



**Hemodynamic disorders part (I)**

### ***I. Microspecimens:***

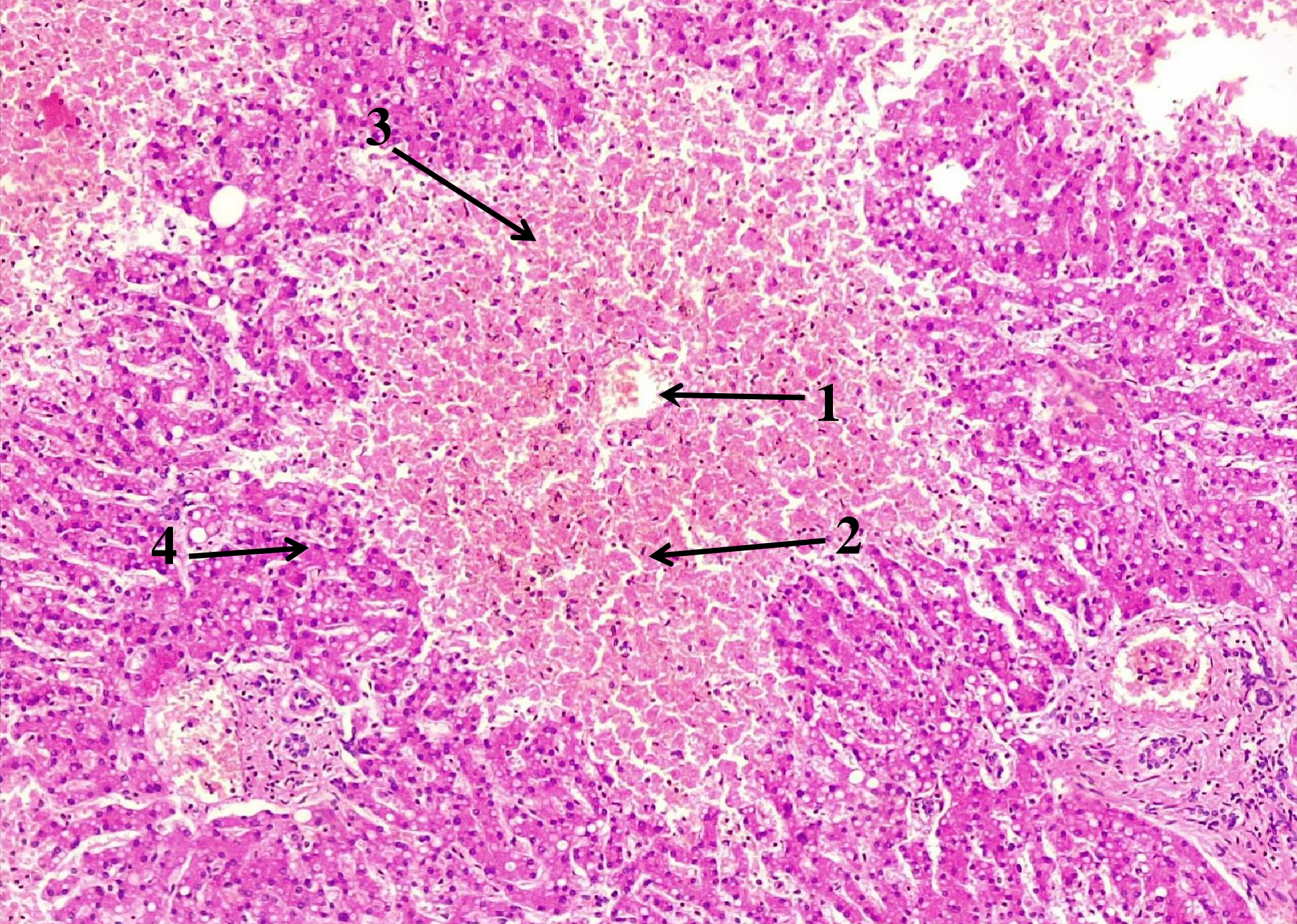
**№ 10. Chronic hepatic congestion (*nutmeg liver*). (*H-E. stain*).**

#### **Indications:**

1. Dilated and congested centrilobular vein.
2. Dilated and congested sinusoidal capillaries.
3. Atrophied hepatocytes in the center of lobule.
4. Unchanged hepatocytes and sinusoidal capillaries at the periphery of the lobule.

In the center of the hepatic lobules the trabecular structure is erased, there are hemorrhages ("blood lakes") around central veins, veins are dilated and congested, the adjacent portions of the sinusoidal capillaries are dilated, filled with erythrocytes, most of the hepatocytes are destroyed (necrosed), preserved hepatocytes are atrophied, macrophages with hemosiderin granules are observed. In peripheral areas, the hepatic tracts have usual appearance, the sinusoids are not congested, in some hepatocytes there are lipidic degeneration (micro-macrovesicular steatosis).

*The selective congestion of the central areas of the lobules is due to the peculiarities of the blood circulation and angioarchitectonics of the liver and it is explained by the fact that the venous stasis mainly comprises the hepatic veins and collecting veins but at the level of lobules - central veins and adjacent portions of the sinusoidal capillaries. However, the stasis does not extend to the periphery of the lobules due to the higher speed and pressure of blood in the peripheral areas of the sinusoids where arterioles enter from hepatic artery system. Therefore, the center of the liver lobule is congested, and the periphery - no.*



**№ 10. Chronic hepatic congestion (*nutmeg liver*). (H-E. stain).**

## **№ 9. Chronic pulmonary congestion. (H-E. stain).**

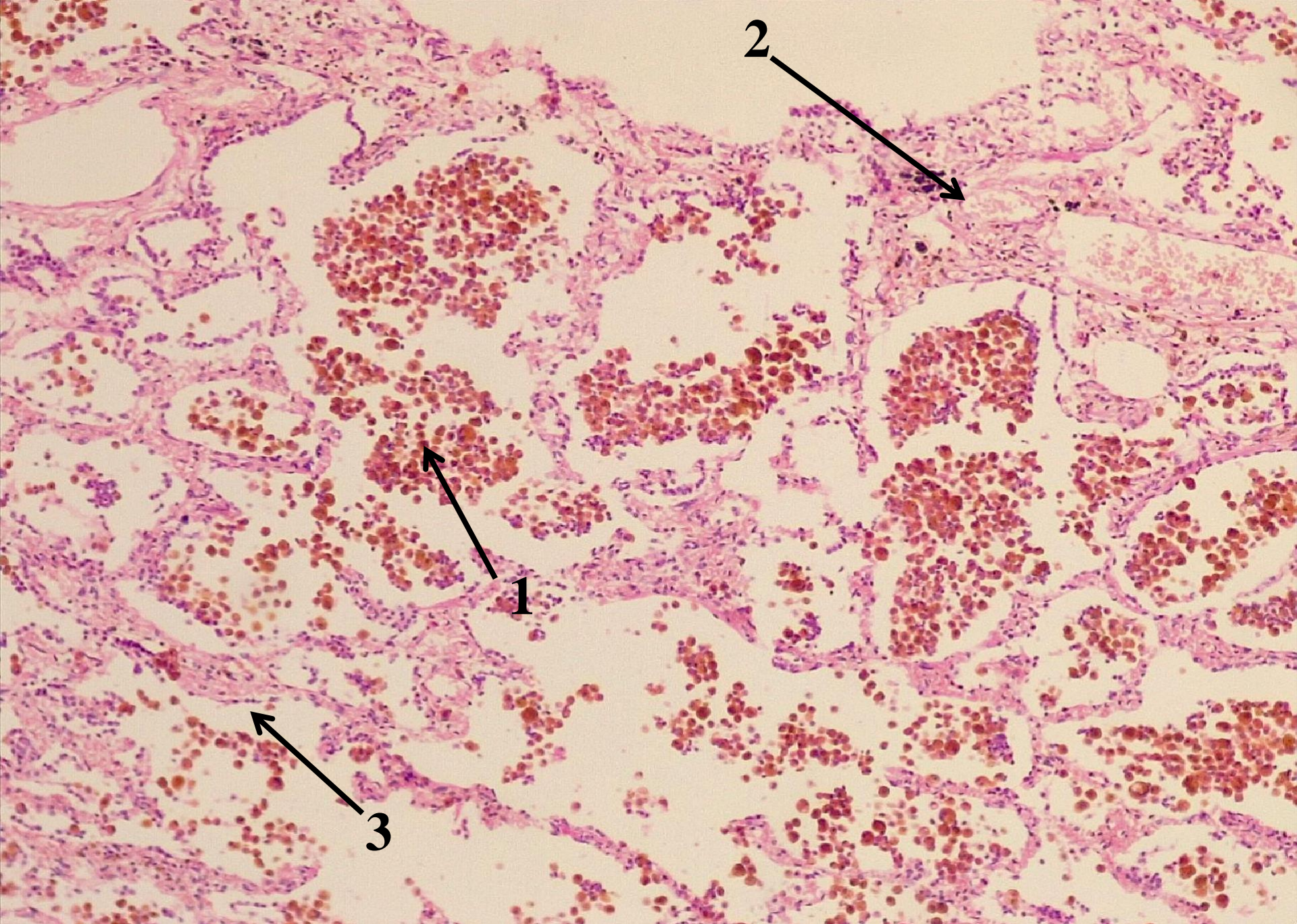
### **Indications:**

1. Clusters of sideroblasts and siderophages in the lumen and walls of the alveoli.
2. Dilated and congested vessels in interalveolar septa.
3. Sclerosed and thickened interalveolar septa.

The interalveolar septa are thickened, sclerosed, the veins and septal capillaries are dilated and congested. Agglomerations of phagocytic cells (alveolar macrophages) loaded with hemosiderin granules of brown color (sideroblasts and siderophages) are observed in the alveoli; part of the alveoli contains eosinophilic edema fluid, erythrocytes or remnants of disintegrated red blood cell.

*Macroscopically the lungs in chronic congestion are enlarged in volume and mass, they have a dense consistency and brown color on section. The increased consistency is due to the excessive proliferation of connective tissue in the alveolar walls, and coloring - due to accumulation of hemosiderinic pigment. The lesions are more pronounced in the postero-inferior areas of the lungs.*

*Chronic venous congestion of the lungs is encountering in left heart failure, mitral stenosis and chronic ischemic heart disease (because of this it is also called "cardiac lung"). In the sputum of patients with heart failure, macrophages with hemosiderin granules in the cytoplasm can be found and they are called "heart failure cells". The presence of hemosiderinic pigment gives to sputum a rusty hue.*



**№ 9. Chronic pulmonary congestion. (H-E. stain).**

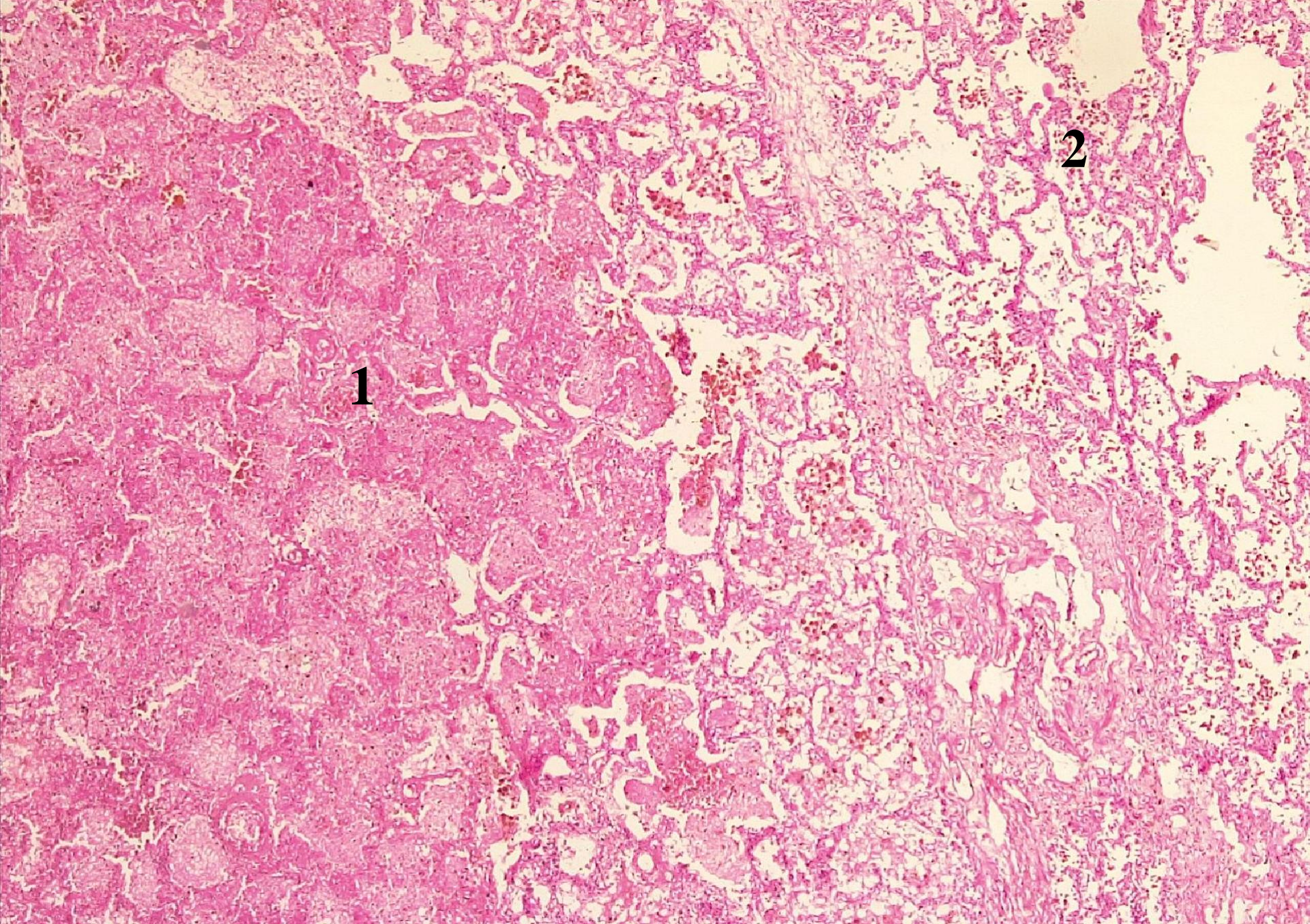
## **№ 1. Pulmonary hemorrhagic infarction. (H-E. stain).**

### **Indications:**

1. Infarction area:
  - a. necrotized interalveolar septa;
  - b. hemolyzed erythrocytes in alveolar lumen.
2. Adjacent lung tissue with venous stasis and edema.

With naked eye in microspecimen a non-aerated area is observed, at the small objective in this area the alveoli are filled with extravasated erythrocytes, most of the them are hemolysed of eosinophilic color, in some alveoli groups of siderophages are observed, the alveolar septa are necrotic, cells are without nuclei (karyolysis) of intensely eosinophilic color. Adjacent lung tissue is with signs of chronic venous congestion.

*The hemorrhagic character of the infarction is determined by two factors: 1) double circulation of the lung tissue: from the pulmonary artery (small circulation) and the bronchial artery (large circulation); between these arteries there are multiple anastomoses, which are not functional under physiological conditions; the obstruction of the pulmonary artery is followed by the reflex opening of the anastomoses and penetration under pressure of the blood from the bronchial artery into the ischemic area, which leads to rupture of the capillary and venules walls of the interalveolar septa and the blood flow into the alveoli; 2) venous stasis, because it favors the retrograde circulation of blood in veins and flooding of the ischemic area (it is most frequently encountered in left heart failure, especially in mitral stenosis).*



**№ 1. Pulmonary hemorrhagic infarction. (H-E. stain).**

## **№ 5. Renal infarction. (H-E. stain).**

### **Indications:**

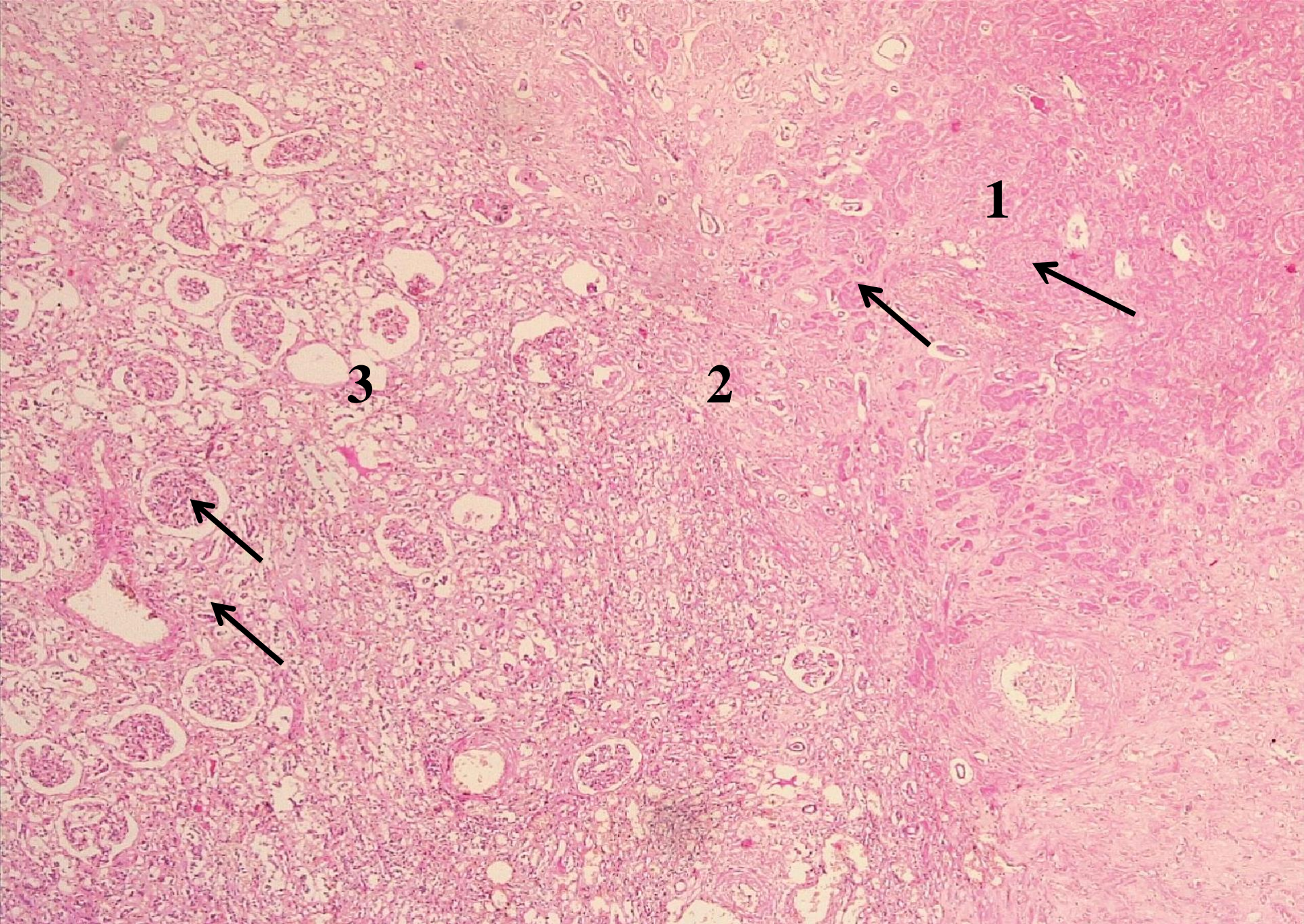
1. Infarction area:
  - a. necrosed glomerulus;
  - b. necrosed tubule.
2. Demarcation zone:
  - a. congested vessels;
  - b. hemorrhages.
3. Adjacent renal tissue:
  - a. unchanged glomerulus;
  - b. unchanged tubule.

In microspecimen a zone of necrosis is determined, in which the glomeruli and tubes of eosinophilic color have been preserved, but without nuclei (karyolysis), at the periphery of this area are hyperemic vessels, haemorrhages, and neutrophilic infiltration (inflammation of the demarcation ), the adjacent renal tissue has normal structure.

*Macroscopically, the renal infarction has yellowish-white color and triangular shape with the tip pointing towards the hilum and the base towards the capsule, on the surface of the capsule may be fibrin deposits. Clinically it is manifested by hematuria.*

*The most common causes are thromboembolism or thrombosis of renal artery. It is encountering in atherosclerosis of the aorta and renal arteries, rheumatic and infective endocarditis, hypertension, myocardial infarction with intracardiac thrombosis. The most common consequence is organization (cicatrization).*





**№ 5. Renal infarction. (H-E. stain).**

## ***II. Macrospecimens:***

### **№ 71. Chronic hepatic congestion (*nutmeg liver*).**

The liver is enlarged in size and mass, has an extended, smooth capsule, increased consistency, rounded anterior margin, with mottled appearance on section, similar to the nutmeg core due to the alternation of small, dark red spotty foci of central areas of lobules with others of brown color (usual color of the hepatic parenchyma) or slightly yellow due to the hepatocyte steatosis - of the peripheral areas of the lobes (the dimensions of the hepatic lobule are  $\sim 2 \times 0.7$  mm).

*It can be seen in right heart failure or global heart failure ("cardiac liver"). Examples: pulmonary hypertension (in pulmonary emphysema, bronchiectasis, diffuse pneumofibrosis, secondary pulmonary tuberculosis, etc.) and pulmonary artery stenosis. An analogous pattern develops in hepatic vein thrombosis (Budd-Chiari syndrome).*

### **№ 141. Ischemic infarction of the spleen.**

The infarct area is well delimited, it has white-yellowish color, dense consistency (coagulative necrosis) and conical shape with the tip towards the spleen hilum and the base towards the capsule, on the capsule are fibrin deposits.

*The most common cause is thrombosis or thromboembolism of the spleen artery. It is found in rheumatic or infective endocarditis, leukemias, ischemic heart disease, atherosclerosis etc.*

*The conical shape and white color are determined by the magistral type of vascularization of the spleen with poor collateral circulation, which excludes the possibility of the blood entering in ischemic area through collaterals.*

*The most frequent consequence is organization (cicatrization) of the infarction with spleen deformation.*



**№ 71. Chronic hepatic congestion (*nutmeg liver*).**



**№ 141. Ischemic infarction of the spleen.**

### **№ 38. Pulmonary hemorrhagic infarction.**

The infarct area has dense consistency, red-dark color, conical shape with the tip towards the hilum and the base towards the pleura. It is compact, non-aerated, filled with blood. Fibrinous deposits are observed on the pleura.

*The cause of pulmonary infarction is obstruction of a branch of the pulmonary artery by thrombosis or embolism (with starting point from the peripheral venous system, especially from the veins of the lower limbs).*

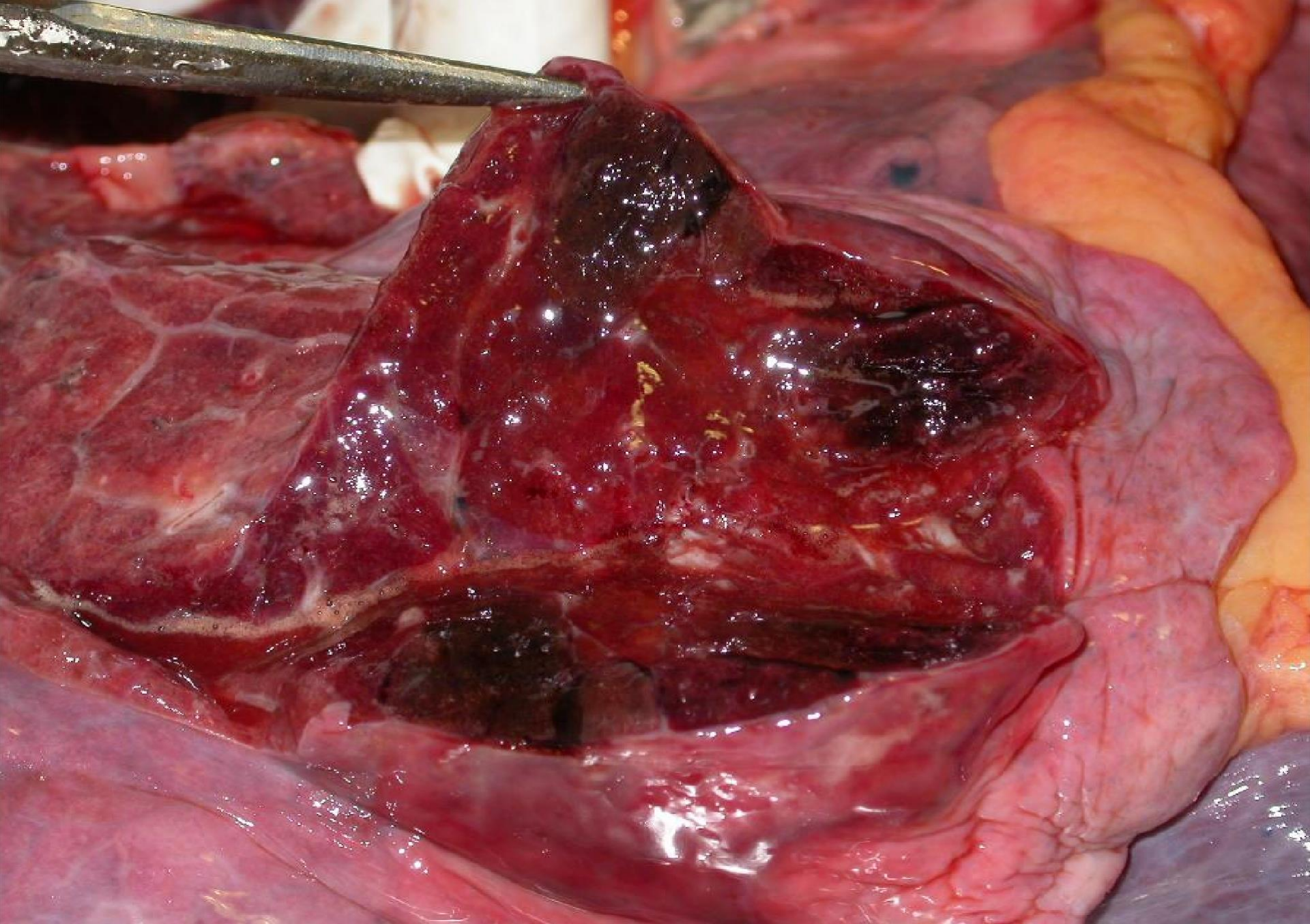
*Clinically, the pulmonary infarction is manifested by hemoptysis (the presence of blood in the sputum) and pleural friction rub at auscultation.*

*The common consequence of pulmonary infarction is organization (scarring). Possible complications: post-infarction pneumonia, pulmonary abscess, pleural empyema, pneumothorax, pulmonary gangrene.*

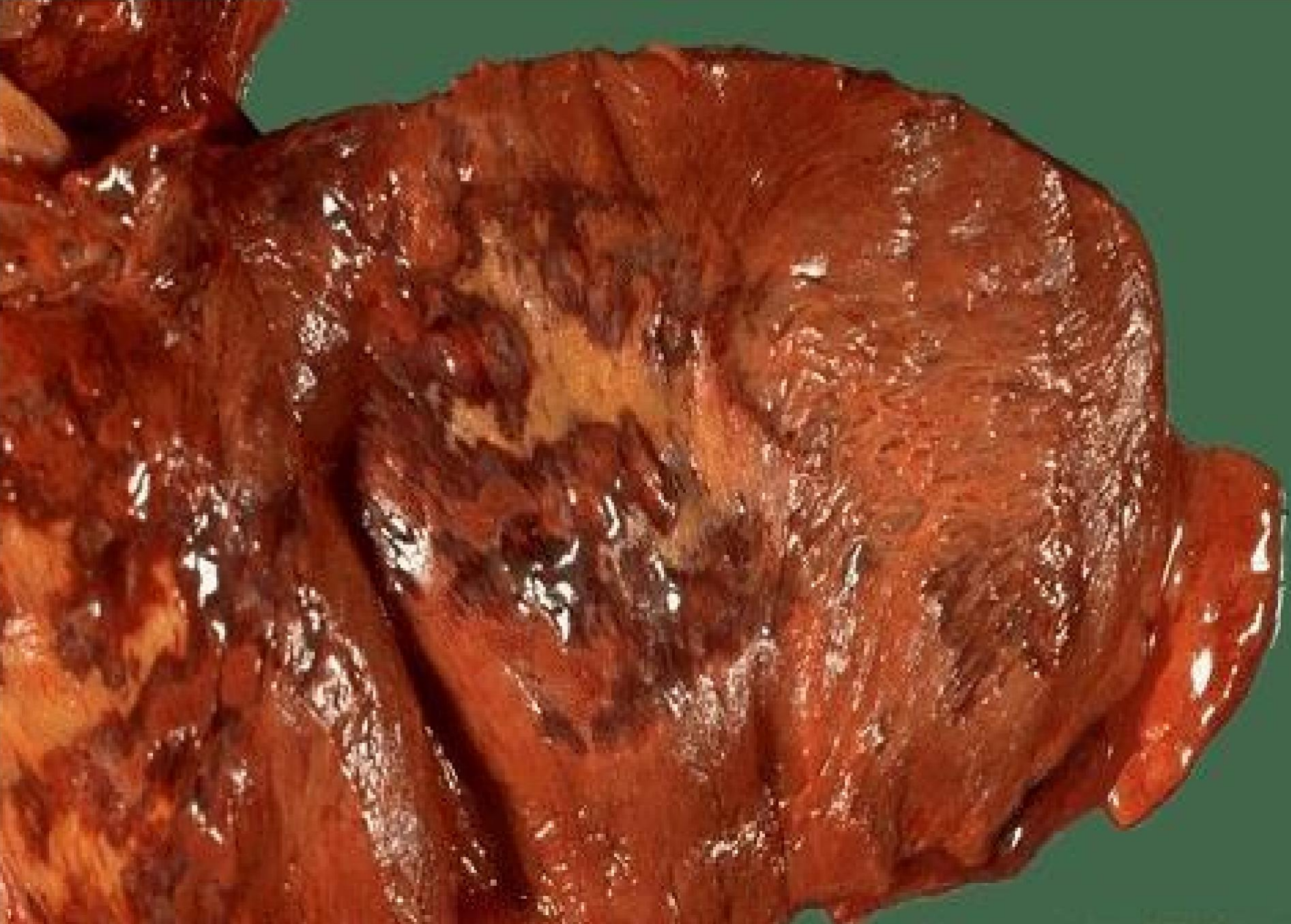
### **№ 9. Myocardial infarction.**

In the wall of the left ventricle on section there is an area of yellow-white color of irregular shape, surrounded by hemorrhages (white ischemic infarction with hemorrhagic rim), the consistency of the infarction zone is decreased (myomalacia).

*In the absolute majority of cases myocardial infarction occurs on the background of stenosing atherosclerosis of the coronary arteries, the causes being thrombosis, angiospasm or thromboembolism. It is the main form of ischemic heart disease. The clinical significance and effects of myocardial infarction depend on its location and extension. The most frequent consequence is organization (cicatritation) of the necrosed area - macrofocal postinfarction cardiosclerosis.*



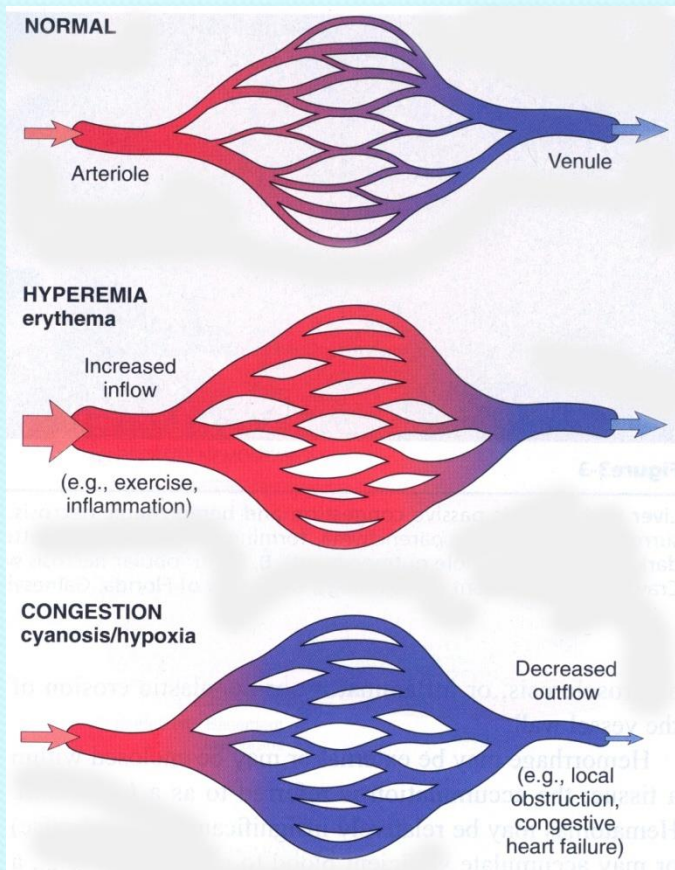
**№ 38. Pulmonary hemorrhagic infarction.**



**№ 9. Myocardial infarction.**

# Arterial hyperemia (hyperemia) & Venous hyperemia (congestion)

Both processes represent an increase pressure and volume of blood in an organ or tissue.



Arterial hyperemia (hyperemia): active process; macroscopically manifested by red color, elevated temperature.

Venous hyperemia (congestion): passive process; macroscopically manifested by dark purple-red (cyanotic) color, low temperature.



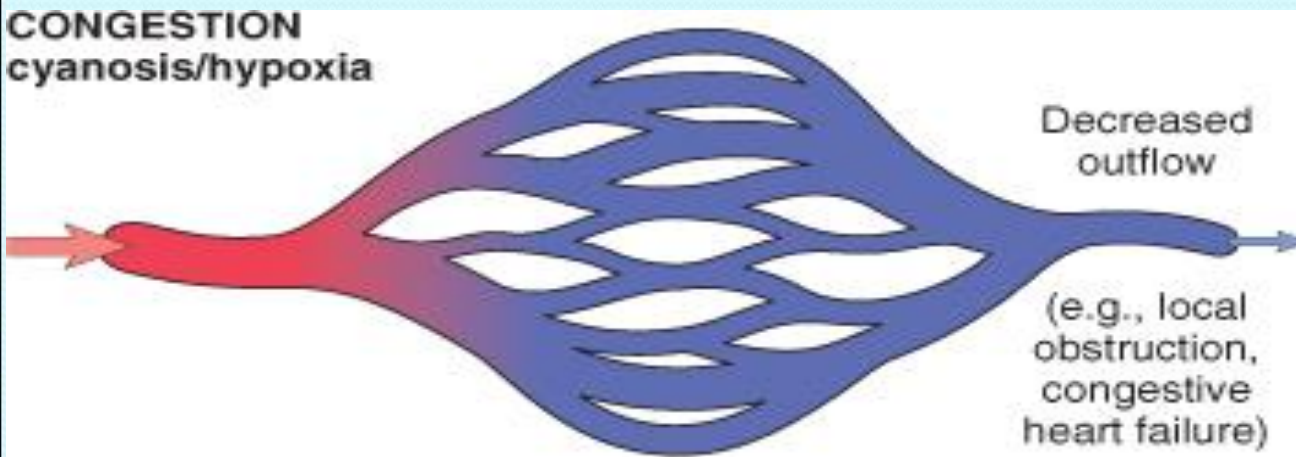


**HYPEREMIA**  
erythema

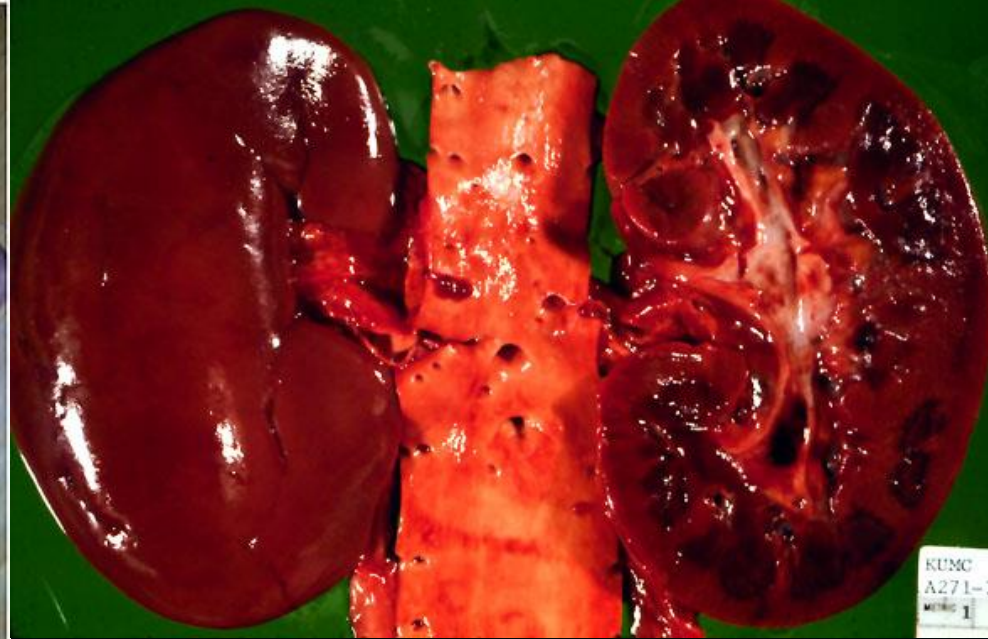


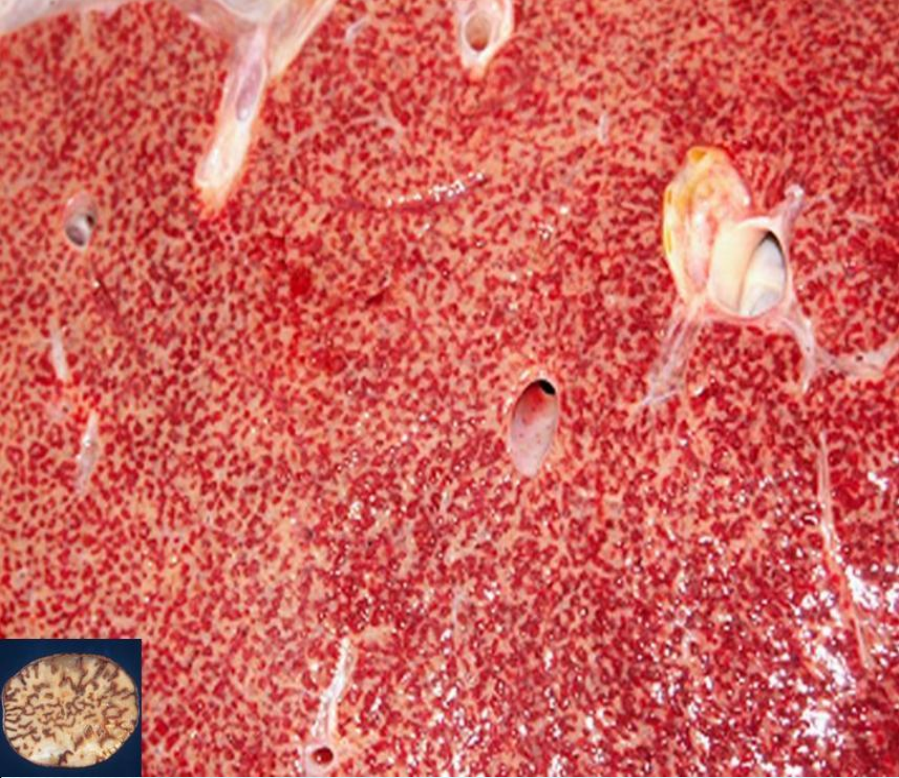
**Arterial hyperemia.**

# Venous hyperemia (congestion).

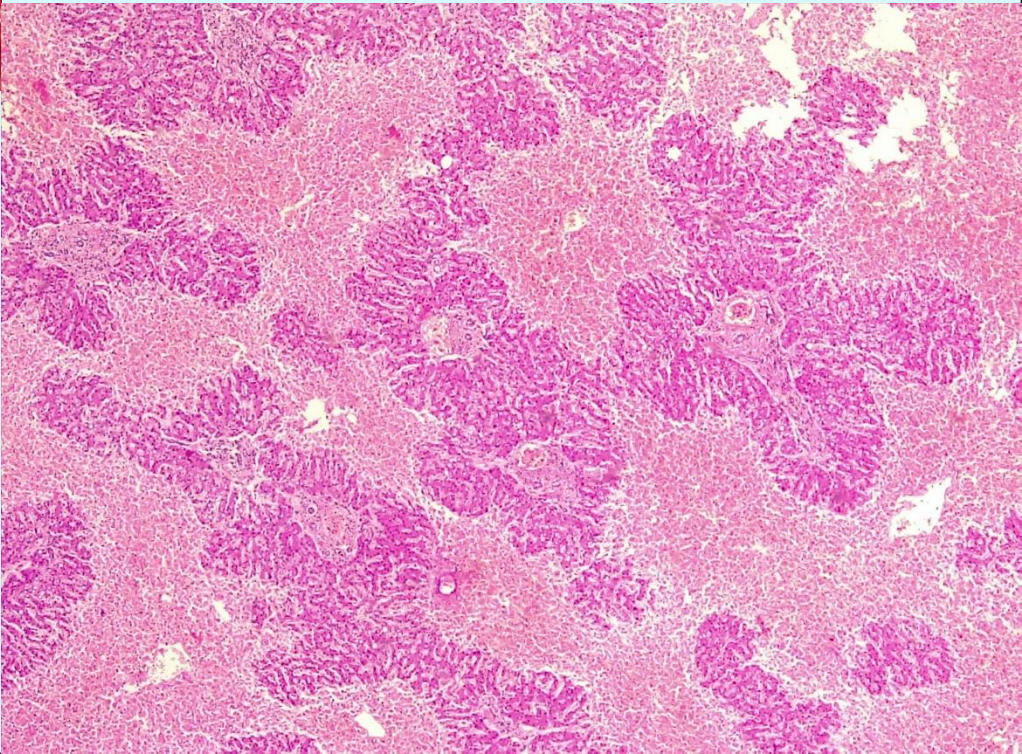


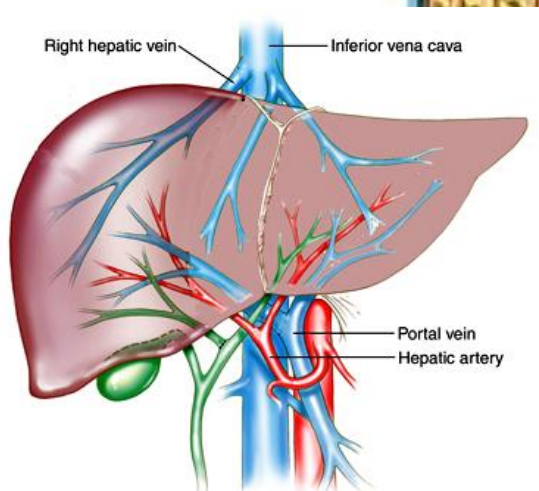
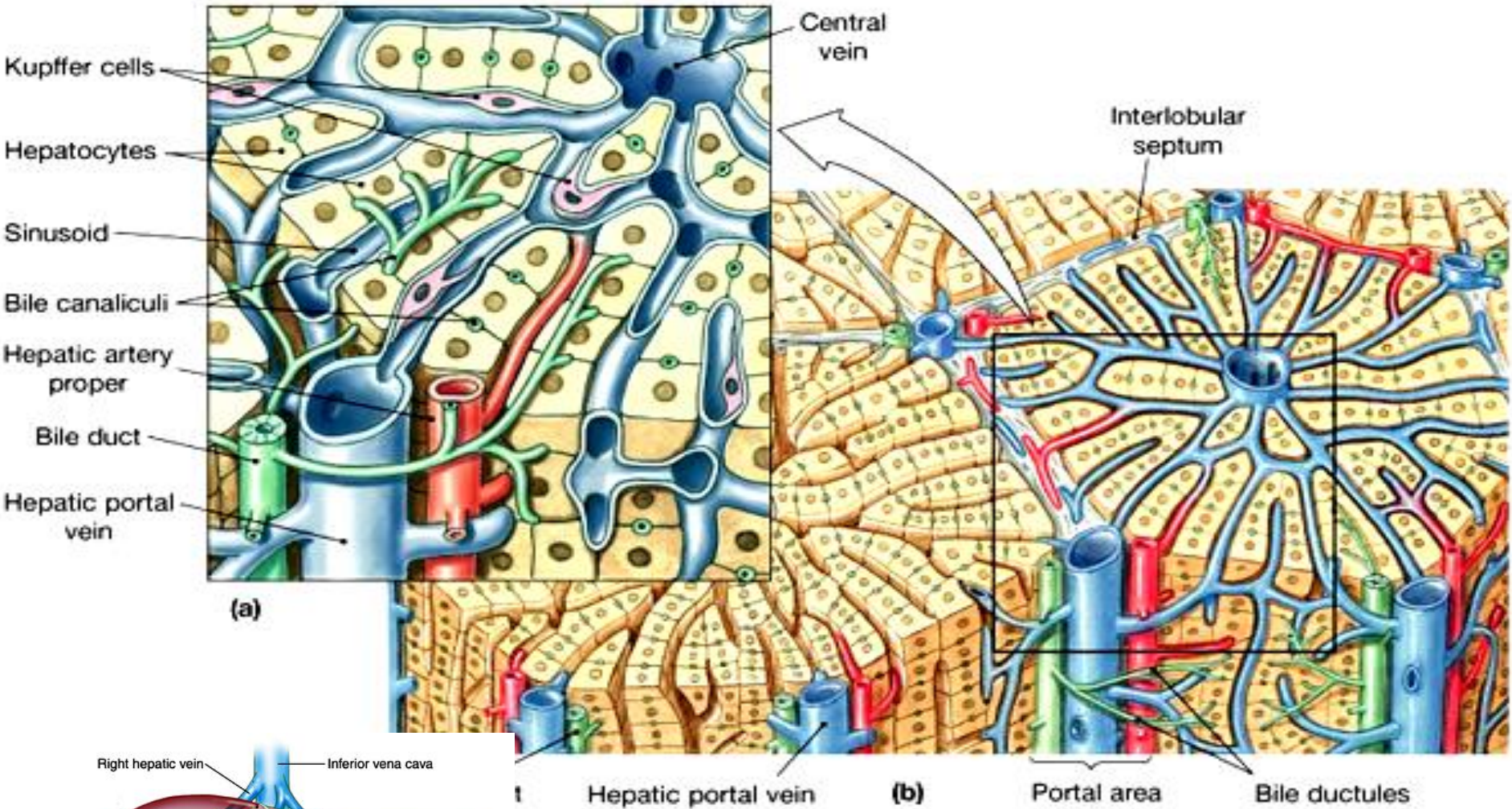
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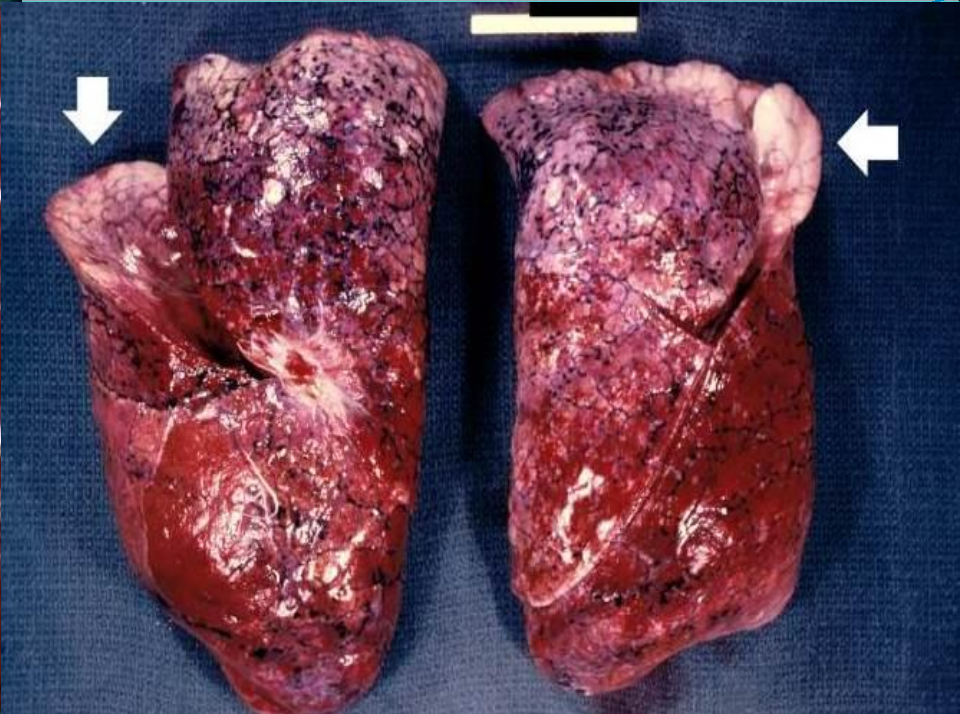


**Chronic liver congestion.**





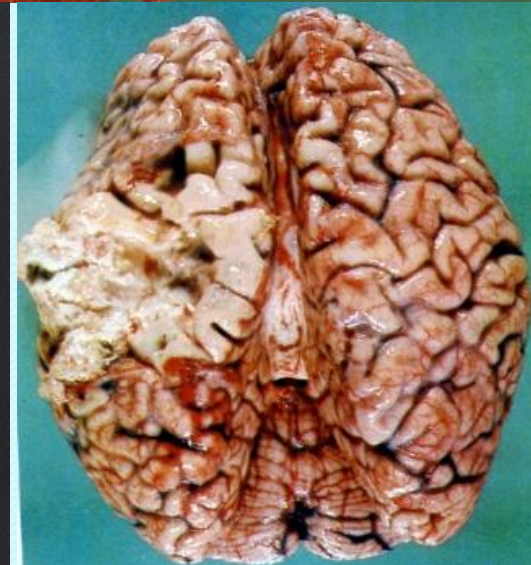
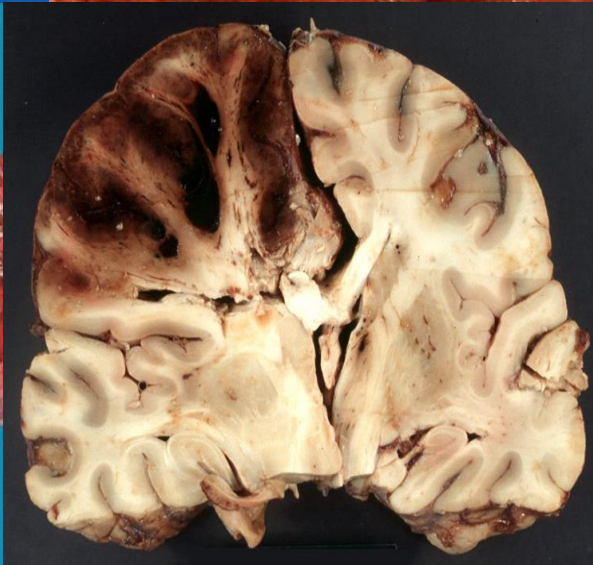
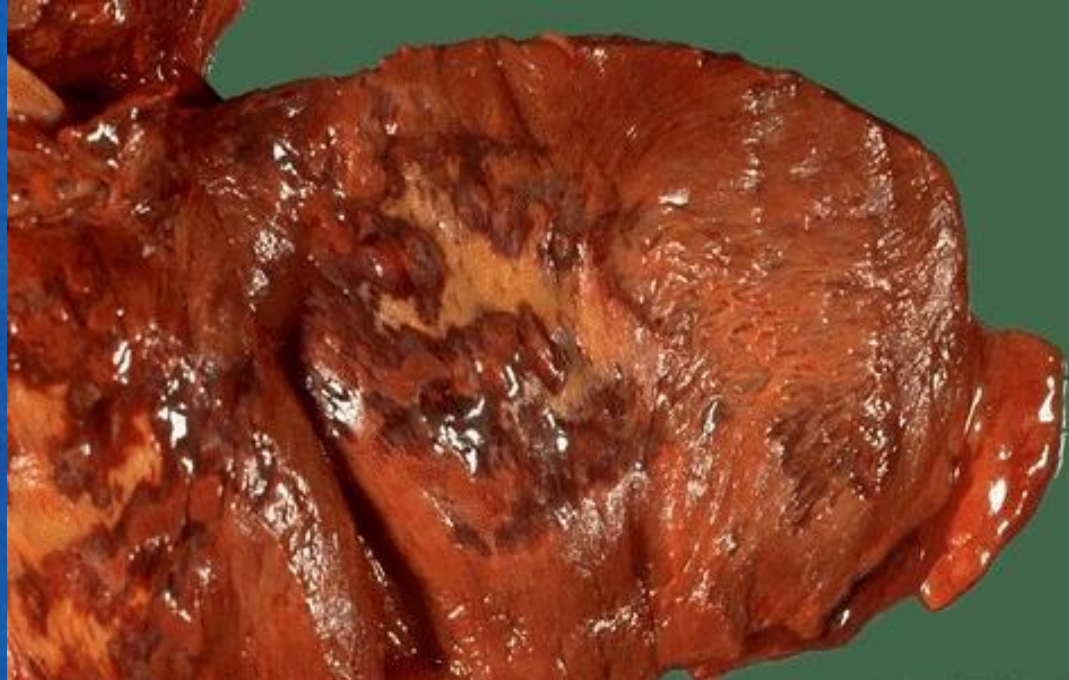
# Hepatic blood circulation.



Chronic venous hyperemia (congestion)  
of the lung  
*(brown induration of the lung).*

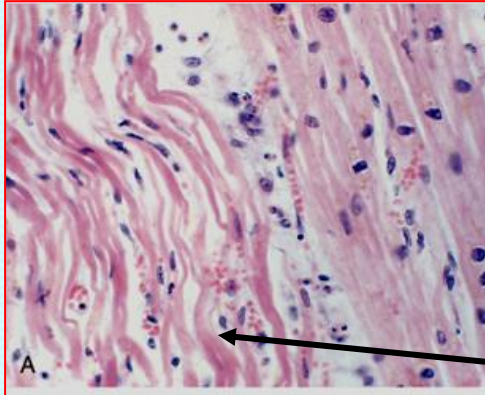
Heavy lungs  
Firm and rusty or brown due to accumulation of hemosiderin

# Infarction.



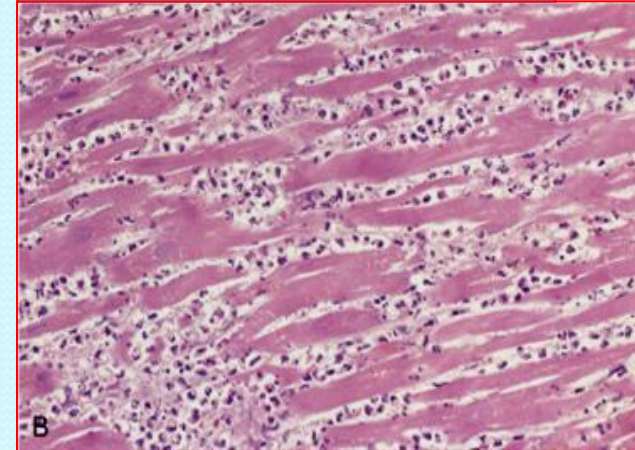
# Acute myocardial infarction.

One-day-old infarct

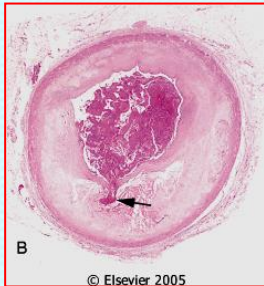


wavy fibers

Up to 3 days duration

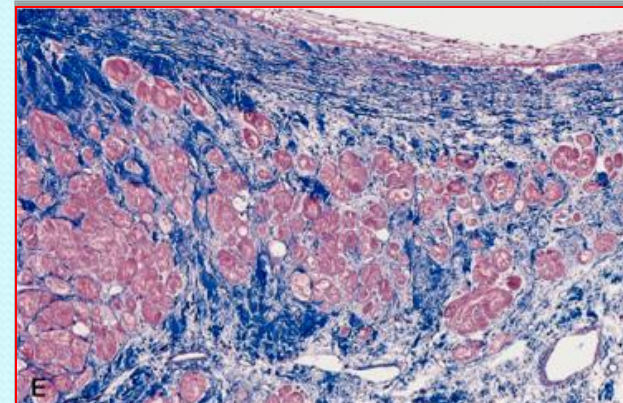


Neutrophilic infiltrate

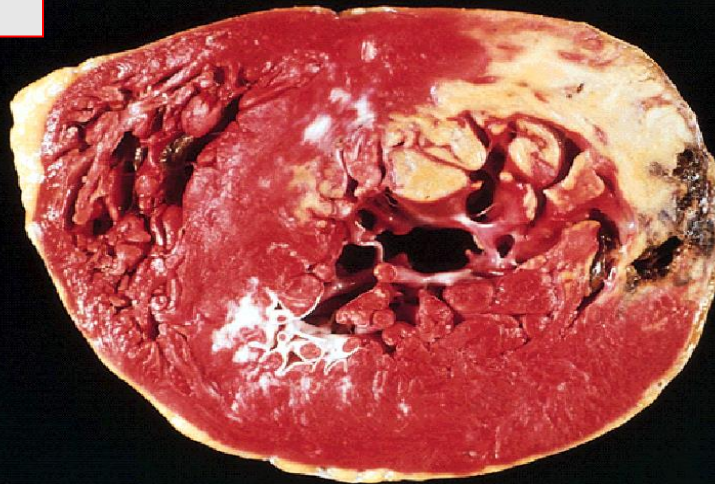


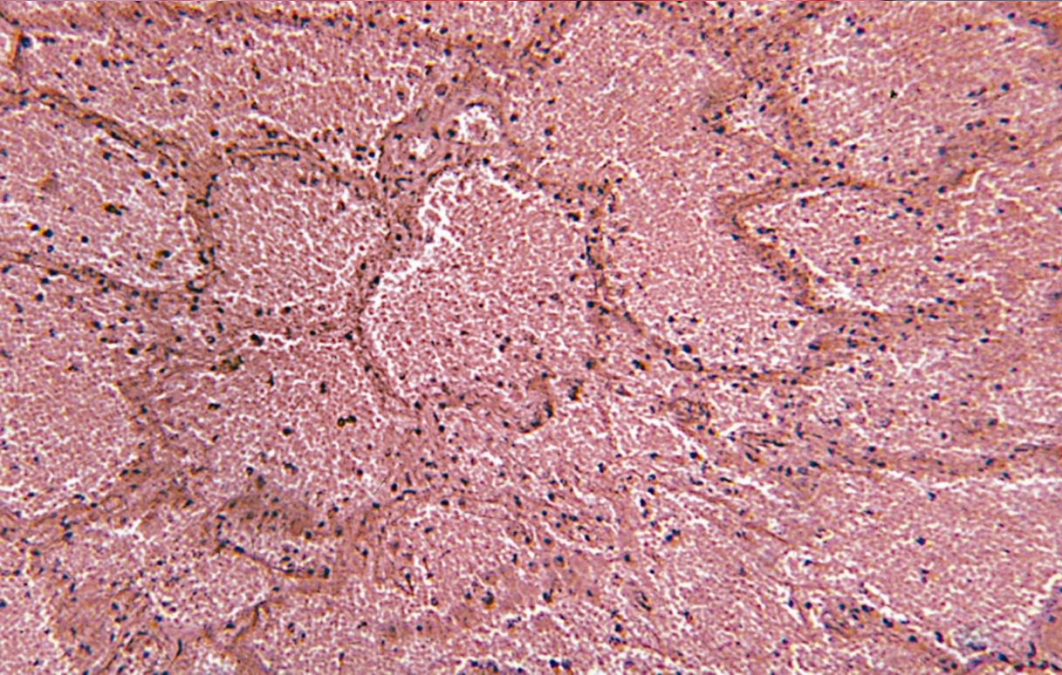
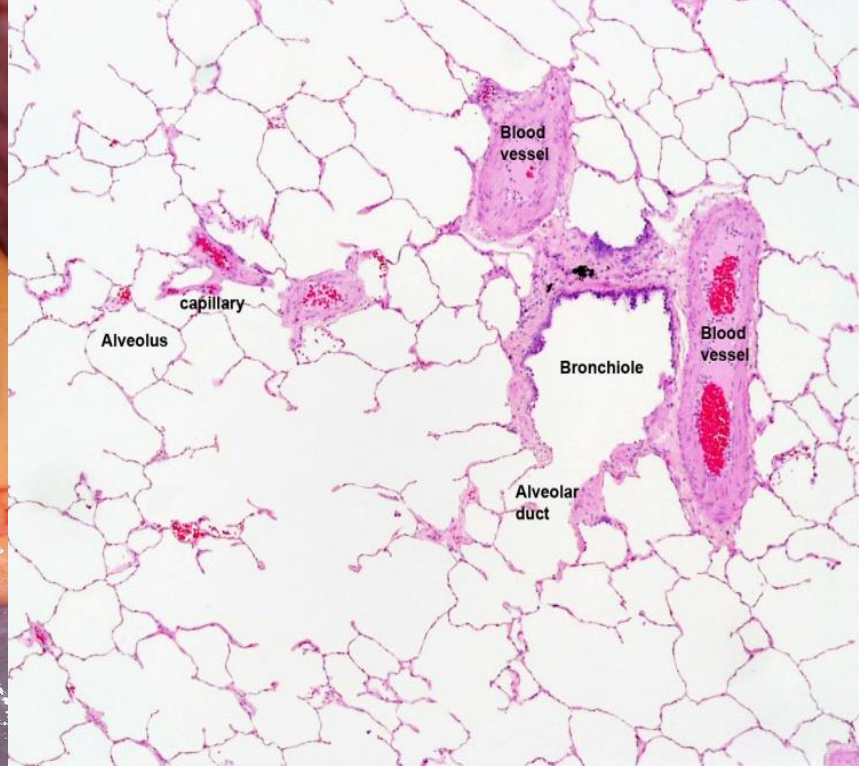
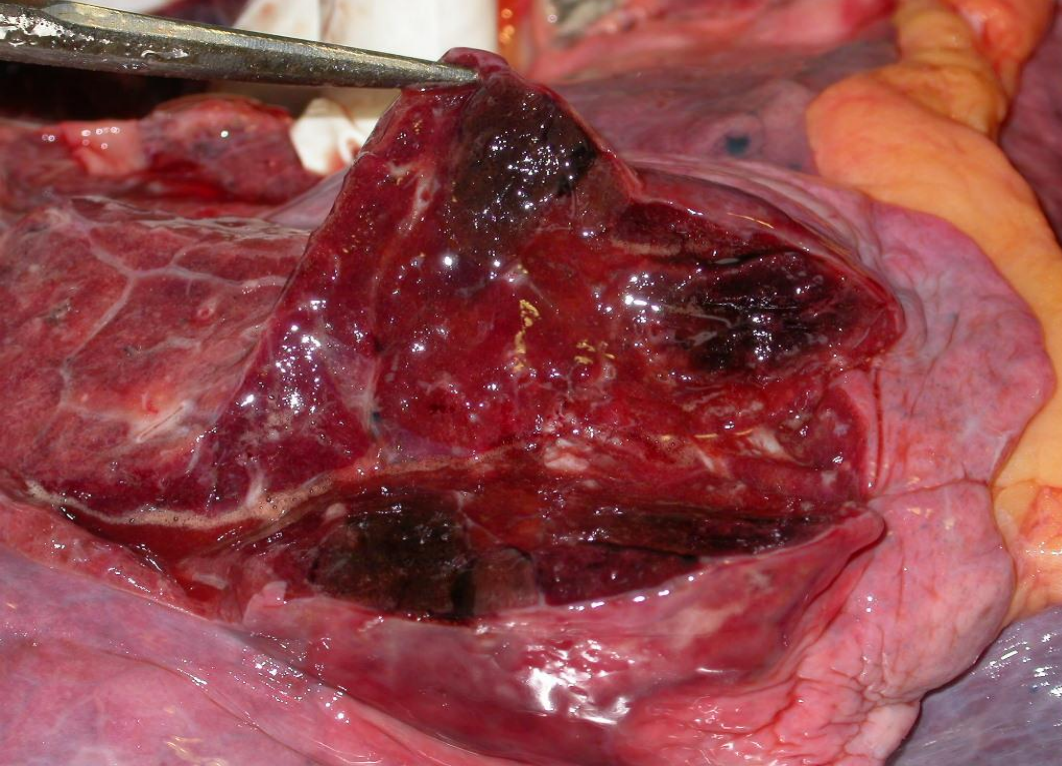
coagulative necrosis

>3 weeks



Scar





**Pulmonary infarction.**





**Renal infarction.**



# **EDEMA**

**“Increased Fluid in the Interstitial  
Tissue Spaces”**

**Also Includes:**

**Hydrothorax, Hydropericardium  
Hydroperitonium or Ascites and  
Anasarca.**

# EDEMA

- **Increased interstitial fluid volume**
- **Two major types**
- **Local** - inflammation
- **Generalised** - anasarca - **Systemic causes.**

# Pathophysiological Classification

- Inflammatory Edema
- Non-Inflammatory Edema
  - 1. Increased Hydrostatic Pressure**
  - 2. Reduced Plasma Osmotic Pressure**
  - 3. Lymphatic Obstruction**
  - 4. Sodium Retention**

# Pathophysiological Classification

*(Continued....)*

- Increased Hydrostatic Pressure:
  - 1. Impaired Venous Return: (e.g. CCF, Constrictive Pericarditis, Liver Cirrhosis, Venous Obstruction)**
  - 2. Arteriolar dilatation: (e.g. Exposure to Heat, Neurohormonal dysregulation)**

# Pathophysiological Classification(*Continued....*)

- Reduced Plasma Osmotic Pressure
  1. **Protein-Losing Glomerulopathies (Nephrotic Syndrome)**
  2. **Liver Cirrhosis (Ascites)**
  3. **Malnutrition**
  4. **Protein-Losing gastroenteropathies**

# Pathophysiological Classification(*Continued....*)

- Lymphatic Obstruction
  1. Inflammatory
  2. Neoplastic
  3. Postsurgical
  4. Postirradiation

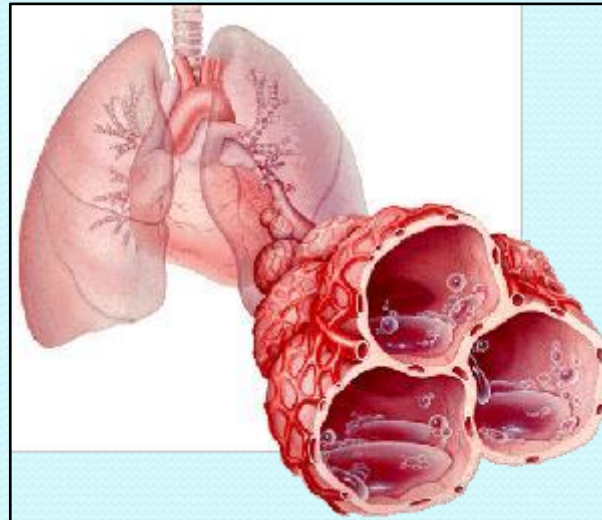
# Pathophysiological Classification

*(Continued....)*

- Sodium Retention
  1. Excessive salt Intake with Renal Insufficiency
  2. Increased Tubular Reabsorption of  $\text{Na}^+$
  3. Renal Hypoperfusion
  4. Increased Renin-Angiotension-Aldosterone Secretion



# EDEMA

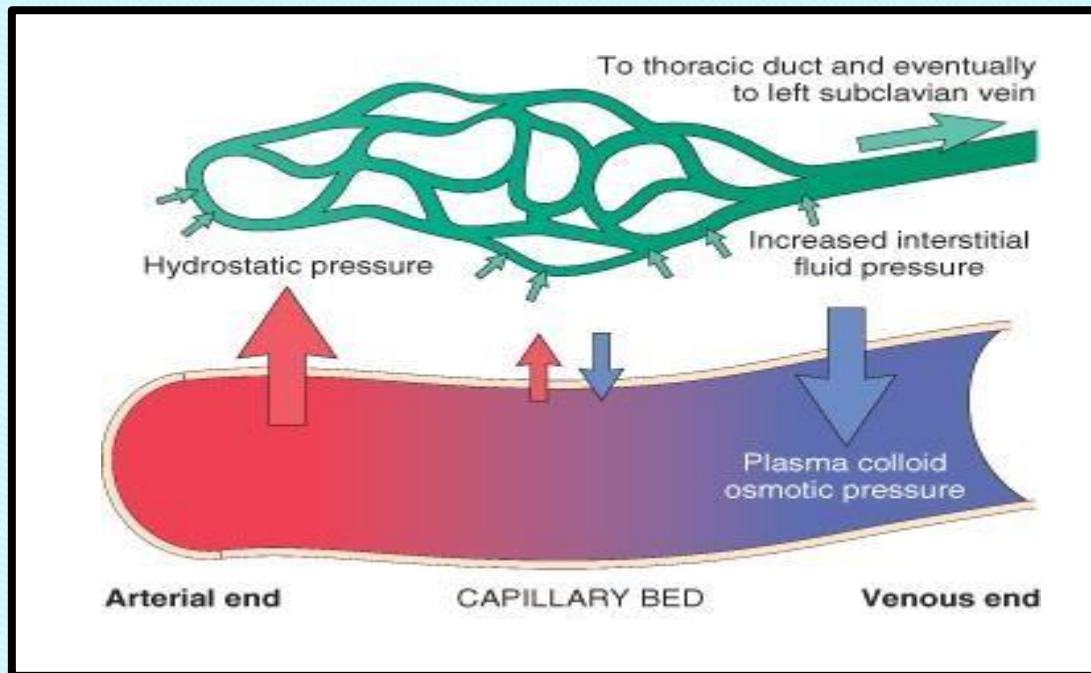


**increased fluid in the interstitial tissue spaces**

# Fetal Anasarca



**Factors affecting fluid balance across capillary walls. Capillary hydrostatic and osmotic forces are normally balanced so that there is no net loss or gain of fluid across the capillary bed. However, increased hydrostatic pressure or diminished plasma osmotic pressure leads to a net accumulation of extravascular fluid (edema). As the interstitial fluid pressure increases, tissue lymphatics remove much of the excess volume, eventually returning it to the circulation via the thoracic duct. If the ability of the lymphatics to drain tissue is exceeded, persistent tissue edema results.**



	<b>TRANSUDATE</b>	<b>EXUDATE</b>
<b>Etiology</b>	<b>Increased hydrostatic pressure</b>	<b>Inflammation</b>
<b>Specific gravity (g/mL)</b>	<b>&lt;1.015</b>	<b>&gt;1.015</b>
<b>Total protein (g/dL)</b>	<b>&lt;3.0</b>	<b>&gt;3.0</b>
<b>Fluid/serum protein ratio</b>	<b>&lt;0.5</b>	<b>&gt;0.5</b>
<b>Fluid/serum LDH ratio</b>	<b>&lt;0.6</b>	<b>&gt;0.6</b>
<b>Fluid/serum glucose ratio</b>	<b>&gt;1.0</b>	<b>&lt;1.0</b>
<b>Cells (leukocytes)</b>	<b>No</b>	<b>Yes</b>

# Shock

■  
■

- *"Depressed vital functions due to decreased circulating blood volume"*
- **Types:**
  - **Hypovolaemic** - true/vasovagal
  - **Cardiogenic** – Heart failure, MI.
  - **Obstructive** – Pulm embolism.
  - **Anaphylactic** – vasodilation due to allergy.
  - **Septic** – capillary damage by infection.

# Shock Features:

- **Hypotension**
- **Tachycardia**
- **Cold clammy skin**
- **Rapid shallow respiration.**
- **Drowsiness, confusion, irritability**
- **Multi organ failure.**

# **HYPERMIA AND CONGESTION**

**“Both indicates a local  
increased volume of blood in a  
particular tissue.”**

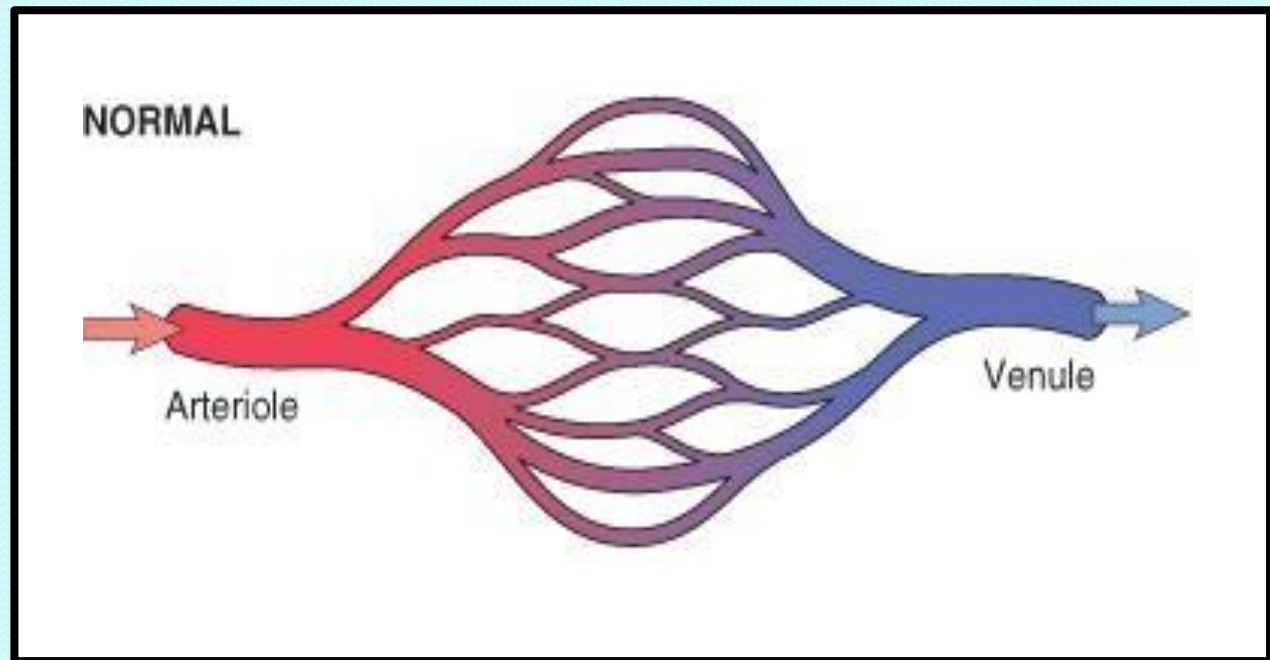
# **HYPERMIA AND CONGESTION**

	<b>HYPEREMIA</b>	<b>CONGESTION</b>
<b>1</b>	<b>An active process</b>	<b>A passive process</b>
<b>2</b>	<b>Increased blood flow (vasodilatation)</b>	<b>Impaired blood flow</b>
<b>3</b>	<b>During exercise &amp; in inflammation</b>	<b>Venous obstruction &amp; cardiac failure</b>
<b>4</b>	<b>Oxygenated blood (Redder)</b>	<b>Deoxygenated blood (Cyanosed)</b>



# Hyperemia versus congestion.

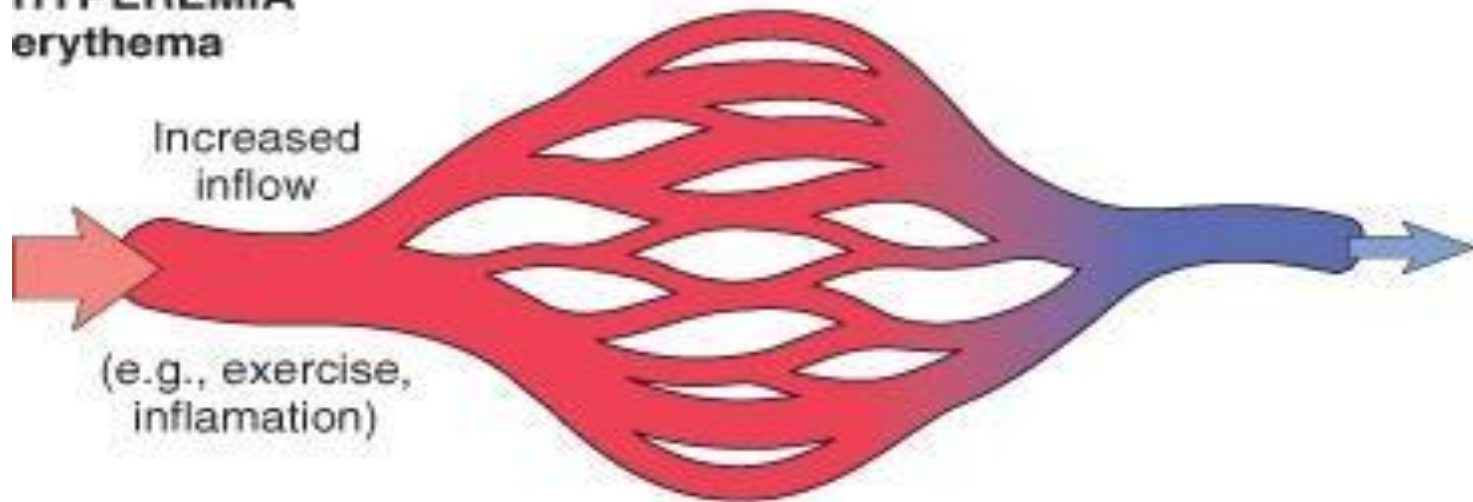
In both cases there is an increased volume and pressure of blood in a given tissue with associated capillary dilation and a potential for fluid extravasation.



# Hyperemia versus congestion.

In hyperemia, increased inflow leads to engorgement with oxygenated blood, resulting in erythema.

**HYPEREMIA**  
erythema

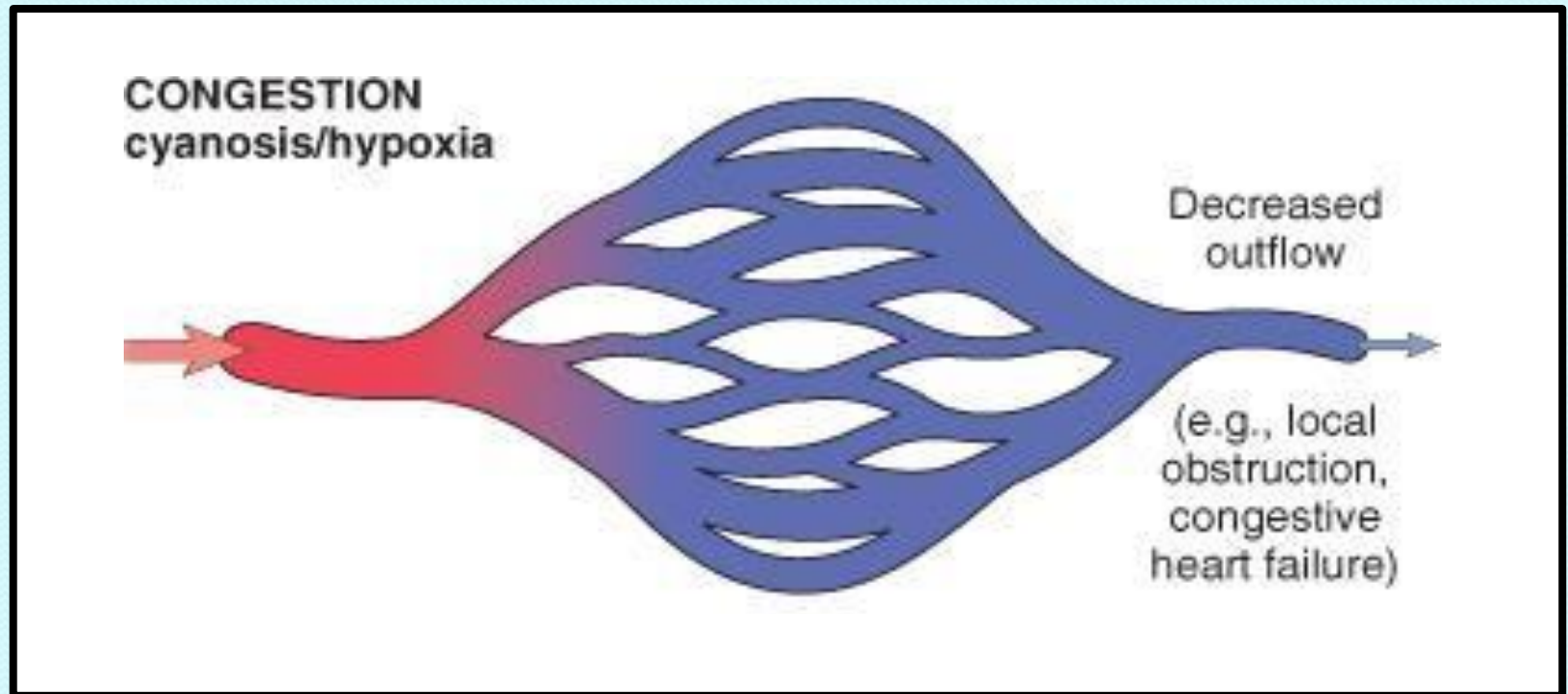


# Hyperemia



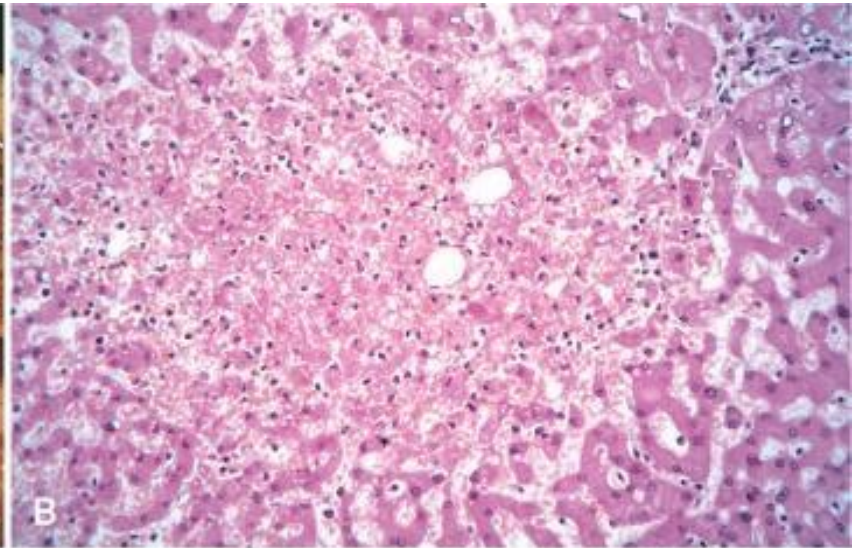
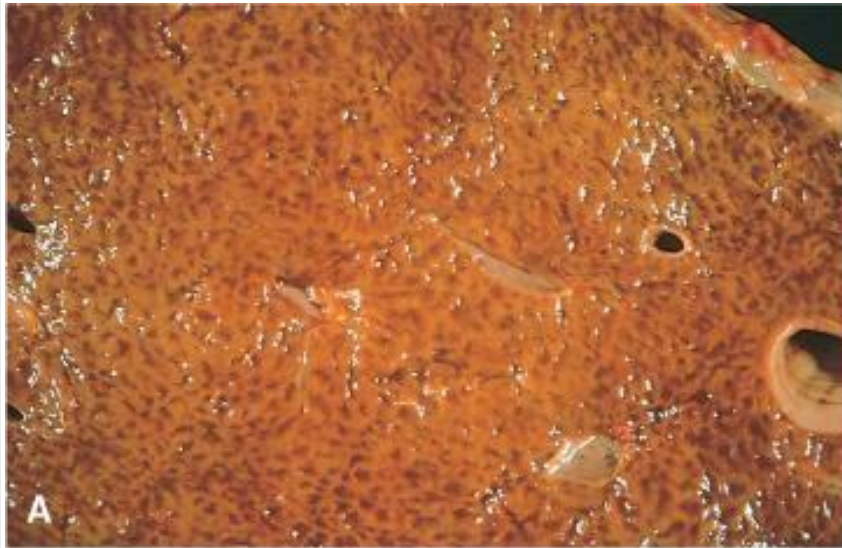
# Hyperemia versus congestion.

In congestion, diminished outflow leads to a capillary bed swollen with deoxygenated venous blood and resulting in cyanosis.



# Liver with chronic passive congestion and hemorrhagic necrosis.

- **A, Central areas are red and slightly depressed compared with the surrounding tan viable parenchyma, forming the so-called "nutmeg liver" pattern.**
- **B, Centrilobular necrosis with degenerating hepatocytes, hemorrhage, and sparse acute inflammation.**



# Ischemia

- **●Greek *ischein* "to restrain" + *haima* "blood"**
- **●Ischemia occurs when the blood supply to a tissue is inadequate to meet the tissue's metabolic demands**
- **●Ischemia has 3 principal biochemical components:**
  - **–Hypoxia (including anoxia)**
  - **–Insufficiency of metabolic substrates**
  - **–Accumulation of metabolic waste**
- **●Therefore, ischemia is a greater insult to the cells and tissues than hypoxia alone**

# Causes of Ischemia:

## Decreased Supply

- **Vascular insufficiency:**

- **Atherosclerosis**

- **Thrombosis**

- **Embolism**

- **Torsion**

- **Compression**

- **Hypotension:**

- **Shock**

- **Hemorrhage**

# Causes of Ischemia:

## Increased Demand

- **Increased tissue mass (hypertrophy)**
- **Increased workload (tachycardia, exercise)**
- **Increased tissue “stress” (cardiac dilatation)**



# Effect of Ischemia Depends on Cell Type

- **“Parenchymal” cells are more susceptible than “stromal” cells**
- **Different parenchymal cells have different thresholds for ischemia:**
  - **Neurons: 3-4 min**
  - **Cardiac muscle, hepatocytes, renal tubular cells, gastrointestinal epithelium: 20-80 min**
  - **Fibroblasts, epidermis, skeletal muscle: hours**

# **Effect of Ischemia** Depends on Microvascular Anatomy

- **Subendocardial hypoxia in the heart**
- **Watershed infarcts in the brain**
- **Ischemia due to countercurrent exchange in the intestinal villi**
- **Resistance in dual perfusion organs**

# Infarction

Latin *infarctus*, pp. of *infarcire* "to stuff"

- An infarct is an area of tissue/organ necrosis caused by ischemia
- Infarctions often result from sudden reduction of arterial (or occasionally venous) flow by thrombosis or embolism
  - Infarctions can also result from progressive atherosclerosis, spasms, torsions, or extrinsic compression of the vessels

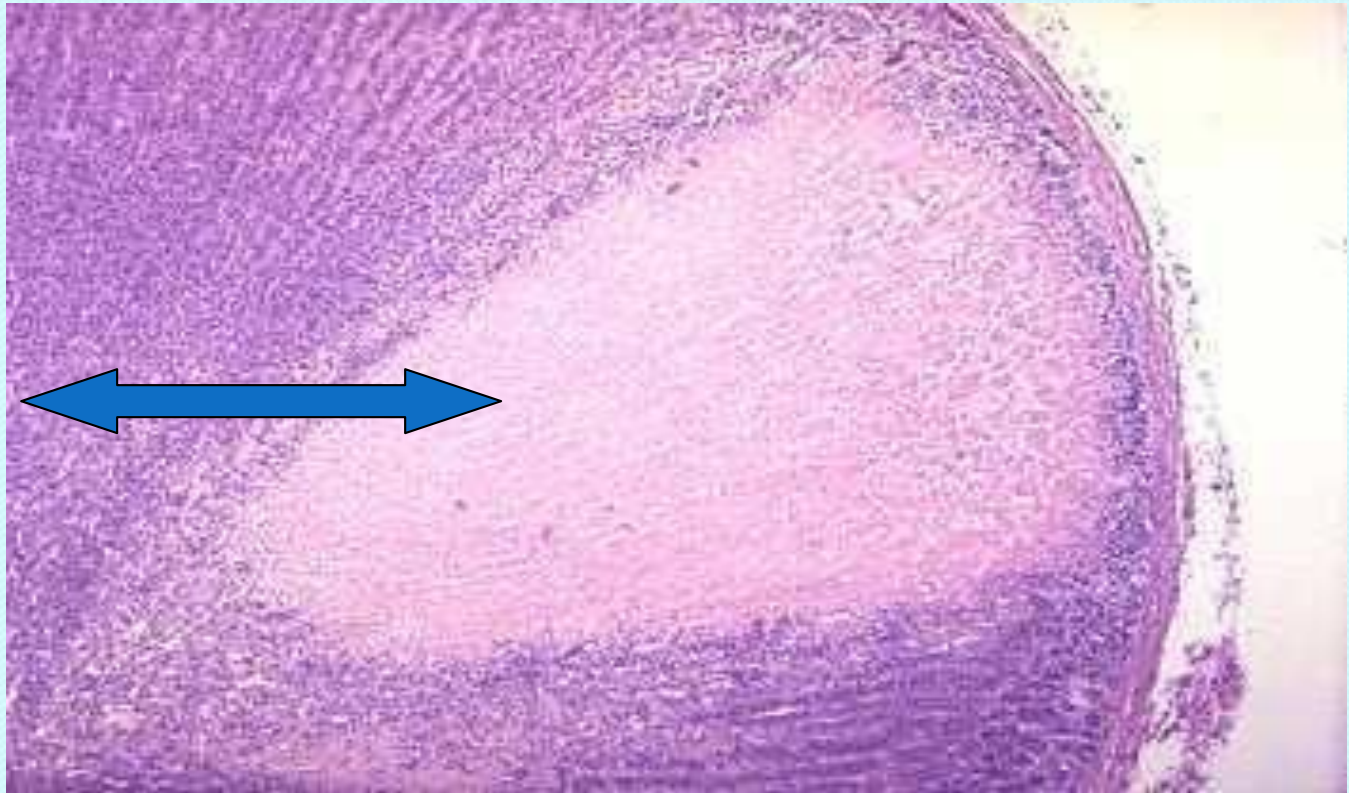
# Morphology of Infarcts

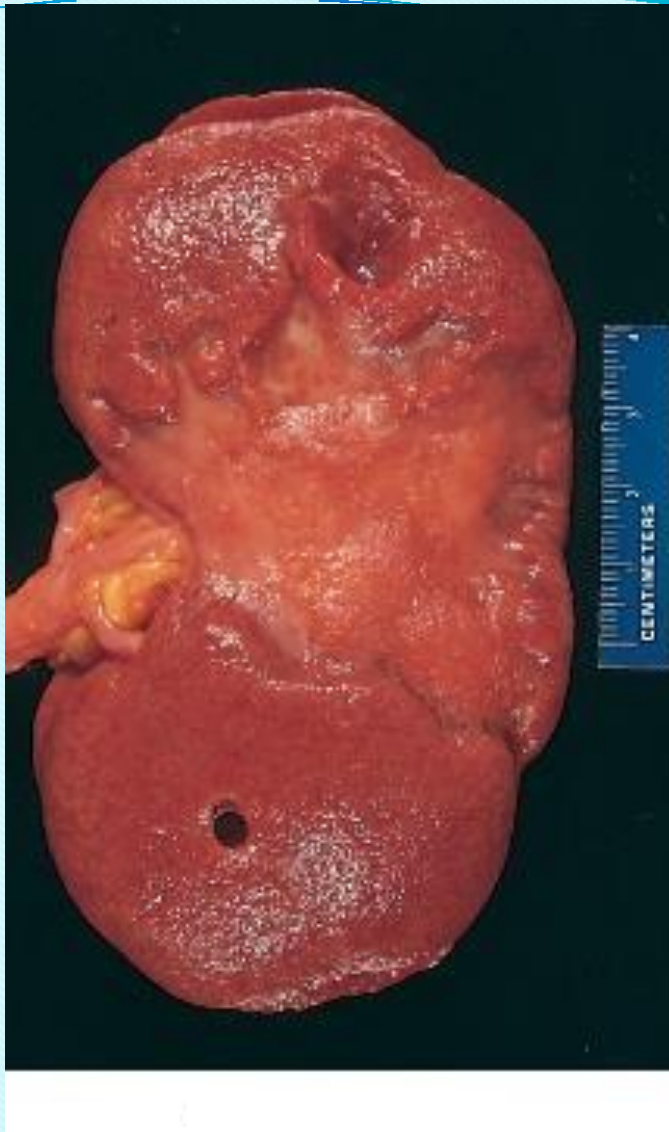
- **●Infarcts can be anemic (white) or hemorrhagic (red)**
- **●White infarcts occur with arterial occlusion of solid organs**
- **●Red infarcts occur with venous occlusion or with arterial occlusions in organs with double or collateral circulation**
- **●White infarcts can become hemorrhagic with reperfusion**

# Renal Infarction



# Renal Infarction

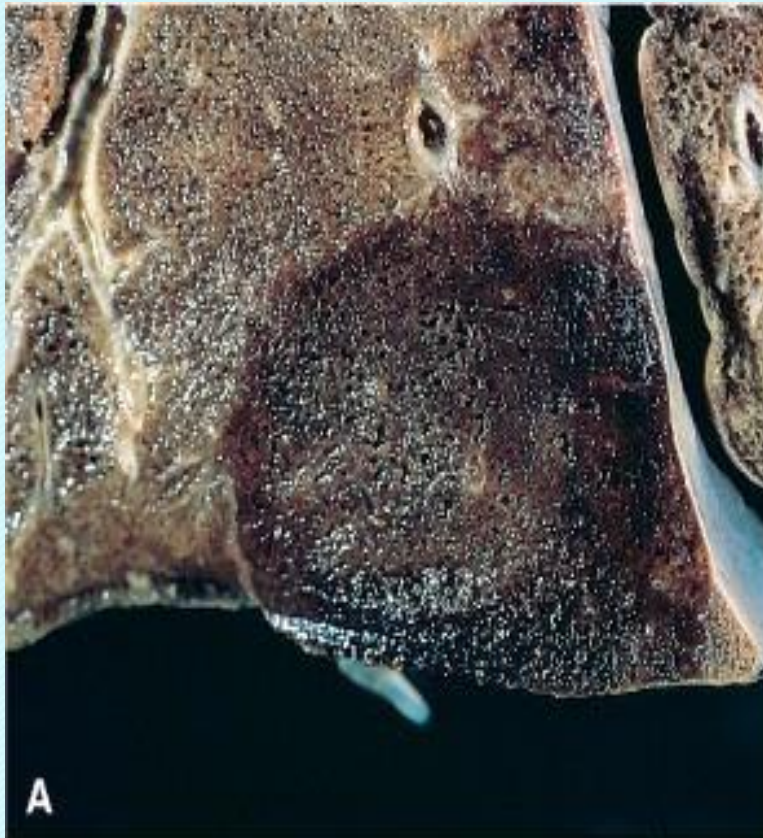




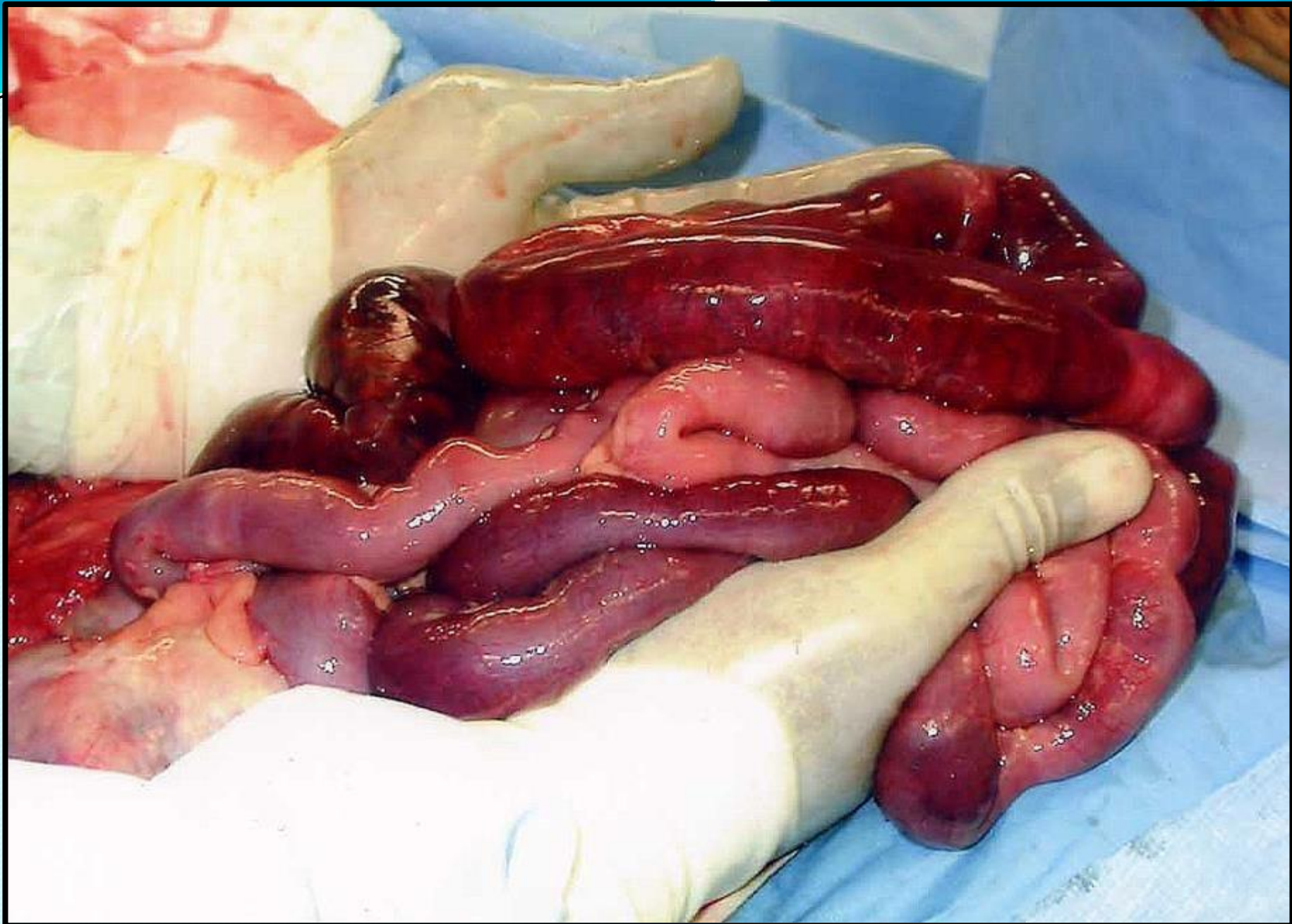
**Remote kidney infarct, now replaced by a large fibrotic cortical scar.**

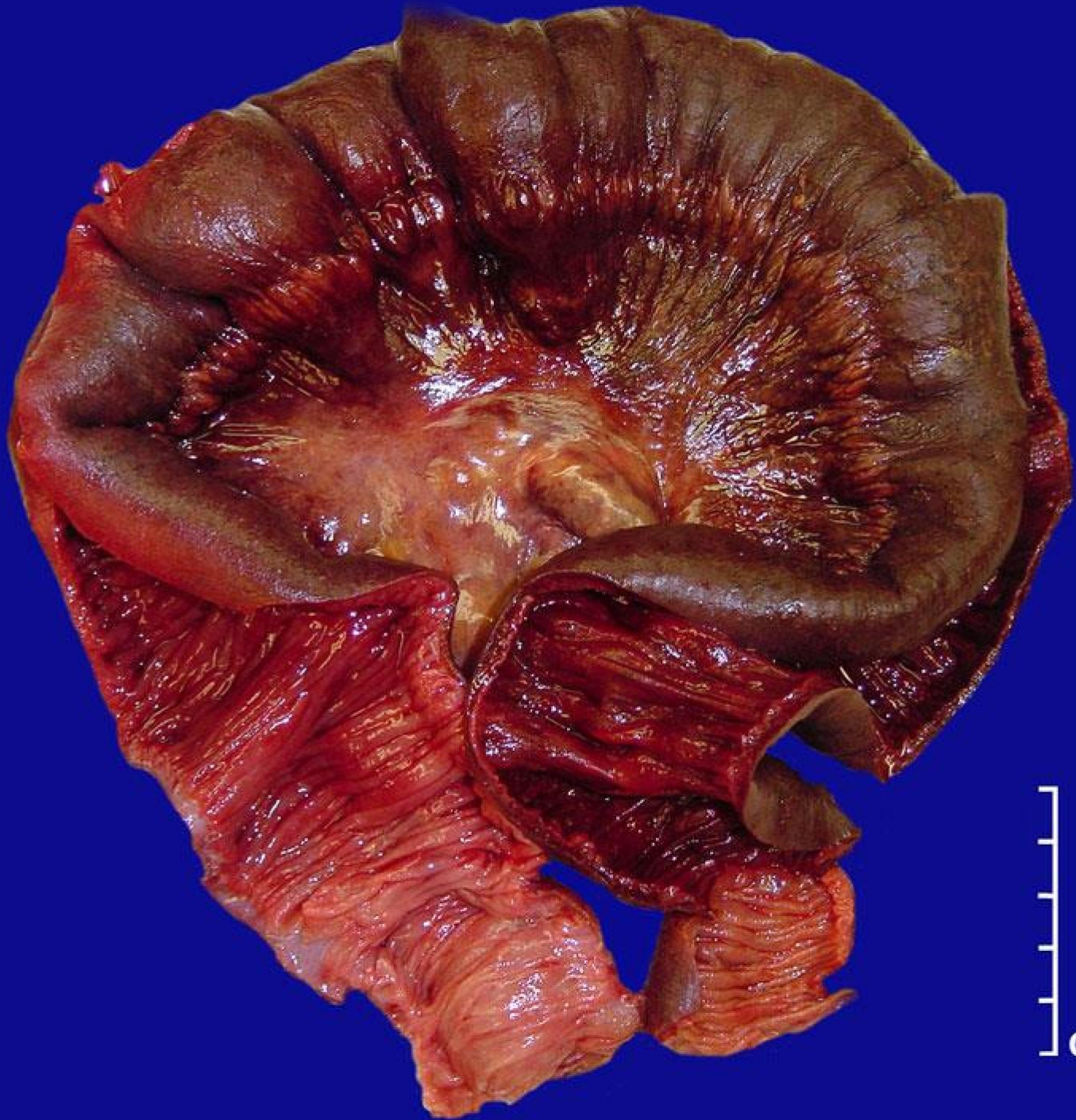
**A, Hemorrhagic, roughly wedge-shaped pulmonary infarct.**

**B, Sharply demarcated white infarct in the spleen.**

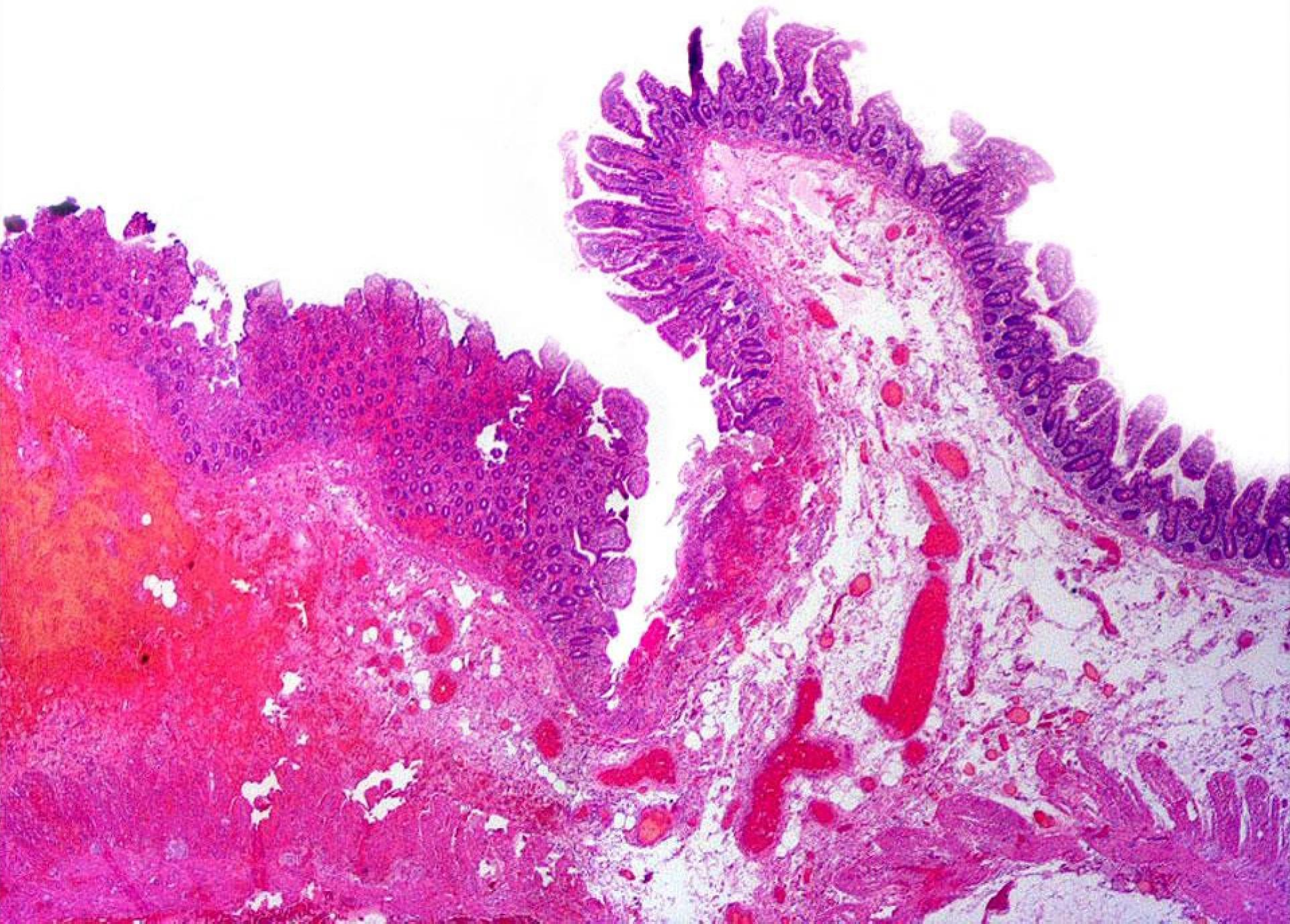


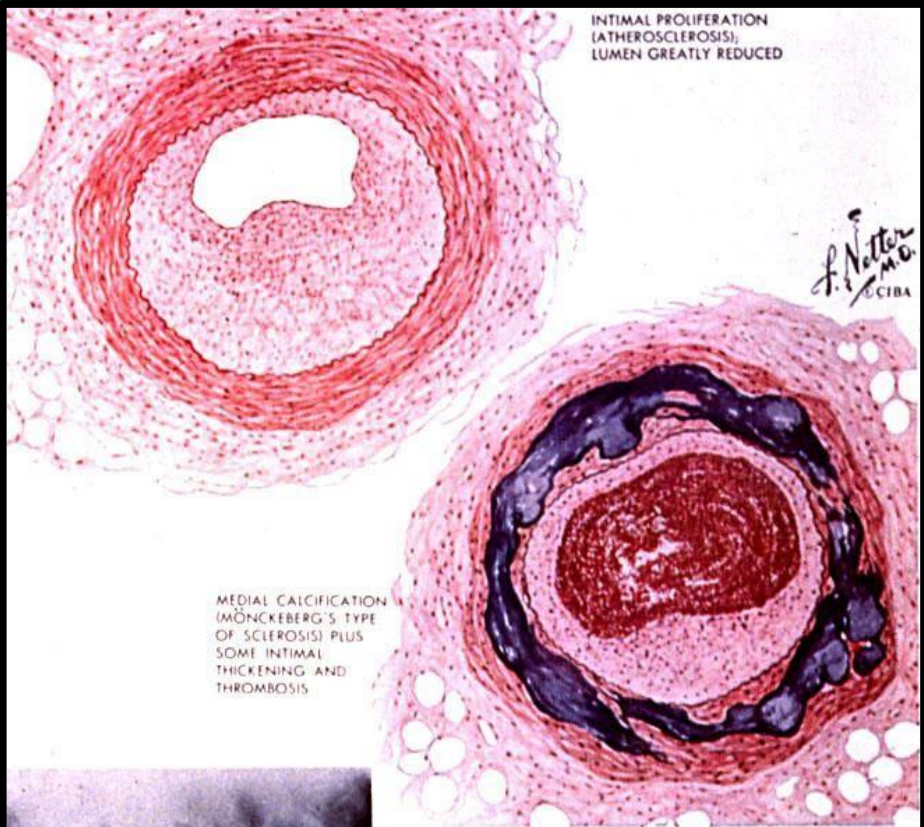






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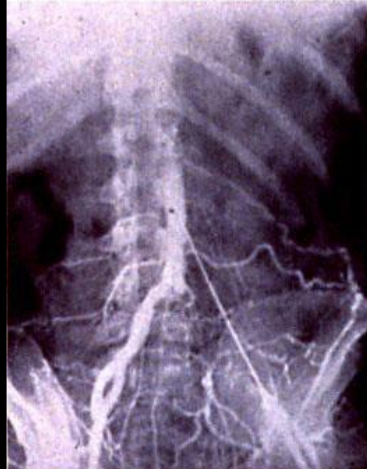




INTIMAL PROLIFERATION (ATHEROSCLEROSIS); LUMEN GREATLY REDUCED

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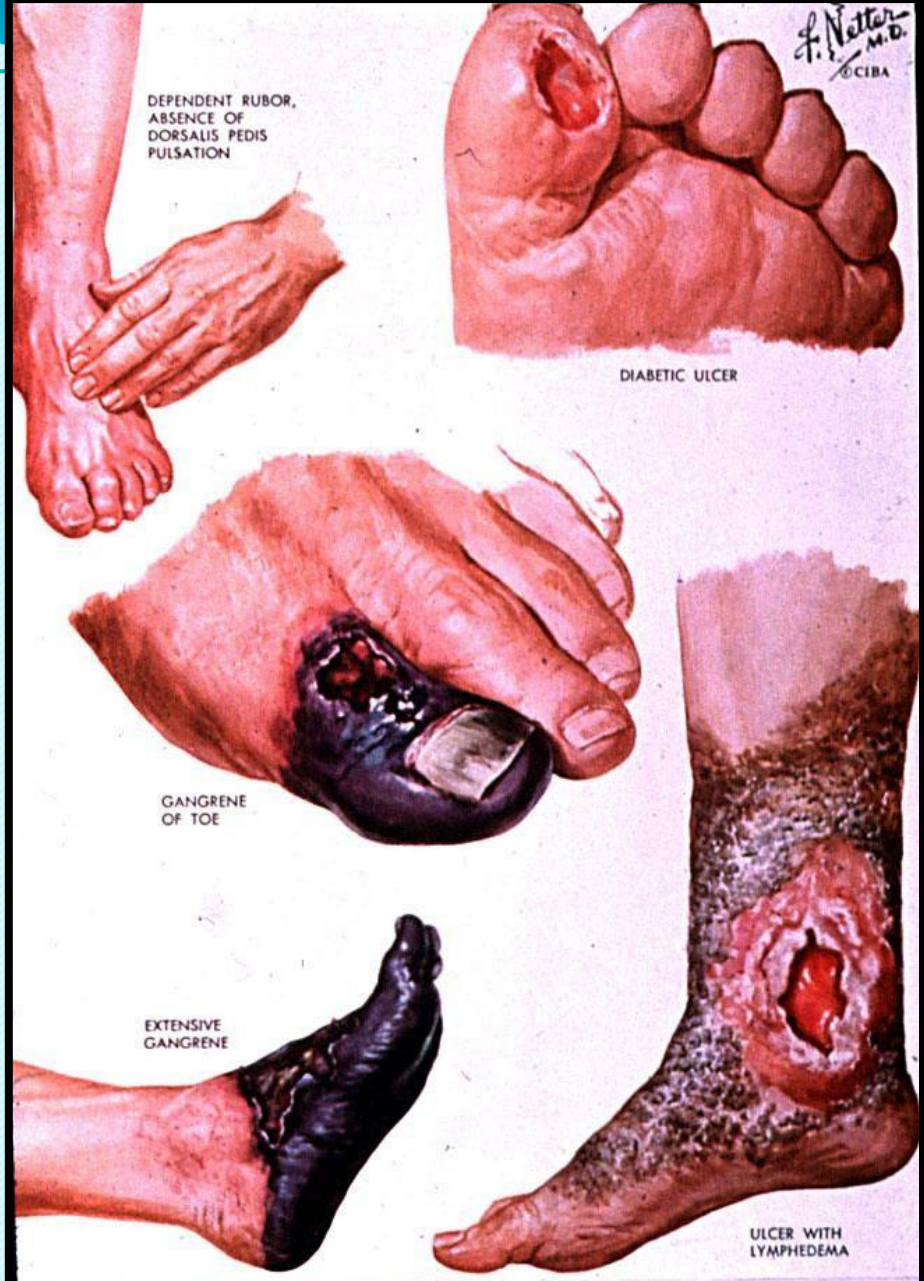
MEDIAL CALCIFICATION (MÖNCKEBERG'S TYPE OF SCLEROSIS) PLUS SOME INTIMAL THICKENING AND THROMBOSIS



AORTOGRAM OBSTRUCTION OF LEFT ILIAC ARTERY IN A DIABETIC



CALCIFIED FEMORAL ARTERY IN A DIABETIC DEMONSTRATED BY X-RAY



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DEPENDENT RUBOR, ABSENCE OF DORSALIS PEDIS PULSATION

DIABETIC ULCER

GANGRENE OF TOE

EXTENSIVE GANGRENE

ULCER WITH LYMPHEDEMA