

Hemodynamic disorders I.

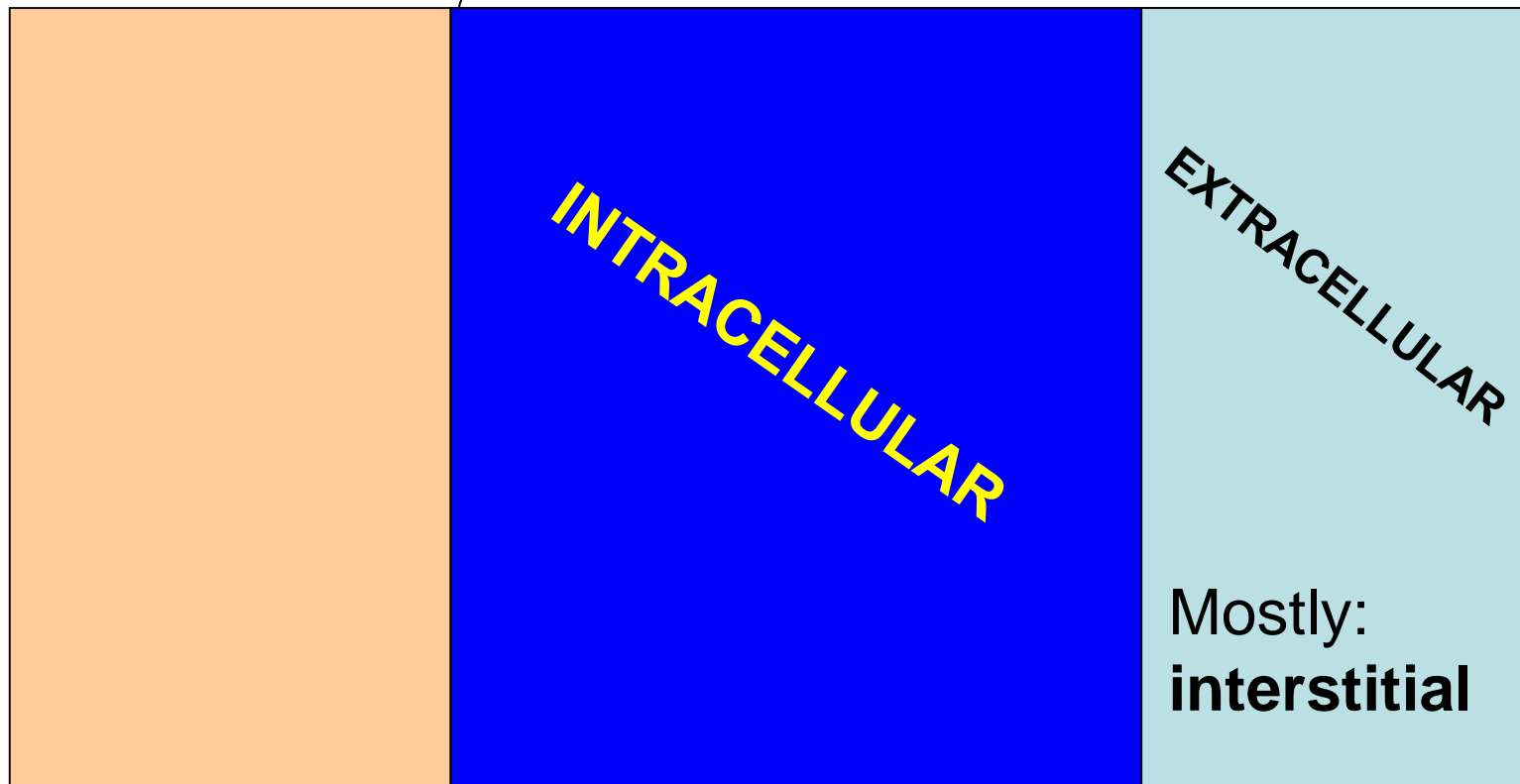
Edema, congestion

Edema: increased fluid content in the interstitium (transudate or exudate)

Transudate: protein content < 3 mg/ml

Exudate: protein content > 3 mg/ml

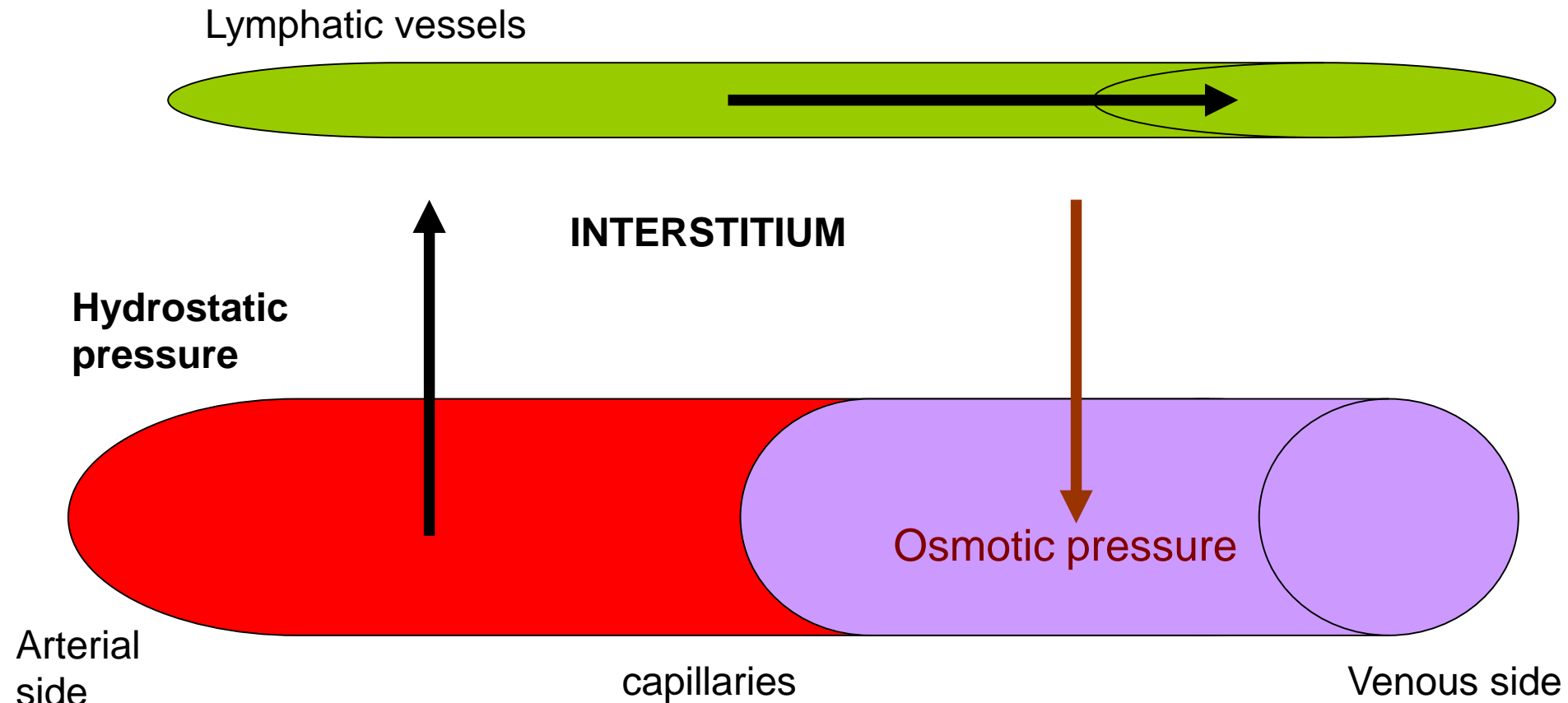
60 % of the body mass is water



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Edema formation



Edema formation - causes

(Inflammation:

- acute/chronic) later...

-inflammation (lymphangitis)

-tumor

lymphoedema

Lymphatic vessels



INTERSTITIUM

Hydrostatic
pressure



- nephrosis-syndrome

- liver failure

- malnutrition

Osmotic pressure
albumin



Venous side

-local: deep vein thrombosis

-generalized: heart failure + renin-AT/ fluid retention

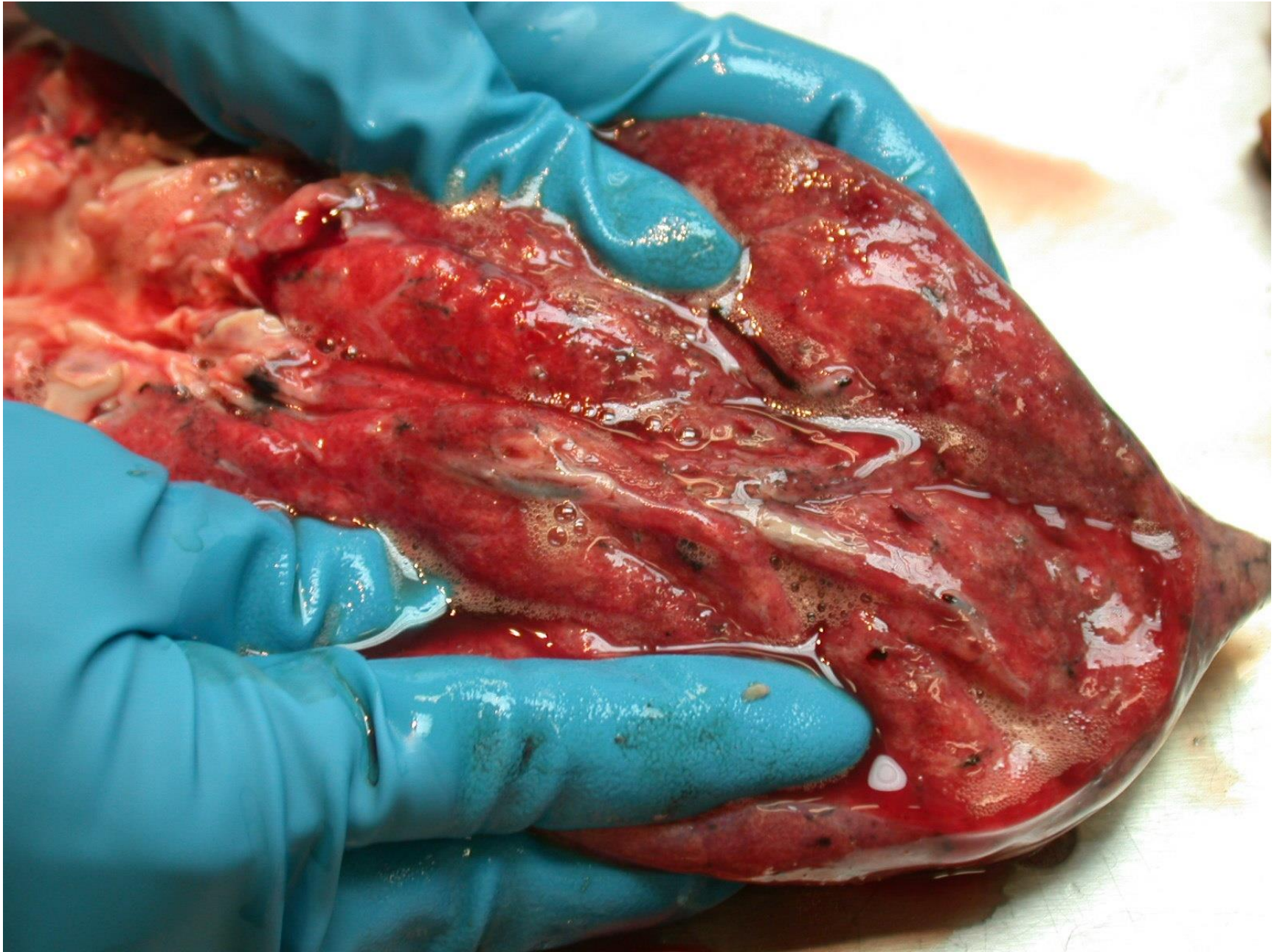
- acute renal failure/salt and water retention

Causes of lung edema

(oedema pulmonis)

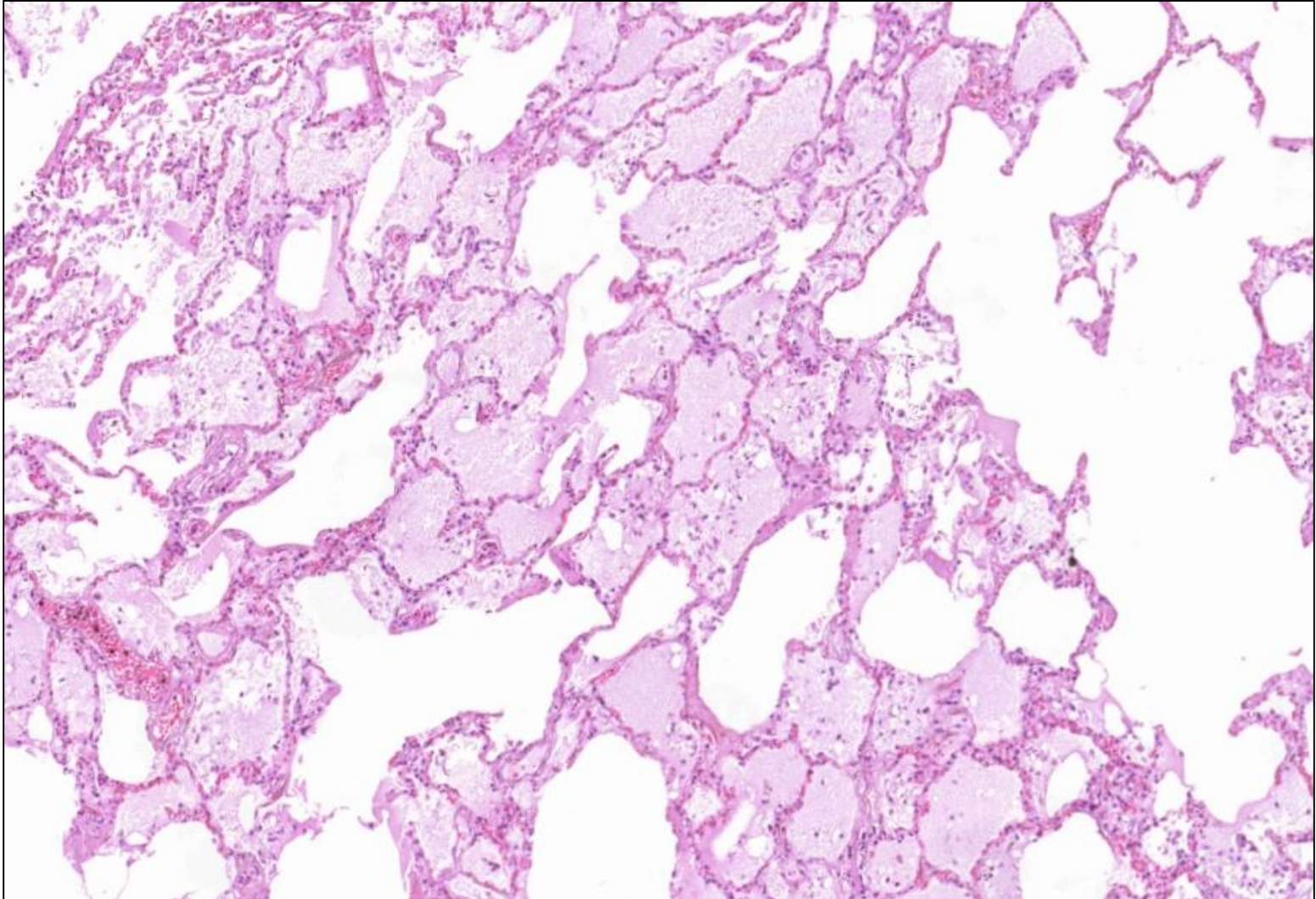
- Cardiogenic: left heart failure: $p_{\text{hydrostatic}}$ ↑
- Non-cardiogenic: vascular permeability ↑
 - Toxic damage
 - Renal failure (uraemia, hypoproteinaemia)
 - Hypoxia
 - Fluid and electrolyte management disorders (iatrogenic: infusion mismanagement)

Oedema pulmonis



Oedema pulmonis

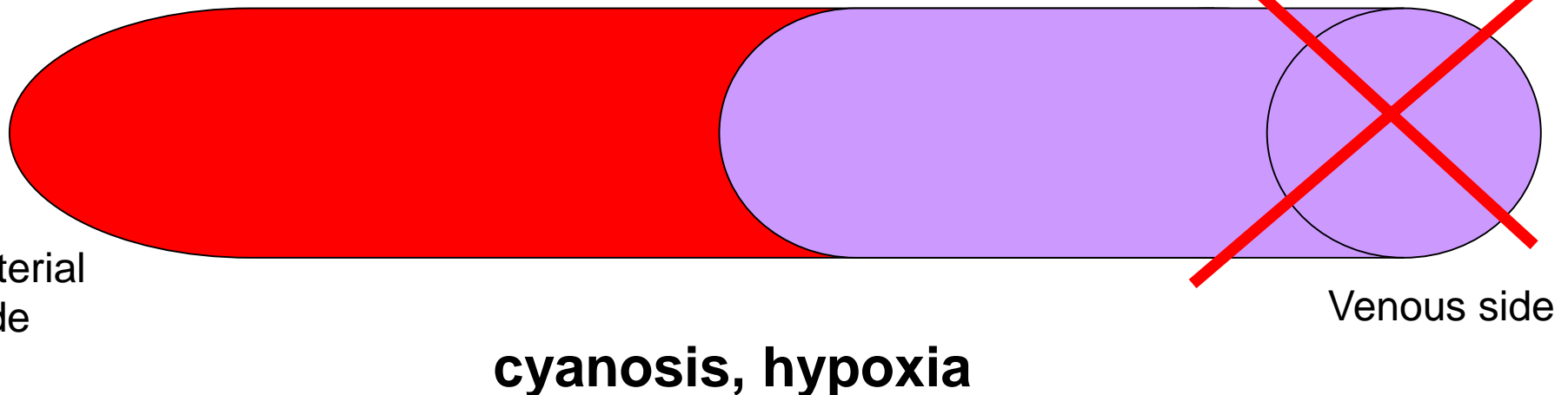
- **Microscopically**
 - eosinophilic transudate in the alveoli



Congestion

= passive hyperaemia

- systemic: heart failure
- local: venous occlusion
- acute
- chronic -> leads to:
parenchymatous degeneration / necrosis



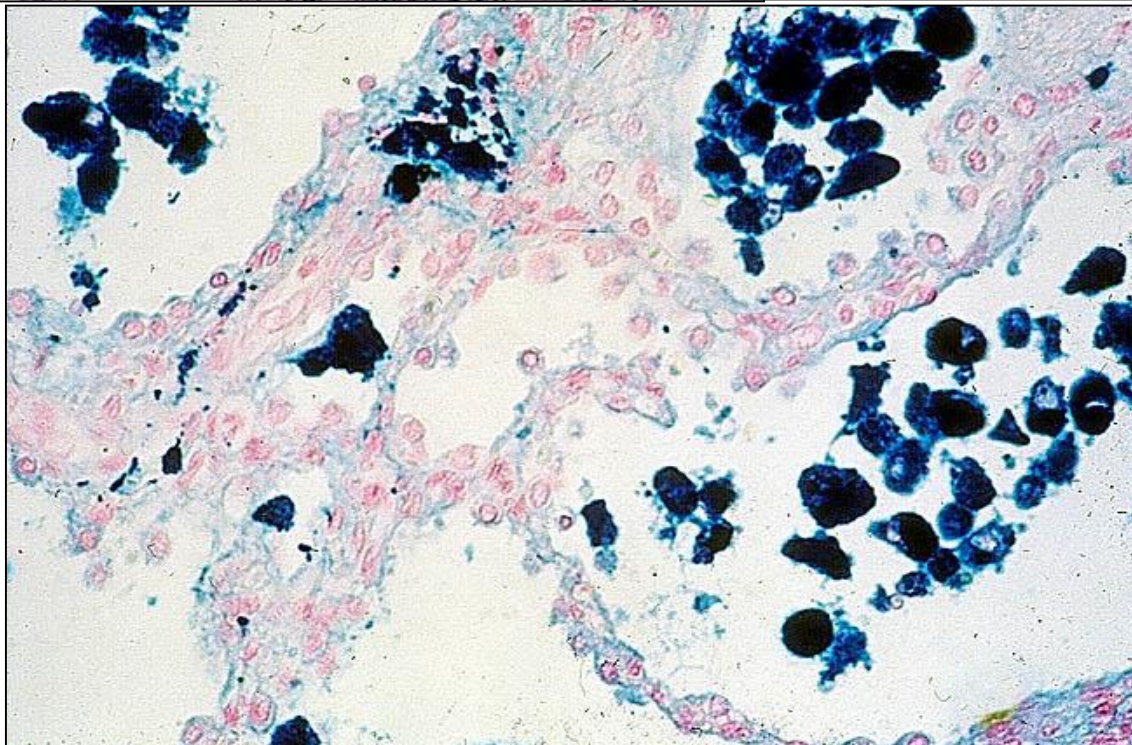
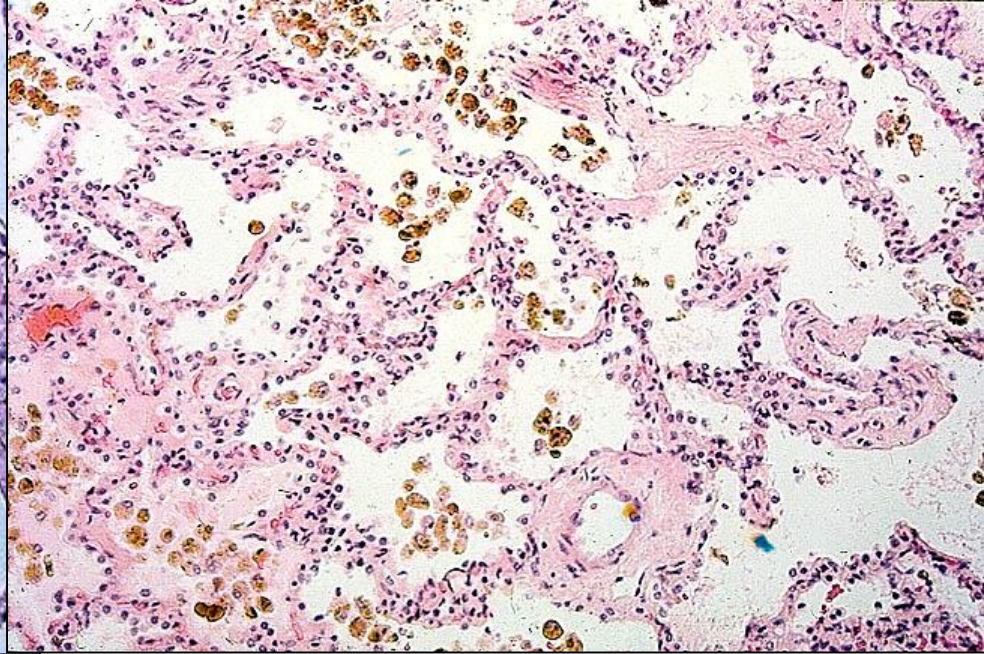
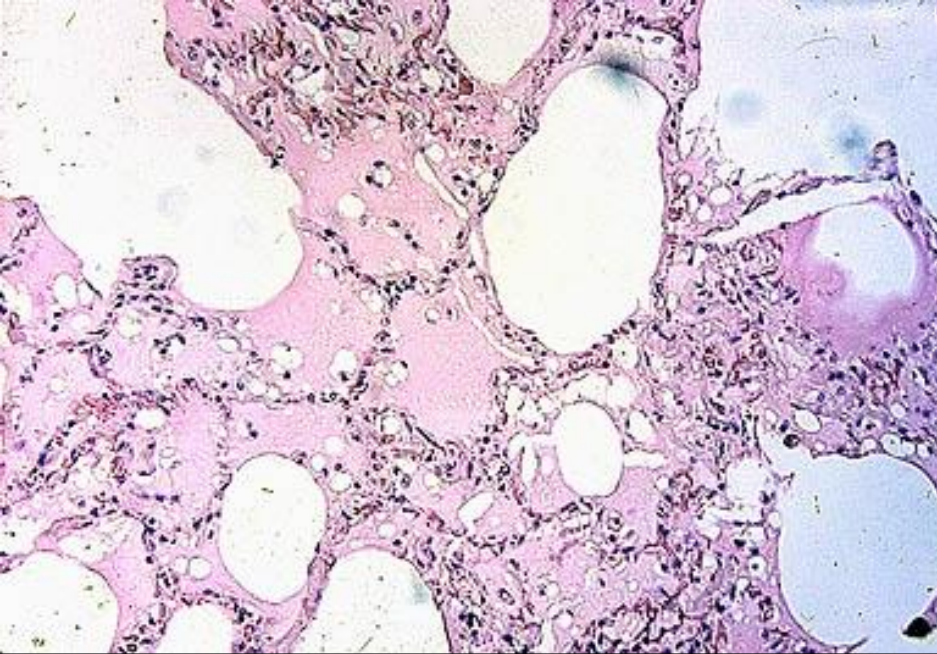
Lung congestion

- Acute
 - Lungs are heavy, firm elastic
 - Alveolar capillaries are engorged with blood
- Chronic:
 - firm, brownish: induratio brunea pulmonis
 - fibrotic septa
 - hemosiderin laden macrophages („heart failure cells”)

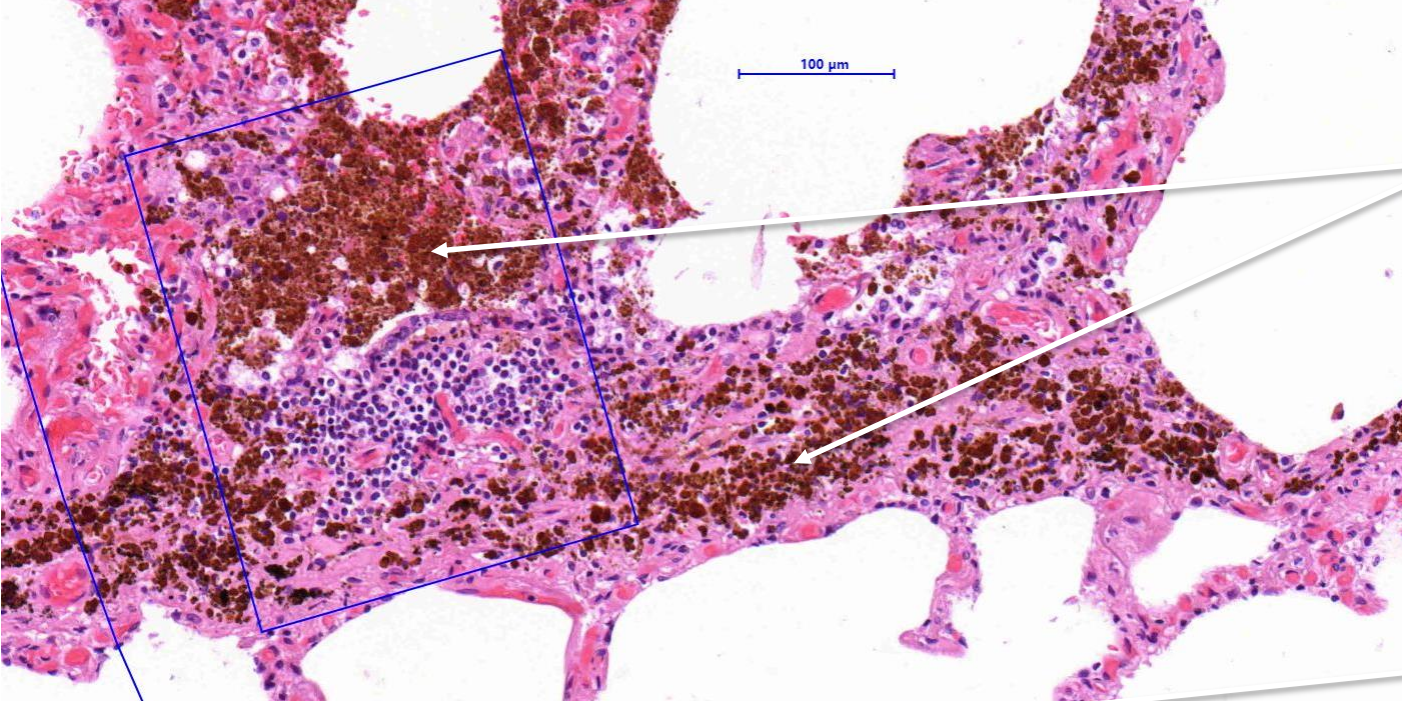
CAUSE: left heart failure

Induratio brunea pulmonis

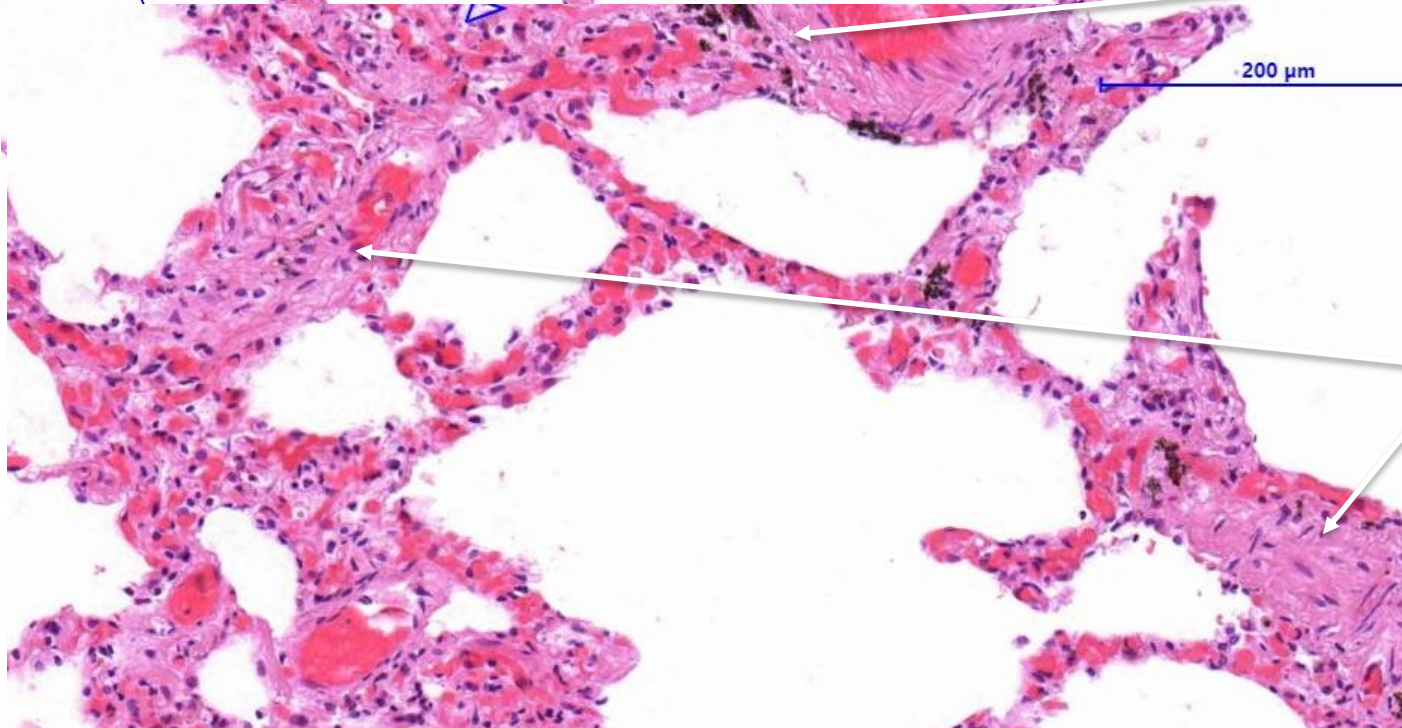




Prussian-blue
reaction



Hemosiderin laden
macrophages (brown
pigment)



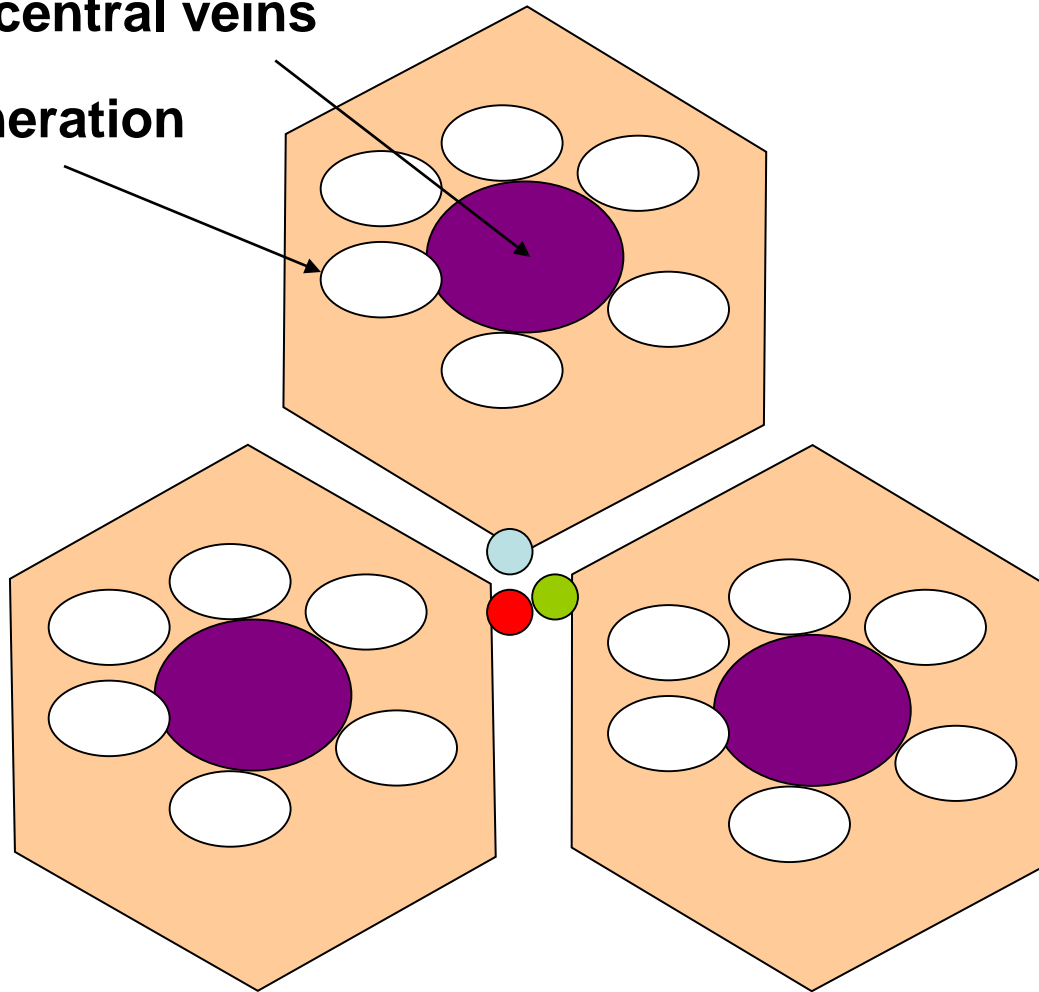
Anthracosis
(black pigment)

Fibrotic septae

Liver congestion

Chronic: nutmeg liver (hepar moschatum)

- dilation of central veins
- fatty degeneration



Hepar moschatum

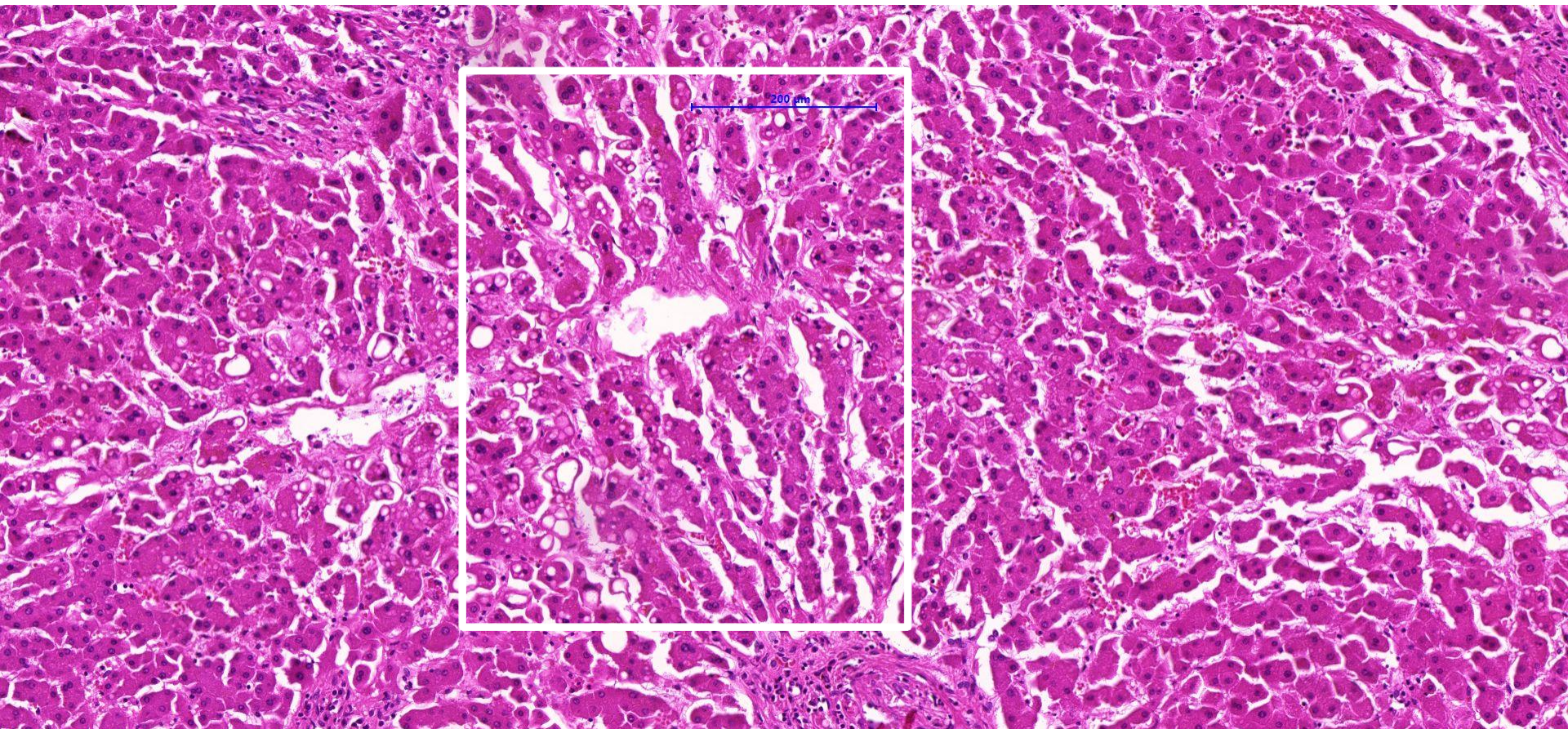




Boldog Kukta

boldogkukta.blogspot.com

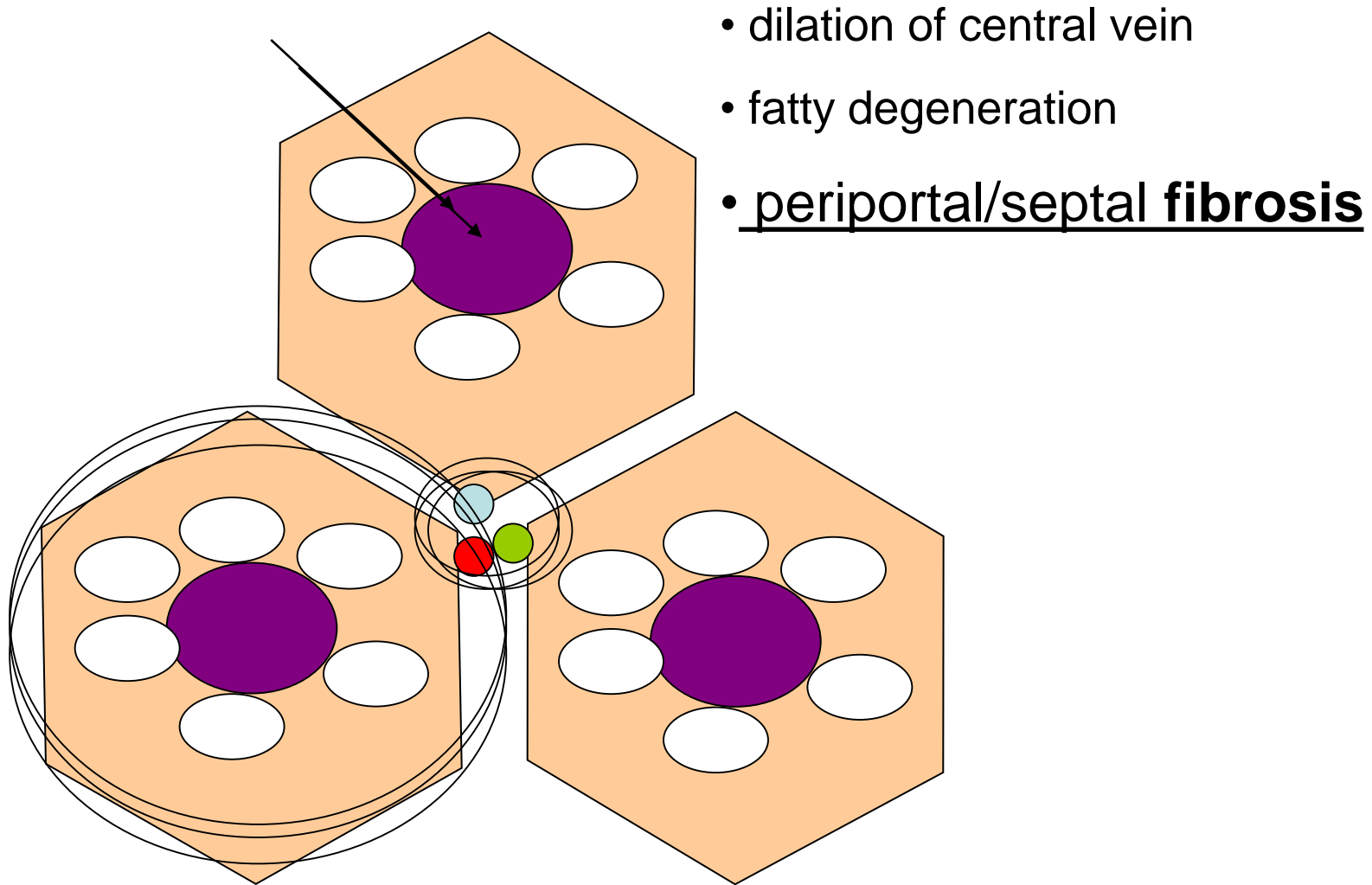
Hepar moschatum (preliminary stage)



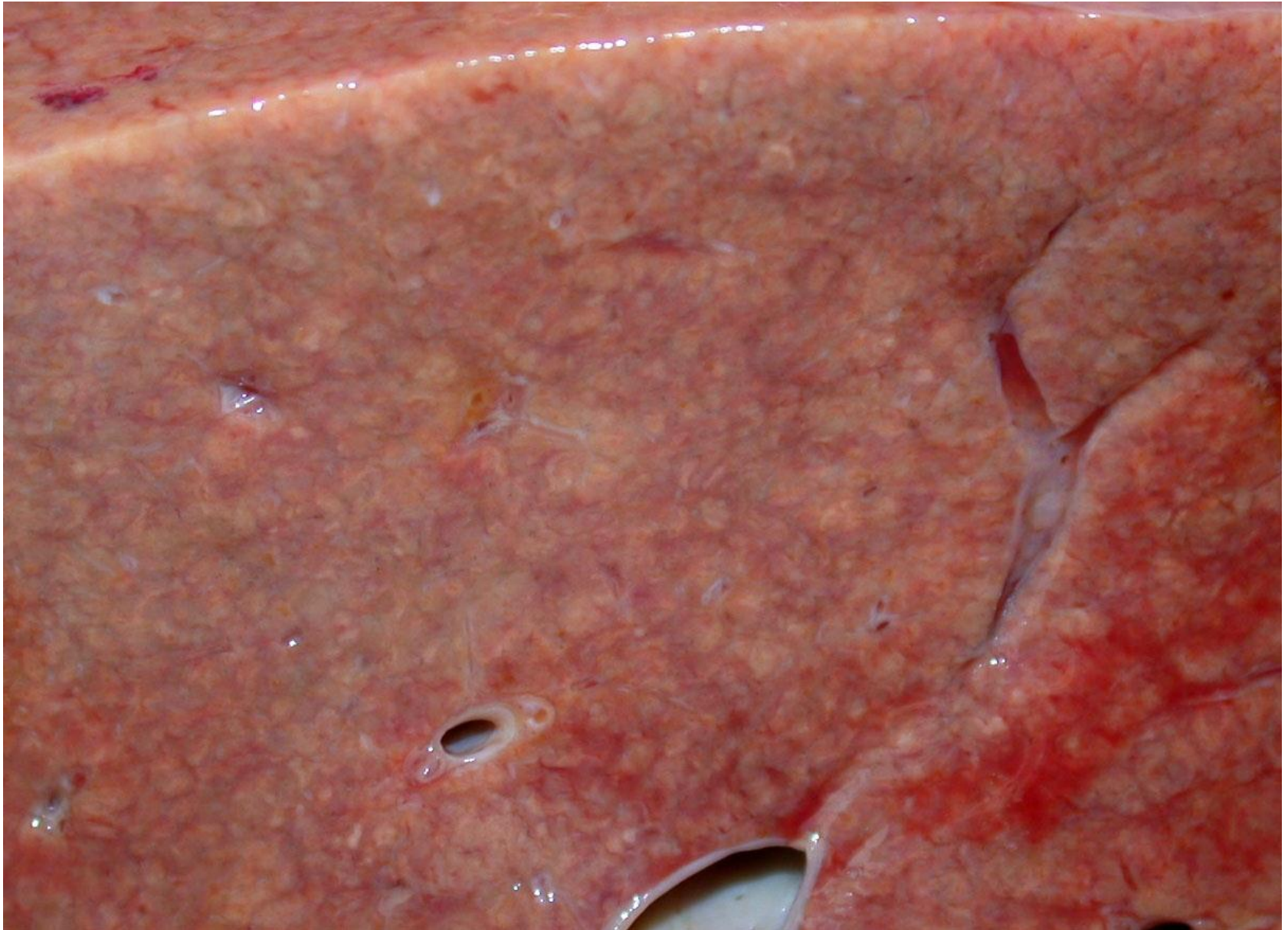
Centrilobular stasis and sinusoidal dilatation

Liver congestion

Chronic complication of congestion : liver fibrosis



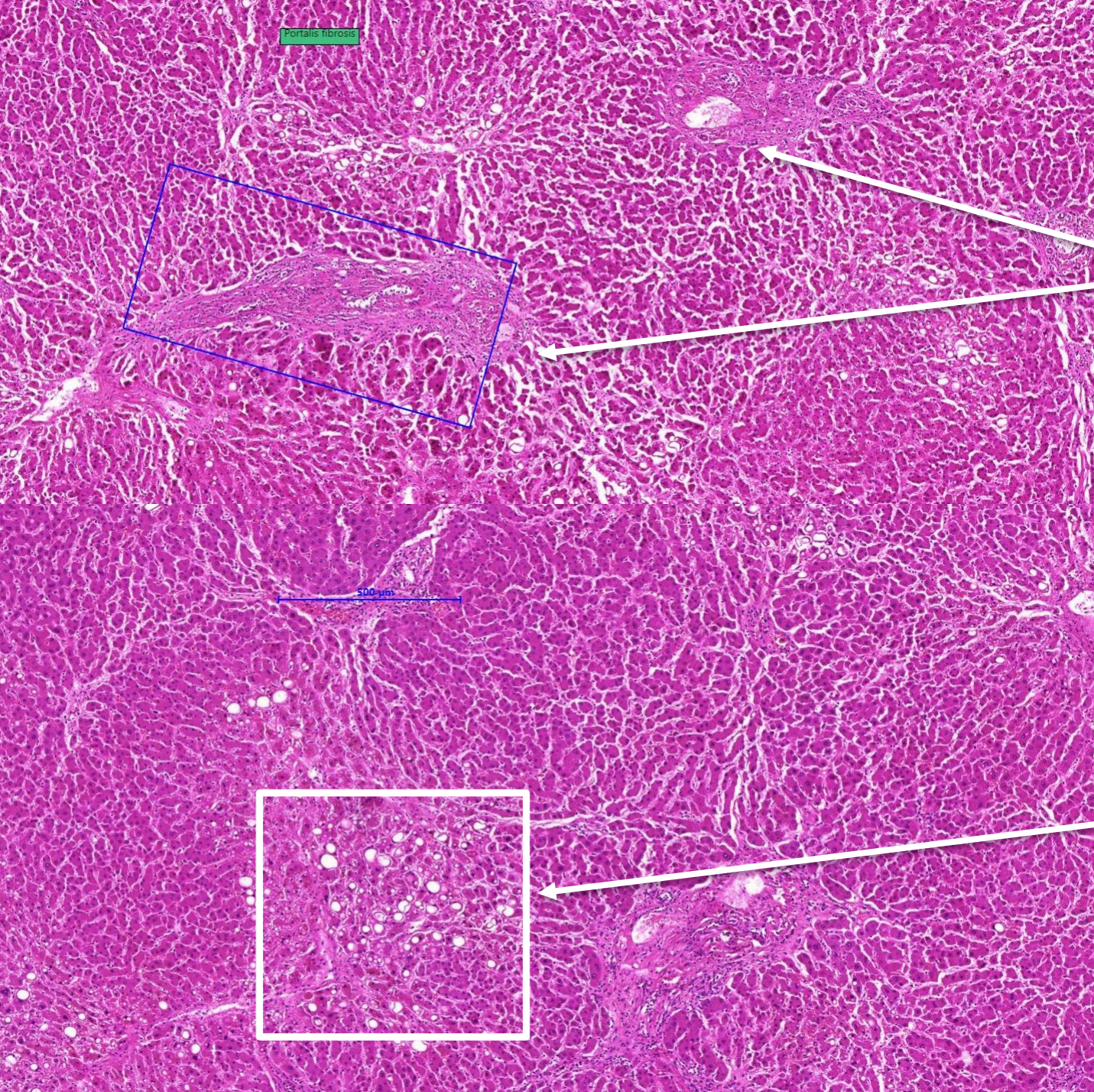
Fibrosis hepatis



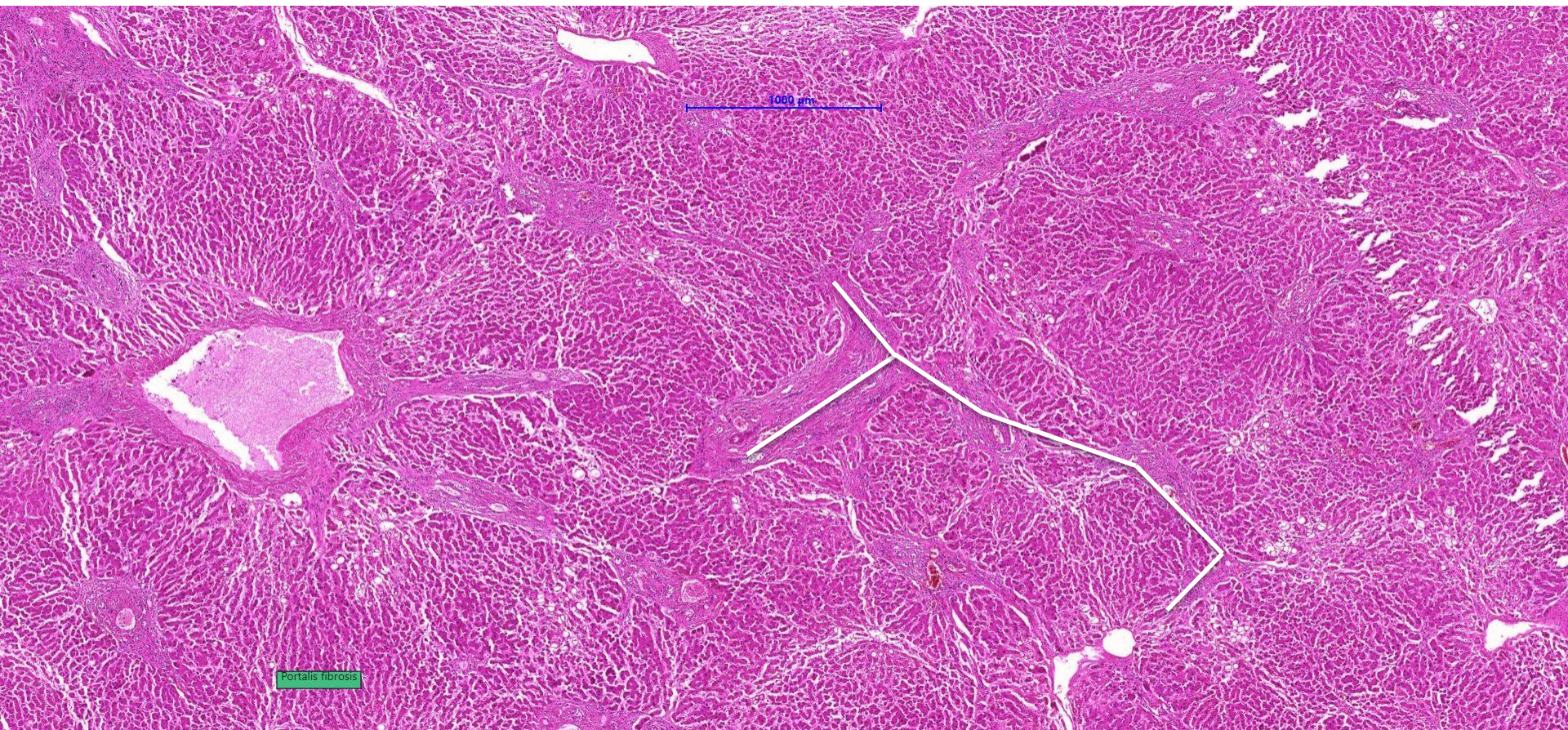
Later stage

Portal fibrosis

Centrolobular necrosis



Last stage: cardiac fibrosis (,cirrhosis') with joining
fibrous septae



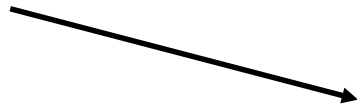
DIC (disseminated intravascular coagulopathy)

Endothelial
damage

Thrombi in the
microcirculatory
system

Coagulation factors,
thrombocytes are
consumed, fibrinolysis is
activated

Severe bleeding



DIC under the microscope (PTAH-staining)

