

Inflammation I.

Dr. József Tímár



Inflammation

The reaction of vascularized living tissue to local injury

Causes of inflammation

- Mikroorganisms (bacteria, fungus, viruses)
- Zoonosis (worms, protozoa, insects)
- Toxic agents
- Mechanic agents
- Termic agents
- Radioactivity
- Endogenous toxins (uraemia)
- Tumours
- Necrosis

Phasis of inflammation

- Acute
- Subacute
- Chronic



Main symptoms of inflammation (GALENUS)

- Rubor
- Calor
- Dolor
- Tumor
- Functio laesa



ACUTE INFLAMMATION

Progress:

- Vascular changes
- Cellular changes

Cause



**Inflammatory
mediators**



Vasoactive mediators:

- histamin
- serotonin
- bradykinin
- anaphylatoxin
- LT, PG, PAF



**Increased
permeability**



OEDEMA

Chemotactic factors:

- C5a
- LTB
- peptids (formilated)
- lymphokins
- monokine



**Chemotaxis + activating
of inflammatory cells**



**ACUT
INFLAMMATION:**

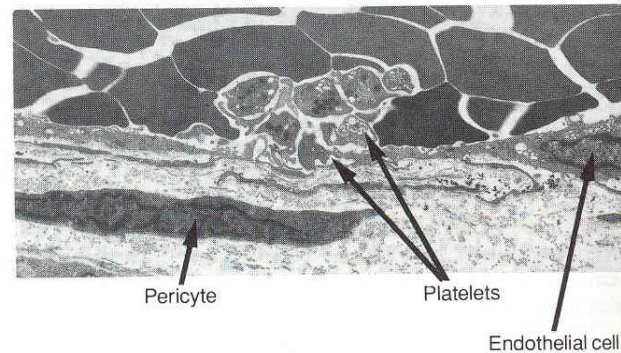
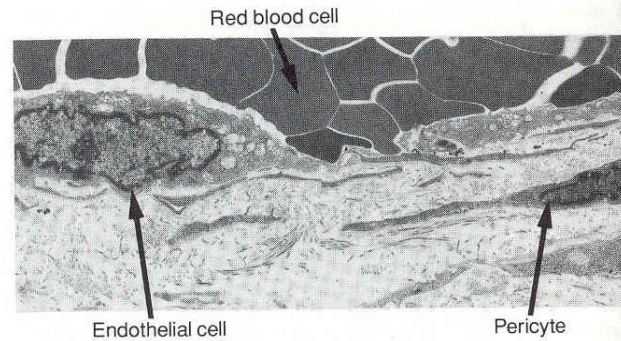
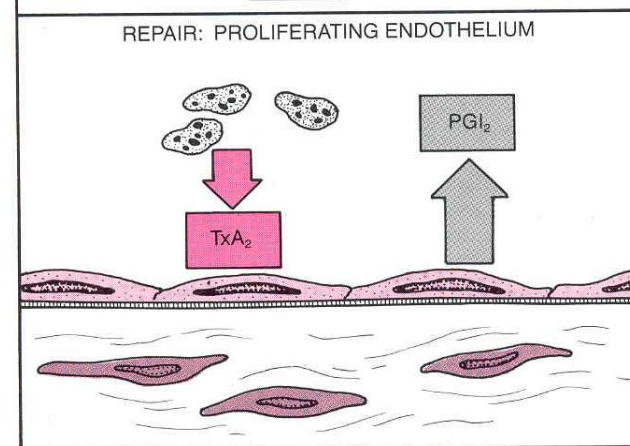
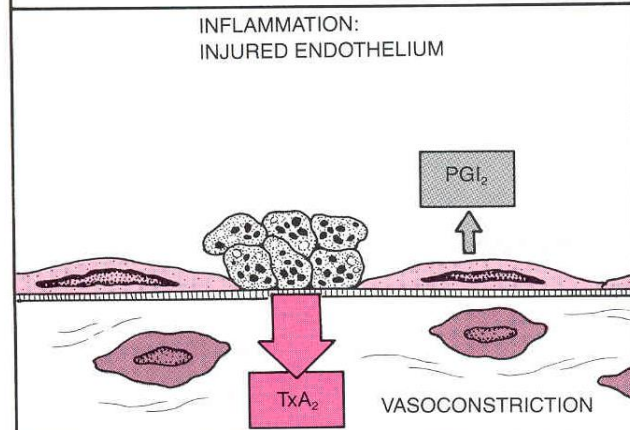
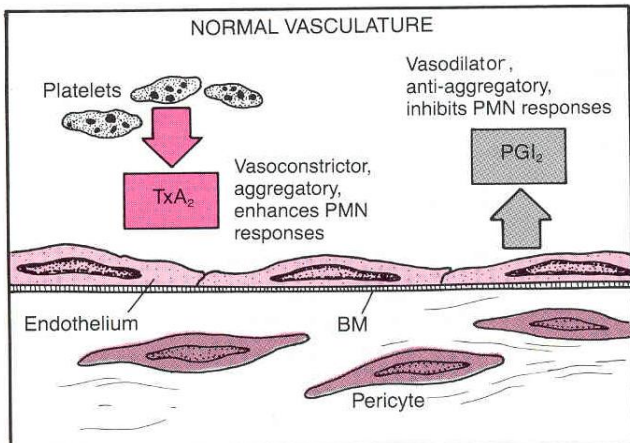
- neutrophils
- platelets

**CHRONIC
INFLAMMATION:**

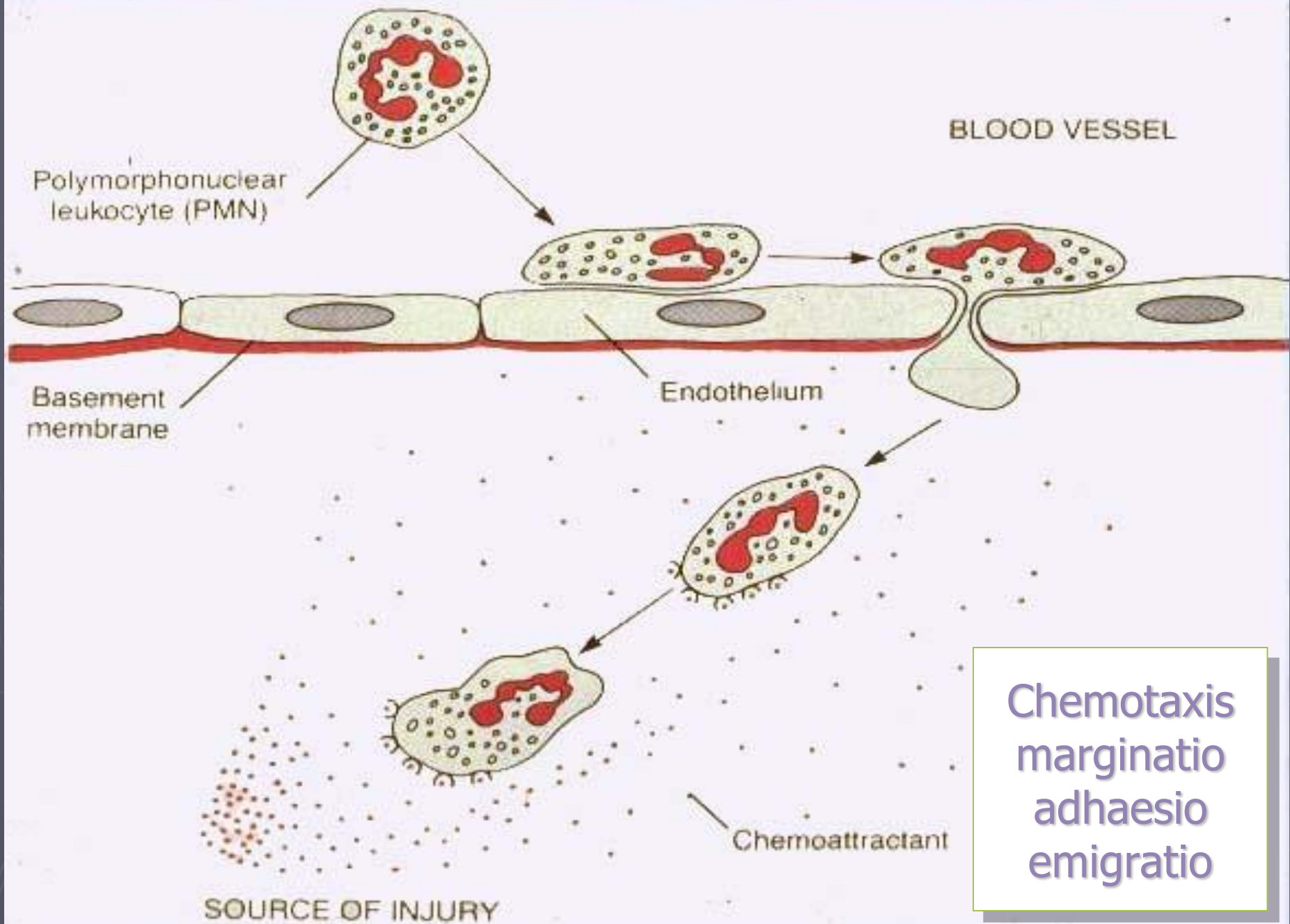
- macrophages
- lymphocytes
- plasma cells

Function of endothelial cells

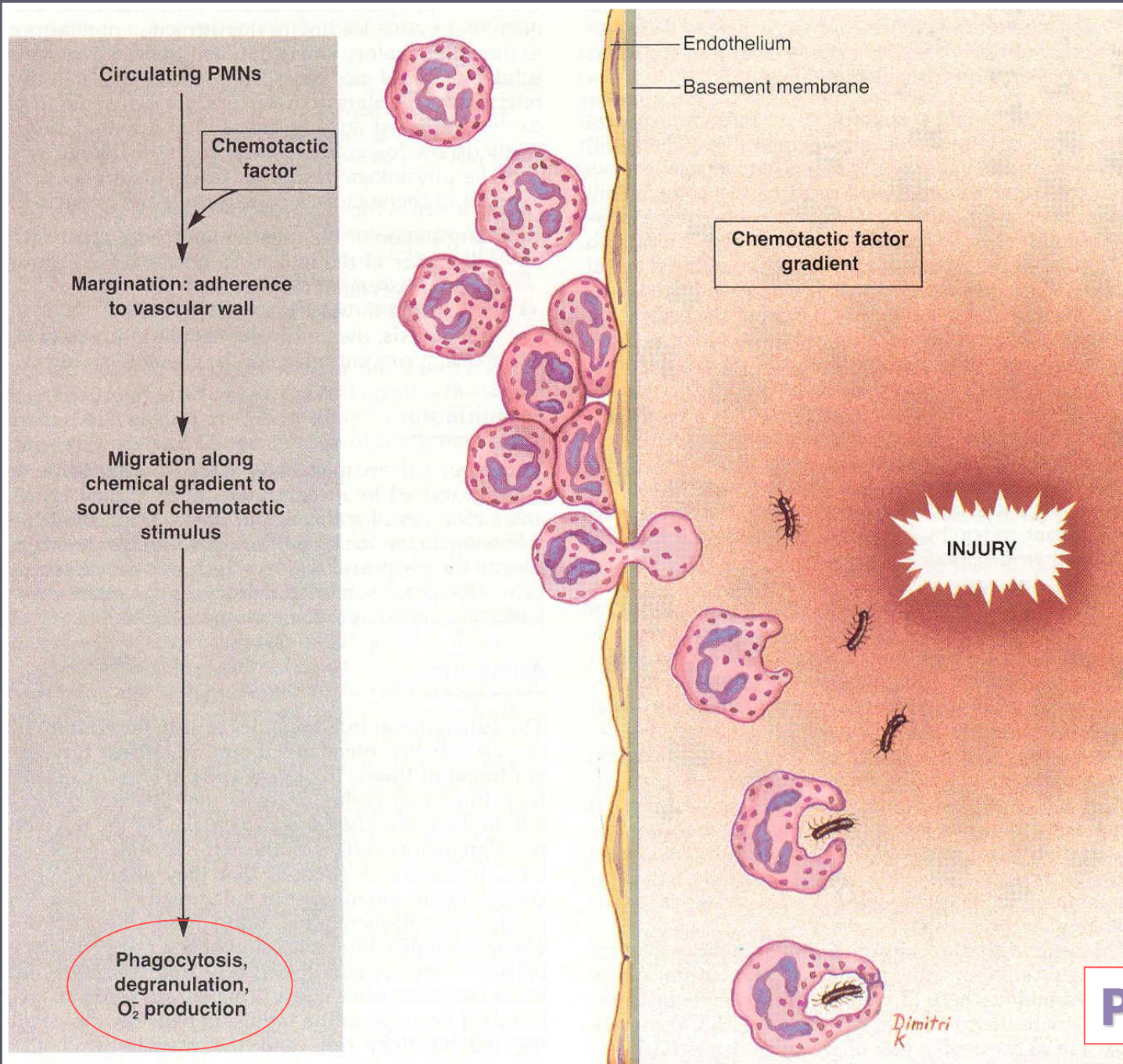
- **Regulation of permeability**
 - Tight junction, gap junction
 - Vesicula
 - Transendothelial channels
- **Metabolic activity**
 - PGI₂, Plasminogen activator
 - VIII. factor, vW factor, ACE
 - EDRF, endothelin
 - Extracellular matrix production
- **Uptake & degradation**
 - Catecholamins, vasoactive amins, LDL



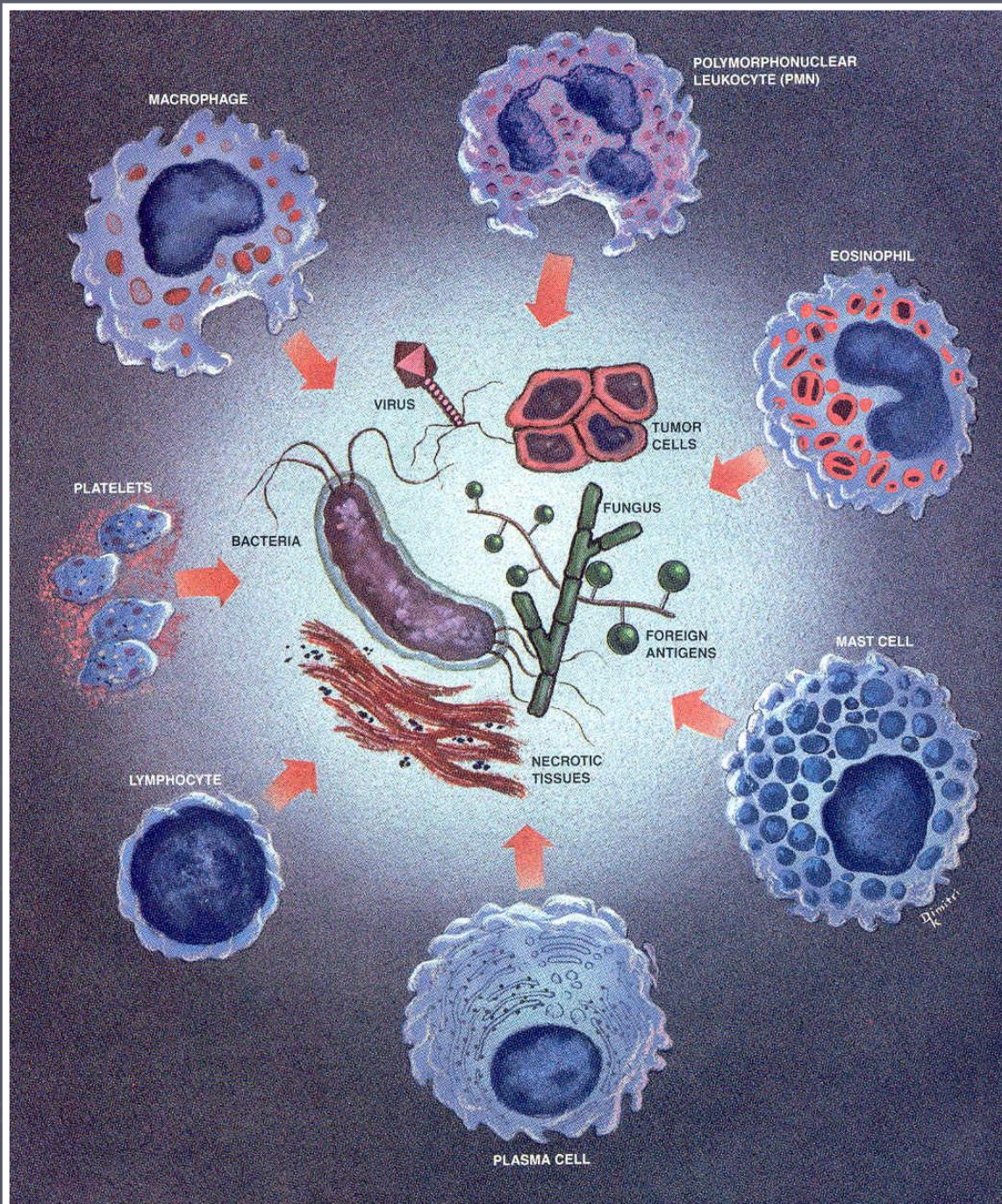
Cells
+
vessels



Chemotaxis
marginatio
adhaesio
emigratio



Phagocytosis



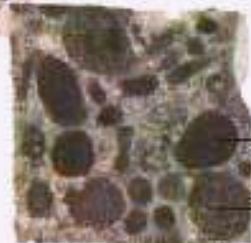
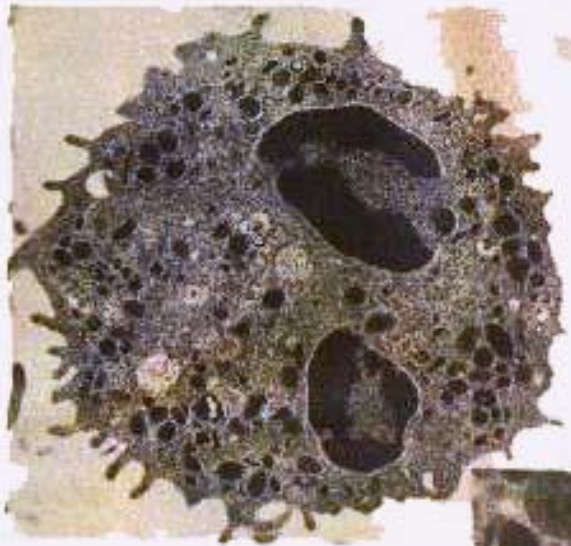
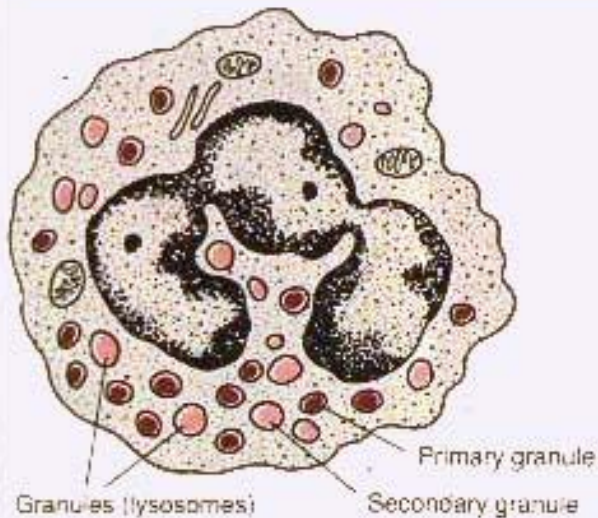
Inflammatory cells

CHARACTERISTICS AND FUNCTIONS

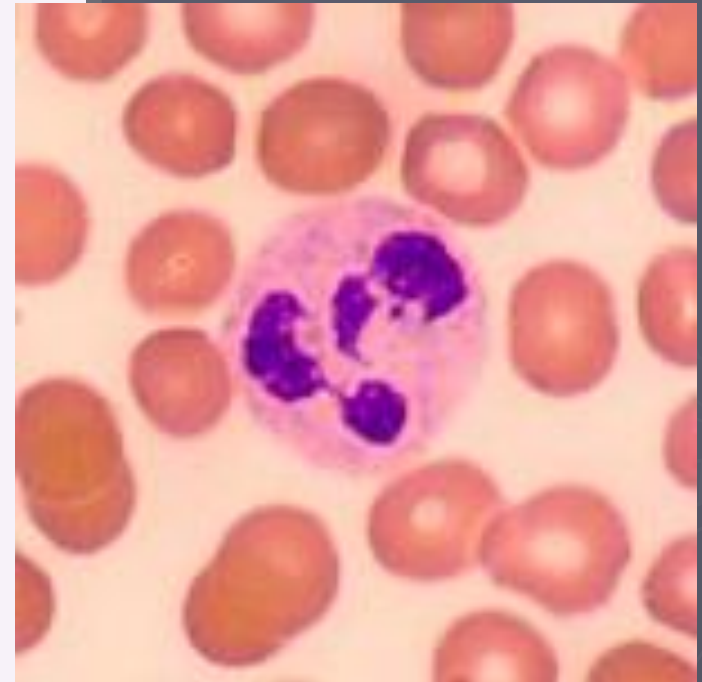
- Acute inflammation
- Bacterial and foreign body phagocytosis

PMN INFLAMMATORY MEDIATORS

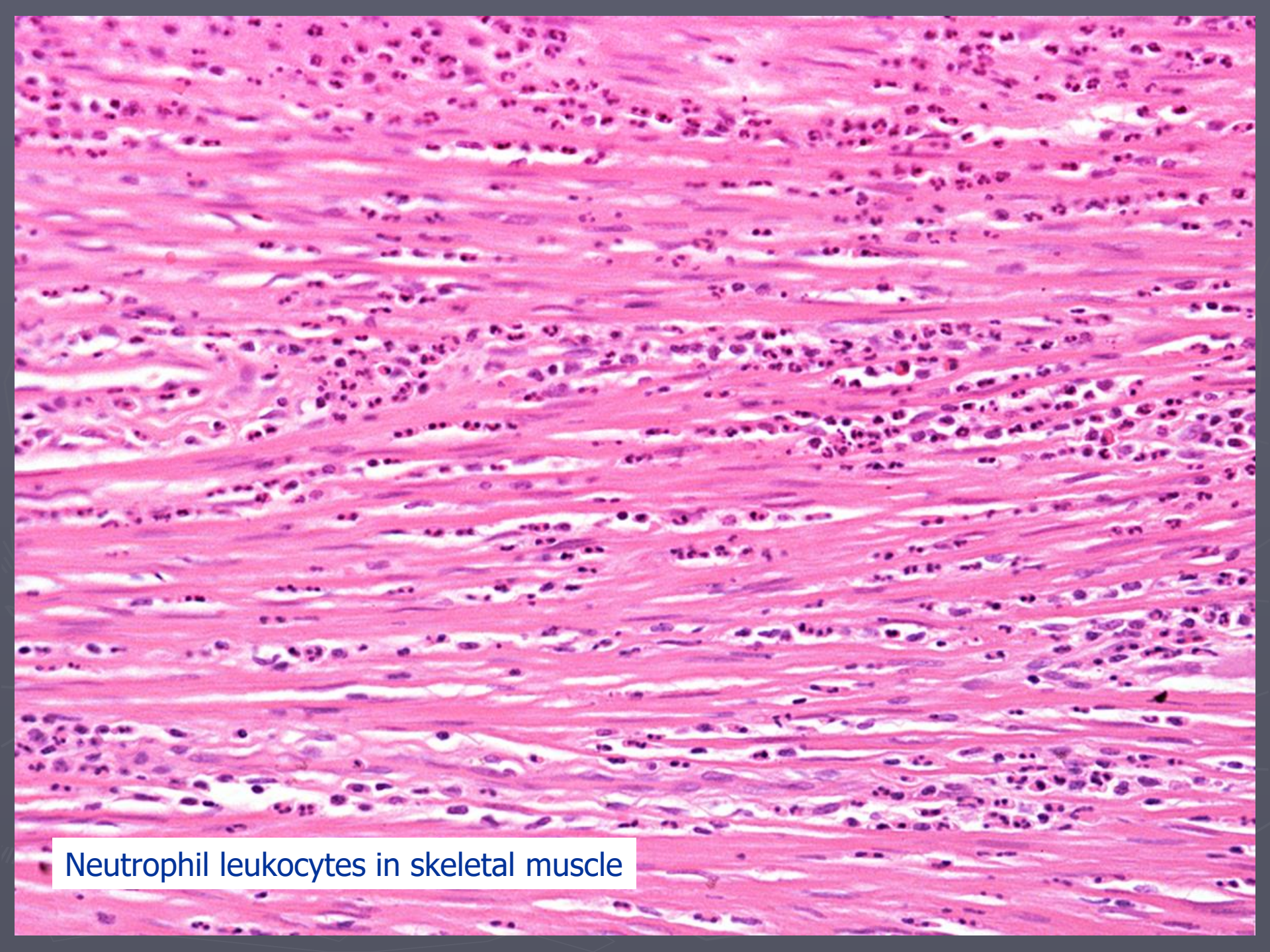
- Reactive oxygen metabolites
- Lysosomal granule contents
 - **Primary granules**
 - Phospholipase A₂
 - Myeloperoxidase
 - Lysozyme
 - Cationic proteins
 - Acid hydrolases
 - Elastase
 - Cathepsins
 - **Secondary granules**
 - Phospholipase A₂
 - Lysozyme
 - Alkaline phosphatase
 - Collagenase
 - Lactoferrin
 - Vitamin B₁₂ binding proteins
 - **Tertiary or C particles**
 - Gelatinase
 - Cathepsins



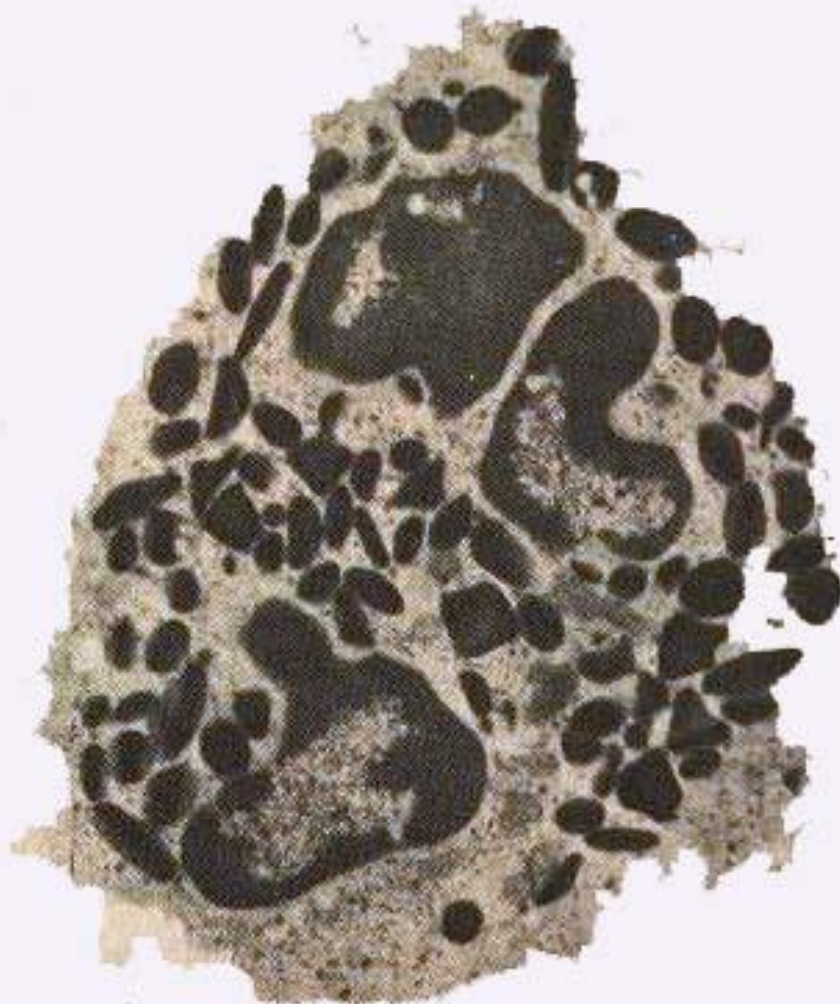
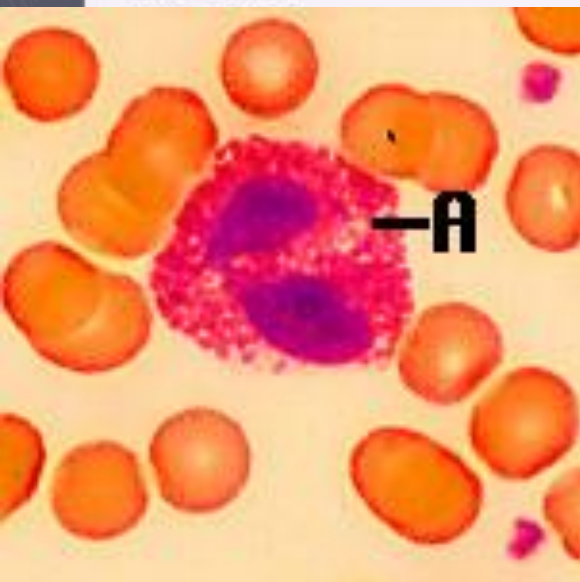
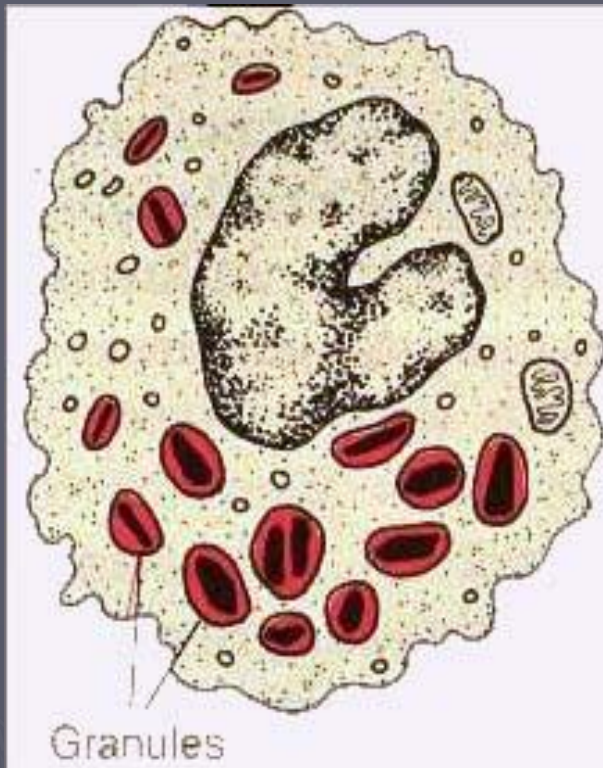
Primary granule
Secondary granule



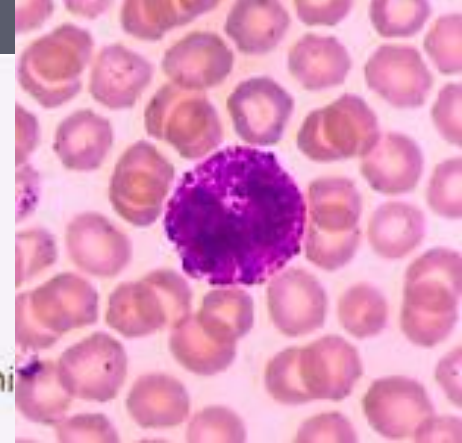
Neutrophil granulocyte

A histological section of skeletal muscle stained with hematoxylin and eosin (H&E). The image shows multiple muscle fibers with varying degrees of eosinophilic staining. Numerous small, dark purple nuclei are visible, many of which are clustered together, indicating a significant inflammatory infiltrate. These cells are identified as neutrophils, which are characteristic of acute inflammation. The overall architecture of the muscle is disrupted by the presence of these leukocytes.

Neutrophil leukocytes in skeletal muscle



Eosinophil
granulocyte



Mast Cell (Basophil)

CHARACTERISTICS AND FUNCTIONS

- Binds IgE molecules
- Contains electron-dense granules

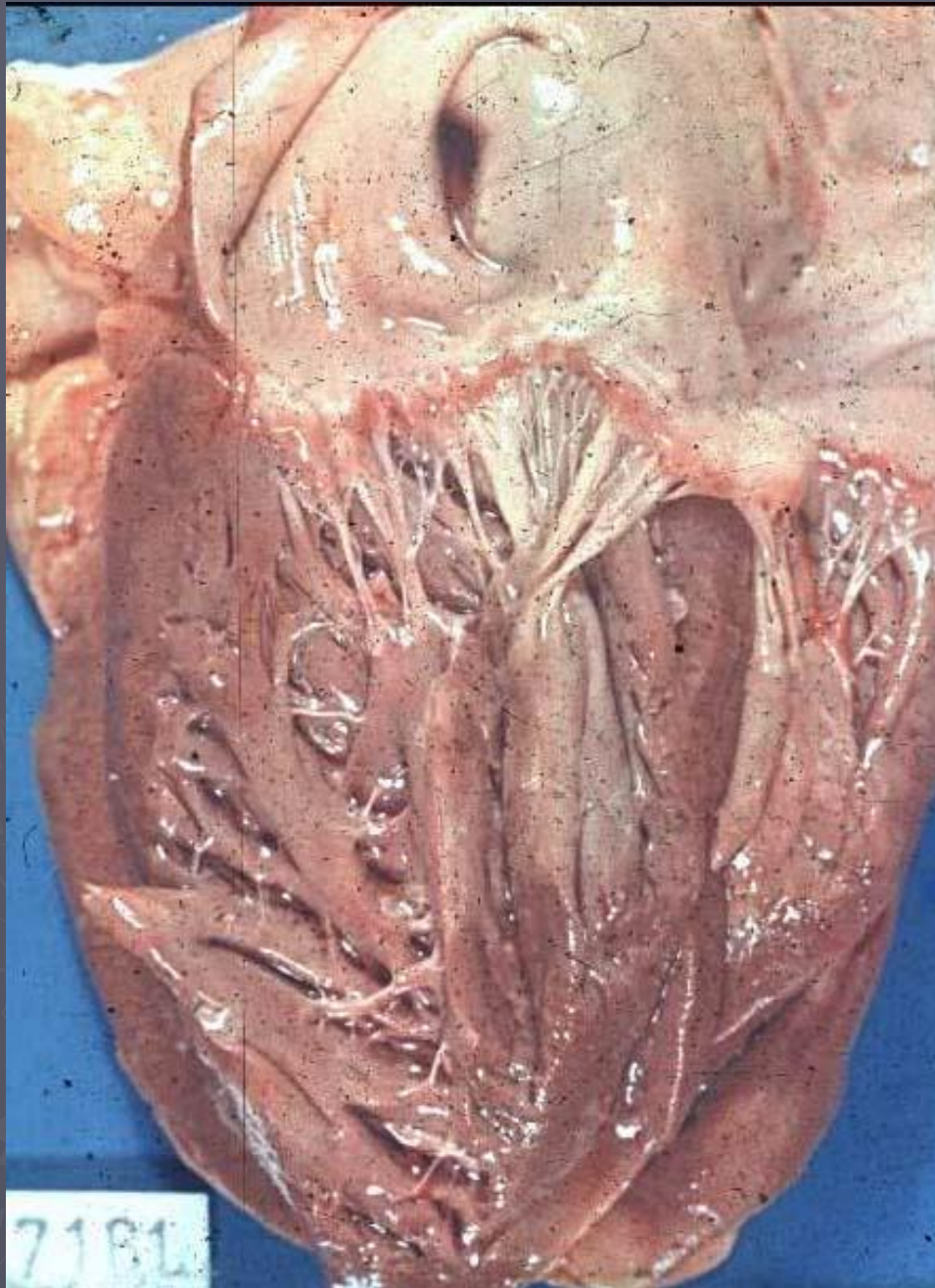
PRIMARY INFLAMMATORY MEDIATORS

- Histamine
- Leukotrienes (LTC_4 , LTD_4 , LTE_4)
- Platelet activating factor
- Eosinophil chemotactic factors

Mast cell (basophil granulocyte)

Types of acute inflammation:

- Serous inflammation – inflammatio serosa
- Fibrinous inflammation – inflammatio fibrinosa
- Purulent inflammation – inflammatio purulenta
- Hemorrhagic inflammation – inflammatio haemorrhagica
- Gangrenous inflammation – inflammatio ichorosa seu gangraenosa



Endocarditis
serosa

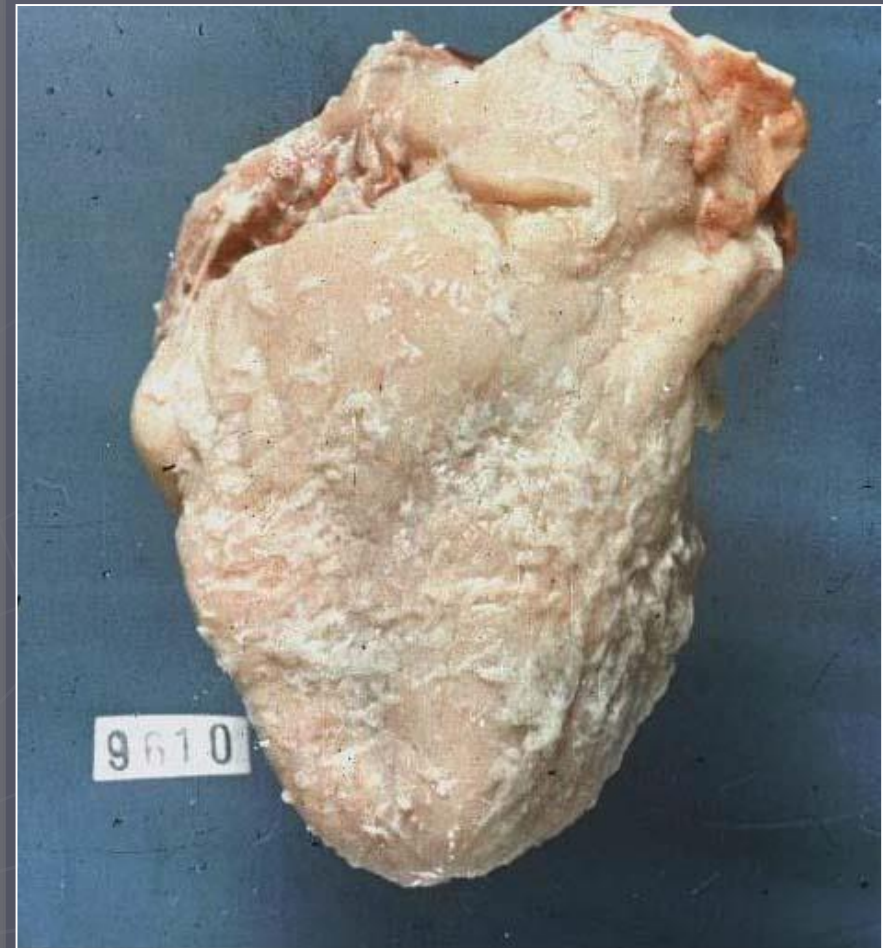


Meningitis
serosa

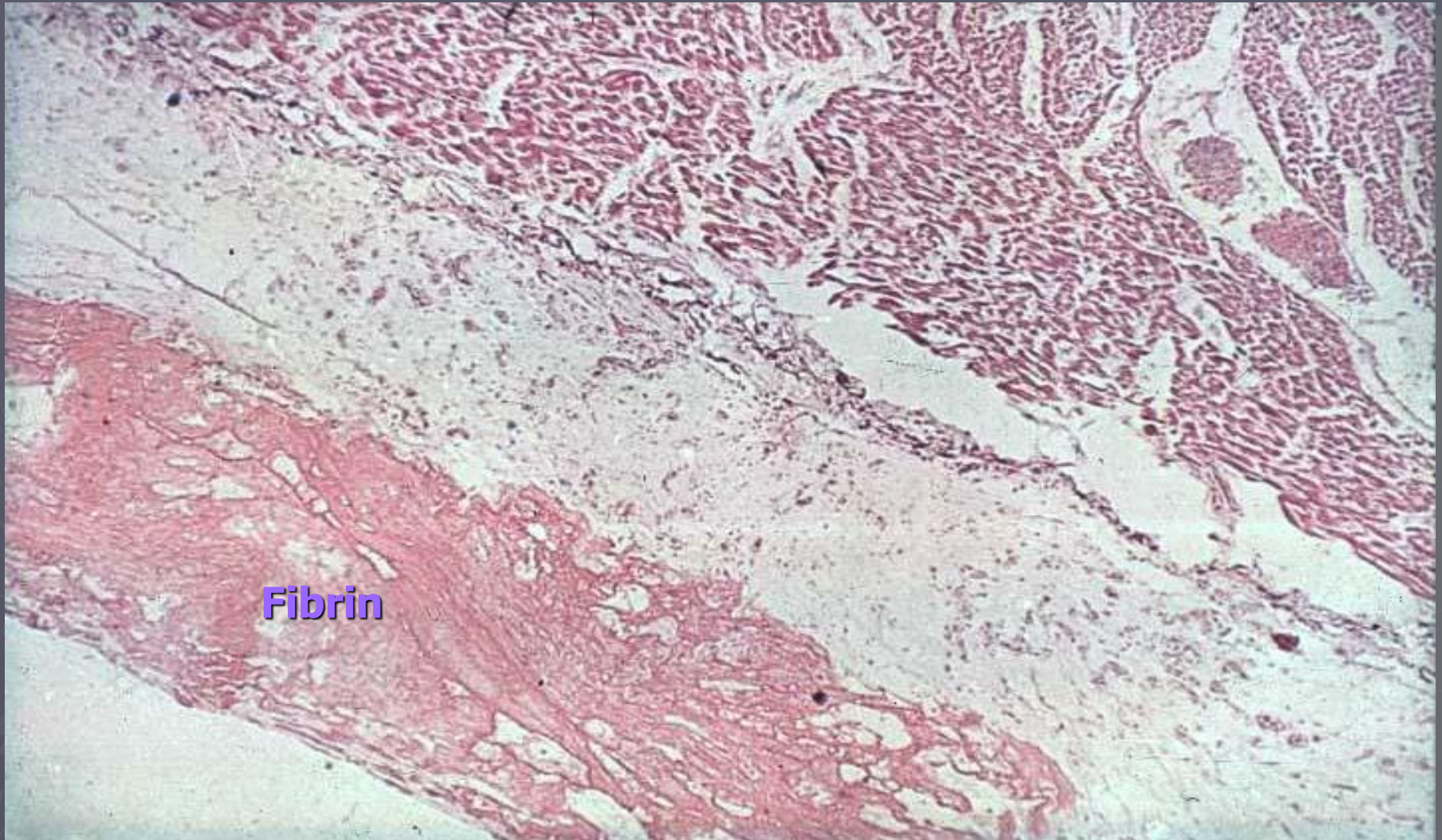
Types of **acute** inflammation:

- Serous inflammation – inflammatio serosa
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Pericarditis fibrinosa



Pericarditis fibrinosa (H&E)

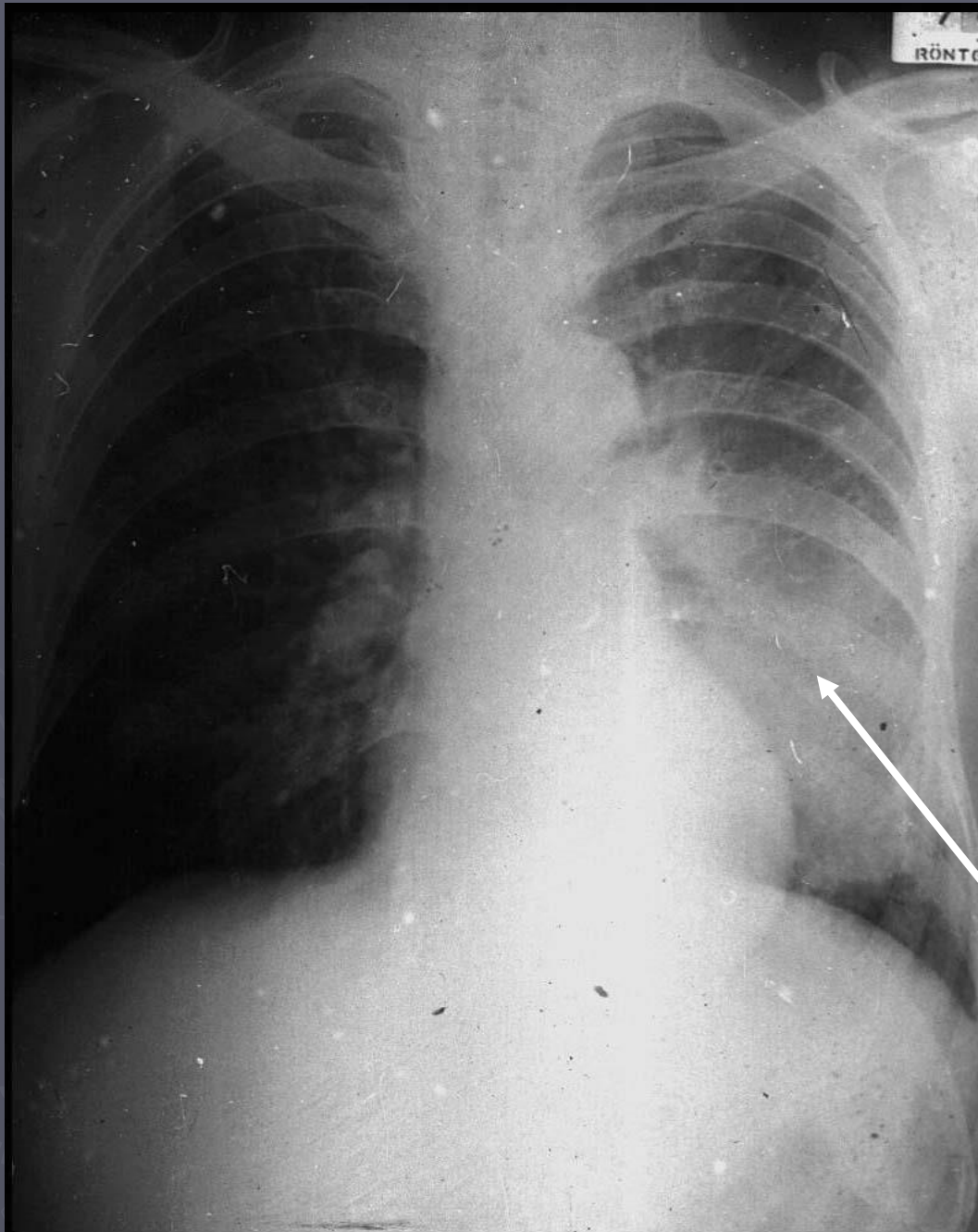




Fibrinous pleuritis



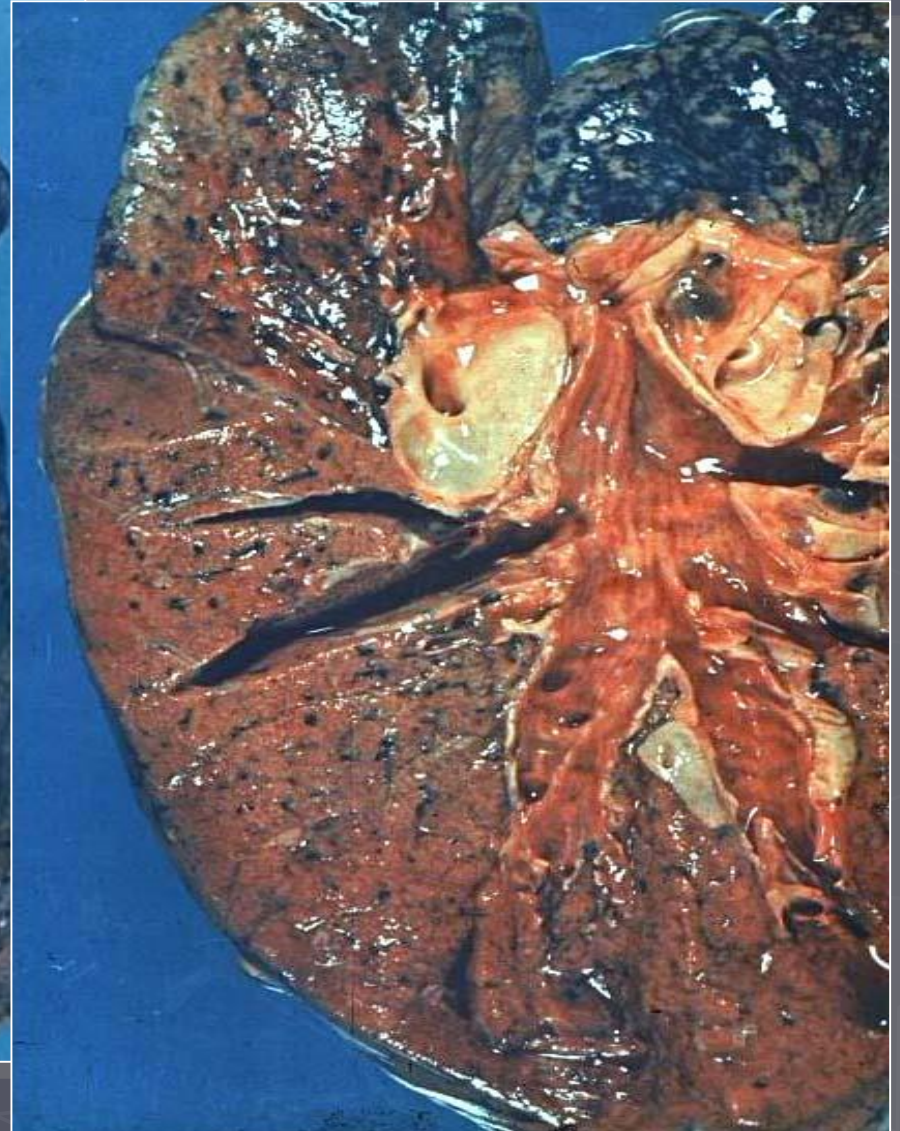
Fibrinous peritonitis

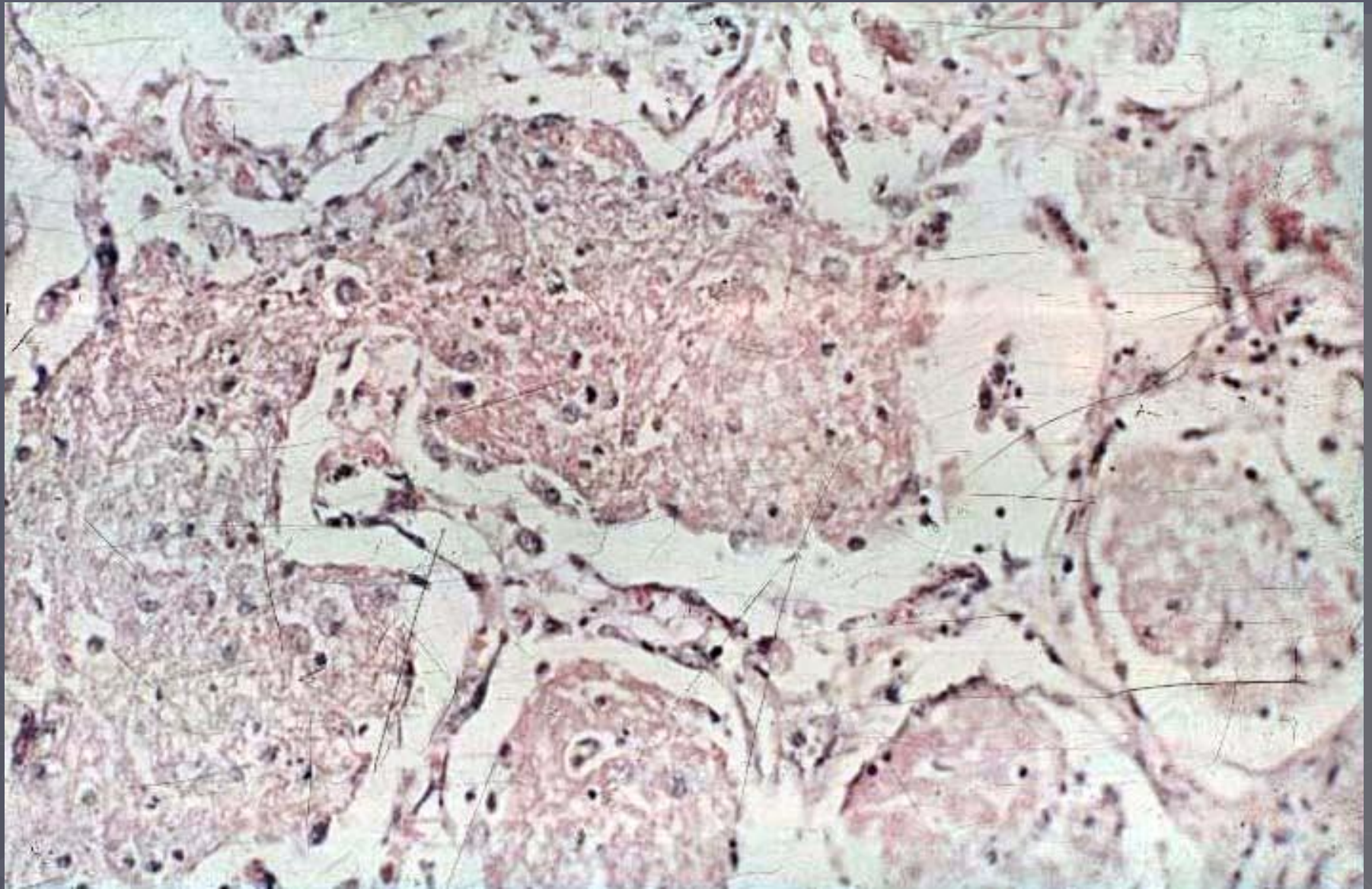


Lobar
pneumonia

Chest X-ray

Lobar pneumonia

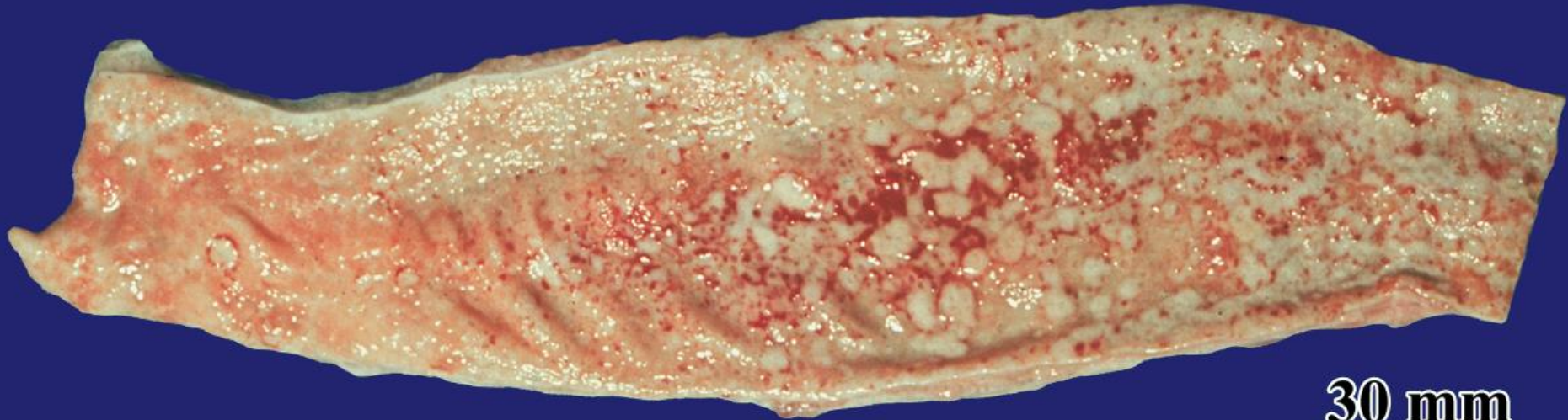




Lobar pneumonia (H&E)

Dysentery

(fibrinopurulent inflammation in large bowel)



30 mm

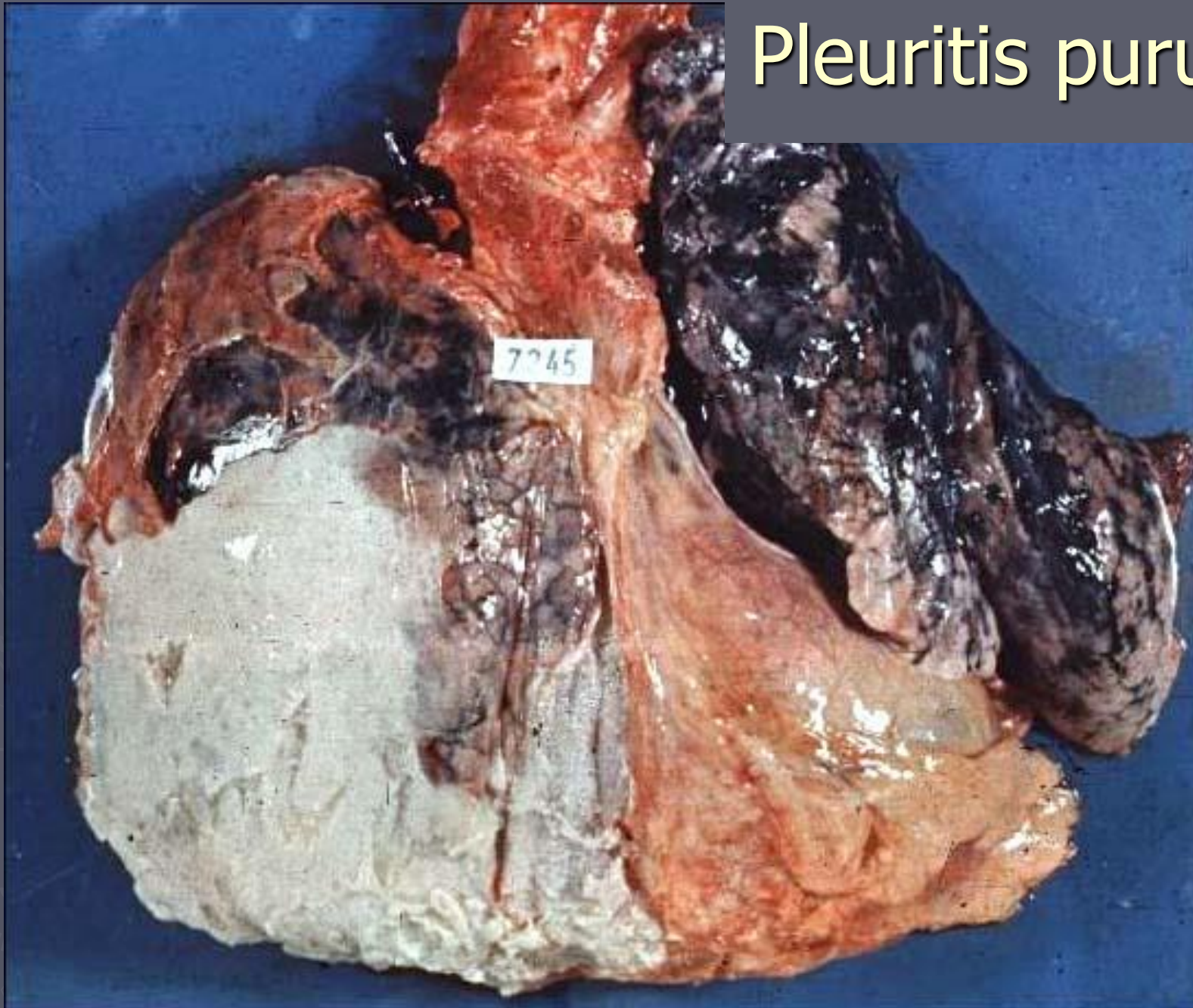
Pseudomembranous inflammation of intestine



Types of **acute** inflammation:

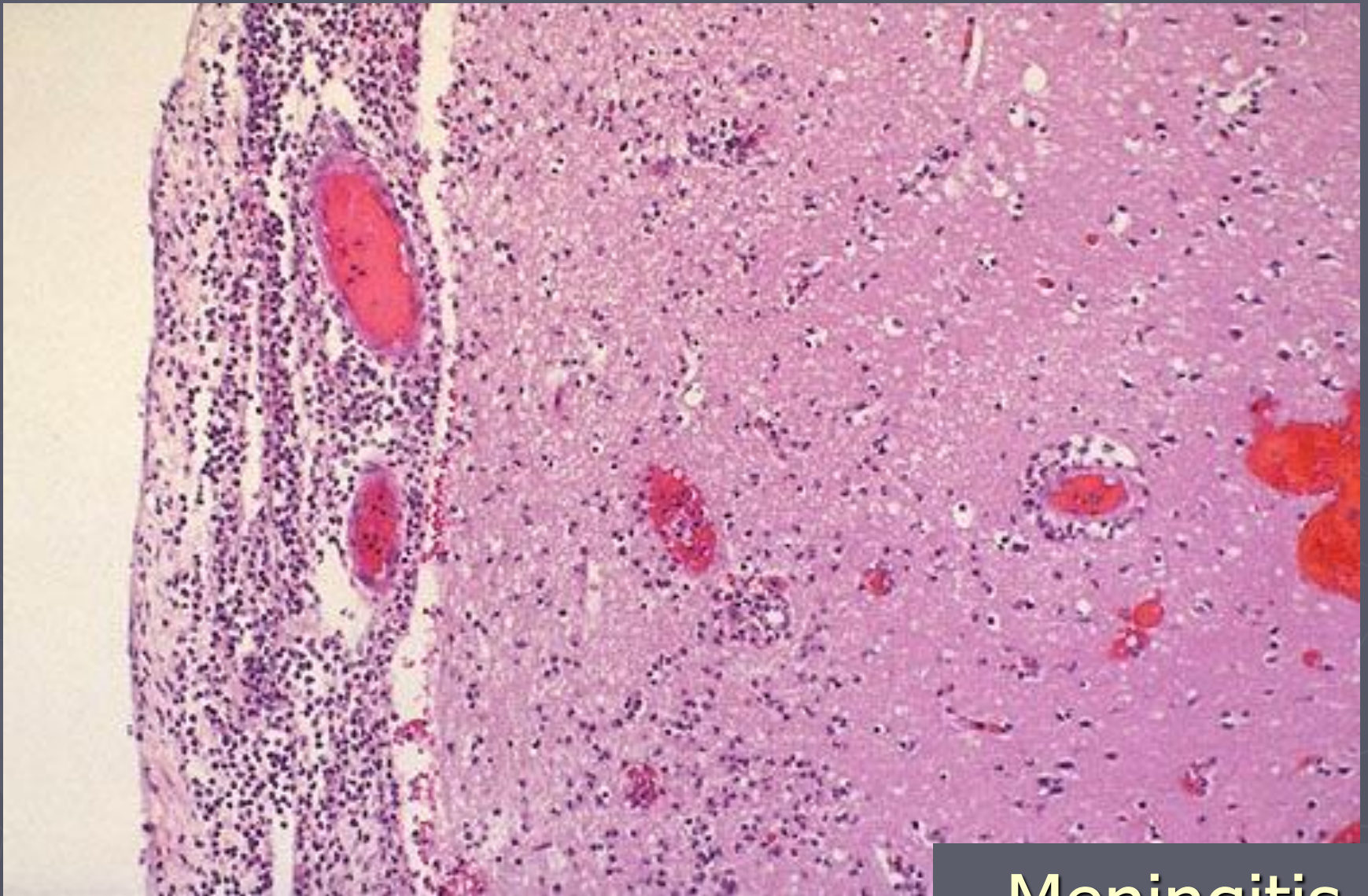
- Serous inflammation – inflammatio serosa
- Fibrinous inflammation – inflammatio fibrinosa
- **Purulent inflammation – inflammatio purulenta**
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Pleuritis purulenta

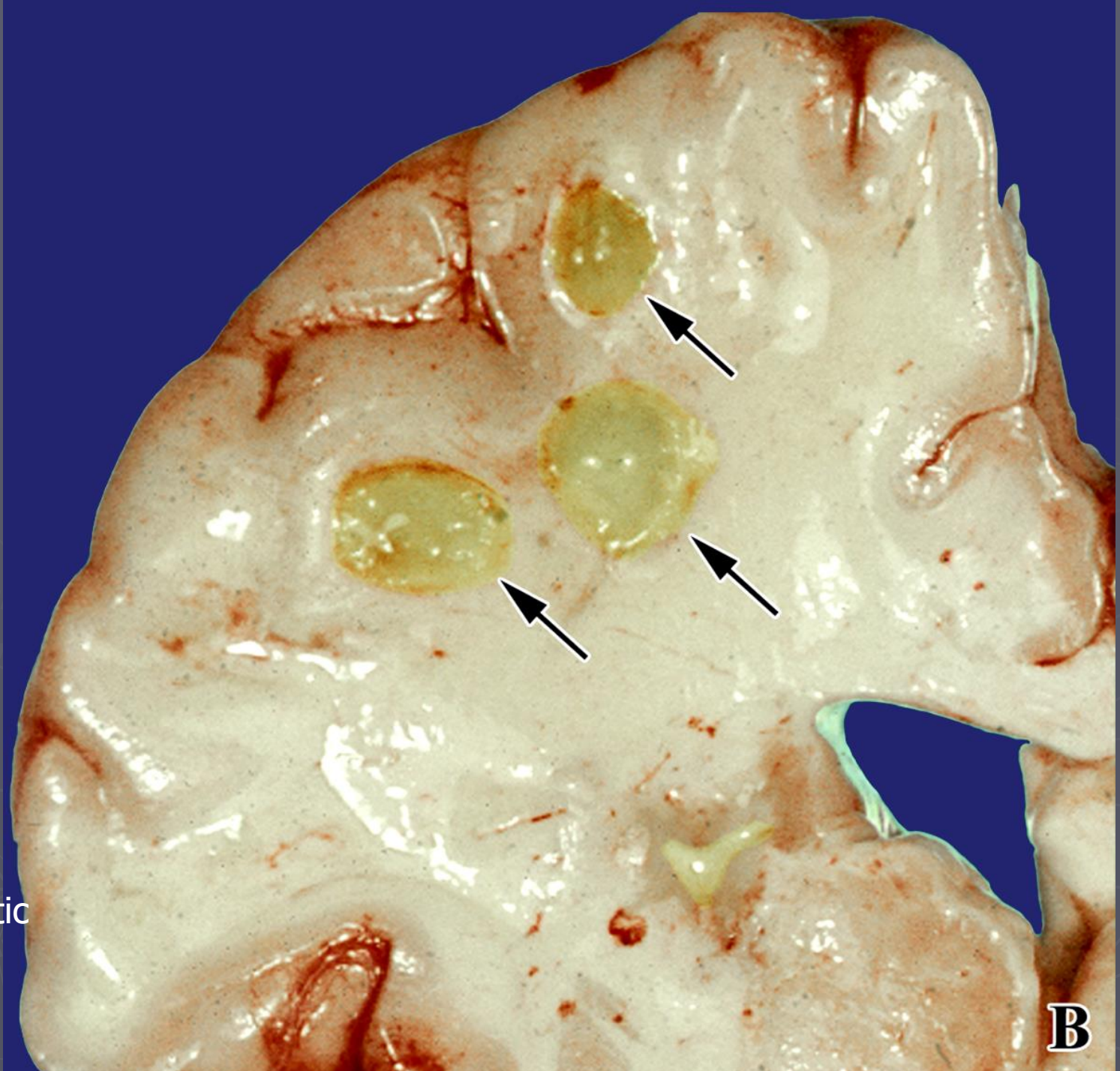




Meningitis
purulenta



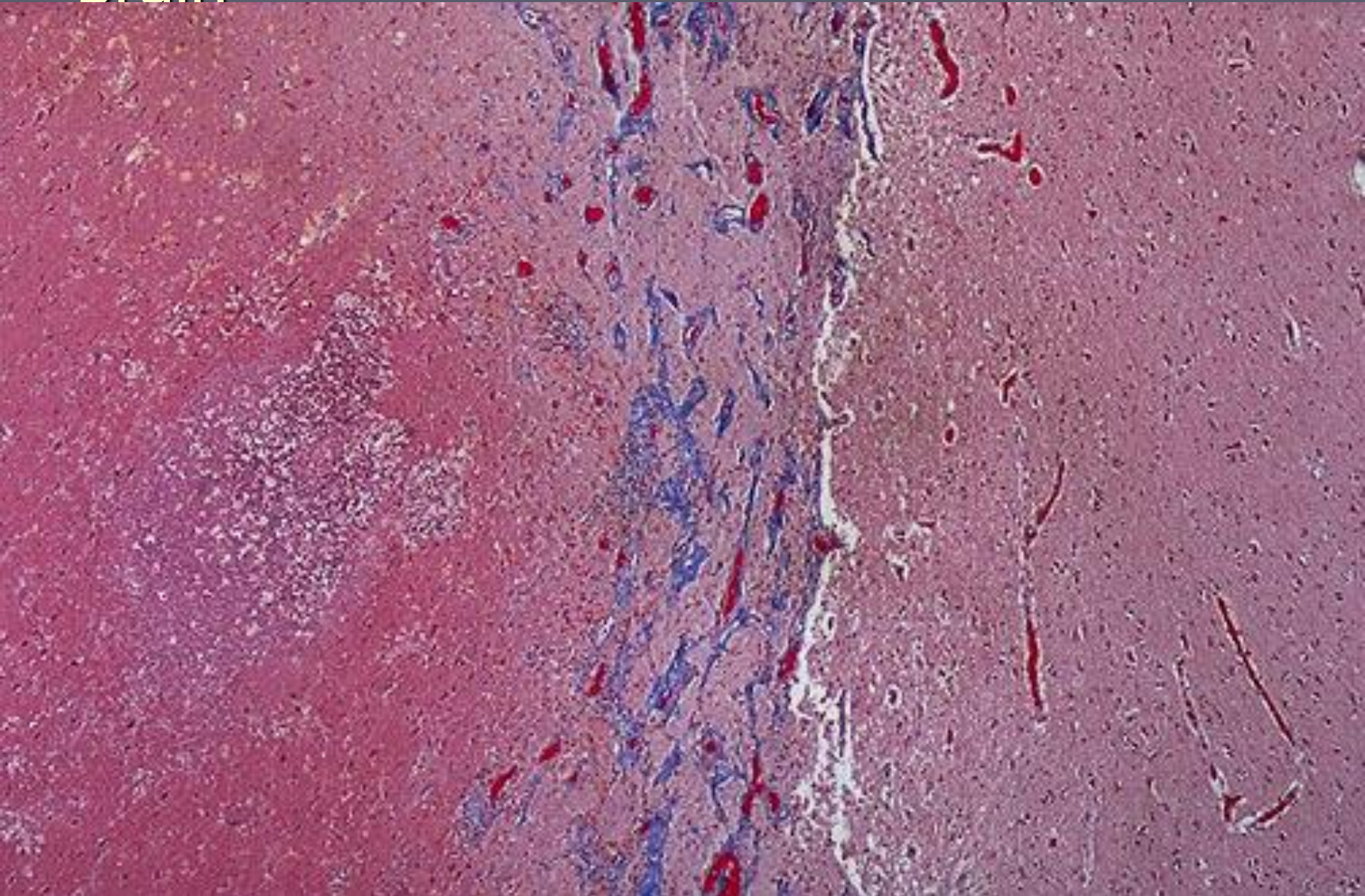
Meningitis
purulenta



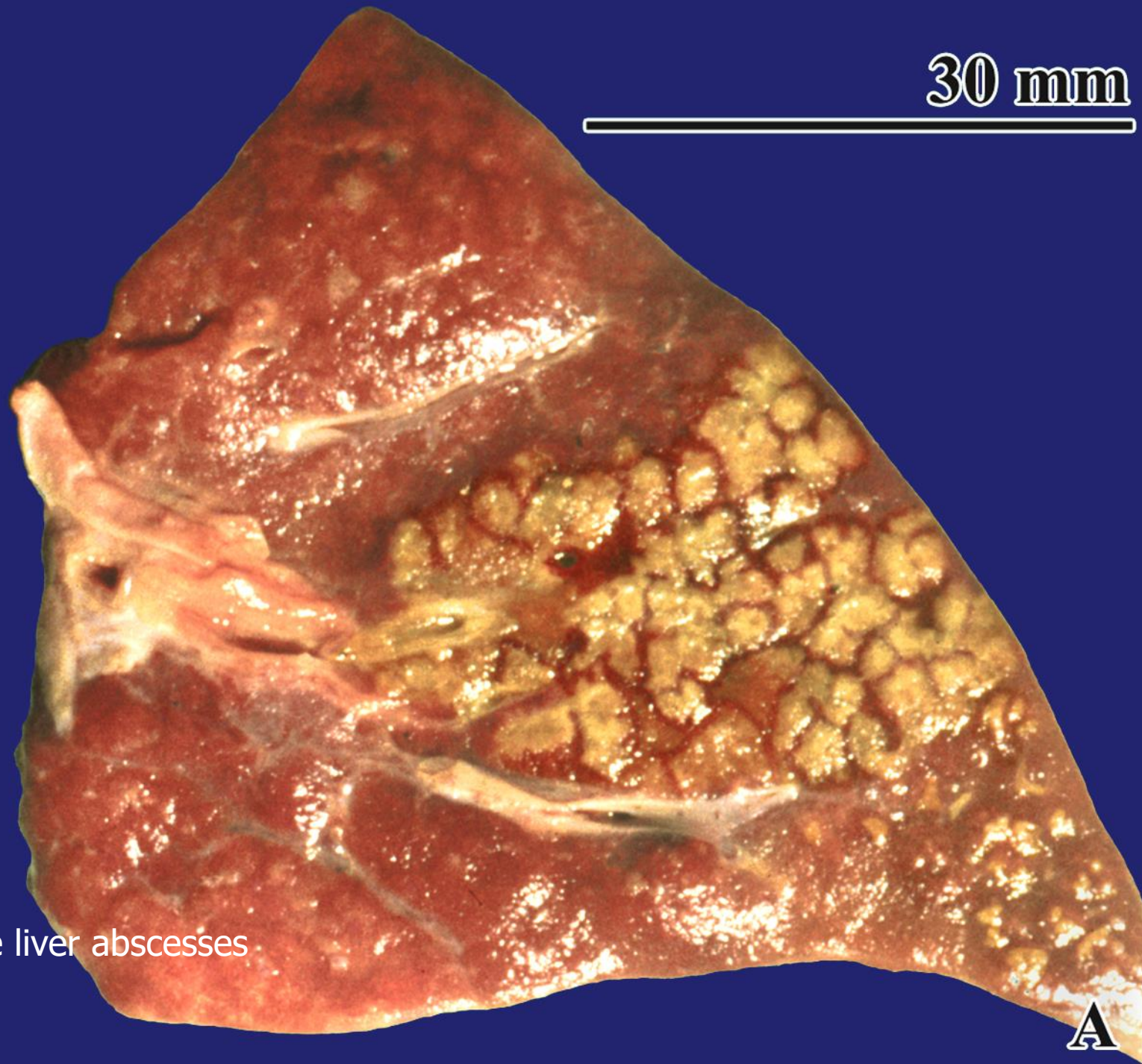
Metastatic
brain
abscess

B

Abscess, brain

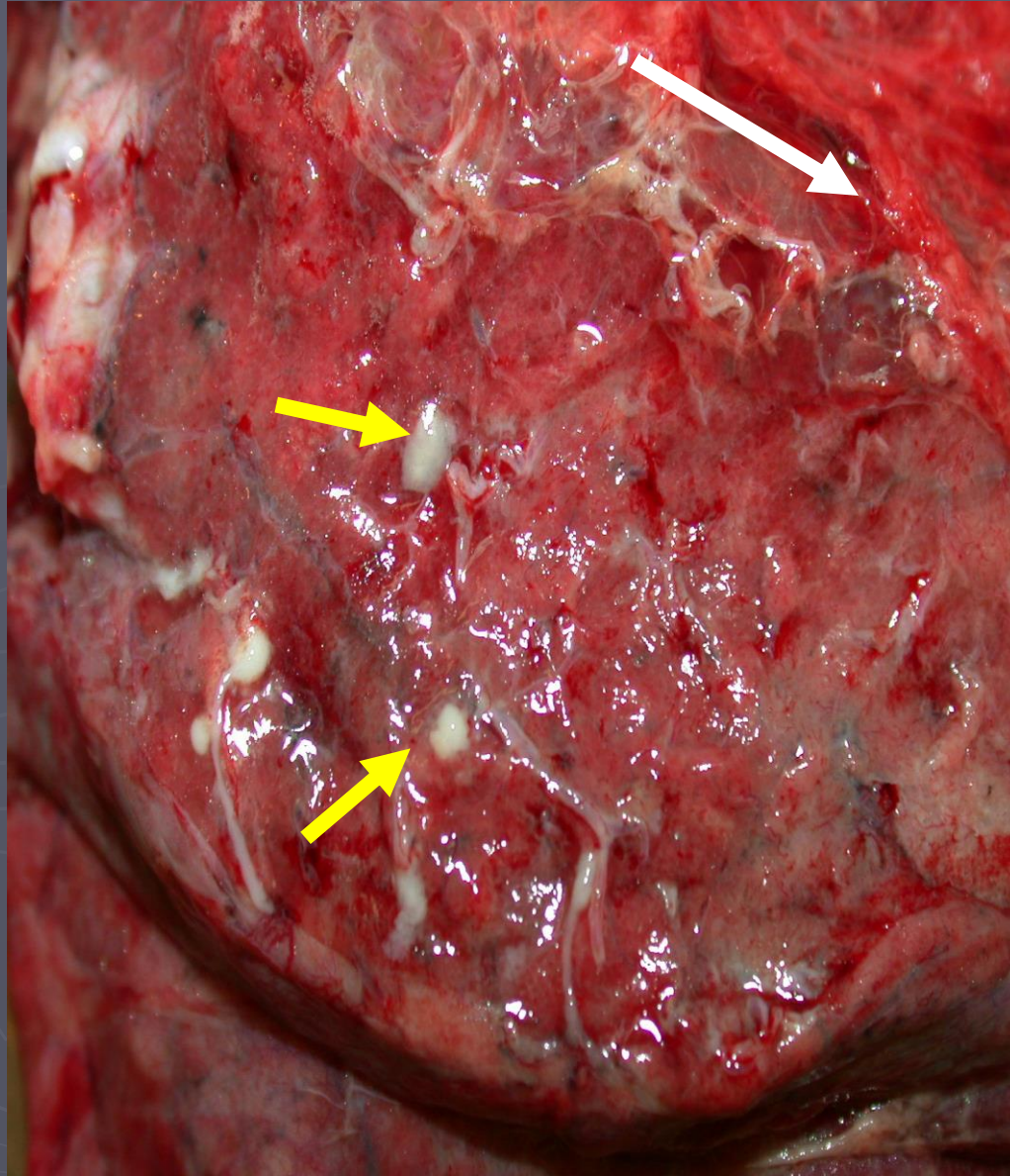


30 mm

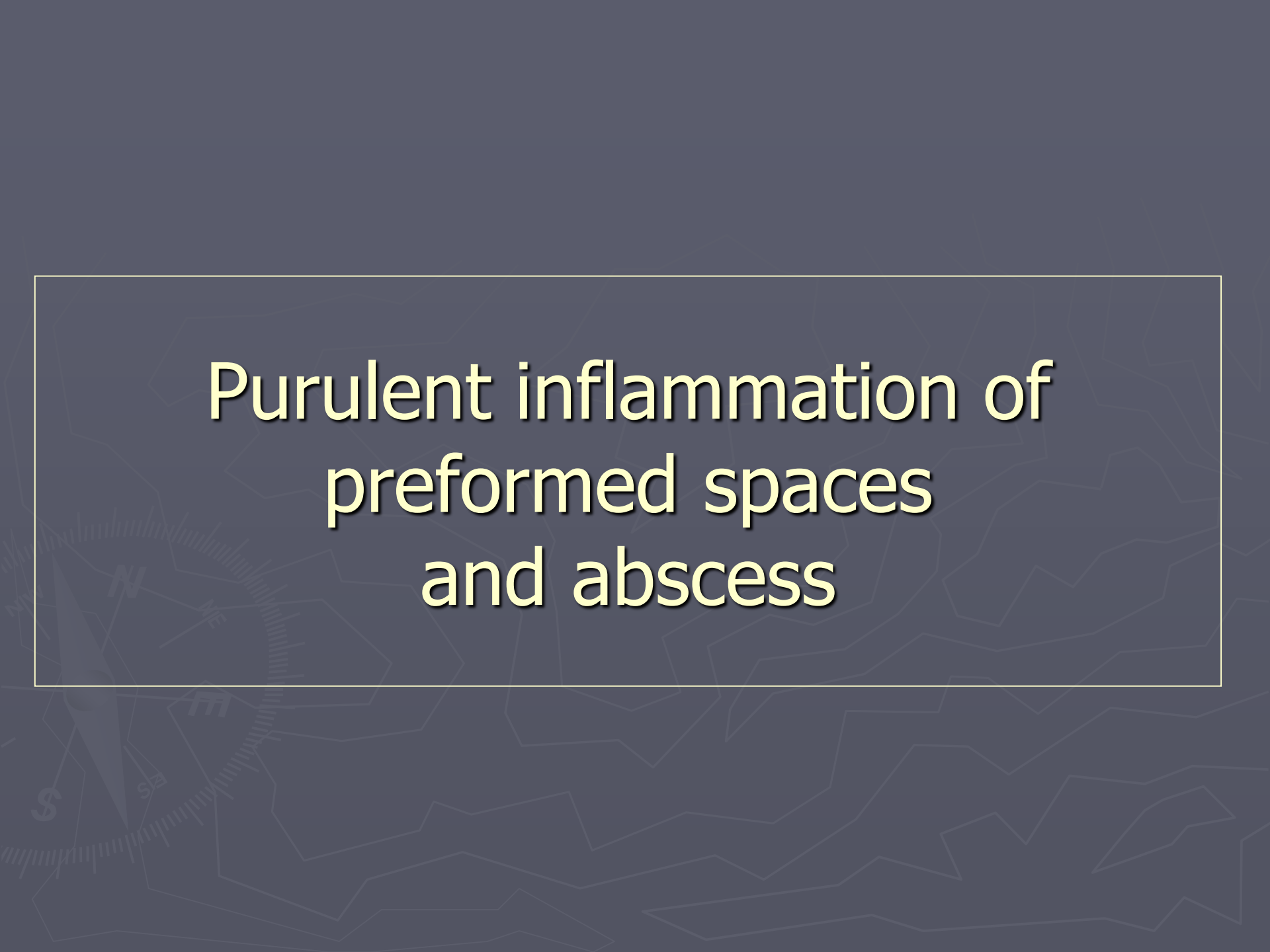


Multiple liver abscesses

Purulent bronchopneumonia



44761.04.24.

The background of the slide is a dark blue-grey color. It features a faint, light-grey graphic on the left side that includes a compass rose with the letter 'N' and a topographic map with contour lines. The main text is centered within a white rectangular border.

Purulent inflammation of preformed spaces and abscess

PUS in an abscess



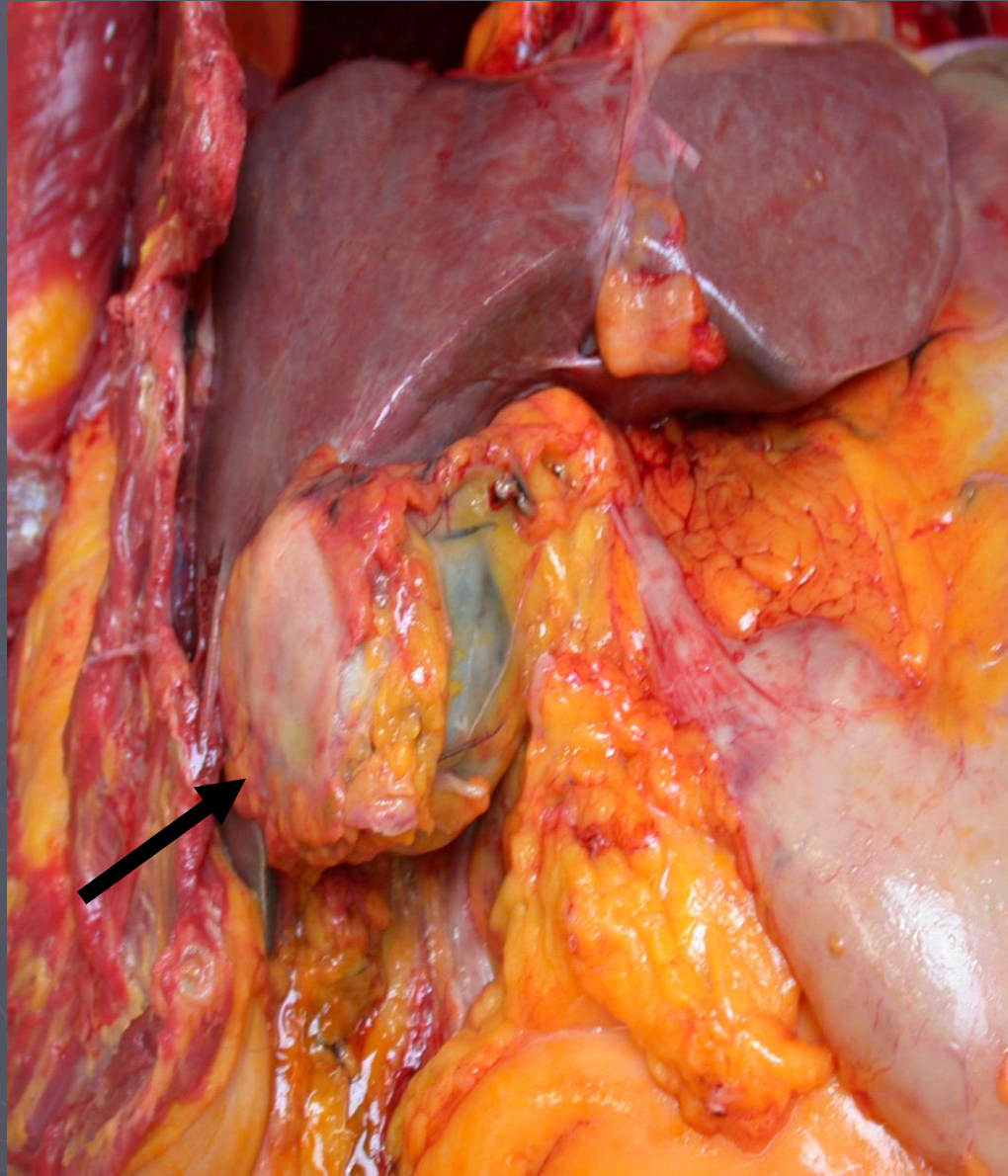
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OPENED ABSCESS



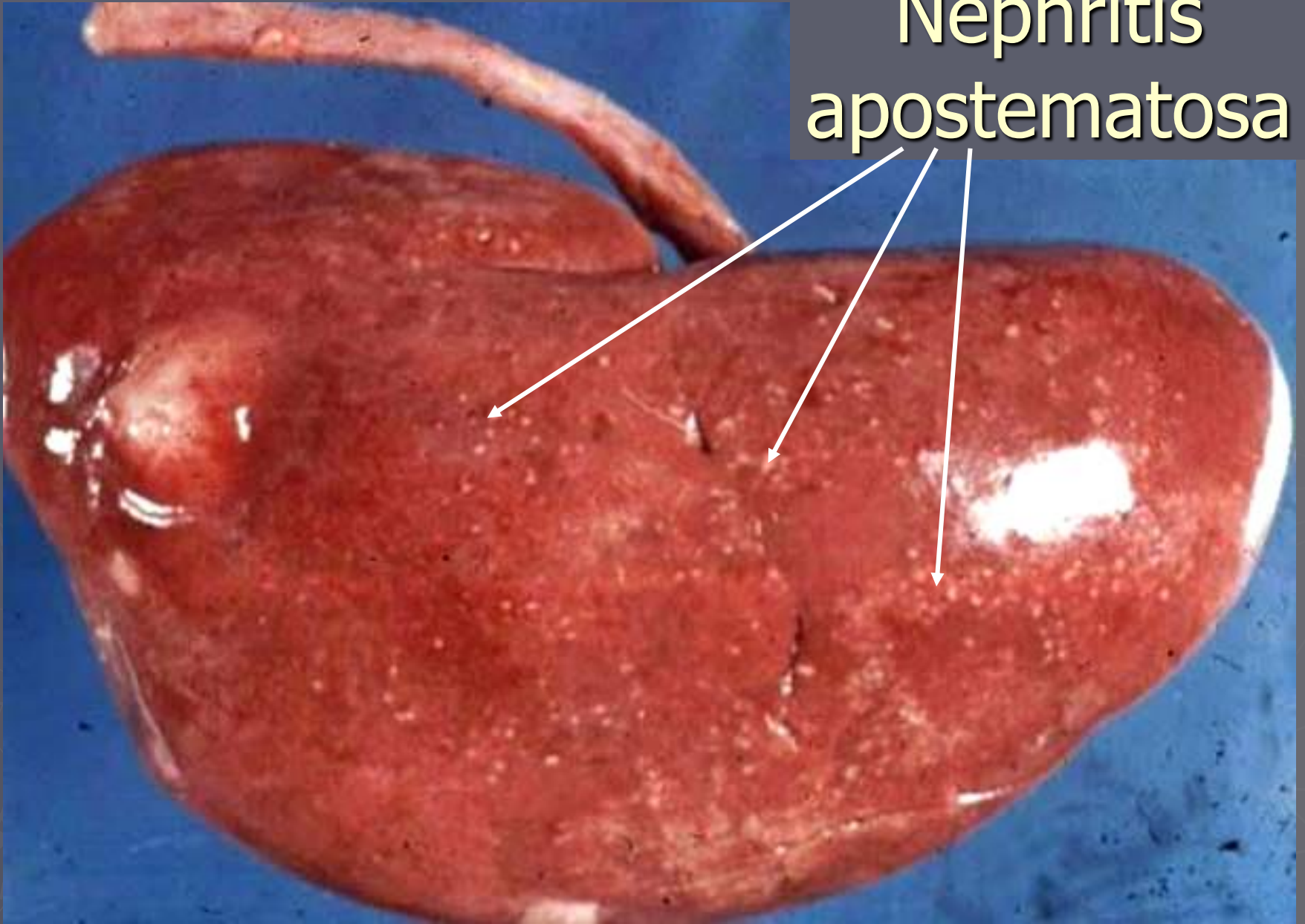
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Empyema vesicae felleae (arrow)

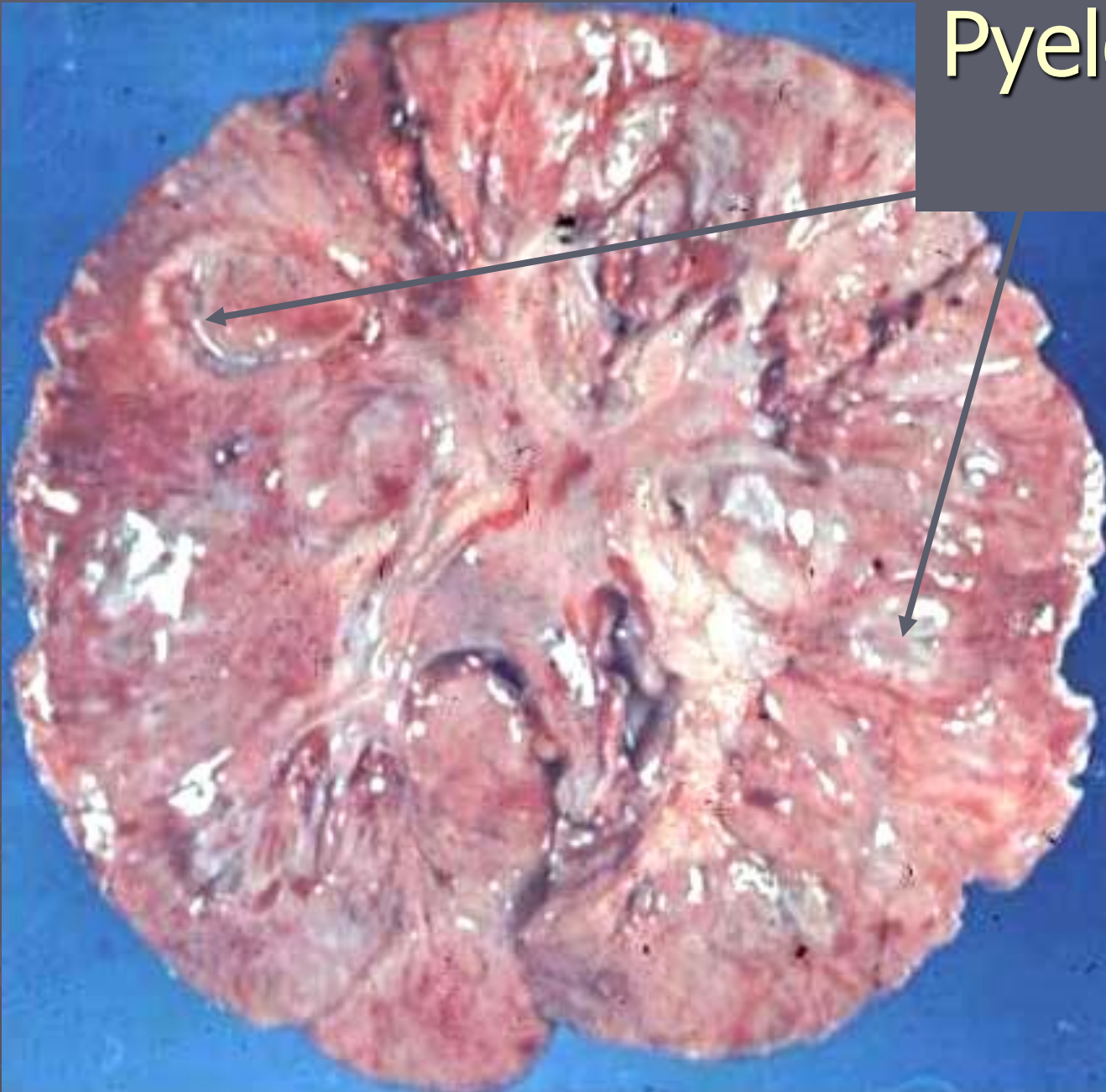


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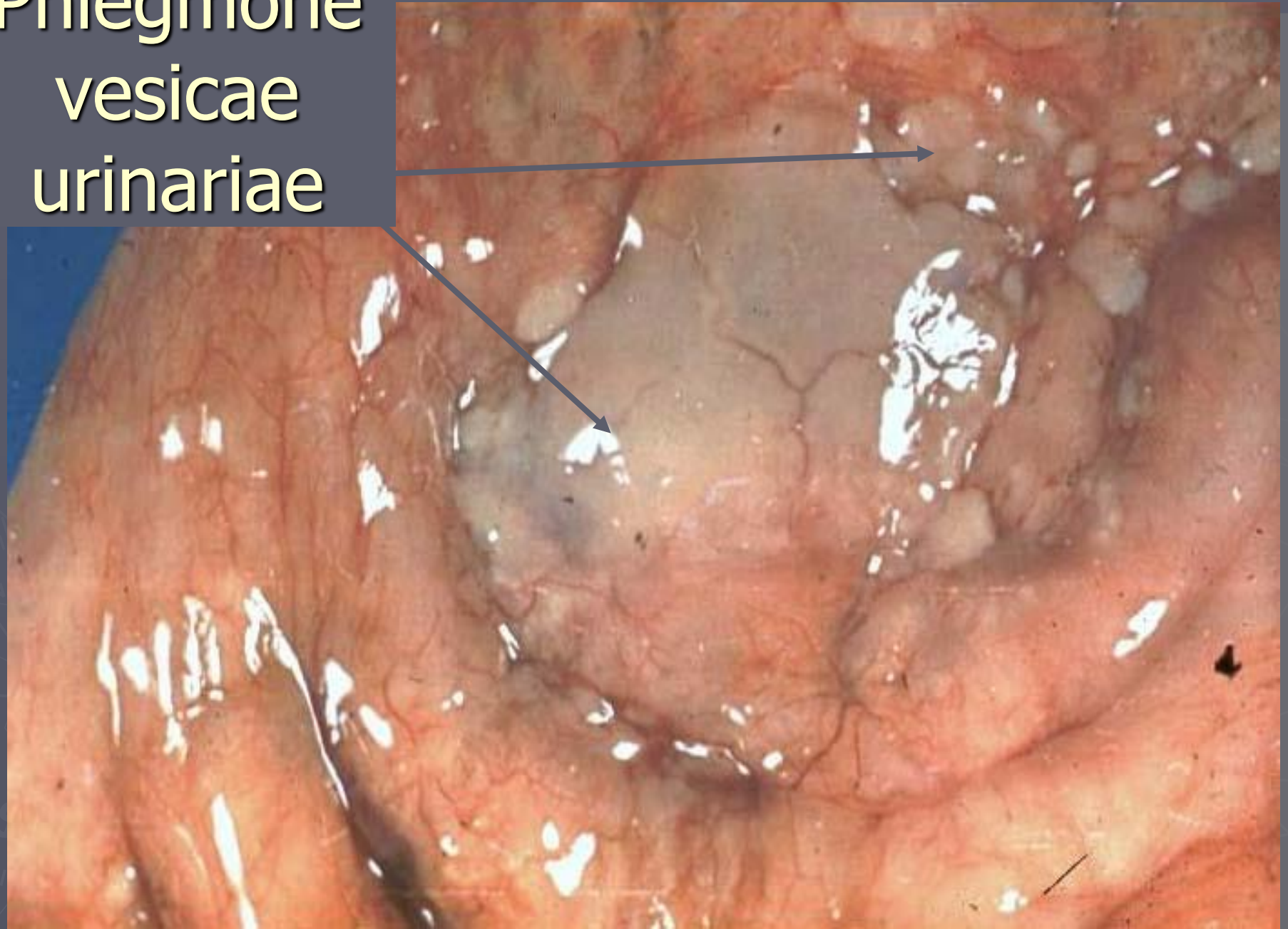
Nephritis apostematosa



Pyelonephritis acuta

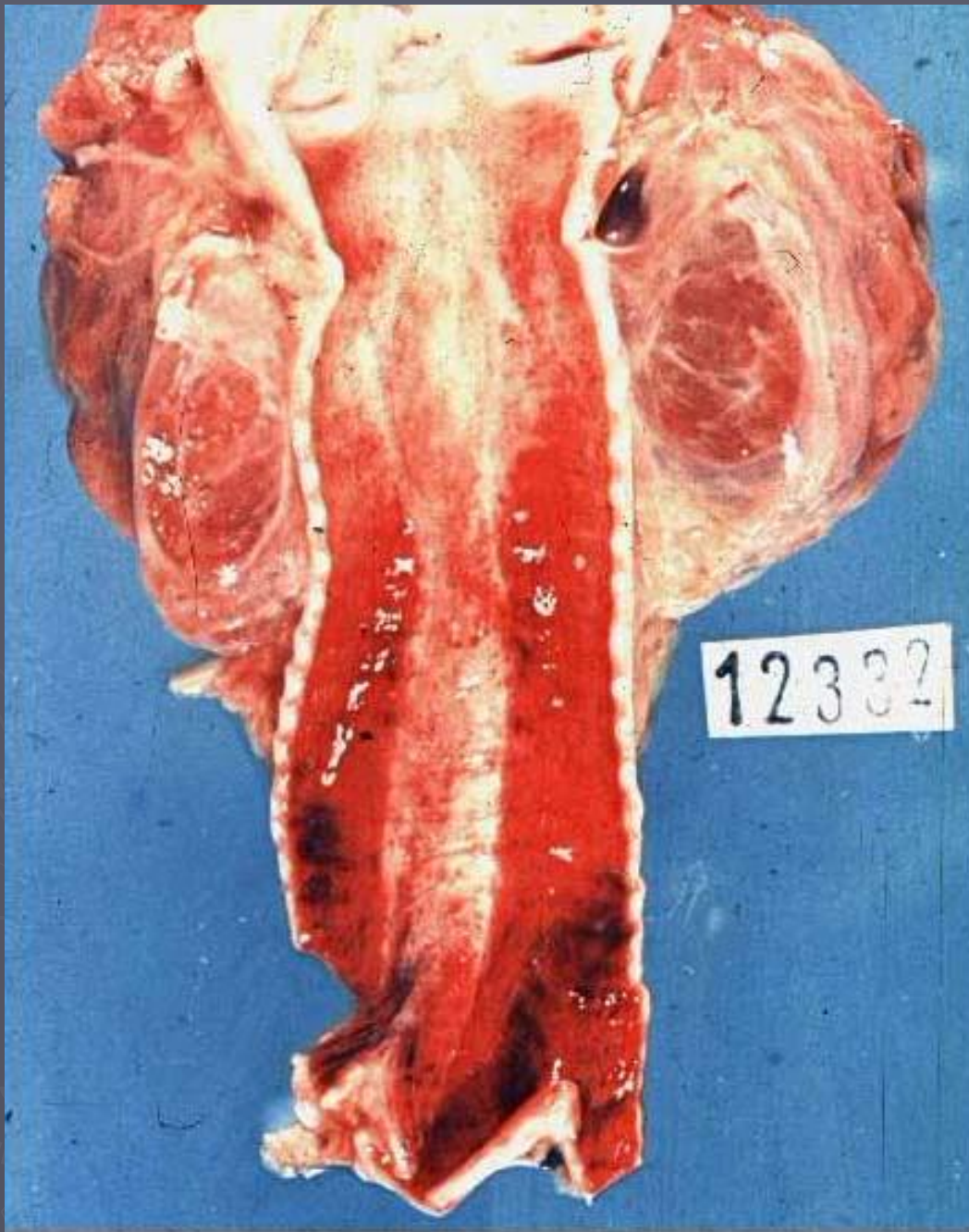


Phlegmone vesicae urinariae

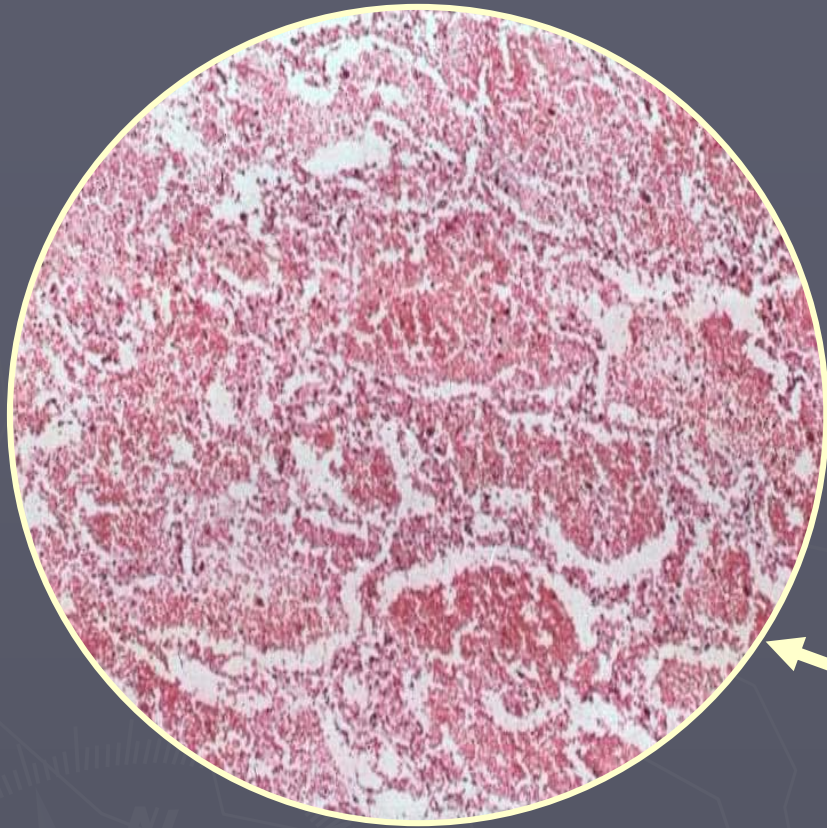


Types of **acute** inflammation:

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Tracheitis
acuta
haemorrhagica



Pneumonia
haemorrhagica

Enteritis haemorrhagica

13545



Anthrax
Haemorrhagic inflammation



Types of **acute** inflammation:

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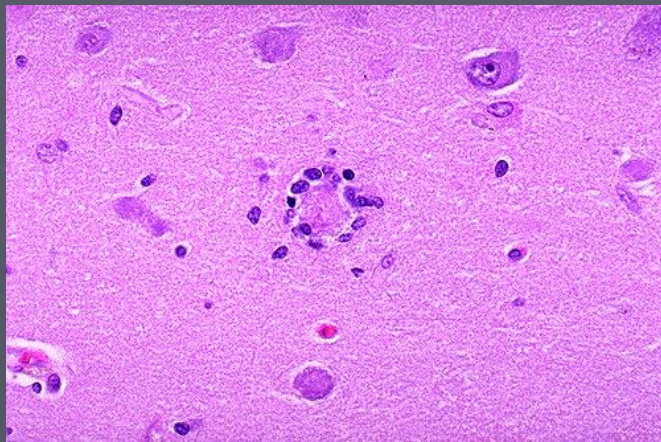
Gangrene
pulmonum

Gangrene

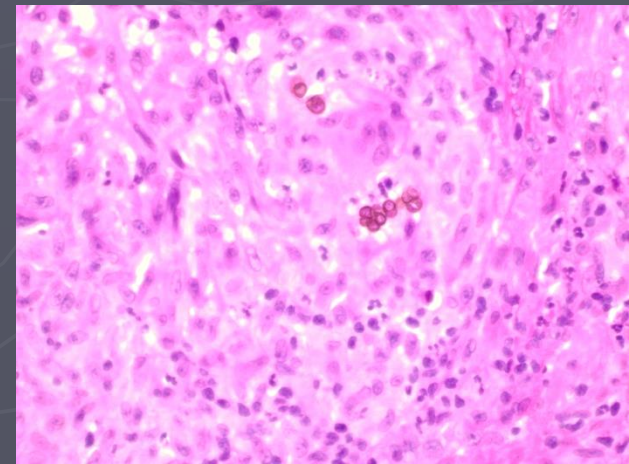


Acute inflammation, exsudate, cause

- ▶ Bacterial: purulent, neutrophyl predominance
- ▶ Viral: serous/hemorrhagic, lymphocytic/MPH



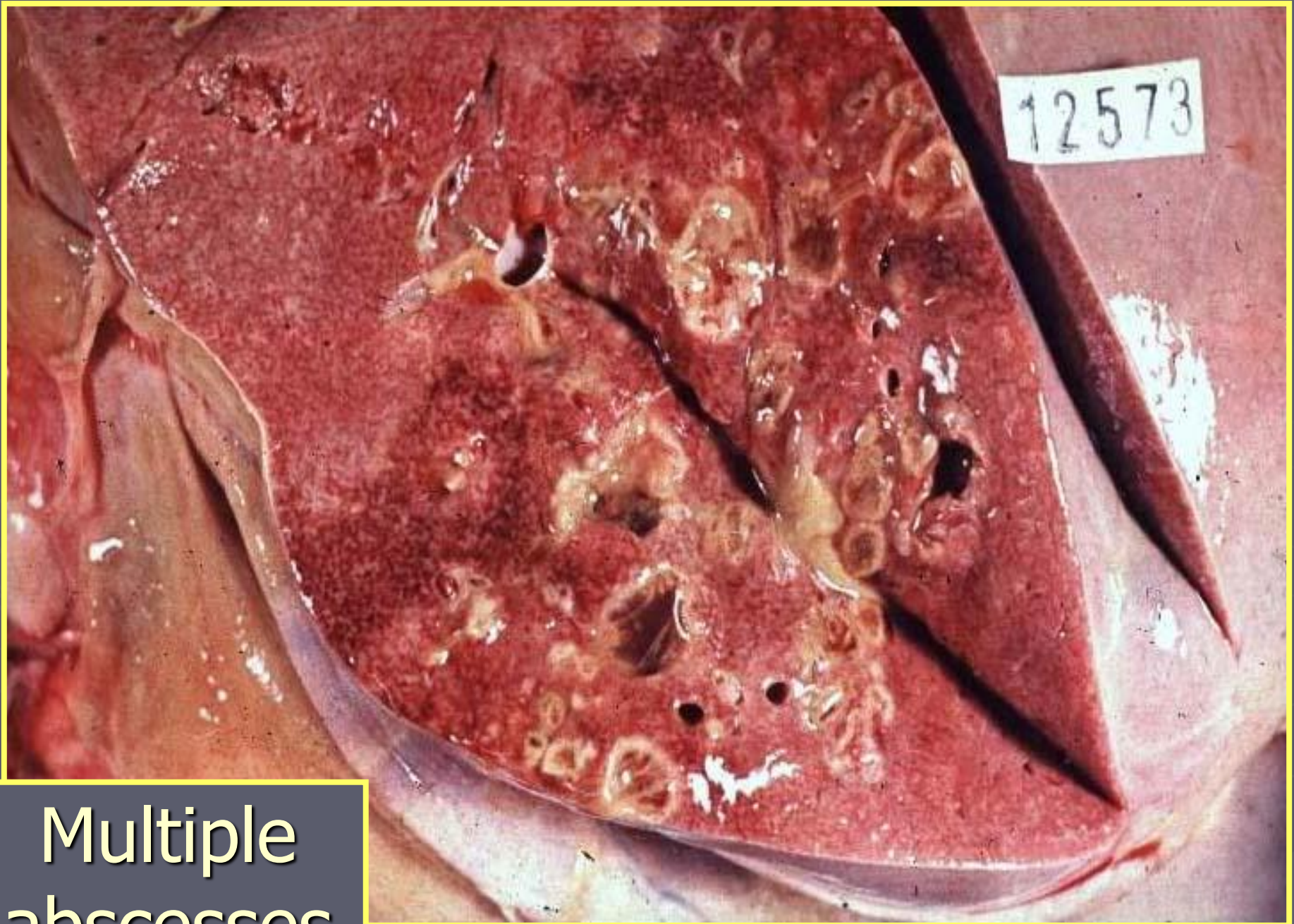
- ▶ Fungi: purulent, neutro+ eosinophyl



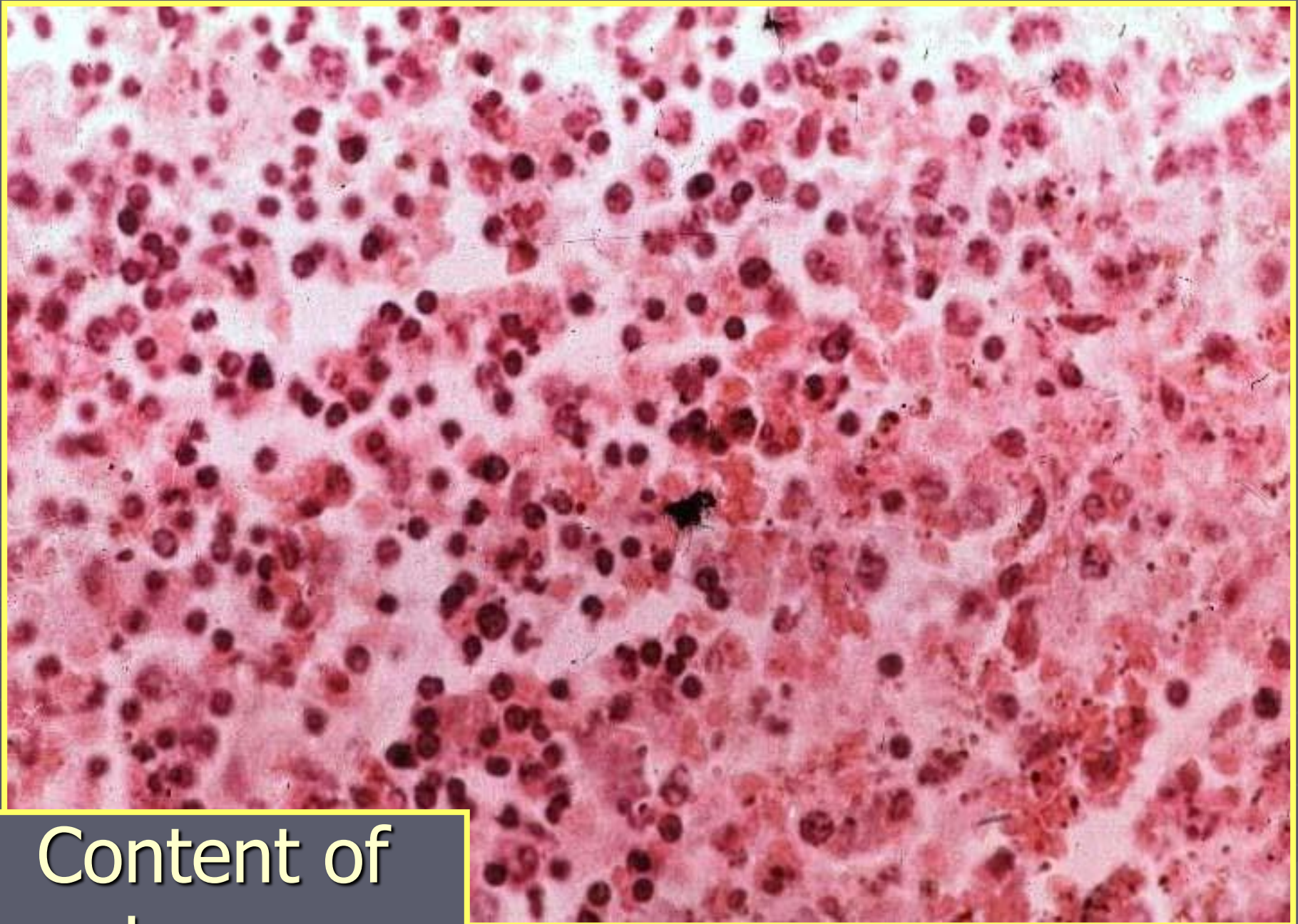


Sepsis

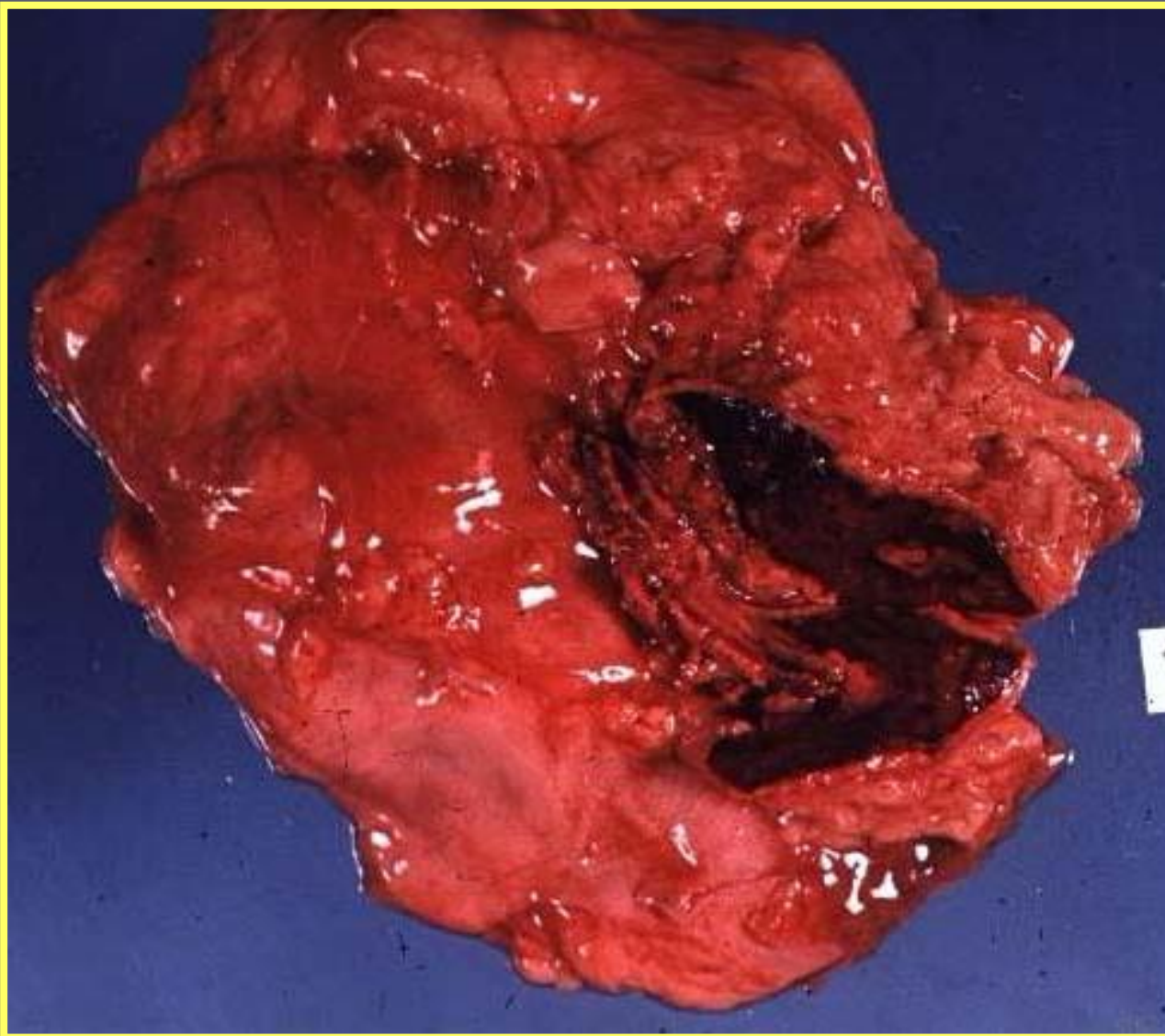
circulating infectious agents
+
clinical symptoms of systemic
acute infections



Multiple
abscesses



Content of
abscess



Adrenalitis haemorrhagica

Neisseria meningitidis
Sepsis

**Waterhouse
Friderichsen sy**

Hemorrhage of adrenal gland
in meningococcus sepsis

Phases of inflammation

- ▶ **Acute**
- ▶ **Subacute**
- ▶ **Chronic**



Subacute inflammation

- ▶ Few weeks or 1-2 months in progress
- ▶ Decreasing exsudate
- ▶ Decreasing edema
- ▶ Eosinophiles, lymphocytes
- ▶ **NOXIOUS AGENT + INFLAMMATION + REPARATION**

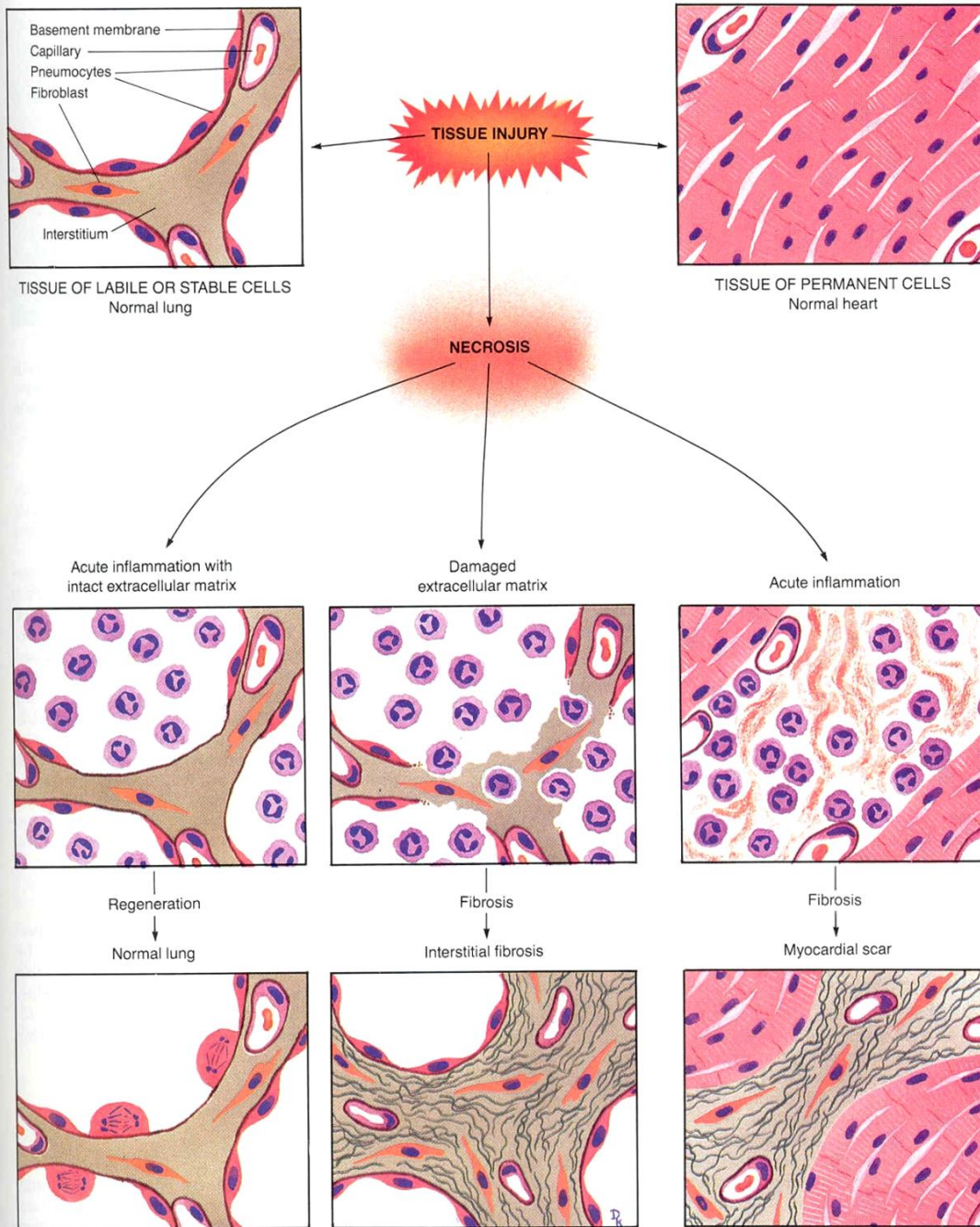
HEALING

REGENERATION:

Is the renewal of a lost tissue or part in which the lost cells are replaced by identical one.

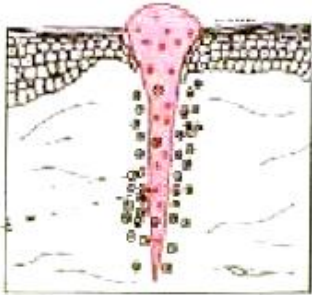
REPAIR

Replacement of injured tissue by scar(connective)tissue

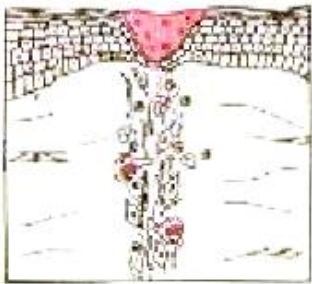


Regeneration of stable és labile cells

Primary healing of wound



- ▶ ***0-24^h: blood clot + neutrophils***

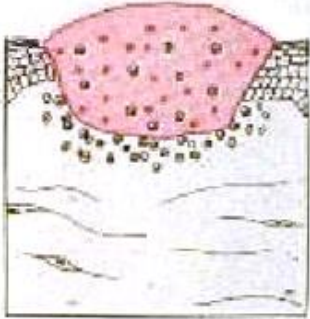


- ▶ ***Few days: granulation tissue, mitosis in epithelium***

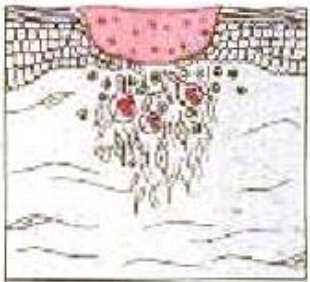


- ▶ ***Few weeks: scar tissue***

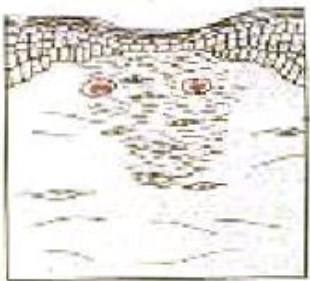
Secondary healing of wound



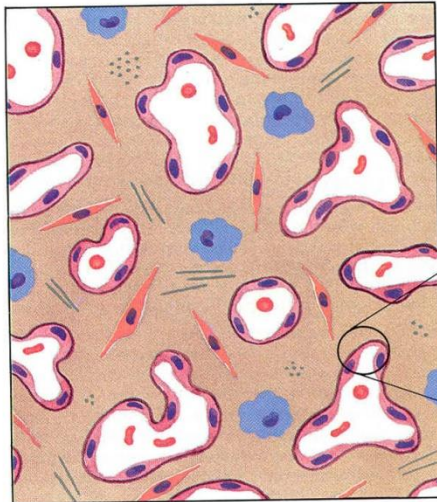
- ▶ ***0-24^h: Necrotic tissue, inflammatory cells, more fibrin***



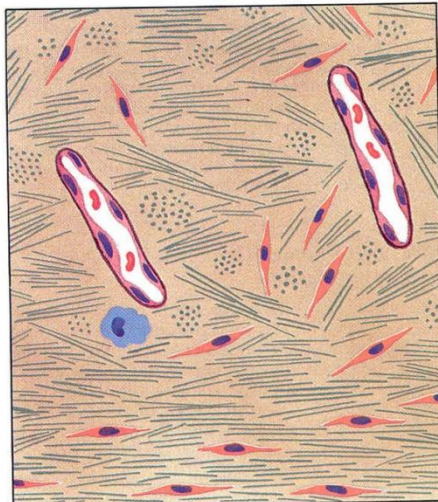
- ▶ ***Few days: granulation tissue***



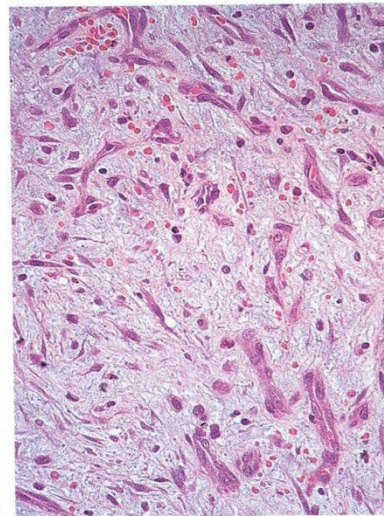
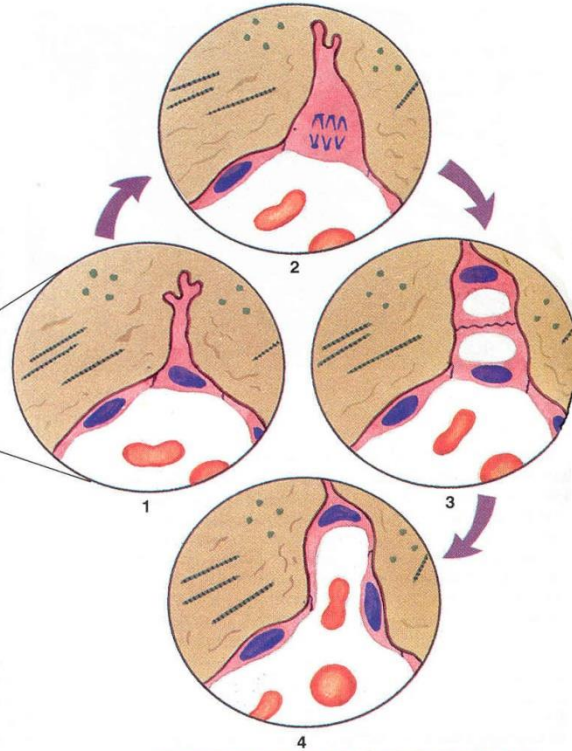
- ▶ ***Few weeks: scar, contraction of the wound***



A

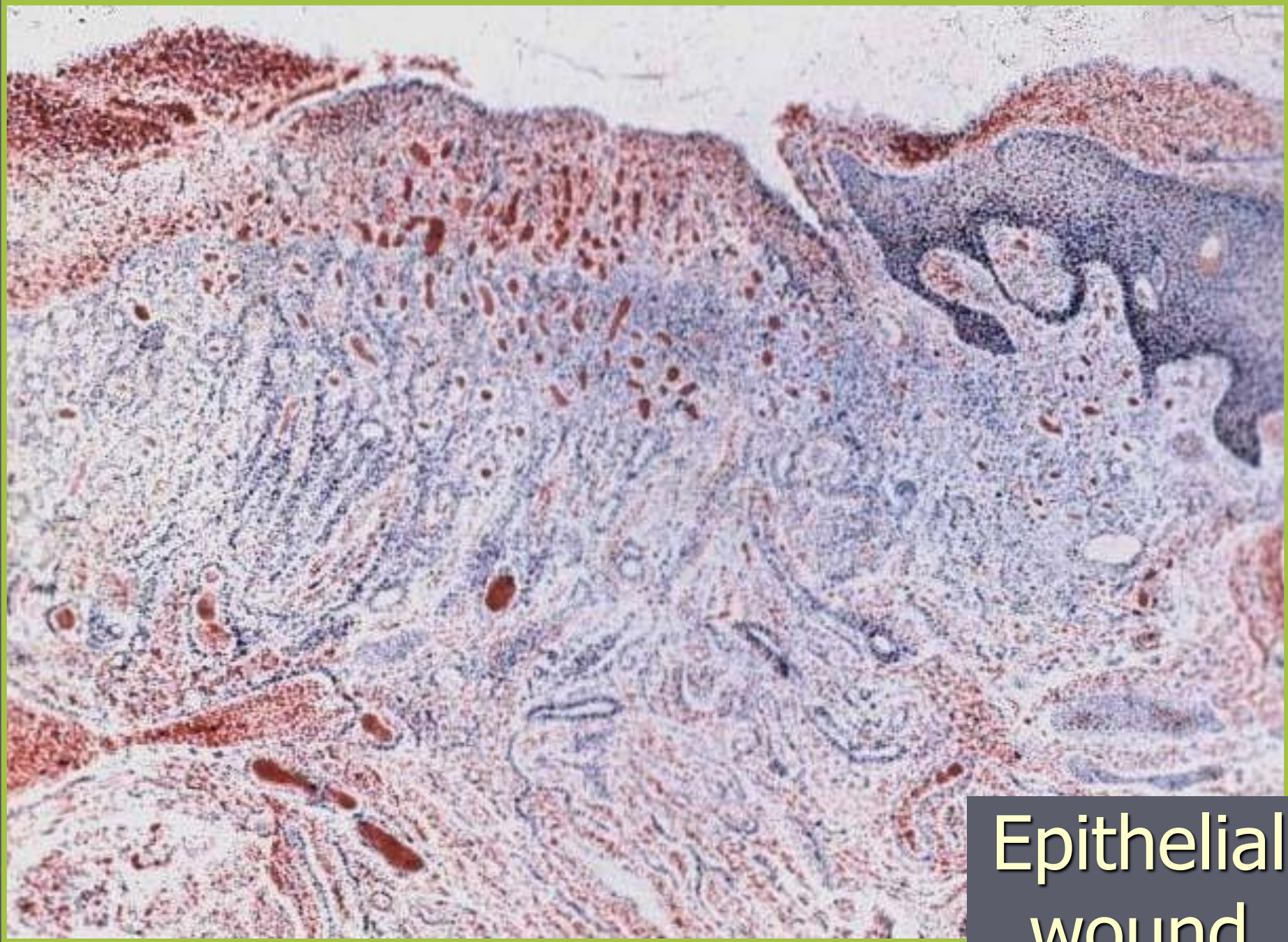


B

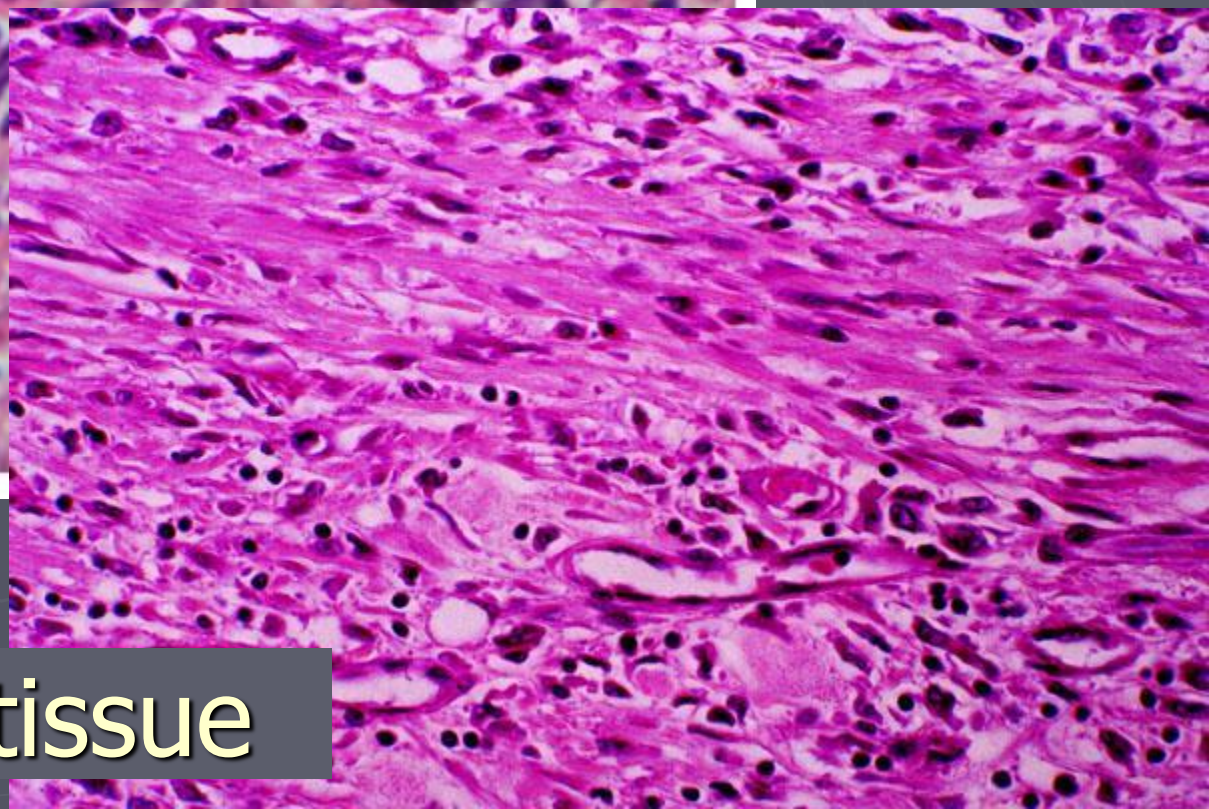
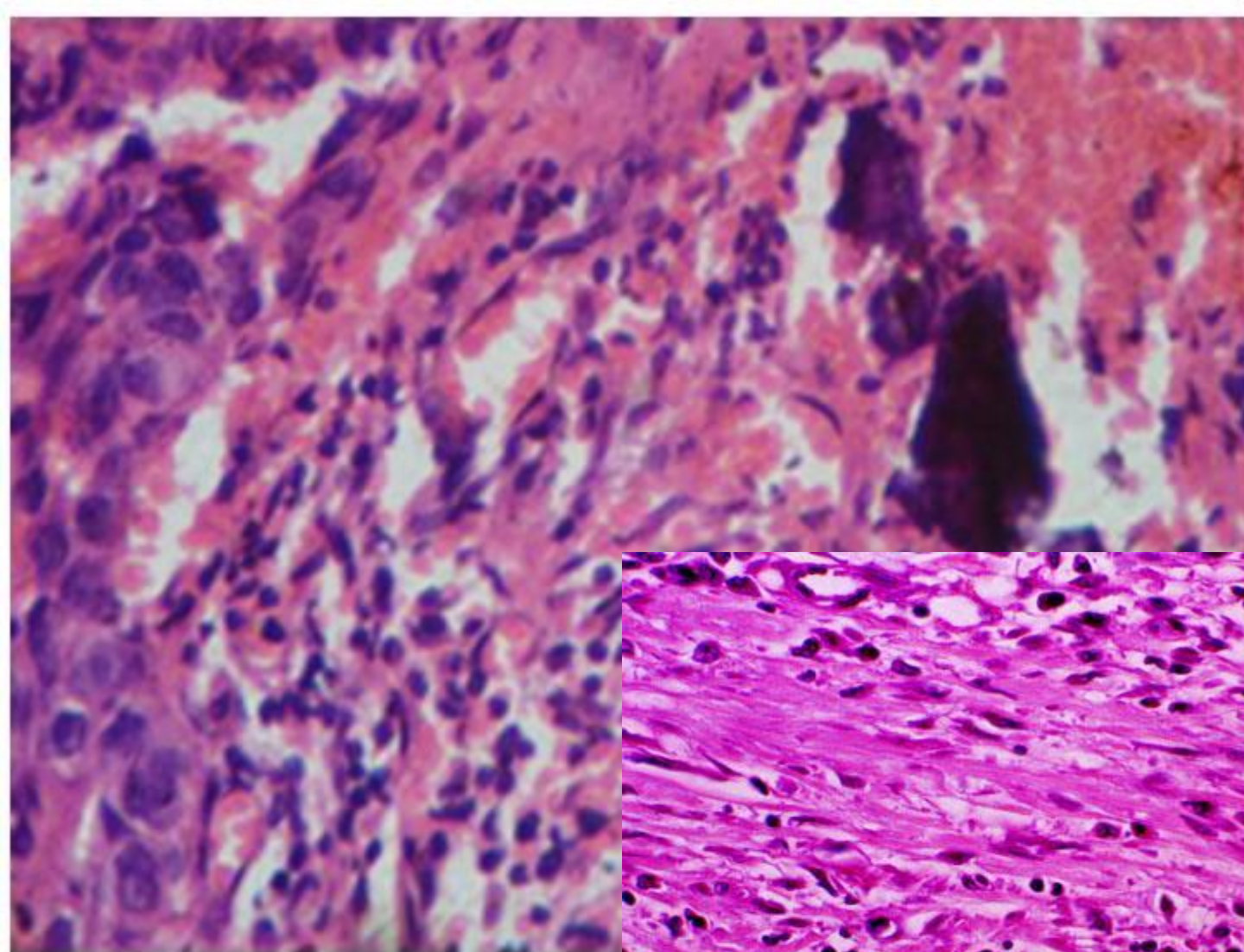


C

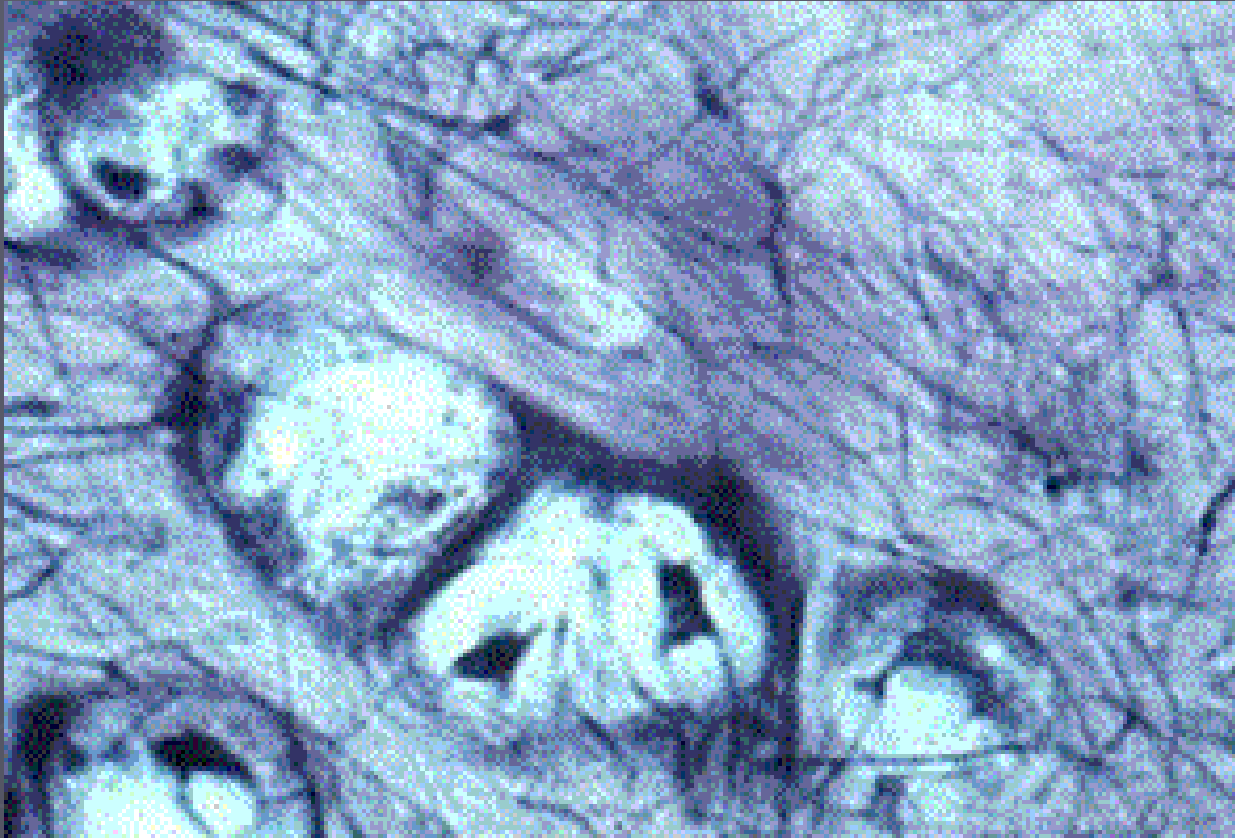
Transition from
granulation
tissue to scar



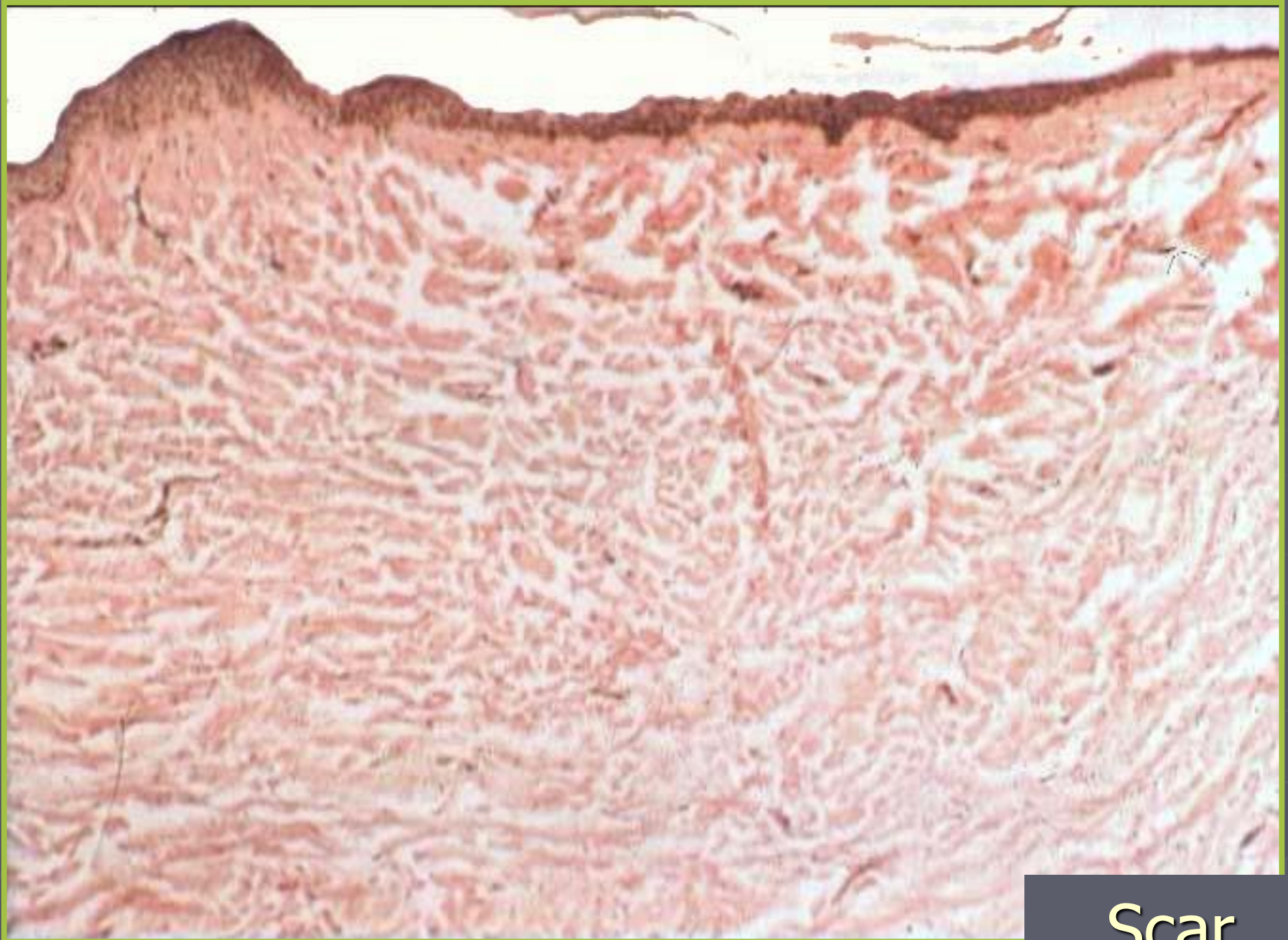
Epithelial
wound



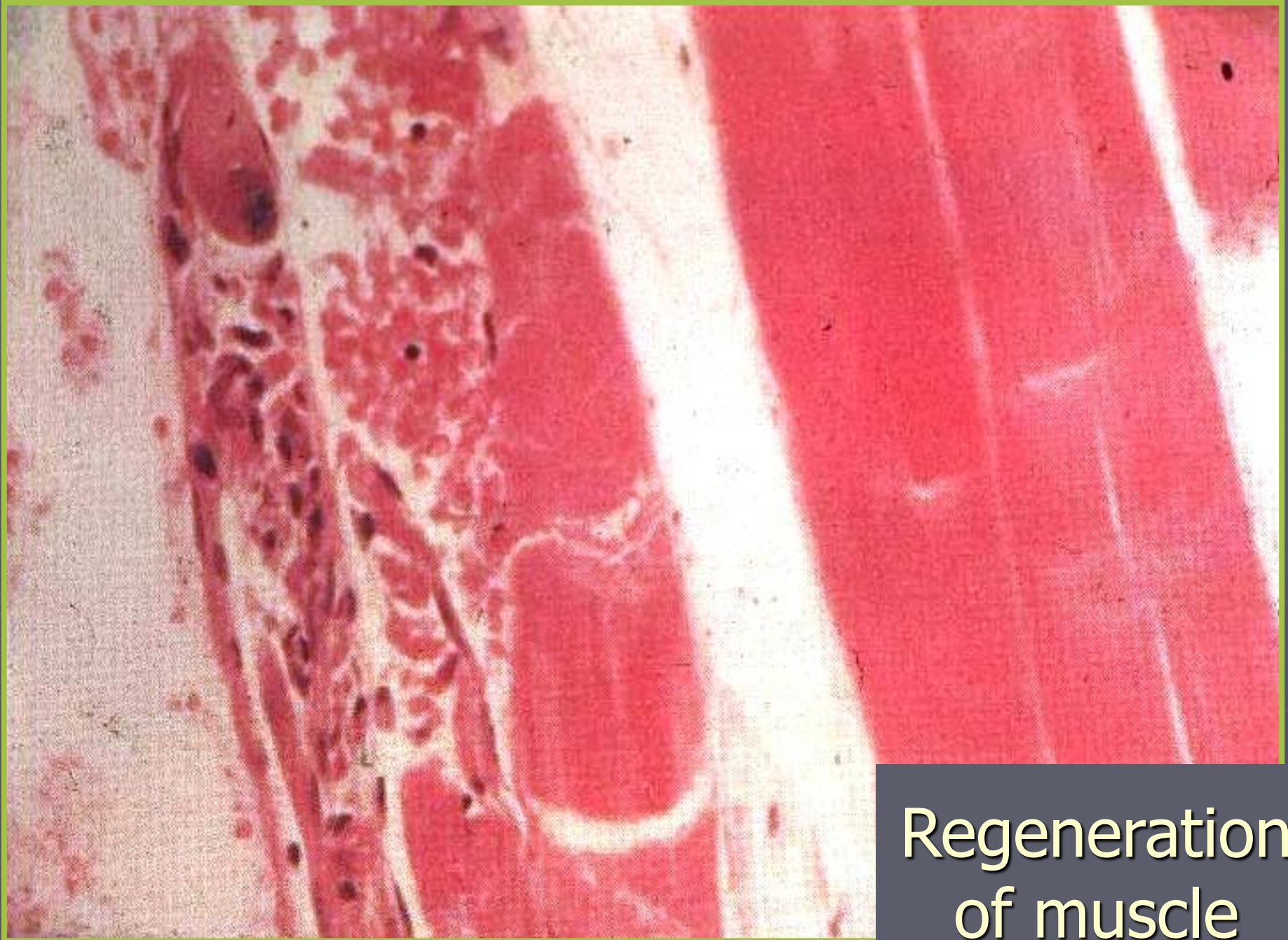
Granulation tissue



Scar - fibroblasts



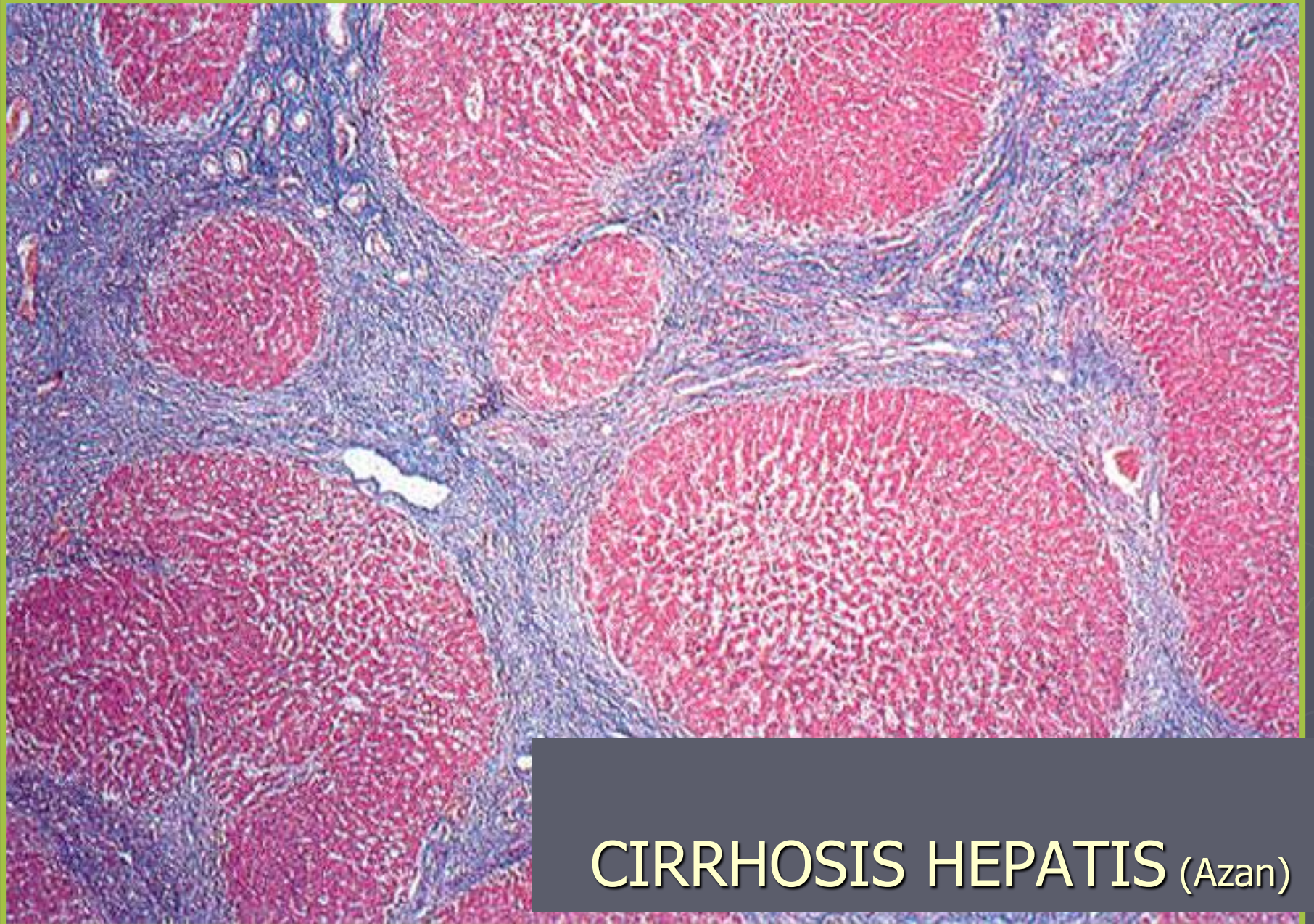
Scar



Regeneration
of muscle



Scar tissue replacing necrotized myocardium



CIRRHOSIS HEPATIS (Azan)

Regeneration of bones 2.

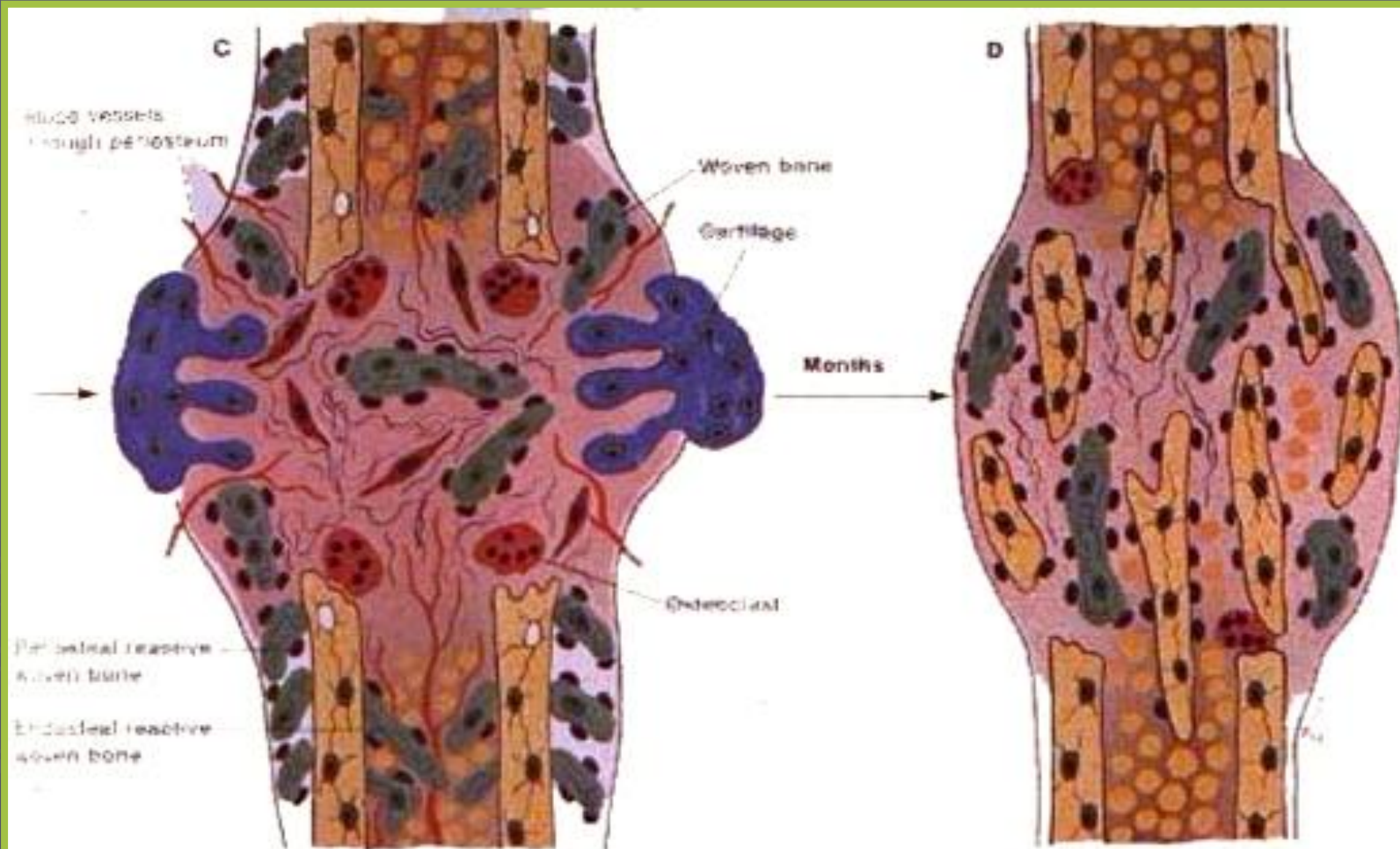


FIGURE 26-15 (continued).

(C) The reparative phase of fracture healing is characterized by the formation of a callus of cartilage and woven bone near the fracture site. The jagged edges of the original cortex have been remodeled and eroded by osteoclasts. The marrow space has been revascularized and contains reactive woven bone, as does the periosteal area. (D) In the remodeling phase, during which the cortex is revitalized, the reactive bone may be lamellar or woven. The new bone is organized along stress lines and mechanical forces. Extensive osteoclastic and osteoblastic cellular activity is maintained.