



Pathological anatomy

**Disorders of
Circulation**

Circulatory disorders:

- Arterial hyperemia, Venous congestion (BV – Narrowing)
- Bleeding, hemorrhage (BV - rupture, aneurism).
- Thrombosis
- Embolism
- Edema
- Shock

Arterial hyperemia

1. Angioneurotic hyperemia,
2. Inflammatory hyperemia,
3. Collateral hyperemia,
4. Post ischemic hyperemia,
5. Vacate hyperemia (after low barometric pressure),
6. Hyperemia due to arterio-venous shunt.
7. Hyperemia after influence of physical or chemical factor.



Angioneurotic hyperemia

Herpes virus neuralgia;

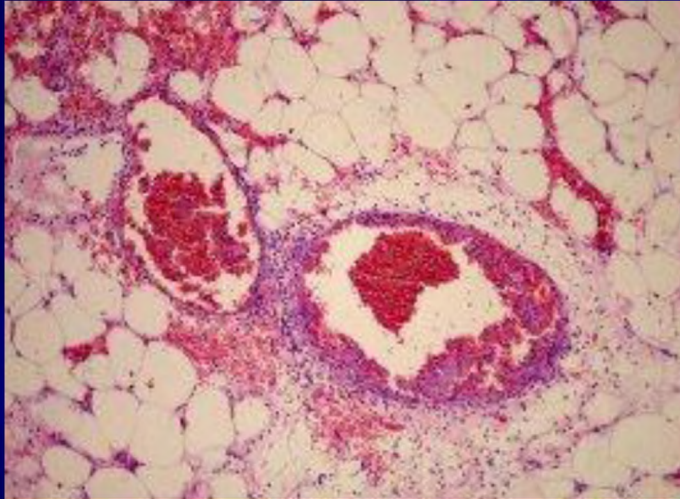


Primary neurovascular
disorder;

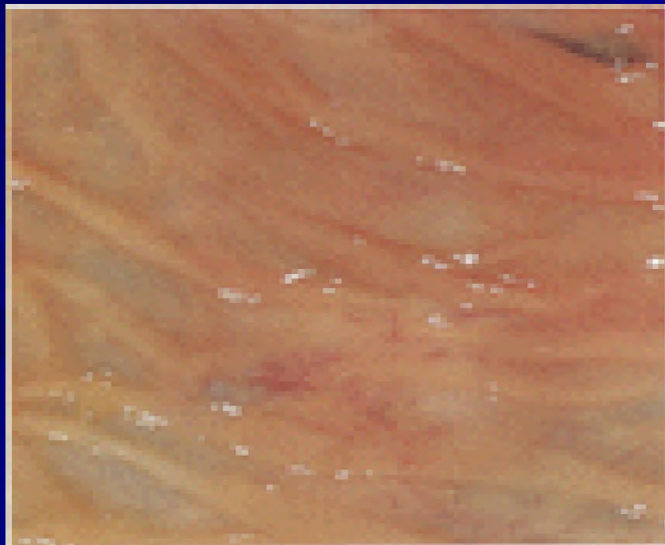


Systemic Lupus
Erythematosus.

Inflammatory hyperemia

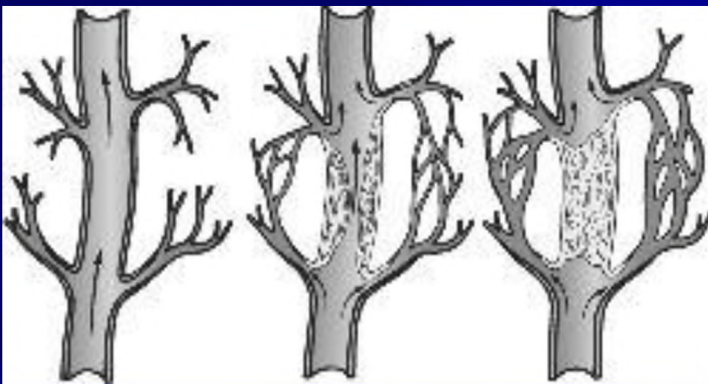
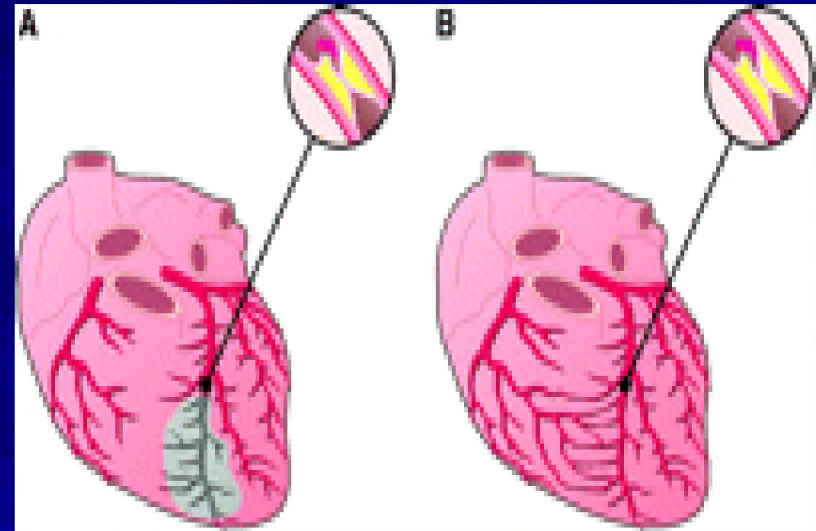


- Compensatory regulation of blood circulation during inflammation.
- Compensation of microcirculatory changes, number of functional capillaries increases.

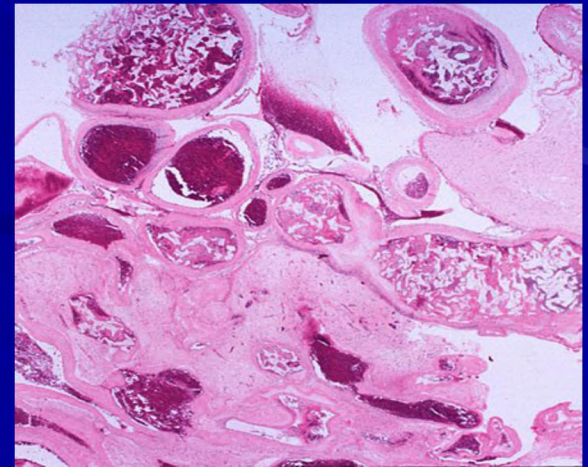
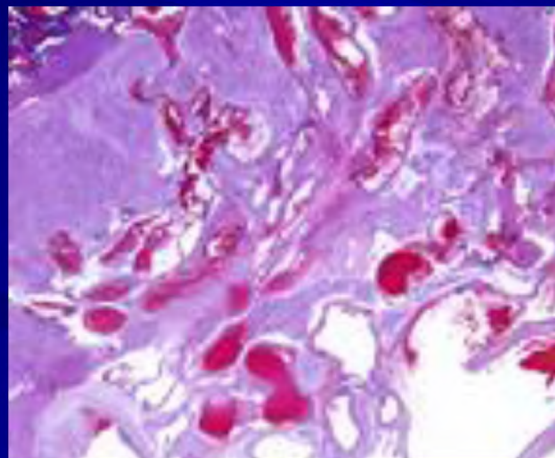
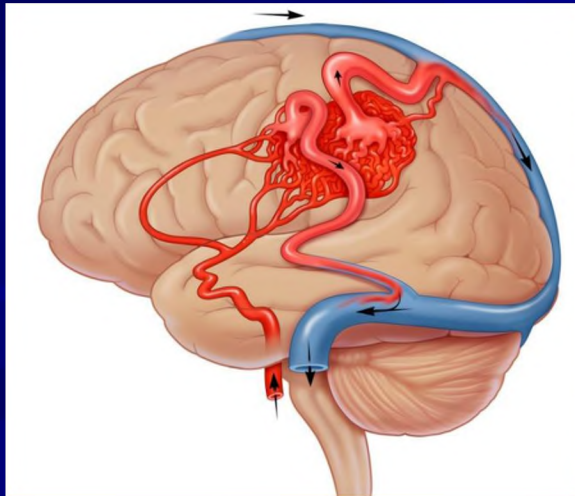
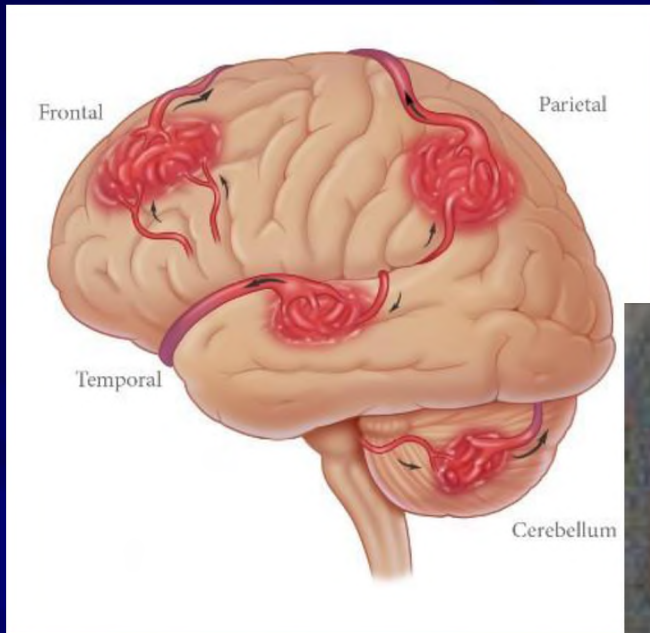


Catarrhal erosive gastritis.

Collateral hyperemia



Collateral hyperemia due to arterio-venous malformation

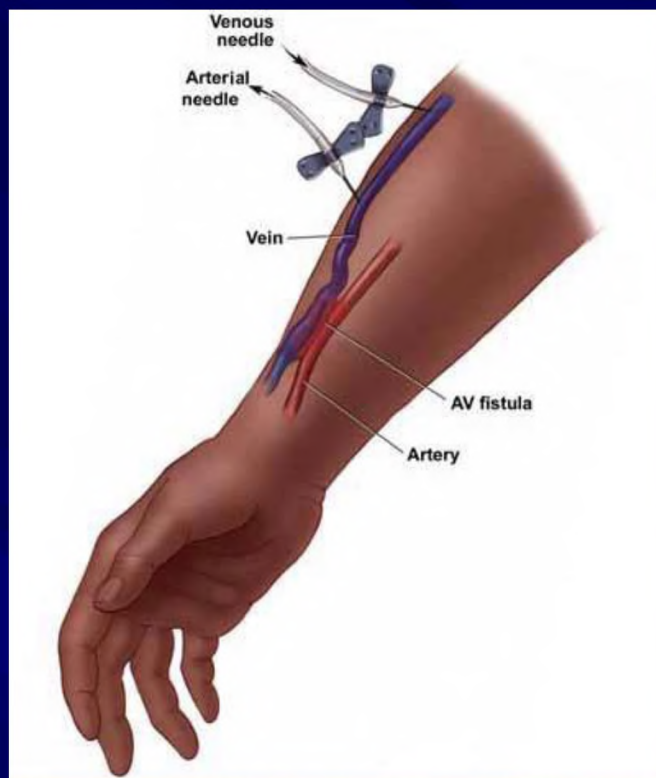


Vacate hyperemia

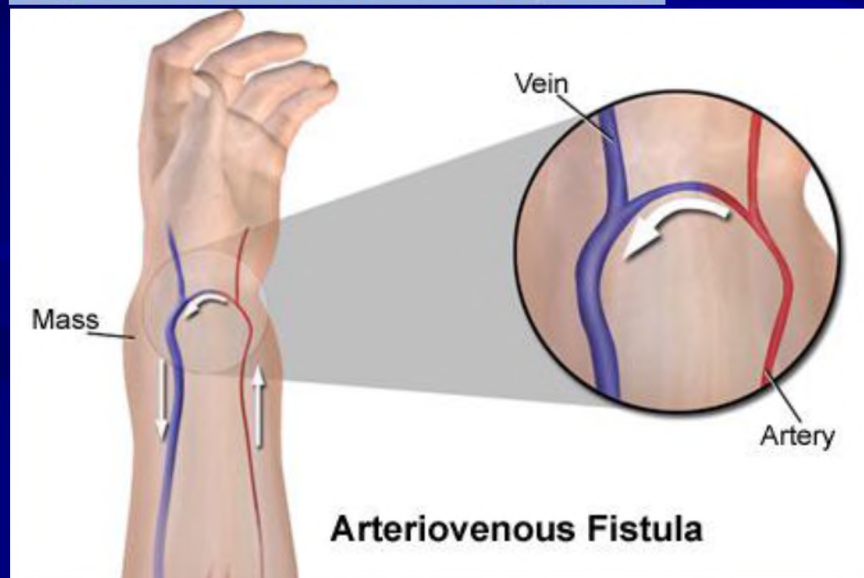


Developed after low
barometric pressure on the
skin.

Hyperemia due to arterio-venous shunt



© Mayo Foundation for Medical Education and Research. All rights reserved.

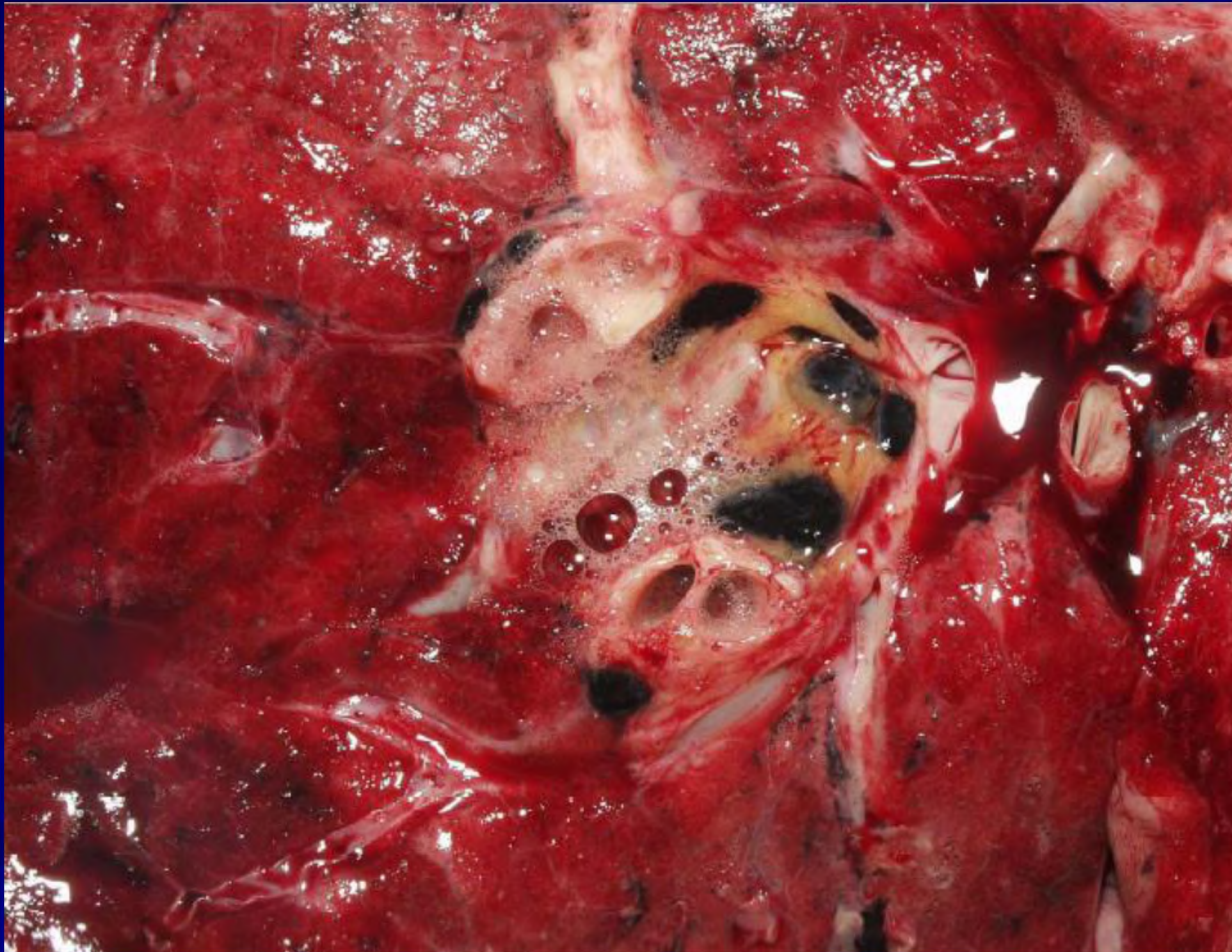


Passive hyperemia, or congestion

Passive hyperemia, or congestion, is engorgement of an organ with venous blood. Acute passive congestion is clinically a consequence of acute left or right ventricular failure.

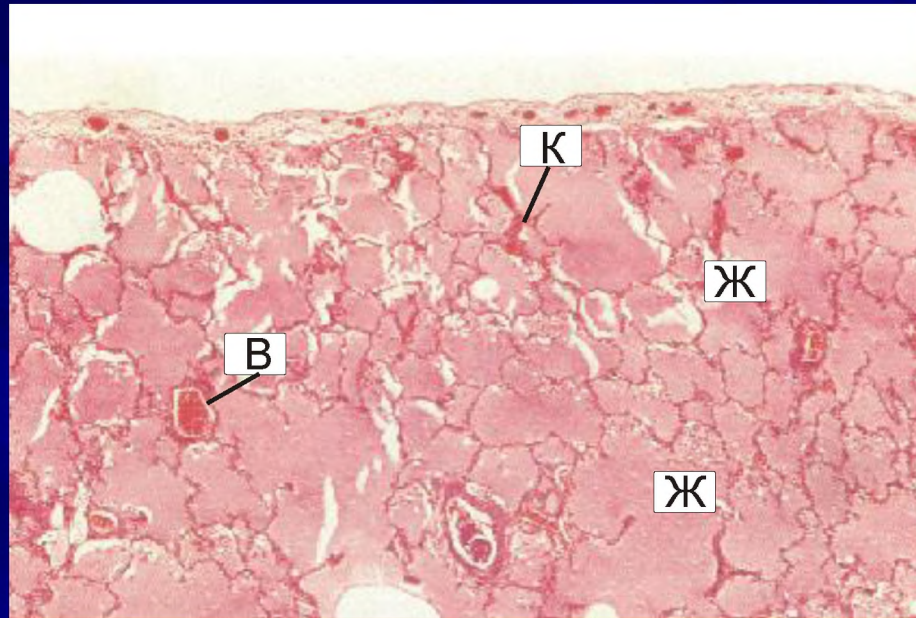
Regarding the former, resultant pulmonary venous engorgement leads to pulmonary edema, or accumulation of a transudate within the alveolar space.

Edema of lung



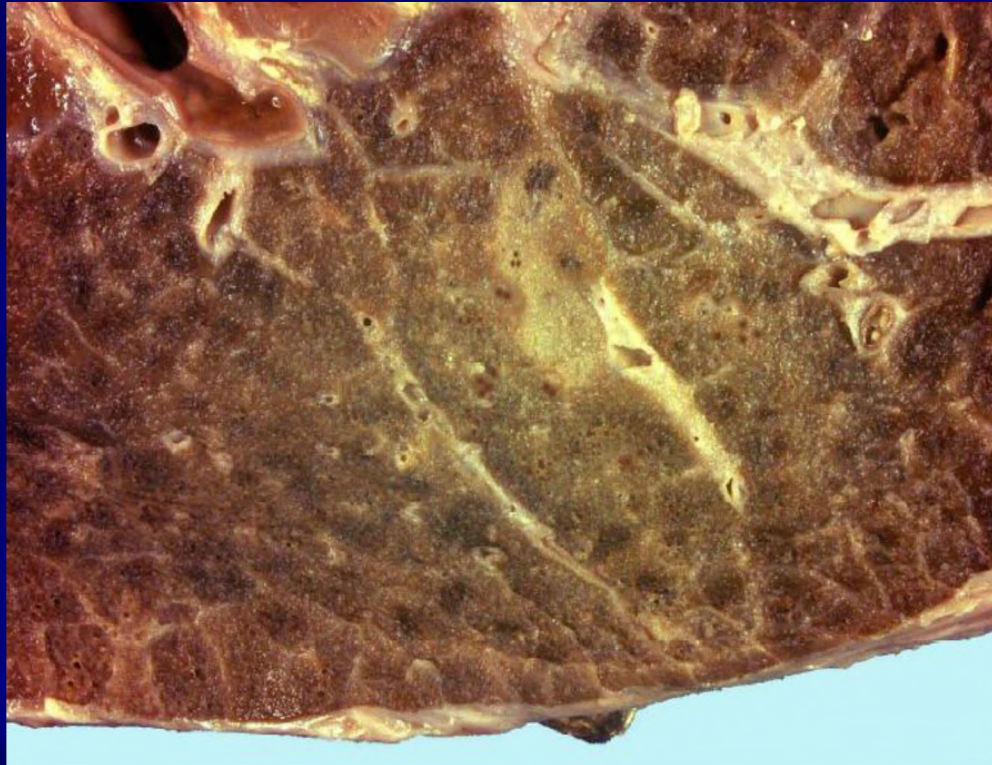
Pulmonary Edema

(hyperaemia universalis venosa)

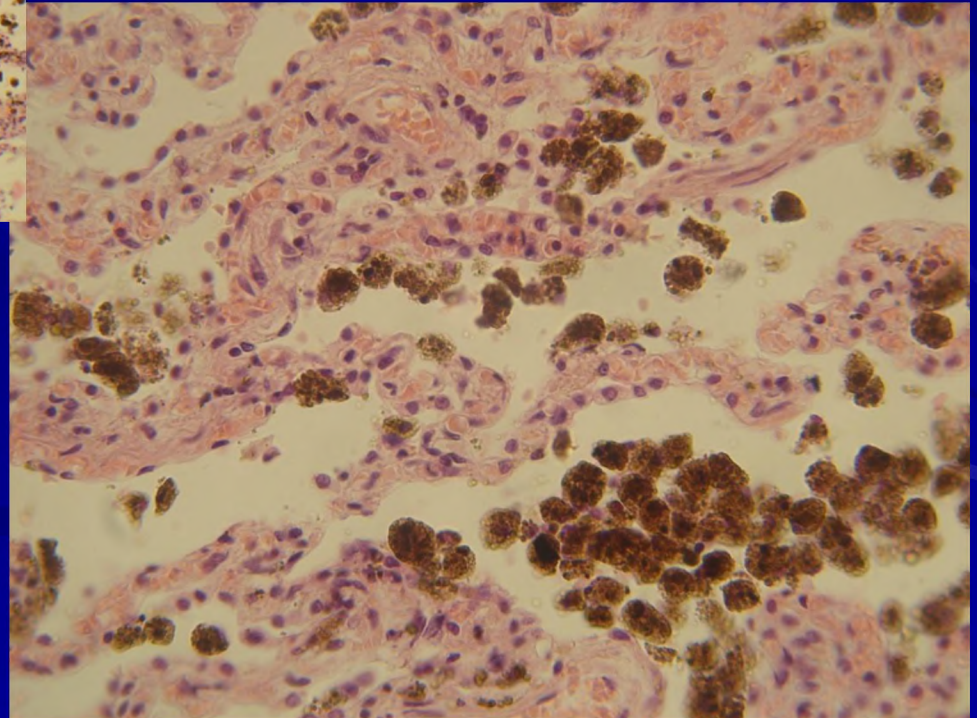
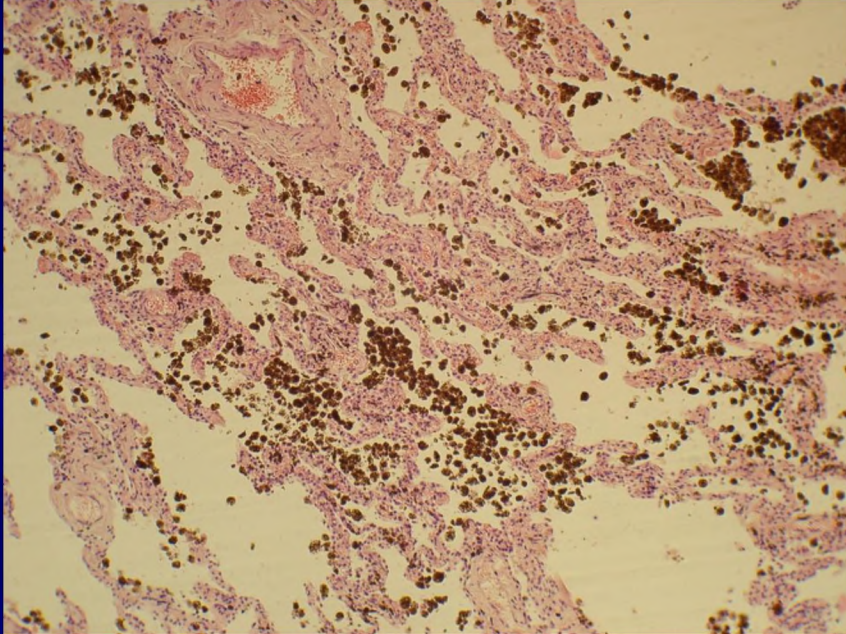


In alveoli accumulated fluid (Ж), characterized by weak acidophilia. Fluid accumulates in the alveoli in pulmonary edema is called transudate, which is transparent and contains less than 2% protein. The capillaries (К) of interalveolar septae and venules (В) dramatically expanded.

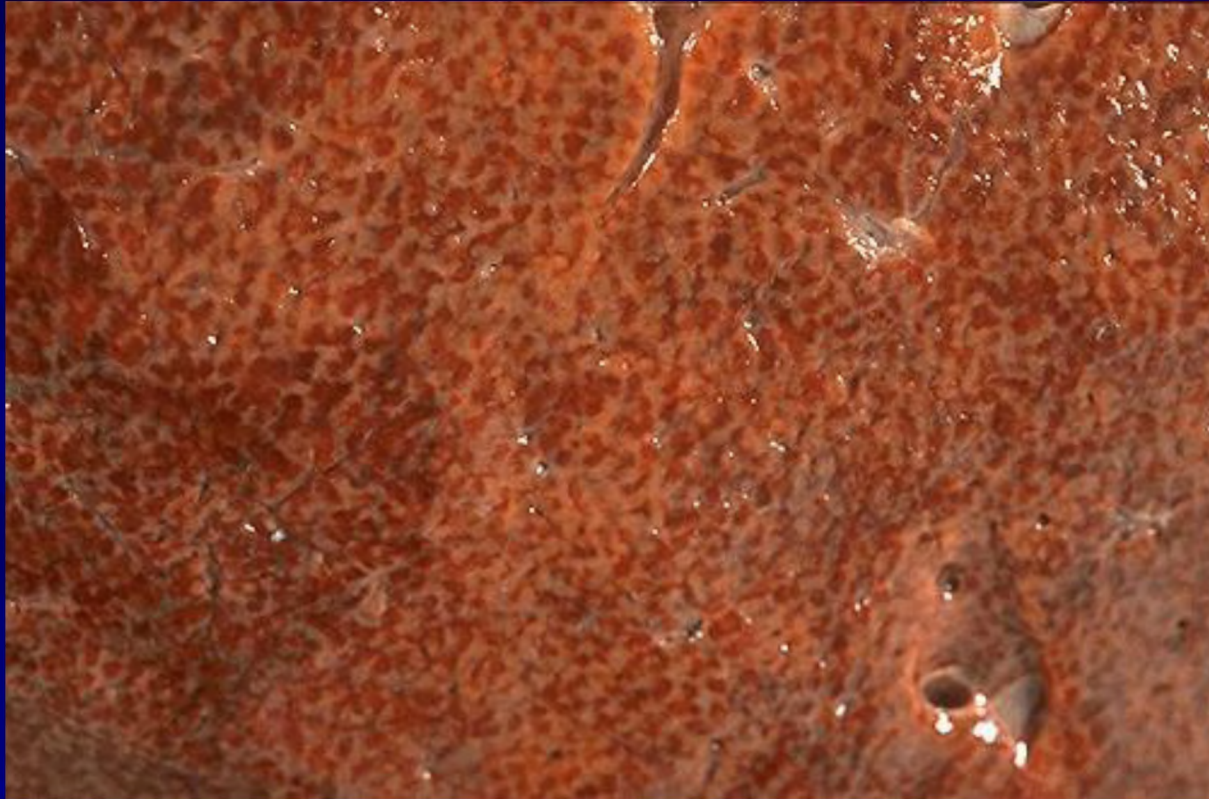
Venous congestion



Venous congestion

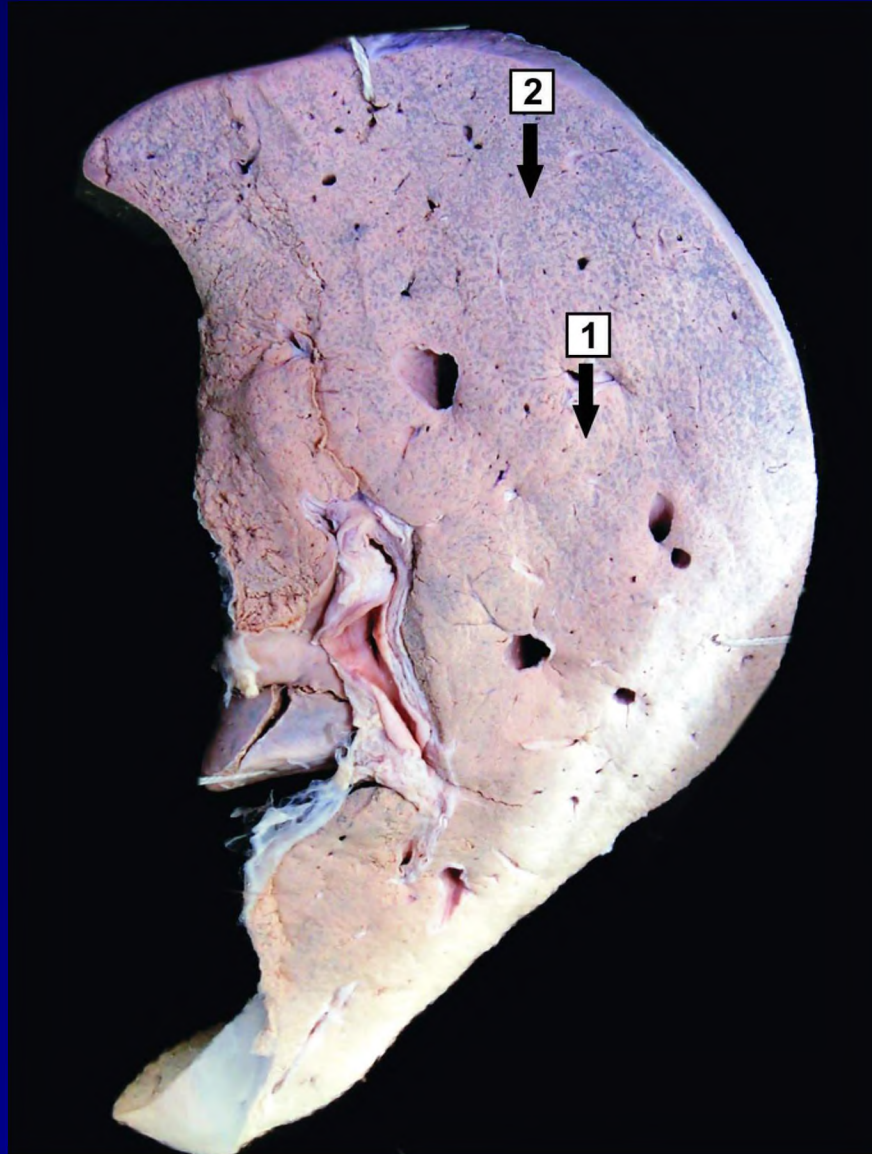


Venous congestion

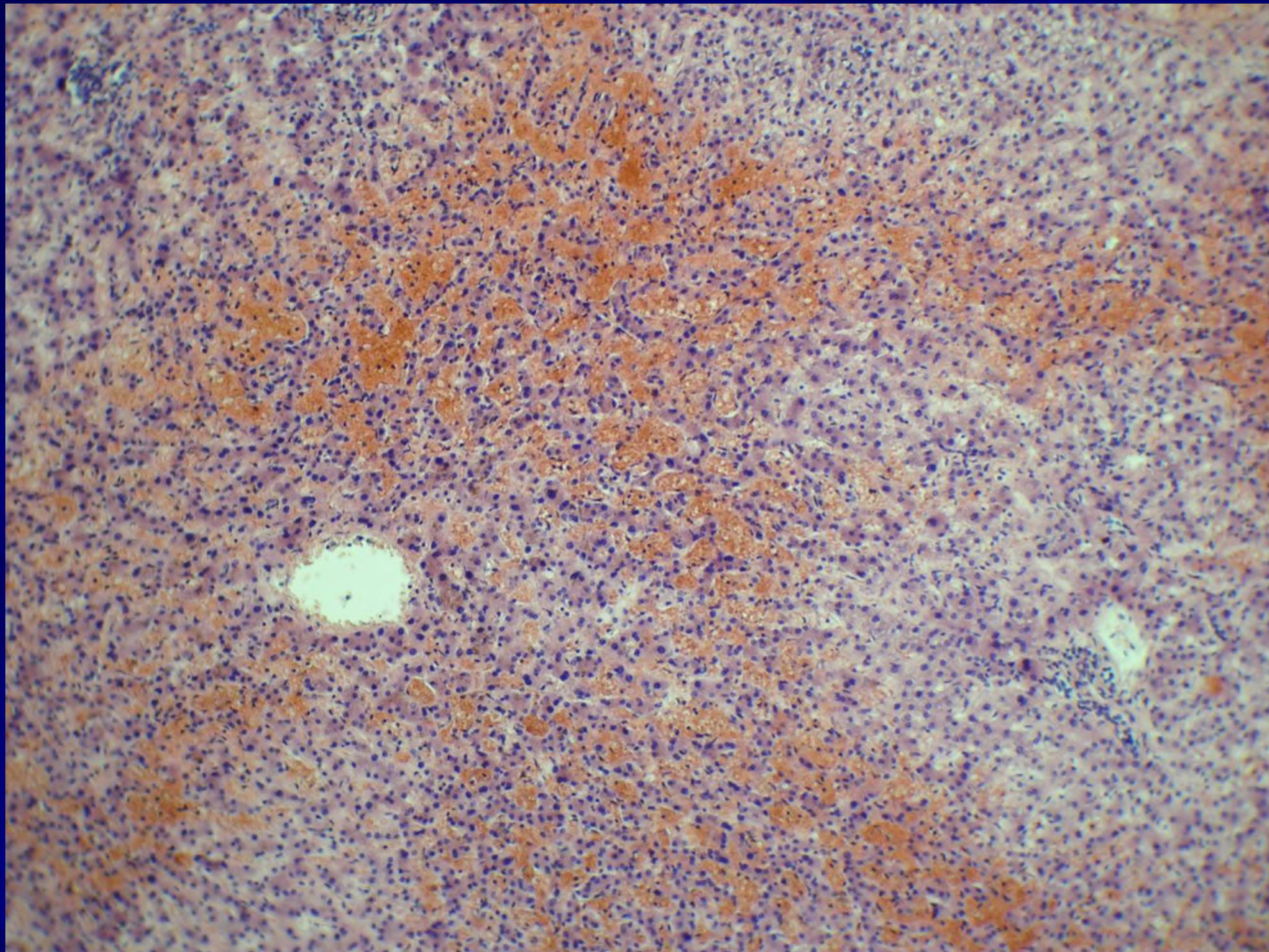


- Here is an example of a "nutmeg" liver seen with chronic passive congestion of the liver. Note the dark red congested regions that represent accumulation of RBC's in centrilobular regions.

“Nutmeg” liver seen with chronic passive congestion of the liver

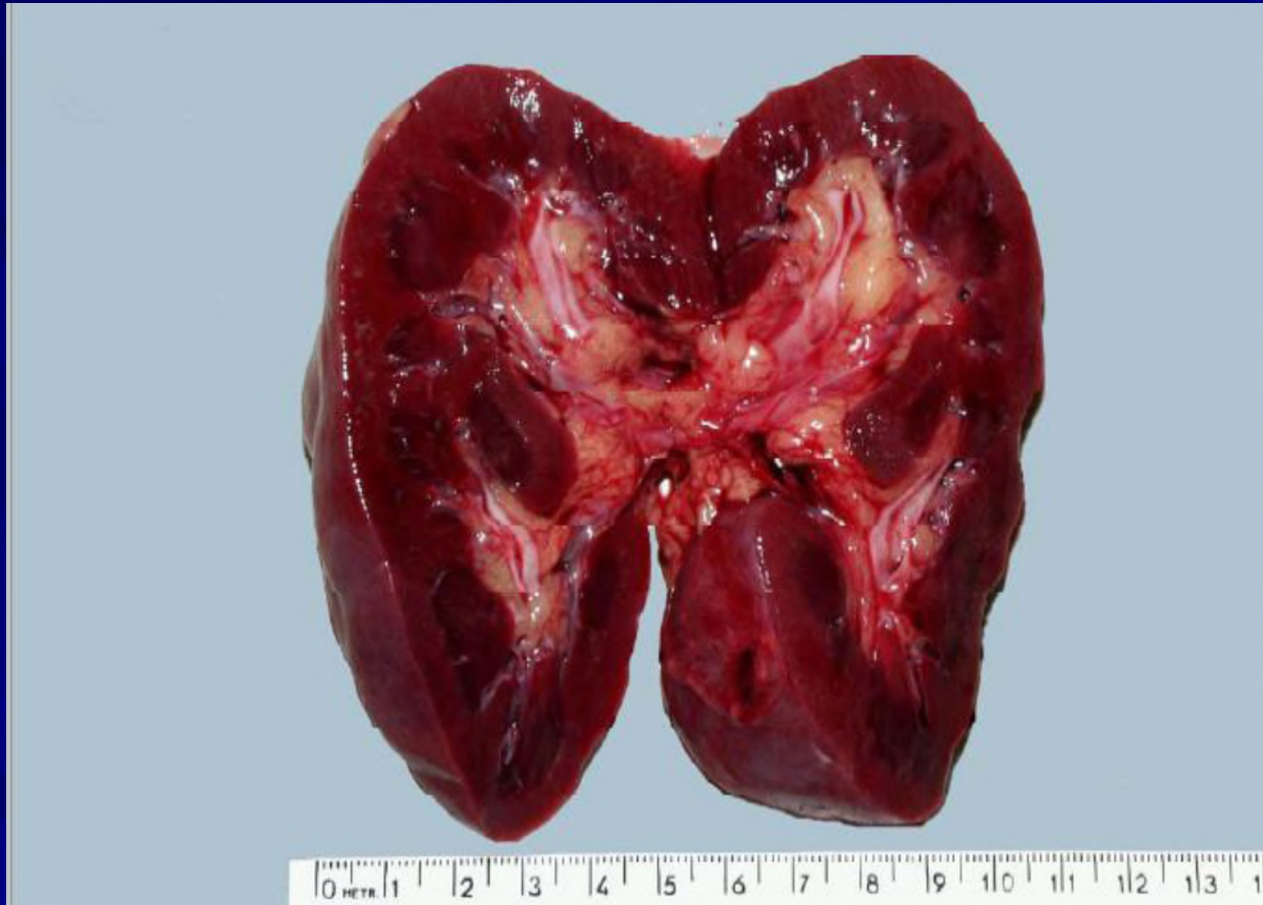


Venous congestion

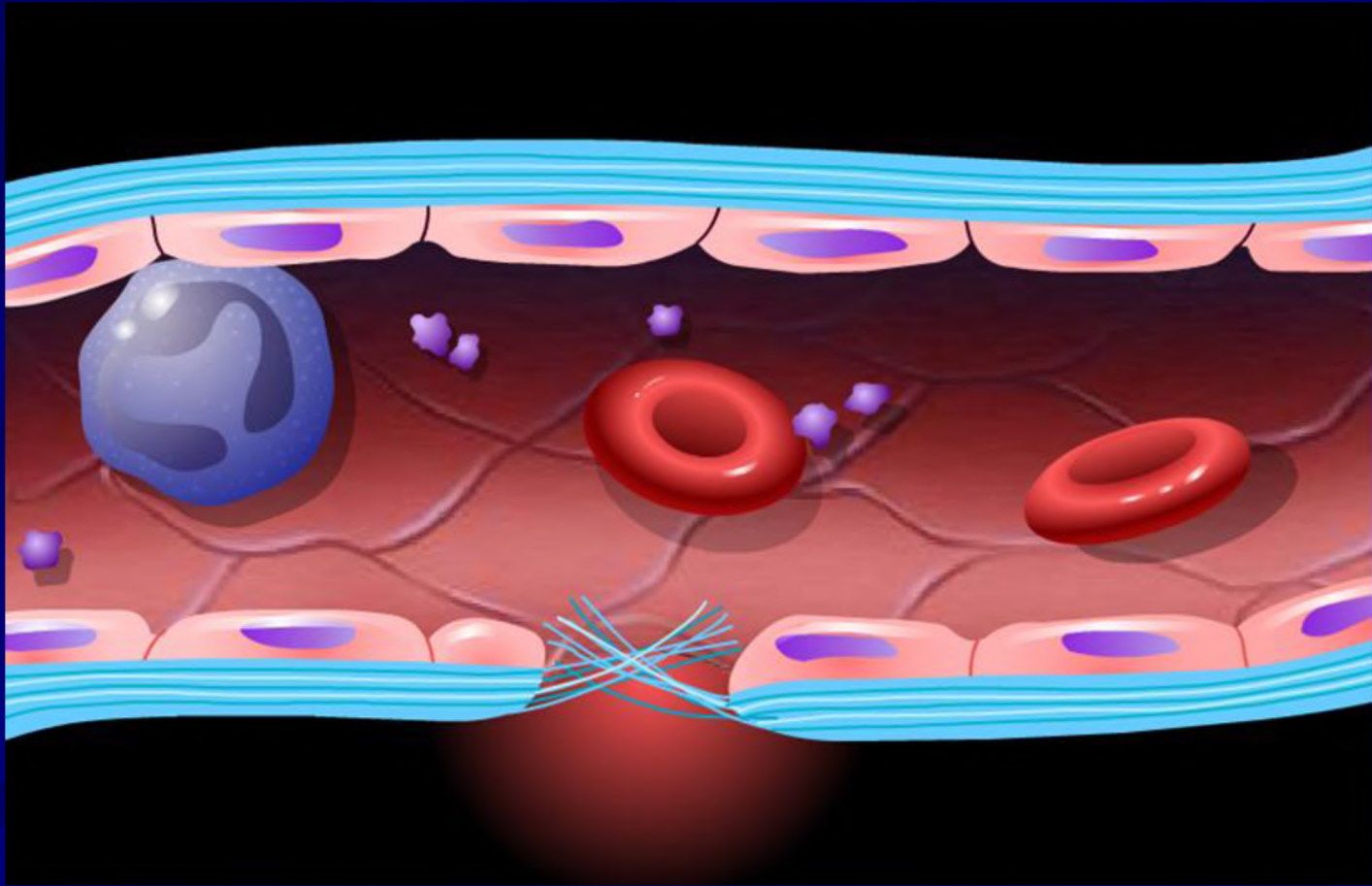


Increased venous pressure causes the central veins of hepatic lobules to dilate and is transmitted to hepatic sinusoids, which dilate, causing centrilobular hepatocytes to undergo pressure atrophy.

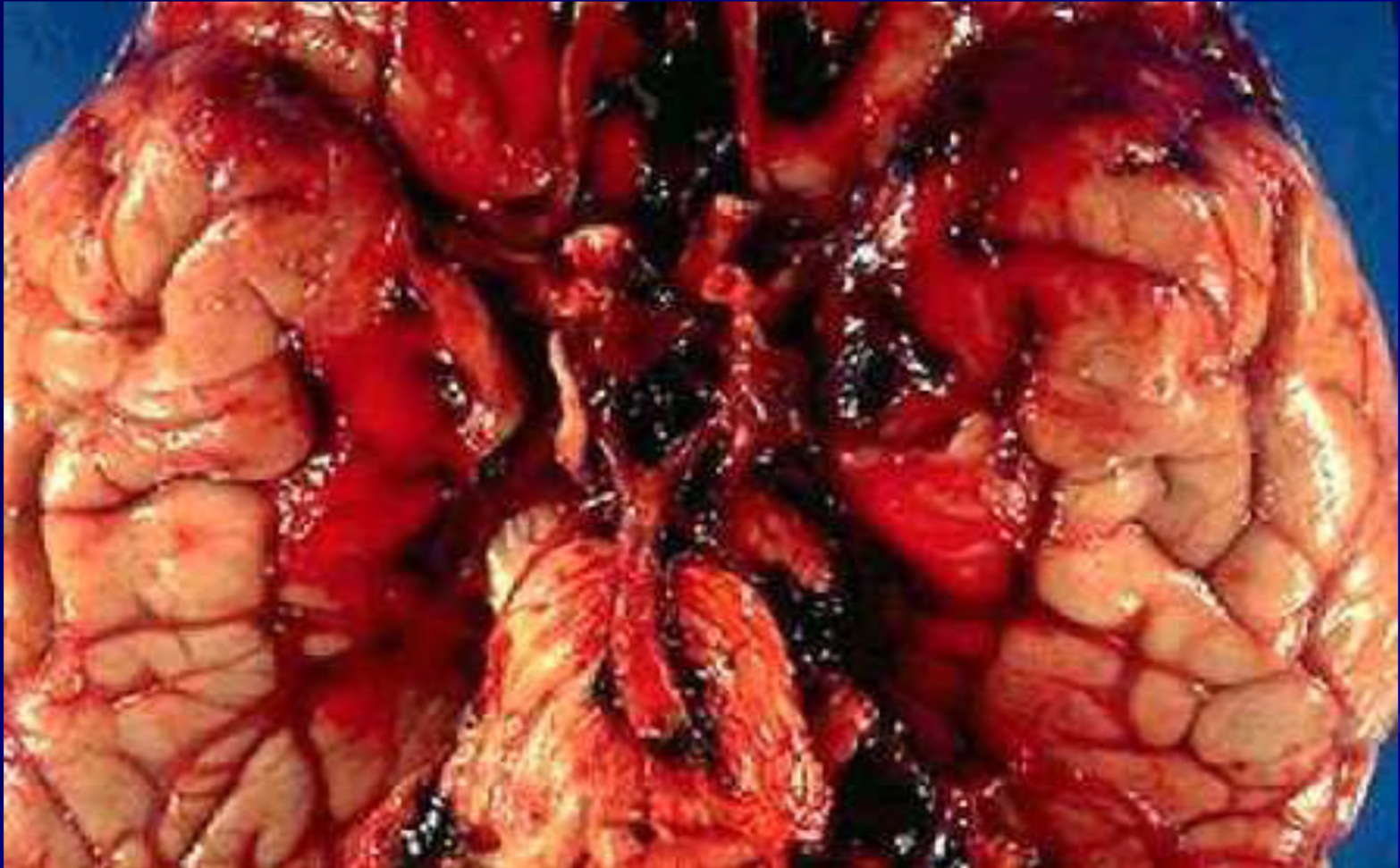
Venous congestion



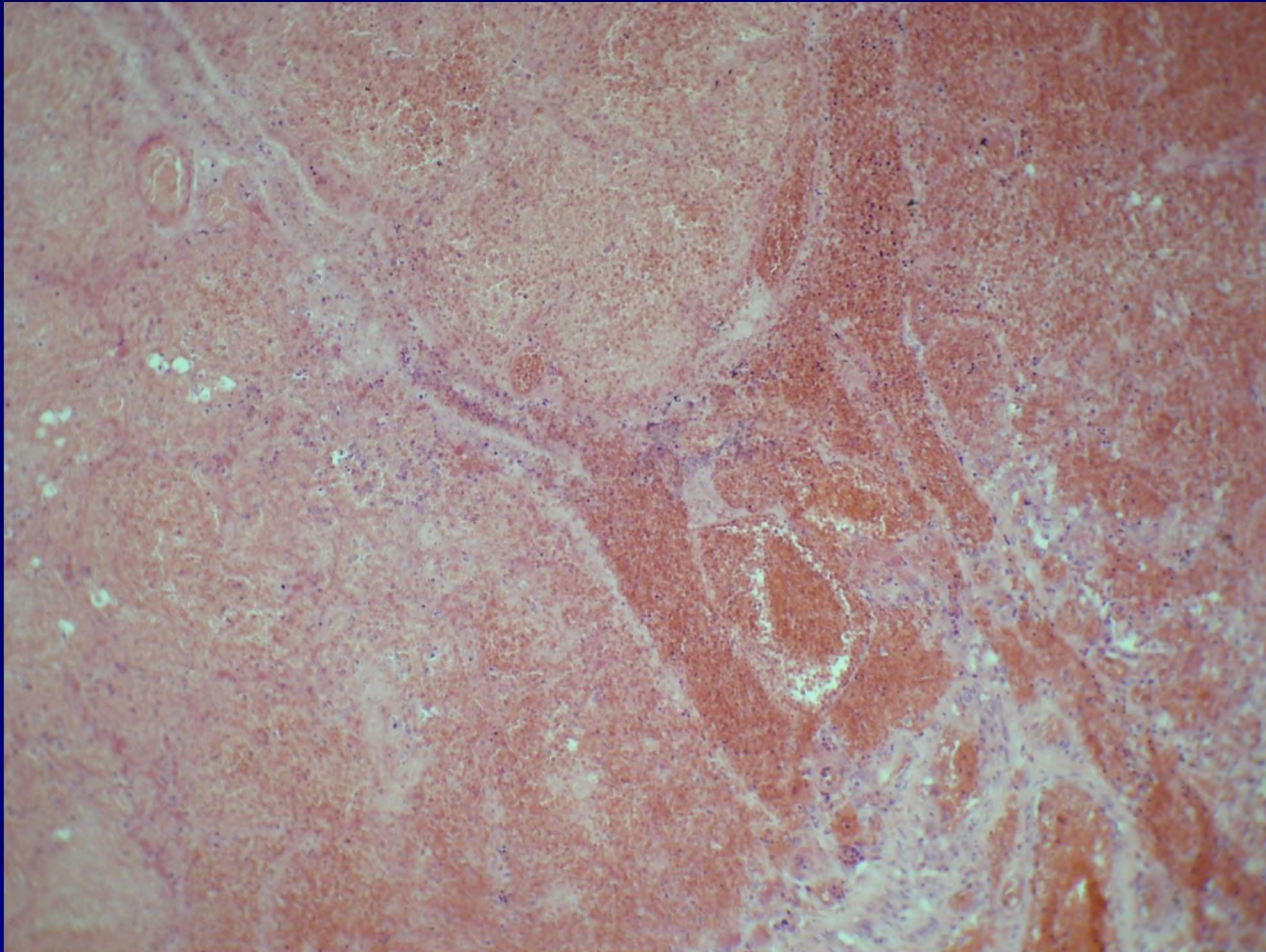
Bleeding Disorders



Subarachnoid Hemorrhage:

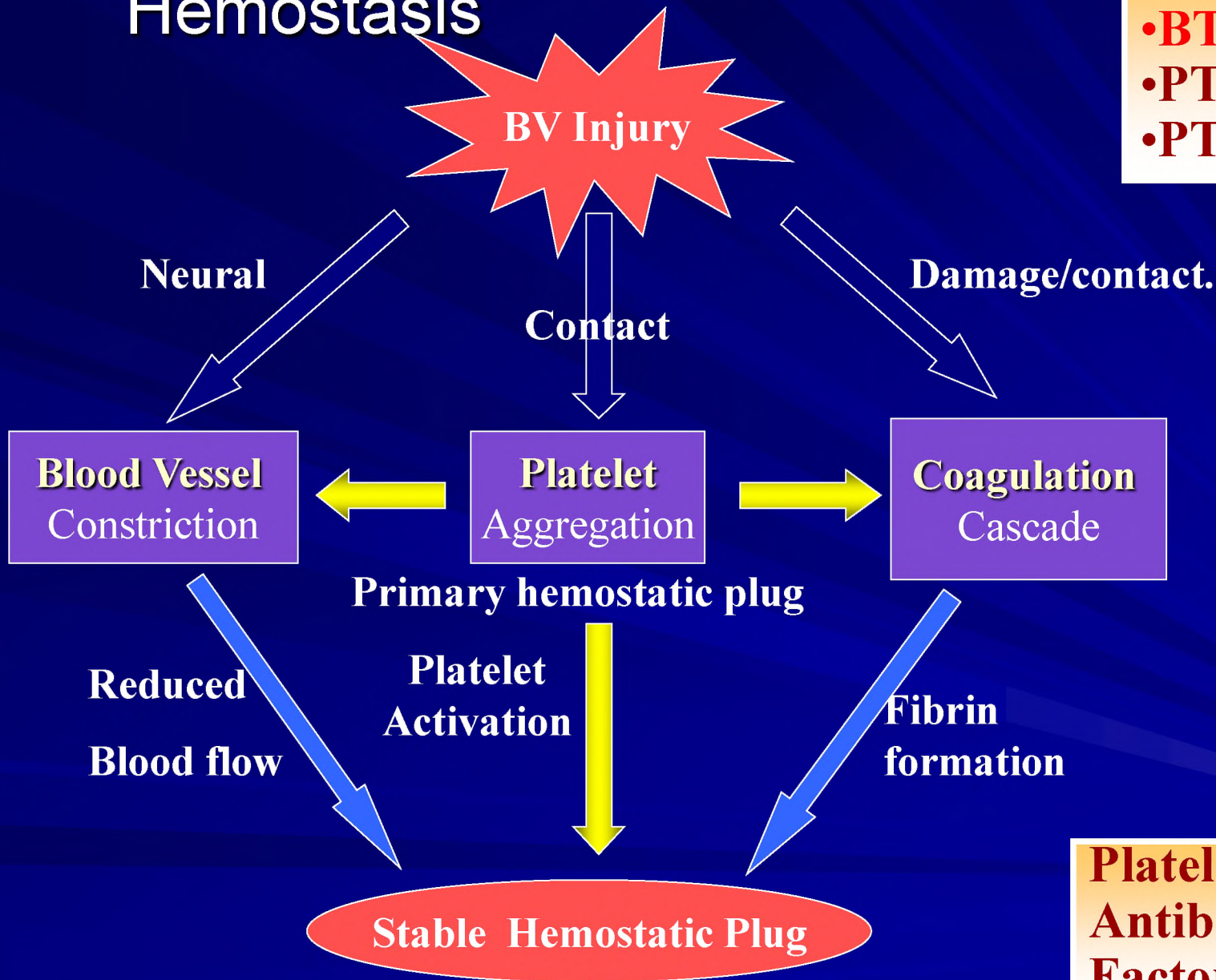


Hemorrhage



Hemostasis

- CBC-Plt
- BT,(CT)
- PT
- PTT



Platelet study
Antibody tests
Factor Assay

HEMOSTASIS

■ Primary Hemostasis

- Blood vessel contraction
- Platelet Plug Formation

■ Secondary Hemostasis

- Activation of Clotting Cascade
- Deposition & Stabilization of Fibrin

■ Tertiary Hemostasis

- Dissolution of Fibrin Clot
- Dependent on Plasminogen Activation

Classification:

- **Disorders of Blood vessels**
 - Scurvy, senile purpura, Henoch-Schonlein syndrome.
- **Disorders of Platelets**
 - Thrombocytopenia ITP, TTP, HUS, DIC.
 - Aspirin therapy, Thrombasthenia,
- **Disorders of Coagulation**
 - Extrinsic, intrinsic, combined.
- **Other disorders**
 - Post transfusion purpura, MPS, MDS.

Tests of Hemostasis:

■ Screening tests:

- Bleeding.T - 10m. Platelet & BV function
- Prothrombin.T – Extrinsic, aPTT – Intrinsic
- Thrombin.T – common path. (DIC)

■ Specific tests:

- Factor assays – hemophilia.
- Tests of thrombosis – TT, FDP, DDA,
- Platelet function studies:
 - Adhesion, Aggregation, Release tests.
- Bone Marrow study

Bleeding: Clinical Features

- Local - Vs - General, spontaneous . .
- Hematoma / Joint Bleeds- Coag
- Skin / Mucosal Bleeds – PLT
- wound / surgical bleeding –
- Immediate - PLT
- Delayed - Coagulation

Platelet



Petechiae, Purpura

Coagulation



Hematoma, Joint bl.

Bleeding

- It is called exit of the blood from the lumen of the blood vessel or cardiac cavity in the environment (external bleeding) or body cavity (internal bleeding).
- **External** bleeding may be open: coughing up blood, vomiting blood, discharge of blood in the faeces, and others.
- **Internal** bleeding is accompanied by the accumulation of blood in the body cavities: the pericardial cavity (hemopericardium), pleura (hemothorax), abdominal (hemoperitoneum). Hemorrhage is a special kind of internal bleeding, it is characterized by the accumulation of blood in the tissues.

The reasons (for R. Virhov):

- **per rhexin** (Gk) by breaking the vascular wall or the wall of the heart (heart attack as a result of rupture of the heart or an aortic aneurysm, and others.);
- **per diabrosin**, by corrosion, i.e. the enzymatic digestion of components of the vessel wall as in hemorrhagic pancreatitis, or by the action of lytic enzymes pus. A defect in the vessel wall may be formed during the decay of the tumor, in the hearth of purulent inflammation, in destructive processes stomach (under the action of gastric juice) and the lungs (TB in the wall cavity), and others.
- **per diapedesin** in the absence of apparent gaps in the vessel wall, due to leakage of red blood cells through the wall, diapedetic hemorrhage due to increased permeability of the capillary walls, venules, arterioles. Observed in hypoxic brain damage, arterial hypertension, systemic vasculitis, infectious and infectious and allergic diseases, and others. With the development of hemorrhagic syndrome diapedetic hemorrhage take systemic nature.

Vascular disorders:

- **Petechiae, purpura, ecchymoses**
- **senile purpura**
- **vitamin C deficiency (scurvy)**
- **Connective tissue disorders**
- **Infections – Meningococcus**
- **Henoch-Schonlein Purpura-Immu**

Senile Purpura

Circulatory Disorders



Petechiae in Vasculitis

(Rocky Mountain Spotted Fever)



Henoch-Schonlein purpura

- Immune disorder
- Children
- Follows infection
- Petechiae with edema and itching.



Henoch-Schonlein purpura

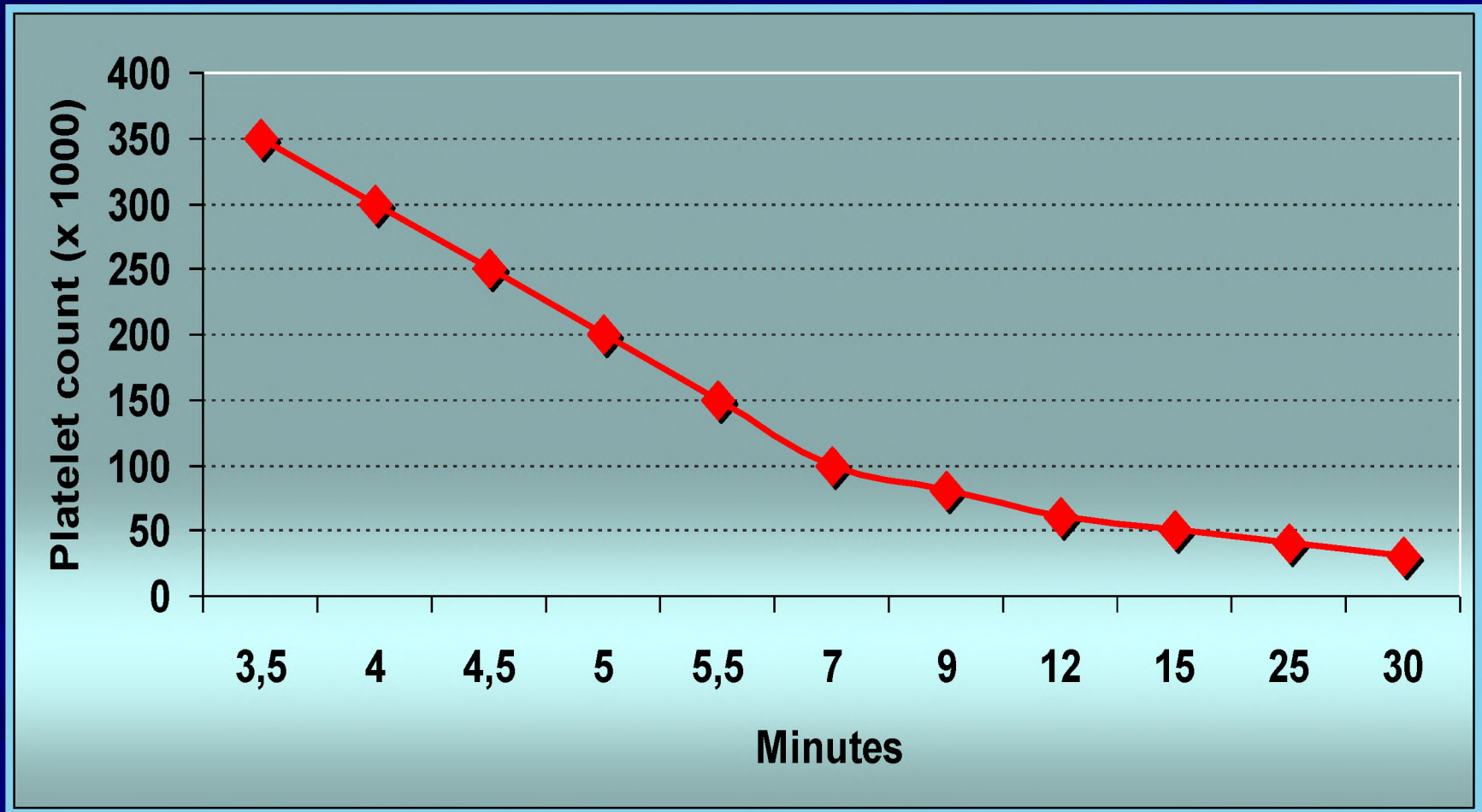


20y Male, fever, painful symmetric polyarthrititis for a day. During the next two days, edema and palpable purpura developed.

Platelet Disorders - Features:

- **Mucocutaneous bleeding**
- **Petechiae, Purpura, Ecchymosis.**
- **spontaneous bleeding after trauma**
- **CNS bleeding (severe ↓ plt)**
- **Prolonged bleeding time (BT)**

BLEEDING TIME vs. PLATELET COUNT





Idiopathic Thrombocytopenic Purpura (ITP)

- **Acute - children (post infection)**
- **Chronic - adults (↑ females, 20-40 yrs)**
- **autoimmune disorder**
- **antiplatelet antibodies (IgG)**
- **IgG coated platelets removed by spleen**
- **Usually ↑ megakaryocytes in BM**

Platelet dysfunction:

Inherited Disorders:

- **Bernard-Soulier disease**
 - large platelets, failure of adhesion
- **Glanzmann's thrombasthenia**
 - normal size, failure of aggregation

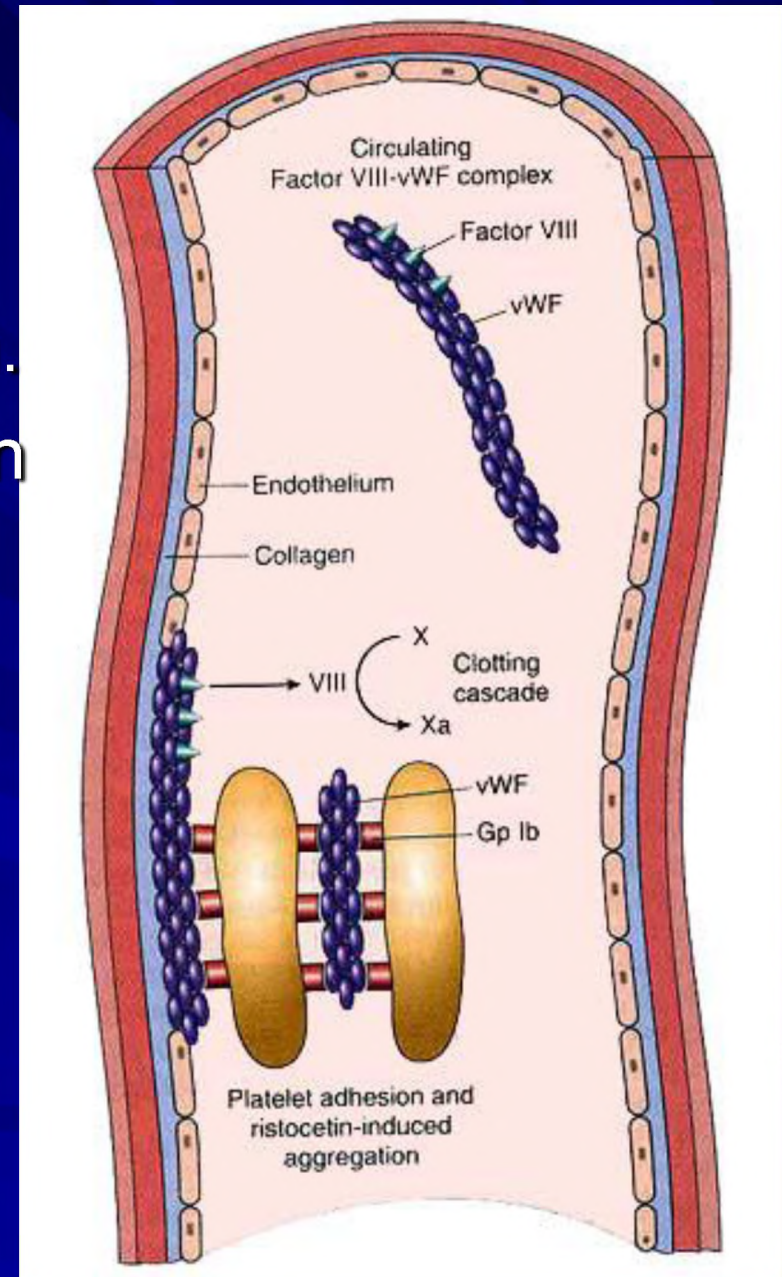
Acquired Disorders:

- **Drugs - Aspirin, Alcohol, Uremia,**

Von-Willebrand Disease:

- **Coagulation + PLT disorder:**
- **Congenital disorder**
- **Deficiency of vWF molecule**
- **Part of FVIII,**
- **Mediates platelet adhesion**
- **Prolonged Bleeding time**
- **Low Factor VIII & long aPTT**
- **Mucocutaneous bleeding**

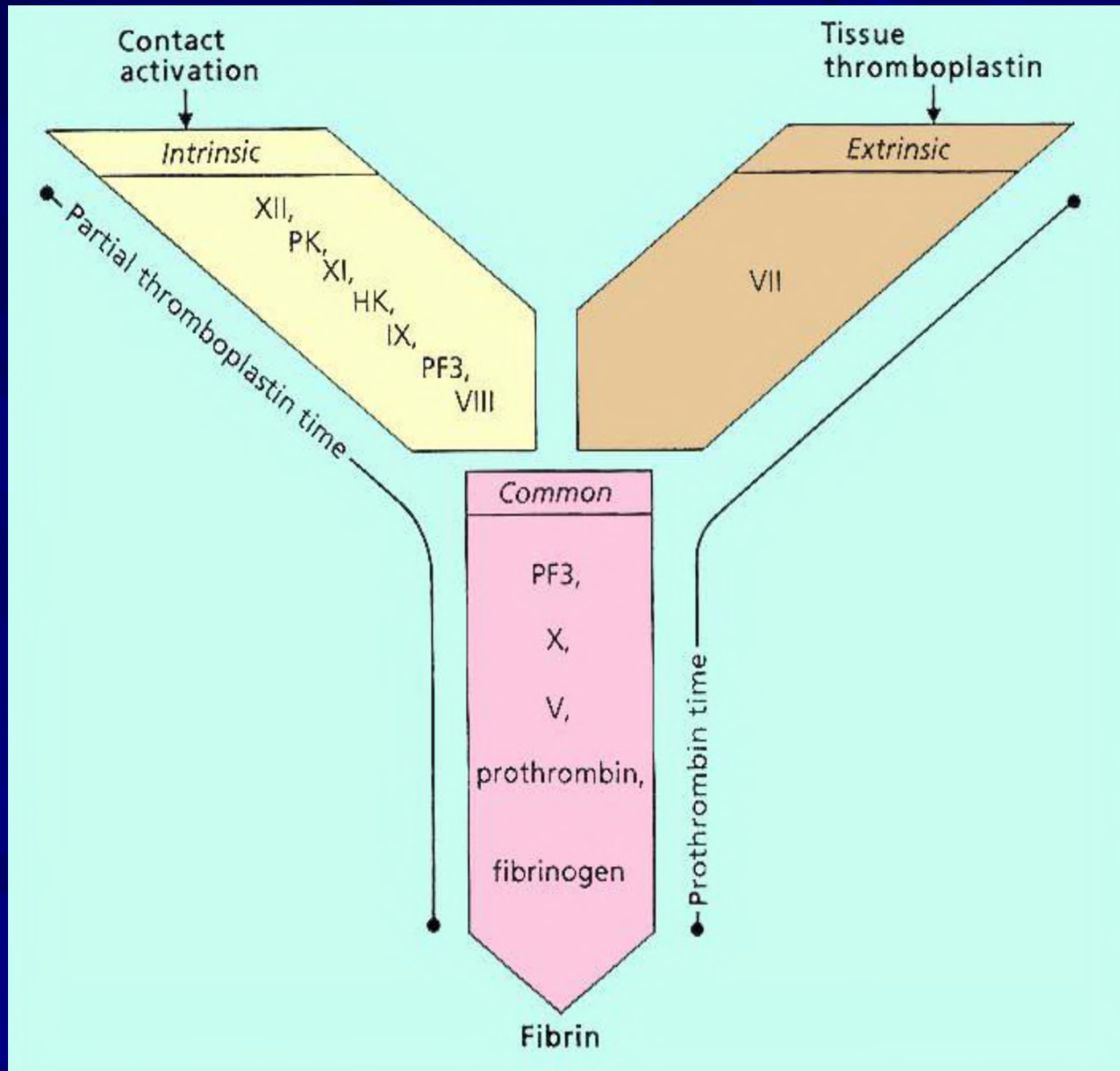
- **Von-Willebrand Disease**
- vWF: F-VIII & PLT function.
- Defective Platelet Adhesion
- Skin Bleeding
- Prolonged Bleeding time.
- Low Factor VIII levels.



Coagulation disorders:

- **Deficiencies of Clotting factors**
- **Onset - delayed after trauma**
- **Deep bleeding**
 - Into joints - Hemarthroses
 - Into deep tissues – Hematoma
 - large skin bleed – Ecchymoses

Blood Coagulation & Tests



CT- Large hematoma of psoas muscle

