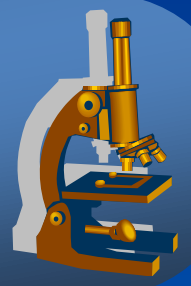
General pathology histopath. practice

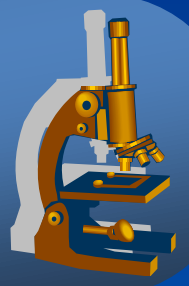


General pathology II.

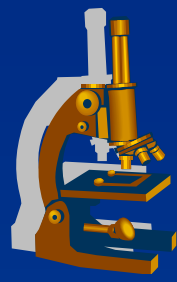
Circulation disorders

Inflammation I

Circulation disorders

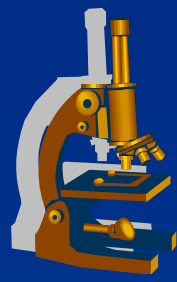


Circulation disorders



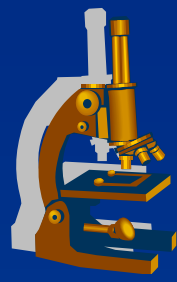
- Local
- Systemic
- Shock

Circulation disorders LOCAL



- Thrombosis
- Embolism
- Haemorrhage
- Oedema
- Hyperemia
- Ischemia
- Infarction

Thrombosis



- main cause of local circulation disorders

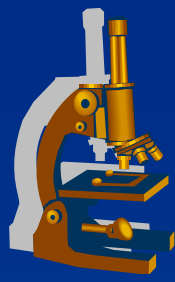
»***intravital*** formation of a pathological blood clot (thrombus) ***within vascular system***, due to platelets aggregation + transformation of fibrinogen into fibrin

possible obstruction of the blood flow through the circulatory system«

Circulation disorders

- Local
  - ⇨***thrombosis***
  - ⇨***embolism***
  - ⇨***hemorrhage***
  - ⇨***oedema***
- Systemic
  - ⇨***cardial***
  - ⇨***extracardial***

Thrombosis



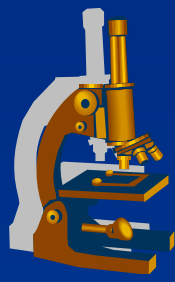
3 basic pathogenetic mechanisms:

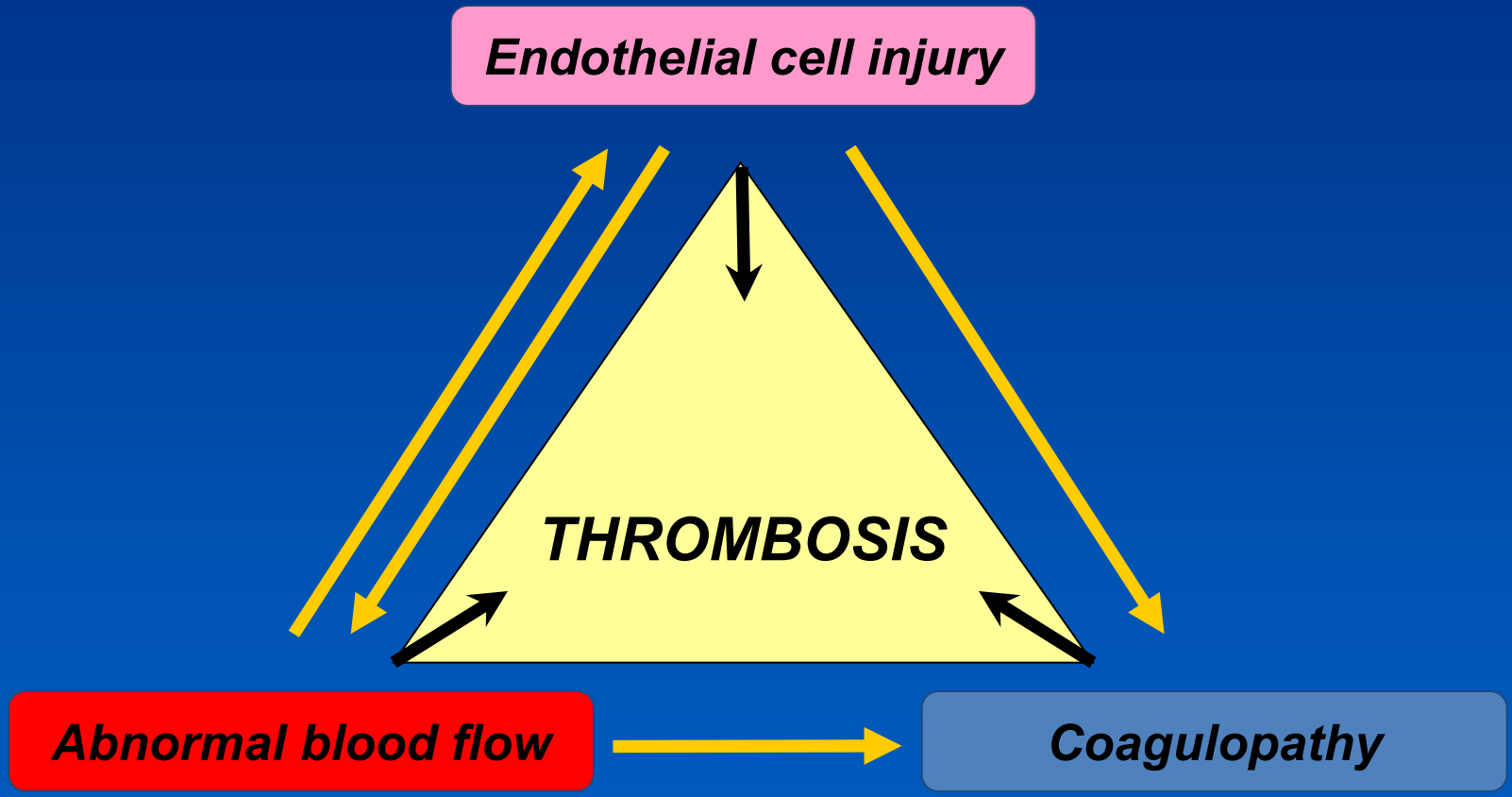
- ⇨**endothelial cell injury**
  - the most importatnt (trauma, AS, immune complexes, viruses, bacteria, inflammations, toxins) – FXII activation
- ⇨**blood flow disturbance**
  - stagnation past the point of injury, venous stasis in leg veins
- ⇨**blood constituents change, coagulopathy**
  - congenital: e.g. genetic mutation FV (Leiden)
  - acquired: hormonal contraception, tumor dissemination, DIC, hyperlipidemia, smoking

Circulation disorders

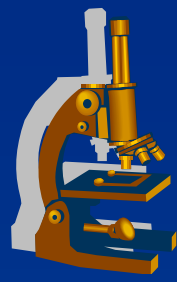
- Local
  - ⇨***thrombosis***
  - ⇨***embolism***
  - ⇨***hemorrhage***
  - ⇨***oedema***
- Systemic
  - ⇨***cardial***
  - ⇨***extracardial***

Pathogenetic mechanism





Thrombosis



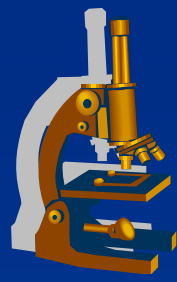
GROSS:

- ***mural thrombus*** *(cardial cavities, arteries)*
- ***obturating*** *(veins)*

Circulation disorders

- Local
  - ***thrombosis***
  - ***embolism***
  - ***hemorrhage***
  - ***oedema***
- Systemic
  - ***cardial***
  - ***extracardial***

Thrombosis



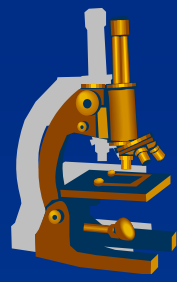
MICRO:

- ***red***
  - erythrocytes, in leg veins - stasis
- ***white***
  - thrombocytes, fibrin – cardial valves – turbulent flow
- ***mixed***
  - combination of both (stratified thrombus, e.g. cardial and aortic aneurysm)
- ***fibrin thrombus***
  - microscopic size, in small calibre arteries and capillaries, fibrin + platelets, CNS, kidney, lungs by shock, infections

Circulation disorders

- Local
  - ***thrombosis***
  - ***embolism***
  - ***hemorrhage***
  - ***oedema***
- Systemic
  - ***cardial***
  - ***extracardial***

Thrombosis



FATE OF THROMBI

- ***vessel wall hypoxia » platelet grow factors » reparative fibroproductive inflammation» thrombus fixation to the vessel wall » fibroblasts » thrombus retraction » recanalization + ORGANIZATION***
- ***growth of the thrombus***
- ***breaking off of the thrombus ⇨ embolism***
- ***fibrinolysis***

Circulation disorders

- Local
  - ***thrombosis***
  - ***embolism***
  - ***hemorrhage***
  - ***oedema***
- Systemic
  - ***cardial***
  - ***extracardial***

Source: Robbins basic pathology

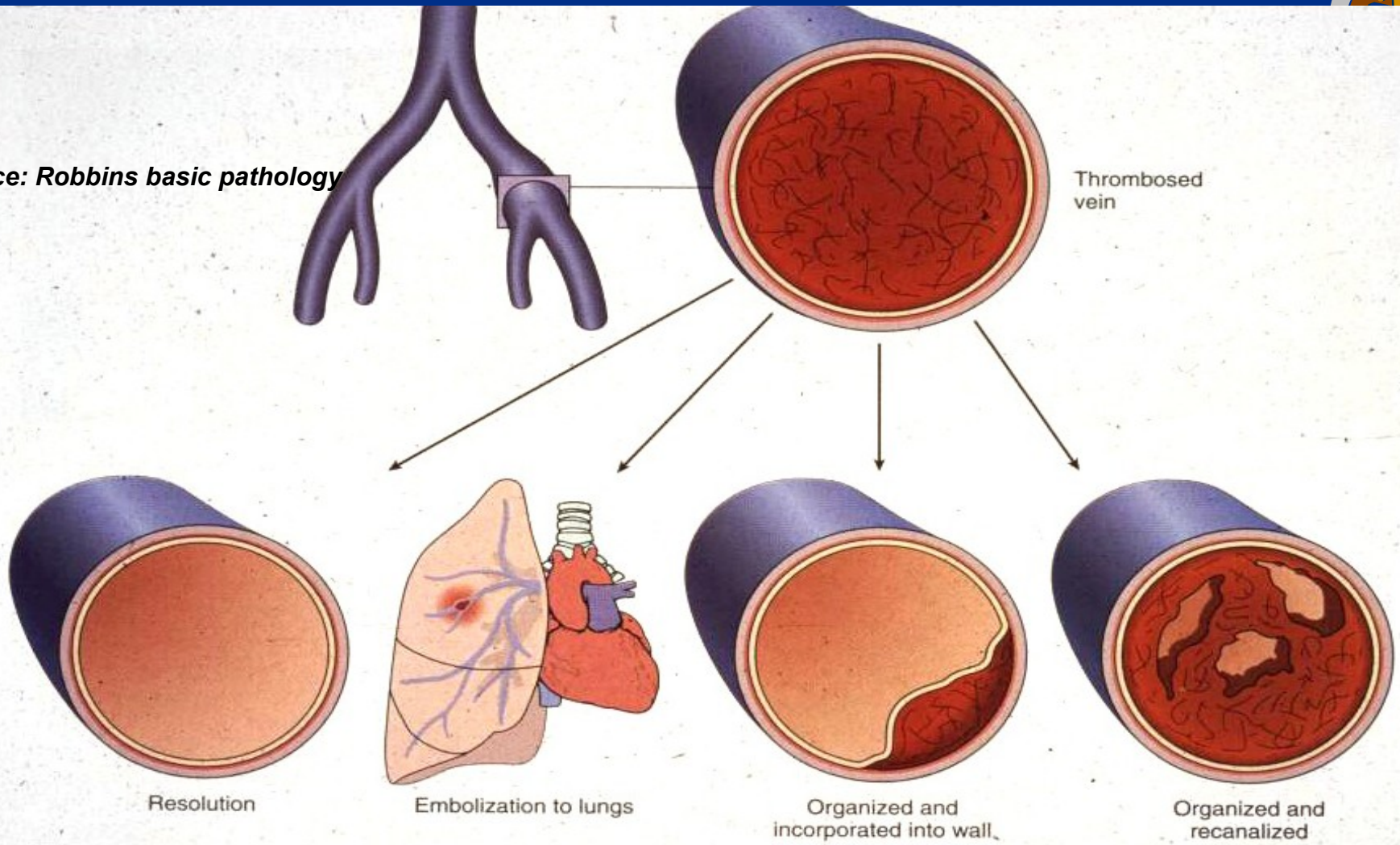
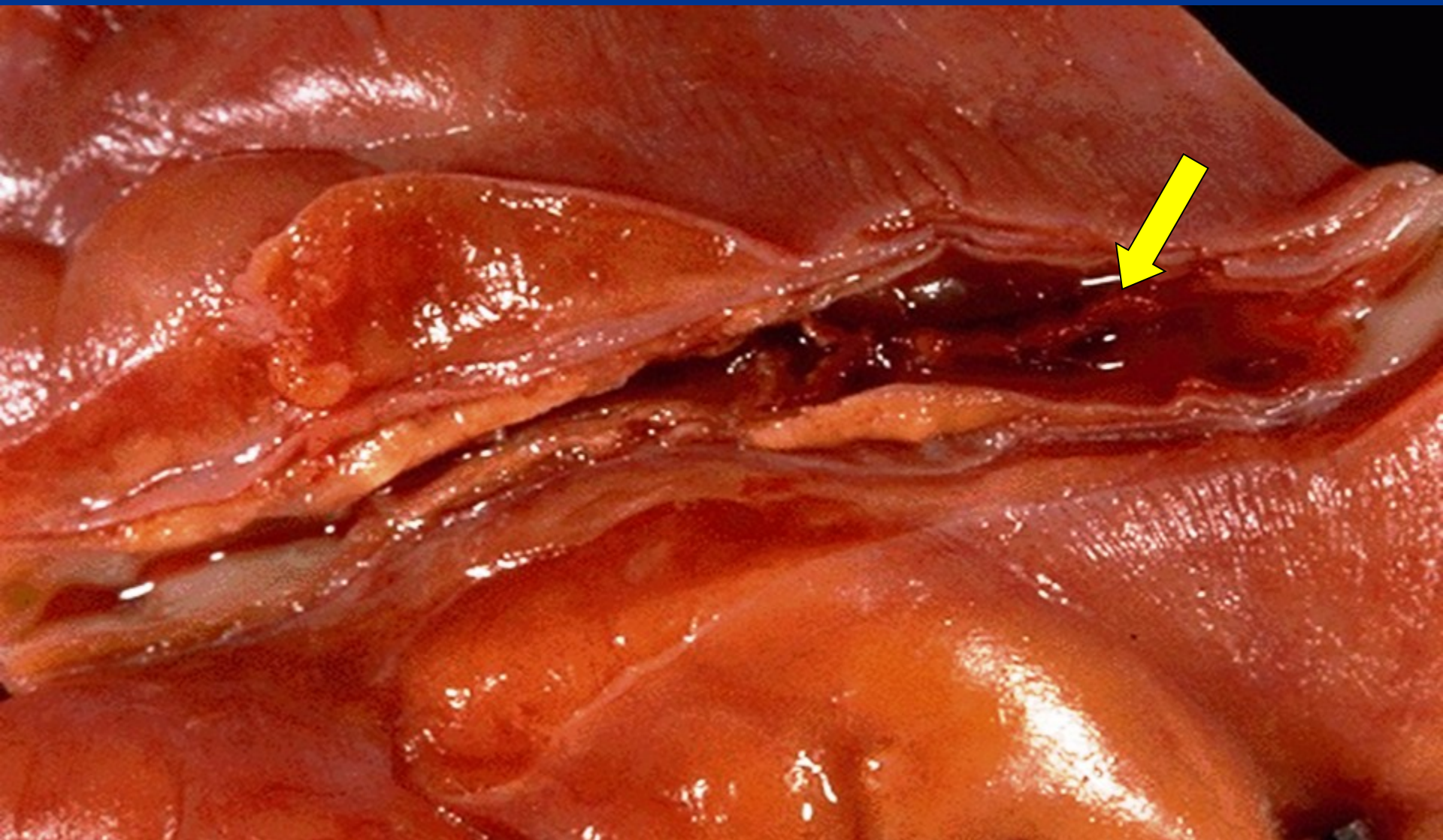
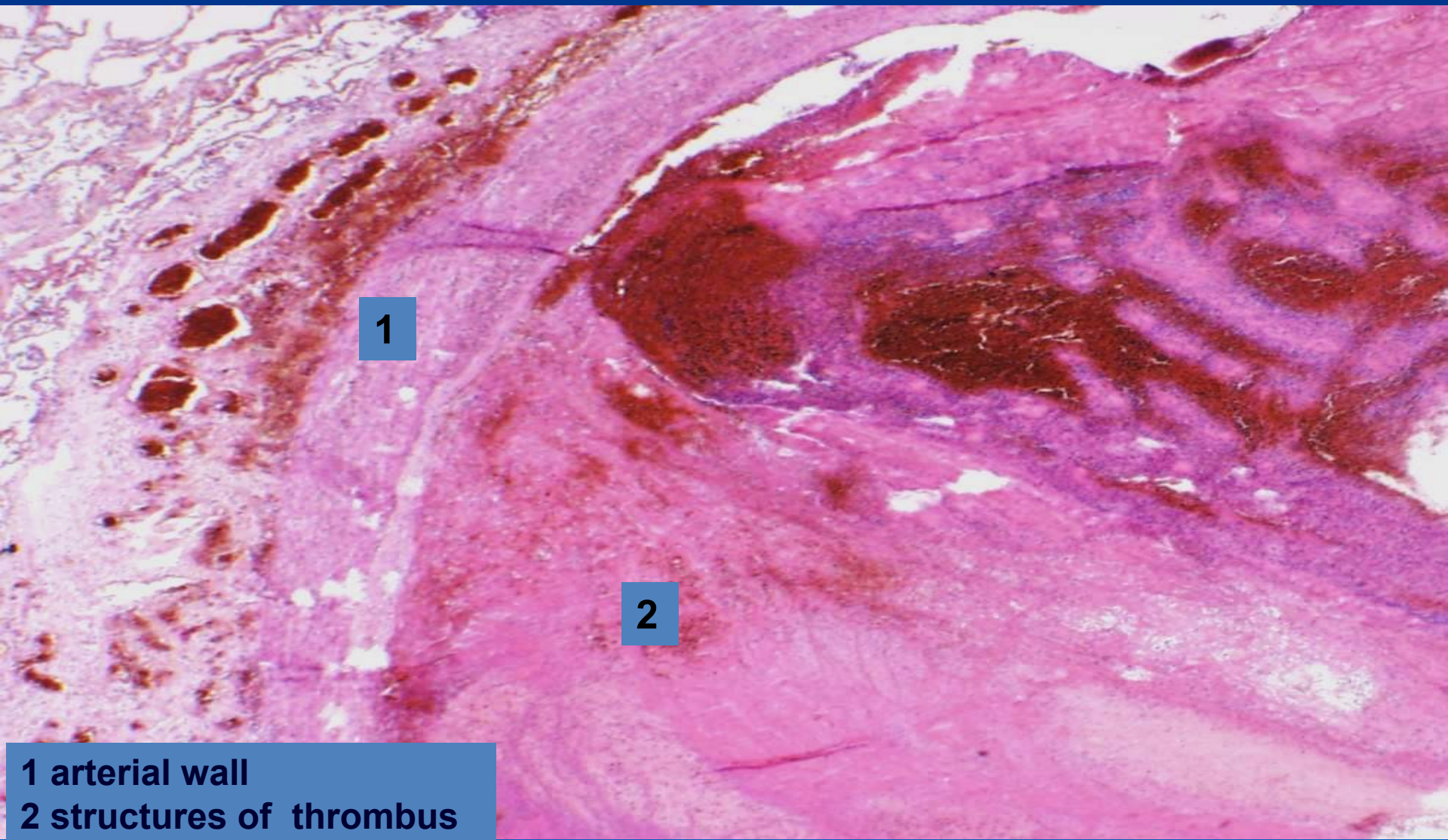


Figure 4–10

Coronary artery thrombosis

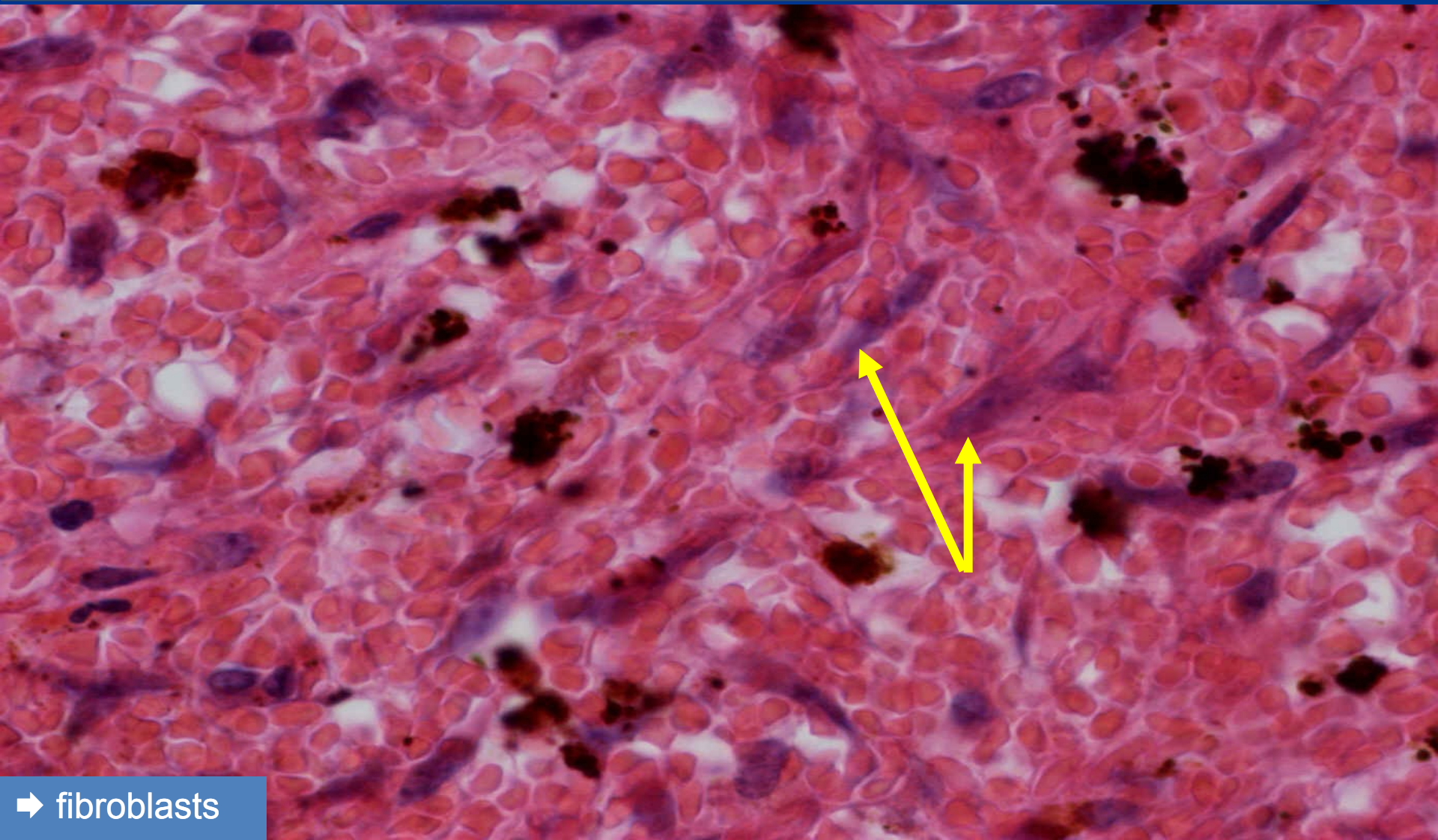


Organized mixed thrombus



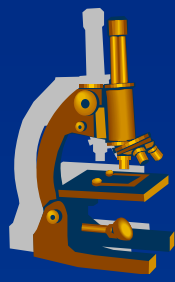
1 arterial wall
2 structures of thrombus

Mixed thrombus - organization
(detail 600x)



➡ fibroblasts

Microcirculation thrombosis DIC

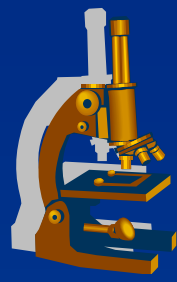


- Acquired coagulopathy, mortality 40%
- Etiopathogenesis:

  release of tissue factor (a part of the cell membranes) into blood or endothelial damage

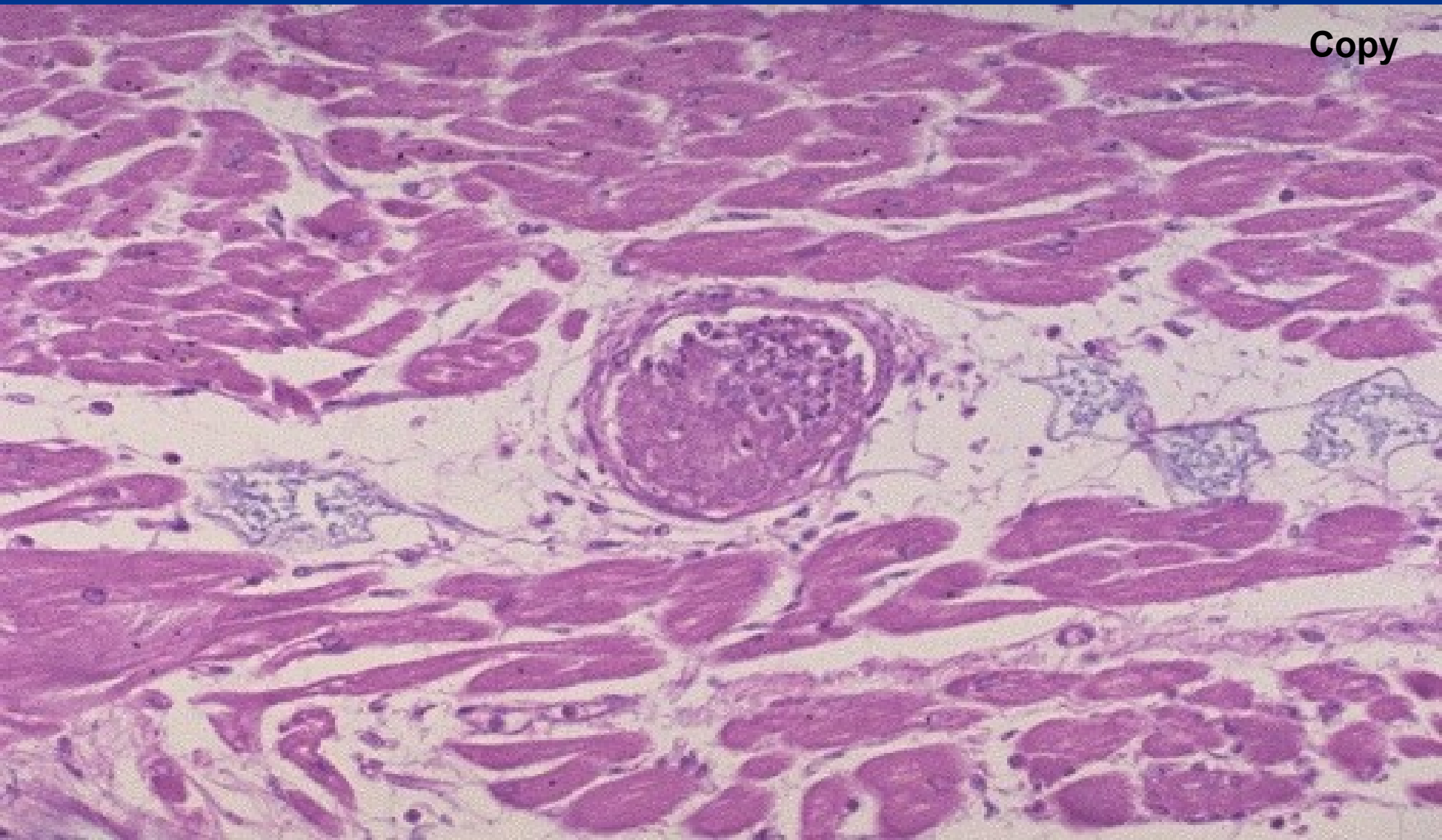
- Causes:
  - ⇨ ***Obstetric complications:***
    - intravenous embolism of amniotic fluid
    - placental abruption
  - ⇨ ***Infections***
    - menigococcal, stafylococcal sepsis, acute pancreatitis
  - ⇨ ***Shock***
  - ⇨ ***Tissue trauma (***burns, major surgery)

DIC

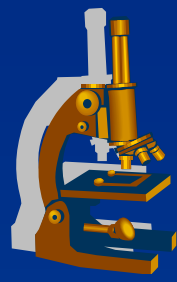


- 1st phase: hypercoagulation
  - ⇨ ***Morphology:***
    - multiple fibrin thrombi in the microcirculation
    - brain, lungs, kidney, heart, liver – ischemia, multi-organ failure
    - microangiopathic haemolytic anaemia
- 2nd phase: hypocoagulation:
  - ⇨ ***consumption of coagulation factors/fibrin degradation products***
  - ⇨ ***extreme hypocoagulability, fatal bleeding (surgical wounds, mucous membranes)***

Fibrin thrombus



Embolism



= a mass of movable material in the vascular system able to become lodged within a vessel and ***block its lumen****.*

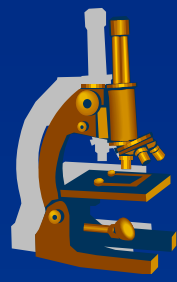
- thrombembolism:
  - ***most commonl***
  - ***from veins (deep v. of legs) into pulmonary arteries***
  - ***from heart/aorta into systemic arteries***

- paradoxical embolism:
  - ***from veins into arteries via foramen ovale apertum***

Circulation disorders

- Local
  - *thrombosis*
  - ***embolism***
  - *hemorrhage*
  - *oedema*
- Systemic
  - *cardial*
  - *extracardial*

Embolism

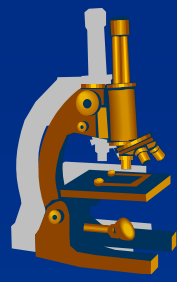


- Localization:
  - *venous embolism*
  - *arterial embolism*
  - *paradoxical embolism*
  - *portal embolism*

Circulation disorders

- Local
  - ***thrombosis***
  - ***embolism***
  - ***hemorrhage***
  - ***oedema***
- Systemic
  - ***cardial***
  - ***extracardial***

Embolism



- Classification:
  - ***thrombotic***
  - ***air***
  - ***fat***
  - ***cellular*** *(tumor dissemination)*
  - ***subcelullar*** *(DNA)*
  - ***amniotic fluid***

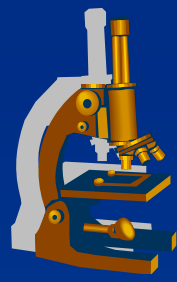
Circulation disorders

- Local
  - *thrombosis*
  - ***embolism***
  - *hemorrhage*
  - *oedema*
- Systemic
  - *cardial*
  - *extracardial*

Partially organized mixed blood clot

1 vascular wall
2 structures of the thrombus
➡vascular lumen

Haemorrhage



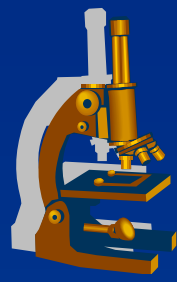
= loss of blood from the circulatory system, accumulation of blood in the intersticium – HAEMATOMA

- localization:
  - ***external x internal***
- origin:
  - ***arterial, venous, capillary***
- appearance:
  - ***petechiae, ecchymosis (subcutaneous), apoplexy (massive)***

Circulation disorders

- Local
  - ***thrombosis***
  - ***embolism***
  - ***hemorrhage***
  - ***oedema***
- Systemic
  - ***cardial***
  - ***extracardial***

Oedema



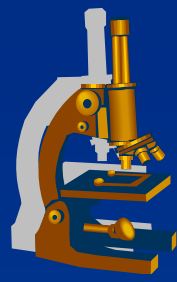
Abnormal accumulation of fluid in the intersticium.

- Types:
  - ***localized***
  - ***generalized***

Circulation disorders

- Local
  - ***thrombosis***
  - ***embolism***
  - ***hemorrhage***
  - ***oedema***
- Systemic
  - ***cardial***
  - ***extracardial***

Oedema

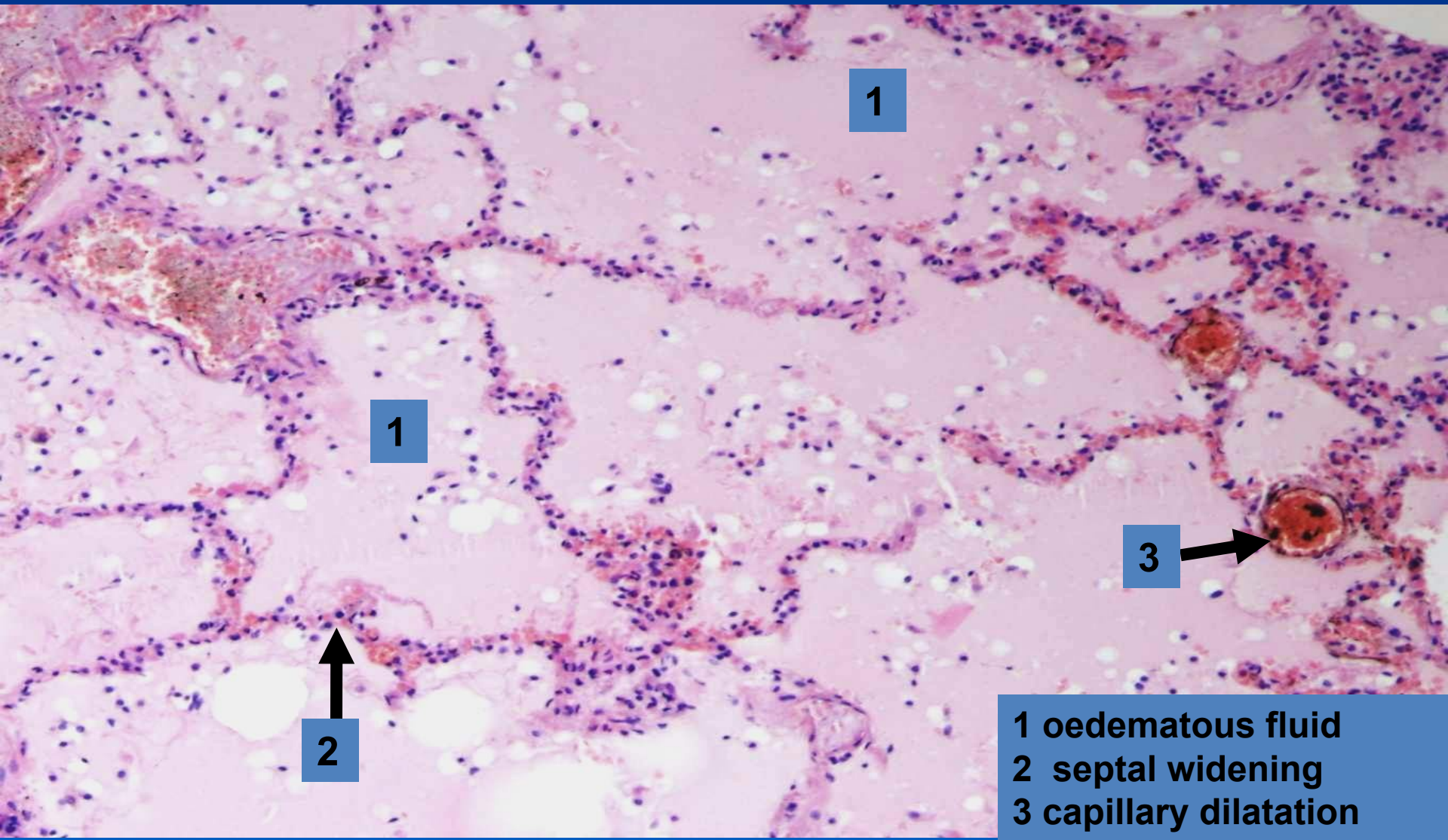


- Causes:
  - ***venous: ↑ intravenous pressure (e.g. chronic cardiac failure)***
  - ***inflammatory: increase of endothelial permeability***
  - ***hypoalbuminaemic: reduced oncotiic pressure***
  - ***lymphostatic oedema***

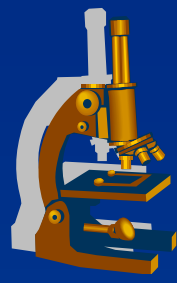
Circulation disorders

- Local
  - *thrombosis*
  - *embolism*
  - *hemorrhage*
  - ***oedema***
- Systemic
  - *cardial*
  - *extracardial*

Pulmonary oedema



Circulation disorders SYSTEMIC



CAUSES:

- **HEART**

  (ischemic heart disease, myocarditis, cardiomyopathy, pericarditis, valvular heart disease, endocarditis)

- **VESSELS**

  (systemic hypertension, pulmonary hypertension)

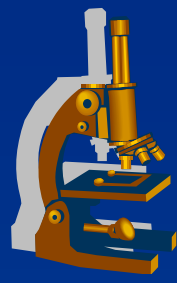
- **BLOOD DISORDERS**

  (polycytemia vera, anemia)

Circulation disorders

- Local
  - ⇨ *thrombosis*
  - ⇨ *embolism*
  - ⇨ *hemorrhage*
  - ⇨ *oedema*
- ***Systemic***
  - ⇨ *cardial*
  - ⇨ *extracardial*

Circulation disorders SYSTEMIC



- All causes subsequently manifest as ***HEART FAILURE***

Left ventricle failure

Right ventricle failure

Congestive heart failure – both ventricles

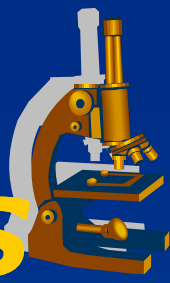
- ⇨ ***„forward“ failure – usually low cardiac output***
- ⇨ ***„backward“ failure – venous congestion***

cardiogenic shock

Circulation disorders

- Local
  - ⇨ ***thrombosis***
  - ⇨ ***embolism***
  - ⇨ ***hemorrhage***
  - ⇨ ***oedema***
- ***Systemic***
  - ⇨ ***cardial***
  - ⇨ ***extracardial***

Circulation disorders
MORPHOLOGICAL ASPECTS

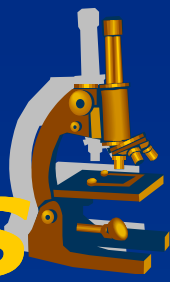


- **HEART**:
  - ⇨ ***Dilatation (without hypertrophy)***
    - acute cardiac failure (pulmonary embolism)
  - ⇨ ***Hypertrophy***
    - ***concentric*** x ***ecccentric*** (+ dilatation)
    - Starling`s principle, depletion of adaptation mechanisms

Circulation disorders

- Local
  - ⇨ ***thrombosis***
  - ⇨ ***embolism***
  - ⇨ ***hemorrhage***
  - ⇨ ***oedema***
- Systemic
  - ⇨ ***cardial***
  - ⇨ ***extracardial***

Circulation disorders
MORPHOLOGICAL ASPECTS

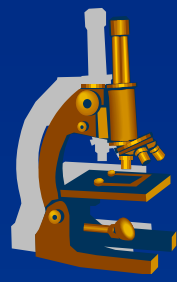


- **EXTRACARDIAL**
  - ⇨ *venous congestion +/- cyanosis*
  - ⇨ *oedema (pulmonary, systemic)*
  - ⇨ *induration*

Circulation disorders

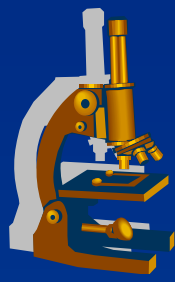
- Local
  - ⇨ *thrombosis*
  - ⇨ *embolism*
  - ⇨ *hemorrhage*
  - ⇨ *oedema*
- Systemic
  - ⇨ *cardial*
  - ⇨ ***extracardial***

Heart failura



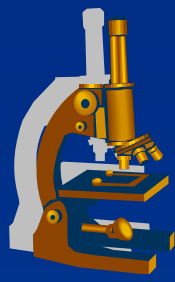
- LV x RV
- ACUTE x CHRONIC failure

Heart failure



- LV failure:
  - ⇨ *acute - morphology:*
    - congestion in pulmonary circulation
    - pulmonary oedema
  - ⇨ *chronic - morphology:*
    - chronic pulmonary venous congestion – „brown“ lung induration (siderophages), later signs of RV failure

Pulmonary oedema

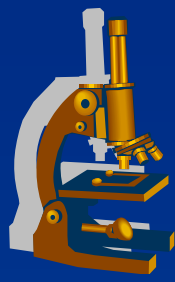


- Left-sided heart failure

  (LV insufficiency, mitral stenosis)

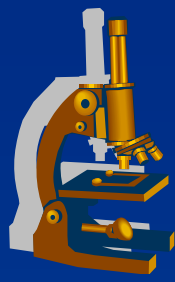
- congestion in pulmonary veins, capillaries

Pulmonary oedema



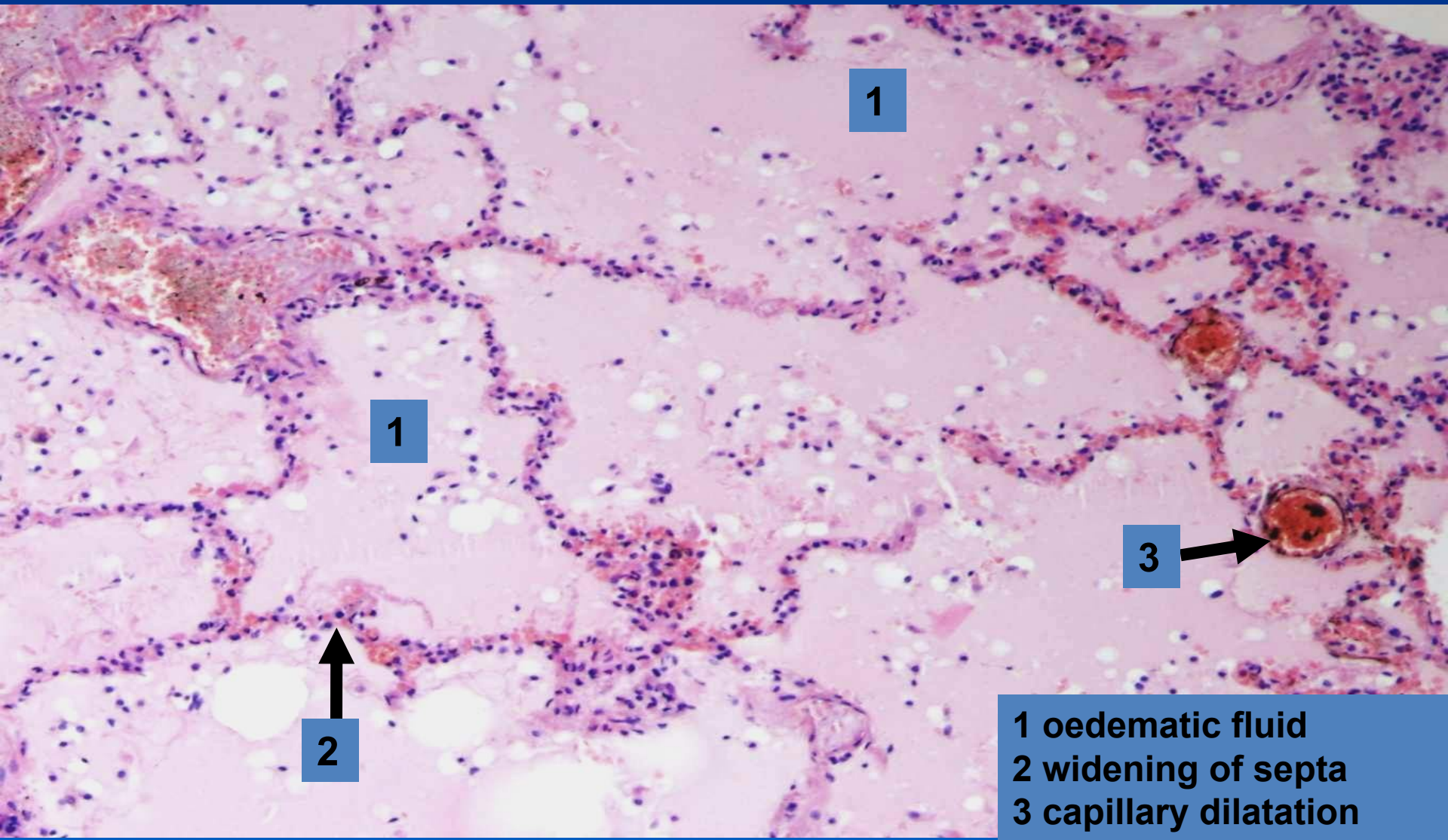
- GROSS:
  - ⇨ *enlarged, heavy lung of tougher consistency,*
  - ⇨ *fluid running out from tissue and bronchi*
  - ⇨ *in chronic venous congestion: superimposed oedema in the setting of tougher, rusty tissue*

Pulmonary oedema

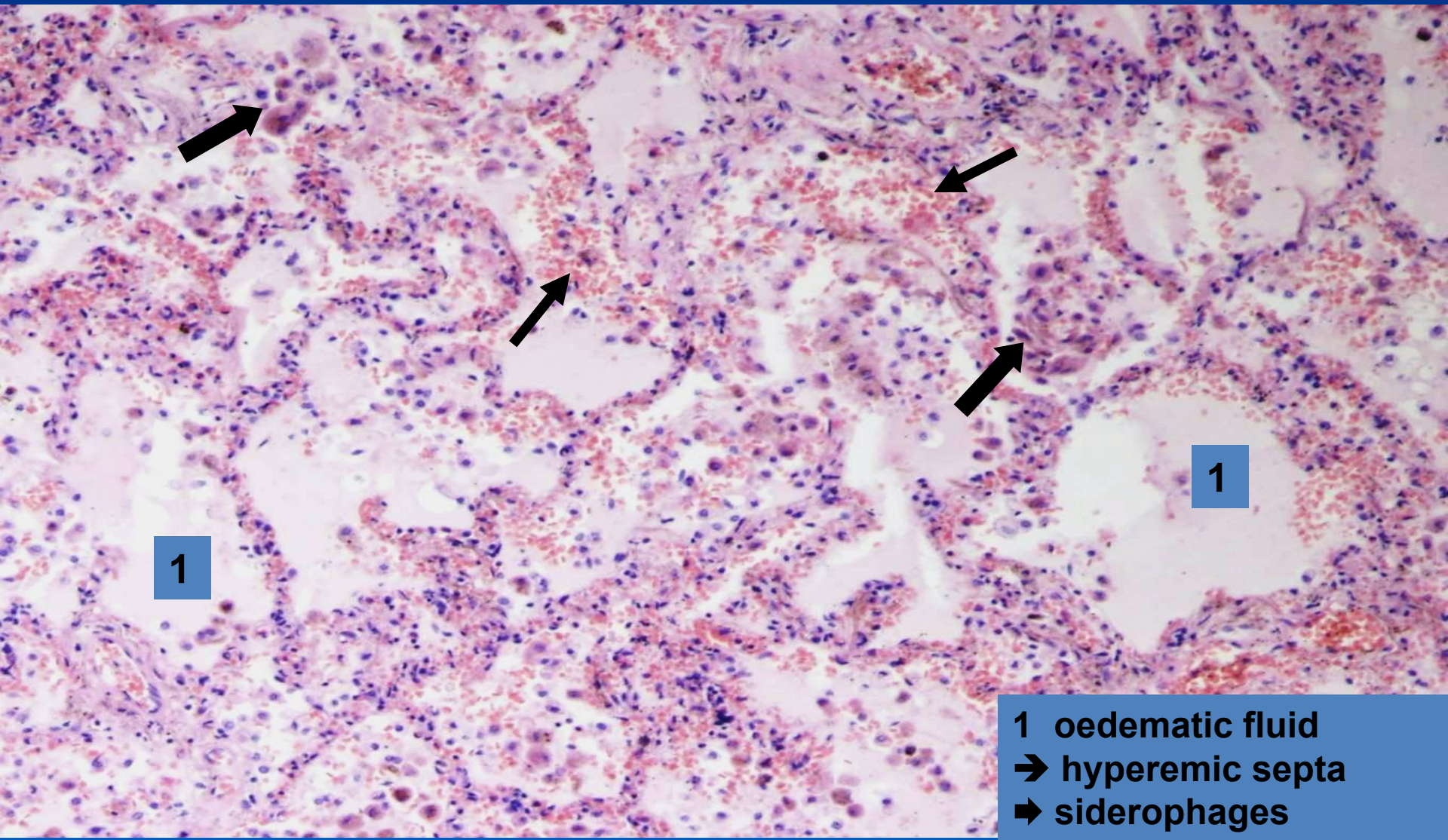


- MICRO:
  - ⇨ *dilatated septal capillaries filled with blood*
  - ⇨ *widened septa*
  - ⇨ *intraalveollar homogennous eosinophilic material (oedematic fluid)*
  - ⇨ *long-standing: extravasation and breakdown of red blood cells (haemosiderin, siderophages)*

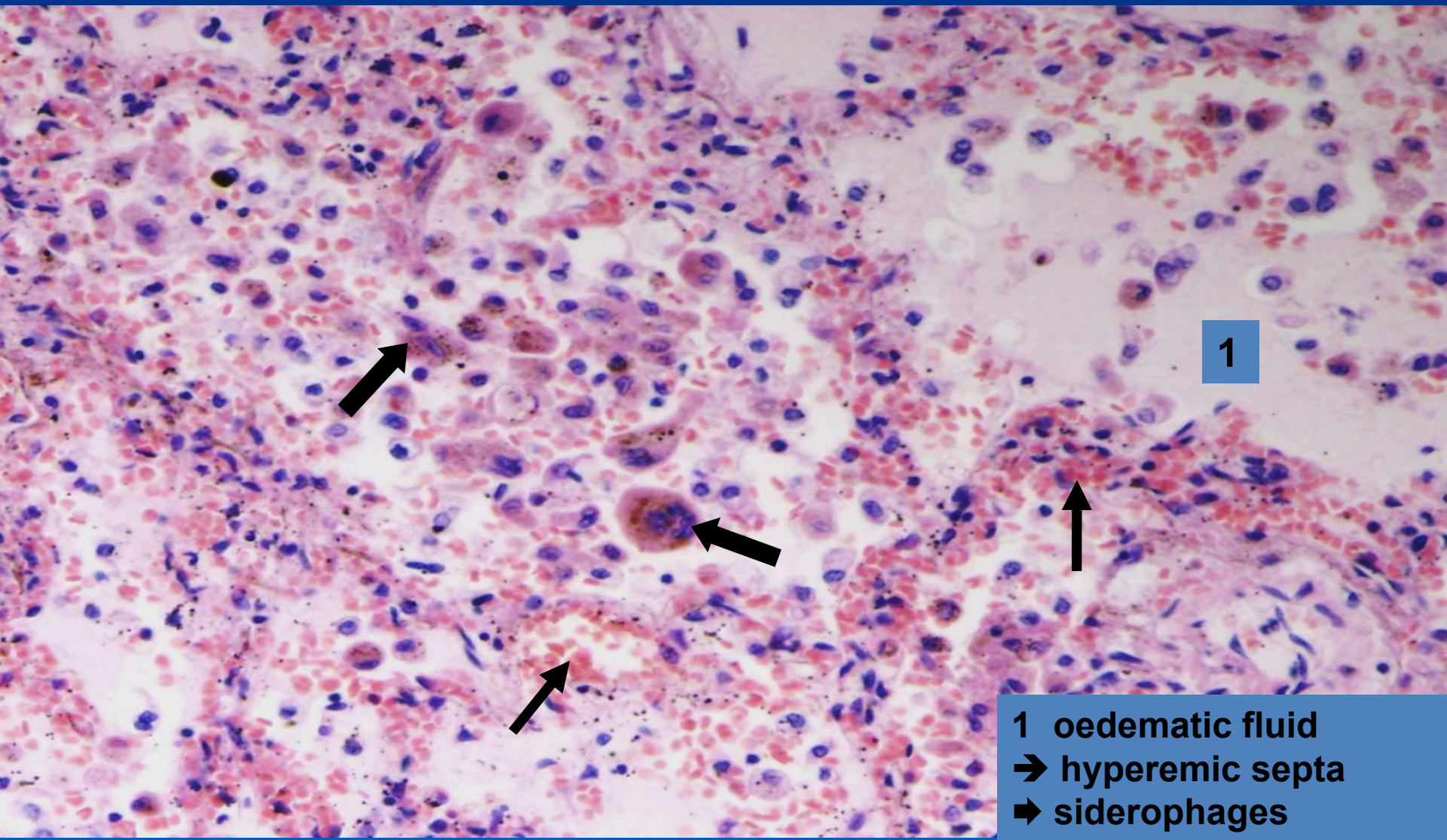
Pulmonary oedema



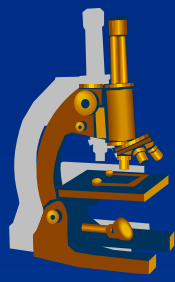
Chronic pulmonary venous congestion



Chronic pulmonary venous congestion *(detail)*

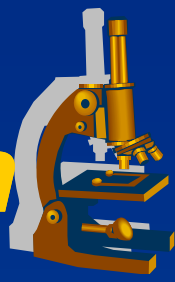


Heart failure



- RV insufficiency:
  - ⇨*acute - morphology:*
    - acute cor pulmonale (massive thrombembolism)
    - acute congestion in systemic circulation (splanchnic region) ⟶ congestive necrosis
  - ⇨*chronic - morphology:*
    - chronic hepatic venous congestion, congestion in portal region (spleen, mucous membranes of GIT), kidneys
    - congestive skin/soft tissue changes – trophic, oedemas
    - venous congestion in brain

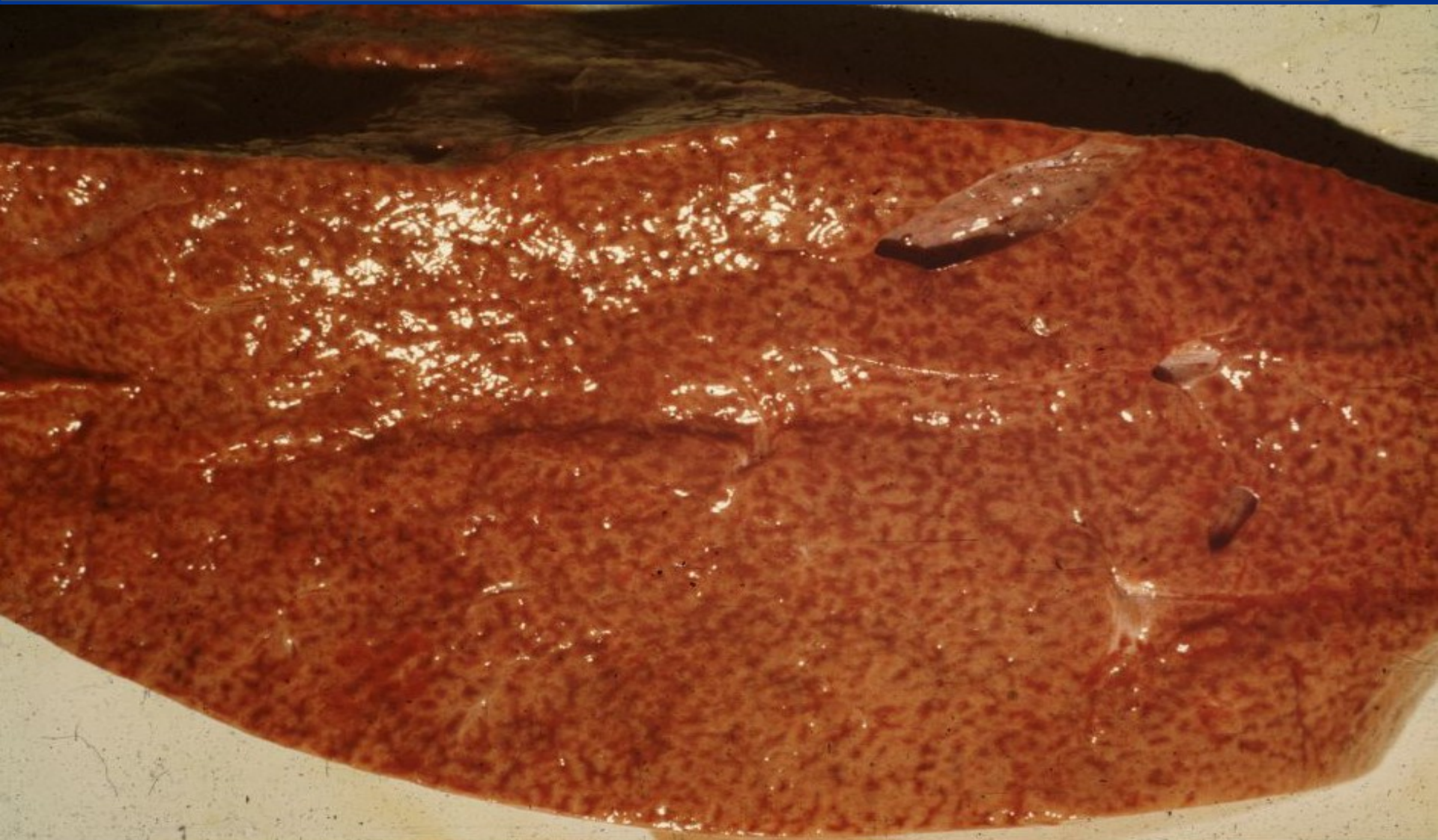
Hepatic venous congestion



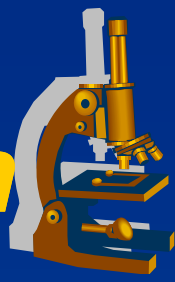
- GROSS:
  - ⇨ *enlarged, heavy liver*
  - ⇨ *dark – reddish brown color*
  - ⇨ *cardiac fibrosis (induration)*
  - ⇨ *combination with chronic hypoxemic steatosis – nutmeg liver*

Hepatic venous congestion

("nutmeg" liver)

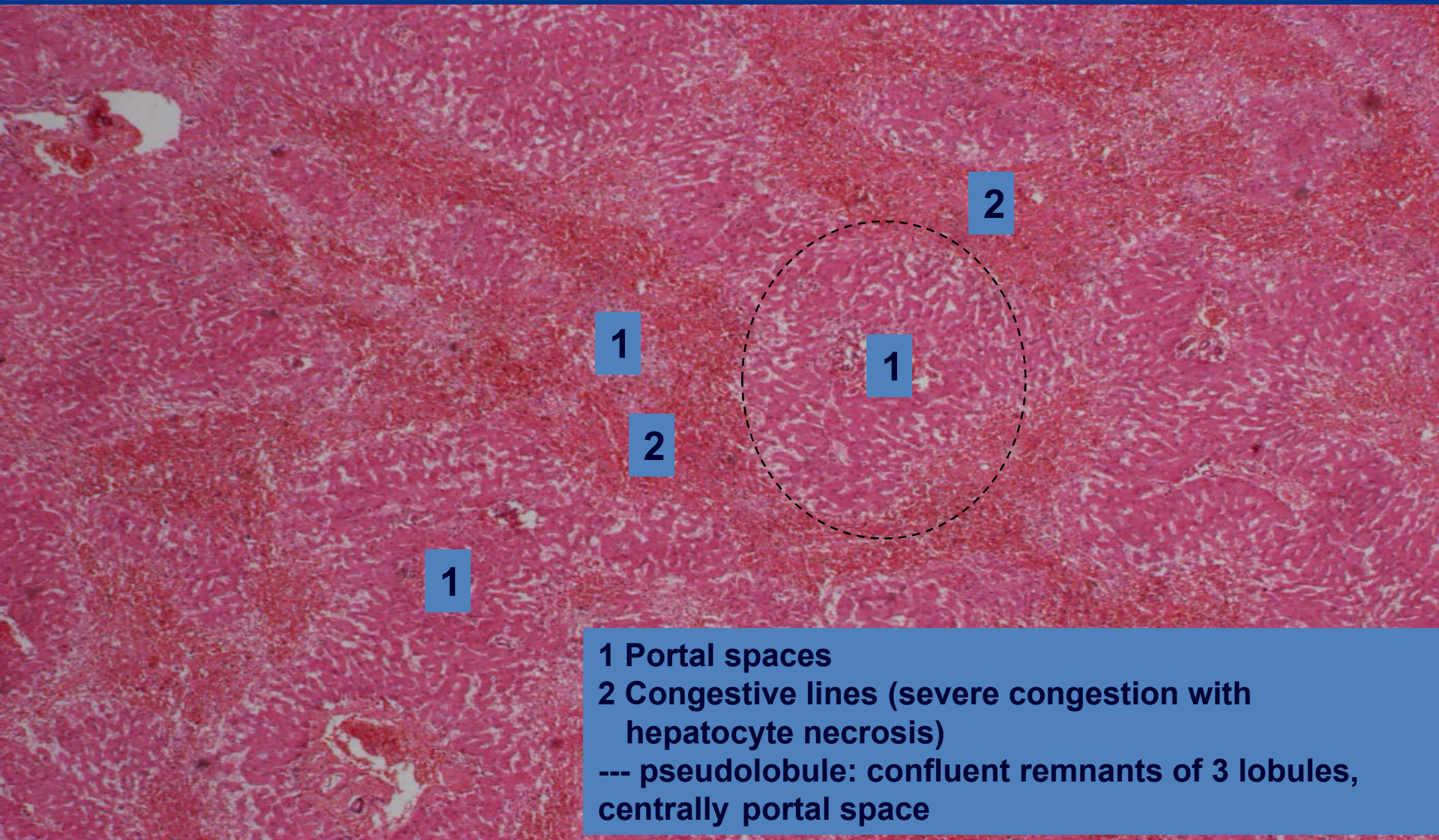


Hepatic venous congestion



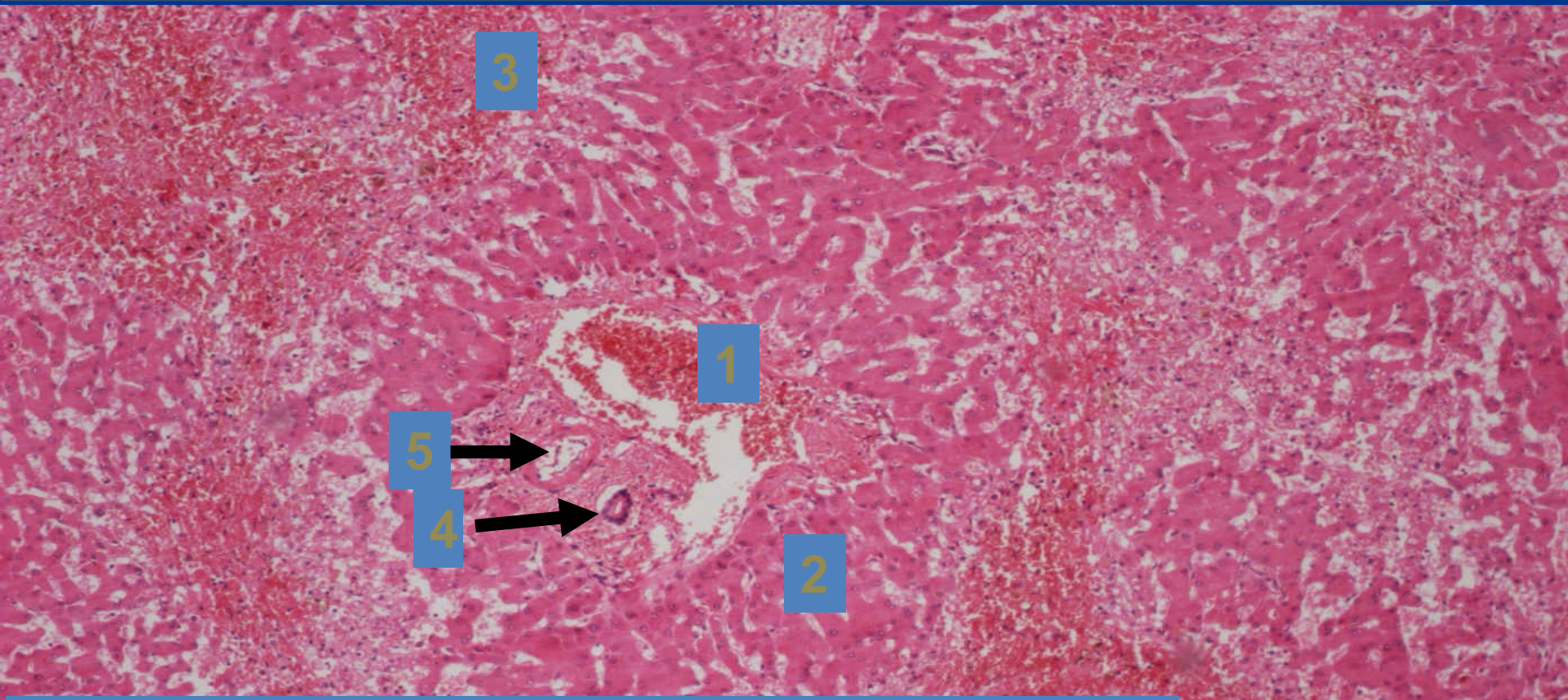
- MICRO:
  - ⇨ *central veins and sinusoidal dilatation*
  - ⇨ *centrolobular hepatocytic atrophy, necrosis*
  - ⇨ *„lines“ of congestion*

Hepatic venous congestion



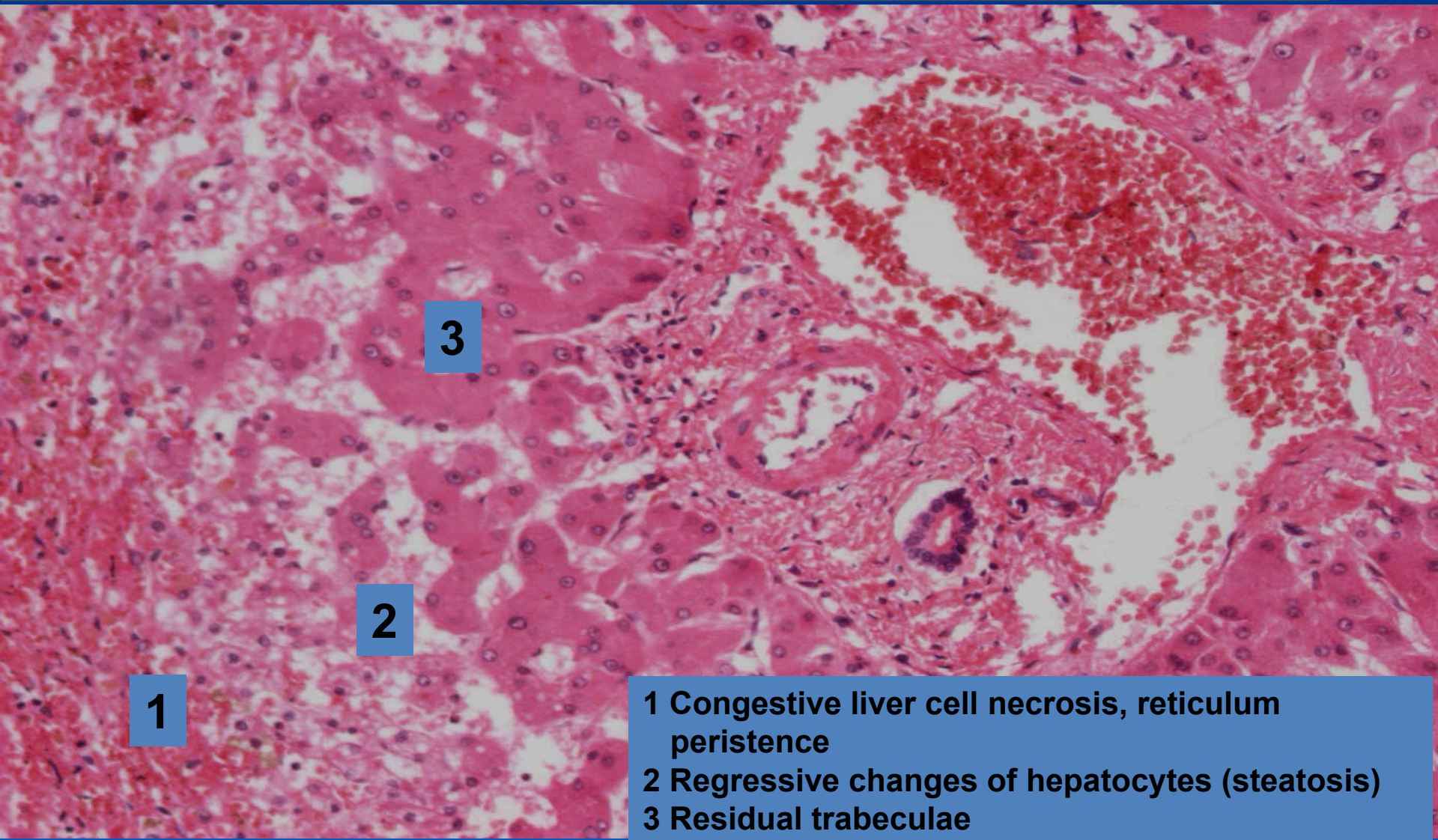
1 Portal spaces
2 Congestive lines (severe congestion with hepatocyte necrosis)
--- pseudolobule: confluent remnants of 3 lobules, centrally portal space

Hepatic venous congestion
("nutmeg" liver), detail



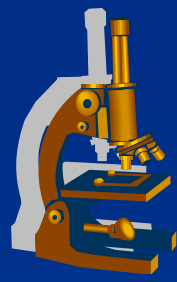
1 Portal space-dilatation of portal vein branch
2 Pseudolobule
3 Congestion lines
4 Bile duct
5 Hepatic artery branch

Hepatic venous congestion
("nutmeg" liver), portal area detail



1 Congestive liver cell necrosis, reticulum peristence
2 Regressive changes of hepatocytes (steatosis)
3 Residual trabeculae

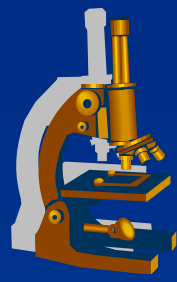
Shock



»Life-threatening medical condition that occurs due to inadequate substrate for aerobic cellular respiration. In the early stages this is generally an inadequate tissue level of oxygen (low organ perfusion).«

- Lethal without adeqaute medical therapy.

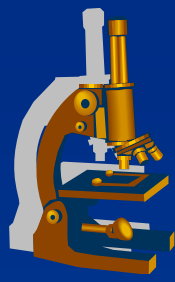
Shock



- Factors:
  - ⇨ ***Insufficient circulating volume (hypovolaemic)***
    - ↓ periferal vascular resistance
    - ↑ vascular wall permeability
    - blood loss
  - ⇨ ***Low cardiac output (cardiogenic)***
    - Acute heart failure

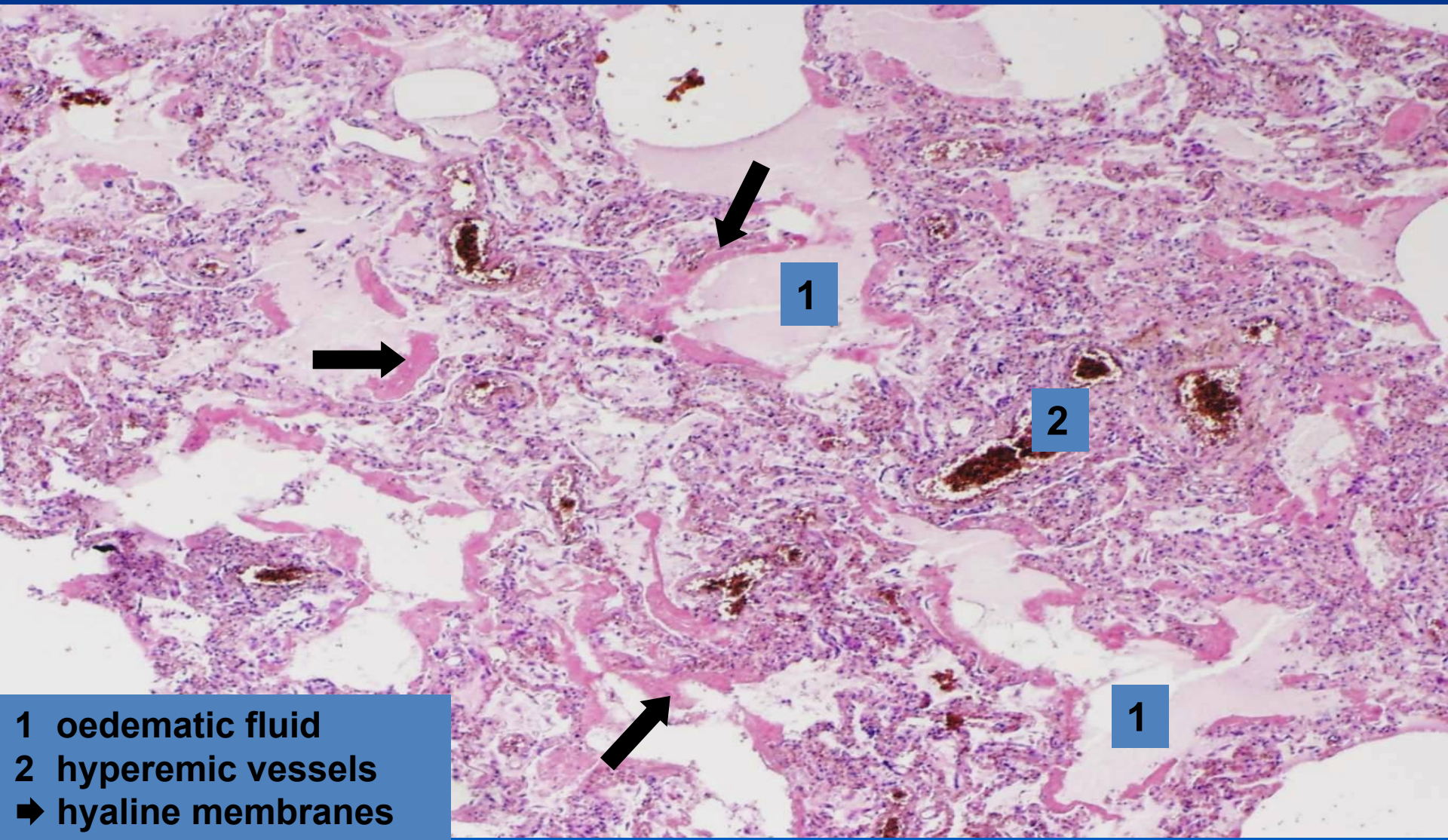
      (acute myocardial infarction, massive pulmonary embolism, cardiac tamponade, tension pneumothorax)
  - ⇨ ***Infection (bacterial toxaemia)***

Shock - MORPHOLOGY



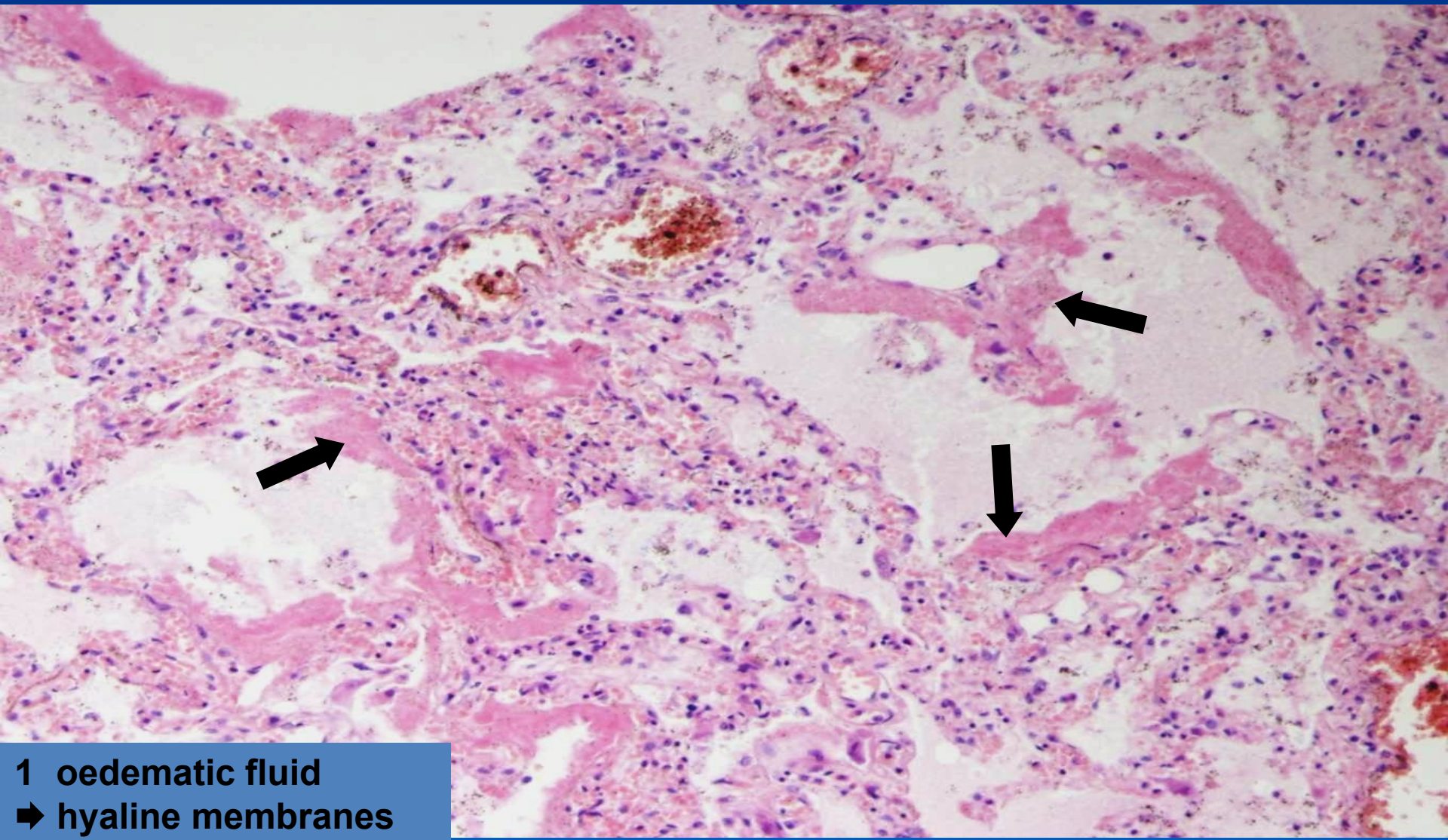
- Multiple organ failure (organ ischemia)
  - ⇨ ***brain***
  - ⇨ ***heart***
  - ⇨ ***kidney – acute tubular necrosis (ATN)***
  - ⇨ ***lungs – diffuse alveolar damage/ acute respiratory distress syndrome (DAD/ARDS)***
  - ⇨ ***adrenal glands, gastrointestinal tract)***

DAD/ARDS



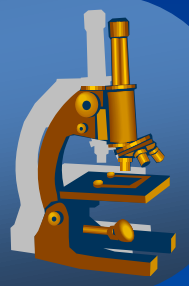
1 oedematic fluid
2 hyperemic vessels
➡ hyaline membranes

DAD/ARDS
(detail)

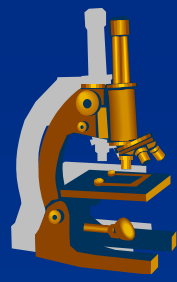


1 oedematic fluid
➡ hyaline membranes

Inflammation



Inflammation

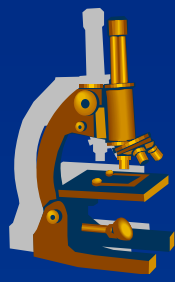


*= PROTECTIVE RESPONSE intended to eliminate harmful agents, accompanied with **alterative, exudative, proliferative components and immune response**.*

- types:
  - ⇨ ***acute inflammation***
  - ⇨ ***chronic inflammation***

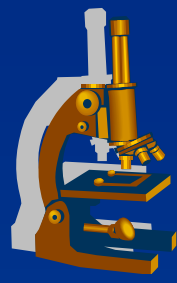
  - ⇨ ***nonspecific inflammation***
  - ⇨ ***granulomatous inflammation (specific)***

Inflammation



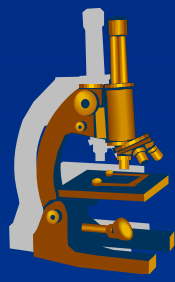
- Macroscopic appearances:
  - ⇨ ***rubor (redness)***
  - ⇨ ***calor (heat)***
  - ⇨ ***dolor (pain)***
  - ⇨ ***tumor (swelling)***
  - ⇨ ***functio laesa (loss of function)***

Inflammation



- Microscopic appearances:
  - ⇨ ***ALTERATION:***
    - regressive changes, necrosis
  - ⇨ ***EXUDATION:***
    - vascular leakage of protein-rich fluid and blood cells
    - ***exudate X transudate***

Inflammation



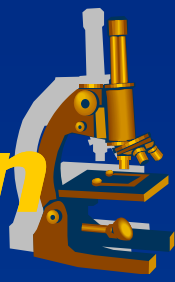
⇨ ***PROLIFERATION:***

- proliferation of fibroblasts and capillaries
- formation of granulation and fibrous tissue

⇨ ***IMMUNE RESPONSE:***

- antigen presentation
- T and B-lymphocytes reaction
- production of antibodies by plasma cells
- memory cells

NONSPECIFIC inflammation



Classification:

⇨ ***alterative:***

- alteration of tissue
- ***viral hepatitis, prion diseases [Creutzfeld-Jacob, BSE], diphteric myocarditis***

⇨ ***exudative:***

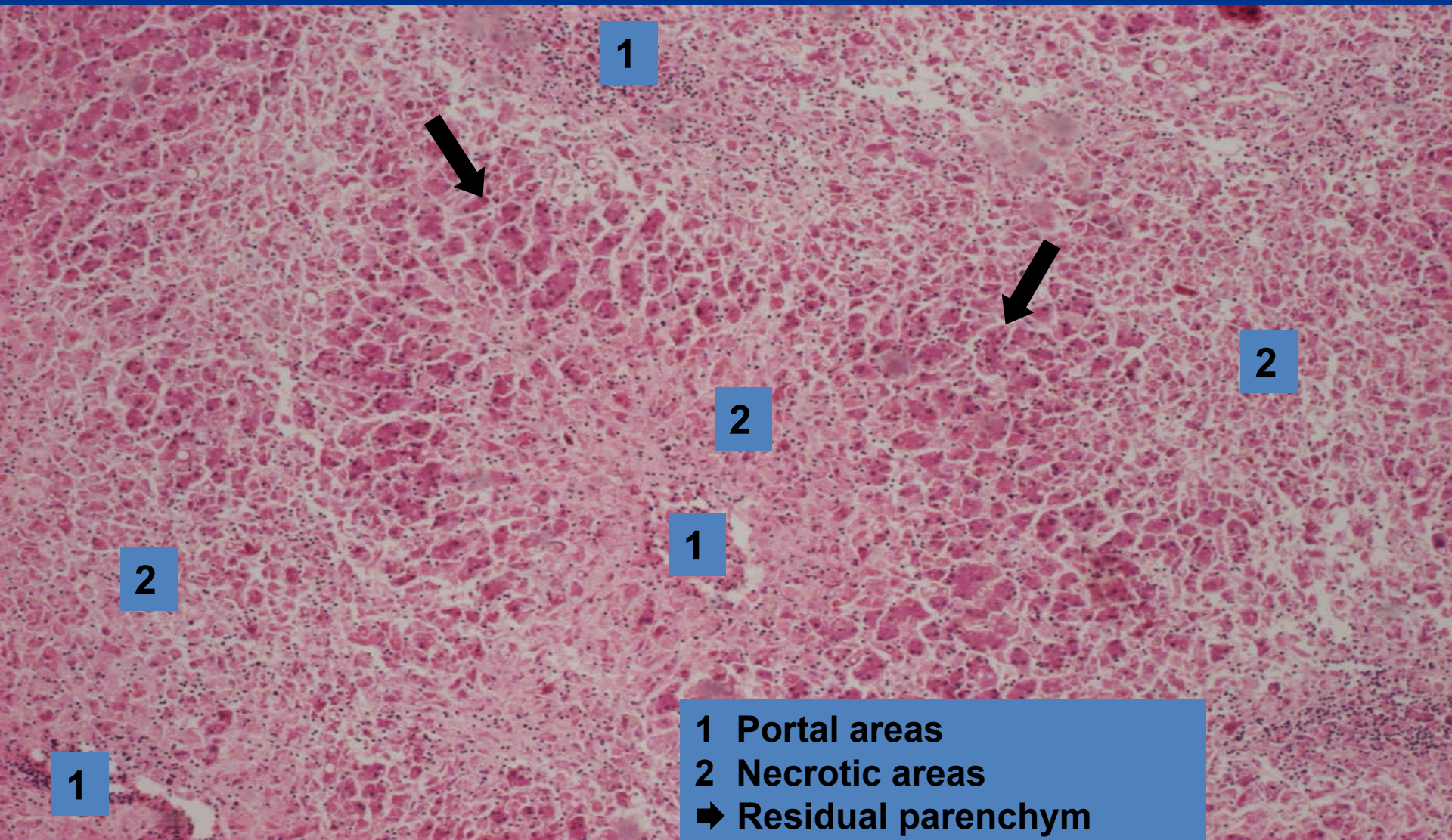
- most common, exudation prevails
- superficial and deep
- ***serous, fibrinous, nonpurulent, purulent, gangrenous***

⇨ ***proliferative:***

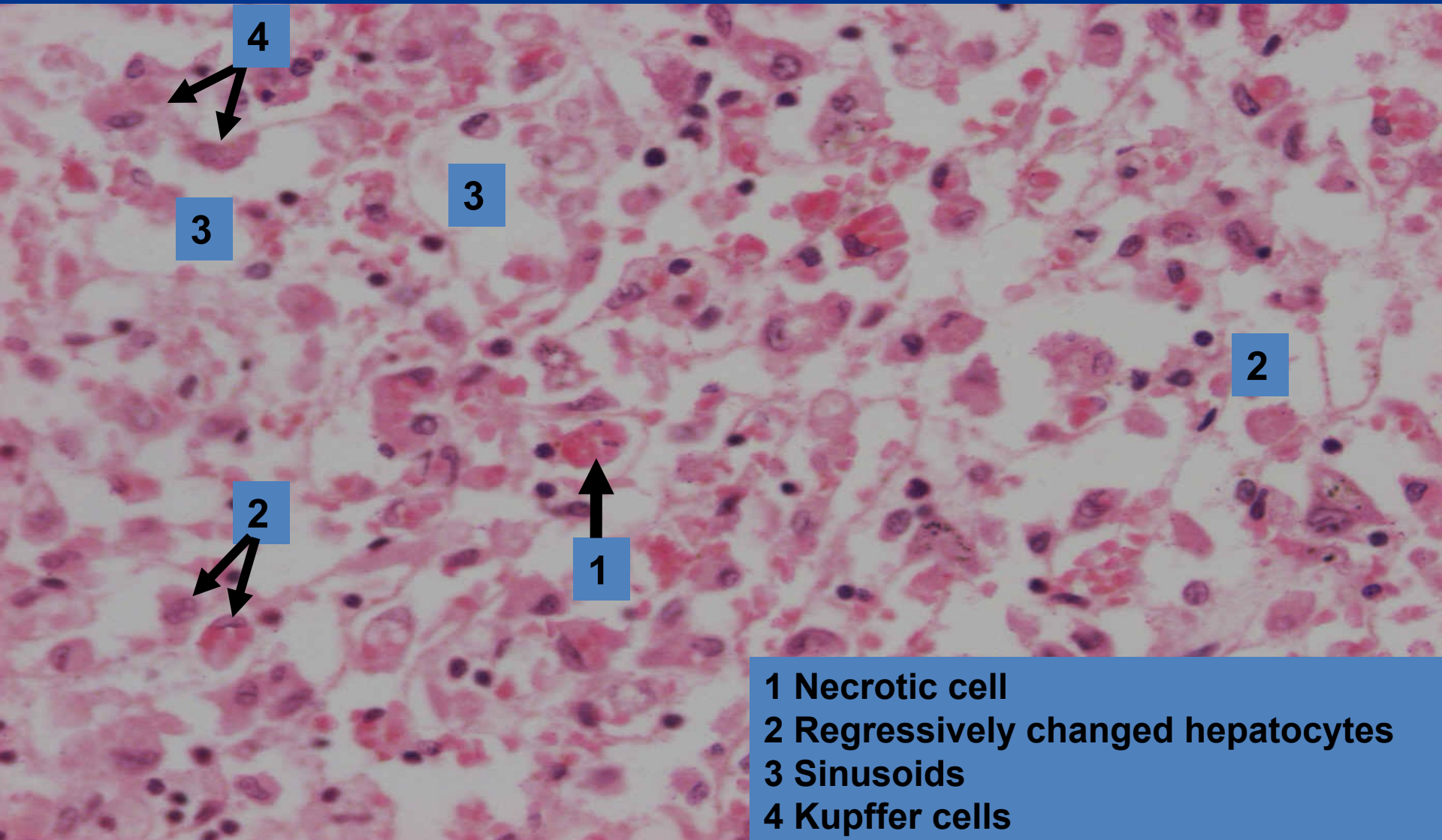
- formation of fibrous tissue

Alterative inflammation
(liver necrosis)

Alterative inflammation
(massive necrosis)

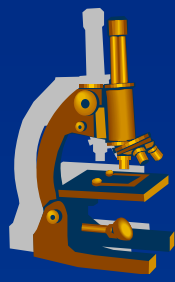


Alterative inflammation
(massive necrosis - detail)



1 Necrotic cell
2 Regressively changed hepatocytes
3 Sinusoids
4 Kupffer cells

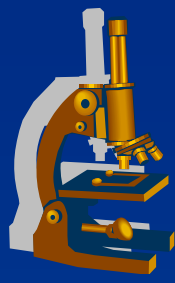
Exudative inflammation



- topography of inflammatory changes:
  - ⇨ ***superficial*** ***(mucous membrane, serous membranes, skin)***
  - ⇨ ***deep*** ***(interstitium)***

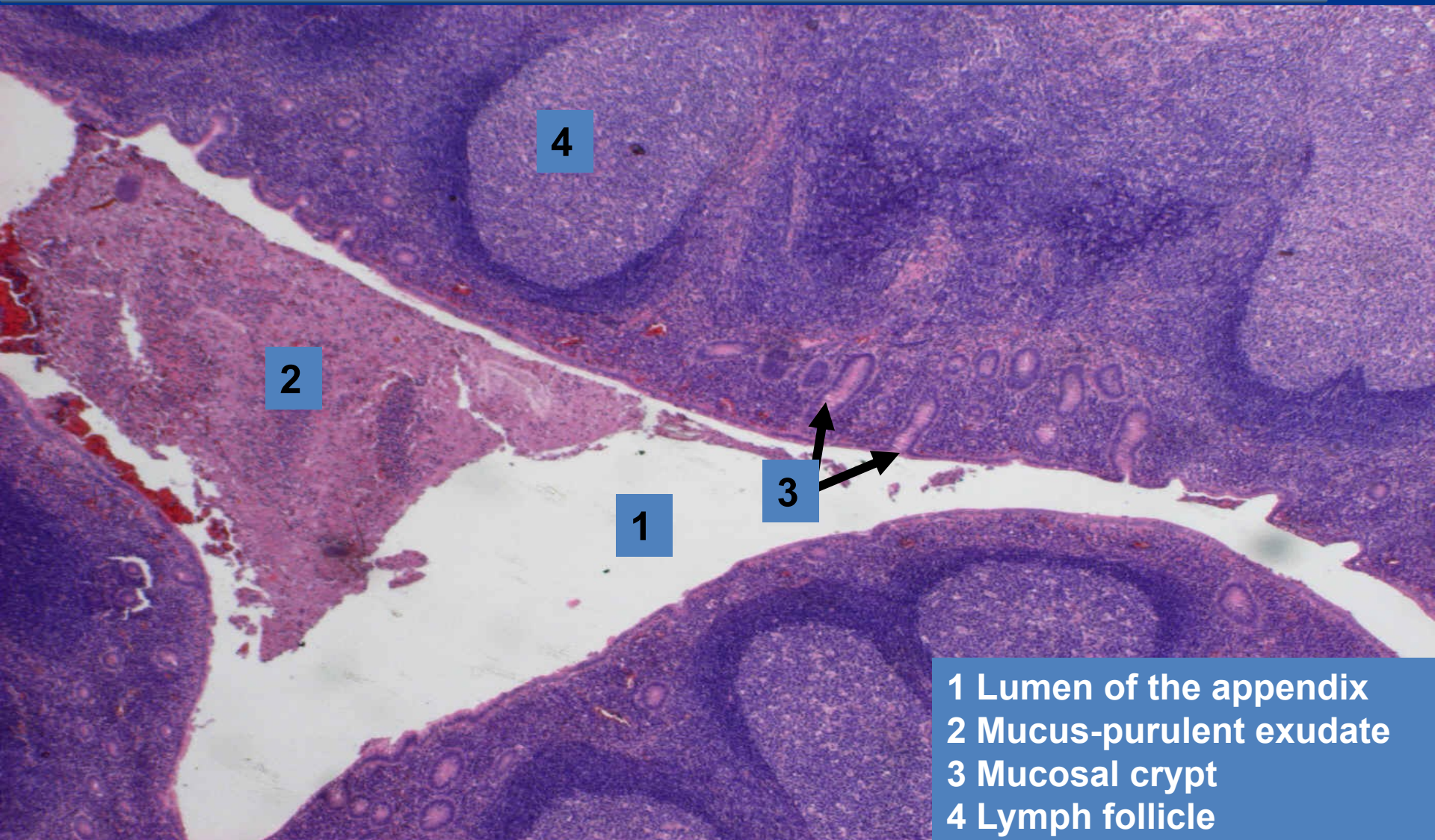
- exudate components:
  - ⇨ ***serous***
  - ⇨ ***fibrinous***
  - ⇨ ***nonpurulent***
  - ⇨ ***purulent***
  - ⇨ ***gangrenous***

Exudative inflammation

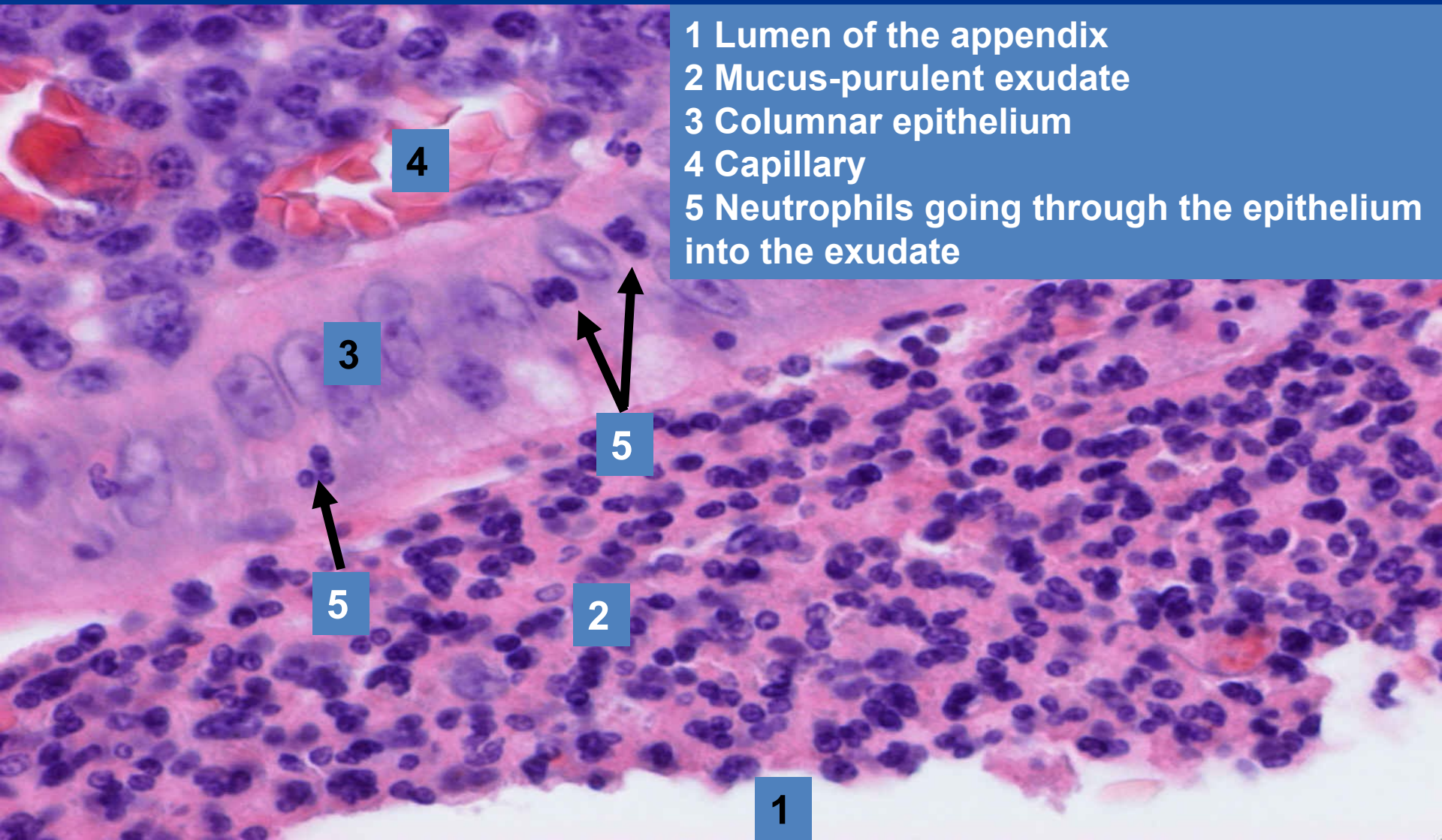


- serous:
  - ⇨ ***watery exudate***
    - few proteins (fibrinogen)
    - in mucous membranes – catarrhal (mucus)
  - ⇨ ***heals by inhibition of exudation***
  - ⇨ ***examples:***
    - superficial: catarrhal appendicitis
    - deep (interstitial): urticaria (hives)

Acute catarrhal appendicitis
(superficial serous inflammation)

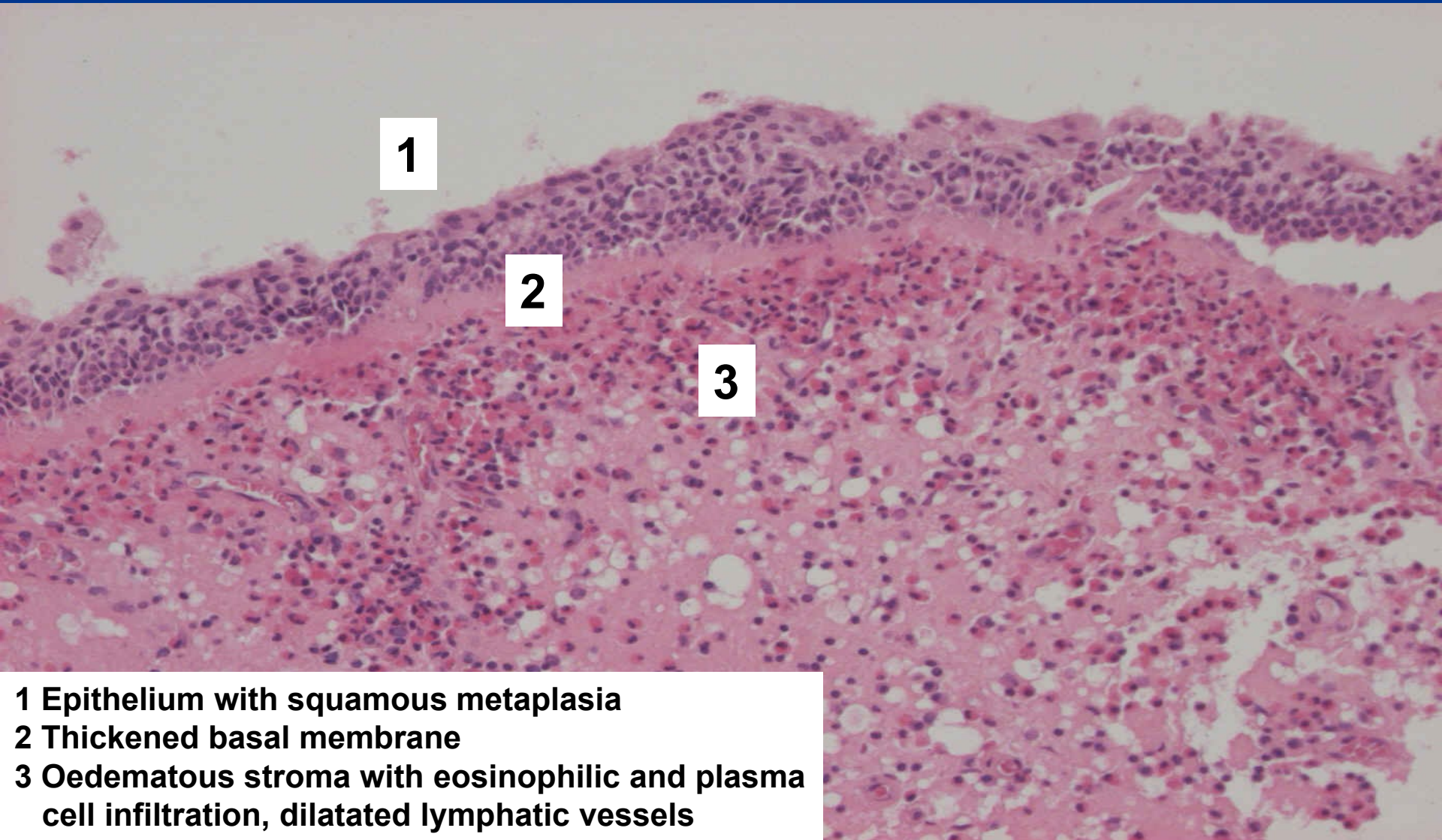


Acute catarrhal appendicitis
(superficial serous inflammation)



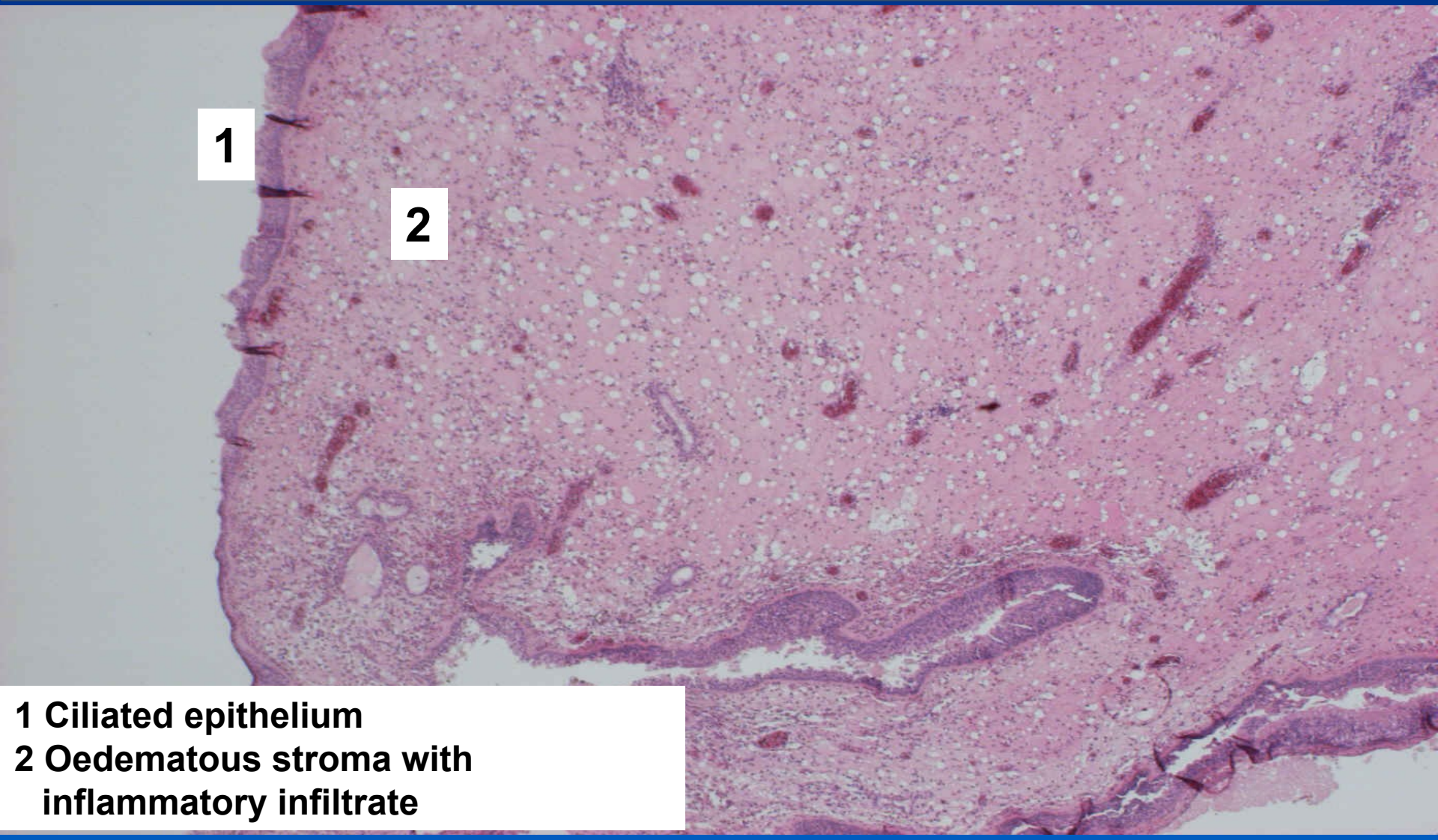
1 Lumen of the appendix
2 Mucus-purulent exudate
3 Columnar epithelium
4 Capillary
5 Neutrophils going through the epithelium into the exudate

Polypous chronic rhinitis
(superficial serous inflammation)



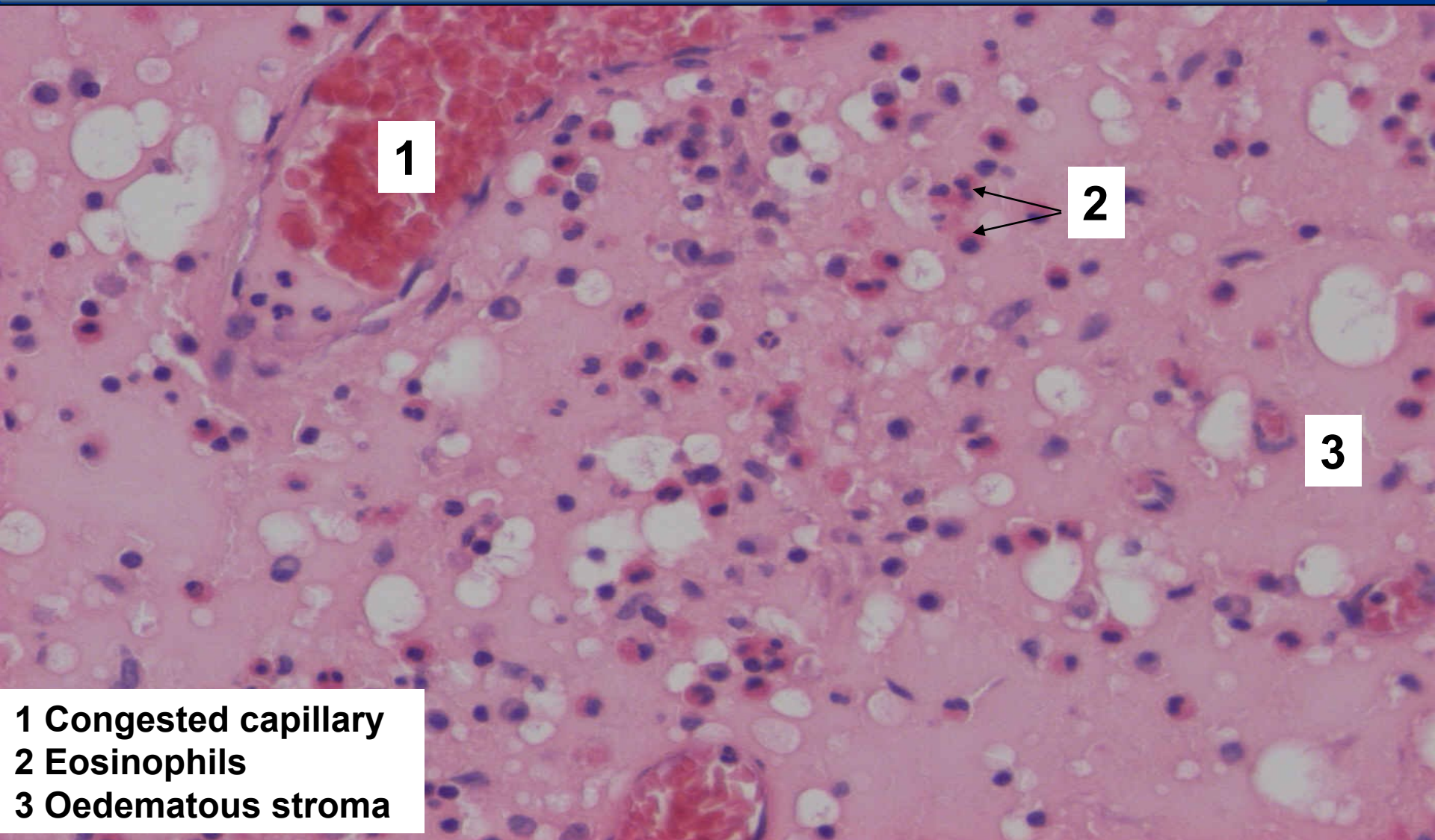
1 Epithelium with squamous metaplasia
2 Thickened basal membrane
3 Oedematous stroma with eosinophilic and plasma cell infiltration, dilatated lymphatic vessels

Polypous chronic rhinitis
(superficial serous inflammation)



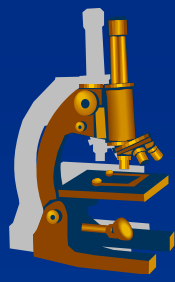
1 Ciliated epithelium
2 Oedematous stroma with inflammatory infiltrate

Polypous chronic rhinitis
(superficial serous inflammation)



1 Congested capillary
2 Eosinophils
3 Oedematous stroma

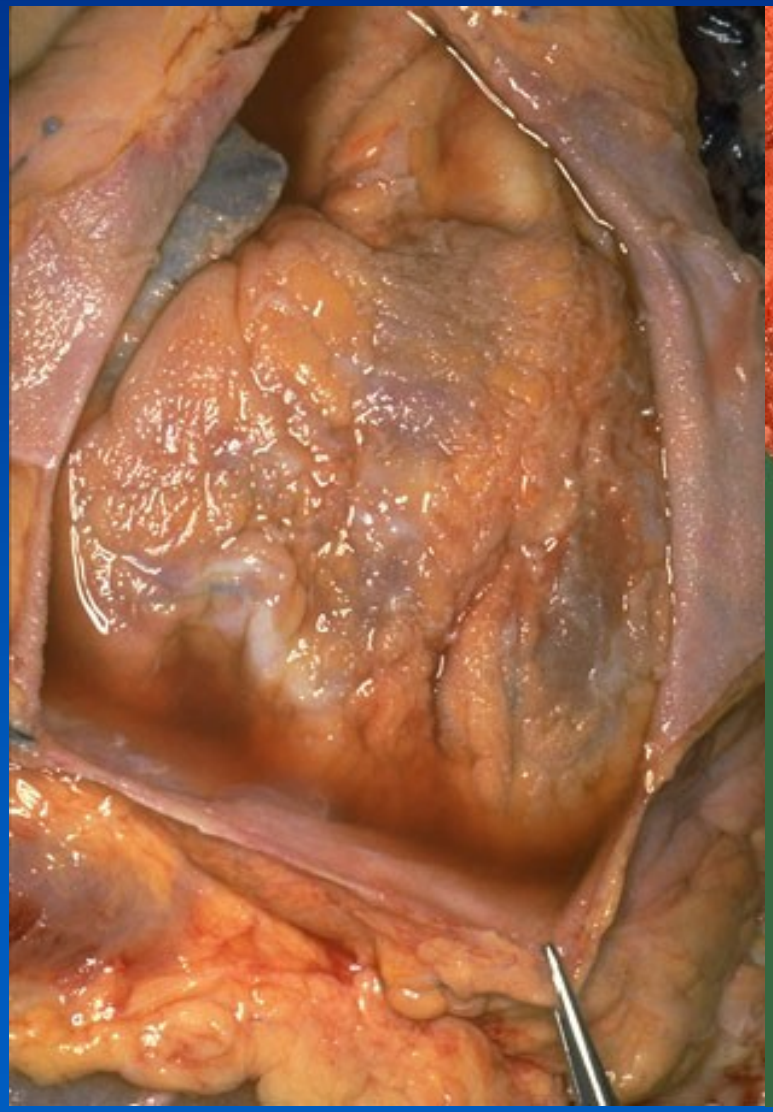
Exudative inflammation

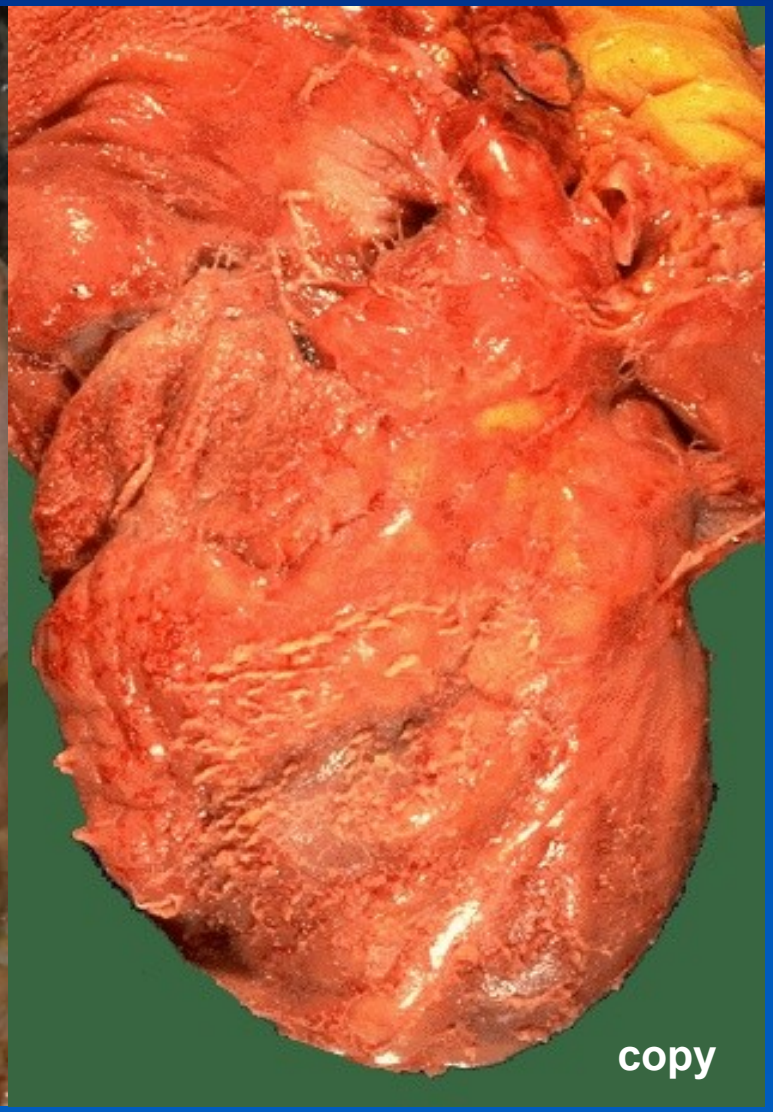


- fibrinous:
  - ⇨ ***content of fibrinogen – fibrin***
    - gross: yellow-greyish ***plaques***
    - micro: eosinophilic fine fibers
  - ⇨ ***healing is more complicated (productive inflammation)***
  - ⇨ ***examples:***
    - superficial inflammation of serous membranes:
      - – **fibrinous pericarditis** (upon uremia) = cor villosum, hirsutum
    - superficial infl. of mucous membranes ***(PSEUDOMEMBRANES)***:
      - – **plaque-like inflammations**
    - deep:
      - – **rheumatic fever**

Fibrinous pericarditis – cor villosum

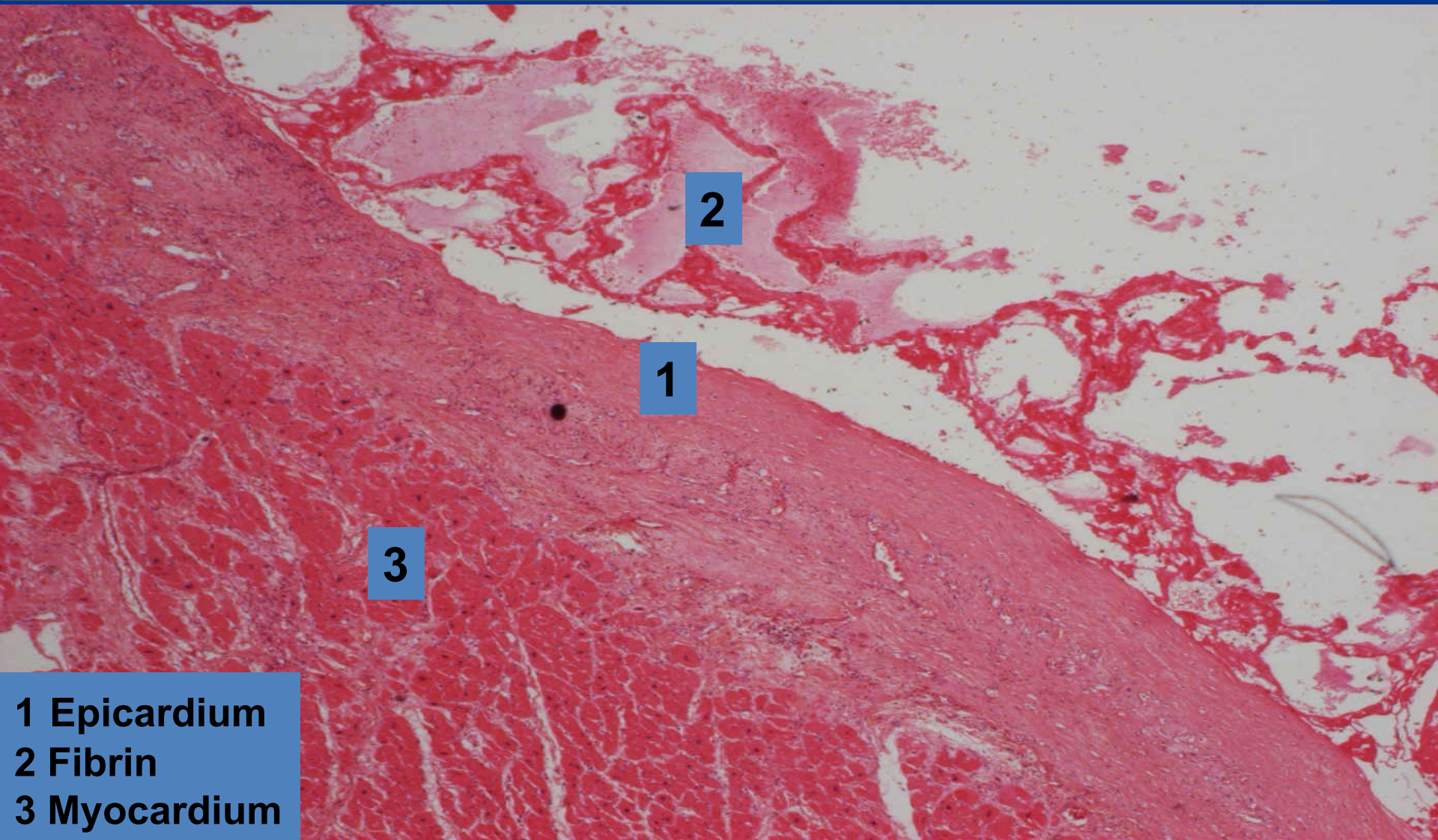
(superficial fibrinous inflammation of serous membranes)





Fibrinous pericarditis

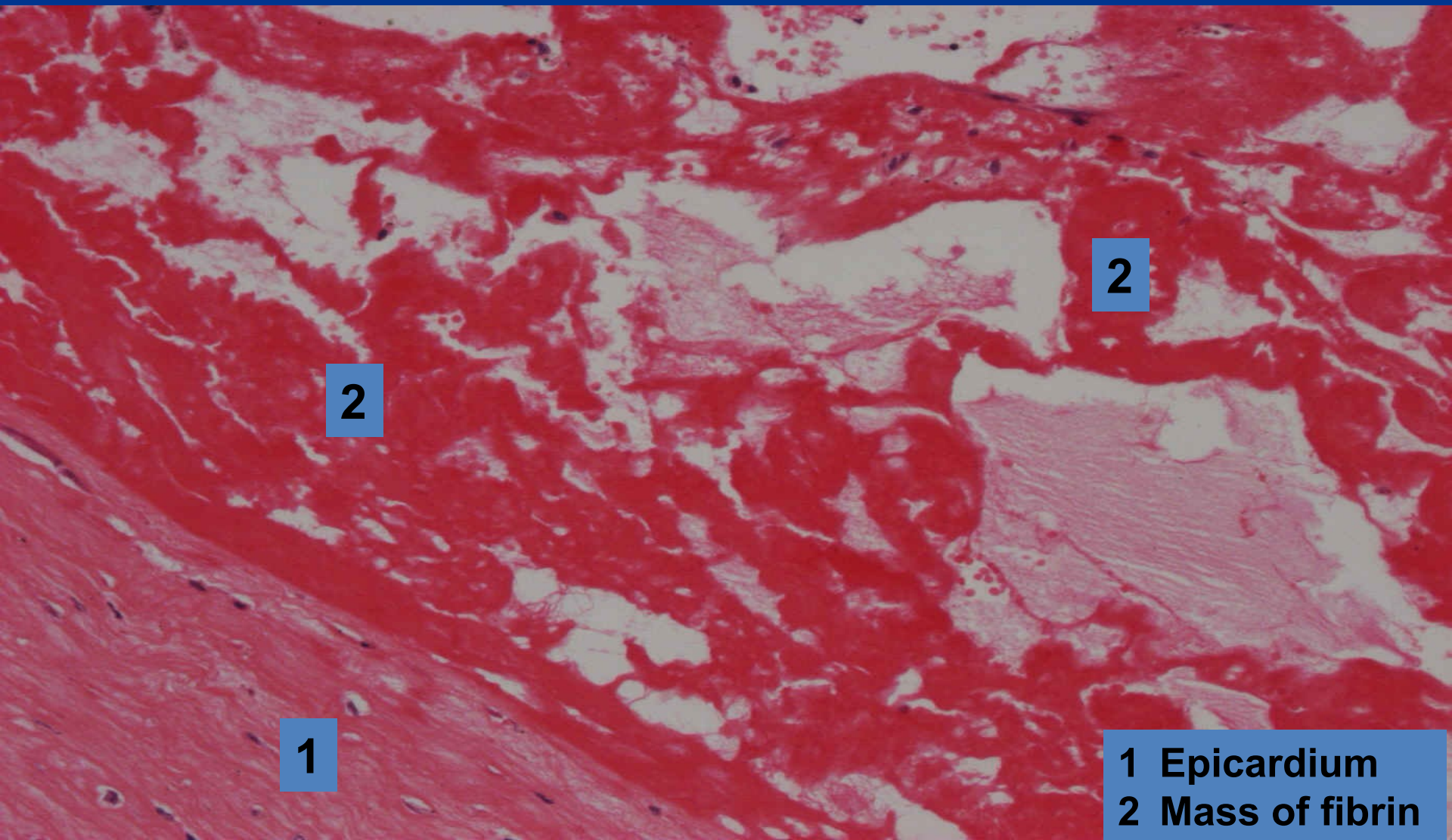
(superficial fibrinous inflammation of serous membranes)



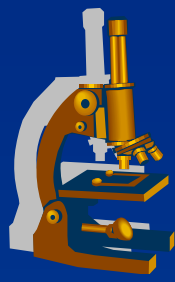
1 Epicardium
2 Fibrin
3 Myocardium

Fibrinous pericarditis

(superficial fibrinous inflammation of serous membranes)



Exudative fibrinous mucosal inflammation



Classification due to mucosal damage:

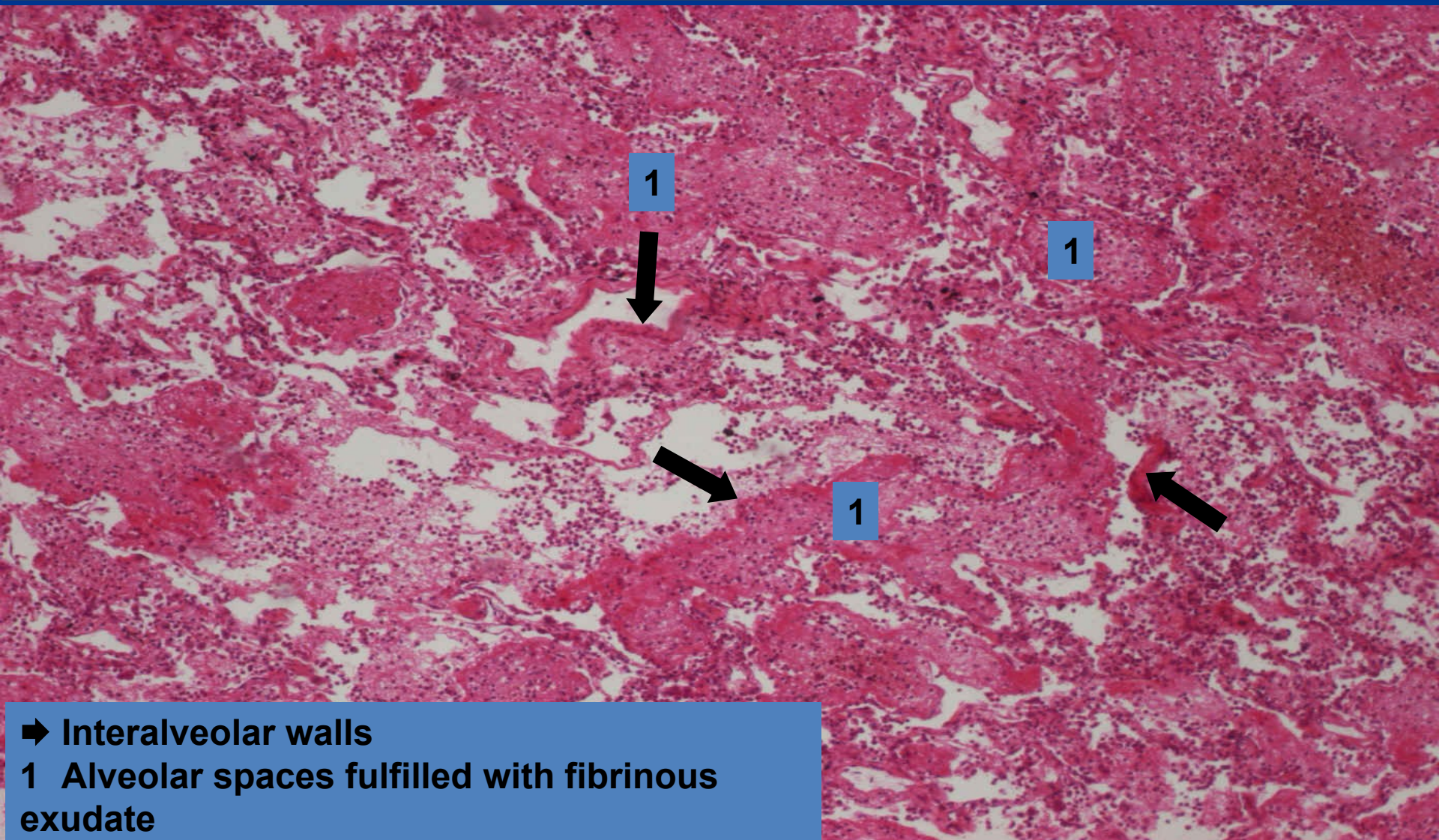
- croupous
  - ⇨***little alteration, plaque is loose on the surface***

    (croupous pneumonia)
- diphteric
  - ⇨***deeper mucosal necrosis, after the pseudomembrane is peeled off » ulcus***

    (pseudomembranous colitis)
- escharotic
  - ⇨***extensive deep necrosis***

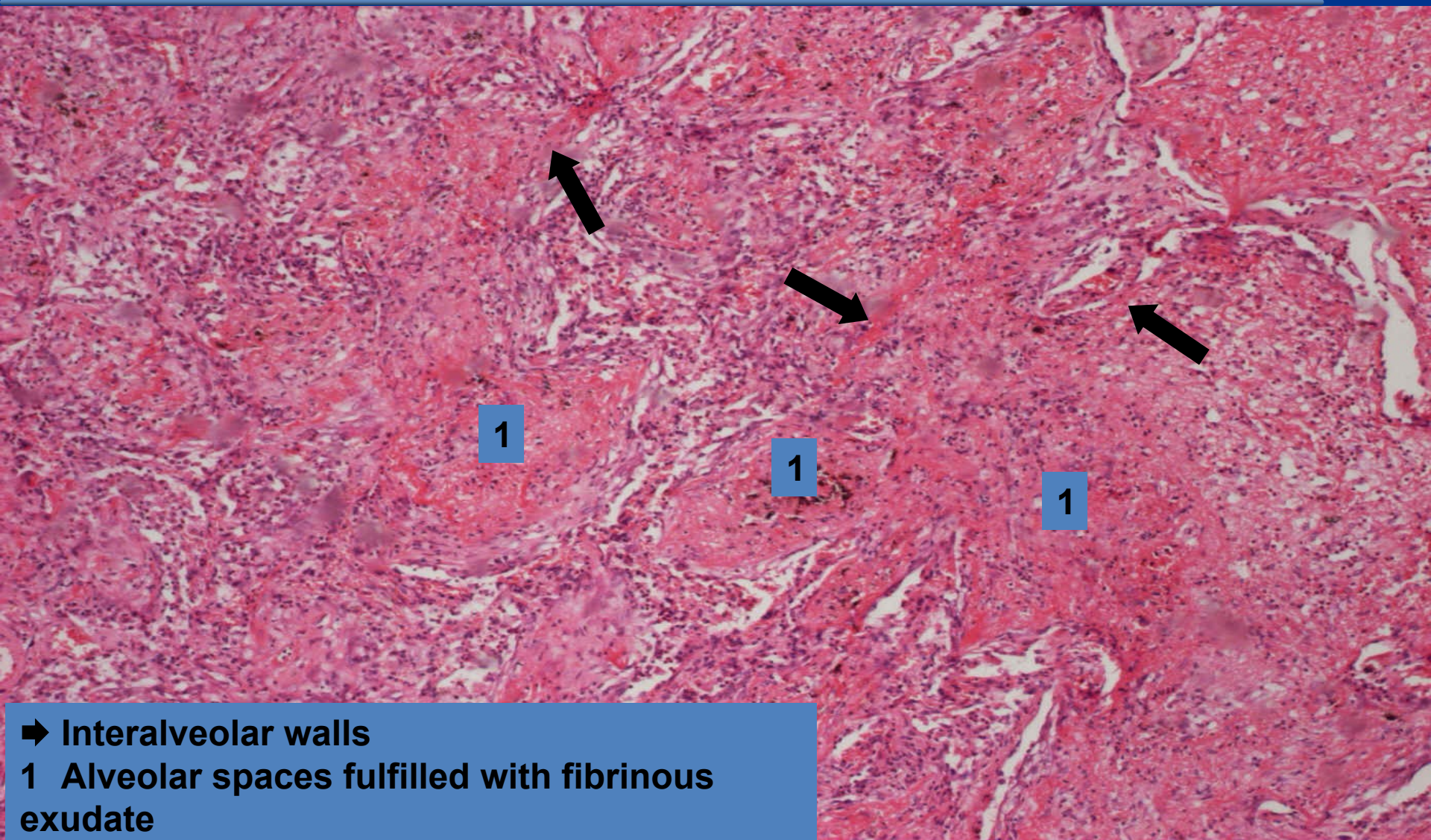
    (necrotising tracheitis in flu)

Lobar pneumonia
(superficial fibrinous mucosal inflammation)



➡ Interalveolar walls
1 Alveolar spaces fulfilled with fibrinous exudate

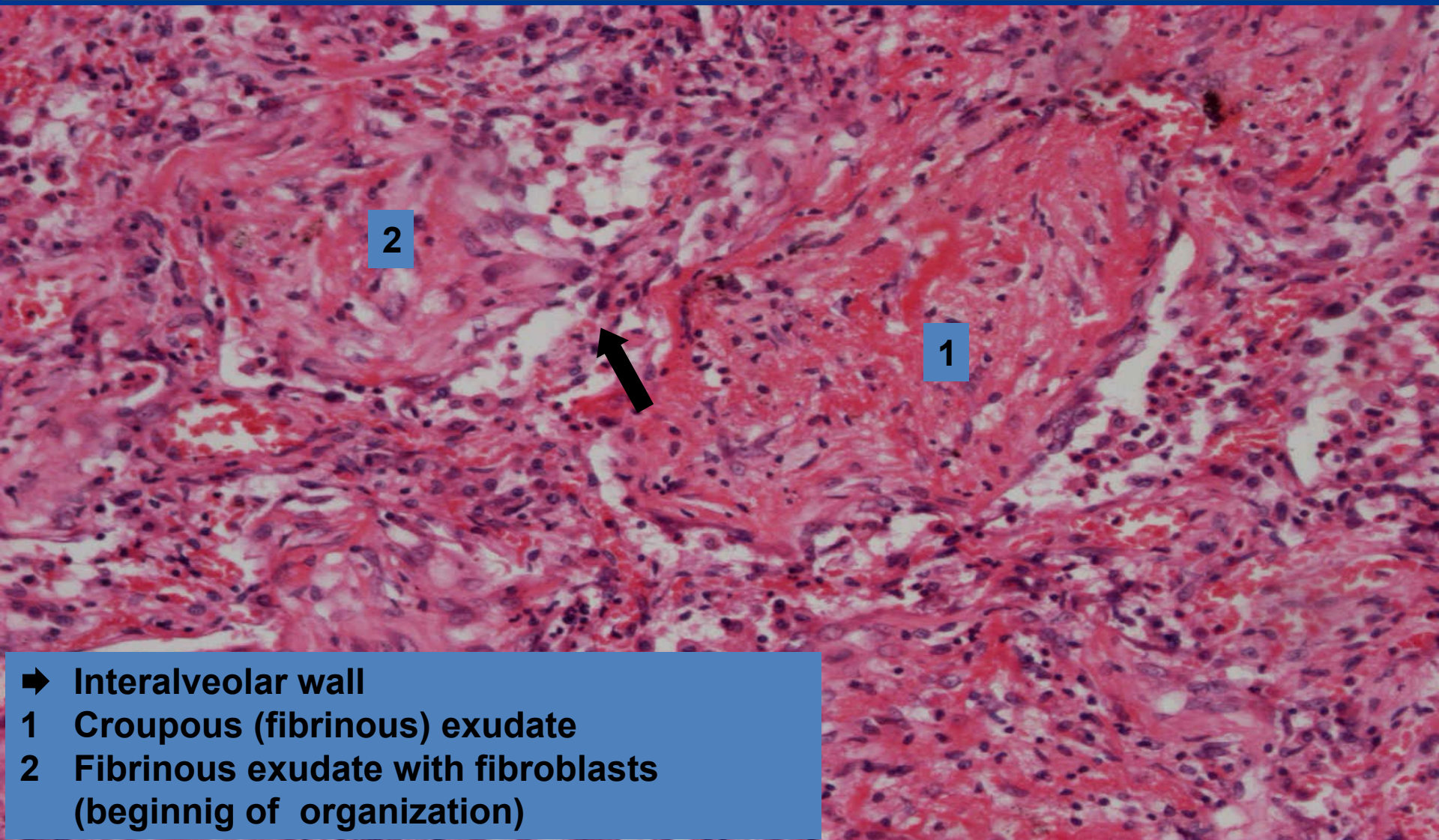
Lobar pneumonia
(superficial fibrinous mucosal inflammation)



➡ Interalveolar walls
1 Alveolar spaces fulfilled with fibrinous exudate

Lobar pneumonia - detail

(superficial fibrinous mucosal inflammation)

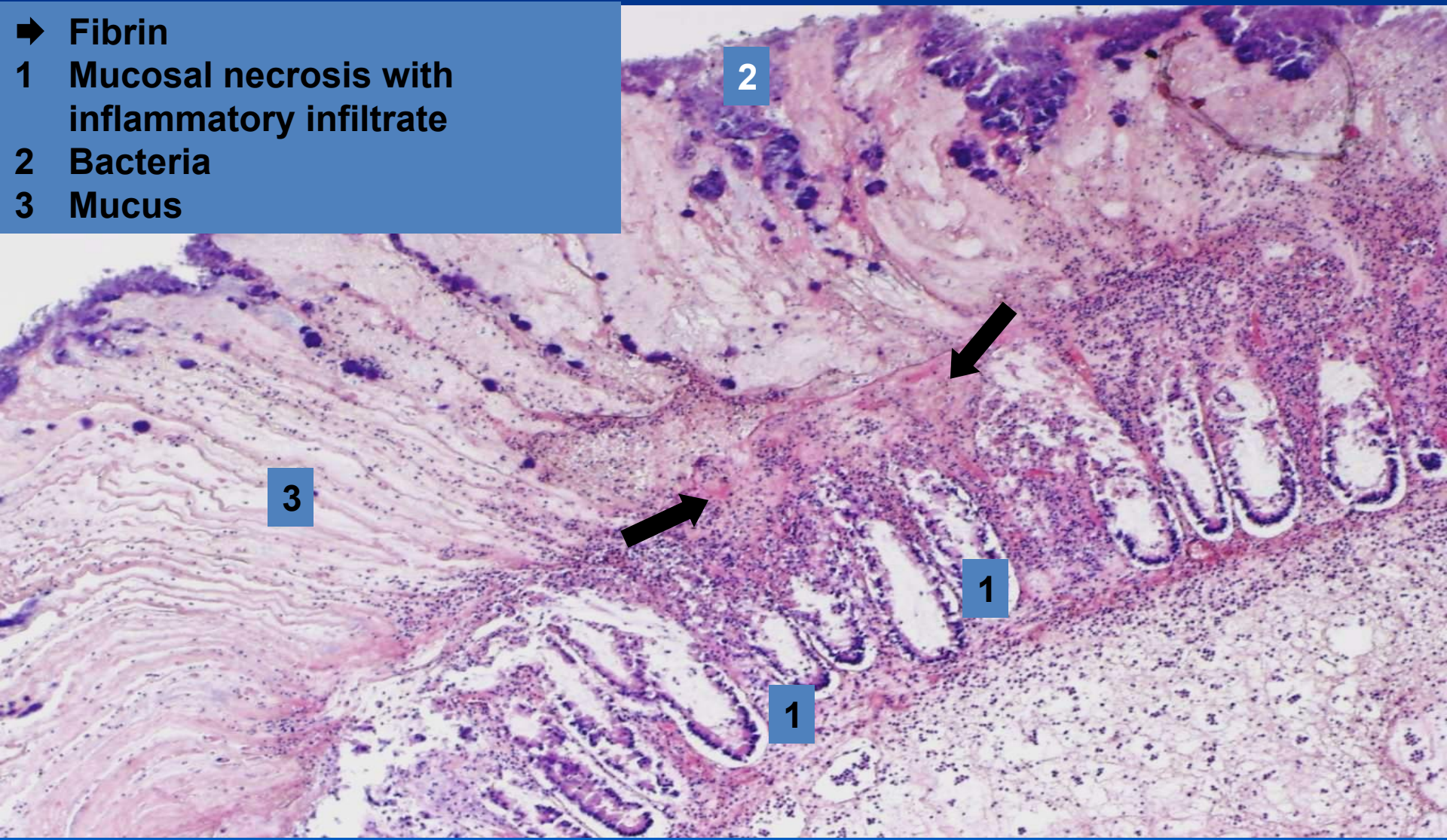


➡ Interalveolar wall
1 Croupous (fibrinous) exudate
2 Fibrinous exudate with fibroblasts (beginnig of organization)

Pseudomembranous colitis

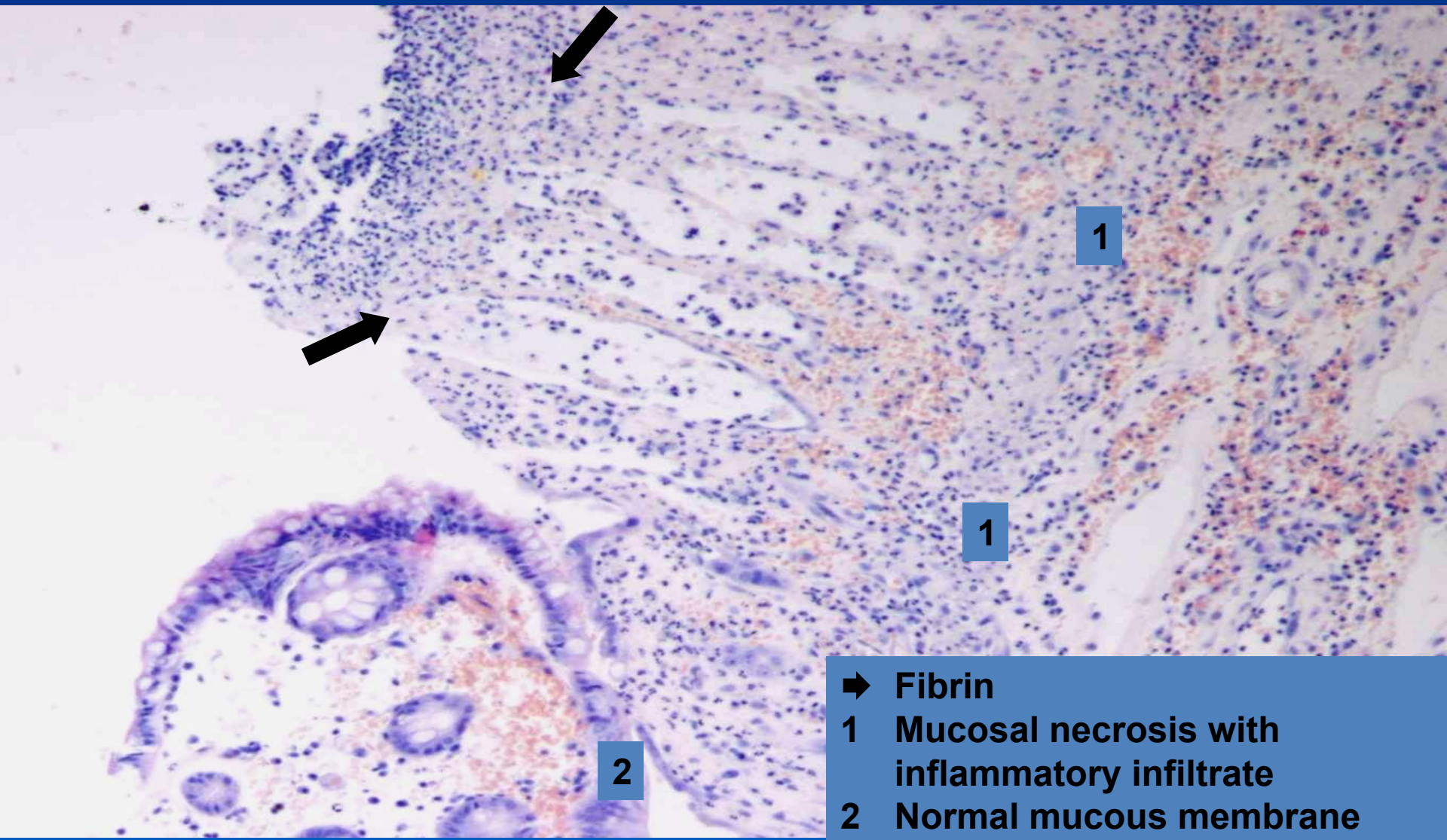
(superficial fibrinous mucosal inflammation)

➡ Fibrin
1 Mucosal necrosis with inflammatory infiltrate
2 Bacteria
3 Mucus



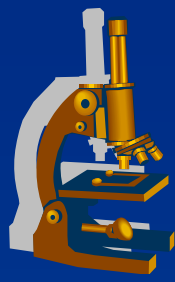
Pseudomembranous colitis

(superficial fibrinous mucosal inflammation)



➡ Fibrin
1 Mucosal necrosis with inflammatory infiltrate
2 Normal mucous membrane

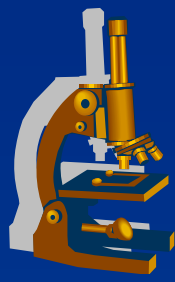
Rheumatic fever



- Relapsing inflammatory disease esp. in children (1-5 weeks after streptococcal A infection)
- forming of antibodies cross-reactive with antigens in the heart, joints…
- dg. based on anamnesis and 2 of 5 main (Jones) criteria:

  erythema marginatum, chorea, carditis, subcutaneous nodules, migratory polyarthritis of the large joints

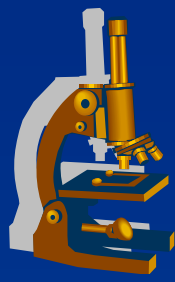
Rheumatic fever



- GROSS:
  - ***vegetations*** *- (friable, bulky, wart-like outgrowths) on the edges of the heart valves (mainly aortic and mitral)*
  - *after years leaflet thickening and commissural fusion*
  - *thickening, shortening and fusion of the chordae tendinae*

- MICRO:
  - ***Aschoff bodies*** *– inflammatory lesion within the heart*
  - *fibrinoid necrosis of fibrous tissue*
  - *central zone of degenerating with surrounding chronic* ***inflammatory infiltrate*** *(lymphocytes, plasma cells, histiocytes, activated macrophages called Antischkow cells)*

Exudative inflammation

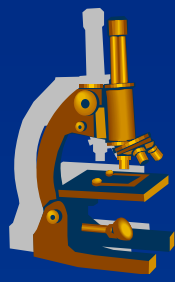


- purulent:
  - ⇨ ***PRODUCTION OF PUS:***

    neutrophil-rich exudate
  - ⇨ ***GROSS:***

    superficial pus, pus accumulation (abscess)
  - ⇨ ***heals by inhibition of exudation and/or by proliferative inflammation***
  - ⇨ ***examples:***
    - **superficial inflammation of meninges:**
      - purulent meningitis
    - **superficial mucosal inflammation:**
      - catarrhal-purulent bronchopneumonia
    - **deep (interstitial):**
      - phlegmona (e.g. phlegmonous appendicitis)
      - abscess

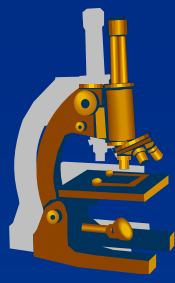
Purulent leptomeningitis



Aetiology:

- *Escherichia coli* and *β streptococci*
  - ⇨ ***in newborns***
- *Haemophilus influenzae*
  - ⇨ ***in infants and children***
- *Neisseria meningitis*
  - ⇨ ***in adolescents and young adults***
- *Streptococcus pneumoniae* and *Listeria monocytogenes*
  - ⇨ ***in old people***

Purulent leptomeningitis

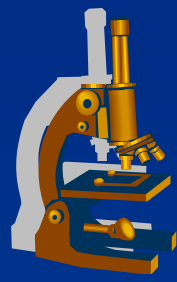


SYMPTOMS:

- meningeal irritation:
  - ⇨ ***headache, photophobia, irritation, nuchal rigidity, consciousness failures***

- lumbar puncture:
  - ⇨ ***opaque or purulent liquor with higher CSF pressure***

Purulent leptomeningitis MORPHOLOGY



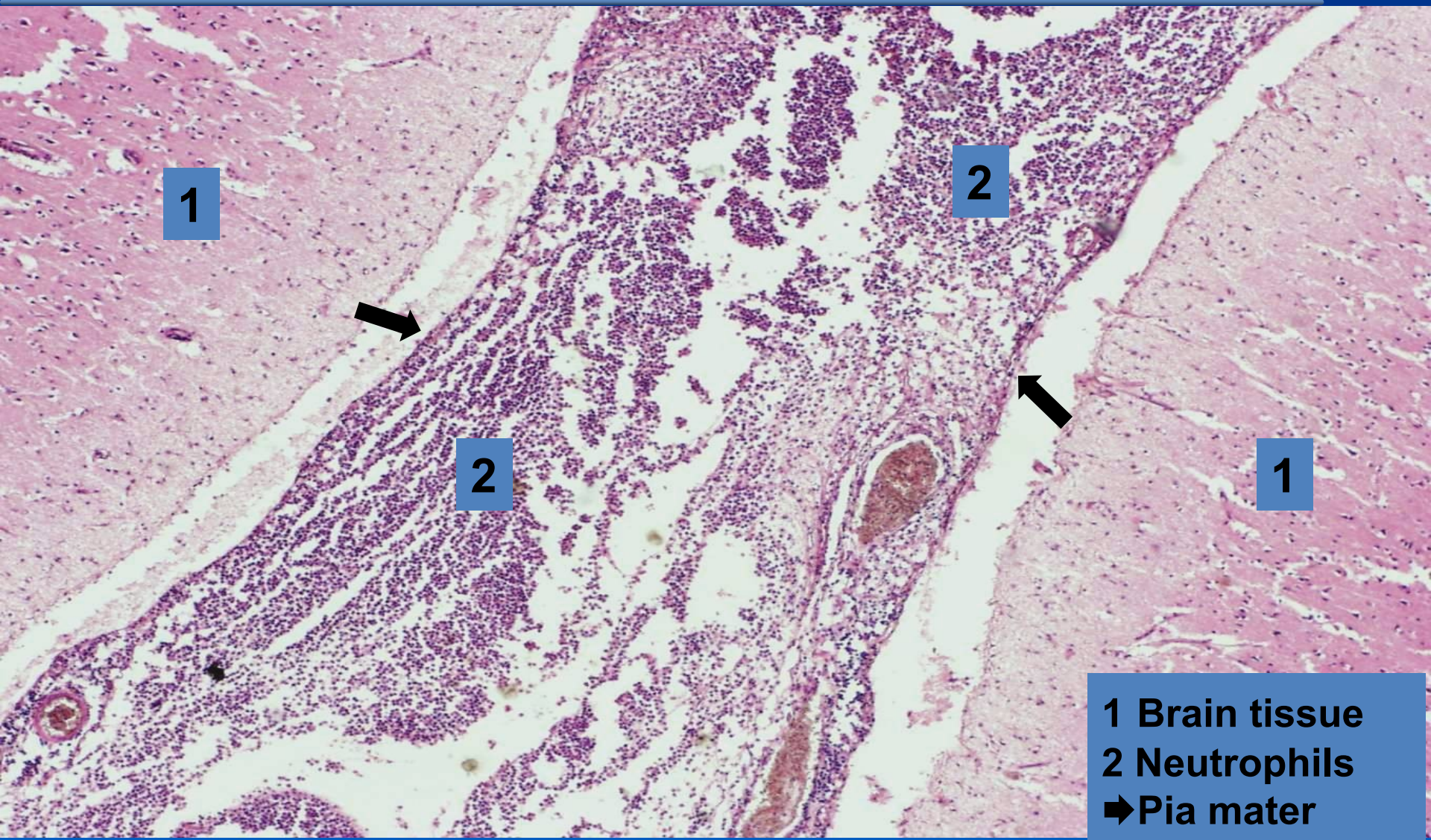
- GROSS:
  - ⇨ *congested and leaked pia mater with pus*
  - ⇨ *purulent-opaque liquor*
- MICRO:
  - ⇨ *meninges infiltrated with neutrophils*
  - ⇨ *vessels congested with blood, thrombosis » hemorrhagic brain infarction*
- COMPLICATIONS:

  possible fibrosis of meninges resulting in hydrocephalus; epilepsy

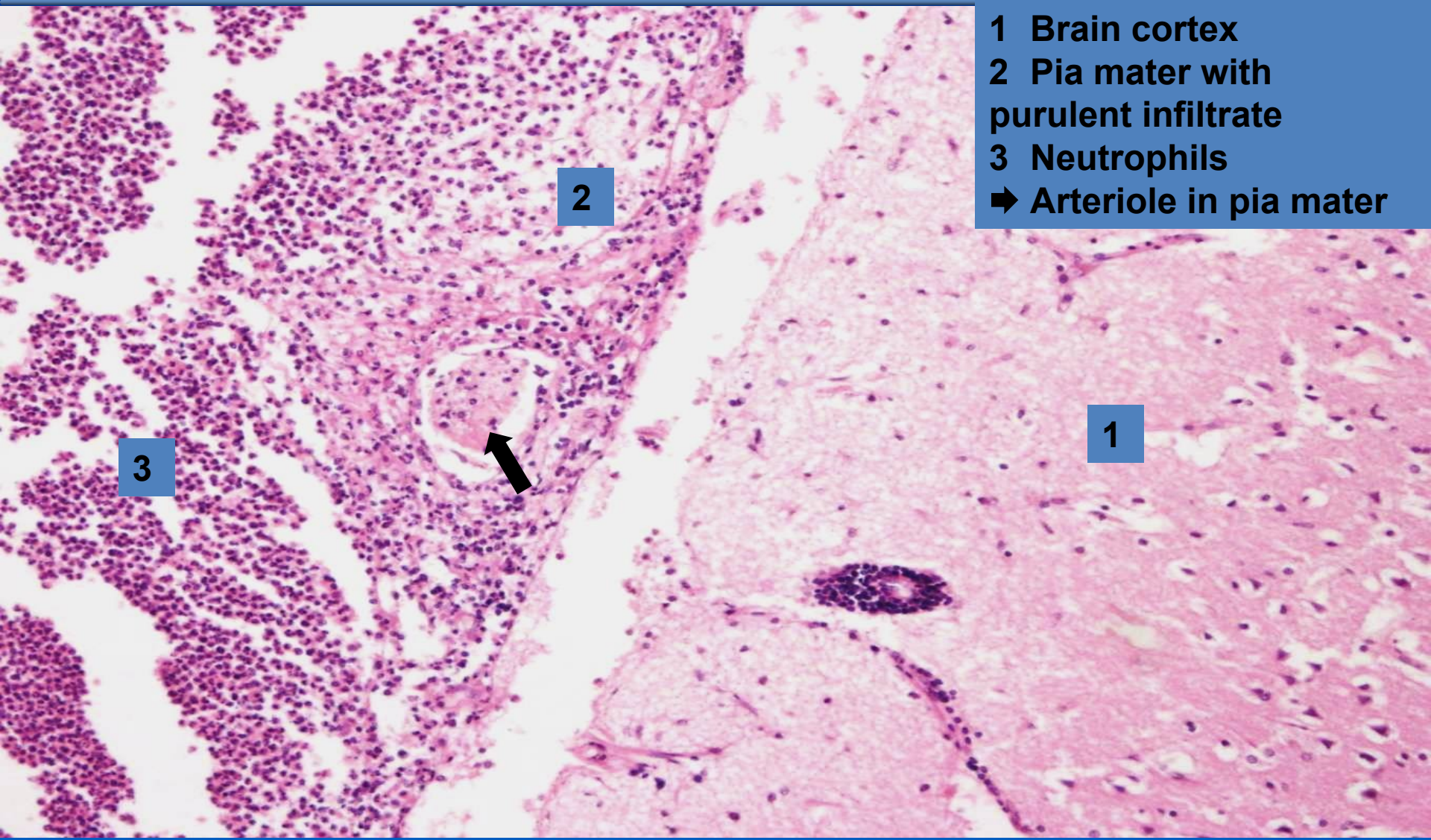
Purulent leptomeningitis
(superficial purulent inflammation)

Purulent leptomeningitis

(superficial purulent inflammation)

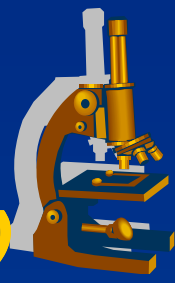


Purulent leptomeningitis
(superficial purulent inflammation - detail)



1 Brain cortex
2 Pia mater with purulent infiltrate
3 Neutrophils
➡ Arteriole in pia mater

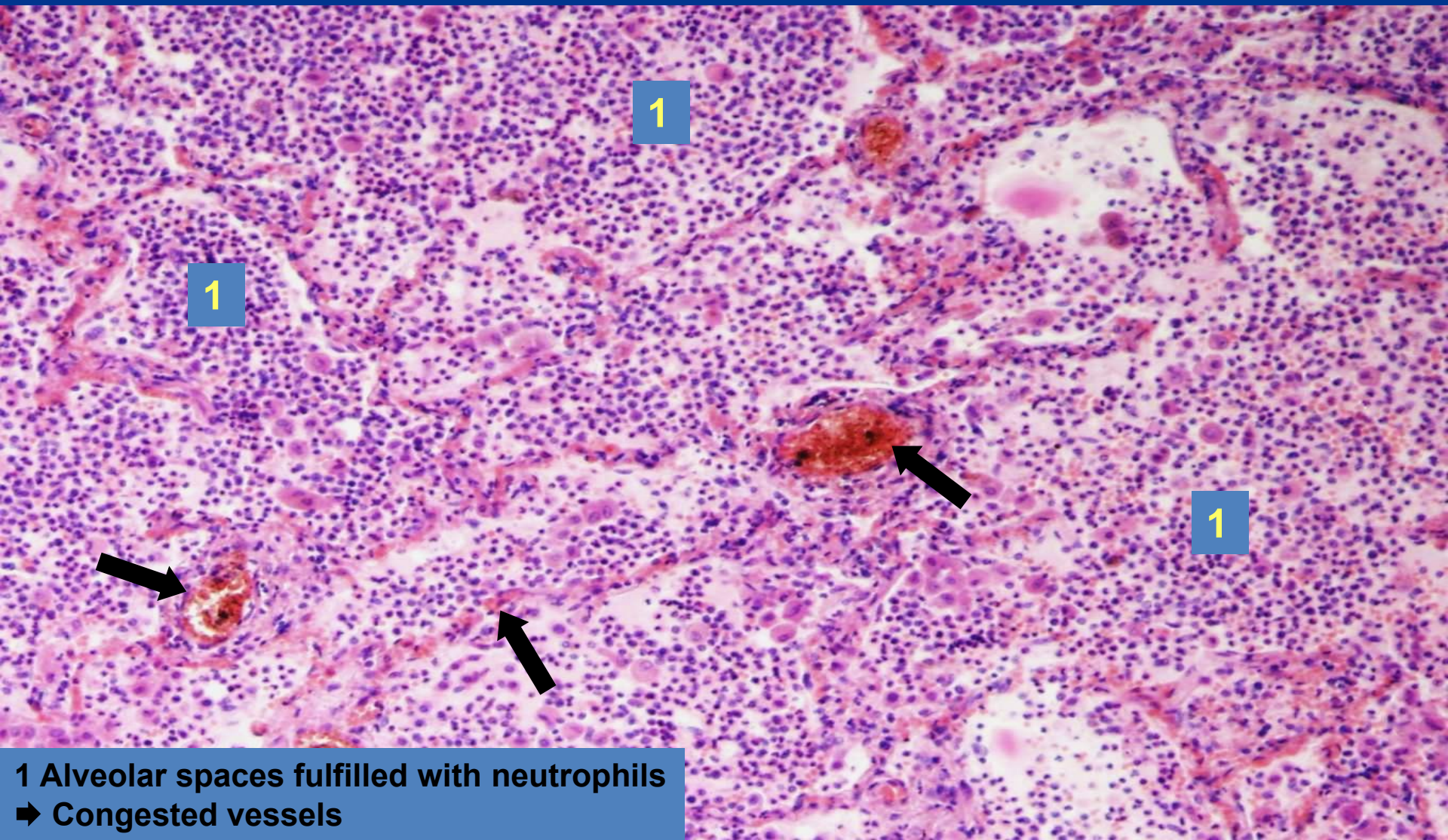
Bronchopneumonia
(Superficial purulent mucosal inflammation)



- GROSS:
  - ⇨ ***various stages of inflammation in the lung at the same time***
- MICRO:
  - ⇨ ***alveolar spaces fulfilled with neutrophils***
  - ⇨ ***small amount of fibrin in the exudate compared to lobar pneumonia***

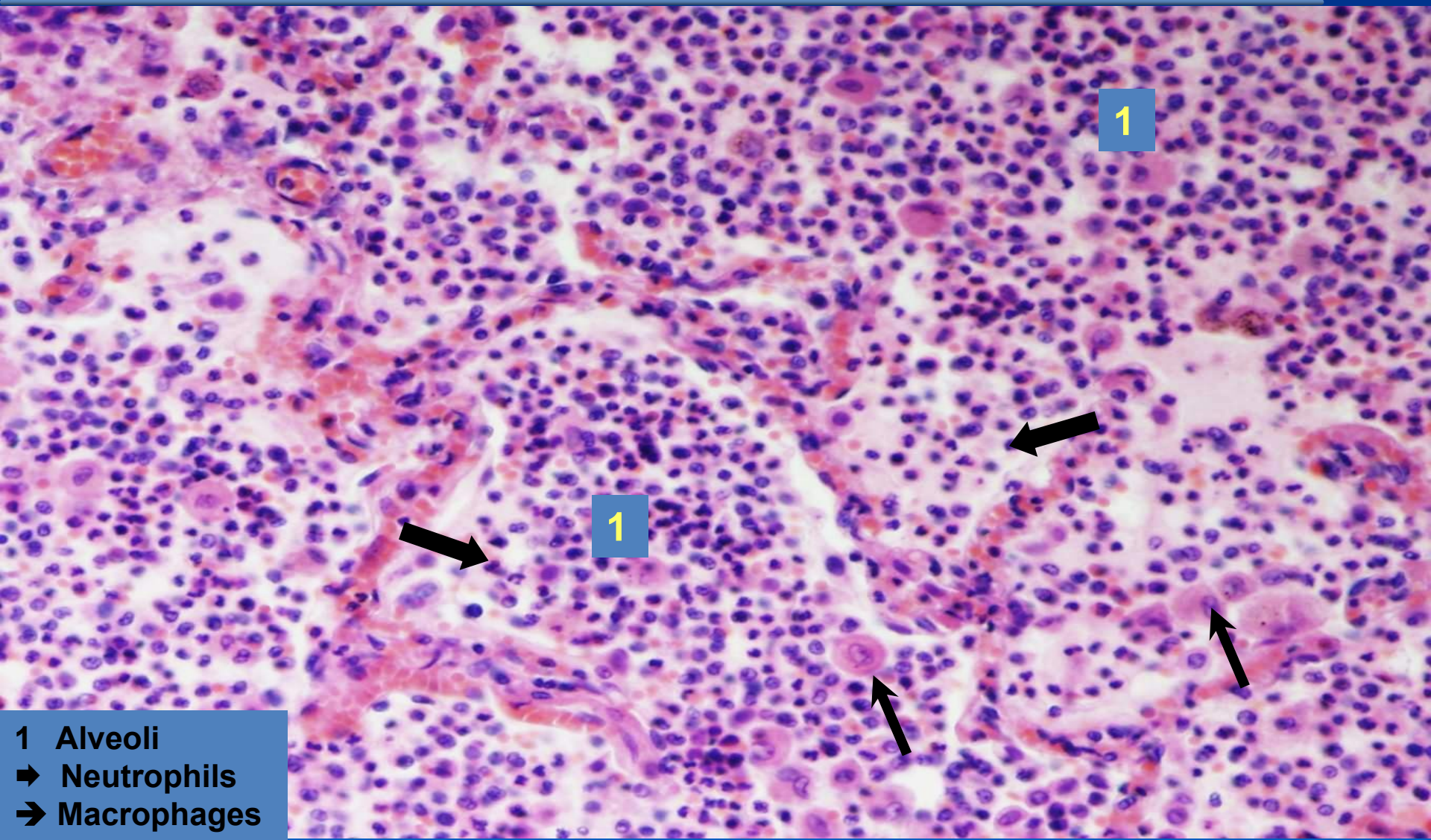
Bronchopneumonia

Purulent bronchopneumonia
(superficial purulent mucosal inflammation)



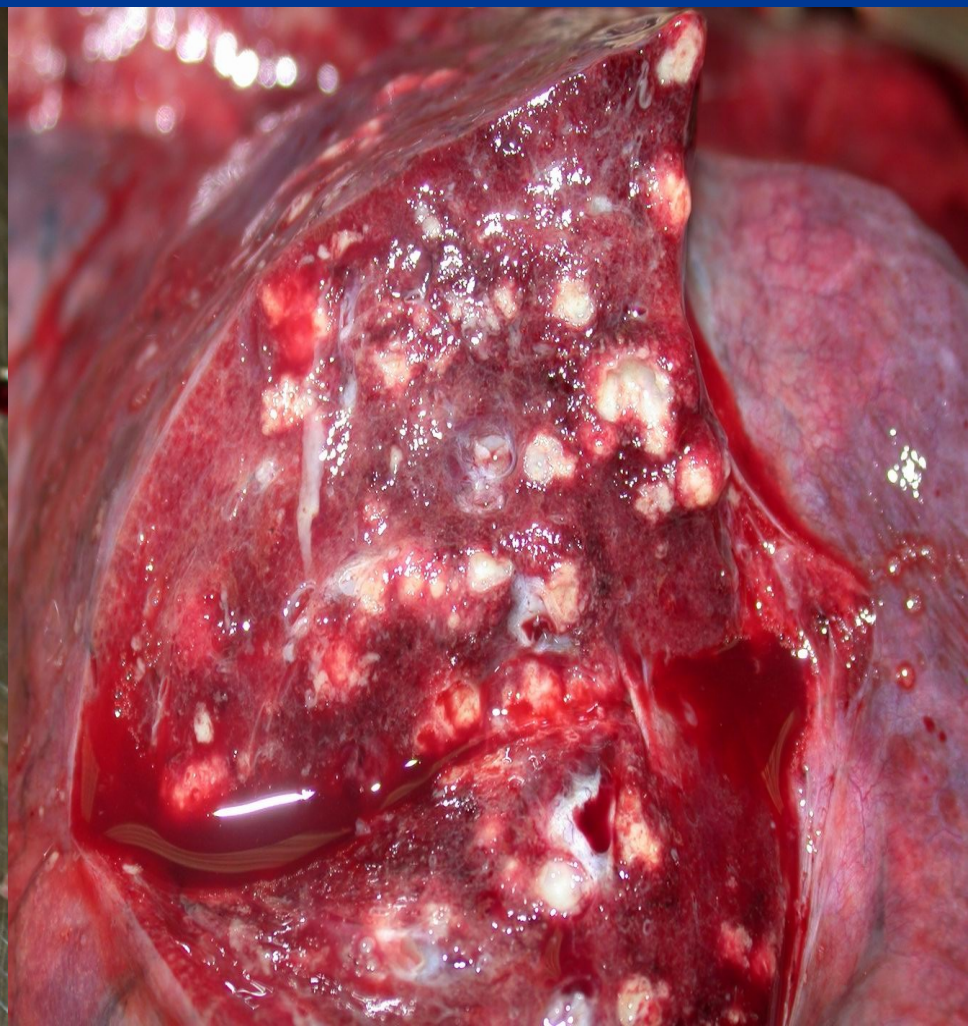
1 Alveolar spaces fulfilled with neutrophils
➡ Congested vessels

Purulent bronchopneumonia - detail
(superficial purulent mucosal inflammation)

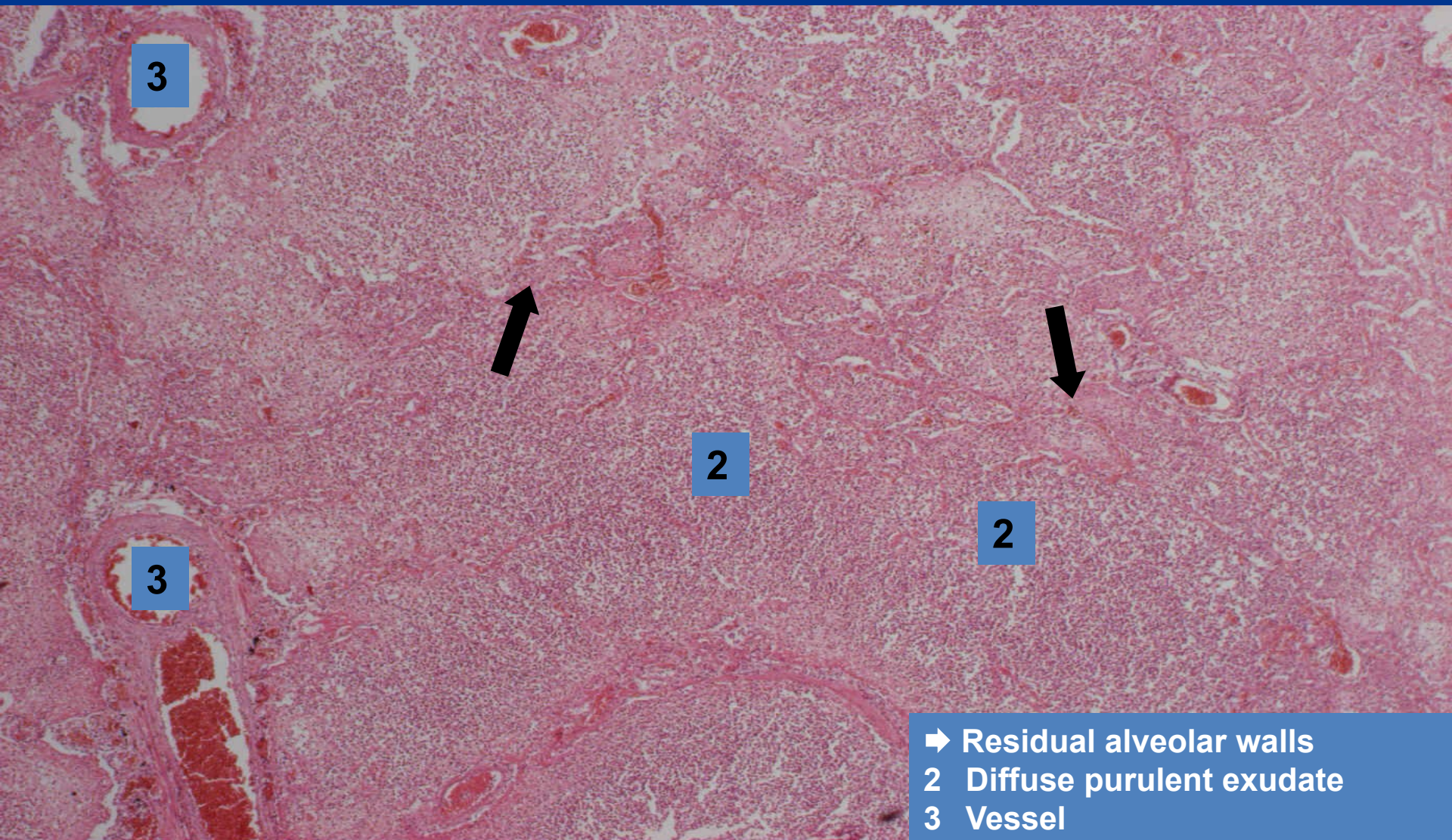


1 Alveoli
➡ Neutrophils
➔ Macrophages

Abscessing bronchopneumonia
(abscessing purulent inflammation)

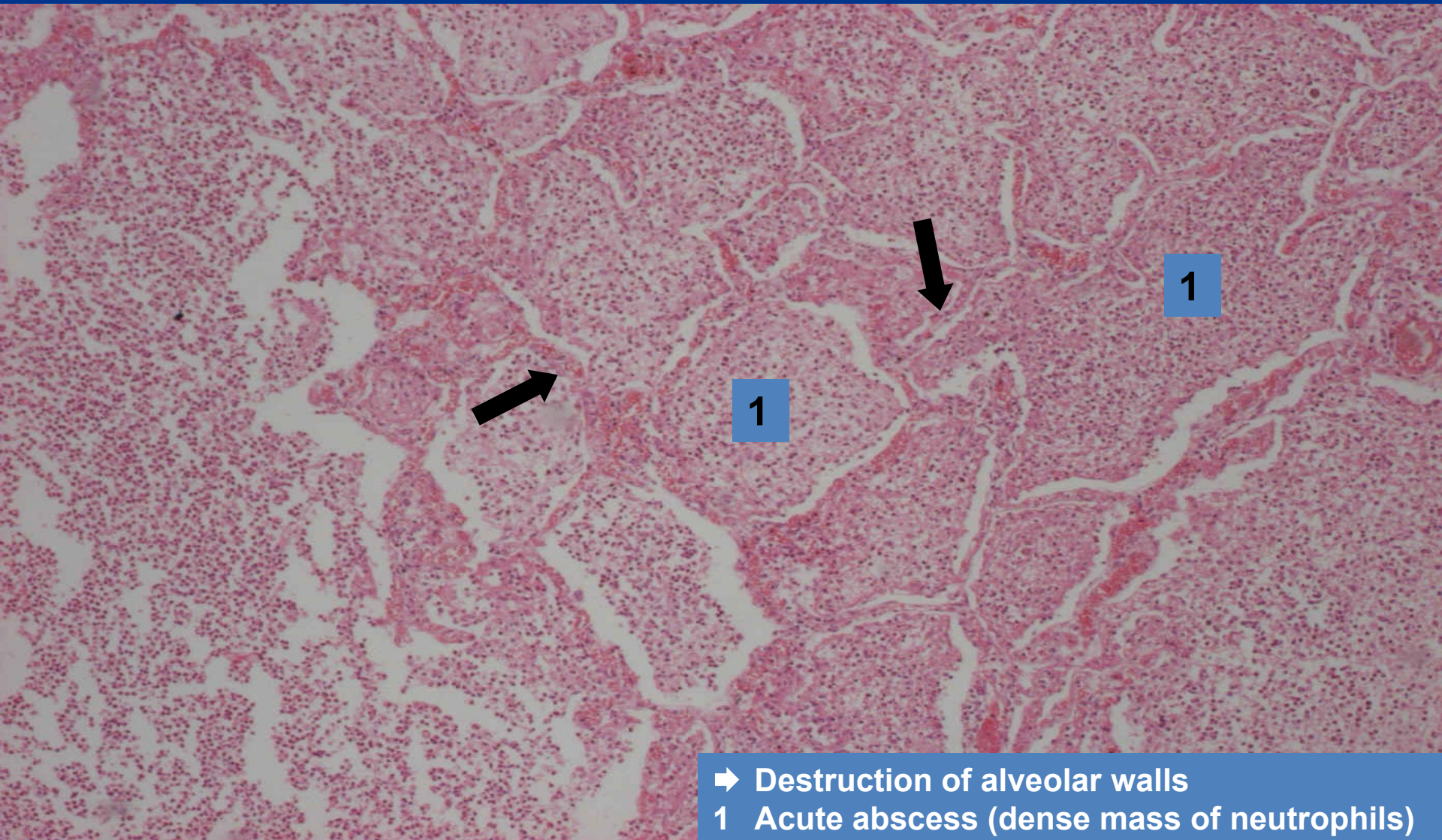


Abscessing bronchopneumonia
(abscessing purulent inflammation)



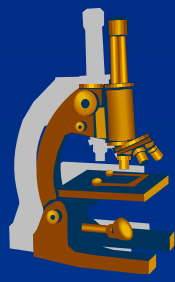
➡ Residual alveolar walls
2 Diffuse purulent exudate
3 Vessel

Abscessing bronchopneumonia
(abscessing purulent inflammation)



➡ Destruction of alveolar walls
1 Acute abscess (dense mass of neutrophils)

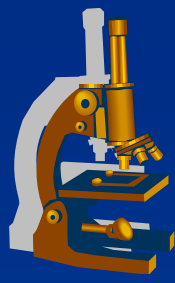
Phlegmonous appendicitis
(interstitial purulent inflammation)



- most common cause of „acute abdomen“, surgical intervention necessary.

- CLINICALLY:
  - ⇨ ***in any age, commonly children – young adults.***
  - ⇨ ***right mesogastric or hypogastric pain; nausea, vomiting, rectal raised temperature, sensitive abdomen, leukocytosis.***

Phlegmonous appendicitis



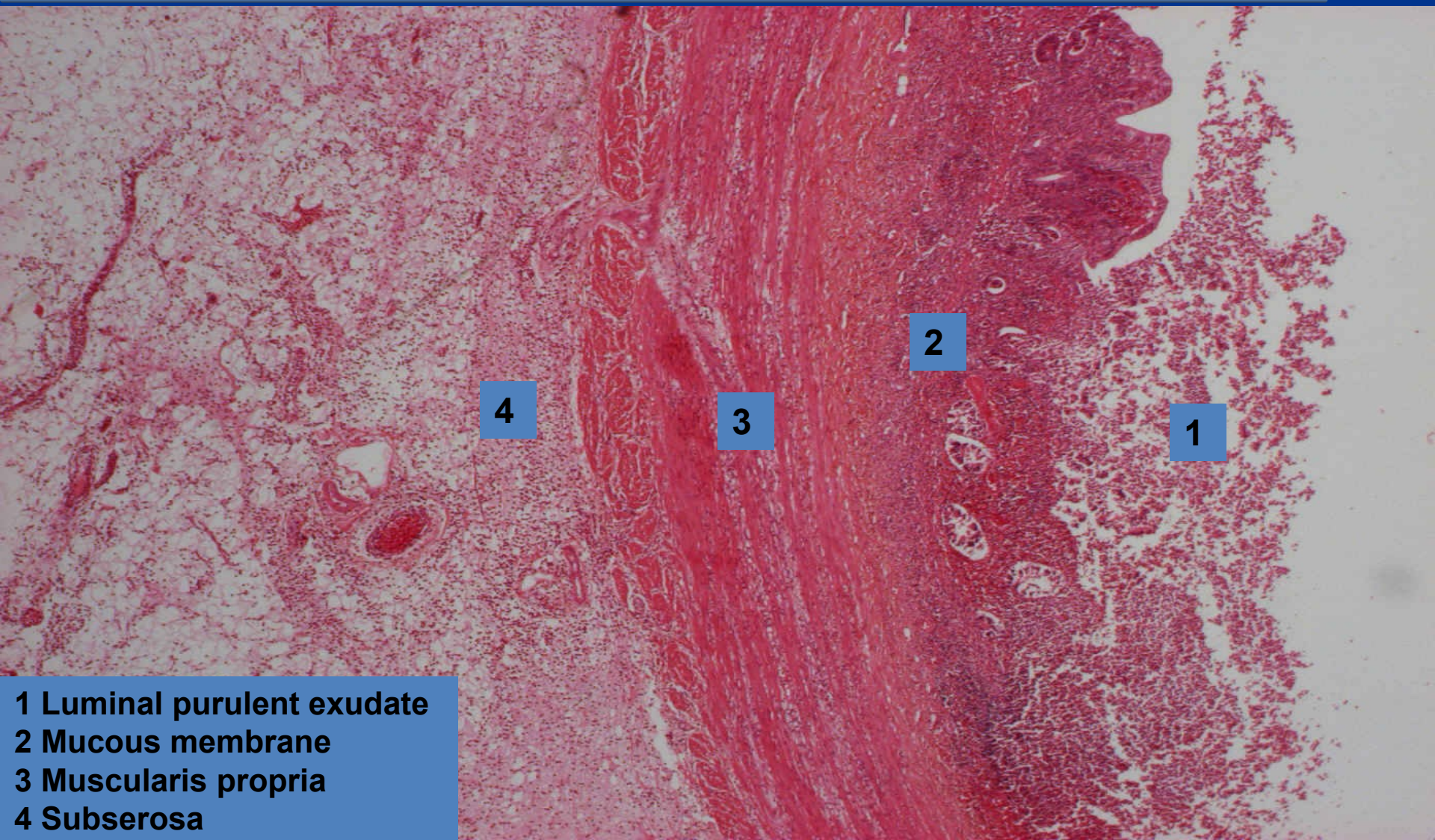
- MORPHOLOGY:
  - ⇨ ***GROSS:***
    - hyperemic serosa, hemorrhage, pus.
  - ⇨ ***MICRO:***
    - diffuse interstitial neutrophilic infiltration (phlegmona), purulent periappendicitis.

- COMPLICATIONS:
  - ⇨ ***perforation, purulent peritonitis, pyelophlebitis, portal thrombosis, pyemia, liver abscessi, sepsis.***

Appendix - normal

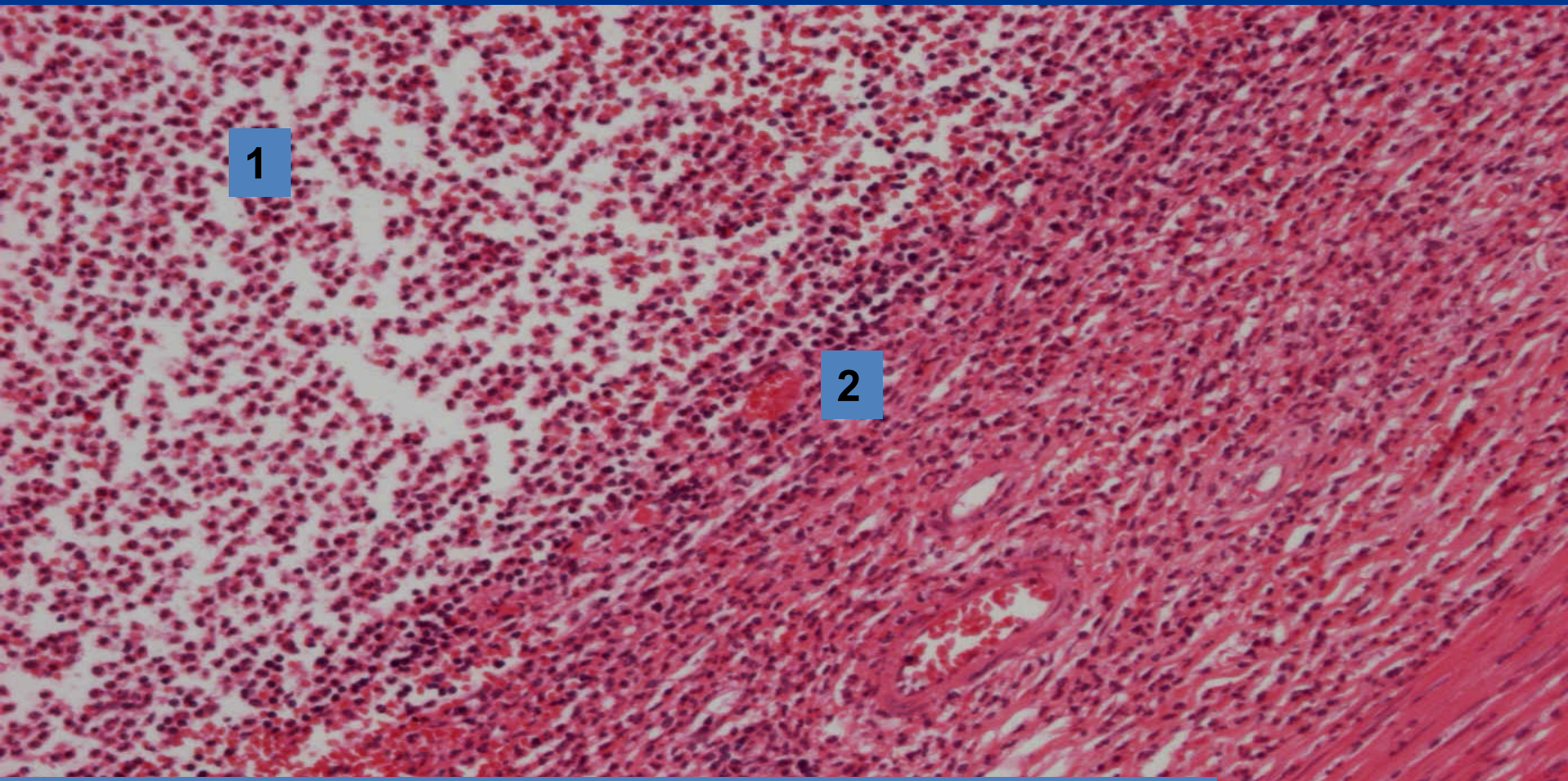
Phlegmonous appendicitis
(interstitial purulent inflammation)

Phlegmonous appendicitis
(interstitial purulent inflammation)



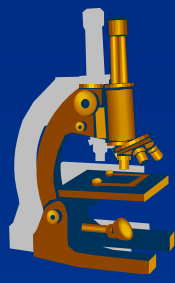
1 Luminal purulent exudate
2 Mucous membrane
3 Muscularis propria
4 Subserosa

Phlegmonous appendicitis
(interstitial purulent inflammation)



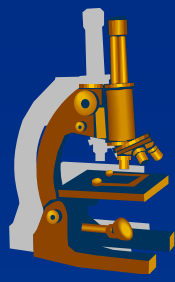
1 Luminal purulent exudate
2 Mucosa with diffuse neutrophilic infiltration – absence of epithelium
3 Muscularis propria

Acute pyelonephritis



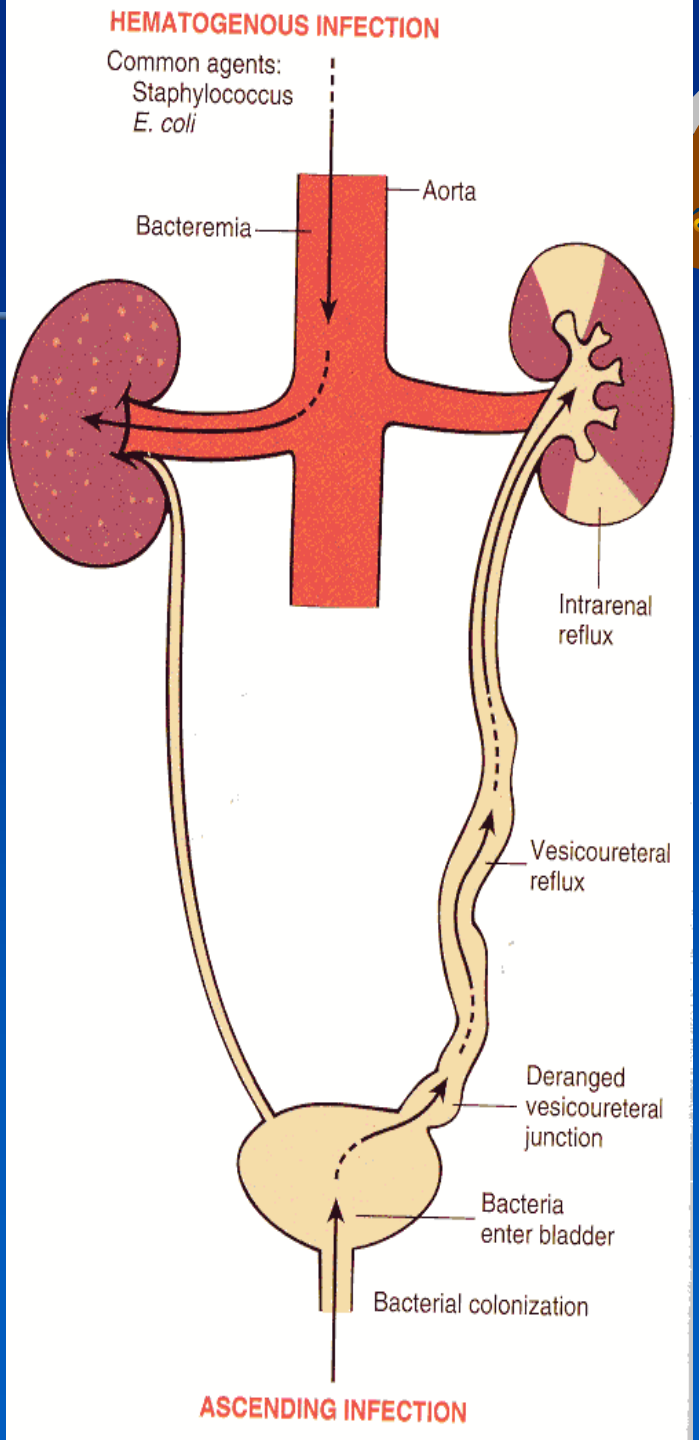
- Common purulent renal inflammation, bacterial infection by *Escherichia coli, Proteus, Klebsiella, Enterobacter*
- **Ascending** infection by urine reflux in urinary tract inflammation
- **Descending (haematogenous)** infection in septicaemia, rare

Acute pyelonephritis

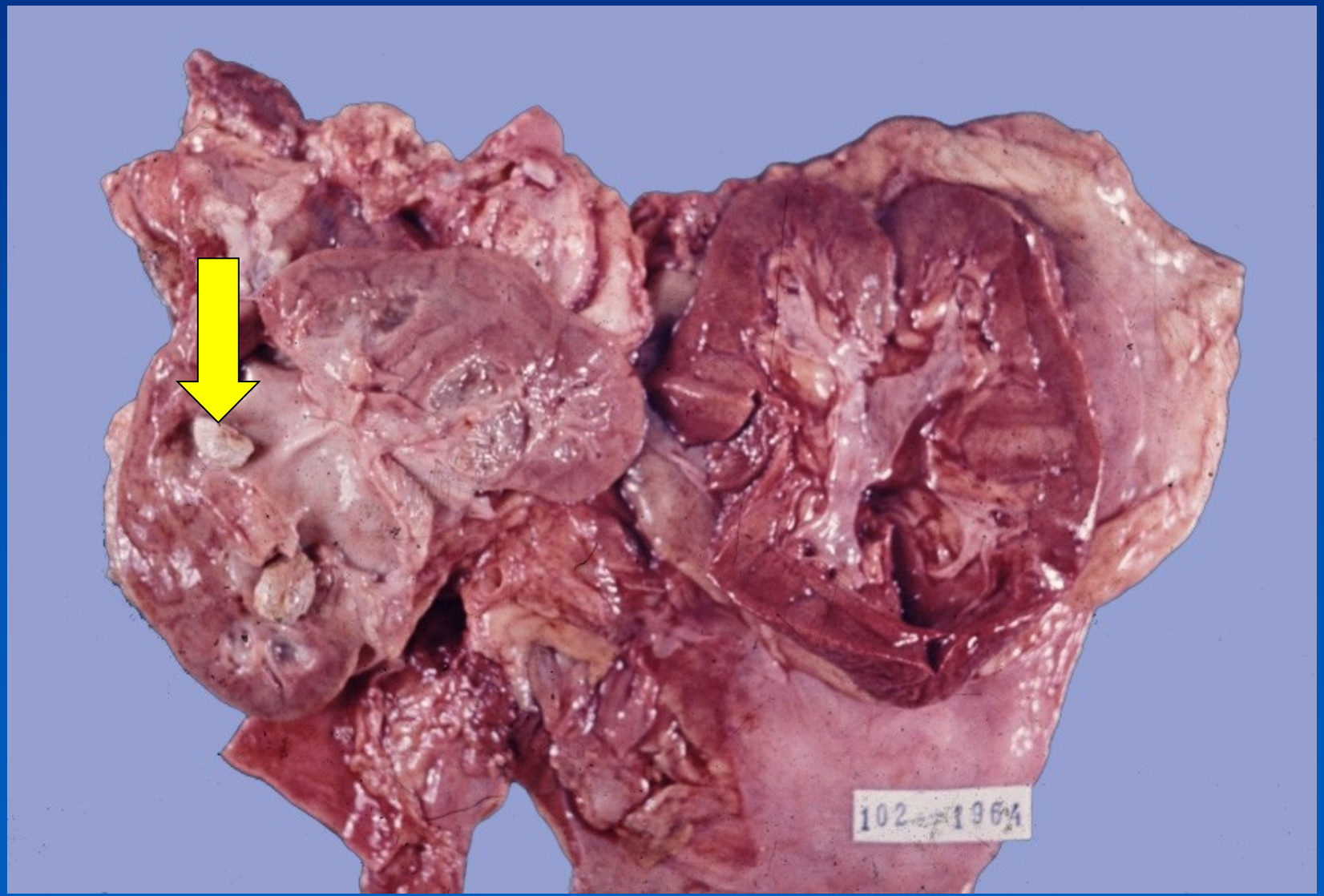


- Facilitated by DM, gout, all causes of obstructive uropathy (e.g. nephrolithiasis, tumors, urinary tract anomalies incl. vesicoureteric and intrarenal reflux)
- Instrumental interventions (cathetrization, cystoscopy)
- GROSS:
  - ⇨ ***enlarged kidney, cortical and medullary abscessses***
- MICRO:
  - ⇨ ***purulent neutrophilic exudate in tubules and interstitium, oedema***

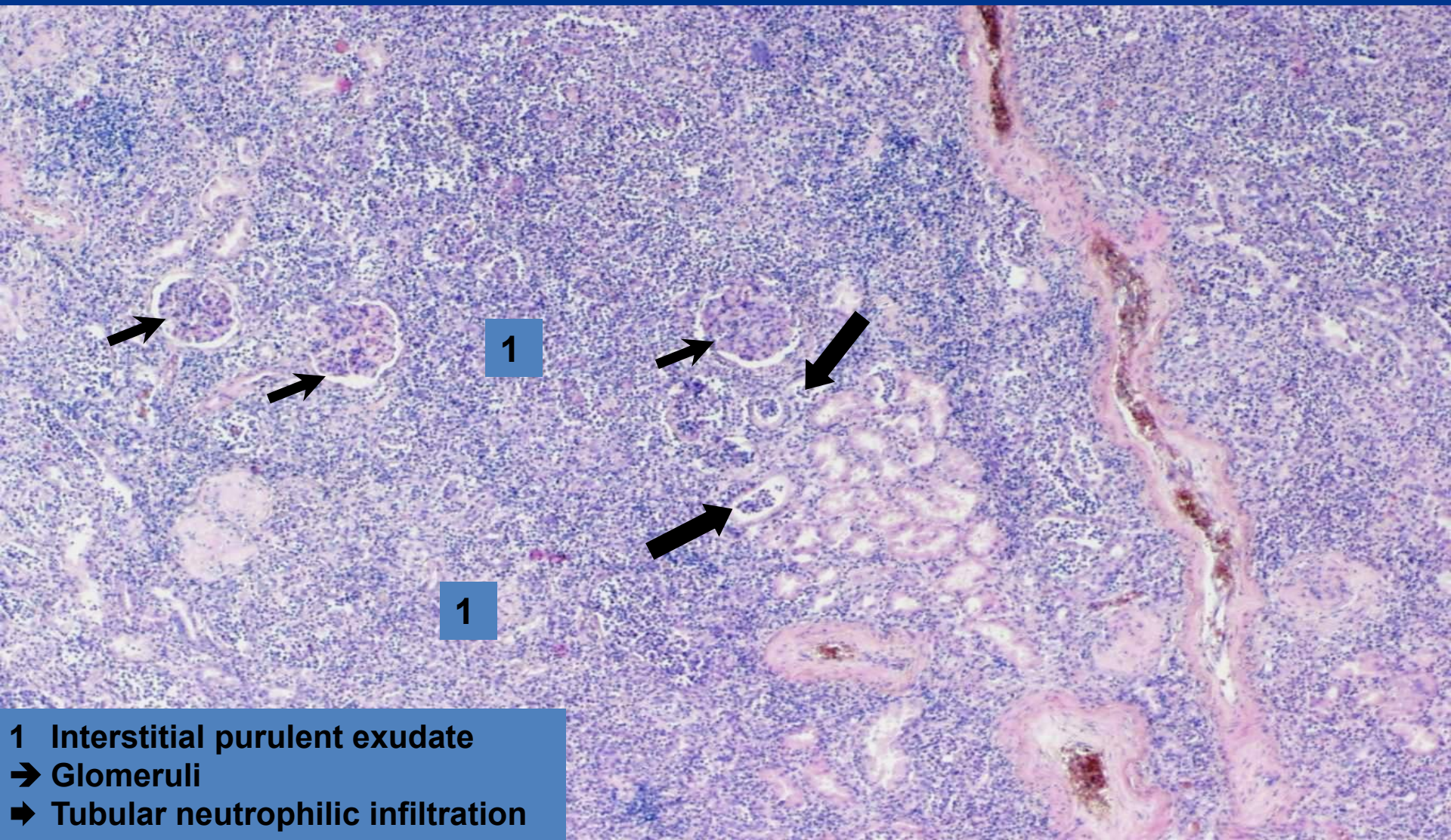
Acute pyelonephritis



Acute pyelonephritis with nephrolithiasis

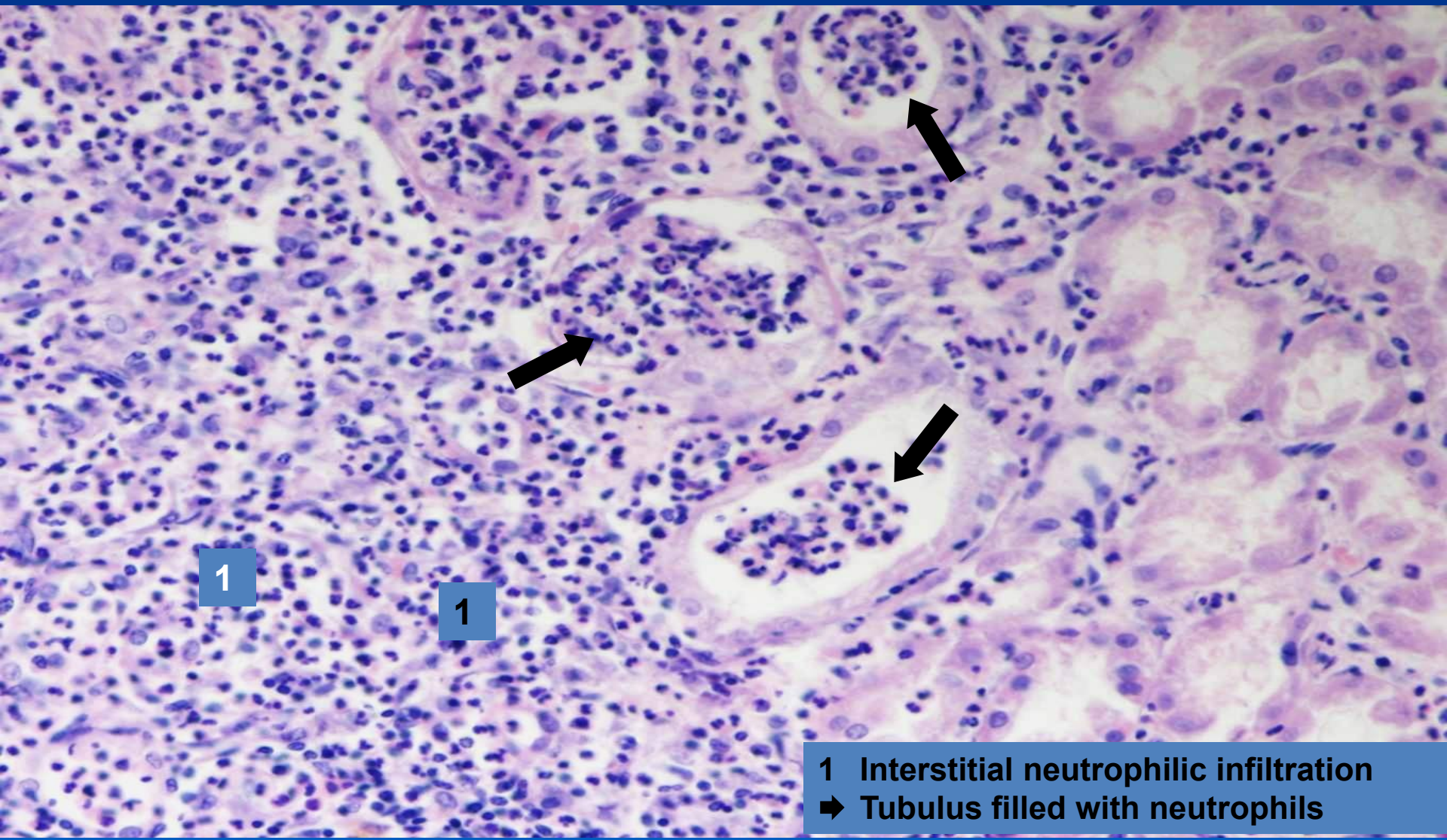


Purulent pyelonephritis
(interstitial purulent inflammation)



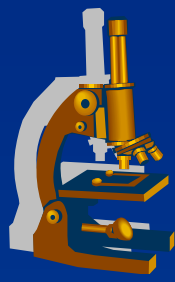
1 Interstitial purulent exudate
➔ Glomeruli
➡ Tubular neutrophilic infiltration

Purulent pyelonephritis
(interstitial purulent inflammation)



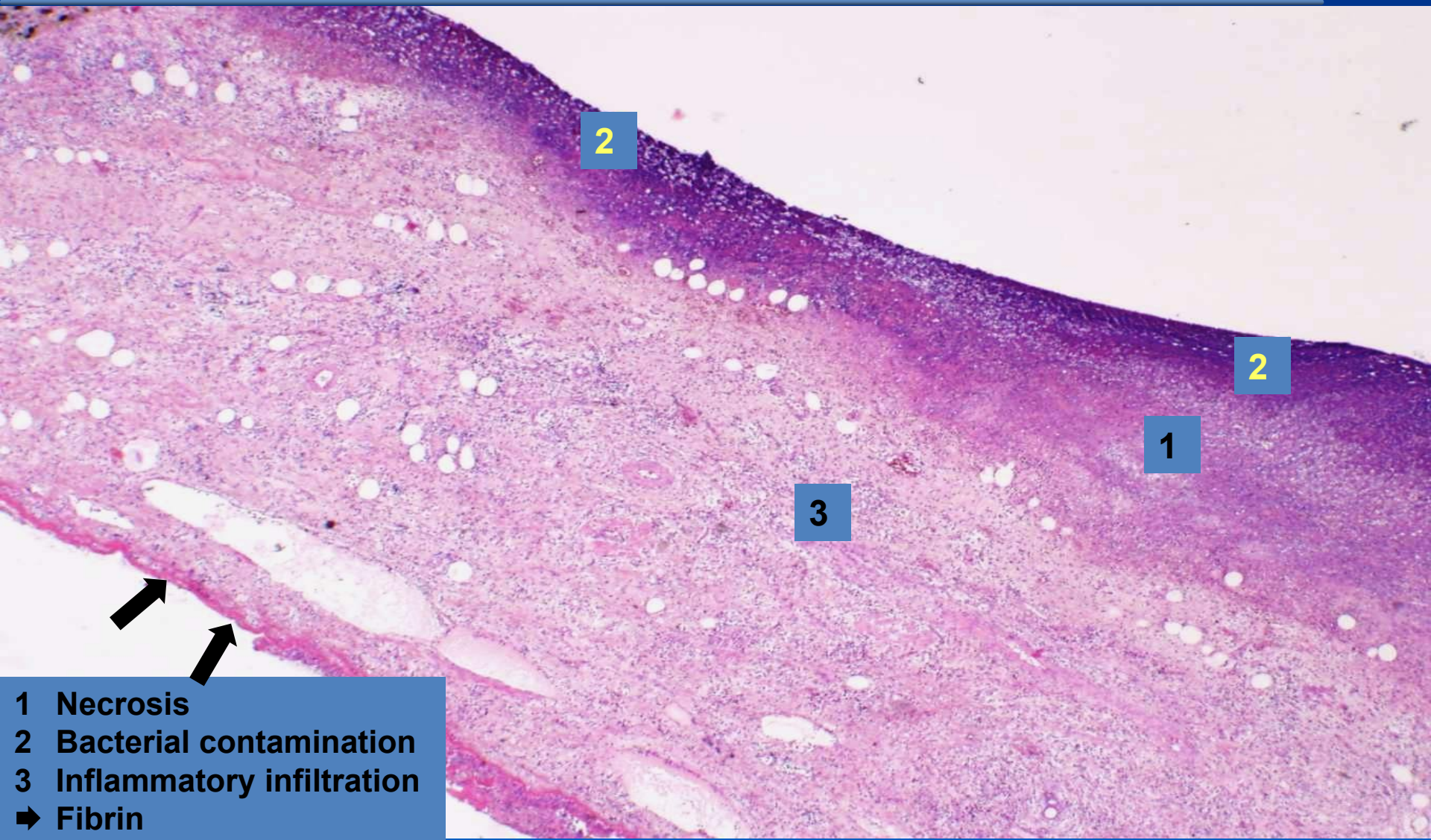
1 Interstitial neutrophilic infiltration
➡ Tubulus filled with neutrophils

Exudative inflammation



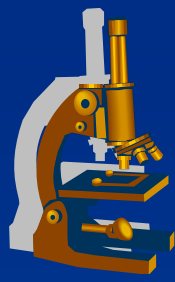
- gangrenous:
  - ⇨ ***necrosis modified with putrid bacteria***
  - ⇨ ***examples:***
    - gangrenous cholecystitis

Gangrenous cholecystitis
(gangrenous inflammation)



1 Necrosis
2 Bacterial contamination
3 Inflammatory infiltration
➡ Fibrin

Exudative inflammation

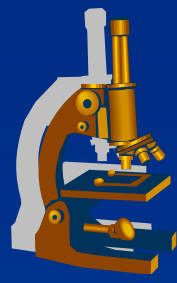


- non-purulent:
  - ⇨ ***exudate made by chronic inflammatory cells***

    (lymphocytes, plasma cells = mononuclear inflammatory infiltration)

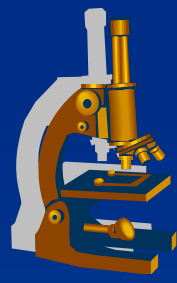
  - ⇨ ***examples:***
    - interstitial pneumonia
    - Hashimoto`s lymphocytic thyreoiditis

Primary (atypical) interstitial pneumonia



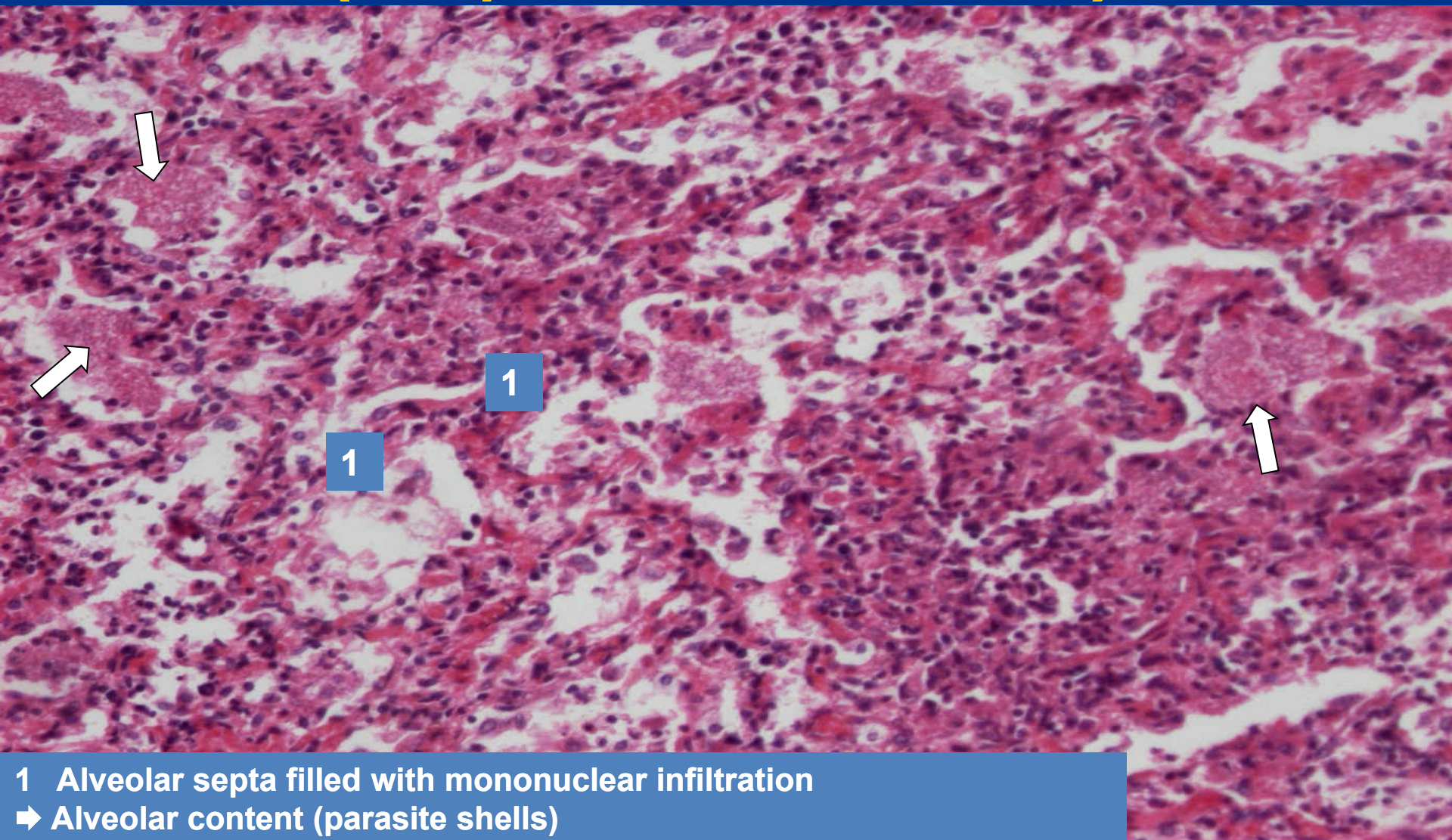
- etiology:
  - ***viral (influenza A, B; RSV, adenoviruses, rhinoviruses, HSV, CMV)***
  - ***small bacteria (*Mycoplasma pneumoniae)**
  - ***fungi (*Pneumocystis carinii*).***
- symptoms:
  - ***fever, dyspnoea, dry cough, auscultation may be normal (empty alveoli), x massive changes on X-ray***
- possible in normal hosts, more common in immunosuppressed

Primary (atypical) interstitial pneumonia



- GROSS:
  - ⇨ *focal or diffuse regions of hyperaemia, in fulminant cases with consolidation (ARDS – adult respiratory distress syndrome).*
- MICRO:
  - ⇨ *interstitial pneumonitis - oedematous septa with mononuclear infiltrate.*
  - ⇨ *hyaline membranes (ARDS)*
  - ⇨ *common secondary bacterial infection*

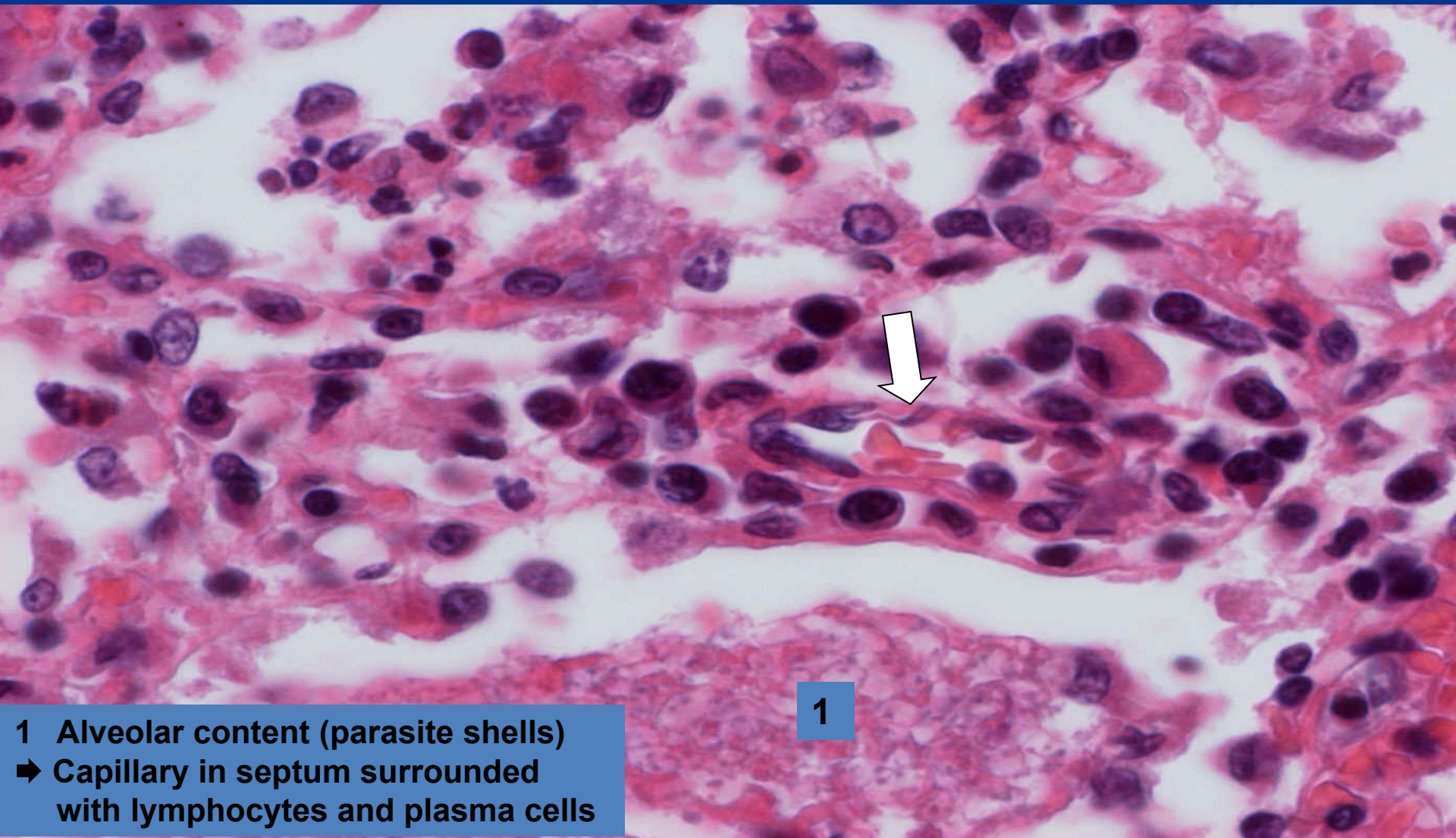
Interstitial pneumocystic pneumonia
(non-purulent inflammation)



1 Alveolar septa filled with mononuclear infiltration

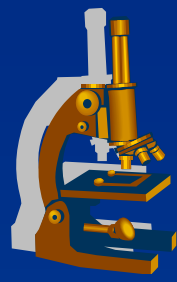
➡ Alveolar content (parasite shells)

Interstitial pneumocystic pneumonia - detail
(non-purulent inflammation)



1 Alveolar content (parasite shells)
➡ Capillary in septum surrounded with lymphocytes and plasma cells

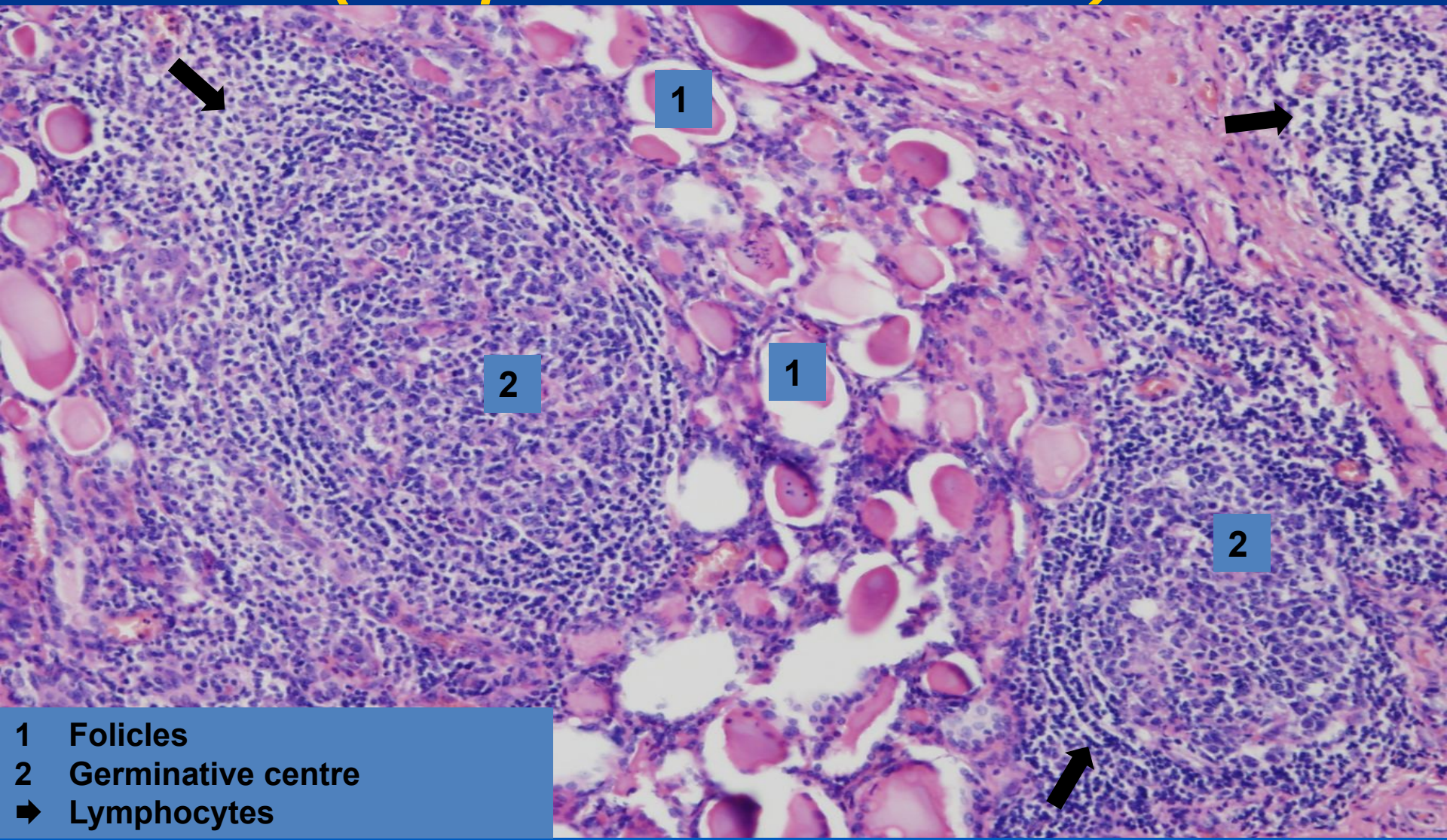
Hashimoto thyroiditis



- autoimmune
- antibodies against various th. antigenes
- loss of th. follicular cells
- lymphocytic infiltration
- neoformation of lymphoid follicles
- risk of other autoimmune diseases, thyroid tumors

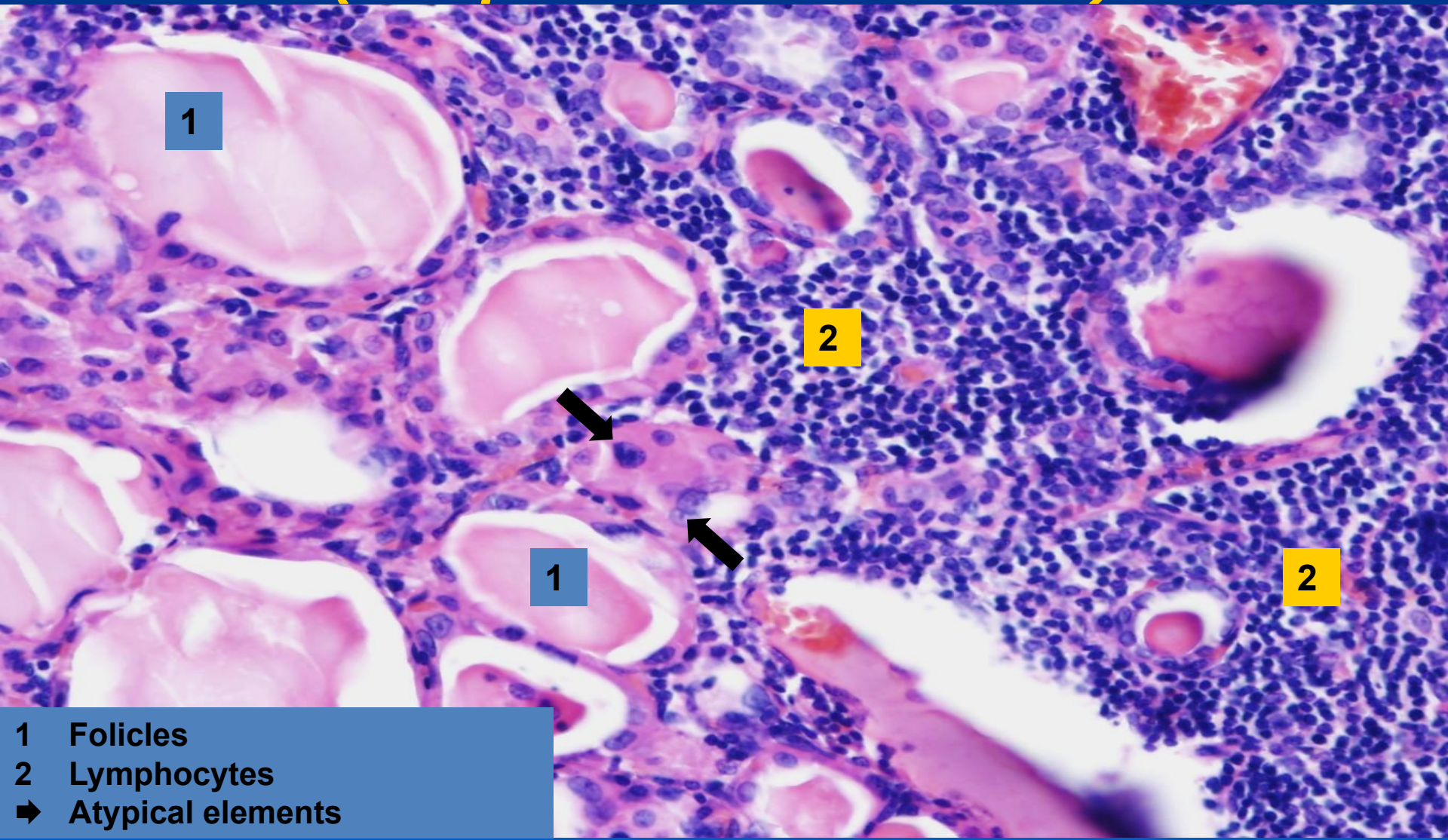
Chronic thyreoiditis

Hashimoto`s lymphocytic thyreoiditis
(non-purulent inflammation)



1 Folicles
2 Germinative centre
➡ Lymphocytes

Hashimoto`s lymphocytic thyreoiditis – detail
(non-purulent inflammation)



1 Folicles
2 Lymphocytes
➡ Atypical elements