral psoas muscle with 2-0 or 3-0 Vicryl suture. A tunneled, antirefluxing anastomosis of the ureter to the posterior wall of the bladder is performed, being certain that an adequate width tunnel is created to prevent obstruction. If the available ureteral length is short, antirefluxing tunneling can be eliminated. Either an internal double-J–type stent or an externalized single-J stent can be used (not shown). (**B**) Psoas hitch ureteral reimplantation for penetrating injury to lower ureter, performed acutely during initial laparotomy in a hemodynamically stable patient. The bladder body can be seen sutured to the left psoas muscle, with the ureter entering cephalad. A single-J ureteral stent and suprapubic cystostomy exit from the bladder in the lower part of the photograph.

More complex techniques of ureteral reconstruction include transureteroureterostomy (TUU) (Fig. 40-27), ileal-ureteral replacement, and renal autotransplantation (Fig. 40-28). TUU is relevant when anastomosis to the bladder is not feasible due to inadequate length of the ureter, the condition of the bladder, or when it is desirable to move the repair away from the ipsilateral hemipelvis due to local conditions such as infection or prior pelvic radiation. Ureteral replacement with the ileum is seldom performed in the acute trauma setting because it is preferable to have a fully prepped bowel when performing this procedure. Renal autotransplantation may be appropriate in the acute trauma setting if appropriate vascular surgical expertise is available and less complex options for ureteral replacement are not feasible. The proximal ureter can be anastomosed directly into the bladder in the case of loss of the majority of the lower ureter, or an anastomosis can be performed to the lower ureter if it is clearly viable and not excessively distal.

When ureteral repairs are performed in direct apposition to adjacent vascular or visceral repairs, separation of the repairs by an omental pedicle or other viable tissue is desirable to prevent a fistula or contact with urine at the site of the adjacent organ injury. External drainage of ureteral injuries, in addition to stenting or diversion, may be desirable, particularly if the repair is tenuous or the vascularity of the repaired tissues is questionable. Some urologists prefer Penrose drains to closed-suction drains. In the postoperative period, antibiotic administration may be desirable, especially if urinary extravasation persists.

As noted later, ureteral injuries are also highly amenable to damage control strategies when the patient is not in suitable condition for repair at the time of the initial laparotomy. An external stent placed through the transected proximal ureteral stump allows maintenance for control of the urinary output while the patient is undergoing resuscitation in preparation for definitive delayed reconstruction.

Bladder

Surgical repair of the bladder is performed for many iatrogenic injuries, for nearly all blunt intraperitoneal injuries, and for selected cases of blunt extraperitoneal rupture. Penetrating injuries to the bladder may also be managed with operative repair.

Intraperitoneal ruptures of the bladder are approached through a midline abdominal incision or laparoscopic repair. The large laceration is nearly always in the dome of the bladder as previously described (Fig. 40-29). The interior of the bladder is palpated and inspected through the laceration to verify that no other injuries are present and that there is clear efflux from both ureteral orifices. The laceration may be

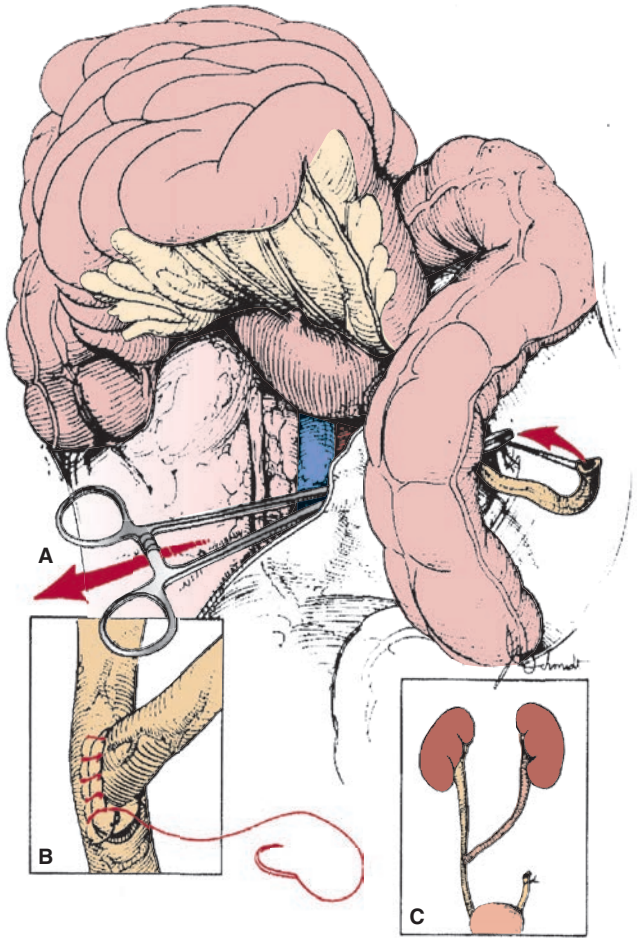


FIGURE 40-27 (**A**–**C**) Transureteroureterostomy (TUU) is performed when the loss of the distal part of the ureter is sizeable and ureteral reimplantation cannot be performed. (**A**) Use of right angle clamp to transpose carefully the left ureter to the right side through a peritoneal window (two fingerbreadth wide to prevent ureteral constriction) at the level of the promontory. (**B**) The injured left ureter is mobilized, spatulated, and reimplanted to the right ureter with mucosa–mucosa apposition, water-tight anastomosis and tension-free. (**C**) The left ureter is ligated at the site of injury prior to the TUU being performed. The left ureter should have a smooth transition at the transposition site to the right side to prevent obstruction as demonstrated in the figure.

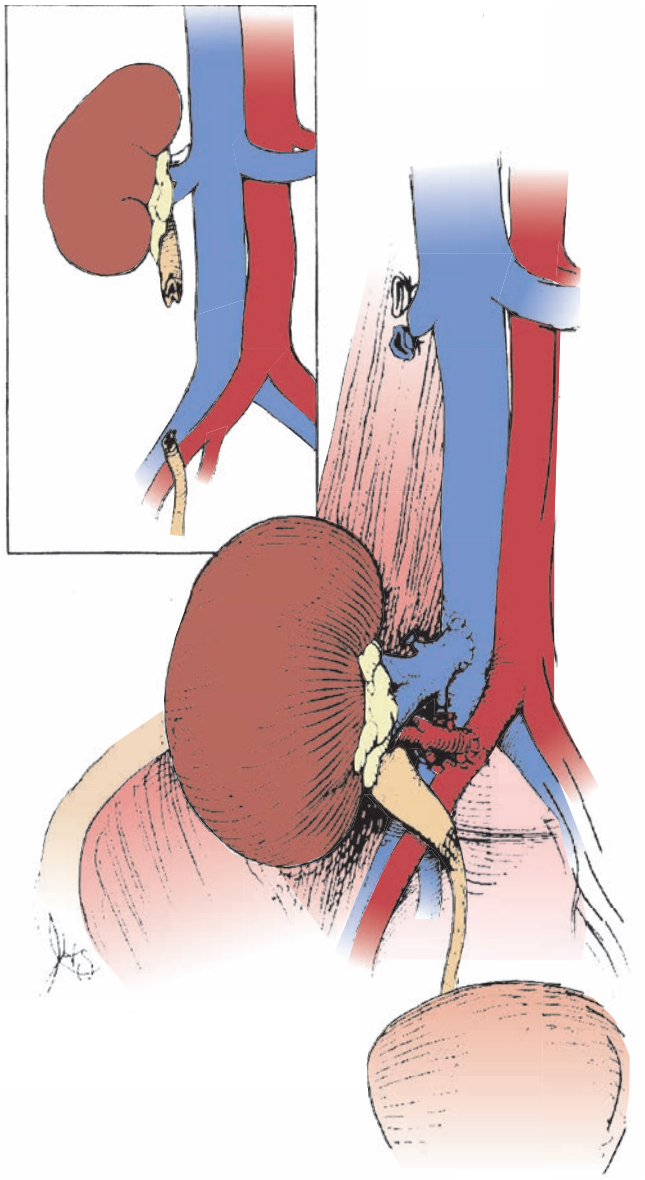
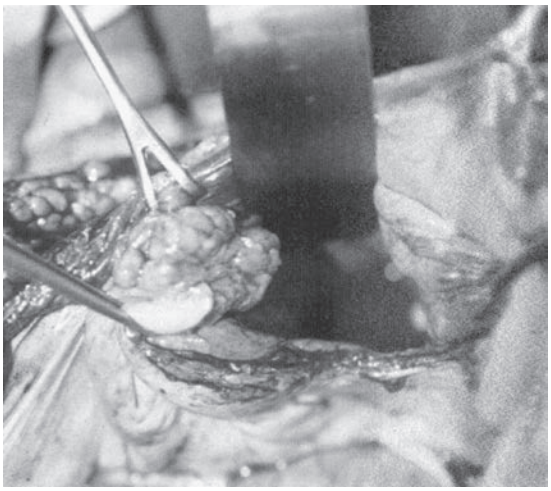


FIGURE 40-28 Renal autotransplantation for reconstruction following extensive loss of mid ureter, making direct union of upper ureter to bladder impossible. Alternative to ileal-ureteral replacement of most of the ureter. Nephrectomy must be tailored to include as much of the renal vessels as possible to aid in anastomosis to iliac vessels (in general, vein generally transected flush with vena cava on right, with artery transected more proximally, behind vena cava than shown here). Anastomosis of proximal ureter to viable lower ureter.

extended into an anterior midline cystotomy if further assessment is needed, but this is not usually necessary. The edges of the bladder laceration may require minimal debridement to remove devascularized tags of detrusor muscle or mucosa. The laceration is then closed using two layers of heavy absorbable suture. An adequate bore bladder catheter is used to allow free drainage of initially bloody efflux that clears in the first few days. The length of catheterization time is usually 5 to 10 days, but the period needed for urinary efflux to clear and the ability of the patient to be ambulatory and void comfortably should be considered. It is prudent to perform a cystogram prior to removal of the catheter following any operative repair and is mandatory with nonoperative management. Because a well-sutured repair carries an extremely low postoperative risk of extravasation, some practitioners remove the catheter without prior contrast imaging with excellent success. Suprapubic cystostomy catheters are not generally needed after repairs of intraperitoneal ruptures. They should be inserted only when there is a need for long-term bladder drainage, such as in a patient with a significant injury to the



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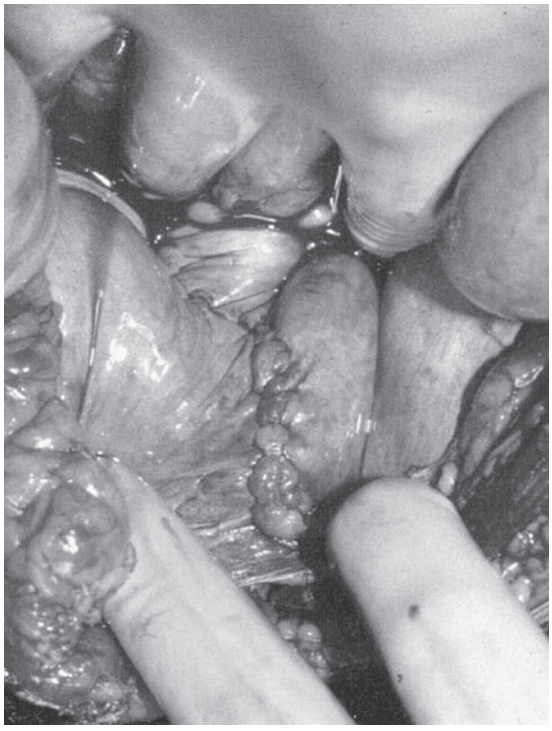

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FIGURE 40-29 (**A**) Intraperitoneal bladder dome rupture from blunt trauma. The retractor is inside of the bladder. (**B**) Cystorraphy should be performed with continuous absorbable suture, water-tight repair without exposed bladder mucosa to prevent fistula formation.

brain, trauma to the pelvis or a lower extremity, or other factors that would be expected to substantially delay a return to ambulation.

For the selected cases in which extraperitoneal rupture of the bladder is managed with operative repair, there are several important differences when compared with intraperitoneal repairs. When operating on the injured bladder during a laparotomy following a pelvic fracture, an effort should be made to avoid entering the retropubic hematoma. This avoids potentially serious hemorrhage from a site that is often tamponaded. If repair of the bladder is necessary in this setting (see the previous section "Nonoperative Management of Genitourinary Injuries"), one should enter the bladder through an anterior cystotomy incision cephalad to the pelvic hematoma. The laceration, which is usually located in the lower anterior or anterolateral bladder, can be sutured transvesically by introducing Deaver or malleable retractors into the bladder and retracting them laterally. Often, only a single-layer, full-thickness closure is possible in this setting. It is useful to communicate with the orthopedic surgeons when operating on extraperitoneal bladder ruptures in the setting of a pelvic fracture to allow for coordinated care.

A penetrating injury to the bladder is most often managed operatively, although occasional patients (as previously described) may be candidates for nonoperative management.[62] If a patient is undergoing laparotomy and has gross hematuria following penetrating pelvic trauma, the peritoneal surface of the bladder is examined first. The retropubic space is then entered, and an anterior, midline cystotomy is created. This may be easier to accomplish if the bladder is partly filled with irrigant. For laparotomies in which bladder surgery is likely, including the genitalia in the sterile field can facilitate whatever manipulation may be necessary without abdominal contamination. Following cystotomy, the interior of the bladder, the ureteral orifices, and the bladder neck are thoroughly examined. The urinary efflux from both orifices should be observed; if bloody or absent, further investigation for trauma to the ureters or upper tract is recommended. Penetrating injuries to the bladder are closed with two layers of absorbable suture, as previously described.

In some patients, an iatrogenic or penetrating injury to the bladder may result in loss of a large portion of the detrusor of the bladder body. Closure over a bladder catheter is still recommended, as the bladder may expand to an acceptable volume with time. If minimal bladder capacity persists following a reasonable period of healing, augmentation cystoplasty can be performed electively.

As for renal and ureteral injuries, injuries to the bladder in the unstable trauma patient are amenable to damage control strategies. These include externalized stenting of the ureters with pelvic packing and delayed repair of complex lacerations.

Certain associated injuries impact the management of bladder trauma. Contiguous injury to the vagina or rectum, for example, requires close collaboration between the clinical services involved in caring for these injuries. When such injuries are suspected, it is helpful to have the patient in a modified dorsal lithotomy position so simultaneous access to the perineum and abdomen can be obtained. During surgical repair, the bladder should be separated from the rectum or vagina by placing an interposition flap of viable tissue if the loss of tissue is significant and the injuries directly overlie each other. This effort of separating the pelvic organs can be difficult in the trauma setting, and if the injuries do not directly overlie each other and tissue loss is minimal, simple transvesical closure is generally adequate. In this setting, longer indwelling catheter times, perioperative antibiotics,

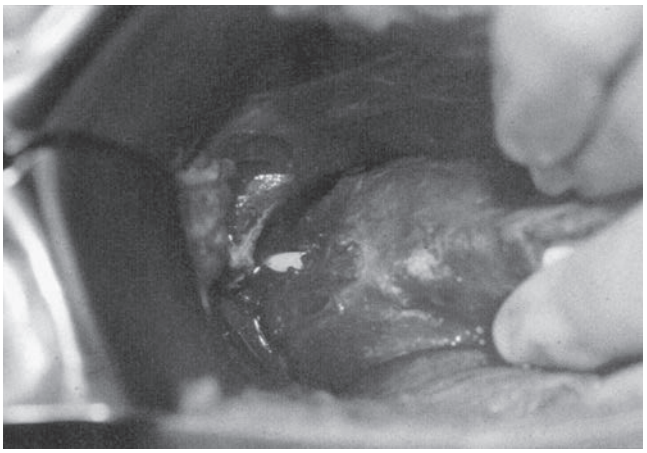


FIGURE 40-30 Bladder neck avulsion injury in an adult female with pelvic fracture. Operative appearance during surgical repair. An anterior midline cystotomy had been performed (to right in photo), with the tip of a Foley catheter protruding from the avulsed bladder neck for demonstration purposes. Anastomosis to urethral stump at level of pelvic floor performed over Foley catheter. The patient was initially managed with a percutaneous suprapubic cystostomy. This repair was performed 36 hours following injury, when the patient was hemodynamically stable and risk of excessive bleeding from the pelvic fracture would be lower.

and radiographic imaging prior to removal of the catheter are recommended. Open pelvic fractures are among the most devastating injuries in orthopedic trauma, and injury to the lower urinary tract may complicate such injuries. A close interaction between the urologist, orthopedist, trauma surgeon, and interventional radiologist is necessary for management of such patients. Chronic disability is common following these injuries.[79]

Avulsion injuries of the bladder neck, which are more common in the pediatric population, require operative repair (Fig. 40-30).[54] Repair for these complex injuries should be delayed until 24 to 72 hours after injury to support a damage control strategy and to minimize the risk of excessive hemorrhage from an associated pelvic fracture.

Urethra

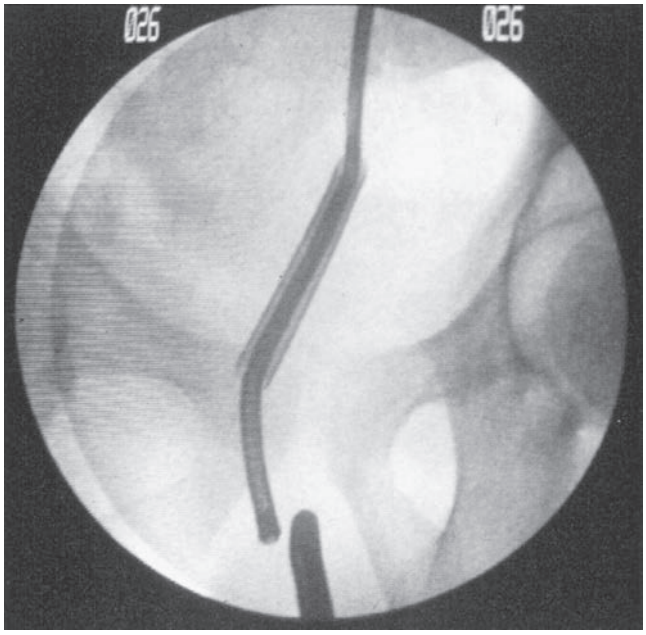
Operative management for urethral trauma includes the broad topic of elective urethral reconstruction following traumatic injuries and surgical repair of urethral strictures, and there are several in-depth reviews available regarding this topic.[80,81] This discussion will focus on immediate and subacute surgical intervention for urethral trauma, and clinical guidelines have recently been reported for these injuries.[82]

Incomplete anterior urethral injuries may be managed with the placement of a transurethral catheter or with suprapubic diversion. Endoscopic guidance for any attempt to catheterize the traumatized urethra is preferred. If a blind attempt at catheterization is performed and any resistance is encountered, an endoscopically guided procedure should follow. Complete ruptures of the anterior urethra from blunt trauma are best managed with suprapubic diversion for 3 months or longer, followed by elective end-to-end urethroplasty when the perineal hematoma and induration have fully resolved. Acute attempts at excision and repair are not recommended because it is unclear how much urethra should be resected due to the crush injury and result in tension-free healthy tissue anastomosis.

Penetrating injuries to the anterior urethra may be managed with local exploration and repair or with suprapubic diversion. With stab wounds or gunshot wounds from low-velocity missiles, performing limited debridement and repair with a spatulated anastomotic technique is usually simple. If the patient is not an appropriate candidate for immediate repair due to more serious injuries or other reasons, suprapubic diversion or endoscopically guided insertion of a transurethral catheter is performed. Extensive loss of the urethra from penetrating trauma or industrial trauma may require a staged repair.

The management of disruption or distraction injuries of the posterior urethra remains controversial. In recent years, there has been increasing interest in early catheter realignment for such injuries. Techniques used have included endoscopic guidance, open surgical approaches, and, historically, the use of interlocking magnetic sounds (Fig. 40-31).[83-85] A potential advantage of endoscopic realignment is the possibility that the injury will heal free of intractable stricture. This would obviate the need for late urethroplasty, shorten the period of urinary intubation, and may improve the anatomic result as compared with the nonintubated state by reducing malalignment. A potential disadvantage of this approach is the risk of infecting the retropubic hematoma because of the indwelling catheter, which can adversely impact late continence and sexual function; in addition, there is a high likelihood that a stricture will form regardless. When selected, catheter realignment should be performed by an experienced team in the operating room with endoscopic and fluoroscopic capability. Results are better for incomplete disruptions than they are for complete disruptions. Most patients managed in this manner develop a stricture that will require endoscopic intervention, often involving multiple procedures. Overall, patients managed with catheter realignment may avoid a subsequent urethroplasty about 50% of the time. Currently, a multi-institutional prospective cohort trial is underway to determine the utility of acute urethral realignment after pelvic fracture urethral injuries. The study is comparing the outcomes between primary endoscopic realignment and suprapubic tube placement.[86]

The traditional approach to a posterior urethral distraction injury is diversion with a suprapubic cystostomy, followed by a period of observation of 3 to 6 months while the pelvic hematoma resolves and the anatomy stabilizes. Repeat antegrade and retrograde urethrograms are then performed, and definitive reconstructive surgery is planned. The ultimate success rate of this approach is over 90%; however, the need for a long-term indwelling suprapubic tube while awaiting surgery may be frustrating for the patient. Nevertheless, endoscopic techniques such as catheter realignment can



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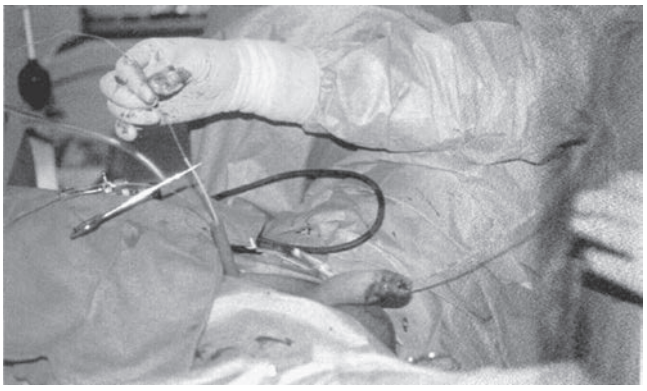


B

FIGURE 40-31 (**A**) Urethral realignment with complete posterior urethral injury. (**B**) Endoscopic realignment under fluoroscopy using flexible and rigid cystoscopies as well as a super stiff guidewire.

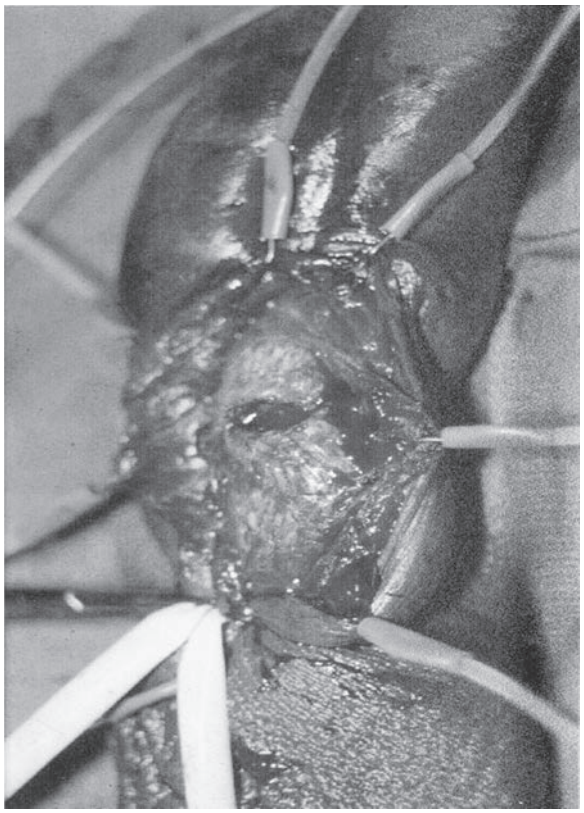


FIGURE 40-32 Penile fracture. Appearance of penis during surgical exploration for penile fracture sustained during sexual intercourse. Patient reported classic findings of pain, swelling, and detumescence following sudden marked bending of erect penis. Note marked swelling of distal phallus with subcutaneous hematoma. Penis is explored through a ventral, midline, penoscrotal incision. Dissection to area of palpable irregularity along penile shaft reveals transverse laceration of tunica albuginea of corpus cavernosum. A penile tourniquet, using a Penrose drain, is in place to reduce bleeding during repair. The hooks are part of a ring-retractor system commonly used in genital surgery. The tunica albuginea defect is closed with running 3-0 Vicryl suture. Early exploration and repair for penile fracture injuries produce the best results. Circumcising subglandular incision is preferred by some surgeons for this type of exploration and repair.

be performed successfully without the need for suprapubic catheters.[87]

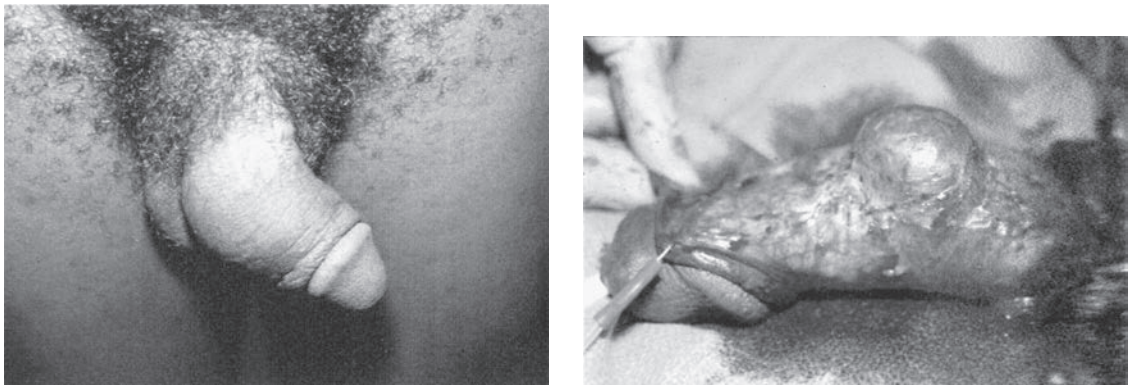
Penis, Testis, and Scrotum

Penile trauma is primarily managed through operative exploration and repair.[88] For blunt penile fractures, the penis is explored through either a ventral midline penoscrotal incision or a circumcising subcoronal incision. The defect in the tunica albuginea is exposed and closed with absorbable suture (Fig. 40-32). The outcomes following early operative repair of penile fractures are far superior to those resulting from nonoperative management. Deformity, painful erection, pseudoaneurysm, and loss of erectile function are common in nonoperative management of such injuries (Fig. 40-33).[63]

Similarly, penetrating penile injuries should be managed with operative exploration and repair (Figs. 40-34 and 40-35).[89] Because combined cavernosal and urethral injury occurs in roughly 10% of penile fractures, a preoperative urethrogram or flexible cystoscopy is useful in planning the repair.

In cases of penetrating penile injury, a similar surgical approach is used with conservative debridement, repair of cavernosal and urethral injury, and microsurgical repair of dorsal neurovascular structures when possible. For limited injuries, direct wound exploration may be a preferable approach. The possibility of an adjacent nonurologic injury (thigh, femoral vessels, pelvic organs) must always be considered in cases of penetrating genital injury.

Penile strangulation injuries due to constricting bands or other devices are managed with removal of the constricting

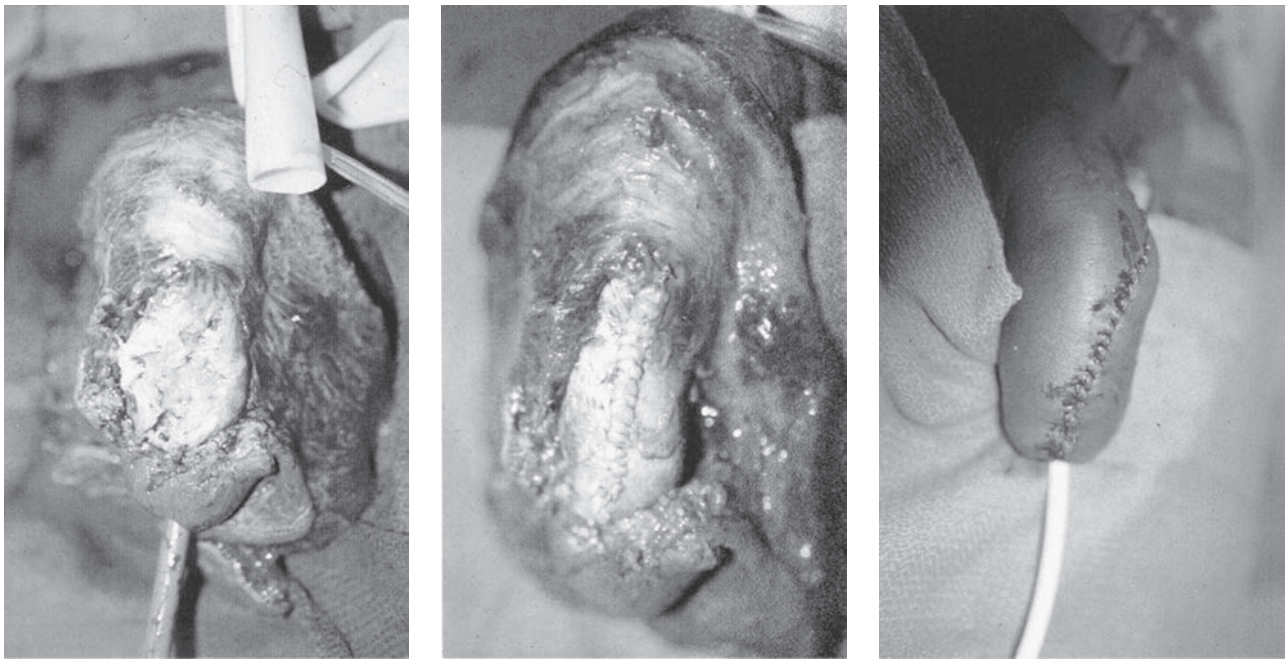


A B

FIGURE 40-33 Delayed presentation following penile fracture. (**A**) Note marked angulation to left with mass effect on right lateral side of penile shaft following untreated rupture. Patient presents 6 weeks after injury; the subcutaneous hematoma has resolved, while the defect in the corpus cavernosum remains, resulting in angulation and pain with erection. (**B**) Appearance of penis at surgical exploration through circumcising incision. Note large encapsulated hematoma under Buck's fascia, which, on incision, still communicates with cavernosal space. Defect repaired with correction of deformity.

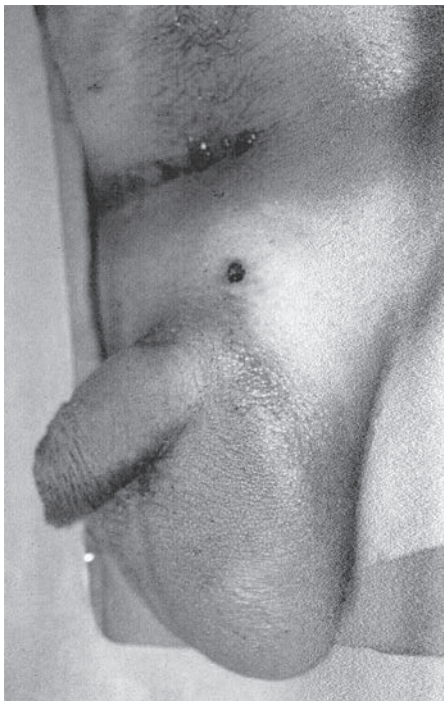
object in the most atraumatic manner possible. Distal penile skin, glans, cavernosal, or urethral necrosis can occasionally occur in such cases. A conservative approach to debridement of tissues of questionable viability and diversion with a suprapubic cystostomy tube (if the urethra is compromised) are principles of management.

Patients with traumatic amputation of the penis require specialized management (Fig. 40-36). Often, patients who suffer traumatic amputation through self-mutilation are psychotic and/or are involved in substance abuse and require psychiatric as well as urologic intervention.[90] The severed organ should be cleansed, kept in cold saline-soaked gauze within a sealed bag, and then placed in ice. Replantation surgery is well described by Jordan and Gilbert.[91] In sequence, anastomosis of the corpora cavernosa, urethra, dorsal blood vessels, and nerves should be performed with appropriate



A B C

FIGURE 40-34 Gunshot wound to penis with entrance at dorsal penile base. (**A**) Extensive injury to skin and subcutaneous tissues and laceration of tunica albuginea of corpus cavernosum. Penile tourniquet in place to allow injury assessment while minimizing bleeding. (**B**) Tunica albuginea has been conservatively debrided and closed with running Vicryl suture. (**C**) Appearance of penis following reconstruction of glans and skin tube. Subsequent scar revision was necessary for necrosis of skin edges (not shown). Preservation of soft tissues and conservative debridement demonstrated.



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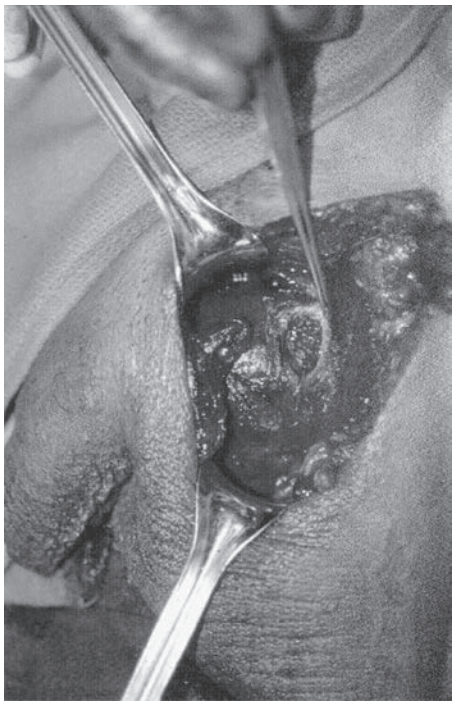
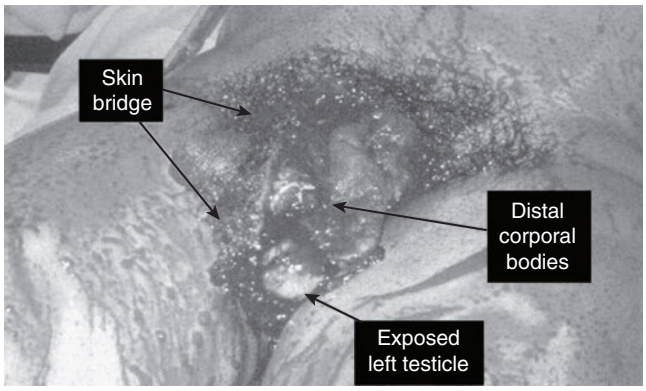


B

FIGURE 40-35 Gunshot wound to mons pubis region, cephalad and to left of penile base. (**A**) No palpable abnormality of penis is recognized. Small left scrotal hematoma present. (**B**) Surgical exploration of wound via oblique scrotal neck incision extends toward groin. Bleeding sites in left spermatic cord were controlled (not shown), followed by evacuation of hematoma resulting in significant bleeding. Dissection revealed complete transection of left corpus cavernosum at penile base, which was repaired. Case demonstrates importance of surgical exploration of penetrating injuries in proximity to male genitalia.



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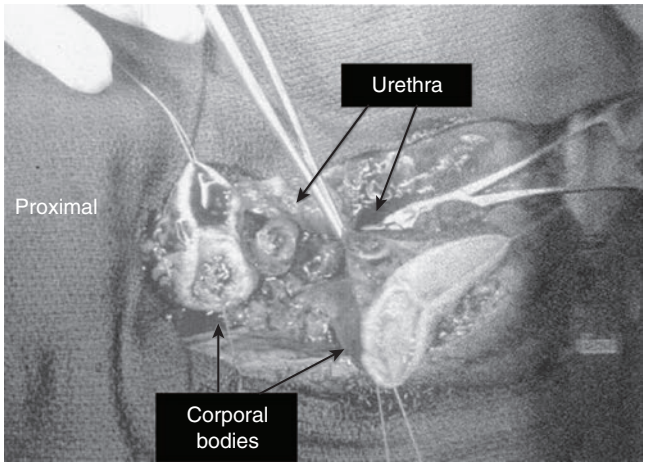

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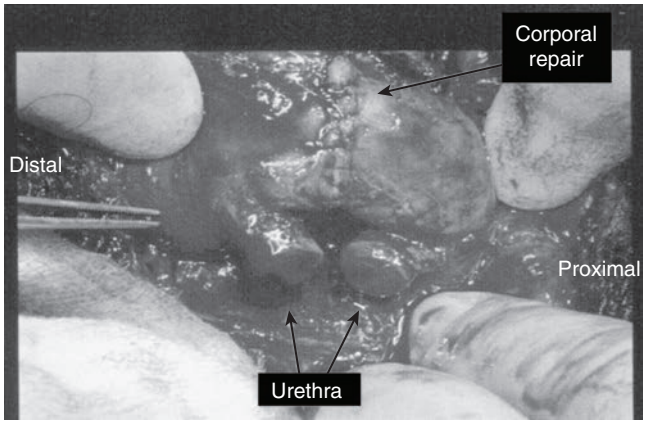
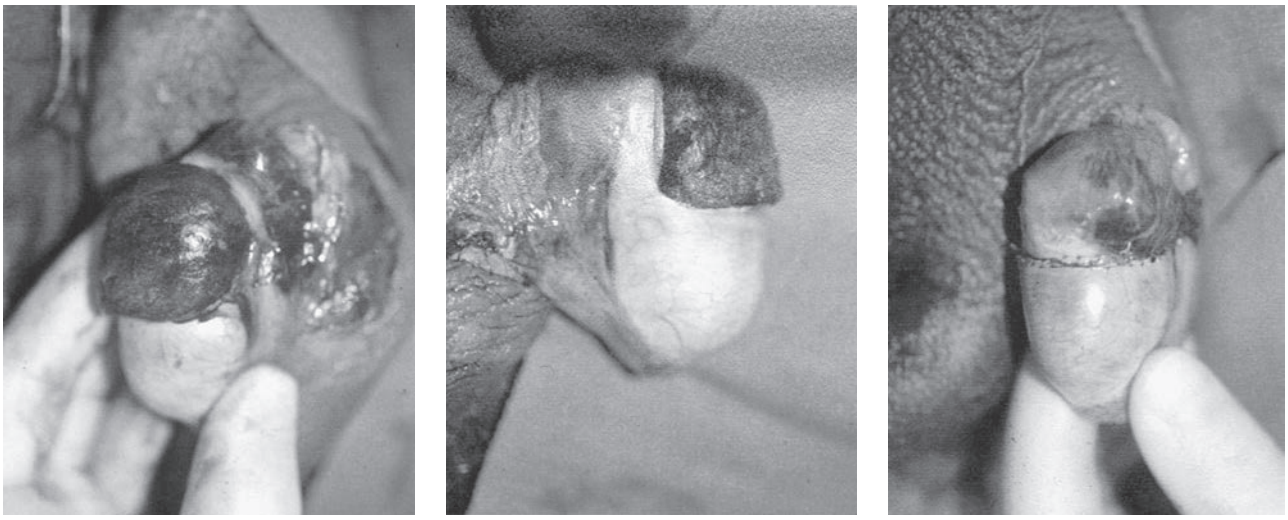


C

FIGURE 40-36 Subtotal penile amputation injury due to assault with knife. (**A**) Photograph demonstrates complete transection of body of penis with right-sided skin bridge attaching distal phallus to body. Left testis is exposed as well. (**B**) Preparing for surgical reconstruction—minimal debridement of corpora cavernosa and urethra, following extensive irrigation. (**C**) Corpora cavernosal anastomosis has been completed; urethral anastomosis about to be completed after spatulation and mobilization of distal ends to avoid tension on repair. Following completion of urethral repair over Foley catheter, microsurgical anastomosis of deep dorsal arteries, deep dorsal vein, and adjacent nerves was performed (not shown).



A B C

FIGURE 40-37 Testis: testicular rupture due to blunt trauma. (**A**) Scrotal hematoma after straddle injury. (**B**) Scrotal exploration: tunica albuginea rupture with extravasation of testicular tissue. (**C**) After testis repair with minimal tissue debridement.

microsurgical expertise. Functional outcomes are variable with such replantation efforts, largely reflecting the condition of the severed organ and the time that elapses prior to replantation.

Scrotal trauma should be explored if there is a concern about testicular rupture. In blunt trauma, a testicular ultrasound may be helpful in deciding if operation is indicated. In penetrating trauma, we often use an oblique upper scrotal incision that provides access to the groin, spermatic cord, penile base, and scrotal contents. Most scrotal injuries should be explored with the goal of evacuation of the hematoma, debridement of devitalized tissue, and repair and salvage of the testicle (Figs. 40-37 and 40-38). Reproductive outcomes are favorable following such management.[92]

Cases of scrotal and other soft tissue loss in the genital region should be managed with a conservative approach to debridement of marginally vascularized skin and soft tissues. Delayed primary closure or reconstruction of significant scrotal loss using meshed split-thickness skin grafting produces favorable results. Human bite wounds have a very high infection rate and should be left open if presenting in a delayed fashion (see Chapter 50).

COMPLICATIONS OF GENITOURINARY TRAUMA

The management of complications of urologic injury is an important issue facing the trauma surgeon. Extensive reviews of this topic are available.[93-95] Complications may be categorized as early and late occurrences and can occur in the setting of an early diagnosis of injury or a delayed diagnosis of injury. Early complications of injury to the upper urinary tract include bleeding, postinjury infections, problems related to urinary extravasation, and ischemic processes. Renal and ureteral injuries may also result in late complications including hypertension, hydronephrosis, and renal insufficiency. Functional abnormalities following trauma to the urinary tract may include a neurogenic bladder, urethral stricture, and sexual or reproductive dysfunction. Appropriate follow-up studies for high-risk injuries are critical in the early detection of complications of urologic trauma.

DAMAGE CONTROL PRINCIPLES IN GENITOURINARY TRAUMA

Damage control in genitourinary trauma is the process of managing immediately life-threatening conditions (ie, major active bleeding from the kidney or renal pedicle or the need for acute urinary diversion), intentionally delaying surgical interventions until the patient is more stable.[96,97] A majority of these delayed procedures are reconstructive surgery of the urinary tract.

The evolvement of diagnostic and interventional radiology, better-understood resuscitation protocols, and the concept of "packing" and "second look" can be applied to the upper and lower urinary tract when there is a need to control renal or pelvic bleeding.

Urinary diversion can be achieved by percutaneous nephrostomy tubes or externalized ureteral stents when major ureteral injuries occur, and suprapubic catheters may be expeditiously performed to drain the bladder when urethral catheters cannot be placed (Fig. 40-39).

Controlling urine leak is important to avoid creatinine elevation due to urine absorption and electrolyte imbalance and maintain potassium levels.

Certain bladder injuries may be difficult to repair at the initial operation as well. Visibility may be compromised because of packing from pelvic bleeding, the complexity of

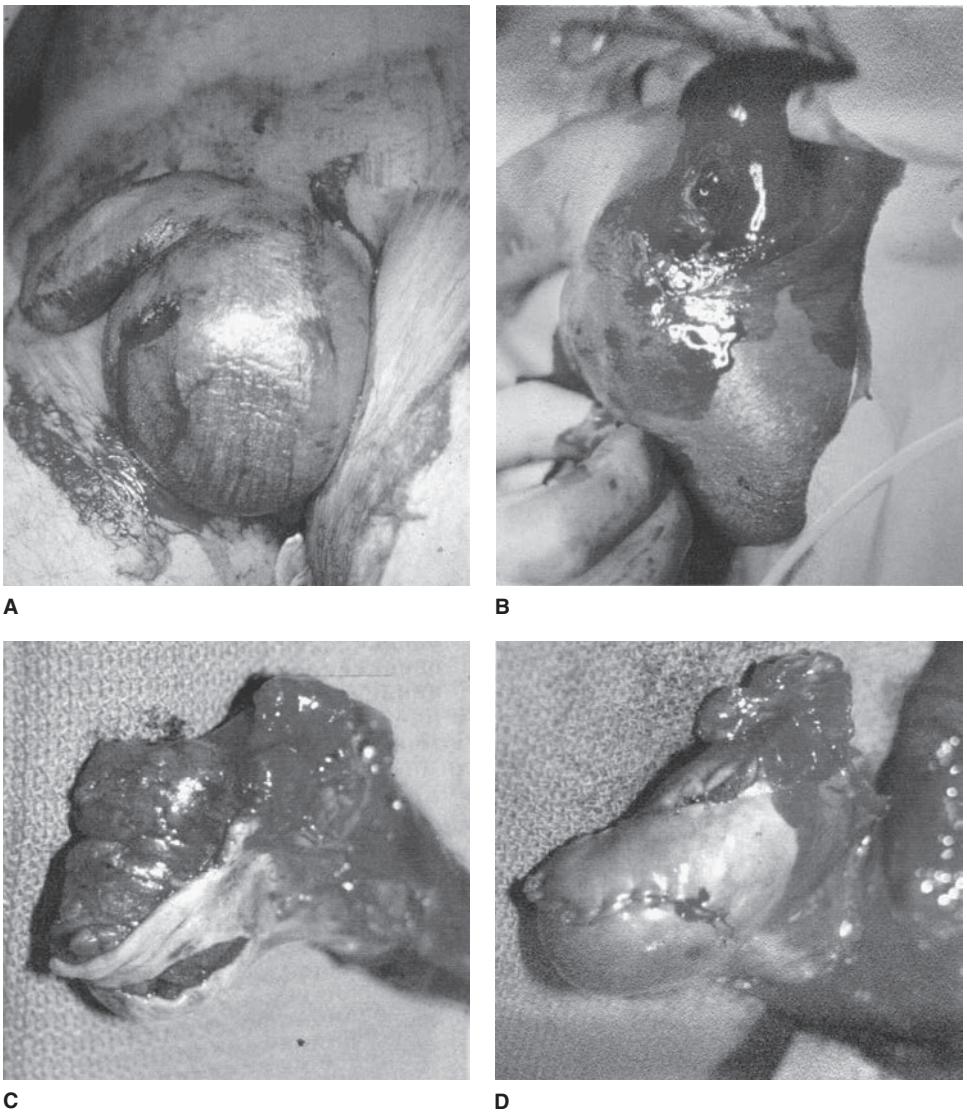


FIGURE 40-38 Scrotal exploration and testicular repair following gunshot wound to scrotum. (**A**) Entrance wound visible lateral to base of penis on left, exit just to the right of the median raphe; note marked left hemiscrotal swelling from hematoma. Surgical exploration is mandatory, and preoperative scrotal imaging is unnecessary. (**B**) Scrotal exploration performed through high oblique scrotal incision for optimal exposure of scrotal contents and possible extension to groin if further spermatic cord exposure proves necessary. Entrance into tunica vaginalis visible. Testis introduced out of scrotum on spermatic cord pedicle. (**C**) Appearance of left testis demonstrating complex laceration of tunica albuginea with extruded testicular parenchyma. (**D**) Appearance of testis following limited parenchymal debridement and reconstruction of tunica albuginea. Testis is then returned to scrotum following evacuation of hematoma and extensive irrigation; Penrose drain placed through inferior stab incision in left hemiscrotum (not shown).

the repair may require more time and blood loss than the patient can tolerate, or the degree of debridement needed may be unclear, as in cases with a high-velocity gunshot wound. Delaying definitive repair may be accomplished by inserting bilateral externalized ureteral stents. The pelvis can then be packed for bleeding by compressing the open bladder against the pubis. Placing an externalized suprapubic catheter (Malecot or Foley) within the injured bladder is also an option. If the catheter prevents tamponade of pelvic bleeding, it can be clamped temporarily and then reopened to drainage when the patient's coagulopathy is corrected. The use of damage control principles for complex penetrating pelvic trauma in the battlefield setting has been recently reported. In this series, 43% of patients had urologic injury, whereas 50% had major vascular injury. A 21% mortality rate in the first week after injury was reported, whereas 36% of patients with combined vascular and rectal injuries died.[98] A staged, multidisciplinary approach to management and reconstruction was shown to be valuable in this experience.

Injuries to the urethra and external genitalia can be temporarily managed with suprapubic catheterization or dressing

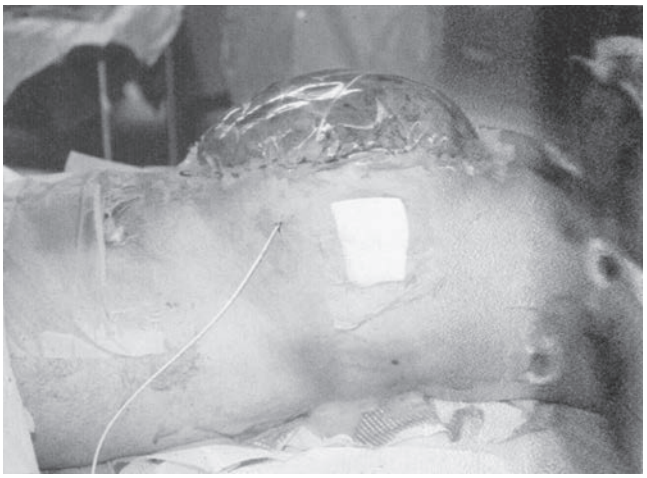


FIGURE 40-39 Patient managed with damage control laparotomy: gunshot wound to abdomen with injuries to small bowel, left iliac artery and vein, and left ureter. Patient was hemodynamically unstable following vascular repair, so ureteral injury was managed with damage control approach. Single-J stent was passed up proximal ureter at injury site and secured to end of ureter with silk tie; stent was externalized, exiting from left lower quadrant, as shown. Abdomen was closed with "Bogota bag" silo due to bowel and mesenteric edema. On return to operating room at 36 hours after injury, formal ureteral repair with psoas hitch and ureteroneocystostomy was performed.

applications pending the patient's return to surgery for definitive management.

The results of damage control management for urologic injuries in appropriately selected patients appear to be acceptable in terms of patient survival, renal salvage, and functional outcome.[99,100]

CONSULTATION AND INTERSERVICE INTERACTION

A specialty such as urology offers skills that are different from those of the general surgeon. These include endoscopic capability and familiarity with reconstruction of the urinary tract. The urology department should be informed of signs of urologic injury as early as possible, preferably from the emergency department. This allows the urologist to be involved in preoperative imaging and interpretation, later operative sequencing, and use of damage control strategies. The experience of large trauma centers in which an interested and capable urologic trauma team is involved often results in reduced rates of nephrectomy and improvements in other outcome measures.

TRANSGENDER PATIENTS

There are an estimated 1.4 million transgender adults in the United States, and this number is increasing.

The definition of *transgender* is as follows: of or relating to people who feel that their true nature does not match their sex at birth. Not everyone who is transgender prioritizes or desires procedures, such as hormone therapy and gender-affirming surgeries.

Eventually, these patients will need acute care in a trauma setting, requiring clinicians to obtain an inclusive history that takes into account possible gender-affirming surgery, managing hormone therapy, and other clinical issues.

Patients who underwent gender-affirming surgery have a unique anatomy that must be considered when they are under emergent and acute surgical care. The interpretation and understanding of laboratory values, surgical anatomy, and possible hormonal management may influence their clinical outcome.[101]

MEDICOLEGAL CONSIDERATIONS

Urologists frequently become involved in the management of injuries that were not diagnosed at the initial operation. These are often recognized later in a patient's clinical course, often after a complication (eg, urinary extravasation, bleeding, azotemia, sepsis) initiates further testing and imaging studies. In this case, it is important to communicate to the patient and family what is occurring and to document the events that have occurred in the medical record. It is important to educate patients and their families that traumatic injuries are complex and that certain complications are common and to be expected. Also, functional outcomes may be disappointing to patients, including urinary incontinence due to damage of urethral sphincter, neurogenic bladder that can occur after lumbosacral injuries and interventions, and erectile dysfunction and/or dyspareunia after pelvic or genital trauma.

Often, reconstructive procedures will occur after a long period of rehabilitation following trauma, and due to innumerous reasons, it is common for these patients to become lost to follow-up until complications arise due to retained ureteral stents (eg, urinary tract infection, stent calcification, obstruction, hematuria) or loss of urinary or renal function. With the advent of electronic medical records, special mobile phone apps, and patient safety culture implementation, ureteral stent track applications have been studied to decrease loss of follow-up and retained ureteral stent complications.[102]

Lastly, patients with major renal injuries may develop hypertension and/or proteinuria following certain injuries; this should be explained and documented.

CONCLUSION

There are very few prospective studies in the urologic literature, leaving levels of evidence at a suboptimal state for evidence-based medical practice. There is a broad international consensus, however, regarding the management of urologic injuries.[1-5] Evolvement in body imaging, endoscopic and laparoscopic approaches, endovascular stenting, and other radiologic and minimally invasive techniques has changed approaches to urologic trauma and selection of patients for operative versus nonoperative management. Over the past decade, the most significant change was the trend

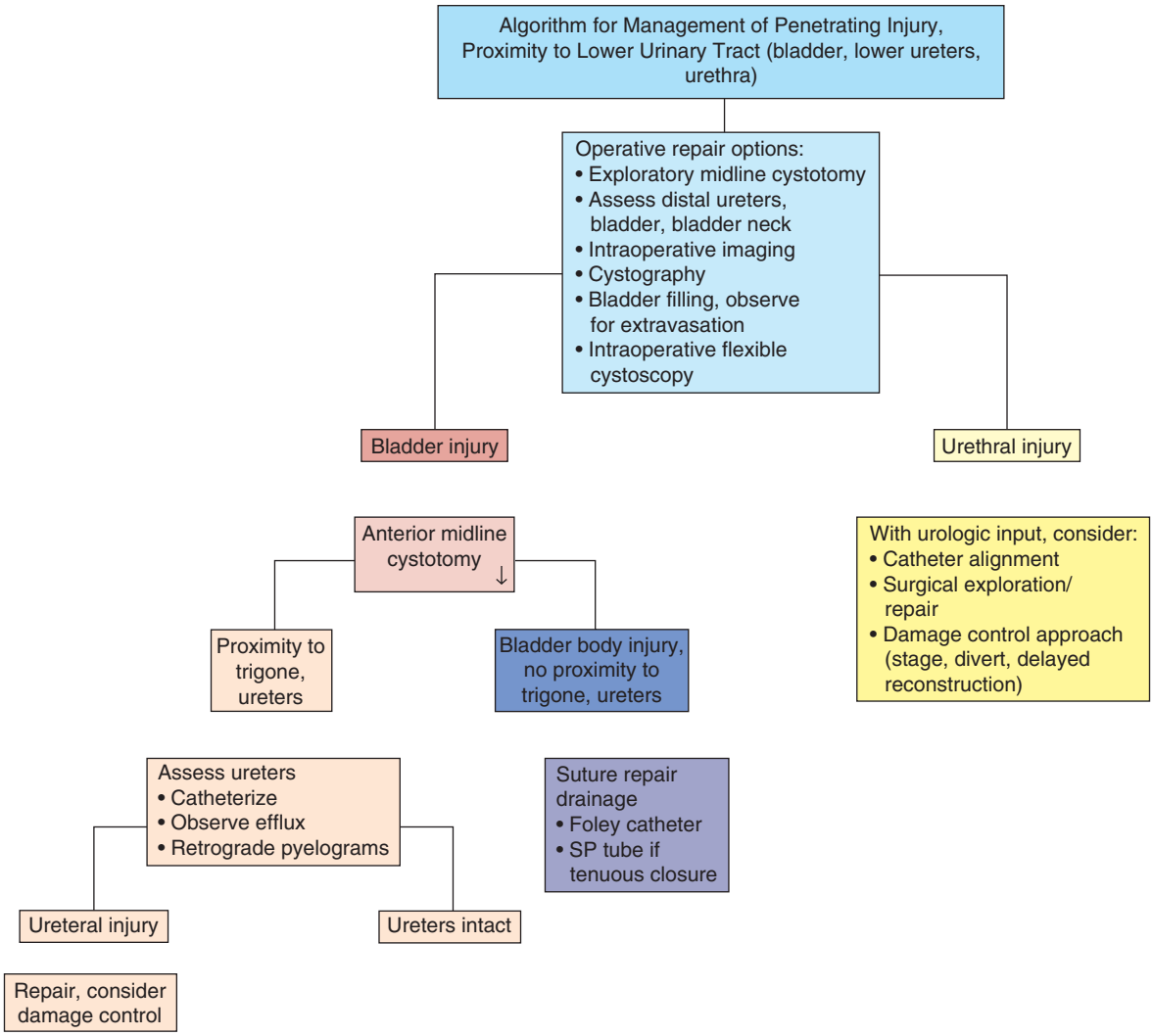


FIGURE 40-40 Algorithm for management of penetrating injury in proximity to lower urinary tract (bladder, lower ureters, urethra). SP, suprapubic.

toward conservative management whenever possible, especially when the kidney is involved; avoiding nephrectomies; and utilization of interventional radiologic vascular technology. Reconstructive surgeries can be achieved endoscopically in cases of urethral realignment and laparoscopically to treat bladder injuries. Further research will continue to impact the urologist's role and approach in dealing with genitourinary injury (Fig. 40-40).

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41

Trauma in Pregnancy

Brian C. Beldowicz • David Leshikar • Christine S. Cocanour

KEY POINTS

- Motor vehicle crashes account for 50% of trauma injuries during pregnancy and 82% of trauma-related fetal deaths, many of which are related to improper seat belt use.
- The disproportionate increase in plasma volume compared to red blood cell volume during pregnancy allows 35% of maternal blood to be lost before the mother exhibits clinical signs of shock.
- When supine, the gravid uterus compresses the inferior vena cava and causes a decrease in venous return to the heart and a drop in cardiac output. Placing the mother in the left lateral decubitus position restores venous flow and improves cardiac output.
- Imaging in the pregnant patient should be similar to the nonpregnant patient if life-threatening injuries are suspected.
- Pelvic fractures are the most common maternal injury that results in fetal death.
- Consider emergency cesarean delivery within 5 minutes in women who are beyond 20 weeks and have cardiopulmonary collapse because the gravid uterus may interfere with maternal hemodynamics.
- Intimate partner violence is the most common form of intentional trauma in pregnancy.

INTRODUCTION

Pregnancy imparts unique considerations in the evaluation and management of trauma patients. These include the following: epidemiologic distinctions from the general population; changes in anatomy and physiology that can alter injury presentation; concerns for fetal and placental injury; ramifications of trauma on the natural course of gestation; and poorly defined implications for the fetus from cumulative ionizing radiation employed in diagnostic imaging. Trauma during pregnancy has increased dramatically over the past 25 years.[1] Injury is now the leading cause of nonobstetrical maternal death in the United States,[2] and adverse fetal outcomes that have been correlated with trauma during pregnancy include preterm delivery, low birth weight, and fetal demise.[3]

Major trauma is associated with a 40% to 50% risk of fetal death, but the severity of trauma does not always predict the severity of injury to mother and fetus. Minor trauma occurs much more frequently but can still pose a significant risk to the fetus. A study of 5352 expectant mothers classified their injuries as major if hospital admission was required or minor if only emergency department evaluation occurred. They found that women in their first or second trimester with minor injury were 20% more likely to have a child with prematurity or low birth weight.[4]

EPIDEMIOLOGY

Trauma complicates an estimated 1 in 12 pregnancies, and 0.4% of pregnant women require hospitalization for their injuries.[5] Motor vehicle crashes account for 50% of all traumatic injuries during pregnancy and 82% of trauma-related fetal deaths.[6]

A major risk factor for injury is the improper use of a seat belt.[7] Over 40% of pregnant women involved in motor vehicle crashes did not receive counseling on appropriate seat belt use in one study.[8] Correct placement has the lap belt underneath the abdominal dome, with the belt contacting both anterior superior iliac spines and the pubic symphysis and decreasing the pressure transmitted across the uterus during a rapid deceleration. The shoulder harness should overlie the clavicle and run between the breasts.

Pregnancy makes women more prone to falls due to inherent changes such as increased joint laxity, weight gain, and dynamic postural stability.[9] It is estimated that one in four

women will fall at least once during their pregnancy.[10] Nearly 80% percent of pregnant women hospitalized after a fall were in their third trimester, and the most common injury was fracture in a lower extremity. Falls are associated with an 8-fold increase in placental abruption, a 4.4-fold increase in preterm labor, a 2.1-fold increase in fetal distress, and a 2.9-fold increase in fetal hypoxia.[11]

ANATOMIC AND PHYSIOLOGIC CHANGES UNIQUE TO PREGNANCY

Understanding the many anatomic and physiologic changes that occur with pregnancy will help the treating physician better manage the mother and the fetus (Table 41-1). The most obvious change seen in pregnancy is uterine growth. Figure 41-1 illustrates the uterine position throughout pregnancy.

At 12 weeks of gestation, the uterus becomes an intra-abdominal organ as it rises above the pelvic brim. At 20 weeks, the top of the uterus is at the level of the umbilicus, and at 36 weeks, it reaches the costal margin. Toward the end of pregnancy, the fetal head drops back into the pelvis, lowering the fundal height. This descent makes the fetal head more susceptible to injury, particularly if the mother suffers a pelvic fracture.[12] Uterine growth also shifts the maternal organs within the abdominal cavity, altering the typical findings on a physical examination and x-rays.

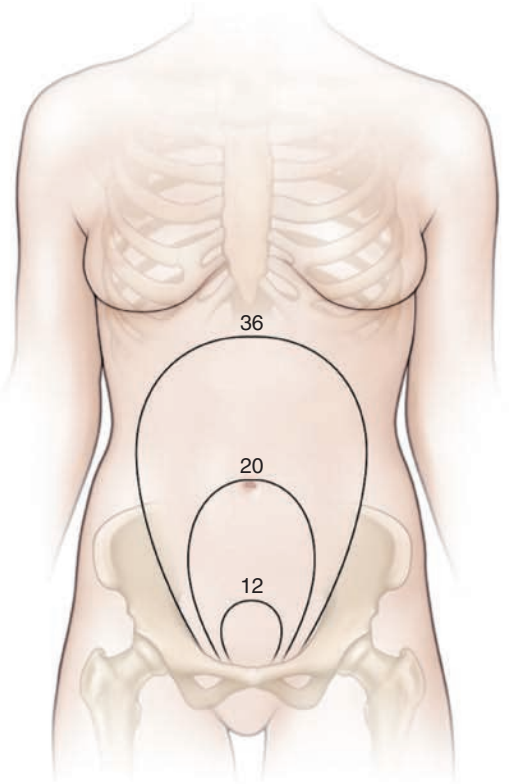

FIGURE 41-1 Representation of fundal height at 12, 20, and 36 weeks of pregnancy.

Cardiovascular System

The growing fetus requires a marked increase in oxygen delivery from the mother, and this is through an increase in

TABLE 41-1: Summary of Normal Physiologic Changes During Pregnancy

System	Change	Potential implication
Cardiovascular	↓ Peripheral vascular resistance, ↓ venous return, ↓ blood pressure (10–15 mm Hg)	Supine hypotensive syndrome (10–15 mm Hg)
Blood volume	↑ Plasma volume, ↑ RBC volume, ↑ WBC (20,000 cells/mm^3)	Physiologic hypervolemia may mask hypotension secondary to blood loss
Coagulation	Hypercoagulable; ↑ fibrinogen; ↑ factors VII, VIII, IX, X, XII; ↓ fibrinolysis	↑ Venous thromboembolism
Respiratory	↑ Subcostal angle (68–103°), ↑ chest circumference (5–7 cm), ↑ diaphragmatic excursion (1–2 cm), elevated diaphragm, ↑ tidal volume, ↑ minute ventilation, ↓ FRC, ↓ Pco_2, ↓ HCO_3	Alteration in FRC and lung volume, chronic compensated respiratory alkalosis
Gastrointestinal	↓ Motility, ↓ intestinal secretion, ↓ nutrient absorption, ↓ sphincter competency (progesterone)	Aspiration
Hepatobiliary	Organ displacement, ↑ gallbladder volume, ↓ albumin, ↑ AP, ↓ bilirubin (free), ↓ GGT	Clinical examination unreliable
Renal	↑ Glomerular filtration rate, ↑ renal plasma flow, ↑ creatinine clearance, ↓ serum creatinine, ↓ BUN	Hydronephrosis, hydroureter, bladder/urethral muscle tone
Endocrine	↑ Parathormone, ↑ calcitonin	↑ Calcium absorption
Musculoskeletal	Pelvic ligaments soften (relaxin, progesterone)	Pelvic widening, lordosis, shift in center of gravity

AP, alkaline phosphatase; BUN, blood urea nitrogen; FRC, functional residual capacity; GGT, γ-glutamyl transferase; RBC, red blood cell; WBC, white blood cell.

oxygen-carrying capacity, cardiac output, and minute ventilation. The maternal plasma volume begins to increase at 10 weeks of gestation and expands by 45% at full term, whereas the red blood cell volume only increases by 18% to 30%.[13] The disproportionate increase in plasma compared to red blood cells is protective because the mother will lose a large amount of blood during delivery. A higher plasma volume relative to red blood cell volume results in fewer lost red cells during hemorrhage. This physiologic anemia of pregnancy, with hematocrit falling to 32% to 34% at its nadir, occurs between the 30th and 34th weeks of gestation. Importantly, the extra blood volume may give a false sense of security for the treating traumatologist because approximately 35% of maternal blood volume may be lost before any clinical signs of shock are observed.

During pregnancy, the normal blood pressure drops by 5 to 15 mm Hg, and the resting heart rate increases by 10 to 15 bpm to increase cardiac output.[14] It is important not to attribute tachycardia or hypotension after trauma to physiologic changes associated with pregnancy without adequately evaluating for sources of hemorrhage. The uterine arteries supply the majority of blood to the fetus and are maximally dilated during pregnancy. Hypovolemia may cause vasoconstriction to divert blood to the mother's vital organs, and this will significantly decrease placental blood flow. Shunting of blood away from the placenta may manifest as fetal distress before systemic signs of hemorrhage are seen in the mother. As the growing uterus displaces the diaphragm, it pushes the heart to the left and upward along its long axis, producing an enlarged cardiac silhouette on chest radiographs. Most pregnant women will have some degree of benign pericardial effusion, while an electrocardiogram will show a slight left axis deviation from the altered position of the heart.[15]

During the middle of the second trimester, maternal position greatly affects cardiovascular physiology. While supine, the gravid uterus compresses the inferior vena cava, leading to a decrease in venous return to the heart and a drop in the cardiac output by as much as 25%. The *supine hypotensive syndrome* leads to dizziness, pallor, tachycardia, diaphoresis, nausea, and hypotension. The uterus also compresses the aorta, causing decreased blood flow to itself. With the uterine arteries maximally dilated during pregnancy, autonomic regulation is lost and blood flow is entirely dependent on maternal mean arterial pressure.[16] Placing the mother in the left lateral decubitus position restores venous flow and improves cardiac output.

Respiratory System

Both hormonal and mechanical changes in pregnancy contribute to increased minute ventilation. As the gravid uterus enlarges, it displaces the diaphragm superiorly approximately 4 cm, decreasing functional residual capacity. Elevated progesterone levels cause increased tidal volume and hyperventilation, which augment minute ventilation. It is common for a woman in her third trimester to have hypocapnia ($Paco_2$ of 30 mm Hg) and a compensated respiratory metabolic alkalosis. It is important to recognize that a pregnant patient with a $Paco_2$ of 35 to 40 mm Hg may be exhibiting signs of impending respiratory failure. The baseline hyperventilation of pregnancy also reduces the mother's ability to compensate for metabolic acidosis.

Renal System

To compensate for the increased blood volume in pregnancy, the renal plasma flow and glomerular filtration rate (GFR) both increase. Paired with hemodilution from a 50% increase in plasma volume, the serum creatinine concentration decreases by an average of 0.4 mg/dL. In pregnancy, a normal creatinine ranges from 0.4 to 0.8 mg/dL.[17] The increased GFR leads to excretion of other metabolic products that may exceed the tubular reabsorption capability and result in proteinuria and glucosuria. As the uterus grows, it can cause partial ureteral obstruction and physiologic hydronephrosis, particularly in the late stages of gestation. The right side is more affected than the left, due to the cushioning of the left ureter by the sigmoid colon.[15]

Coagulation System

Thrombosis in pregnancy is related to two components of Virchow's triad—stasis and hypercoagulability. Stasis occurs as the uterus grows and compresses venous outflow; however, the risk of venous thromboembolic events is the same throughout all stages of pregnancy, suggesting that hypercoagulability is the more significant factor. Increased concentrations of clotting factors cause hypercoagulability in pregnancy and may protect the mother from hemorrhage at childbirth or miscarriage. Pregnant women are at higher risk of thromboembolic events, with a three- to fourfold increase in arterial thromboembolism and four- to fivefold increase in venous thromboembolism.[18] Of interest, deep venous thrombosis represents 80% and pulmonary embolus 20% of venous events. In pregnancy, deep venous thrombosis is more likely to be proximal, massive, and on the left.[18]

Gastrointestinal System

Nausea and vomiting are common in pregnancy. Gastric emptying is unchanged, but pregnant women are more susceptible to aspiration with anesthetics. Alterations in liver function tests include a mild generalized decrease of aspartate transaminase, alanine transaminase, γ-glutamyl transpeptidase, and bilirubin.[19] Alkaline phosphatase activity increases, particularly in the third trimester, whereas serum albumin concentration decreases throughout pregnancy due to hemodilution and can be as low as 3.0 g/dL in the third trimester. Total-body albumin, however, is elevated.[20] As the fetus grows, maternal nutrition needs to increase, so a pregnant patient who is to remain nil per os (NPO [nothing by mouth]) for an extended period of time should receive supplemental nutrition with a goal of 36 kcal/kg/d.[21]

INITIAL ASSESSMENT AND MANAGEMENT

The first priority in treating an injured pregnant patient is the mother. Early and aggressive maternal resuscitation directly correlates with improved fetal outcomes.[22] Fetal mortality increases with the severity of maternal injuries and is more likely after direct fetoplacental injury, maternal shock, pelvic fracture, maternal injury to the brain, or hypoxia.[15] Preterm delivery is a significant risk, and a fetus delivered before 23 weeks is generally considered not viable.

Prehospital Care

The initial assessment of the mother in the field should be the same as with any injured patient. The National Center for Injury Prevention and Control recommends that pregnant women over 20 weeks gestation be transported to a center capable of timely and thorough evaluation and initial management of potentially serious injuries.[23] Evaluation at a designated trauma hospital has been associated with a lower incidence of preterm labor, low birth weight, and meconium at delivery.[24] Supplemental oxygen should target a maternal Sao_2 of greater than 95% to prevent fetal hypoxia.[25] Small improvements in maternal oxygen saturation have a greater effect on the fetus due to fetal hemoglobin's higher affinity for oxygen. Fluids and/or blood products should be given liberally to compensate for blood loss, and during transport, woman who are more than 20 weeks gravid should have a wedge placed beneath their right side or the uterus should be manually displaced to the patient's left to alleviate caval compression. If injuries to the spine are suspected, the wedge can be placed beneath a carefully secured backboard, allowing spine precautions to be maintained while offloading the inferior vena cava.

Regional and institutional protocols may differ, but pregnant trauma patients with a viable fetus (≥23 weeks) and without life- or limb-threatening injuries may be considered for transport directly to a labor and delivery unit with a trauma or orthopedic consultation placed from there, as appropriate. Pregnant patients should be transferred to an emergency department or trauma unit when the severity of injury is unknown, the viability or gestational age of the fetus is unknown, or injuries are known to be severe, or if the mother shows signs of instability.[25]

Primary Survey

The primary survey is performed as for all trauma patients. Lower doses of succinylcholine are required during rapid sequence intubation due to the lower concentration of pseudocholinesterase during pregnancy.[26] Once the patient's breathing has been addressed, two large-bore intravenous catheters are placed. If central venous access is necessary, the femoral vessels should be avoided due to compression of the inferior vena cava by the uterus, which can alter the distribution of fluids and medications.[27] The relative hypervolemia of the mother may mask the clinical signs of shock as previously noted. Resuscitation should be started once intravenous access is secured. Rh-negative blood should be used exclusively until maternal blood typing has been completed. Vasopressors should be reserved only for instances of intractable hypotension unresponsive to aggressive volume resuscitation due to their deleterious effect on uteroplacental perfusion.[25]

Secondary Survey and Maternal Assessment

The secondary survey should begin with a thorough history and physical examination, including a complete obstetric and prenatal history. A history of preterm labor, placental abruption, or placental previa should be ascertained.

Obstetric consultation provides assistance with the evaluation of any fetus deemed potentially viable by gestational age. Urgent consultation is recommended in women greater than or equal to 23 weeks pregnant who present with uterine contractions, suspected placental abruption, or uterine rupture. Measuring fundal height allows estimation of gestational age, particularly when the mother is unresponsive. A discrepancy between the fundal height and known gestational age can also be a sign of uterine trauma, specifically uterine rupture or hemorrhage. The pelvic examination should include a speculum exam, especially if a pelvic fracture is suspected and vaginal bleeding or hematuria is present.[22] If vaginal bleeding is present, placenta previa should be excluded by an ultrasound examination prior to a manual or speculum exam.

X-rays should be ordered as in any trauma evaluation. The uterus is shielded when not being directly evaluated. The focused assessment with sonography for trauma (FAST) examination is a safe and efficient modality to evaluate for intra-abdominal fluid. In a retrospective cohort of more than 2300 FAST exams, the sensitivity and specificity for the detection of free fluid or intra-abdominal injury in pregnant and nonpregnant trauma patients were similar.[28] In addition, fetal ultrasound assesses activity, gestational age, fetal position, and placental location. Fig. 41-2 provides a useful algorithm summarizing the initial evaluation and management of the pregnant trauma patient.

Radiographic Evaluation

The choice of imaging in the pregnant trauma patient should be similar to the nonpregnant patient if life-threatening injuries are suspected. The risk of a missed injury or delayed diagnosis in the fetus is much greater than the risk of radiation. As noted, FAST is a useful tool in the early evaluation of the mother and fetus but has limitations. Computed tomography (CT) remains the mainstay for evaluation of trauma patients and should be used if clinically indicated in the pregnant patient. Effort should be made to decrease unnecessary scans, reduce the overlap of body sections, and avoid multiple passes when possible.[29] Low-dose protocols often give little diagnostic information and should be avoided.

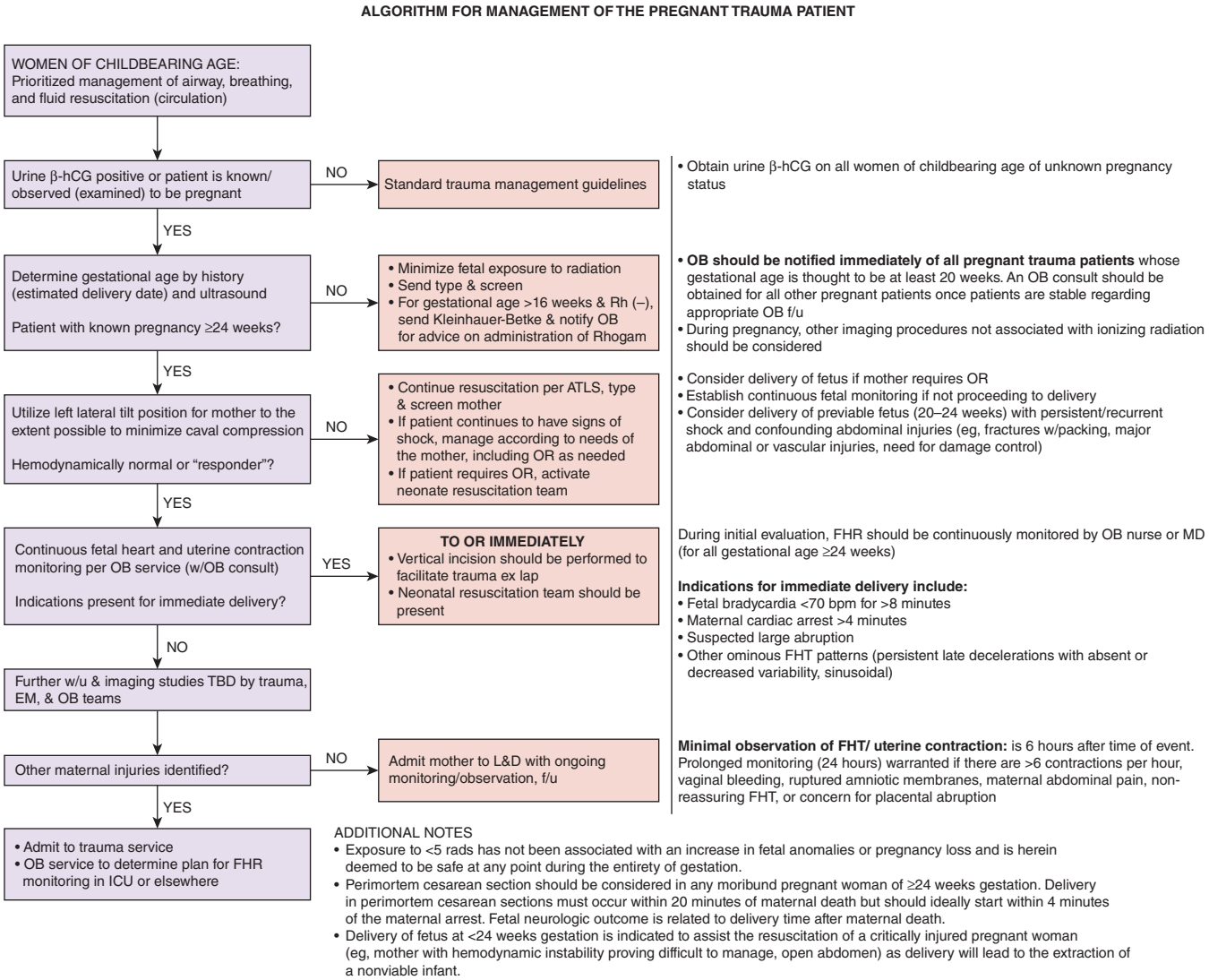

FIGURE 41-2 Algorithm for the management of the pregnant trauma patient. ATLS, Advanced Trauma Life Support; EM, emergency medicine; ex lap, exploratory laparotomy; FHR, fetal heart rate; FHT, fetal heart tone; f/u, follow-up; hCG, human chorionic gonadotropin; ICU, intensive care unit; L&D, labor and delivery; OB, obstetrician; OR, operating room; TBD, to be determined; w/u, workup.

Fetal risks from ionizing radiation include developmental delay, small head size, organ malformations, cancer, and death.[30] The average background radiation that a fetus is exposed to over 9 months of gestation is approximately 1 mGy (0.1 rad). Table 41-2 shows the estimated radiation exposure to the fetus during many common radiographic procedures used to assess injured patients. As expected, radiation exposure increases substantially when the fetus is in the field of view. Radiation dose and gestational age are the two main variables in the risk assessment of the fetus. Data from survivors of the atomic bomb blasts indicate that the risk is greatest between 8 and 15 weeks gestation, the period of fetal organogenesis and neuronal development.[31] There is no proven risk when less than 8 weeks or greater than 25 weeks gestation. Guidelines from the American College of Obstetricians and Gynecologists (ACOG) for imaging during pregnancy and lactation emphasize that exposure to less than 5 rad (50 mGy) has not been associated with fetal abnormalities or pregnancy loss. The complete guidelines are shown in Table 41-3. Radiation exposure is similar whether using 16-slice or 64-slice CT scanners.[32]

EVALUATION OF THE FETAL-PLACENTAL UNIT

Fetal death after maternal trauma results from direct fetal injury, placental abruption, maternal hypotension leading to fetal malperfusion, or trauma-induced preterm delivery. Findings on clinical examination that should raise concern of fetal-placental injury include vaginal bleeding, ruptured membranes, a bulging perineum, presence of contractions, and an abnormal fetal heart rate or rhythm. Vaginal bleeding

TABLE 41-2: Estimated Fetal Exposure from Common Radiologic Procedures

Procedure	Fetal exposure
Chest XR (AP and lateral)	0.00002–0.00007 rad[a]
Abdominal XR (single AP view)	0.1 rad[a]
Pelvis XR (single AP view)	0.25–1.5 rad[a]
Hip XR (single view)	0.2 rad[a]
Complete spine XR series	0.37rad[a]
CT pulmonary angiogram	0.003–0.022 rad[b]
CT abdomen/pelvis	0.47–0.55 rad[b]
CT trauma protocol (head, neck, chest, abdomen, pelvis)	1.01–1.12 rad[b]

AP, anteroposterior; CT, computed tomography; XR, x-ray.

[a]Adapted with permission from Desai P, Suk M. Orthopedic trauma in pregnancy. *Am J Orthop.* 2007;36:E162. Copyright © 2007 Quadrant HealthCom Inc. All rights reserved.
[b]Adapted from Keleranta A, Makela T, Kaasalainen T, Kortesniemi M. Fetal radiation dose in three common CT examinations during pregnancy: Monte Carlo study. *Phys Med.* 2017;43:199-206.

is abnormal before labor and can indicate placental abruption, early labor, premature cervical dilation, or placenta previa, where the placenta's position covers a portion of the cervical os. Rupture of the amniotic sac should be suspected when there is cloudy white or green discharge, which increases the risk of infection. A ruptured amniotic sac can also lead to prolapse of the umbilical cord, which is an obstetric emergency requiring immediate cesarean section.

TABLE 41-3: American College of Obstetricians and Gynecologists Guidelines for Diagnostic Imaging During Pregnancy and Lactation

- Ultrasonography and magnetic resonance imaging (MRI) are not associated with risk and are the imaging techniques of choice for the pregnant patient, but they should be used prudently and only when use is expected to answer a relevant clinical question or otherwise provide medical benefit to the patient.
- With few exceptions, radiation exposure through radiography, computed tomography (CT) scan, or nuclear medicine imaging techniques is at a dose much lower than the exposure associated with fetal harm. If these techniques are necessary in addition to ultrasonography or MRI or are more readily available for the diagnosis in question, they should not be withheld from a pregnant patient.
- The use of gadolinium contrast with MRI should be limited; it may be used as a contrast agent in a pregnant woman only if it significantly improves diagnostic performance and is expected to improve fetal or maternal outcome.
- Breastfeeding should not be interrupted after gadolinium administration.

Source: Reproduced with permission from Committee on Obstetric Practice. ACOG Committee Opinion No. 723: guidelines for diagnostic imaging during pregnancy and lactation. *Obstet Gynecol.* 2017;130(4):e210-e216.

Fetomaternal Hemorrhage

Suspicion of blunt uterine trauma raises concern for transmission of fetal hemoglobin into the maternal circulation. This poses significant risk in Rh-negative mothers who may then form antibodies to the red blood cells of present and future pregnancies. The Kleihauer-Betke (KB) test is used to detect the presence of fetal blood in maternal circulation. Acid is placed on a maternal blood smear to dissolve adult hemoglobin, but fetal hemoglobin, if present, persists and can be quantified. A 300-mcg dose of Rh immune globulin should be administered for every 30 mL of fetal blood estimated in maternal circulation and should be administered within 72 hours of the time of injury.

At least one study has challenged the utility of KB testing in trauma, citing higher rates of positive tests in low-risk pregnancies compared to a population of pregnant trauma patients.[33] Still, the most current recommendations from the Eastern Association for the Surgery of Trauma (EAST) support routine KB testing for all pregnant trauma patients greater than 12 weeks gestation, regardless of Rh status.[34] Some institutions elect to give a 300-mcg dose of Rh immune globulin to all Rh-negative pregnant trauma patients and add additional doses if indicated based on KB test results. ACOG, however, cites that the principal utility of the KB test is in its ability to restrict the use of Rh immune globulin to only those mothers who need it and to identify patients for whom a single 300-mcg dose is insufficient.[35]

Fetal Monitoring/Nonstress Testing

Fetal heart tones can be detected as early as 12 weeks of gestation and are normally between 110 and 160 bpm. Initially, fetal hypoxia manifests as tachycardia, but as the arterial oxygen content decreases, the fetus becomes bradycardic and any rate less than 120 bpm should be recognized as fetal distress. EAST recommends at least 6 hours of fetal heart monitoring for all pregnant trauma patients greater than 20 weeks gestation.[34] Fetal bradycardia or prolonged or late heart rate decelerations during nonstress testing should prompt a trial of resuscitative measures including intravenous fluids, oxygen supplementation, and maternal repositioning. Failure of such interventions to resolve fetal stress should prompt consideration of fetal-placental injury and the possible need for emergent obstetrical intervention. All trauma patients greater than or equal to 23 weeks pregnant who have been observed for at least 6 hours after injury should undergo a formal obstetrical ultrasound prior to discharge.

Placental Abruption

Placental abruption occurs when the relatively inelastic placenta separates from the elastic uterine myometrium, compromising oxygen transfer to the fetus. Clinical examination is often unreliable in diagnosing placental abruption, and vaginal bleeding is present in only 35% of patients.[36] Placental

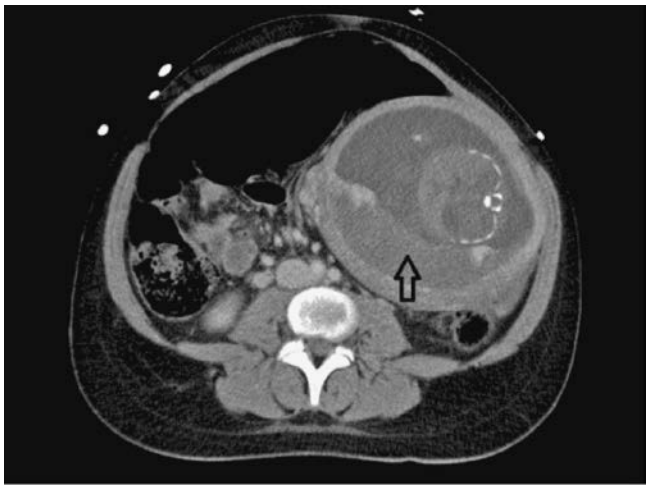

FIGURE 41-3 Example of placental abruption on computed tomography.

abruption may not present until as many as 24 hours after injury but is typically accompanied by at least one contraction every 10 minutes within 4 hours of presentation.[3,37] Monitoring of patients without such contractions can be limited to 6 hours, but should continue for a minimum of 24 hours if frequent contractions, vaginal bleeding, ruptured amniotic membranes, maternal abdominal pain, or nonreassuring fetal heart rate patterns are present. CT is a reliable means of diagnosing and grading placental abruption and should be considered in pregnant patients with abdominal ecchymoses, pain, or tenderness.[38] Fig. 41-3 shows an example of placental abruption on CT. One study in pregnant trauma patients suggested that emergent delivery at the first sign of fetal distress improved fetal outcomes in third-trimester mothers with 50% or less placental surface area contrast enhancement on CT, indicating moderate to severe placental abruption.[39]

MANAGEMENT OF SPECIFIC INJURIES DURING PREGNANCY

Thoracic Trauma

Injuries to the thorax are treated the same in pregnant and nonpregnant patients. It is important to remember that the gravid uterus elevates the diaphragm approximately 4 cm when performing procedures such as a tube thoracostomy. The insertion point for a chest tube should be one to two rib spaces above the usual fifth intercostal space.[22]

Blunt Abdominal Trauma

Similar to thoracic injuries, blunt abdominal injuries are treated the same in pregnant patients as in nonpregnant patients. The primary focus should be on the evaluation and rapid treatment of the mother because maternal shock is associated with an 80% fetal mortality.[5] Injury to a solid organ can be safely observed if the mother remains hemodynamically stable; however, if signs of shock or infection develop, early operative intervention is indicated, as a delay in treatment leads to worse fetal outcomes.

The most devastating fetal injuries following blunt trauma include placental abruption and uterine rupture. Abruption is thought to complicate 1% to 6% of minor injuries and up to 50% of major injuries.[3,15] Uterine rupture is rare, occurring in less than 1% of pregnant trauma patients, and is usually associated with direct impact with substantial force.[15] A previously scarred uterus is more likely to rupture, and the risk increases with gestational age as the uterus grows and becomes an abdominal organ. The increased blood flow that accompanies advanced pregnancy makes rupture a severe complication, which can lead to extensive hemorrhage.[22] The extent of uterine damage is typically not apparent clinically and must be evaluated with operative exploration. Injuries to the skull and brain of the fetus are uncommon but are more likely to occur if there is a maternal pelvic fracture and when the fetal head is engaged within the pelvis.

Orthopedic Trauma

Maternal orthopedic injuries are common, and one study found that 6% of 1067 pregnant women had an orthopedic injury, and this led to an increased risk of preterm birth, placental abruption, and fetal mortality.[40]

Pelvic Fractures

A pelvic fracture is the most common maternal injury that results in fetal death. In a study of 101 pregnant women with pelvic fractures, the fetal mortality rate was 35%.[41] Causes of death included direct fetal injury (20%), placental abruption (32%), and maternal shock (36%). Maternal mortality in the same study was 9% as dilated retroperitoneal vessels place the mother at high risk of bleeding from pelvic fractures. Angiography and embolization of these bleeding vessels often deliver a higher dose of radiation than is usually considered safe, and these women should be counseled on the risk. Operative fixation of both pelvic and acetabular fractures is generally considered safe during pregnancy. Most women can safely attempt vaginal delivery after a pelvic fracture, even in the third trimester. Relative indications for a cesarean delivery include fractures of the pubic rami adjacent to the urethra or bladder, severe lateral compression fractures, and acute fractures of the pelvis with marked displacement.[41]

Penetrating Injuries

In contrast to blunt trauma, maternal mortality is more favorable after penetrating injury as the gravid uterus protects the mother's vital organs from injury.[42] Conversely, fetal mortality is increased and has been reported to be as high as 73%.[43] The thick uterus, however, provides protection to the mother and fetus from low-velocity stab injuries. Although

gunshot wounds to the uterus are much more likely to cause fetal injury, visceral injuries are less likely when the entry site is anterior and below the uterine fundus.[44] Potential abdominal injuries from thoracoabdominal wounds should be suspected at a higher level than normal given displacement by the uterus. Exploration should be considered for any gunshot or stab wound to the upper abdomen, particularly because peritoneal irritation is blunted during pregnancy.[15] Diagnostic laparoscopy is reasonable in the hemodynamically stable patient.

Neurologic Injuries

A moderate or severe traumatic brain injury is associated with an adverse fetal outcome. Ikossi et al[45] found that a Glasgow Coma Scale score less than or equal to 8 was a significant risk factor for death of the fetus. The range of hyperventilation used for traumatic brain injury should be decreased in a pregnant patient because this causes a mechanical reduction in venous return and, therefore, cardiac output, which can have negative effects on the fetus. Hypothermia and mannitol should also be avoided, but the use of hypertonic saline has no known deleterious effects.[46]

Thermal Injuries

Burn injury in the pregnant patient presents a unique management challenge because it causes increased capillary permeability and can lead to rapid fluid loss and hypovolemia. Fetal survival is dependent on fetal age and the extent of maternal injury. It is estimated that once the total body surface area of a burn exceeds 40%, the risk of mortality for the mother and fetus approaches 100%.[47] Early delivery is recommended in the third trimester if the mother has extensive burns.[48]

Wound care in the pregnant patient should limit the use of silver sulfadiazine because it can potentially cause kernicterus in the newborn; however, one author has shown successful use without any adverse effects.[49]

Preparing for the Operating Room

If operative intervention is indicated, it is crucial to operate immediately because a delay in treatment puts the fetus further at risk. Fetal viability should be documented using fetal heart tones or ultrasound pre- and postoperatively, and on-site obstetric consultation is recommended. Intubation can be difficult in the pregnant patient due to physiologic oropharyngeal edema, and inhalation anesthesia has a more rapid onset in pregnant patients, requiring an adjustment in dose.

Presently, there are no conclusive data or expert recommendations regarding the use of tranexamic acid (TXA) for traumatic hemorrhage in pregnant patients. TXA is classified as a pregnancy category B drug. Although a 2011 meta-analysis indicated its use was associated with less blood loss following vaginal and cesarean section deliveries and reduction in postpartum blood transfusion requirements, its safety and efficacy have not been examined in the pregnant trauma population. The CRASH-2 trial did not specifically exclude pregnant patients; however, the study design left the decision of TXA administration to the discretion of prescribing physicians. Females composed only 16% of the study population, and no mention of pregnancy status was included in the study's analysis.[50] A 1993 retrospective study of 256 patients treated with TXA for a bleeding disorder during pregnancy demonstrated no increased risk of thromboembolic complications compared to an untreated cohort of pregnant patients with similar bleeding disorders.[51]

During a laparotomy for trauma, the goals should be the same as in a nonpregnant patient—control of hemorrhage and contamination. The uterus should be retracted inferiorly while exploring the other abdominal organs. Once completed, the uterus is thoroughly inspected for any injuries, particularly in the setting of penetrating trauma. Small lacerations to the uterus can be repaired with chromic sutures. If the laceration extends into the uterus and the fetus is at a survivable age, then a cesarean delivery should be performed. Such delivery can also be considered if the gravid uterus prevents adequate exploration of the mother. After significant blunt trauma, a uterine rupture may occur. Attempts should be made to repair the uterus primarily if possible, but if there is extensive uterine damage or hemorrhage cannot be controlled, hysterectomy should be performed. Fetal survival after extensive uterine damage is rare.[22] An injury to the gastrointestinal tract increases the risk of loss of the pregnancy, and the abdomen should be irrigated extensively in addition to the administration of perioperative antibiotics.[22]

Cesarean Section Following Injury

Indications for cesarean delivery after trauma are vague and must be made on an individual basis. The three possible goals when a caesarean section is indicated include save the mother, save both the mother and child, and save only the child. Cesarean section prior to 23 weeks usually results in fetal death and should only be done to allow successful resuscitation of the mother.[37] After 23 weeks, cesarean section should be performed if fetal distress outweighs the risk of prematurity or if the uterus prevents repair of maternal injuries during laparotomy.[52] A cesarean delivery is not automatically indicated with exploratory laparotomy for trauma, but it is important to have the obstetrical team available for any unforeseen problems.

Cardiopulmonary Resuscitation in Pregnancy

The America Heart Association (AHA) recommends four key interventions to prevent a possible cardiopulmonary arrest in the critically injured pregnant patient as follows: (1) place the patient in full left lateral position to relieve aortocaval compression; (2) give 100% oxygen; (3) establish intravenous catheter access above the diaphragm; and (4) assess for hypotension, defined as systolic blood pressure less than 100 mm Hg, which

reduces placental perfusion.[53] If cardiopulmonary resuscitation (CPR) is required, the mother should be taken out of a left lateral tilt because this reduces the efficacy of chest compressions.[22] To relieve aortocaval compression, manual left lateral and upward displacement of the uterus can be done by a member of the treating team. Chest compressions should be performed slightly higher than normal due to the upward displacement of the thoracic and abdominal structures by the uterus.[54] In one retrospective study, three of seven pregnant patients who achieved return of spontaneous circulation following CPR sustained hepatic lacerations that required surgical or endovascular intervention.[55] The rate of compressions and the use of defibrillation and cardiac medications remain unchanged from the recommendations in a nonpregnant patient.

The AHA and ACOG both recommend cesarean delivery be considered within 4 minutes of maternal cardiopulmonary collapse if there is no return of spontaneous circulation.[52,56,57] Delivery within 5 minutes in women beyond 20 weeks is encouraged in order to facilitate maternal resuscitation. This recommendation is based on the assumption that the compression of the vena cava and aorta by the gravid uterus may interfere with maternal hemodynamics. In one case review, 12 of 18 patients had sudden and often profound improvement in hemodynamic status with return of blood pressure and pulse immediately after a cesarean delivery,[58] changing the concept of delivery from a postmortem cesarean to perimortem cesarean. The 4-minute time frame advocated for postmortem cesarean delivery is often unmet, yet neonatal survival is still likely if delivery occurs within 10 or even 15 minutes of arrest.[57] Earlier cesarean delivery theoretically benefits both mother and neonate by minimizing ischemic neurologic damage.

Emergency cesarean delivery should be performed at the location of arrest. Time should not be taken to check for fetal viability or to move to an operating room. CPR should continue during the procedure in case maternal circulation is restored. Gestational age can quickly be estimated, as the uterine fundus should be two finger breadths above the umbilicus at 24 weeks.[59] To perform delivery, a large midline skin incision from xiphoid to pubis is made, and the uterus is opened with a vertical incision through the upper portion. The surgeon should then place his or her hand between the fetal head and pubic symphysis to deliver the baby and placenta.[15] The uterus should be closed with a single layer of running chromic suture followed by quick closure of the fascia. CPR should continue well after the procedure, and if successful, the mother should be transported to the intensive care unit for recovery. A summary of the guidelines for perimortem cesarean section is provided in Table 41-4.

TABLE 41-4: Predictors of Successful Fetal Outcome Following Postmortem Cesarean Section

1. Duration of gestation.
   Fetal viability generally is defined as 26–28 weeks gestation. This corresponds to a fundal height of approximately 26–28 cm above the pubis and/or uterus, halfway between the umbilicus and costal margin. At this age, the fetus, under optimal conditions, has a 40%–70% estimated chance of survival without major handicap; therefore, cesarean section is indicated shortly after maternal death.
2. Time between maternal death and delivery.
   <5 min, excellent
   5–10 min, good
   10–15 min, fair
   15–20 min, poor
   20–25 min, unlikely

Procedure

1. Establish viability.
2. Complete the CPR sequence.
3. Make a vertical midline incision through the abdominal layers into the uterus.
4. Remove the fetus from the uterine cavity, clamp the cord, and hand the neonate to appropriate personnel for resuscitation.
5. Remove the placenta.
6. Continue CPR and assess for maternal signs of life; maternal survival is still possible after the uterus has been emptied and the supine hypotension syndrome has been resolved.

CPR, cardiopulmonary resuscitation.

Sources: Adapted with permission from Macmillan Publishers Ltd: Higgins SO. Perinatal protocol: trauma in pregnancy *J Perinatol.* 1988;8:288; and adapted with permission from Seldin BS, Burkes TJ. Complete maternal and fetal recovery after prolonged cardiac arrest. *Ann Emerg Med.* 1988;17:346 © Elsevier.

INTIMATE PARTNER VIOLENCE IN PREGNANCY

Intimate partner violence (IPV) is the most common form of intentional trauma in pregnancy and poses a significant risk to both the mother and fetus. ACOG believes that reported rates of IPV during pregnancy do not reflect the magnitude of the problem in our society.[60] Voluntarily reported rates of IPV may be as low as 3.2%,[61] but other estimates range as high as 28%.[62] Risk factors associated with IPV include maternal or intimate partner substance abuse, low socioeconomic status, low maternal education level, unintended pregnancy, history of domestic violence prior to pregnancy, history of witnessed violence as a child, and unmarried status.[44] The National Violent Death Reporting System demonstrates that 54.3% of pregnancy-associated suicides and 45.3% of pregnancy-associated homicides were directly attributable to IPV.[63]

A comprehensive meta-analysis on the impact of IPV on adverse infant outcomes identified twice the rate of preterm birth and low birth weight and a nearly 40% increased risk that a baby would be born small for gestational age.[64]

The prevalence of domestic violence involving pregnant women that results in serious injury has been estimated to be between 10% and 30%.[65] The abdomen is the most common target associated with assault, increasing the likelihood of an intra-abdominal injury and fetal morbidity and mortality. Women who deliver during a hospitalization for assault are at higher risk for preterm birth, maternal death, fetal death, and

TABLE 41-5: Sample Intimate Partner Violence Screening Questions

While providing privacy, screen for intimate partner violence during new patient visits, annual examinations, initial prenatal visits, each trimester of pregnancy, and the postpartum checkup.

Framing Statement

"We've started talking to all of our patients about safe and healthy relationships because it can have such a large impact on your health."*

Confidentiality

"Before we get started, I want you to know that everything here is confidential, meaning that I won't talk to anyone else about what is said unless you tell me that…(insert the laws in your state about what is necessary to disclose)."*

Sample Questions

"Has your current partner ever threatened you or made you feel afraid?"

(Threatened to hurt you or your children if you did or did not do something, controlled who you talked to or where you went, or gone into rages)†

"Has your partner ever hit, choked, or physically hurt you?"

("Hurt" includes being hit, slapped, kicked, bitten, pushed, or shoved.)†

For women of reproductive age:

"Has your partner ever forced you to do something sexually that you did not want to do, or refused your request to use condoms?"*

"Does your partner support your decision about when or if you want to become pregnant?"*

"Has your partner ever tampered with your birth control or tried to get you pregnant when you didn't want to be?"*

For women with disabilities:

"Has your partner prevented you from using a wheelchair, cane, respirator, or other assistive device?"‡

"Has your partner refused to help you with an important personal need such as taking your medicine, getting to the bathroom, getting out of bed, bathing, getting dressed, or getting food or drink or threatened not to help you with these personal needs?"‡

*Family Violence Prevention Fund. Reproductive health and partner violence guidelines: an integrated response to intimate partner violence and reproductive coercion. San Francisco (CA): FVPF; 2010. Available at: http://www.futureswithoutviolence.org/userfiles/file/HealthCare/Repro_Guide.pdf. Retrieved October 12, 2011. Modified and reprinted with permission.

†Family Violence Prevention Fund. National consensus guidelines on identifying and responding to domestic violence victimization in health care settings. San Francisco (CA): FVPF; 2004. Available at: http://www.futureswithoutviolence.org/userfiles/file/Consensus.pdf. Retrieved October 12, 2011. Modified and reprinted with permission.

‡Center for Research on Women with Disabilities. Development of the abuse assessment screen-disability (AAS-D). In: Violence against women with physical disabilities: final report submitted to the Centers for Disease Control and Prevention. Houston (TX): Baylor College of Medicine; 2002. p. II-1–II-16. Available at http://www.bcm.edu/crowd/index.cfm?pmid=2137. Retrieved October 18, 2011. Modified and reprinted with permission.

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uterine rupture, whereas those who are discharged and deliver during a subsequent hospitalization remain at increased risk for placental abruption and hemorrhage compared to mothers with no history of having been assaulted.[66]

The impact of IPV persists long beyond delivery for both mother and child. Nearly half of mothers subjected to IPV during pregnancy meet criteria for postpartum depression.[67] Adverse effects of IPV may persist for years and could preclude effective parenting. IPV victims are at risk for functional disorders such as irritable bowel syndrome, fibromyalgia, and chronic pain syndromes, as well as psychosocial issues such as substance abuse, eating and sleeping disorders, physical inactivity, poor self-esteem, and posttraumatic stress disorder.[68] Mothers who suffer IPV during pregnancy remain at risk of homicide for up to 24 months after delivery compared to the general population.[69]

Victims of IPV are at risk for delayed and inadequate perinatal care, and the interaction with medical providers during a presentation for trauma offers a fleeting opportunity to engage expectant mothers in initiating and continuing appropriate care during pregnancy. ACOG recommends universal screening of all pregnant women regarding IPV, and any pregnant woman who presents with a vague or suspicious history of trauma should raise a concern for assault.[70] Warning signs include frequent office or emergency department visits, depression, substance abuse, discrepancy between injuries and given history, and a partner's insistence on being present for the interview and examination.[12] A guide for IPV screening is provided in Table 41-5.[70]

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42

Trauma Damage Control

Chad G. Ball • David V. Feliciano

KEY POINTS

- *Damage control* is a US Navy term defined as the "capacity of a ship to absorb damage and maintain mission integrity."
- An abbreviated laparotomy with diffuse intra-abdominal packing in patients with significant intra-abdominal injuries and an intraoperative coagulopathy was first described by H. Harlan Stone, MD, at Grady Memorial Hospital, Atlanta, Georgia, in 1983.
- In the modern era, agreed-upon indications for a damage control laparotomy include temperature less than 34°C; arterial pH less than 7.2; an international normalized ratio, prothrombin time, or partial thromboplastin time greater than 1.5 times normal; or a clinically observed coagulopathy in the pre- or intraoperative setting.
- The early coagulopathy of trauma, as demonstrated on laboratory testing, is present upon admission in over 25% of injured patients with a base deficit greater than –6.
- Examples of thoracic damage control include cross-clamping the hilum, a hilar twist after division of the inferior pulmonary ligament, and pulmonotomy with selective vascular ligation.
- Examples of abdominal damage control include perihepatic packing, suture repair and packing of grade I or II splenic injuries, and Whipple procedure or distal pancreatectomy delayed to reoperation.
- Examples of vascular damage control anywhere in the body include insertion of temporary intraluminal shunts, ligation of selected major veins, and early fasciotomy in high-risk patients with injuries to the extremities.
- Abdominal compartment syndrome is defined as a sustained intra-abdominal pressure greater than 20 mm Hg that is associated with new organ dysfunction or failure.
- The temporary silo over open abdomen technique was initially described by Oswaldo Borraez G. at the San Juan de Dios Hospital in Bogota, Colombia, in 1984.
- Indications for an emergent return to the operating room after a damage control laparotomy performed in a patient with blunt trauma include a normothermic patient who is bleeding greater than 2 units of packed red blood cells per hour or the development of an abdominal compartment syndrome with ongoing blood loss.
- Maintenance of the peritoneal domain and prevention of adherence of the viscera to the underside of the abdominal wall are critical to allow progressive closure of the linea alba after an open abdomen.

HISTORY AND DEFINITION

Damage control is a US Navy term defined as "the capacity of a ship to absorb damage and maintain mission integrity."[1] When applied to surgery and critically ill patients, abdominal damage control surgery (DCS) incorporates fundamental tenets that include the following: (1) stopping surgical hemorrhage; (2) controlling gastrointestinal spillage; (3) inserting surgical packs; and (4) applying a temporary abdominal closure. This truncated operation is then followed by immediate transfer to the intensive care unit with subsequent rewarming, hemodynamic stabilization, correction of coagulopathy, and general supportive care for stunned organs. A return to the operating room then occurs 6 to 48 hours later for definitive repairs, exploration for missed injuries, insertion of a feeding tube, and primary fascial closure, if possible. In essence, a typical operative sequence is interrupted by completing only the most crucial aspects during the first operation.

The history of the innovation of DCS mirrors that of the history of trauma surgery in general (Table 42-1). The DCS concept represents one of the most significant innovations in the history of surgery and has now influenced the practices of a large number of surgical subspecialties and radiology.[2] DCS was first initiated due to a growing appreciation from the surgical community in the 1970s to 1990s that major

TABLE 42-1: Author History of the Innovation of Damage Control

Era 1: 1902–1983: Staged laparotomy for packing of liver and/or diffuse intra-abdominal hemorrhage	
Pringle	Calne
Halsted	Eldering
Lucas	Kashuk
Ledgerwood	Feliciano
Walt	Stone
Era 2: 1984–1993: Abbreviated laparotomy and development of rapid damage control techniques	
Stone	Burch
Carmona	Rotondo
Ivatury	Schwab
Feliciano	Moore
Era 3: 1994–2000: The damage control laparotomy learning curve and extrapolation beyond the peritoneal cavity	
Hirshberg	Feliciano
Coburn	Scalea
Borraez	
Era 4: 1990s–2018: Assessment of damage control techniques	

definitive operations were often futile after injured patients developed hypothermia, metabolic acidosis, and a coagulopathy. In addition, rapid conservative operative techniques were associated with improved outcomes in select major trauma patients. Although the adaption of the contemporary *damage control* term to the field of traumatology can be specifically credited to Rotondo, Schwab, McGonigal, and colleagues from the University of Pennsylvania in 1993,[3] its dominant principles are more accurately rooted in the 1975 presentation to the American Association for the Surgery of Trauma by Drs. Charles E. Lucas and Anna Ledgerwood.[4] More directly, they expanded on Pringle's 1908 report of perihepatic packing by describing a small series of patients who underwent laparotomy pad packing of major hepatic injuries.[4] Although many surgeons in the early 20th century placed packs directly into hepatic tears to facilitate hemostasis (sometimes with the parenchyma loosely approximated over top in order to create a "tampon"), this updated technique involved reconstituting the liver as a solid structure by packing around the organ in a compressive fashion. This concept was reiterated shortly thereafter by Calne et al,[5] as well as Feliciano et al in 1981[6] and 1986.[7] Despite these small series outlining the success of perihepatic packing, the visionary extrapolation of this principle to patients with multiple concurrent life-threatening injuries and major coagulopathy was not published until 1983.[8] At that time, Harlan Stone retrospectively described 31 injured patients who developed major bleeding diatheses during an emergency laparotomy at Grady Memorial Hospital in Atlanta, Georgia.[8] Of these, 17 patients were managed with damage control principles of arresting surgical hemorrhage and abbreviating the operative intervention. This led to the survival of 11 patients who were predicted to have a lethal coagulopathy. Once DCS was born, it was quickly marketed into other disciplines that included, but were not limited to, neck,[9] vascular,[10] orthopedic,[11] thoracic,[12] and military injuries[13] because it was clear that the DCS approach leads to improved survival in patients who are approaching intraoperative physiologic exhaustion after sustaining blunt and penetrating injures.[14]

INDICATIONS FOR DAMAGE CONTROL

Despite its obvious utility, the widespread propagation of DCS throughout the trauma community has led to overutilization of the technique. More specifically, multiply injured patients who are *not* approaching physiologic exhaustion are not uncommonly exposed to the potential risks associated with an open abdomen and the DCS process. As a result, the pertinent question remains: Who truly requires DCS? The succinct response is: a patient who is more likely to die from an uncorrected shock state than from failure to complete organ repairs. Depending on the center, these "metabolic cripples" encompass 3% to 8% of all *severely* injured patients (penetrating vs blunt; military vs civilian). In essence, they continue to suffer the sequelae of tissue shock that is manifest as *persistent* hypothermia, *persistent* metabolic acidosis, and nonmechanical (ie, nonsurgical) bleeding. Although this description appears straightforward, opinions regarding the specific indications for DCS remain debated across both global trauma centers (Table 42-2) and an assorted quality of peer-reviewed literature.[15] Further, clinical recognition of these patients based on prehospital reports or on initial glance upon arrival in the trauma bay is notoriously inaccurate.[15] The DCS triggers with most agreement, however, include core temperature less than 34°C, arterial pH less than 7.2, and laboratory-confirmed (international normalized ratio, prothrombin time, and/or partial thromboplastin time >1.5 times normal) or clinically observed coagulopathy in the pre- or intraoperative setting. Other triggers include the administration of greater than 10 units of packed red blood cells (RBCs), requirement for a resuscitative thoracotomy in the emergency department, or identification of a juxtahepatic venous injury or devascularized or destroyed pancreas, duodenum, or pancreaticoduodenal complex during operation.[15] It must be emphasized, however, that not all patients with initial physiologic deficits will need DCS. With rapid arrest of hemorrhage, as well as ongoing resuscitation, some patients will dramatically improve in all parameters on repeated intraoperative blood gases and evaluations. In other words, patients with *unresolved* metabolic failure despite the arrest of hemorrhage via suture, resection, or packing remain our primary targets for DCS. In addition, it should be stated that patients with multiple intra-abdominal injuries do not always develop metabolic failure.

The natural extension of DCS has been damage control *resuscitation* (DCR). This concept includes DCS, but also the early transfusion of blood products, reduced administration

TABLE 42-2: Published Candidate Indications for Use of Damage Control Surgery in Adult Civilian Trauma Patients Who Were Rated to be Appropriate by the Majority of Respondents

Indication
Degree of physiologic insult in the pre- or intraoperative setting
Persistent systolic BP <90 mm Hg or a successfully resuscitated cardiac arrest during transport to hospital
Persistent systolic BP <90 mm Hg in the preoperative setting or during operation
Preoperative core body temperature <34°C, arterial pH <7.2, or INR/PT >1.5 times normal (with or without a concomitant PTT >1.5 times normal)
Core body temperature <34°C and arterial pH <7.2 at the beginning of operation
Persistent core body temperature <34°C or persistent arterial pH <7.2 during operation
INR/PT and PTT >1.5 times normal during operation
Clinically observed coagulopathy during operation
Core body temperature <34°, arterial pH <7.2, and laboratory-confirmed (INR/PT and/or PTT >1.5 times normal) or clinically observed coagulopathy in the preoperative setting, at the beginning of operation, or during the conduct of operation
Estimated blood loss and amount or type of resuscitation provided
Estimated blood loss >4 L in the operating room
>10 units of PRBCs were administered to the patient in the preoperative or preoperative and intraoperative settings
Injury pattern identified during operation
An expanding and difficult-to-access pelvic hematoma
A juxtahepatic venous injury
An abdominal vascular injury and at least one major associated abdominal solid or hollow organ injury
A proximal (ie, Fullen zone I or II) superior mesenteric artery injury
Devascularization or destruction of the pancreas, duodenum, or pancreaticoduodenal complex with involvement of the ampulla/proximal pancreatic duct and/or distal CBD
Multiple blunt or penetrating injuries spanning across more than one anatomic region or body cavity that each require surgery with or without angioembolization

BP, blood pressure; CBD, common bile duct; INR, international normalized ratio; PRBC, packed red blood cell; PT, prothrombin time; PTT, partial thromboplastin time.

of crystalloid fluids, permissive hypotension in selected populations, and immediate control of hemorrhage (whether operative or angiographic). In summary, DCR is a structured, mobile intervention that can be delivered to a critically ill patient in any location (emergency department, interventional radiology suite, operating room, and/or intensive care unit). Basic principles include arresting hemorrhage, restoring blood volume, and correcting hypothermia, acidosis, and coagulopathy.

MASSIVE TRANSFUSION

Although the traditional definition of a massive transfusion is 10 or more units of packed RBCs within a 24-hour period, this has been modified with regard to both the amount of blood products and the associated time interval.[16-18] The 3% and 8% of civilian and military casualties, respectively, who require a massive transfusion have high mortality rates as well (27%–51%).[18] Furthermore, the early coagulopathy of trauma as demonstrated on laboratory testing is a well-recognized entity that is present upon admission in over 25% of injured patients with a base deficit greater than –6.[19] In addition, although coagulopathy was historically viewed as a byproduct of resuscitation, hemodilution, and hypothermia (Table 42-3), the "bloody vicious cycle" is now understood to be significantly more complex.[20] Tissue trauma, shock, hemodilution, hypothermia, acidemia, and inflammation all play key trigger roles in the acute coagulopathy of trauma shock.[19] The improved understanding of interrelationships and recognition of these six key initiators of a coagulopathy support the modern use of massive transfusion protocols (MTPs). In brief, a modern MTP aims to approximate delivery of close to a 1:1:1 ratio of RBCs to fresh frozen plasma to platelets.[20] By addressing the early coagulopathy of trauma, MTPs have been shown to improve mortality in multiply injured populations.[21,22] Although the specific structure of MTPs varies slightly from center to center, they are all approximations of the late George Sheldon's fresh whole blood resuscitation principles from 1975.[22,23] It should also be noted that reasonable scientific concern remains with regard to the apparent improvement in survival associated with MTPs.[24] The possibility of a strong survival bias (ie, surviving long enough to receive the most RBC units) is significant.[24]

Additional benefits of a formal MTP include earlier administration of blood products during resuscitation, improved efficiency of blood banking, decreased utilization of total blood products during a hospital stay, and significant economic savings.[25] Another major benefit is avoiding excess administration of crystalloid fluids.[26,27] This reduction in crystalloid volume during resuscitation minimizes multiple associated

TABLE 42-3: Maneuvers to Prevent or Reverse Hypothermia During Damage Control Operations

Increase operating room temperature to >85°F (29.4°C)
Infuse blood products through a warming device such as a Level 1 fluid warmer
Cover patient's head with a turban or warming device
Cover body parts out of the operative field with a warming device
Employ a heated mattress pad underneath the patient
Irrigate nasogastric and thoracostomy tubes with warm saline during laparotomy
Irrigate open pericardial cavity, pleural cavities, and peritoneal cavity during simultaneous sternotomy or thoracotomy and laparotomy

side effects, including reperfusion injury, increased leukocyte adhesion and inflammation, associated acidosis, and resultant acute respiratory distress syndrome, systemic inflammatory response syndrome, and multiorgan failure.[26-29] In addition, excess administration of crystalloid fluids continues to be an obstacle to obtaining early definitive fascial closure secondary to edema of the abdominal viscera and wall.[30,31]

Interestingly, the evolution of MTP and DCR now appears to be focused upon individual variances between patients in regard to their precise responses to a significant injury. More specifically, using various point-of-care measures such as thromboelastography will assist in providing a significantly more granular analysis of which resuscitation factors a given critically injured patient requires. This would include both the type and amount of a given blood product, in addition to the immediate response to a particular intervention.

In summary, if the initiation of an MTP is accurately based on rapid recognition of physiologic exhaustion secondary to persistent hemorrhage, then it also typically acts as a trigger for the entire damage control process. It must be reemphasized, however, that some severely injured patients will improve dramatically with an MTP and reverse their physiologic derangements to an extent that allows the surgeon to complete operative repairs and closure of the abdominal incision at the first operation.

THORACIC DAMAGE CONTROL TECHNIQUES

Lung (See Chapter 28)

Exsanguinating hemorrhage from the lung is most rapidly controlled by the application of a DeBakey aortic clamp to the hilum.[32] Similarly, a Duval lung clamp is an excellent instrument for more peripheral and readily visible hemorrhage. Another technique that may be helpful in either the emergency department or the operating room includes twisting the hilum after division of the inferior pulmonary ligament to kink the major hilar vessels. This technique is rarely used when manual hand/finger or clamp control works. It is also challenging given the anatomic limitations associated with the inferior pulmonary ligament.

When the site of the blood loss has been a stab wound deep into the pulmonary parenchyma or a gunshot wound completely through a lobe, the technique of pulmonotomy (sometimes referred to a tractotomy) is employed.[33-35] This maneuver requires division of the pulmonary parenchyma between noncrushing vascular clamps or via the use of a linear stapler/cutter to expose injured vessels. After selective ligation of bleeding vessels, the pulmonary parenchyma can be closed using a continuous 2-0 absorbable suture, with reinforcement material added to the staple line, as needed.

Heart (See Chapter 30)

Although compression with a finger is always recommended as the quickest and safest way to control hemorrhage from a small wound or rupture of a ventricle in the emergency department or operating room, application of 6-mm-wide skin staples (Auto Suture 35 W; United States Surgical Corporation, Norwalk, CT) may also be employed.[36,37] It should also be noted that some wounds such as large-caliber holes or injuries proximate to the coronary arteries are not appropriate for stapling. If desired, formal cardiac repair with Teflon pledgets and sutures may be completed over the staples or as they are sequentially removed in the operating room. Larger wounds or ruptures of a ventricle in patients surviving by virtue of a tamponade may be controlled by insertion of a Foley balloon catheter into the hole.[38] With the balloon inflated and extremely gentle traction applied to the catheter, Teflon-pledgeted sutures can then be passed through the ventricle from side to side over the balloon. The thin wall of the right ventricle puts the inflated balloon at significant risk of puncture as each suture is placed. Pushing the catheter and balloon into the ventricle with each bite of the suture will mitigate this complication, although blood loss may be significant. An alternative option is to use a cuffed endotracheal tube. This provides the advantage of increased manual stability while sewing. It must be emphasized, however, that excessive traction on either device can enlarge the initial laceration and create a nonrecoverable scenario. Clamping of the balloon catheter (or endotracheal tube) is also essential to prevent massive blood loss via the lumen of the tube, unless the surgeon wishes to use direct venous access through the catheter by connecting it to intravenous tubing (ie, for medication boluses).

With a longitudinal perforation or significant rupture of a ventricle, the time-honored technique of inflow occlusion is useful in avoiding cardiopulmonary bypass.[39] Curved aortic or angled vascular clamps are first applied to the superior and inferior vena cavae. As the heartbeat slows, horizontal mattress sutures can be inserted rapidly on either side of the defect and then crossed to control hemorrhage if this is still occurring. A continuous suture is then placed to close the defect, and before it is tied down, air is vented out of the elevated ventricle by releasing the clamps on the cavae. The surgeon should ensure internal paddles and all resuscitation tools are readily available, as successful restoration of a perfusing rhythm is unlikely after 3 minutes of occlusion.[40] For injuries to more vulnerable or friable myocardium, manually compressing the right atrium will result in partial inflow occlusion necessary to repair the ventricle.[41] Injuries occupying the lateral wall of the left ventricle, left pulmonary veins, left atrial appendage, or left pulmonary artery are accessed through a "cupping" maneuver to lift the ventricles out from the pericardial well. This should be done fairly slowly to avoid any rapid hypotension by running the fingers of the right hand between the pericardium and the right ventricle and then sweeping them posteriorly and cephalad. The hand cups the apex of the left ventricle, which is subsequently elevated anteriorly and out of the pericardial well. Meanwhile, placing several pericardial retraction sutures is important in order to maximize exposure. Finally, wounds to the posterior heart are particularly challenging. To access these sites, the heart must often be "flipped up" prior to suture repair.

Close communication with the anesthesiologist and rapid surgical technique are essential given the typical induction of complete cardiac arrest after positioning the heart for repair. As a result, intermittent restoration of the heart back into its natural position may be required for cardiac relief during prolonged repairs. Caution in technique is also essential if cardiac massage is required to promote prograde perfusion. An open hand methodology prevents punctures of the heart with excited fingers.

VASCULAR DAMAGE CONTROL TECHNIQUES

General

Although it is clear that stopping ongoing hemorrhage is the most crucial aspect of DCS, *vascular* damage control has been traditionally limited to vessel ligation. More recently, however, balloon catheter tamponade and temporary intravascular shunts (TIVS) have increased in popularity. The impressive utility of balloon catheters for tamponade of exsanguinating hemorrhage has a long history dating back more than 50 years.[2] Although this technique was originally described for esophageal varices,[42,43] it was quickly extended to patients with traumatic vascular and solid organ injuries. Since the initial treatment of an iliac arteriovenous lesion in 1960,[33] balloon catheters have also been used for cardiac,[38,44] aortic,[45] pelvic,[46] neck (carotid, vertebral, and jugular),[47,48] abdominal vascular,[49] hepatic,[50] subclavian,[51] vertebral,[48] and facial trauma.[52] Although this technique was originally intended as an intraoperative endovascular tool, it has since been employed as an emergency department maneuver with the balloon being placed outside of the lumen of the injured vessel such as at the base of the skull.[53,54]

Modern indications for this damage control technique are limited. This is primarily because routine methods for controlling hemorrhage, such as direct pressure, are typically successful. As a result, indications for catheter tamponade currently include the following: (1) inaccessible (or difficult to access) major vascular injuries; (2) large cardiac injuries; and (3) deep solid organ parenchymal hemorrhage (liver and lung). Of interest, the type of balloon catheter (Foley, Fogarty, Blakemore, or Penrose over red rubber Robinson), as well as the duration of indwelling, can vary significantly.

In conclusion, balloon catheter tamponade is a valuable tool for damage control of exsanguinating hemorrhage when direct pressure fails or tourniquets are not applicable. It can be employed in multiple anatomic regions and for variable patterns of injury. Prolonged catheter placement for maintenance of hemostasis is particularly useful for central hepatic gunshot injuries.[54]

TIVS are intraluminal synthetic conduits that offer nonpermanent maintenance of arterial inflow and/or venous outflow.[55] As a result, they are frequently life- and limb-saving when patient physiology is hostile. By bridging a damaged vessel and maintaining blood flow, they address both acute hemorrhage and critical warm ischemia of distal organs and limbs.

Although Eger et al[56] are commonly credited for pioneering the use of TIVS in modern vascular trauma, this technique was initially employed by Nobel Prize winner Alexis Carrel in animal experiments.[57] The first documented use in humans occurred in 1915 when Tuffier employed paraffin-coated silver tubes to bridge injured arteries.[58] This technique evolved from glass to plastic conduits in World War II[59] and continues to vary both in structure and material among today's surgeons.[60]

Modern indications for TIVS include the following: (1) replantation; (2) open extremity fractures with concurrent extensive soft tissue loss and arterial injury (Gustilo IIIC); (3) peripheral vascular damage control; (4) truncal vascular damage control; and (5) temporary stabilization prior to transport.[60,61] Although use of TIVS for military and civilian indications is increasing, the optimal shunt material, dwell time, and anticoagulation requirements remain poorly studied.[60] It can be noted, however, that TIVSs are remarkably durable and rarely clot unless they are too small (diameter), kink because of inappropriate length, and/or are placed in an extremity without appropriate (or shunted) venous outflow (venous hypertension leads to arterial thrombosis).[60,62] Although the literature contains many case series and practical experience using TIVS, one of the most dramatic examples of their utility is the near complete disappearance of limb loss that often followed ligation of the common and/or external iliac arteries in the past.[61] More specifically, despite similar injury and patient characteristics, the improvement in amputation rate from 47% to 0% in 22 patients with the introduction of TIVS at a high-volume penetrating trauma center is remarkable.[61] Further advantages included an observed reduction in fasciotomies (93% to 43%) and extra-anatomic bypasses (six bypasses to zero bypasses).[61]

Of interest, the majority of TIVS (64%) in a series from the National Trauma Data Bank (NTDB) were used in patients injured via a blunt mechanism.[63] Although the kinetic force of a motor vehicle crash (MVC) or MVC-pedestrian collision can be tremendous, TIVS has most often been discussed in the context of extremity damage control for gunshot wounds in patients with hostile physiology.[63] This recent NTDB analysis, however, indicated that most extremity TIVSs are actually placed for blunt vascular trauma associated with extensive orthopedic and/or soft tissue injuries (74%). This is usually a temporizing maneuver to provide distal flow to a limb while orthopedic injuries are assessed and fixated. In the patients who did not undergo TIVS for fractures and soft tissue defects, shunting was employed as an extremity damage control technique in patients with hemodynamic instability and severe base deficits (26%).[63] These patients displayed a much lower level of subsequent amputation when compared to patients after blunt trauma with concurrent fractures and soft tissue trauma who were not shunted.

In addition to using TIVS in patients with blunt injuries, the report from the NTDB also indicates that this technique has been used unevenly across a wide range of hospitals.[63] Of 111 trauma centers employing TIVS, only six used five or more shunts throughout the study period. Additionally, only three centers employed more than 10 shunts.

In conclusion, TIVS appear to be useful in any scenario with a major vascular injury and concurrent hostile patient physiology. This includes patients with blunt trauma with concurrent severe fractures and/or soft tissue injuries in the extremities.

Abdominal Arteries (See Chapter 38)

In any patient with multiple upper abdominal visceral and vascular injuries, a significant injury to the celiac axis is treated with ligation. An injury to the renal artery is also best treated with ligation and nephrectomy in the presence of a palpably normal contralateral kidney and multiple associated injuries. Depending on the comfort of the surgeon, segmental loss of the supra- or infrarenal aorta can be addressed by inserting a large TIVS (ie, thoracostomy tube) or rapid insertion of a 12-, 14-, or 16-mm woven Dacron or polytetrafluoroethylene (PTFE) interposition graft. The superior mesenteric artery or common or external iliac artery is smaller in young trauma patients, and an intraluminal Argyle, Javid, or Pruitt-Inahara TIVS may be rapidly inserted.[64] Should ligation be required for injuries to the common or external iliac artery, a rapid ipsilateral two-incision, four-compartment below-knee fasciotomy may prevent myonecrosis with its associated renal and septic problems in the short term. If the patient survives, the patient should undergo extra-anatomic revascularization within 6 hours.

When life-threatening arterial hemorrhage from either a blunt pelvic fracture or a penetrating wound occurs in the deep pelvis and cannot be controlled by packing, several approaches can be considered. Insertion of a Fogarty balloon into the internal iliac artery beyond a proximal tie on the side of the hemorrhage is followed by sequential advancement and inflation until the hemorrhage stops.[65] Another technique is to ligate the internal iliac arteries. Unfortunately, gluteal necrosis may occur after ligation of bilateral arteries and occasionally after ligation of a single internal iliac artery in patients with crush injuries (see Chapter 39).

Abdominal Veins (See Chapter 38)

Ligation is generally the treatment of choice in the context of major injuries to the common or external iliac veins, infrarenal inferior vena cava (IVC), superior mesenteric vein, or portal vein in a patient with profound shock.[66-70] After ligation of the infrarenal IVC, bilateral four-compartment below-knee fasciotomies should be performed immediately if the anterior compartment pressure is greater than 25 to 35 mm Hg, depending on the patient's hemodynamic stability. Bilateral thigh fasciotomies are also often necessary within 48 hours of ligation. When there are large defects in the sacrum or pelvic sidewall involving numerous pelvic veins or in the paravertebral area, a number of approaches are available to rapidly control hemorrhage. Packing the missile track with several vaginal packs, inserting fibrin glue, or placing a Foley catheter with a 30-mL balloon inflated at the site of hemorrhage may be effective. Placing packs outside of the blast cavity in the deep pelvis or paravertebral area often fails to control hemorrhage in the patient who develops a coagulopathy. Bleeding from presacral veins can be controlled by inserting sterile tacks directly into the visible defect or by suturing a free piece of omentum into the obvious area of perforation.

Extremity

Similar to other areas of the body, damage control operations on an extremity are appropriate when exsanguination has caused physiologic exhaustion, when multisystem injuries have occurred that require emergent treatment (skull, chest, abdomen), or when instability of an open fracture precludes formal repair of the associated vascular injury (as described earlier).[71]

After rapid control of hemorrhage, the best TIVSs for extremity injuries tend to be Argyle, Javid, or Pruitt-Inahara shunts, as previously noted.[72,73] The latter shunt possesses the added benefit of inflatable balloons on either end so that tying the shunt in place is not necessary. Its T-port also allows the infusion of heparin, vasodilators, or contrast for arteriography in the postoperative period.

Although ligation of major extremity venous injuries is often well tolerated in stable patients,[74,75] those requiring damage control may suffer severe sequelae.[71] These include compartment syndrome below the level of ligation in the lower extremity and excessive hemorrhage from soft tissue injuries and fasciotomy sites. Even if a compartment syndrome does not occur immediately, reperfusion injury during resuscitation will typically cause the syndrome to develop. This highlights the rationale for the rapid insertion of TIVS to ensure adequate venous outflow. Short 24F to 28F segments of thoracostomy tubes are excellent conduits for popliteal, femoral, or common femoral veins.

It should be reemphasized that if there has been any delay to restoration of venous flow (ie, long prehospital time, difficulty in controlling hemorrhage) or ligation of the major venous structures is required, ipsilateral four-compartment below-knee fasciotomies are recommended as part of the damage control procedure.

ABDOMINAL DAMAGE CONTROL TECHNQUES

Liver (See Chapter 33)

At rest, the liver has a blood supply approximating 1500 mL/min. In severe injuries, this can make stopping hemorrhage an extremely challenging task for not only a general surgeon, but also an experienced trauma surgeon.[76-81] As a fundamental philosophy of damage control, the techniques applied to major hepatic injuries with ongoing life-threatening hemorrhage should remain as limited and direct as possible.

Although the published history of hepatic trauma is replete with descriptions of various technical maneuvers ordered in a hierarchical scheme (extensive hepatorraphy, hepatotomy with selective vascular ligation, resectional debridement with

selective vascular ligation, rapid resectional debridement),[82-85] very few are commonly relevant in the context of modern trauma care and damage control. More specifically, damage control packing of hepatic hemorrhage controls the vast majority of ongoing bleeding in critically ill patients. The goal of packing is to reconstitute the liver as closely to its normal anatomic state as possible. It may be helpful to leave the falciform ligament intact to provide a medial wall against which to improve packing pressure (especially in blunt trauma to the right lobe). Although persistent perihepatic packing is required in less than 10% of operative liver injuries (ie, with an associated open abdomen), it remains an underappreciated skill. More specifically, it should be performed in an intentional and cautious manner to avoid further injury to the liver. The surgeon's nondominant hand is responsible for reconstituting the liver, followed by the placement of pads in strategic locations by the dominant hand. It should be noted that it is possible to pack the liver so tightly that it impedes venous return to the heart through the IVC and potentiates hypotension. Also, there has also been significant historical concern for interference from the radiopaque markers associated with laparotomy pads in subsequent angiography/angioembolization. Advances in digital subtraction technology and CT and the experience of interventional radiologists have made this less of a problem.

If hemorrhage continues in any scenario, an early Pringle maneuver (clamping of the porta hepatis with a vascular clamp) is mandated as both a diagnostic and potentially therapeutic technique. If bleeding continues despite application of a Pringle clamp, an injury to the retrohepatic vena cava or a hepatic vein is likely. It should be noted that not all critically injured patients in physiologic extremis tolerate extended Pringle maneuvers to the same extent as patients with hepatic tumors undergoing elective hepatic resection (40- to 60-minute upper limit). If the liver hemorrhage responds to packing but continues when unpacking is completed, the patient should be repacked and transferred to the intensive care unit with an open abdomen once damage control of concurrent injuries is complete. Covering the parenchymal or capsular injury with a plastic sheet or sterile x-ray cassette before inserting packs avoids adherence at the time of unpacking.

If control of the hepatic hemorrhage is dependent upon maintenance of a Pringle maneuver despite packing, the surgeon should call for an experienced assistant, mobilize the injured lobe, and see if there is an injury to the retrohepatic vena cava or a hepatic vein. On occasion, a patient with an injury to one of these structures may require total vascular exclusion (TVE) of the liver—that is, complete occlusion of the infrahepatic IVC, suprahepatic IVC, and porta hepatis (Pringle maneuver), as well as an infrarenal aortic cross-clamp within the abdomen. If TVE is pursued without concurrent clamping of the infrarenal abdominal aorta, the patient will often arrest due to a lack of coronary artery perfusion. Technically savvy surgeons prefer to obtain suprahepatic IVC control within the abdomen in patients with a normal length of IVC inferior to the diaphragm. Another option is to access the IVC as it enters the heart within the pericardium. This 2-cm length of IVC is easily accessible by opening the pericardial sac after dividing the diaphragm or after performing a median sternotomy. Veno-veno bypass is a damage control option that may require the assistance of a surgeon with experience in hepatic transplantation. If an atriocaval shunt is contemplated, two experienced surgical teams (one for the thorax and one for the abdomen) are essential. The decision to pursue this damage control shunt must be made early in the exploration process because they rarely result in patient salvage.

Similar to damage control for selected vascular injuries (see earlier discussion), ongoing hepatic hemorrhage that is not easily treated by direct repair and/or is challenging to control with external compression (eg, central hepatic gunshot wounds) may be controlled by intrahepatic balloon catheter tamponade.[85,86] Although techniques vary, a Foley, Fogarty, or inflated Penrose drain over a red rubber catheter is the most useful in controlling hemorrhage from a deep lobar stab or missile track. Inflation of the balloon is performed at different levels of the track until hemorrhage is controlled. Removal of the balloon catheter is performed at a reoperation when the patient's metabolic failure has been corrected. This technique is excellent at stopping hemorrhage and is associated with a reasonably high survival rate given the challenges of these injuries.

Pancreas (See Chapter 36)

Damage control for the pancreas itself is limited to packing of the injured gland if a coagulopathy is present. Ductal transections to the left of the mesenteric vessels that do not involve the splenic vessels are packed and drained, with the distal pancreatectomy delayed until reoperation (Fig. 42-1). Major parenchymal or ductal injuries in the head or neck of the pancreas are also packed or drained, once hemorrhage from the gland or underlying vasculature is controlled. Similar to a distal pancreatectomy, a required pancreatoduodenectomy is reserved for reoperation, as well.[87]

The dominant associated life-threatening scenario for peripancreatic injuries involves hemorrhage from the portal and superior mesenteric veins (and, occasionally, the IVC) (Fig. 42-1). Although these veins can be ligated as part of damage control, repair with 5-0 or 6-0 Prolene sutures is appropriate if rapid vascular control can be obtained. Another damage control option would be insertion of a small chest tube as a TIVS. Central injuries to the splenic vein are ligated, whereas those near the hilum mandate a splenectomy.

Spleen (See Chapter 34)

With American Association for the Surgery of Trauma (AAST) Organ Injury Scale (OIS) grade III, IV, or V injuries, splenectomy remains the safest choice when damage control is necessary.[88,89] Bulk ligation of the hilum via either suture ligature or a vascular stapler (linear or laparoscopic) is safe and rapid. Should an AAST OIS grade I or II injury be present,

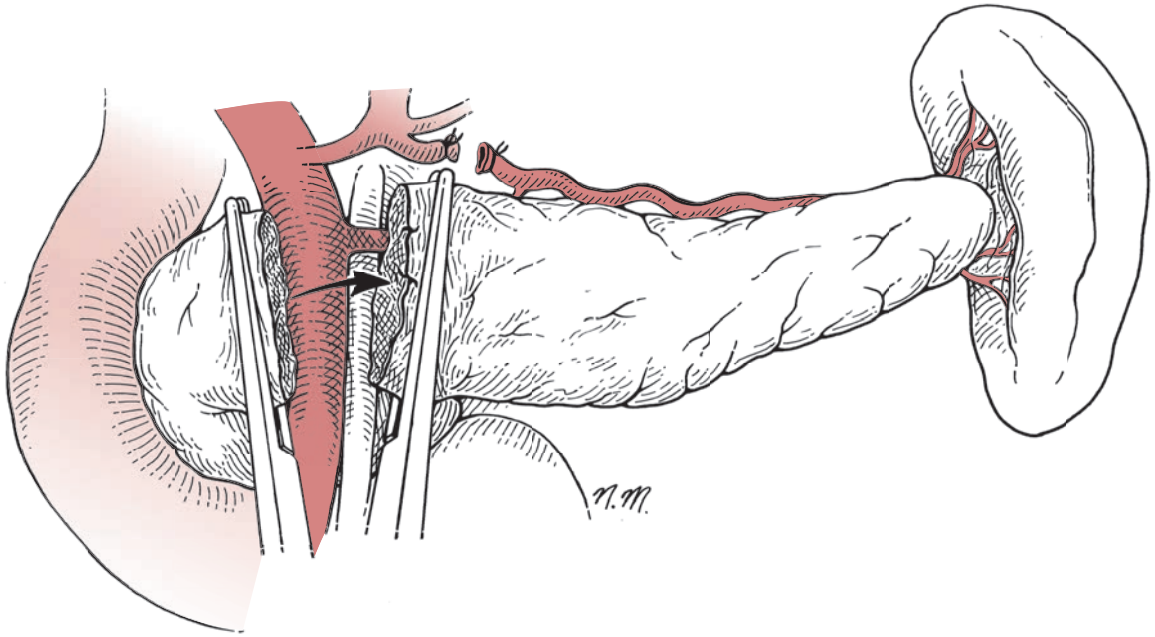

FIGURE 42-1 Distal pancreatectomy and splenectomy with exposure of the portal venous confluence. (Reprinted with permission from Cushman JG, Feliciano DV. Contemporary management of pancreatic trauma. In: Maull KI, Cleveland HC, Feliciano DV, et al, eds. *Advances in Trauma and Critical Care*. St. Louis, MO: Mosby; 1995, vol 10, pp 309-336.)

rapid mobilization and direct suture repair may be faster than splenectomy and will avoid a denuded retroperitoneal area that may be susceptible to hemorrhage in coagulopathic patients. With rupture of the splenic capsule, a multitude of commercially available topical hemostatic agents, as well as a bipolar sealing energy instrument, work well.[90,91]

Hollow Viscera (See Chapters 35–37)

Damage control techniques for hollow viscera include rapid excision for multiple adjacent injuries or temporary staple exclusion of injured segments. In the case of gastric division, the proximal stomach should be decompressed by a nasogastric tube. Near transections of the duodenum are stapled shut, whereas an associated injury to the head of the pancreas is packed. At the reoperation, once metabolic failure has been corrected, duodenal continuity can be restored with an end-to-end anastomosis. A pyloric exclusion with polypropylene suture and an antecolic gastrojejunostomy may be added in selected patients with severe duodenal contusion, narrowing after a suture repair, or a combined pancreatoduodenal injury.[92,93] Distal feeding access must also be considered in particularly ill patients.

If a limited number of enterotomies or colostomies from penetrating wound(s) are encountered, rapid one-layer, full-thickness closures using a continuous 4-0 monofilament suture is appropriate. Multiple large perforations within a short segment of small bowel or colon are treated with segmental resection, using metal clips or an energy ligation device for mesenteric hemostasis and staples to transect the bowel. The small bowel and colon can be left in discontinuity for up to 72 hours with minimal sequelae.[87] With shotgun wounds and multiple partial- and full-thickness perforations of the jejunum, a jejunectomy is appropriate because all of its absorptive capabilities are duplicated by the ileum. On occasion, large-blast or close-range shotgun injuries may cause 50 to 100 holes over the entire length of the small bowel. This particularly problematic scenario may lead to an enterocutaneous fistula after the reoperation due to a missed injury or failed repair. This will require treatment with drains, negative-suction devices, injections of somatostatin analog, nutritional support, and frequent returns to the operating room for control of intraperitoneal sepsis. A jejunectomy will lower the risk of this complication.

ABDOMINAL COMPARTMENT SYNDROME

Abdominal compartment syndrome (ACS) is defined as a sustained intra-abdominal pressure greater than 20 mm Hg that is associated with new organ dysfunction/failure (Fig. 42-2).[94-108] ACS differs from intra-abdominal hypertension (IAH), which is graded (I–IV) and defined as sustained pathologic elevation greater than 12 mm Hg. Symptoms of ACS are extensive and impact every major system within the human body. These include, but are not limited to, cardiovascular (hypotension), renal (acute kidney injury), and respiratory (failure) components. It is interesting to note that many of the dominant risk factors for developing ACS mirror the physiologic triggers for engaging in DCS/DCR. This observation supports the concept that physiologic variables (eg, core temperature, pH, base deficit) are clear markers for

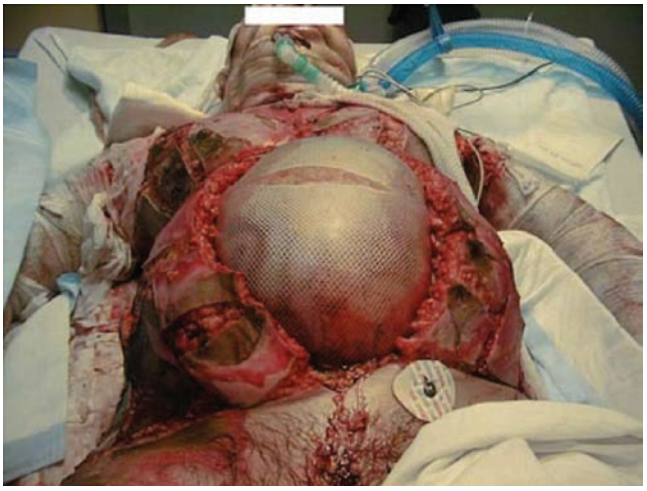

FIGURE 42-2 Decompressive laparotomy and fasciotomies for a patient with severe burns and abdominal compartment syndrome.

the absolute sickest of the sick. Closing abdomens in patients manifesting physiologic extremis often leads to ACS, as well-described by Morris et al[97] in 1993. With closure, these authors described severe abdominal distension in concert with raised peak airway pressures, carbon dioxide retention, and oliguria. Their observed 63% mortality rate associated with reperfusion injury after unpacking was also dramatic. Recurrent or tertiary ACS is an important concept to consider in cases where ACS has already been treated previously but has recurred (ie, often in the setting of additional resuscitation for new hemorrhage or sepsis).[98] Quaternary ACS occurs once the abdominal wall has been closed, but the ACS recurs. Although the incidence of primary ACS has decreased dramatically over the past decade,[94] continued vigilance is crucial to guard against secondary and recurrent ACS. Despite the increased understanding surrounding this anatomic and physiologic complication, it is clear that many clinicians require more education with regard to both monitoring and treating ACS.[99] This concern has led to a recent evidence-based update of the definitions of primary, secondary, and recurrent ACS, as well as of an expert society's therapeutic recommendations.[95] More specifically, in addition to multiple suggestions (Tables 42-4 and 42-5), the World Society of the Abdominal Compartment Syndrome strongly recommends the following: (1) measuring intra-abdominal pressure when any known risk factor for IAH/ACS is present in a critically ill or injured patient using a transbladder technique (grade 1C); (2) using protocolized monitoring and management of intra-abdominal pressure (grade 1C); (3) engaging in a decompressive laparotomy for cases of overt ACS (grade 1D) (Fig. 42-2); (4) attempting to ensure same-hospital-stay abdominal fascial closure (grade 1D); and (5) using negative-suction therapy in patients with open abdominal cavities (grade 1D).[95] It should also be noted that some patients may adequately respond to decompression (nasogastric, colonic, intraperitoneal, increased sedation, and/or paralysis) as primary therapeutic maneuvers. Failure to resolve ACS with these medical therapies, however, should lead to rapid surgical decompression. Measuring intra-abdominal pressures can be easily performed at the bedside with a three-way Foley catheter, pressure transducer, and intravenous tubing.[108,109] The probe should be zeroed at the patient's hip in the supine position.

TABLE 42-4: Consensus Definitions of the World Society of the Abdominal Compartment Syndrome (ACS)

IAP is the steady-state pressure concealed within the abdominal cavity.

APP = MAP – IAP

IAH is graded as follows:

- Grade I, IAP 12–15 mm Hg
- Grade II, IAP 16–20 mm Hg
- Grade III, IAP 21–25 mm Hg
- Grade IV, IAP >25 mm Hg

ACS is defined as a sustained IAP >20 mm Hg (with or without an APP <60 mm Hg) that is associated with new organ dysfunction/failure.

Primary ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiologic intervention.

Secondary ACS refers to conditions that do not originate from the abdominopelvic region.

The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 mL of sterile saline.

IAP should be expressed in mm Hg and measured at end-expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the midaxillary line.

Normal IAP is approximately 5–7 mm Hg in critically ill adults.

IAH is defined by a sustained or repeated pathologic elevation in IAP ≥12 mm Hg.

Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS.

Polycompartment syndrome is a condition where two or more compartments have elevated compartmental pressures.

An open abdomen is one in which the abdominal viscera are exposed to the outside environment or retained by a nonautologous artificial barrier.

Pathophysiologic classification of the open abdomen

1A = clean, no fixation

1B = contaminated, no fixation

2A = clean, developing fixation

2b = contaminated, developing adhesions

3 = "frozen abdomen" with complete fixation

4 = "frozen abdomen" with enterocutaneous fistula

Abdominal wall compliance defines a concept regarding the ease of expansion of the abdominal wall and its contents, which is determined by the overall intra-abdominal volume and elasticity of the abdominal wall.

APP, abdominal perfusion pressure; IAH, intra-abdominal hypertension; IAP, intra-abdominal pressure; MAP, mean arterial pressure.

TABLE 42-5: Summarized World Society of the Abdominal Compartment Syndrome Consensus Management Statements

Recommendations

1. Measure IAP when any known risk factor is present in a critically ill/injured patient.
2. Studies adopt the current and accepted standard IAP measurement technique.
3. Protocolized monitoring and management of intra-abdominal pressure should be used when caring for the critically ill/injured.
4. Efforts and/or protocols should be used to avoid sustained IAH.
5. Use DCL to reduce IAP in cases of overt ACS.
6. Negative-pressure therapy should be used to faciltate earlier fascial closure.

Suggestions

1. Critically ill/injured should receive optimal pain and anxiety relief.
2. Brief trials of neuromuscular blockade may temporize the treatment of IAH.
3. The potential contribution of body position in elevating IAP should be considered in patients with moderate to severe IAH or ACS.
4. Use a protocol to try to avoid a positive cumulative fluid balance in the critically ill with or at risk of IAH.
5. Use an enhanced ratio of plasma to packed red blood cells during resuscitation from massive hemorrhage.
6. Use percutaneous drainage to remove fluid in those with IAH/ACS when this is technically possible whether the alternative is doing nothing *or* a decompressive laparotomy.
7. Physiologicaly exhausted posttrauma laparotomy patients should use a prophylactic open abdomen strategy.
8. Intraperitoneal contamination (even if severe) is **NOT** an indication for the routine or prophylactic use of the OA technique.
9. Biological meshes should **NOT** be routinely used to faciltate early acute fascial closure.

No recommendations

1. No recommendation was made regarding the use of the abdominal perfusion pressure as a resuscitation marker.
2. No recommendation was made regarding the use of diuretics to mobilize fluid in hemodynamicaly stable patients with IAH.
3. No recommendation was made regarding the use of renal replacement therapies to mobilize fluids in hemodynamically stable patients with IAH.
4. No recommendation was made regarding the administration of albumin to mobilize fluids in hemodynamically stable patients with IAH
5. No recommendation could be made to use the component separation technique to facilitate earlier acute fascial closure.
6. No recommendation was made regarding the use of the OA technique in emergency surgery in patients with physiologic exhaustion.

ACS, abdominal compartment syndrome; DCL, decompressive laparotomy; IAH, intra-abdominal hypertension; IAP, intra-abdominal pressure; OA, open abdomen.

In addition to raised pressures within the abdominal compartment, similar manifestations may also occur in closed compartments of the extremities. Akin to secondary abdominal compartment syndrome, secondary extremity compartment syndrome is a result of a massive resuscitation.[110] It is also associated with extremely high morbidity and mortality (35%–70%). Serial measurements of creatine phosphokinase (CPK) and urine myoglobin may be appropriate in high-risk patients as a screening tool. Any individual with persistently high CPK levels or the unexplained development of myoglobinuria may benefit from measurement of extremity compartment pressures and fascial releases as appropriate.

MANAGEMENT OF THE OPEN ABDOMEN (SEE ATLAS FIGURE 67)

The concept of delaying abdominal wall closure is credited to Dr. H. Harlan Stone at Grady Memorial Hospital in Atlanta, Georgia, in 1981.[100] Among 167 patients, mortality approximated 85% in those whose abdomens were closed under tension, compared to only 22% in patients who underwent delayed fascial closure. This truly remarkable report altered the DCS landscape dramatically. Unfortunately, the open abdomen is also responsible for significant short-term (fluid and protein loss, sepsis, intestinal fistulas, nursing care challenges, economic costs) and long-term (chronic physical discomfort, physique embarrassment, delayed return to work, poor quality of life) morbidity.[103,104]

Management of the open abdomen can be divided into two phases. In the acute phase, the goal is to provide some variant of temporary coverage to allow the patient to be taken from the operating room to the intensive care unit for additional resuscitation and stabilization with the intent of returning to the operating room when normal physiology has been restored. In the second phase, which follows reoperation, the ultimate goal is a delayed fascial closure. On occasion, this is not technically possible, and a planned ventral hernia will be required.

Multiple techniques for covering the open abdomen at the index operation are available. These range from simple techniques such as covering the viscera with nylon cloth material followed by "generous gauze packs" (Detroit Receiving Hospital) to commercially available negative-suction devices.[111,112] Because no single technique has been shown to be far superior to others, a given trauma center should employ a single consistent option that ensures familiarity by all surgeons, house staff, and nurses. This allows accurate assessment of fluid losses, as well as intermittent troubleshooting. A simple and

cost-effective choice remains a large sterile x-ray cassette drape with two closed suction drains and a covering plastic adhesive drape. This temporary silo technique was initially described by Oswaldo Borraez G. at the San Juan de Dios Hospital in Bogota, Columbia, in 1984. Alternative materials for coverage include a 3-L plastic irrigation bag that is opened up into a single sheet of plastic, a Silastic sheet, or a PTFE sheet. The use of vacuum-assisted wound closure devices is also now common throughout much of the world. The initial "homegrown" description of this concept was reported by Barker et al[113] in 112 patients. The principal goals of this technique remain coverage of the viscera, rapid removal of peritoneal fluid, and maintenance of some midline traction. In much of the developed world, low-technology versions of commercial products have provided clinicians with an excellent alternative. It is critical to mention, however, that avoiding commercial negative-suction devices during the initial critical phase (ie, following the first laparotomy) may be helpful in the prevention of recurrent ACS.[105] Alternatively, if recurrent ACS does occur, simply turning off the suction pump and therefore converting the negative-suction device into a simple silo often relieves recurrent IAH.

Other options for covering the open abdomen include towel clip or suture closure of the skin. The advantage of these techniques is that they are simple, rapid, and readily available in almost all settings.[114] They can also be employed for closure of chest, abdomen, and groin incisions. Depending on the length of the incision, up to 30 standard towel clips may be necessary to complete the closure. To prevent manipulation of the towel clips and minimize secondary contamination of the chest, abdomen, or groin through the spaces between the towel clips, a large plastic adherent drape is also placed over the towel clips in combination with a closed suction drain lying lateral to the incision. When suture closure of the skin is selected, 2-0 nylon or thicker suture material is selected, depending on the tension. It should also be noted that these skin-only closures are used much less commonly now given their propensity to increase intra-abdominal pressure.

It is clear that as the patient is exposed to multiple subsequent operative interventions, intestinal coverage (via endogenous abdominal wall or skin or split-thickness skin graft) must be achieved as soon as possible to limit the development of enteroatmospheric fistulas. It is also evident that, regardless of technique, severely injured patients more commonly achieve fascial closure during their initial hospital stay than their nontrauma, acute care surgery counterparts. If closed too early, ACS, fascial dehiscence, necrotizing fasciitis, and ventilation challenges are notable complications. Despite the poor methodology inherent in the open abdomen literature (ie, mixed patient cohorts, lack of complete inclusion, ignorance of nonsurvivors, variable individual surgeon effort and interest), it is evident that the application of negative-suction dressings at subsequent operations has improved closure rates and shortened time to closure. The two dominant principles when using negative-suction therapy are maintenance of the peritoneal/abdominal domain and continuous and progressive tension on the midline abdominal wall. These goals are achieved first by insertion of a plastic barrier deep into the paracolic gutters (maximally lateral to prevent adhesions between the colon and abdominal wall) and, second, by generation of midline abdominal wall tension using nonfascial retention sutures or commercial systems. It should also be noted that intra-abdominal pressures often exceed "normal" (>20 mm Hg) immediately after progressive increases in tension at the midline during repeat laparotomy and attempted closure.[107] This typically abates over the subsequent few hours and is considered acceptable in the absence of end-organ ischemia (decreased urine output, increased airway pressures). As a result, it is considered fundamentally different from the acute phase of ACS. If the intra-abdominal pressure does not normalize, however, the abdomen must be reopened to prevent recurrent ACS.

In conclusion, an individual patient with an open abdomen will either continue to improve, mobilize fluid, and allow gradual abdominal closure via repeat laparotomies, or will continue to be challenged with sepsis and multiorgan failure, will not mobilize fluid, and will eventually require coverage with a split-thickness skin graft (Fig. 42-3). Enteroatmospheric fistulas must also be prevented at all costs. This complicates short-term management, as well as eventual reconstruction of the abdominal wall (component separation, modified component separation, retrorectus mesh procedures, indications for the use of biologic mesh implants). If present, these fistulas are best intubated by soft rubber catheters placed within the sponge material of a negative-pressure suction dressing.[115] Over time, this will allow the patient to develop a granulation plate around the fistulas appropriate for a skin graft (ie, conversion into a stoma).

A detailed discussion of the tiered algorithm for abdominal wall reconstruction, as well as the indications for occasional use of biologic materials, is beyond the goals of this review. Clearly, the appropriate timing of reconstruction (6–12 months) is vital to the success of the repair (adhesions vs lateral retraction of the rectus muscle). Extensive experience in reconstructive techniques is also crucial to ensure

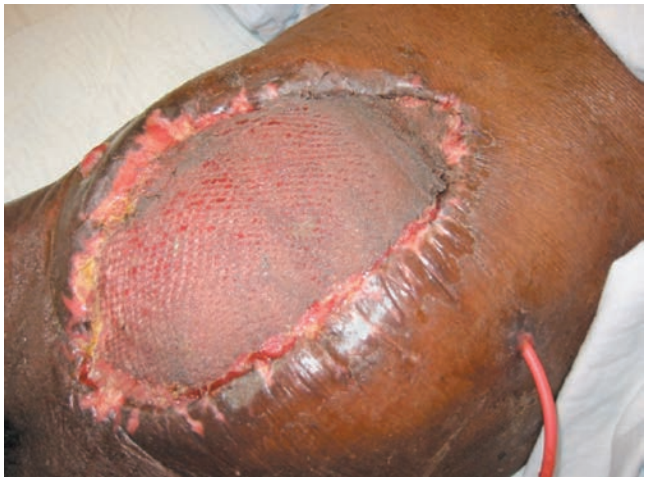

FIGURE 42-3 Split-thickness skin graft on an open abdomen.

acceptable outcomes. These principles include, but are not limited to, timing, sequencing (stoma reversal or fistula closure), ensuring adequate skin coverage, sparing of periumbilical perforators, minimally invasive lateral releases, and management of wound complications. In particular, reconstruction of the gastrointestinal tract (eg, stoma takedown, resection of fistulas) can be undertaken by partial elevation of a healed split-thickness skin graft as a first separate procedure. Three or more months later, a separate reconstruction of the abdominal wall can be safely performed without the risk of gastrointestinal contamination.[116] Consideration may also be given to the use of expensive, but effective, biologic mesh inserts in fields of mild to moderate contamination as a one-stage procedure.

REOPERATION

Following control of surgical bleeding and contamination, patients are transferred to the intensive care unit for ongoing resuscitation and stabilization. The immediate goals are to both provide adequate oxygenation and reverse the effects of inadequate tissue perfusion and resultant metabolic failure. Correction of hypothermia, coagulopathy, and acidosis must be at the forefront. Hypothermia can be treated by external rewarming or, in extreme cases, cardiopulmonary bypass. Our increasingly nuanced understanding of the state of coagulopathy has also ushered in new tests such as thromboelastography.[117] Although significant excitement surrounds these techniques, our true understanding of their clinical role continues to evolve (see Chapter 16).

Reoperations can be divided into emergency or planned. Failure to attain the desired end points of resuscitation in the immediate postoperative period of damage control may reflect continued hemorrhage.[97,118] An early return to the operating room is a difficult decision because physiologic and biochemical stabilization is typically incomplete at this point (Table 42-6). Therefore, the surgeon must postulate whether mechanical or surgical hemorrhage is occurring versus diffuse oozing from a coagulopathy in which an early reoperation may not be indicated. As mentioned, an obvious indication for an early reoperation is the development of ACS (ie, with failure of a decrease in pressure once the negative-suction pump has been turned off).

A planned reoperation typically occurs when postoperative bleeding is not a dominant concern and the patient has achieved normalization of cardiovascular, pulmonary, and metabolic parameters (Table 42-7).[119] Although the specific timing of a return to the operating room remains a source of debate, a patient who is normotensive, without a coagulopathy, and is in the diuretic phase of recovery after resuscitation from shock is the ideal candidate. The more important concept remains returning the patient to the operating room as soon as the patient has stabilized, rather than based on a given surgeon's schedule.

In patients who cannot achieve fascial closure at the initial reoperation (due to incomplete reconstructions, persistent intraperitoneal sepsis, or visceral/abdominal wall edema secondary to the initial resuscitation), a repeat temporary abdominal closure must be applied. Choices remain identical to the initial operation, with the exception of a much lower risk of recurrent ACS with a negative-suction device.[105] Although the literature is composed of a long list of evolving techniques to address the recurrent open abdomen (vacuum-assisted,[120-123] zippers,[124] Wittmann Patch [Starsurgical, Inc., Burlington, WI],[125-128] absorbable meshes,[129-132] or permanent meshes[100,133-136]), the majority of centers use a commercially available negative-suction device. As previously mentioned, maintenance of the peritoneal domain and prevention of adherence of the viscera to the underside of the abdominal wall are critical to allow progressive closure of the fascia with repeated operations. Other nuanced technical concerns include the following: avoidance of injuring the fascia at all operations preceding the final closure irrespective of the technique selected; maintaining progressive midline traction with each successive operation (ie, beyond simply applying a negative-suction device); and avoiding converting the geometry of the wound from an ellipse into a circle (ie, by closing the top and bottom but leaving the middle of the wound open). By following these principles and applying a negative-suction device at reoperation as an adjunctive tool, it is clear that patients have fewer wound issues, better management of peritoneal fluid and contamination, and fewer planned ventral hernias.

TABLE 42-6: Indications for Emergent Return to the Operating Room After a Damage Control Laparotomy

Blunt trauma	Penetrating trauma
Normothermic but bleeding >2 U/h	Normothermic but bleeding >2 U/h
Abdominal compartment syndrome with ongoing blood loss	

TABLE 42-7: Indications for Elective/Planned Return to the Operating Room After a Damage Control Laparotomy

Temperature >36°C (96.8°F)
Acid-base balance
Base deficit corrected to greater than –5 mmol/L if originally less than –15 mmol/L
Serum lactate normal or correcting gradually
Coagulation
Prothrombin time <15 seconds
Partial thromboplastin time <35 seconds
Platelets >50,000/mm^3
Cardiovascular
Cardiac index >3 L/min/m^2, with or without low-dose inotrope
Pulmonary
Fraction of inspired oxygen <0.50
Oxygen saturation >95%

FIGURE 42-4 Resuscitation with Angiography, Percutaneous Techniques, and Operative Repair (RAPTOR) environment.

HYBRID CARE SUITES

Given that DCR is dedicated to our most critically injured patients, it is not surprising that the development of a single location (ie, RAPTOR—Resuscitation with Angiography, Percutaneous Techniques, and Operative Repair) where percutaneous therapies, operative interventions, cross-sectional imaging, and critical care can all be delivered holds significant promise (Fig. 42-4).[137-140] This concept transitions care from a location-based approach to a truly disease- and urgency-based algorithm. The best example remains a patient with ongoing hemorrhage from an unstable pelvic fracture in the context of associated injuries that may or may not require emergent operative intervention. In recent reports, the use of a hybrid suite in severely injured patients with ongoing hemorrhage was shown to reduce both the time to intervention and the overall procedure duration.[139,140] Further, a large survival benefit was also noted in the most critically ill patients.[140] The significant concerns with this technology remain the tremendous cost of building hybrid suites, as well as the training requirement for clinicians to safely perform hybrid procedures.[137,138]

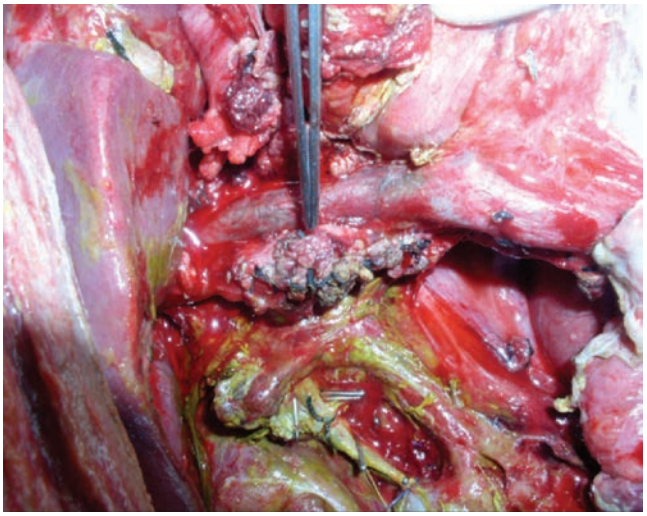

FIGURE 42-5 Resection of the pancreatic head following ligation of the inferior vena cava with synchronous right nephrectomy as an initial damage control procedure.

CONCLUSION

In conclusion, DCR currently includes simultaneous early transfusion of blood products and arrest and/or temporization (ie, balloon tamponade and TIVS) of ongoing hemorrhage in the most challenging patients (Fig. 42-5). As a result, DCR addresses the early coagulopathy of trauma, avoids massive crystalloid resuscitation, and leaves the peritoneal cavity open when a patient approaches physiologic exhaustion without improvement. Although the precise indications for DCS remain debated,[141,142] the future evolution of the DCR concept will include further elucidation of personalized resuscitations (individual blood product ratios based on point-of-care testing), as well as introduction of hybrid angiography-operating suites in centers with resources.

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43

Upper Extremity

Raymond Pensy

KEY POINTS

- The field of hand surgery has evolved via the integration of surgical disciplines specific to bone, tendon, nerve, vessel, and skin.
- A careful inventory of each of these components must be made in every patient to plan treatment and anticipate expected functional recovery of the traumatized hand and upper extremity.
- Assessment of tissue viability should occur through a careful vascular and neurologic examination, both pre- and intraoperatively.
- The skeleton represents the foundation of any reconstruction of the upper limb; a plan for temporizing and/or definitive fixation in every fracture must be established prior to finalizing any other repair.
- A plan for soft tissue coverage should be made early when the skin envelope is compromised.

INTRODUCTION

The origins of hand and upper extremity surgery in the United States as a surgical specialty can be largely attributed to Major General Norman Kirk, MD (Fig. 43-1), the first orthopedic surgeon to be appointed as surgeon general of the armed forces.[1] With his significant experience in treating veterans of World War I and World War II,[2] he appreciated that injuries of the hand and upper extremity commonly involve joint, bone, vessel, tendon, and nerve, and optimal treatment required familiarity with each.

In an effort to improve the outcomes related to the care of the hand and upper extremity, Sterling Bunnell[3] (Fig. 43-2), a friend and contemporary of Norman Kirk, was charged with founding the first group of specialized military training centers for hand surgery.[3] These programs have evolved into the current modern hand fellowships.

In 1989, recognizing that surgery of the hand requires a specific skill set for optimal outcomes, the American Boards of Surgery, Orthopaedic Surgery, and Plastic Surgery[4] initiated a specialty examination process culminating in a Certificate for Additional Qualifications for Surgery for the Hand, now called Subspecialty Certificate in Surgery of the Hand. This certification represents the pinnacle of training and aspirations of clinicians committed to caring for those afflicted with trauma or malady of the hand and upper extremity. This chapter is a reflection of the basic tenets and a brief overview of the current state of this specialty.

THE HAND AND UPPER EXTREMITY AS PART OF THE WHOLE

The approach to the treatment of the trauma victim must follow the standardized, thoroughly investigated, and well-established protocols of basic and advanced life support in the field, as well as Advanced Trauma Life Support (ATLS). Many would agree that the severed or mangled hand, forearm, or arm can be a disturbing sight, but the experienced traumatologist recognizes the importance of first assessing the airway, breathing, and circulatory system, as well as the potential for injuries to the brain, chest, or abdomen. Because these injuries are frequently life-threatening, timely and thorough evaluation of these systems must be completed first, even in those cases where the most obvious injury is that of the extremity and even in cases of amputation.

When considering severe trauma to the upper extremity, assessment of the perfusion status is paramount.[5] In instances where absolute ischemia exists due to a tourniquet or as the result of an incomplete amputation, all appropriate haste must be made to ensure that ischemic time is minimized. The time of injury or tourniquet application time

FIGURE 43-1 Major General Norman Kirk, MD. (From U.S. National Library of Medicine. http://resource.nlm.gov/101420484.)

FIGURE 43-2 Sterling Bunnell, MD.

must be documented, and efforts to minimize this time are paramount.

In all other cases, injuries of the hand and upper extremity can be evaluated with a careful physical examination, thorough radiography, splinting, and, finally, operative treatment as necessary. With these careful assessments, the status of the constituent nerves, bone, tendon, and skin will be determined.

In planning appropriate treatment of the hand or arm of the polytrauma victim, one must consider the influence this injury will have on the patient and his or her recovery as a whole. The experienced reconstructive surgeon will recognize the impact that early functional rehabilitation of the hand and arm will have for the overall care of the patient.[6] The differences in treatment rendered for an isolated injury to the upper extremity, compared to that in a patient with concomitant injuries, can be significant. For example, a patient with a humerus fracture in isolation will do well with splinting, but if a non–weight-bearing injury in a lower extremity is present also, the patient will do better with rigid fixation permitting unrestricted weight bearing of the upper extremity.

In an effort to simplify the approach to examination, diagnosis, and treatment to the injured hand and upper extremity, the author finds it reasonable to consider the upper extremity as a collection of skeletal, joint, vascular, artery, muscle, tendon, nerve, and skin tissues. Each of these requires its own set of independent diagnostic modalities and treatments, and the sections that follow are organized as such.

SKELETON AND JOINT—GENERAL EVALUATION AND TREATMENT

Physical Examination

None would argue that, in the absence of a sturdy, robust, and well-formed foundation, efforts at subsequent reconstruction are fruitless. The first responders will relay a history of deformity if present, as well as other crucial information. Simple inspection will reveal asymmetric posture and malangulation. The awake patient will guard the fractured upper limb and avoid movement, often keeping the limb motionless. The surgeon must examine the patient's extremities for deformity, crepitance, "clunking," and pain to assess for gross signs of fracture or dislocation of a joint.[7] The movements of the upper extremity at all joints and planes should be supple and painless and assessed in every degree of motion, both passive and active. Joints should be examined for swelling, and the skin checked carefully for ecchymoses, lacerations, and abrasions, with particular attention paid to the possibility of an open fracture. Finally, careful and deliberate palpation of each region of the upper limb should be performed and documented. Not infrequently, the polytrauma patient will have concomitant injury to the spine, trunk, or lower extremity, which may distract the examiner from the smaller bones of the hand and feet. Thus, in some instances, a repeat secondary survey with focal radiography is advised.[8]

TABLE 43-1: Imaging Examinations for Shoulder Girdle Region

Conditions	X-ray	Advanced imaging
Sternoclavicular dislocations	AP, serendipity view	CT
Clavicle fracture	AP, apical/oblique views	
Acromioclavicular dislocations	AP, apical/oblique views	
Scapular fracture	True AP, scapular Y, and axillary lateral views	CT
Acromion fracture	True AP, scapular Y, and axillary lateral views	
Glenoid neck fracture	True AP, scapular Y, and axillary lateral views	
Glenoid fracture	True AP, scapular Y, and axillary lateral views	
Scapular body fracture	True AP, scapular Y, and axillary lateral views	
Floating shoulder without associated clavicle fracture	True AP, scapular Y, and axillary lateral views	
Scapulothoracic dissociation	True AP, scapular Y, and axillary lateral views	CT/MRI/angiography
Glenohumeral dislocation	AP/Bloom–Obata modified	Axillary view
	Scapular Y view	CT/MRI

AP, anteroposterior; CT, computed tomography; MRI, magnetic resonance imaging.

Diagnostic Modalities

ORTHOGONAL RADIOGRAPHY

In the trauma patient, plain radiography will reveal the majority of skeletal injuries requiring intervention (Tables 43-1, 43-2, and 43-3). In many cases, the nature of the fracture's appearance on plain films will alert the provider to possible significant injury related to the other components of the upper extremity (Figs. 43-3A and B and 43-4).

In general, any part of the upper limb that demonstrates deformity, lack of suppleness, crepitance, or pain upon palpation should be imaged, as should the joint above and below. Many fractures of the upper extremity will involve dislocation of the joint distal or proximal to the level of an obvious fracture and result in significant morbidity if unrecognized, such as Monteggia and Galeazzi injuries[9] (Fig. 43-5).

Orthogonal radiographs should be acquired in a standardized manner. The fractured arm (humerus) or forearm (radius and/or ulna) is imaged in the anteroposterior (AP) plane, replicating the anatomic position, and then a lateral plane, obtained without rotating the limb. This is accomplished by positioning the cassette and beam 90° from the initial exam and with a provider or assistant carefully lifting the limb and holding the cassette to achieve this orthogonal view. It is generally contraindicated to "twist" or rotate the limb in order to achieve this orthogonal view, as it will cause undue pain,

TABLE 43-2: Imaging Examinations for the Arm, Elbow, Forearm, and Wrist

Conditions	X-ray	Advanced imaging
Proximal humerus fracture	AP, transscapular lateral, Bloom–Obata modified	CT
Humeral shaft fracture	axillary view	
Elbow trauma	AP, lateral	
Distal humeral fracture	AP, lateral	CT
Capitellum fracture	AP, lateral	
Elbow dislocations/fracture-dislocations	AP, lateral	CT
Fracture of proximal ulna	AP, lateral	CT
Radial head fracture	AP, lateral	
The floating elbow	AP, lateral	
Monteggia fracture-dislocations	AP, lateral	
Ulna shaft fracture	AP, lateral of elbow and forearm	
Radial shaft fracture	AP, lateral	
Distal radius injury/fracture	AP, lateral	
Radius/ulna fractures	AP, lateral	
Scaphoid fractures	AP, lateral	
	PA, lateral, oblique, scaphoid	MRI

AP, anteroposterior; CT, computed tomography; MRI, magnetic resonance imaging.

TABLE 43-3: Imaging Examinations for Wrist and Hand

Conditions	X-ray	Advanced imaging
Triquetrum fractures	PA, lateral, oblique	CT/MRI
Pisiform fractures	Carpal tunnel view and/or supinated oblique view	CT
Trapezium fractures	Hyperpronated Roberts view, Bett's view, carpal tunnel view	CT
Trapezoid fractures	PA, lateral, oblique	CT
Capitate fractures	PA, lateral, oblique, and scaphoid view	CT
Hamate fractures	Carpal tunnel view and/or supinated oblique view	CT
Hook fractures	Carpal tunnel view and/or supinated oblique view	CT/MRI
Lunate fractures	PA, lateral, oblique	
Carpal dislocations	PA, lateral, oblique	
Metacarpal fractures	PA, lateral, oblique	
Phalangeal fractures	PA, lateral, oblique	
Metacarpophalangeal dislocations	PA, lateral, oblique	
Interphalangeal joint dislocations	PA, lateral, oblique	

CT, computed tomography; MRI, magnetic resonance imaging; PA, posteroanterior.

result in poor image quality, and, in some cases, further displace the fracture or dislocation.

Static injury films of the limb are acquired in its injured state prior to realignment, splinting, or traction, and provide useful information regarding the force vector that resulted in a given injury pattern. This will often dictate the method of treatment, whether it be in regard to an initial reduction maneuver, application of a three-point mold with splinting or casting, or the application of plates to "buttress" or "neutralize" the deforming forces. Further, injury films document the severity of the injury. To the experienced eye, even these plain radiographs will yield valuable information regarding the possibility of significant compromise of soft tissue above and beyond that which is obvious to the skeleton. In any trauma center with the availability of a physician or other medical personnel capable and trained in performing reductions, injury films should, in most instances, be performed prior to the reduction attempt. Although tempting to reduce the injured part out of a perceived benefit to the patient (to restore blood flow; limit desiccation to exposed cartilage, tendon, or bone; lessen skin tension and ischemia), the few minutes required for radiography will not significantly add to morbidity, as the time from injury to reduction is only modestly lengthened by radiographs that almost always assist in treatment.

Traction views will provide the astute extremity surgeon with additional information, particularly for injuries of the distal humerus, radius, and phalanges.[10,11] In many cases, traction alone can realign fractures of the distal radius and phalanges to acceptable parameters. This may demonstrate fractures that can be treated with less invasive external fixation or other closed techniques. Additionally, traction views will clarify the "reducibility" of fracture elements through ligamentotaxis, which will further delineate treatment parameters such as the surgical approach, choice of surgical implants, and the likely duration of the surgical effort (Fig. 43-6).

COMPUTED TOMOGRAPHY

Computed tomography (CT) has revolutionized imaging and care of the trauma patient, especially in the precise delineation of skeletal injury patterns and particularly in those in periarticular regions.[12]

Specific to the upper extremity, CT is recommended for the following: fractures involving the scapula, particularly those involving the glenohumeral joint; fracture-dislocation of the elbow (fractures involving the primary stabilizers including the coronoid, radial head, and trochlea, associated with dislocation); distal radius fracture in some instances; carpometacarpal joint fractures; and intra-articular injuries of the fingers and those of the carpus, particularly scaphoid fractures.[13-15]

Treatment

The rationale behind skeletal stabilization is simple, yet its effects are profound. First, stabilization of the injured/fractured long bone will substantially mitigate pain.[16] The stabilization of a long bone (femur, tibia, and humerus) limits additional microtrauma to the surrounding muscle, vessels, and nerves, and immediately improves pain. Effective pain control is important in mitigating its effects on the cardiopulmonary and neurologic systems.[17,18]

Continued instability of the injured limb often promotes vasospasm and arterial thrombosis. Finally, in circumstances of severely displaced fractures or malaligned joints, the skin will become tented, resulting in localized ischemia and, in severe cases, necrosis. This will further complicate treatment because of unwanted exposure of neurovascular structures, tendons, or joints.

Stabilization of the fractured or dislocated skeleton is performed with a variety of nonoperative and operative techniques. The orthopedist has a broad array of techniques available (Table 43-4). These techniques have roles specific to

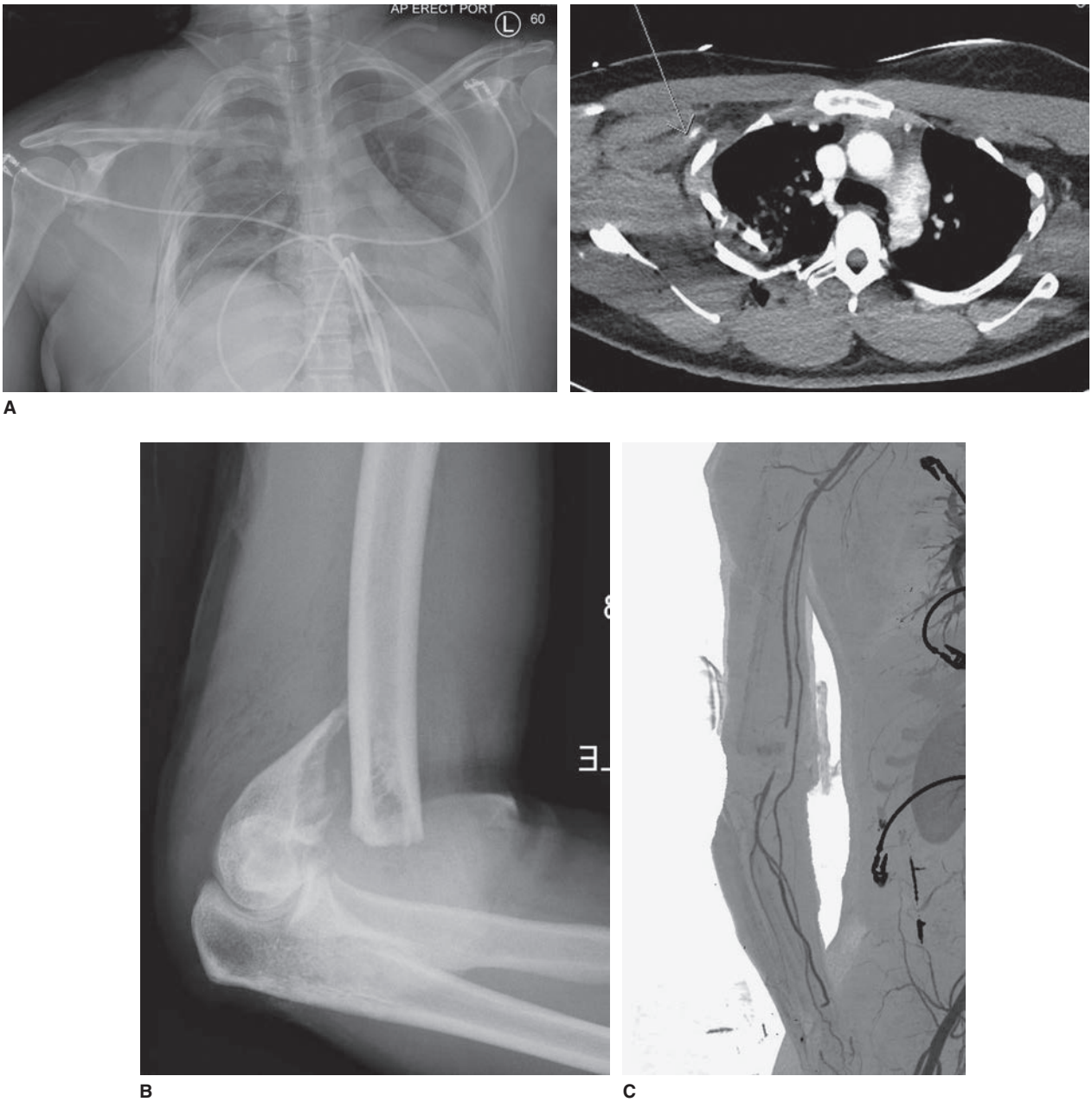


FIGURE 43-3 (**A**) Axillary artery injury with scapulothoracic dissociation. (**B**) Brachial artery injury with extension-type supracondylar humerus fracture. (**C**) Brachial artery injury following ballistic trauma, with maintained perfusion via high/proximal radial artery origin.

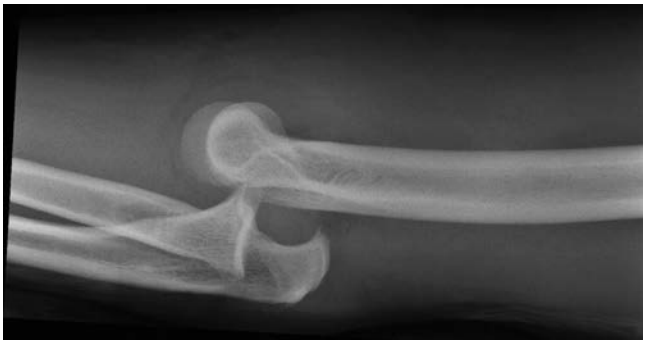
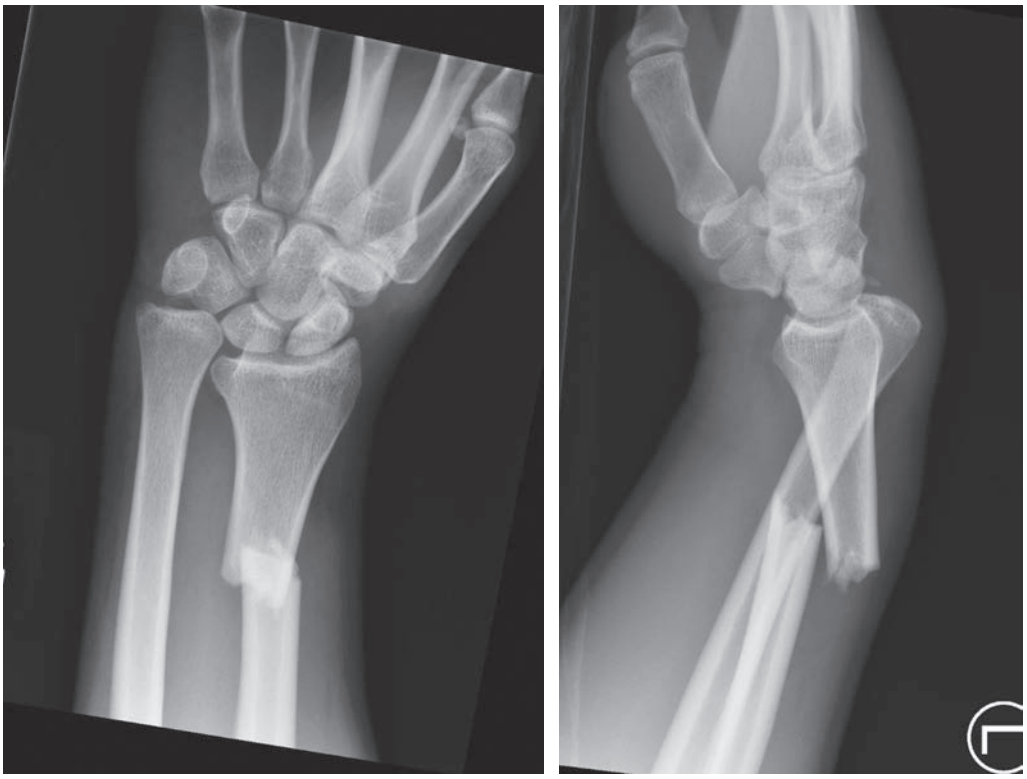


FIGURE 43-4 Brachial artery injury with elbow dislocation.

the bones that require stabilization and have different properties of mechanical rigidity, different amounts of requisite surgical dissection and trauma, different risk profiles, and, of course, differences in the time required to achieve application.

SPLINTING/CASTING

The efficacy of a well-molded splint or cast cannot be overstated. Current widely employed splints of plaster of Paris and moldable fiberglass provide efficient and effective means of stabilizing the injured limb.[19] They provide effective pain control, decrease risk to the overlying skin and the underlying vascular tree, and facilitate patient transport and transfer. In



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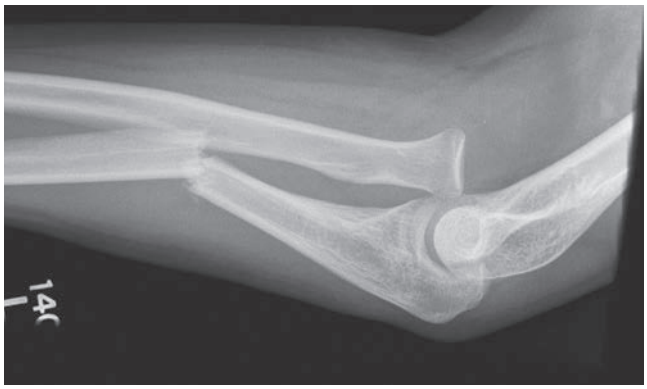


B

FIGURE 43-5 (**A**) Galeazzi fracture dislocation. (**B**) Monteggia fracture dislocation.

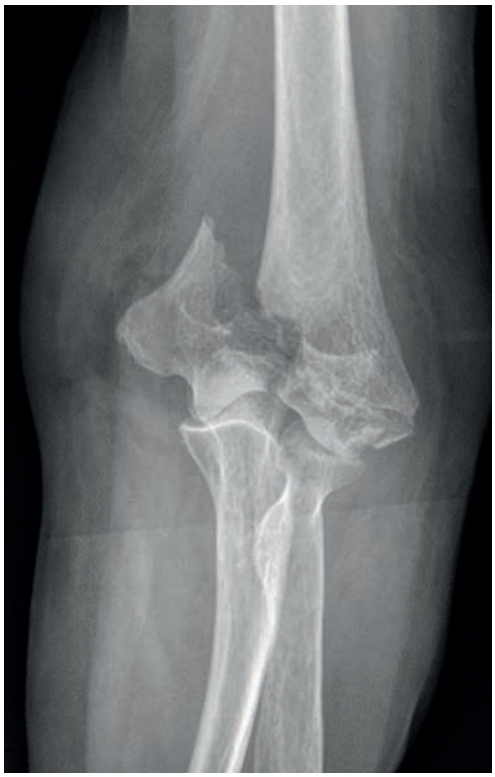
many cases, splinting with conversion to casting will provide definitive treatment in the closed management of fractures and dislocations. In other cases, splints provide optimal initial treatment until surgical stabilization occurs.[20]

Splinting materials should be readily available in any emergency department. Typically, a dedicated work station, platform, or cart should be maintained and well-stocked with undercast padding, differing widths of material, finger traps, and conforming and nonconforming overwrap.

The application of these splints should be performed by those with specific training, as improperly molded splints that are insufficiently padded or without appreciation for the underlying anatomy can be deleterious to both the skin (resulting in pressure sores or decubiti) and vascular tree (compromise secondary to an overly compressive wrap).

TRACTION

The application of traction, particularly to injuries of the lower limb, can also be extremely effective in mitigating pain. In addition, traction will counteract the shortening of the limb, resulting from the reflexive contraction of opposing muscle groups. In the upper extremity, traction is not commonly employed in the developed world, as appropriate splinting will yield to operative treatment for those fractures where anticipated shortening is expected.



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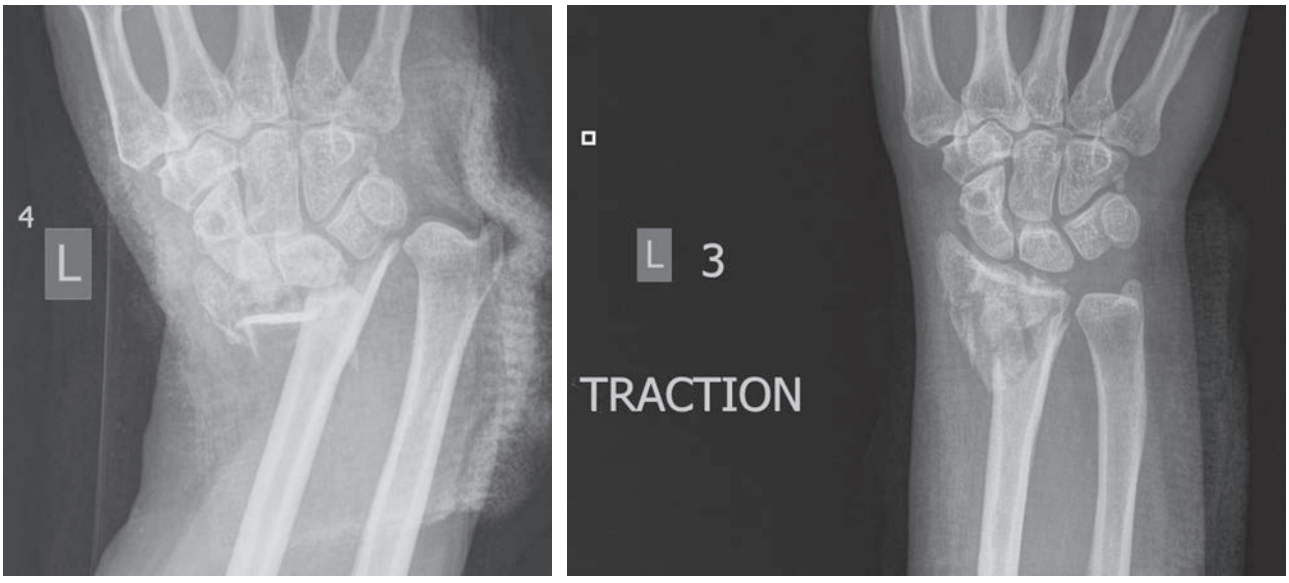

B

FIGURE 43-6 (**A**) Traction view of distal humerus. (**B**) Traction view of distal radius.

EXTERNAL FIXATION

The application of percutaneous wires and pins, tensioned with bridging bars and struts, has been an important component of fracture care for nearly 200 years. More modern techniques of external fixation were implemented by Hoffman.[21] In most patients, the external fixator is used to align fractures, whether as a temporizing or definitive method.[22] In other instances, external fixators are used as an extremely effective method of providing elevation to limit edema or controlling unstable joints to prevent recurrent dislocation and/or preventing disruption of surgical repairs of an artery, nerve, or the skin.

As temporizing treatment, fixators permit operative disassembly and re-creation of the traumatic deformity, allowing

TABLE 43-4: Skeletal Fixation Methods

Technique	Common	Pros	Cons
Plate fixation	Humerus	Anatomic fixation	Operative time
	Radius	Rigid	Blood loss
	Ulna	Immediate range of motion	Technique dependent
	Clavicle	Possible weight bearing	Dissection and soft tissue disruption
	Metacarpals	Low dependence on radiography	
	Metatarsals	No external orthosis required	
Intramedullary fixation	Femur	Strong	Malrotation possible
	Tibia	Early weight bearing typically permitted	Large radiography dependence
	Humerus	Immediate range of motion	Embolization of marrow contents
External fixation	Tibia	Limited blood loss	Pin tract infections
	Femur	Simple technique	Pin loosening
	Humerus	Fast	Expensive
			Bulky
Splint	Any appendicular bone	Cheap	Obscures soft tissue
		Easy to apply	Possible decubiti
		Fast	Immobilizes joint
		No operating room requirement	Least rigid
Cast	Any appendicular bone	Cheap	Obscures soft tissue
		Easy to apply	Decubiti
		Fast	Immobilizes joints

for thorough repeat debridement. For the patient who is critically ill, external fixation provides a means of damage control orthopedics to mitigate the adverse consequences of instability of a long bone (pain, extravasation of marrow contents, inflammatory cascade).[22]

In the upper extremity, external fixation is most commonly employed for fractures of the humeral shaft and distal radius and unstable fractures and dislocations of the elbow.[23]

INTERNAL FIXATION

The modern orthopedic surgeon is trained to incorporate a knowledge of extremity anatomy and biomechanical properties of implants to achieve fixation in the least disruptive means possible. Routine instruction in cadaveric anatomy, formal and informal didactics, and "saw bones" courses provide the orthopedic trainee the opportunity to learn the rapidly evolving techniques of skeletal fixation.

Plating. Although more disruptive to the soft tissues, plating fractures of the long bones of the upper extremity (humerus, radius, and ulna) results in high union rates.[24] Additionally, this technique facilitates early rehabilitation and nursing care. Intramedullary implants, although biomechanically superior in fatigue and load (failure strength) compared to plates, have not become quite as popular. The skeletal structure of the upper extremity does not favor intramedullary implants, because they are less likely to provide adequate rotational or longitudinal stability to permit immediate and unrestricted weight bearing.

Intramedullary Fixation. Intramedullary fixation for almost any tubular bone of the upper extremity is "friendly" to soft tissue. In ballistic injuries of the humerus and in some mangling injuries of the forearm, especially those involving the ulna, intramedullary fixation is preferred. As noted earlier, rotational stability is comparatively difficult to provide to the upper extremity, however, because the intramedullary diameters of the forearm bones do not permit implant sizes amenable to stout locking bolts and targeting guides. Yet, blood loss and surgical time are decreased compared to plating techniques.

Tension Band and Interosseous Cerclage. Interosseous cerclage provides an effective means of restoring skeletal anatomy in certain fracture patterns, particularly those with comminution, small fragments, or ligamentous avulsion injury.[25]

MANAGEMENT OF SPECIFIC FRACTURES AND DISLOCATIONS

A review of the diagnosis and management of fractures and dislocations in the upper extremity is provided in the following sections, and a summary of operative indications is provided in Table 43-5.

Sternoclavicular Dislocation

Injuries of the sternoclavicular (SC) joint occur with high-energy trauma. Notoriously difficult to diagnose with physical examination and plain radiography, posterior dislocations of this joint can result in significant morbidity. For example, reports of injuries to the trachea, esophagus, and brachial plexus are common in patients with untreated posterior SC dislocations.[26]

TABLE 43-5: Important Clinical Presentations and Their Associated Findings: What Not to Miss

Condition	Do not miss!!!	Comment	Treatment
Sternoclavicular dislocation	Posterior displacement into the mediastinum	Thoracic surgeon recommended for reduction	Reduction (closed if possible)
Scapular fractures	High index of suspicion for other serious, possibly life-threatening problems	Rule out injury to the chest, cervical spine, or neurovascular structures	Workup for other associated injuries
Proximal humeral fractures	Nerve injuries are common, especially involving the axillary nerve	A vascular injury may be present even if a radial pulse is palpable, due to the presence of multiple collateral vessels around the shoulder	
Proximal humeral fractures	Open fractures into the armpit may be small and overlooked	Small puncture lacerations are at high risk for severe infections of the chest wall (such as necrotizing fasciitis)	Early recognition of aggressive irrigation and debridement in OR and broad-spectrum IV antibiotic treatment
Humeral shaft fractures		The radial nerve is at highest risk, particularly in the distal third of the shaft	Coaptation splint is effective for most humeral shaft fractures
Elbow trauma	Care should be taken to identify associated injuries, which may include trauma to the median, ulnar, and radial nerves, as well as the brachial artery	Note this is a complex set of articulations including the ulnohumeral, radiocapitellar, and proximal radioulnar joints	
Elbow dislocations	Nerve and vascular injuries	Always assess and note stability during range of motion after reduction	
Elbow dislocation, complex type	The "terrible triad of the elbow"	Fracture-dislocation of the radial head, coronoid, and injury to the collateral ligaments	Surgical treatment is required with reconstruction to restore stability to the elbow
Supracondylar elbow fractures	Nerve injuries most commonly involve the median nerve or its anterior interosseous branch; rule out brachial artery injuries	Nerve injuries often recover with expectant management	
Distal radius fracture	Acute carpal tunnel syndrome	This is a "compartment" syndrome involving the median nerve	Urgent surgical decompression
Proximal radial head fractures	Associated injuries to the distal radioulnar joint with proximal migration of radius and injury to interosseous membrane	This is termed *Essex-Lopresti* fracture-dislocation	Check for elbow and wrist stability and range of motion
Distal radial shaft fractures	Associated distal radioulnar joint dislocation	This is termed *Galeazzi fracture-dislocation* and is more common in children	
Radius and ulna shaft fractures	Associated risk of compartment syndrome has been reported		
Wrist pain	Scaphoid fractures	These can be difficult to see radiographically on presentation	Conventional wisdom is to follow the clinical exam and picture more than initial radiographs
Hook of hamate fractures	Both ulnar and median nerve injuries may be associated with hook fractures	A thorough examination of motor and sensory function should be documented	
Perilunate and carpal dislocations	Associated median nerve injury is common		
"Fight bite" or clenched fist injuries	Septic arthritis of metacarpophalangeal joint	Always assume that the tooth entered deep into the joint	Systemic antibiotic and surgical arthrotomy and irrigation in the OR are the rule

(*continued*)

TABLE 43-5: Important Clinical Presentations and Their Associated Findings: What Not to Miss (*Continued*)

Condition	Do not miss!!!	Comment	Treatment
Felon	Direct extension of infection to periosteum	Associated bone involvement of this deep infection	Deep incisions and drainage
Compartment syndrome	Do not wait for paresthesia, pallor, or pulselessness	Pain out of proportion should be enough to raise suspicion	Fasciotomies should be performed emergently
High-pressure injection injuries	Small puncture wound but these injuries travel proximally down the fascial planes	Verify the type of solution injected because oil-based paints create chemical burns	Wide incision and drainage with debridement of all paint material

IV, intravenous; OR, operating room.

In the patient with tenderness about the SC joint or asymmetry on plain radiography, a CT examination is warranted. In addition, CT angiography provides opacification of the large, proximal arterial and venous structures adjacent to the SC joint (Fig. 43-7).

Reduction of posterior SC dislocations is mandatory, preferably at a center with cardiothoracic surgery available in the unusual event that a major vascular injury is present as well. Suture, transarticular plating, and ligament reconstruction are all reasonable methods of treatment. With closed treatment, recurrent dislocation is possible. Additionally, recurrent instability is difficult to detect and requires follow-up CT, further supporting open reduction and fixation.[27]

Anterior dislocations carry less morbidity and are generally treated with closed reduction. Although recurrent instability can be common, this does not carry the same danger as unreduced or recurrent posterior dislocations.[28]

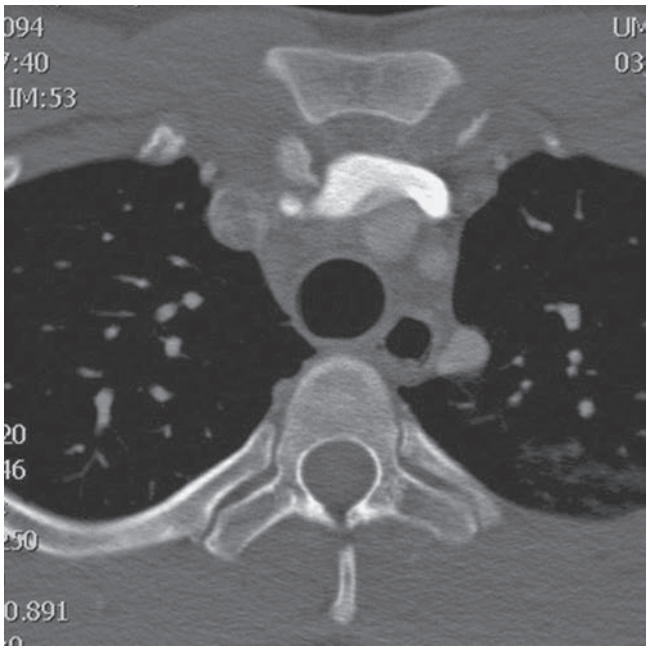

FIGURE 43-7 Axial computed tomography image of posterior sternoclavicular dislocation.

Fracture of the Clavicle

Fractures of the clavicle are extremely common, particularly in the polytrauma patient. Diaphyseal fractures of the clavicle are much discussed and have been the subject of quality randomized controlled trials (RCTs). A recent meta-analysis of these studies suggests that, although functional outcomes are modestly improved with surgical treatment, secondary operations are common, particularly as they relate to symptomatic hardware.[29]

Recent studies suggest that mid-shaft fractures displaced more than 100% may benefit from operative treatment, citing improved shoulder outcome scores. In many surgeons' opinions, this potential benefit is often outweighed by the possible risks of operation, including significant hemorrhage, need for removal of symptomatic hardware, anterior chest wall numbness, and unsightly scarring.[30,31]

No study to date has validated early operative fixation to facilitate rehabilitation. In the author's experience, patients with diaphyseal clavicle factures who are permitted unrestricted weight bearing on the upper limb for transfers, gait training, and activities of daily living (ADLs) do so quickly and without an apparent increased risk of nonunion. At the Shock Trauma Center at the University of Maryland, operative treatment for clavicle fractures is reserved for open injuries, evolving neurologic deficit, and in rare patients, injuries requiring vascular access or stabilization for vascular reconstruction.

Fractures of the lateral third of the clavicle, particularly those that are displaced and involve disruption of the coracoclavicular ligament complex, will often progress to symptomatic nonunion and the case for operative intervention is more substantial.[32]

Fracture of the Proximal Humerus

Neer's original description of proximal fractures of the humerus describes the following four parts: greater and lesser tuberosities, head, and shaft fragments.[33] The fragment qualifies as a "part" if displaced more than 1 cm or angulated more than 45°. Quality research has added to our understanding of the management of these injures, particularly in the elderly, where nonoperative and operative outcomes are similar.

In open injuries (commonly two-part fractures), careful assessment is indicated. Injury to the axillary nerve, brachial

plexus, and axillary or brachial vessels can occur. For two-part fractures in the young polytrauma patient, rigid fixation with either an intramedullary implant or a locking plate permits early functional return and facilitates rehabilitation. The results between these two techniques have been widely studied with similar long-term functional outcomes.[34]

In three- and four-part fractures, increased complications are noted, both in young and aging patients. A recent RCT comparing open versus closed treatment in elderly patients found such an outcome.[35] This study excluded patients who "required" operative treatment. Thus, significant debate remains regarding whether displaced fractures of the proximal humerus are better served with operative versus nonoperative treatment,[36] particularly in those age 60 years or older. In general, fractures in the young patient with displacement more than 1 cm or 45° of angulation are generally deemed operative with careful consideration of comorbidities.

In the elderly patient, fractures of the proximal humerus represent an area of significant controversy. Open reduction results in not uncommon hardware complications, whereas hemiarthroplasty often results in residual pain and inferior shoulder scores. More favorable results have been achieved with reverse arthroplasty, balanced at the expense of significant bone resection and lack of a good option for revision. Operative treatment is indicated for nearly all glenohumeral fracture-dislocations. In fracture-dislocations in young patients, open reduction and fixation is recommended because it preserves the skeleton and associated muscle units. In the elderly patient, good results have been achieved with arthroplasty.[37]

Fractures of the Shaft of the Humerus

In isolation, fractures of the shaft of the humerus are successfully and reliably treated with bracing.[38] Bracing depends on the coactivation of the biceps and triceps muscles, gravity, appropriate careful molding, and routine clinical follow-up. A mild varus deformity can be expected, without significant functional or cosmetic detriment. Although some studies suggest earlier return to function with an open operation, splinting results in acceptable alignment and functional outcomes in the majority of patients.[39]

In the polytrauma patient, treatment of the fractured humerus warrants more careful consideration. In a patient with an injury to the contralateral upper extremity, rigid internal fixation provides immediate functional benefit, as previously noted. Feeding, hygiene, and other routine ADLs are permitted immediately. With robust fixation, the patient with a fractured humerus is also permitted immediate and unrestricted weight bearing, allowing for crutch training, transfers, wheelchair propulsion, and so on.[40] In a patient with an associated injury to a lower extremity, it has been found that plate fixation of the humeral fracture reduces length of hospital stay, allows for discharge to lower level of care facilities with improved access to therapists, and leads to overall decreased inpatient rehabilitation stays.

In certain instances, intramedullary nail fixation of the humerus is preferred, as it generally requires less surgical time and less blood loss. External fixation is also an effective means of treatment. Union rates, functional outcomes, and complication risks are similar across these treatment methods.[41,42]

Fracture of the Distal Humerus

Fractures of the supracondylar humerus, without intra-articular extension, are also amenable to closed treatment. Careful bracing and prescribed therapy are important in achieving satisfactory outcomes. Similar to the humeral shaft, patients who have sustained injuries to both an upper and lower extremity will benefit from operative treatment and rigid internal fixation, in order to permit ADLs.[43] Weight bearing is generally prohibited because fractures in the periarticular region are not as amenable to fixation that provides an adequate biochemical milieu for early healing.

Extension of the distal fracture into the intercondylar area generally requires operative treatment to obtain the best clinical result. Fixation of the components of the joint permits immediate range of motion (ROM), and in many cases, splinting can be avoided and ADLs encouraged.[44]

Dislocation and Fracture of the Elbow

Simple elbow dislocations, which concentrically reduce, are treated closed. Assessment of stability is necessary, in order for a guided rehabilitation program. Typically, in the awake, alert, and cooperative patient, the dynamic stabilizing muscles (biceps, triceps, brachialis) permit immediate ROM to tolerance and ADLs. In the obtunded patient or one lacking in muscle stabilizers, splinting at 90° and assisted ROM are prescribed. Limits are set by the provider assessing the stability of the elbow after reduction, increasing the prescribed ROM over a period of several weeks.

In dislocations involving the key stabilizers of the elbow (radial head, coronoid, trochlea, and capitellum), operative treatment is generally indicated. Restoring anatomic joint relationships is critical in achieving stability and a satisfactory outcome. Mini-plate fixation, intramedullary implants, radial head arthroplasty, and precontoured plates are currently available options.[45]

Patients in whom the elbow will require additional stabilization (external fixation) have undergone ligamentous repair or fixation of small but critical components such as the coronoid or radial head. This is especially true in patients who have a concomitant injury to a lower extremity or are obese. These fixators can be unlocked by the therapist, permitting supervised ROM, and then locked again. This will prevent recurrent dislocation in the patient whose arm is not satisfactorily braceable or in the patient who assumes a functional quadruped status secondary to their other injuries.[46] Methods of external and internal hinged fixation are now available as well.

Fracture of the Shaft of the Radius

Displaced fractures of the diaphyseal radius in adults are fractures "of necessity." The actions of the brachioradialis, supinator,

and pronator muscles act upon the radius to cause a predictable deformity. The resulting malunion and associated disability are not prevented with closed, nonoperative treatment.[47] Therefore, rigid internal plate fixation is required for this fracture.[48]

In certain patients, a fracture of the radial shaft will result in dislocation of the distal radioulnar joint. This is commonly referred to as a Galeazzi fracture (Fig. 43-5A). Anatomic plate fixation of the radius fracture will typically result in reduction of the distal radioulnar joint. The injury is then splinted for a period of several weeks, with gradual resumption of prosupination to allow satisfactory healing of the disrupted distal radioulnar joint.[49]

Fracture of the Shaft of the Ulna

An isolated fracture of the ulna shaft does not usually result in malalignment if appropriate splinting is performed; thus, closed treatment is often satisfactory. In patients with bilateral upper extremity fractures, an argument can be made for operative fixation to permit unrestricted range, weight bearing, and ADLs.[50]

Monteggia Fracture

A fracture of the proximal ulna requires a careful radiographic examination of the proximal radioulnar joint. An associated dislocation of the radial head is termed *Monteggia fracture* (Fig. 43-5B). Difficulty in maintaining reduction of the radial head with closed treatment of the ulnar fracture has led to the wide adoption of anatomic and rigid fixation of the shaft of the ulna. In patients in whom the radial head fails to reduce, open reduction of the proximal radioulnar joint is accomplished by extraction of the entrapped annular ligament.[51]

Combined Fractures of the Shafts of the Radius and Ulna

Displaced fractures of the shafts of the radius and ulna, commonly referred to as *both bones* fractures, are treated with rigid plate fixation. Accounting for the anatomic contours of each bone, plate fixation results in union rates of better than 98%. In many patients, plate fixation will permit unrestricted activities immediately postoperatively, including ADLs and full weight bearing.[52]

Fracture of the Distal Radius

Fractures of the distal radius are grouped into extra- and intra-articular injuries. Restoration of the distal anatomy of the radius, accounting for the relationship of length to the ulna, palmar tilt, radial inclination, and articular congruity, is required for an optimal result in young patients. In sedentary patients, malalignment is felt to be more tolerated. Consensus opinion regarding exact age cut-offs over 50 and the degree of acceptable malalignment that will still permit satisfactory outcome does not exist.[53]

Partial articular fractures generally warrant operative treatment regardless of patient age or activity level. These fractures have a propensity to shorten significantly with resultant "point loading" and rapid degenerative arthritic changes.

High-energy articular fractures with multiple joint fragments are typically treated operatively. In the young patient, optimal outcomes require anatomic restoration of alignment and joint congruity. Current techniques favor neutralization of the forces that lead to shortening and impaction, typically by either external fixation or internal fixation with a spanning bridge plate, followed by anatomic restoration of critical joint fragments using mini-locked plates.[54]

Fracture-Dislocation and Fracture of the Carpal Bones

Perilunate dislocations are common in patients after high-energy trauma. Lunate dislocations are common and frequently misdiagnosed, leading to significant morbidity. A compressive median neuropathy results from complete lunate dislocation, and urgent reduction is required for an optimal result.[55]

Careful examination of wrist radiographs is paramount. Fractures of the other carpal bones in high-energy trauma may be present, and careful inspection of Gilula's lines is mandatory (Fig. 43-8). Disruption of these arcs represents an intrinsic carpal ligament injury. Disruption of other carpal relationships may also represent longitudinal axial disruptions. In patients in whom an abnormality is suspected, contralateral wrist views can aid in the diagnosis of intercarpal instability.[56]

Fractures of the scaphoid can lead to significant disability if not recognized and treated appropriately at presentation. Displaced fractures of the scaphoid (>1 mm) require operative treatment, as union is delayed and nonunion is common with attempts at closed management. Nondisplaced fractures are effectively treated with immobilization of the thumb in a Spica cast for a period of 6 to 8 weeks.[57]

In patients with tenderness in the anatomic snuffbox, a CT scan or magnetic resonance imaging (MRI) can be an effective method for diagnosis, with sensitivity of more than 95%.[58] If any question regarding the diagnosis remains, immobilization with a thumb Spica splint for 3 weeks is appropriate until repeat x-rays are obtained. Delay in immobilization will frequently result in further displacement of the fracture, prolonging the time to union, altering carpal kinematics, and causing a possible nonunion.

Fractures of the carpometacarpal joints, particularly in patients after a motorcycle crash, can represent fracture-dislocations and other complex injuries. The base of the thumb is frequently dislocated by a hyperextension force, resulting in rupture of the palmar oblique and dorsal radial ligaments and chronic instability.[59] Thus, avulsion injuries in the hand require careful assessment for stability.

Fractures of the Metacarpal Bones

Nondisplaced fractures of the metacarpal bones are generally treated with splinting and early mobilization of the

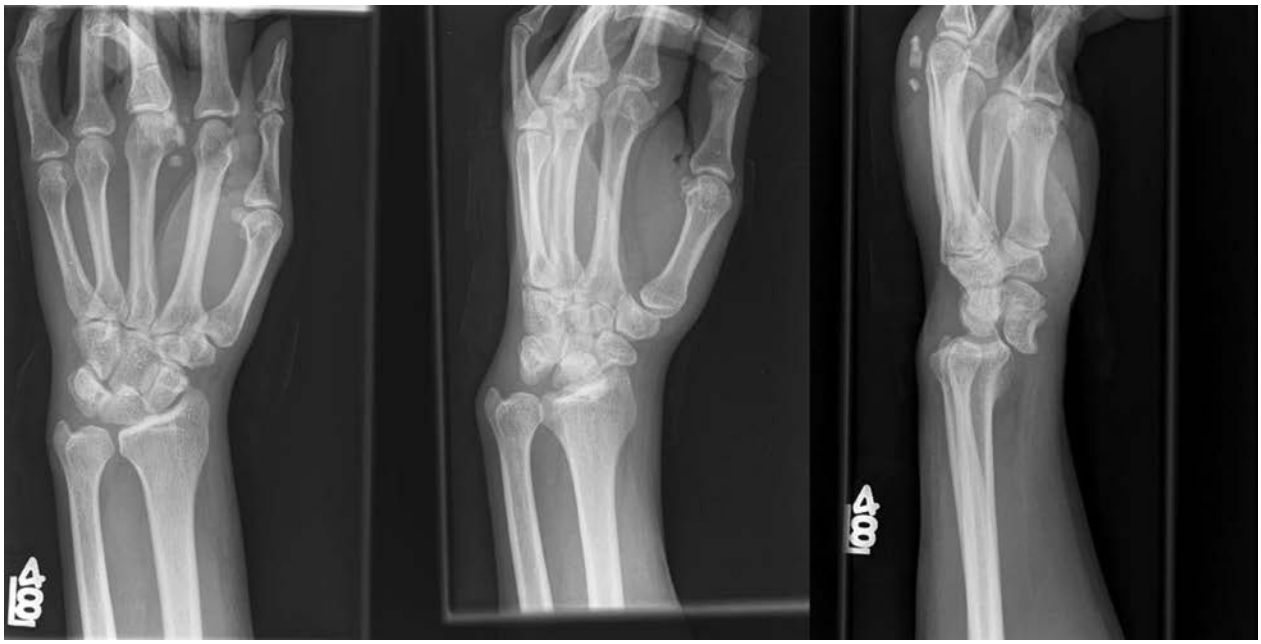

FIGURE 43-8 Anteroposterior radiograph of perilunate dislocation with disruption of Gilula's lines.

interphalangeal joints. These fractures will heal in 3 to 4 weeks, permitting relatively early rehabilitation.[60]

Metacarpal fractures that are displaced and result in significant malangulation or shortening, particularly when multiple, require operative treatment. Restoration of the rotational malalignment is critical. As the patient regains flexion at the metacarpophalangeal joint, malrotation can manifest in disabling "scissoring." Sagittal malangulation of the metacarpal necks (15° for index, 25° for middle, 35° for ring, and up to 45° for the small finger) is accepted as the motion at the proximal carpometacarpal articulations permits compensatory motion in the sagittal plane.[61] Greater malangulations are an indication for operative treatment. Fixation techniques for the metacarpal bones include transmetacarpal pinning, intramedullary "bouquet" pinning, plate fixation, and external fixation.

Fracture of the Phalanges

Nondisplaced fractures of the phalanges are treated closed, also. Stiffness of the proximal phalangeal joints after trauma or surgical dissection is frequent and may take many months to resolve.

Slight malalignment in any plane results in an obvious and disabling clinical deformity. Displaced fractures of the phalanges are typically treated with K wire fixation, although the proximal phalanx is amenable to plate fixation as well.[62]

Displaced fractures involving the metacarpophalangeal joint typically represent ligamentous avulsion or impaction. Displacement of more than 1 mm requires operative treatment. Fractures involving the proximal interphalangeal joint are notoriously difficult to treat. Small (1.0 mm) screws are often required, as are thin Kirschner wires (0.035/0.028). External fixation for fracture-dislocations of the proximal interphalangeal joint can be an option.

VASCULAR INJURIES IN THE UPPER EXTREMITY (SEE CHAPTER 45)

Physical Examination

Assessment of a presumptive vascular injury should be rapid. Patients presenting with a tourniquet in place represent one of the few true limb-threatening emergencies. A thorough history should be obtained from the emergency medical service personnel to include mechanism of injury, presence of pulsatile bleeding, presence or absence of movement of the hand or arm prior to placement of the tourniquet, and exact time of placement. Typically, the upper extremity with an appropriately applied tourniquet will lose motor function approximately a half-hour after application. Field tourniquets are applied as a lifesaving intervention but do not afford controlled application of pressure. In the setting of the appropriately applied tourniquet, one must remember that the limb that was frequently *relatively* ischemic with only collateral flow is now rendered *absolutely* ischemic. Thus, the ongoing muscle ischemia represents an emergent condition. Within 4 hours of ischemia, skeletal muscle cells begin to undergo irreversible cell death, with deleterious increases in serum lactate, potassium, and myoglobin.[63,64]

Once a primary assessment and ATLS protocols have been completed, removal of the tourniquet should be completed in a controlled fashion. If pulsatile bleeding occurs, replacing the field tourniquet with a standard pneumatic tourniquet is necessary. This permits hemostasis with a known pressure over a broader and safer area. If pulsatile bleeding is anticipated by the obvious nature of the wound, immediate operative intervention is required. The tourniquet should be left in place until the patient is positioned on the operating room table and

anesthetized. Then, under appropriate conditions with excellent lighting, the surgical team removes the tourniquet while providing direct pressure, rapid exploration, and vascular control.

In patients in whom the bleeding can be effectively controlled with a compressive dressing, the tourniquet should be removed to permit collateral flow, even in patients with a presumed injury to the brachial artery.[65] In the upper extremity, the presence of collateral flow will permit adequate perfusion to prevent irreversible muscle damage during an appropriate secondary survey and diagnostic testing.[66]

A palpable pulse in the normotensive patient nearly eliminates the possibility of a vascular insult requiring operative intervention. In the patient without a palpable pulse, adequate assessment of the color of the limb under appropriate lighting is valuable. Assessment with an incandescent light source or flashlight to assess capillary refill, turgor, and color of the digits can be very helpful.

In addition to the "hard signs" of vascular injury (eg, pulselessness, pale limb, pulsatile bleeding, expanding hematoma), "soft signs" may alert the clinician to a vascular injury. A penetrating injury immediately adjacent to the course of a major vessel, widely displaced fractures or dislocations, a peripheral nerve deficit, or nonpulsatile hemorrhage should be taken into account and may warrant angiography.[67]

Diagnostic Modalities

PENCIL DOPPLER

In a patient with an absent pulse and color difficult to assess, a pencil Doppler can provide important data. First, one must ensure the Doppler is functional. Using the examiner's own finger as a baseline exam, the pencil Doppler is placed at the "swirl" of the fingerprint. A properly functioning Doppler will easily provide biphasic signal of the small digital arteries in the normotensive healthy examiner and then can be used to verify arterial patency for the injured patient. Once confirmed, a Doppler examination of the brachial artery medial to the biceps tendon, the radial and ulnar arteries, and the palmar arch is completed. A Doppler Allen test, completed by sequential and independent compression of the radial and ulnar arteries at the wrist, can provide data as to the patency of each. A Doppler examination of the fingers can also be completed to assess the perfusion of the digits. The patient who is hypotensive or peripherally vasoconstricted will have altered perfusion of the distal extremities, complicating the examination.

CT ANGIOGRAPHY

CT angiography has generally replaced formal angiography in the initial assessment of the vascular tree. This modality permits rapid and accurate assessment with sensitivities and specificities above 95%.[68,69] In the limb with a well-defined zone of injury, CT angiography is not required for diagnosis.

Treatment

PRIMARY REPAIR

Well-localized vascular disruptions are suitable to primary repair.[70] Commonly, the mechanism of injury alone will provide the surgeon a reasonable plan of action. Injuries that result from a penetrating focal laceration (eg, knife, glass, metal, shrapnel, or iatrogenic transection) are generally amenable to resection of a small portion of the injured vessel and a lateral repair or an end-to-end anastomosis.[71]

SHUNT (TEMPORARY INTRAVASCULAR SHUNT)

Temporary urgent intraluminal shunting as the initial maneuver can often be predicted by the mechanism of injury. An injury to the brachial artery after a rollover motor vehicle crash, shotgun blast, or any crush injury implies a lack of collateral flow, as the resultant zone of injury can be large. In such a patient, insertion of a shunt to expeditiously restore flow is appropriate[72] (Fig. 43-9).

The shunt allows the surgical team adequate time for the critical steps of a tumor-like debridement. In many patients in whom a shunt is required, the zone of injury is significant, as is the potential for contamination and muscle necrosis. A debridement beginning through a zone of normal, uninjured tissue is recommended. Similar to that of resection of a sarcoma, the dissection is begun in normal, noncancerous tissue, respecting neurovascular planes and intervals. In this manner, a complete debridement is ensured. The debridement is often the longest and most critical part of limb salvage and can be completed in a thorough and nonrushed manner through restoration of arterial inflow (and/or venous outflow) by the shunts.

Temporary intravascular shunts are also extremely helpful in the setting of a vascular injury with concurrent fracture of a long bone (ie, Gustilo 3C). By rapidly inserting a temporary intravascular shunt, the orthopedic team can then bring the bone out to length and fixate it at this position in a controlled manner. Once fixed, the trauma/vascular surgeon is then able to reconstruct the vascular injury in a limb that will not stress the subsequent repair.[72]

INTERPOSITION VEIN GRAFT

In instances where injury of the vessel involves more than 2 to 3 cm, vascular reconstruction with an interposition vein graft is recommended. In the upper extremity, the cephalic, basilic, and associated brachial veins are suitable graft sources and rapidly employed. These veins are frequently dissected during the initial

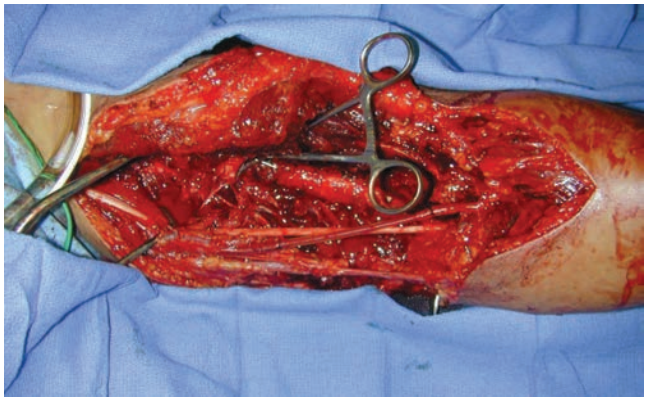

FIGURE 43-9 Shunt of brachial artery.

exposure, and care should be taken to preserve them.[73] Inspection of these veins during the approach will identify obvious trauma. If the local veins are traumatized, a greater saphenous vein from the thigh or ankle is preferred. In order to facilitate a two-team surgical approach, the contralateral greater saphenous vein is harvested, as the vein harvest team will be positioned on the opposite side of the operating table from the injured upper limb. This also highlights the need to prepare and drape the entirety of a lower limb (ie, even with an isolated injury to an upper extremity) in cases that may require a vein graft.

Management of Specific Vascular Injuries

SUBCLAVIAN AND AXILLARY ARTERIES

Traction injuries to the vessels are often devastating because they are frequently associated with severe injuries to the brachial plexus. Fortunately, significant progress has been made with endovascular stenting of proximal arterial lesions.[74]

Injuries that require exploration for repair or grafting will require familiarity with the brachial plexus. As the primary determinant of outcome, an associated injury to the brachial plexus warrants special attention. A perfused but inanimate hand is of little, if any, use to the patient. Therefore, identification and preservation of the brachial plexus are mandatory during open exposure of either the subclavian or axillary artery. Observation of a transection or avulsion should be communicated early to the "peripheral nerve" surgeon, preferably for intraoperative consultation. Generally speaking, the functional outcome of an injured upper extremity is tied to the integrity of major peripheral nerves. Therefore, the most appropriate time for proper identification and inventory is during the initial exposure for arterial repair. In cases of sharp transection of a trunk, division, or cord of the plexus, immediate repair will often result in a reasonable functional outcome. In those instances where repair is not possible, insertion of a nerve graft similarly can provide a good, if not excellent, result. If neither grafting nor repair is possible, identification of the injured nerves is critical. Nerve transfers are now recognized as a valuable tool in providing recovery and can be implemented early, provided the surgeon has an accurate understanding and identification of the nerves that are deemed unreconstructible.[75] Nerve transfers, when performed early, facilitate expeditious recovery.[76]

BRACHIAL ARTERY

For injuries of the brachial artery, shunting is performed when critical ischemia is present. Fortunately, the mechanism of injury will often predict the degree of ischemia, as previously noted.[77] A shotgun blast at the humeral level, crush injury, or entrapment will frequently result in disruption of the profunda brachii artery, as well as the ulnar and radial collateral systems. With disruption of these collaterals, shunting provides immediate reperfusion of the forearm muscles.

Fortunately, the upper extremity maintains a robust collateral system about the elbow (Fig. 43-10A). The superior and inferior collateral systems medially, as well as the profunda brachii and associated radial collateral systems, will maintain perfusion sufficient for at least short-term viability in many cases. Intraoperatively, assessment of perfusion will occur based on the color of the hand, the contractility of the forearm muscles, and back-bleeding from the surgically identified and isolated distal arterial stump.

RADIAL AND ULNAR ARTERIES

An injury to either the radial or ulnar artery, in isolation, will typically not result in ischemia of the hand or forearm (Fig. 43-10B). Standard clinical examination to include assessment of color, capillary refill, palpation of pulses, and an Allen test should be a routine part of every examination of the hand and upper extremity with significant trauma, as previously noted.

Importantly, an innocuous low-velocity gunshot wound or penetrating sharp injury to the forearm can result in a significant hematoma and a possible compartment syndrome. Injury to the anterior interosseous artery, in particular, can result in significant bleeding within the deep compartment of the forearm, resulting in the compartment syndrome cascade of swelling, ischemia, nerve dysfunction, and muscle necrosis.

In instances where either the radial or ulnar artery is injured and subsequently repaired, only 47% are found to be patent at 1 year.[78] In the same study, if both vessels are injured and only one repaired, patency rates approach 100%. In those cases where both were repaired, both remained patent in 41% of patients. This is an obvious demonstration of the robust collateral retrograde supply that the patent vessel maintains through a healthy palmar arch.

In patients in whom the surgeon chooses to maintain perfusion via repair of only one vessel (other vessel thrombosed or ligated), the author will confirm perfusion to each digit with capillary refill, a Doppler signal, and, if exposed, back-bleeding in the unrepaired vessel. Consideration is also given to achieving soft tissue coverage over any vascular repair. Additional consideration is given to the possible need for subsequent microvascular reconstructions (Fig. 43-11).

PALMAR ARCH AND DIGITAL ARTERY

The hand enjoys a robust collateral system, composed of the dual contribution of both the radial and ulnar arteries to both the deep and superficial palmar arches.[79] Each digit receives flow from two digital arteries, either of which is sufficient to maintain viability. In some cases, digital viability is maintained with the dorsal circulation alone.

The common digital and palmar metacarpal arteries provide additional redundancy, such that an isolated lesion of either the deep or superficial palmar arch will not cause critical ischemia (Fig. 43-12).

Again, assessment of color, capillary refill, and Doppler examination are the mainstays of evaluation when determining whether or not to undertake microvascular reconstruction in the hand or digits.

In any patient in whom there is question about the perfusion of the hand, there are many relatively conservative measures that can be taken. Warming the patient, adequate resuscitation to limit hypotension, warming the extremity with a warming blanket, ensuring skeletal stability (either

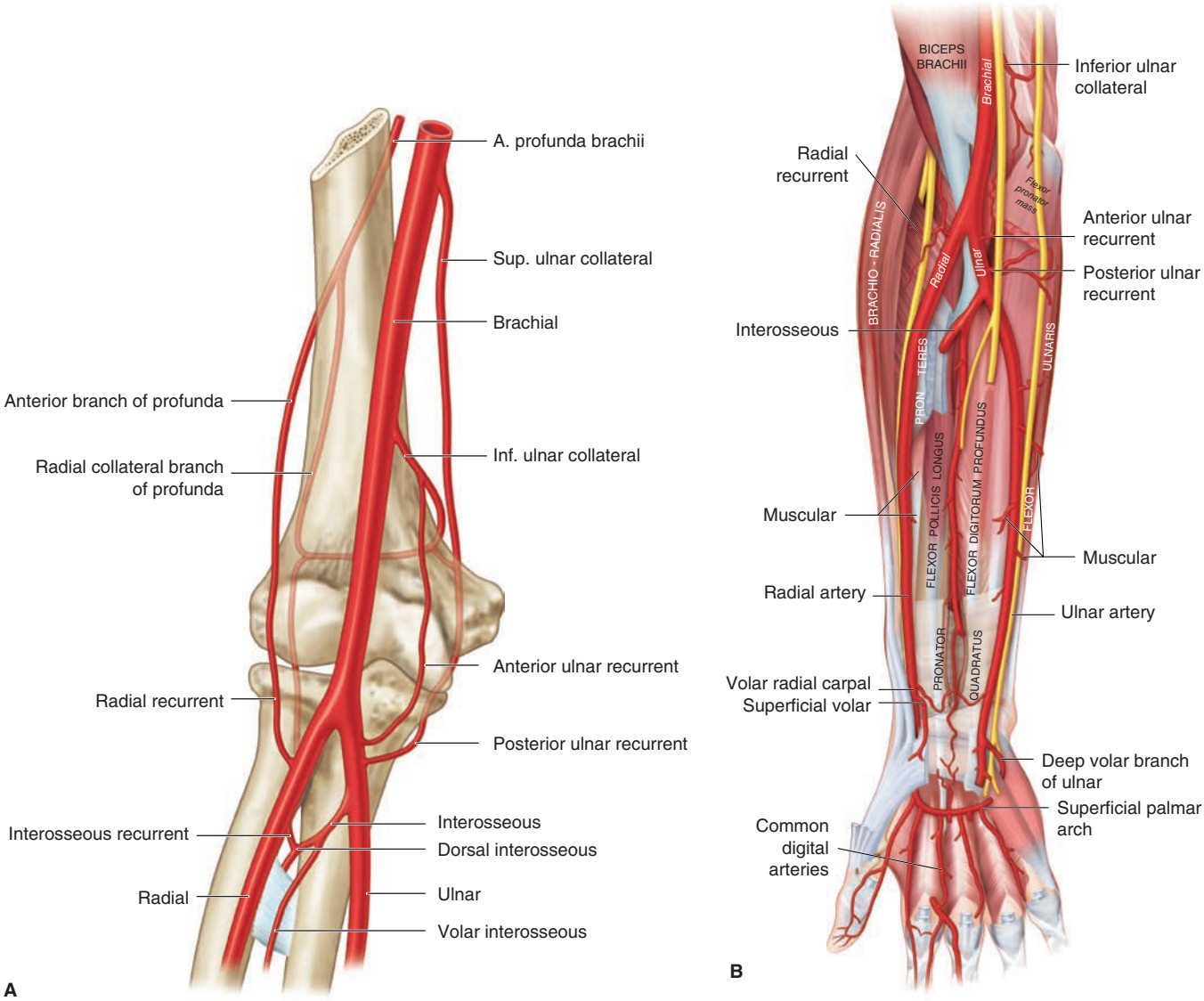

FIGURE 43-10 (**A**) Vascular tree of the arm. (**B**) Vascular tree of the forearm.

through operative fixation or splinting), and providing a regional anesthetic (infraclavicular, axillary, or wrist block) have all been shown to increase perfusion.[80,81]

In cases requiring repair, it is not uncommon to require suture of 8-0 caliber or smaller and the use of an operating microscope or high-power loupes (3.5× or more). In cases of significant trauma, vein grafts from the palmar wrist or dorsal foot are suitable for reconstruction.

NERVE INJURIES IN THE UPPER EXTREMITY

Physical Examination

To provide an accurate assessment of trauma to a peripheral nerve in the upper extremity, a thorough knowledge of anatomy beginning with the brachial plexus and each of its branches is required[82] (Table 43-6). Ideally, a complete examination requires a fully awake and compliant patient; however, even in a patient who cannot follow commands, simple observation of the upper extremity can provide the astute examiner a basic status of the integrity of peripheral nerves. Gross movement of the shoulder, biceps muscle, and wrist and finger motion will give the surgeon a reasonable indication of significant nerve trauma, important to rendering initial care and planning (Table 43-6).

Although the anatomy of the brachial plexus is complex, general injury patterns help the clinician understand the anatomy and pathoanatomy. In general, the plexus can be thought of in three main components corresponding to the trunks: those for shoulder function, elbow function, and wrist/hand function. The upper trunk and respective roots provide motor function via the suprascapular, axillary, and musculocutaneous nerves to the rotator cuff and deltoid,

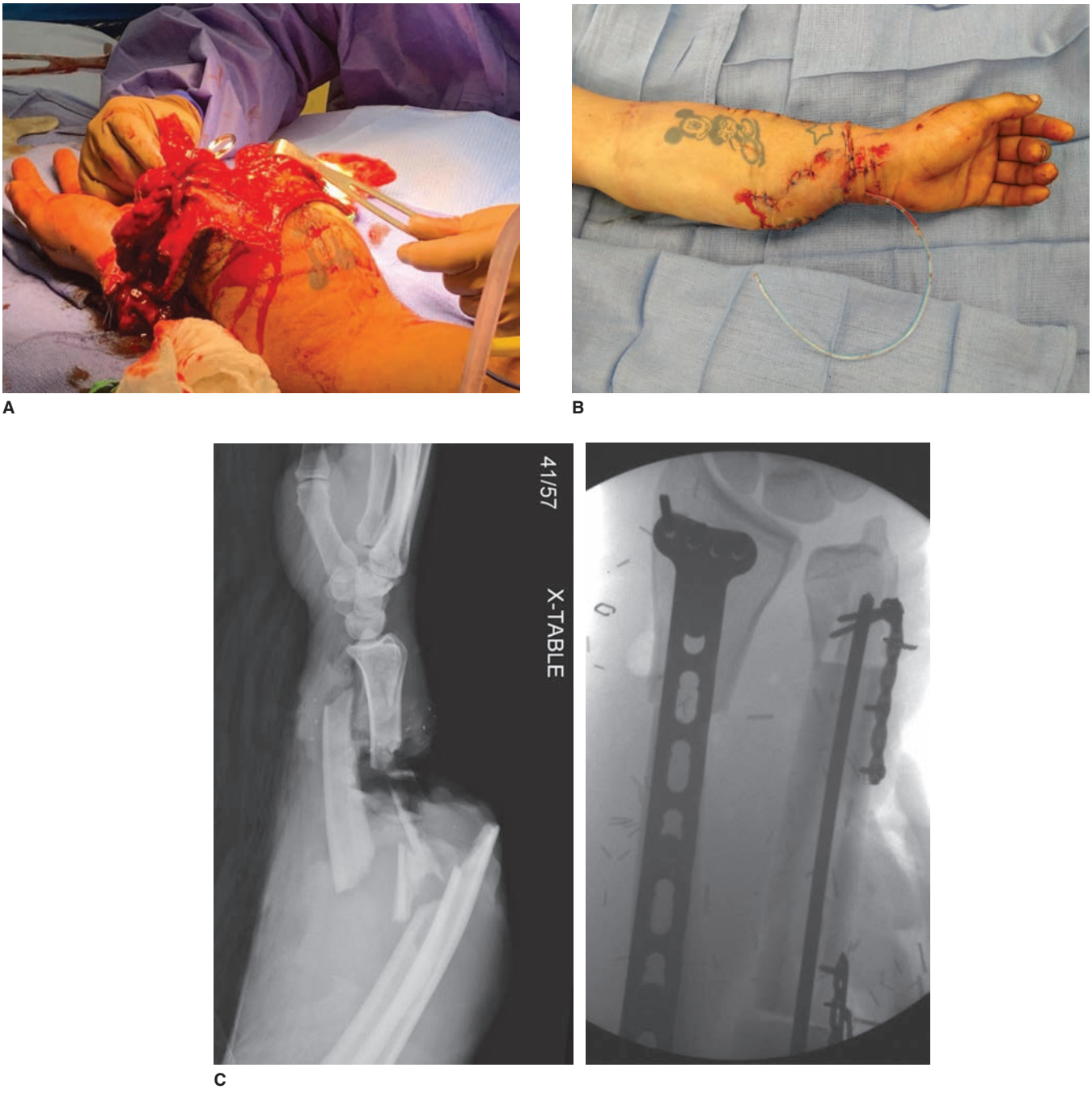

FIGURE 43-11 (**A–C**) Nearly amputated hand, repair of radial artery, and subsequent utilization of the ulnar artery for end-to-end microanastomosis of the osteocutaneous free fibula graft.

biceps, and brachial muscles, respectively. The C7 root and middle trunk are important to elbow and wrist extension through activity of the radial nerve, and the C8 and T1 (lower trunk) roots are important in the grip and fine motor function of the hand.[83]

Injuries to the adult brachial plexus and peripheral nerves can be the result of a variety of mechanisms.[84,85] Therefore, the author finds it helpful to consider the resultant zone of injury in planning and rendering treatment.

An initial part of triage always should include a description of the injury from the patient if possible. In high-energy trauma, however, this is often obtained from the first responder. Traction injuries are common to motorcyclists, and a detailed history from emergency medical service personnel regarding the accident can be helpful. For example, a motorcyclist with abrasions on his or her helmet or head suggest that the head and cervical spine have been forcibly deviated from the shoulder and plexus. This will commonly result

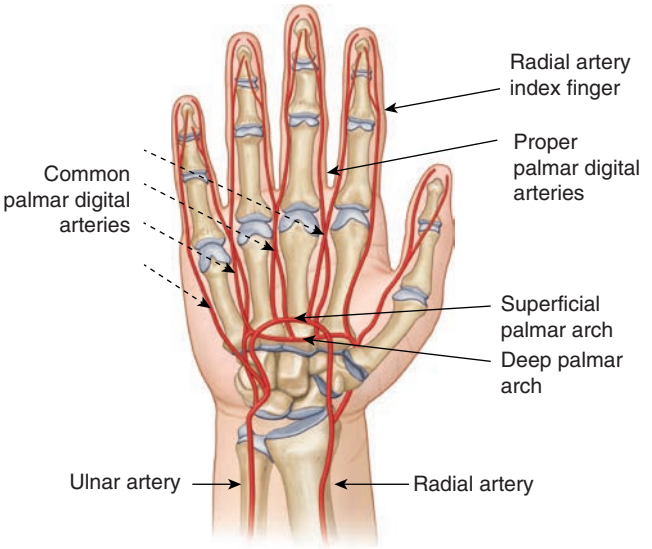

FIGURE 43-12 Vascular tree of the hand.

in an upper trunk lesion with paralysis of the shoulder and elbow.

The examiner should then test all major muscle groups of the upper extremity and their function, including, at a minimum, the following: shoulder abduction (deltoid); external rotation of the shoulder (supra- and infraspinatus, teres minor); elbow flexion (biceps/brachialis); elbow extension (triceps); wrist extension (extensor carpi radials brevis and longus); wrist flexion (flexor carpi radialis and ulnaris); finger extension (extensor digitorum); thumb extension (extensor pollicis longus); finger flexion (flexor digitorum superficialis and profundus); and, particularly, thumb interphalangeal flexion (flexor pollicis longus). The Medical Research Council (MRC) muscle grading system is used to document the degree of strength and deficit (Table 43-7).

Deficits in strength and sensation are documented clearly in the medical record and relayed accurately to other treating physicians so that close monitoring of neurologic function can be accomplished, particularly in patients at risk for a compartment syndrome or an evolving neurologic deficit.

Determination as to whether or not a traction injury is pre- or postganglionic is important. Preganglionic lesions are generally devastating because no useful recovery of an avulsed, preganglionic lesion can be expected. In regard to physical examination, Horner syndrome (ptosis, miosis, and anhidrosis) is a critical finding because it results from a preganglionic avulsion and disruption of the sympathetic innervation controlling these functions and portends a poor outcome.

Diagnostic Studies

Plain radiography and standard CT will not provide specific diagnostic data related to peripheral nerve trauma. A concomitant bone injury, however, can give the clinician an appreciation for the zone of injury or severity of trauma. For example, although the degree of nerve trauma is highly variable in scapulothoracic dissociation, a severely displaced scapula is of course a potential harbinger of a brachial plexopathy. Additionally, several fractures are worth noting as having a high incidence of associated injuries to nerves as follows: clavicle (brachial plexus[86]); shoulder dislocation (axillary and radial nerve injury, uncommonly median nerve[87]); humerus fracture (radial nerve); and Monteggia injury (posterior interosseous nerve).

NERVE CONDUCTION AND ELECTROMYOGRAPHY

Nerve conduction studies will provide objectification of the presence, absence, amplitude, and velocity of the conductive signal propagated by a major nerve in the upper extremity. They are rarely indicated in the immediate posttrauma phase. It will take at least 3 to 4 days for a nerve to demonstrate conduction loss after even severe trauma. Similarly, electromyography (EMG) changes (fibrillations, positive sharp waves) are not apparent until several weeks after nerve trauma. Thus, physical examination remains the mainstay in the diagnosis of acute nerve trauma, whether blunt or sharp.[88]

In the weeks and months following nerve trauma, electrodiagnostic studies (nerve conduction studies and EMG) can provide evidence of nerve recovery, or lack thereof, which will direct the clinician toward either further observation or intervention.[89] The decision to proceed with nerve reconstruction or tendon transfer in the absence of meaningful recovery is based on the wishes of the patient, the surgeon's knowledge of the severity of the nerve trauma, the expected odds of functional improvement with further observation or intervention, the availability of suitable donor nerves or tendons, and the ability of the patient to comply and adhere to the postoperative rehabilitation program.

Importantly, a preganglionic lesion can be characterized by nerve conduction velocities with absence of motor nerve action potentials, as the motor nerve cell body lies within the spinal cord. In contrast, sensory nerve action potentials are maintained, as the sensory cell body lies in the dorsal root ganglion. In the dermatome absent of sensation and corresponding motor deficit and the above findings, particularly with a Horner syndrome, the prognosis is grim.

MRI/CT MYELOGRAM

Advanced imaging of the brachial plexus can include MRI, CT myelogram, or magnetic resonance neurography.[90] In most cases, the history and physical examination will provide the diagnosis, and further clarification as to whether nerve trauma may recover or not will be provided with enough specificity to warrant immediate exploration in cases of blunt trauma.[91,92] In some instances, magnetic resonance neurography can provide images of sufficient quality to discern preganglionic avulsion, prompting early nerve transfers.

TABLE 43-6: Nerves and Muscles of the Upper Extremity

Nerve	Muscles innervated	Test for function	Sensory distribution
Spinal accessory	Sternocleidomastoid	Ipsilateral head tilt, contralateral head rotation; scapular elevation rotation, adduction: head extension, rotation	
	Trapezius		
Dorsal scapular	Rhomboid	Scapular retraction; scapular stabilization	
Suprascapular	Supraspinatus	Arm abduction	
	Infraspinatus	Arm external rotation	
Long thoracic	Serratus anterior	Scapular protraction: scapular stabilization	
Subscapular	Subscapularis	Arm internal rotation, adduction	
Thoracodorsal	Latissimus dorsi	Arm extension, internal rotation	
Pectoral (medical and lateral)	Pectoralis major and minor	Arm internal rotation, flexion, adduction	
Musculocutaneous	Biceps	Arm and forearm flexion; forearm supination	Lateral forearm (lateral antebrachial cutaneous)
	Coracobrachialis	Arm flexion, adduction	
	Brachialis	Forearm flexion	
Axillary	Deltoid	Arm abduction; internal, external rotation	Lateral aspect of shoulder
	Teres minor	Arm external rotation, adduction	
Radial	Triceps	Arm and forearm extension	Dorsoradial hand, thumb (superficial radial)
	Anconeus	Forearm extension	
	Brachioradialis	Forearm flexion	
	Extensor carpi radialis longus and brevis	Wrist extension	
	Extensor carpi ulnaris		
	Supinator	Forearm supination	
	Extensor digitorum communis	Finger, thumb extension	
	Extensor indicis proprius		
	Extensor digiti minimi		
	Extensor pollicis brevis and longus		
	Abductor pollicis longus		
Median	Flexor carpi radialis	Wrist flexion	Volar thumb, index, long, radial half of ring finger; dorsum index, long, radial half of ring finger
	Pronator teres and quadratus	Forearm pronation	
	Flexor digitorum sublimis	Finger proximal interphalangeal joint flexion	
	Flexor digitorum profundus (index, long)	Finger distal interphalangeal joint flexion	
	Abductor pollicis brevis	Thumb abduction	
	Opponens pollicis	Thumb opposition	
	Flexor pollicis brevis (superficial head)	Thumb metacarpophalangeal joint flexion	
	Lumbricals (index, long)	Metacarpophalangeal joint flexion	
		Interphalangeal joint extension	
Ulnar	Flexor carpi ulnaris	Wrist flexion	Entire little finger, ulnar half of ring finger, and ulnar border of hand
	Flexor digitorum profundus (ring, little)	Finger distal interphalangeal joint flexion	
	Abductor digiti minimi	Little finger abduction	
	Flexor digiti minimi	Little finger metacarpophalangeal joint flexion	
	Abductor pollicis	Thumb adduction	
	Flexor pollicis brevis (deep head)	Thumb metacarpophalangeal joint flexion	
	Interossei (volar, dorsal)	Metacarpophalangeal joint flexion, interphalangeal extension	
	Lumbricals (ring, little)	Metacarpophalangeal joint flexion, interphalangeal extension	

TABLE 43-7: Medical Research Council Strength Score

Score	Muscle strength
0	No movement
1	Flicker of movement
2	Movement with gravity eliminated
3	Movement against gravity intact
4	Movement against resistance
5	Full power

0, no contraction; 1, identifiable contraction without movement; 2, identifiable contraction with movement with gravity eliminated; 3, contraction with movement with gravity; 4, contraction with movement against resistance; 5, full power.

Treatment

The treatment for a stretch or concussive injury to the brachial plexus or a peripheral nerve is generally observation for a period of at least 3 to 6 months. Many of these patients will demonstrate a spontaneous recovery. Yet, this algorithm is at odds with the patience of many patients and providers and hopes for an immediate recovery or intervention. Nerve recovery, compared to that of vascular or skeletal treatment, is not immediate. It will take many months for a nerve deficit resulting from a stretch or contusion to recover.

In concussive, blast, avulsed, or crush type injuries, initial treatment is generally observation, therapy directed at maintaining joint suppleness, and splinting. A determination as to whether or not meaningful recovery will occur can be difficult. The presence of a Horner sign, the aforementioned nerve conduction/EMG findings, and lack of motor recovery by 3 to 6 months generally favors surgical management. In instances where pre- or postganglion avulsion can be identified with certainty, early surgical intervention is performed.

Currently, the available literature supports nerve transfers over grafting in the treatment of injuries of the brachial plexus. Injuries that involve the upper trunk are readily addressed with a series of nerve transfers that use expendable and reliably intact nerves. The spinal accessory transfer to the suprascapular nerve will permit reinnervation of the supra- and infraspinatus muscles. The branch of the radial nerve to the medial head of the triceps to axillary transfer will permit reinnervation of the deltoid muscle.[93] Finally, fascicles of the median and ulnar nerves transferred to the branches of the musculocutaneous nerve to the brachialis and biceps muscles will provide elbow flexion.[94,95]

Sharp penetrating injuries result in a partial or complete nerve transection. In these cases, early exploration is indicated. Repair of sharp transections of the brachial plexus, particularly nerves that control the proximal shoulder muscles and elbow flexors, will yield useful function.[96] Similarly, repair of a sharp injury to the radial or median nerve will allow the majority of patients to regain strength against resistance (MRC score of 4).[97]

Fortunately, the majority of gunshot wounds to the upper extremity resulting in a complete deficit of a discrete nerve will recover spontaneously.[98] In some patients, a nerve deficit is associated with either vascular or skeletal trauma, which then leads the surgeon to consider operative treatment. If the nerve is exposed for the treatment of the vascular or skeletal trauma, it is inspected carefully with loupe magnification. If there is obvious disruption of fascicles, repair or grafting is indicated. For injuries where the gross appearance is abnormal but the nerve remains in continuity, observation is indicated. A plan for subsequent reconstruction is made if recovery has not occurred within the expected time. For example, a radial nerve found to be contused during fixation of an associated fracture of the humeral shaft will typically recover within 8 months or less. If no recovery is demonstrated, the patient is advised that future nerve grafting or tendon transfers will be necessary.

MUSCLE AND TENDON INJURIES IN THE UPPER EXTREMITY

Physical Examination

Trauma to the upper extremity almost always includes some degree of injury to muscles or tendons. Contusions are common and will generally heal with a period of prescribed rest and therapy. Lacerations involving tendons of the upper extremity are treated operatively, as there is an excellent potential for recovery and healing.

Any wound deep to the fascia of the shoulder, arm, or forearm should lead to a meticulous physical examination and begin with a "top-down" approach.

Aside from ensuring joint suppleness, the examiner should assess the patient's ability to maintain external and internal rotation and forward elevation against resistance. A drop arm test, performed with passive elevation of the internally rotated humerus with the elbow extended, will localize control of the rotator cuff. With a massive cuff tear, the arm falls abruptly with the action of gravity.[99]

Injures to the antecubital fossa and associated elbow flexors are ominous because transection of the biceps and brachialis muscle will frequently involve injury to the brachial artery and median nerve. The associated findings of an anterior elbow wound and profuse bleeding should alert the clinician to a high probability of injury to each of these critical structures. In addition to the vascular examination, the examiner should pay specific attention to the posture of the hand. The "benediction sign," where the thumb and index finger assume an extended posture, is indicative of a median nerve deficit, where the flexor pollicis and index profundus and superficialis muscles are paralyzed, while the ulnar nerve maintains digital flexion through its innervation of the more ulnar profundi (Fig. 43-13). Injuries to the triceps muscles with loss of extension against gravity and/or resistance indicate need for repair. Injures involving the triceps muscle will frequently include some trauma to the ulnar nerve, so the examiner should assess the patient's sensation about the ulnar digits and the ability to abduct and adduct the fingers.

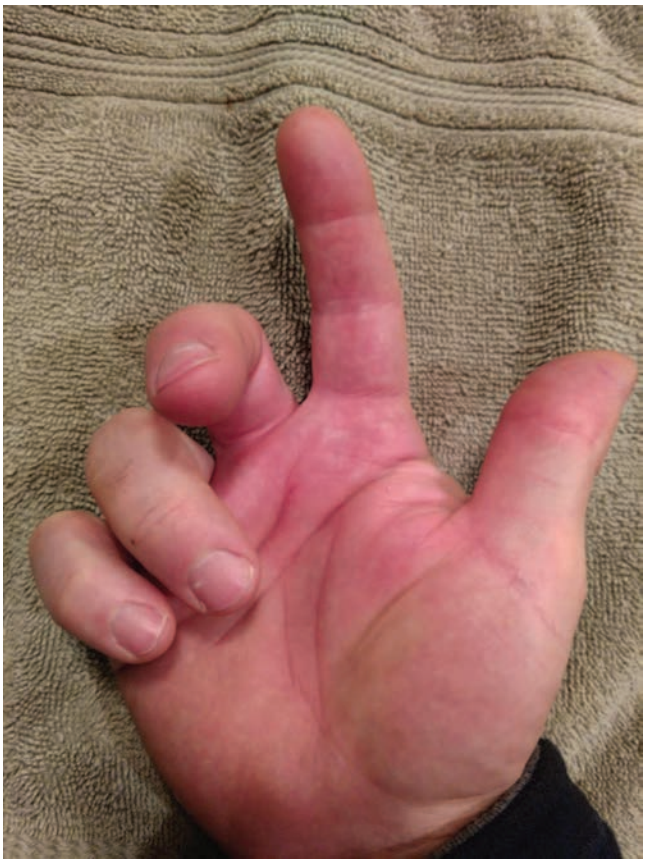

FIGURE 43-13 Benediction sign after compression or injury of the median nerve at the elbow or forearm.

Lacerations deep to the fascia of the forearm or hand will involve injury to a critical tendon or nerve in a large percentage of cases and, thus, should be explored. The extended posture of the digit with transected flexors or the wrist lying in flexion with transected extensors is easy to diagnose. In the awake patient, isolation of individual digital flexion and extension should be performed, "blocking" the other digits and isolating the proximal and distal interphalangeal joints individually to demonstrate the integrity of the flexor systems.

Diagnostic Modalities

The vast majority of tendon injuries in the upper extremity and hand are diagnosed with physical examination alone. In certain cases, such as injury to the rotator cuff, triceps, or biceps muscles, and/or in certain instances involving the digits, a tendon injury can be accurately assessed with MRI. Ultrasound can also aid in diagnosis but requires a well-trained ultrasonographer or institutional familiarity.

Treatment

Ruptures of the pectoralis major are infrequent. Commonly the result of an eccentric contraction, such as during a bench press or forced extension of the shoulder against resistance, these injuries present with a defect overlying the anterior shoulder that is both visible and palpable. Operative treatment is preferred in the young patient for restoration of cosmesis and strength.

Ruptures of the rotator cuff are common in trauma patients, particularly in those over 50 years of age sustaining a first-time shoulder dislocation.[100] Massive tears are frequent and can result in significant dysfunction of the shoulder. These tears require prompt recognition, as the tear will retract to an irreparable level within weeks. Arthroscopic or open repair is indicated for the majority of these injuries.

A proximal injury of the biceps tendon will present as a "Popeye" deformity and is most commonly the result of preexisting tendinopathy of the rotator cuff. Nonoperative management is usually prescribed.

Lacerations of the biceps and triceps tendons at the elbow similarly warrant expeditious repair, as excellent results can be expected. A delay in treatment will result in the need for tendon grafts to overcome the shortening inherent to delay. As noted earlier, these lacerations should be approached with the anticipation of an associated vascular or nerve injury.

FOREARM AND HAND

Lacerations involving tendons in the forearm and hand require an expeditious repair.[101,102] Complete transection results in immediate loss of length, due to unopposed contraction of the muscle. The contracted state will preclude repair in a period as short as 2 to 3 weeks. The flexors and extensors of the wrist and hand provide critical function, and appropriate repair should be by a trained hand surgeon[103] (Table 43-6). Flexor tendon injuries to the palmar surface of the thumb and fingers within "no-man's land" are notoriously difficult to treat, requiring specialized tendon handling, suture repair, and postoperative therapy to achieve a good result.

Tendon repairs of the upper extremity are generally completed using a braided nonabsorbable suture, appropriate to the size of the tendon undergoing repair. For example, tendons within the finger are typically repaired with a 3-0 or 4-0 suture, those in the forearm and wrist typically with a 2-0 or 0 suture, and those in the distal biceps with a #2 suture. Restricting active motion of the repaired tendon is generally prescribed as part of the rehabilitation protocol, whereas passive motion of the crossed joint is initiated immediately. Gradual progression to active motion and then strengthening is permitted over a period of 6 to 8 weeks.

SKIN INJURIES IN THE UPPER EXTREMITY

Physical Examination

Perhaps the most readily assessed component of the upper extremity is the skin. Even so, a careful and experienced eye is required to complete a thorough inspection. Good lighting and palpation are generally all that is required to yield a near-complete assessment. There are, however, many qualities of the skin that require objectification. The clinician, in formulating

a treatment plan, is well served by learning how to articulate these specific and detailed qualities. For the majority of non-burn or chemical injuries to the skin, trauma mechanisms can be divided into ischemic and direct tissue loss categories.[104]

ISCHEMIC CHANGE

Skin, like all other tissues, relies on a continuous source of perfusion and oxygenation. Although skin will tolerate ischemia significantly better than muscle tissue, it remains susceptible to necrosis with a prolonged ischemic time. The speed of progression to frank necrosis is dependent on the degree of hypoxia. Discerning the degree of hypoxia/ischemia can be challenging and is done based on the mechanism of injury, physical examination, and ancillary studies.

A variety of mechanisms are responsible for ischemia of the skin.[105] Direct trauma or thrombosis of the regional artery can result in obvious disruption of perfusion. Pressure on the skin, whether external (eg, ill-fitting splint or cast, bandage, traction device, restraint, or any form of external compression) or internal, will result in relative ischemia to the affected area and some degree of skin loss, depending on the duration and severity of the pressure.[106] Hypoxia can also result from edema or a hematoma. Edema or hemorrhage in the tissue immediately below or within the skin can result in focal pressure, decreased perfusion, and resultant local hypoxia. Finally, skin sustaining a shear and degloving injury will lose the arterial connections to the axial vessels below. If this area is significant, the skin will necrose.

Assessment of skin perfusion is accomplished with an assessment of capillary refill. Skin that is well perfused in the fair-skinned individual will be pink and blanch with modest pressure. Excellent lighting, preferably incandescent, is required to assess the color of the skin, as previously noted. Perfusion assessment of the nonglabrous skin in dark-skinned individuals can be particularly challenging, as the blanching associated with pressure can be difficult to demonstrate. In instances where severe venous occlusion or insufficiency has occurred, the part will become congested, manifesting as rapid capillary refill and a bluish color. In advanced stages, compression of the capillary beds becomes impossible, and no refill phenomenon is present. In late stages, this leads to capillary leaking and "bruising" of the skin, appearing as a port-wine discoloration.

Turgor, or the "fill" of the skin, is another important characteristic. Normal turgor is maintained in the uninjured extremity with appropriate degrees of perfusion and venous return. Comparison of the affected part or extremity to its uninjured partner will provide an accurate gold standard in regard to color and turgor. In instances of venous congestion, the turgor becomes abnormally increased to the point of gross swelling.

Skin that has sustained trauma will frequently blister. These blisters should alert the clinician that the skin has, to some degree, lost perfusion. Blistering around fractures of the knee, ankle, foot, and elbow is common.[107] This is believed to be the result of hypoxia induced by the abnormally elevated subcutaneous pressure from edema or hemorrhage, particularly in those areas where the skin is directly overlying bone, such as the medial tibial plateau or calcaneus.

Skin that has sustained substantial degloving will frequently demonstrate a "tomato skin" color and friability. Predicting whether or not this skin will recover to provide a sufficient barrier for the desired functional recovery can be challenging at best. If the patient is warm and normotensive, with no correctable trauma to an axial vessel, incised skin should bleed. If the skin does not bleed, the surgeon can be assured that the skin will eventually demarcate and turn to eschar. Delaying excision will typically complicate the patient's course. In cases where there is no underlying fracture, hardware, or vascular injury, allowing the skin to "declare" can be preferable. Excising large portions of skin should be done with caution and with a subsequent reconstructive plan in consultation with a soft tissue/microsurgeon.

Skin that has "declared" will be black. These changes typically take several days or, in some cases, weeks. In these instances, particularly in areas where skin coverage is critical (eg, open fracture, hardware, or critical vascular repair), the necrosis is typically debrided and replaced with adjacent, distant, or a free tissue transfer.

Diagnostic Modalities

As described earlier, discerning the degree of perfusion can be challenging. Several adjunctive methods can assist the provider in determining relative degrees of ischemia.

LASER-ASSISTED ANGIOGRAPHY

Indocyanine green dye will fluoresce when exposed to light in the near-infrared spectrum and has been used to assess soft tissue perfusion in cardiac, ophthalmologic, bowel, plastic, biliary, and orthopedic surgery.[108,109] Commercially available cameras and devices using this technology provide an objective assessment of the perfusion of skin with results provided in both relative and absolute values.[110] Through its assessment of perfusion of the skin and flaps, it has been effective in decreasing the risk of soft tissue complications in breast and soft tissue reconstructions.[111] Also, it can be used to quantify differences in skin perfusion with different closure techniques and assess viability in free tissue transfers and frostbitten extremities, and is undergoing significant investigation in predicting soft tissue complications in at-risk fractures.

Thermography using forward-looking infrared cameras has become increasingly accurate, available, and portable.[112] Temperature differences on the order of 0.1°C are easily and reproducibly measured with smart phone adapters. This technology has been used to assess the perfusion of free tissue transfers,[9] burn wounds, and thermal changes in skin during fracture healing.[113,114]

Treatment

Potentially compromised skin should be first addressed by ensuring adequate warmth, stability, and perfusion. This is accomplished by maintaining an appropriate core temperature of the patient and applying a warming blanket to the extremity. Reducing dislocated joints and aligning fractures via either operative or nonoperative measures are imperative

to reduce the risk of vascular torsion, kinking, or spasm. Also, this further limits the catecholamine response from the painful stimulus of an unstable limb. Finally, ensuring adequate volume resuscitation is imperative to maintain adequate perfusion of the extremities.

SKIN GRAFTING

In cases with preservation of the underlying muscle, periosteum, peritenon, or subdermal tissue, skin grafting is appropriate.[115] In those cases with exposed muscle alone (eg, fasciotomy wounds), split-thickness grafting will suffice. Because the degree of contracture is inversely proportional to the thickness of the dermis within the skin graft, graft thickness is an important consideration over areas such as the axilla, neck, palmar hand, and antecubital fossa. These areas are prone to intolerable contracture, and full-thickness skin grafts are indicated.

ADJUNCTIVE AND TEMPORIZING TREATMENTS

In patients with massive skin loss or areas that are deemed not "ready" for skin grafting, dermal matrix substitutes have gained in popularity.[116] These commercially available products are applied to the wound bed to allow for vascular ingrowth over a period of several weeks. The skin graft is then applied to the integrated collagen matrix. These products have led to favorable results over exposed tendon, joint capsule, and large areas of atrophic wound beds. This technique still requires a nutritionally restored patient and adequate perfusion to the extremity. In the author's experience, the products do not provide adequate supple coverage for areas that require tendon excursion for function or elevation for subsequent reconstructions, and are used sparingly.

Allograft skin provides an additional option for temporary wound coverage.[117] When the patient is malnourished or unstable and/or a large wound bed of indeterminate viability and quality is present, allograft skin can be used as a trial run. This skin grafting technique depends on the typical imbibition and inosculation common to all skin grafts, with the added advantage of almost no biologic or surgical morbidity to the patient. It provides the benefit of decreasing insensate losses and dressing changes while providing a biologic barrier. Upon operative removal in 7 to 10 days, the underlying tissue has been "primed" with the ingrowth of a superficial capillary network favoring autograft skin. In these wounds, the quality of the allograft skin "take" will provide the clinician and patient confidence that the wound bed is amenable to precious autograft skin, particularly in patients with large skin defects and severe trauma, where donor skin is in limited supply.

FLAPS

In cases where the underling tissues will not support a skin graft, the transfer of a healthy tissue "flap" is indicated.[118] In the extremities, these are most commonly performed over open fractures, exposed orthopedic implants, vascular repair or reconstructions, tendon, joints, or joint creases such as the antecubital fossa.[119]

Flaps can be divided into local, distant, and free. Local skin flaps of the upper extremity typically include transfer of full-thickness skin using either advancement, transposition, or a pedicled or a propeller-type transfer. Other local flaps include muscle, such as the pedicled flexor carpi ulnaris flap.[120]

Distant flaps, such as the groin flap, rely on vascular ingrowth into the transferred flap from the recipient bed[121] (Fig. 43-14). This flap requires a period of 3 weeks of relative immobility of the hand while attached to the groin region but provides the benefit of not requiring a donor vessel. This is relevant in patients with a severely traumatized limb. In cases where axial vessels will be subsequently used for additional free tissue transfer, the groin flap can provide a suitable source of supple and expendable skin. After an appropriate period, the flap is then divided and inset.

In patients in whom local or distant flaps are deemed unsuitable, a large array of microvascular free tissue options exist, provided there are an adequate recipient artery and vein. Flaps composed of skin, muscle, bone, tendon, nerve, or artery and vein are available and can be tailored to the requirement of the defect. Most commonly, skin is required, and flaps such as the anterolateral thigh or parascapular flap are suitable and provide large volumes of supple skin with minimal donor site morbidity (Fig. 43-15). Smaller defects are amenable to flaps such as the lateral arm, and in cases of

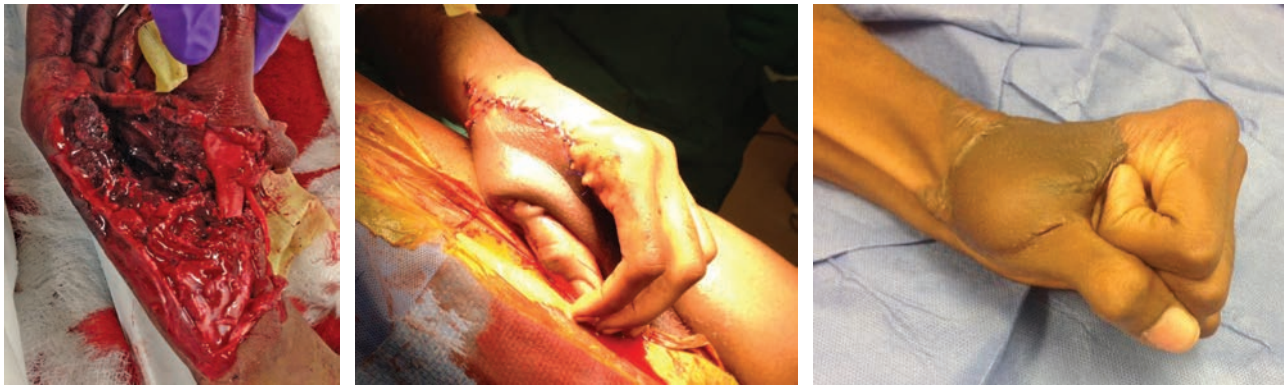

FIGURE 43-14 Groin flap for coverage of a mangled hand and replanted thumb.

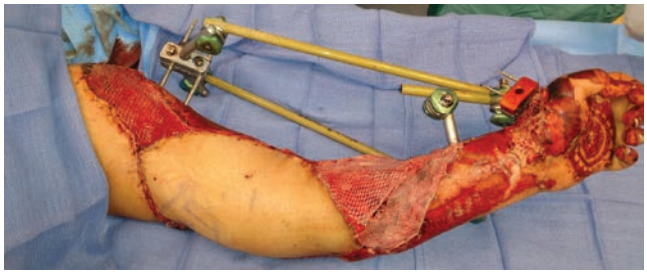

FIGURE 43-15 Anterolateral thigh flap for coverage of a large antecubital wound.

missing diaphyseal bone of the upper extremity, osteocutaneous flaps such as the free fibula are chosen.[122]

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44

Lower Extremity

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KEY POINTS

- Compartment syndrome, mangled extremities, high-grade open fractures, irreducible dislocations, and fractures with vascular compromise require emergent treatment.
- Low-grade open fractures, femur fractures, and hip fractures are ideally treated within 24 hours.
- Evidence shows decreased infection rate for open fractures when antibiotics are administered within 1 hour of admission.
- Extreme diligence is required in the care of knee dislocations due to a 7% to 15% incidence of injury to the popliteal artery.
- Unnecessary testing delays surgery, increases costs, and does not provide clinical benefit in geriatric patients with hip fractures.
- Geriatric hip fractures are often life-changing events, with only 60% of patients returning to preinjury activity level and a 30% 1-year mortality.
- External fixation is an excellent option for rapid initial stabilization of many fractures in the lower extremities.
- Non–life-threatening injuries such as pilon, calcaneus, and Lisfranc fractures can have a dramatic long-term negative impact on a patient's functional status.
- Advances in prosthetics currently make below-knee amputation a better functional option than limb salvage in many patients.

INTRODUCTION

Lower extremity injuries are a common cause of hospital emergency department visits. These injuries and their effect on patient function have a great impact on society.[1] Lower extremity fractures can occur as simple injuries or as complex, high-energy injuries in a polytraumatized patient. Patients with significant injury to their lower limbs are at risk for a wide variety of complications including ischemia, infection, nonunion, chronic pain, and long-term functional deficits. Complex extremity trauma requires prompt assessment of the injury including bone, neurologic, vascular, integument, and muscular evaluations. Technical advances in both osseous and soft tissue reconstruction have led to improvements in treatment, mobilization, and postinjury rehabilitation of severely injured patients. Rehabilitation concepts have changed from prolonged rest to the present emphasis on rapid restoration of skeletal stability allowing prompt mobilization. Appropriately timed management and well-executed surgical interventions are critical to prevent infection, achieve wound healing, promote osseous union, avoid systemic complications, and maximize functional outcome. Many victims of extremity trauma also have associated life-threatening injuries to the head and torso. Therefore, the initial evaluation of lower extremity fractures must focus on the patient as a whole and not exclusively on the injured limb.[2-4]

The wide prevalence of safety belt usage and changes in vehicular design, such as crumple zones and mandatory air bags, has improved the survival rates in high-energy crashes. This has led to an increasing number of severe lower extremity injuries previously "unseen" because the patient did not survive. Additionally, with an aging population, there has been an increase in the rate of fractures in elderly patients, often accompanied by multiple medical comorbidities that can compromise their response to traumatic insults.[5] Many of these patients are on anticoagulants or other medications that can dramatically amplify the effects of even low-energy trauma. These patients require a high level of diligence, from both the trauma and orthopedic surgeons, because of the potential for a larger systemic impact due to medical comorbidities and polypharmacy.

There is considerable controversy surrounding the timing of many orthopedic interventions, which has led to variations in recommended practice over the past 20 years.

Current evidence supports "urgent" management of some injuries historically treated "emergently," including talus fractures, femoral neck fractures, and most open fractures, with a higher emphasis on the quality of the surgical intervention rather than on the timing.[6,7] In contrast, compartment syndrome, fracture-dislocations associated with neurovascular compromise, and severe soft issue injuries including mangled extremities remain true orthopedic emergencies.[7] Failure to recognize the significance of these injuries can lead to severe sequelae such as limb loss or death. Open fractures of the lower extremities require timely surgical treatment by an appropriately skilled team to minimize the risk of infection and limb loss. A quality debridement, including (1) removal of contamination; (2) excision of nonviable tissue; and (3) excision of extra-articular bone fragments lacking soft tissue attachments, is regarded by most orthopedic traumatologists as more important than debridement occurring on an emergent timeline. However, data and widespread clinical practice support the practice of irrigation and debridement of open fractures within 24 hours of injury, with crush injuries and badly contaminated fractures likely benefiting from a more emergent treatment.[6]

The concept of "damage control orthopedics" was established based on the principle that prolonged early definitive treatment of long bone fractures can be detrimental for severely injured patients who are physiologically unstable.[8,9] In these patients, the early mitigation of the "lethal triad" of persistent metabolic acidosis, hypothermia, and coagulopathy represents the prime goal for survival.[2] Resuscitation strategies have improved over the past decade, however, and multiple recent studies have shown improved outcomes using an early appropriate care model, in which early care is titrated based on a patient's overall injury constellation and physiologic status.[10-13] The initiation of surgical care when considered safe has not only improved clinical outcomes, but has also been shown to have a dramatic effect on the overall cost of care.[14]

BIOMECHANICS AND MECHANISM OF INJURY IN LOWER EXTREMITY TRAUMA

Biomechanics is a term that relates physics to the musculoskeletal system and can add to the understanding of how an injury occurred. Fractures occur when the applied load to the bone exceeds its load-bearing capacity. Bone is a complex living tissue consisting of collagen fibers embedded with mineral in an intricate fiber orientation and a varied density of the Haversian system (microscopic functional unit of bone). The ratio of collagen to mineral has a significant effect on the elastic modulus (resistance to deformation) of bone, which decreases with loss of mineral content.[15] The orientation of collagen fibers influences the ability of bone to withstand load in specific directions. The fiber orientation of fracture callus in the initial healing phase is random and disorganized. Fibers progressively reorganize along the direction of load to which the bone is exposed. The density of the Haversian system directly affects bone strength. As bone mineralizes and matures from childhood to adulthood, the bending strength and modulus of elasticity increase. Bone density begins to decrease in the fourth decade of life, which can progress in accelerated fashion in patients with osteoporosis, rendering the bone more fragile and susceptible to fracture with lower energy mechanisms.

There are four basic forces that lead to fracture: compression, tension, torsion, and bending. These forces, when applied purely, cause the bone to fracture in predictable ways. A compressive force results in shortening the length of bone, causing oblique fracture lines or comminution (multifragmentary fractures). Tensile force elongates bone, causing transverse fractures. Bone is weakest in torsion, and spiral fractures can result from relatively low torsional forces, leading to long oblique fractures often with little associated soft tissue injury. When a bending force is applied to a bone, there is a compressive force on the side where the force is applied and a reciprocal tension on the opposite side. The bone initially fails in tension, and as the fracture propagates toward the side of the applied load in which the bone is compressed, the fracture becomes oblique both proximal and distal to the force, creating a wedge-shaped "butterfly" fragment.

The amount of energy that produced a given fracture is suggested by the patient's history and the fracture pattern. Basic physics dictate that kinetic energy = 1/2 (mass)(velocity)^2 (see Chapter 1). Thus, as the velocity or mass increases, an exponentially higher amount of energy is absorbed by the musculoskeletal system. This energy is manifested as comminution and local damage to soft tissues. Obtaining a thorough patient history provides useful information that may alert the physician to a high level of injury severity. Although an accurate history may be difficult or impossible to obtain initially in a seriously injured patient, more detail should always be sought and reconfirmed as the patient improves or more information becomes available. A history inconsistent with the extent of injury suggests either a pathologic fracture or possibility of abuse.

HEALING OF FRACTURES

Fracture healing is a form of tissue regeneration that mimics bone formation from fetal development. It follows specific regenerative patterns and involves changes in the expression of many genes.[16] In general, bone heals by two pathways: direct (intramembranous ossification) and indirect (endochondral ossification). Direct bone healing, also known as primary bone healing, occurs in a low-strain (no motion between fracture fragments) environment and requires anatomic reduction of fracture ends without gap formation and stable fixation. Therefore, it does not commonly occur as a natural process but is typically achieved only by surgical means with open reduction and internal fixation. On the contrary, indirect fracture healing, also known as secondary bone healing, occurs in a moderate-strain environment (moderate but not excessive fragment motion) and does not

require anatomic reduction or rigid stable fixation. Nonoperative and certain surgical treatments that provide relative stability such as intramedullary nailing, external fixation, and bridge plate fixation for comminuted fractures ideally lead to indirect fracture healing.

Indirect fracture healing, in contrast to the regeneration of lamellar bone and the Haversian system in direct fracture healing, follows a specific biological pathway. This pathway involves an acute inflammatory response (interleukin [IL]-1, IL-6, IL-11, IL-18, and tumor necrosis factor-α) followed by mesenchymal stem cell–induced cartilaginous callus formation, which undergoes revascularization and calcification until complete bony remodeling is achieved.[17] Despite the regenerative capacity of bone, the process can fail and lead to nonunion.

CLINICAL EVALUATION OF PATIENTS WITH LOWER EXTREMITY TRAUMA

Patient evaluation according to the Advanced Trauma Life Support (ATLS) protocol provides a systematic method of thoroughly examining the trauma patient, assuring appropriate sequence of care, and minimizing missed injuries.[2,18] In addition, the importance of continuous detailed documentation of the physical findings cannot be overemphasized. Patients with extremity injuries require thorough and serial examination. All assessments should be well documented to facilitate comparison by all members of the care team. Although care of multiply injured trauma patients with extremity injury is primarily driven by the general surgery trauma team, multidisciplinary input and efficient communication among consulting services including orthopedic surgery, neurosurgery, vascular surgery, emergency medicine, anesthesia, and radiology are critical to rapidly institute an appropriate treatment strategy.

The secondary survey is a head, torso, and extremity examination after the life-threatening injuries have been identified and initial stabilization measures have been employed. Examination of extremities starts with an inspection. Contusions, open wounds, and deformities are noted. Patients should be carefully rolled to inspect the posterior portion of the patient for wounds. Palpation of extremities for tenderness and abnormal movement aids in identification of injuries. When possible, asking the patient to move each extremity can be a quick screen for injuries. In high-energy mechanisms, the palpation portion of the exam should include the principle of "touch every bone" to elicit areas of abnormal motion or tenderness, which can go unnoticed in a multiply injured patient. Markers of injury can be easily masked in intubated and sedated/obtunded patients. This vulnerable population cannot complain of pain, neurologic examination is unobtainable, severe life-threatening injuries may take precedence, and assessment of deformity or swelling can be difficult, especially in obese patients. The exam should be revisited when an initially obtunded patient is first extubated. A "tertiary survey" should be completed after the initial resuscitation and operative interventions are complete or when a patient's neurologic function improves. Detailed knowledge of lower extremity anatomy and understanding the function of structures and location in relation to injuries are key elements of the physical exam. The extremity exam should proceed in a methodical order to assess the following components of injury: skin, muscle, tendon/ligament, nerve, vessels, and bone.

Nerve Injuries and Evaluation

The neurologic status of the extremity should be documented as early as possible. Nerve injury is usually in proximity to the extremity injury; however, the possibility of an axial (head, spine) source of injury exists in trauma patients. Starting proximally in the lower extremity, the sciatic nerve may be injured in hip dislocations, acetabulum fractures, and thigh injuries, leading to lack of motor function and sensation below the knee. Approximately 10% of adults and 5% of children have neurologic injuries after traumatic hip dislocation.[19] More commonly, the peroneal branch of the sciatic nerve is injured with sparing of the tibial branch in hip and knee fractures and dislocations. The peroneal nerve is smaller with less neuron redundancy, and it is tethered as it passes around the fibular neck, making it more prone to traction injury. Up to 25% of knee dislocations (with or without fracture) lead to common peroneal nerve palsy.[20] Injury to peroneal division can cause decreased sensation in the lateral leg and lateral, plantar, and dorsal foot and weakness or palsy of foot and toe extension (foot drop). The sensory deficit is most easily identified by numbness in the first dorsal web space and dorsum of the foot. Injury to the femoral nerve results in diminished sensation on the anterior thigh and weakness of hip flexion (psoas muscle) and knee extension (quadriceps muscle). Tibial nerve injury, which is often involved in a mangled extremity or as part of a complete sciatic nerve injury, results in loss of sensation to the heel and plantar foot, accompanied by the inability to plantar flex the foot and toes. Although less common than peroneal nerve injury, tibial nerve injury is much more functionally debilitating because the loss of plantar foot sensation and decreased push-off strength dramatically affect normal gait. Simple screening active motion tests for lower extremity nerve assessment include raising the knee and foot off the bed (hip flexion and knee extension—femoral nerve) and caudal (tibial nerve) and cephalad (peroneal nerve) movement of the foot and toes (plantarflexion and dorsiflexion, respectively). Unfortunately, active motion can be limited by pain, in which case an exam based on passive motion and palpation becomes increasingly important.

Most peripheral nerve injuries of the leg are neurapraxias (temporary damage to the myelin sheath) that result from stretching mechanisms or blast effect secondary to projectiles. The vast majority of neurapraxias resolve without intervention by 6 to 8 weeks. Peripheral nerve recovery is assessed by outpatient serial examination. If there is no recovery, nerve conduction studies and electromyography within 3 to 6 months after trauma can aid in defining the extent and prognosis of injury. Axonotmesis occurs after a crush or stretch injury to

a peripheral nerve and implies axonal damage with maintenance of an intact endoneurium and perineurium. Wallerian degeneration occurs, and axonal regeneration occurs at a pace of approximately 1 mm/d. Neurotmesis refers to complete disruption of the axons and the overlying nerve sheath. Patients with acute neurologic deficits following lacerating trauma may benefit from acute surgical exploration and consideration of acute nerve repair. However, even with modern surgical techniques, prognosis for functional nerve recovery following complete nerve disruption is often poor in the lower extremity. Patient and injury factors such as age, medical comorbidities, nerve injury location, and smoking all play a role in the surgical decision making and recovery of nerve injuries.

Vascular Injuries and Evaluation

A detailed vascular assessment of the injured extremity begins with a pulse examination of femoral, popliteal, dorsalis pedis, and posterior tibial arteries. Capillary filling is not, by itself, adequate clinical evidence of intact vascular flow. Distal pulses may be present after a significant arterial injury. It is crucial to take a detailed history regarding time of injury because delays of postinjury revascularization in excess of 8 hours carry a risk of limb loss as high as 86%, compared to 11% if treated within 8 hours.[21] A high level of suspicion for associated vascular injuries is important in lower extremity trauma. The pulse assessment should be repeated after resuscitation and reduction of deformities, because most patients with abnormal pulses on initial examination demonstrate normalization of the flow following resuscitation, limb reduction, and splinting. Therefore, invasive arterial evaluation should be deferred until limb reduction and splinting are performed unless there will be a significant delay. Even in the absence of an abnormal pulse exam, leaving an obviously deformed lower extremity in its deformed position can have an adverse effect on perfusion, skin tension, and nerve traction. Simple, general realignment of an obviously deformed extremity should be performed early and can be safely done without concern of causing additional injury. Any pulse deficit or measurable reduction in arterial pressure index (API) or ankle-brachial index (ABI) after extremity realignment must be considered a potential vascular injury. The ABI is measured using systolic blood pressure in the extremity distal to the zone of injury divided by the systolic blood pressure in the brachial artery of an uninjured extremity. The API is measured by dividing the Doppler arterial pressure distal to the zone injury by the Doppler arterial pressure in an uninvolved upper extremity. Patients with symmetric peripheral pulses or an API or ABI less than 0.9 should undergo further assessment including imaging studies (eg, computed tomography [CT] angiography, duplex ultrasonography) and prompt evaluation by a surgeon experienced in the repair of vascular injuries.[21] The accuracy of pulse examination alone for the detection of arterial injury is very low. The five clinical hard signs of an arterial injury (Table 44-1) are present in more than two-thirds of all dislocations with an associated vascular injury.[22,23] Injuries with a high incidence of associated vascular injuries such as knee dislocations, displaced distal femur and proximal tibia fractures, open tibial shaft and ankle fractures, and mangled extremities should prompt a high level of diligence in the vascular evaluation.

TABLE 44-1: Signs of Vascular Injury

Hard signs	Soft signs
Active or pulsatile hemorrhage	Asymmetric extremity blood pressure
Pulsatile or expanding hematoma	Stable and nonpulsatile hematoma
Clinical signs of limb ischemia	Proximity of wound to major vessel
Diminished or absent pulses	Peripheral neurologic deficit
Bruit or thrill, suggesting arteriovenous fistula	Presence of shock/hypotension

A common arterial injury in the lower extremity involves the popliteal artery in association with a knee dislocation or periarticular fracture (Fig. 44-1). Knee dislocations will often spontaneously reduce, appearing relatively benign, and thus injury severity may be underappreciated. The popliteal artery is tethered at the adductor hiatus to the distal femur and at the soleus muscle on the proximal tibia, placing it at risk for vascular injury. In particular, fractures of the medial tibia plateau are often underestimated and should be considered akin to a knee dislocation.[24] Late thrombosis of an initially nonocclusive injury may result in limb loss. Frequent assessment of pedal pulses is required for these patients. Posterior knee dislocation often presents with transection of the popliteal artery, whereas anterior knee dislocation generally causes an intimal tear due to traction.[25] Anterior knee dislocation caused by a hyperextension injury is the most common type, accounting for approximately 40% of all knee dislocations, whereas posterior knee dislocation caused by a direct force such as a dashboard injury is the second most common type, accounting for approximately 30%.[26] Risk factors for limb loss include delayed surgery, arterial contusion with consecutive thrombosis, and, most importantly, failed revascularization. In penetrating extremity trauma, the presence of a hard sign of arterial injury was nearly 100% predictive of a significant vascular injury necessitating surgical intervention.[27] The majority (>95%) of arterial injuries occur in proximity to the site of fracture or joint dislocation. Delay in diagnosis secondary to missing a hard sign can result in limb loss.

The use of soft signs (Table 44-1) to detect occult vascular injury is less clear. The incidence of arterial injuries in patients with soft signs ranges from 3% to 25% depending on which soft sign or combination is present.[27,28] The yield of arteriography in the setting of clinical soft signs alone is very low, and the lesions that are typically identified are nonocclusive: intimal flap, contusion, spasm, and pseudoaneurysm. A high level of suspicion is still required, however, for identifying and treating potentially catastrophic vascular injuries in high-risk injuries such as knee dislocations. Persistence of soft signs on

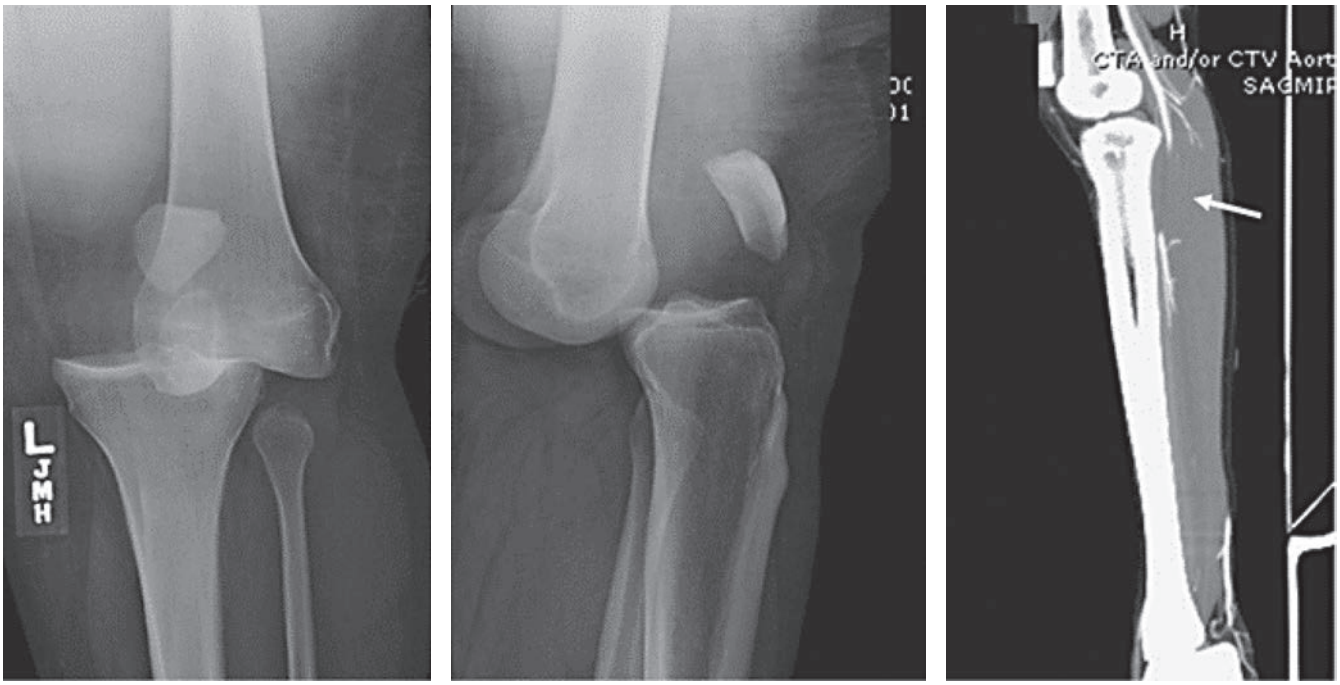


FIGURE 44-1 (**A**) Anteroposterior knee radiograph and (**B**) lateral knee radiograph demonstrating an anteromedial knee dislocation. (**C**) Sagittal computed tomography angiogram image depicting disruption of the popliteal artery just distal to the knee joint (arrow).

serial examinations is an appropriate indication for obtaining a CT angiogram to rule out underlying vascular injuries.

An arterial injury associated with a fracture or dislocation requires a coordinated approach by the trauma surgery and orthopedic teams. A temporary arterial shunt can be placed to restore limb perfusion and minimize tissue ischemia. The limb is reduced out to length and stabilized, often using external fixation. Subsequent to this, arterial repair or bypass is performed. Many institutional protocols leave the shunt after provisional limb stabilization and defer formal revascularization for 12 to 24 hours to allow patient resuscitation and optimization. There is controversy as to whether temporary fracture stabilization should precede vascular repair or vice versa. Clear communication of the treatment plan and sequence among treating surgeons is critical to optimize the care of these limb-threatening injuries. Care must be taken to avoid repairing the artery in a shortened position if vascular repair takes precedence as the repair can be damaged when the limb is lengthened and stabilized. In some fractures, formal osseous repair can be completed in less than 60 minutes, allowing vascular reconstruction to be performed on a stabilized limb that will not require repeat manipulation.

Evaluation of Soft Tissue Envelope

Soft tissue injuries (skin, muscle, nerve, vessels) are key drivers of functional outcome and decisions regarding limb salvage. Reconstruction of large osseous defects is possible through modern techniques including nonvascularized (iliac crest) and vascularized (free fibula) bone grafting, limb lengthening, and bone transport. Advancements in soft tissue reconstruction, most notably muscle and nerve regeneration, trail progress made in the treatment of bone defects. Energy associated with fractures is absorbed into surrounding soft tissues and causes varying degrees of injury. Soft tissue classification systems used to describe such injuries are relatively subjective based on observation, mechanism of injury, and severity of the fracture.[29] Significant swelling and blistering are signs of substantial soft tissue injury. Any wound in proximity to a known fracture is presumed to communicate with the fracture (open fracture) until proven otherwise. Antibiotic treatment in open fractures is critical, as early antibiotic administration has been associated with decreasing incidence of infection.[30] Immediate removal of gross contamination is warranted followed by the application of a sterile dressing. The tenets of wound management are debridement and irrigation, appropriate prophylactic antibiotic coverage, and definitive timely wound reconstruction. Temporary local antibiotic delivery using antibiotic-impregnated cement beads or spacers is occasionally employed in the treatment of open fractures for dead space management. Temporary wound coverage may be facilitated with the use of negative-pressure dressings. Wet-to-dry dressings are rarely indicated in the contemporary care of open fractures. Selection of appropriate soft tissue coverage is based on careful evaluation of the location and extent of injury, associated injuries, and medical comorbidities. Definitive wound coverage options include acute or delayed primary closure, skin grafting, local rotational flap, and free tissue transfer. Definitive wound coverage should be achieved as soon as possible but only after all contamination has been

excised and remaining tissue is perfused and viable. The timing of definitive soft tissue coverage is directly related to the severity of the initial wound. Recent evidence has demonstrated higher rates of deep infection with definitive soft tissue coverage after 7 days.[31]

Compartment Syndrome

While most limb injuries are appropriately prioritized after the care of axial trauma, compartment syndrome is a surgical emergency. Extremity compartment syndrome occurs due to raised pressure within a confined space (leg compartment) that causes permanent and irreversible damage to the contents of the space, including nerve and muscle. It is precipitated by an event that elevates compartmental pressures leading to decreased microcirculatory blood flow, resulting in a positive feedback loop and ultimately muscle and nerve necrosis. This damage may be reversible within the first 4 to 6 hours if an adequate fasciotomy is performed to relieve the pressure. However, there is an increased risk of soft tissue necrosis as time progress, and authors have reported the damage to be irreversible after 8 hours.[32]

Fracture is the most common cause of compartment syndrome, accounting for 69% of cases in one study.[33] Tibial fracture is the most common etiology with a prevalence of compartment syndrome reported up to 15%.[33,34] High-energy injury is typically associated with acute compartment syndrome. High-energy or crush injuries, segmental or widely displaced fractures, severely comminuted fractures, and patients with impaired sensorium are at high risk. Paradoxically, the prevalence of acute compartment syndrome in lower extremity fractures can be significant even in low-energy injuries.[33] Additional common etiologies of compartment syndrome include crush injuries, forearm fractures, vascular injury (reperfusion), intravenous line infiltration, excessively tight circumferential dressings or casts, dog bites, high-pressure injection injuries, burns, prolonged immobilization (found down after drug overdose), and intraoperative lithotomy positioning. Although ballistic injuries carry an overall low incidence of compartment syndrome (3%), isolated proximal tibia and fibula fractures due to gunshots have a 21% chance of developing compartment syndrome.[35]

The key to successful treatment of compartment syndrome is making diagnosis in a timely manner. Compartment syndrome should be suspected when pain is disproportionate to the injury and worsens over time across multiple exams. Other signs of significant soft tissue injury, including fracture blisters, diffuse ecchymosis, swelling, and tissue crepitus, should alert the medical team to a high-risk injury. Pain on passive stretch of the muscles in the affected compartment and paresthesia are the most important physical exam findings suggestive of early compartment syndrome when associated with swelling and firm soft tissues on palpation. Motor deficits and decreased pulses are late findings; however, poor diagnostic sensitivity and specificity of clinical symptoms and signs have been reported.[36,37] Compartment syndrome is widely accepted by most surgeons to be a clinical diagnosis. However, intracompartment pressure measurement can be obtained to assist in the diagnosis of acute compartment syndrome in patients with equivocal exam findings or obtunded/sedated patients. In a recent study of tibia fractures, compartment pressure measurement was found to have a high false-positive rate of 35% using accepted criteria for diagnosis (compartment pressure within 30 mm Hg of systolic blood pressure).[38] Continuous compartment pressure monitoring has reported higher diagnostic accuracy (98% sensitivity, 98% specificity, 93% positive predictive value, and 99% negative predictive value), but these results are interpreted with extreme caution because the diagnosis of "disease" in this study was "escape of muscle" at the time of surgery, which is regarded by most clinicians as highly subjective.[39]

Compartment pressure measurement, when used, should be performed in all compartments and within 5 cm of the fracture.[40] An arterial line setup, slit catheter, or side-port needle can be used, but one must be cognizant of the fact that an arterial line may read 5 to 15 mm Hg higher than a side-port needle. The two most commonly cited thresholds for diagnosis are pressure within 30 mm Hg of the diastolic blood pressure (delta P) and absolute compartment pressure greater than 30 mm Hg.[37,39,40] Clinical exam can be unreliable in an obtunded or anesthetized patient; therefore, compartment pressure measurement has an increased role in this population.[39] Institution-wide protocols (high-risk compartment syndrome protocols) that empower all team members (eg, nurses, residents, faculty) and promote rapid escalation when compartment syndrome is suspected are generally considered best practice.[41] Documentation is important both to compare physical examination findings as well as for medicolegal reasons because prolonged time from onset of symptoms to fasciotomy is associated with an increased medicolegal risk.[42]

Although various fasciotomy techniques are used for the lower extremity, the vital aspect of surgical treatment is complete release of the affected fascial compartments. The thigh is typically treated with a single lateral incision to facilitate release of both the anterior and posterior compartments. Rarely, the medial (adductor) compartment also requires release through a separate incision. The lower leg is typically treated with a dual incision technique, although single-incision techniques have been described.[43] Foot fasciotomies are often performed with two dorsal incisions to access the nine compartments. If feasible, communication between surgical services is helpful to avoid suboptimal placement of fasciotomy incisions that may compromise future fracture procedures and wound care. Tibia fractures with compartment syndrome are more likely to become infected and develop nonunion.[44] Cost and length of the hospital stay are more than double for these patients.[45]

GENERAL MANAGEMENT OF OPEN FRACTURES

An open fracture is defined as a fracture that communicates with the external environment through a traumatic soft tissue defect. It should be emphasized that the soft tissue injury may

not lie directly over the fracture. A fracture in the same region as soft tissue injury should be considered open until proven otherwise. History regarding the environment and extent of contamination, as well as the time elapsed from injury, are vital to appropriate management and determining appropriate antibiotic coverage. Open fractures should be quickly assessed on arrival to the emergency department. The patient should be adequately undressed so that all soft tissue injuries can be identified. This is especially important in patients who are unconscious or under the influence of alcohol or drugs. Size, location, orientation (transverse, longitudinal, irregular), and depth of the wound are documented, along with notation of any exposed bone, tendon, or muscle. The Gustilo-Anderson classification of open fractures is commonly used to convey information and dictate treatment (Table 44-2). It is important to recognize that significant internal soft tissue injury such as extensive periosteal stripping and fracture comminution can be present with even small wounds, suggesting a higher level of energy.

Ideally, photographic documentation of the wound is obtained prior to application of a sterile dressing and splint. This is important not only because visual documentation surpasses written descriptive documentation for planning treatment, but it also prevents exposing the wound to further contamination by removing the dressing multiple times for an evaluation by other team members. A detailed medical and social history is important because many chronic conditions and medications may influence the treatment plan and outcome, including diabetes mellitus, rheumatoid arthritis, immunocompromised state, renal failure, dementia, smoking, and substance abuse.

Early antibiotic administration in patients with open fractures has been shown to decrease the risk of infection.[46,47] There is little quality evidence describing the optimal antibiotic coverage and duration for open fractures. The duration and choice of prophylactic antibiotic therapy have been reviewed by the Orthopaedic Trauma Association, Surgical Infection Society, and the Eastern Association for the Surgery of Trauma, but all recommendations are based on low-level evidence. Prolonged antibiotic therapy may increase the risk of developing antibiotic-resistant systemic and nosocomial infections, including pneumonia and urinary tract infection.[48] Antibiotic treatment is dependent on the severity and contamination of the wound (Table 44-3).[49,50] Most authors advocate directing antibiotic coverage toward gram-positive organisms (first-generation cephalosporins) and adding gram-negative coverage (aminoglycosides) for contaminated fractures including severe type III injuries.[30] Aminoglycosides have an increased risk of nephrotoxicity in trauma patients. Fluoroquinolones are effective against gram-negative bacteria but should be used with caution due to their potential inhibitory effect on fracture healing and dose-dependent cytotoxic effects.[51] Penicillin G can be added for anaerobic coverage in severely contaminated (soil, grass, marine) wounds. As the prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA) increases, special attention should be given to geographic area of practice and community prevalence of MRSA infection.[52] The role of vancomycin in MRSA-colonized patients is currently being investigated.

Following antibiotic administration, dressing application, and adequate resuscitation, the patient should be taken to the operating room for a surgical debridement and fracture stabilization in a timely manner. Extensive exploration or manipulation of exposed bone should not be attempted in the emergency department. Previously it was thought that debridement should be performed within 6 hours of injury. More recent data have shown that time to debridement is not associated with the rate of infection as long as it is performed within 24 hours of injury.[53] While quality of debridement is felt to be more critical than time to debridement, rapid transfer to a trauma center with appropriate resources capable of definitively treating severely injured extremities in an urgent manner has been shown to be beneficial.[53,54] A thorough debridement includes the following: (1) sharp excision

TABLE 44-2: Gustilo-Anderson Classification of Open Fractures

Type	Description
I	Clean wound <1 cm, inside-out perforation, little or no contamination, simple fracture pattern
II	Skin laceration >1 cm, surrounding soft tissue without signs of contusion, vital musculature, moderate to severe fracture instability
III	Extensive soft tissue damage, wound contamination, exposed bone, marked fracture instability due to comminution or segmental defects
IIIA	Adequate soft tissue coverage of the fractured bone
IIIB	Exposed bone with periosteal stripping
IIIC	Any open fracture with associated arterial injury requiring vascular repair

Source: Data from Gustilo RB, Mendoza RM, William DN. Problems in the management of type III (severe) open fractures: a new classification of type III open fractures. *J Trauma.* 1984;24:742-746.

TABLE 44-3: Open Fracture Antibiotic Protocol

Type	Antibiotic	Duration
I	Cefazolin	24 hours
II	Cefazolin +/– aminoglycoside	24–48 hours
IIIA	Cefazolin + aminoglycoside Vancomycin + cefepime	72 hours
IIIB	Cefazolin + aminoglycoside Vancomycin + cefepime	72 hours
IIIC	Cefazolin + aminoglycoside Vancomycin + cefepime	72 hours
Soil contamination	Penicillin	Single dose
Marine contamination	Levaquin	Single dose

of all devascularized skin, subcutaneous tissue, muscle, fascia, and bone; (2) removal of all foreign material; and (3) copious irrigation of the wound bed using normal saline. There has been mixed evidence comparing high- versus low-pressure irrigation.[55] High-pressure lavage removed bacteria more effectively when compared to bulb syringe; however, some studies suggested that high-pressure lavage may further propagate bacteria into soft tissue.[56,57] A recent large multinational randomized controlled study of over 2500 patients demonstrated no difference in reoperation rate in patients treated with very-low-pressure (gravity), low-pressure, or high-pressure irrigation.[58] The same study reported a higher rate of reoperation in patients who received soap-based irrigation solution compared to normal saline irrigation.[58]

Fracture stabilization is performed after soft tissue and bony contamination and devitalization are adequately addressed. Primary closure of all open fracture wounds should be attempted if the surgeon feels all potentially nonviable tissue has been excised. Leaving an otherwise closable wound bed open can lead to the need for flap coverage and an increased infection rate in lower grade open fractures.[59] If there is any concern for ongoing contamination or tissue viability, a repeat surgical debridement should be planned independent of whether the wound is closed or left open. Lower deep infection rates have been observed with negative-pressure wound therapy (NPWT) using vacuum-assisted closure.[60] NPWT is frequently used in conjunction with local antibiotic delivery, such as antibiotic-impregnated cement beads or spacers, for severe open fractures. Wound coverage within 1 week should be the goal. One study demonstrated that flap coverage beyond 7 days is associated with a higher rate of infection that increases daily by 16%, after controlling for injury severity and multiple confounders.[31]

Mangled Extremity: Limb Salvage Versus Amputation

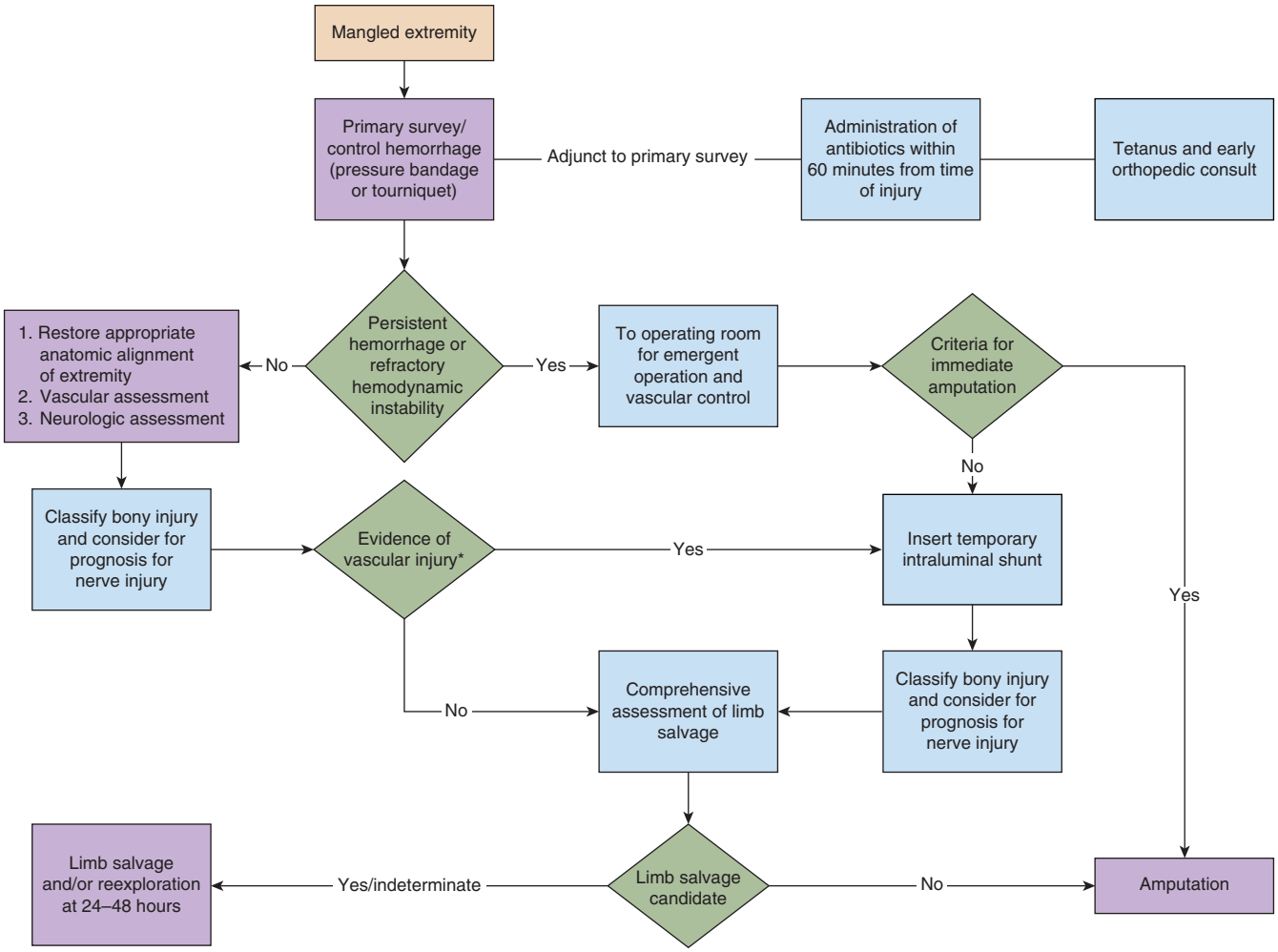
The term *mangled extremity* is used to describe a severely injured limb in which there is significant risk of amputation as a potential outcome. Irreparable injury to one or more structures in a mangled extremity, including muscle, tendons, ligaments, nerves, vessels, joints, or large segments of bone or skin, may significantly impair the function of a limb and lead to disability. Severe open fractures, especially type III open fractures and crush injuries, pose a great risk for significant injury to multiple elements critical to limb function. One of the most challenging decisions involved in the care of patients with a mangled extremity is whether to attempt salvage of a severely injured limb.[61] Initial efforts should be made to preserve functional and anatomic integrity. Amputation and ambulation with a prosthesis, however, may (1) provide better pain relief; (2) lead to a better functional outcome than an impaired native limb; (3) negate the need to undergo multiple procedures for limb reconstruction; and (4) avoid risk of systemic complications related to limb salvage such as infection, thromboembolic events, and anemia leading to blood transfusion(s).

In the acute phase, the decision to amputate will depend primarily on the physiologic condition of the patient and the feasibility of stabilizing and revascularizing the injured limb. An organizational protocol and multispecialty team approach to decision making are useful (Fig. 44-2). In the face of hemodynamic instability, significant hemorrhage, and the inability to revascularize the leg without increasing the risk of death, amputation becomes the primary option. In these cases, a guillotine-type amputation is appropriate, but every effort should be made to preserve length and all viable muscle and skin. Energy expenditure of an amputated limb depends on the level of amputation. Metabolic demand increases as the level of amputation ascends the lower extremity.[62] Proximal amputations have greater functional impairment, although technologic advancements (microprocessor knees) have significantly improved the function of above-knee amputees.

Similarly, meaningful strides have been made over the past decade with respect to foot and ankle prosthetics for below-knee amputees. Below-knee prostheses are highly functional; therefore, efforts to preserve the proximal tibia and knee may yield a better outcome. Free tissue transfers, rotational flaps, and skin grafts can all be used effectively to improve length and provide a durable stump. The decision to amputate during off hours in nonemergent scenarios, without opportunity for consultation with experienced limb salvage surgeons, should be avoided. Multidisciplinary decision making may provide more reconstructive options whether limb salvage or amputation is chosen.

When limb salvage is initiated, but complications arise, thoughtful informed decisions must be made regarding continuation of the salvage effort. Successful reconstruction typically requires multiple operations. The psychosocial factors that play into losing a limb are similar to the patterns of grief associated with losing a loved one.[63] Generally speaking, a patient who has sustained a catastrophic lower extremity injury will never have the same level of function as prior to the injury, whether salvage or amputation is performed. However, the functional recovery of a below-knee amputation can often outperform what can be obtained with a complex multistaged limb salvage. There is no difference in sickness impact profile, a multidimensional measure of self-reported health status, at 2 and 7 years between limb salvage and below-knee amputation.[64] Recovery, regardless of salvage or amputation, is more determined by economic, personal, and social factors than by injury or surgical factors.[64,65] If a patient is low functioning at baseline, a good functional outcome is unlikely regardless of which operation is performed.

Certain clinical findings can be key determining factors in limb salvage decisions. For example, necrosis of the plantar skin requiring free flaps and skin grafts to the sole of the foot are frequently not well tolerated and unlikely to be very functional (Fig. 44-3). Additionally, severe crush injuries to the foot, particularly when combined with severe open tibia fractures, may be best treated with early amputation due to the likelihood of foot and ankle stiffness and chronic pain with ambulation. Historically, several clinical indicators were used to help guide decision making, such as injury scoring



Practice guidelines are a resource of reference and do not replace clinical judgment.

FIGURE 44-2 Schematic algorithm of a mangled extremity protocol.

systems and the presence or absence of plantar sensation. The Lower Extremity Assessment Project (LEAP) found that injury scores were not predictive of a patient's likelihood to have success with limb salvage.[66,67] Similarly, the presence or absence of plantar sensation upon initial presentation was not predictive of plantar sensation after reconstruction.[68] A recent study comparing patients with severe lower limb injuries treated with limb salvage versus below-knee amputation may shed additional light on specific injury characteristics that dominate outcome.[69] Despite improvements in the body of evidence surrounding this difficult subject, much of the decision making remains patient and surgeon specific.

RADIOGRAPHIC EVALUATION OF PATIENTS WITH LOWER EXTREMITY TRAUMA

ATLS protocols suggest anterior-posterior (AP) chest and pelvis and adequate lateral cervical spine radiographs are indicated early in the evaluation and resuscitation of the injured patient during the primary survey.[2,18] The improved imaging capabilities of CT has led some to bypass the pelvis and cervical spine x-rays. CT provides better imaging of the cervical spine, but plain radiographs provide valuable information with pelvic fractures.

Radiographs of injured extremities are part of the secondary survey. Resuscitation of the patient should not be delayed or interrupted for radiographs of the extremities. Radiographs can be taken after urgent surgical treatment for other life-threatening injuries. In the unstable patient, care should be concurrent and not contiguous, such that radiographs and fracture stabilization occur concomitantly in the operating theater or resuscitation bay. If adequate extremity radiographs can be obtained without delaying other essential aspects of the evaluation and treatment of a trauma patient, they can be valuable in making the initial care plan.

Extremity radiographs should include orthogonal views of the entire bone in question (Table 44-4). Generally speaking, radiographs should be obtained prior to formal reduction and splint placement. Not only is the quality of the imaging better without overlying splint material, but knowledge

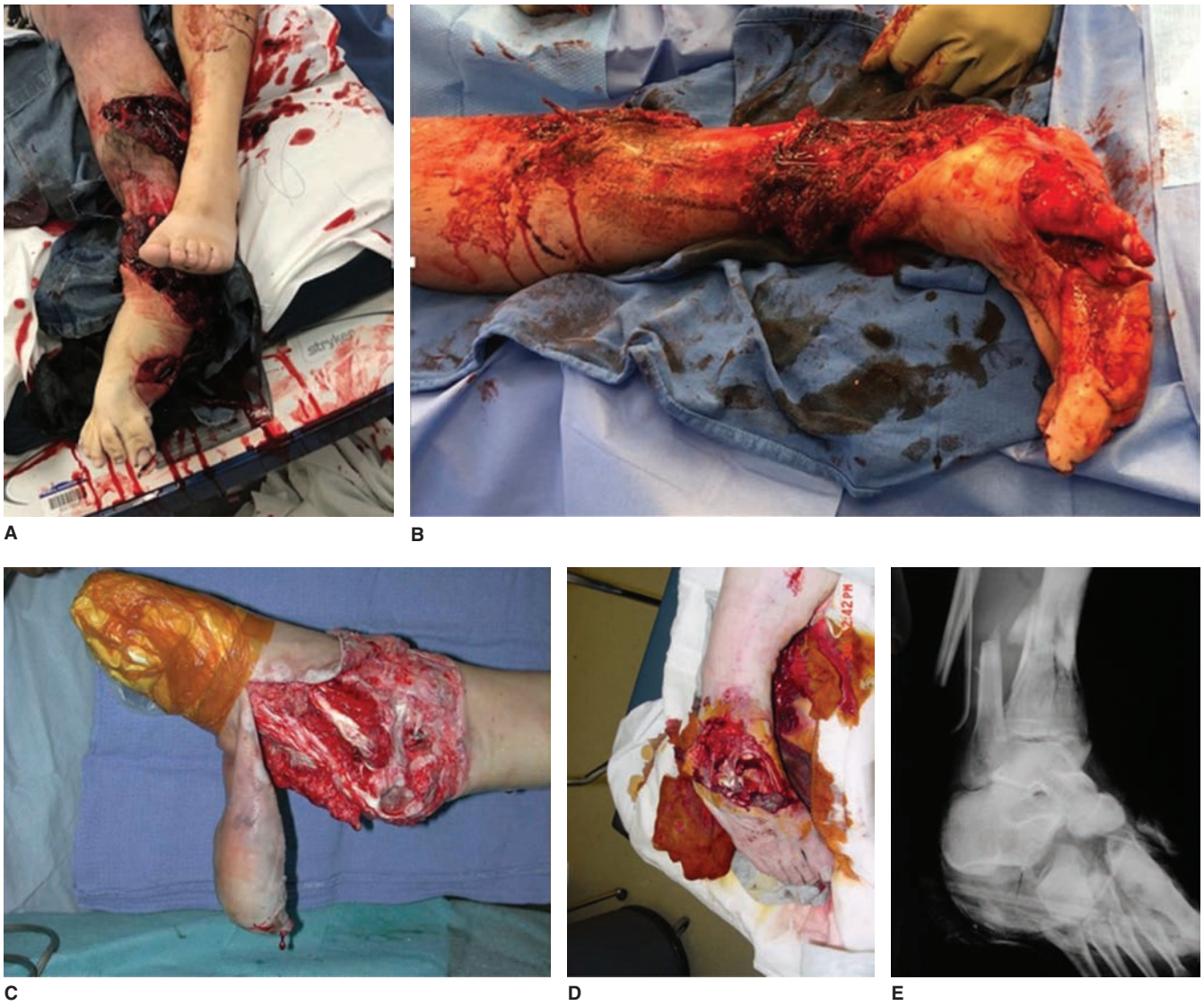


FIGURE 44-3 Examples of several mangled extremities. (**A** and **B**) Clinical images of combined severe tibia and foot trauma with extensive muscular damage, skin loss, and fractures. Clinical images of (**C**) a degloved plantar heel pad, (**D**) crushed foot with dorsal skin loss and tendon injury, and (**E**) corresponding oblique ankle radiograph illustrating the extent of osseous injury to the tibia, ankle, and foot. Severe combined tibia and foot injuries and dysvascular plantar heel pads are common indications for early below-knee amputation.

TABLE 44-4: Radiographic Views of the Lower Extremity

Body part	Standard views
Pelvis	Anteroposterior
Hip	Anteroposterior and cross-table or frog leg lateral
Femur	Anteroposterior and lateral
Knee	Anteroposterior and lateral
Tibia and fibula	Anteroposterior and lateral
Ankle	Anteroposterior, oblique (ankle mortise), and lateral
Foot	Anteroposterior, oblique, and lateral
Calcaneus	Lateral, Harris-Beath (axial)

of the injury pattern can guide surgical decision making. In the event of vascular compromise, reduction should proceed prior to imaging. This is more common with fractures and dislocations about the knee and ankle. After manipulation, reduction, and splint stabilization, the clinical exam should be repeated and radiographs obtained.

Some institutions have incorporated various forms of total-body CT as a rapid, comprehensive evaluation of the skeletal system. This has been common in Europe for many years. Depending on the protocol employed, the entire patient can be rapidly evaluated, obviating the need for multiple trips to the radiology suite, manipulation, and repositioning of the patient. Comprehensive body and extremity scanning may decrease discomfort for the patient, as well as minimize the time the patient is away from the resuscitation

area. Modern CT protocols can accomplish these scans with minimal increase in radiation exposure compared to multiple plain radiographs. Additionally, CT can identify subtle bone injuries that might go unnoticed on plain radiographs. Adoption of this type of imaging protocol requires collaboration between radiology, trauma surgery, emergency medicine, and orthopedic surgery and may require some practitioners to adapt to a different set of images than those to which they are accustomed.

Missed injuries in severely traumatized patients are common. One investigation reported an initial missed injury rate of 15% at a Level I trauma center, of which 70% were fractures.[70] Approximately 50% of missed fractures occurred in the lower extremities, with the foot being the most common.[70,71] Physical examination ("touch every bone"), identifying sites of tenderness, and radiographic imaging correlation can help avoid missed injuries.

Most complex articular fractures are best visualized with CT scan. CT provides detailed information crucial for preoperative planning prior to definitive articular fracture fixation. If a patient is hemodynamically stable and requires CT studies of the chest, abdomen, pelvis, or cervical spine, extremity CT may be obtained at the same time. However, if temporary stabilization with external fixation is planned, the CT is often more useful after restoration of limb length, alignment, and rotation achieved by external fixation. Early involvement of the orthopedic surgeon ensures proper imaging and may avoid unnecessary diagnostic studies.

TIMING AND MAGNITUDE OF FRACTURE FIXATION IN MULTIPLY INJURED PATIENTS

Initial treatment of orthopedic injuries in polytrauma patients is primarily dependent on the patient's evolving physiology. Hemodynamic stability, respiratory status, brain injury, resuscitation, coagulopathy, and additional traumatic injuries are considered prior to implementing a fracture treatment plan. The orthopedic traumatologist should coordinate with the general surgery trauma team and other consultants to formulate an individualized plan of care. Multiply injured patients can follow varying, often unpredictable, clinical trajectories. Continuous physiologic and resuscitative reevaluation is necessary to guide treatment.

Evolution of Early Total Care and Damage Control Orthopedics

In the 1950s, fracture management by traction and prolonged bedrest led to poor outcomes in trauma patients.[72] In the 1980s and early 1990s, early definitive fixation, termed *early total care*, was promoted, which facilitated early restoration of function. Increased complication rates were reported in patients undergoing early total care, and thus more conservative approaches were advocated.[73] In the 1990s and early 2000s, the term *damage control orthopedics* (DCO) was introduced, and studies demonstrated an improved survival in severely injured and underresuscitated patients initially treated with external fixation of femoral fractures in contrast to primary intramedullary nailing.[9,74]

DCO refers to the expedient provisional stabilization of pelvic and long bone fractures, typically via external fixation, in severely injured and physiologically unstable trauma patients. Common indications for DCO include hemodynamic instability (systolic blood pressure <90 mm Hg despite resuscitative efforts), persistent acidosis without trending improvement (pH <7.25, lactate >4.0 mmol/L, base excess –8 mmol/L), severe chest injury requiring significant ventilator assistance, and severe brain injury (Glasgow Coma Scale score [GCS] ≤8). The purpose of DCO is to avoid exacerbating the patient's altered physiologic condition by contributing to ongoing hemorrhage, which could worsen systemic acidosis, induce hypotension, and lead to organ perfusion deficits (eg, decreased cerebral perfusion pressure). DCO also avoids the second-hit phenomenon from major orthopedic surgeries. Immediately after trauma, there is a marked surge of inflammatory mediators.[75-77] The second hit can be a result of a variety of circumstances including infection, sepsis, ongoing hemorrhage (from injury or surgery), and an exaggerated inflammatory response (also from injury and/or secondary surgical procedures). A heightened and dysregulated postinjury inflammatory response has been implicated in the development of several adverse short-term outcomes including acute respiratory distress syndrome (ARDS), prolonged systemic inflammatory response syndrome, nosocomial infection, and multiple organ failure.[8,77-81]

Current Concepts of Early Appropriate Care

There is little debate that hemodynamically unstable and severely underresuscitated trauma patients benefit from DCO measures. Likewise, most agree that it is generally safe to proceed with early definitive fracture fixation in physiologically stable patients without significant head injury. There is considerably more controversy surrounding the care of polytrauma patients with "borderline" physiology (systolic blood pressure 80–100 mm Hg, lactate ~2.5 mg/dL, blood transfusion of 2–8 units, abdominal and/or chest Abbreviated Injury Scale score of 2 or 3). Pape et al[77] reported a lower risk of systemic complications, especially pulmonary dysfunction, with DCO (external fixation) compared to early intramedullary nailing of borderline patients with femur fractures. Recent data by Vallier and colleagues support the concept of early appropriate care, defined as early definitive fixation of spine, pelvis, acetabular, and femur fractures in appropriately resuscitated patients, to minimize systemic complications, including infection, and reduce hospital length of stay.[12,82,83] Early appropriate care of axially unstable fractures is generally recommended when the following physiologic parameters are met: pH greater than 7.25, lactate less than 4.0 mmol/L, base deficit less than 5.5 trending positive, systolic blood pressure greater than 90 mm Hg, and absence of brain injury causing

elevated intracranial pressure.[82-85] Inappropriate timing of surgery not only impacts outcomes in severely injured patients but is also associated with an increased cost of care.[10,14]

Considerations in Polytrauma Patients with an Associated Injury to the Head or Chest

Although some studies have reported higher rates of ARDS (>25%) in patients with lung injuries when early definitive femoral fixation strategies were used, a recent investigation showed no difference in outcome with early total care (2%).[9,13,86] The debate is compounded by the wide spectrum of acute lung injury and subjectivity encompassing the diagnosis of posttraumatic ARDS after fracture fixation. An international panel of experts, the ARDS Definition Task Force, was charged with refining the Berlin Definition, which resulted in improved mortality predictive validity.[87] A careful assessment of a patient's chest injury should be an integral part of decision making when choosing the timing of long bone fracture stabilization.

Secondary brain injury is a result of systemic inflammation, hypoxia, acidosis, fat embolization from a fracture site, and coagulopathy.[88] The GCS is a valuable tool in evaluation of neurologic status and timing of surgery.[88] GCS >13 with a normal CT is appropriate for early definitive treatment of a femur fracture. Patients with a moderate brain injury (GCS 9–13) and minor intracranial pathology are candidates for early appropriate care, but DCO measures should be considered based on recommendations from the neurosurgical and trauma services.[89] Severe traumatic brain injury (GCS <9) patients should be treated with damage control measures, especially in the face of significant intracranial pathology (eg, edema, midline shift, subdural/epidural bleeding).[89] Conversion from external fixation to internal fixation can be planned once GCS is greater than 11 and/or stable intracranial pressure (<20 mm Hg) and cerebral perfusion pressure (>80 mm Hg) over 48 hours are achieved.[89]

Postinjury Immunologic Response of Trauma Patients

Trauma incites a robust systemic inflammatory response. Endogenous "danger" signals, termed *alarmins* or *damage-associated molecular patterns* (DAMPs), are released following injury and propagate inflammation.[90] An exaggerated postinjury inflammatory response, often manifested by the systemic inflammatory response syndrome, has been implicated in adverse outcomes, such as multiple organ failure, nosocomial infection, complicated clinical course, and death.[91]

The two-hit hypothesis refers to an initial traumatic event (eg, tissue damage, hemorrhage) and secondary insults (eg, ischemia/reperfusion, necrotic tissue, infection, additional surgery) that activate the innate immune system in a potentially amplified fashion. This is followed by a compensatory anti-inflammatory response, characterized by suppression of the adaptive immune system. An imbalanced and dysregulated immune response can render host defenses vulnerable to infection and organ dysfunction.[91] An alternative model of the postinjury immunologic response, often referred to as a *genomic storm*, was proposed following a large-scale genomic analysis.[92] Authors described a rapid upregulation of the innate immune system and simultaneous suppression of the adaptive immune system.[92] Recent computational analyses have linked inflammatory biomarker networks with an increased risk of developing nosocomial infection and organ dysfunction.[81,93] Few studies have examined the impact of immunoactive mediators in an orthopedic trauma population.[94-96]

An improved understanding of the immunologic response following trauma may offer additional insight into decisions surrounding the timing and magnitude of fracture fixation. Titration of initial and staged orthopedic interventions in polytrauma patients is a collaborative effort and directed by multidisciplinary input.

MANAGEMENT OF COMMON FRACTURES AND DISLOCATIONS

Acetabular Fractures

Fractures of the acetabulum are articular injuries of the pelvic portion of the hip joint, which can have profound implications on its long-term function. The weight-bearing dome of the acetabulum is defined as the superior 10 mm of the acetabulum as measured on CT, which corresponds to 45° roof arc measurements on radiographs.[97] Fractures with displacement of 2 mm or greater within the dome typically are treated surgically. Fractures involving the lower half of the acetabulum often do not require treatment unless an associated pelvic ring injury requires treatment or there is hip instability. Successful open reduction and internal fixation (ORIF) of displaced acetabular fractures significantly improves the prognosis of these severe injuries. Judet et al[98] developed a classification system that illustrates five simple and five associated fracture types. Their seminal work has significantly improved the understanding and management of acetabular fractures.[98]

An understanding of acetabular morphology and surrounding anatomy is essential for identifying and classifying fracture patterns, formulating a treatment plan, and safely executing surgical treatment. The acetabulum is located in the concavity of an arch (open arms of an inverted "Y") formed by two columns of bone. The anterior column (iliopubic segment) includes the iliac crest, anterior iliac wing (including the anterior superior iliac spine and anterior inferior iliac spine), anterior wall, anterior pelvic brim, and superior pubic ramus. The posterior column (ilioischial segment) consists of the inferior aspect of the sciatic buttress (adjacent to the greater sciatic notch), posterior wall, majority of the quadrilateral plate, ischial spine, and ischial tuberosity. Compact bone between two columns at its apex constitutes the roof of the acetabulum and forms the keystone of the arch. Assessment of radiographic landmarks on the AP pelvis view can allow for rapid identification of acetabular injuries. A break

or discontinuation of any of these radiographic lines should warrant further investigation with obturator oblique and iliac oblique Judet radiographs and CT scan with three-dimensional reconstructions.[99] CT aids in fracture classification, measurement of displacement, identification of incarcerated fracture fragments, assessment of marginal impaction, and determination of the ideal surgical approach. Knowledge of the vascular tree of the true and false pelvis is critical to safely performing acetabular surgery. ORIF of the acetabulum is typically performed by orthopedic trauma surgeons with pelvic operative experience. Cautious identification and protection of the external iliac artery/vein, obturator artery/vein, deep branches of the internal iliac system, and ligation of the corona mortis is critical in anterior approaches. Posterior approaches necessitate careful mobilization of the sciatic nerve, avoidance of the medial femoral circumflex artery, and protection of the superior and inferior gluteal vessels.

Specific acetabular fracture patterns result from the direction of force applied to the hip and the position of the femoral head when it impacts the acetabulum. An axial load to the femur with the hip in the flexed or neutral position will force the femoral head against the posterior articular surface of the acetabulum, leading to a posterior wall fracture, with or without hip dislocation. An abducted leg position directs the force on the acetabulum more medially and will lead to a transverse fracture that is occasionally associated with posterior wall. A transverse fracture of the acetabulum is a major injury that separates the acetabulum into cephalad and caudal fragments. The degree of displacement and comminution in acetabular fractures depends on not only the magnitude of energy involved, but also an individual's bone density. As in most lower extremity injuries, there is a bimodal distribution in acetabular fractures: high-energy fractures typically occur in young patients, and low-energy fractures usually result from falls in elderly patients with poor-quality bone. In high-energy injuries, fracture patterns closely reflect those described by Judet et al.[98] However, in osteopenic patients, a low-energy fall with an impact on the lateral thigh often results in an anteromedial force through the greater trochanter, leading to a displaced anterior column fracture with quadrilateral plate (central portion of acetabulum) involvement. This fracture is classified as an anterior column–posterior hemitransverse pattern. Very low anterior column fractures are typically extensions of superior pubic ramus fractures and are treated as pelvic ring injuries rather than acetabular fractures (Fig. 44-4). The bone quality and fracture mechanism should be considered when evaluating patients with acetabular fractures because there is a high association of other orthopedic

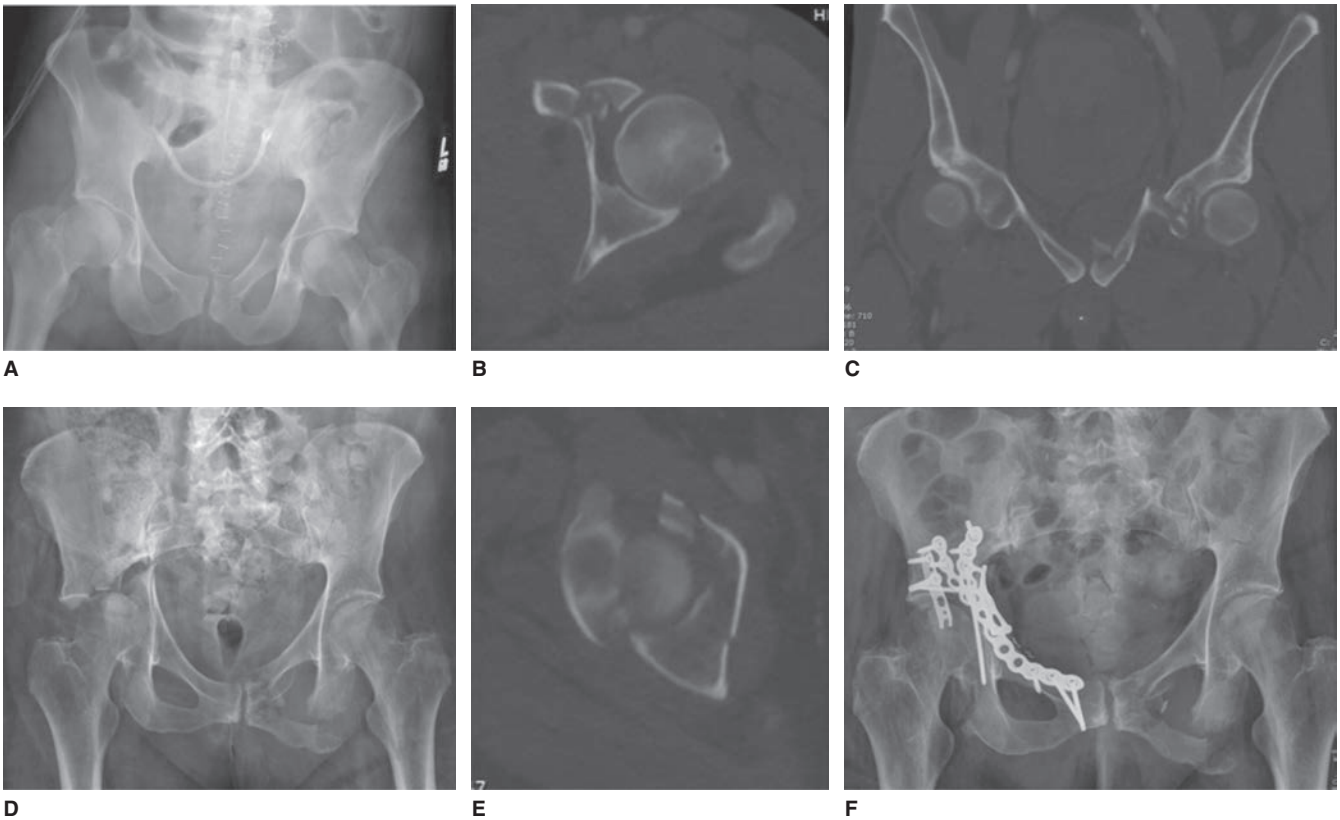


FIGURE 44-4 Low anterior column acetabular fracture of the left hip: (**A**) anteroposterior (AP) pelvis radiograph, (**B**) axial computed tomography (CT) scan, and (**C**) coronal CT scan. Although technically an acetabulum fracture, low anterior column fractures are typically manifestations of pelvic ring injuries and may not require anatomic fixation as in acetabulum fractures that involve the weight-bearing dome. Transverse–posterior wall acetabular fracture of the right hip: (**D**) AP pelvis radiograph, (**E**) axial CT scan, and (**F**) postoperative AP pelvis radiograph following treatment with open reduction and internal fixation via a combined anterior and posterior approach.

and systemic injuries. Careful evaluation of skin overlying the pelvis, hip, and thigh may identify a Morel-Lavallée lesion, defined as a closed, internal degloving injury when the subcutaneous fat avulses off the deep fascia, creating a potential space for fluid accumulation. Nerve palsies, most commonly affecting the peroneal division of the sciatic nerve, occur in up to 15% of acetabular fractures associated with posterior hip dislocations.[100,101]

Common indications for surgical management of acetabular fractures include displacement greater than 2 mm within the weight-bearing dome,[102] instability noted on examination under anesthesia,[103,104] hip incongruity, intra-articular fragments superior to the fovea, and open fractures. The surgical approach is dictated by the fracture pattern and the overall condition of the patient. Options for anterior column and transverse fracture patterns include the ilioinguinal approach, anterior intrapelvic approach (Stoppa),[105] lateral window portion of the ilioinguinal, and limited iliofemoral approach (extension of a lateral window into an anterior approach to the hip). The majority of posterior column fractures and most transverse fractures are exposed via the Kocher-Langenbeck approach. The extended iliofemoral approach is extensile but rarely used due to high rates of infection and heterotopic ossification. Occasionally, combined anterior and posterior approaches are employed to address both anterior and posterior column displacement in associated fracture types (eg, both-column, T-type, transverse-posterior wall patterns) that cannot be reduced by a single exposure. Occasionally the optimal approach must be abandoned due to associated wounds or initial treatments such as laparotomy, suprapubic catheter, and nonselective arterial embolization to major pelvic vasculature.[106] Restoring articular congruity and achieving stable fixation are critical in acetabular surgery as accuracy of fracture reduction has been shown to correlate with hip survival (prevention of posttraumatic arthritis and conversion to arthroplasty).[107] Minimally invasive percutaneous screw fixation represents a viable alternative to ORIF in minimally displaced fractures or in patients at risk for significant wound complications due to an extensive surgical procedure.[108,109]

Timing of acetabular surgery depends on the patient's physiologic condition. Fixation and mobilization within 24 hours in appropriately resuscitated patients carry a lower risk of systemic complications.[82] Acetabular fractures are usually closed injuries, without need for immediate operation. Some authors contend that intraoperative blood loss can be minimized if surgery is delayed for 2 to 3 days. However, two recent investigations found no difference in blood loss when anterior column fractures were treated within 48 hours compared to after 48 hours[110] or when posterior wall fractures were repaired before 24 hours compared to after 24 hours.[111] Short-term traction may be indicated in unstable fracture patterns to maintain hip reduction and prevent femoral head protrusion into the pelvis. Postreduction CT is preferred to prereduction CT when fractures are associated with dislocations, as it not only confirms reduction, but also identifies incarcerated or intra-articular fracture fragments that may influence surgical planning.

Acetabular fractures in osteoporotic individuals pose unique problems. There is frequently significant comminution, protrusion deformity, and poor bone quality, which increases the chance of postoperative fixation failure. In these instances, immediate total hip arthroplasty, either combined with ORIF or specialized acetabular reconstruction devices, allows improved fixation and early weight bearing in elderly patients.[112,113]

Patients with pelvic and acetabular fractures have a significant risk of thromboembolic complications. Intermittent venous compression devices, pharmacologic anticoagulation, and insertion of retrievable inferior vena cava filters for high-risk patients are all appropriate strategies. Prophylaxis against heterotopic ossification following open reduction through a posterior approach is preferred by most surgeons. Evidence supports radiation-based prophylaxis (single dose of 700 cGy) compared to pharmacologic-based prophylaxis (indomethacin), which has been shown to be associated with acetabular nonunion.[114]

Treatment of acetabular fractures is also associated with significant blood loss. Cell saver has been shown to be potentially useful for select anterior fracture patterns when large-volume blood loss is anticipated.[115] Two recent meta-analyses reported that intraoperative administration of tranexamic acid may reduce rates of blood transfusion in orthopedic trauma surgery.[116,117] However, a randomized controlled trial on the use of tranexamic acid in acetabular surgery reported no benefit to its use.[118] Acetabular fracture surgery remains among the most challenging procedures in orthopedics. These difficult and high-risk reconstructive surgeries should be performed in specialized trauma centers with experienced surgeons to ensure optimal treatment.[119]

Hip Dislocation and Femoral Head Fractures

The hip joint depends on bony architecture and soft tissue constraints for its stability. Significant force is required to dislocate the native hip joint. The most common mechanism of injury is a direct posterior force through a flexed knee and adducted hip in a motor vehicle accident. Some patients with pure hip dislocations may have slightly abnormal native anatomy (acetabular retroversion) predisposing them to hip dislocation.[120] The clinical appearance of a patient with a posterior hip dislocation is hip flexion, adduction, internal rotation, and resistance to motion. Patients are usually in significant pain. This appearance may be lacking if a significant fracture of the posterior wall exists. Anterior dislocation results in external rotation and abduction of the affected extremity with slight hip flexion. The typical mechanism of injury leading to anterior dislocation is forced external rotation and abduction of the hip joint. Anterior dislocation occurs less frequently (<10%) compared to posterior dislocation.[121]

The primary blood supply to the femoral head in adults is predominantly from the medial femoral circumflex artery through the retinacular arteries that branch off from an extracapsular ring at the base of the femoral neck. The lateral

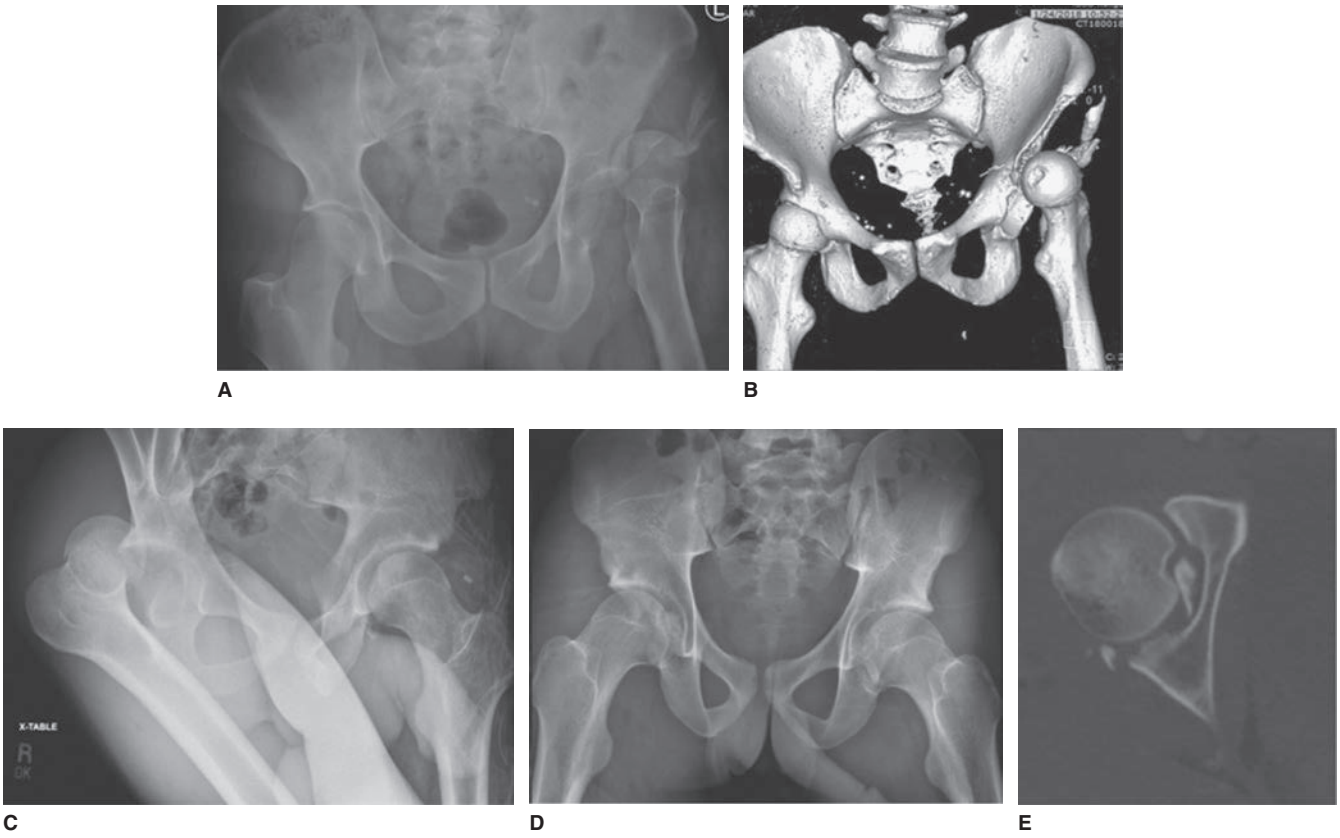


FIGURE 44-5 Examples of hip dislocations. (**A**) Anteroposterior (AP) pelvis radiograph and (**B**) three-dimensional computed tomography (CT) reconstruction of a left anterior hip dislocation. (**C**) AP pelvis injury radiograph of a right posterior hip dislocation. (**D**) Postreduction radiograph. (**E**) Postreduction axial CT scan demonstrating a small posterior wall acetabulum fracture and a large intra-articular fracture fragment requiring excision.

femoral circumflex artery and the superior and inferior gluteal arteries provide minor contributions to femoral head perfusion. High-energy injury involving damage to the blood supply can result in osteonecrosis and resultant collapse of the femoral head. This can be a devastating complication with lifelong implications, particular for young patients, as total hip replacement is usually the only viable treatment. The sciatic nerve runs in close proximity to the posterior aspect of the hip joint. Ipsilateral sciatic nerve injury occurs in 10% to 15% of hip dislocations, with the peroneal trunk being most commonly affected, leading to foot drop. Urgent hip reduction is critical to minimize ongoing compression injury to the nerve and minimize the risk of osteonecrosis of the femoral head. There is no specific treatment for sciatic nerve injuries. Up to 70% to 80% of sciatic nerve palsies resolve over a period of months. Associated orthopedic injuries occur in 60% to 70% of patients with traumatic hip dislocations and fracture dislocations.[122] Ipsilateral knee injuries including ligamentous disruption, patella fracture, distal femur fracture, and proximal tibia fracture are quite common.[123] Other systemic injuries such as head, craniofacial, thoracic aortic, and abdominal visceral injuries may be seen in deceleration injury patterns.[122,124]

An AP pelvis radiograph should be obtained as part of the ATLS protocol. This radiograph usually shows obvious signs of a hip dislocation or fracture-dislocation. A posterior hip dislocation will show a smaller femoral head on the injured side as the femoral head is closer to the cassette. An anterior dislocation, in contrast, will demonstrate a larger femoral head as the femur is further from the cassette, creating a larger radiographic shadow (Fig. 44-5). Anterior dislocations may show combined superior (pubic) or inferior (obturator) dislocation if the hip is in extension or flexion at the time of injury, respectively. Care should be taken to scrutinize radiographs for ipsilateral femoral neck fractures, which are a contraindication for closed hip reduction in the emergency department.

Urgent closed reduction of the hip joint should be performed to decrease the risk of irreversible avascular necrosis or sciatic nerve injury. The importance of prompt reduction less than 6 hours from injury has been demonstrated in retrospective and prospective studies to minimize the risk of avascular necrosis.[125] Closed reduction is best performed with adequate intravenous sedation and muscle relaxation. Ideally, reduction would occur before the patient has the standard trauma head/neck/chest/abdomen/pelvis CT scan because delays can increase the risk of avascular necrosis and sciatic nerve injury. Additionally, all hip dislocations require a postreduction CT scan to evaluate for concentric reduction and identify residual incarcerated bone fragments

(Fig. 44-5). Therefore, if reduction occurs before the trauma scan, an additional pelvic CT scan can be avoided.

There are various described techniques for reduction of the hip joint, with the general principle being in-line traction combined with re-creation of the injury pattern. For posterior dislocations, a direct anterior force applied through a bent knee is often effective while two assistants are holding counterpressure on the patient's anterior superior iliac spines bilaterally. This maneuver should begin with the leg internally rotated and adducted to clear the posterior wall. The clinician should then progress to external rotation once traction is applied. Care must be taken to avoid unnecessary force because iatrogenic fracture, particularly femoral neck fracture, is a rare complication. After relocation of the femoral head, the hip should be taken through a range of motion in order to examine for instability that will guide treatment. Postreduction skeletal traction may be necessary if the hip remains unstable or if incarcerated fragments remain in the joint. Acetabular wall fractures of significant size can result in instability, which is an indication for traction. If the hip is reduced, the surgery can be delayed until the patient is properly resuscitated and optimized. Purely ligamentous injuries are usually inherently stable, and most surgeons recommend a 4- to 6-week period of protected weight bearing and hip precautions based on the direction of the dislocation.

Fracture of the femoral head is a rare injury pattern and often associated with hip dislocations. The presence and size of a femoral head fragment depend on the position of the hip at the time of impact, with a straighter leg and hip associated with larger fracture fragments. These injuries are usually seen in high-energy mechanisms. The incidence of femoral head fractures is increasing as resuscitation protocols improve. Radiographic characteristics of irreducible femoral head fracture-dislocation are posterior-superior dislocation with sagittal plane femoral head fracture, intact posterior wall of acetabulum, and close apposition of proximal femur to the supra-acetabular ilium (Fig. 44-6).[126] Postreduction CT scans should be obtained in order to assess the concentricity of

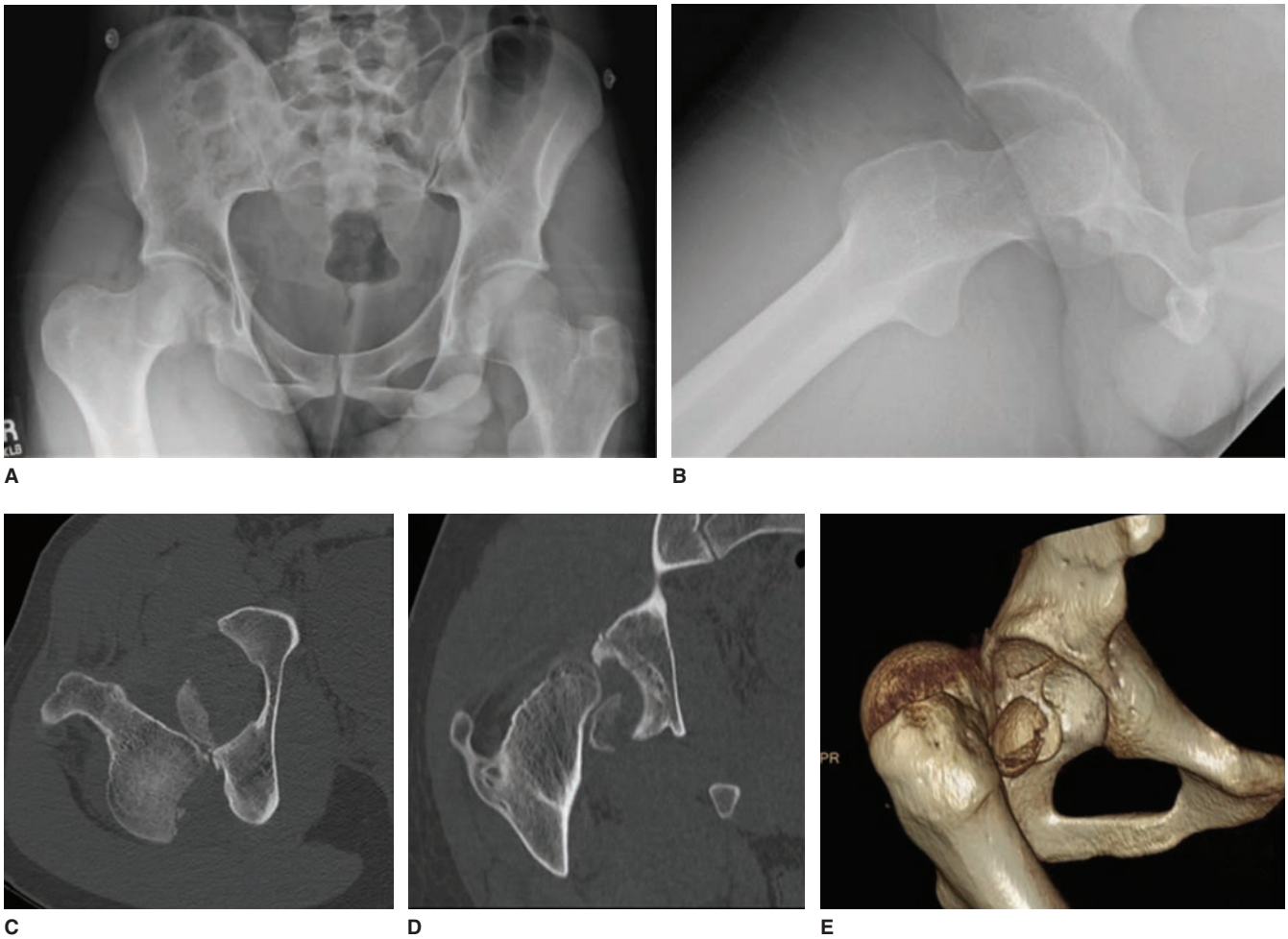


FIGURE 44-6 Irreducible hip dislocation. (**A**) Anteroposterior (AP) pelvis radiograph and (**B**) cross-table lateral radiograph of the right hip demonstrating a right posterior hip dislocation. Note the nearly normal appearance of the right hip joint on the AP image in contrast to the cross-table lateral view that clearly demonstrates the posterior dislocation. (**C**) Axial computed tomography (CT), (**D**) coronal CT, and (**E**) three-dimensional CT reconstruction demonstrating the posterior dislocation with a residual femoral head fracture fragment blocking the reduction of the femoral head.

reduction, the size and location of the femoral head fracture fragment, and for associated bony injuries of the acetabulum and femoral neck. Irreducible femoral head fracture-dislocations should be taken promptly to the operating room for an open reduction, but only after the appropriate orthopedic trauma surgical team is mobilized because the reduction can be technically challenging.[127] Surgical approaches include a posterior Kocher-Langenbeck approach with or without a trochanteric osteotomy and surgical hip dislocation[128] and an anterior Smith-Peterson approach to the hip.

The Pipkin classification describes the femoral head fracture in relation to the fovea (depression in the femoral head where the ligamentum teres attaches) as follows: type 1, fracture inferior to the fovea; type 2, fracture superior to the fovea; type 3, any head fracture with an associated femoral neck fracture; and type 4, any fracture with an associated acetabular fracture.[129] The majority of Pipkin type 3 and 4 fractures are treated operatively, along with most displaced (≥2 mm) Pipkin type 2 fractures. Most minimally displaced Pipkin type 1 fractures can be managed nonoperatively if the hip is concentrically reduced. Nonoperative management consists of a period of protected weight bearing, often with motion restrictions to prevent dislocation. Older patients with osteoporotic bone or comminuted fractures may be treated with arthroplasty to facilitate early mobilization and immediate weight bearing.

Posttraumatic arthritis and avascular necrosis are the most common complications following hip dislocation and fracture-dislocation. These are life-altering complications when they occur in young patients. The treatment for both of these complications is total hip arthroplasty. Although somewhat undesirable in younger patients, arthroplasty provides excellent pain relief and function, with modern implants lasting much longer than earlier generation implants (up to 20–30 years). Heterotopic ossification can be seen, particularly with patients with concomitant head injuries. Hip arthroscopy has improved substantially over the past decade and warrants a consideration in the treatment algorithm to address loose bodies and other intra-articular pathology after hip dislocation and fracture-dislocation.

Fractures of Proximal Femur—"Hip Fractures"

Hip fracture is a term often used to describe femoral neck and intertrochanteric femur fractures (Fig. 44-7). Like most orthopedic injuries, hip fractures have a bimodal distribution. In young patients (<50 years), fractures of the proximal femur are typically high energy with more comminution and displacement. The more common geriatric hip fracture tends to be lower energy. Worldwide, there were 1.7 million hip fractures in 1990, and this is projected to increase to 6.3 million in 2050. In the United States alone, this number is expected to be between 500,000 and 1 million hip fractures per year.[130] Hip fractures are associated with significant morbidity and mortality, often to the surprise of patients and

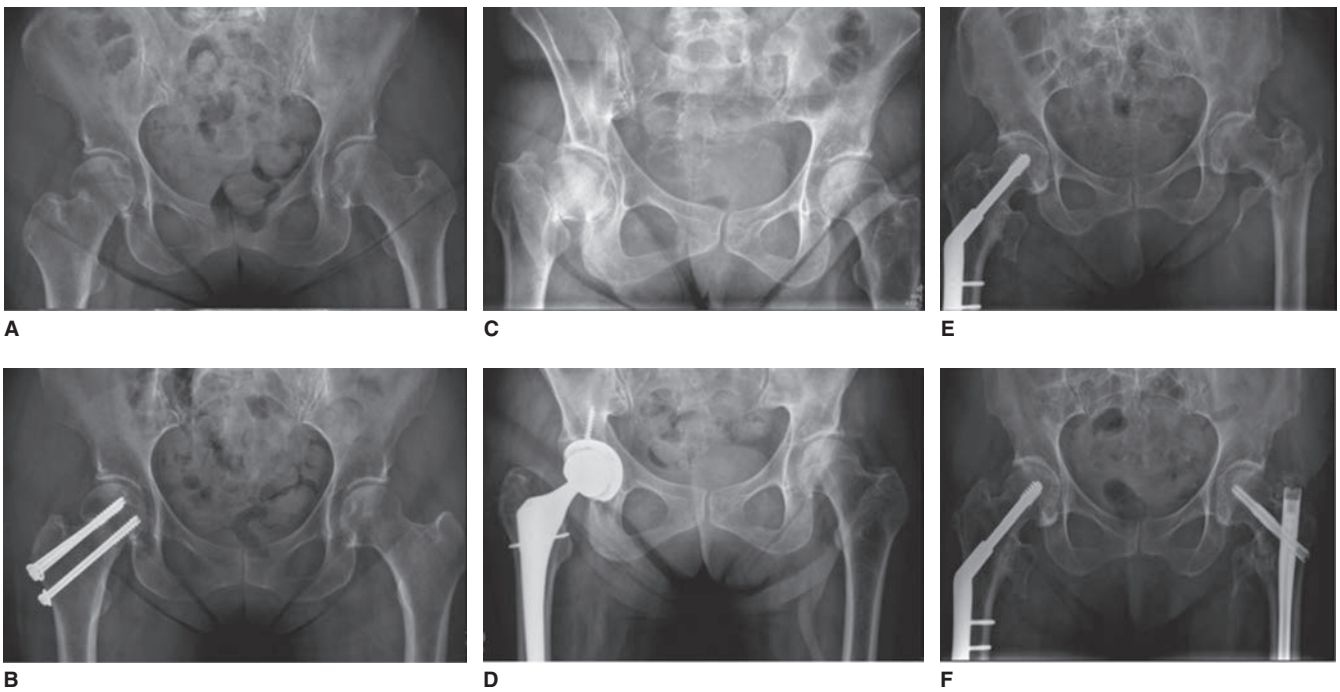


FIGURE 44-7 Three different types of hip fractures. (**A**) Preoperative and (**B**) postoperative anteroposterior (AP) pelvis radiographs of a minimally displaced right femoral neck fracture treated with percutaneously placed cannulated screws. (**C**) Preoperative and (**D**) postoperative AP pelvis radiographs of a displaced right femoral neck fracture treated with total hip arthroplasty. (**E**) Preoperative and (**F**) postoperative AP pelvis radiographs of a stable left intertrochanteric femur fracture treated with a compression hip nail. The right hip was previously treated with a compression hip screw and side plate.

families who suffer this often life-changing injury. Up to 20% of elderly hip fracture patients require long-term nursing home care, and only 40% regain their prefracture level of independence.[131] By definition, a patient over age 55 who sustains a hip fracture has osteoporosis and carries a risk of future osteoporotic fractures as high as 17 times that of age-matched controls.[132,133] Reported mortality rate following the hip fracture surgery is 9% at 30 days, 19% at 90 days, and 30% at 12 months.[134] The mortality associated with osteoporotic fractures is greater than the combined mortality of breast and ovarian cancer.[135] Various classifications based on fracture location, displacement, and stability help define treatment options. Although often grouped together, fractures of the proximal femur can have significantly different treatment methods based on a patient's age, function, and intracapsular or extracapsular location.

Fracture healing rates and prognosis for hip fractures are highly dependent on the vascular supply to the femoral neck and head. The ascending cervical arterial branches that supply the femoral head arise from an arterial ring at the base of the femoral neck. Extracapsular fractures at the base of the neck (basicervical) or distal to it (intertrochanteric) usually do not damage these arteries. Intracapsular fractures proximal to the arterial perforation of the femoral neck can disrupt these vessels or cause local circulatory compromise due to intracapsular bleeding and tamponade. In addition, an intracapsular fracture will be exposed to the synovial fluid within the hip joint, which can interfere with healing. Blood supply to the femoral head may not be compromised in nondisplaced fractures.

The medial aspect of the femoral neck is the compressive side and the lateral aspect the tension side. The average neck-shaft angle is 130° with 10° of anteversion. Hip abductors (gluteus medius and minimus) insert on the greater trochanter, and the iliopsoas muscle inserts on the lesser trochanter. Standard diagnostic imaging consists of an AP pelvis, cross-table lateral hip, and orthogonal femur films. CT scan can aid in diagnosis and preoperative planning in select circumstances.

Femoral neck fractures are classified based on the following: (1) location: subcapital (below the head), transcervical (mid neck), or basicervical (base of the neck); (2) stability: Garden classification; and (3) fracture line orientation (Pauwel's classification). Displaced (Garden 3 and 4) femoral neck fractures portend more instability and are associated with a higher likelihood of fracture fixation failure. The AO Foundation (AO)/Orthopaedic Trauma Association (OTA) classification is often used for intertrochanteric femur fractures.[136] Intertrochanteric fractures without comminution, lesser trochanteric involvement, multiple fragments, or posteromedial buttress extension are considered stable. Fractures that extend laterally below the greater trochanter render the lateral wall incompetent and are considered unstable.[136] Reverse obliquity intertrochanteric fractures are also considered unstable.[136]

Nearly all patients with hip fractures benefit from operative stabilization to promote healing and allow immediate mobilization. Young femoral neck fracture patients are typically treated with closed reduction and internal fixation for nondisplaced fractures or ORIF for displaced fractures. An intracapsular femoral neck fracture in a young patient should be addressed as soon as possible because viability of the femoral head is at risk. Not only can the ascending arteries along the femoral neck be severed or avulsed if the fracture is displaced, but also they can be kinked and subsequently thrombose. These small-caliber vessels cannot be repaired, although timely fracture reduction may potentially preserve vascularity. The efficacy of intracapsular hematoma decompression is controversial. Because there is no way to preoperatively assess femoral head perfusion, attempts to preserve the viability of the femoral head should proceed when the appropriate surgical team is assembled, ideally within 24 hours. Reduction quality is still considered to be more important than timing to fixation; thus, emergent surgery with an inexperienced and ill-equipped team is discouraged.

Avascular necrosis is a complication reported in 10% to 30% of femoral neck fractures. Portions of the subchondral bone may collapse, leading to joint surface irregularity, loss of femoral head sphericity, and posttraumatic arthritis. Total hip replacement is usually the only reconstructive option is these situations. Degenerative changes due to avascular necrosis contribute to a reoperation rate ranging from 17% in young patients to 53% in an older population.[137,138] Despite the complication rates, internal fixation usually remains the best option for patients less than 50 to 55 years old due to the superior function of the native femoral head compared to total hip arthroplasty.

Displaced geriatric femoral neck fractures are usually treated with arthroplasty to minimize the rate of reoperation and facilitate immediate mobilization. In general, high-functioning elderly patients without extensive medical comorbidities should undergo total hip arthroplasty, regardless of age.[138] Lower functioning geriatric patients with minimal ambulatory capacity and multiple comorbidities are better candidates for hemiarthroplasty.[138] Elderly patients with nondisplaced femoral neck fractures can be treated effectively with in situ pinning permitting immediate weight bearing.

Intertrochanteric fractures in patients of all ages are treated surgically. For stable fractures, either a sliding hip screw or cephalomedullary nail (CMN) is appropriate (Fig. 44-7). For unstable fractures (lateral wall incompetence, four-part fracture, and reverse obliquity patterns), however, CMN is the implant of choice due to a higher failure rate of sliding hip screw.[139] Greater than 90% of intertrochanteric femur fractures heal uneventfully, and precise placement of the lag screw in the center of the femoral head close to the subchondral bone minimizes the risk of fixation failure.[140] Recently, an analysis of American College of Surgeons National Surgical Quality Improvement Program illustrated that although the overall outcomes are similar between the sliding hip screw and CMN, patients treated with CMN had a greater than 1 day shorter hospital length of stay after hip fracture, thus negating the extra cost incurred by using CMN.[141]

Geriatric patients with hip fractures benefit from expeditious medical optimization and surgical fixation within 24 to

48 hours from injury. Delay in surgery for patients with hip fractures has been associated with increased rates of mortality, increased in-hospital complications, and slower return to function.[142,143] Surgical delay of more than 24 hours increases the risk of 30-day and 90-day mortality.[134,144] An echocardiogram should be obtained to assess patients with a new-onset or previously undiagnosed murmur because significant aortic stenosis may influence anesthetic management. However, authors have found that most preoperative testing for hip fracture patients often delays surgery and rarely influences management.[145] Communication between the medical and surgical teams and adherence to the American College of Cardiology/American Heart Association guidelines on indications for preoperative testing may avoid delays in surgical treatment by foregoing testing that will not impact the care of the patient.[146,147]

Many hospitals have recently developed a multispecialty hip fracture service to provide comprehensive inpatient and outpatient care for hip fracture patients. This includes rapid, cost-effective preoperative testing and optimization. Surgical decision making focuses on choosing an implant that allows the patient to bear weight immediately postoperatively. A coordinated inpatient team includes the following: orthopedic surgeon, hospitalist or geriatrician, dietician, physical therapist, social worker, and case management. This team focuses on initiating osteoporosis management, delirium prevention, mobilization, and appropriate discharge disposition. Close multidisciplinary follow-up is established to optimize the patient's bone health during the healing stages of the fracture with establishment of an osteoporosis treatment plan. There is considerable evidence to suggest that a comprehensive approach decreases complications and opioid use, shortens length of stay, and decreases readmissions, while improving 30-day and long-term mortality and return to activities of daily living, all without an increase in costs.[148]

Subtrochanteric and Femoral Shaft Fractures

Subtrochanteric femur fractures are defined as fractures involving the region between the lesser trochanter and 5 cm distal, including patterns that extend into the intertrochanteric region and base of the femoral neck. Physiologically, subtrochanteric and femoral shaft fractures are very similar; however, there are some nuances between them that dictate varying treatment considerations.

Femoral shaft fractures are usually due to high-energy trauma and can be associated with blood loss of up to 1500 to 2000 mL in the thigh. Thus, these injuries can cause, or contribute to, hemorrhagic shock. Many patients suffer severe associated injuries to the torso, pelvis, and other extremities. The femur is the strongest bone in the body; therefore, the force required to fracture it in young, healthy patients is significant. Patients present with a shortened, externally rotated limb, pain on palpation, and gross deformity. A detailed neurovascular exam should be documented, and thigh compartment tension should be assessed. Radiographs of the entire femur should be obtained, as femoral neck fracture occurs in association with approximately 9% of femoral shaft fractures (Fig. 44-8).[149,150] Up to 10% of ipsilateral femoral neck fractures in patients with femoral shaft fractures were not identified during the initial assessment and were found after shaft fixation.[151] Although several historical classification systems exist, they are not often used in daily practice. Femoral shaft fractures are typically described by location (proximal one-third, mid-shaft, distal one-third) and pattern (transverse, short/long oblique, segmental, comminuted).

Both femoral shaft and subtrochanteric fractures can be initially treated with skeletal traction prior to definitive fixation if prompt surgical fixation is not possible. Traction can minimize fracture-related blood loss by decreasing the

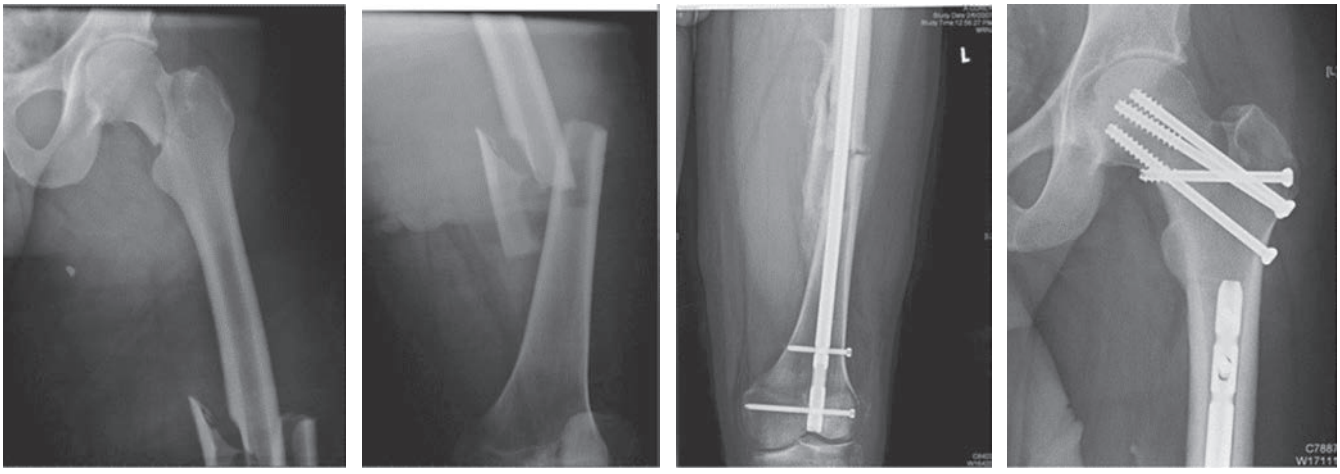


FIGURE 44-8 (**A** and **B**) Injury radiographs of a femoral shaft fracture with an ipsilateral femoral neck fracture. (**C** and **D**) Postoperative radiographs following intramedullary nail fixation of the shaft fracture and cancellous screw fixation of the femoral neck. The femoral neck fracture in this injury is often missed. Close inspection of the hip on injury radiographs, the trauma pelvis computed tomography scan, and intraoperative fluoroscopy can prevent missed injuries.

potential third space and lessen pain. Traction for femur fractures is typically applied through a pin in the proximal tibia to avoid contaminating the distal femur site of future fixation. Early (<24 hours) femur fixation is beneficial in appropriately resuscitated patients without lung and head injuries.[10,12,14,77,82,83] Patients who are too unstable to undergo definitive treatment typically benefit from placement of an external fixator. External fixators can typically be placed in less than 30 minutes, with minimal blood loss and without special positioning. External fixator placement minimizes fracture motion, decreases pain and fat emboli, and allows improved patient mobilization and pulmonary toilet.

The vast majority of femoral shaft and subtrochanteric femur fractures are treated with a reamed, statically interlocked, intramedullary nail.[152] This procedure is associated with high union rates (>95%), low complication rates, and immediate weight bearing. Intramedullary reaming permits use of a larger-diameter nail to provide sufficient strength and durability, minimizing the risk of failure. Awareness of extravasation of reaming debris such as fat, bone marrow fragments, and inflammatory mediators has led to concern that embolization of this debris to the lungs may increase the risk of pulmonary complications.[153] There are conflicting data surrounding the impact of reaming on perioperative pulmonary complications.[154,155] In most circumstances, the clinical benefit of reamed nails is superior to unreamed nails.[156,157] Proximal one-third femur fractures are often treated with an antegrade nail (passage of the nail from the hip to the knee; starting point on the greater trochanter or piriformis fossa) and distal one-third femur fractures are typically treated with a retrograde nail (passage of the nail from the knee to the hip). However, many proximal one-third fractures can be successfully treated with a retrograde nail, including polytrauma patients who benefit from supine positioning. Mid-shaft femur fractures can be treated with either technique. Antegrade nailing is associated with a slightly higher rate of postoperative hip pain, and retrograde nailing is associated with a slightly higher rate of postoperative knee pain.[158]

Subtrochanteric fractures represent challenging injuries as muscle forces on the proximal fragment (flexion, abduction, external rotation) often lead to widely displaced fractures that are difficult to properly align. Intramedually nailing remains the optimal treatment of subtrochanteric fractures; however, open reduction is often necessary to obtain adequate alignment and promote fracture healing. Use of a sliding hip screw is not recommended due to a high rate of loss of fixation and early failure.[159] Multiple recent reports have demonstrated a high failure rate with the use of fixed-angle locking plates in the treatment of peritrochanteric femur fractures.[160]

Atypical subtrochanteric femur fractures may occur in patients who have been taking bisphosphonate medication for the treatment of osteoporosis for several years. The mechanism of action of bisphosphonates interferes with normal bone turnover. Subsequently, the ability of bone to repair itself is altered. Bisphosphonate fractures occur with low-energy or even no trauma, often with prodromal pain. Hallmarks of an atypical femur fracture include a short oblique or transverse fracture pattern, thickening or beaking of the lateral cortex, and a medial spike (Fig. 44-9).[161,162] Radiographs of the contralateral femur should be taken, as these fractures often occur bilaterally. Patients with these fractures should have their bisphosphonates stopped, and medical reevaluation of their osteoporosis is warranted.

Fractures of the Distal Femur

Fractures of the distal femur have historically been a significant treatment challenge because it can be difficult to obtain adequate fixation in the short residual bone segments, especially in osteoporotic patients. Distal femur fractures account for approximately 7% of all femur fractures.[163] Similar to hip fractures, distal femur fractures are mostly seen in two patient populations: young patients sustaining high-energy trauma and low-energy injuries in the elderly. There is a range of injury patterns that vary from complex intra-articular injuries to simple transverse or short oblique extra-articular fractures. Historically, weight bearing was limited following fixation; however, recent reports support immediate weight-bearing protocols in elderly patients to facilitate early mobilization without an increase in fixation failure or impairment of healing.[164]

The most common mechanism for a distal femur fracture is a dashboard-type injury with direct trauma to the flexed knee. These injuries are associated with concomitant acetabular fractures, hip dislocations, and knee injuries such as patella fractures. Because of the close proximity of the tethered femoral artery in the adductor hiatus, vascular injury can occur. Vascular studies are indicated if abnormal pulses are detected or if the ABI is less than 0.9. Radiographic evaluation should include standard AP and lateral films of knee and femur. CT scan is obtained if there is concern for intra-articular extension. The prevalence of a coronal plane fracture of the femoral condyle (Hoffa fracture) has been reported to be as high as 38% (Fig. 44-10).[165] Lateral condyle fractures are more common than medial condyle fractures and can be associated with late displacement and poor functional outcomes if left untreated.[165]

Most patients initially have a knee immobilizer applied for pain control in anticipation of surgery. Similar to femoral shaft fractures and hip fractures, patients benefit from early fracture stabilization and mobilization. In patients with significant soft tissue injuries or polytraumatized patients requiring prolonged resuscitation, a temporary knee spanning external fixator may be applied to maintain length and stability until definitive fixation is appropriate. The primary aim of distal femur fracture treatment is anatomic reconstruction of the articular segment, followed by restoration of appropriate length, alignment, and rotation of the femur. Failure to achieve appropriate articular reduction and alignment may result in an abnormal gait, pain, posttraumatic arthritis, and a poor functional outcome. Treatment options include ORIF with plate fixation and intramedullary nailing.[166] Complex total knee arthroplasty may be warranted in rare circumstances.[167] Elderly periprosthetic distal femur fractures have

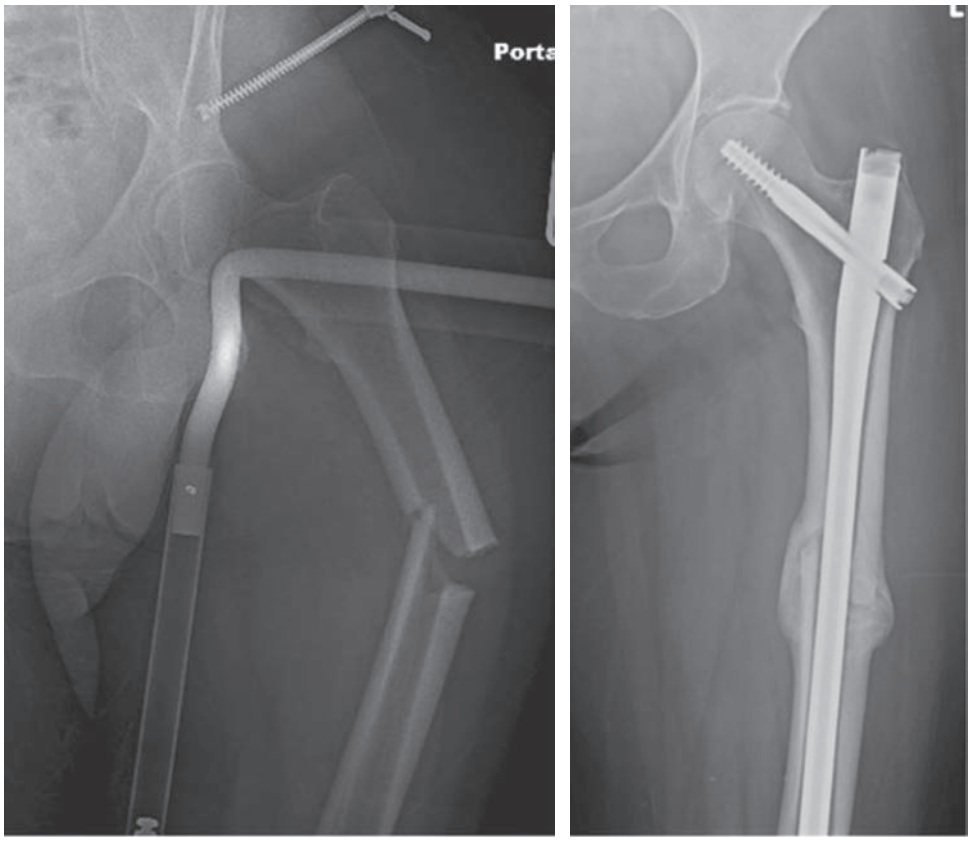


FIGURE 44-9 Atypical femur fracture. (**A**) Anteroposterior left hip radiograph of an atypical subtrochanteric femur fracture often associated with long-term bisphosphonate use. Note the thickened lateral cortex, lateral beak, transverse lateral cortex fracture line, and oblique medial cortex fracture line. (**B**) Six-month postoperative radiograph of the healed fracture after treatment with a cephalomedullary nail.

increased in incidence over the past decade. Most surgeons advocate immediate weight bearing in this population after fracture stabilization with a plate or nail. Patients with fractures that extend into the articular surface typically have their weight bearing limited for 6 to 12 weeks to avoid loss of reduction. Distal femur fractures have historically been characterized by high rates of malunion, nonunion, infection, and hardware failure.[168] Recent advances in surgical technique, coupled with implant designs that are angularly stable, have resulted in improved outcomes.[169]

Patella Fractures and Injuries to the Extensor Mechanism

The patella is the largest sesamoid bone in the body and is encased within the extensor mechanism of the knee. The extensor mechanism allows the quadriceps muscles to extend the knee through pull on the proximal tibia. Patella fractures are clinically significant if they disrupt this mechanism. Quadriceps and patellar tendon ruptures have a similar clinical picture and functional deficit. The patella is stabilized within the trochlear groove of the distal femur by the medial and lateral portions of the quadriceps, as well as retinacular tissue that connects the quadriceps to the joint capsule.

Standard imaging in the setting of suspected extensor mechanism disruption includes AP and lateral knee radiographs. Advanced imaging is rarely indicated in the acute setting, although magnetic resonance imaging (MRI) or ultrasound can be used for diagnostically challenging cases. Patella fractures and extensor mechanism disruptions are diagnosed by history and physical examination. Patients will frequently present with knee pain and history of a direct blow to the knee or popping sensation. On physical examination, pain with palpation of the patella, a knee effusion, and the inability to actively extend the knee are clues that the extensor mechanism may be injured. Some patients might retain some extension in the setting of a patella fracture if the surrounding retinaculum is not injured. A palpable defect distal to the patella and patella alta (high-riding patella) suggest a patellar tendon disruption. Conversely, a palpable defect proximal to the patella and patella baja (low-riding patella) suggests quadriceps tendon rupture.

Patellar fracture management depends on the fracture displacement. Nonoperative treatment is reserved for patients with minimally displaced patellar fractures who are able to

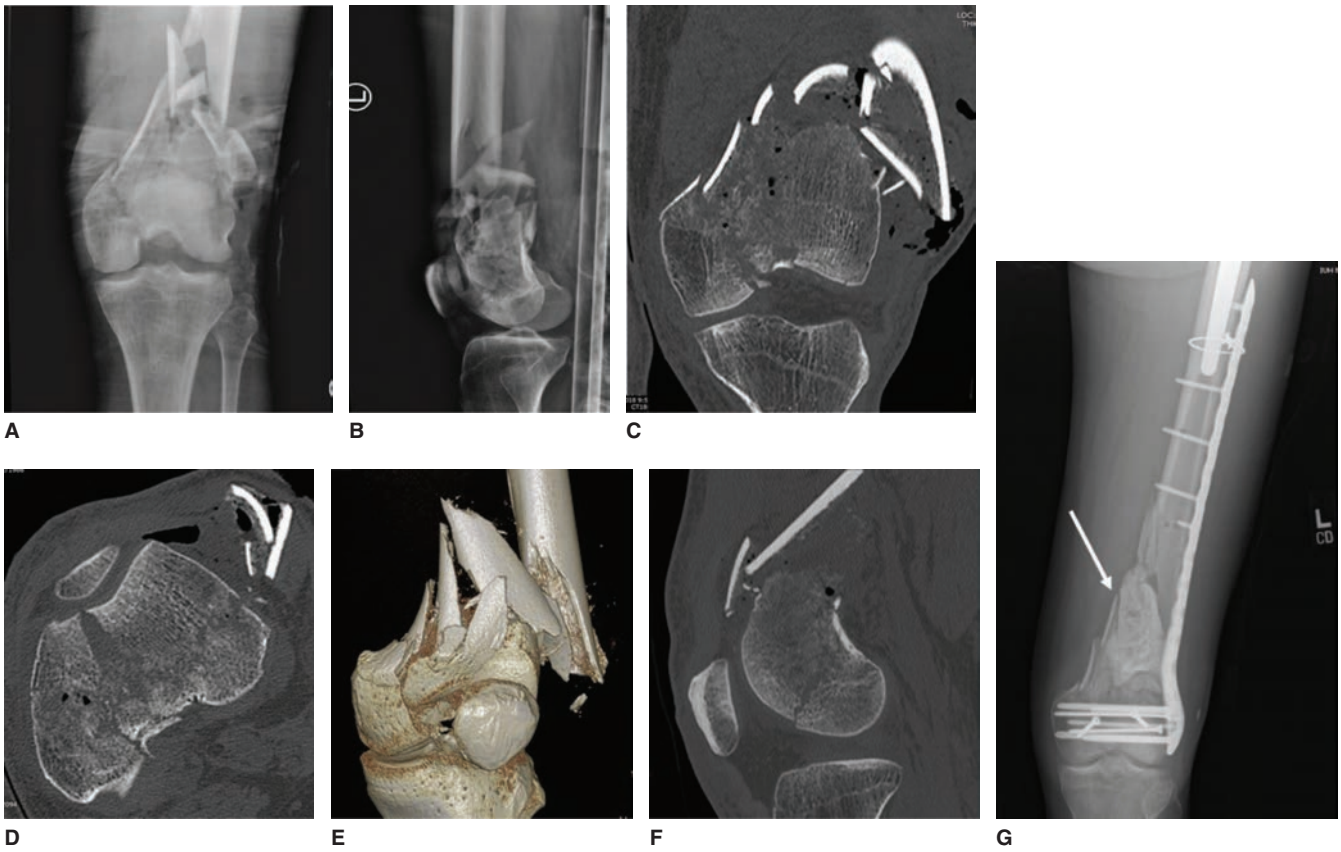


FIGURE 44-10 Complex, open intra-articular distal femur fracture with a Hoffa fragment. (**A**) Anteroposterior (AP) and (**B**) lateral knee injury radiographs. (**C**) Coronal computed tomography (CT), (**D**) axial CT, and (**E**) three-dimensional CT reconstruction. The Hoffa fracture of the lateral condyle can easily be missed on plain radiographs but is demonstrated on the (**F**) sagittal CT. (**G**) AP radiograph after open reduction and internal fixation of the distal femur fracture, also demonstrating an antibiotic-laden cement spacer in the metaphysis of the femur (arrow).

extend their leg against gravity. Nonoperative treatment consists of bracing or casting the knee in extension for 4 to 6 weeks. These patients are allowed to bear weight with the knee fully extended to protect the patella against tension load. Most patients with displaced patellar fractures, full-thickness patellar tendon ruptures, or full-thickness quadriceps tendon ruptures are treated surgically. The method of patellar fixation depends on the fracture pattern, location, and degree of comminution. Operative interventions include wiring, screw-based fixation, plating, partial patellectomy, and rarely total patellectomy. Tendon ruptures are repaired with heavy, braided sutures woven through the tendon and passed to the opposite side of the patella through bone tunnels. Chronic tendon tears typically require tendon lengthening and more complex reconstructions in order to overcome the shortening from quadriceps retraction. These injuries can be missed in polytraumatized patients who are unable to participate in a reliable physical exam. Extensor mechanism injuries typically require prolonged rehabilitation to achieve a functional range of knee motion and regain strength. It is not uncommon for patients with patella fractures that require operative treatments to have persistent pain and functional deficits.[170]

Knee Dislocation

Knee dislocations are severe injuries that involve multiple ligament disruptions of the knee. Knee dislocations are typically caused by high-energy blunt trauma such as a direct blow to a dashboard. Low-energy mechanisms can also lead to dislocation, typically in morbidly obese patients (females > males), which can occur after a seemingly trivial injury such as a ground-level fall.[171] A high index of suspicion is needed when a patient describes a "pop" or "shift" in their knee because knee dislocations can spontaneously reduce and have a normal-appearing radiograph. The knee is completely dependent on ligaments for stability. Ligamentous injuries can avulse fragments of bone seen on radiographs, which can be a sign of a dislocation. Examples include a posterior cruciate ligament (PCL) avulsion and a lateral capsular avulsion (Segond fracture) commonly seen with anterior cruciate ligament (ACL) injuries. MRI is indicated to precisely define the ligaments that are injured and determine the timing of treatment. Physical exam of the knee will demonstrate joint line tenderness and abnormal laxity compared to the contralateral knee. The Lachman test and anterior drawer test are performed to diagnose ACL injury. The posterior drawer test and posterior

sag sign detect PCL injury. Varus and valgus stress tests with the knee flexed 30° evaluate for lateral collateral ligament and medial collateral ligament injury, respectively. A positive Dial test (increased external rotation of the foot with the knee at 30°) is indicative of a posterolateral corner injury.

The proximity of the important neurovascular structures around the knee makes knee dislocations a potentially limb-threatening injury. The early recognition of an associated popliteal artery injury, which has been described in up to one-third of cases with traumatic knee dislocations, is crucial.[172] While a complete arterial disruption may be obvious early after trauma due to clinical signs of peripheral ischemia, an incomplete dissection or intimal injury by stretching forces may be missed. Intimal tears can lead to delayed thrombosis and secondary limb ischemia despite the absence of apparent early clinical evidence for a vascular injury. Because of the often-asymptomatic nature of these popliteal injuries, the amputation rate for blunt vascular trauma is about three times higher than that after penetrating injuries and lies in the range of 15% to 20%.[173] Thus, a high index of suspicion and defined diagnostic algorithms should help establish an accurate diagnosis. ABI should be measured and documented on all knee dislocations. ABI less than 0.9 and/or asymmetric pulses should prompt angiographic evaluation. Based on a large meta-analysis, the accuracy of pulse examination alone is very low, yielding a sensitivity of only about 79% for the detection of an arterial injury.[174] The hard and soft signs for an arterial injury, which are present in about two-thirds of all cases, are outlined in Table 44-1. Injury to the peroneal nerve with a resultant foot drop and impaired dorsal foot sensation is common. Less commonly, tibial nerve injury can occur, leading to absence of foot and toe plantarflexion and plantar foot numbness. This can interfere with recognition of ischemic pain due to arterial occlusion or an acute compartment syndrome. Serial examination and clear documentation and communication between team members are essential when treating these injuries. A popliteal artery injury associated with dislocation of the knee is often addressed in the operating room with both vascular and orthopedic surgeons. Adequate reduction and stabilization of the knee dislocation are required. External fixation is well suited for provisional stabilization because it can be applied rapidly without delaying arterial repair but can also readily be adjusted to allow intraoperative positioning of the knee, facilitating repair. Four-compartment prophylactic fasciotomy is advisable after popliteal artery repair in order to avoid compartment syndrome secondary to ischemia-reperfusion.

Initial management of knee dislocations is based on the knee stability after reduction. A knee dislocation without vascular injury that is concentrically reduced on postreduction radiographs may be treated in a splint or knee brace until definitive knee reconstruction. Foam knee immobilizers provide insufficient support to maintain reduction if there is concern for recurrent instability. Open dislocations, those with vascular injury, and reductions that cannot be maintained should be treated in a joint-spanning external fixator.[175] Definitive ligament reconstruction is typically performed 1 to 6 weeks after injury, depending on the ligaments involved. Outcomes of knee dislocation are more dependent on associated injuries rather than the timing of reconstruction.[176] Numerous protocols exist regarding staging of surgical interventions and selection of ligament to treat. In general, improved outcomes have been demonstrated with staged ligament reconstruction as opposed to acute ligamentous repair. Arthroscopy is avoided within the first 10 to 14 days of injury because fluid extravasation will likely occur due to the disrupted knee capsule and could potentially cause compartment syndrome.

Knee dislocations are devastating injuries, especially to athletes, because few return to the same level of performance. Common complications include knee stiffness (very common), instability (less common with appropriate treatment), and posttraumatic arthritis. Rehabilitation protocols are very patient and surgeon specific.

Tibial Plateau Fractures

Tibial plateau fractures are fractures involving the articular surface and the proximal 7 to 9 cm of the tibial metaphysis. Fracture pattern varies depending on bone quality, the magnitude and direction of force, and the degree of knee flexion at the moment of impact. Coronal plane forces, such as varus and valgus stress, coupled with axial compressive force, create medial and lateral fracture patterns, respectively. Young patients with good bone quality are more likely to sustain split or split-depression fracture patterns with associated ligamentous injury. Older patients with osteopenia are more likely to sustain a pure depression or split-depression fracture (Fig. 44-11). The examiner should carefully evaluate the neurovascular status and soft tissue conditions including swelling because the rate of compartment syndrome in bicondylar tibial plateau fractures approaches 20% and skin breaches may represent an open fracture. Open wounds in this region can also result in traumatic knee arthrotomy, even in the absence of proximal tibia fracture. A saline retention test with at least 150 mL injected into the knee away from the wound can be performed. Leakage of fluid from the open wound confirms diagnosis of an open joint injury.[177] Presence of intra-articular air in CT was found to be highly sensitive and specific for traumatic knee arthrotomy and may replace conventional saline retention test.[178] Presence of compartment syndrome must be considered, especially in high-energy injuries with comminuted fractures, and patients should be evaluated frequently as swelling evolves.

AP and lateral radiographs of the knee should be obtained to evaluate knee pain after injury. Radiographs should be inspected for articular depression, widening of the tibial plateau, metaphyseal fracture, and bony avulsions. CT scan with sagittal and coronal (with or without three-dimensional) reconstruction provides detailed information of the articular injury and aids in preoperative planning. Lower energy injuries typically lead to injury on one side of the joint, whereas higher energy injuries often result in complete injury to both sides of the joint. The AO/OTA classification and Schatzker classification are commonly referred to in daily practice when

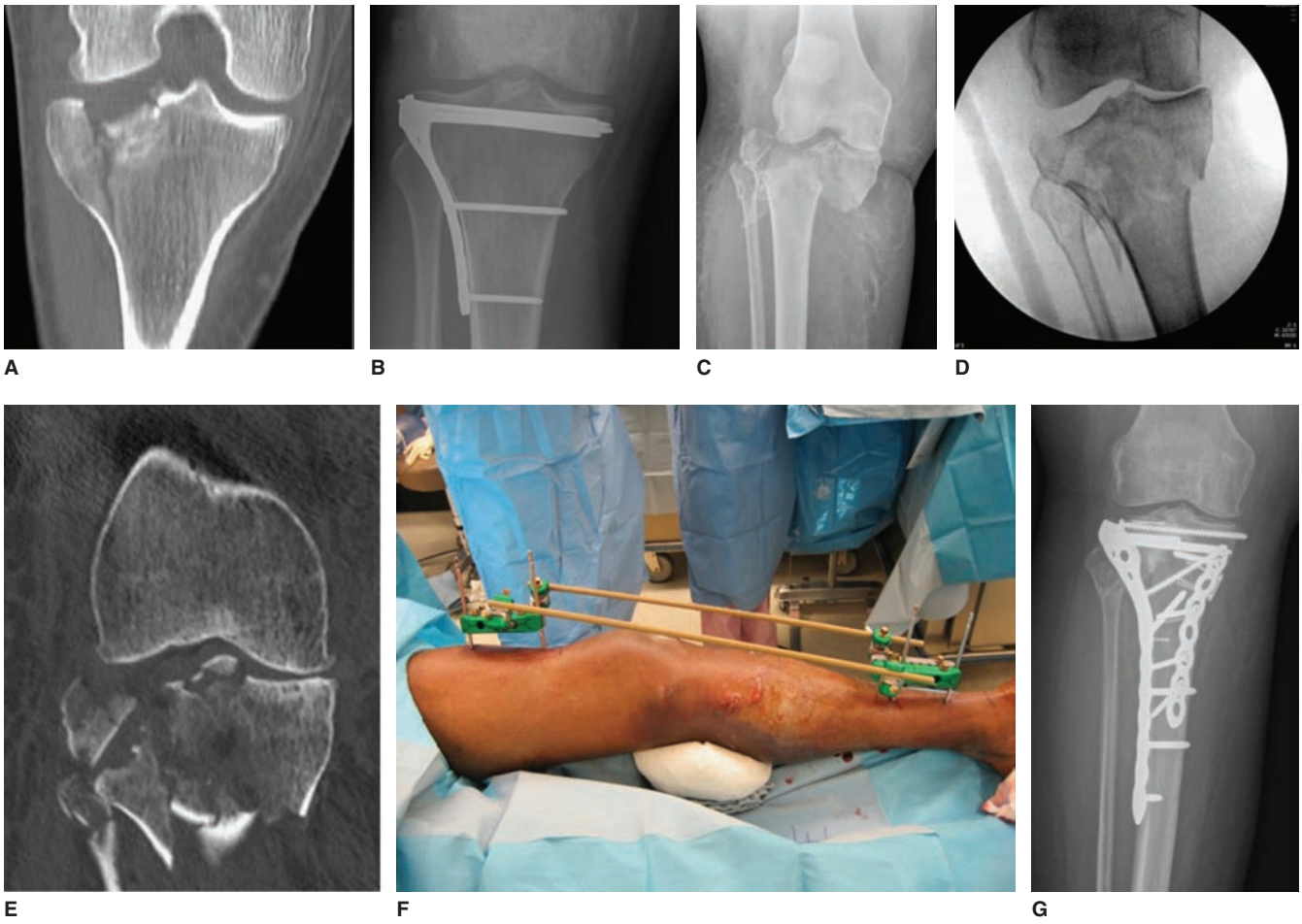


FIGURE 44-11 (**A**) Coronal plane computed tomography scan of a unicondylar tibial plateau fracture and (**B**) AP knee radiograph following open reduction and internal fixation (ORIF). (**C–G**) High-energy, comminuted bicondylar tibial plateau fracture as seen on (**C**) injury AP knee radiograph; (**D**) post–external fixation AP fluoroscopic image; (**E**) coronal computed tomography; (**F**) clinical image demonstrating knee-spanning external fixator and fracture blisters; and (**G**) postoperative AP knee radiograph following ORIF with a dual-incision approach.

formulating treatment plans.[136] Particular attention should be paid to medial tibial plateau fractures (Schatzker IV), which can be associated with a knee dislocation (Fig. 44-12). The stable fragment in these injuries is not the tibial shaft but rather is the portion of the medial tibial plateau that is still attached to the femur by its ligamentous attachments. The remainder of the tibia is dislocated from the femoral articulation. Vigilance in these fractures is critical, and vascular evaluation should be undertaken as described earlier.

Nondisplaced and minimally displaced fractures can be treated with early motion and a period of non–weight bearing in a hinged knee brace for 6 to 12 weeks. Indications for surgical management include open fracture, compartment syndrome, articular displacement, and knee instability. Timing of surgery is dependent on the soft tissue condition. Definitive surgery is delayed if severe swelling or blistering is present to minimize wound complications. Bicondylar tibial plateau fractures with poor soft tissue conditions are typically initially managed with external fixation to restore length and alignment. Historically, most bicondylar tibial plateau fractures were treated in staged fashion; however, recent evidence has shown that many injuries can be treated earlier in the absence of severe soft tissue injury.[179]

Historically, bicondylar plateau fractures were associated with a high rate of infectious and wound complications. More biologically friendly techniques including dual incisions, percutaneous instrumentation, and staged treatment protocols have significantly reduced wound and infectious complication rates from 50% to less than 10%.[180] Most tibial plateau fractures are stabilized by ORIF with plate and screw fixation. Locking screw technology has improved fixation in select patients with comminuted fractures or osteoporotic bone, but universal use of locking implants is not necessary.[181] Select metaphyseal and simple intra-articular plateau fractures are amenable to intramedullary nailing. Although technically challenging, one study demonstrated effective treatment of tibial plateau fractures with ring fixators.[182] At the time of definitive treatment, assessment for other soft tissue injuries of the knee should occur because they are associated with a high rate of meniscal tears and ligament disruptions.[183]

Despite the improved treatment methods detailed earlier, bicondylar plateau fractures have been recently reported to

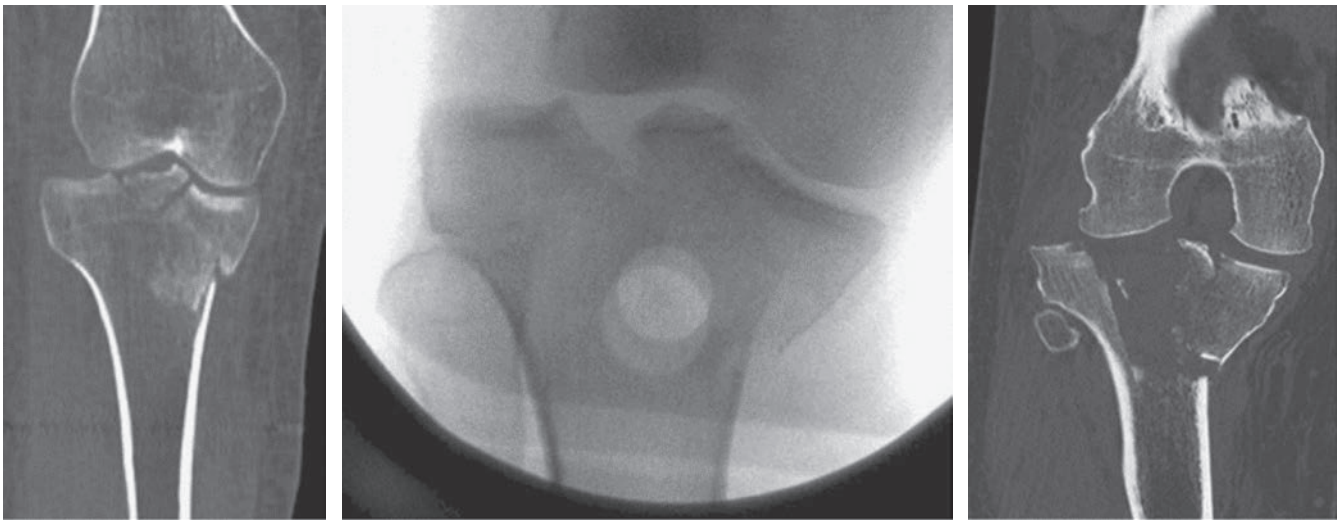


FIGURE 44-12 Examples of Schatzker IV medial tibial plateau fractures. (**A**) Anteroposterior (AP) knee radiograph, (**B**) AP fluoroscopic stress image, and (**C**) coronal computed tomography scan demonstrating variants of medial tibial plateau fractures with joint subluxation suggesting instability. This injury can behave similar to a knee dislocation, and a high degree of suspicion for vascular injury is required.

have an overall complication rate (including infection and nonunion) of 28%.[184] The long-term risk of posttraumatic arthritis has been reported to be as high as 30%; however, many patients remain asymptomatic, and the long-term conversion rate to total knee arthroplasty is 7%.[185] Some loss of motion and persistent knee pain may occur from joint stiffness. Articular injuries typically require 8 to 12 weeks of restricted weight bearing; however, early passive and active knee range of motion is usually encouraged.

Tibial Shaft Fractures

Fractures of the tibial shaft range from low-energy, indirect torsional injuries to severe high-energy injuries such as motor vehicle collisions that are associated with significant soft tissue damage and a high incidence of acute compartment syndrome. The amount of energy absorbed by the leg is suggested by the radiographic appearance and soft tissue condition. The severity of the soft tissue injury, whether open or closed, is closely associated with the overall outcome of tibial shaft fractures. The soft tissue envelope on the medial border of the tibia is very thin; thus, minor open fractures may have major therapeutic implications for soft tissue coverage of the exposed bone. Additionally, the close proximity of numerous vessels, nerves, and tendons can result in associated injuries that impact outcome.

A thorough examination of peripheral nerve (superficial peroneal, deep peroneal, tibial) function and vascular integrity (dorsalis pedis and posterior tibial pulses) should be performed. Clinical evaluation for compartment syndrome is extremely important because it occurs frequently in tibial shaft fractures.[33,34] Compartment syndrome is especially common if the soft tissues have been crushed or if a period of ischemia has occurred. Diagnosis of compartment syndrome is primarily clinical, and a coordinated effort by all members of the medical team is necessary to prevent delay in treatment. Open fractures are common, and the patient should be administered prophylactic antibiotics as soon as possible in the emergency department.[46,47]

Full-length AP and lateral radiographs of the affected tibia should be obtained. AP, lateral, and oblique views of the ipsilateral knee and ankle should be obtained if the fracture extends proximally and/or distally to assess intra-articular involvement. CT scan may be helpful if there is suspicion of fracture extension into either articular surface. Tibia shaft fractures are described by location (proximal, central, distal), shape (transverse, long/short oblique, spiral, segmental, comminuted), and direction of displacement (varus, valgus, apex anterior/procurvatum, apex posterior/recurvatum).

Timing and treatment modalities for tibial shaft fractures are dependent on the severity of injury, associated injuries, comorbidities, and the patient's physiologic condition. Limb-threatening injuries such as arterial disruption with ischemia and compartment syndrome require immediate surgery. As discussed earlier, open fractures should be treated in expeditious fashion by an experienced team because quality of surgical debridement is thought to be more important than timing to debridement as long as debridement occurs within 24 hours of injury.[6]

External fixation is typically indicated as the initial treatment of high-energy open tibia fractures with severe soft tissue injury, of vascular injury requiring repair, and in the setting of unstable polytrauma patients as a damage control procedure.[4] Many open fractures of the tibia require multistage treatment for decontamination, assessment of residual tissue perfusion, fracture repair, and soft tissue coverage. The wound bed is debrided and closed if possible during the initial surgery because delayed closure in adequately

debrided wounds increases infection risk.[186] Long-term use of an external fixator (>14 days) is associated with bacterial colonization of the pin tracts and a risk of infection from subsequent intramedullary nailing. Use of an external fixator for only a few days, however, can safely precede intramedullary nailing for definitive management of tibial shaft fractures.

In closed fractures, provisional closed reduction and application of a long leg splint provide initial immobilization. Historically, the majority of tibial shaft fractures were definitively treated by closed means with reduction and casting.[187] However, with advancements in surgical fixation, especially intramedullary reamed nailing, surgery is indicated in most cases. Intramedullary nailing provides excellent fixation and facilitates restoration of length and alignment in diaphyseal fractures. Nails allow immediate motion of the foot and ankle and permit early weight bearing. Reaming of the tibia medullary canal has a beneficial effect on fracture union.[188,189] The indications for intramedullary nailing have expanded to more proximal and distal metaphyseal fractures as new-generation interlocking nails provide multiplanar interlocking options and far proximal/distal opportunities for screws.[190] Plate fixation of acute fractures of the tibial shaft is generally reserved for periarticular injuries too proximal or distal for intramedullary nailing.[191] Alternatively, tibial shaft fractures with soft tissue compromise can be definitively managed with a ring fixator. Strategies to reconstruct tibial bone loss include secondary Masquelet bone grafting and distraction osteogenesis (via bone transport) with ring fixators.[192]

Knee pain after intramedullary nailing of tibia shaft fracture is a common complication and may persist for a long period of time.[193] Infection and nonunion occur in up to 10% of patients with closed fractures and 25% to 30% of open fractures.[189,194] Managing modifiable risk factors such as cessation of tobacco abuse and strict glucose control in diabetes is recommended in high-risk patients.

Distal Tibial Fractures

Mechanism of injury categorizes distal tibia fractures into two categories. An axial loading force on the tibial articular surface leads to a pilon fracture, whereas a rotational injury typically leads to an ankle fracture.

Ankle injuries represent the most common musculoskeletal injury. The ankle is a hinge joint, in which the body of the talus dorsiflexes and plantarflexes within a mortise-like socket formed by the medial malleolus, plafond of the distal tibia, and the distal fibula (lateral malleolus). Integrity of the mortise is maintained by the lateral ligaments, medial (deltoid) ligaments, and syndesmotic ligaments. The ankle syndesmosis is the joint between the distal tibia and the distal fibula immediately above the ankle joint, which is stabilized by its own ligamentous complex. Widening of this mortise is indicative of syndesmosis disruption and can result in talar instability and predisposition to posttraumatic arthritis. The Lauge-Hansen classification system has historically described four groups of rotational ankle injuries based on the position of the foot and direction of force applied at the moment of injury.[195] Pilon fractures, in contrast, result from a high-energy axial load to the ankle. Pilon fractures are classically described by characteristic fragments (anterolateral, posterolateral, central, medial), and the AO/OTA classification system is commonly referenced when discussing the injury.[136] Significant soft tissue damage can accompany pilon fractures, even in closed injuries. They are challenging intra-articular injuries that often carry a poor functional prognosis.[196,197]

Ankle sprains are ligamentous injuries of the ankle most commonly involving the lateral collateral ligament complex, which provide inversion stability of the talus. Inversion of the foot normally occurs at the subtalar joint. If forced to the limit, however, the lateral collateral ligament stretches or ruptures, producing the typical "sprained ankle" with lateral pain, swelling, ecchymosis, and tenderness over the injured ligaments distal and anterior to the lateral malleolus. It is difficult to differentiate a simple sprain from a fracture in the acute setting due to nonspecific symptoms such as pain, tenderness, and swelling. The Ottawa ankle rules can help determine the need for diagnostic imaging in ankle injuries.[198] Ankle radiographs are suggested in the following settings: (1) bony tenderness at the lateral malleolus, (2) bony tenderness at the medial malleolus, or (3) inability to bear weight for four steps. Three views of the ankle, including AP, mortise (15°–20° internal rotation of the foot), and lateral, are usually sufficient to adequately diagnose most ankle fractures. Tibia radiographs should be obtained in ankle fractures to rule out an unstable Maisonneuve fracture, defined as a spiral fracture of proximal fibula associated with medial malleolus or deep deltoid ligament injury and disruption of the syndesmosis or interosseous membrane. CT is typically obtained in pilon injures. If staged treatment is planned, CT is optimally obtained after ankle-spanning external fixation is placed. This not only improves fracture fragment visualization, but also minimizes radiation to the patient.

Minor ankle sprains can be treated with restricted activities, elevation, ice, and support as needed for comfort. More severe sprains require immobilization. After a brief period of rest, most ligamentous injuries are effectively treated with a functional brace. Ankle fractures should be examined promptly in the emergency department because prolonged deformity can lead to soft tissue injury and blistering. Timely closed reduction and splinting are optimal, especially in fracture-dislocations, to reduce the risk of skin necrosis secondary to osseous displacement. Postreduction radiographs and surgical consultation should be obtained to confirm adequate reduction. Surgical treatment of ankle fractures can occur any time within 10 to 14 days, with fixation deferred in severely swollen ankles.

The lateral malleolus is the prime determinant of talar alignment. Restoration of its proper relation with the distal tibia is the key to treating malleolar fractures. Lateral malleolus fractures can be distal to the joint, at the joint, or proximal to the joint. Isolated, minimally displaced lateral malleolus fracture below the joint can be managed conservatively in a

walking cast or a boot with immediate weight bearing. If the fracture is at or above the joint, stress exam may be helpful to determine a medial deltoid injury. Several authors have determined that a widened medial clear space of more than 4 to 5 mm under stress exam represents an indication for surgical ankle fracture fixation.[199] Patients who require syndesmotic fixation have significantly worse long-term outcomes than patients treated for ankle fractures without syndesmotic injury.[200] Isolated, minimally displaced medial malleolar fractures can be treated nonoperatively. However, displaced medial malleolar fracture should be reduced and surgically stabilized when part of a bimalleolar or trimalleolar fracture pattern (Fig. 44-13). The deltoid ligament does not need to be repaired if the remainder of the joint is reduced and stable. The posterior lip of the tibial plafond, known as posterior malleolus or Volkmann's fragment, is frequently fractured in an ankle fracture-dislocation. The designation of a trimalleolar fracture implies lateral, medial, and posterior fractures or fracture equivalent ligament injuries. Although it is controversial, large posterior tibial plafond injuries that involve more than one-fifth of the articular surface should be reduced and fixed to avoid posterior subluxation of the talus and incongruity of the joint.[201] Weight bearing is variable after ankle fractures, ranging from early weight bearing after suture removal in some rotational injuries to limited weight bearing for 2 to 3 months for syndesmotic and pilon injuries.

Historically, traditional pilon ORIF techniques carried a high risk of wound dehiscence and infection, particularly

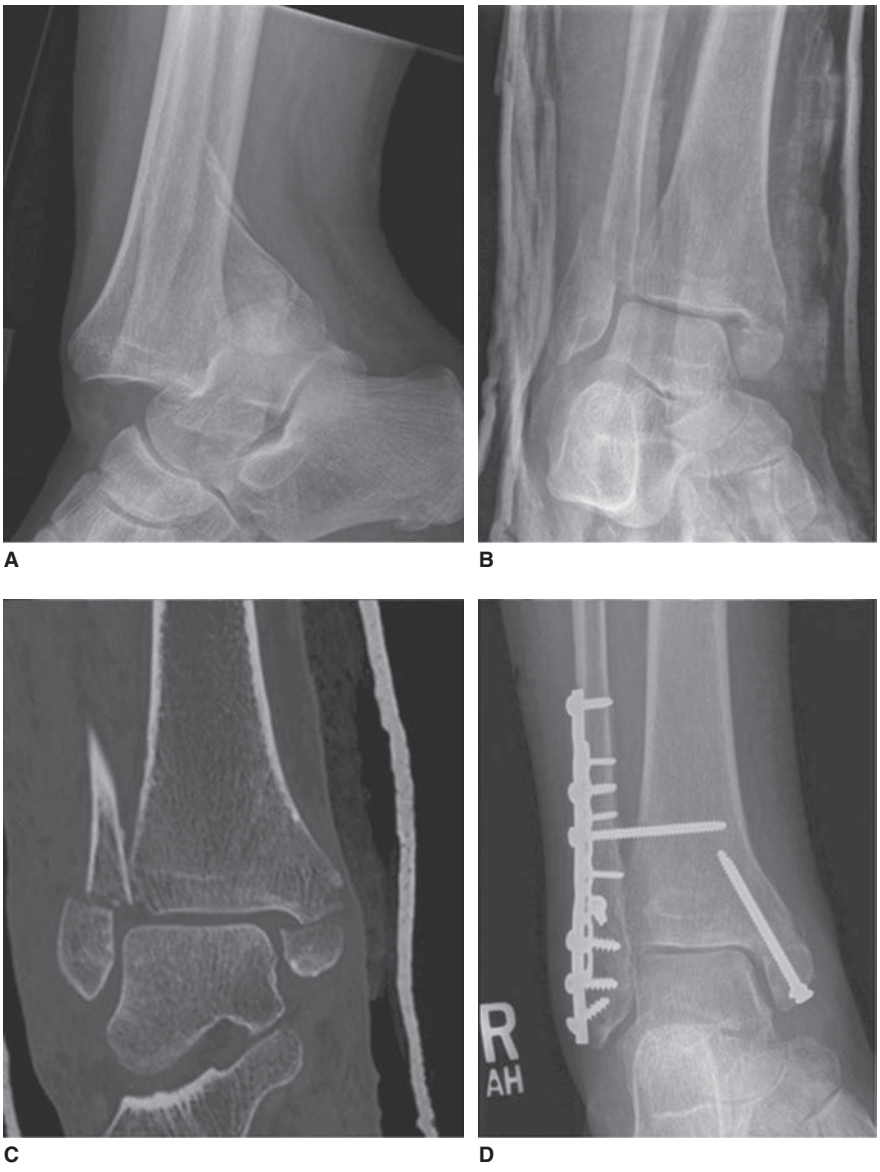


FIGURE 44-13 (**A**) Lateral ankle radiograph of a trimalleolar ankle fracture-dislocation. (**B**) Postreduction anteroposterior (AP) ankle radiograph and (**C**) coronal computed tomography scan. (**D**) Postoperative AP ankle radiograph of the trimalleolar ankle fracture-dislocation and syndesmotic injury treated with open reduction and internal fixation.

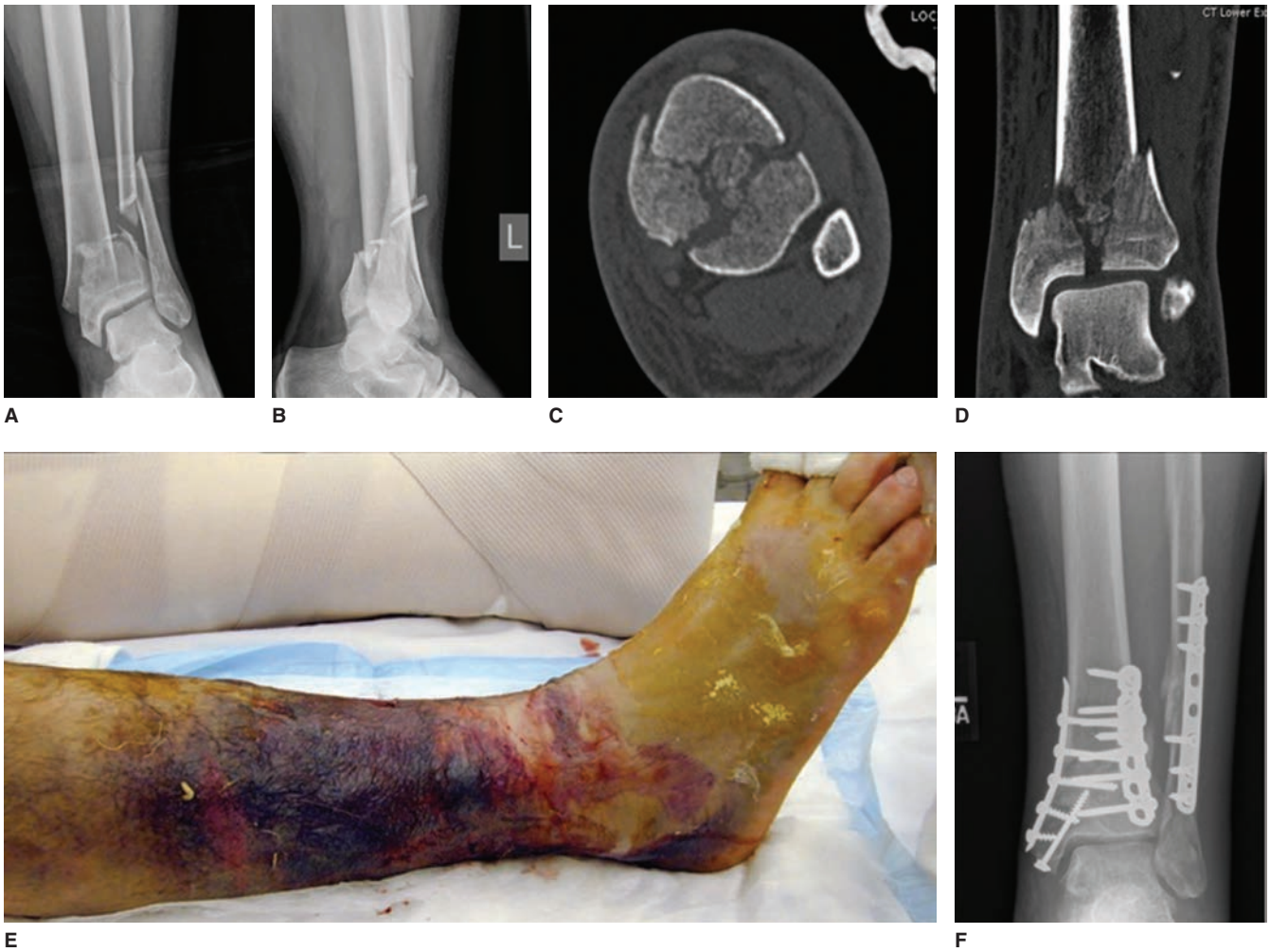


FIGURE 44-14 (**A**) Anteroposterior (AP) and (**B**) lateral ankle radiographs of a comminuted intra-articular pilon fracture with corresponding (**C**) axial computed tomography (CT) and (**D**) coronal CT scan. (**E**) Clinical image demonstrates the severe soft tissue injury that often accompanies this injury. (**F**) Postoperative AP ankle radiograph following pilon open reduction and internal fixation.

if early surgery was performed in the presence of significant soft tissue swelling or blistering.[202,203] Clinical studies have demonstrated an improved outcome of tibial pilon fractures with two-staged approach, typically early external fixation and staged ORIF once the soft tissue condition improved (Fig. 44-14).[204] Recent research has suggested that these fractures can be treated in most circumstances with early fixation without increased risk of complications.[205] Depending on the degree of comminution, bone quality, and extent of soft tissue compromise, the postoperative rehabilitation of pilon injuries may vary. Weight bearing is typically restricted for 10 to 12 weeks.

Outcomes of distal tibia fractures significantly vary depending on the severity and type of fracture. In general, fractures with significant articular involvement have a high incidence of persistent pain, poor function, and symptomatic posttraumatic arthritis.[196,197] Wound and infectious complications can have dire consequences and can potentially lead to loss of limb.

Calcaneus Fractures

The calcaneus is the most commonly fractured tarsal bone. Fracture is often the result of an axial load such as a fall from height. The calcaneus is the largest tarsal bone and is responsible for transmission of body weight to the ground during the heel strike phase of gait cycle. It has a complex shape that includes three subtalar joint facets (posterior facet, middle facet, and anterior facet) that articulate with the talus. The Achilles tendon attaches to the calcaneal tuberosity, forming a strong lever arm for the muscles of the calf.

A detailed history and physical exam are essential in decision making because age, gender, smoking status, worker's compensation injury, and comorbidities including diabetes and peripheral vascular disease play a critical role in outcome.[206,207] Concomitant injuries are common, with 7% to 15% of patients having associated spine fractures.[208] A spine exam should be thorough, with a low threshold for imaging, particularly of the lumbar spine. Calcaneus fractures

require careful soft tissue evaluation and should be initially immobilized in a well-padded splint with the foot in a neutral position. Open fractures of the calcaneus have a high rate of deep infection and are at risk of subsequent complications including amputation.[209] Fractures of the calcaneus can be associated with the development of compartment syndrome of the foot, particularly when the injury is due to a crushing mechanism. Surgical treatment of compartment syndrome of the foot is controversial because fasciotomy carries a significant risk of complications similar to sequelae of compartment syndrome.

AP, lateral, and oblique radiographs of the foot, and a Harris heel view of the calcaneus should be obtained for diagnosis. CT scan is usually required for making treatment decisions. The Sanders classification of intra-articular calcaneus fractures is a useful system that helps guide treatment.[210]

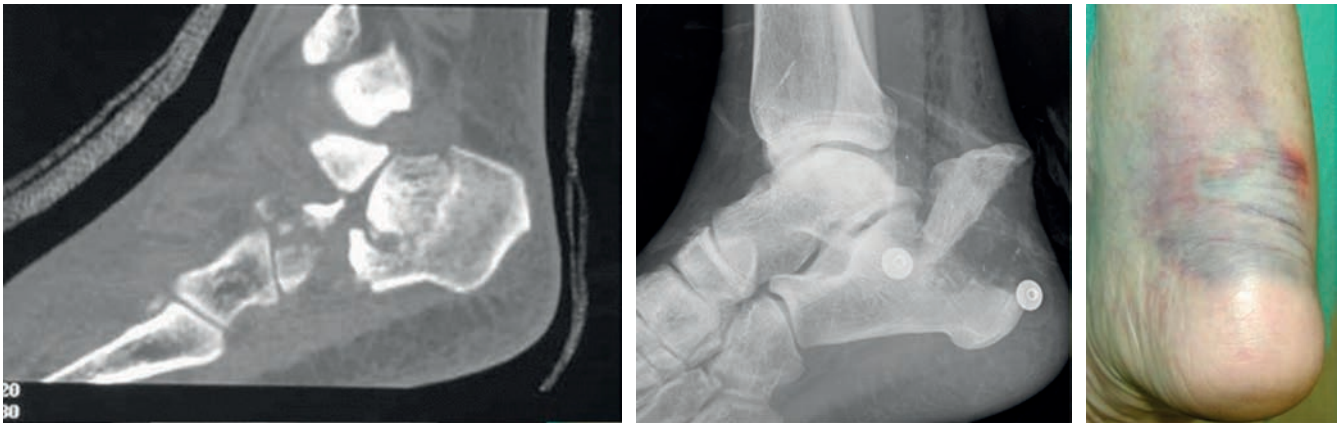
Many nondisplaced and extra-articular fractures of the calcaneus may be treated nonoperatively. Most calcaneal fractures can be treated in delayed fashion; however, the tongue-type variant (Fig. 44-15), which puts pressure on the posterior heel skin, must be reduced urgently to avoid skin loss. One randomized controlled trial comparing operative versus nonoperative treatment of intra-articular displaced calcaneus fractures found surgery to have a beneficial impact on physical function in select groups of patients, including younger patients (<29 years), females, those with comminuted fractures, those with excellent articular reduction (≤2 mm), patients with a lighter workload, and those who were *not* receiving worker's compensation.[206] In another randomized controlled trial with long-term follow-up, surgical treatment was not found to be superior at 1 year but was associated with a decreased rate of posttraumatic arthritis 8 to 12 years postoperatively.[211] Surgical treatment of calcaneus fractures is challenging and can be associated with relatively high complication rates.[209,210]

Historically, the majority of displaced calcaneus fractures were treated 2 to 3 weeks after injury when swelling had significantly subsided. The traditional lateral extensile approach has a risk of skin edge necrosis and wound infection. The blood supply to the skin in this region is tenuous because the angiosome of the heel has a distinctive watershed area at the traditional lateral extensile approach incision.[212] Recently, a limited sinus tarsi (lateral) approach has gained popularity. When compared to the traditional extensile approach, the sinus tarsi approach has been shown to have decreased wound complications.[213] Whether treated operatively or nonoperatively, calcaneus fractures require 6 to 12 weeks of non–weight bearing. Subtalar posttraumatic arthritis can occur after either surgical or nonsurgical treatment. Patients who develop symptomatic arthritis are candidates for fusion of the subtalar joint after failure of conservative treatment measures. Surgeons may elect to perform a subtalar fusion and calcaneus ORIF in the acute setting for comminuted, Sanders type IV intra-articular calcaneus fractures.[214]

Talus Fractures

The talus is the second most common tarsal bone injury after calcaneal fractures. Over 50% of the surface of the talus is articular cartilage. Typical mechanisms of injury involve forced dorsiflexion from falls or motor vehicle accidents. Talar neck fractures are the most common type of talus fractures. Displaced talar neck fractures can be associated with dislocation of the adjacent subtalar, talonavicular, or tibiotalar joints. Ipsilateral lower extremity fractures are common.

AP, oblique, and lateral radiographs of the foot should be obtained. CT is indicated when suspicion is high and aids in preoperative planning through precise determination of displacement and comminution. The Hawkins classification classically described three types of talar neck fractures and associated dislocations, with Canale and Kelly later amending the classification to add a type 4.[215,216] The risk of developing osteonecrosis increases with each type as follows: type 1, talar neck fracture; type 2, fracture with subtalar dislocation;



A B C

FIGURE 44-15 (**A**) Sagittal computed tomography scan of a joint depression-type calcaneus fracture. (**B**) Lateral radiograph of a tongue-type calcaneus fracture and (**C**) corresponding clinical image of the posterior heel demonstrating internal skin pressure due to displaced tongue-type fracture fragment. Tongue-type calcaneus fractures are unique in that they require urgent treatment.

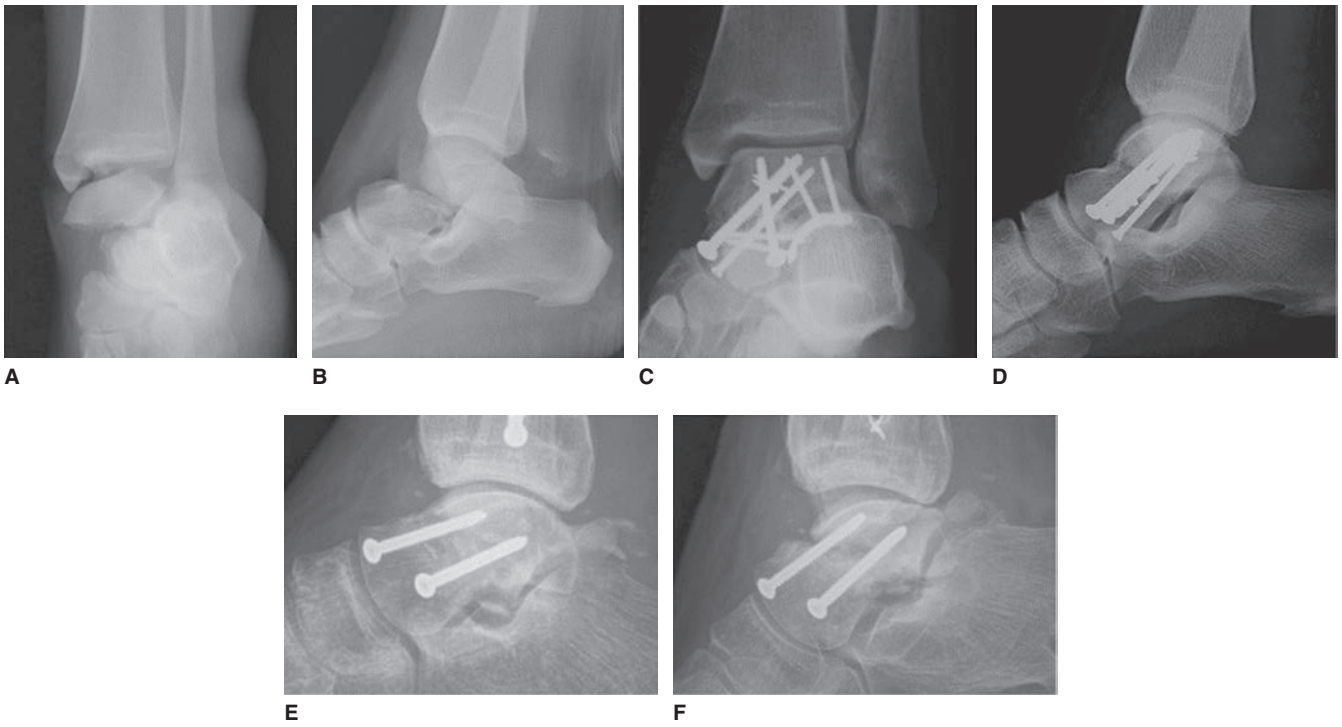


FIGURE 44-16 Talar neck fracture. (**A**) Injury oblique and (**B**) lateral ankle radiographs of a talar neck fracture. (**C** and **D**) Subsequent treatment with open reduction and internal fixation (ORIF). (**E**) Initial and (**F**) late lateral radiographs of a talus treated with ORIF that demonstrated avascular necrosis and collapse on the late radiograph.

type 3, fracture with subtalar and tibiotalar dislocations; and type 4, fracture with subtalar, tibiotalar, and talonavicular dislocations.[215,216]

Blood supply to talus is tenuous because less than half of the talus can be perforated by extraosseous vessels. There are no muscle attachments to the talus, and the blood supply is solely through ligaments and the joint capsule. Disruption of the blood supply to the talus can lead to avascular necrosis with subsequent collapse and posttraumatic arthritis (Fig. 44-16). Therefore, fracture-dislocations of the talus resulting in vascular disruption greatly impact long-term prognosis. Initial fracture displacement and timing of reduction are factors that affect the blood supply. Fracture-dislocations require emergent reduction in the emergency department to potentially restore vascularity. If the fracture cannot be reduced closed, the patient should be taken urgently to the operating room for an open reduction.

Peripheral fractures of the talus include the talar head, body, and lateral process. Unlike talar neck fractures, these fractures usually do not require emergent intervention. These fractures are frequently missed on x-ray; therefore, a CT scan should be obtained in any situation in which there is a high suspicion for a fracture and the radiographs are negative.

Subtalar dislocation is another severe injury of the foot that often involves open wounds, associated fractures of the tarsal bones, and neurologic injury. Urgent reduction is recommended to reduce the risk of permanent vascular insult to the talus and stretch injury to peripheral nerves. The reduction maneuver involves knee flexion, ankle plantarflexion, and either supination or pronation for lateral and medial dislocations, respectively. A postreduction CT scan is required to confirm a congruent reduction, identify associated fractures, and assess for loose fragments in the joint. Purely ligamentous injuries can be treated conservatively in a cast, but fracture-dislocations often require surgical intervention. Posttraumatic arthritis is the most common long-term complication.

Mid- and Forefoot Fractures

The Lisfranc joint complex refers to five tarsometatarsal (TMT) joint articulations between four proximal tarsal bones (medial cuneiform, middle cuneiform, lateral cuneiform, and cuboid) and the five distal metatarsals. The Lisfranc joint complex is composed of several ligaments: the Lisfranc ligament that runs plantarly from the base of the second metatarsal to the medial cuneiform; plantar TMT ligaments (strongest); dorsal TMT ligaments; and intermetatarsal ligaments (no ligament between first and second metatarsal).

There are variable patterns of dislocations and fracture-dislocations that can involve any or all of the TMT joints. Injury mechanism can be either direct or indirect. Direct injury usually involves a crush injury. Excessive plantarflexion and abduction force are the most common indirect mechanisms. They are occasionally associated with severe soft tissue trauma and compartment syndrome. These injuries present with significant swelling, pain, and plantar ecchymosis of the midfoot.

Radiographic findings may be subtle if only one or two TMT joints are injured. AP, lateral, and oblique radiographs of the foot should be obtained. Standing radiographs are obtained in order to stress the Lisfranc ligament when injury suspicion is high but radiographs are normal. A CT scan of the foot can also be obtained to better visualize avulsion fractures. These injuries require urgent closed reduction of the midfoot when dislocation is present. Residual displacement typically requires surgical fixation (Fig. 44-17). Midfoot arthrosis is the most common complication following these injuries.

Metatarsal fractures and forefoot phalanx fractures are common injuries. Due to the stability provided by intermetatarsal ligaments, the majority of these fractures are treated without surgery. In the setting of multiple metatarsal fractures, surgical treatment may be indicated to restore alignment and length of the forefoot, since intermetatarsal ligaments no longer prevent migration and deformity. Multiple metatarsal

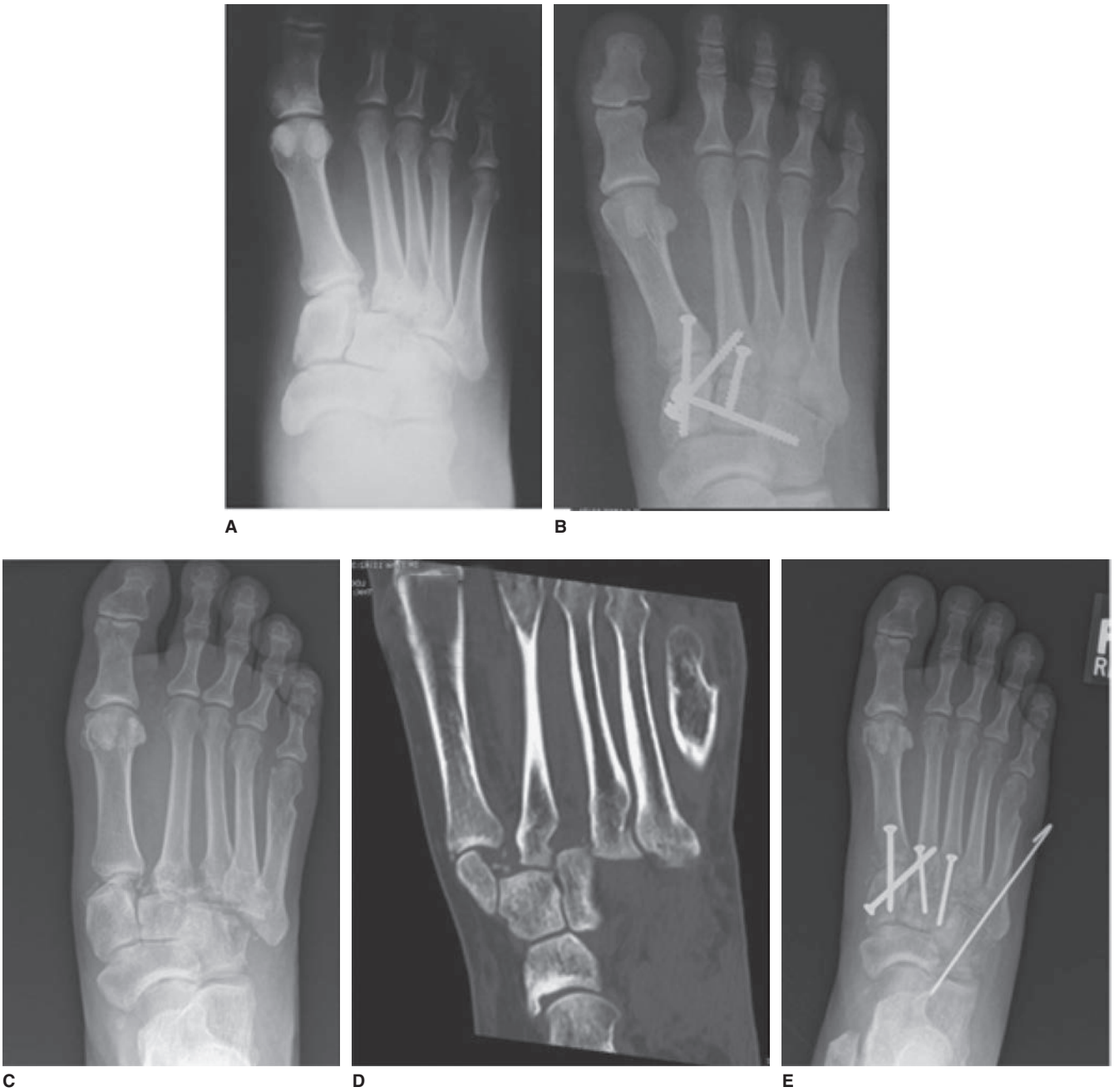


FIGURE 44-17 (**A**) Anteroposterior (AP) foot injury radiograph of a subtle Lisfranc fracture dislocation of the first and second tarsometatarsal (TMT) joints and (**B**) AP foot following Lisfranc open reduction and internal fixation (ORIF). (**C**) AP foot radiograph and (**D**) coronal computed tomography scan demonstrating a significant Lisfranc fracture dislocation of all five TMT joints. (**E**) Postoperative foot radiograph following ORIF of TMTs 1 to 3 and percutaneous treatment of the fifth TMT.

fractures should raise suspicion of a Lisfranc injury. Metatarsal fractures and toe fractures are usually treated with a hard-soled shoe to minimize fracture motion during gait and can usually weight bear as tolerated. An exception is a Jones fracture involving the proximal metadiaphysis of the fifth metatarsal. Nondisplaced Jones fractures can be treated nonoperatively with 6 weeks of immobilization followed by progressive weight bearing, but the nonunion rate has been reported to be as high as 20%. Athletes are usually offered surgical repair to increase the rate of union and promote earlier mobilization.

COMPLICATIONS IN LOWER EXTREMITY TRAUMA

Nonunion

Nonunion occurs when a fracture fails to completely heal. Fractures have different expected healing times depending on the type of fracture and the location. Tibia fractures are relatively slow to heal, particularly when open and highly comminuted.[194] Femur fractures heal more rapidly, due in part to the fact that the femur is circumferentially surrounded by muscle and thus has an excellent osseous circulation. Nonunions can be complicated by bone loss, malalignment, or infection, resulting in difficult challenges for the patient and surgeon. They lead to prolonged recovery for the patient and add significant medical and societal costs. Costs were 2.3 times greater in patients who developed tibia nonunion compared to those who healed primarily.[217]

Mechanical stability and local biology play key roles in fracture healing, as noted earlier. The most common cause of nonunion is infection, which is initially evaluated by serologic markers of inflammation including a complete blood count, erythrocyte sedimentation rate, and C-reactive protein. Metabolic and endocrine abnormalities such as vitamin D deficiency, hyperglycemia (diabetes), malnutrition, hyper-/hypothyroidism, parathyroid disease, disorders of the hypothalamic-pituitary-gonadal axis, and diseases leading to calcium and phosphorous imbalances (including kidney disease) have also been linked to nonunion.[194] Therefore, it is important to screen patients for undiagnosed metabolic and endocrine diseases, especially in cases where uneventful healing is expected. When metabolic or endocrine abnormalities are discovered, a multidisciplinary approach to nonunion treatment including endocrinology and internal medicine consultation may improve a patient's outcome. Polytrauma patients with initial complicated clinical courses are at greater risk of nonunion. It has been proposed that tobacco abuse leads to an increase risk of nonunion.[194] Nonsteroidal anti-inflammatory drugs (NSAIDs) have also been shown to increase the risk of nonunion in some studies, but larger prospective studies are underway to better answer this question.[194,218] This concern must be remembered as the opioid epidemic has influenced surgeons to limit narcotic prescriptions and turn to alternative pain treatment strategies, potentially including the more liberal use of NSAIDs.

Nonunions are categorized as hypertrophic, oligotrophic, and atrophic.[219] These distinctions are critical in that they describe the underlying cause of the nonunion and therefore guide treatment. Hypertrophic nonunions are fractures that have failed to heal despite an adequate local blood supply and obvious formation of callus. Mechanical stabilization alone usually leads to union in this situation. Oligotrophic nonunions show minimal callus formation but no bony resorption. Atrophic nonunions show little to no callus formation and have local bone resorption. Bone grafting along with mechanical stabilization are required in oligotrophic and atrophic nonunions.

Nonunion treatment requires a comprehensive evaluation of both patient physiology and injury factors to arrive at realistic treatment expectations. In some cases, patients are better served with amputation rather than a long, painful, expensive treatment course that can result in a healed extremity that is painful and dysfunctional. Contemporary lower extremity prostheses can allow a wide range of functional activity including running, biking, working, and resumption of normal daily activity, especially in patients who were in good physical condition prior to injury.

Malunion

Malunion is an abnormally healed bone with residual shortening, angulation, or malrotation. The evaluation of a patient with malunion focuses on whether the deformity has a significant impact on the function of the limb. Physical examination should assess joint motion, gait, and coronal and sagittal plane alignment. Radiographic evaluation includes orthogonal radiographs of the affected bone, standing full-length radiographs, and CT scan to identify coronal, sagittal and rotational deformity. Functional limitations are the most common indication for surgical management of malunion in the upper extremity. However, pain as a result of long-term weight bearing on malaligned lower extremities is the primary indication for surgical intervention in the lower extremity. Significant malalignment may predispose a patient to progressive osteoarthritis from asymmetric joint loading. Length leg discrepancy can lead to both joint and low back pain. Complex malunion reconstructions require thoughtful consideration of both patient and injury factors prior to embarking on a prolonged treatment course.

Musculoskeletal Infection

Musculoskeletal infection after fracture surgery is a serious complication that can lead to significant treatment morbidity or even loss of limb. Polytraumatized patients are susceptible to organ dysfunction and infection from altered immunologic responses.[220] Open fractures, wound contamination, soft tissue coverage failure, multiple procedures, prolonged hospital stays, and perioperative malnutrition further increase the risk of infection.[221] The diagnosis of infection after fracture surgery can be challenging in the absence of the typical clinical signs of infection. Serologic testing, specialized radiographic imaging, and fracture site biopsy can all play a role in establishing

the diagnosis. Delayed healing, wound complications, and pain should lead to suspicion of infection. Patients with clinical symptoms of infection and negative culture results should be treated similarly to patients with culture-positive infections.[222] Delay in treatment of infection after internal fixation increases a patient's morbidity and risk for limb loss. Treatment depends on the chronicity of the infection and status of fracture healing. Acute postoperative infection within a few weeks of internal fixation can be treated with surgical debridement, retention of the orthopedic implants, and oral or intravenous antibiotics until fracture union.[223] However, the rate of successful eradication of infection with hardware retention is reported to only be 70%, and patients should be appropriately counseled.[223] Osteomyelitis in the setting of a healed fracture after internal fixation is treated with surgical debridement and removal of implants and a varied course of oral or intravenous antibiotics.[224] Management of septic nonunion should address both infection and mechanical instability. This usually requires multistage surgical treatment and a prolonged course of intravenous antibiotics.[225,226]

Other Sequelae

Lower extremity trauma can lead to loss of function from a variety of causes. Muscle injury can lead to weakness, soft tissue pain, and joint stiffness. Direct injury to articular cartilage, postfixation joint incongruity, and mechanical axis malalignment increase the risk of posttraumatic arthritis. Anatomic reduction of articular injuries and stable fixation that permits early range of motion of injured joints provide the best chance of preventing posttraumatic arthrosis and stiffness. However, an injured articular surface may still develop posttraumatic arthritis despite anatomic reduction due to irreversible cartilage damage at the time of injury. Significant arthritis leads to chronic pain and eventual loss of normal functional activities, necessitating arthroplasty or arthrodesis of affected joints.

Although ankle and subtalar joint arthrosis is usually evident within 1 year, hip and knee deterioration can be more variable. Rapid deterioration of the hip joint can be seen in avascular necrosis after hip dislocation or displaced femoral neck fracture. Joint collapse due to avascular necrosis is typically seen within 3 to 12 months after an injury. Posttraumatic arthritis, however, can take years to develop. Pain and disability from posttraumatic arthritis do not always correlate with x-ray findings. Initial conservative management includes bracing, anti-inflammatory medication, and intra-articular cortisone injection. Arthroplasty or arthrodesis options depend on the anatomic location and are reserved for patients with disabling symptoms. Although arthroplasty of the hip or knee is a highly successful reconstructive procedure for elderly adults with severe symptoms of arthritis, the functional limitations and finite life span of arthroplasty components make it a less attractive option in young patients. However, if no viable alternative options exist and the patient is disabled by pain, arthroplasty is an acceptable option even in young patients.

Contracture and joint stiffness of the hip, knee, and ankle are common complications after lower extremity trauma. This is particularly true for intensive care patients who remain intubated for extended periods. Equinus ankle contractures predictably develop if appropriate splinting, bracing, and/or stretching exercises are not performed. Flexion contractures of the knee can develop rapidly with prolonged bed rest, particularly if pillows are placed under the knees for patient comfort. Contractures of the knee and ankle significantly affect gait and slow the functional recovery. Prevention of contractures is far easier than treatment. A multidisciplinary approach including qualified physical and occupational therapists, nursing, and physicians to focus on early motion and contracture prevention may improve outcomes in this population.

CONCLUSION

Lower extremity trauma has a wide range of presentations, from simple isolated injuries to highly complex multiple injuries in severely injured, polytraumatized patients. Injury characteristics and patient comorbidities lead to an array of treatment paradigms. The treatment course of these patients can span from simple nonoperative measures to prolonged multistage surgical interventions spanning years. Injuries and their associated treatments carry a significant risk of complications, with a wide variation in functional outcome. Optimal results are obtained with a multidisciplinary approach by experienced and dedicated providers.

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45

Peripheral Vessels

Steven R. Shackford

KEY POINTS

- Ischemia of greater than 3 hours can cause irreversible neuromuscular changes; expeditious diagnosis and management are essential to a good outcome.
- Significant prognostic factors related to secondary amputation are duration of ischemia, significant soft tissue loss, blunt mechanism, compartment syndrome, and multilevel arterial injury.
- Computed tomographic arteriograms have replaced catheter-based arteriography for *diagnostic* vascular imaging.
- In the presence of protracted ischemia and the absence of intracranial or intracavitary hemorrhage, systemic preoperative or intraoperative heparinization should be considered.
- In a stable patient with a bruit or thrill, catheter arteriography can provide both a diagnosis and treatment for an acute pseudoaneurysm or acute arteriovenous fistula.

INTRODUCTION

Injury to a major peripheral artery can be limb threatening. If active hemorrhage is present and not urgently controlled, peripheral vascular trauma can be life threatening. In either case, diagnosis and management must be expeditious. This chapter reviews the epidemiology, pathophysiology, clinical presentation, management, and outcome of extremity vascular injuries. Recently, algorithms have been developed for the evaluation and management of vascular injury and are useful for quick reference for the topics discussed in this chapter.[1,2]

EPIDEMIOLOGY

Vascular injuries of the extremities are uncommon. In civilian urban trauma centers, peripheral vascular injuries are present in 1% to 5% of admissions; in rural centers, they are even less common, occurring in less than 1% of admissions.[3,4] Most are penetrating, due to either gunshot or stab wounds, and occur predominantly in males in their third and fourth decades. Blunt trauma sufficient to produce fractures or dislocations is a much less frequent cause. Explosive ordnance and high-velocity projectiles are the predominant wounding agents in the recent military experience.[5]

Because of the increase in endoluminal procedures for diagnosis, vascular control, and therapy, the number of iatrogenic peripheral arterial injuries increased 40% between 1996 and 2003.[6] Iatrogenic arterial injuries occur in approximately 0.6% of patients undergoing endoluminal therapies, and they appear to be specialty related.[7] Most of these injuries involve the groin where access is most commonly obtained for interventional procedures. Iatrogenic vascular injuries can also occur during open operations on the extremities, such as during total joint procedures, intramedullary and external fixation, and plate osteosynthesis. They can present as hemorrhage or ischemia during the procedure or immediately after (usually in the recovery room), or they can present months or years later as claudication or acute limb-threatening ischemia due to thrombosis or emboli.[8] Iatrogenic arterial injuries are definitely not benign. Limb-threatening complications have occurred,[9] and recent reports have documented a 5% to 7% all-cause mortality following iatrogenic arterial injury.[6,10]

PATHOPHYSIOLOGY

Arteries and veins are composed of three tissue layers: the outer adventitia of connective tissue, the central media of smooth muscle and elastic fibers, and the inner intima or endothelial cell layer. Trauma to a blood vessel (artery or vein) can produce hemorrhage, thrombosis, or spasm, either alone or in combination, depending on the mechanism and the magnitude of the force applied to the vessel. Hemorrhage occurs when there is a laceration or puncture of all of three

layers. If the bleeding is tamponaded by the surrounding tissue (ie, muscle or fascia), a localized hematoma will form, which may be pulsatile. If local tamponade is ineffective or only temporarily effective, immediate or delayed hemorrhage ensues, which can be life threatening. Damage solely to the intima occurs when an artery is acutely deformed or angulated. The intima is the least compliant of the three layers; thus, it fractures when the more flexible layers (adventitia and muscle) bend when deformed by an adjacent broken bone or joint dislocation. Intimal injury exposes the subendothelial matrix, which is rich in tissue factor, resulting in activation of the clotting mechanism and subsequent thrombus formation. The thrombus may enlarge or propagate and occlude the vessel or embolize and produce a distal occlusion. The injured intima may also form a flap that can prolapse into the arterial lumen as a result of the forward blood flow dissecting under it. The prolapsed intima can partially or completely obstruct the lumen. Displaced bone from a fracture or dislocation can compress a vessel to the point of completely interrupting flow. Stretching or contusing an artery can produce spasm or segmental narrowing. Bleeding adjacent to a vessel also produces spasm due to the vasoconstrictive effects of hemoglobin on the external surface of an artery. Spasm that reduces a vessel diameter by 50% reduces the cross-sectional area by 75%, producing a significant reduction in distal flow.

Penetrating injuries produce focal injury, whereas blunt injuries tend to be diffuse and injure not only the vascular structures, but also the adjacent soft tissue, bone, and nerves. This adjacent tissue contains small, unnamed blood vessels that would normally provide collateral flow around an injured named vessel. This "collateral" damage worsens or exaggerates ischemia.

Velocity, rather than size, matters in penetrating injury because the energy imparted to a target by a projectile is equivalent to one-half the mass of the projectile multiplied by the velocity of the projectile *squared*.[11] As a result, penetrating mechanisms are classified as either low velocity (<2500 feet per second [ft/s]) or high velocity (>2500 ft/s). Low-velocity wounds include stabs, fragment injuries, and low-velocity gunshot wounds. High-velocity (>2500 ft/s) wounds are most commonly inflicted by a military assault rifle. Because of the imparted kinetic energy, high-velocity weapons are capable of producing significantly more tissue damage than low-velocity weapons.[11,12]

Peripheral vascular injuries can be subtle and go undetected. Symptoms or signs may not be present during the initial phases of care or even during the initial hospitalization. With time, however, they progress insidiously and eventually produce signs and symptoms. The most common late-detected indolent injuries are the arteriovenous fistula and the pseudoaneurysm.

An arteriovenous fistula typically occurs after penetrating trauma that causes a puncture or small laceration to both an artery and an adjacent vein. The high-pressure flow from the artery will follow the path of least vascular resistance into this adjoining vein. Because the fistula is small initially, it is undetected, but with time, it enlarges, diverting nutrient flow from the distal vascular bed. As it enlarges, it produces local, regional, and systemic signs and symptoms such as local tenderness and edema, regional ischemia from "steal," and congestive heart failure if the involved artery and vein are major conduits.[13]

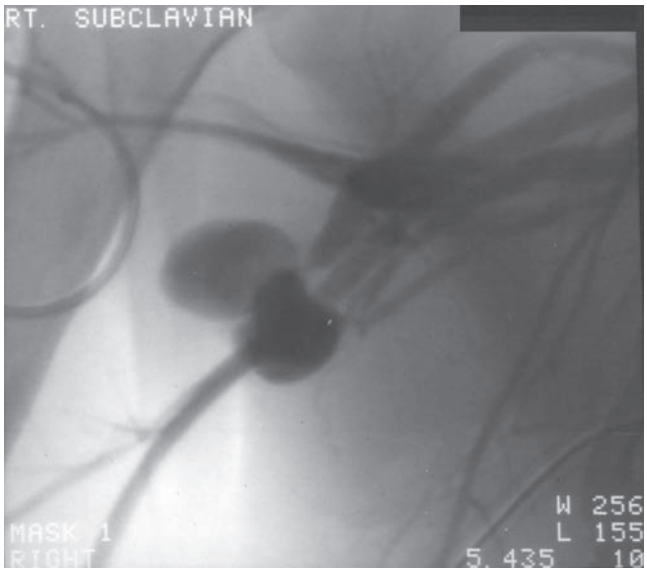


FIGURE 45-1 Acute axillary artery pseudoaneurysm and arteriovenous fistula following stab wound in right axilla. Vessels repaired by simple closure.

A pseudoaneurysm is a result of a puncture or laceration of an artery that bleeds into and is controlled by the surrounding tissue. The artery remains patent; blood flows into and out of the pseudoaneurysm, much like the ebb and flow of ocean water into and out of a tide pool. As a pseudoaneurysm enlarges, it can produce local compressive symptoms, erode adjacent structures, or, rarely, be a source of distal emboli (Fig. 45-1).

Minimal peripheral arterial injuries can heal without an intervention. Dennis and colleagues[14] were the first to demonstrate that small intimal flaps, intimal "irregularities," small pseudoaneurysms, and small arteriovenous fistulae can heal with little residual deformity. Most of these minimal arterial injuries will heal, but it is impossible to predict which ones will progress and eventually develop either acute or chronic symptoms. For this reason, close follow-up with periodic duplex color flow imaging is essential.

A reduction in blood flow produces ischemia when the oxygen demands of the tissue supplied by that artery are not met. The vulnerability of a tissue to ischemia depends on its basal energy requirement, substrate stores, and duration and severity of the ischemic insult. Peripheral nerves are the most vulnerable to ischemia because they have a high basal energy requirement and virtually no substrate (glycogen) stores. Therefore, sensory deficits are often the first manifestations of ischemia. Skeletal muscle is more tolerant of decreased blood flow; histologic changes are not evident unless ischemia has been present for 3 hours or more. In a porcine model, functional derangements and histologic change in both nerve and muscle occur in 3 hours following the onset of ischemia even

if reperfusion is established by the end of that interval.[15] With complete interruption of arterial inflow, such as occurs with occlusion of a major arterial conduit *and* disruption of collateral vessels, and the longer the duration of interrupted flow, the greater is the potential for irreversible ischemic damage.

After prolonged complete ischemia, damage can be extended rather than reversed by reperfusion. This ischemia/reperfusion injury is thought to be initiated by hypoxic disruption or "shedding" of the endothelial glycocalyx, which changes the normal endothelial cell phenotype from anticoagulant and anti-inflammatory to procoagulant and proinflammatory.[16,17] Recent evidence suggests that both the complement and kinin cascades are triggered, which exacerbate the injury by attracting neutrophils. In addition, vascular integrity is lost, resulting in interstitial edema. Interstitial edema raises the interstitial fluid pressure, eventually occluding venules, capillaries, and arterioles and resulting in the "no reflow" phenomenon, compartment syndrome, and myonecrosis or rhabdomyolysis with release of myoglobin and potassium from the irreversibly injured myocytes. Myoglobin is nephrotoxic, and hyperkalemia, if untreated, can lead to a fatal arrhythmia.[18]

PROGNOSTIC FACTORS

There are a several important factors that determine the outcome of an injury to a peripheral vessel. Outcome, in this case, is the need for secondary amputation (amputation following one or more attempts at revascularization). Based on a thoughtful meta-analysis of lower extremity vascular injury, the most significant prognostic factors are amount of significant soft tissue injury, duration of ischemia, presence of a compartment syndrome, high-energy transfer mechanism (ie, gunshot wound or blast injury), multilevel arterial injury, and anatomic location (ie, popliteal artery).[18] Of these, the surgeon can only impact the duration of ischemia, which is the elapsed time from *injury* to *restoration of flow*. Notice that it is the elapsed time from *injury*, not hospital arrival, that is critical, and it is the time to *restoration of flow*, not completion of the arterial repair. Because the time of injury is not always exactly known, it is best to estimate a longer prehospital interval than a shorter one and let this govern the urgency with which management occurs. Based on experimental work using a model of complete vascular occlusion, restoration of flow within 3 hours appears to be optimal to avoid any ischemic changes in nerve and muscle. A delay of greater than 6 hours from injury to restoration of flow results in myonecrosis and moderate Wallerian degeneration of the peripheral nerves.

Other factors that can adversely affect not only limb salvage, but also function include blunt mechanism, associated nerve injury, associated orthopedic injuries, and associated comorbidities. Shock may also be a contributor, but a recent meta-analysis did not find shock to be significant.[19] Compared to the lower extremity, all things being equal, the upper extremity appears to be more tolerant of ischemia, which is likely due to relatively better collateral flow around the shoulder and elbow joints.[20]

CLINICAL PRESENTATION

The presentation of extremity vascular injury varies from obvious life-threatening external hemorrhage from penetrating injury to ischemia from blunt force trauma. As stated previously, penetrating extremity trauma tends to be focal and is usually not accompanied by other injuries. The same is not true for blunt trauma, which is more diffuse and often associated with multiple injuries.

For the patient with a penetrating focal injury, it is important to obtain information from the prehospital providers regarding the approximate time of injury and the agent (eg, stab wound, gunshot wound) and an estimate of the amount of blood lost at the scene and during transport. For a patient with blunt trauma, additional history must include a description of the mechanism of injury (eg, pedestrian stuck, rollover with ejection) and, in the case of motor vehicle crashes, the amount of damage done to the vehicle. This information allows the physician to estimate energy transfer—the greater the energy transfer, the higher the index of suspicion should be for occult vascular injury, not only in the extremity, but also in the torso. Fracture and dislocation patterns can also suggest the possibility of vascular injury. For example, in the upper extremity, a supracondylar fracture of the humerus can be associated with a brachial artery injury. Similarly, in the lower extremity, posterior knee dislocation can be associated with a popliteal artery injury.

The presence of unexplained hemorrhagic shock in patients without evidence of head, neck, or torso injury should direct attention to apparently trivial extremity lacerations. This is particularly important in wounds in the antecubital fossa, groin, and popliteal fossa where initial hemorrhage from a laceration of the deep vessels may have led to hypotension and subsequent thrombosis.

At presentation, the following sequence of steps is recommended: primary survey as described by the Advanced Trauma Life Support (ATLS) course, control of ongoing hemorrhage by compression, or the use of a proximal tourniquet if compression is unsuccessful. Tourniquet use was discouraged in prior military conflicts and used only when all other methods failed primarily because of concerns about improper application.[21] The recent military and civilian experience with tourniquets has shown that proper placement can be lifesaving and improper placement is infrequent.[22,23] Next, a secondary survey, as described by ATLS, is performed, and the primary survey is repeated at regular intervals, particularly if there is a change in the patient's condition.

DIAGNOSTIC EVALUATION

History and Physical Examination

A detailed history and a thorough physical exam can eliminate the need for imaging and contribute to the expeditious management of a threatened extremity. In addition to the elements of the history obtained during the initial survey, the following are also important (particularly in a patient over

the age of 50 years): list of medications (including illicit drugs with vasoconstrictive properties, such as cocaine and methamphetamine), a history of claudication in either or both lower extremities, and a list of current comorbidities.

The physical examination must include systolic blood pressure and temperature, both of which affect the extremity vascular exam. Hypotension, causing peripheral vasoconstriction, will reduce or eliminate the peripheral pulse in an uninjured limb; hypothermia will prolong capillary refill. Therefore, resuscitation and rewarming may improve the pulse exam in the limb without a vascular injury but will have little or no effect in the limb with a vascular injury. Extremity dressings should be removed to assess and document the nature of the underlying wounds. The following should be noted and documented if present or absent (negatives are pertinent for subsequent examinations): active bleeding, hematoma (including whether it is soft or tense), bruit, or thrill. Examination of the uninjured contralateral extremity should precede that of the injured limb to provide the basis for comparison. This includes a vascular and neurologic examination (sensory and motor), careful palpation of peripheral pulses, and assessment of color, warmth, capillary refill, and venous filling. The vascular and neurologic findings in the injured and uninjured limbs must be accurately documented. This provides important information necessary for follow-up examinations, both preoperatively and postoperatively. There is an unfortunate tendency to "overcall" the presence of peripheral pulses. Once any examiner documents that a pulse is present when it is, in fact, absent, there is a tendency of subsequent examiners to do the same. When in doubt, declare the pulse absent and proceed to the use of a continuous-wave (handheld) Doppler device. Venous signals can be heard and mistaken for an arterial signal. Venous signals augment with distal compression; arterial signals do not. The experienced examiner can assess flow based on the character of the audible Doppler signals. However, when there is an abnormal (absent or reduced) pulse, the arterial pressure index (API) should be determined.[24,25] This is done by placing a blood pressure cuff just proximal to the wrist or ankle in the injured extremity. The probe is placed over the distal vessel. The cuff is slowly inflated, and the cessation of the arterial signal indicates the systolic blood pressure at the level of the cuff. The uninjured contralateral extremity and an uninjured arm pressure are then determined. The normal ankle–brachial index is 1.1. Unless the patient has preexisting peripheral vascular occlusive disease, the ankle–brachial index should be at least 0.9, and there should be less than a 20-mm Hg difference between the two lower extremities. An absolute pressure below 50 to 60 mm Hg at the wrist or the ankle indicates limb-threatening ischemia in the patient with a normal systemic blood pressure. The API is not useful in patients with advanced diabetes in whom the proximal conduit arteries are severely calcified making them noncompressible, even at high cuff inflation pressures.

There are very distinct physical findings ("hard signs") that indicate a *high probability* of a vascular injury (Table 45-1). There are equally important "soft signs" that suggest the *possibility* of extremity vascular injury. Of the hard signs, expanding hematoma, pulsatile bleeding, and advanced ischemia require immediate exploration. In the absence of hemorrhage, hemodynamic instability, or advanced ischemia, a bruit or thrill or the presence of one or more soft signs is an indication for further evaluation.

TABLE 45-1: Hard and Soft Signs of Vascular Injury

Hard signs	Indicate a very high probability of vascular injury: • Pulsatile bleeding • Expanding hematoma • Palpable thrill • Audible bruit • Evidence of regional ischemia –Pallor –Paresthesia –Paralysis –Pain –Pulselessness –Poikilothermia
Soft signs	Suggest the possibility of a vascular injury: • History of moderate hemorrhage • Injury (fracture, dislocation, or penetrating wound) • Diminished but palpable pulse • Peripheral nerve deficit

Vascular Imaging

The advent of high-resolution multidetector computed tomography (CT) angiography (CTA) has radically changed the approach to contrast imaging for extremity vascular trauma. Catheter arteriography used *solely* for the diagnosis of a potential vascular injury has been replaced by CTA. Multidetector (64-slice) CTA with the appropriate imaging protocols creates axial, coronal, and sagittal views within minutes.[26] The latest software produces three-dimensional reconstructions without the delays associated with workstation manipulations. In addition to being very accurate, CTA avoids not only the delay necessary to assemble the angiography team, but also the potential complications associated with arterial access. Catheter arteriography is now reserved for patients with suspected vascular injury in whom a catheter-based therapy may be necessary (eg, an arteriovenous fistula or a pseudoaneurysm),[27] patients with blast injuries or shotgun wounds in whom metallic fragments or pellets can produce artifacts on the CTA that obscure the arterial or venous lumen, patients with multilevel injury, and hemodynamically stable patients who have injuries of the thoracic inlet.[28] The indications for vascular imaging have not changed (Table 45-2). In the patient who is neurologically or hemodynamically unstable, a single-injection arteriogram in the trauma room or operating room is a quick and accurate method to evaluate an extremity with a suspected vascular injury (Table 45-3).[29]

TABLE 45-2: Indications for Vascular Imaging in Patients with Extremity Injuries

In patients with blunt force injury

Multilevel extremity fractures with distal ischemia
Crush injury with diminished blood flow
Extensive hematoma with intact distal flow
Presence of a thrill or bruit over an area of injury

In patients with penetrating injury

Significant hematoma in proximity to wound with intact distal flow
Multilevel penetration from a shotgun blast
Presence of a thrill or bruit over an area of injury

TABLE 45-3: Technique for "Single-Shot" Extremity Arteriography

1. Place a radiograph cassette beneath the area of concern in the extremity.
2. Insert and hold steady an 18-gauge needle or short 16-gauge catheter in the femoral or axially artery.
3. Aspirate blood to the level of the syringe containing contrast to avoid air bubbles.
4. Rapidly inject 20 mL of full-strength intravenous contrast agent for the leg and 10 mL for the arm.
5. Delay x-ray exposure for 2 seconds for the proximal upper extremity and proximal thigh, 3 seconds for the forearm and distal thigh, 4 seconds for the popliteal level, and 5 seconds for the tibial vessels.
6. Fluoroscopy with the digital subtraction angiography mode may also be used.

Noninvasive Evaluation

Color flow ultrasound imaging is useful for the delayed diagnosis of chronic vascular injuries, such as a pseudoaneurysm or an arteriovenous fistula, and for the postoperative follow-up of vascular repairs. In patients with severe renal insufficiency in whom the use iodinated contrast might precipitate permanent renal failure, duplex scanning performed and interpreted by a skilled technologist or physician combined with a thorough physical examination can be used to rule out an arterial injury. The quality of the image may be reduced by hematoma, air in the subcutaneous space, and a large wound near the site of injury. If doubt remains, the calculus of harm must be assessed with respect to the risk of a contrast study versus that of an operative exploration.

Practice Recommendation for Extremity Vascular Diagnostic Evaluation

The recommended diagnostic approach is detailed in Fig. 45-2. Physical examination remains the most important

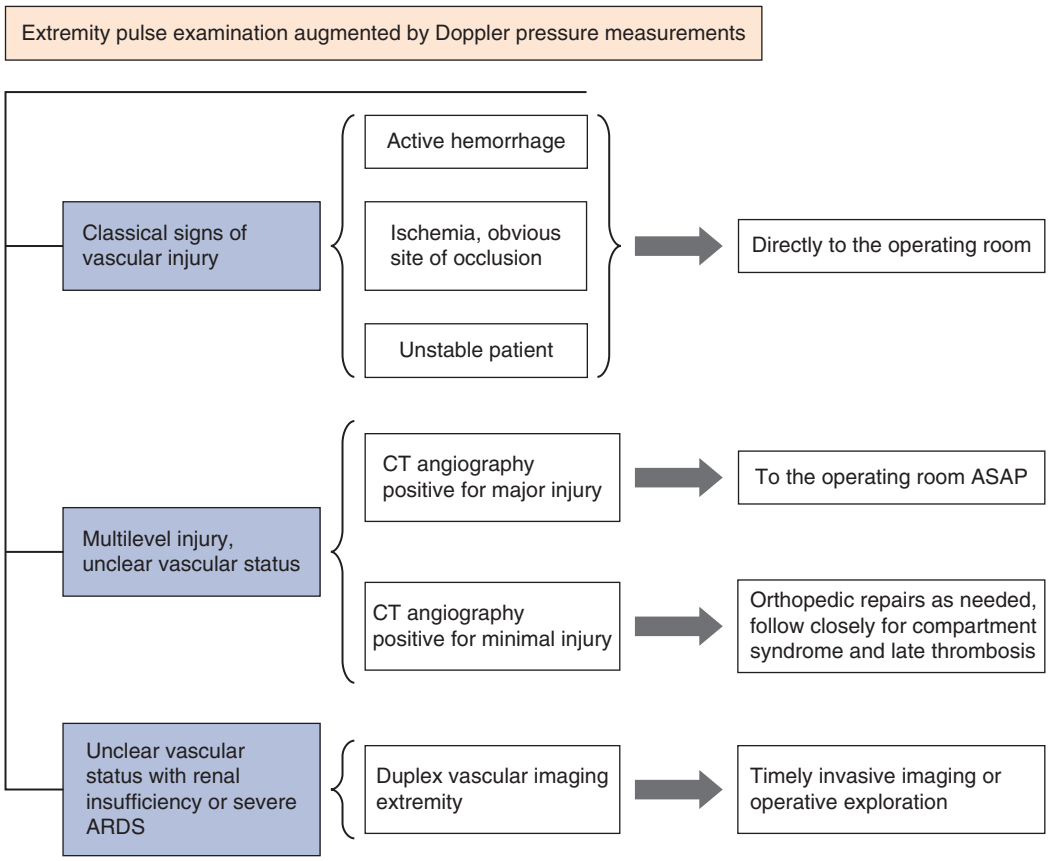


FIGURE 45-2 Diagnostic evaluation of extremity vascular injury. ARDS, acute respiratory distress syndrome; CT, computed tomography.

element of this process. Common sense dictates that patients with pulsatile bleeding, expanding tense hematoma, hemodynamic instability, or advanced ischemia go directly to the operating room to delineate and repair the injury. Expeditious imaging with either high-resolution CTA or catheter arteriography, if a possible endoluminal therapy is needed, must make sense in terms of the expense of time and the value of the results in deciding and directing the management.

GENERAL PRINCIPLES OF MANAGEMENT

Consensus-derived algorithms explaining the rationale for decision making in the evaluation and management of peripheral vascular trauma have recently been published and should be reviewed as a companion to the following information.[1,2]

Minimal Vascular Injury and Nonoperative Management

Minimal vascular injuries are those that are asymptomatic and have the potential to heal without becoming symptomatic. The diagnosis is made following imaging obtained for suspected vascular injury manifested by the soft signs. Minimal vascular injury includes intimal irregularities (eg, small intimal flap), small arteriovenous fistulae, focal spasm, and small pseudoaneurysms.[30] Progression of these lesions to produce symptoms occurs in approximately 5% to 15% of cases and usually occurs early in the postinjury course.[14] Considerable evidence suggests that nonoperative therapy of these asymptomatic lesions is safe and effective. The possibility of progression, while remote, necessitates compulsive inpatient and outpatient follow-up with repetitive physical examinations (including the API) and the liberal use of color flow imaging. Operative therapy is required for thrombosis, ischemia (including ischemic "steal" produced by an enlarging arteriovenous fistulae), and failure of small pseudoaneurysms to resolve.

Endovascular Management

The use of endovascular therapies for extremity vascular injuries is increasing. These therapies include branch coil embolization, vasodilator infusion, and the use of covered and uncovered stents. There are now several small retrospective case series and a single prospective report describing the use of covered stents for the treatment of chronic injuries, such as arterial pseudoaneurysms and arteriovenous fistula, and acute injuries with hemorrhage, dissection, and thrombosis.[5,31-33] The most common injuries treated acutely are those involving the subclavian and axillary arteries, probably due to the relative difficulty in open operative exposure of these arteries. The evidence to support endoluminal therapies for peripheral injuries remains parochial because there is no consensus on the indications (particularly in the case of minimal injury), no uniform definitions of complications, and no comprehensive long-term follow-up of the patients who have been treated.[34] Reports from large databases lack granularity to address some of these important issues.[35] As such, the decision to use an endovascular approach for treatment of acute peripheral vascular injury should be made on an individual case-by-case basis.[2,36]

Operative Management

The successful operative management of extremity vascular injuries requires prompt control of hemorrhage and expeditious restoration of adequate perfusion. These priorities must be orchestrated with the *overall management and care* of the patient. In the neurologically or hemodynamically unstable patient, other priorities will trump *definitive* vascular repair. In either case, *damage control* using a temporary intravascular shunt inserted into the appropriately prepared artery (and vein, if injured) can quickly restore perfusion in an ischemic limb,[37] whereas a tourniquet, appropriately applied (see later), can control hemorrhage.[22] Secondary considerations include adequate tissue coverage of the vascular repair, fracture stabilization, and wound management.

WHO SHOULD REPAIR INJURED BLOOD VESSELS?

Currently, trauma surgeons with general surgery specialty training perform almost 70% of complex vascular repairs of injured arteries, whereas vascular or cardiovascular surgeons perform 27% with similar rates of limb salvage (94% and 95%, respectively).[38,39] In an era of fewer open vascular procedures performed during general surgical training, the repair of extremity vascular injury in the future may not be within the capabilities of many trauma surgeons.[40] It is important that senior trauma surgeons with experience in managing vascular injury train their younger colleagues in the techniques necessary to expose and repair these injuries. Because many surgeons who perform elective vascular surgery are not sufficiently experienced in the *overall management* of a trauma patient with a vascular injury, board certification in vascular surgery does not necessarily qualify a surgeon as capable to handle these injuries, just as the lack of certification does not necessarily disqualify a surgeon. Conversely, there are many trauma surgeons who are very skilled in vascular technique by virtue of their interest and experience. Surgeons with experience in vascular techniques and management of vascular injuries, no matter what the specialty training, should be available at all trauma centers.

OPERATION

Preoperative Preparation

Broad-spectrum antibiotics and, if there is a penetrating wound or an open fracture, tetanus toxoid should be administered as soon as possible. If ischemia of the limb is apparent, greater than 3 hours have elapsed since the time of injury,

and there is no evidence of ongoing bleeding in the limb and no intracranial or intracavitary hemorrhage, consideration should be given to administering systemic unfractionated heparin (70 U/kg) as soon as possible if there is evidence of ischemia.

In controlling hemorrhage, there is no role for "blind" clamp placement in the injured extremity; it is rarely successful and frequently injures adjacent nerves. The operating room has the personnel and the equipment (including lighting and suction) necessary for effective exposure and control. A properly placed tourniquet, a Foley catheter with a 30-mL balloon inserted into the wound and inflated, or a gloved hand compressing the bleeding site during transfer to the operating room will suffice. There are a variety of commercially available disposable tourniquets that are very effective in providing temporary control. The tourniquet is placed proximal to the injury, but as distal as possible to avoid ischemia to tissues that are proximal to the injury. It should not be placed directly over joints or bony prominences because effectiveness may be reduced, and the skin directly under the tourniquet will be at risk for ischemia by direct compression. Finally, it should be applied with pressure sufficient to occlude flow. The time of placement should be recorded to accurately track the occlusion time, which should not exceed 90 minutes to avoid nerve ischemia.[41]

If there is an associated fracture or dislocation, consultation with an orthopedic surgeon will facilitate preoperative planning. The sequence of procedures and conduct of the operation should be discussed. For example, controversy can arise regarding the priority of orthopedic stabilization compared to vascular repair. The use of a temporary vascular shunt to perfuse an ischemic extremity prior to orthopedic stabilization can address both issues.[36] Shunt placement and subsequent detection of Doppler signals in the limb distal to the shunt ensure perfusion and remove the sense of urgency to perform a definitive repair. If there is extensive soft tissue loss, early consultation with a plastic surgeon will facilitate the planning of tissue coverage of the vascular repair.

Once operative priorities have been established, communication with the operating room staff is necessary to ensure the availability of appropriate instrument sets, sutures and graft material, and other ancillary equipment, such as a cell saver for blood retrieval and a patient warming device. Communication with the anesthesiologist is necessary to inform him or her of the patient's resuscitation needs, need for blood products, and estimated duration of the proposed operation.

The surgeon should be present in the operating room when the patient arrives to assist with specific operative preparation, which includes selecting suture and instrumentation appropriate to the proposed procedure[2] and provision of heparinized saline (5000 U of heparin/500 mL) and papaverine hydrochloride (a locally effective vasodilator, 30 mg/mL) for *regional* injection. The surgeon should supervise positioning, prepping, and draping. To ensure that proximal control can *always* be obtained, areas of the adjacent chest and shoulder for upper extremity injuries and the adjacent abdomen (up to and including the umbilicus) for lower extremity injuries should be prepped and draped with the *entire* injured extremity. Because the middle of the night is not the time to pull together the necessary equipment, it is prudent to assemble a standard peripheral vascular trauma set of equipment, sutures, and graft material ahead of time. It is also recommended that the surgeon wear coaxial lighting (headlamp) and 2.5× to 4× magnification loupes, depending on the diameter of the vessel to be repaired (proximal, 2.5× magnification; distal, 4× magnification).

Exposure and Control (See Atlas Figure 76)

Proximal and distal control of the involved artery should be achieved prior to exposure of the injury. The incisions for exposure are those used for elective procedures (see the later section "Management of Vascular Injuries by Anatomic Region"). In proximal extremity injuries with active hemorrhage, the site is chosen to give the fastest exposure of inflow vessel(s) for clamping. A sterile tourniquet can be exchanged for one previously applied. In the operating room, have one team member compress the bleeding site with a gloved hand and a sponge, remove the previously applied tourniquet, and prep the extremity. A sterile tourniquet for use in the operating room (ie, one that contains a bladder for inflation and a gauge for the measurement of cuff pressure) is placed proximal to the wound and inflated, and the pressure and time of inflation are documented. A 5000-U heparin bolus is then given if this is an isolated injury (no evidence of cavitary or intracranial injury). A recent retrospective analysis of the Prospective Observational Vascular Injury Trial (PROOVIT) database demonstrated no difference in either amputation or thrombosis between patients with peripheral vascular injury receiving systemic intraoperative heparinization and those who were not systemically heparinized.[42] However, the group receiving intraoperative anticoagulation contained three times as many patients with an occlusive vascular injury and over twice as many patients with prolonged preoperative ischemia (>3 hours) than did the group not receiving intraoperative anticoagulation. One would have expected higher amputation and thrombosis rates in patients with prolonged ischemia and an occlusive vascular injury.[19] A recent multicenter retrospective study containing more patients than the PROOVIT analysis found significantly improved patency of vascular repairs with no increase in bleeding.[43]

If, after proximal and distal control have been obtained, there is still ongoing hemorrhage from the wound area, an appropriately sized Fogarty balloon-tipped catheter on a three-way stopcock can be gently inserted into the artery above or below the level of injury. The catheter is advanced to the area of the injury (measured beforehand against the Fogarty using the 10-cm markers on the catheter) and the balloon inflated enough to control the bleeding. Extreme care is necessary when inflating the Fogarty balloon—as soon as any resistance to inflation is felt, inflation should stop.

Control of proximal and distal flow is best achieved by "double passing" silastic vessel loops around the vessel above and below the area of injury and gently retracting until flow

ceases. Side branches between the proximal and distal vessel loops are controlled with removable metal clips. If clamps are needed, choose the appropriately sized vascular clamp and close the ratcheted handle only as much as needed to occlude the vessel. Carefully support the clamps to avoid twisting and inadvertent stretching of the vessels.

Before initiating definitive repair of the injury, several sequential maneuvers are necessary. Carefully inspect the injury and debride the injured parts back to normal-appearing intima. Because flow has ceased with proximal and distal control, there may be proximal and distal thrombus in the vessel. Therefore, pass an appropriately sized Fogarty catheter proximally and distally to clear any thrombus. This must be done carefully because the intima can be injured by overdistention of the balloon, as previously mentioned; this is avoided by starting to retract the catheter before starting to inflate the balloon. When the slightest resistance or "drag" is appreciated, stop inflating because the balloon is now in contact with the arterial wall. At this point, continue with retraction of the catheter. Repeat catheter passes until no clot is retrieved from the proximal and distal artery. Inject heparinized saline into the proximal and distal artery using a vessel irrigator (Titus needle or olive-tipped irrigator), first aspirating blood to ensure that the tip is in the lumen. Care should be used when injecting proximally in the brachial and axillary arteries because vigorous flushing of 10 mL may force thrombus or air into the origin of the vertebral artery, potentially causing a posterior circulation stroke.

The debrided and appropriately flushed artery should then be carefully inspected to select the method of repair that should be tension free. Normal arteries in the extremities of young patients are highly elastic and can retract a substantial distance. There is a significant risk of stenosis and thrombosis if undue tension is placed on the artery in an attempt to perform a primary repair of the distracted ends. When there is doubt about tension or about the adequacy of the debridement, it is best to abandon an attempt at primary repair and proceed with interposition grafting, which is the most common type of repair in large series.[35]

Transverse or short oblique lacerations without vessel wall disruption may be repaired with simple interrupted sutures. Longitudinal and long oblique lacerations cannot be closed without compromising the luminal diameter. The injured site should be opened longitudinally for a length sufficient to inspect the intima. The injured or "questionable" intima should be debrided. A vein patch can then be used to close the arterial defect without compromising the diameter of the lumen. If suitable vein is not available, a polytetrafluoroethylene (PTFE) patch is an acceptable alternative in the common and superficial femoral arteries. If a long segment of the anterior arterial wall is debrided, leave the uninjured back wall intact. Leaving the back wall intact, rather than dividing it, prevents retraction of the arterial ends, facilitating vein patch angioplasty or interposition grafting.[20]

When there is complete vessel transection, interposition grafting is usually necessary. The vessel ends should be "spatulated" or beveled to ensure a nonstenotic anastomosis. To ensure a tension-free anastomosis, mobilize proximal and distal segments of the uninjured portion of the artery even if it means sacrificing some minor tributaries. The optimal interposition graft material is autologous greater saphenous vein harvested from an uninjured leg. Native vein graft is preferable because it has elastic properties that make it very compliant with the normal pulsatile flow of an artery. It also has a diameter that approximates that of an extremity artery and produces an adequate size match for grafting in the arm and leg. Venous intima is less likely to be thrombogenic and has superior long-term patency when compared with prosthetic material when used with smaller vessels (popliteal and tibial). When saphenous vein is unavailable, lesser saphenous vein should be used. Cephalic vein has been suggested as a suitable second choice, but cephalic vein is less muscular than the greater and lesser saphenous and will eventually dilate after it has been "arterialized." Both the cephalic vein and the lesser saphenous vein are more difficult to harvest than the greater saphenous. If time of ischemia is a concern, one can insert a temporary shunt into the injured artery and vein and proceed with the harvest.

A PTFE graft is an acceptable second choice. PTFE has a short-term patency of 70% to 90%, and infections are rare even in contaminated wounds.[44] Patency of PTFE grafts is equivalent to that of vein for injuries proximal to the popliteal artery, but inferior to vein for popliteal and more distal vessels. PTFE grafts of greater than 6 mm in diameter should be used.[45] All arterial repairs must be covered with soft tissue to prevent infection or desiccation of autogenous tissue, both of which can lead to hemorrhage or infection.

Single-vessel arterial injuries in the distal forearm and distal calf may be ligated if there is sufficient collateral flow through the remaining vessels. Observing back-bleeding through the distal injured end of the vessel indicates adequate collateral flow. Doppler signals in the hand or forefoot vessels also indicate adequate distal perfusion. When in doubt, perform an intraoperative arteriogram.

Venous Injuries

Injured small veins can be ligated without sequela. Definitive repair of major veins (ie, iliac, femoral, superficial femoral, popliteal, axillary, and subclavian) should be undertaken if the patient is physiologically stable.[46] If the patient is unstable, ligation is expeditious and effective in controlling hemorrhage.[47] A damage control approach can also be used in the unstable patient by inserting a temporary intravascular shunt into the appropriately prepared vein. The shunt should first be placed into the distal end of the injured vein to confirm proximal flow in the vein and to ensure that the distal end of the shunt has not been placed into a valve cusp; the other end is then placed into the proximal vein and flow confirmed using Doppler interrogation. Lateral venorrhaphy, best performed with a running 6-0 or 7-0 Prolene suture, is possible in most venous injuries, taking care to avoid undue tension and "puckering" due to placing stitches too far apart and creating a purse-string effect. Vein patch closure or panel graft

interposition is occasionally required. In the lower extremity, major venous ligation leads to venous hypertension, which can increase the risk for compartment syndrome. Autologous vein patch angioplasty should be considered in the more extensive injuries. The vein patch must be of a generous size to maintain adequate luminal diameter. Uncommonly, stab wounds result in a transversely oriented transaction that may be primarily repaired by simple anastomosis of the cut ends without causing significant stenosis. More extensive circumferential injuries require a saphenous vein panel graft interposition. This is performed by harvesting a long segment of saphenous vein, opening it longitudinally, wrapping it around a chest tube or other appropriate large cylindrical structure, and sewing it in a spiral fashion to create a panel graft. This large-diameter graft is a suitable conduit for venous reconstruction. This technique is tedious and requires significant vascular technical ability and experience. While preparing the panel graft, a temporary shunt should be placed into the severed ends of the vein (as described earlier).

Intraoperative Assessment of Vascular Repairs

Because technical problems can occur following repair of a vessel injury, objective assessment of the repair and the distal vascular runoff bed must occur. Palpation of the distal pulses should be performed (another reason why the *whole* extremity is prepped) followed by a handheld Doppler interrogation of the repair and the vessel immediately distal to the repair. Constant high-pitched signals indicate stenosis and should prompt imaging. Intraoperative duplex scanning is useful but requires significant training and experience to perform and interpret the images. A completion arteriogram with either single-injection radiography or fluoroscopy is useful to detect platelet ("white") thrombus or stenosis at a suture line, kinking or twisting of an interposition graft, or the presence of an intimal flap, all of which may cause early failure.

Role of Tissue Coverage

Desiccation or infection of the inadequately covered vascular repair can lead to suture line disruption followed by hemorrhage. Therefore, all repairs must be covered with healthy tissue, preferably muscle. This is typically not a problem with simple stab wounds or gunshot wounds, but tissue avulsion from automobile or motorcycle crashes or debridement of devascularized tissue as a result of blast injury can compromise adequate coverage. These often require rotation of regional muscle or local advancement of skin flaps. Early involvement of a plastic surgeon facilitates planning because a pedicled transposed muscle flap, free tissue transfer, myocutaneous flap, or fasciocutaneous flap may be indicated. However, complex myocutaneous flaps and free tissue transfer are inappropriate at the initial operation because they are time consuming and can put the patient at risk for hypothermia. These are more safely performed in a delayed fashion when the patient has recovered from the initial physiologic effects of injury. Vascular repairs can be temporarily covered by either cadaver skin graft or porcine xenograft.[48] The homograft or xenograft will adhere temporarily, provide coverage, and often can stay in place for 5 to 7 days or longer. Subsequently, split-thickness skin grafting, tissue rotation, pedicle flaps, or free tissue transfer can be performed.

In extreme cases of a severely contaminated wound, extensive tissue loss, or subsequent disruption of an inadequately covered vascular repair associated with obvious distal ischemia, an extra-anatomic bypass is required. Preferably, an autologous vein bypass can be routed through adjacent healthy tissue in the extremity. Less commonly, externally supported PTFE grafts can be tunneled around the vascular injury site to supply distal perfusion as either definitive revascularization or as a temporizing step to allow healing and later placement of a vein graft through the site of injury. Examples of useful extra-anatomic bypass routes for arterial infections are femoral-femoral bypass for iliac infections and obturator bypass for infections in the groin.

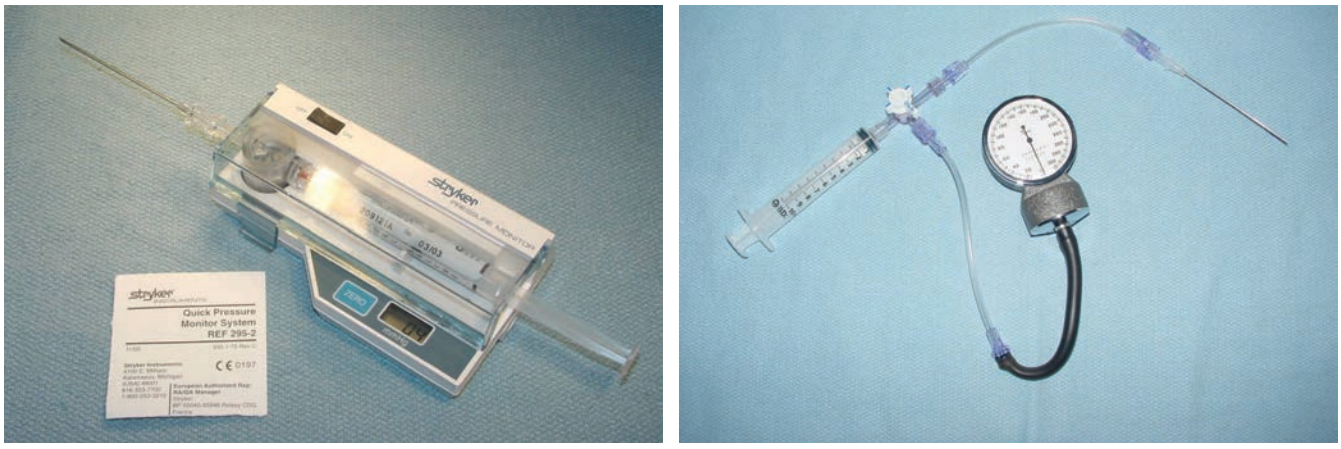
ROLE OF FASCIOTOMY

Unrecognized compartment syndrome following revascularization of an acutely ischemic limb is one of the most common causes of preventable limb loss following extremity trauma.[19] It must be remembered that compartment syndrome can be a manifestation of reperfusion injury (see the section "Pathophysiology," earlier) and may not be clinically apparent immediately after revascularization. Thus, in patients with prolonged ischemia, closed fractures, crush injury, or combined arterial and venous injury, especially if major veins have been ligated, there is a role for "preemptive" fasciotomy in which fasciotomy is performed in conjunction with the initial vascular repair.[49] In patients perceived to be at low risk for compartment syndrome (ie, no crush, no closed fracture, and revascularization in <3 hours), compartment pressure should be measured routinely in the operating room before termination of the anesthetic. A commercially available device (Stryker Surgical, Kalamazoo, MI) facilitates initial and repeat pressure measurements (Fig. 45-3A). If not available, pressure tubing on a three-way stopcock with a blood pressure cuff and monometer may be used (Fig. 45-3B). There is no consensus regarding the compartment pressure indicative of the syndrome; however, any pressure above 25 mm Hg should, at the very least, raise concern and suggest the need for repeated measurements and close observation for signs and symptoms of compartment syndrome (see the later section "Complications and Outcome").

Most trauma surgeons have experience with fasciotomy in the calf. Orthopedic surgeons more commonly perform fasciotomy in the upper arm, thigh, hand, foot, and buttocks.

Calf Fasciotomy (See Atlas Figures 80, 81, and 84)

There are four compartments in the calf to release as follows: anterior, lateral, and deep and superficial posterior



A B

FIGURE 45-3 (**A**) Stryker pressure measurement device (Stryker Surgical). (**B**) Pressure measurement device constructed of connection tubing, syringe, stopcock, and manometer from blood pressure cuff.

(Fig. 45-4). The simplest release is performed using two long incisions (double-incision fasciotomy), one each on the lateral and medial aspects of the calf. Although isolated anterior compartment syndrome occurs rarely, it is recommended to release all four compartments.

The lateral incision should be generous; it begins 2 cm anterior to the fibula and 4 cm below the fibular head to avoid the peroneal nerve and is taken distally to within 2 to 3 cm of the lateral malleolus. The fascia of both the anterior and the lateral compartments can be released through this incision. It is critical to ensure that the anterior compartment is fully released by visualizing and palpating the tibia anteriorly beneath the incised fascia. Misplacing the skin incision posterior to the interosseous membrane can lead to mistaking the lateral compartment for the anterior compartment. This results in failing to release the anterior compartment. This failure can be avoided if the tibia is palpated medially through the releasing incision (beneath the opened fascia), as described earlier, and the intramuscular septum between the anterior and lateral compartment is palpated inferiorly beneath the open fascia of the anterior compartment. If doubt exists about adequate decompression of the lateral compartment, retract the muscles of the anterior compartment and visualize the intermuscular septum separating the anterior and lateral compartment.

The medial calf incision should be made 2 to 3 cm behind the posterior margin of the tibia to avoid lacerating the greater saphenous vein. The fascia over the gastrocnemius is fully released proximally and distally. In the distal calf, the

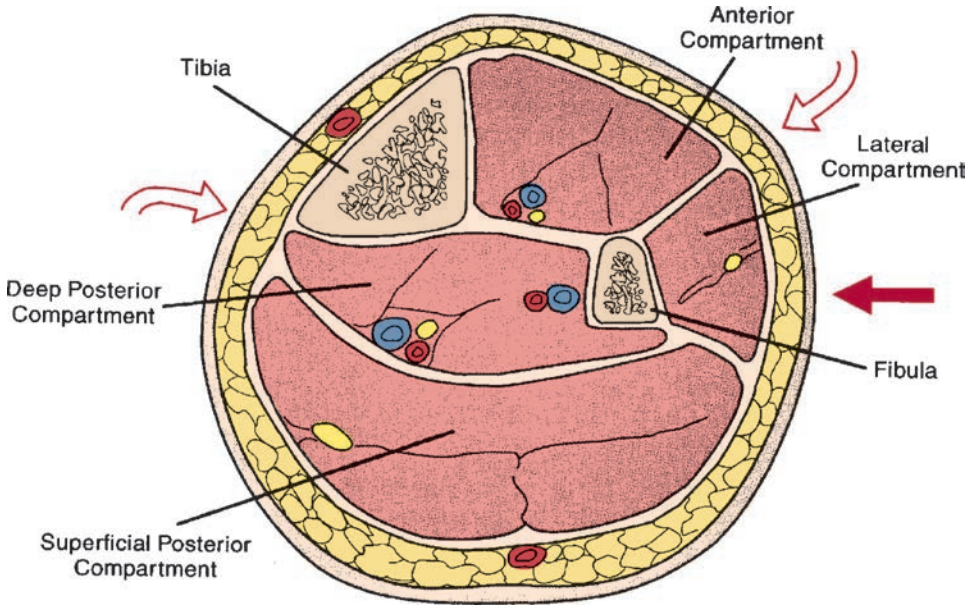


FIGURE 45-4 Cross-section of mid calf showing the four fascial compartments and their contents. Open arrows show sites of double-incision fasciotomy; closed arrow shows site of single-incision fasciotomy. (Reproduced with permission from Frykberg ER. Compartment syndrome. In: Cameron JL, ed. *Current Surgical Therapy.* 5th ed. St. Louis, MO: Mosby-Yearbook; 1995:850, Copyright © Elsevier.)

gastrocnemius and soleus muscles are then retracted posteriorly to expose the deep posterior fascia. This layer is released carefully under direct vision to avoid lacerating the posterior tibial artery.

After the four compartments are released and hemostasis is obtained, a loose dressing is applied; tight dressings will recreate the syndrome when muscle swelling occurs. Postoperatively, the extremity should remain elevated to reduce edema and facilitate wound closure, which can occur within 48 to 72 hours. Early closure can be enabled by commercially available systems (eg, negative-pressure wound therapy [KCI, Dublin, Ireland], skin stretching applications) or by applying Montgomery straps to each skin edge and daily adjusting the tension on the umbilical tapes across the wound. However, split-thickness skin grafting may be required.

Thigh Fasciotomy (See Atlas Figure 83)

Thigh compartment syndrome is less common that that of the forearm and leg. As a result, fasciotomy is only occasionally required. Nevertheless, when a closed femur fracture accompanies an arterial injury, thigh compartment syndrome deserves consideration.

There are three compartments to release: lateral, medial, and posterior, which are encased by the fascia lata. Begin with a generous lateral incision beginning at the intertrochanteric line and ending at the lateral epicondyle. After exposing the iliotibial band, it should be incised completely to release the lateral compartment. Reflect the vastus lateralis superiorly (carefully cauterizing perforating vessels) to expose the lateral intermuscular septum. Incise with a knife or scissors and extend the incision with either long Metzenbaum scissors or a fasciotome. It is worthwhile to repeat the measurement of the medial compartment pressure after release of the lateral compartment, which may be sufficient. If the pressure remains elevated, make a separate medial incision to decompress the medial compartment.

Forearm Fasciotomy (See Atlas Figure 82)

Because an intimate familiarity with the anatomy of the forearm muscles, nerves, and arteries is necessary, orthopedic surgeons or hand surgeons are often consulted to assist with decompression of the three or four forearm compartments depending on local terminology—superficial and deep volar (flexor), dorsal (extensor), and mobile wad (lateral). A dorsal incision and either a volar-ulnar or a volar-curvilinear incision are needed to decompress all compartments (Fig. 45-5). It has been demonstrated experimentally that forearm compartment pressures cannot be normalized through either of the volar approaches only.[50] Individual muscle group epimysiotomy can be undertaken for tense muscle groups, but routinely performing this does not appear to have merit. There are numerous superficial cutaneous nerves that must be carefully avoided. A thorough step-by-step description of the procedure of forearm decompression and a lucid description of the anatomy are available elsewhere.[51]

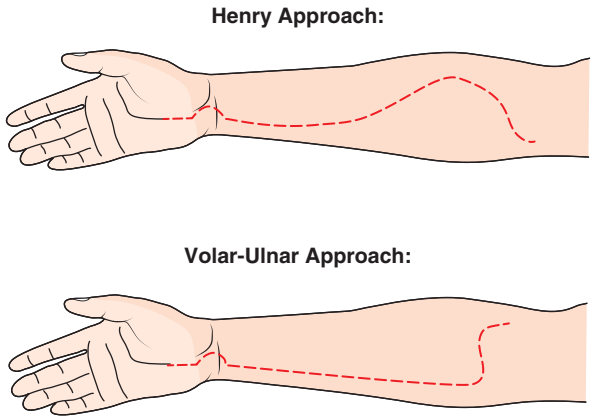


FIGURE 45-5 Curvilinear volar (Henry) and volar-ulnar incisions for decompression of a forearm volar compartment syndrome.

VASCULAR DAMAGE CONTROL

Vascular damage control is necessary when extremity vascular injury is associated with major torso injuries in an unstable patient. It involves rapid control of hemorrhage and prompt restoration of blood flow using temporary intraluminal shunts.[52,53] Fasciotomy may be part of damage control in the high-risk patient (see the earlier section "Role of Fasciotomy"). Ligation should be reserved for those arteries with adequate collateral flow. For example, in the upper extremity, injuries to either the distal radial or ulnar arteries may be treated by ligation provided there is evidence of adequate distal collateral flow assessed by either physical examination or continuous-wave Doppler interrogation. Likewise, in the lower extremity, ligation of a single tibial vessel or the peroneal can be performed following a similar assessment. If there is doubt about the adequacy of collateral circulation (eg, the patient is in profound shock), insert a temporary intraluminal shunt. Ligation of the brachial, external iliac, and superficial femoral or popliteal arteries should be avoided because ligation has a high likelihood of producing limb-threatening ischemia.

There are a variety of commercially available shunts that can be used for damage control (see Atlas Figure 72). The 10F or 12F straight carotid shunts are the most commonly used for this purpose. If these are not available, sterile intravenous tubing or endotracheal suction tubing of adequate size can be used to shunt both the artery and the vein. Venous shunt placement instead of ligation may improve extremity perfusion and lower the risk of compartment syndrome. The common femoral vein may be shunted with a pediatric chest tube.

Damage control shunt placement begins with obtaining adequate proximal and distal control. Thrombus should be cleared, as previously described, followed by the instillation of regional heparinized saline (10 U/mL). The shunt should be placed in a straight line and must be long enough to remain

safely held in place in the proximal and distal vessel with secured umbilical tapes or 2-0 silk ties. Long, looped shunts run the risk of becoming dislodged during subsequent dressing changes or procedures and should be avoided. If a patient is to be transferred to another facility, place a tie around the center of the shunt with one end of the ligature tied to the proximal and one end tied to the distal shunt ligatures that are securing the shunt in the vessel. This will ensure that the shunt is not dislodged during the transport. Securing the shunt with ligatures necessarily injures the intima at the site of their placement; those portions of the artery must be debrided at the time of definitive vascular repair. Temporary shunts have successfully been left in place ("dwell time") for greater than 48 hours. Complications of shunt thrombosis and dislodgement are low, but are likely to increase with longer dwell times.[52,53]

If there is injury to a major proximal vein of the lower extremity, it should not be ligated but should be shunted (see the section "Venous Injuries," earlier). This allows the option of repairing or ligating at a time when the patient is stable. Ligation will result in venous thrombosis, and subsequent venous repair will not be possible. However, in dire situations with a profoundly unstable patient, venous ligation may be the best option. After major lower extremity venous ligation, one should consider preemptive calf fasciotomy.

The timing of definitive vascular repair following damage control procedures is determined by the condition of the patient. Hemorrhage must be controlled and hypothermia, coagulopathy, and acidosis must be corrected prior to returning the patient to the operating room. Concern for shunt patency should not drive the decision to return to the operating room. The patency of temporary shunts used for damage control is 95%, even with protracted dwell times of up to 72 hours.[52]

COMBINED ARTERIAL AND SKELETAL EXTREMITY TRAUMA

Vascular injury complicates less than 2% of extremity fractures and dislocations, but skeletal trauma is present in 10% to 70% of patients with extremity vascular injuries.[54] In both civilian and military series, combined arterial and skeletal extremity trauma carries a substantially higher risk of limb loss than does isolated skeletal or isolated arterial injury.[19]

Successful management requires both prompt diagnosis and coordination of the efforts of both the orthopedic and vascular surgeons. Treatment priorities must be discussed; that is, should fracture fixation go before repair of arterial and venous injuries? Each case must be individualized, but concern regarding the priority of either procedure is allayed by inserting a temporary intraluminal shunt.[52] This can be done relatively quickly, and the orthopedic surgeon may then proceed with fracture fixation. Once the extremity is stabilized, the definitive vascular repair can be performed. Combined injury damage control in hemodynamically or neurologically unstable patients consists of placement of intravascular shunts, fasciotomy, and external fixation.

The Mangled Extremity

The ultimate in combination injuries of the extremity has been euphemistically termed *mangled* because it consists of severe injury to the bones, muscles, soft tissues, nerves, and vessels as a result of high-energy transfer. The amputation rate for mangled extremities remains at 20%.[55]

The grotesque appearance of these threatened limbs can be a distraction from the more severe associated injuries of the head and torso. Focus must be shifted to the initial evaluation or primary survey using ATLS guidelines (see the section "Clinical Presentation," earlier). Hemorrhage from a mangled distal extremity can be controlled with proximal tourniquet application (see the section "Preoperative Preparation," earlier). If extremity hemorrhage cannot be controlled or the patient is persistently hypotensive, proceed to the operating room where cavitary injuries can be addressed and the instrumentation and ancillary equipment necessary for extremity management are available. The complexity of this type of injury mandates collaborative evaluation with orthopedic, neurosurgical, and plastic and reconstructive surgery consultants to address management options—one of which is amputation. If skin and soft tissues are the only attachment of the mangled limb to the torso, immediate amputation is indicated.[56] A management algorithm based on the existing literature and expert opinion recommends the following general principles in the stable patient: restore anatomic alignment of fractures, perform vascular and neurologic assessments (see the section "Diagnostic Evaluation," earlier), obtain multispecialty consultation with regard to individual injury components (ie, bone, nerve, and soft tissue) in the operating room overseen by the trauma surgeon, and make a collaborative decision to attempt salvage or amputate based on that assessment.[56] If a decision cannot be made, damage control procedures should be undertaken, and a planned return to the operating room in 24 hours is scheduled. Photographs and extensive notes articulating the nature of injuries and the decision-making process are recommended at the end of the initial procedure. These are helpful for comparisons at the second operation and are useful for accurate communication with the patient and the family in the interim. The use of scoring systems to reliably predict the need for amputation has not been useful because the limb salvage rate has been consistently higher than predicted.[56]

EARLY POSTOPERATIVE MANAGEMENT

Technical problems with the vascular repair are most likely to present within the first 24 hours postoperatively. Therefore, close clinical surveillance is essential. This includes repeated pulse examination and frequent assessment of the extremity for compartment syndrome (see the later section "Compartment Syndrome"). The onset of new neurologic deficits is an important indicator of continuing ischemia and should prompt assessment of the patency of the vascular repair and the pressure within muscular compartments. Loss of a palpable pulse,

pallor replacing rubor (following ischemia, the reperfused limb should be hyperemic after rewarming), and a loss of or a change in the character of the Doppler signal (monophasic replaces triphasic or biphasic) should prompt a return to the operating room.

The use of systemic anticoagulation and antiplatelet medications in the early postoperative period is discouraged in patients with multiple injuries secondary to blunt trauma. Full anticoagulation with heparin is reserved for isolated penetrating extremity injuries with repair of small vessels.

MANAGEMENT OF VASCULAR INJURIES BY ANATOMIC REGION

The following sections provide information regarding the management of injuries to specific arteries based on their anatomic location. It is assumed that the recommendations for diagnosis and management described earlier are used since they are applicable to all regions. It is advisable to carefully review texts with detailed descriptions of the anatomy of the arteries to be discussed and the techniques used to repair them.[20]

Upper Extremity

Because of their proximity to the nerves of the upper extremity, vascular injury is frequently associated with an injury to the adjacent nerve. When combined injuries do occur, the nerve injury, rather than the vascular injury, determines the quality of the outcome in terms of function and chronic pain (see later section "Posttraumatic Pain Syndromes"). Compared to the lower extremity, the arteries of the upper extremity are less muscular, which means they must be handled with extreme care during operative repair.

SUBCLAVIAN AND AXILLARY VASCULAR INJURIES (SEE ATLAS FIGURE 69)

Subclavian injuries are the least frequent of extremity vascular injuries. The most common mechanism of injury is penetrating to either the chest, base of the neck, or shoulder; 50% of patients with a subclavian injury will be in shock. Blunt mechanisms produce contusions with intramural hematoma, laceration from shards of a fractured first rib or clavicle, or intimal disruption with thrombosis. A chest radiograph with radiopaque markers placed over any penetrating wound(s) is essential; a hemothorax, an apical cap, or an elevated hemidiaphragm (as a result of phrenic nerve injury) should arouse suspicion of a subclavian artery injury.

Exposure requires a wide prepping and draping of the ipsilateral shoulder, lower neck, chest, and arm. Proximal control on the left may require an anterolateral thoracotomy through the third intercostal space. On the right, sternotomy may be required for proximal control. Distal control may require an infraclavicular incision. The site of injury is then approached directly through a supraclavicular incision. The right subclavian is more easily approached than the left because it rises higher relative to the clavicle. An effective adjunct to proximal control in the case of a partial arterial wall laceration is the retrograde introduction of a balloon-tipped catheter through the axillary artery to the site of injury. The subclavian may then be directly approached. Surgical exposure can be difficult because of the clinically important structures closely allied with the subclavian. Division of the anterior scalene should only be done after the phrenic nerve has been identified (tracking medial to lateral across the belly of the anterior scalene) and mobilized carefully. Any direct handing of the phrenic nerve can lead to a temporary paralysis of the involved hemidiaphragm. Thus, use careful blunt dissection under the nerve and surround it with a vessel loop. If the clavicle is obstructing either the exposure or the repair, it can be divided or completely removed without sequela.[57] The subclavian artery has a thin muscular coat and it is very intolerant of heavy-handed traction, imprecise suturing, or excessive tension. The third portion of the subclavian artery (between the lateral border of the anterior scalene and the lateral border of the first rib) may be exposed by dividing the clavicle at the junction of the proximal third with the distal two-thirds and retracting it inferiorly to be reattached later or completely removed.[8]

Exposure of the axillary vessels is obtained through a transverse infraclavicular incision carried down to the pectoralis minor muscle by splitting the fibers of the pectoralis major muscle. The pectoralis minor tendon is divided at its insertion on the coracoid process; it will then retract inferiorly out of the way. The axillary artery and vein are located immediately below the muscle (Fig. 45-6). Care must be taken to avoid the cords of the brachial plexus, which are in close proximity to the axillary vessels. More distal exposure may require completely dividing the pectoralis muscle; however, this is not commonly needed.

Injuries of the subclavian and axillary vessels are rarely amenable to simple suture repair. Subclavian and axillary arterial injuries are preferentially repaired with an interposition of externally supported ("ringed") PTFE. Venous injuries may be ligated unless there is extensive soft tissue injury with disruption of collaterals. Occasionally, vein or PTFE patch repair is possible. The risk of venous hypertension is low, and ligation should be used unless the vein repair can be done expeditiously. Forearm or upper arm fasciotomy is rarely required with vascular injuries at the subclavian and axillary level. However, close follow-up is necessary.

Severe blunt trauma to the shoulder and upper extremity can cause complete disruption of the musculoskeletal support of the shoulder girdle producing what has been termed *scapulothoracic dissociation* or a *closed forequarter amputation*. A common mechanism is high-speed motorcycle crash. The diagnosis can be made from a chest radiograph that shows ipsilateral sternoclavicular dislocation and lateral displacement of the medial border of the scapula. The brachial plexus and either the subclavian or axillary artery and vein can be completely disrupted or severely stretched, producing a large hematoma and a pulseless, flaccid, and insensate upper extremity. The outcome is uniformly poor because of the neurologic injury.[58] Because of the abundant collateral circulation around the shoulder, the loss of the pulse with scapulothoracic dislocation is infrequently associated with

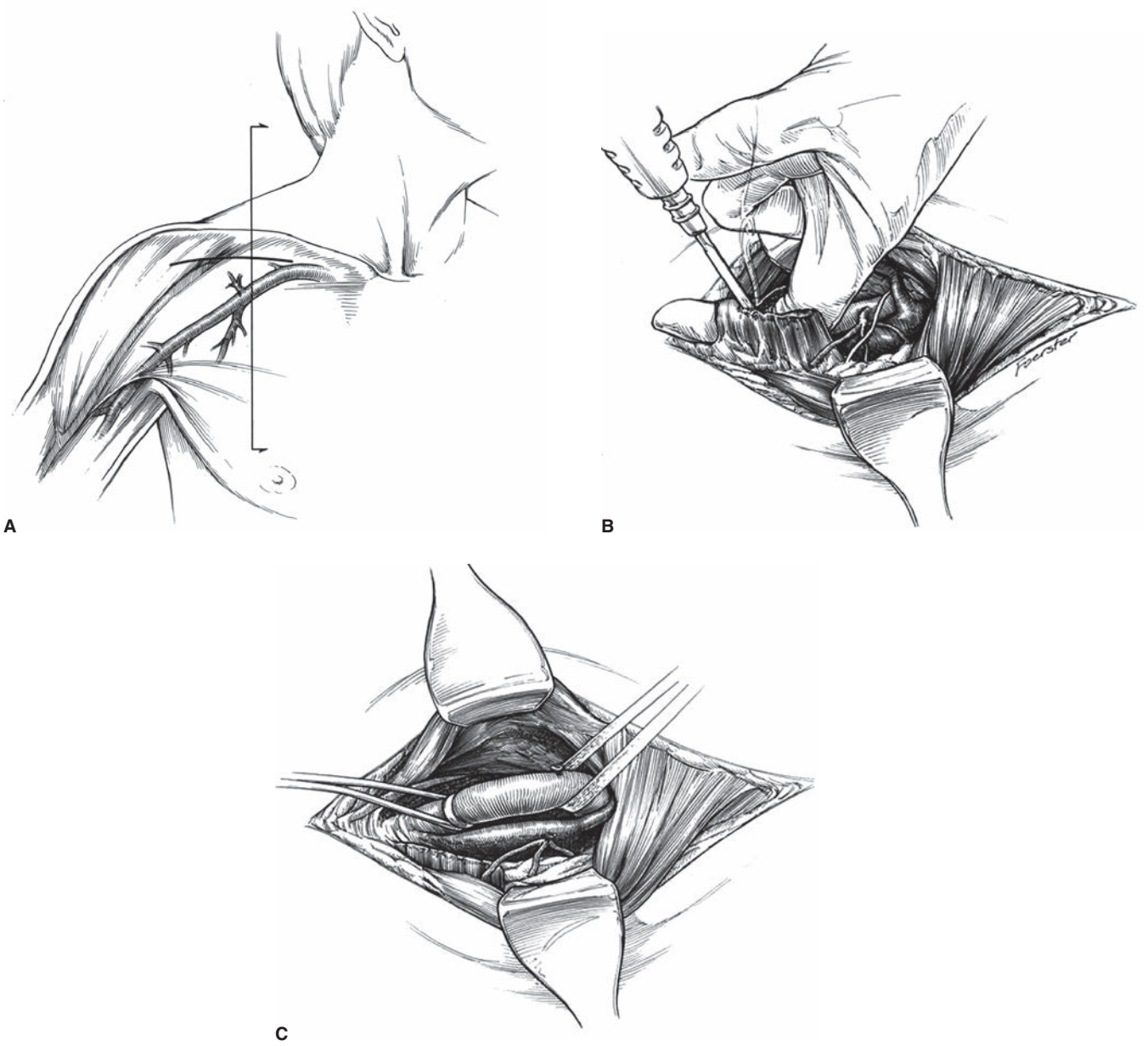


FIGURE 45-6 Approach to the axillary artery for proximal arterial control. (**A**) Location of infraclavicular incision. (**B**) Division of pectoralis minor muscle at coracoid process. (**C**) Exposure and control of axillary artery. (Reproduced, with permission, from Rutherford RB, ed. *Atlas of Vascular Surgery: Basic Techniques and Exposures.* Philadelphia, PA: WB Saunders; 1993. Copyright © Elsevier.)

limb-threatening ischemia. It may even be necessary to ligate the subclavian artery to obtain hemostasis. Recent publications have confirmed that the subclavian artery can be ligated without producing ischemia and that loss of a radial pulse following scapulothoracic dissociation has minimal risk of ischemia.[59,60] Occasionally, patients request amputation because of the burden of an insensate and paralyzed arm that is vulnerable to repeated injury and infection.

Endovascular management has been increasingly reported for axillary and subclavian injuries.[61,62] The apparent and theoretical advantages of endoluminal management include reduced morbidity and blood loss and seem particularly suited for noncompressible hemorrhage from an injury in the thoracic inlet.[61] A recent multicenter study of 223 patients with subclavian and axillary artery injuries revealed that open procedures were more common for the more severe injuries, whereas the endovascular approach was more common for pseudoaneurysms. Outcomes were equivalent with regard to limb salvage and mortality.[62]

BRACHIAL ARTERY INJURIES

Brachial artery trauma is most frequently penetrating from interpersonal violence or laceration from glass shards.

Supracondylar humerus fracture is the most commonly associated orthopedic injury.

Prepping and draping for proximal or distal injuries should include the shoulder and infraclavicular region as well as the entire arm to the fingertips; the distal arm can be draped with a sterile sleeve, which can be rolled up or cut for additional exposure or to palpate the radial pulse.

Exposure is obtained through a longitudinal incision over the course of the artery on the medial aspect of the upper arm.[20] Proximal control for high brachial artery injuries may require control of the axillary artery in the infraclavicular region. Distally, the incision can be extended with an S-shaped extension across the antecubital fossa from ulnar to radial aspect and onto the forearm to expose the origins of the forearm vessels.

Proximal control for injuries of the distal brachial artery and the forearm vessels may be temporarily obtained with a sterile pneumatic tourniquet. This adjunct, however, should be removed as soon as vessel loops or vascular clamps can be applied in order to restore collateral flow.

Simple lacerations may be treated by direct suture repair if it can be performed without tension. Saphenous vein interposition should be chosen whenever vessel injury is extensive or if primary tension-free repair is not possible. There is no role for brachial vein repair unless there is extensive soft tissue injury.

Forearm fasciotomy, particularly in the setting of prolonged ischemia, must always be considered prior at the completion of the brachial artery repair. Intraoperative compartment pressure measurements may be normal, but reperfusion edema and swelling can produce a delayed compartment syndrome. Repeated postoperative follow-up is essential to identify this delayed complication.

FOREARM ARTERIAL INJURIES

The brachial artery gives rise to the ulnar and radial arteries after crossing the antecubital fossa. The ulnar artery is larger than the radial artery in the upper arm and gives rise to the interosseous artery. Although the radial artery is more superficial and easily palpated at the wrist, the ulnar artery is the dominant blood supply to the hand in 60% of patients. Both vessels contribute to the superficial and deep palmar arches. The ulnar artery is the dominant supply to the palmar aspect of the hand, and the radial artery is the dominant supply to the dorsum. The palmar arches are incomplete in up to 30% of patients.

Prepping and draping should include the hand, wrist, and arm to the axilla. Exposure is obtained through a longitudinal incision on the volar aspect of the forearm, taking care to avoid the many cutaneous nerves in this area.[20]

Combined ulnar and radial artery injuries in the forearm require repair of at least one vessel. The larger ulnar artery in the proximal forearm is a better target for saphenous vein bypass. Distally, repair should be performed in whichever vessel is larger or less injured and more amenable to simple repair. Collateral flow should be evaluated by either completion arteriogram or Doppler interrogation, which should include the palmar arch and proper digital arteries.

Isolated ulnar or radial artery injuries can be ligated if there is absolute certainty that flow through the remaining vessel is adequate and supplies the palmar arch. Close inspection of the forearm and hand with palpation of pulses augmented by Doppler interrogation of not only the distal vessel, but also of the palm, is essential prior to ligation. Isolated forearm arterial injury may be repaired if the patient is stable and there are no other pressing management priorities.

Lower Extremity

Lower extremity vascular injuries are most commonly caused by penetrating injury. Supracondylar fractures of the femur or dislocation of the knee should be viewed with a high index of suspicion for an arterial injury. There is a role for liberal use of prophylactic compartment release following lower extremity vascular injuries when there has been prolonged ischemia.

COMMON FEMORAL VASCULAR INJURIES (SEE ATLAS FIGURES 71, 73, AND 74).

Prepping and draping should include the foot and the ipsilateral lower quadrant of the abdomen; this allows exposure of the external iliac for proximal control, if necessary (Fig. 45-7). A longitudinal incision over the common femoral vessels should be generous enough to expose the bifurcation so that both the superficial femoral and profunda femoris arteries can be controlled with encircling double-passed silastic vessel loops. Primary repair is occasionally possible, but most injuries are complex and require either a saphenous vein or PTFE interposition graft.

Femoral vein injuries require careful clamp placement for vascular control. There are numerous lateral, medial, and posterior branches that must be controlled. Vein clamping results in venous hypertension if the artery is not also occluded. Vein patch repair, saphenous vein panel interposition, or heparin-bonded, externally supported PTFE may be required. In unstable patients, venous ligation may be the best course of action. However, if there is sufficient time, shunting of the common femoral vein using a pediatric chest tube may prevent the venous hypertension associated with ligation and, with maintenance of femoral venous return, may augment cardiac filling. Calf fasciotomy should always be considered when the major veins of the lower extremity are ligated.

The profunda femoral artery is well collateralized by branches of the hypogastric (internal iliac) artery.[20] Although simple lacerations should be repaired, more extensive injuries can be ligated unless there is extensive soft tissue injury and loss of collaterals in the buttock and upper thigh. If there is a known preexisting stenosis or occlusion of the superficial femoral artery, repair of the profunda femoral artery is necessary because of its role in providing collateral flow to the leg.

SUPERFICIAL FEMORAL ARTERY AND FEMORAL VEIN INJURIES

Prepping and draping is similar to that presented for common femoral artery injuries. Exposure of the proximal superficial femoral is obtained through a longitudinal groin incision, previously described. The mid portion is located just

FIGURE 45-7 Approach to the external iliac artery in the retroperitoneum for proximal arterial control. (**A**) Location of right lower quadrant incision. (**B**) Retroperitoneal approach by retracting away peritoneum and contents. (**C**) Exposure of external iliac artery. (Reproduced, with permission, from Rutherford RB, ed. *Atlas of Vascular Surgery: Basic Techniques and Exposures.* Philadelphia, PA: WB Saunders; 1993. Copyright © Elsevier.)

behind the sartorius muscle as it travels from superior lateral to inferior medial in the thigh. The sartorius muscle can be retracted either medially or laterally to expose the superficial femoral artery and vein. The incision should be long enough to obtain both proximal and distal control. Although simple repair may be possible in some wounds, most require an interposition graft. Reversed autologous saphenous vein from the uninjured leg should be used if at all possible. The superficial femoral vein should be repaired, if possible. Consideration should always be given to calf fasciotomy in any patient with superficial femoral arterial injury and prolonged (>3 hours) occlusion and patients with ligation of the superficial femoral vein. If fasciotomy is not performed, compartment pressures should be measured before leaving the operating room and repeated frequently in the early postoperative period.

POPLITEAL AND TIBIAL ARTERY INJURIES (SEE ATLAS FIGURES 75, 77, 78, AND 79)

The popliteal artery is fixed in position at the adductor tendon proximally, the geniculate collaterals at its mid portion, and the gastrocnemius distally.[20] These points of fixation at the knee joint place the popliteal artery at risk for injury when the knee is dislocated. In full knee extension, the popliteal artery is under considerable tension. In hyperextension, it is under even more tension—as the back of the tibial plateau moves posteriorly in a dislocation, it impacts and stretches the popliteal artery, often completely disrupting it. An injury to the superficial femoral or popliteal artery occurs in less than 2% of fractures of the femur in young patients (Fig. 45-8). The popliteal vein may suffer the same fate. Because the amputation rate remains 20% to 30% following a posterior dislocation of the knee[63] and because the pulse examination may be insensitive, a CTA with three-dimensional reconstructions is reasonable for this injury.

With the patient in the supine position, prepping and draping are done from the groin to the toes. Adequate exposure requires a medial incision from the proximal popliteal space to the distal popliteal space with care not to injure the greater saphenous vein.[20] Division of the medial head of the gastrocnemius muscle and the semimembranosus and semitendinosus tendons provides a complete view of the popliteal artery and vein and the tibial nerve. This ensures adequate vascular control and the opportunity for successful repair. When closing the wound, approximation of the divided gastrocnemius muscle and semimembranosus and semitendinosus tendons with absorbable sutures will ensure an excellent functional result. Distal popliteal and proximal tibial vessel injuries are approached through a medial incision below the knee along the posterior margin of the tibia. This may be extended distally by dividing the soleus muscle over the course of the tibial-peroneal trunk and the posterior tibial vessels.[20]

Popliteal and tibial artery injuries are usually complex, and primary repair is rarely possible. Saphenous vein interposition is the best method of reconstruction. PTFE should be avoided because it has a high late failure rate when crossing the knee joint.

Empiric four-compartment calf fasciotomy should be considered when the interval of ischemia exceeds 3 to 4 hours. It should always be performed in the setting of combined popliteal arterial and venous injuries.

Some tibial vessel injuries may be ligated if there is adequate flow through the remaining vessels. Caution is advised when considering ligation of the anterior tibial artery following blunt trauma because it has been associated with a much higher rate of amputation than ligation of the peroneal or posterior tibial artery.[64] When in doubt, an intraoperative arteriogram or Doppler interrogation of the dorsalis pedis and posterior tibial arteries in the foot should be obtained. In multiple tibial vessel injuries, a saphenous vein interposition should be performed to the distal tibial vessel that best supplies the pedal arch and that can be most easily covered with healthy soft tissue.

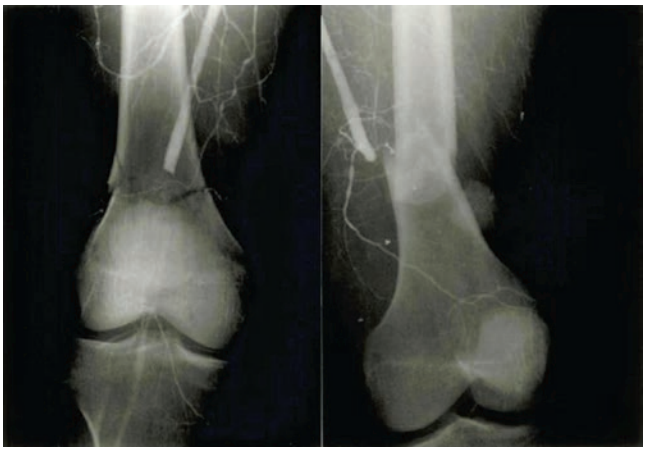


FIGURE 45-8 Bilateral thrombosis of popliteal arteries associated with fractures of the distal femurs. Patient was trapped between wall and oncoming motor vehicle.

COMPLICATIONS AND OUTCOME

Complications following surgery for a vascular injury are those that are generic to any operation, such as a surgical site infection, and those that are parochial to the vascular repair, such as graft thrombosis, and those that may be a combination of the two, such as amputation. Each of the major complications will be discussed in that context.

Surgical Site Infection

The surgical site infection rate following interventions for extremity vascular trauma is not often reported, but some generalizations can be made. It is more common after blunt injury; of the penetrating mechanisms, it is most common after explosive injuries and shotgun wounds, both of which produce significant soft tissue trauma, moderate local contamination, and interruption of the local blood supply to the skin and subcutaneous tissue.

Preventative measures are best practice. These include preoperative broad-spectrum antibiotics with intraoperative scheduled redosing, particularly when the procedure is long (eg, combined with an orthopedic procedure) and the blood loss is substantial. Preemptive debridement of nonviable and potentially nonviable skin and soft tissue at the initial operation is paramount. For severe injuries (see the section "The Mangled Extremity," earlier), scheduled return(s) to the operating room for examination under anesthesia provides an opportunity to perform additional debridement and to assess viability of local tissues.

A surgical site infection that extends to a vascular graft or to the site of a vascular repair or anastomosis can lead to graft thrombosis or exsanguinating hemorrhage. Infection of a vascular suture line may present as a "herald bleed"—a small amount of hemorrhage from a wound considered to be stable and healing. The herald bleed abates only to be followed later with significant blood loss when the vascular repair breaks down or dehisces. Wound dehiscence or wound infection with even remote suspicion of a graft infection merits examination under anesthesia in the operating room. If the graft is not exposed and the deep layers of the wounds appear uninfected, proceed with culture of the wound, aggressive debridement of the superficial layers, wound irrigation, and packing of the open wound. After the first or second dressing change, vacuum-assisted closure can be used. In complicated wounds, a muscle flap to cover the vascular repair site is the best way to avoid infection and vessel erosion with hemorrhage. Once a repair has eroded and bled, it should be debrided back to healthy-appearing artery and ligated, the stump covered with viable tissue, and the graft replaced with an extra-anatomic bypass.

Amputation

Amputation remains the most significant of the complications following extremity vascular trauma. As has been pointed out previously, amputations of the upper extremity are less frequent, most likely because of the extensive collateral circulation around the shoulder.[20] Factors associated with limb amputation are blunt mechanism, time to reperfusion of an ischemic limb, protracted hypovolemic shock, extensive soft tissue injury (see the section "The Mangled Extremity," earlier), popliteal artery injury, failed revascularization, and unrecognized and untreated compartment syndrome (see the section "Compartment Syndrome," later).[19] Technical failures, such as a narrowed anastomosis or a twisted graft (see the section "Early Postoperative Management," earlier), can lead to amputation, but these are not common, particularly if intraoperative completion arteriography is performed (see the section "Intraoperative Assessment of Vascular Repairs," earlier).

Amputation rates during the index hospitalization can vary from 5% to 20%; higher rates are reported from military conflicts with massively destroyed limbs undergoing early amputation in theater, whereas lower rates are reported from civilian centers with mostly isolated penetrating trauma. Long-term follow-up data of patients with vascular injury are sparse, but that void is being addressed with a concurrent multicenter study.[35] The late amputation rate due to severe nerve injury producing chronic pain and allodynia is unknown.

Missile Emboli

Intravascular migration of bullets, shotgun pellets, and other foreign bodies is uncommon and occurs in less than 1% of extremity vascular injuries.[65] The most common occurrence is the migration of small-caliber shotgun pellets from the proximal extremity veins through the right side of the heart and into the pulmonary circulation. Larger bullets may enter arteries and embolize to distal vessels causing ischemia. Bullets that enter the great veins may embolize proximally to the heart or, by the force of gravity, travel in a retrograde fashion into the lower extremity veins.

Diagnosis requires a high index of suspicion and a thorough peripheral vascular examination. Entrance wounds without exit wounds or associated fragments, pellets, or bullets on radiographs of the injured limb should raise the alert that "missile" embolism may have occurred. Arterial obstruction by an offending missile embolus producing acute limb ischemia requires prompt operation for bullet retrieval. In the absence of limb threat, angiography and snare deployment under fluoroscopy can be successful for missile retrieval. A cut-down under local anesthesia at the snare introduction site is usually required to remove the retrieved bullet. Asymptomatic small-caliber shotgun pellets in the extremities are best left alone.

Compartment Syndrome (See Previous Section on Fasciotomy)

All muscle compartments in the extremities are vulnerable to intracompartmental hypertension that can eventually produce a compartment syndrome, leading to nerve and muscle necrosis. A compartment syndrome can be caused by hemorrhage from direct trauma or reperfusion following ischemia (see the section "Pathophysiology," earlier). Compartment syndrome results from swelling of muscle in a confined space (ie, bound by rigid structures, such as fascia and bone). This swelling increases the tissue pressure within the confined space or "compartment," compressing both venous and lymphatic outflow and, eventually, arteriolar inflow, further increasing tissue pressure and reducing perfusion and ultimately resulting in ischemic neuropathy and myonecrosis if not treated promptly. Compartment syndrome occurs most commonly in the muscle compartments of the lower leg. It may be life threatening because of the systemic effect of rhabdomyolysis with subsequent myoglobin-induced renal failure and hyperkalemia.

The anterior compartment of the leg is the most vulnerable. Acute complete traumatic occlusion or ligation of the popliteal vein will increase tissue pressure enough to cause compartment syndrome, particularly in the anterior compartment. The upper extremity has more extensive venous drainage and is less prone to develop compartment syndrome than the lower extremity following venous occlusion or ligation.

Compartment syndrome follows less than 10% of brachial artery injuries and is not a complication of upper extremity venous ligation unless there is extensive soft tissue injury.

Thrombosis of a Graft in an Artery

Despite meticulous techniques, some grafts fail. They fail because of problems with the inflow, the conduit, or the outflow. Intraoperative completion angiography will detect most conduit problems (eg, kinking or a 360° twist of a tunneled vein) but may miss inflow and outflow problems. For this reason, compulsive repeat examinations of the runoff bed are mandatory. A patient who had a palpable distal pulse in the cold operating room but has no palpable pulse in the warm recovery room should raise concern for a technical problem. In this case, check the patient's blood pressure; if the patient is hypotensive and has lost the pulse in the contralateral extremity, the issue is inflow and is systemic and not local. Proceed with resuscitation and investigation of the cause of hypotension. If the contralateral pulse returns with resuscitation but the ipsilateral does not, the problem is local and the patient should be returned to the operating room. If the patient did not have a pulse in the cold operating room and had a satisfactory completion arteriogram and biphasic continuous-wave Doppler signals in the runoff bed, use the Doppler and physical examination to follow the distal circulation. Often a palpable pulse will return and the previously ischemic runoff bed will be hyperemic in the normothermic patient. Postoperative vascular checks must be performed frequently. If there is any doubt about patency, the patient should be returned to the operating room for direct inspection of the repair. If there is no pulse in the graft or distal artery, catheter thrombectomy followed by completion angiography is necessary.

The most common causes of early failure are technical errors. These include intimal flaps, kinking, undue tension, and stenosis at the site of repair or anastomosis. Less commonly, platelet thrombus accumulates on a technically adequate repair. This may occur from the systemic platelet activation associated with injury. It is often associated with extensive muscle or crush injury. Less commonly, it is a manifestation of a heparin allergic reaction, which instigates the process of heparin-induced thrombocytopenia. Low-molecular-weight dextran (40,000) should be given as a 40-mL bolus and then infused at 40 mL/h for 24 hours. Once the thrombus has been removed by thrombectomy, the patient should be followed closely for recurrent occlusion and the cause of heparin-induced thrombocytopenia should be investigated.

Postoperative anticoagulation is not usually helpful in arterial repairs for vascular trauma. However, it may have a role in venous repairs and should be considered in otherwise stable patients.

Posttraumatic Pain Syndromes

The ultimate outcome from extremity vascular injury is largely determined by the associated musculoskeletal and neurologic injuries. The neuropathic or insensate extremity will severely limit functional capacity, not only of the limb, but also of the patient. Complex regional pain syndromes (I and II, previously termed *reflex sympathetic dystrophy and causalgia*, respectively) are disabling neuropathic syndromes that can occur following mild to severe injury. Patients may complain of hyperalgesia or allodynia that may become so severe that they will not touch or move the limb. If the diagnosis is suspected, a diagnostic sympathetic block should be performed. If successful (moderate to complete relief of pain), the patient should be offered a sympathectomy. The earlier this is done in the course of the disease, the more likely sympathectomy will produce long-term benefit. Undiagnosed and untreated neuropathic pain syndromes can lead to a patient-requested amputation.

Ambulatory Venous Hypertension

Late sequelae from venous injuries can be more troublesome than those of arterial injuries. Thrombosis following venous repair is not uncommon, but the vein will usually recanalize, the process of which can lead to valvular dysfunction, venous insufficiency, and ambulatory venous hypertension. If untreated, postphlebitic syndrome occurs, which can be disabling. Compressive support stockings should be given to all patients following venous repair. Because patient compliance with wearing support hose is very poor, patient education is critical in gaining their cooperation.

Pseudoaneurysm and Arteriovenous Fistula

Late development of either a pseudoaneurysm or an arteriovenous fistula is very uncommon due to improved modalities of diagnosis of arterial injury in the acute phase of care. These complications may occasionally be missed or may become apparent weeks to months after injury. For these reasons, patients who have had complex vascular repairs should be followed after discharge with duplex ultrasound. Arteriovenous fistulae and pseudoaneurysms are amenable to either open or endovascular treatment, with endovascular treatment becoming more common.

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Endovascular Commentary to Chapter 45: Peripheral Vessels

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Disclaimer: The viewpoints expressed are those of the authors and do not represent official positions of the University of Michigan, the United States Air Force, or the Department of Defense

It is impossible to overlook the large amount of work and level of expertise that converged in the writing of this chapter on peripheral vascular injury. Dr. Shackford did a masterful job of providing a comprehensive update on all aspects of presentation and management of extremity vascular injury as well as addressing "hot button" topics, such as who is best suited to manage this injury pattern, the greater reliance on computed tomography (CT) imaging for diagnosis, and damage control surgical approaches. Like many types of trauma, the understanding of vascular injury has been advanced from experience and study during the recent wars in Iraq and Afghanistan.[1,2] The sections of this chapter reinforce these lessons learned and make note of a number of still unsettled topics for which our understanding is evolving.

The use of catheter-based, endovascular tools for extremity vascular injury is limited, partly because of the increased use of contrast-enhanced CT angiography (CTA). For decades, traditional catheter-directed angiography has been the gold standard for the diagnosis of vascular trauma. The technique remains useful in some settings, such as the occasional "on the table" angiogram in the setting of an injured extremity. The ubiquitous presence, speed, and ability of CTA to provide a plethora of diagnostic information, however, has allowed it to replace invasive arteriography as the new de facto diagnostic standard. In this era, invasive catheter-based angiography is mostly reserved for instances in which an endovascular procedure (eg, a stent graft or coil embolization) is planned.[3,4]

Endovascular treatment of peripheral injuries is also uncommon and reserved for anatomic locations or patient scenarios in which open exposure and repair are more complicated or morbid. For example, the use of covered stents (ie, stent grafts) has been shown to be particularly useful in the management of select axillo-subclavian artery injuries.[5] In many of these situations, open exposure and repair would be associated with significant blood loss and morbidity due to a combination of incisions and/or working near the brachial plexus. Improved vascular access, imaging, and stent graft technologies now allow for many of these injuries to be crossed and repaired from a transfemoral or transbrachial (ie, retrograde) approach.[3-5]

Most peripheral injuries distal to the thoracic outlet and the inguinal ligament are best managed using open repair techniques. One exception is the use of catheter-directed coil embolization to treat traumatic extremity arteriovenous fistulae. In these cases, if the fistula is large or symptomatic and there are no other indications for an open operation,

arteriography can be performed with an eye toward coiling the inflow vessel as a means of treatment. There are also reports of the effective use of covered stents to treat popliteal artery trauma resulting from a posterior knee dislocation. Unless there are circumstances in which endovascular therapy may provide a particular advantage (ie, morbidly obese patient or need for catheter-directed thrombolytic therapy), however, open repair of this injury pattern is still advised in most cases.

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46

Alcohol and Drugs

Grace F. Rozycki • Laura S. Johnson

KEY POINTS

- Because excessive alcohol consumption and/or opioid pain reliever abuse are frequently observed in injured patients, trauma surgeons should know the common substances abused and the interventions to help the patient.
- An estimated 16 million people in the United States have alcohol use disorder, and an estimated 623,000 of them are adolescents.
- American College of Surgeons Levels I and II trauma centers are mandated to have protocols in place to assess and screen patients who are at risk for alcohol use disorder.
- Prescription opioid overdoses and a recent surge in illicit opioid overdoses (heroin and illegally made fentanyl) are interconnected trends that have fueled America's opioid overdose epidemic.
- Most illicit drugs exert their initial reinforcing effects by activating reward circuits in the brain similar to those involved in alcohol addiction. Continued drug use impairs brain function by interfering with the capacity to exert self-control over drug-taking behaviors.
- Treatment for opioid use disorder includes the following medications: methadone, a synthetic opioid agonist that eliminates withdrawal symptoms and relieves drug cravings; buprenorphine, a partial opioid agonist that has weaker activation of opioid receptors but is well tolerated; and naltrexone, an opioid antagonist that blocks the activation of opioid receptors.
- Surgeons write 36.5% of outpatient prescriptions for opioids, and, therefore, we are uniquely positioned to effect change in the opioid crisis at the local, state, and national levels.

INTRODUCTION

The health consequences of alcohol abuse and nonmedical use of prescription opioid pain relievers are significant. Recent reports indicate that excessive alcohol consumption is responsible for 88,000 deaths annually, making alcohol use the third leading preventable cause of death in the United States. In 2016, 10,497 people died in alcohol-impaired driving crashes, accounting for 28% of all traffic-related deaths in the United States.[1]

Equally concerning is that public health authorities have noted an unprecedented increase in the morbidity and mortality associated with opioid pain relievers. Between 1999 and 2016, more than 630,000 people died from a drug overdose in the United States. In addition, in 2016, a record number (63,632) of drug overdose deaths occurred, a rate of 19.8 per 100,000 persons.[2] These alarming statistics are consistent with the dramatic rise of opioid pain reliever use over the past 15 years. Even the lay press is replete with articles and books documenting this major public health challenge.[3-5]

An integral part of substance abuse, whether alcohol or drugs, is addiction. According to the National Institute on Drug Abuse, drug addiction is a "chronic disease characterized by compulsive, or uncontrollable, drug seeking and use despite harmful consequences and changes in the brain, which can be long lasting."[6] The Centers for Disease Control and Prevention (CDC) cites that 48.5 million persons in the United States, or 18% of persons 12 years of age or older, reported the use of illicit drugs or the misuse of prescription drugs in the past year.[2]

Considering the growing number of people with substance abuse problems and/or addiction, it is likely that trauma surgeons will frequently encounter these patients in the trauma bay or intensive care unit, on the ward, and in the clinic. In order to provide the best care, the surgeon should be aware of an array of medications or substances abused, how to test for them, expected mental status changes, and, in the case of chronic use, the abnormal findings on blood tests and

imaging. Further, the surgeon should understand the need for and application of interventions and their efficacy in helping the patient. Given the magnitude of these major public health challenges, the CDC, the Substance Abuse and Mental Health Services Administration, the National Institute on Drug Abuse, and the National Institute on Alcohol Abuse and Alcoholism have devoted considerable effort to collecting and reporting data on "alcohol-related harms" and "prescription drug overdose" in an effort to better assess the problems, identify trends, and potentially provide solutions. The following sections discuss the substance disorders relative to the scope of the problem, pathophysiology, treatments, screening, and interventions.

ALCOHOL USE DISORDER

Overview of the problem

According to the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5), alcohol use disorder is a "chronic relapsing brain disease characterized by an impaired ability to stop or control alcohol use despite adverse social, occupational, or health consequences."[7] The DSM-5 provides criteria for the diagnosis of alcohol use disorder and its levels of severity (Table 46-1). This classification provides some consistency to a wide range of conditions and helps clinicians with the diagnosis, treatment, and follow-up of patients.

An estimated 16 million people in the United States have alcohol use disorder, and an estimated 623,000 of these people are adolescents. According to the National Institute on Alcohol Abuse and Alcoholism, an alcohol-impaired driving fatality involves a driver or motorcycle rider with a blood alcohol concentration (BAC) of 0.08 g/dL or greater.[8] Although a good marker for acute intoxication, the BAC may vary with the patient's age, sex, race, weight, and food and drugs ingested. Hence, the metabolism of alcohol is no longer considered to be zero-order kinetics. A BAC level of 0.08 g/dL exceeds the legal limit of alcohol for driving and typically occurs after four drinks for women and five drinks for men (of average size and weight) within about a 2-hour period.[9] Mental and physical impairment increases as BAC increases, with the most severe level of intoxication being alcohol poisoning (BAC ≥0.31), a life-threatening impairment characterized by stupor, vomiting, seizures, hypoventilation, and hypothermia (Fig. 46-1).[8] Alcohol poisoning death rates vary substantially by state with a low of 5.3 per 1 million population in Alabama to 46.5 per 1 million population in Alaska. On average, six persons, mostly adult men, die from alcohol poisoning each day in the United States.[10]

Regardless of how much alcohol is consumed, a person can still experience alcohol withdrawal syndrome after abstinence. A major focus of the treatment is the prevention of

TABLE 46-1: *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition, Criteria for Alcohol Use Disorder[7]

In the past year, have you:

1. Drank too much or longer than intended
2. More than once tried to cut down but could not
3. Spent a lot of time drinking or getting over aftereffects
4. Wanted a drink so badly you could not think of anything else
5. Found that drinking interfered with responsibilities
6. Continued to drink even though it caused trouble with relationships
7. Gave up activities in order to drink
8. More than once were in situations while drinking that increased risk of harm
9. Continued to drink even though it made you depressed
10. Had to drink much more than you did to get the effect
11. Found that you had withdrawal symptoms when alcohol effects wore off

The presence of two or more symptoms indicates alcohol use disorder: mild = 2 to 3 symptoms, moderate = 4 to 5 symptoms, and severe = 6 or more symptoms.

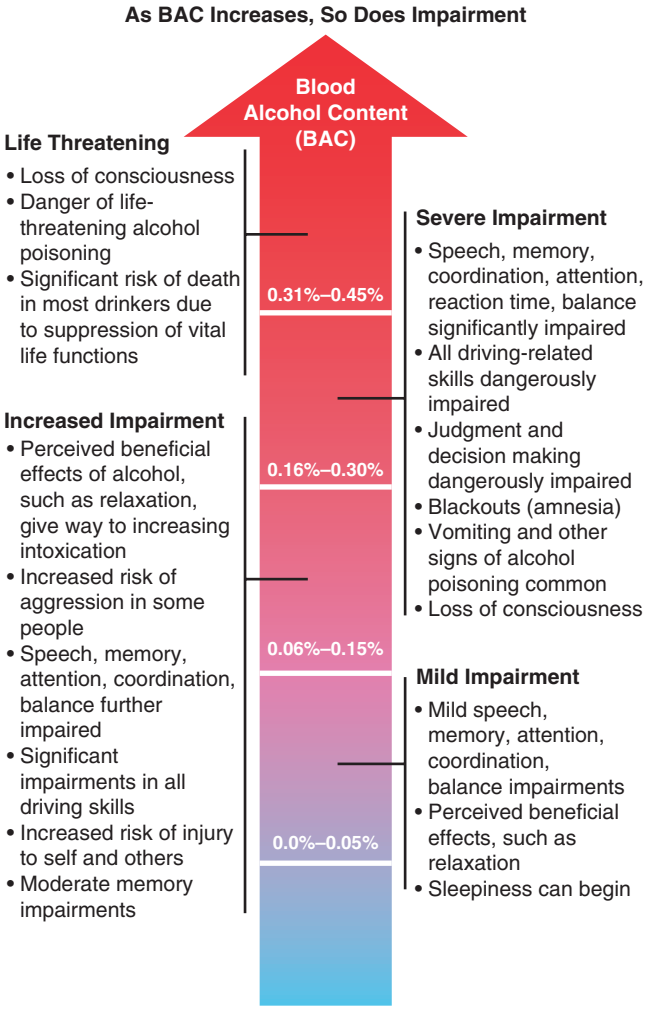

FIGURE 46-1 Signs and symptoms associated with blood alcohol concentration levels.[8] (Reproduced from National Institute on Alcohol Abuse and Alcoholism. Alcohol facts and statistics. https://www.niaaa.nih.gov/alcohol-health/overview-alcohol-consumption/alcohol-facts-and-statistics. Accessed August 21, 2018.)

TABLE 46-2: Spectrum of Alcohol Withdrawal Syndromes[18]

Minor alcohol withdrawal	Alcohol hallucinosis	Alcohol withdrawal seizures	Delirium tremens
Tremor, anxiety, nausea, headache, diaphoresis, palpitations	Visual hallucinations	Seizures	Hallucinations, disorientation, tachycardia, hypertension, fever, agitation, diaphoresis Key manifestation: rapid change in consciousness over short time
Onset within 6–36 hours of abstinence of alcohol	Onset within 12–24 hours of abstinence of alcohol	Onset within 12–48 hours of abstinence of alcohol	Onset within 48–96 hours of abstinence of alcohol

Because of the short action of ethanol, withdrawal symptoms usually begin within 8 hours after blood levels decrease, peak at about 72 hours, and are markedly reduced by days 5 to 8 of abstinence.

delirium tremens, which, if untreated, has about a 15% mortality rate. Table 46-2 lists the spectrum of alcohol withdrawal syndrome, its adverse consequences, and approximate times of onset. In addition to these alterations, recent evidence has focused on the effects of acute alcohol intoxication on coagulation (ie, impaired clot formation and inhibition of fibrinolysis). Identification of this bidirectional effect of alcohol has encouraged caution in interpreting viscoelastic testing and early recognition and treatment of alcohol withdrawal syndrome to prevent adverse coagulation outcomes.[11,12]

Pathophysiology

Understanding the pathologic changes that occur due to alcohol ingestion has relevance in the current and future treatment of alcohol use disorder. In general, alcohol is a central nervous system depressant, but various alterations in neurotransmitters are responsible for the changes that occur following alcohol absorption and during the periods of abstinence. The major neuroinhibitory transmitter of the central nervous system is γ-aminobutyric acid (GABA). Acutely, alcohol inhibits the flow of ions through *N*-methyl-D-aspartate (NMDA)-type glutamate receptors and enhances the activity of GABA receptor channels, producing an overall inhibitory effect on neurons. However, in chronic alcohol use and after abstention from alcohol, GABA receptors are downregulated, contributing to many of the symptoms of alcohol withdrawal. The activity in the glutamate neurotransmitter system, the major excitatory neurotransmitter in the central nervous system, produces many of the signs and symptoms of alcohol withdrawal syndrome. Also, chronic alcohol use increases dopaminergic transmission, which may be responsible for hallucinations.[13,14] Other pathophysiologic processes associated with alcohol abuse include oxidative stress, inflammation, acetaldehyde generation, decreased gut barrier function, impaired anabolic signaling, upregulation of catabolic processes, fibroblast activation, mitochondrial injury, and cell membrane disturbances.[15]

Treatment

The spectrum of alcohol use disorder presents challenges for treatment, as the following factors need to be considered: amount of alcohol ingested per time, chronic abuse, age of the patient, comorbidities, and a history of relapse. For example, a recent study found that age of 55 years or older and severe brain injury (Abbreviated Injury Scale ≥3) were independent predictors of the progression of alcohol withdrawal to delirium tremens.[16] The Clinical Institute Withdrawal Assessment for Alcohol, revised version (CIWA-Ar), is a 10-item scale for the assessment of patients with alcohol withdrawal syndrome.[17] Scores range from 0 to 67, and if the patient's score is less than 8, it indicates mild withdrawal symptoms that rarely require any medications. Scores from 8 to 15 are consistent with moderate withdrawal symptoms and usually respond to benzodiazepines. However, a score greater than 15 indicates severe symptoms and signs and requires that the patient be closely monitored, usually in the intensive care unit. The validity and reproducibility of CIWA-Ar are well documented, and it provides an objective way to monitor the treatment for alcohol withdrawal syndrome.[14,18,19] A key point is the avoidance of delirium tremens with its spectrum of signs and symptoms, including hyperthermia, cardiac dysrhythmias, seizures, hallucinations, and medical disorders, all of which increase the mortality rate.[18] The National Institute on Alcohol Abuse and Alcoholism and the Substance Abuse and Mental Health Services Administration jointly convened a consensus panel of experts to review current evidence on the effectiveness of medications for the treatment of alcohol use disorders. Table 46-3 provides a summary of their findings.[19] When treating alcohol use disorder, the clinician should consider the patient's past experience with medications, level of motivation of abstinence, medical status, and contraindications to the medications. Although the optimal length of treatment is unknown, guidelines for monitoring the patient are provided by the National Institute on Alcohol Abuse and Alcoholism.[19]

Special Groups

THE PREGNANT PATIENT

Simply stated, pregnant patients should not drink alcohol. Avoidance is the best plan because the consequences for the mother and her unborn child are devastating. Alcohol can disrupt fetal development at any stage during a pregnancy

TABLE 46-3: Medications for Treatment of Alcohol Use Disorder[19]

	Disulfiram	Naltrexone	Acamprosate
Frequency of administration	Daily	Daily (oral) Monthly (extended release)	Three times per day
Principal action	Deterrent to drinking alcohol as it causes severe nausea and vomiting when alcohol is ingested	Blocks opiate receptors involved in rewarding effects of drinking	Reduces symptoms of protracted abstinence by counteracting the imbalance between glutamatergic and GABAergic systems associated with chronic alcohol withdrawal
Clinical uses	Patients who are dependent on alcohol who completed alcohol withdrawal	Patients who can discontinue drinking for several days before treatment initiation	Patients who are dependent on alcohol and are abstinent at treatment initiation

and is a leading preventable cause of birth defects and neurodevelopmental abnormalities in the United States. The resulting developmental, cognitive, and behavioral problems can manifest at any time during childhood and last a lifetime. Fetal alcohol spectrum disorders include fetal alcohol syndrome (including partial fetal alcohol syndrome), alcohol-related neurodevelopmental disorder, and alcohol-related birth defects.[20]

Treatment of alcohol use disorder in the pregnant or nursing patient remains a challenge because the medications used to treat the disorder have not been shown to be safe in this population. Pregnant or nursing patients should be referred to an addiction specialist who has experience managing patients with high-risk pregnancies.[21]

OLDER ADULTS

The number of older patients with alcohol use disorder continues to rise. Han et al[22] reported that the number of adults age 50 years or older with substance use disorder is projected to double from 2.8 million (2002–2006) to 5.7 million in 2020.[22] These patients present unique challenges because they frequently have comorbidities that complicate their treatment, increase hospital length of stay, and delay placement to a rehabilitation center. Unique to this age group is the intense shame that they feel about alcohol dependence, and consequently, they will go to great lengths to hide it. Especially in this age group, alcohol dependence may be confused with other medical problems, and so it should be in the differential diagnosis of someone who presents with signs and symptoms of depression or dementia.[19]

ADOLESCENTS

The Federal Uniform Drinking Age Act of 1984 set the minimum legal drinking age to 21 years.[23] Although states follow this law, each state has some latitude on the specifics, such as allowing people younger than 21 years of age to drink with their parents present. According to the 2015 National Survey on Drug Use and Health, an estimated 623,000 adolescents (age 12–17 years) had signs and symptoms of alcohol use disorder.[24] This survey also reported that 5.1 million people age 12 to 20 years admitted to binge drinking in the past month. Underage college drinking (<21 years) is common and takes a toll on the intellectual and social lives of students. An extreme version of excessive drinking on campus occurs when fraternity members "haze" new recruits by encouraging them to consume large amounts alcohol within a short period of time. Recently, hazing has garnered increased public awareness because, in 2017, three college students died from alcohol poisoning in hazing rituals.[25,26] In addition to the tragic loss of life in this young age group, there is also an average of 60 years of potential life lost and hence a tremendous loss to society. Regardless of age, the development of serious medical problems and the consequences of alcohol-related injuries provide incentive for clinicians to assess and screen patients for alcohol abuse in the hope of reducing excessive drinking among adults.

Screening and Assessing Patients

BRIEF INTERVENTIONS

Because alcohol abuse is a significant risk factor for injury, American College of Surgeons (ACS) Level I and II trauma centers are mandated to have protocols in place to assess and screen patients who are at risk for alcohol use syndrome. These time-limited counseling strategies are designed for use in high-volume settings and focus on changing behavior and increasing treatment compliance. Gentilello et al[27] demonstrated a 50% reduction in trauma recidivism when brief alcohol interventions are used. Trauma surgeons have a unique opportunity to capitalize on the teachable moment associated with injury and, hence, implement screening and brief interventions. The screening process contains three steps: (1) identifying the target population and then screening those patients; (2) conducting the brief intervention to assess the patient's understanding of how alcohol contributed to the injury (this may be initiated by informing the patient about his or her admission BAC); and (3) offering professional advice so that the patients can set realistic goals about treatment.[28]

TABLE 46-4: Description of Screening Instruments Adapted from the American College of Surgeons Committee on Trauma Quick Guide[29-31]

	AUDIT[a]	Consumption + CAGE[b]	CRAFFT[c]
Description	10 questions	3 consumption + 4 dependence questions	6 questions about behaviors that are reliable indicators of consumption and risk
Use	Identifies: 1. Unlikely to be at risk 2. At risk; drinks excessively 3. Had problems related to alcohol 4. Likely to have alcohol dependence	Identifies: 1. Likely to be at risk of having alcohol problems 2. Likely to have alcohol dependence	Identifies adolescents likely to have alcohol or drug problems
Score	Ranges from 0–40 16–19: severe alcohol problems ≥20: alcohol dependence	Considered positive if number of drinks per week exceed recommended amount or patient answers "yes" to ≥2 CAGE questions	≥2 positive answers indicate possible problem
Advantages	Sensitive to a wide range of drinking problems Well validated	Provides information on consumption and possible dependence	Designed for adolescent screening Includes alcohol and drug screening

[a]AUDIT: Alcohol Use Disorders Identification Test.
[b]CAGE:
1. Have you ever felt that you should **C**ut down on your drinking?
2. Have people **A**nnoyed you by criticizing your drinking?
3. Have you ever felt bad or **G**uilty about your drinking?
4. Have you ever had an **E**ye-opener first thing in the morning to steady your nerves or get rid of a hangover?

[c]CRAFFT:
1. Have you ever gotten into a **C**ar driven by someone who was high or had been using alcohol or drugs?
2. Do you ever use alcohol or drugs to **R**elax or fit in?
3. Do you ever use alcohol or drugs while you are by yourself (**A**lone)?
4. Do you ever **F**orget things that you did while using alcohol or drugs?
5. Do your **F**amily or **F**riends ever tell you that you should cut down on drinking or drugs?
6. Have you ever gotten into **T**rouble while you were using alcohol or drugs?

In addition to the trauma setting, the Screening, Brief Intervention, and Referral to Treatment (SBIRT) has been shown to be effective in primary care practices, in emergency departments, and among adolescents. Table 46-4 contains a description of three commonly used screening instruments with references for more information.[29-31]

A variation in the traditional screening and brief intervention instruments is the Electronic Screening and Brief Intervention (eSBI). It is conducted using electronic devices, such as computers and mobile devices, and the advantages include the following: (1) the service may be performed across different venues including health care systems or within a community; hence, it expands the reach of the intervention; (2) the delivery of the components may be fully or partially automated, thus overcoming time constraints as a common barrier to implementation; (3) the electronic component facilitates delivery and potentially reduces costs; and (4) individuals may be more likely to participate, especially those who are reluctant to discuss alcohol problems face to face with a physician. A review of 31 investigations on the effectiveness of eSBI showed that in nearly all studies it reduced excessive alcohol consumption and related harms.[32] These studies had the limitations of self-reporting alcohol use and variations in the methods of data acquisition, but according to the Community Guide rules of evidence, eSBI was shown to be an effective screening method.[33]

SCREENING ADOLESCENTS

Because alcohol abuse among adolescents remains a public health and safety problem, the implementation of an age-appropriate screening tool for the early detection of risky behavior can be effective in decreasing alcohol-related deaths and potential years of life lost. The National Institute of Alcohol Abuse outlines a four-step process for alcohol screening and brief intervention for patients age 9 to 18 years. The process includes asking the two screening questions, guiding the patient, assessing the risk, advising the patient, and providing follow-up and support. The uniqueness of this tool is that the questions are simple and direct and that they vary slightly depending on whether the patient falls into the elementary, middle, or high school age group. Also distinctive about this screening tool is that it includes the topic of "friends' drinking," which may be a less intimidating way of approaching the topic.[34] For adolescents, brief motivational interviewing, even a single session, seems to be more successful than other types of interventions.[35] Additionally, a meta-analysis also supported the effectiveness of motivational interviewing–type interventions for adolescents who abuse alcohol or drugs, thus showing promise for this technique in clinical settings.[36] Based on the positive results of these studies, the American Academy of Pediatrics recommends that clinicians who work with children and adolescents regularly

TABLE 46-5: Biomarkers of Alcohol Exposure or Ingestion[38]

Marker	Time to return to normal limits	Type of drinking
γ-Glutamyl transferase	2–6 weeks of abstinence	70 drinks/week for several weeks (a standard drink is equal to 14.0 g or 0.6 oz of pure alcohol)
Aspartate aminotransferase	1 week	Unknown, but heavy
Alanine aminotransferase	Unknown	Unknown, but heavy
Macrocytic volume	Unknown	Unknown, but heavy
Carbohydrate-deficient transferrin	2–4 weeks of abstinence	4 drinks/day

screen for current alcohol use and employ brief intervention techniques during office visits.[37]

Biomarkers

Biomarkers are objective indicators of alcohol exposure or ingestion (Table 46-5). The most commonly known biomarker is the BAC, which is highly sensitive and specific, but only for acute alcohol ingestion. Other biomarkers, although not as specific, may reflect the presence of chronic and/or high-level use of alcohol and may be used as outcome measures to evaluate the effectiveness of medications and behavioral interventions and as evidence of abstinence.

Carbohydrate-deficient transferrin (CDT) is a newer indirect alcohol biomarker. Although the mechanism responsible for the elevation of CDT is not clearly understood, moderately heavy alcohol consumption for about 2 weeks can cause the transferrin molecule to be lacking in the carbohydrate residue in some of its terminal chains.[38] The particular advantage of CDT over γ-glutamyl transferase is that fewer factors other than alcohol use can cause its elevation.

OPIOIDS

Overview of the Problem

The facts surrounding opioid abuse are staggering. Drug overdose deaths, especially those associated with opioids, continue to increase in the United States and globally (Fig. 46-2). The number of deaths from drug overdoses continues to rise among men, women, all races, and adults of nearly all ages. Of the 62,632 deaths due to a drug overdose in 2016, 42,249 of them were due to an opioid.[2] Also in 2016, 2.14 million people age 12 and older had an opioid use disorder, including 153,000 adolescents.[39] From the same report, during the period of July 2016 to September 2017, opioid overdoses increased 30% in 45 states and 70% in the Midwestern area of the United States. On average, 115 Americans die every day

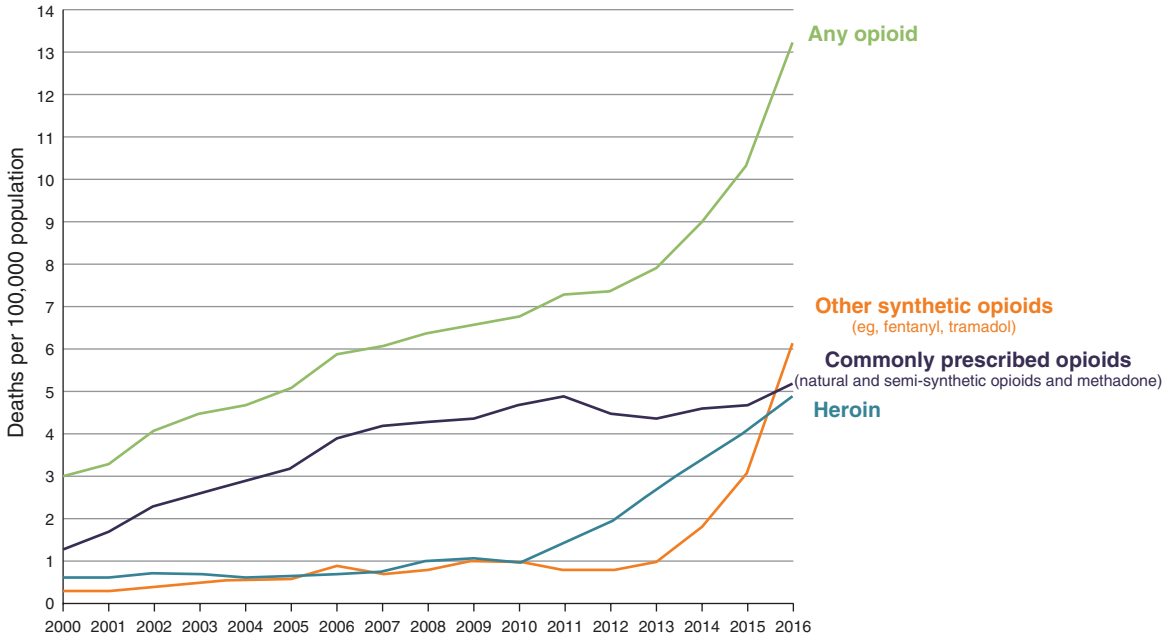

FIGURE 46-2 Overdose deaths involving opioids, by type of opioid, United States, 2000-2016. (Reproduced from the Centers for Disease Control and Prevention.[2])

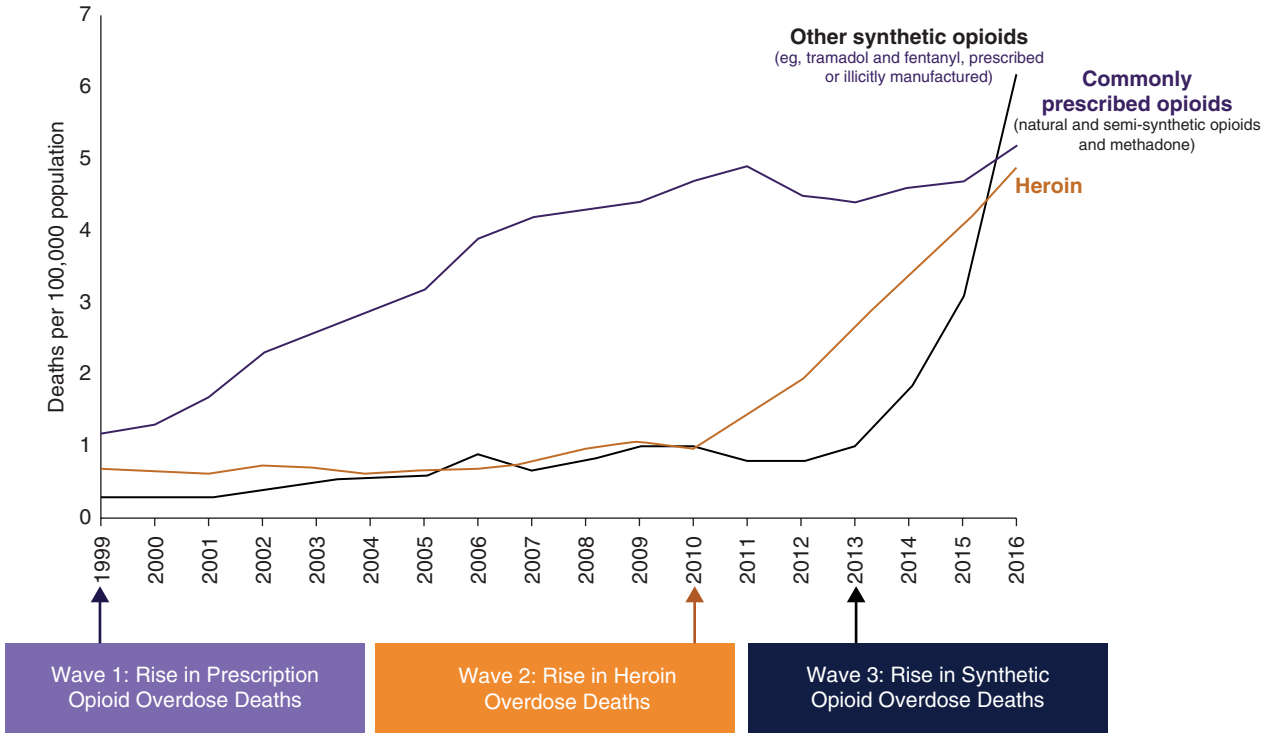

FIGURE 46-3 Three waves of the rise in opioid overdose deaths. (Reproduced from the Centers for Disease Control and Prevention.[40])

from an opioid overdose.[40] These alarming data are consistent with the terms *national crisis* and *public health emergency*.

In order to appreciate the statistics and trends documented by the CDC, it is helpful to understand the following definitions:

1. *Drug poisoning or overdose deaths* include deaths resulting from unintentional or intentional overdose of a drug, being given the wrong drug, taking a drug in error, or taking a drug inadvertently.
2. *Natural and semisynthetic opioids (prescription opioids)* include morphine, codeine, hydrocodone, and oxycodone.
3. *Synthetic opioids* other than methadone include fentanyl, fentanyl analogs, and tramadol.
4. Common *prescription opioids* include:
   - Hydrocodone (Vicodin), oxycodone (OxyContin, Percocet)
   - Oxymorphone (Opana)
   - Morphine (Kadian, Avinza)
   - Codeine
   - Fentanyl

How Did We Get Here? Milestones in the Opioid Crisis

The CDC describes the rise in opioid overdose deaths in the following three "waves": the 1990s—increased prescribing of opioids with overdose deaths involving prescription opioids; 2010—rapid increases in overdose deaths involving heroin; and 2013—significant increases in overdose deaths involving synthetic opioids, especially illicitly manufactured fentanyl (Fig. 46-3).[41] These three "waves" show that interconnected trends are driving America's opioid overdose epidemic (ie, a several-decades-long increase in deaths from prescription opioid overdoses and a recent surge in illicit opioid overdoses driven mainly by heroin and illegally made fentanyl).

In addition to these waves, it is helpful to be aware of the factors that contributed to the evolution of the opioid crisis in order to appreciate how initially good intentions about pain control led to adverse outcomes.[41]

In the mid-1800s, Civil War soldiers used opium tinctures as medicine to treat diarrhea and painful wounds. Considering the soldiers' great demand for pain medication, pharmaceutical companies began producing synthetic opioids in the late 1800s, and about that time, heroin also became available. Later, opium derivatives were recognized as addictive substances, and the United States restricted their import to medical uses only.[42] In the early 1900s, federal restrictions were placed on morphine (International Opium Convention) and, later, heroin (the Heroin Act), prohibiting the manufacturing, importation, and possession of heroin, even for medical purposes.[43] However, opium problems continued as veterans were given opioids for acute pain and many continued to use and misuse opioids after the acute crisis ended. By the 1970s, other opioids such as oxycodone and hydrocodone were developed and marketed for the relief of acute and cancer-related pain. However, a major change occurred in 1980, when a short letter entitled "Addiction Rare in Patients Treated

with Narcotics" was printed in the *New England Journal of Medicine*.[44] Citing their own results but not analyzing any data, the authors of this five-sentence letter stated that only 4 of 11,882 patients became addicted to opioids. Subsequently, this letter was cited over 600 times in support of using opioids to treat chronic pain, although no evidence supported that opinion.[44] Further support of this practice came from a paper describing the treatment of 38 patients with chronic pain who were treated with opioid pain relievers.[45] Despite the small number of patients in that study, the authors concluded that opioid pain relievers could be prescribed safely on a long-term basis. That study was also cited numerous times to support the expanded use of opioids for chronic noncancer pain. In 1995, Purdue Pharma introduced OxyContin (oxycodone hydrochloride), which was a "game changer" in that it was an extended-release formulation of oxycodone. Between 1996 and 2002, Purdue Pharma funded more than 20,000 pain-related educational programs and launched a campaign to encourage the long-term use of opioid pain relievers for chronic noncancer pain. Purdue provided financial support to organizations, and in turn, they advocated for aggressive identification of the treatment of pain, especially using opioid pain relievers. A number of physicians and pain organizations, including the World Health Organization, began advocating for more aggressive use of opioids for chronic pain.[46] Subsequently, between 1997 and 2002, prescriptions for OxyContin increased from 670,000 to 6.2 million.

Another major contributing factor to the prolific use of opioid pain relievers was the 1995 American Pain Society Presidential Address, "Pain as the Fifth Vital Sign."[47] Soon after Dr James N. Campbell delivered his Presidential Address, this concept was endorsed by the US Department of Veterans Affairs and The Joint Commission. Later, the American Academy of Pain Medicine and the American Pain Society issued a consensus statement endorsing opioid pain relievers for chronic noncancer pain. Subsequent legislation included state laws such as the Intractable Pain Act, which removed sanctions for physicians who prescribed long-term opioid therapy.[41] The other initiative that contributed to the opioid crisis was the development of the Hospital Consumer of Healthcare Providers and Systems Survey.[48] This 25-question patient satisfaction survey, endorsed by the Centers for Medicare and Medicaid Services and the Agency for Healthcare Research and Quality, incorporated three questions about pain control. That portion of the survey was intended to measure how well hospital providers managed patients with pain. Unfortunately, there was no evidence that equated the patients' perceptions with quality of care. In addition, hospitals encouraged patients to participate in this survey as full annual reimbursement was directly tied to a high score for pain management. Hence, hospital leadership ensured that the "fifth vital sign" was addressed regularly and that the patient's pain was kept to a minimum.

It should be noted that trends began to emerge regarding opioid abuse and investigations ensued. In 2007, the manufacturer of OxyContin and three senior executives pleaded guilty to federal criminal charges that they misled regulators, doctors, and patients about the risk of addiction associated with the drug.[49]

Pathophysiology

Current evidence shows that most illicit drugs exert their initial reinforcing effects by activating reward circuits in the brain similar to those described earlier in the section on alcohol addiction. Continued drug use impairs brain function by interfering with the capacity to exert self-control over drug-taking behaviors and rendering the brain more sensitive to stress and negative moods. Fig. 46-4 shows the stages of the addiction cycle (ie, binge and intoxication, withdrawal and negative effects, and preoccupation and anticipation or craving).[50]

Treatment of Opioid Use Disorder

Studies show that people with opioid use disorder who follow detoxification with complete abstinence are likely to relapse.[51] This setback is an anticipated step on the path to recovery, but it can also be life threatening. Therefore, an important way to support recovery and maintain abstinence from the addictive drug(s) is through the use of medications that reduce the negative effects of withdrawal and cravings without producing the euphoria that the original drug of abuse caused. The following are commonly used drugs to support that goal.

OPIOID AGONIST

Methadone is a synthetic *opioid agonist* that eliminates withdrawal symptoms and relieves drug cravings by acting on opioid receptors in the brain—the same receptors that other opioids such as heroin, morphine, and opioid pain medications activate. Although it occupies and activates opioid receptors, it does so more slowly, and in an opioid-dependent person, treatment doses do not produce euphoria.[52] It has been used successfully since 1947 to treat opioid use disorder. A comprehensive Cochrane review in 2009 compared methadone and psychosocial treatment to a placebo and psychosocial treatment. The authors found that methadone treatment was effective in reducing opioid use and that patients were 4.44 times more likely to stay in treatment than controls.[52] However, careful follow-up of these patients is needed because, in 2016, 3373 persons in the United States died from drug overdoses involving methadone.[2]

OPIOID PARTIAL AGONIST

Buprenorphine is a *partial opioid agonist*. Although it binds to opioid receptors, its activation of those receptors is less strong. Like methadone, it can reduce cravings and withdrawal symptoms in a person with an opioid use disorder without producing euphoria, and patients tolerate it well.[53]

OPIOID ANTAGONIST

Naltrexone is an *opioid antagonist* (ie, it works by blocking the activation of opioid receptors). Instead of controlling withdrawal and cravings, it treats opioid use disorder by preventing

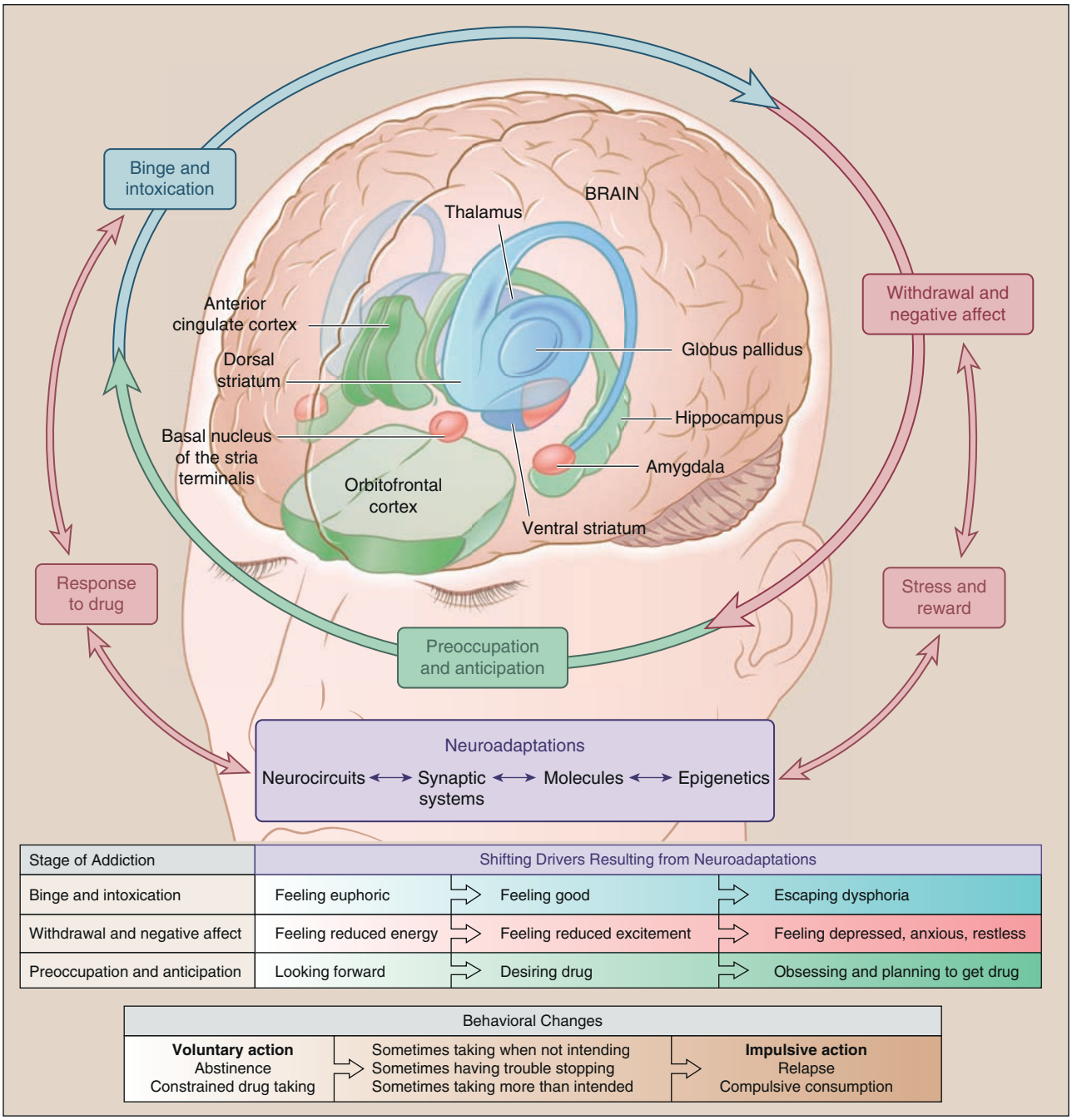

FIGURE 46-4 Stages in the addiction cycle.[50] (Reproduced with permission from Volkow ND, Koob GF, McLellan AT. Neurobiologic advances from the brain disease model of addiction. Review. *N Engl J Med.* 2016;28:374. Copyright © 2016 Massachusetts Medical Society. Reprinted with permission from Massachusetts Medical Society.)

any opioid drug from producing rewarding effects such as euphoria. Its use for ongoing opioid use disorder treatment has been somewhat limited because of poor adherence and tolerability by patients. An injectable, long-acting form of naltrexone (Vivitrol) has been recommended for those who do not have ready access to health care or who struggle with taking their medications regularly. This extended-release injectable form of naltrexone has promise for treating opioid and alcohol dependence but requires full detoxification to be effective.[52]

SYNTHETIC DRUG ABUSE

A particular challenge to clinicians is the identification and treatment of patients with synthetic drug abuse. First, in the United States, it is estimated that five new synthetic drugs are introduced monthly. Second, some of the drug components are available in various forms as over-the-counter medications and thus are easily accessible to the general population. Two of the common over-the-counter drugs that are abused include

TABLE 46-6: Synthetic Illicit Drugs[54]

Type	Common names	Effects
Synthetic cannabinoids	Spice, K2	Seizures, violent behavior, cerebral emboli
Synthetic cathinone	Bath salts, Bliss, Ivory Wave, Vanilla Sky	Insomnia, seizures, tachycardia
3,4-Methylenedioxy-methamphetamine (MDMA)	Ecstasy, Molly	Hallucinations, long-lasting depression
N-Bomb	Smiles, 25i	Hallucinations, seizures, rhabdomyolysis
Ketamine	Cat Valium, Jet, Special K	Hallucinations, amnesia
N-Methylamphetamine	Meth, Ice, Glass	Violent behavior, psychosis
Methadone	Saliva, Waver, Amidone	Cerebellar edema
Fentanyl	King Ivory, Jackpot, Apache	Gait impairment, neurologic deficits
Anabolic androgenic steroids	Pumpers, Gear, Juice	Increased aggression and cognitive impairment
Methcathinone	Ephedrone, Cat, Jeff	Hypertension, tachycardia, ataxia, dysarthria
Desomorphine	Krokodil, Russian Magic	Extreme skin ulcerations, gangrene (Fig. 46-5)

dextromethorphan (contained in cough syrup, antihistamines, and decongestants) and loperamide, an antidiarrheal. Third, the internet is replete with information about how to make synthetic drugs, and they are pharmacologically similar to traditional illicit drugs, such as cocaine, methamphetamines, and marijuana. Many of the drugs are addictive stimulants, and their repeated use causes long-term or irreversible damage to the dopaminergic, adrenergic, and serotonergic pathways in the brain. Some of the severe life-threatening adverse effects include seizures and cardiac dysrhythmias. In fact, illicit drugs should be considered in the differential diagnosis of new-onset seizures.[54] Table 46-6 provides information about synthetic drugs, including their common "street" names.

COMMONLY USED ILLICIT DRUGS

The following is a discussion of some of the drugs that have been found to be associated with fatal injuries.[55] In addition to the testing challenges for illicit drugs previously mentioned in this chapter, a positive drug test could represent an inactive drug metabolite resulting from past use (ie, not proximate to the injury time).

1. As of 2018, marijuana or Δ-9-tetrahydrocannabinol (THC) has been approved for recreational use in eight states. In addition, in 30 states including the District of Columbia, laws exist that legalize THC in some form. Because its recreational use is not fully approved in all states, THC remains the most commonly used illicit drug in the United States. It is made from the dried leaves of the *Cannabis sativa* or *Cannabis indica* plant and is highly lipophilic. Because of this property, it is stored in adipose tissue, liver, muscle, and the spleen and is redistributed into the bloodstream long after ingestion. Hence, marijuana can cause highly potent mental, physical, and toxic effects that are hard to control or predict.[56,57]
2. Cocaine is a powerfully predictive stimulant made from the leaves of the coca plant native to South America. Cocaine increases dopamine in the brain and prevents its recycling. This adverse process causes a buildup of dopamine between neurons, hence inhibiting normal communication. Tachycardia, hyperthermia, and dysrhythmias are among the most serious adverse effects of cocaine. Treatment for cocaine addiction (as well as for heroin) includes those medications discussed in the opioid section.[58]
3. Heroin is an opioid drug made from morphine, a natural substance taken from the seed pod of opium poppy plants. It is highly addictive and can cause serious organ damage, especially cardiac valvular disease. From 2013 to 2014, there were 10,574 heroin-related deaths, a 26% increase. Of note, the strongest risk factor for heroin addiction is opioid abuse.[59]
4. Hallucinogens are a very diverse category of drugs. Common hallucinogens include ayahuasca (hoasca, aya, dimitri), dimethyltryptamine (DMT), D-lysergic acid diethylamide (LSD), peyote (mescaline), 4-phosphoryloxy-*N,N*-dimethyltryptamine (psilocybin), and phencyclidine (PCP). Many of these substances have a wide variety of mind-altering effects including paranoia and psychosis. There is no specific medication to treat addictions to hallucinogens, and so treatment currently relies on behavioral and supportive methods.[60]

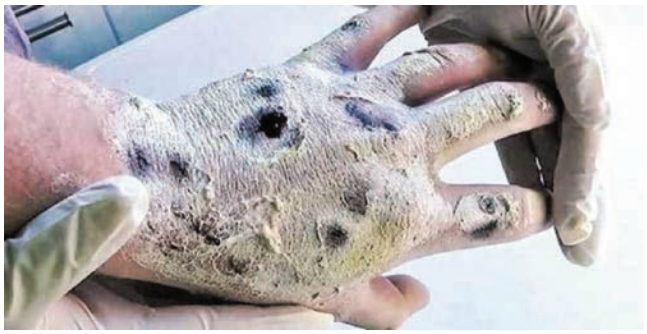

FIGURE 46-5 Hand with gangrenous changes secondary to Krokodil injections. (Reproduced with permission from Alves E, Grund J, Afonso C, et al. The harmful chemistry behind krokodil (desomorphine) synthesis and mechanisms of toxicity. *Forensic Sci Int.* 2015;249:207-213.)

DRUG AND ALCOHOL CRASH RISK

Driving under the influence of drugs is dangerous as side effects include slowed reaction time, impaired judgment, decreased coordination, drowsiness, and even hyperactivity. Any of these impairments can lead to reckless driving, automobile crashes, and/or injury to a pedestrian. According to the 2016 National Survey on Drug Use and Health, 11.8 million people admitted to driving under the influence of drugs.[61] Unlike BAC, there is no universal or single test for illicit drugs because they often contain multiple preparations and may be taken via several routes (intravenous, oral, inhalation) over different periods of time. Hence, the metabolism of these drugs may involve several organs and different kinetic orders. The National Highway Traffic Safety Administration conducted a study on the crash risk of alcohol-positive, drug-positive, and alcohol-plus-drug-positive drivers using a case-control design.[62] Drugs included over-the-counter, prescription, and illegal products. The study used data from crash-involved and non–crash-involved drivers from Virginia Beach, Virginia, over a 20-month period. They found that drivers who drank alcohol were 2.07 times more likely to crash than drivers who drank no alcohol. The drug odds ratios were adjusted for sex, age, race/ethnicity, and BAC, but the presence of drugs did not increase the risk of a crash. A potential explanation for this finding is that some drugs have a long half-life, and although the patient may test positive for the drug, the drug effects have long dissipated. Another study conducted in nine European Union countries showed the highest crash risk was for drivers with high alcohol concentrations, and those positive for marijuana were estimated to be at an elevated risk.[63] Similar results were noted from a study using the Fatality Analysis Reporting System in which the investigators found that during the period 1999 to 2010, the proportion of fatally injured drivers testing positive for narcotics and THC had tripled.[55] These studies and others suggest that driving under the influence of drugs and alcohol is a growing concern and a substantial cause of motor vehicle fatalities.

WHAT IS BEING DONE AND WHAT CAN WE DO?

According to a study examining opioid prescriptions by specialty, surgeons write 36.5% of outpatient prescriptions for opioids.[64] Hence, surgeons are uniquely positioned to effect change in the opioid crisis at the local, state, and national level (Table 46-7). A good starting point for surgeons is to

TABLE 46-7: What Is Being Done and What Can Be Done

Federal government	States	Health insurers	Health providers	Patients
Educating about pain management, addiction, overdose, and providing guidance on safe pain management	Implementing prescription drug monitoring programs	Reviewing programs to address improper prescribing and use of opioids	Using opioids only when benefits are likely to outweigh risks	Considering nonopioid options for pain management
Equipping states with resources to implement and evaluate safe prescribing practices	Implementing and evaluating programs to improve prescribing practices	Increasing coverage for treatments to reduce pain, especially nonopioid pain medicines	Starting with the lowest effective dose of immediate-release opioids	Storing prescription opioids in a secure place
Improving access to addiction treatment and recovery services	Using data to identify and address high-risk prescribing	Covering clinicians' time when they are conducting activities that improve quality and safety of pain management and addressing addiction	Reassessing benefits and risks when considering dose increases for opioids	Disposing of medications properly
Increasing access to overdose-reversing drugs	Enhancing the use of best practices for prescribing	Reducing barriers (eg, prior authorization) to the use of nonopioid pain relievers	Prescribing only the number of days that acute pain is expected to be severe enough to require opioids	Participating in local hospital and community efforts to address the opioid crisis
Tracking opioid-related trends and supporting research on pain management and addiction	Increasing access to medication-assisted treatment	Working closely with hospital systems to develop data bases to track high-risk patients	Using state-based prescription drug monitoring program to identify patients at risk of addiction or overdose	Getting help Substance Abuse and Mental Health Services Administration Helpline: 800-662-HELP (4357)[74]

become familiar with their local hospital and state initiatives. For example, health care executives are assembling teams to develop best practices for their institutions and then collaborating with other hospitals within their regions to ensure that patients do not go from one hospital to another in search of opioids. The physicians at Gundersen Health System have been taking steps toward decreasing opioid abuse for over 10 years. A pain medicine specialist ensured that Gundersen physicians understood the nuances of treating patients with chronic pain, and they implemented guidelines for prescribing narcotics. They formed a committee to address these issues, and much of their success is based on a continuous reassessment of progress as medication standards and patient populations change.[65] In addition to health care providers and hospital leaders strategizing about how to tackle the opioid crisis, the patient needs to be an integral part of the process. Gundersen Health System includes all stakeholders, including the patient, as members of its Chronic Pain Committee. The inclusion of the patient was also helpful in developing their Chronic Pain Registry. Another way that surgeons can contribute to diminishing the opioid problem is to set prescription guidelines for postoperative pain. Orthopedic surgeons at the Mayo Clinic developed opioid prescription guidelines for patients undergoing several common orthopedic procedures. The researchers compared the well-matched groups in the preguideline (1822 patients) and postguideline (751 patients) periods. They found that the median prescription dropped from about 95 pills of 5-mg oxycodone to approximately 50 pills, an overall 48% decrease.[66]

The CDC has published recommendations for prescribing opioids for chronic pain outside of active cancer, palliative, and end-of-life care (Table 46-8). Many of the best practices at hospitals are based on these guidelines, and the reader is referred to the CDC website for the complete publication.[67] Some of these guidelines are supported from studies on the use of opioid pain relievers prescribed for postoperative pain. For example, Waljee et al[68] reviewed the opioid prescriptions given to 296,452 adults who underwent common upper extremity surgical procedures. They found that patients who previously received opioids were more likely to fill a postoperative opioid prescription and renew prescriptions more often.[68] Another study, conducted among the acute care hospitals in Ontario, Canada, found that taking benzodiazepines preoperatively was a risk factor for the prolonged use of opioid pain relievers.[69]

Suggestions made by the ACS include the following: (1) promote the use of prescription drug monitoring programs; (2) encourage research and training developed in collaboration with specialists in pain management for safe prescribing; (3) recognize and address issues specific to veterans; (4) change the direct relationship between provider reimbursement and patient pain control; and (5) support patient safety legislation.[70] Additionally, the ACS published an educational tool for patients regarding opioids so that the patient can understand the importance of his or her role in this addiction crisis.[71] Table 46-9 offers suggestions for multimodal pain management. Other leading organizations, such as the US Food and Drug Administration (the agency that ensures that drugs used by the US public are safe), published

TABLE 46-8: Centers for Disease Control and Prevention Recommendations for Prescribing Opioids for Chronic Pain Outside of Active Cancer and Palliative and End-of-Life Care[65]

1. Prescribe nonpharmacologic and nonopioid therapy for chronic pain.
2. Establish treatment goals with the patient.
3. Discuss with the patients the risks and realistic benefits of opioids.
4. Prescribe immediate-release opioids, not extended-release.
5. Initially prescribe the lowest effective dose.
6. Prescribe as per the expected duration of pain.
7. Evaluate benefits and harms regularly.
8. Evaluate continuation of therapy at appropriate intervals.
9. Review patient's history of controlled substance prescriptions.
10. When prescribing opioids for chronic pain, consider urine drug testing annually.
11. Avoid prescribing opioids and benzodiazepines concurrently.
12. Offer evidence-based therapy for patients with opioid use disorder.

TABLE 46-9: Multimodal Pain Management[75,76]

Preoperative	Intraoperative or after injury	Postoperative or after injury
Appropriate goal setting to target "functional" and not "pain free"	Regional anesthesia	Targeted opioid utilization
Identification of high-risk individuals	Neuraxial anesthesia	Continuous regional nerve blocks
		Nonnarcotic pain management strategies: Gabapentinoids Acetaminophen NSAIDs
		Early rehabilitation
		Cognitive therapy for pain management (guided imagery, relaxation techniques, music therapy)
		Management of psychological disease impact on pain manifestation

NSAIDs, nonsteroidal anti-inflammatory drugs.

a special report outlining their plans to deal with this opioid misuse crisis.[72] All of these initiatives presented convincing evidence that legislative efforts are needed to counter the addiction crisis. An example is H.R. 6, the Substance Use-Disorder Prevention That Promotes Opioid Recovery and Treatment for Patients and Communities Act, passed on June 22, 2018, with substantial bipartisan support. This legislation will help to advance treatment and recovery initiatives, improve prevention, protect communities, and bolster efforts to curb the production of deadly illicit synthetic drugs.

Finally, there is some relatively good news to report. From 2012 to 2017, the overall national opioid prescribing rate declined. In 2017, this rate was the lowest it had been in 10 years, at 58.7 prescriptions per 100 persons, about 191 million total opioid prescriptions. Overall, these numbers are still too high, and some counties had rates that were seven times higher than the overall average, indicating that progress is being made but there is still much work to be done.[73]

SUMMARY

The growing number of people with substance abuse problems and/or addiction qualifies as a national crisis and a major public health problem. Trauma surgeons will frequently encounter these patients in a variety of settings and are uniquely positioned to achieve a positive change toward this crisis. Early screening, assessment, and treatment are important steps in the treatment of alcohol use disorder and the prevention of the high mortality disease, delirium tremens. Underage drinking requires special tactics relative to screening and treatment, especially since years of potential life lost are greatest in this group. Deaths due to drug overdoses continue to rise, and multifaceted strategies such as prescription drug monitoring programs, prescription guidelines, pharmaceutical treatments, counseling, legislative efforts, and patient engagement are essential components of the resolution. Clinical outcomes and basic science research are also necessary components of the solution. The surgeon's responsibility includes becoming aware of the resources on substance abuse and minimizing the patient's pain while addressing the imperative to avoid improper prescribing.

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Social Violence

Amy M. Kwok • James W. Davis

KEY POINTS

- The Centers for Disease Control and Prevention estimate that 12.7 million women and men have been affected by intimate partner violence.
- The World Health Organization reported that 35% of woman worldwide have experienced physical or sexual violence.
- The HITS (Hurt, Insult, Threaten, Scream) questionnaire has been validated for screening for intimate partner violence.
- Older people with cognitive impairments, physical disabilities, or mental health problems are at higher risk for elder abuse.
- Fractures in places besides the hip, vertebrae, or wrists and spiral fractures are suspicious for elder abuse.
- The Elder Abuse Suspicion Index is a six-question screening tool validated in the ambulatory care setting.
- The United States has the highest rate of youth homicides out of all the industrialized nations.
- A gun in the home is associated with a 40-fold greater chance of killing a family member or acquaintance.
- Individuals who participate in gangs are more likely to be involved in fights, take weapons to school, and use drugs or alcohol at school.

INTRODUCTION

Trauma care is, by definition, tertiary prevention or treatment. Understanding the root causes of the injury may aid in better comprehension and treatment of the trauma victim. Improvements in trauma care should incorporate consideration of the psychosocial aspects of such injuries as well as the impact on the larger health care system.

Patients who are victims of family and community violence may have relatively simple traumatic injuries but often have complex psychosocial issues that affect their response to injury. Simply treating the injuries and not intervening with the underlying causes makes recidivism of these patients the likely end result. Early detection and efforts at prevention of interpersonal violence must be part of the trauma center's prevention program.

Violence may be defined as "the intentional use of physical force against another person or against oneself, which results in or has a high likelihood of resulting in injury or death."[1] Its frequency is documented by the following facts:

- Suicides and homicides are the second and third leading causes of death among children and youth under the age of 21.[2]
- Overall, injury and violence remain the leading cause of death for persons age 1 to 44.[2]
- One person dies every 4 minutes as a result of intentional injury.[3]
- Intimate partner violence is the most common cause of injury to women in the United States, causing nearly 2 million injuries a year.[4-7]

The literature is replete with studies identifying risk factors for interpersonal violence.[2,8-10] Despite this potential knowledge base, physicians are often hesitant to use this information.[11-13] Early recognition and intervention may prevent future incidents and decrease rates of complications such as posttraumatic stress disorder.[13-15] The statistics on death and injury from intentional violence are only the tip of the iceberg. The cost to society of violent behavior also includes the price of legal battles, incarceration, and the economic effects on the health care system as a whole, as well as the psychological stress to victims and the families of victims.[3,4]

The purpose of this chapter is to provide practicing surgeons with some basic information on intentional violence with a focus on intimate partner and community violence, so that they may be better providers of care for these patients with special needs.

INTIMATE PARTNER VIOLENCE

Intimate partner violence (IPV), also known as domestic violence, refers to those acts of interpersonal violence resulting in physical or psychological injury to members of the same family or household or to intimate acquaintances in heterosexual or same-sex relationships. According to the Centers for Disease Control and Prevention (CDC), IPV constitutes physical, emotional, or psychological harm in the form of physical or sexual violence, stalking, and/or psychological aggression.[2] Other reports have acknowledged that child[16,17] and elder abuse may also be included in the spectrum of domestic violence.[18] IPV and elder abuse will be covered here, and child abuse will be addressed in the chapter on pediatric trauma.

IPV is not new; it has long plagued mankind. A 15th-century scholar argued that a man should beat his wife "not in rage but out of charity and concern for her soul."[19] English Common Law established "the Rule of Thumb" in 1895, stating that a husband could not beat his wife with a switch greater in diameter than the width of his thumb.[20] The legal right of men to beat their wives was not abolished until 1871 in the United States.[21] Until the 1970s, assaults on wives were considered misdemeanors, when an equal assault against a stranger would have been considered a felony.[22] In 1992, it became a requirement of The Joint Commission that all accredited hospitals have policies and procedures in their emergency departments and ambulatory care facilities for identifying, treating, and referring victims of abuse.[22]

Public awareness campaigns such as No More (nomore.org) have heightened awareness on this issue. Multiple medical professional organizations recognize IPV as a public health problem not limited to specialties. The responsibility resides with all the treating physicians and surgeons to diagnose and aid in providing resources to the victim.[23-25]

Incidence and Prevalence

The actual incidence of IPV is unknown because definitions and estimates vary widely. However, the estimated statistics are alarming. Currently, the CDC estimates more than 12.7 million women and men have been affected by IPV.[26] IPV affects 24 people every minute.[26]

The National Intimate Partner and Sexual Violence Survey from the CDC and National Institute of Justice and Department of Defense estimates that more than 27% of women and 11% of men in the United States experienced physical violence, rape, and/or stalking by an intimate partner in their lifetime. The rates of IPV were highest for persons age 18 to 24 years.[27] Women are more likely to experience rape, stalking, and physical violence, whereas men are more likely to experience physical violence alone.[28] Nearly 1 in 5 women (19.3%) and 1 in 59 men (1.7%) have been raped in the United States.[26] In addition, 51.5% of female rape victims reported being raped by their intimate partner.[28]

These rates are similar to those reported by the World Health Organization (WHO). They found worldwide that 35% of women have experienced physical or sexual violence, with 30% involving IPV.[29] In effect, one out of three women around the world has been beaten, coerced into sex, or otherwise abused during her lifetime.[30]

One report from the National Institute of Justice and the CDC estimated that 1.5 million women are physically assaulted or raped by an intimate partner in the United States annually.[31] The National Violence Against Women Survey also estimated that 5.3 million IPV incidents against women occur annually, with more than 550,000 requiring medical attention, loss of 8 million days of paid work, and loss of 5.6 million days of household productivity.[24] The estimated cost of this violence is more than $8.3 billion.[32,33]

The incidence of IPV in the lesbian, gay, bisexual, transgender, and queer (LGBTQ) community is significantly understudied. However, prevalence rates are estimated to be similar or even higher than in the heterosexual community. The CDC found that LGBTQ persons experienced higher rates of IPV, with a 61% lifetime prevalence in bisexual women compared to 44% of lesbian and 35% of heterosexual women. IPV rates were lower in men when compared to women but still affected 37% of bisexual men, 26% of gay men, and 29% of heterosexual men. Gay and bisexual men also had higher rates of IPV than heterosexual men, with 25% of gay men and 37% of bisexual men experiencing some form of IPV, including rape, physical violence, and/or stalking.[34] Rates of sexual violence were higher in the LGBTQ community compared to heterosexual men and women. Almost half of bisexual men (47%) and 40% of gay men have been victims of sexual violence, compared to 21% of heterosexual men. Bisexual survivors are more likely to experience physical and sexual violence, whereas transgender victims are more likely to experience physical violence. Gay men were more likely to experience IPV in the forms of intimidation and harassment, and the homicide rate is highest among gay men (76.2%) in the LGBTQ community.[34,35]

The *cumulative lifetime prevalence* of domestic violence of women seen in the emergency department is 40% to 60%, and 12% to 25% of visits by women to the emergency department are from domestic violence.[31,36] IPV is responsible for 30% of trauma center admissions for women.[37] Severe physical violence is experienced by 24.3% of women by an intimate partner, including being slammed against something or assaulted with fists or hard objects.[28]

A WHO multicountry study and US studies on domestic violence found that those who reported IPV had a wide range of physical problems, were twice as likely to experience depression and alcohol abuse, and had a 4.5-fold increased risk of suicide attempts.[29,38] In some regions, women were 1.5 times more likely to acquire HIV.[29,30]

Victims are more likely to have long-term health and mental problems such as depression, posttraumatic stress disorder, and drug and alcohol abuse than nonvictims.[24,26] Physical abuse occurs as well in 7% to 20% of pregnancies. IPV continues to be a significant risk factor for violence in pregnant patients, specifically adolescent pregnant patients.[39,40]

In the United States, 17% of all homicides involved IPV. Of these cases, 77% of the victims were female, and 62% of

the female homicide victims were murdered by their intimate partner.[41,42] This averages to four to five women murdered every day by their partner.[43] The Violence Policy Center found that of all murder–suicides, 72% of cases involved an intimate partner and 94% of the victims were female.[41] Worldwide, 38.6% of female homicides were committed by an intimate partner.[38] The majority of female homicides (52%) are committed with guns.[42]

Women are 3.6 times more likely to be shot by a spouse or ex–significant other than by a stranger.[44] Women are six times more likely to be killed if a gun is in the house in IPV homicides.[42] In addition, marital violence is a significant predictor of physical child abuse. In one study, the probability of child battering increased from 5% with one act of marital violence to near certainty with 50 or more acts of wife battering.[45]

Child battering occurs in 59% of the homes with spousal abuse and may be as high as 77% with severe wife abuse.[46,47] The victim is frequently demoralized and is so lacking in self-esteem that it is difficult to leave the situation.[48] Additionally, the threats of retaliation, injury to children or pets, and death increase the victim's fears and helplessness. Indeed, the risk of physical violence actually increases after moving out.[49]

Identifying Intimate Partner Violence

A three-phase cycle has been described for battering. The first phase involves a gradual buildup of tension and then escalation with name calling, intimidation, and mild physical abuse. The second phase involves an uncontrollable discharge, with verbal and physical attack and frequently injury. In the third phase, the abuser apologizes and asks for forgiveness and promises that it will not recur. With repeated cycles, the first phase increases in length, the violence may become more acute, and the third phase decreases. Again, the victim is frequently demoralized and is so lacking in self-esteem that it is difficult to leave the situation.[48]

The second cycle for victims of domestic violence involves the failure to make the diagnosis even after the patient arrives in the emergency department. In a study of battered victims presenting to the emergency department, 23% had presented 6 to 10 times previously and 20% had 11 prior emergency department visits.[50] In 40% of cases with known domestic violence, physicians made no response at all, and in 92% of cases, physicians made no referral for the abuse.[51] Victims of domestic violence view physicians as least effective in helping them compared to women's shelters, social services, clergy, police, and lawyers.[51]

There are some characteristics of injury type and location in domestic violence. Injuries tend to be central (face, head, neck, breast, and abdomen) versus the more peripheral injuries seen in accidents. In one study of injury locations, the head, face, and neck were significantly more frequently injured in victims of domestic violence than in accident victims (P <.001), and an unwitnessed injury is a significant marker for IPV.[52,53]

Because victims of IPV may be fearful or ashamed, nontraumatic complaints predominate as reasons for the physician visit. In one study, 78.4% of IPV victims had medical complaints and 72% were never identified as victims of abuse.[54] Even after a violent episode, only 23% had injury-related complaints. Domestic violence victims rarely volunteer information; after battering, only 13% of victims either told staff or were asked about the possibility of abuse.[55] However, domestic violence victims were not offended when asked about abuse in a nonjudgmental manner.[56] Further, the failure of health care providers to ask about domestic violence may be perceived as evidence of a lack of concern and add to feelings of entrapment and helplessness.[57]

In 2015, a Cochrane review found that routine screening increased identification of women experiencing IPV by nearly threefold.[58] Joseph et al[59] found the prevalence of IPV to be 5.7 cases per 1000 trauma center discharges, with an overall mortality of 6%. He concluded that a mandatory IPV screening should be done in all trauma patients and that a national intervention focus is a necessity.[59] The use of a specific screening tool for domestic violence has been shown to be more effective than routine social services evaluation.[56] Screening for IPV should be approached in a quiet environment, separate from the partner, with a nonjudgmental opening such as, "Because we see a lot of patients coping with abusive relationships, we now ask about IPV routinely."

There are a number of screening tools that have been validated for IPV screening. The Hurt, Insult, Threaten, Scream (HITS) screening tool questionnaire consists of four questions. Answering one or more questions affirmatively indicates a positive screen.[60]

1. Does your partner physically **h**urt you?
2. Does your partner **i**nsult you or talk down to you fairly often?
3. Does your partner **t**hreaten you with harm?
4. Does your partner **s**cream or curse at you fairly often?

The US Preventive Services Task Force (USPSTF) recently updated their recommendations to screen asymptomatic women of childbearing age for IPV and provide referral services to those who screen positive. The USPSTF found adequate evidence that screening can identify abuse, and other studies have found that effective interventions can reduce violence.[61,62] The American College of Surgeons, American College of Physicians, American College of Obstetricians and Gynecologists, American Academy of Orthopedic Surgeons, American Medical Association, Eastern Association for the Surgery of Trauma, and the Western Trauma Association all recommend screening for IPV.[18,23-25]

Treatment and Referral, Documentation, and Reporting

Once the diagnosis of IPV has been made, the responsibilities are to treat the patient, reassure the patient about safety, and make the appropriate referral to social services. It is important to carefully document the injuries in the medical record. Regardless of the legal requirement to report IPV, failure to do so may have lethal consequences. In several studies of women murdered by their spouses or boyfriends, the majority

had accessed the health care system within a year or two of their deaths, most for injury, and even when the diagnosis was made, there was no referral for the abuse.[63,64]

Summary of Intimate Partner Violence

IPV is common and commonly undiagnosed. As recommended in the position statement by the American College of Surgeons, all trauma patients should routinely be screened for IPV.[25] The use of a specific screening tool without the partner present is strongly recommended. Trauma centers and trauma surgeons should have ongoing education about domestic violence to improve the recognition and management of this epidemic problem.

ELDER ABUSE

Elder (>60 years old) abuse is a single or repeated act, or lack of appropriate action, occurring within any relationship where there is an expectation of trust, that causes harm or distress to an older person; elder abuse can involve physical abuse, psychological or emotional abuse, financial or material abuse, sexual abuse, and neglect.[65] Elder abuse and neglect is suspected to be greatly underreported, which may stem from lack of professional awareness, victim reluctance, or even cognitive impairments in reporting.[66,67] One in 10 elders in 2008 reported some form of elder abuse, including emotional, sexual, or physical abuse or potential neglect from caregivers, friends, and/or family.[67]

Health care professionals tend to underestimate elder abuse, and when detected, approximately 50% of elder abuse cases are reported.[66]

Incidence and Prevalence

Similar to IPV, the exact incidence of elder abuse is unknown because it is largely underreported. Recent reports estimate a 7.6% to 10% incidence of elder abuse.[67-69] In one study, 88.5% of abused elders experienced psychological abuse, whereas 19.7% reported physical abuse and 29.5% experienced neglect.[70]

Older people with cognitive impairments, physical disabilities, or mental health problems were found to be at higher risk of being abused.[71-73] Alarmingly, 34% to 62% of caregivers were found to abuse elders with dementia.[70,72,74]

Elder abuse is found to be most common in women and persons older than 75 years of age. A family member is identified as being the abuser in 90% of the reported cases.[75-77] Cultural differences and language barriers in vulnerable groups may hinder identification of abuse, especially in Latino, Asian, and Pacific Islander elders.[78,79]

Worldwide, the incidence of elder abuse is predicted to increase as the global population is expected to more than double to approximately 1.2 billion in 2025.[80] A WHO review found a 4% to 6% combined rate of elder abuse in developed countries.[81] The highest prevalence rate of elder abuse is found to occur in more developed countries rather than developing countries.[77]

The rates of elder abuse in nursing homes are alarming. One study found that over half of the nursing home staff reported physical violence, neglect, and mental abuse of their elderly patients. The majority of these cases involved neglect.[82] Previous studies from 1999 to 2001 found that one-third of nursing homes in the United States were cited for abuse violations.[83]

There is a significant association between elder abuse and risk of morbidities such as depression, increased rates of hospitalization, increased emergency service use, and mortality.[73,84-87] One study found that elder abuse, especially neglect, was associated with significantly increased risk of mortality.[85] One prospective cohort study found a threefold increase in mortality for persons age 65 years and older who were referred to Adult Protective Services for mistreatment and a 1.7-fold increase for those with self-neglect.[88]

Diagnosis

Some of the findings of elder abuse are similar to other forms of abuse (eg, skin injury and tears and bruising). Fractures in the elderly are unfortunately common, but fractures in places besides the hip, vertebrae, or wrists, or spiral fractures may be suspicious of abuse. Malnutrition and dehydration may be signs of neglect in a dependent elder.[89,90]

As with IPV, there are a number of screening tools that may be used for assessment of elder neglect and/or abuse. The Elder Abuse Suspicion Index is a six-question screening tool validated in the ambulatory care setting.[91] The first five questions are answered by the patient and the last question by the physician. Any affirmative response to questions 2 to 6 is considered a positive screen.

1. Have you relied on people for any of the following: bathing, dressing, shopping, banking, or meals?
2. Has anyone prevented you from getting food, clothes, medication, glasses, hearing aids, or medical care or from being with people you wanted to be with?
3. Have you been upset because someone talked to you in a way that made you feel shamed or threatened?
4. Has anyone tried to force you to sign papers or to use your money against your will?
5. Has anyone made you afraid, touched you in ways that you did not want, or hurt you physically?
6. Elder abuse may be associated with findings such as poor eye contact, withdrawn nature, malnourishment, hygiene issues, cuts, bruises, inappropriate clothing, or medication compliance issues. Did you notice any of these today or in the past 12 months?

The American Medical Association recommends elder abuse screening directed at both the elder patient and caregiver.[77] Questions directed toward the caregiver include the following: Are your elder's needs more than you are able to handle? Are you worried that you might hit your elder? Have you hit your elder? When screening the elder patient, questions asked include: Has anybody hurt you? Are you afraid of anybody? Is anyone taking or using your money without

your permission? The responses might lead to further questions, and additional information may need to be obtained.

Treatment and Referral

When elder abuse is suspected, as in IPV, medical treatment comes first. If there is reason to believe that an elder is suffering from abuse, neglect, or exploitation, referral for possible elder abuse should occur. Reporting elder abuse is required in most states. Documentation of injuries on the medical record should be meticulous.

Summary of Elder Abuse

Elder abuse is less common than IPV (7.6%–10%) but continues to trend upward. It is associated with significant morbidity and mortality. Seniors with risk factors should undergo brief screening questions, as listed earlier. If elder abuse or mistreatment is suspected based on answers to the screening questions or physical findings, further assessment is indicated. If there is reason to believe that there is abuse or neglect, reporting and careful documentation are essential.

COMMUNITY VIOLENCE

History of the Problem

The issue of interpersonal violence as a public health problem has gained significant national and worldwide attention. Worldwide, violence causes more than 1.3 million deaths each year and is the fourth leading cause of death for people age 15 to 44 years.[92] The United States significantly exceeds all other high-income nations in violent death.[92] The United States also has the highest youth homicide rate out of all the industrialized nations.[93,94] In 2013, the National Research Council and Institute of Medicine found that US males and females had shorter life expectancies than other economically developed countries, with homicide and suicide accounting for about a quarter of the years of life lost for US men.[94]

Gunshot suicide deaths account for 51% of violent deaths and occur 17 times more frequently in the United States than in Great Britain.[95-98] Young men in the United States age 16 to 19 years are five times more likely and males age 20 to 24 years are seven times more likely to die from violence.[94] Worldwide, more than 500 young adults are murdered daily.[92]

Over 2.4 million emergency department visits are due to violent injury annually in the United States.[99] In 2010, the total cost associated with fatal and nonfatal injuries due to gun violence was estimated to be at least $174 billion.[100] Most violence occurs not between strangers but among intimate partners, friends, and acquaintances.[101-103]

Risk Factors

Injuries resulting from violence occur when people have learned to use force to "solve" problems. Most age and population groups in the United States are actually at relatively low risk. A number of risk factors have been identified for community violence and related injury, including age, socioeconomic status, race, access to firearms, alcohol and other drug use, gang involvement, exposure to domestic violence and child abuse, and media violence. The most significant risk factors are male gender, being an adolescent or young adult, and low socioeconomic status.[92,95] Men younger than 65 years old were fourfold more likely to be murdered than women or people over 65 years of age.

Homicide is the second leading cause of death for men between 15 and 24 years old and the leading cause in African American men between 15 and 24 years old in the United States.[94,95] Sixty-six percent of all homicides involve the use of firearms.[94]

Increased concerns about violence have led to more Americans obtaining and carrying firearms for protection,[104] but this actually leads to increased risk of firearm injury or death. It is estimated that 40% to 50% of American households have guns.[105,106] A gun in the home is associated with a 40-fold greater chance of killing a family member or acquaintance (homicide, suicide, or accidental) than an intruder in self-protection.[107]

Alcohol and drug use are also associated with an increased risk of violence.[108] In one study, individuals who began drinking before age 14 years were significantly more likely to have been in an alcohol-related fight than those who began drinking after age 21.[109] Alcohol is involved in one-half to two-thirds of homicides, half of serious assaults, and more than 25% of all rapes.[110] Approximately 36.7% of homicides involved alcohol, where the majority of victims had a blood alcohol content of 0.08 g/dL or higher.[95]

Gang Violence

A national survey of youth gangs reported more than 30,000 gangs with 850,000 members in 2012, an increase of 20% from the previous year.[111] More than 85% of cities with a population greater than 50,000 have street gangs. Gangs were involved in approximately 16% of all homicides.[111] Individuals who participate in gangs are more likely to be involved in fights, take weapons to school, and use drugs or alcohol at school.[112,113]

The media's glamorized version of violence is seen by millions daily. The use of violence is an acceptable, if not the preferred, method of dealing with conflict and used by heroes as well as villains. A longitudinal study of young adults demonstrated that those with childhood high violence television viewing were significantly more likely to have grabbed, pushed, or shoved their spouses and that the men in this group were three times more likely to be convicted of crimes.[114]

Surgeon's Role

Physicians are in a key position to impact the lives of victims of interpersonal violence.[115] The timing and proximity of our contact with the patient are crucial components of the formula for successful intervention.

There are barriers to intervening, but despite these barriers, physicians can and should address the issue of intentional violence. Many professional organizations have published position statements and/or guidelines. Most guidelines advocate for the routine incorporation of questions regarding safety and exposure to interpersonal violence into the history. Alpert et al[116] have made a list of age-specific screening questions that can be easily covered. The value of this routine questioning is partly about identifying individual victims but also represents an opportunity to improve overall health care. Routine screening provides physicians with opportunities to express concern for the patient and to express the antiviolence message.[117] Indeed, when patients were given an opportunity to tell physicians how to intervene in family conflict, the overwhelming majority thought physicians should ask.[13]

SUMMARY

Violence in our society represents a complex, multifaceted problem. In many ways, violence is like a chronic disease. This "disease" is related to both lifestyle and environment. Sims et al[118] at Henry Ford Hospital found that trauma is a recurrent disease, with 44% having recurrent injury, and the overall 5-year mortality rate of 20% clearly demonstrates that this "disease" should not be ignored.

The enormity of this problem requires an ordered, disciplined approach such as that developed by the public health community to address infectious diseases.[1] This approach should include event surveillance, epidemiologic analysis, intervention design and evaluation, and a focus on prevention. Our educational efforts should be based on clear competencies,[119] and interventions should be evidence based.

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48

Pediatric Trauma

Denis D. Bensard

KEY POINTS

- Injuries are the leading cause of death in the United States for persons age 1 to 19 years and the fifth leading cause of death for newborns and infants age less than 1 year.
- The Broselow emergency tape or Pediatric Advanced Weight Prediction in the Emergency Room (PAWPER) provide estimated weight of a child using length-based estimation without (Broselow) or with (PAWPER) adjustment for body habitus.
- The Shock Index, Pediatric Age-Adjusted (SIPA) identifies injured children with high injury severity, need for blood transfusion within the first 24 hours, and increased in-hospital mortality.
- Criteria to activate a massive transfusion protocol in injured children may include 20 mL/kg of packed red blood cells in the first hour or anticipated blood loss greater than one-half blood volume in 12 hours or greater than one blood volume in 24 hours.
- The motor component of the Glasgow Coma Scale (GCS) alone identifies a child with a serious traumatic brain injury.
- In general, the presence of free fluid on a focused assessment with sonography for trauma (FAST) examination is an indication for computed tomography imaging in a stable injured child.
- Spinal cord injury without radiologic abnormality is particularly hazardous because children may only present with transient neurologic findings, but then go on to develop neurologic deficits hours to days after the initial injury.
- Children with two or more rib fractures are more likely to require a thoracostomy tube, thoracotomy, thoracoscopy, and/or laparotomy.
- For a child with an injury to a solid organ in the abdomen, the success of nonoperative management approaches 95%.
- Suspicion of child abuse should be raised if there is a discrepancy between the history and the extent of injury, if there are explanations that do not fit, or if the amount of time between the incident and presentation for medical attention is significant.

INTRODUCTION

Early in the last century, little distinction was made in the care of children from adults. William E. Ladd, a general surgeon, observed this firsthand as he cared for severely burned children in the Halifax disaster of 1917. "Dr. Ladd was distressed by the quality of surgical care offered to these small patients and was determined to improve it."[1] He and Dr. Robert E. Gross in the mid-20th century pioneered a new field of surgery specific to children, illuminating features of children that merited special attention. They and others recognized that although the care of children shared some similarities to that of the adult, there were distinctly different anatomic and physiologic characteristics and surgical conditions in children that made them unique. In 1962, the first pediatric trauma unit opened at the Kings County Hospital Center in Brooklyn. Yet, the lack of dedicated care and clinical protocols persisted in the United States until the early 1970s. The first designated pediatric shock trauma unit in the United States opened at the Johns Hopkins Children's Center and became incorporated into the statewide University of Maryland Institute of Emergency Medical Systems.[2] In the years that followed, the American College of Surgeons (ACS) Committee on Trauma (COT) established the treatment of pediatric injury as an important component of all regional trauma centers, integrated pediatric trauma into the Advanced Trauma Life Support (ATLS) course, and established minimum requirements for pediatric trauma center designation. As of 2018, the number of ACS designated Level I/II pediatric centers is 109, over twice the number of

43 in 2010. The importance of designated trauma centers for children cannot be overemphasized. Myers et al[3] found that injured children treated at a Level I pediatric trauma center were more likely to survive when compared to those treated at a Level I trauma center. Similarly Notrica et al[4] reported that mortality (20.6/100,000) in states without verified pediatric trauma centers exceeded mortality in the states with Level I/II verified pediatric trauma centers (15.3/100,000) or Level I verified pediatric trauma centers only (12.9/100,000).

C. Everett Koop, a former pediatric surgeon and US Surgeon General, declared, "If a disease were killing our children at the rate unintentional injuries are, the public would be outraged and demand that this killer be stopped." Worldwide, every year, nearly a million children are killed. Nonfatal injuries affect the lives of between 10 million and 30 million more. Each year in the United States, more than 12,000 children die from unintentional injuries, and nearly a million children are treated in emergency departments (EDs) for nonfatal injuries.[5] The cost of unintentional injury can be measured in dollars, permanent disability, and potential lost years in productivity or life. The estimated cost of all injury in the United States in 2013 was $671 billion. Of those costs, which include total estimated lifetime medical and work-loss costs, $6.8 billion and $22.4 billion were a result of fatal and nonfatal injury, respectively, in children age 0 to 14 years. Sadly, these figures do not include the cost associated with future work lost and quality of life, which would be most pronounced in the very young.[6,7]

Injuries are the leading cause of death in the United States for persons age 1 to 19 years and the fifth leading cause of death for newborns and infants age less than 1 year (Fig. 48-1). The death rate for males is almost two times the rate for females. Pediatric trauma patients demonstrate a lower mortality than adults. Children demonstrate differing injury patterns by age, and patterns differ from those observed in adults. By adolescence, however, patterns of injury mirror those seen in adults. The mechanisms of injury affecting each age group are largely influenced by developmental stage. Two peaks in deaths are observed: the toddler years (age 1–4 years) and adolescence (age 15–18 years). Infants are at highest risk of death from inflicted trauma. Strikingly, intentional injury from abusive trauma accounts for more deaths in infants than unintentional injury. Toddlers often suffer injury from falls due to their immature motor and cognitive skills coupled with their higher center of gravity. From ages 5 to 9 years, pedestrian injuries are the leading cause of death, reflecting greater mobility and, thus, risk. Motor vehicle occupant injuries predominate in the 10- to 14-year-old age group. Adolescent death is most often due to a motor vehicle crash as the driver or occupant. This age group is particularly at risk of serious injury or death due to greater autonomy, poor judgment, and alcohol or drug use. Injury death rates for teenagers are significantly higher than for all other age groups. Although penetrating injury is relatively uncommon in young children, it becomes more prevalent in adolescence. Homicide is the second leading cause of death in those older than age 15 years, but in African American youths, it is the leading cause of death. Firearm homicides account for nearly 14.5% of years of potential life lost before age 65 among black males compared to 1.2% among white males.[8]

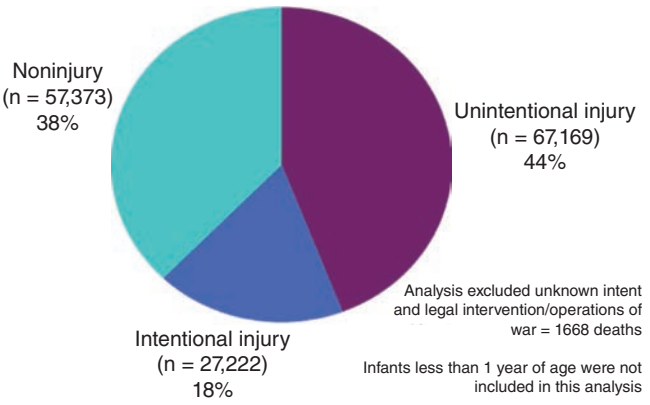

FIGURE 48-1 Cause of death by injury status and intent among children age 1 to 19 years, United States, 2000 to 2005.

An estimated 17.4 million children do not have access to a pediatric trauma center within 60 minutes.[9] In fact, 90% of injured children receive care in facilities other than pediatric trauma centers.[10] Dictated largely by geography and pediatric specialist limitations, most injured children are initially cared for by adult specialists with variable pediatric-specific training or resources. Therefore, it is incumbent on all providers of trauma care to have knowledge in the unique needs of children, recognizing that *children are not just little adults*.

INITIAL ASSESSMENT

The initial evaluation of children does not differ from that in adults. The assessment should proceed in a systematic approach based on the principles of the ATLS program published by the ACS-COT. There are unique characteristics of children that differ from adults that must be considered during the assessment (Table 48-1).

The most common cause of preventable prehospital deaths is airway obstruction or brain apnea.[11] Examining prehospital death and preventability, Oliver et al[12] found that more than 50% of patients suffered a potentially preventable death. Of interest, 76% suffered a neurologic injury, and 19% had evidence of airway obstruction on autopsy. A bystander was present in half the cases; most called emergency medical services, but fewer than half attempted a lifesaving intervention prior to their arrival.[12] Early death (<6 hours) from trauma most often results from lethal traumatic brain injury (TBI) or hemorrhage.[11,13] The Hartford Consensus Conference concluded that the leading cause of preventable death is bleeding and that simple interventions that "stop the bleed" can be lifesaving.[14] Prehospital cardiopulmonary resuscitation (CPR) portends a poor prognosis in the absence of easily reversible airway obstruction, tension pneumothorax, or hemorrhage. In 169 injured children requiring prehospital CPR, 28 survived to discharge, including 16 with full neurologic recovery. Of patients who had prehospital CPR that

TABLE 48-1: Age-Based Vital Signs from Infancy to Adolescence

Age group (years)	Weight range (kg)	Heart rate (bpm)	Blood pressure (mm Hg)	Respiratory rate (breaths/min)	Urinary output (mL/kg/h)
Infant (0–1)	0–10	<160	>60	<60	2.0
Toddler (1–3)	10–14	<150	>70	<40	1.5
Preschool (3–5)	14–18	<140	>75	<35	1.0
School age (6–12)	18–36	<120	>80	<30	1.0
Adolescent (>12)	36–70	<100	>90	<30	0.5

Source: Reproduced with permission from American College of Surgeons. *ATLS Student Manual.* 9th ed. Chicago, IL: American College of Surgeons; 2012:257.

continued upon arrival to the ED, all died.[15] Fallat[16] has suggested that if a child has arrested, resuscitation time exceeds 30 minutes, and the nearest facility is more than 30 minutes away, transport may be futile. Despite current evidence that suggests either death or a poor outcome is inevitable, many children undergoing prehospital CPR will still be transported to the nearest hospital and, if pediatric capability is limited, will be transferred to the pediatric trauma center despite little chance of survival.

Additional factors at presentation alert the physician to a child with life-threatening injury. Low Glasgow Coma Scale (GCS) score, prehospital intubation, and shock have traditionally been used to identify high-risk patients. Falcone et al[17] performed an analysis of 29 commonly used trauma team activation criteria including the six minimum criteria of the ACS-COT for highest trauma team activation. The most predictive of requiring a high-level resource were gunshot wound to the abdomen, blood given before arrival, traumatic arrest, tachycardia/poor perfusion, pre-trauma center IV fluid volume resuscitation greater than 40 mL/kg, age-appropriate hypotension, airway compromise, and GCS score less than 8. Nearly one-half of the injured children who met these criteria needed at least one high-level resource during their ED resuscitation; nearly 25% required two to three resources. The criteria used for trauma team activation not only identify children at risk of life-threatening injury, but also identify those who will likely require one or more emergent interventions.[17] It is in this high-risk group that adequate preparation, skills, and age-appropriate equipment are essential. Team failure is most likely to occur in high-acuity pediatric trauma activations, those without prior notification, or penetrating injury.[18]

In young children (<40 kg), dosing of medications and fluids is weight based. The Broselow emergency tape and Pediatric Advanced Weight Prediction in the Emergency Room (PAWPER) tape are simple tools that provide estimated weight using length-based estimation without (Broselow) or with (PAWPER) adjustment for body habitus. These tools can guide providers in recommended fluid administration, drug dosing, and normal ranges of vital signs by age and guide appropriate size of necessary equipment. However, these tools have limitations. The length-estimated weight tapes appear to accurately estimate ideal body weight but may incorrectly estimate weight in the under- or overweight child. Underdosing of medications in the overweight child or overdosing in the underweight child can occur.[19] Adjusted dosing or redosing of medications may become necessary in some children. Despite their limitations, these tools may be helpful to providers who treat injured children infrequently.

The Primary Survey

AIRWAY

Ensuring an adequate airway is the first priority in trauma resuscitation. In analysis of preventable deaths in the state of Montana, Esposito et al[20] found that most of the preventable deaths occurred in the subgroup of children less than or equal to 14 years of age. Inappropriate trauma care was highest in the ED phase of care and primarily resulted from errors in management of the airway.[20] Similar findings were reported in a subsequent study by Sanddal et al[21] of preventable pediatric deaths in Utah. Among patients surviving to a hospital, the preventable death rate was 11%. Issues with care were again mainly in management of the airway, fluid resuscitation, chest injury diagnosis, and management.[21]

Infants and young children have a prominent occiput that tilts the head forward when supine; in combination with a short neck, floppy epiglottis, and increased lymphoid tissue, upper airway obstruction is common, particularly in the unconscious child. A simple maneuver, the jaw thrust or chin lift, can relieve obstruction caused by the tongue, facilitate bag-mask ventilation, and allow time to prepare for endotracheal intubation (ETI). Children have an increased metabolic rate and oxygen consumption. They have diminished functional residual capacity. Together, this results in a propensity for rapid oxygen desaturation. With little intrapulmonary oxygen reserve, they become hypoxic more precipitously than adults. Supplemental oxygen or oxygen with bag-mask ventilation helps to minimize this characteristic of young children.

After simple support of the airway and administration of oxygen, the need for intubation should be assessed. In spontaneously breathing children, basic airway maneuvers can establish airway patency and restore adequate respirations. Unconsciousness, combativeness, declining GCS, and shock are the most common indications for a definitive airway in an injured child and identify a seriously injured subset of children with an increased risk of death.[22] Nasotracheal

intubation is rarely performed in children. The child is preoxygenated, and rapid sequence intubation (RSI) drugs are administered. ETI generally proceeds orally with direct visualization of the glottis and in-line stabilization of the neck. A neck injury should always be assumed to be present. Standard direct laryngoscopy (DL) is usually performed.

Video laryngoscopy (VL; Glidescope) to facilitate successful intubation in the difficult airway has increased since the introduction of the device. In a meta-analysis of 14 studies by Sun et al,[23] although VL improved glottis visualization in pediatric patients, an increase in time to intubation and number of failures was observed. The use of VL in trauma remains unproven. In a simulation of pediatric trauma patients with cervical spine immobilization, 23 children age less than 10 years underwent DL and VL. The use of VL resulted in a less optimal view of the glottis and required 21 seconds, compared to 7 seconds for DL.[24] These data suggest that DL is not inferior to VL when performed by those experienced in ETI, and caution is warranted before application of VL in the injured child.

The narrowest point of the airway is the cricoid cartilage not at the level of the vocal cords. Therefore, selection of the correct size endotracheal tube (ETT) is essential to successful intubation. Inappropriate size and multiple attempts can lead to laryngospasm and inability to maintain the airway. Oxygen desaturation and laryngospasm are two of the most common complications observed in attempted ETI placement in children. The third most common complication is right mainstem intubation due to the relative short length of the airway. The physician should have some idea of the appropriate depth of the ETT. A general rule of thumb is three times the diameter of the ETT should be the measurement at the lip indicated by the markings on the ETT.

RSI is a commonly used adjunct in trauma emergency airway management.[25] RSI maximizes the chances for success while minimizing the adverse physiologic effects of intubation.[26] The most feared complication of the technique is the inability to intubate and subsequently ventilate the patient. RSI requires familiarity with airway management techniques, sedation agents, neuromuscular blocking agents, supplemental agents, and postintubation management techniques.[27] Children exhibit an exaggerated vagal response to hypoxia, succinylcholine, laryngoscopy, and intubation.[28] To attenuate the physiologic effects of ETI, especially in children with a TBI, preinduction medications (eg, lidocaine, fentanyl, atropine) may be administered.[29] A sedative induction agent (eg, thiopental, ketamine, propofol, fentanyl, or etomidate) is given, followed by a rapidly acting neuromuscular blocking agent (eg, succinylcholine), resulting in unconsciousness and motor paralysis.

Cricoid pressure is applied, and intubation follows. Overcompression can easily occur due to the compressibility of the pediatric airway. Once the ETT is placed, a colorimetric device that measures exhaled or end-tidal carbon dioxide ($EtCO_2$) is connected. A color change from purple ($EtCO_2$ <0.5%) to yellow ($EtCO_2$ >4%), symmetric chest rise, and auscultation of bilateral breath sounds suggest successful intubation. In the child suffering circulatory arrest, a color change may not be observed until the circulation is restored.

If ETI is unsuccessful, there are several rescue maneuvers available. The first and perhaps easiest is the placement of a laryngeal mask airway (LMA) designed for use during anesthesia. The LMA is easy to insert and requires less training than ETI but does not protect against aspiration. Successful use after failed emergency ETI has been reported.[30] When basic interventions or ETI fail, particularly in the paralyzed patient, an emergent surgical airway becomes necessary. Cricothyroidotomy, needle cricothyroidotomy, or translaryngeal jet ventilation offers the last options in securing the airway. Although there is ample literature concerning these techniques in adults, their use in children has been limited, emphasizing the importance of expertise in emergency pediatric airway management.[31]

BREATHING

The first step in assessment of breathing is the rate and quality of respirations. The infant may breathe normally up to 60 times per minute, young children up to 40 times per minute, and older children 20 to 30 times per minute. Young children primarily use the diaphragm to breathe; they have underdeveloped musculature, rendering them prone to muscle fatigue and less likely to be able to maintain a sustained high respiratory rate. Ineffective movement of air may result from poor effort, parenchymal injury, pneumothorax, or airway obstruction. Signs of respiratory distress include stridor, chest retractions, paradoxical motion of the diaphragm, and grunting. When these signs are present, support of respiration becomes necessary.

Pulse oximetry is a noninvasive measure of hemoglobin saturation that correlates well with arterial Pao_2 and is a measure of blood oxygen saturation. At readings of less than 80%, correlation with Pao_2 is lost and severe hypoxemia may be present. Oximetry can provide indirect evidence of ventilation, but objective evaluation requires measurement of arterial Pco_2 or $EtCO_2$. If it is determined that ventilation is inadequate, it becomes necessary to give supplement breaths or assume all ventilation. Breaths should be delivered every 2 to 3 seconds with adequate chest rise. Full ventilation will require adequate sedation and often paralysis. For mechanical ventilation, a tidal volume of 6 to 8 mL/kg is used to reduce the risk of barotrauma, and the FiO_2 is titrated to below 60% to minimize the harmful effects of hyperoxemia. Measurement of end-tidal capnography is useful to guide the rate of assisted breaths. Significant hypocarbia or hypercarbia is deleterious in the brain-injured patient due to the effects on cerebral circulation. Current guidelines suggest that an $EtCO_2$ of 35 to 40 mm Hg is optimal. In a study of children with severe TBI (GCS <8) from Level I pediatric trauma centers, children with an admission $Paco_2$ between 36 and 45 mm Hg had greater discharge survival compared with those with both admission hypocarbia ($Paco_2$ ≤35 mm Hg) or hypercarbia ($Paco_2$ ≥46 mm Hg).[32] Excessive ventilation with low $EtCO_2$ can be minimized with capnography.[33] Inadequate

ventilation or high peak inspiratory pressure (>30 mm Hg) with ventilation may be due to unrecognized pneumothorax. In this setting, evidence of pneumothorax requires rapid decompression with chest tube placement. The Broselow tape or palpation of the intercostal space can guide selection of the appropriate size chest tube. In the absence of pneumothorax or hemothorax, significant pulmonary contusion or aspiration may account for the impaired ventilation, but the treatment remains largely supportive.

The child's chest is highly compliant and poorly muscularized, and the ribs are less ossified, leaving the intrathoracic contents susceptible to injury. External signs of trauma may be absent or minimal. The mediastinum is mobile, and pneumothorax can rapidly deteriorate to tension pneumothorax, especially if positive-pressure ventilation is required. Rib fractures are uncommon in blunt-injured children for these reasons. Rib fractures suggest a significant blunt force injury. Kessel et al[34] found that children with rib fracture were more likely than adults with rib fracture to suffer brain injury, hemothorax/pneumothorax, and spleen and liver injury.

Ventilation may also be compromised by inadequate gastric decompression. The distended stomach interferes with diaphragmatic excursion, especially in the very young (Fig. 48-2). Therefore, an orogastric tube should be placed in any injured child requiring prolonged bag-mask ventilation or ETI. A nasogastric tube should not be placed in any child with signs of basilar skull fracture or facial trauma, so a policy of orogastric tube placement simplifies decision making and reduces the risk of malplacement.

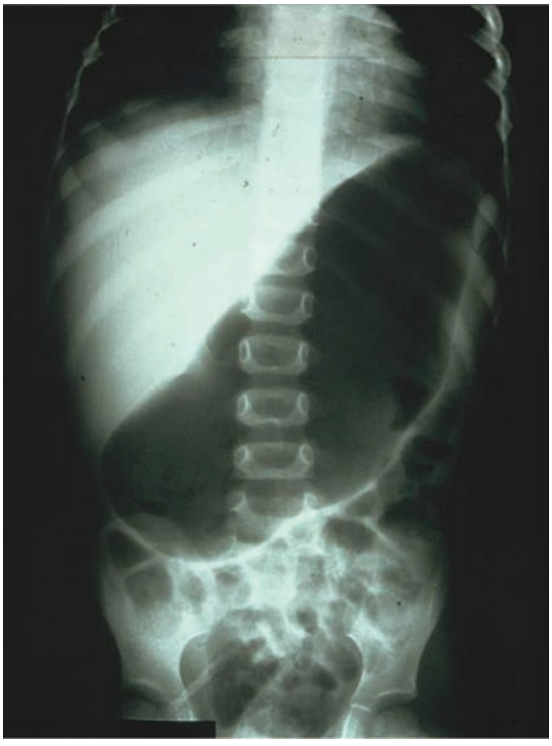

FIGURE 48-2 Acute gastric dilatation in an injured child.

CIRCULATION

Infants and young children have a limited ability to meet increased cardiac output demand. Stroke volume is generally fixed. Thus, an increase in cardiac output requires an increase in heart rate. Compensation for the loss of intravascular volume results in an increase in both heart rate and systemic vascular resistance with shunting of blood to vital organs. In a classic experiment of hemorrhagic shock, puppies were subjected to continuous hemorrhage calculated at 1% blood volume per minute. The response to hemorrhage was gradual and compensated until nearly a third of the blood volume was lost. With continued blood loss, the fall in mean arterial pressure and cardiac output accelerated, followed by death.[35] Children have robust compensatory mechanisms for mild to moderate shock. However, once a critical threshold is passed, decompensation occurs rapidly, emphasizing the need for early recognition.

Assessment of the circulation begins with the examination of the level of consciousness, skin color, heart rate, blood pressure, and peripheral pulses. An adequate blood pressure may be falsely misleading. The assessment of capillary refill allows an estimate of the degree of vasoconstriction and compensation present. Delayed (>2 seconds) capillary refill in the absence of hypothermia suggests that blood pressure is maintained by an increase in vascular resistance and that the child is significantly hypovolemic. A rapid assessment should be made for any external signs of bleeding that can be controlled by direct pressure or a tourniquet. The efficacy of tourniquets to control blood loss in military and civilian adults is well described; however, less is known regarding their efficacy in children. In a large study of prehospital interventions for children injured in Afghanistan, Sokol et al[36] found that tourniquet or hemostatic dressing application for extremity vascular injury or amputation was the most effective intervention and safe. Based on the military experience in both adults and children, if there is major hemorrhage from an extremity or amputation, a tourniquet should be applied. The pelvis should be examined for stability. If a pelvic fracture is suspected in conjunction with signs of shock, the pelvis should be wrapped in a sheet to provide compression that can reduce ongoing bleeding.

Signs of decompensated shock in children include altered mental status, mottled or cyanotic skin, cool pale extremities, weak peripheral pulses, and delayed capillary refill. Review of age-based normative values for heart rate and blood pressure demonstrate changes that occur from infancy to adolescence (Table 48-1). In general, respiratory rate and heart rate are normally higher, whereas blood pressure is lower in the young, but by age 12 years, they approach the normative values of the adult. Mortality is increased in children suffering traumatic injury and hypotension. Gunst reported 2% mortality in hypotensive injured children.[37] Hypotension, even brief episodes, substantially increases mortality in children suffering from TBI.[38]

Injured children generally suffer from one of three types of shock: hemorrhagic, cardiogenic, or distributive. Hemorrhagic and distributive shock are the most common;

TABLE 48-2: Shock Index, Pediatric Age-Adjusted (SIPA) Used to Rapidly Identify Traumatic Shock in an Injured Child

Vital signs by age with calculated cutoff values				SIPA
Age (years)	**HR (bpm)**	**SBP (mm Hg)**		**SIPA cutoff value**
1–3	70–100	90–110		1.2
4–6	65–110	90–110	HR/SBP = SIPA	1.2
7–12	60–100	100–120		1.0
>12	55–90	100–135		0.9

Note: SIPA is easily calculated by dividing heart rate (HR) by systolic blood pressure (SBP). In younger children, the cutoff value is higher due to higher normal HR and lower normal blood pressure. With increasing age, these values approximate those of adults, and thus, after age 12, the SIPA cutoff (0.9) is equivalent to the adult shock index cutoff (0.9).

Source: Adapted with permission from Nordin A, Coleman A, Shi J, et al. Validation of the age-adjusted shock index using pediatric trauma quality improvement program data. *J Pediatr Surg.* 2018;53(1):130-135. Copyright © Elsevier.

cardiogenic shock is rarely the cause of primary traumatic shock in children and, if present, suggests that death is imminent. Although hemorrhage should always be assumed as the source of traumatic shock, it must be remembered that severe central nervous system (CNS) injury in young children can produce hypotension in the absence of hemorrhage.[39] A simple calculation, the Shock Index (SI; heart rate/systolic blood pressure [SBP]), can quantify the degree of shock, stratify the risk of death, and direct triage of injured patients to a trauma center. Cannon et al[40] demonstrated in adults that the risk of death is 16% when the SI is greater than 0.9 compared to a 6% risk of death when the SI is less than 0.9. An increase in SI from the field to the ED nearly doubles mortality.[40] The applicability of an uncorrected adult SI to children is limited given the relatively high values of normal heart rate and low values of normal of SBP in children relative to adults, leading to a falsely elevated SI (>0.9). To correct for age-based normative values, we examined the Shock Index, Pediatric Age-Adjusted (SIPA) in injured children (Table 48-2). We found that, like observations in adults, once SI is adjusted for age, SIPA identifies injured children with high injury severity, blood transfusion within the first 24 hours, high-grade solid organ injury requiring blood transfusion, and increased in-hospital mortality.[41] In a review of the Trauma Quality Improvement Program (TQIP) database of 22,344 children age 1 to 16 years, Nordin et al[42] demonstrated that SIPA was a strong predictor of transfusion needs, injury severity, admission to the intensive care unit (ICU), ventilator use, and mortality for both blunt and penetrating trauma. A rapid SIPA calculation can be derived by simply dividing heart rate by SBP regardless of age-based norms. In any child following injury, a value greater than 1.2 suggests traumatic shock and the likely need for one or more interventions.

Among the most difficult challenges in the care of children is vascular access. Smaller veins coupled with increased subcutaneous fat in children make both palpation and visualization of veins more difficult. Peripheral vein collapse due to hypovolemia, vasoconstriction from hypothermia, failed attempts with resultant hematoma or bruising, and extremity fractures all further complicate attempts at successful peripheral intravenous (IV) access. Ideally, access is obtained with placement of two peripheral IV catheters in the upper extremities. The Broselow emergency tape guides selection of an appropriate IV catheter size. What may appear to be an inadequate catheter size in an adult may be suitable for rapid fluid infusion in a child. The saphenous vein at the ankle is an excellent option for peripheral IV access if attempts in the upper extremity fail.

Pediatric Advanced Life Support (PALS) and ATLS now recommend placement of an intraosseous (IO) line if IV access cannot be established within three attempts or 90 seconds, whichever is sooner.[43] In children, IO placement is generally first attempted in the proximal tibia. Other potential sites include the distal tibia, the proximal humerus, and the distal femur. IO avoids many of the problems associated with central venous access. IO placement with a battery-operated device (eg, EZ IO) is rapid, requires little training, and provides reliable access in which most drugs, fluids, and blood achieve the central circulation in seconds (Fig. 48-3). Complications are low, but IO placement should be avoided in a fractured extremity due to the risk of fluid extravasation and resultant compartment syndrome.

Historically, if peripheral venous access failed, then central venous access using the Seldinger technique based on anatomic landmarks was attempted. This practice has largely been abandoned but remains an option when all other measures fail. Usually attempts are made to access the femoral vein first followed by the internal jugular vein or subclavian vein. Ultrasound is readily available in most EDs, is easy to learn, and permits visualization of the femoral vein or internal jugular vein, facilitating central venous catheter placement. In a randomized trial of children undergoing planned central venous access, ultrasound reduced the number of cannulation attempts with a greater success rate relative to the landmark approach.[44] Regardless, central venous access carries the risk of serious complications and delay and, because of the suboptimal conditions in the ED, requires early catheter removal to avoid central line–associated bloodstream infection. Therefore, central venous access should only be considered if peripheral IV and IO access have failed.

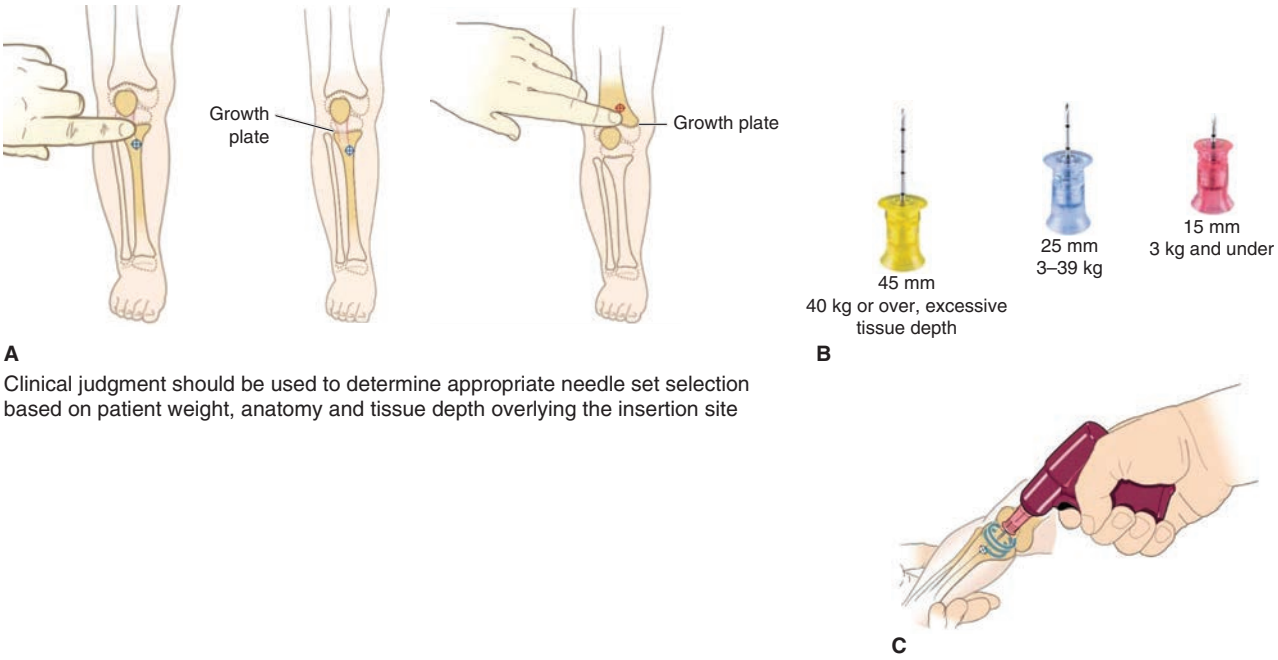

FIGURE 48-3 (**A**) Preferred sites of IO placement in infants and children. (**B**) Recommended weight based EZ IO needle length guide. (**C**) Depiction of correct technique in a child. (Images courtesy of Teleflex Incorporated. © 2020 Teleflex Incorporated. All rights reserved.)

Once IV access is obtained, a 20 mL/kg bolus of 0.9% normal saline (NS) or lactated Ringer's solution is administered. Due to the risk of non–anion gap metabolic acidosis, lactated Ringer's is favored over 0.9% NS. In patients with a TBI, early administration of hypertonic saline (3% or 7.5%) rapidly restores blood volume and may be beneficial in treatment. Two recent randomized trials in patients older than age 15 years, however, failed to demonstrate any benefit of prehospital hypertonic saline over 0.9% NS in patients with either hypovolemic shock (SBP <70 mm Hg or SBP 71–90 mm Hg and heart rate >108 bpm)[45] or TBI (GCS <8).[46] Colloid solutions for the resuscitation of traumatic shock fail to demonstrate consistent benefit. A meta-analysis by the Cochrane Review found no evidence from randomized controlled trials that resuscitation with colloids reduces risk of death compared to resuscitation with crystalloids in patients with trauma. Moreover, the use of the colloid, hydroxyethyl starch, may increase mortality.[47] For now it appears that 0.9% NS or lactated Ringer's should remain the initial resuscitative fluid unless blood transfusion becomes necessary. If the patient does not respond or only transiently responds, a second 20 mL/kg bolus of isotonic crystalloid may be given, but contemporary damage control resuscitation emphasizes early blood and product administration in lieu of repeated crystalloid administration. Blood samples for arterial blood gas, lactate, hemoglobin, coagulation (thromboelastography or prothrombin time/partial thromboplastin time), and type and cross should be obtained.

Under- or overresuscitation with fluids is likely deleterious in trauma patients. The optimal amount of fluid needed in the first 24 hours to adequately resuscitate injured patients remains unclear. However, most patients dying of shock or TBI die within 24 hours from either hemorrhage or lethal brain injury. Most deaths occur despite ongoing resuscitation.[48] In adults, investigators from the Glue Grant found that ventilator days, ICU and hospital length of stay (LOS), risk of acute respiratory distress syndrome, multiple organ failure, bloodstream and surgical site infections, and abdominal/extremity compartment syndromes were all increased in a dose-dependent fashion with aggressive crystalloid administration.[49] In contrast, we reported that high-volume early resuscitation in children, defined as greater than 60 mL/kg/24 h, did not demonstrate an increased risk of acute respiratory distress syndrome, multiple organ failure, abdominal compartment syndrome, or bloodstream infections, but like the Glue Grant investigators, we observed an increase in ventilator days, ICU LOS, anemia, and thrombocytopenia.[50] Edwards et al[51] reported similar findings to both studies in a cohort of pediatric combat causalities. They observed that the adverse effects of crystalloid became most pronounced when greater than 150 mL/kg was administered in the first 24 hours, but administration of 50 to 150 mL/kg was not associated with increased mortality. Not surprisingly, underresuscitation (<50 mL/kg/24 h) led to increased mortality, but this may simply reflect survival bias (ie, children who died prior to hemorrhage control and ongoing fluid resuscitation). Injured children should be judiciously resuscitated, but adequate fluid should be given to ensure adequate tissue oxygenation and cerebral perfusion regardless of the amount of crystalloid needed in the early resuscitative phase. While adult studies have suggested benefit to permissive hypotension, Hughes et al[52] warns that there is no evidence to support permissive hypotension strategies in pediatrics.

In the absence of a sustained response to 40 mL/kg of isotonic crystalloid fluid bolus, ATLS advises the administration of blood. Blood component administration is the resuscitative

fluid of choice in the bleeding patient until hemorrhage control is achieved. The decision to begin blood transfusion must be balanced against its potential delayed adverse effects,[53,54] including a potential negative effect on outcome in children suffering TBI.[55] Injured children requiring early blood administration represent a seriously injured subset. Early blood transfusion (<6 hours) is associated with up to 50% mortality, in stark contrast to the low mortality in those who do not receive blood or undergo delayed blood transfusion.[54] Interestingly, most children who received early blood in this series died of lethal injury to the brain rather than hemorrhage. It may be that the brain-injured child in shock and without hemorrhage might benefit from an alternate resuscitative strategy utilizing vasopressors, as suggested by Di Gennaro et al.[56]

An ongoing requirement for blood suggests noncompressible intracavitary hemorrhage, and massive transfusion becomes more likely. A focused ultrasound examination for pericardial and intra-abdominal fluid (focused assessment with sonography for trauma [FAST]) demonstrating free fluid can assist in directing the child to emergent laparotomy to achieve hemorrhage control.[57] Like traumatic shock, the need for massive transfusion is associated with an increased risk of death in children. Neff et al[58] found in combat-injured children that once a threshold of 40 mL/kg/24 h of blood products is given, critically injured children are more than 2.5 times more likely to suffer early in-hospital death.[58] Similar findings are reported in civilian pediatric victims.[54] Current evidence in adults suggests that in this setting blood components should be transfused in 1:1:1 or 1:2:1 ratio (packed red blood cells to fresh frozen plasma to platelets) until hemorrhage is controlled and there is no evidence of ongoing bleeding.[59] In contrast, in a study of pediatric casualties in Iraq and Afghanistan, Edwards et al[51] found that balanced component resuscitation did not improve outcomes and was associated with higher mortality when all transfused patients were considered. Further studies are needed to reconcile this apparent difference in children from adults.[52,60]

The decision to transfuse blood is based on vital signs, injury patterns, anticipated major bleeding, or risk of continued bleeding after surgical control. In a prospective study of injured adults, Sisak et al[61] found that 9% required early blood transfusion and nearly half had activation of a massive transfusion protocol (MTP). Injury pattern, vital signs, lactate, and anticipated major bleeding were the best predictors of MTP in this cohort, which demonstrated a high rate of emergency surgery (86%) and mortality (14%).[61] Less is known in children. In children, most protocols recommend that MTP be activated in children with signs of bleeding. Criteria may include greater than 20 mL/kg of packed red blood cells in the first hour or anticipated blood loss of greater than one-half blood volume in 12 hours or greater than one blood volume in 24 hours. The Assessment of Blood Consumption (ABC) score consists of four variables (heart rate >120 bpm, SBP <90 mm Hg, positive FAST, and penetrating torso injury) each assigned 1 point. Validated in adults, a score of greater than 2 triggers an MTP and correctly classifies most bleeding adult patients in need of MTP.[62] The ABC score may not be applicable in children due to differing normal values of heart rate and BP with age. In a pilot study using a modified ABC score using SIPA (ABC-S), the authors found that an ABC score of greater than 2 poorly predicted the need for MT, but an ABC-S score of 1 or greater improved prediction of MTP, with sensitivity increased from 29% to 65% and comparable specificity.[63] Although it is evident that MTP results in earlier delivery of blood components, it remains unclear whether it improves outcome in children.[60,64,65]

In the child who presents with signs of life and loses vital signs while in the ED, resuscitative thoracotomy may be considered. First, reversible causes of pulseless electrically activity such as tension pneumothorax or hypovolemia should be excluded. Unfortunately, the outcome in children with blunt injuries who have a cardiac arrest and ED thoracotomy is dismal.[66] The results are more favorable in adolescent patients suffering penetrating injury and witnessed cardiac arrest, particularly those suffering stab wound to the heart.[67] ED thoracotomy may be warranted in children suffering penetrating torso injury who lose vital signs in the ED or have short transport time with prehospital CPR less than 15 minutes.

DISABILITY

The child's level of consciousness, pupillary examination, and neurologic exam define disability. The aim of the neurologic assessment is to identify severe head injury or spinal cord injury that may require urgent neurosurgical intervention. A rapid assessment can be determined using the Alert/Pain/Verbal/Unconscious (APVU) scale. APVU appears to correlate well with the GCS: alert, GCS of 15 (interquartile range [IQR], 15); pain, GCS of 13 (IQR, 12–14); verbal, GCS of 8 (IQR, 7–9), and unconscious, GCS of 3 (IQR, 3).[68] GCS is more typically used and shows good performance in preverbal children compared to older children with blunt head injury. Receiver operator characteristic curves demonstrate good correlation of GCS with brain injury confirmed by computed tomography (CT) or requiring acute intervention.[69] Concerns remain that calculation of the GCS can be difficult in children, is compromised by the need for ETI, and suffers significant interrater variability. Ultimately, the goal is to ascertain quickly which children require an urgent CT scan and perhaps neurosurgical intervention. This has led others to examine the use of abbreviated GCS scales or one of the three components of the GCS to rapidly identify at-risk children.[70] We found that the motor component of the GCS alone identifies children with serious TBI and the elimination of the eye and verbal components did not adversely affect the performance of the tool.[71]

If after assessment by one of the previously described tools the child is felt to have a serious brain injury, the eye examination should be repeated to determine if there is evidence of impending transtenorial brain herniation suggested by unilateral dilation of a pupil. This or rapid progressive neurologic deterioration implies critical intracranial hypertension. If present, temporizing measures such as brief hyperventilation ($EtCO_2$ 25–30 mm Hg), 3% hypertonic saline IV bolus (1–6 mL/kg), or IV mannitol (0.25–1 mg/kg) should be considered until definitive neurosurgical care

is available.[72] Mannitol should not be administered to a child with hypotension or shock. Unlike mannitol, hypertonic saline, a volume expander, can be used in the setting of hypotension. The child is examined for evidence of a spinal cord injury. Respiratory failure and/or shock can be the first signs of injury to the spinal cord but are only attributed to this once all other potential causes are excluded. Symmetric flaccid paralysis of the extremities indicates spinal cord injury, and spinal immobilization should continue.

EXPOSURE

Children are susceptible to hypothermia, more so than adults. Hypothermia can lead to arrhythmias, abnormal coagulation, and metabolic acidosis, with the latter two drivers in the lethal triad of trauma death. Review of the National Trauma Data Bank (NTDB) demonstrated that of those trauma patients in whom temperature was measured, 2% had a core temperature as low as 32°C. Comparison of core temperature versus mortality revealed that as temperature decreased, the mortality rate increased, reaching approximately 39% at 36°C.[73]

The trauma room temperature should be maintained at greater than 80°F. Wet clothes should be removed, the child covered with warm blankets, and exposures for procedures minimized. All IV fluids and blood products should be warmed using fluid warmers. Active external rewarming with a convective air blanket (eg, Bair Hugger) should be initiated when the core temperature falls below 36°C. Despite the importance of exposure and environmental control during pediatric trauma resuscitation, compliance is poor. In children with TBI or requiring ETI where the duration of exposure is prolonged, Kelleher et al[74] found hypothermia to be common. Sundberg et al[75] remind us that a hypothermic patient is 9.2 times more likely to die than a normothermic patient when adjusting for seasonal variation. Therefore, children who are prone to hypothermia or arrive hypothermic must be rewarmed and kept warm. There is no role for hypothermia in the acute setting of trauma despite reports of hypothermia used in cardiac arrest or management of TBI.

THE SECONDARY ASSESSMENT

The secondary assessment begins only after all elements of the primary assessment are satisfied. Periodic review of vital signs, pulse oximetry, $EtCO_2$ capnography, fluid administration, and GCS alerts the surgeon to significant change and the need to interrupt the secondary assessment. Chest and pelvis radiographs are obtained if indicated. The examination generally proceeds systematically from head to toe, as in adults. At this time, a brief medical history, current medications, and immunization status are obtained. For penetrating wounds, it may be necessary to administer tetanus toxoid and/or immunoglobulin depending on the immunization status. At this point, if the child is hemodynamically stable with evidence of external injury or in pain, an analgesic should be given that will address the pain and facilitate the remainder of the examination. In children, pain is considered the fifth vital sign but is often overlooked.[76]

Bleeding from scalp lacerations should be controlled with pressure, surgical staples, or injection of local anesthetic containing epinephrine. Due to the frequency of head injury in children, the child should be examined for bleeding from the ear, bruising posterior to the ear, or orbital ecchymosis—findings that suggest basilar skull fracture. The chest is examined for crepitus, rib fractures, bruising, or penetrating wounds, and the chest auscultated. One or more of the following in children increases the odds of significant chest injury following blunt trauma: low SBP, elevated respiratory rate, abnormal thoracic examination, abnormal chest auscultation, femur fracture, and a GCS score of less than 15.[77] If there is evidence of pneumothorax, tube thoracostomy is performed.

Increased risk of abdominal injury in children is present when examination reveals low SBP, abdominal tenderness, or femur fracture. Abdominal tenderness confers a sixfold increased risk of significant intra-abdominal injury, but the reliability of absent abdominal tenderness to exclude intra-abdominal injury decreases with declining GCS.[78,79] Another important finding in children is bruising of the abdomen from either a seat belt[80] or handle bar.[81] When present, these findings should raise concern for a significant intra-abdominal injury. Lutz et al[82] reported that, among restrained children involved in a motor vehicle crash, those with a bruise were 232 times more likely to have a significant intra-abdominal injury when compared with children without bruising of the abdominal wall. Pelvic fracture is uncommon in children.[83] Nevertheless, the pelvis should be palpated for tenderness and instability. If either is present, an x-ray should be obtained; however, the routine use of pelvic x-ray in the evaluation of blunt-injured children is discouraged.[84,85] As in adults, if a pelvic fracture is present, the meatus in males and the vaginal introitus in females should be examined for blood. In postmenarche females, a urine pregnancy test should be obtained to identify an unknown pregnancy and the potential for fetal compromise. Routine rectal examination, like routine urinary catheterization, is not indicated in children.[86]

When examining the extremities, it is important to remember that children may have fractures with minimal displacement, known as greenstick fractures. Tenderness with or without hematoma, bruising, and displacement should prompt imaging. For displaced fractures, the pulses should be assessed distally, and the fractured extremity splinted to reduce pain and bleeding. It may be necessary to attempt reduction to restore flow if a distal pulse is absent.

The FAST examination, although a diagnostic test, is considered a component of the secondary assessment. In special circumstances, it may be performed earlier, particularly in the child in cardiac arrest or profound shock. In this situation, FAST permits examination of the heart and abdomen that can direct emergent interventions or discontinuation of resuscitative efforts if cardiac activity is absent. In stable children, the FAST cannot be solely relied upon to rule out intra-abdominal injury due to its low sensitivity.[87] Thirty to fifty percent of children with negative FAST will have a solid organ injury.[88] In general, the presence of free fluid on FAST is an indication for CT imaging in the stable child. Some

believe that the utility of FAST to direct CT imaging can be improved if integrated with additional supportive findings, such as elevated hepatic transaminases,[89] a scoring system,[90] or perhaps a step-up approach where imaging is only indicated if additional clinical factors are present.[91] Until these novel approaches that combine FAST with adjunctive findings, tests, or as an element of a scoring scale are validated, FAST should only be relied upon to direct intervention in a child with shock.

The utility of routine laboratory studies in the evaluation of trauma patients to aid in diagnosis of injury is limited and has been largely abandoned. The tests rarely result in a change in management and add unnecessary cost to care.[92] Capraro et al[93] showed that, in children with confirmed abdominal injury by CT, no routine laboratory test demonstrated good sensitivity, specificity, positive predictive value, or negative predictive value, leading the authors to conclude that routine "trauma panels" are not indicated as a screening tool in children with blunt trauma. Selective testing for suspected injury to the liver (aspartate aminotransferase), pancreas (lipase), or kidney (urinalysis) is not unreasonable but may lead to unnecessary CT imaging that only reveals injuries not requiring surgical intervention.[94] Laboratory studies are most helpful in assessing the metabolic state of the patient,[95] degree of acute blood loss anemia, and degree of trauma-induced coagulopathy. Keller et al[96] found that over half of children with severe TBI (GCS 3–8) had abnormal coagulation studies but few needed an intervention.[96] Arterial blood sampling provides valuable information regarding the metabolic condition of the critically injured patient. The degree of blood loss or the need for blood transfusion can be aided by hematocrit measurement,[97] although acute values may be falsely elevated.[61] Serum lactate, a byproduct of anaerobic metabolism, is useful to quantify the degree of metabolic derangement, is prognostic, and can guide resuscitation.[98]

The Environmental Protection Agency reports that nearly half of all radiation in the United States is attributable to medical imaging (Fig. 48-4). The overall average radiation of the US population has doubled in the past 25 years and is largely due to increased utilization of diagnostic CT scanning.[99] The radiosensitive pediatric population is most at risk. Brenner et al[100] predicted that although pediatric patients only represented 4% of all CT imaging, they account for 20% of all CT-related cancer deaths. In 2013, Miglioretti et al[101] quantified the risk of developing a future malignancy based on a child's age at the time of the CT scan. The authors estimated that 1 in 570 to 6130 scans will lead to the development of a new cancer depending on the child's age, sex, and type of scan.[101] In 2007, CT imaging was predicted to cause 29,000 new cases of cancer, of which 15% would result from imaging children. The theoretic prediction of cancer from CT imaging has been validated. A linear relationship between the dose of radiation delivered during a CT scan and the relative risk of developing leukemia and brain cancer was reported in a large cohort of European children.[102] Similar findings were reported in a study of Australian children in 2013.[103] Although the estimated

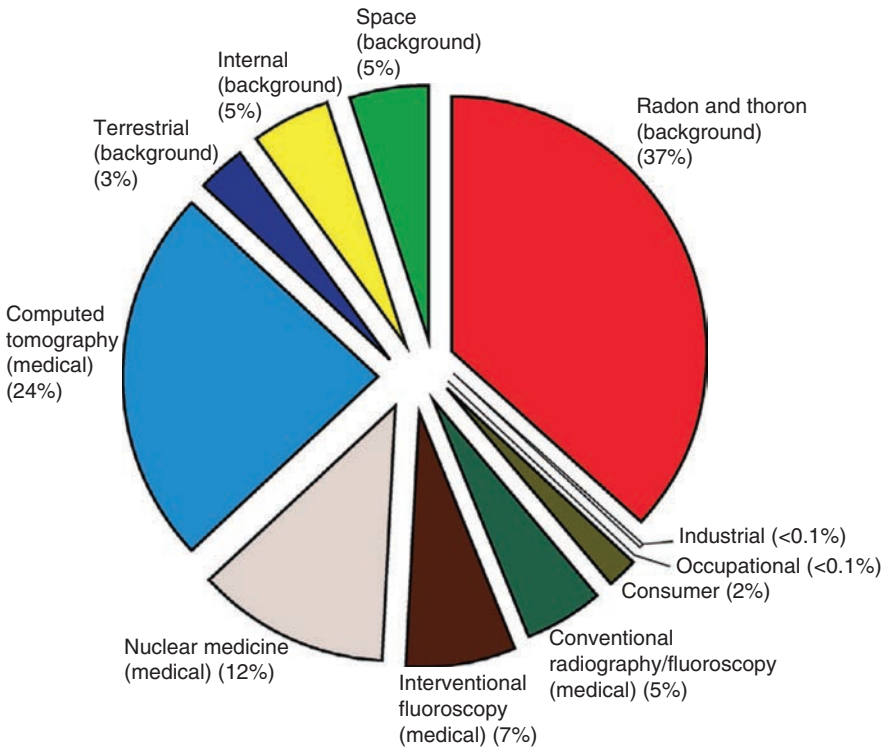

FIGURE 48-4 Sources of annual environmental radiation exposure in the US population. Note that radiation from medical imaging now accounts for 50% of the environmental exposure. (From Environmenal Protection Agency. Radiation effects. http://www.epa.gov/radiation/understand/perspective.html. Accessed January 22, 2020.)

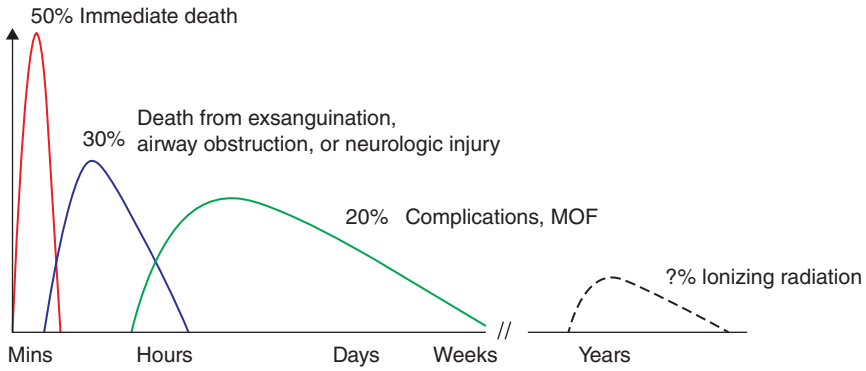

FIGURE 48-5 Hypothetical fourth peak of trauma death due to ionizing radiation from medical imaging in injured children. MOF, multiple organ failure. (Adapted from Trunkey DD. Trauma. Accidental and intentional injuries account for more years of life lost in the U.S. than cancer and heart disease. Among the prescribed remedies are improved preventive efforts, speedier surgery and further research. *Sci Am*. 1983;249(2):28-35.)

future risk of cancer is low, these studies represent only a relatively short period of observation following exposure. Moore et al[104] speculated that if real, late deaths due the effects of ionizing radiation could emerge as the fourth peak in trauma-related mortality, albeit many years later (Fig. 48-5).

Given our increasing knowledge of the risks associated with radiation, a CT scan should only be ordered when the results are likely to influence the care of the patient. To raise awareness and reduce radiation dosing during pediatric medical examination, the Alliance for Radiation Safety in Pediatric Imaging launched the Image Gently campaign in 2008. These and other efforts are intended to decrease the amount of radiation from imaging through specific steps[105] or by eliminating unnecessary imaging. Among the "five things that physicians and patients should question," the ACS advises to avoid the routine use of whole-body diagnostic CT scanning in patients with minor or single-system trauma. Clarifying which patients can safely forgo CT scans to minimize radiation exposure without missing clinically important injuries is critical. Children who suffer blunt trauma and have a normal chest radiograph do not benefit from chest CT.[106] Great vessel injury is rare in children, and typically, an abnormal silhouette on chest radiograph is reported. CT will detect more injuries than a conventional chest radiograph, but management rarely changes. For example, children, like adults, with an occult pneumothorax detected by CT but not seen on plain films do not require treatment but are simply observed.[107] Fenton et al[108] has demonstrated that although children are frequently imaged with CT, few will require a surgical intervention. In a cohort of injured children undergoing evaluation for blunt trauma, no child imaged on the basis of mechanism of injury alone had a significant or life-threatening injury. These children averaged 1.7 CT studies with a radiation exposure nearly three times the accepted annual limit for environmental sources. In contrast, in children with abnormal GCS (<15), abnormal vital signs, or abnormal physical examination, nearly one-fourth of studies were positive for significant or life-threatening injury.[104] Children should not be imaged based on mechanism of injury, but rather, imaging should be directed by findings of the primary and secondary survey.

In an effort to reduce CT utilization, clinical decision rules to identify children at low risk for clinically important injuries requiring intervention have been developed. The use of these decision instruments can effectively reduce unnecessary imaging of minor head injury, the cervical spine, and the abdomen. In the absence of a GCS score less than 14, altered mental status, loss of consciousness of greater than 5 seconds, vomiting, severe mechanism of injury, or skull fracture, children can be observed safely rather than imaged by CT. When studied, the prediction rule demonstrated a negative predictive value of 100% in children over the age of 2 years.[109] In the National Emergency X-Radiography Utilization Study (NEXUS), a decision rule was examined in a prospective, multicenter study. The investigators found that the decision rule correctly identified all pediatric patients with cervical spine injury and correctly designated 20% of patients as low risk, for whom imaging could have been eliminated.[110] The Pediatric Emergency Care Applied Research Network (PECARN) examined a clinical decision rule composed of seven history and physical exam factors (abdominal wall bruising, GCS <14, abdominal tenderness, thoracic trauma, complaints of abdominal pain, decreased breath sounds, or vomiting) that successfully identified children at low risk of clinically important abdominal injuries; 5.4% of children with a seat belt sign or GCS <14 underwent an abdominal intervention (Fig. 48-6). In contrast, if none of the criteria were present, the risk of an abdominal intervention was 0.1%.[111] Although the decision instrument can reduce imaging, the specificity of 42.5% demonstrates that over half of the studies remain negative for clinically significant injury. These data support the opinion that pan-scanning or nonselective imaging of children is unwarranted.

The primary and secondary assessments in children demonstrate subtle differences from those in the adult, but the principles remain largely the same. Seriously injured children typically suffer multisystem injury, and if shock is present, the risk of death is increased. Conversely, many children suffer single-system injury, with a low frequency of needing a craniotomy, thoracotomy, or laparotomy. Careful examination, selective laboratory studies, and the use of decision

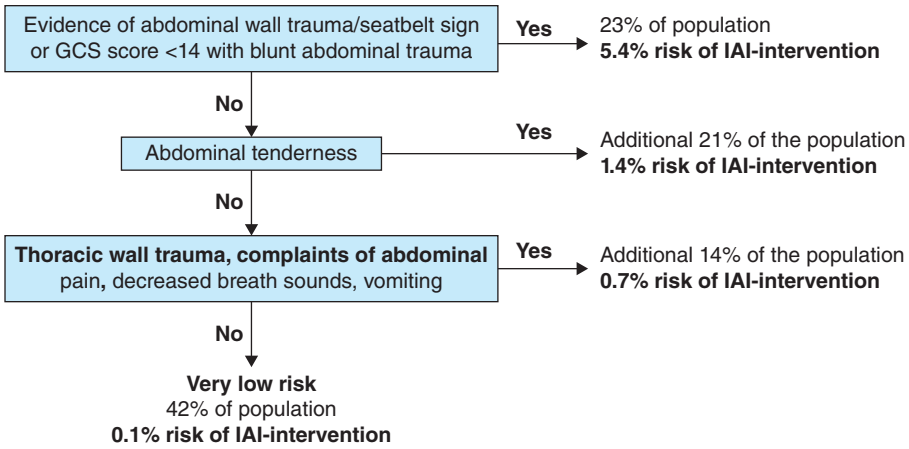

FIGURE 48-6 Example of a validated decision instrument to reduce unnecessary imaging in injured children by identifying at-risk children who likely benefit from imaging. GCS, Glasgow Coma Scale; IAI, intra-abdominal injury. (Reproduced with permission from Holmes JF, Lillis K, Monroe D, et al. Identifying children at very low risk of clinically important blunt abdominal injuries. *Ann Emerg Med*. 2013;62(2):107-116. Copyright © Elsevier.)

instruments can lead to directed imaging with few missed injuries.

MANAGEMENT OF SPECIFIC INJURIES

Neurologic Injury

TBI is a leading type of childhood injury according to the Centers for Disease Control and Prevention. In the United States alone, children age 0 to 14 years make almost half a million ED visits for TBI annually.[112] TBI resulting from unintentional trauma constitutes the primary cause of death among children and young adults. Fortunately, most brain injuries are mild,[113] but even this level of injury is associated with ongoing problems.[114] TBI is a significant financial burden on the economy of the United States. Charges for TBI-related hospital visits are more than $1 billion per year in the United States.[115] Neuropsychological sequelae associated with pediatric TBI can influence key developmental processes, such as learning, emotional awareness, and social functioning.[116] Injury costs for children are often greater than those for adults, because they include expenditures for acute treatment, long-term rehabilitation, and loss of productivity for the parent or guardian.

Children less than age 1 year are at increased risk of abusive head injury. In toddlers (age 1–4 years), head injury most often occurs due to fall. By adolescence, head injury because of a motor vehicle crash is most frequent. Children suffering unintentional serious head injury often have concomitant multisystem injury due to significant blunt force mechanism. Nearly 80% of all trauma deaths in children have associated TBI. Consequently, TBI is the most common cause of disability and death in childhood. Based on the current best estimates, severe pediatric TBI has a 20% mortality, and over half of those who survive demonstrate an unfavorable 6-month outcome.[117] As the severity of injury increases, the risk of death, vegetative state, or decreased functional status rises.

At birth, the head is one-fourth the total body length, whereas in the adult, it is one-seventh (Fig. 48-7). The head is proportionally large and the legs proportionally short during childhood. The center of gravity in an infant and toddler is more cephalad.[118] The skull of the infant is thin and soft, and the closure of sutures is not completed until age 3 years. This heavier head mass, coupled with a higher center of gravity and weak neck, may in part account for the propensity for children to strike the head in a fall and the higher frequency of head injury. The volume of cerebrospinal fluid is smaller than that of the adult, brain water content is increased, and brain myelination is incomplete until age 2 years. As a result, the brain is soft and prone to injury. Due to the anatomy and biomechanical factors observed in young children, acceleration and deceleration injury yields a greater amount of force applied to the brain and greater frequency of shear injury. Children appear to experience age-dependent pathology following pediatric TBI. In infants and young children, subdural hematomas and diffuse injury are more common than focal injuries, particularly in those suffering abusive head trauma.[119]

TBI refers to the primary brain injury that occurs at the time of the inciting event and the subsequent secondary brain injury that arises from derangements in cerebral blood flow, ischemia, and the inflammatory response to injury. Primary brain injury results from the immediate force of trauma. These forces can produce focal injury to the brain. Shearing of blood vessels can produce epidural hematoma, subdural hematoma, and/or subarachnoid hemorrhage. Parenchymal injury is worsened by compression from the mass effect of blood. The result is parenchymal injury exacerbated by increased swelling of the injured brain that is trapped in a confined space. The intracranial vault is incompressible and holds a fixed volume of brain, cerebrospinal fluid (CSF),

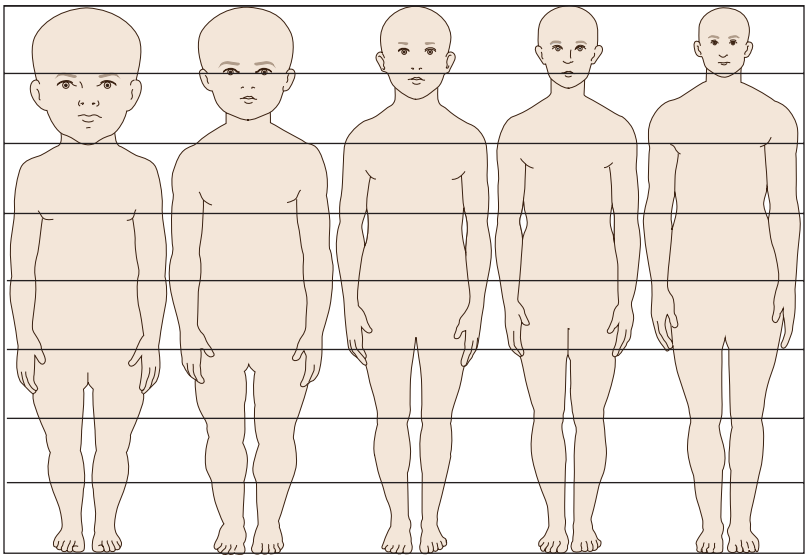

FIGURE 48-7 Graphic representation of head size relative to body size in infants through adulthood. In infants and children, the head is disproportionately larger and the limbs relative smaller, resulting in a higher center of gravity.

and blood. Any increase in volume of one of the cranial constituents must be compensated by a decrease in volume of another. This concept, the Monro-Kellie doctrine, serves as the basis for the management of brain injury.[120]

Initial compensation for the increase in intracranial volume is displacement of CSF to the spinal canal and displacement of venous blood to the jugular veins to reduce intracranial pressure (ICP). Once these compensatory mechanisms are exhausted, even small increases in cerebral edema and intracranial volume lead to significant increases in ICP, further compromising cerebral perfusion. Cerebral ischemia and edema follow and, if uninterrupted, result in brain herniation and death. Care of the primary brain injury may require neurosurgical intervention, but reduction in secondary brain injury can be initiated as early as the prehospital phase of care by nonneurosurgeons. It must be remembered that secondary brain injury develops in the initial minutes following primary brain injury. A myriad of physiologic and metabolic alterations ensue. Hypotension, hypoxia, and hypo- or hypercarbia have been demonstrated to exacerbate secondary brain injury and increase the risk of death.[32] All can be addressed by the nonneurosurgeon by the restoration of respiration and circulation. The acute management of TBI is directed to prevent or ameliorate events that promote secondary brain injury. To date, prevention of delayed secondary brain injury as a result of neuronal cell degeneration and traumatic axonal injury with neuronal cell death remains investigational.[121] Contemporary therapies focus on cerebrovascular dysregulation, swelling, metabolism, and ischemia.

The GCS is a scoring tool that permits assessment and stratification of the severity of brain injury. GCS correlates with the risk of disability or death.[122] Calculation of the GCS and physical examination permit stratification of injured children with mild, moderate, or severe TBI. Validated decision instruments direct the need to obtain brain imaging.[109] In general, any child with an abnormal GCS should undergo brain imaging. Additional history (loss of consciousness >5 seconds, amnesia), symptoms (vomiting, persistent headache), or physical findings (raccoon eyes, battle sign) may prompt brain imaging. The purpose of brain imaging is to exclude or confirm a brain injury, identify extra-axial blood collections, and determine the degree of brain compression. Prompt neurosurgical intervention may be needed for evacuation of extra-axial blood with significant mass effect, decompressive craniectomy for impending herniation or control of ICP, and placement of an intraventricular drain or ICP monitor. Delays in craniotomy or placement of ICP monitoring are associated with worse outcome.[123] Goals of ICP monitoring are to adjust therapies to maintain the ICP less than 20 mm Hg and maintain a minimum cerebral perfusion pressure (CPP) of greater than 40 mm Hg.[124]

Not all children with minor TBI require imaging. For children with minor TBI, a period of observation with scheduled neurologic assessment is recommended. Children with intracranial injuries may initially present with only headache and vomiting due to increased ICP, and thus it is recommended to consider imaging or a period of observation. It must be remembered that even in the setting of minor TBI, there is significant risk of ongoing cognitive and behavioral issues. Decision instruments for selective imaging of children can be used to reserve imaging only for those with abnormal GCS, symptoms of increased ICP (vomiting, headache), or other signs of head injury (skull fracture, battle sign, raccoon eyes).[109] Plain x-rays may demonstrate a skull fracture but provide no information regarding the underlying brain injury and cannot substitute for CT. The CT is reviewed for mass lesions, early signs of brain swelling, or evidence of diffuse brain injury. Conversely, a normal CT scan and brief period of observation may permit discharge with continued observation at home obviating the need for hospitalization.[125]

For moderate to severe TBI, the treatment is based on the Brain Foundation guidelines first published in 2003 and updated in 2012.[124] The current guidelines are derived from Level II/III evidence. Level II evidence supports the use of hypertonic saline and therapeutic hypothermia, but the evidence does not support the use of corticosteroids or immunomodulatory diets. The remaining guidelines provide Level III evidence for a CPP greater than 40 mm Hg; ICP monitor for GCS less than 8; adequate analgesia, sedation, and paralytics; seizure prophylaxis with phenytoin; and avoidance of severe hyperventilation (Pco_2 <30 mm Hg). Initial measures to address secondary brain injury are elevation of the head of the bed 30° to maximize venous drainage, definitive airway management for GCS less than 8 or rapidly declining GCS, administration of oxygen, and restoration of circulation. Even brief episodes of hypoxemia and/or hypotension have detrimental effects and increase mortality.[126,127] All may be initiated prior to neurosurgical intervention. Decreases in cerebral blood flow in the initial 24 hours following severe TBI in infants and young children have been associated with poor outcome.[56,127]

In patients who require mechanical ventilation, a Pco_2 of 35 to 40 mm Hg is optimal. Lower values risk unacceptable reductions of cerebral blood flow and potential increased secondary brain injury due to ischemia. Like mechanical ventilation, early hyperosmolar therapy may be initiated prior to neurosurgical evaluation either with hypertonic saline or mannitol. The advantage of hypertonic saline is that it also serves as a volume expander and can be administered in the hypotensive patient, unlike mannitol. Pediatric patients with severe TBI appear to tolerate a high osmolar load. Serum osmolality should be maintained at less than 360 mOsm/L. Once the mass effect of bleeding or increased ICP has been addressed, care of the brain-injured child becomes largely supportive. Additional adjuncts have been proposed, largely based on adult evidence of neuroprotection and neurorecovery with administration of excitatory amino acids, antagonists to the *N*-methyl-D-aspartate receptor, dopamine agonists, benzodiazepines, β-blockers, anticonvulsants, and antidepressants, but remain unproven.[128] In the event of refractory intracranial hypertension or impending herniation, decompressive craniectomy may be considered. Level III evidence suggests that it may be effective in reversing early signs of neurologic deterioration or herniation and in treating intracranial hypertension refractory to medical management; however, there is insufficient evidence to define patients who may or may not benefit.[124]

TBI impacts the life of individuals and their families. For individuals hospitalized after a TBI, almost half (43%) have a related disability 1 year after the injury. Approximately 5.3 million Americans are living with a TBI-related disability, and the consequences of severe TBI can affect all aspects of an individual's life.[129]

Spine and Spinal Cord Injury (See Chapter 26)

Spinal column and spinal cord injury are uncommon in children. Fractures of the spine constitute less than 1% of all pediatric fractures. In a study of the NTDB, Polk-Williams et al[130] reported that 1.59% of patients suffered an injury to the cervical spine; 0.38% had associated spinal cord injury associated with their spinal column injury, and 0.19% had an isolated spinal cord injury without a radiologic abnormality (SCIWORA). Despite the low incidence of cervical spine injury, immobilization is indicated in all children with significant head injury or multisystem injury. The cervical spine is susceptible to bony and ligamentous injury in the child. The neck is poorly muscularized, the ligaments lax, and the vertebral bodies wedged anteriorly with a tendency to slide forward with flexion. SCIWORA is particularly hazardous since children may only present with transient neurologic findings but then go on to develop neurologic deficits hours to days after the initial injury[131] (Fig. 48-8). The most common mechanisms of injury are road traffic accidents, sports, and falls. A homogenous distribution of spinal cord injuries is observed. Nearly all children who present with intact motor or sensory function recover, but only 37% to 54% with an incomplete injury (American Spinal Injury

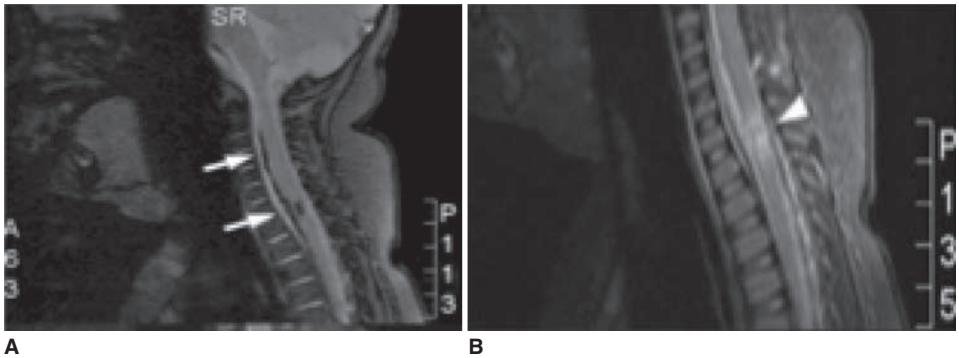

FIGURE 48-8 Spinal cord injury without a radiologic abnormality in a child. The magnetic resonance image demonstrates (**A**) significant spinal cord contusion and hemorrhage from C7 to T1, as well as ventral subdural hemorrhage from C2 through C7 and (**B**) evidence of interspinous ligament rupture between C5 and C6. (Reproduced with permission from Silman E, Langdorf MI, Rudkin S, Lotfipour S. Images in emergency medicine: pediatric spinal cord injury without radiographic abnormality. *West J Emerg Med.* 2008;9(2):124.)

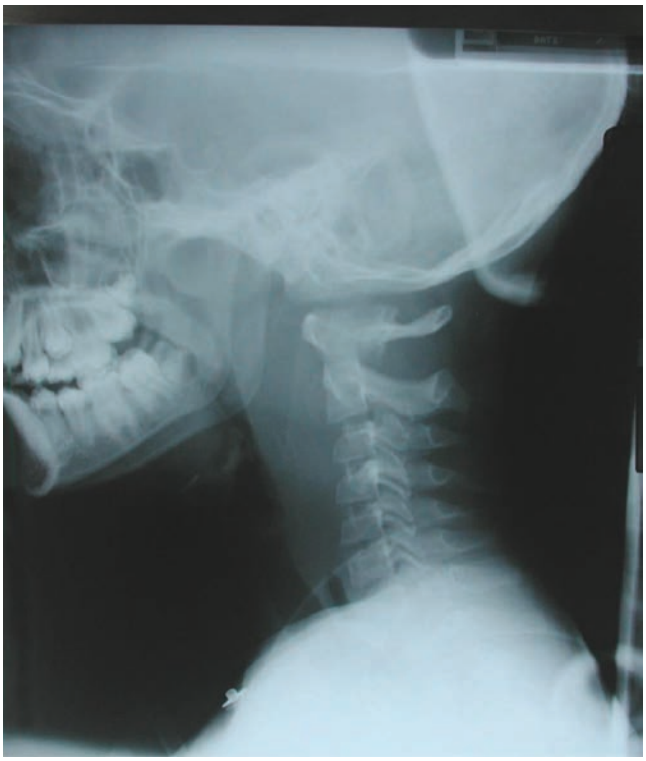

FIGURE 48-9 Atlantoaxial dislocation. This injury may be associated with a high spinal cord injury causing apnea at the scene.

Association Impairment [ASIA] Scale B or C) or 13% with a complete injury (ASIA Scale A) achieve a full neurologic recovery. Magnetic resonance imaging (MRI) is particularly useful in the diagnosis and prognosis of SCIWORA, given its accuracy for ligamentous injury, soft tissue injury, and spinal cord injury.[131] Young children most often suffer upper cervical spine injury, whereas older children and adolescents injure the lower cervical spine like adults[132] (Fig. 48-9).

When evaluating the pediatric spine, anterior displacement of C2 vertebra on C3 may be observed. Pseudosubluxation is a benign variant that may be observed in up to 20% of children suffering polytrauma.[133] In the absence of head injury or distracting injury, the cervical spine can be cleared by clinical examination.[110] All other children require imaging. CT scan has supplanted plain radiographs due to its superiority in detection of injury but at the cost of increased ionizing radiation exposure. Thus, the cervical spine should be cleared by clinical exam whenever possible. Hannon et al[134] have proposed an innovative algorithm specific to children using clinical examination and screening radiographs with focused CT reserved for abnormal radiographs. Such an approach will require validation in a larger cohort of children prior to widespread application.

Thoracic and lumbar spine injury is even less common in children; however, in the child with a lap belt sign, there is a significant risk of a fracture in the lumbar spine. The Chance fracture results from flexion and distraction of the upper lumbar vertebrae around the lap belt during rapid deceleration. Modern restraint design has not mitigated the risk of injury, likely due to submarining of the hips beneath the lap belt and hyperflexion secondary to forward excursion of the pelvis and legs during the collision.[135] There is a high incidence of an associated neurologic deficit that is most often transient but may be permanent in up to 10% of children.[136] Mahan[136a] found that spinal column injury is most common in children over the age of 8 years. Nearly one-third have multilevel injuries, 6% of which are noncontiguous, emphasizing the importance of complete imaging of the spine.

Management of an injury to the spinal cord parallels that of TBI with an emphasis on avoidance of secondary injury. Spine immobilization is essential to avoid further injury. Resuscitation ensures perfusion of the injured spinal cord. High cervical cord injury may produce a distributive form of shock (neurogenic) because of autonomic disruption, loss of sympathetic vascular tone, relative hypovolemia, and decreased peripheral vascular resistance. Fluid administration is usually adequate to restore blood pressure, but in some cases, a vasopressor (eg, phenylephrine) will be added to maintain adequate blood pressure. In rare cases, autonomic dysregulation produces bradycardia that may require administration of a drug with chronotropic effects such as dopamine. As in adults, the use of high-dose corticosteroids has been abandoned and is no longer recommended.[137]

Spinal cord decompression and spinal column stabilization are the most common indications for operative treatment. However, many spinal column injuries in children are managed nonoperatively until adolescence when treatment generally patterns what is recommended in adults. The outcome for children suffering an injury to the spinal cord is related to the level and severity of injury. In a series of children with injury to the cervical spine who were assessed using functional independence measures, most communicated independently, fed themselves independently, and had independent locomotive function following rehabilitation.[132] Review of the literature demonstrates that pediatric patients with traumatic SCI have a better neurologic recovery potential when compared to adults. If the injury is sustained prior to their adolescent growth spurt, unlike adults, they have a higher likelihood of developing scoliosis.[138]

Thoracic Injuries

Thoracic injuries account for 4% to 15% of pediatric trauma admissions. About 80% of thoracic injuries in children are from a blunt mechanism. Penetrating injuries are more likely to be lethal than blunt injuries. Isolated thoracic trauma has a mortality rate of approximately 5%. Children with combined head, thoracic, and abdominal trauma have a mortality rate that approaches 40%. Thoracic trauma is likely to be present in children who present with a low SBP, an elevated respiratory rate, and abnormalities on physical examination of the thorax.

Injuries to the lung and chest wall are the most common type of thoracic problem. Lung injuries, mainly contusions, account for almost half of all chest injuries. Chest wall injuries

(ribs and sternum) and pneumothorax/hemothorax account for about one-quarter each. Most of these can be managed with supportive care (oxygen, pain control, and physiotherapy) and, in some cases, a pleural drain. Injuries to the major airways, heart, and great vessels are much less common and compose less than 1% of all thoracic injuries.[139] They are much more likely to be lethal and often require an operation.

The child's thorax has unique anatomic and physiologic properties that are important to the diagnosis and treatment of thoracic trauma. The chest wall is more compliant in children because the ribs are more easily deformed and there is less muscle. This allows a greater transmission of energy to the intrathoracic organs when a blunt force is applied. The mediastinum is more mobile than in older patients, especially in young children. Unilateral changes in thoracic pressure, such as with a tension pneumothorax, can lead to a shift of the mediastinum that impairs venous blood return to the heart. Because the ribs of young children are more compliant, they tend to bend, not fracture. Consequently, rib fractures are relatively uncommon in young children. For the same reason, injuries to the lung, liver, and spleen lying underneath the ribs are more common.[34]

The assessment of a child with a suspected chest injury is based on the history, physical examination, and plain chest x-ray. A standard anteroposterior (AP) chest x-ray alone will reveal almost all significant chest injuries.[106,140] Although CT scanning will identify more injuries than conventional chest radiograph,[106] the results rarely lead to a change in management. CT scanning can be avoided in most cases of chest trauma to minimize radiation exposure, except for those with abnormal plain films, high-risk mechanisms, or abnormal physical findings.[141] Children who do not meet these criteria are very unlikely to have a significant chest injury.[77]

The history should document the circumstances of the injury, the subsequent course, and any interventions such as IV fluid administration or blood transfusion. The physical examination should include inspection for bruises and other external signs of injury; palpation for rib tenderness, crepitus, and tracheal deviation; and auscultation for diminished or asymmetric breath sounds. All victims of chest trauma should have a chest x-ray and a hemoglobin/hematocrit. If the clinical assessment supplemented by the chest x-ray and hematocrit is normal, no further diagnostic or therapeutic intervention is required. If the clinical assessment and/or the chest x-ray are abnormal, treatment can be initiated as appropriate, or further diagnostic tests can be ordered. Ultrasound examination of the chest (extended FAST [E-FAST]) has been reported for diagnosing a pneumothorax or hemothorax.[142] E-FAST may be more sensitive than a conventional AP chest x-ray in detecting a small pneumothorax or hemothorax. When a specific injury is suspected based on clinical assessment and plain chest x-ray, a CT scan, ultrasound, or transthoracic or transesophageal echocardiography can be used to confirm the diagnosis. Bronchoscopy is indicated in selected patients thought to have a major airway injury that is usually manifested by a massive or continuing air leak. Conventional angiography is seldom needed for diagnostic purposes.

RIB FRACTURES

Rib fractures are less common in children compared to adults.[143] This is believed to be because their ribs are flexible, so they tend to bend but not break. This is also why flail chest is much less common in children. Rib fractures following trauma indicate significant blunt force with an increased likelihood of serious injury. In a study of the NTDB, Rosenberg et al[144] showed that mortality doubled from 1.8% without rib fracture to 5.8% for one rib fracture and then nearly linearly increased to 8.2% for seven fractures. Children with two or more rib fractures were more likely to require a thoracostomy tube, thoracotomy, thoracoscopy, and/or laparotomy.[144] Rib fractures in children less than 2 years of age and posterior rib fractures are highly predictive of nonaccidental trauma.[145] The treatment of rib fractures in children is supportive. Pain management may include systemic analgesics, intercostal nerve blocks, and epidural analgesia.

PULMONARY CONTUSION

One of the most common thoracic injuries in children is a pulmonary contusion, which can occur with blunt or penetrating trauma. Pulmonary contusions decrease lung compliance and cause ventilation/perfusion mismatch. Both can lead to hypoxia. A chest x-ray taken during the initial assessment is the most common means of diagnosing a pulmonary contusion. CT is more sensitive and may show areas of pulmonary contusion not appreciated on a plain x-ray. However, contusions seen on CT but not visible on plain x-rays are of little clinical significance. Conversely, significant pulmonary contusion visible on plain x-ray correlates with impairment of oxygenation, carbon dioxide exchange, and duration of ventilator support.[146] Treatment of a pulmonary contusion includes appropriate fluid resuscitation, supplemental oxygen, pain management, and strategies to prevent atelectasis and pneumonia. Most children recover quickly from this injury, but some patients develop pneumonia or respiratory distress syndrome. In an occasional patient, a large pulmonary contusion may cause life-threatening hypoxia that cannot be supported with conventional or advanced techniques of ventilation including high-frequency oscillation. Extracorporeal life support has been used in extreme circumstances to support patients with severe pulmonary contusions and secondary respiratory distress syndrome.

PNEUMOTHORAX AND HEMOTHORAX

Most pneumothoraces can be definitively treated with a thoracostomy tube appropriately sized for the patient. A small pneumothorax without mediastinal shift that can be seen on CT, but not plain x-ray, does not require treatment.[107] A tension pneumothorax should be treated emergently as in an adult with a needle thoracostomy in the second intercostal space in the mid-clavicular line, followed by a chest tube in the anterior axillary line at the nipple level (Fig. 48-10). A hemothorax should be drained. Historically, a large chest tube was placed, but contemporary evidence suggests that a

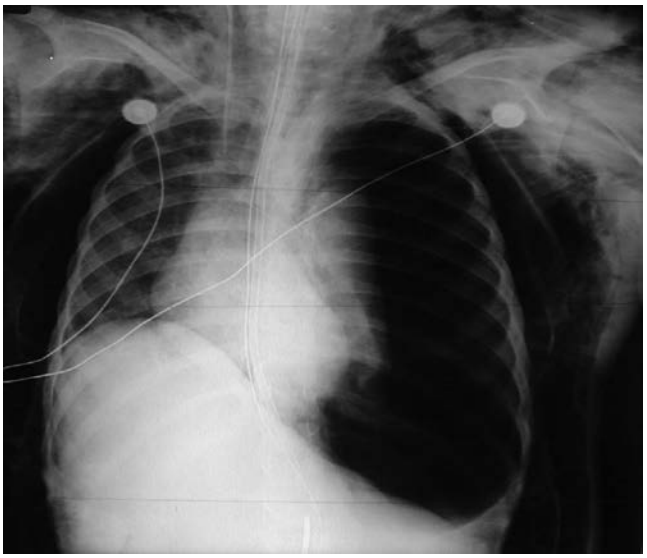

FIGURE 48-10 Left tension pneumothorax. Note flattening of the left diaphragm and deviation of the mediastinum to the right.

small-bore chest tube is equally effective as a large-bore tube and may reduce the risk of empyema[147] (Fig. 48-11).

Duration of tube thoracostomy and retained hemothorax appear to be the greatest risk factors for posttraumatic empyema.[148] Therefore, it is imperative that if a small-bore catheter is used, the chest is effectively drained and the chest tube removed as early as possible. Thoracotomy is indicated to control the bleeding if more than 15 mL/kg of blood drains immediately or if bleeding continues at a rate of greater than 2 to 3 mL/kg/h for 3 or more hours; this is rarely necessary in children.

MEDIASTINUM

Although extremely rare, tracheobronchial, cardiac, major blood vessel, and esophageal injuries do occur and should be ruled out. Major tracheobronchial injuries typically occur in the membranous part of the trachea or mainstem bronchus. They may cause a major air leak that leads to a tension pneumothorax and a massive ongoing air leak after placement of one or more chest tubes. When a lobe or lung fails to expand after placement of a chest tube, a major airway injury should be ruled out (Fig. 48-12).

These patients should be taken to the operating room for bronchoscopy and possible thoracotomy. Temporary control may be achieved by advancing an ETT beyond the leak, but open repair is often needed. Primary repair of a major airway injury is ideal, but when a lobar bronchus is completely avulsed, a lobectomy may be a better option.

Blunt cardiac injuries are rare. The most common type is a myocardial contusion for which there is no specific therapy.[149] Free wall rupture and valve disruption have also been reported, but survivors are rare. Free wall injuries may cause pericardial tamponade, which is manifest by pulsus paradoxus. FAST exam or echocardiography can confirm the diagnosis of pericardial effusion. Contusions may cause dysrhythmias and hypotension. The diagnosis of blunt cardiac contusion is largely based on clinical suspicion supplemented by electrocardiogram and serum cardiac enzyme levels. An echocardiogram is unnecessary except in the rare cases of cardiac failure or valve disruption.

Injuries to the great vessels do occur in children injured in high-speed highway crashes and other mechanisms characterized by great force and rapid deceleration, but they are rare. In a review of the NTDB, Heckman et al[139] found that only

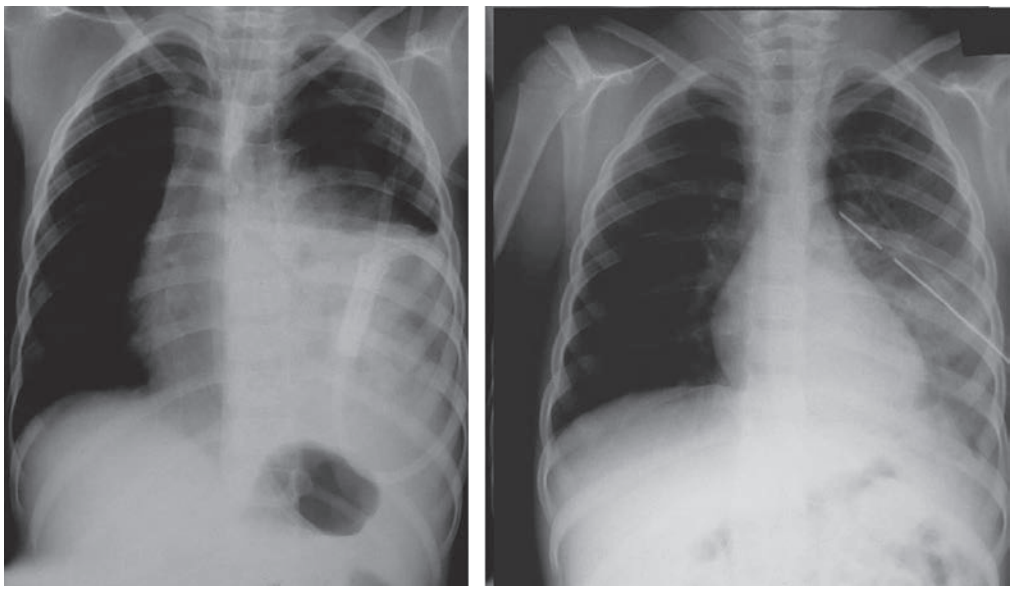

FIGURE 48-11 (**A**) Hemopneumothorax. (**B**) Complete resolution with no further intervention after evacuation of the air and blood by a chest tube.

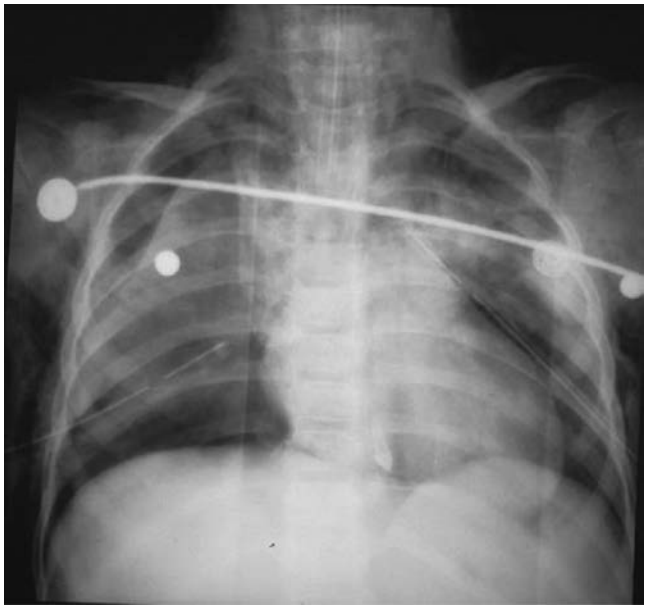

FIGURE 48-12 Major bronchial injury with large air leak and persistent pneumothorax despite intercostal drain.

34 (0.1%) of 26,940 patients suffered injury to the thoracic aorta, but the observed mortality was high (41%). The true incidence may be underestimated due to prehospital death.

Clinical predictors of thoracic aorta injuries include a low SBP on admission; increased respiratory rate; abnormal thoracic exam, especially on chest auscultation; an associated fracture of the femur; and a GCS of less than 15. If there is a high index of suspicion based on history, physical examination, or an x-ray of the chest, further testing is required. Helical chest CT and transesophageal ultrasound (echocardiography) have been found to be equally sensitive when compared with aortography in the detection of an aortic injury. Once the injury is found, the treatment of thoracic aortic injuries is like that in adults. β-Blockers should be started early. Treatment options include nonoperative management for minor intimal tears and operative management for more extensive injuries. Operative repair can be accomplished by thoracotomy and direct repair, usually with an interposition graft. Endovascular stent graft repairs have definite advantages, if a correctly sized graft is available.[150] The long-term outcome of endovascular stenting in children is unknown.

TRAUMATIC ASPHYXIA

This injury is caused by compression of the chest and upper abdomen that produces a marked elevation of pressure in the superior vena cava and its feeding branches. The clinical manifestations are swelling of the face and petechial hemorrhages in the skin above the nipple line and in the conjunctivae.[151] Occasionally, respiratory arrest due to hypoxia ensues, but most children recover uneventfully with supportive measures including elevation of the upper body and oxygen by mask or nasal cannula.

COMMOTIO CORDIS

A direct blow to the anterior chest, typically in the mid-sternal area, may cause sudden cardiac collapse from ventricular fibrillation. Mortality is high.[152] Young children appear most at risk due to their thin, pliable anterior chest that affords decreased protection to the heart. Age and participation in sports that risk a blow to the anterior chest appear to be the most important risk factors. In a review of the US commotio cordis registry, Maron et al[153] reported an overall mortality of 72%. From 1970 to 2012 survival has steadily increased from 10% to 58%, perhaps due to more rapid response times, access to defibrillation in the field, and improved public awareness of the condition.[153]

Abdominal Injuries

Blunt or penetrating forces can cause abdominal injuries, but blunt injuries are much more common in children. The most common causes are falls, motor vehicle crashes, and direct blows to the abdomen. Physical abuse or nonaccidental trauma is another important cause of abdominal injury in children.[154,155] Solid organ and hollow organ injuries are equally common in child abuse, but solid organ injuries are much more frequent in unintentional trauma. The most common abdominal injuries are to the solid abdominal organs—the spleen, liver, and kidneys. Injuries to the hollow structures—the stomach, duodenum, small bowel, colon, biliary and pancreatic ducts, renal pelvis, ureters and bladder, and major blood vessels—are relatively rare. Significant intra-abdominal injury may result from a relatively minor force, especially in young children, because of the lack of effective protection by the muscle layers of the body wall and the ribs primarily for the liver and spleen. The seat belt syndrome is a rare but important type of blunt abdominal trauma caused by compression of the bowel and/or the pancreas by a seat belt positioned above the pelvis against the spine in deceleration crashes. It may be associated with an unstable fracture-dislocation of the lumbar spine (Chance fracture) and occasionally with spinal cord injury. The hallmark is a transverse bruise across the lower abdomen.[156]

In general, there are two important types of intra-abdominal injury: solid organ injury (liver, spleen, kidney) that can cause bleeding and hollow organ injury (intestine, bile or pancreatic duct, renal pelvis, ureter, bladder) that can cause peritonitis and sepsis. Thus, it is useful to think about the assessment of abdominal trauma using this framework. Is there evidence for bleeding or peritonitis, the two key threats to life from abdominal trauma and the two key indications for an operation?

Clinical assessment is the most important step in the diagnosis of abdominal injuries because it is the best way to detect signs of bleeding or peritonitis. Abdominal CT with IV contrast is the best way to determine the exact nature of an intra-abdominal injury, but CT should be limited to patients in whom there is reason to suspect such an injury.[104] Not every "trauma" patient needs an abdominal CT scan.

The indications for CT scanning should ideally be defined by hospital-specific decision rules to avoid unnecessary radiation exposure.[77,111,141]

Clinical assessment is also the most useful guide to the treatment of abdominal trauma. CT will typically reveal the anatomy of an intra-abdominal injury, but it is not very helpful in deciding how to manage the injury. A CT scan is very useful in documenting the exact nature of an intra-abdominal injury, but the decision to treat nonoperatively or by laparotomy is almost always made on clinical grounds, not by the CT scan. We have found that children who present to the ED after blunt abdominal trauma by a nonmotorized force with a normal GCS and SIPA are unlikely to have a solid organ injury that will require intervention. For injured children who meet these criteria, it is safe to forgo a CT scan.[157]

The history should document the circumstances of the injury, especially the mechanism, the subsequent course, and any interventions, such as IV fluid administration or blood transfusion. The mechanism may point to a specific injury. For example, a blow from a bicycle handlebar may cause an injury to the gastrointestinal tract or pancreas. Blunt abdominal trauma without a clear mechanism in an infant should prompt consideration of child abuse.

The physical examination should include inspection for bruises, especially lap belt bruises and other external signs of injury, distension, presence or absence of bowel sounds, and focal or diffuse tenderness with or without rigidity. The seat belt sign, a well-defined transverse bruise, band of erythema, or skin abrasion across the lower abdomen, is associated with an increased risk of intra-abdominal organ injury, especially to the bowel and pancreas.[111,158,159] All victims of abdominal trauma should have a urinalysis and a hemoglobin/hematocrit. If the clinical assessment supplemented by the urinalysis and hematocrit is normal, no further diagnostic or therapeutic intervention is required. If there is hemodynamic instability with frank evidence of intra-abdominal bleeding or clear evidence of peritonitis, a laparotomy is indicated. For the majority of patients who fall between these extremes in whom the diagnosis is in doubt, further observation with repeat clinical evaluation and further diagnostic tests are indicated. This is especially true for patients with an abnormal GCS score. Nasogastric or orogastric drainage is indicated in all children requiring a laparotomy and in selected children undergoing observation and further workup, especially if they are obtunded, distended, or vomit frequently.

Liver function tests, serum amylase, and lipase may be helpful in patients with mild to moderate abdominal pain and tenderness who are suspected of having an intra-abdominal injury or injuries; however, an abnormal result is not a clear indication for a laparotomy or imaging. A CT with IV contrast may also help in these circumstances, especially if consciousness is reduced, as noted earlier. Conventional FAST examinations lack sensitivity in detecting abdominal organ injuries, and up to one-third of solid organ injuries will be missed by FAST.[57] Contrast-enhanced ultrasound (CEUS) may be an exception because there is evidence that CEUS may be more accurate than conventional ultrasound in detecting injuries to the liver, spleen, kidneys, and pancreas.[160] CEUS may be especially useful in the assessment of abdominal trauma due to low-energy mechanisms.[161] A conventional FAST examination may help to establish the presence of abnormal amounts of fluid in the abdomen; however, FAST has not been shown to be accurate enough for definitive diagnosis of abdominal injuries. FAST can be helpful in physiologically unstable patients who have other injuries that could be the cause of their instability but are too unstable for CT scanning.[57] Injuries to the liver and spleen are graded based on CT findings according to the organ injury scaling system developed by the American Association for the Surgery of Trauma.[162] This is the same system used for adults.

Minimally invasive surgery has been successfully applied to both blunt and penetrating abdominal injuries in adults and children both for diagnosis and definitive therapy. It should only be considered as an option in children who are hemodynamically stable. Laparoscopy can reduce the rate of nontherapeutic laparotomy, especially in penetrating trauma where the indication for surgical intervention depends more on the mechanism than the physiologic impact as in blunt trauma. Even in cases where conversion to an open procedure proves necessary, laparoscopy may allow better placement of a smaller incision instead of the conventional long midline laparotomy. On the down side, the risk of missing an injury to the bowel may be higher with laparoscopy than with an open laparotomy.

In cases of abdominal trauma in which the patient has lost physiologic and metabolic reserve evidenced by continuing shock with metabolic acidosis, hypothermia, and coagulopathy from massive blood loss, damage control surgery may be lifesaving. Damage control is most often needed in penetrating trauma cases with major vascular and/or intestinal injury or blunt liver injury with major damage to the hilum or retrohepatic venous structure. Damage control surgery aims to save the patient's life by doing only what is necessary to reverse the acidosis, hypothermia, and coagulopathy cycle. This is accomplished in three stages:

1. Focused operation to stop bleeding and further contamination
2. Resuscitation in the ICU including rewarming, transfusion, and hemodynamic support
3. Reoperation for definitive repair and wound closure

Switching to damage control should be considered when the patient's core temperature falls below 34°C, arterial pH falls below 7.2, international normalized ratio/prothrombin time are greater than 1.5 times normal, abnormal bleeding is observed, and cumulative blood loss exceeds 40 mL/kg or 50% of the baseline blood volume.[163]

DIAPHRAGM

Rupture of the diaphragm is rare in children, and most occur on the left side.[164] The usual mechanism is a high-energy abdominal injury such as can occur in a motor vehicle crash or fall from a great height. Children with a diaphragmatic

injury often suffer multiple injuries with high severity.[164] When the diaphragm is ruptured, the liver and spleen are often also injured. The diagnosis is most often made on plain abdominal or chest radiographs, but an apparently normal plain x-ray cannot exclude it. Diaphragm injuries are obvious when gas-filled bowel loops are seen in the chest, but they may be subtle. When suspected, repeat plain radiographs, ultrasound, or CT scan should confirm the diagnosis. Diaphragm injuries should be repaired urgently by a transabdominal approach.

GASTROINTESTINAL TRACT

Injuries to the bowel are relatively uncommon compared to injuries to the liver and spleen. Most are caused by a direct blow to a focused area of the abdominal wall from a seat belt, fist, handlebar, or butt end of a hockey stick or similar mechanism. The lap belt complex is a constellation of injuries due to a deceleration injury in a restrained child (Fig. 48-13). These children are often improperly restrained with the seat belt across the mid abdomen rather than the bony pelvis. A lap belt ecchymosis identifies a child at risk of pancreas injury, hollow viscus injury, major vascular injury, and spinal cord/column injury.[82,111,165] Holmes et al[111] reported that 5% of children suffering blunt injury required an intra-abdominal intervention when a lap belt ecchymosis was present. Contrast-enhanced CT is highly sensitive in the diagnosis of solid organ injury, but it is less sensitive in diagnosing injuries to the small and large bowel. Pneumoperitoneum and extravasation of enteral contrast indicate intestinal perforation, but they occur in less than half of cases. Suspicious CT findings include bowel enhancement/thickening, mesenteric stranding, and free fluid in the absence of a solid organ injury, but none of these is considered diagnostic. Occult intestinal perforation will cause peritonitis.[165] Diagnostic delay is not uncommon but does not appear to adversely affect outcome in children,[156,166] unlike in adults.[167] Repeated clinical assessment is performed with attention to unexplained tachycardia, fever, and/or peritoneal irritation.[168] Diagnostic peritoneal lavage or laparoscopy can be used to identify hollow viscus injury in the child with CNS injury, multiple injury, or equivocal findings of intestinal perforation.

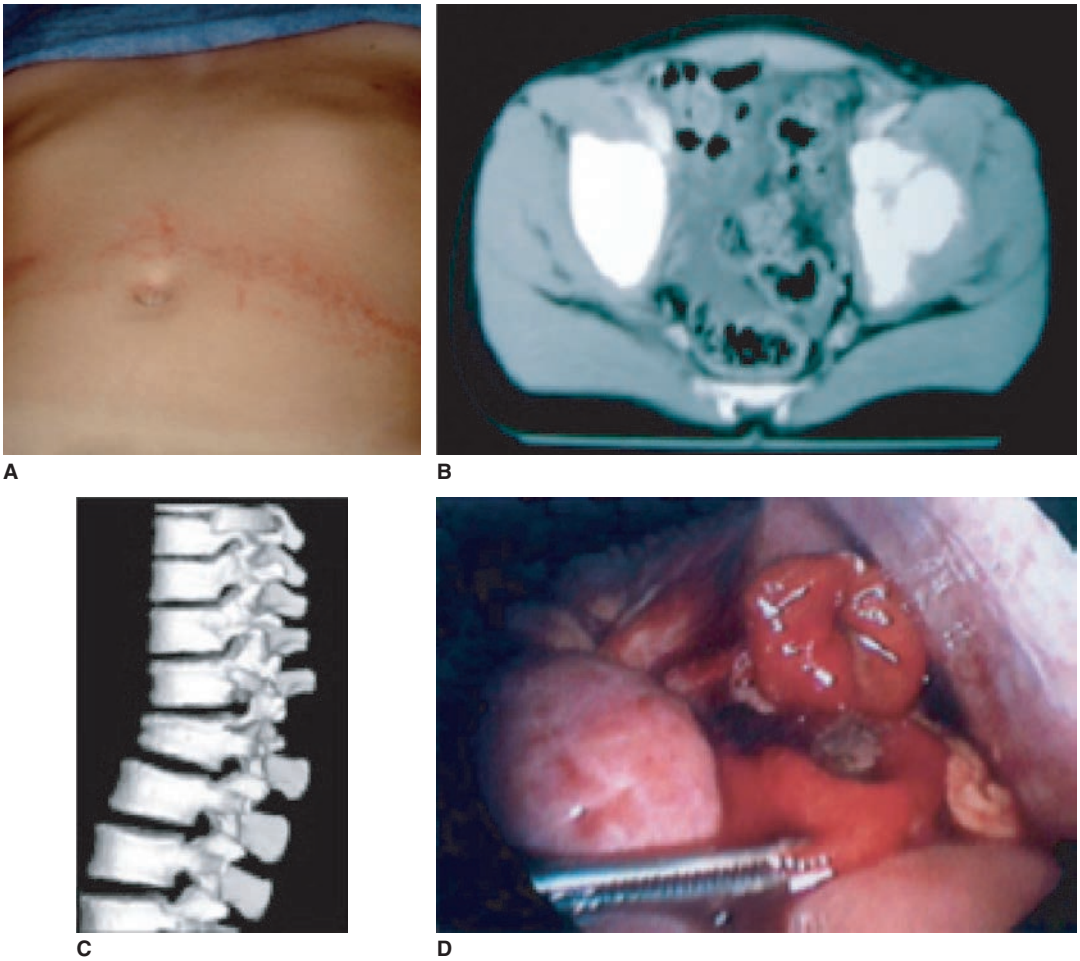

FIGURE 48-13 Lap belt complex in a 12-year-old back seat passenger in rollover motor vehicle crash. (**A**) Characteristic abdominal wall ecchymosis demonstrating malposition of the restraint across the mid abdomen. (**B**) Abdominal computed tomography scan with free fluid without solid organ injury. (**C**) L2 compression fracture (Chance fracture). (**D**) Transection of jejunum visualized by laparoscopy.

Blunt force trauma can cause a contusion to the bowel that results in a mechanical obstruction, avulsion of the mesentery, or frank perforation. Delayed mechanical obstruction from a stricture or delayed free perforation may occur in an area of devascularization. Duodenal hematomas cause feeding intolerance and vomiting that is usually temporary. Ultrasound, upper gastrointestinal series, or CT can confirm the diagnosis of duodenal hematoma. The hematoma will usually resolve after a period of bowel rest. Surgical intervention is seldom needed. Contusions to the intestine may occur, and cases have been reported of delayed presentation of intestinal obstruction due to stricture or adhesions.[168] In the case of intestinal perforation, the injury is generally amenable to resection of the perforation and primary anastomosis.

LIVER

Liver injuries are very common but fortunately seldom require operative treatment. Almost all are caused by blunt trauma. The main risk is bleeding, but this is rarely life threatening since most liver injuries involve the parenchyma only, not the named perihepatic or intrahepatic vessels or biliary system. Most injuries to the liver heal spontaneously. Liver injuries are most commonly found on an abdominal CT scan. In rare cases, a major hilar, intrahepatic or retrohepatic blood vessel injury will present with massive blood loss that requires immediate laparotomy. A useful criterion for operative intervention is the estimated loss of greater than 50% of the circulating blood volume. Operations for massive liver bleeding can be very difficult, even lethal. It is very important to avoid intraoperative hypothermia, acidosis, and coagulopathy. An MTP should be invoked, and red blood cells, fresh frozen plasma, and platelets should be transfused in a 1:1:1 ratio. Direct repair of major hepatic and perihepatic vascular injuries should be attempted if it can be accomplished promptly with appropriate vascular control before the lethal triad of hypothermia, acidosis, and coagulopathy develops. Damage control surgery should be considered as an option, as in adult liver trauma. Liver packing and adjunctive therapeutic embolization have been reported in children.[169]

SPLEEN

Injuries to the spleen are also very common (Fig. 48-14). Almost all are caused by blunt forces. Patients with splenic injuries tend to fall into one of two groups: those with isolated splenic trauma often from a direct blow and those with splenic trauma in the context of multiple other significant injuries. The former almost always heal spontaneously and almost never need an intervention. The latter are more likely to require a blood transfusion and, in some cases, operative intervention. Some authorities have suggested that splenectomy rates be used as a quality measure of care given that splenectomy rates are higher in nontrauma centers compared to designated trauma centers.[170] Nonoperative management is recommended for all patients with blunt splenic injury who are hemodynamically stable. The main indication for operative intervention is failure to restore hemodynamic

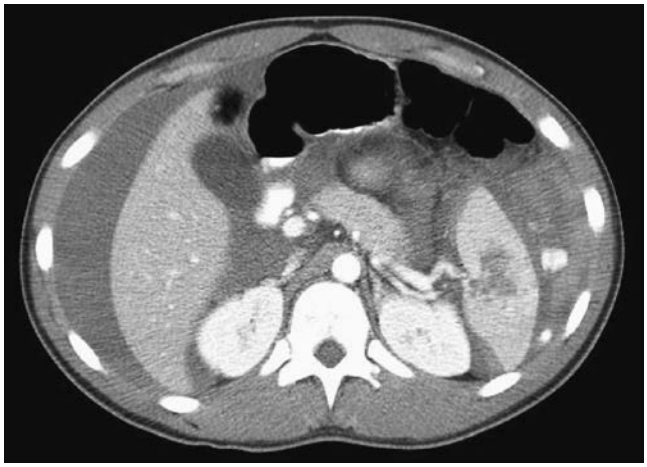

FIGURE 48-14 Splenic injury with a vascular "blush" sustained in a football game. The bleeding stopped after one transfusion.

physiology (pulse rate and blood pressure) in the normal range for the patient's age. As in liver trauma, a useful criterion for operative intervention is the estimated loss of greater than 50% of the circulating blood volume. If an operation is indicated, an attempt should be made to salvage the spleen by splenorraphy or partial splenectomy. Transarterial angioembolization (AE) has been used successfully in adults as an alternative to operative intervention. AE has been reported in children for various indications including active bleeding with an arterial blush on CT, hemodynamic instability, high-grade injury on the organ injury scale, and pseudoaneurysm formation, but experience in children is minimal and the indications for AE in children are not established.[171] If a laparotomy is undertaken, an attempt should be made to stop the bleeding and salvage the spleen by direct repair or partial splenectomy, leaving at least one-third of the splenic parenchyma with an intact blood supply. Prompt splenectomy should be carried out if the bleeding is massive or if there are other significant intra-abdominal injuries that need attention. Patients who require a splenectomy should receive appropriate immunization against the encapsulated organisms *Haemophilus influenzae* type B, pneumococcus, and meningococcus. For children with solid organ injury (liver, spleen, kidney), the success of nonoperative management approaches 95%.[172] Children who fail nonoperative management typically fail early, within the first 24 hours of injury. Children who are hemodynamically stable are admitted for a period of observation based on injury grade and discharged home with activity restrictions for 2 to 6 weeks (organ injury grade + 2 weeks). Follow-up imaging is not performed routinely.

PANCREAS

Blunt injuries to the pancreas are the fourth most common abdominal organ injury after the spleen, liver, and kidneys. They usually result from a direct blow from an object, such as a bike handlebar, or other similar mechanism that compresses the pancreas against the spine. Pancreatic injuries are difficult

to diagnose and may be missed at initial presentation. They are classified according to the organ injury scaling system developed by the American Association for the Surgery of Trauma.[162]

The most common symptoms suggesting pancreatic injury are fever, epigastric pain that radiates to the flank or back, anorexia, nausea, and vomiting. There is usually tenderness in the epigastrium. However, the symptoms and signs may be minimal in the initial 12 to 24 hours after injury even when the main duct has been disrupted. In the first few hours after injury, serum amylase levels have low sensitivity and specificity for major pancreatic trauma; persistent and rising elevations are more predictive.[173] CT scanning detects most major pancreatic injuries, although they may be missed on the initial study. Endoscopic retrograde cholangiopancreatography (ERCP) can be used to confirm and define the nature of a ductal injury[174] (Fig. 48-15). Magnetic resonance cholangiopancreatography (MRCP) is reported to be accurate in the identification of pancreatic ductal injury and may help to clarify the need for ERCP.[175] Stent placement by ERCP may be beneficial in cases of proximal duct injury.

Most pancreatic injuries, especially grade I and II injuries, will resolve spontaneously with the important exception of injuries to a major intrapancreatic duct. The management of pancreatic injury with ductal transection in children is controversial.[174,176] Pancreatic ductal injury can be successfully managed conservatively but usually only after a long period of pain, feeding intolerance, and parenteral nutrition. A pseudocyst may develop, requiring drainage. To avoid a prolonged recovery and complications, many experts advocate early distal pancreatectomy based on CT evidence of a transection of the main duct in the mid body of the pancreas. A multicenter study of the management of pancreatic injury in children found that children managed with distal pancreatectomy had earlier return to enteral feeding, shorter hospital stay, fewer procedures, and fewer pancreatic-related complications.[176] This multicenter experience suggests that pancreatic resection is superior to nonoperative management when the pancreatic duct is transected.

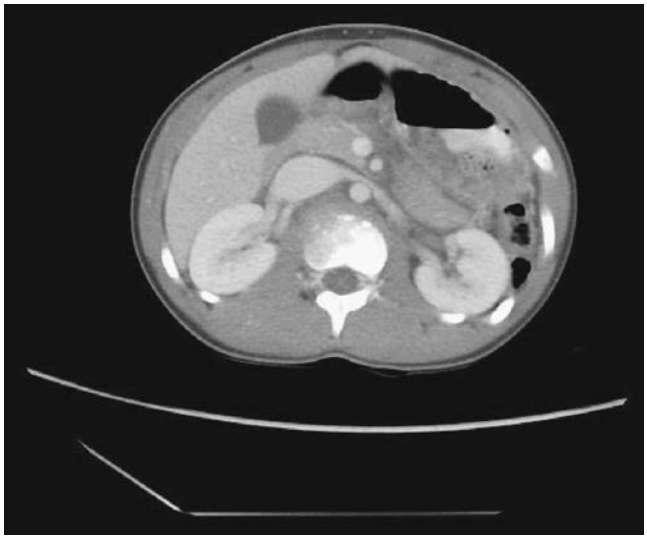

FIGURE 48-15 Transection of the mid body of the pancreas after a direct fall onto a bicycle handlebar. Major duct disruption was confirmed by endoscopic retrograde cholangiopancreatography. The child recovered promptly and uneventfully after a laparoscopic distal pancreatectomy.

KIDNEY

Blunt injuries to the kidneys are common. Some, but not all, result in gross or microscopic hematuria.[177] Contusions are the most type of blunt kidney injury. Almost all heal spontaneously, but in rare cases, massive damage to the kidney results in major bleeding that produces hemodynamic instability and requires surgical intervention, usually a nephrectomy. Four-phase abdominal CT IV contrast is the recommended diagnostic test to define kidney injuries.[178] There are limited reports of therapeutic angiographic embolization to control renal hemorrhage.[169] In some cases, a urine leak from the collecting system is noted on the CT scan. Most of these will also heal spontaneously. Ureteral stenting may facilitate healing of urine leaks. Injuries involving the renal arteries, either frank disruption or an intimal tear that impairs blood flow, are another type of rare kidney injury. These are seldom recognized promptly enough to salvage the kidney.

BLADDER

Bladder injuries are uncommon in children.[179] They typically occur in the setting of pelvic fracture or direct blows to the lower abdomen in the presence of a full bladder.[180] Injury is suggested by gross hematuria.[181] Extraperitoneal injuries generally resolve with urethral drainage. Intraperitoneal injuries require surgical repair.[179]

VASCULAR INJURY

Vascular injuries are uncommon in children. In a review of the NTDB, Barmparas et al[182] found that the incidence of vascular injury was 0.6% in children less than 16 years of age. Over the age of 16 years, the incidence increased to 1.6%. The most common site of vascular injury occurred in vessels of the upper extremity. The incidence of thoracic aortic injury was extremely low (0.03%).[182] Iatrogenic injury is most common in young children usually from diagnostic catheterization, cannulation for extracorporeal membrane oxygenation, vascular access, or arterial/venipuncture.[183] Collateralization is more rapid in infants and the very young. Therefore, a nonoperative approach with anticoagulation and observation is generally pursued in iatrogenic injury if the limb is not threatened.[184] If successful, periodic reevaluation becomes necessary to ensure that limb length discrepancy or claudication does not develop over time due to inadequate blood supply in the growing extremity.

Noniatrogenic injury increases with age. Most are penetrating. In a large civilian experience, Corneille et al[185] reported that 1.4% of injured children suffered vascular injury with a mean age of 12.7 ± 4.1 years. Nearly two-thirds sustained

vascular injuries following penetrating trauma. One-third of patients with torso vascular injury died. Primary repair was the most common technique used in arterial repair, and limb salvage was possible in 97.4%.[185] Outcome of extremity vascular injury is determined by early diagnosis and treatment. Delayed diagnosis is associated with poorer outcome. Assessment proceeds as in the adult with examination directed to hard or soft signs of vascular injury. An arterial pressure index (API) should be measured in children without absolute indications for exploration. An API less than 0.9 suggests that a vascular injury may be present, and a CT arteriogram (CTA) should be considered. CTA can be used in children with comparable results to adults.[186]

Blunt pediatric vascular injury is less common than iatrogenic or penetrating vascular injury. Supracondylar fractures are the most common cause of limb ischemia in children, with neurovascular complications reported in up to 24%. Due to collateral circulation at the elbow, obvious limb ischemia and injury to the brachial artery may not be evident. Louahem et al[187] reported a 14% incidence of ischemia in supracondylar fractures prior to reduction; 1% demonstrated delayed return of pulse after reduction, but none required revascularization. The authors suggest that anatomic reduction of the fracture, early fixation, and a conservative approach will result in good outcome.[187] In a review of the NTDB of children sustaining blunt brachial artery injury managed either with observation or arterial reconstruction, Tan et al[188] found that both approaches resulted in similar outcomes, suggesting that arterial reconstruction does not confer significantly better outcome than nonoperative management, particularly in those age 0 to 6 years. Mommsen et al[189] reported that lower vascular injury following blunt injury was associated with severe limb trauma and a lower limb salvage rate of 69%. Arterial injuries were managed with primary repair, patch, or graft for revascularization, with the most common site being the femoral artery. The impact of mangled extremity and blunt force mechanism on limb salvage is demonstrated by 100% limb salvage when mangled extremity severity score is less than 7 that falls to 33% with a score of greater than 7.[189]

Blunt cerebrovascular injury (BCVI) occurs in approximately 0.1% of adults following blunt trauma. If asymptomatic patients are screened, the incidence increases to 1%.[190] The incidence appears lower in children. In a review of the NTDB, Lew et al[191] reported that 0.03% of blunt-injured children suffered BCVI. Injuries associated with an increased risk included chest trauma (fourfold), combined head and chest trauma (sixfold), basilar skull fractures (fourfold), intracranial hemorrhage (sixfold), and clavicle fractures (eightfold), but these differ from most screening criteria used in adults. A third suffered neurologic complications. Since most asymptomatic children do not undergo screening, the true incidence is unknown. In a study of six Level I pediatric trauma centers, Azarakhsh et al[192] found that only 17% of children with one or more adult screening criteria underwent diagnostic imaging (CTA or magnetic resonance angiography), likely due to concerns for radiation exposure from imaging. Jones et al[193] reported in a series of children with BCVI that 22% developed a stroke 17 ± 6 hours after injury (range, 1–72 hours) and adult screening indications were absent in two-thirds. In asymptomatic children who underwent screening CTA and treatment with antithrombotic agents, 31 of 32 remained asymptomatic.[193] In contrast, Kopelman et al[194] found in that in children with BCVI, Eastern Association for the Surgery of Trauma screening criteria were present in 91% of the patients, and of those studied, the incidence of BCVI was 21%. As in the study by Jones et al,[193] no patient treated suffered a stroke.[194] The failure to detect the injury prior to the onset of symptoms leads to a marked increased risk of stroke, arguing that children at risk of BCVI should undergo screening. Using computer modeling, Malhotra et al[195] concluded that selective CTA in high-risk patients and selective anticoagulation are the optimal strategy in children.

For arterial reconstruction in young children, primary repair or autologous vein graft is generally preferred. For extremity vessels in the young, microsurgical repair may be necessary. Vein grafts in children are prone to aneurysmal dilatation. Prosthetic grafts may be used for larger vessel (>6 mm) reconstruction, but with growth, the graft may become a fixed point of stenosis requiring later revision. One of the largest experiences of pediatric vascular injury was accrued by the military in the management of wartime injuries in the Iraq and Afghanistan conflicts from 2002 to 2011; 155 children sustained penetrating vascular injury involving the extremity (65.9%), torso (25.4%), and neck (8.6%). Survivors with extremity vascular injury underwent vessel ligation or arterial reconstruction using greater saphenous vein or synthetic grafts. Limb salvage was achieved in 95%. The authors attributed the high limb salvage rates to damage control resuscitation, use of vascular shunts, regional or systemic anticoagulation, and liberal use of fasciotomy. A mortality rate of 18% was observed for both upper or lower extremity vascular injury, but mortality was increased fourfold in children with penetrating torso vascular injury. Good short-term results were achieved, but as the authors recognize, the long-term outcome of these children undergoing vascular reconstruction remains unknown.[196]

Successful endovascular repair of a pediatric aortic injury or other great vessel injury has been reported[150] (Fig. 48-16). Current access systems, small access or target vessels, and available stent or endograft size in conjunction with growing blood vessels that can lead to graft migration limit widespread application in children. Current limitations suggest that it is unlikely that the endovascular approach will supplant open repair except in adolescents with aortic injury, in those with access vessels and target vessels of adequate size, and in children in whom further growth is unlikely, reducing the risk of graft migration. Since the natural history of endografts or stents in children is unknown, treatment with endograft placement will necessitate lifelong surveillance.

Venous Thromboembolism

Injured children have a low incidence of venous thromboembolism (VTE) relative to adults. A decreased capacity to

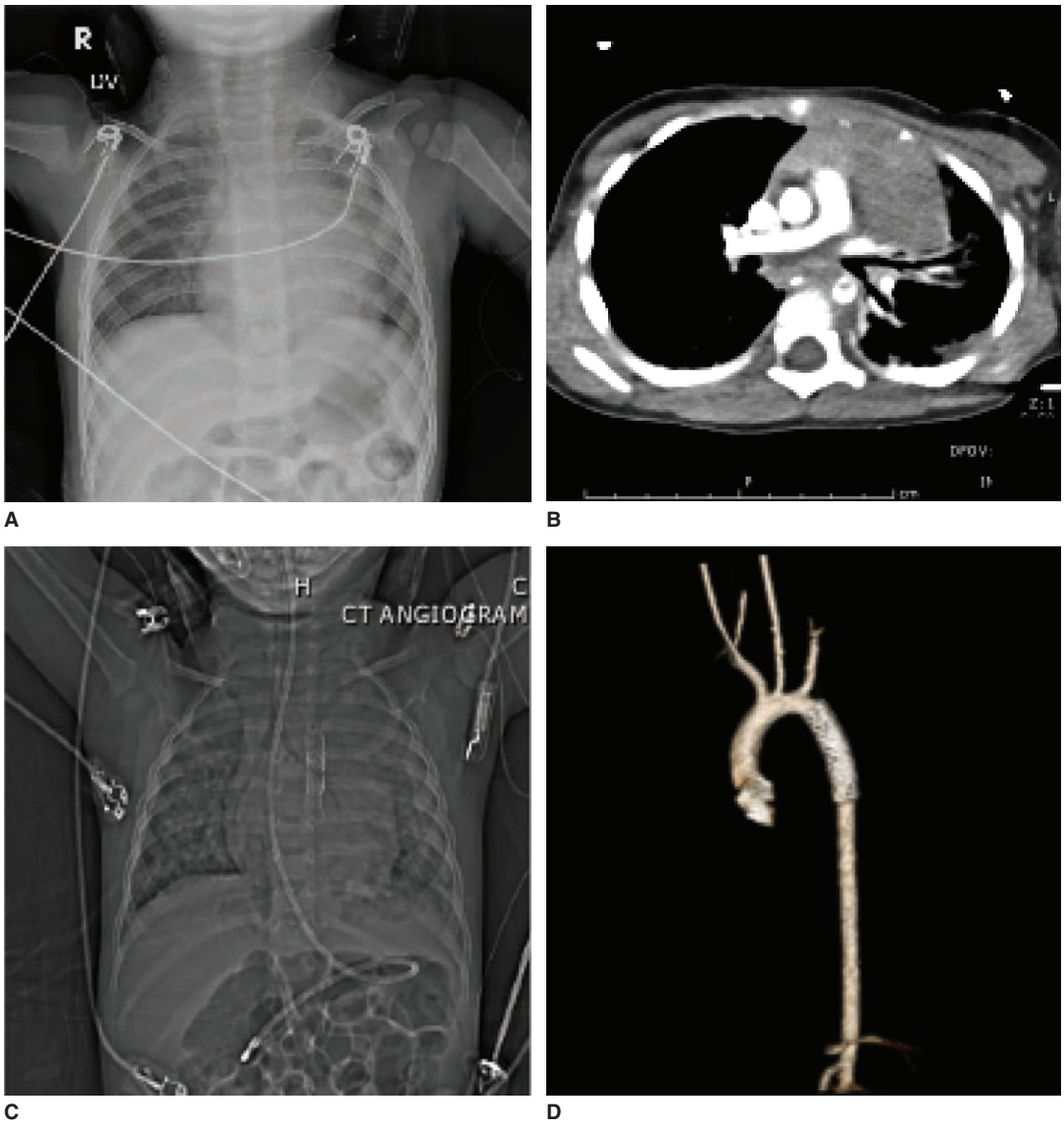

FIGURE 48-16 Two-year-old crushed by a trailer at low speed. (**A**) Chest radiograph demonstrating right rib 1 to 3 fractures and loss of the aortic knob. (**B**) Computed tomography (CT) angiogram demonstrating periaortic hematoma with dissection. (**C**) Radiograph following successful percutaneous placement of aortic stent. (**D**) CT angiogram three-dimensional reconstruction demonstrating aortic stent without extravasation.

generate thrombin, increased capacity of α_2-macroglobulin to inhibit thrombin, and enhanced antithrombotic potential by the vessel wall have been implicated as possible reasons for the low incidence of VTE during childhood.[197] The annual incidence is 5 per 10,000 hospital admissions of children.[198] Two peaks are observed, the first in the neonatal period and the second in adolescence. Teenage girls demonstrate double the rate of VTE compared to teenage boys, which is in part attributed to the effects of pregnancy or contraceptives. Central venous catheter is the most important risk factor for VTE in children, including trauma patients. Hanson et al[199] found that 67% of VTEs occurred at the site of a previous or existing central venous line. The rate of VTE in injured children is 3.3 per 1000 admissions. Children with VTE tend to be older and have higher Injury Severity Scores. Additional risk factors include thoracic injury and spinal injuries.[200] In a study of pediatric trauma patients, Van Arendonk et al[201] found that the risk of VTE was low in children less than 12 years old, but increased to 0.3% in those age 13 to 15 years and to 0.8% in those age 16 years or older. In support of this observation, Liras et al[202] measured hypercoagulability using thromboelastography maximal amplitude in trauma patients. They found that hypercoagulability and the risk of VTE increased with age in children up to age 15 years (1–12 years, 1.5%; 13–14 years, 2.3%, 15 years, 5.1%) but fell to 3.6% in those age 16 to 30 years.[202] In one study, children at high risk of bleeding were defined as those older than age 13 years with four or more of the following: projected immobility greater than 5 days, GCS less than 9, presence of a central venous line, spinal cord injury, complex lower extremity

Summary of EAST and PTS recommendations for VTE prophylaxis in children hospitalized following trauma

Question	Recommendation
1. Pharmacologic Prophylaxis in Children Hospitalized after Trauma	1. In children hospitalized after trauma who are at low risk of bleeding, we conditionally recommend pharmacologic prophylaxis be considered for those >15 y old and in younger postpubertal children with ISS >25. We conditionally recommend against the use of routine pharmacologic prophylaxis in prepubertal children, even with ISS >25.
2. Mechanical VTE Prophylaxis in Children Hospitalized after Trauma	2. In children hospitalized after trauma, we conditionally recommend mechanical prophylaxis be considered for those >15 y old and in younger postpubertal children with ISS >25 versus no prophylaxis or in addition to pharmacologic prophylaxis.
3. Active Ultrasound Surveillance for VTE in Children Hospitalized after Trauma	3. In children hospitalized after trauma, we conditionally recommend against active surveillance for VTE with ultrasound compared with daily physical examination alone for earlier detection of VTE.

FIGURE 48-17 The Pediatric Trauma Society (PTS) and the Eastern Association for the Surgery of Trauma (EAST) evidence-based recommendations for venous thromboembolism (VTE) prophylaxis for children hospitalized after trauma in 2017. ISS, Injury Severity Score. (Adapted with permission from Mahajerin A, Petty JK, Hanson SJ, et al. Prophylaxis against venous thromboembolism in pediatric trauma: a practice management guideline from the Eastern Association for the Surgery of Trauma and the Pediatric Trauma Society. *J Trauma Acute Care Surg*. 2017;82(3):627-636.)

fracture, operative pelvic fracture, use of inotropes, CPR during resuscitation, exogenous estrogen, chronic inflammatory state, history of previous clot, known thrombophilia, and current malignancy. Patients with a low risk of bleeding were administered chemoprophylaxis with low-molecular-weight heparin subcutaneously twice daily. High-risk patients in danger of bleeding underwent screening ultrasound on pediatric ICU (PICU) day 7. A decrease in total VTE and clinical VTE was observed after guideline implementation without an increase in bleeding complications.[199] Unlike adults, there are a limited number of studies in children regarding the risks and benefits of VTE prophylaxis. Applying the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) methodology, the Pediatric Trauma Society and the Eastern Association for the Surgery of Trauma proposed evidence-based recommendations for VTE prophylaxis for children hospitalized after trauma in 2017 (Fig. 48-17).[203] More definitive guidelines will likely emerge only after the conduct of clinical trials examining the benefit of VTE prophylaxis in critically ill or injured children.

Musculoskeletal Injuries

Musculoskeletal trauma is common in children and accounts for up to 25% of all traumatic injury in children. Contusions, open wounds, sprains/strains, and fractures are the leading reasons for musculoskeletal evaluations in children and adolescents, and approximately 15% will be admitted to the hospital. Of children hospitalized, Galano et al[204] found in review of the Kids' Inpatient Database that the most common injuries were femur fracture (21.7%), tibia and/or fibula fracture (21.5%), humerus fracture (17.0%), radius and/or ulna fracture (14.8%), and vertebral fracture (5.2%). Most significant injuries result from falls, motor vehicle crashes, and auto–pedestrian accidents, but in young children or infants, fractures from child abuse must be considered.[205] In children less than 18 months of age treated for lower extremity fracture in a Level I pediatric trauma center, 41 of 55 lower extremity fractures were attributed to abuse, and the most common fracture was of the femur.[206]

Pediatric bone is less dense and more porous than adult bone. The increased porosity of pediatric bone allows for greater energy absorption and decreases propagation of fracture lines, which in part explains the infrequency of comminuted fractures in children. The increased porosity, however, leads to increased susceptibility to compression force. The periosteum is thick relative to adults and minimizes fracture displacement. The periosteum may tear on the side of the fracture but remains intact on the opposite side, resulting in a unique fracture pattern not observed in adults. In a greenstick fracture, the bone is fractured with only partial disruption of the periosteum, resulting in angulation without displacement. In the torus fracture or buckle fracture, there is compression of the bone. Finally, injury can occur without visible evidence of a cortical fracture, and the periosteum remains intact but a bend in the bone is observed.

The physis or growth plate of developing bones provides longitudinal growth. It is the weakest part of the bone. Compression, avulsion fractures, or fractures involving the articular surface may occur. Up to a third of fractures in children involve the growth plate.[207] The Salter-Harris (SH) classification stratifies injury to the physis and guides management. Type I injuries are separations of the growth plate without bony fracture. Type II fractures include a variable amount of metaphseal bone with the epiphyseal fragment, and the fracture line is the same as in Type I fractures, except where it extends into the metaphysis. Type III injuries include a physeal component as in type I, with an extension of the fracture line through the epiphysis into the joint. Type IV injuries are characterized by the fracture line extending through the epiphysis, physis, and metaphysis. These injuries will result in premature arrest if not anatomically reduced. Type V injuries are crush injuries to the plate (Fig. 48-18).

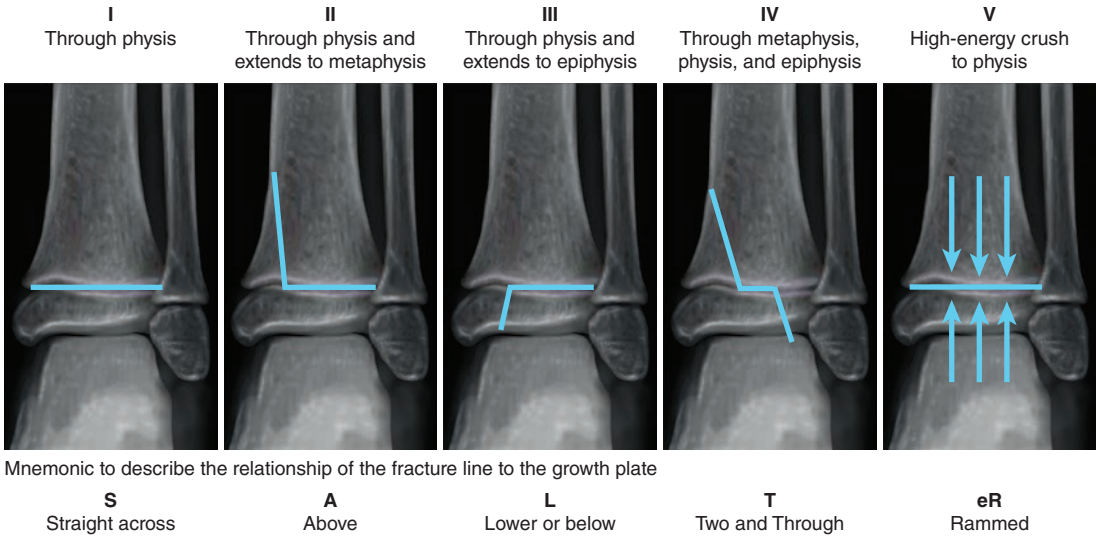

FIGURE 48-18 Salter-Harris classification of physeal fractures. (Reproduced with permission from Jones C, Wolf M, Herman M. Acute and chronic growth plate injuries. *Pediatr Rev.* 2017;38(3):129-138. Copyright © 2017 by the AAP.)

Complete injury to the physis may result in arrest of longitudinal growth. Incomplete injury may result in angular deformity. Other complications include malunion, nonunion, or avascular necrosis. SH types III and IV fractures that involve the resting layer of the physis are most at risk of growth disturbance. The most frequent sites of physeal fracture associated with abnormal growth are the distal femur, proximal and distal tibia, and distal radius. In a review of the literature, Basener et al[208] found that 52% of distal femoral growth plate fractures had some form of growth disturbance and 22% developed a leg length discrepancy of greater than 1.5 cm. SH I fractures had the lowest incidence of growth disturbance, whereas SH IV fractures had the highest rate of growth disturbance.

Examination is directed to sites of deformity, swelling, tenderness, ecchymosis, and/or soft tissue injury. Minimal findings may be present in children, and a high index of suspicion is necessary for diagnosis. In the fractured extremity, a neurovascular examination distal to the area of suspected fracture should be performed. The fracture should be splinted to reduce pain and avoid further injury. Plain radiographs are used to confirm fractures and should include the joint proximal and distal to the fracture. If there is overlying soft tissue and proximity to a joint, then involvement of the joint should be excluded. Management of the fracture may include open or closed reduction and, less commonly, skeletal traction or external fixation. The choice in treatment is influenced by age, location of the fracture, and the extent of soft tissue injury. Healing of fractures in children is more rapid than in adults. A stable union can be achieved in a neonate within 1 week. This is attributed to the thick periosteum that reduces the extent of bone injury, stabilizes the fracture, and is the major contributor to new bone formation. Children demonstrate the ability to remodel, and young children demonstrate the greatest remodeling potential. However, children are also at increased risk of overgrowth, deformity, or growth arrest, with the latter associated with injury to the growth plate.

In children with significant crush injury, ischemia, or closed fracture with significant bleeding, the extremity should be assessed for signs of compartment syndrome. Significant pain, especially with flexion, suggests the diagnosis. Paresthesia, pallor, paralysis, and pulselessness are later findings. Ferlic et al[209] reported a 3% incidence of acute compartment syndrome in children with lower extremity fracture that resulted from high-energy transfer. The time to diagnosis averaged 17 hours but was observed as late as 65 hours after injury.

Pediatric pelvic fractures are infrequent in children and account for only 1% to 2% of fractures. Pelvic fracture generally follows motor vehicle crash or auto–pedestrian accident. Children with pelvic fracture and concomitant multisystem injury demonstrate the same risk of death as adults (5%).[210] Chia et al[211] found that 78% of children with pelvic fracture had an associated injury, with a third requiring surgery. Similarly, Spiguel et al[212] reported a high incidence of associated organ injury involving two or more organ systems: neurologic, 54%; vascular, 39%; pulmonary, 31%; and genitourinary, 15%. AP radiographs and CT are used to identify the fracture and associated injuries. Hemorrhage or death from exsanguination from pelvic fracture is rare in children.[83] The use of therapeutic AE[213] and preperitoneal pelvic packing[214] to control bleeding in children with pelvic fracture has been reported. Similarly, the risk of genitourinary injury appears low. In a series of 212 children with pelvic fracture, Tarman et al[180] found that 14% had hematuria but only 2 (0.9%) of 212 sustained significant injury to the bladder or urethra. Treatment is individualized based on patient age, fracture classification, and stability of the pelvic ring. Pelvic ring injuries tend to be more stable, as the relatively thick periosteum restricts bony displacement. Most pelvic injuries in children are treated nonoperatively with weight bearing and gradual return to activity. Operative fixation is recommended for acetabular fractures with displacement, intra-articular fracture, and pelvic ring fracture with significant displacement.

CHILD ABUSE

In 1962, Kempe et al[215] published a landmark article titled "The Battered-Child Syndrome," which described a collection of injuries from repeated beatings. This article led to the mandate that all physicians should be required to report confirmed or suspected instances of child abuse.[215] Since then, the incidence of child abuse, often referred to as nonaccidental trauma or sometimes as nonaccidental injury, has increased exponentially. Annually, 3 million cases of child abuse and neglect are reported to child welfare systems in the United States each year. One-third are substantiated (around 12 per 1000 children).[216] Pediatric hospitals, especially trauma centers, receive and care for many abused children. Pediatricians with specialized training in this field are commonly asked to provide their expertise in the workup and identification of the perpetrator, potential mechanisms and outcomes of physical abuse, and long-term care. There is often a strongly associated social component. Rubin et al[217] reported that, in a cohort of abused children, 72% came from single-parent households, 37% had mothers whose age was less than 21 years, and 26% had a history of prior child welfare involvement in their families. Regardless, it is important to emphasize that the potential for child abuse is present in all socioeconomic classes. An organized approach with participation of abuse specialists, social workers, and child protective services is essential in assessing the circumstances that contributed to the child abuse and facilitate placement of the victim and siblings into a safe environment after hospital discharge. Some medical disorders may mimic child abuse, including accidental bruising, bleeding disorders, and skeletal diseases such as osteogenesis imperfecta and rickets, but these are rare.[218]

Abusive injuries have been reported for virtually every organ in the body. Infants are at the greatest risk for abusive head trauma (AHT) or shaken baby syndrome, the leading cause of fatal head injuries in children less than 2 years of age. A recent consensus report by experts in the field put to rest a number of alternative theories that have been proposed to explain some cases of suspected AHT.[219] As children age, soft tissue injury, fractures, and thoracic and abdominal visceral injuries become more prevalent. In a systematic review of children suffering abusive visceral injury, Maguire et al[154] found that abused children with visceral injury were younger (2.5–3.7 years) and more likely to die (53%) in comparison to children suffering unintentional injury (age 7.6–10.3 years; mortality 21%). Child abuse represents 3% to 4% of all cases seen in pediatric trauma centers but accounts for a disproportionate share of mortality in the very young. In one recent report from two Level I pediatric trauma centers, physical child abuse caused more than 50% of all infant trauma deaths.[220]

Clinicians must be familiar with the signs of child abuse to protect and care for those who cannot protect themselves. A thorough history and physical examination are the key to the diagnosis of physical child abuse (Table 48-3). Suspicion should be raised if there is a discrepancy between the history and the extent of injury, if there are explanations that do not fit, or if there are long periods between the time of the incident and the time to medical attention. Often the reported mechanism of injury is a fall from a crib, couch, or bed while the child was unattended. The risk of death for infants and children up to age 5 years from falls less than 1.5 m is less than one in a million.[221] Tarantino et al[222] showed that few children falling from heights of less than 4 ft suffer serious or life-threatening injury, so if a life-threatening injury is present with a history of a fall from less than 4 ft, the likely cause is abuse. Any significant injury occurring after a relatively minor mechanism should prompt further investigation. Unexplained events such as loss of consciousness should be questioned. A history of repeated trauma treated at different EDs with "doctor shopping" is suspicious. Fractures from different time periods as well as long bone fractures in children under 3 years of age should alert the clinician to the possibility of child abuse. A history that changes over time or between caregivers and inappropriate responses by the parents or other caregivers to the medical opinions given should raise concern for abuse. Finally, it is important to observe the relationship between the child and the parents to judge whether the relationship appears strained or inappropriate.

On examination, it is very important to look for and document bruising. Multicolored bruises suggest repeated injury over time. Bruising patterns can distinguish abusive from accidental injury. Pierce et al[223] found that bruising was common in both groups; however, bruises of the torso, ear, or neck in

TABLE 48-3: Components of the History and Physicial Examination Suspicious for Child Abuse

History components for physical abuse

- No explanation or vague explanation given for significant injury
- Explicit denial of trauma in a child with obvious injury
- Details of the history change
- Explanation provided is inconsistent with pattern, age, or severity of injury
- Explanation is inconsistent with child's physical or developmental capabilities
- Unexplained delay in seeking medical care
- Different witnesses provide different explanation for injuries
- Patient, caregiver, or witness gives history of inflicted injury

Physical examination components for physical abuse

- Any injury to a preambulatory infant, including bruises, mouth injury, fracture, and intracranial or abdominal injury
- Injuries to multiple organ systems
- Multiple injuries in different stages of healing
- Patterned injuries
- Injuries to nonbony locations, such as the torso, ears, face, neck, or upper arms
- Significant injuries that are unexplained
- Additional evidence of child neglect or failure to thrive
- Different forms of injury present (eg, burns, fractures)

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a child 4 years of age or younger and bruises in any region for an infant less than 4 months of age are predictive of abusive injury. Peculiar injuries such as bites, cigarette burns, or rope marks as well as sharply demarcated second- and third-degree burns in patterns that suggest forceful immersion in hot water indicate abusive injury. Other concerning injuries are those in odd places such as the perioral, genital, or perianal areas. Old scars or healed fractures on x-ray are suspicious for repetitive abuse. Long bone fractures in children younger than 3 years old, multiple subdural hematomas, especially without a new skull fracture, and retinal hemorrhages are all highly predictive of abuse. Head injury is more severe and mortality increased in AHT when compared to accidental head injury[224] (Fig. 48-19). Roach et al[119] found that children with AHT had higher rates of diffuse axonal injury, subdural hemorrhage, skull fractures, and epidural hemorrhages when compared to unintentionally brain-injured children. Moreover, mortality was threefold greater in the AHT cohort.[119] Similarly, an acute abdomen in a young child following minor trauma should arouse suspicion. Approximately 10% of abused children will suffer visceral injury, and nearly half of those will require a laparotomy due to the frequency of intestinal perforation.[155]

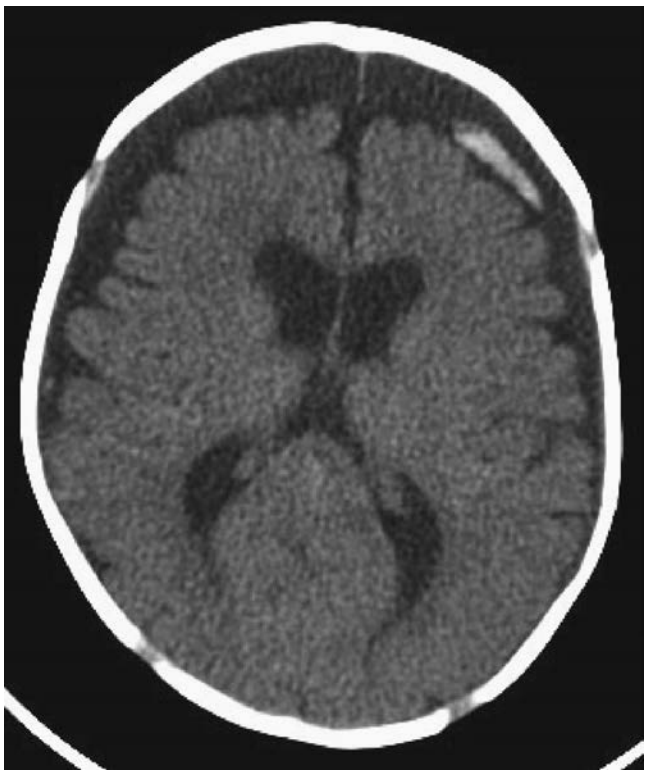

FIGURE 48-19 Axial image of a computed tomography scan of the brain performed without intravenous contrast shows large, bilateral, low-density, extra-axial fluid collections that are subdural hematomas. In the frontal aspect of the left chronic subdural hematoma, a linear focus of high density is seen, and this represents an area of acute subdural hematoma within the chronic subdural hematoma. (Reproduced with permission from D'Alessandro DM, D'Alessandro MP. How old are those subdural hematomas? December 3, 2007. Pediatric Education.org. https://pediatriceducation.org/2007/12/03/how-old-are-those-subdural-hematomas/. Accessed January 22, 2020.)

Any child suspected of suffering nonaccidental injury should undergo comprehensive evaluation that includes a skeletal survey to identify new and old fractures, careful examination of the skin, and, in the setting of TBI, a fundoscopic examination for retinal hemorrhages. CT scans and MRIs should be obtained according to clinical indications. It is important to consider that up to a third of confirmed cases of abuse will not be recognized by physicians during the initial evaluation.[225] The presence of acute respiratory compromise before admission; bruising of the torso, ears, or neck; bilateral or interhemispheric subdural hemorrhages or collections; and any skull fractures other than an isolated, unilateral, nondiastatic, linear, parietal fracture in acutely head-injured children age less than 3 years old admitted to the PICU is predictive of child abuse.[226] Examining physicians in all 50 states are required to report any case of suspected abuse to Child Protective Services (CPS). Once reported, it is up to CPS to investigate and proceed with the appropriate measures. In a series of children who survived AHT, a third were discharged to foster care (39%) or relatives (16%), but nearly a fourth of abused children were returned to their home and parents. Even more alarming, successful prosecution of the assailant occurred in less than a third of all cases despite the fact that the perpetrator was known in 42% of cases.[224]

CONCLUSION

Pediatric trauma is a major public health issue. Injury rates and deaths have declined significantly over the past 30 to 40 years, but trauma is still the leading cause of death for children in most parts of the world. Efforts should continue to expand pediatric prehospital resources and pediatric trauma centers within the United States and around the world. The spectrum of injuries and the systematic approach to the diagnosis and treatment of injured children are modeled after the care of the adult trauma victim. As in adults, most pediatric trauma patients are otherwise healthy, and a good recovery is achievable except for those with severe CNS trauma. There are many unique aspects to pediatric trauma. Most injuries are blunt. Child abuse is common in infants and toddlers. The child's airway is easily compromised. Massive blood loss may be masked by a normal or near-normal SBP. A tool such as SIPA can facilitate shock recognition. CT scans account for most of the radiation exposure of trauma patients, with the greatest risk from exposure in children.

> *"When I approach a child, he inspires in me two sentiments—tenderness for what he is and respect for what he may become."*
>
> —Louis Pasteur

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Geriatric Trauma

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KEY POINTS

- For the year 2016, the National Trauma Data Bank noted that 43% of all patients in the registry were 55 years or older and the mortality for this group was 58% of all deaths reported.
- There is evidence that immune function is significantly attenuated during the aging process and that cytokine response is impaired as well.
- In the event of hypovolemia secondary to trauma, the "stiffer" heart with atherosclerotic changes in elderly patients may be unable to compensate with tachycardia and an increase in cardiac output.
- The Trauma-Specific Frailty Index includes reviews of comorbidities, daily activities, health attitude, sexual function, and albumin level.
- In the National Trauma Triage Protocol, a systolic blood pressure of 110 mm Hg in an injured patient 65 years of age or older is a criterion for transport to a trauma center.
- When performing rapid sequence intubation in an elderly patient, the doses of induction agents may need to be reduced between 20% and 40% to minimize the risk of cardiovascular depression.
- Elderly patients on anticoagulants who have had a negative computed tomography (CT) scan of the brain after injury should have a follow-up CT of the brain before discharge from the emergency department, although the time frame for observation is controversial.
- Many of the falls in older trauma patients are from a standing height but still lead to significant injuries requiring hospitalization.
- Preexisting conditions such as cirrhosis, congenital coagulopathy, chronic obstructive pulmonary disease, ischemic heart disease, and diabetes mellitus have a significant negative impact on mortality in older trauma patients.
- Mortality in elderly trauma patients is significantly reduced in an intensivist-model intensive care unit (ICU) as compared to an open-model ICU.

INTRODUCTION

According to census projections, the elderly population in the United States, defined as age more than 65 years, is experiencing the largest growth in history. Members of the post–World War II "baby boom" (75 million people born from 1946 to 1964) were 46 to 64 years old in 2010.[1] In 2014, there were more than 40 million elderly Americans (age ≥65 years) representing 14.5% of the US population. By the year 2060, it is projected that the elderly sector of the population will more than double to about 98 million.[2]

The ever-increasing mobility and active lifestyles of today's elderly place them at increased risk for serious injury. In fact, data from the National Trauma Data Bank (NTDB) for the year 2016 revealed that 43% of all patients in the registry were 55 years old or older and the mortality for this group was 58% of all deaths reported to the NTDB.[3] Injury is now the seventh leading cause of death in the elderly population.[4]

The elderly have a higher morbidity and mortality, have more preexisting medical problems, and demonstrate a senescent physiologic response to injury when compared to younger individuals. The reasons for the differing response are unknown; the literature is contradictory in places, and there are a limited number of prospective randomized trials that focus specifically on the elderly. This is best demonstrated by a lack of consensus on the definition of what age constitutes elderly. Historically, geriatric patients were considered to be patients over the age of 65 years, as noted earlier; however, there are a variety of organ-specific injuries that demonstrate rising morbidity and mortality at chronologic ages less than 65 years. As such, elderly patients should be assessed by the

degree of frailty and viewed from the vantage of the physiologic response to an injury or injury complex rather than a specific age. Despite these limitations, this chapter focuses on an overview of care for the injured geriatric patient.

AGING

Declining cellular function is part of the aging process. Eventually, this will lead to organ failure. The aging process is characterized by impaired adaptive and homeostatic mechanisms, resulting in an increased susceptibility to the stress of injury. This is commonly perceived as decreased physiologic reserve. Insults commonly tolerated by younger patients can lead to devastating results in the elderly patient. Differences in the metabolic response to injury were studied by Frankenfield et al.[5] In their study, they compared injured patients by dividing them into those older than 60 years and those who were younger. These investigators concluded that the metabolic response to injury is significantly attenuated in the elderly population. This was demonstrated by the older group having less fever, less oxygen consumption, more hyperglycemia, and more azotemia.[5] This may be driven by the fact that there is evidence that immune function is significantly attenuated during the aging process and that cytokine response is impaired as well. This immune senescence is, in part, characterized by reduced neutrophil function. Butcher et al[6] investigated a group of patients who sustained mild trauma (hip fracture) and were older than 65 years of age. Neutrophil phagocytic function was assessed immediately after injury, and patients were then followed for 5 weeks for clinical infection. When compared to a younger cohort, the older patients had a significant reduction in neutrophil phagocytic function as measured by significantly depressed superoxide production.[6] Additionally, nearly half of the elderly population suffered bacterial or fungal infection within the study period, compared to no infection in the younger patients. These authors suggested that the defect in the aging immune system may be a result of decreasing dehydroepiandrosterone (DHEA); that is, in the presence of the physiologic stress of injury, patients have an obligatory rise in corticosterone levels, which is immune suppressive.

ORGAN FUNCTION AND AGING

Cardiac

Cardiovascular comorbidities are most frequently seen in the elderly patient. Cardiac function declines by 50% between the ages of 20 and 80 years. The declining function is combined with a decreased sensitivity to catecholamines, so the expected cardiovascular response to hypovolemia may not be apparent.[7] This may be further complicated by a variety of medications, including β-blockers.[8] The cellular elements of the conductive system and the myocytes themselves are gradually replaced by fat and fibrous tissue. The resultant less resilient stiffer heart is more prone to dysfunction and dysrhythmias. Atherosclerotic changes in the arteries are common, and valvular anatomy is changed by tissue thickening. In addition, the increased afterload causes an increase in systolic blood pressure (SBP) and enlargement of the heart. The system is generally well compensated while at rest; however, in the event of hypovolemia, elderly patients generally are unable to compensate with tachycardia and an increase in cardiac output. There is a blunted response to adrenergic stimulation as well, because there are decreased baroreceptor reflexes in response to hypovolemia. As such, the response by the aged heart is generally characterized by an increase in systemic vascular resistance. Despite "normal" blood pressure, many of these patients have evidence of tissue hypoperfusion.[9] The end result of these age-related changes is a decreased ability to respond to the stress of injury or critical illness.

Pulmonary

There are significant anatomic and physiologic changes associated with aging that occur in the respiratory system. With age-related loss in bone density, there is development of thoracic kyphosis, and rib calcification is associated with a decrease in transverse thoracic diameter. In addition, muscle mass is reduced and the elastic recoil of the lung decreases with age. These anatomic changes result in decreased compliance of the chest. This results in a reduction in functional residual capacity and gas exchange.[10] The elderly also have a decreased cough reflex, decreased function of the mucociliary epithelium, decreased response to foreign antigens, and an increase in oropharyngeal colonization with microorganisms. These changes place the elderly patient at risk for hospital-acquired pneumonia.[11]

Although the number of alveoli remains constant with aging, alveolar surface area decreases after the age of 30 years. This results in decreased alveolar surface tension, which ultimately interferes with alveolar gas exchange. The alveoli are also noted to flatten and become shallow, thereby decreasing the effective surface area for gas exchange. Diffusion capacity is decreased because of the decrease in effective surface area and an increase in alveolar-capillary membrane thickness.[12]

Renal

Above the age of 50 years, renal mass is lost as progressive sclerosis of the glomeruli occurs with a corresponding fall in glomerular filtration rate. Between the ages of 50 and 80 years, there is a decrease in glomerular filtration rate by about 45%.[7] Generally, this is not detected by routine renal function testing, as it is accompanied by a decrease in total muscle mass and production of creatinine. Measurement of creatinine clearance by serum creatinine may be lowered as a result of decreased muscle mass and can give a false indication of renal function. The Cockroft-Gault formula should be used with caution to estimate the degree of dysfunction in the basal state and should not be used in the acutely stressed patient. Furthermore, the endocrine response of the kidney to antidiuretic hormone and aldosterone is abnormal. This

results in a decrease in the ability of the kidney to concentrate urine. Elderly patients may be able to maintain a deceptively adequate urine flow despite hypovolemia. As such, urine output should be used cautiously as a surrogate for renal perfusion.[13,14]

Many elderly patients are azotemic at baseline. The normal thirst response to dehydration may be impaired, and this is especially true in patients with underlying neurologic disease. These age-related changes in renal function put the older patient at significant risk for acute kidney injury following trauma and the confounding nephrotoxic insults following injury. Likewise, the elderly are at risk for developing untoward effects of aggressive volume resuscitation such as hyperchloremic metabolic acidosis and volume overload. Care must be maintained when dosing renally excreted drugs to these patients.

Skin/Soft Tissue and Musculoskeletal System

Older people undergo an obligatory loss of lean body mass. This loss is estimated to be about 4% every 10 years after the age of 25. This loss then increases to approximately 10% after the age of 50. The loss of muscle is accompanied by a proportional increase in adipose tissue. From a skeletal aspect, osteoporosis is a common feature of aging that results in a loss of 60% of trabecular bone and 35% of cortical bone.[15] This makes the elderly individual at risk for fractures, especially those involving the vertebrae, hip, and distal forearm. There are age-associated changes of the joints and cartilages resulting in osteoarthritis and other degenerative features affecting nearly all joints, as well. Degenerative changes in the cervical spine are particularly worrisome in the older population, and mobility is greatly affected, putting this area at risk for injury, particularly during oral intubation.[16]

Skin changes with aging are well documented. As the epidermis become thinner, changes in the blunted epidermal-dermal rete pegs place patients at risk for a shear injury. There is a decrease in the skin adnexa, resulting in increased skin dryness and slower healing as well. Overall, there is decreased sensation, decreased vascularity, and impaired lymph flow, all impacting normal wound healing. Skin changes and depressed wound healing may complicate underlying fractures, specifically pelvic fractures and open fractures.[17]

Endocrine

Normal adrenal function is critical in the response to the stress of injury. Basal, circadian, and stimulated cortisol secretion remain intact with aging. There is a decrease in DHEA production, which may predispose the physiologically stressed elderly patient to a hypercortisone state (Table 49-1). Additional age-related changes that affect endocrine function include decreased production of thyroxin and tissue responsiveness as a result of senescence.[7]

Additionally, there is some evidence for age-related decrease in testosterone contributing to increased fragility in men older than age 50. Testosterone levels are significantly decreased in men older than age 70, suggesting that this testosterone change could result in lack of energy and decreased strength, which may increase an elderly male's risk for injury.[16]

Functional Reserve

Maintaining homeostasis in the face of physiologic stress is a demonstration of good functional reserve. Declining functional reserve in the elderly may precipitate a decline in performance when the patient is exposed to chronic or acute illness. When faced with a physical insult, homeostasis may be poorly maintained (Fig. 49-1). The decline in functional reserve is heterogeneous, and a variety of factors will impact the magnitude of the loss of functional reserve. These include age-related disease and their treatments, genetics, lifestyle choices, and environmental factors. It is clear that older patients do not tolerate injury as well as younger patients, and they have substantial complication rates. What is not clear is the reason for this discrepancy, as previously noted. The impact of preexisting medical problems,[18-20] in combination with a declining functional reserve, often results in a poor outcome after trauma. This impact on outcome may be reduced by early intensive care unit (ICU) admission and an aggressive management approach to the patient.

Frailty

Surgeons have intuitively recognized the impact of aging on the patient's ability to tolerate operations and trauma. Recently, there has been a growing body of literature to objectively evaluate the aged patient for risk stratification purposes.[20-22] Although we would like to be able to use functional reserve clinically, there is no standardized method for measuring and quantifying this heterogeneous parameter. There is wide variability in the age at which individuals have physiologic deterioration. Conceptually, the closer a patient is able to approach maximal organ function in the face of stress, the better the likelihood for recovery and a favorable outcome. Because functional reserve cannot be quantified, frailty is now recognized as a surrogate that can be used clinically. Frailty is recognized as a unique aspect of health status that can be a marker of decreased reserves and increased vulnerability in elderly patients. In other words, frailty is a phenotypic expression of physiologic reserve and resistance to stressors. Recently, the American College of Surgeons National Surgical Quality Improvement Project developed guidelines on preoperative assessment of the geriatric patient.[21] Included among a variety of evaluations and assessments is frailty scoring. Fried et al[22] proposed a widely accepted operational definition that is composed of five parts (Fig. 49-2).

Understanding the impact of frailty on an older trauma patient is important. It can predict discharge disposition and may have a role in goals-of-care discussions with patients and families. Investigators from the University of Arizona have developed and validated a Trauma-Specific Frailty Index (TSFI). Over a 2-year period, 200 patients were evaluated

TABLE 49-1: Organ System Changes with Aging

Organ system	Functional changes	Implications for care
Cardiac	Declining function	Lack of "classic" response to hypovolemia
	Decreased sensitivity to catecholamines	Risk for cardiac ischemia
	Decreased myocyte mass	Dysrhythmias
	Atherosclerosis of coronary vessels	Elevated baseline blood pressure
	Increased afterload	
	Fixed cardiac output	
	Fixed heart rate (β-blockers)	
Pulmonary	Thoracic kyphoscoliosis	Increased risk for respiratory failure
	Decreased transverse thoracic diameter	Increased risk for pneumonia
	Decreased elastic recoil	Poor tolerance to rib fractures
	Reduced FRC	
	Decreased gas exchange	
	Decreased cough reflex	
	Decreased mucociliary function	
	Increased oropharyngeal colonization	
Renal	Loss of renal mass	Routine renal labs will be normal (not reflective of dysfunction)
	Decreased GFR	Drug dosing for renal insufficiency
	Decreased sensitivity to ADH and aldosterone	Decreased ability to concentrate urine
		Urine flow may be normal with hypovolemia
		Increased risk for acute kidney injury
Skin/soft tissue/ musculoskeletal	Loss of lean body mass	Increase in body fat
	Osteoporosis	Increased risk for fractures
	Changes in joints and cartilages	Decreased mobility
	Degenerative changes (including c-spine)	Difficulty for oral intubation
	Loss of skin elastin and subcutaneous fat	Risk of skin injury due to immobility
		Increased risk for hypothermia
		Challenges in rehabilitation
Endocrine	Decreased production and response to thyroxine	Occult hypothyroidism
	Decreased DHEA	Relative hypercortisone state
		Increased risk of infection

ADH, antidiuretic hormone; DHEA, dehydroepiandrosterone; FRC, functional residual capacity; GFR, glomerular filtration rate.

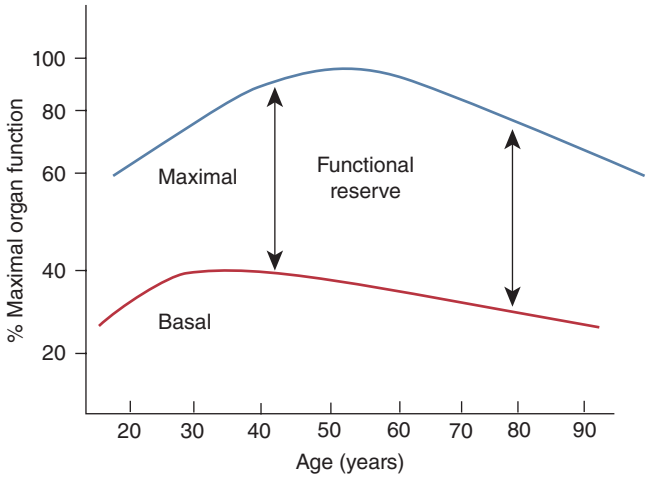

FIGURE 49-1 The functional reserve is the difference between basal function (red line) and maximal function (blue line). Even in healthy individuals, this functional reserve is reduced. (From Muravchick S. *Geroanesthesia: Principles for Management of the Elderly Patient*. St. Louis, MO: Mosby; 1997, with permission. Copyright © Elsevier.)

using their 15-variable TSFI (Table 49-2), and patients were assessed for favorable or unfavorable discharge dispositions. The investigators were able to demonstrate that the TSFI reliably predicted a patient's discharge disposition, whereas age itself was not predictive.[23] This same group enrolled 350 patients, again used the TSFI, and found that frail patients were more likely to have had a trauma-related readmission (odds ratio [OR], 1.4; 95% confidence interval [CI], 1.2–3.6) and/or repeated falls (OR, 1.6; 95% CI, 1.1–2.5) over the 6-month period. Overall 6-month mortality was 2.8% (n = 10), and frail elderly patients were more likely to have died (OR, 1.1; 95% CI, 1.04–4.7) compared with nonfrail patients.[24] Maxwell et al[25] evaluated the influence of physical frailty and cognitive decline by sampling patients age 65 years or older admitted to a Level I trauma center with a primary injury diagnosis. Surrogates of 188 patients were interviewed within 48 hours of hospital admission to determine preinjury cognitive and physical frailty impairments. Follow-up completed on 172 patients at 6 months and 176 patients at 1 year to determine posthospitalization status and outcomes revealed

Frailty score: Operational definition	
Criteria	**Definition**
Shrinkage	Unintentional weight loss ≥10 pounds in past year
Weakness	Decreased grip strength
Exhaustion	Self-reported poor energy and endurance
Low physical activity	Low weekly energy expenditure
Slowness	Slow walking

Interpretation of the frailty score

The patient receives 1 point for each criterion met.

0–1 = Not frail
2–3 = Intermediate frail (pre-frail)
4–5 = Frail

Frail patients are at much higher risk of adverse health outcomes.

Intermediate frail patients are at elevated risk (less than frail ones) but are also at more than double the risk of becoming frail over three years.

FIGURE 49-2 Frailty score: operational definition.[24] (Reproduced with permission from Chow WB, Rosenthal RA, Merkow RP, et al. Optimal preoperative assessment of the geriatric surgical patient: a best practices guideline from the American College of Surgeons National Surgical Quality Improvement Program and the American Geriatrics Society. *J Am Coll Surg.* 2012;215:453-466. Copyright © American College of Surgeons. Published by Elsevier, Inc. All rights reserved.)

that overall median physical frailty scores did not return to baseline in the majority of survivors at 1 year. Further study analysis revealed that preinjury cognitive impairment and preinjury physical frailty are independently associated with physical function (frailty).[25] This study adds additional support to the idea that the assessment of frailty in the elderly trauma patient will have an increasing role for patient care, resource utilization, and outcomes-based research.

GENERAL MANAGEMENT OF THE ELDERLY PATIENT WITH INJURIES

Triage

Improper triage seems to contribute to the poor outcomes experienced by some elderly trauma patients. The effectiveness of triage can be evaluated by looking at the interaction between injury severity and complication rate, mortality, or requirement for intervention.[26] According to the American College of Surgeons Trauma Quality Improvement Program (ACS TQIP), elderly patients have an increased risk of undertriage by both emergency medical services and emergency department personnel. Undertriage is associated with a twofold

TABLE 49-2: Trauma-Specific Frailty Index (TSFI)

Fifteen Variable Trauma-Specific Frailty Index

Comorbidities			
Cancer history	Yes (1)	No (0)	
Coronary heart disease	MI (1)	CABG (0.75)	PCI (0.5)
	Medication (0.25)	None (0)	
Dementia	Severe (1)	Moderate (0.5)	Mild (0.25)
	No (0)		
Daily activities			
Help with grooming	Yes (1)	No (0)	
Help managing money	Yes (1)	No (0)	
Help doing housework	Yes (1)	No (0)	
Help toileting	Yes (1)	No (0)	
Help walking	Wheelchair (1)	Walker (0.75)	Cane (0.5)
	No (0)		
Health attitude			
Feel less useful	Most time (1)	Sometimes (0.5)	Never (0)
Feel sad	Most time (1)	Sometimes (0.5)	Never (0)
Feel effort to do everything	Most time (1)	Sometimes (0.5)	Never (0)
Falls	Within last month (1)	Present not in last month (0.5)	None (0)
Feel lonely	Most time (1)	Sometimes (0.5)	Never (0)
Function			
Sexual active	Yes (0)	No (1)	
Nutrition			
Albumin	<3 (1)	>3 (0)	

CABG, coronary artery bypass graft; MI, myocardial infarction; PCI, percutaneous coronary intervention.

Source: Reproduced with permission from Joseph B, Pandit V, Zangbar B, et al. Validating trauma-specific frailty index for geriatric trauma patients: a prospective analysis. *J Am Coll Surg.* 2014;219:10-18. Copyright © American College of Surgeons. Published by Elsevier, Inc. All rights reserved.

increase in the risk of death. To mitigate late recognition of significant injuries, a lower threshold for trauma team activation should be used for the elderly trauma patients. In many cases, this approach would require elevating the level of activation based on age.[27] Elderly patients with severe injuries who are not treated with full trauma team activation are considered undertriaged. Multiple studies have shown a larger incidence of undertriage in older patients as compared to younger patients, so undertriage can be viewed as a modifiable risk factor for poor outcome in the older patient.[26-32] Lehmann et al[29] demonstrated that the classic physiologic criteria for trauma team activation, that is, blood pressure and heart rate, both failed to independently predict hospital mortality or the need for urgent interventions. These authors attributed the older patient's "pseudostability" to declining functional reserve and the interaction of premorbid medications. Use of initial vital signs in the elderly population can be misleading. In a study from Los Angeles, patients 70 years of age and older who were admitted to the trauma center were reviewed. The hypotension or tachycardia criteria for trauma team activation were not met in 63% of patients with an Injury Severity Score (ISS) of greater than 15% and 25% of patients with an ISS greater than 30. In this study, the overall mortality rate in "stable" patients not meeting any of the standard trauma team activation criteria was 16%.[26] The National Trauma Triage Protocol now suggests that an SBP of 110 mm Hg be used as a criterion for transport to a trauma center ("110 SBP is the new 90") for patients above the age of 65 years old. Brown et al[33] from Pittsburgh recently evaluated data from the NTDB and were able to show that the new criterion of an SBP of 110 mm Hg in patients older than 65 increased the sensitivity in predicting mortality in geriatric trauma patients. In a similar approach, the group at the University of Arizona has suggested that the Shock Index (SI = heart rate/SBP) can be used as a field triage tool for elderly patients. Using data from the NTDB, these investigators were able to demonstrate that an SI greater than 1 reliably predicted mortality with statistically better fidelity than the individual components of the index.[34]

Interestingly, "minor" injury can have major impact on the older patient. Rib fractures and pulmonary contusions can lead to an abrupt decompensation, and injuries such as intracranial hemorrhage are commonly underappreciated. It has been suggested that a patient age 70 or older be used as a criterion for trauma team activation.[26] The age at which triage and management issues become problematic is controversial also. The current recommendation of the Advanced Trauma Life Support (ATLS) program is 55 years of age.[35] This is based on data from the Major Trauma Outcome Study (MTOS), which noted a significant increase in mortality between the ages of 45 and 54 years.[36] The Trauma Injury Severity Score (TRISS) uses a similar age cutoff, although a recent work examining TRISS methodology seems to indicate that an older age is more accurate. Using the Ohio Trauma Registry, Caterino et al[30] examined mortality trends. Regression analysis identified 70 years of age to be the most promising cutoff for predicting increased odds of mortality.[30]

Initial Assessment

AIRWAY

The airway of the elderly patient poses specific challenges for providers because these individuals have a significant loss of protective airway reflexes. Patients may have dentures or be edentulous, with the former making bag-mask ventilation easier, whereas arthritic changes may make mouth opening difficult. Finally, when performing rapid sequence intubation, the doses of induction agents may need to be reduced between 20% and 40% to minimize the risk of cardiovascular depression.[37]

BREATHING

After airway control, oxygenation and ventilation are the next priority in the elderly trauma patient. The anatomic and physiologic changes in the respiratory system associated with aging are reviewed earlier. Changes in the compliance of both the lungs and the chest wall result in an increased work of breathing with aging. These changes associated with the possibility of nutritional deficits and the supine position place the elderly trauma patient at high risk for respiratory failure. Given a suppressed heart rate response to aging, respiratory failure may present in a more insidious fashion. Diagnosis can sometimes be difficult when interpreting clinical and laboratory information in the face of preexisting respiratory disease or nonpathologic changes in ventilation associated with age. Frequently, decisions to secure a patient's airway and provide mechanical ventilation may be made prior to fully appreciating underlying premorbid respiratory conditions. As noted earlier, the risk of ventilator-associated pneumonia and the possibility of prolonged ventilation are significant.[37]

CIRCULATION

Age-related changes in the cardiovascular system place the elderly trauma patient at significant risk for being mislabeled as being "hemodynamically normal." Scalea et al[38] found significant measurable hemodynamic compromise in elderly patients who were clinically stable after initial evaluation. Since the elderly patient may have a fixed heart rate and cardiac output, the response to hypovolemia will occur by increasing systemic vascular resistance. To further demonstrate the lack of classic symptoms as they relate to cardiovascular pathology, Chong et al[39] evaluated troponin I levels following emergency orthopaedic surgery in 102 patients over the age of 60, and 52.9% had elevated levels. The majority of patients with elevated troponin levels had no cardiac symptoms but had an increased mortality within 1 year of the event. Furthermore, because many elderly patients have preexisting hypertension, the seemingly "acceptable" blood pressure may truly reflect a relative hypotensive state. As such, identifying the patient who has significant tissue hypoperfusion is mandatory. Several measurements continue to be used to make this diagnosis. These include base deficit, serum lactate, age-adjusted SI, and tissue-specific end points.[10,40-42] Resuscitation of the geriatric hypoperfused patient is the same as all other patients

and based on appropriate fluid and blood administration. Fluid overload in the elderly population once adequate perfusion is established should be considered because there may be a fine line between hypovolemia and cardiac failure. The elderly trauma patient with evidence of circulatory failure should be assumed to be bleeding. Given the incidence of elderly people with preexisting disease states, however, one should keep in mind that some physiologic event may have triggered the incident leading to injury. Ultimately, an aggressive approach to resuscitation of the elderly patient with overt shock or tissue hypoperfusion should result in acceptable outcomes. Less aggressive measures based on the patient's age are not acceptable.

DISABILITY

Traumatic brain injury (TBI) is a problem of epidemic proportion in the elderly population, and older age is a known variable for a poor outcome following brain injury.[43] Age-related atrophy will cause the dura to become more adherent to the skull, causing an increased risk for subdural hematomas. Additionally, older patients are more commonly prescribed anticoagulant and antiplatelet medications for preexisting medical conditions. These two factors place the elderly individual at high risk for intracranial hemorrhage. Atherosclerotic disease is common with aging and may contribute to a primary or secondary brain injury. Moderate cerebral atrophy may permit intracranial pathology to initially present with a normal neurologic examination. This increase in potential space due to atrophy may mask the early signs of mass effect from intracranial hematoma. Additionally, age-related pupillary abnormalities may make initial evaluation of brain stem reflexes inaccurate. Early identification and timely appropriate support including correction of therapeutic anticoagulation can improve outcomes.[44]

SPINAL CORD INJURY

Traumatic injuries to the spinal column and/or spinal cord account for a significant number of traumatic injuries in the elderly population. Typically, older patients sustain an incomplete spinal cord injury (SCI), as opposed to complete injuries, likely due to the less severe mechanism of injury. Upper cervical injuries are the most common location of fractures and SCIs in the elderly population.[45] Three types of injury that appear to be relatively specific to the elderly population include (1) central cord syndrome; (2) cervical extension/distraction injuries; and (3) odontoid fractures. Management of elderly patients with spinal injuries must take into account their preexisting conditions, pathophysiologic characteristics, and age-related disorders that affect the population.[45]

EXPOSURE

Musculoskeletal changes associated with the aging process pose special concerns during the initial assessment of the elderly trauma patient. Loss of subcutaneous fat, nutritional deficiencies, chronic medical conditions, and associated medical therapies will place the elderly patient at risk for hypothermia and the risks associated with immobility (pressure ulcers, delirium). Rapid evaluation and early mobilization will minimize morbidity.

MANAGEMENT OF SPECIFIC INJURIES

Traumatic Brain Injury

In the elderly age group, TBI accounts for more than 140,000 emergency department visits each year, of which a majority result in hospitalization.[46] Falls are the leading cause of TBI for people over the age of 65 years (59.4%), followed by motor vehicle collisions (11.6%), based on the National Health Statistics Reports for 2016.[47]

Although the guidelines for the treatment of TBI have been established, they do not address the challenges of managing TBI in older patients, and research is limited. An investigation using the New York State Trauma Registry compared mortality and functional outcome in elderly versus younger patients.[48] In this study, Susman et al[48] demonstrated increased mortality in patients older than 65 years but also showed that mortality increased as patients aged. These investigators also showed that a majority of elderly patients sustain TBI from falls and appear to have only a mild TBI at admission but still have a much higher mortality compared to younger patients. This same group then looked at the total effect of age on mortality after TBI.[49] They concluded that mortality from TBI increases after 30 years of age but has a sharp rise after the age of 70. Given the anatomic changes associated with aging on the brain and the fact that a majority of elderly patients present with a Glasgow Coma Scale (GCS) score consistent with a mild brain injury, a high index of suspicion must be maintained with the elderly patient presenting with any mechanism of head trauma. Antipsychotic dopamine antagonist drugs and medications for glaucoma may affect neurologic examination, including evaluation of pupil size and responsiveness. To further address this, Mack et al[50] investigated the use of computed tomography (CT) of the brain in elderly patients. This study specifically looked at mild brain injury (GCS 13–15). In their study of 133 elderly patients, 14.3% had radiographic evidence of acute intracranial pathology. The authors noted that there were no useful clinical predictors of intracranial injury. The American College of Emergency Physicians recommends a noncontrast CT scan of the head for all patients age 65 years or older who present with a mild head injury. The use of anticoagulation in many geriatric trauma patients complicates the recommended indications for imaging. In this group, the incidence of intracranial hemorrhage is higher; therefore, a more liberal use of early CT scanning is warranted. Additionally, patients on preinjury anticoagulants should have repeat CT scanning even in the absence of intracranial hemorrhage on initial imaging if they are going to be discharged from the emergency department, although the time frame for observation and repeat imaging is controversial.[51] Outcome following TBI, most often measured as mortality, has historically been

considered to be worse in the geriatric population. Age, admission GCS, Charlson comorbidity index, Head Abbreviated Injury Scale (AIS) score, and ISS have all been found to be reliable for predicting mortality.[51] The Extended Glasgow Outcome Scale score has been reported to gradually improve for at least 1 year in most age groups after TBI, except in the geriatric population. Functional outcome and independence, cognitive function, mental health, and quality of life are important outcome measures in TBI. In a multi-institutional trial, a group of elderly patients surviving their initial moderate to severe brain injury (Head AIS = 3) were evaluated following discharge from acute care. In this cohort, there were few patients with a low GCS score who survived, and this left patients with a GCS of 13 to 15 to be evaluated. Functional outcome for these patients, as measured by the Glasgow Outcome Scale and modified Functional Independence Measure, was good to excellent. Older patients, however, required more inpatient rehabilitation and took longer to recover when compared to younger patients.[52] Aggressive initial management including inpatient and long-term rehabilitation in the elderly patient with TBI can dramatically influence outcomes.

Rib Fractures

Fractures of the bony thorax are common injuries seen in older patients. Rib fractures in the elderly pose a significant risk for morbidity and mortality when compared to younger patients who, in general, suffer little morbidity. The morbidities in the elderly include inadequate pain management, need for intubation, prolonged ventilatory support, and the development of pneumonia. Bulger et al[53] investigated the impact of rib fractures after blunt chest trauma in the elderly. This study showed a linear relationship between age, number of rib fractures, complications, and mortality. In a similar study, Holcomb et al[54] retrospectively evaluated 171 patients. These authors demonstrated an increase in negative outcomes based on increasing age and number of rib fractures. By grouping ages and number of rib fractures, their data revealed that patients with more than four rib fractures who are older than age 45 years had increased morbidity (ICU length of stay [LOS], total LOS, ventilator days, and pulmonary complications). The University of Washington Harborview Medical Center evaluated different interventions for reduction of morbidity and found the literature supported a bundled rib fracture management protocol that they developed and instituted at their facility. This rib fracture management protocol incorporates many of the tenets proposed by Todd et al,[55] but with broader inclusion criteria, early initiation of multimodal pain therapy, and frequent function-based scoring driven by nursing staff and the patient. Multivariate analysis by Todd et al[55] demonstrated that post-pathway implementation patients had significantly decreased ICU LOS, hospital LOS, pneumonia, and mortality. The recommendations for pain management from the Eastern Association for the Surgery of Trauma and Trauma Anesthesiology Society include the use of epidural analgesia and multimodal analgesia (ie, use of different classes of analgesics, including combinations of opioids with other agents such as nonsteroidal anti-inflammatory drugs, pregabalin/gabapentin, and acetaminophen) versus opioids alone to treat pain.[56] Further work is required in this area, and readers are encouraged to refer to Galvagno et al[57] on evidence-based guidelines for analgesia. In an evaluation of data from the NTDB of the American College of Surgeons Committee on Trauma, Flagel et al[58] reviewed a large patient population. These investigators showed that the overall mortality rate for patients with rib fractures was 10%. This rate increased for each additional rib fracture independent of age. A similar trend noted increasing pulmonary complications with additional rib fractures. The incidence of pneumonia in patients with up to five rib fractures ranged from 3% to 5.2%. This increased to 6.8% to 8.4% for patients with six or more rib fractures. These authors were unable to demonstrate that age is a risk factor for mortality in patients with rib fractures. Most recently, data from a multicenter study of 1621 patients were published by the Research Consortium of the New England Centers for Trauma.[37] The investigators evaluated patients over the age of 50 years with nearly isolated rib fractures. Of interest, 35% of the patients were admitted to the ICU with an average ICU LOS of 16.5 days and a total hospital LOS of 27.5 days. Intubation was required in 12% of patients, and 4.3% went on to require tracheostomy. Univariate analysis of the data revealed that risk factors for mortality were preexisting coronary artery disease or congestive heart failure (CHF), increasing age, ISS, number of ribs fractured, and increasing AIS for associated body regions. On multivariate analysis, the strongest predictors of mortality were admission to a high-volume trauma center, preexisting CHF, intubation, and increasing age. Carver et al[59] evaluated 683 patients with rib fractures and vital capacity performed within 48 hours; average patient age was 58 years old, and the median ISS was 13. The investigators found that every 10% increase in vital capacity was associated with a 36% decrease in likelihood of pulmonary complications. Additionally, the presence of fractured ribs and vital capacity of less than 30% were significantly associated with pulmonary complications.[59] Taken together, this body of literature supports the identification of elderly patients with rib fractures and early recognition of respiratory failure with aggressive supportive maneuvers to potentially reduce morbidity and mortality.

Abdominal Injury

Nonoperative management of solid organ injuries in the elderly should follow the same basic tenets of management for all hemodynamically stable patients; however, this approach must be used with caution in the elderly trauma patient. The classical physiologic response to hemorrhage, which may be used as a criterion to attempt nonoperative care or as a marker for "failure" of nonoperative management, may not be present. In a 6-year retrospective analysis of patients sustaining blunt splenic injuries, Albrecht et al[60]

evaluated the utility of nonoperative management of these injuries in patients older than 55 years. In this small study of 37 patients meeting inclusion criteria, 13 patients went directly to the operating room. Of the remaining 23 patients, nonoperative management was successful in 15 (62.5%) and failed in 8 (33.3%). Characteristics of the group that failed nonoperative management included higher American Association for the Surgery of Trauma splenic organ injury scale grade and large hemoperitoneum. In a larger retrospective study of 1482 patients, 15% (n = 224) of whom were age 55 years or older, the mortality of the older population was significantly higher than the younger group (43% vs 23%). In this study, 80% of patients over the age of 55 years were successfully managed nonoperatively, with 24 patients of the original 132 patients subsequently requiring exploration. Although not statistically significant, there was a trend toward an increased failure rate with increasing grade of injury. When evaluated by grade of injury, grade I injuries had a success rate for nonoperative management, which was similar for younger and older patients. Success rates for older patients were lower for grade II (73% vs 54%) and grade III (52% vs 28%) injuries. All elderly patients with grade IV and V injuries required operation, either immediately or for failed nonoperative management.[61] The data suggest that nonoperative management of splenic injury in the elderly patient should be undertaken with caution. An appreciation of the grade of injury and assuring hemodynamic stability are absolute requirements. Overall mortality is higher in the injured elderly than for younger patients for both operative and nonoperative management.[62]

Pelvic Fractures

Osteoporosis, falls, and fractures are major public health issues among the elderly population. Fractured bones are extremely common after trauma, and women are at particular risk because of lower bone density. Pelvic fractures pose a significant special risk for elderly patients, with challenges involving the acute phase of fracture management, timing of operation, and functional outcome. The group from the R Adams Cowley Shock Trauma Center compared outcomes of patients (<55 vs >55 years old) sustaining pelvic fractures over a 2-year period.[63] A blood transfusion was required in 62% of older patients compared to 36% of the younger population (P <.0035). This study showed that lateral compression fractures, which are generally considered less serious injuries in younger patients, occurred 4.6 times more frequently than anteroposterior (AP) fractures, and 8.2 times more frequently in older patients undergoing transfusion as compared with AP compression. Further analysis revealed that patients who received transfusion were 2.8 times more likely to be over the age of 55. Mortality for the younger group was 6.2%, compared to 20.5% for the older group (P = .0034). Even after adjusting for ISS, death was four times more likely in the elderly patients. The authors concluded that elderly patients with pelvic fractures are more likely to have substantial bleeding and transfusion requirements and require angioembolization and ICU admission. The authors suggested that every elderly patient with a pelvic fracture should be considered hemodynamically unstable until proven otherwise.

Extremity Injury

Attention to extremity injuries is a high priority in the elderly because ambulation and activities of daily living may be profoundly affected. Distal radius and proximal humerus fractures are common among the elderly population; they are the second and third most common fractures in adults over the age of 66,[64] with hip and pelvic fractures ranking number 1.[65] Hip fracture prognosis is quite poor, with 1-year mortality rates estimated to range from 20% to 30%. Among patients who were independent prior to fracture, 1 year after hip fracture, 25% remained in nursing homes and 60% required assistance in one or more activities of daily living.[65]

Consideration in the management of the elderly patient with orthopedic trauma is the timing of operation. The decision to proceed with an orthopedic operation is made based on extraorthopedic injuries, physiologic status, and the magnitude of the operation planned. Early fracture fixation should be accomplished as soon as life-threatening injuries are addressed. In a series of 367 elderly patients with hip fractures, a delay in operation for more than 2 days was associated with more than double the risk of death within the first postoperative year.[66] Current standard of care is that operative fracture fixation should occur as soon as possible when physiologic conditions have been optimized to allow earliest possible mobilization.

Burns

The elderly burn patient has a higher mortality for a given size burn than younger patients. In fact, predictors of survival from burn injury continue to rely on age as a significantly weighted variable alongside sex, percentage total body surface area (TBSA), and concomitant inhalation injury influencing elder burn victim morbidity and mortality. Despite significant advances in the science and management of burn injury, the LD_{50} (burn size with a lethality of 50% of patients) for burns in the 65 years or older population remains approximately 35% TBSA. Older individuals are at significant risk for burn injury due to impaired mobility, diminished senses, and slower reaction time, all of which make it difficult to reach safety, ultimately leading to deeper and more extensive burns.

Predicting survival in older burn patients is important. It provides a framework for discussing realistic clinical expectations with patients and their families and may allow for appropriate utilization of resources associated with the intensive care of burn patients. Wibbenmeyer et al[67] attempted to address this predictive model in relation to modern burn care. They reviewed 308 burn patients over the age of 60 years. A majority of these patients sustained flame burns, including 41.4% in household incidents. At least one preexisting medical problem was present in 64% of the patients, and the

median TBSA size was 13%. The mortality for the cohort was 30.2%, with an LD_{50} of 30%. When this was further evaluated, the LD_{50} was age dependent. Specifically, it was 43.1% for patients age 60 to 69.9, 25.9% for age 70 to 79.9, and 13.1% for age 80 and older. As expected, the presence of an inhalation injury had a significant negative impact on survival. These authors concluded that death was significantly related to age, TBSA burn, and presence of inhalation injury, whereas comorbidities did not impact mortality. The Baux score, calculated as the sum of the age of the patient and the TBSA burn, is an estimate of the percent mortality and is supported by data from this study. The Abbreviated Burn Severity Injury (ABSI) score, calculated as the weighted sum of age, gender, TBSA, percentage of full-thickness injury, and presence of inhalation injury, was also predictive of survival, but less so than the Baux score. The authors were unable to demonstrate an improvement in survival over the 20-year period of data collection.

Controversies remain regarding aggressive, early excision (24–72 hours after burn) of deeply burned tissues and the benefits of early skin grafting in terms of decrease in infections, shorter hospital stay, and early functional recovery. Ho et al[68] reported outcome data on 94 patients over the age of 60 years from their regional burn center. When compared to a younger population of burn patients, the older patients had a longer LOS, a mortality of 7.4%, and no differences in outcome based on early versus late excision. Deitch[69] had shown earlier that early wound closure decreased LOS and number of septic complications and improved mortality. The studies from Ho et al[68] and Wibbenmeyer et al[67] did not show the benefits of early excision. Finally, gender seems to impact outcomes in elderly burn patients. Chang et al[70] from the University of Utah demonstrated a higher mortality, longer LOS, and less likelihood of being discharged home for older female patients when compared to older men.

Maintaining an aggressive approach to the older burn patient is warranted in the context of survivable injuries. Advanced age, larger burns, and the presence of inhalation injury are all negative predictors of survival in this population. Realistic therapeutic expectations are important for patients, their families, and the burn team when taking care of these very challenging patients.

Falls

In 2013, 2.5 million nonfatal falls among older adults were treated in emergency departments, and more than 734,000 patients were hospitalized. In addition, approximately 25,500 older adults died from unintentional fall-related injuries in the same year.[71] For older trauma patients, many of these falls are from a standing height but still lead to significant injuries requiring hospitalization.[72] Preexisting medical conditions, medications, and other variables play a significant role in ground-level falls. Unfavorable discharge dispositions and mortality are significant in this group of patients. The group from Harborview Medical Center examined 1352 elderly patients admitted to their trauma center after a ground-level fall. Deaths occurred in 12% of the patients during the index admission. Of the survivors, 50% were discharged to a skilled nursing facility (SNF), and only 6% were discharged to home. Nearly 45% of patients were readmitted within 1 year following discharge, with patients requiring an admission to the ICU during the index admission to be at highest risk for readmission. The overall 1-year mortality for the entire group, including those who died during the index admission, was 33%. Patients discharged to an SNF were three times more likely to die than patients discharged home.[73]

Because the societal and financial impact for older adults sustaining falls is significant, developing effective prevention measures is mandatory. Optimizing medications to prevent hypotension and hypoglycemia; encouraging physical exercise to prevent osteoporosis and prevent skeletal muscle loss; instituting environmental modifications, such as grab bars and clutter removal; and reducing environmental hazards all play a significant role in preventing falls. Fall prevention programs aim to detect individuals at high risk for falls and to remove those risk factors that can lead to falls in the elderly.

Penetrating Trauma

With the growth in the proportion of elderly people, there will be an obligatory rise in the number of elderly trauma patients. By far, blunt trauma is the predominant mechanism for injury; however, there are still a significant number of people over the age of 65 years who are victims of penetrating injury. Most of these patients are victims of self-inflicted injuries mediated by chronic illness and depression. When examining patients over the age of 55 between the years 1982 and 1987 in the original MTOS, elderly patients had a higher mortality when compared to a younger cohort.[74] In addition, Finelli et al[75] reported a significantly higher mortality in the elderly (52%) compared to younger patients (20%) sustaining gunshot wounds. In contrast, Roth et al[76] from Los Angeles County Hospital and the University of Southern California retrospectively reviewed 79 patients over the age of 55 years who suffered penetrating injuries. Their data showed no difference in mortality between elderly and younger patients (23% vs 18%). Interestingly, 50% of the elderly patients who died presented with "normal" vital signs.[76]

Elder Abuse

The discussion of geriatric trauma would not be thorough without considering elder abuse. Elder abuse includes not only physical violence, but also acts of neglect. This may include withholding food, medications, hygiene, or monetary or emotional support. A high index of suspicion is important because signs and symptoms of elder abuse may be attributed to chronic diseases. On the secondary survey of the elder trauma patient, a complete history and physical examination noting unusual features should be documented. Findings that should arouse suspicion for abuse may include delays in seeking treatment, coming to medical attention without the caregiver, a caregiver who dominates the conversation, unkempt

appearances, unusual wounds such as multiple bruises, "glove and stocking" injuries, and genital injuries.[77]

CRITICAL CARE

In a large retrospective statewide trauma registry review of 22,571 patients with blunt trauma, of whom 7117 were elderly, ICU utilization was evaluated.[78] The entire population had an ICU admission rate of 42.7% with a mean ICU LOS of 5.77 ± 8.86 days. In contrast, the elderly patients had a lower ICU admission rate of 36.7% when compared to the younger population (45.5%). Interestingly, elderly patients admitted to the ICU had a significantly longer ICU LOS. The lower utilization of ICU resources in the elderly may be explained by a higher early mortality and/or end-of-life decisions that may have precluded admission to an ICU setting.

Preventable complications in the elderly trauma patient significantly impact outcome. DeMaria et al[79] demonstrated an association of complications with death (32%) and an association of deaths from multisystem organ failure (62%). The relationship between senescence and infection deserves mention. Bochicchio et al[80] showed an increase risk of infection in elderly patients (39%) when compared to a younger population (17%) (P <.05). Once infected, the elderly patient is at much higher risk for death (28%) than his or her younger counterparts (5%) (P <.005). An aggressive approach to the older patient with infection is essential for improved outcomes.

Scalea et al[81] suggested that early invasive monitoring of the geriatric trauma patient can improve outcome. Noninvasive monitoring of elderly patients has been investigated in the critical care setting, but little has been concluded regarding the critically ill trauma patient.[82] There is a movement in emergency medicine and critical care as well as surgery toward use of point-of-care ultrasound. Murthi et al[82] looked at the utility of cardiac ultrasound to evaluate hemodynamics and resuscitation. In this study, these investigators compared focused rapid echocardiographic evaluation, a hybrid of the point-of-care examination and transthoracic echocardiography, versus assessment using a vascular catheter. They were able to show that the echocardiographic assessment correlated well with data from a pulmonary artery catheter, with the benefit of noninvasiveness and real-time evaluation after therapeutic interventions. Although not specifically studied in elderly patients, this noninvasive method will likely become a mandatory diagnostic technique for intensivists.

Because the elderly will fail to demonstrate the classic physiologic responses to shock, it is important to maintain an aggressive approach toward monitoring and resuscitation. The utility of invasive monitoring and aggressive critical care is demonstrated in a study on outcomes of elderly patients with the acute respiratory distress syndrome (ARDS). In this study, Eachempati et al[83] evaluated a protocol approach to ARDS using lung protective ventilator strategy and invasive hemodynamic monitoring. In a group of 210 elderly patients with ARDS, the investigators were able to demonstrate a lower mortality when compared to historical controls despite a higher severity of illness.[83]

Delirium in the elderly trauma population is now recognized as a significant risk factor for morbidity and mortality in the ICU. Delirium is characterized as a disturbance of consciousness associated with a fluctuating mental status and disorganized thought, occurring in at least 20% of hospitalized patients over the age of 65 years. In addition, it increases hospital costs by $2500 per patient, resulting in $6.9 billion of Medicare hospital expenditures.[84] There is also a threefold higher mortality over 6 months following a single episode of delirium in the ICU.[85] Understanding the impact of delirium in the trauma patient has only recently been investigated. Pandharipande et al[86] undertook a prevalence study of delirium in their trauma and surgical ICUs. Using the Confusion Assessment Method for the ICU (CAM-ICU), a validated screening tool, these investigators found an overall prevalence of delirium of 70% (73% in surgical patients and 67% in trauma patients).[86] The Intensive Care Delirium Screening Checklist is another validated tool for use in ICU patients. This scale assesses delirium symptoms over an 8- to 24-hour period because delirium typically fluctuates throughout time, and the "spot" nature of the CAM-ICU assessment may miss an episode of delirium if the symptoms are not present at the time of assessment. The group from the trauma center at Denver Health Medical Center performed a 4-month study of 69 patients admitted to their trauma ICU following injury. They found a 59% incidence of delirium overall and a higher incidence if the patient was mechanically ventilated. Upon univariate analysis, age proved to be a predictor for the development of delirium; however, on multivariate analysis, the strongest predictors for transitioning into delirium were lower arrival GCS, higher packed red blood cell transfusion amount, and higher multiple organ failure score.[87] Most recently, a two-center study examining the impact of delirium on outcomes was performed. A total of 134 patients were evaluated, and 63% progressed to delirium during their ICU stay. The patients with delirium had more ventilator days and longer ICU and hospital LOS. These investigators were unable to show a relationship between increasing age and the development of delirium.[88] The association of advanced age with delirium is strong in most studies, and delirium and dementia are highly interrelated; however, the nature of this interrelationship remains poorly examined.[84] The association of delirium with opioid narcotics and benzodiazepines is well established, and care must be taken in prescribing these medications to all patients but especially the elderly. In addition, the issue of appropriate and goal-directed analgesia and sedation is commonly faced by the ICU staff. Finally, delirium represents one of the most common preventable adverse events among older persons during hospitalization,[89] implying that the development of delirium in a patient may reflect processes of care in the hospital, and, therefore, may be a reflection of the quality of care of that individual. Those caring for elderly injured patients must be aware of the development, diagnosis, prevention, and, if needed, appropriate treatment of delirium in order to provide the highest quality of care to this population.[90]

OUTCOMES

Injured elderly patients admitted to the hospital consume significant health care resources. Average hospital LOSs are generally around 10 days, and usually the LOS in the ICU is longer than for younger patients (except for early deaths). In addition, hospital-acquired complications are independent predictors of mortality in this age group. In 2002, Richmond et al[91] reported their 10-year retrospective review of geriatric trauma from a statewide trauma registry. They evaluated all patients age 65 years or older who were included in the registry. Nearly 62% of the patients were injured as a result of a fall, whereas 22.6% were injured in a vehicle collision. Operation was required in 28% of patients, and 37% of the cohort had a preexisting medical condition. The average LOS was 11.5 days, one-third of the group required admission to the ICU, and 10% of all patients died. These investigators found that as age increased, the patients had higher mortality and more complications and more required discharge to a facility other than home. The patients who died had a greater number of injuries with more body regions involved and a resultant higher ISS.

Admission physiologic markers may predict those at risk for mortality. In a statewide trauma registry review from Pennsylvania, elderly patients who were hypotensive, had a GCS of 3, or had a respiratory rate of less than 10 all had a significantly increased risk of death.[78] The nine-center Prognostic Assessment of Life and Limitations After Trauma in the Elderly (PALLIATE) consortium has validated the Geriatric Trauma Outcome Score (GTOS) as a prognosis calculator for injured elders. A total of 10,984 subjects were included, with the identified subjects age 65 to 102 years admitted from 2000 to 2013. GTOS was specified using the following formula: [GTOS = age + ISS × 2.5) + 22 (if transfused packed red blood cells at 24 hours)]. Subjects in the GTOS validation study showed that increasing age and injury severity both contributed to mortality. The penetrating trauma subgroup (n = 150) had a higher mortality rate, at 20.0%.[25]

Although the literature is not unanimous concerning factors that correlate with mortality or poor functional outcomes, several items that have generally been shown to be predictive have been described. These include advanced age, increasing severity of injury, shock, prolonged mechanical ventilation, male gender, significant head injury, and sepsis. Trauma also appears to have not only short-term but also long-term effects on elderly survival. A study by Xu et al[92] found that survival time for seriously injured older adults was significantly shorter when compared with survival times for the minor and no injury groups in the elderly. The study used an integrated version of the public use National Health Interview Survey (NHIS) produced by the Integrated Health Interview Series (Minnesota Population Center and State Health Access Data Assistance Center, 2016) consisting of participants age 65 years and older. A total of 79,504 older adults were used to estimate mortality risk. There was a clear difference in mortality risk by injury status. More than 38% of seriously injured older adults died within 11 years, 27.9% of those with minor injuries died, and 24.9% of those with no injury at baseline died. The survival time for the minor injury group was also significantly shorter than for the non-injury group. The probability of death reached 25% by 14 quarters (or 3.5 years) after baseline for seriously injured older adults, by 21 quarters (or 5.25 years) for patients with minor injuries, and by 26 quarters (or 6.5 years) for the non-injured group. At 44 quarters, or 11 years, the probability of survival dropped to 40.3% for seriously injured older adults, 52.6% for older adults with minor injury, and 55.9% for non-injured older adults.[92] In summary, elderly trauma patients are at greater risk for mortality, prolonged institutional care, lack of return to preinjury status, and reduced midterm survival.

PREEXISTING CONDITIONS

The interaction between injury and patient factors has been studied extensively, but data are conflicting regarding the impact of these factors on mortality. Despite this, there is a large body of evidence documenting that preexisting conditions (PECs) will impact morbidity and, likely, mortality. Morris et al[20] identified five PECs that appeared to influence outcomes. In that study of over 3000 patients, one-fourth of patients over the age of 65 years had one of the five PECs. These investigators identified cirrhosis, congenital coagulopathy, chronic obstructive pulmonary disease, ischemic heart disease, and diabetes mellitus as PECs that influenced outcomes in trauma patients. Patients with one or more of these PECs were nearly two times more likely to die than those without PECs. The same authors then reported the interaction between injury and host factors, which included age, gender, and PECs.[19] Although injury severity was the primary determinant of mortality, host factors also played a significant role. These studies were later corroborated in a study by Grossman et al,[18] in which an ISS greater than 30 was considered the LD_{50}. Each additional year of age greater than 65 years carried a mortality increase of 6.8%, and PECs with the greatest impact on mortality were hepatic disease, renal disease, cancer, and CHF.[18] The impact of PECs was demonstrated in a study from the group at Vanderbilt University in which they studied a predictive mortality model incorporating PECs (University Health System Expected Mortality [UHC-EM]) and compared it to TRISS methodology. The UHC-EM is derived from data on diagnosis-related groups and includes both anatomic injury and PECs, whereas TRISS uses only unadjusted vital signs and pattern of injury. The study was a 7-year retrospective review of all patients admitted to the Vanderbilt Trauma Center, and elderly patients were compared to a younger cohort. The UHC-EM was able to predict mortality better than TRISS in elderly patients, demonstrating the impact of PECs.[93]

COMPLICATIONS

There seems to be a relationship between PECs and the development of postinjury complications that increase mortality. In a study reviewing a large statewide trauma registry

by Taylor et al,[78] 6.2% of elderly patients developed pneumonia, with preexisting pulmonary disease and increased ISS as risk factors. Pneumonia predicted both an increased ICU and hospital LOS. In this study, 5.9% of patients developed an acute kidney injury, which resulted in a greater than 10-fold increase in mortality. Finally, the development of sepsis, which occurred in only 1.2% of patients, significantly increased mortality, ICU LOS, and hospital LOS. The only risk factor identified for the development of sepsis was an increased ISS.[78] The impact of failure to rescue in the elderly has been investigated in a large database of emergency surgical patients. The failure to rescue rate was twofold higher in the elderly compared to younger patients, correlating with a similar disproportionate increase in mortality.[94] For elderly trauma patients who survive their initial injury, cardiovascular, pulmonary, and infectious complications are common and contribute significantly to mortality. Complications are a significant sequela in this group of high-risk patients.

RESOURCE UTILIZATION

The association between age as an independent predictor and outcome as documented by increased mortality and hospital LOS is well supported in the literature. Although hospital LOS is increased, ICU utilization is less than in a younger population.[78,95] As previously noted, this is due to an increased early mortality seen in the elderly as well as decreased ICU admission resulting from advanced directives. Data from the Healthcare Financing Administration demonstrated that ICU use decreased with age and was least likely for patients over 85 years old.[96] Young et al[97] looked at outcome in a cohort of elderly trauma patients. Although the older group had a higher mortality rate, there were no differences in hospital or ICU LOS or in ICU admission rate. In the study by Taylor et al,[78] the elderly group had a lower ICU admission rate (36.7%) compared to younger trauma patients (45.5%); however, elderly patients, once admitted to the ICU, had a longer ICU LOS. The impact of withholding or withdrawing care may influence ICU LOS.

STRATEGIES FOR IMPROVING OUTCOMES

Triage

There are numerous studies suggesting that elderly trauma patients are "undertriaged" (underestimation of injury severity) to trauma centers despite the proven mortality benefit of trauma center care. Statewide data from Maryland demonstrated that fewer elderly trauma patients were transported to trauma centers despite meeting physiologic criteria for trauma triage.[98] The group of undertriaged elderly patients had a significantly higher mortality, discharge disability rate, and complication rate when compared to a group of younger patients. Meldon et al[99] demonstrated a significant survival benefit for severely injured elderly patients when treated in a trauma center (56%) versus a nontrauma center (8%).[99] Undertriage of older patients could be due to inadequate training, unfamiliarity with protocol, and possible age bias; however, assessment of this group is less likely to demonstrate hypotension or tachycardia than are younger patients, as previously noted.

Medications

The increased awareness in medication reconciliation and its impact on patient safety is prominent in current health care literature. The acuteness of the hospital admission and, at times, the initial underappreciation of significant anatomic injury result in a potentially modifiable situation highlighting the importance of knowledge of a patient's premorbid medications and the potential interaction of any newly prescribed medications. The Beers criteria are used by consensus for safe medication use in elderly patients.[100] Using this system along with expert input from geriatricians and pharmacists may reduce adverse drug reactions in older patients.

There are a number of medications specifically related to the injured patient that deserve mention. β-Blockers are used in approximately 20% of elderly patients with coronary artery disease and 10% of patients with hypertension. The inherent physiologic blockade of the expectant response to hypovolemia may provide obstacles to triage and treatment. Additionally, the impact of prolonged β-blockade on the progression and outcome of trauma and burn injury is not fully understood.

Anticoagulant medications include warfarin, heparin, low-molecular-weight heparin, the antiplatelet agent clopidogrel, and anti-Xa inhibitor oral anticoagulants. Aspirin and nonsteroidal anti-inflammatory medications may also cause coagulopathy. Anticoagulation with warfarin and/or antiplatelet therapy poses significant problems for the bleeding patient. Warfarin therapy carries a 1% per year risk of spontaneous intracranial hemorrhage even in the absence of trauma.[101] Assessment of the therapeutic level is important because many patients will be sub- or supratherapeutic. Many studies assessing the impact of warfarin on trauma and, specifically, trauma to the brain are limited in that they do not address the clinical level of anticoagulation. To address this limitation, a study of 3242 trauma patients over the age of 50 years was performed. In this retrospective analysis, data on the international normalized ratio (INR) were used as a surrogate for warfarin anticoagulation. These investigators demonstrated a mortality rate of 22.6% in the group with an INR greater than 1.5 compared to 8.2% for those with an INR less than 1.5. After adjusting for age and ISS, the odds of death for each one-unit increase in INR was 30%.[101] Patients with supratherapeutic anticoagulation fare even worse. For reversal of warfarin, prothrombin complex concentrate (PCC) is now recommended over fresh frozen plasma (FFP). The use of PCC leads to shorter time for normalization of the INR, reduction of clinical progression of intracerebral hemorrhage, and reduced mortality rates as compared with treatment with FFP.[102]

Antiplatelet therapy is less well studied. This class of drugs includes aspirin and clopidogrel and is increasingly used

for a wide variety of cardiovascular problems. In a registry review of patients over the age of 50 years who sustained a TBI, patients taking aspirin or clopidogrel were compared to those not receiving antiplatelet therapy. The group receiving antiplatelet therapy had a mortality of 23% compared to 8.9% (P = .016) in the control group. Risk factors for death in this study included age greater than 76 years and GCS less than 12.[103] Peck et al[104] from San Diego retrospectively looked at a group of elderly patients with blunt TBI. They compared patients taking anticoagulants and/or antiplatelet medications at the time of injury with patients not taking such medications. The anticoagulation/antiplatelet group had a higher mortality, and the authors attributed this to the use of antiplatelet agents, with the most frequently used medication being clopidogrel.[104] Patients sustaining a TBI while taking clopidogrel have been shown to have progression of intracranial hematomas (ICHs). Joseph et al[105] retrospectively reviewed 71 patients with an ICH who were taking clopidogrel at the time of injury. There was a fivefold increase in progression of ICH and a twofold increase in progression of ICH based on clinical deterioration. These authors suggested routine repeat head CTs in patients with an ICH who had taken clopidogrel.[105] Management of TBI patients on preinjury antiplatelet agents remains controversial. One randomized trial of platelet transfusion in patients with intracranial hemorrhage demonstrated improved outcomes, whereas other studies have failed to show an impact of platelet transfusion on clinically relevant outcome measures.[51]

Rapid reversal of anticoagulation with transfusion of FFP is beneficial. Reversal of an elevated INR to normal within 2 hours reduced mortality from 48% to 10% in one study.[106] Fortunately, and contrary to initially expected results, large recently published series showed that trauma patients receiving new oral anticoagulants had a better outcome than those anticoagulated with vitamin K antagonists.[107] Reversal of newer oral anticoagulant agents, which include factor Xa inhibitors and direct thrombin inhibitors, is not well established because there are limited data. This highlights the need for specific screening protocols for coagulopathy in this setting. The Neurocritical Care Society and the Society of Critical Care Medicine have assembled guidelines intended for use by clinicians providing care to intracranial hemorrhage patients exposed to antithrombotic agents. This 13-person group analyzed the quality of literature evidence and provided treatment recommendations based on the Grading of Recommendations Assessment, Development and Evaluation (GRADE) system. This guideline addresses commonly encountered antithrombotic-associated intracranial hemorrhage scenarios for which data and experience have been reported. Refer to the summary of recommendations for further information.[108]

Specialists

The role of the geriatrician in the management of the elderly trauma patient is logical. There are age-specific nuances in the care of the elderly that make the geriatric specialist an invaluable member of the extended trauma team. There have been publications noting improvement in outcomes of geriatric trauma patients when a geriatrician became involved in their care.[106] Fallon et al[109] described the idea of a "geriatric trauma team." This involved mandatory consultation from a group of interested and dedicated geriatricians. The consultants were able to address a variety of medical conditions in elderly trauma patients at study sites. These included pain management, rehabilitation, delirium, and advanced care planning, to name a few. Most notably, the group of patients seen in consultation had a lower mortality than a group of elderly trauma patients not seen by a geriatrician. To study this concept prospectively, Lenartowicz et al[110] from Toronto established a Geriatric Trauma Consultative Service (GTCS) that was composed, at a minimum, of an advanced practice nurse in geriatrics and a geriatrician who performed a comprehensive geriatric assessment. All patients age 60 years and older were evaluated by the GTCS. The authors looked at geriatric-specific complications (falls, delirium, and restraint use) and trauma-specific complications (pressure ulcers, venous thromboembolism, myocardial infarct, pneumonia, cardiac arrest, and missed injury). There were less consult requests to internal medicine and psychiatry, and ultimately, there was less delirium and a decreased incidence of patients being discharged to long-term care facilities.[110]

There are age-specific interventions that may improve outcome in the elderly injured population. As previously noted, one of the first attempts to address this was work from the trauma service at Kings County Hospital Center in Brooklyn, New York. In this study, the authors were able to demonstrate an outcome benefit from early invasive monitoring and intervention.[81] A study from Nathens et al[111] investigated the impact of an intensivist-model ICU on trauma-related mortality. In this large multicenter study, the authors concluded that an intensivist-model ICU when compared to an open-model ICU significantly reduced mortality. Furthermore, the greatest risk reduction was seen in the elderly population (relative risk, 0.55; 95% CI, 0.39–0.77).

End of Life

The ICU is an environment in which end-of-life decisions are made on a regular basis. In fact, 38% of all hospitalized patients who die in the United States spent at least 10 days in the ICU in one review.[112] Older patients, having an understanding of their own wishes regarding health care, frequently use advance directives and health care proxies. Often, patients know these legal entities through interaction with an astute family physician or by way of a previous elective hospital admission. Understanding the logistics of these documents is mandatory when caring for trauma patients. Given the acute onset of trauma and a patient's inability to predict future injury, trauma practitioners must rely on a written document that may have been written years prior or by interactions with a health care proxy. The health

care proxy is an individual who is appointed by a competent adult. In the event that the individual is no longer able to make health care decisions, the proxy would "speak" on behalf of the patient. There is evidence that physicians frequently have differing opinions regarding a clinical outcome compared to the health care proxy, as well as personal beliefs that can influence end-of-life decisions.[112] Withholding and withdrawing life support is commonly practiced in ICUs. Common reasons for this are futility, patient suffering, and the anticipation of a poor quality of life.

Futility is a concept with a distinct ethical definition. Unfortunately, the term is applied frequently in situations that are strongly influenced by the local environment and culture. What may be viewed as futile by one physician may not be seen the same way by another. As such, utilization of an ethics consultation will be important. The perception of suffering of elderly trauma patients is a cause of emotional distress for the ICU staff.[113] The ubiquitous use of analgesics and sedatives in the ICU is necessary, but the science is imprecise. Most surgical ICU patients who survive indicate that they would repeat the experience again if necessary.[114] End-of-life decisions based on this argument are not indicated. Using the argument of a poor quality of life is commonly based on the perceptions, beliefs, and personal interpretation of quality of life of the health care providers. Care must be taken to not interject one's own beliefs when counseling families. Most ICU survivors indicate that they have an acceptable quality of life and would undergo treatment again.[115] Data on the quality of life following ICU treatment after injury are not widely available. Limiting care in the elderly trauma patient in whom survival is unlikely is necessary. Importantly, this care at the end of life may not be consistent with patient preferences and values and may place unnecessary emotional, physical, and financial burden on dying patients and their family members. Using standard ethical concepts and precise diagnoses will help facilitate discussions with families. Palliative care consultations have been used successfully with trauma patients, which can improve family and staff satisfaction with end-of-life care.[116] There is emerging evidence that advance care planning early during an acute care hospitalization can reduce ICU admissions and that time-limited trials and proactive early palliative care consultation in the ICU can reduce the LOS and intensity of treatment for patients who die in the ICU.[117]

ORGAN DONATION

Donation of organs from the elderly requires "expanded criteria" that are specific to the Organ Procurement Organization. From San Diego and Imperial Counties, patients older than 60 years without hypertension or a serum creatinine greater than 1.5 mg/dL can be considered for donation. In the interval between 2009 and 2013, 11% to 14% of annual donors from the San Diego and Imperial County Organ Procurement Organization were older than 60 years.[118] Thus, it seems reasonable to consider otherwise healthy seniors as suitable candidates for organ donation.

CONCLUSION

As the elderly population continues to grow and lead an active lifestyle, the trauma surgeon will be increasingly called upon to treat injured geriatric patients. An understanding of the unique anatomic, physiologic, and psychiatric aspects of the aging adult is mandatory. This, coupled with an appreciation of a variety of PECs, the impact of medications, and respect for advanced medical decisions, should form the basis of our approach to care.

Early identification of the critically injured elderly patient, physiologically based resuscitation, and timely intervention for correctable injuries will optimize utilization of resources, decrease complications, and improve outcomes in this unique and special population.

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IV

SPECIFIC CHALLENGES IN TRAUMA

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Wounds, Bites, and Stings

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KEY POINTS

- The four phases of wound healing are hemostasis, inflammation, proliferation, and maturation/remodeling.
- Significant influences on wound healing include age, nutrition, body habitus, and social history.
- The decision to use antibiotics for a wound will depend on host factors (eg, age, comorbidities, immunocompromised state), wound characteristics, and location.
- Wounds with significant contamination, ischemia, or tissue loss are left open to heal by secondary intention.
- Transmission of HIV or hepatitis C through saliva from a bite is rare, and HIV testing after human bites is not necessary.
- Jaws of larger dogs are capable of generating 200 to 450 pounds per square inch of pressure, and fractures under the injury to soft tissue should be ruled out.
- Rabies is a vaccine-preventable viral disease primarily caused by the bite of a rabid animal and has a worldwide distribution.
- In up to 40% of patients, the bite of a brown recluse spider will lead to necrosis of soft tissue due to local ischemia.
- Pit vipers including rattlesnake, water moccasin, and copperhead snakes have a heat-sensing pit located behind their nostrils.
- Although the mouth of a snake is colonized with bacteria like all animals, local infection following a snakebite is rare.

INTRODUCTION

Millions of patients are treated for traumatic wounds annually. These wounds are managed by a variety of practitioners, including surgeons, specialty surgeons and physicians, and advanced practice providers. The majority of wounds are straightforward and heal satisfactorily regardless of the technique used; however, there are wounds that require special care or attention, which will be discussed in this chapter. Wound management priorities include healing without infection, maintenance of normal function, and acceptable cosmesis. The simplest technique that will meet these requirements should be employed in treating a wound and will vary with the nature of the wound, its anatomic location, and the clinical situation.

WOUNDS

Wound Healing

Wounds, whether created electively or as a result of traumatic injury, are an integral component of the surgical patient; thus, understanding the pathways and mechanisms of wound healing is critical for the optimal care of surgical patients. This fund of knowledge is applicable to the care of patients with acute wounds and facilitates the development of therapeutic options for patients with chronic or nonhealing wounds. Wound healing is often divided into phases in order to aid understanding of the complex process. These phases are hemostasis, inflammation, proliferation, and maturation and remodeling (Table 50-1). Although these phases are often described as discrete events, it is important to realize that characteristics and elements of these phases overlap. Wound repair is a dynamic and complex process of inflammation characterized by a well-coordinated pattern of cell migration, proliferation, and differentiation, along with angiogenesis and matrix remodeling. The essential characteristics of the early phase include hemostasis and inflammation. The intermediate phase is characterized by cell proliferation, migration, angiogenesis, and epithelialization, whereas the late phase involves collagen production with contraction of the wound. Ultimately, all wounds undergo continuous remodeling for the rest of the patient's life.

TABLE 50-1: Phases of Wound Healing and Activity

Stage	Predominant cells	Activity	Time course
Hemostasis	Platelets	Vascular congestion Platelet plug formation	Immediate
Inflammation	Neutrophils	Clearance of necrotic tissue/microbes	Hours 6–48
	Macrophages	Wound debridement Angiogenesis	Hour 48–day 4
	Lymphocytes	Matrix synthesis Cell immunity/antibody production Lymphokines regulate collagenase	Day 5
Proliferation			Days 4–12
Angiogenesis	Endothelial cells	Vascular growth	Hour 48–day 4
Fibroplasia	Fibroblasts	Matrix/collagen synthesis	Day 3–3 months
Epithelialization	Epithelial cells	Wound coverage	Variable
Maturation/remodeling	Fibroblasts/myofibroblasts	Matrix deposition Collagen remodeling Vascular maturation/regression	Day 21–1 year

A chapter on wounds would not be complete without expansion on the four phases of healing briefly described earlier. The first phase, or hemostasis (coagulation) and inflammation phase, occurs to minimize hemorrhage from disrupted blood vessels. Hemostasis precedes inflammation. Vasoconstriction occurs from the effects of vasoactive mediators (epinephrine, norepinephrine, prostaglandins, serotonin, and thromboxane). Adherence and aggregation of platelets and the activation of the coagulation cascade result in the generation of fibrin, leading to clot formation. Activated platelets degranulate and release chemotactic and growth factors from α granules, such as platelet-derived growth factor (PDGF), transforming growth factor-β (TGF-β), platelet-activating factor (PAF), serotonin, and fibronectin.

The inflammatory phase is characterized by increased vascular permeability, chemotaxis of cells from the circulation into the wound, release of cytokines and growth factors, and the activation of migrating cells. Inflammatory cells are attracted by chemokines released by platelet activation. Vasoconstriction dissipates and a period of vasodilation commences mediated by histamine, prostaglandins, kinins, and leukotrienes. Blood flow is increased, and inflammatory cells (neutrophils, macrophages, lymphocytes) and factors to mitigate infection are abundant in the wound. The predominate cell in the first 24 to 48 hours, the neutrophil, releases the inflammatory mediators and bactericidal oxygen free radicals, decreasing bacterial counts in the wound. Macrophages, derived from circulation monocytes, migrate into the wound and peak at 3 days, and persist until wound healing is complete. Macrophages phagocytize debris and bacteria and secrete collagenases, elastases, and cytokines. These cytokines further stimulate chemotaxis and proliferation of fibroblasts and smooth muscle cells. Endothelial cells are attracted to the wound by further macrophage substance release for promotion of angiogenesis. Additionally, cytokines mediate nitric oxide synthesis, which has antimicrobial properties and stimulates collagen synthesis.[1,2]

T-lymphocytes are attracted to the wound around day 5 by the cellular release of interleukin (IL)-1 from neutrophils, playing a role in cellular immunity and antibody production, in addition to secretion of lymphokines (heparin-binding epidermal growth factor and fibroblast growth factor). IL-1 also contributes to the regulation of collagenase activity.

The proliferative phase occurs from about day 4 to day 12, overlaps with the inflammatory phase, and includes epithelialization, fibroplasia, angiogenesis, and contraction. Revascularization occurs in parallel with fibroplasia. Healthy tissue adjacent to the wound sprouts capillary buds that extend into the wound. Endothelial cells from the side of the venule closest to the wound begin to migrate in response to angiogenic stimuli. The newly formed venules diverge along one of two pathways; they either differentiate into arterioles or undergo apoptosis and ingestion by macrophages. Mediators of angiogenesis, endothelial cell growth, and chemotaxis include cytokines produced by platelets, macrophages, and lymphocytes; low oxygen tension; wound acidemia; and high lactate levels.[3,4] Fibrin proliferation and collagen deposition replace the provisional fibrin matrix and provide a stable extracellular matrix at the wound site consisting of collagen, proteoglycans, and fibronectins. This activity is regulated by PDGF and TGF-β, with the latter having a central role in wound healing.[5] Granulation tissue formation deep in the wound provides a transitional replacement for normal dermis. It consists of a dense network of blood vessels and capillaries, elevated cellular density of fibroblast and macrophages, and randomly organized collagen fibers. Epithelialization is occurring at the wound surface and begins within a few hours of injury. Epithelial cells arising from the wound margins and dermal epithelial appendages become activated and migrate over the viable connective tissue. Basal keratinocytes lose their tight

attachments and begin to migrate across the wound. The migration of these edge basal cells during wound closure has been described as being akin to cells "leapfrogging" over one another, rather than crawling across the wound to cover the defect.[6,7] As the migrating epithelial cells advance and begin to cover the wound, proliferation begins to ensure that sufficient cells are available to cover the wound. This is one of the areas of wound healing that is impeded by steroids. It has been shown that glucocorticoids impede epithelial growth factor (EGF)–mediated cellular migration.[8] Once the wound has been bridged, the migrating epithelial cells change shape to become more columnar, followed by surface keratinization.[9] Epithelialization is complete in approximately 48 hours in approximated wounds, but completion of epithelialization can depend on the size and depth of the wound, tissue defect, and contamination or infection. Investigations remain ongoing regarding the signals or stimuli that bring the proliferative phase of wound healing to an end.

Collagen formation and deposition are critical to the reconstructive phase of wound healing, and transition to the remodeling phase of wound healing is delineated by a state of collagen equilibrium. Fibroblasts are also responsible for the production of other matrix constituents, including fibronectin and the glycosaminoglycans.[10] Wound collagen synthesis is increased for at least 4 to 5 weeks.[11,12] Initially, there is an increase in type III collagen followed by a decline and increased deposition of type I collagen, the dominant fibrillar collagen in skin.[13] Wound breaking strength, as compared to uninjured or normal tissue, is 3% at 1 week, approximately 20% at 3 weeks, 50% at 4 to 6 weeks, and if healing occurs without complications, 80% at 10 to 12 weeks, and has no further increase thereafter. The tensile strength of a wound will never reach that of uninjured tissue.[14-17] Increased collagen turnover continues for at least several years.[18]

The final phase a wound undergoes is the remodeling or contraction from the edges of the wound to the center in a centripetal fashion, resulting from a complex interplay between fibroblast and the extracellular matrix material. During reepithelialization, the wound edges progress toward each other at a rate of 0.6 to 0.75 mm/d, and the rate of contraction is dependent on a wide variety of factors. The correct balance between too little contraction, which leads to nonhealing wounds, and too much contraction, which leads to contractures, is important for optimal healing. New research is questioning longstanding theories on the mechanism of wound contraction. Modified fibroblasts with smooth muscle (SM)–like features, including the expression of α-SM actin (ACTA2), were first observed by Gabbiani and colleagues in granulation tissue of healing wounds. This led to the theory that myofibroblasts promote wound contraction and collagen production.[19,20] Subsequent in vitro studies demonstrated that myofibroblasts generate increased contractile force as compared to fibroblasts, and that the mechanism is related to ACTA2 incorporation into stress fibers and increased focal adhesion size.[21] ACTA2 levels were found to associate with a fibroblast's ability to wrinkle a deformable substrate and enhance collagen gel contraction.[22] The compelling hypothesis that myofibroblasts are essential for wound contraction is balanced by a counterhypothesis that myofibroblasts are not essential for wound contraction. This counterhypothesis is supported by studies in human sacrococcygeal pilonidal sinus wounds noting contraction without a high density of myofibroblasts being present; studies showing that full-thickness excisional wounds in rats capable of unwounded wound contraction in the absence of myofibroblasts, fibroblasts, and myofibroblasts produce similar contractile forces; studies demonstrating ACTA2 absence in free-floating collagen lattice contraction; studies showing that suppression of ACTA2 expression in rodents treated with vanadate did not alter wound contraction; and most recently, two studies demonstrating that ACTA2 expression is not necessary for wound closure.[23,24] Thus wound contraction mechanisms remain an active area of research for approaches to promotion of healing in chronic wounds or reducing scar contractures and fibrosis.

Patient Factors That Affect Wound Healing (Table 50-2)

The speed of wound healing and tensile tissue strength are affected by the patient's overall health status and the specific structure or organ that is wounded or injured. Skin and fascia are the strongest tissues in the body but regain tensile strength more slowly than other tissues. The viscera, stomach, and small intestine have weaker tensile strength but progress faster through the healing cycle, and in the colon, the speed is similar but the tensile strength varies from the cecum to the sigmoid. Patient factors are important in the discussion of risks of wound closure and complications.

The overall health or the disease state of the patient has a significant influence on wound healing, including age, nutrition, body habitus, and social history.[25]

AGE

The thinning of the skin and tissues in the elderly predisposes them to injury. Age and coexisting chronic disease

TABLE 50-2: Patient Factors That Affect Wound Healing

Age
Gender
Weight
Nutritional status
Hydration status
Alcohol use (acute and chronic)
Smoking tobacco (nicotine, carbon monoxide, hydrogen cyanide)
Immune responses (cancer, AIDS, chemotherapy, catabolic steroids)
Chronic disease (diabetes mellitus, cirrhosis, chronic kidney disease)
Radiation exposure
Ischemia/hypoxemia
Specific structure or organ injured

slow the phases of wound healing. Every phase of healing undergoes age-related changes, which includes enhanced platelet aggregation, increased secretion of inflammatory mediators, delayed infiltration of macrophages and lymphocytes, impaired macrophage function, decreased secretion of growth factors, delayed reepithelization, delayed angiogenesis and collagen deposition, reduced collagen turnover and remodeling, and decreased wound strength.[26] Additionally, sex hormones play a role in age-related wound healing, with studies showing that aged males have further delays when compared to aged females. Estrogen has been found to improve age-related impaired wound healing in both men and women, whereas androgens negatively regulate cutaneous wound healing.[27]

OBESITY

Many of the wound issues surrounding obesity result from increased wound tension and pressure combined with perfusion deficits and hypoxemia resulting in ischemia in the subcutaneous tissues. Obesity is also associated with altered immune and inflammatory responses with discoveries of induced changes in adipose tissue that may initiate and perpetuate a chronic low-grade inflammatory state. Phenotypic switching of protective M2 macrophages to proinflammatory M1 macrophages exacerbates the inflammatory process. Adipose tissue itself excretes angiogenic inhibitors and fibrotic mediators, thus creating extracellular matrix that prevents angiogenesis and further adding to hypoxia. There are supportive findings that obesity-related changes in cellular and immune function are improved by weight loss.[25,28]

NUTRITION

The primary sources of energy for wound healing include fats and carbohydrates. The major fuel for adenosine triphosphate (ATP) synthesis is glucose. Protein is a source of energy for capillary formation, fibroblast proliferation, proteoglycan synthesis, collagen synthesis, and wound remodeling. The semi-essential amino acid arginine is required during periods of maximal growth, severe stress, and injury, and the most abundant amino acid is a major source of metabolic energy for rapidly proliferating cells. Glutamine is critical in the inflammatory stage of wound healing. β-Hydroxy-β-methylbutyrate (HMB), a metabolite of the essential amino acid leucine, is responsible for inhibiting muscle proteolysis, modulating protein turnover, and enhancing collagen deposition.

Vitamins C, A, and E have antioxidant and anti-inflammatory effects important to wound healing, and deficiencies result in impaired wound healing. Micronutrients such as magnesium, zinc, and iron are also important in acute and chronic wound healing.

The nutrients needed for wound healing are complex, suggesting that nutritional support including supplementation of these critical components may benefit wound healing. A summary of studies has reported that high-dose supplementation of zinc and vitamin C, in addition to a regular diet, may support the healing of surgical wounds in healthy subjects, and formulations enriched with glutamine and antioxidant micronutrients may support the healing of traumatic wounds and can improve the healing of burns.[29] Glutamine supplementation has been shown to improve wound breaking strength, increase levels of mature collagen, and decrease complications in colorectal and colon cancer treatment.[30] The combination of arginine, glutamine, and HMB has been shown to increase hydroxyproline levels and improve bursting pressure in a colon anastomosis model, improve muscle strength following surgery, reduce acute inflammation from radiation, and impact secondary wound healing in ischemic and diabetic wounds. There are few studies, however, evaluating the use of this combination in acute wound healing models, and recent studies question the benefit of these supplements in the critically ill population.[31]

ALCOHOL AND SMOKING

The exposure to ethanol is most apparent in the proliferative phase of wound healing with most effects on angiogenesis, collagen production, alterations in protease balance at the wound site, and impairment of the early inflammatory response increasing the incidence of infection.[32] Smoking affects wound healing in the proliferative and inflammatory phases. The inflammatory healing response is attenuated by a reduced inflammatory cell chemotactic responsiveness, migratory function, and oxidative bactericidal mechanisms. In addition, the release of proteolytic enzymes and inhibitors is imbalanced. The proliferative response is impaired by reduced fibroblast migration and proliferation in addition to downregulated collagen synthesis and deposition.[33]

Priorities in Wound Management

The management of acute traumatic wounds should be thought of in a three-phase decision algorithm, and while progressing through the algorithm the wound should be protected from further injury and contamination (Fig. 50-1).

The first phase of wound management includes an assessment of the injured patient directed toward identifying and rapidly treating life-threatening injuries. The most readily apparent wound is not always the most important injury that will need to be addressed. Even the most dramatic of wounds should not distract the physician from first addressing fundamental life-threatening injuries. Advanced Trauma Life Support principles should be applied with purposeful attention to the primary ABCDEs (airway, breathing, circulation, disability, exposure) followed by a directed secondary survey to establish appropriate priorities. If there is ongoing hemorrhage contributing to the patient's current or potential hemodynamic stability, techniques may include direct pressure, packing with plain gauze or hemostatic agents, pressure dressings, and temporary or partial wound closure of scalp, face, junctional, truncal, and extremity wounds, as well as reduction and splinting of fractures. Finally, potential junctional or extremity tourniquet placement may need to be considered for extremity wounds with hemorrhage that is uncontrolled

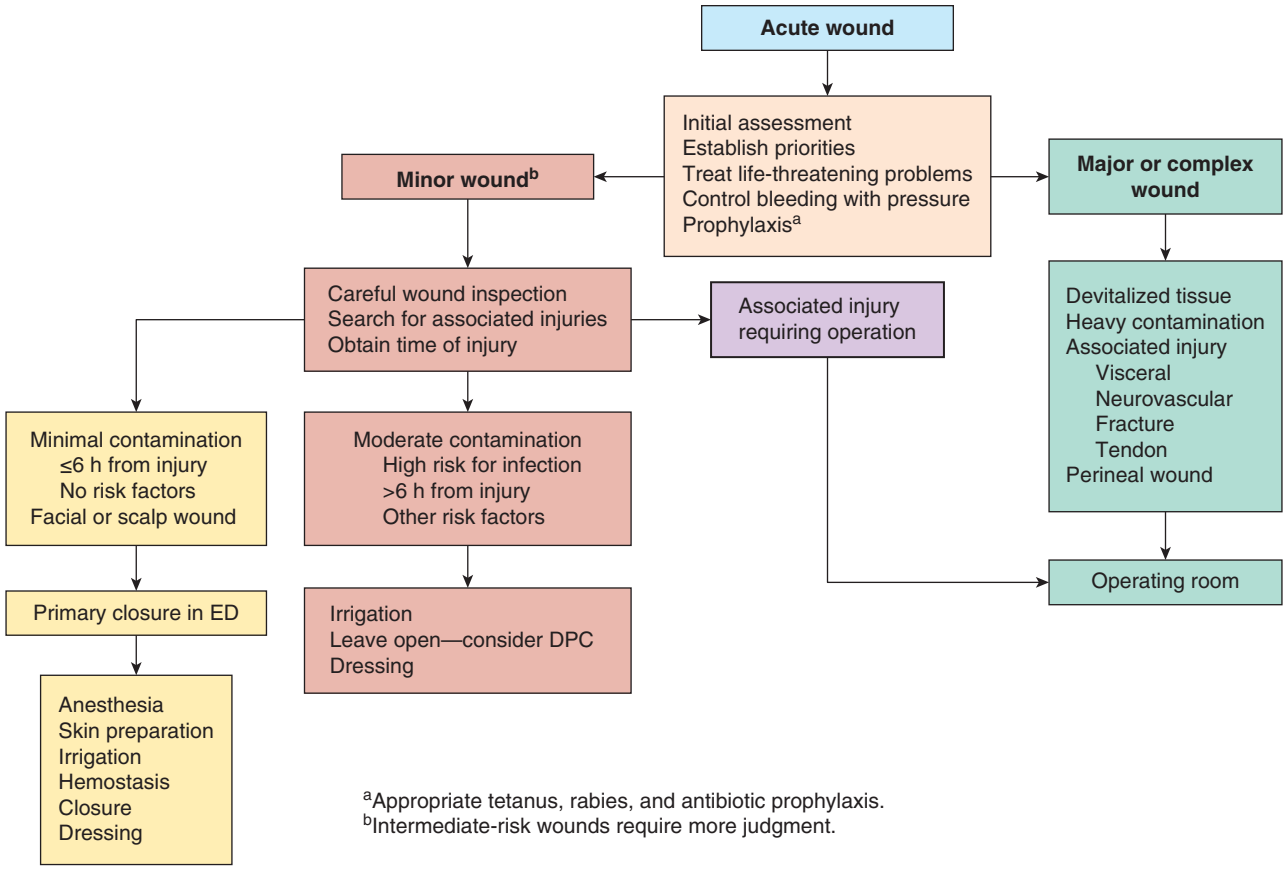


FIGURE 50-1 Algorithm for the treatment of an acute wound. DPC, delayed primary closure; ED, emergency department.

with these prior methods. Hemorrhage from scalp lacerations may lead to significant anemia and shock, especially in the elderly and children. This may be prevented with pressure dressing application, hemostatic temporary closure, or application of disposable or reusable Raney clips. Junctional tourniquets for axilla and inguinal hemorrhage control are in trials in the military and prehospital settings,[34] and current widespread training is ongoing regarding the indications and application of tourniquets for extremity trauma.[35]

Once life-threatening injuries are treated or are being treated, the second phase of the wound management algorithm concerns how to deal with the wound itself. Key questions to be considered in this phase are as follows: (1) Should the wound be managed in the operating room? (2) Are there associated injuries related to the wound (fractures or injuries to tendons, fascia, muscle, nerves, or vascular injuries)? (3) Can the wound be closed primarily, or should it be managed by delayed primary closure or by healing through secondary intention? (4) Should preventative antibiotics be given? (5) Does the patient need tetanus or rabies immunizations and/or prophylaxis?

The third phase of the algorithm relates to techniques of closure and the type of dressing. This is the last phase due to the utmost importance of the first two phases. Almost all truly poor or bad outcomes related to management of wounds can be linked to errors of judgment rather than to technique.

Wound Management in the Operating Room Versus the Emergency Center (Table 50-3)

Factors influencing the decision about whether or not to manage a wound in the operating room relate to the size of the wound, degree of contamination, presence of devitalized or ischemic tissue, anatomic location, associated injuries, and patient-specific factors.

The associated injury to an adjacent structure or cavity may be more important than the wound itself. The goals of traumatic wound management are to avoid infection, assist in hemostasis, and provide the safest closure technique; in addition, it is also important to consider the aesthetics of the wound.

Classification of Wounds

Surgical site infection events occurring following an operative procedure that takes place in an operating room are reportable events within the Centers for Disease Control and Prevention National Healthcare Safety Network. Important to this reporting is the initial classification of the wound. Wounds are divided into four classes, as listed in the following sections.

TABLE 50-3: Conditions for Wound Management in the Operating Room

Large or complicated soft tissue injury
Extensive amount of necrotic or ischemic tissue
Heavy contamination
Associated injury
Vascular
Tendon or fascia
Nerve
Fracture
Viscera
Intra-articular
Perineal wounds
Compartment syndrome
High-pressure injection injuries
Patient factors
Age
Length of sedation
Volume of topical anesthetic
Physiologic status
Mental/psychological status

CLEAN

A clean wound is an uninfected operative wound in which no inflammation is encountered and the respiratory, alimentary, genital, or uninfected urinary tracts are not entered. In addition, clean wounds are primarily closed and, if necessary, drained with closed drainage. Operative incisional wounds that follow nonpenetrating (blunt) trauma should be included in this category if they meet the criteria.

CLEAN-CONTAMINATED

Clean-contaminated wounds are operative wounds in which the respiratory, alimentary, genital, or urinary tracts are entered under controlled conditions and without unusual contamination. Specifically, operations involving the biliary tract, appendix, vagina, and oropharynx are included in this category, provided no evidence of infection or major break in technique is encountered.

CONTAMINATED

Contaminated wounds are open, fresh, accidental wounds. In addition, operations with major breaks in sterile technique (eg, open cardiac massage) or gross spillage from the gastrointestinal tract and incisions in which acute, nonpurulent inflammation is encountered, including necrotic tissue without evidence of purulent drainage (eg, dry gangrene), are included in this category.

DIRTY OR INFECTED

Dirty or infected wounds include old traumatic wounds with retained devitalized tissue and those that involve existing clinical infection or perforated viscera. This definition suggests that the organisms causing postoperative infection were present in the operative field before the operation.

Management of the Wound

Once the critical associated injuries have been excluded or addressed, the decision is made about adjuncts to wound management, such as antibiotics and tetanus prophylaxis, skin preparation, irrigation methods, and type of wound closure. Additionally, the history should document the following: the event, environment, and time of the injury, and location, width, length, and depth, including the tissues involved such as subcutaneous fat, fascia, muscle, tendon, bones, nerves, and vascular structures. The appearance of the wound edges, wound bed, and the neurovascular and functional status should also be documented.

When deciding if antibiotics are needed, several factors need to be considered, including host factors such as age, comorbidities, and immunocompromised states, and wound characteristics and location (Table 50-4). In regard to the latter, the thigh and leg have the highest infection rate, whereas the scalp has the lowest (Table 50-5).

A meta-analysis conducted to determine whether prophylactic systemic antibiotics prevented infection in patients with nonbite wounds managed in the emergency department found that patients treated with antibiotics had a slightly greater incidence of infection compared to untreated controls (odds ratio, 1.16; 95% confidence interval, 0.77–1.78). Thus, from the nine randomized trials analyzed, it was concluded that there is no evidence the prophylactic antibiotics offer protection against infection of nonbite wounds in patients treated in emergency departments.[36] However, patients with wounds with high risk for infection due to location, contamination, tissue destruction, delayed presentation, and high-risk patient factors should receive antibiotics

TABLE 50-4: Characteristics That Indicate Need for Antibiotics

Host characteristics	Wound characteristics
Age	Crush/devitalized
Comorbidity	Stellate laceration
Diabetes mellitus	Contamination
Chronic kidney disease	Retained foreign body
Obesity	Delayed presentation
Malnutrition	Exposed tendon, joint, bone
Cirrhosis	Location
Prosthesis (heart, joints)	Intraoral
Immunocompromised	Bites
Illness	
Cancer	
Medications	
AIDS	
Lymphedema	

TABLE 50-5: Wound Location and Infection Rates

Location	Infection rate (no.)
Arm/forearm	15.3% (157)
Back	8.3% (12)
Chest/abdomen	11.8% (17)
Ear/nose	3.6% (28)
Face	3.9% (383)
Foot/toe	12.5% (21)
Hand/finger	5.7% (192)
Scalp	1.7% (233)
Thigh/leg	23.0% (87)

Source: Adapted with permission from Lammers RL, Hudson DL, Seaman ME. Prediction of traumatic wound infection with a neural network-derived decision model. *Am J Emerg Med*. 2003;21(1):1-7. Copyright © Elsevier.

at presentation and potentially an additional dose prior to debridement and closure if this time is prolonged from over the half-life of the initial dose (Table 50-6). The less time that elapses between injury and the administration of antibiotics, the lower is the risk of infection, especially in open fractures. Patients with open fractures receiving antibiotics within 3 hours of injury had decreased infection rates compared to those who received antibiotics beyond 3 hours (4.7% vs 7.4%).[37] In most settings, a first-generation cephalosporin, antistaphylococcal penicillin, or macrolide is adequate. If the wound has an intraoral component, a penicillin is appropriate. For more austere contamination and unusual incidents involving lakes, ponds, rivers, natural disasters (earthquakes, tornadoes), or agricultural or combat injuries, more resistant organisms should be considered, including nonfermenting gram negatives (*Acinetobacter*, *Pseudomonas*), *Enterobacter*, *Serratia*, and yeast or fungi (mucormycetes, *Apophysomyces*, *Aspergillus*). In these situations, additional agents such as quinolones, extended-spectrum penicillin, and antifungals should be considered and tailored to postculture findings and sensitivities.[38,39]

Tetanus immunization should be reviewed when a patient presents with an acute injury or wound, and prophylaxis administered as indicated (Table 50-7). Administration of the diphtheria–tetanus–acellular pertussis (DTaP) vaccine is routinely recommended in adolescents, with a single booster dose of a vaccine containing tetanus toxoid, reduced diphtheria toxoid, and acellular pertussis (Tdap, Adacel, or Boostrix 0.5 mL intramuscularly [IM]) recommended for 11- to 12-year-olds, followed by tetanus toxoid and the reduced diphtheria toxoid in the form of Td recommended at 10-year intervals throughout life. In the United States, a tetanus and diphtheria toxoids (Td) vaccine (0.5 mL IM) is recommended every 10 years for all adults with complete prior immunization against tetanus and diphtheria. The Advisory Committee on Immunization Practices also recommends a single dose of a vaccine containing tetanus toxoid, reduced diphtheria toxoid, and acellular pertussis (Tdap) for all adults age 19 years and older who have not received Tdap previously. When indicated, Tdap should be administered regardless of the interval since the last dose of Td. The subsequent Td booster can be resumed 10 years after the Tdap administration. Adults who have not been previously vaccinated against tetanus and diphtheria should receive a series of three vaccines (one Tdap and two Td vaccines).[40] The preferred schedule is a Tdap vaccine dose followed by a Td vaccine dose 4 weeks later and another Td vaccine dose 6 to 12 months later. In addition to tetanus immunization, human tetanus immune globulin is indicated in individuals who have sustained a wound that is more severe than a clean and minor wound (eg, wounds contaminated with dirt, feces, soil, or saliva; puncture wounds; avulsions; wounds resulting from missiles, crushing, burns, or frostbite) and who have either received fewer than three doses of tetanus toxoid previously or in whom the number of previous doses is unknown.

The process and type of skin preparation for wound closure must be considered to decrease trauma to the wound and decrease the incidence of infection. Use of a depilatory or clippers to remove hair from the area is preferred over shaving to decrease infection[41] and foreign body contamination to the wound. If the wound can be closed without disruption of the surrounding hair or tissue, that would be most preferable; if not, limited removal for adequate closure is acceptable. The most common antiseptics—povidone-iodine solution (betadine), chlorohexidine gluconate solution (Hibiclens), and hydrogen peroxide—are all cytotoxic, have antibacterial activity, and have been shown to inhibit wound healing. These wound antiseptics should be restricted to the intact skin surrounding the wound and not in contact with the wound itself, unless at very dilute concentrations. Chlorhexidine gluconate is a potent broad-spectrum germicide that is effective against nearly all nosocomial bacteria and yeasts,[42] and bacterial resistance to chlorhexidine is rare. Chlorhexidine has a low skin irritancy and sensitization potential, has a strong affinity for the skin, and demonstrates prolonged duration of antimicrobial effect. Unlike povidone-iodine, the antibacterial activity of chlorhexidine persists for hours after topical application. In contrast to iodine-containing compounds, chlorhexidine is not neutralized by contact with proteinous solutions, such as blood. Chlorhexidine should not be used in the facial region due to risk of contact injury to the cornea.

Wound irrigation and removal of debris are probably the most important steps in wound preparation for closure. Methods include mechanical, biological, technical, and surgical. Each method has different potential benefits and risks. The advantages of all irrigation methods are removal of debris, hydration of the wound, and assistance in visualization of structures for repair or control. The classic techniques include bulb syringes, syringes with attached cannulas, puncturing holes in the cap of liter bottles of normal saline, gravity flow irrigation from intravenous solution bags, and a number of commercial devices on the market that provide high-pressure or alternating pulsatile pressure irrigation. The optimal irrigation pressure is unknown. There

TABLE 50-6: Initial Selection of Antimicrobial Therapy in Patients with Risk Factors and High-Risk Austere Environment Wounds

	First-line agent		Alternative agent	
	Standard-risk factors	**High-risk factors**	**Standard-risk factors**	**High-risk factors**
Skin, soft tissue, no fracture	Cefazolin 1–2 g	Cefazolin 1–2 g	Clindamycin 900 mg	Clindamycin 900 mg
Skin, soft tissue				
Open joint	Cefazolin 1–2 g	Cefazolin 1 g	Clindamycin 900 mg	Clindamycin 900 mg
Open fracture, Gustilo I/II	Cefazolin 1–2 g load Ceftriaxone 1 g	Cefazolin 1–2 g Cefazolin 1 g	Clindamycin 900 mg Clindamycin 900 mg	Clindamycin 900 mg Clindamycin 900 mg
Open fracture, Gustilo III	or Ampicillin-sulbactam 3 g or Cefazolin 1-2 g and an aminoglycoside	or Cefazolin 1–2 g and an aminoglycoside, plus penicillin G 4 U	or Clindamycin 900 mg and an aminoglycoside or aztreonam or Quinolone	or Clindamycin, an aminoglycoside, or aztreonam
Soft tissue with concern for soil or waterborne organisms	NA	Bacterial coverage: Piperacillin-tazobactam 4.5 g Antifungal: Posaconazole	NA	Bacterial coverage: Meropenem 1 g or Trimethoprim-sulfamethoxazole Antifungal: Amphotericin
Thoracic cavity	Cefazolin 1 g	Consider cefazolin 1 g depending on intrathoracic component	Clindamycin 900 mg	Consider clindamycin 900 mg depending on associated intrathoracic component
Abdomen				
Suspected viscus injury	Cefoxitin 1–2 g or Piperacillin-tazobactam 4.5 g	Cefoxitin 1–2 g or Piperacillin-tazobactam 4.5 g	Ciprofloxacin 400 mg and metronidazole 500 mg or Levofloxacin 750 mg and metronidazole 500 mg	Ciprofloxacin 400 mg and metronidazole 500 mg or Levofloxacin 750 mg and metronidazole 500 mg
Penetrating viscus injury with associated spinal cord injury	Below choices for at least 24–48 hours, pending additional need for treatment of identified abdominal injuries	NA	Below choices for at least 24–48 hours, pending additional need for treatment of identified abdominal injuries	NA
Esophagus	Cefoxitin 2 g or Cefazolin 2 g and metronidazole 500 mg		Clindamycin 900 mg Levofloxacin 500 mg and metronidazole 500 mg or Clindamycin 900 mg and aminoglycoside	
Stomach	Cefoxitin 2 g		Levofloxacin 500 mg and metronidazole 500 mg	
Small bowel	Cefoxitin 2 g		Levofloxacin 500 mg and metronidazole 500 mg	
Colon	Cefoxitin 2 g or Cefazolin 2 g and metronidazole 500 mg		or Metronidazole 500 mg and aminoglycoside	

(*continued*)

TABLE 50-6: Initial Selection of Antimicrobial Therapy in Patients with Risk Factors and High-Risk Austere Environment Wounds (*Continued*)

	First-line agent		Alternative agent	
	Standard-risk factors	**High-risk factors**	**Standard-risk factors**	**High-risk factors**
Penetrating pelvic fracture Associated anus/rectum injury Associated genitourinary injury	Cefazolin 2 g Extended-spectrum penicillin or Cefazolin 2 g and metronidazole 500 mg Extended-spectrum penicillin	NA	Clindamycin 900 mg Ciprofloxacin 400 mg and metronidazole 500 mg Cefazolin 2 g and metronidazole 500 mg	NA
Maxillofacial				
Open fractures and fractures of the tooth-bearing maxilla/mandible	Cefazolin 2 g or Ampicillin-sulbactam 3 g	Cefazolin 2 g or ampicillin-sulbactam 3 g	Clindamycin 900 mg No antibiotics	Clindamycin 900 mg NA
Closed fractures (uncomplicated, non–tooth-bearing regions) and maxillary sinus fractures	No antibiotics	NA		
Central nervous system				
Penetrating brain injury Penetrating spinal cord injury	Ceftriaxone 2 g Cefazolin 2 g or Ceftriaxone 2 g	Cefazolin 2 g, aminoglycoside, and penicillin	NA	Vancomycin and ciprofloxacin 400 mg
Eye				
Abrasion Penetrating	Topical erythromycin or bacitracin ophthalmic ointment for symptoms Consider levofloxacin 750 mg	Fluoroquinolone drops QID × 3–5 days Consider levofloxacin 750 mg	Topical erythromycin or bacitracin ophthalmic ointment for symptoms	Fluoroquinolone drops QID × 3–5 days
Burn	No antibiotics			

NA, not applicable; QID, four times a day.

TABLE 50-7: Guide to Tetanus Prophylaxis in Routine Wound Management

No. of doses of adsorbed tetanus toxoid–containing vaccines	**Clean and minor wound**		**All other wounds[a]**	
	DTaP, Tdap, or Td[b]	**TIG**	**DTaP, Tdap, or Td[b]**	**TIG[c]**
Unknown or <3	Yes	No	Yes	Yes
≥3	No[d]	No	No[e]	No

[a]Such as, but not limited to, wounds contaminated with dirt, feces, soil, and saliva; puncture wounds; avulsions; and wounds resulting from missiles, crushing, burns, and frostbite.
[b]DTaP is recommended for children age <7 years. Tdap is preferred to Td for persons age ≥11 years who have not previously received Tdap. Persons age ≥7 years who are not fully immunized against pertussis, tetanus, or diphtheria should receive one dose of Tdap for wound management and as part of the catch-up series.
[c]Persons with HIV infection or severe immunodeficiency who have contaminated wounds should also receive TIG, regardless of their history of tetanus immunization.
[d]Yes, if ≥10 years since the last tetanus toxoid–containing vaccine dose.
[e]Yes, if ≥5 years since the last tetanus toxoid–containing vaccine dose.

DTaP, diphtheria and tetanus toxoids and acellular pertussis vaccine; Td, tetanus and diphtheria toxoids; Tdap, tetanus toxoid, reduced diphtheria toxoid, and acellular pertussis; TIG, tetanus immune globulin.

Source: Adapted from Liang JL, Tiwari T, Moro P, et al. Prevention of pertussis, tetanus, and diphtheria with vaccines in the United States: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Recomm Rep.* 2018;67(2):1.

remains ongoing debate given concern that if the pressure is too high, tissue destruction will occur and that low pressure fails to remove the debris and bacterial load, leading to a higher infection rate. Management should be based on the location, characteristics, injury environment, available equipment, and location of treatment.[43] A 2015 study in the orthopedic literature has shed a little more light on irrigation, showing that very-low-pressure irrigation (1–2 pounds per square inch [psi]) had similar complication rates, including infection, and was acceptable compared with low-pressure irrigation (5–10 psi) and high-pressure irrigation (>20 psi).[44] Thus, no method was superior to the other. High-pressure and alternating pulse irrigation systems have been noted to be associated with soft tissue damage and deeper bacterial propagation into the soft tissues. A study comparing high-pressure pulse lavage (HPPL) of greater than 20 psi, bulb irrigation, and suction irrigation noted that HPPL caused significantly more soft tissue damage and that bulb irrigation and suction irrigation were capable of removing more inorganic contamination than HPPL.[45] This supports the concept that sharp debridement and low-pressure irrigation, as opposed to high-pressure irrigation systems, may be the treatment of choice for most wounds. Judgment of the wound, including whether it is heavily contaminated, and concerns regarding soft tissue damage should dictate the use of higher pressure systems.

The solution for irrigation should be nontoxic and allow adequate assessment of the wound during the irrigation process. As previously noted, antiseptic solutions, such as povidone-iodine, chlorhexidine, hydrogen peroxide, and sodium hypochlorite (Dakin's solution) may be toxic to the tissues and impede wound healing. An additional agent used for open fracture management, castile soap, had a higher reoperation rate when compared to saline irrigation.[44] Normal saline has been the primary solution favored for wound irrigation given that it is isotonic, available, and does not interfere with normal wound healing processes. A recent review has reported no evidence that using potable (drinking/tap) water to cleanse an acute wound increases infection rates and some evidence that it reduces infection rates.[46] Thus, this may be an option if more sterile solutions are not available.

Wound Closure

The "golden period" for primary wound closure, defined as less than 6 hours from the time of injury, has been called into question. The initial recommendation of 6 to 8 hours was based on laboratory and clinical studies on the doubling time of bacteria and from clinical outcomes demonstrating decreased risk for infection after debridement during that period. Exceptions to this rule were the scalp and face, given the lower incidence of infection (Table 50-5). Quinn et al[47] reported that diabetes, wound contamination, length greater than 5 cm, and location on the lower extremity are the most important risk factors for wound infection. The time from injury to wound closure is not as important as previously thought. Improvements in irrigation, debridement, and decontamination over the past 30 years may have led to this change in outcome.[47] Wound closure may be performed immediately with sutures, tapes, tissue adhesives, or staples to allow healing by primary intention. Staples are most commonly used in areas less prone to cosmetic considerations such as the scalp, trunk, and extremities. Sutures are used in most other locations, with the type dependent on the location, wound tension, complexity, and possibly compliance of the patient to return for removal. In the latter population, fast-absorbing gut or other absorbable sutures may be appropriate for an interrupted or subcuticular closure. Complex wounds may be closed in multiple layers, using an absorbable suture to approximate fascia or the dead space of the soft tissues. The technique for skin closure should allow precise alignment of the skin edges, with the minimum amount of foreign material in the wound. For a cosmetic closure of facial wounds, 6-0 monofilament suture with 1- to 2-mm bites in either a running or interrupted fashion is used. These sutures are usually removed in 3 to 5 days to minimize suture marking (Table 50-8).

In the area of the mouth, the repair should approximate the vermillion border of the lip. Tissue adhesives are less painful and quicker to apply but may be associated with more dehiscence than sutures. The most frequent areas of use of these adhesives are for low-tension wounds, especially on the face. Adhesive tapes for closure must have a dry surface for best result, work best in areas of low tension, and can be associated with premature removal, traction blisters, and wound dehiscence. Patient compliance should be considered with tissue adhesive and adhesive tape closures.

Wounds with significant contamination, ischemia, or tissue loss may be left open to heal on their own by secondary intention. Wounds in this category may also need repetitive returns to the operating room for serial debridement of nonviable tissue as determined by the color, consistency, and circulation, and when evaluating muscle viability, contraction is also an important component. Wounds are treated with various methods of debridement, including excisional, abrasive, or nonexcisional, and with the use of irrigation or hydrosurgery. Surgical debridement involves the excision of necrotic tissue with the use of scalpels, scissors, curettes, rongeurs, saws, or burrs. Bleeding is common in this approach, which is usually performed in the operating room. Hydrosurgery,

TABLE 50-8: Timing for Suture removal

Location	Time[a]
Face	3–5 days
Trunk	7 days
Scalp	7–10 days
Extremity	7–10 days
Joints	14–21 days

[a]Given usual healing conditions.

unlike high-pressure lavage, consists of a waterjet that passes parallel to the wound that debrides tissue, which is captured by a suction evacuator system. The system simultaneously irrigates and debrides tissue.[48,49] There is debate regarding whether hydrosurgery is cost effective for debridement and wound bed preparation.[50,51]

Wound dressings for open wounds treated by secondary closure are additionally variable. One of the traditional dressings for open wounds has been a moist gauze application and a dry gauze as a cover to wick away any exudate and remove debris—the traditional wet to dry dressing. The goal of newer dressings is to produce a moist but not macerated wound that is free of infection, toxic chemicals, and foreign material while maintaining an optimum temperature and pH. Wound dressings can be broken down into several major groups, which include gauze, semipermeable film dressings, foam dressing, alginates, and hydrocolloids (Table 50-9).

A number of dressing types are appropriate in a granulating wound, and the amount of wound exudate often guides wound care recommendations. Dabiri et al[52] published a practical guideline for dressing use in *Advances in Wound Care* in 2016. In granulating wounds with mild to moderate exudate, a hydrocolloid dressing will maintain granulation tissue and aid in epithelialization by absorbing liquid and exudate by formulating a soft gel. Foams are recommended for wounds with moderate exudate due to their hydrophilic component. Calcium alginates, hydrofibers, and polymeric membrane dressings are also useful in exudate wounds. Fibrinous wound beds require dressings that aid in slough removal, such as wet to dry dressings and proteolytic, enzymatic, or autolytic products. Finally, iodine-based and silver-releasing agents have been formulated into nontoxic antimicrobial dressings to assist in wound healing and enhance bactericidal properties.[53] The current recommendations for silver dressings is a 2-week period of use followed by reevaluation; if there is improvement, the dressings are continued, and if not, an alternative technique should be considered.[54]

Negative-pressure wound therapy (NPWT), also known as vacuum-assisted closure or microdeformational wound therapy, uses a dressing that applies continuous or intermittent

TABLE 50-9: Wound Dressing Materials

Classification	Material	Functions	Use	Examples	Disadvantages	Advantages
Gauze	Cotton gauze	Absorbs fluid, removes exudate with changes	Open or infected wounds, fibrinous wounds	Kerlex fluffs 4×4	Frequent changes, foreign body in wound, desiccation	Readily available
Hydrocolloid	Hydrophilic colloidal particles with adhesive matrix	Maintains moist wound environment with optimum temperature and pH, adherence without adhesion, promotes granulation and epithelialization, seals and protects wound, wound debridement by autolysis	Full- or partial-thickness wounds, decubitus ulcers, primary dressing for closed wounds	Duoderm NuDerm Comfell Hydrocol Cutinova Replicare Tegasorb Granuflex Intrasite Biofilm	Some seepage with heavy exudate, difficult-to-seal wounds with complex shape, malodorous yellow gel formation	Optimal wound environment, improved healing of some wounds, infrequently needs changing, decreased pain, no foreign body residual
Alginates	Calcium alginate fibers	Absorbs fluid and exudate, maintains moist wound environment	Open or infected wounds	Algiderm Algisite Algisorb Kaltostat Curasorb Sorbsan Melgisorb SeaSorb Kalginate	Painful removal when minimal exudate, require a secondary dressing	May be used in infected or contaminated wounds, infrequent dressing changes

(*continued*)

TABLE 50-9: Wound Dressing Materials (*Continued*)

Classification	Material	Functions	Use	Examples	Disadvantages	Advantages
Films	Semipermeable membranes (polyurethane or copolyester)	Occlusive dressing, gas permeable	IV site dressing, partial-thickness, minimal exuding wounds	Opsite Tegaderm PolySkin Bioclusive Blisterfilm Ominderm Proclude Mefilm Carrafilm Transeal	Not absorptive, surrounding skin maceration, adhesive may damage uninvolved skin	Shield from bacterial invasion
Hydrogels	Cross-linked hydrophilic polymers bathed in water-rich environment, promote autolytic debridement	Soften and liquefy hard eschar and slough	Nonsurgical treatment of eschars	Vigilon Nu-Gel Tegagel FlexiGel Clearsite Curafil Curasol Elasto-gel Hypergel Normgel 2nd Skin Transigel	Frequent change to prevent skin maceration	Minimal pain or trauma during dressing changes
Foams	Thicker dressing of a bilaminate structure with a hydrophilic surface		Wounds over bony prominence, moderate fluid or exudate	Allevyn Biopatch Curafoam Hydrasorb Mepilex	May be too drying to a wound if minimal or mild exudate, malodorous	
Hydrofibers	Hydrophilic carboxymethylcellulose fibers	Absorbs fluid and exudate, maintains moist wound environment	Wounds with moderate to excessive exudate	Aquacel Versiva	Painful removal when minimal exudate, require a secondary dressing	Less lateral wicking and maceration than alginate dressings
Polymeric membrane dressings (PMD)	Hydrophilic polyurethane membrane matrix with a continuous semipermeable film backing	Contain ingredients that continuously cleanse wounds and expedite healing	Donor graft sites and superficial abrasions	Polymem		Nonadherent, may decrease wound pain
Silver dressings	Silver ions incorporated into a dressing	Silver ions bind and disrupt bacterial cell walls, intracellular and nuclear membranes, denature bacterial DNA and RNA	Infected wounds for 2 weeks, if improvement continues	Silvasorb Aquacel Ag Mepilex Ag Silvercel Algicate Ag SilverAlginate Dynaginate AG Suprasorb Ag Acticoat Medifoam Silver Allevyn Ag Polymem Silver	Must be moist to release the active agent	Multiple products with additional wound treatment advantages

negative pressure to a wound. NPWT, similar to the previously discussed dressings, provides wound coverage, prevents desiccation, facilitates wound drainage, and contributes to decreasing the wound bacterial burden. It is not an alternative to debridement, and in fact, if necrotic tissue is present, this mandates additional excisional debridement to assist in healing. NPWT has been shown to decrease hospital length of stay and cost of wound care, improve time to wound closure, and decrease overall complication rates.[55,56] The mechanisms by which NPWT facilitates wound contraction and closure are complex. The negative pressure draws out the excess fluid from the wound and may be a means of clearing excessive quantities of inflammatory mediators, which, it is believed, may preclude the natural progression of the wound healing pathways. Further, edema fluid exerts an adverse compressive effect on the microcirculation; thus, drawing the edema fluid from the wound may improve perfusion and oxygenation to the wound. NPWT has also been shown to decrease bacterial burden within the wound[57] and to act via mechanosensing and mechanotransduction effects, applying traction forces that promote the central migration of factors essential for wound coverage, including wound fibroblasts.[58,59] Keratinocytes, endothelial cells, and fibroblasts have all been shown to respond to the mechanical forces generated by NPWT upon the wound. NPWT also induces production of proangiogenic factors including vascular endothelial growth factor (VEGF) and fibroblast growth factor-2 (FGF-2), and this modifies endothelial morphology and proliferation with an associated improvement in angiogenesis, vessel formation, and ultimately wound perfusion.[60,61] Although the amount of collagen in the wound does not appear to be affected by NPWT, it is clear that NPWT improves collagen organization within the wound. It appears that the cytokine milieu of the wound is changed by the application of NPWT,[62] as evidenced by reduction in tumor necrosis factor levels,[63] whereas IL-8, a potent chemokine and regulator of neutrophil and macrophage migration, was noted to be elevated in the wound without a concomitant elevation of systemic IL-8 levels.[61]

In essence, the NPWT devices commercially available involve placement of a foam or interface material of varying porosity and chemical composition on a wound and then connection to a suction device. Polyurethane ether (PE) black foam is hydrophobic, whereas polyvinyl alcohol (PA) white foam is hydrophilic, and both are used with commercially available NPWT devices. PE is used for fluid drainage and to promote granulation, whereas the PA sponge is used over delicate tissues such as vasculature, tendons, or bowel. Modifications of NPWT devices include the ability to instill a variety of solutions, including antibiotics, through the device into the wound. NPWT has been used in conjunction with other wound healing modalities including being applied over a split-thickness skin graft as well as in combination with processed allogenic materials. NPWT induces macrodeformation of the wound through the centripetal force exerted upon the wound edges and microdeformation forces that are exerted upon the individual cells at the edges and within the body of the wound. This combined effect has the overall effect of transitioning a large complex wound into a smaller wound that may be manageable with lesser surgical reconstructive techniques.[64,65]

In wounds healing by secondary intention, excessive granulation tissue may form preventing epithelialization. The excess granulation tissue may need to be removed by surgical, cauterization (heat or silver nitrate), or laser techniques versus hypertonic NaCl dressings, permeable nonocclusive dressings, or topical steroids.[66] Wounds that heal by secondary intention either contract and epithelize, or if the wound is too large to completely epithelize, once the surface is appropriate a skin graft may be used.

The final choice in wound management may be to leave the wound open following debridement to viable tissue with a planned delayed primary closure. The wound is inspected periodically over the next several days until it is determined that there is no further devitalized tissue or inflammation, and then closed. Most wounds are closed within 48 to 72 hours, but some are closed after up to 5 to 7 days. The goal is to decrease infection and simplify wound care following closure; occasionally, as a result of dead space issues, drains will be placed in the depths of the wound. These wounds will need monitoring for secondary infection, and if this occurs, they need to be opened and allowed to heal by secondary intention. The benefit of this method is that the entire wound may not need to be opened, decreasing final time to healing and closure.

BITES

Human Bites

Mammalian bites are a significant health issue worldwide. In the United States alone, there are approximately 2,000,000 to 5,000,000 bites per year, accounting for 1% of all emergency department visits.[67,68] Human bites are the third most common cause of bite wounds seen in hospital emergency departments, behind dog and cat bites, and account for approximately 2% to 20% of all bite wounds. Human bite wounds are classically underreported and are notorious for causing infection at the site of injury; they also pose a risk for transmission of infectious diseases.

EPIDEMIOLOGY

Human bite wounds can result from incidental (nail biting) or purposeful injury. Studies have found that human bites are more common in males, with the peak incidence occurring in individuals between the ages of 10 and 34 years old.[69] Human bites can be classified according to mechanism, including occlusive bites and clenched-fist injuries (fight bite). Clenched-fist injuries occur predominantly in males, whereas occlusive injuries occur with equal frequency in males and females.[69,70] Human bites are often multiple, located on the upper extremity, and associated with both the greatest loss of function and risk of infection.

PATHOPHYSIOLOGY

Human bites can be classified by mechanism of injury into occlusion bites and clenched-fist injuries. Occlusion bites are similar to animal bites and occur when the teeth close over the skin with sufficient force to breach the integrity of the skin. Human occlusive bites are usually seen as a semicircular or oval area of ecchymosis or erythema that usually compresses tissue rather than avulses it, as is more common in animal bites. This classic pattern is caused by positive pressure from the closing teeth that first disrupts blood vessels and by the negative pressure from suction and tongue thrusting.[69,71] Clenched-fist injuries are not true bites but are clinically similar to bite wounds because the skin is disrupted by teeth. Clenched-fist injuries occur when a closed fist makes impact with another individual's teeth, classically leaving an injury over the dorsal aspect of the third, fourth, or fifth metacarpophalangeal joints. Although clenched-fist injuries are quite small, they are highly prone to infection given the proximity of skin over the knuckles to the joint capsule and the relative avascular nature of these structures.

CLINICAL EVALUATION AND MANAGEMENT

Most human bite wounds can be treated in the emergency department, with approximately 1% to 2% requiring hospitalization. The key components of initial evaluation and management of mammalian bites include a history, thorough physical examination, irrigation, debridement, and closure if indicated. Early treatment, appropriate antibiotic prophylaxis, and surgical evaluation if indicated are the key to achieving optimal outcomes for this vulnerable class of wounds. The first step in evaluation is an accurate history, and the essentials to elicit include both the timing and circumstances surrounding the bite event, location of bite wounds, any treatment rendered, significant past medical history that could alter the host response, tetanus vaccination status, and HIV or hepatitis carrier status of the person inflicting the bite if known (Table 50-10).[69] Occlusive bites in women should give concern for the possibility of domestic or sexual abuse, and although occlusive bites in children are often related to other children, the possibility of abuse needs to be considered.

TABLE 50-10: Essentials of Patient History After Human, Dog, or Cat Bites

Time and location of bite event
Circumstances surrounding bite
Anatomic location
Prehospital treatment (irrigation)
Type of dog or cat and health status (health, rabies vaccination history, behavior, known location)
HIV and hepatitis status of person inflicting human bite if known
Patient factors
• Tetanus status
• Rabies vaccination history
• Immunocompromised state
• Diabetes
• Peripheral vascular disease

TABLE 50-11: Clinical Management After Human, Dog, or Cat Bites

Inspect all bite wounds
• Depth
• Range of motion (very important for hand bites to identify occult injury)
• Associated devitalized tissue
Irrigate all wounds thoroughly with saline
Debride necrotic tissue
Obtain cultures: blood and wound if clinical signs of infection (abscess, cellulitis, sepsis)
Obtain radiographic studies if indicated (identify associated fractures)
Initiate prophylactic antibiotics if indicated
Assess tetanus status and administer if indicated
Assess need for rabies postexposure prophylaxis
Assess need for postexposure prophylaxis and counseling for potential HIV/hepatitis exposure
Hospitalization
• Fever
• Severe cellulitis
• Edema
• Sepsis
• Significant crush injury

Physical examination should include a measurement of vital signs and careful inspection of every bite wound to identify the depth of penetration, the presence of devitalized tissue, neurovascular involvement, and any signs of infection including surrounding erythema, edema, and purulent drainage (Table 50-11). Local anesthesia is usually necessary for an adequate physical examination to minimize patient discomfort. Occlusive or clenched-fist injuries on the hand or fingers are particularly prone to infection as penetration into deeper tissue including the underlying extensor mechanism, joint capsule, or deeper connective tissue is noted to occur in as many as 75% of clenched-fist injuries to the hand.[72] In hand bites of any etiology, it is critical to perform a complete assessment of extension and flexion of the hand and fingers to adequately examine the underlying structures in their natural state, minimizing missed injuries. Although this assessment may be painful for the patient, it is essential and includes recreating the clenched fist to examine the wound and underlying tendons and tissue, which can retract and/or be masked by viewing only in the relaxed position. A thorough exam, although important for every bite, is crucial in this vulnerable anatomic subset of wounds that are prone to infection and devastating complications if diagnosis or management is delayed.

Physical examination is crucial in determining appropriate ancillary studies in patients who present with human bite wounds. Routine laboratory studies, including a white blood cell count, C-reactive protein, and erythrocyte sedimentation

rate, may be elevated but do little to either rule out or rule in infection and are not indicated. Aerobic and anaerobic blood cultures are only indicated in patients who present with physical exam findings consistent with an infected bite wound and signs of systemic infection prior to initiation of antibiotic therapy (Table 50-11). Empiric wound cultures of the noninfected bite wound are not clinically indicated, and the results from those cultures do not correlate with the likelihood of infection or the pathogen that is present if the wound subsequently becomes infected.[73,74] Plain x-rays are indicated in wounds near joints to evaluate for fractures and evidence of foreign bodies and in wounds with signs of marked infection to detect fractures, subcutaneous gas, and changes associated with osteomyelitis (Table 50-11).

Key to achieving optical outcomes is expedient treatment. Irrigation and debridement of necrotic tissue are the most important means of infection prevention and are most important in treating bite wounds to the hand (Table 50-11). Irrigation should proceed with isotonic sodium chloride irrigation under pressure; approximately 100 to 200 mL of irrigation solution per square inch appears to be an adequate rule of thumb.[69,75] For most bite wounds, a 19-gauge blunt needle and 35-mL syringe provide adequate pressure and volume. If surgical sharp debridement is needed, it should include devitalized, infected, and necrotic tissue. Delayed presentation and treatment leads to worse outcomes, particularly in closed-fist injuries where delay beyond 8 days is associated with an amputation rate of 18%.[76] Although an important part of infection prevention, care should be taken when debridement is necessary in order to avoid unnecessary enlargement of the wound preventing potential closure, particularly in areas of cosmetic concern.

A majority of human bite wounds are left open to heal by secondary intention due to the high risk of infection. This is most important for bites to the hand. Primary closure can be considered on clinically uninfected facial wounds less than 24 hours old where cosmesis is paramount and infection is uncommon secondary to the excellent blood supply to the face. This has been anecdotally reported, but a paucity of evidence is present in the literature. In one study of a limited number of patients with primary closure of facial human bites after irrigation and debridement, despite the duration of injury being delayed up to 4 days, wound healing was complete in 90% of patients at the time of suture removal.[77]

Appropriate prophylaxis is of paramount importance in minimizing complications in patients who suffer human bite wounds. All patients with human bite wounds that penetrate the dermis, regardless of appearance of the wound, should undergo antibiotic prophylaxis because of the higher risk of infection. One randomized controlled trial that studied the utility of antibiotic prophylaxis in human bite injuries to the hand found that 47% of patients in the placebo group developed infection versus none in the antibiotic prophylaxis group.[78] It is of particular importance in occlusive and clenched-fist injuries to the hand, wounds near a bone or joint, wounds with associated crush injury, wounds requiring surgical repair, and wounds in immunocompromised patients. Patients receiving antibiotic prophylaxis should receive the first dose as soon as possible after the injury and continue the antibiotic course for 3 to 5 days. Antibiotic prophylaxis should include activity against the common human bite wound pathogens, including *Streptococcus* species, *Staphylococcus aureus*, *Eikenella*, *Fusobacterium*, *Peptostreptococcus*, *Prevotella*, and *Porphyromonas* species (Table 50-12). In addition to antibiotic prophylaxis, tetanus prophylaxis should be administered if indicated (Table 50-7). Transmission of HIV and hepatitis C through saliva is rare, and routine HIV testing after human bites is not necessary; however, counseling regarding postexposure prophylaxis may be appropriate in certain settings (Table 50-11).[79-81] Rates of transmission of hepatitis B virus are higher, and administration of an accelerated course of hepatitis B virus vaccine is recommended.

TABLE 50-12: Antibiotic Guidelines After Dog, Cat, or Human Bites

Infection not established

- Amoxicillin + clavulanate (child, 22.5 + 3.2 mg/kg up to 875 + 125 mg) orally, 12 doses hourly for 5 d
- If commencement of above is likely to be delayed, procaine penicillin (child, 50 mg/kg up to 1.5 g) intramuscularly, as 1 dose, followed by above

Infection established

- Metronidazole (child, 10 mg/kg up to 400 mg) orally, 12 doses hourly for 14 d + EITHER
- Cefotaxime (child, 50 mg/kg up to 1 g) intravenously daily for 14 d OR
- Ceftriaxone (child, 50 mg/kg up to 1 g) intravenously daily for 14 d OR
- Piperacillin + tazobactam (child, 100 + 12.5 mg/kg up to 4 + 0.5 g) intravenously, 8 doses hourly for 14 d OR
- Ticarcillin + clavulanate (child, 50 + 1.7 mg/kg up to 3 + 0.1 g) intravenously, 6 doses hourly for 14 d

For patients with immediate penicillin hypersensitivity

- Metronidazole (child, 10 mg/kg up to 400 mg) orally, 12 doses hourly for 14 d + EITHER
- Doxycycline (child > 8 y, 5 mg/kg up to 200 mg) orally for the first dose, then 2.5 mg/kg up to 100 mg orally, 12 doses hourly OR
- Trimethoprim + sulfamethoxazole (child, 4 + 20 mg/kg up to 160 + 800 mg) orally, 12 doses hourly OR
- Ciprofloxacin (child, 10 mg/kg up to +500 mg) orally, 12 doses hourly

Source: Reproduced with permission from Aziz H, Rhee P, Pandit V, et al. The current concepts in management of animal (dog, cat, snake, scorpion) and human bite wounds. *J Trauma Acute Care Surg.* 2015;78(3):641-648. © 2015 Wolters Kluwer Health, Inc. All rights reserved.

COMPLICATIONS

The most feared complication following human bite wounds is the development of infection. Human bites are a challenging problem given the wide variety of bacteria (>50) and the number of organisms, with almost 10^8 microbes/mL found in saliva.[68,82] Higher rates of infection are seen in human bites with delayed presentation, bites on the hand, or bites on relatively avascular structures (ear cartilage). When infections do

occur, they tend to be polymicrobial, and common organisms include *Streptococcus viridans*, *Staphylococcus* species, *Eikenella corrodens*, *Bacteroides* species, and microaerophilic streptococci. Antibiotics should cover the most common pathogens and provide broad anaerobic coverage. In addition, they should include a β-lactamase inhibitor because 50% to 80% of *Prevotella* species and staphylococci isolated from human bite wounds were β-lactamase producers.[68,83] Antibiotic prophylaxis, as mentioned previously, should be continued for 3 to 5 days and extended if infection is present during follow-up or on initial presentation if delayed. This course is extended for 10 to 14 days when cellulitis is present, for 3 weeks for tenosynovitis, for 4 weeks for septic arthritis, and for 6 weeks for osteomyelitis (Table 50-12).

Dog Bites

Dog bites are the most common animal bites seen in the emergency department in the United States each year (60%–90%), and the medical costs for the treatment of dog bites alone is estimated to be $1 billion per year in the United States. In addition, dog bites cause between 20 and 35 deaths each year in the United States, primarily in infants and young children.[69]

EPIDEMIOLOGY

Dog bites are frequently caused by animals known to the victim. Dog bite wounds are more common in males and in children, with the highest incidence in males between the ages of 5 and 9 years. The age of the dog bite victim is indicative of location of injury. The most common sites of dog bites are to the head and neck in children through the age of 9 years. In children older than 10 years and adults the most common location of dog bites is to the arms, particularly the dominant hand, and legs.[84-86]

PATHOPHYSIOLOGY

Dogs can cause a wide range of injuries, from relatively minor wounds, including scratches and abrasions, to complicated wounds, including deep puncture wounds and lacerations, tissue avulsions, and crush injuries. The severity of injury is most commonly related to the size of the dog, with the jaws of larger dogs capable of generating 200 to 450 psi. The substantial pressures exerted on tissue during the bite result primarily in significant crush injuries and avulsions.

CLINICAL EVALUATION AND MANAGEMENT

As discussed thoroughly in the previous section, the key components of initial evaluation and management of mammalian bites include a history, thorough physical examination, irrigation, debridement, and closure if indicated (Tables 50-10 and 50-11). Dog bites have markedly lower rates of infection than bites from humans or cats. Despite the lower risk of infection, dog bites require copious irrigation. Facial dog bite wounds are usually closed, whereas wounds in other locations are managed with delayed primary closure or healing by secondary intention.

Although the infection risk is lower in dog bites than in human or cat bites, prophylaxis should be considered in patients who present more than 8 hours after the bite or patients who have moderate or severe wounds, diabetes mellitus, an immunocompromised state, hand bites, or deep puncture wounds. Common infecting organisms include *Pasteurella multocida*, *S viridans*, *Bacteroides* species, *Fusobacterium*, and *Capnocytophaga*. Antibiotics should cover the most common organisms; ampicillin plus a β-lactamase inhibitor provide reasonable empiric coverage. Treatment for empiric coverage should be 3 to 5 days and should be extended if infection is present during initial presentation or in follow-up (Table 50-12). As with most bite wounds, treatment can occur mainly as an outpatient; however, if significant infection or debridement is warranted, the patient may require hospital admission and utilization of intravenous antibiotics. Tetanus and rabies prophylaxis should be administered if indicated in all patients presenting with a dog bite (Tables 50-7, 50-13, and 50-14).

Cat Bites

Cat bites are the second most common type of animal bite in the United States, accounting for approximately 5% to 20% of animal bite wounds.

EPIDEMIOLOGY

Cat bites occur with an estimated annual incidence of approximately 400,000 and usually present in a delayed fashion after complications develop.[87] Cat bites occur most commonly in females and the elderly over the age of 75.[88] Over two-thirds of cat bites occur on the upper extremities, with decreasing incidence on the lower extremities, head and neck, and trunk. Similar to human bites, cat bites are heavily contaminated with bacteria.

PATHOPHYSIOLOGY

Cat bites do not carry as much force as seen in dog bites and thus are not associated with the same degree of crush and soft tissue injury. Due to their sharp and narrow teeth, cat bites present with small puncture wounds that can be deceptively deep.

CLINICAL EVALUATION AND MANAGEMENT

The evaluation and management of cat bites are similar to other bite wounds and include a history, thorough physical examination, irrigation, and debridement (Tables 50-10 and 50-11). Closure is not indicated in cat bites. As mentioned previously, cat bites are heavily contaminated, and the wounds they inflict, although small, can be deep. Thorough exploration of the depth of penetration is extremely important, because deep abscess and osteomyelitis are more common in cat bites.

As mentioned previously, cat bites are prone to infection. Common organisms include *Pasteurella multocida* and *Staphylococcus* species. Antibiotic prophylaxis that includes

TABLE 50-13: Treatment Recommendations and Estimates of the Risk of Rabies According to Type of Exposure and Geographic Area

		Treatment recommendations[b,c]		Cases of rabies/10,000 untreated exposures[c]	
Geographic area	**Animals[a]**	**Bite**	**No bite**	**Bite**	**No bite**
Group 1: Rabies endemic or suspected in species involved	Bats anywhere in the United States; raccoons, skunks, foxes, mongooses in Puerto Rico, dogs in most developing countries and in the United States along the Mexican border (3%–80% rabid)	Treat	Treat	150–5000	0.3–160
Group 2: Rabies not endemic in species involved in the exposure, but endemic in other terrestrial animals in the area	Most wild carnivores (wolves, bobcats, bears) and groundhogs (2%–20% rabid)	Treat	Treat or consult	10–1200	0.2–40
	Dogs and cats (0.1%–2% rabid)	Observe or consult	Observe or consult	0.5–120	0.01–4
	Rodents and lagomorphs except groundhogs (0.01% rabid)	Consult or do not treat	Do not treat	0.05–0.6	0.001–0.02
Group 3: Rabies not endemic in species involved in the exposure or in other terrestrial animals in the area	Dogs, cats, many wild terrestrial animals in Washington, Idaho, Utah, Nevada, and Colorado (0.1%–0.01% rabid)	Consult or do not treat	Consult or do not treat	0.05–6	0.001–0.2

[a]Percentages are the approximate proportions of indicated species found rabid when submitted and tested in state health department laboratories.
[b]"Consult" denotes consultation with a state or local health department. If the risk of rabies in the species involved in the exposure is low and the animal's brain is available, treatment is sometimes delayed up to 48 hours pending the results of laboratory testing. A healthy domestic dog or cat that bites a person should be confined and observed for 10 days. Any illness in the animals should be evaluated by a veterinarian and reported immediately to the local health department. If signs suggestive of rabies develop, treatment is begun immediately. If the animal is a stray or unwanted cat or dog, it should be killed immediately and the head removed and shipped under refrigeration for examination by a qualified laboratory.
[c]Rabies develops in 50% to 60% of untreated humans bitten by a rabid animal and in 0.1% to 2% of those exposed to rabid animals but not bitten (scratched or licked on an open wound or mucous membrane). The number of cases per 10,000 untreated patients was derived by multiplying the prevalence of rabies in a geographic area by the proportion of untreated humans in whom rabies would be expected to develop after exposure to a rabid animal.

Source: Adapted from Fishbein D, Robinson L. Rabies. *N Engl J Med.* 1993;329:1636. Adapted from Centers for Disease Control and Prevention. Rabies Prevention—United States, 1991. Recommendations of the Immunization Practices Advisory Committee (ACIP). *MMWR.* 1991;40(RR03):1-19. Table 2.

ampicillin and a β-lactamase for 3 to 5 days is indicated, with extended duration for established infection. Tetanus and rabies prophylaxis should be given if indicated (Tables 50-11 and 50-12). It is important to remember when evaluating and managing cat bites that approximately 6% of cat bites will require hospital admission.

Rabies

Rabies is a vaccine-preventable viral disease primarily caused by the bite of a rabid animal. A number of different strains of highly neurotropic viruses cause rabies infection. Most of the viruses belong to a single serotype in the genus *Lyssavirus*, which belongs to the Rhabdoviridae family, and induce a rapidly progressive encephalitis. There is no known effective treatment once infection is established, and rabies has the highest per-case fatality rate of any infectious disease.

EPIDEMIOLOGY

Rabies has a worldwide distribution and is found on every continent except Antarctica.[89] A majority of rabies cases are acquired through exposure to saliva from an animal bite. A minority of cases can occur via nonbite exposures, including aerosolized virus in bat caves, handling virus in laboratories, and transplantation of tissue or organs from a donor with unrecognized disease. The World Health Organization estimates that between 30,000 and 70,000 people die of rabies each year, with a majority of these occurring in Asia and Africa. Dogs are the main source of human rabies deaths, accounting for over 99% worldwide.[90] In the United States, vaccination programs have been responsible for a dramatic decline in rabies acquired from domestic cats and dogs. Since the late 1960s, rabies exposures in the United States have been most commonly linked to wild animals, including bats (most common), raccoons, skunks, and foxes (Table 50-13).[91-93]

TABLE 50-14: Schedule of Prophylaxis Recommended in the United States After Possible Exposure to Rabies

Vaccination status	Regimen[a]
Not previously vaccinated	
Local wound cleansing	Immediate cleansing with soap and water.
Rabies immune globulin	20 IU/kg of body weight (if anatomically feasible, up to half the dose should be infiltrated around the wound or wounds and the rest should be administered intramuscularly in the gluteal area. Never give more than the recommended dose. Do not use the syringe used for vaccine or inject into the same anatomic site.)
Vaccine	1.0 mL of HDCV or RVA intramuscularly in the deltoid area[b] on days 0, 3, 7, 14, and 28.
Previously vaccinated[c]	
Local wound cleansing	Immediate cleansing with soap and water.
Rabies immune globulin	Should not be given.
Vaccine	1.0 mL of HDCV or RVA intramuscularly in the deltoid area[b] on days 0 and 3.

[a]The regimens are applicable to all age groups, including children.
[b]The deltoid area is the preferred site of vaccination for adults and older children. For younger children, the outer aspect of the thigh may be used. Vaccine should never be administered in the gluteal area.
[c]"Previously vaccinated" indicates previous vaccination with HDCV or RVA, or any other type of rabies vaccine and a documented history of antibody response.

HDCV, human diploid cell vaccine; RVA, rabies vaccine adsorbed.

Source: From Fishbein D, Robinson L. Rabies. *N Engl J Med*. 1993;329:1636. Adapted from Centers for Disease Control and Prevention. Rabies Prevention—United States, 1991. Recommendations of the Immunization Practices Advisory Committee (ACIP). *MMWR*. 1991;40(RR03):1–19. Table 2.

In the United States, there are approximately two to three reported cases of human rabies per year.[94-96]

PATHOPHYSIOLOGY

The rabies virus has a predilection for neural tissue and spreads via peripheral nerves to the central nervous system. Rabies has a prolonged average incubation period of 1 to 3 months, with reports ranging from several days to 7 years. The relatively slow replication in the infected muscle or peripheral nerve allows for marked immune system evasion. Once in the central nervous system, the virus replicates rapidly and travels to the salivary glands, where productive viral replication and shedding occur. There are many host factors that influence the susceptibility to rabies infection. Host factors that increase susceptibility include virus variant, amount of viral inoculum, degree of innervation at the site of the bite, and host immunity and genetics.[97,98]

CLINICAL EVALUATION AND MANAGEMENT

In humans, established disease is almost always fatal. The dismal prognosis of rabies encephalitis emphasizes the importance of appropriate use of the vaccine and immunoglobulin preparations to prevent infection when indicated in humans presenting with animal bite wounds. The risk of acquiring infection depends on the probability of rabies infection in the animal and the amount of inoculum delivered into the wound. It is of utmost importance for treating physicians to be familiar with the local incidence of rabies in their region and to integrate this knowledge into the patient history of present illness. In general, low-risk bites include those from immunized animals and healthy cats and dogs in nonendemic areas. The risk of rabies inoculation is increased in domestic animals when the bite was unprovoked or if the animal was behaving erratically prior to the bite. Patients who present with bites from wild animals including bats, skunks, raccoons, and foxes should be considered high risk for rabies, and prophylaxis should be given. Tables 50-13 and 50-14 summarize the risk of transmission of rabies and treatment. Upon initial presentation, all bite wounds should be thoroughly cleansed with soap and water. This treatment alone has been shown to protect 90% of experimental animals from infection following inoculation of rabies virus into a wound.

The mainstay of treatment is a high index of suspicion and administration of postexposure rabies prophylaxis upon initial presentation, as the early symptoms are often mild. Early symptoms are subtle and vague, consisting of mild fatigue and behavioral changes. Overt symptoms, occurring approximately 7 days from the bite, include pain at the bite site, dysphagia, pharyngeal spasms, excessive muscle movements or flaccidity, and dysautonomia. Neurologic deterioration continues rapidly and includes progressive motor paralysis and death, usually within 3 days of the presence of overt symptoms. Rabies infection is uniformly fatal in humans; therefore, postexposure prophylaxis is critical. Although rabies prophylaxis is costly, there has been no recorded failure of the cell-based postexposure prophylaxis since 1970 if promptly administered.[99,100]

Spider Bites

Spider bites are very rare medical events. There are thousands of spider species, and only a few cause problems in humans. All spiders have a venomous component to their bite, which is essential for neutralizing and killing their prey, but only a few species have muscles powerful enough to penetrate human skin and have venom that affects mammalian tissues.

EPIDEMIOLOGY

There are many common disorders that mimic a spider bite, so the incidence is often exaggerated and overreported. The two most common species of spiders dangerous to humans in the continental United States include the brown recluse spider (*Loxosceles reclusa*) and the black widow (*Latrodectus mactans*). Brown recluse spiders are limited to the midwestern

and southern portions of the United States, and the black widow spider is found in warm climates worldwide.

CLINICAL EVALUATION AND MANAGEMENT

A presumptive diagnosis of a spider bite is often based on history and clinical presentation. Most patient reports are unreliable, and the spider was never visualized. The diagnosis of a spider bite can only be considered definitive if a spider was observed inflicting the bite; the spider was recovered, collected, and properly identified; and a skin lesion or systemic finding typically associated with the type of spider bite is present.[101] Physicians evaluating patients with suspected spider bites should be knowledgeable about spiders indigenous to their area and offer tetanus prophylaxis when indicated.

The brown recluse spider can be identified as a brown spider with three pairs of eyes and a violin-shaped carapace on its body, although this may be difficult to see.[102] Initial symptoms are often very mild and cause little pain. In up to 40% of cases, local tissue ischemia progresses and a very painful necrotic wound can develop. Aggressive surgical management is contraindicated, and allowing for definitive demarcation can minimize wound size and tissue loss. Rarely, wounds resulting after debridement will require skin grafting. Systemic symptoms are mild and respond well to supportive care.

The black widow spider can be identified as a large black shiny spider with a red hourglass on the underside of the abdomen. Only the female black widow spider has enough venom to envenomate a human. Bites by the black widow spider are characterized by a systemic toxic reaction. Symptoms of envenomation are rapid and usually occur within 1 hour of the bite and include pain, muscle rigidity, altered mental status, and seizures. The pain is often intense but resolves within 72 hours. The treatment is mainly supportive; however, there are commercially available antivenoms that can be given to patients unresponsive to supportive care. Antivenom appears to reduce pain duration and severity and the need for hospitalization.[103,104] The mortality rate for black widow spider bites has been reported as 5%.

Snakebites

Snakebites are a significant problem in the United States. Successful treatment requires prompt definitive medical evaluation, careful clinical assessment, and timely administration of antivenom in selected patients. Consultation with a medical toxicologist or other clinician with experience in poisonous snakebites is recommended. There are approximately 120 known snake species that are indigenous in the United States, but only 40 of these species are believed to be venomous to humans. Approximately 5000 snakebites are reported to the American Association of Poison Control Centers annually.[105] Most of these occur in adult males. Clinical effects are more severe in children and in victims of rattlesnakes or water moccasin (cottonmouth) envenomation. Most snakebites occur in the summer months when snakes are most active. Southern and western states with warmer climates report the greatest

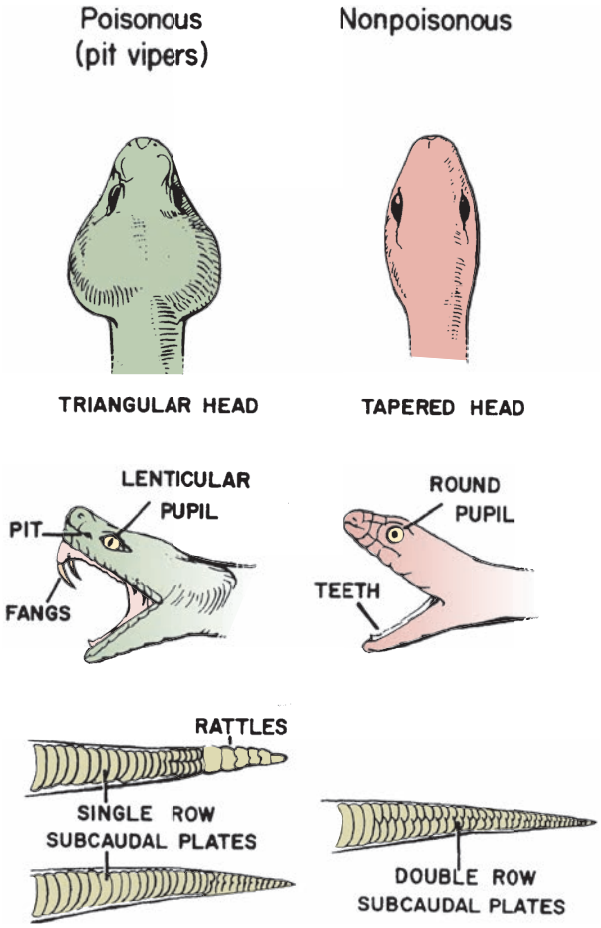


FIGURE 50-2 Characteristics of poisonous versus nonpoisonous snakes.

number of venomous snakebites, including Texas, Florida, California, Arizona, Louisiana, Georgia, and North Carolina.

The Viperidae and Elapidae families are primarily responsible for all venomous bites to humans (Fig. 50-2). The Elapidae family includes the king cobra, the world's longest venomous snake, measuring up to 18 ft in length. Elapids possess a pair of fangs through which they can inject their venom, a potent neurotoxin that induces paralysis, making elapid bites potentially lethal. Coral snakes are also members of the Elapidae family, and these snakes are relatively small and are known for their bright distinctive coloration forming rings around the body of the snake. The venomous coral snakes may be distinguished from a similarly appearing benign snake by the sequence of the coloration. If a red band is adjacent to a yellow band, then this is a venomous snake, whereas if the red band touches a black band, then the snake is lacking venom. This has led to the oft-stated idiom "Red on yellow, kill a fellow; red on black, venom lack"; however, this association of coloration and presence of venom only applies to coral snakes found in North America. Coral snakes have short fangs, which may have difficulty penetrating thick clothing, and bites from these snakes are relatively rare due to their rather reclusive, burrowing, and nonaggressive nature.

TABLE 50-15: Crotalinae of North America

Genus	Common name	Characteristics	Range
Agkistrodon	Moccasins, copperheads	No rattles, large plates on crown	North America, southeastern Europe, Asia
Crotalus	Rattlesnakes	Rattles, small scales on crown	North, Central, and South America
Sistrurus	Massasaugas and pygmy rattlesnakes	Rattles, large plates on crown	North America

Despite their timid behavior and limited fangs, coral snakes possess some of the most potent venom among snakes in North America; thus, they should not be taken lightly or else significant morbidity and mortality may ensue.

Rattlesnakes, water moccasins (cottonmouths), and copperheads are members of the family Viperidae, subfamily Crotalinae (Table 50-15). They are also commonly called pit vipers, a name that refers to the heat-sensing pit located behind the nostrils. Rattlesnakes, water moccasins (cottonmouths), and copperheads are the most numerous and cause the most snakebites in the Unites States. Elapids (coral snakes) and imported exotic snakes cause a smaller number.[106] Characteristic features of copperheads include a broad triangular head with a thick body and facial pits. Rattlesnakes possess the characteristic rattles often heard before an attack, and these may aid in distinguishing a rattlesnake attack.

An understanding of the effects of snake venom and US distribution of snakes is important when assessing and treating patients. Symptomatic Crotalinae (rattlesnake, water moccasin, or copperhead) envenomation is best considered as an envenomation syndrome.[107] Local tissue damage consists of ecchymosis and progressive tissue swelling. Nonspecific systemic effects are nausea, vomiting, diarrhea, weakness, lightheadedness, diaphoresis, and chills. Coagulopathy and rhabdomyolysis with nephrotoxicity can also occur. Increased vascular permeability, tachycardia, tachypnea, and hypotension can also be seen, as well as neurotoxicity.[108]

The venom of the Viperidae, and subspecies of rattlesnakes, water moccasins, and copperheads described earlier, often induces tissue necrosis because the venom possesses over 50 active components, including considerable amounts of metalloproteinases, phospholipases, and inflammatory mediators.[109] Symptoms of Viperidae envenomation include pain, especially with movement, swelling, erythema, and nausea. Moderate to severe envenomation is associated with tissue destruction from the enzymes present in the venom, vomiting, and bleeding with resultant tachycardia. Tissue destruction is related to matrix metalloproteinases (MMPs), phospholipases, and the inflammatory mediators inducing vascular endothelial damage as well as activation of platelets and complement. The MMPs in snake venom have been shown to be diverse and have a variety of adverse effects upon the coagulation/fibrinolytic pathways in humans, and may progress to a severe consumptive coagulopathy.[105,110] The degree of local destruction is considered a marker of the degree and nature of the envenomation. The cause of death from viper envenomation is commonly from cardiovascular collapse. The degree of tissue destruction and severity of the bite may vary considerably and are dependent on numerous factors including duration from bite to treatment, dose of venom in the wound, anatomic location of the wound, and the victim's immunologic response to either the venom or the antivenin. Greater consideration for possible extensive tissue necrosis must be given when the victim of the snake bite is a child, given the smaller volume of distribution in children for the same volume of injected venom.

All patient snakebite assessments begin with consideration of whether an envenomation has occurred. Examining for the presence of fang marks is very helpful, as the lack of fang marks effectively rules out a possible envenomation, but even if fang marks are identified, envenomation will have occurred in only 25% of bite victims. The following measures are recommended for US snakebite victims prior to definitive hospital care: remove the patient from the snake's territory, and keep him or her warm, at rest, and calm. Immobilize the injured body part in a functional position at the level of the heart initially.[111,112] Placing the extremity below the level of the heart may lead to increased tissue damage in some patients but may be appropriate in patients who have systemic effects of envenomation.[105] In contrast, elevation of a swollen extremity to prevent acute swelling in patients without systemic symptoms may subsequently increase systemic venom absorption but may be acceptable if the time to definitive care, including antivenom administration, is short.[111] Remove any rings, watches, or constrictive clothing; do not apply pressure immobilization, tourniquets, or constrictive dressings; cleanse the wound; and withhold alcohol and drugs that may confound clinical assessment. Transport the patient in the supine position to the nearest medical facility as quickly as possible, preferably using emergency medical services. In addition, attempts to identify the snake should not endanger the patient or rescuer and should not delay transport to a medical facility.[106]

Clinical evaluation begins with an assessment of the wound site and the adjacent tissues. "Dry bites," in which no venom was injected, occur in approximately 25% of snakebites in the United States and show only minimal local irritation. True Crotalinae envenomation produces swelling, pain, ecchymosis, and blister. Proximal spread of these signs suggests progressive toxicity. Systemic reactions, such as nausea, vomiting, abdominal pain, paresthesia, and dizziness, suggest more severe envenomation. Hypotension and altered mental status are ominous findings.[113] Elapidae envenomation, such as with Mojave rattlesnakes, does not typically produce significant local tissue reaction, and neurologic findings are pathognomonic. Onset of neurologic findings can

be delayed up to 12 hours.[114] Because symptoms of Elapidae envenomation rarely occur immediately, as the neurotoxins circulate, the effects manifest as tingling and numbness in the extremities, dysarthria, lethargy, and shallow respirations. Patients with suspected Elapidae envenomation who present with neurologic symptoms should be transported to a center of care capable of offering critical care, since these neurotoxic effects may progress to respiratory failure and the need for intubation and mechanical ventilation. Given the fact that the effects of Crotalinae envenomation have a relatively fast onset of action, it is reasonable to assume that a lack of symptoms upon arrival at a care center is highly associated with a lack of envenomation, and thus practitioners may withhold antivenin treatment or other advanced treatment options and follow with close clinical examination. It is important, however, to remember that the neurologic effects of Elapidae envenomation can be delayed up to 12 hours.[114]

The initial response to a snakebite, whether in the prehospital setting or following arrival to a definitive care center, should be focused on minimizing further harm from "therapeutic maneuvers." A spectrum of commonly employed measures undertaken at the scene may worsen the clinical situation; thus, field management of snakebites now focuses on rapid and expedient transport to a medical facility rather than attempting care in the field.[115,116] Methods such as tourniquets, incisions and oral suction, mechanical suction devices, stun gun therapy, immersion of the affected limb in ice, cryotherapy, surgery, and electric shock therapy have been tried in the past but are no longer recommended[117] (Fig. 50-3).

Incision and attempted aspiration of possible venom is fraught with potential harm, as this is likely occurring prehospital outdoors with nonsterile hunting equipment by an individual with very limited knowledge of anatomy. Given the fact that 25% of snakebites will result in little or no envenomation, the risk of creating a potentially serious soft tissue infection far outweighs any potential theoretical benefit. Venom removal by mechanical suction is minimal and only reduces the total body venom burden by 2%.[118] Electric shock therapy administered from a stun gun, largely advocated by investigators from Ecuador, has also been proven to be ineffective as both clinical data and animal studies have demonstrated no benefit of electric shock therapy in snakebite wounds. Placing the affected limb into ice was considered a potential therapeutic option based on decreasing the temperature and diminishing the activity of the enzymes contained within the venom; however, not only has this been shown to be ineffective, but it has also led to considerable numbers of snakebite victims having the whole extremity immersed in ice with significant ice-related tissue loss and destruction with no evidence of actual envenomation. In addition to the previously mentioned therapies to avoid, another therapy that is no longer recommended is pressure immobilization. Patients with snakebites should not receive pressure immobilization. Pressure immobilization, originally described in 1979 for neurotoxic envenomation in Australia, remains controversial.[119] Pressure immobilization has failed to show a benefit in other regions of the world including in the United States, calling into question the Australian experience. In Australia, where travel to definitive care can involve traveling over vast areas and the rate of neurotoxin envenomation is considerably higher, this may theoretically work, but it has been discouraged in the United States where the predominance of true envenomation is tissue destructive rather than neurotoxic. Pressure immobilization refers to a procedure in which an elastic bandage is applied to the affected limb with a goal of achieving a bandage pressure of 55 to 70 mm Hg. This method may delay systemic absorption of snake venom by preventing lymphatic spread in the affected limb; however, this may cause increased tissue damage, especially with Crotalinae envenomation, due to ischemia from the pressure immobilization in combination with the local tissue damage caused by the venom.[117] No studies have demonstrated benefit in envenomated humans, and if anything, studies have shown worse outcomes due to sequestering the venom in the affected limb.[120] Furthermore, application of the pressure immobilization to ensure the correct pressure is exceedingly difficult to achieve. When incorrectly applied with elevated pressures, pressure immobilization becomes effectively an arterial tourniquet; thus, the use of a pressure immobilization is greatly discouraged.

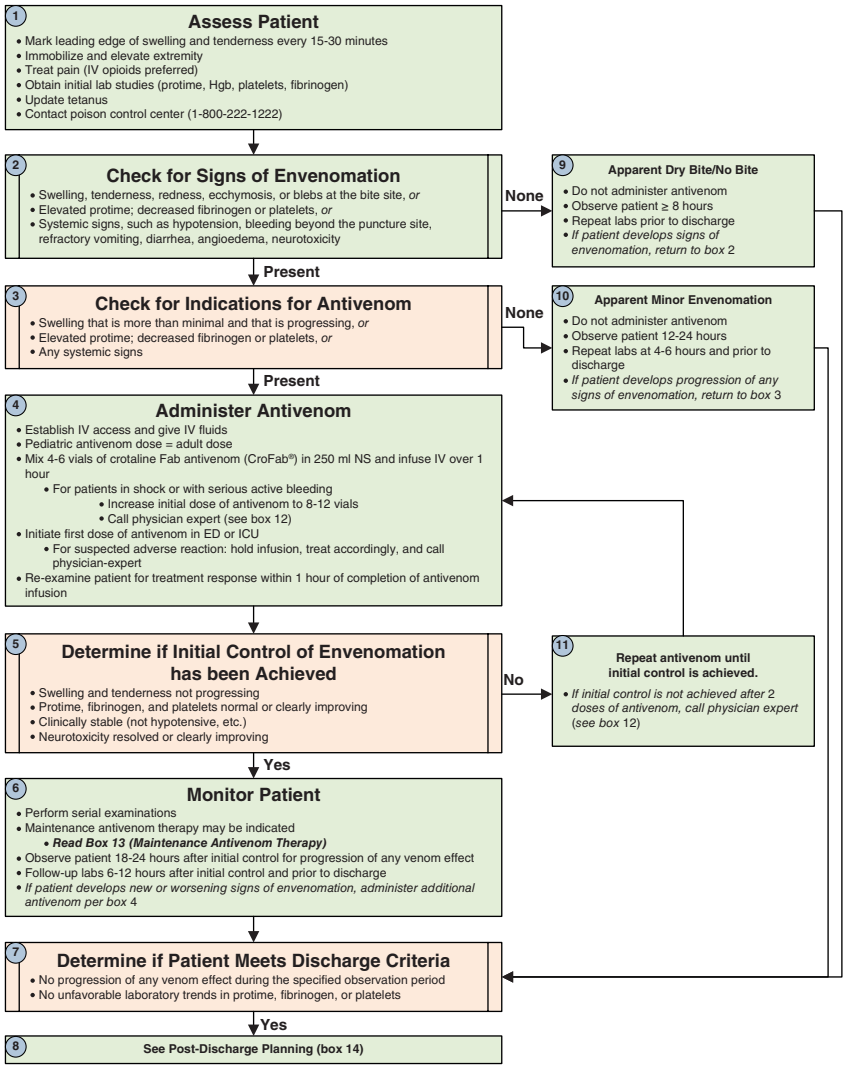
Tourniquet application has been advocated and employed by many first responders, but the application of an arterial tourniquet for greater than 2 hours may result in severe ischemic damage to the extremity with potential limb loss from the tourniquet.[113] Venous tourniquets, loosely applied, have the potential theoretical benefit of impairing venom outflow while allowing arterial inflow to perfuse the extremity; however, these tourniquets are often difficult to correctly apply in the field due to lack of knowledge or experience. The proper application of a tourniquet in this situation is loose enough to allow the passage of one to two fingers while also being tight enough to impede venous return, but it should be noted that animal models showing a theoretical benefit have not been proven in clinical studies.[113] Furthermore, it is postulated that containing the venom in a restricted region may increase the potential for local tissue destruction; therefore, the application of a tourniquet is best avoided in all cases of snakebites.

Once the patient arrives at the site of definitive care, care should commence with supportive measures such as gaining intravenous access, fluid resuscitation, pain control, and a clinical assessment and good documentation of the presence and/or extent of tissue injury, which will aid in following for progression of tissue loss. Lab work should be sent (including complete blood count, serum electrolytes, creatinine, blood urea nitrogen, serum creatine kinase, prothrombin time, partial thromboplastin time, international normalized ratio, D-dimer, fibrinogen, and urinalysis) and electrocardiogram performed to help identify coagulopathy, rhabdomyolysis, and acute kidney injury.

Ongoing supportive care should encompass fluid resuscitation, pain control, administration of tetanus prophylaxis, and correction of coagulopathy. Although the mouth of a snake is colonized with bacteria like all animals, infection

Emergency Department and Hospital Management of Pit Viper Snakebite

Includes: Rattlesnakes, Copperheads, and Cottonmouths (Water Moccasins)



12 When to Call a Physician-Expert

Direct consultation with a physician-expert is recommended in certain high-risk clinical situations:

- **Life-threatening envenomation**
  - Shock
  - Serious active bleeding
  - Facial or airway swelling
- **Hard to control envenomation**
  - Envenomation that requires more than 2 doses of antivenom for initial control
- **Recurrence or delayed-onset of venom effects**
  - Worsening swelling or abnormal labs (protime, fibrinogen, platelets, or hemoglobin) on follow-up visits
- **Allergic reactions to antivenom**
- **If transfusion is considered**
- **Uncommon clinical situations**
  - Bites to the head and neck
  - Rhabdomyolysis
  - Suspected compartment syndrome
  - Venom-induced hives and angioedema
- **Complicated wound issues**

If no local expert is available, a physician-expert can be reached through a certified poison center (1-800-222-1222) *or the antivenom manufacturer's line* (1-877-377-3784).

13 Maintenance Antivenom Therapy

- Maintenance therapy is additional antivenom given after initial control to prevent recurrence of limb swelling
  - Maintenance therapy is 2 vials of antivenom Q6H x 3 (given 6, 12, and 18 hours after initial control)
- Maintenance therapy may not be indicated in certain situations, such as
  - Minor envenomations
  - Facilities where close observation by a physician-expert is available.
- Follow local protocol or contact a poison center or physician-expert for advice.

14 Post-Discharge Planning

- Instruct patient to return for
  - Worsening swelling that is not relieved by elevation
  - Abnormal bleeding (gums, easy bruising, melena, etc.)
- Instruct patient where to seek care if symptoms of serum sickness (fever, rash, muscle/joint pains) develop
- Bleeding precautions (no contact sports, elective surgery or dental work, etc.) for 2 weeks in patients with
  - Rattlesnake envenomation
  - Abnormal protime, fibrinogen, or platelet count at any time
- Follow-up visits:
  - Antivenom not given:
    - PRN only
  - Antivenom given:
    - Copperhead victims: PRN only
    - Other snakes: Follow up with labs (protime, fibrinogen, platelets, hemoglobin) twice (2-3 days and 5-7 days), then PRN

15 Treatments to Avoid in Pit Viper Snakebite

- Cutting and/or suctioning of the wound
- Ice
- NSAIDs
- Prophylactic antibiotics
- Prophylactic fasciotomy
- Routine use of blood products
- Shock therapy (electricity)
- Steroids (except for allergic phenomena)
- Tourniquets

16 Notes:

- **All treatment recommendations in this algorithm refer to crotalidae polyvalent immune Fab (ovine) (CroFab®).**
- This worksheet represents general advice from a panel of US snakebite experts convened in May, 2010. No algorithm can anticipate all clinical situations. Other valid approaches exist, and deviations from this worksheet based on individual patient needs, local resources, local treatment guidelines, and patient preferences are expected. **This document is not intended to represent a standard of care.** For more information, please see the accompanying manuscript, available at www.biomedcentral.com.

FIGURE 50-3 Algorithm for the management of a pit viper snakebite. ED, emergency department; ICU, intensive care unit; IV, intravenous; NS, normal saline; PRN, as needed; Q6H, every 6 hours. (Reproduced with permission from Lavonas EJ, Ruha AM, Banner W, et al. Unified treatment algorithm for the management of crotaline snakebite in the United States: results of an evidence-informed consensus workshop. *BMC Emerg Med.* 2011;11:2.)

following a snakebite is rare. In a placebo-controlled trial, there was no difference in infectious outcomes with routine administration of an antimicrobial agent following snakebites; thus, routine prophylactic antimicrobial agents are not indicated.[121] Secondary bacterial infections are much more likely to occur in patients who had their wound manipulated in the field in an attempt to "extract" the venom. Antibiotics should only be administered for established infections or for heavily contaminated wounds.[106] Tetanus prophylaxis should be administered to snakebite patients according to the recommended immunization schedule (Table 50-7).

Coagulopathy is a more frequent complication of envenomation and should be corrected based on laboratory values of clotting times and blood products levels as well in response to evidence of bleeding. The coagulopathy associated with Crotalinae (rattlesnake, water moccasin, or copperhead) envenomation is due to thrombin-like glycoproteins within the venom as well as thrombocytopenia.[107] This is in contrast to true disseminated intravascular coagulation where fibrinolysis is activated by increased levels of endogenous thrombin. Transfused platelets and coagulation factors in fresh frozen plasma are inactivated by Crotalinae venom and should be avoided in patients with envenomation-induced coagulopathy unless significant bleeding occurs that is uncontrolled by high-dose antivenom administration.[107] Thus, antivenom administration, and not coagulation factor replacement, is the primary treatment for Crotalinae-induced coagulopathy.

Tissue destruction caused by snake envenomation may lead to muscle cell breakdown (rhabdomyolysis) with significant risk for renal failure. Patients with rhabdomyolysis typically present with the triad of pigmented granular casts in the urine, a red to brown color of the urine supernatant, and a marked elevation in the plasma level of creatine kinase. Primary treatment goals consist of fluid repletion and evaluation for significant electrolyte abnormalities (hyperkalemia, hyperphosphatemia, hypocalcemia).[113]

The surgical management of envenomation has changed over the past 20 years, as excising the region of the bite attempting to remove the venom-laden fangs combined with empiric fasciotomy has fallen out of favor. Now, surgical intervention is limited to debridement of necrotic or devitalized tissue and wound care. Elevated tissue or compartment pressures may complicate Crotalinae bites. Any dressing, constrictive band, splint, cast, or other restrictive covering should be removed. Venom is usually introduced into the subcutaneous tissues, and edema occurs in this space. Tissue pressures may increase because of the massive amounts of subcutaneous tissue fluid and because the skin has limits of elasticity. Swelling, pain, and paresthesia may occur in patients after Crotalinae snakebites even in the abscesses of elevated tissue compartment pressures.[113] Thus, surgical intervention based on clinical findings alone is inappropriate. Increased compartment pressures result from extrinsic pressure and can be reduced with the administration of adequate amounts of antivenom and elevation. Elevation is usually avoided in true compartment syndromes and prior to antivenom administration in patients with venomous snakebites. After antivenom administration, however, elevation results in the drainage of subcutaneous edema and contributes to the reduction of the source of increased tissue pressure.[106] The prior rationale for early aggressive operative intervention lay in the postulation that the tissue destruction was a local effect amenable to locally excising the infused venom with the surrounding necrotic muscle and decompressing the adjacent healthy tissues pending resolution of the inflammation.[122] However, it has been clearly demonstrated that empiric operative intervention or empiric fasciotomy is not indicated for envenomation and that muscle necrosis is more likely the result of the ongoing venom rather than the presence of compartment syndrome.[123] Although the risk of compartment syndrome is low, it still occurs. There is a wide range in the literature on the rate of rattlesnake envenomation progressing to require fasciotomy and decompression (0%–15%), but it is estimated that the incidence of true compartment syndrome needing fasciotomy rather than just "prophylactic fasciotomy" is considerably lower, at approximately 1% to 2%.[124,125] Compartment syndrome is more likely to occur in the anterior leg, fingers, or hand and is usually associated with deeper bites. True compartment syndromes may result from direct compartmental injection of venom, particularly with the anterior tibial compartment, which is very close to the skin. In these instances, antivenom and elevation may still reduce compartment pressures by the reduction of extrinsic pressure, but persistent intercompartmental pressures may remain high. The indications for fasciotomy in this context are unclear. An animal model of direct compartmental injection of venom demonstrated improved outcomes with antivenom alone versus antivenom plus fasciotomy.[126] In this model, fasciotomy was performed immediately after venom injection. The diagnosis of compartment syndrome may be difficult given the overlap in clinical findings, such as pain with passive motion, pain out of proportion to findings, swelling, and firmness to exam, between severe local tissue inflammation and true compartment syndrome. Thus, it is highly recommended to base the decision to intervene surgically on objective measures of the compartment pressures rather than exclusively on physical examination.[124] Therefore, surgical intervention for elevated compartment pressures following Crotalinae snakebites is controversial and should be guided by a medical toxicologist and surgeon with extensive experience caring for victims with snakebites.[127] With fasciotomy, acute debridement is to be discouraged, as injured muscle may improve with the concomitant administration of antivenin and supportive care. Despite the infrequency of progression to fulminant tissue necrosis and/or compartment syndrome, snakebites should still be considered a potential surgical disease, and early surgical consultation is critical given the consequences of missing progression to compartment syndrome.[128]

It must be recognized that the majority of snakebites that occur in the United States are nonvenomous, and given the potential for adverse reactions to antivenin, an attempt should be made to identify the snake, preferably by an individual trained in herpetology. Originally created in 1954, commercial Crotalinae antivenin has undergone extensive

modifications over the years to minimize its side effects. The original antivenin contained whole immunoglobulin G including the Fc fragment, which led to severe hypersensitivity reactions including bronchospasm and cardiovascular collapse. Anaphylactic reactions were reported to occur in as many as 50% of administrations, and much higher rates of serum sickness were also noted.[127] This early product was also limited in its ability to work against the rapidly spreading neurotoxin or in controlling the coagulopathic and hemorrhagic components of the envenomation. Modifications in antivenin design allowed for cleavage of the Fc and the Fab segments, isolating the Fab preparations for the final antivenin, which minimized its side effects, with serum sickness being reported in about 15% of patients. Polyvalent Crotalinae ovine immune Fab (FabAV, Crofab, Protherics) is the antivenom currently commercially available in the United States for Crotalinae (rattlesnake, water moccasin, or copperhead) envenomation.[127] Equine F(ab)2 antivenom (Anavip) has been used successfully to treat US pit viper envenomation in one study and may reduce the risk of late coagulopathy, and is now available for clinical use. FabAV consists of the purified Fab fragments of sheep immunoglobulin (IgG) raised against the venom of four snakes.[127,129] When infused, these Fab fragments bind venom in the intervascular space and are renally excreted. The half-life of FabAV is shorter than that of Crotalinae venom substances. Thus, recurrent toxicity is possible despite initial control of local and systemic effects and may necessitate repeated FabAV administration. It is most effective when given within 6 hours of envenomation.[130]

It is recommended that patients with Crotalinae bites (rattlesnake, water moccasin, cottonmouth, or copperhead bites) and moderate to severe toxicity or with bite sites that present a significant possibility for airway obstruction from local tissue swelling (bites to the face or neck) receive FabAV therapy.[113] In these patients, the benefits of treating life-threatening toxicity clearly outweigh the potential risk of hypersensitivity. Guidelines have suggested that patients with mild envenomation not receive antivenom, but some evidence suggests that it may provide short-term benefit, primarily a more rapid return to normal function of the involved limb.[117,131] The use of antivenin is discouraged in cases of mild envenomation, and antivenin should not be administered to individuals with a history of allergic reactions to antivenin or in settings where a severe life-threatening allergic reaction cannot be managed.[132] Administration should be through slow infusion while watching for an allergic reaction and stopping immediately upon detecting any hypersensitivity. Antivenin treatment, although ideally administered before injury, remains effective if given up to 6 hours following the envenomation, but there have been anecdotal reports of effective antivenin therapy when administered later. In life-threatening envenomation, antivenin has been administered up to 24 hours following the bite with variable benefit.[132]

Even when used correctly, many of the existing antivenins are associated with considerable allergic side effects that, on occasion, may induce severe organ failure. Even with milder cases of allergic reactions to antivenin, the adverse effects may last up to several months.[132,133] The dose of antivenin administered remains empiric and is based on a clinical judgment of the amount of venom delivered from the bite; thus, adults and children are dosed based on the amount of venom from the bite and not on patient weight. Multiple administrations may be necessary, with 10 vials or more potentially required for patients with large exposures and severe envenomation. In patients with progressive clinical manifestations, repeat doses may be required.[113] Thus, the use of antivenin for patients with mild envenomation should be individualized on a case-by-case basis with input from a medical toxicologist or other snakebite expert. For patients with Crotalinae envenomation but minimal and nonprogressive swelling, pain, or ecchymosis, and in whom the decision is to withhold antivenom, we suggest close observation for 12 to 24 hours to ensure that progression of toxicity, as indicated by worsening local or systemic effects, does not occur. All patients who receive antivenom should receive close observation for recurrent toxicity over the first 24 hours after antivenom administration.[134] Recurrent toxicity may occur despite initial control with antivenom. This is related to the short half-life of Fab fragments or possibly dissociation of Fab fragment–venom complexes.[135] As a result, guidelines suggest that those who receive FabAV administration with moderate-severe envenomation should receive scheduled doses of 2 g every 6 hours for three doses to prevent recurrent toxicity.

The clinician should immediately stop antivenom infusion in patients who experience signs of acute hypersensitivity (anaphylactic shock, oropharyngeal swelling, bronchospasm, and urticaria). These patients should receive treatment for anaphylaxis. Because acute reactions are often nonimmunologic in nature, the antivenom infusion may be resumed cautiously and completed at a lower infusion rate once initial signs of acute hypersensitivity have been treated.[113] Allergic reactions can be secondary to venom sensitivity, particularly in those who have been bitten before. In all cases of anaphylaxis, cessation of antivenom administration is part of the management of anaphylaxis. Once anaphylaxis is controlled, restarting the antivenom should be based on risk-benefit analysis, and if the decision is made to restart, then it should be administered at a lower infusion rate.[132] Again, consultation with a medical toxicologist experienced in the management of snakebites is strongly recommended for these patients.

Considerable limitations have developed over the past 10 years with respect to coral antivenin since its production has ceased in the United States due to the rare incidence of coral snake envenomation. Production of coral antivenin in the United States ceased in 2003, and most stocks of coral antivenin in the United States expired roughly 5 years later. Although licensing and expiration dates have been amended several times, there is no longer any coral snake antivenin available.

Overall the use of antivenin has increased over the past 15 years, but this may change as stocks of antivenin expire and are not being reproduced. Despite increasing awareness of the potential benefit of antivenin, many institutions do not

stock, or are unaware of the availability of, the appropriate antivenin.[113]

STINGS

Venomous Fish

The seas and oceans contain several potential stinging or biting hazards to humans. These include venomous fish, jellyfish, sea snakes, coral, sea urchins, and stingrays. These envenomations are painful. The degree of pain is largely determined by the amount of venom injected and the animal encountered. The puncture wounds associated with marine envenomation often have retained foreign bodies and are frequently contaminated. Most wounds are superficial, although stingrays can cause deep penetrating injuries. Marine envenomation may induce a spectrum of manifestations including allergic reactions, neurotoxic effects such as paralysis, and cardiac depression due to cardiotoxins.[136]

Coral are cnidarians, like jellyfish, and members of the class of Anthozoa.[137] Stings and lacerations are common among snorkelers, surfers, and scuba divers who touch or step on coral.[138] Coral stings are usually only mildly toxic with local pain and erythema. The sting is caused by venomous capsules called nematocysts and can be treated similarly to jellyfish stings. Lacerations from coral are prone to secondary infection, including cellulitis, lymphangitis, fever, and ulceration of the wound within a few days of injury.[137,139]

Sea urchins are echinoderms and the most significant member of this phylum in the United States. Other members include starfish and sea cucumbers, which have no venom apparatus (poisonous rather than venomous). Sea urchins have long, sharp spines made of calcium carbonate that easily penetrate flesh, rubber-soled shoes, or wetsuits and can break off in the wound. Some species of sea urchins have venom-producing glands at the tips of the spines and thus are venomous.[140] Clinical findings include redness, swelling, bleeding, and intense pain, which can last up to 24 hours.[138] Discoloration of the wound can indicate retained sea urchin spines. If this discoloration resolves in 48 hours, it is unlikely that a spine is still lodged in the skin.[141] Imaging can help identify retained foreign body.

There are over 150 species of stingrays worldwide, accounting for thousands of injuries every year.[137] Stingrays are flat, cartilaginous fish with a long, tapered whiplike tail with a furrowed, serrated spine containing secretory venom cells and sacs. This tail is noted to contain multiple barbs containing venom, and this venom is noted to be cardiotoxic. It also contains proteolytic and hyaluronidase activities, which may contribute to tissue necrosis and skin loss around the site of the sting. The flat-shaped nature of the stingray body allows it to easily burrow under a shallow layer of sand and thus remain unexposed and difficult to visualize, leading to stings; however, gentle agitation of the sand will disturb the stingrays, prompting them to move away, as stingrays are not aggressive by nature. When a stingray is stimulated or frightened, it flings the barbed tail upward, embedding the spine in the victim, which releases the venom.[142] Most stingrays possess one or more barbed stingers located on the tail that are loaded with venom and are used by the stingray as a defense mechanism; thus, these animals should be left alone in order to minimize the likelihood of a sting.[137] Most stings occur from accidentally stepping on a stingray while walking in the surf and wading at shallow ocean depths. Injuries to the upper limbs are less common but can occur when stingrays are removed from fishing lines. Thoracoabdominal trauma can occur rarely to divers. Although most stingray stings are not fatal, they do induce considerable pain from both the site of the sting as well as from venom-induced muscle cramps.[142] Pain may radiate up the limb and usually persists for hours. The wound may be erythematous and dusky. There is also usually significant bleeding, which depends on the site of injury. Although numerus systemic effects are reported, they are uncommon and usually related to the systemic response to pain.[136] The size and length of the stingray spine will dictate the extent of injury, and larger spines increase the risk of trauma and secondary infection. Death or severe injury may occur if the spine penetrates a major blood vessel, heart, or other vital organ.[143] Thoracoabdominal injuries should be managed like major penetrating trauma but have the additional risk of foreign body retention and infection; as such, those wounds that extend through the chest or abdominal wall will need exploration in the operating room.[144] Imaging is undertaken to assess for possible remaining fragments of the barb, and wound exploration should be completed as the barb from the tail may break off and remain lodged in the stung extremity. Stingray wounds should be left open to heal by secondary intention, and surgical debridement may be indicated in cases of significant necrosis. Prophylactic antimicrobial agents are administered given the relatively high rate of infection. Tetanus prophylaxis should be updated, but currently there is no available stingray antivenin.[136]

Venomous fish have a specific apparatus designed to inject poison. Stings occur in the marine and freshwater environment and also from private aquariums.[145] Spines cover integumentary sheaths, which contain venom glands. When the spine penetrates the victim's skin, the surrounding integument covering the spine ruptures and venom is injected into the wound. There are two families of venomous spiny fish; the Scorpaenidae include stonefish and lionfish, and the Trachinidae contain the weeverfish.[136] Stonefish and weeverfish usually cause injuries when they are stepped on by swimmers or divers because they are bottom-dwellers, and lionfish usually cause envenomation of the hands or fingers during routine care and feeding by their handlers.[146] Lionfish are often kept as part of home aquariums, which increases the incidence of stings caused by this tropical fish, and stings from lionfish are the most common marine-related sting called in to poison centers in the United States. The pain of these venomous fish stings may resolve over 1 hour in minor injuries, but with more severe stings, the pain may persist for over 24 hours and may radiate. Systemic effects have been reported in more severe cases, including nausea, vomiting, headache, sweating, hypotension, and syncope. However, these are most

likely secondary to the severe pain. Most patients experience moderate to severe pain at the sting site, and stings from lionfish may induce significant skin necrosis.[147] Stonefish toxins may induce local tissue necrosis, severe muscle contracture, and neurotoxicity and may even induce cardiotoxicity with severe cardiac depression and death.[146] The weeverfish inhabits the waters of Europe and the Mediterranean and can inflict a painful sting, typically to the foot or ankle due to humans stepping on them at low tide. These stings require local wound care only, and there are no confirmed fatalities due to this fish.[148] Fatalities have been attributed to many kinds of venomous fish; however, they are usually due to septicemia from secondary infection.

Sea snakes reside in a variety of tropical regions, including the warmer portions of the Indian and Pacific Oceans from the east coast of Africa to the west coast of the United States and Central and South America. They are found close to land in coral reefs or along islands.[149] The bite is often painless with limited local tissue injury; however, after a dormant period that can last up to several hours, systematic effects become apparent, including muscle pain, tenderness, trismus, weakness, rhabdomyolysis in severe cases, acute kidney injury, and hyperkalemia.[150] Sea snake envenomation may warrant treatment with antivenom depending on severity.

Cnidaria comprise four different classes including Cubozoa (box jellyfish), Scyphozoa (true jellyfish), Anthozoa (anemone), and Hydrozoa (Portuguese man-o-war). All species of Cnidaria have hollow, sharp-pointed, coiled thread tubes surrounded by venom called cnidae. The jellyfish and sea anemones do not cause puncture wounds. After a jellyfish sting, a linear red urticarial lesion typically develops a few minutes later, although these lesions sometimes do not appear for several hours. "Tentacle prints" may be seen on examination of the skin. Nematocysts can be obtained by skin scrapings or by applying sticky tape to the sting site when diagnosis is unclear. Sea anemone stings present as circular and painful urticarial lesions, which may have a central pallor. Most jellyfish stings induce an acute dermatitis and are self-limiting; however, box jellyfish, residing mainly off the coast of Australia, are the deadliest of jellyfish and some have contended that box jellyfish may be the world's most venomous creature. Most reported cases of box jellyfish stings involve painful lesions that may progress to skin necrosis within 12 to 18 hours. Although the exact mechanism of action of the venom is largely unknown in humans, it is postulated that the venom causes cellular pore formation, leading to massive and swift sodium and calcium fluxes into cells with rapid cellular compromise. This venom leads to rapid hypotension, cardiovascular collapse, and death, which has been reported to occur in as little as 60 seconds after the sting. Treatment of all jellyfish stings involves nematocyst removal using seawater and gentle rubbing. It is not recommended to immerse in cool fresh water or vigorously rub the wound because this will cause the nematocysts (tentacles) to fire. After their removal, it is recommended that the pain from the stings of the box jellyfish and bluebottle jellyfish (man-of-war) be treated with hot water immersion, acetic acid, or papain meat tenderizer. Hot water immersion should be for approximately 20 minutes.[151]

Fire Ants

Fire ants, both red (*Solenopsis invicta*) and black (*Solenopsis richteri*), were originally imported into the southern United States in the 1920s, but these insects continue to march forward, and colonization has extended across vast areas of the United States. Red imported fire ants are the most abundant and damaging species.[152] The habitat of the black imported fire ant is relatively limited. Stings occur most frequently in the summer and typically on the lower extremities. Off-season stings often do not cause as much pain and may go unnoticed. This may reflect seasonal differences in fire ant venom.[153] Fire ant bites will not sting through fabric, stockings, or tights.[154] Thus, wearing shoes with socks and long pants is a helpful avoidance measure. Hands and arms should be covered with gloves and sleeves when gardening or doing outdoor work. Insect repellents are not effective. Approximately 5% of victims of fire ant bites will require medical attention, and of those, approximately a fifth will have systemic allergic reactions.[155] The fire ant is capable of multiple stings during one attack if not removed quickly, leading to a characteristic pattern of bites in a circular or linear pattern of pustules on the victim. The victim experiences an immediate, intense burning and itching at the sting site. The multiple stings in succession give the feeling of "fire" along the site, and the majority of the fire ant venom is composed of nonprotein piperidine alkaloids, which lead to the characteristic pustules following the stings. The venom is 95% water-insoluble alkaloids by volume, with the remaining 5% being an aqueous solution of proteins. The alkaloids cause the sterile pustule but are not allergenic. The allergenic portion of the venom is the protein component.[156] Following a fire ant sting, immediate washing of the stung area will help to reduce the venom load. Akin to other stings, the effects can be classified as local, local large, or systemic, with about 2% of patients presenting after a fire ant sting with a severe systemic reaction. Patients manifesting systemic symptoms require prompt medical attention since these reactions may progress to full-blown anaphylaxis.[155] If a patient develops a severe local reaction to the bite or survives a systemic reaction to the venom, then referral to an immunologist for workup of a fire ant venom allergy, possible skin testing, and immunotherapy to minimize the response to repeat exposure to fire ant venom is warranted.

Bee Stings

Hymenoptera species that sting humans include bees, wasps, yellow jackets, hornets, and imported fire ants. Most people develop only minor local reactions, but patients with venom allergy are at risk for systemic allergic reactions (anaphylaxis), which can be severe and is the leading cause of death from stings. There are also several uncommon and delayed types of reactions that may develop after Hymenoptera stings, such as serum sickness.[157] Hymenoptera generally sting people in

self-defense or to protect their nests or hives. Their stings are acutely painful. Identification of the species can be difficult after the sting since the resultant lesions are similar in appearance. Bees (and some yellowjackets) have a barbed stinging apparatus that can lodge in the skin and rip away, along with the venom sac, from the insect's body.[158] Venom is released almost immediately, so if the stinger can be removed immediately, it may help limit the amount of venom injected. After this, immediate stinger removal is not critical, because the venom will have already been fully expelled.[159] Honey bees differ from wasps in that the stinging mechanism of the bee is strongly barbed and is designed to remain in the flesh, whereas wasp stings are not intended to remain in the victim, which facilitates multiple stings. The presence of a retained stinger distinguishes bee and wasp sting, aiding in identifying those who will benefit from allergic reaction therapies.[160] Local reactions consist of symptoms that are confined to the tissues contiguous with the sting site. Stings from bees and wasps are relatively common during the warmer months of the year, and the vast majority of these remain unreported, requiring only local wound care or topical analgesics. They are usually mild and transient, although some patients develop large local reactions or rarely secondary bacterial infections.[161] Ten percent of individuals who sustain a wasp or bee sting develop a large local reaction, and approximately 1% to 3% of individuals who are stung will display a life-threatening allergic reaction.

Stings from bees can be categorized as minor local, major local, or systemic. Local symptoms include pain and erythema, but the presence of symptoms distal to the site of the sting defines a systemic response. A typical local reaction to a Hymenoptera sting is redness and an area of painful swelling (1–5 cm) at the site of the sting that develops within minutes and resolves within a few hours. Occasionally, swelling may last 1 to 2 days. Uncomplicated local reactions may be treated with cold compresses. Large local reactions result in exaggerated redness and swelling at the site of the sting that gradually enlarges over 1 to 2 days. These reactions peak at approximately 48 hours and then gradually resolve over 5 to 10 days. The area of swelling typically measures about 10 cm in diameter.[162] Treatment of a large local reaction is based on symptoms and includes cold compresses, elevation, oral prednisone as a single dose or rapid taper, nonsteroidal anti-inflammatory drugs, and oral antihistamines.[163] Hymenoptera stings are considered "clean" for the purposes of tetanus vaccination. Large local reactions and anaphylaxis are believed to be immunoglobin E mediated. Systemic symptoms include nausea, vomiting, and bronchospasm, and ultimately may progress to anaphylaxis and cardiovascular collapse. The incidence of anaphylactic reaction and deaths from bee stings is exceedingly rare yet constitutes more deaths per year than from all other venomous animals. Anaphylaxis may be defined as a serious allergic reaction that is rapid in onset and may cause death.[164] A single sting is sufficient to precipitate a severe reaction in a venom-allergic individual, and insect stings are a leading cause of fatal anaphylaxis, requiring multiple doses of epinephrine.[164] The reported deaths from bee stings are usually related to anaphylaxis and a delay in seeking medical care once the symptoms start. Small children are especially at risk of larger allergic reactions since the venom load is consistent among the bee population and is not based on the weight of the victim. Immunoprophylaxis is available, but there are several limitations for its use.[164] Any person who has exhibited bee sting anaphylaxis should be equipped with an emergency allergy kit and epinephrine pen and should be referred for consideration of immunoprophylaxis (Table 50-16).

TABLE 50-16: Treatment for Anaphylaxis

1. Airway control.
2. Epinephrine, 0.5 mg SQ (may be given IV with major anaphylactic reaction). May be repeated every 20 min.
3. Volume expansion.
4. H_1 antagonists (diphenhydramine, 25–50 mg IV or IM).
5. H_2 antagonists (ranitidine, 50 mg IV).
6. Glucocorticoids (hydrocortisone, 125 mg IV). No effect for several hours, but may prevent recurrence of symptoms.
7. Inhaled β_2-agonist for recurrent bronchospasm (albuterol, 0.5 mg).
8. Removal of bee stinger.

IM, intramuscularly; IV, intravenously; SQ, subcutaneously.

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51

Burns and Radiation

Benjamin Levi • Gary A. Vercruysse

KEY POINTS

- Early resuscitation after burn injury is key to mitigate shock. Follow the Parkland formula for resuscitation in burns greater than 20% total body surface area (TBSA).
- Use lactated Ringer's solution, not normal saline, as a resuscitation fluid to avoid hyperchloremic metabolic acidosis.
- Do not administer bolus intravenous fluids during initial burn resuscitation. This can lead to abdominal compartment and secondary extremity compartment syndromes.
- Consider early use of colloid (packed red blood cells, fresh frozen plasma, or albumin) in patients with low urine output despite adequate fluid resuscitation.
- Communicate with local burn center before performing escharotomy.
- Intubate prior to transfer in patients with extensive head, neck, or facial burns; elevated carbon monoxide levels (>30%), large burns (>40% TBSA), obtundation, or symptoms of early airway obstruction.
- In austere conditions, resuscitate orally when possible, graft only full-thickness burns and in small aliquots, and minimize ventilator use.

BURNS

Introduction

Approximately 1.25 million people are burned annually in the United States, of whom 30,000 are admitted to burn centers and 3400 succumb to their injury.[1,2] Traditionally, the highest incidence of burn injury occurs at the two extremes of age. During the first few years of life, burns are primarily due to liquid scalds and then, after the age of 60, injuries are commonly due to both flame and scald burns.[2-4]

Between 1971 and 1991, burn deaths decreased by 40% with a concomitant 12% decrease in deaths associated with inhalation injury.[5] Since 1991, burn deaths per capita have decreased 25% according to the Centers for Disease Control and Prevention (https://www.cdc.gov/injury/wisqars/index.html). These improvements are in part due to prevention strategies resulting in fewer burns of lesser severity as well as significant advances in treatment, particularly in children. Current reports indicate a 50% mortality for 98% total body surface area (TBSA) burns in children age 14 years and younger.[6] A healthy child with any size burn can be expected to survive.[7] The same is not true for those age 45 years or older, where improvements have been more modest, particularly in patients over 65 years of age, in whom a 35% TBSA burn still results in a 50% mortality.[8]

Burn Center Referral Criteria

The decision for in-hospital care of a burned victim varies with burn size, age, premorbid conditions, the patient's home situation, and access to local expertise. Patients with larger burns may benefit from treatment in specialized burn centers. These centers have dedicated resources and the required multidisciplinary approach to maximize outcomes from such devastating injuries.[9] The American Burn Association and the American College of Surgeons Committee on Trauma have established guidelines to identify patients who should be transferred to a specialized burn center, and these include the following[10]:

- Partial-thickness burns greater than 10% TBSA
- Burns that involve the face, hands, feet, genitalia, perineum, or major joints
- Third-degree burns in any age group
- Electrical injury, including lightning injury
- Chemical burns

- Inhalation injury
- Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality
- Any patient with burns and concomitant trauma in whom the burn injury poses the greatest risk of morbidity or mortality
- Burned children in hospitals without qualified personnel or equipment for the care of children
- Burn injury in patients who will require special social, emotional, or rehabilitative intervention

In an effort to mitigate unnecessary transfer of minor burns, efforts are underway by the American Burn Association to create consultative criteria, rather than transfer criteria, so that minor burns can be cared for at nonburn centers with burn center input, follow-up, and transfer as necessary. This patient-centered approach is designed to provide optimal patient care without the added expense of unnecessary transfers. If successful triage can be accomplished, a great opportunity for savings in the health care system can be achieved while still rendering safe and effective burn care.[3,4,11] In the past several years, advances in video-based consultation using smartphone technology have allowed for accurate initial consultation. This can then be used to facilitate appropriate triage decisions and guide initial care to a significant proportion of the patients previously triaged to burn centers.[12-14]

Pathophysiology

Burns are classified into six causal categories, three zones of injury, and five depths of injury (Table 51-1). The causes include fire, scald, contact, chemical, electrical, and radiation. Fire burns can be further divided into flash and flame burns, whereas scald burns can be divided into those caused by liquids, grease, or steam. Liquid scald burns can be due to spill or immersion scalds.

Flame, scald, and contact burns cause cellular damage primarily by the transfer of energy that induces coagulative necrosis. On the other hand, direct damage to cellular membranes is the cause of injury in chemical and electrical burns.

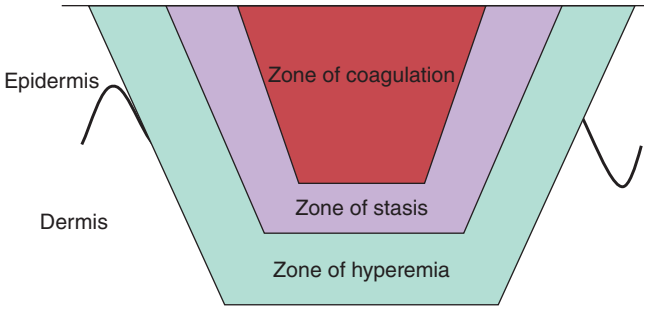

FIGURE 51-1 Zones of injury after burn. Factors likely to affect the zone of stasis determine the extension of injury from the original zone of coagulation.

The skin generally provides a barrier to limit transfer of heat energy to underlying tissues; however, after the source of burn is removed, the response of local tissues can lead to further injury. The necrotic area of a burn is termed the *zone of coagulation*. The area immediately surrounding the necrotic zone has a moderate degree of injury that initially causes a decrease in tissue perfusion. This area is termed the *zone of stasis* and, depending on the environment of the wound, can progress to coagulative necrosis if local blood flow is not maintained. Thromboxane A_2, a potent vasoconstrictor, is present in high concentrations in burn wounds, and local application of thromboxane inhibitors has been shown to improve blood flow and may decrease this zone of stasis.[15,16] Antioxidants and inhibition of neutrophil-mediated processes may also improve blood flow, preserve tissue, and decrease the depth of injury.[17,18]

Endogenous vasodilators such as calcitonin gene-related peptide and substance P, whose levels are increased in the plasma of burn patients, may also play a role.[19] Practically, the most important factor to minimize conversion of zones of stasis into zones of coagulation is adequate fluid resuscitation. The last area, the *zone of hyperemia*, is formed as a result of vasodilation from inflammation surrounding the burn wound. This zone contains clearly viable tissue from which the healing process begins (Fig. 51-1).

TABLE 51-1: Definition of Burn Types, Zones, and Depth of Injury

Burn	Categories	Zones of injury	Burn depth
Fire	Flash	Zone of coagulation	First degree (superficial)
	Flame		
Scald	Liquid	Zone of stasis	Superficial second degree (superficial partial thickness)
	Spill		
	Immersion		
	Grease	Zone of hyperemia	Deep second degree (deep partial thickness)
	Steam		Third degree (full thickness)
Contact			Fourth degree (deep organ involvement)
Chemical			
Electrical			
Radiation			

Inflammatory Response

There is a profound hypermetabolic response and massive release of inflammatory mediators following burns. This hypermetabolism is associated with alterations in blood glucose as well as lipids and typically occurs in the ebb phase (first 14 hours) and flow phase (until 5 days after burn).[20,21] Many mediators have been proposed to explain the changes in permeability after burns, including prostaglandins, catecholamines, histamine, bradykinin, vasoactive amines, leukotrienes, and activated complement.[22] Mast cells in the burned skin release histamine in large quantities immediately after injury, causing a characteristic response in venules by increasing the space in intercellular junctions.[23,24] The use of antihistamines in the treatment of burn edema, however, has had limited success with the possible exception of H_2-receptor antagonists.[25] In addition, aggregated platelets release serotonin, which plays a major role in the formation of edema. This agent acts directly to increase pulmonary vascular resistance and indirectly aggravates the vasoconstrictive effects of various vasoactive amines. Serotonin blockade has been shown to improve cardiac index, decrease pulmonary artery pressure, and decrease oxygen consumption after burns.[26]

Another mediator likely to play a major role in vascular hyperpermeability and tone is thromboxane A_2. Levels of thromboxane increase dramatically in the plasma and wounds of burn patients.[27,28] This potent vasoconstrictor leads to platelet aggregation in the wound, contributing to expansion of the zone of stasis. Also, it causes prominent mesenteric vasoconstriction and decreased blood flow to the gut in animal models with compromised gut mucosal integrity and immune function.[29]

Patients with major burn injury have the highest metabolic rate of all critically ill or injured patients.[30] The metabolic response to a severe burn injury is characterized by a hyperdynamic cardiovascular response, increased energy expenditure, loss of lean body mass and body weight, accelerated breakdown of glycogen and protein, lipolysis, and immune depression.[31] This response is mediated by increases in circulating levels of catabolic hormones including catecholamines, cortisol, and glucagon.[32]

Pharmacologic agents have been used to attenuate catabolism and stimulate growth after burn injury. To further minimize erosion of lean body mass, administration of anabolic hormones such as growth hormone, insulin, oxandrolone, fenofibrate, and catecholamine antagonists such as propranolol has been studied. These agents contribute to maintenance of lean body mass and promote wound healing.[33-38]

Changes in Organ Function

Major burn injury affects multiple organs. Cardiac effects include marked loss of plasma volume, increased peripheral vascular resistance, and decreased cardiac output; pulmonary effects include a decrease in pulmonary static compliance, and these changes are associated with mild direct cardiac damage.[39-41] Renal blood flow decreases with a fall in glomerular filtration rate, which may result in renal dysfunction. Metabolic changes are characterized by an early depression followed by a marked sustained increase in resting energy expenditure as well as increased lipolysis, proteolysis, and oxygen consumption. This is driven, in part, by an increase in production of catecholamines, cortisol, and glucagon.[42] Increased peripheral lipolysis results in hepatic steatosis, and a fatty liver is caused by breakdown of triglycerol into free fatty acids. Like steatosis, peripheral lipolysis contributes to morbidity and mortality through fatty infiltration of various organs. Burn-related metabolic responses can last more than 2 years after burn.[21] There is a generalized impairment in host defenses, with depressed production of immunoglobulin, decreased opsonic activity, and depressed bactericidal activity.[43] This leads to an increased susceptibility to infections.

Initial Care

The patient should be removed from the source of the burn to stop the burning process, and clothing and jewelry taken off. The patient should be kept warm by wrapping in a clean sheet or blanket. Upon arrival to the emergency department, the immediate treatment of a burn patient should proceed as with any trauma patient to include the ABCDEs (airway, breathing, circulation, disability, exposure), and any potential life-threatening injuries should be identified and treated.

Airway and Breathing

Assessment starts with the airway, as upper airway obstruction can occur after burn injury. In most patients, there is a 4- to 6-hour period of time before postburn edema will compromise the airway. It is important make the decision for intubation in a relatively elective fashion. Oral tracheal intubation is preferred. It is important to place as large an endotracheal tube (ETT) as possible because burn patients frequently require pulmonary toilet via bronchoscopy, and smaller tubes can become occluded. It is not possible to perform bronchoscopy on an adult with an ETT smaller than a size 7, or a 4.5 in a child. Once placed, it is important to carefully secure the ETT to avoid dislodgement. Stridor, wheezing, tachypnea, and hoarseness indicate impending airway obstruction due to an inhalation injury or edema, and immediate treatment is required. Severe hypoxemia can result from flame burns, so oxygen should be administered and saturations closely monitored. Refractory hypoxemia or labored breathing also signal the need for intubation. In-line stabilization of the neck should be maintained if injury to the cervical spine is suspected.

Hypoxemia can also result from an elevated carbon monoxide (CO) following prolonged smoke exposure. Arterial blood gas and carboxyhemoglobin (COHb) levels should be obtained when inhalation injury is suspected. CO has an

affinity for hemoglobin that is over 200 times that of oxygen, and COHb can falsely elevate oxygen saturation levels determined colorimetrically. Therefore, use of a pulse oximeter may not be effective because patients with CO poisoning may show falsely normal oxygen saturation levels. Use of a pulse CO-oximeter enables measurement of absorption at several wavelengths to distinguish oxyhemoglobin from COHb saturation and determines blood oxygen saturation more reliably.[44] Levels of CO higher than 30% to 40% are indicative of severe exposure. The treatment for CO inhalation is 100% oxygen. This will decrease the half-life of CO from 4 to 6 hours at room air to 40 to 80 minutes with 100% oxygen. In three atmospheres absolute 100% oxygen in a hyperbaric chamber, the half-life decreases further to 15 to 30 minutes.

Full-thickness circumferential burns of the chest can interfere with ventilation, and bilateral expansion of the chest should be observed to document equal air movement. If the patient is ventilated, airway pressure and P_{CO_2} should be monitored. Rising airway pressure and P_{CO_2} indicates compromised ventilation, and a chest wall escharotomy should be performed. This is preferably performed at a burn center.

Managing the airway of an acute burn patient is one of the most difficult aspects of initial burn care. In general, intubation should strongly be considered for the following: elevated COHb associated with depressed mental status and/or persistent metabolic acidosis; burns greater than 40% TBSA, especially in children; burns with a perioral full-thickness facial component of more than 50%; full head burns (face and scalp); and clinically significant smoke inhalation. Relative indications for intubation include the following: change in voice or hoarseness, carbonaceous sputum, partial-thickness burns to the face, and extremes of age (<1 or >75 years of age). Neither chest x-ray findings nor the presence of singed nasal hairs is particularly helpful. Singed nasal hairs can occur with a flash burn or burn in an enclosed space. Bronchoscopic findings are relatively unhelpful if there is no history of inhalation injury and no spontaneously expectorated carbonaceous sputum.

Circulation

All patients with significant burns should have large-bore intravenous (IV) lines placed for resuscitation. If an IV cannot be placed in the field, an intraosseous line can be placed in the humeral head, proximal tibia, or sternum. If required, IVs can be placed through the burned skin for initial resuscitation. Because noninvasive measurement of blood pressure may be difficult in patients with burned extremities, there should be early consideration of an arterial line.

Disability and Exposure

Patients should undergo the standard trauma evaluation with complete exposure to allow all areas of skin to be examined. Burned areas should be cleansed with soap and warm water or Hibiclens solution if available, and then dressed with antibiotic cream (bacitracin or gentamycin ointments, silver sulfadiazine, or Sulfamylon) and wrapped in gauze or other clean dressing material. A Foley catheter should be placed for large (>10% TBSA in children or >20% TBSA in adults) burns or burns to the genitalia. During this process, it is important to avoid hypothermia. Resuscitation areas and IV fluids should be warmed, exposure minimized, and warm blankets and thermal coverings employed. Finally, tetanus status should be updated as necessary.

Determination of Burn Size

Accurate determination of burn size is critical to ensure treatment and resuscitation are performed properly. The most commonly used method of determining the burn size in adults is the "rule of nines" (Fig. 51-2). Each upper extremity and the head and neck are 9% of the TBSA. Each lower extremity, the anterior trunk, and posterior trunk are 18% each. Finally, the perineum and genitalia are 1% of the TBSA. Another method of estimating burn size is using the patient's open hand including the digits, which is approximately 1% TBSA. Many burn centers will use the Lund-Browder chart, which adjusts for differences based on age (Table 51-2). Regions should be marked for burn depth with the understanding that this may change with time. Children have a relatively larger portion of body surface area in the head and neck and a smaller surface area in the lower extremities. Infants have 21% of the TBSA in the head and neck and 14% in each leg, which incrementally approaches adult proportions with increasing age. These are estimates for the initial fluid rate. Subsequent adjustments are based on the patient's response to resuscitation.

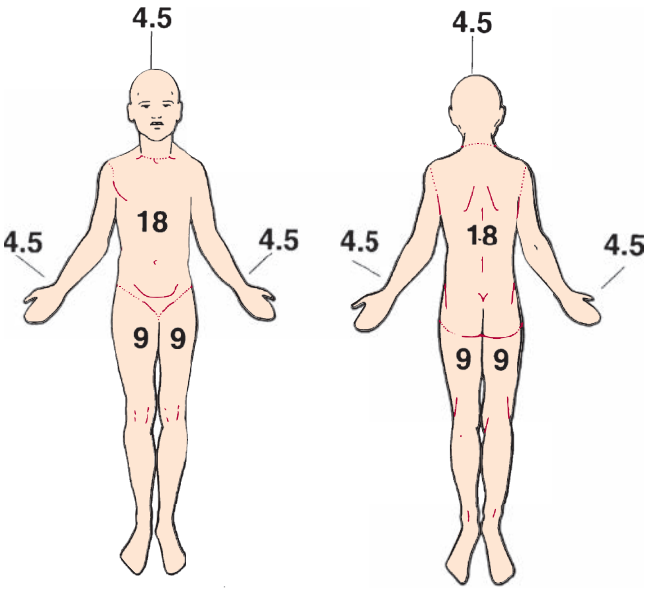

FIGURE 51-2 Determining burn size by the "rule of nines."

TABLE 51-2: Lund-Browder Chart for Estimation of Burn Size in Children

Area	1 y	1–4 y	5–9 y	10–14 y	15 y
Head	19	17	13	11	9
Neck	2	2	2	2	2
Anterior trunk	13	13	13	13	13
Posterior trunk	13	13	13	13	13
Buttock	2.5	2.5	2.5	2.5	2.5
Genitalia	1	1	1	1	1
Upper arm	4	4	4	4	4
Forearm	3	3	3	3	3
Hand	2.5	2.5	2.5	2.5	2.5
Thigh	5.5	6.5	8	8.5	9
Leg	5	5	5.5	6	6.5
Foot	3.5	3.5	3.5	3.5	3.5

FIGURE 51-3 Right hand showing superficial partial-thickness burn after the typically occurring blister has been removed. Superficial partial-thickness burn has a wet, pink, and moist appearance.

Determination of Burn Depth

Clinical manifestations of burn injury are determined by depth and breadth of tissue destruction. The two independent systems of describing the depth of burn injury include degree and thickness. Both are interchangeable.

SUPERFICIAL BURN (FIRST-DEGREE BURN)

Superficial burns do not penetrate the epidermis. Similar to a sunburn, the skin is warm, erythematous, and painful. It is also dry without blisters or eschar. The epithelium remains intact but may slough over time. These burns are self-limited and have no potential for scar. Treatment is primarily related to comfort.

PARTIAL-THICKNESS BURN (SECOND-DEGREE BURN)

Partial-thickness burns penetrate into but not through the dermis. These burns appear wet, weeping, and erythematous, and are exquisitely painful, with blisters or sloughing of the epidermal remnant. They are further divided into two categories based on the depth of dermal penetration.

Superficial Partial-Thickness Burn. These burns involve the epidermis and papillary dermis. They blanch to touch and often form blisters, which may not occur for some hours following injury (Fig. 51-3). Typical healing occurs in about 2 weeks with appropriate wound care, and they are low risk for scar and pigment changes. These can be managed conservatively with dressing changes or xenograft to facilitate healing.

Deep Partial-Thickness Burn. These burns involve the epidermis, papillary dermis, and reticular dermis. They are nonblanching, less painful, pink or pale, and require more than 3 weeks to heal (Fig. 51-4). These burns are frequently managed surgically because they are at high risk of hypertrophic scar and pigment change. Outcomes may be improved by early tangential excision and grafting.

FULL-THICKNESS BURN (THIRD-DEGREE BURN)

Full-thickness burns destroy the dermis and penetrate into the subcutaneous fat. These burns are dry, leathery, waxy, nonblanching, and insensate, and eschar is frequently shades of brown, white, gray, or black (Fig. 51-5) The transition from adjacent partial-thickness burn is clear by the lack of tissue edema. These burns will not heal alone without significant scarification and contracture, and warrant surgical excision with skin grafting or tissue transposition. Despite early excision and grafting, a large full-thickness burn is still at risk for sequelae such as contractures and hypertrophic scars.[45] It is now common practice to surgically debride third-degree burns within the first 3 to 5 days after burn to avoid

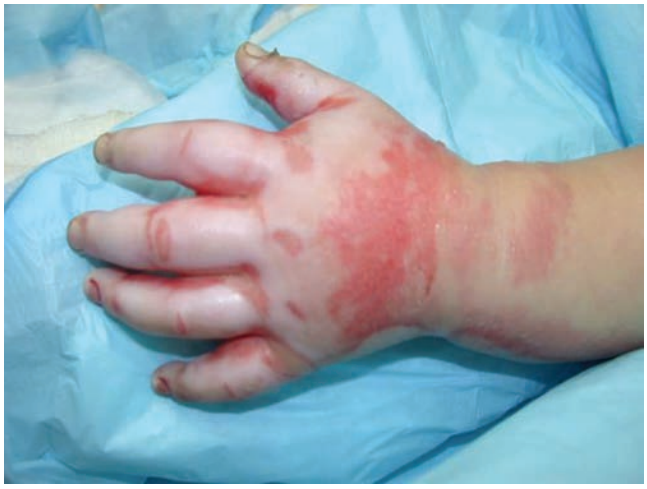

FIGURE 51-4 Left hand showing deep partial-thickness burn, typically with mottled pink or white areas.

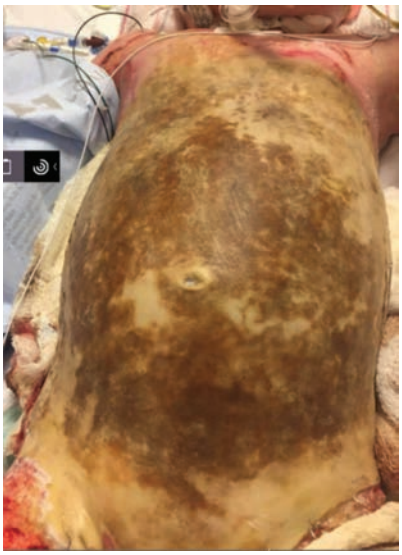

FIGURE 51-5 Third-degree burn of the abdominal wall. Full-thickness burns destroy the dermis and penetrate into the subcutaneous fat. These burns are dry, leathery, waxy, nonblanching, and insensate, and eschar is frequently shades of brown, white, gray, or black.

cellulitis and burn wound sepsis, especially in large burns. Fourth-degree burns refer to burns that extend into muscle, tendon, or bone. They have a charred appearance that usually results from high-voltage electrical injury or from prolonged duration of contact with fire or a heated object. Skin grafting alone is not adequate treatment for burns of this severity, and limb loss may occur.[46]

Burn Resuscitation

Burn resuscitation is essential for large burns, defined as greater than 10% TBSA in those younger than 5 or older than 50 years of age and 20% TBSA in those 5 to 50 years of age. Patients with smaller burns who can tolerate oral intake including oral rehydration do not require supplemental IV fluids.[47]

With larger burns, there is a systemic capillary leak that increases with burn size. Capillaries usually begin to regain competence after 18 to 24 hours if resuscitation has been successful. Lactated Ringer's is the fluid of choice in adults. Normal saline will predictably result in a severe hyperchloremic metabolic acidosis and should be avoided. Children less than 2 years of age should receive fluids containing 5% dextrose, such as 5% dextrose in half-normal saline, to prevent hypoglycemia. This can be accomplished by giving 5% dextrose as maintenance fluid and lactated Ringer's solution as resuscitation fluid.

Different formulas have been devised to assist the clinician in determining the proper amount of resuscitation fluid. Early work by Baxter[48] formed the basis for modern fluid resuscitation protocols. His work showed that edema fluid in burn wounds is isotonic and contains the same amount of protein as plasma. Additionally, the greatest loss of fluid is into the interstitial fluid compartment. Various volumes

TABLE 51-3: Resuscitation Formulas

Formula	Crystalloid volume	Colloid volume	Free water
Parkland	4 mL/kg/% TBSA burn	None	None
Brooke	1.5 mL/kg/% TBSA burn	0.5 mL/kg/% TBSA burn	2.0 L
Galveston (pediatric)	5000 mL/m^2 burned + 1500 mL/m^2 total	None	None

TBSA, total body surface area.

of intravascular fluid were tested in a canine burn model to determine the optimal delivered amount in terms of cardiac output and extracellular volume. These findings led to the Parkland formula in resuscitating burn patients (Table 51-3); Baxter also concluded that colloid solutions should not be used in the first 24 hours after burn until capillary permeability returns closer to normal. Others have argued that normal capillary permeability is restored somewhat earlier after a burn (8 hours), and thus colloids could be used earlier.[49] Some burn centers are now in fact using oncotic fluids such as albumin or plasma.[50,51]

The late colonels John A. Moncrief and Basil A. Pruitt, Jr. studied the hemodynamic effects of fluid resuscitation in burns, which resulted in the Brooke formula (Table 51-3). They showed that fluid loss in moderate burns results in an obligatory 20% decrease in both extracellular fluid and plasma volume during the first 24 hours after injury. In the second 24 hours, plasma volume returns to normal with the administration of colloid. Cardiac output is low on the first postburn day despite resuscitation but subsequently increases to supernormal levels as the flow phase of hypermetabolism is established.[52] Since these studies, it has been found that much of the fluid needs are due to capillary leak that permits passage of large molecules and water into the interstitial space. Intravascular volume follows the gradient into the burn wound and nonburned tissues. Approximately 50% of fluid resuscitation needs are sequestered in nonburned tissues in patients with 50% TBSA burns.[53]

Most burn centers use guidelines similar to the Parkland or Brooke formulas, with varying amounts of crystalloid and colloid solutions being administered for the first 24 hours after burn. These formulas are guidelines to the amount of fluid necessary to maintain adequate perfusion. All of them calculate the amount of volume given in the first 24 hours, with half being given in the first 8 hours and the other half being given over the next 16 hours.

In pediatric burns, the commonly used formulas are modified to account for changes in surface area–to–mass ratios. Compared to adults, children have a larger body surface area relative to their weight and generally have greater fluid needs during resuscitation. The Galveston formula for children based on body surface area uses 5000 mL/m^2 TBSA burned for resuscitation plus 1500 mL/m^2 TBSA for maintenance in the first 24 hours (Table 51-3). This formula accounts for

both the resuscitation fluid requirements and maintenance needs of children with burns.

An alternative resuscitation formula popularized in the military is the Rule of Ten.[54] Burn size is estimated to the nearest 10% TBSA. The initial fluid rate for patients who weigh 40 to 80 kg is %TBSA × 10. For patients who weigh more than 80 kg, the rate should be increased by 100 mL/h for every 10 kg.

The main indicator of the adequacy of resuscitation in burn patients remains the hourly urine output. In general, the goal for urine output in these patients should be 0.5 mL/kg/h in adults and 1 mL/kg/h in children. Fluid rates should be adjusted to match the desired urine output. If urine output is greater than the stated goal, the fluid rate can be adjusted down 20% per hour until urine output is within the goal range. To avoid oliguria, it is not recommended to decrease the fluid rate below the maintenance rate. If urine output is less than expected, fluid rates should be adjusted up by 20% per hour until urine output goals are met.[55] Patients at risk of low urine output due to heightened needs for resuscitation are those with deep burns, smoke inhalation, alcohol intoxication, or associated injuries, and children.[50] In addition to urine output, standard measures of resuscitation, such as lactate, base deficit, pulse pressure variation, and CVP, should be monitored. Crystalloid boluses should be avoided because these will only contribute to resuscitation morbidity and potentially lead to increased pulmonary edema or secondary extremity or abdominal compartment syndromes.[56-59]

Fluids should be decreased by one-third after the first 24 hours and can be divided between colloid and crystalloid. This will help to mitigate unnecessary interstitial edema.[60] Patients with chronic renal failure will clearly not meet the normal targeted urine output and may require some type of objective measure to guide resuscitation to avoid fluid overload.

Resuscitation morbidity is a consequence of overresuscitation. Excessive fluid administration can lead to abdominal compartment syndrome, which, in the burn patient, is associated with a greatly increased mortality, especially when a decompressive laparotomy is required.[61] Additional complications of fluid overload include extremity compartment syndrome, airway edema, and malperfusion of burn wounds. This constellation of complications associated with excessive fluid administration has been coined "fluid creep."[57]

Escharotomies

With circumferential constricting burns to an extremity, peripheral circulation to the limb can be compromised. Development of generalized edema beneath a nonyielding eschar impedes venous outflow and will have a tourniquet effect on the arterial inflow to the distal beds. This can be recognized by numbness and tingling in the limb and increased pain in the digits. Arterial flow can be assessed by pulse oximetry and determination of Doppler signals in the digital arteries and the palmar and plantar arches in affected extremities. Capillary refill should also be assessed. Escharotomies may be needed for patients with deep second-degree and third-degree burns that are circumferential around the chest, extremities, abdomen, penis, or neck.[62-64] In the extremity, the release of a burn eschar is performed by making lateral and medial incisions on the affected extremity using electrocautery. The entire constricting eschar must be incised to relieve the obstruction to blood flow. If the hand is involved, incisions should be made as they would for a fasciotomy. These include over the index and ring finger metacarpal and along the thenar and hypothenar eminence at the junction of the glaborous and nonglaborous skin. If needed in the fingers, the incisions should be placed at the border of the glaborous and nonglaborous skin. Chest wall escharotomies are made in the lateral chest bilaterally with a connecting incision across the chest to relieve constriction and allow adequate ventilation. Incision placement for commonly required escharotomies is shown in Fig. 51-6.

An overview of burn triage and initial care is presented in Fig. 51-7.

Inhalation Injury

Inhalation injury occurs in 8% of cutaneous burn injuries and remains a significant predictor of outcome following massive burns. There has been improved survival over the past few decades, which is attributed to early diagnosis, better airway management, gentle ventilation, and pulmonary hygiene.[65]

In most inhalation injuries, damage is caused primarily by inhaled toxins. Heat is generally dispersed in the upper airways, whereas cooled smoke particles and released toxins are carried distally into the bronchi and alveoli. The injury is principally chemical in mechanism, and the response is an immediate increase in blood flow in the bronchial arteries with formation of edema and increases in lung lymph flow. The resulting edema is associated with an increase in lung neutrophils, and it is postulated that these cells may be the primary mediators of pulmonary damage with this injury.[66] Neutrophils release proteases and reactive oxygen species that can produce conjugated dienes by lipid peroxidation. High concentrations of these conjugated dienes are present in the lymph and pulmonary tissues after inhalation injury, suggesting that increased neutrophils are active in producing cytotoxic materials.[67]

Another hallmark of inhalation injury is separation of the ciliated epithelial cells from the basement membrane followed by formation of exudate within the airways. The exudate consists of proteins found in the lung lymph and eventually coalesces to form fibrin casts.[68] Clinically, these fibrin casts can be difficult to clear with standard airway suction, and bronchoscopy is often required. These casts can also add barotrauma to localized areas of the lung by forming a "ball valve." During inspiration, the airway diameter increases, and air flows past the cast into the distal airways. During expiration, the airway diameter decreases, and the casts effectively occlude the airway, preventing the inhaled air from escaping. Increasing volume leads to localized increases in pressure that are associated with numerous complications, including pneumothorax and decreased lung compliance. Therapy is aimed at clearing the airway and minimizing complications from hyperinflation.

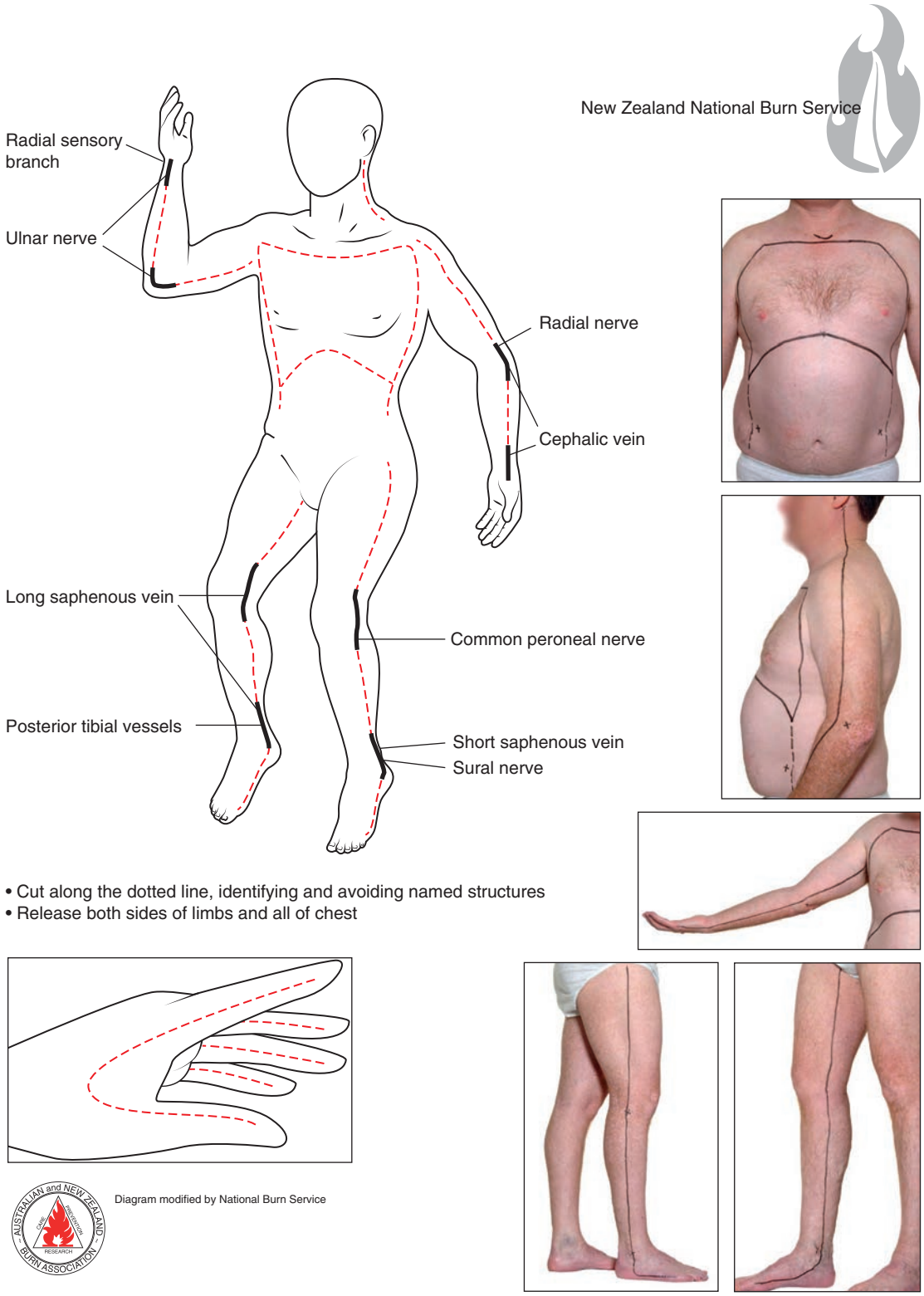

FIGURE 51-6 Typical escharotomy incisions. (Reproduced with permission from the Australian and New Zealand Burn Association.)

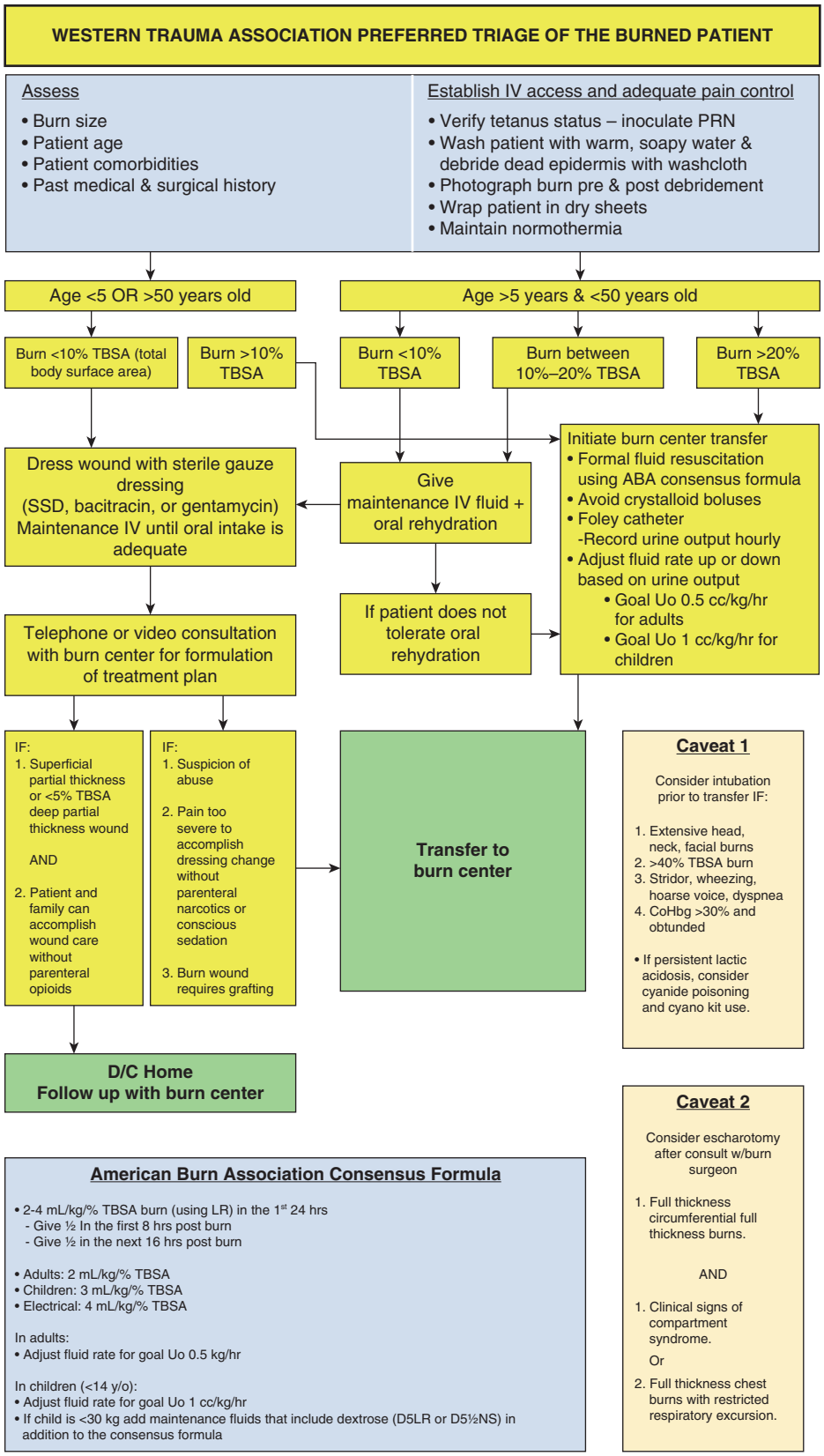

FIGURE 51-7 Algorithm for triage of the burned patient. This algorithm is an up-to-date tool to help care for and appropriately triage patients who arrive at a trauma center. ABA, American Burn Association; IV, intravenous; LR, lactated Ringer's; NS, normal saline; PRN, as needed; SSD, silver sulfadiazine; Uo, urine output. (Reproduced with the permission of the Western Trauma Association.)

Patients with smoke inhalation often present with a history of exposure to smoke in a closed space. Signs and symptoms include stridor, hoarseness, wheezing, carbonaceous sputum, facial burns, and singed nasal vibrissae. Each of these findings has poor sensitivity and specificity[69]; therefore, the diagnosis is often established with bronchoscopy. Bronchoscopy can reveal early inflammatory changes such as erythema, edema, ulceration, sloughing of mucosa, and prominent vasculature in addition to infraglottic soot.[70] Mechanical ventilation may be needed to maintain gas exchange, and repeated bronchoscopies often reveal continued ulceration of the airways with the formation of granulation tissue and exudate, inspissation of secretions, and edema.

Management of an inhalation injury is directed at maintaining open airways, clearing secretions, and maximizing gas exchange while the lungs heal. An awake patient with a strong cough and patent airway can clear secretions very effectively, and efforts should be made to treat patients without mechanical ventilation when possible. If respiratory failure is imminent, intubation should be instituted early, with frequent chest physiotherapy and suctioning as needed. Pharmacologic treatment of inhalation injury includes β-agonists, nebulized acetylcysteine and heparin, and nebulized racemic epinephrine.[71-73]

Alternative ventilator therapies such as high-frequency percussive ventilation have been used in patients who fail conventional ventilation strategies. High-frequency percussive ventilation minimizes barotrauma, enhances secretion clearance by loosening inspissated secretions, and recruits alveoli at lower airway pressures. It combines standard tidal volumes and respiratory rates (ventilator rates 6–20 breaths/min) with smaller high-frequency respirations (200–500 breaths/min); however, there are no data that this method is superior over basic lung-protective ventilation strategies.

Electrical Burns

The two kinds of electrical burns are arc and current. Arc burns are flash burns produced by heat generated by an electrical arc. These types of burns are much more common than current burns. There usually is no history of tetany in involved muscle groups. The neurologic exam of the affected extremity is normal. There is no entrance or exit wound and no deep tissue damage. Arc burns are treated the same as thermal burns.[74]

Current burns are caused by electricity passing through the body, leading to tissue destruction. They are characteristically deep and well circumscribed, with visible entrance and exit wounds that are typically on widely different areas of the body (eg, entrance at hand and exit at foot). There is usually a history of tetanus muscle contracture. The neurologic exam of the involved extremity is usually abnormal.[75] An acute neuropathy is common and may be permanent. Development of cataracts is also common after severe electrical current burns and may be delayed for months; therefore, close ophthalmologic follow-up is necessary. Patients with current burns are at risk for a compartment syndrome, which would require a fasciotomy, not escharotomy, to decompress the affected extremity. If myoglobin is present in the urine, a brisk urine output should be maintained. Current burns are debrided surgically much like necrotizing soft tissue infections. Most of the patients will need operative debridement within 48 hours of admission and will require multiple surgeries.

Chemical Burns

It is important to accurately identify the chemical, how the exposure occurred, and type and duration of exposure. Prompt treatment is imperative in minimizing tissue damage in chemical burns, and the area of the burn should be copiously irrigated with water. Care must be taken to direct the drainage of the irrigating solution away from unburned areas to limit the area of exposed skin. Contaminated clothing should be removed and disposed of, and care should be taken not to get any chemical on providers. If the chemical composition is known, monitoring the irrigated solution pH will give an indication of the effectiveness of the irrigation. Attempts at neutralization of either acidic or basic solutions can result in heat production and extend the injury. Generally, acids cause coagulative necrosis and are confined to the skin, while basic solutions cause liquefactive necrosis and extend further into tissues. After the chemical injury has been controlled, the remaining burn is treated in the same way as thermal injuries. Assessment of burn depth is often difficult but is typically deeper than it appears. For chemical exposures to the eyes, a visual acuity test and fluorescein dye test should be performed following irrigation. Ophthalmology should be consulted on any patient with a significant eye chemical exposure.

HYDROFLUORIC ACID

Hydrofluoric acid (HF) is used in chrome and rust cleaners and in industry for etching glass. It is corrosive, and the fluoride ion penetrates deeply into tissues to cause progressive tissue destruction.[76] Burns are usually seen when HF concentrations are greater than 20%. Even small amounts of HF swallowed, inhaled, or absorbed through the skin can cause systemic symptoms. The fluoride ion precipitates calcium and may cause systemic hypocalcemia; however, the classic physical signs of hypocalcemia do not usually occur. Instead, patients will have severe pain at the site of exposure (typically hands), and over time, if untreated, patients will develop ventricular arrhythmias that are resistant to pharmacologic therapy and tend to recur after cardioversion. The arrhythmias occur even after the abnormalities in calcium and magnesium have been corrected. It is thought that the fluoride ion is directly cardiotoxic.[77] In addition to undergoing copious irrigation of the burned area, the exposed skin should be treated with 2.5% calcium gluconate gel to provide pain relief and limit the spread of the fluoride ion. Magnesium and ionized calcium levels should be followed closely. Patients who present with acute HF burns that are obviously full thickness and are greater than 1% TBSA should be taken immediately to surgery to excise the wound and thus remove the toxin.

Inhaled HF causes severe pulmonary edema and destruction of lung parenchyma. Aerosolized calcium gluconate has been used with mixed results.

HF hand burns are typically seen after direct exposure to industrial-strength cleaning solutions. As for all HF burns, 2.5% calcium gluconate gel can be applied topically. For more severe burns (tissue blanching or bleb formation) not on the digits, 10% calcium gluconate solution can be injected into the subcutaneous tissues (0.5 mL/cm^2 of burned area). If HF digital burns progress to bleb formation or blanching, intra-arterial calcium infusion has been advocated. These burns will rarely be severe enough to need palmar or digital fasciotomies.[78]

WHITE PHOSPHORUS BURNS

White phosphorus is an incendiary agent used in fireworks and military ammunitions. It is found in certain bullets, mortars, rockets, improvised explosive devices, and bombs. White phosphorus burns as long as oxygen is available. It is highly lipolytic, which allows it to continue to penetrate into deep tissues producing systemic effects. If still burning, soft tissue may need rapid debridement until all visible white phosphorus has been removed. Wounds should be soaked with saline or water to avoid exposure to oxygen; then 2% copper sulfate solution should be applied if available. Calcium and phosphorus levels should be monitored as the exothermic reaction generated by burning white phosphorus releases phosphates and oxidates, which bind calcium. Sudden death from electrolyte abnormalities has been seen for burns greater than 10% TBSA.[79,80]

PHENOL BURNS

Phenol is a solvent used by industry that acts as a topical anesthetic, so that delayed recognition of the burn is possible. The burned area should be cleansed with polyethylene glycol (PEG 300 or 400), propylene glycol, or vegetable oil to remove the phenol, followed by application of a topical antibiotic such as silver sulfadiazine. Phenol can be rapidly absorbed through the skin, resulting in systemic toxicity such as renal failure, intravascular hemolysis, and hepatic dysfunction.[81,82]

Care of the Burn Wound

ANTIMICROBIAL AGENTS

Care of the burn wound is based on burn depth. In general, after initial debridement of the epidermis, topical antimicrobial dressings should be applied with the goal to limit bacterial overgrowth.

Silver sulfadiazine cream (Silvadene) (SSD) combines the agents sulfonamide and silver ion. It is the most common ointment used for initial wound care. It can also be used for care of donor sites.[83-85] SSD is applied only once a day and has fair eschar penetration. It should not be used on the face or ears. SSD has good gram-positive, gram-negative, and antifungal properties. It is painless but can cause leukopenia that is self-limited. Its use is contraindicated in patients with true sulfa allergies. The sulfonamide component of SSD can be absorbed through the skin. This is especially true in small children who have a greater area-to-mass ratio. In addition, sulfonamides can cause kernicterus in newborns. For these reasons, SSD is contraindicated in children under 2 months of age. Instead, gentamicin or bacitracin ointment should be used.

Mafenide acetate (Sulfamylon) is used as a 2% solution, penetrates eschar well, is painful when applied, and is rapidly absorbed. Sulfamylon has broad-spectrum antimicrobial qualities and is bactericidal. It has good gram-positive and gram-negative activity, including *Clostridium* species, but has little antifungal activity. Sulfamylon is a strong carbonic anhydrase inhibitor, causing loss of bicarbonate in the urine.[86] If used on large-percent TBSA burns, pH should be monitored, and often supplemental bicarbonate is needed. Sulfamylon is safe for use on ear burns and can prevent auricular chondritis.[87]

Silver nitrate solution (0.5%), although rarely used today, is safe for patients with sulfonamide allergies, does not penetrate eschar, and is minimally absorbed. Broad-spectrum antimicrobial resistance is uncommon. Disadvantages include the use of large bulky dressings to keep wounds moist, and black staining of all exposed surfaces. It can also cause hyponatremia and hypokalemia as well as methemoglobinemia.[88-90]

Bacitracin ointment is typically used on the face or in children where keeping dressings in place is a challenge. It is useful for superficial partial-thickness burns or clean donor sites.

Nystatin is a polyene antibiotic structurally similar to amphotericin B. It binds ergosterol in the fungus cell membrane. It has a broad-spectrum antifungal effect but little antibacterial effect. It is used with SSD in cases of heavy fungal colonization of the burn wound or in invasive fungal burn wound infections. It is not systemically absorbed, and resistance is rare.

BASIC BURN DRESSINGS

The application of topical antimicrobials is an effective method of decreasing skin colonization, but dressing changes are painful. Recently, silver nylon or foam dressings (Silverlon, Silverseal, or Mepilex Ag) are being used once the burn wounds have been determined to be clean, stable, and likely to heal. The main advantages of these types of dressings are ease of application, elimination of painful daily dressing changes, and documented antimicrobial effectiveness. In some cases, this will allow home care rather than inpatient burn wound care.

Surgical Management of the Burn Wound

Deep partial-thickness burns may not heal and full-thickness burns will not heal in a timely fashion without autografting. Early excision and skin grafting of these wounds are now standard. For wounds less than 20% TBSA, skin grafts are either unmeshed or meshed, with a narrow ratio (2:1 or less). The advantage of unmeshed is that there is less contraction. In major burns, widely expanded autograft skin (4:1 or greater ratio) is needed. Although widely meshed skin is applicable for most large burn wounds, cosmetically important areas such as the hands, face, and neck should be grafted with non-meshed sheet grafts to optimize appearance and function.

Cultured epidermal autograft is a process by which a patient's own keratinocytes are cultured in vitro and then used as a graft to cover wounds.[91] The potential for donor coverage is potentially unlimited, but the process is time consuming and expensive. This technique can be used as rescue in patients with very large burns and limited donor sites, and works best when applied over a 6:1 meshed autograft.[92] Prolonged drying time is required each day, adherence is as low as 15%, and the grafts are subject to significant degradation by mechanical forces as well as by colonizing bacteria.[93] Recently, the FDA has approved cultured epidermal autografts which can be sprayed over the excised burn wound within the same operation of the harvest. Like cultured epidermal autografts, outcomes are better if the cells are sprayed over widely meshed autograft.

Wounds that cannot be covered with meshed autograft skin due to a scarcity of available donor skin can be temporarily covered with an allograft. Porcine xenograft or human allograft (cadaveric) has the advantage of being readily available. These temporary dressings may also be used for coverage of partial-thickness wounds to reduce pain and maintain joint mobility while allowing the wound to heal. Dermal substitutes such as Integra (Integra LifeSciences Corporation, Plainsboro, NJ) and Alloderm (LifeCell Corporation, The Woodlands, TX) are also being used. They remain in place for approximately 2 weeks, after which time the outer silastic layer is removed and a thin allograft is placed over the underlying neodermis.

Nutritional Management of Burn Patients

As previously noted, profound hypermetabolism is a physiologic response unique to burn injuries. This hypermetabolism is characterized by increased body temperature, glycolysis, gluconeogenesis, proteolysis, lipolysis, and prolonged substrate cycling.[94,95] Overall, the basal level of glucose is elevated despite a high insulin state, which can complicate outcomes and exacerbate muscle catabolism. Lipolysis also occurs at a rate that is higher than normal due to abnormal substrate cycling. This results in an accumulation of triglycerides causing steatosis. There has been a report that propranolol will decrease this peripheral lipolysis.[96] Proteolysis is also increased in burn patients compared to nonburn patients. This is due to a combination of increased muscle breakdown and increased plasma protein production by the liver. As wound healing requires an increase in protein synthesis, early enteral nutrition with supplemental proteins is recommended. Protein requirements are typically from 2 to 2.5 g protein/kg/d, and calorie needs may be as high as 40 kcal/kg/d, although these will vary with metabolic demand, age, and percent TBSA. The use of anabolic agents, particularly oxandrolone, can reduce catabolism and improve muscle mass after burn injury.[97]

Special Problems Seen in Burn Patients

STRESS ULCERS

Since the near-universal use of histamine type 2 receptor blockers or proton pump inhibitors in burn patients, stress ulcers, or Curling ulcers, are now rarely seen. Some centers have advocated for conservative use of stress ulcer prophylaxis in small burns, but this has not been well studied.

BURNS OF THE EAR

The ear is unique in that it contains cartilage that is just under a thin layer of skin. Because cartilage has a poor blood supply, it is very susceptible to infection following burns. Sulfamylon penetrates into the cartilage better than other topical antibiotics and is recommended for use on ear burns. If auricular chondritis does develop, it should be promptly treated. Signs and symptoms include pain, tenderness, and erythema and an increase in the auriculocephalic angle such that the ear may protrude laterally. Chondritis usually occurs 2 to 6 weeks after the burn, often after the patient has been discharged from inpatient care. The most common organism seen in auricular chondritis is *Pseudomonas aeruginosa*, but gram-positive infections are possible, and cultures should always be sent. Treatment includes antibiotics, drainage of any abscesses, and debridement of necrotic cartilage. Infections can be challenging to eradicate, and they often recur.[98]

TAR BURNS

Tar or pitch blend is used in sealing roofs or repairing roadways. Burns are usually on the hands and forearms. Significant pain and loss of skin will result if the tar is simply peeled off of the skin. Petroleum ointment or other emulsifying agents will adhere to the tar, solubilize, and then dissolve the tar. Underlying burns are then treated with SSD or another topical antibiotic.[99,100]

Care of the Burn Patient in Austere Conditions

Caring for victims of burns in austere conditions such as the military environment or after natural disasters or other humanitarian crises can be challenging. Evacuation to a burn center or other treatment facility may not be feasible. The following are practical and safe alternatives to care in such settings. For additional information related to combat casualty care, see Chapter 53.

For patients who can be evacuated, care should be optimized prior to long flights or travel in suboptimal medical conditions.[101] Patients should undergo dressing changes each day prior to and including the day of transport. Prior to departure, the patient should be wrapped in clean sheets and blankets, with Bair Huggers, fluid warmers, and ancillary warming devices used when possible to help maintain normothermia during transport. Other than an escharotomy, operative burn care should not be undertaken prior to evacuation.

Burn patients who cannot be evacuated face additional challenges. Recent US military experience treating burn casualties abroad has led to clinical practice guidelines citing 40% to 45% TBSA as the largest survivable burn size. Resuscitation capabilities and blood are limited, dressing supplies

are precious, infection control is rudimentary, supplemental nutrition may not be available, and physicians, nurses, and physical therapists (if available) may have variable to no burn experience. When a burn patient arrives, burn evaluation, cleansing, and dressing should ideally be performed in the operating room. Field lines should be changed, and assessment of burn wound depth and extent should be performed as per normal care. Resuscitation is similar to civilian burns with a prescribed formula such as outlined in Table 51-3.[102] Smaller size burns may be amenable to primary excision and closure. Once larger burns have demarcated, excision and grafting of small portions of full-thickness burns can commence. This should be undertaken approximately every 3 days until all full-thickness sites are covered with graft and until all partial-thickness wounds are healing.

Reconstruction

As survival of patients with large surface area burns has increased due to acute burn care and improved critical care, surgeons care for an increased number of patients with secondary burn deformities. Hypertrophic scarring is a major complication after burn injury with several risk factors that contribute to its development including the following: location of the burn injury, burn depth, time to healing, and skin color.[103,104] Although the precise mechanism by which hypertrophic scarring occurs remains unclear, expression of transforming growth factor-β1 and its receptors has been associated with its development. Recent investigations have also identified the role of mechanotransduction through focal adhesion kinase as playing a central role.[105]

The key to improving hypertrophic scars is to relieve tension by either lengthening the scar through local tissue rearrangement or providing additional tissue through thick split-thickness grafts, full-thickness grafts, biologic substitutes, or free-tissue transfer. Z-plasty adds length to the scar at the expense of width and is a form of local tissue rearrangement to release linear burn scar contractures.[106,107] In addition to lengthening a scar, it is also useful in redirecting a scar, flattening a raised or depressed scar, and recreating a webspace.[45,46,108] The classic Z-plasty design incorporates angles of 60° with three equal limbs to achieve a theoretical 75% lengthening. This configuration balances the desired increase in length of the contracture by taking advantage of the laxity of lateral skin. The corner of the Z-plasty should be 90° to maximize blood flow and minimize the risk of skin necrosis, as shown in Fig. 51-8. Laser therapy can also be used to treat hypertrophic burn scars, including both pulsed dye lasers (PDLs) and fractional carbon dioxide lasers. PDLs increase vascularity and reduce redness, whereas carbon dioxide lasers improve pliability of the scar to permit increased range of motion.[109] Often, use of both PDLs and a fractional carbon dioxide laser is required.

Broad burn scar contractures will require a generous transverse release to restore structures to their normal anatomic positions for a proper correction. As a general rule, most burn scar contractures that cross a mobile structure, such as an eyelid or a joint, will require an incision across the entire axis of rotation and further than one might expect. Scar excision should rarely be contemplated as this will create an even larger defect, and the same forces will be present that led to the initial scar.

Autologous fat grafting involves transfer of fat from one area on the body to another. For burn reconstruction, it can aid in replacing tissue loss in a burned area. Fat is usually removed by liposuction, processed into liquid, and then injected into the scarred or grafted area. Fat grafting has shown promise to improve function and appearance. Also, flaps can be used to fill in a postburn tissue defect. They have the advantage of not having to rely on the blood supply of the recipient bed to survive. Flaps can also be used to cover exposed nongraftable structures such as tendon or bone.

Tissue expansion is a valuable tool for burn reconstruction when a large adjacent piece of skin is needed either as a full-thickness graft or flap. The basis for tissue expansion is the gradual placement of mechanical stress to the overlying skin. Complications of tissue expansion are common, especially in

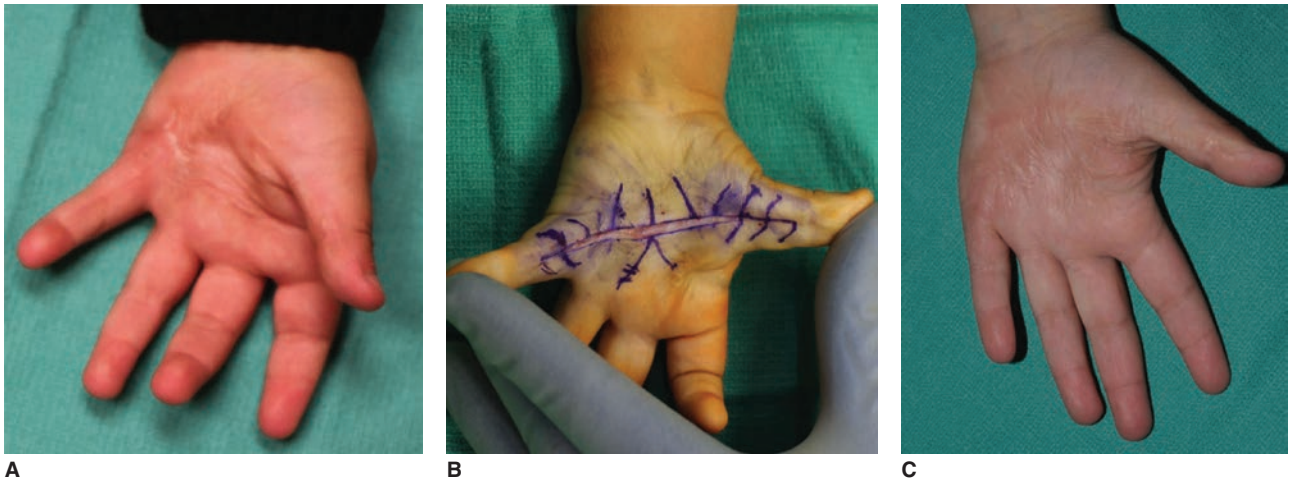

FIGURE 51-8 Series of Z-plasties with a central five-flap "jumping man" to relieve a palmar contracture. (**A**) Preoperative hand in extension. (**B**) Intraoperative markers. (**C**) Postoperative hand in extension.

the extremities, and include infection, implant exposure, and flap ischemia.

RADIATION

Therapeutic Radiation

Ionizing radiation therapy has become a central component of cancer treatment. In radiation-treated areas, close to 85% of patients will have a moderate to severe skin reaction, known as acute radiation dermatitis, that can lead to blistering, ulceration, and erosion.[110] There is a grading system developed by the National Cancer Institute to classify radiation dermatitis, and it includes four stages. Grade 1 injuries have faint erythema or dry desquamation. These are treated with hydrophilic moisturizers, while pruritus/irritation is treated with low-dose steroids. Grade 2 injuries have moderate to brisk erythema with patchy desquamation confined to creases and skin folds. Grade 3 injuries have moist desquamation outside skin folds with pitting edema and bleeding from minor trauma. Grades 2 and 3 injuries are treated with a hydrogel dressing and hydrocolloids with the goal of decreasing infection. Grade 4 injuries are characterized by full-thickness skin necrosis or ulceration with spontaneous bleeding. These wounds may be treated like full-thickness burn injuries and can require debridement with skin grafts or flaps.

Measuring Exposure to Radiation

Radiation consists of both particles (α, β, and neutrons) and photons (γ- and x-rays), which have specific energies and tissue penetrance (Table 51-4). The potential for biological injury for each of these depends on the amount of energy transmitted by the particle or photon when it interacts with the target. Each type of radiation has different qualities, and each is absorbed into tissue to varying degrees. Distance, time, and shielding are the ways to reduce exposure from a radiation source. Dosimetry is the measurement of radiation exposure by detectors that indicate the type, quality, flux, and rate of exposure dependent on the distance of the detector from the source.

Pathophysiology

Cell damage from radiation is caused by the transfer of kinetic energy from particles or photons to existing molecules, causing ionization of mostly oxygen and formation of free radicals such as the hydroxyl radical. These highly toxic compounds react with normal biologic molecules to cause cellular damage, primarily to phospholipid membranes and deoxyribonucleic acid.[111] Different cell types have different sensitivities to radiation. Cells with high proliferation rates are the most sensitive, while those with low proliferation rates are relatively resistant (Table 51-5).

The overall effect depends on the extent of cellular mass exposed, the duration of exposure, and the homogeneity of the radiation field. Radiation injuries are either localized or whole body, depending on the circumstances of the exposure. The term *localized radiation injury* refers to an injury involving a relatively small portion of the body that does not lead to systemic effects.[112] This is mostly associated with local exposure to low-energy radiation.

The dose of exposure determines injury severity. The onset of vomiting is useful for estimating the dose received from a single exposure. Vomiting in the first 10 minutes of the exposure means that the dosage was more than 8 Gray (unit of ionizing radiation dose, or Gy), vomiting within 10 to 30 minutes, 6 to 8 Gy; within 1 hour, 4 to 6 Gy; within 1 to 2 hours, 2 to 4 Gy; and over 2 hours, less than 2 Gy.[113] Aside from vomiting, erythema of the skin can be an initial sign of exposure, with the sooner the appearance of erythema, the higher the dose of exposure. Depilation, or hair loss, is another way to determine the extent of localized injury and may occur as early as 7 days after exposure.

TABLE 51-4: Types of Radiation, Relative Energies, Penetrance, and Relative Hazard

	Relative energy (MeV)	Penetrance into tissue (cm)	Relative hazard
α	1–5	0.0007–0.004	Low
β	0.2–1.0	0.017–0.34	Moderate
γ	0.1–10	20–150	High

TABLE 51-5: Radiosensitivity of Human Cell Types

Radiosensitivity	Cell types
Very high	Lymphocytes
	Hematopoietic cells
	Intestinal epithelium
	Spermatogonia, ovarian follicular cells
High	Urinary bladder epithelium
	Esophageal epithelium
Intermediate	Endothelium
	Fibroblasts
	Pulmonary epithelium
	Renal epithelium
	Hepatocytes
Low	Hematopoietic stem cells
	Myocytes
	Chondrocytes
	Neural cells

Acute Radiation Syndrome

Detonation of a nuclear device can result in enough radiation exposure to cause immediate death for individuals within the lethal area of the blast. The severity of injury from acute

TABLE 51-6: Four Phases of Acute Radiation Syndrome

Prodromal phase	Nausea, vomiting, fever
Latent phase	Symptom-free interval following acute nausea/vomiting
Manifest phase	Symptoms of hematopoietic, gastrointestinal, and neurologic injury
Recovery phase	Variable

radiation exposure is directly related to the effective dose of radiation to the whole body. Radiation exposure less than 1 Gy is associated with minimal symptoms and no mortality, but exposure to greater than 8 Gy has a 100% mortality.[114] The hematopoietic and gastrointestinal systems are particularly affected by radiation because they have rapidly dividing cells. Therefore, loss of stem cells and rapidly dividing cells from hematopoietic and gastrointestinal tissues can lead to bleeding, infection, and diarrhea.

Acute radiation syndrome has four phases of severity of signs and symptoms (Table 51-6). Onset of the prodromal phase is related to the total dose of radiation received. It can be minutes, if a lethal dose is received, to hours for lower doses. Fever, nausea, vomiting, and anorexia characterize this phase, which may last for 2 to 4 days or longer. When signs and symptoms regress, the latent phase starts. This symptom-free period may last a few hours to several weeks depending on the dose of radiation. As dose increases, the latent phase becomes shorter. Symptoms of hematopoietic, gastrointestinal, and neurologic syndromes are expressed in the third, or manifest, phase. Nausea, vomiting, diarrhea, and bleeding characterize this phase. Recovery is the last phase, is variable, and may last weeks to months. If the dose is high enough, death ensues.

Whole-Body Exposure

Effects of whole-body exposure depend on the dose of radiation absorbed by all tissues of the body. As opposed to local exposure to just the skin, whole-body exposure will lead to much more absorption of energy. Effects are primarily seen on the cardiovascular, hematopoietic, gastrointestinal, and central nervous systems. With relatively lower doses, bleeding, infection, and loss of electrolytes can occur from damage to the intestinal mucosa and blood cell components. Higher doses will cause cardiovascular collapse and circulatory failure.

The dose absorbed will lead to one of the three following courses:

1. *Hematopoietic syndrome*—Exposure to 1 to 4 Gy causes pancytopenia with an onset of 48 hours and a nadir at 30 days. Spontaneous bleeding can occur from thrombocytopenia, and opportunistic infections can occur from granulocytopenia.
2. *Gastrointestinal syndrome*—Exposure to 8 to 12 Gy will cause gastrointestinal symptoms in addition to pancytopenia. Severe nausea, vomiting, abdominal pain, and watery diarrhea occur within hours of the exposure. Once these symptoms resolve, the mucosa of the intestine sloughs and causes bloody diarrhea, loss of the intestinal barrier, and translocation of bacteria. Sepsis and massive fluid losses ensue with subsequent hypovolemia, acute renal failure, and death.
3. *Neurovascular syndrome*—Exposure to more than 15 Gy causes immediate total collapse of vascular tone that is superimposed on the preceding syndromes. This may be caused by the massive release of vasodilatory mediators or destruction of the endothelium, which progresses rapidly to shock and death.[115]

Assessment

Effective field triage, rapid evacuation of casualties, and appropriate decontamination are essential and should be performed as soon as possible.

During stabilization of the patient, information about the incident should be obtained, including the type of radiation, the duration of the exposure, the distance from the source, and whether direct contact with the source occurred. This information will be necessary to calculate the dose of radiation. Other important information is the background radiation level of the involved area at the time of the incident. The average radiation background in the United States is 360 mrem/y (rem = the quantity of any ionizing radiation that has the same biologic effectiveness as 1 rad of x-rays). Any radiation above the background level is considered contamination.

The individual radiation dose is assessed by determining the time to onset and severity of nausea and vomiting, rate of decline in absolute lymphocyte count, and appearance of chromosomal aberrations in peripheral lymphocytes. Documentation of clinical signs and symptoms affecting the hematopoietic, gastrointestinal, neurovascular, and cutaneous systems over time is essential for triage of victims, selection of therapy, and assignment of prognosis.

A complete blood count with differential should be performed with particular attention to the total lymphocyte count (TLC), which is the most accurate indication of radiation injury. The rate of decrease in TLC varies inversely with the dose of radiation and, therefore, portends the prognosis. Increases in serum amylase and diamine oxidase (specific to enterocytes) may be helpful in determining injury to the intestinal mucosa.

TRIAGE

Like other disaster situations, major radiation accidents and exposures can quickly overwhelm any response system. Treatment facilities may be destroyed, supply distribution may be hampered, and health care workers can be among the injured. For these reasons, triage with resources directed toward those likely to survive is paramount to limit casualties. Unfortunately, the extent of radiation injury may not be initially apparent.

Victims should be evacuated as quickly as possible to limit exposure. The patient's injuries, including burn or traumatic injury, should be treated, and then symptomatic treatment for the radiation illness should commence.

Thermal burns are likely to occur in combination with radiation injury, which can be a lethal combination. With the addition of a significant radiation injury, survival following burns is reduced and only those individuals with relatively small (<30% TBSA burns) burns may be expected to survive.[116] Under most circumstances, the immediate resuscitation and treatment of life-threatening injuries supersede treatment for radiation exposure, as the effects of radiation are relatively delayed.

DECONTAMINATION

The first priority in the treatment of radiation injury is the stabilization of the patient. Once the resuscitation has begun and the initial assessment is complete, decontamination should be started and occur before access to the treating facility. Removal of clothing and jewelry and irrigation or washing of the body is an effective way of removing any radioactive contamination. Open wounds should be covered with a clean dressing. These wounds are assumed to be contaminated with radioactive material and should be gently irrigated with copious amount of water, saline, or 3% sodium hydroxide solution. The irrigant should be rinsed to a safe drain with the goal of diluting any radioactive particles without spreading them to adjacent uncontaminated areas. Irrigation is continued until the area indicates a steady-state absence of radiation with a Geiger counter or reaches the background radiation level.

Attention should then be turned to the intact skin, where decontamination involves gentle scrubbing of the contaminated sites with a soft brush under a steady stream of water. Following this, povidone-iodine or hexachlorophene solution is applied. This should be repeated until the skin has a steady-state level of radiation. After two rounds of the preceding treatment, a diluted mixture of one part commercial bleach to 10 parts water can be used to remove further radioactive particles.

Management of Local Injuries

Local injuries to the skin evolve very slowly over time, and symptoms may not occur for days to weeks after the exposure. Mild erythema should be treated conservatively with dressings, if needed. The lesion may progress to ulceration or chronic radiodermatitis.

Management of Whole-Body Injuries

The management of whole-body irradiation is aimed primarily at symptoms of cellular loss. Except for removal of internal radiation, no treatment can interrupt the process. Antiemetics are given to provide symptomatic relief from nausea and vomiting. Resuscitation may be needed to maintain euvolemia because of volume losses.

Gastrointestinal bleeding, diarrhea, infection, anemia, and diffuse bleeding from pancytopenia may occur with varying degrees of severity. If the exposure is more than 2 Gy, a bone marrow transplant as a salvage maneuver may be considered.

Infections become problematic as these patients become profoundly immunosuppressed, and opportunistic organisms are often the cause. All blood products should be irradiated to prevent graft-versus-host disease. Transplants should be performed at the peak of immunosuppression, which is between 3 and 5 days after a high-dose exposure. The use of hemopoietic growth factors such as granulocyte colony-stimulating factor (G-CSF) and granulocyte-macrophage colony-stimulating factor (GM-CSF) can speed restoration of granulocytes.

SUMMARY

The treatment of burns and radiation injuries is complex. Knowledgeable physicians can treat minor injuries in the community; however, moderate and severe injuries require treatment in dedicated facilities with resources to maximize the outcomes from these often devastating injuries. The care of patients has markedly improved over the past 40 years, and most patients with massive injuries now survive. Challenges for the future will be in modulation of scar formation and in shortening the time to a functional and visually appealing outcome.

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52

Temperature-Related Syndromes

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KEY POINTS

- The standard definitions of hypothermia are mild (>32°C), moderate (28–32°C), and severe (<28°C).
- Warming techniques to reverse hypothermia include passive, active and noninvasive, and active and invasive.
- Depressed platelet function, impaired platelet delivery, slowed activation of coagulation enzymes, activation of protein C, and activation of fibrinolysis occur in hypothermic patients.
- Initial hospital management of frostbite should include rapid rewarming of the affected body part, in a 38°C to 40°C water bath.
- Surgical debridement and amputation should be delayed until demarcation has occurred after a frostbite injury, unless the patient develops wet gangrene, overwhelming infection, or a necrotizing soft tissue infection.
- The normal core body temperature is maintained at approximately 37°C by conduction, convection, evaporation, and radiation.
- As heat exhaustion progresses, the cutaneous blood vessels paradoxically vasoconstrict and sweating stops in many, but not all, patients.
- In patients with heat stroke, rapid cooling improves morbidity and mortality.
- Cellular changes, including protein denaturation, begin to take place at approximately 41.6°C to 42.3°C.
- Malignant hyperthermia is caused by exposure of susceptible individuals with a unique genetic composition to halogenated anesthetic agents.

INTRODUCTION

Humans as homeothermic mammals must maintain a stable internal body temperature within narrow range to allow function of enzymes. Regional and seasonal variations in the environment mandate the ability to either lose or generate heat in order to maintain temperature within this range. Mechanisms to either conserve heat (surface vasoconstriction, shivering, and piloerection) or lose heat (surface vasodilation, sweating with evaporation) allow adaptation to a colder or warmer environment. Three specific adaptive behaviors permit existence in a cold environment and have allowed the spread of humankind across six of the seven continents. These are fabrication of clothing, shelter-building, and control of fire. However, environmental extremes, abnormal endocrine function, or infection and injury can result in the inability of the organism to maintain body temperature within the normal range, leading to significant functional disturbances.

HYPOTHERMIA

It is important to understand the distinction between environmental hypothermia (due to exposure to cold) and hypothermia associated with injury. Hypothermia due to exposure can be lethal when it is severe and ongoing (consider Napoleon's army during the retreat from Moscow[1]), but with modern medical care, hypothermia without injury has a significant survival rate even in the setting of cardiac arrest (50%).[2] In patients after injury, however, the effects of hypothermia are profound, with hypothermia being an important component of the "bloody vicious cycle" first described by Kashuk and

TABLE 52-1: Hypothermia and Associated Physiologic Changes

	System					
Temperature	Cardiovascular	CNS	Renal	Coagulation/ hematologic	Respiratory	Other
35–36°C	Vasoconstriction Tachycardia ↑ Cardiac output	Shivering			↑ Resp rate	↑ Metabolic activity
32–34°C	Bradycardia ↓ Cardiac output	↓ ICP Abn EEG Confusion Lethargy	Cold diuresis	"Enzymatic" coagulopathy[a] Hemoconcentration (2% per 1°C decrease)	Bronchorrhea Inhibited ciliary function	↓ Metabolic activity
28–32°C	↑ Myocardial irritability "Osborne J" waves on ECG		Hypokalemia Hypomagnesemia	Thrombocytopenia (sequestration) Increased infectious risk	↓ Resp rate	Hyperglycemia Abn drug metabolism
≤28°C	Ventricular arrhythmias Cardiac arrest	Obtundation		Apnea		

[a]As a broad estimate, for every degree Celsius below 34°C, coagulation factor activity decreases by 10%.

Abn, abnormal; CNS, central nervous system; ECG, electrocardiogram; EEG, electroencephalogram; ICP, intracranial pressure; Resp, respiratory.

colleagues.[3] In one large historical series, there were no survivors of hypothermia and serious trauma if initial core body temperature was less than 32°C.[4] This distinction mandates a different approach for patients with hypothermia and injury.

Definitions

The normal core body temperature for humans is 37°C, with a circadian variation of approximately 0.5°C to 1°C.[5] The standard definition of hypothermia, developed for environmental hypothermia, describes mild hypothermia as a core body temperature above 32°C, moderate from 28°C to 32°C, and severe less than 28°C. The lowest reported temperature for an adult survivor of hypothermia is 13.7°C. In trauma patients with hypothermia, the scale is shifted due to significant changes in mortality, with temperatures of 34°C to 36°C defined as mild, 32°C to 34°C as moderate, and less than 32°C as severe.

Measurement of temperature should be performed using a reliable technique. The most readily available and accurate techniques include a bladder catheter with thermistor tip or an esophageal monitor. Rectal temperature measurements are accurate but not as responsive to changes in core body temperature as the previously noted techniques. Tympanic thermometers are readily available and noninvasive but are of limited use at subnormal body temperatures. Oral and axillary temperature measurements are not reliable in hypothermic patients and should not be used.

Environmental Hypothermia

Environmental hypothermia (also known as accidental hypothermia) occurs if a normal person is exposed to cool temperatures with inadequate clothing or shelter. The incidence of environmental hypothermia in both the United States and Europe is increasing with risk factors including advanced age, mental illness, male sex, and alcohol intoxication.[6,7] Unsurprisingly, hypothermia is highly associated with the winter season.[7] Cooling is accelerated in the presence of windy conditions, and submersion in cold water can result in heat loss of more than 30 times that of the same air temperature.[8] Although exceptional athletes can swim for hours in cold water, an unconditioned person may become unconscious within 30 minutes of immersion in 4°C water. Case reports and small series suggest improved survival of children with cold water immersion undergoing cardiopulmonary resuscitation,[9] with a major issue being anoxic brain injury.[10,11]

The physiology of hypothermia is significantly dependent on the variation from normal body temperature[12] (Table 52-1). Initially there is an intense cutaneous vasoconstriction to reduce heat loss, and the exposed skin can rapidly cool to the ambient temperature. The patient experiences thermogenic shivering, which markedly increases oxygen consumption and depletes glycogen stores.[13] As the temperature drops, metabolism progressively slows and shivering ceases. The patient becomes confused, lethargic, and cold to the touch. Urine production is profuse due to impaired production of antidiuretic hormone (ADH) and increased vascular tone. The patient exhibits bradycardia, electrocardiogram (ECG) changes,[14] hypotension, hypovolemia, and metabolic acidosis with elevated blood lactate. Agitation, irrational behavior, and combativeness are replaced by obtundation and finally coma. When cardiac arrest occurs, death is not immediate but is inevitable without medical intervention.

MANAGEMENT OF ENVIRONMENTAL HYPOTHERMIA

Warming Techniques

There are a variety of techniques available for prevention and treatment of hypothermia. These include both passive and active warming techniques (Table 52-2). Passive techniques, consisting of warmed blankets and environment, are more applicable for prevention of hypothermia and for treatment of patients with cold stress (temperature >35°C). Specifically, ambient operating room temperature has been shown to have no effect on patient core temperature during emergency surgery and may in fact contribute to surgeon distraction.[15,16] Active methods can be defined as either external or internal. One of the most common external methods in use is forced-air rewarming (Bair-Hugger, 3M, St Paul, MN, or similar). Current broadly used internal methods include use of arterial-venous rewarming through catheters placed in femoral artery and vein with the blood passed through a fluid warmer, or continuous venovenous rewarming (using a continuous hemofiltration dialysis unit). A more recently developed option is use of a femoral venous catheter designed for therapeutic hypothermia (Thermogard XP, Zoll Medical, Minneapolis, MN), which circulates warmed or cooled fluid through the catheter. For unstable patients or those who have developed cardiac arrest, extracorporeal membrane oxygenation (ECMO) and cardiopulmonary bypass (CPB) are appropriate options.

For the responsive patient with a core temperature above 35°C, passive rewarming measures and supportive care are adequate. For patients with mild hypothermia (32–35°C), normal mentation, and preserved vital signs, passive or active external warming techniques can be effectively used. Trauma patients with mild hypothermia should be transported to a trauma center if feasible and should receive active warming. Patients with hypothermia should receive warmed parenteral fluids only. In patients with moderate hypothermia, a Foley catheter with an integral temperature probe is optimal to monitor core temperature and measure urine output. Urine output will typically remain brisk during rewarming and is *not* an indicator of adequate intravascular volume. Subclavian or internal jugular catheter placement is avoided because the guide wire can trigger ventricular fibrillation of the cold myocardium. Rewarming can be performed using either active internal or external techniques. Patients with severe hypothermia require endotracheal and nasogastric tubes to protect the airway and prevent aspiration. Irrigation of the chest and abdominal cavities with sterile warmed saline may be an effective warming technique in severe hypothermia. If any perfusing rhythm can be detected, administer pressor agents but avoid chest compressions that may trigger intractable ventricular fibrillation. In patients showing signs of severe hypothermia in the field (eg, unconsciousness, minimal vital signs), consideration should be given for transport to a center with the ability to perform ECMO or CPB, as these are the most effective techniques for both support of the cardiopulmonary system and rewarming, with survival in small series of 50% to 100%.[17,18] Figure 52-1 presents an algorithm of the previously discussed management strategies, adapted from a number of society guidelines.[19-21]

TABLE 52-2: Warming Techniques and Estimated Rates of Rewarming

Warming technique	Rate of temperature increase
Passive	
Blankets, warm room	0–0.5°C/h
Active noninvasive	
Forced air rewarming	1–2.4°C/h
Resistive heating blankets	1–2.5°C/h
Circulating water blanket	1.5–2°C/h
Active invasive	
Cavity lavage	1–4°C/h
Intravascular venous (Thermogard XP Catheter)	3–5°C/h
Cardiopulmonary bypass	2°C/5 min under optimal circumstances

The classic adage for the hypothermic trauma patient is that the patient is not dead until they are warm and dead; however, there are a number of identified markers strongly correlated with death despite aggressive resuscitation. These include elevated serum potassium of more than 12 mEq/L, lethal traumatic injury, core temperature less than 10°C, unwitnessed cardiac arrest, or chest wall too stiff to permit cardiopulmonary resuscitation.[20,21]

There are a number of potential complications associated with rewarming. These include core temperature afterdrop, rewarming-associated hypotension, pulmonary edema, hypoglycemia, electrolyte imbalances, renal failure, disseminated intravascular coagulation (DIC), and rhabdomyolysis.[22] An expected major sequela of rewarming of patients with hypothermic cardiac arrest is potential for anoxic brain injury.[23] A mortality of 0% to 50% is reported.[17,18,22,23]

Patients with hypothermia routinely present with an extremity temperature more than 15°C lower than the core temperature, so that even in a warmed environment, the core temperature can continue to fall, a phenomenon referred to as *afterdrop*.[24] Afterdrop may be clinically relevant in the setting of a patient with borderline severe hypothermia. In this setting, a further drop in cardiac temperature may result in cardiac arrest. An afterdrop of up to 5°C has been reported.[25] For this reason, until rewarming is achieved, patients with moderate or severe hypothermia should not be mobilized to minimize return of cold peripheral blood to the central circulation.

Hypothermia in Trauma

Hypothermia is associated with increased risk of death after serious trauma. A classic study performed by Jurkovich et al[4]

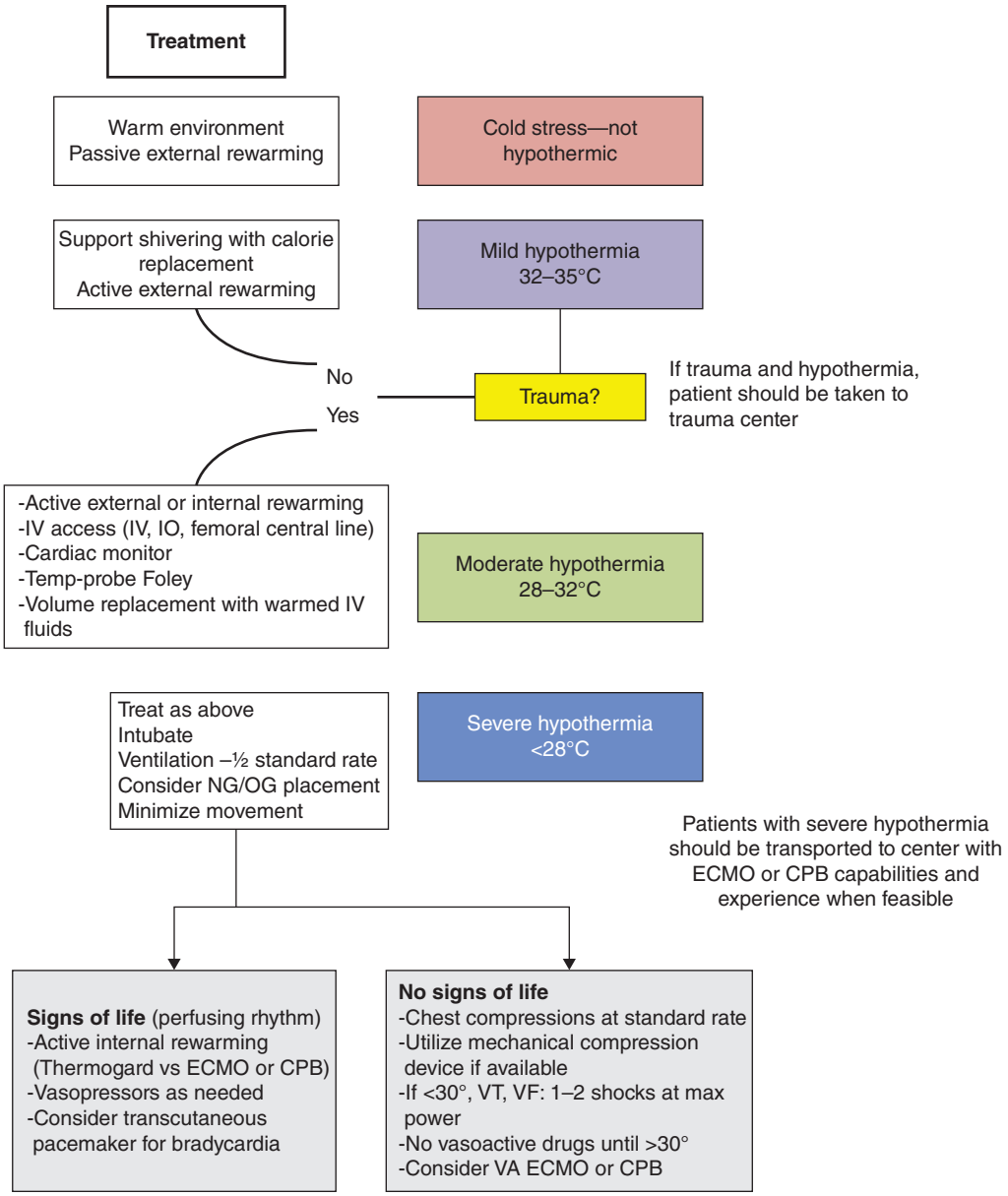

FIGURE 52-1 Algorithm for treatment of environmental hypothermia. CPB, cardiopulmonary bypass; ECMO, extracorporeal membrane oxygenation; IO, intraosseous; IV, intravenous; NG, nasogastric; OG, orogastric; VA, venoarterial; VF, ventricular fibrillation; VT, ventricular tachycardia.

in 1987 reported a 40% mortality in trauma patients with a core temperature less than 34°C, which increased to 100% in patients with a core temperature less than 32°C. Wang et al[26] reported data from analysis of 38,520 patients in the Pennsylvania Trauma Outcome Study, with an odds ratio for death of 3.03 associated with hypothermia. Similarly, postoperative hypothermia after cavitary surgery in severe trauma patients was also associated with increased mortality.[27] In an effort to determine whether or not hypothermia was a risk factor for death or a marker of complications, Shafi et al[28] evaluated nearly 40,000 patients in the National Trauma Data Bank and found that hypothermia was independently associated with increased mortality. In one large multicenter, prospective evaluation, hypothermia in severely injured trauma patients was found to be independently associated with multiple organ failure but not mortality.[29] In a randomized trial of active internal versus external rewarming techniques, Gentilello et al[30] noted improved early survival in patients receiving active internal rewarming; however, this early benefit did not lead to improved survival to discharge.

In contrast to these results in humans, there are a number of animal studies showing benefit to hypothermia in animal models of traumatic injury and shock.[31-33] The discrepancy between animal and human studies is initially puzzling but is

key to understanding the differences between accidental and traumatically induced hypothermia. The primary driver of hypothermia after injury is insufficient intrinsic heat generation due to mitochondrial anoxia during anaerobic metabolism, resulting in a drop of the organisms' core temperature toward that of the ambient environment. This is in distinction to induced hypothermia, which is sparing of adenosine triphosphate (ATP) levels, resulting in protection of the cellular environment during anaerobic conditions. For example, ATP levels are lower and remain depressed longer in hypothermic trauma patients compared to persons with hypothermia following elective surgery[34] or patients with therapeutic hypothermia induced under anesthesia.[35]

In the case of animal models, most involve induction of hypothermia over a short period of time, close to the time of injury/shock, and frequently in the setting of a standardized single-organ model, whereas in the human condition, hypothermia develops over a variable period of time, with the patient in shock, and with varied periods of transport and exposure to environmental cooling prior to arrival for definitive medical care. Until more evidence exists defining appropriate patient populations for therapeutic hypothermia, we recommend aggressively warming multiply injured patients to normothermic levels.

Hypothermia and Coagulation

Coagulation disorders are the most concerning physiologic abnormality in trauma patients, and hypothermia exerts profound effects on the coagulation system. It is difficult to separate the effects of hypothermia alone in the setting of the "bloody vicious cycle" of traumatic coagulopathy; however, the effects of hypothermia on the intrinsic and extrinsic coagulation systems, and their laboratory measures, have been extensively evaluated. Cosgriff et al[36] identified coagulopathy in massively transfused patients and noted in multivariate analysis that hypothermia was a significant risk factor, with an odds ratio of 8.7. These clinical results have been confirmed in a number of animal and human ex vivo studies,[37,38] demonstrating significant impairments of coagulation measurements with plasma or blood in cold conditions that resolve with warming of the plasma. In an in vitro study using viscoelastic testing to evaluate the individual roles of hypothermia, fibrinolysis, hemodilution, and acidosis in trauma-induced coagulopathy, hypothermia in the presence of fibrinolysis was found to have a significant effect on clot formation, clot strength, and lysis time.[38,39] Monitoring of coagulation parameters in the hypothermic patient is a challenge because decreasing temperature has been shown to have a significant inverse effect on prothrombin and activated partial thromboplastin time. This is relevant for clinicians, as coagulation tests are performed after warming of the plasma to 37°C, suggesting that these tests in hypothermic trauma patients are likely to underestimate the degree of coagulopathy. These results were confirmed clinically in a group of 112 seriously injured trauma patients in which core body temperature was correlated with coagulation studies.[40] It was noted that coagulation enzyme activity decreased below a threshold temperature of 34°C.

A host of additional mechanisms related to hypothermia have been implicated, including depressed platelet function, impaired platelet delivery, slowed activation of coagulation enzymes, activation of protein C, and activation of fibrinolysis.[41] Hypothermia plays a significant role in the coagulopathy of trauma and significantly complicates surgical management in these patients. In patients at high risk for coagulopathy, "damage control resuscitation" with component therapy to mimic whole blood should be used along with aggressive measures to rewarm the patient.[42] In patients requiring operative control of their injuries, damage control procedures should be used, with definitive correction delayed until the patient's coagulopathy, acidosis, and hypothermia have been corrected.[43]

THERAPEUTIC HYPOTHERMIA

There is increasing clinical evidence to support the use of therapeutic hypothermia (also known as targeted temperature management) in different clinical conditions. Hypothermia has a variety of physiologic effects that are potentially useful, including reduction in metabolic rate, inhibition of apoptosis, reduction of oxidative stress, inhibition of neuroexcitatory pathways, and decreased inflammation.[44] Since two landmark trials published in 2002 demonstrated a benefit in terms of neurologic outcomes after out-of-hospital cardiac arrest in patients treated with mild hypothermia,[45,46] there has been a growing literature suggesting potential benefit in a variety of other settings[47,48] (Table 52-3).

As a result of the two trials noted earlier, therapeutic hypothermia is included as a part of guidelines in the United States[49] and internationally for treatment of patients suffering out-of-hospital cardiac arrest with an initial cardiac rhythm of ventricular tachycardia or fibrillation. Data on its use in other

TABLE 52-3: Clinical Uses of Therapeutic Hypothermia and Type of Clinical Evidence

Clinical scenario	Efficacy	Evidence
Cardiac arrest	Effective	RCTs; cohort studies
Neonatal hypoxic ischemic encephalopathy	Effective	RCTs
Increased ICP	Effective	RCTs
Hypoxic encephalopathy after hanging	Feasible	Case series
Ischemic stroke, hemorrhagic stroke, SAH	Feasible	Small RCTs, case series
TBI	Unknown	RCTs with conflicting results
Traumatic exsanguination	Unknown	Case series

ICP, intracranial pressure; RCT, randomized controlled trial; SAH, subarachnoid hemorrhage; TBI, traumatic brain injury.

settings of cardiac arrest (eg, arrest with pulseless electrical activity, asystole, in-hospital arrest[50]) are less compelling, but it is the practice of these authors to consider therapeutic hypothermia in most cases of successful initial resuscitation after cardiac arrest. Cooling can be initiated in this setting with rapid infusion of 1 to 2 L of intravenous cold saline while a decision about whether or not to undergo cardiac catheterization is made and central and arterial lines are placed. Cooling can be sustained with either active external cooling devices (Arctic Sun, Medivance, Louisville, KY, and others) or an intravascular cooling device inserted via the femoral vein (Thermogard XP, Zoll Medical, Minneapolis, MN). Current recommendations include cooling the patient to 32°C to 36°C for 24 hours with an 8-hour rewarming period, followed by another 48 to 72 hours of normothermia. Reaching the targeted temperature within 6 hours of the arrest has been associated with a favorable outcome. A large randomized trial demonstrated equivalent outcomes for targeted temperature of 34°C and 36°C.[51]

Therapeutic hypothermia has also been studied extensively in the setting of traumatic brain injury with elevated intracranial pressures (ICPs). A meta-analysis from 2012 highlights the effectiveness of decreasing ICP in this patient population.[52] In this meta-analysis, which included 18 studies and 1773 patients, there was a decrease in ICP with therapeutic hypothermia in all studies, with an average reported decrease in ICP of approximately 11 mm Hg. Unfortunately, although this intervention results in a significant decrease in ICP, a subsequent randomized multicenter trial failed to show improved outcomes using the Extended Glasgow Outcome Scale in patients treated with hypothermia.[53] A recent systematic review found no difference in mortality or poor outcome in patients with traumatic brain injury treated with hypothermia, echoing the variable outcomes of available literature.[54] In a multicenter randomized controlled trial, Hifumi et al[55] found that fever control with a moderate temperature maintenance at 35.5°C to 37°C, compared with hypothermia therapy, resulted in improved traumatic brain injury–related mortality in patients with Abbreviated Injury Scale 3 and 4 head injury. Although the effect on outcome remains unclear, these results support the importance of fever control and suggest that therapeutic hypothermia should only be considered in patients with refractory intracranial hypertension unresponsive to other therapy.

FROSTBITE

Frostbite occurs when tissues cool to the point of freezing for a sustained period. At about 24°C, ice crystals form in the intracellular or extracellular fluid, depending on the rate of cooling. The incidence of frostbite is increasing with a more mobile society partaking of a growing number of outdoor sports. The hands and feet account for 90% of reported injuries, and this injury occurs most commonly in adults between the ages of 30 and 49 years.[56] Frostbite is classified by grade based on severity at the time of exam[57] (Table 52-4).

TABLE 52-4: Classification of Frostbite Injury

Grade	Symptoms and findings
First degree	Numbness, erythema White or yellow raised plaque without blister
Second degree	Superficial skin vesiculation Clear or milky fluid in blisters Initial lesion on distal phalanx
Third degree	Deeper hemorrhagic blisters Initial lesion on proximal phalanx
Fourth degree	Extends into subcutaneous tissue Initial lesion on carpal/tarsal

Initial hospital management of frostbite should include rapid rewarming of the affected body part, most optimally in a 38°C to 40°C water bath to quickly restore blood flow to tissues as the metabolic rate increases. This is painful and will frequently require parenteral narcotics. Ibuprofen should be administered at a suggested dose of 12 mg/kg/d twice daily to a total daily dose of 2400 mg to provide inhibition of prostaglandins and thromboxanes. Blisters should not be debrided, although some authors suggest aspirating nonhemorrhagic blisters with a fine needle.

In patients with frostbite at risk for tissue loss (grade 3 or 4 injury), thrombolytic therapy and intravenous prostacyclin therapy may be considered. This therapy should be administered in a setting that includes intensive monitoring. A recent report of angiography and thrombolytic therapy included angiography in 69 patients over 14 years.[58] Angiography was performed within 24 hours of rewarming, and catheter-directed lytic therapy was used for intra-arterial thrombi (n = 472 digits). Of 198 digits with restored blood flow, 4 required amputation. In the digits with partial or no improvement, amputation rates were 37% and 69%, respectively. Another trial randomized 47 patients to aspirin plus either buflomedil (a vasoactive agent used to treat claudication), intravenous prostacyclin, or prostacyclin plus systemic lytic therapy (recombinant tissue plasminogen activator [r-tPA]).[59] The risk of amputation was 60% in the buflomedil group, 0% in the prostacyclin group, and 16% in the prostacyclin plus lytic therapy. Either intravenous iloprost or thrombolysis with r-tPA (or both) should be considered for use in patients with severe frostbite and high risk of tissue loss. These interventions should be administered within a 24-hour window of injury if possible and should be administered in a facility with appropriate monitoring.

Surgical debridement and amputation should be delayed until demarcation has occurred unless the patient develops wet gangrene, overwhelming infection, or spreading necrotizing infection. The majority of amputations can be performed 1.5 to 3 months after the initial injury. Long-term sequelae of frostbite include severe arthritis and joint pain secondary to loss of articular cartilage associated with prolonged severe cold sensitivity or even Raynaud phenomenon.

Cold Contact or Flash Freeze Injuries

Exposure to cold or pressurized gases (eg, carbon dioxide, oxygen, nitrogen, or propane) or contact with a cold object can produce an irreversible freeze injury.[60] Plantar injury occurs when walking on ice or snow with unprotected feet. Huge ice crystals form both extracellularly and intracellularly, immediately rupturing the affected cells. The injury may have the appearance of a thermal burn and can affect any body part because escaping gas can pass through layers of clothing. The wounds are treated with topical antimicrobials, and skin grafts are applied if the wounds granulate. Lytic agents have no benefit and are contraindicated in these patients.

Nonfreezing Cold Injuries

Rapid cooling of the exposed skin causes severe pain and pale skin color called frostnip, which has little clinical significance. Immediate rewarming prevents tissue freezing, but the tissue remains sensitive to changes in temperature. Repeated episodes of nearly freezing, especially in wet conditions, cause chilblains (also known as pernio), with intense vasospasm and damage to local nerves. Subsequent cold exposure triggers marked pain with features of Raynaud syndrome, which increases the risk of a secondary freezing injury. Treatment is symptomatic.[61]

SYSTEMIC HYPERTHERMIA

The normal human core body temperature is maintained at approximately 37°C by conduction, convection, evaporation, and radiation. Skin temperature is approximately 35°C, thus facilitating heat dissipation down this temperature gradient. Conduction and convection allow the body to gain or lose heat in response to the respective external climate. Radiation causes heat transfer to or from the body by electromagnetic waves.[62] Evaporation, the body's most effective cooling mechanism, causes a reduction in skin temperature of 0.58 kcal/mL of sweat.[63] Hyperthermia results when the internal core temperature is elevated above normal ranges and the body's compensatory methods are ineffective, impaired, or overwhelmed by intrinsic or extrinsic heat loads.

Primary Hyperthermia

Susceptibility to primary hyperthermia depends on both patient and environmental factors. Exercise increases heat production, which can exceed the cooling capacity of the body. Additionally, climate variability has been shown to impact the incidence of heat-related illness.[64] Increasing humidity decreases the body's most effective defense mechanism of sweating. Hyperthermia is common in military training or combat and strenuous contact or endurance sports. Patients with significant cardiovascular comorbidities, diabetes, dysfunctional compensatory mechanisms, or altered behavior are at higher risk of heat-related illness as well. External heat causing hyperthermia presents on a continuum

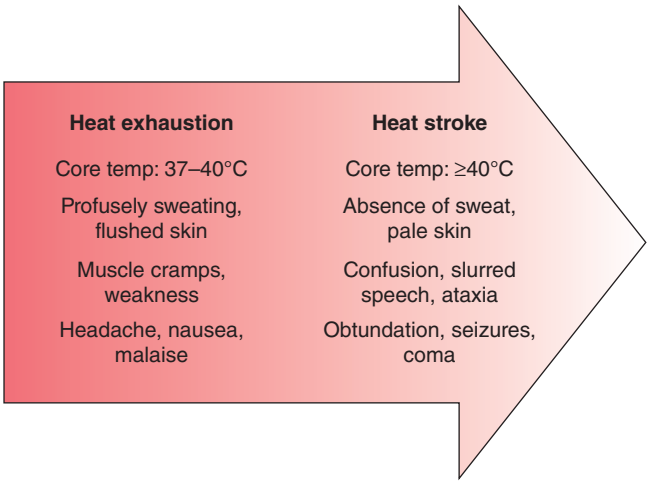

FIGURE 52-2 Continuum of severity of heat-related illness.

of severity (Fig. 52-2). Systemic hyperthermia begins as heat stress and progresses to heat exhaustion as fluid losses mount and core temperature increases. The patient is flushed, sweating profusely, and feels acutely ill. The patient may experience intense thirst, weakness, muscle cramps, nausea, and vomiting. Syncope may occur from functional orthostasis due to hypovolemia from sweating and peripheral shunting of intravascular fluid from vasodilation of skin vessels. Symptoms persist until the core temperature is lowered and fluids and electrolytes are replaced.[65]

As heat exhaustion progresses, the cutaneous blood vessels paradoxically vasoconstrict, and sweating stops in many, but not all, patients.[66] This more severe presentation of heat stroke is less commonly seen, albeit more deadly. An exact definition of heat stroke is difficult to pinpoint due to the range of severity and symptoms that may present; however, a generally accepted definition is a core temperature of at least 40°C and presence of central nervous system symptoms.[62,67] It is traditionally conceptualized as two distinct types. Exertional heat stroke is associated with generally healthy patients undergoing strenuous exercise. Classic heat stroke is typically a more passive process occurring in patients at extremes of age or in individuals with impaired mechanisms to escape or compensate to exposure to excess heat. The clinical picture can be mistaken for a cerebrovascular accident because the patient can be pale, cool to the touch, disoriented, paretic, or comatose. Prolonged, extreme elevation of core temperature may produce irreversible central nervous system injury.

Heat stroke accounts for 2% of sudden deaths among competitive athletes[68] and occurs in epidemic form during heat waves.[60] Two primary goals of treatment are cooling and cardiovascular support. Studies have shown that rapid cooling improves morbidity and mortality of patients.[70,71] Immersion in an ice bath is optimal, however, this may not be available or not be possible for patients with significant comorbidities requiring invasive monitoring. Additionally, patients may demonstrate variable levels of consciousness and combativeness and are prone to vomiting and diarrhea from

splanchnic hypoperfusion; therefore, immersion in water may prove difficult or dangerous to patient and staff in some cases. Removal of clothing (to facilitate sweat evaporation) and exposure to any cold object are essential, particularly near large vessels such as in the neck, groin, and axilla. Nearly all patients are hypovolemic from fluids lost through sweating; thus, this should be followed by fluid and electrolyte replacement. Resuscitation with normal saline is generally indicated due to hyponatremia from excessive sweating, which is often compounded by the patient's intake of large volumes of hypotonic solutions during early rehydration efforts. Laboratory workup may include assessment of electrolytes, liver and renal function, creatinine kinase, and/or coagulation factors. Further biochemical analysis or radiographic workup may be prudent in patients with significant comorbidities or suspicion for other concomitant causes of hyperthermia.

The downstream pathologic changes resulting from excess heat can have multiorgan system effects. Cellular changes, including protein denaturation, begin to take place at approximately 41.6°C to 42°C.[72] The brain and liver are thought to be the most vulnerable to hyperthermia; however, multisystem organ effects are seen due to hypoperfusion from centrally mediated shunting of blood to the skin for heat exchange, as well as the cellular effects of the excess heat.[62] Direct endothelial damage, cytokine release, and cell death contribute to the cytotoxicity and systemic inflammatory response that may be seen depending on the level of severity. At advanced stages of hyperthermia, activation of the coagulation cascade with progression to DIC has been seen. The mechanism is thought to be multifactorial, including direct heat damage to the endothelium, heat-mediated platelet aggregation, and fibrin deposition in arterioles.[72-74] Extremely high core temperature may additionally cause rhabdomyolysis and myoglobinuria, associated with compartment syndrome in affected muscles. Survival in such cases is rare.

Additionally, drugs such as cocaine, amphetamines, and 3,4-methylenedioxy-methamphetamine (MDMA or ecstasy) may cause or exacerbate hyperthermia. This is important in trauma treatment because hyperthermia has been shown to increase the risk of death after cocaine administration. The mechanism behind cocaine-induced hyperthermia is incompletely understood. This multifactorial process is likely a combination of a hypermetabolic state resulting in elevated heat production, disordered behavioral adjustment due to altered heat perception, and impaired mechanisms of heat dissipation through diminished sweating and cutaneous vasodilation.[75]

Iatrogenic Hyperthermia

Malignant hyperthermia (MH) is caused by exposure of susceptible individuals with a unique genetic composition to halogenated anesthetic agents. The unfettered release of calcium from the sarcoplasmic reticulum results in abnormal activation of skeletal muscle and increased metabolic rate. There is substantial variation in the reported prevalence of MH susceptibility. It has been estimated to be as high as 1 in 3000, although a recent epidemiologic study in New York state from 2001 to 2005 estimated a prevalence of 1:100,00[76] and even lower in subsequent years.[77] The heat released causes a precipitous spike in the core temperature that can be rapidly lethal. Patients present with increase in end-tidal carbon dioxide, increased oxygen consumption, tachycardia, muscle rigidity, and respiratory and metabolic acidosis.

The treatment follows several steps. The anesthetic agent must first be stopped, followed by ventilation with pure oxygen to drive off the anesthetic, treat acidosis, and reverse anaerobic metabolism. Occlusive drapes should be removed and active cooling initiated. Infusion of dantrolene, a hydantoid derivative muscle relaxant, is recommended to suppress the abnormal sarcoplasmic reticulum calcium release of MH, although the exact binding site of action is incompletely understood.[78] It may also be necessary to administer glucose to replace the depleted glycogen reserves. Patients with the genetic abnormality can safely receive general anesthesia with alternative anesthetic agents, nitrous oxide, and nondepolarizing muscle relaxants.[79]

Malignant neuroleptic syndrome (MNS) is a rare event in patients receiving antidepressant medications that block dopamine. It has been reported in trauma patients treated with haloperidol for agitation.[80] It is also caused by atypical antipsychotic agents or sudden withdrawal of dopamine agonists such as levodopa. The risk is increased with lithium administration, dehydration, or low serum iron levels. The patient presents with high fever and intense muscle rigidity; lesser features include depressed mentation or coma, tremors, autonomic dysfunction, or dysphagia. The illness is easily mistaken for meningitis, MH, or drug-induced delirium. Laboratory tests are not diagnostic, although creatinine kinase levels are elevated. The treatment is rapid cooling and rehydration, discontinuation of dopamine-blocking agents or restoring dopamine agonists, and supportive measures in the intensive care unit. The reported mortality is 10%.[81]

Serotonin syndrome is caused by an excess of intrasynaptic serotonin in different areas of the brain and can be caused by several mechanisms. Three features predominate in this condition, including altered mental status, hyperactivity of the autonomic nervous system, and abnormal neuromuscular excitation. Patients present with rapid onset of delirium, agitation, hyperthermia, diffuse sweating, flushing, tachycardia, hyperreflexia, tremor, shivering, and muscle rigidity.[82] Medications that may precipitate serotonin syndrome include serotonin reuptake inhibitors, tricyclic and atypical antidepressants, and monoamine oxidase (MAO) inhibitors.[83] The trauma surgeon encounters the syndrome in association with the use of cocaine, ecstasy, amphetamines, tramadol, or linezolid. Fentanyl has also been implicated in two case reports of serotonin toxicity, although controversy remains as to the risk associated with this drug interaction.[82] Nausea and diarrhea indicate a mild illness, but delirium, hyperthermia, and autonomic instability can be life threatening. These patients exhibit agitation rather than the stupor of MNS and require benzodiazepines for sedation, combined with cessation of the causative agent and supportive measures.

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53

Modern Combat Casualty Care

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KEY POINTS

- The Joint Trauma System (JTS) has been identified by Congress in the National Defense Authorization Act for Fiscal Year 2017 as the reference body for trauma care in the Department of Defense.
- *Died of wounds* is the term used for mortality of casualties who arrive at a medical treatment facility alive and succumb to their injuries.
- The case fatality rate historically has trended with percent killed in action.
- The transition from gunshot wounds to blast injuries such as from the improvised explosive devices used in Iraq and Afghanistan has been in evolution since the US Civil War.
- Almost 90% of combat casualties who succumb to their wounds do so before they arrive at a medical treatment facility.
- Combat units that have trained all of their unit members, not just medics, in tactical combat casualty care have achieved the lowest incidence of preventable death on the battlefield in the history of modern warfare.
- In military roles of care, role 2 is the first level of care that has surgical capability, such as the forward surgical teams used by the US Army in the recent conflicts.
- A Critical Care Air Transport Team refers to the augmented aeromedical evacuation-type platform with specific capabilities for intensive care unit care.
- For transfusion of whole blood to military casualties, only group O whole blood donors with low titers of anti-A and anti-B antibodies are used.
- Kaolin-based dressings like Combat Gauze work by accelerating clotting pathways.

INTRODUCTION

He who wishes to be a surgeon must first go to war.
Hippocrates (460–377 BC)

How varied was our experience of the battlefield and how fertile the blood of warriors in rearing good surgeons.
Thomas Clifford Allbutt (1836–1925)

The military surgeon is faced with the challenge of preservation of life in an environment of destruction where loss of life is far too commonplace. Historically war comes at the cost of human life; battlefield medics deploy to this environment to alleviate pain and suffering, care for the wounded, preserve the fighting strength, and save lives. Medicine and surgery have seen great advances during times of large casualty volumes; the Mayo brothers are credited with the quote: "The only victor in war is medicine." The current conflicts in the Middle East have spanned over two decades and represent the longest conflicts in US history. During this period, there have been significant advances in prehospital care, hemostasis, resuscitation, evacuation, damage control surgery, and transfusion strategies. Vice Admiral Alasdair Walker, Surgeon General of the British Armed Forces, described a concept now referred to as the "Walker Dip" during the 2013 Military Health System Research Symposium. The Walker Dip describes the recurrent historical cycle where medical care improves during conflicts, the lessons are forgotten after, and then are relearned again during the next war. Unfortunately, the Walker Dip did not spare the current generation, and lessons from previous conflicts were relearned, such as the use of whole blood and providing blood far forward. It is the duty of our generation to learn from past experiences and build on the current knowledge so that the lessons learned (and relearned) are not forgotten but rather codified into future education and training in order to decrease preventable death from trauma.

Regardless of the nation, era, or location, combat casualty care has unique attributes that distinguish it from civilian trauma care. It tends to occur in austere, resource-limited

environments under hostile conditions. The structures, equipment, supplies, and personnel are often required to be both mobile and few. Patients frequently are moved by helicopter or plane to receive definitive care. Care on the battlefield may require tactical decisions to take precedence over clinical care depending on the nature of the hostilities. By nature, ongoing hostile gunfire may threaten treatment activities for patient and provider alike. In addition, the mechanisms of injury in combat trauma are mostly penetrating and blast associated.

Proximity to surgical care drives much of modern battlespace planning. Warfighters are often in remote locations in today's unconventional dispersed battlefield based on counterinsurgency operations. The Golden Hour mandate started in 2009 demonstrated that there was a decrease in mortality with decreased time to capability (hemorrhage control, transfusions, surgical intervention).[1] With the purpose of decreasing evacuation times and bringing capabilities closer to casualties, surgical care has been pushed further forward with a smaller footprint (facilities and personnel) to support remote tactical operations. Figure 53-1 is typical of this type of unit, in this case a forward surgical team (FST). In recent years, the FST is often divided into two teams in order to provide split operations at two different locations. The three service branches have also developed smaller and more mobile surgical teams for performing resuscitation and damage control surgery in even more remote and austere conditions. Damage control resuscitation and surgery is implemented with the goal of stabilization and transport to the next higher levels of care. A chain of medical treatment is established from the point of injury to the final medical treatment facility (MTF) in the United States. Currently this chain of treatment and transport evacuation is typically completed within 72 hours.

Over the course of this chapter, the current status of combat casualty care will be discussed. The battlefield is a dynamic environment, and clinical care evolves rapidly based on continuous performance improvement implemented by the Department of Defense (DoD) Joint Trauma System (JTS). The JTS has been identified by Congress in the National Defense Authorization Act (NDAA) for fiscal year 2017 section 707 as the reference body for trauma care in the DoD; the most updated battlefield clinical practice guidelines can be found on the JTS website (https://jts.amedd.army.mil). Unique aspects of military care will be emphasized as well as their translation to civilian trauma care. Special attention is devoted to recent discoveries and newly implemented practices. There continues to be a close bidirectional relationship between military and civilian care for the injured patient, each constantly informing the other with expertise and innovation.

Benchmarking and Progress

The ability to assess combat casualty care outcomes over time is necessary for continuous performance improvement and, when trended over time, provides a perspective and method

FIGURE 53-1 Medical evacuation (MEDEVAC) helicopter loading (top left), air drop of supplies (top right), the operating room tent (bottom left), and medical personnel of the 67th Forward Surgical Team (Airborne) (bottom right); FOB Todd/Bala Murghab, Afghanistan.

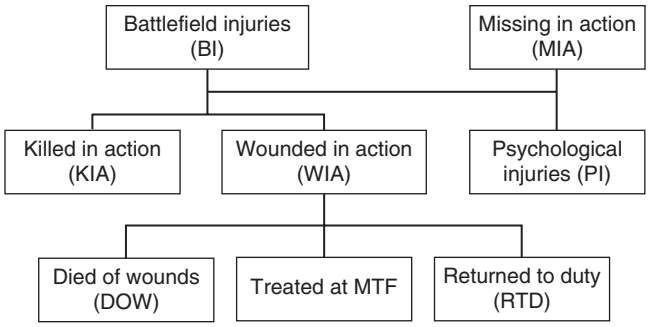

FIGURE 53-2 Classification of battlefield injuries. MTF, military treatment facility.

for comparison. Objective outcome assessment is foundational for evidenced-based care and requires standardization. The principal combat casualty care statistics that summarize the dynamic interplay between battlefield lethality and trauma care delivery are the case fatality rate (CFR), the percent died of wounds (DOW), and the percent killed in action (KIA) (Fig. 53-2).[2] Each summary statistic pivots on multiple dynamic factors such as prehospital care, transfusion capability, transport timing, protective gear and vehicles, surgical teams, and enemy tactics.

Killed in Action

Those with battlefield injuries who do not survive arrival to a military MTF are classified as KIA. Factors that directly influence the KIA rate are weapon lethality, prehospital care, and transport time to surgical capability. Devastating and sophisticated weaponry increases the KIA rate, while better prehospital care and shorter transport times decrease it. Efforts to decrease the KIA rate include body and vehicle armor, field care advances, and decreased evacuation times.

Died of Wounds

The DOW rate is a measure of mortality for casualties that arrive to the MTF alive and succumb to their injuries. It considers those wounded who die after reaching an MTF and has the potential to broadly, without unit or facility granularity, assess MTF effectiveness. Since the percent DOW (%DOW) formula relates those who succumb to their wounds to all the wounded, a surfeit of minimally injured patients decreases the %DOW. Key to its use as a comparative measure is the inclusion or exclusion of patients who are minimally wounded and return to duty. There is a reciprocity between the KIA rate and %DOW in regard to the quality and rapidity of prehospital care resulting in more patients with fatal injuries surviving to arrive at the MTF. Recent analysis of the %DOW demonstrates that it has not decreased since the Vietnam conflict, and unlike the other combat casualty care statistics, %DOW did not have a downward trend over the last decade in Iraq.

% Killed in Action (KIA)

$$\%\ \text{KIA} = \frac{\text{KIA} + \text{DOW}}{\text{KIA} + (\text{WIA} - \text{RTD})} \times 100$$

Died of Wounds (DOW)

$$\%\ \text{DOW} = \frac{\text{Died of wounds after reaching MTF}}{(\text{WIA} - \text{RTD})} \times 100$$

Case Fatality Rate (CFR)

$$\text{CFR} = \frac{\text{KIA} + \text{DOW}}{\text{KIA} + \text{WIA}} \times 100$$

FIGURE 53-3 Calculations for some of the common measures of combat casualty. MTF, military treatment facility; RTD, returned to duty; WIA, wounded in action.

Case Fatality Rate

The CFR is an overall summary statistic. The CFR provides a proportional measure of battlefield lethality and is the most comprehensive of the combat casualty statistics because it provides a summary measure of overall battlefield lethality as well as trauma care capability and delivery. Deaths from both KIA and DOW are included. The denominator includes all those injured on the battlefield—KIAs and wounded in action. It is expressed alongside the other equations in Fig. 53-3. When used to compare scenarios with similar battlefield characteristics, it can provide additional insights into the aggregate effectiveness of pre-MTF and post-MTF arrival casualty care.

The CFR historically has trended with the percent KIA (%KIA). CFR and %KIA, although valuable measures, are inherently limited in their capacity to provide accurate and granular assessment of MTF-level trauma care delivery. During the recent conflicts in the Middle East, the significant decrease in the CFR and %KIA likely reflect both clinical (point of injury care, en route care capabilities, and transport times) and tactical (improvised explosive device [IED] detection and defeat technology, body armor, safer vehicles such as mine-resistant ambush-protected [MRAPs] vehicles) improvements. Although the decreases in the CFR and %KIA have been attributed to improvements in battlefield medical care, this is certainly true for prehospital hemorrhage control but does endorse improved MTF or surgical battlefield care. Finally, although all statistic measures pivot based on the complex and multifactorial nature of the combat environment, the CFR is a composite statistic and can change significantly based on medical and many nonmedical factors.

Research comparing the current operations with prior conflicts has been limited due to variation in amount of data, how those data are recorded, and inconsistency of terminology. Recent comparisons of DOW rate to the DOW rate in Vietnam demonstrate that it has likely increased, especially when patients with minor injuries who return to duty are excluded from the calculations; additionally, the %DOW increased from 2003 to 2014.[3] It is not known exactly what this analysis means (shorter transport times bring sicker

patients to the MTFs vs changes in surgical care capability); further investigation and ultimately real-time unit performance improvement will help discern the discrepancies in the DOW rate. The civilian methods for benchmarking performance improvement and capturing data for analysis on a large scale are usually registry based and performed at local and regional levels (eg, the National Trauma Data Bank [NTDB] and the Trauma Quality Improvement Program). The DoD Trauma Registry (DoDTR) was created with these civilian models in mind. Since 2004, all comprehensive, standardized data are collected from the point of injury onward and maintained by the JTS.

PREVENTION AND PREPARATION

Injury Patterns and Injury Epidemiology

The IED has become the characteristic weapon of the recent conflicts in Iraq and Afghanistan. The IED and the countermeasures to mitigate its effects have shaped the distribution of injuries sustained. This transition from gunshot wounds to blast injuries has been in evolution since the US Civil War.[4] Other explosive devices, beside IEDs and gunshot wounds, still remain a significant source of morbidity and mortality.

IEDs are common in the recent and ongoing conflicts in the Middle East as well as in terrorist events in the United States and abroad. Increased adoption of their use has been driven by a combination of available materials, resources, and an understanding of how to maximize their destructive power. While their forms vary widely, they all share four essential components denoted by the acronym PIES: **p**ower source, **i**nitiator, **e**xplosive, and **s**witch (Table 53-1).[5] Adjuncts, such as formed charges or fragments, are often added. All items required for construction are readily available, making their creation easy and prevention problematic.

TABLE 53-1: Components of a Standard Improvised Explosive Device

Component	Examples
Power source	Battery, match
Initiator	Blasting cap, fuse
Explosive	Conventional artillery, "homemade" explosives, chemical
Switch	Cell phone, alarm clock, timers, circuits

A common utilization of IEDs has been in the form of roadside bombs. As the recent conflicts progressed, efforts were made to decrease the high number of injuries sustained in high-mobility multipurpose wheeled vehicles. This spurred the development of the MRAP vehicle with characteristic armor and a V-shaped hull to deflect blast energy (Fig. 53-4). Additionally, this protective vehicle has resulted in an epidemiologic shift of injury patterns, in that more closed fractures of the extremities and spine are seen in patients exposed to blasts who are in MRAP vehicles.

As coalition troops became less susceptible to roadside bombs with the advent of improved vehicle armor, the use of IEDs shifted. "Dismounted" IEDs became more common, targeting soldiers on foot patrols. IEDs have also been used via a variety of delivery devices, including vehicle-borne IEDs and suicide bombers. This interplay between development of weapons and the evolution of defensive mechanisms is observed in conflicts throughout history.

The torso is particularly vulnerable to penetrating projectiles such as IED-created fragments or firearm rounds. In response, modern body armor provides protection to this area (Fig. 53-5). The effectiveness of body armor used by the

FIGURE 53-4 Mine-resistant ambush-protected (MRAP) vehicles.

FIGURE 53-5 Example of improved outer tactical vest (IOTV) body armor.

US military has changed the distribution of combat injuries. Areas without protection, such as the extremities, are the more common sites of injury. The increased use of explosive devices by insurgent forces described earlier has also evolved in response to weaknesses in armor coverage. A recent review of 5 years of injuries in Iraq and Afghanistan revealed the presence of extremity injuries in 51.9% of combat casualties, and the vast majority of wounds (74.4%) were due to explosive devices.[6]

Education and Resources

CLINICAL PRACTICE GUIDELINES

As initial surgical interventions for combat casualties occur over a wide variety of dispersed locations, the challenge of standardization and education increases. As a response, JTS has created a series of clinical practice guidelines (CPGs). These guidelines are modeled after civilian guidelines published by several of the national trauma associations. As of this writing, there are over 40 evidence-based CPGs created by subject matter experts on a wide variety subjects (Table 53-2). The CPGs are updated routinely based on the available literature. The complete list can be easily found on the JTS website and is available as a portable device application.[7]

TABLE 53-2: List of Joint Trauma System Clinical Practice Guidelines Available in Multiple Formats from the US Army Institute of Surgical Research[7]

Acoustic trauma and hearing loss
Acute respiratory failure
Amputation
Battle/nonbattle injury documentation resuscitation record
Blunt abdominal trauma
Burn care
Catastrophic care
Cervical spine evaluation
Cervical and thoracolumbar spine injury
Clinical management of military working dogs
Compartment syndrome and fasciotomy
Damage control resuscitation
Concussion management algorithm cards
Department of Defense policy guidance for management of mild traumatic brain injury/concussion in the deployed setting
Emergent resuscitative thoracotomy
Fresh whole blood transfusion
Frozen blood
High bilateral amputations
Hypothermia prevention
Infection control
Inhalation injury and toxic industrial chemicals
Initial care of ocular and adnexal injuries
Intratheater transfer and transport
Invasive fungal infection in war wounds
Management of pain anxiety and delirium
Management of patients with severe head injury
Management of war wounds
Neurosurgical management
Nutrition
Pelvic fracture care
Postsplenectomy vaccination
Prehospital care
Prevention of deep venous thrombosis
Radiology
Resuscitative endovascular balloon occlusion of the aorta for hemorrhagic shock
Trauma airway management
Trauma anesthesia
Unexploded ordnance management
Urologic trauma management
Use of electronic documentation
Use of magnetic resonance imaging in management of mild traumatic brain injury in the deployed setting
Ventilator-associated pneumonia
Wartime vascular injury

INITIAL TREATMENT

Tactical Combat Casualty Care

When US military personnel are wounded on the battlefield, the prehospital phase of care is critical, in that almost 90% of combat casualties who succumb to their wounds do so before they arrive at an MTF with a surgical capability. This highlights the importance of the battlefield trauma care provided by combat medics, corpsmen, and pararescuemen (PJs). In addition, the care provided by other nonmedical unit members or the casualties themselves may be essential to survival.

FIGURE 53-6 Tactical combat casualty care logo. (Reproduced with permission of the Committee on Tactical Combat Casualty Care [CoTCCC]. Copyright © CoTCCC. All rights reserved.)

If the prehospital care provided to the casualty enables him or her to survive long enough to reach the care of a surgeon, the likelihood is very high that he or she will survive. Combat units that have trained all of their unit members, not just medics, in tactical combat casualty care (TCCC) have achieved the lowest incidence of preventable death on the battlefield in the history of modern warfare.[8]

TCCC was developed initially by a research project begun by the Naval Special Warfare Command in 1992 and continued by the US Special Operations Command. During this period, battlefield trauma care was comprehensively reviewed, and updated recommendations for care were subsequently published in *Military Medicine* in 1996.[9] The TCCC guidelines are continually reviewed and updated as needed by the Committee on TCCC (CoTCCC; Fig. 53-6). Trauma surgeons, emergency medicine physicians, combat medics, PJs, and corpsmen with combat deployment experience from all branches of the US military participate in the CoTCCC. The TCCC for Medical Personnel course is the only battlefield trauma care training course to have been endorsed by the US military, the American College of Surgeons Committee on Trauma, and the National Association of Emergency Medical Technicians. The CoTCCC is now one of the three trauma care committees in the JTS. The wide adoption of TCCC (including many international countries) has contributed to a remarkable decrease in prehospital mortality in recent combat operations.[8,10-12] TCCC makes battlefield trauma care recommendations in a tactical context, with basic management plans outlined for Care Under Fire, Tactical Field Care, and Tactical Evacuation Care, but caveated by noting that combat medical personnel may have to modify these recommendations as dictated by the tactical considerations of the specific casualty scenario.[9,13]

STAGES OF TACTICAL COMBAT CASUALTY CARE: CARE UNDER FIRE

In the Care Under Fire phase of care, TCCC recommends that the following actions be taken: (1) Return fire and take cover. (2) Direct or expect the casualty to remain engaged as a combatant if able. (3) Direct casualty to move to cover and apply self-aid if able. (4) Try to keep the casualty from sustaining additional wounds. (5) Casualties should be extricated from burning vehicles or buildings and moved to places of relative safety; do what is necessary to stop the burning process. (6) Stop life-threatening external hemorrhage if tactically feasible. Direct casualty to control hemorrhage by self-aid if able. Use a CoTCCC-recommended limb tourniquet for hemorrhage that is anatomically amenable. Apply the limb tourniquet over the uniform clearly proximal to the bleeding site(s). If the site of the life-threatening bleeding is not readily apparent, place the tourniquet "high and tight" (as proximal as possible) on the injured limb and move the casualty to cover. (7) Airway management is generally best deferred until the Tactical Field Care phase.

Stages of Tactical Combat Casualty Care: Tactical Field Care[8-12,14,15]

When the injured warfighter is no longer under effective hostile fire or has been moved to a location that provides cover, combat medical personnel can provide the additional interventions recommended for Tactical Field Care after a security perimeter has been established by the unit. These interventions include, in part:

- Disarming casualties with an altered state of consciousness
- Immediate control of massive external hemorrhage using limb tourniquets (Fig. 53-7), hemostatic dressings, junctional tourniquets, and/or XStat as required
- Allowing casualties who have direct maxillofacial trauma, but who are conscious and able to protect their airway by sitting up, leaning forward, and self-clearing the airway to do so

FIGURE 53-7 Combat application tourniquet.

- Protecting the airway in casualties who are unconscious but who have no direct airway trauma, through use of a nasopharyngeal or extraglottic airway
- Performing a surgical airway and inserting a CricKey in casualties when less invasive airway interventions are not sufficient to protect the airway
- The aggressive use of needle thoracostomy with a 10- or 14-gauge, 3.25-inch needle to treat suspected tension pneumothorax
- Applying vented chest seals for casualties with open pneumothoraces
- Reevaluation of external bleeding sites and the use of additional measures to control continued bleeding as needed
- Applying pelvic binders for casualties suspected of having sustained pelvic fractures
- Attempted conversion of limb tourniquets to other methods of hemorrhage control if the tourniquet has been in place for 2 hours
- Obtaining intravenous (IV) access only when that is required for medications or fluid resuscitation
- The use of intraosseous access when vascular access is needed but peripheral venous access is difficult to obtain
- Early administration of tranexamic acid (before fluid resuscitation) for casualties who are in, or judged to be at significant risk of, hemorrhagic shock
- Performing fluid resuscitation with whole blood or with red blood cells (RBCs) and plasma in a 1:1 ratio
- Faster, safer, and more effective relief of the pain of combat wounds through the use of the TCCC "Triple Option" analgesia plan than was achieved using the previous battlefield standard of intramuscular (IM) morphine; the Triple Option analgesia approach includes the use of ketamine for casualties in severe pain but with hemodynamic or pulmonary compromise and/or oral transmucosal fentanyl citrate lozenges for casualties who are in severe pain but who are otherwise stable
- IV or IM ondansetron in casualties with nausea and vomiting as a result of opioid use or the trauma itself
- Prevention of hypothermia with its attendant coagulopathy through use of the Hypothermia Prevention and Management Kit or other measures
- Administration of moxifloxacin or ertapenem to reduce morbidity from wound infections
- Bilateral needle decompression of both sides of the chest should the casualty suffer a traumatic cardiac arrest
- Documentation of care using the DoD Form 1380 (TCCC Casualty Card) and the electronic TCCC Medical After-Action Report[8]

Stages of Tactical Combat Casualty Care: Tactical Evacuation Care

Eventually, the casualty will be transported from the point of injury to a higher level of care using aeromedical, ground, or maritime mobility platforms. Evacuation should provide the opportunity to have additional and possibly more highly trained medical personnel as well as medical equipment such as electronic monitoring of vital signs, supplemental oxygen, and whole blood or 1:1 RBCs and plasma if these fluids were not available during Tactical Field Care.

ROLES OF CARE

Role 1

Initial care in the role 1 (prehospital) phase is TCCC provided by either the casualty him- or herself, nonmedical unit members, unit members with additional medical training (combat lifesavers), or combat medical personnel (medics, corpsmen, or PJs). The injured warfighter is then taken to a casualty collection point, battalion aid station (BAS), or shock trauma platoon for additional care prior to tactical evacuation.

The BAS is similar for the US Army and US Marine Corps. At the BAS, a physician, physician assistant, or medic/corpsman makes triage, treatment, and evacuation decisions. The goal of treatment is to return to duty or to stabilize and allow for the evacuation to the next role of care. There is no surgical or patient holding capacity at role 1.

The Shock Trauma Platoon is a small emergency medical unit that supports the Marine Expeditionary Force. Like the BAS, it has no surgical capacity; however, the Shock Trauma Platoon is staffed by two emergency medicine physicians and can hold patients for up to 48 hours, depending on the severity of their wounds.

Role 2

Role 2 care is the first level of care that has surgical capability. In the concept design for the battlefield, role 2 care was introduced in the 1990s when it was recognized that bridging the time and space gap from point of injury to definitive care would decrease death from hemorrhage on the battlefield. During recent conflicts, role 2 care has been heavily used and has the primary mission of damage control surgery and damage control resuscitation. Additionally, there is an introduction of basic primary care in some of the role 2 configurations in the Army and Air Force, which can include optometry, dental care, combat and operational stress control, behavioral health, laboratory services, and radiology services. This occurs when the role 2 is part of an area support medical company. Each branch of the Armed Forces has slightly different organization and capabilities.

For the US Army, the role 2 MTF is operated by the medical treatment platoon of medical companies. The medical company is assigned to support a brigade or division. Here, the warfighter is examined and evaluated. Those who can be treated and return to duty within 72 hours or who require immediate damage control surgery to preserve life, limb, or eyesight are held for treatment. The warfighter can receive blood transfusions, dental care, limited radiographic studies, clinical laboratory services, combat and operational stress control, and preventive medicine. The FST is a 20-person team with three general surgeons, one orthopedic surgeon,

two nurse anesthetists, critical care nurses, surgical scrub technicians, and combat medics. It is designed to provide life-saving resuscitative general and orthopedic surgery. An FST is deployed by ground, fixed or rotor wing aircraft, or airborne operations. A fully supplied FST can operate continuously for 72 hours on two operating tables for a maximum of 30 cases. However, an FST is not designed or equipped for stand-alone operation and thus must rely on the medical company for logistical support (electricity, water, and fuel) and security. In the last few years of Operation Iraqi Freedom and Operation Enduring Freedom, FSTs have been split to create two teams. The US Army is transitioning from the FST to a Forward Resuscitative Surgical Team. In this model, the 20-person team consists of two general surgeons, two orthopedic surgeons, two emergency medicine physicians, two nurse anesthetists, and additional nurses, medics, and operating room technicians. This staffing model provides for a symmetrical split of the team when necessary. For a smaller footprint team in support of Special Operations Command missions, the Golden Hour Offset Surgical Treatment Team (GHOST-T) was developed and implemented. The GHOST-T is tasked to support a Special Forces Group (Airborne) Operational Detachment Alpha Team and provide resuscitation and damage control surgery in a far-forward setting outside of the 60-minute medical evacuation (MEDEVAC) ring. Personnel in the GHOST-T include a surgeon, nurse anesthetist, operation room technician, medic, and nurse. The total number of team members on the GHOST-T can range from 5 to 10, depending on the length of mission and number of warfighters being supported.

In the US Air Force (USAF), the Mobile Field Surgical Team (MFST) is composed of five team members: a general surgeon, orthopedic surgeon, anesthetist, emergency medicine physician, and an operating room nurse or technician. Much like an FST, the MFST cannot participate in stand-alone operations and supports an aid station or flight line clinic. In order to accommodate the need for austere surgical support in ground combat operations, the Ground Surgical Team (GST) was established. The GST has six team members consisting of a general surgeon, anesthesiologist, emergency medicine physician, critical care nurse, operating room technician, and medic. Compared to the MFST, the GST sacrifices some surgical capability in exchange for patient holding capacity. To support special operations requirements, the Special Operations Surgical Team (SOST) was developed. This six-person team is composed of a general surgeon, emergency medicine physician, anesthesiologist, critical care nurse, cardiopulmonary technician, and operating room technician. The SOST is coupled with Special Operations Critical Care Evacuation Team (SOCCET) to provide resuscitation, damage control surgery, and critical care while aboard evacuation platforms. The 10-person Small Portable Expeditionary Aeromedical Rapid Response (SPEARR) team is an MFST with an additional three-person Critical Care Air Transportation Team (CCATT) and two-person preventive medicine team. A SPEARR team is stand-alone capable for 7 days and can perform up to 10 surgeries in 48 hours. The SPEARR team is designed to provide care during the early phase of deployment and is highly mobile. The Expeditionary Medical Support (EMEDS) Basic team builds on the SPEARR team, with an additional 15 personnel. In addition to resuscitative surgery, EMEDS Basic can provide 24-hour sick call, dental care, and limited radiology and clinical laboratory services. There are four holding beds, two operating room tables, and three climate-controlled tents. An EMEDS+10 facility has six additional holding beds for a total of 10 beds and 56 personnel but retains the same surgical capability as an EMEDS Basic.

For the US Navy (USN), role 2 care consists of a Casualty Receiving and Treatment Ship (CRTS) or aircraft carrier battle group. The CRTS is part of a three-ship Amphibious Ready Group (ARG). With 45 ward beds, 4 operating rooms, and 17 intensive care unit (ICU) beds, the CRTS has a 176-person Fleet Surgical Team with 1 surgeon, 1 nurse anesthetist, 1 ICU nurse, 1 operating room nurse, 1 general medical officer, and 12 support staff. The CRTS can be augmented with an additional two orthopedic surgeons and one oral maxillofacial surgeon. Support services include frozen RBC availability, radiology, and clinical laboratory capabilities. The CRTS can triage up to 50 patients and can only hold patients for 72 hours. The aircraft carrier battle group role 2 assets do not typically support ground forces and are not casualty receiving ships. The 52 ward beds, 3 ICU beds, and single operating room are staffed by one surgeon and five additional medical officers. For the delivery of trauma care afloat or ashore near the point of injury, the USN has the Expeditionary Resuscitative Surgical System (ERSS). The ERSS has three components: the Expeditionary Surgical Team (EST), Expeditionary Trauma Team (ETT), and the En Route Care Team (ERCT). In total, the ERSS has a general surgeon, anesthesia provider, critical care nurse, operating room technician, two emergency medicine physicians, and two independent duty corpsmen.

US Marine Corps role 2 care consists of a surgical company that provides surgical care for one infantry regiment of the Marine Expeditionary Force. The surgical company is composed of four Forward Resuscitative Surgical Systems (FRSS), four shock trauma platoons (role 1 providers), and four en route care teams. The FRSS is the basic surgical capability module that can doctrinally provide resuscitative surgery for 18 patients for 48 hours without resupply. Each FRSS has two surgeons, one anesthetist, one ICU nurse, two operating room technicians, and two corpsmen. Like the US Army FST, the FRSS is not a stand-alone unit and relies on the main unit for resupply and logistical support. The ERCT is composed of an ICU nurse and corpsman. They can transport and care for up to two critically injured warfighters (one requiring mechanical ventilation).

Although each service branch has its own form (or multiple forms) of role 2 care, the primary purpose is to control hemorrhage and implement damage control surgical and resuscitation techniques in order to stabilize life-threatening

injuries and move the patient along the continuum of care. Role 2 teams have adapted and been flexible with the mission sets. Many role 2 teams have provided definitive care for host national patients and participated in humanitarian surgical care when the battlefield medical rules of engagement allowed humanitarian care.

Role 3

Role 3 care comprises the highest level of care on the battlefield. The most common role 3 MTF is referred to as the Combat Support Hospital (CSH). The US Army is transitioning the CSH to the field hospital with the 2017 Force Design Update; the field hospital and CSH include the same capabilities. Role 3 care includes medical and surgical subspecialty care. The MTFs typically have neurosurgical, ophthalmology, vascular, and orthopedic capabilities. Although the size of the CSH varies depending on the operational need, doctrinally, a role 3 facility is resourced to care for up to 248 patients. Four ICUs and support staff can care for up to 48 critically ill patients, and 10 wards with intermediate nursing care for up to 200 patients. In terms of surgical capability, doctrinally, role 3s have six operating room tables, albeit in the current theaters of operations the capacity has been reduced. In addition to surgical expertise, radiology, clinical laboratory, blood banking, psychiatry, public health, pharmacy, and nutrition support are usually available at this role of care. Medical teams, medical detachments, or hospital augmentation teams can augment capabilities of the CSH. Augmented services can include hemodialysis, clinical pathology, infectious disease, and head and neck surgery teams (neurologic surgery, ophthalmology, and otolaryngology).

In the USAF, role 3 doctrinally, care consists of EMEDS+25 and the USAF theater hospital. With 84 support personnel, two operating room tables, and 25 patient beds, EMEDS+25 can provide up to 20 operations in 48 hours. Additional modules with subspecialty capabilities such as otolaryngology, neurologic surgery, ophthalmology, cardiothoracic, and vascular can be added to the EMEDS+25. Currently in Afghanistan, the Air Force theater hospital at Bagram Air Base provides the largest surgery capability and critical care support in theater.

Role 3 care for the USN consists of expeditionary medical facilities and the USNS *Mercy* and USNS *Comfort* hospital ships. Each hospital ship has 12 operating rooms and 999 beds with 68 ICU, 20 postanesthesia care unit, 11 respiratory isolation, 400 intermediate care, and 500 minimal care beds. These minimal care beds are usually upper bunk beds reserved for patients ready to return to full duty; 273 officers and 943 enlisted personnel staff the ship with full clinical laboratory, radiology (with computed tomography [CT] scanner), and blood banking services with frozen blood stores. The expeditionary medical facility has four operating rooms, 40 ICU beds, and 110 ward beds. Clinical support services and surgical specialties for the expeditionary medical facility are similar to the US Army CSH.

Role 4

There is currently only one role 4 OCONUS (outside the continental United States) role of care and multiple role 4 CONUS (within the continental United States) MTFs. Landstuhl Regional Medical Center (LRMC) in Germany is the only role 4 OCONUS MTF providing the first level of care outside the combat zone. All casualties (medical and surgical) pass through LRMC as part of the evacuation from the combat theater. Role 4 CONUS facilities are located throughout the United States and provide definitive, full-spectrum care for combat casualties.[16,17]

COMBAT CASUALTY EVACUATION

The en route care environment can be risky for severely injured casualties. There have been significant advances in the evacuation capabilities, and much work is ongoing in this arena. Patients are transferred intratheater usually by rotary wing aircraft (helicopters) and travel out of theater typically on fixed-wing aircraft (USAF C-17 or C-130). Evacuation and transport of combat casualties is a multiservice and occasionally a multinational endeavor. The current doctrine of US military evacuation from the point of injury is centered on the Golden Hour concept. All battlefield injuries doctrinally must be transported to a surgical capability (role 2 or role 3 MTF) within 60 minutes. Evacuation vehicles and surgical assets are geographically placed to meet these demands. Patients are transported from the point of injury serially to higher echelons of care. An arrangement of various transport platforms has been assembled, and a nonexhaustive list follows (Fig. 53-8).

Casualty evacuation (CASEVAC) refers to patient movement in a vehicle not specifically designated for medical care, essentially a vehicle or helicopter of opportunity. In CASEVAC transport, personnel may have little, if any, medical training. CASEVAC platforms are chosen when speed is more important than en route medical equipment or capabilities.

MEDEVAC pertains to vehicles that are in effect military ambulances—either by air or ground. The MEDEVAC platform most often used in recent operations is the Sikorsky HH-60M Black Hawk helicopter (Fig. 53-9). Nicknamed "DUSTOFF," a title coined during the Vietnam era, MEDEVAC helicopter crews are designed to expediently bring patients from the point of injury to the nearest surgical capability. Early in the conflicts, these vehicles were manned by basic medics, but they are now manned by advanced paramedics and en route critical care nurses. Resuscitation and stabilization of the patient continue while in the air. Hostile fire is often encountered in this process, and as a medical transport, minimal defense capabilities are available for protection.

Aeromedical evacuation (AE) is the term for USAF fixed-wing aircraft used to transport patients either within theater or out of theater. Examples would be from a role 2

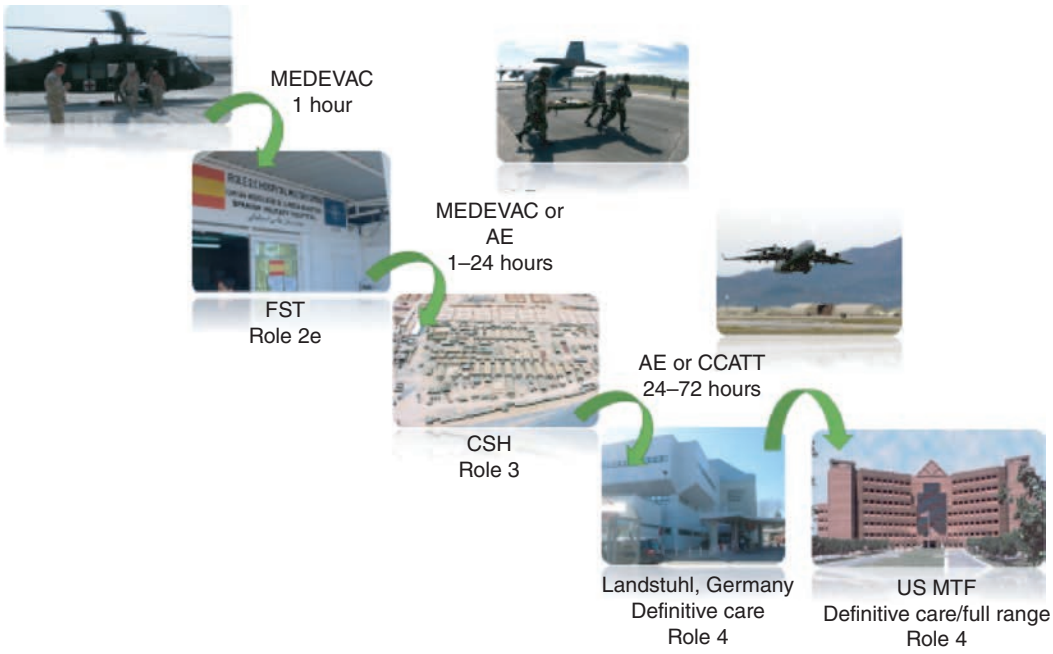

FIGURE 53-8 Schematic of roles of care and evacuation platforms. AE, aeromedical evacuation; CCATT, critical care air transport team; CSH, Combat Support Hospital; FST, forward surgical team; MEDEVAC, medical evacuation; MTF, military treatment facility.

facility to a role 3 (intratheater) or from Afghanistan to Germany. Physiologic capacity for flight and appropriate triage of patients via AE is determined by the flight surgeon, who is familiar with the demands and restrictions of care at altitude. The most common air frames used for AE include the C-130 and C-17 (Fig. 53-10). AE is used for patients who would generally meet criteria for ward or outpatient status within a hospital setting; they may be ambulatory or "litter patients" (named for the lightweight transport cot used).

Critical Care Air Transport Team (CCATT) refers to the augmented AE-type platform with specific capabilities for ICU care. Each team consists of an intensivist physician, a critical care nurse, and a cardiopulmonary technician (for ventilator and invasive monitor management). They are able to provide en route care for three to six critical patients for periods ranging from under an hour to over 16 hours. Specifically designed low-profile and air/altitude-worthy equipment, such as ventilators and monitors, is cleared for use prior to employment as part of the CCATT tool set. Despite the severity of injuries, long transport times, and additional constraints of providing care at altitude, the CCATT program has had an en route mortality of less than 0.02%.[17-20]

FIGURE 53-9 Approaching a medical evacuation Black Hawk helicopter with a casualty.

Medical Emergency Response Team–Enhanced

As the military continues to push high-level care further forward, the logical next step is physician presence on evacuation aircraft. The United Kingdom's Medical Emergency Response Team–Enhanced (MERT-E) represents this capability. It operates out of a CH-47 Chinook, a larger two-rotor helicopter. The team is designed to meet the needs of the most critically injured patients when evacuation times and distances may be long. A study evaluating the period from July to November 2008 recorded 324 missions for 429 patients. A physician was present for 90% of missions. A total of 64 physician interventions were performed including tube thoracostomy, death pronouncements, sedation, and blood product administration. In certain cases, the increased medical capability may be offset by the larger rotary wing platform that is unable to land in a "hot zone"; DUSTOFF MEDEVAC has less medical capability but will land in hot zones, which results in shorter transport times to role 2 or role 3 care. The benefit of physician presence overall is

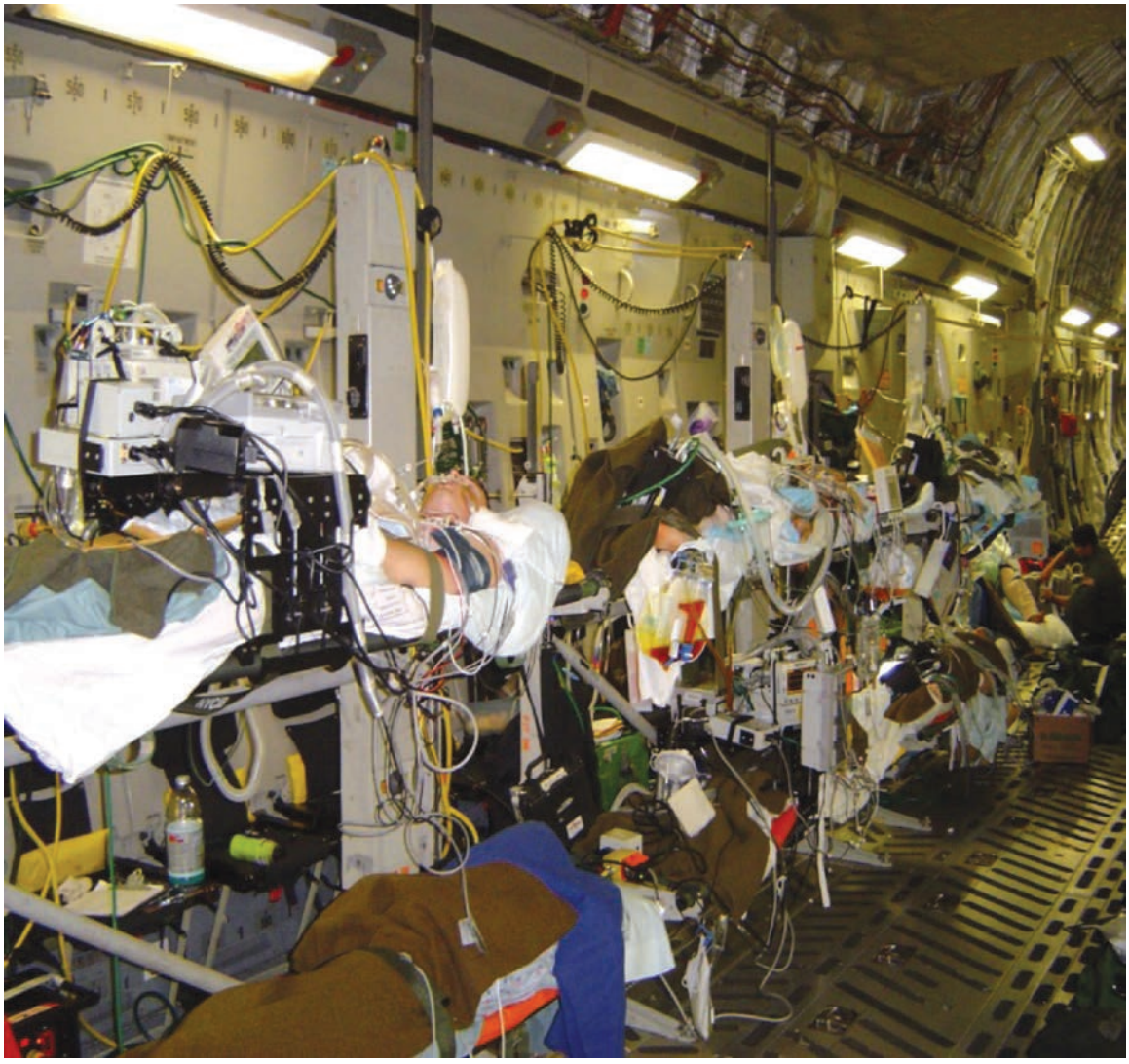

FIGURE 53-10 Critical care air transport team (CCATT) assembled in a US Air Force C-17 aircraft.

unclear as the decision not to perform advanced interventions can sometimes be important and is more difficult to measure. Additionally, the MERT-E model is equipment and personnel resource intensive in environments that often have minimal resources.[21] A comparative evaluation of forward AE platforms found lower mortality for all Injury Severity Score groups with the MERT-E platform.[22]

DAMAGE CONTROL RESUSCITATION

One of the greatest advances in the recent conflicts is the adoption of an overall damage control approach to battlefield care. The introduction of role 2 surgical capability is the battlefield planning aspect of damage control. Although the concept was introduced in 1983[23] in the description of an abbreviated laparotomy for a patient with a coagulopathy, the term *damage control* was introduced 10 years later in the landmark publication by Rotondo et al.[24] The refinement of damage control resuscitation (DCR) principles represents one of the major advances in trauma surgery during recent years, and the military has adopted the concept of damage control along the entire continuum of care. The high tempo and large volumes of casualties during combat operations lasting over a decade have provided a crucible for this change. Damage control starts with tourniquet use at the point of injury and also includes hemostatic resuscitation with blood component ratios that replicate whole blood, early whole blood use, prehospital transfusion, short transport times, minimal/no crystalloid use, and damage control surgical techniques. Patients with trauma necessitating a massive transfusion require vigilant attention throughout their treatment—from point of injury to the first MTF and definitive surgery, and throughout the continuum of care, including the en route care environment.[25]

Damage Control Criteria Recognition: The Lethal Triad

Acute coagulopathy of trauma (ACT) is caused by the combination of severe tissue injury and shock, and is mediated by activated protein C.[26] The infusion of high-chloride-containing crystalloid solutions exacerbates ACT by inducing the lethal triad, producing trauma-induced coagulopathy. The lethal triad is defined by the presence of acidosis, hypothermia, and coagulopathy in the setting of traumatic hemorrhage. It is a vicious cycle of clinical findings associated with increased mortality compared with noncoagulopathic patients.[27] Once this vicious cycle is induced, it is difficult to reverse as each component of the triad results in worsening of the others. There is tremendous interest in developing criteria to identify patients who require DCR early. Unfortunately, there is no single scoring system that will distinguish all traumatic coagulopathy patients requiring DCR.[28] Mechanism and clinical suspicion remain important factors for early recognition. Current predictors used by the US military as established by the CPG on DCR at forward facilities are included in Table 53-3 and are discussed in the following sections.[7]

ACIDOSIS

Acidosis, generally recognized as a pH less than 7.25, represents part of the lethal triad. A base deficit of greater than 6 has been demonstrated as an independent risk factor for coagulopathy and is associated with increased mortality.[29] Recognized via point-of-care testing, acidosis provides an early indication of metabolic derangements from profound hemorrhage. Acidosis impairs both platelet function and the coagulation cascade.[28]

COAGULOPATHY

Coagulopathy is concerning in the setting of trauma as both an indicator of increased mortality and a barrier to hemorrhage control. An international normalized ratio (INR) greater than 1.5 is an independent predictor of massive transfusion and is usually available in the resuscitation bay.[30] Impaired coagulation requires replacement of clotting factors and platelets rather than red cells alone.

TABLE 53-3: Clinical Predictors for Recognizing Patients Requiring Damage Control Resuscitation per the Most Recent US Military Clinical Practice Guidelines

Acidosis	pH <7.25
Coagulopathy	INR ≥1.5
Anemia	Hematocrit <32%
Hypotension	Systolic blood pressure <110 mm Hg
Tachycardia	Heart rate >105 bpm
Uncontrolled hemorrhage	

INR, international normalized ratio.

HEMOGLOBIN AND HEMATOCRIT

Anemia secondary to acute blood loss is another measure easily determined in the trauma bay. Traditional teaching dictates that the hemoglobin and hematocrit will not initially change immediately after hemorrhage as red cells and plasma are lost in equal proportions. Based on this premise, anemia would not be evident until after plasma levels equilibrate for the lost whole blood volume. However, when hemoglobin measures are obtained within the first 30 minutes of arrival to the emergency department, nearly 90% of trauma patients with major bleeding will be identified.[31] A hemoglobin less than 11 g/dL has been determined to be an independent predictor of massive transfusion.[30]

SYSTOLIC BLOOD PRESSURE

The presence and degree of hypotension provide measures for diagnosis, prognosis, and treatment in the bleeding trauma patient. Systolic blood pressure (SBP) less than 90 mm Hg is commonly used as the definition of shock. Advanced Trauma Life Support teaching also advocates for the use of blood pressure in its classification of hemorrhage. However, once a SBP below 90 mm Hg is reached, this already corresponds to a 40% loss of circulating volume—a late finding.[32] Earlier changes in blood pressure have been demonstrated to be indicative of changes in prognosis. A large study of NTDB patients found the prognostic cutoff to be 110 mm Hg, below which mortality increased by 5% for every decrement of 10 mm Hg.[33] An important note, however, is that prognostic criteria cutoffs do not necessarily translate into therapeutic goals. *Permissive hypotension* is an example of this. A goal of DCR is to keep the SBP at approximately 90 mm Hg prior to definitive surgical hemorrhage control. Previous experiences with SBP goals at normal levels translated into increased mortality.[34] Although an SBP of less than 110 mm Hg is useful to determine severity of hemorrhagic insult, it should not be used as an early resuscitation target.

Damage Control Intervention: Resuscitative Fluid Strategies

CRYSTALLOID MINIMIZATION AND SELECTION

Crystalloids have long been used in trauma as volume expanders to support blood pressure. However, select resuscitative fluids can propagate acidosis, and normotension may exacerbate hemorrhage. Rather than being resuscitative as intended, crystalloids can increase morbidity and mortality.

In far-forward prehospital settings, blood products are rarely available and crystalloids remain one of the options available for fluid resuscitation. In these situations, it is important to consider which type of crystalloid to use. The most common fluids used are normal saline (NS) and lactated Ringer's (LR). There are several key differences between the two, but the most notable is the fluid pH.

Saline infusions were developed in 1831 after observed "decreased fluidity" of blood among cholera patients. Despite the widespread use of NS, there is no available scientific basis

TABLE 53-4: Comparison of 0.9% Sodium Chloride (Normal Saline) Versus Lactated Ringer's Solution[30]

	Normal saline	Lactated Ringer's
pH	5.0	6.5
Osmolarity (mOsm/L)	308	272
Na (mEq)	154	130
Cl (mEq)	154	109
Potassium (mEq)	0	4
Calcium (mEq)	0	3
Lactate (mEq)	0	28
Coadminister with blood	Yes	No

for its initial use. The closest evidence is an in vitro study by a Dutch chemist, Jakob Hamburger, in 1888 noting a shared freezing point between human plasma and 0.9% NS of –0.52°C.[35] Ringer's solution was developed in the 1880s by Sydney Ringer with the purpose of maintaining activity of a frog myocardium using electrolyte constituents resembling plasma.[36] Later, lactate was added to Ringer's original solution (by Alexis Hartmann) to combat the effects of acidosis through conversion to bicarbonate in the liver.

Several animal studies over the past decade have supported improved outcomes with LR over NS.[37-39] Counterintuitively, hyperkalemia is increased with NS as opposed to LR. Hyperkalemia is attributed to extracellular shift of potassium due to the low pH of 5.0 found in NS (Table 53-4).[40] With both LR and NS, there is concern for dilution of clotting factors compounding coagulopathy. There are situations when judicious crystalloid resuscitation is appropriate. Part of this strategy includes the target blood pressure for resuscitative efforts.

BLOOD TRANSFUSION STRATEGIES

The primary tenant of the current concept of DCR is the use of blood—either whole blood or blood products in ratios replicating whole blood. This strategy comes directly from military experience after observing mortality trends associated with blood product ratios. Borgman et al[41] reviewed 246 patients at a US Army CSH in Iraq evaluating ratios of plasma to red cell units and association with mortality rates. This study revealed a significant decrease in mortality as the ratio approached 1:1. Results of the study by Borgman et al[41] provided confirmation for changes in the CPGs, which had already taken effect during 2004.

The move away from transfusion of low plasma to red cell ratios marked an important transition from previous practice. The use of individual blood product components had been dictated by laboratory parameters rather than the patient's clinical situation.[25] Patients would receive plasma for an elevated INR or partial thromboplastin time rather than the presence of severe hemorrhage. Similarly, platelet administration had been guided by measured thrombocytopenia. The transition to higher ratios was based on a paradigm shift toward matching the whole blood that was being lost. Despite changes in both military and civilian DCR guidelines, the ideal ratio is unknown.

The goal of the Prospective Observational Multicenter Major Trauma Transfusion (PROMMTT) study was to provide further evidence regarding blood component ratios, particularly early transfusion of plasma and platelets. A total of 1245 patients from 10 US Level I trauma centers receiving at least 1 unit of RBCs within 6 hours of admission were in the original study group. Multiple separate subgroup analyses have been published from the data obtained. Among 905 patients from the main study group who also received greater than or equal to 3 component unit transfusions, increased plasma-to-RBC ratios were independently associated with decreased 6-hour mortality. Hemorrhagic deaths predominated in the first 6 hours. Patients with high ratios either tended to receive a 1:1:1 or 1:1:2 ratio of plasma to platelets to RBCs.[42,43]

The Pragmatic Randomized Optimal Platelet and Plasma Ratios (PROPPR) trial further addressed the question of ideal transfusion ratios. A total of 680 patients were randomized to a 1:1:1 versus a 1:1:2 ratio during active resuscitation at 12 Level I trauma centers in the United States. There was no statistically significant difference in 24-hour or 30-day mortality. However, the 1:1:1 group achieved hemostasis more frequently and were less likely to die of hemorrhage in the first 24 hours.[44]

The use of an early balanced and hemostatic resuscitation strategy with blood products has been a significant paradigm shift that came directly from the experience, data analysis, and results from the wars in Iraq and Afghanistan and has been validated in civilian trauma. Hemostatic resuscitation is designed to replicate whole blood, and the military experience in treating combat casualties has demonstrated that whole blood is associated with superior outcomes when compared to component therapy. Anecdotally, fresh whole blood has been coined a *physiology changer* in the operating room.[45-48]

Prehospital Transfusion. Initiatives to bring blood far forward, as close as possible to the point of injury, have proven to be lifesaving. In 2012, a prehospital transfusion protocol initiated in Afghanistan placed RBCs and fresh frozen plasma (FFP) on MEDEVAC helicopters. Criteria for transfusion were signs of shock or mechanism of injury. Results from this protocol implementation demonstrated that prehospital blood product transfusion of combat casualties improved early and 30-day survival.[45,49] The retrospective analysis of US military casualties injured in Afghanistan and transported by en route care platforms with a transfusion capability demonstrated that transfusion within minutes of injury significantly reduced mortality. This study was unique compared to other prehospital transfusion studies in that all deaths in this military population were captured; this type of study design proved to be crucial when considering early hemorrhagic

deaths given they are the cohort of patients predicted to most benefit from prehospital transfusion. The study demonstrated that the median time to initiation of transfusion, 36 minutes after injury or 7 minutes after MEDEVAC, was associated with improved survival. This study supports sustainment of a system of care that reliably supports prehospital transfusion and implementation and investigation of similar systems in civilian trauma settings. Very recent results looking at prehospital plasma transfusion demonstrated decreased mortality, especially with longer transport times.[50]

Given that the vast majority of combat deaths occur in the prehospital environment prior to reaching a surgical capability, these are the combat casualties who will most benefit from blood far forward. Whole blood is the logical choice for a nearly perfect resuscitative fluid in the far-forward environment given that it has oxygen-carrying capacity, coagulation factors, and platelets in the same ratios that are lost during exsanguination. It is logistically easier to carry and transfuse 1 unit of whole blood compared to multiple units of components. In the current theaters of operation, blood transfusion capability continues to mature at both point of injury and in the en route care environment. In 2013, the Norwegian Special Operations Forces instituted a remote DCR protocol that includes far-forward collection and transfusion of whole blood. A similar protocol for tactical DCR in order to transfuse low-titer type O whole blood at point of injury was adopted by US Army Ranger Regiment: Ranger O-Low-Titer Type O (ROLO). Currently, US Special Operations Forces carry low-titer universal donor whole blood on select missions, and there is increasing availability of type O low-titer whole blood in the forward deployed environment.[45,51-53] Transfusion far forward is an essential capability that saves lives of combat casualties.

WHOLE BLOOD VERSUS COMPONENT THERAPY

Whole blood has a long history of use on the battlefield and was the primary resuscitation fluid for hemorrhage in World War I, World War II, the Korean War, and early in the Vietnam War. During the first two world wars, a combination of fresh whole blood (FWB) and stored whole blood (SWB) was used, with a peak of 2000 units per day in 1945.[54,55] The transformation to crystalloid therapy and component product resuscitation lacked clinical evidence of superior efficacy or improved outcomes; nevertheless, it was widely adopted until data from the wars in Iraq and Afghanistan resulted in the paradigm shift to a resuscitation strategy that replaces components in a manner mimicking the composition of whole blood. Despite this paucity of initial evidence, component therapy became the standard for blood banking.[56] Early in the recent wars, whole blood was used primarily by forward deployed teams that were equipped with limited units of packed RBCs (PRBCs); therefore, during this period, whole blood transfusion capability was a necessity driven by component therapy availability and was not necessarily clinically compelled. At the role 3 CSHs, whole blood was initially used because of the lack of platelet availability. Over time, through both focused empiricism and investigations of comparative efficacy that demonstrated improved survival with whole blood,[57] battlefield hospitals no longer use whole blood solely because of lack of component therapy availability, but also secondary to its clinical superiority. There continues to be enthusiasm for whole blood use in both military and civilian settings, and it is being considered, studied, and reestablished as the optimal therapy for hemorrhage. As of November 2018, over 10,550 units of whole blood have been transfused during the wars in Iraq and Afghanistan. These transfusions have almost exclusively occurred in the "hospital" setting: role 2 (FSTs) or role 3 (CSHs). Not only have complications been exceedingly low, but whole blood has repeatedly demonstrated improved outcomes in casualties with hemorrhagic shock.[58]

The reexamination of whole blood in the current era was also led by the military community. Logistical restraints are imposed on effective component therapy transfusion at forward operating bases owing to long supply chains and limited processing and storage of components. Platelets are stored at room temperature, require constant agitation, and have a shelf life of 5 days due to increasing risk of bacterial contamination. FFP and cryoprecipitate must be stored at –20°C and require at least 30 minutes to thaw. Additionally, the storage capacity for all components is limited.[59] Whole blood addresses many of these limitations as it eliminates the need for multiple products and storage conditions.

The theoretical clinical benefits of whole blood include avoidance of dilution, absence of anticoagulant additives, consistency with damage control surgery principles, and blood product age. Although a 1:1:1 transfusion ratio with component therapy attempts to approximate whole blood, it creates a dilute blood mixture with a hematocrit of 29% and platelet count of 90,000/μL, and 62% of coagulation factors found in fresh whole blood are achieved. As with component therapy, whole blood transfusion must have donor RBCs that are ABO compatible with the recipient in order to prevent acute hemolytic transfusion reactions. Group O whole blood donors have RBCs that are compatible with all recipients, but the plasma contains anti-A and anti-B antibodies that may cause hemolysis in a non–group O recipient. Therefore, only group O whole blood donors with low titers of anti-A and anti-B antibodies (<1:256 saline dilution method) are used. This whole blood can be considered universal and is known as low-titer O whole blood (LTOWB). Donors of LTOWB should be retested annually at minimum in order to ensure titers remain below 1:256. Additionally, LTOWB is collected from males and nulliparous women to lessen the risk of transfusion-associated lung injury in recipients.

FWB is whole blood collected from a walking blood bank (WBB) on an emergency basis. The WBB consists of volunteer donors (prescreened when possible) who are not needed immediately for warfighting activities or active resuscitation of the patient(s). FWB can be kept at room temperature up to 24 hours prior to use. Due to emergency conditions under which FWB is collected, it does not undergo transfusion-transmitted disease (TTD) testing prior to use. For this reason, FWB is not US Food and Drug Administration

(FDA) approved because it carries an increased risk of disease transmission.

SWB is whole blood that is collected, stored, and tested for TTD and is FDA approved. The Armed Services Blood Program provides SWB only as LTOWB to locations outside the continental United States (OCONUS). It can be stored for 21 days in CPD (citrate, phosphate, dextrose) at 1°C to 6°C and 35 days in CPDA-1 (citrate, phosphate, dextrose, adenine) at 1°C to 6°C. However, after 14 days, the hemostatic function of the platelets may be less effective and supplementation with fresher whole blood may be necessary. Of note, the use of LTOWB is now permitted by the American Association of Blood Banks (AABB), and it is used by several civilian trauma centers in the United States.

For DCR at the point of injury or in en route care, remote locations, or MTF, the order of priority of fluid administration should be as follows: (1) LTOWB, (2) component therapy at 1:1:1 ratio, (3) RBC and plasma at 1:1 ratio, (4) plasma and RBC at any ratio, and (5) RBC alone. Current US practice is guided by the US Army Institute of Surgical Research Clinical Practice Guideline for Massive Transfusions.[7]

ADJUNCTS AND ALTERNATIVES TO COMPONENT THERAPY: LYOPHILIZED PLASMA AND CRYOPRESERVED RED BLOOD CELLS

Storage conditions remain a limiting constraint on the availability of blood products during military operations. For the combat medic, weight is paramount because everything needs to be carried and refrigeration is rarely available. At the FST or CSH, the refrigerated shelf life of PRBCs can limit availability of this product.

The benefits of early plasma transfusions have been recognized with new information on component therapy ratios. The most common form of plasma, FFP, must be stored at –18°C and then undergo a thawing process in a warming bath for approximately 15 to 20 minutes.[60] These processes require specialized bulky equipment that is difficult to house or completely absent at far-forward facilities and for first responders. However, lyophilized (freeze dried) plasma represents a lightweight, easily carried option that can be stored at room temperature. Lyophilized plasma represents another rediscovery of previous military advances finding new uses again today. In World War II, lyophilized plasma was used due to the same concerns as today—transport and shelf life. Unfortunately, pooled plasma that was not screened for viral pathogens was used at that time—with associated increased viral infection risk. Thus, the practice was abandoned. With the ability to screen and test for disease more effectively, lyophilized plasma is being reinvented. Recent work evaluating the properties of lyophilized plasma has demonstrated 86% of prelyophilization coagulation factor activity[17] and decreased blood loss compared with FFP in a combat-relevant swine model.[61,62] European production of lyophilized plasma has continued in recent years with notable use by the French and Germans in overseas military activities at role 1, 2, and 3 facilities and MEDEVAC platforms.[63] In July 2018, the FDA issued an Emergency Use Authorization to allow for the use of pathogen-reduced leukocyte-depleted freeze-dried plasma (French FDP) to treat hemorrhage or coagulopathy during a medical emergency involving military combat when plasma is not available. This product is a lyophilized, pathogen-reduced, leukocyte-depleted pooled apheresis plasma product collected from volunteer donors. The packaged unit contains one flask of freeze-dried plasma, one flask of 200 mL of sterile water, one transfer set, a sterile tubing set with an in-line 200-µm filter, and a needle for product infusion. French FDP can be stored between 36°F and 77°F for 2 years. At the time of use, the FDP and sterile water should be brought to room temperature. The sterile water flask is spiked first, followed by the FDP flask. If the FDP is spiked first, the vacuum is lost and subsequent water transfer becomes more difficult. Once the water has filled the FDP flask, gentle mixing without generating foam is completed over the span of 3 to 5 minutes.

Deglycerolized RBCs (DRBCs) were first used on a large scale by the US military during the Vietnam War after large volumes of standard PRBC preparations were being discarded due to age. The technique currently approved for use by the FDA involves the use of a glycerol buffer to prevent crystal formation–associated membrane disruption and cellular damage due to large temperature changes. The red cells are stored at –80°C with 40% glycerol weight/volume within 6 days of collection. They can be stored for up to 10 years before they are thawed and deglycerolized for patient use.[64,65] The storage lesion is minimized while also allowing for larger inventories of blood in settings with sporadic or unpredictable use. Despite these benefits, including FDA approval, DRBCs are only being employed in rare specific situations today. This is likely attributed to the increased time (~90 minutes) and cost associated with the thawing and deglycerolizing process. They have been used increasingly in current military operations with hundreds of units provided and are used in an equivalent manner to PRBCs. They are also part of the response plan for potential large-scale natural or manmade disasters domestically.

Damage Control Interventions: Temporary and Definitive Direct Hemorrhage Control

The general principle of damage control in the combat environment is to provide only the interventions required to safely get the patient to the next echelon/role of care. This approach begins at the point of injury with the teachings of TCCC, including tourniquets and topical hemostatic agents. It continues through the FST with a variety of vascular control and repair techniques. The goal is to salvage potentially survivable injuries, over 90% of which are associated with hemorrhage.[12]

TOPICAL HEMOSTATIC AGENTS

Research and development of novel topical hemostatic agents has seen much interest during the current era of combat casualty care. The agents primarily used in theater are either chitosan (a positively charged compound derived from shrimp shells) or

kaolin (aluminum silicate mineral) based. Chitosan-based dressings, such as Celox, work as mucoadhesive agents that stick to open wounds, sealing bleeding. Kaolin-based dressings, such as Combat Gauze, work by accelerating clotting pathways. When used as a straight powder or impregnated gauze, these agents can provide hemorrhage control alone or as an adjunct to tourniquet use. As addressed in the earlier section on tactical field care, Combat Gauze is used as the primary hemostatic by first responders based on CoTCCC guidelines. The gauze has been selected over powder formulations for ease of use and ability to provide compression in cavitary wounds. Hemostatic gauze products continue to be useful in the operating room as well and have become common in civilian settings. There has been an explosion of improvements and options for topical hemostatic agents in recent years with several new agents being investigated currently.

Self-expanding foam represents a promising new technique to provide hemostasis for otherwise noncompressible intra-abdominal hemorrhage. A polyurethane expanding foam that is introduced into the abdomen as two liquids that combine and polymerize providing pressure on bleeding sites has recently been FDA approved for early clinical trials to assess for efficacy and safety. This intracavitary foam is designed to temporize hemorrhage until definitive surgical control can be achieved.[66]

Expandable sponges under the label XStat (RevMededx, Wilsonville, OR) have also been developed through military research, with FDA approval for use in 2014 (Fig. 53-11). The device consists of a large syringe that can deliver many pill-sized, nonabsorbable, radiopaque sponges, which expand within 20 seconds of contact with moisture. The volume of material delivered and the size of the delivery device provide tamponade control of junctional wounds in the groin and axilla.

TRANEXAMIC ACID

The Clinical Randomization of an Antifibrinolytic in Significant Hemorrhage (CRASH-2) study, an international prospective randomized trial of over 20,000 trauma patients, demonstrated a mortality benefit with tranexamic acid.[67] When studied in a military role 3 hospital, the Military Application of Tranexamic Acid in Trauma Emergency Resuscitation (MATTERs) study also revealed a mortality advantage.[68] In both studies, the benefit was seen only if treatment occurred early after injury (<3 hours). As such, tranexamic acid is currently endorsed in the TCCC guidelines as well as the DCR CPG.[7]

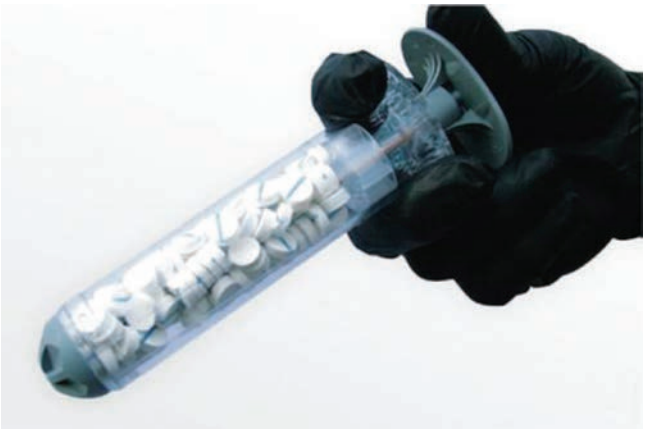

FIGURE 53-11 XStat expandable sponge delivery device.

TRAUMATIC ARREST AND PROFOUND SHOCK

Combat casualties have a high frequency of penetrating injury from mechanisms designed to be lethal. Traumatic arrest and profound shock en route to the first surgical facility are realities that must be anticipated. Aortic occlusion represents a means to limit hemorrhage and maintain perfusion to neurologic and central structures. However, this remains a very aggressive maneuver, and patient selection is paramount.

RESUSCITATIVE THORACOTOMY

The indication for resuscitative thoracotomy is usually loss, or impending loss, of vital signs in the initial resuscitation setting after penetrating trauma. The specific indications for this extreme measure vary between institutions and remain an area of contentious debate. It is promoted as the "last opportunity" to save a life. However, the nature of the procedure poses risk to medical personnel in terms of infectious disease exposure from sharp rib fractures or instruments. A large series of emergency thoracotomies during a 4-year period in Iraq revealed an overall survival rate of 11%.[69] The current US military indication for emergency thoracotomy is limited to penetrating trauma, without isolated head injury and not if vital signs have been lost for over 10 minutes.[7]

RESUSCITATIVE ENDOVASCULAR BALLOON OCCLUSION OF THE AORTA

The use of intra-arterial balloon occlusion of the aorta was first reported in 1954 by Colonel Carl Hughes.[70] However, it has only recently gained popularity in the current era of expanded endovascular technologies. Resuscitative endovascular balloon occlusion of the aorta (REBOA) has several potential advantages over emergent thoracotomy. In terms of risk to providers, this catheter-based technique reduces the opportunities for direct exposure to biohazardous materials through puncture by broken ribs. As the balloon can be inflated at different locations, selective occlusion—either in the chest or inferior to the renal arteries—can be performed. Particularly for the patient with exsanguinating isolated pelvic trauma, obtaining the benefits of aortic occlusion while maintaining renal and visceral blood flow is possible. REBOA does require a specialized skill set and equipment that may not be available in all circumstances. Recent promising experiences with REBOA have prompted the creation of military-specific indications for its use (CPG).

OPERATIVE RESUSCITATION

Operative intervention in the setting of DCR is an extension of prehospital and initial MTF intervention goals of

hemorrhage and coagulopathy management. Unlike most civilian settings, *both* tactical and physiologic considerations mush be considered. A prime example is the FST, which often operates in a split-based configuration (two general surgeons or one general surgeon and one orthopedic surgeon) with one operating room and only 6 hours of holding capacity after surgery. The focus includes rapidly assessing the patient, controlling hemorrhage, minimizing contamination, and facilitating evacuation to the next higher level (role/echelon) of care. Because time and resources are limited, only those interventions that are required to stabilize the patient for transport to the next level of care are performed.

Damage control laparotomy is a familiar component of DCR in the setting of intra-abdominal or lower extremity junctional hemorrhage. Hemorrhage is controlled by ligation, repair, compression, or shunting. Injured bowel requiring resection is left in discontinuity. The abdomen is packed and left open in a nearly universal manner. This allows for an abbreviated operative time, facilitating expedient return to the ICU for further resuscitation. Additionally, the operating room is made available more rapidly for the next patient(s)—who regularly arrive in multiples. The chaos inherent in a war zone can adversely affect communication concerning the patient's injuries. An open abdomen allows the next-higher receiving MTF to directly visualize the injuries sustained when communication is impaired. The abdominal pack dressing is routinely labeled with indelible ink as an additional means to ensure the full extent of injuries is recognized and can be appropriately treated (Fig. 53-12).

Major arterial injury presents conflicting priorities of control to prevent exsanguination and preservation of flow to maintain viability of tissues distant to the injury. The primacy of life over limb remains. When the limb can be salvaged while preserving the patient's life, the intervention used is contingent upon the resources available. Extra time spent on a formal repair may place other casualties needing operative intervention at unacceptable risk. Generally speaking, at role 2 facilities, the employment of temporary vascular shunts is encouraged over formal repair, which is best left to the role 3 or higher. Exploration, proximal and distal control, thrombectomy, and local heparinization are still used for shunting (CPG for vascular injuries). Not all vessels can or should be shunted; a list of major vessel considerations for shunting is provided in Table 53-5.[18] When an extremity's vascular inflow is maintained through shunting or repair, a fasciotomy is currently recommended. The amount of soft tissue compartment swelling after injury in young, muscular patients is often profound.

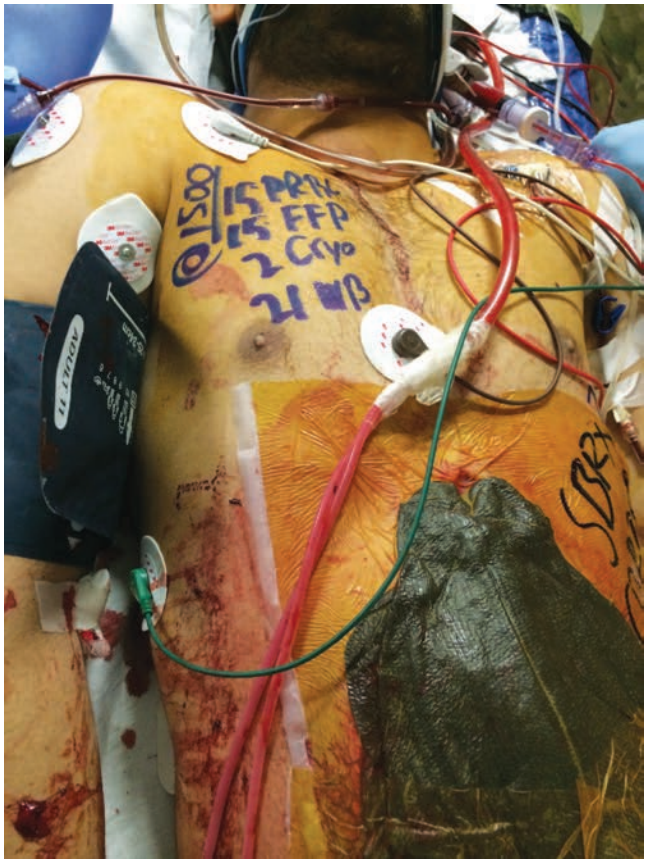

FIGURE 53-12 Open abdomen labeled for transport.

SPECIAL MISSIONS

Acute Lung Rescue Team/Extracorporeal Membrane Oxygenation in Transport

Combat trauma-associated acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) are present in 26% to 33% of critically ill casualties.[71,72] ALI and ARDS among combat-wounded patients are attributed to sources including blast injury, pulmonary contusion, transfusion complications, fat/marrow emboli, pneumonia, inhalation injuries, sepsis, near-drowning, and large overall injury burden. CCATT had been established for the transport needs of critical patients out of the theater of operations. Most patients with ALI/ARDS are responsive to standard low-volume ventilation lung-protective strategies based on

TABLE 53-5: List of Vessel Considerations for Temporary Vascular Shunts

Consider for shunting	Poor candidates for shunting
Subclavian artery (difficult)	Radial and ulnar arteries
Axillary artery	Profunda femoris artery
Brachial artery	Tibial arteries
Common femoral artery	Vertebral arteries
Superficial femoral artery	Portal vein
Popliteal artery	Mesenteric vessels
Large extremity veins	Jugular vein
Aorta	
Iliac arteries	

ARDS Network guidelines.[73] However, there remains a subset of patients with such severe pulmonary injury that they either have to wait for improvement to meet CCATT criteria or die prior to evacuation. In 2005, the Acute Lung Rescue Team (ALRT), a joint service endeavor, was implemented to meet the needs of these patients. The ALRT involves an intensivist-led team available for consults from the combat zone and capable of activating a team to meet and transport severely lung-injured patients. Composed of two critical care physicians, two critical care nurses, and two respiratory therapists with specialized experience, the ALRT team is able to provide over 24 hours of continuous care. Adjuncts to standard mechanical ventilator support employed include prone positioning, buffer therapy, inhaled prostacyclin, and use of the Volumetric Diffusive Respiratory (VDR-4) (Percussionaire Corporation, Sandpoint, ID).[74]

Eventually, pump-driven venovenous extracorporeal membrane oxygenation (ECMO) has been employed with cannulations being performed at the role 3 hospitals in Iraq and Afghanistan. The most recent ECMO transfer was from Baghdad, Iraq, to San Antonio, Texas, in November 2017 (Fig. 53-13). The miniaturization of the ECMO filter and circuit combined with improvement in the electrical/computer interface have made these advances and transcontinental transports possible.[71,75,76]

Care of the Military Working Dog

Military working dogs (MWDs) are regularly employed for detection of explosives and illicit materials. They are an integral component of special operations and military police activities and often involved in far-forward activities. In the setting of injury or illness, the ideal responders are military veterinary personnel. However, other health care providers may be the only available resource for preservation of life, limb, or eyesight. The scope of this chapter does not allow for an exhaustive description of battlefield canine care, but there are some cogent considerations below.[77] Upon stabilization, the MWD should be evacuated to a military veterinarian at the earliest convenience.

- Demographics and clinical information should be confidential, similar to MWDs' human counterparts.
- The MWD's handler should be present during care for the protection of the animal and the treating staff (Fig. 53-14). Muzzles should be used during examination and treatment.
- Direct communication with a military veterinarian should occur prior to any surgical procedure to provide guidance.
- A thin or fenestrated mediastinum is present in dogs, requiring *bilateral* chest tubes in the setting of a unilateral pneumothorax.

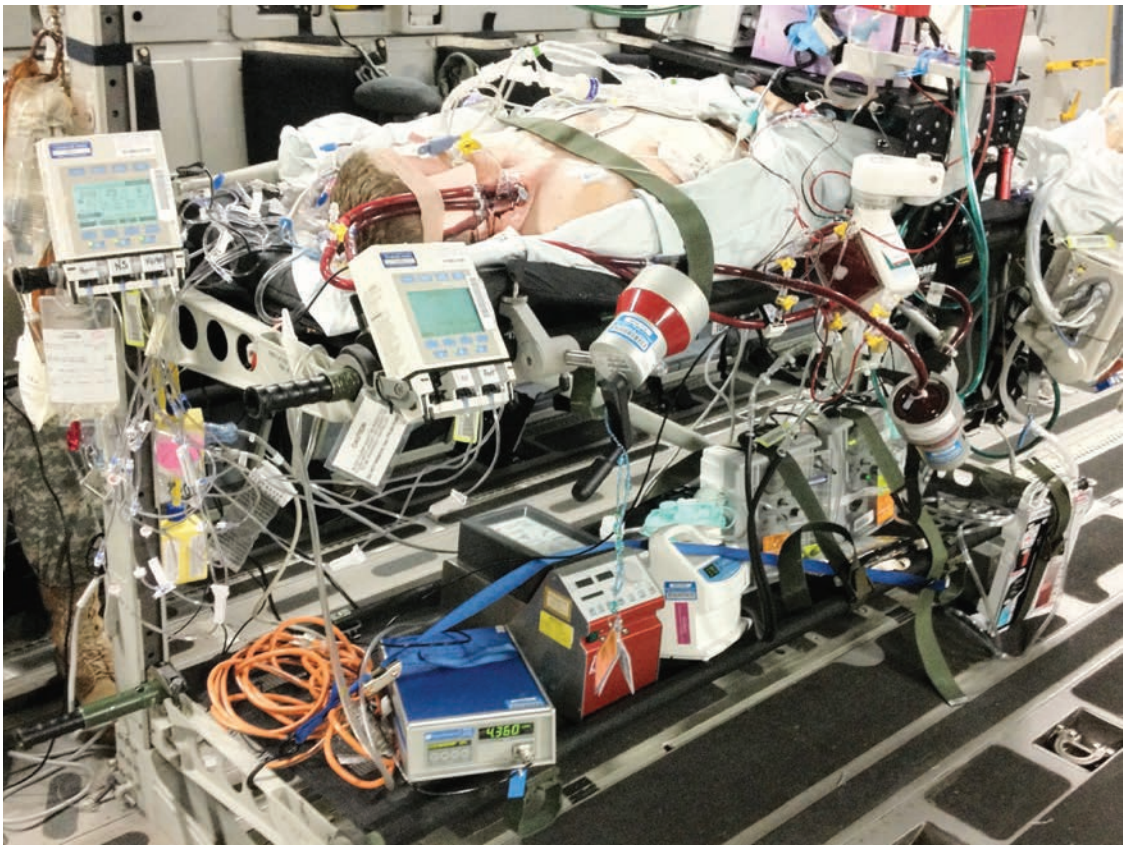

FIGURE 53-13 Extracorporeal membrane oxygenation (ECMO) patient being transported via critical care air transport team.

FIGURE 53-14 Military working dog receiving medical care attended by their handler.

- Pulse oximetry can be placed on the tongue in a sedated dog.
- Gastric dilatation-volvulus syndrome is a common canine surgical emergency. Prophylactic gastropexy is performed in most MWDs, but not all, and is still subject to failure. Clinical presentation includes abdominal distention, ineffective vomiting, and signs of shock (due to decreased venous return). Lateral thoracic radiography aids in the diagnosis. Treatment consists of immediate decompression by a percutaneous gastric trocar (14- to 18-gauge IV) decompression. Definitive therapy requires exploratory laparotomy, detorsion, gastropexy, and resection of ischemic tissue, preferably performed by a veterinarian if urgent evacuation is possible after decompression.[78]
- Acetaminophen and ibuprofen can cause liver toxicity in MWDs—DO NOT USE.
- MWDs are subject to canine posttraumatic stress disorder–like syndrome; listen to the handler for changes in behavior after significant events.
- MWDs may be euthanized in the case of catastrophic wounding with poor prognosis for recovery and in order to relieve the MWD from undue suffering.

Recent evidence confirms that MWDs can be adequately resuscitated and/or treated by nonveterinarians. A study of gunshot wounds sustained by MWDs in Iraq and Afghanistan revealed 29 such injuries. Half of the wounds were thoracic but otherwise had a similar distribution to humans. The died of injury rate was 38%, and 17 of the 18 MWDs killed died prior to evacuation or treatment. Of the 11 survivors, all were returned to duty.[79]

Joint Trauma System and Department of Defense Trauma Registry

One of the biggest advances of the recent conflicts was the development of the military trauma system. When the United States entered into the conflicts in the Middle East in 2002, the military did not have a codified trauma system that unified the levels of care and set the standards for battlefield trauma care delivery. While trauma care has advanced through the exchange of innovation and experience between civilian and military medicine going back to antiquity, statutory changes that have occurred in 2017 should stand the test of time helping to ensure that the lessons learned from these conflicts will not be forgotten. Examples of the reciprocity between the development of military and civilian trauma systems were seen during the Vietnam War when prehospital interventions and decreased evacuation times due to aeromedical platforms influenced civilian trauma care. During the same era, the American College of Surgeons shaped civilian trauma management through the publications *Early Care of the Injured Patient* followed by *Optimal Resources for Care of the Injured Patient.*[80,81] These resources became the guide for the development of civilian trauma centers that have been associated with improved outcomes. The trauma system is defined as "an organized effort in a geographic region to deliver a full range of trauma care."[82] Organization within an area of operations or activity allows for increased oversight and information availability across the spectrum from the prehospital setting through definitive care. Areas covered by an integrated trauma system have shown a decrease mortality of 15% to 20%.[82]

Shortly after the military conflicts began in the Middle East, medical and military leaders looked toward the successes of civilian trauma systems and began to build a military system that emulated the successes seen in the civilian system.[83] The military's trauma system, the JTS, was established in 2004 and grew out of efforts in the US Army Institute of Surgical Research. Military surgeon leaders such as Colonel (Ret) John Holcomb, Colonel (Ret) Brian Eastridge, and Colonel (Ret) Don Jenkins set the stage for building on a trauma system that was informed by performance improvement with the patient at the epicenter. Given that casualties would travel 5000 to 8000 miles to reach the stateside hospital where they would undergo the majority of their care, one of the pillars of the military's trauma system was a weekly performance improvement video teleconference to discuss casualties among all the providers involved in the surgical, medical, and critical care air transport of the patients. This effort evolved over time, and CPGs were developed using the clinical casualty data. The stated mission of the JTS is "to provide evidence-based process improvement of trauma and combat casualty care, to drive morbidity and mortality to the lowest possible levels, and to provide evidence-based recommendations on trauma care and trauma systems across the Department of Defense (DoD)."

The three pillars of the JTS are described in the following sections.[84,85]

Analysis of Clinical Data from the DoDTR

Previously known as the Joint Theater Trauma Registry (JTTR), after the US Army Institute of Surgical Research program was adopted by all military services, it became the DoDTR. The registry was initially launched the same year as the JTS (2004), and it was modeled after a civilian trauma registry that was modified to capture military-relevant clinical data. Some of the strengths of civilian programs were the ability to compare outcomes to each other and over time in order to make incremental improvements and, when necessary, abandon nonbeneficial practices. The DoDTR electronically captures data in the following categories: demographics, mechanism/circumstances of injury, diagnosis, treatment, and outcomes. The patients included are comprehensive—regardless of US citizenship, military/civilian status, or presence of a wartime environment. This store of information is used for official activities, for monitoring of continuous metrics, and as a source for queries and research similar to the NTDB. Over 250 peer-reviewed publications, most relating to performance improvement, have used data from the DoDTR. As of 2018, the DoDTR has over 75,000 patient entries.

Trauma Care Delivery

The delivery of battlefield trauma care and en route combat casualty care occurs throughout the continuum of care and ideally with clinical data that get captured in the DoDTR. Standards for trauma care delivery are set by the JTS CPGs. Trauma care delivery has seen changes throughout the recent conflicts, as described earlier, in regard to the full-spectrum implementation of the principles of damage control to include early transfusion of blood products and whole blood. The refinement of battlefield trauma care is through process improvement and analysis of DoDTR data. Efforts are constantly underway to obtain increased granularity of the data with a specific interest in gathering detailed prehospital and en route care data.

Performance Improvement

Inherent in all of the JTS activities is the aspect of performance improvement (PI). Whereas this is a general motivation behind the comprehensive data acquisition and management activities of the DoDTR, specific activities and positions within the JTS are organized around PI. The development and monitored implementation of the CPG is one of the more visible PI initiatives. The effect of CPG use has been measured with reassuring results, including decrease in mortality from 32% before implementation to 21% after CPG implementation for the DCR guidelines addressed earlier.[70] A form of ongoing PI has been the use of the weekly video teleconference. This conference connects providers from every aspect of care including prehospital, the FST, CSH, Landstuhl, and military hospitals in the United States to discuss current critically ill patients to assess the quality of care and provide 360° follow-up. Also under the umbrella of PI are the constant education and readiness efforts of the JTS, including predeployment training to continuing medical education.

THE FUTURE OF THE JOINT TRAUMA SYSTEM AND THE BATTLEFIELD HEALTH CARE SYSTEM

The amount of clinical research, PI, and guideline development generated by the recent conflicts has been voluminous. The system of care that evolved and the JTS came to be without any mandate, law, or requirement. It was not until 2016 that a DoD Instruction (DoDI) required that a military trauma system (ie, the JTS) exist. This was codified in the NDAA of 2017 that established the JTS by law. From a historical perspective, it is a credit to the military leaders who had experience with the civilian trauma system that such a complex system of care grew into existence without any requirement to function. The JTS is now well established and known for establishing battlefield standards of care through the CPGs throughout the military, civilian, and international trauma communities.

Valuable lessons have been learned from prior conflicts, but the records of those lessons are too often incomplete; with the establishment of the JTS, it is hoped that this will be mitigated during future conflicts. Ultimately, preservation of the many costly lessons learned is contingent upon continuation of the systems and ideals instituted through the JTS and DoDTR. Future challenges faced by military surgeons include maintenance of trauma surgery skills in a period when the military tempo decreases and when the majority of cases done during peacetime do not support the needed trauma skill set. Elements of the new JTS are poised to address these issues by advocating (and requiring) military–civilian collaborations for trauma skill sustainment. As the military and civilian trauma systems continue to grow and collaborate with a focus on the preservation of valuable wartime lessons learned, both civilian and military members benefit; additionally, this focus on trauma readiness helps to ensure that in future conflicts the surgical force will be well poised to care for severely injured combat casualties.

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54

Organ Donation from Trauma Patients

Naeem Goussous • Joseph R. Scalea

KEY POINTS

- Solid organ transplantation is the treatment of choice for end-organ dysfunction in many organs and provides advantages in survival and improved quality of life.
- The use of organs from HIV- and hepatitis C virus–infected donors along with advancements in organ preservation will help to narrow the gap between organ supply and demand.
- The Organ Procurement and Transplantation Network is the organization that oversees these activities.
- Trauma and acute care surgeons play a pivotal role in early management of potential organ donors.
- The transplant team does not manage the donor in donation after cardiac death until the patient dies.
- Brain death is associated with a series of events that affect every organ system, but especially the cardiovascular and respiratory systems.
- The early adoption of aggressive donor management protocols will allow maximization of organ utilization and expansion of the donor pool.
- Unmanned aircraft systems are a promising modality for more efficient and safe organ transportation.

INTRODUCTION

Advances in the field of immunosuppression, organ preservation, surgical techniques, and critical care have resulted in significant improvements in the outcomes of transplant recipients (Fig. 54-1). Indeed, in most cases, solid organ transplantation provides a survival advantage and improved quality of life when compared with standard medical therapies. It has become the treatment of choice for end-stage kidney, liver, islet, intestine, heart, and lung failure. With improved medical treatments and improved transplant outcomes, increased demand for transplantation has resulted, yielding longer waitlists. As such, there is a woeful disparity between the number of patients who require transplants and the number of available donor organs. According to the Organ Procurement and Transplantation Network (OPTN) official website (http://optn.transplant.hrsa.gov), a new patient is being added to the transplant waiting list every 10 minutes, and on average, 20 people die on the waiting list every day. Currently in the United Sates, there are 124,370 patients on the transplant waiting list (Fig. 54-2).

Major innovations in organ replacement sciences have occurred in the past decade. Living and deceased donor allogenic transplants, however, remain the favored approach for patients with organ failure. Indeed, innovative approaches such as bioengineering and organ regeneration are still in experimental stages.[1] Xenotransplantation has seen a resurgence of interest, perhaps as a result of clustered regularly interspaced short palindromic repeats (CRISPR) technology, which allows investigators to manipulate multiple animal genetic loci simultaneously. In this way, several large public–private partnerships have suggested that the first pig-to-human transplants may occur in the next 5 years.

Recent innovations in infectious disease management have changed the field of transplantation as well. In 2013, the senate passed the HIV Organ Policy Equity (HOPE) act, which allowed organs from HIV infected donors to be transplanted into patients with HIV infection.[2] Several transplant centers have initiated HIV-positive to HIV-positive transplants under research protocols. The use of organs from HIV infected donors will have the potential to expand the donor pool with 500 to 600 extra donors annually. Further, transplant centers are also routinely transplanting hepatitis C virus (HCV)–positive donor organs into HCV-negative recipients. Although counterintuitive, the development of the new direct-acting antiviral medications for the treatment of HCV infection has resulted in transformation in the management of HCV with successful eradication of the virus in greater than 98%

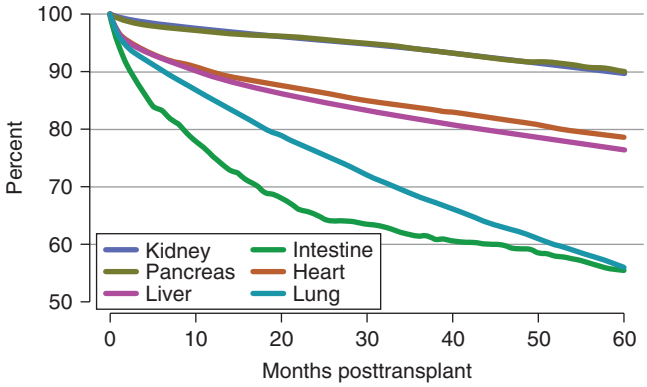

FIGURE 54-1 Patient survival among all transplant recipients, 2008 to 2012, by organ. (Reproduced from Organ Procurement and Transplantation Network. OPTN/SRTR annual report 2017. https://optn.transplant.hrsa.gov. Accessed June 2019.)

of patients. With the introduction of these highly effective anti-HCV medications, many centers are adopting policies to transplant organs from HCV-infected donors to HCV-negative recipients and treat HCV infection afterward. With this approach, the donor pool is expanded with good-quality organs.[3] The opioid epidemic has changed organ donation, as well. With the increasing number of narcotic overdoses, more and more organ donors are considered at increased risk for the transmission of viral infections (including HCV) by the US Public Health Service.

Exciting innovations in biotechnology have also advanced transplantation in recent years. Promising results with utilization of normothermic machine perfusion devices will allow more liberal use of marginal liver allografts (livers from older donors, fatty livers, and livers recovered from donors after circulatory arrest), thereby increasing the pool for all forms of organ transplantation.[4] Beyond devices for prolonging the time between explant and implant, recent exciting data suggest that organs may be able to move unmanned using remotely piloted or unmanned vehicles (ie, drones). This is of

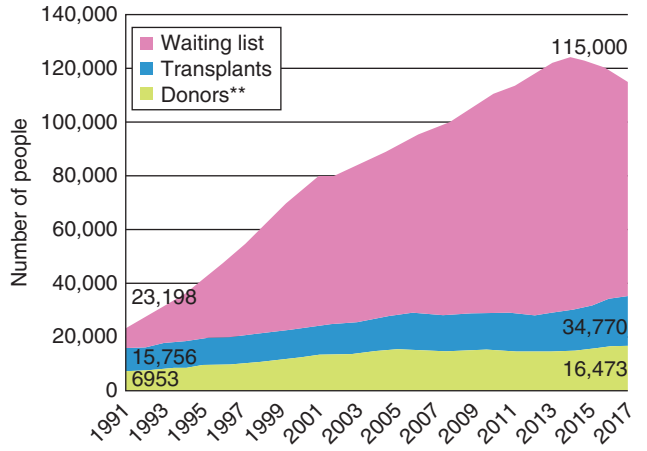

FIGURE 54-2 Total number of patients on the waiting list in the United States and the number of donors and transplants from 1991 to 2017. **Includes both living and deceased donors. (Reproduced from Organ Procurement and Transplantation Network. https://optn.transplant.hrsa.gov. Accessed June 2019.)

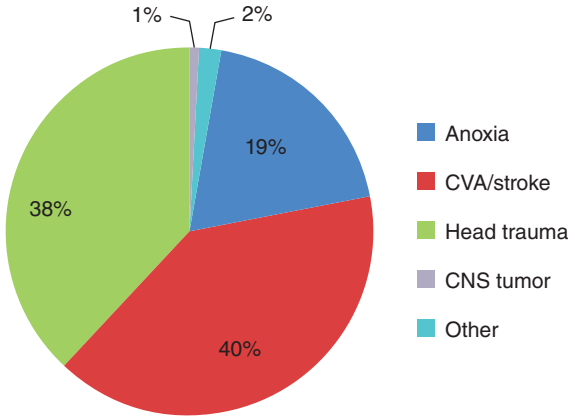

FIGURE 54-3 Pie chart demonstrating categories of deceased organ donors by cause of death in the United States from 1994 to 2014 (total number of donors = 142,822; donors with unreported cause of death are excluded). CNS, central nervous system; CVA, cerebrovascular accident. (Reproduced from Organ Procurement and Transplantation Network. http://optn.transplant.hrsa.gov. Accessed February 4, 2020.)

substantial potential benefit to the field because current shipment practices are expensive, time insensitive, and potentially unsafe for transplant recovery teams. Taken together, recent innovations are improving the delivery of transplant care.

INJURY AND DONATION

Traumatic brain injury is the second most common cause of death leading to cadaveric solid organ donation, closely following cerebrovascular accidents/stroke (Fig. 54-3). Clinicians caring for severely injured patients play a key role in the initiation and implementation of the organ donation process. Early recognition of potential organ donors is critical to maximizing the available pool of donor organs and the number of transplantable organs per donor. It is essential for those caring for potential organ donors to be knowledgeable about the criteria and process for declaring brain death and the physiologic effects of brain death. Familiarity with local organ procurement organizations (OPOs) is important because of the vital role they play in counseling the families of potential organ donors and coordinating the transplant process. Lastly, following the declaration of brain death, treatment priorities aimed at minimizing brain injury require adjustment. Afterward the priority shifts to providing physiologic support to maintain perfusion of potentially transplantable organs, and timely initiation of this support is crucial to increasing the probability of successful transplantation.

THE INFRASTRUCTURE AND ORGANIZATIONS INVOLVED IN ORGAN DONATION

Under the National Organ Transplant Act, the US Congress established the OPTN.[5] The OPTN is a unique public–private partnership linking professionals involved in the organ

donation and transplantation system. The OPTN is governed by the United Network for Organ Sharing (UNOS). UNOS is a private nonprofit charitable organization contracted by the Health Resources and Services Administration of the US Department of Health and Human Services to develop organ transplantation policy. UNOS facilitates organ transplantation by organizing the medical, scientific, public policy, and technologic resources required to maintain an efficient national transplantation system. UNOS is responsible for developing recipient priority policies and for managing the national transplant waiting lists. UNOS maintains the national transplant database, plays a very important role in raising public awareness of the importance of organ donation, and helps to keep patients informed about transplant issues and policy.

To alleviate the shortage of organs available for transplantation, the Revised Uniform Anatomical Gift Act requires OPOs and donor hospitals to have the necessary policies and procedures in place to preserve the option of donation for all patients and their families.[6] In an effort to maximize donation opportunities, the American College of Surgeons (ACS) qualitatively evaluates each hospital's organ donation practices during the trauma center verification process.[7] Specifically, the ACS requires verified trauma centers to establish a relationship with an OPO, develop policies and clinical triggers for notification of the OPO about patients with the potential for neurologic death, have a formal process to review organ donation rates, and implement protocols for the declaration of neurologic death. Organ donation, allocation, and procurement require a closely coordinated and complex series of efforts. In the United States, this process is coordinated by independent local OPOs. The United States is split into 11 UNOS regions and is covered by 58 OPOs. OPOs are responsible for two main functions within their designated service area as follows: (1) increasing the number of registered donors; and (2) coordinating the donation process when actual donors become available. OPOs employ specially trained professionals who assist with the following: (1) evaluation of potential organ donors; (2) the declaration of brain death; (3) counseling of donor family members; (4) management of the donor; (5) organ allocation; and (6) the procurement process. When an organ donor is identified, the local OPO serves to ensure that brain death has been established and assists in obtaining consent for organ donation. Thereafter, coordination of organ placement and the procurement of the organs are facilitated by the OPO. For organ allocation, UNOS maintains a centralized computer network, UNet, which links all OPOs and transplant centers in a secure, real-time environment using the Internet. Each organ waiting list incorporates specific criteria to establish individual patient ranking on the list. In general, waiting lists incorporate factors such as geography, patient's blood type, waiting time, severity of illness, and human lymphocyte antigen matching in cases of kidney allocation.

Trauma surgeons play an important role in organ donation. It has been shown that hospitals with trauma surgeons on their organ donor councils had a significantly higher number of donors per trauma admissions as well as significantly more donors per admissions.[8]

UNOS CRITICAL PATHWAY FOR ORGAN DONORS

UNOS has developed the Critical Pathway for management of organ donors, which is available through local OPOs and UNOS.[9] Preliminary data from a pilot study using this pathway showed an increase in the number of organs procured per donor. The Critical Pathway has five distinct but partially overlapping phases as follows:

Phase I (referral): When a patient with a severe brain injury is identified as a potential organ donor, critical care staff should initiate the Critical Pathway. This step establishes contact between the hospital and the local OPO and initiates referral before the potential organ donor becomes brain dead.

Phase II (declaration of brain death and acquisition of consent): Once the potential donor is confirmed brain dead, the patient's choice about organ donation is evaluated through the donor registry (usually through the motor vehicle registry) and the family is provided with this information. If no first-person consent is found, the family is approached about the option of organ donation. First-person consent supersedes a family declining the option of donation in many states and OPOs in the United States.

Phase III (donor evaluation): After consent for organ donation is obtained or confirmed through a donor registry, donor evaluation and management protocols are initiated.

Phase IV (donor management): As the donor is being evaluated, optimal management needs to be started simultaneously to achieve adequate perfusion and oxygenation of organs.

Phase V (organ recovery): Organ procurement takes place after potential recipients are identified. Various procurement teams work together to maximize organ recovery.

IDENTIFYING A POTENTIAL ORGAN DONOR

Federal law requires that every death in a hospital be called in to their respective OPO, and the OPO coordinators evaluate every call to assess suitability for donation. All patients who have suffered severe brain injuries and are either brain dead or likely to progress to brain death should be considered for organ donation regardless of their age, underlying cause of illness, and overall social history.

According to the Centers for Medicare and Medicaid Services (CMS), hospitals and respective OPOs must agree upon "triggers" to identify potential brain dead donors, although CMS does not specify these triggers. For instance, some OPOs consider an intubated patient with a Glasgow Coma Scale score of 5 or less a potential organ donor. If a patient meets initial criteria, the donation case moves on

TABLE 54-1: Absolute Contraindications to Organ Donation

- Active malignancy with evidence of spread outside affected organ
- Primary intracerebral lymphoma and secondary intracerebral tumors
- Melanoma
- Active hematologic malignancy
- Tuberculosis: active and untreated
- West Nile virus infection
- Coroner exclusion

to the next step in the process, which is referral to the local OPO. Although perceived contraindications to donation may exist, they should be discussed with a representative of the local OPO before concluding that a given patient is not a candidate for organ donation (Table 54-1). Bacteremia, fungemia, and high-risk social history are not absolute contraindications. For increased-risk donors, a nucleic acid test is performed to detect recently acquired HIV, HCV, or hepatitis B virus (HBV) infections when routine serology tests are negative. Sometimes, donors with HCV, HBV, and HIV are considered suitable for organ donation. For instance, an otherwise normal liver or a kidney from a donor with HCV infection can be transplanted into an HCV-infected recipient as previously noted. Trauma to the chest and/or abdomen is not uncommon in potential organ donors. Appropriate surgical management is required to control bleeding and prevent complications such as a pneumothorax. Typical interventions include insertion of a thoracostomy tube, laparotomy, or splenectomy. A retroperitoneal hematoma, hepatic and splenic lacerations, and an open abdomen are not contraindications to utilization of an abdominal organ; however, such circumstances demand careful evaluation of organs at the time of procurement. A computed tomography (CT) scan using intravenous contrast is important in the assessment of severity of injury and vascular integrity.

The physician caring for a potential organ donor is responsible for notifying the local OPO of such patients. All OPOs employ personnel who are responsible for advising health care providers on the suitability of an individual patient for organ donation. Communication with local forensic authorities is extremely important. The OPO will contact the medical examiner or coroner in order to obtain permission to proceed with organ donation. Once a donor is identified, the OPO is responsible for obtaining family consent for organ donation. Organ procurement specialists are trained in counseling families about the importance and process of organ donation, and it is advisable to refer families to these specialists when potential organ donation is discussed. Also, these individuals perform a careful review of the potential donor's social and past medical history. The circumstances leading to brain death are very important, as is any history of the occurrence and duration of cardiopulmonary arrest. Screening also includes an extensive laboratory and serologic evaluation to exclude chronic diseases and transmissible infections. A donor profile is then generated and includes current hemodynamics as well as an assessment of current organ function. The assessment of organ function is individualized to the donor based on the donor profile, the specific organs under consideration, and the level of medical support required to maintain the donor. The overall profile that is generated is crucial for transplant physicians who must evaluate the suitability of a given organ donor for the individual recipient.

DECLARATION OF BRAIN DEATH

Brain death occurs when a person has an irreversible, catastrophic injury which causes total cessation of brain function. Some causes of brain death include (but are not limited to) the following: (1) trauma to the brain (eg, severe head injury caused by a motor vehicle crash, gunshot wound, fall, or blow to the head); (2) cerebrovascular accident (eg, stroke or rupture of an aneurysm); (3) anoxia (eg, respiratory arrest, drowning); and (4) brain tumor. Ethical standards in the United States mandate that all organ donors must be declared dead before organ donation can proceed. Brain death must therefore constitute a sufficient basis on which to declare a person legally dead. The vast majority of cadaveric organs are procured from donors whose deaths are declared on the basis of brain death. Consequently, cadaveric organ donation is dependent on the ability to reliably determine that a patient is brain dead. Unfortunately, many clinicians remain poorly informed about brain death and how it is defined. The current concept of brain death used in the United States is based on guidelines published in 1981 by the President's Commission for the Study of Ethical Problems and adopted under the Uniform Determination of Death Act (Table 54-2).[10] This act states that death has occurred when there is irreversible cessation of all functions of the brain including the brainstem. Each state government has adopted these guidelines in legislating local criteria for the determination of brain death. The qualifications and number of physicians who must agree on the diagnosis of brain death in order to legally declare a patient brain dead vary considerably among different states.

TABLE 54-2: Defining Brain Death

Preconditions

Coma with known cause

Documentation of irreversible structural brain injury

Exclusions (preclude diagnosis of coma)

- Hypotension
- Hypothermia
- Hypoxemia
- Drug intoxication
- Metabolic derangements

Tests

Absence of brainstem function; pupillary, corneal, oculovestibular, gag, and cough reflexes absent

Apnea (strict definition)

Electroencephalogram not indicated

Some states require two separate declaration procedures with a defined time interval between the two examinations. Some states require that two separate physicians make the declaration. In other states, a single physician may declare a patient brain dead on the basis of one examination. In no case can the declaring physician take part in the recovery or transplantation of organs from the donor. Most hospitals have established policies within state guidelines for physician qualifications required to make the diagnosis of brain death. Physicians caring for these patients should be aware of local requirements and hospital guidelines in order to facilitate the declaration process and allow termination of care for brain dead patients who will not be organ donors.

Symptoms that support the diagnosis of brain death are the absence of brainstem reflexes, absence of cortical activity, and demonstration of the irreversibility of this state.[11] Therefore, in order to declare brain death, there must be a proof of the cause of the brain injury; otherwise, the irreversibility requirement cannot be met. Secondly, all reversible causes of coma must be excluded. Causes of reversible coma include hypothermia, hypoxia, hypoglycemia, hyperglycemia, uremia, hepatic failure, Reye syndrome, hyponatremia, hypercalcemia, myxedema, adrenal failure, and central nervous system (CNS) depressants. The presence of CNS-depressing agents such as narcotics, sedatives, anticonvulsants, anesthetics, and alcohol must be assessed. If any of these agents are present, confirmatory testing is usually required to declare brain death.[12] In the brain dead patient, all cranial nerve functions will be absent. The absence of brainstem reflexes must be confirmed by careful neurologic examination. Neuromuscular conduction must be intact in order to allow adequate examination; consequently, the presence of neuromuscular blocking agents must be excluded.

The most definitive finding supporting the diagnosis of brain death is the presence of apnea. The apnea test remains one of the most important parts of the neurologic evaluation of potential organ donors.[13] To perform a reliable apnea test, the $Paco_2$ is normalized to 40 mm Hg. The patient is preoxygenated with 100% oxygen for at least 5 minutes. The patient is then disconnected from the ventilator and placed on 100% oxygen delivered passively to the endotracheal tube via a T-piece at 8 to 12 L/min. The $Paco_2$ is allowed to rise to 60 mm Hg, confirmed by a blood gas drawn after approximately 10 minutes. If hemodynamic instability occurs, the patient should be immediately returned to mechanical ventilation and a blood gas should be drawn to assess the $Paco_2$. If there is any evidence of respiratory activity, the patient is not brain dead and should be immediately returned to the ventilator. If there is no evidence of spontaneous respiratory activity, the $Paco_2$ has reached 60 mm Hg, and the pH is acidotic, apnea is established and is strongly supportive of brain death.

In many cases, confirmatory testing must be performed in addition to a careful neurologic examination in order to firmly establish the diagnosis of brain death. Patients with cervical fractures above the level of C4 may not have intact diaphragmatic function, precluding a reliable apnea test. Apnea testing is also unreliable in patients with overdoses of substances that depress respiratory drive such as alcohol, antiseizure medications, and sedatives. Hemodynamic instability during apnea testing will also preclude the establishment of the diagnosis of brain death on the basis of apnea. In other cases, local requirements and/or hospital policy may dictate the use of additional confirmatory testing. Confirmatory tests may also be useful in demonstrating a clear etiology for brain death or severe anatomic damage and can decrease the observation period required to establish the diagnosis of brain death.[12]

Confirmatory tests of brain death include an electroencephalogram (EEG) and methods of demonstrating the absence of cerebral blood flow. EEGs are not entirely reliable and are now rarely used for this purpose with the exception of brain death determination in young infants. Demonstration of the absence of cerebral blood flow is the most common confirmatory test currently in use. Methods used to make this determination include cerebral angiography, Doppler ultrasound scanning, and radionuclide cerebral blood flow scanning. The latter two methods are noninvasive, low risk, relatively inexpensive, and more readily available than cerebral angiography. These tests are highly accurate in verifying the absence of cerebral blood flow and are useful in reducing the time required to establish the diagnosis of brain death. Conversely, an examination that indicates continued cerebral blood flow does not necessarily exclude the diagnosis of brain death. Uncommonly, cerebral blood flow may persist despite brain death due to testing before increasing intracranial pressure completely shuts down flow, skull pliability in infancy or in the presence of decompressing fractures, ventricular shunts, ineffective deep brain flow, reperfusion, brain herniation, jugular reflux, the presence of emissary veins, and pressure injection artifacts.[14]

Appropriate documentation of brain death is very important in facilitating organ donation for a brain dead patient. The diagnosis of brain death must be documented in writing, and it must be unequivocal. The circumstances leading to brain injury, the specific findings of the neurologic examination, and the results of any confirmatory tests should be clearly recorded. Lastly, the date and time of the declaration of brain death must be noted before OPO personnel may obtain the permission of local authorities and the consent of the potential donor's family. Despite the presence of evidence indicating a person's desire to be an organ donor, family consent for donation must be obtained. Family refusal is the most common reason that otherwise suitable donors do not become organ donors. If consent for donation is declined, appropriate testing and documentation of brain death are necessary in order to initiate the withdrawal of care.

The most common form of deceased donation is donation after brain death (DBD). For DBD, donors are brought to the operating room intubated and frequently on vasoactive medications. Surgical recovery teams confirm brain death testing, brain death documentation, laboratory values, and blood group. Thereafter, the recovery teams work in concert with recipient teams and hospitals to confirm the logistics of presumed aortic cross-clamp and organ recovery times. This

is important because organs need to be transplanted expediently to reduce cold ischemia time. In some patients with catastrophic brain injuries, the intracranial pressure (ICP) is maintained and brain death does not occur; however, in these patients, the severity of neurologic injury is such that no meaningful neurologic recovery is anticipated. As such, regardless of the disease process, families may elect to withdraw life support because of a hopeless prognosis. In these cases, patients can still donate under a practice known as donation after cardiac death (DCD).

DCD can be controlled or uncontrolled. Controlled DCD involves planned withdrawal of ventilatory and organ perfusion support in the face of catastrophic illness, usually in the operating room. Uncontrolled (currently not practiced in the United States) DCD involves unexpected cardiopulmonary arrest and/or unsuccessful resuscitation (Maastricht classification I, II, or IV).[15] Controlled DCD offers the patient and the family the opportunity to donate organs when criteria for brain death are not met prior to cardiac death. The procurement/transplant team plays no role in whether or when support will be withdrawn.

PHYSIOLOGIC CONSEQUENCES OF BRAIN DEATH

Brain death is usually associated with increased ICP, causing physiologic responses, mainly in the cardiovascular and respiratory systems.[16] Disruption of normal autonomic regulation may lead to profound vasodilation and hypotension. In turn, decreased organ perfusion is injurious to potentially transplantable organs.

Cardiovascular

Increased ICP is associated with marked sympathetic stimulation (catecholamine storm), resulting in arterial hypertension, intense vasoconstriction, raised systemic vascular resistance, tachycardia, and direct myocardial depression.[16] Echocardiographic evidence of myocardial dysfunction is seen in 40% of brain dead donors being considered for heart donation.[17] After the catecholamine storm, there is a loss of sympathetic tone causing peripheral vasodilatation and hypotension, resulting in hypoperfusion of all organs and leading to significant injury that may preclude organ donation if not corrected in a timely fashion.

Respiratory

Respiratory dysfunction associated with brain death will have profound effects on all other organ systems due to hypoxia. Impaired gas exchange is common in brain dead patients who have required mechanical ventilation for any length of time. Pulmonary infection and/or aspiration injury frequently occur in potential organ donors. Neurogenic pulmonary edema is another cause of impaired gas exchange that may accompany brain death.[18] Myocardial dysfunction may also contribute to lung dysfunction due to increases in left atrial pressure, thereby potentiating pulmonary edema. Hypoxemia will further impair myocardial function, leading to a precarious hemodynamic state.

Endocrine

A number of hormonal changes occur after brain death and reflect anterior and posterior pituitary failure. There is early depletion of antidiuretic hormone and development of diabetes insipidus in almost 80% of brain dead organ donors. This is characterized by inappropriate diuresis, severe hypovolemia, hyperosmolality, and hypernatremia. These changes may exacerbate neurogenic pulmonary edema and lead to tissue hypoperfusion. If not managed appropriately, diabetes insipidus will have a profound effect on fluid and electrolyte balance. A rapid decline in free triiodothyronine (T_3) is seen after brainstem death. Insulin levels fall after brain death, leading to a decrease in intracellular glucose concentration, the development of an energy deficit, and a shift toward anaerobic metabolism and acidosis. Significant decreases in cortisol levels occur after brain death as a result of a decreased release of the stimulating factor adrenocorticotropic hormone (ACTH) from the anterior pituitary. The administration of exogenous corticosteroids has been associated with stabilization of organ function.

Other interrelated effects of brain death include endothelial activation, systemic release of proinflammatory cytokines, complement activation, and consumptive coagulopathy. Without appropriate intervention, brain death is followed by severe injury to all other organs, and circulatory collapse will usually occur within 48 hours.

MANAGEMENT OF THE DONOR

Prior to the declaration of brain death, the treatment of patients with severe brain injury is directed at controlling ICP and preserving cerebral perfusion pressure in order to limit secondary brain injury. Once the diagnosis of brain death is established and a patient is recognized as a potential organ donor, treatment priorities must shift in favor of preserving the function of potentially transplantable organs. Preservation of organ function requires maintenance of adequate organ perfusion. The most common derangements requiring interventions are the previously mentioned hypothermia, hypotension, and diabetes insipidus. Because of the profound effects on the cardiovascular system, invasive hemodynamic monitoring is essential to guide the appropriate resuscitation of brain dead patients. Central venous access should be obtained to allow measurement of volume status and to provide a reliable means of administering vasoactive drugs. Arterial access is required to facilitate continuous monitoring of blood pressure and frequent measurement of acid–base status, gas exchange, and serum biochemical parameters.

One of the strategies with proven success in increasing organ donation is utilization of an aggressive donor management (ADM) protocol, by decreasing the number of donors

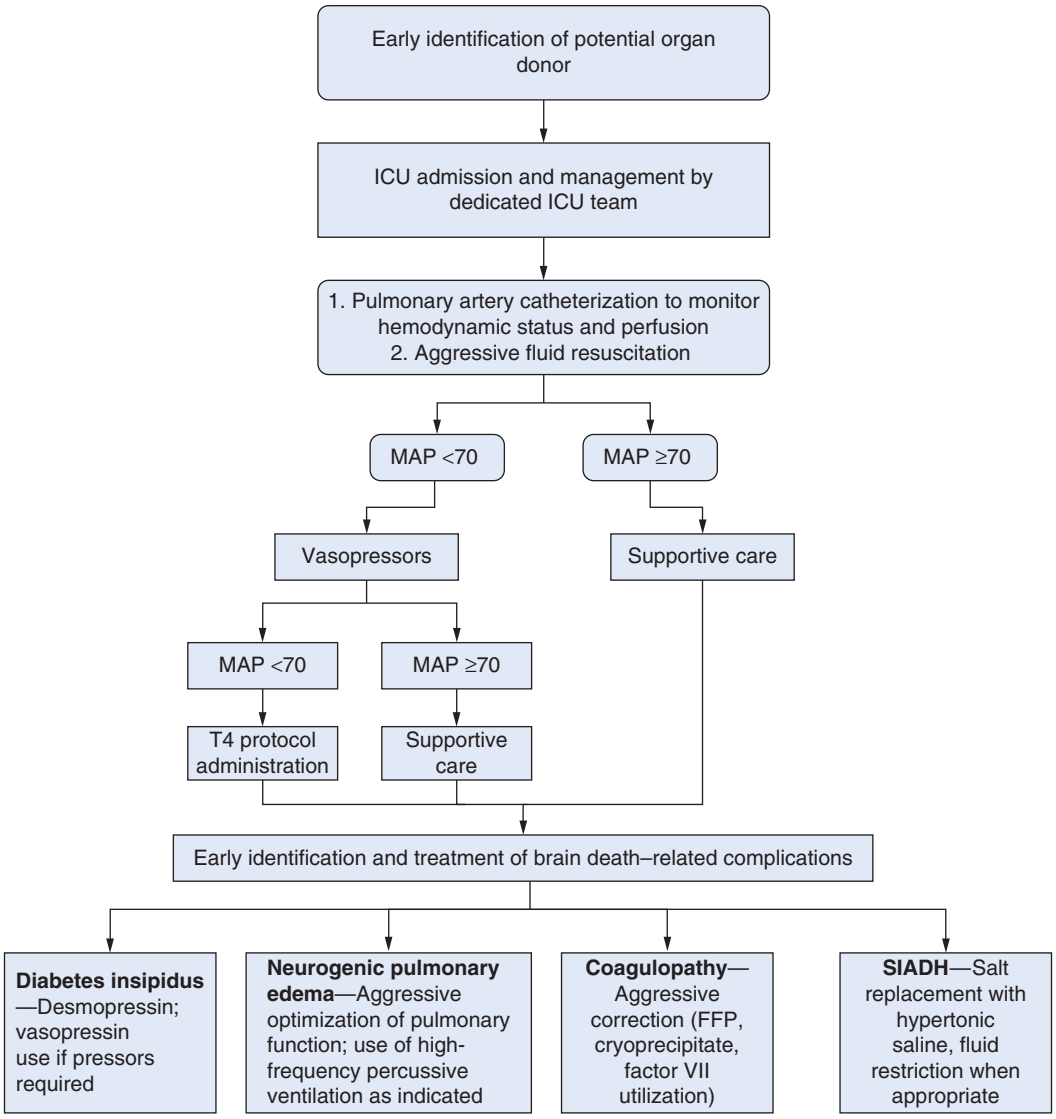

FIGURE 54-4 Protocol for aggressive donor management. FFP, fresh frozen plasma; ICU, intensive care unit; MAP, mean arterial pressure; SIADH, syndrome of inappropriate antidiuretic hormone; T4, thyroxine. (Reproduced with permission from DuBose J, Salim A. Aggressive organ donor management protocol. *J Intensive Care Med*. 2008;23(6):367-375.)

lost as a result of cardiovascular collapse.[19] The components of ADM include the following (Fig. 54-4): (1) early identification of potential donors; (2) admissions and management by a dedicated team; and (3) early and aggressive resuscitation with fluids, vasopressors, and hormone therapy.

Hypovolemia is common in brain dead patients due to vasodilation and, in some cases, blood loss as a result of trauma or fluid loss due to diabetes insipidus. In general, patients should be resuscitated with crystalloid solutions and appropriate blood products based on measurements of hematocrit and coagulation status. Resuscitation should be guided by frequent monitoring of central venous pressure or pulmonary capillary wedge pressure. Avoiding excessive fluid loading in donor management is associated with increased numbers of transplantable lungs and pancreas.[20] Body temperature must be monitored and should be maintained above 36°C using passive warming as needed. Urine output should be maintained at 2 mL/kg/h. Renal dose dopamine (3–5 mcg/kg/min) is recommended for nearly all donors unless higher doses are required to maintain perfusion. Vasopressin should be infused to treat diabetes insipidus when present, and fluids should be adjusted on the basis of electrolyte measurements. Serum sodium and osmolarity should be kept as normal as possible. Replacement fluids should be based on measurements of serum electrolytes and urine output. Frequent serum glucose measurements are required to facilitate treatment of hyperglycemia.

Inotropic support should be introduced to support blood pressure if initial volume resuscitation fails to restore adequate perfusion pressure.[21] Dopamine is usually the first agent of choice, as noted earlier. Pure vasoconstrictors such as norepinephrine should be avoided whenever possible due to

deleterious effects on myocardial and splanchnic perfusion. Dobutamine is frequently added to increase cardiac output. If these agents are not effective at restoring hemodynamic stability, an epinephrine infusion should be initiated and titrated to the lowest dose required to achieve adequate support. The use of high doses of norepinephrine in donors is associated with increased cardiac graft dysfunction, particularly right ventricular performance, and higher early and late mortality in the recipients. Thyroxine (T_4) or T_3 infusions are commonly used due to their inotropic effects and potentiation of myocardial catecholamine sensitivity.[22-24]

Ventilator management in potential organ donors is guided by the respiratory function of the donor. Pulmonary injury is common in brain dead patients, and all attempts should be made to preserve the possibility of lung donation. As mentioned previously, fluid overload must be avoided and infusions should be guided by measurements of central venous pressure. Chest x-rays should be obtained to assess pulmonary expansion, guide appropriate intervention to counteract atelectasis, and assess the suitability for transplantation. A nasogastric tube should be placed and kept on suction in order to minimize gastric distension and the risk of aspiration. An endotracheal tube with an internal diameter of 7.5 mm or larger should be inserted whenever possible in order to accommodate a therapeutic size fiberoptic bronchoscope. This will assist in the evaluation of potential lung donation and will facilitate clearing secretions to optimize lung expansion. Excessive mean airway pressures should be avoided. Tidal volumes and physiologic levels of positive end-expiratory pressures (PEEP) should be maintained to provide adequate lung expansion and minimize atelectasis. High concentrations of inspired oxygen are injurious to the lungs and should be avoided. The inspired oxygen concentration should be titrated to maintain an oxygen saturation of 95%. In patients with significant pulmonary injury, lung donation is unlikely; therefore, increased levels of PEEP and higher inspired oxygen concentrations may be required (Table 54-3).

TABLE 54-3: Donor Management Goals

- Mean arterial pressure: 60–100 mm Hg
- Central venous pressure: 4–10 mm Hg
- Ejection fraction: 50%
- Vasopressors: ≤1 and low dose[a]
- Arterial blood gas pH: 7.3–7.45
- Pao_2:Fio_2: >300
- Serum sodium: 135–155 mEq/L
- Blood glucose: <150 mg/dL
- Urine output: 0.5–3 mL/kg/h over 4 h

[a]Low dose of vasopressor was defined as dopamine ≤10 mcg/kg/min, neosynephrine ≤60 mcg/kg/min, and norepinephrine ≤10 mcg/kg/min.

Source: Data from Malinoski DJ, Patel MS, Daly MC, Oley-Graybill C, Salim A; UNOS Region 5 DMG Workgroup. The impact of meeting donor management goals on the number of organs transplanted per donor: results from the United Network for Organ Sharing Region 5 prospective donor management goals study. *Crit Care Med.* 2012;40:2773-2780.

ORGAN PROCUREMENT

The actual process of multiorgan procurement occurs in the operating room environment under the usual sterile conditions. Representatives of the local OPO will coordinate the use of the operating room with various transplant procurement teams involved with a particular donor. Frequently, this procedure may involve several teams whose members are operating together for the first time. In addition, the initial phases of the recipient operations for heart and lung recipients are commonly carried out simultaneously with the donor procurement procedure in order to minimize graft ischemia time.

The logistics of organizing the donor operating room fall to the OPO. Frequently, the timing of the organ procurement is dependent on the wishes of the donor family. Donor operating room timing is also informed by the availability of the recovering surgeons as well as the needs of the transplant recipients. For example, a heart recipient with a ventricular assist device may have operative challenges that limit the timing of both the donor and the recipient operating room procedures. Operating room timing may also be affected by the availability of rooms and staffing, particularly at smaller hospitals.

Procurement procedures may be as short as 30 minutes for a DCD in which kidneys alone are recovered or as long as 4 to 5 hours when both abdominal and thoracic organs are recovered. Unexpected delays at the donor facility or any extra tests or procedures needed (eg, liver biopsy, bronchoscopy, and/or blood gas results for lung transplantation) may affect the length of the donor procedure as well. Beyond medical and technical delays, potential intraoperative organ declines by a procuring team and the need to reallocate the organ to different teams may also occur, adding time to recovery and transport.

Before the start of the recovery procedure, the donor history, labs, and imaging are reviewed and the death note is reviewed by procuring teams, as previously noted. The instruments needed for a procurement include a major abdominal tray with a large sharp Balfour retractor, an electrical sternal saw or a Lebsche knife, and a Finochietto retractor. An anesthesiologist is required to provide physiologic support to the donor during the procurement procedure. The donor patient is prepped and draped to allow an incision from the sternal notch to the pubis. In this manner, the thoracic and abdominal organs can be evaluated and procured simultaneously. In all cases, the final determination of organ suitability for transplantation is made after visual inspection and manual palpation of the organ and other viscera to rule out any malignancy. Biopsy of any suspicious lesions should be performed, and pathologic evaluation of frozen sections must be confirmed before the recipient transplant operation is initiated. Similarly, biopsies of liver and kidneys may be required to evaluate the quality of organs in some donors.

In the case of brain dead donors, the intended organs are prepared for rapid removal after exsanguination of the patient. For this purpose, the important vascular structures of the

respective organs are identified and the organs are mobilized. The patient is systemically anticoagulated with a large dose of heparin. Appropriate vascular cannulas are inserted in the abdominal and thoracic vessels to allow rapid flushing of the organs being procured. There are a number of preservation solutions in use, and these vary according to the organ being procured and the preference of the procuring institution. All preservation solutions are kept at approximately 4°C because hypothermia is a key element of organ preservation. When all members of the procuring teams are prepared, the superior vena cava, ascending aorta, and supraceliac aorta are clamped. The inferior vena cava is transected at the cavoatrial junction, and blood is suctioned from the open inferior vena cava, exsanguinating the patient. The left atrium is vented in order to prevent left ventricular distension and back pressure on the pulmonary vascular bed. Preservation fluids are infused through the previously placed cannulas, flushing the procured organs of blood and rapidly lowering their temperature. Topical ice is applied, augmenting rapid cooling. Thereafter, the heart and lungs are usually removed first. Donor hepatectomy is then carried out, followed by removal of the kidneys. Variations in this sequence are required when the pancreas and/or small bowel are also procured. The iliac vessels and occasionally segments of the descending thoracic aorta are then removed to provide additional vascular conduits that are sometimes necessary for complex vascular reconstructions at the time of organ implantation. All organs are inspected on the back table to ensure that no surgical damage has occurred. Finally, the organs are placed in sterile containers containing cold preservation solution. The containers are then packed in ice in preparation for transport to the location where transplantation will occur. Once all solid organs are removed, any additional tissues approved for donation are removed.

In case of procurement from DCD, hospitals have their own protocols for withdrawal of life support and declaration of cardiac death. In principle, the withdrawal of life support occurs in the operating room. Heparin is administered prior to withdrawal of support, except in the rare case when it might be expected to hasten death and/or is prohibited per local procurement protocol. Morphine and/or other analgesics may be given at the discretion of the patient's treating care team if the purpose is to minimize discomfort according to accepted end-of-life protocols. The initial required instruments are set up on a Mayo stand to expedite the first steps of the procurement, which involves entry into the abdominal and thoracic cavities and gaining access to the abdominal aorta. Procurement team members do not participate in decisions regarding the use of such agents and/or declaration of death. Members of the procurement team are not present in the operating room at the time of withdrawal of support and until the declaration of death. During this period, the OPO coordinators document hemodynamic measurements every minute. After cessation of cardiorespiratory function, the patient's treating physician declares death. Subsequently, a waiting period, typically 2 to 5 minutes, is observed prior to initiation of procurement to ensure that cardiac activity does not resume. After this waiting period, the procuring teams rapidly enter the abdominal and thoracic cavities and cannulate the respective blood vessels for flushing the organs with preservation fluid. Following this, the procurement proceeds similar to that in brain dead donors. When performed, premortem cannulation of the femoral artery and femoral vein prior to withdrawal of support decreases the rush inherent with the rapid recovery technique and may decrease warm ischemia time. If the patient has not been declared dead within the time frame stipulated by the local procurement protocol (usually 60 minutes), then the donation is aborted and the patient is returned to the intensive care unit for comfort care.[25]

Some centers use premortem cannulation in conjunction with postmortem extracorporeal membrane oxygenation (ECMO) to restore the flow of warm oxygenated blood to the intra-abdominal organs during the interval between death and organ procurement, possibly improving graft function. This is done by cannulation of the femoral artery and vein on one side for the ECMO circuit and the contralateral groin is cannulated with an intra-aortic balloon occluder that is placed in the descending thoracic aorta above the diaphragm to prevent cardiac reanimation. The cannulation and confirmation of intra-aortic balloon placement are done in the intensive care unit premortem. After withdrawal of life support and after the mandatory waiting period is observed, the balloon occluder is inflated and the ECMO circuit is initiated. The donor is then transferred to the operating room where a standard organ procurement is performed and the organs are flushed in a retrograde fashion with cold preservation solution via the arterial cannula.[26]

ORGAN PRESERVATION AND SHIPMENT

Organ shipment in the United States is arranged by OPOs. Organs are shipped by ground transport, helicopter, and airplane, depending on the time the organ needs to arrive at the transplant center. Culturally, hepatic and thoracic surgeons recover their recipient's organs and subsequently perform the transplant procedure as well. As a result of redistricting processes, however, this cultural norm may be changing because of the increased frequency with which organs are traveling further distances.

Historically, organs have been preserved using "cold storage," wherein organs are placed in preservation solution and packed in ice. This process has the effect of reducing the tissue metabolic rate of the organ and prolonging the organ's "shelf life." With normothermic perfusion, organs are placed on special pumps that perfuse the organ with warmed, oxygenated blood after recovery and prior to transplantation. This practice mimics posttransplant physiology and may prolong the allowable time between explant and implant. There are several devices in use that have shown promise in hepatic and kidney transplantation.[4] Hypothermic perfusion is performed as well. In this scenario, kidneys are perfused with a hypothermic solution rather than being placed in static cold

storage. Of interest, these organs have lower rates of delayed graft function after transplantation.[27]

With changing organ allocation systems, solid organs are moving further distances and experiencing longer cold ischemia times in many cases. This has led to several problems, including the challenge of finding on-demand small aircraft. To this end, investigators have recently begun testing organ shipment using unmanned aircraft systems (UAS). The benefits of UAS organ shipment would be reduction of cold ischemia time, improved safety for the organ recovery team (only the organ travels, not the team), decreased cost, improved outcome, and better access to organ location and estimated time of arrival. While experimental, these technologies have shown great promise.[28]

CONCLUSION

Organ transplantation benefits thousands of people every year in the United States. There is, however, a critical shortage of organs available for lifesaving transplantation with no increase in the numbers of deceased donors in recent years. A significant number of deceased donors in the United States are victims of trauma. Therefore, health care providers who care for injured patients will continue to play an important role in the identification and critical care management of potential organ donors. It is imperative that trauma surgeons and personnel be aware of the critical steps involved in organ donation and advanced protocols in the management of potential organ donors.

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Rehabilitation

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KEY POINTS

- The fundamental principles of rehabilitation are founded on mitigating and preventing (when possible) the effects of immobility.
- Daily range-of-motion, flexibility, and muscle strengthening exercises help maintain the appropriate balance of muscle activity across joints and can be used to both prevent and treat muscle atrophy, disuse osteoporosis, and contracture formation.
- Immobilized patients have a reduction in blood volume, venous return, and stroke volume.
- Decreased gastric transit time in immobilized patients may cause symptoms of gastroesophageal reflux, regurgitation, and heartburn.
- To determine pain level, most health care organizations use a visual analog scale typically ranging from 0 (no pain) to 10 (worst imaginable pain).
- Approximately 12,000 spinal cord injuries occur nationally each year, and approximately 259,000 trauma survivors are living with spinal cord injuries in the United States.
- Spinal cord injuries are classified by their severity and level of injury, using the American Spinal Injury Association Standard Neurological Classification Worksheet.
- Between 25% and 80% of patients with a spinal cord injury eventually develop a decubitus ulcer.
- It is estimated that 19% to 23% of military service members who deploy overseas sustain a concussion or mild traumatic brain injury.
- Vascular occlusive diseases cause over 80% of amputations and account for nearly 30,000 new cases annually.

INTRODUCTION

The goal of rehabilitation is to optimize an individual's physical, cognitive, and psychological recovery from a disease, injury, or traumatic event to maximize the patient's functional independence. This is achieved through an interdisciplinary team of professionals who use therapeutic exercise, assistive technology, patient and family education, and fundamental rehabilitation principles to prevent secondary injury, optimize pain control, and achieve established patient goals. Although the first priority in treating trauma casualties is to preserve life, early initiation of rehabilitation can have a significant positive impact on recovery, length of stay, community reintegration, and quality of life.[1]

Trauma patients, especially those who sustain spinal cord injury (SCI), traumatic brain injury (TBI), burns, and/or amputation, often experience polytrauma and are particularly vulnerable to secondary complications, which are best addressed by early preventative strategies. Rehabilitation professionals are especially skilled at mitigating the risks in developing secondary complications frequently associated with trauma; therefore, consultation to rehabilitation specialists should be initiated upon early hospitalization. In fact, evidence demonstrates that early mobilization in the intensive care setting results in fewer ventilator days, fewer intensive care unit and hospital days, less hospital complications, less sedation and delirium days, improved overall functional outcomes, reduced overall hospital cost and mortality, and increased likelihood of being discharged home.[2] The rehabilitation team can also be very helpful regarding other critical decisions during the acute management of trauma patients, including pain control strategies, bowel and bladder management, selection of definitive amputation levels, facilitating patient and family education, and disposition considerations, including the timing of transfer to a rehabilitation center.

As in other areas of medicine, subspecialty designation within the field of rehabilitation is common. Many physicians, therapists, nurses, and counselors receive subspecialized

training and board certification in areas such as spinal cord medicine, neurologic rehabilitation, amputee care, and TBI. Rehabilitation facilities themselves receive special accreditation by the Commission on Accreditation of Rehabilitation Facilities (CARF), which helps to ensure quality.[3] The National Institute on Disabilities and Rehabilitation Research (NIDRR) also recognizes excellence in rehabilitation institutions with their Models Systems Programs for Burns, SCI, and TBI.[4]

Trauma providers should not wait until the resolution of all medical and surgical issues before engaging in rehabilitation interventions; rather, they should be initiated early during hospitalization and remain an integral part of every trauma patient's care. Furthermore, health care professionals should recognize that the quality of the medical, surgical, and rehabilitative interventions provided during the acute phase of trauma casualty care will likely have longlasting implications for the patient's overall recovery, functional independence, and quality of life.

EFFECTS OF IMMOBILITY

The fundamental principles of rehabilitation are founded on mitigating and preventing (when possible) the effects of immobility. The physiologic and psychological effects of immobility lead to adverse organ system changes that may complicate healing and recovery. A thorough understanding of these potential consequences will help optimize any treatment plan.

Musculoskeletal Effects

MUSCLE ATROPHY AND WEAKNESS

Muscle responds to alterations in loading conditions. Whereas increased activity leads to muscle fiber hypertrophy, less activity may result in disuse atrophy. The muscles most affected by such disuse atrophy during immobilization are the antigravity muscles of the lower limbs and trunk. Thus, muscles with different functional roles atrophy at different rates during unloading.[3] In addition, during immobilization, the rate of protein synthesis declines while proteolysis increases. The resulting loss in total body protein is accompanied by a significant remodeling of muscle architecture, including loss of sarcomeres both in series and in parallel, leading to reduced muscle thickness and length.[5] As a result, it is estimated that immobilization leads to a loss of strength at a rate of 10% to 15% per week.[6] It is also recognized that muscle strength decreases at a faster rate than muscle size, indicating that, in addition to atrophy, other factors contribute to muscle weakness,[7] such as a decrease in mitochondrial volume density. This can result in reduced muscle oxidative capacity and loss of endurance.[8,9]

DISUSE OSTEOPOROSIS

Bone, like muscle, is sensitive to loading stresses. Bone mass increases under mechanical stress and decreases in the absence of muscle activity or gravitational forces. During bedrest, an unloaded skeleton leads to a decrease in bone formation with little to no impact on the rate of bone reabsorption, resulting in disuse osteoporosis because of a mismatch between bone growth and bone loss. It is estimated that bone mineral density reduces at a rate of 1% to 2% each month during immobilization.[10,11] Patients with preexisting osteopenia or osteoporosis are especially susceptible to bone mineral turnover during immobilization, resulting in a greater risk of osteoporotic-related fractures. Individuals with major limb loss are also susceptible to significant bone mineral loss, particularly when the amputation is performed at the proximal transfemoral level.[12] This is further complicated with any delay in weight bearing from poor soft tissue healing, infection, fracture, or difficulty with prosthetic fitting.[13] Finally, because of the high rate of bone reabsorption, trauma casualties are also at risk for hypercalcemia; therefore, calcium levels should be monitored closely.[14]

CONTRACTURES

A contracture is defined as a shortening of a muscle or connective tissue, which prevents movement through the normal range of motion. Joint contractures are classified according to etiology as either myogenic or arthrogenic. *Myogenic* contractures are caused by changes in muscle, tendon, or fascia. Although reduction in muscle length clearly contributes to the formation of a myogenic contracture, remodeling of intramuscular connective tissue during immobilization also plays a significant role. Such changes have only been observed when the muscle is immobilized in a shortened position, suggesting that limb positioning plays a direct role in connective tissue properties.[15] *Arthrogenic* contractures are caused by changes in bone, cartilage, synovium/subsynovium, capsule, or ligaments. Proliferation of intra-articular connective tissue, adaptive shortening of the capsule, and increases in cross-linking of collagen fibrils promote this type of contracture formation.[16]

PREVENTION AND TREATMENT

The best way to prevent the deleterious effects of immobility on the musculoskeletal system is to keep immobilization to a minimum. Strength, endurance, and flexibility programs are integral to both the prevention and treatment of musculoskeletal damage. Physical therapists, occupational therapists, and the nursing staff should promote activity as soon as it is determined to be safe by the medical and surgical teams. Resistance exercise has been shown to both maintain muscle protein synthesis and increase bone mass, reducing the incidence of muscle atrophy and disuse osteoporosis during immobilization.[7] Electrical stimulation may also be helpful by passively contracting skeletal muscle and maintaining muscle oxidative capacity.[17] Daily stretching has been shown to prevent serial sarcomere loss and reorganization of tissue components, leading to a reduced risk of muscle atrophy and contracture formation. Daily range-of-motion, flexibility, and muscle strengthening exercises help maintain the appropriate

balance of muscle activity across joints and can be used to both prevent and treat muscle atrophy, disuse osteoporosis, and contracture formation.[18]

Cardiovascular Effects

SHIFT OF BODY FLUIDS

Lying in a supine position eliminates the gravitational gradient normally present during standing, leading to a fluid shift of approximately 1 L from the legs to the thorax. The resultant increase in pressure within the heart's ventricles causes an increase in diastolic filling and stroke volume. As a consequence, the regulatory mechanisms within the heart react to this increase in intrathoracic volume, triggering urinary excretion, reduced thirst, resetting of the cardiopulmonary baroreflex, and overall reduction in plasma volume.[19]

CARDIAC EFFECTS

The circulatory system is also negatively affected by immobility, as skeletal muscles play an important role in compressing veins, forcing blood return to the heart. Therefore, loss of skeletal muscle due to injury or during bed rest impairs venous return, decreasing diastolic pressure and stroke volume. In response, heart rate typically increases and may lead to tachycardia. In fact, after 4 weeks of bed rest, the resting heart rate may increase by approximately 10 bpm.[20] Similar to skeletal muscle, cardiac muscle is also plastic in response to stress; therefore, as stroke volume decreases, myocytes begin to atrophy. The resulting reduction in cardiac mass and myocardial thinning lead to an overall decreased cardiac pump effectiveness.[21]

ORTHOSTATIC HYPOTENSION

Immobilized patients have a reduction in blood volume, venous return, and stroke volume. These factors in conjunction with cardiac deconditioning and myocardial thinning lead to the development of orthostatic hypotension.[20] Orthostatic hypotension is characterized by an excessive fall in stroke volume on assuming an upright position and has been reported after as little as 20 hours of bed rest.[22,23] Orthostasis is magnified in patients with SCI because a loss of sympathetically mediated vasoconstriction compounds venous pooling in the abdomen and lower limbs. This results in dizziness, lightheadedness, or loss of consciousness when assuming an upright position. Symptoms of orthostatic hypotension may significantly interfere with a patient's ability to participate in therapy or perform activities of daily living (ADLs) and may persist even after a protracted period of time, particularly for elderly patients.

AEROBIC CAPACITY

Maximal oxygen consumption (VO_{2max}) is a measure of cardiovascular fitness and has been shown to decrease in proportion to the duration of immobilization.[24] Individuals with preexisting cardiovascular disease present a higher risk for cardiac complications when recovering from a traumatic event and require explicit cardiovascular guidelines for participation in therapy. Typical guidelines for initiating therapeutic exercise include not exceeding a greater than 10 to 15 mm Hg rise in systolic blood pressure or greater than 60% to 65% maximal heart rate. Perceived exertion scales are often used to help monitor effort, and when needed, supplemental oxygen and monitoring with pulse oximetry should be used during therapy. Prior to initiating an appropriate exercise program, additional considerations should be made regarding the patient's preinjury conditioning, other cardiovascular risk factors, and medication use.[25]

VENOUS THROMBOSIS AND PULMONARY EMBOLUS

The incidence of deep venous thrombosis (DVT) after major trauma has been reported to be as high as 58%.[26,27] Without prophylaxis, its incidence after a motor complete SCI may be as high as 72%.[28] Typical features of DVT include swelling, erythema, and pain in the affected extremity. A pulmonary embolism (PE) is a potentially fatal consequence of a DVT, caused by part of the clot breaking away from the vein wall and lodging in a pulmonary artery. Typical symptoms include tachycardia, dyspnea, and chest pain. Unfortunately, both DVT and PE may manifest silently without producing any traditional symptoms, especially in patients with paralysis, sensory loss, or impaired consciousness.[29] Computed tomography (CT) pulmonary angiography has emerged as the preferential test in diagnosing a PE due to its high accuracy, ease of use, and ability to provide alternate diagnoses.[30] Other tests for assessing the presence of DVT or PE include ultrasound, lung ventilation/perfusion scans, and blood tests including D-dimer, but these are not as accurate in trauma patients.

PREVENTION AND TREATMENT

Minimizing the effects of cardiovascular deconditioning is achieved by reintroducing activities as soon as possible during the initial care of an injured patient. Management of orthostatic hypotension begins with preventing venous pooling in the abdomen and lower limbs by using abdominal binders and lower limb compression stockings. If necessary, the use of medications such as midodrine or fludrocortisone may also be helpful in treating or preventing episodes of orthostatic hypotension.[31] The incidence of DVT can be reduced to 7% to 10% with appropriate prophylaxis.[28] The current recommendation for the prevention of DVT and PE following trauma is to initiate pharmacologic anticoagulation as soon as possible after injury, provided no contraindications exist (eg, bleeding, ongoing surgical procedures, or progressive changes in the brain on CT). Anticoagulation may be accomplished with low-molecular-weight heparin or adjusted-dose unfractionated heparin in patients with impaired renal function. In addition to pharmacologic prophylaxis, use of mechanical prophylaxis such as compression hose or intermittent compression boots should be initiated as soon as possible and continued for at least 2 weeks. If contraindications to

pharmacologic prophylaxis exist, a vena cava filter may be inserted to minimize PE. Duration of pharmacologic prophylaxis is dependent on the extent of trauma and continued risk of thrombus formation. Guidelines for the management of individuals with SCI suggest the following indications to continue prophylaxis: (1) until hospital discharge for those with uncomplicated motor incomplete SCI; (2) 8 weeks for those with uncomplicated motor complete SCI; and (3) 12 weeks for those with complicated SCI (eg, lower limb fracture, age >70 years, or history of thrombosis, cancer, heart failure, or obesity).[32] Lifestyle modifications such as avoiding immobility and dehydration, tobacco cessation, and maintaining normal blood pressure can also help prevent DVT and PE.

Pulmonary Effects

LUNG VOLUME AND STRUCTURAL CHANGES

Patients take fewer deep breaths when lying down due to decreased respiratory muscle strength during immobilization. This decreased tidal volume (amount of air inhaled or exhaled during normal respiration) contributes to a 25% to 50% decrease in total respiratory capacity.[33] A similar reduction in the diameter of the airways leads to a decrease in functional residual capacity (the amount of air left in lungs after expiration), which can restrict airways and decrease gas exchange.[34] Further complicating respiratory function, bedridden patients have difficulty clearing secretions, leading to accumulation of mucus in the air passages and secondary atelectasis and pneumonia.[35]

PREVENTION AND TREATMENT

Promoting deep inspiration with an incentive spirometer is important for all trauma patients. Chest physiotherapy, vibration, and postural drainage techniques may be administered by a respiratory therapist and should be considered for any patient with acquired pneumonia while on bed rest. Clearing secretions is especially challenging for patients with SCI. Respiratory therapy, postural drainage, tracheal suction, manual cough support, and mechanical insufflation–exsufflation should be considered.[36] Special coughing techniques, such as the "quad cough," where after maximal inspiration the patient initiates a cough, while simultaneously receiving assistance from an attendant, who gently pushes inward and upward on their abdomen, should also be taught to the patient and family to help better manage respiratory secretions.[35,37]

Urinary System Effects

URINARY RETENTION, STONES, AND URINARY TRACT INFECTION

The urge to urinate is often lessened when supine, even when the bladder is full, and this contributes to urinary retention. An overdistended bladder may cause the stretch receptors to lose sensitivity, further reducing the urge to urinate. The aforementioned bone loss due to immobilization results in hypercalciuria, increasing the risk of forming bladder and renal stones. Urinary retention also encourages the growth of bacteria that raise the pH of urine and increase the risk of infection as well as precipitation of calcium that exacerbates stone formation.[38,39]

PREVENTION AND TREATMENT

For most trauma patients, an indwelling catheter is placed to ensure adequate emptying of the bladder, prevent fluid stasis and subsequent infection, and prevent a high-pressure system that may lead to hydronephrosis and renal damage. An indwelling catheter allows the trauma team to monitor fluid output and assess fluid status but should be removed as soon as medically possible to prevent urethral or bladder damage and to avoid reducing bladder compliance and storage capacity. After removing an indwelling catheter, it is important to monitor initial postvoid residual (PVR) bladder volumes to ensure adequate emptying. A PVR greater than 50 to 100 cm^3 should raise concern and consideration of replacement of the indwelling catheter.[40] Patients with a neurogenic bladder, whether from an injury to the central or peripheral nervous system, can develop either bladder contraction or distension; therefore, an appropriate bladder training program should be initiated as soon as the indwelling catheter is removed (see the later section "Spinal Cord Injury").

Gastrointestinal Effects

APPETITE AND GASTRIC TRANSIT TIME

Bed rest negatively impacts taste acuity, leading to a reduction in food intake.[41] In addition, immobilization results in structural and functional changes of the gastrointestinal tract, including atrophy of the mucosal lining and reduced glandular capacity.[42] Gastric transit time is prolonged during bed rest. The resultant decreased rate of evacuation from the stomach may cause symptoms of gastroesophageal reflux, regurgitation, and heartburn. The rate of intestinal peristalsis is also slowed during bed rest, further delaying intestinal motility and increasing water absorption, and may lead to constipation and fecal impaction.[39,43] These effects are likely to be magnified with the use of opioids or other pharmacologic agents that may also slow intestinal motility.

PREVENTION AND TREATMENT

Minimizing immobility, promoting activity, and ensuring adequate hydration are essential components of proper bowel management. The use of a bedside commode or bathroom toilet rather than a bedpan is encouraged. Bowel management should be targeted to the specific dysfunction that is present. Effective bowel programs include dietary modifications, timing evacuation about 30 minutes after a meal to take advantage of the gastrocolic reflex, and manual disimpaction, especially for individuals with neurogenic bowel dysfunction from an SCI. If further treatment is required, medications such as bulk-forming agents (eg, Metamucil), enemas, colonic irritants (eg, Dulcolax), and intestinal motility agents (eg, Senna) may be used. The ability of the patient

to independently perform manual disimpaction or self-administer enemas or suppositories will depend on his or her level of impairment. A surgical colostomy may be necessary to provide continence in certain patients, and appropriate colostomy care should be taught to the patient or caregiver.

Integumentary System Effects

DECUBITUS ULCERS

Immobilization is strongly associated with the formation of decubitus ulcers. Bony prominences such as the occiput, shoulder blades, sacrum, and heels are at greatest risk in supine patients.[44] Ulcerations that occur over the ischial tuberosities are generally attributable to increased seating pressure. Because immobilized patients are unable to make natural postural changes that alleviate pressure on susceptible body parts, blood flow to tissues may become obstructed, leading to necrosis of soft tissue and skin.[45] Patients with impaired cognition or sensation are particularly susceptible to the formation of pressure ulcers, and between 25% and 80% of patients with an SCI eventually develop pressure ulcers, with up to 8% developing fatal complications.[35] In addition, urinary and fecal incontinence may lead to excessive skin maceration and subsequent breakdown.[46] When present, pressure ulcers may have a significantly negative impact on rehabilitation, recovery, and quality of life.[47]

PREVENTION AND TREATMENT

Frequent skin inspection, appropriate hygiene, achieving urinary and fecal continence, proper fitting of orthotics, and avoiding excessive pressure through a regular turning schedule are all critical aspects of caring for the skin of a trauma patient. Providers should remain particularly attentive to the pressure-sensitive areas such as the sacrum, occiput, and heels/ankles. The utilization of specialty beds, padded orthotics, and seating cushions is encouraged but should not replace vigilant medical and nursing care. Many trauma centers have a dedicated skin and wound care team. These professionals should be consulted at the earliest signs of skin breakdown and should be involved in continuous education of the medical, nursing, and therapy staff. The primary treatment of pressure ulcers should focus on prevention through frequent position changes by a caregiver or the patient. This requires early patient and caregiver education, as well as the use of specialized equipment, including wheelchairs with adjustable tilting features.

Unique Complications of Trauma Patients

HETEROTOPIC OSSIFICATION

Heterotopic ossification (HO) is the formation of mature lamellar bone in tissue that is not normally ossified.[48] It commonly develops in patients with TBI, SCI, burn injury, or blast-related amputations.[49-51] The most common site of HO following SCI is the hip, but it may also occur in the elbow, shoulder, and knee.[52] For patients with burns, 92% of cases occur in the elbow,[53,54] and up to 80% of patients who sustain an amputation from a blast injury may develop HO.[55] The clinical presentation of HO may include a decrease in range of motion and erythema and swelling about the involved joint. Radiographs, bone scans, and monitoring of serum alkaline phosphatase levels may aid in the diagnosis. Prophylaxis with bisphosphonates, nonsteroidal anti-inflammatory drugs (NSAIDs), and local irradiation may prevent HO.[56] Treatment of HO involves active and passive range of motion to prevent worsening; however, in severe cases, surgical excision may be required to improve functional outcomes.[52,57] Even when treated aggressively, HO may still result in significant restrictions in range of motion and subsequent disability.

SPASTICITY

Spasticity is defined as a velocity-dependent increase in muscle tone that occurs following injury to upper motor neurons and is associated with increased deep tendon reflexes and other signs of upper motor neuron disease. Spasticity occurs frequently in patients with TBI and SCI and may significantly impede successful rehabilitation if not treated appropriately. During the acute stage, however, a patient with SCI may have diffusely diminished reflexes below the level of injury. This period of hypotonicity and hyporeflexia is referred to as spinal shock. In the weeks following SCI, reflexes return and gradually increase while spasticity develops. The incidence of spasticity following SCI at 1 year is estimated to be 65% to 78%.[58] Spasticity may result in significant pain, impaired mobility, difficulty with dressing and hygiene, and an increased risk of skin breakdown. In certain circumstances, however, spasticity may be of benefit to the patient, particularly when lower limb tone is used to aid in transfers. Therefore, the decision to treat spasticity must be based on improving patient function, hygiene, and/or care.

Conservative treatment of spasticity begins with positioning, stretching, splinting, and range-of-motion exercises. Medications used for spasticity include γ-aminobutyric acid (GABA) agonists (eg, baclofen), centrally acting α_2-agonists (eg, tizanidine), and medications that inhibit skeletal muscle contraction (eg, dantrolene). Botulinum toxin or phenol injections may also be helpful in the management of spasticity. Implanted pumps may be used to deliver highly concentrated doses of medications directly to the spinal cord through an intrathecal catheter, which minimizes systemic side effects, and surgical treatment of spasticity may be required to correct fixed deformities.[58] The appropriate choice of medication, however, should be based on the patient's response as well as the presence of adverse side effects.

PAIN MANAGEMENT

Background

Inadequate pain control is associated with delayed healing, increased complications, prolonged hospitalization, poor sleep, and diminished quality of life.[59] It has also been shown

to be a significant cause for hospital readmission.[60] Therefore, attentive pain management should be a priority for any trauma team. Unfortunately, many patients are undertreated for their pain.[61] Multiple professional organizations, including the American Pain Society (APS) and the Agency for Healthcare Research and Quality (AHRQ), have developed and disseminated clinical practice guidelines to address the insufficient treatment of pain in America's health care delivery system.[62] In addition, The Joint Commission introduced new standards for pain assessment and management, advocating pain management as a patient right and requiring health care organizations to employ effective pain management policies.[63] Despite these initiatives, significant challenges still remain in changing clinical practice.[64] Postoperative pain is a significant problem for most patients and demands special attention from the trauma team.[65] Although a multitude of pain interventions currently exist, numerous barriers prevent patients from receiving optimal care.[66] Patients may underreport their level of pain, particularly if they expect it as part of their disease process or treatment. Pain medications prescribed on an "as-needed" basis can cause a lag in treatment and a "peak and trough" pain pattern of analgesia. Such poor pain management may lead to significant anxiety, poor sleep, and loss of appetite. In addition, undertreating pain may impair the patient's ability to participate in rehabilitation. Once severe pain has been established, it is more difficult to treat. Therefore, aggressively treating perioperative pain early with the use of multimodal and scheduled analgesia can be very helpful in avoiding the development of more complex pain syndromes. Furthermore, uncontrolled or protracted acute pain may lead to chronic pain conditions, which may have long-term negative implications on function, return to work, and quality of life.[67,68]

Providers traditionally underestimate a patient's level of discomfort. Therefore, it is important to establish a standardized method of communication between the patient and provider. To determine pain levels, most health care organizations use a visual analog scale typically ranging from 0 (no pain) to 10 (worst imaginable pain). In addition to communicating pain levels, this tool may also be used to assess the effectiveness of pain interventions and establish therapeutic goals for each patient. Determining pain levels for children or individuals with cognitive impairment may be challenging. In such cases, providers may use the FACES Scale, in which images of faces ranging from smiling to grimacing reflect pain scores.[69] A speech-language pathologist may be of great assistance to the patient and treatment team by developing an effective communication system for more challenging communication barriers. Family members should also be engaged with the treatment team to help provide feedback on the patient's nonverbal expression of pain and response to therapeutic interventions.

Phantom Limb Pain

Although pain affects all patients with traumatic injury, phantom limb pain (PLP) is unique to individuals with acquired amputation. PLP is the experience of a painful sensation (eg, cramping, burning, aching) within a missing (phantom) limb. The phenomenon occurs in up to 90% of patients following amputation of a major limb and is thought to be the result of central nervous system reorganization or hypersensitivity. PLP symptoms may occur immediately after limb loss or may be delayed by several weeks. Although most individuals experience a gradual decrease in PLP over time, many individuals will report persistent symptoms. Most common treatments include medications that are typically used for neuropathic pain. In addition, mirror therapy has also been shown to be an effective treatment. This involves the patient placing a mirror in front of them in a way that the reflection of their intact limb appears to represent their phantom (missing) limb, which they are then able to voluntary move and, through visual feedback, trick their brain that the phantom limb is moving, which helps decrease their pain.[70]

Terminology and Pathophysiology

Although a detailed discussion is beyond the scope of this chapter, the proper treatment of pain requires a basic understanding of common pain terminology and a familiarity with nervous system physiology (Table 55-1).

Treatment

Pain management strategies should target the suspected source of nociception (Fig. 55-1). Issues such as positioning, poor sleep, anxiety, and depression may augment the perception of pain. In addition, chronic underlying conditions such as migraine headaches, diabetic neuropathy, osteoarthritis, or other musculoskeletal injuries may be exacerbated by trauma, adding to a patient's discomfort. Therefore, a thorough history and physical examination are critical in evaluating every trauma patient. It is possible that other injuries may have been overlooked during the initial trauma screen due to a distracting injury; therefore, a tertiary survey is necessary for all trauma patients. Pain management strategies should be readily discussed among the trauma team, and a pain specialist should be consulted for patients with complex pain problems. Treatment strategies are often multimodal and include pharmacologic and nonpharmacologic approaches.

PHARMACOLOGIC

With the current opioid crisis, clinicians are encouraged to use a multimodal pain regimen to minimize the use of opioids while still providing adequate pain control.

- *Opioids* are the most common analgesic medications used during acute care of the trauma patient and may be administered orally, intramuscularly, or intravenously. Morphine remains the most frequently used opioid because it is inexpensive, is readily available, and has clear analgesic, amnestic, and sedative effects. Although fentanyl is more expensive and not as readily available, it offers a shorter half-life and less sedation than morphine, which may be

TABLE 55-1: Common Pain Terminology

Term	Definition
Nociception	The neural process of encoding and processing noxious stimuli
Hyperalgesia	An increased sensitivity or response to a painful stimulus
Allodynia	A painful response to a normally nonpainful stimulus
Dysesthesia	An unpleasant or painful sensation that occurs spontaneously
Paresthesia	A sensation of a tingling or numbness of the skin or region of the body that occurs spontaneously
Epicritic sensations	Nonpainful sensations such as light touch, vibration, and position sense, transmitted through low-threshold, large-diameter myelinated nerve fibers
Protopathic sensations	Painful sensations transmitted through high-threshold, small-diameter unmyelinated nerve fibers
Visceral pain	Pain generated through internal nociceptors as a result of internal organ damage
Somatic pain	Pain generated from peripheral nociceptors within the skin, muscle, soft tissue, and bone
Acute pain	Pain associated with an acute event, generally lasting <30 d
Chronic pain	A pain syndrome lasting beyond 3–6 mo or pain that extends beyond the expected period of healing

better in some trauma casualties.[71] Patients on chronic opioids may be safely treated with intravenous opioid bolus dosages up to 20% of their total daily use; however, as when using any opioid, caution should be observed because of the risk of addiction and respiratory depression. The coadministration of opioids and benzodiazepines may have a synergistic effect on increasing respiratory depression and delirium.[72] Evidence suggests that short-acting opioids may be favorable to long-acting opioids, especially during acute care hospitalization. Reliance on long-acting opioids at time of hospital discharge has a 27.3% probability of continued use after 1 year, whereas short-acting oxycodone demonstrated only a 4.7% probability.[73]

- *Continuous infusion analgesia and anesthesia* may be achieved with intravenous patient-controlled analgesia (PCA) or with regional anesthesia through a peripherally placed catheter. Intravenous opioid administration through PCA helps to avoid the "peak and trough" phenomenon of inadequate pain management. Continuous infusion of an anesthetic agent through a catheter in the region of a peripheral nerve or plexus offers outstanding pain control for limb injuries and avoids the secondary cognitive effects of systemic opioids. Regional anesthesia catheters should be monitored for effectiveness as well as signs of infection and have been reported to be safe for up to 34 days in combat-related injuries.[74]

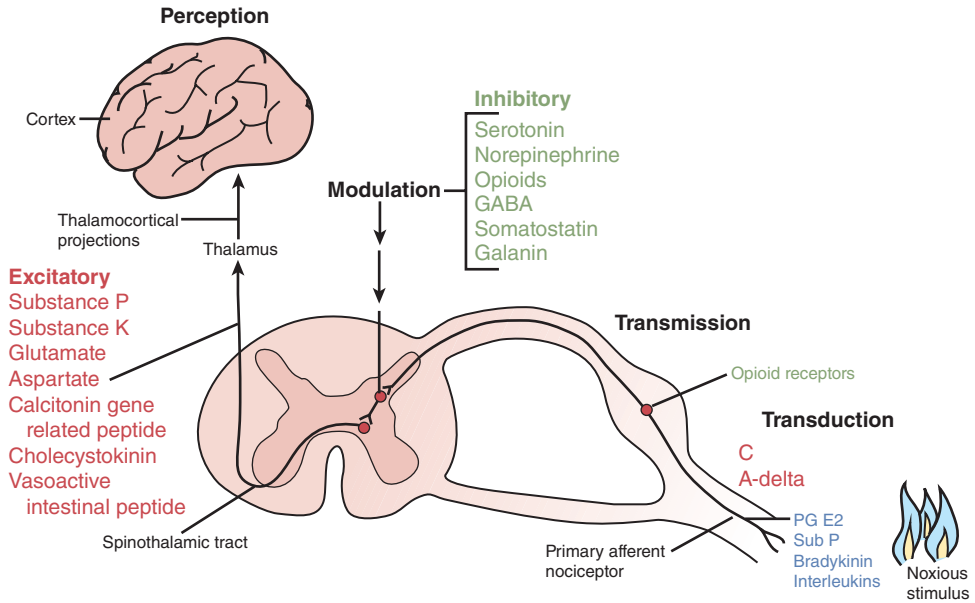

FIGURE 55-1 Afferent nociception pathway: demonstrates pain signals originating from a peripheral noxious stimulus, traveling along sensory afferents through the dorsal root ganglion and into the spinal cord, where both excitatory (red) and inhibitory (green) neurotransmitters modulate impulses before the signal is perceived within the brain. GABA, γ-aminobutyric acid; PG E2, prostaglandin E2; Sub P, substance P.

- *NSAIDs* can be very helpful in relieving peripherally generated pain by blocking cyclooxygenase and the subsequent formation of prostaglandins, which promote pain and inflammation. There are two isomers of cyclooxygenase (COX-1 and COX-2), each involved in a range of physiologic functions. The prostaglandins produced by COX-1 protect the stomach and support platelet aggregation. Therefore, nonselective COX inhibitors should be used with caution in the injured patient to avoid the possible side effects of peptic ulceration and dyspepsia. Ketorolac (Toradol) is the only NSAID in the United States available in an injectable form. Its administration, either intravenously or intramuscularly, has analgesic effects similar to morphine. All NSAIDs should be used with caution in the presence of complex fractures, particularly those with high propensity for nonunion, although data are conflicting.[75]
- *Acetaminophen* is a nonopioid analgesic without the antiplatelet and anti-inflammatory properties of NSAIDs. When used in combination with opioids, it has been shown to lower opioid requirements by up to 20%.[76]
- *Muscle relaxants* such as tizanidine and baclofen are often used in the trauma setting, particularly for painful spasms and spasticity due to upper motor neuron injury.[72] α_2*-Adrenergic agonists* such as clonidine can also help to reduce spasticity and also help decrease anxiety and opioid withdrawal symptoms. However, care should be taken to avoid hypotension and bradycardia.[76]
- *N-Methyl-*D*-aspartate (NMDA) receptor antagonists* such as ketamine are thought to inhibit nociopathic pathways within the central and peripheral nervous system, with evidence suggesting similar analgesic effects to morphine and reducing the overall need for opioids.[72] Administration is intravenous and requires continuous monitoring for potential tachycardia, hypertension, agitation, and hallucinations.[77]
- *Antiepileptic medications* such as gabapentin, pregabalin, carbamazepine, valproic acid, and oxcarbazepine are often used to help treat neuropathic pain and may also provide relief from PLP. These medications act as membrane stabilizers to the axons in the central and peripheral nervous systems through modulation at the sodium and calcium channels.[76]
- *Antidepressant medications* have long been used to help augment pain management, particularly for neuropathic pain. Tricyclic antidepressants (TCAs) are effective but have sedative effects. They also have anticholinergic properties, which may lead to secondary problems such as confusion and urinary retention, especially in the elderly. Therefore, side effects should be monitored and the dose of medication adjusted accordingly. Duloxetine is an antidepressant medication with fewer side effects than TCAs and is approved for the treatment of diabetic nerve pain. Although the effect of selective serotonin reuptake inhibitors on acute or chronic pain is unclear, these medications should be considered to treat depression or anxiety, which may be a contributing factor to the patient's pain.[72,76]
- *Sympathetic blockade* may be helpful in patients with a sympathetically mediated complex regional pain syndrome (CRPS). CRPS should be suspected if the pain in an extremity persists long after what would be normally expected in patients with similar injuries. Symptoms typically include regional pain, allodynia, hyperalgesia, swelling, and local dysautonomia (sweating, discoloration, or temperature changes). Because prolonged immobility may be a precursor to CRPS, early rehabilitation may prevent its occurrence. When CRPS is suspected, a pain specialist consultation is recommended.

NONPHARMACOLOGIC

- Modalities such as heat or ice may provide effective pain relief for injured patients and should be made available as needed. Caution should be observed when using these modalities for patients with injuries to the central or peripheral nervous system or those with cognitive impairment. Lack of protective sensation or awareness my lead to a skin injury and/or burn.
- Interventions such as acupuncture, desensitization techniques, self-hypnosis, biofeedback, and music therapy may also provide some pain relief for injured patients. Such therapies are generally well received and can be administered without fear of side effects or adverse interaction with other medications.

FUNCTIONAL IMPROVEMENT THROUGH INTERDISCIPLINARY TEAM CARE

The early application of rehabilitation principles helps prevent secondary injury and facilitates patient recovery. Consultation with rehabilitation specialists should be considered for every trauma patient. An interdisciplinary rehabilitation team employs a variety of specialists from physiatry, physical therapy, occupational therapy, mental health, and speech-language pathology. For patients with cognitive impairment, a neuropsychologist may also offer invaluable assistance in identifying cognitive deficits and formulating management decisions. In addition to providing holistic care, rehabilitation providers spend additional time with patients and family members to perform a comprehensive functional assessment to determine preinjury functional capacity as well as current functionality. Rehabilitation professionals will assess any potential barriers that might prevent timely hospital discharge (eg, cognition, mobility, safety, architectural barriers) or independent living with or without assistance. Once these barriers are identified, therapeutic interventions may be initiated, and appropriate assistive equipment can be ordered in a manner to fit with establishing short- and long-term rehabilitation goals.

Physical Medicine and Rehabilitation Specialists (Physiatrists)

Physiatrists are board-certified physicians who specialize in neuromusculoskeletal disorders and rehabilitation. These

health care providers are skilled at integrating medical and surgical treatment plans with the rehabilitation team. Physiatrists are also helpful in establishing safety precautions for the patient, managing pain and bowel/bladder issues, and aiding in appropriate discharge planning and disposition. Subspecialty certifications currently exist in brain injury, neuromuscular, pain, SCI, sports, and pediatric rehabilitation medicines.

Physical Therapists

Physical therapists evaluate motor function and mobility. This includes assessing range of motion of joints and motor strength as well as a patient's balance and ability to sit, stand, transfer, or walk independently or with an assistive device. Treatment should begin early during the acute care hospitalization and continue until goals are met. Physical therapy assessments are very helpful in guiding appropriate discharge planning. It is important for the trauma physician to provide the therapist with guidance on appropriate weight bearing, range of motion, or activity precautions.

Occupational Therapists

Occupational therapists assess a patient's ADLs and instrumental ADLs (IADLs). ADLs include feeding, toileting, dressing, and hygiene. IADLs refer to activities beyond basic personal care, typically requiring devices such as a telephone, kitchen utensils, or appliances. Examples of IADLs include managing finances, preparing meals, and doing laundry. A patient's independence in ADLs and IADLs will help determine appropriate discharge planning. Occupational therapists are also skilled at helping those with extremity trauma or nervous system injury regain both gross and fine motor hand function.

Speech-Language Pathologists

Speech-language pathologists assess, diagnose, and treat patients with difficulties related to speech, language, swallowing, or cognitive–communication deficits. Because adequate nutrition is necessary for tissue healing and recovery, evaluating a patient's ability to swallow safely may help guide appropriate interventions to ensure appropriate caloric intake.

Additionally, the speech-language pathologists may develop ways to improve the necessary communication between the treatment team and the patient.

Orthotists and Prosthetists

Orthotists and prosthetists are certified in evaluating, fabricating, and custom fitting orthopedic braces (orthotics) and artificial limbs (prostheses). Orthotics are devices used to align, support, protect, or improve the function of a body part. Examples of orthotics used in trauma casualties include halo cervical traction splints, ankle-foot orthotics for ambulation and pressure relief,[78] and thoracolumbosacral orthotics. Orthotics may be either static (rigid) or dynamic (assist with desired motion). Prostheses may be used for upper or lower limb amputations and are composed of a socket, suspension, joint(s), shank, and terminal device (hook, hand, or foot). Early prosthetic fitting may enhance acceptance and functional recovery.

Mental Health Professionals

Mental health professionals should be a part of any interdisciplinary team. Traumatic events have a significant psychological impact on the patient, family members, and caregivers. Nearly all survivors of major traumatic events will exhibit stress-related symptoms, and up to 40% may have a psychiatric disorder at 1 year following trauma.[79] For combat casualties, rates of comorbid posttraumatic stress disorder (PTSD) have been reported to be 16.7%,[80] but may be as high as 30% to 45% in those sustaining amputation or SCI.[81] Fear of isolation, lack of wholeness, and concerns about the future are common but can be eased by supportive family members and caregivers. A preventative medicine approach to psychiatry helps to decrease the stigma that is often associated with seeing a behavioral health specialist. Fostering appropriate mental health care acutely likely helps to prevent the development of disabling psychiatric disorders often associated with trauma.[82] Special considerations should also be made to the children and other family members of those who suffer trauma, particularly those with disfiguring injuries.

Rehabilitation Engineers and Assistive Technology Specialists

Rehabilitation engineers specialize in the fabrication, fitting, and training of assistive technology devices. Assistive technology devices provide essential support for individuals with disabilities, allowing them to perform ADLs and return to work, sports, and recreational activities. They include manual and powered wheelchairs, seating/standing systems, adaptive vehicles, augmentative communication systems, electronic aids, and assistive robotics. As technology becomes more sophisticated, expertise is needed in these areas. The Rehabilitation Engineering and Assistive Technology Society of North America (RESNA) currently provides credentialing to suppliers, health care providers, and engineers to ensure quality in products and service delivery.

Peer Support Visitors

Peer support visitors are trauma survivors who receive training to support other patients and their families. Peer visitors generally have suffered a similar life-changing injury as the patient and demonstrate a successful recovery by adapting and returning to community participation. Peer support visitors are especially beneficial for individuals with paralysis, vision loss, disfiguring scars, or limb loss. Not-for-profit organizations, such as the Amputee Coalition, offer specialized peer support training programs, which may be helpful

for individuals or institutions interested in initiating such a program.

Vocational Rehabilitation Specialists

Vocational rehabilitation specialists help match career goals with functional capacity, assistive technology, and professional talents. Community reintegration and return to vocation should be in the long-term goals of all trauma patients. Although controversy exists as to the optimal timing of intervention, discussing vocation during early hospitalization may facilitate engagement in rehabilitation and hasten recovery.

Case Managers and Social Work Services

Case managers are essential to adequately address discharge planning and transfer to the next level of care, coordinate equipment needs, and provide patient and family education. Therefore, these professionals should be an integral part of the trauma treatment team.

SPINAL CORD INJURY (SEE CHAPTER 26)

Introduction

Injury to the spinal cord results in impaired motor strength and sensation below the level of injury. SCI is often associated with bowel and bladder dysfunction and may cause sexual impairment as well as dysfunction of the autonomic nervous system. Approximately 12,000 SCIs occur nationally each year, and approximately 259,000 trauma survivors are living with SCI in the United States. Males account for approximately 80% of those with SCI, and motor vehicle accidents represent the largest proportion of injuries, followed by falls, violence, and sports injuries. The societal impact of SCI is staggering, resulting in total lifetime costs of between $1 million and $4.6 million per patient.[83]

Classification

SCIs are classified by their severity and level of injury, using the American Spinal Injury Association (ASIA) Standard Neurological Classification Worksheet.[84] This classification system provides guidelines for measuring key motor levels and sensory examination points that represent function at a given spinal level. According to this method of classification, patients are given two scores, with one based on level and the other on the degree of impairment. The neurologic level of injury is defined as the lowest level of normal functioning. The impairment is rated as *A*, *B*, *C*, *D*, or *E*. An *A* is assigned to a patient with a complete injury, with no motor or sensory function in the S4 or S5 levels. *B* represents an incomplete injury with sensory sparing but no motor activity below the level of injury. A patient with some motor (nonfunctional) strength below the level of injury is assigned a *C*, whereas a *D* classifies a patient with more motor sparing below the level of the lesion, as indicated by antigravity strength in greater than 50% of those muscles spared. A classification of *E* represents normal strength and sensation.

Acute Management

Treatment of acute SCI in a trauma center has a significant positive impact on survival and long-term functional recovery.[85] Immediate spinal stabilization is essential to prevent further neurologic compromise. Although not all patients require surgery after SCI, current literature suggests that surgical treatment relieving spinal cord compression can prevent further neurologic deterioration.[86]

The routine use of corticosteroids for the treatment of acute SCI is no longer recommended. In 1990, the National Acute Spinal Cord Injury Study recommended that high-dose methylprednisolone be administered for 24 to 48 hours. Although prior recommendations endorsed the use of high-dose methylprednisolone following acute SCI,[87] increasing concerns about infection, gastrointestinal bleeding, and steroid myopathy led some trauma centers to question the safety and efficacy of this protocol. As result, the 2013 guidelines of the American Association of Neurological Surgeons and Congress of Neurological Surgeons recommended against the routine use of methylprednisolone following acute SCI.[88] In addition, a meta-analysis in 2015 failed to demonstrate a benefit in functional outcome in those receiving methylprednisolone versus a placebo at both the 6-month and 1-year follow-ups.[89] Currently there is no international consensus for the application of high-dose methylprednisolone in acute traumatic SCI, and recent recommendations consider steroids at best as an option rather than standard of care.[90,91]

Regenerative Medicine

When an SCI occurs, the axons of the spinal cord nerves are destroyed and the surrounding oligodendrocytes die. Because the body cannot replace these cells, a functional defect results. Stem cells have become a source of research as a potential way to repair the nerve cells of the spinal cord and surrounding structures. They not only have the theoretical ability to rebuild the physical defect in structure, but they also have anti-inflammatory and immunomodulatory effects that are postulated to have a positive effect following SCI.[92-94]

In preclinical studies, embryonic stem cells transplanted into rodent models of SCI have demonstrated some recovery of function; however, concerns about the use of embryonic stem cells are both logistical (the plausibility of translating rodent models to humans) and ethical (the use of embryonic tissue). Mesenchymal stem cells are found in the bone marrow and may also exist in adipose and muscle tissue. They are not only easier to isolate, but their usage has fewer ethical concerns than embryonic stem cells; however, a major concern is the potential for spontaneous malignant transformation.[95] Preclinical studies thus far have had mixed success in improvements

in functional outcome following the implantation of mesenchymal stem cells into rodent models.[96] The potential of stem cells to provide a cure for SCI has resulted in many clinical trials over the past decade. Thus far, the question as to their efficacy is still unanswered, and there is no consensus about which type of stem cell will be the most effective therapeutically.[97]

Functional Outcomes After Spinal Cord Injury

The expected level of independence and functional capacity after an SCI depends largely on the level of injury and the impairment rating. Although lifelong mechanical ventilation is typically required for patients with complete injuries at the level of C3 or above, it is often not necessary for those with injuries at C4 or below. An injury at the C5 level is expected to result in independent mobility in an electrically powered wheelchair and driving in a van with appropriate modifications. C7 level injuries allow independence with transfers, weight-shifting maneuvers, and bowel and bladder management. Although individuals with thoracic level injuries have full function of the upper limbs, they typically require the use of a manual wheelchair with varying degrees of truncal support. Patients with an L1 or L2 injury generally also require a manual wheelchair but have independent truncal control. Patients with L3 or L4 injuries may be able to ambulate with assistive devices, and those with injuries at L5 or below should be able to independently ambulate.[85]

New technologies are emerging to facilitate rehabilitation and assist those with SCI to achieve better functional outcomes. Multiple motorized exoskeletal systems are currently available to augment mobility. Evidence suggests that these systems may also offer improvement in cardiovascular, gastrointestinal, and bladder function, as well as improved bone health and psychological well-being.[98,99] Brain–machine interfaces have also been investigated as a technology to achieve functional restoration of limb movement for those with an SCI. These devices allow the patient to have control of assistive devices by analyzing brain signals.[100] In addition, spinal cord stimulation has been investigated as a way of activating networks to induce locomotor-like movements in the legs, with promising results in preliminary animal models.[101]

Complications After Spinal Cord Injury

The management of patients with SCI is focused on the prevention of complications that may interfere with successful rehabilitation. The recognition of and attention to these complications are critical components of a successful rehabilitation program.

AUTONOMIC DYSREFLEXIA

Autonomic dysreflexia (AD) is a severe and life-threatening complication of SCI that occurs in patients with lesions at or above T6. AD typically occurs as a result of a noxious stimulus below the level of the injury, which triggers a sympathetic reflex that goes "unchecked" or uncorrected because of injury to the descending inhibitory tracts within the spinal cord. This results in elevated blood pressure, bradycardia or tachycardia, headache, and sweating or piloerection above the level of the injury. If not recognized and treated properly, stroke or death may occur. Once AD is recognized, the patient's head should be elevated and the noxious stimulus (eg, tight or constricted clothing, pressure on the skin, bladder distension, or bowel impaction) should be identified. If an indwelling catheter is in place, it should be thoroughly examined for constriction or kinks that may cause distension. If replacement is needed, it should be inserted with the use of lidocaine gel to decrease the amount of noxious stimuli. A rectal exam should be performed to assess for fecal impaction, and if present, manual disimpaction may be required. If blood pressure remains elevated, pharmacologic management may be necessary. Typical first-line medications include nitroglycerin paste (which can be easily removed to prevent rebound hypotension) or chewable calcium channel blockers. During the management of AD, blood pressure should be continually monitored, and the patient should be continually reexamined for sources of noxious stimulation.[31,102,103]

HYPERCALCEMIA

Hypercalcemia as a result of upregulation in osteoclast activity may occur in individuals with SCI, especially adolescents and young adult males. This may result in lethargy, abdominal pain, nausea, vomiting, psychological changes, polydipsia, and polyuria. If hypercalcemia is suspected, serum calcium levels should be monitored and treated appropriately.[104]

BLADDER DYSFUNCTION

Bladder dysfunction following SCI may lead to incontinence and urinary retention. In a typical upper motor neuron lesion, the bladder will become spastic and inadequately store urine, resulting in frequent episodes of small-volume incontinence. In lower motor neuron lesions, the bladder may become flaccid, resulting in failure to empty and overflow incontinence. Many patients with SCI develop detrusor–sphincter dyssynergia, characterized by a detrusor contraction against an unrelaxed sphincter, leading to failure to empty and a high-pressure urinary system. Management of bladder dysfunction aims to prevent incontinence and urinary reflux and infection, while ensuring adequate voiding at socially acceptable times. Options for bladder voiding include indwelling catheters (eg, Foley catheter or suprapubic catheter) or clean intermittent catheterization. Crede maneuver is also helpful, in which the patient or a caregiver provides manual pressure to the suprapubic region to facilitate emptying of the bladder. Pharmacologic management of a hyperactive bladder consists primarily of anticholinergic medications to relax the detrusor muscle. Intravesical administration of botulinum toxin may also be considered to treat detrusor spasticity. Surgical options include augmentation cystoplasty, cutaneous conduits, and urinary diversions.[105]

BOWEL DYSFUNCTION

Bowel dysfunction is common after SCI due to a loss of bowel control from the central nervous system, resulting in constipation, delayed gastric emptying, and poor colonic motility.[106] Both incontinence and failure to empty may result (see the earlier section "Effects of Immobility").

SPASTICITY, ORTHOSTATIC HYPOTENSION, SKIN BREAKDOWN, AND HETEROTOPIC OSSIFICATION

These are also significant problems for patients with SCI. The reader should refer to the previous discussion of these topics.

TRAUMATIC BRAIN INJURY (SEE CHAPTER 22)

Introduction

TBI is the leading cause of death and disability in young adults in the United States, with over 1.4 million individuals presenting to emergency and other acute medical settings each year. Of these, 275,000 require hospitalization and 52,000 cases are fatal.[107] In addition, it is estimated that 19% to 23% of service members who deploy overseas sustain a concussion or mild TBI.[108] Reports estimate that there were approximately 313,816 service members diagnosed with TBI between 2000 and 2014 from the wars in Iraq and Afghanistan.[109] TBI may occur from multiple mechanisms including blunt, penetrating, or blast trauma. The presence and severity of TBI may be difficult to diagnose, particularly if the injury was not witnessed. Additionally, secondary effects of trauma, such as hypovolemia, anoxia, and metabolic changes, may lead to significant brain injury. TBI may occur in discrete lesions or more diffusely such as in diffuse axonal injury. Neuroimaging, including CT, magnetic resonance imaging (MRI), functional MRI, and diffusion tensor imaging, is used to confirm the presence of a mechanical lesion that may require surgical evacuation.[110]

Classification

Much debate surrounds the optimal way to classify TBI. Most trauma centers rely on the Glasgow Coma Scale (GCS) or length of posttraumatic amnesia (PTA) to determine severity of the TBI (Table 55-2). PTA is the time between injury and the development of new memories, demonstrated by the patient's ability to recall daily events. It may be more formally assessed using tools such as the Galveston Orientation and Amnesia Test (GOAT).[111] The Ranchos Los Amigos Scale is often used to describe a patient's level of awareness, cognition, behavior, and interaction with the environment after a TBI (Table 55-3).[112] Patients who are mobile, but confused or agitated, will likely require a secured inpatient setting.

Predicting outcomes after TBI is extremely challenging because of imprecise classification systems. In addition, issues such as heterogeneity of injury patterns, premorbid cognitive and physical functioning, family support, and psychosocial factors all play differential roles in recovery.[113] Reports indicate that 38% to 80% of patients experiencing mild TBI will develop postconcussive syndrome, characterized by headache, fatigue, anxiety, and impaired memory, attention, and concentration.[114] The best predictor of outcome after TBI is the patient's speed of recovery and response to treatment; therefore, monitoring patient performance in multiple functional domains is important. Furthermore, an extended duration of loss of consciousness immediately following TBI has been negatively correlated with long-term rehabilitative potential.[115] Numerous outcome instruments are currently in practice including the Glasgow Outcome Scale (GOS), Functional Independence Measure (FIM), Community Integration Questionnaire (CIQ), Craig Handicap Assessment and Reporting Technique (CHART), and Disability Rating Scale (DRS). For a more comprehensive reference on TBI outcome tools, the reader is referred to the National Institute of Neurological Disorders and Stroke's Traumatic Brain Injury Common Data Element Standards.[116]

Treatment

A comprehensive discussion of treatment strategies for individuals with TBI is beyond the scope of this chapter. Trauma teams should consider the effects of immobility as described earlier and apply the appropriate rehabilitation principles. Patients with severe TBI should be transferred to a specialized rehabilitation facility as soon as medically feasible. Comprehensive acute inpatient rehabilitation programs provide intensive medical, physical, cognitive, and behavioral therapy regimens, which are coordinated in an interdisciplinary fashion.[117] Because of the frequent association of other sensory disturbances, these treatment teams are often augmented by highly skilled audiologists, neuropthalmologists, optometrists, and vision rehabilitation specialists. In addition, music and art therapists may be invaluable in not only engaging the patient in therapy, but also in improving cognitive and communication therapies. Transfer to a skilled nursing facility may

TABLE 55-2: Traumatic Brain Injury Classification

Injury severity	Glasgow Coma Scale	Posttraumatic amnesia	Structural imaging	Loss of consciousness	Alteration of consciousness
Mild	13–15	<24 h	Normal	0-30 min	A moment up to 24 h
Moderate	9–12	1–7 d	Normal or abnormal	>30 min and <24 h	>24 h
Severe	3–8	>7 d	Normal or abnormal	>24 h	>24 h

TABLE 55-3: Ranchos Los Amigos Scale

Cognitive level	Outcome	Patient response
I	No response	No response to sounds, sights, touch, or movement
II	Generalized response	Limited response, which is inconsistent and nonpurposeful; responses to sounds, sights, touch, or movement
III	Localized response	Inconsistent but purposeful response in a more specific manner to stimuli; may follow simple commands
IV	Confused and agitated	Confused and often frightened; overreactions to stimuli by hitting or screaming; highly focused on basic needs (eg, eating, toileting); difficulty following directions
V	Confused and inappropriate	Appears alert and responds to commands; easily distracted by the environment; frustrated and verbally inappropriate; focused on basic needs
VI	Confused and appropriate	Follows simple directions consistently; may have some memory but lacks details, attention span of about 30 min
VII	Automatic and appropriate	Follows a set schedule; does routine self-care without help; attention difficulty in distracting or stressful situations; problems in planning and following through
VIII	Purposeful and appropriate	Realizes difficulties with thinking and memory; less rigid and more flexible thinking; able to learn new things; demonstrates poor judgment; may need guidance for decisions

be necessary for those with impairments that prohibit transfer to an acute inpatient rehabilitation facility (eg, unable to tolerate a minimum of 3 hours of rehabilitation per day).[118,119] Patients who sustain severe trauma but deny TBI or cognitive difficulties may still be determined to have significant cognitive deficits during formal neuropsychological testing. Common symptoms of TBI include headache, visual and hearing disturbances, balance difficulties, poor sleep, irritability, and impaired cognition.[108,111] In addition, comorbidities such as depression, PTSD, and generalized anxiety disorder often accompany TBI and have overlapping symptoms.[118]

Pharmacologic interventions for neuroprotection and management of neurobehavioral disorders following TBI continue to lack significant scientific evidence in the medical literature. Neuroprotective agents seek to prevent death of neurons after injury. While some agents have shown promise in animal studies, large clinical trials in humans have not revealed strong evidence in support of a single agent. In particular, although thought to be effective in preventing secondary injury after TBI, level I evidence now exists demonstrating that the use of high-dose methylprednisolone in individuals with severe TBI is contraindicated due to increased mortality risk.[119]

In addition, numerous medications have been advocated to help facilitate recovery of functional deficits after TBI. Clinicians should consider the following pharmacologic management: (1) for *arousal*, methylphenidate, amantadine, modafinil, and zolpidem; (2) for *attention and memory*, methylphenidate, dextroamphetamine, amantadine, physostigmine, and donepezil; and (3) for *agitation, irritability, and aggression*, propranolol, quetiapine, clozapine, valproate, and antidepressants.[120]

Posttraumatic seizures (PTS)/epilepsy occur in 2% to 47% of patients with TBI. PTS are typically characterized as *immediate* (within the first 24 hours), *early* (within the first week), or *late* (occurring after the first week). Risk factors for early PTS include intracerebral hematoma, subdural hematoma in children, younger age, severity of injury, and alcoholism, whereas risk factors for late PTS include early PTS, intracranial bleed, severity of injury, and age more than 65 years.[121] Management of seizures is usually achieved with antiepileptic drugs such as phenytoin, levetiracetam, carbamazepine, and valproate, although these should be monitored for their frequent side effects, including fatigue, dizziness, and lightheadedness.[122]

Patients with TBI are also at significant risk for developing HO, DVT/PE, spasticity, bowel and bladder incontinence, and skin breakdown. The reader should refer to discussion of these topics earlier in the chapter.

BURN INJURIES (SEE CHAPTER 51)

Introduction

Burns often occur simultaneously with other traumatic injuries including orthopedic injuries, TBI, SCI, or amputations. Severe disability, altered body image, and numerous physical and medical complications often lead to a diminished quality of life among survivors of burns.[123] Burn injuries account for 40,000 hospital admissions annually in the United States and are the fifth most common cause of unintentional death.[124] Advances in early resuscitation techniques, topical chemotherapy, early wound excision, isolation practices, infection control, antibiotics, and grafting techniques have contributed to the improved survival rates from severe burns.[125]

Classification

Burns are classified by size and thickness. The size of a burn is measured as a percentage of total body surface area, with the *rule of nines* providing a rough estimation of the area that has been burned in an adult. Using this rule, the head and

neck and both upper extremities account for 9% each of the total body surface area, the lower extremities and the anterior and posterior trunk are 18% each, and the perineum and genitalia are 1%. Thus, an estimate of the total body surface area involved in a burn can be quickly calculated. The thickness of a burn is classified as superficial, partial thickness, or full thickness. A superficial burn affects only the epidermis, whereas a partial-thickness burn extends through the epidermis to part of the dermis. A full-thickness burn affects the epidermis and entire dermis and may also involve underlying muscle, tendon, fascia, or bone. [125,126]

Complications

After acute treatment, the focus of burn care shifts to rehabilitation and restoration of function. Attention to the prevention and treatment of the following complications is an important aspect of burn rehabilitation.

HYPERTROPHIC SCARRING

Excessive scarring following burns may result in significant joint contractures and disfigurement, negatively impacting functional capabilities and quality of life. Despite little evidence of its efficacy, the most widespread method of preventing excessive scarring is the application of pressure garments. Other preventative measures include splinting, stretching, and range-of-motion exercises.[127]

WEAKNESS AND PROTEIN CATABOLISM

Following severe burns, a dramatic increase in protein catabolism results in weakness, decreased exercise tolerance, and functional deficits. Treatment and prevention consist of strength training and endurance exercises, which may be effective in increasing strength and function.[128] In addition to exercise programs, treatment with anabolic agents such as oxandrolone has been observed to reduce or prevent weakness in a burn population.[129]

HEAT INTOLERANCE

The ability to regulate and tolerate heat is often decreased following burn injury.[130] The mechanism for heat intolerance may be rooted in changes in central and peripheral thermoregulation; however, studies have suggested the involvement of still yet unknown factors.[131] Despite the uncertainty surrounding the etiology of heat intolerance in burn patients, it must be recognized and incorporated into the rehabilitation program of this population.

PAIN AFTER BURN INJURIES

Pain after a burn depends on the depth of the injury. In a partial-thickness burn, nerve endings in the dermis may remain intact, resulting in significant pain. In contrast, pain may be less or absent in areas of full-thickness burns because of loss of nerve endings. Approximately 35% of burn survivors continue to report significant pain more than a year following the injury (see the earlier section "Pain Management").[125]

AMPUTATION(S)

Despite aggressive limb preservation procedures after a burn, the extent of tissue damage or secondary complications such as infection or ischemia may mandate amputation. The combination of comorbid burns and amputation presents unique rehabilitation challenges, especially with regard to the prosthetic socket interface and skin breakdown.

PSYCHOLOGICAL COMPLICATIONS

Following burn injuries, emotional complications including depression, body image dissatisfaction, and PTSD may complicate recovery and rehabilitation. Furthermore, hypertrophic scarring may result in significant facial or limb disfigurement, leading to significant dissatisfaction with body image and difficulty with community reintegration. In the 1 month after a burn injury, 54% of patients report depression, whereas 43% of patients continue to report emotional distress 2 years after injury.[132] PTSD has been reported to occur in 13% to 25% of patients with burn injuries. Although there is little evidence for specific treatment recommendations, it has been established that "debriefing" sessions may actually be harmful or increase the rate of PTSD following traumatic events.[133] The recognition and treatment of psychological complications of burn injuries are crucial to the design and implementation of a successful rehabilitation program.

AMPUTATION

Introduction

In the United States, approximately 2 million people live with limb loss. Demographic projections estimate that by the year 2050, nearly 3.6 million Americans will be living with major limb loss (1 in 144 persons).[134] These high estimates are due to vascular occlusive diseases, which have been known to cause approximately 82% of amputations and account for nearly 30,000 new cases annually. The majority (>60%) of these patients also have diabetes mellitus.[135,136] Traumatic amputations from war wounds have accounted for over 1700 service members losing a major limb from wounds sustained in either Iraq or Afghanistan.[137] In addition to their distinct mechanism of injury (blast), these injured service members represent a unique patient population when compared to other civilians with acquired limb loss in terms of demographics, location of amputation, and presence of comorbid injuries.[138] Trauma remains the highest contributor to pediatric and upper limb amputations, whether in civilian or military populations. In fact, 68.6% of all traumatic amputations occur in the upper limb. Numerous physical, psychological, and functional challenges accompany major limb loss and create additional challenges for successful rehabilitation and reintegration.

The decision to amputate or salvage a limb depends on the extent of injury and tissue viability. Evidence suggests that, at 2 and 7 years after injury, there are no significant functional outcome differences between those who undergo limb salvage surgery and those who undergo amputation. Limb salvage

patients, however, are more likely to have longer hospital stays, more operative procedures, and a higher complication rate in the short term. Additionally, long-term follow-up of both groups demonstrates a lower quality of life compared with age-matched controls.[139] Individuals with acquired major limb amputation have a higher lifetime incidence of various pain syndromes (eg, residual limb pain, PLP, back pain), skin problems in the residual limb, overuse injuries, cardiovascular disease, and diabetes mellitus.[138] Of particular concern for children with limb loss are psychological acceptance, altered self-image, and the development of bony overgrowth of the amputated limb.

Treatment

For a comprehensive review of amputee care, the reader is referred to the textbook titled *Military Medicine: Care of the Combat Amputee*.[140] Although there have been great advances in the rehabilitation methods and prosthetic devices that are currently available for individuals with major limb amputation, the acute medical and surgical care of these patients has longlasting implications for community reintegration and quality of life. Decisions such as optimizing limb length, balancing muscles, appropriately managing transected nerves, and achieving adequate soft tissue coverage are fundamental surgical principles that will greatly affect prosthetic fitting and training. Aggressive acute pain management, physical and occupational therapy, and balance and gait training, along with reduction of cardiovascular risk and nutritional counseling, will likely reduce long-term complications. Introduction of a prosthesis early during the course of treatment is essential, especially for individuals with upper limb loss, in order to promote lifelong bimanual activity and help avoid overuse injuries of the intact limb. Prosthetic fitting and training for children with amputation(s) are especially challenging and should be guided by the child's developmental milestones (eg, reaching, standing, and walking). It is important to introduce play, sport, and recreational activities with appropriate adaptive equipment to help facilitate socialization and reintegration into the community as well.

PERIPHERAL NERVE INJURIES AND COMPLICATIONS

Introduction

Traumatic injuries to the peripheral nervous system may contribute to a significant loss of function and independence, which may not be readily evident during the initial trauma screen. Peripheral nerve injuries may occur as a complication of medical care. Reported causes have included improper bed positioning, compressive casts, poor-fitting orthotics, excessive pressure during surgery, or inadvertent needle sticks. Common sites of nerve injury include the brachial plexus, ulnar nerve at the elbow, and peroneal nerve at the fibular head.

Cognitive impairment or injury to the spinal cord may limit the ability to fully assess the peripheral nervous system. In addition, confounding conditions such as a critical illness neuropathy or myopathy may contribute to sensory or motor dysfunction, especially for patients requiring extended intensive care. Electrodiagnostic testing may be helpful in assessing the presence and extent of peripheral nerve damage as well as establishing the prognosis for recovery. Typically, nerve injuries are classified as either complete or incomplete. *Neuropraxia* refers to an incomplete injury, characterized by demyelination, and has an excellent prognosis for recovery. Damage to the axon (*axonotmesis*) signifies a more severe injury with a poorer prognosis. Axon regeneration can be estimated to occur at a rate of 1 to 5 mm/d or 1 inch per month.

Prevention and Treatment

A discussion of repair or grafting for peripheral nerve injuries is beyond the scope of this chapter; however, if repair is undertaken, proper postoperative positioning, splinting, and activity precautions should be explained to the patient and treatment team to help support healing and prevent damage. Peripheral nerve care and injury prevention require serial neurologic examinations, attention to patient positioning, and frequent monitoring of pressure-sensitive areas. Positioning is particularly important during surgical procedures. Frequent turning by nursing staff may be necessary if the patient's injuries do not allow independent changes in position. Special attention is required when placing casts or external fixation devices. The use of ultrasound to guide interventional procedures may help to avoid inadvertent iatrogenic injuries.

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Trauma, Medicine, and the Law

Kenneth L. Mattox • Carolynn Jones

KEY POINTS

- When in doubt and time and life are running out, *treat.*
- Have specific instructions for judicial intervention and emergency psychiatric detention available in the trauma center.
- Be mindful of Hippocrates's admonition, "Which ought not be spoken of abroad, I will not divulge, as reckoning that all should be kept secret."
- Compliance with local reporting laws is essential to avoid potential criminal penalty and civil liability.
- The police department's duty is containment and control, whereas the trauma team's duty is care and cure. To each his own.
- Avoidance of malpractice claims depends on the exercise of skill based on knowledge of reasonable, ordinary, prudent physicians under similar circumstances.
- An unfavorable outcome does not, of necessity, imply or result in a legal lawsuit; if such were the case, 50% of all attorneys in court cases would be so guilty.
- Standing orders, best practices, protocols, guidelines, and electronic recommended practices all have value, but the ultimate hallmark of the professional is the exercise of sound judgment in any particular case.
- Records must be made for the patient's benefit, not for the attending physician, hospital, attorneys, or quality surveillance. Corrections to records can be made, dated, and timed, but the original entry must not be removed.
- Freely use consultation and escalation of concerns when indicated. There are always persons, processes, and resources to provided unknown, unfamiliar, or clarified information.

INTRODUCTION

Interactions between the medical and legal professions increase in both complexity and number with each passing year. No longer is the focus only on fear of being sued, testifying in court, and preparing for a deposition. In-hospital legal counsels, now in most hospitals, add another dimension and play a significant role in any medicolegal situation. This chapter will focus on the major concerns a physician must have when treating an injured patient—the intersection/interaction among trauma, forensic medicine, and the law. The trauma patient with physical and emotional injuries can bring legal issues, some of which can be more challenging to the trauma team than the clinical problems. Legal issues can range from civil rights to criminal law, and the team needs to have some basic knowledge to appropriately address medicolegal issues that may arise. Despite differing and ever-changing laws from state to state, some basic guiding concepts can be set forth.[1]

CONCEPTS, PRINCIPLES, AND DEFINITIONS

It should be noted that legal and medical definitions or concepts may differ and that such variances are important to medical personnel interacting with forensic and legal personnel, especially in a courtroom, deposition, or adversarial situation. Some general definitions are as follows:

- Abandonment—Terminating care of a patient without assuring that a continuum of the same or higher level of care exists.
- Assault and/or battery—Unlawful touching of a person or patient without appropriate consent for that contact.
- Confidentiality—Prohibits the health care provider from disclosing information about the patient's case to others without permission. Also, providers and health care systems are encouraged to take steps to ensure that only authorized access occurs, which, in this era of the

electronic medical record, can be challenging. Additionally, state law requires reporting certain communicable/infectious diseases, as well as suspected cases of child, dependent adult, and elder abuse, and gunshot wounds. What is reportable can change, and the health care provider must be cognizant of requirements where he or she practices.

- Competence—Ability of a patient to understand the questions put to them by health care personnel and to understand the implications of the treatment recommendations and decisions made.
- Negligence—Deviation from an accepted standard of care that would be rendered by a similar practitioner under similar circumstances.

Duty to Treat

In the "modern" version of the Hippocratic Oath, the phrase **"…there is the obligation to all my fellow human beings, to treat those of sound mind and body as well as the infirm"** establishes the tone of the regulations for a duty to treat any patient who presents with an immediate, life-threatening condition.[2] Wide interpretation and discrepancies exist regarding "duty to treat," depending on urgency of clinical condition, availability of appropriately skilled clinicians, availability and infrastructure of the treating facility, and availability of a higher level of care within the geographic area. In addition, the availability of funding for hospitals and clinicians has resulted in local, state, and federal regulations to protect patients, practitioners, and hospitals. Once a patient enters a treatment facility and is found to have an immediate, life-threatening condition on initial evaluation, the obligation to continue the treatment is understood, as is the now established physician–patient and hospital–patient relationship. That relationship continues until physician or patient terminates, by mutual consent, until services are no longer needed, or until the physician properly withdraws from the relationship.[3] The Emergency Medical Treatment and Active Labor Act (EMTALA), passed by Congress in response to hospitals refusing to treat patients based on their ability to pay, requires hospitals and physicians to provide a medical screening examination and stabilizing treatment for patients who present with an emergency medical condition to a hospital with a dedicated emergency department.[3,4] The physician's duty to treat and/or stabilize the patient for transfer to a facility with a higher level of care is clear. Should a physician misrepresent a patient's condition to facilitate a transfer or violate any requirements of EMTALA, civil penalties exist, including fines and exclusion from participating in Medicare and Medicaid programs.[3,4] The Americans with Disabilities Act of 1990 prohibits the denial of an individual's access to health care based on a disability, unless providing said care poses a direct threat to the health and safety of others that cannot be eliminated by adequate precautions.[3,5] Implicit in these laws is that inability to pay is not an acceptable (or legal) reason for nontreatment of any patient presenting with a life-threatening condition. While implicit to all emergency facilities, this is especially pertinent to designated trauma centers of all levels that provide care to critically injured patients.

The duty to treat a patient does not change, even if the patient is the perpetrator of a violent crime. The fact that the patient was injured or injured others while driving intoxicated or while wielding a gun is irrelevant. The physician has a duty and ethical obligation to treat the patient, regardless of the situation surrounding the injury.

Consent

Numerous terms and concepts apply to the many consent issues involved in patient care. Appropriate informed consent protects the legal rights of patients and guides the ethical practice of medicine. Since the risk of litigation often depends on patient dissatisfaction due to lack of communication with the physician, strong practices for informed consent may actually prevent such suits from being filed.[6,7] *The informed consent process should be documented thoroughly, using an electronic medical record, procedure-specific consent forms, patient education materials, and other options whenever possible*.[7] Except in urgent life-threatening situations, at its simplest, consent is required whenever a health care provider intends to render care to a patient.[6] The physician's (and hospital's) duty extends beyond obtaining a patient's signature. Informed consent includes providing enough knowledge to the patient about the procedure, the risks and benefits, and any alternative treatments for the patient to make an "informed" decision.[8,9] As outlined by The Joint Commission, this process has several components, which include the nature of care, medications, procedures, possible risks and benefits, and any limitations on confidentiality of information learned from or about the patient.[10] As one enters an emergency center or is admitted to the hospital, consent to be evaluated and treated is usually obtained. Many patients do not completely read the "fine print" above this simple signature block and are often in a hurry to get on with an emergency evaluation. Beyond this simple entrance signature, additional informed consent is required for more detailed treatment (such as an operation) once that more complex treatment is known to be required.

EXPRESS CONSENT

A patient directly communicates his or her permission for recording a history, having a physical examination or tests, and treating the conditions discovered. The doctor or other knowledgeable health care worker must describe the reason for the examination and treatment, the treatment to be rendered, the risks of such treatment, and possible side effects and complications. The consent form should include the physician's name, location of the treatment, and date and time of treatment, as well as a description of the treatment. Oral (without any written or electronic documentation) consent is difficult to prove/defend in a court of law.

IMPLIED CONSENT

In contrast to express consent, implied consent is when a patient's actions and the facts and circumstances of the situation act as the patient's consent for the medical treatment. For example, a patient who extends their arm to have blood drawn has given presumed or implied consent in an emergency situation[8]; as another example, when a patient presents with traumatic injuries and cannot understand or communicate and a family member cannot be reached, it is assumed that consent to lifesaving emergency treatment would be desired. Of course, if this patient should present with a living will or an advanced directive specifically requesting that no heroic lifesaving measures be undertaken, there is no implied consent.[10] Often, only after a blood transfusion has begun, does the team become aware that the patient or his or her family is opposed to such intervention. In these cases, the intervention is stopped, and the situation is carefully documented. The members of the treating team should, if time permits, attempt to ascertain the presence of any advanced directive or official treatment requests.

In obtaining consent, there are several considerations for the surgeon. In certain circumstances, minors, for example, may provide consent without parental involvement.[11] Individual state regulations also outline other situations in which minors may give consent, such as treatment for sexually transmitted infections, abortions, contraception, and prenatal care.[11] It is incumbent upon health care providers to know the laws and policies of their state and hospital.

WAIVER OF CONSENT

In certain cases of emergency care, federal regulations allow the local institutional review board to approve a consent procedure, which does not include, or which alters, some or all of the elements of informed consent, or to waive the requirement to obtain any informed consent. Most complete waivers of consent involve studies in which there are minimal risks to subjects, but complete waivers are also possible in emergency care and a few other circumstances. In such circumstances, it is appropriate to inform the patient and the family as soon as possible after the emergency and after treatment has been rendered. The surgical team should then obtain written documentation that the patient and/or family understands and gives consent, assent, or understanding.

SURROGATE CONSENT

Both state and hospital policies define who (and in what priority) may give informed consent in defined circumstances for patients whose clinical condition does not allow them to speak for themselves or understand the implications of the information given to them. In the event a patient lacks the capacity to consent, health care decisions may be made by the patient's legal representative such as a parent, family member, guardian, or another with medical power of attorney.[8] Hospital policies define the priority of surrogate consent, and these policies usually reflect local or state laws.

Competency in medicine is different from the legal connotation. Competency or capacity refers to the patient's ability to understand what is best and right for him or her, and the decision is autonomous.[10] Competency is generally not an issue unless the patient suffers from a cognitive deficit, such as Alzheimer disease. Substance abuse or withdrawal from a substance that has caused altered mental status will affect competency, as well.[9] All attempts should be made to obtain consent. For example, hospital administrators may assist when there is an issue with competency and no patient representative is available. Under such circumstances, an *ad litem* (court-appointed) representative of the patient might be considered. Local, state, and federal regulations exist defining the process and payment for *ad litem* representation.

REFUSAL OF CONSENT

The adult patient who fully understands the implications of his or her decision has the right to refuse to be examined, to be admitted to undergo laboratory tests or procedures, or to be treated in other ways.[10] Under some circumstances, local law or hospital policy requires the local ethics committee or a legal *ad litem* attorney to become involved, especially if the adult refusing treatment has minor children or is mentally incompetent. When evaluation and/or treatment is refused, it is important to carefully and completely document the conditions of the refusal and the various persons involved in advising and interacting with the patient. While troubling and frustrating for the physician, the right to refuse treatment must be recognized and respected. Refusal to consent is usually reserved for a patient who has reached majority. In the case of a parent issuing a "refusal to consent or treat" for a minor child, the legal system often becomes involved with hospital administrations and physicians to determine what is permissible. In refusal to consent situations, the physician must consider competency a subjective assessment, and judgment of such falls to medical professionals.[12] The physician must ensure all information is provided and any questions answered so the patient or surrogate has the data to make an informed decision.

Health Insurance Portability and Accountability Act, the Electronic Medical Record, and Social Media

Federal law (Health Insurance Portability and Accountability Act [HIPAA]), state laws, and hospital policy protect patient privacy, hospital medical records, and physician confidentiality in matters of peer review. With the introduction of the electronic medical record (EMR) and ever-expanding availability of social media networks/devices, new and stricter surveillance and control have become necessary to assure privacy and confidentiality.

All hospital and personal electronic devices (eg, computers, smart phones, smart pads) have the capacity for misuse and transmission, deliberately or accidentally, of sensitive patient or medical staff information to public social networks or

hackers. Internal controls must be in place for surveillance, detection, and rapid response if violations are discovered.

Many hospitals have developed policies and protocols to allow, and even encourage, use of smart phones to take appropriate photographs of forensic, intraoperative, pathologic, and other material that is clinically significant. Software and "apps" exist that then allow secure transfer of this data to the EMR, with subsequent deletion of the material from the original source. Health care providers must be aware of HIPAA social media rules and should receive training specific to same.

Forensic and Medical Implications

Forensic and legal issues surface daily in trauma care but may be overlooked or unidentified due to the urgency of the situation. Health care providers are able to and, indeed, need to provide lifesaving measures and "think forensically" at the same time. By considering the forensic and legal implications, evidence that may be vital to the outcome of a legal case is preserved without impeding appropriate medical care.

Trauma team and law enforcement activities repeatedly intersect in various locations for a variety of reasons including system and organizational regulation and review, patient complaints and undesirable results, and quality and peer review, to name a few.

Each year, over 1.6 million lives are lost worldwide to violence, and countless others sustain injury.[1] By understanding the law and the health care forensic implications of the law, identifying and preserving evidence during the course of evaluation and treatment, and accurately describing wounds, the physician is an indirect advocate for the patient and may provide information that will be used by law enforcement and the justice system.

The very nature of traumatic injuries leads one to consider forensic or legal implications. For example, preserving the clothing of a gunshot victim may be critical to the outcome of courtroom proceedings. Obtaining blood samples from a potentially intoxicated driver is considered essential in some political jurisdictions. The local laws concerning a hospital or physician's rights and responsibilities in ordering laboratory data for the sole purpose of obtaining forensic evidence are inconsistent, and clinicians must be cognizant of hospital, local, and state regulations. Traditionally, forensic issues have not received much, if any, priority, since lifesaving measures are the primary objective; however, physicians and other providers are quite able to render necessary care while also taking care to appropriately preserve evidence.

Forensic Nursing and Trauma Systems

Proper evidence collection and preservation may be accomplished with the assistance of forensic nursing. The forensic nurse is specially educated and trained in both the health care and legal arenas and provides direct services to patients and providers.[13] Those include injury identification and documentation, forensic photography, evidence collection, death investigation, and addressing the psychosocial needs of the patient and family. The forensic nurse may be present in the trauma room to collect and preserve evidence and document injuries through photographic means or using body diagrams.[14,15] For example, clothing that is removed is handed to the forensic nurse, who places and appropriately labels each piece in separate containers to prevent contamination. In addition, bullet fragments removed during surgery may be given to the forensic nurse, who ensures chain of custody and releases the evidence to the investigating agency.

Patients presenting with traumatic injuries may not be able to provide a history of the precipitating event. The forensic nurse anticipates the types of evidence that need to be preserved, and his or her presence in the trauma room allows the surgeon to focus on lifesaving measures, while confident that evidence is appropriately preserved. Information including photographs obtained for forensic purposes is logically available during the trauma quality review process. Also, once identification markers have been removed (HIPPA requirements), such materials might also be very helpful in clinical scientific preparations for publication.

Injury Identification

The trauma center evaluates injuries and underlying nontrauma conditions, assesses risks, and classifies and grades injuries. This latter task using an established injury classification schema such as those developed by professional organizations (American Association for the Surgery of Trauma, American College of Surgeons, and others) may impact later legal actions. It is not always immediately clear, however, that a wound is caused from a blunt or penetrating source or even the identity of a specific wounding agent. Therefore, the final coding by the trauma service is determined at the end of the hospital admission.

For cases involving gunshot wounds when it is known or suspected the patient may have been the shooter, bagging the hands for later examination by forensic investigators is advocated, if possible.

When bullets and fragments are removed during emergency treatment, they are evidence. In preserving the evidence and assuring appropriate "chain of evidence," there are several cautions and principles to follow when removing a metallic foreign body:

- Avoid using a metal instrument to extract the metal foreign body. Metal on metal creates "scoring" on the missile that might distort the forensic evidence.
- Avoid dropping a metallic missile into a metal container for the same reason. The foreign body should be gently placed into a plastic specimen cup available in hospitals, assuring that patient labels are on the side and cap of the specimen cup.
- Do not scratch your initials and the date on the foreign body. Use appropriate/approved methods of documentation, in accordance with the established hospital policy of managing forensic evidence and the chain of custody.

- The physician removing the foreign body should record the details of the foreign body removal, to whom the missile was given, and the instructions given to this individual.
- The person *receiving* the foreign body and placing it into the plastic cup should also document the same in the record, as well as to whom the container was given (hand-off).
- The exact process and the offices where forensic evidence is maintained are determined by each hospital and clearly stated in that hospital's "Chain of Evidence" policy.

With victim's clothing, avoid cutting through defects or holes, which can destroy evidence. Cuts should be made around the defect or along the seam, and individual items of clothing should be placed in separate paper bags. The top of the bag is then folded over, sealed with tape, and labeled with the examiner's initials. If available, the forensic nurse can expedite and assure proper procedure in handling, photographing, packaging, and labeling clothing. Once bagged, the clothing will be released to law enforcement or stored in a locked area of the hospital.

INTERACTIONS WITH UNIFORMED OFFICERS (POLICE, FIREMEN, OTHERS)

In the course of their normal activities, law enforcement and medical professionals will find themselves in juxtaposition and occasional confrontation as to who has specific authority in some instances. Even with existing policy from local or state law, law enforcement agencies, hospitals, or state medical boards, it is probable that such laws or policies may be in opposition or have different interpretations.

Blood Specimen Collection for Law Enforcement Purposes

In some instances, the patient is the alleged perpetrator of a crime. Primarily for legal rather than clinical reasons, there is a need to identify any illegal substances or high levels of alcohol, since toxicology results impact charges and sentencing of defendants. Forensic toxicology studies are then performed in crime laboratories where assays identify and quantify submitted specimens. It should be remembered that obtaining specimens for legal reasons is different from routine laboratory testing. Local and state laws differ with regard to the need for permission from a patient to obtain blood, urine, tissue, or exhaled air specimens. All members of the treating team should know the dictates of local/state law, as well as related individual hospital policies. When laws and/or policies do mandate consent from a patient/surrogate consent, the physician must assure that the patient's cognitive condition allows for informed consent. Some states have mandatory requirements, while others require a search warrant or court order for collection of specimens.[16]

Blood samples should be obtained within 3 to 4 hours of a trauma incident to provide accurate blood alcohol content, and it is critical to assure that time of specimen collection is accurately recorded.[16] Commercial specimen collection kits are available to aid in collection and chain of custody.

Chain of Custody

Chain of custody is the "paper trail" that documents each person who has custody and control of a particular piece of evidence or a specimen. The chain begins with the person who collects the evidence and continues with each subsequent person who handles the evidence until it reaches the crime laboratory. The following information must be documented in the medical record[16]:

- Each person who handled the specimen(s)
- Name and badge number of the officer receiving the specimen/evidence
- Other law enforcement agency personnel who might be present
- Date and time the specimen/evidence is released

Failure to maintain and appropriately document the chain of custody opens the door for attorneys to dispute the validity of the evidence.

Sexual Assault Examinations

Victims of sexual assault most often seek examination in a trauma center or emergency department. During the examinations, collections are made from physical evidence, and biologic specimens are obtained.[17] Such collections are saved per protocols and submitted to law enforcement officials. The responsibility of the trauma service is to assure that the patient's clinical needs are met and that appropriate collections are performed to comply with the hospital's protocols for sexual assault examinations.[17-19]

Officers Entering Health Facilities

Uniformed officers may be in a health facility as contract security officers, because they were called by hospital personnel, or because there is a personnel safety issue. Hospital operations and policies are usually under the direction of the administration and the medical staff. When uniformed officers enter the buildings under emergency conditions, however, the duty of emergency management shifts to the uniformed officers with tight communication and input from the hospital's incident command.

Fire Alarms

Fire in any public building is a crisis, whether notification is caused by a public alarm or an internal fire box alarm. Such an alert or alarm from a hospital prompts an immediate response from local and regional fire stations. Then, fire chiefs and police authorities enter the hospital with practiced protocols in action. The fire chief who responds to a hospital

fire alarm is in charge of hospital operations and functions and will issue safety orders to maximize employee and patient safety.

Active Shooter in a Public Place

Protocols regarding an active shooter in a hospital are widely distributed and practiced. During such an event, it is local security officers and uniformed police who are responsible for security operations within the hospital and will issue directions that will maximize personnel safety. Ideally, local police officers will have drilled with in-hospital security officers as well as with administrative and clinical personnel, including physicians. There have been ethical and moral concerns from physicians and nurses regarding abandoning helpless patients with the "run, hide, fight," policy. New collaborative protocols interacting with uniformed officers to produce an initial "contain and control" environment, especially in the emergency center, obstetrics, operating room, and intensive care units, are being investigated and implemented.[20]

Officers Accompanying Prisoners

The laws and rules regarding prisoners' rights do not change because they are in the hospital; however, the prisoner as a patient has the usual rights regarding privacy, confidentiality, and so on.

Undocumented Aliens as Patients

One recent area of concern relates to persons who are in the United States without official or legal visas or papers and who become patients. Concern exists among clinicians that immigration officers might learn of such "undocumented aliens" and "extract" them from the hospital or request that they be dismissed prematurely from the hospital. As in all areas of clinical concern, a well-understood hospital policy should be created regarding the clinical responsibilities for such patients.

LEGAL CONSIDERATIONS DURING DISASTERS

The principles of interaction between medicine and the law should not change in the face of a disaster. The principles relating to licensure, credentialing, and consent continue to apply, even in extreme or special circumstances. The fact that a physician (or other health worker) may be from an outside geographical or political jurisdiction does not remove the organizational and personal responsibility to comply with local credentialing and licensing laws. During disasters, the flow of supplies, equipment, and personnel may be altered, but the responsibility for the physician and the clinical team to comply with professionalism and standard best practices within their capability and resources still applies. Record keeping may be a challenge, but detailed and accurate records must be the undisputed goal during these times.

During a disaster, all personnel working in the hospital are subject to the bylaws and policies of the hospital, as well as state and local law. All questions relating to medical authority, supervision, coordination, and action during the time of a declared disaster in regional shelters are subject to the regional incident command structure and its medical branch. Any outside agency must function under the authority of the regional authority, unless subject to a federal law.

INTERACTING WITH ATTORNEYS

Hospital Bylaws, Committees, Credentialing, and Policies

As part of any hospital, clinic, or health department function, and especially in any trauma care facility, it is axiomatic that surgeons and other trauma team members will interact with attorneys on a frequent basis. Most hospitals, medical schools, and medical facilities have an in-house or consulting legal counsel that assists, among other things, in the preparation of bylaws, policies and procedures, credentialing, outside regulatory review, and the interpretation of local, state, and federal laws. These attorneys are hospital employees, and the interests of the hospital are their primary concern. The medical staff bylaws govern and "belong to" the organized medical staff. As bylaws are revised to adapt to changing regulations and policies, it is imperative that the bylaws committee members review all in careful detail, with the goal of keeping the bylaws in the best interest of the organized medical staff. In some instances, it is advisable for the organized medical staff to employ their own legal counsel to assure that the medical staff's interests have not been compromised when bylaws are written or modified.

Professional Liability, Depositions, and Testifying in Court

Despite best intentions to practice the highest quality of medicine possible, dissatisfaction, complications, and misadventures will occur in all treatment environments. At the hospital level, such events are reviewed during the hospital quality review process, including mortality and morbidity meetings, performance improvement committees, peer review committees, and sentinel event committees. Except for criminal cases, the minutes and proceedings of these reviewing entities are protected from discovery. Physicians should participate in these hospital activities, including reviews of their own cases. In the course of caring for trauma patients, being named in a malpractice suit, being asked to peer review records of another physician, and being asked to testify in court about an injury are not uncommon. On receipt of a subpoena, the first step is to contact your attorney and/or in-house counsel and insurance carrier. From that moment on, talk to no one else about the case without the advice and/or presence of your attorney. Preparatory to a deposition, review all related records to the depth and detail you would in preparing for a board exam.

At the time of a deposition or court testimony, listen carefully to every question asked and answer that question. Do not volunteer any additional information (even when sorely tempted)! If you do not understand the question, so indicate. If you are interrupted by the judge or an attorney, stop talking (in mid-sentence, if necessary) and listen carefully to the nature of any objection or interruption. Do not continue until your attorney or the judge advises you to do so. You may ask that a previous response or question be read back to you by the court recorder. If you have authored or co-authored published articles, be prepared to defend your writings.

The two categories of witnesses are "fact" and "expert." A fact witness is called to enter into the record facts as they remember them, discover them in the medical record, or otherwise glean information. An expert witness is certified by the court as having special knowledge and is called upon to elaborate on information pertinent to the case from the vantage of his or her special knowledge, background, and experience. An expert witness might also be a fact witness, but not usually vice versa. Again, the expert witness must be prepared to have all he or she ever said, wrote, or did come under very careful scrutiny.

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V

MANAGEMENT OF COMPLICATIONS AFTER TRAUMA

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57

Principles of Critical Care

Lena M. Napolitano • Raul Coimbra • Jay Doucet • Leslie Kobayashi

KEY POINTS

- The primary goal of critical care in the trauma patient is restoration of hemodynamic stability and organ function via continuation of early trauma care, hemostatic resuscitation, organ support, and specific injury management.
- The ultimate goal of optimal intensive care unit (ICU) care is to achieve zero preventable deaths after injury.
- High-intensity ICU physician staffing is associated with significantly reduced ICU and hospital mortality and significantly lower failure to rescue rates.
- Although Advanced Trauma Life Support has standardized trauma care in the initial hour after injury, there is little standardization subsequently in the ICU.
- ICU admission order sets should be used to optimize early ICU care on admission.
- Damage control resuscitation and massive transfusion protocols decrease mortality from hemorrhagic shock, which is the most common cause of early mortality in trauma.
- National evidence-based guidelines should be used to guide postinjury ICU care.
- Daily goals checklist and the ABCDEF Bundle enhance ICU patient safety and progress.
- All preventive strategies should be implemented to prevent hospital-acquired infections.
- Traumatic brain injury management is based on the Brain Trauma Foundation guidelines.
- Septic shock has a mortality rate of 40%; the Surviving Sepsis Campaign (SSC) guidelines and 2018 SSC bundle provide recommendations to improve outcomes.
- Acute respiratory distress syndrome (ARDS) has a high mortality rate, and use of national ARDS guidelines reduces mortality.

INTRODUCTION

We have had great advances in the care of the injured patient from prehospital and emergency department care to care in the intensive care unit (ICU). Care in the ICU is designed to provide optimal resuscitation, reestablish homeostasis, and minimize secondary and iatrogenic complications (particularly organ failure). Over the past 20 years, critical care has matured greatly, resulting in dramatically higher survival rates for our critically injured patients. Excluding early deaths in the operating room, most trauma hospital deaths will occur in the ICU. In the ICU, the outcome of critically injured patients is dependent on a solid understanding of the pathophysiology and evolution of traumatic injuries, optimal postinjury resuscitation, and prevention of complications. Meticulous attention to detail in all ICU care provided to trauma patients is essential if we are to achieve the ultimate goal of *zero preventable trauma mortality*.

MORTALITY IN TRAUMA

Despite an organized system of trauma care in the United States, the number of trauma deaths increased by 22.8% from 2002 to 2010 for those age 25 years and older, with a concurrent increase in the US population of 9.7%. The largest increase in trauma deaths was in the 50- to 60-year-old cohort.[1] The two most common causes of early trauma mortality are hemorrhagic shock and traumatic brain injury, both of which require extensive ICU care. The 2016 National Academies of Sciences, Engineering, and Medicine report, "A National Trauma Care System: Integrating Military and Civilian Trauma Systems to Achieve Zero Preventable Deaths After Injury,"[2] presents a vision for a national trauma care system driven by the clear and bold aim of zero preventable deaths after injury and minimal trauma-related disability.

Standardization of evidence-based ICU care in trauma is of vital importance with a goal to improve trauma outcomes.

Studies have documented low compliance rates (10%–40%) with standardized protocols in trauma. It has been confirmed that major deviations from guideline-based clinical care are associated with significantly higher mortality after injury and increased organ failure.[3] The implementation of standard operating procedures (SOPs) in the Glue Grant documented significant improvements in morbidity and mortality rates with increasing compliance with published SOPs.[4] Although we have standardized trauma care in the initial hour after injury with use of Advanced Trauma Life Support, there is little standardization subsequently.[5]

Although major quality improvement initiatives have been undertaken at the national level to improve care at trauma centers (American College of Surgeons [ACS] Trauma Quality Improvement Program [TQIP]), major variations in outcomes between trauma centers persist.[6] The fact that some hospitals perform better than others suggests that some patients are not receiving the best possible care, which may result in preventable deaths. A recent study estimated that if all hospitals delivered the highest quality of care to trauma patients, an estimated 167,746 lives (95% confidence interval [CI], 164,534–170,861 lives) could potentially be saved. This report focused on preventable in-hospital trauma deaths, which were presumed due to differential quality of care.[7]

Although evidence-based practice guidelines are recognized to minimize unwarranted health care variation and promote best practices, guideline publication alone does not guarantee practice change. The best method to improve in-hospital and ICU trauma care is to implement patient care protocols and bundles of care developed from evidence-based guidelines.

In the past several years, increasing emphasis has been placed on quality of care indicators and physician staffing models for ICUs. A modern surgical ICU provides evidence-based critical care using protocols, algorithms, clinical practice guidelines (CPGs), and checklists; uses cutting-edge technology for physiologic monitoring; and has a robust quality improvement process to continuously evaluate its outcomes and identify opportunities for improvement. This chapter reviews principles of critical care essential to the management of the injured patient and important critical care and trauma national and international guidelines to provide optimal evidence-based ICU care to improve trauma patient outcomes.

PHYSICIAN STAFFING AND MODELS OF CARE IN THE INTENSIVE CARE UNIT

A growing body of scientific evidence suggests that quality of care in hospital ICUs is strongly influenced by (1) whether "intensivists" are providing care, and (2) how the staff is organized in the ICU. Intensivists are familiar with the complications that can occur in the ICU and, thus, are better equipped to minimize errors.

Several patterns of ICU organization are currently in use. The closed ICU model relies on an attending intensivist and critical care team for ICU patient care. Comprehensive management including responsibility for all orders and procedures is assumed by the ICU team, with other services providing care as consultants as required by the clinical condition of the ICU patient. In the open ICU model of care, individual physicians manage their ICU patients, depending on their institutional privileges. Consultative involvement of a board-certified intensivist is at the discretion of each primary attending physician and is not required or expected. Many ICUs use a semiopen, collaborative care model in which the ICU is staffed with attending intensivists and an ICU team who are responsible for writing ICU orders, but who also coordinate care and communicate closely with primary surgeons and their teams. There are often specific areas of designated critical care autonomy, such as the management of mechanical ventilators, invasive hemodynamic monitoring, pain management, and sedation. Although the ultimate responsibility for the patient remains with the primary attending surgeon, ICU patient care is a collaborative effort. This model combines the advantages of critical care expertise for trauma and surgery patients while maintaining primary surgical service responsibility for overall patient management. This is consistent with the Guidelines for the Optimal Care of the Injured Patient recommended by the ACS Committee on Trauma for Level I trauma centers.[8]

There is now substantial evidence examining the relationship between ICU staffing models and patient outcomes. Mortality rates are significantly lower in hospitals with closed ICUs managed exclusively by board-certified intensivists. A recent distinction has been made between high-intensity and low-intensity physician staffing ICU models. A high-intensity model involves 24/7 dedicated ICU physician staffing and mandatory ICU team involvement with patient management and includes closed ICUs and most semiopen ICUs. In contrast, open units typically use low-intensity ICU physician staffing. In a meta-analysis of 26 studies, high-intensity ICU physician staffing was associated with significantly reduced hospital and ICU mortality (relative risk [RR], 0.71 [95% CI, 0.62–0.82] for hospital mortality; RR, 0.61 [95% CI, 0.5–0.75] for ICU mortality) for both adults and children.[9]

Similar results were found in a study of the effect of high-intensity ICU staffing on outcomes following major trauma.[10] Using prospective cohort data from 68 trauma centers and after adjusting for differences in baseline patient characteristics, the RR of death in intensivist model (closed and semiopen) ICUs was 0.78 (95% CI, 0.58–1.04) compared with an open ICU model. The effect was greatest in the elderly (RR, 0.55; 95% CI, 0.39-0.77), in units led by surgical intensivists (RR, 0.67; 95% CI, 0.50–0.90), and in designated trauma centers (RR, 0.64; 95% CI, 0.46–0.88). The authors concluded that injured patients are best cared for using an intensivist model of dedicated critical care delivery, a criterion that should be considered in the verification of trauma centers.

The mortality rate among surgical patients who develop complications, called *failure to rescue*, is also reduced by the presence of intensivists in the ICU. Failure to rescue is a quality measure that looks at the hospital mortality rate of surgical patients with complications. Hospitals with intensivists on their rapid response teams or in their ICUs have lower failure

TABLE 57-1: Leapfrog Group Intensive Care Unit (ICU) Physician Staffing (IPS) Standard

Hospitals fully meeting the IPS Standard will operate adult or pediatric general medical and/or surgical ICUs and neuro-ICUs that are managed or co-managed by intensivists who:

1. Are present during daytime hours and provide clinical care exclusively in the ICU; and,
2. When not present on site or via telemedicine, return notification alerts at least 95% of the time (1) within 5 minutes and (2) arrange for a physician, physician assistant, nurse practitioner, or a FCCS-certified nurse to reach ICU patients within 5 minutes.

FCCS, Fundamental Critical Care Support.

Source: Reproduced with permission. Copyright © The Leapfrog Group 2019.

to rescue rates.[11] The presence of surgical intensivists also reduces complications and improves National Surgical Quality Improvement Program general surgery quality measures.[10]

Current ICU staffing requirements for hospitals with adult or pediatric ICUs were developed and implemented by The Leapfrog Group, recommending the ICU Physician Staffing (IPS) Standard (Table 57-1).[12] Unfortunately, only 47% of hospitals reporting in the 2015 Leapfrog Hospital Survey have the recommended intensivist coverage in place and met the Leapfrog IPS Standard.

CONTINUOUS QUALITY IMPROVEMENT IN THE SURGICAL INTENSIVE CARE UNIT

Quality assurance and performance improvement in a surgical ICU are complex processes requiring the ongoing identification of outcome measures or performance indicators, data collection and analysis, and the development of action plans to correct deficiencies and subsequent monitoring. The underlying goal of delivering high-quality ICU care also depends on specialization (critical care specialists), provider education and training, excellent communication, and collaborative interaction between specialty and ancillary services. Existing critical care quality assurance programs have used a variety of clinical indicators or "filters" as a measure of the quality of care. These indicators may reflect process measures (eg, percentage of eligible patients receiving venous thromboembolism [VTE] prophylaxis in a timely manner) and outcome measures (eg, number of central line-associated bloodstream infections or ventilator-associated pneumonia cases). Illness severity indices have been developed for risk adjustment to aid in predicting outcomes in critically ill patients and are used as benchmarking tools, allowing participating units to compare their actual ICU outcomes with those predicted.

The ACS TQIP[13] is a recent addition to the many quality improvement collaboratives that have been established in surgery. Now in its ninth year in 2018, ACS TQIP uses risk-adjusted benchmarking to provide over 800 participating hospitals with accurate national comparisons.[14] Aggregate outcomes by center are reported, as well as outcomes in two distinct subsets: multisystem blunt trauma and penetrating trauma. Additional TQIP reports focus on patients with traumatic brain injury (TBI) or shock or the elderly population. TQIP reports regarding the processes of care are also provided, including the use of intracranial pressure (ICP) monitors for TBI patients, the use and timing of angiography for shock patients, and timing of VTE pharmacologic prophylaxis.

In addition to improved instruments for assessing critical care outcomes, the development and implementation of clinical protocols and CPGs directed at reducing undesirable treatment variability are being linked to ICU performance improvement. Protocols and guidelines, once developed and implemented, can later be analyzed in terms of their clinical efficacy and cost-effectiveness and can be modified further to improve efficiency.

The effectiveness of CPGs has been demonstrated for a variety of conditions including ventilator weaning, pneumonia, nutrition, and sedation. Most institutions experience difficulties with the implementation of CPGs, since it requires the development of brief protocols or bundles of care. Current methods of improving implementation and compliance with CPGs include ongoing education, defined order sets, the assignment of some management responsibilities to specialized teams (eg, nutrition and respiratory therapy), and the use of advanced practice providers (nurse practitioners and physician assistants) in the ICU.

Several fields in medicine, motivated by an increased awareness of errors in medical care delivery, have incorporated lessons learned from aviation to clinical practice to improve quality and patient safety. A checklist is just one of the tools used in the aviation industry that have been tested in many ICUs to potentially improve safety, quality, and consistency of ICU care.[15]

Daily Goals Checklist in the Intensive Care Unit

A daily goals checklist for morning ICU rounds is an excellent aid to ensure optimal communication among all ICU personnel. A recent study examined the use of a daily goals checklist in a 15-bed closed ICU in a tertiary care, university-affiliated hospital that provided care for medical-surgical ICU patients. The daily goals checklist was perceived to improve the management of ICU patients by creating a systematic, comprehensive approach to patient care and by setting individualized daily goals. The daily goals checklist also enhanced patient safety and daily progress by improving interprofessional communication and practice and encouraging momentum in recovery from critical illness. Daily goals checklist review also prompted teaching opportunities for multidisciplinary learners on morning rounds.[16]

A checklist using the mnemonic FASTHUG (feeding, analgesia, sedation, thromboembolic prevention, head of the bed elevation, stress ulcer prophylaxis, and glucose control), which is reviewed daily during multidisciplinary ICU rounds to ensure protocol adherence, has been effective.[17] Similarly,

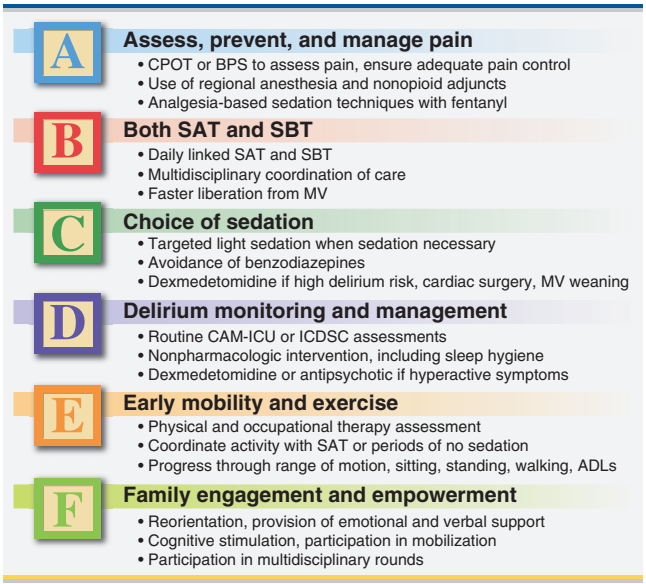


FIGURE 57-1 The ABCDEF Bundle. ADLs, activities of daily living; BPS, Behavioral Pain Scale; CAM-ICU, Confusion Assessment Method for the Intensive Care Unit; CPOT, Critical Care Pain Observation Tool; ICDSC, Intensive Care Delirium Screening Checklist; MV, mechanical ventilation; SAT, spontaneous awakening trial; SBT, spontaneous breathing trial.

implementation of the Pain, Agitation, and Delirium (PAD) Guidelines is facilitated in many ICUs via the ABCDEF Bundle (Fig. 57-1).[18] The ICU Liberation Collaborative[19] is a real-world large-scale quality improvement initiative to implement the Society of Critical Care Medicine (SCCM) PAD Guidelines across 76 ICUs using the ABCDEF Bundle.

In a study of 6064 patients in seven community hospitals, implementation of the ABCDEF Bundle was associated with significantly improved outcomes. For every 10% increase in total bundle compliance, patients had a 7% higher odds of hospital survival (odds ratio [OR], 1.07; 95% CI, 1.04–1.11; P <.001). Likewise, for every 10% increase in partial bundle compliance, patients had a 15% higher hospital survival (OR, 1.15; 95% CI, 1.09–1.22; P <.001). These results were even more striking in a sensitivity analysis removing ICU patients identified as receiving palliative care (12% and 23% higher odds of survival per 10% increase in bundle compliance, respectively; P <.001). Patients experienced more days alive and free of delirium and coma with both total bundle compliance (incident rate ratio, 1.02; 95% CI, 1.01–1.04; P = .004) and partial bundle compliance (incident rate ratio, 1.15; 95% CI, 1.09–1.22; P <.001).[20]

STANDARD TRAUMA/SURGICAL INTENSIVE CARE UNIT ADMISSION PROCEDURES

When a trauma patient is initially admitted to the ICU, a standard and meticulous assessment must be completed by the ICU team to assess all injuries and current physiologic state, ensure that all invasive lines and catheters are in appropriate position, and institute appropriate lifesaving resuscitation and organ-supportive treatment strategies. Standardized trauma/surgical ICU admission orders are required to ensure optimal care (Table 57-2).

The first priority during ICU admission is to assess for ongoing hemorrhagic shock and need for damage control

TABLE 57-2: Trauma/Surgical ICU Admission Orders

Obtain CXR, assess ETT position, thoracic/lung injuries
Obtain abdominal x-ray if NGT present
Peripheral venous lines—assess/document
Central venous lines—assess/document or insert if indicated
Arterial lines—assess/document or insert if indicated
ICU admission, ICU labs, including ABG; evaluate baseline lactate if ongoing resuscitation
If ongoing/past hemorrhage, obtain ROTEM/TEG to assess trauma-induced coagulopathy
Massive transfusion protocol if ongoing hemorrhage
Hypothermia reversal protocol if hypothermia present
Mechanical ventilation comprehensive orders
Sedation if intubated—assess, determine optimal medications (propofol, fentanyl)
Vital signs—frequency; continuous ECG, pulse oximetry, and temperature monitoring
Arterial line and central venous pressure monitoring
Neurologic checks and vascular checks—frequency
NGT/OGT to low continuous suction
Chest tubes to –20 cm H_2O continuous suction
Foley catheter to dependent drainage
Intake/output every 1 hour
Spine precautions, if necessary
HOB elevated >30° for VAP prevention
VTE prophylaxis: pneumatic compression devices and pharmacologic prophylaxis
Stress ulcer prophylaxis if risk factors (intubated/mechanical ventilation, coagulopathy)
Supplemental oxygen therapy
Intracranial pressure (ICP) monitoring if TBI and ICP monitor in place
Glucose management protocol, serial blood glucose monitoring
Anemia management protocol if hemorrhage control achieved
Intravenous maintenance fluids; ICU electrolyte protocol if normal creatinine
Activity: early mobility versus bedrest or other activity restrictions
Daily weights
Consult physical therapy, occupational therapy, social services, geriatrics
Order additional radiologic studies as required based on injuries sustained

ABG, arterial blood gas; CXR, chest x-ray; ECG, electrocardiogram; ETT, endotracheal tube; HOB, head of bed; ICU, intensive care unit; NGT, nasogastric tube; OGT, orogastric tube; ROTEM, rotational thromboelastometry; TBI, traumatic brain injury; TEG, thromboelastography; VAP, ventilator-associated pneumonia; VTE, venous thromboembolism.

TABLE 57-3: National and International Damage Control Resuscitation (DCR) Guidelines

Guideline	Recommendations
Eastern Association for the Surgery of Trauma Damage Control Resuscitation Guideline	• We recommend the use of a MT/DCR protocol in hospitals that manage such patients and recommend that the protocol target a high ratio of plasma and platelets to RBCs. This is best achieved by transfusing equal amounts of RBCs, plasma, and platelets during the early, empiric phase of resuscitation. • We cannot recommend for or against the use of recombinant factor VIIa based on the available evidence. • We conditionally recommend the in-hospital use of TXA early in the management of severely injured bleeding patients.
Joint Trauma System Clinical Practice Guidelines on Damage Control Resuscitation	Hemostatic resuscitation with blood products for the early treatment of hemorrhage in trauma. DCR is limited to keep blood pressure at approximately 90 mm Hg, preventing renewed bleeding from recently clotted vessels. Plasma is used as a primary resuscitation fluid in at least a 1:1 or 1:2 ratio with PRBCs. Ongoing hemorrhage is treated with use of MT protocol for blood product resuscitation. Crystalloid use is minimized.
ACS TQIP Massive Transfusion in Trauma Guidelines	Recommend a foundation ratio in acute trauma resuscitation: • Transfuse in a ratio between 1:1 and 1:2 plasma to RBCs.
Major Trauma: Assessment and Initial Management Guideline	• Transfuse 1 single donor apheresis unit or random donor platelet pool for each 6 units of RBCs. • For patients with active bleeding, use a restrictive approach to volume resuscitation until definitive early control of bleeding has been achieved.
National Clinical Guideline Centre (UK), National Institute for Health and Care Excellence (UK): Clinical Guidelines, February 2016	• For adults, use a ratio of 1 unit of plasma to 1 unit of RBCs to replace fluid volume. • In hospital settings, do not use crystalloids for patients with active bleeding • For patients with active bleeding, start with a fixed-ratio protocol for blood components and change to a protocol guided by laboratory coagulation results at the earliest opportunity. • Use intravenous TXA as soon as possible in patients with major trauma and active or suspected active bleeding. Do not use intravenous TXA more than 3 hours after injury in patients with major trauma unless there is evidence of hyperfibrinolysis. • Hospital trusts should have specific major hemorrhage protocols.

ACS, American College of Surgeons; MT, massive transfusion; PRBCs, packed red blood cells; RBC, red blood cells; TQIP, Trauma Quality Improvement Program; TXA, tranexamic acid; UK, United Kingdom.

resuscitation because hemorrhage is the most common cause of early mortality in trauma. As the initial assessment is completed, if intravenous access is inadequate, the ICU team will place additional central venous access for blood product resuscitation and arterial line for hemodynamic monitoring. The initial arterial blood gas on ICU admission provides significant information regarding degree of shock (arterial lactate, base deficit). Baseline viscoelastic testing (rotational thromboelastometry or thromboelastography) on ICU admission also provides information regarding the presence of trauma-induced coagulopathy and possible need for specific blood products or factors to assist with early definitive hemorrhage control. The ICU team must be able to provide minute-to-minute resuscitation as provided in the emergency department and in the operating room.

DAMAGE CONTROL RESUSCITATION AND MASSIVE TRANSFUSION GUIDELINES

For patients with hemorrhagic shock, a liberal transfusion strategy with hemostatic damage control resuscitation (DCR) is required until hemorrhage control is achieved. Trauma-related deaths due to hemorrhage occur early, with median times to hemorrhagic death of 2 to 2.6 hours in four prospective resuscitation trauma studies.[21] Based on these data and outcome studies comparing transfusion ratios, multiple national and international guidelines for DCR have been developed (Table 57-3).

Most recently, it was confirmed that the prehospital administration of thawed plasma resulted in a lower 30-day mortality compared to standard care resuscitation (23.2% vs 33.0%; difference, –9.8 percentage points; 95% CI, –18.6% to –1.0%; $P = .03$) in injured patients at risk for hemorrhagic shock during air medical transport. Kaplan-Meier curves showed an early separation of the two treatment groups that began 3 hours after randomization and persisted until 30 days after randomization (log-rank χ^2 test, 5.70; $P = .02$).[22]

The Joint Trauma System Clinical Practice Guidelines on Damage Control Resuscitation were the first DCR guidelines developed and implemented in combat casualty care in 2004 and recommended hemostatic resuscitation with blood products for the early treatment of hemorrhage in trauma.[23] DCR is limited to keep blood pressure at approximately 90 mm Hg, preventing renewed bleeding from recently clotted vessels. Plasma is used as a primary resuscitation fluid in at least a 1:1 or 1:2 ratio with packed red blood cells (RBCs). Ongoing hemorrhage is treated with use of a massive transfusion (MT) protocol (Fig. 57-2) for blood product resuscitation. Crystalloid use is minimized.

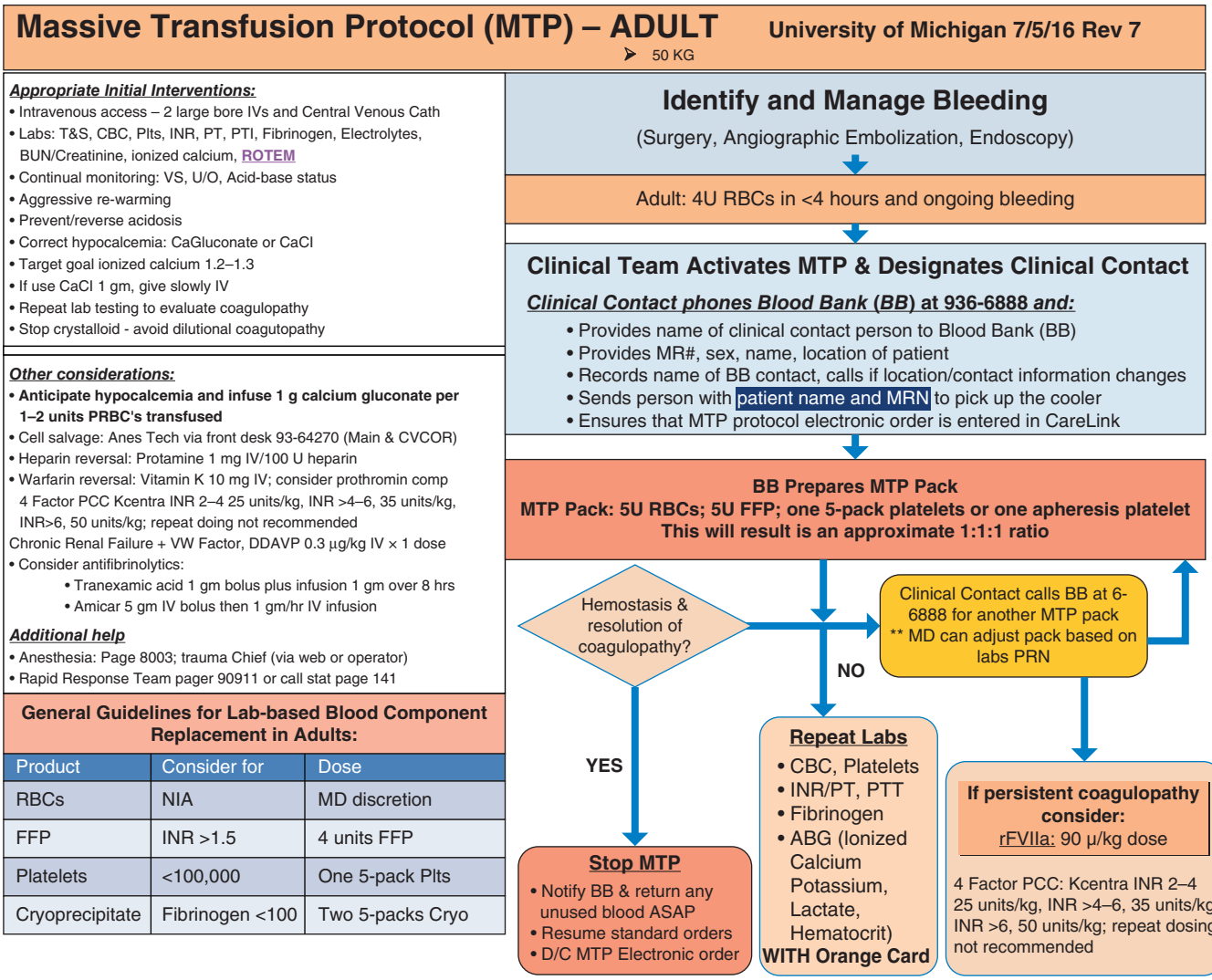


FIGURE 57-2 University of Michigan massive transfusion and damage control resuscitation (DCR) protocol. (Reproduced from the University of Michigan.)

The recent Eastern Association for the Surgery of Trauma Damage Control Resuscitation Guidelines recommend the use of an MT/DCR protocol to target a high ratio of plasma and platelets to RBCs, best achieved by transfusing equal amounts of RBCs, plasma, and platelets during the early empiric phase of resuscitation.[24] This guideline included 31 studies that met criteria for quantitative meta-analysis. This analysis confirmed that mortality decreased with use of an MT/DCR protocol versus no protocol (OR, 0.61; 95% CI, 0.43–0.87; $P = .006$) and with a high ratio of plasma to RBCs and platelets to RBCs (relatively more plasma and platelets) versus a low ratio (OR, 0.60; 95% CI, 0.46–0.77; $P < .0001$; OR, 0.44; 95% CI, 0.28–0.71; $P = .0003$). They concluded that DCR can significantly improve outcomes in severely injured bleeding patients.

The ACS TQIP Massive Transfusion in Trauma Guidelines also recommend a foundation ratio in acute trauma resuscitation: "transfuse in a ratio between 1:1 and 1:2 plasma:RBC; transfuse one single donor apheresis unit or random donor platelet pool for each 6 units RBCs."[25]

The National Institute for Health and Care Excellence Guideline on Major Trauma: Assessment and Initial Management for the United Kingdom provides guidance on the assessment and management of major trauma, including resuscitation following major blood loss associated with trauma. It includes an important section on management of hemorrhage and management of shock, and the guidelines are similar to the ACS TQIP Guidelines.

Optimal dose, timing, and ratio of blood products in MT are still controversial with a recent systematic review (including 16 randomized controlled trials) concluding that "there is insufficient basis to recommend a 1:1:1 over a 1:1:2 ratio or standard care for adult patients with critical bleeding requiring massive transfusion," and additional high-quality studies are warranted.[26]

PHYSIOLOGIC MONITORING IN THE INTENSIVE CARE UNIT

Intensive physiologic monitoring can be provided in the ICU to provide optimal critical care. Monitoring devices can be used to determine if the patient has stable organ function and to detect anomalies that may indicate impending organ dysfunction. In recent years, there has been a dramatic increase in the ability to monitor a wider variety of physiologic parameters. Monitors can be useful in determining if a patient's trajectory is deviating from the anticipated or optimal course; however, all data obtained from physiologic monitors require interpretation and integration with the overall clinical picture by the critical care team. Monitoring itself can be associated with serious complications, including device-related complications and errors in interpretation that lead to errors in clinical decision making and treatment. The questions that must be addressed with any form of monitoring include "When?" (indication and timing), "What?" or "How much?" (which specific monitoring tools or techniques), and "Why?" (an analysis of the associated risks and benefits).

Hemodynamic Monitoring (See Chapter 58)

Hemodynamic monitoring is directed at assessing the results of resuscitation and used as a guide to reestablishing and maintaining tissue and organ perfusion. The restoration of normal arterial blood pressure, central venous pressure (CVP), pulmonary arterial (PA) pressure, and cardiac output (CO) provides some reassurance that there is adequate organ perfusion. This reduces the likelihood of death and serious morbidity due to complications of hemorrhagic shock including postinjury inflammatory response syndrome and multiple organ failure. However, the phenomenon of regional circulation and inadequate perfusion of specific organs, particularly the gut, in the presence of normal arterial pressures remains a concern. New types of monitors are becoming available that can monitor CO and intravascular volume status in a relatively noninvasive fashion and may be able to determine perfusion at the tissue level.[27]

ARTERIAL PRESSURE MONITORING

Noninvasive blood pressure monitoring is widely available using automated blood pressure cuffs. These devices can be set to perform repetitive blood pressure measurements as frequently as every minute. However, they are usually considered insufficient in patients with significant hypotension due to poor sensitivity at low blood pressures as well as the intermittent nature of the readings. Insertion of an arterial intraluminal catheter or arterial line allows instantaneous measurement and continuous display of arterial blood pressure, even at very low blood pressures.

Arterial lines have a wide variety of indications (Table 57-4) and are the preferred method of arterial blood pressure management in patients receiving continuous infusions of vasoactive drugs as they allow better drug titration. Arterial lines are typically placed in the radial artery, although they can also be placed in the dorsalis pedis, femoral, or axillary arteries. Infection of arterial lines is less common than that of central venous lines; however, sterile technique should still be used to avoid the risk of catheter-related bloodstream infections. Thrombotic complications can also occur at any arterial line site and may lead to tissue ischemia, including loss of digits. This is particularly common in patients with prolonged hypotension or sepsis or who have been treated with vasopressors. Although Allen tests are frequently performed before the insertion of a radial arterial line, these do not exclude the possibility of this complication.

Minimally invasive continuous arterial pulse waveform analysis devices are available that quantify pulse pressure variability (PPV), and stroke volume variation (SVV) by analyzing the variations in arterial waveform with respiration and the area under the arterial pressure waveform. These devices can also use the calculated stroke volume to provide an

TABLE 57-4: Indications (Suggested) for Hemodynamic Monitoring

Arterial line monitoring
Shock states (any)
Cardiac
Septic
Neurogenic
Hypovolemia/hemorrhage
Monitoring intraoperatively/postoperatively
Head injury
Serial monitoring of ABG
Labile hemodynamic status
Respiratory failure
Hypertensive crisis
Vasoactive drug infusions
High-risk or elderly trauma patient
Central venous pressure monitoring
Volume status in acute head injury
Complex trauma/pelvic fracture
Shock dates
Acute renal failure
Vasoactive drug infusions
Monitoring during acute ICU hemodialysis
Cardiac pacing
Elderly trauma patient
Massive transfusion
Pulmonary contusions
PA catheter monitoring
Refractory septic and cardiogenic shock states
Severe hypoxemia, pulmonary edema/embolism/ARDS
High-risk intraoperative monitoring
Barbiturate coma (iatrogenic myocardial depression)
Moderate or mixed venous O_2 (Svo_2)
Diagnoses: explained shock, hypoxemia, renal failure

ABG, arterial blood gas; ARDS, acute respiratory distress syndrome; ICU, intensive care unit; PA, pulmonary artery.

estimate of CO without need of a PA catheter. Recent meta-analyses found that PPV and SVV of at least 11% to 13% both had excellent ability to identify critically ill patients who were likely to respond to fluid challenge with an area under the curve (AUC) of 0.94 and 0.84, respectively.[28,29] However, it should be kept in mind that PPV and SVV are most reliable in patients on mechanical ventilation with no spontaneous respirations and may be less reliable in patients with arrhythmias, patients with spontaneous respirations, and those on low tidal volume ventilation.

CENTRAL VENOUS PRESSURE MONITORING

Monitoring CVP is most useful in patients at high risk of over- or underresuscitation. These include patients with limited cardiac reserve, including the elderly and those with cardiac disease. Patients with TBI are another group in whom CVP monitoring is frequently performed to avoid hypotension associated with underresuscitation or overresuscitation resulting in increased cerebral edema. Although guidelines suggest CVP goals between 8 and 12 mm Hg,[30] these end points may be less reliable in patients on positive-pressure ventilation, making trends of CVP over time in response to therapy, rather than static measurements, more useful in guiding overall resuscitation.

The central venous catheter can also be used to obtain a mixed venous blood gas measurement. Since the mixed venous oxygen saturation ($Scvo_2$) is usually slightly lower in the superior vena cava as compared with that in the inferior vena cava due to the relatively low oxygen consumption (Vo_2) of the kidneys, the blood oxygen saturation obtained from a central venous catheter is usually slightly higher than that obtained from a PA catheter. Central venous catheters that have an oximetric tip can measure the saturation of blood at the tip of the central venous catheter or $Scvo_2$, allowing continuous measurement of this marker of global perfusion.

ECHOCARDIOGRAPHY

The formal transthoracic echocardiogram (TTE) is extremely valuable in assessing the function and structure of the heart and its components including valves and chambers. Limited TTE can also detect such conditions as pneumothorax and pleural effusions.[31] However, in about 50% of trauma patients, it is difficult or impossible to perform a complete TTE due to chest trauma, subcutaneous air, obesity, or surgical dressings. Transesophageal echocardiography (TEE) can overcome many of these limitations and can be used in the detection of such injuries as blunt aortic rupture and blunt myocardial injury.[12,17-19] Frequent use of TEE in the ICU can be limited, however, due to availability, cleaning requirements, and the skill required by the operator. However, there are now US Food and Drug Administration (FDA)–approved TEE devices using a disposable probe that can be left in the esophagus or stomach for up to 72 hours, allowing for repeated exams over time. Studies of this catheter demonstrate excellent image quality and utility in directing therapeutic decision making.[32]

Focused critical care echocardiography (CCE) is now essential in provision of optimal ICU care, and the ability to perform a prompt bedside TTE on ICU arrival for all trauma patients is extremely helpful in determining their cardiac function on ICU admission, particularly in geriatric trauma patients.[33] CCE provides unique morphologic and functional information in real time to determine the specific etiology of shock and/or cardiac dysfunction in ICU patients, as in Fig. 57-3. CCE identified that one patient had shock and cardiac dysfunction due to hypovolemia and responded to fluid resuscitation, whereas the other patient had shock and cardiac dysfunction due to acute cor pulmonale and required administration of inhaled nitric oxide and diuresis to improve cardiac performance.

CCE is performed and interpreted by the intensivist at the bedside to help in the diagnostic workup and to guide therapy for patients with acute circulatory and respiratory failure. CCE includes expertise in both TTE and assessment of preload and volume status by assessment of inferior vena cava diameter and compressibility. Due to the statement of educational standards, the validation of echocardiography against alternative hemodynamic monitoring tools, and continuous technologic improvement, CCE is increasingly being considered as an unparalleled technique to hemodynamically assess critically ill patients since it can be performed bedside and in a serial manner to assess response to therapies instituted.

Basic CCE should be part of the initial training of all intensivists, whereas advanced CCE is an optional component of competency since it requires a more extensive and specific training. The National Board of Echocardiography announced that the first administration date for the new Examination of Special Competence in Critical Care Echocardiography was Tuesday, January 15, 2019 (https://www.echoboards.org/EchoBoards/News/2019_Adult_Critical_Care_Echocardiography_Exam.aspx).

PULMONARY ARTERY CATHETERS

The Swan-Ganz PA catheter was first introduced in 1970; however, in the mid-1990s, many studies questioned their efficacy and reported complications including pulmonary infarction and pulmonary artery thrombosis, perforation, bleeding, and pseudoaneurysm. More recent studies have not demonstrated increased major adverse sequelae associated with the PA catheter.[35] However, these studies also failed to demonstrate any significant improvement in outcomes associated with the use of PA catheters. Currently, PA catheters are primarily used in patients who have known or suspected myocardial disease or dysfunction.

An alternative to invasive PA catheter placement is the FloTrac/Vigileo system (Edwards Lifesciences, Irvine, CA), which uses arterial waveform analysis to calculate SVV, stroke volume, and CO. Meta-analyses confirm that this technology has better concordance with PA catheter and echocardiographic estimates of CO, with a percentage of error less than or equal to 30%. However, optimal concordance only occurs in patients with normal ejection fraction (≥40%) and systemic vascular resistance (SVR). Other minimally invasive devices use

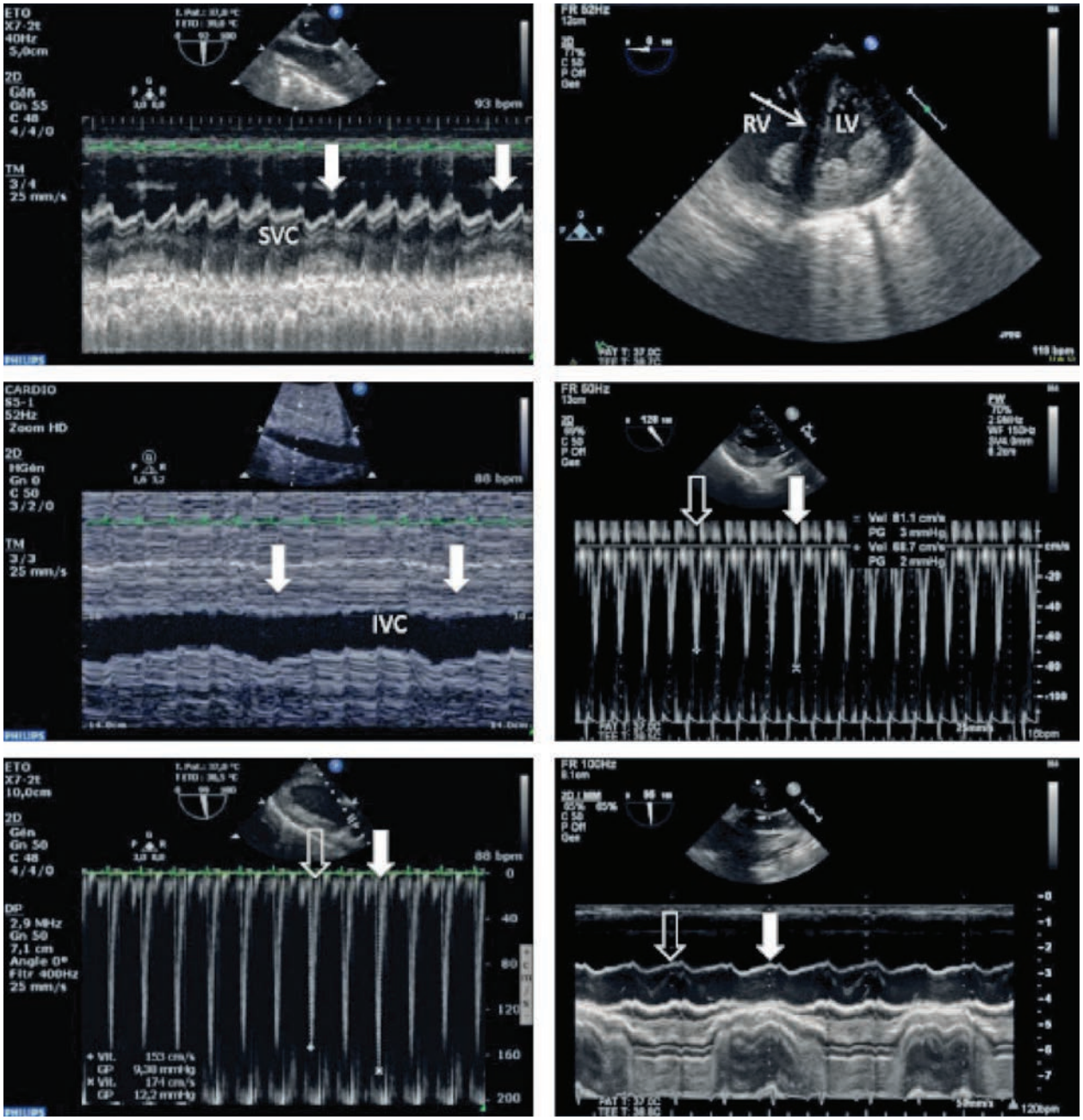


FIGURE 57-3 Focused critical care echocardiography (CCE) by transthoracic or transesophageal approach. IVC, inferior vena cava; LV, left ventricle; RV, right ventricle; SVC, superior vena cava. (Reproduced with permission from Vignon P. What is new in critical care echocardiography? *Crit Care.* 2018;22:40.)

arterial waveform analysis in conjunction with lithium dilution (LiDCO, Cambridge, United Kingdom) and thermodilution (PiCCO, PULSION Medical System, Munich, Germany) to estimate SVV and CO. However, these devices require placement of a central venous catheter and calibration every 8 hours and are also less accurate among patients with arrhythmias, valvular disease, depressed cardiac function, and high or low SVR.[35]

BIOIMPEDANCE CARDIOGRAPHY

Transthoracic impedance, also known as bioimpedance cardiography, uses an electrical current applied to the thorax. Changes in bioimpedance to this current are used to measure heart rate, left ventricular ejection fraction, and intrathoracic fluid content and calculate stroke volume, CO, and SVR. However, the technology is limited by patient movement and arrhythmias and is inaccurate in pregnancy, obesity, pleural fluid or gas, heart failure, severe valvular disease, and pulmonary edema. A new device is available that uses impedance electrodes on the outside surface of an endotracheal tube, the Endotracheal Cardiac Output Monitor (ECOM; ConMed, Irvine, CA). This allows impedance measurements without interference from abnormalities or motion of the chest wall.

However, studies have demonstrated an inability to accurately estimate or trend CO when compared to thermodilution.[36]

A number of other hemodynamic monitoring methods are available for use in the ICU (Table 57-5), and each ICU must review the optimal technology for use in their individual ICU based on the patient population, knowledge and experience of the staff, and willingness to increase use of central venous lines, which might increase risk for central line–associated bloodstream infection.

Neurologic Monitoring (See Chapter 22)

One of the principal indications for admission to the ICU for trauma patients is the need for frequent neurologic

TABLE 57-5: Overview of Hemodynamic Monitoring Methods in the ICU

Method	Examples of commercial name	Calibrated or not	Major advantages	Major disadvantages
Invasive methods				
Pulmonary artery catheter		Calibrated	Direct measurements in right atrium and pulmonary circulation	Delay in determining CO, most invasive, and risks involved
Less-invasive methods				
Transpulmonary thermodilution	PiCCO® VolumeView®/ EV1000® LiDCO®	Calibrated	Intermittent and continuous CO, added variables	Need for specialized arterial and central venous line, LIMITS (PiCCO® system)
Ultrasound flow dilution	COstatus®	Calibrated	Continuous CO, added variables, can detect intracardiac shunts	Requires AV loop
Pulse contour and pulse pressure variation	FloTrac®/Vigileo® ProAQT®/Pulsionflex® LiDCOrapid®/pulseCO® Most Care®/PRAM	Non-calibrated	Continuous CO	Lack accuracy in unstable patients or during use of vasoactive drugs
Partial CO_2-rebreathing	NiCO®	Non-calibrated	No need for intravascular devices	Only in sedated patients under volume control ventilation, interference from pulmonary disease
Transesophageal echocardiography		Operator dependent	Real-time images of the cardiac structures and blood flow	Learning curve, (low) risk of complications
Esophageal Doppler		Operator dependent	Real-time CO and afterload data, added variables	Risk of dislocation
Non-invasive methods				
Transthoracic echocardiography		Operator dependent	Direct measurement of CO and visualization of cardiac structures	Ultrasound characteristics often suboptimal in ICU patients
Non-invasive pulse contour systems	T-line® ClearSight®/Nexfin®/ Physiocal® CNAP®/VERIFY®	Non-calibrated	Non-invasive, simple tool	Less accurate, needs more validation
Bioimpedance		Non-calibrated	Simple tool, providing data concerning CO and fluid overload	Changes intrathoracic fluid content and SVR influence measurements
Estimated continuous cardiac output®	esCCO®	Non-calibrated	Uses widely available variable to estimate CO	Is only estimate, inadequate accuracy
Ultrasonic cardiac output monitoring®	USCOM®	Non-calibrated	Short learning curve and only few risks	Only estimate, uses standard valve areas which can differ in patients

AV loop, arteriovenous fistula; CO, cardiac output; ICU, intensive care unit; SVR, system vascular resistance.

Source: Reproduced from Huygh J, Peeters Y, Bernards J, Malbrain ML. Hemodynamic monitoring in the critically ill: an overview of current cardiac output monitoring methods. *F1000Res.* 2016;5:pii: F1000 Faculty Rev-2855.

assessments in those with known or suspected TBI. There are two goals for monitoring in TBI: first, the avoidance of secondary brain injury by avoidance of hypoxia and hypotension and maintenance of cerebral perfusion; and second, the detection of increased ICP due to cerebral edema or expanding intracranial hematomas. Reassessment should be done frequently as subtle changes may herald increases in ICP or cerebral ischemia. Severe TBI is a common cause of trauma mortality.

ICP MONITORING

ICP monitoring with a ventriculostomy catheter or subdural bolt is a common practice in most major centers for patients who have a neurologic exam that is unavailable or unreliable following TBI. The Brain Trauma Foundation (BTF) guidelines[37] recommend early and aggressive monitoring of ICP and the calculated cerebral perfusion pressure (CPP), which is the difference between the mean arterial pressure and ICP. Monitoring of the ICP and CPP can be used to guide therapy such as vasopressors, sedation, paralysis, hyperosmolar therapy, or cerebrospinal fluid drainage and to provide surveillance for increasing cerebral edema or intracranial hemorrhage (Fig. 57-4). Adherence to the BTF guidelines, including compliance with placement of ICP monitors, has been associated with improved mortality in patients with TBI.[38-40]

STAGE 1

Admission to the ICU
Ventilation PaO_2 ≥11 kPa, $PaCO_2$ 4.5–5.0 kPa
Sedation
Analgesia ± paralysis
30° head of bed elevation
Intravenous fluids ± inotropes to maintain MAP ≥80 mm Hg

Ventriculostomy ± CSF drainage
Surgical removal of SOL
± Prophylactic anticonvulsants

STAGE 2

Mannitol (maintain serum osmolarity <315 mOsm)

Hypertonic saline (avoid in hyponatremic patients, caution in patients with cardiac or pulmonary problems)

Inotropes to maintain CPP >60 mm Hg

Monitor blood magnesium levels and replace as required

Barbiturates not permitted
± Therapeutic hypothermia

STAGE 3

Barbiturate therapy

Decompressive craniectomy

FIGURE 57-4 Management of severe traumatic brain injury by Brain Trauma Foundation Guidelines. CPP, cerebral perfusion pressure; CSF, cerebrospinal fluid; ICU, intensive care unit; MAP, mean arterial pressure; SOL, space occupying lesion.

Investigational noninvasive monitoring systems for ICP include transocular ultrasound monitoring of optic nerve sheath diameter.[41] The optic nerve sheath is an extension of the intracranial dura matter and has been shown to become distended with elevated ICP. Continuous transcranial Doppler ultrasound, brain tissue oxygen, and cerebral microdialysis monitoring are other investigational approaches for ICU monitoring of TBI that may contribute to decision making and eventually improve outcomes in these patients.[42]

RESPIRATORY FAILURE AND MECHANICAL VENTILATION (SEE CHAPTER 59)

General Principles

The need for mechanical ventilation is the most common indication for admission to the ICU. The general principles for intubation and mechanical ventilation are the following:

1. Secure and establish an airway.
2. Decrease the work of breathing (WOB).
3. Improve oxygenation.
4. Improve ventilation (carbon dioxide [CO_2] gas exchange) and maintain control of partial pressure of arterial CO_2 ($Paco_2$), especially in TBI.
5. Anticipate worsening respiratory status or airway patency such as the need for large-volume resuscitation, severe neck/thoracic trauma, upper torso/facial burns, and inhalation injury.

Current ventilator technology is diverse and advanced; however, the essential physical principle of mechanical ventilation is pushing oxygen-rich air into the lungs by positive pressure and removal of waste CO_2 by reduced pressure. Ventilator management can change rapidly; therefore, a nucleus of critical care expertise is needed not only to optimize respiratory support, but also to interpret acute changes in pulmonary mechanics that can often be a harbinger of systemic pathology.

MONITORING OF GAS EXCHANGE

Rapid assessment of arterial oxygen tension (Pao_2) is essential for both evaluating and managing the adequacy of alveolar–arterial oxygen gas exchange. Oxygen (O_2) is driven from the alveolar airspace into the pulmonary capillaries along a diffusion gradient between the respective tissue beds. Calculating the efficiency of pulmonary oxygen exchange can be cumbersome since the equation for the A–a gradient requires alveolar and arterial CO_2 concentrations, shunt fraction, water vapor pressure, and body temperature. A more convenient and simple bedside index of oxygen exchange is the partial pressure of oxygen (Pao_2)/fraction of inspired oxygen (Fio_2) (P/F) ratio that adjusts for a fluctuating Fio_2 and helps in defining lung injury. The use of pulse oximetry to determine arterial oxygen saturation (Sao_2) has replaced continuous arterial blood gas measurements as a real-time, noninvasive method to assess

arterial oxygenation. There are limitations to Sao_2, and its nuances are important in interpreting the significance of an absolute Sao_2 percentage. First, because of the kinetics of oxygen–hemoglobin binding, Sao_2 and the oxygen dissociation curve are sigmoidal and not linear. Therefore, small changes in Sao_2 may reflect a much larger drop in Pao_2. Second, under instances of carbon monoxide poisoning or severe circulatory shock, oxygen delivery will be abnormally low despite a near-normal Sao_2.

In addition to oxygenation-adequate ventilation, end-tidal (Et) CO_2, and resultant arterial CO_2 tension (pco_2) have important physiologic and clinical implications and must be monitored in mechanically ventilated patients. The immediate detection of CO_2 through qualitative capnography relies on the lower pH of $EtCO_2$-rich air changing the color of pH-sensitive filter paper in the capnograph. As a rule, a disposable capnograph remains purple if $EtCO_2$ is less than 0.5% and turns yellow when $EtCO_2$ is greater than 2.0%.[43] Normal $EtCO_2$ is greater than 4%; therefore, capnography turns yellow when the endotracheal tube is positioned in an airway. This device is very sensitive unless the patient is in circulatory arrest and adequate pulmonary perfusion is compromised. The presence of a large volume of acidic gastric contents may give a false impression of successful endotracheal intubation in some cases of esophageal intubation. In these circumstances, initial detection of $EtCO_2$ decreases rapidly with subsequent tidal volumes. Continuous quantitative capnography analyzes $EtCO_2$ and, depending on the alveolar–arterial gradient, can give an indication of the arterial pco_2. In patients without preexisting pulmonary disease, the normal alveolar–arterial gradient is between 1 and 3 mm Hg. Using quantitative capnography can be helpful in brain injury where hyperventilation may curtail rising ICP. However, $EtCO_2$ measurements are affected by pulmonary dead space fraction and pulmonary perfusion that can be greatly altered in cases of hemorrhagic shock, thoracic trauma, and increased airway resistance. In a prospective study of 180 recently intubated trauma patients, $EtCO_2$ measurements were correlated with pCO_2 from blood gas analysis.[44] Using regression analysis, there was a direct correlation between $EtCO_2$ and pco_2; however, patients ventilated with an $EtCO_2$ of 35 to 40 mm Hg were likely to have a pCO_2 greater than 40 mm Hg 80% of the time and a pco_2 greater than 50 mm Hg 30% of the time. In severely injured patients, when an increase in pulmonary shunt may exist, the measurement of pCO_2 through blood gas analysis remains the most accurate measure of assessing adequacy of ventilation.

Causes of hypoventilation can be multifactorial. Increased dead space from pulmonary contusion, acute respiratory distress syndrome (ARDS), pulmonary embolism, and oversedation can be contributing factors causing hypercapnia, especially during the weaning phase of mechanical ventilation.

ASSESSMENT OF LUNG MECHANICS AND INSPIRATORY/EXPIRATORY PRESSURE

Assessing pulmonary mechanics can significantly aid in diagnosing and treating a patient's sudden or progressive pulmonary insufficiency. One of the most important parameters to measure is compliance and the ability to distinguish between static and dynamic compliance. Compliance, the change in volume produced by a change in pressure, is calculated based on measurements taken from the ventilatory circuit itself. In general, a sudden decrease in compliance will mean a concomitant rise in both plateau pressure (PP) and peak inspiratory pressures (PIP) for a given tidal volume (V_T) as seen in pneumothorax or abdominal compartment syndrome. However, measuring the individual changes of either static or dynamic compliance may indicate a specific pulmonary pathology.

PULMONARY COMPLIANCE

In static compliance, the change of volume produced is measured from the inspiratory hold pressures or plateau pressure using the following formula: $Compliance_{static} = V_T/PP - PEEP$ (positive end-expiratory pressure). Static compliance reflects the alveoli and chest wall, rather than the airways. Cases of decreased static compliance (normal = 50–100 mL/cm H_2O) with normal or elevated PIP generally indicate intra-alveolar pathology such as ARDS, ALI, or pulmonary edema.

Dynamic compliance uses the PIP ($Compliance_{dynamic} = V_T / PIP - PEEP$) to estimate resistance of the airways. Sudden decreases in dynamic compliance or a measured difference between static and dynamic compliance reflects increased airway resistance during bronchospasm, mucous plugging, kinked endotracheal tube, or foreign body aspiration.

PRESSURE–VOLUME CURVES

A well-accepted principle in mechanical ventilation is the importance of increasing alveolar recruitment to improve oxygenation. Assessment of the pressure–volume curve in mechanical ventilation has been used to determine the mean pressure needed for the inflection point, or P_{flex} (Fig. 57-5). This point represents the critical pressure needed to open collapsed alveoli and corresponds to the zone of optimal alveolar recruitment. The pressure reading at the inflection point may reflect an optimal PEEP setting. In ARDS and pulmonary edema, the normal pressure–volume curve is shifted, leading to de-recruitment of alveolar units and increasing pulmonary

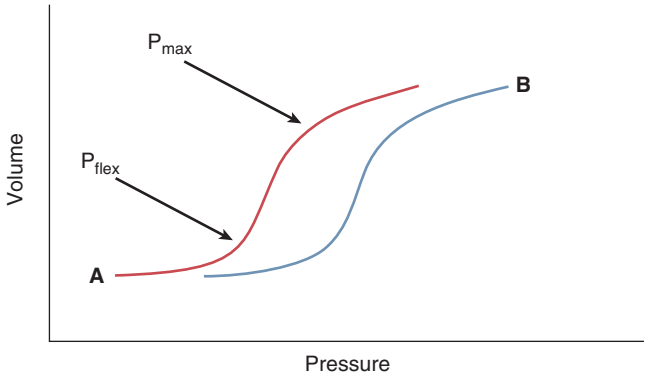


FIGURE 57-5 Pressure–volume curves.

shunt. During these instances, increasing driving pressure and PEEP are often used to maximize gas exchange.

VENTILATORY CAPACITY

Ventilator capacity encompasses the ability of a patient to generate muscular forces needed to meet the ventilatory work required for physiologic demand. Assessing ventilatory capacity in the mechanically ventilated patient can be an important physiologic indicator of clinical improvement and can be used to predict successful extubation, particularly in patients who have been intubated for prolonged periods of time or have neurologic (spinal cord injury and myasthenia gravis) or mechanical (flail chest and rib fractures) injury affecting the thorax. Forced vital capacity and negative inspiratory force (NIF) are the most frequently measured indices of ventilator capacity. A normal vital capacity is between 65 and 75 cm^3/kg depending on ideal body weight and sex. The NIF estimates inspiratory muscle strength, and a value of –25 to –30 cm H_2O is indicative of adequate inspiratory force to maintain airflow after extubation.[45]

Mechanical Ventilation Modes

Spontaneous breathing uses negative intrathoracic pressure to fill alveoli (Fig. 57-6). In contrast, mechanical ventilation uses positive pressure to fill alveoli. Positive-pressure mechanical ventilation can improve gas exchange by recruiting atelectatic alveoli, increasing functional residual capacity, and reducing areas of ventilation/perfusion mismatch, thereby decreasing pulmonary shunt fraction. Negative effects of mechanical ventilation vary according to ventilatory mode; however, adverse effects common to all positive-pressure modes include barotrauma, ventilator-induced lung injury, and impairment of CO from decreased venous return. It is important to understand the physical mechanics with different modes of mechanical ventilation since correct management will reduce duration of mechanical ventilation and improve patient outcomes. In choosing the ideal mode of mechanical ventilation for an individual patient, it is helpful to evaluate the patient's current oxygenation, ventilation, pulmonary compliance, muscular strength, and mental status.

MANDATORY VOLUME CONTROL VENTILATION

Both continuous mandatory ventilation (CMV) and assist-control (AC) mode ventilation are preset volume-cycle modes that deliver a fixed tidal volume at a specific respiratory frequency. The individual names of these modes are commonly referenced interchangeably; however, CMV delivers a set tidal volume exclusive of a patient's ventilatory effort, whereas AC synchronizes delivery of a set tidal volume to each patient-initiated breath. Consequently, an awake and breathing patient will have episodes of dysynchrony causing significant discomfort, breath stacking, or barotrauma. Because of this, CMV is rarely used. In AC, the ventilator recognizes a patient-triggered breath (either flow or pressure mediated) and delivers a synchronous, identical preset tidal volume (Fig. 57-7). Most modern ventilators are "flow triggered"; a continuous flow of gas passes around the breathing system, and patient inspiration deflects this flow and triggers the ventilator. AC modes also have a set rate to assure ventilation in an apneic, paralyzed, or heavily sedated patient; in these circumstances, AC becomes indistinguishable from CMV. In an awake or lightly sedated patient, full synchronous respiratory support will be achieved with each initiated breath, thereby decreasing WOB and assuring adequate ventilation. Awake or agitated patients may trigger several full tidal volume breaths, causing high minute ventilation and respiratory alkalosis.

SYNCHRONIZED VENTILATION MODES

Synchronous intermittent mandatory ventilation (SIMV) is similar to AC but allows for spontaneous ventilation in addition to the preset breaths (Fig. 57-8). In contrast to AC ventilation, where every patient-initiated breath receives the full tidal volume, SIMV mode allows for respiratory work between the preset tidal volumes. The degree of ventilatory support can be increased or decreased by adjusting the set rate and amount of pressure support delivered by the ventilator with patient breaths. SIMV can be useful in reducing minute ventilation in patients who are significantly overbreathing during AC ventilation causing respiratory alkalosis.

Excessive airway pressures (>30 cm H_2O) can increase the incidence of barotrauma and lung injury, especially in states

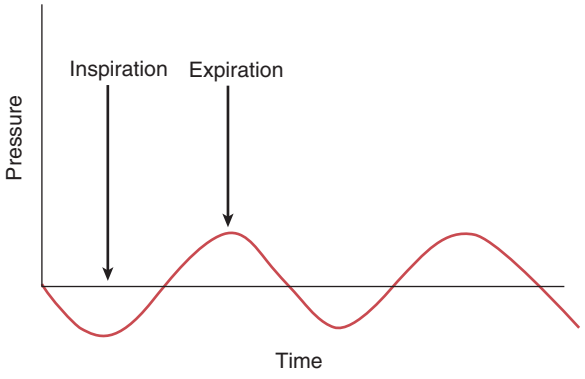


FIGURE 57-6 Spontaneous mode of breathing.

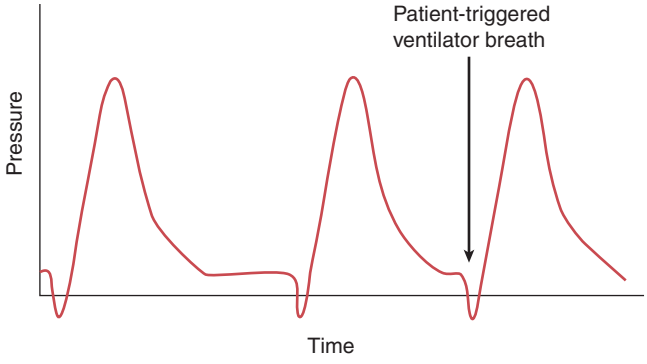


FIGURE 57-7 Assist-control (A/C) mode ventilation.

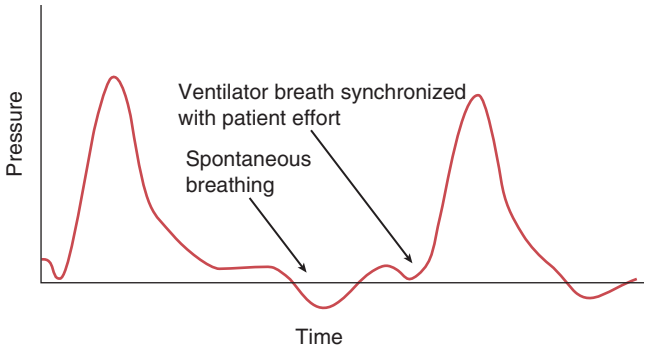


FIGURE 57-8 Synchronous intermittent mandatory ventilation (SIMV).

of decreased pulmonary compliance. Pressure-regulated volume control ventilation modulates the inspiratory flow pattern during a volume-controlled breath, thus reducing PIP. This flow pattern ("decelerating inspiratory flow") is in contrast to pure AC/CMV modes where there is constant inspiratory flow resulting in a continuous rise in airway pressure.

Pressure support ventilation (PSV) provides a baseline level of inspiratory airway pressure and decreases the WOB by augmenting spontaneous respiration (Fig. 57-9). The patient, therefore, is aided in overcoming the resistance of the ventilatory circuit and has complete control over the rate and tidal volume. Each PSV breath is supported by a specific flow limited by a preset pressure that is triggered by patient inspiration. Because this mode requires spontaneous breathing, PSV can only be used in lightly sedated or awake patients without paralytic therapy or neuromuscular disease. Although PSV can be used as a sole ventilation mode, it is most frequently used during ventilator weaning (see the later section "Weaning from Mechanical Ventilation"). Since national guidelines currently recommend light sedation of intubated patients, PSV ventilation can be used as the primary mode and has the advantage of maintaining endogenous diaphragmatic function in ICU patients, preparing them for extubation.

PRESSURE-CONTROL VENTILATION

In patients with ALI/ARDS with poor pulmonary compliance, the limitations of volume-cycled ventilation are increasingly recognized. To achieve a specific preset tidal volume, progressively higher airway pressure must be delivered as compliance worsens, increasing the likelihood of barotrauma. In contrast to volume control, time-cycled pressure control (PC) ventilation delivers breaths at a fixed flow rate dictated by a preset pressure (driving pressure). Regardless of pulmonary or chest wall elasticity, the PIP is fixed and will not exceed the set driving pressure; however, the delivered tidal volume will vary as a function of compliance. PC is useful in ARDS patients to limit plateau pressures below 30 cm H_2O and maintain low tidal volume ventilation.[46] Despite the theoretical advantages of using pressure modes of ventilation in patients with poor compliance, the literature to date has failed to identify a statistically significant benefit to mortality or barotrauma with either mode of ventilation.[47]

In conventional AC/CMV mode, the inspiratory-to-expiratory ratio is approximately 1:2, thereby minimizing airway pressures and allowing adequate ventilation. In severely hypoxic patients, the ratio can be reversed (2:1 or higher)—inverse ratio ventilation (IRV). In principle, IRV allows maximal recruitment of alveoli by increasing the inspiratory time. When using IRV, it also is important to be cognizant of rising intrinsic PEEP and mean airway pressures that can lead to barotrauma, hypercapnia, or a decrease in venous return affecting CO.

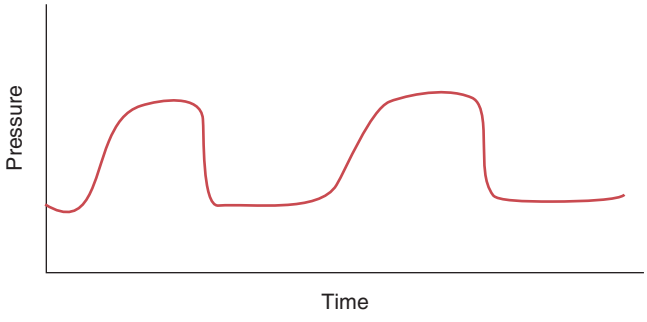


FIGURE 57-9 Pressure support (PS) ventilation.

AIRWAY PRESSURE RELEASE VENTILATION

Airway pressure release ventilation (APRV), or bilevel ventilation, is essentially continuous positive airway pressure (CPAP) ventilation with the exception that the pressure setting is generally higher (P_{high}) for a longer duration (T_{high}) than typical CPAP and there is a short "release" of high pressure (T_{low} and P_{low}), allowing for ventilation and CO_2 exhalation. The long duration of P_{high} (as long as 6 seconds) may improve alveolar recruitment, functional residual capacity, and gas exchange by increasing mean airway pressure without the expense of high PIP. APRV's greatest benefit is the presence of a floating release valve that allows for patients to breathe spontaneously during the prolonged P_{high} phase, which may provide greater patient comfort and decrease need for sedation. Studies have demonstrated safety and improved oxygenation with APRV, and some have shown a mortality benefit compared to other modes of ventilation when lung-protective strategies are used.[48] Time-controlled adaptive ventilation is a new preemptive ventilation strategy recommended to "cast" the lung maintaining homogeneous ventilation using an extended time at inspiration and a brief time at expiration.[49]

Respiratory Failure

PULMONARY CONTUSION AND HYPOXEMIA

Parenchymal disease is the most common cause of hypoxemia, including aspiration pneumonia, hospital-acquired pneumonia, pulmonary contusion, or ARDS. Pneumothorax and/or hemothorax may also manifest as hypoxemia but generally occur during the early phase of resuscitation.

Pulmonary contusion consists of a direct injury to the lung, and the contusion evolves over the first 24 hours as alveolar hemorrhage and edema accumulate, such that the P_{O_2} progressively decreases during that time period. The contused lung has leaky capillaries, and aggressive fluid resuscitation, particularly with colloids, may result in further deterioration of pulmonary function. A restrictive fluid strategy is recommended to prevent severe hypoxemia in patients with pulmonary contusion. The biggest pitfall in the management of pulmonary contusions is failure to anticipate injury progression. Computed tomography (CT) scanning of the chest can be used to estimate the amount of injured lung and potential need for mechanical ventilation.[50]

SEVERE HYPOXEMIA AND ARDS

It is very important to identify ARDS early in the ICU after injury because the associated mortality rate of severe ARDS (P/F ratio ≤100) is high (40%). The Berlin definition of ARDS (see Table 59-4) classifies ARDS into mild, moderate, and severe categories and includes specific amounts of PEEP at which the P/F ratio is calculated.

Other Options in Respiratory Failure

PRONE POSITIONING

Poorly or nonaerated lung units localize in dependent lung zones while in the supine position. Prone positioning improves gas exchange and ventilation/perfusion mismatch by expanding atelectatic portions of the lung akin to the zones of West. Prone positioning also prevents ventilator-induced lung injury by prevention of alveolar hyperinflation, more homogeneous distribution of transpulmonary pressure and ventilation, and prevention of atelectotrauma through improved alveolar recruitment compared to supine positioning. The most recent meta-analysis confirmed a significant survival benefit and improved oxygenation with prone positioning in ARDS patients with severe hypoxia.[51] However, prone positioning may be associated with complications, including pressure ulcer and endotracheal tube dislodgment.

The PROSEVA (Proning in Severe ARDS Patients) trial[52] confirmed a significant survival benefit with the prone position protocol (prone position for 16 hours per day) compared to patients who remained supine. Patients in the prone group underwent their first prone positioning session within 55 ± 55 minutes after randomization. The average number of sessions was 4 ± 4 per patient. Twenty-eight-day mortality (16.0% vs 32.8%; P <.001) and 90-day mortality (23.6% vs 41.0%; P <.001) were both significantly decreased in the prone group. Due to the effectiveness and simplicity of placing patients in a prone position, the early use of prone positioning should be considered in all patients with moderate to severe ARDS.

PHARMACOLOGIC AGENTS

Two agents used to improve hypoxia in severe ARDS are inhaled nitric oxide and inhaled epoprostenol. The mechanism of action is likely related to the relaxation of vascular smooth muscle in the lung, thereby improving shunt fraction and optimizing the ventilation/perfusion ratio by selectively vasodilating aerated lung zones. Clinical trials have shown both agents to transiently improve oxygenation, but neither agent has been shown to improve survival.[53] Other pharmacologic therapies for ARDS, including sildenafil, corticosteroids, and exogenous surfactant, have not been shown to be consistently efficacious.

NEUROMUSCULAR BLOCKADE

A multicenter, double-blind trial of 340 patients with severe ARDS confirmed that early administration of a neuromuscular blocking agent (cisatracurium) improved 90-day survival and increased time off mechanical ventilation without increasing muscle weakness compared to placebo.[54] Enthusiasm for this approach is tempered with concern because current data do not address long-term follow-up for critical illness polyneuropathy and long-term mortality.

To address these issues, the Clinical Trials Network for the Prevention and Early Treatment of Acute Lung Injury (PETAL) launched the ROSE (Reevaluation of Systemic Early Neuromuscular Blockade) trial, a large, multicenter randomized controlled trial comparing early neuromuscular blockade and deep sedation with lighter sedation and no routine neuromuscular blockade. The estimated study completion date for all final data collection of the ROSE trial is April 2019.[55]

Current national guidelines by the American Thoracic Society and the Society of Critical Care Medicine recommend increasing intensity of treatment intervention for increasing severity of ARDS, with low tidal volume ventilation and higher PEEP in mild and moderate ARDS, and consideration of evidence-based treatment strategies including neuromuscular blockade, prone position, inhaled nitric oxide, recruitment maneuvers, and venous-venous extracorporeal membrane oxygenation (ECMO) in severe ARDS (P/F ratio ≤100) by Berlin criteria (Fig. 57-10).[56] For ICUs that manage severe ARDS and serve as regional ECMO referral centers, it is helpful to have an ARDS algorithm (Fig. 57-11; also see Fig. 59-4) for optimal evidence-based ARDS treatment.[43]

Weaning from Mechanical Ventilation

Managing and weaning a patient from mechanical ventilation are highly variable and the duration and success of weaning is dependent on each specific clinical scenario. It is additionally important to separate the principles of weaning from extubation despite the fact that the terminology is used interchangeably. As a set of general principles (1) weaning should be considered early in the course of a ventilated patient, (2) pressure support weaning is well tolerated, and (3) spontaneous breathing trial is the best diagnostic test to assess progress of weaning and determine if the patient is ready for liberation from mechanical ventilation and extubation.

Several factors influence successful weaning, including the type of injury sustained, preexisting medical problems,

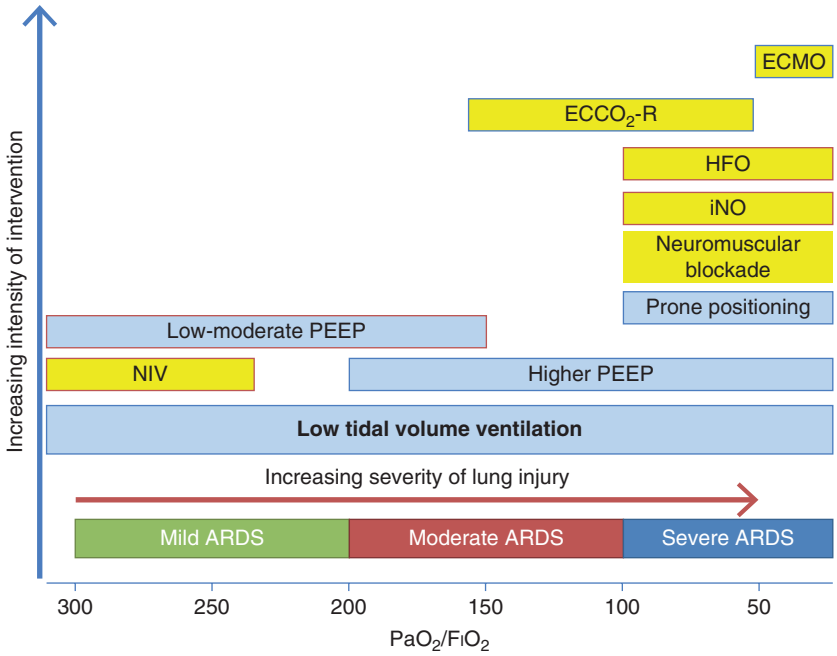


FIGURE 57-10 Increasing intensity of treatment intervention. ARDS, acute respiratory distress syndrome; $ECCO_2$-R, extracorporeal carbon dioxide removal; ECMO, extracorporeal membrane oxygenation; HFO, high-frequency oscillation; iNO, inhaled nitric oxide; NIV, noninvasive ventilation; PEEP, positive end-expiratory pressure.

patient toxicology, and the general state of health. Weaning should be directed toward decreasing WOB and improving ventilatory capacity (Table 57-6).

WOB is a function of the force required to expand the airways, lungs, and chest wall to an appropriate volume in order to meet oxygen and physiologic demands (pressure × volume/time). Excess WOB may be secondary to inappropriate ventilator settings, increased airway resistance, and decreased pulmonary or thoracic wall compliance. Additionally, increased WOB may also reflect higher CO_2 production from sepsis, fever, or overfeeding.

METHODS AND PROTOCOLS FOR WEANING

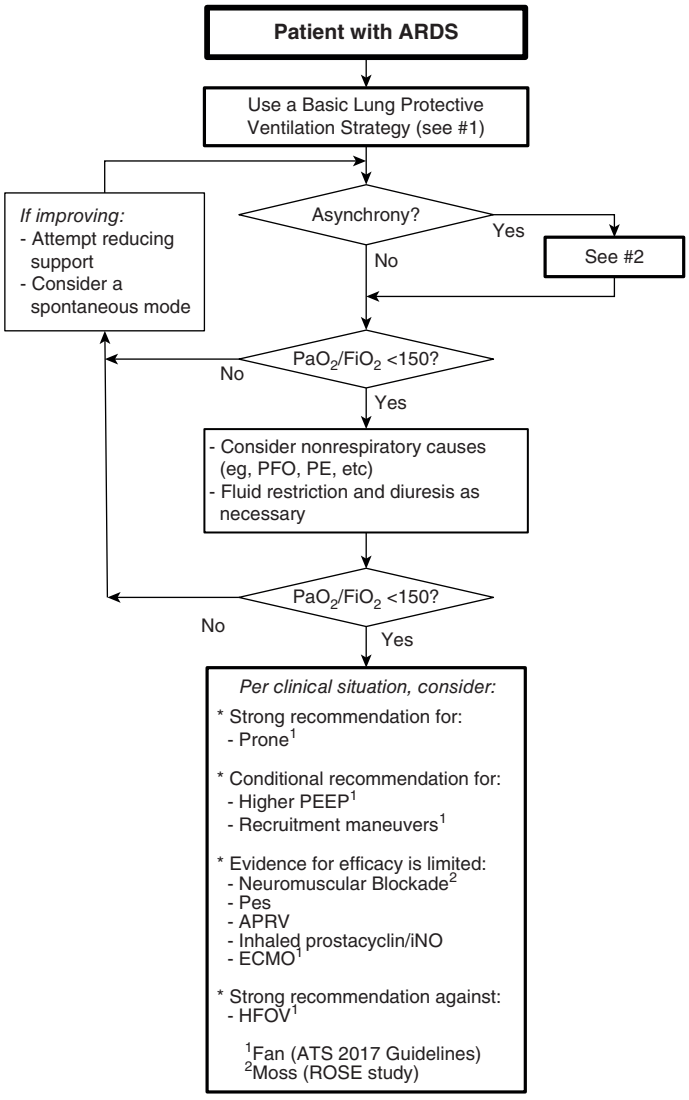
The process of weaning can be long and variable and may account for over 40% of total ventilator time.[44] There remains a lack of uniformity in weaning protocols, and there is a paucity of data available to support standardization. Nevertheless, weaning should focus on conditioning and strengthening the muscles of breathing. The methodology of weaning has evolved in the past three decades from a physician-driven approach to a modern protocol-based approach. Options include a "stepwise" methodology where patients' ventilatory assistance (pressure support or rate) is decreased in a stepwise manner. However, in the stepwise approach, there is no set period of unassisted respiratory exercise, and this approach may prolong mechanical ventilation, leading to chronic fatigue and deconditioning. This method is in contrast to the "sprint" approach (spontaneous breathing trials) where intermittent trials of spontaneous breathing are used once or twice daily and an assessment of WOB and ventilatory capacity is determined accordingly.

In an effort to empirically evaluate optimal weaning techniques, the Spanish Lung Failure Collaborative Group analyzed four weaning methods in an important, large (n = 546), multicenter, prospective trial. Patients were randomized in a stepwise progressive mode: (1) intermittent mandatory ventilation (IMV) where patients had a set mean frequency of 10 breaths/min and decreased, (2) pressure support (PS) where support was initially a mean of 18 cm H_2O and decreased; or into an intermittent spontaneous breathing method, (3) greater than 2/day with PS less than 5 cm H_2O, or (4) once-daily T-piece for 2 hours only. Both of the spontaneous breathing methods had higher rates of successful weaning than the stepwise methods.[57] Among the three most popular weaning modes, T-piece wean, PS wean, and IMV wean, current data suggest a trend toward longer duration of ventilation with IMV weaning compared to spontaneous trials; however, it is important to note that few data have shown any link between weaning method and mortality.[58]

The benefit of a formal protocol to hasten weaning is clear.[59] These protocols reduce physician practice variability and have been shown in several studies and meta-analyses to reduce ventilator and ICU days and weaning duration. Implementing an aggressive weaning protocol should incorporate the Screen, Trial, Exercise, Evaluate, and Report (STEER) principle. Using this protocol, a team of highly trained respiratory therapists, in conjunction with the ICU team, implement the following daily regimen on each mechanically ventilated patient:

Overview of ARDS Ventilator Management Strategies

University Hospital Respiratory Care
Michigan Medicine, Ann Arbor MI



1: Basic Lung Protective Ventilation

- ARDS Network ventilation strategy:
  a. Use VCV or PCV, targeting VT 6 mL/kg PBW
  b. Maintain Pplat ≤30 cm H_2O
     - Reduce VT to 5 or 4 mL/kg if necessary
  c. PEEP/FiO_2 per table (see bottom of page)
- Consider maintaining driving pressure ≤12-15 cm H_2O
- If consolidation is asymmetrical, consider placing 'good lung' in dependent position

2: Patient-Ventilator Asynchrony

* Consider minor ventilator adjustments (eg, flow rate & pattern, inspiratory pause)
* Assess potential to treat with pharmacologic agents (eg. sedation, NMB), especially in pt with severe ARDS and strong respiratory drive (double-trigger)
* For double-triggering, consider increasing VT 1 mL/kg (max 8 mL/kg), provided Pplat ≤30 cm H_2O
* For flow asynchrony, consider a variable flow pressure breath mode of ventilation:
  - Volume targeted PC (PRVC, VC+, Autoflow)
  - Pressure control, pressure support

FiO_2/PEEP Tables

Lower PEEP/Higher FiO_2 table

Step:	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7	0.7	0.8	0.9	0.9	0.9	1.0	1.0	1.0	1.0
PEEP	5	5	8	8	10	10	10	12	14	14	14	16	18	18	20	22	24

Higher PEEP/ Lower FiO_2 table (from ROSE study)

Step:	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
FiO_2	0.3	0.4	0.4	0.4	0.4	0.4	0.4	0.5	0.5	0.5	0.6	0.7	0.8	0.8	0.9	1.0	1.0
PEEP	5	5	8	10	12	14	16	16	18	20	20	20	20	22	22	22	24

Consider Lower PEEP for patients with low PEEP-responsiveness potential (ie, P/F ≥150); Higher PEEP if higher PEEP-responsiveness potential (P/F <150) or BMI >35

Prone Positioning

* Consider after initial 12-24 hrs of stabilization
* Use 16 hr/day (generally 4 pm to 10 am)
* Discontinue when:
  - Instability in prone position
  - Supine × 4 hr, PaO_2/FiO_2 >150 on FiO_2 ≤0.60 & PEEP ≤10

Higher PEEP

* For pts with PaO_2/FiO_2 <150, consider higher PEEP table

Recruitment Maneuvers

* Consider for pts with clear de-recruitment, negative Ptp or PaO_2/FiO_2 <150
* Recommend PCV with: 1) 40/20-25 for 1-3 min (as tolerated) or 2) delta-P of 15 and increase PEEP by 5 up to PIP of 40
* If CPAP method used, limit to 15-30 seconds
* Provider should be at bedside if pressures >40 cm H_2O used

Neuromuscular Blockade

* No benefit of routine use of NMB in moderate-severe ARDS.
* Consider use if significant asynchrony and concern for VILI.

Esophageal Pressure (Pes) Guided Therapy

* Informs of transpulmonary end-inspiratory (Ptp-plat) and end-expiratory (Ptp-PEEP) pressures
* Requires AVEA ventilator & placement of Pes catheter

Airway Pressure Release Ventilation (APRV)

* Increases Pmean with lower Pplat; lacks outcomes benefit
* Concern for P-SILI in pt with strong respiratory drive

Inhaled Nitric Oxide (iNO)

* Start at 10 ppm
* If positive response (improved oxygenation) or brought in by Survival Flight:
  - Maintain at 10 ppm and reduce FiO_2 down to 0.8, then titrate iNO down, or consider Veletri or iloprost, per Respiratory Care policy
* If no response, discuss with team to consider stopping

NOTE: *iNO is a very costly drug compared to alternatives*

Extracorporal Membrane Oxygenation (ECMO)

* Absolute contraindications: irreversible pulmonary process
* Evaluate, but lower survival if on vent 7-10 days pre-ECMO
* Consider if: PaO_2/FiO_2 <50 ×3 hrs or <80 ×6 hrs, or pH <7.25 w/ $PaCO_2$ >60 ×6 hrs

High Frequency Oscillatory Ventilation (HFOV)

* Strong recommendation against routine use; may have benefit if PaO_2/FiO_2 <64; goal is to increase Pmean

FIGURE 57-11 Acute respiratory distress syndrome (ARDS) mechanical ventilation algorithm. (Reproduced from the University of Michigan.)

TABLE 57-6: Factors Contributing to Respiratory Failure

Physiologic factors	Causes
Thoracic injury	Pain, disruption of chest wall stability (flail chest), large hematomas, persistent pneumothorax/hemothorax
Abdominal injury	Pain, disruption of abdominal accessory muscles, abdominal hypertension or compartment syndrome, ascites
Spinal cord injury	Loss of diaphragm and accessory motor strength, loss of airway protection capacity
Excessive agitation/anxiety	Inappropriate sedation or analgesia, post-TBI effects, acute alcohol or drug withdrawal, sepsis
Decreased pulmonary compliance	ARDS/ALI, edema, pulmonary fibrosis, empyema, severe pneumonia
Increased airway resistance	Bronchospasm, endotracheal obstruction or narrowing, mucus plugging of airway or bronchioles
Refractory hypoxia	Pneumonia, pulmonary emboli, ARDS/ALI, sepsis, barotrauma, pneumothorax/hemothorax
Hypercapnia	Sepsis, overfeeding syndrome, inadequate weaning parameters or settings
Malnutrition/metabolic	Persistent catabolic state, respiratory muscle atrophy, electrolyte abnormalities, pulmonary edema
Cardiac insufficiency	Decreased cardiac output (MI, blunt contusion, preexisting disease) causing hypoxia or pulmonary edema

ALI, acute lung injury; ARDS, acute respiratory distress syndrome; MI, myocardial infarction; TBI, traumatic brain injury.

- **S**creen for weaning contraindications (hypoxia, fevers, hemodynamic instability, high WOB, uncontrollable ICP, and sedation)
- **T**rial of minimum support breathing (ie, PS <5 cm H_2O)
- **E**xercise according to set protocol (ie, a 2-hour PS trial or T-piece trial)
- **E**valuate progress
- **R**eport the results to the ICU team

The Clinical Practice Guideline for Liberation from Mechanical Ventilation in Critically Ill Adults provides discrete evidence-based recommendations for all ICU patients (Table 57-7).[60]

EXTUBATION

The weaning process begins shortly after mechanical ventilation is initiated. However, extubation, the final removal of the artificial airway, should not be confused with weaning. Liberation from mechanical ventilation should be considered if the following parameters are met:

1. Reversal of underlying cause of respiratory failure
2. Adequate oxygenation (P/F ratio >150–200 with PEEP <5–8 cm H_2O, FIO_2 <0.4–0.5, and pH >7.25)
3. Hemodynamic stability
4. The capability to initiate a respiratory effort

TABLE 57-7: Guideline for Liberation from Mechanical Ventilation

Recommendation	Strength of recommendation	Certainty in the evidence (ie, quality of evidence)
1. For acutely hospitalized patients ventilated >24 h, we suggest that the initial SBT be conducted with inspiratory pressure augmentation (5-8 cm H_2O) rather than without (T-piece or CPAP).	Conditional	Moderate certainty in the evidence
2. For acutely hospitalized patients ventilated >24 h, we suggest protocols attempting to minimize sedation.	Conditional	Low certainty in the evidence
3. For patients at high risk for extubation failure who have been receiving mechanical ventilation for >24 h, and who have passed a spontaneous breathing trial, we recommend extubation to preventive NIV.	Strong	Moderate certainty in the evidence
4. For acutely hospitalized patients who have been mechanically ventilated for >24 h, we suggest protocolized rehabilitation directed toward early mobilization.	Conditional	Low certainty in the evidence
5. We suggest managing acutely hospitalized patients who have been mechanically ventilated for >24 h with a ventilator liberation protocol.	Conditional	Low certainty in the evidence
6a. We suggest performing cuff leak test in mechanically ventilated adults who meet extubation criteria and deemed high risk for PES.	Conditional	Very low certainty in the evidence
6b. For adults who have failed a cuff leak test but are otherwise ready for extubation, we suggest administering systemic steroids at least 4 h before extubation. A repeat cuff leak test is not required.	Conditional	Moderate certainty in the evidence

CPAP, continuous positive airway pressure; NIV, noninvasive ventilation; PES, postextubation stridor; SBT, spontaneous breathing trial.

TABLE 57-8: Criteria for Extubation

Criteria	Physical findings
Ability to maintain oxygenation	O_2 saturation above 95% with F_{IO_2} <0.4 on minimal CPAP or T-piece
Ability to maintain ventilation	RSBI <100 f/V_t, NIF >–30 cm H_2O, forced TV >10 mL/kg, $Paco_2$ <45mm Hg on minimal CPAP or T-piece
Ability to maintain airway	Appropriate mental status, adequate strength, intact gag and cough, positive endotracheal leak with deflated cuff
Ability to clear secretions	Paucity of secretions, adequate gag and cough reflex, adequate strength
Physiologic considerations	No indication of sepsis, adequate sedation and analgesia, no anticipated decrease in mental status

CPAP, continuous positive airway pressure; NIF, negative inspiratory force; RSBI, rapid shallow breathing index.

Prediction of successful extubation varies greatly, and factors that impact on successful extubation include tidal volume, NIF, vital capacity, rapid shallow breathing index (RSBI), and minute ventilation.[61] Patients with RSBI >105 breaths/min/L are 95% likely to fail, whereas those with an RSBI of less than 100 breaths/min/L are 80% likely to succeed.[61] Additional criteria should be considered when deciding upon extubation, including ability to protect the airway, secretion control, and sustainability (Table 57-8). TBI patients can be particularly difficult to manage because neurologic symptoms can change, making criteria for airway and secretion control difficult to evaluate. A Glasgow Coma Scale (GCS) score of greater than 8 is generally associated with the greatest extubation success. In TBI patients, early tracheostomy may be a consideration to establish definitive airway control (see the later section "Tracheostomy").

UNPLANNED EXTUBATIONS

Despite rigorous patient observation and appropriate sedation, unplanned extubations (UEs) remain a serious complication, with a reported incidence of 0.1 to 3.6 events per 100 intubation days.[62] Consequences of UE include aspiration, pneumonia, vocal cord trauma, hypoxia, cardiopulmonary arrest, and death. Risk factors for UE include male gender, chronic obstructive pulmonary disease, agitation, lower sedation, and less experienced nurses. UEs require reintubation in 33% to 58% of cases, with the majority (62%–74%) occurring within 1 hour. When required, reintubation increases mortality. Predictors of reintubation include GCS less than 11, age greater than 65 years, P/F ratio of less than 200, and pneumonia. Preventing UE requires attention to endotracheal tube security, particularly during transfers, bedside procedures, or prone positioning. Adequate sedation and analgesia are paramount, and physical restraints, especially in patients with high sedative tolerance, may be temporarily necessary. Protocols and checklists to prevent UE including nursing and physician education, standardization of procedures, patient surveillance, and identification of high-risk patients can reduce the risk of UE by 22% to 53%. An emergency ICU protocol should be in place for UE because reintubation may be difficult and a team of experts with appropriate experience that can provide a surgical airway should be immediately present.

NUTRITION SUPPORT (SEE CHAPTER 62)

Current national guidelines indicate the need for nutritional assessment, support, and monitoring for ICU patients.[63] Early enteral nutrition, which may help preserve gut mucosal barrier function, is the preferred approach for trauma patients. Nutritional assessment should begin with an assessment of the degree of preinjury malnutrition as well as current requirements. The literature to date suggests serial weight measurements and serum albumin do not predict outcomes in the critically ill. Calculation of the Nutrition Risk in the Critically Ill (NUTRIC) score is the best nutritional assessment measure for ICU patients. Indirect calorimetry (metabolic cart) provides an assessment of caloric requirements and metabolic rate to determine optimal nutrition requirements by calculation of the respiratory quotient (RQ). The RQ can be used to determine the prominent nutritional substrate being used, with excess carbohydrates leading to RQs of 1.0 or more and RQs below 0.8 indicating possible excess lipid utilization.

GLYCEMIC CONTROL IN THE INTENSIVE CARE UNIT

Hyperglycemia with or without insulin resistance is common in critically ill patients. Studies have also shown that hyperglycemia in trauma patients correlates with higher mortality rates.[64] The trial by van den Berghe et al[65] demonstrated that tight glucose control (≤110 mg/dL) with intensive insulin therapy improved mortality and reduced complications such as infection rate, multiorgan failure rate, ventilator days, and morbidity. However, the intensive insulin regimen initially proposed by van den Berghe et al[65] was associated with increased hypoglycemia and failed to show a mortality benefit in subsequent multi-institutional studies, including the Glucontrol and Normoglycemia in Intensive Care Evaluation Survival Using Glucose Algorithm Regulation (NICE-SUGAR) trials.[66,67] In the NICE-SUGAR trial, 90-day mortality was increased in patients assigned to intensive insulin therapy, as compared with an intermediate target range for blood glucose. Current national guidelines recommend improved glucose control in ICUs; however, a moderate protocol (goal glucose level ≤180 mg/dL) is recommended

to prevent hyperglycemia without inducing higher mortality or hypoglycemic events.[68]

PREVENTION OF HOSPITAL-ACQUIRED INFECTIONS (SEE CHAPTER 21)

Mortality after trauma in the ICU is frequently related to infection. Infections may be related to specific injuries (eg, open fractures), specific procedures required (eg, anastomotic leak after intestinal resection and anastomosis), or hospital-acquired infections (HAIs) during ICU management (eg, ventilator-associated pneumonia [VAP], central line–associated bloodstream infection [CLABSI], and catheter-associated urinary tract infection [CAUTI]). In a study using multiple regression analysis, the most common predictors of infection were central venous catheters, mechanical ventilation, chest tubes, and open fractures.[56] Patients at risk for infections after injury should be routinely tested by culture and examined for clinical indications such as fever, leukocytosis, change in physical examination, pyuria, and development of purulent sputum or new infiltrate on chest x-ray. HAIs (VAP, CLABSI, and CAUTI) have standardized definitions by the National Healthcare Safety Network and the Centers for Disease Control and Prevention. All HAI preventive strategies should be implemented in optimal ICU care and treatment, including VAP prevention (Table 57-9).[69]

The decision to start presumptive (empiric) antibiotics should be based on risk factors, the expected sequelae of injury, and prior infections. Presumptive treatment should be started, if indicated; stopped at a defined end point for culture-negative patients; and appropriately deescalated when final culture information and sensitivities are available. Fungal sepsis should be considered in patients with prolonged ICU stays, multiple prior antibiotic therapies, and immunosuppression. Measurement of procalcitonin levels when a fever workup is initiated can be helpful in determining the likelihood of infection as the etiology of fever in the ICU.

TABLE 57-9: Ventilator-Associated Pneumonia Prevention Strategies

Ventilator bundle: 10 elements
1. Adherence to hand hygiene guidelines
2. Daily assessment of readiness to wean
3. Elevation of head of bed between 30° and 45°
4. Lack of gastric overdistention
5. Subglottic suctioning (CASS endotracheal tubes)
6. Endotracheal cuff pressure of at least 20 cm
7. Stress ulcer prophylaxis
8. Comprehensive oral care with chlorhexidine
9. Prevent condensate in ventilator circuits
10. Deep vein thrombosis prophylaxis

CASS, continuous aspiration of subglottic secretions.

Infectious Complications

Two particularly troublesome and expensive nosocomial infections that occur during critical care after major trauma are CLABSI and VAP. One of the principal issues regarding VAP is its effect on outcome after major injury. There is evidence that pneumonia may exacerbate multiple organ failure and ARDS and could be associated with increased mortality, as has been found in other studies. CLABSI rates range from 1.2 to 5.6 per 1000 central venous catheter days with a marked increase in risk after 9 catheter days. CLABSIs are also associated with longer length of stay, increased cost, and an attributable mortality of 12% to 25%.[70]

ANEMIA PREVENTION AND MANAGEMENT IN THE INTENSIVE CARE UNIT

One source of anemia well within the control of the surgical intensivist is phlebotomy-related blood loss. Average ICU phlebotomy losses were 436 mL for all ICU patients and 679 mL for patients with an ICU length of stay of at least 4 days.[71] Studies have also demonstrated that small changes in the amount of blood drawn, as little as 3.5 mL/d, can significantly increase risk for blood transfusion in the ICU. All efforts should be made in the ICU to reduce the number of serial labs, eliminate unnecessary studies, and use low-volume adult or pediatric testing tubes for all necessary tests. Blood reinfusion devices (eg, the SafeSet device) should be used on all arterial lines to avoid discard of waste blood with diagnostic laboratory testing. Additionally, technology such as continuous noninvasive hemoglobin monitors may avoid unnecessary serial blood tests and reduce the percentage of patients receiving transfusions as well as the average volume of blood transfused per patient.

Blood transfusions are common in the ICU for treatment of anemia, with 30% to 62% of critically ill patients receiving an average of 2 to 4 units of packed red blood cells. Blood transfusions independent of shock or injury/disease severity are associated with worse outcomes. Increased infection, multiple organ dysfunction, and mortality are directly correlated with the amount of blood transfused.[72] Lowering target hemoglobin from 10–12 to 7–9 g/L was associated with improved outcomes in ICU patients in the Transfusion Requirements in Critical Care (TRiCC) study.[73] The TRiCC trial results have been reaffirmed by multiple subsequent studies and meta-analyses in various ICU and surgical populations including cardiac disease, respiratory failure, and those on mechanical ventilation. In addition to improved survival, restrictive transfusion strategies were associated with reductions in rebleeding, acute coronary syndrome, pulmonary edema, and bacterial infections without adversely affecting functional recovery.[74,75]

Despite initial support for liberal transfusion among septic patients, subsequent studies have failed to demonstrate any benefit to transfusion triggers above 7 g/L within this

population. The Surviving Sepsis Campaign Guidelines from 2016 support a transfusion trigger of less than 7.0 g/dL with a target hemoglobin concentration of 7 to 9 g/dL.[15]

The appropriate trigger for patients with TBI remains unclear.[76] However, recent studies have demonstrated increased mortality with transfusions in TBI patients with hemoglobin levels greater than 10 g/dL, no benefit in neurologic outcome, and a significantly increased risk of thromboembolic events and transfusions with a trigger of 10 g/dL when compared to a trigger of 7 g/dL.[77,78]

The 2016 Clinical Practice Guidelines for Red Blood Cell Transfusion Thresholds from the American Association of Blood Banks (AABB)[79] recommended the following: (1) it is good practice to consider the hemoglobin level, the overall clinical context, patient preferences, and alternative therapies when making transfusion decisions regarding an individual patient; and (2) a restrictive RBC transfusion threshold in which the transfusion is not indicated until the hemoglobin level is 7 g/dL is recommended for hospitalized adult patients who are hemodynamically stable, including critically ill patients, rather than when the hemoglobin level is 10 g/dL (strong recommendation, moderate quality evidence). A restrictive RBC transfusion threshold of 8 g/dL is recommended for patients undergoing orthopedic surgery or cardiac surgery and those with preexisting cardiovascular disease (strong recommendation, moderate quality evidence). The restrictive transfusion threshold of 7 g/dL is likely comparable with 8 g/dL, but randomized controlled trial evidence is not available for all patient categories. These recommendations do not apply to patients with acute coronary syndrome, severe thrombocytopenia (patients treated for hematologic or oncologic reasons who are at risk of bleeding), and chronic transfusion-dependent anemia (not recommended due to insufficient evidence).

Erythropoiesis-Stimulating Agents

The anemia of critical illness resembles that of chronic inflammatory disease; low circulating erythropoietin levels and high hepcidin levels are the causative factors, which result in iron-restricted erythropoiesis.[80] Erythropoiesis-stimulating agents (ESAs; epoetin-alfa and darbepoetin) are used for treatment of anemia due to chronic kidney disease but are currently not indicated for the treatment of anemia of critical illness. Initial clinical trials (EPO-1 and EPO-2) documented improved hemoglobin levels or transfusion reduction compared to placebo without use of a transfusion protocol.[81,82] Subsequent studies (EPO-3) confirmed an increase in hemoglobin with ESA treatment without a reduction in transfusions.[83] A subset analysis of the trauma patients in the EPO-2 and EPO-3 studies suggested there was an approximately 50% reduction in 29-day trauma mortality with ESAs in the trauma cohort.[84] This survival benefit was redemonstrated in patients with TBI.[85-87]

A recent meta-analysis[88] included nine eligible studies that randomly assigned 2607 critically ill patients after trauma to an ESA or placebo (or no ESA). Compared with placebo (or no ESA), ESA therapy was associated with a substantial reduction in mortality (risk ratio, 0.63; 95% CI, 0.49–0.79; P = .0001, I = 0%). In patients with TBI, ESA therapy did not increase the number of patients surviving with moderate disability or good recovery (risk ratio, 1.00; 95% CI, 0.88–1.15; P = .95; I = 0%). With the dosing regimens employed in the included studies, ESA therapy did not increase the risk of lower limb proximal deep venous thrombosis (risk ratio, 0.97; 95% CI, 0.72–1.29; P = .78; I = 0%). The authors concluded that the administration of ESAs to critically ill trauma patients was associated with a significant reduction in mortality without an increase in the rate of lower limb proximal deep venous thrombosis. Given the worldwide public health significance of these findings, additional research to validate or refute these findings is required.

PAIN, AGITATION, AND DELIRIUM IN THE INTENSIVE CARE UNIT

Patients with traumatic injury may be suffering severe pain and may require directed and effective analgesia. Significant trauma may lead to prolonged critical illness, mechanical ventilation, and long-term sedation. Many trauma patients also have a history of substance abuse and are at risk for acute withdrawal symptoms and may have opiate and benzodiazepine tolerance. Inadequate control of pain, agitation, and delirium (PAD) can lead to patient suffering, additional complications, and unnecessarily prolonged intubation, mechanical ventilation, and ICU stay. Consensus CPGs exist for management of PAD[89] and were updated in 2018.[90-92]

The need for adequate analgesia and sedation must be balanced against the risks of oversedation. Oversedation may result in hemodynamic instability, increased length of stay and costs, increased respiratory complications including VAP, and possibly long-term decreases in cognitive function. Oversedation may also increase the risks of delirium and possibly posttraumatic stress disorder. Delirium is related to increased length of hospital stay, increased health care costs, and higher mortality. ICU care may lead to pain and discomfort due to catheters, drains, endotracheal tubes, and performance of routine nursing care such as airway suctioning, physical therapy, dressing changes, and an enforced mobilization. Most academic ICUs have adopted algorithms for assessment and management of PAD. An example of an ICU patient assessment and management PAD algorithm is shown in Fig. 57-12.

Pain and Choice of Parenteral Opioid for Analgesia

Pain is almost universal in trauma patients; it has been described as an unpleasant sensory or emotional experience that is associated with tissue damage or described in terms of tissue damage. Many patients later recall unrelieved pain when interviewed about their ICU stays. It is a standard of practice to regularly and frequently assess pain; this is readily

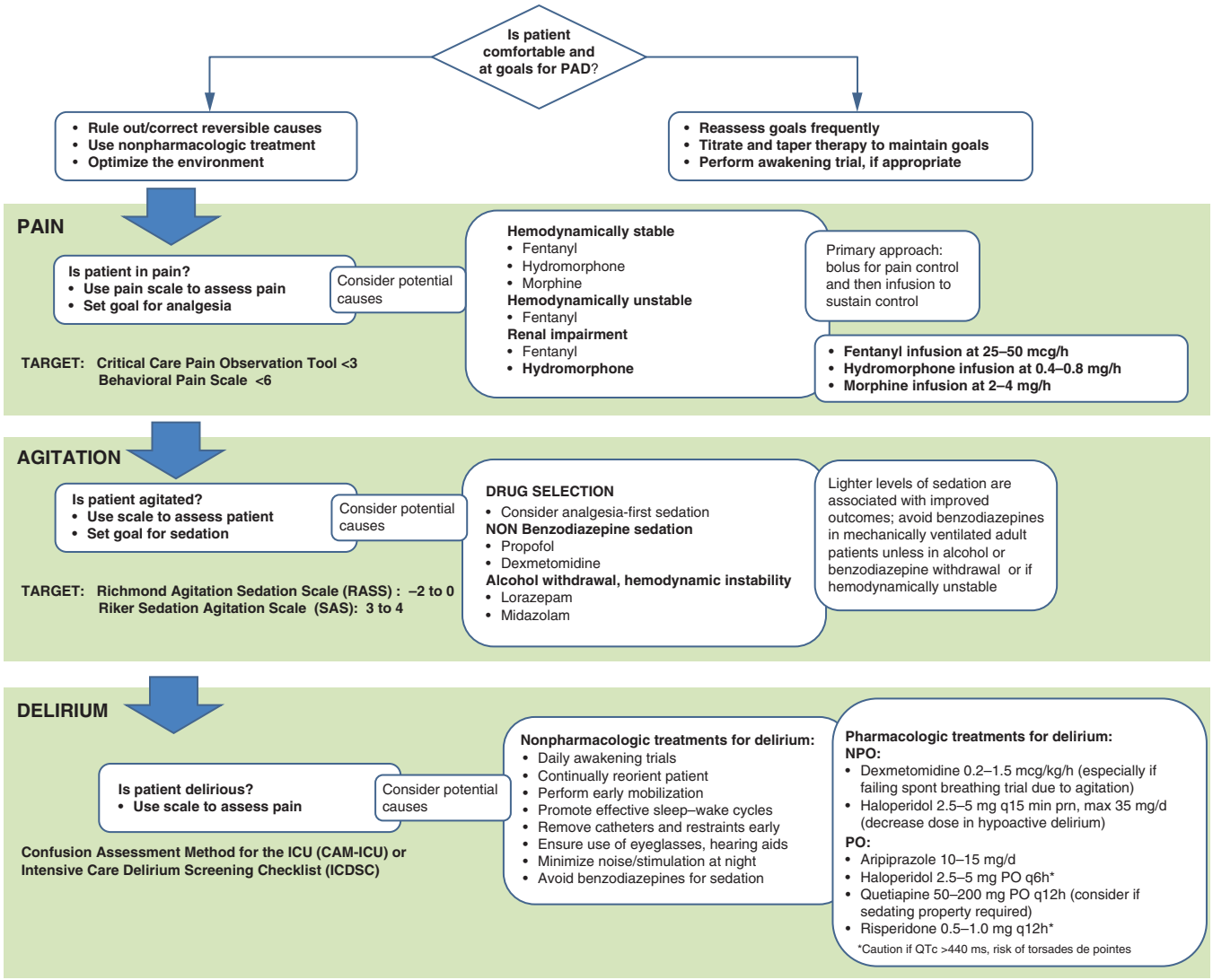


FIGURE 57-12 Algorithm for pain-agitation-delirium (PAD) management in the University of California, San Diego surgical intensive care unit (ICU). NPO, nothing by mouth; PO, oral.

accomplished by the use of validated pain scores. The Joint Commission has labeled the pain score "the fifth vital sign." The Behavioral Pain Scale (BPS) and the Critical-Care Pain Observation Tool (CPOT) are the most valid and reliable behavioral pain scales for monitoring pain in medical, postoperative, or trauma (except for TBI) adult ICU patients who are unable to self-report and in whom motor function is intact and behaviors are observable.[93,94] Parenteral opioids have a long history of efficacy and safety for pain and ICU patients. Because bioavailability and efficacy are variable in ICU patients, the intravenous route is to be used whenever possible. Intramuscular and subcutaneous routes should be avoided due to irregular absorption and highly variable serum levels. Patient-controlled analgesia may be of great utility in patients able to use it due to the self-control and immediate administration provided to the patient. Recent guidelines have recommended preferred parenteral agents in the treatment of pain for adult ICU patients. Table 57-10 lists characteristics of the preferred agents in the SCCM PAD guideline: morphine, fentanyl, and hydromorphone (Dilaudid).

Morphine remains the most commonly used opioid analgesic in the ICU setting due to clinician familiarity in dosing and low cost. It penetrates the blood–brain barrier slowly due to its poor lipid solubility. This results in a delayed peak effect and a long-acting effect as compared with more lipid-soluble agents such as fentanyl. Morphine does not have a direct negative inotropic effect; however, it has predictable effects of arterial and venous dilation. These effects suggest morphine may be advantageous in the hemodynamically stable or hypertensive patient with myocardial ischemia or cardiogenic pulmonary edema. Disadvantages of morphine and other opioid agonists are the opioid receptor–associated side effects, the most serious of which is centrally mediated respiratory depression. Other effects include sedation, nausea, and

TABLE 57-10: Suggested Intravenous Opioid Doses for Adult ICU Patients[a]

Agent	Approximate equivalent single IV dose (mg)	Typical infusion rate	Onset peak effect (min)	Duration	Average price per day[a]	Comments
Fentanyl	100–200 mcg	50–200 mcg/h	2–5 min	0.5–2 h	$26/d at a rate of 100 mcg/h	Fastest onset and shortest duration, least hemodynamic effect.
Hydromorphone	1.5–2 mg	0.2–3 mg/h	20–30 min	3–4 h	$23/d at a rate of 1.6 mg/h	5–10× more potent by weight than morphine.
Morphine	10 mg	2–10 mg/h	20–30 min	3–4 h	$20/d at a rate of 10 mg/h	Avoid in hypotension. Active metabolite accumulates in renal dysfunction. May cause itching due to histamine release (not a true allergy). Decreases preload, which may be beneficial in pulmonary edema.

[a]UCSD Medical Center 2/2015, will vary depending on institution.
ICU, intensive care unit; IV, intravenous.

sphincter of Oddi spasm. Adverse effects can be reversed if necessary with careful titration of the opioid receptor antagonist naloxone. Naloxone must be administered with care because access effect is associated with undesirable hemodynamic effects such as hypertension, tachycardia, and myocardial ischemia as well as acute pulmonary edema. Morphine is also associated with a non–receptor-associated effect of histamine release, and this can result in undesired hypotension, tachycardia, and possibly exacerbating bronchospasm in patients with reactive airway disease. Prolonged use of morphine, especially in renal impairment, can result in the accumulation of a metabolite, morphine-6 glucuronide, and may result in a prolonged sedative effect.

Fentanyl is a lipid-soluble synthetic opioid. Fentanyl is 10 times more potent than morphine and has a more rapid onset of action, requiring vigilance with its use. It is also inexpensive, with costs similar to morphine. With small doses, the duration of action is short due to redistribution. When large doses are administered, especially by continuous infusion, termination of effect requires elimination, probably due to the saturation of poorly perfused adipose tissue. The pharmacokinetics of fentanyl are not much altered by the presence of cirrhosis, and clearance appears to remain normal in renal failure. Hemodynamically, fentanyl maintains cardiovascular stability and does not have significant negative inotropic effect. In the presence of high sympathetic tone, fentanyl may decrease blood pressure indirectly by decreasing central sympathetic output. Fentanyl also predictably causes a decrease in heart rate by a central vagotonic effect. The receptor-associated side effects of fentanyl and their management are the same as described for morphine. Unlike morphine, fentanyl does not release histamine and therefore may be a better choice in patients who are hemodynamically unstable or who have reactive airway disease. For ICU patients, fentanyl has been recommended as an alternative to morphine in situations of hemodynamic instability, known allergy to morphine, or previous histamine release with morphine.

Hydromorphone is a semisynthetic opioid agent that is lipophilic and 5 to 10 times more potent than morphine. Time to onset of action and duration of action are similar to those of morphine, and hydromorphone's terminal half-life is 184 minutes. Hydromorphone appears to have minimal hemodynamic effect and does not result in release of histamine. Like morphine and fentanyl, hydromorphone is inexpensive and is recommended as a third-line agent after morphine and fentanyl.

Agitation

The SCCM PAD guidelines recommend regular assessment and response to therapy.[90] The appropriate target level of sedation is a calm patient who can be easily aroused with maintenance of the normal sleep–wake cycle. Some patients require deeper levels of sedation to facilitate mechanical ventilation or reduce ICP. Use of a sedation scale such as the Richmond Agitation Sedation Scale (RASS) or Riker Sedation Agitation Scale (SAS) is common in most ICUs.[95] The choice of intermittent or continuous sedation is important; intermittent sedation relies on recognition of agitation and subsequent administration of a sedative. Continuous sedation reduces the likelihood of delay in administration of sedatives; however, continuous sedative infusions for ICU patients have been shown to increase the duration of mechanical ventilation and length of ICU stay. Weaning of patients from mechanical ventilation is often hampered by oversedation.

The benzodiazepines (midazolam and lorazepam) have previously been the most commonly used agents in the ICU for sedation. Propofol and dexmedetomidine have expanded the number of agents available for ICU sedation. The provision of anxiolysis and amnesia are of major importance for

critical care patients undergoing intermittently painful procedures or mechanical ventilation. It has been demonstrated that the majority of patients surviving prolonged mechanical ventilation found memory of the experience to be unpleasant.

The PAD guidelines recommend the use of propofol or dexmedetomidine and reduced use of benzodiazepines for sedation due to a possible increased risk of delirium. A meta-analysis of six trials ranked as moderate to high quality suggested that sedation with benzodiazepines may increase ICU length of stay by approximately 1.62 days (P = .0007) and duration of mechanical ventilation by 1.9 days (P <.00001) compared with nonbenzodiazepine sedation.[96] Benzodiazepines may have their greatest utility in the ICU for procedural sedation and in management of substance abuse withdrawal. Characteristics of commonly used benzodiazepines, dexmedetomidine, and propofol are found in Table 57-11.

Propofol is a lipid-soluble alkyl phenol intravenous anesthetic that is insoluble in water and formulated in a lipid emulsion. It has hypnotic, amnestic, and antiemetic properties but is devoid of analgesic effect. Propofol has minimal active metabolites, even with repeated administration. The sedative effect is rapid and predictable, and recovery occurs quickly when the drug is terminated. Compared to benzodiazepines, duration of mechanical ventilation is significantly shorter (2.6 days) in patients sedated with propofol.[96] Propofol has predictable hemodynamic effects, including arterial and venous dilation, decreased inotropic effect, and decrease in systolic blood pressure by 20% to 30%. Propofol given as a loading dose will cause profound ventilatory depression and may induce hypotension. For this reason, propofol is limited to mechanically ventilated patients only.

The 2018 SCCM PAD guidelines recommend the use of either propofol or dexmedetomidine over benzodiazepines for sedation in critically ill, mechanically ventilated adults. Patients on propofol for longer than 24 hours or requiring high doses need to be monitored carefully for propofol infusion syndrome. Hypertriglyceridemia and lipid-induced pancreatitis have been reported during prolonged infusion of propofol in the ICU, suggesting that serum triglyceride levels should be monitored in these patients. A lipid solution of propofol also supports rapid bacterial growth at room temperature, and a number of postoperative bacteremias have been linked to poor administration technique.

Dexmedetomidine is an imidazole compound that is a highly selective agonist of the α_2-adrenergic receptor with eight times greater affinity than clonidine. It is also shorter acting than clonidine, allowing use as an intravenous infusion. Initially evaluated as an anesthetic, it was found to be associated with excess bradycardia and hypotension. Lower doses produce reliable sedation, and dexmedetomidine is approved in the adult ICU patient as a sedative infusion. Patient selection and proper drug infusion are needed to avoid significant hemodynamic effects. Avoiding the initial bolus dose can reduce the incidence of significant bradycardia and hypotension on administration. Dexmedetomidine produces sedation but easy arousal, analgesic-sparing effect, and minimal depression of the respiratory drive. These characteristics are unique in that patients appear to be sedated but are readily roused and interactive and can follow commands. This makes the drug highly suited for patients who are being weaned from the ventilator, especially those who become agitated when other sedation is reduced. Dexmedetomidine is cost-effective because it reduces patient ventilator days, delirium, and ICU length of stay as compared with midazolam.

Monitoring and Daily Awakening

Monitoring of sedation within the ICU can be performed by the Ramsey or RASS scales (Table 57-12). The scales have midrange scores that indicate a calm, cooperative patient, with high and low scores indicating excess agitation or oversedation. A common sedation scale should be used throughout the institution for consistent and effective use. Daily interruption of continuous intravenous sedation until awakening of mechanically ventilated patients decreases duration of mechanical ventilation and ICU length of stay. Combination of daily awakening with spontaneous breathing trials results in patients spending less time on mechanical ventilation, less time in a sedative coma, and less time in the ICU and in the hospital. Daily awakening trials are most effective when following a standardized nurse-driven protocol. Patients who should be excluded from a daily awakening trial include those with increased ICP or neuromuscular blockade, those with hemodynamic instability, those requiring high levels of ventilatory support, and those who are post–coronary artery bypass grafting.

The daily awakening period should be synchronized with spontaneous breathing trials; in a study of 336 mechanically ventilated ICU patients, doing so achieved significant reduction in ventilator-free days and ICU and hospital length of stay.[97] The daily awakening also provides an opportunity for early exercise and mobilization (physical and occupational therapy) (Table 57-13). A recent study of early mobilization during daily awakening in 104 mechanically ventilated patients demonstrated increased independent functional status at hospital discharge, shorter duration of delirium, and increased ventilator-free days during the 28-day follow-up period compared with controls.[98]

The 2018 PAD Guidelines suggest using light sedation (vs deep sedation) in critically ill, mechanically ventilated adults. A recent trial of no sedation[99] in mechanically ventilated patients compared no sedative medications with control patients on continuous midazolam drips. Most of the eligible ICU patients could not be enrolled because they had medical indications for deep sedation. Patients in the no sedation group were allowed morphine boluses and had frequent nonpharmacologic antianxiety interventions such as reassurance by nurses and reassessment by physicians of pain orders and the need for tubes and catheters. Patients in the no sedation group had significantly shorter duration of mechanical ventilation and hospital and ICU length of stay but also had increased delirium and increased use of haloperidol.

TABLE 57-11: Suggested Intravenous Sedation Agents for Adult ICU Patients[a] (San Diego Patient Safety Council)

Agent	Typical IV bolus dose	Typical infusion rate	Onset peak effect	Duration	Average price per day[a]	Comments
Propofol	0.03–0.15 mg/kg (max 20 mg)	5–80 mcg/kg/min	1–2 min	<20 min	$36/d at a rate of 50 mcg/kg/min	Fastest onset and shortest duration. Avoid in hypotension. Dose/rate related hypotension/ bradycardia. Avoid IV push bolus due to increased risk of hypotension (if bolus required and low risk of hypotension, limit dose to 10–20 mg). Monitor triglycerides. Provides 1.1 kcal/mL. Monitor for propofol-related infusion syndrome.
Dexmedetomidine	Bolus not recommended: 1 mcg/kg over 20 min	0.2–1.5 mcg/kg/h	30 min	2–4 h	$408/d at a rate of 0.8 mcg/kg/h	No respiratory depression—consider for patient failing spontaneous breathing trial due to agitation/anxiety. Dose/rate related hypotension and bradycardia–bolus not recommended. May cause hyper/hypotension. 10 times cost of midazolam, may not be suitable during resuscitation due to hypotensive effect.
Lorazepam	1–3 mg	2–10 mg/h	15–20 min	2–4 h	$38/d at a rate of 4 mg/h	Slower onset but longer duration. Risk of propylene glycol toxicity with high doses (anion-gap acidosis, ↑ serum creatinine, ↑ lactate). Monitor serum osmolality if rate >6 mg/h and consider possible PG toxicity if osmol gap >10–15. Lorazepam is associated with increased risk of delirium and should be avoided in elderly and mechanically ventilated patients.
Midazolam	1–6 mg	1–10 mg/h	5–10 min	1.5–2 h	$70/d at a rate of 8 mg/h	Fast onset—amnestic effect. Active metabolite accumulates in renal dysfunction. Midazolam 2–3 mg is approximately equivalent to 1 mg lorazepam. Midazolam is associated with increased risk of delirium and should be avoided in elderly and mechanically ventilated patients.

[a]UCSD Medical Center 2/2015, will vary depending on institution.

ICU, intensive care unit; IV, intravenous; PG, propylene glycol.

TABLE 57-12: Richmond Agitation Sedation Scale (RASS)

Target RASS value	RASS description
+4 Combative	Combative, violent, immediate danger to staff
+3 Very agitated	Pulls or removes tubes or catheter; aggressive
+2 Agitated	Frequent nonpurposeful movement; fights ventilator
+1 Restless	Anxious, apprehensive but movements not aggressive or vigorous
0	Alert and calm
−1 Drowsy	Not fully alert, sustained awakening to voice (eyes open and contact >10 s)
−2 Light sedation	Briefly awakens to voice (eye opening and contact <10 s)
−3 Moderate sedation	Movements or eye opening to voice (but no eye contact)
−4 Deep sedation	No response to voice; moves or eyes open to physical stimulation
−5 Unarousable	No response to voice or physical stimulation

TABLE 57-13: Daily Awakening Trials—Summary Recommendations

Components recommended for a Daily Awakening Trial:
- Consistency—should follow a standardized nurse-driven protocol
- Continuity—need to ensure a set time for the patient during daylight hours
- Coordination—need to ensure Daily Awakening Trial is coordinated with other disciplines, specifically physical therapy, occupational therapy, and respiratory therapy activities

Exclusions to a Daily Awakening Trial:
- Increased intracranial pressure issues
- Neuromuscular blockade
- Pressure-regulated pulmonary ventilation with an inverse ratio
- Coronary artery bypass graft immediate postoperation

Process for weaning drug (per drug)[a]:
- Target—use sedation scale targets (−1 on the RASS)
- Sedatives—decrease by 50% or off
- Narcotics—consider reducing narcotics if still sedated (or SAS of 3–4)

Assess as part of Daily Awakening Trial:
- Pain
- Agitation
- Delirium
- Spontaneous Breathing Trial/rapid shallow breathing index

If continued sedation required, start at lower dose than previously—50% of original or lowest effective dose during titration—the most recent titration dose (before reaching −1 RASS)
- Bolus and titrate up to reach target goal as appropriate; do not resume at previous rate

[a]Abort Daily Awakening Trial if patient becomes physiologically unstable during procedure.

RASS, Richmond Agitation Sedation Scale; SAS, Riker Sedation Agitation Scale.

Delirium

Delirium is defined as fluctuation in mental status such as inattention, disorganized thinking, hallucinations, disorientation, and an altered level of consciousness. It occurs in up to 65% of hospitalized patients and up to 87% of patients admitted to the ICU. Delirium in the elderly patient is associated with a doubling of mortality. Delirium can increase hospital length of stay and increase health care cost. There has been discussion of ICU delirium rates as being a possible quality measure.

Delirium must be considered when assessing pain and sedation in the ICU. Delirium is further defined as follows: (1) *hyperactive* delirium: characterized by hypervigilance, restlessness, anger, irritability, and uncooperativeness, and associated with better overall outcomes; (2) *hypoactive* delirium: the more common and deleterious, characterized by a lack of awareness, decreased alertness, sparse or slow speech, lethargy, decreased motor activity, and apathy; and (3) *mixed* delirium: apparent in patients with a mixed clinical picture. Hypoactive delirium may be mistaken for depression or stupor and therefore is often not recognized, which may contribute to higher 6-month mortality of 32.0% versus 8.7% for mixed delirium.[100]

Delirium occurs in patients typically 24 to 72 hours after admission to the ICU. Preexisting risk factors for delirium include cognitive impairment, chronic illness (including hypertension), age greater than 65 years, depression, smoking, alcoholism, and severity of illness. Risk factors arising during hospitalization include congestive heart failure, sepsis, prolonged restraint use, immobility, withdrawal from substance abuse, seizures, dehydration, hyperthermia, head trauma, intracranial mass lesions, and use of lorazepam, midazolam, morphine, fentanyl, and propofol.

Assessment of delirium should be carried out in association with pain and sedation assessments and can be facilitated by the use of a validated delirium score such as the Confusion Assessment Method for the Intensive Care Unit (CAM-ICU) or the Intensive Care Delirium Screening Checklist (ICDSC); these tools may be most usefully administered during a daily awakening.[90] In addition, the patient should be assessed for QTc prolongation on electrocardiogram (ECG) if therapy is being considered because many antidelirium agents are associated with QTc prolongation and risk of torsade de pointes. CAM-ICU and ICDSC allow rapid consistent assessment for altered level of consciousness, disorganized thinking, inattention, and other delirium features.

Nonpharmacologic measures are the primary treatment used to manage delirium and include daily awakening trials, continuous reorientation of the patient by nursing staff, keeping clocks and calendars visible to the patient, promoting effective

sleep–awake cycles, timely removal of restraints and catheters, ensuring use of glasses and hearing aids, minimizing ICU noise and stimulation, and avoiding use of benzodiazepines. There are no double-blind, randomized, placebo-controlled trials that are adequately powered to prove the efficacy or safety of any antipsychotic agent in the management of delirium in ICU patients, although these are still commonly used by intensivists in the United States (Table 57-14).[101] The 2018 PAD Guidelines recommend dexmedetomidine, which may be especially useful in patients failing spontaneous breathing trials secondary to agitation. Haloperidol is *not* currently recommended and has been associated with a reduced seizure threshold, extrapyramidal reactions (dyskinesia), and laryngeal dystonia. It has also been associated with malignant neuroleptic syndrome. Large doses of intravenous haloperidol should only be administered in a monitored critical care setting and with monitoring of QTc intervals. Oral agents are longer acting and include aripiprazole, risperidone, or oral haloperidol. These agents are associated with QTc prolongation and should only be administered if the measured ECG QTc is less than 440 milliseconds. Another oral delirium agent with greater sedative properties is quetiapine; it is not strongly associated with QTc prolongation.

TRACHEOSTOMY

Tracheostomy in a chronically ventilated patient can offer several advantages including improved oral care, improved pulmonary toilet, decreased sedation requirements, improved patient comfort, decreased oral and vocal cord trauma, decreased airway resistance and dead space, and the ability to more aggressively wean ventilation. However, tracheostomy can be complicated by infection, subcutaneous emphysema, hemorrhage, tracheal stenosis, tracheoinnominate and tracheoesophageal fistula, and tracheomalacia.

Significant controversy exists regarding optimal timing for tracheostomy. Definitions of early tracheostomy and late tracheostomy vary, with early tracheostomy ranging from 48 hours to 8 days and late tracheostomy generally being greater than 10 days. Early tracheostomy confers no mortality benefit and results in no difference in pneumonia or ventilator duration in most studies, including the largest TracMan trial.[102] Based on evidence from recent large randomized trials, it is reasonable to wait at least 10 days to be certain that a patient has an ongoing need for mechanical ventilation before consideration of tracheostomy.[103]

Early tracheostomy may be effective in patients with a TBI, particularly when an airway is required but the patient does not have the need for mechanical ventilation. Tracheostomy may be performed via open or percutaneous method (see Atlas Figure 14). The technique used should be based on patient needs and clinician experience as multiple large studies in heterogeneous ICU populations have shown both methods to be safe and effective, with a possible morbidity benefit to percutaneous methods over open techniques. Percutaneous tracheostomy with flexible bronchoscopy guidance is recommended and is easily performed at the bedside in the ICU.[104]

COMPLICATIONS AND PITFALLS IN THE INTENSIVE CARE UNIT

Critical care complications interpose a substantial cost burden to the health care institution and society and worsen patient outcomes. There is increasing public awareness of this issue; preventable complications are often reportable to regulatory authorities, and many insurers, including Medicare, may refuse to pay hospital or physician charges in case of patients with certain preventable critical care complications. Health care consumers can readily obtain ICU complication rates for their health care institutions from a variety of media. The need to reduce preventable complications of critical care has resulted in guidelines released by professional and regulatory organizations and local hospitals.

Critical Illness Polyneuropathy and Myopathy

Prolonged neuromuscular weakness associated with critical illness was reported as early as the 1950s. Found to be associated with sepsis, hypotension, or multiple organ dysfunction, critical illness polyneuropathy (CIP) may prolong weaning from mechanical ventilation, delay return to ambulation, and significantly affect overall recovery from posttraumatic critical illness. The syndrome is characterized by the development of diffuse neurogenic muscle weakness over a several-week course of severe critical illness. The neurologic manifestations may include unexplained failure to wean from mechanical ventilation, decreased/absent deep tendon reflexes, tetraparesis, muscle atrophy, decreased fibrillations, compound muscle action potentials, and axonal damage on electrophysiologic testing. Nerve conduction velocities are near normal, and histologic evaluation of peripheral nerves has shown acute diffuse neurogenic atrophy in muscles and axonal degeneration in nerve tissue. CIP may be far more common than is currently recognized and may frequently affect ventilator weaning and recovery.[91] It should be considered as a cause of weaning failures or generalized weakness in the setting of critical illness. Electrophysiologic evaluation of muscle and nerve function is important for the diagnosis. Although not conclusive, available data suggest that the avoidance of long-term use of neuromuscular blocking agents (eg, pancuronium and vecuronium), particularly in combination with corticosteroids or aminoglycoside antibiotics, may be an important preventative measure. Recovery from CIP, although prolonged, may be nearly complete from a clinical standpoint. Follow-up electromyographic studies have shown changes with chronic neurogenic damage.

The Geriatric Trauma Patient in the Intensive Care Unit

Mortality in elderly trauma patients is significantly higher than that of their younger counterparts and has been associated with cardiovascular and septic complications.[105]

TABLE 57-14: Pharmacology of Antipsychotics Commonly Used in Intensive Care Unit (ICU) Delirium

						Adverse effects (incidence)				
Antipsychotic agent	Dosage form	Metabolism	Metabolizing enzyme	Equiv. dosages (approx) (mg)	Max dose (mg/d)	QT prolongation potential[a,b]	Sedation	Dopaminergic affinity/ extrapyramidal effects[b]	Anticholinergic effects	Orthostatic hypotension
Haloperidol (Haldol)[c]	Tab, IV injection	T½:: 21 h hepatic	CYP3A4, 2D6	2	20	Low	Low	High	Low	Low[d]
Quetiapine[e] (Seroquel)[c]	Tab	T½:: 6 h hepatic	CYP3A4	125	400	Moderate	Moderate	Low	Moderate	High
Risperidone (Risperdal)[c]	Tab, ODT tab, soln (1 mg/mL)	T½:: 3 h hepatic	CYP2D6, 3A4	1	4	Moderate	Low	High	Low	Moderate
Aripiprazole (Abilify)[c]	Tab, soln (5 mg/mL), IM injection	T½:: 75 h hepatic	CYP2D6, 3A4	5	10–30 mg	Low	Low	Low	Low	Low

[a]Low: 3–10 ms, medium: 10–15 ms, and high: >15 ms.
[b]Dose-related effect.
[c]Note the following FDA Black Box Warning applies to haloperidol and all atypical antipsychotics: "Increased mortality seen when used in elderly patients with dementia-related psychosis due to cardiovascular or infectious complications." The use of these agents for prevention of delirium in ICU patients has not been tested in high-quality trials.
[d]Increased with IV formulation.
[e]Caution: Bone marrow suppression; blood dyscrasias.

FDA, US Food and Drug Administration; IM, intramuscular; IV, intravenous; ODT, orally dissolving tablet; $T_{1/2}$, half-life.

Therefore, aggressive monitoring is warranted as it may help in diagnosing physiologic deterioration and in assessing the effectiveness of various therapies deployed in an attempt to improve outcomes. End-of-life and goals-of-care discussions are particularly important in this patient population.

Sepsis and subsequent multiple organ system failure cause most late deaths following trauma in the elderly. Urosepsis and pneumonia are common in elderly trauma patients, and what would be otherwise a relatively simple problem to treat in the young healthy individual may be the trigger to a cascade of events in the elderly patient, which may culminate with multiple organ dysfunction and death. For these reasons, aggressive and early treatment of these infections, initially with broad-spectrum antibiotics followed by culture-based de-escalation or adjustment of therapy, is a critical determinant of good outcome.

One of the most common causes of death in the geriatric trauma population is pneumonia following blunt chest trauma and rib fractures. Due to decreased pulmonary reserve and associated comorbidities, the elderly trauma patient is generally more susceptible to the development of pneumonia due to an inability to effectively clear secretions and take deep breaths. Two aspects in the early initial care in the ICU in these patients are important: avoidance of fluid overload and adequate analgesia. To this end, patient-controlled narcotic analgesia and/or epidural administration of opiate analgesics or local anesthetics (rib blocks or chest wall pain catheters) may be helpful in appropriately selected trauma patients. Another option is surgical stabilization of rib fractures, but indications are not agreed upon, and data to date have not been able to demonstrate a consistent outcomes benefit.[93]

Geriatric patients are also at increased risk for thromboembolic complications following trauma. Patients in the high-risk group for thromboembolic events (TBI, spinal cord injury, complex pelvic fractures, bilateral lower extremity fractures, prolonged immobilization, or a previous history of deep venous thrombosis or pulmonary embolism) should receive pharmacologic prophylaxis with low-molecular-weight heparin as soon as possible.

Reversal of Oral Anticoagulation in Acute Hemorrhage

Trauma centers in the United States have recently experienced an epidemic of elderly falls. The main clinical trials on anticoagulant therapies rarely include the frail elderly; however, many such patients arrive after taking these drugs. Common agents such as aspirin, clopidogrel, and warfarin have been joined by direct oral anticoagulants (DOACs) such as dabigatran, rivaroxaban, and apixaban. DOACs may require specific antidotes, if available, or use of concentrated clotting factors such a prothrombin complex concentrates (PCCs). Even patients with mild to moderate TBI are at a much higher risk of developing fatal intracranial hemorrhage due to the use of these drugs and because of "increased space" for hematoma expansion due to brain atrophy. Rapidly obtaining a brain CT scan and quickly assessing coagulation parameters for reversal of anticoagulation are critical in early management. Patients with active hemorrhage should undergo rapid reversal with PCC and specific DOAC reversal agents, including idarucizumab for dabigatran and andexanet alfa (approved by FDA in May 2018) for rivaroxaban and apixaban. In a life-threatening or critical site bleed or in situations in which bleeding cannot be controlled, reversal of oral anticoagulants is required with national evidence-based recommendations from the 2017 ACC Expert Consensus Decision Pathway on Management of Bleeding in Patients on Oral Anticoagulants (Table 57-15).[106]

TABLE 57-15: Recommendations for Reversal of Oral Anticoagulants

Vitamin K antagonist (warfarin)	Direct thrombin inhibitor (dabigatran)	Factor Xa inhibitor (rivaroxaban, apixaban, edoxaban)

• Administer 4-factor PCC IV based on INR
  • INR 2–4, 25 units/kg
  • INR 4–6, 35 units/kg
  • INR > 6, 50 units/kg
• Or 4-factor PCC low fixed-dose option
  • 1000 units for any major bleed
  • 1500 units for intracranial hemorrhage
• If 4-factor PCC not available, use plasma 10–15 mL/kg
	• Administer 5 g idarucizumab IV
	• If idarucizumab is not available, administer 4-factor PCC or aPCC 50 units/kg IV
	• Consider activated charcoal for known recent ingestion (within 2–4 h)
		• Administer 4-factor PCC IV, 50 units/kg IV
		• If 4-factor PCC not available, consider aPCC 50 units/kg IV
		• Andexanet alfa (FDA approved for reversal of apixaban or rivaroxaban for life-threatening bleeding), bolus (400 or 800 mg) and infusion (4 vs 8 mg/min for up to 120 min)
		• Consider activated charcoal for known recent ingestion (within 2–4 h)

aPCC, activated prothrombin complex concentrate; FDA, US Food and Drug Administration; INR, international normalized ratio; IV, intravenous; PCC, prothrombin complex concentrate.

Missed Injuries

The initial evaluation of the trauma patient centers on recognizing abnormal physiology and the pattern of injury. Missed injuries are the most common cause of preventable death; however, the true incidence of missed injury is difficult to determine. Surgical intensivists caring for trauma patients must recognize potential injuries likely to occur given a particular mechanism of injury. The challenge becomes the rapid identification of occult injuries before the clinical condition of the patient deteriorates. Missed injury is a major pitfall in the care of trauma, and an unexpected deterioration in the condition of the patient in the ICU should prompt a reexamination for possible missed injury, among other causes. Trauma patients in the ICU should undergo a tertiary survey and careful review of imaging results to reduce the risk of missed injury.[107]

Postresuscitation Hypotension in the Intensive Care Unit

Any patient developing hypotension postresuscitation is bleeding until proven otherwise, and aggressive investigation of the etiology is mandatory. Such episodes should prompt a reevaluation of the patient's workup and raise the specter of a missed intra-abdominal injury or solid organ injury rebleeding due to overresuscitation and dislodgment of the initial hemostatic clot. It is a common pitfall to ignore such an episode and assume that resuscitation has been completed and hypotension is due to other causes such as TBI or bleeding due to long bone fractures.

There are certain injuries that may require ongoing resuscitation with blood products, most prominent among them a vertical shear-type posterior element pelvic fracture. However, in the absence of any such known injury, a diligent attempt must be made to exclude a missed injury in the abdomen or perhaps an injury whose magnitude was underestimated.

The traditional end points of resuscitation include an adequate urinary output, the trend of the base deficit on the arterial blood gas, and normalization of arterial lactate levels. There are situations in which the urinary output may be misleading. The intoxicated patient will have good urinary output even in the face of hypovolemia, because alcohol inhibits the release of antidiuretic hormone from the posterior pituitary; in addition, it is hypertonic and leads to peripheral arterial vasodilation. Another situation in which the urinary output will be misleadingly elevated is in the setting of hyperglycemia. Whether the patient is a diabetic or he or she has received high-dose steroids for a spinal cord injury, the resultant hyperglycemia will cause a misleadingly comforting urinary output. A serum blood sugar over approximately 180 to 190 mg/dL results in glycosuria, and this pitfall must be recognized.

Similarly, the base deficit can also be misinterpreted. The etiology of metabolic acidosis in the injured patient is, until proven otherwise, due to hypoperfusion from hemorrhagic shock; it must be understood, however, that the base deficit can be due to ketosis, non–anion gap acidosis including hyperchloremia due to excess saline infusion, renal dysfunction, and sepsis.

SYSTEMIC INFLAMMATORY RESPONSE SYNDROME, SEPSIS, AND SEPTIC SHOCK

Injury leads to the activation of the inflammatory response, which may be aggravated by the severity of shock, degree of tissue injury, and secondary insults. Severely injured patients commonly develop systemic inflammatory response syndrome (SIRS). SIRS is defined by manifestation of two or more of the following conditions: (1) temperature greater than 38°C or less than 36°C, (2) heart rate greater than 90 bpm, (3) respiratory rate greater than 20 breaths/min or $Paco_2$ less than 32 mm Hg, and (4) white blood cell count greater than 12,000/mm^3, less than 4000/mm^3, or greater than 10% immature forms. The treatment of SIRS is supportive, and the condition resolves over time.

Sepsis was previously defined as the presence of a presumed infection in a patient with SIRS. Severe sepsis included sepsis and organ failure, while septic shock was previously defined as sepsis-induced hypotension and hypoperfusion, refractory to volume replacement and requiring vasopressors. The new Sepsis-3 definitions include only sepsis and septic shock[108] and are listed in Fig. 57-13. The 2016 Surviving Sepsis Campaign Guidelines use the new sepsis definitions.[109]

The source of sepsis in the injured patient relates to the type of injuries. It is extremely rare for a patient to have septic shock early, unless there is an obvious infection, such as an aspiration pneumonia or perforated viscus. The patient who has leukocytosis with bandemia, fever, and clinical deterioration must be investigated closely for a source of infection. The diagnosis of an infection following major trauma is the biggest pitfall since the cardinal signs of infection such as fever, leukocytosis, and hyperdynamic hemodynamic state can be, and frequently are, the result of the inflammatory cascade in response to tissue trauma. The pitfall lies in the differentiation between sepsis and SIRS. The consequences of liberal use of antibiotics to broadly cover for presumptive sepsis are real, including drug resistance, antibiotic-related colitis, and fungemia. The consequences of not treating a patient with fever, hyperdynamic state, and signs and symptoms of infection, in the absence of positive cultures or a clear source, are equally daunting, as the patient may indeed be harboring an infection, but the yield of blood cultures and the other surveillance tests are poor.

Unfortunately, there is no reliable method to distinguish between SIRS and sepsis until a definitive infectious source is identified. The usual sources of infection in the ICU are the lungs, indwelling vascular catheters, the urinary tract, and wounds. Each of these sites must be examined meticulously for infection.

The optimal management of sepsis is compliance with the Surviving Sepsis Campaign bundle (Table 57-16) to improve

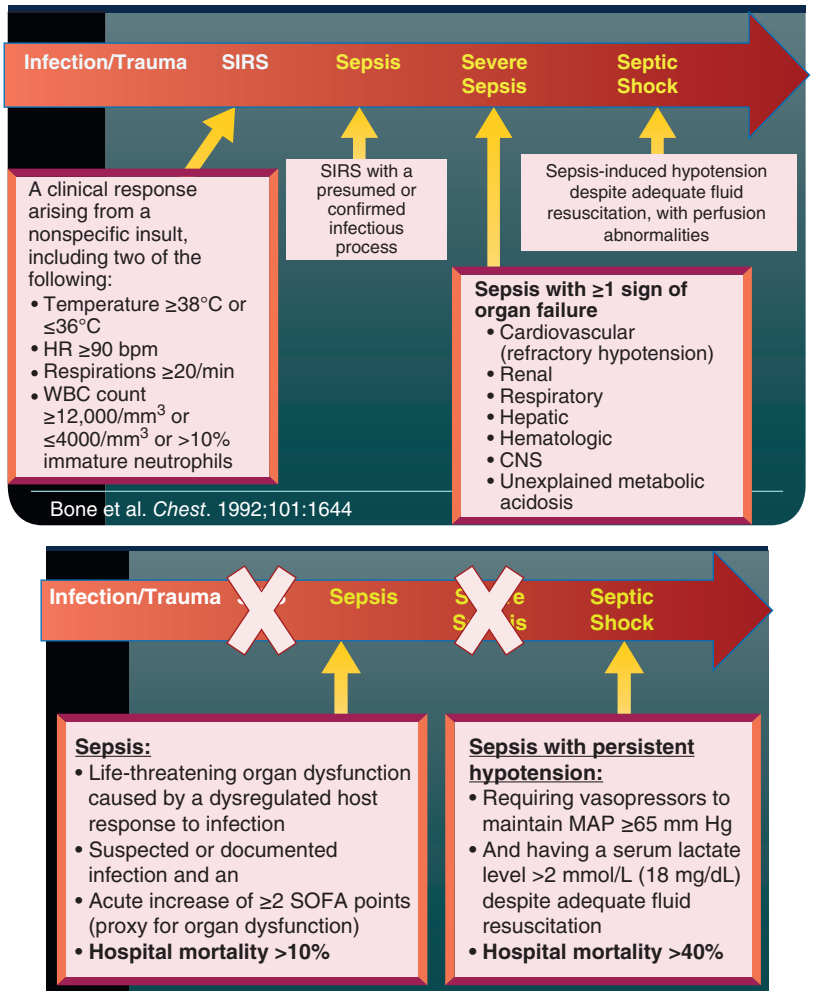


FIGURE 57-13 Sepsis-3 new definitions (2016) compared to prior sepsis definitions. CNS, central nervous system; HR, heart rate; MAP, mean arterial pressure; SIRS, systemic inflammatory response syndrome; SOFA, Sequential Organ Failure Assessment; WBC, white blood cell.

TABLE 57-16: Hour-1 Surviving Sepsis Campaign (SSC) Bundle

- Measure lactate level. Remeasure if initial lactate is >2 mmol/L.
- Obtain blood cultures prior to administration of antibiotics.
- Administer broad-spectrum antibiotics.
- Begin rapid administration of 30ml/kg crystalloid for hypotension or lactate ≥4 mmol/L.
- Apply vasopressors if patient is hypotensive during or after fluid resuscitation to maintain MAP ≥65 mm Hg.

* *"Time zero" or "time of presentation" is defined as the time of triage in the Emergency Department or, if presenting from another care venue, from the earliest chart annotation consistent with all elements of sepsis (formerly severe sepsis) or septic shock ascertained through chart review.*

Note. The most important change in the revision of the SSC bundles is that the 3-hour and 6-hour bundles have been combined into a single "hour-1 bundle" with the explicit intention of beginning resuscitation and management immediately.

MAP, mean arterial pressure.

Source: Reproduced with permission from Levy MM, Evans LE, Rhodes A. The Surviving Sepsis Campaign Bundle: 2018 update. *Crit Care Med.* 2018;46(6):997-1000.

outcomes.[15,110] Lower mortality was observed in high (29.0%) versus low (38.6%) resuscitation bundle compliance sites (P <.001) and between high (33.4%) and low (32.3%) management bundle compliance sites (P = .039).[111] Additional evidence-based recommendations from the recent Surviving Sepsis Campaign Guidelines differ from the prior guidelines (Table 57-17) and include more recommendations labeled as "Strong" (Table 57-18).

ETHICAL ISSUES IN THE INTENSIVE CARE UNIT

Ethics may be regarded, in part, as a series of societal formulations predicated on moral values designed to produce a theoretical "optimal good." With the ability of modern ICUs to maintain physiologic support and homeostasis well beyond the bounds of reasonable sentient existence, physicians are increasingly called on to participate in sometimes difficult live-or-die, withdraw/withhold support decisions. While ICU ethics also include other considerations, such as consent

TABLE 57-17: Surviving Sepsis Campaign Guideline Changes Comparing 2012 and 2016 Recommendations

	2012	2016
Sepsis definition	Systemic manifestation of infection + suspected infection; severe sepsis: sepsis + organ dysfunction.	Life-threatening organ dysfunction caused by dysregulated response to infection. No severe sepsis definition.
Initial resuscitation	At least 30 mL/kg in the first 3 hours; crystalloid fluid (no specific recommendation for fluid type). Albumin if patients require substantial fluids.	
	Early goal-directed therapy protocolized care including CVP, $ScvO_2$.	Use dynamic resuscitation markers (passive leg raise, TTE). Target MAP 65 mm Hg.
	Normalize lactate.	Reassess hemodynamic status to guide resuscitation. Normalize lactate.
Vasopressors	Target MAP 65 mm Hg.	
	Norepinephrine vasopressor of choice; epinephrine if not at target MAP or vasopressin to reduce norepinephrine requirement. Avoid dopamine in most patients.	
Steroids	Only indicated in septic shock refractory to adequate fluids and vasopressors.	
Antibiotics	Administration of effective IV antimicrobials within the first hour of recognition of septic shock and severe sepsis without septic shock.	We recommend that administration of IV antimicrobials be initiated as soon as possible after recognition and within 1 hour for both sepsis and septic shock.
	Initial empiric anti-infective therapy of one or more drugs that have activity against all likely pathogens. Combination empirical therapy for neutropenic patients with severe sepsis and for patients with difficult-to-treat, multidrug-resistant bacterial pathogens such as *Acinetobacter* and *Pseudomonas* spp.	Initial IV broad-spectrum antibiotics to cover all potential pathogens. The addition of a second gram-negative agent to the empiric regimen is recommended for critically ill septic patients at high risk of infection with multidrug-resistant pathogens (eg, *Pseudomonas, Acinetobacter*) to increase the probability of at least one active agent being administered.
	Antimicrobial regimen should be reassessed daily for potential deescalation. Use of low procalcitonin levels or similar biomarkers to assist the clinician in the discontinuation of empiric antibiotics in patients who initially appeared septic but have no subsequent evidence of infection.	May use procalcitonin to guide deescalation of antibiotic therapy.
Source control	Achieve within 12 hours, if feasible.	Achieve as soon as medically and logically feasible.

CVP, central venous pressure; IV, intravenous; MAP, mean arterial pressure; $ScvO_2$, central venous oxygen saturation; TTE, transthoracic echocardiography.

issues and donor organ procurement, this section focuses on the withdrawal/withholding of physiologic support including the application of do not resuscitate (DNR) orders in the surgical ICU.

Ethical Principles Applicable to Medical Decision Making

Ethical decision making should involve the careful application of established principles. The temptation to apply one's individual value system to the decision-making process is strong, but the personal values of the patient are paramount, and the personal judgments of both family members and physicians must be considered in the context of the ethical principles involved. With the possible exception of the principle of distributive justice, each of the following principles should be applied in any given decision-making process:

- Beneficence: The principle of "doing good" as applies to a particular patient, individual, or situation.
- Nonmaleficence: The principle of avoiding harm or wrongdoing, as applied to a particular situation or individual.
- Autonomy: Perhaps the most important operative principle in critical care ethics is the right of self-determination—the inherent right of individuals to make decisions regarding their own treatment options (or withholding thereof) and ultimately make decisions that will impact their survival.
- Full disclosure: The principle of accurate communication of information that will allow individuals (or their surrogates) to exercise autonomy or surrogate decision making.
- Social (distributive) justice: The principle whereby benefits to an individual, if associated with burdens to another individual or individuals, must be weighed in terms of the most "good" done to the society or group as a whole. This last principle typically involves decisions regarding the distribution of scarce resources to allow the "optimum" treatment of not a single individual, but a population of individuals (society). Such a principle may apply during wartime casualty triage or civilian mass-casualty triage.

TABLE 57-18: Strong Recommendations from the Surviving Sepsis Campaign 2016 Guidelines

- 30 mL/kg crystalloid fluid resuscitation within the first 3 hours
- Crystalloids as fluid of choice for initial resuscitation
- Against the use of hydroxyethyl starches for intravascular volume replacement
- Initial target mean arterial pressure of 65 mm Hg in septic shock requiring vasopressors
- Norepinephrine as first-line vasopressor
- Administer antibiotics within 1 hour of recognition
- Empiric broad-spectrum antimicrobial therapy to cover all likely pathogens
- Red blood cell transfusion only when hemoglobin <7 unless extenuating circumstances (myocardial infarction, severe hypoxemia, acute hemorrhage)
- Target tidal volume 6 mL/kg for ARDS, plateau pressure upper limit 30 cm H_2O
- Conservative fluid strategy in ARDS in patients without hypoperfusion
- Against the use of pulmonary artery catheter for patients with sepsis-induced ARDS
- Prone position for sepsis-induced ARDS with Pao_2/Fio_2 ratio <150
- Against use of β_2-agonists for patients with sepsis-induced ARDS without bronchospasm
- Against use of HFOV in adult patients with sepsis-induced ARDS
- Raise head of bed 30–45° in mechanically ventilated patients, spontaneous breathing trials, and a weaning protocol
- Blood glucose control via protocol targeting blood glucose <180 g/dL
- Pharmacologic VTE prophylaxis, unfractionated or low-molecular-weight heparin
- Stress ulcer prophylaxis for patients with risk factors for gastrointestinal bleeding
- Early enteral nutrition, against parenteral nutrition in the first 7 days
- Against use of omega-3 fatty acids as an immune supplement
- Incorporate goals of care into treatment planning using palliative care principles where appropriate

ARDS, acute respiratory distress syndrome; HFOV, high-frequency oscillatory ventilation; VTE, venous thromboembolism.

Withdraw/Withhold Support Decisions

Modern ICUs have developed the increasing technical capability to support homeostasis, even in the face of what would otherwise be overwhelming disease. Increasingly in the trauma population of ICU patients, patient demise is the result of a decision to withdraw or withhold life support. The consequences and irreversibility of such withdraw/withhold decisions in the ICU mandate that they be considered very carefully and predicated on the careful application of ethical and legal principles.

The importance and impact of withdraw/withhold decisions are considerable. In a review of such decision, Smedira et al[112] found that in a mixed population of surgical and medical ICU patients, support was withheld in 1% and withdrawn in 5%. The resultant deaths accounted for 45% of all ICU deaths in the two institutions under study. The general setting under which withdraw/withhold decisions are made is poor prognosis, which would include a low chance of survival and high likelihood of poor cognitive function. Specific criteria that may allow withdrawal/withholding include the following:

- Provision of further treatment is considered medically futile with respect to achieving well-defined therapeutic goals.
- The patient with decision-making capacity (DMC), in exercising his or her autonomy, chooses to have support withdrawn/withheld.
- A legal surrogate decision maker (legal guardian or durable power of attorney) chooses to have support withdrawn/withheld under medically appropriate circumstances.
- The decision is made based on a prior written medical directive stipulating circumstances under which support may be withdrawn/withheld.
- The physician, acting as a surrogate decision maker in conjunction with next of kin, other consultants, and possibly an ethics committee, elects to withdraw/withhold support. The decision should be based on the following considerations:
  - Information regarding the patient's wishes: What the patient would be likely to decide if he or she had DMC. This may be obtained from family, friends, or other providers with a history of relevant contact with the patient.
  - The benefits/burdens test: The benefits of continued treatment are outweighed by the burdens to the patient of such treatment in accomplishing the defined therapeutic goals.
  - Substituted judgment: The "reasonable person test." In the absence of other available information regarding a patient's wishes, a withdraw/withhold decision should be regarded as one likely to be made by any "reasonable person."
  - Best interests test: The decision to withdraw/withhold must be medically appropriate and should not significantly diminish opportunity for recovery within the bounds of benefits/burdens.

Futility

The concept of futility refers to the inability to accomplish a therapeutic goal through medical interventions regardless of the duration of such interventions or the frequency with

which they are repeated. The concept of futility is central to initial withdraw/withhold support decisions. Determination of futility requires two elements: (1) the establishment of an agreed-upon therapeutic goal and (2) determination of the probability, given the application of medical therapy, of reaching these therapeutic goals. Therapeutic goals should be outlined as specifically as possible and may incorporate elements of both physical and cognitive functions. Patients with DMC must be allowed to define their own therapeutic goals, regardless of how "unreasonable" or stringent these goals may seem. The determination of the probability of medical therapy in achieving those goals should be left to the team of treating physicians. Once a given treatment or treatments are deemed to be futile, based on established therapeutic goals, the physician is under no legal or ethical obligation to provide such treatment. Current writing in medical ethics, public policy, and more recently by case law has supported this perspective, although some ethicists do not support this medical prerogative. Individual circumstances, however, particularly in regard to family considerations and consistent with the principle of beneficence, may occasionally be indications for short-term delivery of what otherwise would be considered medically futile care.

During the early ICU phase of care, determination of futility often is achieved in patients with severe nonsurvivable brain injuries (transaxial gunshot injuries or open skull fractures with loss of brain tissue and GCS of 3).

Patients with significant comorbidities, particularly those with end-stage liver or renal disease, and those with malignancies are not candidates for extreme resuscitation efforts or massive transfusion. To the same extent, blunt trauma victims with significant intra-abdominal bleeding and associated nonsurvivable brain injury, greater than 60% total body surface area burns, and cervical spinal cord transactions should not be aggressively resuscitated. Even if some of these patients survive, they will do so with a very poor quality of life, and it is the trauma surgeon's responsibility to provide the family with such information.

Patients who received massive blood transfusions in the operating room without complete hemostasis usually present to the ICU as hypothermic, acidotic, and coagulopathic (see Chapter 13). Although difficult to estimate, the rapidity of bleeding and transfusion requirements greater than two blood volumes have traditionally been used as markers of irreversibility, and most surgeons would agree that in these circumstances, if bleeding is nonmechanical and hypothermia and coagulopathy have not improved over a period of 6 to 8 hours, further resuscitation efforts are probably futile and will serve only to consume precious resources.[113]

Late in the course of ICU care, defining futility in patients with neurologic injury is difficult, as it is almost impossible to predict those who will not achieve a meaningful recovery. Age and comorbidities should be considered when presenting the facts to family members, and they may facilitate the family's decision to withdraw care.

In patients with multiple organ dysfunction, determination of futility is often achieved in those with three or more organ failures as mortality in these circumstances approaches 100%.

Decision-Making Capacity and Autonomy

DMC, which has different implications than the legal term *competent*, requires the following:

- The patient must be able to comprehend and communicate information relevant to making a decision.
- The patient must be able to comprehend his or her alternatives and the benefits and burdens (risks) associated with each.
- The patient must be able to reason and deliberate about these alternatives against a background of stable personal values.

The determination of DMC may generally be made by the primary physician or team, but occasionally, particularly in the presence of underlying psychiatric illness, psychiatric consultation may be of benefit in making this determination. The exercise of autonomy is best accomplished by allowing any patient with DMC to participate in withdraw/withhold or DNR decisions. In many cases, a patient's specific wishes may not be known and legal surrogates or medical directives may be unavailable. With careful adjustment (lightening to the extent possible) of any conscious sedation and directed efforts made in communicating the alternatives (full disclosure), many patients can make decisions or at least provide valuable information allowing surrogates to do so.

The written medical directive provides an alternative means by which a patient may exercise autonomy. Medical directives may be structured in a variety of ways—depending on known medical conditions, age of the patient, and so forth—but they typically contain language stipulating preferences (or not) for life-sustaining treatment as a function of expected physical and cognitive outcomes.[114]

Decision Making in the Absence of Decision-Making Capacity

In situations where the patient does not have DMC and no specific medical directive is available, a surrogate decision-making process is used. Participants in the surrogate decision-making process may include family members, friends, other health care providers, clergy, and members of the institutional ethics committee. Family members invested with durable power of attorney may make clinically appropriate decisions independently on behalf of the patient. Surrogate decision making should involve close collaboration and extensive documented communication between health care providers and the family to the extent possible. The specific role of the family, however, in the absence of durable power, is not to actually make withdraw/withhold decisions but to provide information allowing providers to best formulate decisions consistent with what the patient's desires would be. This information

may include knowledge of the patient's values, goals, religious or philosophical beliefs, or previously expressed wishes with respect to medical care. The importance of this information should not be underestimated; in the absence of durable power of attorney, family members have no recognized legal authority to dictate care or to make decisions that are medically or ethically inappropriate. Health care professionals also have a primary responsibility to act in what they perceive to be the best interests of the patient, which may occasionally be in conflict with wishes expressed by the family or, on rare occasions, the wishes expressed by a durable power agent.

In the absence of any information pertaining to the presumptive wishes of the patient, substituted judgment may be applied based on several considerations:

- What would a "reasonable" person want under similar circumstances? Physicians should rarely be making this judgment independently, and the involvement of colleagues or a formal ethics committee consultation may be appropriate.
- What would the likely benefits of continued treatment be (treatment outcome), weighed against the burdens imposed by both the treatment and the treatment outcome? The anticipated degree of functional recovery and resultant quality of life are important factors in this consideration. Therapeutic intervention designed to prolong life in a setting in which the quality of that life—because of severity of pain, lack of cognitive function, and so forth—is regarded as being excessively burdensome to an individual may not be medically appropriate.

In many cases, the outcome from a critical illness and specific degree of disability, at a given point in time, cannot be predicted with any degree of certainty. The best interests test prevents premature withdraw/withhold decisions from being made that might deprive a patient of an opportunity for satisfactory recovery. Additional professional consultation, including the institutional ethics committee, may help resolve the more difficult cases.

Conflict Resolution

With good ongoing communication, full disclosure of relevant information and prognosis, and sensitivity to patient/family dynamics, withdraw/withhold support decisions can generally be made smoothly. The liberal use of consultants, particularly those involving the neurosciences, may be a valuable adjunct under such circumstances. Ethics consultation services have also been shown to affect management outcome when they are made available. Conflicts may arise, however, over issues of futility, therapeutic goals, and the conduct of surrogate decision making. These conflicts are often based on misunderstanding or mistrust and exacerbated by the lack of good communication.

In situations in which conflict between care providers and family members appears to be irreconcilable, even with ethics committee and institutional risk management/legal consultation, a number of alternatives should be offered to the family. These alternatives may include the following: (1) procurement of additional intramural or extramural medical, religious, or ethical consultations; (2) transferring the care of the patient to another provider within the institution; (3) transfer of the patient to another institution, under the care of another provider; and (4) procurement of a court order mandating a course of treatment.

Do Not Resuscitate Orders

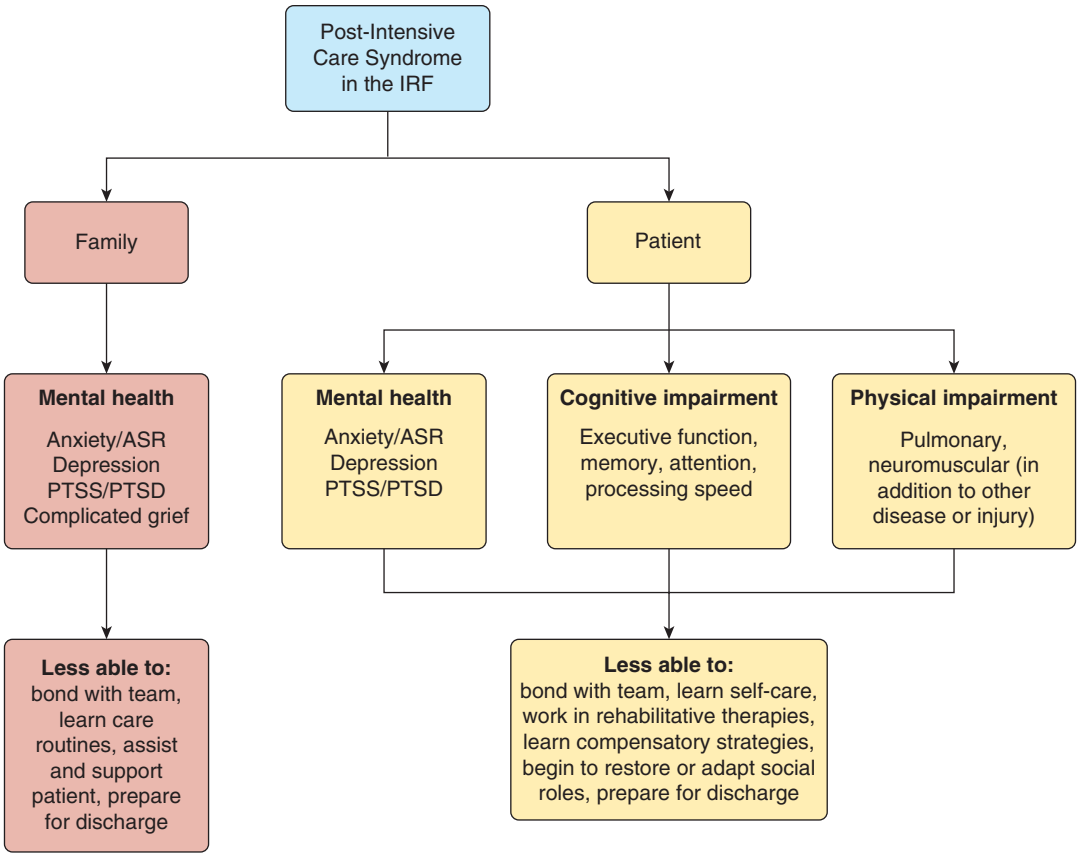
The institution of DNR medical directives has been primarily based on the desire to avoid the indignity of futile cardiopulmonary resuscitation in the setting of inexorable progression of underlying known disease processes. Such orders are frequently applied to patients with long-standing terminal disease (eg, cancer, end-stage AIDS, and irreversible multiple organ failure). DNR orders are implicitly coupled to futility judgments about the benefit of cardiopulmonary resuscitation to achieve prospectively defined therapeutic goals. Under such circumstances, these orders are entirely appropriate and desirable in the variety of clinical disease states. The intensity of critical care interventions with mechanical ventilators and the use of complex drug regimens, including vasoactive drugs, may lead to unexpected (iatrogenic) complications unrelated directly to the underlying disease process for which a DNR order may have been originally placed. In addition, the critical illness being treated in the surgical ICU, particularly for trauma patients, results from a discrete event (surgery or injury) as opposed to complications or exacerbations of chronic disease, as is often the case for medical patients. The presumed reversibility of the physiologic sequelae constitutes a basis for ICU treatment of the trauma patient. As such, DNR for the trauma patient should be applied very carefully to expected complications, or physiologic changes due to inexorable progression of known underlying disease, as opposed to unexpected, very reversible iatrogenic complications.

DNR orders for trauma patients may be more appropriately linked to withdraw/withhold support conditions than more generally to patients with a poor but potentially reversible prognosis. The variability with which DNR orders are interpreted and the "message" such orders send to providers also raise concerns about their applicability for trauma patients. In addition to the potential for the inappropriate application of DNR orders in an ICU, application may result in less aggressive care on the part of ancillary providers.[115,116]

DNR orders constitute the prospective application of a specific "withhold support" decision and should be made with the same care and consideration, as described in the earlier section, as any decision involving the limitation of critical care.

POST–INTENSIVE CARE SYNDROME (PICS)

Millions of trauma survivors experience de novo suffering and disability called the post–intensive care syndrome (PICS). Patients with PICS are robbed of their normal cognitive,



This model, adapted from a model developed in 2010 by the Long-Term Consequences Task Force of the Society of Critical Care Medicine (SCCM), shows the physical, cognitive, and mental health problems affecting inpatient rehabilitation in patients PICS.

FIGURE 57-14 Post–intensive care syndrome (PICS). ASR, acute stress reaction; IRF, inpatient rehabilitation facility; PTSD, posttraumatic stress disorder; PTSS, posttraumatic stress symptoms.

emotional, and physical capacity and cannot resume their previous life (Fig. 57-14). Muscle weakness, memory problems, depression, insomnia, physical pain, and nightmares are just a few examples of the problems that patients may experience following critical illness. Symptoms such as these, which affect emotional, physical, and cognitive health, are now being recognized as PICS. Efforts to educate health care providers, patients, and families about PICS are underway. Some studies have documented that a multidisciplinary, complex, 5-week, peer-supported rehabilitation program, including pharmacy, physiotherapy, nursing, medical, and psychology input, was associated with improved patient outcomes.[117] Additional studies are required to identify optimal treatment for ICU survivors with PICS, but it is our responsibility as intensivists to first identify the symptoms of PICS in our ICU survivors.

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Cardiovascular Failure

Nori L. Bradley • Bryan A. Cotton

KEY POINTS

- Cardiac output (heart rate × stroke volume) is determined by rate, preload, afterload, and contractility.
- Ventricular filling determines left ventricular end-diastolic volume, which in turn determines the force of ventricular contraction.
- Hemodynamic transesophageal echocardiography (TEE) is performed with a miniaturized two-dimensional monoplane disposable TEE probe that can be left in place for up to 72 hours.
- Right heart failure is characterized by increased right ventricular (RV) afterload (increased pulmonary resistance), which decreases the ability of the RV to pump blood forward to the left heart, thus decreasing left ventricular preload.
- In valvular aortic stenosis, symptoms of left heart failure include dyspnea on exertion, angina, lightheadedness, and syncope.
- Management of myocardial dysfunction includes vasopressors (eg, norepinephrine, vasopressin, phenylephrine), inotropes (eg, epinephrine, dobutamine, milrinone, dopamine, digoxin), and intra-aortic balloon pump counterpulsation.
- A low mixed venous oxygen saturation occurs with decreased oxygen delivery (decreased hemoglobin, oxygen saturation, or flow) or increased oxygen demand (hyperthermia, pain, seizure, or shivering).
- In the management of acute heart failure, nitroprusside reduces afterload by acting on arteriolar smooth muscle and has a rapid onset of action, and its effects cease within minutes of stopping the infusion.
- In diastolic dysfunction, β-adrenergic blockade or calcium channel blockade reduces heart rate and increases left ventricular filling, which reduce mortality.
- Cardiac death or a myocardial infarction occurs in 1% to 5% of unselected patients undergoing noncardiac surgery, and this concern is the most common indication for a preoperative cardiac evaluation.

INTRODUCTION

Cardiovascular failure is deterioration of cardiac function or vascular tone that results in impaired end-organ perfusion. In the acute phase of trauma, most shock is due to hemorrhage. Hemorrhagic shock causes decreased preload and thus decreased heart filling volumes, which are compensated by tachycardia. Without appropriate volume resuscitation and control of bleeding, hemorrhagic shock may progress to hypotension and end-organ hypoperfusion. In contrast, cardiogenic shock is due to impaired myocardial contractility with elevated cardiac filling pressures and low cardiac output that progresses to end-organ hypoperfusion.[1] Unlike hemorrhagic shock, cardiogenic shock often does not respond simply to volume/blood product resuscitation and control of hemorrhage, and may often worsen as the patient is "driven off" the Starling curve.[2,3] Cardiogenic shock solely from impaired contractility is most often due to an acute myocardial infarction or acute-on-chronic heart failure.[4] Therapy to support the failing cardiovascular system is directed at the etiology of the shock state and includes fluid management (optimize preload) as well as pharmacologic modulation of vascular tone (optimize afterload), contractility (with inotropes), and heart rate (with chronotropes). This chapter will explain the physiologic components of cardiovascular failure and the evaluation, treatment, and monitoring of patients with cardiovascular failure (Fig. 58-1).[5]

DETERMINANTS OF CARDIAC OUTPUT

Cardiac output (CO) is defined as the quantity of blood ejected into the aorta by the heart each minute and is calculated as heart rate multiplied by stroke volume (CO = heart

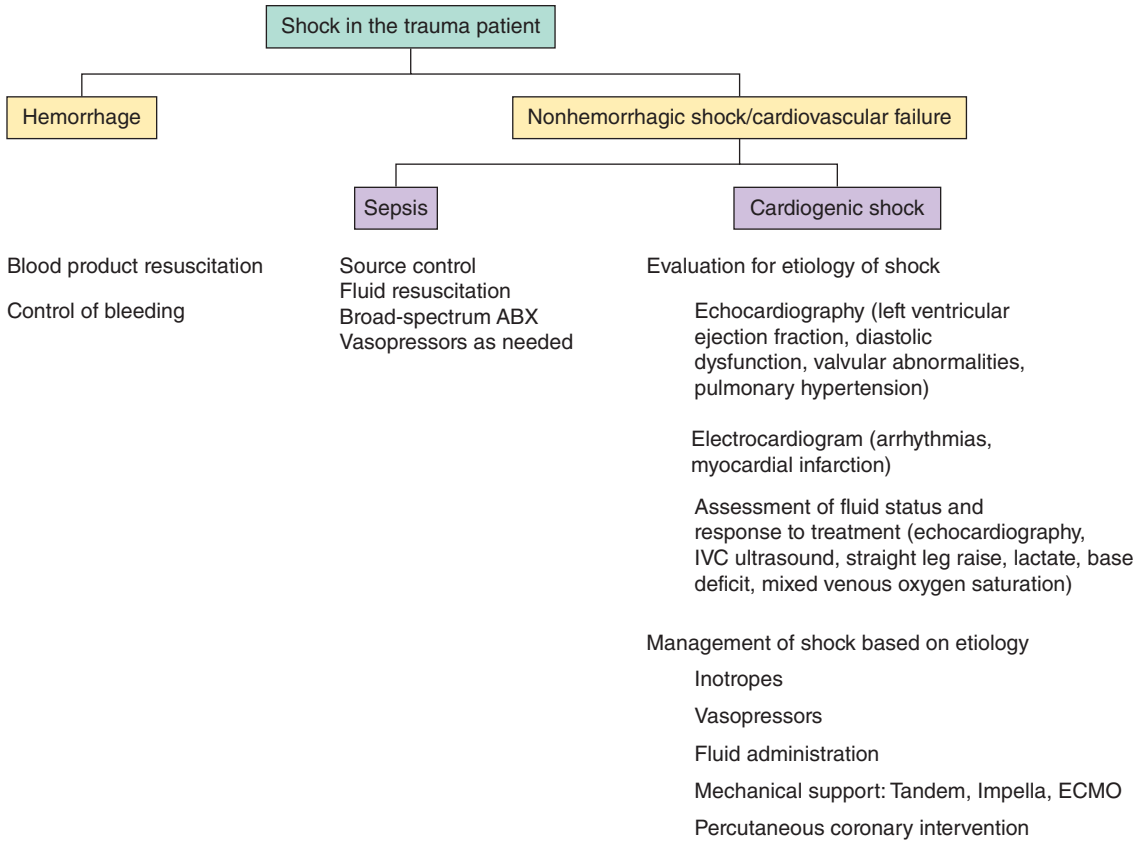

FIGURE 58-1 Algorithm for the approach to the patient in the surgical intensive care unit (SICU) with cardiovascular failure. ABX, antibiotics; ECMO, extracorporeal membrane oxygenation; IVC, inferior vena cava.

rate × stroke volume). This is the quantity of blood that flows through the circulation and is responsible for oxygen and nutrient transport to the tissues. The primary determinants of CO are listed in the box below. Accordingly, the majority of therapies for augmenting CO aim to (1) control heart rate, (2) restore filling pressures, and (3) optimize the ability of heart to contract.[5]

Determinants of Cardiac Output (CO)

CO = heart rate × stroke volume

- Preload
- Afterload
- Contractility

Heart rate = number of beats per minute
Preload = ventricular filling
Afterload = the forces opposing ventricular contraction
Contractility = force with which myocardium contracts

The average CO for adults is approximated as 5 L/min. However, CO varies with activity and is influenced by level of metabolism, exercise state, age, size of the individual, and other factors. CO in women is generally stated as being 10% to 20% lower than in men. CO increases in proportion to increasing body surface area. Therefore, to standardize CO measurements between individuals, cardiac index (defined as CO divided by body surface area in meters squared) is employed.[6]

Preload

Preload is the degree of myocardial stretch prior to contraction and most commonly is volume dependent. The Frank-Starling relationship shows that as cardiac sarcomere length increases, the force of contraction increases. However, when the sarcomere is stretched beyond its maximal length (overfilling), the force of contraction decreases. The Frank-Starling relationship provides a paradigm by which the cardiovascular system and its derangements can be approached. Ventricular filling determines left ventricular end-diastolic volume (LVEDV), which in turn determines the force of ventricular contraction. Factors influencing ventricular filling include intravascular volume status, intrathoracic pressure, intra-abdominal pressure, ventricular compliance, valvular disease, atrial kick, pericardial compliance, and ventricular wall thickness[5] (Table 58-1).

LVEDV can be directly measured with echocardiography. However, it is most frequently estimated via the left ventricular end-diastolic pressure (LVEDP). Assuming unaltered ventricular compliance, LVEDP theoretically approximates

TABLE 58-1: Factors Influencing Ventricular Filling

Factor	Clinical example	Effect on preload	Effect on CVP	Effect on PAWP
Intravascular volume	Hemorrhage	↓	↓	↓
	Sepsis[a]	↓←→	↓←→	↓←→
Intrathoracic pressure	Positive-pressure ventilation	↓	↓	←→ or ↑
	Tension PTX			
Intra-abdominal pressure	Abdominal compartment syndrome	↓	↑	↑
	Pregnancy			
Ventricular compliance	Diastolic dysfunction (↓)	↓	↑	↑
Ventricular hypertrophy	HTN → LVH	↓	←→ or ↑	←→↑ or ↓
	PHTN → RVH	↓	↑	
Valvular disease	MR	↓	←→ or ↑	↑
	AS?	↑		
Atrial kick	Atrial fibrillation	←→ or ↓	↑ (?)	←→ or ↓
Pericardial compliance	Tamponade	↓	↑	↓
	Constrictive pericarditis		↑	

[a]Sepsis can present as intravascular hypovolemia with decreased preload, CVP, and PAWP. However, once resuscitated, if septic cardiomyopathy ensues, ventricular volume may increase but with accompanying ventricular dilation and increased compliance that decreases filling pressures.

AS, aortic stenosis; CVP, central venous pressure; HTN, hypertension; LVH, left ventricular hypertrophy; MR, mitral regurgitation; PAWP, pulmonary artery wedge pressure; PHTN, pulmonary hypertension; PTX, pneumothorax; RVH, right ventricular hypertrophy.

the LVEDV. Traditionally, a pulmonary artery catheter (PAC) was used to measure the pulmonary arterial wedge pressure (PAWP), which approximates LVEDP. Alternatively, measuring central venous pressure (CVP) with a central venous catheter at the cavoatrial junction reflects right ventricle end-diastolic pressure (RVEDP). Again, assuming unaltered ventricular compliance, RVEDP theoretically approximates right ventricle end-diastolic volume (RVEDV). With normal pulmonary compliance and cardiac valves, RVEDV approximates LVEDV. Unfortunately, in the setting of critically ill patients, factors such as myocardial ischemia, heart failure, and myocardial edema can decrease ventricular compliance, rendering measurement of PAWP and/or CVP as surrogates for end-diastolic volume unreliable. In addition, valvulopathy and poor pulmonary compliance can influence right- and left-sided filling volumes differentially. In any of these situations, normal or elevated PAWP and/or CVP does not rule out inadequate preload as a cause of low CO. Therefore, multiple measurements of cardiovascular function, including the pulmonary artery pressure, CVP, and inferior vena cava filling, straight leg raise, response to fluid challenge, and the clinical scenario should be integrated to gauge preload when caring for these complex patients.[7] See later section, "Evaluation of Volume Status and Cardiac Function."

Afterload

Afterload is the force that opposes ventricular contraction. It is primarily dependent on vascular tone, arterial compliance, and ventricular transmural systolic pressure. Resistance is the primary determinant of vascular tone, and afterload is often estimated using systemic vascular resistance (SVR). SVR is calculated using the hemodynamic equivalent of Ohm's law (see the following box).[7]

Determinants of Systemic Vascular Resistance (SVR)

$$SVR = (MAP - CVP)/CO$$

MAP = mean arterial pressure
CVP = central venous pressure
CO = cardiac output

This equation provides an approximation of vascular impedance. However, there are several limitations in using SVR to estimate afterload. SVR values from a PAC are calculated, not directly measured. The calculation assumes nonpulsatile blood flow and does not account for blood viscosity, arterial elastic properties, or microvascular resistance. Despite these limitations, in a critically ill trauma patient, knowledge of SVR may help differentiate between different or overlapping types of shock and guide management (Table 58-2).[7]

Ventricular transmural systolic pressure is the intrathoracic pressure that the ventricular cavity must overcome to achieve ejection. This is notable for the trauma patient undergoing positive-pressure ventilation. Positive end-expiratory pressure (PEEP) decreases the intrathoracic pressure and thus decreases afterload for the left ventricle. For the patient in cardiovascular failure with adequate preload, increasing positive pressure may improve CO. Similarly, removing positive pressure (eg, extubation) increases afterload. For the failing (or recovering) heart, this increased afterload may contribute to end-organ hypoperfusion or extubation failure.[8]

TABLE 58-2: Differentiating Between Overlapping Types of Shock

	Hypovolemic	Distributive	Cardiogenic
Preload (CVP/PCWP)	↓↓	↓↓	↑↑
Cardiac output	↑↑	↑↑	↓↓
Afterload (SVR)	↑↑	↓↓	↑↑
Clinical example	Hemorrhage	Sepsis, spinal cord injury	Heart failure, myocardial infarction

CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure; SVR, systemic vascular resistance.

Cardiovascular failure due to a reduction in afterload is referred to as distributive shock. Etiologies include sepsis, neurologic deficit, and anaphylaxis. In the absence of a PAC, clinical history and physical exam of peripheries and pulse pressure can help assess afterload; warm peripheries and wide pulse pressure would support the diagnosis of distributive shock.[9] In trauma patients, distributive shock in the immediate posttrauma period should raise the suspicion of severe central nervous system injury. Later in the hospital course, sepsis is highest on the differential diagnosis, and the source should be identified and treated accordingly.

Contractility

Contractility, also known as inotropy, is the force with which the myocardium contracts. The inotropic state of the myocardium can be visualized by the construction of a left ventricular pressure–volume loop (Fig. 58-2). This loop is bounded by the four phases of the cardiac cycle: isovolemic relaxation, diastolic filling, isovolemic contraction, and systolic ejection. The difference between end-diastolic volume and end-systolic volume is the stroke volume. The area bounded by this loop is the stroke work, or the external work done by the ventricles to eject the stroke volume. The maximal pressure that can be developed by the ventricle at any given left ventricular volume represents the end-systolic pressure–volume relationship (ie, inotropy). The slope created by this relationship varies directly with inotropic state of the ventricle.[7]

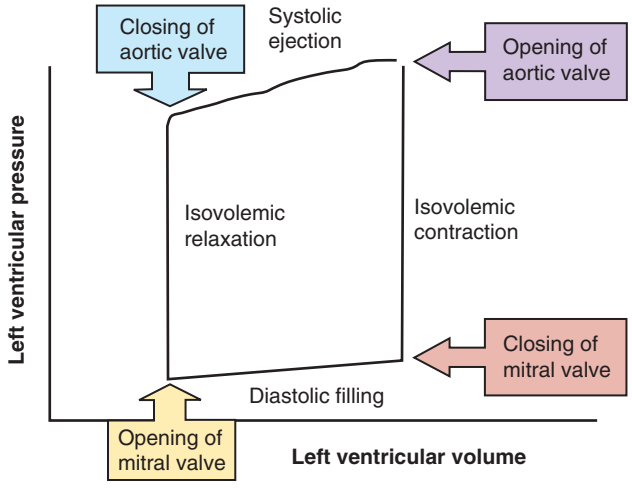

FIGURE 58-2 The relationship between left ventricular pressure and left ventricular volume during a stylized cardiac cycle.

The main factors determining contractility are autonomic nervous system activity, disease, and drugs, mainly by influencing intracellular calcium stores. Sympathetic nerves regulate inotropy and are augmented by epinephrine release during times of stress, such as trauma. Conversely, cardiac disease such as systolic heart failure is associated with reduced peak and sarcoplasmic reticulum calcium uptake. Myocardial ischemia and hypoxia lead to decreased adenosine triphosphate (ATP) production and decreased sarcoplasmic reticulum calcium function. Drugs such as digoxin, β-adrenoceptor agonists, and phosphodiesterase inhibitors influence intracellular calcium by a variety of pathways in order to increase inotropy. However, excessive inotropic stimulation can overwhelm the capacity of myocytes to participate in aerobic metabolism, which can ultimately result in aerobic lactate production, acidosis, and further impairment of sarcoplasmic reticulum function.[7] Clinically, this points toward manipulation of contractility to balance myocardial oxygen supply, demand, and delivery.

Heart Rate

Heart rate is a key determinant of CO. In the setting of constant stroke volume, increasing the number of cardiac ejections per unit time results in increased CO. In addition, increasing heart rate can overwhelm the Na/K-ATPase, allowing intracellular calcium to accumulate via the sodium–calcium exchanger, further increasing inotropy. However, ventricular filling occurs during diastole. In the setting of myocardial failure, high heart rates can shorten the diastolic interval, decrease ventricular filling (ie, preload), and result in decreased CO.[7] High heart rates can also overwhelm myocyte aerobic metabolism, further worsening contractility and CO, as described in the previous section. Therefore, appropriate rate control becomes paramount in ensuring matched oxygen delivery and utilization in patients with cardiovascular failure. Rate control is best initially managed by addressing the underlying cause, such as hypovolemia, anemia, control of pain or agitation, or pulmonary embolism (PE), before initiating rate or rhythm control agents.

EVALUATION OF VOLUME STATUS AND CARDIAC FUNCTION

Because ventricular filling is a key determinant of CO and because knowing the degree of ventricular filling can help rule in or rule out specific causes of shock, assessment of volume status is essential to understand cardiac function in real time. Multiple measurements and tools are available to determine volume status.

Central Venous Pressure

CVP monitoring has been used for many years as a surrogate measurement of preload; however, recent reviews have showed low positive predictive value for CVP as a measure of fluid responsiveness.[10,11] In the young trauma patient with normal cardiac function and ventricular compliance, the CVP monitor provides an assessment of right-sided filling pressure (or preload) that should be used in conjunction with other measures to guide fluid resuscitation.[10] A CVP monitor is usually placed percutaneously as a central venous catheter into the superior vena cava via the subclavian or internal jugular vein. Normal CVP in a nonventilated patient is between 0 and 4 mm Hg; however, response to fluid is more useful than an absolute number. A CVP less than 8 mm Hg has a moderate likelihood of fluid responsiveness.[11] Conversely, an elevated CVP is less likely to respond to fluid or suggests an alternate shock state, such as cardiogenic or obstructive. However, CVP is a static measurement and is affected by multiple variables, which may contribute to its limitation in assessing volume status. Malposition of the catheter, right- or left-sided congestive heart failure (CHF), pneumothorax, lung recruitment techniques or high PEEP, PE, cardiac tamponade, increased intrathoracic pressure, and/or increased intra-abdominal pressure may all lead to increases in CVP measurements, which do not reflect the true overall volume status (Table 58-1).[11] With the current availability of other techniques, CVP should be interpreted in conjunction with measurements such as ultrasound/echocardiography, pulse pressure variation, and passive leg raise in order to assess volume status and cardiac function.[10,11] These are discussed later in this chapter.

Pulmonary Artery Catheters

PACs, first introduced for human use in 1970, have been widely accepted as the standard for invasive monitoring of not only volume status, but also cardiac function by measurement and approximation of left-sided cardiac pressures. Measurement of the PAWP is used as an indirect measurement of LVEDP and LVEDV; however, these measurements often show poor correlation and can be altered by myocardial ischemia, ventricular outflow obstruction, mitral regurgitation, vasodilator therapy, or severe heart failure.[12] Similar to CVP, PAWP can be affected by decreased pulmonary compliance and high PEEP (as seen in lung recruitment strategies). Commercially available PACs currently incorporate multiple calculations to derive other indices of cardiac function and tissue perfusion such as mixed venous oxygen saturation (Svo_2), SVR, oxygen delivery (Do_2), and oxygen consumption (Vo_2).[13] However, a large randomized clinical trial reported that PAC-guided treatment was not superior to that of central venous catheter–guided treatment; mortality and intensive care unit (ICU) length of stay were similar, but the PAC group had a higher rate of complications.[14] A 2013 Cochrane Review analyzing 13 studies found no difference in mortality or hospital or ICU length of stay with the routine use of PAC and found that PAC use was associated with higher costs.[15] Although routine use of PAC may not be indicated in trauma patients, its use should be considered in certain situations, such as in patients with ongoing hemodynamic instability of unclear etiology, patients with a history of CHF or pulmonary hypertension who are not responding as expected to traditional therapy, and patients with suspected hemodynamic changes from blunt cardiac injury.

Echocardiography

Echocardiography, either transthoracic echocardiography (TTE) or transesophageal echocardiography (TEE), has gained wide acceptance in the monitoring of cardiac function and has emerged as an effective tool in the ICU as a guide to resuscitation and monitoring fluid responsiveness. In addition, information about structural abnormalities of the heart, aorta, and main pulmonary arteries can be obtained, as well as assessment of ventricular dysfunction (eg, ejection fraction), ventricular filling, valvular abnormalities, ventricular hypertrophy, and PE. For the critically ill patient in shock, TEE may be able to help differentiate hypovolemic shock (eg, empty/underfilled ventricle) from cardiogenic shock (eg, new wall motion abnormality), obstructive shock (eg, D-shaped septum), and distributive shock (eg, global hypokinesis). Continuous TEE can give accurate measurements of both right- and left-sided filling volumes of the heart and may provide a better assessment of myocardial preload when compared to CVP or PAC measurements.[16] Placed in the esophagus, Doppler probes can accurately predict CO using the diameter of the aorta and measurements of blood flow velocity. The finding of short mitral deceleration time (≤140 ms) is highly predictive of pulmonary capillary wedge pressure of ≥20 mm Hg, which is consistent with cardiogenic shock.[17] A prospective, multicentered study in 2013 demonstrated that the performance of at least 31 supervised examinations over a 6-month period was required to achieve competence in TEE-driven hemodynamic evaluation of a critically ill patient.[18] TEE has been found to be a safe procedure that can be done at the bedside by intensivists and has been found to affect management of the critically ill patient in up to 80% of patients.[19]

Hemodynamic Transesophageal Echocardiography

Hemodynamic TEE (hTEE) is essentially point-of-care echocardiography that has become available for assessment of cardiac structure and function for the intensivist. As with conventional echocardiography (TEE or TTE), hTEE provides information regarding both the overall structure and function of the myocardium, although on a limited scale. Recently developed devices, such as the miniaturized two-dimensional monoplane disposable TEE probe (ClariTEE, ImaCor-USA, Uniondale, NY), allow for continuous less invasive hemodynamic and cardiac monitoring that can be

left in place for up to 72 hours. These devices use a miniature TEE probe (mTEE) with an associated ultrasound system to obtain three cross-sectional views of the heart (midesophageal four-chamber view, transgastric short axis) as well as visualization of the superior vena cava. Two-dimensional transverse, 0-degree images with associated ante- and retroflexion can be viewed.[20]

When compared to TTE or TEE, hTEE/mTEE provides several benefits. Whereas TEE and TTE require extensive training, are more invasive, pose a small risk of complications, and are limited in duration of use, mTEE devices are smaller in size (approximately 5.5 mm in diameter) and can be left in position for intermittent and continuous evaluation for up to 72 hours.[21,22] Multiple studies have demonstrated the learning curve for adequate operator proficiency in both performing and interpreting TEE and TTE. Whereas Charron's group[23] reported that 31 completed and supervised TEEs were adequate for competency in the ICU, the National Board of Echocardiography requires a minimum of 150 exams and interpretation of 300 studies, and the British Society of Echocardiography requires 250 cases over 24 months before accreditation can be sought.[24] Cioccari et al[21] proceeded to demonstrate that proficiency with hTEE/mTEE by ICU practitioners without any prior TEE experience could be obtained after a short training course. Furthermore, the rates of interrater agreement (kappa) between novice TEE practitioners and cardiologists for the three views in hemodynamically unstable patients were all statistically significant (all three views, P <.0001). Associated clinical implications of hTEE use in the ICU and trauma population are present, although limited in the reported literature. hTEE has been used to guide clinical decision making in the care of extracorporeal membrane oxygenation (ECMO) patients, cardiac surgery patients, and trauma patients. The use of hTEE has been shown to aid diagnostic and therapeutic decision making 50% to 66% of the time with respect to critically ill patients, and when compared to standard hemodynamic monitoring, hTEE is able to identify critical issues 66% of the time.[25-28] Unfortunately, only one study has described the use of hTEE in the trauma population to date. Griffin et al[29] demonstrated that the use of hTEE in posttrauma ICU care reduced use of continuous renal replacement therapy (CRRT) as well as days of CRRT use. Although this was a well-powered study, the conclusions drawn regarding hTEE are not easily extrapolated to the posttrauma ICU patient, and further studies involving the trauma population and hTEE are needed.

Ultrasound of the Inferior Vena Cava

Ultrasound evaluation of the inferior vena cava (IVC) can give an estimate of the volume status of the patient and help predict a beneficial response to fluid. The IVC should be identified in a transverse plane with the cardiac probe, and then rotated 90° to the longitudinal plane. IVC diameter should be measured 1 to 2 cm caudad from the hepatic vein–IVC junction (3–4 cm from the caval atrial junction).[30] Static measurements of IVC diameter have been compared to CVP. In a spontaneously breathing patient, an IVC diameter of less than 2 cm is associated with a CVP of less than 10 mm Hg, whereas a diameter of greater than 2 cm is associated with elevated CVP. However, static IVC measurements fail to predict fluid responsiveness.[30] Conversely, dynamic measurements of IVC diameter during the respiratory cycle have been validated to predict fluid responsiveness in both fully ventilated and spontaneously breathing patients. Once the IVC is identified in the manner described earlier, the time-motion (M-mode) feature is used to measure changes in IVC diameter during the respiratory cycle at end expiration and early inspiration. An average of three measurements should be taken. In ventilated patients, positive pressure causes IVC dilation during respiration. An increase of IVC diameter of 12% to 18% during the respiratory cycle of fully ventilated patients indicates fluid responsiveness. In spontaneously breathing, nonventilated patients, negative pressure causes the IVC collapse during respiration. A decrease in IVC diameter greater than 40% is predictive of fluid responsiveness.[30] Multiple studies have demonstrated that ultrasound of the IVC can be a useful noninvasive technique for volume status measurement and fluid responsiveness.[31,32] Serial measurements can also assess response to interventions. However, at this time, the use of IVC ultrasound to predict volume responsiveness in ventilated but spontaneously breathing patients has not been reliably validated.

Pulse Pressure Variation and Stroke Volume Variation

An arterial catheter can be a useful adjunct in the evaluation of a trauma patient's fluid status and heart function (eg, FloTrac Monitor, Edwards Lifesciences, Irvine, CA). Specialized devices can monitor trends in CO and cardiac index, but even simple arterial catheters can provide information regarding a patient's volume responsiveness, via monitoring of the pulse pressure variation (PPV) and stroke volume variation (SVV). Excessive variations in arterial blood pressure caused by the specific interactions of the heart and lungs under positive-pressure ventilation are a clinically well-known sign of hypovolemia.[33] Variations in PPV are exaggerated when the left ventricle (LV) is on the steep portion of the Frank-Starling curve; small preload changes cause large stroke volume changes (see the following box).

Calculation of Pulse Pressure Variation

Pulse pressure variation (PPV):

$$PPV\ (\%) = (PP_{max} - PP_{min})/[(PP_{max} + PP_{min})/2] \times 100$$

where PP_{max} is the largest pulse pressure during inspiration and PP_{min} is the smallest pulse pressure during expiration

This variation is also influenced by ventilator tidal volumes.[11] PPV greater than 8% in low tidal volume patients

(<7 mL/kg) and 11% in high tidal volume patients (≥7 mL/kg) is indicative of fluid responsiveness.[11] PPV is a surrogate of SVV; however, there are limitations in measuring PPV.[34] Pulse pressure can vary independently of the true cardiorespiratory physiology during nonsinus rhythms and may be falsely increased with bradycardia or right ventricular failure. PPV is proportional to stroke volume and inversely related to aortic compliance. Therefore, PPV may vary highly even if SVV is small when aortic compliance is decreased. Finally, spontaneous respiration influences variability and makes PPV difficult to interpret.

In contrast, stroke volume is a volume, not a pressure, measurement. Therefore, SVV may be more accurate than PPV. Multiple studies of SVV have shown improved prediction of fluid responsiveness over CVP and PAWP and have validated SVV and PPV as helpful adjuncts used to improve CO.[35,36] Despite the promise of encouraging initial results in intraoperative patients, the use of SVV has its limitations due to questions of validity at high respiratory rates, with tachycardia, in the presence of β-adrenergic blockade, and in the presence of an over- or underdampened arterial curve.[37]

Arterial pulse contour analysis has also been clinically validated for continuous CO measurement and is now available for the real-time quantification of LV SVV and CO.

Passive Leg Raise

An additional noninvasive method of measuring response to changes in preload is the passive leg raise (PLR) test. The appropriate (and validated) method has the following five steps: (1) Position the patient semi-recumbent with the trunk at 45°. (2) Use the bed controls to both flatten the trunk supine and raise the legs to 45°. (3) Assess the change in CO or pulse pressure and heart rate over 60 seconds. (4) Return the patient to semi-recumbent position with the trunk at 45°. (5) Reassess the hemodynamic status for return to baseline.[38] This method transfers approximately 300 mL of fluid from the lower body to the right heart, and thus PLR simulates the heart's response to a fluid challenge but without adding actual volume. In a recent meta-analysis, a 10% increase in CO during PLR had the highest positive predictive value and lowest negative predictive value for likelihood of fluid responsiveness. Similarly, a 10% increase in PP during PLR had moderate predictive value for fluid responsiveness. These results were fairly robust during both spontaneous and controlled ventilation.[11]

PLR results, however, can be falsely elevated by sympathetic stimulation, the shock state, or vasomotor tone. If the test induces pain, cough, or awakening, an increase in CO or pulse pressure may not be due to the fluid bolus; this is suggested by an elevated heart rate during the PLR.[38] Failure of the CO or pulse pressure to return to baseline after the PLR may indicate the hemodynamic change is influenced by the disease state rather than increased preload with leg raise. Changes in pulse pressure specifically are affected by arterial compliance (increased PPV seen with decreased compliance), as described in the previous section.[38]

MYOCARDIAL DYSFUNCTION AND SHOCK

Myocardial dysfunction can be defined on the basis of perturbed preload, afterload, contractility, or heart rate, as described earlier. Systemic hypovolemia causing inadequate preload, due to hemorrhage or third-space losses, is the most common etiology in the postsurgical or trauma patient. Other causes of decreased CO may arise from acute heart failure or decreased contractile function of the heart. Impaired contractility can be due to the evolution of direct muscle ischemia or from any insult that results in intrinsic metabolic derangements at the cellular level. The latter typically occurs in the settings of sepsis, post–cardiac arrest, post–cardiopulmonary bypass, or traumatic brain injury.[39] Direct physical injury to the myocardium, as occurs following blunt chest injury, may produce contused cardiac muscle, which can lead to contractile dysfunction and decreased CO.

Right Heart Failure

One of the more difficult scenarios to diagnose and manage is dysfunction of the right ventricle (RV) following inferior wall myocardial infarction (MI). Although cardiogenic shock results from RV infarction infrequently, mortality from RV infarction is high (23%–53%).[40] In addition, patients with inferior MI have much higher rates of arrhythmias including atrial fibrillation, ventricular fibrillation and tachycardia, and second- or third-degree atrioventricular block.[41]

The pathophysiology of right heart failure can be explained in terms of end-diastolic pressure. Increased RV afterload (increased pulmonary resistance) decreases the ability of the RV to pump blood forward to the left heart, thus decreasing LV preload. Decreased LV preload results in decreased LVEDP. Increased blood volume in the RV eventually leads to RV dilatation and increases RV preload. Increased RV preload results in increased RVEDP. When RVEDP is greater than LVEDP, the ventricular septum bows from right to left during diastole, further decreasing LV filling volume and LVEDP. This clinical result is decreased CO and, ultimately, cardiogenic shock. In this setting, fluid resuscitation only further increases RVEDP and exacerbates the already decreased LV filling.[7] In critical illness, right heart failure is mainly due to excessive preload, excessive afterload, or insufficient myocardial contractility. These factors can cause increased pressure load or increased volume load on the right heart. For trauma patients, common causes of pressure overload are chronic left heart failure that worsens in the ICU or PE. Volume overload can be caused by excessive fluid resuscitation or right-sided valvular abnormalities. Both overload states can lead to ischemia, but the right heart tolerates pressure overload very poorly. Increased pressure impairs RV wall perfusion and exacerbates ischemia. Impaired contractility, either as a primary cause or physiologic sequela, further reduces CO.[7] Diagnosis requires a multimodality assessment to determine preload, afterload, and contractility. RV preload can be determined by volume status assessments, as described

previously; echocardiography; and clinical resuscitation details. Afterload can be assessed directly by elevated mean pulmonary artery pressure via PAC but is now more often inferred by using chest x-ray, computed tomography (CT) of the chest, and arterial blood gas to identify pathology that induces pulmonary hypoxia (eg, acute respiratory distress syndrome [ARDS]) or obstruction (eg, PE). Contractility can be directly measured using echocardiography, and signs of right heart strain or ischemia can be seen via electrocardiogram (ECG) and troponin. A 15-lead ECG may be necessary to confirm right-sided ischemia. Additional tests, such as ventilation/perfusion scan or abdominal ultrasound, are more often used in the outpatient setting. Once the diagnosis is made, treatment goals are to optimize right-sided filling pressures (preload), reduce right heart afterload, and augment contractility if needed.[42] Details are discussed later in "Management of Specific Cardiac Problems."

Pulmonary hypertension (PH) may develop after significant chest trauma, primarily due to blunt cardiac injury or severe pulmonary contusions and development of ARDS. With blunt cardiac injury, this is most often due to either an acute valvular or papillary disruption, formation of a ventricular septal defect,[43] or myocardial damage to the RV. With contusions/ARDS, the hypoxia-induced vasoconstriction increases RV afterload and can induce the metabolic sequelae leading to elevated pulmonary artery pressures. PH is technically diagnosed with right heart catheterization and mean pulmonary artery pressure of greater than 25 mm Hg at rest; however, formal catheterization is often not feasible in critically ill patients.[42] Therefore, physical examination findings (eg, new and worsening jugular venous distention, a new murmur, unexplained hypotension or hepatic failure, a new arrhythmia) in the proper traumatic setting should prompt further testing with echocardiography. In fact, as many as 27% of major chest trauma patients may have PH suggested on TTE.[44] Signs include a dilated RV and right atrium with normal or small LV, a D-shaped septum, and increased tricuspid regurgitant jet velocity on Doppler measurement (>2.9 m/s suggestive and >3.4 m/s likely).[45] Chest CT scan can help rule out underlying causes such as chronic PE or a preexisting lung pathology.[42] Treatment is based on treating the underlying cause and using the principles described later for management of PH and right heart failure.

Valvular Pathology and Left Heart Failure

Acute traumatic injury to a cardiac valve is rare and is discussed elsewhere (see Chapter 30). Knowledge of common valvular pathology and the impact on trauma resuscitation is important because valve abnormalities can significantly impact the response to fluids, vasopressors, and inotropes. Three of the more common valve disorders (aortic stenosis, mitral regurgitation, and tricuspid regurgitation) will be discussed in this section. In aortic stenosis, narrowing of the aortic valve most often leads to symptoms of left heart failure with patients complaining of dyspnea on exertion, angina, lightheadedness, or syncope. These patients have a fixed CO due to the limited aortic valve area and dilated LV. Thus, persistent and excessive fluid resuscitation or failure of diuresis during recovery may exacerbate the symptoms of aortic stenosis. Similarly, hypotension is poorly tolerated due to decreased LV compliance.[7] Induction of anesthesia or rapid sequence intubation should ensure adequate preload and hemodynamically neutral agents in order to avoid drops in blood pressure that can lead to cardiac arrest. Epidural catheters can similarly cause significant hemodynamic effects that must be anticipated if placement is necessary. Chronic mitral regurgitation may lead to decompensated LV failure. Often, due to left atrial enlargement, these patients suffer from atrial fibrillation or can readily go into atrial fibrillation if the atria are overstretched by volume resuscitation. As with aortic stenosis, responsible fluid management in these patients is necessary. Excessive fluid resuscitation may lead to the development of pulmonary edema and right heart failure. Acute mitral regurgitation can result from either acute MI or, rarely, blunt cardiac injury damaging the mitral valve.[46] Acute mitral regurgitation can lead to the rapid development of cardiogenic shock, pulmonary edema, and hemodynamic instability. Given the failure of the mitral valve, the pressures within the left atrium and ventricle will equalize, impairing ventricular filling. Treatment is generally emergent and includes afterload reduction for hemodynamic stabilization and surgical repair.[47] Tricuspid regurgitation is common and infrequently symptomatic in the general population but is the least common traumatic valvular injury.[48] Tricuspid regurgitation can lead to clinical signs of jugular venous distention and abdominal and ankle edema. Without judicious resuscitation, fluid overload will lead to right heart failure, which can impair CO with deficits in end-organ perfusion.

Unstable Arrhythmias

Critically ill patients are at high risk for developing arrhythmias, due to elevated catecholamines, fluctuations in intravascular volume, electrolyte disturbances, and inflammatory states, such as surgery and sepsis.[49] The development of an arrhythmia in the ICU is associated with increased mortality, with a recent study showing a 17% in-hospital mortality for patients without the development of an arrhythmia, 29% for those who developed a supraventricular arrhythmia, and 73% for those with a ventricular arrhythmia.[50] New onset of an arrhythmia in the ICU also leads to increased length of stay by up to 33%.[51] Identification of any arrhythmia should prompt an evaluation of electrolyte levels, assessment of the rhythm with an ECG, review of medications, and clinical exam for hypotension or signs of impaired perfusion (mental status changes). An echocardiogram may also be indicated if no recent exam exists or the arrhythmia is new. The management of arrhythmias follows the most recent Advanced Cardiac Life Support (ACLS) guidelines.[52]

The most common tachyarrhythmia in the critically ill patient is atrial fibrillation.[53] Given a 25% prevalence of atrial fibrillation in the community and an aging population,

either preexisting or new-onset atrial fibrillation may be seen in up to one-third of critically ill patients. Atrial fibrillation is thought to occur via an arrhythmogenic atrial substrate and an arrhythmogenic trigger that ultimately leads to atrial fibrosis and electrical remodeling. In critical illness, fibrosis and remodeling can be accelerated due to infection and/or inflammation. Risk factors for atrial fibrillation in the ICU include atrial stretch (volume overload), excessive adrenergic stimulation (vasopressors, infection, β-agonists, ventilator dyssynchrony), myocyte trauma/injury (ischemia, contusion), altered intracellular ions (electrolyte abnormalities), inflammation (surgery, sepsis), and increasing injury severity.[53] Thus, clinical assessment should include identifying and modifying risk factors where possible, including an evaluation as described earlier.

The treatment algorithm then depends on whether the patient is hemodynamically stable or unstable. For *hemodynamically stable* patients, medical cardioversion with rate control (calcium channel blockers, β-blockade, or digoxin) or rhythm control agents (magnesium or amiodarone) can be used.[53] In patients with *hemodynamically unstable* presentations, such as hypotension and mental status changes, emergent electrical cardioversion is indicated. While many recommend starting with 100 J, our practice is to proceed directly to 200 J with more immediate resolution of atrial fibrillation. In addition, we recommend premedicating the patient with intravenous midazolam (2–4 mg) to provide perievent amnesia; however, in postoperative patients requiring cardioversion, successful conversion to sinus rhythm occurred in 71%, and only 43% and 23% of patients remained in sinus rhythm at 1 and 2 hours after cardioversion, respectively.[54] Therefore, concurrent rate or rhythm control therapy should be considered to reduce the risk of recurrence.

Bradyarrhythmias in the critically ill patient can be life threatening. Most commonly, bradyarrhythmias are caused by elevated intracranial pressure, exaggerated vagal activity, hypothermia, electrolyte abnormalities, and medications (β-blockers, calcium channel blockers, clonidine, opioids, and dexmedetomidine).[55] Assessment, as described earlier, should be initiated. Management includes removing the offending agent, if possible. Otherwise, atropine or urgent transcutaneous pacing should be emergently organized if there is evidence of hypoperfusion.

Septic Myocardiopathy and Neurogenic Stunned Myocardium Syndrome

In the subacute phase of severe trauma, patients are subject to septic complications related to injury, surgery, or their ICU stay. In some patients with severe sepsis or septic shock, inflammatory cytokines can trigger a nitric oxide cascade and mitochondrial dysfunction that ultimately causes depressed cardiac contractility. This septic cardiomyopathy has three distinct features: LV dilatation (with normal or low filling pressures), depressed ejection fraction, and recovery in 7 to 10 days. Treatment is generally supportive by treating the underlying infection, using targeted volume resuscitation, and using vasopressor support when needed.[56]

Patients with traumatic brain injury and subarachnoid hemorrhage have a systemic catecholamine "storm" triggered by the central nervous system axis. The subsequent massive sympathetic outflow can cause a complex cascade of autonomic and inflammatory responses that can ultimately result in adverse cardiac effects. The neurogenic stunned myocardium syndrome (NSM) is a neurologically mediated cardiac injury characterized by ECG changes, arrhythmias, LV dysfunction, and elevated cardiac biomarkers. It is caused by excessive norepinephrine release from myocardial sympathetic nerve terminals, independent of circulating catecholamine levels. The severity of NSM and cardiac insult is related to the severity of the underlying brain injury. There is a spectrum of cardiac injury, from ECG changes to cardiogenic shock and pulmonary edema. NSM is more common after subarachnoid hemorrhage but should be on the differential diagnosis for traumatic brain injury patients showing signs of cardiac abnormalities with low suspicion of baseline coronary artery disease.[39]

ECG changes include ST-segment changes, flat or inverted T waves, U waves, and QTc prolongation and are most common the first few days after injury. Electrical changes generally do not follow a vascular ischemic territory. Arrhythmias, including sinus tachycardia, atrial fibrillation, premature atrial and ventricular contractions, and atrioventricular dissociation, often occur within the first week after injury. Most are benign, but careful attention to electrolyte levels and QTc-prolonging medications is important. Elevations of cardiac troponin I can be seen early after brain injury, and troponin I usually peaks below the level consistent with MI.[39] Ventricular dysfunction can be detected echocardiographically; includes impaired LV contractility, hypokinesia, and decreased ejection fraction; and often occurs within 3 days of brain injury. The key feature that differentiates NSM from a primary cardiac event is the pattern of regional wall motion abnormalities (RWMAs), which involve the basal and middle portions of the anteroseptal and anterior ventricular walls with apical sparing. The RWMAs follow the sympathetic nerves instead of the vascular territories and resolve spontaneously within days.[39]

Cytopathic Hypoxia

During the acute resuscitative phase of care, cardiovascular failure in trauma patients is the result of hemorrhage, and reversal of this failure occurs only with control of hemorrhage and transfusion of blood products. However, some patients remain in cardiovascular failure despite hemorrhage control and adequate blood product resuscitation. These patients often remain acidotic with large base deficits. The underlying derangement in this circumstance may be cytopathic hypoxia.[57,58] Although an infrequent cause of cardiovascular failure, it should be considered in the adequately resuscitated but hypotensive and acidotic patient. In fact, cytopathic hypoxia may manifest as profound vasoplegia

(see later in chapter) with potential etiologies being mitochondrial dysfunction, problems with nitric oxide metabolism, and alterations in hypoxia-induced pathways. Although no specific therapy is available, treatment must be systemic and target all cells (eg, methylene blue, ubiquitin, therapeutic hypothermia).

MANAGEMENT OF MYOCARDIAL DYSFUNCTION

As described previously, myocardial dysfunction should be managed based on optimizing preload, afterload, contractility, and heart rate. However, sometimes vasopressors, inotropes, and mechanical support are necessary to augment the failing heart until recovery or necessary interventions can occur.

Vasopressors

The use of vasopressors during acute resuscitation after severe injury should be avoided if possible because hemorrhage is the source of almost all shock in the immediate postinjury period (Table 58-3).

NOREPINEPHRINE

Norepinephrine is an endogenous sympathetic neurotransmitter with α- and β-adrenergic effects. α-Receptors are primarily in vascular smooth muscle, and activation leads to vasoconstriction. In cardiac myocytes, β_1-receptor stimulation leads to increased inotropy and chronotropy, whereas in vascular smooth muscle, β_2-receptor stimulation results in vasodilation. At high doses, α-adrenergic effects predominate, with less β_1 effects and even less β_2 effects. Thus, increased SVR and increased blood pressure result. Specifically, in the setting of RV failure, low-dose norepinephrine improves cardiac function without adversely affecting visceral perfusion.[59] Norepinephrine is typically the vasopressor of first choice during or after fluid resuscitation[5] and in the setting of sepsis.[9] In cardiogenic shock, norepinephrine, when compared to dopamine, is associated with decreased mortality and rates of arrhythmia.[60] The latter is likely due to lower β_1 effects than dopamine.[61] Norepinephrine is widely used in the care of the head-injured patient in shock because its vasoconstrictive effects do not extend to the cerebral vasculature, making it an ideal agent for maintaining cerebral perfusion pressure. However, norepinephrine's β effects can worsen hypoxic pulmonary vasoconstriction and thus should be spared in the patient with concomitant hypoxemia, respiratory failure, or PH.[62]

VASOPRESSIN

Vasopressin is a natural hormone produced by the posterior pituitary that acts as a potent vasoconstrictor, acting on V1 receptors in vascular smooth muscle.[61] Vasopressin is recommended as the second vasopressor during septic shock that is refractory to norepinephrine according to the Surviving Sepsis Campaign.[9] When vasopressin is combined with norepinephrine, outcomes in the treatment of catecholamine-resistant cardiovascular failure in septic shock are superior to therapy with norepinephrine alone. In the postoperative cardiotomy patient, vasopressin significantly reduces the need for both pressor and inotropic support and the development of arrhythmias.[63]

Vasopressin also has pulmonary vasodilatory properties, likely mediated via a nitric oxide pathway. The clinical effects are thought to be a reduced pulmonary vascular resistance (PVR)/SVR ratio. In contrast, norepinephrine has been found to increase the PVR/SVR ratio. Thus, vasopressin should be considered early in patients with hypotension and significant hypoxia or pulmonary hypotension.[62]

PHENYLEPHRINE

Phenylephrine is a pure α-agonist and has no β activity. Phenylephrine is often administered in the bolus form for acute hypotension but may also be infused in patients with hypotension and serious arrhythmias that may be triggered

TABLE 58-3: Dosage, Mechanism, and Actions of Pharmacologic Agents Commonly Used in the Treatment of Cardiovascular Failure

Agent	Typical dosage	HR	MAP	CO	SVR	α_+	β_1	β_2
Vasopressors								
Norepinephrine	0.5–30 mcg/min	+	++	+	++	++	+	
Vasopressin	0.04 U/min		++	–	++			
Phenylephrine	0.5–1 mcg/kg/min		++		++	++		
Inotropes								
Dobutamine	2–20 mcg/kg/min	+	±	++	–		++	+
Epinephrine	0.5–10 mcg/min	++	++	+	++	++	++	++
Milrinone	0.125–0.75 mcg/kg/min			+++	–			
Dopamine	3–5 mcg/kg/min	+	+	++			++	
	10–20 mcg/kg/min	++	++	+	+++	++	+	

CO, cardiac output; HR, heart rate; MAP, mean arterial blood pressure; SVR, systemic vascular resistance.

by other vasopressors with β activity.[57] It is also favored to correct hypotension in patients with aortic stenosis, hypertrophic obstructive cardiomyopathy, or vagally mediated reactions.[61] Phenylephrine has a duration of action of up to 20 minutes, making it a useful agent for periprocedural hypotension.[64] At high doses, phenylephrine administration may lead to reflex bradycardia[65] and visceral hypoperfusion.

METHYLENE BLUE FOR PROFOUND VASOPLEGIA

Profound vasoplegia occurs when hypotension and inadequate end-organ perfusion persist despite adequate fluid resuscitation, high CO, and the use of multiple vasopressors.[66] The exact physiologic mechanism of profound vasoplegia is poorly understood but is most commonly encountered in cardiac surgery patients, especially those who have required cardiopulmonary bypass. Mortality rates, wound infections, days on mechanical ventilation, and lengths of stay are all increased in cardiac patients who have profound vasoplegia.[30] Profound vasoplegia may occur in the trauma and postoperative surgical patient as well. Unfortunately, additional treatment modalities for profound vasoplegia are limited. Methylene blue has been used with some success to help to reverse profound vasoplegia. Methylene blue inhibits nitric oxide–mediated vasodilation and can be administered as a slow infusion (2 mg/kg over 20 minutes) for this purpose. Outside of cardiac surgery, methylene blue has been used to reverse vasoplegia in burn patients,[67] following liver transplant,[68] and for septic shock.[69] It is also first-line treatment for symptomatic methemoglobinemia, or methemoglobin levels greater than 20%. Although it should not be considered standard of care, methylene blue may be used in a trauma patient with profound shock unresponsive to fluids or vasopressors as a last resort.

Inotropes

EPINEPHRINE

Epinephrine is an endogenous catecholamine with significant α- and β-adrenergic activity on cardiac and vascular smooth muscle. At high doses, epinephrine has more α-adrenergic effect, causing intense vasoconstriction. At low doses, it exerts primarily β-adrenergic effects, increasing contractility and reducing SVR. Despite this, there is little evidence that epinephrine is superior to dobutamine in the treatment of low-output states. However, it is the first-line agent when managing cardiac arrest, as it rapidly increases diastolic blood pressure to help resume organized myocardial contraction.[61] In patients with septic shock who have been adequately fluid resuscitated, epinephrine increases heart rate and stroke volume (and therefore CO) and systemic oxygen delivery without altering vascular tone. Care must be taken when using epinephrine because renal vasoconstriction, cardiac arrhythmias, reduced splanchnic blood flow,[59] and increased myocardial oxygen consumption and demand may result.[70] Additionally, metabolic abnormalities are common, including dyskalemias, hyperglycemia, and ketoacidosis.[71]

DOBUTAMINE

Dobutamine is a synthetic catecholamine with primarily β-adrenergic effects, although it does possess some α_1-adrenergic properties. Its predominant β_1 effects make it a strong inotrope, increasing contractility, with weaker chronotropic effects. Dobutamine also has β_2-adrenoreceptor activity, producing peripheral vasodilatation. This combination of increased contractility and reduced afterload results in improved CO. However, the increase in CO occurs with an increase in myocardial oxygen consumption (as seen with its exercise-mimicking effects). Thus, dobutamine utility may be limited in settings of cardiac ischemia, and malignant arrhythmias can occur at any dose.[61] Because of the vasodilatory effects, dobutamine is ideally suited for use in low-output cardiac states. For these reasons, dobutamine should be considered the first-choice inotrope for patients with low CO once adequate preload has been confirmed.[9] Its short half-life also makes it easily titratable. In addition, dobutamine may be added in the setting of septic shock if cardiac dysfunction is considered to be a contributor to the shock state.

MILRINONE

Milrinone selectively inhibits phosphodiesterase-3, which leads to increased cyclic adenosine monophosphate (cAMP) and increased calcium intracellularly to promote more efficient contractility. Overall, milrinone administration leads to pulmonary and systemic vasodilation with increased inotropy. The pulmonary vasodilation effects make it a useful agent in PH or high RV afterload states. Milrinone is also used more frequently in the patient with well-established cardiac failure because the half-life is long (2.5 hours) and it is not a rapidly titratable medication, unlike dobutamine or epinephrine. As with dobutamine, hypotension may result from either bolus or excessive milrinone use, especially in patients who are hypovolemic. Although there is increased risk of arrhythmia with milrinone, it is slightly less intense than with dobutamine due to less significant chronotropic effects.[61]

DOPAMINE

Dopamine is an endogenous catecholamine that has several cardiovascular effects, including increased heart rate, increased contractility, and peripheral vasoconstriction. It has historically been used primarily for inotropic support, although it is no longer a first-line (or second-line) agent. Dopamine acts on α- and β-adrenoreceptors as well as DA1 and DA2 dopamine receptors, and its actions can be classified based on dose. At doses of 1 to 3 mcg/kg/min, dopamine acts at primarily DA1 receptors in renal, mesenteric, coronary, and cerebral vascular beds, resulting in vasodilation. At moderate doses (3–5 mcg/kg/min), dopamine stimulates primarily cardiac β-adrenoreceptor, increasing contractility and thus CO. At higher doses of dopamine (10 mcg/kg/min), peripheral vasoconstrictive effects from stimulation of α-adrenergic receptors predominate. This can result in significant coronary vasoconstriction resulting in angina, vasospasm, and increased PAWP. Additionally, increasing afterload from

vasoconstriction coupled with an increased heart rate seen at this dose result in increased myocardial oxygen consumption and demand. Individual variation in the pharmacokinetics of dopamine due to weight-based dosing typically results in poor correlation between blood levels and administered dose. Tachycardia can occur with any dose of dopamine, particularly in the hypovolemic patient. Due to the variable effects of dopamine, the dosage ranges used to define which receptors it affects are to be used as broad guidelines only, with the awareness that low-dose dopamine may have the unwanted effects of medium- or high-dose dopamine on an individual patient. Low-dose dopamine is no longer used for renal protection.[72]

DIGOXIN

Digoxin is a cardiac glycoside that is traditionally used as an oral agent in the outpatient setting. However, it can have several uses in the critically ill patient. In myocardial cells, digoxin binds to the α-subunit of Na/K-ATPase and inhibits the Na/K pump, ultimately increasing intracellular calcium and contractility. Thus, it can work as an inotrope to improve cardiac contractility in the patient with systolic heart failure. It is in this setting that our group has found digoxin to be most useful in weaning other cardiovascular therapeutics and chemical agents. Digoxin also increases vagal tone and thus can be used to decrease ventricular rates in patients with supraventricular tachycardias, including atrial fibrillation. While other agents are more commonly used to manage atrial fibrillation in the critical care setting, digoxin should be considered if patients have contraindications to other agents or have refractory atrial fibrillation. Note that digoxin is contraindicated in the atrial fibrillation secondary to Wolf-Parkinson-White syndrome or in hypertrophic cardiomyopathies. Due to risk of toxicity and a narrow therapeutic window, digoxin levels should be monitored closely in the critically ill, and dosing adjustments should be considered in the setting of geriatric patients or those with renal insufficiency.[61]

Mechanical

INTRA-AORTIC BALLOON PUMP COUNTERPULSATION AND MECHANICAL SUPPORT

Until recently, when myocardial failure had an underlying, surgically correctable, anatomic cause and pharmacologic methods were ineffective in augmenting CO, the use of intra-aortic balloon counterpulsation (IABP) was quite common.[73] However, recent studies, including the IABP-SHOCK II trial, showed no benefit in mortality for IABP use in patients in cardiogenic shock.[74] The 2013 American College of Cardiology Foundation and American Heart Association guidelines on post–ST-segment elevation myocardial infarction (STEMI) care downgraded the recommendation for use of IABP.[75] However, there are still some cases where this type of mechanical support is used, and surgeons managing these patients should be familiar with its use and function. IABP involves placement of a balloon catheter into the proximal descending aorta (distal to the origin of the left subclavian artery) via the femoral artery. The balloon catheter is connected to a pumping device that, in synchrony with the ECG, inflates the balloon during cardiac diastole and deflates it during systole. By filling during diastole, the balloon displaces approximately 40 mL of blood retrograde into the coronary arterial circulation and antegrade into the descending aorta. The balloon is abruptly deflated at the beginning of systole, decreasing LV afterload and facilitating the LV to eject its stroke volume. These dual functions of augmenting coronary arterial flow while decreasing afterload thereby reduce myocardial oxygen consumption and increase systemic blood pressure. Use of IABP is considered a bridge until there is myocardial recovery or a different type of treatment can be obtained. It also requires anticoagulation to prevent clot burden on the balloon.[76]

Other methods of providing direct inotropic support to the acutely failing heart include the Impella and Tandem Heart devices. These devices are inserted percutaneously and deposited into the left heart to provide circulatory support for a failing LV.[77] Like the IABP, both of these devices are used to bridge the patient until more appropriate, longer-term therapy is available and require systemic anticoagulation, which may limit use in the trauma patient.

EXTRACORPOREAL MEMBRANE OXYGENATION

ECMO can provide temporary respiratory support (venovenous [V-V]) or cardiorespiratory support (venoarterial [V-A]) as the last step in resuscitation.[78] Essentially, ECMO can provide the necessary blood flow to maintain oxygen delivery to the brain and end organs (eg, large RV infarct) while also oxygenating and removing carbon dioxide from the blood if the lungs are unable to adequately do so (eg, severe ARDS). Traditionally, ECMO is run with an anticoagulant as the ECMO circuit is thrombogenic and common complications of ECMO are intracranial, surgical, gastrointestinal, or pulmonary bleeding.[46] Given the need for anticoagulation, the use of ECMO in trauma patients who often have injuries that prevent anticoagulation has been limited. However, with newer circuits that have anticoagulant coatings, recent reports have shown that ECMO with or without anticoagulation[47] can safely and successfully be performed in trauma patients.[79,80] With this in mind, though, the use of ECMO in trauma patients should be reserved for centers well experienced in the delivery of ECMO.

OPTIMAL END POINTS OF RESUSCITATION

Mixed Venous Oxygen Saturation

Mixed venous oxygen saturation (Svo_2) is a measurement of the oxygen saturation in the venous return to the heart. It is an indirect measurement of oxygen utilization by the end

organs, as the Svo_2 reflects the amount of oxygen "left over" after tissue extraction. A true Svo_2 is taken from the distal tip of a PAC. This reflects oxygen utilization from the upper body (superior vena cava), lower body (IVC), and the coronary veins (coronary sinus). It becomes "mixed" by the time it reaches the pulmonary artery and thus reflects global oxygen consumption. Normal values are approximately 65% to 80% and are calculated by using a derivation of the Fick equation to determine the difference between measured oxygen delivery and calculated oxygen consumption. Central venous oxygen saturation measurements ($Scvo_2$) are obtained from internal jugular or subclavian central catheters. $Scvo_2$ reflects oxygenation from the head and upper limbs; thus, normal values are slightly higher than mixed venous oxygenation (>70%).[81]

Determinants of Mixed Venous Oxygen Consumption

Variables determining mixed venous oxygen consumption

$$VO_2 = CO \times Hb \times (Sao_2 - Svo_2)$$

VO_2 = oxygen consumption
CO = cardiac output
Hb = hemoglobin
Sao_2 = oxygen saturation
Svo_2 = mixed venous oxygen saturation

As expected by the equation, changes in the above variables may increase or decrease Svo_2.

High Svo_2 can occur with:

- Increased oxygen delivery (increased Fio_2, hyperoxia, hyperbaric oxygen)
- Decreased oxygen demand (hypothermia, anesthesia, paralytic)
- High-flow states (sepsis, hyperthyroidism, severe liver disease)

Low Svo_2 can occur with:

- Decreased oxygen delivery
  - Decreased hemoglobin (anemia, hemorrhage, dilution)
  - Decreased oxygen saturation (hypoxemia)
  - Decreased flow (shock, arrhythmia)
- Increased oxygen demand (hyperthermia, shivering, pain, seizure)

However, Svo_2 can be falsely high despite end-organ hypoxia. In the trauma ICU, this can occur acutely with burn patients subjected to cyanide poisoning or subacutely with sepsis and significant microvascular shunting. Even though the cell may have inadequate perfusion, the inability to use oxygen can result in a normal or elevated Svo_2.[81]

Lactate and Base Deficit

Elevated serum lactate, or lactic acidosis, can be an indicator of cellular hypoxia or shock. When cells can no longer function normally due to hypoxia or decreased blood flow, the normal mechanism of ATP generation by aerobic metabolism is shifted to anaerobic metabolism. This inefficient method of ATP production yields increased amounts of pyruvate, which is converted to lactate. Therefore, increased levels of lactate may be a reflection of ongoing tissue hypoxia. The use of serum lactate measurements as a guide to resuscitation is limited due to the time necessary for laboratory results to return if point-of-care tests are not available. However, in critically ill patients past the acute resuscitation phase of treatment, serum lactate levels may be useful in determining the onset of organ dysfunction.[82] In clinical practice, multiple other factors may also affect lactate levels. Following severe injury, increased circulating epinephrine may cause an increase in lactate production by aerobic glycolysis in response to increased activity of the Na^+/K^{1+}-ATPase, despite adequate tissue perfusion.[83] Hepatic dysfunction may increase serum lactate levels due to inability of the liver to metabolize lactate to carbon dioxide. Seizures, such as after traumatic brain injury, may cause an acute elevation in lactate that then clears quickly. An undiagnosed bowel injury or ischemia may also cloud the picture in the setting of a rising or persistently elevated lactate.

The base deficit is the difference between the standard value of 24 mEq/L and the serum bicarbonate level. This value is typically negative in hypovolemic shock, and therefore, the term *base deficit* is widely used. The magnitude of the base deficit has been used to quantify the magnitude of acidosis, with mild acidosis having a base deficit of –3 to –5 mEq/L; moderate acidosis, –6 to –10 mEq/L; and severe acidosis, less than 10 mEq/L. As the magnitude of the base deficit increases, the resuscitation volumes of both blood and fluid increase, as does mortality.[82] Patients who are able to clear their base deficits in less than 48 hours have decreased mortality when compared to patients whose base deficits persist beyond this time frame. Persistent base deficits despite normal vital signs may be a signal of compensated shock requiring further resuscitation. If left untreated, it may lead to multiorgan dysfunction and increased morbidity and mortality. New-onset lactic acidosis and increasing base deficit in the critically ill patient may be early signs of a low-flow state, cellular hypoxia, and/or end-organ injury; therefore, every effort should be made to find the cause and address it as quickly as possible.

Capnography

Defined as the measurement of carbon dioxide (CO_2), capnography is most commonly used during endotracheal intubation to ensure proper placement of the endotracheal tube. Other studies have reported its use to predict prognosis following cardiopulmonary arrest, to assess resuscitation efforts, to detect alveolar dead space changes, and to monitor sedation and paralytic therapy.[81,82] Under normal conditions, exhaled CO_2 closely correlates with arterial blood CO_2 levels. Due to unmatched areas of ventilation and perfusion in the lungs, the arterial $Paco_2$ will usually be slightly higher than the exhaled CO_2.

End-tidal CO_2 ($EtCO_2$) has been shown to predict resuscitation efforts during cardiopulmonary arrest.[82] Higher $EtCO_2$ levels correlate with increased survival after cardiac arrest. Original research found that nonsurvivors with prehospital arrests after 20 minutes of ACLS were found to have an average $EtCO_2$ of 3.9 mm Hg, whereas survivors had $EtCO_2$ values of 31 mm Hg.[84] An $EtCO_2$ value of 10 mm Hg has been determined to provide a 100% sensitivity to predict return of spontaneous circulation (ROSC).[85] However, subsequent guidelines have advocated thresholds of 10 to 20 mm Hg to improve delivery of chest compressions. A more recent meta-analysis found that an $EtCO_2$ of 25 mm Hg better predicted ROSC and that further research is needed to optimize $EtCO_2$ targets and improve outcomes following ROSC after cardiorespiratory arrest.[86]

MANAGEMENT OF SPECIFIC CARDIAC PROBLEMS

Management of Acute Heart Failure

The shock state precludes vasodilation; however, in acute heart failure without shock, afterload reduction with vasodilators may be advantageous to global perfusion while a rapid reduction in preload should help with a heart that cannot handle whole-body fluid demands. The most commonly used intravenous agents in the critically ill include nitroprusside and nitroglycerin.[2]

Nitroprusside acts primarily on arteriolar smooth muscle, reducing afterload. The onset of action of nitroprusside is rapid, and its effects cease within minutes once the infusion is discontinued. When titrating the dose of nitroprusside, SVR should be decreased with a concomitant increase in CO, thereby maintaining a relatively constant systemic arterial pressure. Although the effects of nitroprusside favor arterial dilatation, it does have mild venous dilatory effects that can lead to an increase in venous capacitance and decreased preload. Care must be taken when using nitroprusside because cyanide toxicity is a known side effect. This is generally seen with infusion rates in excess of 10 mcg/kg/min, with prolonged therapy (several days), or in the setting of renal or hepatic dysfunction. Treatment is with sodium nitrite and is aimed at providing an alternate substrate for the cyanide ion. Sodium nitrite also converts hemoglobin to methemoglobin, producing a ferric ion that competes with the ferric ion in the cytochrome system for the cyanide ion. Methylene blue can be administered to treat the methemoglobinemia that results from sodium nitrite treatment.[87]

Nitroglycerin, a potent arteriolar and venous smooth muscle dilator, is a useful agent when both preload and afterload are elevated. The cardiovascular effects are dose dependent, with low doses (5–20 mcg/min) primarily increasing venous capacitance and higher doses (>20 mcg/min) relaxing arterial tone. Side effects are generally the result of an overly rapid reduction in venous or arterial tone and are readily reversed by cessation of the medication.[2,88]

Management of Acute-on-Chronic Heart Failure

As our population ages, the number of elderly patients in the ICU will increase. In fact, the number of elderly patients (>65 years old) is expected to double in the next three decades. With an aging population, there are age-related changes in physiology, exacerbations of chronic illnesses, and effects of therapeutic drugs, which need to be taken into consideration when caring for the traumatically injured patient. Often, these patients will present with chronic CHF. Acute-on-chronic heart failure in the geriatric patient, which may develop in the posttrauma period, needs to be considered in the setting of low urine output despite fluid resuscitation or worsening pulmonary edema. In this case, fluid status and medication administration need to address both the traumatic injury and heart failure. This is a difficult balancing act that requires a combination of the cardiac monitoring and multiple volume status assessments, as previously discussed in this chapter. In addition, the 2017 American College of Cardiology (ACC)/American Heart Association (AHA) guidelines for heart failure recommend obtaining admission levels of B-type natriuretic peptide (BNP) or N-terminal pro-BNP (NT-pro-BNP) to assist in prognostication of acutely decompensated heart failure. Higher admission levels of natriuretic peptide are associated with greater risk for morbidity and mortality (all-cause and cardiovascular).[89]

From a chronic heart failure perspective, many patients will present on an angiotensin-converting enzyme inhibitor (ACE-I), angiotensin receptor blocker (ARB), or angiotensin receptor–neprilysin inhibitor (ARNI). However, these agents can be nephrotoxic and should be held in the setting of acute kidney injury, which is common in critically ill patients. A second-line approach that can be used in the patient with heart failure but without shock is hydralazine and nitrates (isosorbide dinitrate).[89] In addition, these patients often present on diuretics, including mineralocorticoid antagonists. For the trauma patient immediately post hemorrhagic shock, diuresis is counterintuitive (and potentially dangerous), and risk of hyperkalemia with acute kidney injury is a real concern. Thus, frequent assessments of volume status and consideration for early, goal-directed diuresis must be part of the clinical care plan, with resumption of heart failure medications as soon as appropriate.

Management of Diastolic Dysfunction

The syndrome of CHF typically brings to mind an enlarged heart and decreased systolic function; however, systolic function is normal, or near-normal, in nearly 50% of patients diagnosed with CHF. The presence of other clinical manifestations of CHF, such as orthopnea, dyspnea, increased jugular venous pressure, and abnormal heart sounds on auscultation, leads to the diagnosis of diastolic dysfunction (DD). DD is one of the underlying causes of heart failure with preserved ejection fraction (HFpEF). DD is associated with hypertension, diabetes mellitus, female gender, coronary artery

disease, and chronic atrial fibrillation. Aging itself has been associated with reductions in elastic properties of the great vessels, decreased ventricular filling, diminished relaxation and compliance, and decreased β-adrenergic receptor density. The diagnosis of DD or HFpEF is generally made clinically and is often one of exclusion. Objective testing should include echocardiography or cardiac catheterization. Because the normal ejection fraction is not a standardized number and given that systolic dysfunction often accompanies DD, the diagnosis of DD is often not clear-cut. Therefore, the diagnostic "gold standard" for DD is cardiac catheterization demonstrating increased ventricular diastolic pressure with normal systolic function and volumes. Echocardiography, being noninvasive and practical, can be used to exclude systolic dysfunction. The majority of clinical studies have been geared toward management of systolic dysfunction, with a relative paucity of information regarding the specific management of DD.[90]

In general, the treatment principles for patients diagnosed with DD include antihypertensive therapy, reduction of volume overload, decreasing heart rate, maintenance of sinus rhythm, and the reduction of ischemia. With hypertension being the primary predisposing factor for CHF, neurohormonal regulation has been shown to play a role in CHF and DD. The renin–angiotensin system influences CHF indirectly by causing hypertension and LV hypertrophy (LVH) and directly by both angiotensin II and endothelin contributing to LVH and impaired myocardial relaxation. Blockade of the renin–angiotensin system improves diastolic distensibility in both human and animal studies. Volume overload can be reduced with diuretics or renal replacement therapy in patients with renal failure. In DD, the use of β-adrenergic blockade or calcium channel blockade to reduce heart rate and increase LV filling time reduces mortality. Digoxin decreases hospitalization of patients with CHF with and without systolic dysfunction; however, because digoxin is a negative chronotrope, the benefit may be due to the rate-lowering effect of the drug rather than to its other hemodynamic properties. Although ACE-I therapy is beneficial in patients with hypertensive disease and in patients with mixed systolic and diastolic dysfunction, there is no direct evidence that ACE-Is reduce mortality or morbidity in patients with DD.[90] However, hypertension control is important for treatment; thus, use of β-blockers, ACE-Is, and ARBs are part of the ACC/AHA 2017 guidelines for management of HFpEF.[89] If a patient has both DD and rate-altering arrhythmias, such as atrial flutter or atrial fibrillation, rate control will lead to increased filling times once normal sinus rhythm is restored. Myocardial ischemia can also contribute to DD; thus, patients with drug-resistant ischemic DD may benefit from percutaneous coronary intervention (PCI) or coronary artery bypass grafting. Aldosterone has also been shown to contribute to CHF with detrimental effects on endothelial function as well as inducing a vasculopathy. The latest guidelines suggest that patients with ejection fraction greater than 45%, elevated BNP levels on admission, and adequate renal function be considered for aldosterone receptor antagonists in order to reduce hospitalizations.[89]

Management of Right Heart Failure and Pulmonary Hypertension

As described earlier, right heart dysfunction may progress from underlying pathology or acquired injury in the setting of trauma and critical illness. Management is based on targeting areas of dysfunction with respect to preload, afterload, and contractility.

OPTIMIZING RV PRELOAD

The right heart needs to be full enough, but not too full as to impair LV filling (as described earlier). Judicious fluid resuscitation and objective measures of volume status to assess ventricular filling are advised. Ventilator PEEP can also drop venous return and reduce RVEDP and may need to be reduced. If the heart is overfull, aggressive diuresis or CRRT may be required to remove adequate volume.[7]

OPTIMIZING RV AFTERLOAD

Reversing conditions that increase pulmonary vascular tone can improve RV output. Alveolar hypoxia, hypercapnia, and acidemia should be normalized. Limit vasopressors with pulmonary vasoconstriction (eg, norepinephrine). Add selective pulmonary vasodilators, such as inhaled nitric oxide. Treat underlying lung pathology, such as appropriate antibiotics for pneumonia or anticoagulation for PE. Of note, empiric anticoagulation is no longer first-line management for PH in the absence of PE.[91]

OPTIMIZING RV CONTRACTILITY

Overstretched myocytes are at a mechanical disadvantage, deranged myocardial metabolism can decrease myocardial contractile force, and impaired coronary perfusion impairs myocyte oxygen delivery, especially during the increased demand of critical illness. It is important to keep systemic arterial pressure close to, but greater than, RV systolic pressure in order to maintain RV perfusion. Adequate RV coronary perfusion requires systemic pressures greater than the RV free wall tension. If vasopressors are required to support adequate systemic pressure, an agent with minimal pulmonary vasoconstricting properties, such as vasopressin, is preferred. The RV is dependent on atrial kick because it generally cannot change its stroke volume. Thus, maintaining sinus rhythm at a rate of at least 80 bpm is key for RV contractility. Cardioversion may be required if sequential pacing is not in place to ensure sinus rhythm. If all the above interventions fail to improve RV contractility, inotropes, such as milrinone or dobutamine, can then be added. The increased contractility and chronotropy can be arrhythmogenic and/or worsen myocyte metabolism and ischemia (especially with dobutamine), and thus, inotropes should be used judiciously. Epinephrine will obviously also increase contractility and heart rate but significantly increases cardiac work and is more recommended in the setting of shock and bradycardia with right heart failure. The final method to treat ongoing cardiogenic shock from right heart failure that fails to respond to

these interventions is mechanical support with venoarterial ECMO.[7]

More advanced therapies also exist in the treatment of PH. Phosphodiesterase type 5 inhibitors, such as sildenafil, prolong the vasodilatory effect of nitric oxide and can be helpful in the treatment of PAH. The typical dose of sildenafil used for treatment of PH is 20 mg orally (or 10 mg intravenously) three times daily. Its onset of action is rapid, with effects seen within 1 hour; the half-life is approximately 3 to 4 hours. In stable patients, when it is used alone or in combination with inhaled nitric oxide, the CO will increase. It is contraindicated in patients receiving systemic nitrates because of the potential for severe hypotension, and its use should be closely monitored in patients with liver dysfunction (since it undergoes hepatic metabolism). Although use of calcium channel blockers in the management of chronic PH is effective for control of heart rate, there are no studies of their use in the critically ill patient with PH. Because of their negative inotropic effects, they may precipitate fatal worsening of RV failure. Other therapies, used in isolation or combination, include endothelin receptor antagonists (eg, bosentan), prostanoids (eg, epoprostenol), and guanylate cyclase stimulants. Initiation of these agents should follow diagnosis of a clear underlying etiology of PH, and they should be initiated in concert with a heart failure team.[91]

Perioperative Risk

Patients at high risk for cardiac events during noncardiac surgery have increased mortality from perioperative arrhythmias and MI. Cardiac death or MI occurs in 1% to 5% of unselected patients undergoing noncardiac surgery and is the most common reason for a preoperative cardiac evaluation. The pathophysiology of MI in the perioperative setting differs from that seen in nonsurgical patients. In the latter, MI usually follows rupture of atherosclerotic plaques in the coronary arteries, leading to platelet aggregation and thrombus formation. MI in the perioperative setting is due to plaque rupture approximately 50% of the time; the remainder of MIs are triggered by myocardial ischemia from decreased myocardial oxygen supply and increased demand in the presence of atherosclerotic coronary artery disease. These demands are often exacerbated by anemia, hypotension, hypoxia, hypertension, and tachycardia. Perioperative shifts in intravascular volume, withdrawal of anesthesia, and postoperative pain are all factors that may contribute to the increased demand of oxygen in the perioperative setting. Postoperative tachycardia, arrhythmias, and MI most commonly occur 3 days after an operation, when fluid shifts are at their greatest.

Until recently, reducing cardiac events has centered on preoperative risk assessment with clinical recommendations and often cancellation or postponement of procedures. Perioperative ischemia has been linked to postoperative MI with a 21-fold increase in risk and, thus, has been a target of medical strategies. Mixed results have been obtained in studies using intraoperative calcium channel blockers and nitroglycerin. However, strong evidence has accumulated regarding β-blockers. Two randomized controlled trials (RCTs) have shown that β-blocker therapy reduces perioperative cardiac complications. Mangano et al[92] performed a randomized, double-blinded, placebo-controlled trial using atenolol in 200 patients with known coronary artery disease and/or risk factors for atherosclerosis who underwent noncardiac surgery. Although acute perioperative mortality did not differ between the two groups at 6 months, eight deaths occurred in the placebo group and none in the atenolol group (P <.001), with the difference being sustained at the 2-year follow-up period.[92] In a landmark 1999 study, patients with clinical risk factors and ischemia demonstrated by dobutamine stress echocardiography who were to undergo major vascular procedures were randomly assigned to bisoprolol or placebo.[93] The study was terminated early when investigators noted that bisoprolol markedly reduced perioperative mortality (17% vs 3.4%; P = .02) and MI (17% vs 0%; P <.001). A 2002 meta-analysis of RCTs concluded that β-blockade may be beneficial in preventing perioperative cardiac morbidity despite the heterogeneity of the trials.[94] Subsequent studies have shown that withdrawal of β-blockers increases the risk of morbidity and mortality and that initiation of β-blockers in the preoperative setting requires titration to blood pressure and heart rate control. Although perioperative β-blockade has been demonstrated to decrease perioperative cardiac morbidity, there also is an increased risk of stroke, bradycardia, hypotension, and (potentially) all causes of death. Furthermore, perioperative metoprolol has been demonstrated in multiple studies to have worse outcomes than those associated with more selective agents, such as bisoprolol or atenolol.[95] Therefore, the 2014 ACC/AHA recommendations are aimed at continuation of β-blocker therapy in the perioperative period for those patients already taking a β-blocker. The initiation of selective β-blockade should be strongly considered in patients who are undergoing high-risk surgery and have a history of coronary artery disease or evidence on stress test of reversible ischemia.[96]

Current Treatment of Acute Myocardial Infarction

The definition of acute MI encompasses a spectrum of clinical entities ranging from non-STEMI (also known as acute coronary syndrome [ACS] or unstable angina) to acute STEMI. The pathophysiology for ACS differs from that of STEMI, as does the treatment. Whereas ACS in nonsurgical patients is commonly due to a partial occlusion of the coronary arteries or transient ischemia (due to plaque rupture with platelet aggregation), in the surgical patient, ACS is more commonly secondary to decreased oxygen supply or increased demand due to anemia, tachycardia, intravascular fluid shifts, hypotension, or arrhythmias. Acute STEMI refers to complete occlusion of the coronary arteries with myocardial injury.[96]

During workup of suspected MI, the interpretation of elevated troponins can be confounded by a variety of factors, including recent chest trauma, chest compressions, and/or demand ischemia, all of which can cause mild elevations in

troponin levels. Significant troponin elevations in the presence of ECG changes should prompt evaluation by a cardiologist. Mild troponin elevation in the absence of ECG changes, especially in the presence of suspected demand ischemia, should be trended with levels checked every 6 hours.[96] However, keep in mind that troponin clearance will be prolonged in patients with renal insufficiency.

Despite marked advances in diagnosis and treatment, there is still an approximately 6.3% in-hospital mortality associated with acute MI. Reduced mortality has been shown to result from adherence to three basic tenets of treatment for acute MI: (1) prompt diagnosis, (2) immediate aspirin and oxygen therapy, and (3) rapid restitution of blood flow to the infarcted myocardium. Whereas prompt diagnosis and institution of aspirin and oxygen therapy are relatively straightforward, the reestablishment of blood flow to the affected myocardium has evolved into two primary therapies: pharmacologic therapy with thrombolytic agents and interventional therapy with PCI.[97] With its ease of administration, early studies on the use of thrombolytic therapy consistently showed decreased mortality and improved myocardial performance compared with placebo; however, thrombolytic therapy has well-documented limitations and contraindications.[98] Intracranial bleeding resulting in death or stroke occurs in 0.6% to 1.4% of patients who receive thrombolytic therapy. Thrombolytic therapy is also associated with a reocclusion rate of 30%, resulting in reinfarction of the affected area within 3 months.[98] In the multiply-injured patient with an increased risk of bleeding, head injury, or multivessel disease, thrombolytic therapy is contraindicated.

PCI has been shown to result in better clinical outcomes when compared to thrombolysis.[99] In addition to improved outcomes, PCI offers direct visualization of the affected anatomy and also provides specific hemodynamic and functional data that can be used to guide further therapy. It can also quickly identify patients who should not undergo reperfusion therapy, which include patients with minimal residual stenosis, spontaneous reperfusion, coronary vasospasm, myocarditis, and aortic dissection involving the ostia.[100] The 2015 ACC Foundation/AHA updated guideline for the management of STEMI still recommends PCI for any patient with an acute STEMI, provided that the procedure can be performed in a timely fashion; this is defined as within 90 minutes at a PCI-capable hospital or within 120 minutes for patients transferred from a non–PCI-capable hospital to a PCI-capable hospital.[96] Unfortunately, PCI is not available in all medical centers, and there is wide variability in the use of PCI even in those centers that are PCI capable.[101] Complications from PCI include major bleeding (7%), vascular complications that require surgical repair (2%), and renal failure (13%). The incidence of renal failure rises with increasing age, volume of contrast material, decreased baseline renal function, and hypovolemia. When comparing thrombolytic therapy to PCI, primary PCI had been found to be more effective in reducing both short- and long-term outcomes, including death. The use of platelet glycoprotein (GP) IIb/IIIa inhibitors (eg, abciximab) or platelet adenosine diphosphate chemoreceptor inhibitors (eg, clopidogrel bisulfate) at the time of PCI reduces the rates of subacute thrombosis and recurrent ischemia and the need for repeat revascularization procedures during the first month after PCI with or without stents. However, clinical outcomes do not differ at 6-month follow-up. Despite the heterogeneity of these trials, including whether or not stents were used and the use of platelet inhibitors versus the various thrombolytic agents, the accumulated data appear to favor primary PCI in the treatment of acute STEMI.[96] The addition of platelet inhibitors should be based on clinical judgment in the multiply-injured trauma patient with ACS. A recent meta-analysis compared immediate versus delayed stent placement after STEMI. Although "delayed" varied from hours to 7 days, outcomes were similar for major cardiac events, bleeding, and target vessel revascularization and improved for delayed stenting in long-term LV ejection fraction. Given the risk of bleeding after major trauma or surgery (especially intracranial or spinal injury), these data may provide increased flexibility in timing of PCI for trauma patients when antiplatelet and GP IIb/IIIa inhibitors are contraindicated.[102]

For patients with non-STEMI, treatment is based on the presence or absence of hemodynamic instability. Hemodynamically unstable patients require treatment with aspirin (ASA) and vasopressors and cardiac catheterization with or without PCI. In patients with no hemodynamic instability and minimal risk factors for recurrence, medical management with ASA, β-blockers, ACE-Is, and statins is first-line therapy. Low-dose anticoagulation may also be considered in patients for whom anticoagulation is not contraindicated.[103] Although the initial results from studies regarding the initiation of statin therapy following ACS were encouraging, a 2014 Cochrane Review of 18 RCTs comparing statins to placebo demonstrated no significant reduction in death or MI after 4 months. There was, however, a reduction in unstable angina with statin administration.[104] For patients with multiple risk factors, cardiac catheterization may be indicated when the patient is able to be systemically anticoagulated.[96]

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Respiratory Failure

Ben E. Biesterveld • Aaron M. Williams • Hasan B. Alam

KEY POINTS

- The Lung Injury Score (LIS) is based on the chest x-ray, hypoxemia, positive end-expiratory pressure (PEEP), and respiratory compliance, and each component is scored from 0 to 4.
- The Berlin definition of acute respiratory distress syndrome (ARDS) (2012) includes timing (within 1 week of a known clinical insult), bilateral opacities on chest x-ray, respiratory failure not explained by cardiac failure or fluid overload, and mild, moderate, or severe hypoxemia.
- A consistent histopathologic feature of ARDS is neutrophil infiltration of the pulmonary microvasculature, interstitium, and alveoli.
- With ARDS, it is not unusual to see greatly diminished lung compliance on the order of 10 to 30 mL/cm H_2O.
- The current standard of care for ARDS is lower tidal volume ventilation and the use of incremental inspired oxygen–PEEP combinations to achieve oxygenation goals.
- PEEP is used in ARDS to allow for adequate oxygen delivery to tissues while reducing the concentration of inspired oxygen to nontoxic (below 0.6) levels.
- The most common type of extracorporeal life support ([ECLS]; or extracorporeal membrane oxygenation [ECMO]) is venovenous, where blood is withdrawn from and returned to the venous system.
- Inhaled nitric oxide (iNO) is a selective pulmonary vasodilator with no systemic side effects.
- Any use of "rescue" steroids for late ARDS must be individualized and, optimally, delivered before disease day 14.
- The mortality of ARDS in several randomized controlled trials over the past two decades has decreased to 20% to 25%.

INTRODUCTION

The maintenance of gas exchange may be tenuous in the injured patient because of dysfunction in three key elements of the respiratory system. First, the central nervous system (CNS) may be impaired, resulting in inadequate respiratory drive, or inability to maintain patent proximal airways. Second, injury to the torso can produce changes in compliance, ineffective respiratory effort, and pain that impact the patient's ability to complete the work of breathing. Third, primary and secondary insults to the lung result in ineffective gas exchange. In practice, it is common for patients to suffer simultaneous insults affecting all three elements. Respiratory failure that relates primarily to injury to the CNS is discussed at length in other chapters and will not be extensively covered here. This chapter will focus on insults that affect work of breathing and gas exchange. The syndrome of acute respiratory distress syndrome (ARDS) and current management are the major focus.

HYPERCAPNIC PULMONARY FAILURE

The neurohormonal response to injury (see Chapter 63) results in a remarkable increase in cellular metabolism. This creates a substantial increase in carbon dioxide (CO_2) production that must be matched by increased elimination from the lungs. While a resting adult eliminates 200 $cm^3/kg/min$ of CO_2, postinjury hypermetabolism results in CO_2 production in the range of 425 $cm^3/kg/min$.[1] Thus, the minute ventilation required to maintain eucapnia may rise from a resting rate of approximately 5 L/min to more than 10 L/min. This represents a 100% increase in the work of breathing simply to meet the metabolic demands.

Additionally, injured patients typically have an increase in physiologic and anatomic dead space regions that do not participate in gas exchange. In a normal adult, the proportion of each breath that is dead space (V_d/V_t) is approximately 0.35. In the intubated, ventilated patient, the V_d/V_t can be accurately determined using Fowler's nitrogen washout technique[2]

or more commonly calculated by a number of techniques including the Bohr-Enghoff method (V_d/V_t = [{$Paco_2$ – mean expired CO_2}/$Paco_2$]).[3] For practical purposes (since mean expired CO_2 is not commonly measured), this is a reflection of the minute volume required to achieve a given $Paco_2$. In ventilated patients with pulmonary failure, the V_d/V_t often exceeds 0.6. This extra dead space is a burden because each breath is less effective at eliminating CO_2, and therefore, minute ventilation requirements in the 12 to 20 L/min range are not uncommon in the postinjury setting.

The above increase in respiratory demand might be met by a healthy adult; however, the injured patient faces several challenges in completing this additional work. CNS dysfunction from injury impairs respiratory drive, as do many medications routinely used for sedation and analgesia. Decreased thoracic compliance from abdominal distension (eg, as part of the abdominal compartment syndrome), chest wall edema, and recumbent positioning increase the energy required to complete a respiratory cycle. Decreased pulmonary compliance from an increase in extravascular lung water and pleural collections (effusions/hemothorax) also contributes. Muscular weakness from impaired energetics (hypothermia, acidosis, coagulopathy, cardiovascular failure, mitochondrial dysfunction, and oxidant stress) or fatigue may be an insurmountable challenge. Finally, pain from torso injuries or operative interventions makes the increased ventilatory demand a substantial burden to the patient.

The net effect of increased demand and diminished capacity to execute the work of breathing is hypercapnic respiratory failure. In the early postinjury period, patients most commonly present with a mixed acid-base disorder where ventilation is inadequate to maintain physiologic pH in the face of a metabolic acidosis. This frequently occurs in the absence of major hypoxia, and therefore, caution is warranted in relying on oximetry alone to assess adequacy of pulmonary function. Particularly in patients with tachypnea, blood gas analysis is the best method to quickly identify patients who are not meeting their ventilatory demands. While efforts to diminish the work of breathing should be routine, most patients with hypercapnic failure require some form of mechanical ventilation to meet their demands.

Two injury patterns that precipitate hypercapnic respiratory failure are worthy of special mention, including spinal cord injury and flail chest/pulmonary contusion. In spinal cord injury, conventional wisdom asserts that lesions below C5 should not result in pulmonary failure, because innervation to the diaphragm remains intact. In practice, however, most complete cord lesions in the cervical and upper thoracic regions frequently result in failure, requiring mechanical ventilation.[4] The genesis is multifactorial, including loss of vital capacity, ineffective cough with retention of secretions due to loss of innervation of intercostal and abdominal musculature (important accessory muscles of respiration), pneumonia in the setting of multiple injuries, associated pulmonary contusion, and aspiration at the time of the initial insult. Furthermore, vagal nerve autonomic dysfunction results in increased secretions, bronchospasm, and pulmonary edema. This patient population requires aggressive mobilization and pulmonary care, because recurrent lobar collapse and pneumonia are certain and lethal if not aggressively prevented.

Early operative stabilization of the spine is advocated because it has been shown to decrease the need for mechanical ventilation and length of stay in the intensive care unit (ICU).[5] Other adjuncts such as noninvasive ventilation, bronchodilators, mucolytics, and percussion should be considered. Flail chest and pulmonary contusion can be thought of as a single entity. This is a challenging injury pattern, because it impacts both the patient's ability to execute the work of breathing (from pain and mechanical instability of the thoracic) and gas exchange (from the pulmonary contusion). Isolated pulmonary contusion rarely requires mechanical ventilation. Since minor contusions are frequently identified by computed tomography (CT), it is important to realize that this tends to take a relatively benign course (see Chapter 28). It is clear that the number of rib fractures is strongly associated with the development of pulmonary failure, ARDS, and mortality, which profoundly impact the elderly population.[6] Early pain control, preferably beginning in the emergency department with regional anesthesia, has been shown to be effective in reducing the impact of multiple rib fractures and should be routinely applied, especially in patients over the age of 65 who have more than three continuous ribs fractured.[7] Admission to a high-volume trauma center, use of patient-controlled analgesia, epidural- or paravertebral catheter–administered analgesia, routine tracheostomy, and an algorithm-driven approach are associated with improved survival.[8,9]

HYPOXIC PULMONARY FAILURE, INCLUDING ACUTE RESPIRATORY DISTRESS SYNDROME

Hypoxic pulmonary failure is a substantial contributor to respiratory failure in the trauma setting. The etiologies are diverse, including aspiration pneumonitis, pneumonia, pulmonary embolism, congestive heart failure, blunt thoracic trauma, and ARDS. Most of these entities are discussed elsewhere; however, ARDS will be the focus of this chapter. Indeed, the mechanisms at work in ARDS share many common features with these other processes that affect the alveolar–capillary interface.

ARDS is a clinical syndrome of inflammatory lung injury that can be thought of as the final common pathway of diverse systemic processes. In the past two decades, major progress has been achieved in defining the underlying ARDS pathophysiology and optimal supportive care. Specific therapies that address the underlying mechanisms responsible for ARDS development remain elusive at present.

DEFINITIONS OF ACUTE RESPIRATORY DISTRESS SYNDROME AND THEIR LIMITATIONS

The recognition of ARDS as a distinct clinical entity resulted from the description by Ashbaugh et al[10] in 1967. Subsequent descriptions include five principal criteria: (1) hypoxemia

TABLE 59-1: Lung Injury Score[a]

Chest radiograph score	
No alveolar consolidation	0
Alveolar consolidation confined to one quadrant	1
Alveolar consolidation confined to two quadrants	2
Alveolar consolidation confined to three quadrants	3
Alveolar consolidation confined to four quadrants	4
Hypoxemia score	
Pao_2/Fio_2 ≥300	0
Pao_2/Fio_2 225–299	1
Pao_2/Fio_2 175–224	2
Pao_2/Fio_2 100–174	3
Pao_2/Fio_2 <100	4
Positive end-expiratory pressure (PEEP) score (when ventilated)	
PEEP ≤5 cm H_2O	0
PEEP 6–8 cm H_2O	1
PEEP 9–11 cm H_2O	2
PEEP 12–14 cm H_2O	3
PEEP ≥15 cm H_2O	4
Respiratory system compliance score (when available)	
Compliance ≥80 mL/cm H_2O	0
Compliance 60–79 mL/cm H_2O	1
Compliance 40–59 mL/cm H_2O	2
Compliance 20–39 mL/cm H_2O	3
Compliance ≤19 mL/cm H_2O	4

[a]The final value is obtained by dividing the aggregate sum by the number of components that were used: no lung injury, 0; mild to moderate lung injury, 0.1–2.5; severe lung injury (acute respiratory distress syndrome), 2.5.

Source: Murray JF, Matthay MA, Luce JM, Flick MR. Pulmonary perspectives: an expanded definition of the adult respiratory distress syndrome. *Am Rev Respir Dis.* 1988;138:720. Reprinted with permission of the American Thoracic Society. Copyright © 2020 American Thoracic Society. All rights reserved. The American Review of Respiratory Disease is an official journal of the American Thoracic Society.

refractory to oxygen administration; (2) diffuse, bilateral infiltrates on chest radiograph; (3) low static lung compliance; (4) absence of congestive heart failure; and (5) presence of an appropriate at-risk diagnosis.

Since the initial description of ARDS 50 years ago, the optimal definition of ARDS remains a controversial subject. The Lung Injury Score (LIS), proposed by Murray et al[11] in 1988, has been a commonly used measure of lung injury severity in previous clinical studies. The LIS is based on four components: chest radiograph, hypoxemia, positive end-expiratory pressure (PEEP), and respiratory compliance (Table 59-1).[11] Each component is scored from 0 to 4. The LIS is calculated by summing the scores of the available components and dividing by the number of components used. ARDS (or severe lung injury) is defined as an LIS greater than 2.5. Zero represents no lung injury, and 0.1 to 2.5 represents mild to moderate lung injury.

The first standard definitions were developed in 1994 by the Consensus Conference of American and European Investigators (AECC). Acute lung injury (ALI) was defined as respiratory failure of acute onset with a Pao_2:Fio_2 (P:F) ratio

TABLE 59-2: American–European Consensus Conference (AECC) Definitions of Acute Lung Injury and Acute Respiratory Distress Syndrome

Acute lung injury criteria

Timing: acute onset

Oxygenation: Pao_2/Fio_2 ≤300 mm Hg (regardless of positive end-expiratory pressure)

Chest radiograph: bilateral infiltrates on anteroposterior chest radiograph

Pulmonary artery occlusion pressure ≤18 mm Hg or no clinical evidence of left atrial hypertension

Acute respiratory distress syndrome criteria

Same as acute lung injury except:

Oxygenation: Pao_2/Fio_2 ≤200 mm Hg (regardless of positive end-expiratory pressure)

of less than or equal to 300 mm Hg (regardless of the level of PEEP), bilateral infiltrates on frontal chest radiograph, and a pulmonary capillary wedge pressure less than or equal to 18 mm Hg (if measured) or no evidence of left atrial hypertension. ARDS was defined identically except for a lower limiting value of less than 200 mm Hg for P:F (Table 59-2).[12] The AECC definition of ALI/ARDS has been used in many of the ARDS Network clinical trials; however, debate still exists as to the usefulness of the AECC diagnostic criteria. Criticism of the AECC definition includes lack of including criteria about timing of ARDS development from injury, not including how ventilator settings could affect Pao_2/Fio_2, subjectivity of reading chest radiographs, and challenges to exclude volume overload as the cause for respiratory failure.[13]

An alternative definition of ARDS developed by a consensus panel of senior investigators using the Delphi method appears to be more specific than AECC criteria.[14] Briefly, the authors include PEEP in the consideration of hypoxia and require either the absence of congestive heart failure or the presence of a recognized risk factor for ARDS. The degree of hypoxia required is a P:F less than 200 mm Hg with PEEP greater than or equal to 10 cm H_2O; therefore, it excludes patients who are hypoxic purely because of derecruitment or suboptimal PEEP (Table 59-3). By allowing patients with high filling pressures in the presence of a recognized ARDS risk factor, the definition recognizes the prevalence of high left atrial pressure during the course of ARDS and includes patients with concomitant congestive heart failure and ARDS.

TABLE 59-3: The Delphi Definition of Acute Respiratory Distress Syndrome (ARDS)

Timing: acute onset

Oxygenation: Pao_2/Fio_2 ≤200 mm Hg with PEEP ≥10 cm H_2O

Chest radiograph: bilateral infiltrates

Absence of CHF or presence of recognized risk factor for ARDS

CHF, congestive heart failure; PEEP, positive end-expiratory pressure.

TABLE 59-4: The Berlin Definition of Acute Respiratory Distress Syndrome

Timing: Within 1 week of a known clinical insult or new or worsening respiratory symptoms

Chest imaging: Bilateral opacities—not fully explained by effusions, lobar/lung collapse, or nodules

Origin of edema: Respiratory failure not fully explained by cardiac failure or fluid overload

Oxygenation

Mild: 200 mm Hg < Pao_2/Fio_2 ≤ 300 mm Hg with PEEP or CPAP ≥5 cm H_2O

Moderate: 100 mm Hg < Pao_2/Fio_2 ≤ 200 mm Hg with PEEP or CPAP ≥5 cm H_2O

Severe: Pao_2/Fio_2 <100 mm Hg with PEEP or CPAP ≥5 cm H_2O

CPAP, continuous positive airway pressure; PEEP, positive end-expiratory pressure.

TABLE 59-5: Risk Factors for Acute Respiratory Distress Syndrome (ARDS)

Risk factor	Frequency of ARDS (%)
Direct	
Aspiration	12–36
Pneumonia	12–31
Pulmonary contusion	5–22
Toxic inhalation	2–17
Indirect	
Sepsis syndrome	11–80
Multiple transfusions	5–36
Multiple fractures	2–21
Pancreatitis	7–18
Disseminated intravascular coagulation	23

The definition of ARDS was most recently revised by a panel of specialists in a joint effort of the European Society of Intensive Care Medicine, the American Thoracic Society, and the Society of Critical Care Medicine. Their collective aim was to address the current limitations of the AECC definition and explore other defining ARDS variables. The panel agreed to maintain the previous conceptual model, which defined ARDS as a syndrome of acute, diffuse lung inflammation, with edema because of increased permeability of the alveolar-capillary membrane, and clinically characterized by decreased oxygenation because of increased venous admixture, decreased lung compliance, increased physiologic dead space, and bilateral radiographic opacities. The newest and most widely accepted ARDS criteria, also referred to as the Berlin definition of ARDS, are provided in Table 59-4.[15]

EPIDEMIOLOGY OF ACUTE RESPIRATORY DISTRESS SYNDROME

The estimated incidence of ARDS in population-based studies is on the order of 40 cases per 100,000 person-years.[16-18] In the United States, this represents about 200,000 cases per year, and about 15% of ICU admissions. The LUNG SAFE international trial in 50 countries found that ARDS occurred in 10% of ICU admissions and mortality was 35%, 40%, and 46% for patients with mild, moderate, and severe ARDS.[19]

RISK FACTORS FOR ACUTE RESPIRATORY DISTRESS SYNDROME

Clinical Risk Factors

Clinical risk factors for ARDS can be broadly categorized into direct and indirect pulmonary factors (Table 59-5). Direct pulmonary factors are those primarily associated with local pulmonary parenchymal injury and include inhalation injury, pulmonary contusion, aspiration, and pulmonary infection. Indirect pulmonary factors are those thought to be associated with systemic inflammation and resultant lung injury. These include trauma, shock, burn injury, extrapulmonary sepsis, transfusion of blood products, multiple long bone fractures, pancreatitis, and others.[16,20]

The King County Lung Injury Project (KCLIP) identified pulmonary and extrapulmonary sepsis as the most common risk factors, with 46% and 33% of ARDS cases, respectively. Following sepsis in descending order of frequency were aspiration (11%), trauma (7%), transfusion (3%), drug overdose (3%), and pancreatitis (3%). The highest mortality rates were noted in aspiration-associated (44%) and pulmonary sepsis–associated lung injury groups (41%), with the lowest rate in trauma-associated lung injury (24%).[18]

Demographic Risk Factors

Several demographic factors have been identified as risk modifiers for development of ARDS. The incidence of ARDS increases with age, and older patients have consistently higher mortality rates, although it is unclear whether this simply represents diminished physiologic reserve in older patients.[21,22] Alcohol abuse, active cigarette smoking, and exposure to secondhand smoke have also been linked to ARDS incidence.[23,24] Higher body mass index has been associated with increased incidence of ARDS,[25] with data suggesting a decrease in mortality.[26]

Treatment Risk Factors

It is well established that high tidal volume ventilation increases mortality for patients with established ARDS.[27] Several studies have shown that increased tidal volume is an independent risk factor for the subsequent development of ARDS in a dose-responsive manner.[28,29] This phenomenon, termed *ventilator-induced lung injury* (VILI), has been a target to decrease the development of ARDS.

TABLE 59-6: National Heart, Lung, and Blood Institute Consensus Conference Definition of Transfusion-Related Acute Lung Injury (ALI)

Onset of signs or symptoms ≤6 h after transfusion.
No ALI may be present prior to transfusion.
Alternative ALI risk factors may be present, but the patients' clinical course should determine whether the ALI is mechanistically related to the transfusion.

Transfusion of blood products is a major iatrogenic risk factor for ARDS. Multiple studies have shown increased risk of ARDS in ICU patients receiving packed red blood cells, fresh frozen plasma, and/or platelet transfusions,[25,30] a phenomenon termed *transfusion-related acute lung injury* (TRALI). In addition to ARDS, the National Heart, Lung, and Blood Institute definition of TRALI requires additional criteria (Table 59-6): (1) A patient must develop ARDS during or within 6 hours of transfusion, and (2) no ARDS may be present before transfusion. (3) Alternative ARDS risk factors may be present, but the patient's clinical course should determine whether the ARDS is mechanistically related to the transfusion.[31]

PATHOLOGY OF ACUTE RESPIRATORY DISTRESS SYNDROME

The current paradigm of systemic inflammation leading to ARDS posits that a variety of insults, both infectious and noninfectious, can result in an unbridled hyperinflammatory response. This leads to organ injury from indiscriminate activation of effector cells that subsequently release oxidants, proteinases, and other potentially injuring compounds. If the initial insult is severe enough, early organ dysfunction results ("one-hit" or single-insult model). More often, a less severe insult results in a systemic inflammatory response that is not by itself injurious. These patients appear, however, to be primed such that they have an exaggerated response to a second insult, which leads to an augmented/amplified systemic inflammatory response and multiple organ dysfunction ("two-hit" or sequential insult model; see Chapter 63).[32]

Inflammatory models provide a unifying hypothesis for ARDS and multiorgan failure (MOF); however, the precise relationship between ARDS and MOF remains to be further refined. MOF is a frequent occurrence and the most common cause of mortality in patients with ARDS.[33] It is also now clear that ventilator strategies that inadvertently promote lung injury may produce systemic inflammation, perhaps leading to other organ failures.[34]

Inflammatory lung injury leads to the pathologic lesion of diffuse alveolar damage. This prototypic lesion of ARDS is at the alveolar–capillary interface, which results in epithelial and endothelial damage as well as high-permeability pulmonary edema. The histologic appearance of this lesion can be divided into three overlapping phases as follows: (1) the exudative phase, with edema and hemorrhage; (2) the proliferative phase, with organization and repair; and (3) the fibrotic phase.[35]

The exudative phase generally encompasses the first 3 to 5 days but may last up to a week. The initial histologic changes include interstitial edema, proteinaceous alveolar edema, and intra-alveolar hemorrhage. The exudative phase is characterized by the appearance of hyaline membranes, which are composed of fibrin, immunoglobulin, and complement. Electron microscopy reveals endothelial injury with cell swelling, widening intercellular junctions, and increased pinocytotic vesicles. In addition, there is disruption of the basement membrane.

The alveolar epithelium usually exhibits extensive loss of type I cells, which slough and leave a denuded basement membrane. While some loss may be from necrosis, it appears that apoptosis contributes substantially. Activation of matrix metalloproteinases, toll-like receptors, and oxidative stress pathways initiates programmed cell death in these cells. Demonstration of soluble Fas ligand in bronchoalveolar lavage (BAL) fluid early in ARDS supports this concept.[36] Loss of the alveolar epithelial barrier results in alveolar edema, as the remaining cells are unable to drive sodium from the alveolar into the interstitial compartment.

During the proliferative phase, typically occupying the second 2 weeks after the onset of respiratory failure, type II cells divide and cover the denuded basement membrane along the alveolar wall. This process may be seen as early as 3 days after the onset of clinical ARDS. Type II cells are also capable of differentiating into type I epithelial cells. Fibroblasts and myofibroblasts proliferate and migrate into the alveolar space in the third phase. Fibroblasts change the alveolar exudate into granulation tissue, which subsequently organizes and forms dense fibrous tissue. Eventually, epithelial cells cover the granulation tissue. This whole process is called fibrosis by accretion and is important in lung remodeling. Septal collagen deposition by fibroblasts and "collapse induration" also contribute to fibrous remodeling of the lung in ARDS.

Many patients with ARDS recover lung function 3 to 4 weeks after initial injury; however, some enter a fibrotic phase characterized by thickened, collagenous connective tissue in the alveolar septa and walls. Pulmonary vascular changes occur as well, with intimal thickening and medial hypertrophy of the pulmonary arterioles. Complete obliteration of portions of the pulmonary vascular bed is the end result. Clinical sequelae include an increased risk of pneumothorax, decreased lung compliance, and increased pulmonary dead space. This may lead to increased long-term support on mechanical ventilators and/or need for supplemental oxygen.

PATHOGENESIS OF ACUTE RESPIRATORY DISTRESS SYNDROME

Lung injury in ARDS involves components of inflammation, coagulation, vasomotor tone, and other systems (see Chapter 63). The pivotal cellular mediators appear to be

leukocytes, with both local and humoral mediators orchestrating their function. Activation of these leukocytes results in release or activation of multiple cytokines, chemokines, oxidants, and proteases that result in the final common pathway of tissue injury in ARDS.

Neutrophils

A consistent histopathologic feature of ARDS is neutrophil infiltration of the pulmonary microvasculature, interstitium, and alveoli. Neutrophil infiltration may result in lung injury due to neutrophil-mediated release of reactive oxidant species (ROS) and resulting oxidant stress.[37] Furthermore, neutrophils are an important source of proinflammatory cytokines. Persistence of neutrophils in serial BAL fluid samples from patients with ARDS suggests unbridled inflammation and portends poor prognosis. In animal models, neutrophil depletion prior to an insult markedly attenuates resulting lung injury.[38] Evidence from clinical studies supporting excess ROS in ARDS include findings of increased hydrogen peroxide in exhaled breath,[39] decreased levels of glutathione in lung lavage fluids,[40] and increased lipid peroxidation products in plasma.[41] Neutrophils obtained from trauma patients with ARDS also are able to more effectively break down the endothelial layer.[42]

Macrophages

The lung contains large numbers of fixed tissue macrophages that are a critical component of the inflammatory response in ALI. Activated macrophages can cause tissue injury by releasing the same toxic mediators as neutrophils (ROS and proteases). Probably more important is the macrophage capability to synthesize multiple proinflammatory mediators, such as complement fragments, cytokines, and chemokines, leading to neutrophil and monocyte recruitment. Thus, macrophages are thought to have a major role in amplifying and perpetuating the inflammatory response.[43]

Endothelium

The pulmonary endothelium is not a passive bystander in the pathogenesis of ARDS, but actively participates in initiating and perpetuating the inflammatory response. Endothelial cells increase the expression of adhesion molecules (ICAM-1, ICAM-3, and E-selectin) following exposure to an activating stimulus. These ligands serve as tethering and signaling molecules by binding to their associated leukocyte membrane proteins. Thus, the endothelial cell actively coordinates trafficking, firm adhesion, and transmigration. In the setting of systemic inflammation, inappropriate endothelial cell activation may lead to indiscriminate leukocyte recruitment and parenchymal inflammation. Moreover, endothelial cells produce and release vasoactive substances, such as prostacyclin, nitric oxide (NO), and endothelins. These substances may mediate much of the pulmonary vascular dysfunction characteristic of ARDS. The activated endothelium expresses procoagulant activity, which also contributes to intravascular coagulation and microvascular dysfunction.[35] Thrombin, in turn, has proinflammatory effects on leukocytes. Endothelial injury, then, may be both a proximate cause and a marker for ALI.

Mediators and Markers of Acute Respiratory Distress Syndrome

COMPLEMENT

Systemic complement activation secondary to trauma or sepsis is considered a major early factor in ARDS.[44] C5a, a product of complement activation, is a powerful neutrophil chemoattractant. Moreover, C5a induces neutrophil aggregation and activation, leading to pulmonary neutrophil sequestration and lung injury. Clinically, BAL proteomic analysis reveals markedly differential expression of the complement pathway in ARDS survivors versus nonsurvivors.[45]

ENDOTHELIAL INJURY

The pulmonary endothelium is recognized as an active participant in the development of ALI. As such, markers of endothelial activation or injury have been investigated as predictors of the development of ARDS.[35] von Willebrand factor antigen (vWF:Ag) has been studied fairly extensively as a marker of endothelial dysfunction. vWF:Ag is synthesized largely by vascular endothelial cells and has been shown to be a sensitive marker of endothelial injury or activation and predictive for the development of ALI in injured burn patients.[46]

Following activation, endothelial expression of adhesion molecules, including ICAM-1, VCAM-1, E-selectin, P-selectin, and Ang-2, is upregulated. These compounds are susceptible to proteolytic cleavage and may exist in the circulation in a soluble form. Therefore, these molecules represent a measure of endothelial activation or damage. Ang-2 is released by activated endothelial cells, increases vascular permeability and sensitizes the cell to inflammatory stimuli, and has been shown to be predictive of ARDS mortality.[47] Elevated ICAM-1 levels have been found in severely injured patients who subsequently developed MOF.[48] In contrast, plasma levels of soluble E- and P-selectins measured at admission were not useful in predicting lung injury, and levels of L-selectin were downregulated in those who would go on to develop ARDS.[47]

Markers of leukocyte activation have also been measured in plasma and BAL fluid of patients in an attempt to predict development of ARDS. Gordon et al[49] noted markedly elevated plasma elastase levels very early after multisystem trauma. Subsequent studies supported a causative role for neutrophil elastase in ARDS and have led to the clinical development of human elastase inhibitor (see the section "Pharmacologic Therapy of Acute Respiratory Distress Syndrome").[50]

CYTOKINES

Circulating and BAL fluid levels of cytokines are also inherently attractive as predictors of ARDS; whether they are markers or mediators remains an important question. Direct measurement of BAL or pulmonary edema fluid in ARDS patients has shown that both fluids consistently contain elevated levels of tumor necrosis factor (TNF)-α and interleukin (IL)-1; additionally, high levels of TNF-α and IL-1 in plasma and in BAL have been associated with an increased risk of death in patients with ALI and ARDS.[35] Persistently elevated IL-6 levels have also been associated with an increased risk of death in ARDS patients. In the ARDS Network (ARDSNet) trial, there was a significantly steeper fall in IL-6 levels over the initial 72 hours in the lung-protective treatment group with decreased mortality compared to the higher-tidal-volume treatment group. Of all the inflammatory cytokines, IL-8 in lavage fluid has been shown to be the strongest predictor of ARDS mortality. IL-10 is a potent inhibitor of proinflammatory molecules including TNF-α and IL-1. Lower levels of IL-10 in blood and lung lavage fluid have been associated with development of ARDS.[51]

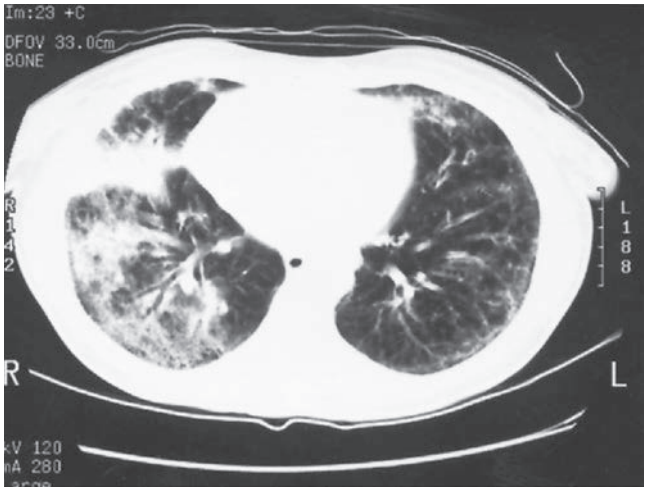
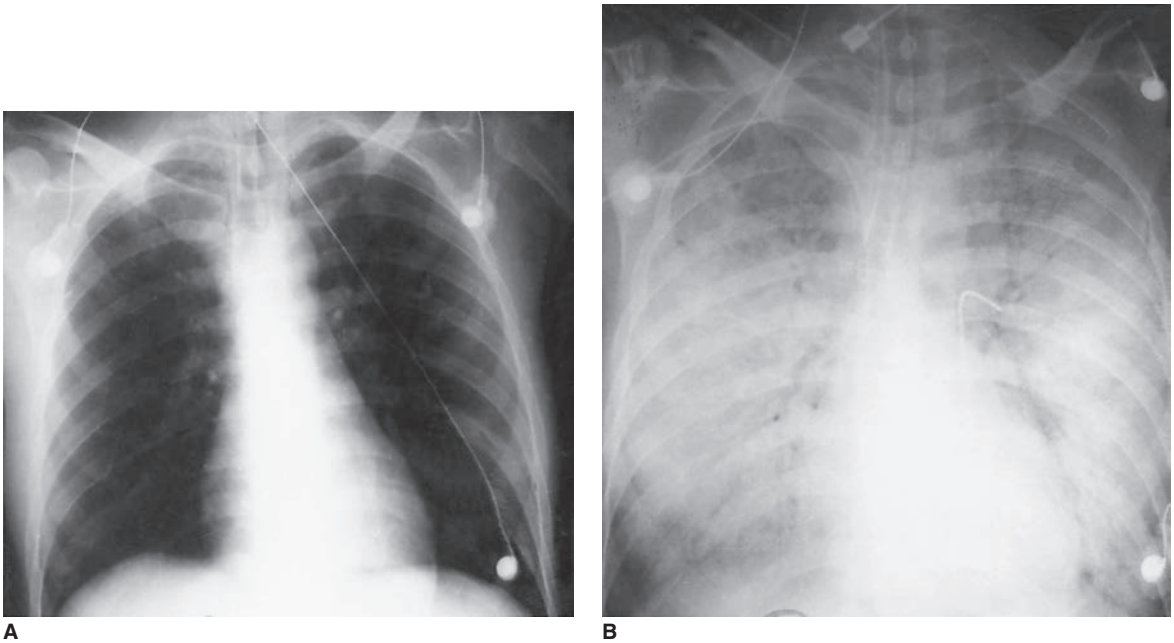


FIGURE 59-2 Chest computed tomography scan of a patient with acute respiratory distress syndrome shows marked inhomogeneity of the process with areas of dense consolidation and essentially normal intervening pulmonary parenchyma.

PATHOPHYSIOLOGY OF ACUTE RESPIRATORY DISTRESS SYNDROME

ARDS is characterized by diffuse, patchy, panlobular pulmonary infiltrates on plain chest radiograph (Fig. 59-1). CT of the chest will demonstrate that the parenchymal changes are heterogenous with the dependent lung regions most affected (Fig. 59-2). The inhomogeneous distribution of parenchymal densities led to the concept of a three-compartment model of the lung in ARDS.[52] One compartment is substantially normal (healthy zone), one is fully diseased without any possibility of recruitment (diseased zone), and, finally, the third compartment is composed of collapsed alveoli potentially recruitable with increasing pressure (recruitable zone). Increased airway pressure is necessary to recruit collapsed alveoli. In the 1990s, however, it was already recognized that in the heterogeneously injured lung, VILI may be damaging



A B

FIGURE 59-1 **(A)** Normal admission chest x-ray in a 27-year-old trauma patient with multiple lower extremity fractures. **(B)** Chest x-ray from the same patient following onset of respiratory insufficiency. Note the bilateral, dense pulmonary infiltrates consistent with severe acute respiratory distress syndrome.

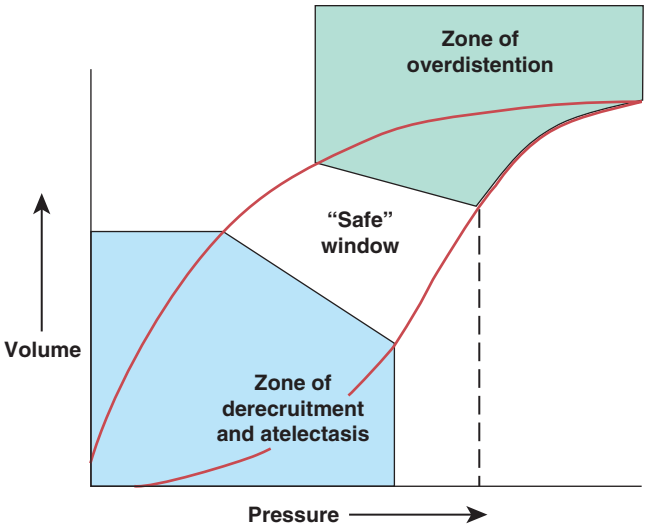


FIGURE 59-3 Idealized static pressure–volume curve of the lungs in acute respiratory distress syndrome. At low pressures and volumes, derecruitment, atelectasis, and lung injury from repetitive opening and closing of gas exchange units are a concern. At high pressures and volumes, overdistention, increased shunt, and lung injury from excessive stretch predominate. In between these two extremes is the optimal zone, where lung stays recruited but is not overstretched.

to the healthy zone. This injury is thought to be responsible for severe protracted ARDS, as well as perpetuation of systemic inflammation and MOF.[53]

A standard method used to describe the mechanical properties of the lung is to determine the static pressure–volume (PV) curve during inflation and deflation (Fig. 59-3). In early ARDS, the lower inflection point represents the airway pressure at which considerable alveolar recruitment occurs. The upper inflection point is where near-maximal inflation occurs such that further increases in airway pressure result in alveolar overdistension and little change in volume. In practice, however, inflection points in individual patients have been difficult to consistently measure.

Pulmonary Edema

Increased pulmonary capillary permeability is a consistent feature. Increased permeability promotes alveolar flooding with protein-rich edema fluid, as well as release of proteins generally confined to the lung into the systemic circulation. In ARDS, the pulmonary epithelium is injured, leading to altered fluid transport. Multiple studies have documented elevated total protein concentrations in BAL fluid from patients with established ARDS.[54] One study observed higher BAL fluid protein concentrations in those who died, suggesting that more severe lung injury is associated with greater endothelial and epithelial permeability.[52]

Gas Exchange

Hypoxemia in ARDS results from ventilation/perfusion mismatching and intrapulmonary shunting of blood flow. Shunting results from blood that passes through the systemic venous to pulmonary arterial system without going through the normal gas exchange units in the lung (ie, right–left shunt). Normally, the shunt fraction is less than 5%; however, in ARDS, it may exceed 25%. Because blood flowing through a shunt is not exposed to alveoli participating in gas exchange, supplemental oxygen is ineffective in increasing arterial oxygen concentration. Other techniques designed to restore ventilation to diseased lung regions, such as PEEP or continuous positive airway pressure (CPAP), are thus necessary to improve oxygenation. Hypoxic pulmonary vasoconstriction is a protective mechanism that limits perfusion to poorly ventilated alveoli and minimizes shunting. In ARDS, hypoxic pulmonary constriction is impaired, resulting in greater intrapulmonary shunt. Multiple factors may contribute to loss of hypoxic pulmonary vasoconstriction in lung injury, including local prostaglandin or NO production.[55]

In early ARDS, most patients are tachypneic from hypoxemia and are secondarily hypocapneic. With disease progression, hypercapnia may become a prominent feature because of physiologic dead space and CO_2 production. Increased dead space ventilation is invariably present. As discussed earlier, the normal dead space–to–tidal volume ratio is approximately 35%, but in ARDS, it can approach 60%. Exacerbating the ability to ventilate is the surfactant dysfunction and terminal bronchiolar spasm contribution to loss of ventilated alveoli.

Pulmonary Mechanics

Lung compliance is defined as the change in lung volume per change in transpulmonary pressure. Normal lung compliance in a mechanically ventilated patient ranges from 60 to 80 mL/cm H_2O. With ARDS, it is not unusual to see greatly diminished lung compliance on the order of 10 to 30 mL/cm H_2O. Initially, reduced compliance is the result of interstitial and alveolar edema with alveolar flooding. In later ARDS, interstitial fibrosis and parenchymal loss further reduce pulmonary compliance.

Hemodynamics

Pulmonary hypertension is a frequent finding in patients with ARDS and can lead to increased interstitial pulmonary edema, right ventricular dysfunction, and impaired cardiac output. When severe, pulmonary hypertension has been observed to be a marker of poor outcome. The etiology of pulmonary hypertension in ARDS is multifactorial. Early in ARDS, the predominant mechanism is most likely impaired vasorelaxation related to hypoxemia, acidosis, and vasoactive mediators, in concert with obstruction from microvascular coagulation. In late ARDS, fibrosis and obliteration of the pulmonary vascular bed are most likely responsible. Wiedemann et al[56] noted markedly depressed right ventricular function in nonsurvivors of ARDS. The authors related this to reduced myocardial contractility and not to pulmonary hypertension.

They also noted that oxygen delivery was more related to cardiac performance than to pulmonary gas exchange.

PRESENTATION OF ACUTE RESPIRATORY DISTRESS SYNDROME

The diagnosis of ARDS is based on the criteria used to define the syndrome and has historically been divided into four clinical phases with radiographic, clinical, physiologic, and pathologic correlates. In the initial phase, dyspnea and tachypnea are evident, with a remarkably normal chest examination (radiographically and clinically). Arterial oxygen saturation is preserved, and hypocapnia from hyperventilation is frequently noted.

The second phase quickly follows (12–24 hours), with physiologic and pathologic evidence of lung injury. The chest x-ray now shows bilateral patchy alveolar infiltrates with hypoxemia evident on arterial blood gas determination. If ARDS persists and progresses, the third clinical phase becomes evident. Acute respiratory failure necessitates mechanical ventilation with increasing inspired oxygen concentrations. There is an increase in physiologic dead space and rising minute ventilation. Patients at this point may develop sepsis syndrome with a hyperdynamic hemodynamic pattern. The radiographic picture worsens with more diffuse infiltrates, consolidation, and air bronchograms.

Without resolution, progressive pulmonary failure and fibrosis characterize the fourth phase. Hypercapnia may worsen and become more difficult to control. MOF commonly develops and is the most common cause of death.

MANAGEMENT OF ACUTE RESPIRATORY DISTRESS SYNDROME

Because there is no proven specific treatment for ARDS, therapy primarily involves supportive measures to maintain life while the lung injury resolves. Such measures include identifying and treating predisposing conditions, mechanical ventilatory support with oxygen, nutritional support, nonpulmonary organ support, and hemodynamic monitoring as necessary. Attention to detail is necessary to avoid nosocomial infection and iatrogenic complications.

Fluid Management and Hemodynamic Support

One of the hallmark pathologic changes in ARDS is increased endothelial and epithelial permeability, resulting in extravasation of fluid and plasma elements, including albumin, in the interstitium and alveolar space. This leads to hypoxemia, atelectasis, decreased lung compliance, and increased pulmonary artery pressures.[55] Fluid management is one of the most important measures shown to impact ARDS. Based on the known pathophysiologic mechanisms, current management is in favor of limiting the forces that favor fluid filtration from the capillaries or augmenting those factors implicated in fluid reabsorption, which can limit fluid accumulation in the lung.

One of the most important trials in the treatment of ARDS has been the Fluid and Catheter Treatment (FACTT) trial.[56] This study randomized 1000 patients to liberal versus conservative fluid management, based on targeted central venous pressure or pulmonary capillary wedge pressure. There was no significant 60-day mortality difference between the groups, but patients in the conservative strategy group had significantly more days alive, days free of mechanical ventilation, and shorter ICU stay. Importantly, there was no increase in organ failures in these patients at 7 and 28 days.

Because ARDS is a common complication in patients with sepsis, proper fluid management is essential for optimal outcomes. However, the correct assessment of fluid status in ICU patients is still a challenge. A tool in estimating lung fluid balance is extravascular lung water (EVLW), which is calculated using the single thermodilution method.[57] Traditionally, EVLW has been indexed to actual body weight (mL/kg); however, a recent observational cohort study of patients with ARDS has shown that baseline EVLW when indexed to predicted body weight is a better prognostic indicator of mortality, after adjusting for severity of illness and other important factors, than EVLW indexed to actual body weight.[58] EVLW may also be a tool to predict those at risk for developing ARDS in postoperative patients.[59]

With respect to colloids, there is no evidence that their use for acute resuscitation improves outcome. A study in hypoproteinemic patients suggests that gas exchange can be improved in late ALI by using colloid in concert with diuretics to mobilize interstitial fluid and promote diuresis.[58] A 2006 randomized controlled trial (RCT) suggested that critically ill patients with profound hypoproteinemia have fewer organ failures (by Sequential Organ Failure Assessment score) when given colloid therapy.[60] Short-term improvement in physiology is not, however, accompanied by an improved outcome in these investigations. The very large Australian SAFE study revealed no difference in mortality and other significant clinical end points for patients resuscitated with albumin, as compared to those resuscitated with normal saline; however, the patient population in this study was heterogeneous.[61] Further large randomized studies of colloid use only in ARDS patients are needed.

Mechanical Ventilation

Most patients with ARDS require endotracheal intubation for mechanical ventilation. This technique reduces shunt physiology and administers high concentrations of oxygen. Emergent intubation is associated with significantly higher morbidity and mortality. Accordingly, early elective intubation should be considered in all patients with deteriorating gas exchange or mental status. Noninvasive positive-pressure mask ventilation should be used very cautiously, if at all, for hypoxemic respiratory failure as several studies have shown a higher mortality and increased risk of intubation-related complications.[62,63]

Respiratory support with a mechanical ventilator is a cornerstone in the supportive management of patients with or at risk for ARDS. The goals of mechanical ventilation in ARDS are to maintain oxygenation while avoiding oxygen toxicity. Typically, this involves maintaining oxygen saturation in the range of 85% to 90% and decreasing F_{IO_2} to less than 65% in the first 24 to 48 hours. It has long been recognized that the ventilator settings can contribute to VILI by worsening lung injury and causing barotrauma (eg, mediastinal emphysema, pneumothorax). This recognition supported many early experimental studies in animals showing that high tidal volume (V_T) ventilation caused injury morphologically similar to ARDS in humans, and even multiorgan system failure in otherwise healthy animals, which was associated with release of inflammatory cells and proinflammatory cytokines.[64,65] Early RCTs examining low V_T strategies were inconclusive with limited sample size and lack of statistical power. The hallmark clinical study by the ARDSNet was the first to conclusively show the benefit of low V_T ventilation compared to traditional strategies.[27] In this trial, volume-controlled, continuous mandatory ventilation was used, and V_T was set based on ideal body weight (IBW). In the group treated with traditional V_T, the target V_T was 12 mL/kg IBW. In the group treated with lower V_T, the target V_T was 6 mL/kg IBW if the plateau pressure did not exceed 30 cm H_2O and 4 mL/kg if plateau pressure was greater than or equal to 30 cm H_2O. For patients with severe dyspnea, the V_T could be increased to 8 mL/kg IBW. The trial was stopped after the enrollment of 861 patients because mortality was significantly lower in the group treated with lower V_T than in the group treated with traditional V_T (31% vs 39.8%). Importantly, plasma IL-6 levels and the number of organ failure–free days were lower among the low V_T group.

Whereas previous studies employing low V_T allowed permissive hypercapnia and acidosis to achieve the protective ventilation goals of low V_T and inspiratory airway pressure, the ARDSNet study allowed increases in respiratory rate as the V_T was decreased and administration of bicarbonate to correct acidosis. This may explain the positive outcome in this study compared to earlier studies that had failed to demonstrate a benefit. However, the ARDSNet trial was criticized for relying on strict ventilator protocols for the higher tidal volume group.

With the results of the ARDSNet trial, the current standard of care now includes a lower tidal volume ventilatory strategy ("ARDSNet lung-protective strategy") and the use of incremental F_{IO_2}-PEEP combinations to achieve oxygenation goals.[53]

Permissive Hypercapnia

Controlled hypoventilation (relative to CO_2 production) with increased P_{aCO_2} is referred to as permissive hypercapnia. The widespread acceptance of the ARDSNet lung-protective strategy has led to a shift in clinical paradigms regarding hypercapnic acidosis (HCA)—from intolerance to acceptance. In fact, not only is it accepted that permissive HCA is well tolerated, there are emerging data regarding the benefits of permissive hypercapnia in experimental models of lung injury.

HCA has multiple physiologic effects on different organs, particularly the pulmonary, cardiovascular, and cerebrovascular systems. Because CO_2 diffuses freely across cell membranes, an increase in extracellular P_{CO_2} will result in intracellular acidosis. Gradual increases in P_{aCO_2} are usually well tolerated, provided renal compensation is adequate (see Chapter 61), and severe acidosis (pH <7.1) usually does not occur. Three mechanisms are responsible for this regulation: (1) physiochemical buffering, mainly due to proteins and phosphates; (2) reduced intracellular generation of protons; and (3) changes in transmembrane ion exchange. Physiochemical buffering is immediate, while the other mechanisms require 1 to 3 hours. These regulatory mechanisms are remarkably powerful and efficient. As a result, normoxic HCA has only limited potential for resulting in intracellular acidosis and is generally well tolerated.

Moderate HCA enhances tissue perfusion and oxygenation through multiple mechanisms. In both normal and diseased lungs, there is evidence that HCA reduces ventilation/perfusion heterogeneity, likely through increasing compliance and directing ventilation to underventilated lung regions with higher alveolar P_{CO_2}. The overall hemodynamic response to acute HCA in human experiments is increased cardiac output, heart rate, and stroke volume, with decreased systemic vascular resistance.[66]

HCA has multiple effects on the CNS. CO_2 is an important regulator of cerebrovascular tone. It has long been recognized that hypercarbia increases cerebral blood flow and intracranial pressure secondary to diminished vascular tone and enlargement of cerebral blood volume.[67] Indeed, traditional management of traumatic brain injury (TBI) historically included hypocapnia to reduce cerebral blood volume and intracranial pressure. Accumulating evidence, however, has shown that sustained hypocapnia reduces cerebral oxygen supply and increases brain ischemia, vasospasm risk, and seizures.[66] Studies have shown that prehospital hypocapnia worsens outcomes after severe TBI.[68,69]

The direct contribution of HCA to the protective effects observed with lung-protective ventilator strategies remains an area of debate. A secondary analysis of ARDSNet data, using multivariate logistic regression and controlling for comorbidities and severity of lung injury, showed that HCA on day 1 was associated with reduced mortality in patients ventilated with 12 mL/kg but not in patients ventilated with 6 mL/kg.[70] Recent studies have introduced the concept of *therapeutic hypercapnia*, whereby deliberately elevated P_{aCO_2} can lessen lung and systemic organ injury. Substantial evidence demonstrates that moderate HCA directly reduces VILI through several mechanisms including prevention of stretch-induced lung inflammation.[71,72] HCA has been shown to directly attenuate indices of ALI in in vivo and ex vivo models of primary and secondary ischemia-reperfusion lung injury.[73,74] In contrast to the observed beneficial effects of HCA described earlier, there are data to suggest that HCA can be deleterious

in prolonged, untreated pneumonia through reduction of neutrophil- and macrophage-mediated bacterial killing.[75] Also, HCA appears to impair pulmonary epithelial wound healing by diminishing cellular migration through inhibition of NF-κB.[76] Despite these concerns, the potential for therapeutic HCA in attenuating lung injury is promising.

A major area of controversy in regard to HCA is the role of buffering with sodium bicarbonate infusion, a common practice in many ICUs. Although bicarbonate may correct arterial pH, it may actually worsen intracellular acidosis because the CO_2 produced from reaction of metabolic acids with bicarbonate diffuses readily across cell membranes.[77] Accumulating evidence also suggests that the protective effects of HCA are attenuated by pH buffering,[77-79] suggesting that these benefits are a function of pH, rather than elevated CO_2.

There has been a lack of human trials to establish guidelines for the use of buffering during therapeutic hypercapnia. In most institutions, sodium bicarbonate would not be administered unless the pH was less than 7.2. With increased experience, however, many centers reserve bicarbonate infusion for pH less than 7.0. A previous alternative was to use tris(hydroxymethyl)aminomethane (THAM), which does not increase CO_2 production but is not specifically labeled for this use.[80] However, the manufacturer of THAM has recently discontinued its production. Sedation is mandatory with permissive hypercapnia in mechanically ventilated patients in order to control respiratory drive and prevent discomfort. Even with heavy sedation, however, respiratory drive may be insufficiently suppressed, resulting in patient–ventilator dyssynchrony. Neuromuscular blockade is often necessary in these patients.

Positive End-Expiratory Pressure

PEEP is one of several methods of increasing mean airway pressure and improving oxygenation. It improves oxygenation by enhancing lung volume and increasing functional residual capacity (FRC) through recruitment of collapsed alveoli. Lung compliance may also be improved. The use of PEEP in patients with ARDS has two primary goals: adequate tissue oxygen delivery and the reduction of F_{IO_2} to nontoxic (generally <0.6) levels. Increasing PEEP above a certain level, however, may have significant adverse effects. By raising intrathoracic pressure, PEEP may significantly reduce venous return and cardiac output.[63] This may result in decreased tissue oxygen delivery despite improvement in arterial oxygen saturation. This effect is accentuated in the hypovolemic patient and can usually be reversed with intravascular volume expansion. Cardiac depression is rarely seen with PEEP levels less than or equal to 10 cm H_2O. If PEEP greater than 10 cm H_2O is required, assessment of intravascular volume status and cardiac function is warranted.

PEEP may also result in alveolar overdistention with compression and obliteration of surrounding pulmonary capillaries. This alveolar overdistention may actually worsen oxygenation by increasing the shunt fraction. Moreover, dead space ventilation may be increased, resulting in a higher minute ventilation requirement. Finally, PEEP may cause a maldistribution of tidal volumes and pressures, creating overdistension of normally aerated lung regions. This hyperinflated form of lung injury may result in barotrauma referred to as *volutrauma*.

The optimal approach to PEEP for ARDS remains a matter of debate. In the ARDSNet study, patients ventilated with lower V_T required higher levels of PEEP (9.4 vs 8.6 cm H_2O) to maintain oxygen saturation greater than 85%, leading some to speculate that the improved survival rates were due to higher levels of PEEP. However, three subsequent RCTs, including the ARDSNet trial of higher versus lower PEEP levels in patients with ARDS, have failed to demonstrate additional benefit of higher PEEP in patient outcomes.[81-83] It is likely, however, that these trials enrolled patients who had both a lower and a greater potential for alveolar recruitment. Gattinoni et al[84] found that in patients with ARDS, the percentage of potentially recruitable lung differs widely. With the use of higher levels of PEEP, there can be an associated increase in plateau pressure in patients who have regions of lung that are nonrecruitable with a higher risk of overdistention of already recruited alveoli. Patients with recruitable lungs have proportionally less increase in plateau pressure when higher levels of PEEP are used and may benefit from PEEP with less risk of overdistention.[85]

In the individual patient, the application of PEEP needs to be balanced between providing a sufficient inspiratory plateau pressure (peak alveolar pressure) to maintain adequate oxygenation and at the same time avoiding derecruitment of the alveoli during exhalation. Today, many centers have adopted the algorithm-driven approach adopted by the ARDSNet investigators where PEEP was individually titrated with combinations of PEEP and F_{IO_2} selected for a target level of arterial oxygenation. The advantage of a ratio approach is that it can be done without measurement of pulmonary compliance curves or invasive hemodynamic monitoring. One disadvantage is that it is not necessarily applicable to an individual patient with individual physiology. Despite this criticism, this approach has been used in all of the trials conducted by ARDSNet and carries validity.

The PV curve has also been used to select optimal PEEP. In this approach, static PV curves are used to select a PEEP level above the lower inflection point to prevent alveolar end-expiratory collapse. Although this approach is intriguing, static PV curves can be difficult to obtain as well as to implement. Measurement of the PV also requires sedation and often paralysis as patient effort may confound measurements of inflection points. Further evidence is needed before the PV curve can used routinely to determine optimal PEEP. Other methods for selecting PEEP include use of the stress index (the shape of the pressure–time curve during constant flow volume-controlled ventilation), use of esophageal pressure monitoring to maintain positive transpulmonary pressure, and use of imaging techniques such as CT, electrical impedance tomography, and ultrasound to evaluate PEEP settings.[85]

Recruitment Maneuvers

A recruitment maneuver is a transient increase in transpulmonary pressure to promote reopening of collapsed alveoli. The use of recruitment maneuvers in ARDS for the symptomatic treatment of hypoxemia is still a matter of debate. Complications secondary to recruitment maneuvers such as desaturation or hypotension are common, but serious complications, such as a new air leak through an existing chest tube, are infrequent.[86] A systematic review and meta-analysis including 10 RCTs reported a decrease in hospital mortality with the use of recruitment maneuvers; however, there is concern about the risk of bias in the included studies and the use of concomitant therapeutic strategies.[87] Some groups advocate use of recruitment maneuvers as rescue therapy in patients with ARDS who have life-threatening hypoxemia.

New light was shed on this debate with the recent results of the Alveolar Recruitment for Acute Respiratory Distress Syndrome Trial (ART). Investigators randomized patients with moderate to severe ARDS to low PEEP and low tidal volume as standard treatment versus an experimental treatment group consisting of incremental high PEEP recruitments up to 45 cm H_2O. After recruitment, PEEP was decreased to what was best respiratory compliance to determine optimal PEEP, then followed by another recruitment maneuver. Thereafter, tidal volume was adjusted to optimal PEEP, low tidal volume (4–6 mL/kg), and plateau pressures below 30 cm H_2O. At 28 days, death was higher in the intervention group (55%) compared to the control group (49%). Death was still higher at 6 months in the intervention group (65%) compared to the control group (60%). Patients being recruited also had a higher incidence of pneumothorax and requirement of pneumothorax drainage.[88] The Sigh Ventilation to Increase Ventilator-Free Days in Victims of Trauma at Risk for Acute Respiratory Distress Syndrome (SiVent) trial is a large ongoing trial that randomizes trauma patients on mechanical ventilation to standard mechanical ventilation or the treatment group of a "sigh" breath every 6 minutes. The sigh breath gives a tidal volume that produces a plateau pressure of 35 cm H_2O (or 40 cm H_2O in morbidly obese patients or those with significant abdominal distention). Notably, the time at higher PEEP for recruitment will be markedly shorter in this trial, although more frequent. The SiVent trial with a different recruitment strategy will give important insights regarding regular recruitment in trauma patients and how it could potentially decrease rates of ARDS.

Equally as important as recruiting new alveoli is maintaining recruitment. Our institution uses a protocol of pressure support and PEEP of 40/20 for 2 minutes as a recruitment maneuver and then will keep the PEEP at 2 cm H_2O above what the previous value PEEP was prior to the recruitment. A common pitfall we have seen leading to significant derecruitment is when ventilator tubing is exchanged, such as when patients are being transported to and from the operating room. We commonly clamp the endotracheal tube prior to disconnecting from the ventilator to prevent the airway pressure drop from an open airway and subsequent derecruitment.

Prone Ventilation

Prone positioning has been used for more than 30 years in patients with acute hypoxemic respiratory failure and ARDS. Initially, prone positioning in ARDS patients was found to be an efficient technique to improve oxygenation as a rescue treatment in case of life-threatening hypoxemia; it is also now evident that prone positioning is able to prevent VILI. Therefore, prone positioning is a strategy that covers the two major goals of ventilator support in ARDS patients: maintaining safe oxygenation, and preventing VILI.

Since the initial description by Bryan[89] of the beneficial effects of the prone position on arterial oxygenation more than 40 years ago, it is remarkable that it took 20 years for the first RCT to be performed. Several early RCTs showed an improvement in survival with prone positioning.[90-93] However, meta-analyses[94,95] have since shown benefit in severe ARDS subgroups. A recently published RCT by the Proning Severe ARDS Patients (PROSEVA) Study Investigators[96] assigned 466 patients with severe ARDS to undergo prone-positioning sessions of at least 16 hours or to be left in the supine position. The 28-day mortality was 16.0% in the prone group and 32.8% in the supine group (P <.001). Furthermore, unadjusted 90-day mortality was 23.6% in the prone group versus 41.0% in the supine group (P <.001), with a hazard ratio of 0.44 (95% confidence interval [CI], 0.29–0.67). Importantly, there was no significant difference in the incidence of complications between the groups, except for the incidence of cardiac arrests, which was higher in the supine group.

The mechanisms by which prone positioning improves survival in patients with severe ARDS are multifactorial.[97] Pappert et al[98] used the multiple inert gas elimination technique and showed that the improvement in oxygenation was the result of decreased intrapulmonary shunt fraction. Albert and coworkers have investigated the mechanism of prone ventilation in a canine oleic acid model of ARDS. The prone position consistently reduced shunt fraction compared with the supine position. The improvement in gas exchange was independent of changes in cardiac output or FRC between the two positions. Subsequently, pulmonary blood flow was shown to be distributed preferentially to dorsal lung regions in the supine and prone positions.[99] In supine animals, pleural pressure increases from nondependent to dependent regions. This may lead to dependent atelectasis in the highly perfused dorsal lung regions, resulting in intrapulmonary shunt and hypoxemia. In the prone position, the pleural pressure gradient is less, and the dorsal (now nondependent) regions are exposed to a lower pleural pressure. This results in opening of previously atelectatic alveoli. Intrapulmonary shunting is reduced because perfusion of the dorsal lung regions is maintained.[100] Using chest CT, Gattinoni et al[90] showed that in the supine position, gasless lung was found predominantly in the dorsal regions. With prone positioning, densities redistributed to the ventral areas and dorsal regions were well aerated. Thus, prone positioning results in recruitment of previously atelectatic dorsal lung regions.

In addition to improvement in oxygenation, there is increasing evidence regarding the effects of prone position on respiratory mechanics and lung volume. In adult humans with ARDS, chest wall elastance, which contributes to set the end-expiratory lung volume (EELV), has consistently been found to be higher in the prone than in the supine position.[97] Pelosi et al[101] found that sighs superimposed on the prone position further increased EELV and oxygenation only in the prone position, suggesting that the prone position extends the potential for recruitment. Lung CT scan studies have also shown that the prone position promotes lung recruitment as compared to the supine position regardless of whether PEEP was low or high in the supine position.[84]

Because vascular dysfunction is a major independent factor associated with ARDS mortality, the beneficial effects of prone positioning on hemodynamics have been shown to be important. These effects include the reduction of the transpulmonary gradient (the difference of mean pulmonary arterial pressure relative to pulmonary artery occlusion pressure).[102] Also, the increase in pulmonary arterial occlusion pressure induced by prone positioning is thought to result in pulmonary vascular recruitment, thus decreasing dead space, another factor independently related to ARDS mortality.[102] There is also evidence that prone positioning unloads the right ventricle in severe ARDS,[103] which may play a role in patient outcome.

Another important benefit of prone positioning appears to be prevention of VILI. Broccard et al[104] showed that prone positioning attenuates and redistributes VILI in dogs. Papazian et al[105] also found lower concentrations of proinflammatory cytokines in BAL fluid in ARDS patients after 12 hours in the prone position compared to supine position in similar settings.

The technical aspects of prone positioning require a coordinated team effort. Prone positioning must be performed with care to avoid inadvertent extubation or loss of intravenous lines or chest tubes. Transient hemodynamic instability and desaturation also may occur during repositioning. Cardiopulmonary resuscitation is difficult, if not impossible, in the prone position. Placement of multifunction electrode pads, which allow defibrillation, cardioversion, and pacing, has been recommended to facilitate cardiopulmonary resuscitation in the prone position. Other areas of concern that accompany prone positioning include facial and eyelid edema, peripheral nerve injury, tongue injuries, and skin necrosis. Multiply injured patients may present unique problems due to the presence of incisions, drainage tubes, extremity fractures, cervical spine fractures, or facial fractures, and the like.

High-Frequency Ventilation

Recognition of the impact of mechanical ventilation on VILI naturally led to an interest in the use of high-frequency oscillatory ventilation (HFOV); after all, this might be considered the ultimate in "low tidal volume ventilation." High-frequency ventilation techniques such as HFOV use very small tidal volumes (1–5 mL/kg) delivered at rates of 60 to 3600 cycles/min. Although peak airway pressures are reduced compared with conventional modes, mean airway pressures, barotrauma, and hemodynamic compromise appear unchanged. The OSCILLATE (OSCillation in ARDS Treated Early) trial[106] showed that HFOV did not reduce, and may increase, in-hospital mortality. HFOV was also associated with higher mean airway pressures and greater use of sedatives, neuromuscular blockers, and vasoactive drugs. The OSCAR (High-Frequency Oscillation in ARDS) trial[107] also showed that HFOV failed to reduce 30-day mortality in patients undergoing mechanical ventilation for ARDS. The disappointing results of these two major trials have cast doubt on the benefits of HFOV in ARDS. A recent meta-analysis again showed increased mortality in patients with mild to moderate ARDS when HFOV was used; however, results are less clear in severe ARDS.[108]

In light of the available evidence and until further trials address the previously discussed issues, HFOV should likely be reserved for a subset of patients with refractory hypoxemia. When using HFOV, monitoring lung recruitment and right ventricular function to ensure achievement of appropriate physiologic goals is crucial.

Extracorporeal Life Support (See Atlas Figures 38 and 39)

Extracorporeal life support (ECLS), also called extracorporeal membrane oxygenation (ECMO), has been in clinical use since 1972. The most common type of ECLS used in ARDS patients is venovenous (V-V) ECLS, where blood is both withdrawn from and returned to the venous system. Blood is withdrawn into an extracorporeal circuit by a mechanical pump before entering an oxygenator. A membrane within the oxygenator provides a blood–gas interface for diffusion of gases. The "flow" of blood to this membrane helps to control oxygenation and can be dialed up or down depending on the support needed. The "sweep" gas at this membrane is typically 100% oxygen or carbogen (5% CO_2, 95% O_2) and blows away CO_2 that has diffused from blood and similarly can be fine-tuned to control patient blood CO_2 levels. The oxygenated extracorporeal blood can be warmed or cooled as needed and then returned to the body.

The results of two early RCTs in patients with ARDS (published in 1997[109] and 1994[110]) did not show a survival benefit with ECLS, including extracorporeal CO_2 removal (a related technique). The old ECLS circuits were difficult to manage and potentially hazardous, with a relatively high complication rate. Recently, technologic advancements in circuitry, including introduction of dual lumen percutaneous catheters and better pumps and oxygenators, have been instrumental in improving patient outcomes and wider adaptation of this therapy. Recent observational studies have suggested a benefit in severe cases of ARDS, with survival rates ranging from 47% to 66%.[111-113] Widespread interest in use of ECLS intensified with the H1N1 influenza epidemic in 2008 to 2009 that resulted in thousands of cases of severe respiratory failure, often with concomitant septic shock generated.

The use of ECLS in these critically ill patients was associated with an impressive 70% survival rate.[114-116]

The Conventional Ventilator Support Versus ECMO for Severe Adult Respiratory Failure (CESAR) trial[117] was the first controlled clinical trial using modern ECMO technology. In this trial, 180 patients were allocated to either continued conventional management or referral to a specialized center with a standardized management protocol that included consideration for treatment with ECLS—75% of those allocated patients eventually received ECLS. Of the patients randomized to ECLS, 63% survived to 6 months without disability compared to 47% of those assigned to conventional management. However, this study was not a randomized trial of ECLS compared with standard lung-protective ventilation. In fact, only 70% of patients in the conventional management group received lung-protective ventilation. Despite these limitations, this trial provides support for transferring patients with severe ARDS to a tertiary care center that is capable of advanced ventilator management techniques and not necessarily the ability to carry out ECMO.

The ECMO to Rescue Lung Injury in Severe ARDS (EOLIA) trial results were just recently published, and the trial attempts to provide clarity on the utility of early ECMO compared to standard ventilation of severe ARDS.[118] As opposed to the CESAR trial, nearly all of the patients randomized to ECMO were in fact cannulated. This trial also did a better job of standardizing treatment for the control group including lung-protective ventilation. The trial was stopped early because it was determined that the preset absolute risk reduction of 20% would not be met, and the authors concluded that early ECMO was not superior to conventional treatment. Because the trial was ended early, it was likely underpowered, but there is still much to learn from it. Before trial termination, mortality at 60 days was 35% in the ECMO group and 46% in controls. Importantly, 28% of patients in the conventional treatment arm were allowed to cross over to ECMO for refractory hypoxemia, which could dilute the ECMO treatment effect. It also showed that complications between the groups were the same, highlighting that ECMO can be used safely.

The CESAR and EOLIA trials highlight the uncertainty regarding which patients with ARDS are the best candidates for this treatment and the optimal timing for initiation of therapy. In general, indications for ECLS in patients with ARDS include one or more of the following: severe hypoxemia, uncompensated hypercapnia, and the presence of excessively high end-inspiratory plateau pressures (Table 59-7).[118] According to the 2017 Extracorporeal Life Support Organization (ELSO) guidelines,[119] the use of ECMO should be considered when the ratio of Pao_2 to Fio_2 is less than 150, and ECMO is indicated when the ratio is less than 80. CO_2 retention despite a plateau pressure greater than 30 cm H_2O is also considered an indication for ECMO in patients with ARDS, as are severe air leak syndromes, need for intubation for patients listed for lung transplant, or acute cardiac or respiratory collapse. There are no absolute contraindications; however, the following should be thoughtfully considered if ECMO is considered because they portend a poorer outcome: mechanical ventilation at high settings for 7 days, major pharmacologic immunosuppression, recent or expanding CNS hemorrhage, nonrecoverable comorbidity, and increasing age.[119]

Obtaining vascular access is done by cannulation of large vessels in the neck or groin. Where and how to place cannulas depends on the status of the patient. For patients with adequate cardiac function, V-V ECMO will be chosen and drainage and return cannulas will be placed in the femoral and jugular veins. These cannulas have the advantage of ease and can be placed percutaneously or via cut-down, without the need for fluoroscopy. Groin lines, however, limit the ability for patients to ambulate. Most centers have shifted to the use of a single (dual lumen) large cannula placed under fluoroscopic guidance as the preferred approach for adults requiring V-V ECMO.[120] The Avalon cannula is commonly used at our institution and drains the superior vena cava and inferior vena cava, and has a side hole on the reinfusion lumen that is oriented toward the tricuspid valve. Proper placement requires adequate imaging, typically fluoroscopy. Without proper placement, recirculation will be an issue and limit the effectiveness of ECMO therapy. The main advantage of these cannulas is that mobility of patients is greatly facilitated. If a patient is unstable for transport to the operating room for dual lumen cannula placement, two single-lumen cannulas can certainly be exchanged for a dual lumen cannula at a later date. Similarly, cannulas can be added at any time if blood flow resistance is too high such that there is inadequate venous drainage. If there is cardiac instability, venoarterial ECMO can be considered with cannulation typically of the femoral artery that provides pressurized blood return to augment cardiac function. It should be noted that in patients with severe respiratory compromise, cardiac function will often improve on V-V ECMO as the patient's respiratory status improves.[119]

Cannulas and lines are thrombogenic. Thrombotic and embolic risks are real with ECMO, and as such, patients are anticoagulated. Just prior to cannulating, a heparin bolus is given (ELSO recommends 50–100 units/kg), followed by a continuous infusion during the run of ECLS. An activated clotting time goal of 1.5 times normal is a typical goal. Partial thromboplastic time and thromboelastography may also be used to titrate effect of heparin. If bleeding is uncontrollable and anticoagulation must be stopped, flow should be kept high and a replacement circuit should be nearby if the circuit clots.[119]

The most appropriate strategy for weaning patients with ARDS from ECMO is a matter of debate. Typically, weaning from ECMO starts when improvement is noted in oxygenation, chest radiography findings, or lung compliance. At that time, the flow rate of sweep gas is decreased to compensate for increase in lung ventilation. Lung-resting ventilation is then transitioned to standard lung-protective settings or pressure-support ventilation. When ECLS support is less than 30% of total, native lung function may be adequate and a trial off is indicated.[119]

TABLE 59-7: Indications and Contraindications for ECMO in Severe Cases of ARDS

Indications:

1. In hypoxic respiratory failure due to any cause (primary or secondary), ECLS should be considered when the risk of mortality is 50% or greater and is indicated when the risk of mortality is 80% or greater
   a. 50% mortality risk is associated with a Pao_2/Fio_2 <150 on Fio_2 >90% and/or Murray score 2–3
   b. 80% mortality risk is associated with a Pao_2/Fio_2 <100 on Fio_2>90% and/or Murray score 3–4 despite optimal care for 6 h or more
2. CO_2 retention on mechanical ventilation despite high Pplat (>30 cm H_2O)
3. Severe air leak syndromes
4. Need for intubation in a patient on lung transplant list
5. Immediate cardiac or respiratory collapse (pulmonary embolus, blocked airway, unresponsive to optimal care)

Contraindications:

There are no absolute contraindications to ECLS, as each patient is considered individually with respect to risks and benefits. There are conditions, however, that are associated with a poor outcome despite ECLS and can be considered relative contraindications.

1. Mechanical ventilation at high settings (Fio_2 >0.9, Pplat >30) for 7 days or more
2. Major pharmacologic immunosuppression (absolute neutrophil count <400/mm^3)
3. Central nervous system (CNS) hemorrhage that is recent or expanding
4. Nonrecoverable comorbidity such as major CNS damage or terminal malignancy
5. Age: no specific age contraindication but consider increasing risk with increasing age

ARDS, acute respiratory distress syndrome; CO_2, carbon dioxide; ECLS, extracorporeal life support; ECMO, extracorporeal membrane oxygenation; Pplat, plateau pressure.
Source: Based on Extracorporeal Life Support Organization (ELSO) guidelines for adult respiratory failure.

The usual duration of ECLS in patients with ARDS is 7 to 10 days; however, survival rates of 50% to 70% with native lung recovery are increasingly reported in cases of prolonged-duration ECLS (>14 days).[112,121] With advances in component technology and the techniques used to perform ECLS, serious complications have been reduced significantly. In the CESAR trial, only one serious adverse event related to ECLS was reported (a death related to vessel perforation during cannulation). In the EOLIA trial, bleeding requiring transfusion was the most common adverse event (46%), although massive bleeding was rare (2%). Other common complications in the ECMO group included thrombocytopenia (40%), ventilator-associated pneumonia (2%), and cardiac rhythm disturbances (31%). Stroke incidence was rare (2%).[118]

PHARMACOLOGIC THERAPY OF ACUTE RESPIRATORY DISTRESS SYNDROME

The diversity of approaches to pharmacologic therapy for ARDS reflects the complex pathophysiology. Many therapies have been tested in randomized trials in humans including NO, corticosteroids, immunomodulating agents, pulmonary vasodilators, antioxidants, and surfactants.

Inhaled Nitric Oxide and Prostacyclin

NO is responsible for regulation of basal vascular tone. Inhaled NO (iNO) is a selective pulmonary vasodilator with no systemic side effects. Moreover, delivery of iNO exposes only ventilated alveoli to its vasodilatory effects. Selective vasodilation of pulmonary vessels in ventilated areas diverts blood away from nonventilated areas, improving ventilation/perfusion matching and hypoxemia. Inhaled NO decreases mean pulmonary artery pressure and, in doing so, lessens the hydrostatic pressure for pulmonary edema.[122] For all of these reasons, iNO has seemed very attractive for use in severely hypoxemic patients; however, data from clinical trials in humans have been disappointing. The largest multicenter RCT of iNO for treatment of ARDS assigned 385 patients to placebo (nitrogen gas) or iNO at 5 ppm until 28 days, discontinuation of assisted breathing, or death. iNO at a dose of 5 ppm resulted in short-term oxygenation improvements in patients with ALI not due to sepsis and without evidence of nonpulmonary organ system dysfunction; however, there was no substantial impact on the duration of ventilatory support or mortality.[123] A Cochrane Review of 14 prospective RCTs of iNO versus placebo or standard therapy showed a statistically significant but transient improvement in oxygenation in the first 24 hours but no statistically significant effect on overall mortality. Concerningly, iNO appeared to increase the risk of renal impairment among adults.

Currently, the role of iNO should be limited to short durations (24–96 hours) in patients with severe, refractory hypoxemia or pulmonary hypertension in whom iNO may act as a "bridge," allowing short-term physiologic support. Application of iNO in these situations may allow for possible patient survival until other therapies may be employed such as pronation or alternative modes of ventilation.

Aerosolized prostacyclins are potent vasodilators with similar effects as iNO including reduction of pulmonary arterial hypertension, improvement in right-heart function, and improvement in ventilation/perfusion matching.

There has been an interest in use of aerosolized prostacyclins as an alternative to iNO due to various advantages (cost, setup, and administration). Similar to iNO, trials of aerosolized prostacyclin have shown transient improvement in oxygenation but no significant differences in mortality, ventilator-free days, or reduction in disease severity.[124] These trials have been limited by small sample sizes, lack of appropriate control subjects, and varying doses, timing, and modes of delivery of the drugs.

Corticosteroids

The ability of corticosteroids to attenuate the inflammatory response would seem to make them ideal treatment for ARDS. From the first description of ARDS, corticosteroids were suggested as possible therapeutic agents. Controversy remains as to whether low-dose corticosteroids can reduce the mortality and morbidity ARDS without increasing the risk of adverse reactions. Early RCTs to assess the use of corticosteroids in patients with respiratory failure at risk of developing ARDS showed no role for steroids in the prevention of ARDS.[125] Large prospective clinical trials have also failed to show any survival benefit with high-dose steroids in early ARDS. The earliest trial, by Bernard et al,[126] tested 30 mg/kg of intravenous methylprednisolone every 6 hours for a total of four doses, with the hypothesis that this treatment would reduce 45-day mortality. However, the study was stopped early after enrolling 99 patients, with 60% mortality in the methylprednisolone group and 63% in the control group.[126] Another RCT by Meduri et al[127] in 2007 that examined the effect of low-dose methylprednisolone given during the first 72 hours of onset of ARDS showed improvement in pulmonary outcomes, including LIS and ventilator-free days, but no significant difference in mortality.

With evidence mounting against treatment with corticosteroids for prevention and early treatment of ARDS, trials have shifted toward evaluating the utility for treatment of the late, proliferative phase of ARDS, with the understanding that the pathogenetic mechanisms initiating early ARDS differ from those of the late stages. A number of anecdotal reports and small series have suggested that high-dose corticosteroids may be of some benefit during late ARDS.[128,129] The ARDSNet conducted a multicenter RCT of 180 patients with ARDS of at least 7 days in duration who were assigned to either methylprednisolone or placebo. Methylprednisolone treatment did increase the number of ventilator-free days, improved oxygenation and respiratory compliance, and resulted in less vasopressor use without a difference in ICU length of stay. There was no overall difference in 60-day mortality; however, among patients enrolled after day 13 of ARDS, mortality was 12% in the placebo group compared with 44% in the steroid group, suggesting that steroids may be harmful if administered too late in the course of ARDS. Interestingly, the rate of infectious complications was no different between the groups, but the methylprednisolone group had a higher incidence of neuromuscular weakness and hyperglycemia. Given the challenges of this patient group, any use of steroids for late ARDS must be individualized and, optimally, delivered before disease day 14. Several meta-analyses have failed to conclusively support routine use of corticosteroids for treatment of early or late ARDS.[125,130-132] Well-designed RCTs are needed to understand the potential benefits of steroid treatment in the face of short- and long-term risks, including delayed recovery and impairments in physical function and health-related quality of life.

Neuromuscular Blockade

Neuromuscular blockade or paralysis can improve ventilator dyssynchrony and also prevent phenomena such as breath stacking that can cause injury. The ACURASYS study used cisatracurium for 48 hours early in ARDS and found improved 90-day mortality, as well as more ventilator-free days in patients receiving paralytic. These data need to be confirmed, and we will have important insights after the Reevaluation of Systemic Early Neuromuscular Blockade (ROSE) trial is completed.[133]

Exogenous Surfactant

Surfactant dysfunction with quantitative and qualitative abnormalities of phospholipids and proteins is a pathologic feature of ARDS. Results for surfactant therapy in infants with the respiratory distress syndrome of prematurity were dramatic, with improvement in gas exchange and lung mechanics and decrease in CPAP requirement and barotrauma. Unfortunately, results in adult ARDS patients have not been consistent. This is likely due to differences in pathophysiology because ARDS is characterized by surfactant inhibition by plasma constituents and increased breakdown by activated oxidative and hydrolytic pathways, whereas the deficiency of surfactant is the primary pathology in premature infants.[134] Several RCTs of surfactant replacement in adults with ARDS have shown improvements in oxygenation indices but have failed to produce any demonstrable survival benefits, as noted by a recent meta-analysis of nine RCTs.[135] Further understanding of which patients may benefit the most from this type of therapy as well as refinement of surfactant preparations may offer hope in the future use of this therapy.

Antioxidants

It is well known that indices of oxidative damage, including neutrophil recruitment, lipid peroxidation, and protein degradation, are higher in patients who die of ARDS. Natural antioxidants such as glutathione have been shown to be depleted in the lungs of ARDS patients. This has been the rationale behind using agents that increase glutathione levels such as *N*-acetylcysteine (NAC) and procysteine. A Cochrane

analysis of five studies using NAC that randomized 239 patients showed no statistically significant effect of NAC on early mortality and inconclusive results regarding duration of mechanical ventilation or ventilator-free days to day 28.[132] Currently, no clear evidence exists for use of NAC or procysteine in the treatment of ARDS patients.

Anti-Inflammatory Therapies

Neutrophil elastase, a serine protease with antimicrobial and anti-inflammatory actions, has been implicated in the pathogenesis of ARDS. Silvelestat, a neutrophil elastase inhibitor, was investigated in the STRIVE trial, an international, multicenter RCT.[136] The study was stopped prematurely due to an increase in 180-day all-cause mortality. A recent meta-analysis of eight clinical trials (including STRIVE) has shown no benefit in short-term mortality and a worse outcome for 180-day mortality.[137]

Statins have also been tested as anti-inflammatories to treat patients with ARDS. Two large trials have compared either rosuvastatin (SAILS trial) or simvastatin (HARP-2 trial) to placebo. No difference was found in either mortality or ventilator-free days, suggesting statins should not be used routinely in ARDS.[138,139]

Antiplatelet Therapy

Because of the observational data showing platelets may contribute to lung injury and aspirin may prevent lung injury, a randomized trial was done giving aspirin therapy to patients deemed at being at elevated risk for ARDS development. Unfortunately, this trial did not show any benefit to aspirin in regard to ARDS development, ventilator-free days, or survival.[140]

β-Agonist

Because of data showing β_2-agonists can accelerate resolution of pulmonary edema, trials have been performed using salbutamol in ARDS patients and to prevent ARDS. It can in fact decrease pulmonary edema; however, the trials have failed to show a survival benefit, and one was terminated early because of increased mortality in patients receiving salbutamol.[141,142]

Nutritional Support

Nutrition support in critically ill patients with ALI and ARDS is a vital aspect of treatment. The proinflammatory and hypercatabolic response seen with ALI/ARDS can lead to significant nutrition deficits. Proper nutrition support is necessary to prevent cumulative caloric deficits, malnutrition, loss of lean body mass, and deterioration of respiratory muscle strength. Early enteral nutrition has been shown to attenuate disease severity and immune response to injury. The optimal dosage, composition of fatty acids, and ratio of individual immune-modulating nutrients in specialized enteral formulations remain controversial; however, a meta-analysis of three trials showed that a diet enriched with eicosapentaenoic acid (EPA), γ-linolenic acid (GLA), and antioxidants resulted in reduction in mortality, increase in ventilator- and ICU-free days, improvement in oxygenation, and reduction in the risk of developing new organ failures.[143] The Omega arm of the ARDSNet EDEN-Omega study, which was studying enteral omega-3, GLA, and antioxidant supplementation, was terminated early due to futility.[144] The EDEN arm compared enteral trophic feeds to full enteral feeds for the first 6 days after ALI and found no difference in mortality or ventilator-free days between the groups.[145] However, based on existing data, American Society for Parenteral and Enteral Nutrition/Society of Critical Care Medicine guidelines, and the Canadian clinical practice guidelines, an enteral diet enriched with omega-3 fatty acid, GLA, and antioxidant supplementation should be considered in patients with ALI/ARDS.[146]

Careful monitoring of nutritional support may be necessary in patients with tenuous respiratory status. An indirect calorimeter can be helpful in providing estimates of CO_2 production and the respiratory quotient. The respiratory quotient should be kept below 0.9 by appropriate adjustment of the proportion of lipid and total calories administered. Overfeeding patients or administering excess carbohydrates can lead to excess production of CO_2. In the setting of marginal ability to execute work of breathing, this may, in theory, precipitate or prolong hypercapnic pulmonary failure. Our goals for nutritional support are to deliver 21 to 25 nonprotein cal/kg/d and 0.25 to 0.30 g of nitrogen/kg/d. The primary goal of carbohydrate administration should be a rate of less than 5 mg/kg/min.

OUTCOME OF ACUTE RESPIRATORY DISTRESS SYNDROME

Mortality associated with ARDS has historically been reported from 30% to 60% with significant variation due to age and risk factors (see the section "Risk Factors for Acute Respiratory Distress Syndrome"). The majority of deaths are related to sepsis and MOF (see Chapter 63). Respiratory failure is a cause of death in only 15% of patients. There has been a gradual improvement in overall incidence and mortality of ARDS patients across several RCTs in the past two decades, with the most recent rates as low as 20% to 25%.[147] Going beyond mortality, survivors of ARDS have significant physical limitations, even at 5 years after their illness despite normal or near-normal lung function, showing the physical impact an episode of ARDS can have on long-term outcomes.[148]

The principal therapy that improves outcome in patients with ARDS appears to be low tidal volume ventilation,[149] but other improvements in critical care delivery and goal-directed care reduce ARDS incidence as well (Fig. 59-4).

NIH NHLBI ARDS Clinical Network Mechanical Ventilation Protocol Summary

PART I: VENTILATOR SETUP AND ADJUSTMENT

1. Calculate predicted body weight (PBW)
   - **Males** = 50 + 2.3 [height (inches) -60]
   - **Females** = 45.5 + 2.3 [height (inches) -60]
2. Select any ventilator mode
3. Set ventilator settings to achieve initial V_T = 8 ml/kg PBW
4. Reduce V_T by 1 ml/kg at intervals ≤ 2 hours until V_T = 6 ml/kg PBW.
5. Set initial rate to approximate baseline minute ventilation (not > 35 bpm).
6. Adjust V_T and RR to achieve pH and plateau pressure goals below.

OXYGENATION GOAL: **PaO_2 55-80 mmHg or SpO_2 88-95%**
Use a minimum PEEP of 5 cm H_2O. Consider use of incremental FiO_2/PEEP combinations such as shown below (not required) to achieve goal.

Lower PEEP/higher FiO_2

FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7
PEEP	5	5	8	8	10	10	10	12

FiO_2	0.7	0.8	0.9	0.9	0.9	1.0
PEEP	14	14	14	16	18	18-24

Higher PEEP/lower FiO_2

FiO_2	0.3	0.3	0.3	0.3	0.3	0.4	0.4	0.5
PEEP	5	8	10	12	14	14	16	16

FiO_2	0.5	0.5-0.8	0.8	0.9	1.0	1.0
PEEP	18	20	22	22	22	24

pH GOAL: 7.30-7.45
Acidosis Management: (pH < 7.30)

- **If pH 7.15-7.30**: Increase RR until pH > 7.30 or $PaCO_2$ < 25 (Maximum set RR = 35).
- **If pH < 7.15**: Increase RR to 35.
  If pH remains < 7.15, V_T may be increased in 1 ml/kg steps until pH> 7.15 (Pplat target of 30 may be exceeded).
  May give $NaHCO_3$

Alkalosis Management: (pH > 7.45): Decrease vent rate if possible.

I: E RATIO GOAL: Recommend that duration of inspiration be ≤ duration of expiration.

PLATEAU PRESSURE GOAL: ≤ 30 cm H_2O
Check Pplat (0.5 second inspiratory pause), at least q 4h and after each change in PEEP or V_T.
If Pplat > 30 cm H_2O: decrease V_T by 1ml/kg steps (minimum = 4 ml/kg).
If Pplat < 25 cm H_2O and V_T < 6 ml/kg: increase V_T by 1 ml/kg until Pplat > 25 cm H_2O or V_T = 6 ml/kg.
If Pplat < 30 and breath stacking or dys-synchrony occurs: may increase V_T in 1ml/kg increments to 7 or 8 ml/kg if Pplat remains ≤ 30 cm H_2O.

PART II: WEANING

A. Conduct a SPONTANEOUS BREATHING TRIAL daily when:

1. FiO_2 ≤ 0.40 and PEEP ≤ 8 OR FiO_2 ≤ 0.50 and PEEP ≤ 5.
2. PEEP and FiO_2 ≤ values of previous day.
3. Patient has acceptable spontaneous breathing efforts. (May decrease vent rate by 50% for 5 minutes to detect effort.)
4. Systolic BP ≥ 90 mmHg without vasopressor support.
5. No neuromuscular blocking agents or blockade.

B. SPONTANEOUS BREATHING TRIAL (SBT):
If all above criteria are met and subject has been in the study for at least 12 hours, initiate a trial of UP TO 120 minutes of spontaneous breathing with FiO_2 ≤ 0.5 and PEEP ≤ 5:

1. Place on T-piece, trach collar, or CPAP ≤ 5 cm H_2O with PS ≤ 5
2. Assess for tolerance as below for up to two hours.
   a. SpO_2 ≥ 90: and/or PaO_2 ≥ 60 mmHg
   b. Spontaneous V_T ≥ 4 ml/kg PBW
   c. RR ≤ 35/min
   d. pH ≥ 7.3
   e. No respiratory distress (distress = 2 or more)
      - HR > 120% of baseline
      - Marked accessory muscle use
      - Abdominal paradox
      - Diaphoresis
      - Marked dyspnea
3. If tolerated for at least 30 minutes, consider extubation.
4. If not tolerated resume pre-weaning setting

Definition of UNASSISTED BREATHING
(Different from the spontaneous breathing criteria as PS is not allowed)
1. Extubated with face mask, nasal prong oxygen, or room air, OR
2. T-tube breathing, OR
3. Tracheostomy mask breathing, OR
4. **CPAP less than or equal to 5 cm H_2O without pressure support or IMV assistance.**

FIGURE 59-4 ARDS Network mechanical ventilation protocol. CPAP, continuous positive airway pressure; HR, heart rate; PEEP, positive end-expiratory pressure; Pplat, plateau pressure; PS, pressure support; RR, respiratory rate. (Reproduced with permission from National Institutes of Health [NIH] National Heart, Lung, and Blood Institute [NHLBI] ARDS Network.)

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Gastrointestinal Failure

Jose J. Diaz • Bryan Collier

KEY POINTS

- In the critically ill or injured patient, gut dysfunction commonly occurs and can take various forms, from food intolerance to life-threating conditions that can progress to multiple organ dysfunction syndrome (MODS) or persistent inflammatory, immunosuppressed, catabolic syndrome (PICS).
- Gut injury can be described as an ischemia/reperfusion event that can present as severe as nonocclusive small bowel ischemia to the abdominal compartment syndrome.
- More commonly, gastrointestinal (GI) dysfunction will manifest as gastroesophageal reflux, gastroparesis, duodenogastric ileus, or malabsorption as a manifestation of food intolerance.
- Recognition of the human microbiome as an important component of gut health and the alterations in the microbiome during critical illness has spurred renewed interest in the microbiology of the GI tract.
- The chapter will review the pathophysiology and the current best practice recommendations for medical management.

INTRODUCTION

In the critically ill or injured patient who survives the initial 48 hours in the intensive care unit (ICU), multiple organ dysfunction syndrome (MODS) and multiple organ failure (MOF) are the leading causes of death in the ICU (see Chapter 63). First, there is strong evidence that during the early stages of shock and reperfusion, episodes of abdominal compartment syndrome (ACS) and gut hypoperfusion can lead to organ dysfunction and death.[1-3] Second, there is growing evidence that the gut microbiome changes soon after injury. The proximal gut, which is typically sterile, develops heavy colonization that can be harmful. These bacteria can have harmful effects on the gut homeostasis and barrier function.[4-12] Third, gut-focused medical therapies such as selective gut decontamination, early enteral nutrition, and most recently immune-enhancing enteral diets have been shown to reduce these nosocomial infections.[6,13-18] The focus of this chapter is to provide an overview of the gut dysfunction of the injured and critically ill patient and how it contributes to overall morbidity and mortality. The discussion will then focus on the pathogenesis and clinical monitoring of specific gut dysfunctions. Based on this information, potential therapeutic strategies to prevent and/or treat gut dysfunction to enhance patient outcome will be discussed.

HOW GUT DYSFUNCTION CONTRIBUTES TO ADVERSE PATIENT OUTCOME

Multiple Organ Failure

MOF results from a dysfunctional, hyperinflammatory response producing two distinct patterns (ie, early vs late) (see Chapter 57). Soon after a traumatic insult, patients are found to be in a state of systemic hyperinflammation, referred to as the systemic inflammatory response syndrome (SIRS).[19-21] The intensity of SIRS is dependent upon (1) innate host factors (genotype/phenotype), (2) the degree of shock, and (3) the amount of tissue injured (mechanism of injury). Of the three, shock is the predominant factor that produces a maladaptive, overexuberant SIRS response.[22,23] The inflammatory response to injury is an evolutionary developed, expected, and maintained response in all organisms. Mild-to-moderate SIRS is most likely beneficial and probably necessary at some basic physiologic level. Severe SIRS has the potential to result in early organ failure and death.[24] As time proceeds, negative feedback systems downregulate certain aspects of acute SIRS to restore homeostasis and limit potential autodestructive inflammation (see Chapter 63).

In a Glue Grant Study by Tompkins et al,[25] blood samples of 167 patients at varying time intervals were taken to better understand the genomic makeup after severe trauma or burn using microarray analysis. They referred to what they found as "genomic storm" in that of "circulating white cells of the 167 patients studied when compared with those of the 35 normal volunteers, more than 80% of the WBC [white blood cell] genes changed significantly."[25] Tompkins et al[25] further described the genomic patterns of complicated patients (eg, who developed MOF or nosocomial infections) as demonstrating higher levels of deviation from normal gene expression, and these changes quantitatively persisted longer than those same genes in the uncomplicated patients. They concluded there were 5136 genes changed at a twofold level in the circulating WBCs of patients compared with normal volunteers. These changes were long-lasting, and in the complicated patients, more than 50% of these genes remained abnormal even at 28 days after injury."[25] The question remains regarding what drives the inflammatory response after the acute insult to potentially cause MOF or death.

In all three phenotypes of MOF (SIRS, compensatory anti-inflammatory response syndrome [CARS], and persistent inflammatory, immunosuppressed, catabolic syndrome [PICS]), it is hypothesized that the gut can be both an instigator and victim of the characteristic dysfunctional inflammatory response.[1,4-6,25-28] Shock is associated with obligatory gut ischemia as a commensurate physiologic response to hypotension. The gut ischemia may lead to essential mucosal damage.[7] With a robust resuscitation, reperfusion results in a locally intense inflammatory response that can further injure the gut, setting the stage for ACS or MOF.[2,3] The reperfused gut releases inflammatory mediators, including proteins such as cytokines and lipids such as those derived from phospholipase A_2, via the mesenteric lymph, that amplify SIRS.[29] Moreover, for patients undergoing laparotomy, bowel manipulation and anesthetics cause further gut dysfunction.[30] Finally, standard ICU therapies (narcotics, proton pump inhibitors, H_2-antagonists, catecholamines, and broad-spectrum antibiotics) and intentional disuse (delayed start of enteral feeds, inappropriate use of the open abdomen, delayed bowel anastomosis, use of total parenteral nutrition rather than enteral nutrition) promote additional gut dysfunction. The result is a progressive dysfunction characterized by gastroesophageal reflux (GER), gastroparesis, duodenogastric reflux, gastric alkalization, decreased mucosal perfusion, impaired intestinal transit, impaired absorptive capacity, increased permeability, decreased mucosal immunity, increased colonization, and gut edema. As time goes on, the normally sterile upper gut becomes heavily colonized, mucosal permeability increases, and local mucosal immunity decreases. Intraluminal contents (eg, bacteria and their toxic products) disseminated by aspiration or translocation can cause systemic sepsis, which promotes further gut dysfunction. The normal gut microbiome with its relationships with immunity, the brain, and injury becomes an important component of gut heath. It has also been recognized to be altered within hours of injury in small animal models. Earley et al[31] demonstrated that burn injury induces a dramatic dysbiosis of the intestinal microbiome of both humans and mice and allows for similar overgrowths of gram-negative aerobic bacteria. The bacteria increasing in abundance have the potential to translocate to extraintestinal sites. McDonald et al[32] collected and processed several samples as part of the Global Gut Study. They demonstrated that critical illness may be associated with the loss of normal, "health-promoting" bacteria, allowing overgrowth of disease-promoting pathogenic bacteria (dysbiosis), which, in turn, makes patients susceptible to hospital-acquired infections, sepsis, and organ failure.[32]

If this dysfunction goes unchecked for long enough, a certain threshold is reached that propels a patient into a vicious cycle of PICS. This persistent inflammation is characterized by increased production of interleukin (IL)-6, a persistent acute phase response, neutrophilia with increased immature granulocyte count, anemia, lymphopenia, and, often, tachycardia.[8,9] Unfortunately, this deranged inflammatory, immunosuppressive state consumes a large amount of energy derived from protein catabolism. The catabolic state despite aggressive nutritional support gives way to the new phenotype that produces substantial lean body mass loss and proportional decrease in functional status.[33,34] It is the PICS phenotype that clinically depicts chronic critical illness (CCI—defined as requiring >14 days of ICU care).[35-37]

A patient meets PICS criteria if he or she is in the ICU more than 14 days with evidence of organ dysfunction and has persistent inflammation, defined by C-reactive protein concentration greater than 50 μg/dL and retinol binding protein concentrations less than 1 mg/dL; immunosuppression, crudely defined by a total lymphocyte count less than 0.80×10^9/L; and a catabolic state, defined by serum albumin concentrations less than 3.0 g/dL, creatinine height index less than 80%, and weight loss of more than 10% or body mass index less than 18 kg/m^2 during the current hospitalization.[8,9] Although these clinical markers are not direct measurements of inflammation, immunosuppression, or protein catabolism, they can serve as surrogates that are readily available in most critical care settings.

Moore and colleagues defined PICS as a new phenotype that has replaced late-appearing MOF. The major challenges for this new paradigm are: (1) to identify PICS early in its course, (2) to understand its underlying pathophysiology, and (3) to initiate appropriate multimodal therapies that target specific components of the syndrome.[38] Medical care resource consumption associated with PICS has yet to be measured but is likely to be a large multiple of the costs associated with the short-term treatment of trauma, severe sepsis, and septic shock.[39] The incidence of PICS will likely increase as our population ages and our ICU technology improves.[40] Characterization and management of PICS will require technologies for direct monitoring and modulation of the patient's nutritional status and immune responses. PICS is likely to be the new horizon for surgical intensive care (Fig. 60-1).[41]

After the initial insult, there is a simultaneous SIRS/CARS response. Some patients develop severe SIRS and proceed to the early MOF and fulminant early death trajectory. Modern

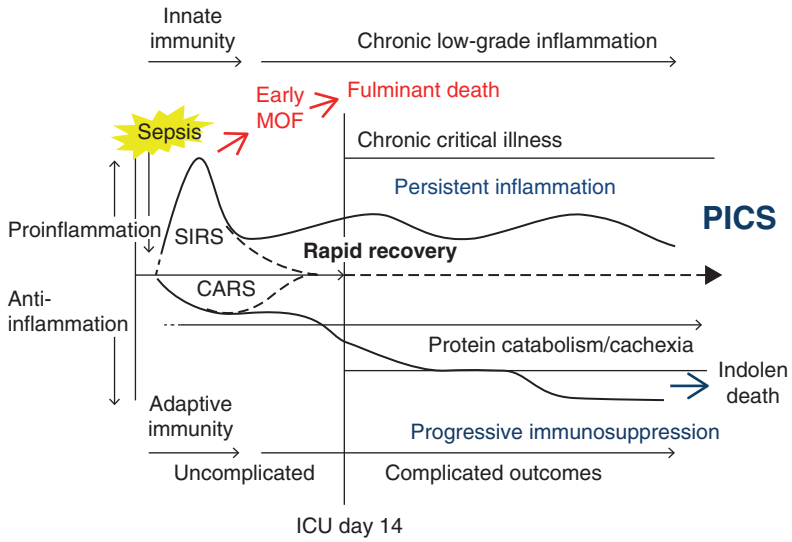

FIGURE 60-1 Persistent inflammatory, immunosuppressive, and catabolic state (PICS) algorithm. CARS, compensatory anti-inflammatory response syndrome; ICU, intensive care unit; MOF, multiple organ failure; SIRS, systemic inflammatory response syndrome.

ICUs, however, are becoming increasingly effective in preventing the full expression of this phenotype (ie, early death). Some patients rapidly recover, but most patients linger in the ICU with manageable organ dysfunctions for prolonged periods and develop CCI. A substantial portion of these CCI patients (40%–60%) exhibit ongoing protein catabolism with poor nutritional status, poor wound healing, and recurrent infections.[8,9] In addition, they have persistent low-grade inflammation, with defects in innate and adaptive immunity. It is likely this response is driven in large part by bone marrow dysfunction, with expansion and release of the myeloid-derived suppressor cells (MDSCs).[42]

Although expansion of the MDSC population can be explained in part by increased myelopoiesis, defects in the maturation and differentiation of this cell lineage render them ineffective at fighting infections and are responsible for producing a persistent low-level inflammatory state through cytokine secretion. The University of Florida Sepsis and Critical Illness Research Center is investigating the genomic makeup of PICS, trying to predict patients at high risk of developing PICS, elucidate the role of MDSCs, and recommend possible interventions. With a recently awarded a P50 grant by National Institute of General Medical Sciences entitled, "PICS: A New Horizon for Surgical Critical Care," the funding will provide a strong foundation to make this achievement possible. It is believed that PICS patients who are ultimately discharged to long-term acute care facilities rarely rehabilitate, hardly ever return to functional life, and usually experience prolonged decline and an indolent death.[36,39,43-52]

Abdominal Compartment Syndrome

Intra-abdominal pressure (IAP) is monitored by urinary bladder pressure measurements (gold standard). As these pressures approach 20 to 25 cm H_2O, organ dysfunction begins (see Chapter 42). Although intra-abdominal hypertension can occur during the entire ICU stay, IAP on the first day of admission appears to have greatest impact on mortality.[53] When IAP is coupled with any organ dysfunction, by definition, this is ACS. There are three types of ACS: primary, secondary, and tertiary.[2,3,54-56] Primary ACS occurs in patients with abdominal injuries who typically have undergone "damage control" laparotomy (where obvious bleeding is rapidly controlled and the abdomen is packed) and have entered the "bloody vicious cycle" of coagulopathy, acidosis, and hypothermia, which promotes ongoing bleeding (see Chapter 16). Intra-abdominal sepsis with significant volume free air, contamination, ascites, and/or bowel wall edema can also present as primary ACS. Accumulation of blood, worsening bowel edema from resuscitation, and the presence of intra-abdominal packs all contribute to increasing IAP that causes ACS. Secondary ACS occurs when extra-abdominal bleeding (eg, mangled extremity or pulmonary hilar gunshot wound) or extra-abdominal pathology (eg, burns or necrotizing soft tissue infections) requires massive resuscitation that causes bowel edema, increasing IAP and eventually resulting in secondary ACS. Tertiary ACS occurs in the setting of a temporary abdominal closure and ongoing IAP increases or a new ACS that follows closure of the abdominal wall after previous open abdomen care. The increased use of the open abdomen has likely moved this scenario from a gastrointestinal failure to a treatment for a current or potential gastrointestinal failure. However, what is important to note is that markedly elevated IAP decreases gut perfusion, which may adversely affect a variety of gut functions. Clinical studies have clearly documented the poor outcome of patients developing ACS and the frequent association of ACS and MOF.[2] In addition, the decreased gut perfusion creates a potential cycle of ileus or decreased gut transport and less than ideal nutrient

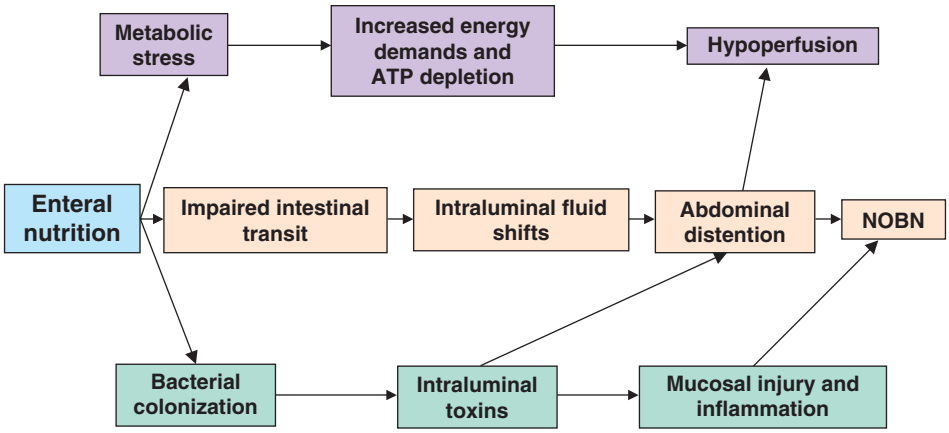

FIGURE 60-2 Proposed pathogenesis of nonocclusive bowel necrosis (NOBN). ATP, adenosine triphosphate.

absorption. Clinically, these patients typically receive less than goal caloric provision.[57,58]

Nonocclusive Small Bowel Necrosis

Nonocclusive small bowel necrosis (NOBN) is a relatively rare but frequently fatal entity that is associated with critical illness. In addition, vasoactive agents while in the critical care unit and drugs such as methamphetamines can create an environment of normal patent arteries, but "clamped" down vessels can decrease oxygen supply. Combined with any shock state that early on diverts blood away from the gut, an imbalance between supply and demand can create ischemia or even necrosis. In some studies, the use of enteral nutrition in critically ill patients has been implicated.[59]

Patients present with complaints of cramping abdominal pain and progressive distention, at times associated with SIRS or severe SIRS. ACS, pressor requirement, and oliguria can also be markers of NOBN.[60] Computed tomography (CT) may reveal pneumatosis intestinalis or thickened dilated bowel in more advanced stages. In 2015, Lasierra in a review described a need for clinical, laboratory, and radiographic evaluation in critically ill patients with a potential for NOBN.[61] For patients who progress and require exploratory celiotomy, ischemia and/or patchy necrosis of the small bowel is found, usually on the antimesenteric border. Pathologic analysis of the resected specimens yields a spectrum of findings from acute inflammation with mucosal ulceration to transmural necrosis and multiple perforations.

The association with enteral nutrition indicates that inappropriate administration of nutrients into a dysfunctional gut may play a pathogenic role. There are three popular hypotheses (Fig. 60-2).[9] First, metabolically compromised enterocytes become adenosine triphosphate (ATP) depleted as a result of increased energy demands induced by the absorption of intraluminal nutrients, leading to hypoperfusion and subsequent NOBN.[62] The second hypothesis is that when nutrients are delivered into the dysmotile small bowel, fluid shifts into the lumen as a result of the presence of hyperosmolar enteral formula, leading to intestinal distention, which when severe progresses to NOBN. Third, bacterial colonization leads to intraluminal toxin accumulation, which can result in mucosal injury and inflammation and, if significant, NOBN. Despite these hypotheses, it is well accepted that a critically ill surgical/trauma patient should receive appropriate euvolemic resuscitation and early (<48 hours) initiation of enteral nutrition. Most of the time, the benefits of this early initiation of enteral nutrition outweigh the risk of small bowel ischemia or necrosis.

SPECIFIC GUT DYSFUNCTIONS

The gut is a complex organ that performs a variety of functions, some of which are vital for ultimate survival of critically ill patients (ie, barrier function, immune competence, and metabolic regulation). Unfortunately, gut dysfunction in critically injured patients is poorly characterized, and routine monitoring of gut function is crude. Currently, the best parameter of normal gut function is tolerance to enteral nutrition (see Chapter 62). Intolerance results in a spectrum of critically ill patients from stress gastritis to intolerance of tube feeds manifested as either distention, diarrhea, or obstipation. First, tolerance to enteral nutrition requires integrative gut functioning (eg, secretion, digestion, motility, absorption). Second, locally administered nutrients improve perfusion and optimize the recovery of other vital gut functions (eg, motility, barrier function, mucosal immunity). Third, tolerance correlates with patient outcome, and improving tolerance will likely improve patient outcome. Fourth, refined therapeutic interventions to improve enteral nutrition tolerance will lessen the need to use parenteral nutrition and decrease its associated complications (see Chapter 62).

Gut dysfunction certainly contributes to enteral feeding intolerance. The pathophysiology and pathogenesis of each dysfunction and how they may be monitored clinically will be reviewed. This can provide the rationale for therapeutic strategies to improve tolerance to enteral nutrition.

Gastroesophageal Reflux

GER is an important contributing factor to aspiration of enteral feedings, which is a common etiology of pneumonia in ICU patients. GER occurs for several reasons in the critically ill patient. Decreases in resistance at the lower esophageal sphincter can be due to relaxation of the sphincter muscle in response to many stimuli, including mediators released during injury and resuscitation. Additional contributing factors include (1) forced supine position, (2) the presence of a nasoenteric tube, (3) hyperglycemia, and (4) narcotics. Pressure differences between the stomach and esophagus become great enough to overcome the resistance offered by the lower esophageal sphincter. GER may occur as a result of increased IAPs due to crystalloid resuscitation and bowel edema.

Delayed gastric emptying in critically ill patients results in abnormal delivery, digestion, and potentially absorption of nutrition. There are preclinical data regarding the pathophysiologic role of the enterohormones peptide YY and ghrelin in this population. Studies have not clearly demonstrated a connection, but critically ill patents' ghrelin levels tend to be lower.[63]

Historically, clinical monitors include laboratory testing for presence of glucose in tracheal secretions or observing blue food dye that has been added to the enteral formula in the setting of tracheal aspiration. Detection of glucose lacks specificity. False-positive results can occur with high serum glucose levels or presence of blood in tracheal secretions. The blue food dye is no longer recommended and carries a US Food and Drug Administration black box warning. It was also poorly standardized, as indiscriminate amounts were added to tube feeds, lacking sensitivity, and it is considered dangerous to patients. Several reports document that absorption of blue food dye in critically ill patients is associated with death.[64] This is potentially associated with toxic effects that blue food dye has on mitochondrial function. A recent consensus conference recommended that the technique be abandoned.[65] Unfortunately, there are no simple monitors of GER other than observing for vomiting or regurgitation, which are not very sensitive. The head of the bed should be elevated 30° to 45° to decrease the risk that when GER occurs that it is less likely to result in pulmonary aspiration (see Chapter 59). Gastric residual volumes (GRVs) should be monitored with the presumption that a distended stomach will lead to higher volume GER.

Gastroparesis and Duodenogastric Reflux

Gastroparesis is common in ICU patients and predisposes to increased duodenogastric reflux (a potential contributing factor for gastric alkalization) and GER (a contributing factor for aspiration). The mechanisms responsible for gastroparesis in critical illness have not been well studied. The current model is an impairment of the esophageal peristalsis. In addition, there is a hypotensive lower esophageal sphincter pressure. This is in the setting of isolated pyloric pressure waves resulting in increased pyloric tone.[66] This results in gastric stasis and, in the setting of H_2-receptor blockers or proton pump inhibitors, bacterial overgrowth. In addition, potential factors include (1) medications (eg, narcotics, pressors), (2) sepsis mediators (eg, nitric oxide), (3) hyperglycemia, and (4) increased intracranial pressure/traumatic brain injury.

The common clinical monitors for gastroparesis are intermittent measurement of GRVs when feeding into the stomach or measurement of continuous suction nasogastric tube output when feeding postpyloric. First, it must be performed with a standard sump nasogastric tube. Small-bore single-lumen tubes do not adequately empty the stomach and should not be used to measure GRV. The practice of using GRV is poorly standardized and is a major obstacle to advancing the rate of enteral nutrition.[67] GRVs appear to correlate poorly with gastric function, and GRVs less than 200 mL generally are well tolerated. GRVs of 200 to 500 mL should prompt careful clinical assessment and the initiation of a prokinetic agent. With GRVs of more than 500 mL, enteral nutrition was traditionally stopped. After clinical assessment excludes small bowel ileus or obstruction, placement of a post–ligament of Treitz feeding tube should be considered (see Chapter 62). However, a study by Reignier et al[68] in *JAMA* demonstrated that not monitoring GVR and allowing patients to advance to goal tube feed rates for adequate nutritional supplementation was not inferior to checking GVR in preventing ventilator-associated pneumonia.

Critically ill mechanically ventilated patients are known to have a high incidence of gastroduodenal reflux. It is seen in patients who are mechanically ventilated while being fed via nasojejunal feeding tubes and can occur during fasting and feeding periods.[69,70] In a study of antral, duodenal, and proximal jejunal motility, Toumadre et al[71] demonstrated that postoperative gastroparesis occurs after major abdominal surgery and is associated with discoordinated duodenal contractions, of which 20% migrated in a retrograde fashion. Heyland et al[72] administered radiolabeled enteral formulas through a standard postpyloric nasoenteric feeding tube in ventilated ICU patients and documented an 80% rate of radioisotope label reflux into the stomach, 25% reflux rate into the esophagus, and a 4% reflux rate into the lung. Finally, Wilmer et al[70] reported monitoring bile reflux in the esophagus of ventilated ICU patients using a fiberoptic spectrophotometer that detects and quantifies bilirubin concentration. Endoscopy was performed and documented erosive esophagitis in half of the patients, of whom 15% had pathologic acid reflux and 100% had pathologic bile reflux. These studies provide convincing evidence that duodenogastric reflux is a common event in ICU patients.

Gastric Alkalization

After major traumatic injuries, patients can develop gastric luminal alkalization, which may be related to bile acid duodenal gastric reflux.[73,74] The stomach, through secretion of hydrochloric acid, normally has a pH below 4.0. This acid environment has been correlated with the relatively low

bacterial counts found in the stomach. Reviews of several studies have shown that alkalization of the stomach through the use of antacids, H_2 antagonists, and proton pump inhibitors results in gastric colonization by bacteria not normally found in the stomach, and several, but not all, studies have shown that gastric colonization predisposes patients to ventilator-associated pneumonia[75] and can increase the risk of community-acquired *Clostridium difficile*–associated disease.[76]

Several animal studies have shown that both lipopolysaccharide administration and mesenteric ischemia/reperfusion result in the gastric accumulation of an alkaline fluid.[77] This most likely results from a decrease in gastric acid secretion with continued gastric bicarbonate secretion and the reflux of duodenal contents into the stomach. Even more recently, reports have indicated that the pH of gastric contents in trauma patients is also elevated, possibly due to similar events.[73] Thus, even without the administration of antacids or inhibitors of acid secretion, gastric alkalization and bacterial colonization of the stomach are likely in this group of patients. When this is combined with the gastroparesis often seen in these patients (see earlier discussion), it is easy to envision the stomach becoming a major source of bacteria for ventilator-assisted pneumonia and perhaps translocation to other organs.

Impaired Mucosal Perfusion

Current resuscitation techniques from hemorrhagic shock cause a persistent and progressive splanchnic vasoconstriction and hypoperfusion despite hemodynamic restoration with intravenous fluid therapy or blood products. The pathophysiologic response to shock is a decrease in splanchnic blood flow. Hemorrhage-induced activation of endothelial cell Na^+/H^+ exchanger results in cellular swelling, which physically impedes capillary filling and compromises gut perfusion. Paradoxical endothelial cell swelling occurs early during hemorrhagic shock because of activation of the Na^+/H^+ exchanger. This cellular edema, which is not resolved by correction of the vascular volume deficit, explains the persistent postresuscitation endothelial cell dysfunction and gut hypoperfusion.[78]

The gut mucosa appears to be especially vulnerable to injury during hypoperfusion. The arterioles and venules in the small bowel mucosal villi form "hairpin loops." This anatomic arrangement improves absorptive function, but it also permits a countercurrent exchange of oxygen from the arterioles to the venules in the proximal villus. Under hypoperfused conditions, a proximal "steal" of oxygen is believed to reduce the Po_2 at the tip of the villi to zero. The gut mucosa is further injured during reperfusion by reactive oxygen metabolites and recruitment of activated neutrophils (see Chapter 63). This mucosal injury, however, appears to quickly repair itself. Mucosal blood flow does not always return to baseline with resuscitation, and this is in part due to defective vasorelaxation. The gut mucosa is also vulnerable to recurrent episodes of hypoperfusion from ACS, sepsis, and use of vasoactive drugs. Whether recurrent hypoperfusion results in additional ischemia/reperfusion injury is not known, but it is reasonable to assume that hypoperfusion would decrease gut nutrient absorption and render the patient more susceptible to NOBN.

Monitoring gastric mucosal perfusion in the clinical setting can be done by gastric tonometry. With hypoperfusion, intramucosal carbon dioxide (CO_2) increases due to insufficient clearance of CO_2 produced by aerobic metabolism or due to buffering of extra hydrogen ions produced in anaerobic metabolism. As intramucosal CO_2 accumulates, it diffuses into the lumen of the stomach. The tonometer measures the CO_2 that equilibrates in a saline-filled balloon (a newer monitor uses an air-filled balloon) that sits in the stomach. This is the regional CO_2 tension ($PrCO_2$) and is assumed to equal the intramucosal CO_2 tension. Using this measured $PrCO_2$ and assuming that arterial bicarbonate equals intramucosal bicarbonate, the intramucosal pH (pHi) is calculated by using the Henderson-Hasselbalch equation. Numerous studies have documented that a persistently low pHi (or high $PrCO_2$ level) despite effective systemic resuscitation predicts adverse outcomes and that attempts to resuscitate to correct a low pHi do not favorably influence mortality.[79]

Direct peritoneal resuscitation (DPR) has been shown to increase survival after intestinal ischemia and reperfusion injury in small animal models. DPR has been demonstrated to have significant therapeutic potential for improving mesenteric perfusion, intestinal injury, and the local inflammatory response after the intestinal ischemia/reperfusion event.[80-83]

Impaired Intestinal Transit

Laboratory models of shock, bowel manipulation, and sepsis demonstrate that small bowel transit is impaired.[84] This impairment in turn is expressed as a decrease in the number and/or force of contractions or as an abnormal pattern of contractions. Although the results in animal models are convincing, surprisingly, clinical studies indicate that small bowel motility and transit are more often than not well preserved after major elective and emergency laparotomies.[85] This observation coupled with the observation that small bowel absorption of simple nutrients is relatively intact provided the rationale for early jejunal feeding (see Chapter 62).

In addition to injury, patients undergoing laparotomy are likely to have postoperative ileus, which is assumed to result from myeloid cells infiltrating the intestinal muscularis externa. In a small animal study, during the postlaparotomy recovery phase, monocyte-derived MΦs acquired proresolving features that aided in the resolution of inflammation. This study showed a critical role for monocyte-derived MΦs in restoring intestinal homeostasis after surgical trauma.[86]

Clinical studies have documented that the majority of critically ill patients tolerate early jejunal feeding.[87] In a recent study, severely injured patients had jejunal manometers and feeding tubes placed at secondary laparotomy.[85] Surprisingly, 50% had fasting patterns of motility that included components of the normal migrating motor complexes (MMCs). These patients tolerated advancements of enteral nutrition without problems. The other 50% who did not have fasting MMCs did not tolerate early advancement of enteral

nutrition. Of note, none of the patients converted to a normal-fed pattern of motility once they reached full-dose enteral feeding. This could be due to infusion of caloric loads insufficient to bring about conversion. On the other hand, the failure to develop fed activity, a pattern of motility promoting mixing and absorption, might explain why diarrhea is a common problem in this patient group.

Although manometry can be used to monitor motility, it is not practical. Unfortunately, simpler indicators of motility such as the presence of bowel sounds or the passing of flatus are unreliable. Other minimally invasive methods to monitor transit are needed. Contrast studies through the feeding tubes are relatively simple but not validated.

Impaired Gut Absorptive Capacity

Small bowel absorption of glucose, lipids, and amino acids is depressed after trauma and sepsis.[88] Multiple factors have been identified including (1) cytosolic calcium overload, (2) ATP depletion, (3) diminished brush border enzyme activity, (4) decreased carrier activity, (5) decreased absorptive epithelial surface area, and (6) hypoalbuminemia. In an animal study, intestinal ischemia/reperfusion caused significant mucosal injury and significant depletion of mucosal ATP.[51] When this was combined with exposure of the bowel to a nonmetabolizable nutrient, the damage and ATP depletion were more severe, and the absorption of glucose was impaired. In contrast, exposure of the bowel to metabolizable nutrients (eg, glucose) preserved ATP levels, protected against mucosal injury, and improved gut absorptive capacity (GAC).[89]

There does appear to be some degree of GAC that is maintained after injury. In a small animal model after burn injury, intestinal absorption of the dipeptides was preserved 24 hours following thermal injury.[90] These findings support the use of less complex enteral diets in the critically ill patient.

The clinical significance of these observations remains unclear because most patients tolerate enteral nutrition when delivered into the small bowel. However, decreased GAC may be a cause of diarrhea and may explain why patients commonly experience diarrhea with reinstitution of enteral nutrition after prolonged bowel rest. Unfortunately, there are no easily performed clinical monitors for GAC. D-Xylose absorption is clinically available but is used most frequently to diagnose chronic malabsorption.

Diarrhea may be indicative of depressed GAC, but there are other causes for diarrhea in the critically ill. These include impaired transit, bacterial overgrowth (ie, presence of *C difficile*), contaminated enteral formulas, abnormal colonic responses to enteral nutrition (ie, ascending colon secretion rather than absorption, or impaired distal colon motor activity), and administration of drugs that contain sorbitol (eg, medication elixirs) or magnesium (eg, antacids).

Increased Gut Permeability

Trauma results in hemorrhagic shock, which leads to mucus damage and gut dysfunction through the generation of free radical species.[91]

Enhanced paracellular permeability represents a type of barrier dysfunction that allows increased passage between viable cells and may induce an inflammatory cascade. The major components of the epithelial barrier are tight junctions, which bind cells together and serve as the gateways to the underlying paracellular spaces. The integrity of the tight junctions is modulated by the actin cytoskeleton. Under conditions of ATP depletion, such as would occur during shock, disruption of the actin cytoskeleton with consequent opening of the tight junctions and loss of the integrity of the permeability barrier can occur.[62]

Intestinal barrier dysfunction has been suggested as a means by which inflammatory cytokines can lead to SIRS and MOF. Additionally, increased intestinal permeability has been documented in high-risk patients after burns, sepsis, and shock, in most but not all studies.[92]

Decreased Gut Mucosal Immunity

During periods of intestinal disuse, critically injured patients are subject to a reduction in gut mucosal immunity. Lack of enteral stimulation (eg, during starvation or with use of parenteral nutrition) quickly leads to lack of immunologic protection by mucosal-associated lymphoid tissue (MALT), which normally provides protection for both the gastrointestinal (GI) and respiratory tracts against microbial flora and infectious pathogens. Kudsk[93] has demonstrated a link between intestinal immunoglobulin A (IgA), intestinal cytokine production, and the vascular endothelium of the GI tract. With enteral stimulation, IL-4 and IL-10 production from the lamina propria of the small intestine stimulates production of IgA on the mucosal surface and inhibits intracellular adhesion molecule-1 (ICAM-1) of the vascular endothelium and subsequent neutrophil-associated inflammation and injury.[93]

Increased Gut Colonization

With progressive gut dysfunction, the normally sterile upper GI tract becomes colonized with organisms that become pathogens in nosocomial infections. Gastric alkalization, paralytic ileus, loss of colonization resistance due to broad-spectrum antibiotics, and decreased local gut immunity have all been proposed mechanisms by which the upper GI tract becomes colonized.

In an effort to decrease the incidence of infectious complications, selective digestive decontamination (SDD) has been proposed. SDD generally consists of topical nonabsorbed antibiotics along with a short course of parenteral antibiotics. There have been numerous clinical trials and at least six meta-analyses addressing this practice. Most studies have examined the incidence of ventilator-associated pneumonias and mortality and, in general, have demonstrated a decrease in both. Despite rather impressive results of these trials, most of which have been performed in Europe, intensivists in the United States have generally avoided use of SDD due to concerns of inducing antimicrobial resistance.[94]

The human microbiome has emerged as a critical component of health and disease, and the microbial communities

within the intestinal tract appear to contribute to it.[95] Head injury is known to induce a hypercatabolic state, disimmunity, and septic complications that increase morbidity and mortality. In a small animal model, head injury changed the gut microbiota homeostasis and thus could contribute to infection.[96]

Gut Edema

From the 1950s and through the Korean War and the Vietnam conflict, intravenous fluid administration was associated with improved mortality.[97,98] The mechanism at that point in time was improved resuscitation via improved cardiac output and subsequently better gut perfusion. However, the lessons learned after the Vietnam conflict in both clinical and laboratory models included the concept that excess fluids produced acute respiratory distress syndrome and GI dysfunction in the form of ascites, bowel edema, ACS, and at times, poor healing of bowel anastomosis. A number of clinical studies, although not performed in resuscitated trauma patients, have demonstrated the benefit of fluid restriction on postoperative bowel function and complications.[99]

The precise mechanisms by which bowel edema adversely affects bowel function have not been fully elucidated. Laboratory investigations by Moore-Olufemi et al[100] have shown that bowel edema alone (not associated with gut ischemia/reperfusion or hemorrhagic shock) is associated with impaired intestinal transit. This can be reversed with enteral feeding. In addition, evidence indicates that enteral nutrition with the open abdomen is associated with fewer complications and early closure of the abdomen.[101] This reinforces the notion that although enteral nutrition may be associated with small bowel ischemia/necrosis, the benefits of early enteral nutrition are greater than the risks.

During the resuscitation of the hemorrhagic shock patient, a multipronged approach has been adopted to prevent overresuscitation, bowel edema, ACS, bowel ischemia, and the perpetuation of the open abdomen. Through improved timely hemorrhage control, damage control resuscitation with the use of early blood products in the form a massive transfusion protocol, and less crystalloid resuscitation, patients have suffered subsequently less open abdomen care and greater primary fascial closure. This also has translated into less GI failure, by means of fewer ACS and ICU days, less enterocutaneous fistulae, and subsequently fewer long-term (9–12+ months) abdominal reconstruction projects.

STRATEGIES TO IMPROVE GUT DYSFUNCTION

Gut-Specific Resuscitation and Monitoring for Abdominal Compartment Syndrome

Traditional resuscitation is aimed at optimizing systemic perfusion, with initial administration of 2 L of isotonic crystalloids and then addition of packed red blood cells at a ratio of 3:1 crystalloid to blood (see Chapter 16).

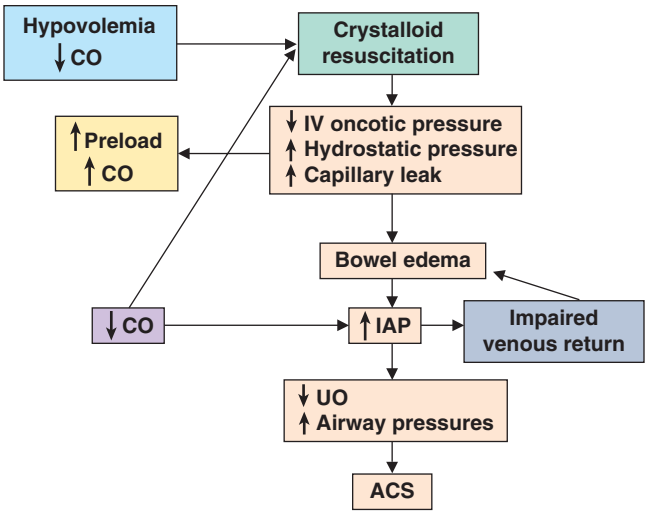

FIGURE 60-3 Crystalloid vicious cycle leading to abdominal compartment syndrome. ACS, abdominal compartment syndrome; CO, cardiac output; IAP, intra-abdominal pressure; IV, intravenous; UO, urine output.

If–then resuscitation protocols need to be devised to optimize early gut perfusion and prevent reperfusion injury. Although this approach is effective in some patients, it is associated with problematic bowel edema in patients at high risk for MOF and subsequent shock-induced gut hypoperfusion, leading to gut dysfunction. As edema worsens and IAP increases, bowel perfusion becomes further impaired, setting up a vicious cycle that leads to ACS (Fig. 60-3).[55]

IAPs should be measured routinely in patients in whom significant resuscitation is anticipated, because intra-abdominal hypertension can be predicted early in at-risk patients.[21] A high index of suspicion and knowledge of these predictors is warranted. In the past, surgeons have not decompressed the abdomen until clear signs of organ dysfunction were present (ie, decreased urine output, decreased Pao_2/Fio_2 ratio, decreased cardiac output despite volume loading) in part because of fear of creating an open abdomen with consequent need for planned ventral hernia and delayed reconstruction (see Chapter 42). Thus, based on prediction models,[3] patients who meet defined high-risk criteria and require ongoing aggressive resuscitation may be considered for a "presumptive" decompressive laparotomy. However, medical or nonsurgical strategies have been proposed to address and effectively treat rising IAPs, subsequently avoiding an open abdomen. If the medical strategy is not effective, early decompressive laparotomy can occur. In fact, a recent prospective study demonstrated that early decompression in at-risk patients reduced mortality from intra-abdominal hypertension/ACS.[102] In addition, current critical care treatment plans for patients with an open abdomen include strategies to address not only organ dysfunctions that are affected by intra-abdominal hypertension but also the actual IAP and the systemic inflammation associated with ACS. Included are resuscitation strategies that utilize early use of blood products

for hemorrhage and coagulopathy, which may lessen bowel edema and therefore reduce the incidence of ACS.[103]

Analgesics and Sedatives

There are three types of opioid receptors—δ, κ, and μ—and all have been identified as having GI side effects including delayed gastric emptying and delayed transit time in both the small bowel and colon. One major cause of ileus is stress-related stimulation of opioid receptors. In animal models and humans, both endogenously released and exogenously administered opioids act on receptors in both the central nervous system and in the enteric nervous system to alter intestinal function, especially motility. Although actions at both the central nervous system and enteric nervous system are involved, recent studies indicate that if opioid actions at the enteric nervous system are blocked, ileus may be prevented or resolved without interfering with the desired opioid actions on the central nervous system and other systems. An investigational opioid receptor antagonist that has limited systemic absorption after oral administration and minimal access to the central nervous system has been shown to speed recovery of bowel function and shorten the duration of hospitalization after surgery.[104] There is also a peripherally acting μ-receptor antagonist, alvimopan, that has recently been shown in a prospective, randomized controlled trial to accelerate time to recovery of GI function in patients undergoing elective major abdominal surgery.[105] μ-Receptor antagonists are attractive agents for postoperative patients in that they are peripherally acting (gut specific), thereby maintaining centrally mediated pain reduction. A recent Cochrane Review on μ-opioid antagonists for opioid-induced bowel dysfunction concluded that both alvimopan and methylnaltrexone were effective in reversing increased transit time and constipation. Additionally, alvimopan is safe and efficacious in treating postoperative ileus.[106] However, long-term data are lacking, and further studies are indicated. Both of these agents need to be studied in patients at high risk for postoperative bowel dysfunction, particularly patients who have undergone major resuscitation from shock.

Ketamine, an antagonist of the *N*-methyl-D-aspartate receptor, combines both analgesic and sedative effects and may represent an alternative to benzodiazepines for sedation and opioids for pain control in ICU patients. There are reports in burn patients of the opiate-sparing effect of ketamine minimizing prolonged gut dysfunction and ileus.[107] Additionally, both proinflammatory cytokines and mediators have been suppressed in laboratory models of sepsis following ketamine administration.[108]

ENTERAL AGENTS

Benefits of Enteral Versus Parenteral Nutrition

Several prospective randomized controlled trials (PRCTs) performed in the late 1980s and early 1990s had significant impact on clinical practice in surgical, and particularly trauma, ICUs. These single-institution trials all randomized trauma patients to early enteral nutrition or parenteral nutrition and demonstrated that patients receiving early enteral nutrition had significantly fewer infectious complications (see Chapter 62). A meta-analysis that combined data from eight PRCTs (six published and two unpublished) was then conducted to assess the nutritional equivalence of enteral nutrition compared to parenteral nutrition in high-risk trauma and/or postoperative patients.[22] Similar to the single-institution trials, fewer infectious complications developed in patients receiving enteral nutrition. Even when patients with catheter-related sepsis were removed from the analysis, a significant difference in infections between groups remained. Taken together, these trials provide convincing evidence that enteral nutrition is preferred to parenteral nutrition in patients sustaining major torso trauma. A recent meta-analysis evaluating the effect of early versus delayed enteral nutrition in acutely ill (medical and surgical) patients also confirmed a decrease in infectious complications in patients receiving early enteral nutrition.[109]

Based on available data, we can now explain how enteral nutrition interrupts this sequence of events to prevent late nosocomial infections and MOF. In a variety of models (ie, sepsis, hemorrhagic shock, and gut ischemia/reperfusion), intraluminal nutrients have been shown to reverse shock-induced mucosal hypoperfusion.[110] In the laboratory, we have also shown that enteral nutrition reverses impaired intestinal transit when given after a gut ischemia/reperfusion insult.[111] Improved transit should decrease ileus-induced bacterial colonization. Moreover, enteral nutrition attenuates the gut permeability defect that is induced by critical illness.[112] Finally, and most important, the gut is a very important immunologic organ, and infections may be lessened by feeding the gut. Kang and Kudsk[113] performed a series of laboratory studies that have nicely elucidated a mechanistic explanation of how this occurs. Enteral nutrition supports the function of the MALT that produces 70% of the body's secretory IgA. Naive T and B cells target and enter the gut-associated lymphoid tissue (GALT) where they are sensitized and stimulated by antigens sampled from the gut lumen and thereby become more responsive to potential pathogens in the external environment. These stimulated T and B cells then migrate via mesenteric lymph nodes to the thoracic duct and into the vascular tree for distribution to GALT and extraintestinal sites of MALT. Lack of enteral stimulation (ie, use of total parenteral nutrition [TPN]) causes a rapid and progressive decrease in T and B cells within GALT and simultaneous decreases in intestinal and respiratory IgA levels. Previously resistant TPN–fed laboratory animals, when challenged with pathogens via respiratory tree inoculation, succumb to overwhelming infections. These immunologic defects and susceptibility to infection are reversed within 3 to 5 days after initiating enteral nutrition.

The severely injured or open abdomen patient undergoes tremendous systemic inflammation. Therefore, by at least one definition, this patient is suffering from severe protein-calorie malnutrition.[114] Secondarily, these patients are typically fed

less than goal, even by criteria of permissive underfeeding. Frequent operative interventions and resuscitations typically preclude adequate nutrition provision. Therefore, it is not uncommon for the clinician to ask whether parenteral nutrition should be started.

With the large prospective randomized trial published in 2011, supplementation with early (<48 hours) parenteral nutrition in the critically ill has been generally avoided. Patients who were randomized to early parenteral nutrition had higher costs and increased mortality risk compared to patients who had delayed initiation of parenteral nutrition until day 5 to 7.[115] Similar conclusions were found by Kutsogiannis et al,[116] who stated that critically ill patients receiving parenteral nutrition suffered worse outcomes. However, other studies focusing on supplemental parenteral nutrition have not reached the same conclusions. Harvey et al[117] showed no difference in infectious complications or mortality in the critically ill when parenteral nutrition was initiated within that 48-hour window. Doig et al[118] demonstrated that patients who had a "contraindication" for enteral nutrition and had parenteral nutrition started early also did not suffer an increased risk of mortality. In fact, the early parenteral nutrition group had reduced ventilator days. Two systemic reviews also did not demonstrate an increased mortality between early and late parenteral nutrition.[119,120] Further analysis showed fewer ventilator days but increased infectious complications.

Unfortunately, no study has focused on abdominal surgical patients, much less on those with open abdomen care. Guidelines support early enteral nutrition, even in open abdomen patients, as not only safe but also beneficial.[121] In addition, through extrapolation of the previously mentioned studies, it is recommended to delay parenteral nutrition for approximately 5 to 7 days. In the scenario of the open abdomen for the first 5 to 7 days, attempts should be made to optimize provision of early enteral nutrition, even in low "trophic" (>30% goal) amounts and between operative interventions.[122,123]

Modified Enteral Formulas

Basic and clinical research suggests that the beneficial effects of enteral nutrition can be amplified by supplementing specific nutrients that exert pharmacologic immune-enhancing effects beyond the prevention of acute protein malnutrition. The proposed immune-enhancing agents include glutamine, arginine, omega-3 polyunsaturated fatty acids (PUFAs), and nucleotides, although the individual contributions of each have not been well investigated. Glutamine has received the most attention in the trauma patient and critical care patients with mixed recommendations. It is actively absorbed across the intestinal epithelium and then metabolized in the small bowel to ammonia, citrulline, alanine, and proline and serves as an energy source for the enterocyte. Glutamine is therefore acknowledged to be the preferred fuel of the enterocyte and stimulates lymphocyte and monocyte function. The demand for glutamine is increased during stressed states, and supplementation at pharmacologic doses may be required.

Eight published PRCTs tested high doses (0.3–0.5 g/kg) of enteral glutamine in critically ill patients. The majority demonstrated improved clinical outcomes and primarily decreased infection. Additionally, findings associated with enteral glutamine were decreases in (1) urinary lactulose/mannitol ratios, (2) serum diamine oxidase levels, (3) circulating endotoxin levels, and (4) gram-negative bacteremia, all of which suggest that glutamine is achieving these benefits through a gut-specific mechanism.

In a recent meta-analysis, glutamine (parenteral and enteral) administered to critically ill and surgical patients resulted in a lower mortality, less infectious complications, and shorter hospital stay.[124] High-dose and parenteral glutamine had the greatest effect, although the study was not designed to examine these parameters. A randomized trial of glutamine-enriched enteral nutrition in severely injured patients demonstrated a decrease in pneumonia, sepsis, and bacteremia.[125] However, with heterogeneity of the surgical critically ill patient, some doubt exists about the need for routine supplementation in this patient population.[126] In addition, recent data from a large prospective randomized trial suggested supplemental glutamine in high doses may, in fact, be toxic with increased mortality in patients with acute renal injury and MOF.[127] Although this conclusion included severe sepsis patients, enthusiasm surrounding the use of glutamine has decreased tremendously.

Enteral Glutamine During Shock Resuscitation

Patients in shock are not given enteral nutrition because of concerns over the safety of feeding the hypoperfused small bowel. However, evidence supports the feasibility of enteral nutrition in this setting.[128] In addition, despite the presence of vasopressors, enteral nutrition has been noted to be tolerated and absorbed. An alternative concept that has been studied in the laboratory and then pursued clinically is enteral administration of glutamine in the setting of shock.[129] There are several reasons why this would be beneficial. First, intraluminal glutamine infusion reverses shock-induced splanchnic vasoconstriction. Second, glutamine is a preferred fuel source and promotes protein synthesis in the gut mucosa. Glutamine is also a preferred fuel for lymphocytes and is a precursor for glutathione and nucleotides. Glutathione protects against oxidant stress, and nucleotides are required for rapid cellular proliferation of enterocytes and lymphocytes under stressful conditions. Third, glutamine induces a variety of protective mechanisms. Glutamine protects against oxidant- and cytokine-induced apoptosis.[130] Glutamine has been shown to induce antioxidant enzymes (ie, heat shock protein and heme oxygenase-1). In a rodent gut ischemia/reperfusion model, intraluminal glutamine provided protection via a novel molecular mechanism of activating the anti-inflammatory transcription factor peroxisome proliferator activator receptor gamma (PPARγ).[103] Of note, enteral glutamine also favorably modulates SIRS. In a well-established model in which manipulation of the small bowel initiates local gut inflammation and

dysfunction that ultimately causes remote lung inflammation and dysfunction, pretreatment with oral glutamine abrogates both local and remote events.[131] Finally, glutamine plays a crucial regulatory role in enterocyte proliferation, which restores villous surface area. This is critical in restoring gut digestive and absorptive capacity and the ability to tolerate enteral nutrition. As noted previously, negative studies with glutamine and septic shock have blunted the enthusiasm for its use.

Prokinetic Agents

Because gastroparesis and small and large bowel ileus are commonly seen postoperatively and following resuscitation, they can complicate initiation of enteral feeding. Therefore, agents to restore motility have been sought. Evaluation of such prokinetic agents is difficult because it is not enough to just stimulate contractions, but contractions at adjacent sites must also be coordinated in order for normal digestion, absorption, and transit to take place. Coordinated contractions are under the control of hormonal and neural, both central and peripheral, pathways, and it is these pathways that are affected by the cytokines and other mediators that are upregulated following a traumatic insult. Prokinetic strategies are aimed at either blocking these mediators or overriding them by stimulating normal pathways.

Agents such as erythromycin that act on receptors for motilin, the naturally occurring hormone responsible in part for regulating normal GI motility, have been shown to enhance gastric emptying and intestinal transit in animal models and in some clinical trials. Although clinical studies have documented their effectiveness in promoting gastric emptying, their effectiveness in reducing postoperative ileus has been disappointing.[132] A recent Cochrane Review of systemic-acting prokinetic agents to treat postoperative ileus after abdominal surgery failed to recommend any of the currently available agents.[133] Erythromycin showed uniform absence of effect, whereas there was insufficient evidence to recommend cholecystokinin-like drugs, dopamine-antagonists, propranolol, or vasopressin. Most trials had small sample size and inadequate reporting of methods to draw meaningful conclusions. In addition, it appears that combining agents such as erythromycin and metoclopramide or erythromycin and neostigmine is more effective than using a single agent.[134-136] Lower gastric residual volumes and increased tolerance to enteral nutrition are appreciated; however, outcomes such as infections and mortality remain unchanged. However, Taylor et al[137] showed that distal feeding access was superior after failed single-agent metoclopramide was used. In addition, although gastric versus distal access feeds continue to be a debate, gastric feeds have been shown to be safe, if not more than adequate, to start enteral nutrition.[138]

A promising new peptide, ghrelin, has been shown in a rodent model to not only accelerate gastric emptying and small intestinal transit in unoperated animals, but also reverse postoperative gastric ileus.[106] Ghrelin has been shown to be decreased in critically ill patients.[63] However, small clinical trials examining the efficacy in postoperative and critically injured patients have yet to demonstrate enough benefit for ghrelin or its analogs/agonists to be used in the critical care population.[139,140]

Antioxidants

The cycle of organ hypoperfusion during shock followed by reperfusion during resuscitation results in the formation of detrimental reactive oxygen species. Thus, it is logical to propose that administration of antioxidants could prove beneficial. In many animal models, administration of agents such as superoxide dismutase, ethyl pyruvate, and melatonin limits damage induced by ischemia/reperfusion.[84] In an animal study, administration of α-melanocyte–stimulating hormone preserved both the function and the structural integrity of the intestine following mesenteric ischemia/reperfusion.[84]

Clinically, antioxidant replacement strategies have demonstrated overall reduction in mortality and specific end-organ protection. A randomized prospective trial of antioxidant supplementation with vitamins C and E to critically ill surgical patients, primarily trauma patients, demonstrated a significant reduction in organ failure.[141] Likewise, Collier et al[142] demonstrated a significant risk reduction in mortality in severely injured patients who received high-dose antioxidants compared to historical controls. A systemic review of aggregated clinical trials in critically ill patients demonstrated an overall reduction in mortality with antioxidant supplementation.[143] After subgroup analysis, selenium appeared to be the predominant antioxidant responsible for the positive effects.

The REDOXS study demonstrated worse outcome with glutamine and antioxidants in severe sepsis, especially with acute kidney injury. In addition, the effect appeared to be related to glutamine and not the antioxidants.[127] Despite these results, the use of antioxidants prior to sepsis, organ failure, or specifically kidney injury has been discouraged clinically at the bedside. When the literature is arrogated, early use of antioxidants is still supported in the trauma population.

Probiotics and Prebiotics

A probiotic is defined as a live microbial feed supplement that improves the host's intestinal microbial balance. Commonly used probiotics include lactobacilli, bifidobacteria, and *Saccharomyces*. A prebiotic is defined as a nondigestible food ingredient that beneficially affects the host by selectively stimulating the growth and/or activity of specific bacteria in the colon. Probiotics are usually nondigestible oligosaccharides. The most extensively studied are the fructooligosaccharides (FOS) such as oligofructose. FOS are fermented in the colon, which promotes the proliferation of bifidobacteria with a reduction in clostridia and fusobacteria. Manipulation of the colonic microflora may reduce the incidence of enteral nutrition–associated diarrhea by suppressing enteropathogens.

Clinically, enteric formulas containing probiotics have shown a significant reduction in a variety of postoperative complications. One randomized clinical trial demonstrated

a significant decrease in postoperative infections in patients who have undergone major abdominal surgery and received postoperative enteral formulas containing probiotics.[144] Results of clinical trials employing probiotics, prebiotics, or a combination in critically ill and burn patients, however, have been disappointing.[145]

Synbiotics are a combination of pro- and prebiotics, and the combination is postulated to improve the survival of the probiotic organism by having a specific substrate readily available for probiotic fermentation. In a study in trauma patients, patients receiving symbiotic supplementation had decreased intestinal permeability and lower combined infection rates than those receiving other immunomodulating formulas. The authors postulated that the presence of synbiotics in the GI tract reduced pathogenic flora and thereby decreased the incidence of pneumonia.[146] These findings represent the immunomodulatory potential for synbiotic enteral formulas in the setting of severe systemic inflammation. A recent study by Shimizu et al[147] demonstrated that, in septic patients, prophylactic synbiotics modulated the gut microbiota and environment and may have preventive effects on the incidence of enteritis and ventilator-associated pneumonia.[147]

Great enthusiasm has emerged regarding changes with microbiota in the critically ill.[148] However, more than one meta-analysis failed to demonstrate sufficient evidence to recommend prebiotic, probiotics, or synbiotics to critically ill patients.[149]

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Renal Failure

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KEY POINTS

- Eighty-five percent of renal blood flow perfuses the outer cortical glomeruli, whereas the remaining 15% perfuses the juxtamedullary glomeruli.
- The final concentration and volume of urine vary with plasma volume, serum osmolality, release of antidiuretic hormone (ADH), and other factors.
- Renal failure is classified by the RIFLE (Risk, Injury, Failure, Loss, and End Stage), AKIN (Acute Kidney Injury Network), or KDIGO (Kidney Disease: Improving Global Outcomes) systems.
- The incidence of renal failure after trauma is 3% to 9%.
- Causes of renal failure after trauma include hemorrhage, hypovolemia, functional prerenal renal failure, toxins (eg, antibiotics), contrast nephropathy, sepsis, and rhabdomyolysis.
- Current KDIGO guidelines promote the use of isotonic crystalloid solutions over colloids for the initial volume resuscitation in patients at risk for or with acute kidney injury.
- Overall energy use is not affected by renal failure alone, and calorie repletion should not be increased based solely on this finding.
- Much as with patients with chronic kidney disease, indications for acute renal replacement therapy include hyperkalemia resistant to usual therapy, acidosis, uremia, and severe volume overload.

INTRODUCTION

Fluid is distributed in the human body through three separate compartments: the intracellular fluid (ICF), and the extracellular fluid (ECF), which is itself broken down into the interstitial and intravascular compartments. The majority of the fluid, roughly two-thirds, is in the intracellular compartment. The extracellular one-third is itself divided such that roughly one-third to one-fourth of the fluid is in the intravascular space as the solute portion of plasma and the remaining two-thirds to three-quarters is interstitial fluid. There is a relatively volumetrically insignificant volume of fluid in the transcellular space, which is to say secretions, cerebrospinal fluid, and ocular fluid.[1,2]

Fluid and some small molecules move in and out of the intravascular space, both in normal and pathologic states. In normal cases, fluid outside of the vasculature distributes between the interstitial and intracellular compartments and can return to the vascular compartment. Vascular endothelial injury leads to vascular leak, with escape of larger molecules and proteins that would normally stay within the vasculature. This extravascular fluid accumulates and becomes edema when more fluid that can be reabsorbed through the lymphatic system is lost into the interstitial space.[1] Functionally, this space, when pathologically engaged, is the so-called "third space."[2] This fluid is trapped and is essentially nonfunctional, or unable to participate in the essential functions of intravascular volume expansion.

In the setting of direct injury, such as traumatic swelling, this can be well circumscribed and have a negligible volume. In critical pathologic states, especially sepsis, this is a widespread process that makes volume estimation and replacement a complex process. There is no clear measure of "third-space" losses, only surrogates, such as ultrasound, physical exam, or patient weight. When this extravascular fluid accumulation happens in organ tissues, this can also contribute to organ dysfunction, as seen in bowel edema or pulmonary edema, which can lead to ileus or respiratory compromise. These states exist in an even more complex interplay with the body's endocrine system and the kidney.

In the prototypical 70-kg person, the 42 L of fluid is divided into 28 L into the ICF and 14 L in ECF, with 11 L in the interstitial fluid space (IFS). The cardiac output in this 70-kg person is 5 L/min with 20% of this flow going to the

kidneys, which leads to a renal blood flow (RBF) of 1250 mL/min. This unusually large RBF reflects the vital renal role in regulating the ICF and ECF, controlling fluid and electrolyte balance, modulating acid-base balance, and excreting undesirable catabolites.[3,4]

NORMAL RENAL FUNCTION

The 1250 mL/min of RBF passes through the renal artery into the interlobar, the arcuate, and, finally, the intralobar arteries; 85% of the RBF perfuses the outer cortical glomeruli; the remaining 15% of RBF perfuses the juxtamedullary glomeruli (Fig. 61-1). The glomeruli (Bowman's capsules) are like capillaries except that proteins, normally, are not filtered.[4,5] While passing through the glomeruli, 20% of the plasma is filtered as a cell-free, protein-free filtrate. The effective renal plasma flow (ERPF) through these tubular vessels is determined by the clearance of para-aminohippurate (PAH) that is filtered and secreted but not reabsorbed by the renal tubules. Normally, 91% of PAH is cleared in one passage; 9% remains bound to the plasma protein. Renal oxygen consumption parallels ERPF, which averages 650 mL/min. True renal plasma flow is calculated by dividing ERPF by 0.91 and averages 710 mL/min. The extraction ratio of PAH, however, may vary with injury and sepsis.

The normal glomerular filtration rate (GFR) averages 125 mL/min (180 L/d) and is measured by the renal clearance (urine concentration × volume/plasma concentration) of exogenous inulin (C_{in}) or endogenous creatinine (C_{cr}), both of which are completely filtered; the C_{in} is slightly higher than the C_{cr} since creatinine in humans is partially reabsorbed by the renal tubules.[5,6] The work of GFR is performed by the heart. The protein-free filtrate passes through the proximal convoluted tubules, where approximately 80% of the sodium and water are reabsorbed by active sodium transport and the passive movement of water, thereby maintaining an iso-osmolar state. The nonfiltered blood, now protein rich, perfuses the efferent arterioles and the peritubular vessels, and augments tubular reabsorption and secretion.[7]

The juxtaglomerular nephrons that receive 15% of the RBF are unique; each has a loop of Henle with descending and ascending straight segments that pass into the inner

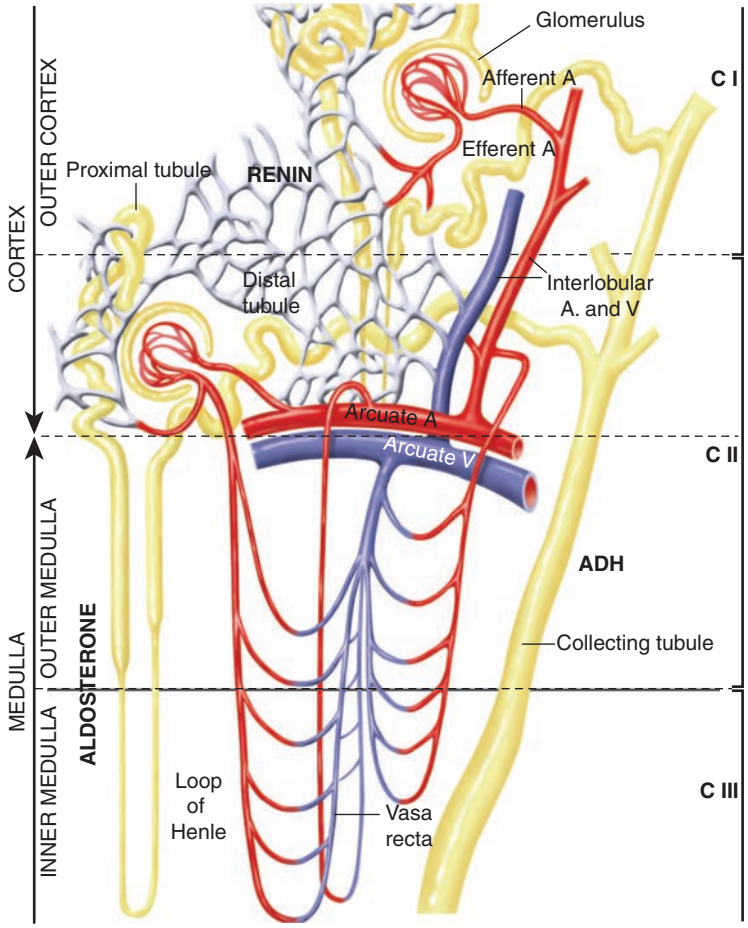

FIGURE 61-1 The kidney is divided into three components. The outer cortex (CI) contains 85% of the glomeruli. The inner cortex/outer medulla (CII) contains the remaining juxtamedullary glomeruli whose peritubular vessels extend to the vasa recta in the inner medulla (CIII) that establishes the hyperosmolality within the loops of Henle.

medulla.[4,5] These segments actively reabsorb sodium against a gradient and, thereby, create a hypertonic medullary interstitium that facilitates the subsequent concentration of glomerular filtrate and the preservation of salt and water in response to antidiuretic hormone (ADH) and aldosterone. Glucose and electrolytes, namely, chloride, phosphate, and potassium, are likewise absorbed at this site. This hypertonic medullary interstitium is further regulated by the peritubular vessels and vasa recta passing close to the loop of Henle (Fig. 61-1). Rapid blood flow through these vessels may cause a "washout" of sodium ions and osmoles, resulting in transient "paralysis" of the renal concentrating mechanism; inadequate blood flow with ischemia injury to these tubular cells impedes sodium reabsorption against a gradient, thus preventing medullary hypertonicity and normal filtrate preservation.[4,5]

Approximately 20% (ie, 36 L/d) of protein-free filtrate and sodium enters the distal convoluting tubules, where additional sodium is reabsorbed by active transport as chloride follows passively.[5,7] This process is facilitated by aldosterone. The distal tubular aldosterone effect is estimated by dividing the free water clearance (C_{H2O}) by the C_{H2O} plus sodium clearance (C_{Na}) in patients with a positive C_{H2O}. When the product is greater than 0.73, the "aldosterone" effect has been blocked. The distal tubule also exchanges sodium for either potassium or hydrogen depending on pH and potassium load. Each distal tubule returns to its own glomerulus at the afferent arteriole where the macula densa and Polkissen body, known as the juxtaglomerular apparatus (JGA), are located. The JGA serves as a "feedback" loop for each nephron affecting sodium and water balance, which are mediated by distal tubular sodium concentration, afferent arteriole pressure, afferent arteriole pulse pressure, and others.[5-8]

After passing through the distal convoluted tubules, the remaining hypotonic filtrate (18 mL/min or 36 L/d) enters the collecting ducts where water reabsorption occurs; this is facilitated by ADH as the filtrate passes through the hypertonic inner medullary interstitium to the renal pelvis.[5] The final concentration and urine volume, therefore, vary with plasma volume, serum osmolality, ADH release, and other factors; urine concentration may range from isosmolar to 1400 mOs/L. Alteration of this highly integrated system occurs with hemorrhagic shock so that the renal concentrating ability may be restricted to 600 mOs/L.[9] The collecting ducts normally reabsorb approximately 17 mL/min or 34 L/d, with the remaining 1 mL/min (1440 mL/d) being excreted as urine.

The renal handling of sodium, osmoles, and water is expressed as the clearances of sodium (C_{Na}), osmoles (C_{osm}), and free water (urine volume – C_{osm}).[4] With normal water and solute intake, the C_{Na} and C_{osm} average 1% to 3% of the GFR; C_{H2O} is usually negative, reflecting the excretion of concentrated urine. A decrease in RBF or plasma volume causes C_{Na} and C_{osm} to fall due to sodium preservation.[3] Likewise, a breakdown in the countercurrent mechanism brought about by a selective insult to the juxtamedullary nephrons and their loops of Henle causes increased C_{Na} and C_{osm} from impaired tubular concentration of sodium.[5,9]

DEFINING ACUTE RENAL FAILURE

The struggle to classify renal failure has been long lasting, but in the 21st century, great strides have been made at codifying the criteria into a meaningful system. In the past two decades, the most successful attempts have been the RIFLE (Risk, Injury, Failure, Loss, and End Stage) criteria, the AKIN (Acute Kidney Injury Network) classification, and the KDIGO (Kidney Disease: Improving Global Outcomes) definition. Unsurprisingly, each of these has benefits and drawbacks.

The RIFLE classification was the product of the Acute Dialysis Quality Initiative, whose aim was to find easily applied, sensitive, and specific criteria based on serum creatinine, GFR, and urine output[10] (Table 61-1).

GFR is a difficult thing to accurately measure, but it is routinely estimated based on endogenous filtration markers, such as creatinine and inulin. Rising serum creatinine, another measure of renal function, has been shown to be independently associated with mortality, possibly through volume derangements or increased risk of infection or anemia.[11] As serum creatinine rises, GFR falls, or urine output decreases, the patient is classified as being at risk of kidney injury, having kidney injury, or having renal failure; to be acute, this has to occur within 7 days of kidney insult and be sustained for a least 24 hours. If the patient suffers complete loss of function for 4 weeks or 3 months, they are said to progress to loss or function or end-stage kidney disease, respectively. This is most useful in the setting of known, preinsult serum creatinine, but in the absence of that information, estimates of baseline values are allowed. While this method has been validated to correlate with outcomes (eg, mortality and length of stay), it has limitations, which are particularly germane to the trauma population. The need for a baseline serum

TABLE 61-1: RIFLE Classification

Class	SCr and GFR	Urine output
Risk	↑ SCr × 1.5 *or* ↓ GFR >25%	<0.5 mL/kg/h × 6 h
Injury	↑ SCr × 2 *or* ↓ GFR >50%	<0.5 mL/kg/h × 12 h
Failure	↑ SCr × 3 or ↓ GFR >75% *or* (if baseline SCr ≥353.6 µmol/L [≥4 mg/dL]) ↑ SCr >44.2 µmol/L (>0.5 mg/dL)	<0.3 mL/kg/h × 24 h or anuria × 12 h
Loss of kidney function	Complete loss of kidney function >4 weeks	Anuric
End-stage kidney disease	Complete loss of kidney function >3 months	Anuric

GFR, glomerular filtration rate; RIFLE, Risk, Injury, Failure, Loss, and End Stage; SCr, serum creatinine.

TABLE 61-2: AKIN Classification

Stage	SCr	Urine output
1	↑ SCr ≥26.5 µmol/L (≥0.3 mg/dL) or ↑ SCr ≥150%–200%	<0.5 mL/kg/h (>6 h)
2	↑ SCr ≥150%–200%	<0.5 mL/kg/h (>12 h)
3	↑ SCr ≥300% *or* (if baseline SCr ≥353.6 µmol/L [≥4 mg/dL]) ↑ SCr ≥44.2 µmol/L (≥0.5 mg/dL)	<0.3 mL/kg/h (>24 h) *or* anuria (12 h)

Note. All patients on RRT, despite SCr or Urine output, belong in stage 3.
AKIN, Acute Kidney Injury Network; SCr, serum creatinine.

TABLE 61-3: KDIGO Classification

Stage	SCr	Urine output
1	1.5–1.9 times baseline *or* ≥0.3 mg/dL (≥26.5 mmol/L) increase	<0.5 mL/kg/h for 6–12 h
2	2.0–2.9 times baseline	<0.5 mL/kg/h for ≥12 h
3	3.0 times baseline *or* Increase in SCr to ≥4.0 mg/dL (≥353.6 mmol/L) *or* Initiation of renal replacement therapy *or* In patients >18 years, decrease in eGFR to <35 mL/min/1.73 m^2	<0.3 mL/kg/h for ≥24 h *or* Anuria for ≥12 h

eGFR, estimated glomerular filtration rate; KDIGO, Kidney Disease: Improving Global Outcomes; SCr, serum creatinine.

creatinine and GFR makes this system very problematic in trauma. Methods used to estimate these values are validated in chronic kidney disease, but not in acute injury or trauma.[12] Despite these limitations, its ease of application and intuitive nature have kept it in use in many institutions.

The AKIN classification was created at a 2005 meeting of nephrologists, intensivists, and other interested parties and published in 2007 (Table 61-2).[13] In many ways, this method was just a clarification or modification of the RIFLE criteria, which stipulated that it could only be applied after adequate hydration and exclusion of urinary obstruction; it also removes GFR from consideration and does away with baseline creatinine in favor of two separate serum creatinine measurements within 48 hours of kidney injury. It continues to use urine output in the classification system.

This classification system stratifies renal failure into three stages, simply described as stage 1 through 3, based on rising serum creatinine and urine output. Stage 3 of this system also requires the initiation of renal replacement therapy. This system is more sensitive to small creatinine increases, which were found to correlate to outcome. Again, this system correlates with length of stay, but there is little evidence that there is improved stratification relative to RIFLE. Furthermore, if the creatinine elevation occurs over more than 48 hours, it is difficult to apply the AKIN classification. The inclusion of renal replacement therapy also complicates the utility of this system, as patient presentations and caregiver practices are variable.[12]

The final diagnostic classification system to consider is the KDIGO criteria, which take into consideration aspects of both of the previously discussed criteria including GFR (similar to RIFLE) and a change in creatinine over 48 hours with three levels of stratification (similar to AKIN)[11] (Table 61-3).

Per the recommendations, acute kidney injury (AKI) is defined by certain thresholds of increased creatinine or falling urine output within hours to days. The working group also recommends managing risk and injury based on stratification of risk and stage of AKI. In high-risk patients, this means mitigating exposure to nephrotoxins, ensuring volume status and perfusion pressure, avoiding hyperglycemia, monitoring creatinine and urine output, and potentially using functional hemodynamic monitoring. As patients progress into stages of injury, further escalation of care, including renal replacement and intensive care unit (ICU) admission, are considered. In comparison studies between all three classification systems, KDIGO has proven equivalent at diagnosis with AKIN and RIFLE.[14,15] There is also very little from an outcome standpoint to differentiate between these systems, especially KDIGO and AKIN, which have similar predictive qualities in length of hospital stay and mortality. This is not surprising because all of these systems are based on the same laboratory parameters.

CAUSES OF ACUTE RENAL FAILURE IN TRAUMA

Hemorrhage

Early mortality in trauma is strongly associated with hemorrhage. In the prehospital setting and in the first 24 hours following injury, hemorrhage is the primary cause of mortality following trauma.[16] Among patients who survive the initial insult, hemorrhage continues to be a contributor to long-term morbidity and mortality. With respect to kidney injury, the body's ability to preserve function through homeostatic regulation of RBF across a wide range of blood volumes represents a robust protective mechanism. These neuroendocrine and hydrostatic-driven mechanisms, however, primarily preserve GFR through alterations in vascular resistance to increase filtration fractions.[17,18] Other compensatory adaptations occur simultaneously to maintain cardiac output, but short of rapid restitution of renal plasma flow, these are measures that are unlikely to be effective in avoiding renal failure.[19] These mechanisms are always at work, even within the normal range of blood volumes that occur in the absence of trauma, but as hemorrhage progresses to hypotension, they

fall short and can lead to cell death, tubular necrosis, and acute renal failure.[18] Platelet and red cell aggregates in renal arterioles have been described in trauma patients who progress to renal failure.[17] The damage to the kidney in trauma also includes direct ischemic insult, inflammatory response, and resultant immune response.[18] In the case of patients with preexisting conditions that predispose them to renal injury, the risk of renal failure is even greater.[20] Still, in acute posttraumatic renal failure, hypovolemia is the most commonly described cause and, for the most part, represents a failure to achieve adequate resuscitation.

In the era of damage control resuscitation, it is increasingly common for trauma patients with significant blood loss to have limited crystalloid resuscitation. Although crystalloid resuscitation is still part of the protocol for some prehospital response teams, it has been shown to increase mortality in trauma and is falling out of favor.[21] In high-volume blood loss, most trauma centers will try to achieve a ratio of 1:1:1 of packed red blood cells to fresh frozen plasma to platelets.[21-23] It is widely agreed that these and other improvements in trauma care have likely reduced the incidence of renal failure after trauma.[20] These advances in trauma care were not strictly aimed at curbing the relatively low incidence of posttraumatic acute renal failure, but it has been serendipitous that the same measures taken to capture the patient dying of massive hemorrhage have simultaneously improved our understanding of renal injury in trauma and, in the right hands, decreased its incidence.

Even as our understanding of trauma and our methods continue to improve, there will always be patients who exist at the outer limits of our resuscitation efforts, so vigilant attention must be maintained. While some centers practice permissive hypotension in the setting of hemorrhage in trauma, for example, the degree of hypotension and length of time of that therapy are questionable and not necessarily easily controlled or the same in all patients.[21,24] Taken to extremes, permissive hypotension is likely to contribute to renal failure to some degree, but its utility in the setting of trauma may still make the risk of renal failure worth it. While hemorrhagic posttraumatic renal failure should be avoided whenever possible, it is not unexpected in a subset of patients who survive massive injury through heroic resuscitation.

Hypovolemia

Prerenal renal failure is caused by inadequate renal perfusion. As perfusion to the kidney decreases beyond compensatory mechanisms, GFR is reduced, leading to increased fluid and salt reabsorption.[25] There are several pathologic states that can lead to renal hypoperfusion, which can be broadly divided into third spacing, decreased effective volume, and decreased extracellular volume. Trauma patients are susceptible to all of these pathologic states but are especially likely to suffer from decreased circulating volume. This can occur due to hemorrhage early in their course, but even beyond that timeline, they remain at risk of hypovolemia. Vomiting, diarrhea, limited oral intake, diuresis, and, more recently, open abdomen management associated with damage control surgery can all contribute to hypovolemic renal failure.[26] While shock unquestionably represents a significant risk, even normotensive patients who remain in a state of diminished extracellular volume are at risk of renal failure due to prolonged exposure to compensatory mechanisms to maintain GFR, including efferent arteriole vasoconstriction.

TABLE 61-4: Urinary Indices for Diagnosing Acute Renal Failure

Measure	Prerenal	Intrinsic	Postrenal
Urine specific gravity	>1.020	Roughly 1.010	Roughly 1.010–1.000
Urine osmolality (mOsm/L)	>400–500	280–320	260–340
Urine sodium	<20	>40	<30
FE_{Na} (%)	<1%	>1%	1%–4%
BUN/Cr	20:1	10:1	10:1
Urine Cr/plasma Cr	>40	<20	<20
Urine urea/plasma urea	>8	<3	N/A
FE_{Urea}	<35%	>50%	N/A

BUN, blood urea nitrogen; Cr, creatinine; FE_{Na}, fractional excretion of sodium; FE_{Urea}, fractional excretion of urea; N/A, not applicable.

Diagnostically, prerenal azotemia is characterized by a physical exam consistent with renal hypoperfusion and confirmed by several urinary and blood test findings (Table 61-4). Specifically, an elevated urine osmolality (>500 mOsm/L); urine specific gravity (>1.020); ratios of urine/plasma osmolarity (>1.5), urine/plasma urea nitrogen (>8), and urine/plasma creatinine (>40); and a blood urea nitrogen (BUN)–to–creatinine ratio of 20 or greater are suggestive of a prerenal cause in an oliguric patient.

The fractional excretion of sodium (FE_{Na}), however, remains the most sensitive diagnostic test.[27,28]

$$FE_{Na} = (U_{Na} \times P_{Cr}/P_{Na} \times U_{Cr}) \times 100$$

The FE_{Na} test quantifies the amount of sodium excreted in the urine relative to the amount filtered by the kidney, which, in a purely prerenal state, should be low since the kidney is normal and can avidly reabsorb the majority of filtered sodium. When patients are treated with diuretics, which can alter the active transport of sodium, fractional excretion of urea (FE_{Urea}) is the preferred diagnostic method. In theory, because urea is primarily reabsorbed in the proximal nephron, diuresis should have less of an effect on the sensitivity of this test than does FE_{Na}; however, its clinical utility is controversial at best.[28,29]

$$FE_{Urea} = (U_{Urea} \times P_{Cr}/P_{Urea} \times U_{Cr}) \times 100$$

Burns

The association between severe burn injury and acute renal failure has long been observed and studied. As we understand

it, burns (especially large ones) lead to a profound inflammatory response, which leads to mediator release, capillary leak, changes in microvascular hydrostatic pressure, and protein loss, all of which contribute to potential renal failure. This combines with the significant open surface area associated with burns, which leads to sizable evaporative fluid loss. The incredible advances in burn care throughout the 20th century, such as categorization by total body surface area and the prioritization of early excision and grafting, have all positively influenced the outcome of renal failure in burn patients.[30] The introduction of the Parkland formula, a strategy that integrates many of these concepts into an early resuscitation guide, has specifically been recognized as a driving force in the drop in the incidence of renal failure after significant burns. The burn community has come to realize that the Parkland formula may in fact lead to excessive crystalloid resuscitation, which comes with several negative side effects, including acute respiratory distress syndrome, heart failure, and multiorgan system failure.[30-32]

Burns can cause renal failure through other mechanisms beyond hypovolemia. Burn eschar can act as a volume-limiting compartment and, much like fascial compartment syndrome, can cause rhabdomyolysis, myoglobinuria, and intrinsic renal injury. Electrical injuries also present a risk of rhabdomyolysis. Burns associated with traumatic injuries, sepsis, and significant comorbidities may also predispose patients to renal failure. These etiologies only explain the incidence of early renal failure in burns, but a second spike in renal failure has also been described. This renal failure is often initially polyuric, less easily tied to a prerenal state or myoglobinuria, and occurs around 5 days after the time of injury.[33,34] As early resuscitation became more standardized, this late form of renal failure became more prominent, but the etiology remains somewhat unclear and may be multifactorial. For example, as patients live longer, they are more likely to develop sepsis or be exposed to nephrotoxic agents. Evidence of mixed tubular and glomerular damage, as well as glomerular microthrombosis, indicates that late renal failure may in fact be intrinsic.

Although burn-related renal failure is less common than it previously was, it does still come with a concerningly high mortality. The mortality of acute renal failure in burns has been reported to be anywhere between 50% and 100%, but is most frequently cited at around 85%, and is attributable to multiple organ failure in most cases.[33-35] Even with renal replacement therapies, this mortality rate makes avoidance of renal failure an important hallmark of burn care.

Functional Prerenal Renal Failure

Prerenal causes of acute renal failure are not limited to extracellular volume status. Decreased effective volume, even in the setting of adequate intravascular volume, may lead to renal failure. This is easily conceptualized in the medical patient, but the perception that trauma patients are generally young and previously healthy, whether true or not, tends to place this farther down the differential diagnosis, but acute coronary syndromes and myocardial infarctions do occur in the trauma population. As the population ages, more people continue to present as trauma patients with preexisting cardiac pathologies, including heart disease, arrhythmias, valvular disease, and polypharmacy with cardiac consequences.[36] In the setting of trauma, these conditions prime the patient for decreased renal perfusion significant enough to cause renal injury. Even among patients who fit the prototypical trauma demographic of young and previously healthy, thoracic injury, and especially significant blunt cardiac injury, can lead to a cardiogenic limitation of effective circulating volume.[37] This can potentially be severe enough to cause acute renal failure. Cardiac injury through dissection of coronary occlusion can also happen as a result of trauma but is rare.

As with the elective surgical population, operative intervention also exerts a cardiac stress on trauma patients but without the opportunity to identify and optimize risk. On top of the risks from trauma, the negative inotropy and vasodilation associated with induction and continuation of general anesthesia can precipitate a cardiac event.[26] This is likelier in older patients, but in emergent trauma operations, it is not uncommon that the patient is still in the midst of active resuscitation and is relatively volume-down at the time of operation, so it is not out of the question that an acute coronary syndrome sufficient to lead to renal failure is possible.[38]

It is important to remember that prerenal azotemia, while potentially reversible, will progress to intrinsic disease if not managed appropriately.[26] Prolonged limitation of RBF will eventually cause an ischemic injury to the renal tubule. Once this occurs, restoration of the renal perfusion will no longer be sufficient to reverse the renal failure. This is especially true if ischemia progresses to cortical necrosis or microvascular coagulation. This is true no matter what the prerenal mechanism initially was; once prerenal patients progress to acute tubular necrosis, they are effectively on a different trajectory of injury and recovery.

Toxins

Intrinsic renal failure occurs when there is injury to the renal tubule, interstitium, vessels, or glomerulus. This is most commonly seen as acute tubular necrosis secondary to ischemia, which is generally from prolonged prerenal azotemia. The second most common cause, however, is toxic tubular necrosis.[26] In the trauma patient who survives the initial resuscitation, there are several common medications that can bring this on. Antibiotics are commonly cited causative agents of toxic acute renal failure in hospitalized patients. Aminoglycosides, which are still used for gram-negative coverage, can produce tubular necrosis in the proximal tubule. They also have a prolonged tissue half-life and can continue to cause injury beyond their immediate usage. Dose, duration, and concomitant use of other nephrotoxic agents can perpetuate this injury, so vigilance is necessary to avoid renal injury when using aminoglycosides.[39] Vancomycin, which is used for resistant gram-positive pathogens, which are more common in surgical and bloodstream infections, is also a known

nephrotoxin. As with aminoglycosides, the mechanism is not completely elucidated but is believed to include oxidative stress. Again, vigilant monitoring of renal function and potential change to an alternative agent are necessary when using these medications.

Nonsteroidal anti-inflammatory drugs (NSAIDs) remain a vital part of pain management. As the true risk of opioid exposure is better understood, the use of NSAIDs is likely to only grow.[39] Renal failure with NSAIDs follows two mechanisms. In one case, inhibition of cyclooxygenase leads to inhibition of renal prostaglandin synthesis. Under normal conditions, this is unlikely to cause renal failure, but in patients dependent upon prostaglandin for renal perfusion, including patients with any prerenal insult, such as acute hemorrhagic anemia, the resultant afferent arteriole vasodilation can cause a drop in the GFR and result in renal failure.[40] NSAIDs can also cause an acute interstitial nephritis, which can progress to nephrotic syndrome.

Angiotensin-converting enzyme inhibitors and angiotensin II receptor antagonists are generally well tolerated, but they also cause efferent arteriole vasodilation. When added to their desired effect of lowering systemic blood pressure, this may lead to a hemodynamic cause of acute renal failure.[39] Although this is a well-known complication, it is not common that trauma patients get put on angiotensin-converting enzyme inhibitors and angiotensin receptor blockers in the hospital setting.

Methicillin is classically implicated in a direct insult to the kidney leading to acute interstitial nephritis, but this can also occur with penicillin, cephalosporin, sulfonamides, and NSAIDs. Toxic acute interstitial nephritis presents with elevated creatinine, fever, possible rash, and urine sediment of red blood cells, white blood cells and their casts, and eosinophils. There may be mildly increased protein excretion.[39] If renal function does not improve with supportive therapy and medication discontinuation, renal biopsy to identify tubulointerstitial damage would be called for, as well as possible steroid administration.

Other medications implicated in renal injury are acyclovir, sulfonamides, antifungals, some chemotherapeutic agents, and some volatile fluorinated anesthetics, especially halothane.[39] The mechanisms involved are variable and not completely understood, but in general, they work through some combination of renovascular changes and direct insult. Colloids also present an underappreciated risk to the kidney. Just the plasma oncotic pressure increase from colloids can potentiate acute renal failure, especially in the hypovolemic patient. Although all colloids can cause this, the least likely offender is albumin. Colloids may also cause tubular obstruction or direct tubular toxicity.

Environmental agents, such as venom, or endotoxins, such as heme pigment, myeloma chain proteins, or other products, can also be nephrotoxic. Nephrotoxic renal failure tends to cause a nonoliguric nephritis and, in general, is associated with a lower mortality than oliguric renal failure.[40] Broadly speaking, most cases of toxic kidney injury can be reversed with withdrawal of the offending agent and limitation of other renal injuries, as well as treatment of underlying disease. If allowed to progress, however, an irreversible renal cortical necrosis may occur.[26]

The classic presentation of toxic nephropathy can be muddied by the simultaneous presence of ischemic insults to the kidney, such as patients with sepsis or HIV. This occurs because patients with acute illnesses or complicated comorbidities are more likely to have exposure to several, potentially nephrotoxic medications.[26] Patients with any underlying renal insufficiency or diabetes are very susceptible to renal injury from toxin exposure, as are the elderly. Hydroxyethyl starch, for example, is relatively unlikely to cause renal injury in a patient with preserved renal function, but in patients with already compromised renal function from sepsis or cardiac surgery, there was found to be a significant worsening of renal function after it was infused.[39]

Many of these discussed toxins are necessary or common medications that can and should be given when they are needed, but they do warrant close monitoring. In some cases, that means monitoring levels, such as gentamicin or vancomycin, but in other cases, it may simply mean watching renal function and having a high index of suspicion for renal injury secondary to the prescribed medication. Appropriate supportive treatment, such as judicious fluid administration, early withdrawal of injurious agents, and electrolyte management, are all necessary measures to avoid or mitigate toxic nephropathy.

Contrast Nephropathy

Every clinician will eventually have to face the risk or the fallout of radiocontrast nephropathy at some point, but it is a particularly ever-present issue for the trauma surgeon. The nature of the presentation of the trauma patient is such that you do not routinely get the details of their past medical history, previous exposures to contrast, preinjury laboratory findings, or occasionally, even their name. Within those limitations, you have to decide on a moment's notice whether they are or are not appropriate for computed tomography (CT) and whether they will or will not get contrast. In most American College of Surgeons designated Level I trauma centers, patients who come in as high-level activations and are deemed appropriate for CT will get at least intravenous (IV) contrast at presentation, despite their limited presentation, because the CT can be instrumental to clinical decision making.

The historical literature on contrast-induced AKI (CI-AKI) is primarily concentrated on iodinated contrast media and focused on cardiac angiography. Iodinated contrasts are water-soluble benzene rings linked to iodine atoms and, in their earliest formulations, were hyperosmolar.[41] The suggested mechanism of contrast-induced nephropathy is medullary hypoxia due to contrast-induced vasoconstriction and direct renal tubular toxicity through oxidative stress, cell failure, impaired calcium management, and increased apoptosis.[41,42] As with other nephrotoxins, patients with normal preexposure renal function are less likely to progress to renal failure. Other risk factors are diabetes, old age, hypertension, heart failure, volume depletion, hemodynamic instability, and concurrent use of other nephrotoxins.[11]

The clinical relevance of this proposed pathophysiology is now being questioned. As clinical use of hyperosmolar contrast has fallen almost completely out of practice, it has been replaced primarily with either low-osmolar or isosmolar contrast media, both for CTs and for traditional angiography.[11] In the modern era, both of these contrast media have been shown by meta-analysis to have much lower risk of CI-AKI than was previously thought.[43,44] It has even been suggested that the very idea of contrast-induced kidney injury is more a matter of our failure to understand the risk factors for kidney injury in hospitalized patients than a legitimate pathophysiologic state. Aycock et al,[45] in a meta-analysis, demonstrated no difference in renal dysfunction between patients who underwent CT either with or without contrast. While even this study contends that high-osmolar agents are associated with kidney injury, they are all but completely out of use, and the new agents seem to be very safe. The authors pointedly recommend moving away from even using the phrase "contrast-induced nephropathy."

The AKI guidelines from the KDIGO group support this finding and further point out that significant variation is found in the serum creatinine of hospitalized patients, irrespective of either their exposure to contrast or their diagnosis of kidney injury.[11] This variation is great enough that up to 35% of patients who did not receive contrast would qualify for the diagnosis of CI-AKI at the same serum creatinine if they had received contrast. Similarly, the focus on contrast exposure secondary to cardiac angiography may have been misleading. Not only are the indications and protocols different between cardiac angiography and CT contrast exposure, but also it is difficult to dissociate the risk of the contrast from the risk of cardiac dysfunction, hypotension, arrhythmia, renal artery embolization, hemorrhage, or atheroma dislodgement that may be encountered in cardiac angiography.[46]

Gadolinium-based contrast agents used in magnetic resonance imaging are clinically believed to be safer than iodinated contrast from the standpoint of nephrotoxicity, although there is some suggestion that they may be nephrotoxic in high-risk patients. However, in patients with chronic kidney disease or end-stage renal disease, they are known to cause a nephrogenic systemic fibrosis, which can be lethal.[42]

In the end, our feeling is that the data have significantly overstated the risk of kidney injury secondary to contrast administration. This is based on data that were found with contrast media of only historical significance or with the conflation of cardiac angiography and CT-related exposure. Our practice relies heavily on diagnostic CT early after presentation in most high-level trauma contacts, well before kidney function labs return, but we remain confident that the greater risk to these patients is in having undiagnosed pathology or nontherapeutic operations and interventions as a result of avoiding what is likely a safe CT.

Sepsis

The incidence of sepsis in trauma patients has been cited at anywhere from 2% to 14.4%, but what is certain is that septic patients have worse outcomes than patients without sepsis, with mortality of up to 23%.[47-49] This is despite the fact that we have become better in treating both trauma and sepsis over time. Injury Severity Score, Revised Trauma Score, and admission Glasgow Coma Scale score were all independent predictors of sepsis in trauma patients, and once a patient had sepsis, not only their mortality, but also their ICU and hospital length of stay, significantly increased. Progression from sepsis to organ dysfunction occurs through several factors, including impaired tissue oxygenation through hypotension, red blood cell dysfunction, and microvascular thrombosis.[50] Other factors, including proinflammatory and anti-inflammatory processes, mitochondrial damage, and capillary leak, also contribute to worsening organ dysfunction. These are the same processes at work in trauma patients with septic renal failure.

Renal failure has been demonstrated to occur with greater incidence in patients with worse sepsis, with 19% incidence in moderate sepsis and 51% in patients with septic shock and positive blood cultures.[6] The arterial vasodilation that occurs with sepsis activates neurohormonal and sympathetic pathways, leading to activation of the renin-angiotensin-aldosterone system (RAAS) in an effort to maintain perfusion. In brief, RAAS is stimulated by low RBF at the renal juxtaglomerular cells (through shock or any other mechanism) triggering conversion of prorenin to renin. That leads to hepatic angiotensinogen being converted to angiotensin I, which is itself converted by angiotensin-converting enzyme into angiotensin II. This triggers the secretion of aldosterone from the adrenal cortex, which leads to reabsorption of sodium and water in the renal tubules and excretion of potassium, which should lead to volume expansion. That and the vasoconstrictive effects of angiotensin II should support blood pressure. However, the renal vasoconstriction and retention of sodium and water may lead to worsening renal failure. Use of vasoactive agents to support blood pressure may also contribute by worsening RBF and limiting GFR. In patients who undergo excessive volume administration, increased interstitial volume and pulmonary edema may precipitate acute respiratory distress syndrome and still lead to multiple organ dysfunction.

A phenomenon of "inappropriate polyuria" has previously been described as occurring in septic patients and, in some cases, preferentially occurring in patients with sepsis following trauma.[17] It has been proposed that these patients exhibit decreased RBF, leading to reduced filtration fraction and oncotic pressure. In response, there is less salt and water reabsorbed in the distal tubules and a resultant diuresis and enuresis.[9] In these patients, polyuria of up to 400 mL/h has been reported. These findings were primarily described in the 1970s, however, and it may be the case that these patients were likely suffering from nonoliguric renal failure.[40] These patients are not unique to sepsis and, in fact, are more commonly seen in nephrotoxic exposure, but also have a high-output renal failure and can progress to oliguric renal failure. The cause of this trajectory of renal failure is unclear, but it has been described throughout most of the 20th century.

The combination of sepsis and renal failure has a very high associated mortality of 70%.[6] For this reason, predictors and markers for renal failure in sepsis have been sought, but no clear evidence has been found for a method better than a high clinical suspicion and appropriate supportive care. Controlling blood glucose and associated immune dysfunction and avoiding disseminated intravascular coagulation and microthromboses also help prevent progression to, or mitigate the effects of, renal failure in sepsis.[6]

Rhabdomyolysis

The definition of rhabdomyolysis is leakage of contents of the muscle cells such as electrolytes, myoglobin, and sarcoplasmic proteins into the circulation. There are several categories of this disease entity. However, this chapter will focus on trauma-related rhabdomyolysis from crush syndrome, limb compression, and/or major artery occlusion.[51]

Myoglobinuria is unique to rhabdomyolysis. In this circumstance, the urine appears tea colored when serum myoglobin levels reach 100 mg/dL. In more severe cases with prolonged muscle ischemia or necrosis prior to treatment, the urine appears black.[52] AKI associated with myoglobinuria is the most serious complication of severe rhabdomyolysis and may be life-threatening. Approximately 7% to 10% of all cases of AKI in the United States are related to rhabdomyolysis.[53]

The mechanism by which rhabdomyolysis causes impaired renal function is thought to be simultaneous vasoconstriction, tubular ischemia, and obstruction primarily at the level of the distal tubule. Hypovolemia and vasoconstriction augment myoglobin accumulation along the renal tubules. The nephrotoxic effects of myoglobin appear to only occur in an acidotic environment.[51,54,55] Major crush injury or prolonged ischemia of a limb causes massive necrosis of the muscle. The patient usually presents with limb swelling, weakness, and severe myalgia. As mentioned, the urine appears dark brown to black in the setting of markedly elevate serum creatine kinase (CK) levels (Fig. 61-2).

Although no specific CK level determines if the patient will develop AKI, some data support the theory that as the CK rises, the incidence of AKI increases. The risk of AKI is very low if the admission CK levels are below 15,000 to 20,000 units/L. Heme-positive urine without red cells confirms the production of myoglobin.[54,56,57] Oliguric or anuric renal failure may occur in severe cases. Rapidly rising plasma creatinine is a common finding. Major electrolyte abnormalities can occur when components of muscle breakdown are released into the circulation and can contribute to the severity of the AKI that may ensue (Table 61-5).

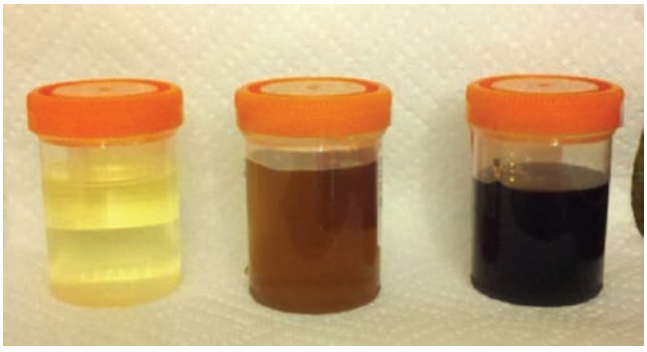

FIGURE 61-2 Urine progression from clear (normal) to tea colored (myoglobinuria) to black (rhabdomyolysis).

TABLE 61-5: Electrolyte Abnormalities Associated with Rhabdomyolysis

Hyperkalemia, can be life-threatening
Hyperphosphatemia
Hyperuricemia
Metabolic acidosis, high anion gap
Hypermagnesemia
Hypocalcemia
Hypercalcemia, after recovery from acute kidney injury

Hyperkalemia can occur rapidly and may reach life-threatening levels. One unique finding during the recovery from rhabdomyolysis-induced AKI is hypercalcemia, which occurs when calcium is mobilized after being deposited in the muscle during the ischemic event.[58]

The literature on this disease entity clearly shows that early, aggressive fluid resuscitation is the mainstay of treatment. Hypovolemia causing the renal vasoconstriction and ischemia decreases adenosine triphosphate production due to hypoxia in the renal tubules This causes cellular necrosis and accumulation of cellular debris in the tubular lumen with precipitation of myoglobin that obstructs the distal convoluted tubules. Volume loading and "flushing" of the casts is the proposed benefit of fluid resuscitation.[55,59]

The key to avoiding AKI is to start fluid therapy as soon as possible. Reports of starting fluids in the field with crush injuries demonstrate improved outcome. The earlier the fluid management, the better are the outcomes. Up to 10 L of fluid per day may be required depending on the severity of the rhabdomyolysis.[54]

There have not been any randomized studies determining which type of fluid replacement is favored. The benefits of alkalization of the urine are based on animal studies, and there is no evidence that it is any better than simple volume replacement.[51,54,60] Normal saline and half normal saline with sodium bicarbonate are the most frequently used salt solutions. There are several suggested steps in the management of myoglobinuria and rhabdomyolysis (Table 61-6).

Diuretics should not be used during the initial management of rhabdomyolysis. Mannitol is the preferred diuretic and should only be used after maximum fluid replacement has been achieved. Its benefit stems from the fact that myoglobin toxicity affects the kidney by lipid peroxidation and the production of oxygen free radicals. Tubular obstruction by myoglobin is also associated with AKI. Mannitol has the theoretical benefits in this setting of being a free radical scavenger and an osmotic diuretic improving urinary flow and

TABLE 61-6: Steps in Management of Myoglobinuria and Rhabdomyolysis

Measure serum creatine kinase, urine dipstick, myoglobin, electrolytes, BUN, and Cr.
Initiate volume repletion as soon as possible with normal saline at rate between 200 and 1000 mL/h.
Target urine output at 200–300 mL/h.
Check electrolyte levels, especially potassium and calcium, frequently.
Check urine pH and attempt to keep above 6.5. Consider adding sodium bicarbonate if urine remains acidotic or systemic acidosis.
Mannitol only if needed to maintain desired urine output despite adequate fluid administration.
Maintain volume resuscitation until myoglobin is cleared.
Use CRRT for symptomatic, resistant hyperkalemia, volume overload, severe acidosis, and oliguria/anuria despite fluids and mannitol.

BUN, blood urea nitrogen; CR, creatinine; CRRT, continuous renal replacement therapy.

flushing of tubular toxins.[61,62] Long-term survival in patients who develop rhabdomyolysis-induced AKI is favorable, with 80% recovering full renal function.[63]

Severe acidosis, volume overload, or intractable life-threating electrolyte abnormalities in the setting of AKI requires hemodialysis. Continuous renal replacement therapy (CRRT) can rapidly correct electrolyte abnormalities and helps clear myoglobin effectively from the renal tubules and the bloodstream. Despite the usefulness of CRRT in clearing myoglobin and correcting electrolyte levels, mortality in this setting has not improved with the use of hemodialysis.[55,64-67]

PREVENTION AND TREATMENT

Resuscitation as Treatment

The topic of fluid management as it relates to renal injury is one of balance. Hypotension and, in turn, renal hypoperfusion can lead to temporary renal impairment or permanent injury. Conversely, volume overload can also potentiate harm and poor outcomes. Thus a balance between adequate fluid resuscitation, with vasopressor support as needed, and attention to volume overload must exist.

The ideal resuscitation fluid has yet to be determined and is certainly related to the specific patient and the patient's underlying problem. In the hemorrhagic trauma patient, blood and its components must be administered, but in the complex ICU patient, fluids may vary. The Saline versus Albumin Fluid Evaluation (SAFE) study noted no difference in the use of 4% albumin versus normal saline with regard to 28-day mortality. The fluids were also similar in relation to organ failure rate, ICU and hospital days, mechanical ventilation days, and days of renal replacement therapy (RRT).[68] Similar findings were noted in the Colloids Versus Crystalloids for the Resuscitation of the Critically Ill (CRISTAL) trial, which again noted no difference in 28-day mortality between colloid and crystalloid administration in the hypovolemic shock patient. Of note, 90-day mortality was found to be lower in the colloid group, a finding that must be further evaluated.[69] Development of AKI in critical patients requiring fluid administration was also addressed in the 0.9% Saline versus Plasma-Lyte 148 for ICU Fluid Therapy (SPLIT) trial to determine if buffered crystalloid therapy would offer any advantage. No difference in the development of AKI was found between these two groups.[70]

Therefore, current KDIGO guidelines promote the use of isotonic crystalloid solutions over colloid products for the initial volume resuscitation in patients at risk for or with AKI, excluding those patients in known hemorrhagic shock.[11] This is based on the lack of evidence proving colloid administration to be superior. Colloids may, however, offer an advantage in certain patient populations or those at risk for volume overload.[71]

Fluid overload may also be problematic. The Program to Improve Care in Acute Renal Disease (PICARD) group determined that critically ill patients with volume overload, defined as a greater than 10% increase in body weight from baseline, were at much higher risk of 60-day mortality.[72] Additionally, they found that among patients requiring dialysis, those with a lower overall fluid accumulation at the start of dialysis had a lower rate of mortality. Similar findings were noted by the FINNAKI study group, which found twice the 90-day mortality in patients with volume overload (again defined as >10% increase from baseline body weight) at the time of RRT initiation.[73]

Glucose Control

Hyperglycemia is a marker of critical illness. Stress hormones, inflammatory mediators, and counterregulatory hormones all function to disrupt the signaling of insulin and promote insulin resistance.[74] Determination of hyperglycemia as cause of organ dysfunction and mortality in critically ill patients has remained in question.

Higher glucose levels were found to be associated with increased risk of death in critically ill trauma patients by Wahl et al.[75] Using glucose ranges of greater than 200, 141 to 200, and 140 mg/dL or lower, the mortality rates were 40%, 20%, and 3.3%, respectively. Van den Berghe et al[76] determined that tighter glucose control, defined as at or below 110 mg/dL, resulted in decreased morbidity and mortality in surgical ICU patients. However, their subsequent studies comparing medical and surgical ICU patients showed no difference in mortality with tight glucose control.[77] There was a reduced incidence of AKI and need for RRT in the intensive insulin therapy surgical ICU group,[76] but only a reduction in AKI without an associated reduction in use of RRT in the intensive insulin therapy medical ICU group.[77] The Efficacy of Volume Substitution and Insulin Therapy in Severe Sepsis (VISEP) study observed that critically ill septic patients treated with either tight glucose control (glucose range, 80–110 mg/dL) or conventional glucose control (glucose range, 180–200 mg/dL)

showed no difference in AKI, 28- or 90-day mortality, or Sequential Organ Failure Assessment (SOFA) scores.[78] Notably, severe hypoglycemia (glucose <40 mg/dL) was found in the intensive insulin therapy group. Similar results were found by Thomas et al,[79] showing no significant difference between the intensive versus conventional insulin therapy groups in 28- or 90-day mortality, AKI rates, or SOFA scores, with hypoglycemia again found more frequently in the intensive insulin therapy group. A meta-analysis of randomized controlled trial data, including the NICE-SUGAR (Normoglycemia in Intensive Care Evaluation—Survival Using Glucose Algorithm Regulation) study,[80] again showed that intensive insulin therapy significantly increased the risk of hypoglycemia without reduction in mortality benefit in critically ill patients, although surgical ICU patients may need further evaluation.[81]

Based on the variability of findings and the risk of potential patient harm, KDIGO guidelines recommend insulin use in critically ill patients, with a glucose target of 110 to less than 150 mg/dL in critically ill patients.[11] Further evaluation of target ranges and specific patient populations may be warranted in the future.

Nutrition

Renal failure can include alterations in metabolism, a state of inflammation, and nutrient imbalance. Malnutrition is predictive of mortality among patients with AKI.[82] AKI is frequently found in the setting of other comorbidities, and all organ dysfunction must be taken into account.

Overall energy use is not affected by renal failure alone, and caloric repletion should not be increased based solely on this finding. Additional comorbidities, multiple systems of organ dysfunction, and/or sepsis may increase total energy expenditure to a maximal addition of 25% to 30%.[83] Current American Society for Parenteral and Enteral Nutrition (ASPEN) and KDIGO guidelines recommend standard total energy intake of 25 to 30 and 20 to 30 kcal/kg/d, respectively.[11,84] Overfeeding does not necessarily result in positive nitrogen balance and may result in complications associated with hypertriglyceridemia, hyperglycemia, and volume overload.[85]

Carbohydrate metabolism in acute renal failure is often associated with the finding of hyperglycemia secondary to an increase in muscle protein degradation,[86] alterations in hepatic glucose uptake, and insulin resistance.[87] Recommendation of 3 to 5 g carbohydrates/kg body weight with close glucose monitoring is advisable.[11]

Lipid metabolism in AKI is affected by lipolysis inhibition resulting in hypertriglyceridemia. There may also be slowed clearance of supplemented lipids.[88] Recommendation for fat administration is 0.8 to 1 g/kg body weight.[11]

Protein catabolism in acute renal failure is multifactorial and related to increases in muscular release and degradation of amino acids with negative nitrogen balance, resistance to growth factors, inflammation, and increased hepatic uptake of amino acids, insulin resistance, acidosis, and other factors. Overall catabolism is also affected by underlying disease processes, type and degree of renal dysfunction, and, if applicable, the type of RRT used.[88]

Simply increasing protein supplementation may not achieve positive nitrogen balance and may in fact produce azotemia and further acidosis without improvement in overall outcome.[89] With the use of CRRT, fluid balance and azotemia and higher protein intake can be better tolerated.[90,91] KDIGO guidelines recommend 0.8 to 1.0 g/kg/d of protein in noncatabolic AKI patients without dialysis, 1.0 to 1.5 g/kg/d in patients with AKI on RRT, and up to a maximum of 1.7 g/kg/d in patients on CRRT and hypercatabolic patients.[71] ASPEN guidelines advise a general goal of 1.2 to 2 g/kg/d and up to a maximum of 2.5 g/kg/d for those on CRRT or frequent hemodialysis, with weight determined by usual weight for average-weight patients and ideal body weight for obese and critically ill patients.[84]

Trace elements, such as selenium and copper, and certain water-soluble vitamins may need to be replaced, especially with RRT use. Vitamin C supplementation in patients with AKI should be 50 to 100 mg/d or higher with CRRT. Other micronutrients may need to be monitored and supplemented as required.

Adequate nutritional support offers a reduction in mortality and morbidity for critically ill patients. The enteral route for nutrition is preferred whenever possible. Enteral nutrition promotes gut motility, protection, and immune function.[92,93] Early enteral feeding should be used and supplemented with parenteral nutrition if needed to offer complete nutritional support.[94] ASPEN guidelines suggest start of nutritional support with enteral nutrition within 24 to 48 hours of admission to ICU.[84]

Renal Replacement

AKI remains a complicating factor for many trauma patients, with shock and sepsis being major risk factors for RRT.[95] Indications for RRT in the acute setting may differ from those in chronic kidney disease. Metabolic disturbances such as hyperkalemia not responsive to therapeutic interventions, acidosis, uremia, and severe volume overload may require RRT consideration. Certain toxins and drug overload may also require intervention. Ideally, RRT can serve to promote fluid and electrolyte balance, offset dysfunction in other organ systems, and prevent further renal injury.

Volume overload, in particular, may result in poor outcome in the setting of AKI. The Program to Improve Care in Acute Renal Disease (PICARD) group noted that dialyzed survivors had significantly less fluid overload when dialysis was started compared to nonsurvivors. Patients not undergoing dialysis also had less volume overload at the time of peak creatinine, whereas those with fluid overload at the time of peak creatinine were less likely to recover kidney function. Fluid overload at the time of AKI diagnosis resulted in less recovery of kidney function.[72]

Although there is certainly benefit to RRT, complications may arise resulting in prolonged renal dysfunction or

poor recovery of renal function. Associated hypotension with impaired RBF may result in ischemic renal injury.[96] Fluid and electrolyte imbalance and access-related complications may also be problematic.[97] Therefore, timing of RRT must be carefully considered.

The PICARD group conducted a multicenter study of critically ill patients requiring dialysis for severe AKI. Therapy timing was determined by BUN concentration at the start of RRT, with the early group defined by a BUN of 76 mg/dL or lower and the late group defined by a BUN of greater than 76 mg/dL. Mortality within 60 days was found to be lower in the early RRT group, with a relative risk for death in the late therapy group of 1.85 (95% confidence interval, 1.16–2.96).[98]

Another study evaluated the timing of RRT in surgical ICU patients after major abdominal surgery with utilization of the RIFLE classification system.[10] Simplified RIFLE classification was used to differentiate the early dialysis group (RRT with sRIFLE-R [risk] or sRIFLE-0) and the late dialysis group (RRT with sRIFLE-I [injury] or sRIFLE-F [failure]). Late initiation of RRT was determined to be an independent predictor for in-hospital mortality.[99]

Regarding type of RRT for use in AKI, each has advantages and disadvantages. CRRT allows for slower fluid removal with less abrupt solute shifts. Hemodynamic stability can be maintained throughout, and titration can be performed based on the patient's overall status. Intermittent RRT (IRRT) can be performed more quickly with rapid effect and offers opportunity for patient mobilization for other clinical interventions.

In hemodynamically stable patients, IRRT versus CRRT does not appear to make a significant difference.[100-102] A Cochrane Collaboration review evaluating 15 randomized controlled trials determined no survival advantage of CRRT over IRRT, as well as a similar degree of renal function recovery. Of note, the CRRT group had a higher mean arterial pressure at treatment end and less elevation in vasopressor support.[103]

In patients with tenuous hemodynamic status or those with acute brain injury, CRRT may be beneficial. CT imaging performed before and after intermittent hemodialysis (IHD) and continuous veno-venous hemofiltration (CVVH) showed that significant changes in the density of white and gray matter were observed after IHD, indicating brain edema.[104] Cerebral perfusion pressure may be altered by changes in mean arterial pressure or alteration in intracranial pressure from rapid solute shifts. These issues can be avoided or handled more consistently with the use of CRRT.[105]

Determination of the timing of the ending of renal support can be difficult and depends on the type of RRT used. In IHD, clearance of solutes is variable between treatment sessions, whereas CRRT allows for more reliable determination of kidney function. Values including urine output, creatinine excretion, and change in BUN and serum creatinine must be evaluated. Wu et al[106] evaluated dialysis termination in postoperative patients and determined that the risk of redialysis was reduced with increased urine output, shorter duration of dialysis, age less than 65 years, and lower disease severity score. The BEST Kidney (Beginning and Ending Supportive Therapy for the Kidney) study evaluated multiple centers and also found that urine output at the time of CRRT cessation was the most important predictor of successful discontinuation. Of note, the use of diuretic was found to negatively affect the predictive ability of urine output, consistent with lack of effect on overall GFR. In addition, patients weaned successfully without subsequent need for further dialysis treatment had better outcomes.[107] Overall, RRT is associated with significant impact on morbidity and mortality, and goals of care often come into play with regard to continuation of care.[108]

MAJOR RENOVASCULAR INJURIES AND NEPHRECTOMY AFTER TRAUMA

Major renovascular injuries are uncommon, and thus there is significant controversy related to the appropriate treatment and the subsequent outcomes related to observation, repair, or immediate nephrectomy. Attempts at renal arterial revascularization for trauma are generally poor, and the potential for subsequent renal impairment, including renovascular hypertension and renal failure, is high[109] (Fig. 61-3).

A Western Trauma Association multicenter report on major renovascular injuries suggests that the majority of patients have associated abdominal injuries, and over half arrived in the emergency department in hemorrhagic shock. An algorithm suggests that all patients with grade V renal injuries and hemodynamically unstable patients with grade IV renal injuries should undergo nephrectomy.

The best results for renal salvage for grade IV injuries are with renal vein and parenchymal repair only.[110] Case reports of endovascular repair and stenting of renal arterial injuries are rare and seldom result in successful outcome.[111,112]

The only studies related to long-term outcomes after nephrectomy are for patients undergoing live kidney donation. A recent analysis showed that life expectancy may be reduced by 0.5 to 1 year in this patient population.[113] There is no literature, however, describing the long-term risks related to nephrectomy as a result of trauma, including chronic kidney disease, end-stage renal disease, or life expectancy. Long-term follow-up studies are needed to determine this potential risk.

OUTCOMES AFTER RENAL FAILURE

While the incidence of renal failure in trauma has declined (now in the single-digit range of 3%–9%), when it does occur, the outcomes remain unsatisfactory.[20,114] In critically ill patients, acute renal failure has been linked to a greater than 20% mortality, which is even higher when considering all in-hospital mortality.[115] In fact, stable survival is not achieved until after 30 to 60 days.[10] In one study, risk of mortality was about three times greater in patients with acute renal failure than those without renal failure, whether they were in the ICU or the ward.[116] That study also linked AKI to earlier onset of multiorgan system failure (24 vs 48 hours after admission).

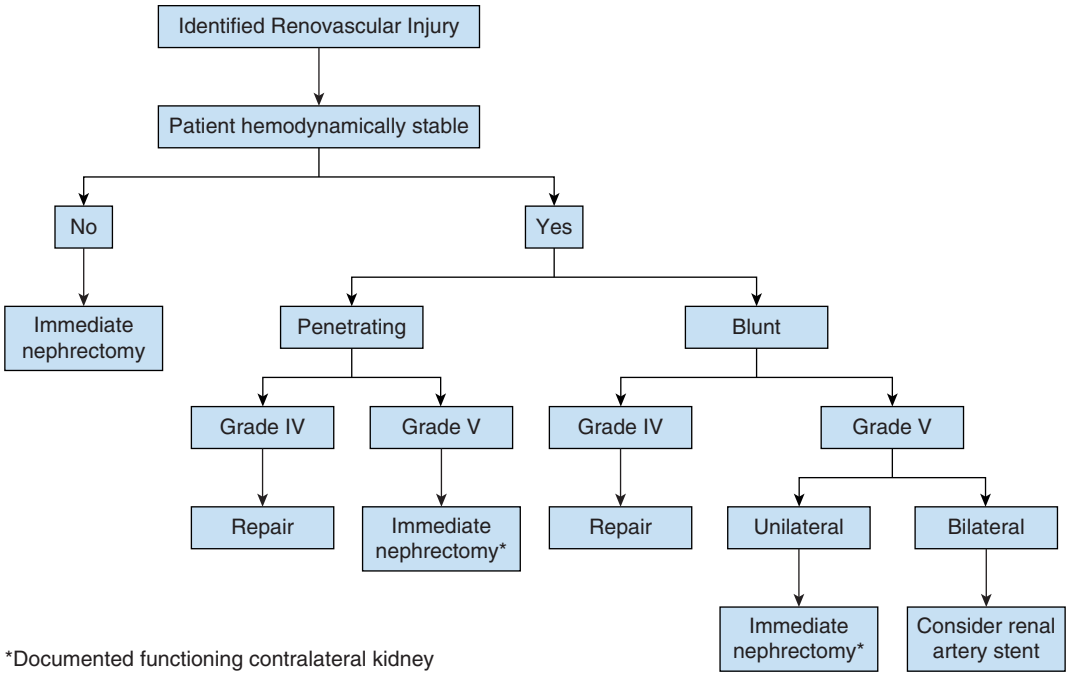

FIGURE 61-3 Algorithm for renovascular injury. (Reprinted with permission from Knudson MM, Harrison PB, et al. Outcome after major renovascular injuries: a Western Trauma Association multicenter report. *J Trauma.* 2000;49(6):1116-1122.)

Among survivors, it is important to understand what the renal-specific outcomes may be; in other words, who is discharged free of renal replacement. The rate of patients requiring dialysis after discharge has been previously stated to be in the mid-30% range, but this may not be the case.[117,118] A more recent study demonstrated a rate of 18.6%, which they attributed to continuous renal replacement rather than intermittent replacement.

Patients who require RRT have been shown to have even higher mortality and higher level of care, based on diagnostic and therapeutic needs associated. The associated mortality is also fourfold that of patients who did not progress to RRT, even when matched to patients with similar levels of care.[119] This supports the idea that renal failure is an independent risk factor for these poor outcomes. Proposed causes in the RRT group are physiologic dysfunction associated with renal failure, versus the negative direct effects of renal replacement itself. Predictably, secondary diagnoses requiring critical care, such as cardiac irregularity or mechanical ventilation, were associated with even worse outcomes. The risk associated with continuous versus intermittent renal replacement is unclear.[119]

These findings, however, were primarily studied in nontrauma patients, rather than in the trauma population. In this group, the causes are multifactorial and the trauma itself is an additional risk, which makes it difficult to assign incidence specific to trauma.[115] Unfortunately, there is no work that specifically predicts the outcome of renal failure among trauma patients and helps predict outcome of those who survive their hospitalization. This represents a potential area of future research.

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62

Nutritional Support and Electrolyte Management

Robert D. Becher • Kimberly A. Davis

KEY POINTS

- Nutritional therapy should be integrated into the overall management of all trauma, surgery, and critically ill patients.
- Nutritional needs and requirements are different in healthy patients compared to the critically ill and severely injured, hypermetabolic trauma patients.
- Early enteral/oral feeding is a proactive therapeutic strategy that may reduce disease severity, decrease complications, and favorably impact outcomes.
- Enteral/oral feeding is the preferred mode of nutrition for trauma, surgical, and critically ill patients.
- Early parenteral nutrition should be considered if enteral feeds are contraindicated or are not expected to be started for 3 days.
- Avoid long periods without enteral or parenteral nutrition..
- Underfeeding during the stress-related catabolic phase or the immediate postoperative period is a risk factor for complications.

INTRODUCTION AND HISTORICAL PERSPECTIVE

Dietary choices impact disease. In the outpatient setting, dietary choices influence our risk for heart disease, cancers, obesity, diabetes, and stroke[1,2]; poor dietary choices increase the chance for developing these chronic and acute conditions, many of which are among the leading causes of morbidity and mortality in the United States and the world.[3,4] Five of the 10 leading causes of death in the United States are directly associated with dietary factors (heart disease, cancer, stroke, diabetes, and atherosclerosis).[5] In addition, dietary interventions can be a primary treatment for these conditions, with outcomes equivalent to or better than drug therapy.[6]

In the inpatient, hospital-based setting, a patient's nutritional status also impacts clinical outcomes. This notion was first reported in surgical patients in the year 1936, in a study demonstrating that patients with baseline malnutrition undergoing peptic ulcer surgery had mortality rates of 33% compared to 3.5% in well-nourished controls.[7] This pattern has been reported repeatedly since then, with preoperative malnutrition leading to increased morbidity and mortality in the surgical and trauma literature. With the prevalence of baseline malnutrition among hospitalized medical and surgical patients as high as 40%,[8,9] many patients are at high risk for poor outcomes and complications.

Anticipating and addressing hospitalized patients' dietary needs can therefore substantially impact their disease as well as their outcome. Simply put, providing adequate nutrition is essential to high-quality patient care, and given the extreme physiologic, metabolic, and homeostatic changes that can occur after severe injury, nowhere in medicine is this more true than in trauma, surgery, and critical care.

This chapter is about nutrition, the inflammatory stress responses to injury, and the nutritional needs of the polytrauma patient, for both macro- and micronutrients. The first major section on nutritional support provides a thorough, up-to-date guide on nutrition: first, nutritional needs and requirements are addressed in both the healthy patient and the critically ill, hypermetabolic trauma patient; second, we outline the specifics of nutritional intervention, including both enteral and parenteral nutritional support; and last, we cover some special patient populations. The second major section of this chapter on electrolyte management is divided into the management strategies of four major electrolytes and their associated abnormalities: sodium and disorders of water balance; potassium; phosphorus; and magnesium.

NUTRITIONAL SUPPORT: OVERVIEW

Normal Nutritional States of Health

Normal health implies homeostasis, when there is balance among the metabolic regulatory mechanisms that act to keep the body in a smooth, continuous condition of regular physiologic function. Part of homeostasis involves ensuring that the body has adequate energy reserves as well as other nutrients to maintain this physiologic normalcy.[10] We discuss nutrient utilization and requirements in a state of normal health and provide a conceptual framework underlying the basis and terminology of nutritional needs support. We cover the three principle classes of food—namely proteins, carbohydrates, and fats (collectively the macronutrients)—as well as the other fundamental compounds without which humans could not survive: water and the micronutrients (vitamins and minerals). See Box 62-1.

NUTRITIONAL SUPPORT: NUTRIENT UTILIZATION AND REQUIREMENTS IN A STATE OF NORMAL HEALTH

Nutrient Utilization and Needs

Humans need energy to live: to do physical work; to maintain body temperature and concentration gradients; and to transport, synthesize, degrade, and replace molecules that make up our body's tissues.[11] This energy is generated by the oxidation of various organic substances consumed in the diet, primarily the macronutrients: carbohydrates, fats, and proteins. The energy produced in the human body by the oxidation of macronutrients is the same as the heat of combustion of these substances in a lab. The major difference is that the body's oxidation is carried out though multiple steps, allowing humans to capture the energy in an intermediate chemical form.

A human's daily energy requirement allows for maintenance of metabolism and activity. This requirement is the level of energy intake from food that will balance energy expenditures. Although the word *energy* is often used repeatedly in a discussion of nutrition, its use in this sense is overly general and nonspecific. *Energy* can refer to food energy; basal energy expenditure; potential energy; energy density; or many other aspects of nutrition. As it relates to what we eat, food energy (as measured in kilocalories or kilojoules) specifically refers to the energy derived from protein, carbohydrates (ie, sugars), and fat.

In addition to energy requirements, humans also have nutrient requirements and therefore need protein (for essential amino acids), fats (for essential fatty acids), water, vitamins, and minerals to survive. Macronutrients and micronutrients, along with water, build the basis of nutrient utilization and needs. Each will be reviewed here.

ENERGY = CALORIES = KILOCALORIES

A patient's daily kilocaloric needs (ie, total energy requirement) are often referred to as their total energy expenditure (TEE); strictly speaking, this is not completely accurate, as the energy needs (ie, requirements) are *determined by* the

BOX 62-1: The Confusing Language of Food Energy

There are two widely accepted units of measuring and quantifying food energy:

- The International System of Units (SI; aka the "modern" metric system), which is the official system of measurement in almost every country in the world, measures energy in joules (J).[13,14] Within this system, food-related quantities of energy are typically expressed in kilojoules (kJ).
- The "old" metric system measures energy in calories; as a unit of energy, it officially is the upper-case Calorie, or equally kilocalorie (kcal): 1 kcal (aka 1 Calorie) is equal to 1000 calories. The scientific definition of 1 *kilocalorie* is the amount of energy needed to raise the temperature of 1 *kilogram* of water 1 degree Celsius from 14.5°C to 15.5°C at 1 atmosphere. A conversion from the old to the new (SI) metric system is: 1 kcal = 1 Cal = 4.184 kJ.

The terminology is confusing. When speaking of nutrition and food energy, it is common to use the term "calorie," with an implied meaning of kilocalories. For example, in the United States, the daily recommended value (DRV) for an average adult female is 2000 calories per day; this more accurately should be written 2000 kcal (2000 kcal = 2000 Calories). Therefore, it is technically incorrect to use calorie interchangeably with kilocalorie; the calories counted by clinicians and dieticians for nutritional purposes are really kilocalories.[15]

To make it even more confusing, the standard units used for food energy vary by region of the world, depending on a country's adoption of the SI system. In some parts of the world, including the United States, food labels use the older system, and list Calories or kcals (even though, as noted earlier, the US Food and Drug Administration's (FDA) Food Labeling Guide drops the "kilo-" and simply refers to food energy incorrectly as "calories"[16]). In most other parts of the world, food energy is expressed in the SI unit of kilojoules. In the European Union, food labels list both kJ (kilojoules) and kcal (kilocalories).[17]

In nutrition circles, including the Committee on Nomenclature of the International Union of Nutritional Sciences,[18] it has been proposed that the SI unit of the kilojoule replace the old metric unit of kilocalorie as the unit of choice for defining the energy value of food. Such a change would align the nomenclature of food sciences with that of most other sciences, which use SI units and the metric system. In this chapter, however, we will not adopt the modern metric system; rather, we will use the language common in the United States: the kilocalorie.

energy expenditure. A better way to think about this is to appreciate that energy requirements are based on estimates of energy expenditure.[10]

The basal metabolic rate (BMR) reflects the energy needed to sustain and maintain metabolism; it is the energy cost of living. The BMR "describes the rate of energy expenditure that occurs in the post-absorptive state, defined as the particular condition that prevails after an overnight fast, that subject having not consumed food for 12 to 14 hours and resting comfortably, supine, awake, and motionless in a thermoneutral environment. This standardized metabolic state corresponds to the situation in which food and physical activity have minimal influence on metabolism."[11] Basal energy expenditure (BEE), expressed as kcal/24 hours, is the BMR extrapolated to 24 hours.

TEE in healthy subjects is made up of three components: resting energy expenditure (REE) (and thus BEE), activity-induced energy expenditure (AEE), and diet-induced energy expenditure (DEE).[11]

1. REE, or energy expenditure under resting conditions, is about 60% of TEE. Note that REE is made up of three variables: BEE, thermoregulation, and energy expended in depositing new tissue. As such, REE is usually about 10% to 20% higher than BEE. Therefore, REE includes all the homeostatic reactions in the human body.
2. AEE is about 30% of TEE. AEE is highly variable, depending on baseline physical activity level as well as a person's physical capacity. For example, in obese patients, with a decreased level of activity, an AEE estimate of 30% TEE is accepted to be too high. However, in very active individuals, AEE can be over 50% of TEE.[11]
3. DEE is about 10% of TEE. This is also referred to as the thermic effect of food.

From these three components, we see that four factors determine an individual's daily energy requirements: gender, age, body size, and level of activity. Males require more kilocalories than females; the young require more kilocalories than the old; large people with more body mass require more kilocalories than smaller individuals; and the more active you are, the higher your kilocaloric needs.

For example, a nonactive, frail 85-year-old woman who weighs less than 100 lb and is 4 ft tall requires only 1000 kilocalories per day, whereas a college-age male athlete weighing 220 lb and standing over 6 ft tall needs over 3000 kilocalories per day. On average, most adult men need about 2500 kilocalories per day, and most adult women need about 2000 kilocalories per day.

Estimated energy requirements (EER) should, as far as possible, be based on estimates of energy expenditure. Energy expenditure can be derived in two ways: direct measurement or predictive equations (mathematical formulas).

The first method to determine EER is by direct measurement of TEE. This can be done by whole-body calorimetry (the gold standard), indirect calorimetry, or the doubly labeled water (DLW) technique. These methods are rarely used nowadays and will not be discussed in this chapter; a thorough discussion can be found elsewhere.[11-13]

The second method to determine EER is by predictive equations that estimate TEE. Three common formulas are as follows: a simplified approach using ideal body weight; a more complex and traditional approach using the Harris-Benedict equation (rarely used clinically anymore but presented for its historical significance); and a more modern predictive equation developed by the Institute of Medicine (IOM). Other excellent predictive equations not presented here include the Mifflin-St. Jeor equation[14] and the Penn State equations.[15,16]

IDEAL BODY WEIGHT TEE EQUATION

The easiest formula to use to calculate energy requirements is based on a predictive equation for TEE using a patient's ideal body weight (IBW):

$$\text{24-hour kcal requirement} = \text{TEE} = \text{IBW (in kg)} \times 30 \text{ kcal/kg}$$

This will give you a person's kilocalorie energy needs over 24 hours. To find a person's IBW, IBW tables exist, as do user-friendly smartphone applications. This simple formula is useful for clinical purposes as an initial estimate. Given the components of TEE, however, the fixed value of 30 kcal/kg will vary depending on the clinical circumstances, such as during the postinjury stress response or after a major burn.

It is important to know that IBW, much like daily caloric needs, is derived from a calculation. There are various methods to calculating IBW; a few of the more well-known methods to calculate IBW were derived by Lorentz[17] in 1929, Robinson et al[18] in 1983, Devine[19] in 1974, and Hamwi[20] in 1964. They all produce approximately the same value of IBW[21] and are all derived by calculations based on height and gender. IBW should not be confused with two other types of body weight calculations, namely the adjusted body weight (ABW)[22] and the lean body weight (LBW).[23]

Although IBW is recommended in this simplified predictive formula for calculating kilocaloric needs, there are exceptions. In obese patients, IBW is much lower than their actual body weight. If a patient is overweight to the point that actual body weight is greater than 30% over calculated IBW, the ABW is recommended for determining daily caloric needs (ABW can also be found in tables, smartphone/tablet applications, or online calculators).

LBW, meaning the total weight of all tissue in the body excluding fat (sometimes also referred to as fat-free mass), is the weight measure most closely correlated to BEE, REE, and TEE.[11] LBW explains about 70% to 80% of the variance in energy expenditure. The other 20% to 30% is affected by age, gender, baseline nutritional state, inherited variations, and different endocrine states.

LBW is extremely variable at different stages of life. For example, from birth to adulthood, the brain increases its LBW fivefold; the liver, heart, and kidneys, which are even more metabolically active, increase 10- to 12-fold; and muscle multiplies its LBW by about 40-fold.[24] The lean tissue content of the body declines with age, and this accounts in part for a progressive fall in BMR in relation to body size.[25]

In the elderly, there is a decrease in the proportion of skeletal mass, as well as muscle.[26] For all of these reasons, IBW is used to predict energy expenditure, not LBW.

There are times when it is important to calculate a healthy patient's basal energy requirements (BEE). To do this via the IBW method, a patient's BEE would be:

$$\text{BEE} = \text{IBW (in kg)} \times 24 \text{ kcal/kg}$$

BEE is thus IBW multiplied by a smaller factor of 23 to 24 kcal/kg. TEE equation of 30 kcal/kg represents a 25% increase over BEE of 24 kcal/kg.[27,28] Very limited activity increases the requirement to 28 kcal/kg.

HARRIS-BENEDICT EQUATION FOR BEE

The Harris-Benedict equations (HBEs)[29] are perhaps the best known equation to determine a patient's total kilocaloric needs over a 24-hour period. The equations were derived using indirect calorimetry. There are separate equations for males and females, and each is based on weight (in kilograms), height (in centimeters), and age (in years). The HBEs predict BEE (not TEE):

$$\begin{aligned}\text{Male 24-hour kcal requirement} = {} & (13.7516 \times \text{weight}) \\ & + (5.0033 \times \text{height}) \\ & - (6.755 \times \text{age}) \\ & + (66.4730)\end{aligned}$$

$$\begin{aligned}\text{Female 24-hour kcal requirement} = {} & (9.563 \times \text{weight}) \\ & + (1.8496 \times \text{height}) \\ & - (4.6756 \times \text{age}) \\ & + (655.095)\end{aligned}$$

Once the BEE has been calculated from the HBE, one needs to apply an activity factor multiplier to determine the TEE; the predicted value for TEE is then used as the EER for 24-hour kilocalorie needs. To do this, the baseline HBE values are multiplied as follows (the multipliers are the same for males and females)[30]:

24-hour baseline HBE BEE kcal requirement ×
- 1.2 (little to no exercise)
- 1.375 (low activity; exercise 1–3 d/wk)
- 1.55 (moderately active; exercise 3–5 d/wk)
- 1.725 (very active; exercise 6–7 d/wk)
- 1.9 (very active; vigorous exercise daily and physical job)

IOM TEE EQUATIONS

The IOM has published a set of formulas that predict TEE based on age, height, weight, and physical activity level. They were derived using the DLW method for directly measuring TEE and represent a more modern approach to determining EER[11] (the IOM equations were defined in 2005, whereas the HBE was derived >100 years ago, in 1918[29]). These IOM TEE equations have replaced the Schofield equation[31] to define and develop the US Food and Drug Administration's (FDA) dietary guidelines and formulate Recommended Dietary Allowances (RDAs).

The IOM TEE prediction formula to calculate EER for males age 19 years and older is as follows (with age in years, weight in kilograms, and height in meters):

$$\begin{aligned}\text{Male 24-hour kcal requirement} = {} & 662 - (9.53 \times \text{age}) \\ & + [\text{PA} \times \{(15.91 \times \text{weight}) + (539.6 \times \text{height})\}]\end{aligned}$$

where PA is the physical activity coefficient:

PA = 1.00 if sedentary
PA = 1.11 if low active
PA = 1.25 if active
PA = 1.48 if very active

The IOM TEE prediction formula to calculate EER for females age 19 years and older is as follows (with age in years, weight in kilograms, and height in meters):

$$\begin{aligned}\text{Female 24-hour kcal requirement} = {} & 354 - (6.91 \times \text{age}) \\ & + [\text{PA} \times \{(9.36 \times \text{weight}) + (726 \times \text{height})\}]\end{aligned}$$

where PA is the physical activity coefficient:

PA = 1.00 if sedentary
PA = 1.12 if low active
PA = 1.27 if active
PA = 1.45 if very active

It has been suggested that the HBE and IOM equations for TEE do not add that much more accuracy beyond the simple IBW equations; this is confirmed in multiple guidelines, which base TEE recommendations on predictive formulas[32-36] (Table 62-1).

TABLE 62-1: Comparison of Various Methods Used to Determine 24-Hour Kilocalorie Energy Requirements

Method[a]	Female[b]	Male[c]
IBW-based TEE	1695	2100
HBE (baseline BEE)	1429	1799
HBE TEE, sedentary	1715	2159
HBE TEE, low activity	1965	2474
HBE TEE, moderate activity	2216	2789
HBE TEE, very active	2466	3104
IOM TEE, sedentary	1907	2529
IOM TEE, low activity	1984	2672
IOM TEE, moderate activity	2081	2854
IOM TEE, very active	2197	3152

[a]Methodologies, equations, activity level multipliers, and references for calculating energy requirements can be found in the text.
[b]Female TEE in kcal was calculated for averages in United States: 40 years old, 69 kg, 163 cm (5 ft, 3 in); IBW (Lorentz) 56.5 kg.
[c]Male TEE in kcal was calculated for averages in the United States: 40 years old, 81.6 kg, 176.1 cm (5 ft, 9 in); IBW (Lorentz) 70 kg.

BEE, basal energy expenditure; HBE, Harris-Benedict equation; IBW, ideal body weight; IOM, Institute of Medicine; TEE, total energy expenditure.

Protein

Proteins are large, nitrogenous organic compounds that are made up of long chains of amino acids; there are 20 different amino acids in our body's proteins. Proteins exist in every cell, tissue, and organ in the human body and are an essential part of all living organisms. Proteins are major structural components of muscle, bones, skin, and collagen; along with amino acids, proteins function as enzymes, antibodies, membrane receptors, hormones, and carriers of nutrients in the blood. Protein balance is often referred to as nitrogen balance.

Humans obtain proteins solely from the diet; once ingested, proteins are digested into their component amino acids. Dietary proteins come in two general forms: complete proteins and incomplete proteins. *Complete proteins* (ie, whole protein; high-quality protein) come from animal-based products and supply all the amino acids, most importantly a group called the essential amino acids. There are nine *essential amino acids* (ie, indispensable amino acids) that cannot be synthesized de novo by humans: histidine, isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan, and valine. *Incomplete proteins* come from plants and lack one or more of the nine essential amino acids. When two or more incomplete proteins are combined to provide adequate amounts of all nine essential amino acids, they are referred to as *complementary proteins.*

Unlike fat (stored as adipose tissue) and carbohydrates (stored as glycogen in the liver and skeletal muscle), protein is not stored in the body. In contrast to other sources of energy, if more protein is ingested than is needed for metabolic purposes, all that excess nitrogen is metabolized and the end products are excreted. If less protein is ingested than is needed for homeostasis, leading to a persistent negative nitrogen balance, the body adapts by breaking down muscle, leading to a loss of LBW.[10] See Box 62-2.

What is a safe level of intake of dietary protein per 24 hours, and how are the nutritional protein needs determined? The protein requirement of an individual is defined as the lowest level of dietary protein intake that will balance the losses of nitrogen from the body (principally in the urine, but also in feces and through the skin) in persons maintaining energy balance at modest levels of physical activity.[24] Furthermore, we cannot simply maintain nitrogen balance; rather we must ensure that the required dietary protein intake includes both the essential amino acids and the nonessential amino acids. The amount of essential amino acids required in a healthy adult diet is 27.7%.[37]

There are two general methods for determining the daily recommended allowance for protein: direct measurement or predictive mathematical formula. The first method is to measure nitrogen loss, when the diet contains no protein, which will provide an estimate of nitrogen requirement. This has been done in nitrogen balance studies by collecting nitrogen losses in the urine, feces, skin, and sweat/secretions and subtracting these losses from measured nitrogen content of protein intake; the process is repeated multiple times, across several levels of protein intake, over many days, each time ensuring metabolic steady state has been reached. This

BOX 62-2: The Relationship Between Energy and Protein

Conceptually, there can be confusion about the link between energy and protein. On the one hand, food energy *includes* the energy from protein, which makes sense because protein not only provides a source of nitrogen and amino acids for the body, but also provides energy in the form of 4 kcal/g protein. In fact, the FDA's daily recommended value for protein is based on 10% of kilocaloric energy coming from protein.[16] However, on the other hand, nutritionists *separate* energy requirements from protein requirements. This practice seems to imply that the energy derived from protein is not included in the total energy needs, which is not true.

In practice, the total energy requirement (as determined by TEE) is calculated first, with no differentiation in how that energy will be derived (all three of the macronutrients can supply energy in the form of kilocalories). Next, the requirement for protein is calculated; one method to determine daily protein requirements is as a percentage of the TEE (see Protein section for specifics). In this manner, total energy requirements are determined independently of protein requirements, although protein requirements can be dependent on the total energy requirement calculation.

Energy and protein are fundamentally different in terms of how the body maintains and stores each. For energy, an individual's intake must match his output if he is to remain in a steady state, and physiologic mechanisms exist by which this balance is normally maintained. For example, when energy intake is below the requirement level, the body uses stores of energy to adapt. The opposite is true as well: when energy intake is above the required level, the body will store that excess energy, mostly as fat.

For protein, in contrast, there is no regulatory mechanism that matches intake to requirement. This is partly due to the fact that humans do not have protein reserves in the same way we have energy stored as glucose and fat; we are therefore dependent on protein intake to achieve required levels, as there are no protein stores to break down. In addition, if we take in more protein than meets the needs of the body, then this excess nitrogen is excreted.

Therefore, the body handles excess protein very differently compared to how it handles excess energy. This is a fundamental concept, and these considerations have led to very different approaches to deciding the amount of dietary protein to maintain nitrogen balance versus deciding the amount of dietary energy to maintain energy balance.

- For energy requirements, one intake achieves energy balance; we therefore determine the *average* requirement for energy.
- For protein requirements, on the other hand, there is a range of intakes to choose from to achieve nitrogen balance; therefore, we determine one intake within a *safe range of intake* for protein.[11]

process is quite impractical, although it is this process that forms the basis for estimating protein requirements using the predictive formula methodologies.[10]

The second method is to use one of two different mathematical formulas: the first based on a person's weight, and the second based on daily energy requirements.

PROTEIN REQUIREMENT USING WEIGHT-BASED METHOD

Determining dietary protein needs for healthy subjects using IBW is based on long-term nitrogen balance studies.[37] Specific equations have been derived from these studies, by both the World Health Organization[38] and the IOM,[39] and provide nearly identical results. The RDA for protein is as follows[38-40]:

24-hour protein requirement (in grams)
= 0.8 g protein/kg IBW

There are three factors that went into determining this 24-hour requirement value. The first is the average amount of high-quality protein needed to maintain nitrogen balance: 0.6 g protein/kg IBW/d. The second is a safety factor to ascertain that 95% of the healthy population's protein needs are covered: 0.15 g protein/kg IBW/d. The third is a buffer to allow for intake of proteins that are not high-quality proteins: 0.05 g protein/kg IBW/d.[37]

The equation is the same for both healthy adult men and women. Although differences in LBW exist between genders, as well as older and younger individuals, these differences are offset partially by differences in weight.[39] Accordingly, there is simply one equation for all healthy adults.

PROTEIN REQUIREMENT BASED ON ENERGY NEEDS

The most recent dietary reference intakes[11,39] for macronutrients are designed to reflect a broadened view of protein needs in relation to carbohydrate and fat requirements. This method, termed the *acceptable macronutrient distribution range* (AMDR), more clearly reflects the interrelation between the macronutrients and allows for some level of flexibility in diet planning. If an individual consumes below or above the AMDR range, there is potential to affect long-term health by increasing the risk of chronic diseases as well as increasing the risk of insufficient intakes of essential nutrients.[41] See Table 62-2 for AMDRs for all macronutrients.

The AMDR for dietary protein is a safe range of intake, from 10% to 35% of the total daily kilocalorie requirement.[39] This affords the ability to provide protein intake in excess of the RDA but within the AMDR; this has spawned the term *flexible calories*, because a clinician can use the AMDR for all three macronutrients to create varied dietary plans while still achieving kilocalorie needs.[40]

This alternative to the weight-based approach to determining protein needs is therefore based on the calculation for daily energy requirements. For example, the AMDR for protein is 10% to 35%; the FDA bases the daily recommended value (DRV) for protein on all food labels in the United States on a 10% rule (the low end of the AMDR for protein): 10% of 24-hour energy needs (as measured in kilocalories) should come from protein.[42] The equation for the 10% rule is:

24-hour protein requirement (in grams)
= (24-hour kilocaloric needs × 0.10)/4 kcal/g

A simpler way to calculate this is:

24-hour protein requirement (in grams)
= (24-hour kilocaloric needs × 0.025)

Although the FDA uses 10%, which is at the RDA level of 0.8 g protein/kg/d, one needs to consider the patient circumstances and thus consider other values within the AMDR for protein. For example, there are good data to show that physically active people should have protein intakes in the range of 1.2 to 2.0 g protein/kg/d (15%–25% of energy from protein).[43-45] There is other research that now demonstrates the benefits of increased protein intake in elderly adults to levels nearly double the RDA of 0.8 g protein/kg/d, the goal being to preserve lean body mass and promote functional ability with age.[46-48]

TABLE 62-2: Acceptable Macronutrient Distribution Ranges (AMDRs) for Macronutrients

Macronutrient	AMDR (as a percentage of 24-hour energy requirements)[a]
Carbohydrate	45%–65%
Protein	10%–35%
Fat	20%–35%
n-3 polyunsaturated fatty acids (linolenic acid)	0.6%–1.2%
n-6 polyunsaturated fatty acids (linoleic acid)	5%–10%

[a]Please see text for a full discussion of the meaning of AMDRs and the values for each macronutrient, as well as references for more information about AMDRs.

Carbohydrates

Carbohydrates are organic compounds that contain only carbon, hydrogen, and oxygen atoms in a ratio usually of 1:2:1; most carbohydrates, but not all, follow the general chemical formula of $C_n(H_2O)_n$. What distinguishes one carbohydrate from another is the different ways the atoms combine.

Carbohydrates (also called carbs) are the most important energy source for humans, particularly in the brain, which is a carbohydrate-dependent organ. In this manner, carbs can be thought of as the storage and transportation form of energy. Carbohydrates, however, are more than simply energy. Carbs also play major roles in our immune system, reproductive system, blood clotting, and cell structure; DNA and RNA are carbohydrate-based chains of molecules. The chemistry behind the structure of sugars is extremely complicated, and some have maintained that the genome is simple compared to the "glycome." The study of the glycome is referred to as *glycomics*, and the science of carbohydrates' role in life is termed *glycobiology*.

Carbohydrates are often referred to as sugars, or *saccharides*. Naturally-occurring saccharides are produced by photosynthetic plants. Unnatural sugars (eg, processed sugars, refined sugars, artificial sweeteners) are simply chemical compounds made in a lab and lack vitamins and minerals; they are referred to as "empty calories." The body digests most carbohydrates in the diet into glucose ($C_6H_{12}O_6$; ie, blood sugar). Glucose is then used in two ways: either it is used as a source of energy via cellular respiration, or it is stored as glycogen in the liver, skeletal muscle, and other tissues (excess glucose can also be stored as fat).

A *monosaccharide* is a carbohydrate in its elemental form, with one sugar unit. When two monosaccharides, or sugar units, combine they form a *disaccharide*. When 3 to 10 monosaccharides join, they form *oligosaccharides*, which have complex, varied, and critical biological functions. *Polysaccharides* are built from any combination of monosaccharides and disaccharides; examples of polysaccharides are glycogen, the storage form of glucose, and starch.

Carbohydrates come in two types—simple or complex—with the difference being their underlying chemical structure. *Simple carbohydrates* are sugars in their simplest form, either one (monosaccharides) or two (disaccharides) sugars. Examples of monosaccharides include glucose, fructose (ie, fruit sugar), and galactose; examples of disaccharides include sucrose (ie, table sugar) and lactose (ie, milk sugar).

Complex carbohydrates are made up of three or more simple carbohydrates bonded together into one larger compound; polysaccharides and oligosaccharides are complex carbs. Complex carbs come in two dietary forms: starch and dietary fiber. *Starches* are digested into sugars for energy; in addition to providing kilocalories, starches also contain vitamins and minerals. Complex carb–containing foods are popularly referred to as "starchy" foods.

Dietary fibers (ie, roughage; bulk; nondigestible carbohydrates) are plant-derived complex carbs that the human body cannot digest or absorb, meaning fiber traverses the gastrointestinal (GI) tract without being broken down. The health benefits of fibers are numerous and include helping to control weight by causing satiety, aiding digestion, preventing constipation, attenuating blood glucose levels, and decreasing serum cholesterol.[39] There are two general types of fiber: soluble and insoluble. *Soluble fiber* dissolves in water and forms a gel-like substance in the GI tract that helps to lower blood cholesterol and glucose levels. *Insoluble fiber* does not dissolve in water and therefore promotes movement of material through the GI tract and helps with constipation and laxation.

What is an average requirement for carbohydrates per 24 hours, and how are the nutritional needs for carbohydrates determined? In general, carbohydrate needs are determined as a percentage of 24-hour energy requirements.[11,39]

CARBOHYDRATE REQUIREMENT BASED ON ENERGY NEEDS

The RDA for carbohydrates is based on a minimum amount necessary to cover the glucose needs of the carbohydrate-dependent central nervous system.[11] With this in mind, when the FDA set the DRV for carbohydrates on all food labels in the United States, they used a very conservative value of 60%: 60% of 24-hour kilocalorie energy needs should come from carbohydrates.[42]

A more modern approach to determining daily carbohydrate needs is to consider the IOM's AMDR for carbohydrates.[11] The AMDRs delineate upper and lower bounds for the percentage of daily calories provided from all macronutrients; these safe ranges recognize the interrelation between the macronutrients and allow flexibility in creating customized diets to individual patient needs.

For carbohydrates, the AMDR ranges from 45% to 65% of total daily kilocalorie requirements[39] (see Table 62-2). This means that carbohydrate-derived energy can safely make up anywhere from 45% to 65% of total energy needs. The calculation for determining 24-hour carbohydrate requirements is based on 4 kcal of energy being produced from every gram of carbohydrate consumed (note that for parenteral nutrition calculations, the conversion is 3.4 kcal/g). For example, using the FDA's DRV of 60%, the equation is as follows:

$$\text{24-hour carbohydrate requirement (in grams)} = (\text{24-hour kcal energy needs} \times 0.60)/4 \text{ kcal/g}$$

To determine other values, one simply substitutes for any percentage, from 45% (use 0.45 in the equation) to 65% (use 0.65 in the equation).

Fat

Fats are an essential nutrient and a key part of our diet. They are characterized by being insoluble in water, nonvolatile, and greasy to touch. Fats are sometimes referred to as lipids, although technically speaking, fats are a member of the lipid family.

Lipids are a group of naturally occurring molecules that are derived from one of two common biochemical subunits (ketoacyl or isoprene). Lipid compounds include *fatty acids* (from which fats are derived); *glycerolipids* (triglycerides; diglycerides; monoglycerides; collectively glycerides); *glycerophospholipids* (ie, phospholipids); *sterol lipids* (eg, cholesterol and steroids); *saccharolipids* (eg, lipopolysaccharides in gram-negative bacteria); and others (see IOM Standing Committee on the Scientific Evaluation of Dietary Reference Intakes[11] for more details).

Fats provide a needed source of energy for the human body. As an energy source, fat produces 9 kcal of energy per gram, which is over twice as many kilocalories as can be derived from carbohydrates (4 kcal/g enterally; 3.4 kcal/g parenterally) and protein (4 kcal/g). For this reason, fat is referred to as nature's storehouse of energy. Fats are necessary for the body to absorb the fat-soluble vitamins A, D, E, and K. Fats help insulate the body and keep skin and hair healthy.

Fats also provide humans with the *essential fatty acids*: linoleic acid and linolenic acid (note that linoleic acid is the

precursor to arachidonic acid, so technically, arachidonic acid is not considered essential). Humans can synthesize all but these two essential fatty acids. Fats can be synthesized from the breakdown products of proteins, carbohydrates, or other fats (using acetate as an intermediate metabolite).

The essential fatty acids are crucial for membrane structure lipids, cell signaling pathways, brain development, controlling inflammation, and blood clotting. Prostaglandins are hormone-like compounds derived from arachidonic acid and are involved in many vital functions in the human body.

As a chemical compound, fats contain carbon, hydrogen, and oxygen, with the defining feature that they are arranged as a hydrocarbon chain skeleton, with a carboxyl group (-COOH) at one end and a methyl group (CH_3-) at the other. Fats are made from various fatty acids, which contribute approximately 95% of the total weight to various fats; the other 5% is from a backbone molecule to which the fatty acids bond, glycerol (and other occasionally attached molecules). Fatty acids are differentiated by the number of carbon atoms in their carbon skeleton as well as the number of carbon-to-carbon (C=C) double bonds. These differences lead to variations in structure and function. There are more than 100 different fatty acids in the human body, although less than 20 contribute to the majority of fats.

Fats are classified as either unsaturated or saturated. *Unsaturated fats* have at least one C=C double bond in the carbon skeleton, which gives them a low melting temperature. They are often liquids at room temperature, known as oils. There are two types of unsaturated fats: monounsaturated and polyunsaturated. *Monounsaturated fats* have only one C=C double bond in the carbon skeleton. *Polyunsaturated fats* have two or more C=C double bonds in the carbon skeleton. Polyunsaturated fats include the two *essential fatty acids*: omega-3 polyunsaturated fats (linolenic fatty acid) and omega-6 polyunsaturated fats (linoleic fatty acid).

Synthetic fats are unnatural, unsaturated fats created in an industrial lab when hydrogen is added to liquid vegetable oil (a natural unsaturated fat) to create solid fat. These fats are used mainly as preservatives for food and to add texture. Synthetic fats come in two types: *hydrogenated fats* and *partially hydrogenated fats* (ie, trans fats; trans fatty acids). Trans fats have been proven to increase cholesterol and the risk for heart disease[49,50] and have a detrimental effect on the brain and nervous system.[51] In short, they have no benefit to human health.[11] It should be noted that there are some trans fats that are naturally occurring, found in animal fats.

Saturated fats are so named because the carbon skeleton in these fats is saturated with hydrogen bonds, and therefore they have no C=C double bonds. These fats are solids at room temperature, indicating a high melting temperature. They are generally referred to as the solid fats and come from two general sources. The primary source is animals, including meats and dairy products (all animal-based saturated fats also contain cholesterol). The second source is certain plants. Saturated fats were once thought to be linked to coronary heart disease and increased cholesterol levels, although that is being questioned by more recent data.[52]

Cholesterol is an organic sterol lipid molecule synthesized by all animal cells. Cholesterol is an essential component of animal cell membranes and is the precursor to steroid hormones, bile salt (it was first discovered in gallstones), and vitamin D. It is found in all foods containing animal fat; cholesterol is not found in significant amounts in plant sources.

Triglycerides are lipid compounds derived when one molecule of glycerol combines with three fatty acids. Triglycerides are the major storage form of fat in human adipose tissue; the hydrolysis of triglyceride ester bonds is the first step in fat metabolism.

What is an average requirement for fat per 24 hours, and how are nutritional fat needs determined? Neither an adequate intake (AI) level nor an RDA exists for total fat. This is because there are not sufficient data to determine a level below which intake is inadequate or above which chronic diseases are prevented.[11] The AMDR for fat for healthy adults is 20% to 35% of the total daily kilocalorie requirement.[39] Accordingly, fat needs are determined as a percentage of 24-hour energy requirements.

FAT REQUIREMENT BASED ON ENERGY NEEDS

The AMDR for fat is 20% to 35% (see Table 62-2). This range was set to ensure that the essential fatty acids were consumed in adequate amounts. The FDA has based its DRV for fat on a value of 30%: 30% of the 24-hour energy needs (as measured in kilocalories) should come from carbohydrates.[42] The equation to calculate 24-hour fat requirements (using 30%, which can change depending on the percentage desired) is as follows:

$$\text{24-hour fat requirement (in grams)} = (\text{24-hour kilocaloric needs} \times 0.3)/9 \text{ kcal/g}$$

To ensure intake of adequate dietary amounts of the essential fatty acids, approximately 10% of total daily kilocalorie intake should come from longer-chain polyunsaturated fats[39] (see Table 62-2):

- Omega-3 polyunsaturated fat (linolenic fatty acid) intake should be in the range of 0.6% to 1.2% of 24-hour kilocalorie needs.
- Omega-6 polyunsaturated fats (linoleic fatty acid) should be in the range of 5% to 10% of 24-hour kilocalorie needs.

Current dietary recommendations also maintain that humans should consume less than 10% of total energy from saturated fats.

Water

The human body is made up of 60% water, and on average, 60% of an adult's total body weight is water.[53] Water is the basis of all fluids in our body: for example, bile is 90% water; blood is 92% water; and cerebrospinal fluid is 99% water. Water is also the basis of all organs and tissues in our body:

bone is 31% water; the brain and heart are 73% water; muscles and kidneys are 79% water; and lungs are 83% water.[54] Total body water (TBW) is distributed between the intracellular fluid (65% TBW) and extracellular fluid (35% TBW); of the TBW in the extracellular fluid, 79% is in the interstitial space and 21% is intravascular in plasma.

Water is made up of three atoms: two hydrogen and one oxygen. The simplicity of the water compound belies its importance, its versatility, and its complexity, in terms of both its physical and chemical properties. Water is the solvent, the medium, and the participant in most biochemical and physiologic reactions occurring in our bodies; it absorbs and releases metabolic heat; it is attracted to itself and many other substances; and it has tremendous surface tension and strength.

Human life and cellular homeostasis depend on water: we could live roughly a month without macronutrients; we can live only a few days without water. Once reaching 10% to 14% dehydration, risk of death increases rapidly, approaching irreversibility.[53] It is via water that carbohydrates and proteins are metabolized, Aqueous solutions are the universal transporter of macronutrients, oxygen, and waste. Cellular hydration is a crucial signal regulating cell metabolism and gene expression. Water aids in digestion, is a lubricant for joints, and is a shock absorber for the brain and spinal cord; and water helps regulate body temperature through sweating and respiration.[53,54]

What is an average requirement for water per 24 hours, and how are the nutritional water needs determined? There is no RDA for water as there are not sufficient data to determine a level above which chronic diseases are prevented.[53] The major risk associated with low intake of total water is dehydration, primarily in the acute setting, which can lead to metabolic and functional abnormalities. Accordingly, an AI has been established for water below which intake is inadequate. These AIs were defined to cover minimal losses from temperate climates for a sedentary individual.

The AI for total water per 24 hours to prevent dehydration is 3.7 L in adult men and 2.7 L in adult women[53]; 80% of this is typically consumed by liquids, meaning healthy adult men should drink 3.0 L (13 cups) per day and women 2.2 L (9 cups); the remaining balance of water comes from food. This intake of water will replace respiratory, urinary, fecal, and insensible fluid losses. Higher intakes should be achieved in physically active people or those exposed to hot environments.

Daily consumption of water below the AI may not translate into an added risk of dehydration because normal hydration can be achieved and maintained over a wide range of intake. Importantly, therefore, the AI is not a specific requirement.[53] In healthy adults, fluid intake, driven by consumption of liquids and food at meals as well as by thirst, maintains TBW at normal levels, preventing dehydration. Body water balance is achieved when water gain and water loss are equal. Water gain occurs from consumption (sources of water include drinking water, water in beverages, and water that is part of food) and production (metabolic water). Water loss occurs from respiratory loss, urinary/renal loss, fecal/GI tract loss, skin loss, and insensible loss.

The interrelationship between the macronutrients, other substances we consume, and water demonstrates the interconnectedness of homeostatic processes in humans. Increased or decreased intake of macronutrients as well as these other substances (eg, caffeine, alcohol, and sodium) can and will affect water requirements.[41]

For example, for dietary proteins and amino acids, the major end product of their metabolism is urea. Urea requires water for excretion by the kidneys. Therefore, increased protein intake requires increased water intake. For dietary carbohydrates, 100 g/d are required to prevent ketosis; consuming fewer carbs can increase ketone bodies, which require water to be excreted. Therefore, increased ketosis requires increased water. Finally, fecal water losses are increased with higher loads of dietary fiber; with increased fiber intake, more water needs to be consumed to prevent dehydration.

Micronutrients: Vitamins

The term *micronutrients* is used to refer to both vitamins and minerals. Vitamins are organic substances made by plants or animals; they are essential for growth and development. Vitamin deficiencies can be seen in the elderly (especially frail or institutionalized people with malnutrition), alcoholics (with associated malnutrition), illicit drug users, impoverished populations, and those in developing countries.

There is a long list of vitamins that humans need[41,55] (Tables 62-3 and 62-4), and they can be grouped together as follows:

- Fat-soluble vitamins:
  - Vitamin A (retinol, including the carotenoids)
  - Vitamin D
  - Vitamin E
  - Vitamin K
- Water-soluble vitamins:
  - B vitamins
    - Biotin
    - Folate (folic acid)
    - Niacin (nicotinic acid)
    - Pantothenic acid
    - Riboflavin (vitamin B_2)
    - Thiamin (vitamin B_1)
    - Vitamin B_6 (pyridoxine)
    - Vitamin B_{12} (cobalamins)
  - Vitamin C (ascorbic acid)
  - Choline

Micronutrients: Minerals

Minerals, on the other hand, are inorganic elements that come from the earth, soil, and water; all minerals are found in the periodic table of elements.[56] Minerals are absorbed by pants from the ground, and humans consume minerals from eating these plants. Minerals are essential for human homeostasis, as the body uses them for normal bone, muscle, heart, and brain function as well as for making hormones.[41]

TABLE 62-3: Vitamins: Functions, Deficiencies, and Toxicities[a]

Nutrient	Vitamin functions	Effect of deficiency	High risk deficiency patients and notes	Effect of toxicity
Fat-soluble vitamins				
Vitamin A (retinol)	Important for vision (formation of rhodopsin, a photoreceoptor in the retina), gene expression, reproduction, embryonic development, growth, immune function. Also integrity of epithelia; lysosome stability; glycoprotein synthesis.	Xerophthalmia (an irreversible drying of the conjunctiva and cornea) leading to night blindness; perifollicular hyperkeratosis; keratomalacia; increased morbidity and mortality in children.	People with high alcohol intake, preexisting liver disease, hyperlipidemia, or severe protein malnutrition may not be protected by the UL set for the general population, because the requirements for vitamin A are based on the assurance of adequate liver stores of vitamin A.	Hypervitaminosis A may be acute or chronic: headache; peeling of skin; hepatosplenomegaly; bone thickening; intracranial hypertension; papilledema; hypercalcemia; teratogenicity
Vitamin D	Involved in bone health (mineralization and repair). It aids in the absorption of calcium and phosphorus in the tubules of the kidneys, thereby helping maintain normal serum levels of these minerals. Also insulin and thyroid function, improvement in immune function, reduced risk of autoimmune disease.	Impair normal bone metabolism, which may lead to rickets in children and osteomalacia in adults. It is also implicated in osteoporosis in adults.	Older adults, especially those who live in northern industrialized cities of the world, are more prone to developing vitamin D deficiency (due to lack of sunlight).	Hypervitaminosis D: Hypercalcemia, hypercalciuria, and calcification of soft tissues, such as blood vessels and certain organs
Vitamin E	Functions as a chain-breaking antioxidant in the body by preventing the spread of free-radical reactions. Is an intracellular antioxidant; scavenger of free radicals in biologic membranes.	Peripheral neuropathy.	Generally occurring only as the result of genetic abnormalities of vitamin E metabolism, fat malabsorption syndromes, or protein-energy malnutrition.	Hemorrhagic toxicity (tendency to bleed)
Vitamin K	Functions as a coenzyme for biological reactions involved in blood coagulation (formation of prothrombin, other coagulation factors) and bone metabolism.	Classic sign of vitamin K deficiency is a vitamin K–responsive increase in prothrombin time and, in severe cases, bleeding; osteopenia.	Clinically significant vitamin K deficiency is extremely rare in the general population, with cases being limited to individuals with malabsorption syndromes or those treated with drugs known to interfere with vitamin K metabolism.	
Water-soluble vitamins				
B Vitamins				
Biotin	Functions as a coenzyme in bicarbonate-dependent carboxylation reactions.	Dermatitis, alopecia, conjunctivitis, and abnormalities of the central nervous system.	Individuals consuming raw egg whites over long periods and in patients receiving total parenteral nutrition (TPN) solutions that do not contain biotin.	NA

(*continued*)

TABLE 62-3: Vitamins: Functions, Deficiencies, and Toxicities[a] (*Continued*)

Nutrient	Vitamin functions	Effect of deficiency	High risk deficiency patients and notes	Effect of toxicity
Folate (folic acid)	Functions as a coenzyme in the metabolism of nucleic and amino acids (purines, pyrimidines, and methionine); maturation of red blood cells; development of fetal nervous system.	Decreased erythrocyte folate concentratin leads to macrocytic anemia (first evidenced by a low erythrocyte count and eventually by a low hematocrit and hemoglobin); neural tube birth defects; confusion.	Coexisting iron or vitamin B_{12} deficiency may interfere with the diagnosis of folate deficiency.To reduce the risk of neural tube defects, women able to become pregnant should supplement folic acid.	NA
Niacin (nicotinic acid)	Involved in many biological reactions (oxidation-reduction reactions), including intracellular respiration and fatty acid synthesis; carbohydrate and cell metabolism.	The classic disease of severe niacin deficiency is pellagra (dermatitis, glossitis, GI and CNS dysfunction), which in industrialized nations generally only occurs in people with chronic alcoholism or conditions that inhibit the metabolism of tryptophan.	People with an increased need for niacin include those with Hartnup disease, liver cirrhosis, carcinoid syndrome, and malabsorption syndrome, as well as those on long-term isoniazid treatment for tuberculosis or on hemodialysis or peritoneal dialysis. Also, pregnant females who are carrying more than one fetus or breastfeeding more than one infant may require additional niacin.	Flushing, nausea and vomiting, liver toxicity, and impaired glucose tolerance; however, most of the data on adverse effects has come from research with patients with special conditions who were treated with pharmacologic preparations.
Pantothenic acid	Functions as a component of coenzyme A (CoA), which is involved in fatty acid metabolism.	Irritability and restlessness, fatigue, apathy, malaise, sleep disturbances, hypoglycemia; neurobiological symptoms, such as numbness, paresthesias, muscle cramps, and staggering gait.	Pantothenic acid deficiency is rare and has only been observed in individuals who were fed diets devoid of the vitamin or who were given a pantothenic acid metabolic antagonist.	NA
Riboflavin (vitamin B_2)	Functions as a coenzyme in numerous oxidation–reduction reactions in several metabolic pathways (especially carbohydrate and protein metabolism) and in energy production; integrity of mucous membranes.	Sore throat, hyperemia and edema of the pharyngeal and oral mucous membranes, cheilosis, angular stomatitis, glossitis, seborrheic dermatitis, and normocytic anemia associated with pure erythrocyte cytoplasia of the bone marrow.	Riboflavin deficiency is most often accompanied by other nutrient deficiencies, and it may lead to deficiencies of vitamin B_6 and niacin, in particular. Diseases such as cancer, cardiac disease, and diabetes mellitus are known to precipitate or exacerbate riboflavin deficiency.	NA
Thiamin (vitamin B_1; aneurin)	Functions as a coenzyme in the metabolism of carbohydrates, fat, branched-chain amino acid, glucose, and alcohol metabolism; central and periphera nerve cell function; myocardial function.	Beriberi (peripheral neuropathy, heart l failure); Wernicke-Korsakoff syndrome.	The classic disease of thiamin deficiency is beriberi, which is sometimes seen in developing countries. Severe thiamin deficiency in industrialized nations is often associated with chronic heavy alcohol consumption and presents as Wernicke-Korsakoff syndrome.	NA

(*continued*)

TABLE 62-3: Vitamins: Functions, Deficiencies, and Toxicities[a] (*Continued*)

Nutrient	Vitamin functions	Effect of deficiency	High risk deficiency patients and notes	Effect of toxicity
Vitamin B_6 (pyridoxine)	Functions as a coenzyme in the metabolism of amino acids (protein, nitrogen), glycogen, and sphingoid bases (transaminations, porphyrin and heme synthesis, tryptophan conversion of niacin).	The signs and symptoms of vitamin B_6 deficiency are seborrheic dermatitis, microcytic anemia, epileptiform convulsions, and depression and confusion.		Sensory peripheral neuropathy and dermatologic lesions.
Vitamin B_{12} (cobalamins)	Functions as a coenzyme for a reaction that converts homocysteine to methionine and for a separate reaction in the metabolism of certain fatty acids and amino acids; maturation of red blood cells; neural function; DNA synthesis; myelin synthesis and repair.	Macrocytic anemia; neurologic deficits (confusion, parethesias, ataxia).	The major cause of vitamin B_{12} deficiency is pernicious anemia, a condition in which the gastric mucosa of the stomach does not produce intrinsic factor. The hematologic effects that occur with this deficiency are identical to those observed in folate deficiency.	NA
Vitamin C (ascorbic acid)	Acts as an antioxidant and a cofactor in enzymatic and hormonal processes. It also plays a role in the biosynthesis of carnitine, neurotransmitters, collagen, and other components of connective tissue (bone and blood vessel health and wound healing), and modulates the absorption, transport, and storage of iron.	The classic disease of severe vitamin C deficiency is scurvy (follicular hyperkeratosis, petechiae, ecchymoses, coiled hairs, inflamed and bleeding gums, perifollicular hemorrhages, joint effusions, arthralgia, and impaired wound healing).	Severe vitamin C deficiency is rare in industrialized countries, but it is occasionally seen in people whose diets lack fruits and vegetables or in those who abuse alcohol or drugs.	Diarrhea and other gastrointestinal disturbances.
Choline	Required for the structural integrity of cell membranes. It is also involved in methyl metabolism, cholinergic neurotransmission, transmembrane signaling, and lipid and cholesterol transport and metabolism.	Liver damage.	Few data exist on the effects of inadequate dietary intake in healthy people.	Fishy body odor (trimethylaminuria), sweating, salivation, hypotension, and hepatotoxicity in humans.

[a]Sources referenced in text.

CNS, central nervous system; GI, gastrointestinal; NA, not applicable; UL, upper limit.

SIX MACROMINERALS

These are needed in large amounts (in the range of grams per day) and are usually simply referred to as *electrolytes* (will be discussed in a later section). The four cation electrolyte macrominerals are as follows: sodium (Na; element 11; an alkali metal); potassium (K; element 19; an alkaline earth metal); calcium (Ca; element 20; an alkaline earth metal); and magnesium (Mg; element 12; an alkaline earth metal).

The two anion electrolyte macrominerals are as follows: chloride (Cl, chlorine; element 17; a nonmetal halogen gas) and phosphorus (P; element 15; a nonmetal solid).

TABLE 62-4: Vitamin Dietary Reference Intakes by Life Stage Group[a]

	Females (age range in years)				Males (age range in years)				UL[b]		
Nutrient	**14–18**	**19–50**	**51–70**	**71+**	**14–18**	**19–70**	**51–70**	**71+**	**14–18**	**19–70**	**71+**
Fat-soluble vitamins											
Vitamin A (retinol; mcg RAE/d)[c]	700	700	700	700	900	900	900	900	3000	3000	3000
Vitamin D (mcg/d)[d]	5	5	10	15	5	5	10	15	50	50	50
Vitamin E (mg/d)[e]	15	15	15	15	15	15	15	15	800	1000	1000
Vitamin K (mcg/d)[d]	75	90	90	90	75	120	120	120	—	—	—
Water-soluble vitamins											
B Vitamins											
Biotin/(mcg/d)[d]	25	30	30	30	25	30	30	30	—	—	—
Folate (mcg/d)[e]	400	400	400	400	400	400	400	400	800	1000	1000
Niacin (mg/d)[e]	14	14	14	14	16	16	16	16	30	35	35
Pantothenic acid (mg/d)[d]	5	5	5	5	5	5	5	5	—	—	—
Riboflavin (vitamin B_2; mg/d)[e]	1.0	1.1	1.1	1.1	1.3	1.3	1.3	1.3	—	—	—
Thiamin (vitamin B_1; mg/d)[e]	1.0	1.1	1.1	1.1	1.2	1.2	1.2	1.2	—	—	—
Vitamin B_6 (pyridoxine; mg/d)[e]	1.2	1.3	1.5	1.5	1.3	1.3	1.7	1.7	80	100	100
Vitamin B_{12} (cobalamins; mcg/d)[e]	2.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4	—	—	—
Vitamin C (ascorbic acid; mg/d)[d]	65	75	75	75	75	90	90	90	1800	2000	2000
Choline (mg day)[d]	400	425	425	425	550	550	550	550	3000	3500	3500

[a]Sources referenced in text.
[b]UL, tolerable upper intake level; ULs are the largest amount of a nutrient that most adults can consume without risk of adverse health effects. If blank, data were insufficient to set a UL.
[c]Values represent Recommended Dietary Allowance (RDA); RAE, retinol activity equivalent. Note that food and supplement labels usually state vitamin A levels in international units (IUs). One IU of retinol = 0.3 mcg retinol = 0.3 mcg RAE. Further note that 1 mcg RAE = 1 mcg retinol = 12 mcg beta-carotene = 24 mcg alpha-carotene or beta-cryptoxanthin. The RAE for dietary provitamin A carotenoids in foods is twofold greater than retinol equivalents (RE), whereas the RAE for preformed vitamin A in foods is the same as RE.
[d]Values represent adequate intake (AI) level; when data to calculate an RDA are insufficient, AIs are determined based on the intake of healthy people.
[e]Values represent RDA; RDAs are set to meet the needs of 97% to 98% of healthy people.

NINE TRACE MINERALS (TABLES 62-5 AND 62-6)[41,57]

Nine trace minerals, which are needed in small amounts (in the milligram or less per day range), are as follows: chromium (Cr; element 24; a transition metal); copper (Cu; element 29; a transition metal); fluoride (F, fluorine; element 9; a nonmetal halogen gas); iodine (I; element 53; a nonmetal halogen); iron (Fe; element 26; a transition metal); manganese (Mn; element 25; a transition metal); molybdenum (Mo; element 42; a transition metal); selenium (Se; element 34; a nonmetal solid); and zinc (Zn; element 30; a transition metal).

OTHER MINERALS

Other minerals used by the body (not discussed in depth in this chapter; for more information, see IOM report referenced here[41]) are as follows: sulfate (S, sulfur; element 16; a nonmetal solid); arsenic (As; element 33; a metalloid); boron (B; element 5; a metalloid); nickel (Ni; element 28; a transition metal); silicon (Si; element 14; a metalloid); and vanadium (V; element 23; a transition metal).

NUTRITIONAL SUPPORT: ABNORMAL STATE OF HEALTH

Just as dietary factors contribute to increasing the risk for certain diseases, the reverse is also very true: disease processes can have a profound impact on nutritional status and needs. The optimal dietary requirements for a normal healthy adult (and thus the trauma patient with minor injuries), as discussed in the last section, may need to be adjusted for both acute and chronic conditions, in both the inpatient and outpatient setting, and for both medical and surgical disease.

TABLE 62-5: Trace Minerals: Functions, Deficiencies, and Toxicities[a]

Nutrient	Function	Effect of deficiency	Effect of toxicity
Chromium	Promotes glucose tolerance	Possibly impaired gluocse tolerance	NA
Copper	Enzyme component, hematopoiesis, bone formation	Anemia in undernourished children, Menkes syndrome	Wilson disease, copper poisoning
Fluorine	Bone and tooth formation	Predisposiiton to dental caries, possibly osteoporosis	Fluorosis mottling and pitting of permanent teeth, exostoses of spine
Iodine	Thyroxine (T_4) and triiodothyronine (T_3) synthesis, development of fetus	Simple goiter, cretinism, deaf-mutism, impaired fetal growth and brain development	Hyperthyroidism or hypothyroidism
Iron	Hemoglobin and myoglobin formation, cytochromc enzymes, iron-sulfur proteins	Anemia, pica, glossitis, angular cheilosis	Hemochromatosis, cirrhosis, diabetes mellitus, skin pigmentation
Manganese	Healthy bone structure Component of manganese-specific enzymes: glycosyltransferases, phosphoenolpyruvate carboxykinase, manganese-superoxide dismutase	NA	Neurologic symptoms resembling those of parkinsonism or Wilson disease
Molybdenum	Component of coenzyme for sulfite oxidase, xanthine dehydrogenase, and one aldhyde oxidase	Tachycardia headache nausea obtundation (sulfite toxicity)	NA
Selenium	Component of glutathione peroxidase and thyroid hormone iodinase	Keshan disease (viral cardiomyopathy), muscle weakness	Hair loss, abnormal nails, nausea, dermatitis, peripheral neuropathy
Zinc	Enzyme component, skin integrity, wound healing, growth	Impaired growth and delayed sexual maturation, hypogonadism, hypogeusia	Red blood cell microcytosis, neutropenia, impaired immunity

[a]Sources referenced in text.

NA, not applicable.

TABLE 62-6: Trace Mineral Dietary Reference Intakes by Life Stage Group[a]

	Females (age range in years)				Males (age range in years)				UL[b]			
Nutrient	14–18	19–30	31–50	51+	14–18	19–30	31–50	51+	14–18	19–30	31–50	51+
Chromium (mcg/d)[c]	24	25	25	20	35	35	35	30	—	—	—	—
Copper (mcg/d)[d]	890	900	900	900	890	900	900	900	8000	10000	10000	10000
Fluorine (mg/d)[c]	3	3	3	3	3	4	4	4	10	10	10	10
Iodine (mcg/d)[d]	150	150	150	150	150	150	150	150	900	1100	1100	1100
Iron (mg/d)[d]	15	18	18	8	11	8	8	8	45	45	45	45
Manganese (mg/d)[c]	1.6	1.8	1.8	1.8	2.2	2.3	2.3	2.3	9	11	11	11
Molybdenum (mcg/d)[d]	43	45	45	45	43	45	45	45	1700	2000	2000	2000
Selenium (mcg/d)[d]	55	55	55	55	55	55	55	55	400	400	400	400
Zinc (mg/d)[d]	9	8	8	8	11	11	11	11	34	40	40	40

[a]Sources referenced in text.
[b]UL, tolerable upper intake level. If blank, data were insufficient to set a UL.
[c]Values represent adequate intake level.
[d]Values represent Recommended Dietary Allowance.

This necessary alteration to nutritional needs occurs because in a state of abnormal health there can be a profound imbalance among the metabolic regulatory mechanisms that act to keep the body in a condition of normal physiologic function. The imbalance in metabolic and physiologic processes leads to changes in energy expenditures and thus energy requirements, driven by complex feedback mechanisms.[11] These changes to energy balance, triggered by metabolic and behavioral responses to disease, are crucial since bodily function depends on energy transformations. As such, changes to energy metabolism can profoundly alter baseline dietary requirements in an effort to return the body to normal function.

A body in homeostasis implies there is balance of physiologic function, and the body has adequate energy reserves and nutrients. Mild disturbances in homeostasis lead to *adaptation*, defined as a process by which a new or different steady state is reached, without loss of function.[24] An example is the increase in hemoglobin concentration that occurs when individuals live at high altitudes.[11] Another example, in response to a change or difference in the intake of food and nutrients, is the decrease in REE during starvation. Adaptation therefore involves changes in body composition that occur over a more extended period of time, without any discernible detriment to health.[11,24]

More severe disturbances in homeostasis may be detrimental if they exceed adaptive capacity; these severe alterations to optimal function lead to *accommodation*. Accommodation involves relatively short-term adjustments, with the loss of less vital physiologic function and attempts by the body to preserve and maintain the most essential functions, all in an effort to achieve homeostasis.[10] Accommodation is therefore an adaptive response to a disturbance that allows survival but results in some degree of serious consequences to health or physiologic function.[11,24] An example of accommodation, with a resultant initial breakdown in homeostatic mechanisms, is the hypermetabolic state, which can be induced by major trauma and injury.

The Stress Response to Trauma and Injury

In addition to the anatomic injuries produced by a traumatic accident, injured patients are susceptible to developing a profound and systemic physiologic inflammatory response. This is known as the stress response to trauma and can last for days to months after the initial injury. The stress response is multifaceted, driven by metabolic changes as well as alterations to the nervous, endocrine, and immune systems.[58,59] These changes can have severe negative downstream consequences, including inflammatory, immunologic, hematologic, and hemodynamic effects.[59,60] These changes can catalyze the systemic inflammatory response syndrome (SIRS), multiple organ dysfunction syndrome, the postinjury multiple organ failure (MOF), and, at its worst, death.[61] The stress response can also affect protein, carbohydrate, and fat metabolism throughout the body,[62] which impacts the nutritional needs for both macronutrients and micronutrients.[63]

The metabolic response to trauma was first described in the early 1930s by a Scottish veterinarian-physiologist named Dr. David Cuthbertson (he was later knighted for his work).[64,65] Cuthbertson showed increased protein metabolism after injury and demonstrated that this was due to increased muscle catabolism. This hypercatabolic state was most pronounced from postinjury days 2 to 8 and, in some patients, lasted up to 2 months. The hypermetabolism was associated with corresponding physiologic changes to heart rate, body temperature, and oxygen consumption.[64,65]

The systemic stress response to trauma is extremely complex and driven by both the primary injury (tissue ischemia/reperfusion and tissue disruption) as well as secondary insults (blood transfusions, delayed operative procedures, infection), and is further compounded by innate gene expression and genetic polymorphisms.[66] Traditionally, the posttraumatic metabolic response is divided into two phases: an ebb phase and a flow phase, with the flow phase having both a catabolic period and an anabolic period.[67] These phases define the metabolic shifts after a major injury, define the deleterious systemic effects a local injury can produce, and define the alterations to energy requirements and nutritional needs after an injury.[67]

The Ebb Phase

The ebb phase is the body's short-term attempt to preserve energy after an injury. This period is marked by depressed cellular metabolism, with decreased body temperature and oxygen consumption.[67] This stage is the period of traumatic shock, dominated by circulatory fluctuations that require resuscitation with fluids and blood products.[67,68] It represents the immediate consequences of the inciting injury and can lead to whole-body ischemia/reperfusion injury and activation of a nervous system response with neuronal and humoral mediators.[66] The ebb phase of the stress response is also known as the nervous system phase, the ischemia/reperfusion phase, the immediate or first phase, and the resuscitation phase.[58,66]

As an example of the profound neuroendocrine changes during the ebb phase, alterations occur to thermoregulatory capacity of the hypothalamus, leading to a drop in core body temperature. The neuroendocrine-driven hypothermia from an overwhelming traumatic injury does not induce shivering, as would normally occur in environmentally driven hypothermia; it has therefore been compared to hibernation.[58] This is indicative of the protective *hypometabolism* of this resuscitation phase, as the body conserves energy to increase the chances of cellular survival.[69]

The ebb phase is typically measured in hours, usually from 8 to 24 hours.[60] The variable intensity and duration of the ebb phase are related to the severity of the injury[69]: the more severe the injury, the shorter the ebb phase and the quicker the onset of the flow phase. Before returning to a normal

metabolic state after an injury, the body must pass through the flow phase.

The ebb phase can be so severe as to overwhelm the body's adaptive mechanisms to a point beyond which the body cannot recover. This terminal ebb phase is simply referred to as irreversible shock. Physiologic mechanisms at work include profound hypothermia, depressed oxygen consumption below BEE, fall in cardiac output, vasoconstriction, increased blood viscosity, intravascular coagulation, profound buildup of lactate due to anaerobic metabolism, failure of buffering capacity and concomitant acidosis, and eventual death.

TARGETED METABOLIC THERAPY FOR THE EBB PHASE

Reducing initial damage caused by the early postinjury pathophysiologic processes in the ebb phase could determine a more favorable outcome,[58] especially for patients with evidence of postinjury early MOF.[70] The overall goal of targeted metabolic and nutritional therapy in this phase is to diminish the deleterious effects related to ischemia/reperfusion. Interestingly, the link between the hypermetabolic response and the postinjury MOF is evidenced by the fact that postinjury MOF was once referred to as the "hypermetabolism organ failure complex."[71] Concepts such as damage control surgery[72] and damage control resuscitation[73] have evolved as therapeutic stopgaps to improve survival during this phase.

Although the one-hit model of MOF (examples of "one hits" are severe organ and soft tissue injury, hemorrhagic shock, and profound hypoxia) stresses the importance of an overwhelming initial insult that precipitates severe SIRS and then MOF, there is also a two-hit model. In the two-hit hypothesis for the development of MOF, the first hits are less severe than in the one-hit model (examples are milder hypotension, ischemia, and resuscitation), leading to a whole-body proinflammatory reaction, but not SIRS. A second hit then occurs (eg, sepsis, blood transfusion, second operation, mechanical ventilation), leading to SIRS and MOF.[61]

The first hits in both models occur during the ebb phase. Initial targeted treatment goals are to dampen the impact of the second hits.[59] For example, global hypoperfusion during the ebb phase can cause GI tract hypoxia, which primes the intestinal microvasculature, rendering it more susceptible to a secondary challenge.[58,74] Once that secondary challenge occurs in the form of reperfusion during resuscitation, the ischemic gut releases cytokines and proinflammatory mediators. This eventually leads to mobilization of neutrophils and possible MOF,[61,75] as they cause direct tissue damage as well as systemic damage via release of cytokines.

Theoretically, identification of patients at high risk for MOF could facilitate institution of early metabolic and/or nutritional therapies to dampen the proinflammatory response.[61] Supplementation with intraluminal glutamine has proved controversial,[66,76] and pulse steroids have been unsuccessful.[77] Evidence of the benefits of early β-blockers is mainly for burn patients.[66]

Catabolic Flow Phase

What follows the ebb phase is the postshock, postresuscitation catabolic flow period, defined by systemic posttraumatic inflammation and increased metabolism as the body tries to repair itself.[68] These integrated metabolic, inflammatory changes occur in virtually all organs and tissues in the body.[62] The need for injury repair stimulates the hypercatabolic state, driven by cytokine mediators released from lymphocytes and macrophages, dominated by interleukin (IL)-6.[60,62] This leads to a functional redistribution of body cell mass to provide amino acids for gluconeogenesis and protein synthesis[60,62,78] (Fig. 62-1). The catabolic state is maintained by proinflammatory cytokines and catabolic hormones for periods long after the acute trauma.[79]

The hypermetabolic changes are associated with neuroendocrine and immune system reactions.[58,59] The neuroendocrine component of the hypermetabolic response includes raised blood concentrations of stress hormones: the glucocorticoid cortisol (also a catabolic hormone/steroid; released from adrenal cortex) and the catecholamine epinephrine (from the adrenal medulla).[60,62] The immune component is multifaceted and diffuse, with a variety of hormones and proinflammatory cytokines bridging the innate and adaptive immune response.[80] Over time, the immune-inflammatory response can lead to SIRS and, if severe enough, postinjury MOF and compensatory anti-inflammatory response syndrome.[59,61,80,81]

This phase's collective actions are characterized by a hyperdynamic, hypermetabolic response, with increased oxygen consumption (Fig. 62-2), increased energy expenditure, increased body temperature, and increased heart rate as well as proteolysis, glycogenolysis, and lipolysis.[58,67] The resultant accelerated catabolism causes breakdown of skeletal muscle, with negative nitrogen balances and loss of body weight (referred to as auto-cannibalism).[60,82] There is mobilization of stored carbohydrate (glycogen via glycogenolysis) and peripheral insulin resistance leading to hyperglycemia.[66,69] Additionally, there is mobilization of fat (triglyceride via lipolysis) with resulting increase in the plasma concentration of fatty acids and triglycerides[66,69]; the rate of fat oxidation is twice that in a normal human.[78]

Overall, it is the initiation of the innate cellular immune system (monocytes, macrophages, neutrophils, endothelium) coupled with other immunologic changes (activation of complement and coagulation cascades, with resultant release of myriad mediators, including cytokines) that feed the hypermetabolic process.[63] In addition to driving the metabolic response, these factors also drive microvascular thrombosis, mitochondrial dysfunction, cellular necrosis and apoptosis, and secondary remote organ dysfunction (Fig. 62-3).[66]

The catabolic flow period lasts for at least 7 days and, at its most severe, up to 3 weeks or longer (note that in burn patients the hypermetabolism can last up to 2 years).[63,69] The proportion of this inflammatory response is directly related to the intensity of the injury. This intermediate phase is also known as the immune phase (due to activation of the innate immune system) or the leukocytic phase.

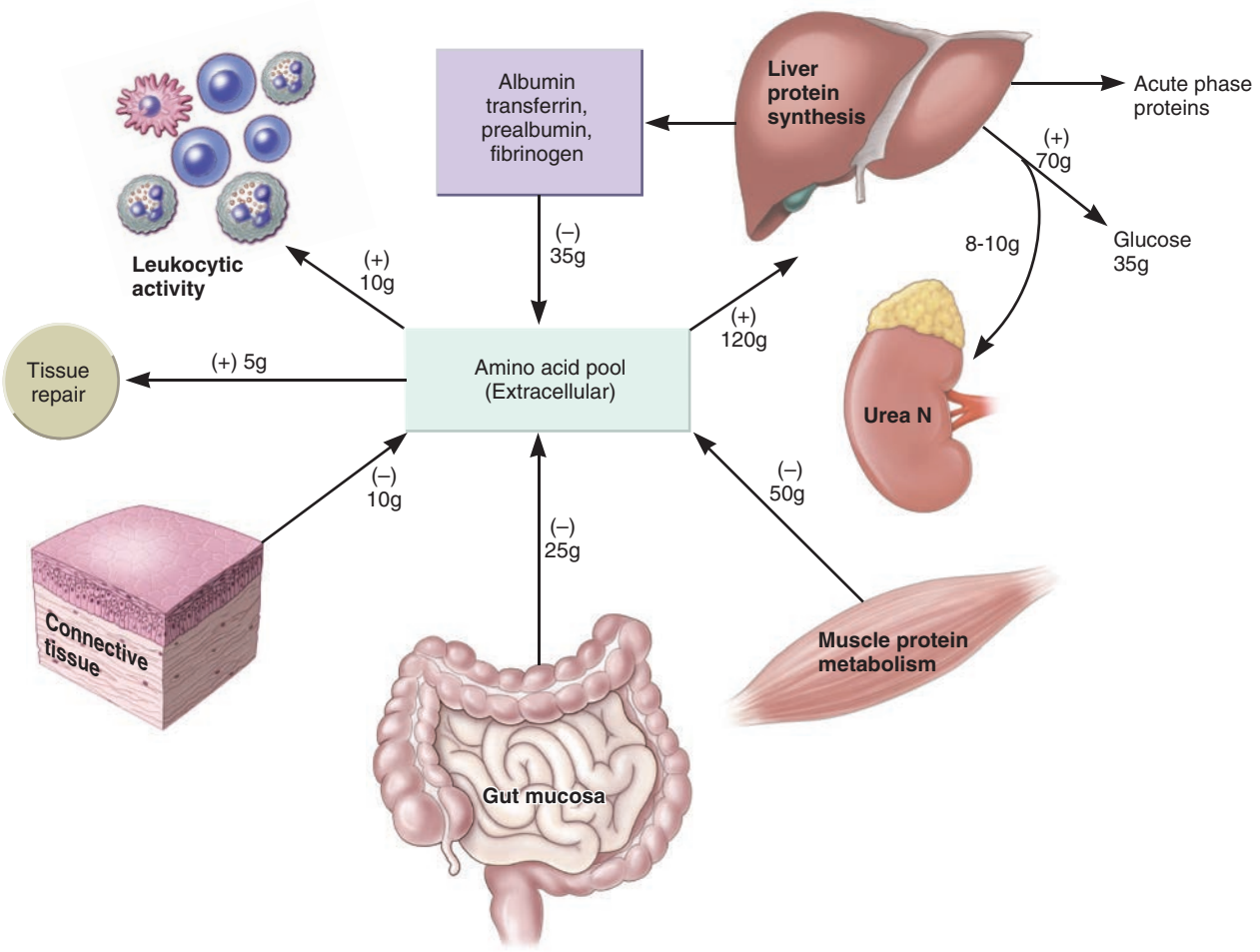

FIGURE 62-1 Functional redistribution of body cell mass after injury provides nitrogen for protein synthesis. Arrows reflect the net release (–) in grams from connective tissue, gut mucosa, and muscle as well as uptake (+) of amino acids into tissues whose net metabolism is associated with survival. The conversion of protein into glucose and urea is a minor source of energy but an important part of the role of the liver to produce the heat necessary to maintain core temperature. (Reproduced with permission from Blackburn GL. Metabolic considerations in management of surgical patients. *Surg Clin North Am*. 2011;91(3):467-480. Copyright © Elsevier.)

TARGETED METABOLIC THERAPY FOR THE CATABOLIC FLOW PHASE

The metabolic changes during this catabolic flow phase catalyze a redistribution of macronutrients and micronutrients (see Fig. 62-1). Labile reserves of protein (skeletal muscle) and fat (adipose tissue) are broken down to provide energy to more active tissue (liver and bone marrow) for host defense, visceral protein synthesis, and heat production.[60] These metabolic alterations in the severely injured patient need to be recognized and addressed during their initial and acute stages.[66,82] The hypermetabolic state increases overall kilocalorie energy requirements, and the increased catabolism with skeletal muscle breakdown increases dietary protein requirements. Early enteral or parenteral nutrition has proven beneficial in this regard (discussed in detail later).

Given the importance of the immune system in driving and maintaining the catabolic flow phase, nutritional therapies to directly alter and target immune function and immune mediators are in theory both logical and possible. Recommendations for glutamine, omega-3 polyunsaturated fatty acids, and other immune-enhancing supplements for trauma patients have been supported in the past. However, new data are changing the landscape of immunonutrition (discussed in detail later), and there is now true clinic equipoise with regard to its use.

This phase's hypercatabolic state is associated with severe complications related to hyperglycemia, hypoproteinemia, and immunosuppression.[60,66] These processes can lead to multisystem organ dysfunction, SIRS, and postinjury MOF.[61] For these reasons, early initiation of glucose protocols with sliding scale insulin is essential, as is attention to nutritional therapy.

Anabolic Flow Phase

The anabolic flow phase gradually occurs as the patient's posttraumatic metabolism shifts from catabolism to synthetic

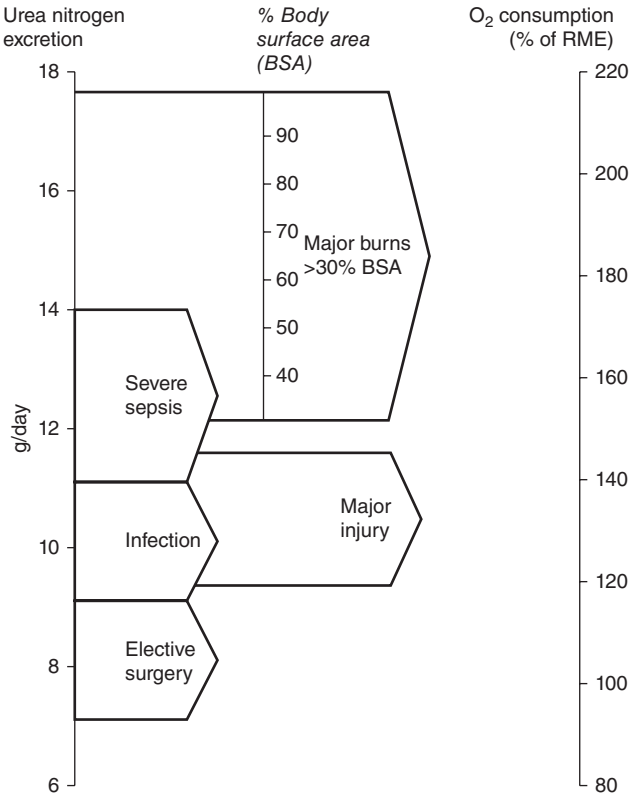

FIGURE 62-2 Rates of hypermetabolism estimated from urinary urea nitrogen excretion. Degree of hypermetabolism differs greatly: for elective surgery, ~100%–110% of resting energy expenditure (REE); for infection and major injury/trauma, ~120%–145% of REE; for severe sepsis, ~140%–170% of REE; and for major burns (>30% total body surface area), ~150%–220% of REE. REE concept explained in the text. RME, resting metabolic expenditure. (Reproduced with permission from Blackburn GL. Metabolic considerations in management of surgical patients. *Surg Clin North Am*. 2011;91(3):467-480. Copyright © Elsevier.)

activities and reparative processes.[63] This phase reprioritizes protein synthesis in the liver, known as the acute phase response. Mediating this phase is increased adrenal cortical hormone secretion as well as a variety of cytokines (IL-1, IL-6, tumor necrosis factor-α), which stimulate the liver's synthesis of the acute phase reactants, including fibrinogen and C-reactive protein,[69] initially at the expense of constitutive proteins such as albumin.[60]

Eventually a convalescence is achieved, as the hypercatabolic syndrome is progressively downregulated with reduction of catabolic hormones (catecholamines, proinflammatory cytokines, cortisol, glucagon) and increase of anabolic hormones (insulin, growth hormones, insulin-like grown factor 1, anabolic steroids).[58] With time, there is an exponential increase in the levels of positive acute phase proteins and a decrease in levels of negative acute phase proteins.[63]

This phase is also characterized by a return of oxidative metabolism, leading to angiogenesis in the injured tissues and organs, facilitating tissue repair and regeneration.[66] For this reason, the phase is also known as the angiogenic phase or the endocrine phase, as it is the endocrine functional system that facilitates oxygen transport.[58] With time, there is full recovery of the endocrine system (hypothalamic-pituitary-organ-hormonal axes), the nervous system (autonomic nervous systems), and the immune system (innate and adaptive).[58] These responses make possible the complex process of resolution of inflammation as well as a return to normal homeostasis.

Nutrient Utilization and Needs After Trauma and Injury: Seven Key Questions

Depending on the severity of the injury, traumatically injured patients are at risk for developing a hypermetabolic posttraumatic stress response (discussed in the last section). If the metabolic insults persist, the body goes into a hyperdynamic state, with breakdown of skeletal protein and rapid loss of lean body mass. Potential complications from this state of hypercatabolism associated with posttraumatic hyperglycemia, hypoproteinemia, immunosuppression, and the multitude of other effects include onset of protein-calorie malnutrition, infectious morbidity, multiorgan dysfunction, prolonged hospital stays, postinjury MOF, and disproportionate death.[34,35,61,83-86]

The inflammatory stress response can be broken down into three phases: ebb, catabolic flow, and anabolic flow. Each of these phases induces distinct pathophysiologic changes that can require interventions to eliminate or minimize their untoward consequences.[63] One of the most fundamental and important interventions is nutrition.[63,82,83,87-90]

WHY IS NUTRITIONAL SUPPORT IMPORTANT?

Early recognition of the hypermetabolic state and adequate early nutritional support are essential to not only meet the patient's increased nutritional needs (and thus preserve lean body mass), but also to facilitate recovery and healing, restore the body's capacity for optimal immune function, prevent oxidative cellular injury, attenuate the inflammatory stress response, and prevent complications.[83] In this sense, early nutrition has gone from supportive care to outright therapeutic care[35] and can be thought of as "metabolic control."[83,84,88]

WHO NEEDS POSTINJURY NUTRITIONAL SUPPORT?

All trauma patients will benefit from nutritional support, especially early in the hospitalization in those who are severely injured or those with baseline malnutrition prior to the injury. Severely injured patients are at highest risk for developing a hypermetabolic state and subsequent malnutrition.

The metabolic response to trauma in clinical terms is an "all-or-none" response: the patient with an Injury Severity Score (ISS) of 18 is metabolically similar to the patient with an ISS of 50.[78] For this reason, some have advocated using an ISS of greater than 16 to identify severely injured patients who are at risk for hypercatabolism and malnutrition.[36] Although this is certainly possible, more often identifying a severely injured patient is done by clinical judgment. It is

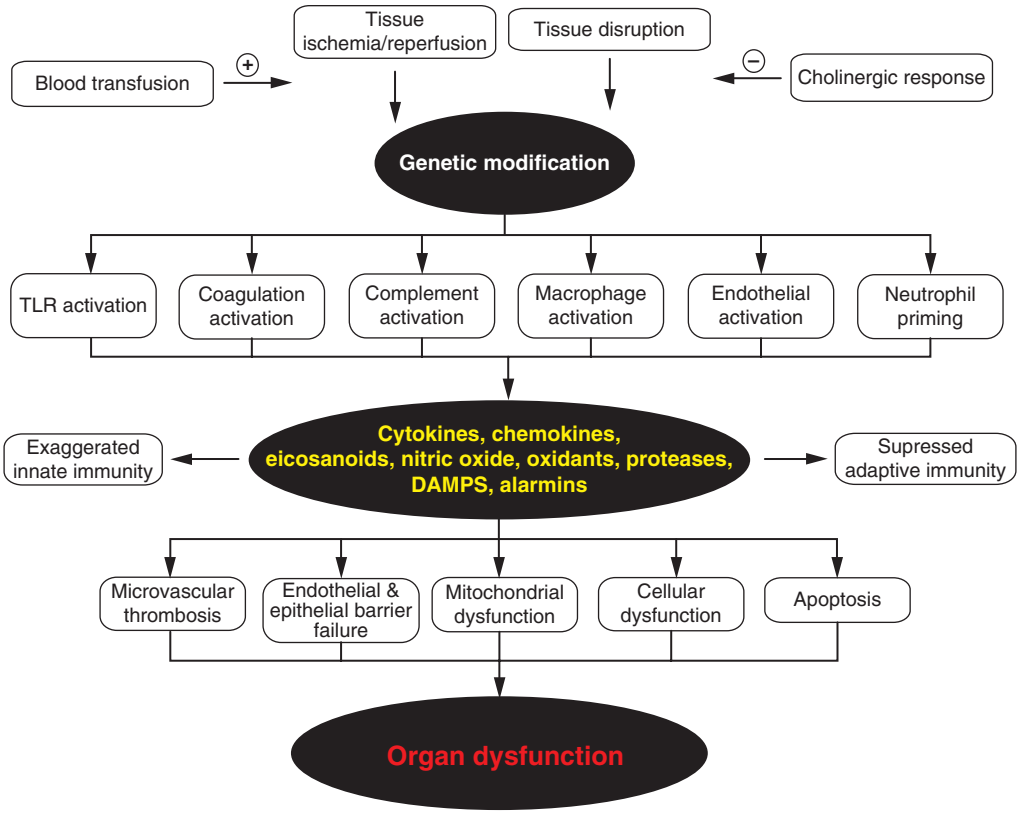

FIGURE 62-3 Simplified schematic representing the current understanding of the pathophysiologic reactions to major trauma. These reactions lead to secondary remote organ dysfunction, as outlined in the text. DAMPs, damage-associated molecular patterns; TLR, Toll-like receptor. (From Stahel PF, Flierl MA, Moore EE. "Metabolic staging" after major trauma: a guide for clinical decision making? *Scand J Trauma Resusc Emerg Med.* 2010;18:34. Reproduced with permission.)

these patients who need to be assessed for specific nutritional formulations with higher protein intake.

There are some unique trauma patient populations with unique nutritional needs and requirements. These include traumatic brain injury patients, burn patients, and others. Please see the Special Patient Populations section later for details and recommendations. This section also has details on how to perform a nutritional assessment to identify patients with baseline malnutrition.

WHEN TO IMPLEMENT NUTRITIONAL SUPPORT?

Prior to starting any nutritional support, the end points of traumatic shock resuscitation should be achieved and the patient stabilized.[32,35,83,91] Once achieved, *early* (within 24–48 hours of injury) enteral nutrition should be started as soon as possible.[32,35] While there are different definitions of early (within 12 hours,[92] within 24 hours,[93] or within 48 hours[32,94] of injury or admission to intensive care unit [ICU]), in general, within 48 hours or earlier is accepted. Early enteral nutrition is so fundamental to good patient management that it should be the final component of traumatic resuscitation.[94]

The severely injured polytrauma patient who does not receive nutritional support in the first few days after the injury can develop worsening energy and protein deficits, which contribute to risk of complications.[83-85,95] Multiple prospective randomized controlled trials have shown that early nutrition can decrease infection rates, hospital lengths of stay, and cost, and improve outcomes.[95-98] Trauma patients with an open abdomen without an associated bowel injury who were started on enteral nutrition once resuscitation was completed had decreased complications and improved survival[99] (Fig. 62-4).

WHAT SHOULD BE USED AS NUTRITIONAL SUPPORT?

Choosing the ideal nutrient formulations for injured and/or critically ill patients is increasingly complex. There are many options for both enteral nutrition (EN) and parenteral nutrition (PN), from the basics to specialized formulations. Please see the sections on nutrition intervention for details and specifics.

WHERE IN THE BODY TO FEED: GI TRACT OR PARENTERALLY?

Patients who have an injury, disability, or critical illness that will preclude their ability to initiate oral feeding (ie, feed themselves) beyond hospital day 5 should be started on enteral tube feedings as soon as safely possible, once resuscitated and stable.[35,36,83,99]

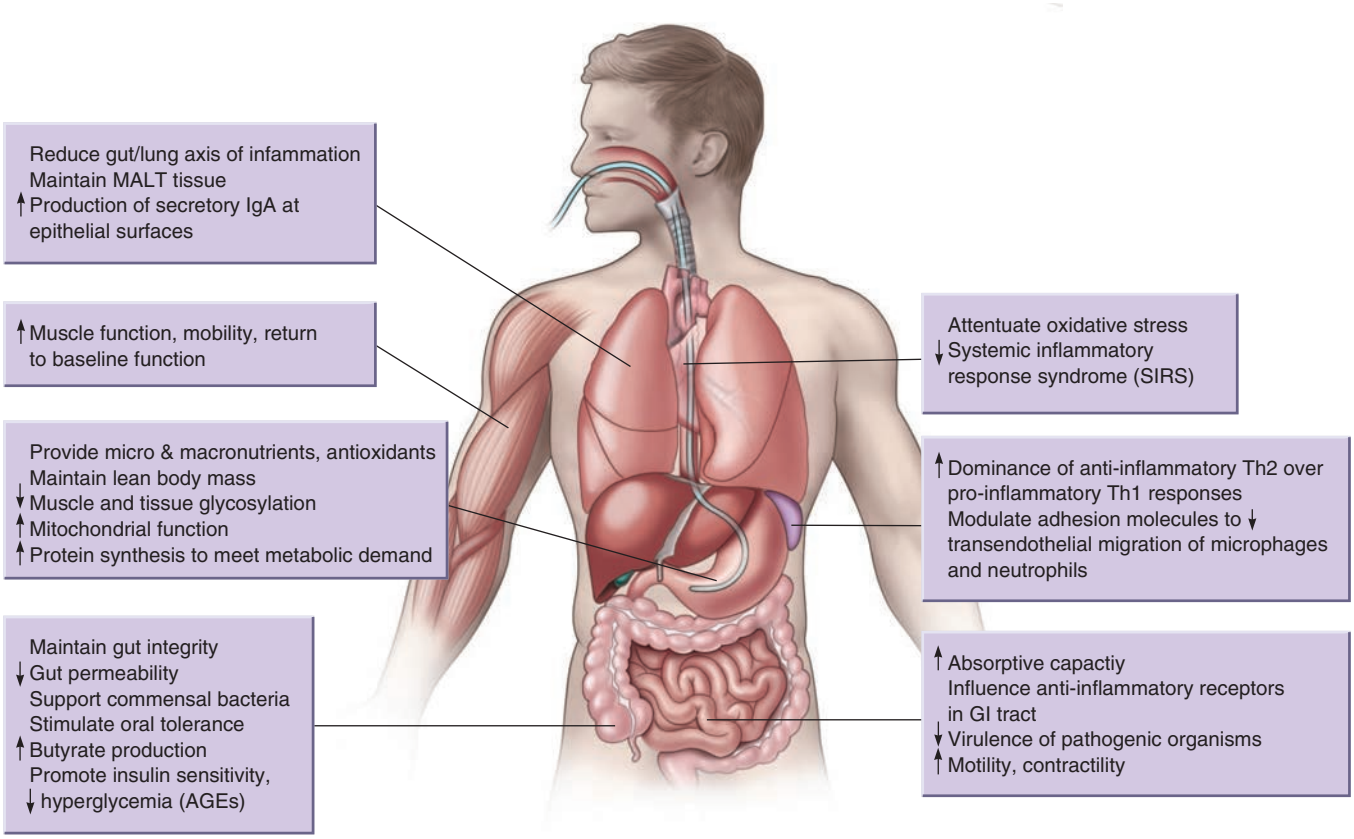

FIGURE 62-4 Nutritional and nonnutritional benefits of early enteral nutrition. AGE, advanced glycolytic end products; GI, gastrointestinal; MALT, mucosal-associated lymphoid tissue. (Reproduced with permission from McClave SA, Martindale RG, Rice TW, Heyland DK. Feeding the critically ill patient. *Crit Care Med*. 2014;42(12):2600-2610.)

Early EN via either gastric feeds or postpyloric feeds is acceptable.[32,34,35,92,97,100-103] In the largest multicenter randomized controlled trial to compare gastric versus small bowel EN in critically ill patients, there was no difference in clinical outcomes between groups, including length of stay, mortality, nutrient delivery, and incidence of pneumonia.[103a] For this reason, our practice is to start with gastric feeds, if possible. This can be achieved through either continuous or intermittent tube feeds, as discussed later in the chapter. The benefits of the enteral route as compared to the parenteral route include fewer infections (eg, pneumonia, catheter-related bloodstream infection), decreased possibility of bacterial translocation, prevention of gut mucosal atrophy, avoidance of complications of PN, avoidance of complications of intravenous access for PN, and reduced cost.[32,35] There is no morality benefit to EN over PN.

Although the enteral route is preferred, there are situations when PN is indicated. If enteral feeds are contraindicated, have failed, or are not expected to be started for 3 days, then *exclusive* PN should be considered.[33,36,104] Note that the 3-day cutoff for initiating exclusive PN is now more widely accepted, and has become a practice when EN is not able to be started. For many years, the European Society for Clinical Nutrition and Metabolism (ESPEN) has used a cutoff of 3 days.[33] The American Society for Parenteral and Enteral Nutrition (ASPEN) and the Society of Critical Care Medicine (SCCM) guidelines, however, use a cutoff of 7 days to start PN in previously healthy patients (prior to their critical illness and injury)—though the evidence to support this stance is reported as "very low."[35] The most recent SCCM/ASPEN guideline does recommend *early* exclusive PN (meaning as soon as possible after admission to the ICU) in patients with baseline protein-calorie malnutrition and when EN is not feasible.[35]

If enteral feeds will be inadequate for the first 3 to 10 days (inadequate defined as achieving only 25% of 24-hour kilocalorie goal), then one option is use of *complementary PN*, meaning the use of PN in addition to EN (complementary PN is also referred to as supplemental PN, dual-modality therapy, or top-off total PN). This topic is somewhat controversial. In 2011, a large, prospective randomized controlled trial found no benefit to early (day 3 of ICU stay) versus late (day 8) initiation of complementary PN.[105] For this reason, ASPEN/SCCM guidelines recommend the use of supplemental PN starting on surgical ICU day 10 only if the patient is unable to meet greater than 60% of their nutritional needs by enteral route alone.[35]

The need for supplementary PN must be interpreted with data demonstrating that trophic EN feeds (which by definition have a negative nitrogen balance) for 7 days in critically ill nontrauma patients have outcomes equivalent to goal tube feeds over the same time period.[106-108] Therefore, if a patient can tolerate even trickle tube feeds, achieving nitrogen balance with complementary PN is not needed. While somewhat controversial (ASPEN/SCCM cite data demonstrating higher infectious morbidity and rates of ARDS when using complementary PN in patients already receiving 1000 kcal/d of EN,[109] and ESPEN cite data demonstrating a strong correlation between negative nitrogen balance and morbidity and mortality in critically ill patients[110,111]), at present, there is not sufficient evidence to make a best recommendation on complementary PN[36,112] beyond the fact that it should not be started before hospital day 10.[105]

Enteral or parenteral modes of nutritional therapy are different means to the same end. In this manner, attention must be paid to the patient's tolerance of one route and, if necessary, switching to the other route.

HOW TO DELIVER NUTRITIONAL SUPPORT?

Please see section on nutritional intervention for details and specifics on the delivery and administration of EN, including access choices, monitoring, and complications.

HOW MUCH ENERGY AND MACRONUTRIENTS ARE REQUIRED?

Energy. Most critically ill trauma patients have in common an increased metabolic rate. The hypermetabolic response coupled with increased protein catabolism create an amplified energy requirement in the severely injured patient. The increase in energy requirements may range anywhere between 30% and 70% above normal, although even the most experienced clinicians are unable to predict the extent to which trauma or injury will amplify an individual's energy requirements.[60]

Based on studies calculating nitrogen losses, needs are increased approximately 30% in major injury, 50% in sepsis, and 75% to 100% with severe burns[60] (see Fig. 62-2). Translating these increased metabolic rates into higher kilocalorie requirements is a bit more nuanced. This is because increasing evidence suggests that critically ill patients have lower energy requirements than expected. For example, while injury and infection increase REE, in most cases, the increase is modest and largely offset by immobility.[10]

Therefore, the old strategy of meeting or exceeding energy requirements after a major trauma compounds the metabolic alterations of the stress response and worsens outcomes.[113] A more modern nutritional doctrine is that overnutrition has detrimental complications.[32-36,114] For this reason, the use of injury factors for the HBE to calculate EER is not recommended (an old dogma was to multiply the 24-hour kilocalorie HBE BEE by factors for postoperative and/or injured patients, such as 1.20 if elective surgery, 1.35 if trauma, 1.60 if major sepsis, or 2.1 if major burn).[115]

A nutritional support goal of 20 to 25 kcal/kg IBW/d is enough to be beneficial during the acute and initial stress response in the critically ill trauma patient.[32,113] This is in keeping with guidelines that highlight reaching at least 50% of total daily goal kilocalories over the first week of the hospitalization, if goal nutrition cannot be achieved.[35] During the anabolic recovery phase (ie, anabolic flow period; convalescence), the aim should be to provide slightly more energy, from 25 to 30 kcal/kg IBW/d.[32] Other guidelines recommend a daily kilocalorie increase of 40% above BEE, which translates into a nutritional support goal of 25 to 30 kcal/kg IBW/d.[32,33,35,36]

The ASPEN/SCCM guideline endorses both predictive equations as well as indirect calorimetry for calculating target energy goals, as does ESPEN. If these goal values cannot be achieved, trophic enteral feeds at lower rates (ranging from 10–30 mL/h) have huge benefits over starvation and have outcomes that are as good as EN at goal.[106-108]

Finally, the question arises regarding which body weight one should use in nutritional calculations in polytrauma and critically ill patients: IBW, actual body weight, LBW, or ABW. This was briefly mentioned in the first section of the chapter. The ESPEN guidelines strongly recommend using IBW for all weight-based calculations ("it is therefore wise to consider ideal body weight when calculating energy requirements").[32,33,91,116] Using IBW avoids the pitfall of overfeeding patients. The ASPEN/SCCM guidelines use the actual body weight for patients with a body mass index (BMI) less than 30, and both the actual body weight and the IBW for those with a BMI of 30 or greater (see Special Patient Populations section later for nutritional requirements in obese patients). Note that in patients with an actual body weight BMI of less than 30, that approximates to the IBW, and thus IBW can be used without dire consequences.

Protein Needs After Trauma. Protein is thought to be the most important macronutrient for wound healing, immune function, and preventing loss of lean body mass in the injured and critically ill patient.[35] The hypermetabolic, hypercatabolic state of posttraumatic stress induces a profound breakdown and redistribution of body protein (see Fig. 62-1); protein needs to be replenished and levels maintained.[60] If such needs are not appropriately addressed, it can lead to ongoing erosion of lean body mass and negative nitrogen balances.

In the midst of a severe inflammatory stress response, once-normal synthetic reactions may require substantially different patterns of amino acid usage: proline for collagen synthesis, aromatic amino acids for synthesis of antibodies and acute phase proteins, and glutamine for rapidly dividing cells.[10] In such conditions, amino acids that are not usually essential can become conditionally essential due to limited synthetic capacity.[10] The amount and composition of protein required to maintain nitrogen balance in postinjury critically ill polytrauma patients may differ substantially from that in healthy subjects. For example, if loss of LBW is severe, adults can have a requirement for essential amino acids resembling that of a growing child due to the needs for tissue rebuilding.[10]

During the acute stress response, a nutritional support goal ranging from 1.2 to 2.0 g protein/kg IBW/d will decrease the

loss of LBW and prevent negative nitrogen balances.[32,33,113] The ESPEN guidelines specifically recommend 1.3 to 1.5 g/kg IBW/d in critically ill trauma patients.[32,33] The latest ASPEN/SCCM guidelines recommend "an ongoing evaluation of the adequacy of protein provision be performed."[35] This amounts to protein of 1.5 g/kg body weight/d if BMI is less than 30; 2.0 to 2.5 g/kg IBW/d if BMI is 30 to 40; and 2.5 g/kg IBW/d if BMI is 40 or greater.[35]

Note that some guidelines use a nitrogen-based recommendation, and the conversion is that 1 g of nitrogen is equal to 6.25 g of protein. Some critically injured polytrauma patients will demonstrate such significant and rapid muscle losses that they have protein requirements three times the normal level, meaning closer to 2.5 g/kg/d.[63] The desire to simply add supratherapeutic levels of protein must, however, be buffered with the fact that it will not prevent the hypercatabolic state in critically ill trauma patients.[117]

The distribution of nutrition kilocalories among the macronutrients during the stress response is not firmly established. It should be adjusted to particular circumstances and for individual patients, keeping in mind the AMDR (see Table 62-2).

Standard recommendations call for calculation of protein kilocalories first based on a protein requirement of 1.3 g/kg IBW/d.[32,33] Then calculate the carbohydrate and fat energy distribution (see later). When calculating energy provision for artificial nutrition support by either EN or PN, do not consider energy provided as protein as separate from energy given as nonprotein calories.

Carbohydrate Needs After Trauma. Carbohydrates should constitute 50% to 70% of nonprotein calories.[36] See specifics on EN and PN later in this chapter.

Fat Needs After Trauma. Fat should constitute 20% to 30% of nonprotein calories.[36] See specifics on EN and PN later in this chapter.

Water Needs After Trauma. The impacts of dehydration are amplified in the critically ill and postinjury patient.[53] Dehydrated trauma patients (those who have lost 7%–10% of body weight as water) are more susceptible to fever, have increased cardiovascular strain and reduced tissue perfusion, and are at increased risk of death.[53] Euvolemia should be maintained as discussed in the healthy adult section.

NUTRITIONAL INTERVENTION: GENERAL

Choosing and constructing the ideal nutritional therapy for injured and critically ill patients is increasingly complex. Expanded knowledge of the pathophysiology of the posttraumatic stress response as well as critical illness and the growing field of nutritional sciences have made the possibility of disease-targeted nutrition a near reality. Enteral and parenteral formulas today are not only expected to provide balanced nutrients to meet energy requirements, but are also supposed to modulate the immune system, enhance neuroendocrine function, mitigate the hypermetabolic response to injury, and offset postinjury catabolism.

In metabolically stressed polytrauma patients, the anabolism-catabolism balance becomes severely weighted toward catabolism, leading to rapid depletion of body tissue stores and critical protein elements, such as immunoglobulins. This is characteristic of protein-calorie malnutrition. The primary goal of nutritional intervention is therefore to minimize the net negative energy and protein balances and their negative downstream consequences, while appreciating that lean tissue loss is unavoidable in severe trauma patients even with aggressive nutritional therapy. The secondary goal, which is increasingly controversial, is to target immune and neuroendocrine function via pharmaconutrition.

Before reading further, please ensure you have read the last section entitled Nutrient Utilization and Needs After Trauma and Injury: Seven Key Questions, which addresses the why, who, when, what, where, how, and how much of trauma patient nutritional support. For additional resources, multiple medical, surgical, trauma, and critical care organizations have developed and published nutrition guidelines which are of benefit, as are their websites:

- SCCM and ASPEN[35]
- ESPEN[32,33,91,116]
- Canadian Critical Care Society[93]
- Eastern Association for the Surgery of Trauma[87]
- Spanish Society of Intensive Care Medicine and Coronary Units–Spanish Society of Parenteral and Enteral Nutrition[36]
- German Association for Nutritional Medicine[118,119]

If nutritional requirements are properly attended to, the polytrauma patient should get the right type of nutritional substrate, in the right amounts, at the right time. This is easier said than done, however, as nutrition and nutritional science is a modern, complex, and ever-changing field of medicine. Nutrition, after all, is not intuitive,[120] and nutritional assessments and interventions demand that one's clinical knowledge is up to date.

NUTRITIONAL INTERVENTION: ENTERAL NUTRITION

Meeting the increased energy requirement with early EN is essential to the adequate care of the injured patient.[66] Prospective, randomized controlled trials have clearly proven the positive effect of early EN in the polytrauma patient, with decreased posttraumatic infection rates, a shorter hospital length of stay, and improved morbidity and mortality (Fig. 62-5).[95-98]

Patient Selection

All patients should ideally receive nutritional support within 24 to 48 hours of injury. Before starting enteral feeds, a thorough history (including past medical and surgical history)

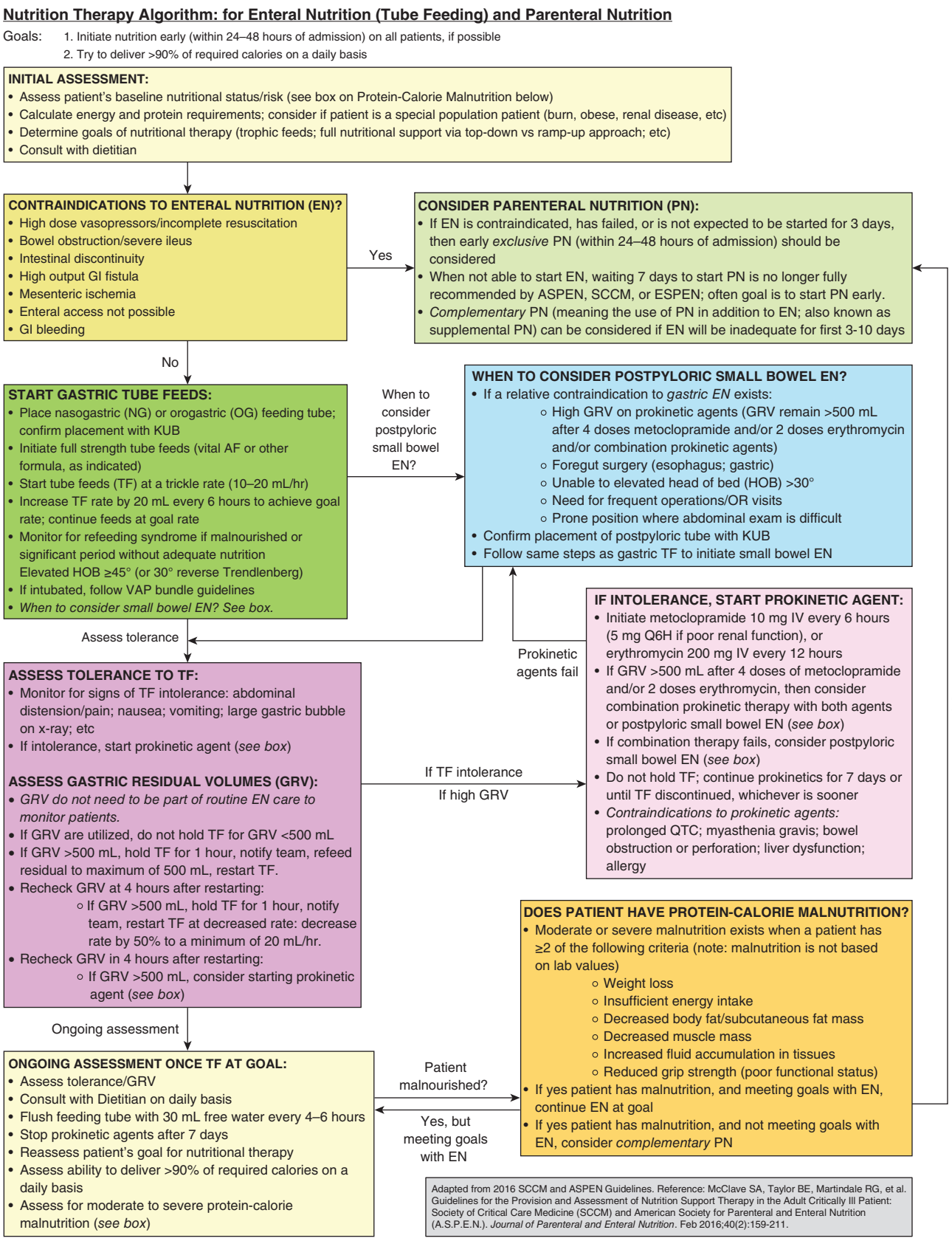

FIGURE 62-5 Clinical algorithm for initiating nutritional therapy.

and physical exam should be completed. Conditions such as heart, liver, and/or kidney disease as well as alcoholism and malnutrition will impact the approach to enteral feeding, as will key operative history such as short gut or gastric bypass. Please see the later Special Patient Populations section for more specifics; particularly important is the section titled Patients with Baseline Malnutrition or High Nutritional Risk.

Meeting Nutritional Requirements by Enteral Route

How to meet the macronutrient and micronutrient requirements of both healthy and critically ill, hypermetabolic trauma patients was discussed earlier in the chapter.

Enteral Formulas and Choices

Highly sophisticated enteral nutritional formulas were developed long ago as part of NASA's space program.[121] Back then, the goal was to develop a diet that would leave no fecal residue. Such enteral formulas eventually became known as elemental diets, and although astronauts rejected them as tasteless, medicine has embraced them as revolutionary. And it is clear why: they can be given in defined concentrations, they contain all macro- and micronutrients, and some require very little enzymatic activity on the part of the GI tract. Enteral nutritional formulas (ie, artificial nutrition, chemically defined diets) today cover a full spectrum of products, many claiming pharmacologic effects in addition to standard nutrient delivery; hundreds are available.

Choosing an appropriate enteral formula is mostly based on meeting the energy (kilocalorie) and protein requirements of the patient (these requirements are discussed at length earlier in the chapter). Other considerations include baseline nutritional status, electrolyte balance, digestive and absorptive capacity, disease and inflammatory state, ongoing or completed resuscitation, and comorbidities including cardiac, pulmonary, hepatic, and renal function.

Enteral formulas are classified in three ways: standard, predigested, or specialized. There are many choices within each category. In general, if patients have normal GI function, they can get standard formulas. If their GI function is compromised, then one should consider the predigested formulas. If they have significant baseline comorbidities, then a specialized formula can be considered; the specialized formulas are for both normal and abnormal GI function.

STANDARD FORMULAS

Standard formulas (ie, polymeric formulas) have intact protein with balanced amounts of macronutrients. Their nutrient compositions are meant to match what is recommended in healthy individuals (within the AMDR; see Table 62-2). They come in various kilocalorie concentrations (called kilocaloric density, ranging from 1.0–2.0 kcal/mL), which translate into higher or lower water content formulas. They are made with and without fiber supplementation; the fiber can be soluble and/or insoluble. The sources of carbohydrates, proteins, and fats are variable. Standard formulas require complete digestive capacity, as they have proteins in their original, natural form. These formulas are often much cheaper than others.

For stressed polytrauma patients with systemic inflammation, high-protein (ie, high-nitrogen) formulas are necessary, meaning they usually have a nonprotein calorie–to–nitrogen ratio of 70:1 to 100:1 (much lower than the usual 150:1 ratio).[35] Additionally, two sources of energy are believed to benefit high-stress situations: medium-chain triglycerides (MCT) and branched-chain amino acids (BCAA).

PREDIGESTED FORMULAS

Predigested formulas are designed for patients with decreased absorptive capacity who need predigested nutrients, thus requiring less enzymatic activity by the GI tract and leaving no residue behind. The protein source in these formulas has already been broken down into either free amino acids (in the case of elemental formulas) or short peptides (in the case of peptide-based formulas, with either dipeptides or tripeptides). These formulas are also called hydrolyzed or partially hydrolyzed formulas, as well as elemental or semi-elemental formulas.

SPECIALIZED FORMULAS

Specialized formulas are designed for a variety of clinical situations and conditions. These formulas can be generally categorized as either disease-specific formulas or immune-modulating formulas (IMFs), although there is overlap. The latest fad in specialized EN is "natural formulas."

SPECIALIZED DISEASE-SPECIFIC FORMULAS

The disease-specific formulas are specially designed for renal disease, liver disease, diabetes/hyperglycemia, pulmonary disease (eg, chronic obstructive pulmonary disease, acute respiratory distress syndrome), and hypoallergenic patients. They can be useful for the stressed polytrauma patient with significant comorbidities.

SPECIALIZED IMMUNE-MODULATING FORMULAS

Pharmaconutrition is the practice of using both nutrition and nutritional supplements as pharmaceuticals, with the intention of improving an outcome or curing a disease. An arm of pharmaconutrition is *immunonutrition*: the practice of using the diet to affect both the nutritional and the immune status of a metabolically stressed patient.[60]

The role of IMFs (ie, immune-enhanced nutrition) was, in the recent past, considered routine clinical care for critically ill trauma and surgery patients.[32,91] Today, however, it is more controversial,[63] as the data on the true benefits of immunonutrition are changing rapidly. Part of the difficulty in interpreting this literature is that supplement dosing, timing, and route of administration are not standardized, the supplements can be given alone or as part of a combination

therapy, and the patient populations are heterogeneous.[122] In short, at present, there is true clinical equipoise with regard to immune-enhanced nutrition.

The latest 2016 guideline from ASPEN/SCCM[35] supports the use of IMFs in trauma patients with a traumatic brain injury as well as in perioperative surgical ICU patients, noting, however, that the evidence for the use of IMFs is very low. In several meta-analyses, adding immunomodulating nutrients to enteral feeding formulas was associated with reduced infectious complications and improved recovery compared with standard EN. IMFs have therefore gained widespread use in severely injured polytrauma patients. Supplements that have been researched include arginine,[123-125] glutamine,[76,126] omega-3 polyunsaturated fatty acids,[125] nucleic acids,[127] and micronutrient antioxidants (eg, selenium),[76] among others.

The most recent ESPEN guideline, on the other hand, has concluded there is no general indication for administering immune-modulating nutrients to critically ill patients.[32] Two recent studies support the ESPEN stance: both the 2013 REDOX study of glutamine and antioxidants[76] and the 2014 MetaPlus Trial of high-protein IMF[128] show an increased risk of mortality with IMFs compared to standard high-protein formulas. Although in theory glutamine, arginine, nucleic acids, omega-3 polyunsaturated fatty acids, selenium, and antioxidants should help as immunonutrition, in practice, their use may be harmful.

"NATURAL FORMULAS"

EN is big business. The global enteral feeding formulas market size was valued at $4.39 billion in 2016 alone[129] and is only expected to get bigger in time. Out of this large corporate market has grown a small, boutique industry for "natural" and "real food" enteral formulas. These new formula companies offer everything from organic to plant-based to vegan EN options. Examples include "Liquid Hope," "Kate Farms," and "Orgain."

Enteral Access

Multiple access options exist for the administration of EN. They can generally be divided into either temporary (ie, short term) or long-term feeding access devices.

Temporary access devices are one of two types: prepyloric or postpyloric. Prepyloric options include simple nasogastric tubes or smaller-diameter feeding tubes placed into the stomach. Postpyloric tubes are also inserted through a nostril, although they are placed past the pylorus and into the duodenum or jejunum.

Long-term access devices are also one of two types: prepyloric or postpyloric. Prepyloric tubes are gastrostomy tubes; they mainly differ by their physical characteristics and the method by which they are inserted (percutaneously, laparoscopically, or open) (see Atlas Figure 68). Postpyloric tubes can originate in the stomach (combination gastric-jejunostomy tube) or in the jejunum (jejunostomy tubes).

In general, there are few contraindications to feeding the stomach. There are no good data to advocate for postpyloric feedings as the delivery option of choice in polytrauma patients. Although it makes sense that postpyloric feeds would decrease the risk of aspiration and pneumonia, this has not been proven[130,131] (please see later in chapter for in-depth discussion on gastric residual volumes). Do not delay the initiation for EN because of a lack of postpyloric access.

The major indication for long-term access (gastrostomy or jejunostomy tubes) is the anticipated inability of a patient to tolerate oral feedings beyond 1 month after the time of injury.

Mechanically ventilated critically ill patients should have the head of the bed elevated to at least 30° when receiving EN[34] to decrease risk of aspiration and pneumonia.

Delivery and Administration of Enteral Nutrition

The delivery of enteral nutritional is achieved by either continuous or intermittent tube feeds. There are no data to support one method of delivery as superior to the other.

Goal-directed continuous tube feeds (with a target goal of full kilocalorie nutritional support) are initiated by one of two strategies: the top-down approach (ie, aggressive approach) or the ramp-up approach.[132] The *top-down aggressive approach* is akin to an hourly volume-based approach: immediately start the rate of tube feeding right on the maximal hourly goal and deescalate if needed. The *ramp-up approach* is more traditional and involves initiation of tube feeds at a lower rate (often 15–25 mL/h) and increasing to the goal rate over a period of time.[83]

More recently, a third strategy has been espoused: trophic feeds. *Trophic feeds* do not target full kilocalorie support, but rather a lower level of both kilocalories and protein to provide the GI tract the benefits of nutritional support (improved gut integrity, better contractility, increased brush border enzymes, restoration of the commensal bacteria[100]) without putting the patient at risk for aspiration, GI intolerance, or other complications of higher volume feeds. With trophic feeds, the rate is set low (10–20 mL/h, representing only 15%–25% of goal kilocalories) and kept low for days; it can be thought of as a very delayed ramp-up to goal.

Studies comparing these methodologies of continuous tube feeds have failed to demonstrate improved outcomes with increased amounts of enteral kilocalories via the aggressive approach compared to underfeeding with a trophic approach.[106-108] Trophic feeds, however, were not shown to be superior to a regimen of full target feeding. Additionally, these methods have not been studied in trauma patients, meaning patients with a hypermetabolic response getting trophic feeds may have ongoing erosion of lean muscle mass, resulting in impaired recovery and worse clinical outcomes.[100] As such, iatrogenic underfeeding with trophic feeds appears to be safe in the very short term, although timely advancement to goal remains the aim of early nutritional therapy in polytrauma patients.

Trophic feeds by definition mean that the patient is getting less than goal energy requirements and less than goal protein requirements (typically 15%–25% of goal). A fourth strategy is *permissive underfeeding*, when the patient gets goal rates of protein requirements but restriction of nonprotein kilocalories. Recent studies have shown promise with permissive underfeeding in comparison to full feeding in critically ill patients,[133] although earlier studies lack both internal and external validity.[100]

From a practical standpoint, there are key considerations to choosing a strategy. One is that hypertonic enteral formulas will not be well tolerated if started at full volume and full concentration. A gradual buildup is necessary (over days) for the GI tract to accept this unnatural source of energy without severe diarrhea (and thus risk of dehydration). Another is that the jejunal route may require more patience before full strength is tolerated, as the jejunum can be sensitive to tonicity, fat concentrations, and protein sources. Additionally, gastric feeds can be higher tonicity, with higher administration rates, and given as bolus feeds, thus giving the provider more options for nutritional support.

It is not natural for the GI tract to be exposed to food/nutrition continuously for 24 hours a day. For this reason, there is a school of thought that interrupting continuous tube feeds for 4 to 6 hours a day makes them better tolerated by patients and is healthier for the GI tract. In this manner, the full kilocalorie feeding regimen would be given continuously for only 18 to 20 hours per day. There are at present no data to support this practice.

Intermittent infusions (ie, bolus feeds) are delivered at intervals (every 4–6 hours), with a large volume given at each interval (often 200–500 mL). Bolus feeds are well tolerated with a prepyloric feeding tube, especially in the outpatient setting. Most inpatients are administered tube feeds continuously. Jejunostomy tube feeds are not well tolerated as intermittent feeds (due to abdominal pain, distention, and diarrhea) and therefore must be given continuously.

PROKINETIC AGENTS

Prokinetic drugs, such as erythromycin and metoclopramide, can be used to promote motility to achieve effective administration of EN.[34-36,134] These agents help in emptying the stomach and in tolerating EN but have not been shown to improve or change outcomes.[35]

GASTRIC RESIDUALS

The practice of regularly holding or interrupting prepyloric EN for elevated gastric residual volumes (GRV) in mechanically ventilated patients has long been used. The rationale is that if, during the initiation or maintenance of enteral feeding, a threshold GRV was reached (anywhere from 200–500 mL has been used), this was proof of gastroparesis, delayed gastric emptying, or GI dysfunction, and warranted stopping the EN. Recent randomized controlled trials have questioned this practice.[130,131] However, checking GRV is by no means a dead practice, especially in a surgical/trauma ICU with patients at high risk for GI dysfunction.[132] The latest ASPEN/SCCM guideline[35] strongly recommends against checking GRV as part of routine care to monitor EN. They instead recommend monitoring for tolerance ("by physical examination, passage of flatus and stool, radiologic evaluations, and absence of patient complaints such as pain or abdominal distention") versus intolerance ("defined by vomiting, abdominal distention, complaints of discomfort, diarrhea, reduced passage of flatus and stool, or abnormal abdominal radiographs").[35] However, in the surgical ICUs that do still use GRV, the guideline recommends a GRV of greater than 500 mL to hold EN.

MONITORING

Once tube feeds are initiated, patients need to be closely monitored. Monitoring for fluid balance and losses, metabolic and inflammatory status, positioning (of both patient and tube), and early detection of complications is required. Blood glucose should be checked at least every 6 hours. Electrolytes should be checked daily, especially with increased GI tract losses. Trace element and vitamin levels should be checked only as clinical concern warrants.

During acute illness, the short-term goals of feeding are to restore and maintain function while limiting further loss of lean tissue. During the weeks of convalescence, the aim is to restore lean mass as well as function Nitrogen balance may be indicative of loss or gain of body protein but is not a goal in itself. Nonetheless, in the absence of specific tests for adequacy of protein intake, some measure of nitrogen balance is useful in various clinical settings, since a prolonged state of negative nitrogen balance is not compatible with life.[10]

COMPLICATIONS

Although in general EN is extremely safe, there are potential complications. The complications fall into one of two categories: access complications (either during insertion or mechanical) or diet-related complications.

Insertion-Related Complications. Feeding tubes of various types can be placed at the bedside, by interventional radiology, by gastroenterologists, and surgically. Each of these methodologies has inherent complications. These include bleeding, infection, nasal damage, intracranial insertion, pulmonary insertion, and perforation. While real and significant, we will not highlight the multiple potential surgical complications of gaining feeding access (these operations include percutaneous endoscopic gastrostomy tubes, laparoscopic and open gastrostomy tubes, and placement of jejunostomy tubes).

Mechanical Complications. Various mechanical complications relating to the tube itself can occur. The lumen of tube can get obstructed. This is often the result of poor

maintenance or lack of tube hygiene. Obstruction can be prevented with free water flushing protocols and ensuring no crushed medications are administered through the tube. Tubes can also be accidentally dislodged or removed, and they can migrate. Over time, tubes can cause discomfort, erosions, fistulae, and strictures.

Diet-Related Complications. Diet-related complications (ie, formula related) are specifically associated with the enteral formula itself (including a host of metabolic issues) or to the unnatural process of being fed via a tube. These include aspiration, diarrhea, GI intolerance, electrolyte disturbances, micronutrient deficiencies, hyperglycemia, hypertriglyceridemia, refeeding syndrome, malabsorption, hypertonic dehydration, hyperosmolar nonketotic coma, liver dysfunction, renal dysfunction, overfeeding, infection, bacterial translocation, pneumatosis intestinalis and necrosis, fluid overload, and hypoprothrombinemia (from vitamin K deficiency), among others.

NUTRITIONAL INTERVENTION: PARENTERAL NUTRITION

In 1968, Wilmore and Dudrick[135] proved—first in beagles and later in a newborn girl with intestinal atresia[135]—that patients could grow and meet their nutritional needs by receiving exclusive intravenous nutrition. Their discovery of what is now known as PN (ie, total PN [TPN]; formerly referred to as hyperalimentation or hyperal) has greatly expanded the ability of physicians to treat malnutrition in patients who were otherwise not candidates for enteral feeds. In this section, we discuss when to use PN, who will benefit from PN, how to order and initiate the use of PN to meet the needs if the patient, and how to monitor patients on PN to ensure few complications with good nutritional support.

Patient Selection

All patients should ideally receive early nutritional support within 24 to 48 hours of injury. As discussed in detail previously in this chapter, PN should be prescribed if:

- Enteral feeds are contraindicated (usually due to nonfunctional GI tract from dysmotility, discontinuity, ischemia, fistula, or obstruction)
- Enteral feeds have failed (either because of nonfunctional GI tract or because EN is not adequately or consistently achieving nutritional goals)
- Enteral feeds are not expected to be started for 3 days in the postinjury, inflammatory stress response patient or patient at high nutritional risk

Contraindications to starting PN, even if one of the above requirements is met, are ongoing resuscitation, shock, and/or high-dose vasopressors. Nutritional therapy is vitally important, but it is never an emergency.

Meeting Nutritional Requirements by Parenteral Route

How to meet the macronutrient and micronutrient requirements of the polytrauma patient with PN is discussed in the following sections.

CENTRAL VENOUS ACCESS

Central venous access, with a dedicated line not to be used for anything else, is fundamental to PN administration. Compulsive attention to sterile technique for both placement and maintenance will prevent complications. Parenteral nutritional preparations have very high tonicity (often >1000 mmol/L, and usually close to 2000 mmol/L) and are better tolerated centrally.

PARENTERAL NUTRITION PRESCRIPTION

There are five steps to writing orders for PN. Figure 62-6 provides an example of PN calculations.

Step 1: Total Kilocalories and Fluid Volume Over 24 Hours. Total energy requirements for the critically ill polytrauma patient were outlined in the last section, specifically the 24-hour kilocalorie requirement and the distribution between the macronutrients. The one major difference between EN and PN in terms of nutritional support is that a strategy of "permissive underfeeding" should be pursued at first with PN; to do this, 80% of total daily nonprotein energy requirement should be given at first.[35]

Total fluid volume of PN preparations will vary depending on the patient's clinical condition, with patients in the immediate postresuscitation hypercatabolic phase needing more. In general, patients need a minimum of 30 to 50 mL fluid/kg of actual body weight to maintain hydration; as discussed earlier in the chapter, in adult men, this is 3.7 L, and in adult women, it is 2.7 L. Additionally, when initially starting PN delivery, volumes may be lower over the first few days (please see later Administration and Delivery section) (Fig. 62-6).

Step 2: Amino Acids and Protein. The next step is to calculate protein requirements and their contribution to total energy requirements. As noted earlier, 1.3 g/kg IBW/d is recommended in critically ill trauma patients,[32,33] and no more than 2.0 g protein/kg IBW/d.[32,33,113] Simply adding huge levels of protein will not prevent the hypercatabolic state in critically ill trauma patients.[117]

With PN, protein is delivered in the form of a balanced amino acid mixture of both essential and nonessential amino acids. The contributions of these amino acids, including BCAA, is variable. In septic patients, for example, TPN fortified with BCAA at high concentrations (0.5 g/kg/d or higher; up to 45% of total protein) has been shown to lower morbidity and mortality when compared to standard TPN.[136]

Supplementation of PN solutions with immune-enhancing amino acids is controversial and in flux. Although at one point it was considered standard of care, more recent data

Calculating Parenteral Nutrition Formulations

(Assuming normal renal, hepatic, and cardiac function)

STEP 1: TOTAL KILOCALORIES AND FLUID VOLUME OVER 24 HOURS

Determine dosing weight:

a. If BMI 18.5–25, using actual weight
b. If BMI 25.1–29.9, use Hamwi equation
    1. Male: 48 kg (for first 5 feet) + (2.72 kg × each inch over 5 feet)
    2. Female: 45.5 kg (for first 5 feet) + (2.27 kg × each inch over 5 feet)
c. If BMI <18.5, use Hamwi equation –10%
d. If BMI 30–39.9, use Hamwi equation +10%
    - *Example:* 60 kg patient with BMI of 23, dosing weight = 60 kg

Determine energy needs:

a. If BMI ≤39.9, use 30 kcal/kg dosing weight
b. If BMI 40–49.9, use 11–14 kcal/kg actual weight
c. If BMI ≥50, use 25 kcal/kg dosing weight
    - *Example:* 60 kg dosing weight × 30 kcal/kg = 1800 kcal/day

Determine PN volume:

a. 25–35 mL/kg dosing weight or 1 mL:1 kcal fed
b. Divide total mL/day by 24 hours to determine hourly rate
c. Divide total mL/day by 1000 mL to determine PN volume in L
    - *Example:* 1800 kcal/day = 1800 mL/day ÷ 24 h/day = 75 mL/h x 24 h/day = 1.8 L/day

STEP 2: AMINO ACIDS AND PROTEIN

Determine protein/nitrogen requirements:

a. If BMI ≤39.9, use 2 g/kg dosing weight
b. If BMI 40–49.9, use 2–2.5 g protein/kg dosing weight
c. If BMI ≥50, use 2.5 g/kg dosing weight
    - *Example:* BMI = 23 → 2 g/kg × dosing weight

Determine protein provisions:

a. Multiple protein dose/kg × dosing weight = grams of protein/day
b. Multiple grams of protein/day × 4 kcal/g = kcal from protein
    - *Example:* 2 g/kg × dosing weight (60 kg) = 120 g/day protein × 4 kcal/g = 480 kcal/day

STEP 3: AMOUNTS OF DEXTROSE AND LIPIDS

Determine lipid dosing:

a. Multiply total energy needs × 0.3 or ≤30% of total kcal = total kcal from lipids
b. Divide total kcal from lipids by 10 kcal/g = grams of lipids/day
c. Divide grams of lipids/day by dosing weight to determine final lipid dosing = g lipid/kg
    - *Example:* 1800 kcal/day x 0.3 = 540 kcal/day from lipids ÷ 10 kcal/g = 54 g/day ÷ dosing weight of 60 kg = 0.9 g/kg

Determine dextrose dosing:

a. Subtract kcal from proteins and kcal from lipids from daily energy requirement = kcal from dextrose/day
b. Divide kcal from dextrose/day by 3.4 kcal/g = grams of dextrose/day
c. Divide grams of dextrose/day by PN volume in L = grams of dextrose/L
d. Calculate percent dextrose concentration by dividing grams of dextrose/L by 10 to = % dextrose concentration
e. Determine final dextrose provisions by multiplying grams of dextrose/L × PN volume × 3.4 kcal/g
    - *Example:* 1800 kcal – 480 kcal – 540 kcal = 780 kcal/day from dextrose ÷ 3.4 kcal/g = ~230 g/day dextrose ÷ total volume of 1.8 L = 128 g/L dextrose × 10 = 12.8% dextrose; 128 g/L × 1.8 L = 230.4 g dextrose × 3.4 kcal/g = 783 kcal/day

STEP 4: ELECTROLYTES, VITAMINS, AND TRACE ELEMENTS

STEP 5: MEDICATION ADDITIVES

LASTLY: Write PN order

- *Example:* P2g/kg D12.8% L0.9 g/kg at 75 mL/h → based on dosing weight of 60 kg provides 1.8 L, 1803 kcal, 120 g protein

FIGURE 62-6 Clinical calculations for parenteral nutrition.

are questioning that practice (see earlier section on EN for detailed discussion). For example, TPN solutions enriched with glutamine have been shown to increase jejunal mucosal weight and nitrogen levels, decrease atrophy of intestinal villi, reduce hepatic steatosis, reduce pancreatic atrophy, and decrease gut bacterial translocation.[63] However, two large multicenter randomized controlled trials published in 2013 and 2014 found that ICU patients with multisystem organ failure receiving immunonutrition had significantly increased risk of death.[76,128]

Step 3: Amounts of Dextrose and Lipids. The next step is to outline the nonprotein kilocalorie distribution between the two major energy sources with PN: dextrose and fat (note that protein as amino acids also provides an energy source but at much lower amounts). Most kilocalories will come from carbohydrates, which are convenient and safe,[33] with only a small amount of fat emulsion to provide the essential fatty acids (Fig. 62-6).

The protein:fat:glucose kilocaloric ratio in surgical patients should approximate to 20%:30%:50%, respectively.[32,33] The common three-in-one or two-in-one formulations will therefore need to be modulated. The three-in-one PN regimen contains all three macronutrients: typically 10% to 15% energy as protein, 30% to 45% as fat, and 35% to 55% as carbohydrate. A two-in-one regimen contains no fat (fat is given separately) and has 15% to 25% energy as protein and 75% to 85% as carbohydrate.

Dextrose in PN should provide 50% to 70% of the daily energy requirements. A basal requirement is estimated to be roughly 2.0 g dextrose/kg IBW/d,[33] much of this for brain metabolism, which oxidizes glucose at a rate of 100 to 120 g/d. During the stress response, the hypermetabolic state creates oxidative limits of energy utilization; for this reason, dextrose should not exceed 5 g/kg IBW/d.[87] If exceeded, this can lead to overfeeding and fatty liver. One method to monitor overfeeding is to check a respiratory quotient (RQ) by indirect calorimetry. The RQ is the ratio of carbon dioxide (CO_2) production to oxygen consumption. Excessive CO_2 production is evidence of overfeeding with carbohydrate and will drive the RQ to be greater than 1. With an elevated RQ, increased kilocalorie contributions from fat should be considered (as can happen in patients with pulmonary failure as well) as fat metabolism generates less CO_2.

Fat emulsions in PN should provide 30% to 50% of the daily energy requirement; total dose of PN fat should be in the range of 1.0 to 2.0 g/kg IBW/24 h.[33] Fat emulsions are usually in the form of long-chain triglycerides (LCT), although in the polytrauma patient, it is best to use a mixture of both LCT and MCT.[33] As discussed previously, during the stress response, MCTs are preferred because they do not exert negative effects on neutrophil and macrophage function or hinder the reticuloendothelial system.

During the systemic inflammatory stress response, the addition of intravenous fish oil to the lipid emulsion, which provides omega-3 fatty acids (both eicosapentaenoic acid and docosahexaenoic acid), has been shown to mitigate multiple aspects of the inflammatory response.[33] Much has been written about the metabolic benefits of omega-3 polyunsaturated fatty acids in fish oils[137,138]; they have been shown to decrease ICU length of stay and have been recommended in Europe since 2005.[33] Although they have long been recommended for and available in EN,[32,34,35] only in July of 2016 did they become FDA approved for PN in the United States. Known here as "Smoflipid," it is an alternative injectable lipid emulsion composed of four different oil sources that spell "smof": soy oil 30%; MCT 30%; olive oil 25%; and fish oil 15%.

If central access is not possible, PN preparations with very limited kilocaloric intake, primarily derived from fat, can be given into the peripheral vasculature. This is known as peripheral PN.

Step 4: Electrolytes, Vitamins, and Trace Elements. The major issue in specifying amounts for electrolytes, vitamins, and trace elements is their contribution to osmolar load. While daily requirements should be met,[33] amounts should not exceed that level unless indicated for other reasons.

Supplementation of PN solutions with immune-enhancing and antioxidant micronutrients is also controversial and in flux. During the hypermetabolic stress response, there is an increased need for vitamins and trace elements, but just how much of an increase is not well defined.

Step 5: Medication Additives. Certain medications can be added to parenteral nutritional formulations to simplify what is often a long list of intravenous and enteral medications in the critically ill patient. Examples include proton pump inhibitors and insulin.

Administration and Delivery

PN cannot be started at full goal intake from day 1; rather, the rate of administration must be gradually increased over a few days to ensure tolerance. The factor limiting immediate goal delivery is glucose and the risks of hyperglycemia.

Monitoring and Complications

Following a patient on PN involves monitoring the metabolic response to the infused nutrients and anticipating or preventing such complications. The major complication is hyperglycemia, and therefore glucose control and monitoring blood sugar levels are of paramount importance.[33,35] A high-risk period is while the volume and glucose load are being increased, before steady levels of administration have been reached. Glucose management can be compounded in diabetics, septic patients, or those on steroids. Hyperglycemia can lead to a hyperosmolar coma, which should

be avoided at all costs. Insulin administration may be necessary.

By its very nature, the aim of PN is to induce an anabolic state (occurs eventually in the hypermetabolic, hypercatabolic polytrauma patient). As such, serum electrolytes will need regular daily monitoring. Patients are at high risk for hypokalemia, as well as alterations in phosphate, calcium, and magnesium. Micronutrient and essential fatty acid deficiencies can also develop. Tonicity and acid-base balance need monitoring as well; TPN solutions have a pH of 4 to 5 and can be balanced by the addition of more base, such as sodium or potassium acetate. Bloodstream infection and complications from venous access (eg, venous thromboembolism) are always a threat. Lastly, although not major issues in the short term, over the long term, liver dysfunction and hypertriglyceridemia are major concerns.

In time, the goal is to transition the patient off of the PN and onto EN. While patients are on PN, EN can be gradually introduced. Once greater than 60% of total daily kilocalorie requirements are being delivered enterally, PN can be stopped.[35]

SPECIAL PATIENT POPULATIONS

Obese Patients

The energy requirement for critically ill and injured obese patients (BMI >30) is lower than that for a nonobese patient and should not exceed 70% of total daily 24-hour energy requirements. The rationale behind this is to achieve some degree of weight loss in this patient population, which may in turn increase insulin sensitivity and reduce the risk of comorbidities.[35] This translates into target kilocalorie requirements of 22 to 25 kcal/kg IBW/d.[35,36,114] Protein requirements for BMI of 30 to 40 are in the range of 2.0 to 2.5 g protein/kg IBW/d; if BMI is greater than 40, then greater than 2.5 g protein/kg IBW/d is required.[35]

Patients with Baseline Malnutrition or High Nutritional Risk

Malnutrition is common: it is estimated that up to 40% of hospitalized patients fit the diagnosis.[8,9] It occurs for two major reasons: because of a patient's primary underlying baseline condition (before they were admitted to hospital, such as alcoholism or malignancy) or because they do not receive adequate nutritional intake for prolonged periods of time (typically while patients are hospitalized, such as after injury or postoperatively). Malnutrition is associated with worse outcomes, including infection (eg, pneumonia, abscess, sepsis), respiratory failure, poor wound healing and pressure ulcers, increased length of hospital stay, and death. These patients are at huge risk for complications if they become critically ill and often need slightly increased energy requirements, especially in the initial and acute phase of their injury.

A baseline nutritional risk assessment must be made and subsequently used to guide nutritional therapy. Randomized controlled trials have shown that nutritional intervention in patients identified to be undernourished or at high nutritional risk improves outcomes.[139,140] Determining the nutritional status of trauma and critically ill patients can be challenging, but knowledge of baseline malnutrition and malnourishment is exceptionally valuable[141,142]: those patients at moderate to severe nutritional risk are most likely to benefit from early nutrition and most likely to be harmed by iatrogenic underfeeding.[35]

Validated scoring systems for nutritional risk include the NUTRIC score (Nutrition Risk Score in ICU[143,144]) as well as the NRS 2002 (Nutritional Risk Screening 2002 tool).[141] The ASPEN/SCCM guideline[35] identifies patients as high nutritional risk if the NUTRIC score is 5 or greater or the NRS 2002 score is 5 or greater. The guideline's section on nutritional assessment is worth reading, as is the consensus statement[145] from the Academy of Nutrition and Dietetics and ASPEN on identifying malnutrition (Fig. 62-7).

Alcoholism

Alcohol use is a major cause of malnutrition; 20% to 60% of patients with alcohol dependence will be malnourished. This has major implications for the polytrauma patient population. These patients often need significantly increased energy requirements, especially once stabilized: kilocalorie goals are 35 to 40 kcal/kg IBW/d with protein intake of 1.2 to 1.5 g/kg IBW/d.

Spinal Cord Injury Patients (See Chapter 26)

Please see Chapter 26 for full specifics on meeting the nutritional needs of the patient with spinal cord injury. In quadriplegic patients, daily energy requirements are met with 20 to 22 kcal/kg IBW/d; in paraplegic patients, it is a bit higher at 23 to 24 kcal/kg IBW/d.[36,146]

Renal Patients (See Chapter 61)

In patients with acute kidney injury, there are multiple adjustments that should be considered to daily nutrition, especially PN. These include decreasing the grams of protein if blood urea nitrogen is greater than 100 mg/dL; decreasing magnesium, potassium, and phosphorus; increasing acetate; decreasing overall kilocalories if on peritoneal dialysis; decreasing fat kilocalories if triglycerides levels are high; and maximally concentrating all solutions.

Intubated Patients (See Chapter 59)

In intubated patients, avoid overfeeding to prevent excessive CO_2 production (which will make weaning off of the ventilator difficult) and try to maximally concentrate all solutions.

Nonsevere Malnutrition/Malnutrition of a Moderate degree in Adults			
	Acute Illness/Injury	**Chronic Illness**	**Social/Environmental**
Weight Loss	1%–2%/1 week 5%/1 month 7.5%/3 months	5%/1 month 7.5%/3 months 10%/6 months 20%/1 year	>5%/1 month >7.5%/3 months >10%/6 months >20%/1 year
Energy Intake	<75% for >7 days	<75% for ≥1 month	<75% for ≥3 months
Body Fat	Mild depletion	Mild depletion	Mild depletion
Muscle Mass	Mild depletion	Mild depletion	Mild depletion
Fluid Accumulation	Mild	Mild	Mild
Grip Strength	N/A	N/A	N/A

Severe Protein Calorie Malnutrition in Adults			
	Acute Illness/Injury	**Chronic Illness**	**Social/Environmental**
Weight Loss	>2%/1 week >5%/1 month >7.5%/3 months	>5%/1 month >7.5%/3 months >10%/6 months >20%/1 year	>5%/1 month >7.5%/3 months >10%/6 months >20%/1 year
Energy Intake	≤50% for ≥5 days	≤75% for ≥1 month	≤50% for ≥1 month
Body Fat	Moderate depletion	Severe depletion	Severe depletion
Muscle Mass	Moderate depletion	Severe depletion	Severe depletion
Fluid Accumulation	Moderate → Severe	Severe	Severe
Grip Strength	Measurably reduced	Measurably reduced	Measurably reduced

FIGURE 62-7 Identifying patients with malnutrition. N/A, not applicable. (Adapted and reprinted with permission. Copyright © Academy of Nutrition and Dietetics. From Skipper A. Malnutrition coding. In: Skipper A, ed. *Nutrition Care Manual.* Chicago, IL: Academy of Nutrition and Dietetics, 2019 edition.)

In patients with overfeeding, indirect calorimetry will demonstrate an RQ of greater than 1.

Low-Flow States and Hypoperfusion (See Chapters 15 and 58)

Historically, EN has been avoided in patients in low-flow states because of the risk of worsening intestinal mucosal ischemia during periods of hypoperfusion. This practice is evolving, as there is evidence to support the safe and beneficial use of trophic feeds even when a patient is in nonrefractory shock on low to moderate dose vasopressors.[107,147]

Hepatic Patients (See Chapter 60)

In patients with liver dysfunction, there are multiple adjustments that should be considered to daily nutrition, especially PN. These include decreasing protein levels in patients with encephalopathy; using BCAA if encephalopathy is unresponsive to medical treatment or is worsening; using both carbohydrate and fat to meet energy requirements; providing 150 g of carbohydrate per day; and maximally concentrating all solutions.

Preoperative Patients and Holding Tube Feeds

For critically ill patients receiving EN, stopping tube feeds at midnight prior to operative interventions and diagnostic tests is unfortunately a routine practice. It is thought to decrease the risk of aspiration when the patient is in the supine position during the operation, and therefore at risk for aspiration. This is of particular concern in the polytrauma patient, as they are frequently in the operating theatre for a multitude of reasons at the start of their hospitalization, and thus tube feeds are frequently interrupted.

Perioperative management of EN should be evidence-based, and established guidelines for preoperative fasting guidelines should be followed.[148-154] For patients who are not intubated, solid foods should be stopped 6 hours prior to an operation, and clear fluid/EN should be stopped 2 hours prior to an operation; nil per os (NPO) beginning at midnight prior to an operation is no longer recommended or justified.

For patients who are intubated with a protected airway, tube feeds (whether gastric or small bowel EN) can safely be stopped just prior to transport to the operating room, ensuring that the tube is placed to suction and/or aspirated immediately before transport. Established exceptions to this practice are operations with planned airway manipulation (such as tracheostomy), operations requiring prone positioning, and operations on the upper intestinal tract.

Regularly stopping tube feeds at midnight leads to patients receiving at most 50% of goal energy requirements from one day to the next.[148] One study in burn patients found that those randomized to have tube feeds continued during their operative debridement (as compared to those who had EN stopped at midnight) had statistically fewer wound infections and reduced caloric deficits.[149] The ESPEN guideline for enteral nutrition in surgical patients does not endorse stopping liquid diet or tube feeds until 2 hours prior to operation.[91]

ELECTROLYTE MANAGEMENT

Most feeds contain adequate electrolytes to meet the daily requirements of sodium, potassium, calcium, magnesium, and phosphate, although specific requirements can vary enormously. Malnourished or metabolically stressed individuals often develop electrolyte derangements.

Sodium and Disorders of Water Balance

Sodium and chloride are necessary to maintain extracellular fluid volume and plasma osmolality.[41,53] The most common electrolyte problems noted in trauma patients are derangements in sodium. While diabetes insipidus or inappropriate antidiuretic hormone (ADH) may occur with head injuries, producing hypernatremia and hyponatremia, respectively, the most common cause for abnormalities in sodium levels is an excess in sodium administration, fluid restriction in patients with traumatic brain injuries, or administration of large volumes of fluid containing low sodium. In the first case, prolonged use of normal saline or lactated Ringer's as a maintenance fluid in association with other hidden sources of sodium administered through multiple antibiotics, H_2-blockers, and so on, in saline produces a gradual and progressive hypernatremia. Many antibiotics contain large amounts of sodium, and since specific admixtures for medications are rarely ordered, intravenous "piggybacks" can reach 2 to 2.5 L/d in some patients. If this is administered in normal saline, hypernatremia develops. Likewise, if medications are mixed in 5% dextrose and water, significant hyponatremia develops.

Assessment of volume status in concert with the low serum sodium concentration will usually identify the patient as hypovolemic, isovolemic, or hypervolemic. Hypovolemic patients usually respond to normal saline or lactated Ringer's infusions. If losses of fluid are chronic and similar to serum (eg, ileostomy losses), additional sodium may be needed in the nutrient solutions. Isovolemic, hyponatremic patients often need little treatment beyond increasing the sodium content in intravenous fluids. In severe cases where urine sodium is elevated at 100 to 200 mEq/L, restriction of free water is necessary by decreasing fluid administration and concentrating the nutrient solution. This problem is most commonly seen with severe head injury or pneumonia and resolves as the patient recovers. Hypervolemic, hyponatremic patients should have nutrient formulas concentrated as much as possible. Other therapy such as diuretics may occasionally be needed.

A less common cause of hyponatremia after trauma is inappropriate ADH secretion. It is usually associated with central nervous system effects induced by head injury, meningitis, subarachnoid hemorrhage, anesthetics, meperidine, carbamazepine, or tricyclic drugs. In addition, a decrease in the vascular volume, secondary to use of diuretics in patients who are on high levels of positive end-expiratory pressure or have large fluid losses from the GI tract, open abdominal wounds, and so on, can also lead to increased ADH secretion and increased sodium loss. Typically, serum chloride concentrations decrease with the hyponatremia, and a hypokalemic metabolic alkalosis with a high serum bicarbonate occurs, especially when diuretic induced. The diagnosis of inappropriate ADH is made by a combination of hyponatremia, a decrease in serum osmolality, a urine osmolality that is elevated relative to serum osmolality, a urine sodium greater than 20 mmol/L, and, if measured, an increase in ADH. Because of the effect of ADH on the kidney, water is absorbed without sodium so that urine sodium and tonicity are high relative to serum. The appropriate therapy is water restriction.

Hypernatremia is also relatively common in the critically ill trauma patient, especially in patients with severe head injury, where a mild hyperosmolar state is often used to decrease intracranial pressure. After bedside assessment, most of these patients can be categorized as hypovolemic, isovolemic, or hypervolemic. Patients with hypovolemic hypernatremia are usually treated with lactated Ringer's solution first to ensure adequate organ perfusion, and then with solutions containing substantial free water (eg, dextrose 5% in water, 0.22% or 0.45% sodium chloride injection). During free water administration, it is appropriate for the sodium to be reduced in the nutrient solution. Patients with isovolemic hypernatremia usually need free water, and sodium should be removed from the PN. Patients with hypervolemic hypernatremia should have intake minimized by concentrating the nutrient formula. Exogenous sodium should be eliminated from PN, medications, and other infusions to the extent possible.

Potassium

Potassium is the main intracellular cation in the body and is required for normal cellular function.[41,53] Hypokalemia is very common after trauma, especially in patients with normal renal function who require aggressive resuscitation with crystalloid. Loss of GI fluids rich in hydrogen and chloride aggravates this hypokalemia. Patients with prolonged nasogastric suction will lose considerable HCl, resulting in metabolic alkalosis and substantial renal wasting of potassium. Drug therapy with loop diuretics, amphotericin B, antipseudomonal penicillins, and corticosteroids has been reported to aggravate renal wasting of potassium. Other drugs, such as inhaled β-agonists (eg, albuterol) and insulin, drive potassium into the cell, also resulting in hypokalemia in some patients. All these conditions will require additional potassium added to the nutrient solution above the standard of 30 to 40 mEq/L that is commonly used in PN or present in enteral formulas. Occasionally, up to 120 mEq of potassium/L must be added to nutrient solutions of patients who were receiving three or four of the previously mentioned drugs to keep them in potassium balance.

Body potassium needs are not proportionate to serum levels. Each 0.25-mEq drop in serum potassium levels between 3.0 and 4.0 mEq/L represents a 25- to 50-mEq deficit in total body potassium. Between 2.5 and 3.0 mEq/L, each 0.25-mEq drop represents an additional 100- to 200-mEq deficit, which must be replaced to avoid precipitous drops with refeeding.

Hyperkalemia is less common than hypokalemia after trauma and is usually associated with compromise in renal function. In general, hyperkalemia from acute renal failure warrants potassium removal from the nutrient solution. Once levels have decreased to 4.0 mEq/L, potassium should be added back in modest doses (eg, 10 mEq/L). Other causes of hyperkalemia are hemolysis of the blood sample and drugs known to cause this disorder, even when renal function is normal. Most laboratories will identify hemolyzed samples that do not require treatment other than repeat analysis. Heparin and trimethoprim have been reported to cause hyperkalemia in patients. Heparin is an aldosterone antagonist that causes sodium wasting and potassium retention. This drug–nutrient interaction occurs with both systemic and low-dose heparin, especially in patients with diabetes and chronic renal dysfunction. Trimethoprim is a component of the combination product of trimethoprim/sulfamethoxazole used frequently for gram-negative systemic infections. It is a weak diuretic with potassium-sparing activities. Patients who experience these interactions should have potassium decreased in the nutrient solution, even when renal function is normal.

Phosphorus

Phosphorus helps maintain a normal pH in the body and is involved in multiple metabolic and homeostatic processes.[41,53] Hypophosphatemia is a common metabolic complication of critically ill patients receiving nutritional support. While most practitioners add phosphorus to PN solutions routinely, several patient populations require greater amounts, including patients with a history of alcohol abuse, poor nutritional status before injury, or chronic use of antacids or sucralfate. Even when serum phosphorus concentrations are monitored closely, hypophosphatemia occurs in approximately 30% of patients receiving PN. Treatment of hypophosphatemia is dictated by the severity, and intravenous replacement doses are most frequently used. The enteral route should be considered in mild cases of hypophosphatemia by adding 5 to 10 mL of Fleet's phosphosoda to each liter of the enteral formula in patients requiring additional phosphate. In patients requiring both potassium and phosphate, potassium phosphate (usually 15–22.5 mmol/L) can be added to the formula. For isolated potassium depression, potassium chloride can be added to the enteral formula.

Hyperphosphatemia is much less prevalent than hypophosphatemia in trauma patients and is usually associated with renal compromise when it does occur. Phosphorus should be decreased or removed from the nutrient solution.

Magnesium

Magnesium is involved in more than 300 enzymatic processes in the body, as well as bone health and the maintenance of intracellular levels of potassium and calcium.[41,53] The development of hypomagnesemia is underappreciated. Magnesium is rapidly depleted in stress, particularly when diuretics and antibiotics such as aminoglycosides are administered. Dysrhythmias, hypocalcemia (an unusual problem in trauma patients), and irritability are avoided with magnesium monitoring and appropriate treatment. Patients with a history of alcohol abuse or lower GI losses, such as diarrhea, are particularly prone to develop hypomagnesemia. Amphotericin B, aminoglycosides, and loop diuretics (and in addition cisplatin and cyclosporine) have all been reported to cause renal wasting of magnesium. Intravenous magnesium replacement therapy is usually necessary in patients with moderate to severe magnesium deficiency due to poor absorption of oral magnesium salts. Magnesium has a renal tubular threshold similar to glucose, so rapid administration over a short period of time will invariably result in high urinary losses.

Magnesium status should also be considered in evaluating a hypokalemic patient, as magnesium is an important cofactor for the Na/K-ATPase pump. It may be necessary to administer magnesium replacement therapy for low-normal serum magnesium concentrations in the presence of hypokalemia, because magnesium is an intracellular cation and serum concentrations may not accurately reflect intracellular status.

Hypermagnesemia usually occurs in association with renal dysfunction or failure. Magnesium should be removed from the PN of these patients.

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63

Postinjury Inflammation and Organ Dysfunction

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KEY POINTS

- Multiple organ failure (MOF) is defined as dysfunction of greater than one organ system.
- Although multiple scoring systems of MOF exist (Marshall, Sequential Organ Failure Assessment, Denver), there is no gold standard.
- MOF accounts for approximately 50% of delayed deaths in trauma.
- The pulmonary, cardiac, and renal systems are affected first, usually within 3 days, followed later by derangements in hepatic function (>3 days).
- Fifty percent of MOF mortality occurs within 3 days, with 80% of mortality occurring within 7 days.
- Traumatic injury and resuscitation from hemorrhagic shock result in proinflammatory and anti-inflammatory responses, which affect multiple organ systems and can contribute to sequential organ dysfunction.
- Prevention of MOF is directed at early resuscitation, avoidance of hypotension, protective lung ventilation, renal protection, and prevention of secondary infection.

HISTORICAL PERSPECTIVE ON MULTIPLE ORGAN FAILURE

With advances in emergency medical services, pharmacology, technology, surgical techniques, and other treatment modalities, the ability of the physician to keep a severely injured patient alive is ever increasing. As more patients have survived initial resuscitation, more patients potentially develop postinjury multiple organ failure (MOF), which has emerged as the leading cause of late trauma deaths.[1-3]

Many advances in trauma care, treatment modalities, and shock were initially stimulated by military need and experience only to be later refined in civilian trauma centers. In World War I, late battlefield casualties were attributed to the release of toxins from dead or dying tissue. Physicians during this time period, particularly Walter B. Cannon, first proposed the concept that hypovolemia is the inciting event that results in organ failure.[4] This concept of hypovolemic shock was not expanded until many years later. In the 1930s, Alfred Blalock demonstrated that reduced circulating blood volume was the main cause of shock and mortality.[5] This knowledge drove treatment modalities toward the restoration and normalization of blood pressure through volume expansion. Patients were resuscitated with freeze-dried plasma and later with stored whole blood. With early restoration of blood pressure, the rate of cardiac arrest observed in World War I decreased, but survivors more frequently developed renal failure. This practice of plasma and blood resuscitation continued into the Korean War, where additional advances in rapid battlefield transport helped improve overall battlefield survival. Patients who once died from hemorrhagic shock now survived initially, although many patients succumbed to late deaths from oliguric renal failure.

These late deaths led G. Tom Shires and others in the 1960s to propose that not only did hypotension lead to shock, but also extracellular or third space fluid deficits compounded the magnitude of traumatic shock. He demonstrated improved survival in animals with the addition of balanced salt solutions to shed blood during resuscitation.[6] From this point until recently, crystalloids were used liberally in addition to blood and plasma resuscitation with the end points of resuscitation focused on maintaining adequate urine output. During this time period, advancements in helicopter evacuation in the Vietnam War enabled rapid transport of the severely injured patient, further decreasing battlefield mortality but resulting in the emergence of more organ failure. Prevention of oliguric renal failure with greater fluid administration, particularly crystalloids, however, presented a new challenge of late deaths from "shock lung." The concept of the acute respiratory distress syndrome (ARDS) was thus born.[7]

During the 1970s, improvements in organ support, specifically mechanical ventilation, vasoactive drugs, parenteral nutrition, and hemodialysis, gave physicians the tools to further sustain the critically ill. Death from isolated single-organ failure became rare, and a new syndrome of "multiple, progressive, or sequential systems failure" emerged.[8] By 1977, the term *multiple organ failure* was coined by Ben Eiseman, who provided the first clinical description of patients with progressive organ dysfunction.[9] Initially the cause of MOF was thought to be the "fatal expression of uncontrolled infection."[10] Concurrent studies at the time pointed toward organ failure as a bimodal phenomenon, with a rapid single-phase MOF from massive tissue injury and shock, or a delayed, two-phase MOF due to moderate trauma and shock followed later by secondary infection and sepsis.[11,12] Clinicians believed that sepsis remained the main culprit of organ failure. By the mid-1980s, however, more evidence demonstrated that organ failure could occur in the absence of infection, and the concept of "generalized autodestructive inflammation" emerged.[13,14]

Noninfectious inflammatory models of MOF, such as trauma or severe pancreatitis, were the focus of investigation in the 1990s. The hypothesis was that patients were resuscitated into an early state of systemic hyperinflammation, the systemic inflammatory response syndrome (SIRS). SIRS is defined clinically by the American College of Chest Physicians/Society of Critical Care Medicine consensus conference as two or more of the following: (1) temperature less than 36.8°C or greater than 38.8°C, (2) heart rate more than 90 bpm, (3) respiratory rate more than 20 breaths/min or P_{CO_2} less than 32 mm Hg, and (4) white blood cell count less than 4000/μL or greater than 12,000/μL or with greater than 10% immature forms.[15]

Like the bimodal distribution of MOF noted earlier, the inflammatory cascade could cause two potential responses to the inciting traumatic event. In a one-event model, a massive traumatic insult overwhelms the ability of a patient to respond to the resuscitative efforts and precipitates early organ failure. In a two-event model, patients may have a moderate response to the initial resuscitation, but this leaves the patient "primed" for a second insult, or "second hit," that dysregulates the immune system, leading to a hyperinflammatory response and sequential organ failure. Examples of second hits include infection, sepsis, abdominal compartment syndrome (ACS), fat embolus, mechanical ventilation, blood transfusions, and subsequent surgical procedures, such as long bone fixation.[16,17] ACS became almost epidemic because of massive fluid resuscitation based on the work of Shires et al[6] and the attempt to achieve supranormal oxygen delivery promulgated by William Shoemaker's group.[18] The administration of massive amounts of crystalloids to achieve supranormal oxygen delivery clearly contributed to the development of ACS.[19,20] Focusing on early, rapid administration of blood products while limiting the amount of crystalloids has led to a decreased incidence of ACS.[20]

By the late 2000s, evidence for the development of two opposite responses to trauma had emerged: a proinflammatory response (SIRS) and its converse, the compensatory anti-inflammatory response syndrome (CARS).[21] Because these responses appear to begin simultaneously, CARS may be a misnomer because it does not seem to be truly compensatory. The *systemic anti-inflammatory response syndrome* may be a better term. The development of MOF is related to the intensity and the balance between pro- and anti-inflammatory states. In severe SIRS, unbalanced early proinflammation from the innate immune system causes early MOF and can result in a fulminant, proinflammatory death. Conversely, early anti-inflammation inhibits the adaptive immune system and creates a preconditioned state in which the patient may be protected against second, inflammatory hits. When the body attempts to counter unbalanced proinflammation, the persistent, severe anti-inflammatory response sets the stage for immune paralysis, impaired wound healing, recurrent infections, and eventual late MOF.[22,23] Current laboratory and translational research provides insights into each specific component and biomarkers of the inflammatory and anti-inflammatory cascades. Thus, each stress, trauma, or septic insult affects cellular functioning and pathways in what has been called the genomic storm.[24] At the cellular expression level, each new gene expression that is decoded adds another potential target for blocking or stimulating agents that may help prevent MOF.

With our current advances in resuscitation and organ system support, we are now observing patients who have survived even these late effects of SIRS and CARS, only to linger for weeks to months in the intensive care unit (ICU) with what has recently been termed the persistent inflammation-immunosuppression catabolism syndrome (PICS).[23] In the next few sections, we will describe more details about MOF, SIRS/CARS, and PICS, including potential mitigating or therapeutic approaches (Fig. 63-1).

DEFINING MULTIPLE ORGAN FAILURE

Simply put, MOF is defined as the sequential dysfunction of multiple organ systems. There is no gold standard scoring system used to identify and measure MOF, particularly the progressive and successive nature of organ dysfunction. Although imperfect, there are a number of scoring systems that measure the total amount and degree of each organ system affected. These scores help establish a baseline degree of organ dysfunction and allow physicians to trend organ dysfunction over time. They also allow stratification of subjects for research purposes.

The most common of these scoring systems are as follows: (1) the Marshall Multiple Organ Dysfunction Score (MODS),[25] first developed in a Canadian ICU; (2) the Sequential Organ Failure Assessment (SOFA),[26] developed by the European Society of Intensive Care Medicine in 1994 and then further revised in 1996; and (3) the Denver Score,[27] developed from a long-term, prospective database from the Denver Health Medical Center. Other intensive care scoring systems exist; however, these often focus on predicting patient

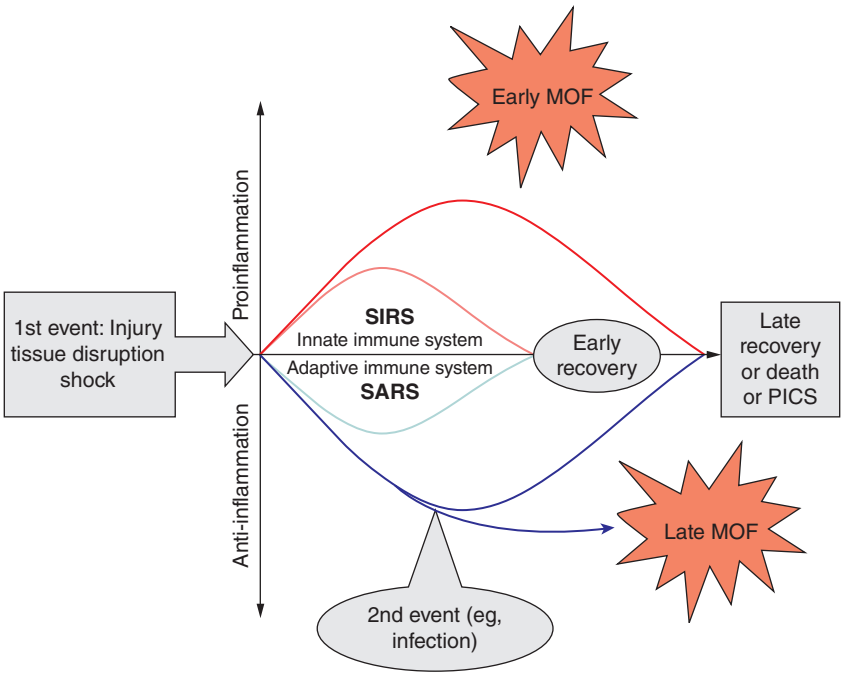

FIGURE 63-1 Theoretical framework for postinjury multiple organ failure (MOF): the synchronous immunoinflammatory model. PICS, persistent inflammation-immunosuppression catabolism syndrome; SARS, systemic anti-inflammatory response syndrome; SIRS, systemic inflammatory response syndrome.

outcomes, length of stay, and overall in-hospital mortality, as opposed to quantifying specific organ failures.

Both the MODS and SOFA scoring systems evaluate the degree of organ dysfunction based on six organ systems. The data of the cardiovascular, pulmonary, hepatic, hematologic, renal, and central nervous system are scored on a scale from 1 to 4. The original Denver Score, based on eight organ systems, was refined to include just four systems, graded 0 to 3, and is assessed every day (Table 63-1).

Multiple analyses of the SOFA score have helped to define and predict outcomes in patients with sequential organ derangement. With MOF defined as dysfunction of two or more organ systems, the lowest rate of mortality is observed in patients with respiratory failure alone. If the respiratory system remains intact but the other organ systems have failed, nearly all potential combinations lead to a mortality between 65% and 74%.[28]

The European Sepsis Occurrence in Acutely Ill Patients (SOAP) multicenter trial examined data from roughly 3100 adult, mixed medical and surgical ICU admissions to evaluate the incidence of MOF. The investigators noted that patients who had severe sepsis and organ failure had the highest mortality. In this group of patients, the highest to lowest mortality groups by system failure were coagulation system (53%), hepatic (45%), central nervous system (44%), cardiovascular (42%), renal system (41%), and respiratory system (35%).[29]

When using SOFA to score patients, the highest, mean, and change in score are useful predicators of outcomes. Higher mean and maximal SOFA scores during the first 48 hours of patient presentation predicted an increased risk for mortality.[30] If the SOFA score increased at 48 hours from presentation, mortality was at least 50%. Conversely, if the score decreased at 48 hours, mortality declined below 27%. If the score did not change over 48 hours, patient mortality remained at 27% to 35%.[31] Cumulative and trending SOFA scores are better at discriminating patient outcome compared to a single organ dysfunction score at one time point.[32]

INCIDENCE

The incidence, presentation, and time course of MOF have changed greatly as new treatment modalities and resuscitation techniques have evolved. Donald Trunkey described a trimodal pattern of death from trauma in 1982.[33] The first peak, which was the tallest, occurred in the first hour following injury and was predominantly caused by severe traumatic brain injury (TBI) and mostly irreparable major cardiovascular injuries. The second peak, over the subsequent 1 to 4 hours, was from ongoing hemorrhage or TBI. The third peak, occurring 1 week or more after injury, was thought to be caused by MOF. Although this peak has significantly decreased over the years, it has not disappeared.[34,35] Despite improvements in resuscitation and critical care, some patients will develop a deranged physiology of inflammation and anti-inflammation, leading to sequential dysfunction of multiple organ systems and late death.

MOF likely accounts for more than 50% of delayed deaths following trauma, although the true incidence and development of posttraumatic MOF are unknown.[36,37]

TABLE 63-1: Denver Postinjury Multiple Organ Failure Score, Sequential Organ Failure Assessment (SOFA) Score, and Marshall Multiple Organ Dysfunction Score

		Score allocation				
Organ system	**Variable (units)**	**0**	**1**	**2**	**3**	**4**
Denver						
Respiratory	PaO_2/FiO_2	>250	250–175	175–100	<100	
Renal	Creatinine, μmol/L	<159	160–210	211–420	>420	
Hepatic	Bilirubin, μmol/L	<34	34–68	69–137	>137	
Cardiac	Inotropes	None	1 inotrope small dose	1 inotrope moderate dose or >1 inotrope small dose	1 inotrope large dose or >2 inotropes moderate dose	
SOFA						
Respiratory	PaO_2/FiO_2		<400	<300	<200 and respiratory support	<100 and respiratory support
Renal	Creatinine, μmol/L		110–170	171–299	300–440	>440
Hepatic	Bilirubin, μmol/L		20–32	33–101	102–204	>204
Cardiac	Inotropes μg/kg per min		Mean arterial pressure <70 mm Hg	Dopamine ≤5 or dobutamine any dose	Dopamine >5 or epinephrine ≤0.1 or norepinephrine ≤0.1	Dopamine >15 or epinephrine >0.1 or norepinephrine >0.1
Coagulation	Platelets, $\times 10^3/mm^3$		<150	<100	<50	<20
CNS	GCS		13–14	10–12	6–9	<6
Marshall						
Respiratory	PaO_2/FiO_2	>300	226–300	151–225	76–150	≤75
Renal	Creatinine, μmol/L	≤100	101–200	201–350	351–500	>500
Hepatic	Bilirubin, μmol/L	≤20	21–60	61–120	121–240	>240
Cardiac	PAR = (HR/MAP) × CVP	≤10.0	10.1–15.0	15.1–20.0	20.1–30.0	>30.0
Coagulation	Platelets, $\times 10^3/mm^3$	>120	81–120	51–80	21–50	≤20
CNS	GCS	15	13–14	10–12	7–9	≤6

CVP, central venous pressure; GCS, Glasgow Coma Score; HR, heart rate; MAP, mean arterial pressure; PAR, pressure-adjusted heart rate; SOFA, sequential organ failure assessment.

This difficulty is due to varying definitions, differences in treatment strategies, and a lack of consensus on a gold standard scoring system. A few studies have attempted to investigate and determine the true incidence of MOF in posttraumatic populations. In a review of 1 million hospital discharges from verified trauma centers, the incidence of posttraumatic MOF varied from 6% to 25%.[36,37] Patients who developed MOF had increased morbidity and were nearly four times as likely to require ongoing assistance with activities of daily living at discharge.[36]

Not all patients who sustain a traumatic injury will develop MOF. There exists a multimodal distribution, with the highest peak of MOF occurring within the first 3 days after injury and a small peak that follows afterward. The organ systems affected most commonly within 3 days are pulmonary, cardiac, and renal systems. Hepatic failure usually occurs after 3 days. Early MOF is associated with worse outcomes. Fifty percent of patient deaths from MOF occur within the first 3 days, and 80% of deaths occur within the first week. Patients who survive MOF have longer clinical courses (mean ICU stays of 18 vs 8 days) and have increased ventilator days (14 vs 5 days) compared to their non-MOF counterparts.[38]

Although mortality would seem to be a straightforward end point, there are important confounders that need to be considered. First, following initial resuscitation and stabilization, patients rarely die while aggressive organ system support is provided. More often than not, late deaths in the ICU occur after the medical team and the family agree to withdraw life-sustaining therapies. Involvement of a palliative care service can facilitate this process when appropriate.[39] Prognostication in the ICU is extremely difficult. It should also be clear that involvement of palliative care services does not change the percentage of patients who die but has been shown to decrease the time to death and ICU length of stay.[40] Second, with the push to transfer patients out of the tertiary or quaternary hospitals to long-term acute care hospitals or rehabilitation centers, patients may still die of their injuries, unbeknownst to the original care team.[41]

PATHOPHYSIOLOGY

Initial Response

Trauma frequently leads to a SIRS response from the combination of tissue injury and ischemia/reperfusion (Fig. 63-2). This response is similar to the generalized inflammatory response observed following infection or nontraumatic, noninfectious conditions such as necrotizing pancreatitis. The SIRS response following trauma is complex but seems to be initiated by a release of endogenous damage-associated molecular patterns (DAMPs).[42] This is similar to the response seen following sepsis, in which microbial products induce a release of pathogen-associated molecular patterns (PAMPs).

Numerous molecules have been identified that activate the inflammatory system after trauma. Hemolysis commonly occurs after trauma, and extracellular hemoglobin can become a redox-reactive DAMP molecule, binding and triggering toll-like receptor–mediated signal transduction leading to increased inflammation.[42-44] Additionally, mitochondrial DNA, which has chemical similarity to bacterial DNA, released from lysed cells can activate circulating neutrophils, worsening organ injury.[42]

Reperfusion injury following hemorrhagic shock–induced hypoperfusion adds to the degree of inflammatory dysfunction. Ischemia/reperfusion induces proinflammatory markers, including cytokines, lipids, and chemokines, which all serve to activate polymorphonuclear neutrophils (PMNs).[17] These factors result in continued activation of the immune system and activation of leukocytes. Smaller studies have shown persistent stimulation of the immune system 28 days after injury in patients without signs of sepsis or a notable second hit.[24]

Polymorphonuclear Response

Patients who develop MOF tend to have a different PMN response to trauma than those who do not develop MOF. Immediately after injury, there is a relatively similar response between MOF and non-MOF patients with regard to total PMN activation. However, as time progresses, patients who go on to develop MOF have increased levels of PMNs.[45] This PMN response occurs as early as 6 hours after the traumatic event. Early demargination, accumulation, and degranulation of PMNs in tissue result in increased local cytotoxic effects by the release of nitric oxide and reactive oxygen species. Degranulation further increases production of systemic proinflammatory cytokines, promoting further inflammation. Early accumulation of PMNs in tissue is facilitated by an increase in endothelial-expressed adhesion molecules allowing for demargination.[46] Interestingly, blockade of the early expression of adhesion molecules significantly reduces

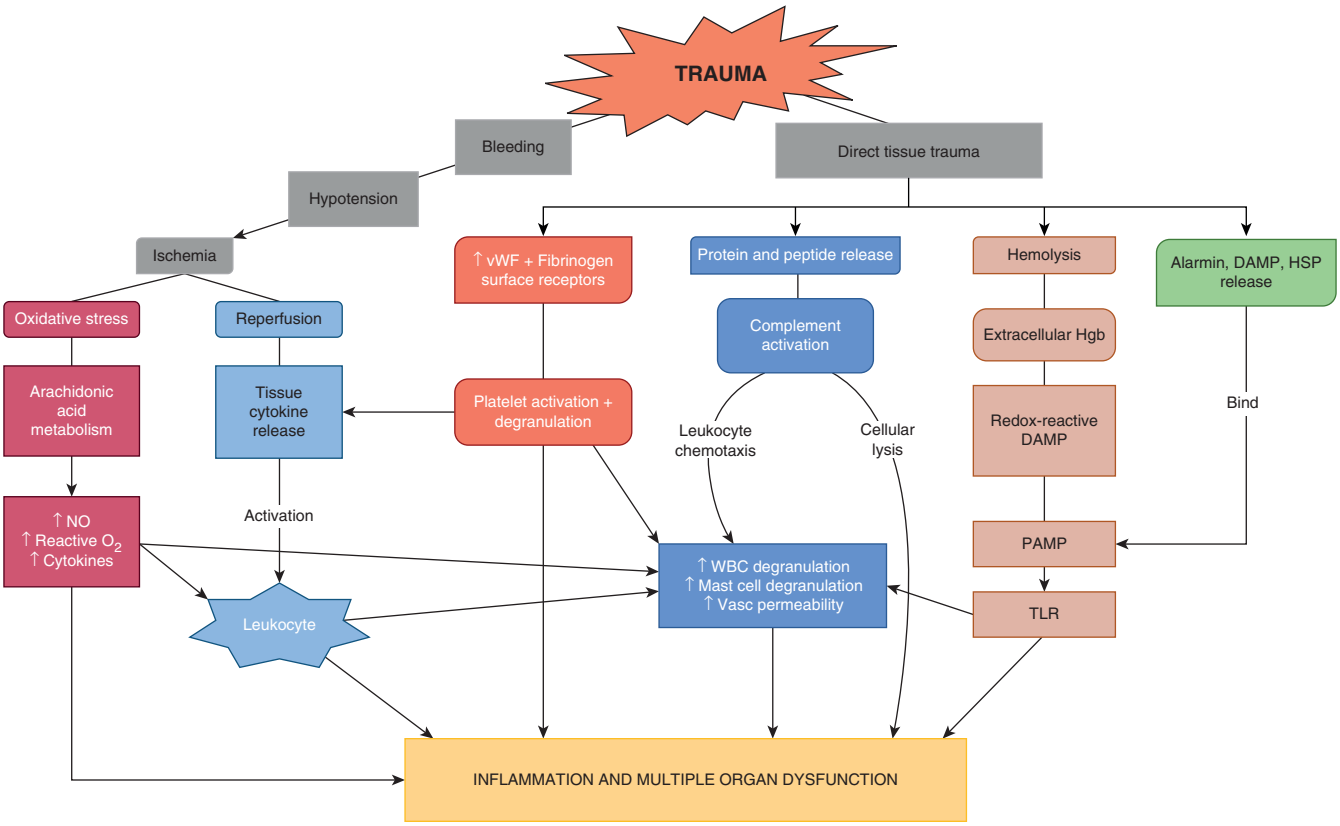

FIGURE 63-2 Pathophysiology of trauma-induced inflammation and development of multiple organ failure. DAMP, damage-associated molecular patterns; Hgb, hemoglobin; HSP, heat shock proteins; NO, nitric oxide; PAMP, pathogen-associated molecular patterns; TLR, toll-like receptors; Vasc, vascular; vWF, von Willebrand factor; WBC, white blood cells.

acute lung injury in experimental sepsis models, suggesting that PMN adhesion and demargination is a critical step in tissue injury during inflammation.[46]

Macrophages

Macrophages play an important role in the development of inflammation, contributing to MOF, following trauma or sepsis. As macrophages are activated, they produce key proinflammatory cytokines including tumor necrosis factor-α and interleukin (IL)-2, which contribute directly to inflammation. They also produce large quantities of nitric oxide, which leads to vasodilation and hypotension and contributes to fluid shifts in sepsis.[47,48] Macrophage signaling is affected during periods of sepsis. In sepsis, macrophages are initially activated by bacterial wall proteins (eg, lipopolysaccharides [LPS]). In late stages of sepsis, however, macrophages are less responsive to LPS.[47-49] This tolerance to LPS contributes to the immunosuppressive stage of sepsis, often seen in patients prior to succumbing. Novel therapies targeting epigenetic regulation and other aspects of macrophage metabolism could better control inflammation and decrease MOF.[49]

Cytokines

Cytokines are local and systemic inflammatory mediators produced from systemic immune cells and cells at sites of injury. They help regulate hemodynamic, metabolic, and immune responses through cellular activation. Activation is achieved by binding specific membrane proteins that result in upregulation of gene transcription to ultimately influence activation, differentiation, proliferation, and survival of immune cells. Cytokines are classified into two types: proinflammatory and anti-inflammatory (Table 63-2). Both types have feedback mechanisms that result in downregulation of the respective pro- or anti-inflammatory counterpart.

Timing and degree of the cytokine release are important in the development of MOF. In one study of the temporal cycling of cytokine production in patients who suffered severe torso trauma, proinflammatory cytokine levels were higher at every time point in patients who developed MOF than in those who did not.[50] Peak cytokine production also appeared to separate patients who develop early (<3 days) MOF versus those who develop late (>3 days) MOF, with patients who developed early elevated cytokine levels having worse outcomes.[51] A rapid initial peak of proinflammatory cytokines, specifically those that activate PMNs (IL-6 and IL-8), was seen in patients who developed early MOF. Patients who developed late MOF had a secondary, delayed increase in inflammatory cytokines.[51] Substantial differences in cytokine levels have been seen as early as 90 minutes after injury. These differences may help differentiate patients who go on to develop MOF from those who do not.[52]

TABLE 63-2: Pro- and Anti-inflammatory Cytokines

Anti-inflammatory	Proinflammatory
TGF-β	TNF-α
IL-4	IL-1
IL-5	IL-6
IL-10	IL-8
IL-11	IL-21
IL-13	IL-22
bFGF	IL-26

bFGF, basic fibroblast growth factor; IL, interleukin; TGF, transforming growth factor; TNF, tumor necrosis factor.

Complement

The complement system constitutes a major component of the innate immune system. Complement functions to help mediate inflammation, clear immune complexes and apoptotic cells, and identify and eliminate microorganisms.[53] Complement also enhances the adaptive response, connects the innate and adaptive immune systems, and links the coagulation and immune systems together.[54,55]

Tissue trauma induces the release of proteins and peptides. These peptides activate the alternative pathway of the complement system, which leads to the chemotaxis of leukocytes, degranulation of white blood cells (including mast cells), and increased vascular permeability.[44,53] In addition, complement production and binding of peptides result in the formation of complement complexes that mediate cell lysis and enhance hemostatic capacity. This continues to activate the inflammatory cascade as complement triggers oxygen free radical production, metabolites, and cytokine formation.

Not only does trauma initiate the complement cascade, but it also has an effect on complement regulation. Regulatory proteins usually exist to help modulate and protect against unwarranted complement-mediated tissue destruction. In trauma patients, however, these regulatory proteins are altered, creating a state of complementopathy with increased complement factors, tissue destruction, and inflammation.[56-60]

Pathogen- and Damage-Associated Molecular Patterns and Heat Shock Proteins

Stressed and injured cells play a direct role in activating the immune system by triggering innate and adaptive responses through release of PAMPs, DAMPs, and alarmins. PAMPs, true to their name, are exogenous microbial molecules that trigger intracellular signaling pathways through toll-like receptors that are located on the membrane of the immune cells of the adaptive and innate immune system. Damage to the body's cells causes release of endogenous molecules that act similarly to PAMPs, but are named alarmins due to their endogenous release. Examples of major DAMPs include high mobility group box protein-1 (HMGB1), heat shock proteins, uric acid, and released DNA. DAMPs have been shown to be elevated 30 times above normal in trauma patients

within 1 hour after injury and have been associated with SIRS and end-organ damage in animals.[61-63]

Another important cellular consideration in immune modulation is that of the role of mitochondrial DNA. Mitochondrial DNA arose from endosymbiotic bacteria. Thus, release of mitochondrial DNA from cellular damage may activate the immune system identically to the bacterial pathways signaled through PAMPs, making tissue trauma akin to inflammation and sepsis.[42]

Role of Platelets

Inappropriate platelet activation and degranulation has been implicated in the development of MOF. Worsening thrombocytopenia has correlated with patient deterioration, with a higher likelihood of MOF development.[64,65] In patients who go on to develop MOF, there is upregulation of platelet surface factors for fibrinogen and von Willebrand factor, which leads to activation and degranulation of platelets, promoting microvascular fibrin deposition.[66] Increased degranulation of platelets strongly correlates with increased severity of organ dysfunction. Increased platelet degranulation causes further activation of neutrophils, oxidative stress, and damage to end organs.

The impact of platelet activation on organ dysfunction is quite notable in models of transfusion-related acute lung injury (TRALI). In lung injury, as discussed earlier, platelet activation and degranulation stimulate neutrophils and result in oxidative stress, local tissue damage, and respiratory distress. In rat models, when platelet activation was blocked with aspirin, local tissue damage and acute lung injury were prevented.[67,68] Similarly, targeting and blocking another platelet activating receptor, P2Y12, protected against acute lung injury in another TRALI mouse model.[68] Pretreatment with antiplatelet therapy helped decrease the risk of MOF in high-risk patients with blunt trauma who received blood transfusions.[69] Antiplatelet therapy (aspirin) was additionally associated with a reduction of mortality in ICU patients with ARDS.[70] In contrast, the Lung Injury Prevention with Aspirin Study Group (LIPS-A)[71] found no benefit of aspirin over a placebo in the development of ARDS at 7 days in patients in the emergency department who were at risk for the development of ARDS (defined as a lung injury prediction score >4).

Role of the Gut

The gastrointestinal tract appears to have a significant relationship with the development of postinjury respiratory dysfunction. The gut is the last organ to have restoration of perfusion after an ischemic insult.[22,44] Ischemia and reperfusion promote the disruption of the barrier between enterocytes and intraluminal bacteria. Gut-derived inflammatory mediators are not only released from intestinal bacteria, but are also released from the intestinal cells secondary to ischemia/reperfusion injury. These pathogenic components enter the systemic circulation through the mesenteric lymph via the thoracic duct. The first organ system encountered is the lungs.[72-74] This is a key reason why respiratory failure commonly precedes development of failure in other organs (heart, kidney, liver).[75]

The microbiome of the intestinal tract has emerged as an important contributor to patients' overall homeostasis. During critical illness, the composition of the microbiome changes.[76] For example, in patients with SIRS, the fecal microbiome may change into one of three patterns—a diverse, single, or depleted pattern. These changes are associated with clinical outcomes, as patients with depleted or single patterns have a significantly higher mortality compared to patients who retain diverse patterns.[76,77] With a better understanding of the effect of the microbiome on the development of MOF and even mortality following trauma and critical illness, the microbiome has become one more potential target for therapeutic interventions in the future.

Oxidative Stress

Reactive oxygen intermediates (ROIs) are generated by mitochondrial oxidation, arachidonic acid metabolism, nicotinamide adenine dinucleotide phosphate (NADPH) oxidase in phagocytic cells, and, lastly, activation of xanthine oxidase. ROIs serve to function in cellular homeostasis, mitosis, differentiation, and cellular signaling.[44] Excess ROIs can overcome the endogenous antioxidant defenses and lead to oxidative stress.[78] Oxidative stress can lead to injury by disrupting cellular proteins, nucleic acids, and cell membranes.[78,79]

Ischemia and reperfusion injury are seen in nearly every hypotensive and resuscitated trauma patient, leading to increased production of reactive oxygen species. These molecules are generated by shifts in cellular energy generation from aerobic to anaerobic metabolism during periods of hypoxemia and ischemia. As the cell decreases energy production (loss of adenosine triphosphate [ATP]), changes in cell membrane permeability result in increased intracellular sodium, causing resultant cell swelling and membrane damage. Decreased ATP results in increased cytosol calcium levels, which activates phospholipases and proteases that perpetuate cell membrane damage. Lastly, ATP hydrolysis increases cellular adenosine monophosphate (AMP) and purine metabolites.[80] As reperfusion increases the availability of oxygen, the purine metabolites are oxidized into urate and superoxide radicals, which contribute to cellular stress and damage. Additionally, ischemia/reperfusion stimulates ROI secretion from PMNs, which further induces cytokines and chemokines, causing more cell and tissue damage.[44]

PREVENTION AND TREATMENT

Despite decades of experimental studies and clinical trials, there are no specific treatments for preventing and treating MOF in patients. Management consists of supporting physiology and optimizing organ system function. Following severe trauma, the initial insult has occurred and management is focused on controlling hemorrhage, resolving shock, supporting each organ system, and preventing any further secondary insults. This is done by maintaining tissue

TABLE 63-3: Measures to Prevent Multiple Organ Failure

System or condition	Interventions
Pulmonary	Lung-protective tidal volume (6 mL/kg) and plateau pressure (<30 torr)
	PEEP management
	Prevention of cyclic opening and closing of airways
	Fluid-restrictive resuscitative strategies
Renal	Rapidly treat shock
	Avoid nephrotoxic agents
	Appropriate dosing of known nephrotoxic drugs
	Hydration prior to contrast agents
Hospital-acquired infection prevention	
Ventilator-associated pneumonia	HOB >30°, chlorhexidine oral rinse, daily interruption of sedation and aggressive weaning strategies such as daily breathing trials
CLABSI and CAUTI	Sterile technique; removal as soon as possible
Nutritional support	Early enteral feeding
Glycemic control	Blood glucose range 140–180 mg/dL
	Avoid hypoglycemia
	Minimize glycemic variability

CAUTI, catheter-associated urinary tract infection; CLABSI, central line–associated bloodstream infection; HOB, head of bed; PEEP, positive end-expiratory pressure.

oxygenation, preventing and treating infections, adequately resuscitating patients while avoiding overresuscitation, providing early enteral nutrition, and minimizing any iatrogenic complications (Table 63-3).

Resuscitative Strategies

Episodes of hypovolemia are common following trauma and in septic patients, and hypotension or circulatory shock is a common inciting event occurring prior to the development of MOF. Timely restoration of intravascular volume and oxygen delivery is imperative to help prevent the development of MOF. The optimal type and amount of fluid used in resuscitation is constantly under discussion (see Chapter 17).

The ideal fluid choice in patients is replacement of the fluids that have been lost. Thus, in trauma patients, blood products should be used for resuscitation. Fresh whole blood may be the ideal and is now becoming available again in the civilian setting. Traditionally, packed red blood cells (PRBCs) were administered first, followed by fresh frozen plasma (FFP), cryoprecipitate, and platelets (PLT) as needed. More recent studies have demonstrated a benefit of proactive, balanced blood product administration, now termed *damage control resuscitation*. Early blood product administration in a 1:1:1 (FFP:PLT:PRBC) ratio increased the frequency of hemostasis, decreased mortality from hemorrhage within 24 hours of injury, and improved early (<24 hours) survival in patients receiving more than 3 units of blood compared to a 1:1:2 (FFP:PLT:PRBC) ratio.[81,82] Initiating FFP transfusion as soon as possible, even outside the hospital, may be advantageous.[83]

When choosing fluids, other than blood, the initial choices are crystalloids or colloids (starches and albumin). Multiple trials, including the Scandinavian Starch for Severe Sepsis/Septic Shock (6S) and the Crystalloid Versus Hydroxyethyl Starch (CHEST) trial, compared the use of colloids (starches) with that of crystalloid (modified Ringer's lactate).[84,85] Not only did these trials fail to show a survival benefit with starches, but they also demonstrated an increased risk of acute kidney injury. Resuscitation with albumin compared to crystalloid showed no improvements in 28- or 90-day mortality.[86] Dextrans and hetastarches can also alter platelet function and increase renal failure.[87] Solutions containing large chloride concentrations, compared to balanced salt solutions, promote a hyperchloremic metabolic acidosis and have been associated with worsening renal function and increased need for renal replacement therapy.[88,89]

In the 1980s and 1990s, resuscitation of trauma patients was focused on reversing hypovolemia and preventing extracellular fluid loss. Supranormal hemodynamic targets for resuscitation were promoted through the use of pulmonary artery catheters.[90] These practices led to injudicious and often harmful quantities of fluid administration, dilution of coagulation factors, pulmonary dysfunction, worsening MOF, and infectious complications.[6,90,91] Additionally, the association between positive fluid balance and mortality in a range of critical illnesses has been well established.[92]

In general, fluids should be administered if the patient is hypotensive and/or demonstrates evidence of hypoperfusion. They should be administered by bolus infusions in the ICU, so that proper responses can be monitored to assure that therapeutic end points are being achieved. Prolonged maintenance fluid therapies should be avoided to decrease the risk of excess fluid administration, which contributes to worse outcomes of organ failure.

A variety of resuscitation end points have been studied, including normalization of acid-base status (lactate, base deficit, pH) and urine output.[93] Oxygen delivery and cardiac output have traditionally been monitored with pulmonary artery catheters. Two promising, less invasive technologies are focused ultrasound assessment and arterial pulse waveform analysis. Focused ultrasound assessment, although user dependent, allows for real-time assessment of cardiac filling, diameter and collapsibility of the inferior vena cava, cardiac function, and the presence of pulmonary edema. Arterial pulse waveform analysis provides a beat-to-beat estimate of cardiac output and stroke volume variation. These technologies provide real-time analysis of volume status to guide adequate treatment, although further studies are needed to demonstrate efficacy.[91]

Multiple strategies have been adopted for limiting fluid resuscitation, and the notion of "deresuscitation" has been

promoted, particularly in the setting of positive fluid balance.[90] One such study by Silversides et al[92] evaluated 400 patients from 10 mixed ICU sites. The use of deresuscitation techniques, including diuretics and renal replacement therapies, to achieve negative fluid balance on day 3 of ICU stay was associated with improved patient outcomes and a lower mortality.

Although restoration of organ perfusion in trauma patients initially requires fluid resuscitation, there is a role for the judicious use of vasoactive agents (eg, vasopressors or inotropes) in many patients. The use of sedation or anesthesia to facilitate procedures can cause vasodilatation and hypotension. In addition, prolonged shock can lead to vasoplegia. Focused ultrasound, arterial pulse waveform analysis, or pulmonary arterial catheterization can help with titrating these therapies, although early use of vasopressors, especially in the setting of hypovolemia, is associated with increased mortality.[94]

ORGAN SYSTEM SUPPORT

Mechanical Ventilation

Mechanical ventilation, although lifesaving for patients with critical illness, can contribute to worsening lung pathology and organ failure (see Chapter 59). Overdistension of the alveoli, by either high pressures (barotrauma) or high volumes (volutrauma), will lead to direct lung injury and an inflammatory response that can further damage other organ systems.[95-97] A lung-protective strategy of low tidal volumes (6 mL/kg) and low plateau pressures (<30 cm H_2O) for patients with ARDS can increase ventilator-free survival, decrease organ failure, and improve survival.[98] Applying this strategy in patients at risk for ARDS may also be beneficial.[99]

Respiratory cycling of opening and collapsing inflamed airways (atelectrauma) can further precipitate damage to the stressed pulmonary system. Positive end-expiratory pressure (PEEP) has been used to help prevent the cyclic collapse, as PEEP can be adjusted to just above the inflection point where alveoli collapse or de-recruitment occurs.[100] This method of PEEP adjustment has shown some improvement in mortality compared with controls. In patients with moderate to severe ARDS, a higher PEEP was associated with improved survival.[101,102] Lastly, a conservative fluid strategy has been shown to improve ventilator-free days and oxygenation at no risk to increasing other organ failures.[103]

In patients requiring increased ventilatory support with the development of severe ARDS, the early use of short-acting paralytics and prone positioning can improve outcomes.[104,105] Extracorporeal membrane oxygenation (ECMO) may play a role in the management of some patients with refractory hypoxemia.[106]

Renal Support

Acute kidney injury (AKI) commonly develops following trauma and is associated with a mortality rate between 40% and 80% (see Chapter 61).[107] Acute tubular necrosis, stemming from hypoperfusion (a prerenal insult), is the cause of acute renal failure (ARF) in the majority of patients (75%). Knowing this, it is imperative to adequately and expeditiously resuscitate patients. Any use of nephrotoxic drugs during the periods of shock and hypovolemia should be avoided if possible. Dosing should be based on pharmacokinetic principles using drug levels if available.[108,109] This is especially important in patients receiving antimicrobials that have been implicated in AKI, including vancomycin, aminoglycosides, and amphotericin B. It is also worth keeping in mind that some drugs cause direct toxic effects on nephrons and others cause indirect injury (eg, interstitial nephritis from penicillins). The attributable risk of AKI from antimicrobials has been difficult to determine in critically ill patients because sicker patients tend to develop more infections and receive more antibiotics. The impact is likely far less than that of other more definitive risk factors, including shock, sepsis, and prior renal dysfunction.

Once patients develop AKI, the use of any nephrotoxic agent should be reassessed. In addition, dosing of all medications that have any renal clearance should be adjusted appropriately. Renal replacement therapy (RRT), either intermittent or continuous, may be needed for management of fluid overload, electrolyte abnormalities (particularly hyperkalemia), refractory metabolic acidosis (after adequate hemodynamic stabilization), or uremia (particularly pericarditis). Theoretically, high flux RRT or RRT using specifically designed adsorbing materials could remove many of the mediators of MOF. Unfortunately, studies using these approaches have not demonstrated any benefit.[110]

Early Fracture Stabilization

The approaches to operative management of fractures in patients with multiple trauma has involved either early total care (ETC) or damage control orthopedic surgery (DCO). In ETC, patients have definitive fixation of all fractures as soon as feasible. ETC, especially of a femur fracture, has been shown to decrease pulmonary complications and promote early patient mobilization when compared to traditional nonoperative management.[111] When ETC is compared to delayed fixation, ETC is also associated with fewer pulmonary complications plus decreased ICU and hospital lengths of stay.[112] Although early fracture fixation in trauma patients has its benefits, many polytrauma patients are too unstable and cannot undergo early, definitive fixation. The importance of patient selection cannot be overstated, as attempting ETC in unstable or poorly resuscitated patients is associated with high rates of pulmonary complications.[113] In addition, these procedures are often lengthy and associated with a great deal of blood loss, potentially setting up patients for a "second hit" and the development of MOF. This led to the concept of DCO, which includes early abbreviated fracture stabilization, usually with splints and external fixator devices.[114] This provides long bone fixation, allows early mobilization, and helps to prevent the prolonged operative times that can lead to a second hit and precipitate MOF. Because posttrauma days 2 to 4 are "fraught with peril," because patients have immunologic changes, ongoing fluid shifts, tissue edema, and may not have achieved normal physiology,[115] timing of definitive

fixation after DCO is usually performed between posttrauma days 5 and 10. In one study, definitive fixation between days 2 and 4 was associated with more frequent development of MOF compared to fixation between days 6 and 8.[116] If possible, definitive fixation should be accomplished within 2 weeks, as infections in external fixator pin sites increase after this time point.[117]

Prevention of Hospital-Acquired Infections

Patients on mechanical ventilation are at risk for ventilator-associated pneumonia (VAP), which can play a significant role in the development of MOF. Bundled measures aimed at the prevention of VAP include elevation of the head of the bed, optimization of sedation with daily spontaneous awakening trials, daily spontaneous breathing trials, aggressive weaning protocols, and chlorhexidine oral rinse.[118-120]

Central line–associated bloodstream infections and catheter-associated urinary tract infections (CAUTIs) are two catheter-related sources of iatrogenic infection. In order to reduce the risk of central line infection, catheters should be inserted following maximal sterile technique (handwashing, cap, mask, sterile gloves and gown, large sterile drape) and skin preparation with an alcohol-based chlorhexidine solution; femoral insertion should be avoided.[121-123] Use of antibiotic-impregnated catheters and chlorhexidine sponge dressings and expeditious and early removal of catheters help reduce infection risk.

The best prevention for CAUTIs is minimizing the use of urinary catheters. Recommendations include a daily assessment of the need for an indwelling catheter, consideration for alternate urine collection methods, emphasis on sterile technique during insertion, and proper maintenance after insertion.[124]

Nutrition

Trauma and sepsis are prominent stressors and increase the metabolic demand of the patient. This can lead to a state of catabolism, resulting in unwanted tissue breakdown and proteolysis. Malnutrition can further contribute to the morbidity and mortality of MOF. Early nutrition, started within 24 to 48 hours of admission, can help suppress this process (see Chapter 62), shift the body toward anabolism, and decrease the risk of developing MOF.[125] Enteral nutrition, which is more physiologic and helps maintain gut integrity, is preferred over parenteral nutrition. The body's hypermetabolic response to trauma and sepsis is driven by the release of endogenous catecholamines, and early administration of catecholamine blocking agents (β-blockers) has been demonstrated to reverse the hypermetabolic response and protein catabolism in burn patients.[126]

Glycemic Control

Hyperglycemia has reproducibly been associated with worsened outcomes in the critically ill, particularly surgical patients. Intensive glucose control in postoperative populations may impact survival and surgical complications.[127,128] In 2001, Van den Berghe et al[129] compared tight glucose control (blood glucose 80–110 mg/dL) with the common management goal at the time of less than 180 mg/dL in a medical ICU in which many of the patients received early parenteral nutrition. The tighter glucose control reduced hospital mortality by 3% to 4% compared to the control. Tight glucose control was also associated with reductions in bloodstream infections, acute kidney injury, critical illness polyneuropathy, hyperbilirubinemia, anemia, and the duration of mechanical ventilation.[129,130] Subsequent studies have not been able to reproduce these results. The NICE-SUGAR trials, which randomized over 6000 mixed medical and surgical ICU patients to tight (81–108 mg/dL) versus conventional (<180 mg/dL) glucose control, demonstrated a higher 90-day mortality and 13-fold increased risk of hypoglycemia with tight control.[131] Hypoglycemia has been associated with increases in cardiovascular mortality and an increased risk of overall mortality among diabetic and nondiabetic patients.[132-134]

Glycemic variability, defined as acute fluctuations in glucose, is another factor that may be more important than previously thought. These fluctuations are often a direct result of the treatment of hyperglycemia. This leads to increased oxidative stress, causing endothelial dysfunction and vascular damage, and worsens the inflammatory cascade.[133] In nondiabetic, critically ill patients, glycemic variability confers an increased mortality risk of 26%.

The current management of glucose control is aimed at decreasing glycemic variability and stopping episodes of hypoglycemia. For now, the most prudent approach seems to be to maintain the blood glucose below 180 mg/dL by initiating interventions to control blood glucose when the level is 150 mg/dL or higher and to maintain blood glucose above 70 mg/dL.[135,136]

SUMMARY

Patients with severe trauma are at high risk of developing SIRS and CARS following resuscitation, leading to late deaths from MOF or the development of PICS. Currently, there are no specific treatments to prevent or treat SIRS or CARS. Support of failing organs and prevention of secondary insults are the best management strategies.

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Trauma and Global Health

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KEY POINTS

- At least 250 million disability-adjusted life-years (DALY) are attributed to traumatic injuries around the world each year.
- In 2018, an estimated 1,350,000 individuals died from road traffic injuries around the world.
- Ninety percent of the world's traffic-related fatalities occur in low- and middle-income countries.
- In 2015, the World Health Assembly passed Resolution 68.15 calling for emergency surgical care as a part of universal health coverage.
- The Residency Review Committee and the American Board of Surgery approved international rotations to fulfill graduation requirements for surgical residents in 2011.
- The "P" values of successful international research collaborations include strong **p**artnerships, knowledge of the **p**eople, requirement for **p**atience and di**p**lomacy to build **p**roductive programs.
- Initiatives from the Global Health Advocacy Incubator have resulted in 11 countries passing new or improved road safety laws, thus protecting over 3.36 billion people

". . . a new and sweeping utopia of life, . . . where the races condemned to one hundred years of solitude will have, at last and forever, a second opportunity on earth."

—Gabriel Garcia Marquez

INTRODUCTION

Trauma is a public health challenge of global proportions. Roughly 5 million individuals lose their lives each year from road traffic collisions and interpersonal violence, and most of these events occur in low- and middle-income countries (LMICs).[1] The challenges of treating traumatic injury are not divorced from the broader challenges of health systems and lack of surgical access. Much of the death and disability is attributed to inadequate prehospital and hospital systems that are understaffed and unequipped to bring patients to receive care or to provide emergent surgical intervention. Comparing mortality rates for Injury Severity Score–matched injuries between high-income countries (HICs) and LMICs suggests that up to 1.9 million (almost 40%) of these lives could be saved with improved health systems.

Acute care surgeons are well positioned to promote improvements in trauma and emergency surgical care, injury prevention, regionalization of acute care, and implementation of data systems to drive process improvement in global surgery.[2] Trauma surgery already has a long history of contributors and beneficiaries of international partnerships.[3]

Dr. Oswaldo Borraez was a young attending surgeon in Bogota, Colombia, in the 1980s when he first thought of using an inexpensive, sterile plastic bag as a means of temporary coverage of the open abdomen. He showed this to a visiting surgeon from the United States, Dr. David Feliciano, who later introduced the technique to the trauma community as the "Bogota bag" in the United States. Variants of this methodology continue to be used worldwide.[4] Similarly, trauma surgeons from LMICs routinely visit major trauma centers in high-resource trauma settings to learn about regionalization and advances in care.[5]

Unfortunately, for too long surgical care has been considered too expensive for provision in the world's poorest regions. Surgeons, too, were largely silent in the debates over investment in health resources. This is beginning to change. Surgical trainees are increasingly seeking meaningful international experiences. Many academic departments of surgery have begun to recognize the value of a variety of global health endeavors.[6-8] The concept of "global surgery" has evolved from simple outreach volunteerism on the part of individuals, nongovernmental organizations (NGOs), and faith-based organizations to a mature academic discipline. This discipline encourages formal postresidency training oriented toward

establishing international partnerships and long-lasting institutional multidisciplinary collaborations.[7] Recent work from the Lancet Commission on Global Surgery, the World Bank's Disease Control Priorities Third Edition (DCP3), and the World Health Organization (WHO) Assembly Resolution 68.15, among others, have framed global surgery as part of worldwide initiatives for health equity. We believe that the term *global surgery* now defines a discipline that seeks to improve the surgical needs of all marginalized and vulnerable populations, irrespective of their regional, national, or subnational geographic location. For the purposes of this chapter, however, we will focus on vulnerable populations in geographic areas of the world with the most disparity (LMICs) as defined by the World Bank.

In this chapter, we will detail the global burden of emergent and traumatic surgical disease, contextualize the recent surge of interest in global surgery, and outline critical areas for collaboration between trauma and emergency general surgeons from across the world.

THE GLOBAL BURDEN OF SURGICAL DISEASE AND TRAUMA

There is an immense need for surgical services worldwide. The *global burden of disease* refers to the total volume of ailments that affect humans, and researchers estimate that between 28% and 32% of it will require surgical expertise for treatment.[9] The AIDS patient who develops an empyema necessitating drainage will require surgical services just the same as the trauma patient with a shattered spleen. At least 15% of pregnancies will result in complications that need emergency obstetric care, including surgical management.[10] The disability-adjusted life-year (DALY) is a composite measure developed to quantify the years of life lost from a premature death or the years of life lived at diminished potential due to morbidity from illness. At least 250 million DALYs are attributed to traumatic injury each year (DCP3, Chapter 2), surpassing the 214 million DALYs per year from AIDS, tuberculosis, and malaria, and the 130 million DALYs per year from ischemic heart disease.[11,12] This is not surprising, as surgery is such an effective treatment modality. The presence of a functional surgical infrastructure including systems-based care for prehospital and posthospital coordination allows for the treatment of a broad range of diseases. Over half of the 45 million deaths in LMICs each year could be potentially averted with improved prehospital or emergency care. This translates into a staggering 1023 million DALYs, or 932 million years of life lost to premature mortality.[13]

In 2018, an estimated 1,350,000 people died from road traffic injuries alone.[14] More than half of these deaths were pedestrians, bicyclists, or motorcyclists. Also, road traffic injuries are the leading cause of death among people in the core years of workforce productivity (ages 15–29 years). Astounding as these numbers are, they represent only a small fraction of the disability generated from road traffic injuries. Roughly fourfold more people are disabled than die from road traffic injuries. As economic development brings more and more people into the middle class, the number of motor vehicles and the number of road traffic incidents are projected to increase.[15] LMICs already are home to 90% of the world's traffic-related fatalities with just about half of the world's vehicles.[16] It is estimated that road traffic injuries will become the seventh *overall* cause of death by 2030, and the economic cost will reach a staggering 3% of the gross domestic product in certain countries.[17]

Crime, drug abuse, alcoholism, and social unrest continue to fuel interpersonal violence in low-income countries and HICs, alike. A population-based survey showed that 10% to 69% of women worldwide reported being physically assaulted by an intimate male partner at some point in their lives.[18] Gun violence, mass shootings, and domestic and international terrorism add additional pressures on training programs both at the residency and fellowship level to ensure proficiency in the management of severe penetrating injuries. In fact, the need may be greater in lower resource settings. The rate of violent death in LMICs is more than twice the rate in middle-income countries.[19] Of the top 50 cities in the world with the highest murder rates excluding current war zones, 17 are in Brazil, 12 in Mexico, 5 in Venezuela, and 5 in the United States (Fig. 64-1).

POOR ACCESS TO TRAUMA CARE IN THE CONTEXT OF POOR SURGICAL ACCESS AND THE RISE OF GLOBAL SURGERY

Most of the world's population does not have access to surgical care when needed. Initial estimates have reported that at least 2 billion people do not have access to surgical care based on operating theater capacity alone.[20] When additionally accounting for timeliness, safety, and affordability, this number rises to more than 5 billion, or 70% of the world's population[21] (Fig. 64-2). In the world's poorest regions, including parts of South Asia and Africa, over 95% of the population are without access. The challenge to improve access is exacerbated by pervasive deficits in the infrastructure, workforce, and financing necessary to provide surgical care (Fig. 64-3).

Aware of the challenges of reaching a surgical hospital with affordable care, many patients delay seeking treatment when faced with illness.[22] Over 90% of deaths from abdominal conditions in India occur in rural areas, with over 70% occurring at home.[23] Unfortunately, too many patients in these environments live hundreds of kilometers away from the nearest surgical hospital.[24] Accessing care will require many hours or days of travel on multiple modes of transportation including bullock carts, bicycle, and on foot. If patients do make it to the hospital, they will likely find equipment and medication shortages; insufficient numbers of physicians, nurses, and

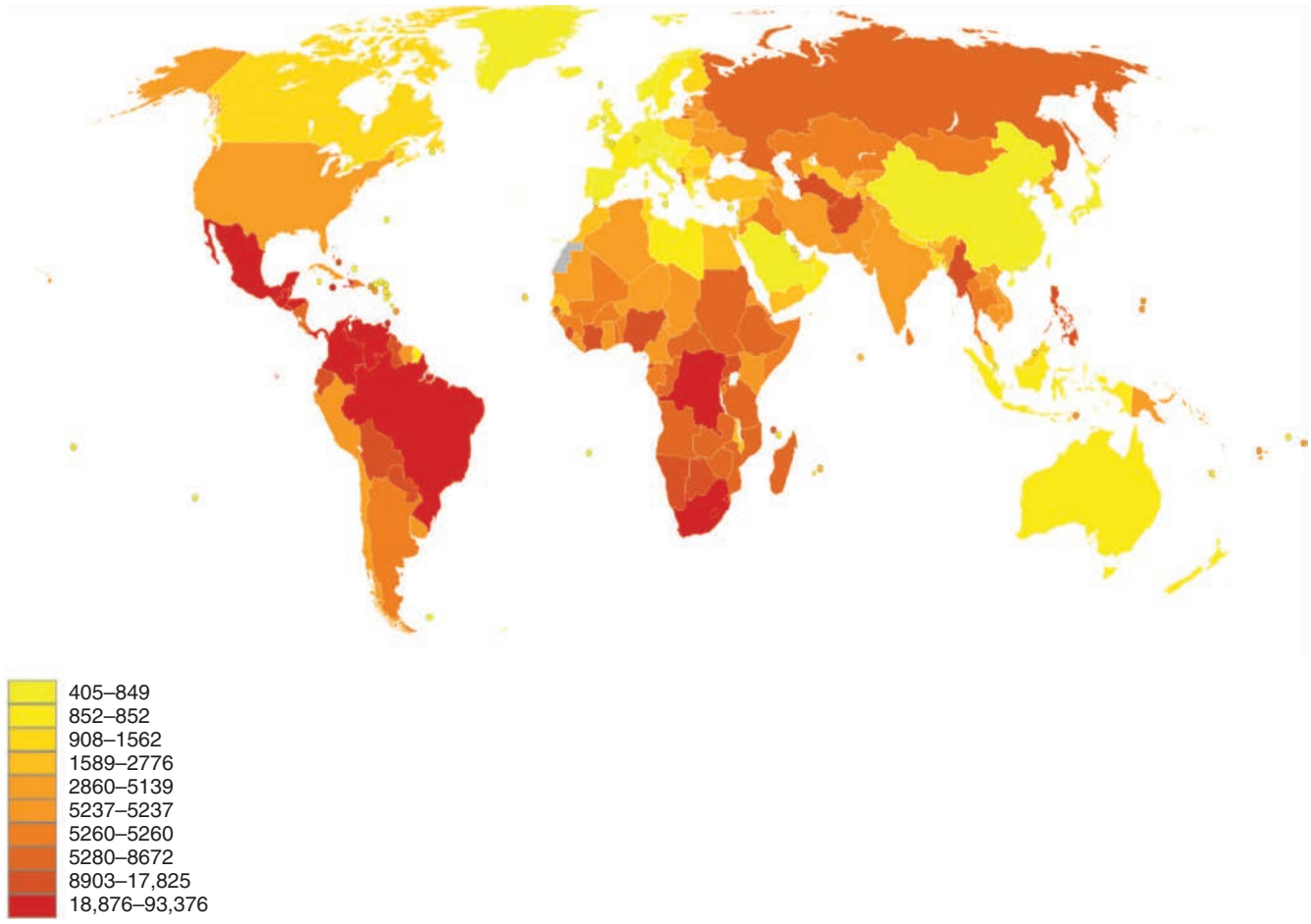

FIGURE 64-1 Disability-adjusted life-years (DALYs) lost from interpersonal violence in 2012 per million males.

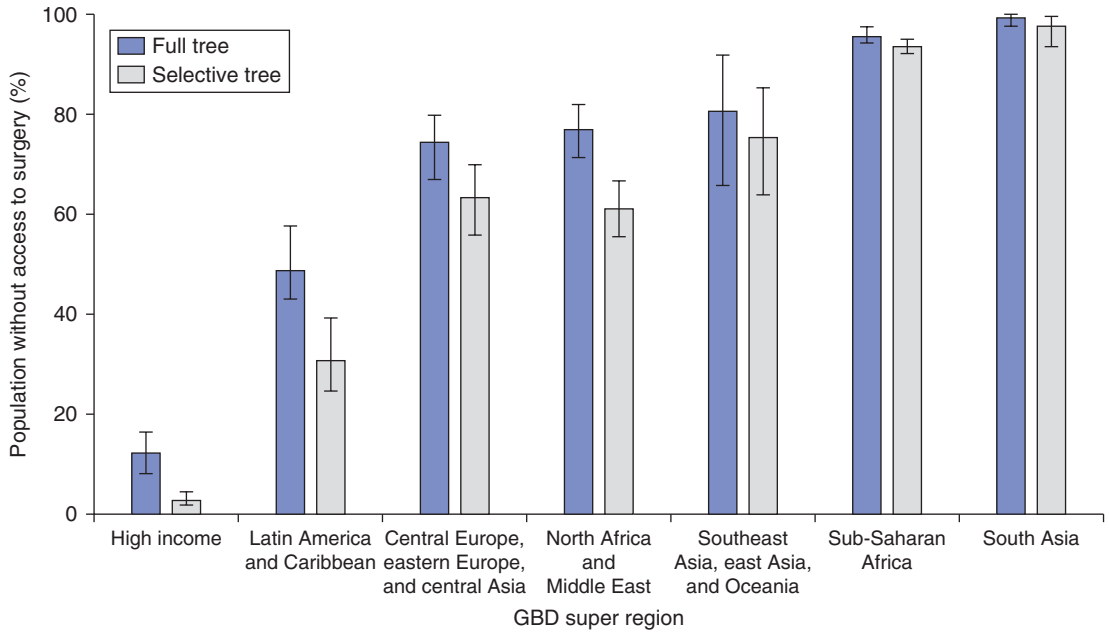

FIGURE 64-2 Over 90% of the populations of sub-Saharan Africa and South Asia do not have access to surgery when taking into account availability, timeliness, safety, and affordability. (Reproduced from Alkire BC, Raykar NP, Shrime MG, et al. Global access to surgical care: a modelling study. *Lancet Glob Health*. 2015;3(6):e316-e323.)

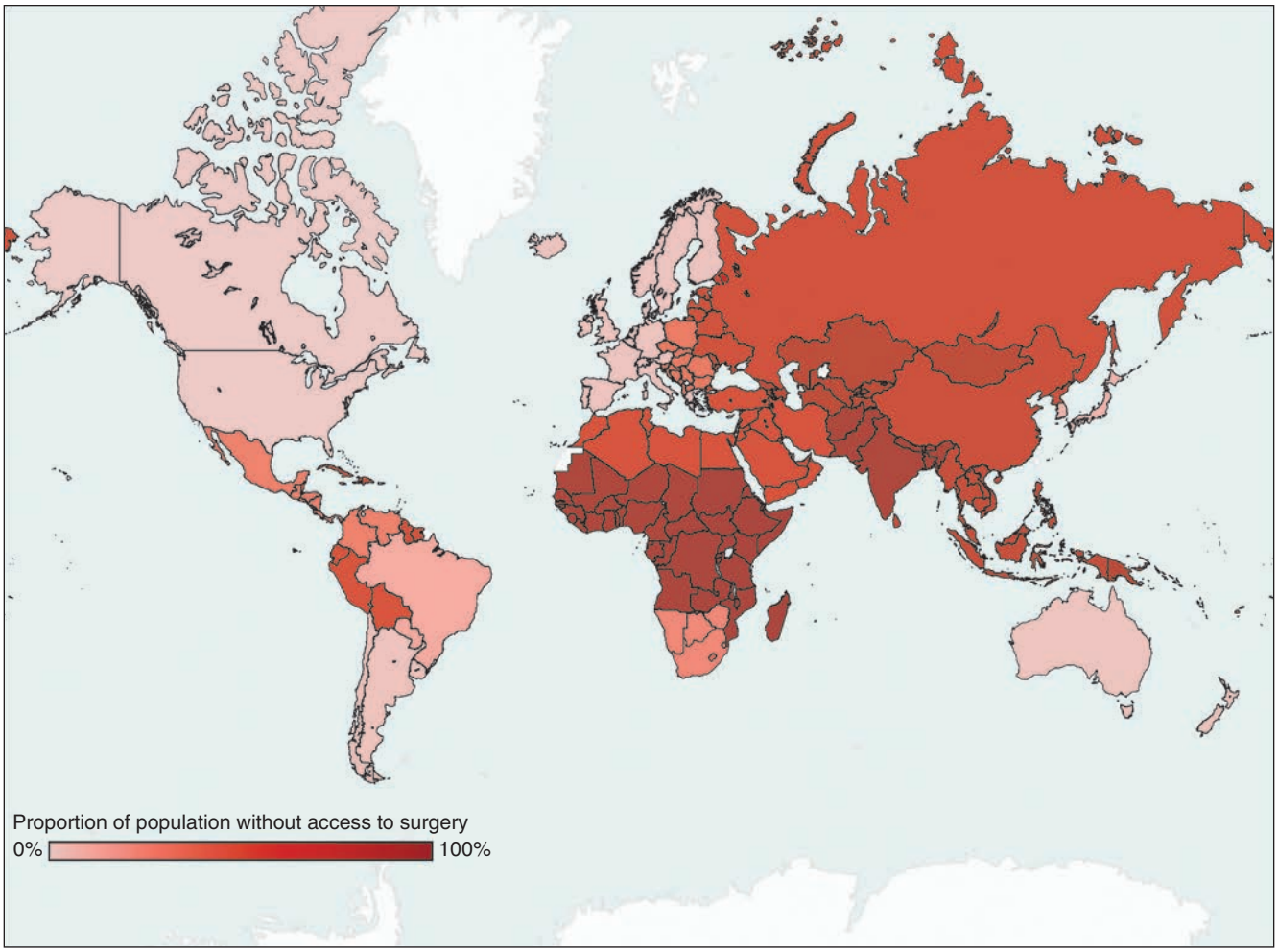

FIGURE 64-3 Unfortunately, the areas of poorest access often overlap with areas of greatest rates of trauma and violence, as depicted in Fig. 64-1. (Reproduced from Alkire BC, Raykar NP, Shrime MG, et al. Global access to surgical care: a modelling study. *Lancet Glob Health*. 2015;3(6):e316-e323.)

staff; and unaffordable costs.[9,25] A quarter of those who successfully obtain surgical care are bankrupted by it.[10]

Accompanying these deficits has been the lack of policy focus. Paul Farmer and Jim Kim famously described surgery as the "neglected stepchild" of the global health community. This alluded to decades of international efforts from the WHO, World Bank, NGOs, and HIC governments prioritizing funds for nonsurgical treatments for AIDS, tuberculosis, malaria, dehydration, and malnutrition.[26] In the United States, the National Academy of Sciences, part of the National Academies of Sciences, Engineering, and Medicine (NASEM), plays a major role in shaping US policy in global health and, for the better part of this century, considered addressing the surgical burden of disease a low priority. As of 2014, less than half of national health strategic plans in sub-Saharan Africa had any mention of trauma, and only 2% of mentioned health targets related to surgical conditions or surgical care.[27] Corruption, violence, and war also threaten the stability of the poorest regions of the world, resulting in the destruction of any existing health system and impeding public health interventions.[28]

The Rise of Global Surgery

Fortunately, there have been recent strides in the prioritization of surgery within the global health landscape. In early 2015, the World Bank released its third iteration of the Disease Control Priorities, dedicating, for the first time, an entire volume to surgical diseases (previous versions in 1999 and 2006 either made no mention or consisted of a single chapter).[29] Also, in 2015, the Lancet Commission on Global Surgery released its seminal report, culminating a 2-year process of data gathering and priority setting involving over 500 experts in 100 countries. There was critical involvement from the World Bank, WHO, and multiple ministries of health.[10] Both the World Bank and WHO revised their core data collection requests from national governments to include surgical indicators from the Lancet Commission's

report. The latest NASEM report, published in May 2017, addresses matters pertaining directly or indirectly to trauma and surgery in 4 of its 14 core recommendations.[30] In 2007, WHO recognized the importance of trauma and emergency care systems with World Health Assembly Resolution 60.22, and in 2015, the World Health Assembly passed Resolution 68.15 calling for emergency surgical care as a part of universal health coverage.[31]

Several national governments have already taken heed of the global drive to strengthen health systems and include surgery. Zambia became the first to undergo a formal national surgical planning process, establishing a National Surgical, Obstetric, and Anesthesia Plan into its national strategic health plan for 2017 to 2021.[32] Ethiopia and Mongolia have embarked on similar processes.[33] Advocates of the rural poor have combined forces with academics, private industry, NGOs, and government officials to drive a grassroots process in India.[34]

THE ROLE OF THE ACUTE CARE SURGEON

Trauma surgeons have had a longstanding record of public health engagement. Never before have the opportunities for the trauma and acute care surgeon interested in global health coincided with the global momentum for surgery. The very nature of combining surgical expertise with the provision of emergency care places the acute care surgeon at center stage to promote appropriate improvements in injury prevention, trauma and emergency care, development of information systems, and workforce training. In fact, continued improvement will require coordinated action led by acute care surgeons in the clinical, training, research, and policy partnerships.

Clinical Partnerships

The massive unmet need for surgery will require a coordinated approach that focuses on enhancing capacity for delivery of surgical care in hospitals and communities worldwide. Hospitals in the health system are usually categorized into first-level (district), second-level (regional), and third-level (tertiary) hospitals. The first level is closest to the communities it serves and is the entry point for the critically ill within the broader health system.[35] Broad consensus exists in the global health community that a functional first-level hospital should be the base for a strengthened surgical system.[36]

The Lancet Commission on Global Surgery has adopted these recommendations and further suggests local clinicians and surgeons stratify priorities for surgical scale into categories of "must do," "should do," and "can do." In fact, it defines abdominal laparotomy, cesarean delivery, and treatment of open fracture as the bellwether procedures, markers of a hospital's surgical functionality.[37] Local clinicians and policymakers and their international partners should focus on context-specific improvements to provide year-round coverage for "must do" and "should do" procedures. In certain hospitals, this may focus on securing a reliable energy source or running water. In other localities, attracting and retaining expert talent including nurses and pharmacists may take top priority. In yet other settings, focus may shift to developing protocols and guidelines dictated by local experts and applicable to the local resources to maximize efficiency in triage or treatment. The specific improvements and priority setting should be left to clinicians and policymakers with a nuanced understanding of local context and disease burden in the community.

Acute care surgeons interested in partnering in these efforts should seek structured, longitudinal collaborations, built through both individual relationships and leveraging institutional resources on both sides. The interest in "surgical volunteerism"—short-term engagements, a staple model for global surgery engagement in the past—should be channeled into structured engagements focused on providing dependable, year-round (long-term) support. Short-term camp-based engagements may continue to have a role, particularly for more specialized, elective "can do" procedures, but the need for and timing of these spurts in surgical activity should be guided by local clinicians so they do not distract from the broader mission of reliably functional first-level hospitals.[38]

Training Partnerships

Educational and training partnerships can present opportunities to enhance provision of care at the first-level hospital with bilateral benefit.[39] International experiences are common among medical trainees worldwide, and recent US medical school curricula have been adapted to include global health and cultural competency.[40] Departments of surgery, however, lag behind other disciplines in offering international experiences for their trainees, with only 34% of US surgical residency programs offering such opportunities, and often they require the use of vacation time.[41] Likewise, acute care surgery programs in the United States are finding increasing difficulty in securing adequate exposure for trainees in penetrating operative trauma. Some programs are actively seeking opportunities for their fellows to travel and participate in clinical rotations where they can be exposed to challenging penetrating caseloads.

Novel partnerships may provide supervised case exposure in low-resource settings for surgical trainees from higher resource settings while coupled with level-appropriate clinical service by advanced trainees or partner faculty.[42] A board-certified American College of Surgeons (ACS) fellow could conceivably provide general surgery support at a first-level institution while gaining operative trauma experience at the trauma center. The Residency Review Committee and the American Board of Surgery (ABS) approved international rotations to fulfill graduation requirements for surgical residents in 2011.[43,44] This approval is contingent on specific requirements, such as Internet access, literary resources, and the supervision of an ABS-certified surgeon. Unfortunately, the regions of the world that will benefit the most from these cultural and educational exchanges are the very regions

that lack the presence of ABS-certified surgeons. Innovative thinking is needed to balance these realities with the need to ensure trainees continue in a safe training environment and are not extended beyond their level of training without adequate supervision. Global health has a complicated history that includes accusations of colonialism. The onus will be on training programs from higher resource partners to ensure trainee presence does not supplant local trainee opportunities or cause disruption without accountability to local partners.[45]

IMPORTANCE OF RECIPROCITY

Opening the doors for surgeons and trainees from the United States to travel to and operate in busy trauma centers in LMICs does require fulfilling several regulatory steps including special permissions and liability protection. In most of these centers, the visiting surgeons and trainees will not be asked to validate their training, although they may need to obtain temporary licenses, often under the purview of the host institution. Regardless, almost universally, they are able to participate in operations and play a pivotal role in the decision making required to provide comprehensive care. Unfortunately, a truly reciprocal experience cannot be offered to foreign medical graduates (surgeons) when invited to visit trauma centers in the United States. Foreign medical graduates specialized in surgery outside the United States and Canada must redo their complete surgical residency in the United States to be able to practice as surgeons, and their interactions in North American trauma centers are subject to regulations from the ABS and state and federal licensing boards.

As international educators whose trainees benefit tremendously from their experiences overseas, we must commit to creating mechanisms to offer valuable experiences for surgeons trained overseas who are invited to the United States. The underlying reason to invite trauma surgeons or residents from LMICs to visit trauma centers in the United States is to provide exposure to mature, academic trauma systems. As long as visitors are supervised and not expected to engage in independent decision making, they should be allowed to participate in clinical rounds and assist in the operating room. Most importantly, they should be provided immersive experiences in the multidisciplinary academic, investigative, and performance improvement efforts that distinguish American academic trauma centers.

Needless to say, most will recognize that the mere technical experience of assisting in a surgical case in isolation from the broader system employed to take care of these patients will be an incomplete strategy.

THE NEED FOR INSTITUTIONAL PARTNERSHIPS IN MEDICAL EDUCATION AND THE ROLE OF PROFESSIONAL SOCIETIES IN SUB-SAHARAN AFRICA

Medical school enrollments are increasing across the African continent. Kenya, for example, had two medical schools for its first 50 years after independence, but the last decade has seen seven more, a 350% increase. Concurrently, the class size for these medical schools has risen annually, with increases of 200% to 300%.[46] The average medical school in Kenya enrolls 80 to 350 students per year.

While this is an encouraging development for a region in dire need of medical professionals, it presents challenges to the medical and surgical community. How will they provide a quality education and postgraduate training with limited faculty already stretched to provide clinical care and administrative duties?

University-to-university collaborations are primed to meet some of these educational challenges.[47,48] Broad collaborations signed at the highest levels between institution leadership can cut across disciplines and provide a steady pool of visiting faculty and leverage resources from online resources to course curricula. Lasting partnerships, after all, mandate that the mechanisms for faculty to participate at partner sites are built into their job descriptions and do not rely solely on volunteerism.

The College of Surgeons of East, Central, and Southern Africa (COSECSA) was formed in 1999 to provide a standard training pathway and internationally recognized surgical qualification, currently in existence across 12 countries in sub-Saharan Africa. COSECSA has graduated over 250 surgeons since 2004 and currently has 575 trainees enrolled across the region. Beyond postgraduate training to scale up the African surgical workforce, COSECSA has also developed initiatives in faculty development, research and peer-reviewed publication, and policy formation in conjunction with national medical boards.[49]

Global partnerships have been critical to COSECSA's successes. The Association of Surgeons of Great Britain and Ireland collaborated with COSECSA for more than 10 years to provide multidisciplinary team training in basic surgical skills and management of surgical emergencies.[50] The Pan-African Association of Christian Surgeons partners with COSECSA, allowing its constituent surgeons and hospitals under faith-based organizations to participate in regional surgical training and leadership while contributing critical additional trainers and training sites.[51] Since 2007, the Royal College of Surgeons of Ireland (RCSI) has collaborated with COSECSA to design and administer examinations (along with a global cohort of expert surgeon-examiners), develop e-learning and logbook resources, and manage administration and business of the college. The RCSI-COSECSA partnership has also cooperatively collected and published data on the surgical needs of the region.[52,53] The ACS has also initiated a scholarship program to support the training of women surgeons through COSECSA.[54]

Research Partnerships

Clinicians in the frontlines often lament that they are both literally and figuratively "operating in the dark." Faced with overwhelming patient loads and working in remote hospitals, providers and administrators rarely receive feedback to assess benchmarks or drive process improvement. Unfortunately, not only is the data void demoralizing, but it also leads to ineffectual choices with investment for scale-up.

Well-intentioned initiatives based on successes in one part of the world do not necessarily transfer well. Ambulance systems, for example, a hallmark of high-income prehospital trauma systems, have failed to improve outcomes in lower-resource settings where injury patterns, traffic and population density, and hospital capabilities may differ.[55]

In fact, organized trauma systems require robust data collection (trauma registries) for optimization of the system. The establishment of the ACS National Trauma Data Bank in the United States continues to enhance trauma care and direct resources toward high-efficiency interventions. Benchmarking trauma systems in the low-resource world will require novel methodology for data collection and new scales to identify system deficiencies.[56] Unfortunately, policymakers and clinicians in most low-resource environments are unable to direct significant investment toward data/informatic systems.

Academic partnerships capitalizing on the expertise of high-income universities in creating, financing, and sustaining trauma registries with electronic injury surveillance applications may provide a hard reset. Such research relationships may also serve as an avenue for reciprocity in global health, leveraging the strong traditions of academic medicine in higher resourced settings with the strong clinical expertise of surgeons from LMICs. As more surgeons-in-training gain valuable surgical experience in low-resource countries, mechanisms must be developed to provide a commensurate experience in high-income institutions for partner surgeons from low-resource settings. Providing immersive learning opportunities in research and quality improvement, with degree opportunities and full access to learning materials, research groups, and faculty typical of academic centers in the United States and Europe, may not only provide benefit to the visiting physician, but must also serve as a longer term investment in research infrastructure in LMICs.

Unfortunately, funding for international trauma research has traditionally been scarce. Several academic surgical departments in the United States have used clinical revenue to support a diversity of international endeavors, particularly at the level of midterm surgical residents or junior faculty.

A few developments, however, offer hope. Since its inception in 1968, the National Institute of Health's international health coordinating entity, the Fogarty International Center, has promoted international collaborations built in the United States by funding research training grants and capacity building in international/global health. The Fogarty International Collaborative Trauma and Injury Research Training Program was designed in 2005 and awarded in 2006 to foment relationships between universities in the United States and medical schools in LMICs, which are reissued in 5-year cycles.[7] Similar programs have been established in Europe and Australia, with direct eligibility linked to research in specific partner countries. The Australia–India Trauma Systems Collaboration is a multiyear partnership funded by a diversity of donors with aims to improve trauma systems through data systems in both countries.[57]

With newfound recognition of the importance of this research within the global health community, investigators will need to employ creativity in responding to dissimilar but related funding requests for applications, in government and nongovernmental sectors, and create new opportunities through advocacy.

CASE EXAMPLE OF CAPACITY BUILDING FOR TRAUMA RESEARCH: FOGARTY INTERNATIONAL CENTER, D43 TRAINING GRANTS

The University of Pittsburgh was awarded a D43 training grant in 2006 in collaboration with the Universidad del Valle in Cali, Colombia. Under this grant, Colombian trainees engage in research training at the University of Pittsburgh. They are offered several short-term research capacity rotations, as well as long-term training in master's and PhD programs in clinical research and clinical translational sciences. This academic training has been pivotal in creating a new culture of trauma research in Colombia, a country where trauma surgeons had developed astonishing clinical proficiency but did not have the opportunity to examine this experience or submit it in a rigorous academic format required for international scientific forums.

Since inception of the program, Colombian trauma research participation has surged at the American Association for the Surgery of Trauma, the Clinical Congress of the American College of Surgeons, and the European Society of Trauma and Emergency Surgery. Colombian trauma surgeons have become independent clinical investigators and regional leaders, collaborating with faculty from other countries in the region including Guatemala and Paraguay. Studies have been diverse and varied, ranging from clinical trials to epidemiologic analyses of alcohol policies and homicides rates.[58-60] The success of this program relies on what we recognize as the "P" values of successful international collaborations. These are strong **p**artnerships that must be based on mutual collaboration. It requires in-depth knowledge of the **p**eople, local cultural characteristics, and cultural idiosyncrasies. It requires **p**atience and di**p**lomacy to build **p**roductive and successful long-lasting research programs. These collaborations must be multidisciplinary by nature among a variety of health professionals from both HICs and LMICs to result in significant and sustainable trauma research contributions relevant to LMICs and beyond.

Policy Partnerships

Advancing surgery among national political priorities will continue to be a key task for those interested in strengthening surgical systems. Funding, resources, and political prioritization required for true systems-level change cannot occur in isolation within clinical and academic partnerships; it requires true engagement of the health system and its decision makers.

Many of the recent examples of national surgical planning (Zambia, India) have been shepherded through academic partnerships in the aftermath of the Lancet Commission on Global Surgery, itself a collaboration of many societies across

the globe.[33] International partners can often provide much-needed research and organizational support to efforts driven by local partners and priorities.

International trauma societies in high- and low-resource settings have already taken up the mantle of global collaboration with the purpose of addressing needs in surgical workforce, research development, and emergency surgical care. The West African College of Surgeons has been in existence since the 1960s, and COSECSA, described previously, provides a standard training pathway and internationally recognized surgical qualification for many countries across Africa. Both societies have collaborated extensively with organizations in the United States and Europe to provide faculty development, and particularly to provide training in clinical research knowledge and skills.[61] Encompassing many of the tenets of acute care and trauma surgery, the Association of Rural Surgeons of India has been an advocate for the rural poor since 1992 and in 2005 formed the International Federation of Rural Surgery in partnership with the Association of Rural Surgeons of Tanzania, the Association of Rural Surgical Practitioners of Nigeria, and the German Society for Tropical Surgery.[62,63] This organizational collaboration not only provides critical professional connectivity to rural acute care, but also has shaped policy in areas such as legislation for blood banking in India and pioneered low-cost surgical innovations such as the use of mosquito nets for use in hernia repair.[64]

The last few years have seen new partnerships between the American Association for the Surgery of Trauma and the European Society for Trauma and Emergency Surgery and other international organizations such as the International Association for Trauma and Intensive Care and the Panamerican Trauma Society. Operation Giving Back was initiated by the ACS in 2004 as an effort to coordinate surgical volunteerism. It now has evolved into an organization supporting broader outreach and working closely with WHO and surgical professional societies in Africa and Asia. Specific to trauma, the ACS's Committee on Trauma has created the International Injury Care Committee (I2C2), uniquely positioned to leverage historically successful endeavors in trauma systems, team training (Advance Trauma Life Support [ATLS]), rural trauma, and quality improvement in the United States with worldwide dissemination. I2C2 should now focus on developing an ATLS-like program to offer basic educational material for the creation of low-cost, effective trauma systems adapted for low-resource settings.

The private and nongovernmental sectors have been key in this process. The Bloomberg Group Philanthropies, for example, have partnered with WHO and other groups to create a Global Health Advocacy Incubator.[65] These initiatives have resulted in 11 countries passing new or improved road safety laws, thus protecting over 3.36 billion people. From 2007 to 2009, Bloomberg Philanthropies funded a pilot program in Cambodia, Mexico, and Vietnam to see if proven road safety interventions could be adapted and used on a global scale. This effort was expanded in 2010 to support the implementation of these interventions and successfully reduce road traffic fatalities and injuries in 10 LMICs that account for half of the global road crash fatalities (Brazil, Cambodia, China, Egypt, India, Kenya, Mexico, Russia, Turkey, and Vietnam). In 2015, Bloomberg Philanthropies launched phase 2 of the Initiative for Global Road Safety, which will address road traffic safety in 10 cities (Accra, Ghana; Addis Ababa, Ethiopia; Bandung, Indonesia; Bangkok, Thailand; Bogota, Colombia; Fortaleza and Sao Paolo, Brazil; Ho Chi Minh City, Vietnam; Mumbai, India; and Shanghai, China), five countries (China, India, Philippines, Tanzania, and Thailand), and four vehicle market regions (Africa, Latin America, India, and Southeast Asia) with the primary goal of reducing road traffic fatalities and injuries.

CONCLUSION

There has never been a better time to be a surgeon interested in global surgery. Acute care surgeons can make meaningful strides extending surgical access for the world's poor through innovative clinical, policy, and academic partnerships. It is imperative that surgical department chairs and professional society leadership embrace and encourage participation in these efforts.

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VI

ATLAS OF TRAUMA

Introduction to the Atlas

Each preceding chapter has figures, tables, and drawings that aid the author and the editors in imparting and clarifying the messages of the chapter. In this edition of *Trauma,* the editors have devoted a special section to an atlas. It is not intended to include an exhaustive artistic rendering of every operation, but, rather, to focus on procedures that are commonly used in major trauma operations, along with related anatomical drawings. Some procedures that may have been commonly practiced in the past but are rarely currently done are not included. However, a few relatively infrequently used concepts and procedures have been included in this section because of sufficient need to clarify the opinions of the editors. The art has been kept in as simple a form as possible, so it is expeditiously available when needed for a quick refresher in anatomy, anatomical relationships, and/or surgical approach in the "heat of battle."

HEAD AND NECK

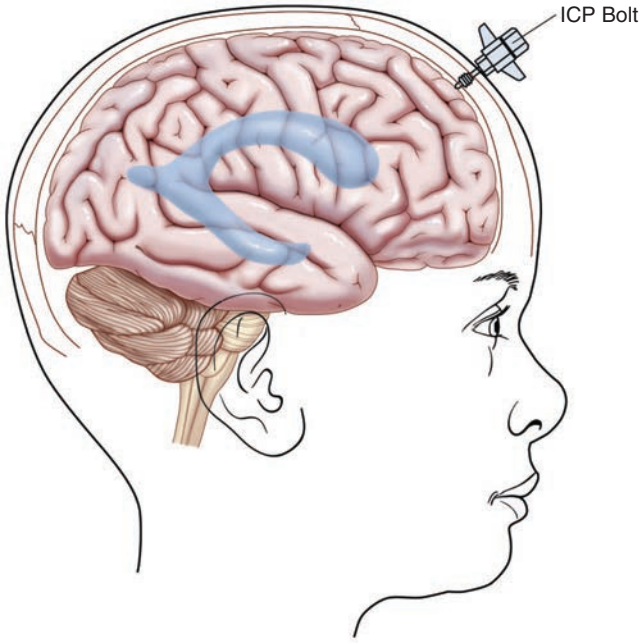

FIGURE 1 ICP Lateral
From a lateral perspective, this shows an ICP bolt inserted into the space around the brain via the skull bone to facilitate continuous ICP monitoring.

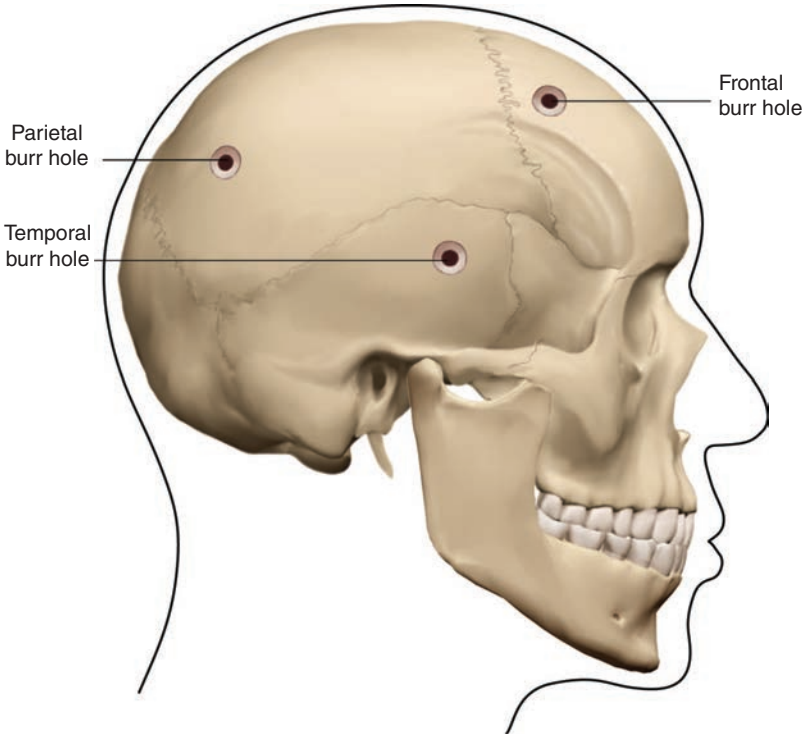

FIGURE 2 Burr Hole
The ideal location of a cranial burr hole depends on the anticipated injury in the three skull bones.

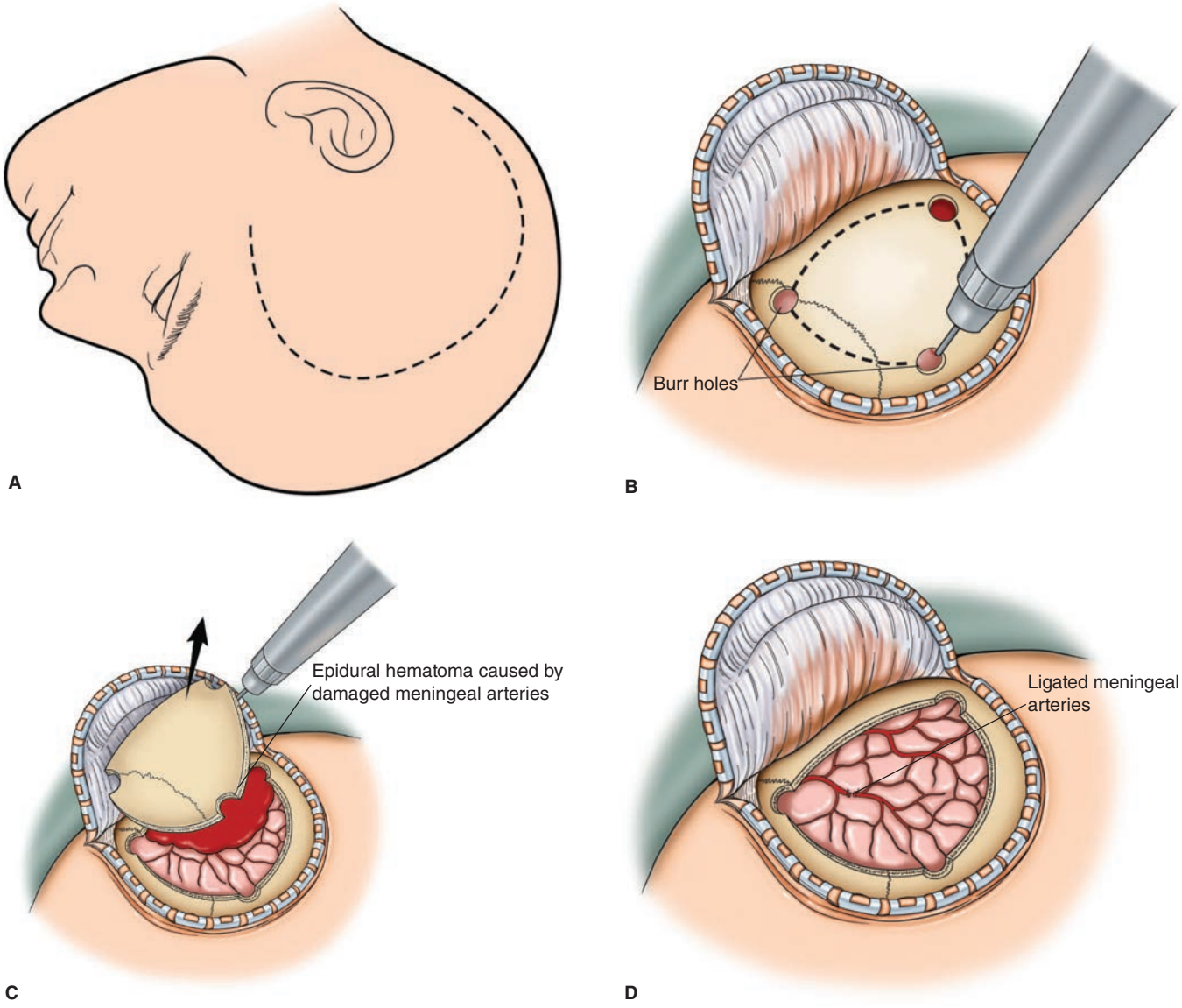

FIGURE 3 Steps in Performing a Lateral Parietal Craniotomy

A. The exact location and size of the skin flap vary, depending on extent of the wound, but must not extend to the midline at the top of the skull.

B. Skin clips are placed for hemostasis, and burr holes elevate the skull bone flap.

C–D. The bone flap is removed, the dura mater is opened to expose and release an epidural hematoma, and bleeding vessels are ligated. In the absence of significant brain swelling, the skull plate is reattached once hemorrhage is controlled and other necessary procedures have been accomplished. With significant brain swelling, the dura is closed, sometimes using dural substitutes, and the bone flap is not replaced at initial operation (decompressive craniotomy).

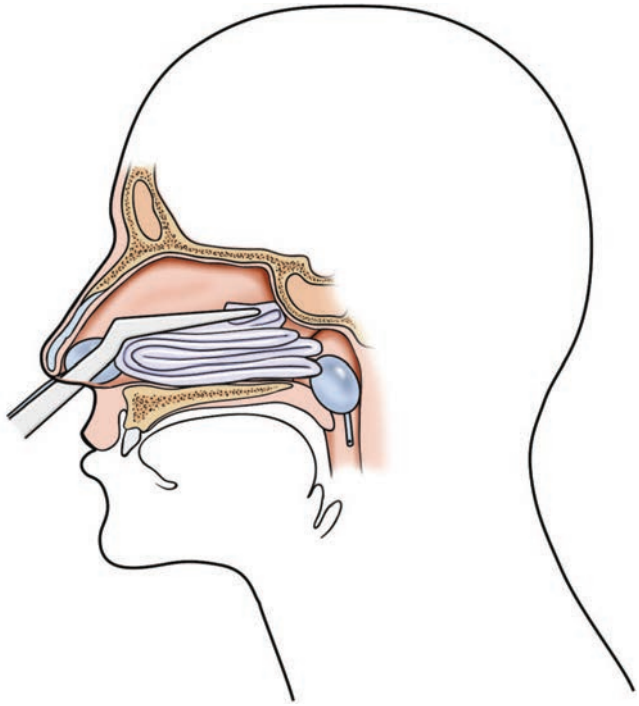

FIGURE 4 Nasal Packing for Hemorrhage Control
Under general or topical anesthesia, gauze impregnated with Vaseline to facilitate insertion is layered into a bleeding nasal passage to achieve hemostasis. Balloon devices are commercially available to provide posterior and anterior nasal packing.

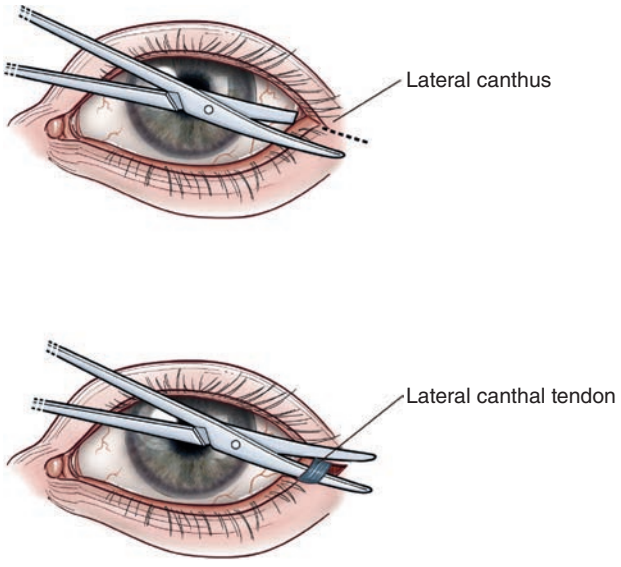

FIGURE 5 Lateral Canthotomy
This drawing of the eye demonstrates the location for creation of a lateral canthotomy in the eye covering. Note the lateral ligament, which may be cut with ocular scissors.

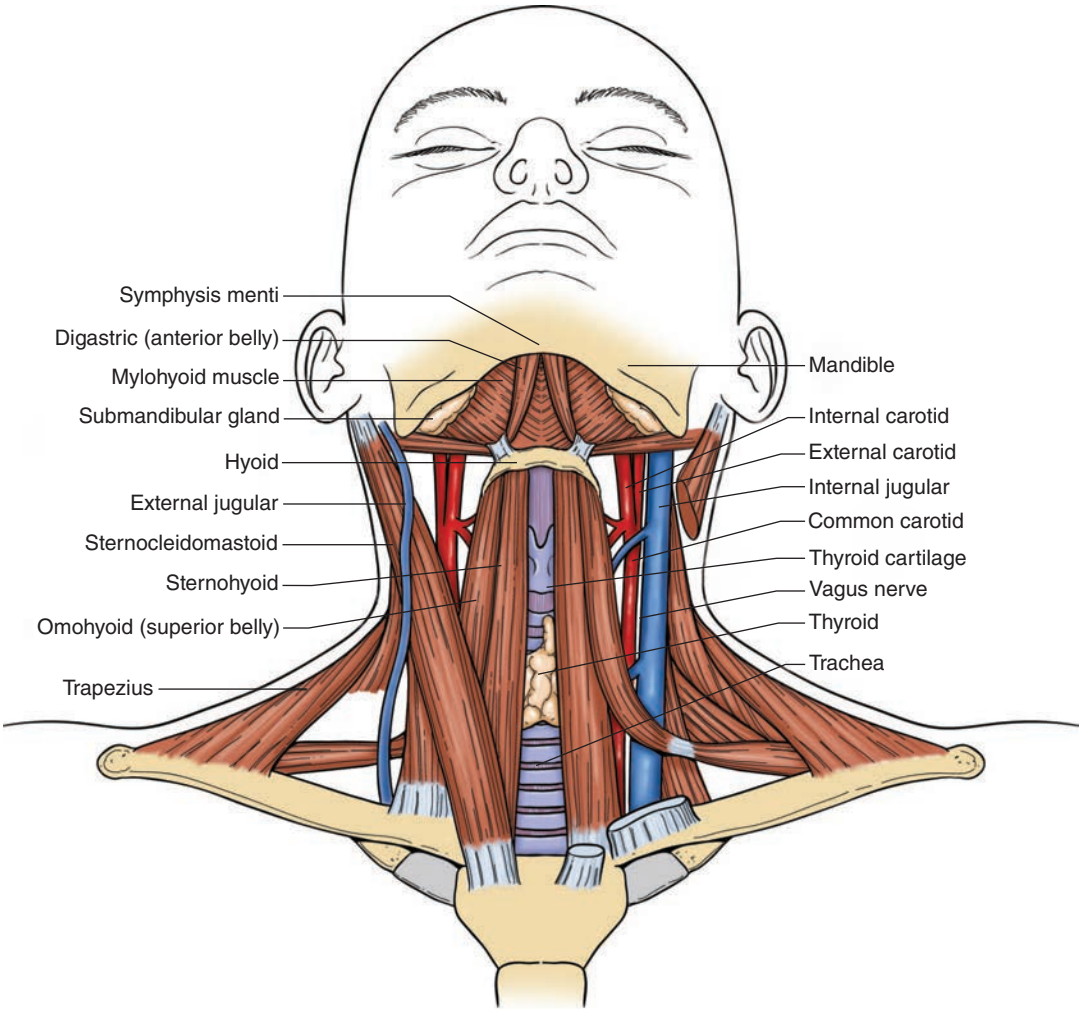

FIGURE 6 Anatomy of the Neck
Anterior perspective—Although usually approached from incisions just anterior to the sternocleidomastoid muscle, the surgeon must always review the cervical anatomy and its structural relationships prior to incision. The external jugular vein is a subcutaneous structure, and the internal jugular vein and carotid arteries are deep and medial in the neck.

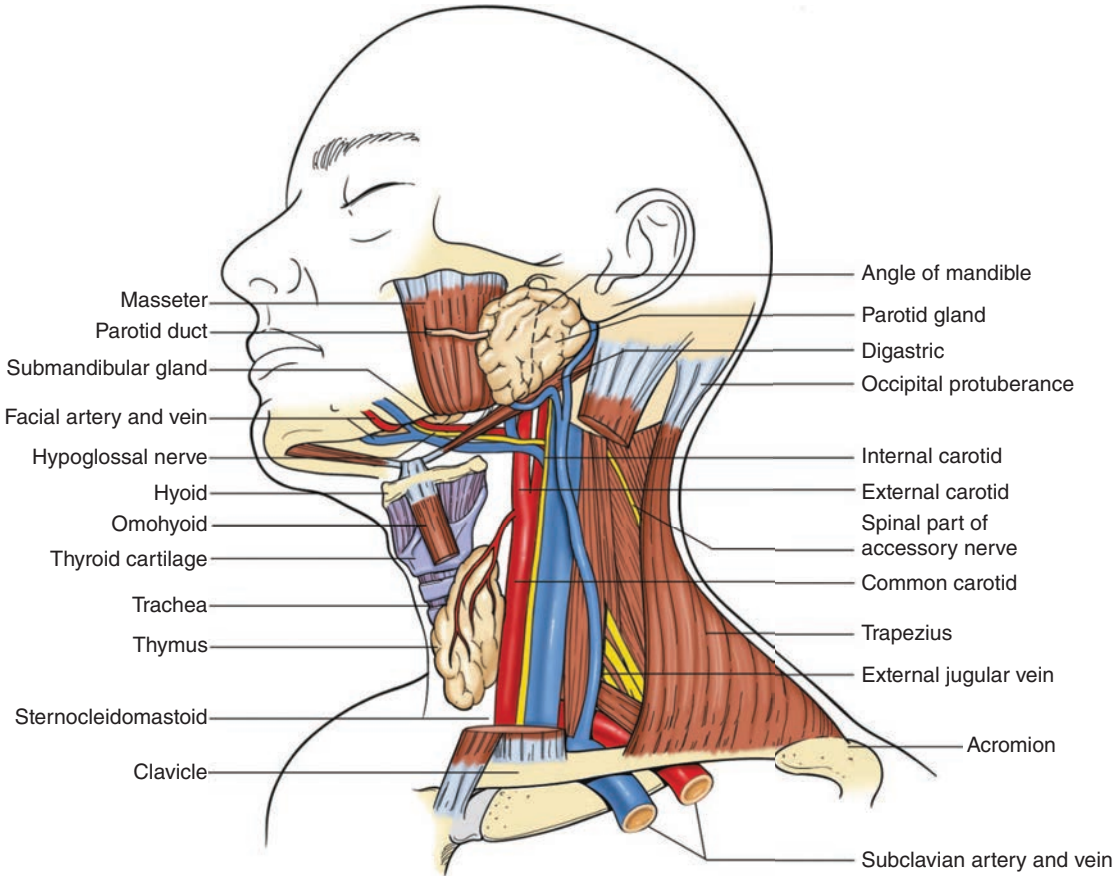

FIGURE 7 Lateral Anatomy of the Neck
This is the anatomy of the neck as seen from a left lateral view. Note the location and course of the facial vein, the division of which is the key to exposing the mid to upper structures in the deep neck.

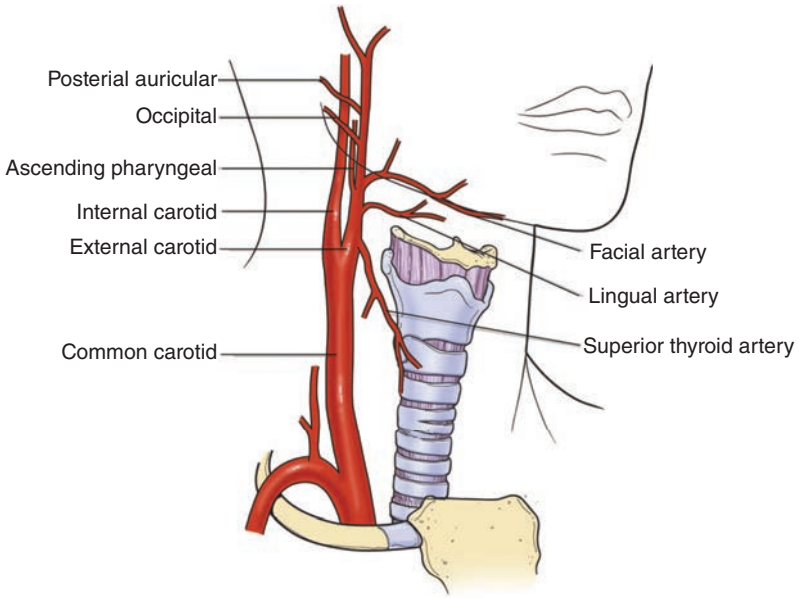

FIGURE 8 Carotid Artery
There are six branches of the external carotid artery. The right carotid artery is the mirror image of the left. Note that the internal carotid artery has no extracranial branches and is always lateral to the external carotid artery. The height of the bifurcation of the common carotid artery in the neck is variable. The facial vein, which can be divided between clamps with impunity, is not shown in this drawing. This division of the facial vein opens the deep anatomy of the mid lateral neck in the area of the bifurcation of the common carotid artery.

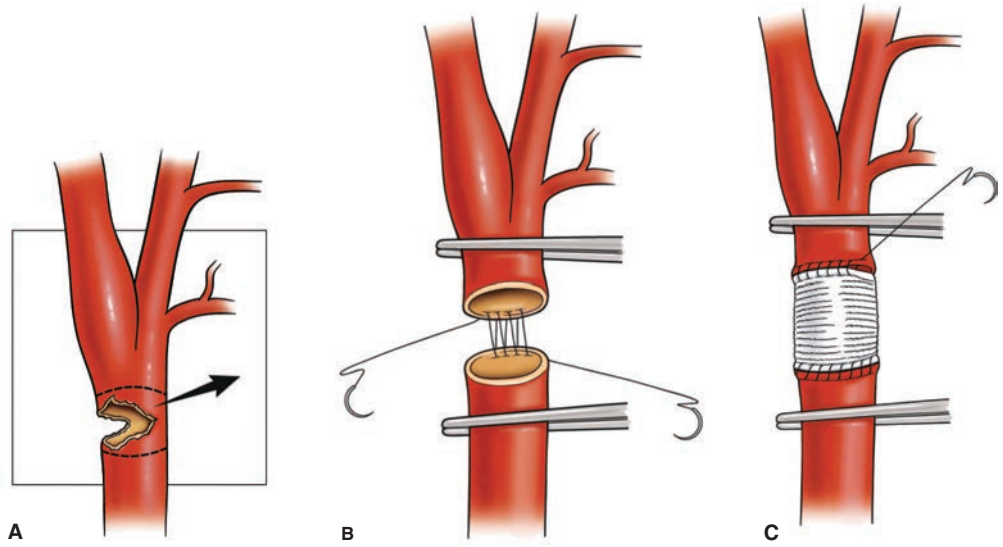

FIGURE 9 Repair Techniques for Injury to the Common Carotid Artery Proximal to the Bifurcation

A. The injured vessel is debrided after proximal and distal control is obtained. Local heparinized saline is injected distally.

B. If the injury is not extensive, end-to-end anastomosis with 5(0) polypropylene running suture using a "parachuting" technique is possible. Note, with adequate arterial collateral circulation, a temporary carotid artery shunt is not required.

C. If the artery is going to be under tension or stretched, an interposition graft of Dacron (shown), polytetrafluoroethylene (PTFE), or scavenged vein is inserted.

Prior to completely releasing the clamps and before prograde flow is reestablished to the brain, any microclots that might have formed during the procedure are adequately flushed out, both proximally and distally, then flow is directed into the external carotid artery, and, lastly, flow is directed into the internal carotid artery.

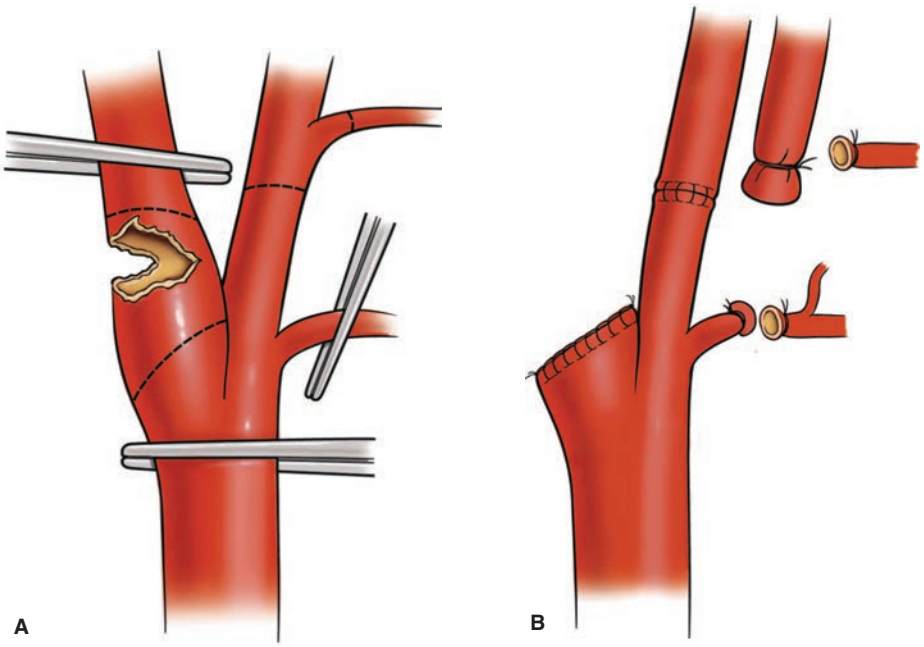

FIGURE 10 Transposition Technique for an Injury to the Proximal Internal Carotid Artery

A. Following proximal and distal control and instillation of local heparinized saline, the area of injury is resected, and the external carotid artery is mobilized for a distance sufficient to bridge the gap for the injured internal carotid artery.

B. The origin of the proximal internal carotid artery is oversewn, and one anastomosis is accomplished using the mobilized external carotid artery. Ligation of one or two branches of the external carotid artery may be required to accomplish significant mobilization.

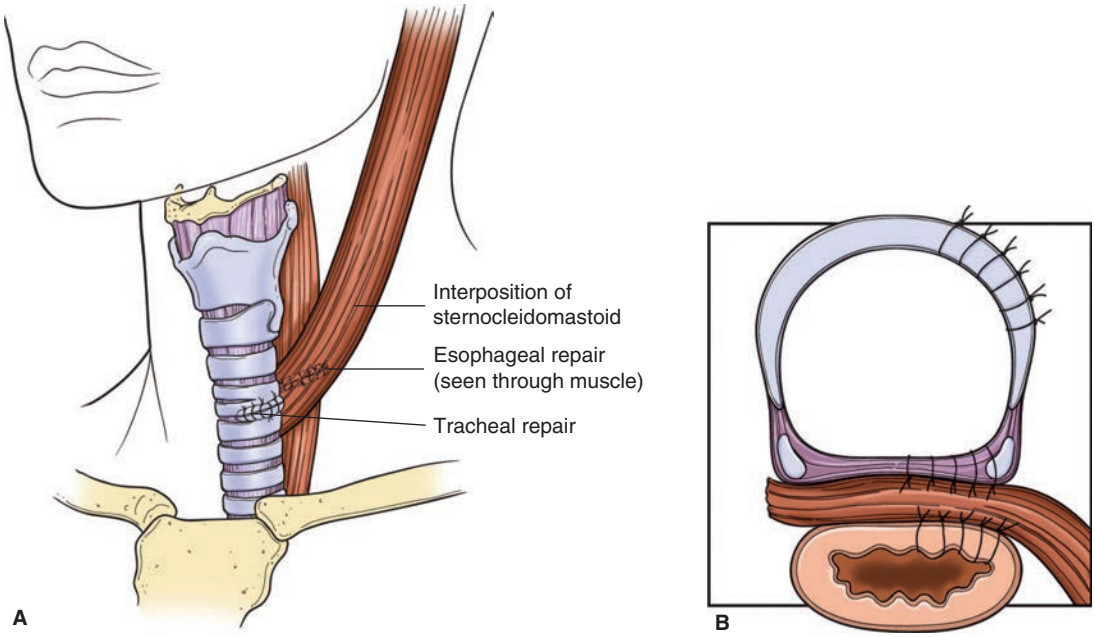

FIGURE 11 Repair of a Combined Injury to the Trachea and Esophagus

A. Following repair using interrupted absorbable sutures on the trachea and esophagus, a vascularized muscle pedicle (such as the sternal head of the sternocleidomastoid muscle) is interposed between these two tubular structures to reduce the postrepair complication of fistula formation.

B. The procedure demonstrated in cross section.

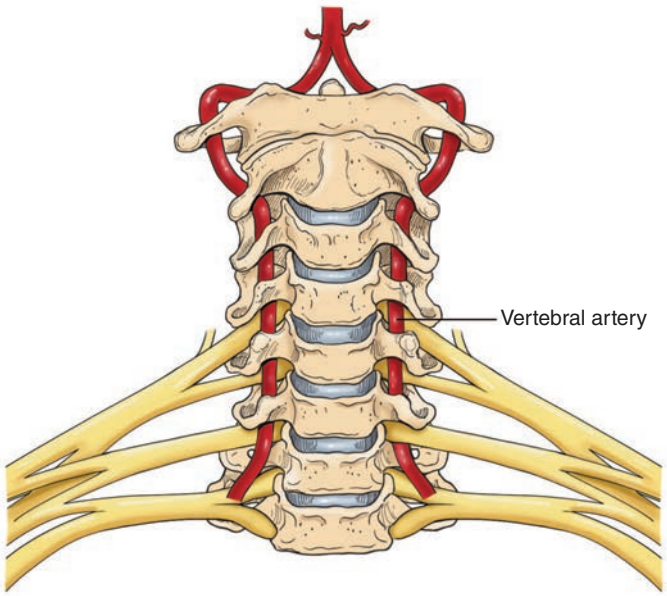

FIGURE 12 Vertebral Artery

This demonstrates the bilateral vertebral arteries arising as the first branch off the corresponding subclavian artery in the neck, entering the transverse foramina of the sixth cervical vertebra, and exiting the transverse foramina of the second cervical vertebra, to join the opposite vertebral artery to form the basilar artery. Note that all portions of the vertebral arteries are anterior to the cervical nerves.

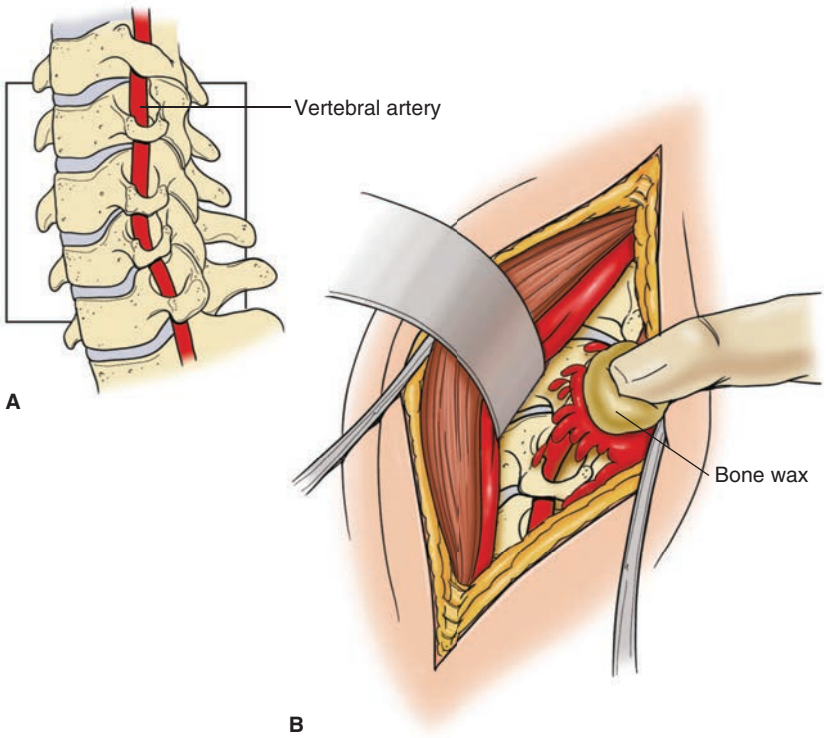

FIGURE 13 Control of Injured Vertebral Artery

A. The vertebral artery lies deep in the neck inside the transverse foramina of the cervical vertebrae. For uncontrolled bleeding from an injured vertebral artery within the transverse foramina of the neck, dissection and unroofing of this bony covering can be difficult and even produce additional injury and complications.

B. Bone wax pressed into the area of injury can rapidly control persistent bleeding.

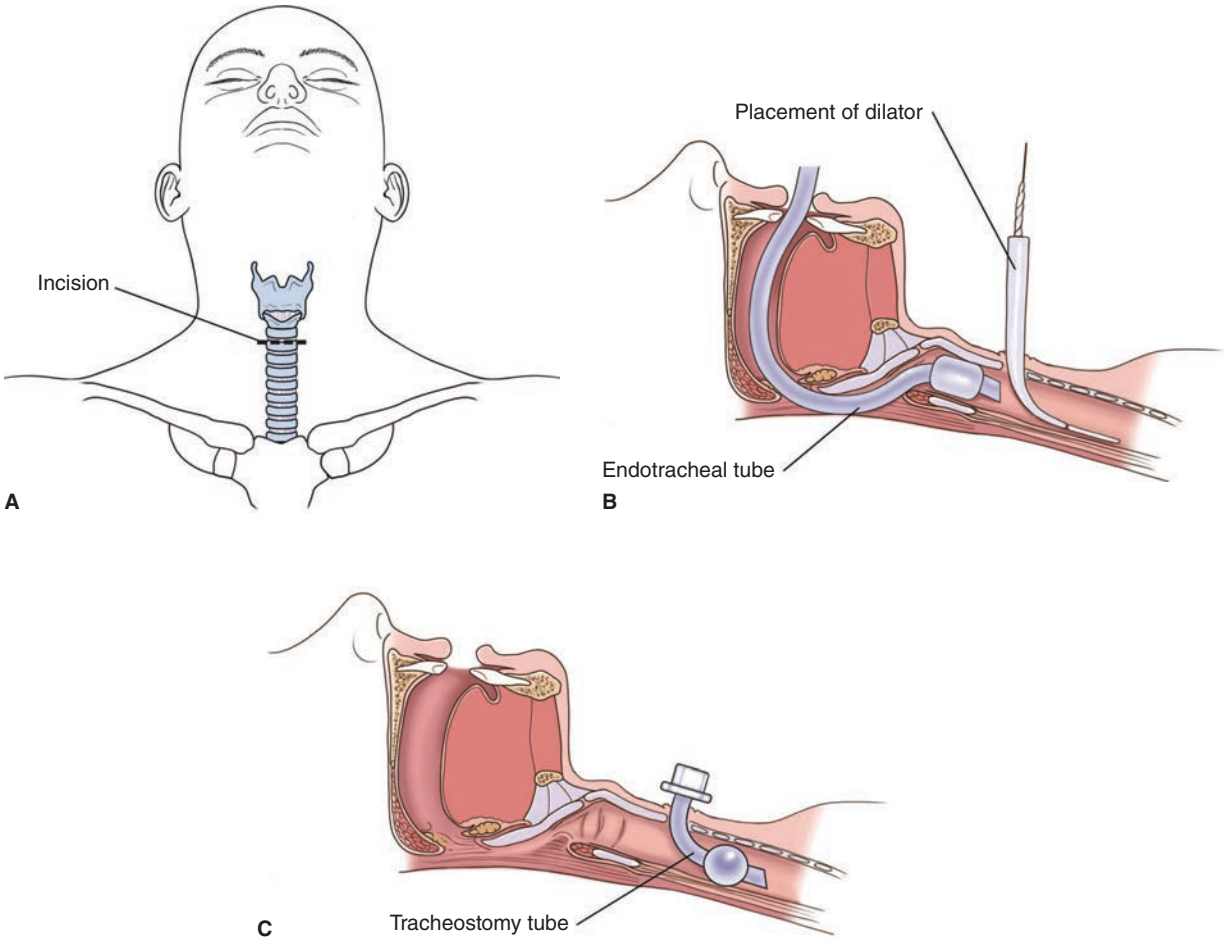

FIGURE 14 Percutaneous Tracheostomy

A. This drawing depicts the initial approach to a percutaneous tracheostomy. The trachea, location of the larynx, and the first few tracheal rings are palpated. The initial needle insertion site is between the first and second tracheal rings.

B. This is a lateral view of the dilation maneuver of the trachea as part of a percutaneous tracheostomy. A needle has been inserted between the first and the second tracheal rings, with a wire inserted through the needle. A dilating device is inserted over this wire into the trachea, and a tracheostomy tube is inserted into this stoma. During the insertion of the initial needle, care is taken to avoid perforation of the occluding balloon on the endotracheal tube.

C. This is a lateral view of a completed percutaneous tracheostomy. Note that a tracheostomy tube has been inserted through the orifice created by the dilator inserted between the first and second tracheal rings. The endotracheal tube inserted via the mouth has been removed.

CHEST AND THORACIC OUTLET

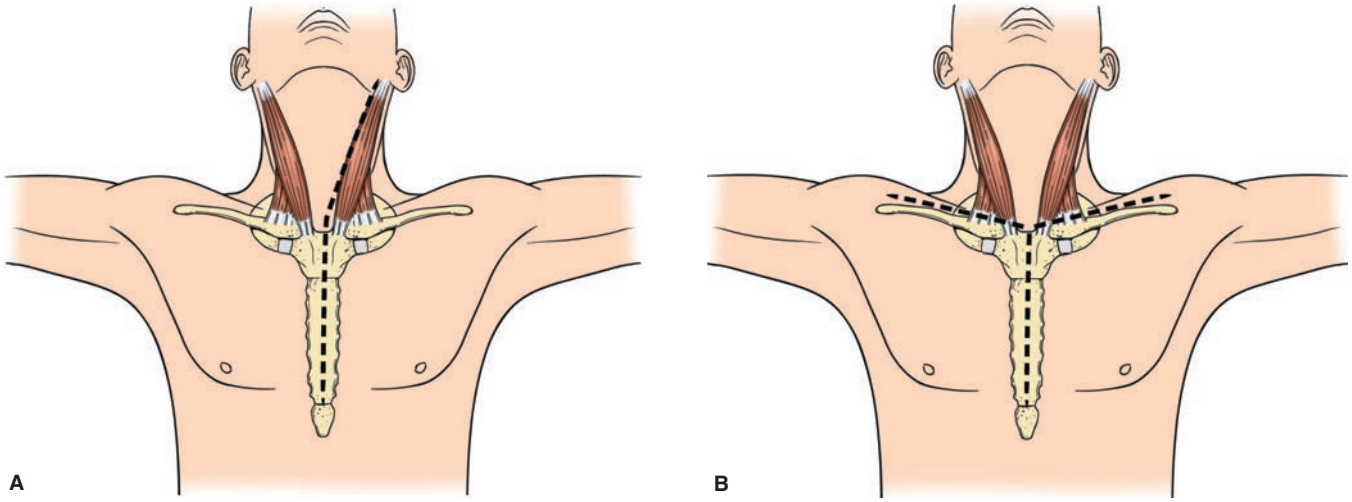

FIGURE 15 Cervicothoracic Incisions

A. For thoracic outlet injuries (zone 1 cervical injuries), median sternotomy may be combined with either a right or left classic anterior neck incision, which allows for proximal vascular control.

B. For injury to the proximal extrathoracic subclavian artery, supraclavicular extension of a median sternotomy allows for proximal control as well as exposure should division or removal of the clavicle be required for exposure and repair of the injury.

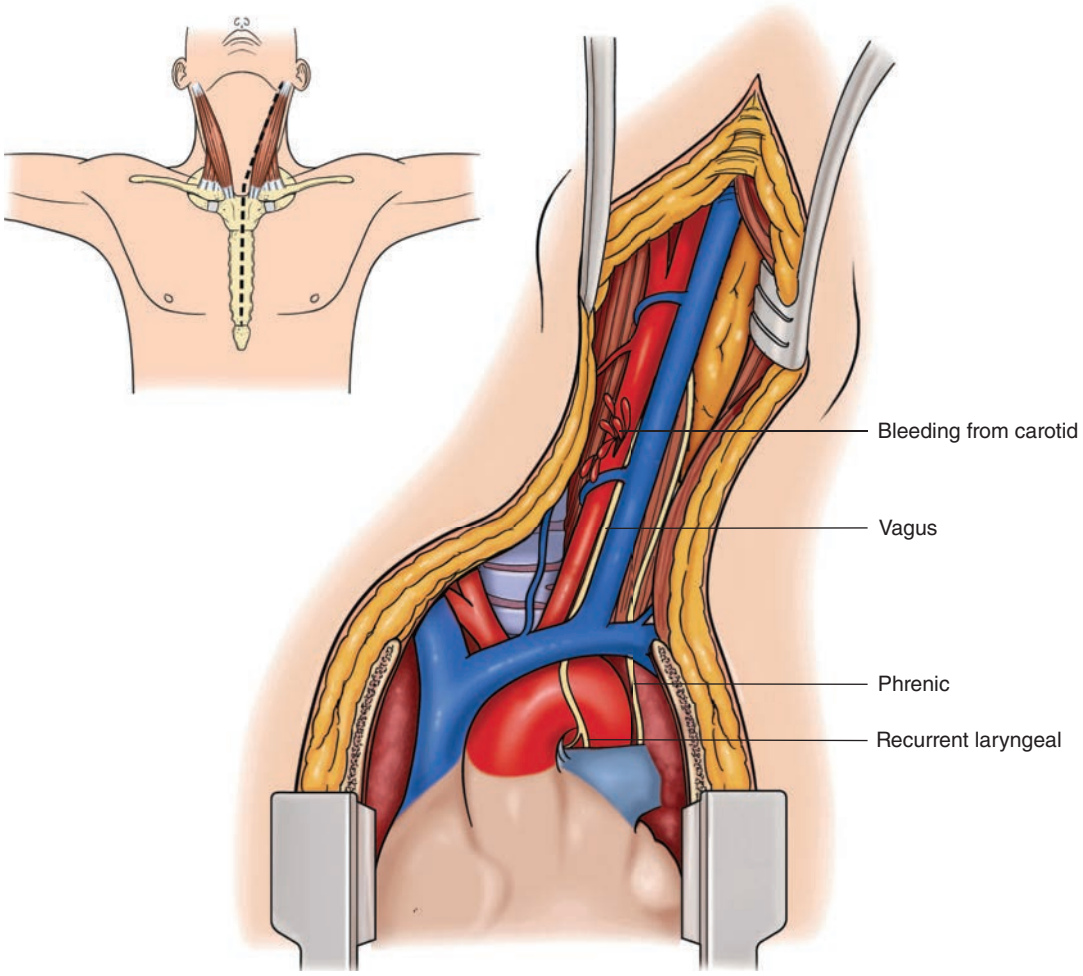

FIGURE 16 Median Sternotomy with a Cervical Extension for Access to an Injury to the Left Proximal Common Carotid Artery
A neck incision or a median sternotomy, alone, is insufficient for control and reconstruction of injury to the left proximal common carotid artery; however, a combined incision, aided by two retractors, affords excellent exposure. Note the location of the left vagus nerve with its recurrent laryngeal nerve around the aortic arch. Also, note the lateral location of the left phrenic nerve.

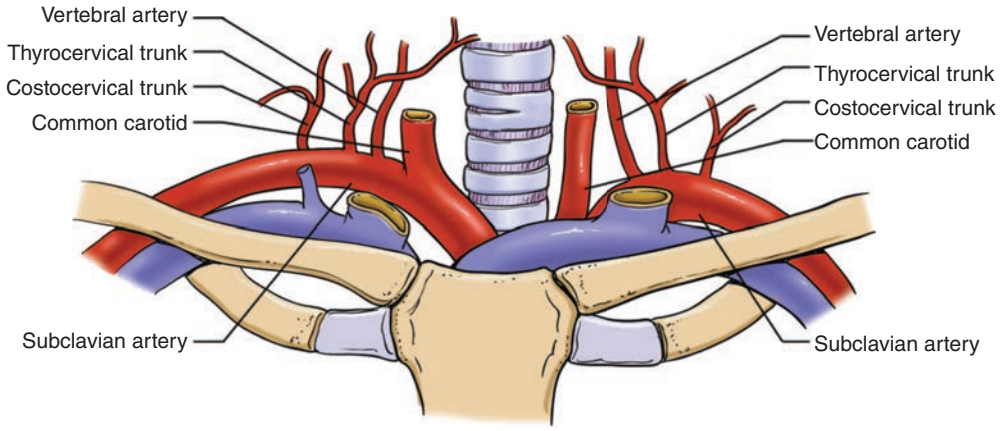

FIGURE 17 Subclavian Artery Anatomy
This illustrates the cervical anatomy of the subclavian arteries, with arms at the patient's sides, which places the clavicles at their lowest positions. The arms in the outward, extended positions result in clavicles covering the subclavian arteries. Note that both the right and left subclavian arteries have four branches (internal mammary arteries coming off the undersides of the subclavian arteries are not shown).

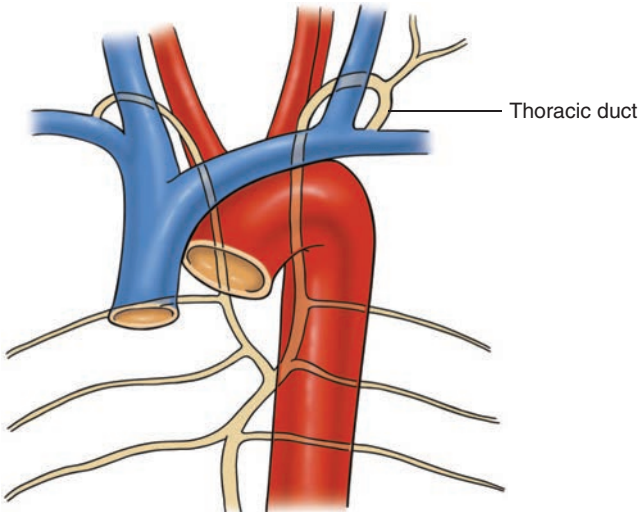

FIGURE 18 Anatomy of the Thoracic Duct
At the base of the mesentery, anterior to the abdominal aorta and very near the left renal vein, the cisterna chyli collects lymph from the mesenteric lymphatic channels and carries lymph upward, continuing anterior to the thoracic aorta on to the thoracic outlet, via the thoracic duct. Other numerous lymphatic collateral channels join this thoracic lymphatic duct, where it bifurcates in the upper posterior chest. Although the left thoracic duct is the larger, a thoracic duct empties into the right superior subclavian vein just as it receives the corresponding internal jugular vein. Note that the thoracic duct is anterior to the subclavian artery and posterior to the subclavian vein.

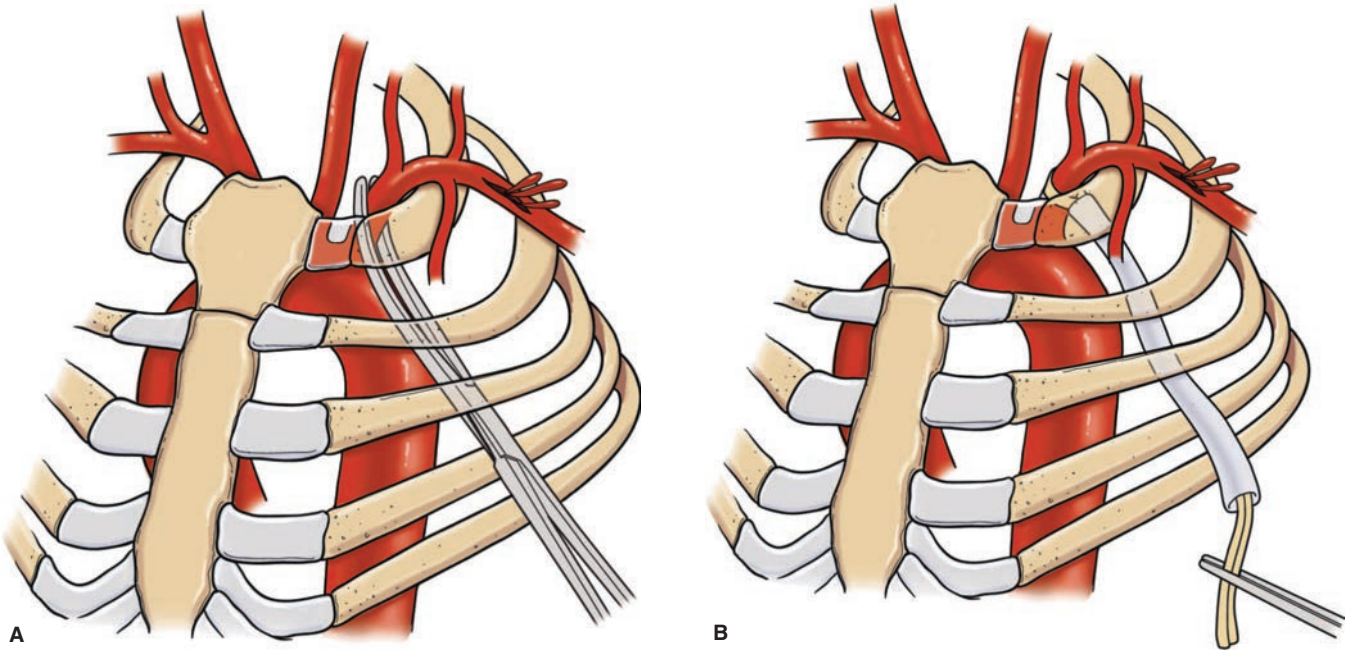

FIGURE 19 Controlling Hemorrhage from the Left Subclavian Artery
A. Temporary vascular control is achieved via short anterior third interspace incision with a vascular clamp applied to the intrathoracic left subclavian artery.
B. Alternately, control can be achieved using a Rumel tourniquet.
Note: In the emergency room, this injury can be immediately temporarily controlled with the tamponading finger or an inflated 30-mL Foley balloon.

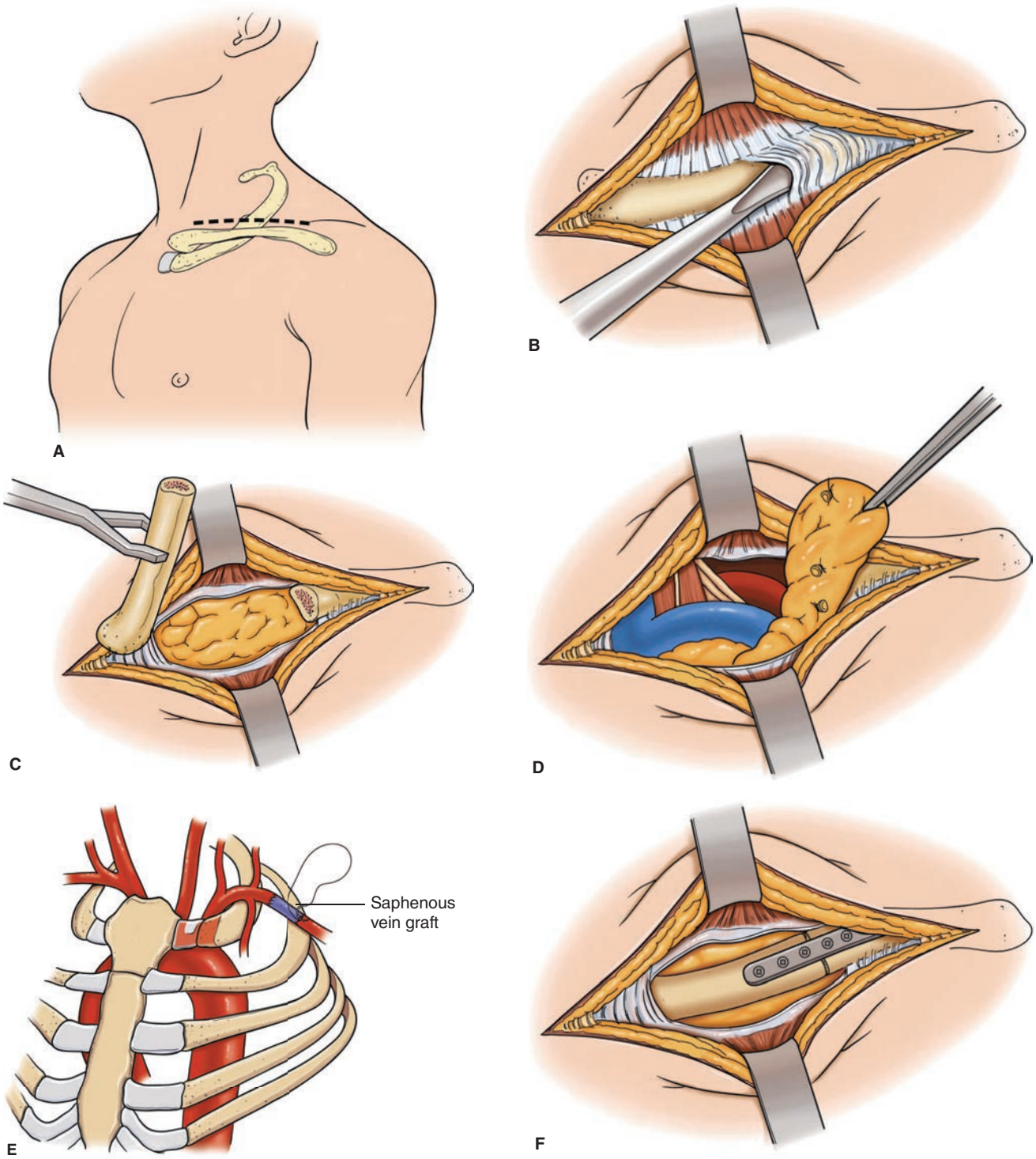

FIGURE 20 Exposure and Repair of an Injury to the Subclavian Artery

A. Although endovascular stent graft options are increasingly being used for injury to the subclavian artery, they are not always available, so direct exposure may be required. With the arm prepped free but positioned at the patient's side, clavicular division or resection might aid in exposure. A liberal supraclavicular incision is made over the clavicle.

B. The bone is exposed using a periosteal elevator.

C. The clavicle is divided in its middle portion, and the proximal end may be retracted medially.

D. The key to the fossa containing the subclavian artery is to remove the scalene fat pad. The phrenic nerve lays on the middle one-third of the scalene anticus muscle, the lateral half body of which is divided to aid exposure.

E. The subclavian artery is extremely fragile, and a saphenous vein is the preferred conduit to use in an open reconstruction.

F. Following vascular reconstruction, the clavicle can be reconstructed with an orthopedic plate or a sternal wire with the twisted knot placed anteriorly.

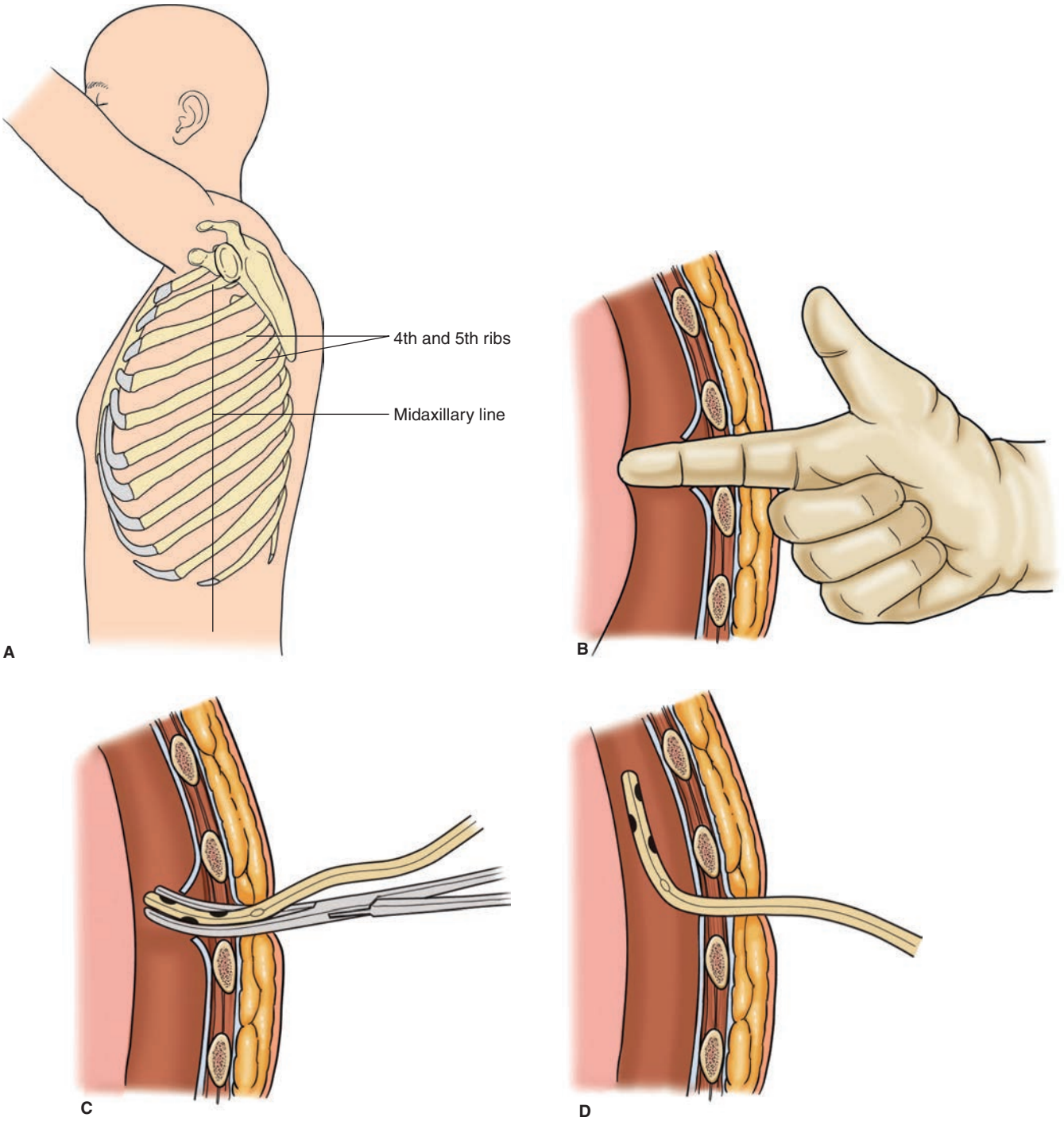

FIGURE 21 Tube Thoracostomy, the Most Commonly Performed Thoracic Operative Procedure

A. The lateral anatomy in the auscultatory triangle at the fourth intercostal space in the midclavicular line is the point for chest tube insertion.

B. Following adequate local anesthesia, a transverse incision through the skin and subcutaneous tissue is made. A large Kelly clamp is then used to spread the intercostal muscles and make a hole in the pleura. Up to 25% of patients have some element of pleural symphysis, and entering the pleura with a trocar or other similar instrument risks producing an iatrogenic lung injury. A finger digital exploration to localize the pericardium or a diaphragmatic injury and/or release pleural adhesions is performed before the thoracostomy tube is inserted.

C. After an appropriately sized hole is created, a chest tube is introduced with the aid of a large curved clamp attached to the tip and directing the tube to the posterior apex location of the pleural space. The tube is attached to an appropriate collection, water seal, and negative pressure device.

D. The chest tube is aimed toward the apex of the pleura, with the last hole in the tube inside the chest wall.

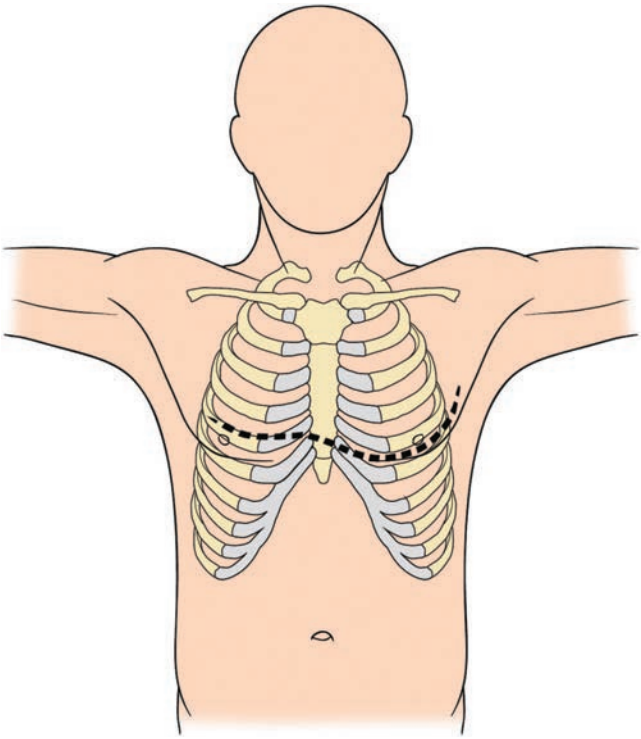

FIGURE 22 Resuscitative Thoracotomy 1
This depicts the standard left anterolateral thoracotomy. The dotted line depicts the incision for a left anterolateral thoracotomy into a bilateral anterolateral (clamshell) thoracotomy. Note that the left incision is in the area of the fourth or fifth interspace, and below the mammary crease.

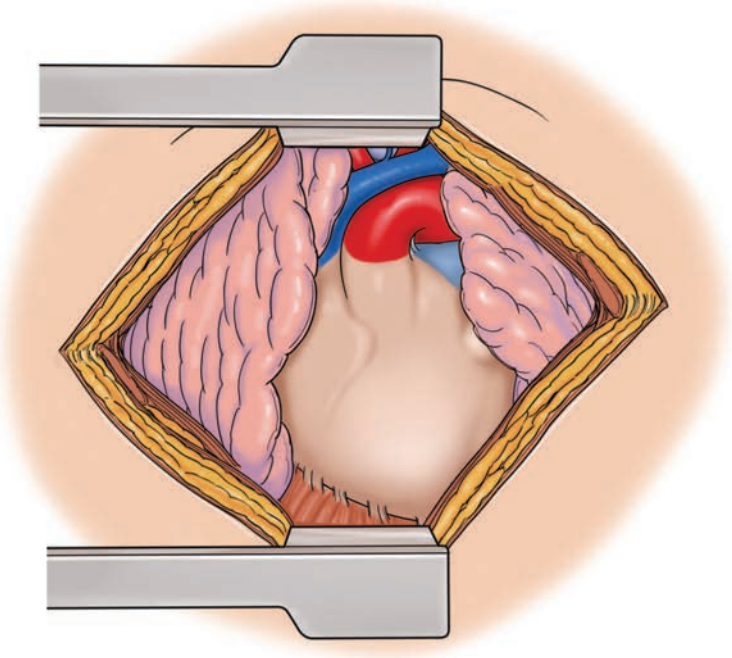

FIGURE 23 Resuscitative Thoracotomy 2
This is the anterior mediastinum and both anterior thoraces following bilateral anterolateral or clamshell thoracotomy. Note that one can visualize both lungs, the pericardium, the heart and all of its chambers, the superior and inferior vena cava, the ascending aorta, the pulmonary artery, the innominate vein, the innominate artery, and the left common carotid artery. This drawing does not show, but assumes the ligation of both divided ends of the right and left internal thoracic (mammary) arteries.

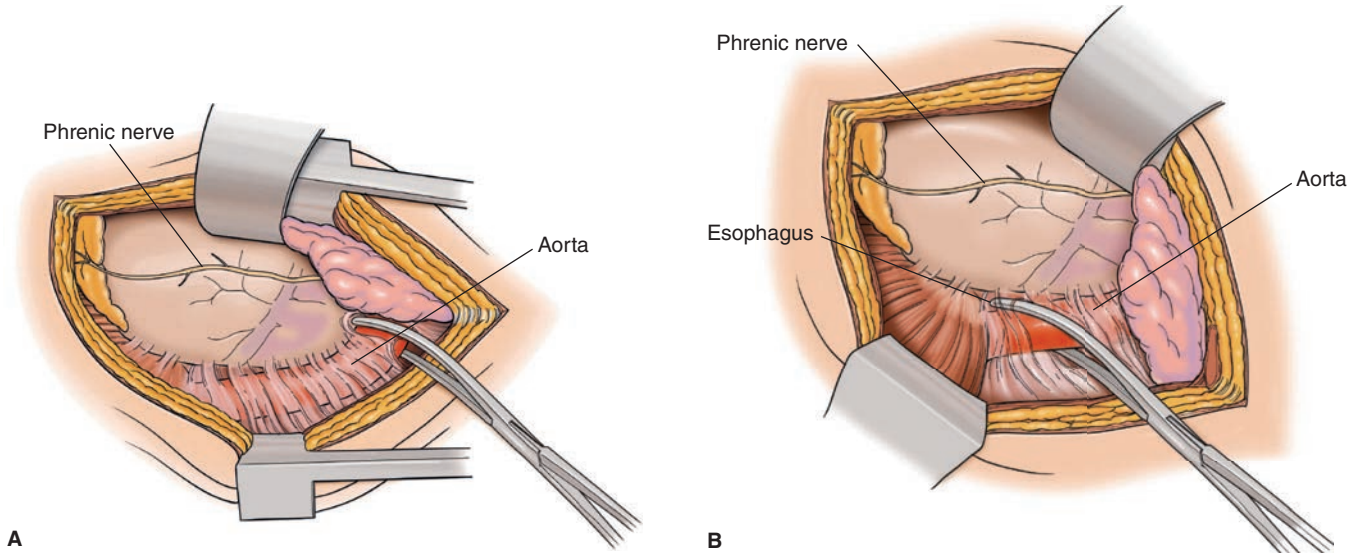

FIGURE 24 Cross-clamping of the Descending Thoracic Aorta

A. With the patient in the supine position, a left fourth or fifth interspace curvilinear incision is made beneath the nipple and breast fold aiming to the axilla. The aorta is clamped higher in the chest, at the proximal descending thoracic aorta, using a vascular clamp.

B. Alternately, the aorta may be cross-clamped low in the chest. The lung is pushed upward, showing the heart within the pericardium, the phrenic nerve, the diaphragm, and the aorta. The esophagus is seen anterior to the aorta and the spine, with the segmental arteries coming off at each interspace. The aorta is cross-clamped low in the thorax using a vascular clamp.

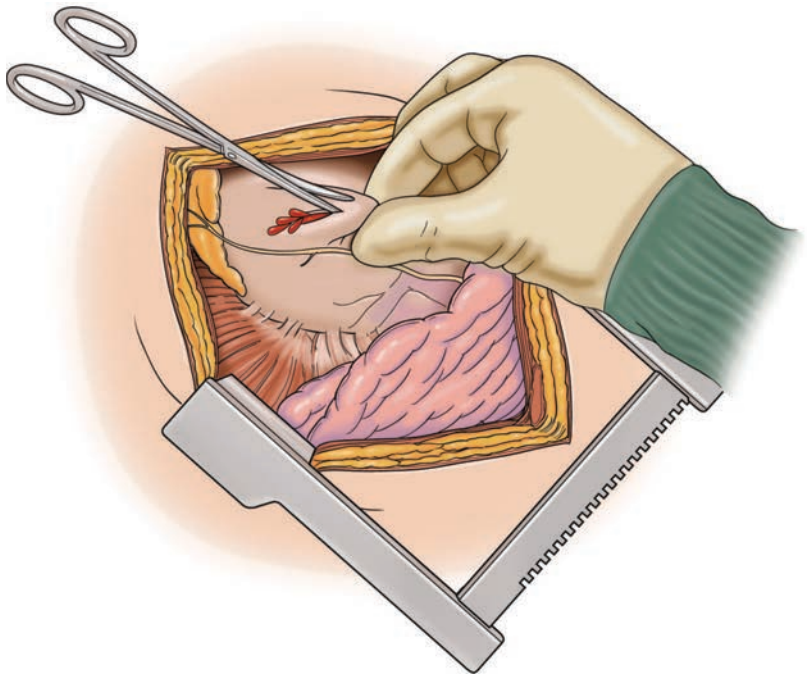

FIGURE 25 Opening the Pericardium to Explore the Heart

Using scissors or a scalpel, the left pericardium is incised, with the lung positioned posteriorly. The incision is made anterior and parallel to the phrenic nerve. With a tense hemopericardium, purchase of the pericardium is difficult, making this maneuver challenging. The ratchet mechanism of the chest wall retractor is positioned posteriorly.

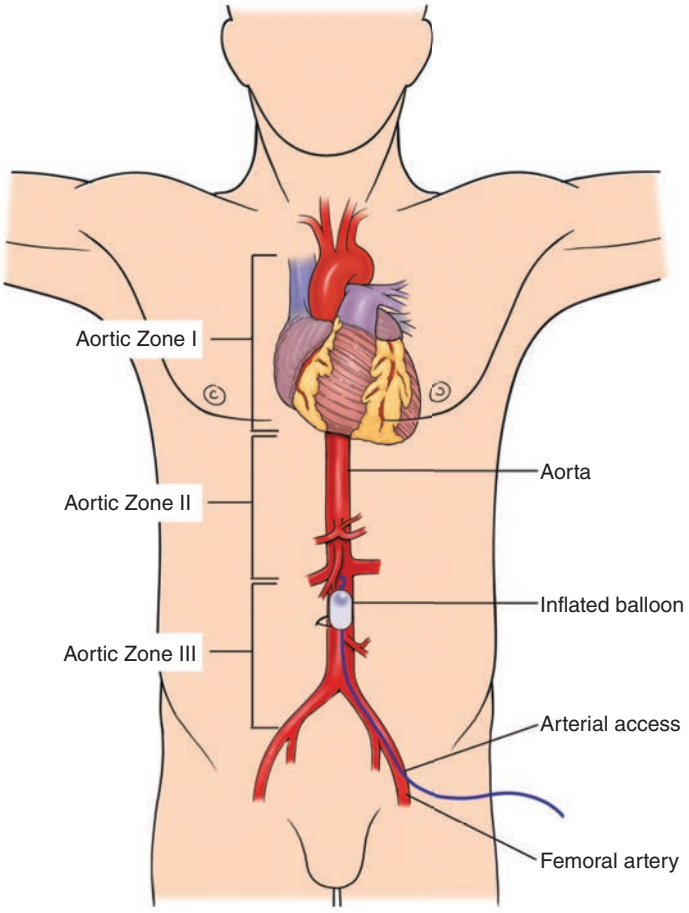

FIGURE 26 REBOA (Resuscitative Endovascular Balloon Occlusion of the Aorta)
REBOA is an occluding aortic balloon introduced via the common femoral artery either percutaneously or by direct cutdown. In this instance, the balloon has been inflated distal to the renal arteries (zone III).

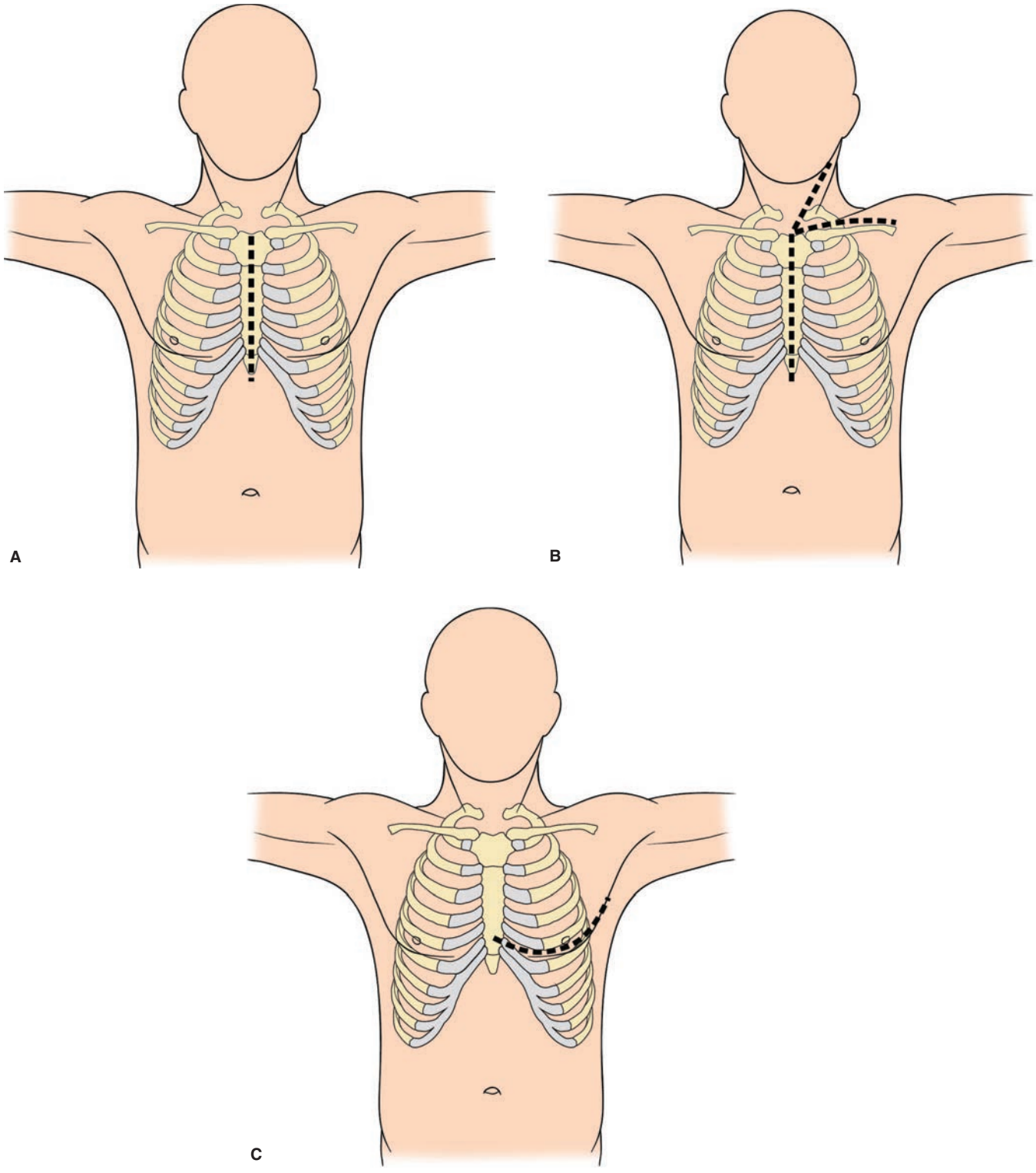

FIGURE 27 Options for Thoracic Incisions

A. The median sternotomy is the standard incision for anterior cardiac and thoracic outlet vascular injury, but is not an appropriate incision for approach to posterior mediastinal structures or the pulmonary hilum.

B. A median sternotomy with an anterior neck or supraclavicular extension is used for thoracic outlet great vessel injuries to zone I of the neck.

C. The anterolateral incision, particularly on the left, is the utility emergency thoracotomy for trauma and resuscitation. It is made from the sternal edge, under the mammary fold, and in a curvilinear fashion toward the axilla, staying in close proximity to the fourth or fifth intercostal space. This incision should not be a straight line incision nor be carried through the female breast.

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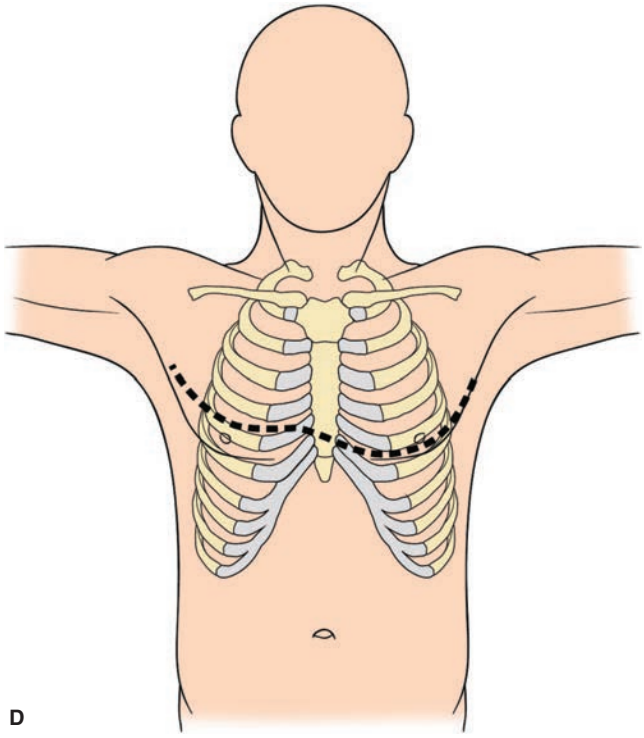

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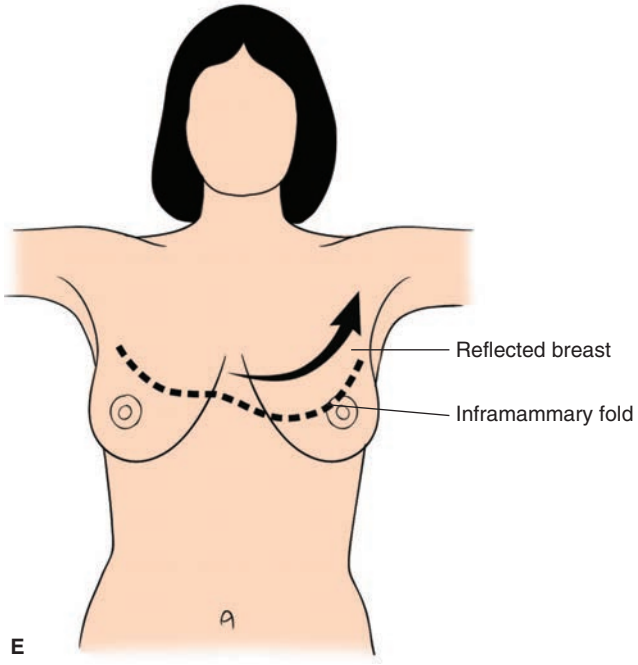

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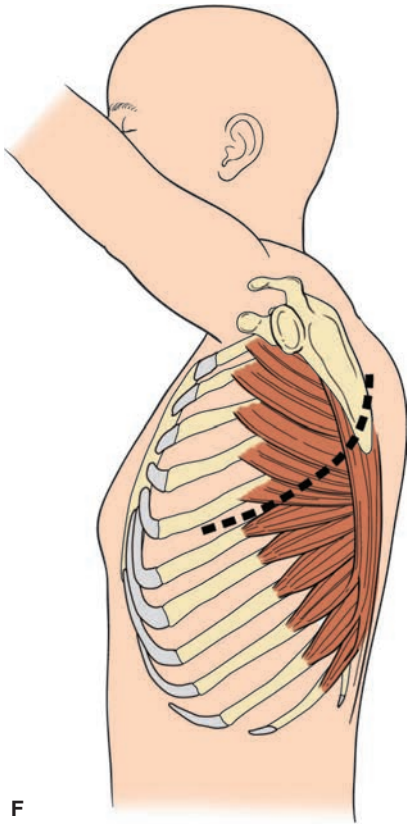


F

FIGURE 27 Options for Thoracic Incisions (*continued*)

D. Bilateral anterolateral incisions may be either separate or combined and transternal. When transternal, the sternum is traversed with a Gigli saw or other cutting device, and the internal thoracic arteries are ligated on both sides on the upper and lower incision sites (four ligatures). Both incisions should be curvilinear, with the transternal cut high enough on the sternum to expose the mid-portion of the heart and also with sufficient sternum to accomplish a solid bony closure. On occasion, when a right-sided injury is suspected high in the pleural cavity, the incision might even be above the right nipple.

E. In the female, the anterolateral incision is at the inframammary fold and is accomplished by moving the breast tissue cranially.

F. With the patient in a lateral decubitus position, a posterolateral fourth or fifth interspace incision can be made from near the area of the nipple, laterally around to near the spinal canal. This incision for trauma traverses the latissimus dorsi muscle and portions of other chest muscles. The scapula must be retracted superiorly to achieve fourth or fifth interspace incisions. This incision provides exposure of posterior mediastinal structures, such as the aorta, lung hilum, esophagus, trachea, and azygos vein.

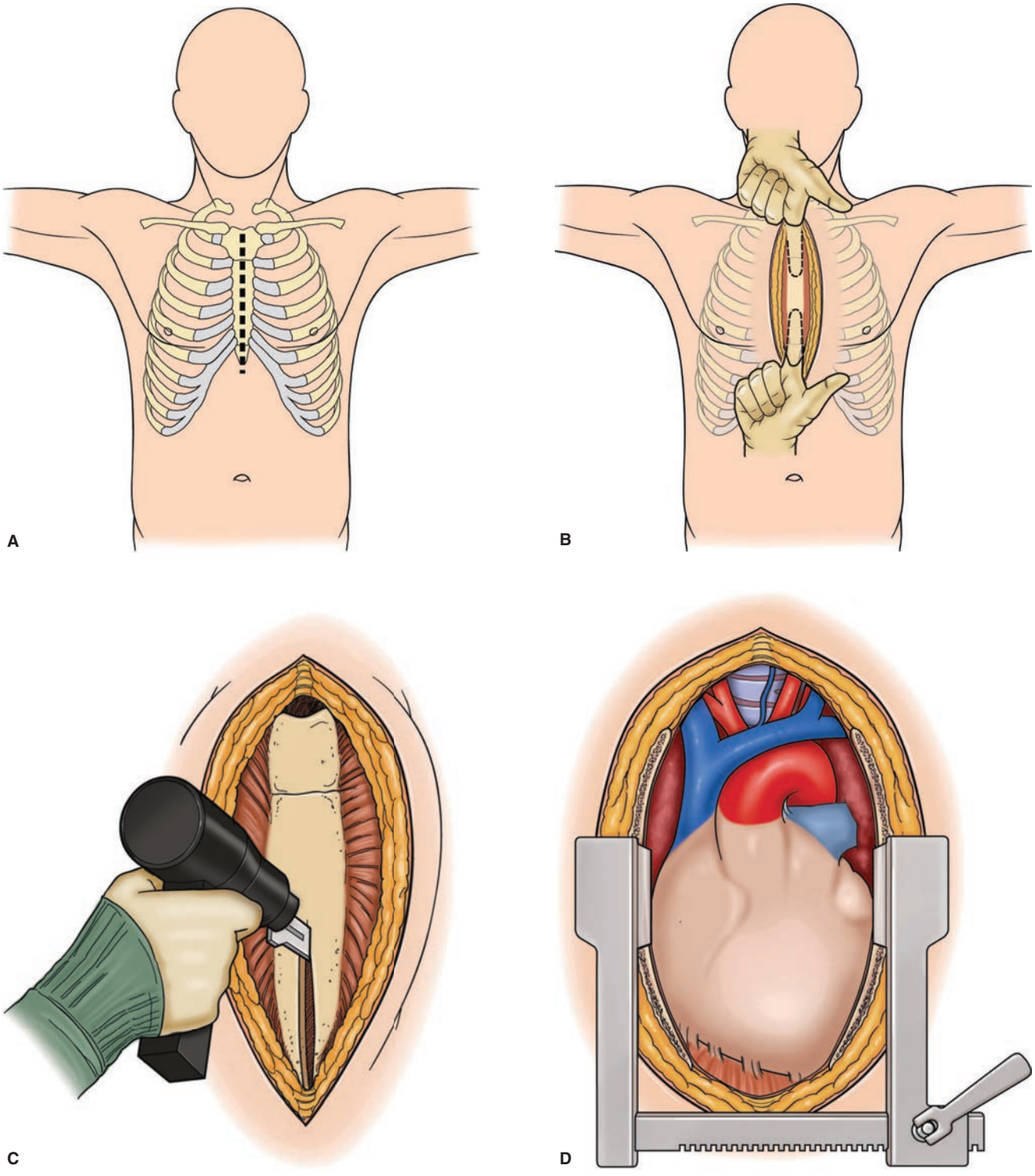


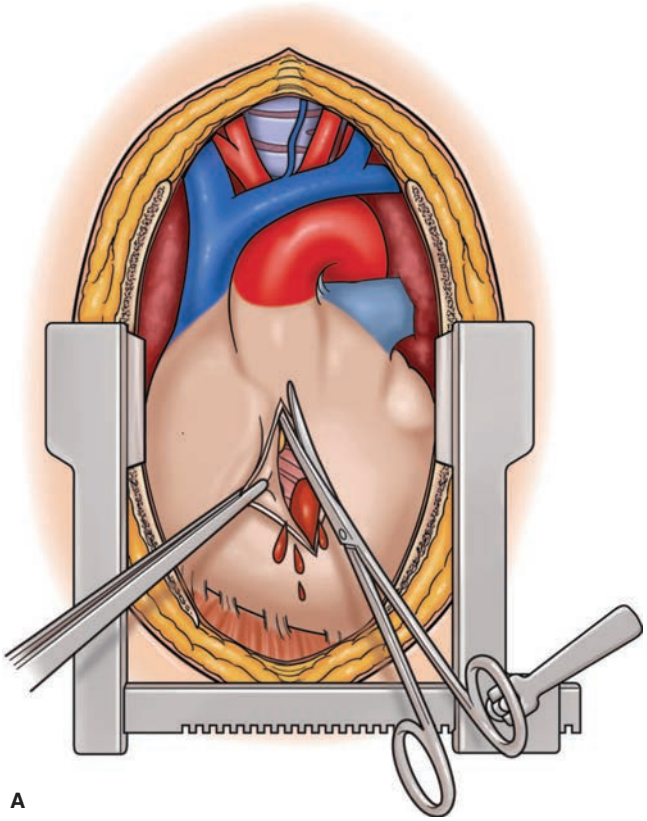
FIGURE 28 Technique for Performing Median Sternotomy

A. With the patient appropriately prepped and draped, a skin incision is made from the manubrial notch to below the xiphisternum.

B. Using blunt dissection, the fingers are inserted just beneath the sternum from below and above, carefully dissecting the pericardium and loose fatty tissue away from the back of the sternum.

C. Using a sternal saw, keeping in the midline of the sternum, and exerting upward pressure on the saw, the total length of the sternum is cut. Care is taken not to divert into the right or left chest cavity.

D. A sternal retractor is placed into the incision, first with the two blades touching each other, and then slowly opening the sternum to avoid fracture of the sternum or any ribs. The pericardium, the thoracic vena cava, ascending aorta, pulmonary artery, and lower neck structures are now exposed. The left innominate vein, in the upper extent of the incision, is enclosed in fatty and thymic tissue, and care must be taken not to injure this vein.



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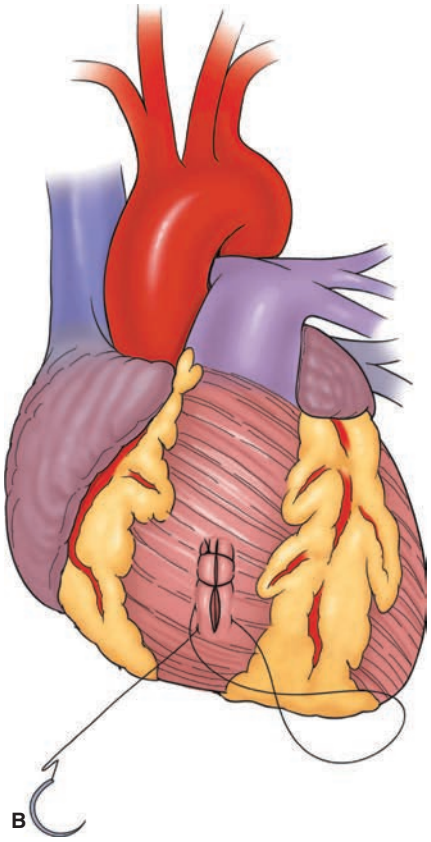
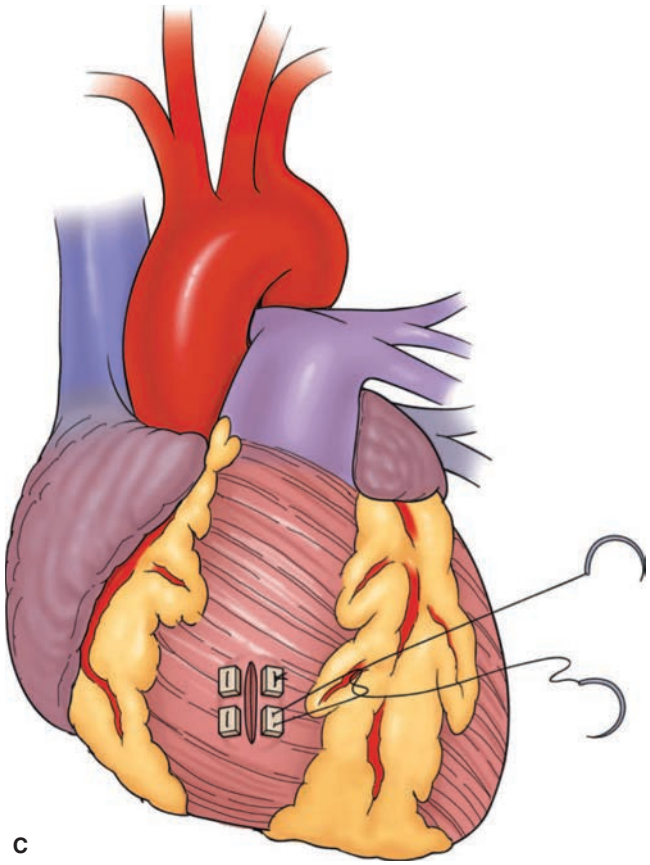


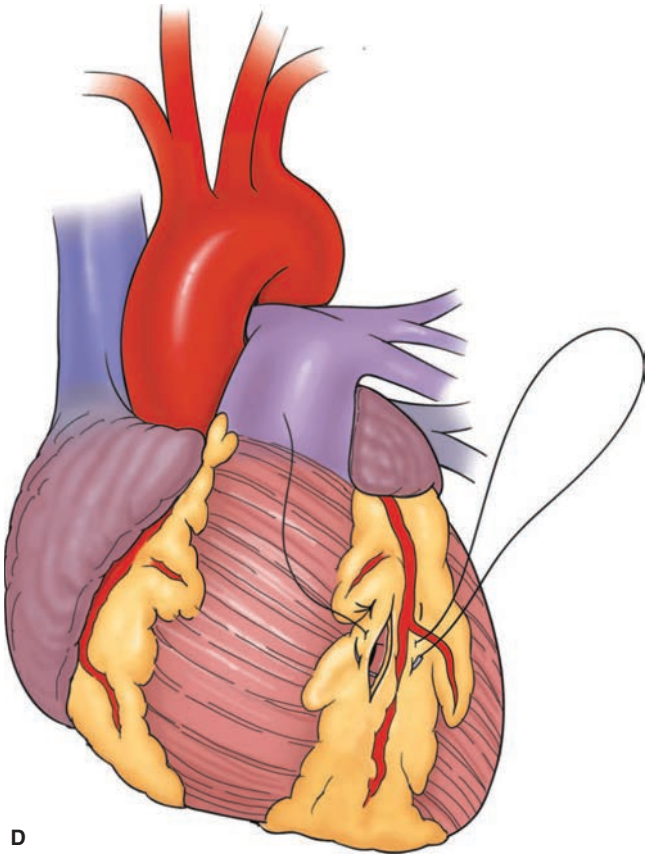
B

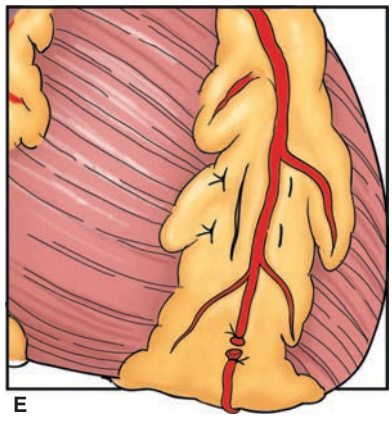
FIGURE 29 Cardiorrhaphy

A. Through a median sternotomy, the tense pericardium is incised in the midline and the clot extracted as digital hemorrhage control is accomplished.

B. Cardiorrhaphy is accomplished by simple direct closure of the penetrating wound using 4(0) polypropylene suture. Repair is accomplished prior to cardiac defibrillation. Often, traction on a figure-of-eight suture will cause all bleeding to stop, and the suture is simply tied.







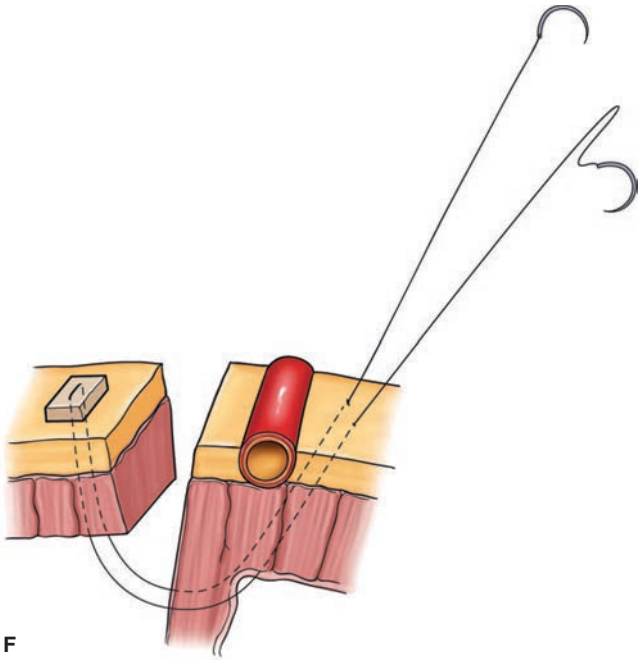


FIGURE 29 **Cardiorrhaphy (*Continued*)**

C. Some surgeons prefer to accomplish cardiorrhaphy with pledgets, an adjunct most useful on the left ventricular wall exposed to high pressures.

D. For injury to the heart, very near a coronary artery, it is possible to use the double needle armed suture to go under the artery with both needles and tie the suture (with or without pledgets) lateral to the injury. For a distal coronary artery injury, simple ligation is sufficient.

E. Completed closure and ligated distal coronary artery injury.

F. Cross section demonstrating the coronary artery undercircling technique.

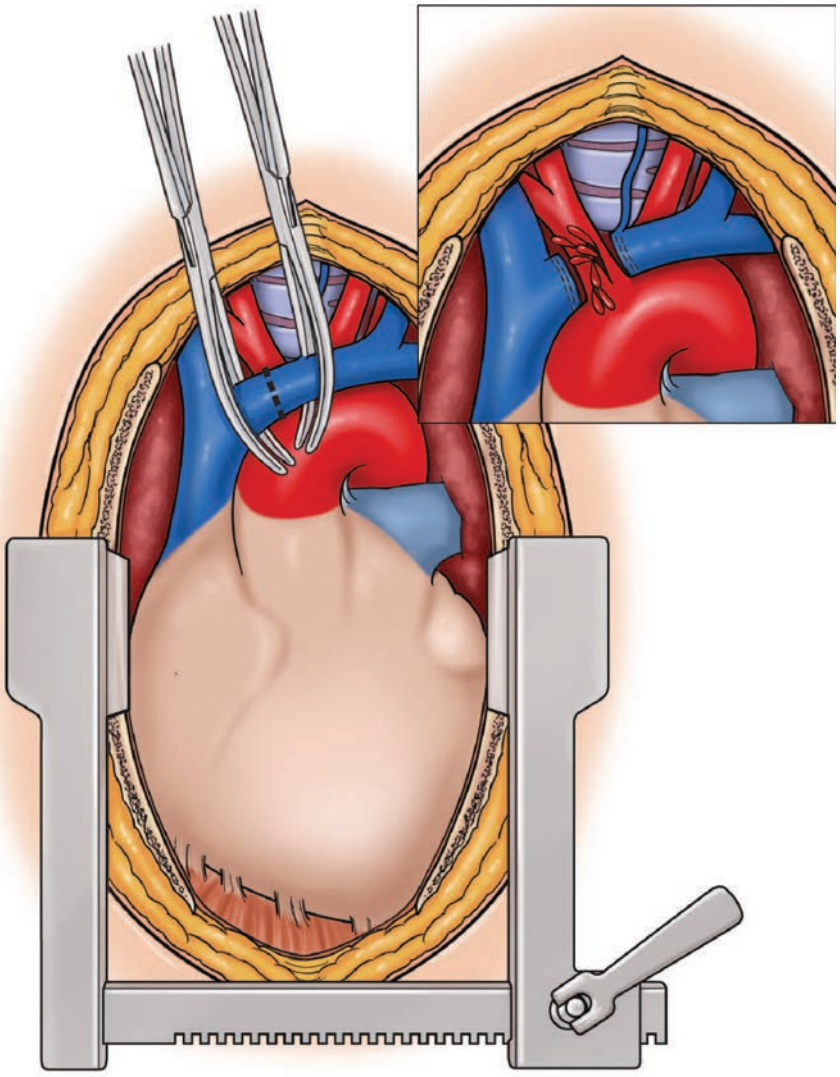


FIGURE 30 Penetrating Injury to the Innominate Artery
When a bullet or knife traverses the left innominate vein and injures the underlying artery, the vein can be divided for access. Optimally, the vein should be reconstructed, but this is not essential if the right innominate vein is normal.

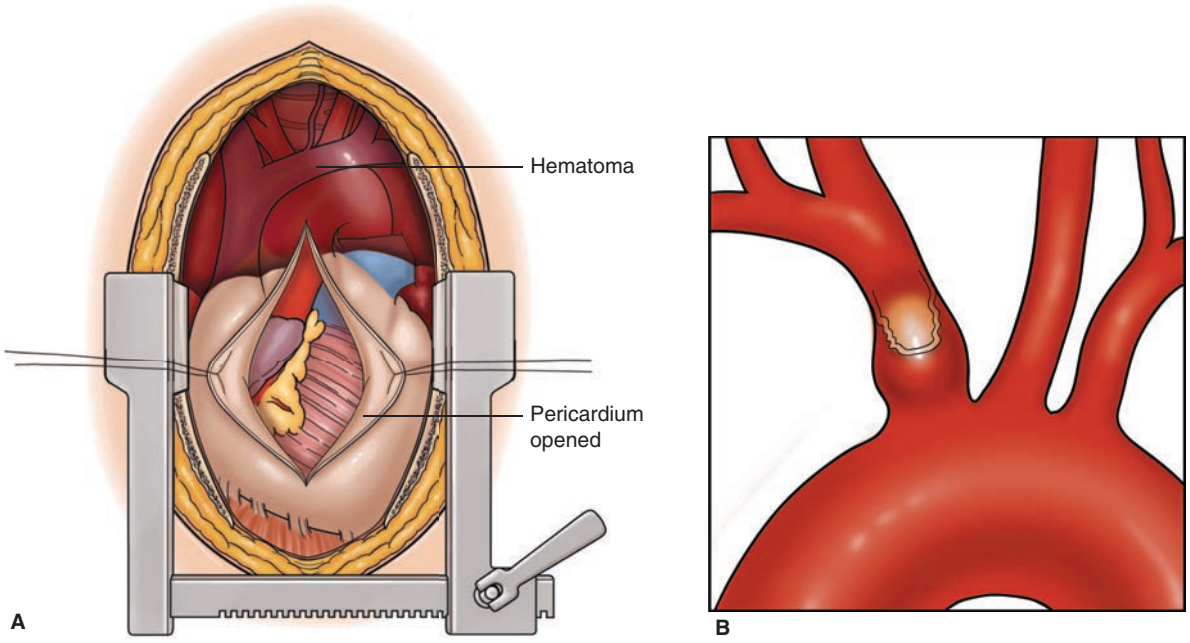
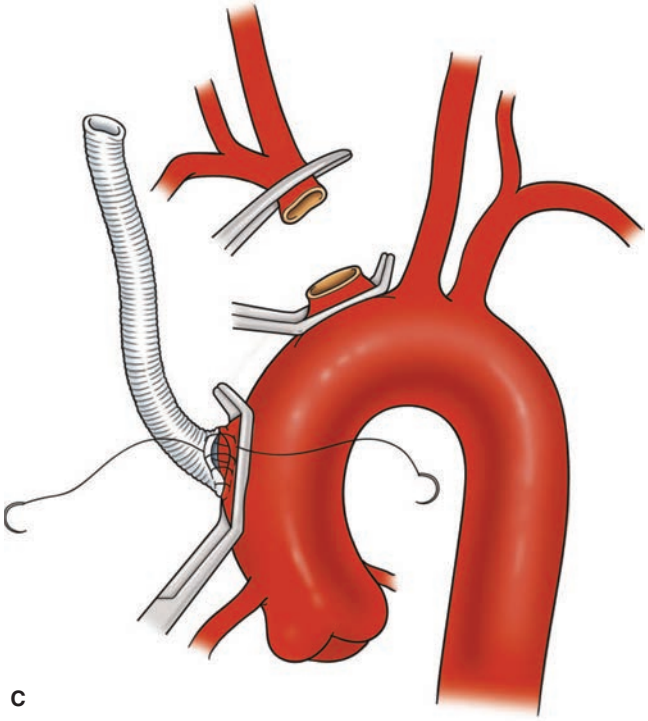


FIGURE 31 Exposure and Management of Injury to the Innominate Artery

A. The pericardium is opened vertically via a median sternotomy with the sternal retractor in place to expose the anterior heart and ascending aorta. The source of intrapericardial bleeding must be determined. The pericardial incision extends superior to the ascending aorta near the aortic arch. At the thoracic outlet, note the hematoma, which is encountered with an injury to the innominate artery or vein or proximal common carotid artery. In this drawing, the upper limits of the incision are extended into the neck (can be either to right or left of midline) so that the distal control of the thoracic outlet great vessels can be obtained. Note, also, that the innominate vein is in the center of the hematoma.

B. A blunt injury to the proximal innominate artery is actually a fracture of the aortic arch intima at the takeoff of the innominate artery. The intima of the innominate artery is rolled up with the presentation of the arterial discoloration, and injury appears to be in the innominate artery. Through a median sternotomy with a right neck extension, the ascending aorta, aortic arch, innominate vein, innominate artery, subclavian artery, and right and left carotid arteries are exposed.

(continues on next page)



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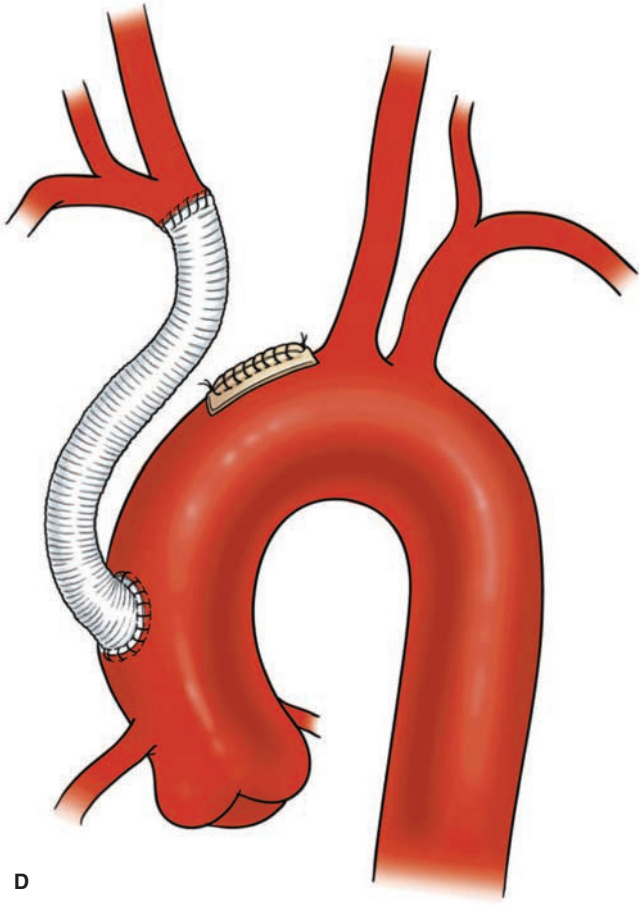


D

FIGURE 31 Exposure and Management of Injury to the Innominate Artery (*Continued*)

C. Without systemic heparinization or hypothermia, a Dacron graft is sutured end to side to the ascending aorta. In some instances the innominate vein might be divided to expose the area of injury. A partially occluding clamp is placed on the aortic arch proximal to the takeoff of the innominate artery and another vascular clamp is placed just proximal to the bifurcation of the innominate artery.

D. The graft is sutured to the distal innominate artery, taking care to back-flush and clear the graft of any clot prior to the completion of the suture line. The aortic plate proximal to the injury is oversewn using two pledget strips.

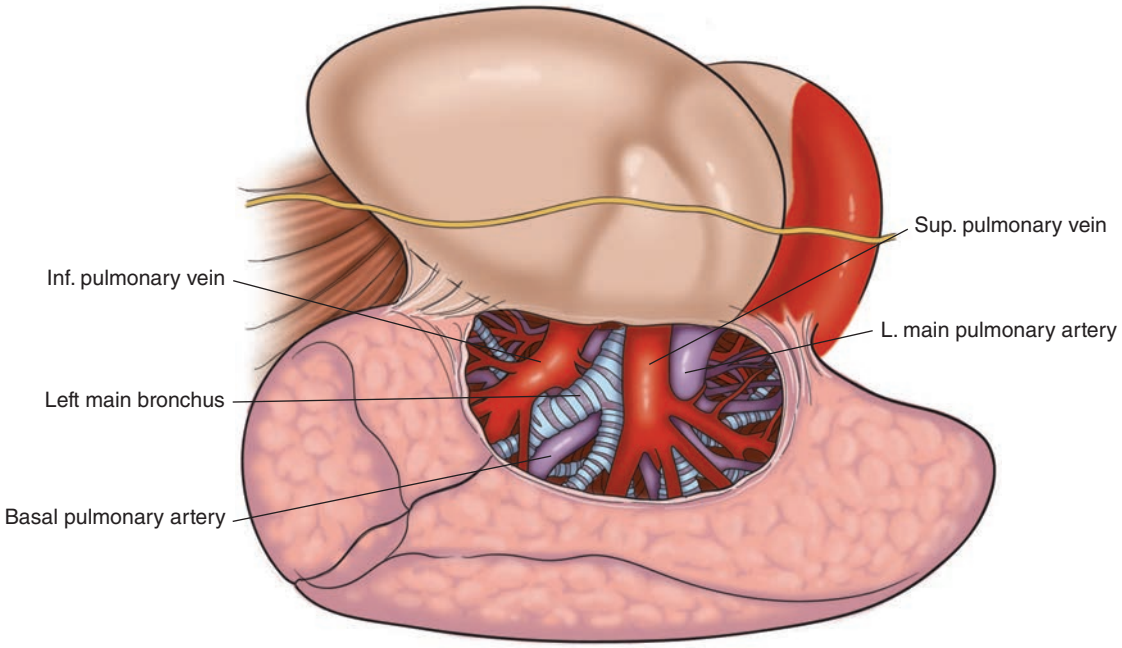


FIGURE 32 Anatomy of Left Pulmonary Hilum
The anatomy of the two pulmonary hila are slightly different. Before performing a trauma lobectomy or even pneumonectomy, a review of the related anatomy can reduce catastrophic iatrogenic bleeding and prevent bronchial injury. With the lung displaced posteriorly and inferiorly, the highest structure in the left pulmonary hilum, the main left pulmonary artery, is exposed and then sharply separated from the aortic arch. This artery goes behind the left superior pulmonary vein (draining the left upper lobe), and the artery continues downward, giving off branches to the left upper lobe and then to its terminal branches to the left lower lobe. The left mainstem bronchus is behind the superior pulmonary artery. Both the superior and inferior pulmonary veins drain into the left atrium.

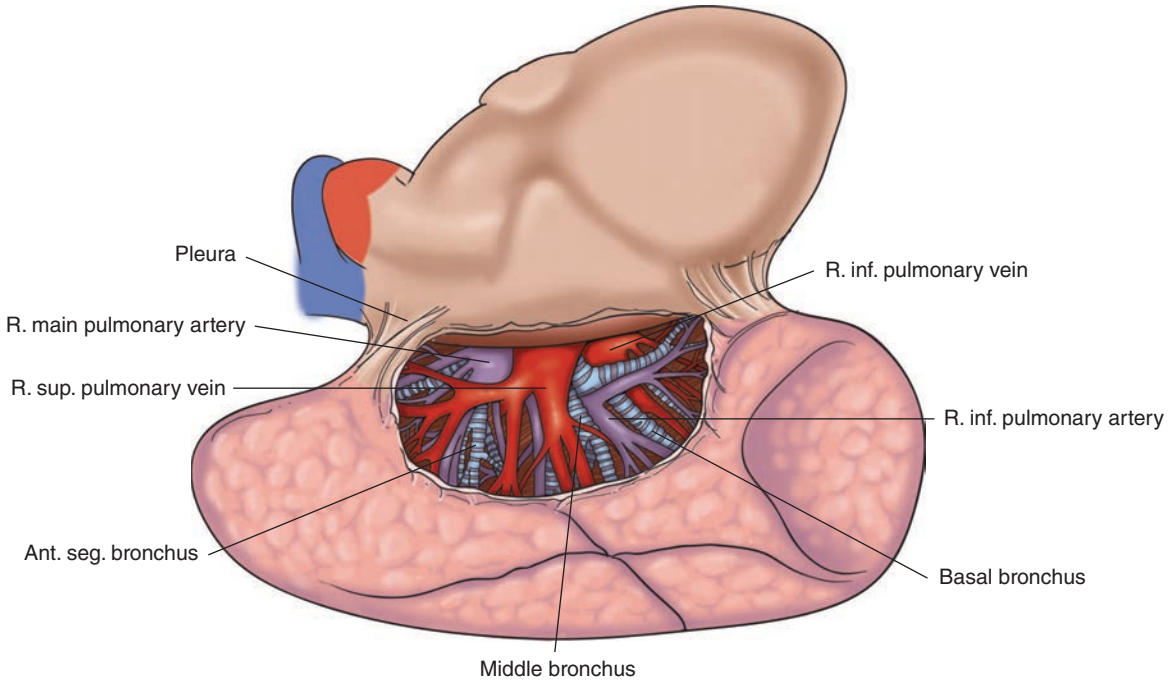
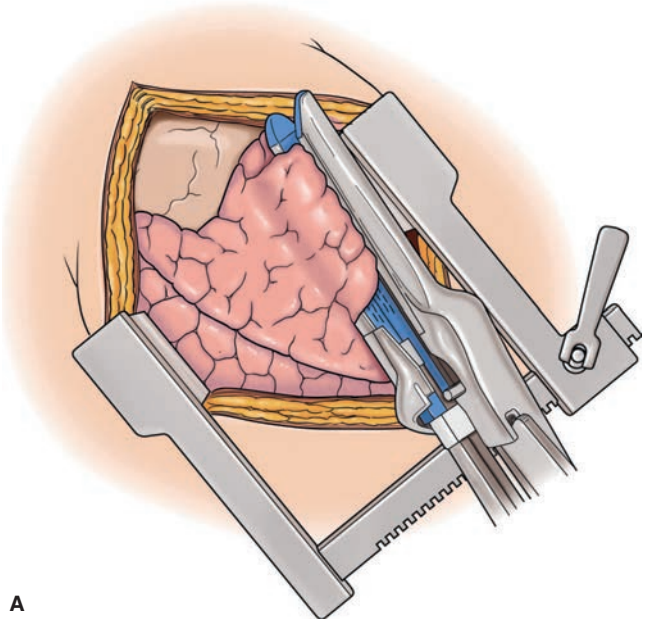
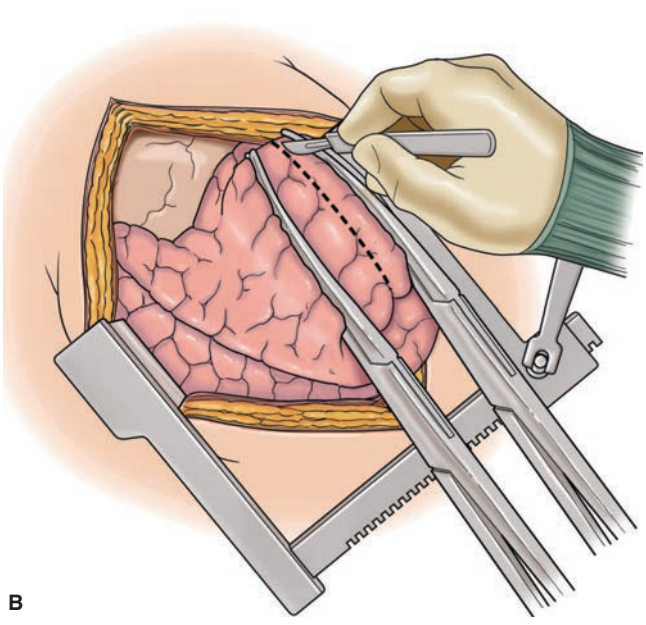


FIGURE 33 Anatomy of Right Pulmonary Hilum
The right lung has three lobes, so the anatomy is different from that of the left lung. To expose either hilum, the pleura covering the vessels and bronchus is mobilized. The highest vessel in the hilum is the right pulmonary artery, giving off branches to the upper and middle lobes, and then continuing to the arborization into the lower lobe. The middle lobe bronchus and artery come off the bronchus intermedius and right intermedius pulmonary artery in an anterior fashion. The inferior pulmonary vein is in a similar position to that on the left.



A



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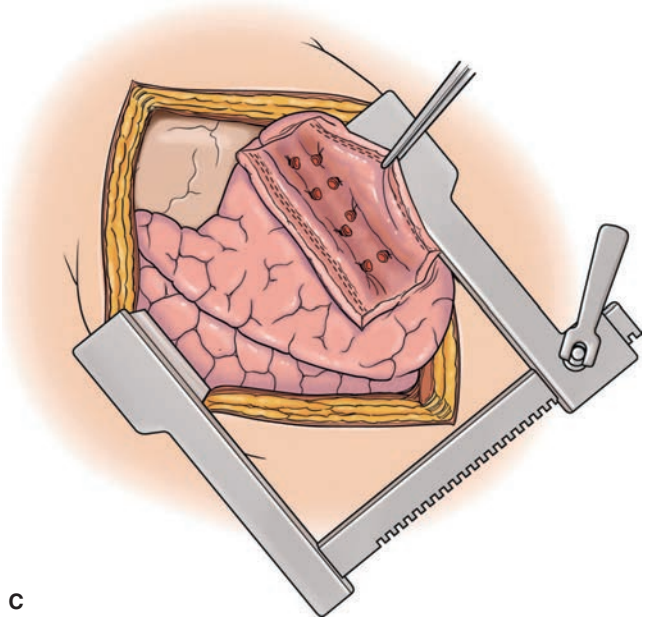


C

FIGURE 34 Pulmonotomy

A. A through-and-through hole to the lung or even a large tangential laceration might be controlled with clamps or a linear stapler. The linear stapler is inserted into the tract, thereby joining the two holes. The stapler is fired and the lung cut, exposing the base of the tract.

B. A pulmonotomy can also be accomplished by placing two large vascular clamps through the tract and cutting the lung between clamps. A polypropylene suture is run back and forth under the clamps, and other sutures control the base of the tract in a manner similar to when the stapler is used.

C. Persistent bleeding points and air leaks are closed with 4(0) polypropylene suture. This nonanatomic procedure is extremely well tolerated.

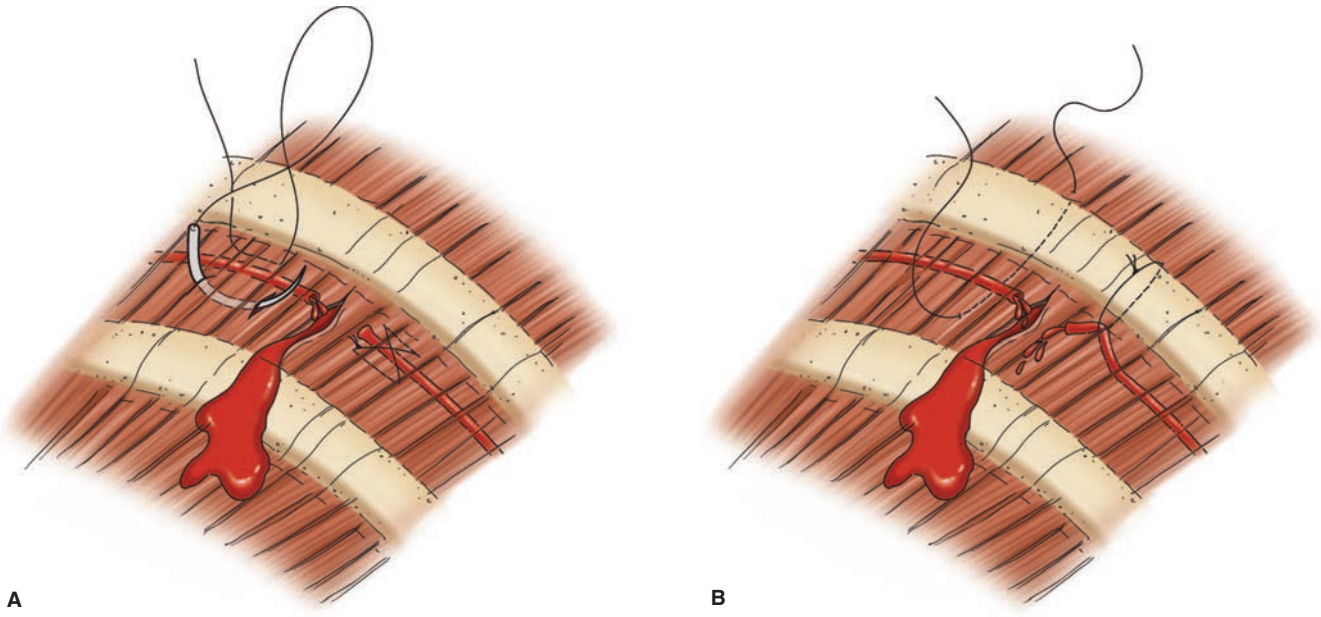


FIGURE 35 Controlling Bleeding from an Intercostal Artery

A. Bleeding from an intercostal artery, injured by a knife, missile, or fractured rib, can be extremely difficult to expose and control. One approach is a figure-of-eight suture ligation of the bleeding intercostal artery at the inferior border of the rib, where the injury occurred.

B. Alternatively, absorbable suture can be used to encircle a rib lateral to the bleeding points, pressing the vessel up against the inferior groove of the rib with the encircling suture.

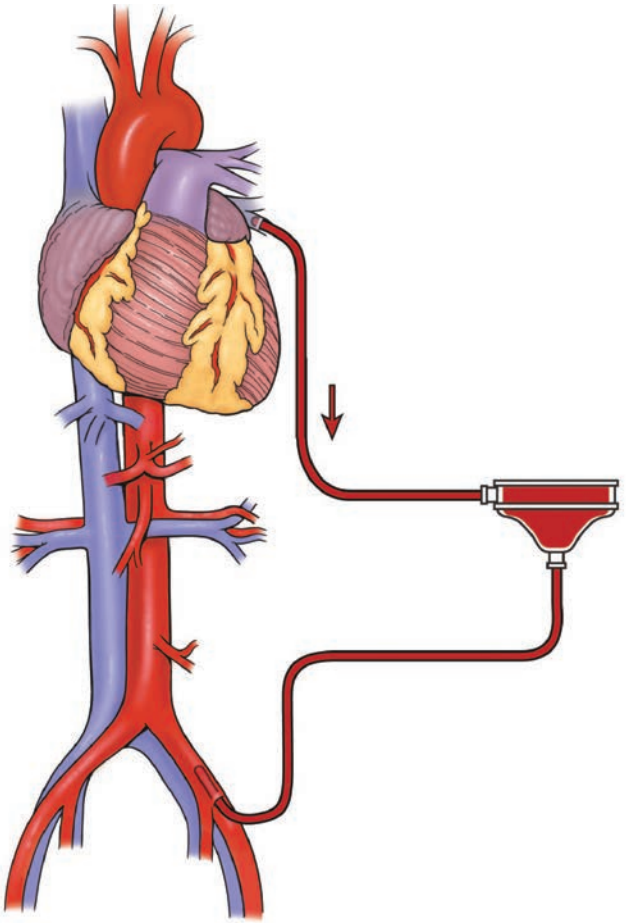


FIGURE 36 Partial Left Heart Bypass 1
Partial left heart bypass using a centrifugal pump is often accomplished without heparin or on low-dose heparin. The outflow is from a cannula inserted into the left atrial appendage via the superior pulmonary vein, and the inflow is via a cannula in the left common femoral artery. This bypass is most often used in cases requiring open repair of a blunt injury of the descending thoracic aorta.

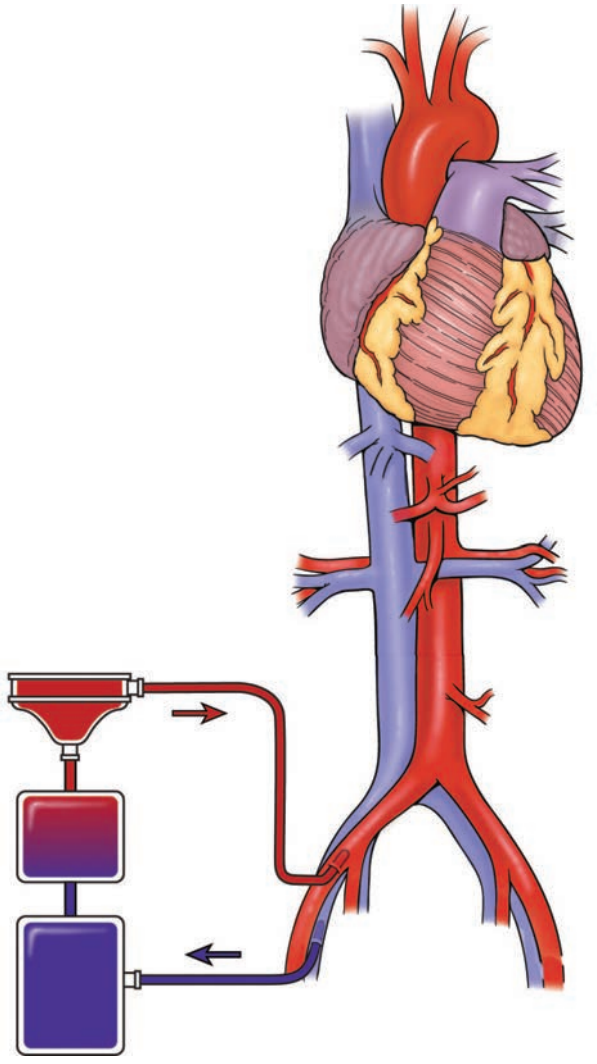


FIGURE 37 Partial Left Heart Bypass 2
One of the options for partial left heart bypass, used for reconstruction of the aorta, is the femoral vein to femoral artery circuit with a centrifugal pump often with an inline oxygenator.

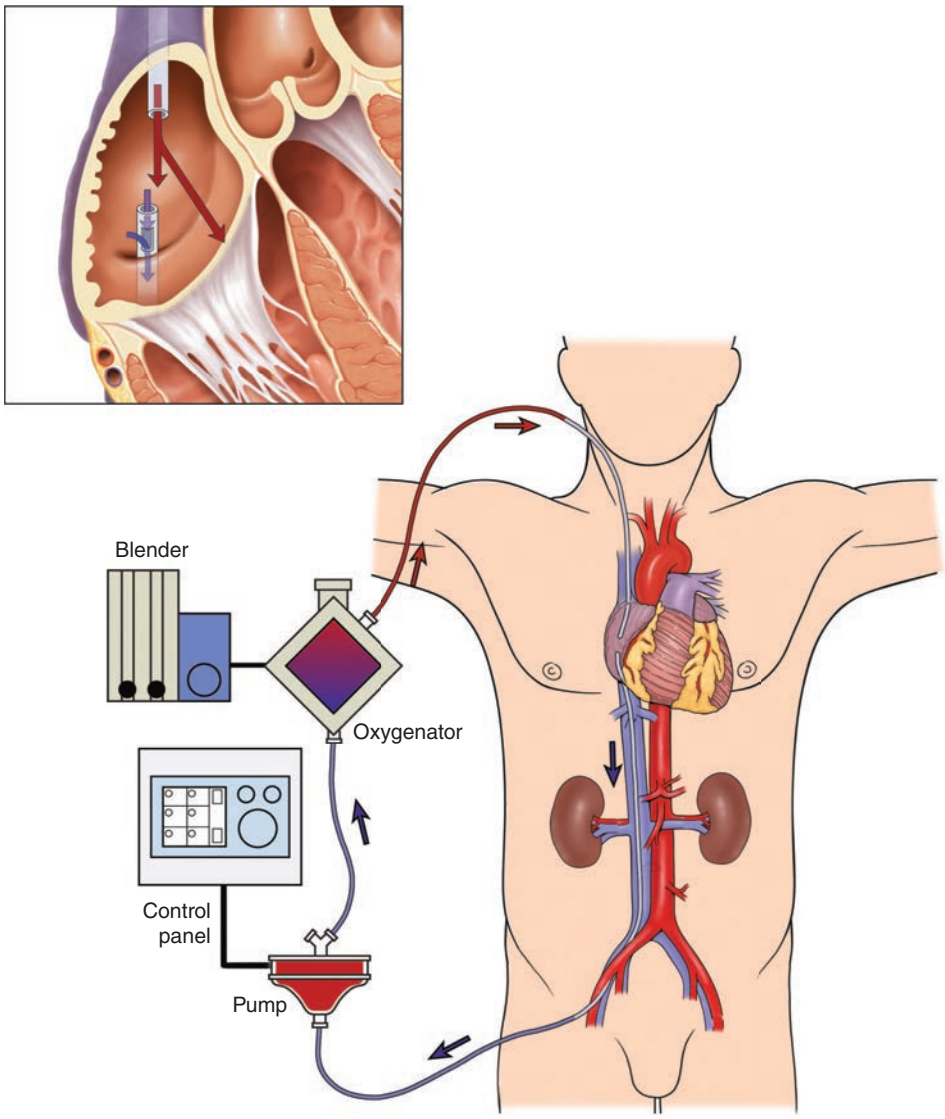


FIGURE 38 ECMO (Extracorporeal Membrane Oxygenation) 1
This drawing demonstrates the two-catheter technique of inserting catheters into the right atrium. The lower catheter is inserted into the femoral vein and threaded into the right atrium for the purpose of draining venous blood to the oxygenation and mixer device. The upper catheter is inserted via the subclavian vein or internal jugular vein into the right atrium, near the tricuspid valve, for the purpose of delivering oxygenated blood into the right-sided circulation of the heart.

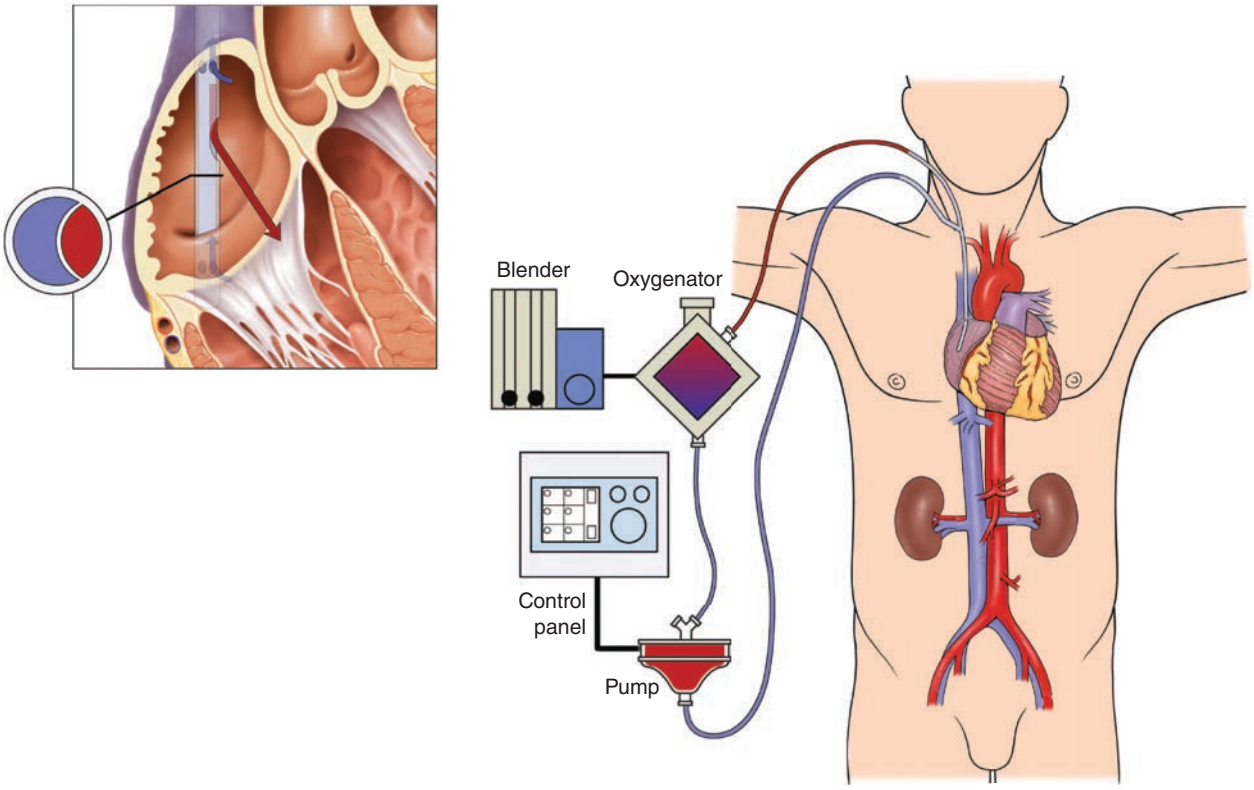


FIGURE 39 ECMO (Extracorporeal Membrane Oxygenation) 2
This drawing demonstrates the single-catheter technique in performing ECMO. Note that a special double lumen catheter is inserted into the right atrium via the internal jugular vein or the subclavian vein, and extended into the intrathoracic inferior vena cava. This single catheter allows for the withdrawal of nonoxygenated blood and the delivery of oxygenated blood after it has been processed via the oxygenator/mixer.

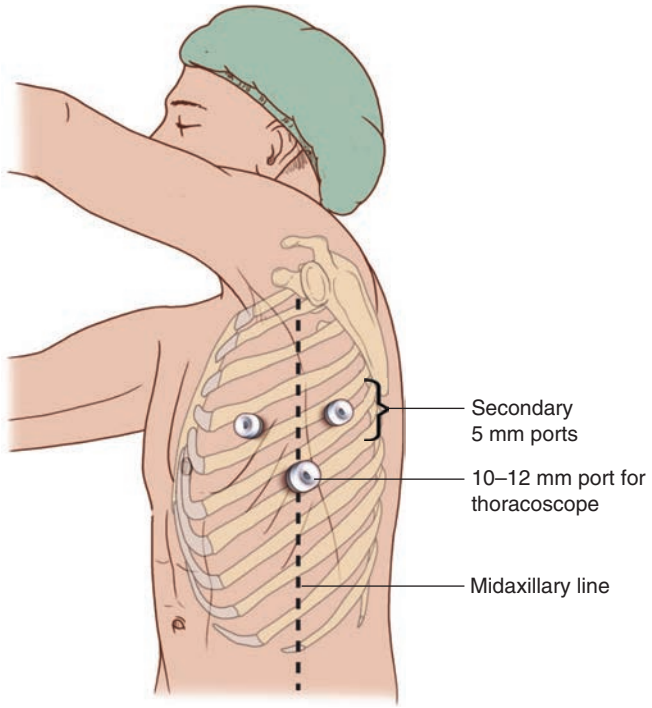


FIGURE 40 VATS (Video-Assisted Thoracoscopic Surgery)
The drawing of a patient in the right lateral decubitus position demonstrates the insertion of ports for performing a VATS.

ABDOMEN AND PELVIS

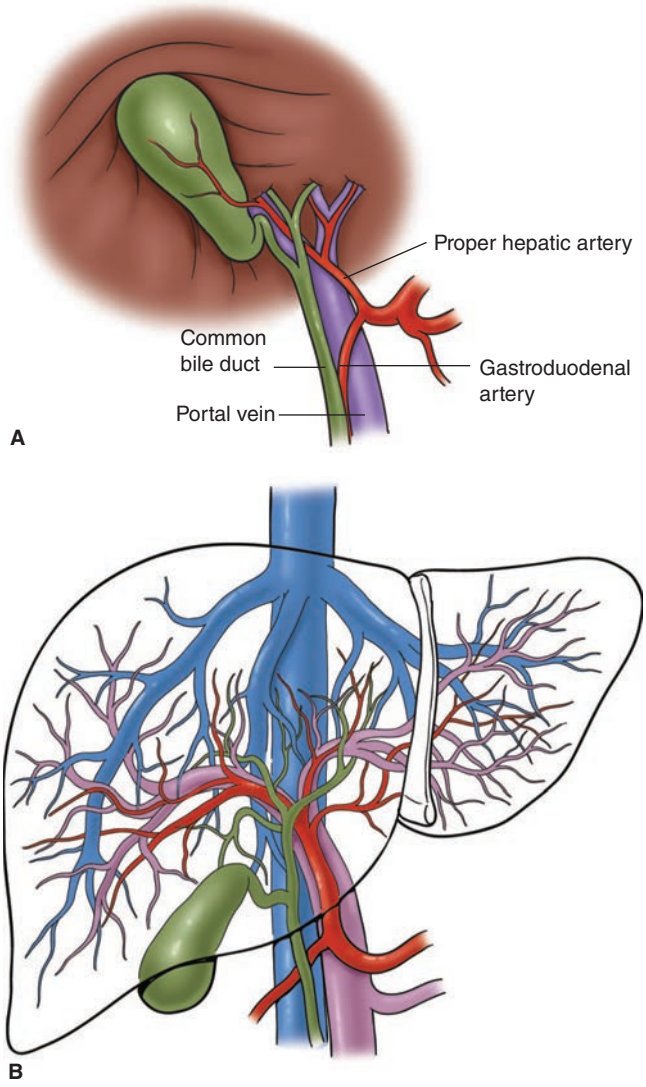


FIGURE 41 Biliary and Vascular Anatomy of the Liver

A. This drawing depicts the anatomy of the gallbladder and porta hepatis. Although many variations may exist, review of this area can be helpful prior to operating on an injury in this location.

B. This drawing illustrates the vascular anatomy of the liver.

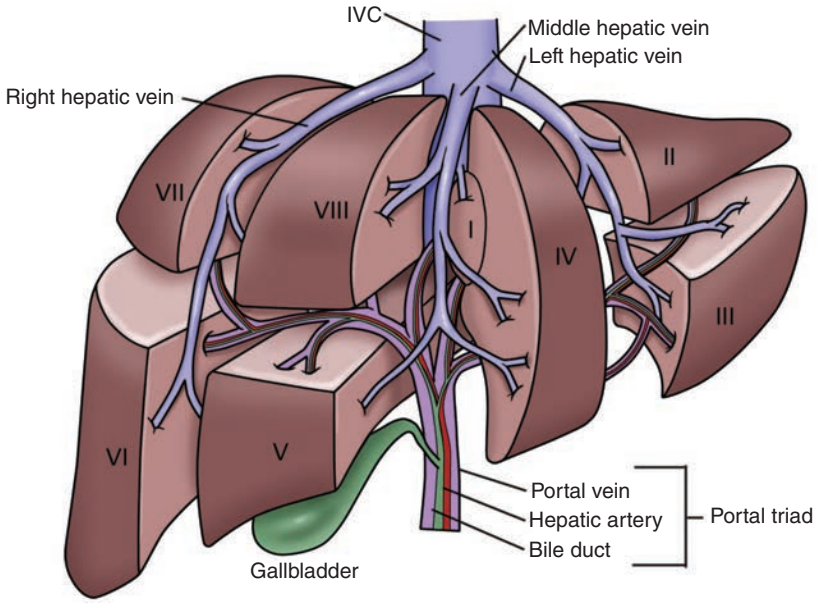


FIGURE 42 Hepatic Segments
These are the eight segments of the liver, defined by their portal circulation. Note that there are at least three major venous outflows from the liver. Not shown here are several direct venous outflows directly from the liver to the retrohepatic IVC.

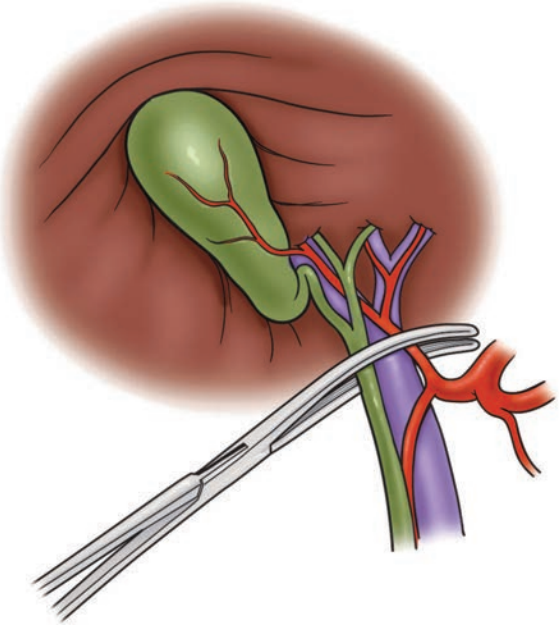


FIGURE 43 The Pringle Maneuver
Using a noncrushing (vascular) clamp, the entire porta hepatis can be occluded to decrease bleeding from an injured liver. This is a temporary occlusion of the portal vein, hepatic artery, and common bile duct. It is accomplished by palpating the foramen of Winslow and precisely placing the clamp on only the desired structures.

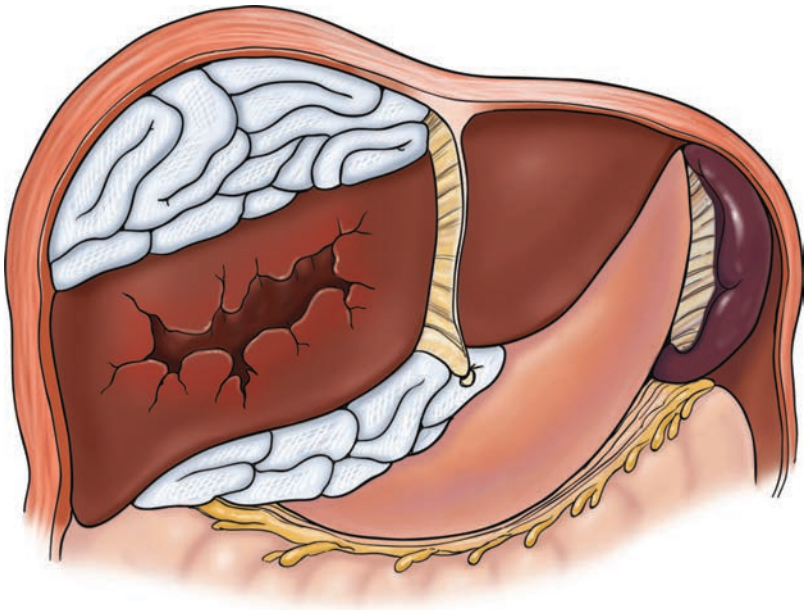


FIGURE 44 Perihepatic Packing
Temporary packing of a bleeding liver is the prime example of damage control. Laparotomy packs are placed above and below a bleeding area in the liver, making a "liver sandwich." Care is taken not to obstruct the inferior vena cava or to produce too much constriction, leading to liver necrosis.

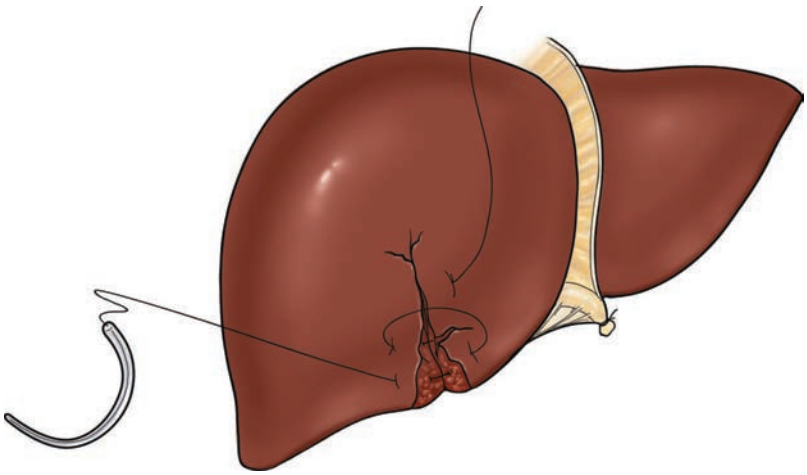


FIGURE 45 Hepatorrhaphy
Using a large, special blunt "liver" needle on an absorbable suture, a deep figure-of-eight suture can stop troublesome bleeding. Sutures should be tied loosely, rather than snug and tight, since the liver swells postoperatively, and tight sutures can cause hepatic necrosis.

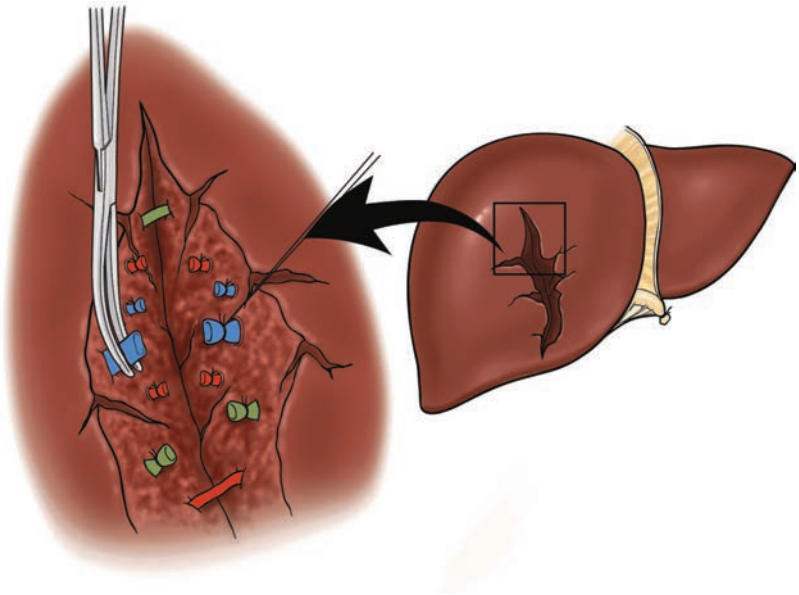


FIGURE 46 Hepatotomy
Gross, large ligatures to the liver may cause liver necrosis and postoperative fever. The injury is unroofed by performing a hepatotomy, with direct ligation of the biliary and vascular structures.

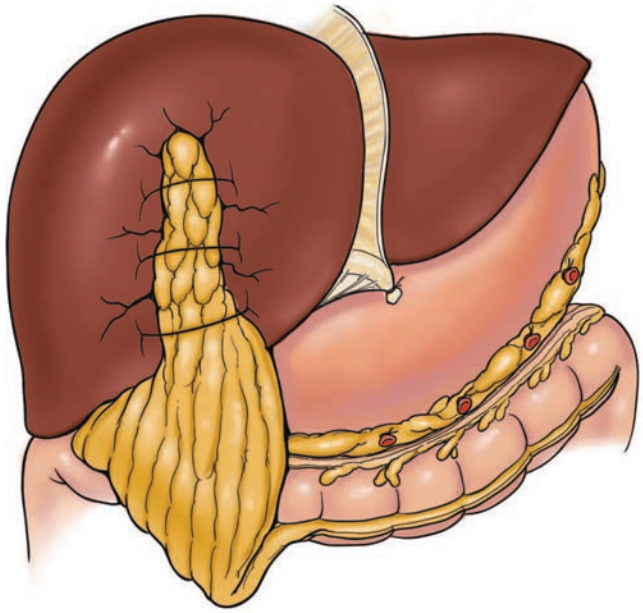


FIGURE 47 Omental Pedicle Packing of Hepatic Laceration or Hepatotomy Site
The left side of the omentum is mobilized off of the transverse colon, preserving a vascular pedicle from the right side of the omentum. The sutures attaching the omentum to the liver are loosely applied, so as not to strangulate the omentum.

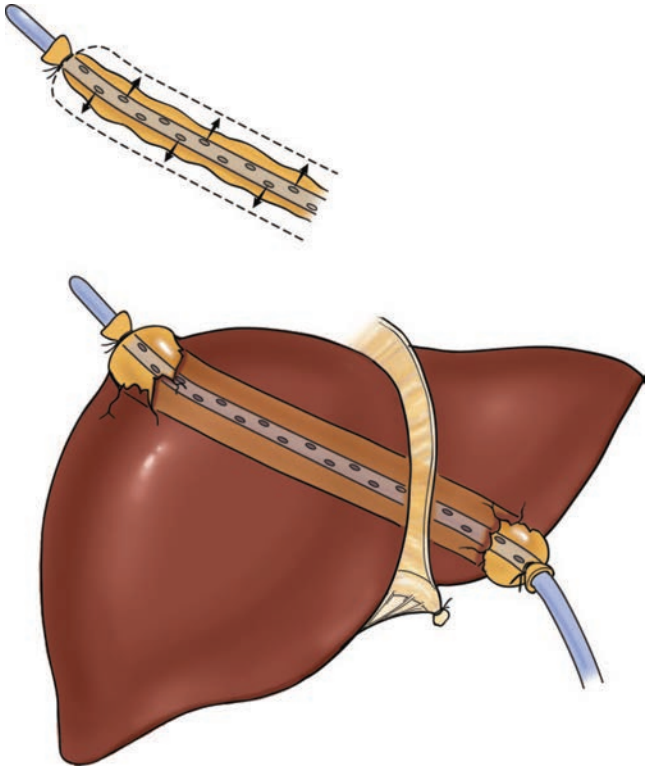


FIGURE 48 Hepatic Balloon Tamponade
A custom, temporary balloon is fashioned using a Penrose drain and a rubber catheter, sealing off the ends of the drain placed over the catheter. After insertion into the body of the liver, the catheter is then filled with saline.

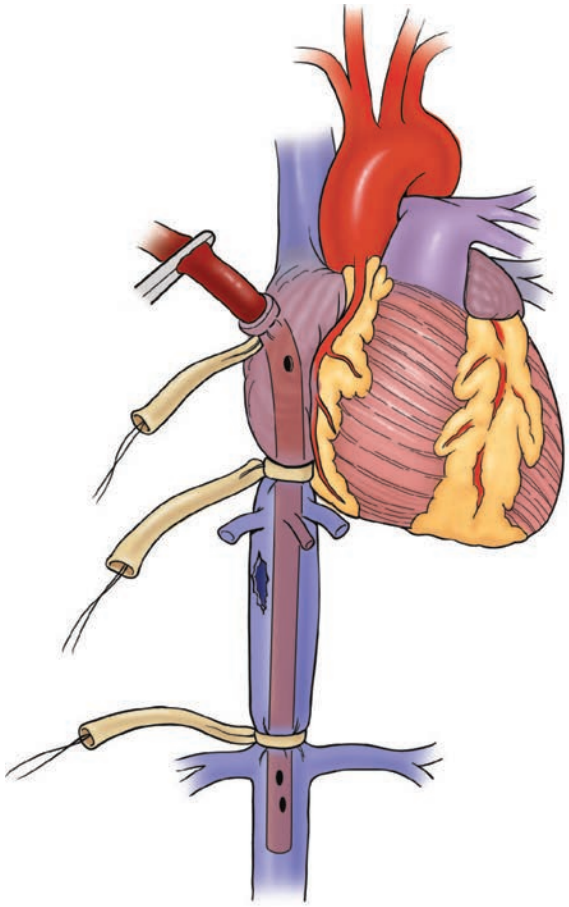


FIGURE 49 Atriocaval Shunt
Isolation of uncontrolled bleeding from the retrohepatic vena cava can be accomplished with an atriocaval shunt. The underlying principle is shunting lower body blood to the right atrium. A 38F chest tube with an extra hole cut near the base end and clamped at the base is inserted via a purse string suture into the right atrial appendage and advanced into the infrarenal inferior vena cava. Assure the last hole in the tube is below the area of the suprarenal inferior vena cava Rumel tourniquet. A second Rumel tourniquet is placed around the intrathoracic intrapericardial inferior vena cava. Finally, a Pringle maneuver accomplishes total hepatic and near-total retrohepatic vena cava vascular isolation to allow repair or reconstruction.

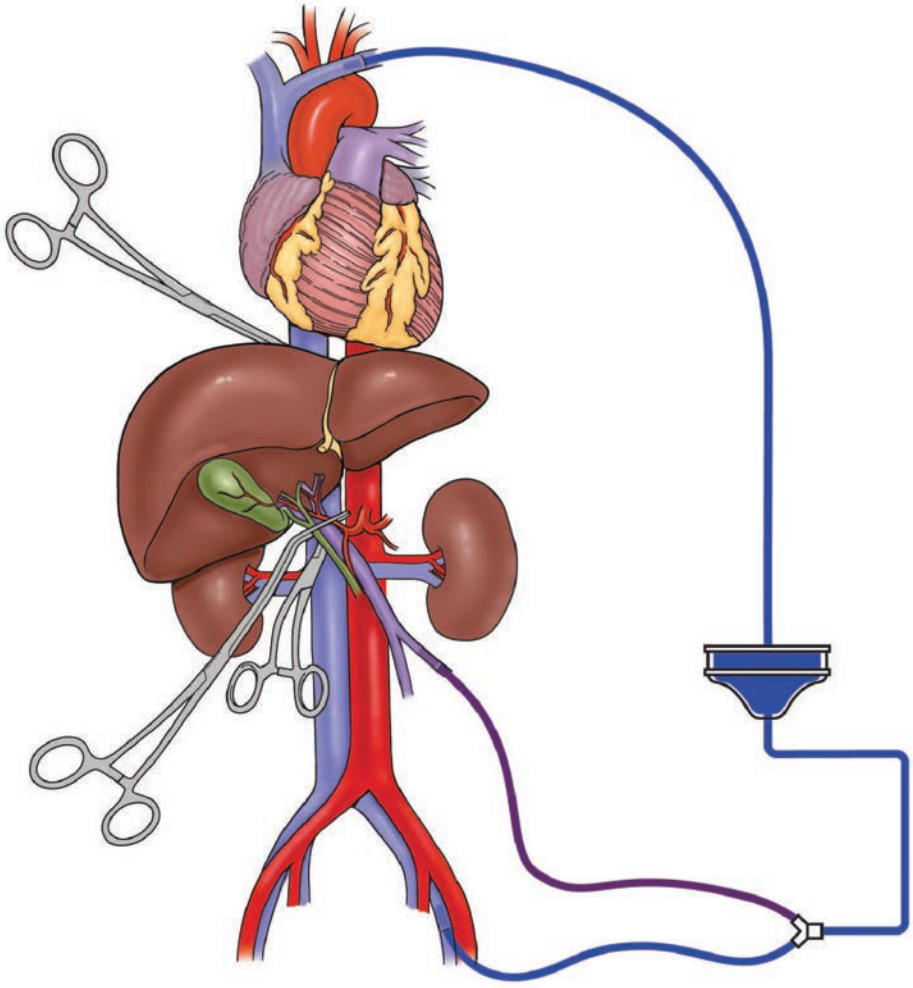


FIGURE 50 Venovenous Bypass
This illustrates a venous bypass circuit using a centrifugal pump for a patient with a complex injury to the retrohepatic vena cava. Cannulae are inserted into the inferior mesenteric, portal, and femoral veins, respectively. This venous blood is pumped, via a centrifugal pump, into the right heart using a cannula inserted into the subclavian vein, innominate vein, or the right atrial appendage.

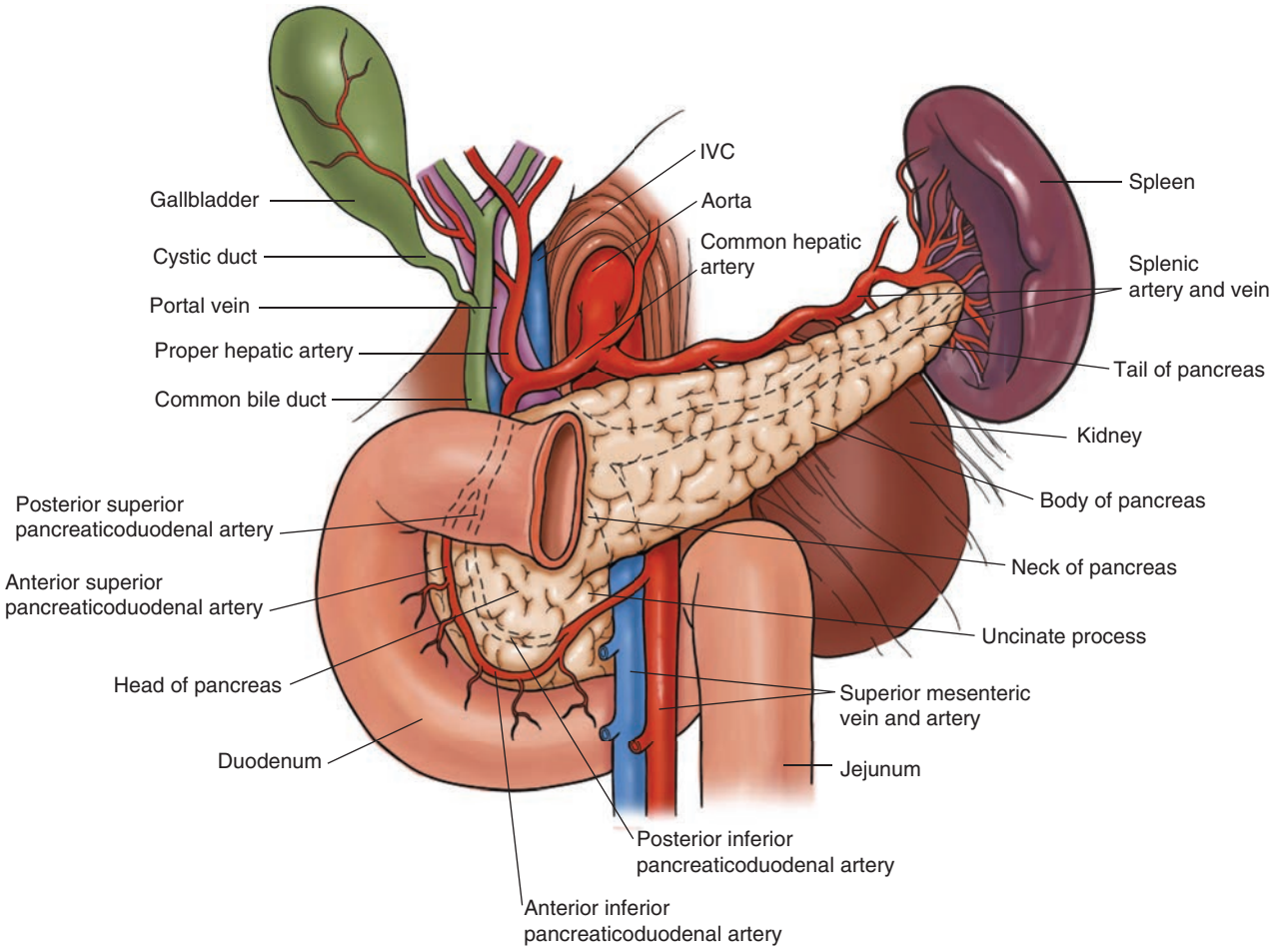


FIGURE 51 Anatomy and Vascular Supply of Duodenum, Pancreas, and Spleen
Complex anatomy of duodenum, pancreas, and spleen in upper abdomen makes management difficult when multiple injuries are present.

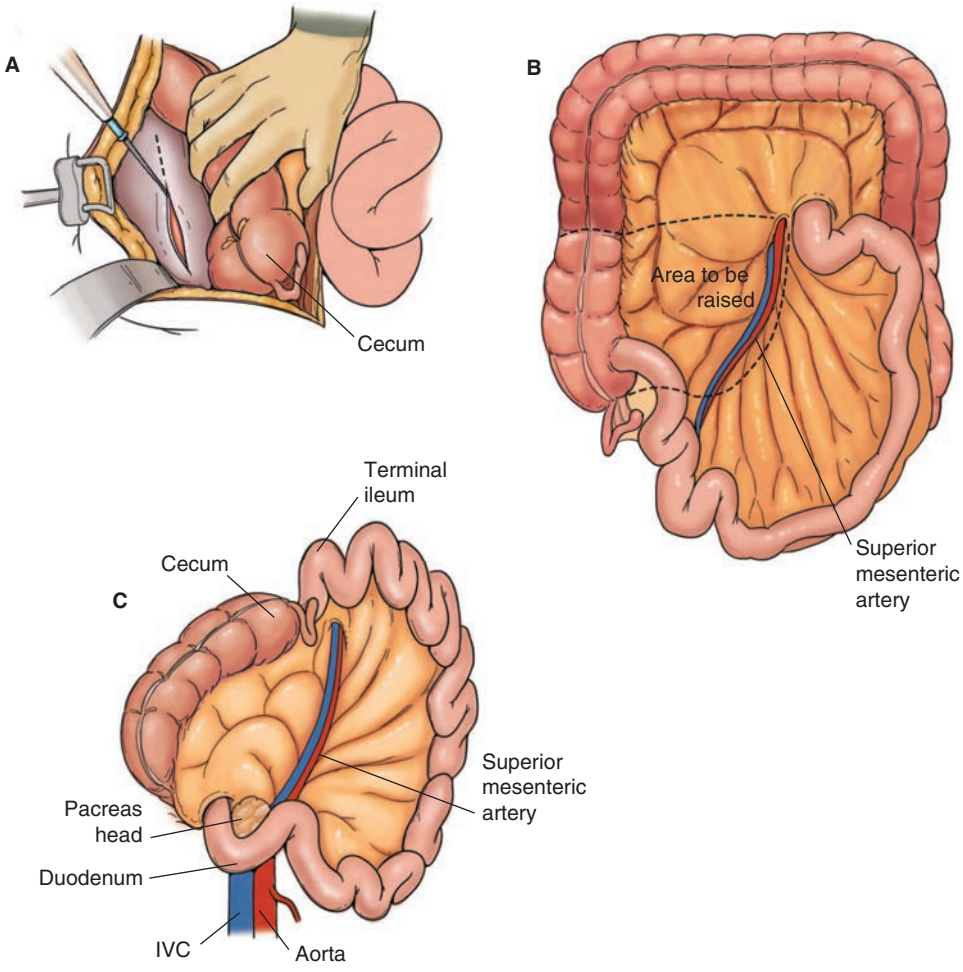


FIGURE 52 Right Medial Visceral Rotation Extended into Cattell-Braasch Maneuver

A. An extensive Kocher maneuver, division of the right line of Toldt, and sharp and blunt dissection toward the midline will expose the posterior head of the pancreas, anterior D_3, infrahepatic inferior vena cava, renal veins, right kidney, and right ureter.

B. Division of the retroperitoneal attachments of the small bowel mesentery from the right lower quadrant to the left upper quadrant completes the Cattell-Braasch maneuver.

C. Using this combination of maneuvers, the structures named above, posterior D_3, and the origin of the superior mesenteric artery can be visualized as well.

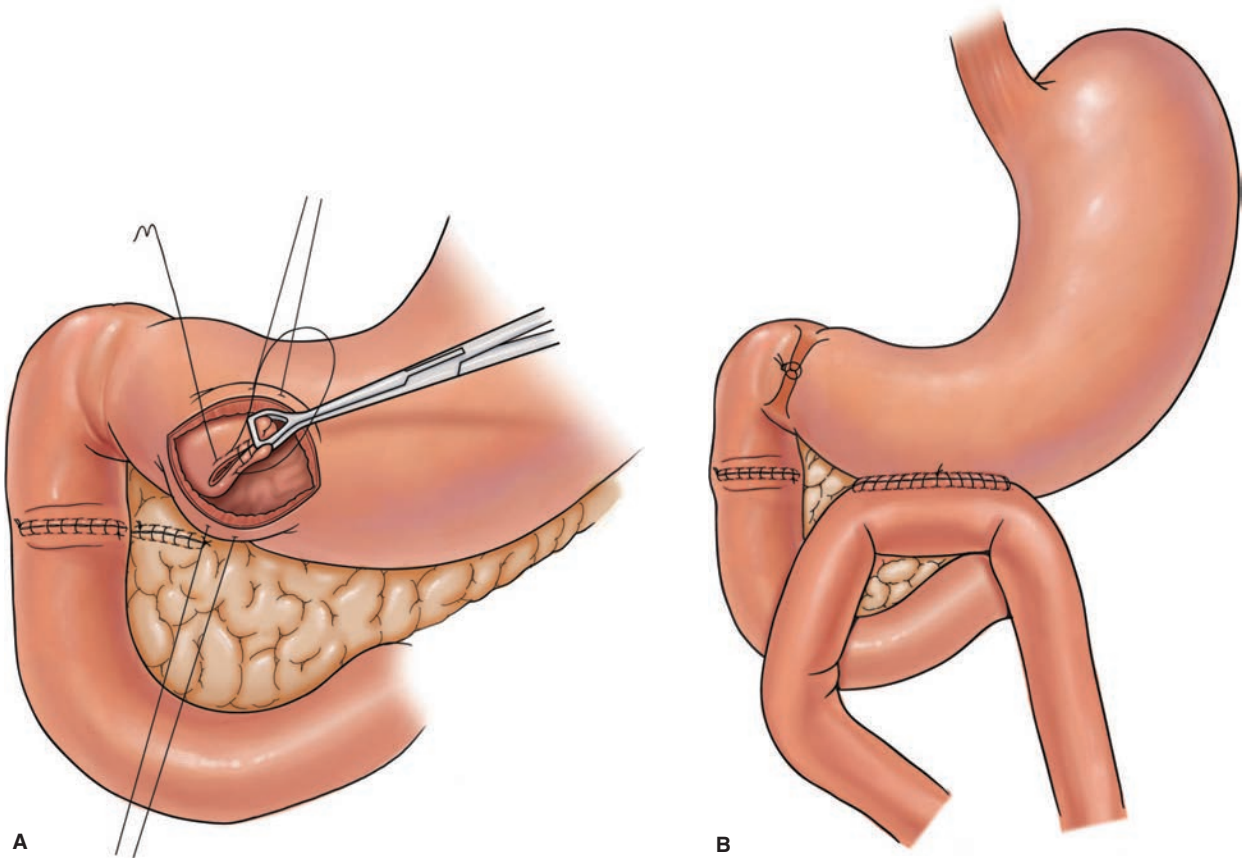


FIGURE 53 Pyloric Exclusion with Gastrojejunostomy

A. For a complex combined injury to the head of the pancreas and duodenum, pyloric exclusion is performed to decrease the risk of a postoperative lateral duodenal fistula. After control of the injury of the duodenum and pancreas has been accomplished, a dependent gastrotomy is made in the antrum of the stomach. The pylorus is palpated and grasped with Babcock clamps and closed with a #1 polypropylene suture. Almost every suture tends to cut through the pylorus, and the gastroduodenal outlet reopens in 2 to 3 weeks.

B. Following closure of the pylorus, a dependent antecolic gastrojejunostomy is created at the gastrotomy site. Patient should be screened for the presence of *Helicobacter pylori* postoperatively and treated if positive.

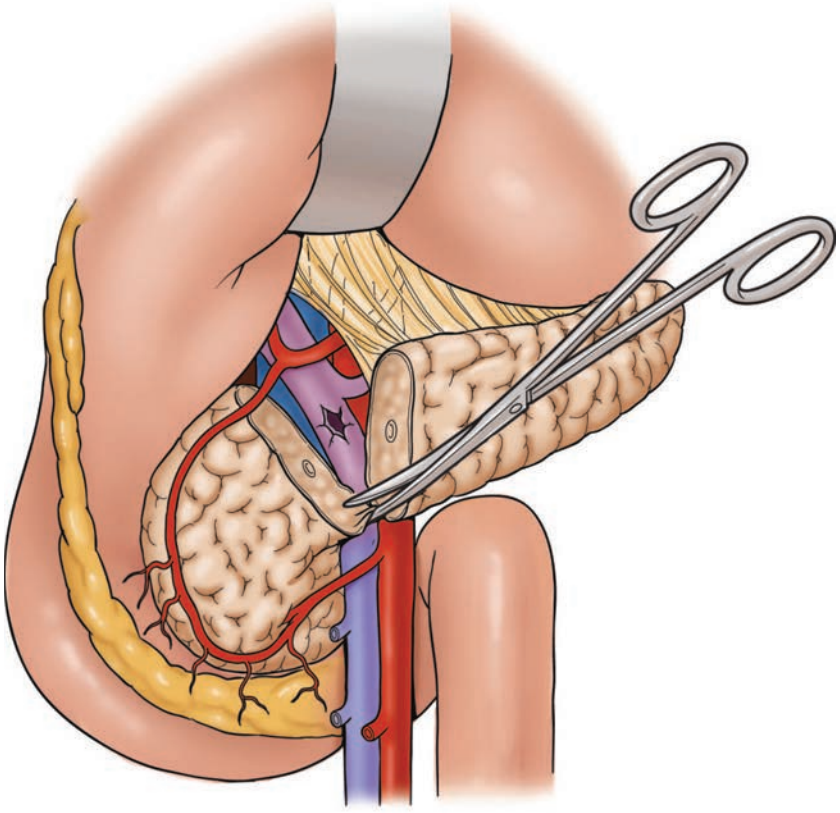


FIGURE 54 Division of Neck of Pancreas to Expose the Portal Vein
The portal vein is formed behind the neck of the pancreas as the superior mesenteric vein is joined by the splenic vein. Bleeding from this area following penetrating trauma presents an exposure problem, but there are very few sizable veins entering the portal vein from the posterior surface of the pancreas. After the superior mesenteric vein is exposed, a dissecting finger can be placed behind the pancreas, and anterior to the superior mesenteric/portal vein. Using scissors or a stapler, the neck of the pancreas is deliberately divided over the dissecting finger under direct vision. This exposes the extent to the proximal portal vein, and appropriate vascular control and repair can be accomplished. The distal end of the pancreas can then be resected with the spleen or reconstructed as a distal end-to-end pancreatojejunostomy.

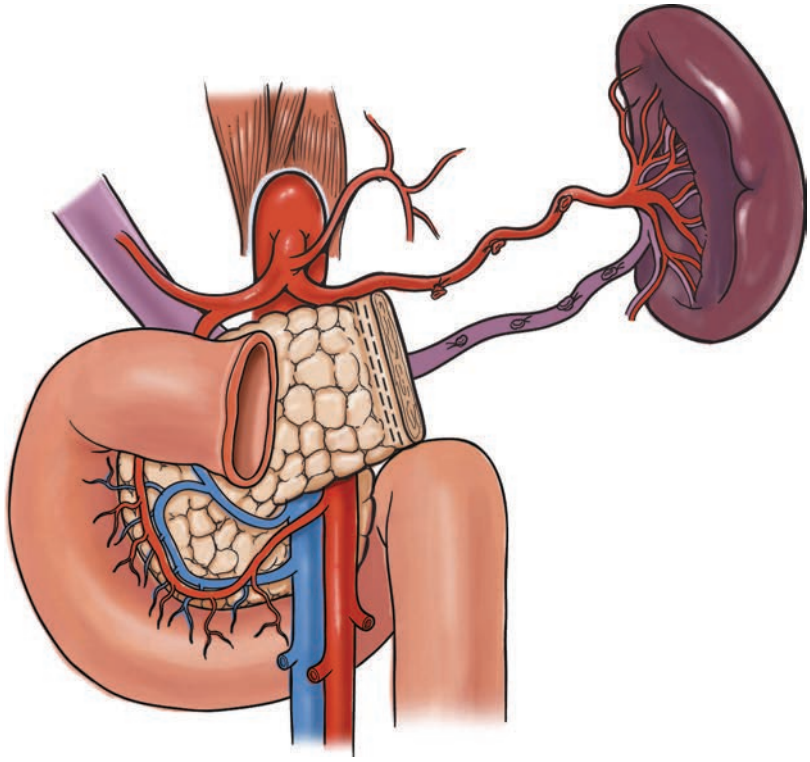


FIGURE 55 Distal Pancreatectomy without Splenectomy in a Child
Because of the immature immune status of children under the age of 2 and possibly up to the age of 10, OIS grade III pancreatic injuries (ductal injury) are treated with spleen-saving distal pancreatectomy.

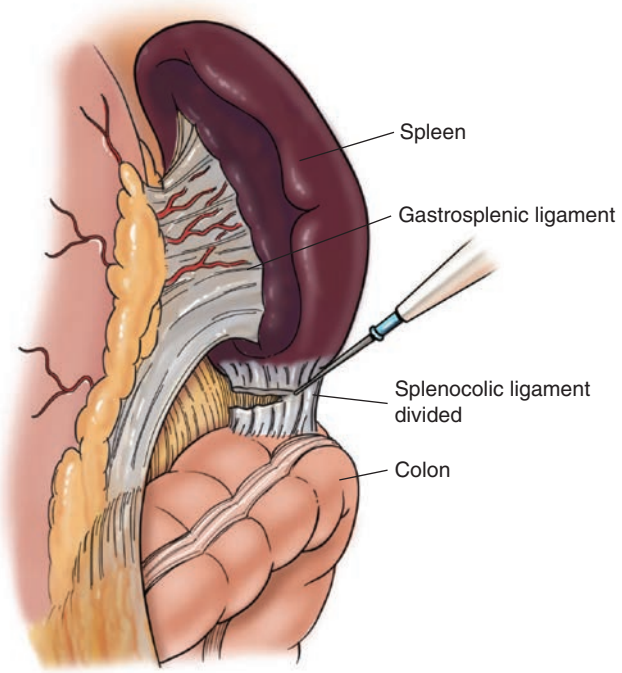


FIGURE 56 Spleen 1
This is the splenic regional anatomy, knowledge of which is necessary to accomplish mobilization of the spleen. The spleen can be mobilized by dividing the splenocolic ligament and ligating the vasa brevia arteries between the stomach and spleen. In this view, the tail of the pancreas and the splenic artery and vein are not demonstrated. Care must be taken to avoid damaging the colon, stomach, and pancreas.

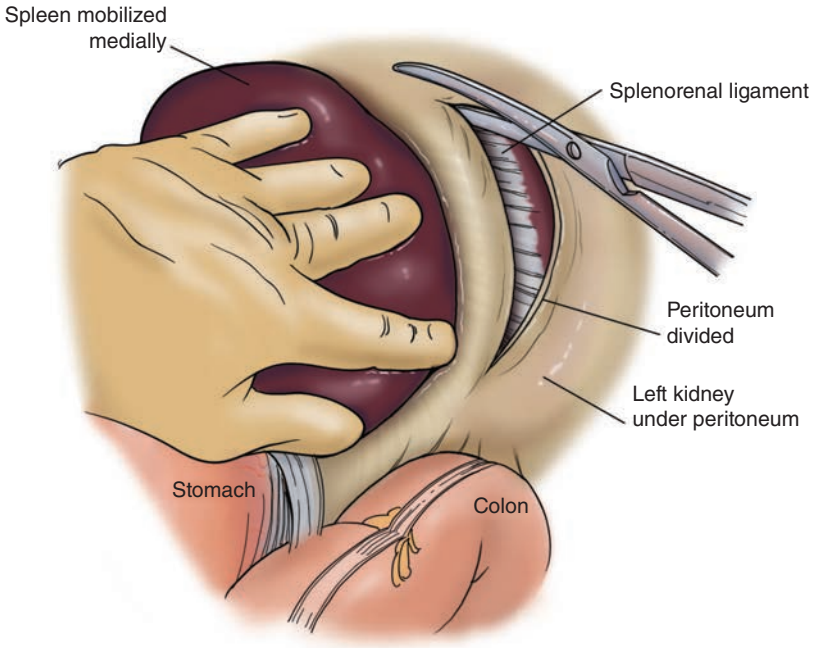


FIGURE 57 Spleen 2
These are additional maneuvers in mobilization of the spleen. Division of the splenorenal ligament and peritoneum overlying the left kidney is illustrated. Often, this maneuver can be accomplished bluntly.

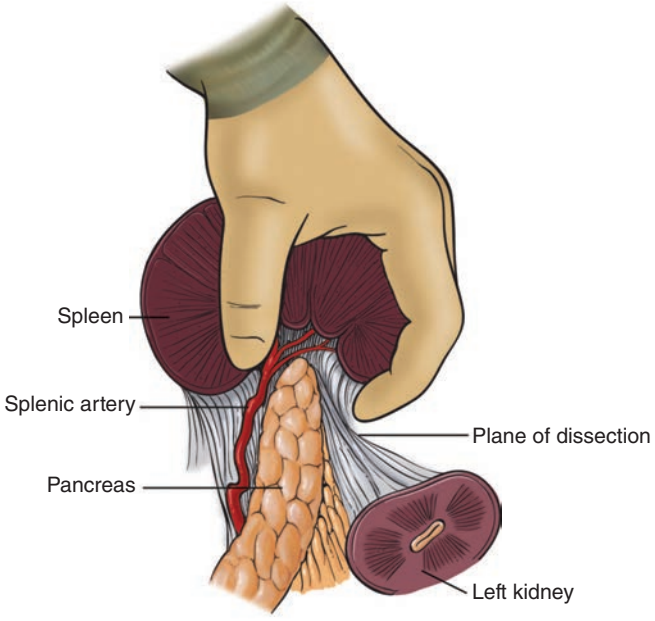


FIGURE 58 Spleen 3
The final movement of the mobilized spleen is by continuing blunt dissection with the tips of the fingers, separating the kidney from the spleen, bringing the tip of the tail of the pancreas and the splenic artery and vein branches in the hilum of the spleen to the midline of the anterior abdomen. In this location, these vessels can be divided, with care taken to avoid injury to the tail of the pancreas.

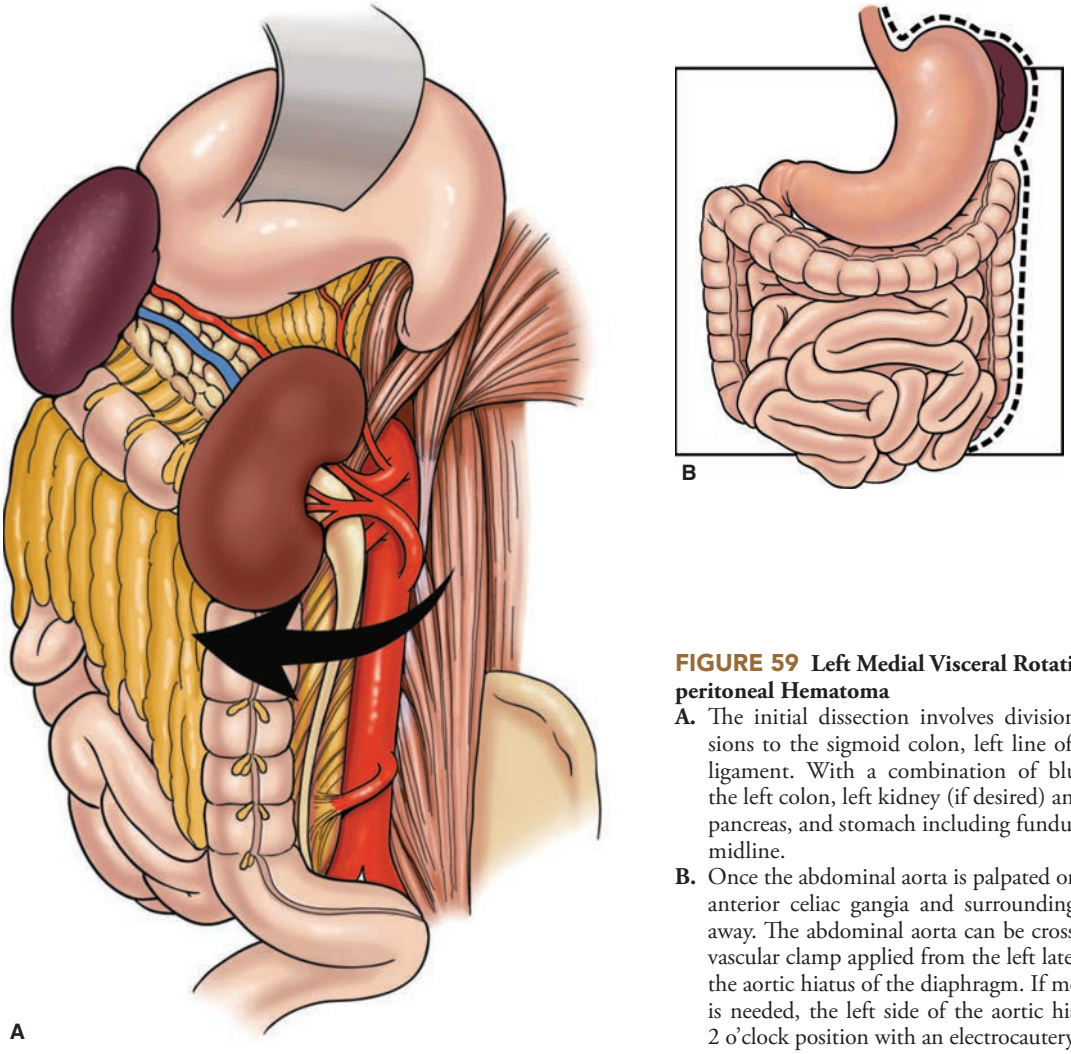


FIGURE 59 Left Medial Visceral Rotation for Suprarenal Retroperitoneal Hematoma
A. The initial dissection involves division of the congenital adhesions to the sigmoid colon, left line of Toldt, and the lienorenal ligament. With a combination of blunt and sharp dissection, the left colon, left kidney (if desired) and ureter, spleen, tail of the pancreas, and stomach including fundus are mobilized toward the midline.
B. Once the abdominal aorta is palpated on the lumbar vertebrae, the anterior celiac gangia and surrounding lymphatics are dissected away. The abdominal aorta can be cross-clamped with a DeBakey vascular clamp applied from the left lateral position just inferior to the aortic hiatus of the diaphragm. If more proximal aortic control is needed, the left side of the aortic hiatus can be divided at the 2 o'clock position with an electrocautery.

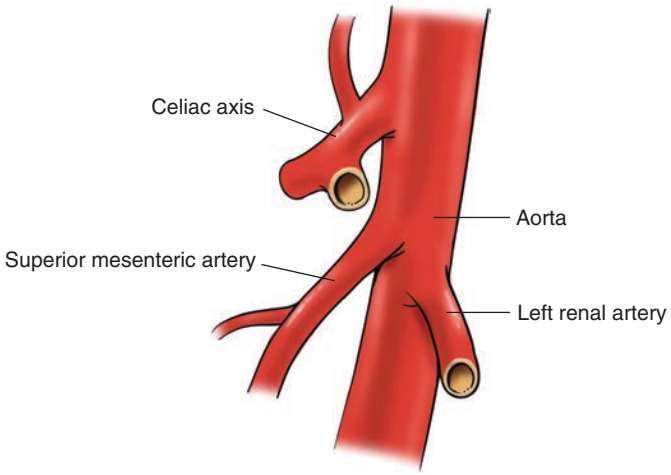


FIGURE 60 Superior Mesenteric Artery
This is the surgical anatomy of the celiac axis, superior mesenteric artery (SMA), and renal arteries. Note that the muscle decussations at the celiac axis are contiguous with the diaphragmatic crura and are located at the level of the diaphragm. Also note the very close proximity of these four arteries. The celiac ganglia around the celiac axis and SMA can be quite thickened and appear as fibrous tissue. They are composed of nerves coming from the greater, middle, and lesser splanchnic nerves that originate from thoracic vertebrae.

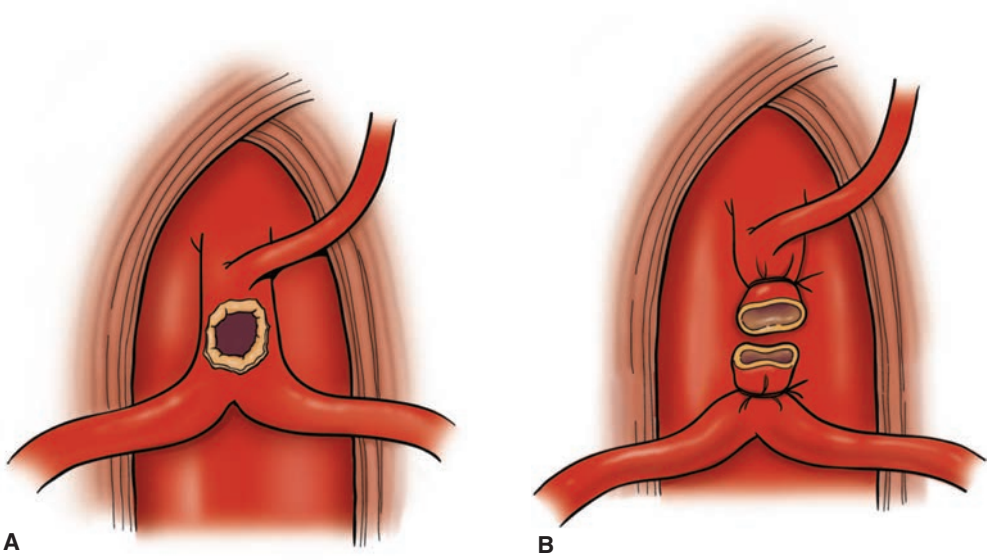


FIGURE 61 Bleeding from the Celiac Axis
A. Drawing illustrating injury to the celiac axis.
B. Ligation of celiac axis with preservation of left gastric artery.

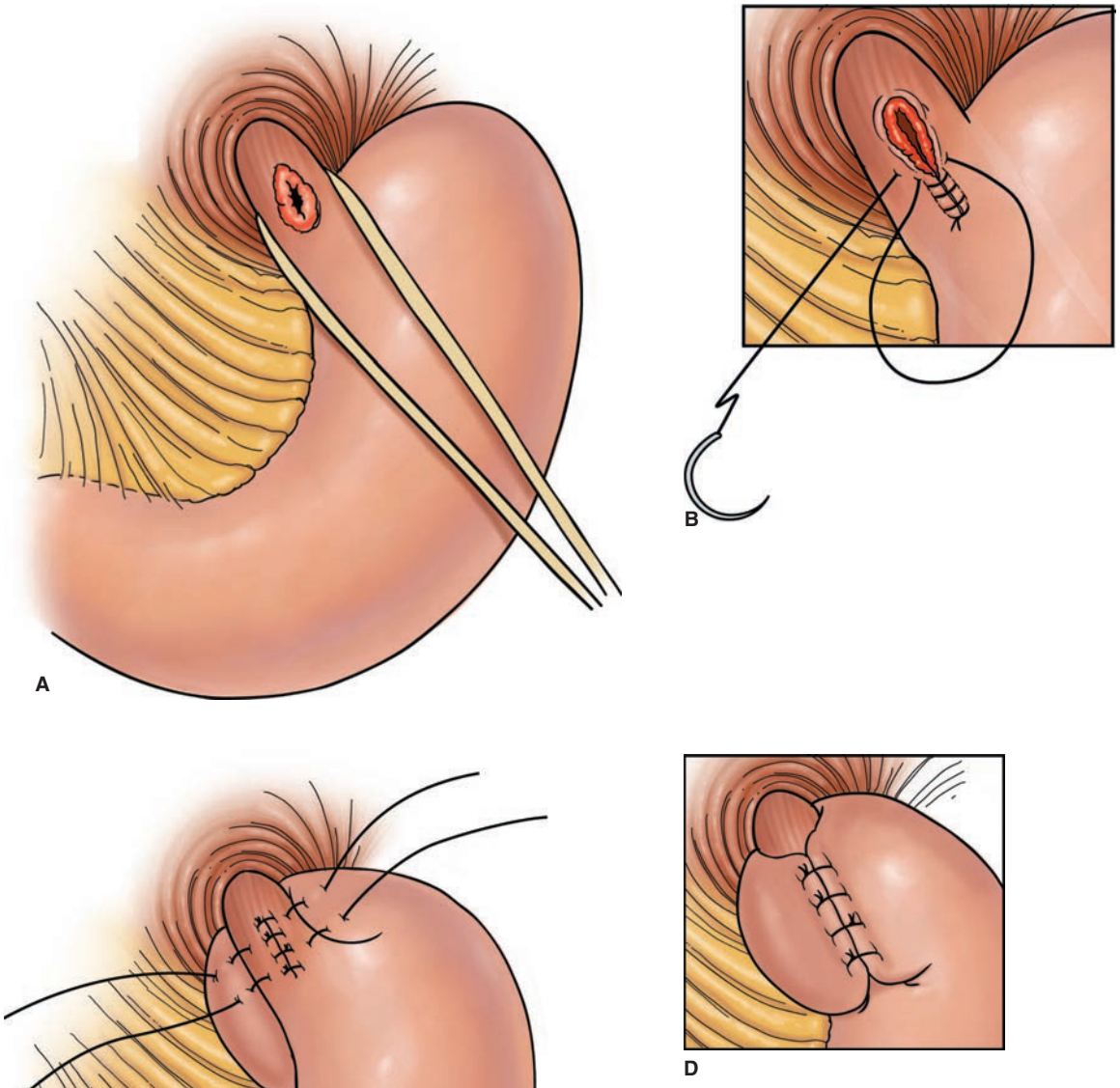


FIGURE 62 Exposure and Repair of an Injury at the Esophagogastric Junction

A. High gastric injuries can be difficult to expose and repair. Following mobilization of the left lobe of the liver by lysing the left triangular ligament, the liver is retracted and the esophagus encircled with a Penrose drain for downward traction.

B. Anterior holes can be suture repaired, taking care not to cause a stricture at this location.

C–D. For an extensive esophagogastric junction injury, the greater curvature of the stomach is mobilized, ligating the vasa brevia vessels, and a fundoplication might be added for a more secure closure.

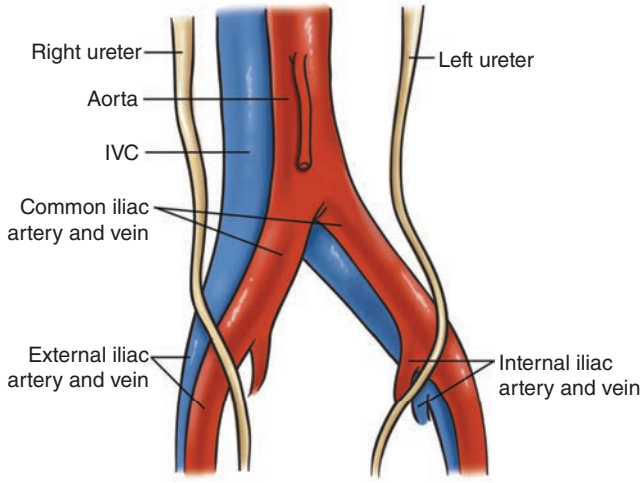


FIGURE 63 **Iliac Anatomy**
This illustrates the distal abdominal aorta, the proximal inferior vena cava, and the common and external iliac arteries, as well as the accompanying iliac veins. The orifices of the internal iliac arteries and veins can be seen as the ureters cross anterior to the iliac arteries at this location.

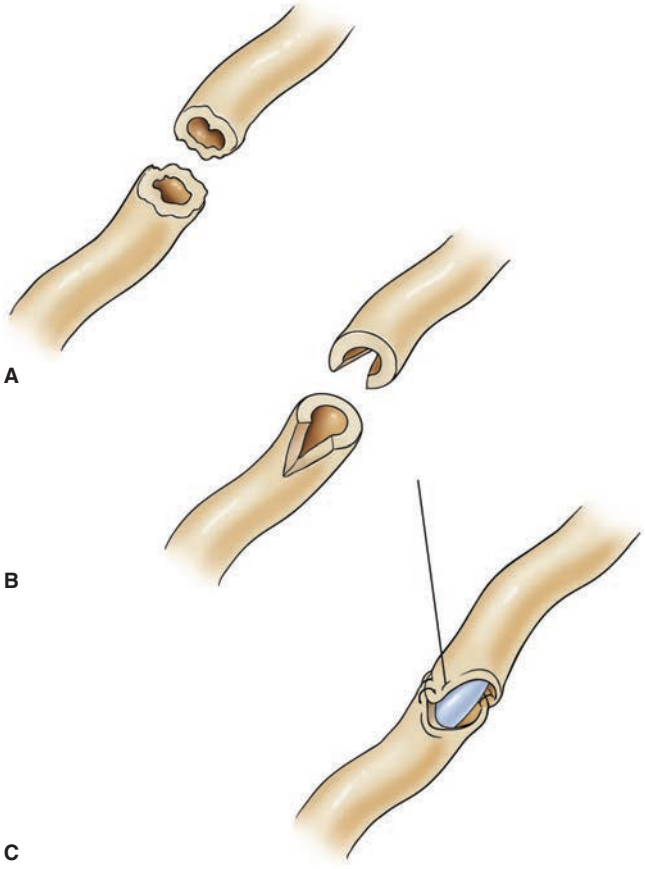
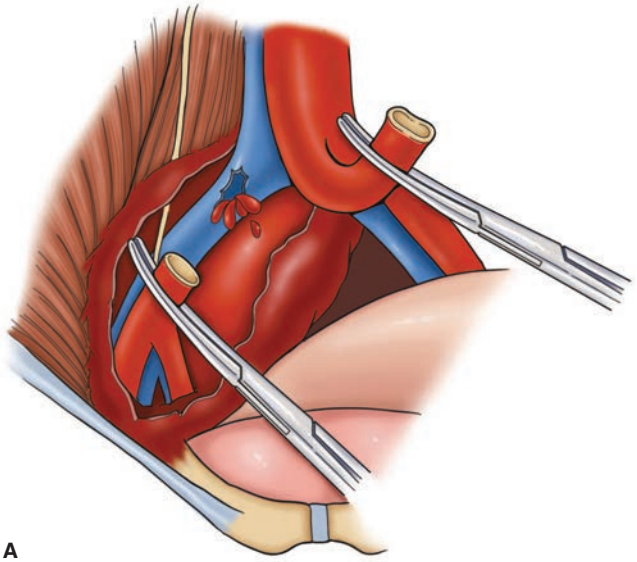


FIGURE 64 **Repair of the Transected Ureter**
A. Drawing of a transected ureter.
B. Spatulated transected ureter.
C. Direct absorbable suture repair of transected ureter over stent.



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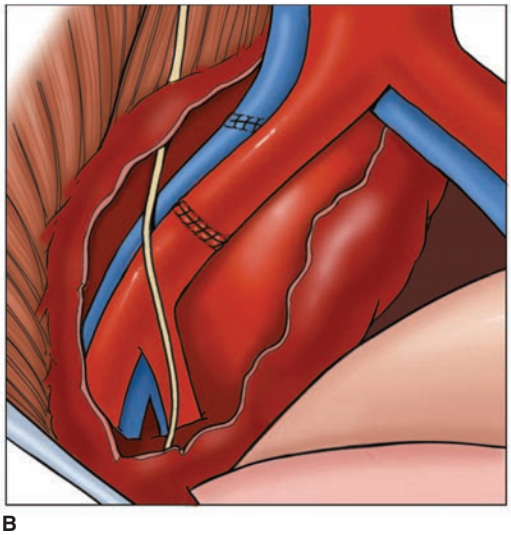
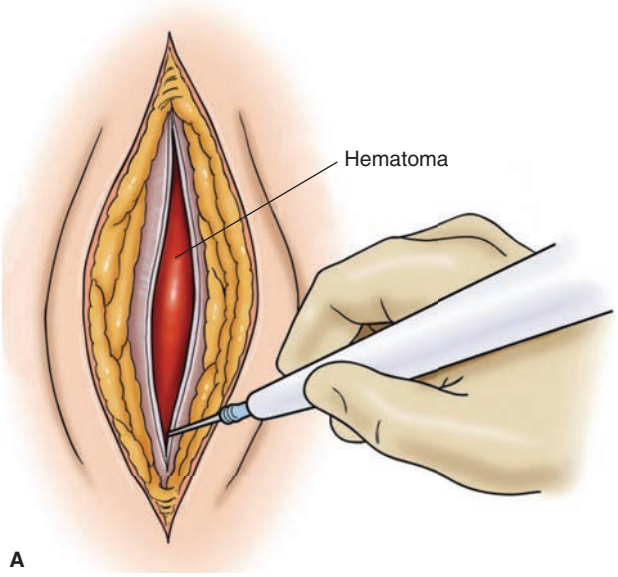


B

FIGURE 65 Deliberate Division of the Right Common Iliac Artery

A. Exposure of an injury to the right common iliac vein is very difficult. Deliberate division of right common iliac artery between vascular clamps facilitates exposure and repair of an underlying venous injury.

B. Following venous control, the right common iliac artery is reanastomosed.



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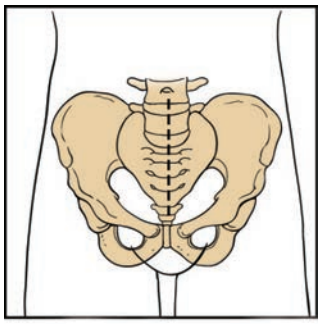
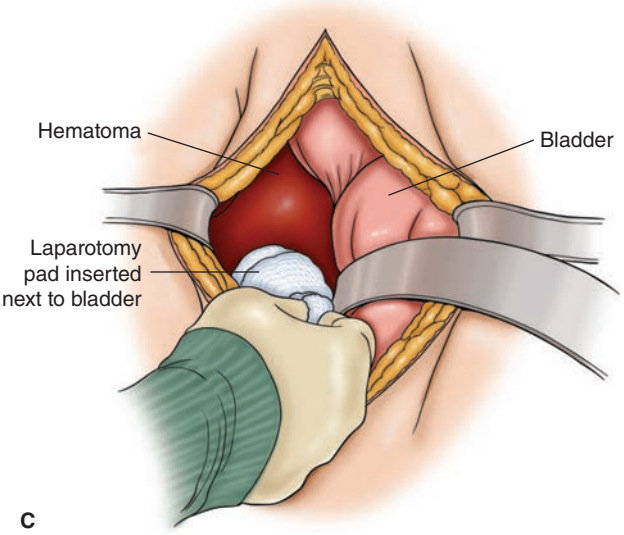


B 6-8 cm midline incision



C

FIGURE 66 Pelvic Packing for Fracture with Retroperitoneal Bleeding

A–B. Through a low midline incision, the area of the extraperitoneal bladder is approached, taking care to stay outside the peritoneal cavity.

C. Any free blood is removed, and the sides of the bladder are retracted.

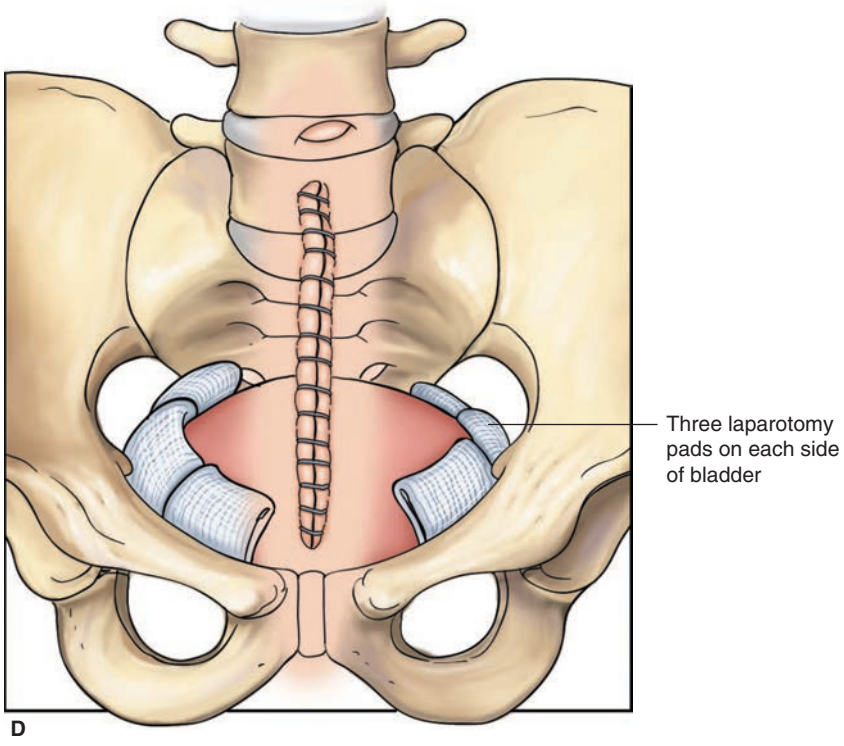


FIGURE 66 Pelvic Packing for Fracture with Retroperitoneal Bleeding (*Continued*)

D. Three laparotomy pads are placed on each side, lateral and posterior to the bladder. The laparotomy pads are left in place as a damage control tactic (to be removed later), and the lower midline incision is closed. Some surgeons also ligate the internal iliac arteries bilaterally. Others recommend leaving these vessels intact to provide a route for embolization should bleeding continue.

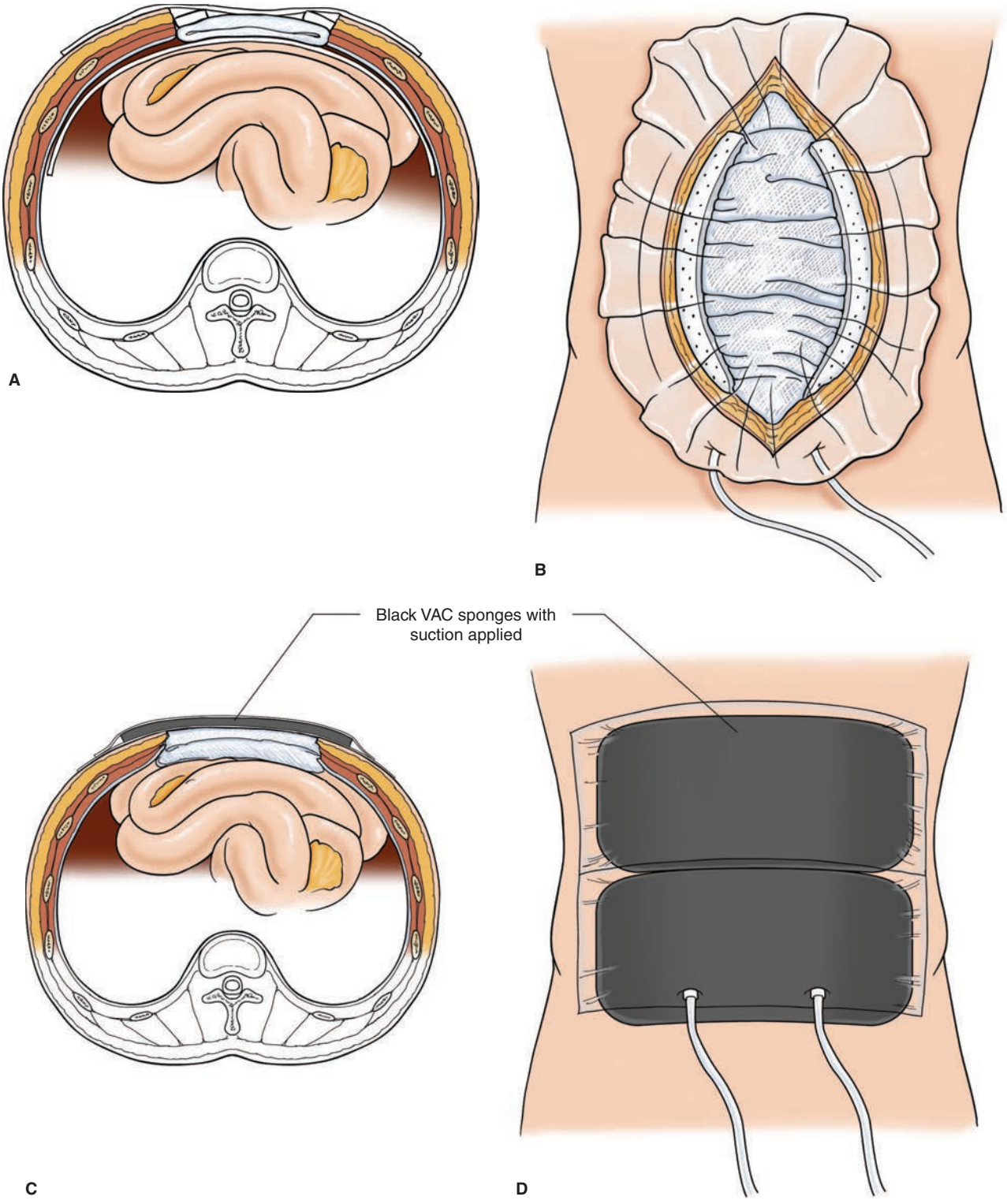


FIGURE 67 Temporary Abdominal Wall Closure Following Damage Control Laparotomy

A. This is the first of a series of drawings demonstrating approaches to management of the abdominal wall after a damage control laparotomy and one in which there is concern for an abdominal compartment syndrome. A transverse depiction of the abdomen is shown with a plastic drape covering the intestines, a series of cloth packings in the middle of the incision, and a plastic drape covering over the entire abdomen, with this drape resting on the skin.

B–D. In **B,** the covering is seen from a frontal view and two suction tubes are seen to emerge from the lower abdomen. Alternatively to the drawings using available pads, plastic sheeting, and suction tubing in every operating room, some surgeons, even at the first operation, might choose to use the same principle, but employ a commercially available device (**C** and **D**). Other surgeons might elect to use such a commercial device at the second or third damage control laparotomy.

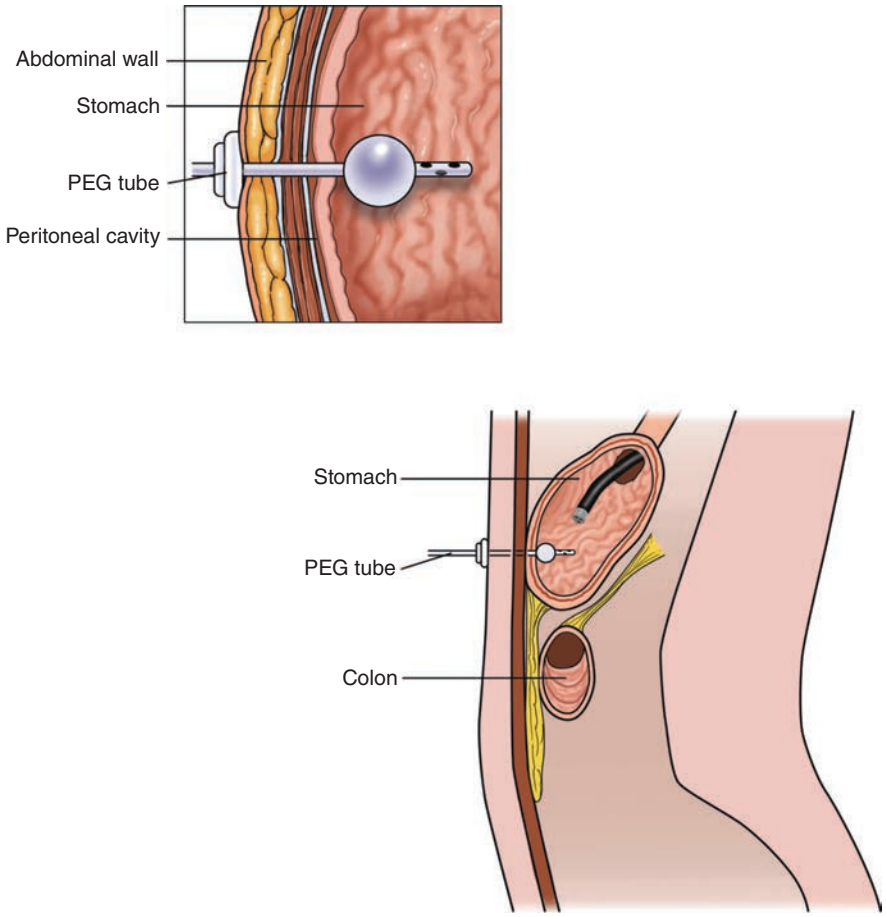


FIGURE 68 PEG (Percutaneous Endoscopic Gastrostomy)
A feeding gastrostomy can be placed with the guidance of a gastroscope to confirm optimal position of the tube. A snare is introduced via the gastroscope to pull a percutaneous wire introduced in the left subcostal region where the stomach transilluminates. The gastrostomy tube is then pulled transorally into the stomach.

EXTREMITY VASCULAR

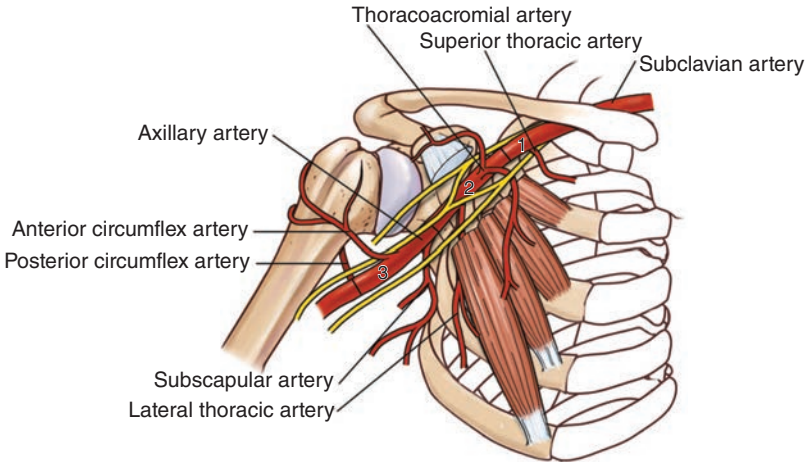


FIGURE 69 Axillary Artery Anatomy
The axillary artery has six branches. The pectoralis major and pectoralis minor muscles, which cover the axilla, are not shown in this drawing.

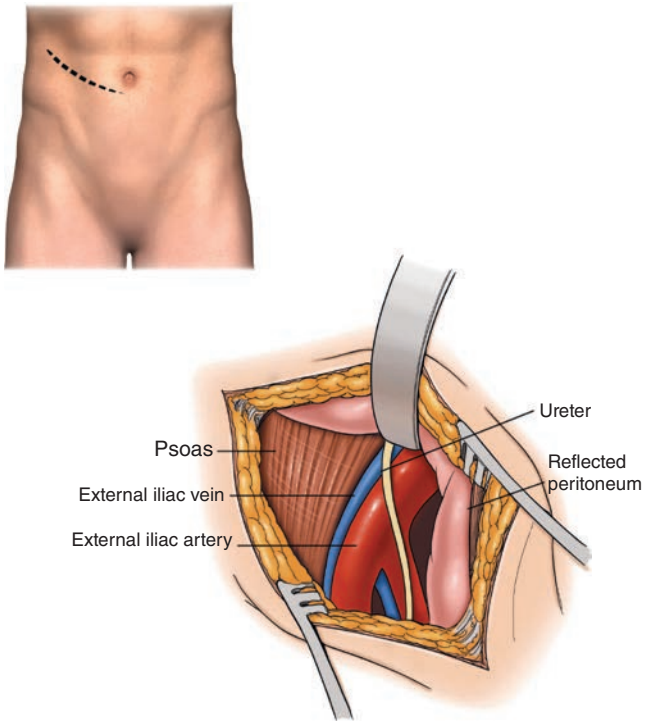


FIGURE 70 Retroperitoneal Exposure of Iliac Artery
This is a lateral lower abdominal incision often used for kidney transplants, which allows for exposure of the retroperitoneal fossa at the bifurcation of the iliac artery. The common, external, and internal iliac arteries are visualized, with the accompanying iliac veins behind the arteries. Note that the ureter crosses anterior to the iliac artery where the iliac artery bifurcates.

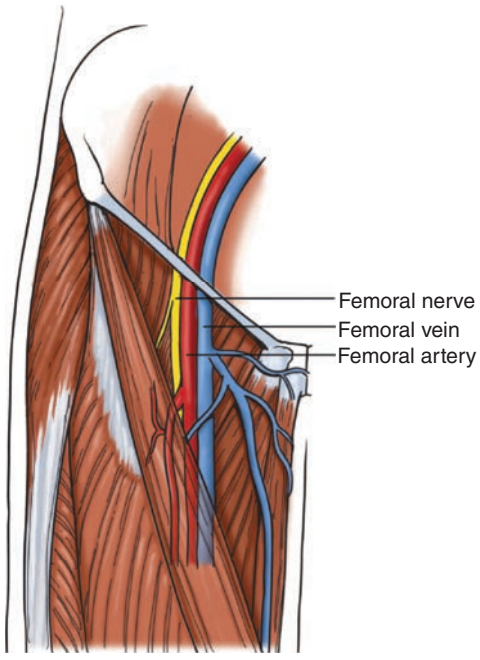


FIGURE 71 Femoral Artery
This is the anatomy of the femoral nerve, artery, and vein, just distal to the inguinal ligament. These anatomical relationships are important to remember when inserting various lines and catheters. Note the arterial branches just proximal to the inguinal ligament. These can produce troublesome bleeding if inadvertently injured during dissection.

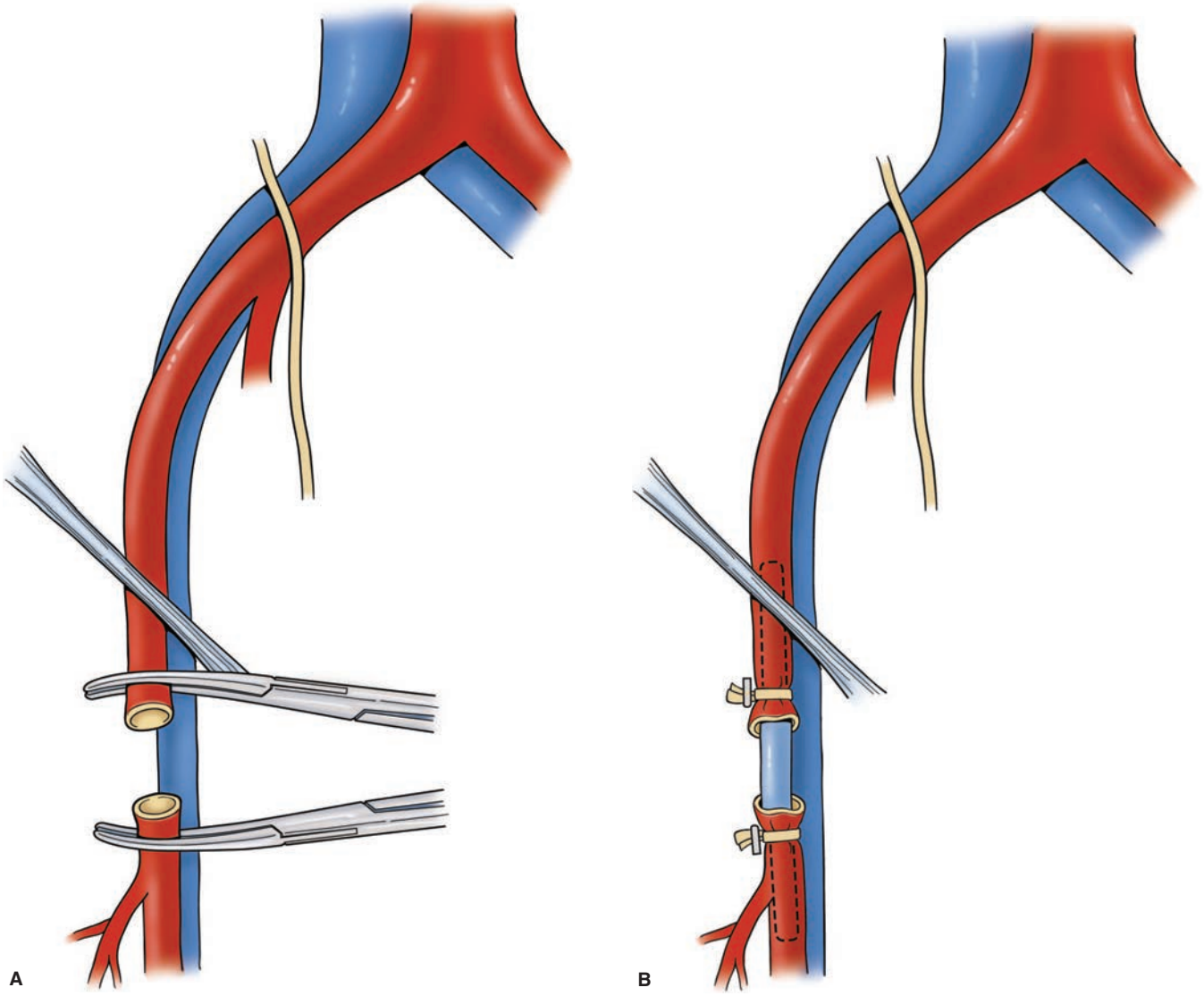


FIGURE 72 Temporary Intraluminal Vascular Shunt

A. Used in most major named vessels of the body, a temporary intravascular shunt is pictured here in the common femoral artery. This vascular damage control tactic provides oxygenated blood distal to a vascular injury, while other injuries are addressed or, if necessary, the patient is transferred for a higher level of care.

B. Using appropriately sized plastic tubing, that is, commercially available carotid shunts, the shunt is secured with encircling tapes.

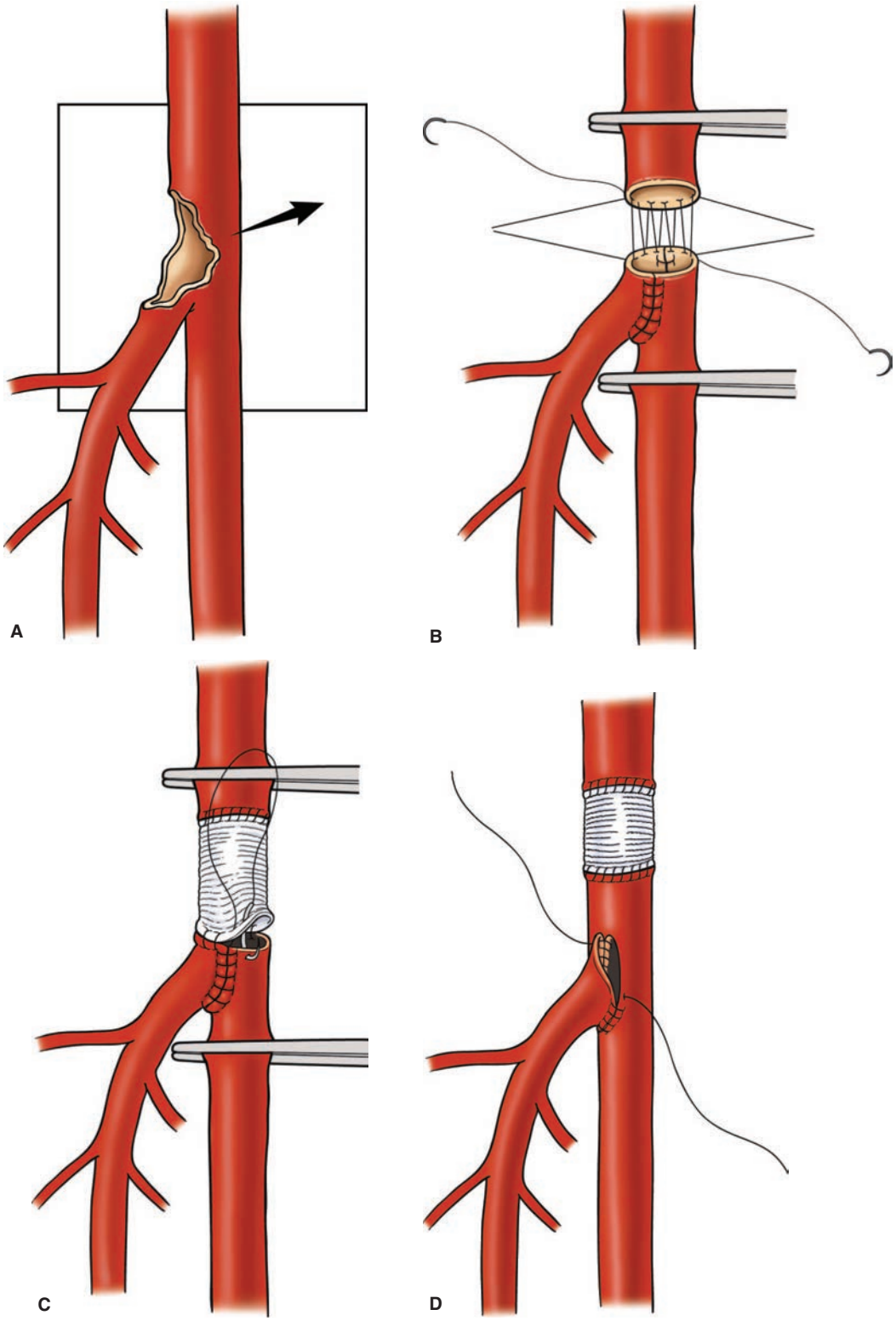


FIGURE 73 Complex Reconstruction of the Femoral Artery

A. Drawing illustrating a complex injury to the femoral artery.

B. The area of injury is debrided, and the sides of the superficial femoral and profunda femoris arteries are sewn together to produce a large single orifice. Depending on the tension, an end-to-end anastomosis can be performed.

C. The artery may also be reconstructed using an interposition graft, here depicted with Dacron.

D. On occasion, a bridge of prosthetic material can be sutured to the proximal superficial femoral artery, with the foreshortened profunda femoris artery sutured end-to-side to a more distal location in the superficial femoral artery.

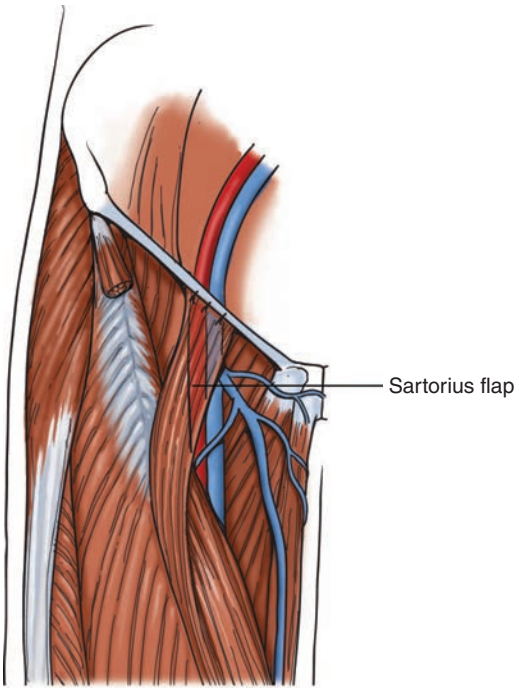


FIGURE 74 Sartorius Muscle Flap in the Groin
This depicts the anatomy of the sartorius muscle, with its accompanying arterial supply. A flap of the sartorius muscle is useful to cover nearby vessels and nerves.

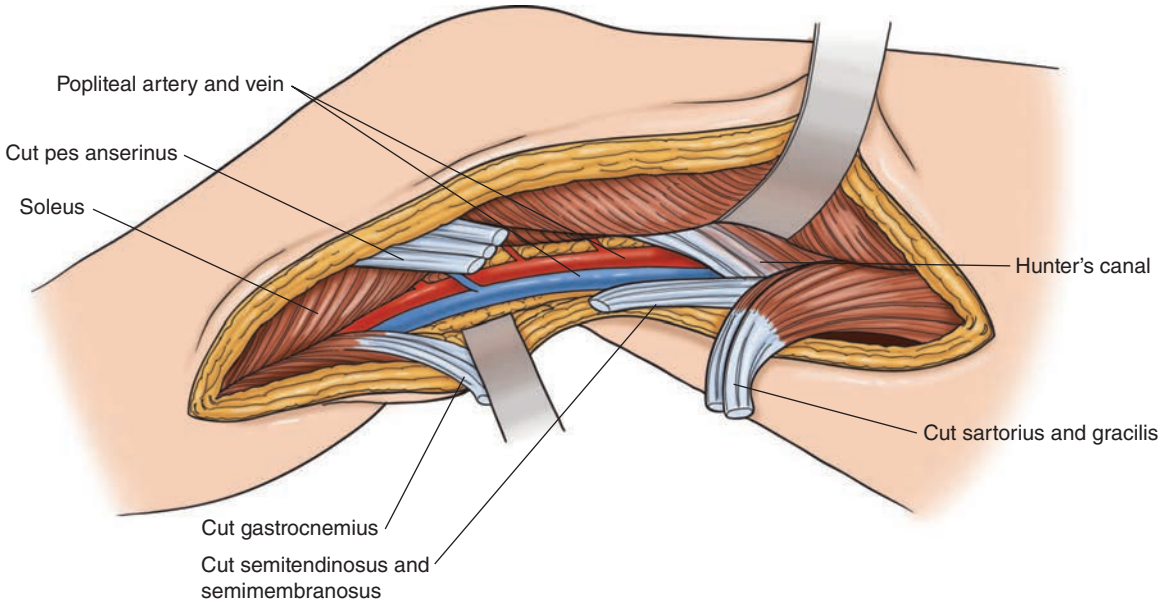


FIGURE 75 Exposure and Repair of Injuries to the Popliteal Artery and Vein
Opening the entire distal medial thigh and proximal medial leg from the area of Hunter's canal proximally to below the area where the popliteal artery divides exposes the popliteal artery and vein. If the injury is directly behind the knee joint, it may be necessary to divide and mark the tendons of the sartorius, gracilis, and semitendinosus muscles approximately 3/8 inch from the bone. After vascular repair, the tendons can be reapproximated with permanent sutures.

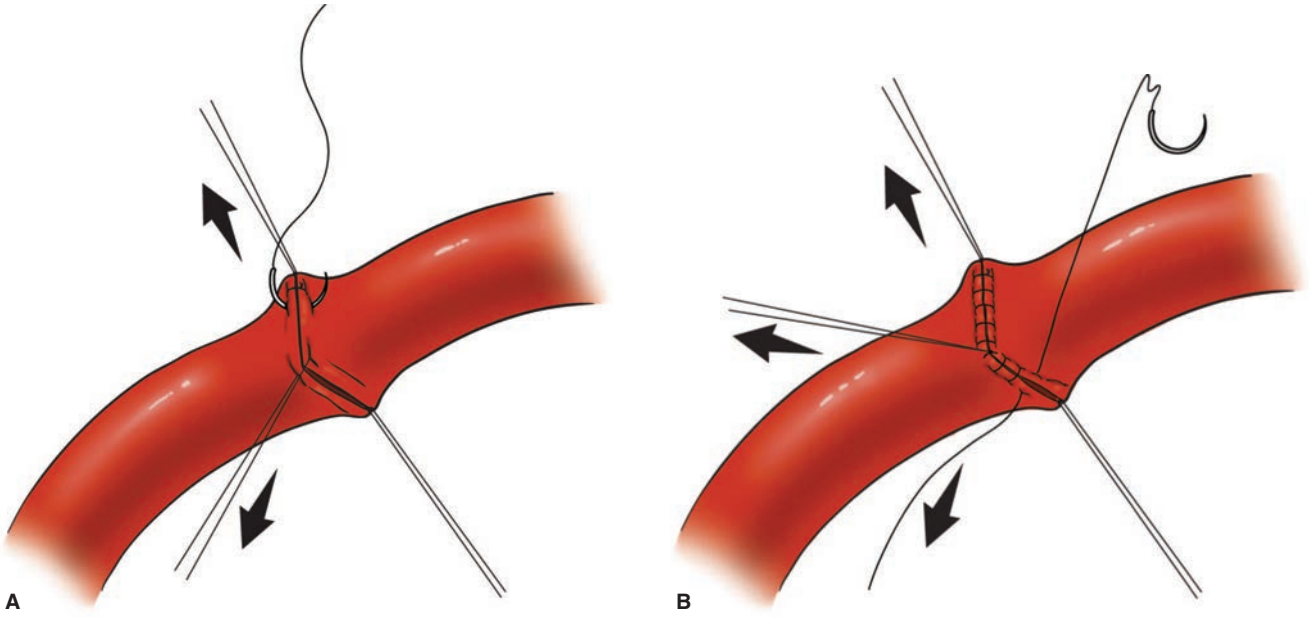


FIGURE 76 Triangulation Technique for End-to-End Anastomosis of Small Vessels

A. A triangulated end-to-end vascular anastomoses, a technique initially described by Alexis Carrel and Charles C. Guthrie.

B. Three temporary stay sutures may be tied as each section of the anastomosis is completed. Some surgeons prefer an interrupted anastomosis (not shown).

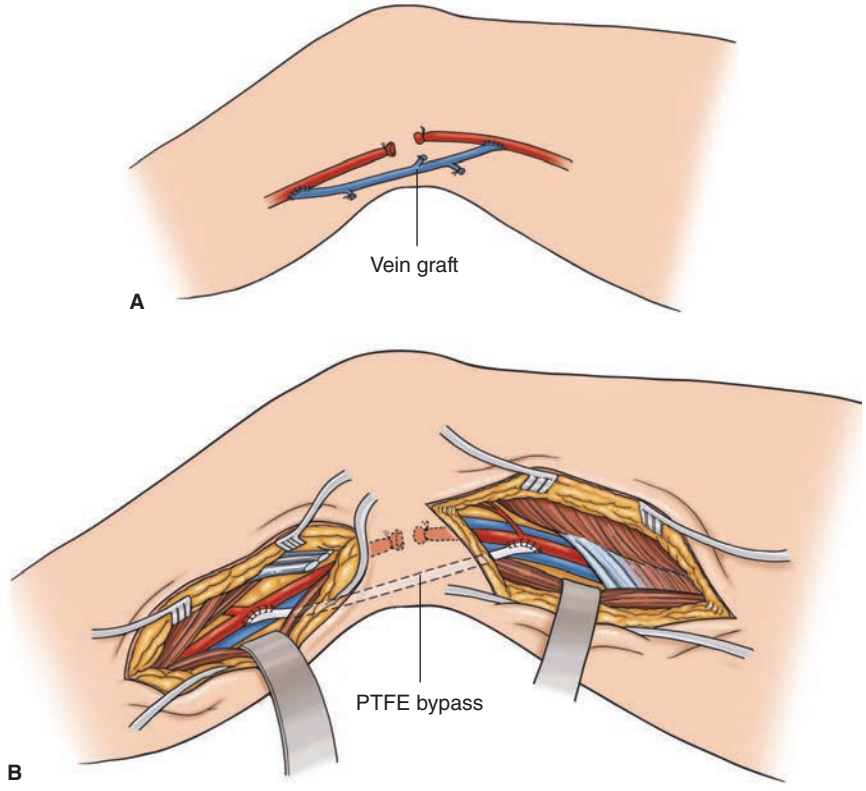


FIGURE 77 Popliteal Artery Injury Behind the Knee

A. Among the several options to reconstruct this injury, ligation of the two ends of the vessel with simple bypass using the saphenous vein from the other leg preserves the muscles and tendons at the medial side of the knee. Depending on presence of associated injuries, the bypass saphenous graft may be passed subcutaneously or deep in the normal traverse of the popliteal artery. Care must be taken to assure the graft does not kink when the knee is flexed.

B. On occasion, when vein is not available, an appropriately sized ringed polytetrafluoroethylene (PTFE) graft is used.

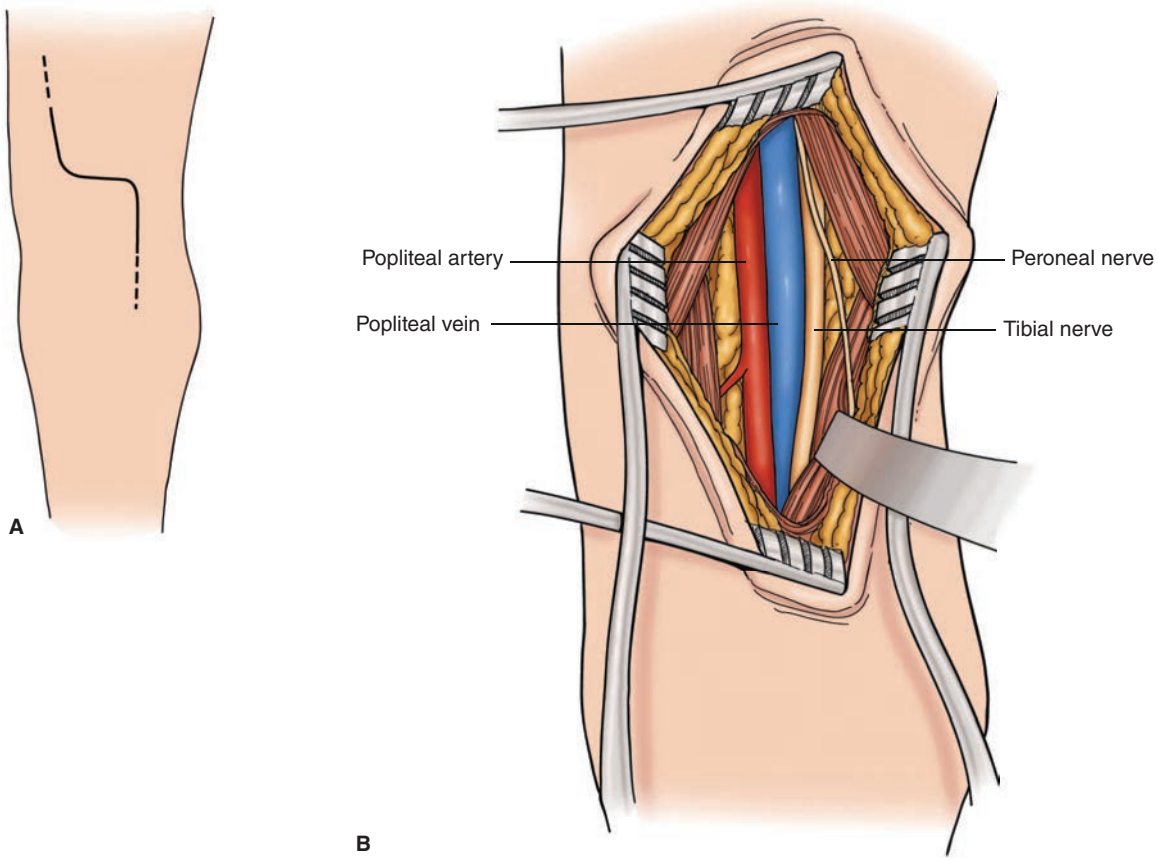


FIGURE 78 Posterior Exposure for Popliteal Artery Injuries

A. On rare occasions, a posterior approach to the popliteal fossa and its vessels may be desired. This approach prevents access to the groin and often reduces the opportunity for salvage of the greater saphenous vein.

B. Anatomy as seen for posterior approach to popliteal space.

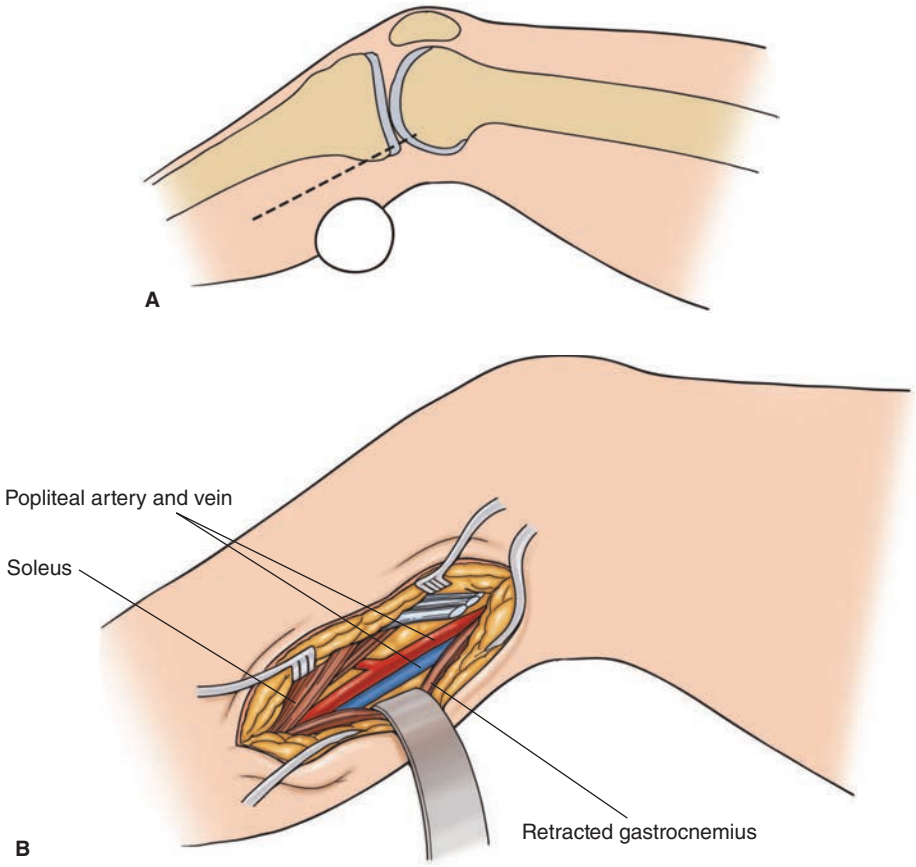


FIGURE 79 Exposure and Repair of Distal Popliteal and "Trifurcation" Injuries

A. Care is taken that the upper portion of this medial incision does not injure the subcutaneous saphenous vein and that it is sufficiently long enough for exposure of the proximal shank arteries.

B. The medial head of the gastrocnemius is taken down and the soleus muscle is exposed. The popliteal artery first gives off the anterior tibial artery, which traverses anteriorly through the interosseous membrane. Once precise vascular control is obtained in the area of the injury, reconstruction can be accomplished.

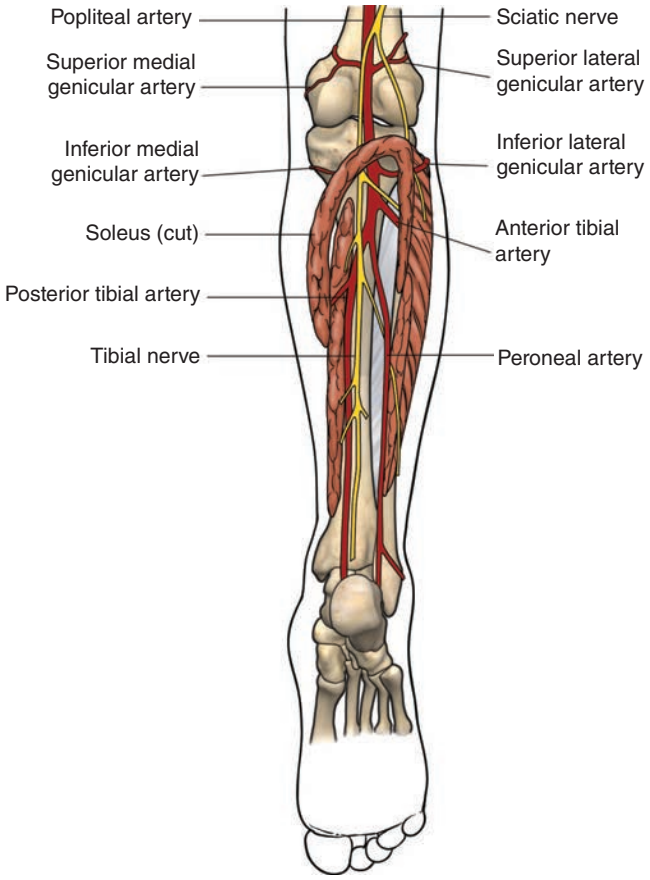


FIGURE 80 Anatomy of the Posterior Leg
Note the course of the popliteal artery and its association with the branches of the sciatic nerve.

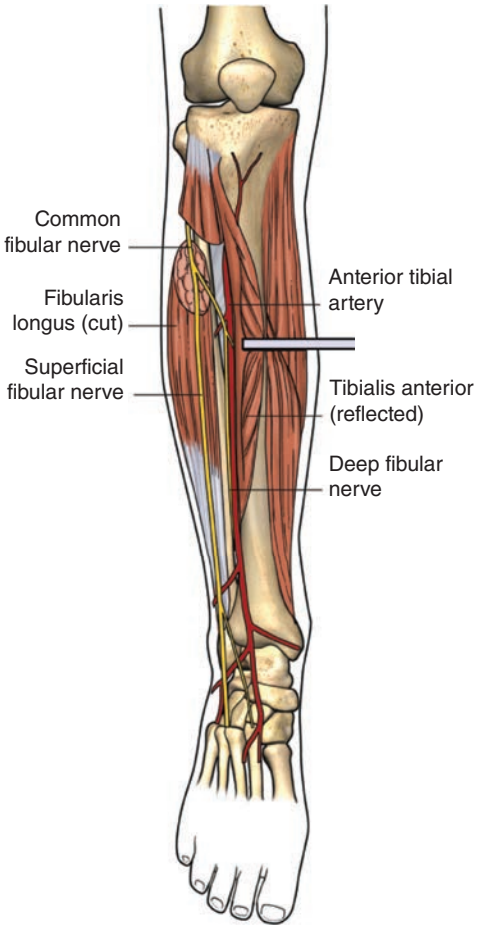
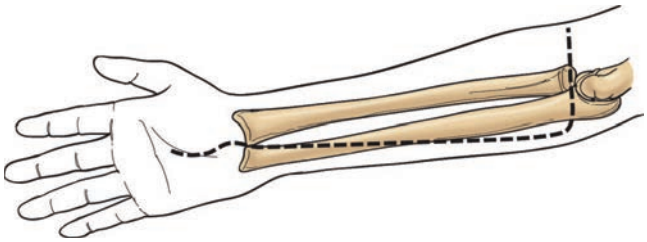
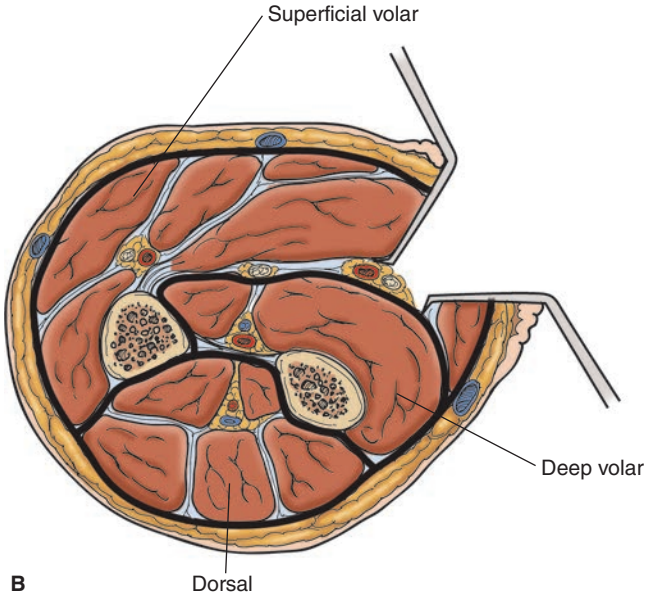


FIGURE 81 Anatomy of the Anterior Leg
The nerves of concern are located laterally, and care is taken during a lateral fasciotomy to prevent iatrogenic injury. Note that the anterior tibial artery has traversed through the interosseous membrane to the anterior compartment.



A



B

FIGURE 82 Forearm Volar-Ulnar Fasciotomy to Decompress Mobile Wad and Superficial and Deep Volar Compartments

A. A single incision beginning on the radial side of the antecubital area extends transversely to the ulnar side. The incision then curves toward the hand and goes down the ulnar side of the volar forearm aiming for the mid-wrist.

B. The transverse antecubital fascial incision decompresses the mobile wad, while the volar-ulnar fascial incision decompresses the superficial volar compartment. Through this latter incision, the flexor carpi ulnaris and flexor digitorum sublimis muscles are separated, the ulnar artery is avoided, and the muscles in the deep volar compartment are individually decompressed.

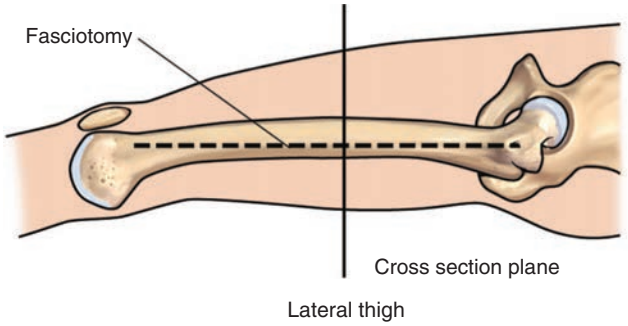


FIGURE 83 Thigh Fasciotomy to Decompress Anterior (Extensor) and Posterior (Flexor) Compartments

One anterolateral thigh skin incision along the iliotibial tract and opening the fascia over the vastus lateralis muscle and mobilizing it superiorly allows for decompression of the extensor and flexor compartments of the thigh.

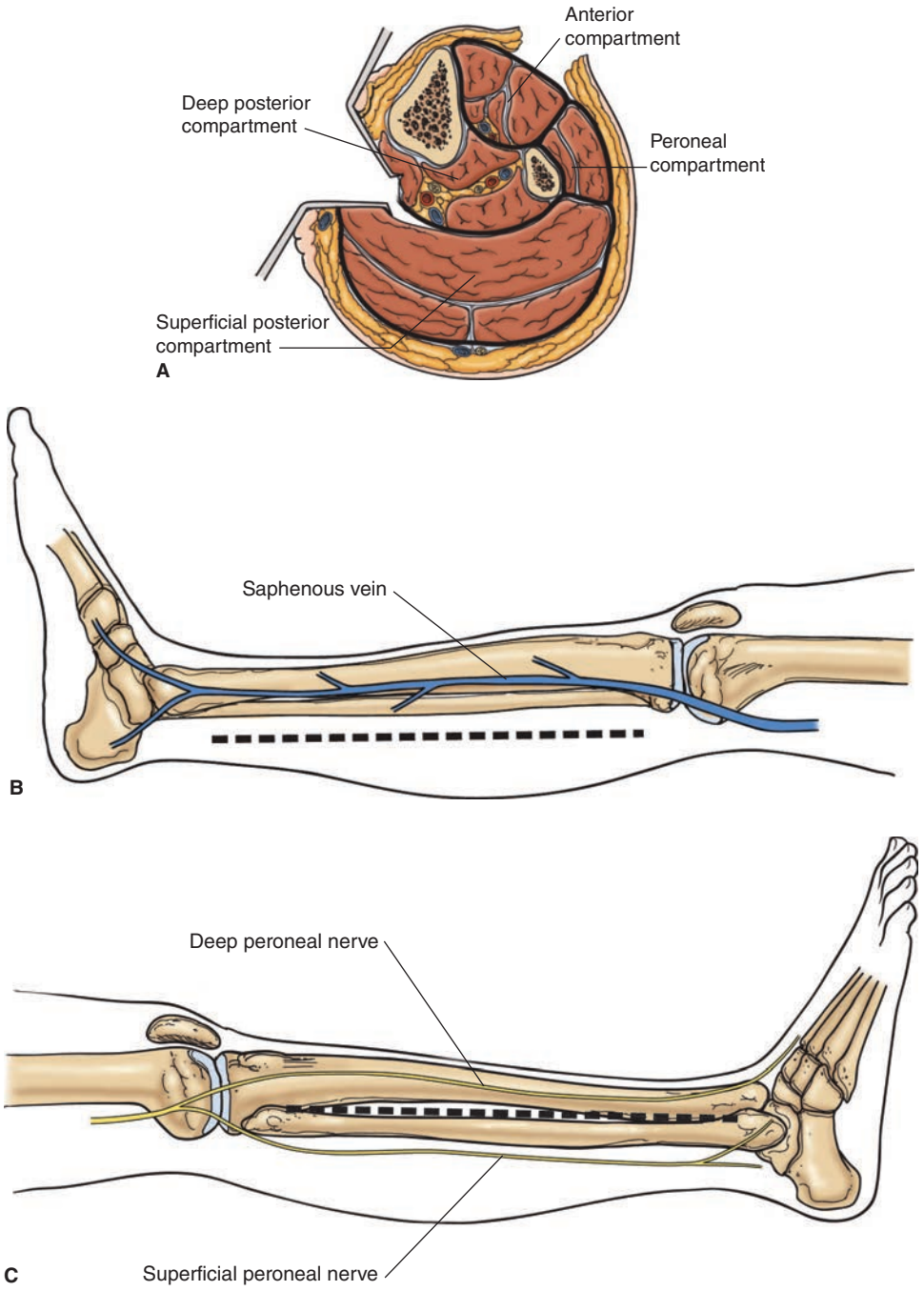


FIGURE 84 Calf Fasciotomy to Decompress Anterior, Peroneal, and Superficial and Deep Posterior Compartments

A. Cross section of the four compartments of the calf.

B. The lateral skin incision is made 2 cm anterior to the superior edge of the fibula curving anteriorly as the incision goes toward the knee to avoid injury to the superficial peroneal nerve. The anterior and peroneal compartments are opened longitudinally (about 4–5 cm apart) through this one lateral skin incision.

C. The medial skin incision is made 2 cm posterior to the inferior edge of the tibia avoiding injury to the greater saphenous vein. Once the superficial posterior compartment is decompressed, the soleus muscle is detached from the lateral tibia to allow for decompression of the deep posterior compartment immediately under the shaft of the tibia.

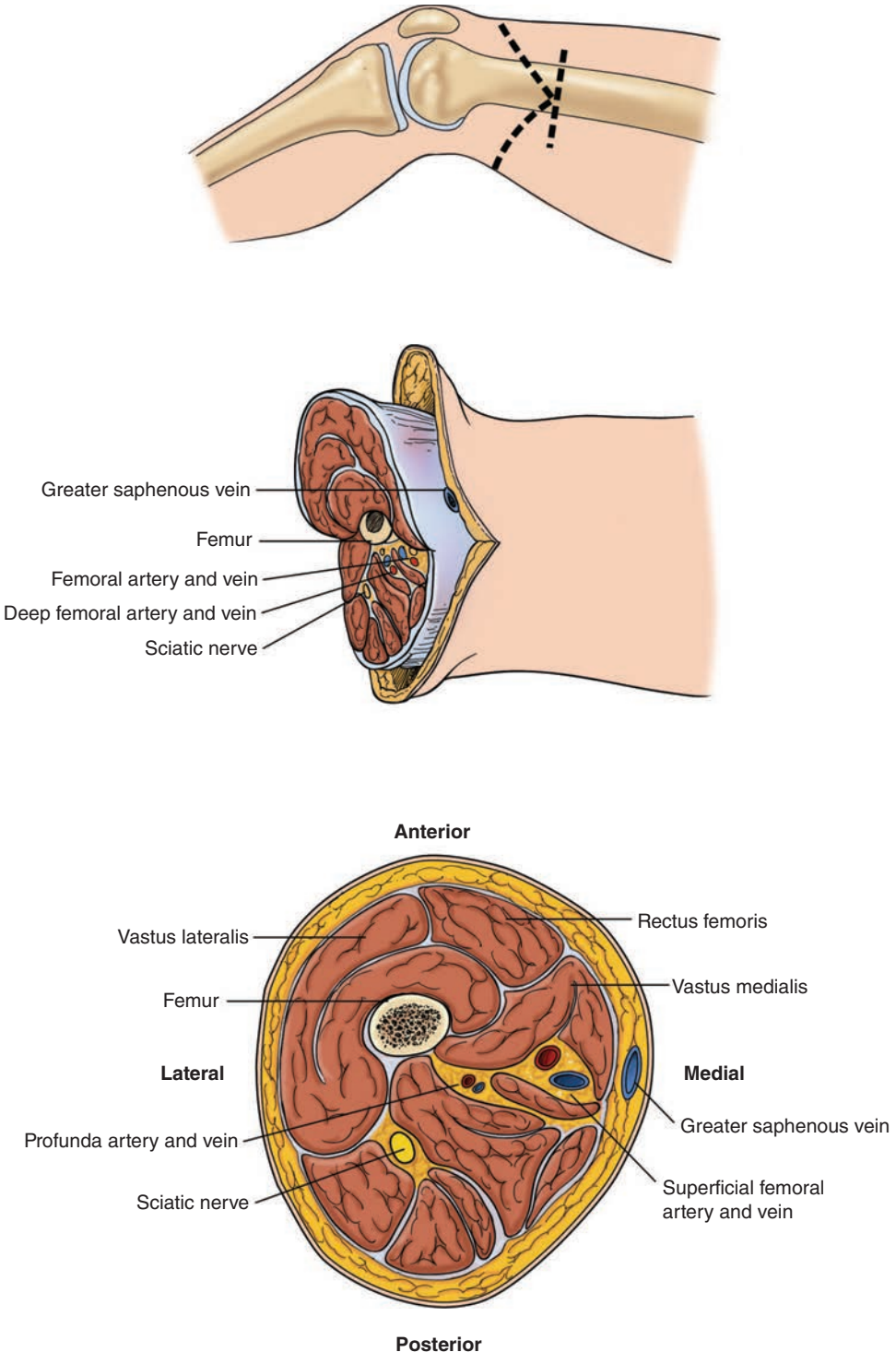


FIGURE 85 Above the Knee Amputation
These drawings illustrate an above the knee amputation. Note that the femur is amputated higher than the skin flaps to achieve tension-free coverage. The regional anatomy is shown.

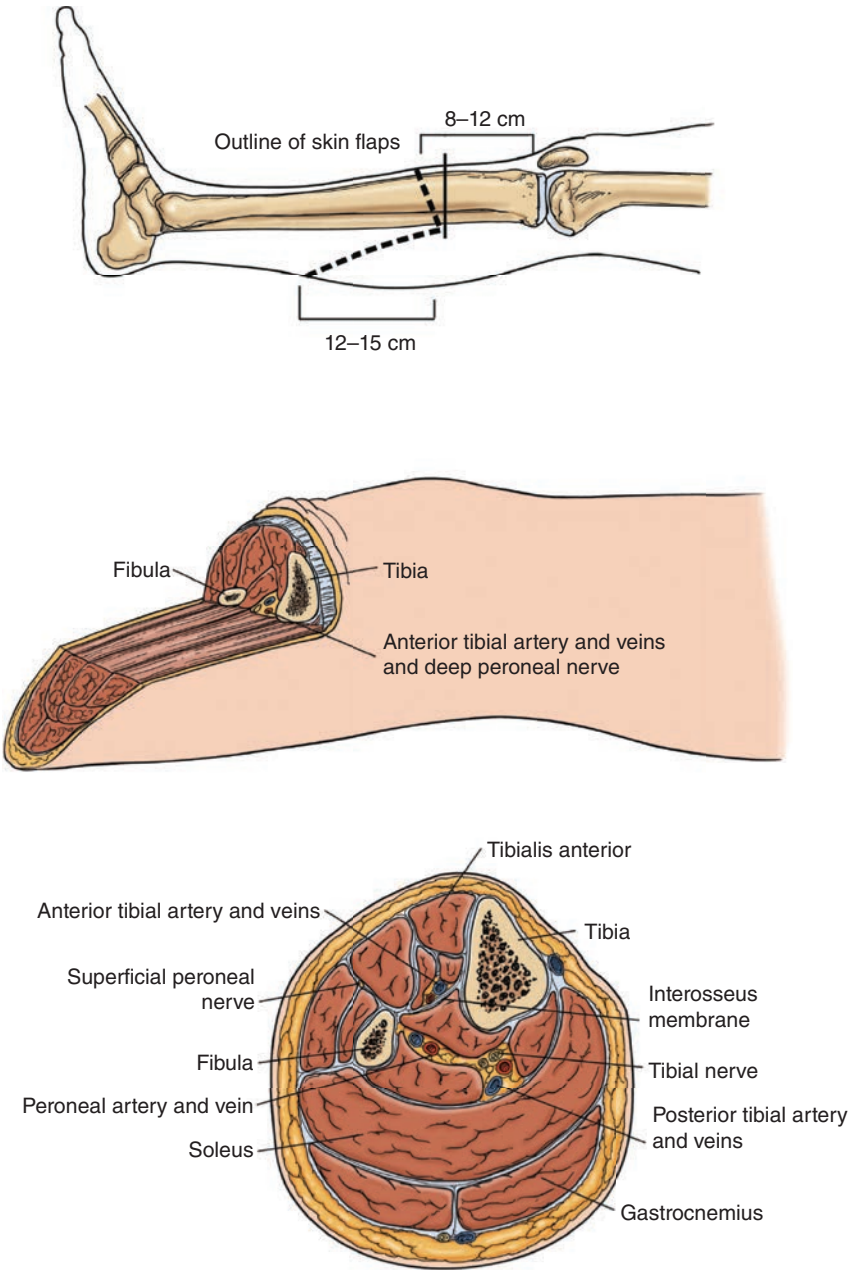


FIGURE 86 Below the Knee Amputation
These drawings illustrate a below the knee amputation. Note the long posterior flap to allow coverage of the amputation stump and provide an adequate cushion for the weight-bearing surface.

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Note: Page numbers followed by *b, f,* and *t* indicate boxes, figures, and tables, respectively.