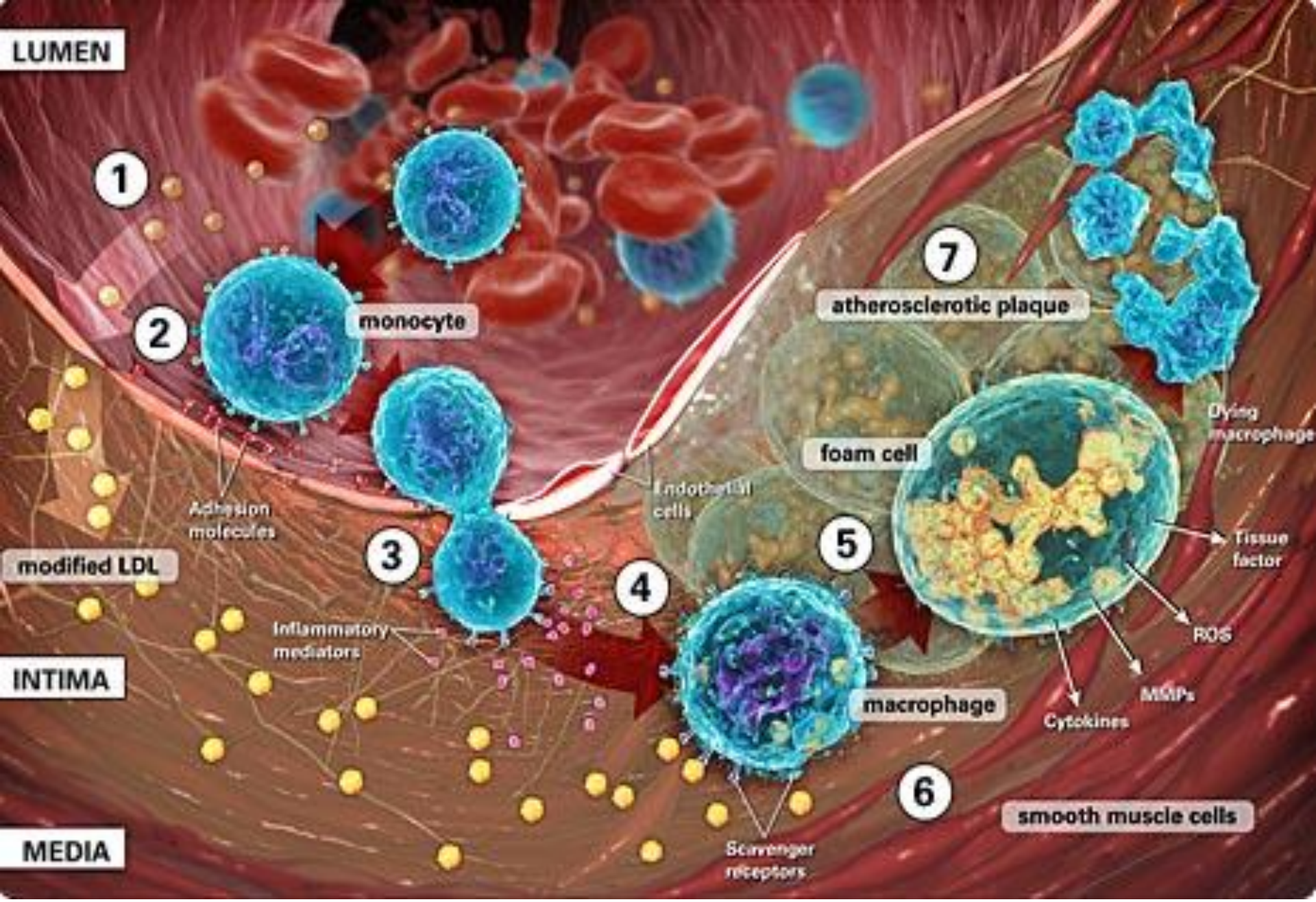


Acute and chronic inflammation.

Acute and chronic inflammation.

I. Microspecimens:

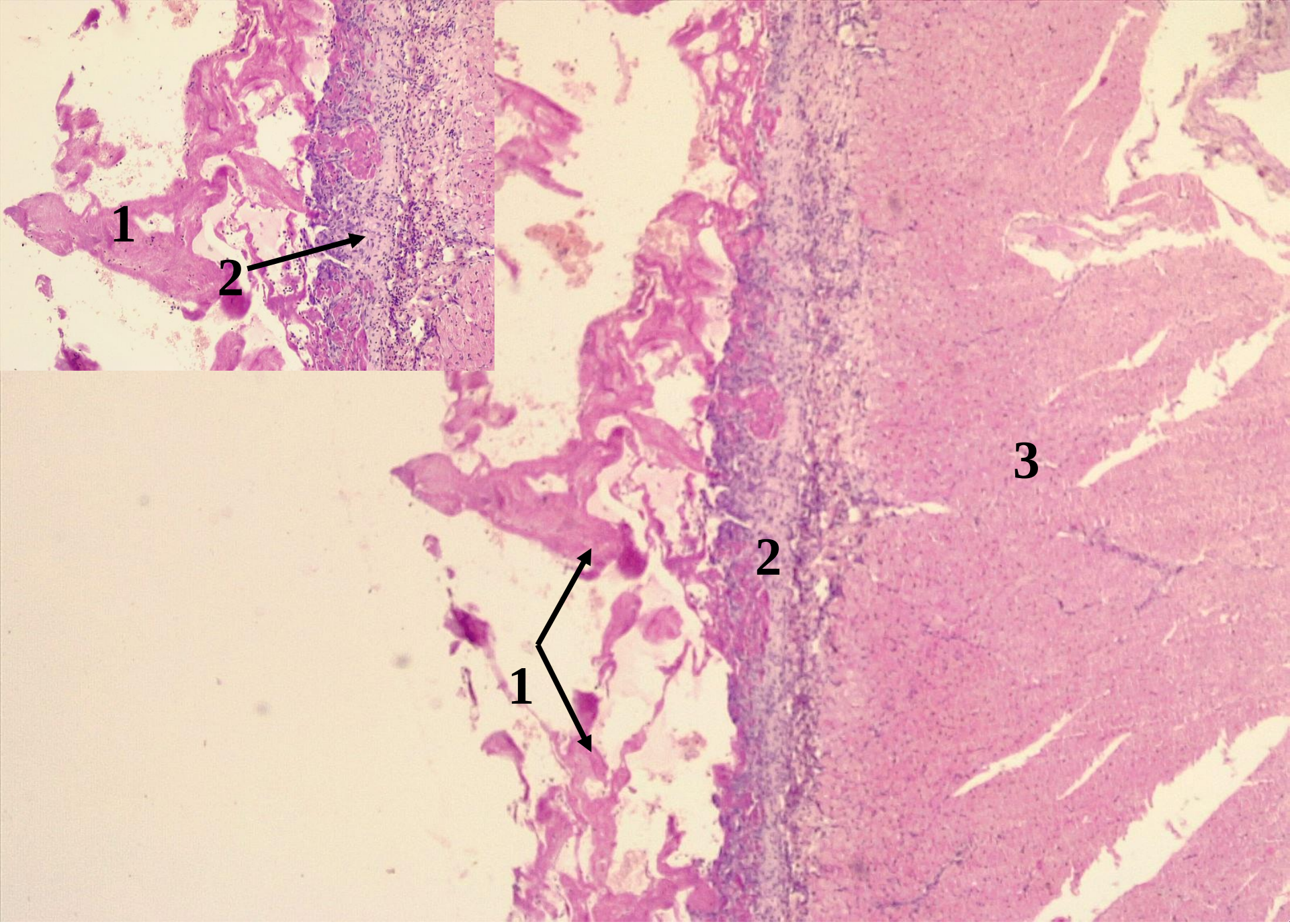
№ 62. Fibrinous pericarditis. *(H-E. stain).*

Indications:

1. Fibrin deposits on the surface of the epicardium.
2. Leukocytic infiltration of epicardium.
3. Myocardium.

On the surface of the epicardium, there are fibrin deposits of eosinophilic color, with irregular appearance, rough due to contractile movements of the heart. In the underlying tissue hyperemia of the vessels, edema, inflammatory infiltrate with neutrophilic leukocytes, lymphocytes and macrophages can be seen. Macroscopically, the heart becomes hairy - villous heart (see macrospecimen number 11).

Fibrinous inflammation occurs in the case of severe tissue injury, leading to marked increase of vascular permeability, which favors extravasation of fibrinogen. The extravasated fibrinogen coagulates into fibrin under the action of thromboplastin, which is removed following tissue necrosis. Fibrin is the predominant component of the exudate. It is most commonly found on mucous and serous membranes, but also in parenchymal organs, eg in the lungs, kidneys. The consequences of fibrinous inflammation can be varied: in some cases complete resorption of the exudate due to the fibrinolytic action of leukocyte enzymes, in other cases fibrin does not resorbed, its organization occurs with the appearance of scars on mucous membranes or adhesions between serous sheets with partial or total obliteration of the cavities (pericardial, pleural, peritoneal) and functional disorders of the respective organs.



№ 62. Fibrinous pericarditis. *(H-E. stain).*

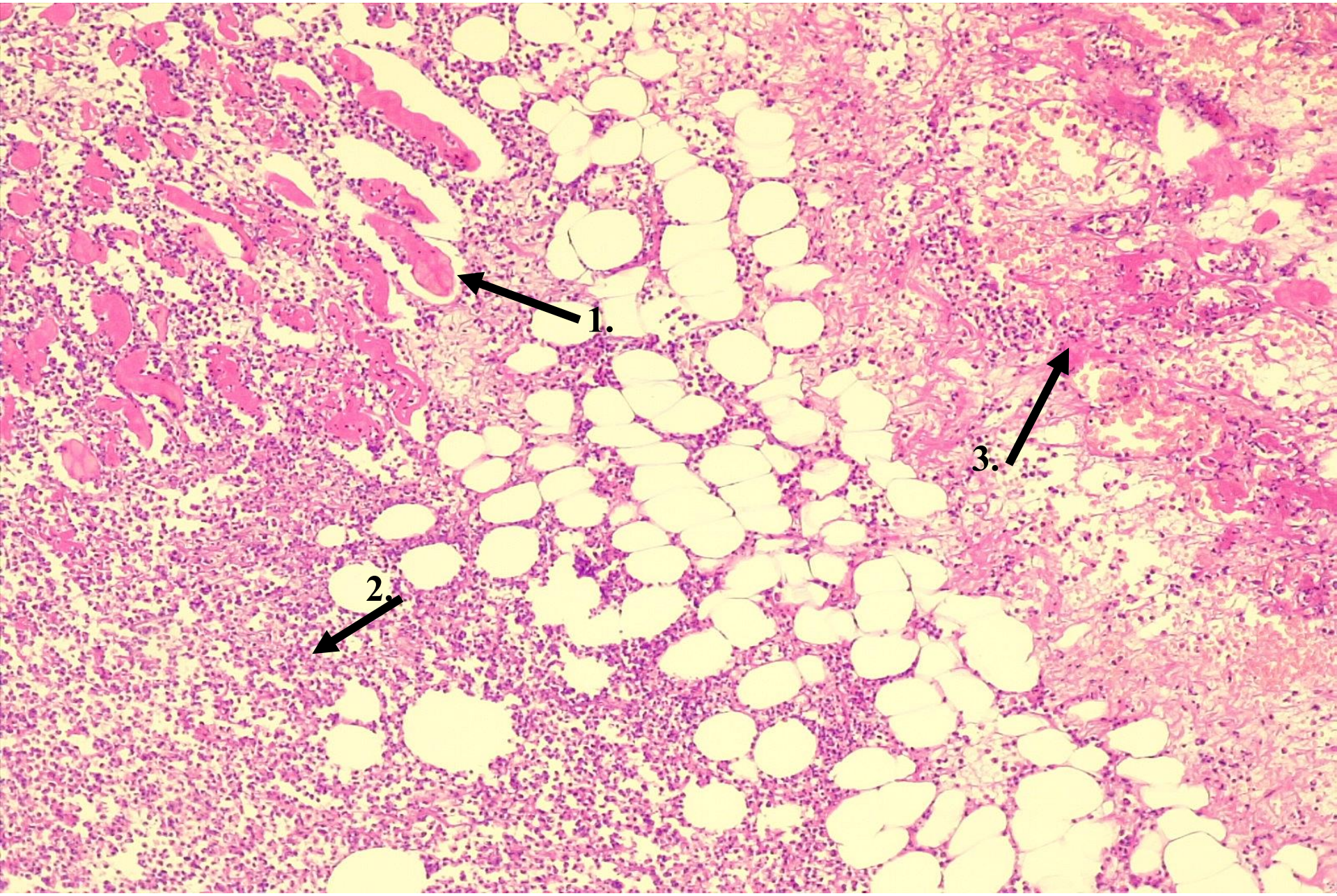
№ 141. Phlegmon of the striated muscle. *(H-E. stain).*

Indications:

1. Necrotic muscle cells (cariolisis).
2. Diffuse infiltration of striated muscle with neutrophil leukocytes.
3. Hemorrhagic foci.

The microspecimen shows diffuse infiltration of striated muscle tissue with neutrophilic leukocytes, edema, dilated blood vessels, hyperemia, hemorrhage; are present necrotic foci of muscle cells with caryolysis.

The phlegmon of the striated muscle is an example of purulent phlegmonous inflammation - inflammation without precise delimitation, in which the exudate spreads diffusely among the tissue elements. The pus spreads along the intermuscular spaces, celluloid tissue, neuro-vascular trunks, etc., the effect of spreading the inflammatory process is favored by substances such as hyaluronidase, which is eliminated by bacteria. It is found in adipose tissue, muscles, walls of cavitary and tubular organs (vermicular appendix, gallbladder, stomach, intestine). The most common causative factors of purulent inflammation are pyogenic microorganisms (which form purulent exudate (pus)), primarily staphylococci. Phlegmon of striated muscle tissue can be seen in septic foci, infected wounds, etc. Consequently, may occur either exudate resorption and complete recovery or fibrosis and sclerosis of the lesion foci.



№ 141. Phlegmon of the striated muscle. *(H-E. stain).*

№ 12. Interstitial myocarditis. *(H-E. stain).*

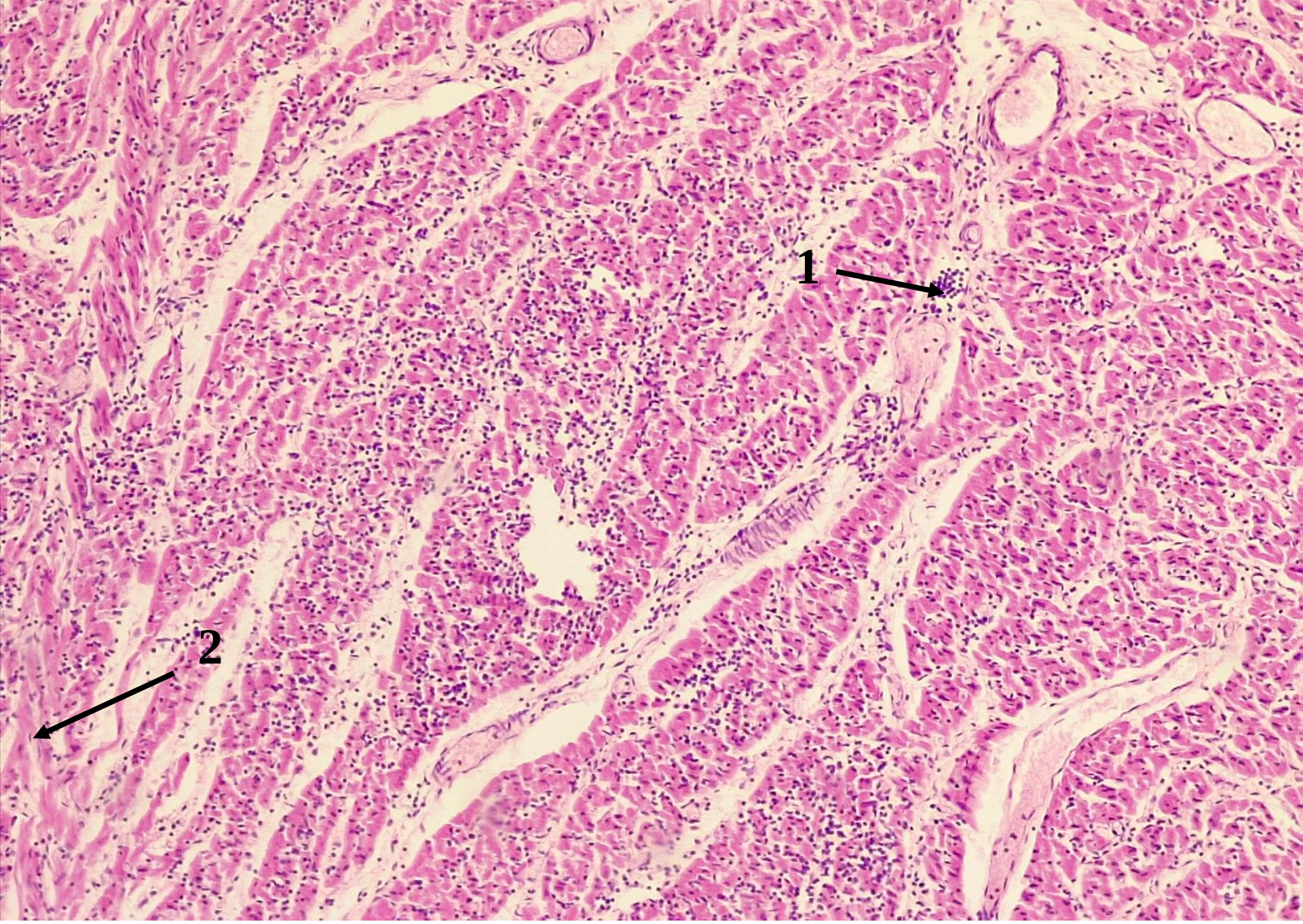
Indications:

1. Infiltration with inflammatory cell of the myocardial stroma.
2. Muscle fibers.

In the interstitial tissue of the myocardium, cellular agglomerations are observed, consisting of lymphocytes, monocytes, macrophages, plasmocytes, fibroblasts. Cellular infiltration is more pronounced around the vessels (perivascular), especially in the subendocardial and subepicardial zones. Degenerative lesions occur in cardiomyocyte sarcoplasm.

It is encountered in viral infections (measles, rubella, influenza), bacterial infection (scarlet fever, exanthematic typhus, meningococcal infection, typhoid fever, brucellosis, septicemia, etc.), fungal and parasitic infections. Clinically it can manifest by signs of heart failure and rhythm disorders. As a consequence of interstitial myocarditis, complete restoration of myocardium or development of cardiosclerosis may occur. Interstitial inflammation is a variant of chronic proliferative inflammation, in which the inflammatory process is localized in the stroma (interstitium) of parenchymal organs. Most common localization: myocardium, kidneys, lungs, liver (name - interstitial - myocarditis, nephritis, pneumonia, hepatitis). The morphological substrate of proliferative inflammation is the inflammatory cell infiltrate.

Consequences of proliferative inflammation: fibrosis - proliferation of connective tissue without organ induration, sclerosis - proliferation of connective tissue, leading to diffuse or local induration of parenchymal organs and cirrhosis - proliferation of connective tissue with pronounced deformation of organs.



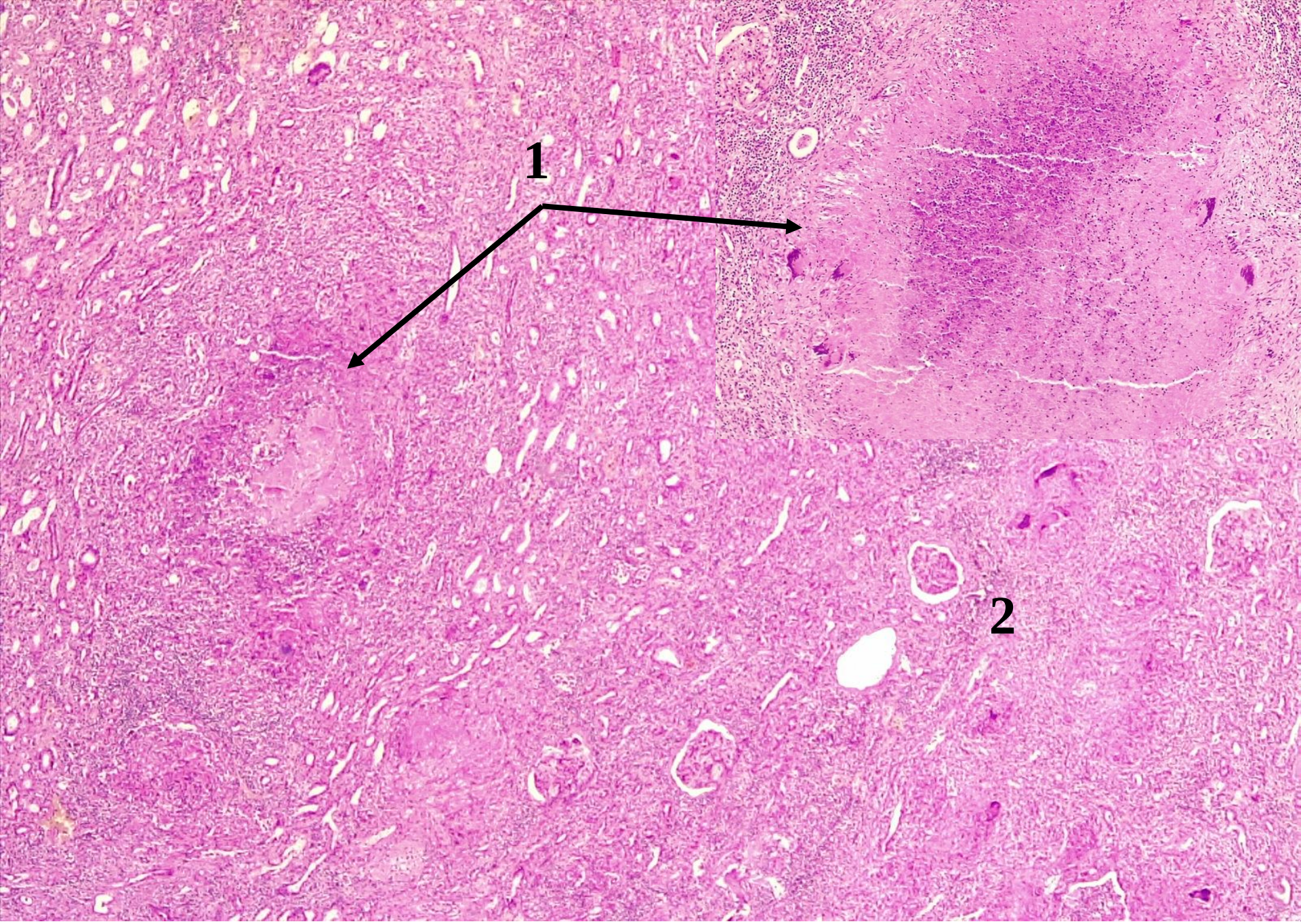
№ 12. Interstitial myocarditis. *(H-E. stain).*

№ 82. Renal milliary tuberculosis. *(H-E. stain).* **Indications:**

1. Tuberculous granuloma:
   a. focus of caseous necrosis in the center of granuloma;
   b. layer of epithelioid cells;
   c. giant cells Langhans;
   d. layer of lymphoid cells .
2. Adjacent renal tissue.

In the renal tissue, tuberculous granulomas are observed that have in the center a zone of eosinophilic, amorphous, structured caseous necrosis witout nuclei, surrounded by a crown of cells arranged from the center to the periphery in the following order: immediately around the necrosis are epithelioid cells, with elongate, pale nuclei, radially disposed (resembling the cells of the spinous layer of the epidermis, hence the name), which are macrophages of monocyte origin, between them, Langhans giant cells can be seen with eosinophilic cytoplasm and nuclei placed as horseshoe or crown. Langhans cells are typical for tuberculosis, in their cytoplasm, Koch bacilli are found. At the periphery of the granuloma is a belt of lymphoid cells (small lymphocytes), including macrophages and plasmocytes. The lack of blood capillaries in the tuberculous granuloma and the persistence of reticulin fibers is characteristic.

Tuberculous nodules can have different sizes, ranging from the size of a millet grain in miliary tuberculosis to larger formations than a few cm. in diameter. The consequences may be different: in cases of favorable evolution (tuberculostatic treatment, high resistance of the organism) resorption, organization, encapsulation or petrification and ossification of the lesion foci may occur, and the unfavorable evolution may be manifested by secondary caseous necrosis and granuloma softening.
Miliary tuberculosis of the kidneys is found in cases of hematogenous dissemination of primary or secondary tuberculosis.



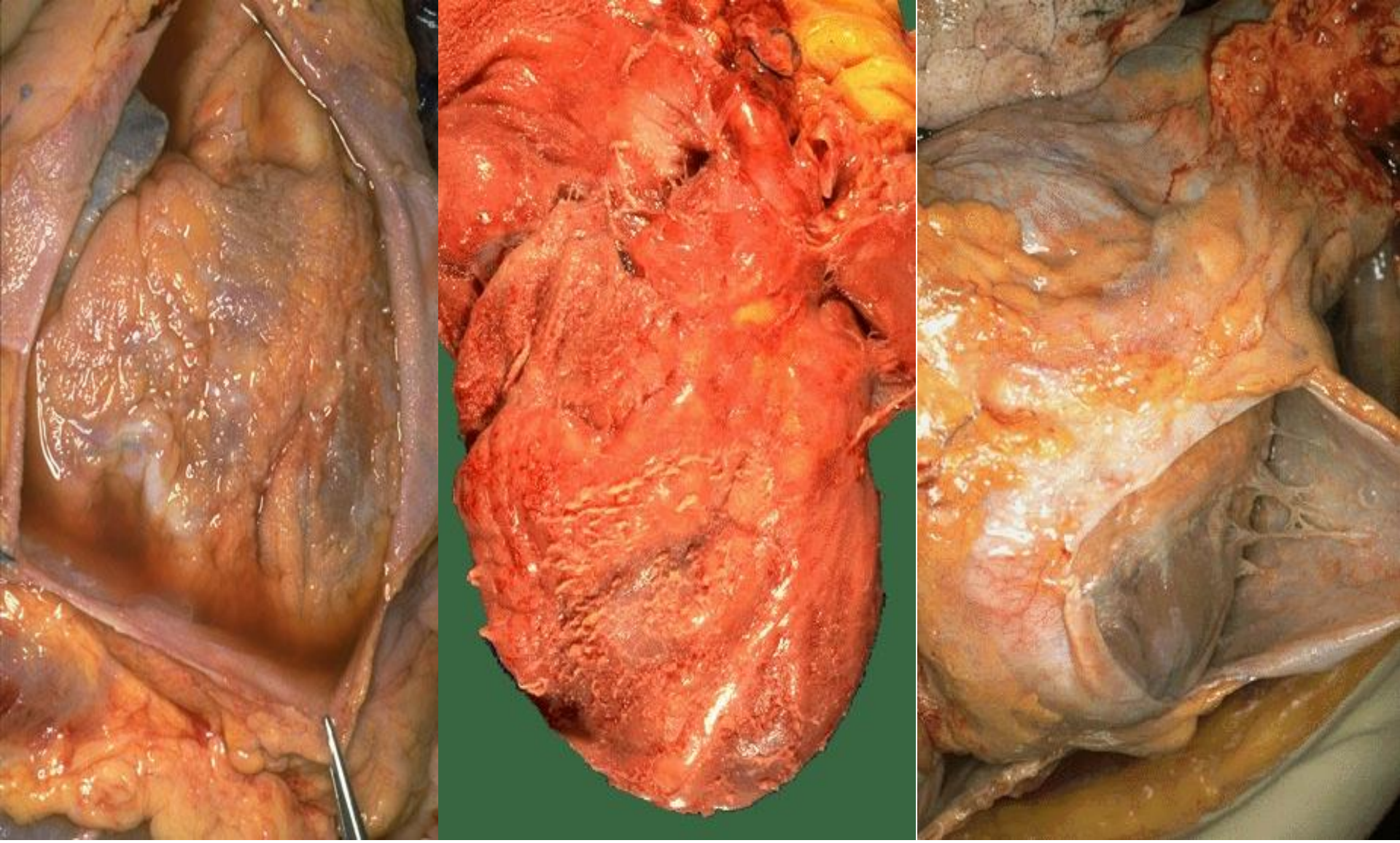
№ 82. Renal milliary tuberculosis. *(H-E. stain).*

II. Macrospecimens:.

№ 11. Fibrinous pericarditis.

The epicardium is opaque, the surface is irregular, covered with yellowish-white deposits of fibrin in the form of villi, which appear due to the contractile movements of the heart. The heart gets a hairy or " tongue of a cat" appearance (villous heart). Fibrin deposits are flaccid and detach slightly (croupous inflammation).

Fibrinous pericarditis is encountered in rheumatic fever, tuberculosis, transmural myocardial infarction, uremia, etc. At auscultation it is manifested by pericardial friction noise. Consequences: resorption of fibrinous exudate due to the fibrinolytic action of leukocyte enzymes or its organization with formation of adhesions between pericardial leaves and obliteration of the pericardial sac. Over time, calcium salts are deposited in the sclerosed pericardium and the "heart in cuirass" appears, which is clinically manifested by progressive chronic heart failure.



№ 11. Fibrinous pericarditis.

№ 33. Lobar pneumonia (grey hepatization stage).

The affected lobe is enlarged in size, not aerated, of firm consistency (similar to the consistency of the liver), the section has a granular appearance, gray color due to the deposit in the alveoli of the fibrinous exudate with a rich content of neutrophilic leukocytes and macrophages; fine deposits of fibrin (parapneumonic fibrinous pleurisy) are observed on the pleura.

The gray hepatization occurs over 4-5 days after the onset of the disease. Subsequently, in the uncomplicated cases, in 8-9 days the lysis of the exudate begins by the fibrinolytic action of leukocytes and macrophages and its elimination by lymphatic drainage and expectoration. Finally, the affected lung is cleansed and the ventilation is restored, which may take 1-3 weeks. Pleural fibrinous exudate is resorbed or organized with the formation of fibrous adhesions between the pleural sheets. In about 3% of cases, alveolar exudate does not liquefy and is replaced by granulation tissue, which is transformed into mature connective tissue (organization) - post-pneumonic fibrosis. Other possible pulmonary complications are pulmonary abscess and pleural empyema. Extrapulmonary complications: purulent pericarditis, mediastinitis, bacterial endocarditis, hematogenous dissemination of infection with development of otitis media, meningitis, brain abscess, purulent arthritis. Complications usually develop in patients with low immunity.

№ 33. Lobar pneumonia (grey hepatization stage).

№ 34. Fibrinous pleuritis.

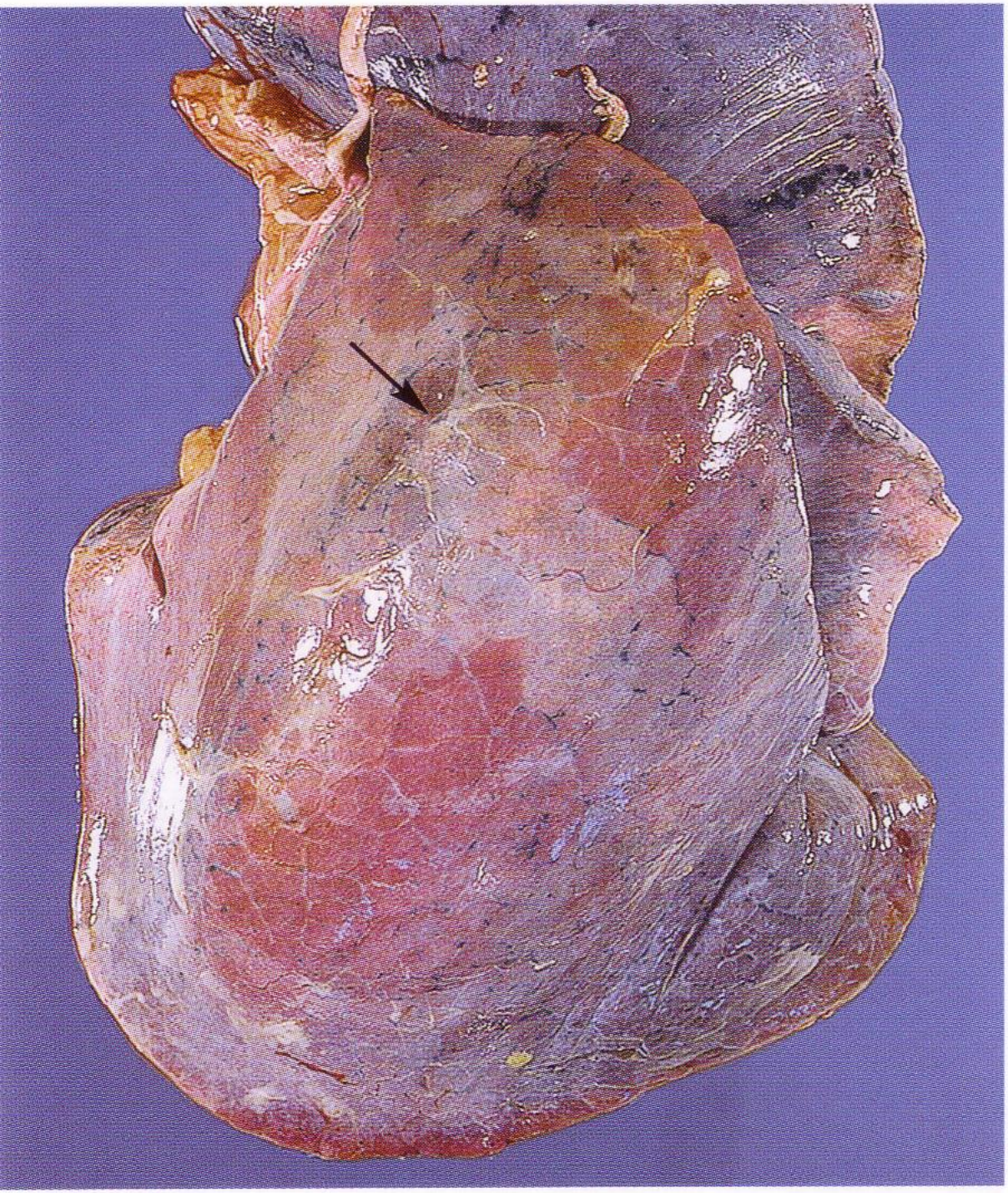
Visceral sheet of the pleura is covered with a fine membrane of whitish fibrin, partially attached to the pleura which gives it a rough appearance. Fibrinous pleuritis manifests at auscultation through pleural friction noise.

It is encountered in tuberculosis, pneumonia, infarction and abscess of lungs, uremia, rheumatoid arthritis, systemic lupus erythematosus. Consequences: resorption of the exudate or fibrous organization with the appearance of adhesions between the pleural sheets with partial or total obliteration of the cavity. The formation of adhesions in the pleura reduces the amplitude of the respiratory movements of the lungs.

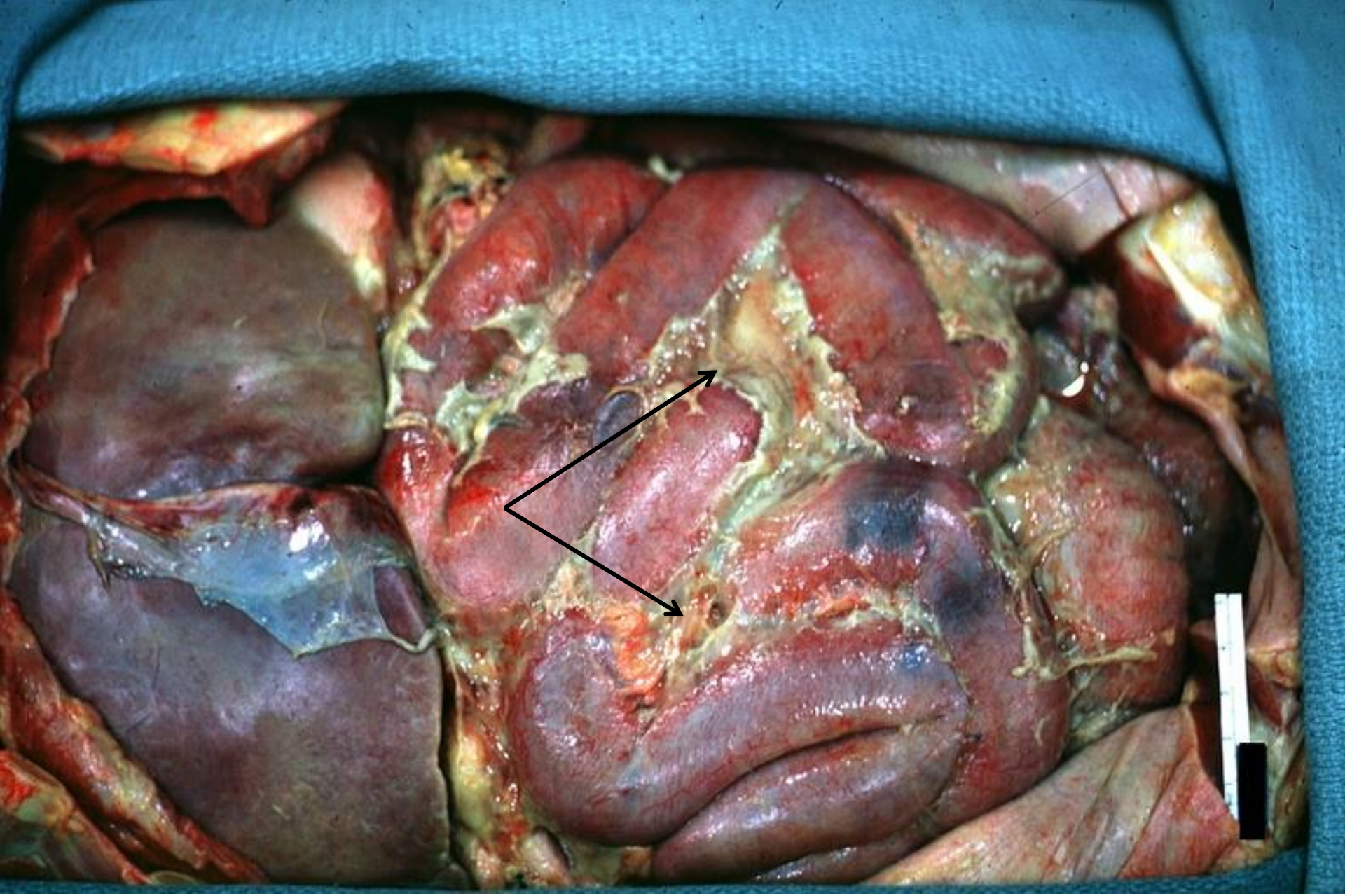
№ 152. Fibrinous peritonitis.

In macrospecimen is a segment of small intestine, the serous membrane has opaque appearance, rough surface, the intestinal loops adhere tightly to each other.

Fibrinous peritonitis can be localized or generalized. It is encountered in appendicitis, cholecystitis, acute pancreatitis, gastric ulcer with perforation, intestinal gangrene, tuberculosis, uremia. Consequences: resorption of fibrinous exudate or its organization with the installation of an adhesive process in the abdominal cavity, which can be complicated by intestinal occlusion.



№ 34. Fibrinous pleuritis.



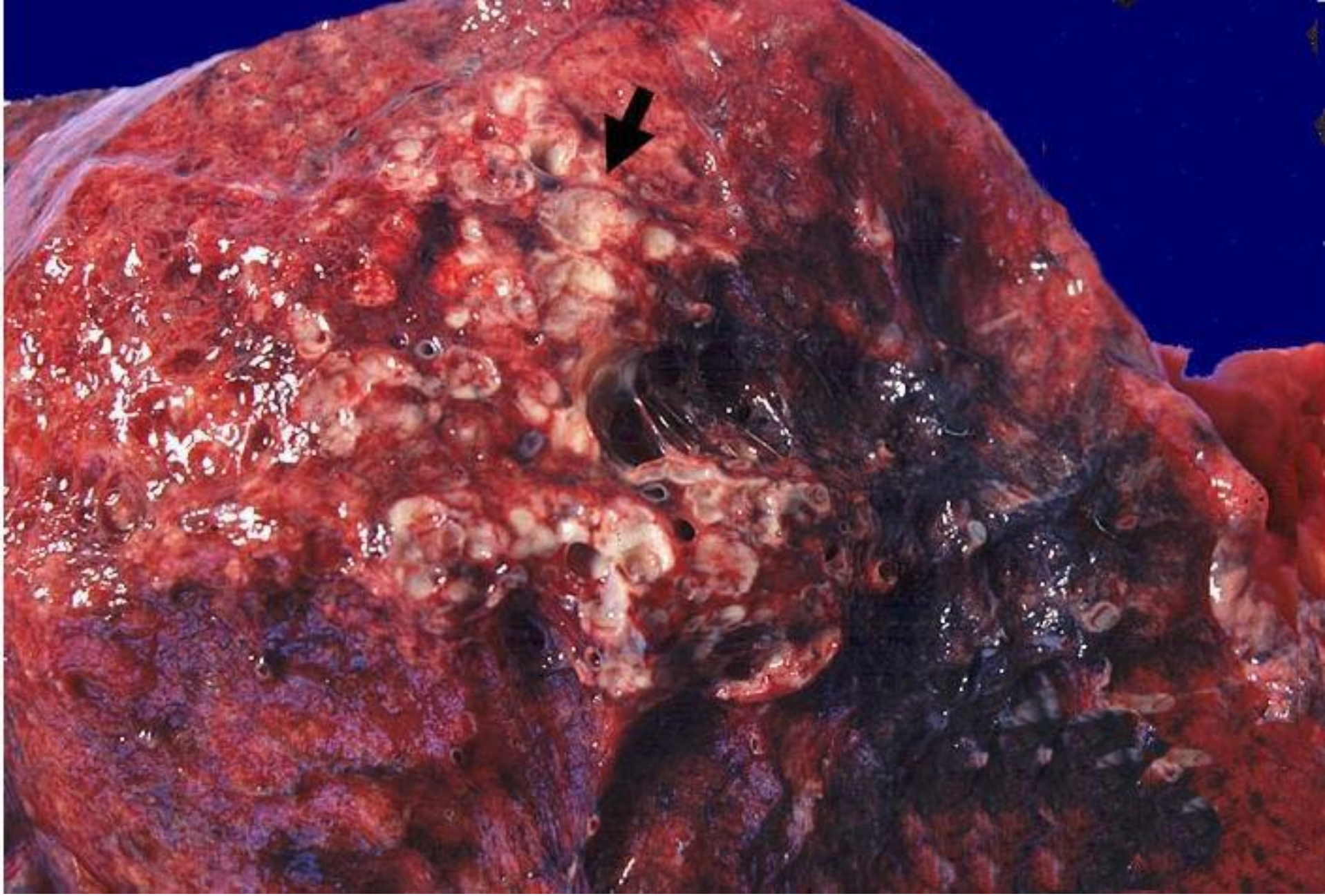
№ 152. Fibrinous peritonitis.

№ 32. Focal pneumonia with abscess formation.

On the section of the lung, there are multiple spread, non-aerated foci of pneumonia which have whitish-gray color and 2-3 cm in diameter, slightly raised, separated by intact lung tissue. In some of these foci there are irregularly shaped cavities, ranging in size from 0.5 to 1-1.5 cm, filled with pus or without content - abscesses. On pleura, fibrin deposits may be seen in case of subpleural localization of pneumonia.

Abscess appears as a result of necrosis, destruction and lysis of the necrotic tissue. The necrosis is due both to the direct injurious action on the tissues of the toxins of the pyogenic bacteria, as well as to the circulatory disorders related to the thrombosis of the vessels and their compression by the inflammatory edema. Histolysis (proteolysis) is produced by proteolytic enzymes eliminated by neutrophil leukocytes. Following the lysis of the altered and necrotic tissues, a viscous, semi-liquid mass of yellow color appears - pus.

Abscess is one of the pulmonary complications of pneumonia, primarily bronchopneumonia or focal pneumonia. Bronchopneumonia is the most common form of pneumonia, which begins with the initial inflammation of the bronchi and bronchioles with subsequent expansion into the adjacent alveoli (bronchoalveolitis).
Bronchopneumonia with abscess formation is usually caused by staphylococci and streptococci. It is most commonly seen in patients with different concomitant conditions, eg congestive heart failure, chronic lung disease, diabetes, immunodeficiency, especially in the elderly. Consequences of acute pulmonary abscess: organization, calcification, chronic evolution (chronic abscess).



№ 32. Focal pneumonia with abscess formation.

№ 12. Difuse cardiosclerosis.

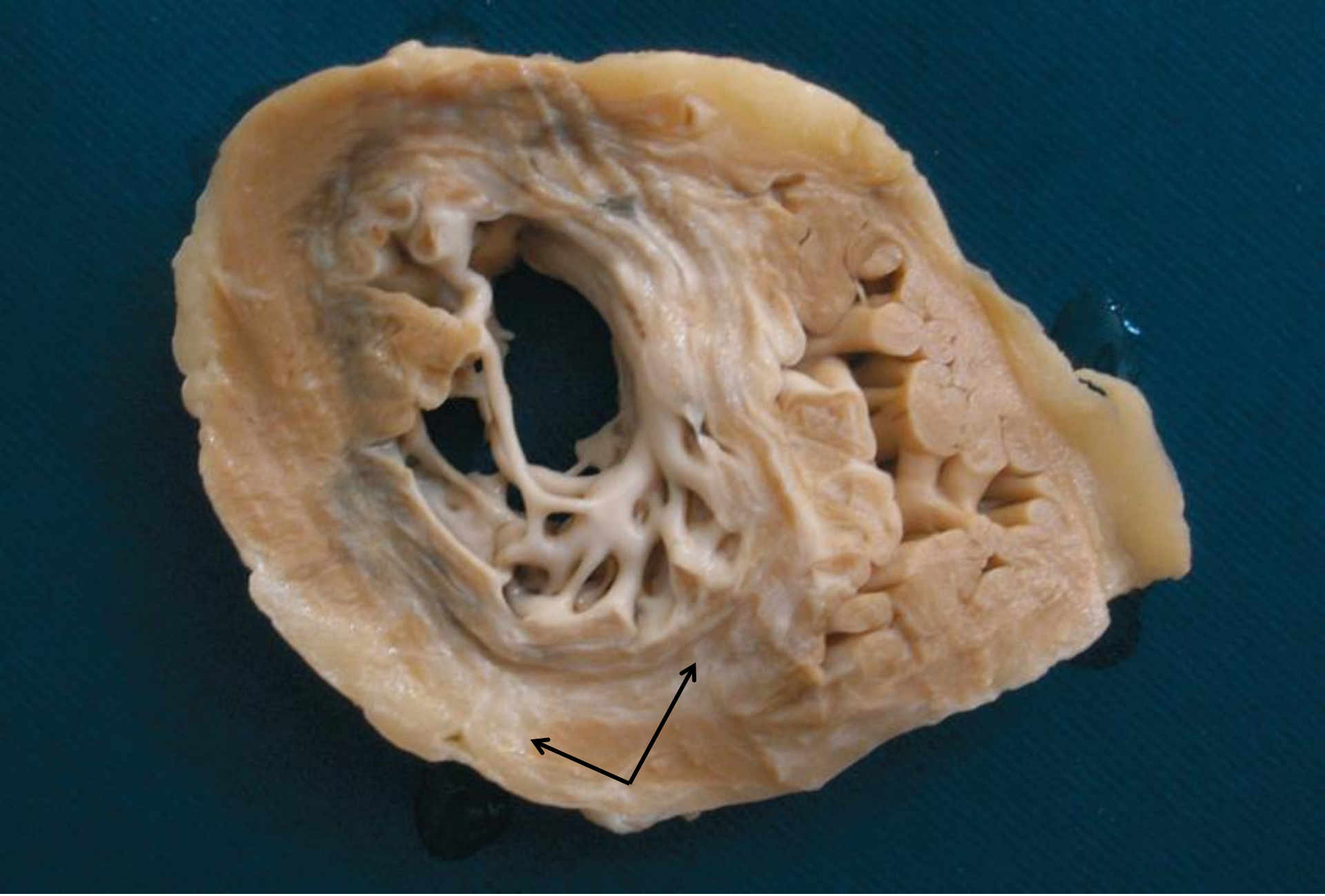
On myocardial section of the left ventricular wall, multiple thin bands of whitish fibrous conjunctive tissue are observed.

Diffuse cardiosclerosis is a process of diffuse excessive proliferation of connective tissue in the heart wall. It may be a consequence of interstitial myocarditis, eg, in rheumatic fever, diphtheria, influenza, measles, sepsis. It is also encountered in chronic ischemic heart disease, caused by stenosing atherosclerosis of coronary arteries. Possible complications: congestive heart failure, heart and rhythm disorders.

№ 51. Gastric polyp.

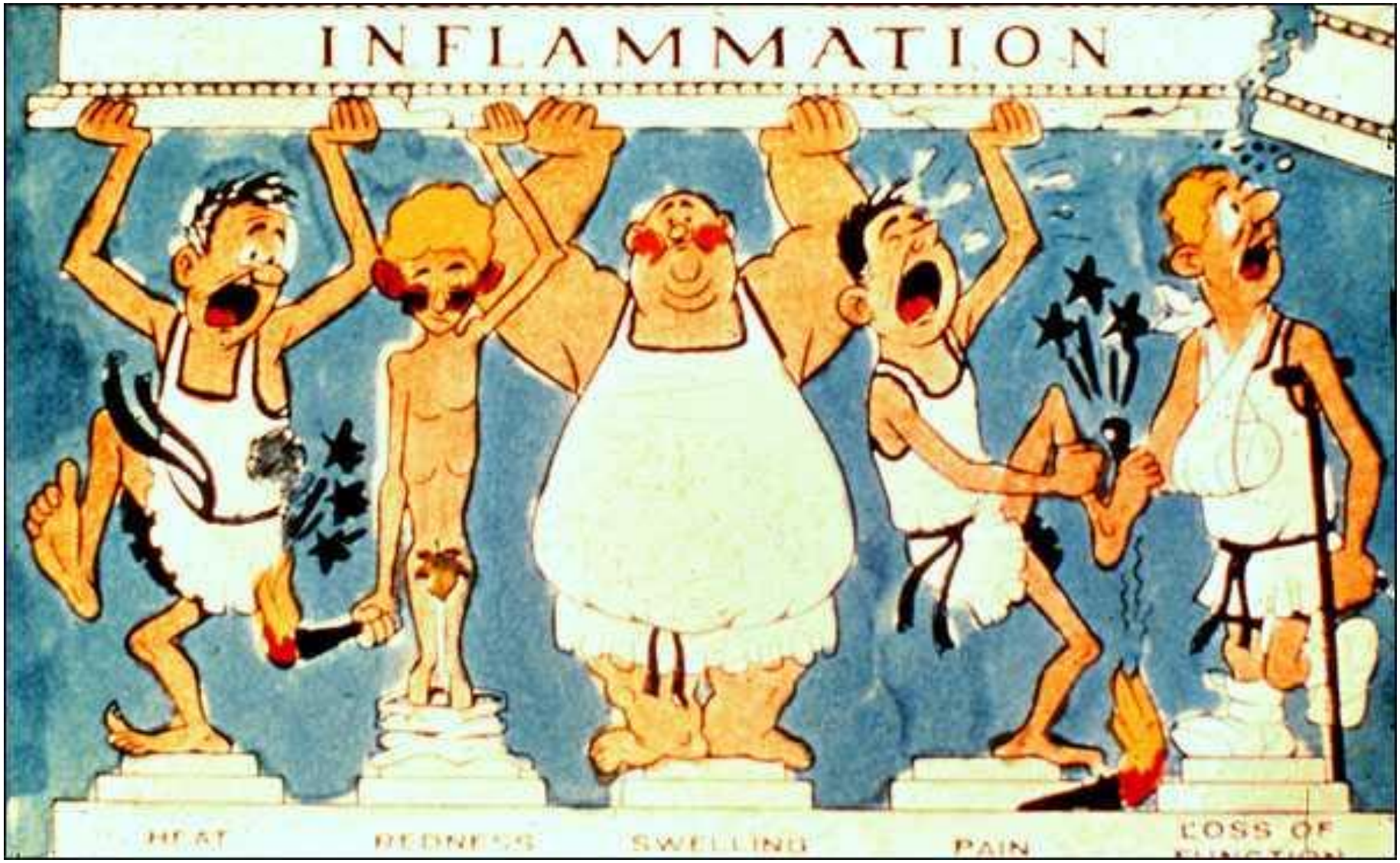
On the surface of the gastric mucosa there are multiple prominent formations, with thin base (peduncled polyps) or wide (sessile polyps), dimensions from a few mm to 1-1.5 cm, oval shape, smooth surface, flaccid consistency, in some cases hemorrhagic foci can be seen.

Gastric polyps are more frequently located in the anthro-pyloric region, they can be solitary or multiple. The absolute majority of gastric polyps (≈90%) are of inflammatory origin, non-neoplastic (hyperplastic polyps). Microscopically they consist of hyperplastic glands, irregularly arranged, some cystically dilated and elongated; are covered with superficial gastric epithelium, but can also parietal and main cells can be noted, the stroma is edematous, hyperemic, with moderate lympho-histiocytic infiltration. No cellular atypia is observed and, as a rule, it has no malignant potential. However, in polyps larger than 1.5 cm there is a risk of gastric epithelial dysplasia, which is a premalignant lesion. Hyperplastic polyps can be complicated by superficial erosions and gastric hemorrhage.



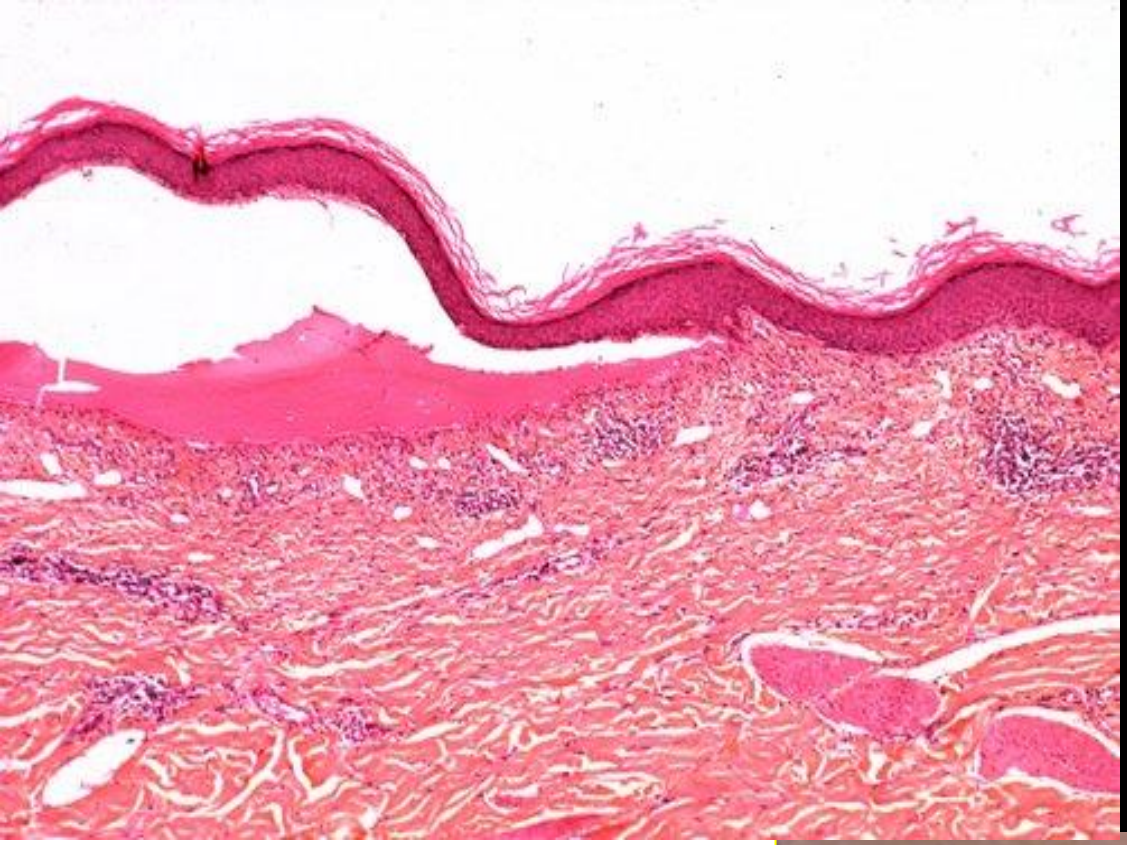
№ 12. Difuse cardiosclerosis.

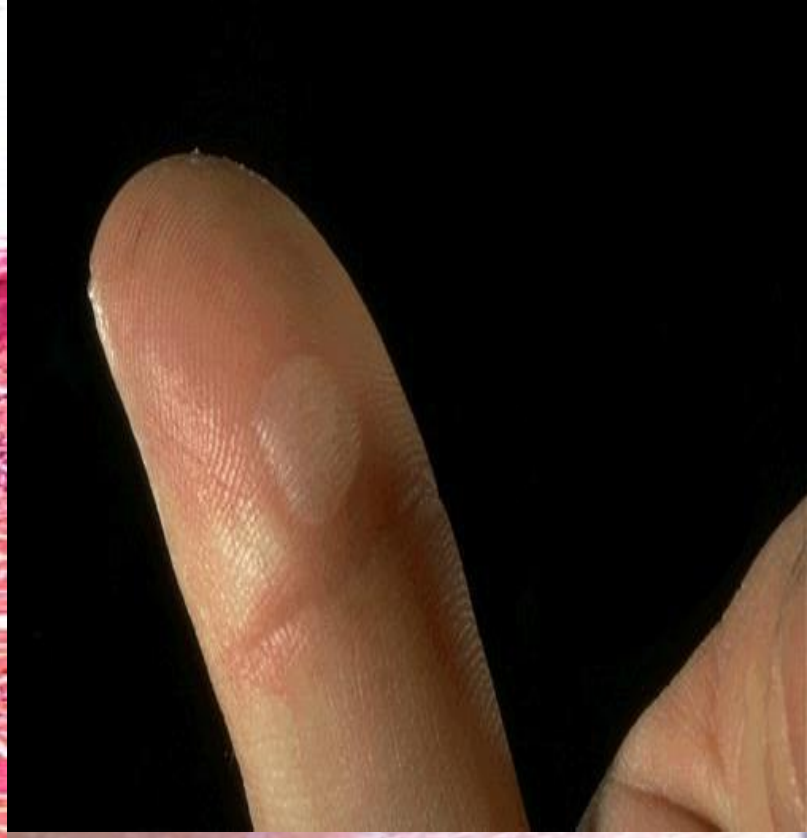
№ 51. Gastric polyp.



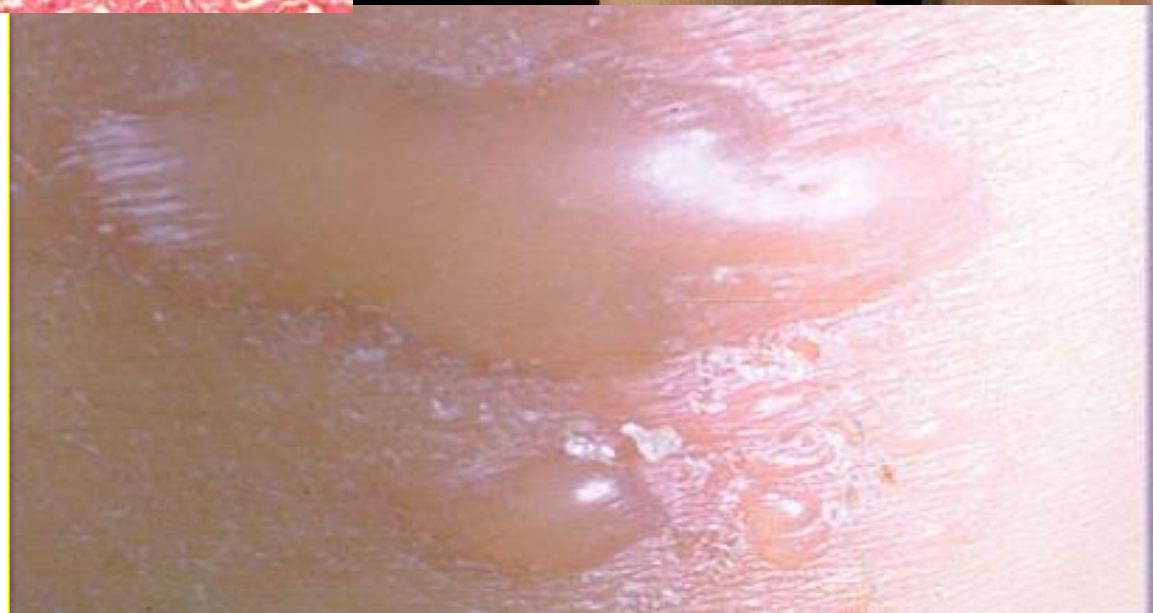
Clinical signs of inflammation

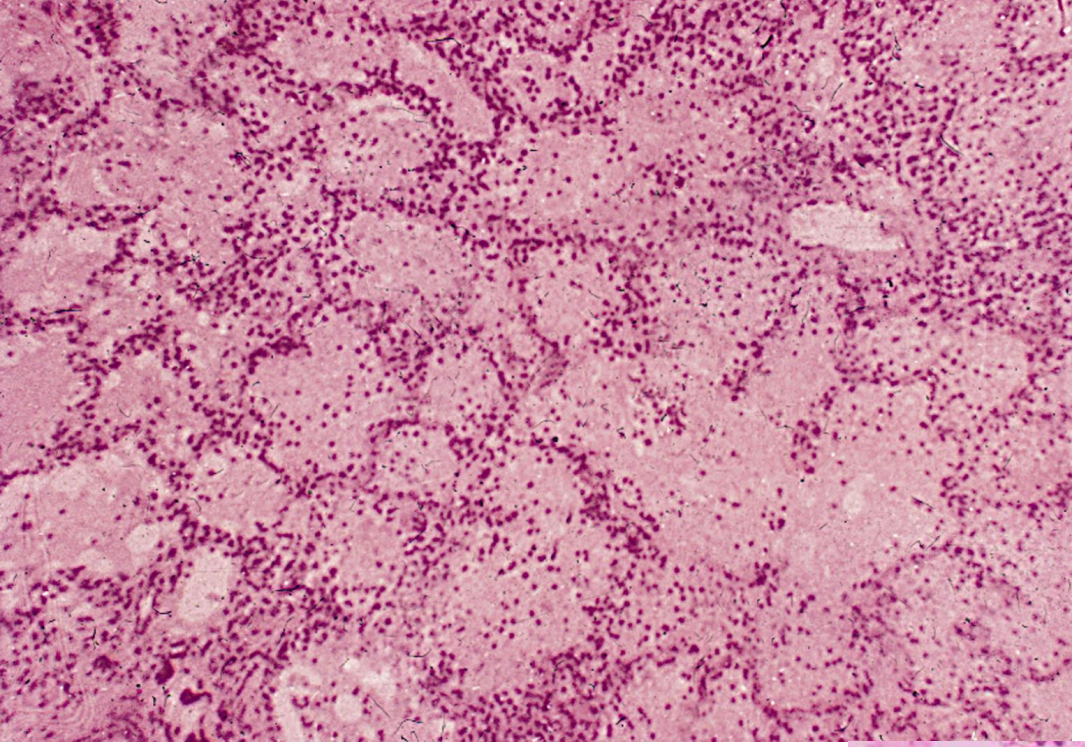
rubor (redness), calor (increased heat), tumor (swelling), dolor (pain), and functio laesa (loss of function)



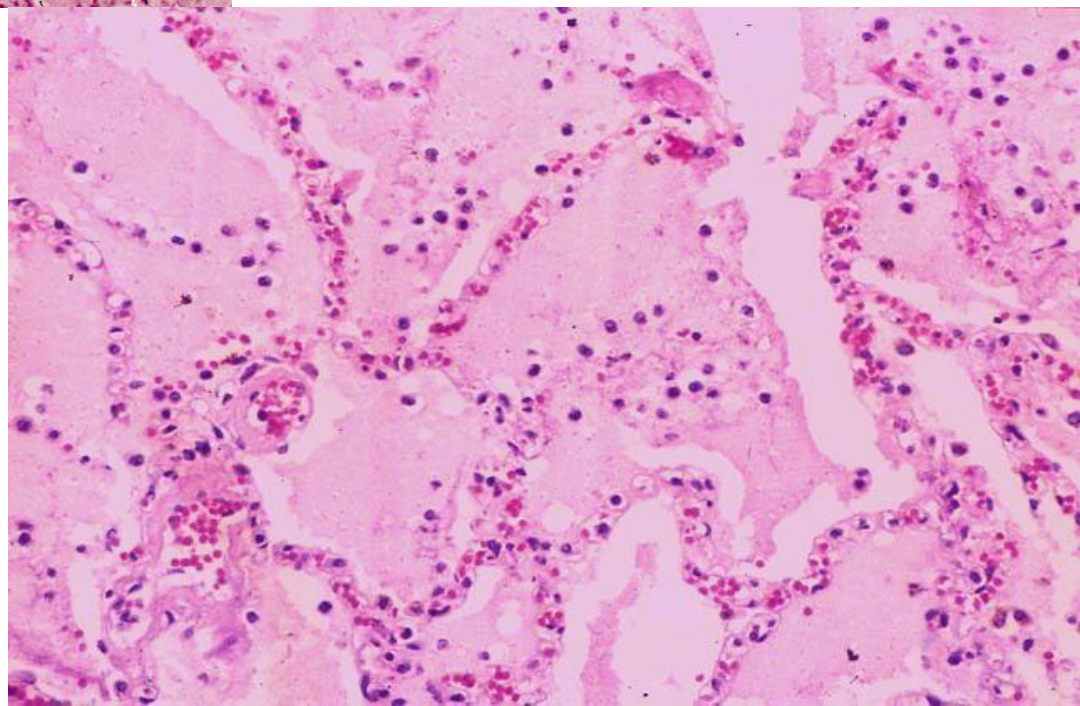


Epidermal vesicle with serous exudate.

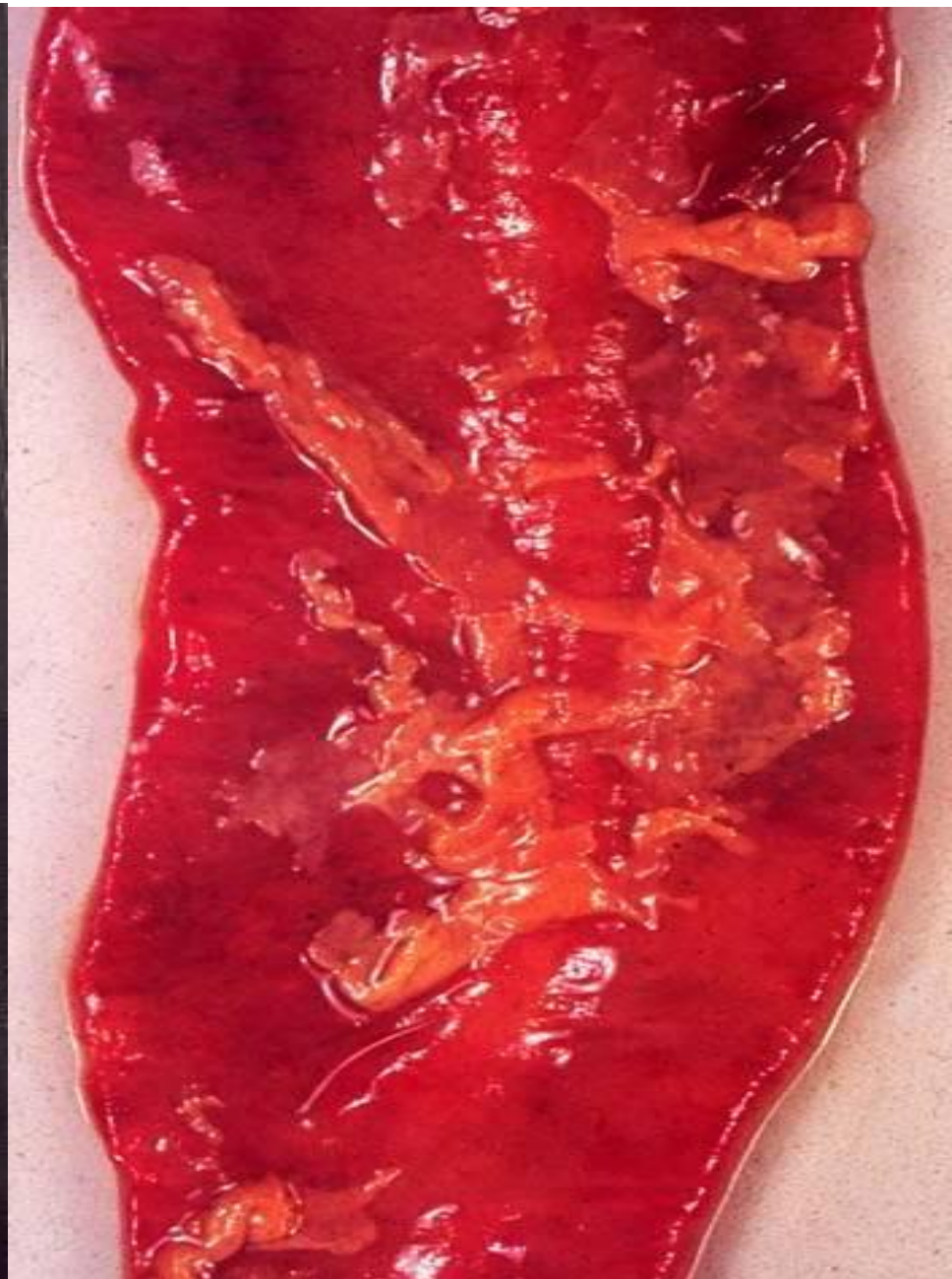




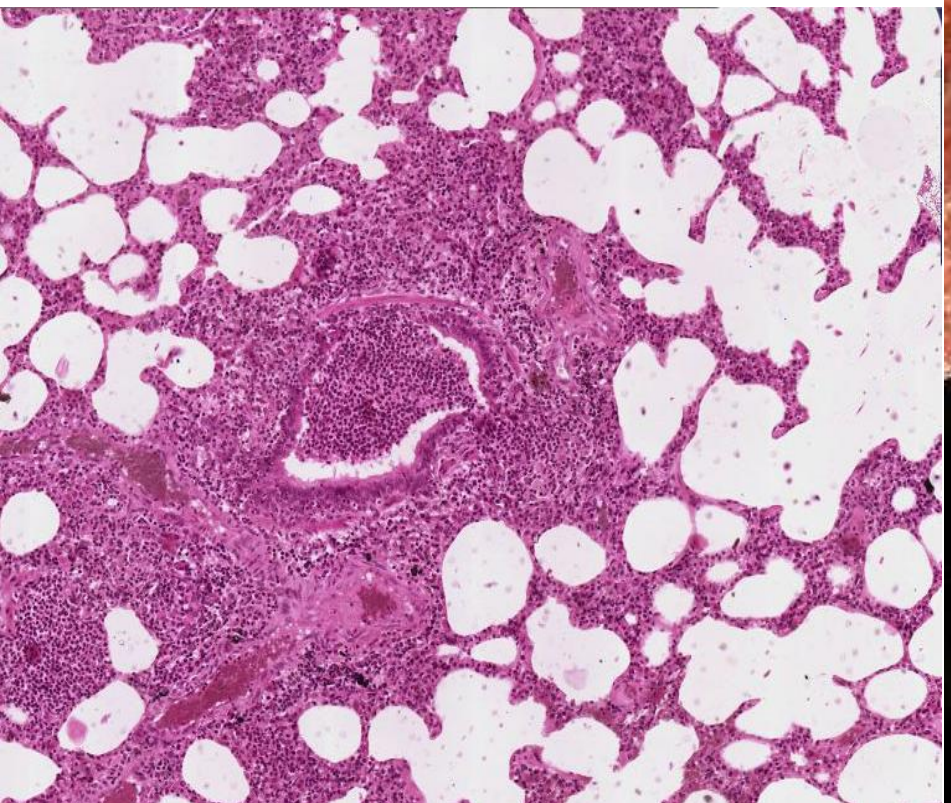
Serous focal pneumonia.

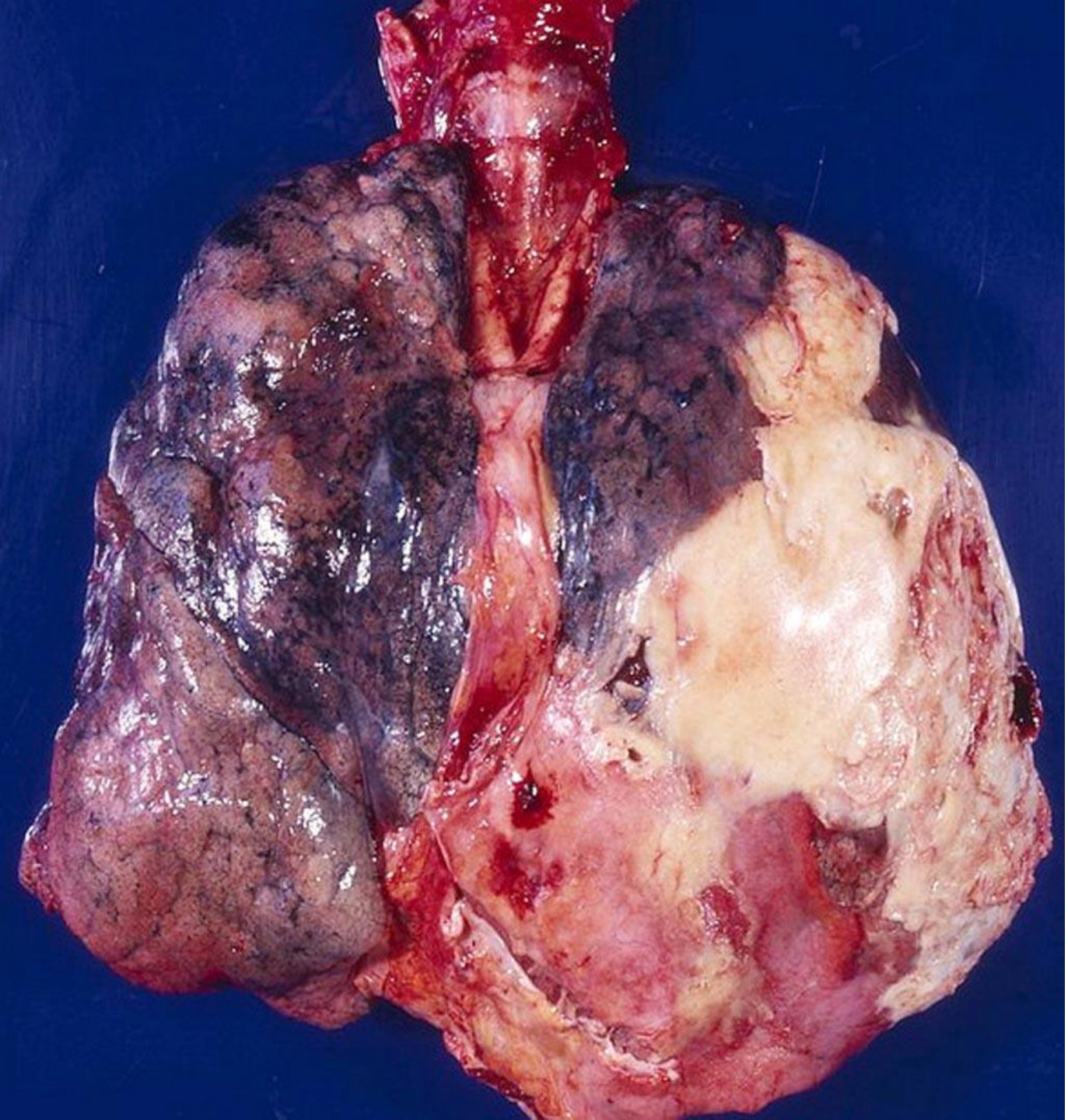


Fibrinous inflammation.



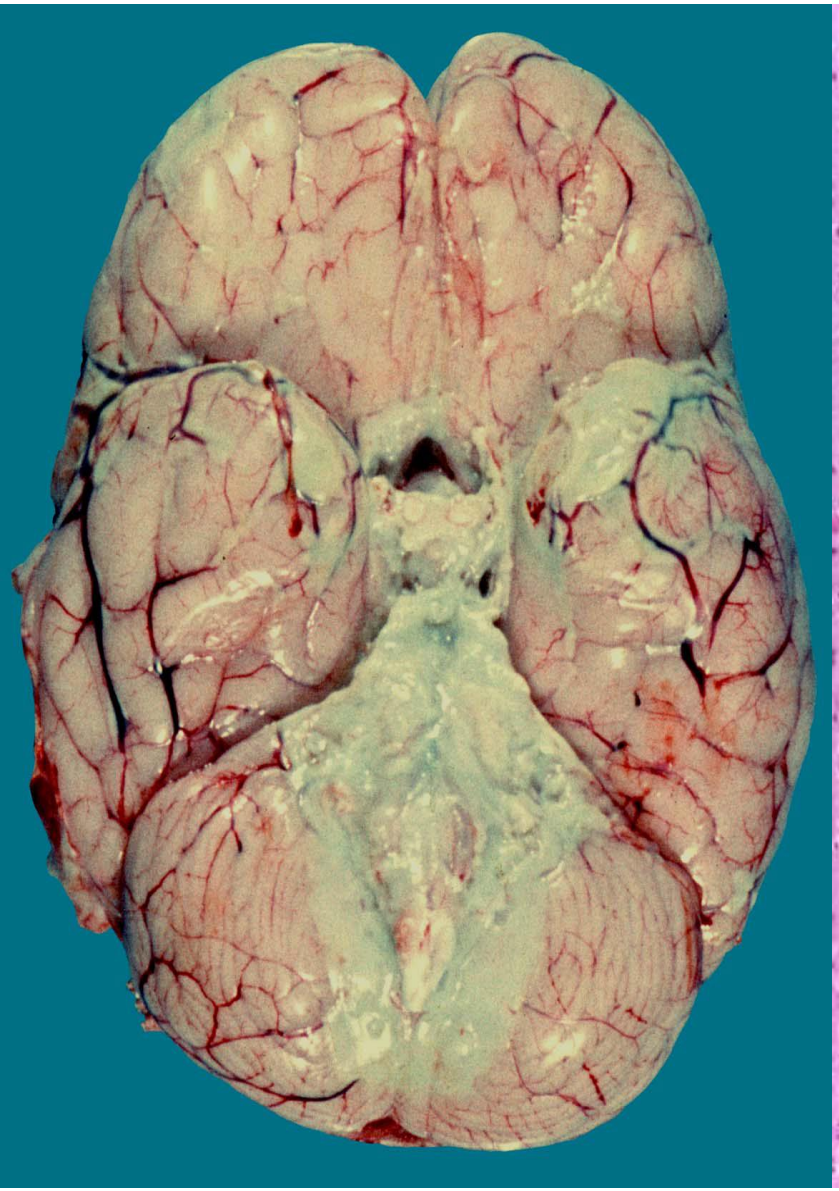
Focal pneumonia.

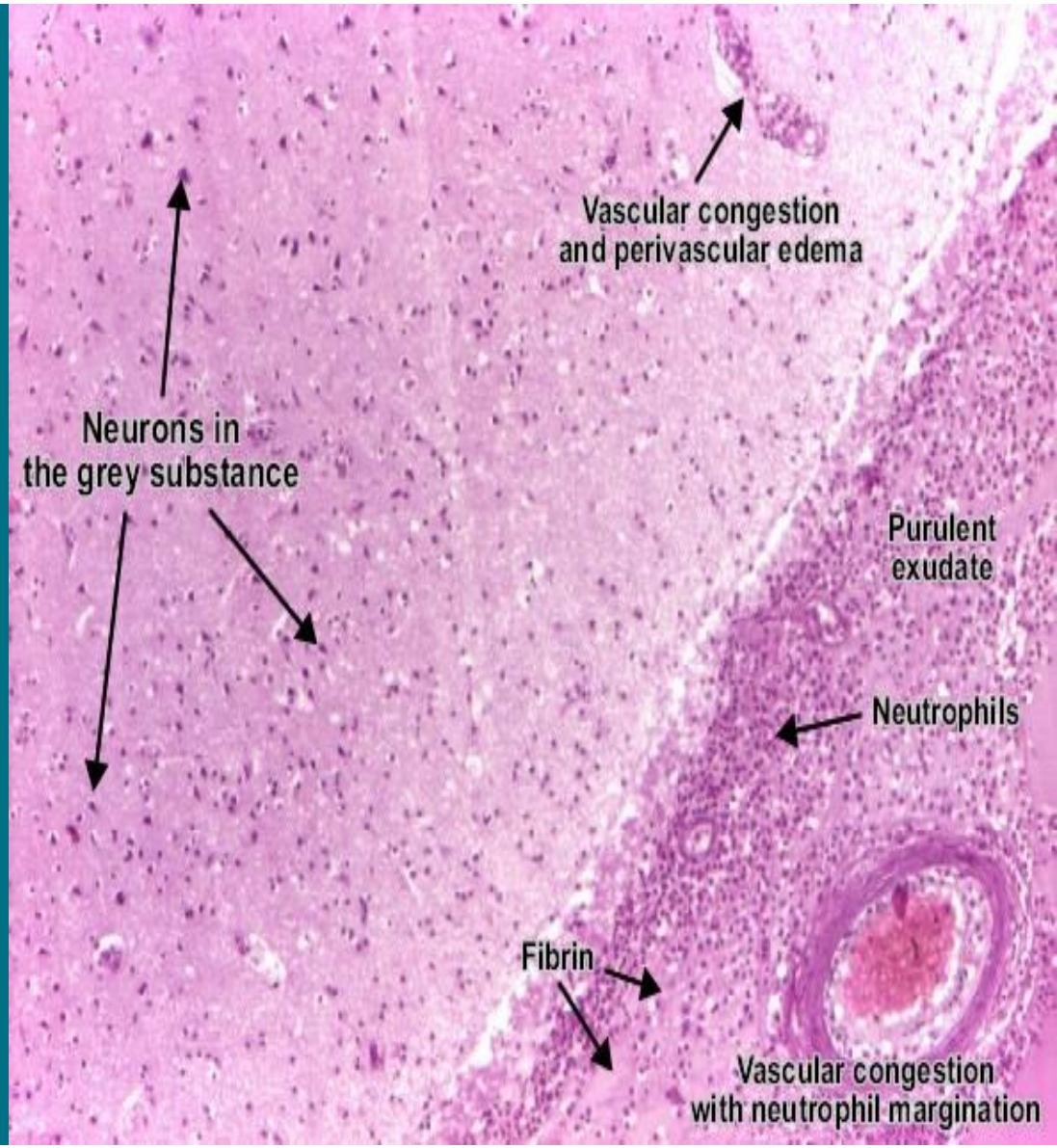


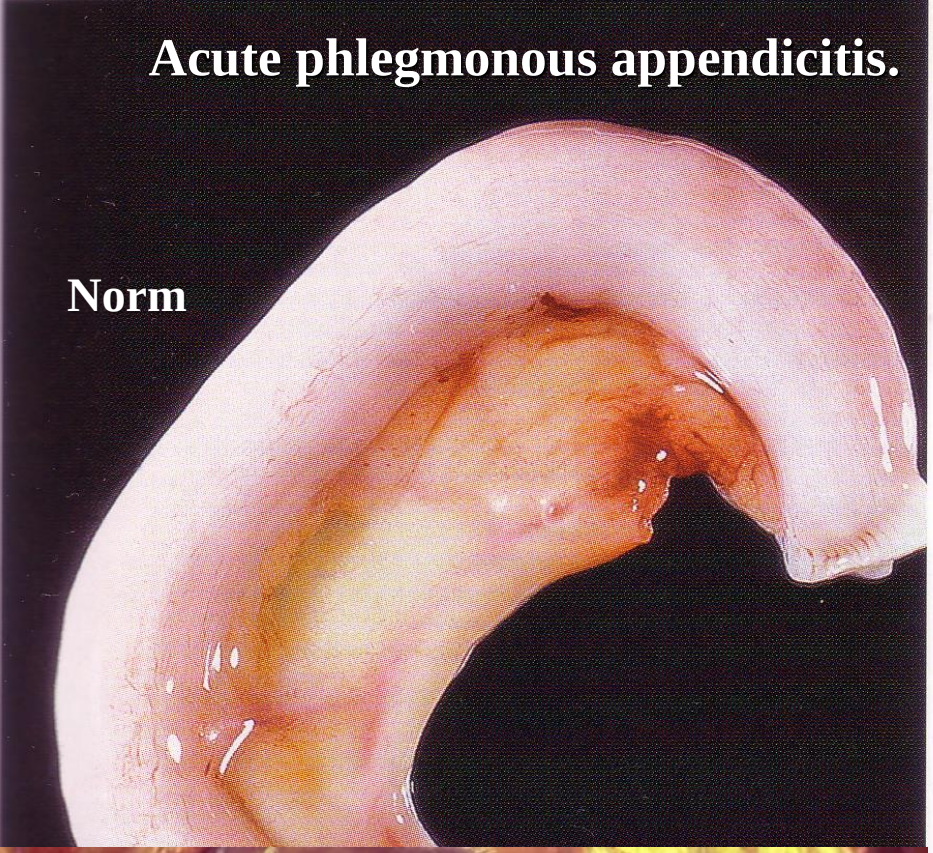


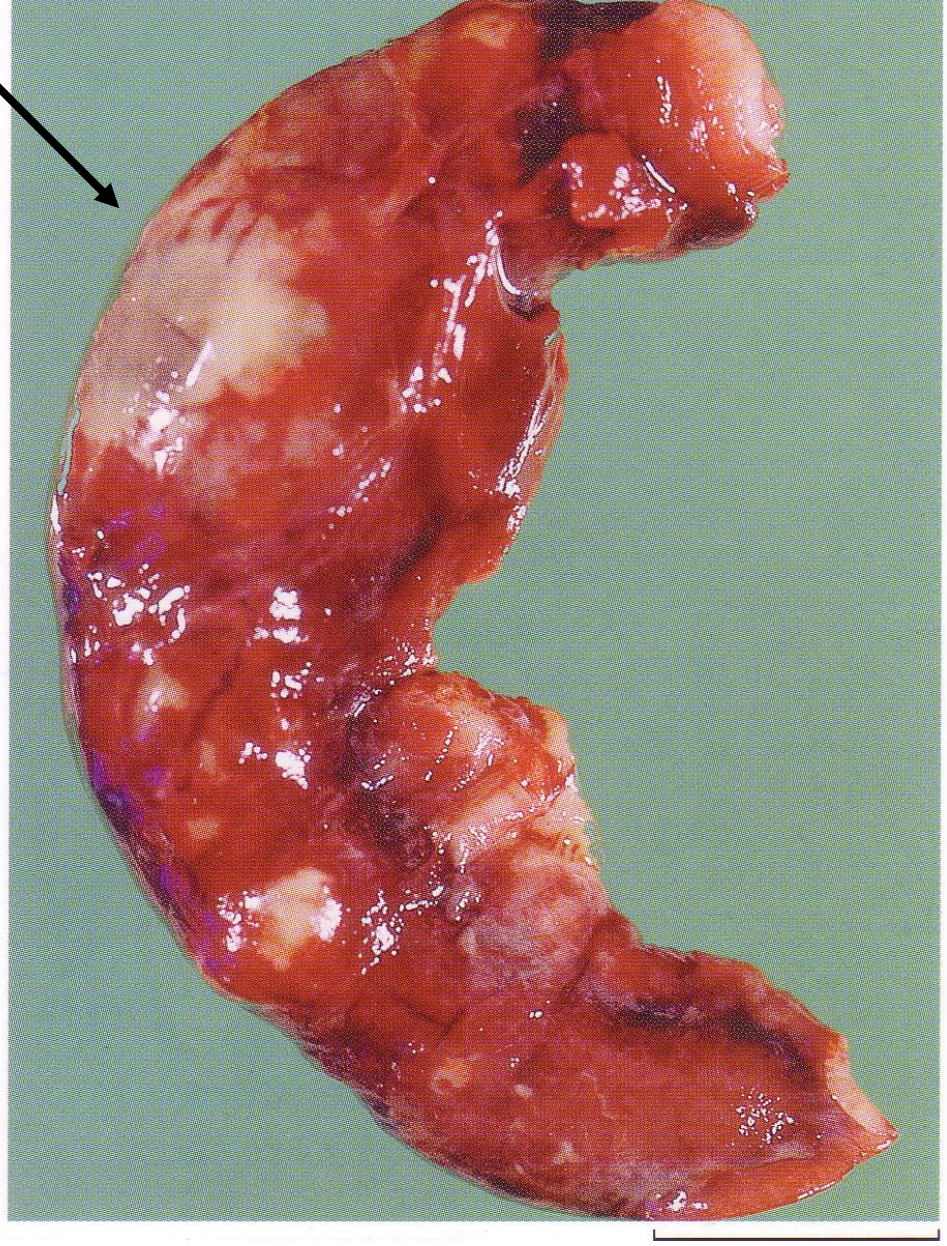
Purulent pleurisy (pleural empyema).

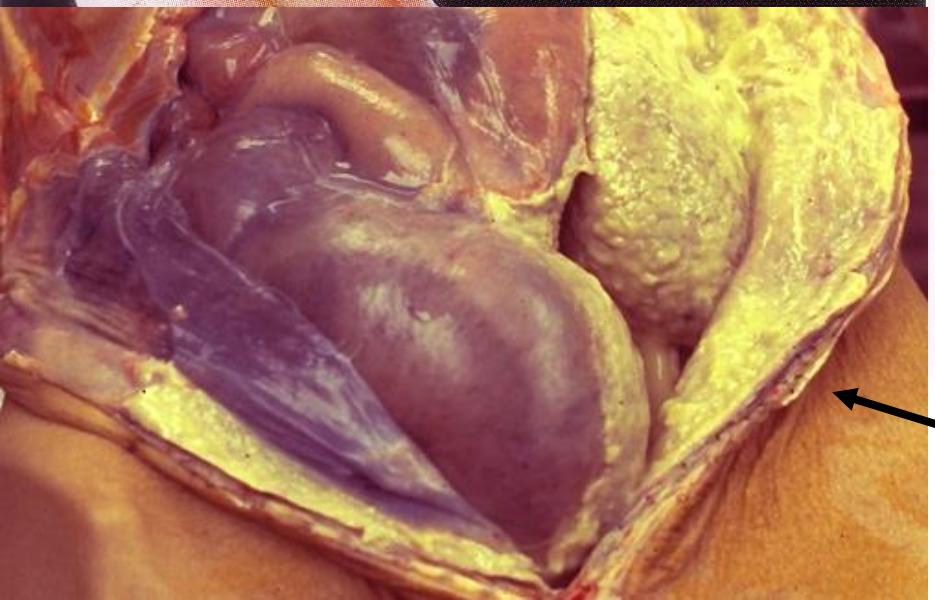
Purulent leptomeningitis.



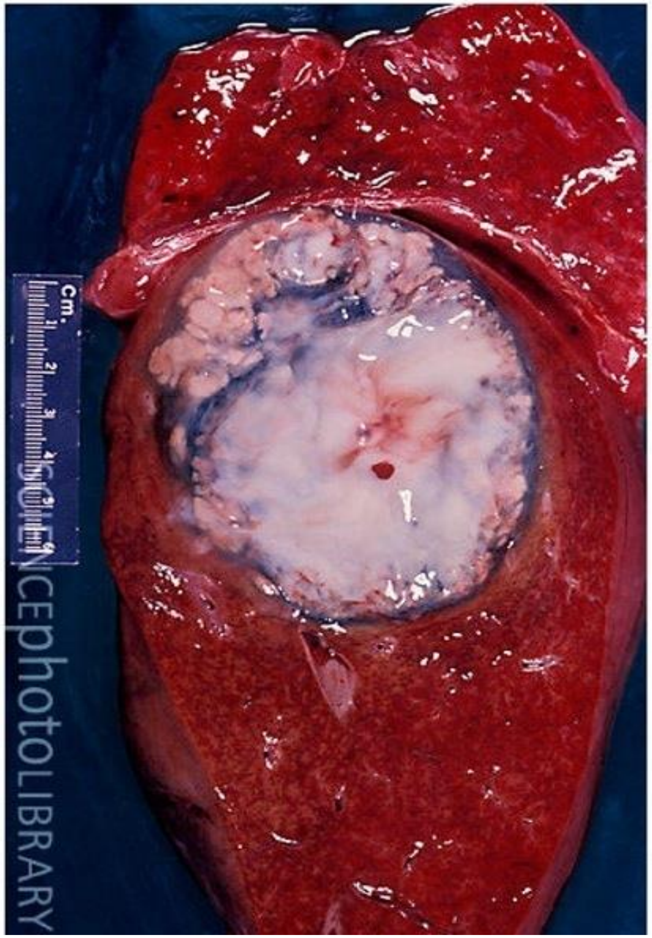








Purulent peritonitis (complication).



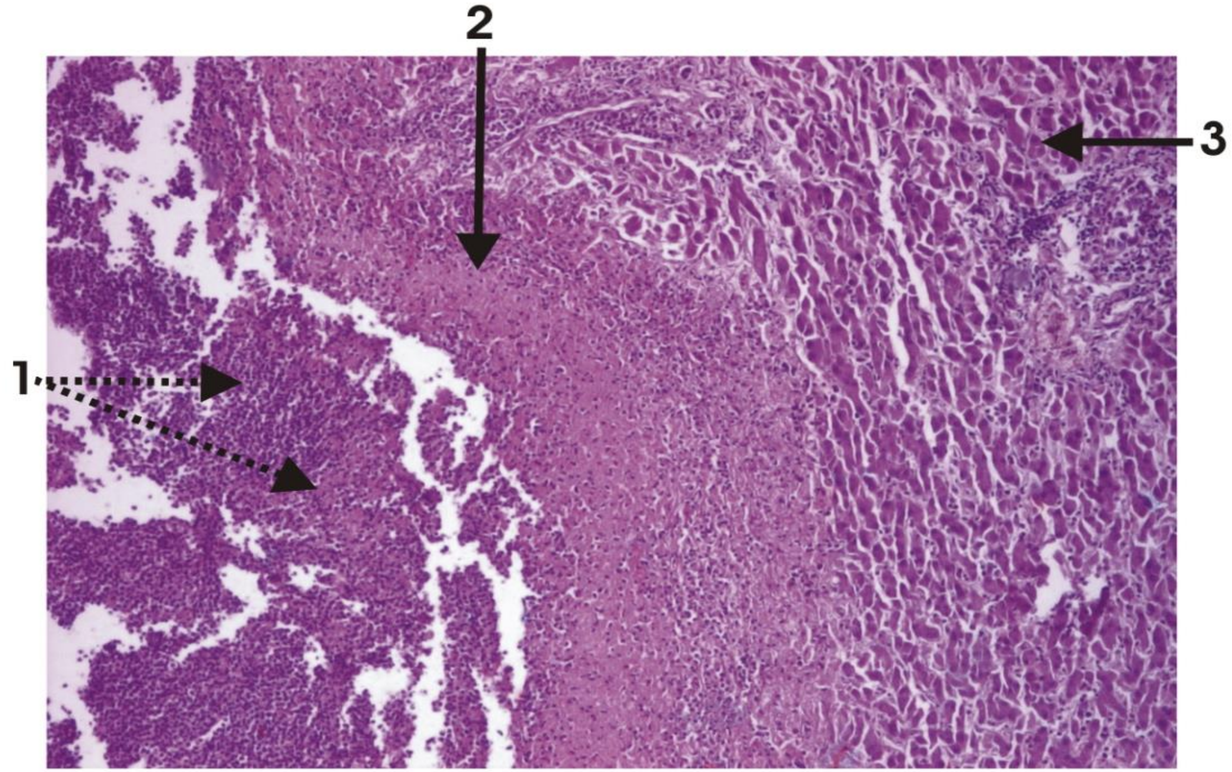
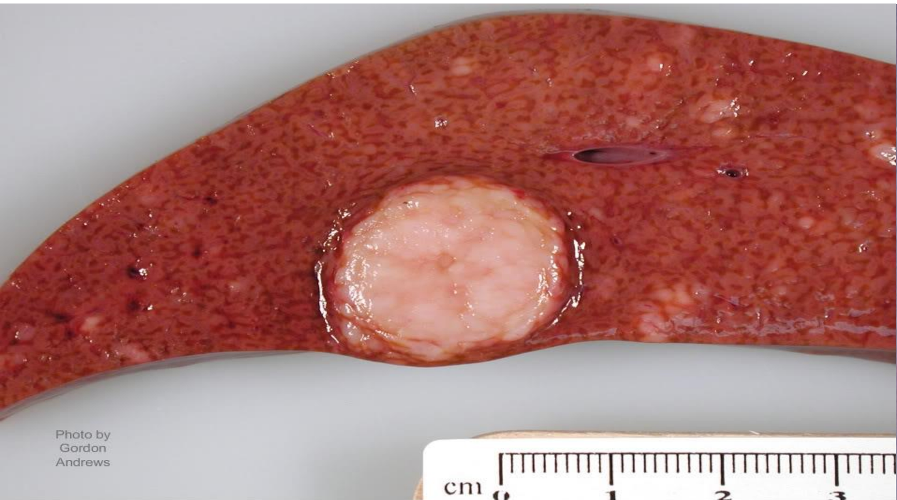
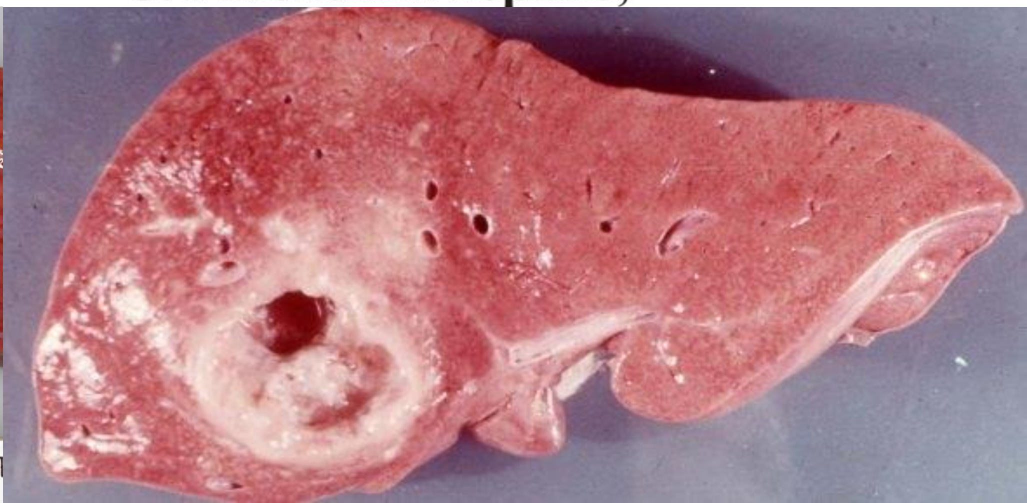
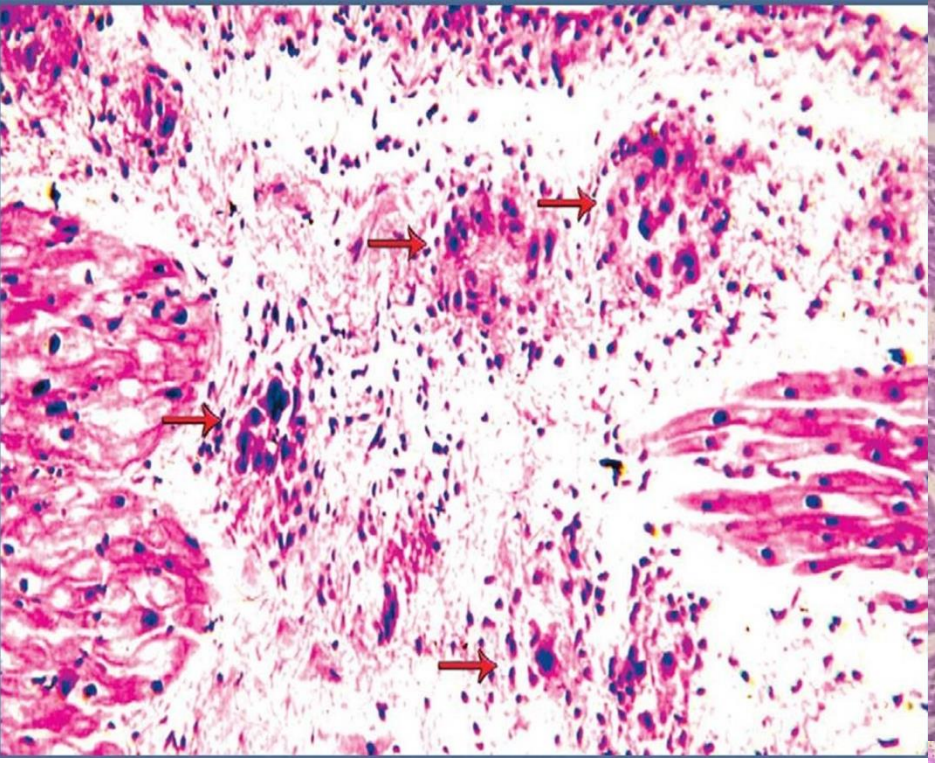
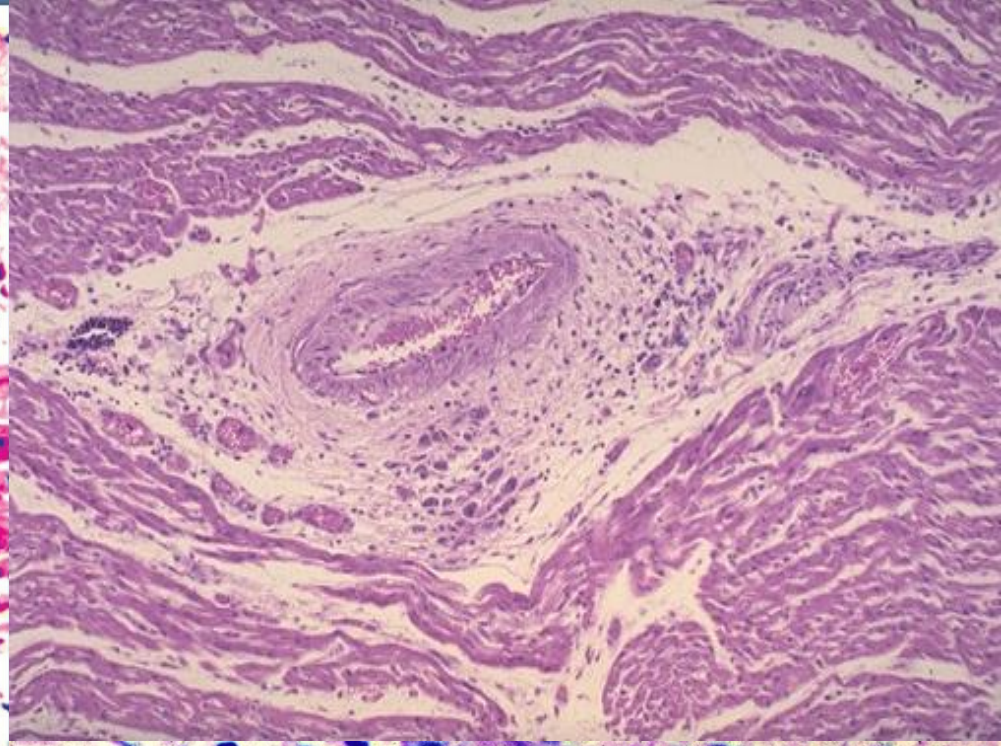


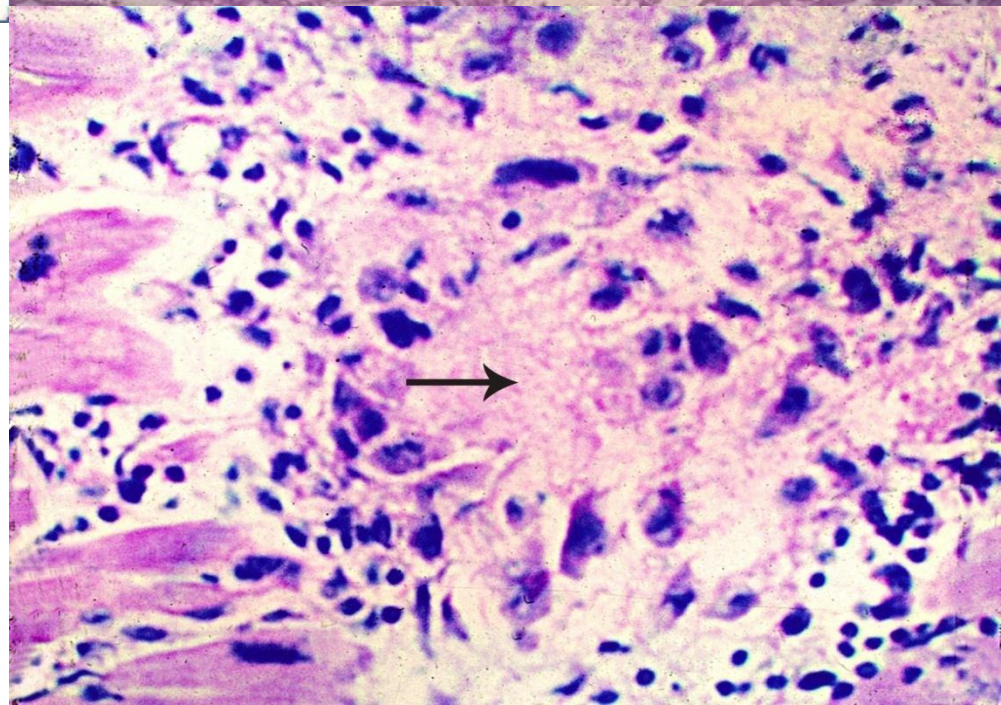
Fig.IV.A.5 Abces hepatic, col. H.E., Ob. 4
1. Detritus necrotic si PMN-uri; 2. Capsula piogena;
3. Parenchim hepatic;



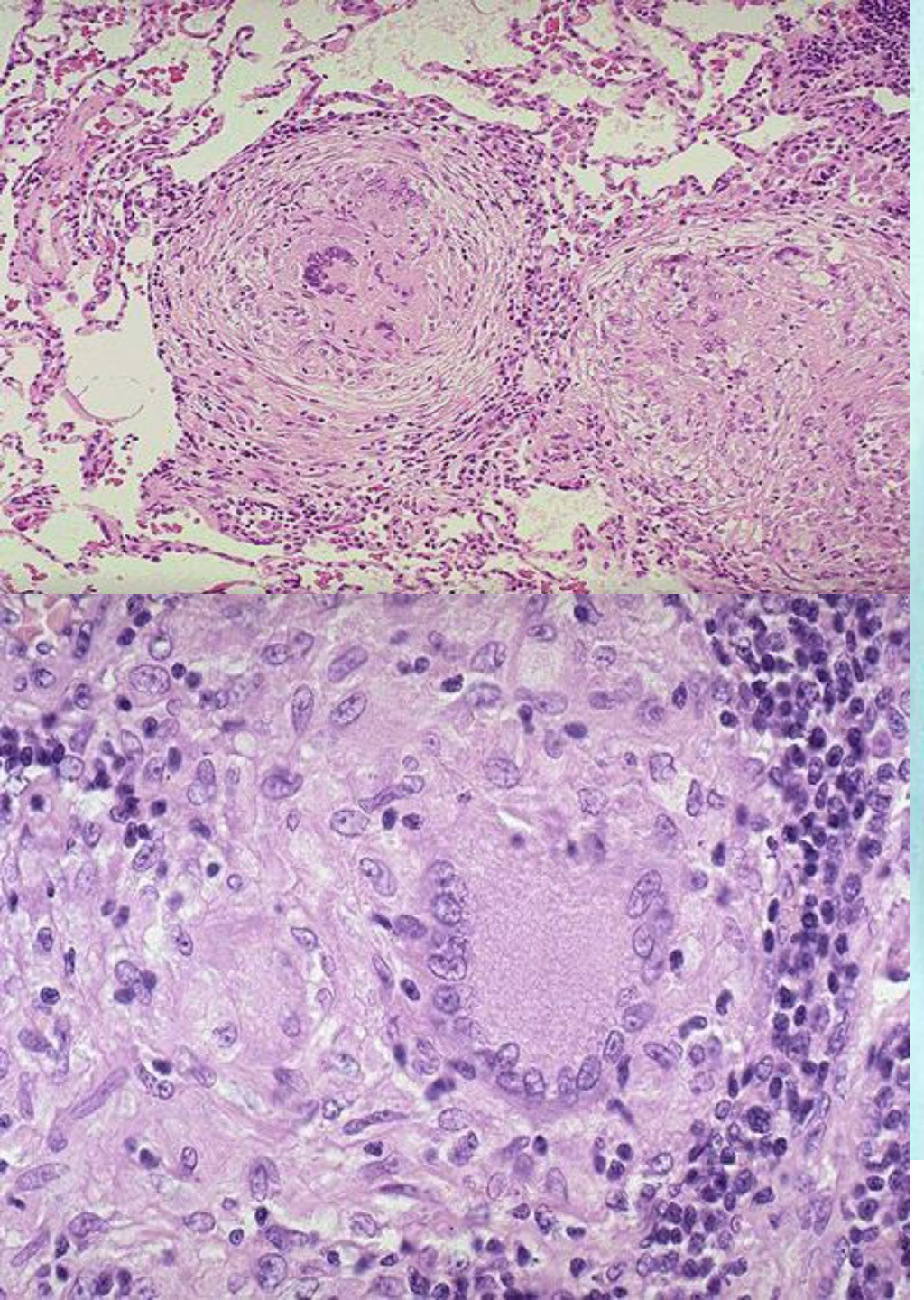


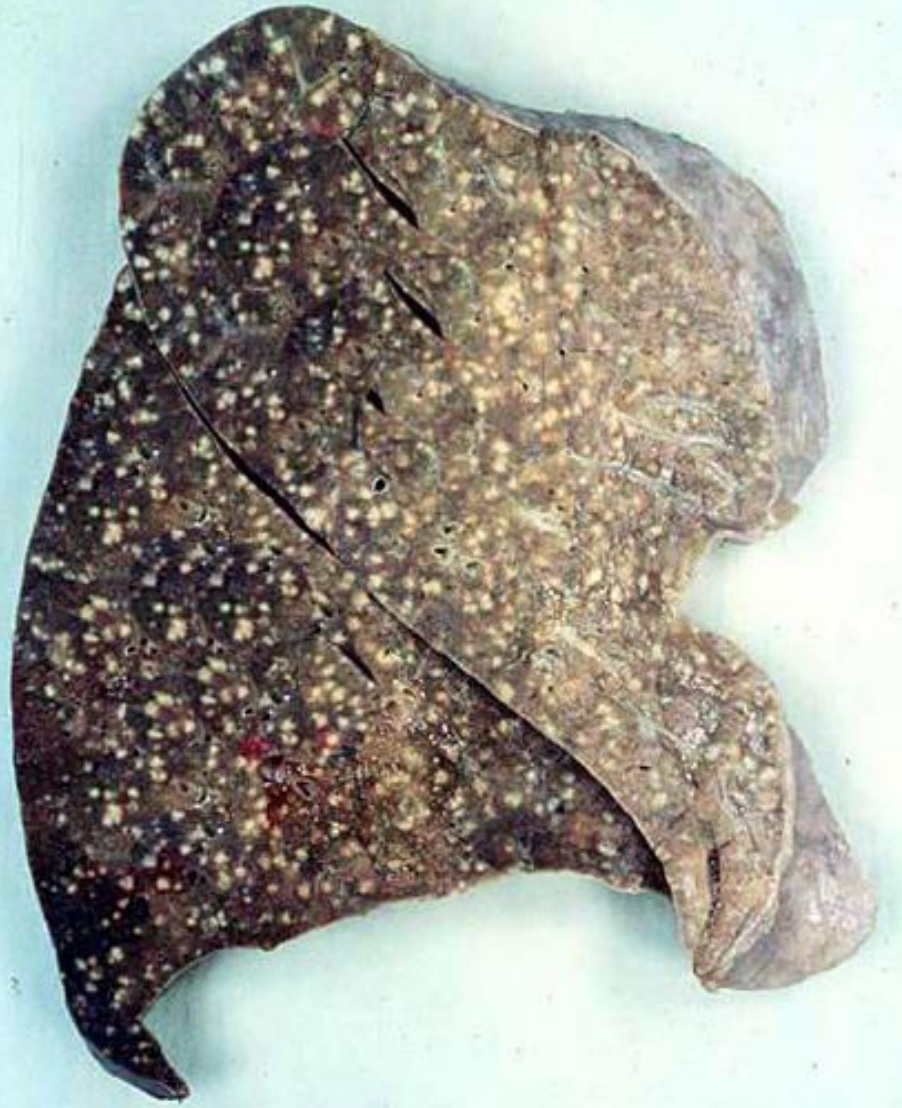




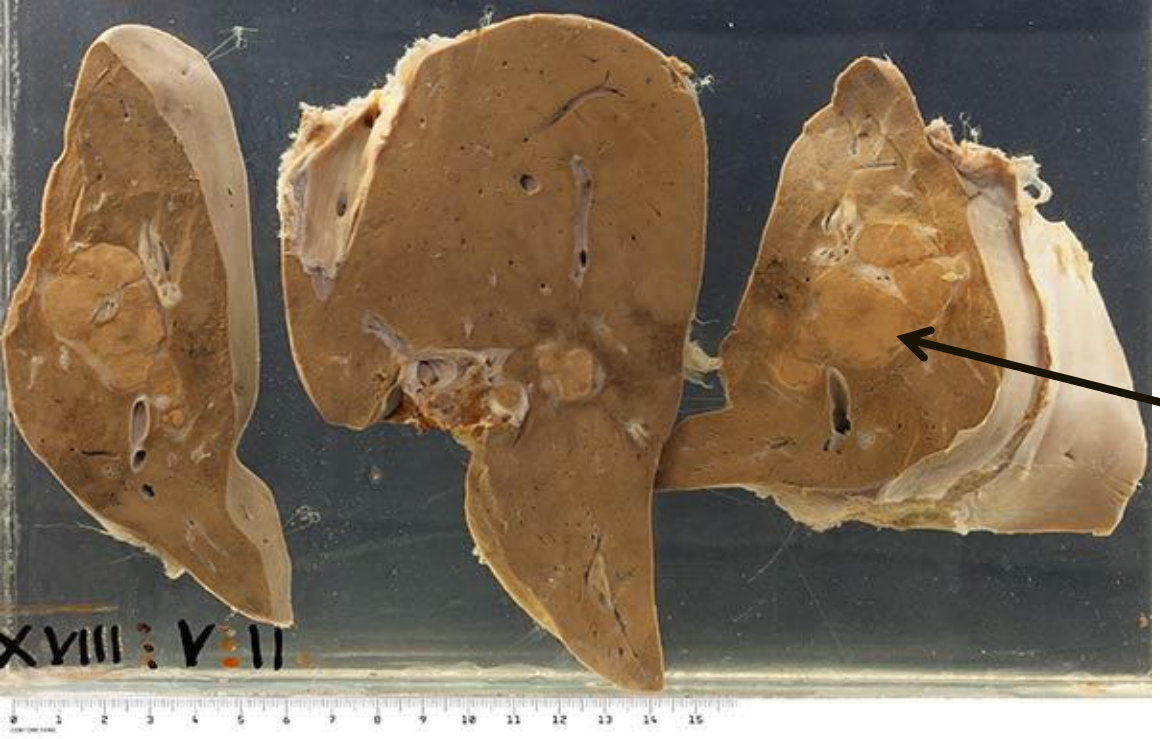


Granulomatous inflammation of the endocardium and myocardium in rheumatic fever. *(H-E stain).*



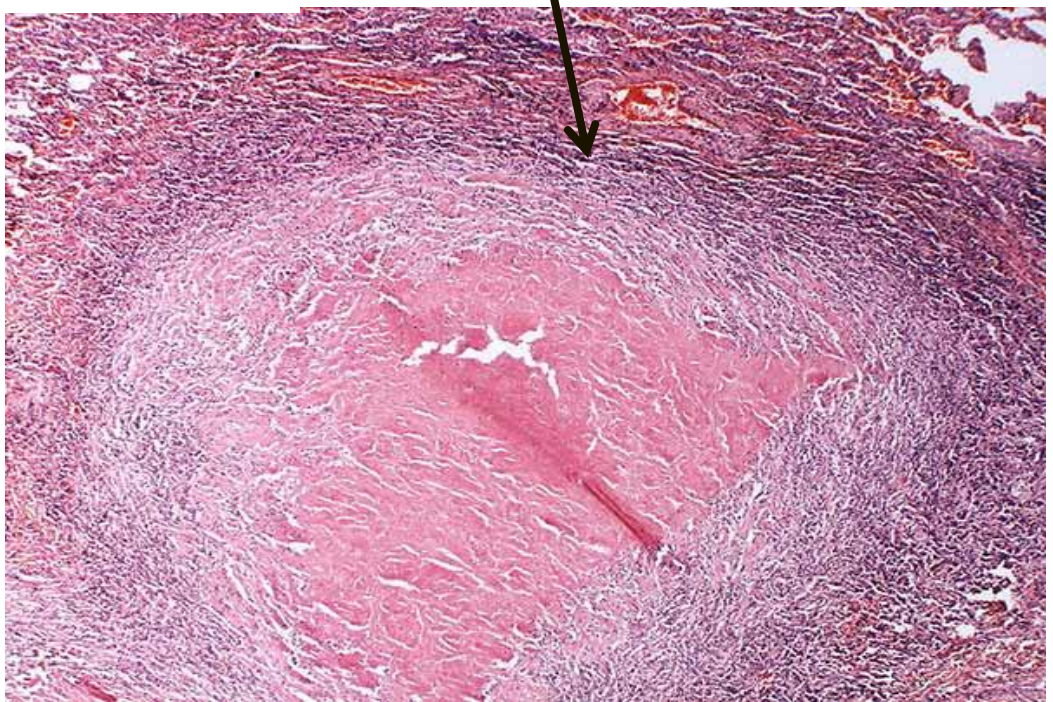


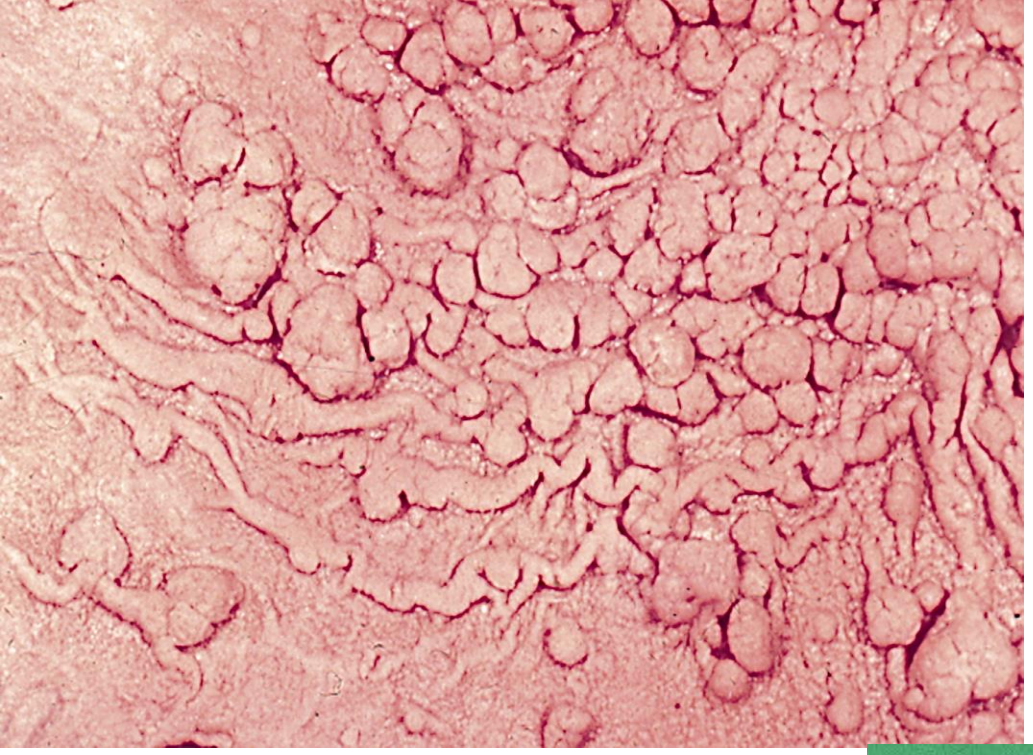
Tuberculous granulomas with giant cells Langhans.



Syphilitic gumma

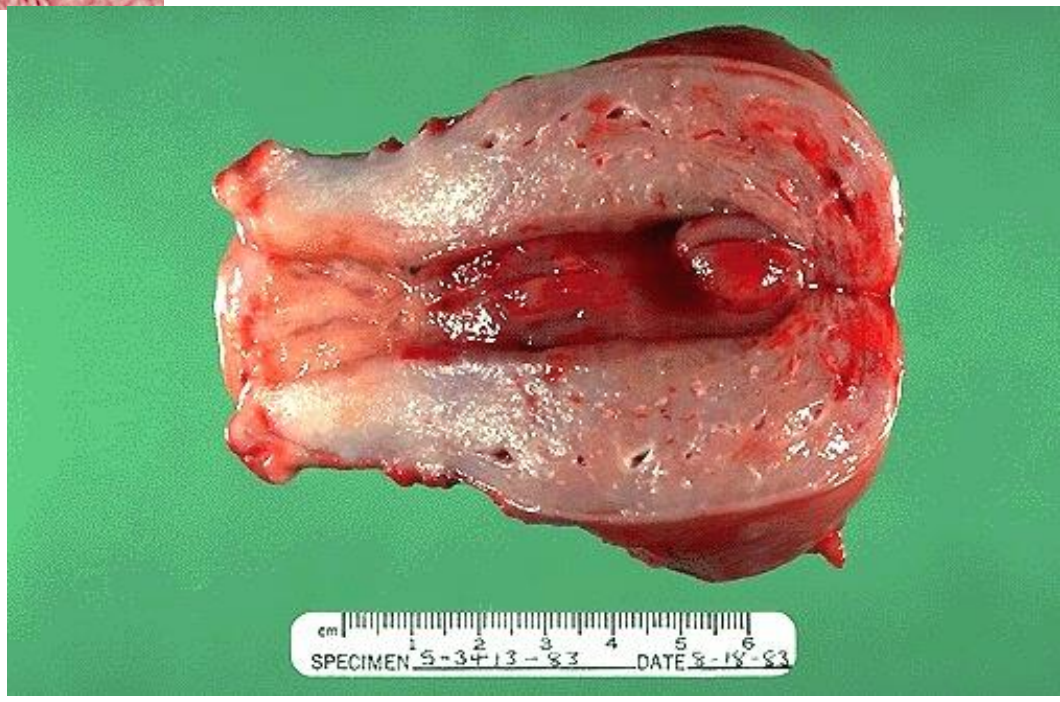
Liver.





Gastric polyposis.

Endometrial polyp.



INTRODUCTION:

- “Inflame” – to set fire.
- Inflammation is “dynamic response of vascularised tissue to injury.”
- Is a protective response.
- Serves to bring defense & healing mechanisms to the site of injury.

Acute inflammatory reactions are triggered by a variety of stimuli:

- Infections (bacterial, viral, parasitic) and microbial toxins
- Trauma (blunt and penetrating)
- Physical and chemical agents (thermal injury, e.g., burns or frostbite; irradiation; some environmental chemicals)
- Tissue necrosis (from any cause)
- Foreign bodies (splinters, dirt, sutures)
- Immune reactions (also called hypersensitivity reactions)

The nomenclature used to describe inflammation in different tissues employs the tissue name and the suffix "*-itis*"

e.g

pancreatitis

meningitis

pericarditis

arthritis

Inflammation

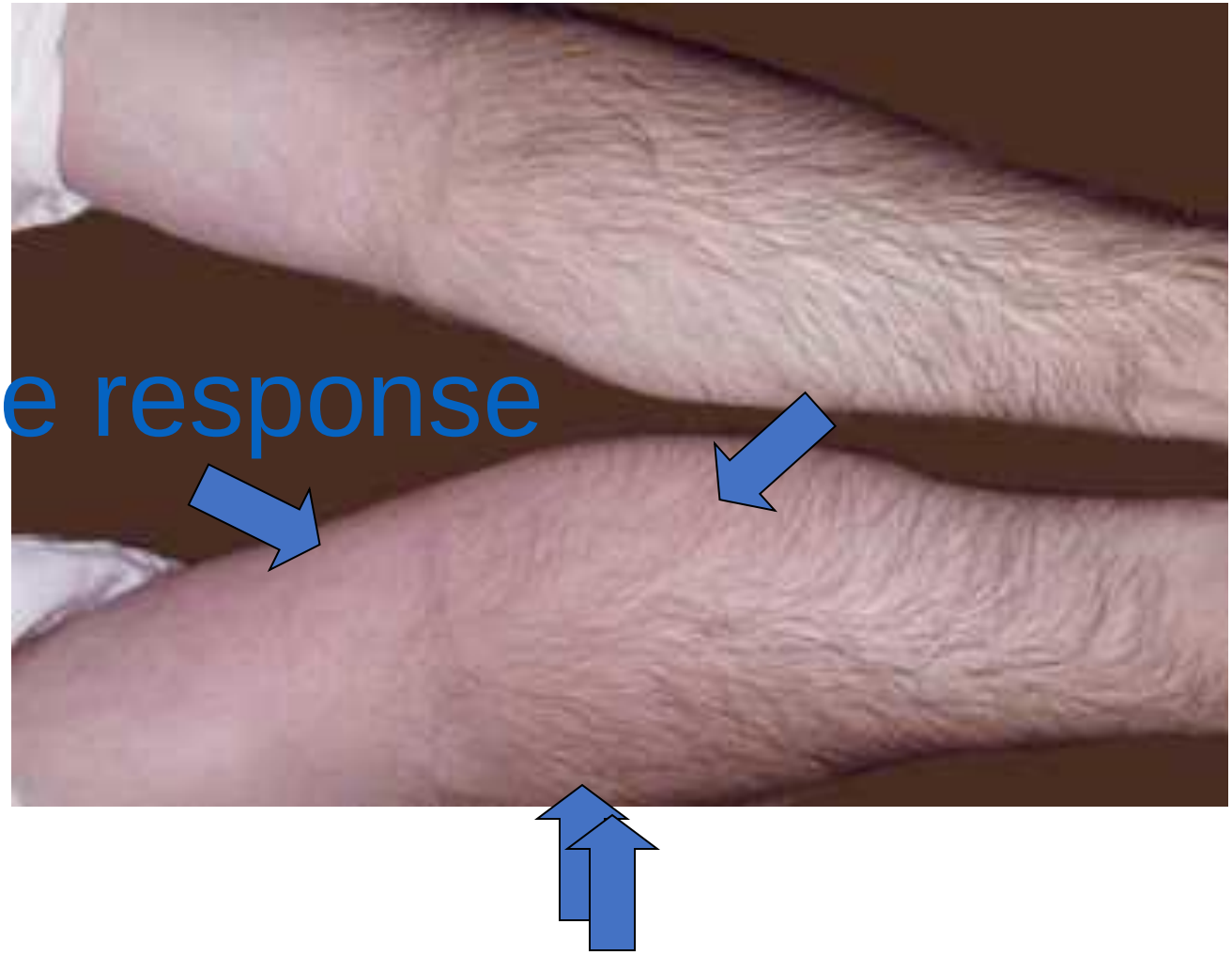
- **provoked response to tissue injury**
  - **chemical agents**
  - **cold, heat**
  - **trauma**
  - **invasion of microbes**
- **serves to destroy, dilute or wall off the injurious agent**
- **induces repair**
- ***protective response***
- ***can be potentially harmful***

Lewis Triple Response:

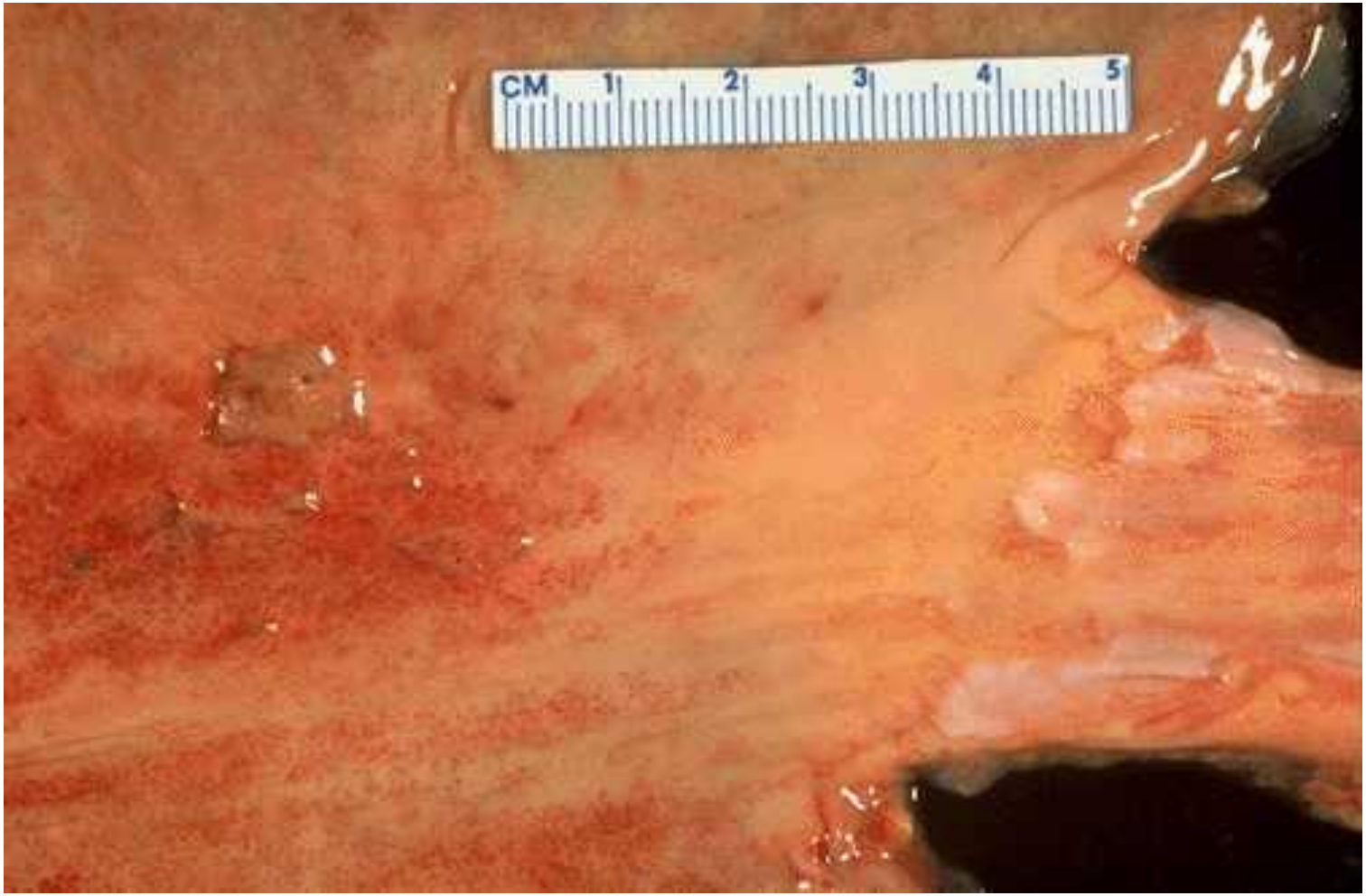
- **Flush** capillary dilatation.
- **Flare** rteriolar dilatation.
- **Weal** xudation, edema.

Red, Warm & Swollen

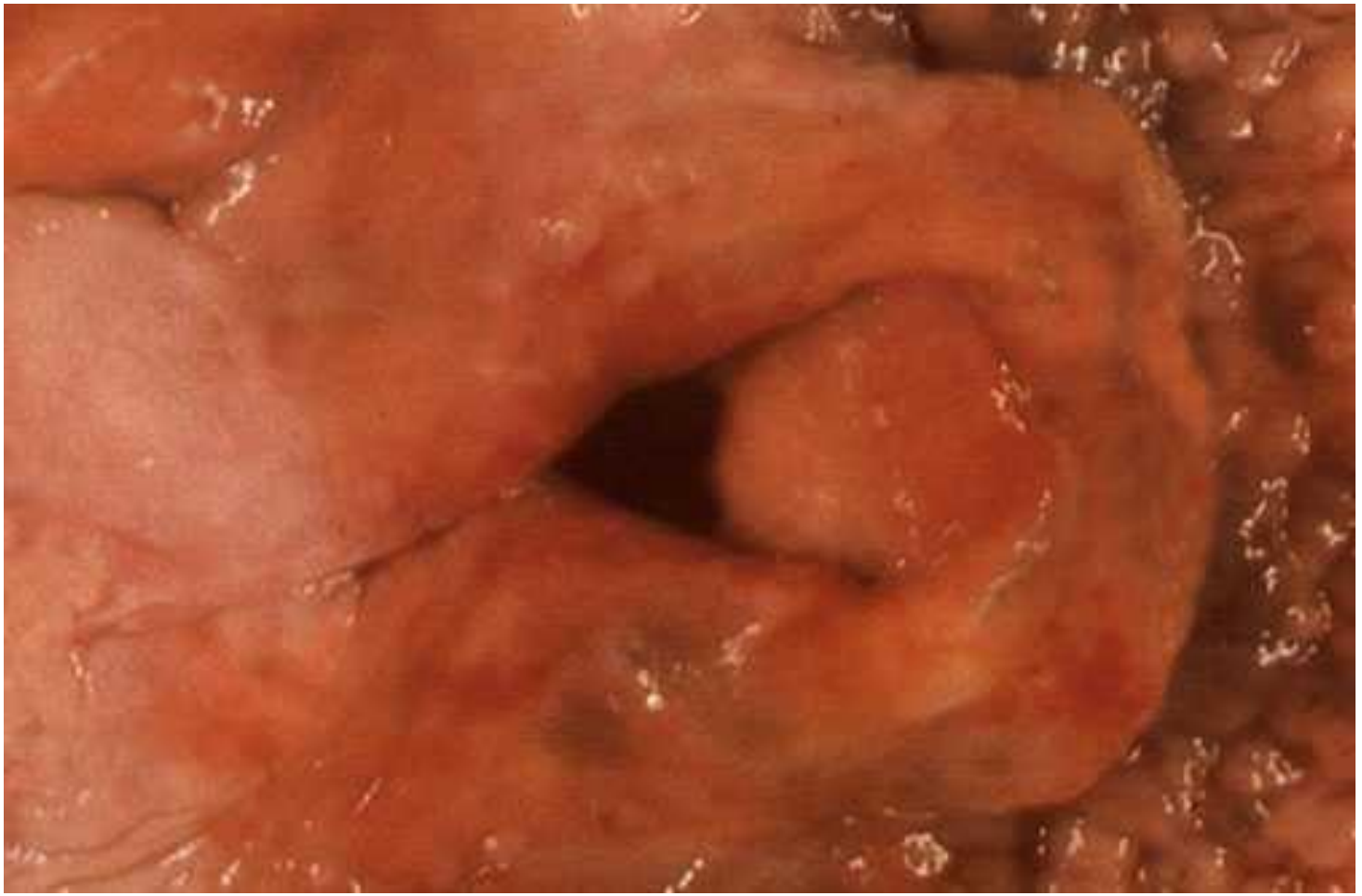
(Flare, Flush & Weal – Lewis)



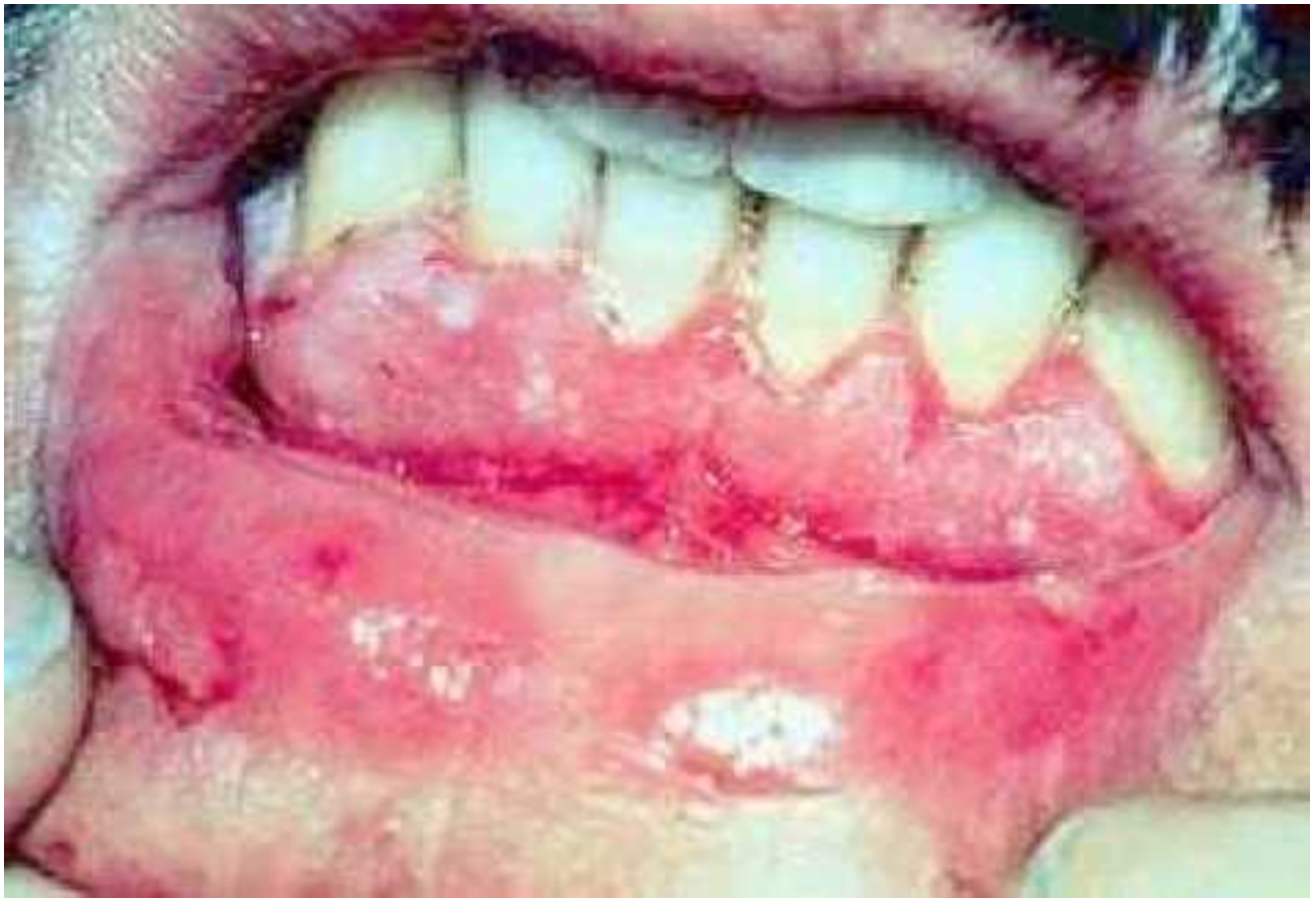
Gastric Ulcer:



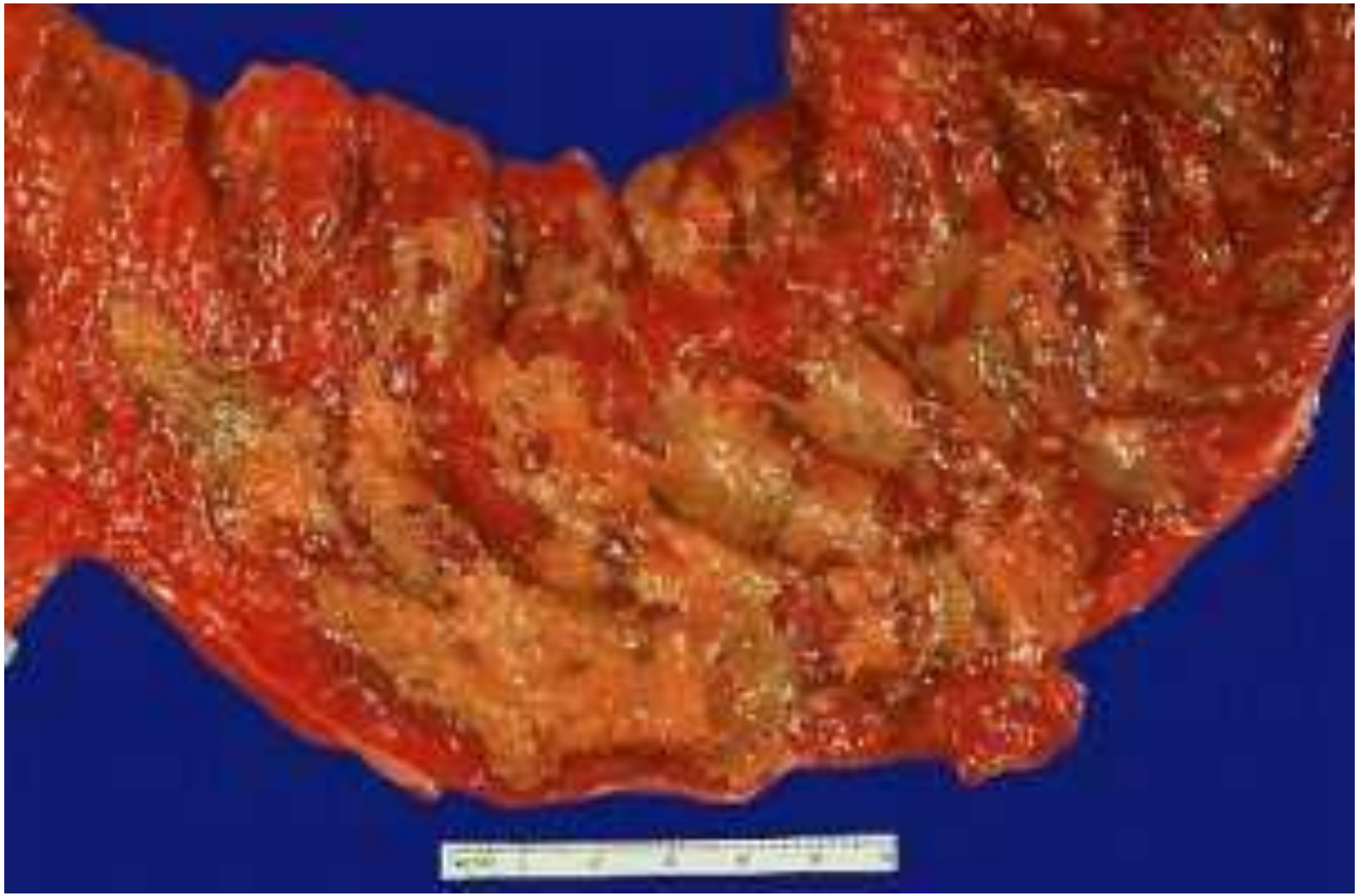
Laryngitis:



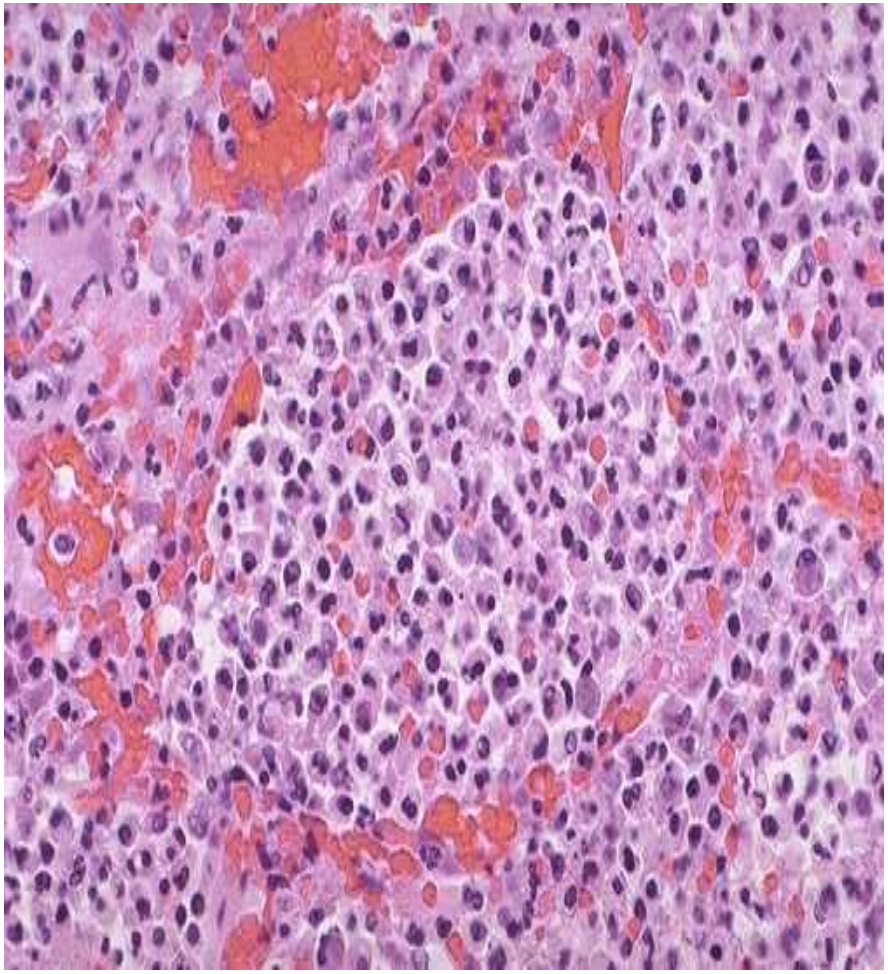
Mouth Aphthus ulcer



Acute Enteritis:



Pneumonia



Cardinal Signs of Inflammation

Celsus, a Roman writer of the first century AD, first listed the four cardinal signs of inflammation:

- ***Rubor*** **(Redness)**
- ***Calor*** **(Warmth)**
- ***Tumor*** **(Swelling)**
- ***Dolor*** **(Pain)**
- ***Functio laesa*** **(Loss of function, later added by Virchow)**

Acute Inflammation

Acute Inflammatory Response

- Clinical indications
  - Generalize malaise
  - Fever
  - Pain often localized to the inflamed area
  - Rapid pulse rate
- Lab values
  - Raised neutrophil count in peripheral blood
  - Increased erythrocyte sedimentation rate
  - Increased acute phase proteins in the blood
    - Increase greatly in acute inflammation
    - Induced by IL-1 and produced by the liver
    - C-reactive protein (liver) is the most common
      - **Used to monitor patients with acute myocardial infarction**

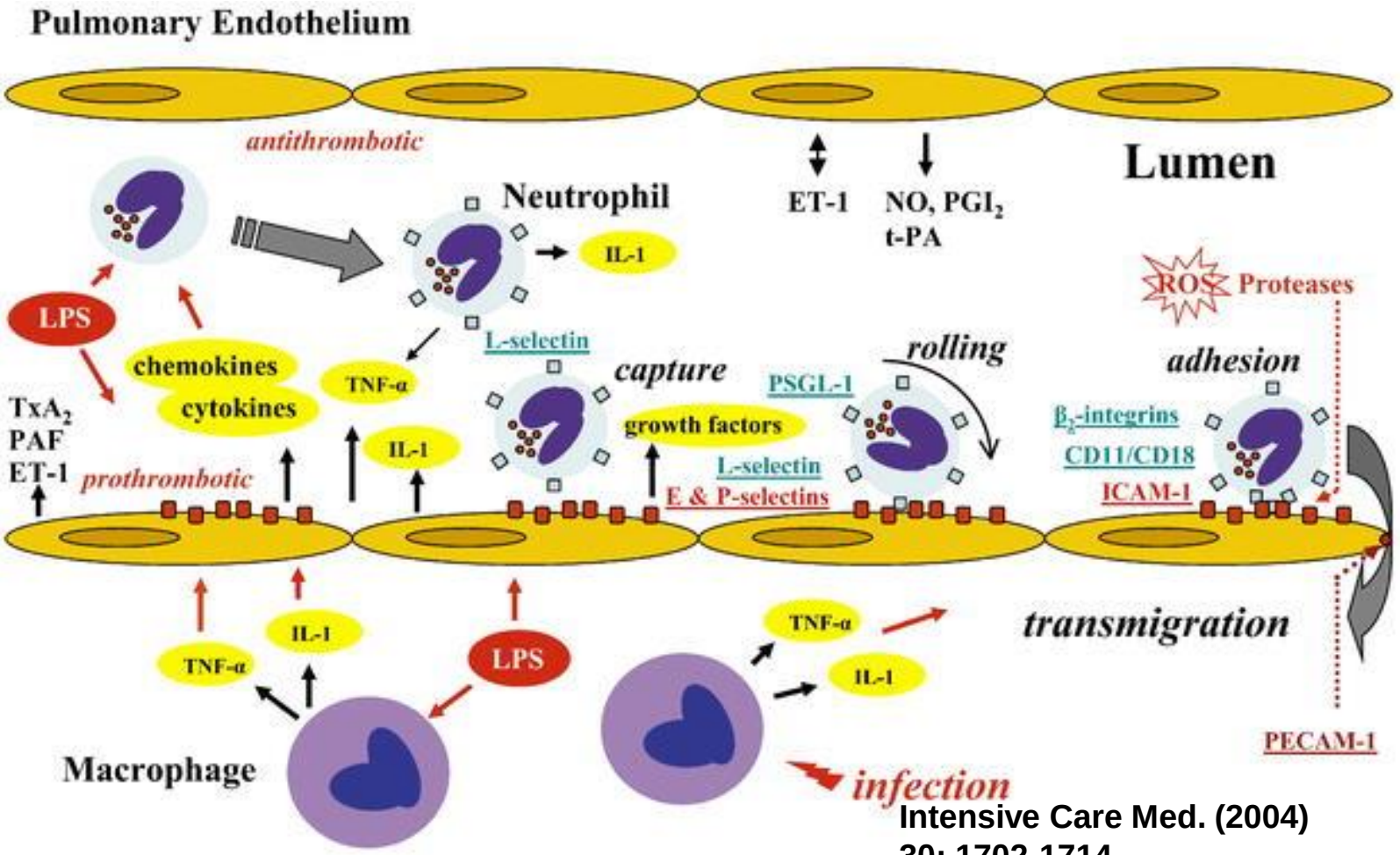
11

Acute Inflammation

PATHOGENESIS: **Three main processes occur at the site of inflammation, due to the release of chemical mediators:**

1. **Increased blood flow (redness and warmth)**
2. **Increased vascular permeability (swelling, pain & loss of function)**
3. **Leukocytic Infiltration**

The inflammatory response consists of a vascular and a cellular reaction



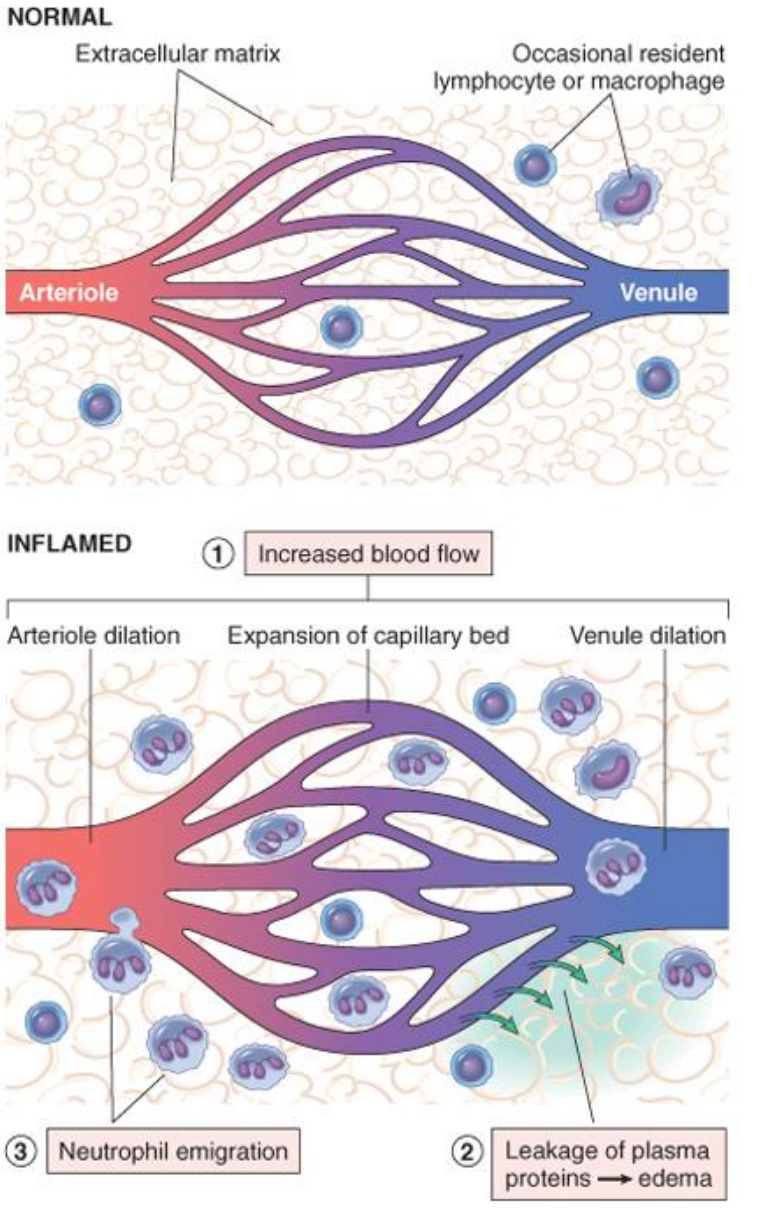
Intensive Care Med. (2004) 30: 1702-1714

Acute inflammation involves:

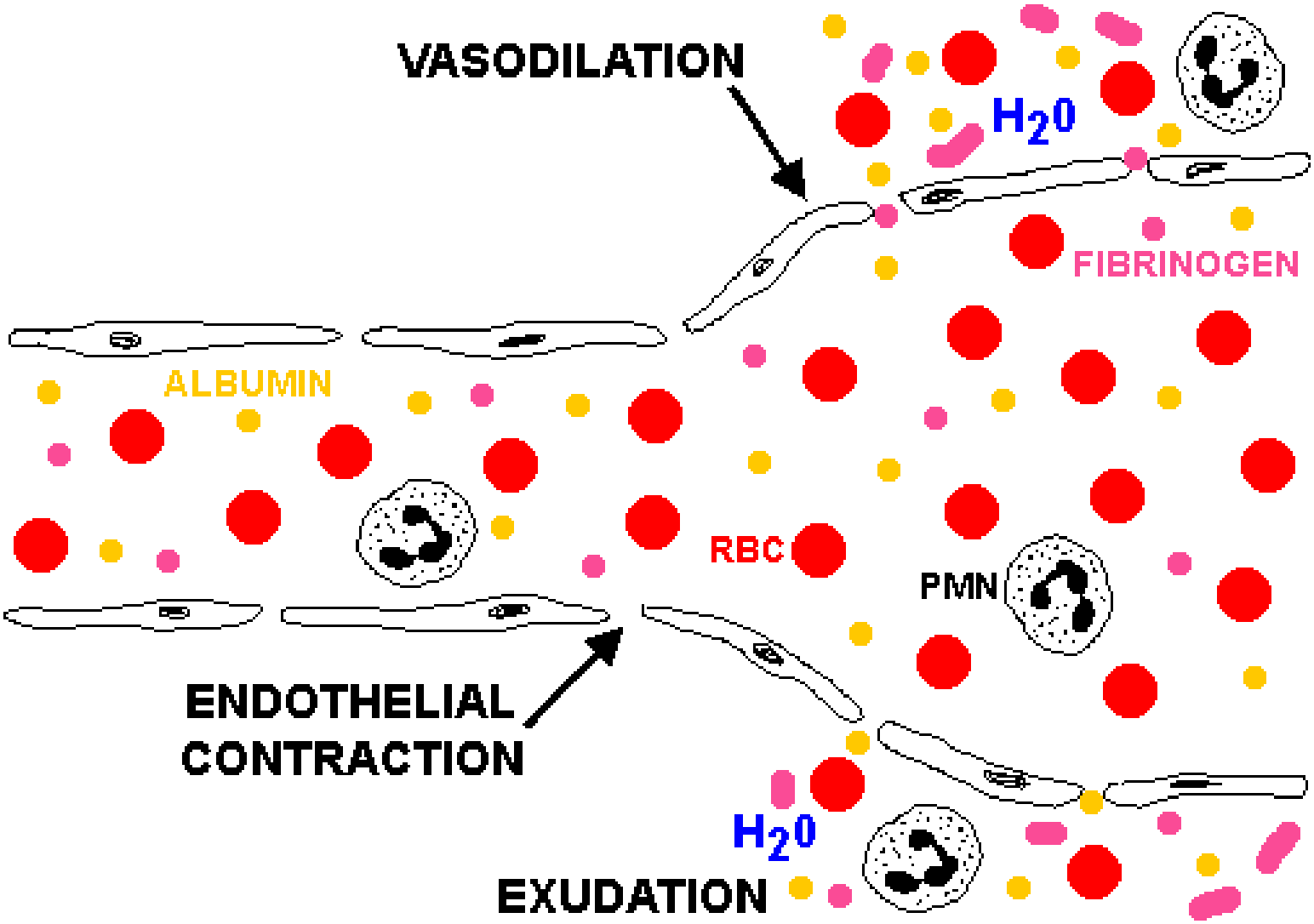
alteration of vascular caliber
following very brief vasoconstriction (seconds), vasodilation leads to increased blood flow and blood pooling creating redness and warmth (rubor and calor)

changes of microvasculature
increased permeability for plasma proteins and cells creating swelling (tumor). Fluid loss leads to concentration of red blood cells and slowed blood flow (stasis)

emigration of leukocytes from microcirculation
due to stasis and activation leads migration towards offending agent



Mechanism of Inflammation:



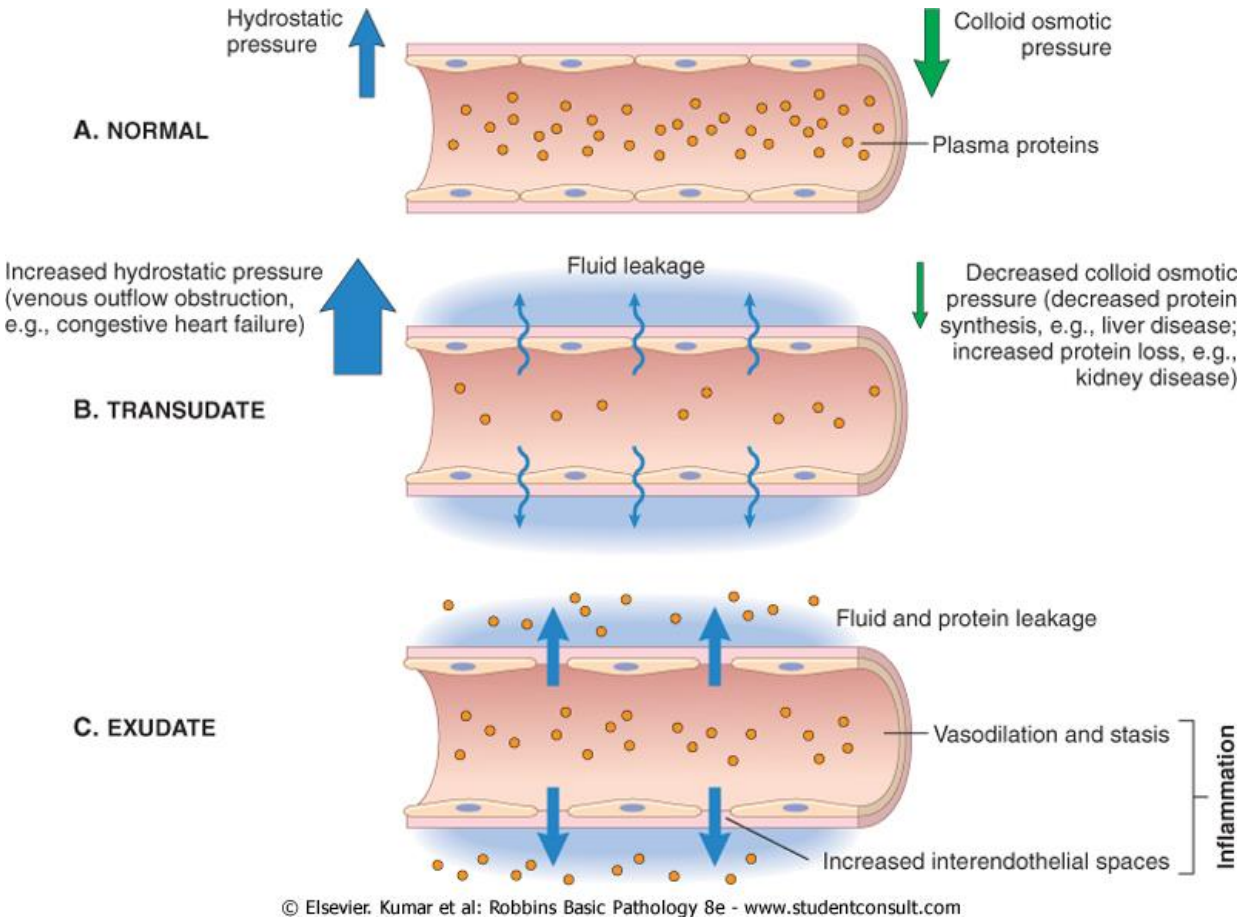
Vascular changes and fluid leakage during acute inflammation lead to *Edema in a process called Exudation*

Transudate

- **result of hydrostatic or osmotic imbalance**
- ultrafiltrate of plasma
- Low protein content
- **specific gravity < 1.015**

Exudate

- **result of inflammation**
- vascular permeability ↑
- high protein content
- **specific gravity >1.020**

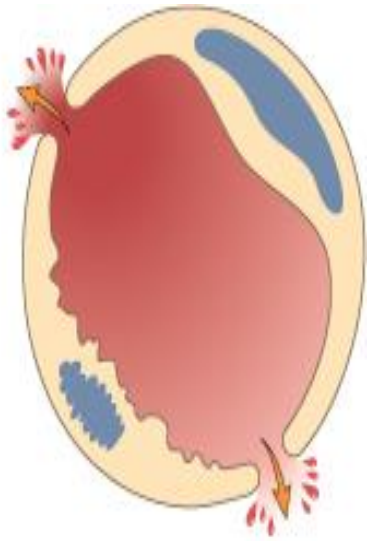


Increased vascular permeability and edema: a hallmark of acute inflammation

- Leakage is restricted to venules of 20-60μm in diameter
  - caused by endothelial gaps
  - usually an immediate and transient response (30 min.)

Gaps due to endothelial contraction

- Venules
- Vasoactive mediators (histamine, leukotrienes, etc.)
- Most common
- Fast and short-lived (minutes)



- Gaps occur due to contraction of e.g myosin and shortening of the individua endothelia cell

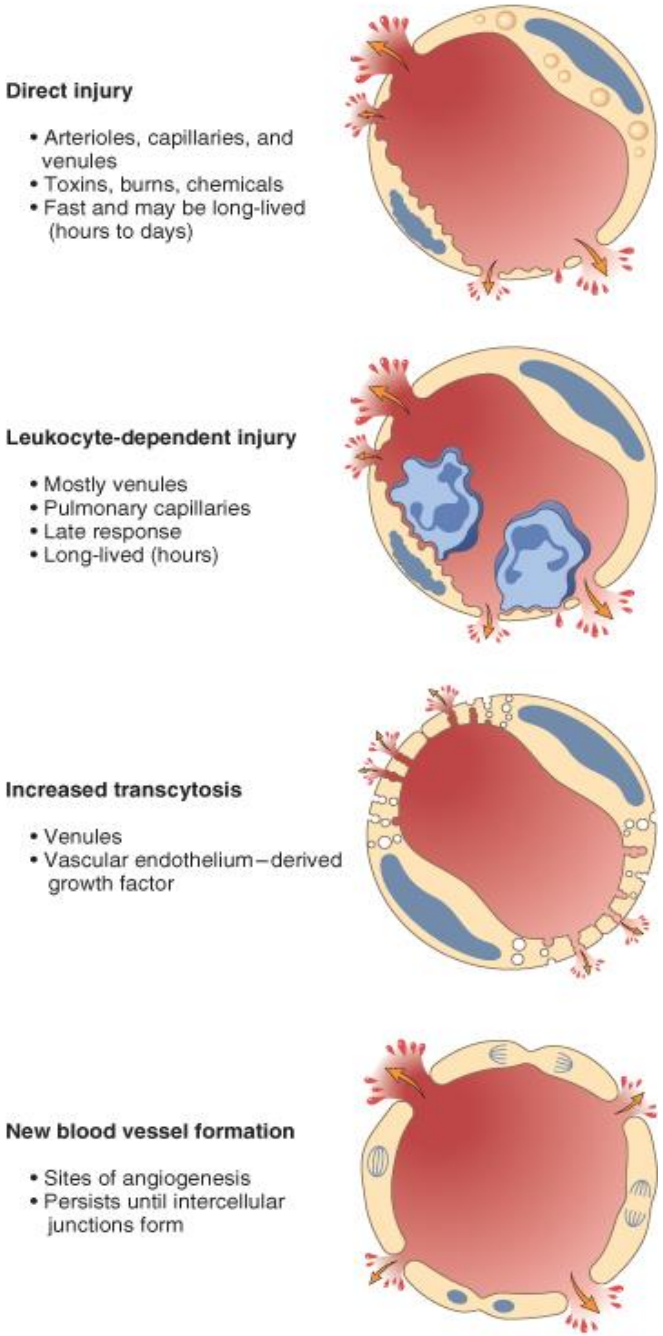
- loss of protein from plasma leads to edema
  - due to reduced osmotic pressure in the vasculature
  - and increased osmotic pressure in the interstitium

- **direct endothelial injury causing necrotic cell death will result in leakage from all levels of microcirculation (venules, capillaries and arterioles)**
    - **This reaction is immediate and sustained**

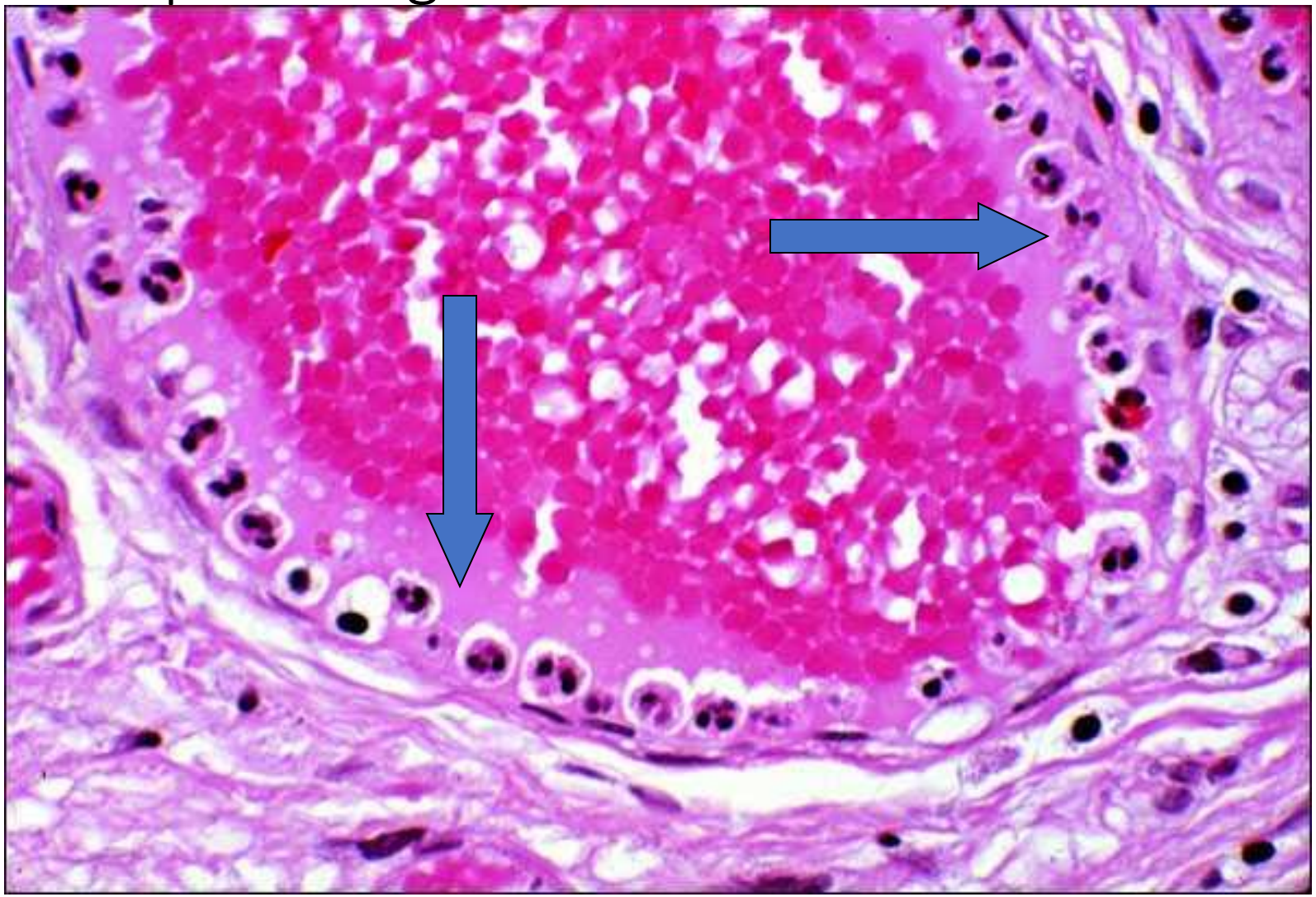
- **Delayed prolonged leakage begins after 2-12 hours and can last several days due to thermal-, x-ray radiation or ultraviolet radiation (sunburn) and involves venules and capillaries**

- **Leakage from new blood vessels during tissue repair (angiogenesis) due to immature endothelial layer**

All these described mechanisms may occur in one wound (e.g burns) and can be life threatening



Neutrophil Margination

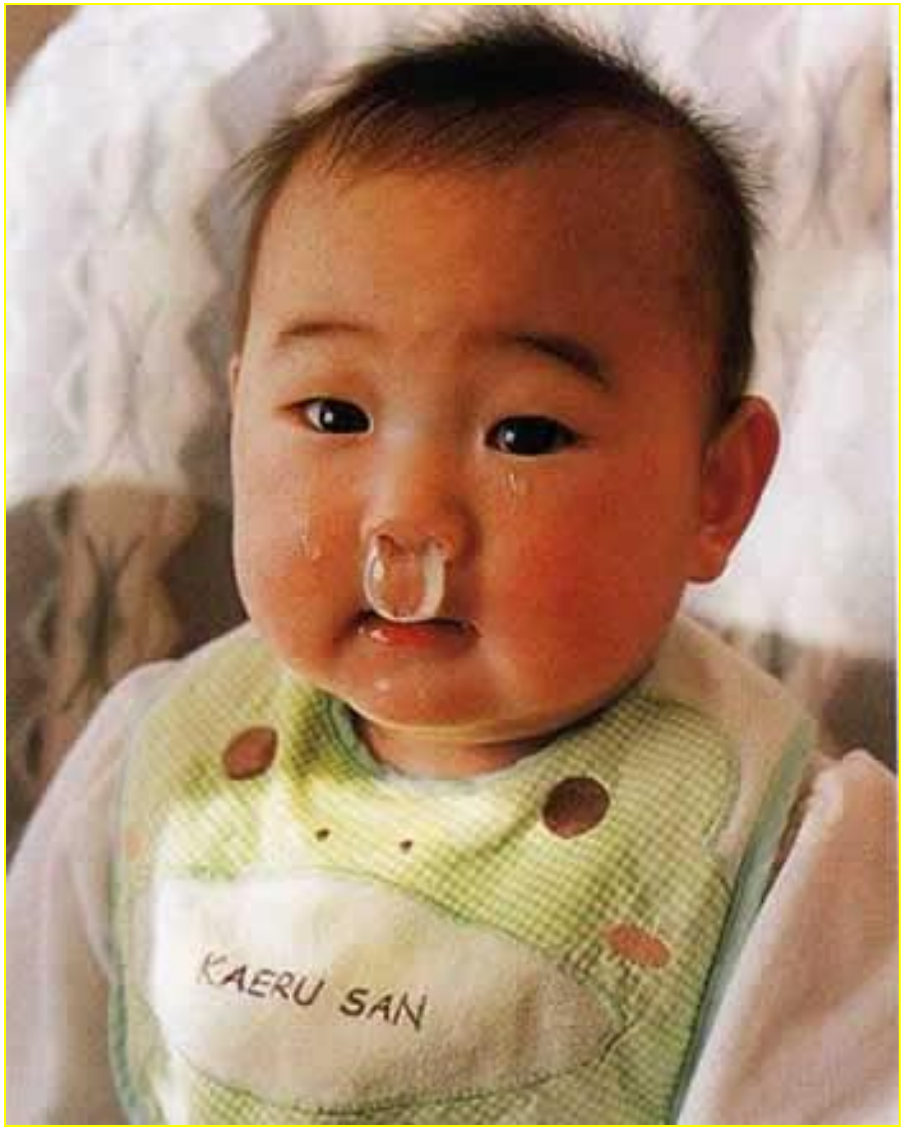


Acute inflammation

PATTERNS

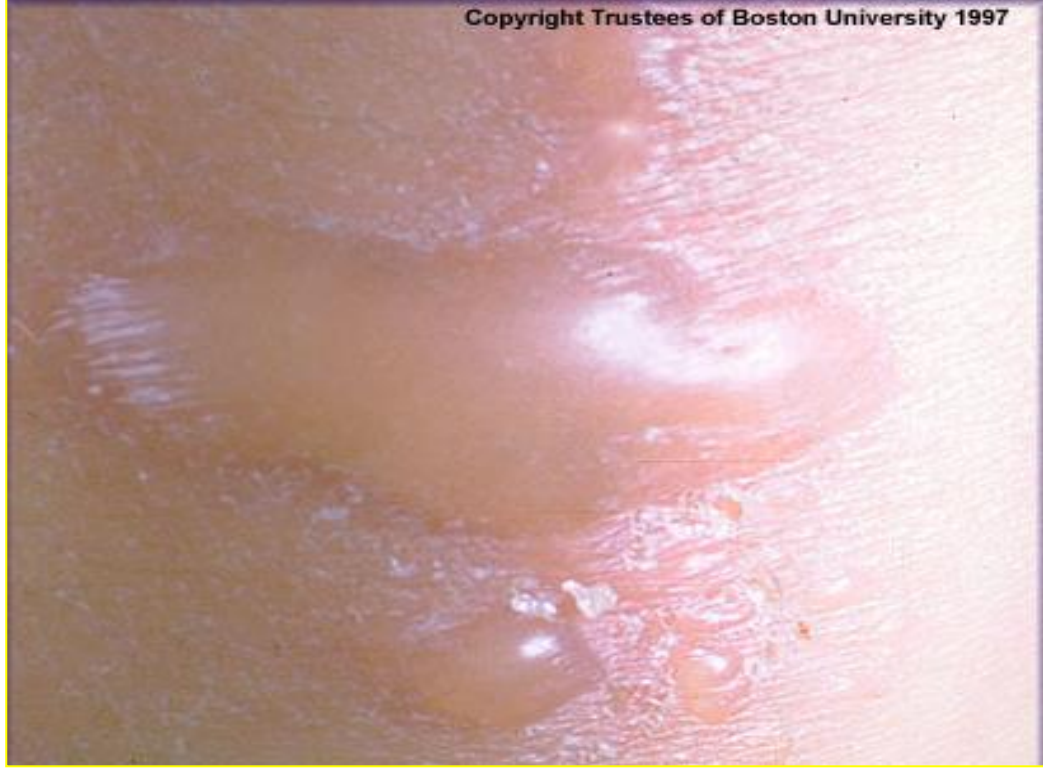
- Serous (high fluid, low protein and cell content)
- Catarrhal
- Fibrinous (exudate is high in plasma proteins especially fibrin; seen in membrane-line body cavities
- Hemorrhagic (Purpura)
- Suppurative or purulent (exudate is rich in neutrophils; abcess, phlegmon, empyeme)
- Ulceration (necrotic and eroded epithelial surface underlying acute and chronic inflammation; trauma, toxins, vascular insufficiency
- Gangrenous
- Pseudomembranous

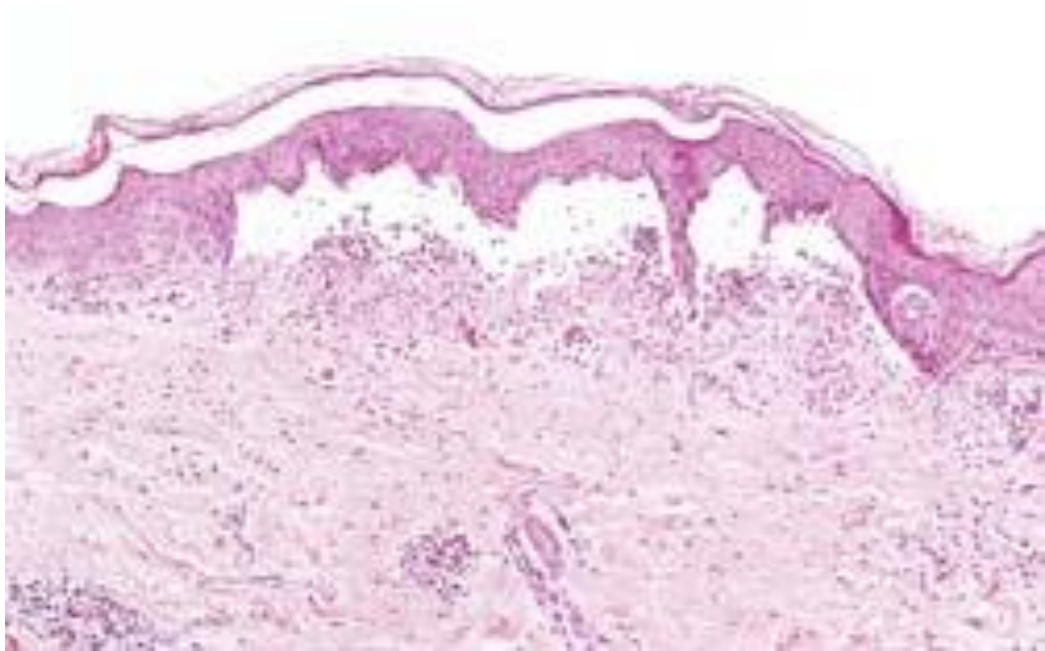
Catarrhal



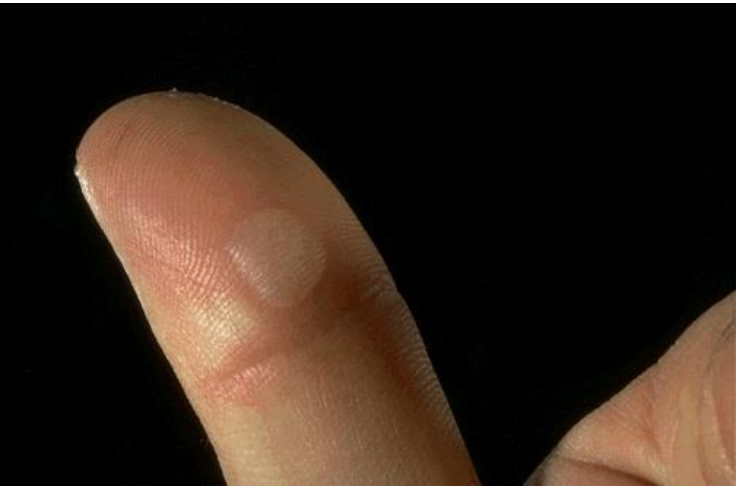
Serous inflammation:

- outpouring of a thin fluid
- is derived from either the plasma or the secretions of mesothelial cells lining the peritoneal, pleural, and pericardial cavities (called *effusion*).

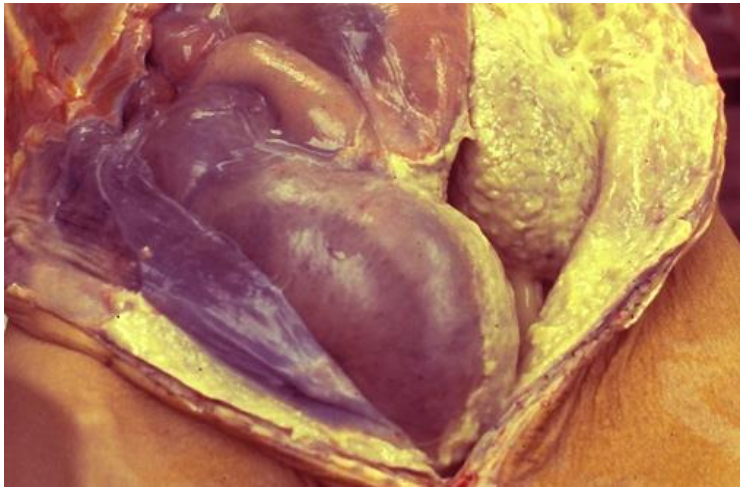




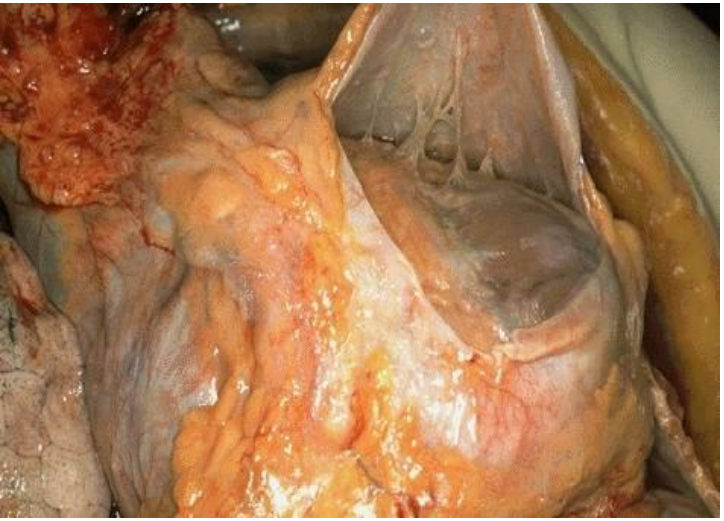
Different morphological patterns of acute inflammation can be found depending on the cause and extend of injury and site of inflammation



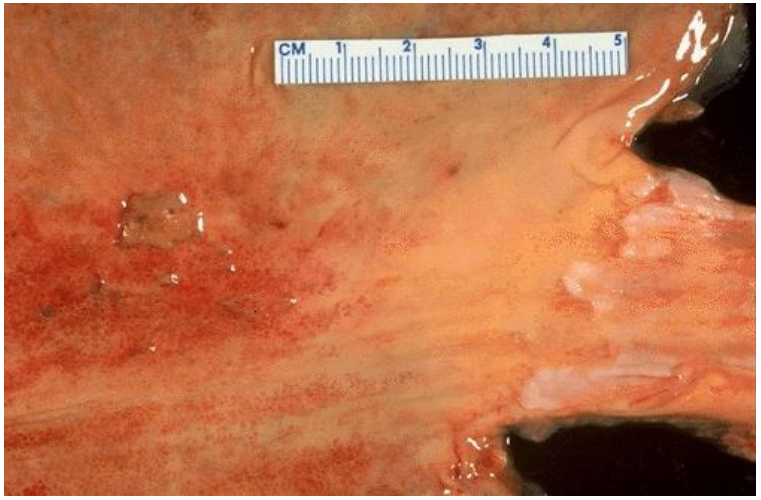
Serous inflammation



Purulent inflammation

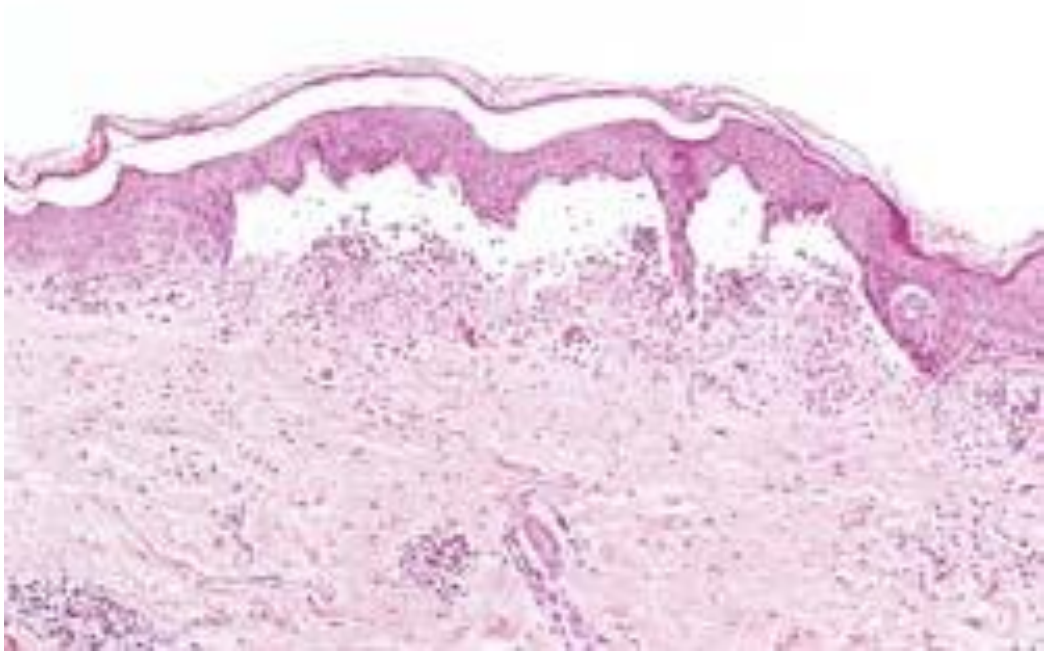


Fibrinous inflammation

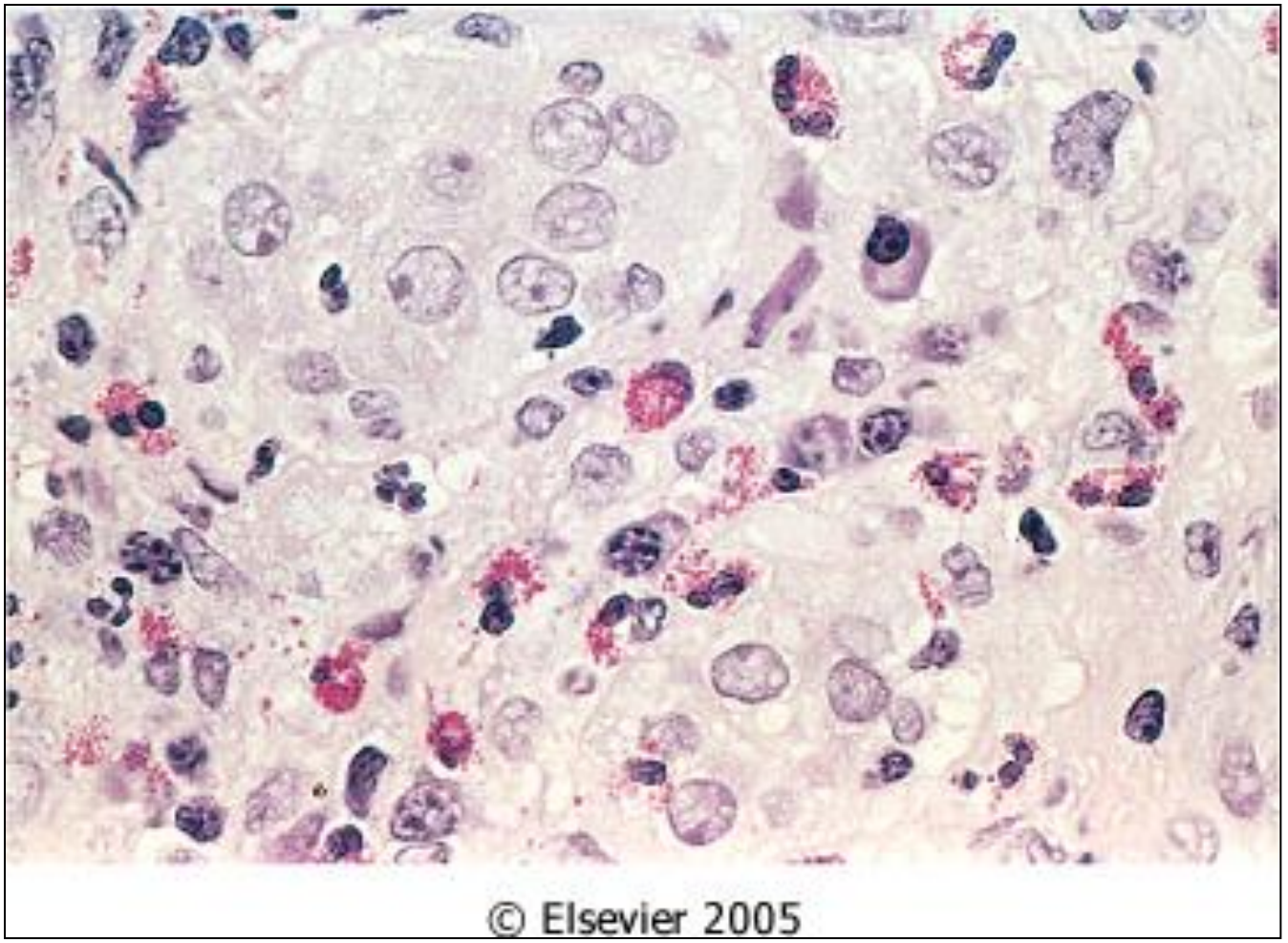


ulcers

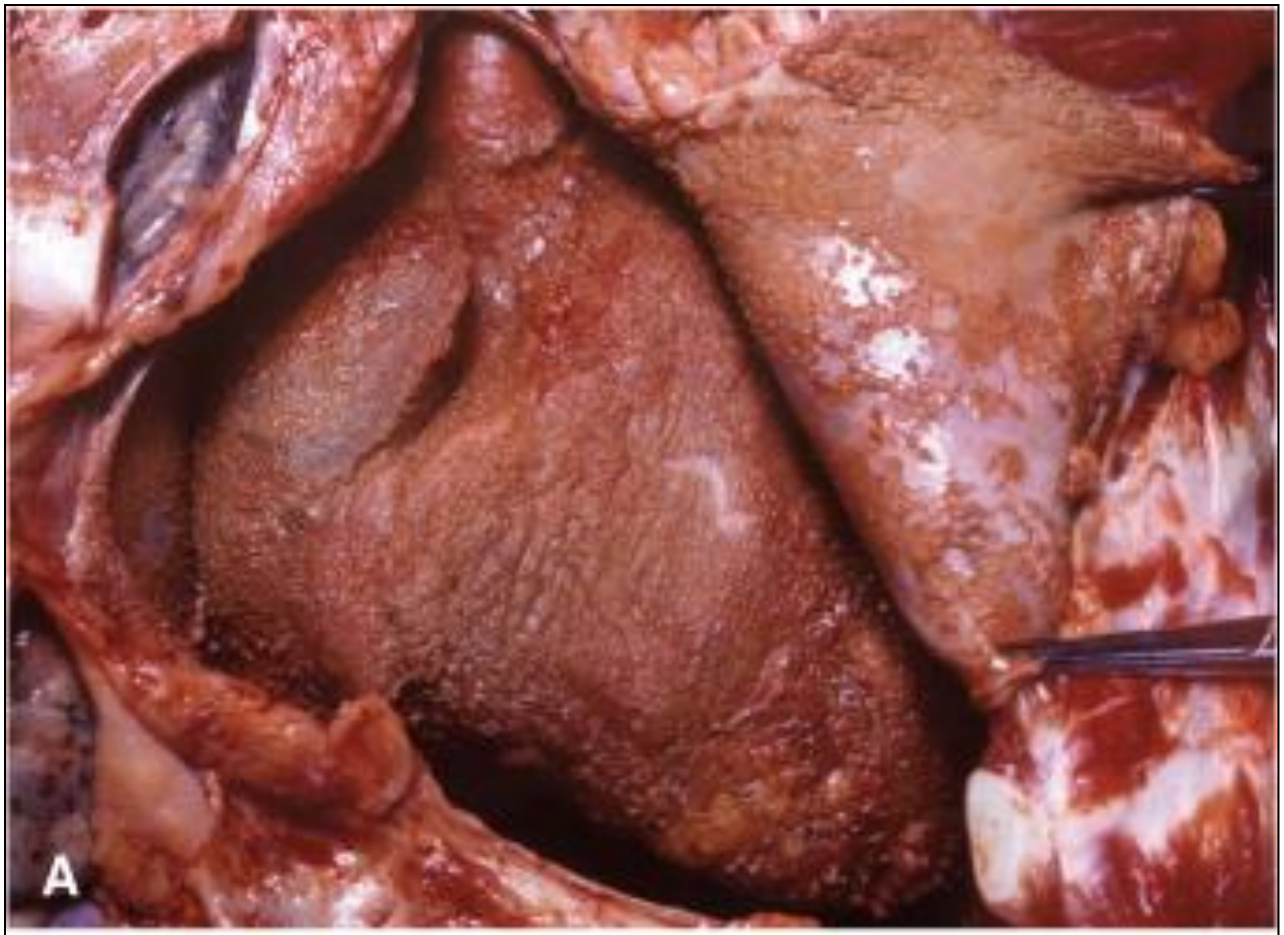
Serous inflammation is marked by the outpouring of a thin fluid that, depending on the size of injury, is derived from either the plasma or the secretions of mesothelial cells lining the peritoneal, pleural, and pericardial cavities (called *effusion*).



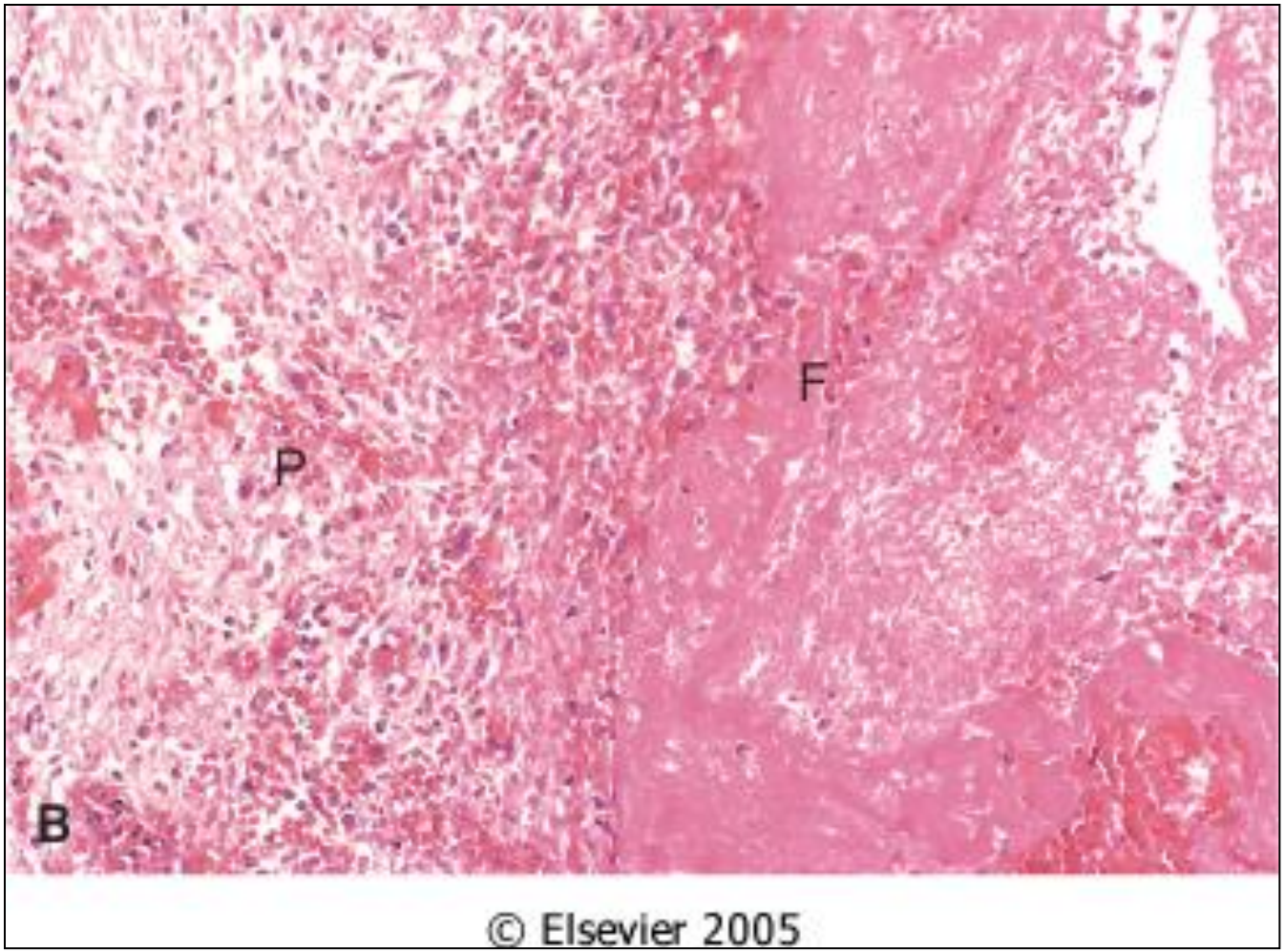
A focus of inflammation showing numerous eosinophils

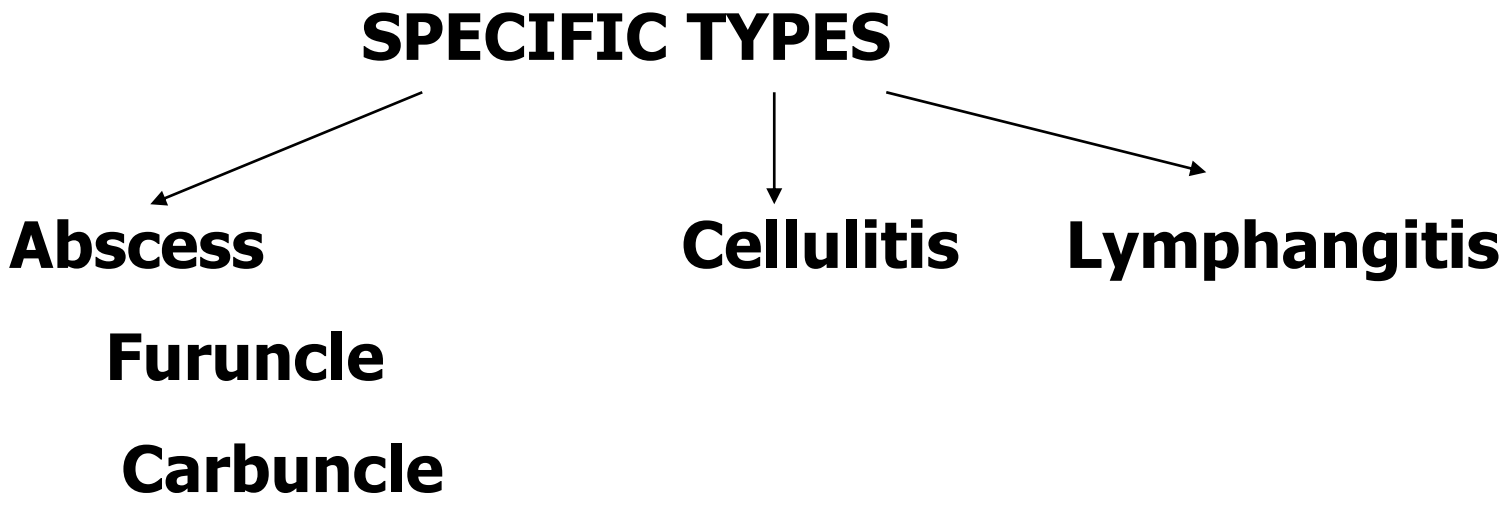


Fibrinous pericarditis
Deposits of fibrin on the pericardium.



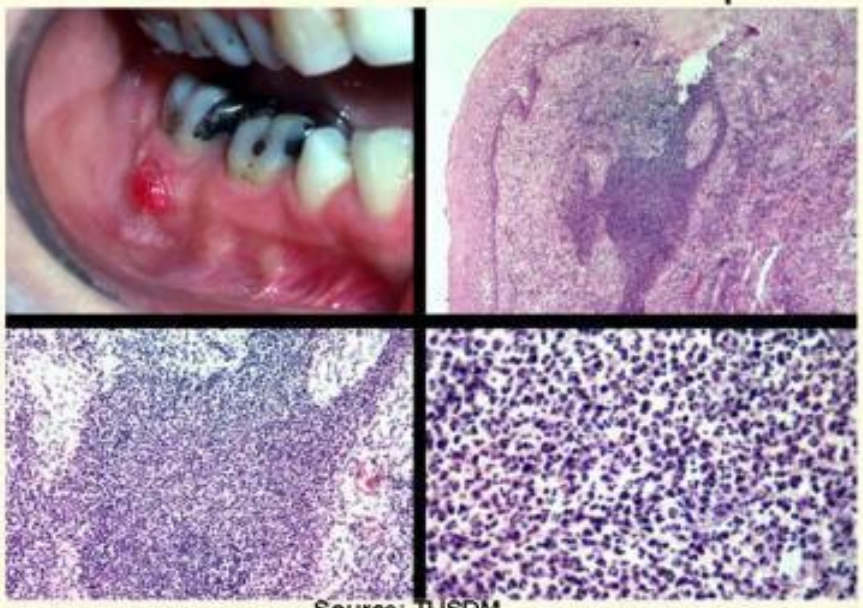
Fibrinous pericarditis: A pink meshwork of fibrin exudate (F) overlies the pericardial surface (P).




SPECIFIC TYPES
Abscess
Furuncle
Carbuncle
Cellulitis
Lymphangitis

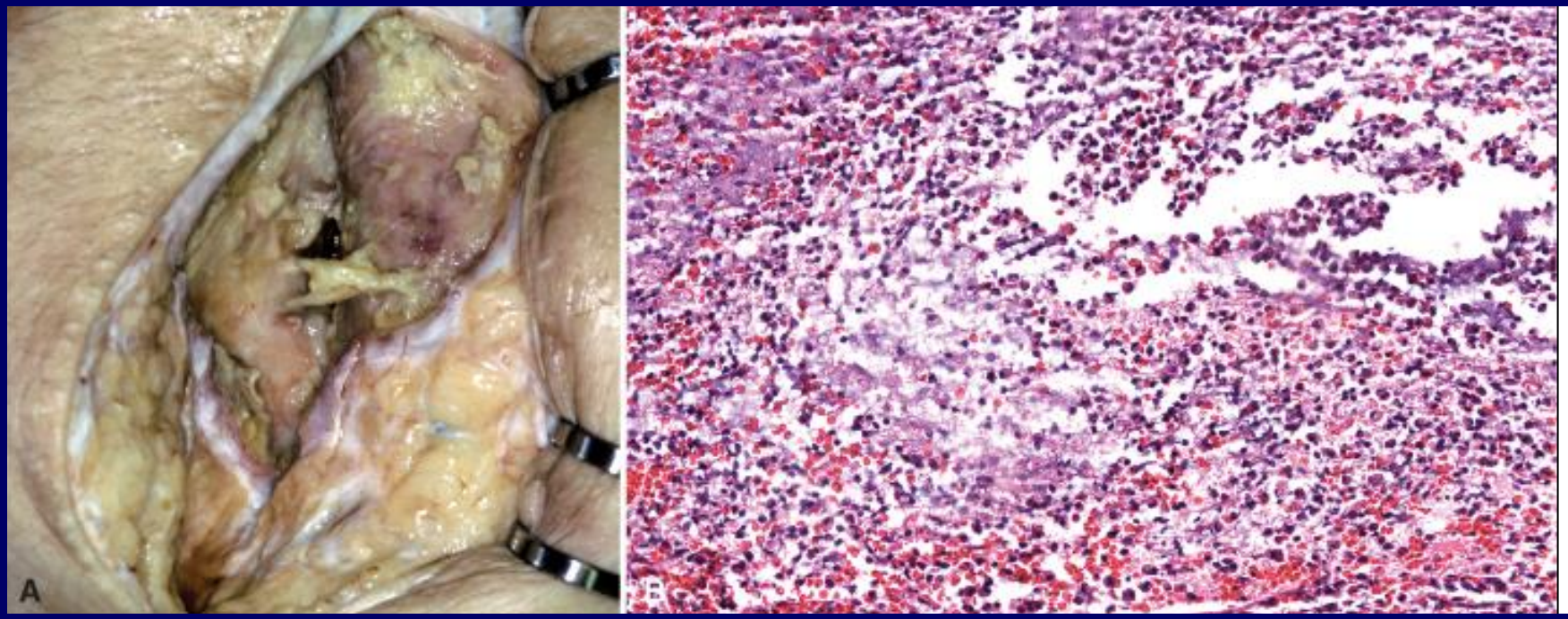
SPECIFIC TYPES

Parulis (gum boil; abscess on the gingiva) = localized accumulation of neutrophils

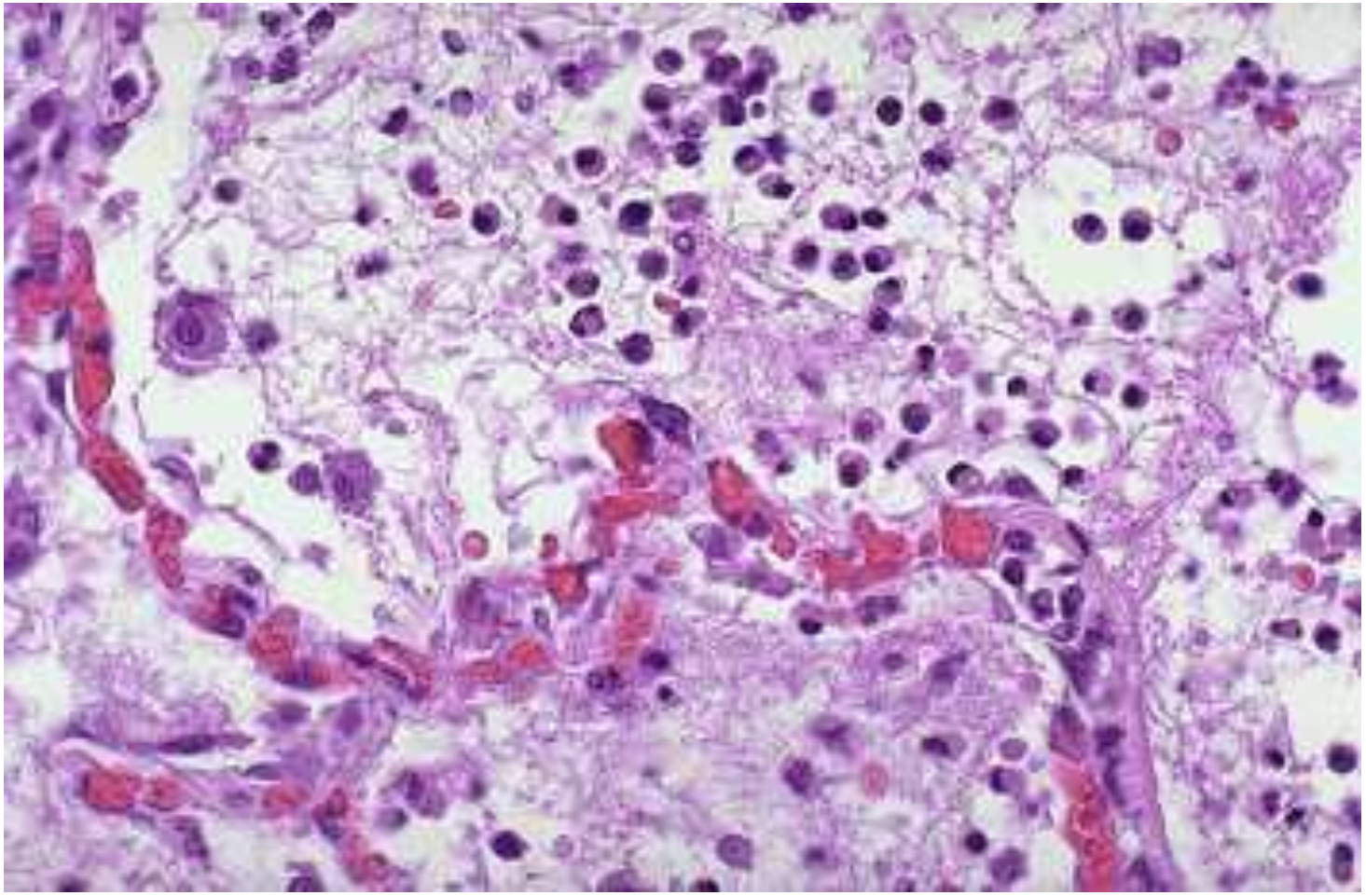


Source: TUSDM

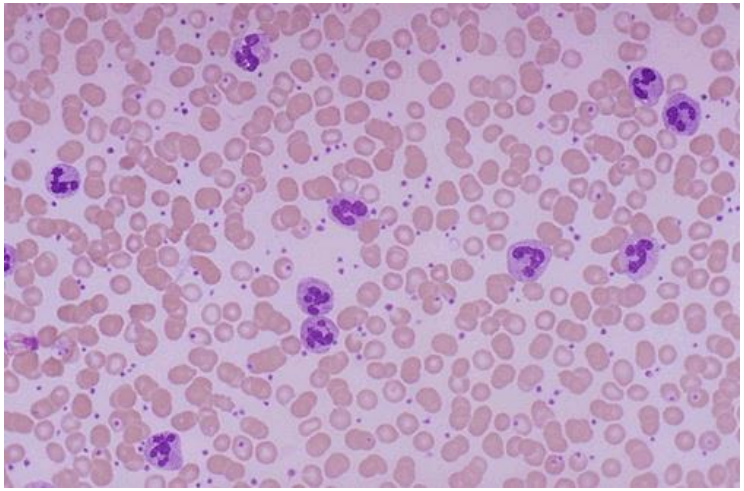
Purulent (Suppurative) inflammation
A, A subcutaneous bacterial abscess with collections of pus.
B, The abscess contains neutrophils, edema fluid, and cellular debris.

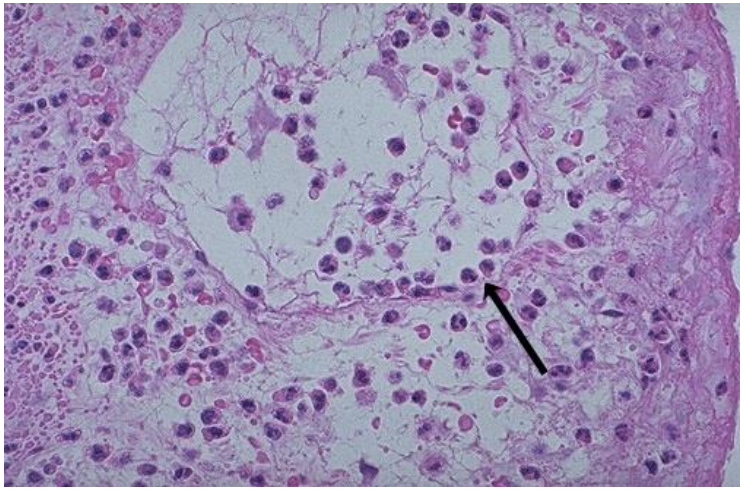


Vascular changes



A critical function of the vascular inflammatory response (stasis and vascular permeability) is to deliver leukocytes to the site of injury in order to clear injurious agents





Neutrophils are commonly the first inflammatory cells (first 6-24 hours) recruited to a site of inflammation.

Extravasation of leukocytes is a coordinated event of:

margination

rolling,

adhesion,

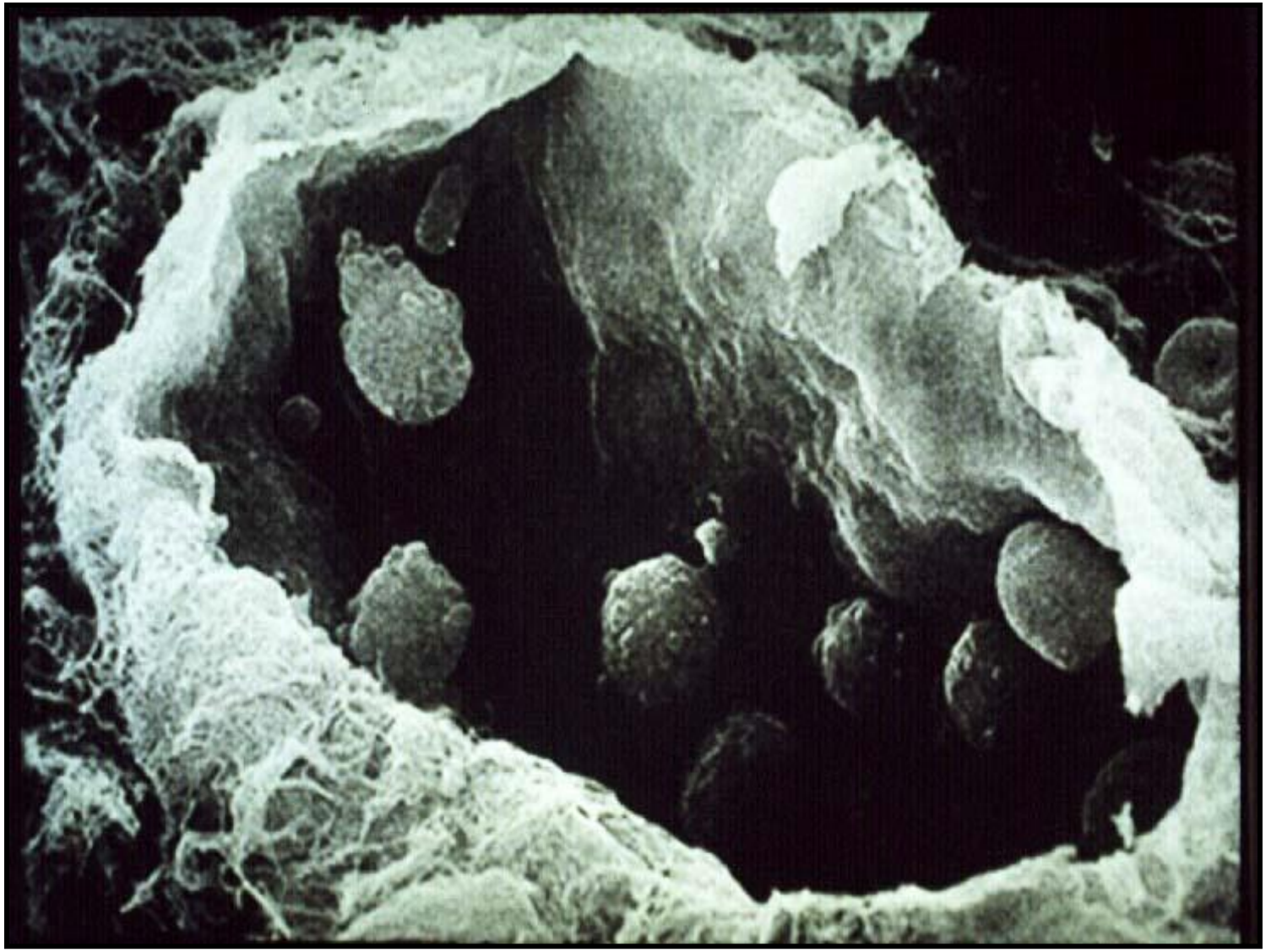
transmigration (diapedesis)

migration.

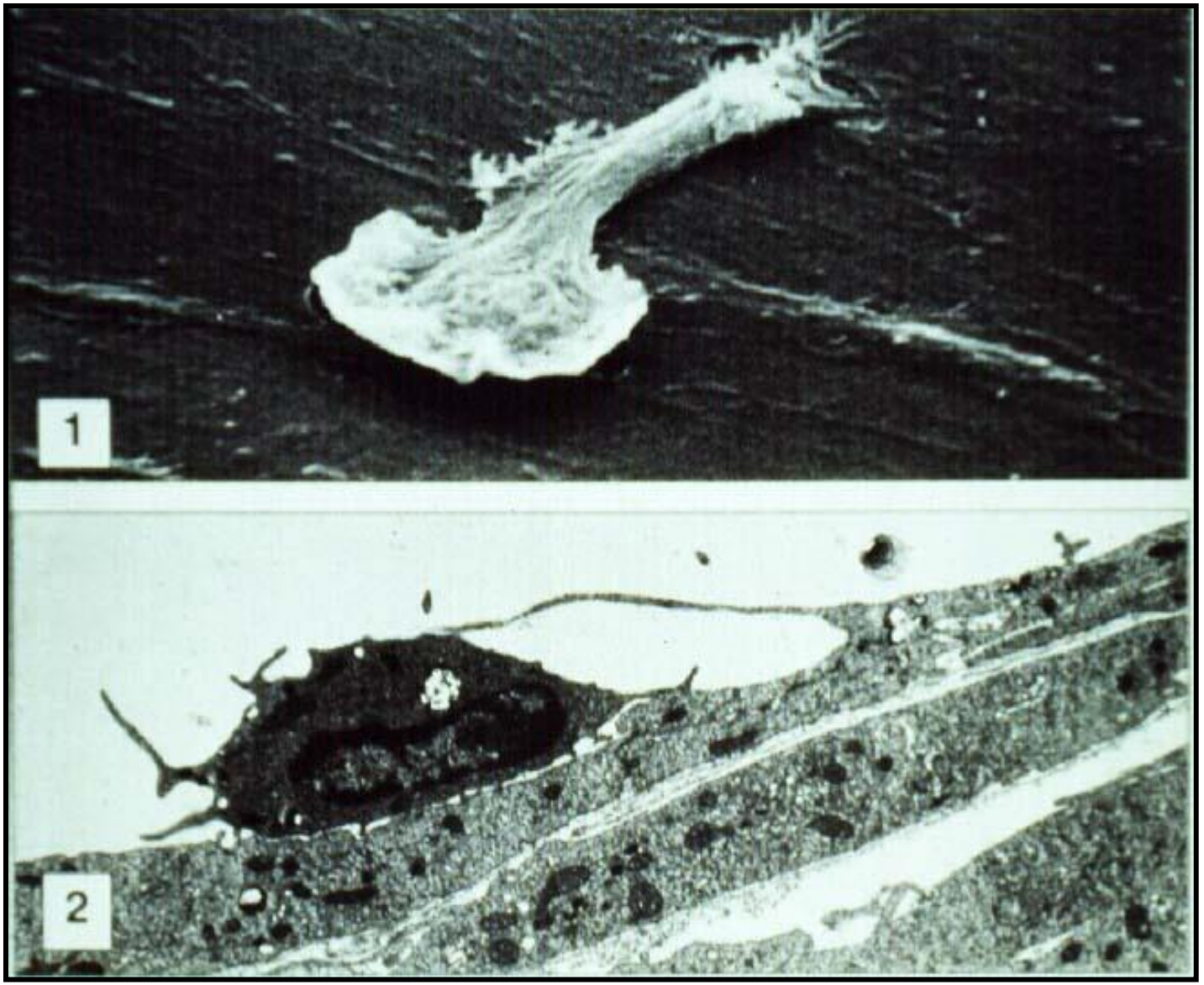
The sequence of events in the journey of leukocytes from the vessel lumen to the interstitial tissue

1. In the lumen: margination, rolling, and adhesion to endothelium. Vascular endothelium normally does not bind circulating cells or impede their passage. In inflammation, the endothelium has to be activated to permit it to bind leukocytes, as a prelude to their exit from the blood vessels.
2. Transmigration across the endothelium (also called diapedesis)
3. Migration in interstitial tissues toward a chemotactic stimulus

Immune cells within a blood vessel



Immune cell traversing endothelium

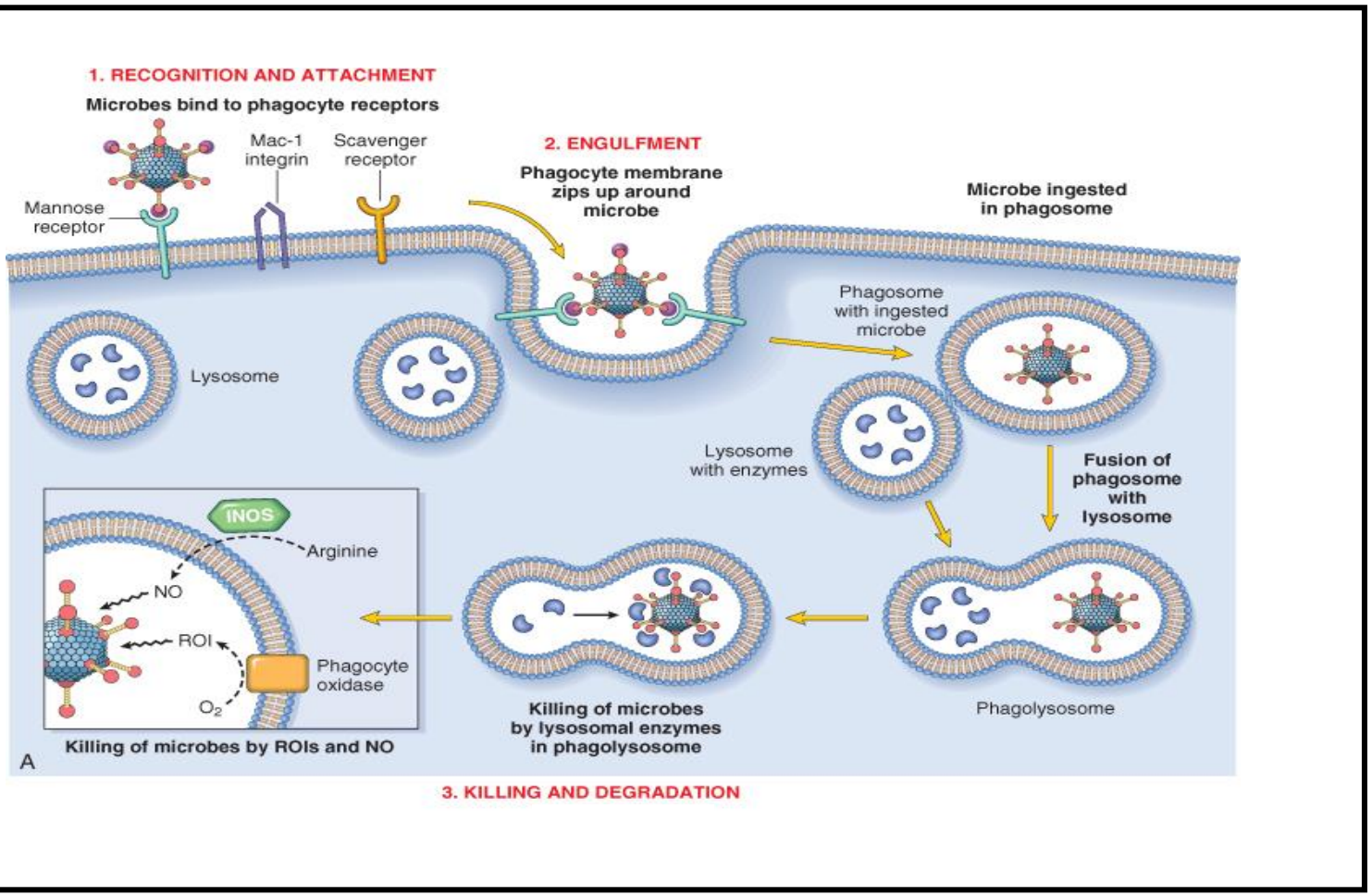


Phagocytosis (engulf and destroy)

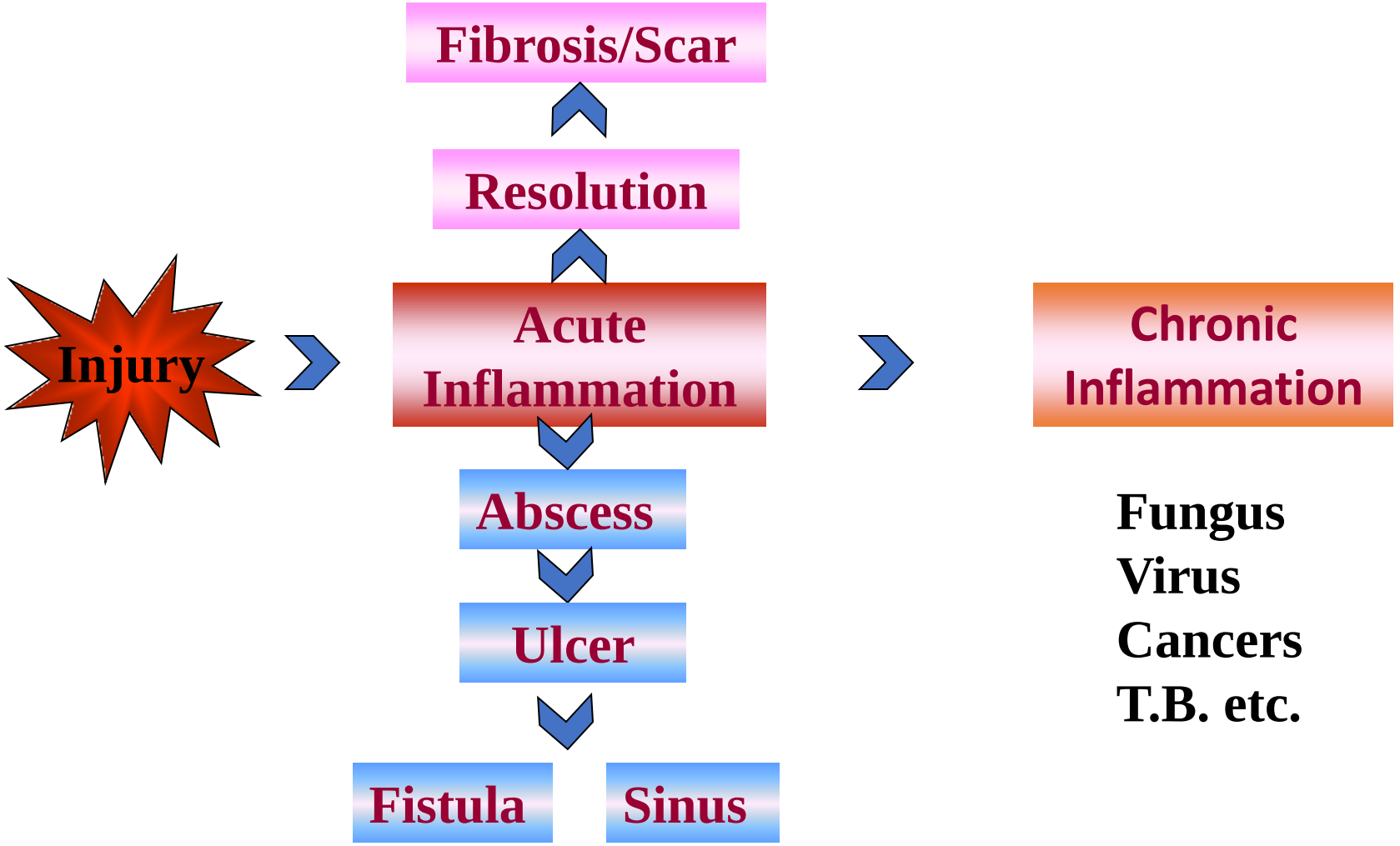
1. Recognition & attachment
   Opsonins (IgG and C3) coat target
2. Engulfment

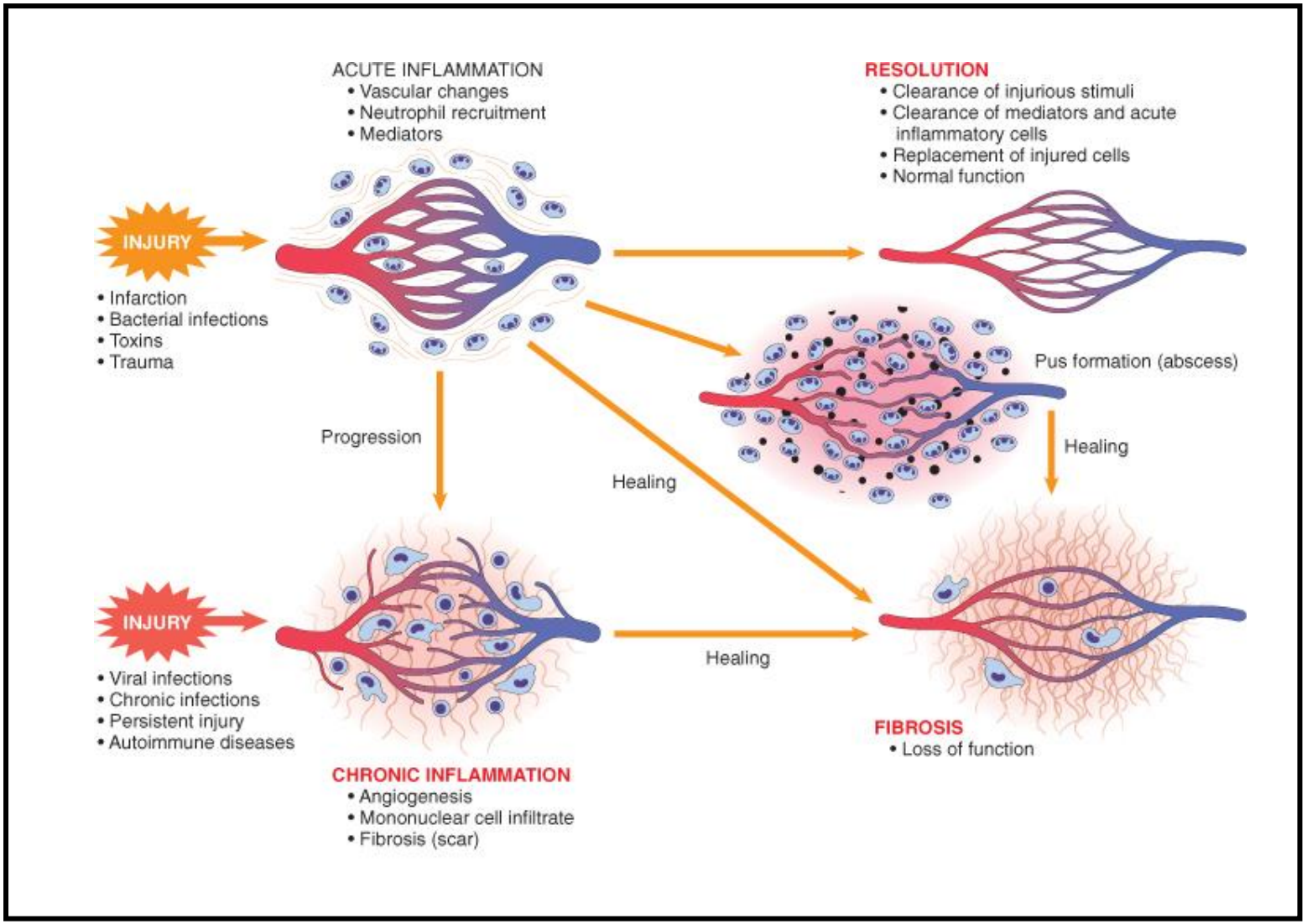
   Pseudopods flow around the particle to be engulfed. Particle is engulfed and fuses with lysosome
3. Killing/degradation
   - O_2 dep: Reactive O_2 species in lysosomes
   - O_2 indep: Bactericidal permeability agents, lysozyme, MBP, lactoferrin

A, Phagocytosis of a particle (e.g., bacterium) involves attachment and binding of Fc and C3b to receptors on the leukocyte membrane, engulfment, and fusion of lysosomes with phagocytic vacuoles, followed by destruction of ingested particles within the phagolysosomes. Note that during phagocytosis, granule contents may be released into extracellular tissues.

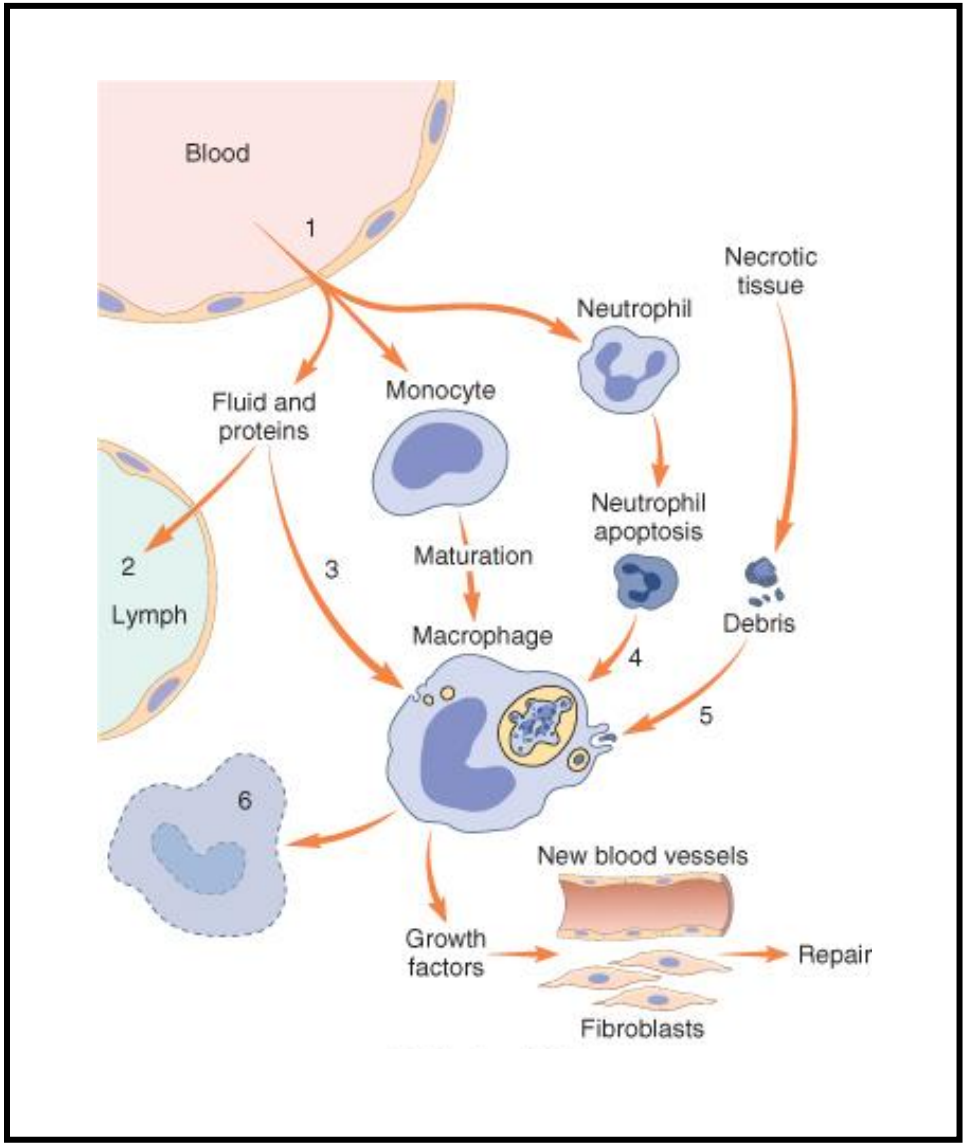


Inflammation Outcome





Outcomes of acute inflammation: resolution, healing by fibrosis, or chronic inflammation



Events in the resolution of inflammation: (1) return to normal vascular permeability; (2) drainage of edema fluid and proteins into lymphatics or (3) by pinocytosis into macrophages; (4) phagocytosis of apoptotic neutrophils and (5) phagocytosis of necrotic debris; and (6) disposal of macrophages. Macrophages also produce growth factors that initiate the subsequent process of repair.

“Never let the competition define you. Instead, you have to define yourself based on a point of view you care deeply about.”

– Tom Chappel


Chronic Inflammation:

Chronic Inflammation

Although difficult to define precisely, *chronic inflammation* is considered to be *inflammation of prolonged duration* (weeks or months) *in which active inflammation*, *tissue destruction*, *and attempts at repair are proceeding simultaneously*. Although it may follow acute inflammation, chronic inflammation frequently begins insidiously, as a low-grade,
smoldering, often asymptomatic response. This latter type of chronic inflammation is the cause of tissue damage in some of the most common and disabling human diseases, such as rheumatoid arthritis, atherosclerosis, tuberculosis, and chronic lung diseases.

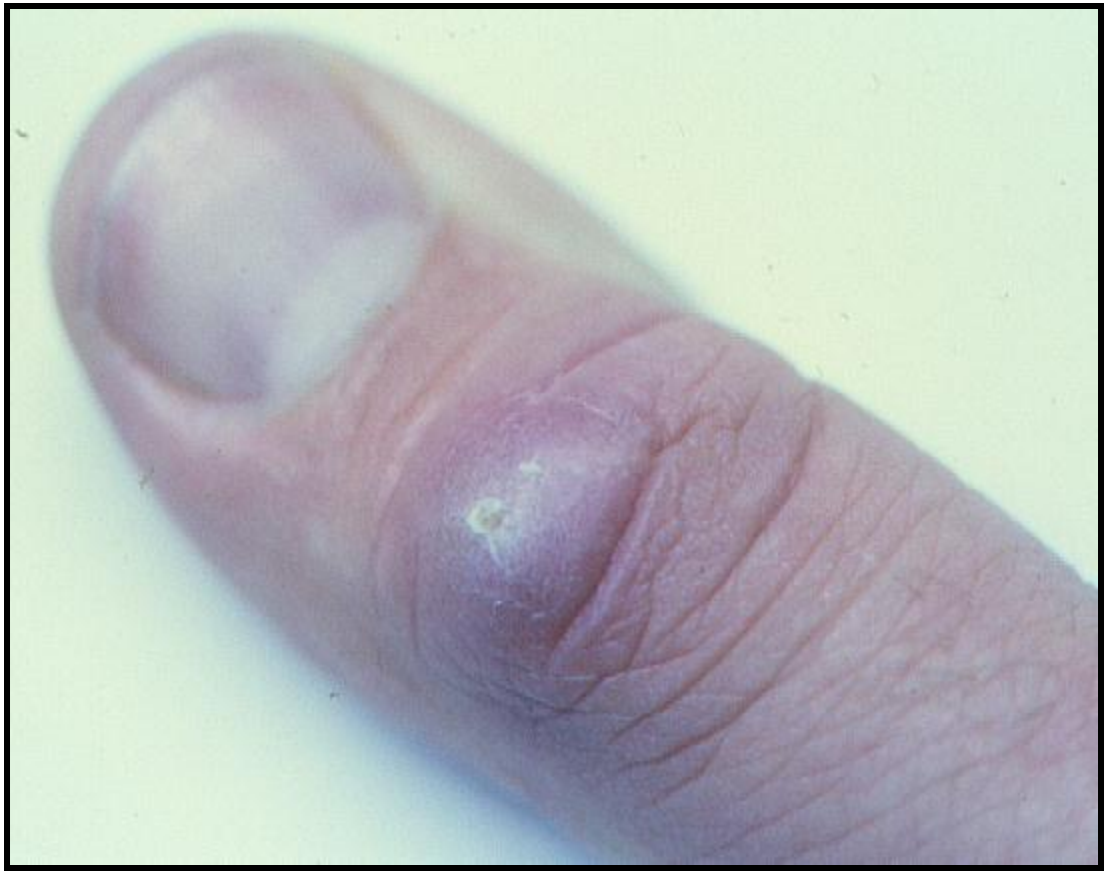
In contrast to acute inflammation, which is manifested by vascular changes, edema, and predominantly neutrophilic infiltration, *chronic inflammation is characterized by:*

- *Infiltration with mononuclear cells*, which include macrophages, lymphocytes, and plasma cells.
- *Tissue destruction*, induced by the persistent offending agent or by the inflammatory cells.
- Attempts at *healing by connective tissue replacement of damaged tissue*, accomplished by proliferation of small blood vessels (*angiogenesis*) and, in particular, *fibrosis*

Table 5–1. Differences between Acute and Chronic Inflammation.

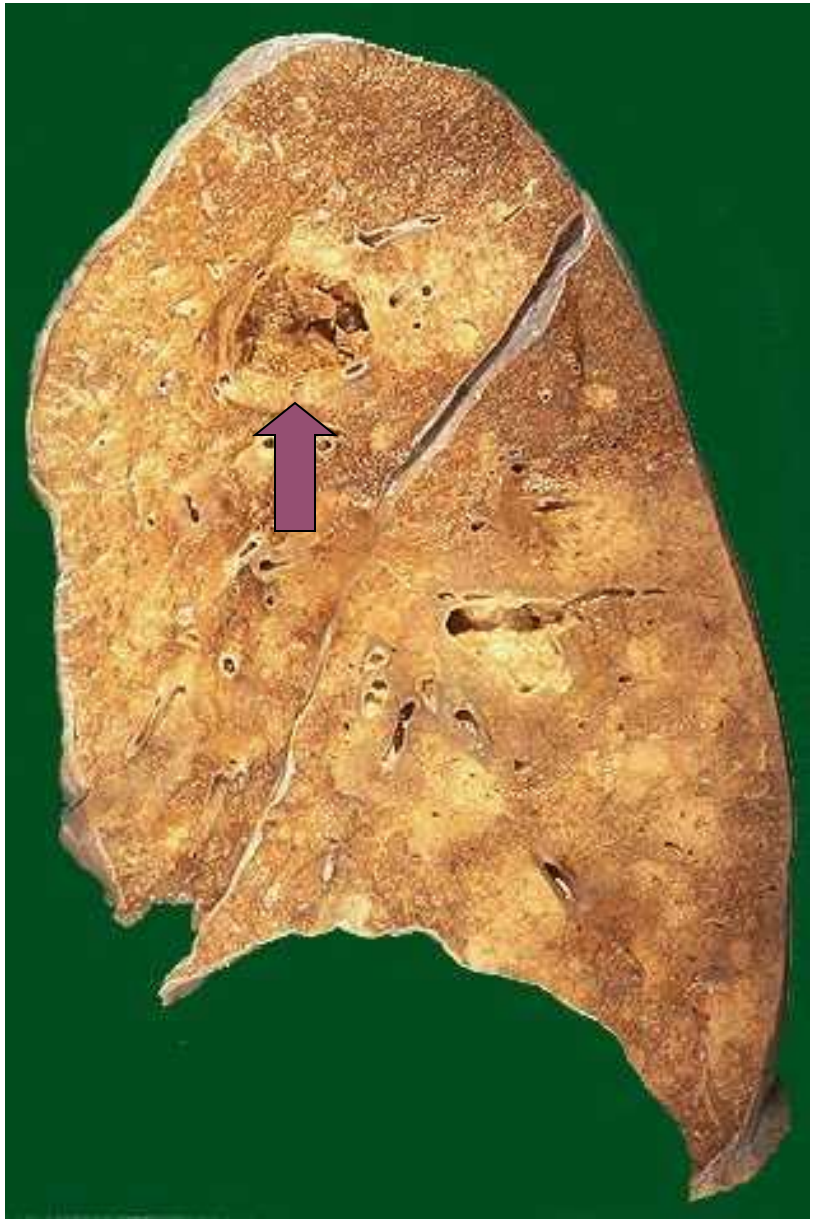
	Acute	Chronic
Duration	Short (days)	Long (weeks to months)
Onset	Acute	Insidious
Specificity	Nonspecific	Specific (where immune response is activated)
Inflammatory cells	Neutrophils, macrophages	Lymphocytes, plasma cells, macrophages, fibroblasts
Vascular changes	Active vasodilation, increased permeability	New vessel formation (granulation tissue)
Fluid exudation and edema	+	–
Cardinal clinical signs (redness, heat, swelling, pain)	+	–
Tissue necrosis	– (Usually) + (Suppurative and necrotizing inflammation)	+ (ongoing)
Fibrosis (collagen deposition)	–	+
Operative host responses	Plasma factors: complement, immunoglobulins, properdin, etc; neutrophils, nonimmune phagocytosis	Immune response, phagocytosis, repair
Systemic manifestations	Fever, often high	Low–grade fever, weight loss, anemia
Changes in peripheral blood	Neutrophil leukocytosis; lymphocytosis (in viral infections)	Frequently none; variable leukocyte changes, increased plasma immunoglobulin

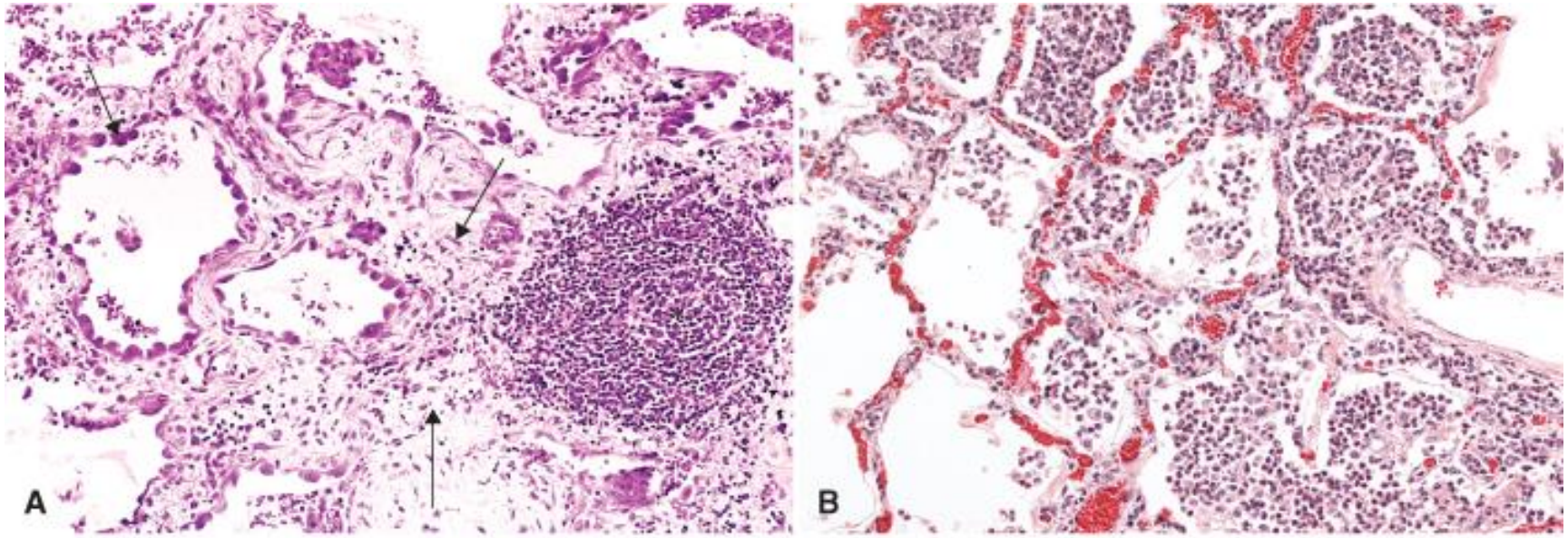
Fish Tank Granuloma
Mycobacterium marinum



Chronic Inflammation:

Lung Abscess

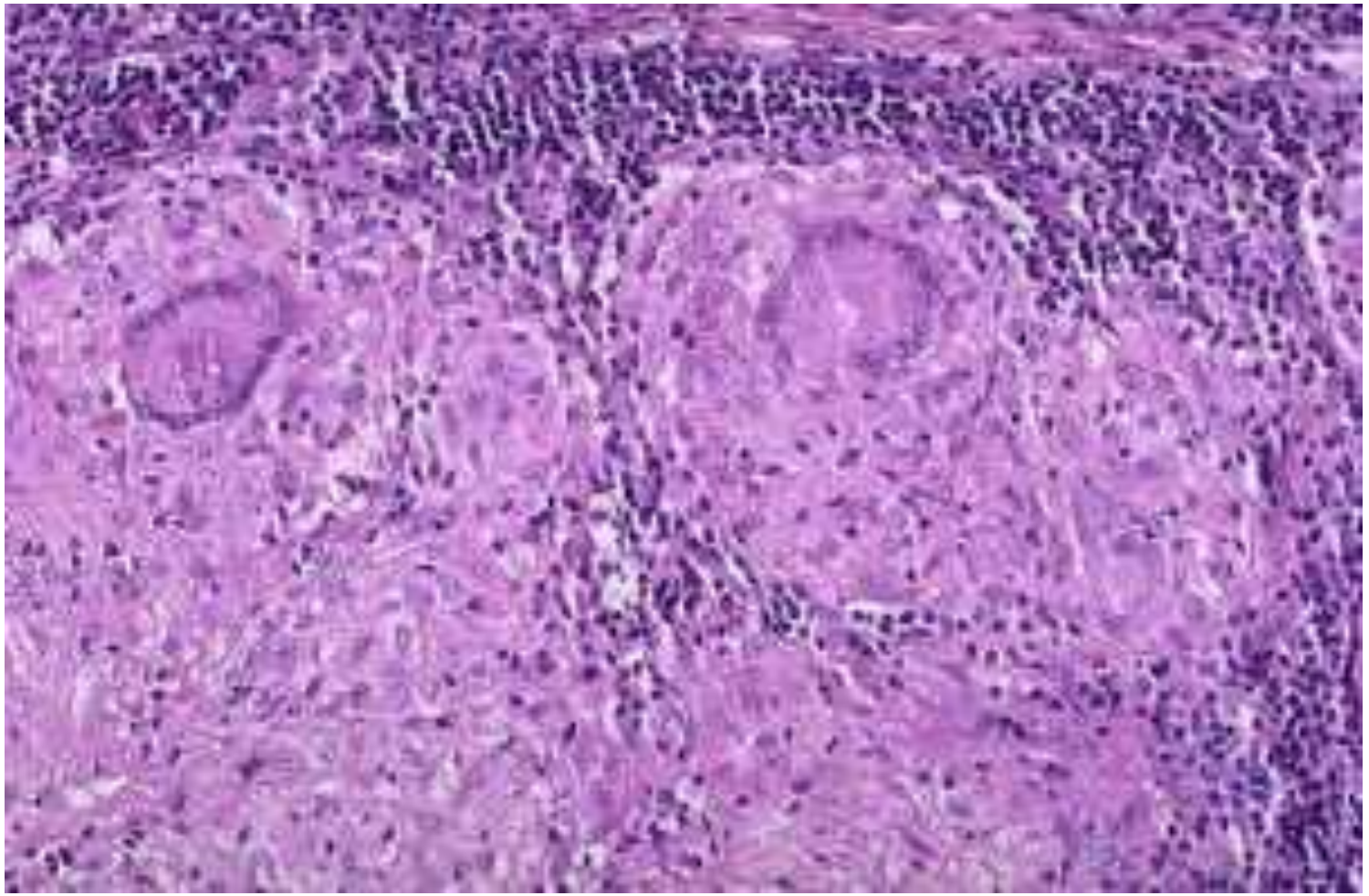


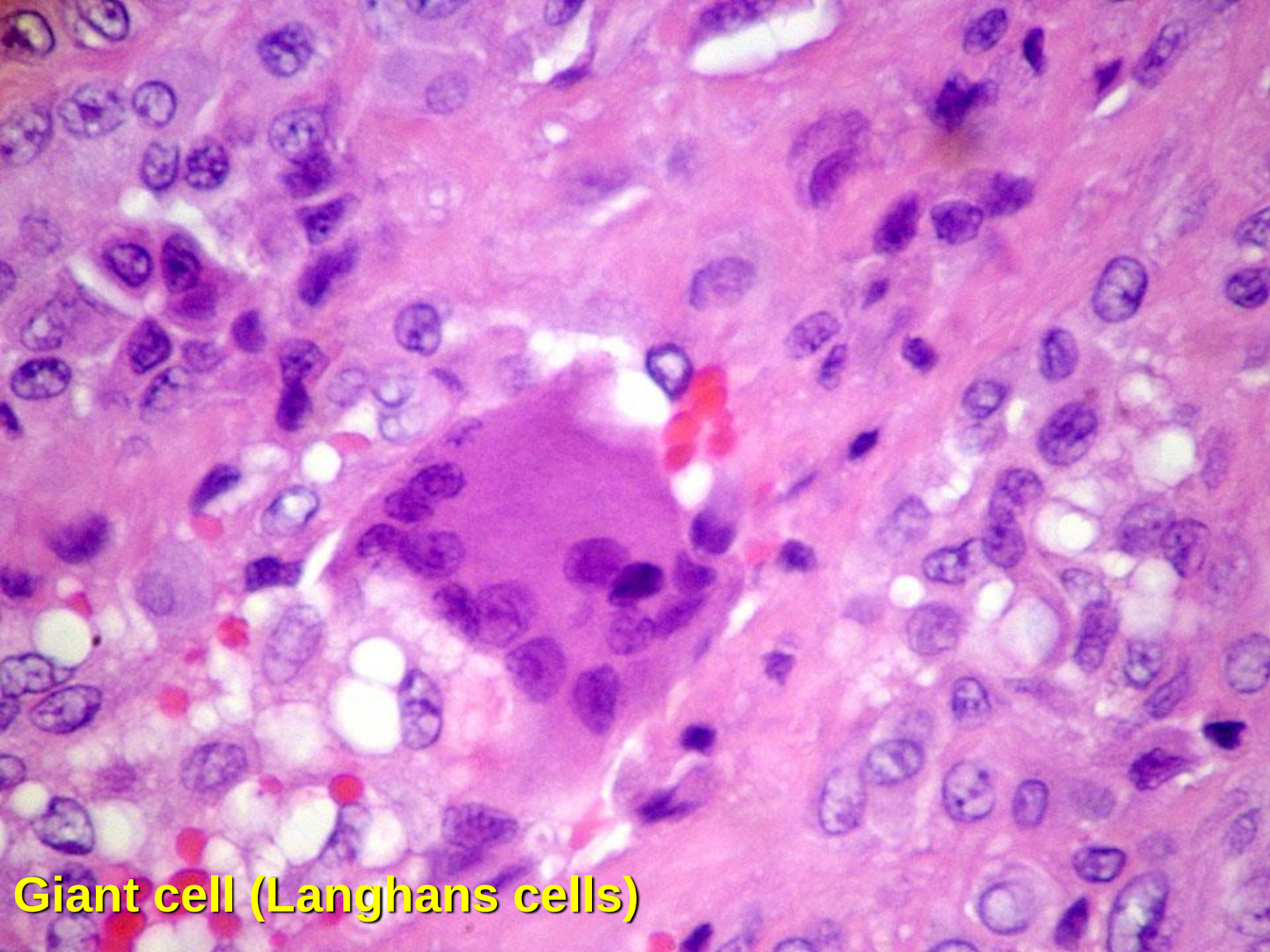


A, Chronic inflammation in the lung, showing all three characteristic histologic features: (1) collection of chronic inflammatory cells, (2) destruction of parenchyma (alveoli are replaced by spaces lined by cuboidal epithelium, arrowheads), and (3) replacement by connective tissue (fibrosis, arrows).

B, By contrast, in acute inflammation of the lung (acute bronchopneumonia), neutrophils fill the alveolar spaces and blood vessels are congested.

Granuloma:




Giant cell (Langhans cells)