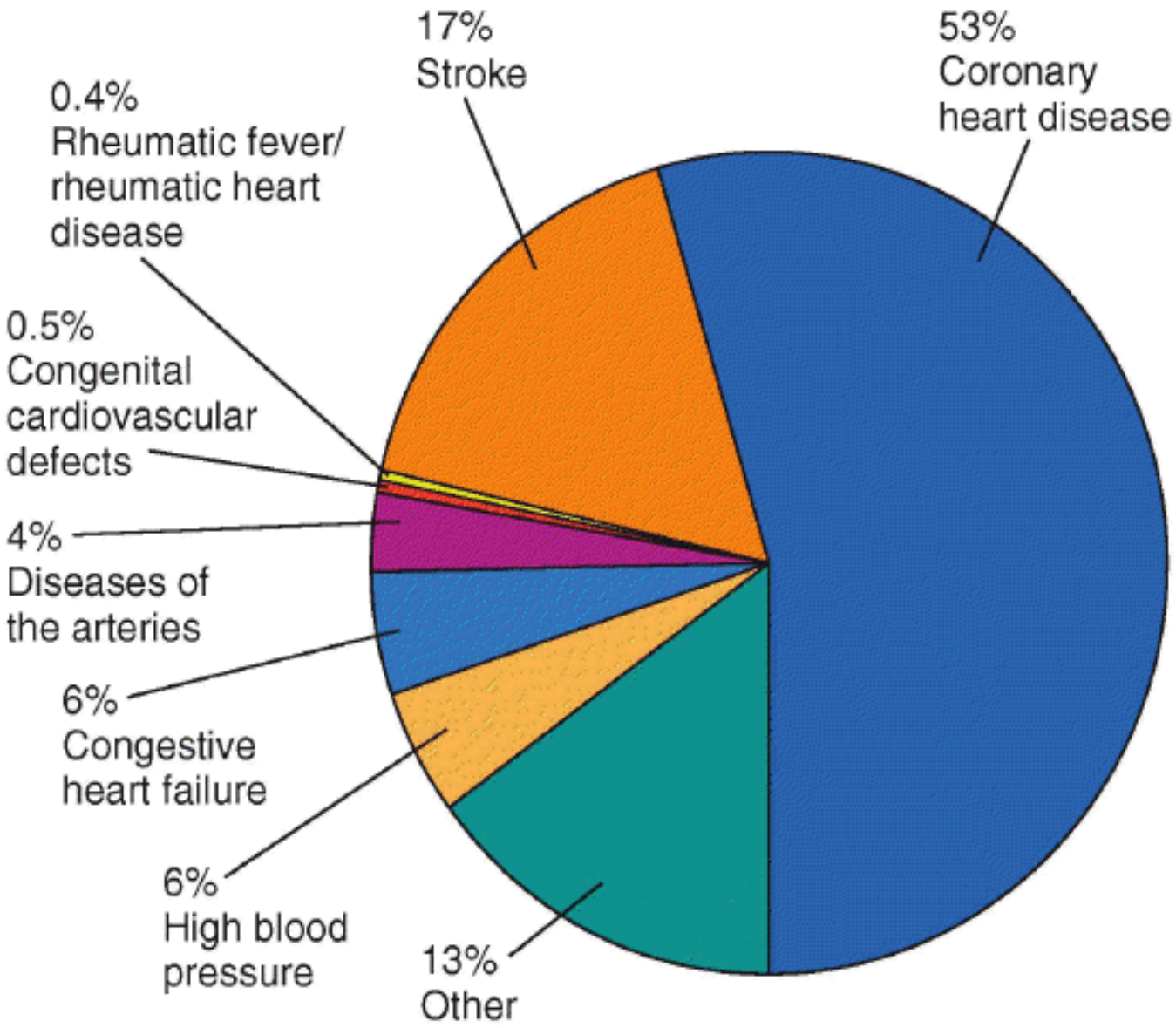
IHD-Ischemic Heart Disease

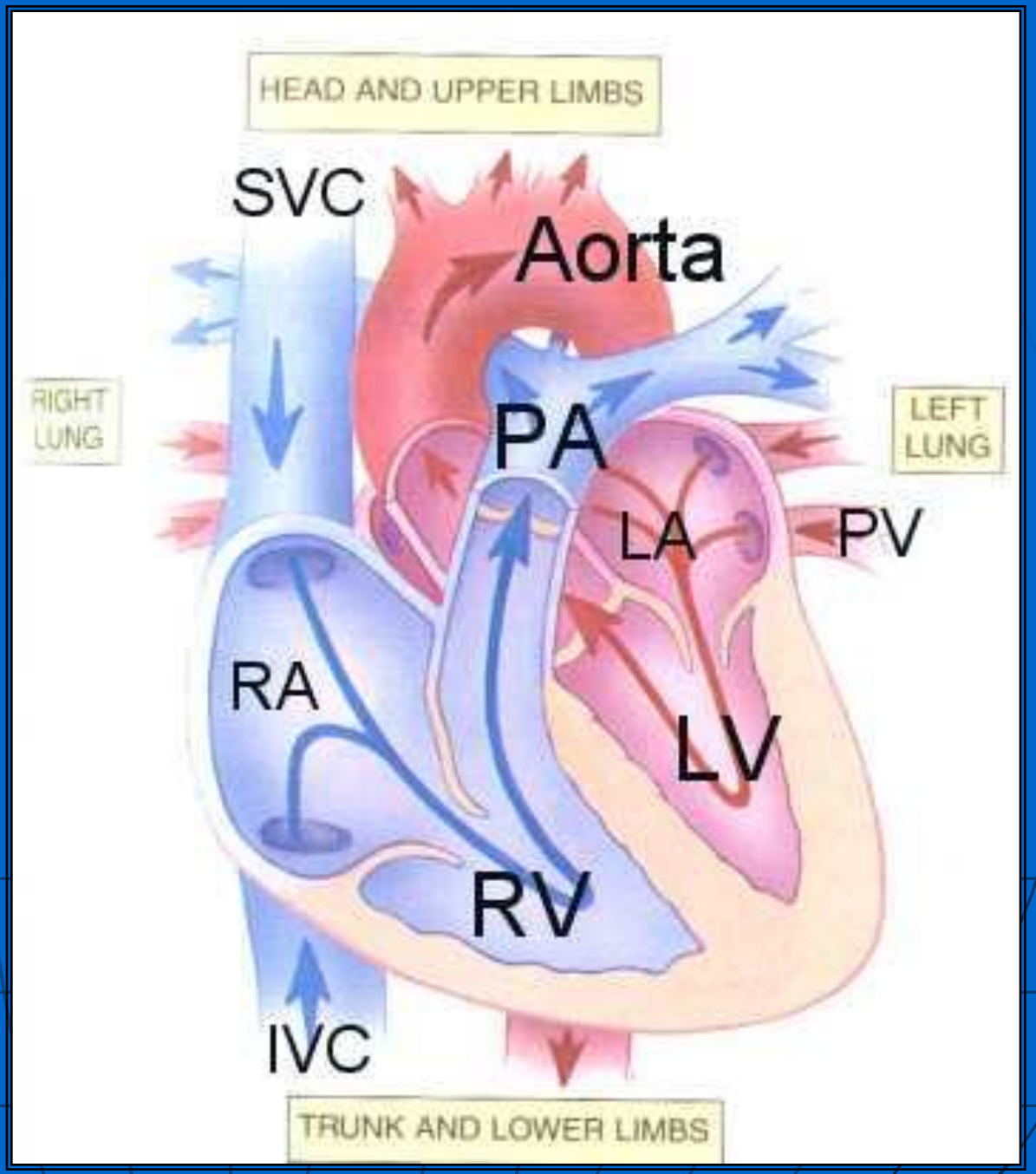
Ischemic Heart Disease

- This is the biggie in the western countries, Russian Federation.
- Atherosclerosis of coronary arteries.
- Acute vs. chronic ischemia.
- Four basic patterns
  - Angina pectoris
  - Myocardial infarction
  - Chronic ischemia leading to CHF and cardiosclerosis
  - Sudden death from arrhythmia

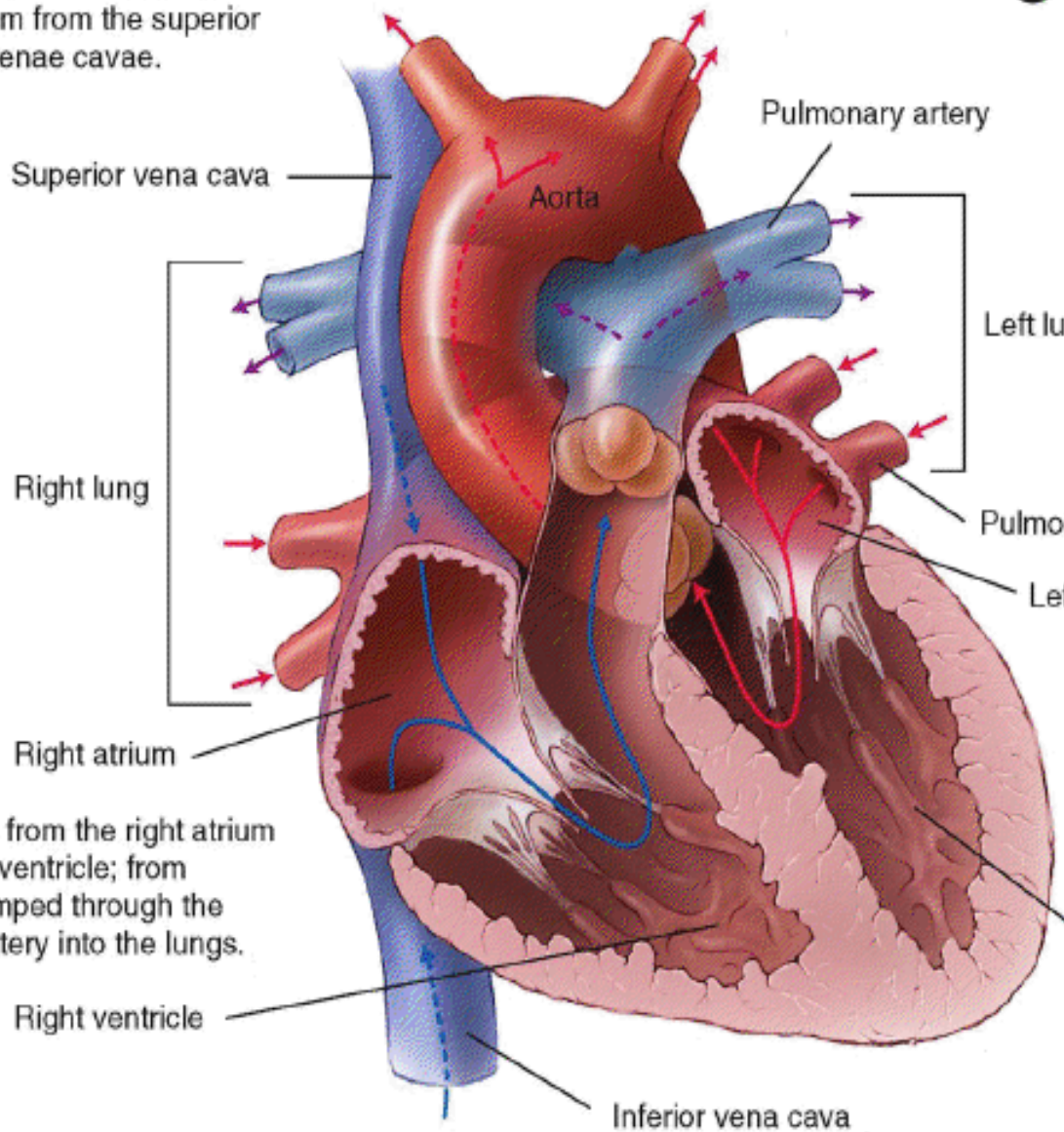
Percentage Breakdown of Deaths from Cardiovascular Disease in the United States, 2001



Anatomy

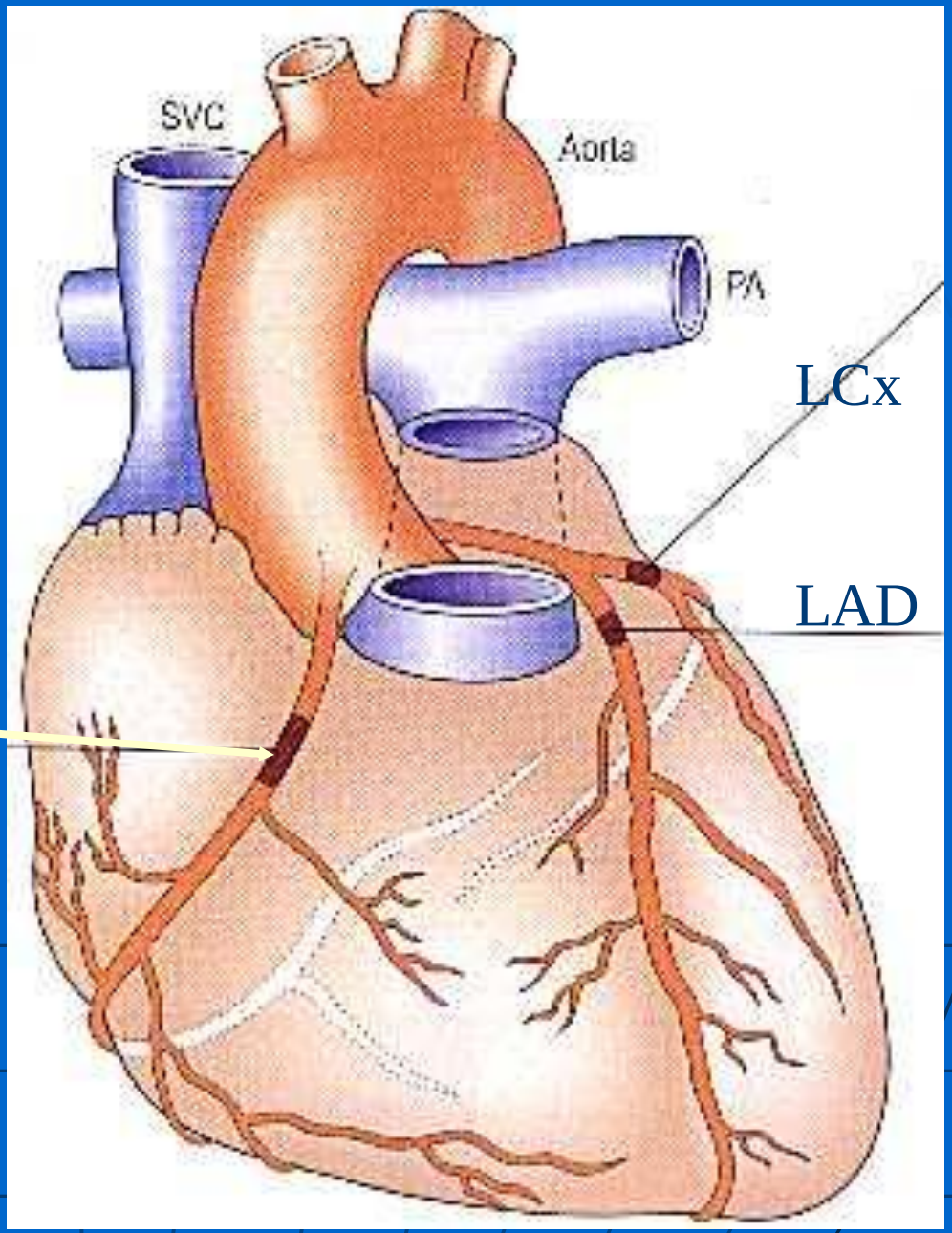

HEAD AND UPPER LIMBS
SVC
Aorta
RIGHT LUNG
PA
LEFT LUNG
LA
PV
RA
LV
RV
IVC
TRUNK AND LOWER LIMBS

Anatomy of the Heart



Coronary Arteries

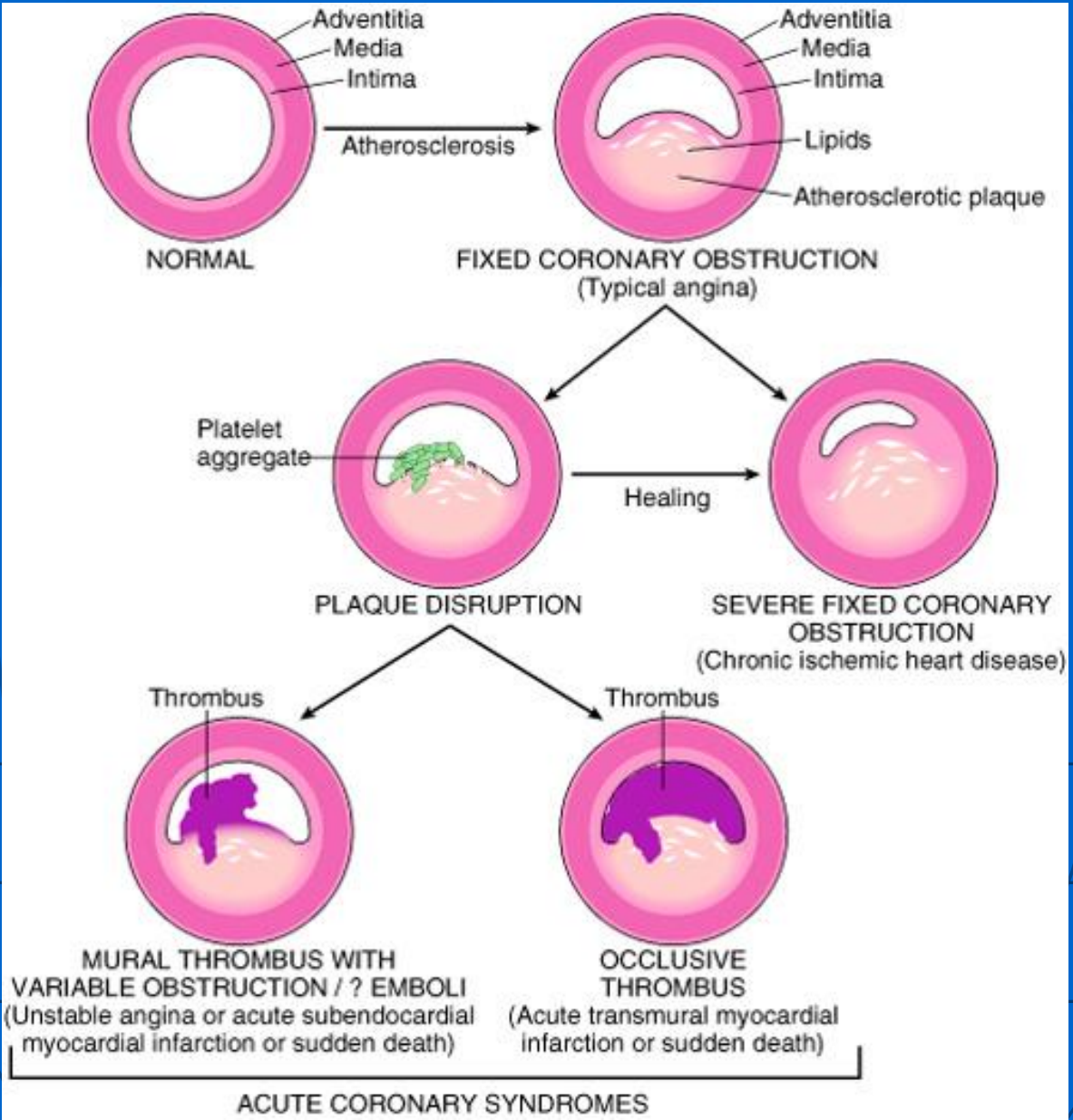
- Left Coronary A.
  - L.A.Descending
  - Left Circumflex
- Right Coronary A.



Ischemic Heart Disease

- Common Health problem.
- High Mortality & Morbidity.
- Etiology – common Atherosclerosis
- Two major types Angina & MI.
- Risk factors –
  - Hypertension
  - Hypercholesterolemia
  - Diabetes
  - Smoking, Life style, Diet, Genetic.

Coronary Atherosclerosis



Patterns of IHD:

- Angina Pectoris
- Myocardial Infarction
- Sudden cardiac death
- Chronic ischemic heart disease (heart failure without the pain, i.e. dyspnea, ankle edema etc., but no pain)

Ischemic heart disease

- deficiency of oxygenated blood, mostly commonly caused by inadequate supply due to coronary artery disease
- causes:
  - coronary artery disease
  - increased myocardial demand
  - systemic: shock
  - reduced oxygen carry capacity of blood (e.g. anaemia)
  - diversion: cyanotic congenital heart diseases
  - reduced oxygen: severe lung disease

Pathogenesis:

- Obstruction to blood flow.
  - Atheroma, Thrombosis Embolism
- Diminished coronary perfusion.
- interaction between
  - fixed atherosclerotic narrowing of coronary arteries
  - intraluminal thrombosis
  - vasospasm
- Ischemia – Angina
- Necrosis -Infarction
  - Inflammation
  - Granulation tissue
  - Fibrous scarring.

Fixed atherosclerotic obstruction

- significant obstruction is defined as 75% reduction of the cross sectional area of the artery
- at this level of obstruction, vasodilatation can not compensate for the reduced blood flow

Role of acute plaque change

- fixed obstructions - collateral have time to develop and compensate
- acute changes to plaques - too rapid for compensation by collaterals
- consists of:
  - intraplaque haemorrhage
  - fissuring
  - ulceration
- consequences of acute change:
  - superadded thrombosis +/- embolism
  - vasoconstriction - damaged endothelium is unable to secrete relaxing factors

Causes of acute plaque events (e.g. ulceration)

- vasospasm fracturing of the plaque
- tachycardia causing physical stress
- stresses producued by blood flow in stenotic vessels
- some plaques more prone to rupture:
  - those with eccentrically positioned plaque
  - those with a large core of necrotic debris and lipid
  - those with a thin fibrous cap
- uncertain sometimes

Clinical syndromes of ischemic heart disease

- angina pectoris
  - stable angina
  - Prinzmetal's or variant angina
  - unstable angina
- myocardial infarction
- sudden death
- chronic ischaemic heart disease

Angina pectoris

- stable angina:
  - reproducible chest pain caused by increased physical activity or emotional excitement and relieved by rest or vasodilators
  - caused by reduction of coronary perfusion by a stable atherosclerotic disease
  - over a long period, repeated episodes of impaired flow may lead to development of fine fibrosis in the myocardium, with death of individual cardiac muscle fibers
  - anastomotic vessels frequently develop to compensate for areas of vascular stenosis
- variant angina:
  - chest pain occurs at rest, responds to vasodilators
  - thought to be caused by vasospasm
- unstable angina:
  - chest pain with progressively less effort, often at rest, prolonged duration
  - caused by fissuring, ulceration or haemorrhage into a plaque with thrombosis, embolisation or vasospasm
  - fissuring = plaques breakdown resulting in a deep cleft in a lipid-rich plaque that either precipitates thrombus development in the lumen or causes bleeding into the body of the plaque, with resultant ballooning into the lumen

Angina Pectoris

- Ischemia – reduction of the heart's blood and oxygen supply
- The more serious the oxygen deprivation the more severe the pain
- Nitroglycerin – drug used to relax (dilate) the veins
- Beta blockers control potential overactivity of the heart muscle

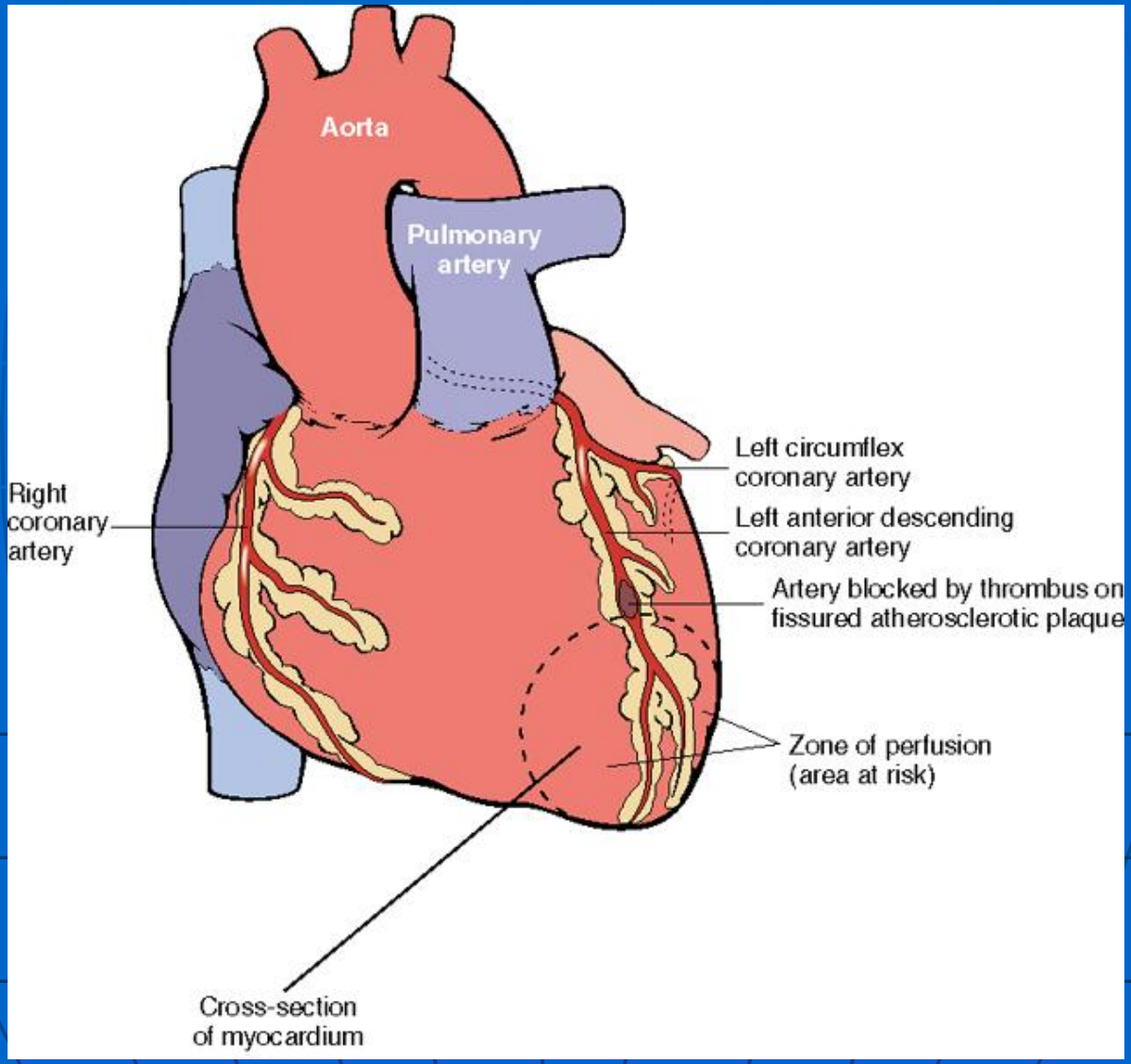
Myocardial infarction

- necrosis of cardiac mucsle due to lack of oxygen
- risk factors:
  - family history
  - hypercholestrolaemia
  - cigarette smoking
  - diabetes mellitus
  - hypertension
  - age
  - male gender

Coronary Heart Disease

- Myocardial infarction (MI) or heart attack – blood supplying the heart is disrupted
- Coronary thrombosis – blood clot in the artery
- Embolus – when the blood clot is dislodged and moves through the circulatory system
- Collateral circulation - if blockage to the heart is minor, an alternative blood flow is selected

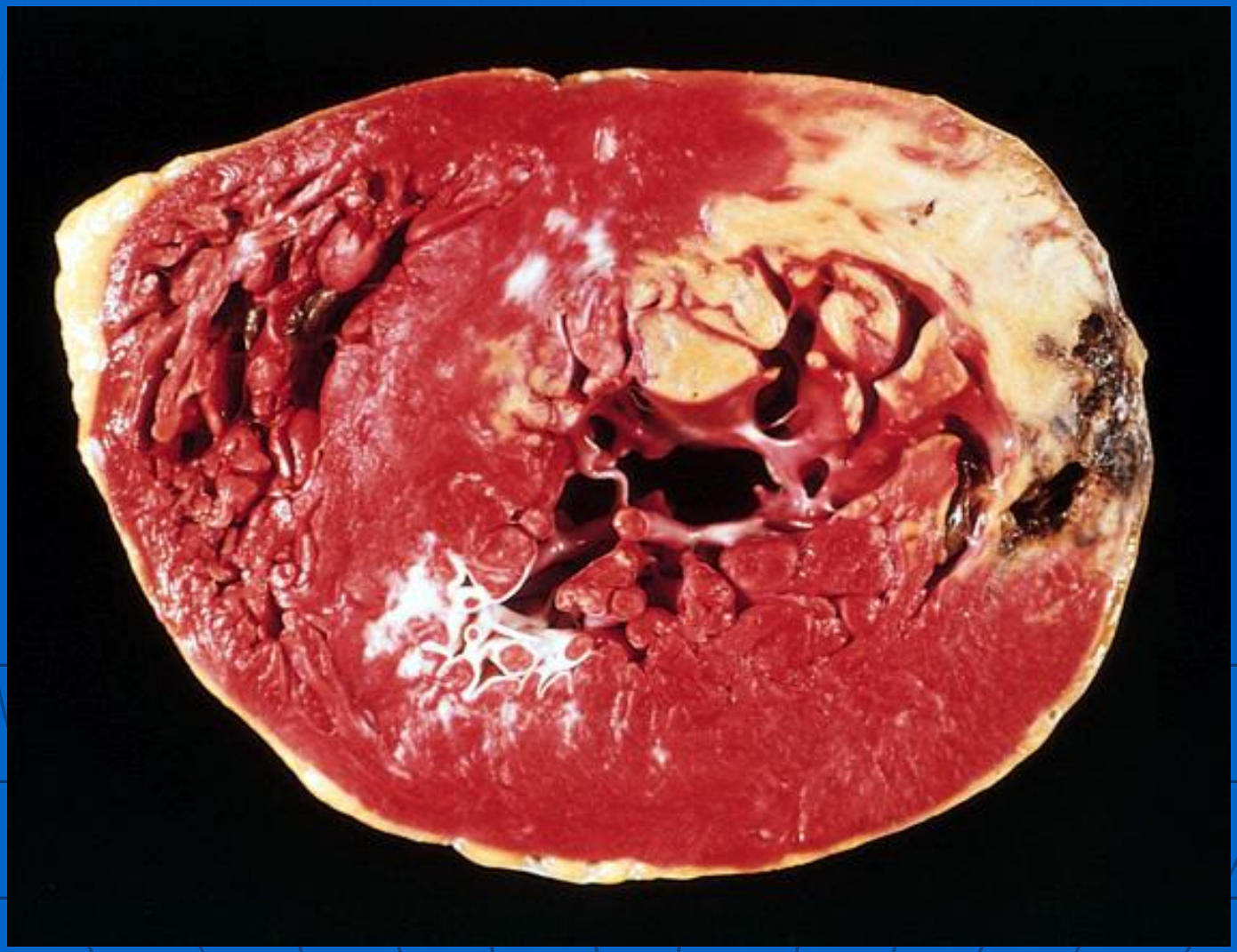
Myocardial Infarction



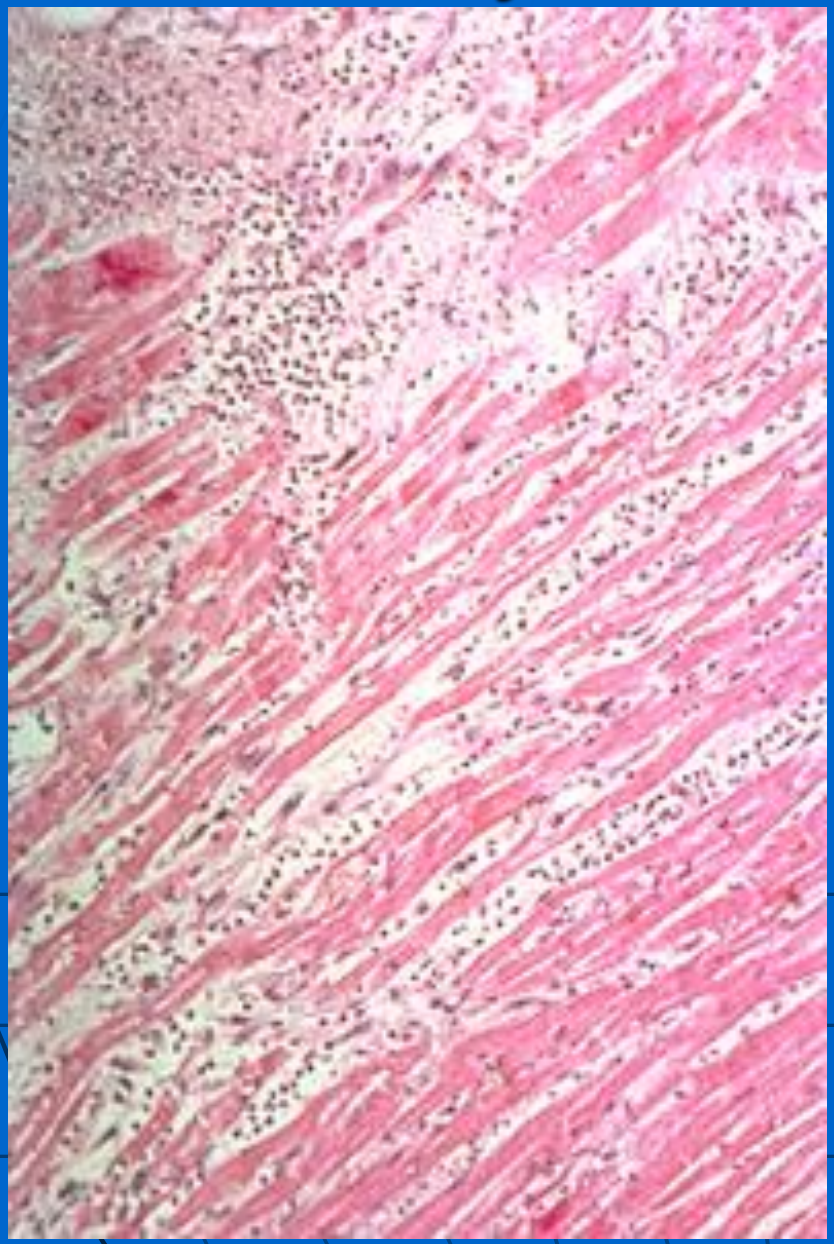
MI - clinical presentation

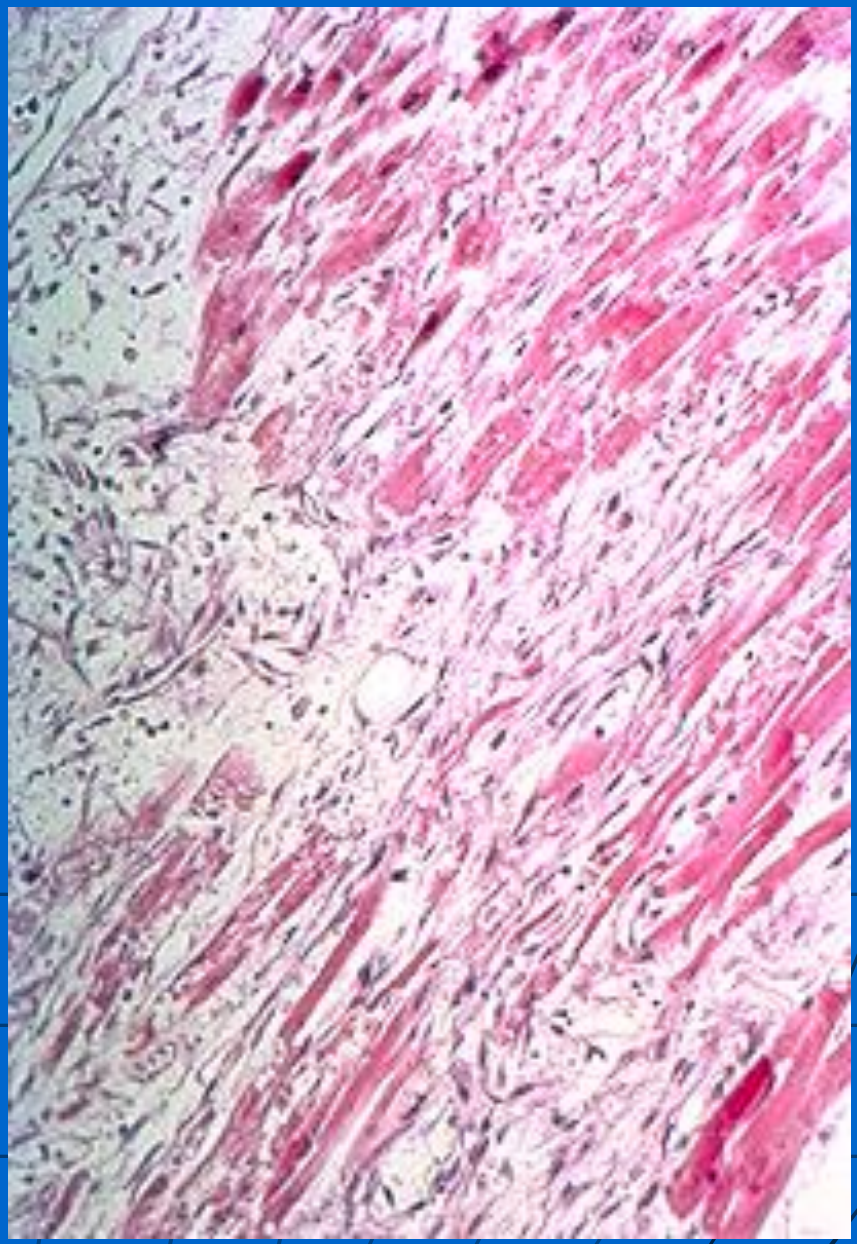
- sudden and devastating chest pain, radiating to left shoulder, arm, jaw
- +/- vomiting, nausea, sweating, shortness of breath
- burning epigastric discomfort, "indigestion"
- 10% asymptomatic, discovered later

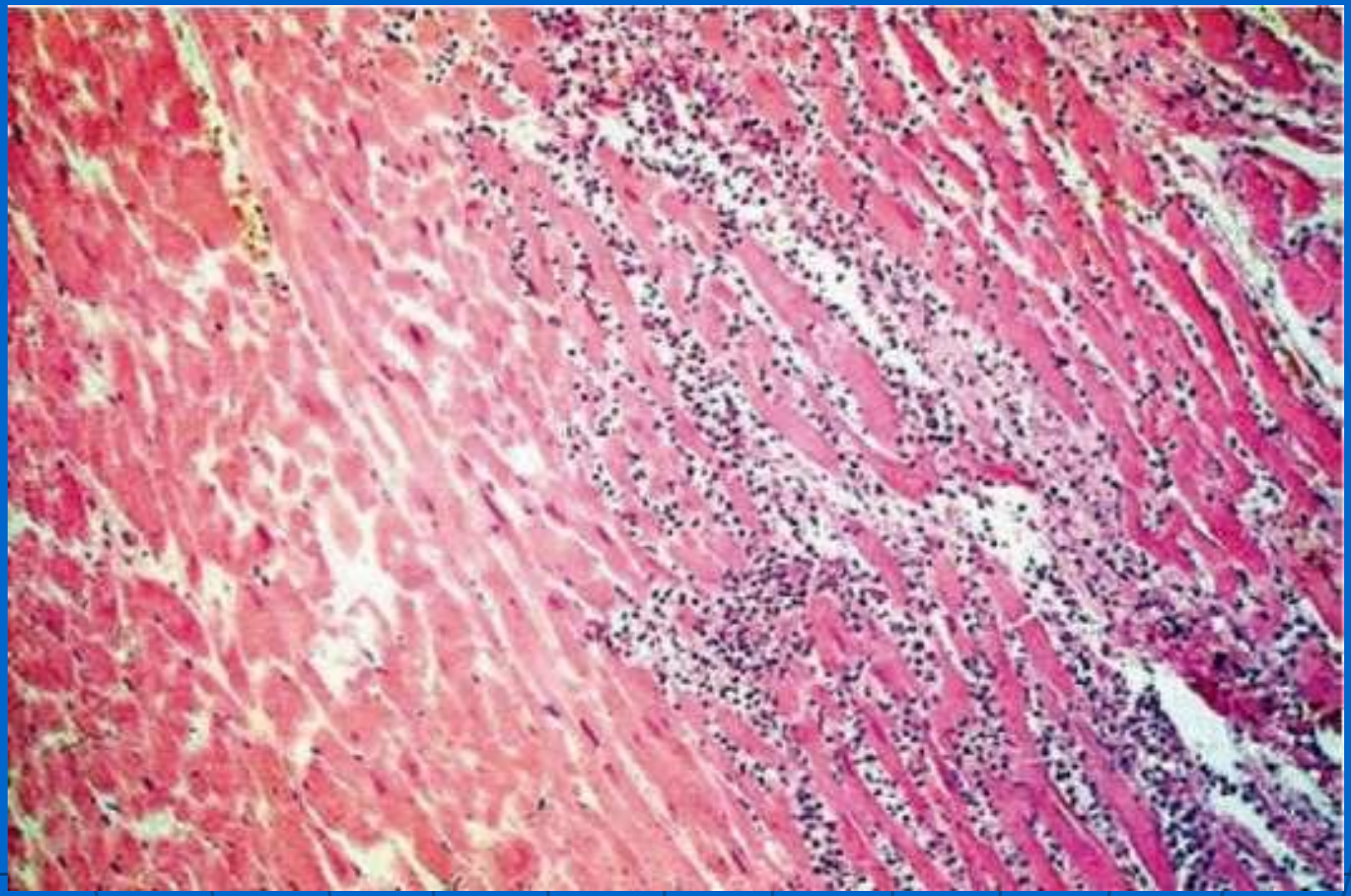
Myocardial Infarction



Myocardial Infarction







The focus of necrosis of cardiac muscle tissue. Around the focus of necrosis demarcation inflammation is in the form of severe leukocyte infiltration and vascular hyperemia. Stained with hematoxylin and eosin.

Types of MI

- transmural infarction (regional infarct) - 90% of cases
  - infarcted area is the full thickness of the muscle in distribution of a specific coronary artery(s)
  - caused by thrombosis superadded to acute plaque events
  - *i.e. thrombus --> blockage of entire coronary artery --> infarct of all the muscle supplied by that coronary (therefore full thickness of wall)*
  - *note: if there is lysis of the thrombus or a collateral supply to the myocardium, the infarct will be limited to the subendocardial zone - i.e. regional subendocardial infarction*

Types of MI

- subendocardial infarction (circumferential infarct) - 10% of cases
  - the ischaemic necrosis is limited to the inner one-third or one-half of the cardiac wall, extending beyond territory of a single artery
  - caused by diffuse, stenosing atherosclerotic coronary artery disease and a reduction in overal cardiac blood flow
  - *i.e. atherosclerosis of coronary arteries --> there is a general hypoperfusion of the main coronary arteries --> normally, enough blood reaches heart, BUT, due to an episode requiring increased blood flow (e.g. exercise), perfusion is inadequate, and an infarct occurs, but because the coronary arteries travel near the pericardium, enough oxygen reaches the outer parts of the muscle, but not the inner parts of the muscle)*

Myocardial response to ischaemia

- dependent on duration and severity of ischaemia
  - reversible phase:
    - 60 seconds - loss of contractility
    - minutes - ultrastructural changes
  - irreversible:
    - 20-40 minutes of severe ischaemia
    - myocyte necrosis
    - a wavefront of coagulative necrosis, starting at the subendocardial zone

Myocardial Infarction-MI

- "Death of heart tissue due to lack of blood supply"
- Atherosclerosis is the common cause.
- Coagulative necrosis – intact cell shape.
- Severe chest pain, breathlessness & sweating
- Complications – cardiogenic shock, Death or Cardiac failure.

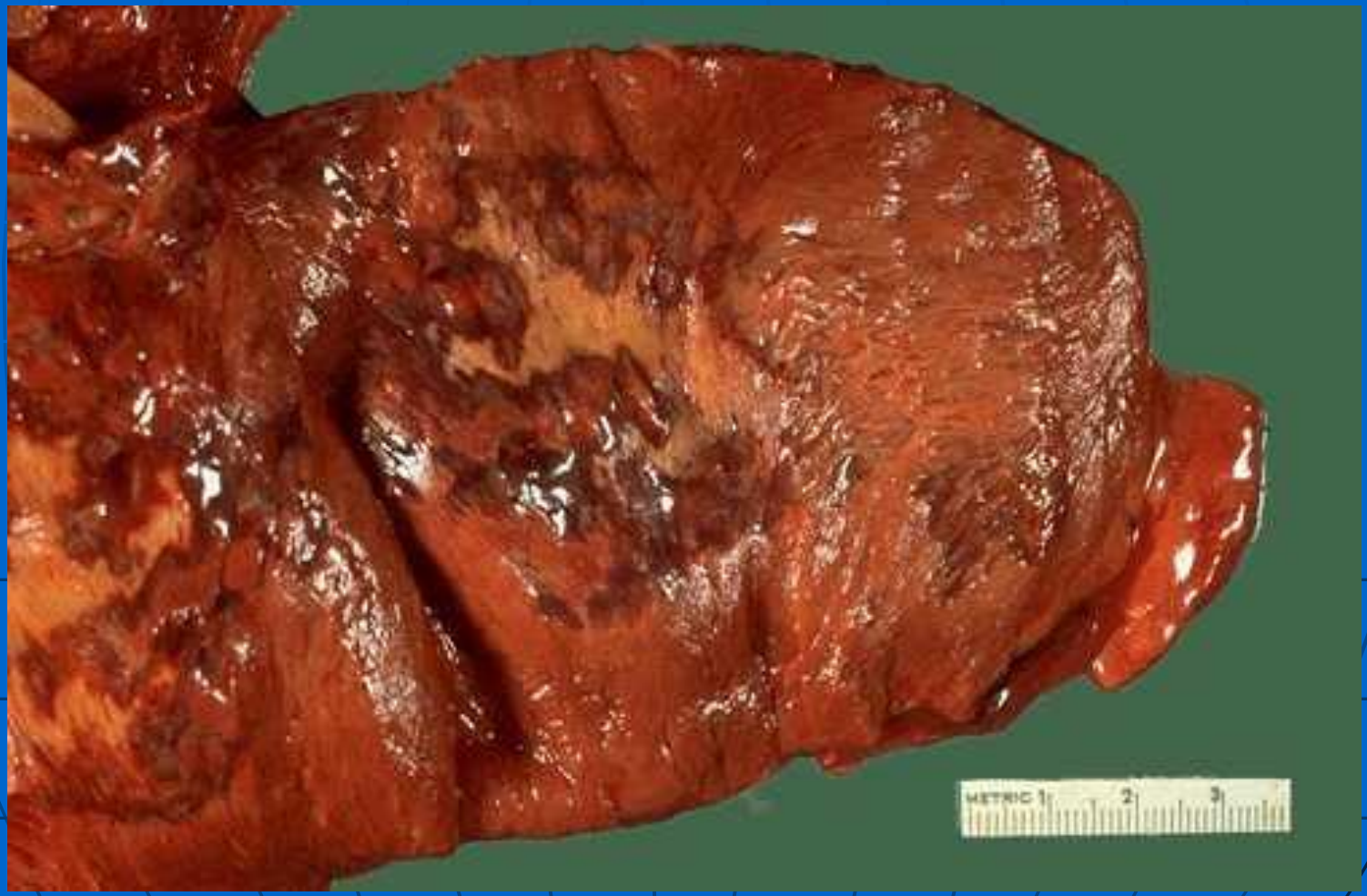
Processes occuring after MI:

- over following 8 weeks
- acute inflammation
- organisation
- scarring

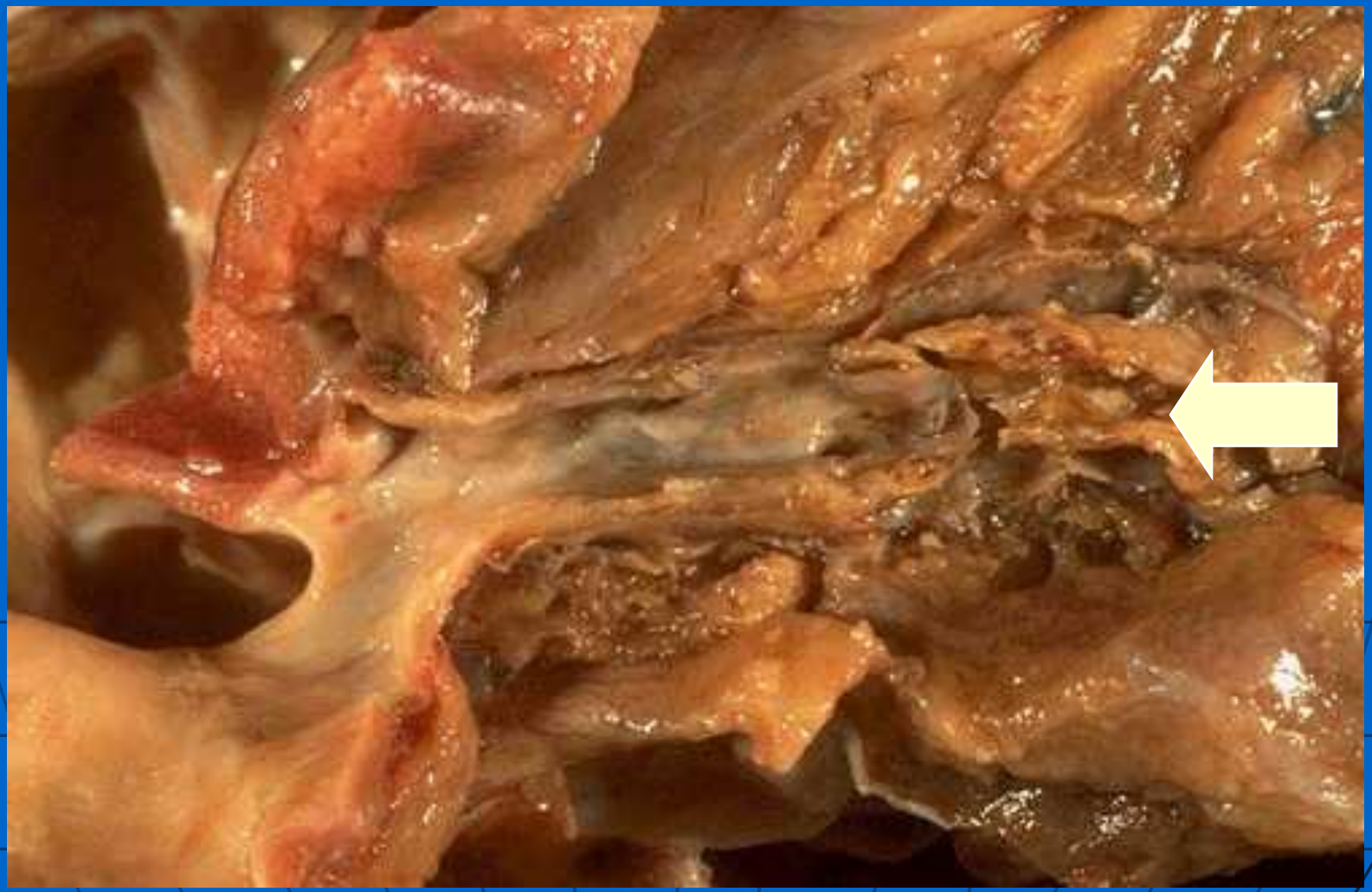
Gross appearance

- *0-12:* inapparent (but, within *3 hrs* triphenyltetrazolium chlordide (TTC) will not stain the affected part of heart because dehydrogenase is depleted [note TTC normally stains a heart blue/black])
- *12-24:* blotchy areas that are palor and congested
- *1-3 days:* the dead area appears soft and pale with a slight yelow colour
- *3-14 days:* softened yellow central area with a hyperaemic rim - granulation tissue (can rupture to cause haemopericardium); note that in many pots the granulation tissue extends throughout the dead muscle
- *weeks:* fibrotic white thin scar (can for aneurysm)

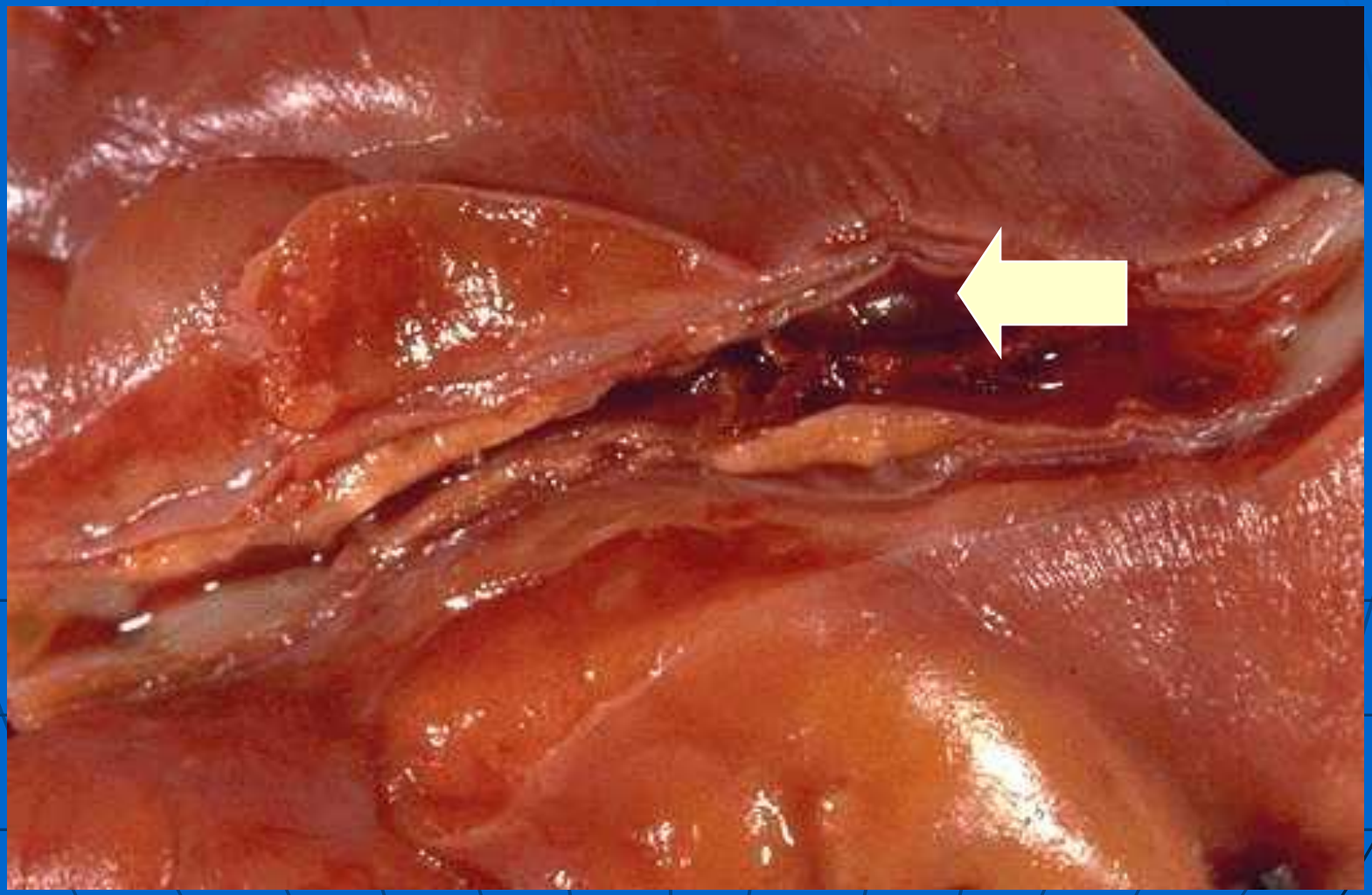
Myocardial Infarction - Gross



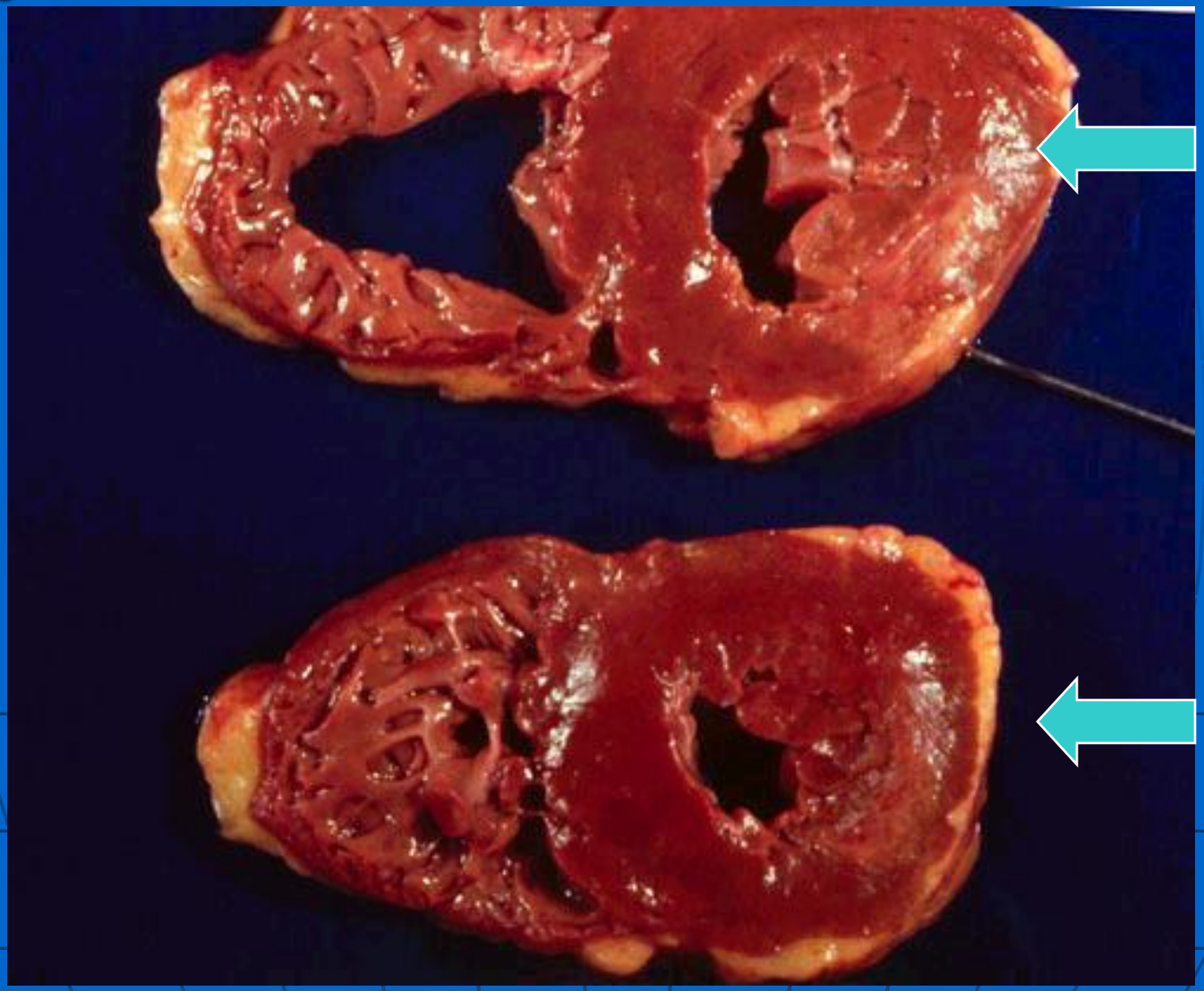
Coronary Atherosclerosis



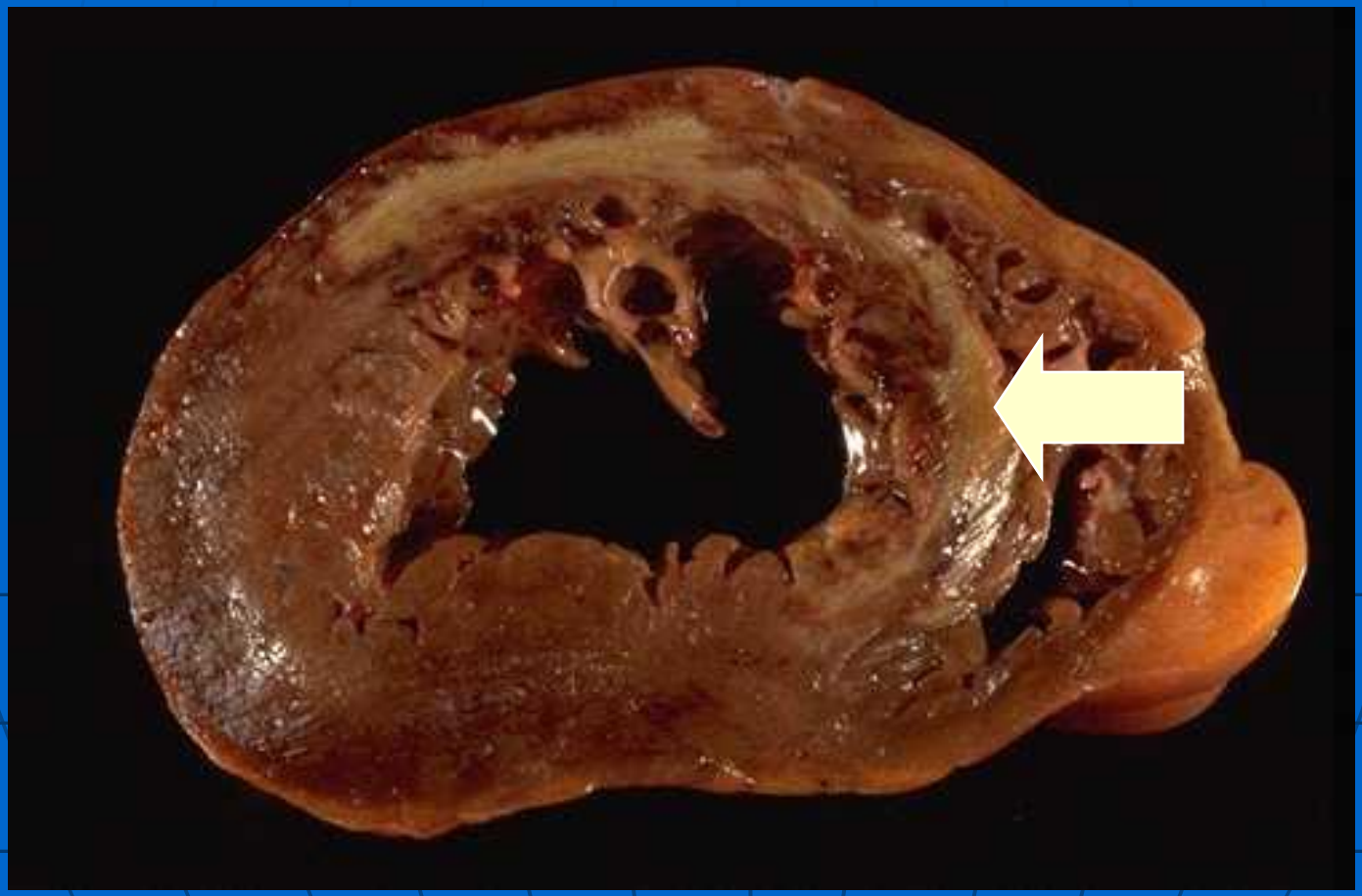
Coronary Atherosclerosis with Thrombosis



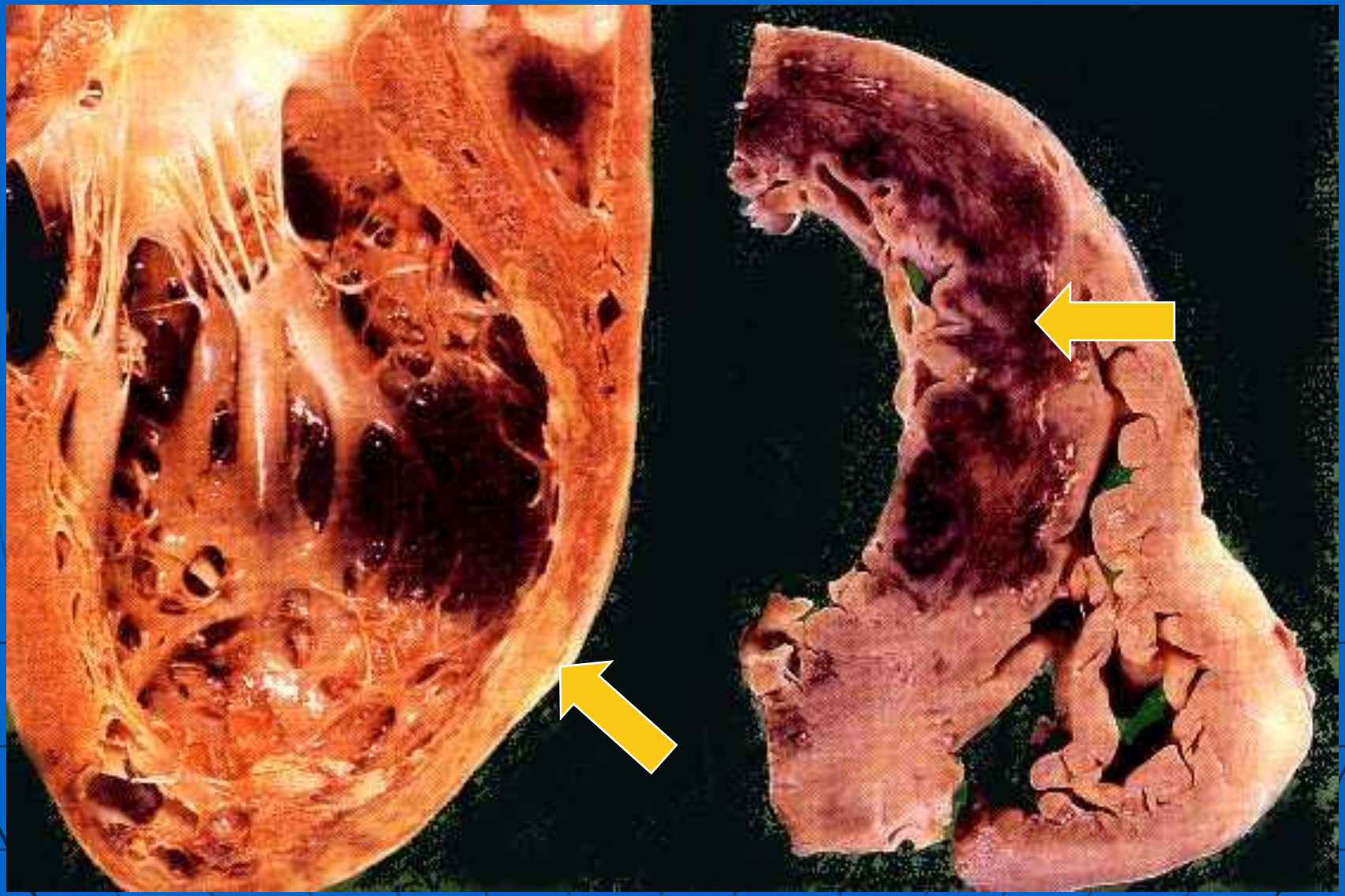
Myocardial Infarction – 1 wks.



Myocardial Infarction - CS



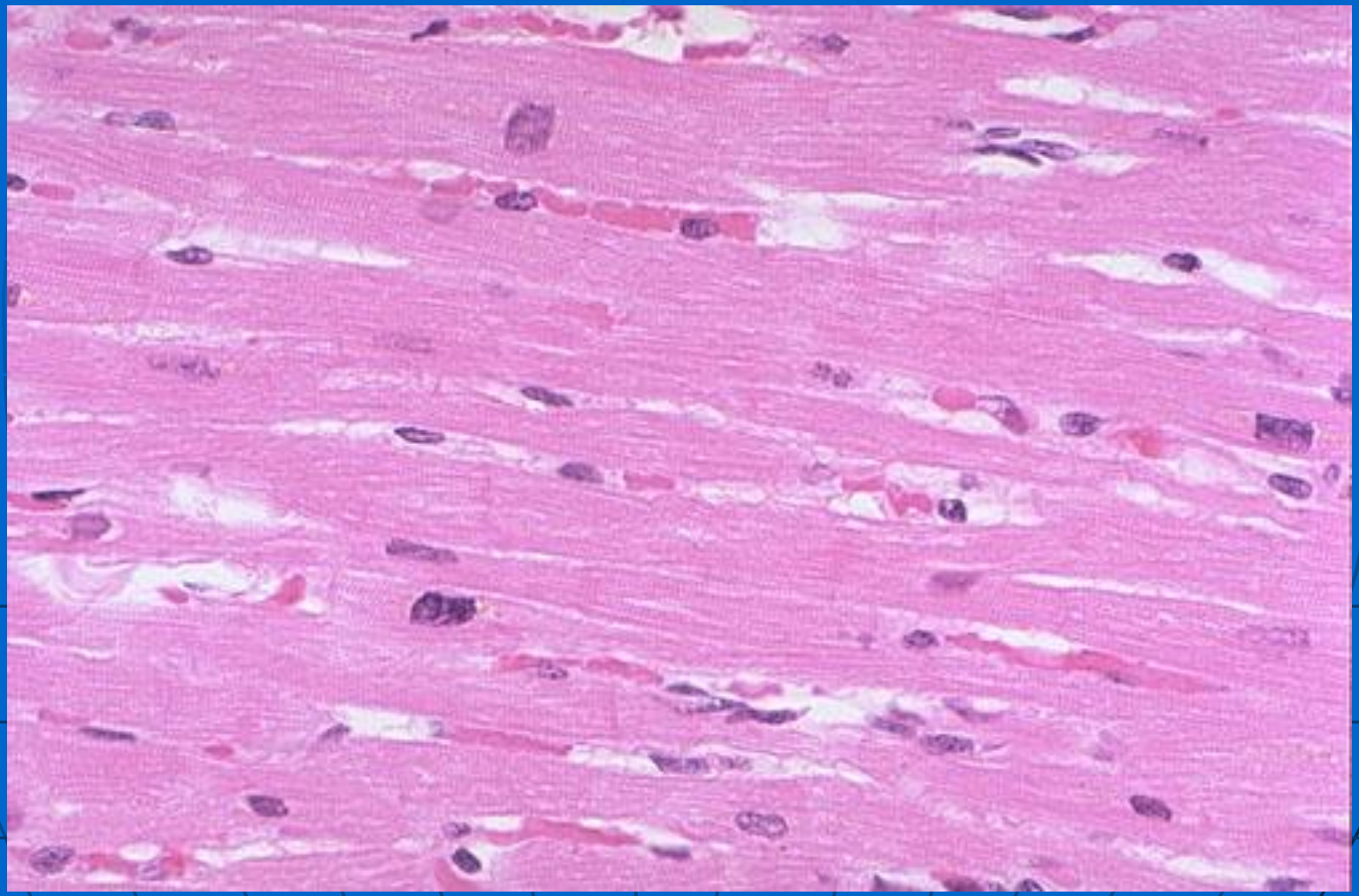
2wk - Myocardial Infarction - 3d



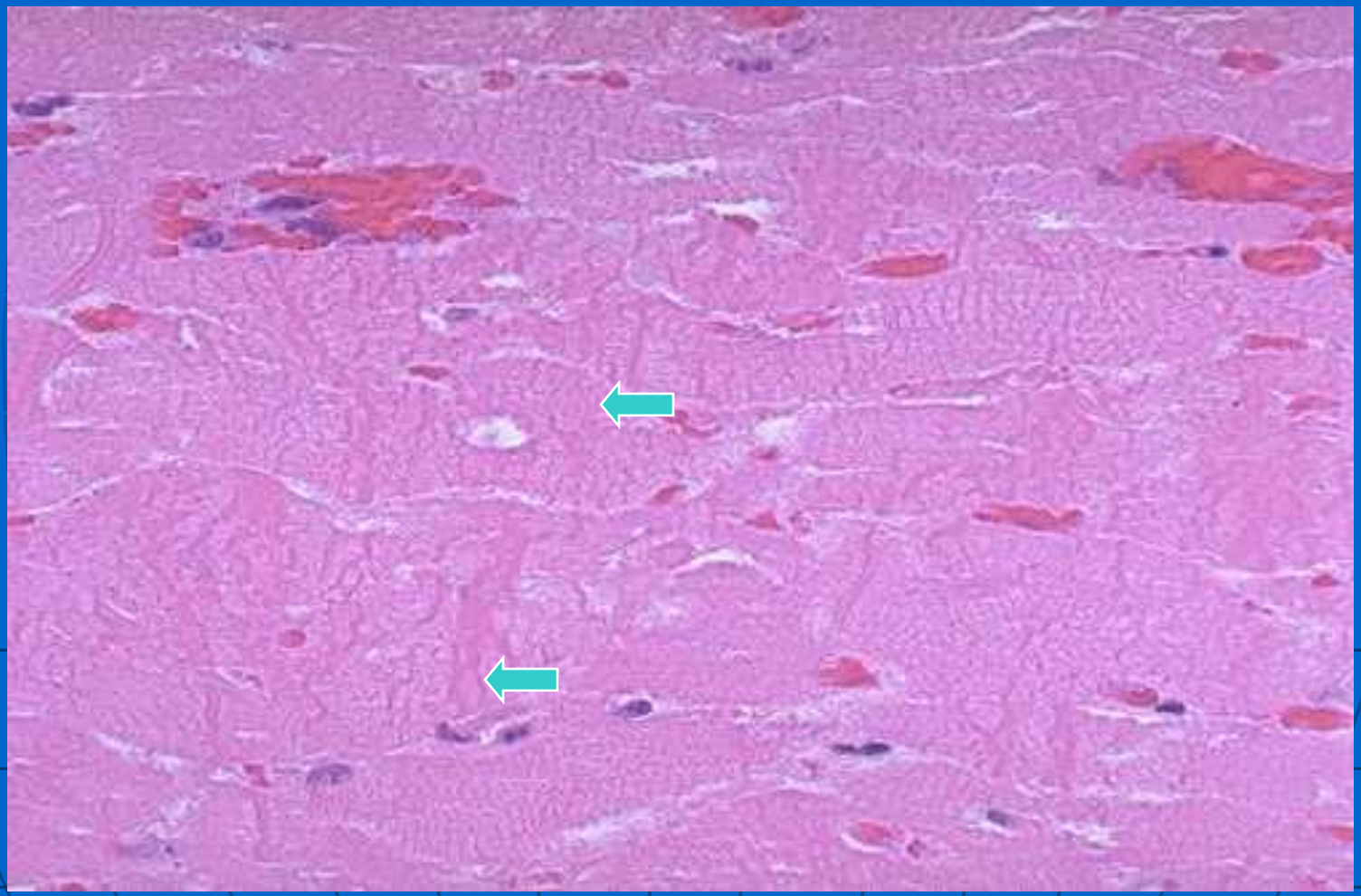
Microscopic appearance

- *4-12 hrs:* wavy fibres, myocytolysis (large vacuoles in cells)
- *12-24 hours:* infarcted muscle is brightly eosinophilic with intercellular oedema
- *1-3 days:* acute inflammation (polymorphs infiltrating dead muscle cells)
- *4-10 days:* removal of dead cells by macrophages, with the beginning of vascular granulation tissue formation
- *2-4 weeks:* repair-granulation tissue, becoming more fibrous and less vascular over time
- *4-6 weeks:* established scarring (collagen)

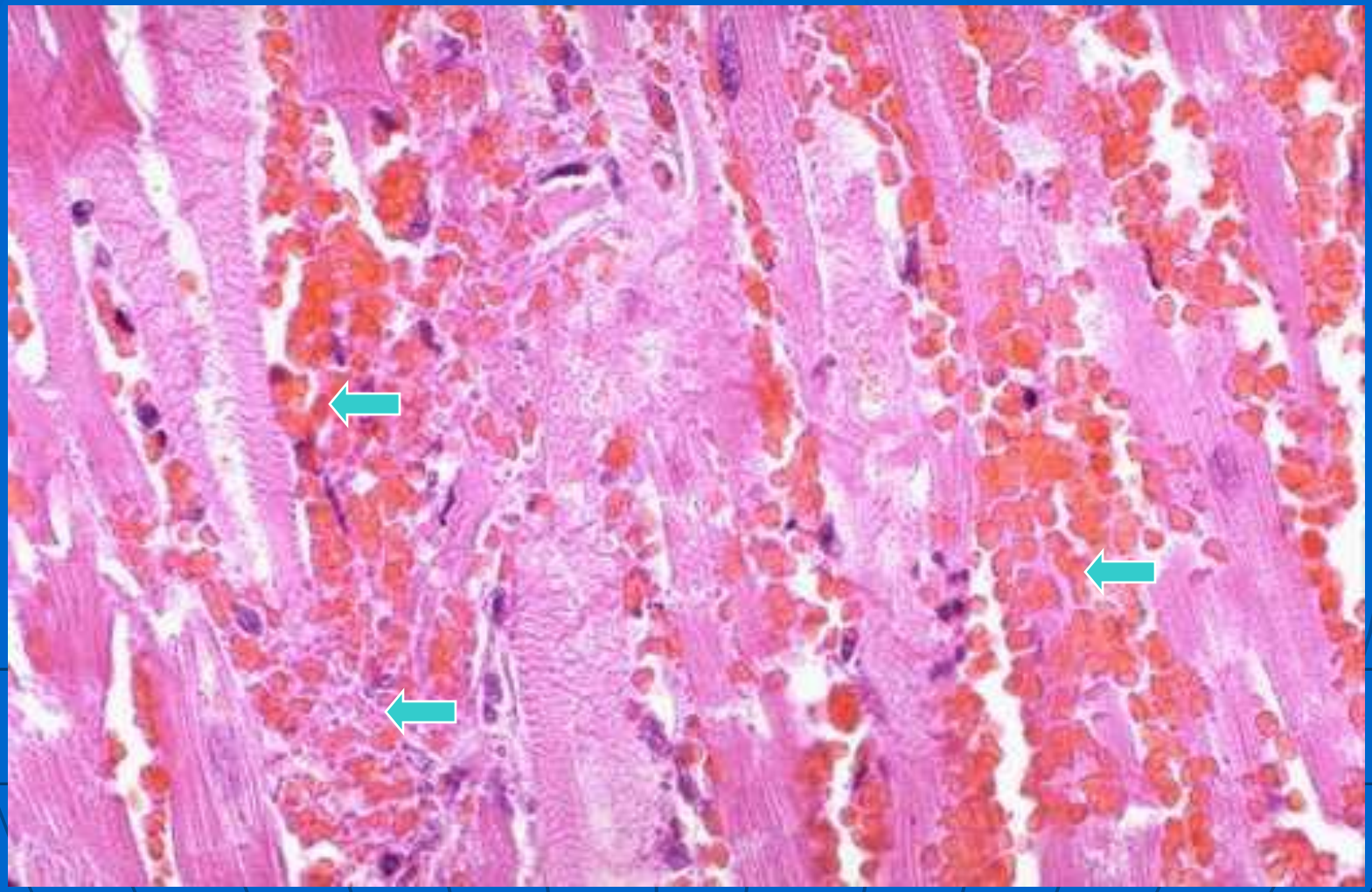
Normal Myocardium:



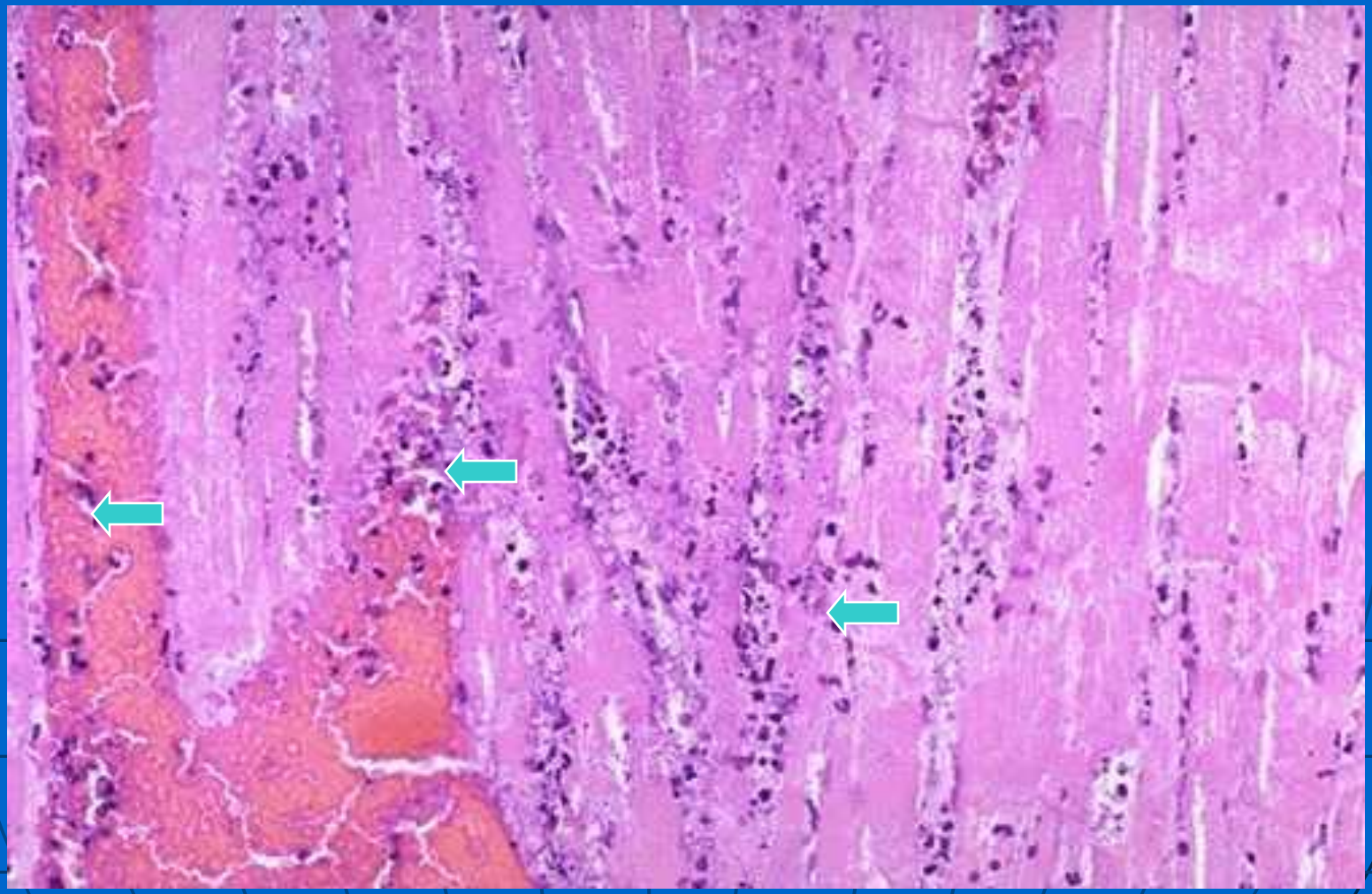
MI 18-24 hr loss of nucleus, contaction bands, coagulative necrosis.

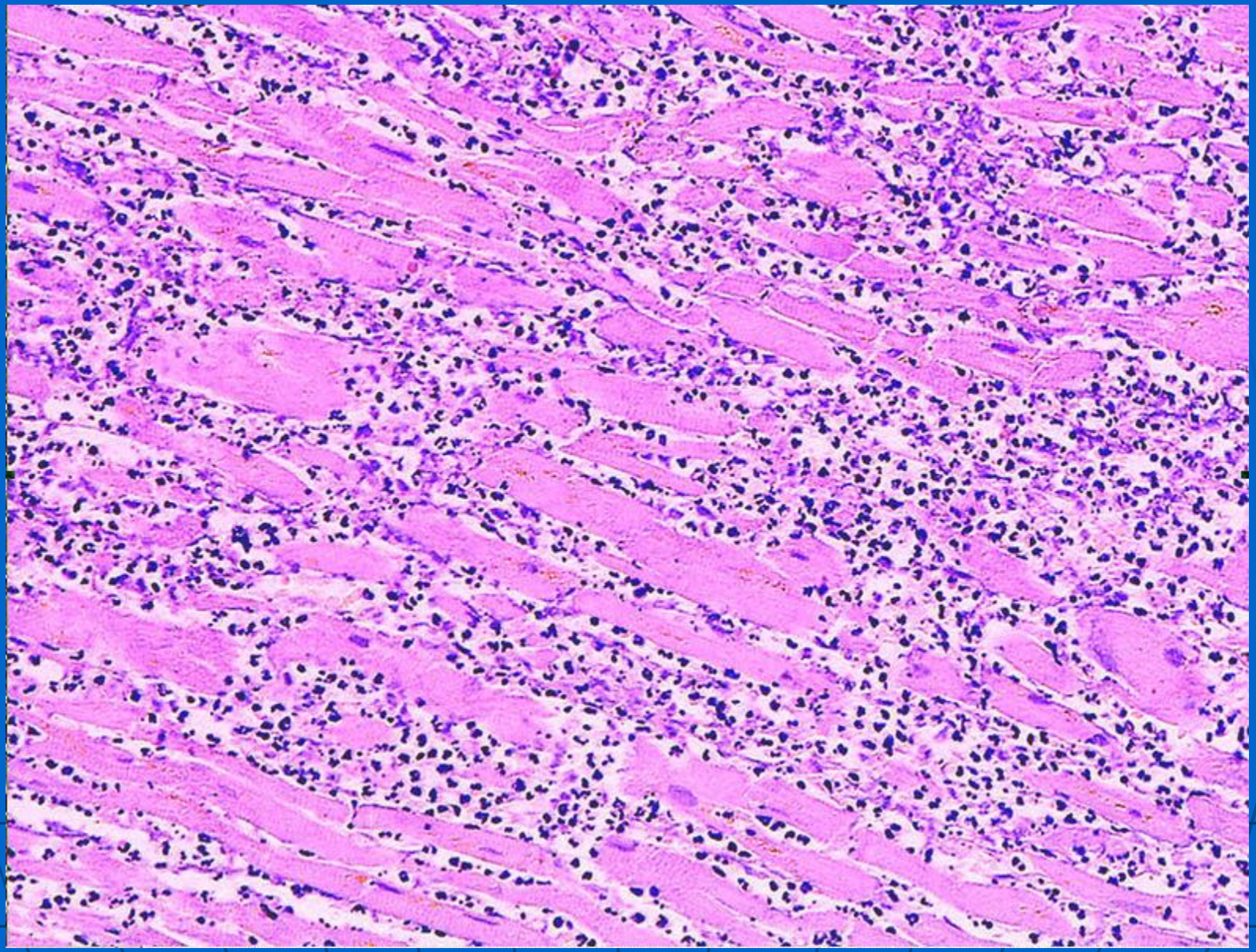


MI 3-4 day – Hemorrhage, inflammation.



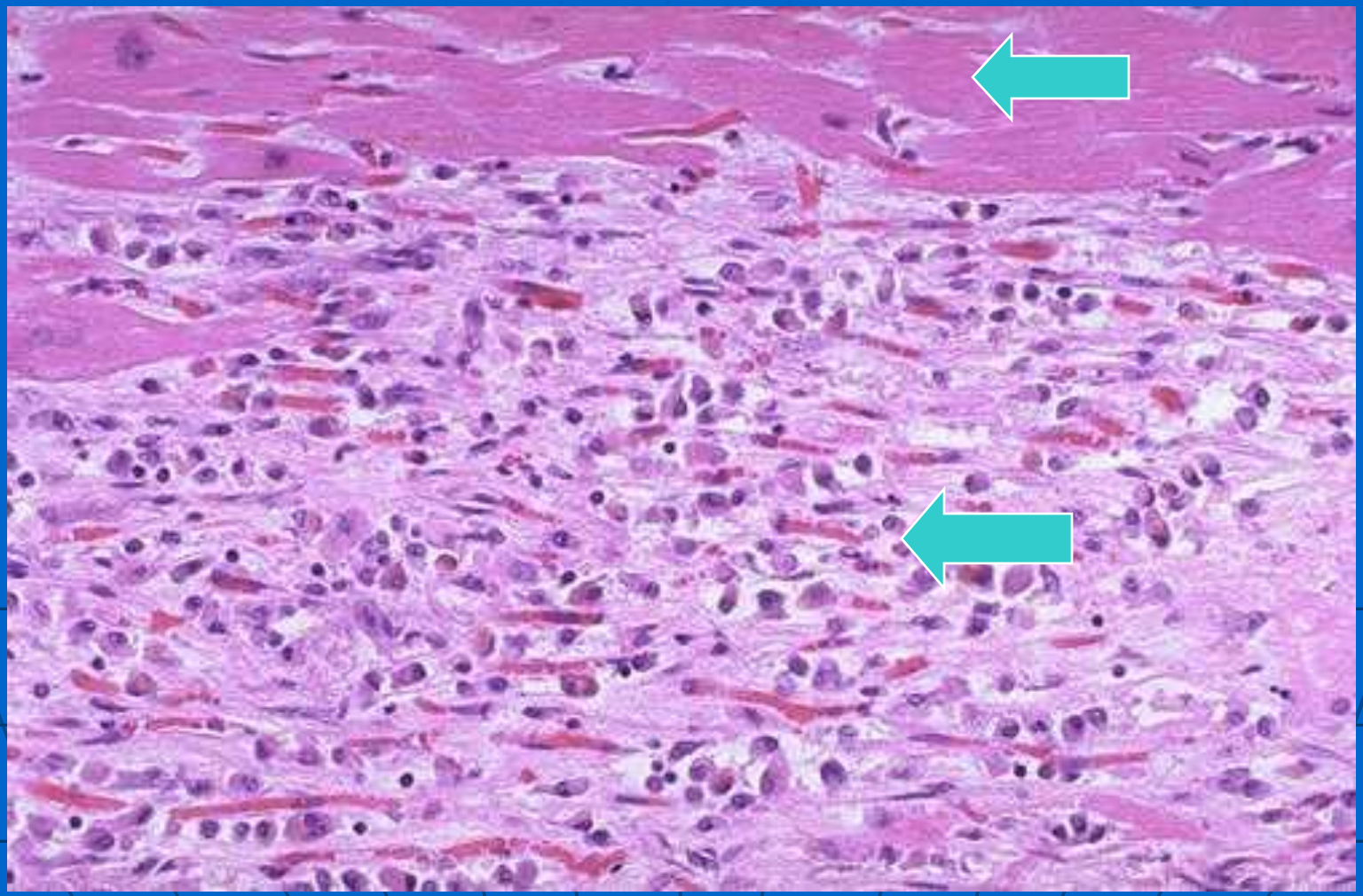
MI 1 w – PNC, macrophages



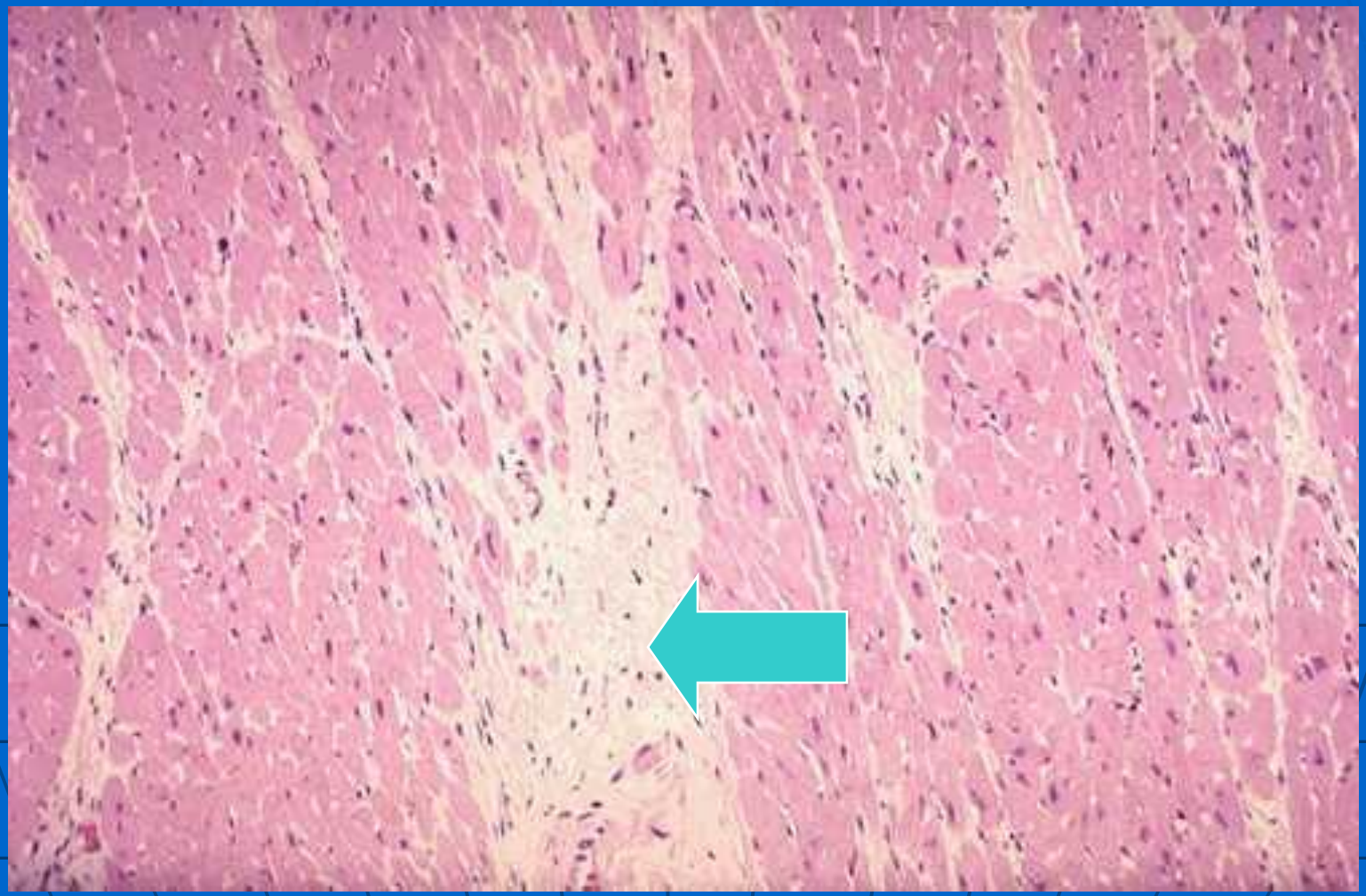


MI 1 w – PNC, macrophages

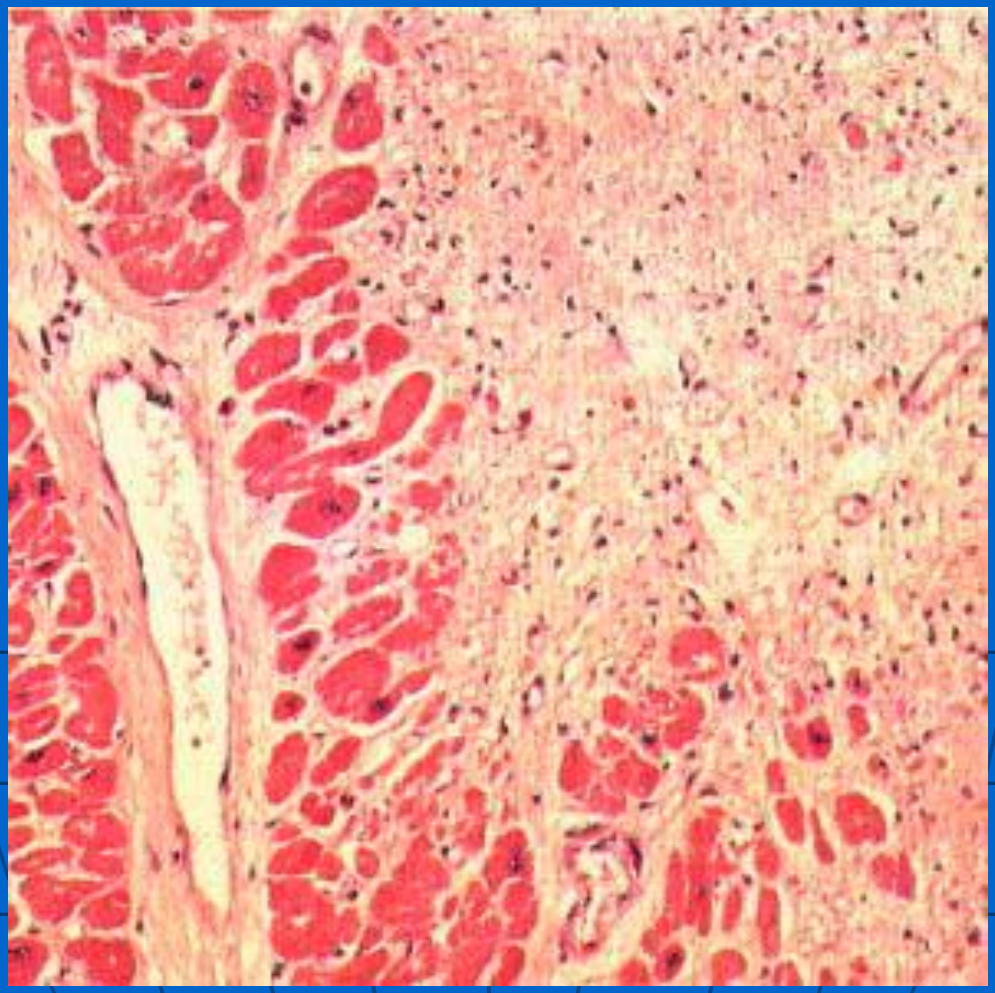
MI 2-4 W - Resorption, fibrosis



Collagen Scar after MI >4-6 W



Scar after MI >8 W



Complications of MI

- sudden death (ventricular fibrillation, tamponade, septal rupture)
  - within 1-2 hours (20% of patients)
- cardiac arrhythmias (80%) - within 2 weeks
  - *patients with old infarcts tend to develop cardiac arrhythmias arising from muscle adjacent to an area of old scarring*
  - *acute myocardial iscaehmia also precipitates arrhytmias*
  - *death is often due to ventricular fibrillation*
- left ventricular congestive failure, pulmonary oedema (60%) - within 2 weeks
- cardiogenic shock (15%) - within 2 weeks
  - monitoring in coronary care unit

Arrythmias

- An irregularity in heart rhythm
- Tachycardia – racing heart in the absence of exercise or anxiety
- Bradycardia – abnormally slow heartbeat
- Fibrillation – heart beat is sporadic, quivering pattern

Complications of MI

- rupture of cardiac muscle (1-5%) - within 3-8 days, particularly during early organisation and sofeting
  - can lead to:
    - tamponade (rupture through anterior, posterior or lateral walls) *(i.e. blood bursts through wall, instantly causing haemopericardium; this sudden rise in intrapericardial cavity pressure prevents cardiac filling, and leads to sudden death)*
    - left to right shunts (rupture through medial (septal) wall)
    - acute mitral regurgitation
- thromboembolism (15-40%) - within 2 weeks
  - *mural thrombus formation on the inflamed endocardium over the area of infarction may occur*
  - *fragments can break off and embolise to various organs (part. brain, spleen, kidney, gut, lower linbs) producing infarction*

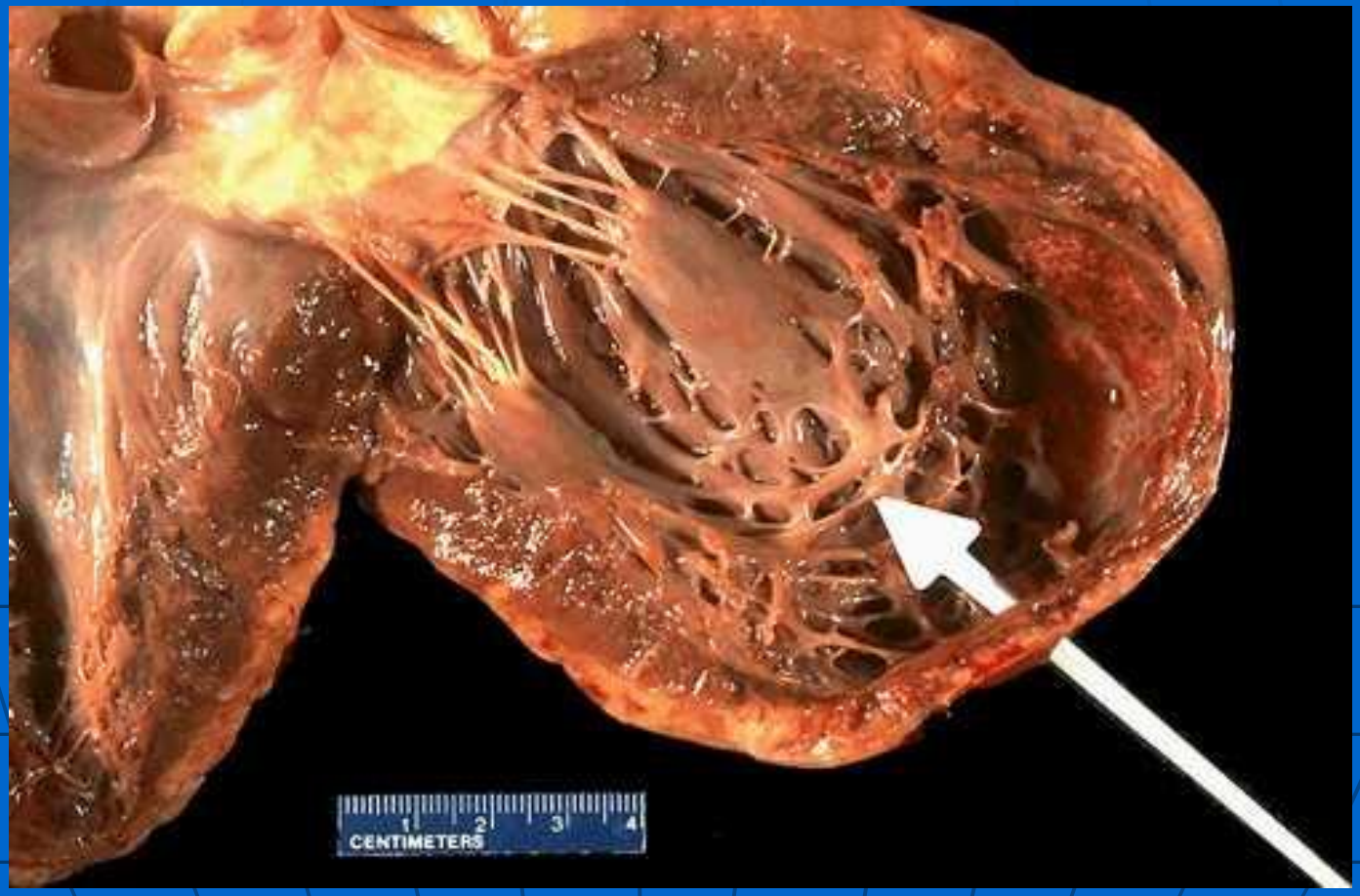
Complications of MI

- fibrinous pericarditis
  - day 2-3
    - a response to underlying infarct
  - *as a long term complication*
    - *Dressler's syndrome is a form of immune-mediate pericarditis associated with a high ESR*
    - *it develops in a very small number of cases after infarction (2-10 months after MI)*
- infarct extension and reccurrent myocardial infarction - long term complication
  - new necrosis adjacent to an existing infarct
- infarct stretching - long term complication
  - thinning of fibrotic myocardium occurs as organisation and repair are undertaken
  - the collagenous scar is weak and inelastic, and may stretch
  - eventually a ventricular aneurysm will become evident and slowly progress
  - it paradoxically bulges during systole
  - overlying thrombosis may occur in this area because of haemostasis (but embolic complications are uncommon)
- chronic intractable left heart failure due to inadequate ventricular pumping action - long term

Myocardial Infarction

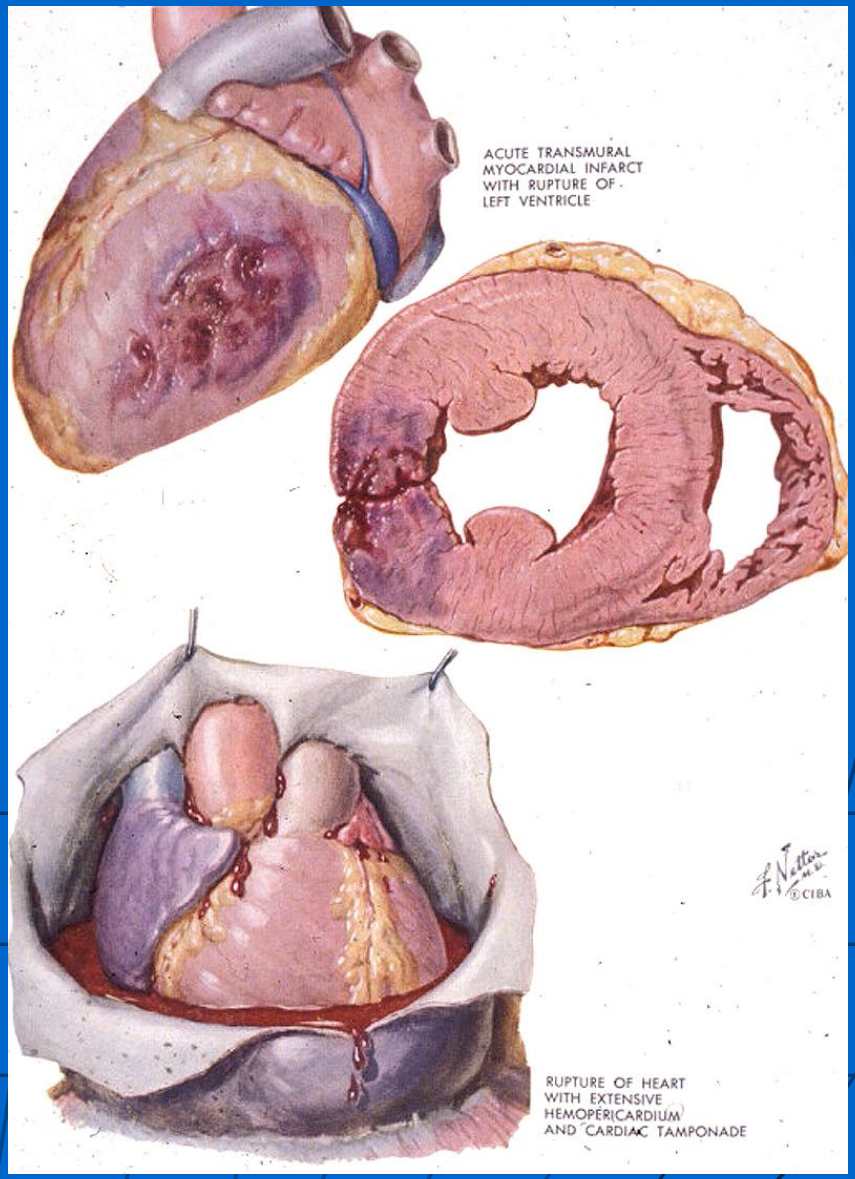
- Complications of MI
  - Cardiogenic heart failure
    - Sudden loss of pumping strength.
  - Arrhythmias
    - Irritable conduction system.
  - Valvular dysfunction
    - involvement of papillary muscle
  - Rupture and tamponade

MI - Rupture



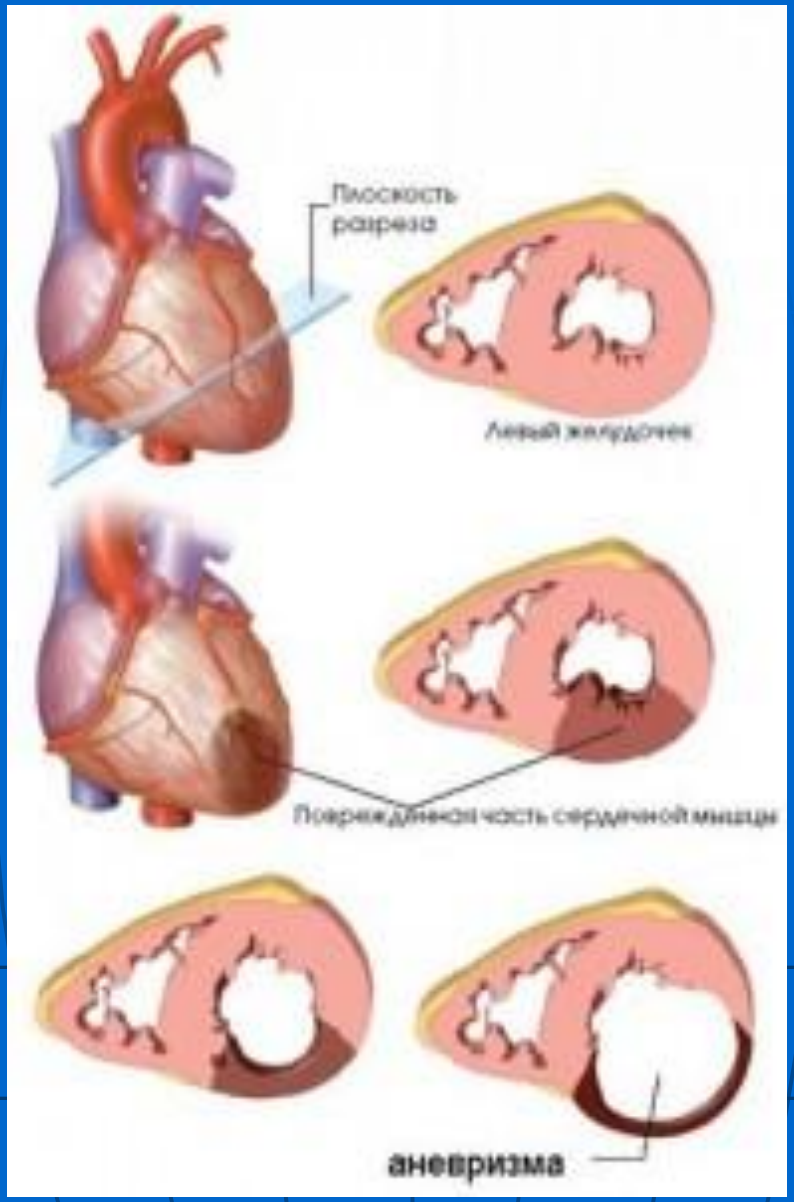
Hemopericardium

- Ruptured or perforated heart
  - MI
  - Penetrating wounds
- Lacerated aortic root
  - Auto accident
  - Rotatory motion unscrews heart from aorta



Heart Failure

- Diminished out volume of either ventricle.
  - Systolic failure
    - Loss of pumping strength.
    - Backup of blood behind weakened ventricle.
    - Atherosclerosis leading to chronic ischemia.
  - Diastolic failure
    - Reduced ability of ventricle to fill.
    - Constriction of trapping of ventricle

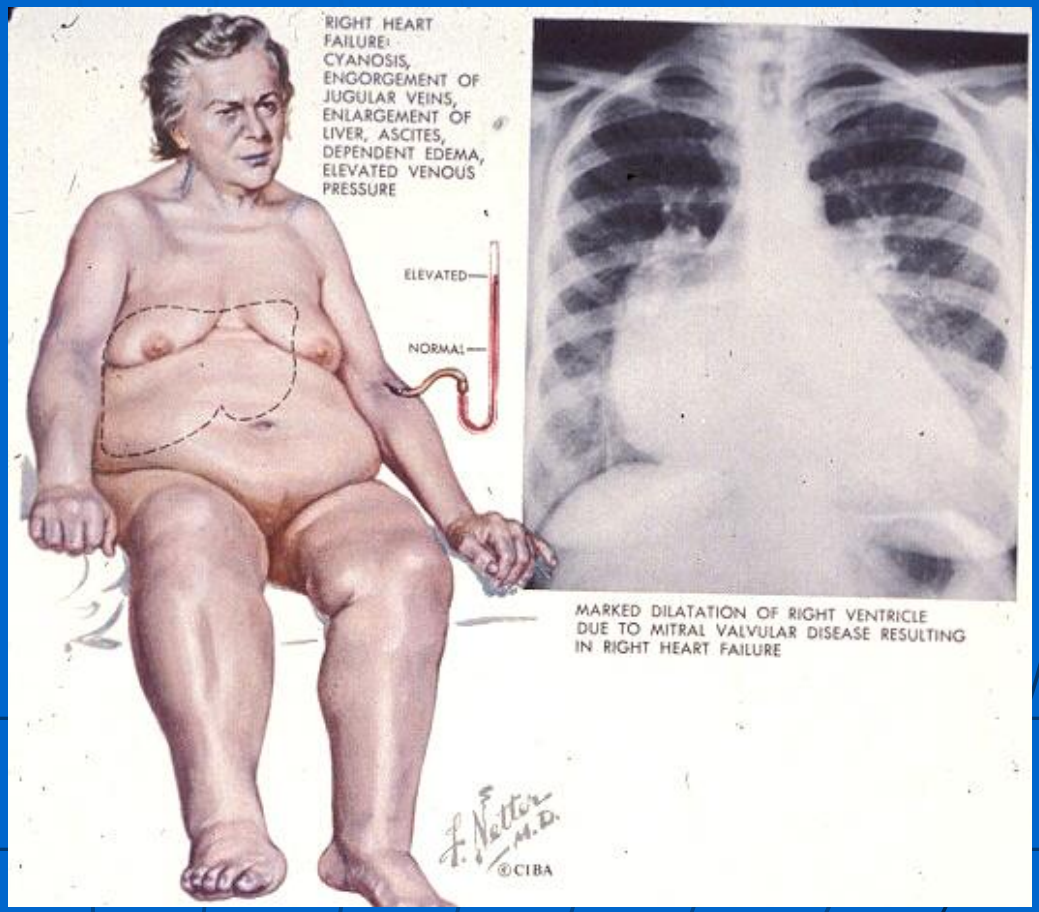

Плоскость разреза
Левый желудочек
Поврежденная часть сердечной мышцы
аневризма

Congestive Heart Failure (CHF)

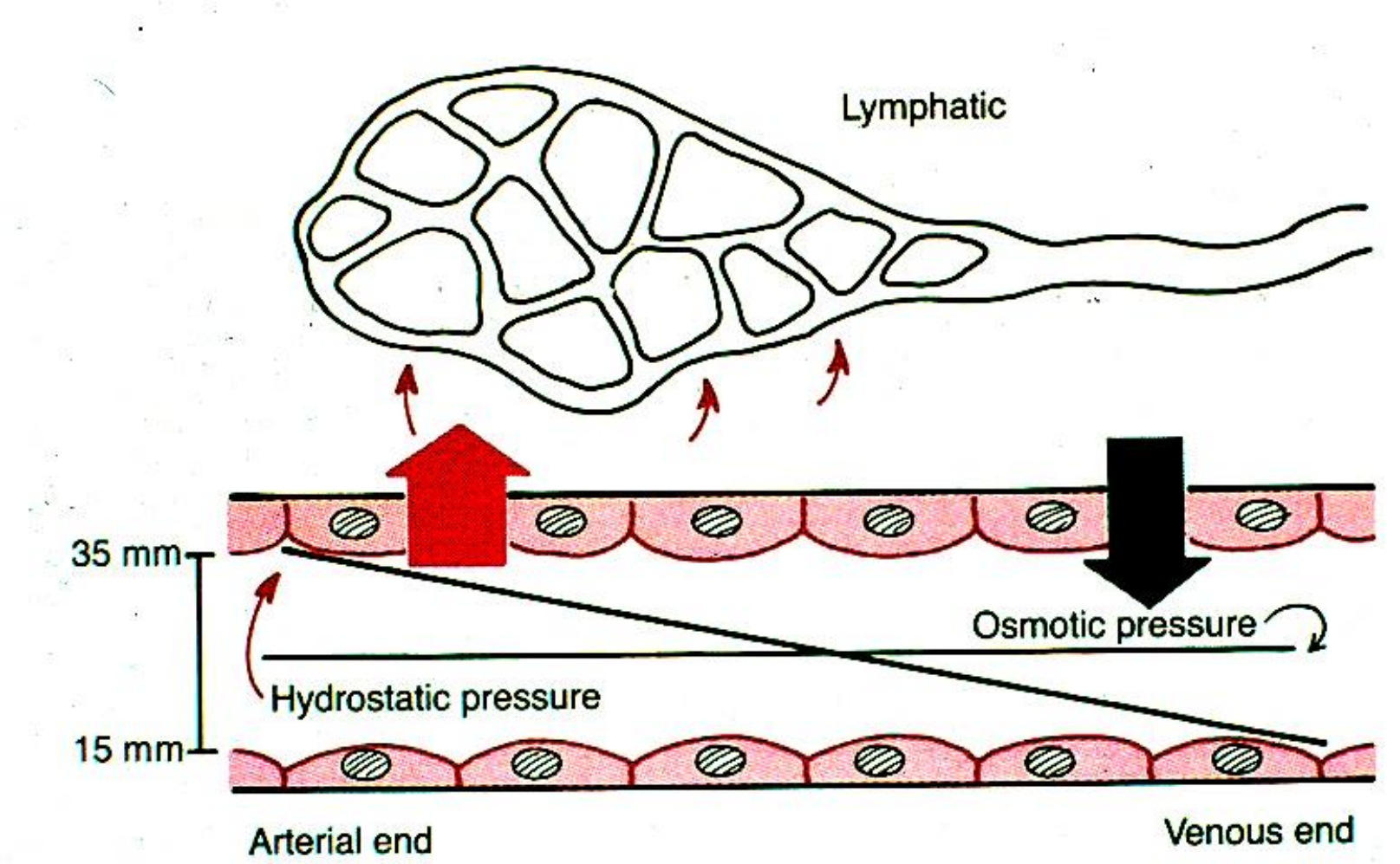
- Damaged or overworked heart muscle is unable to keep blood circulating normally
- Affects over 5 million Americans
- Damage to heart muscle may result from: rheumatic fever, pneumonia, heart attack, or other cardiovascular problem
- Lack of proper circulation may allow blood to accumulate in the vessels of the legs, ankles, or lungs
- Diuretics relieve fluid accumulation

Congestive Heart Failure

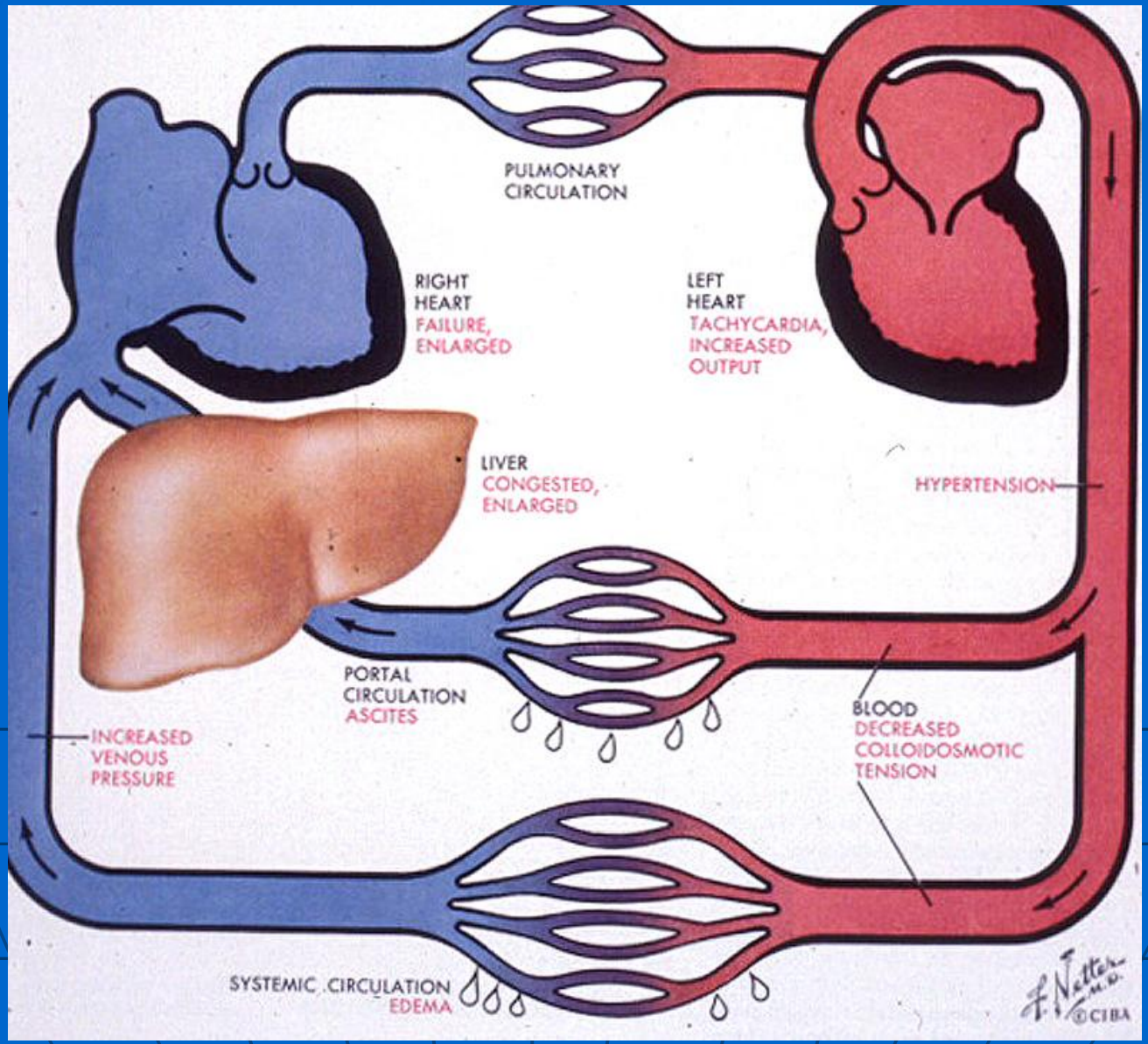
- Diminished of pumping ability of left ventricle.
- Back up of blood in pulmonary vasculature.
- Pulmonary edema
- Peripheral edema



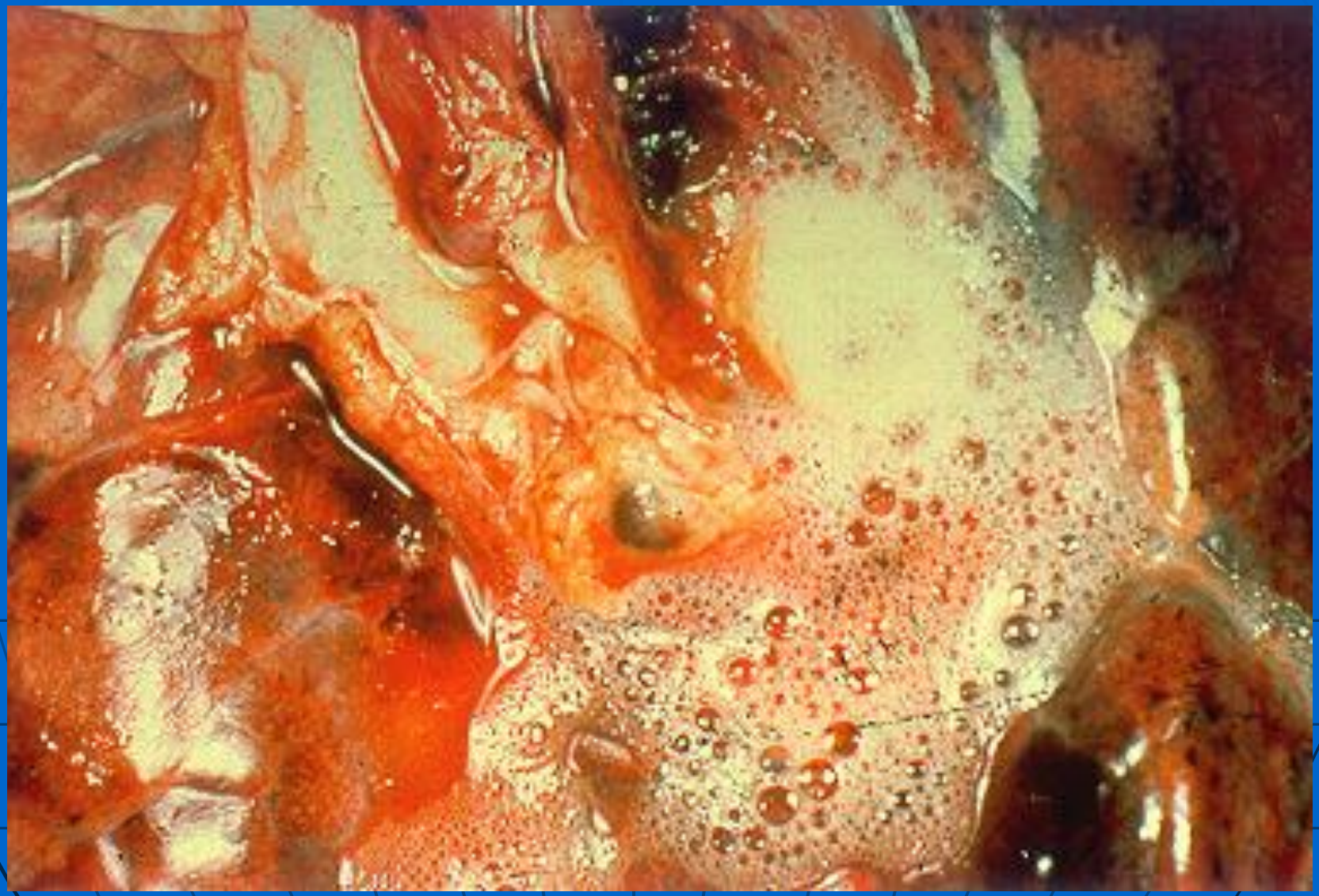
Back Pressure



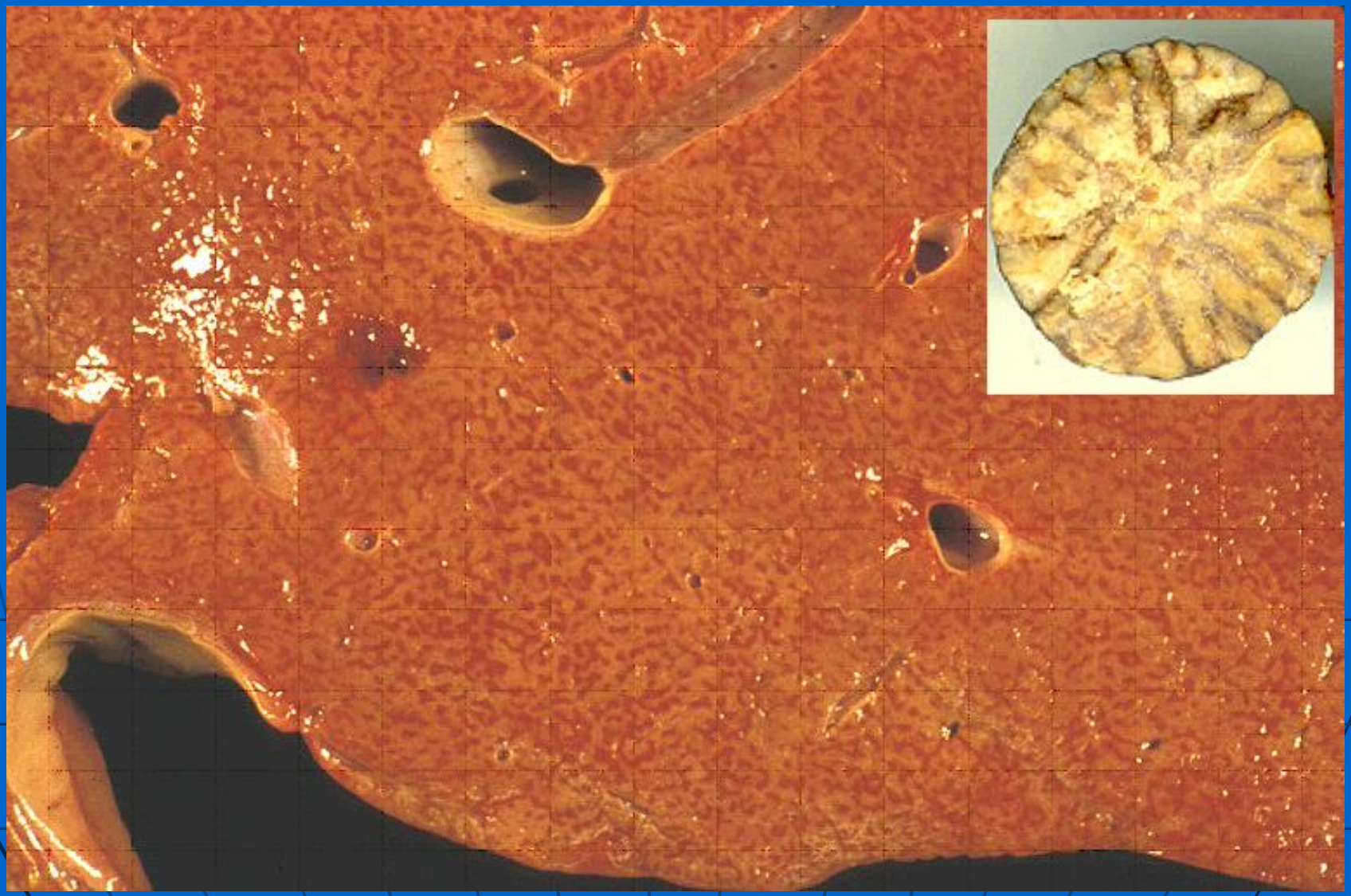
CHF and Edema Formation



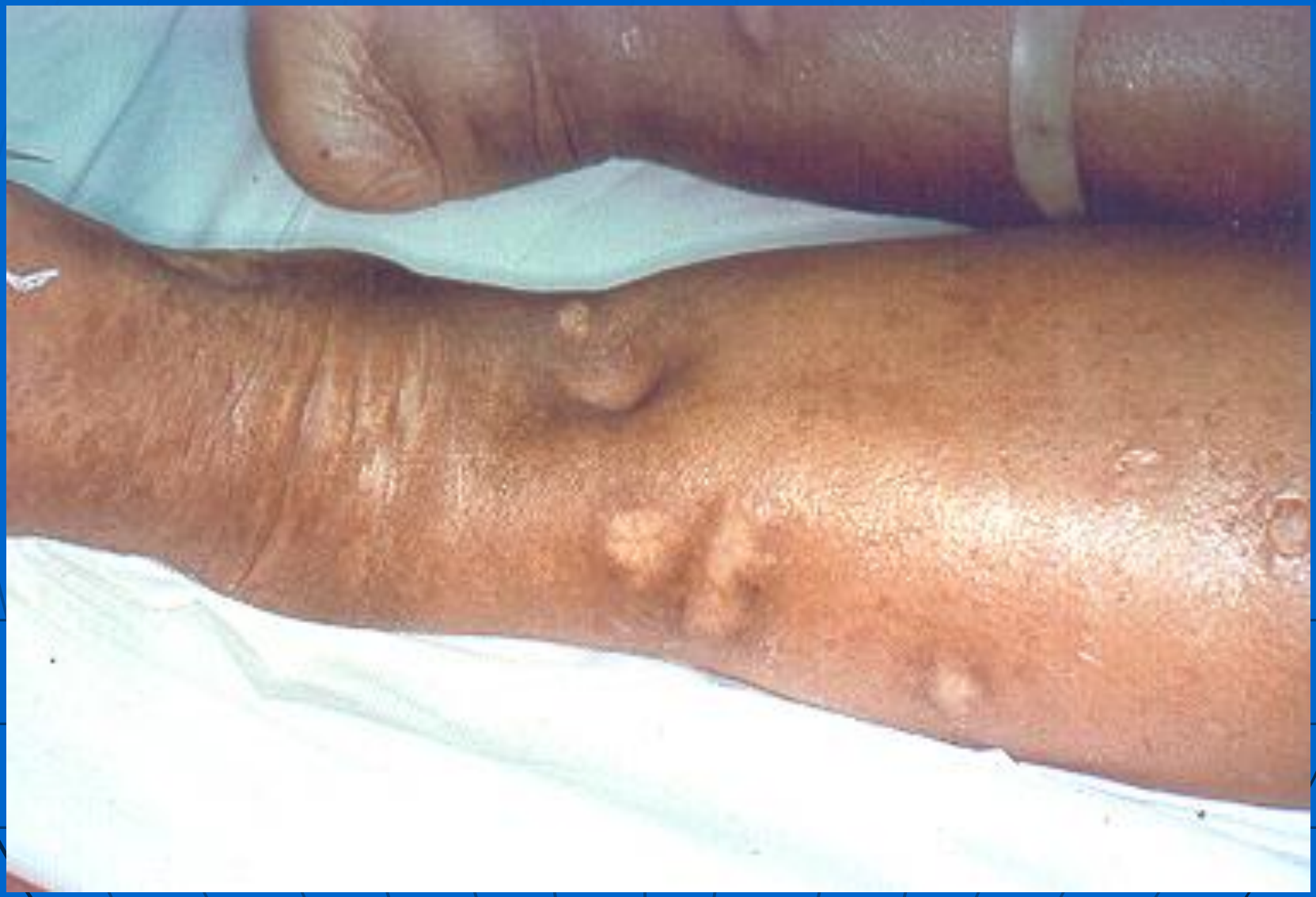
Pulmonary Edema



Liver Chronic Passive Congestion



Pitting Edema



Laboratory Diagnosis

- LDH - 1-5 (1 - 2 flip)
- CK (Creatine kinase)- Isoenzymes (Fractions)
  - MM - Muscles
  - MB (creatine kinase myocardial band) - Cardiac muscle.
  - BB - Brain
- Troponins

Management:

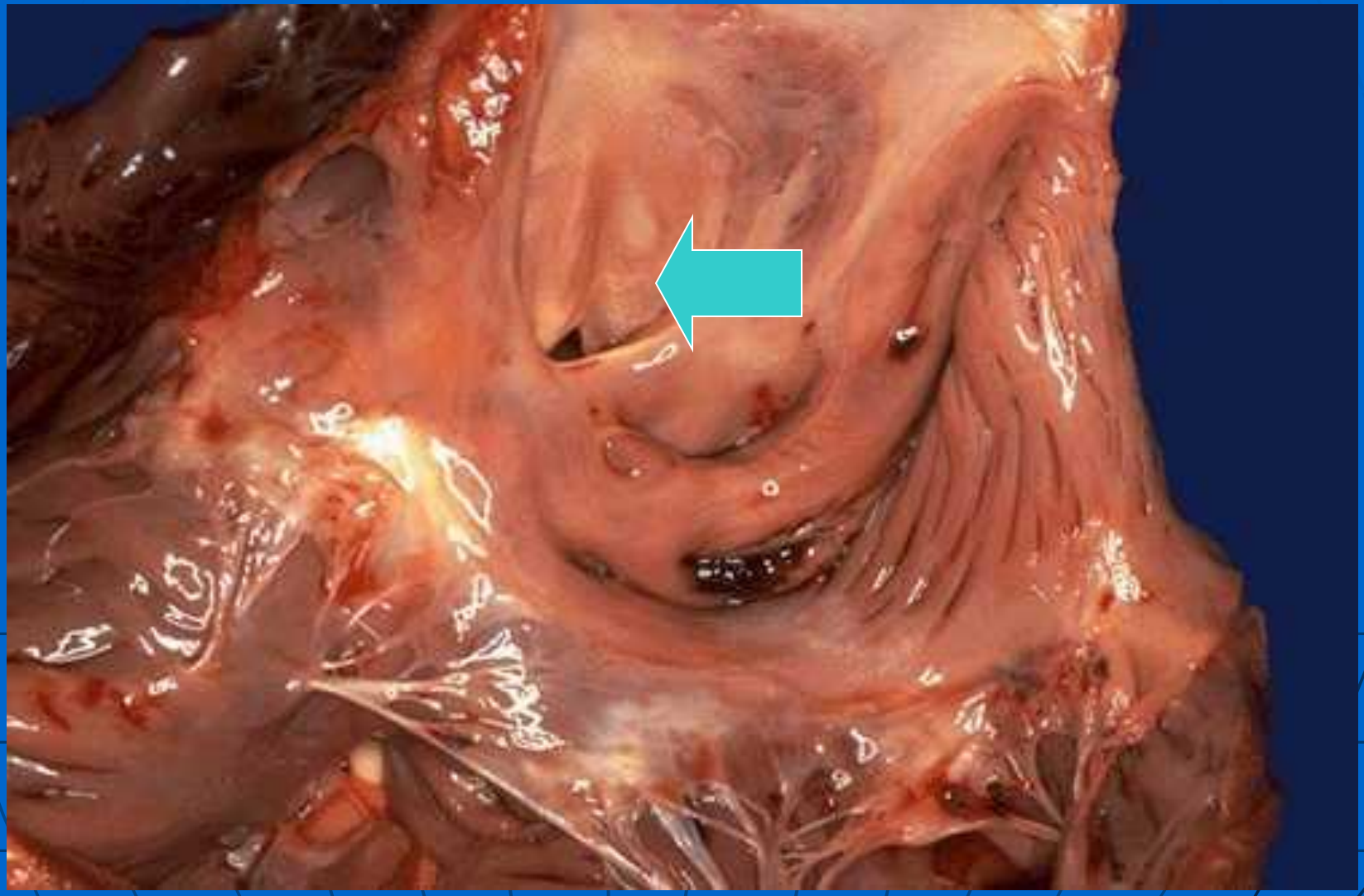
- Aimed to prevent complications.

1. Rest & sedation*
2. Supportive mesures
3. Thrombolytic agents - Streptokinase

Congenital Heart Diseases:

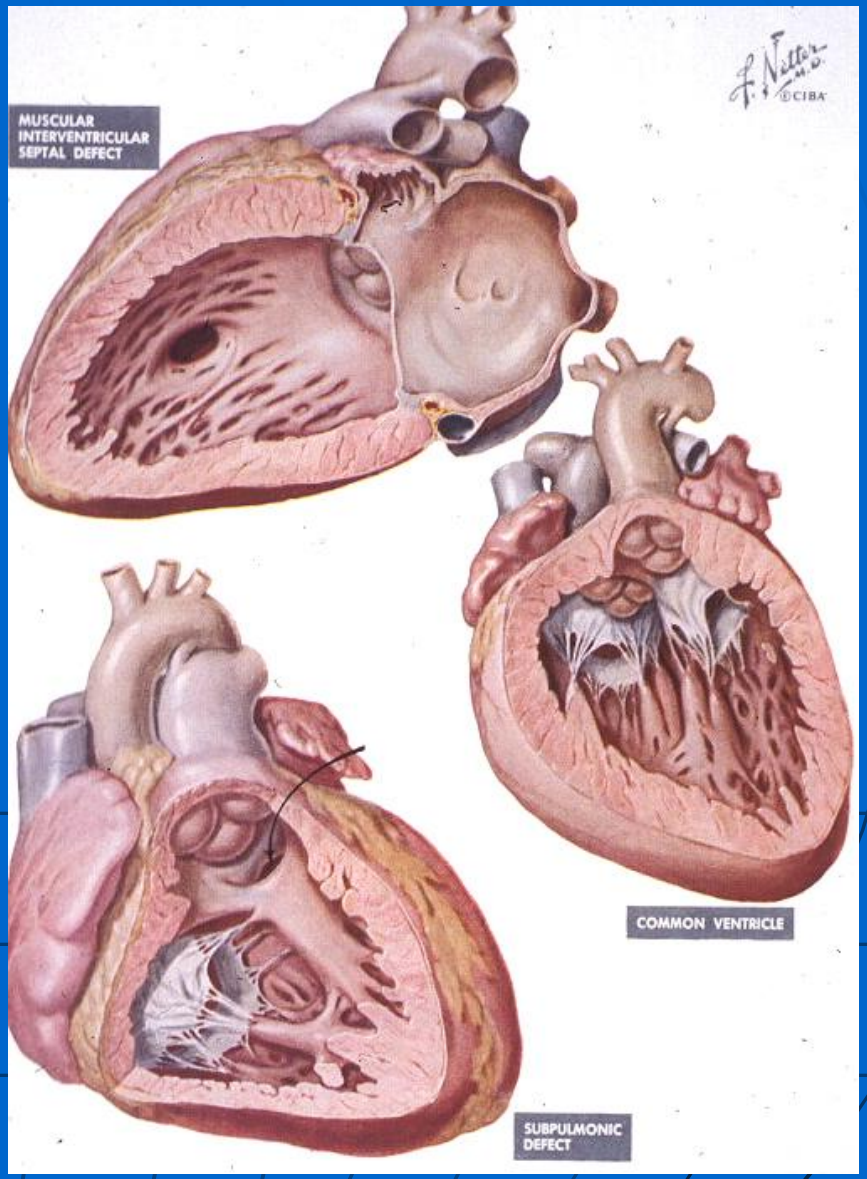
- **Left-to-Right shunts.**
  - Atrial Septal Defect (ASD)
  - Ventricular Septal Defect (VSD)
  - Patent Ductus Arteriosus (PDA)
- **Right-to-Left shunts**
  - Transposition of Great Arteries
- **Obstructions**
  - Coarctation of Aorta
- **Others.**

Atrial Septal Defect (ASD)

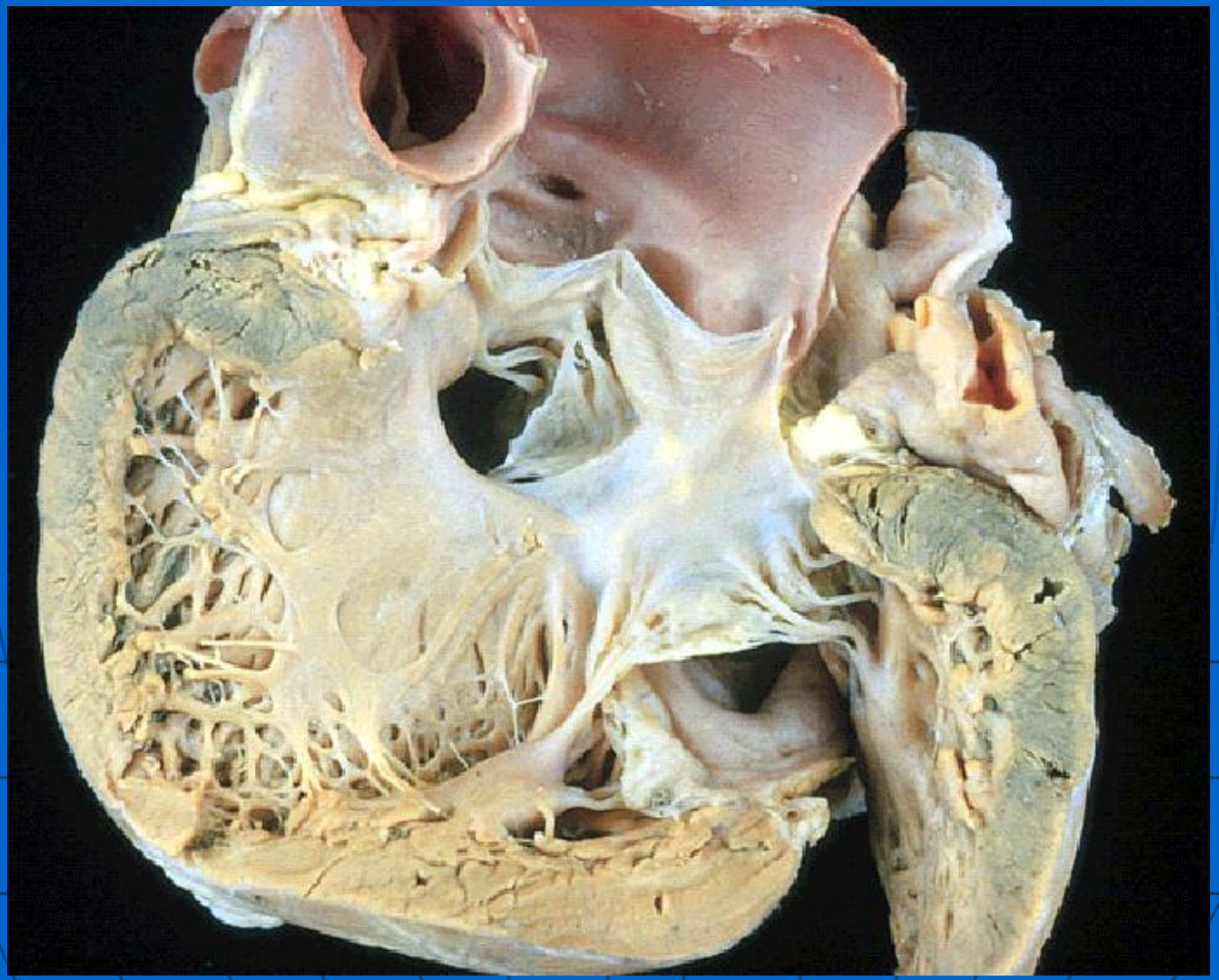


Ventricular Septal Defect

- Left to right shunt
- Depending on size will lead to Eisenminger reaction.
- Later becomes right to left shunt.
- Possible infections.

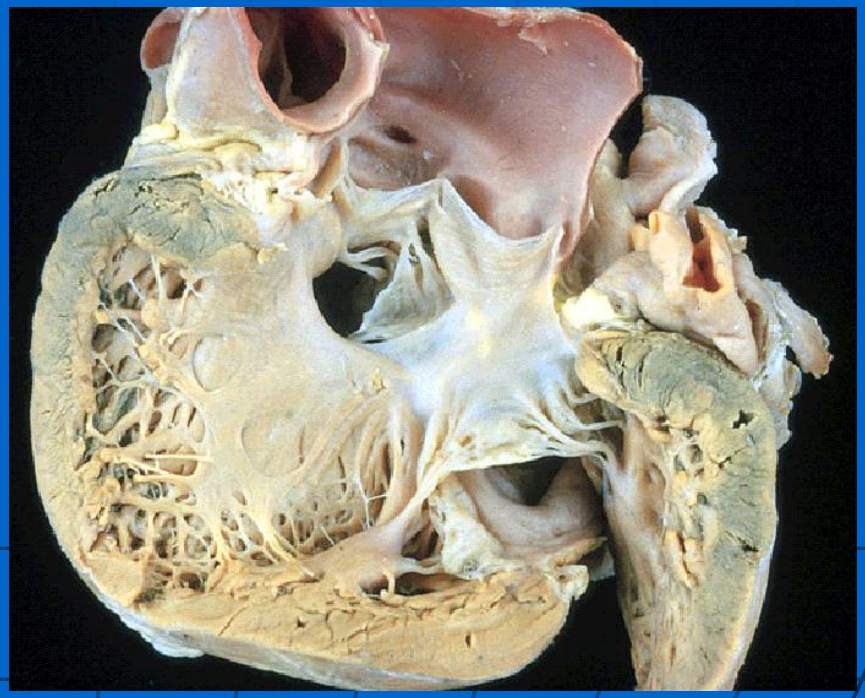


Ventricular Septal Defect



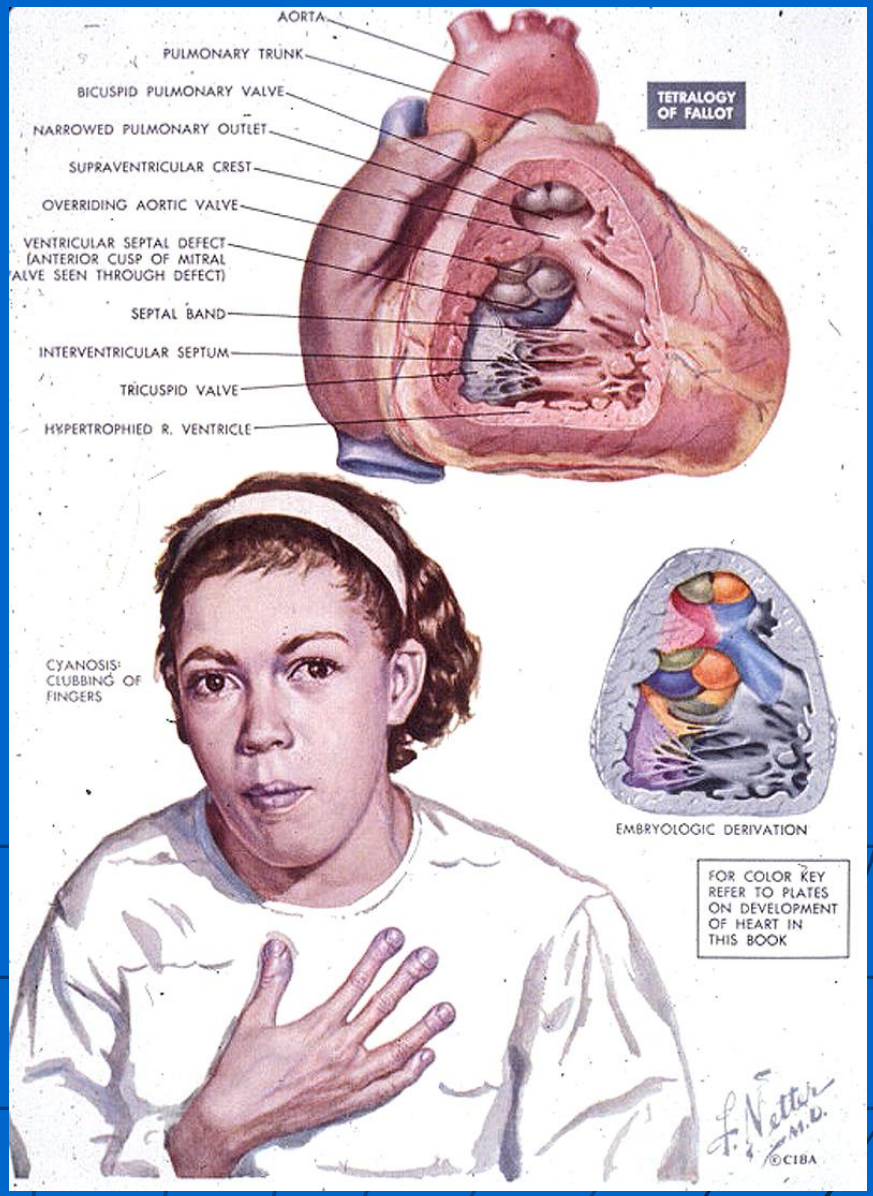
Ventricular Septal Defect

- Colorized Doppler
- Note jet at top of frame.
- Infection on downstream side.
- Eisenminger?



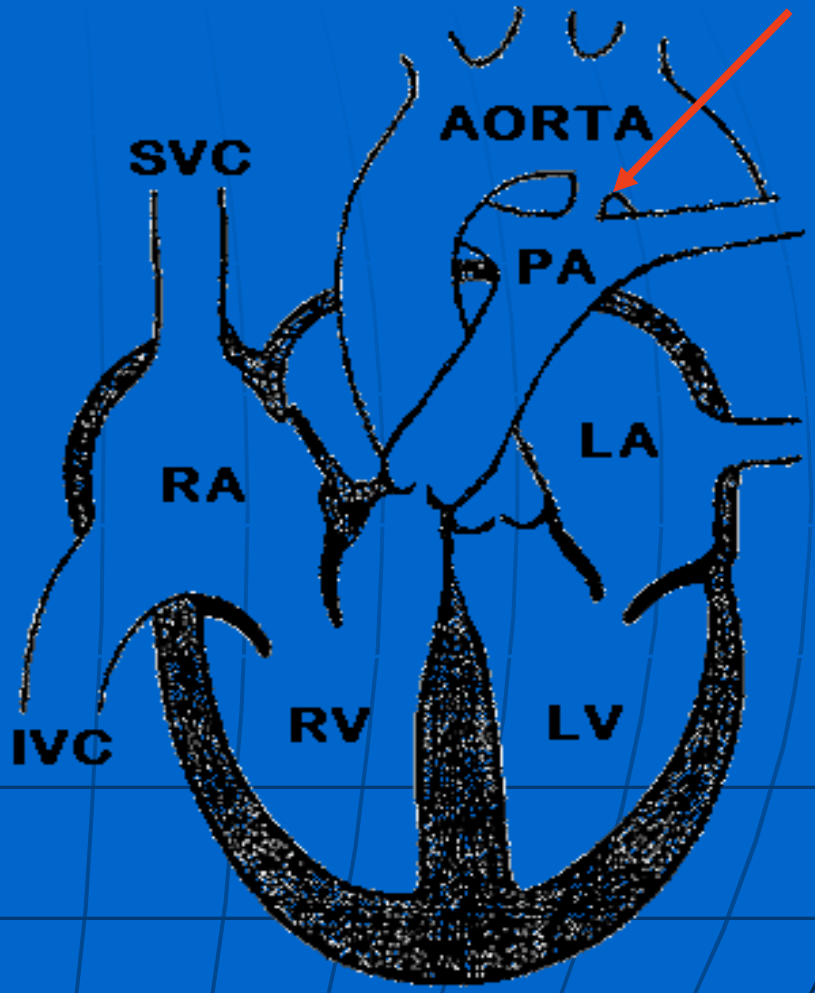
Tetralogy of Fallot

- Four problems
  - 3 defects
  - 1 compensatory
- VSD
- Narrowed pulmonary outflow tract
- Over-riding aorta
- RV hypertrophy



Patent Ductus Arteriosus (PDA)

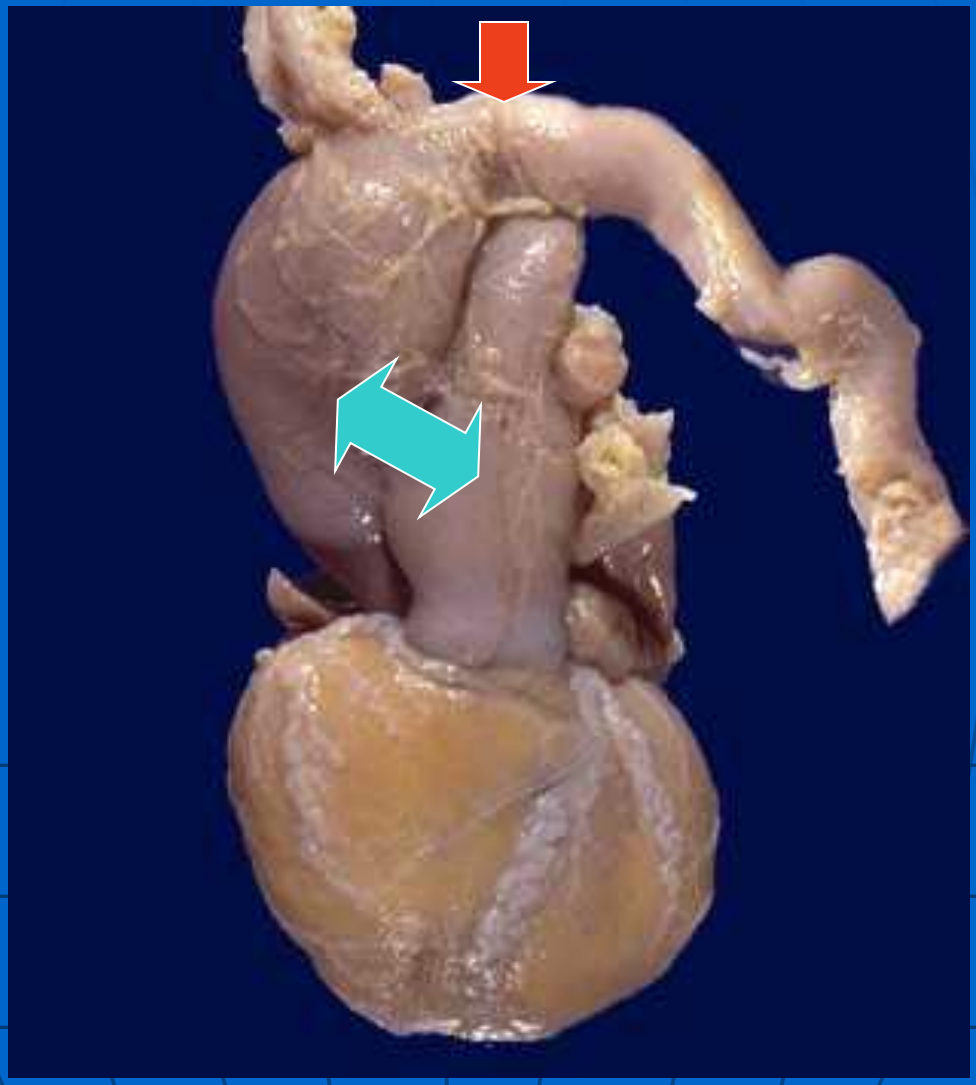
- The ductus arteriosus, serves to shunt blood from pulmonary artery to aorta during intrauterine life. Persistence of ductus, which normally closes soon after birth, results in left-to-right shunt.



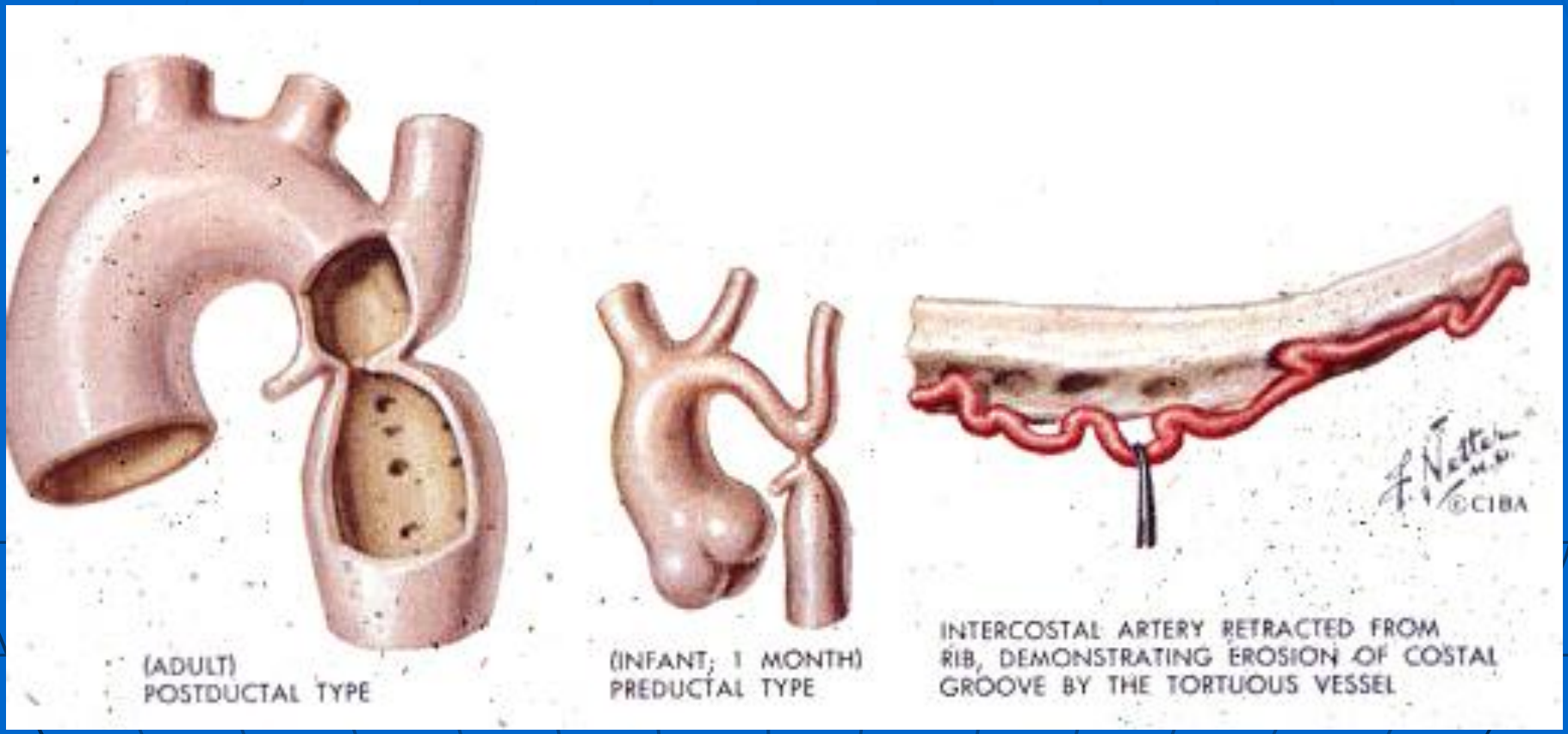
Coarctation of Aorta

- Coarctation of the aorta — or aortic coarctation — is a congenital condition, a narrowing of the aorta.

Coarctation of Aorta - Infant



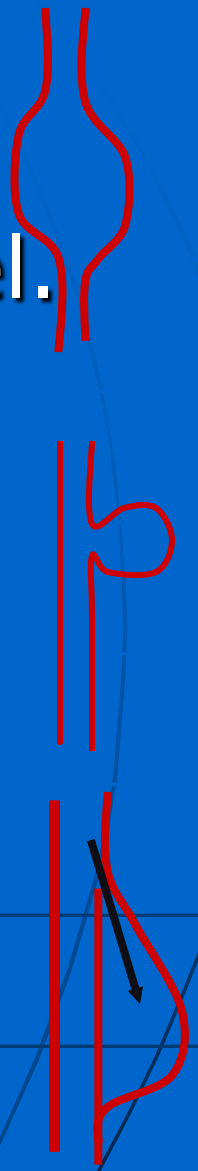
Coarctation of Aorta



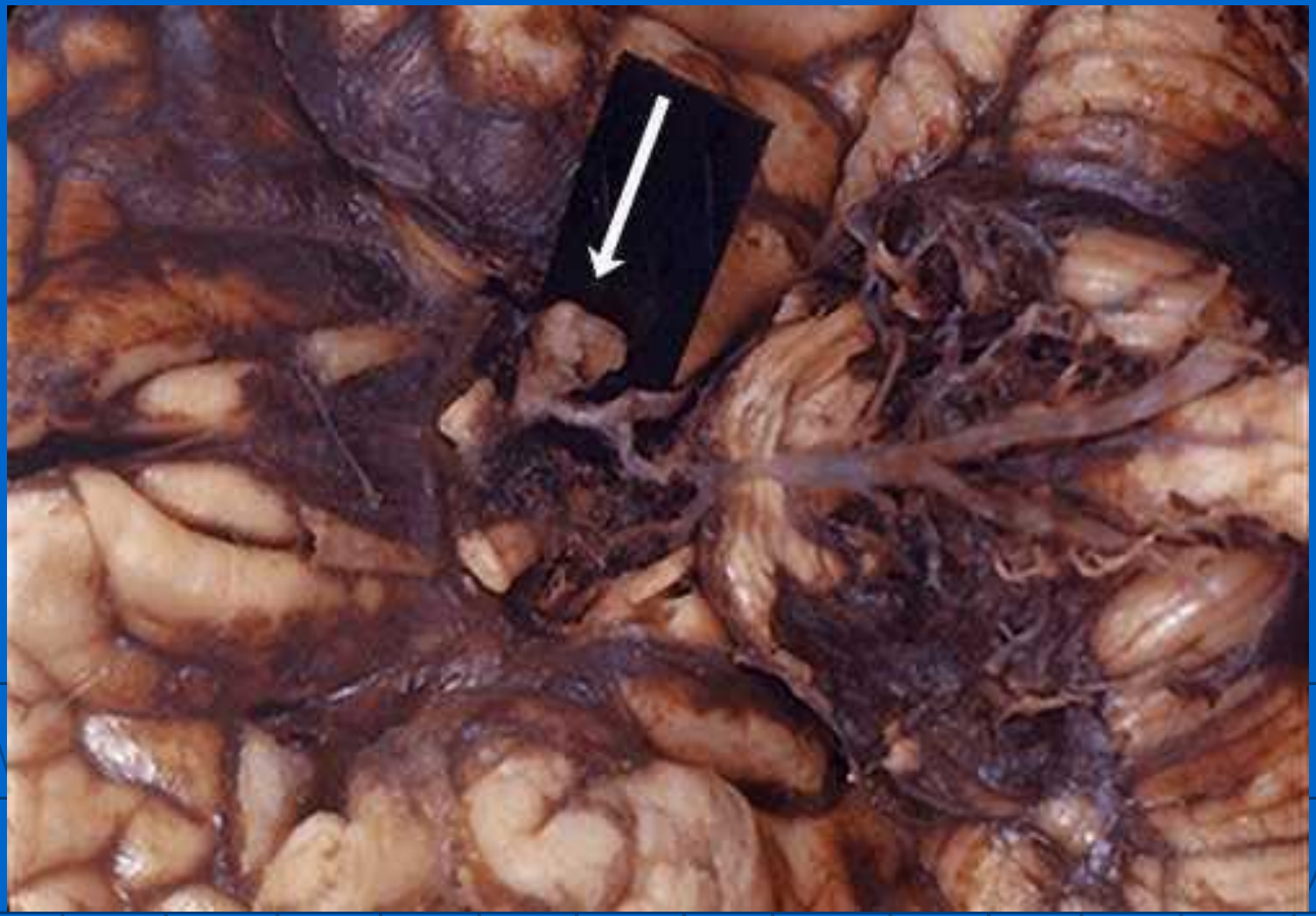
Aneurysms & Varicose Veins

Aneurysms:

- Abnormal dilatation of blood vessel.
- Fusiform, Saccular & dissecting.
- Atherosclerosis, Syphilitic & congenital.
- Berry aneurysms – Base of brain.
- Complications:
  - Thrombosis
  - Embolism
  - Rupture



Berry Aneurysm:



Berry Aneurysm:

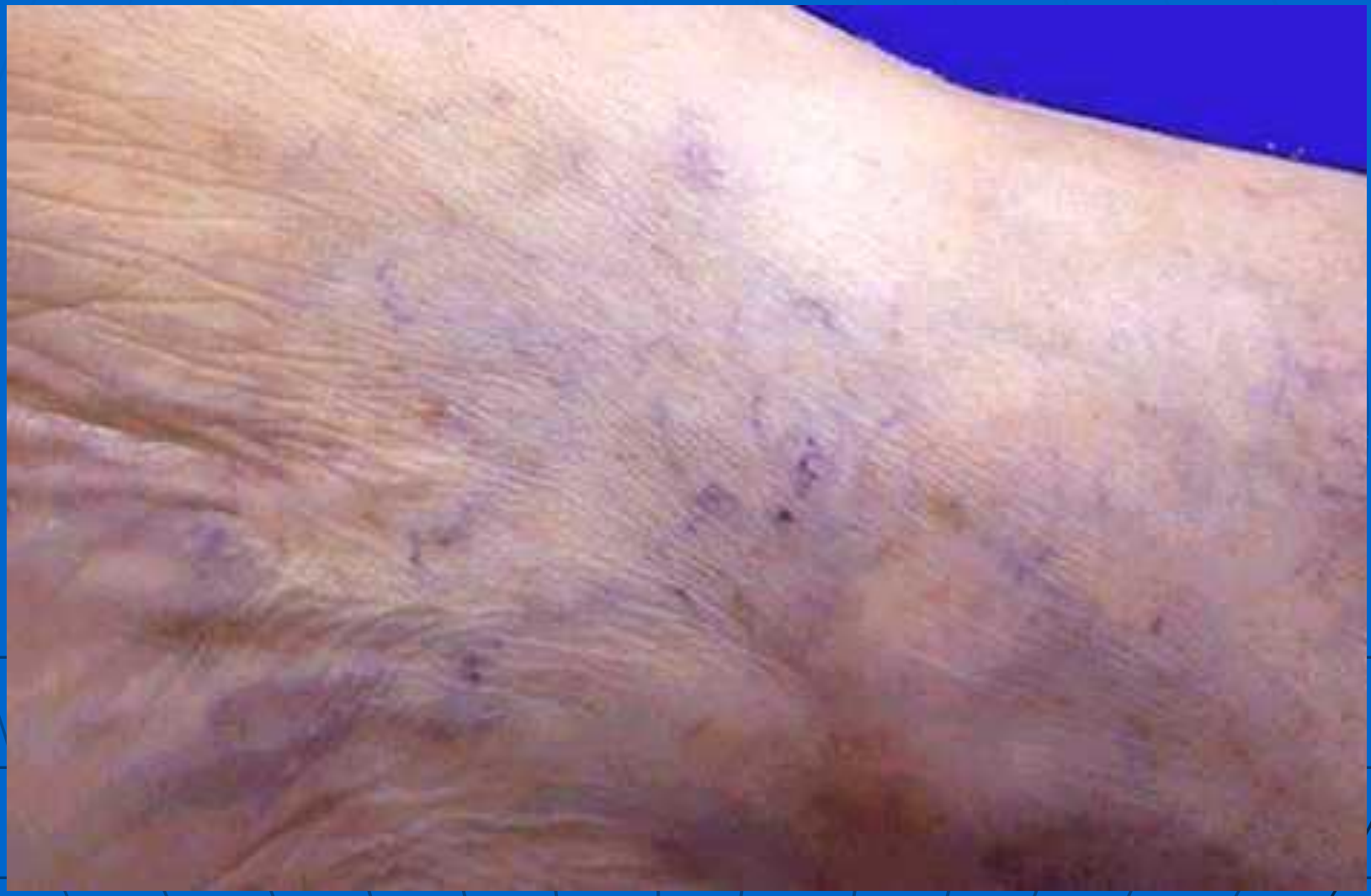
Complications of Aneurysms:

- Thrombosis
- Embolism
- Rupture

Varicose Veins:

- Abnormal diffuse dilatation of veins.
- Lower limbs- common
- Congenital or acquired
- Pathogenesis:
  - Damage to valves
  - Stagnation
  - Increased pressure → dilatation.
- Chronic ulcers.

Varicose Veins:



Thank You…