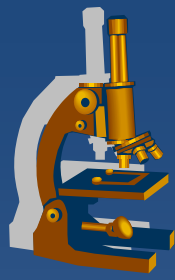
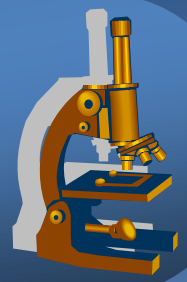
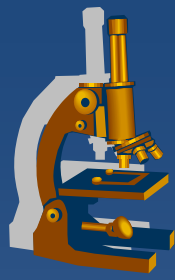


Systemic Pathology



CARDIOVASCULAR system

ATHEROSCLEROSIS

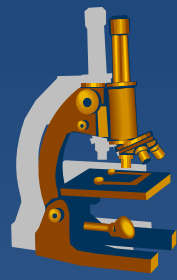


- disease of large and medium-sizes arteries with lipid deposition into intima
- active inflammatory process

- **endogennous risk factors, mostly noninfluenceables :**
  - *age, MxF (estrogen?), familiar factors (f. hypercholesterolemia), hereditary homocysteinemia*

- **exogennous risk factors**:
  - *hyperlipidemia (LDL) ←← hypothyreosis, nephrotic sy;*
  - *hypertension, diabetes mellitus, life style smoking (nicotine, CO), sedentary life, food + obesity; ↑CRP*

Atherosclerosis - pathogenesis



1. **Endothelial injury**
   - *mechanic (↑BP, turbulence)*
   - *endotoxins, immune complexes, exogennous toxins (cig. smoke), ↑ cholesterol*

   ↑ expression of cell adhesion molecules, ↑ permeability, ↑ thrombogenicity

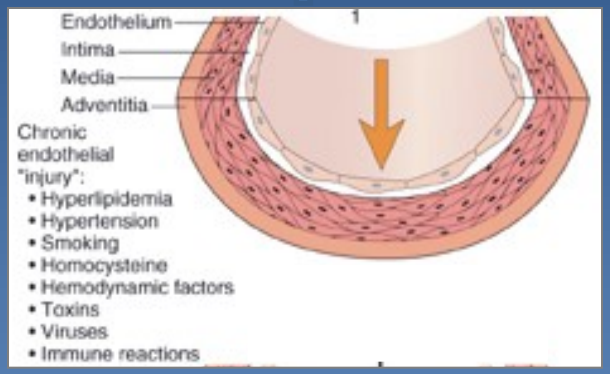
2. **Lipoprotein insudation (LDL) – oxidation** in intima

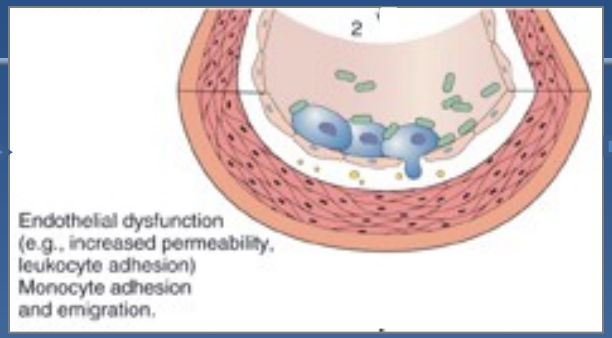
3. **Inflammation**
   - *blood monocytes (→foam cells), T-cells, platelets, smooth muscle cells*

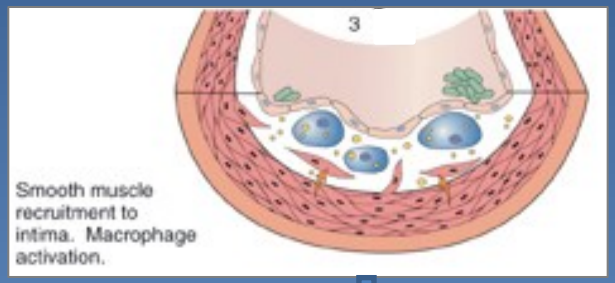
4. **Repair - proliferation of myointimal cells**
   - *synthesis of collagen, elastin, proteoglycans → **fibrotic plaque**, + lipid accumulation - **atheromatous plaque***

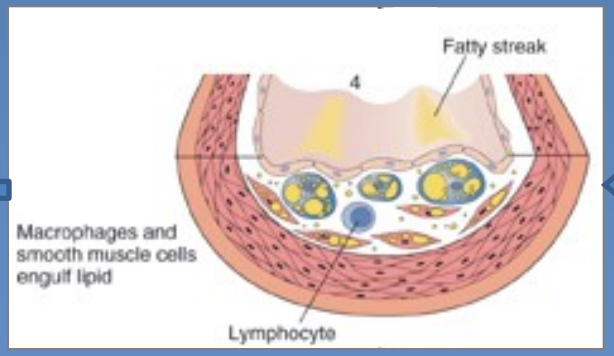
stable plaque under repeated inflammation turns into unstable plaque – fibrous cap + endothelium rupture - thrombus

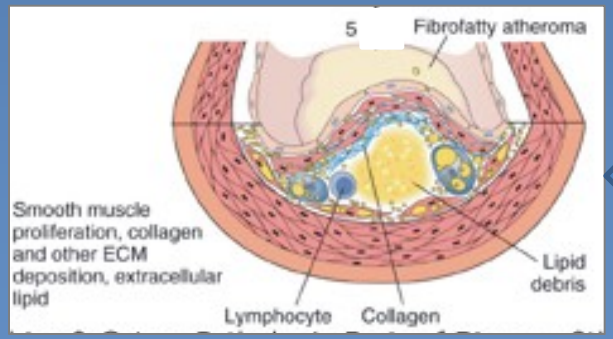
Atherosclerosis - pathogenesis



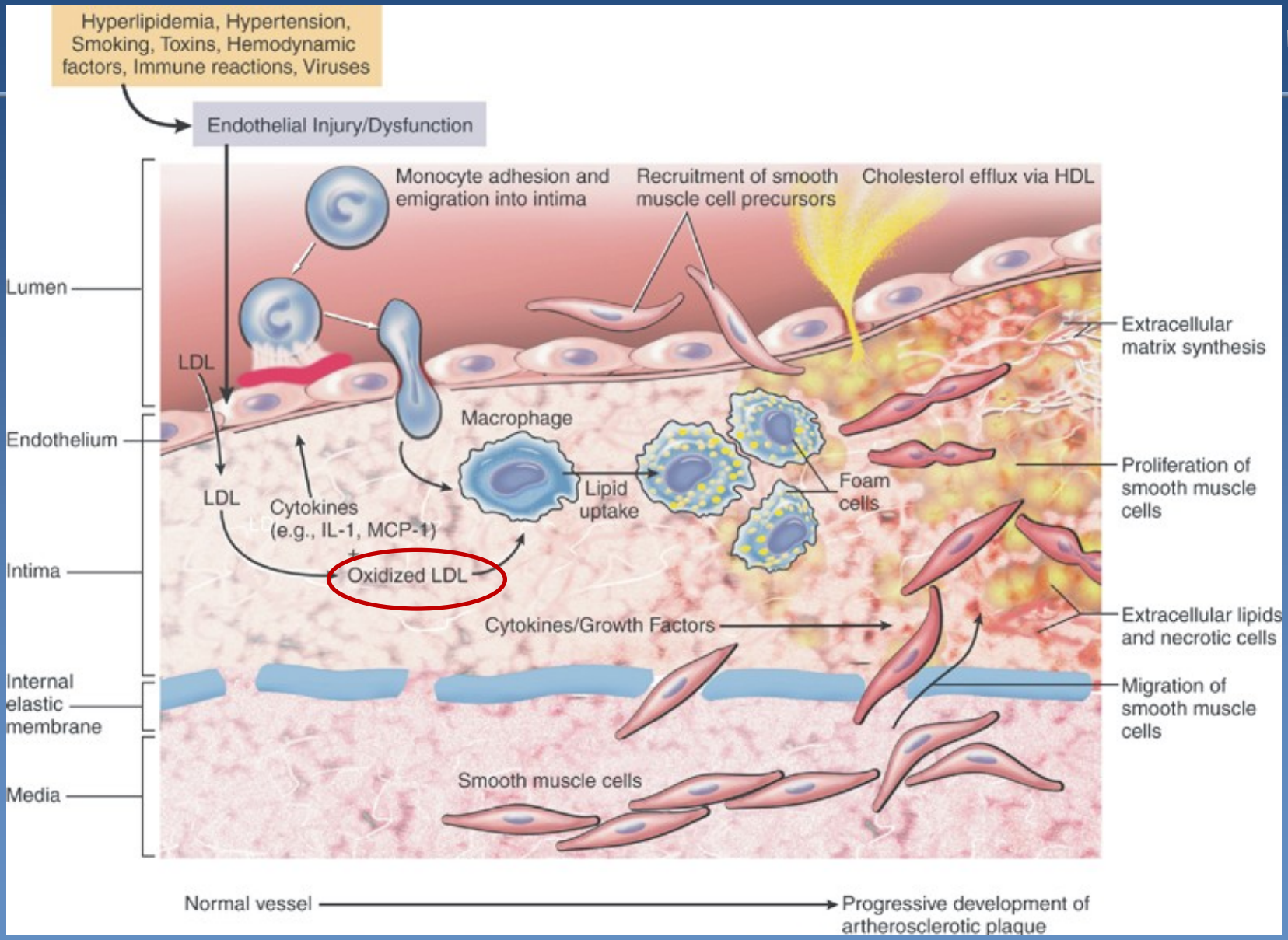




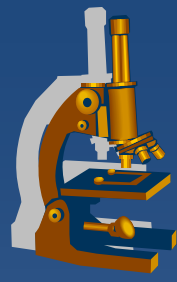




atherosclerosis – cell interactions in an atheromatous plaque

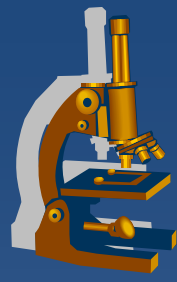


Atherosclerosis



- fatty streak
- fibrotic plaque
- atheromatous plaque
- complicated atheromatous plaque (ulceration, calcification, thrombosis)

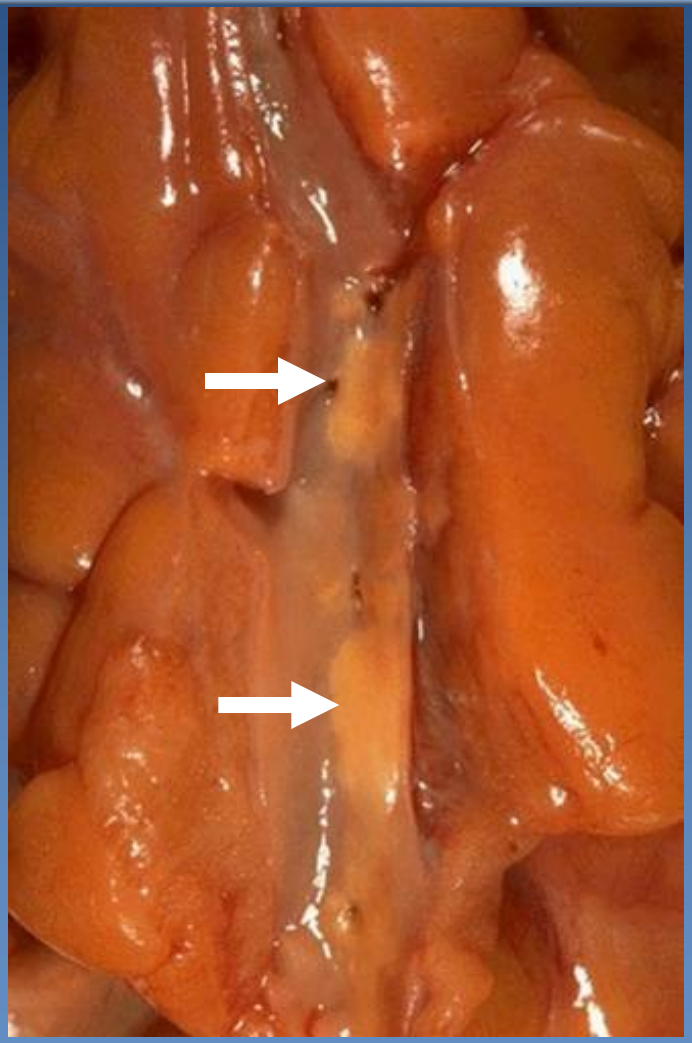
Atherosclerosis

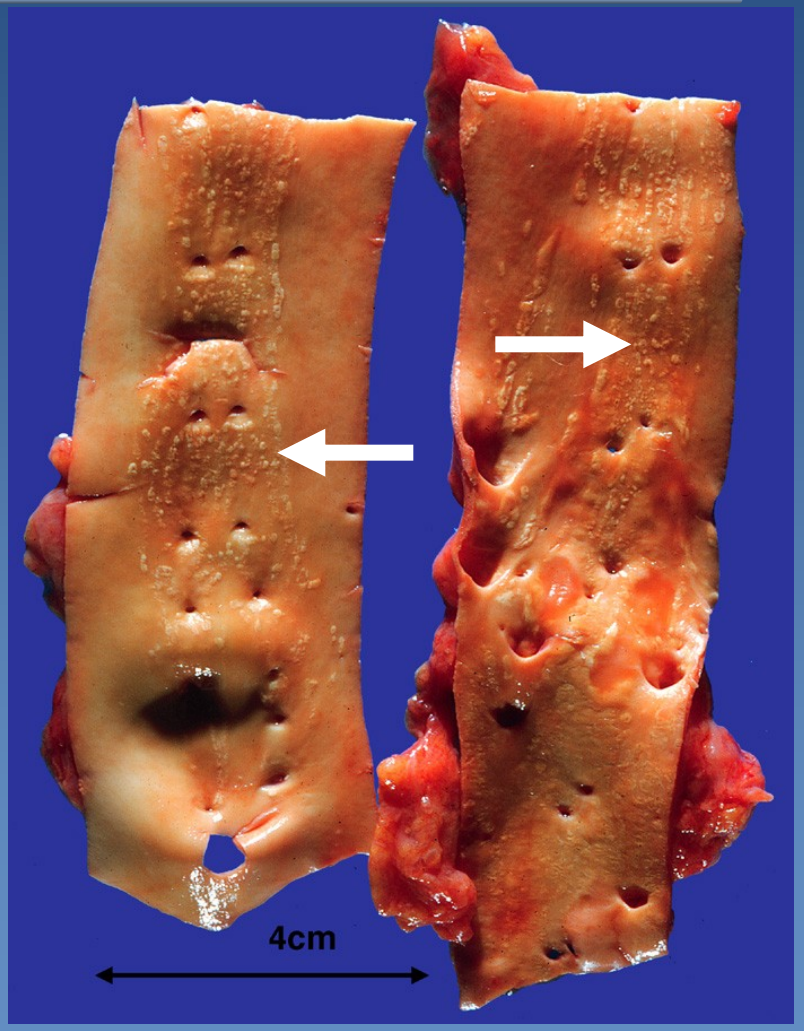


SEQUELS: arterial occlusion in situ

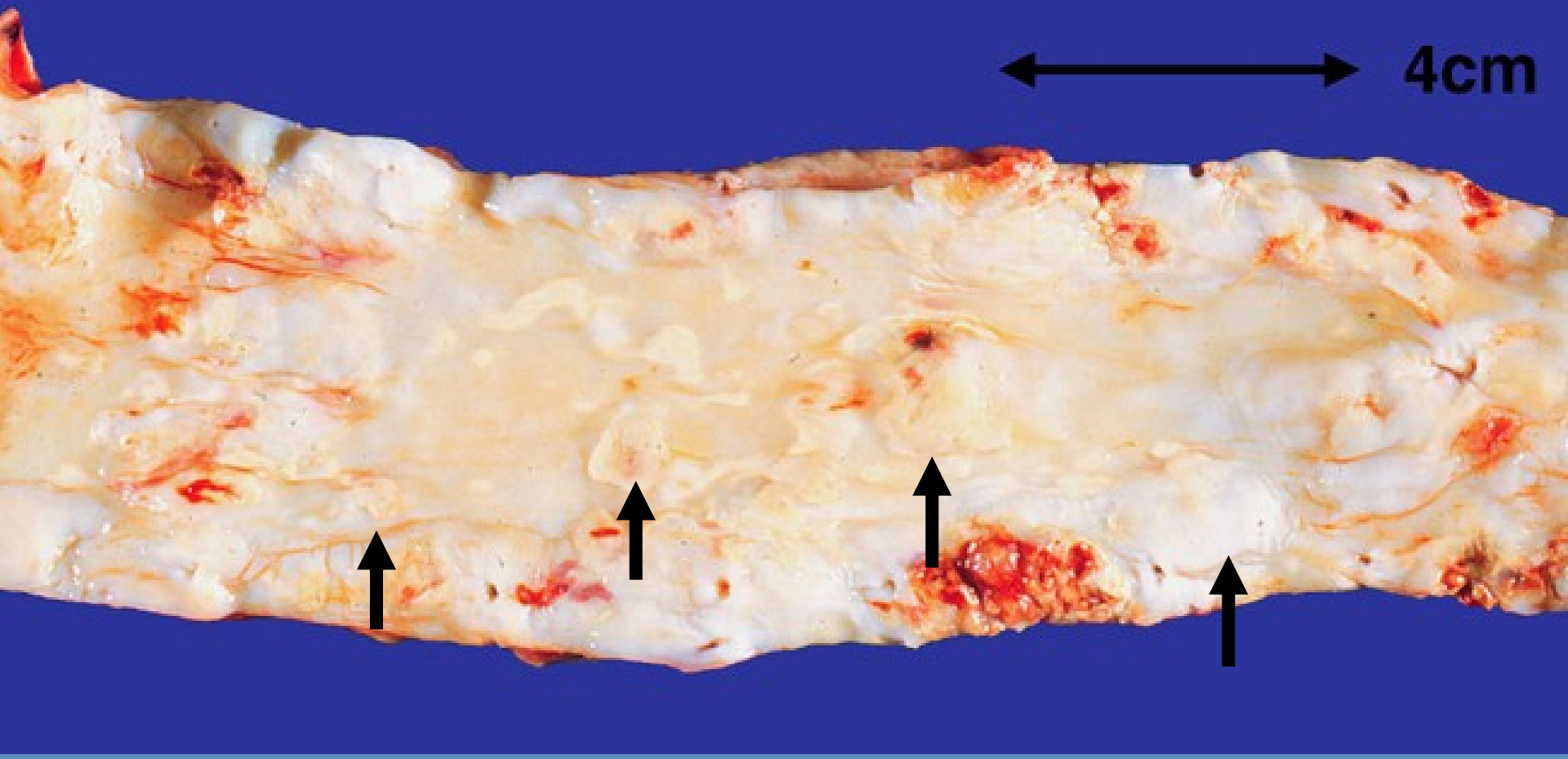
- chronic (→ hypoxia, atrophy)
- acute (→ ischemia, infarction, encephalomalatia)
- embolism (thrombus, plaque material)
- weakening of arterial wall (aneurysm), risk of rupture
- bleeding (from plaque, fissured wall)
- calcification (hypertensive factor)

Atherosclerosis– fatty streak

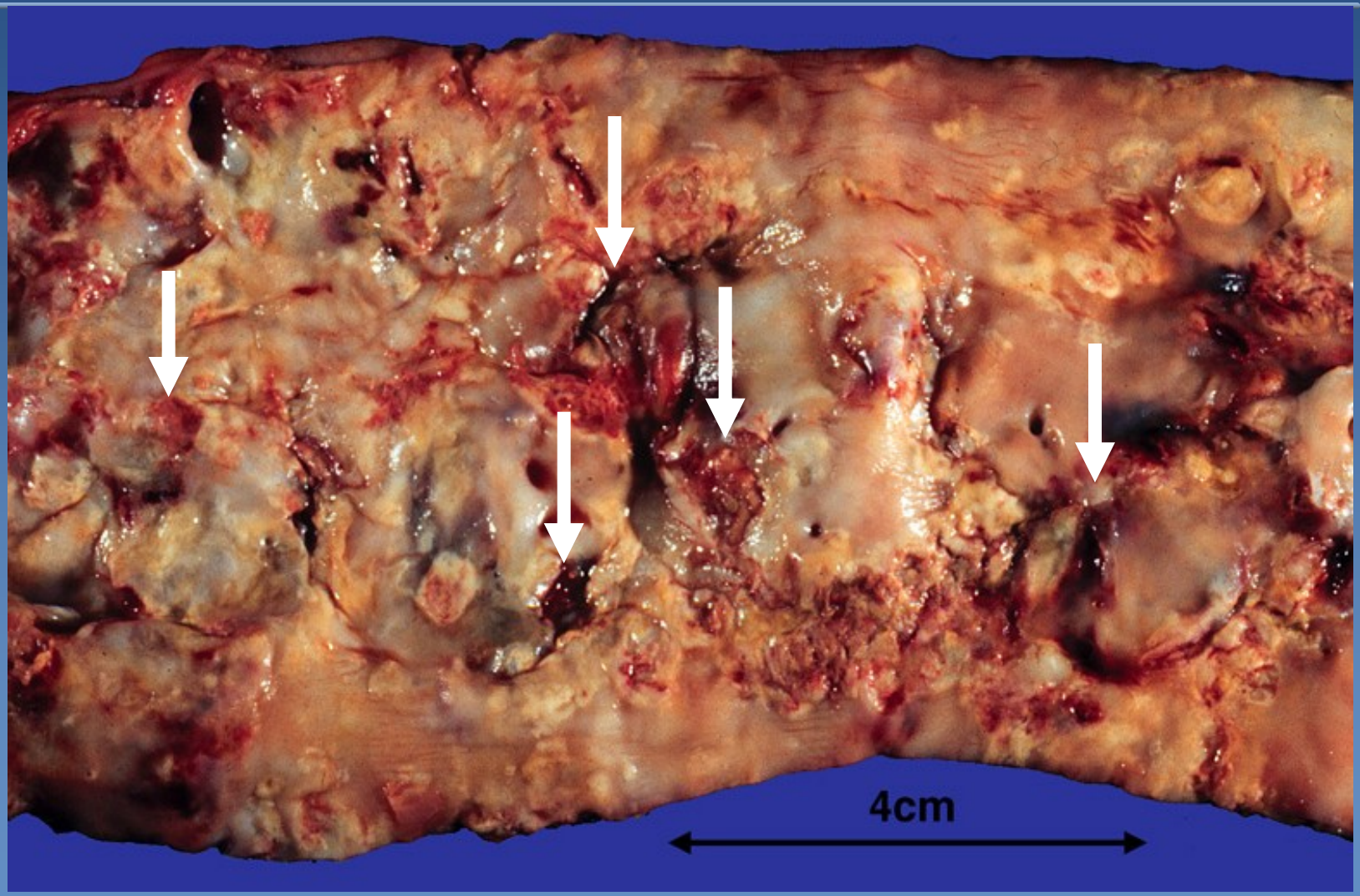




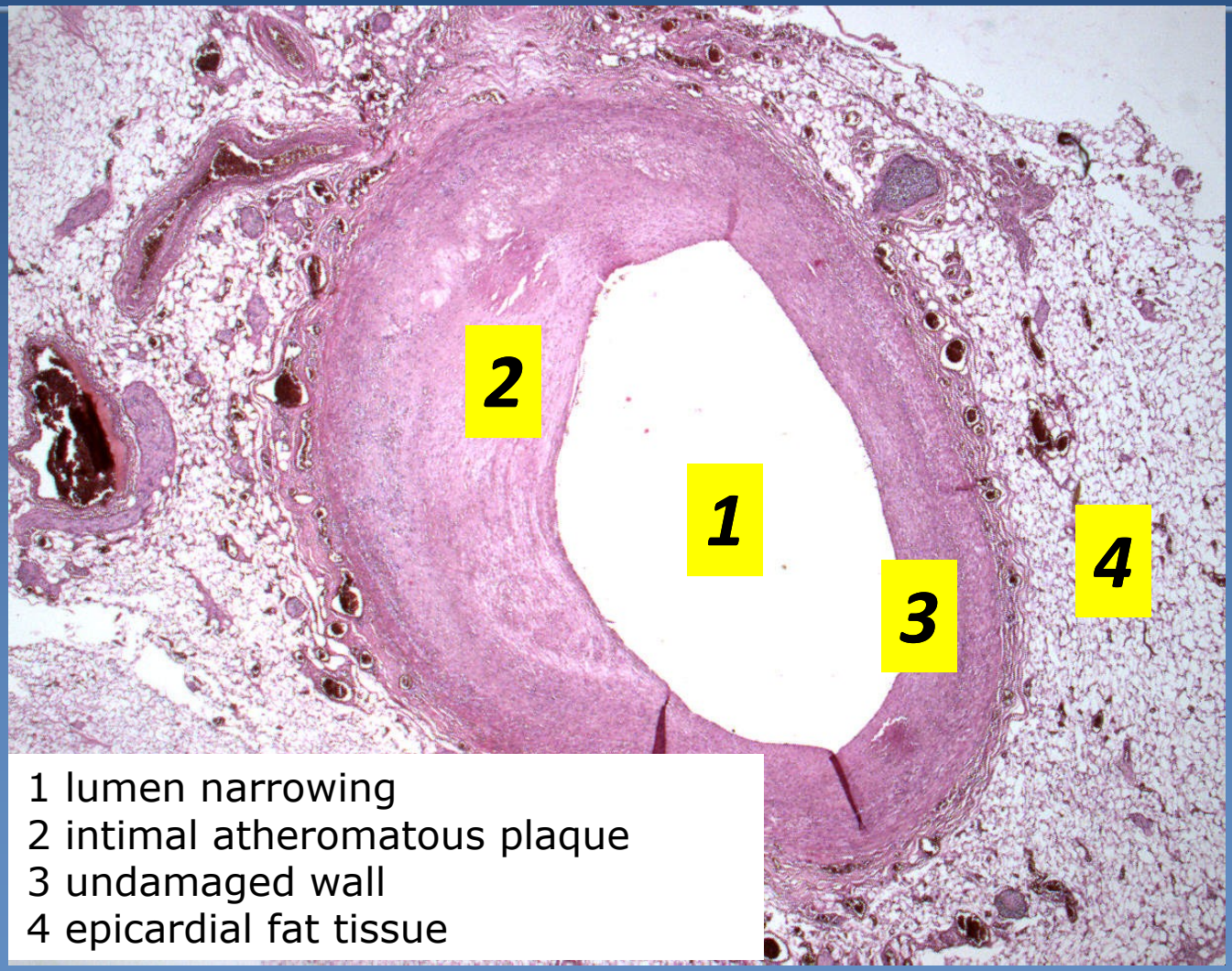
Atherosclerosis –
fibrous and atheromatous plaques



Atherosclerosis–
plaque ulceration, mural thrombosis

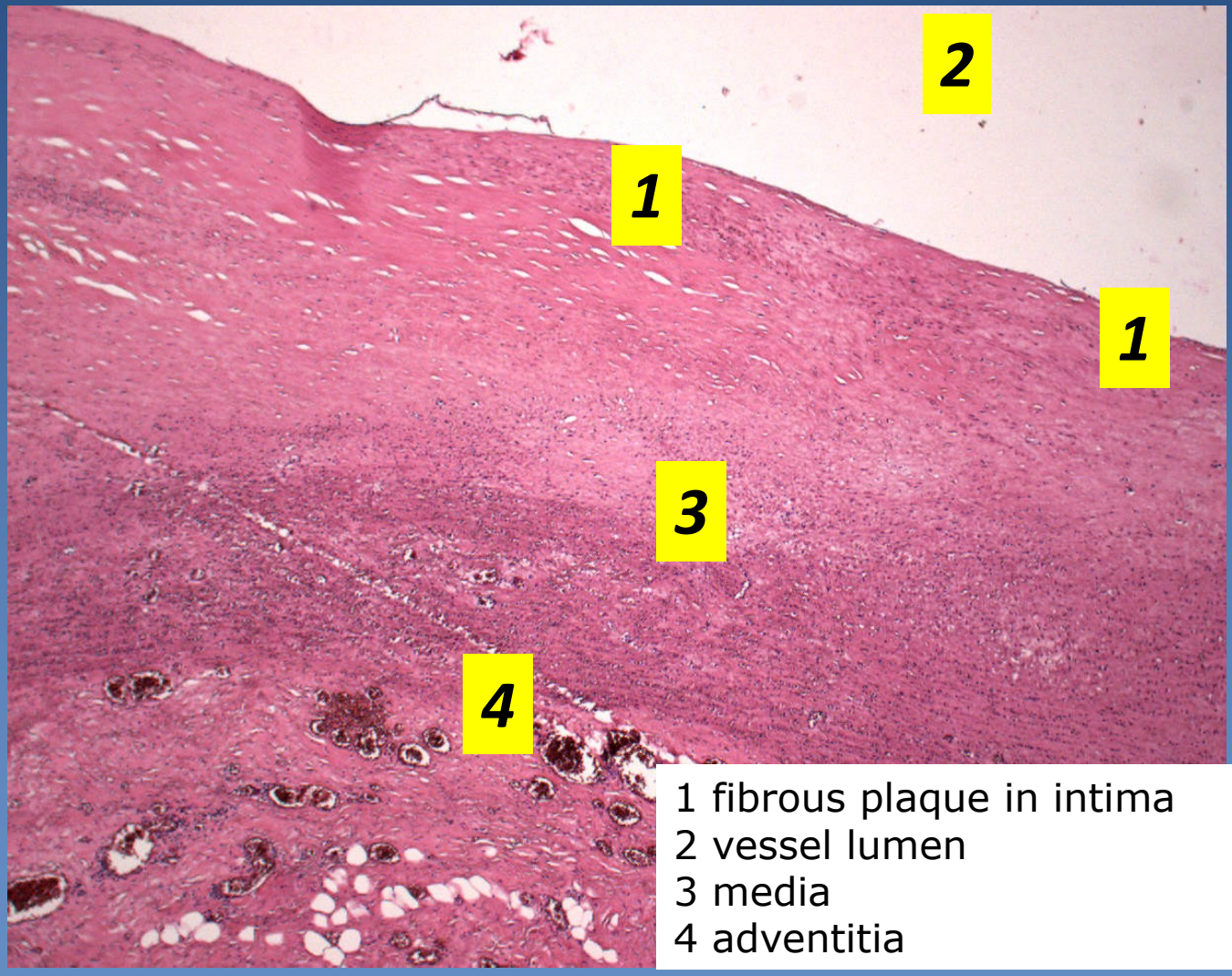


Atherosclerosis–
coronary artery



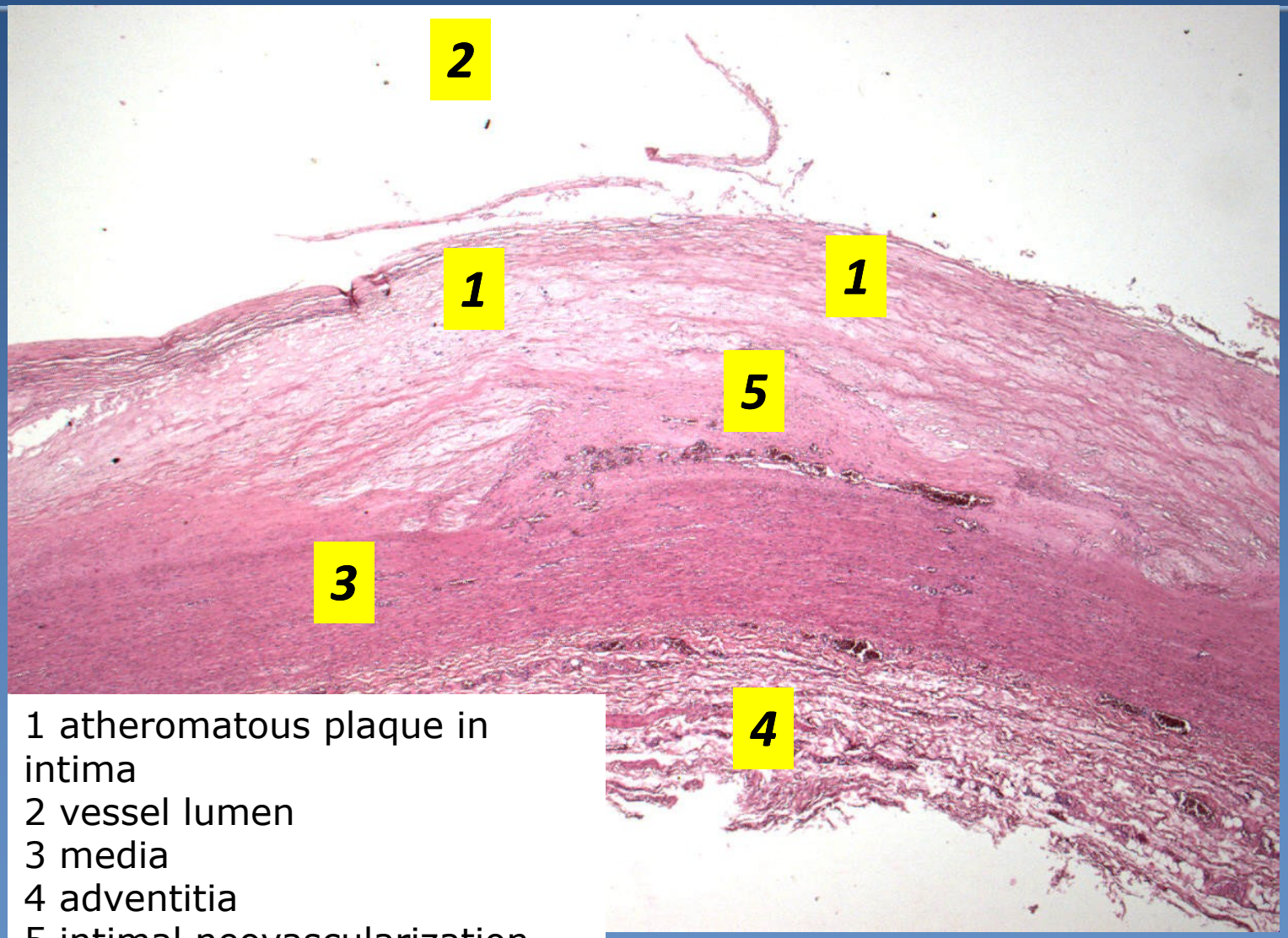
1 lumen narrowing
2 intimal atheromatous plaque
3 undamaged wall
4 epicardial fat tissue

Atherosclerosis – fibrous plaque



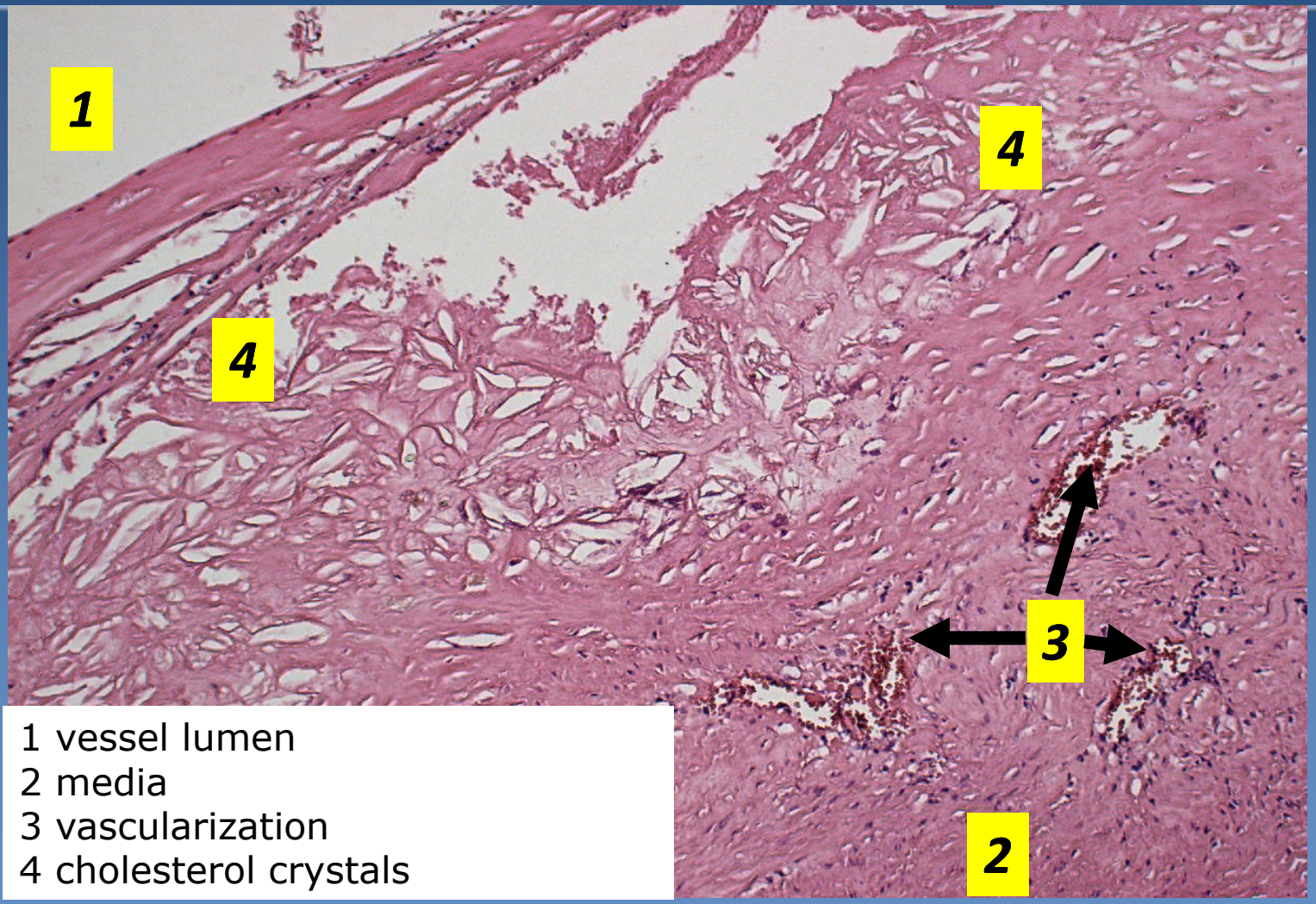
1 fibrous plaque in intima
2 vessel lumen
3 media
4 adventitia

Atherosclerosis – atheromatous plaque



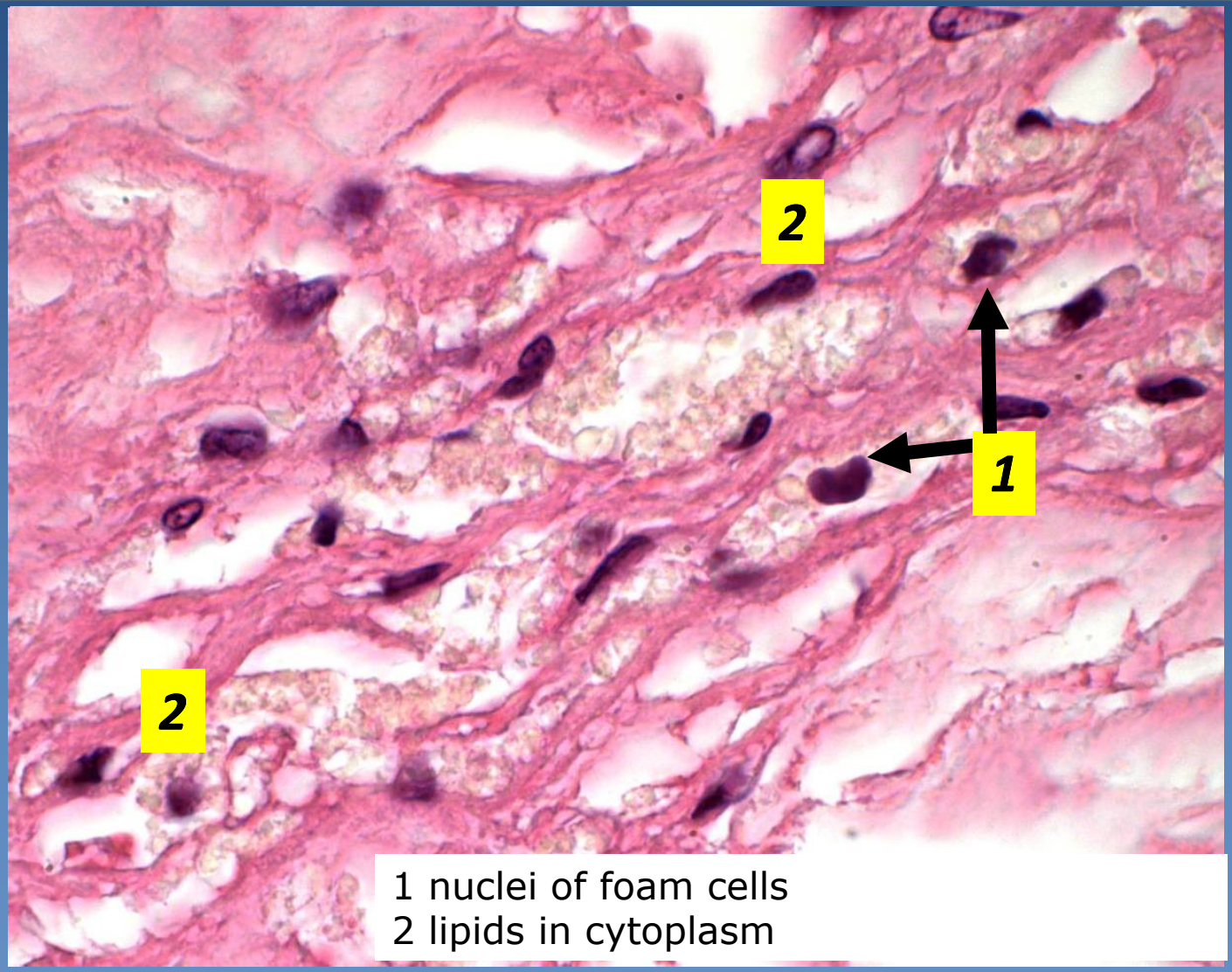
1 atheromatous plaque in intima
2 vessel lumen
3 media
4 adventitia
5 intimal neovascularization

Atherosclerosis – atheromatous plaque, intimal neovascularization



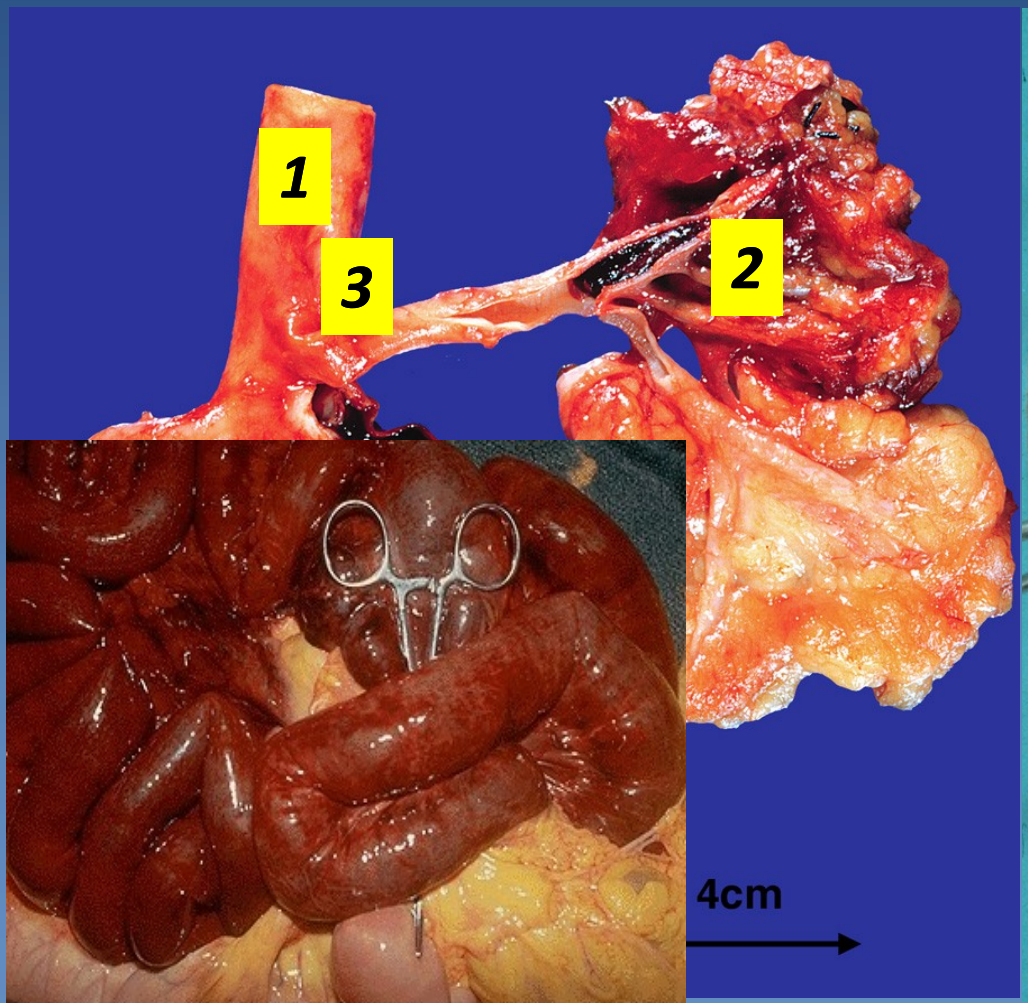
1 vessel lumen
2 media
3 vascularization
4 cholesterol crystals

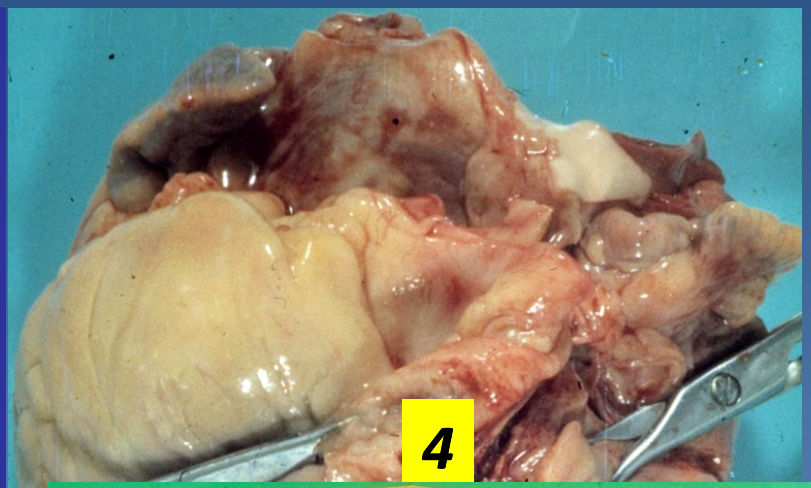
Atherosclerosis – foam cells in atheromatous plaque

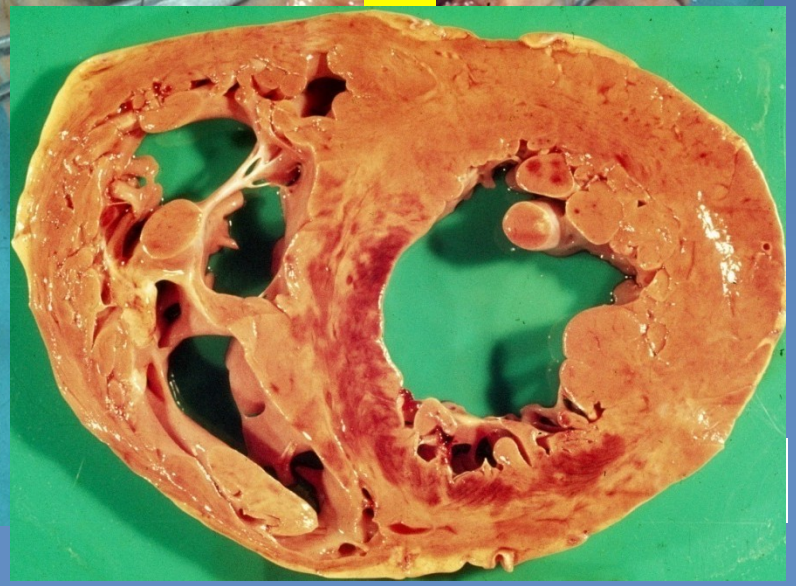


1 nuclei of foam cells
2 lipids in cytoplasm

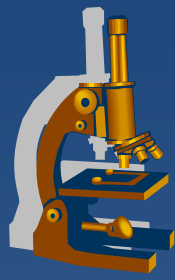
Atherosclerosis – complications
thrombosis/thrombembolia





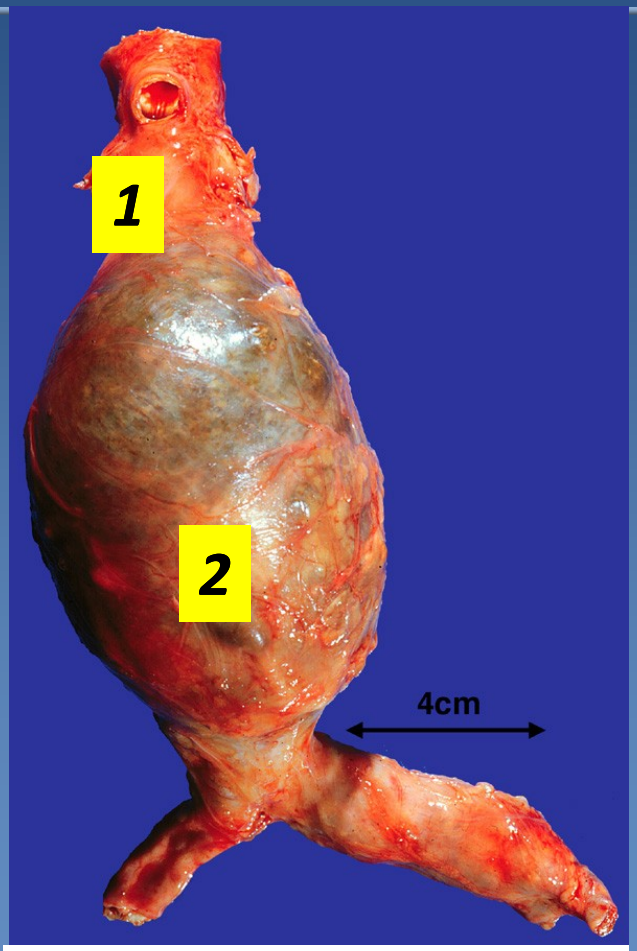


aneurysm

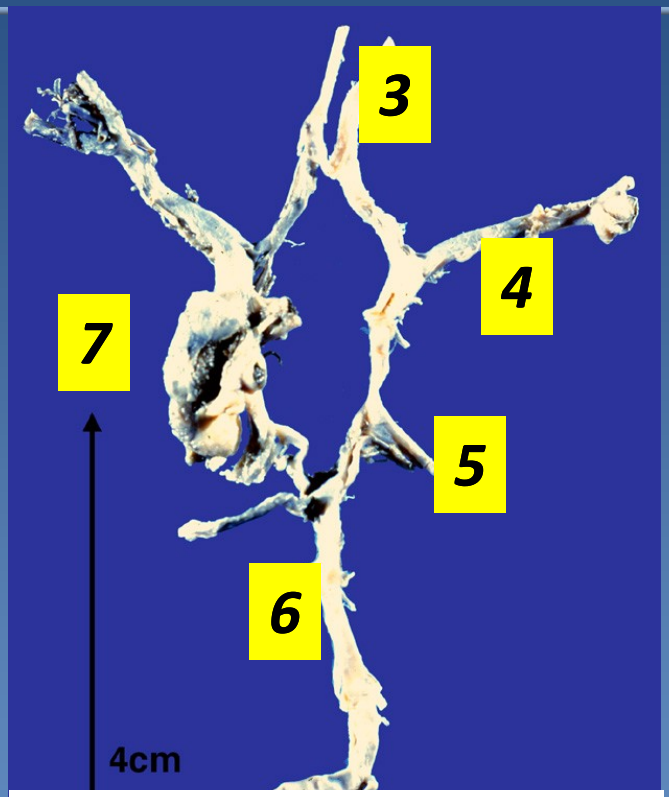


- localized, blood-filled balloon-like bulge in the wall of a blood vessel.
  - ⇨ *the circle of Willis in the brain, thoracic and abdominal aortic aneurysm*
- atherosclerotic aneurysm x syphilitic
- etiology:
  - ⇨ *hereditary defects in the structure, atherosclerosis, inflammation, disease process, accidents …*
- false aneurysm
- serpentine aneurysm, arteriovenous aneurysm

Atherosclerosis – complications– aneurysm

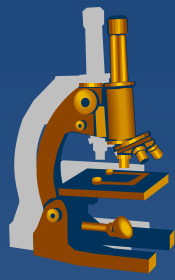


1 abdominal aorta
2 aneurysm



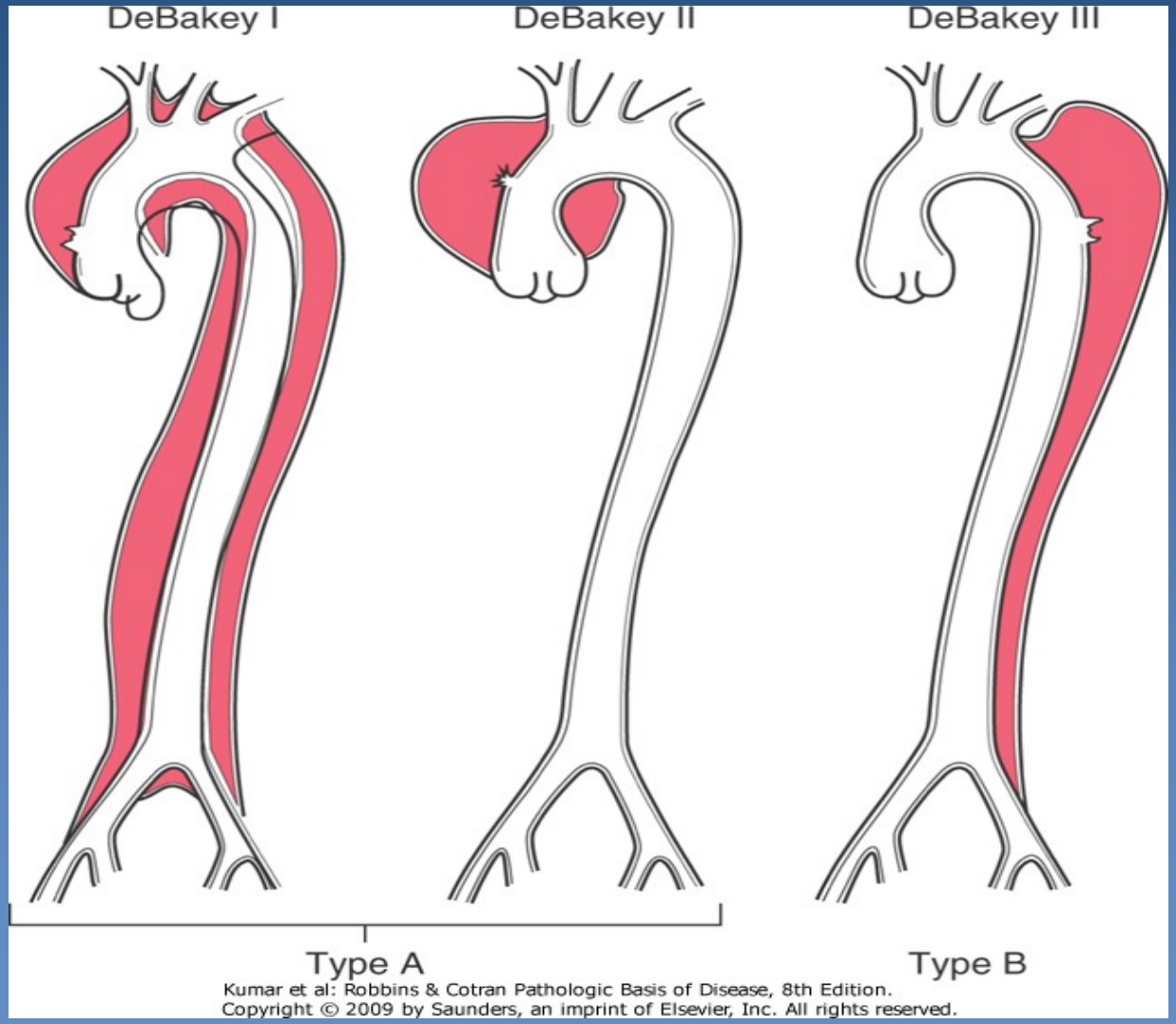
3 a. cerebri anterior
4 a. cerebri media
5 a. cerebri posterior
6 a. basilaris
7 aneurysm

Aortic dissection

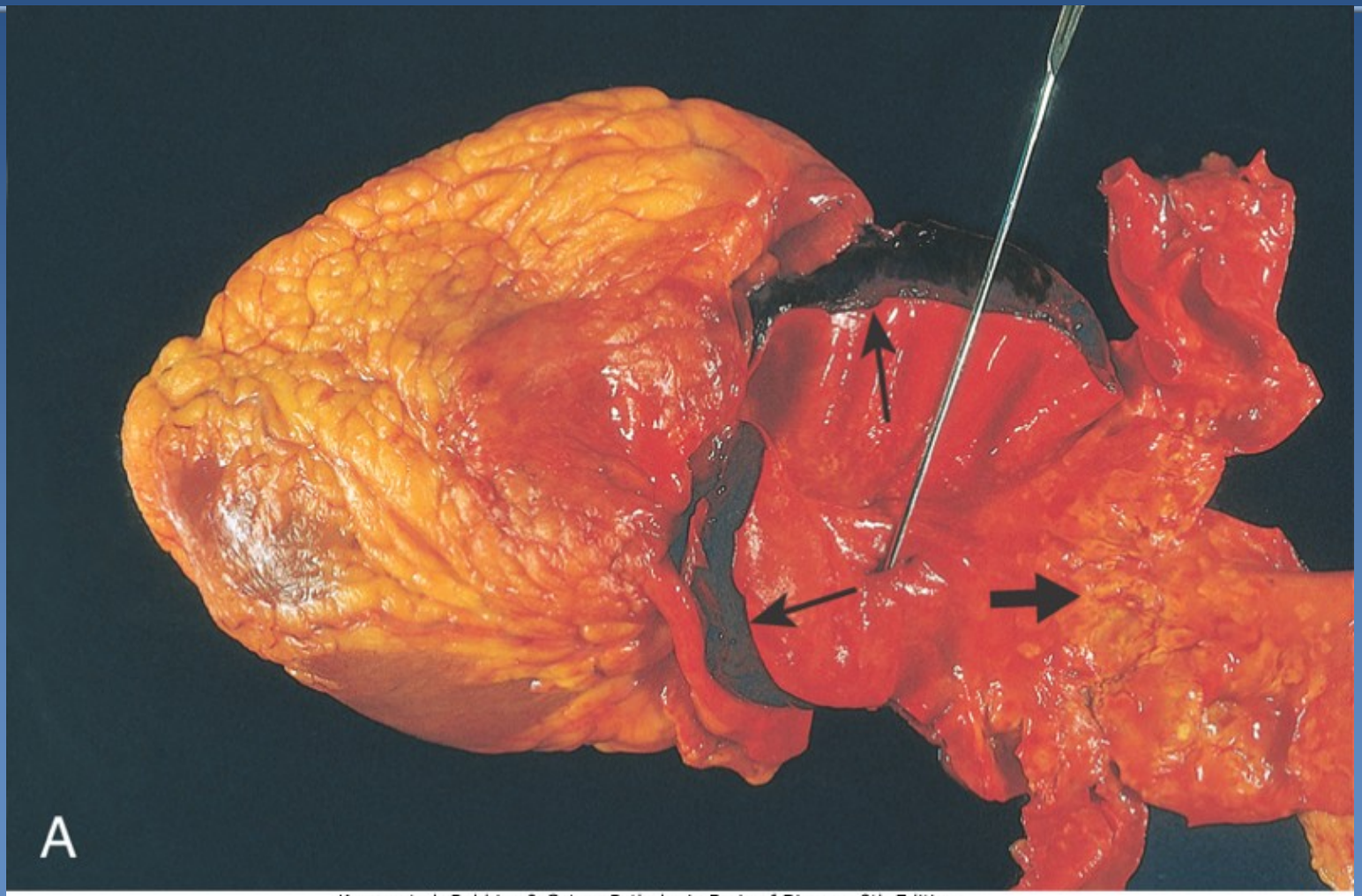


- tear in aortic intima - intramural bleeding through media, false lumen, possible „double-barreled“ aorta
- typic in ascending aort, 1–8 cm above aortic valve
- ante– and retrograde spread to the aortic root
- common thrombosis in false lumen
- risk of external rupture (➔ hemoperikardium), progression at the aortic branches (➔variable organ‘s ischemia), heart failure
- predisposition – hypertension, Marfan sy, cystic medial necrosis, …

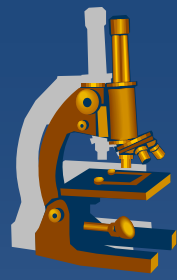
Aortic dissection



Aortic dissection

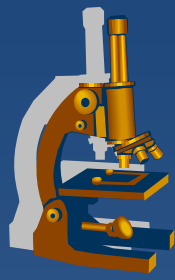


VASCULITIS



- Vessel wall inflammation
- Classification according cause: infectious x non-infectious (commonly immune-mediated, ANCA+/ANCA-)
- Affected organs : all organs with vessels
- Type (size) of vessel involved: Large-vessel
  Medium-vessel
  Small-vessel

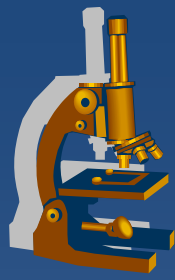
Vasculitis



- **ANCA^{+} vasculitis** (dangerous, even fatal within a few years, if not recognised)
  - ⇨ *Wegener granulomatosis*
  - ⇨ *Churg-Strauss syndrome*
  - ⇨ *microscopic polyangiitis*

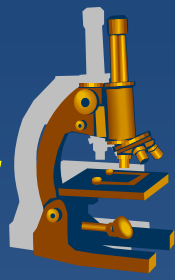
- **ANCA^{-} vaskulitis:**
  - ⇨ *polyarteritis nodosa*
  - ⇨ *Kawasaki disease*
  - ⇨ *giant-cell arteritis (Horton, temporal))*
  - ⇨ *Takayasu arteritis*
  - ⇨ *thrombangiitis obliterans (Bürger disease)*
  - ⇨ *leukocytoclastic (alergic) vasculitis – cca 30%*

Etiology



- **autoimmune process**
- **infection**
  - ⇨ *ie. streptococcus, …*
  - ⇨ *direct cause of infective v., or trigger factor of pathological immune processes*

Possible clinical signs of systemic vasculitis



ORL: **- repeated respiratory tract inflammation**
- exudate rich in plasma cells + eosinophils

Kidney: - glomerulonephritis

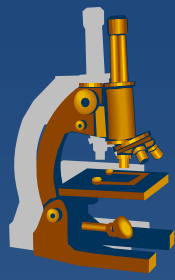
Lung: - variable presentation of lung diseases + hemoptysis

Skin: - ulceration, necrosis, petechiae-purpura

GIT: - ischemic ulcerations (sharply demarcated, without HP, minimal inflammation)

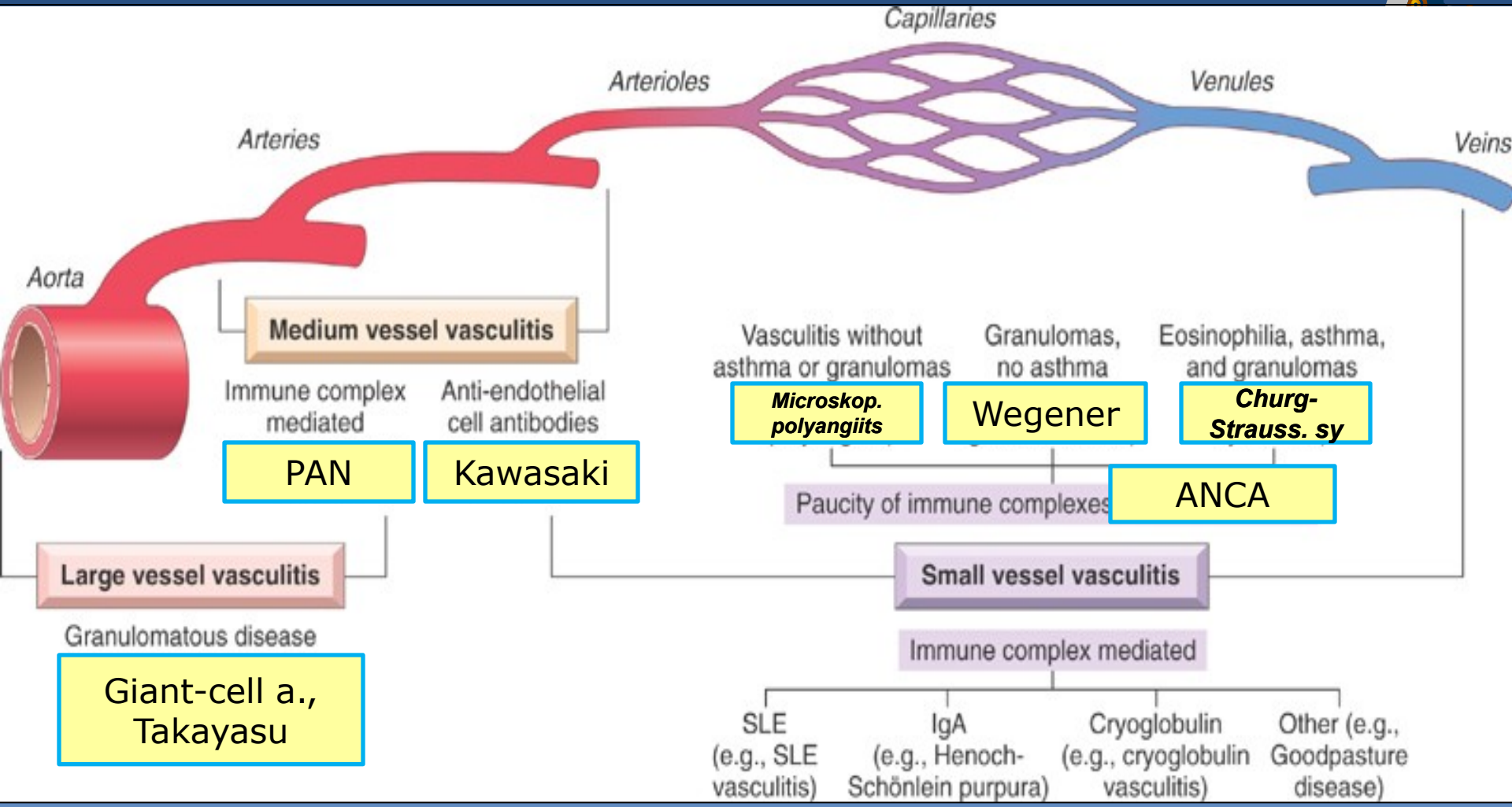
Chronic debilitating disease – clinical signs of tumor!!

Patient presentation

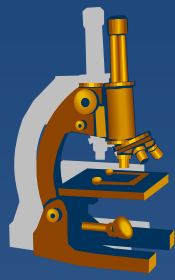


- **fever, nausea, myalgia, arthralgia**
- **skin purpura**
- **signs of nephritis**
- **abdominal pain**

general malaise (~ severe influenza, long duration, resistant to usual therapy)
sinusoid course (relapse --- remission --- relapse--)


Capillaries
Arterioles
Venules
Arteries
Veins
Aorta
Medium vessel vasculitis
Immune complex mediated
Anti-endothelial cell antibodies
PAN
Kawasaki
Vasculitis without asthma or granulomas
Granulomas, no asthma
Eosinophilia, asthma, and granulomas
Microskop. polyangiits
Wegener
Churg-Strauss. sy
Paucity of immune complexes
ANCA
Large vessel vasculitis
Granulomatous disease
Giant-cell a., Takayasu
Small vessel vasculitis
Immune complex mediated
SLE (e.g., SLE vasculitis)
IgA (e.g., Henoch-Schönlein purpura)
Cryoglobulin (e.g., cryoglobulin vasculitis)
Other (e.g., Goodpasture disease)

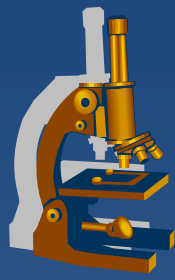
ANCA+ vasculitis



- incidence ????
  - ⇨ ***≤20/1mil. inhabitants***
  - ⇨ ***age 65+ - 53/1mil. inhabitants***

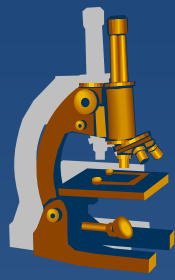
- prognosis:
  - ⇨ *untreated ANCA$^+$ vasculitis ≥80% fatal in 2 yrs*
  - ⇨ *treated ANCA$^+$ vasculitis : ≥80% survives 5 yrs*
  - ⇨ *renal failure in elders >70 yrs - in 40% due to ANCA$^+$ vasculitis*

granulomatosis with polyangiitis (Wegener granulomatosis)

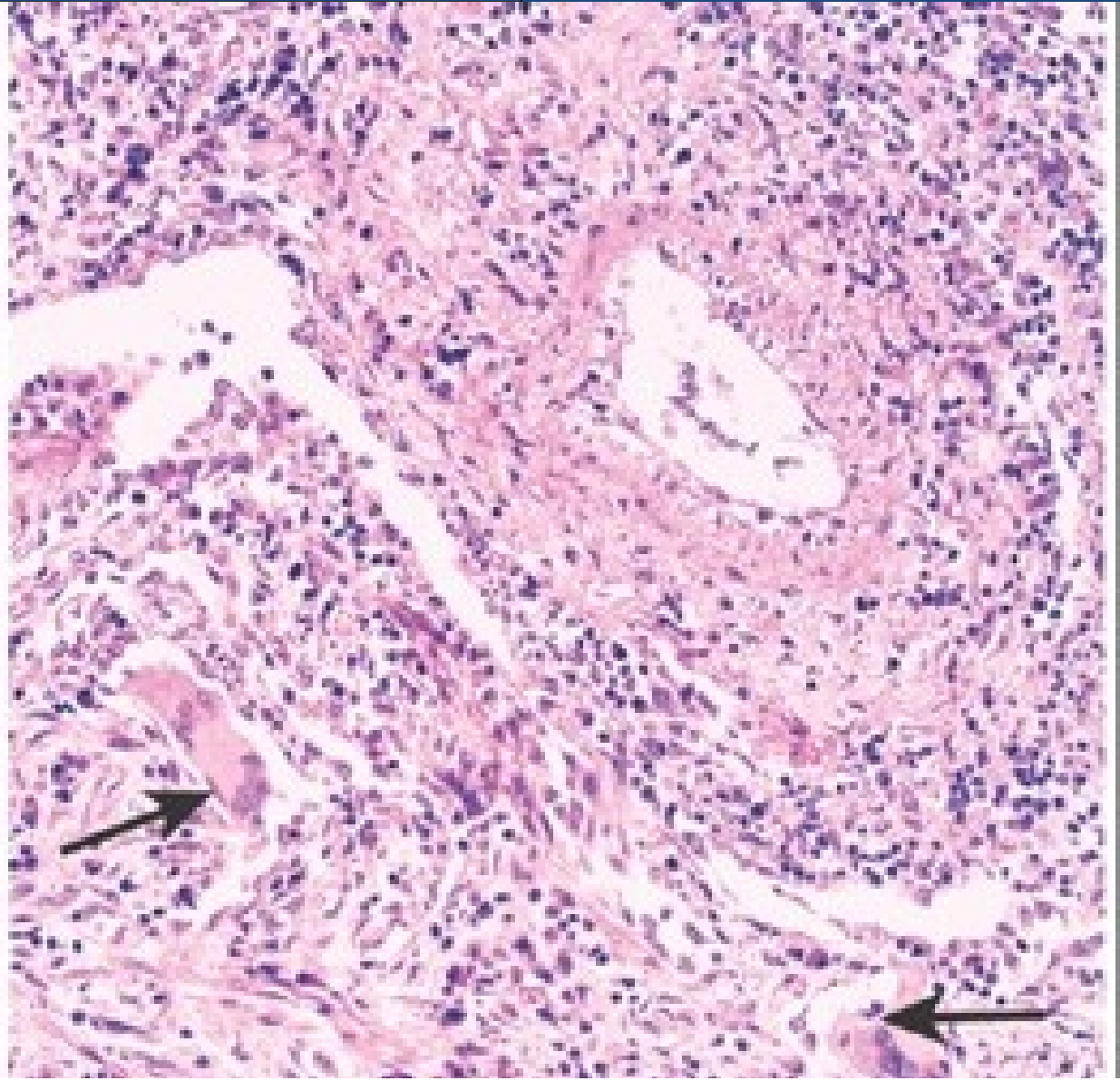


- clinically as pneumonitis, persistent X-ray with bilat. nodular infiltrates, chronic sinusitis with mucosal ulcerations nasopharynx (sometimes destructive axial structures), ARI / CHRI (focal necrosis, sickle cell GLN)

granulomatosis with polyangiitis (Wegener granulomatosis)

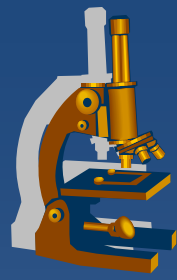


- persistent pneumonitis (95%) – nodular infiltrates
- chronic sinusitis (90%) – ulcerations, event. Destructive
- renal disease (80%) – glomerulonephritis
- other features: rashes, muscle pains, articular involvement, mono-/polyneuritis



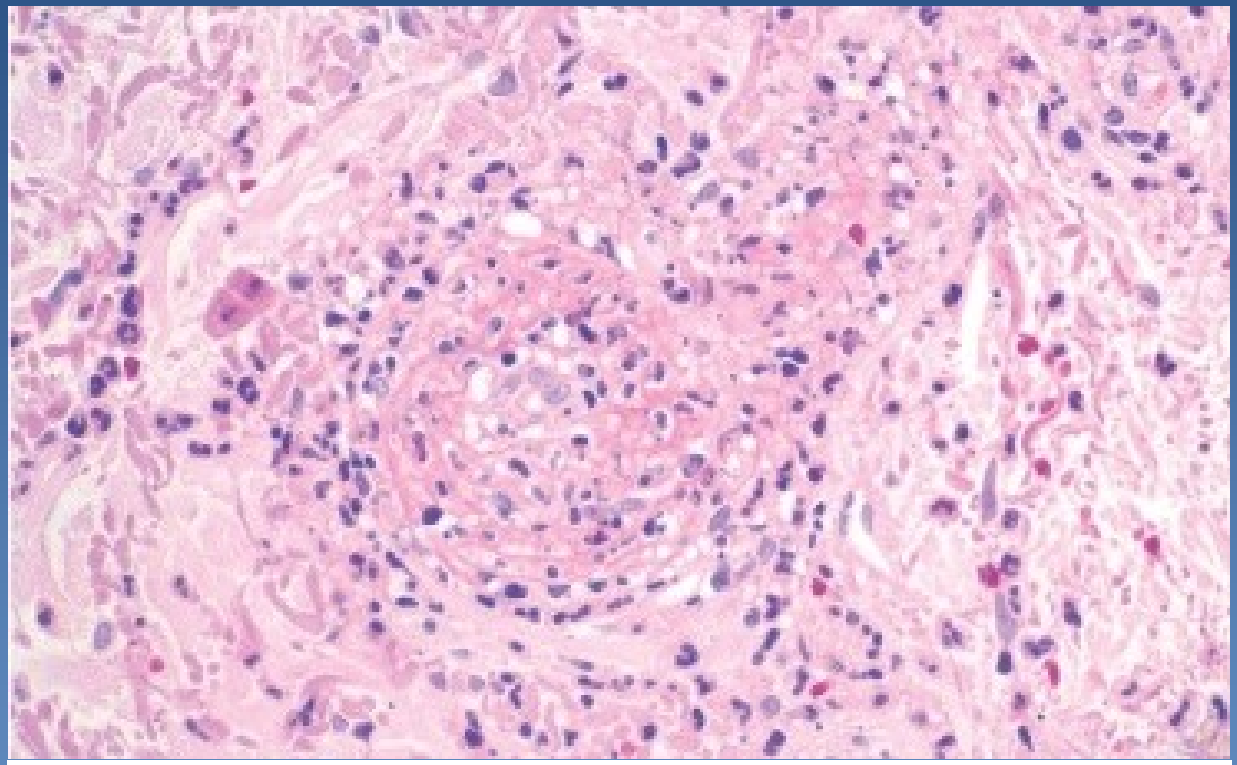
Small vessel vasculitis with giant-cell granulomatous reaction

ANCA+ VASCULITIS:
microscopic polyangiitis



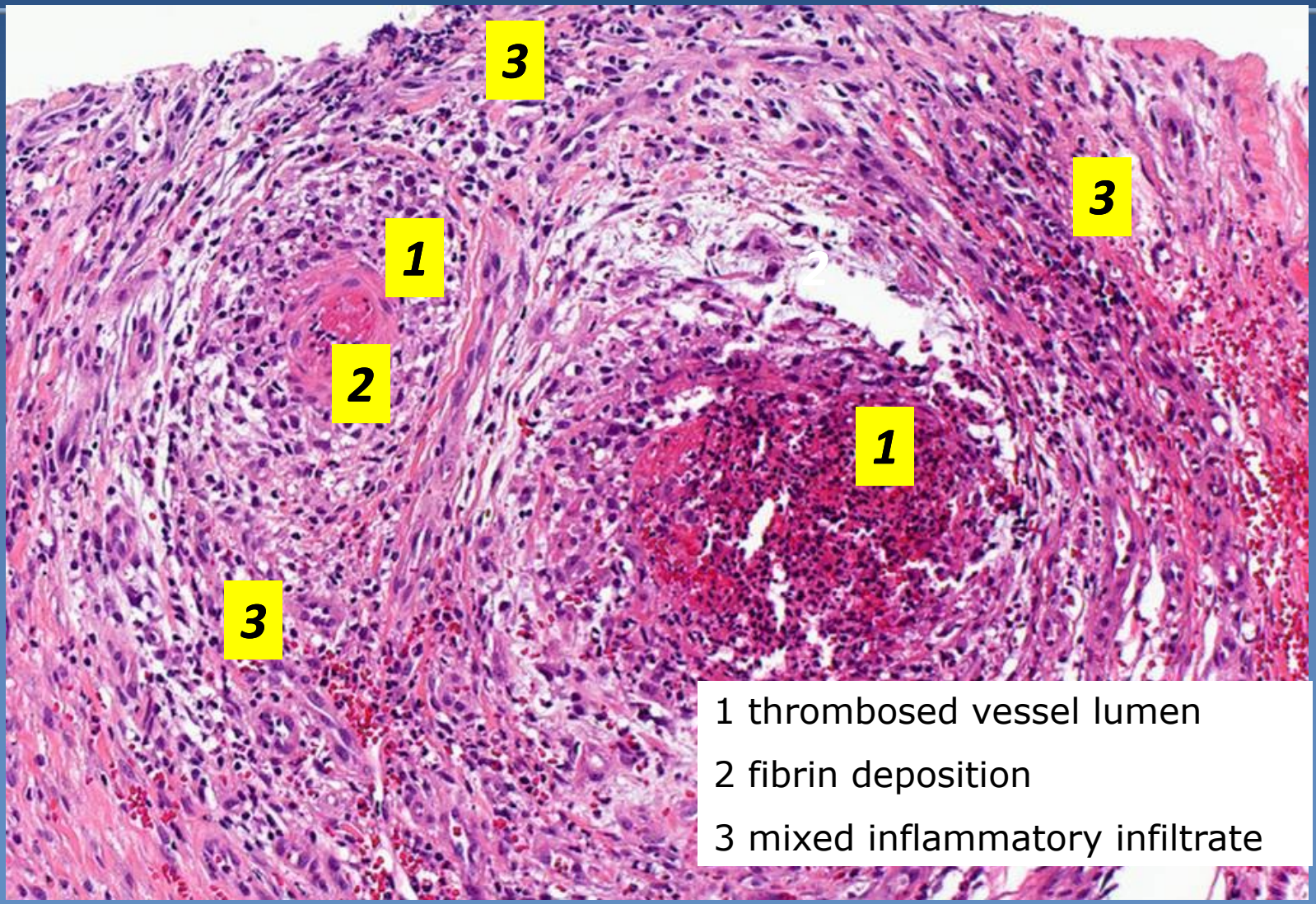
- **ANCA** in approx. **70%** (remaining by immunecomplexes or antibodies)
- = **necrotizing vasculitis** arterioles, capillaries, venules (synonyms: leukocytoclastic v., hypersensitive v., allergic v.)
- : **SKIN**, kidney, lung, GIT, brain…
- highly variable etiopathogenesis (part of systemic connective tissue diseases; alergic response to exogennous antigens – bacteria, viruses, drugs)
- micro:
  - ⇨ *fibrinoid necrosis of vessel wall with neutrophils and chromatin fragments from neutrophil‘s nuclei - leukocytocllastic)*
  - ⇨ ***all lesions in the same stage of evolution (X polyarteritis nodosa)***

leukocytoclastic vasculitis



nuclear fragments from neutrophils in a small vessel wall

polyarteritis nodosa

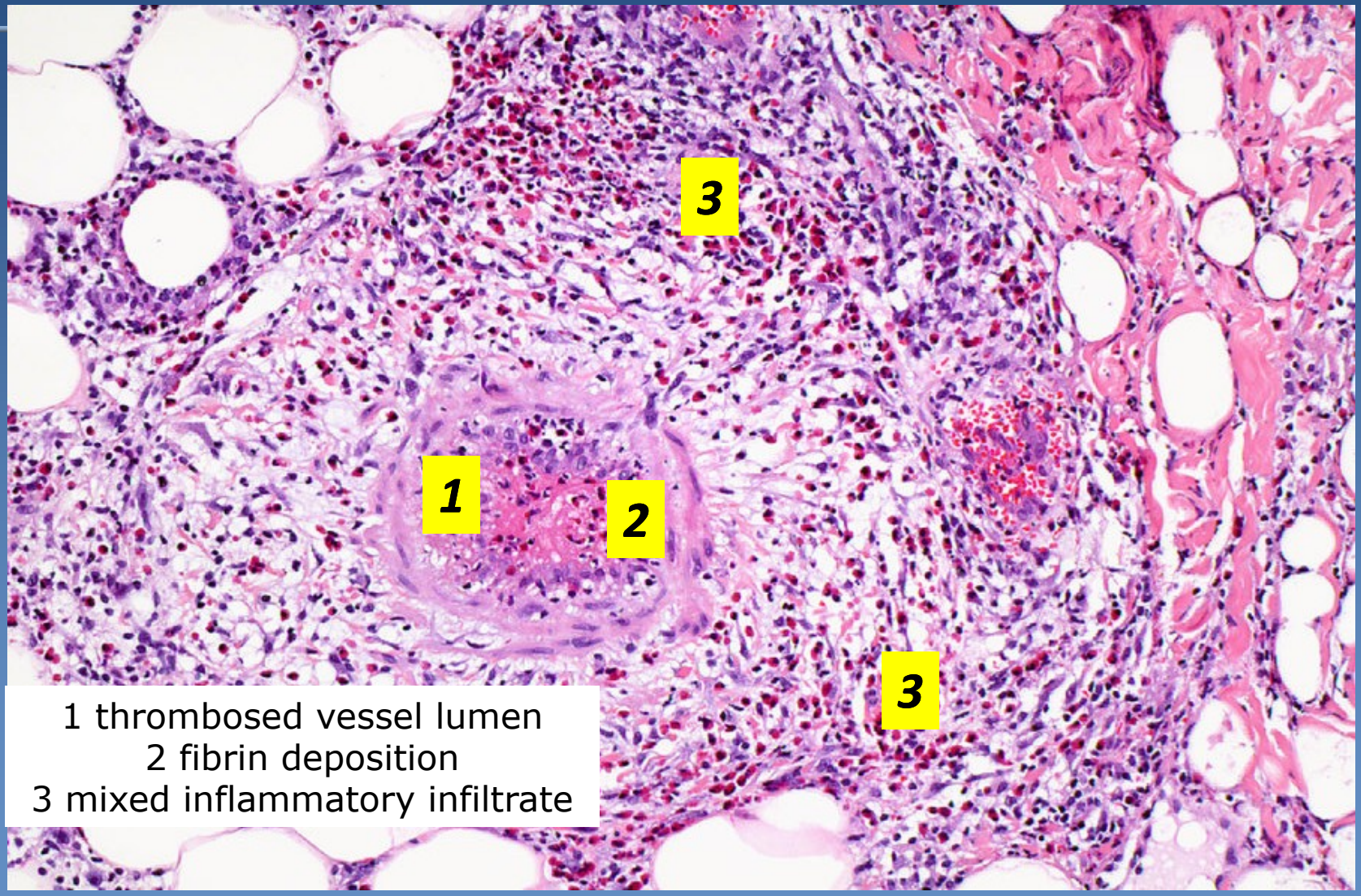


1 thrombosed vessel lumen

2 fibrin deposition

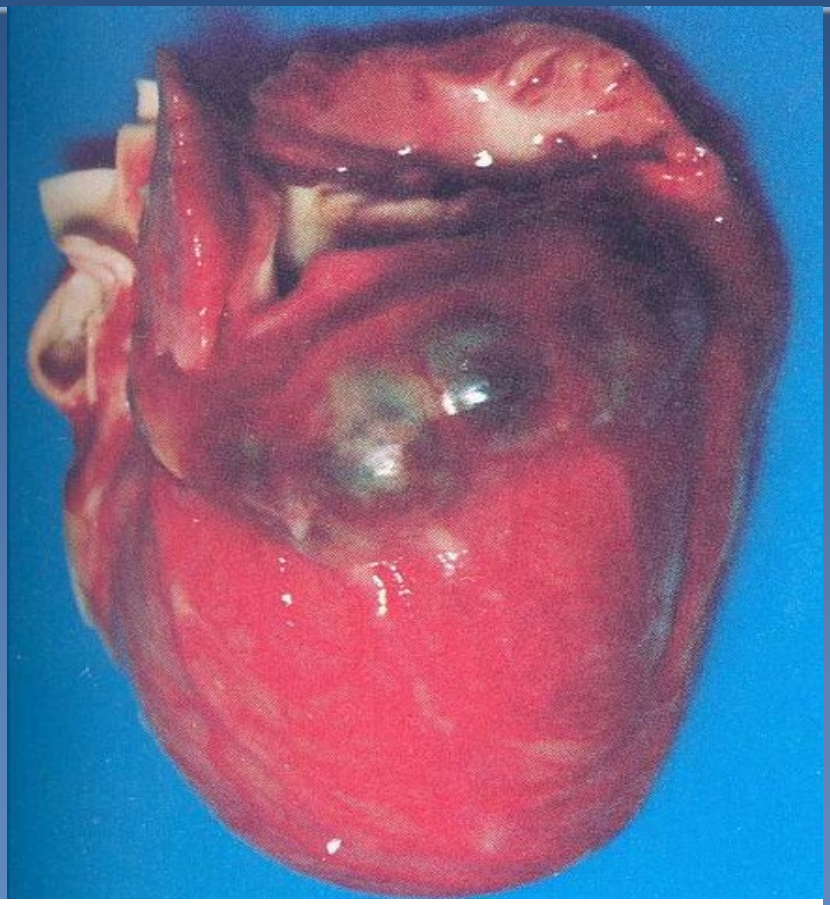
3 mixed inflammatory infiltrate

polyarteritis nodosa



1 thrombosed vessel lumen
2 fibrin deposition
3 mixed inflammatory infiltrate

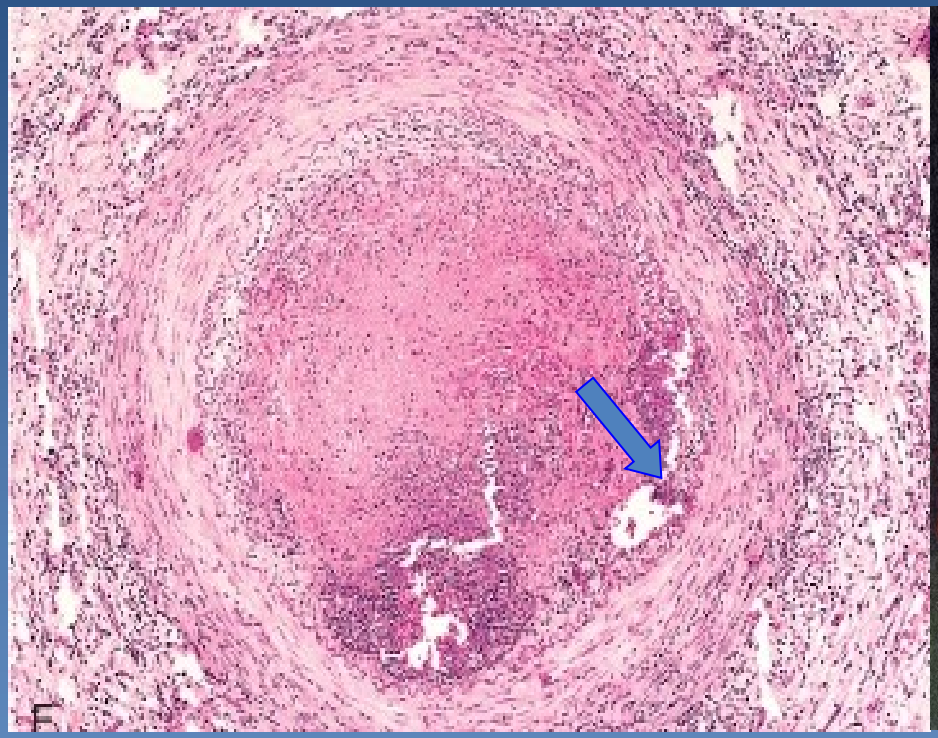
Kawasaki disease



Coronary aneurysms in a child‘ heart

coronary artery with lamina elastica interna defects (arrows) and thrombotized aneurysms

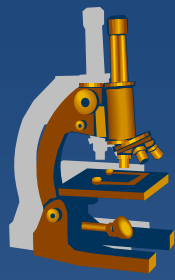
Thrombangiitis obliterans (Bürger disease)



Obliterative thrombosis with granuloma with central microabscess (arrow)

acral necroses

infectious vasculitis

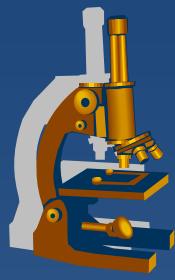


- rare

- arise:
  - ⇨ ***transfer of infection from surrounding tissues***
  - ⇨ ***infected emboli during pyemia***

- examples:
  - ⇨ ***aortitis luetica***
  - ⇨ ***bacillary angiomatosis = opportunistic infections (eg AIDS)??***

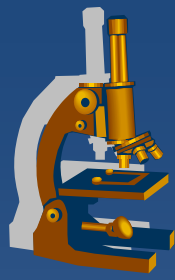
Infectious vasculitis



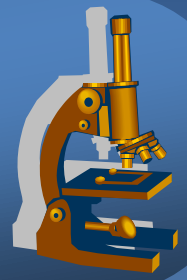
- direct invasion of vascular wall by inf. pathogen

- primary angioinvasive microorganism
  Fungi: Aspergillus, Mucor - thrombosis→ ischemic necrosis

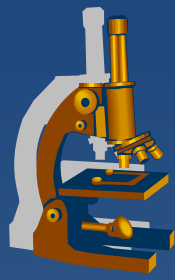
- secondary vasculitis - localized vasculitis in focal infection
  - ⇨ ***purulent*** – *meningitis*
  - ⇨ ***pneumonia***
  - ⇨ ***abscess, fasciitis*** – *pyogenic bacteria*
  - ⇨ ***granulomatous***
    - obliterative endarteritis – TBtertiary syphilis, l
    - Lepra
  - ⇨ ***lymphocytic vasculitis*** – *rickettsia (spotted fever, Q fever etc.)*
  - ⇨ ***recurrent herpes, CMV***
  - ⇨ ***necrotizing vasculitis*** – *anthrax*



Cardiac pathology



Morphology



- pericardial sac – cca 30ml clear yellowish fluid

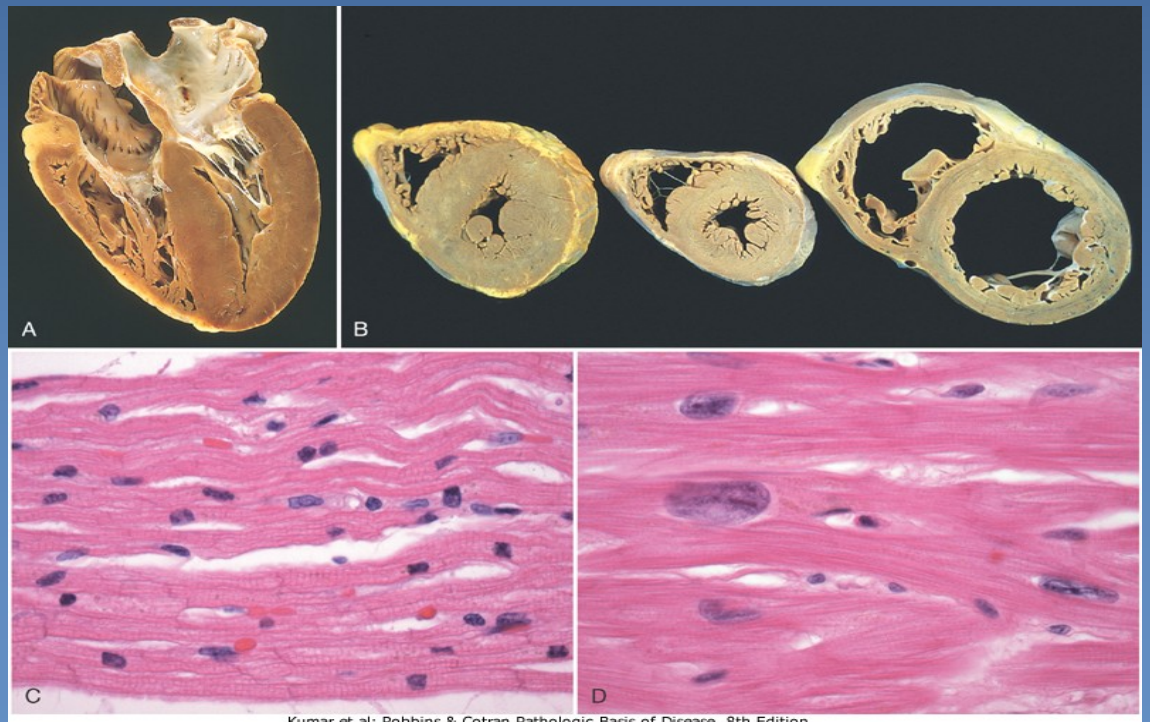
- **male = 300 – 350 g**,
  - *hypertrophy > 400g*

- **myocardium:**
  - ➔ RV 3 – 4 mm
  - ➔ LV 12 – 15 mm

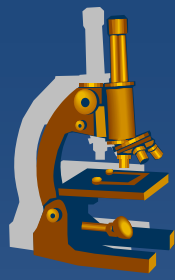
- **foramen ovale**
  - *closed x opened ➔ paradoxical embolia*

Systemic hypertension and heart

- *90–95% essential , risk factor for AS*
- *work overload ➔ LV adaptation to ↑peripheral resistance = **cor hypertonicum** (concentric LV hypertrophy) ➔ limited compensatory mechanisms ➔ **cor hypertonicum decompensatum** (dilatation of hypertrophic LV)*
- *➔ heart insufficiency ← relative coronary incompetence*



Heart failure

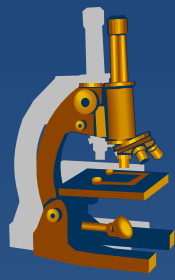


- heart unable to pump blood at a rate sufficient for metabolic demands of the tissues

- systolic dysfunction - ↓ myocardial contractile function (ischemic injury, pressure or volume overload – valvular disease, hypertension, cardiomyopathy
- diastolic dysfunction - inability to dilatate sufficiently (massive LV hypertrophy, myofibrosis, amyloidosis)

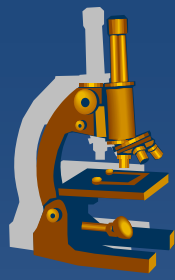
- cardial – extracardial pathologic changes

Heart failure



- failure of normal pumping action of the heart
- failure of forward and backward ➔ to cardiogenic shock
- manifestations of the heart and heart out

Cardial changes

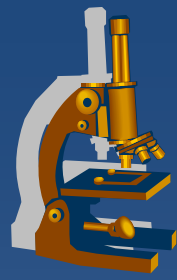


- **disproportion between heart function and peripheral vascular resistance**

- differ according rapidity of development:
  – sudden ➔ *acute dilatation*

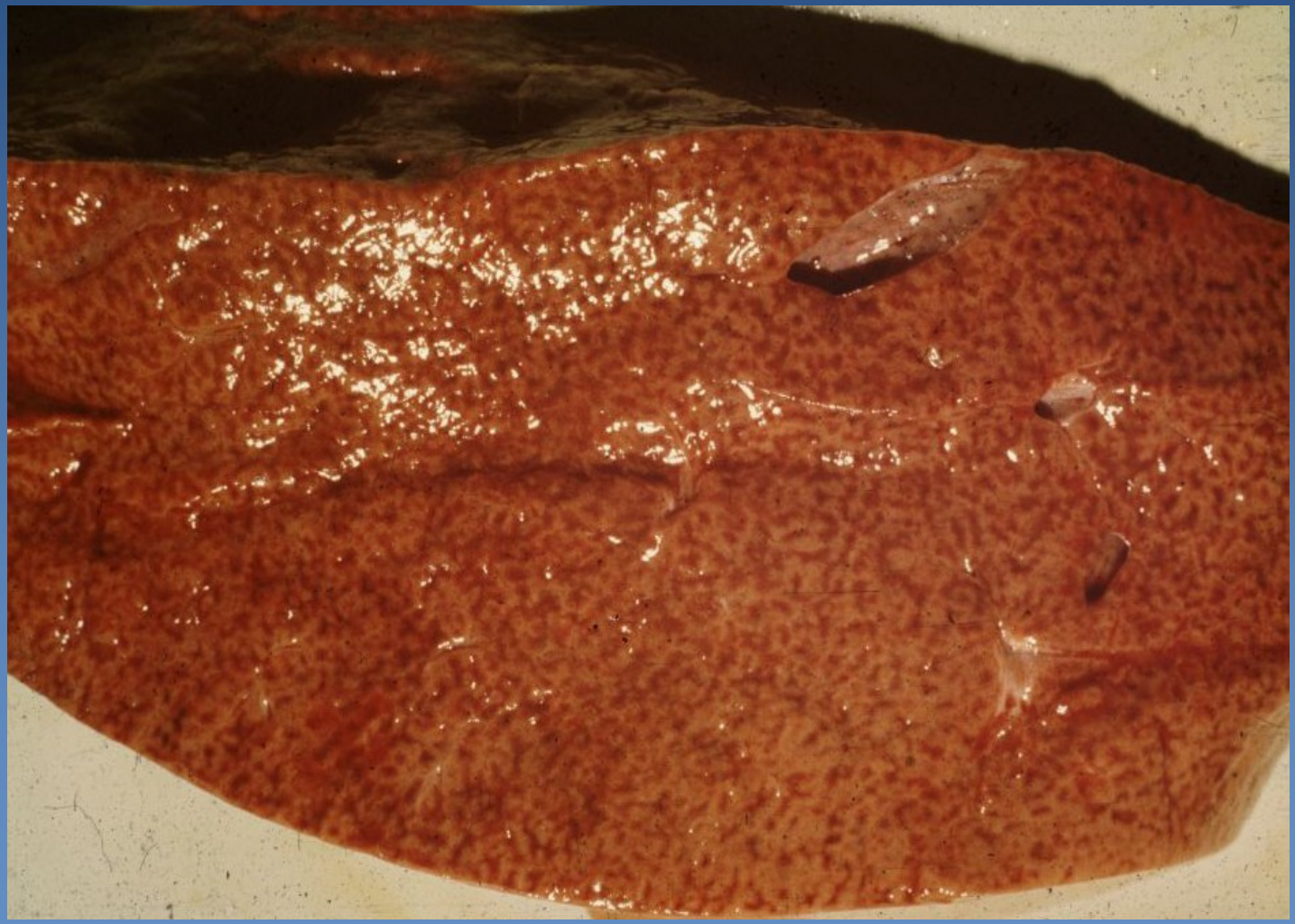
  – chronic ➔ *adaptation* ➔ ➔ ➔
  myocardial hypertrophy (↑ nutritional demands) +/- ventricular dilatation (enhanced contractility – Frank-Starling mechanism), + activation of neurohumoral systems (norepinephrin, renin-angiotensin sy, atrial natriuretic peptide

Extracardial changes

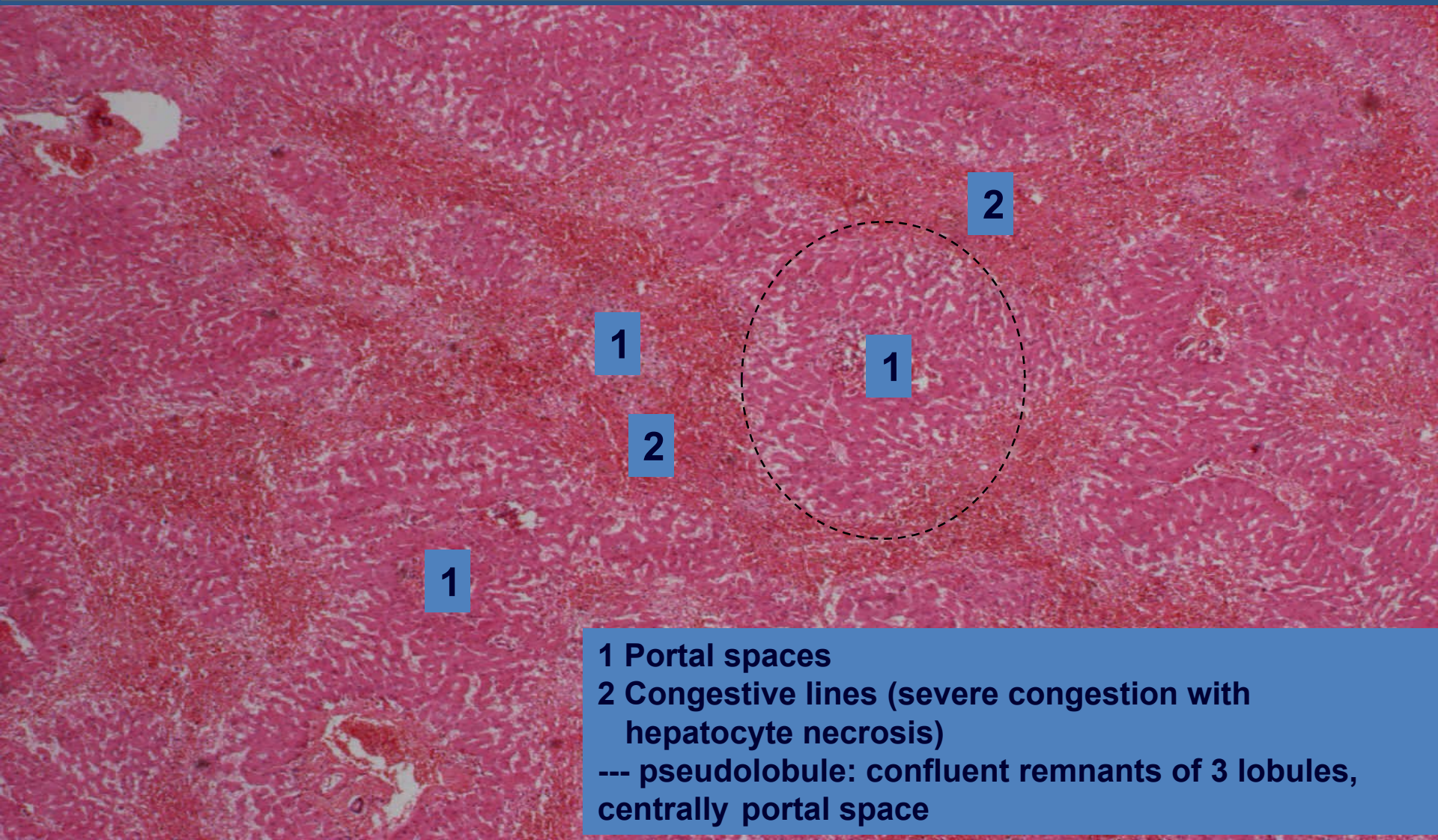


- **venoous congestion** – *e.g. liver (-> hepar moschatum)*
- **induration** – *fibroproduction (liver, spleen, kidney)*
- **oedema** –
- **cyanosis** – *visible on acral parts*

Chronic venous congestion (nutmeg liver - hepar moschatum)

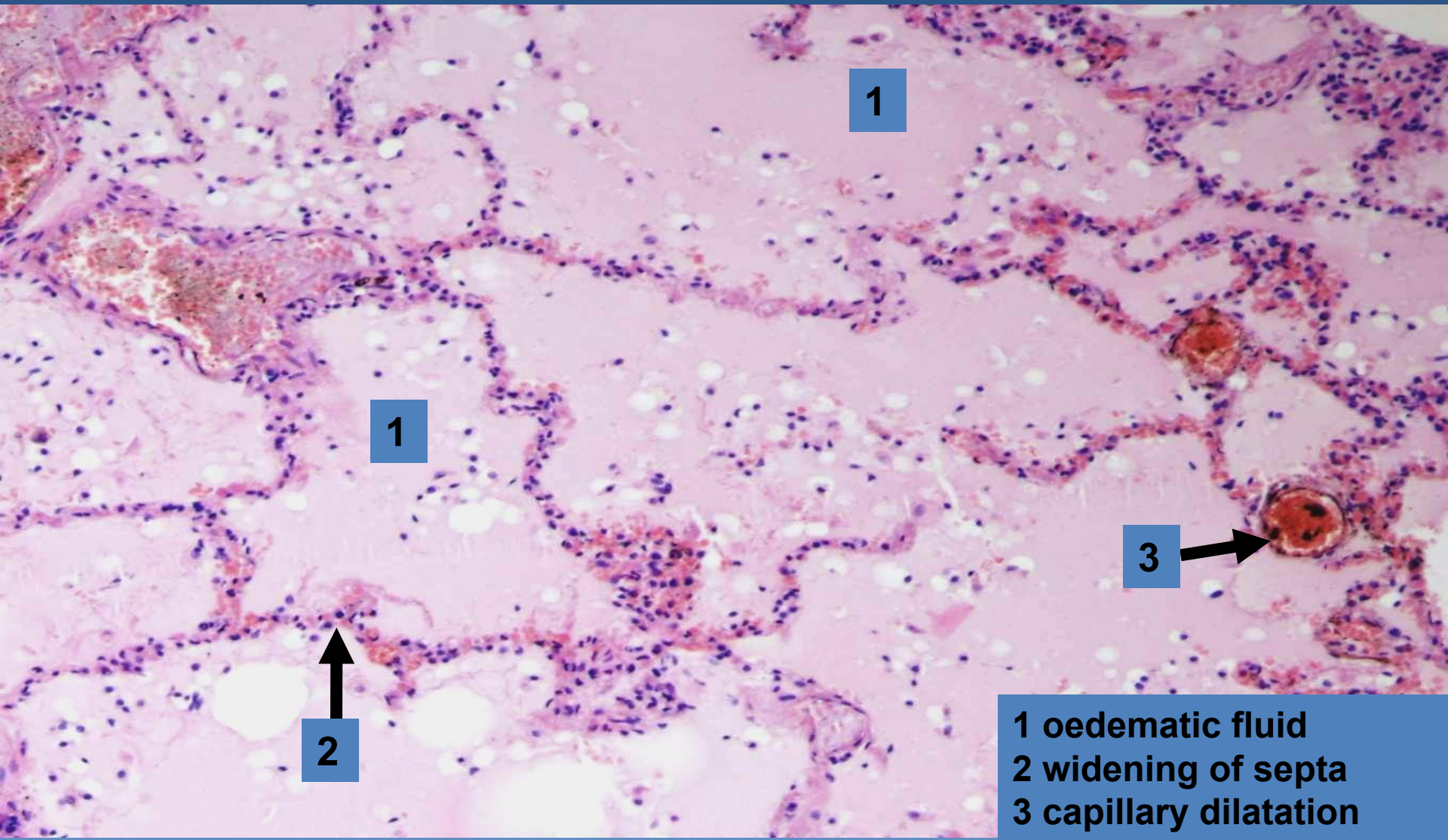


Hepatic venous congestion

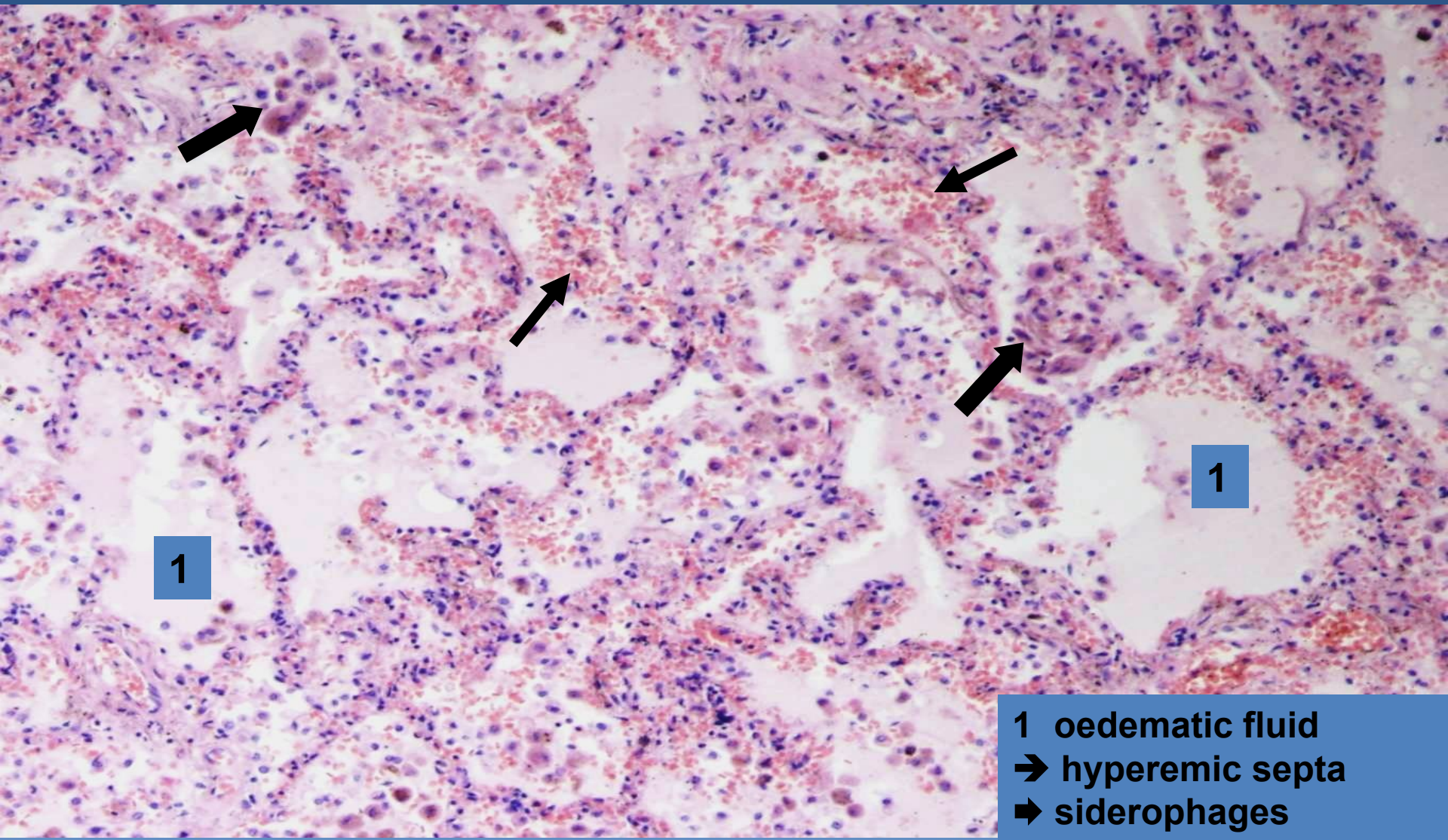


1 Portal spaces
2 Congestive lines (severe congestion with hepatocyte necrosis)
--- pseudolobule: confluent remnants of 3 lobules, centrally portal space

Pulmonary oedema

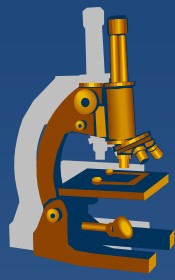


Chronic pulmonary venous congestion



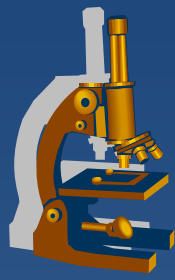
1 oedematic fluid
➔ hyperemic septa
➡ siderophages

Ischemic heart disease (IHD)



- group of pathophysiologically related syndromes resulting from **myocardial ischemia** (hypoxia or anoxia, ↓ nutrients, ↓ removal of metabolites)
- imbalance between the demand and supply by coronary arteries.
- important factor – coronary AS
- forms:
  - ⇨*angina pectoris*
  - ⇨*myocardial infarction (MI)*
  - ⇨ *chronic IHD with heart failure*
  - ⇨ *sudden cardiac death*

Pathogenesis of IHD

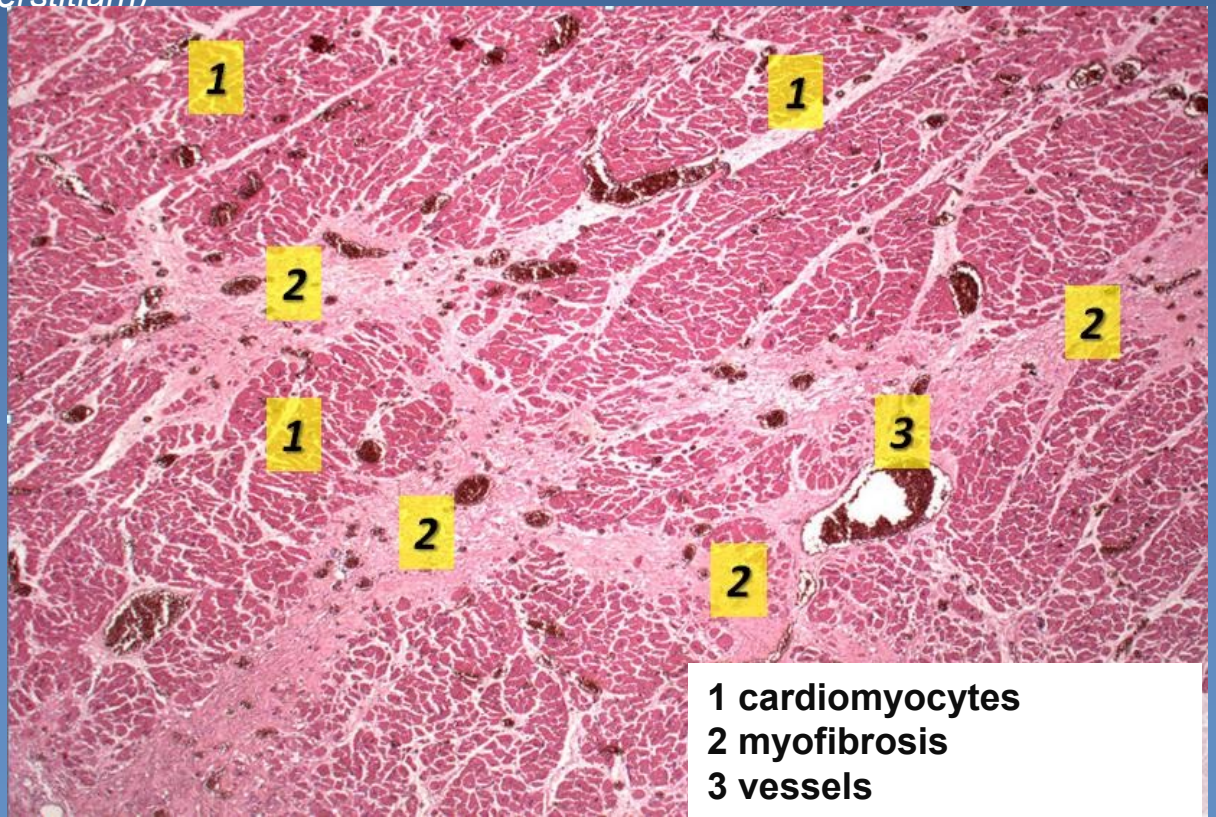


1) **AS of coronary aa.**
   - *– commonly at a. branching*
   - *– fixed obstruction by plaque (fibrous, atheromatic)*
   - *– acute plaque change (rupture, erosion, haemorrhage, thrombosis)*
   - *– 75% stenosis – ischemia during ↑ workload – stable angina pectoris*
   - *– 90% stenosis –ischemia even at rest – ustable angina - preinfarction*

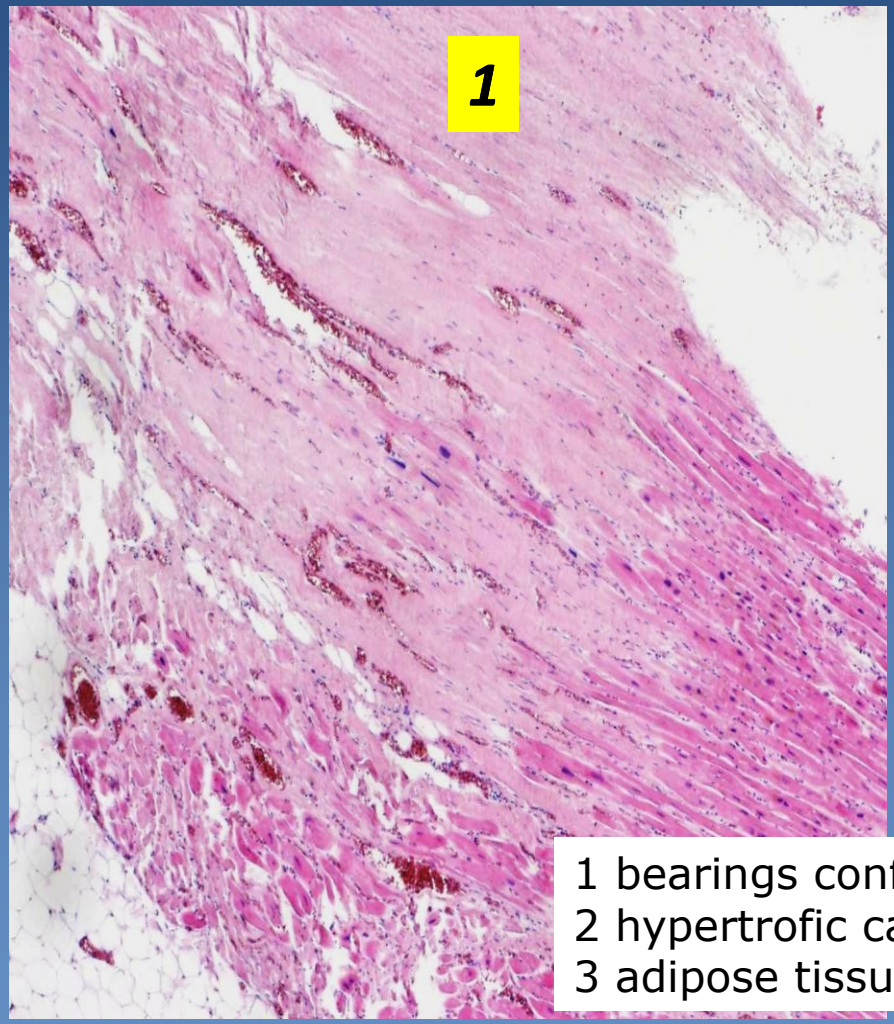
2) **non-atherosclerotic**
   - *– coronary emboli – endocarditis, atrial fibrillation, mural thr., paradoxical e.*
   - *– coronary vasospasm*
   - *– aortic dissection*
   - *– coronary vasculitis*
   - *– congenital coronary aa. defects*
   - *–hematologic disorders, amyloidosis, shock, etc.*

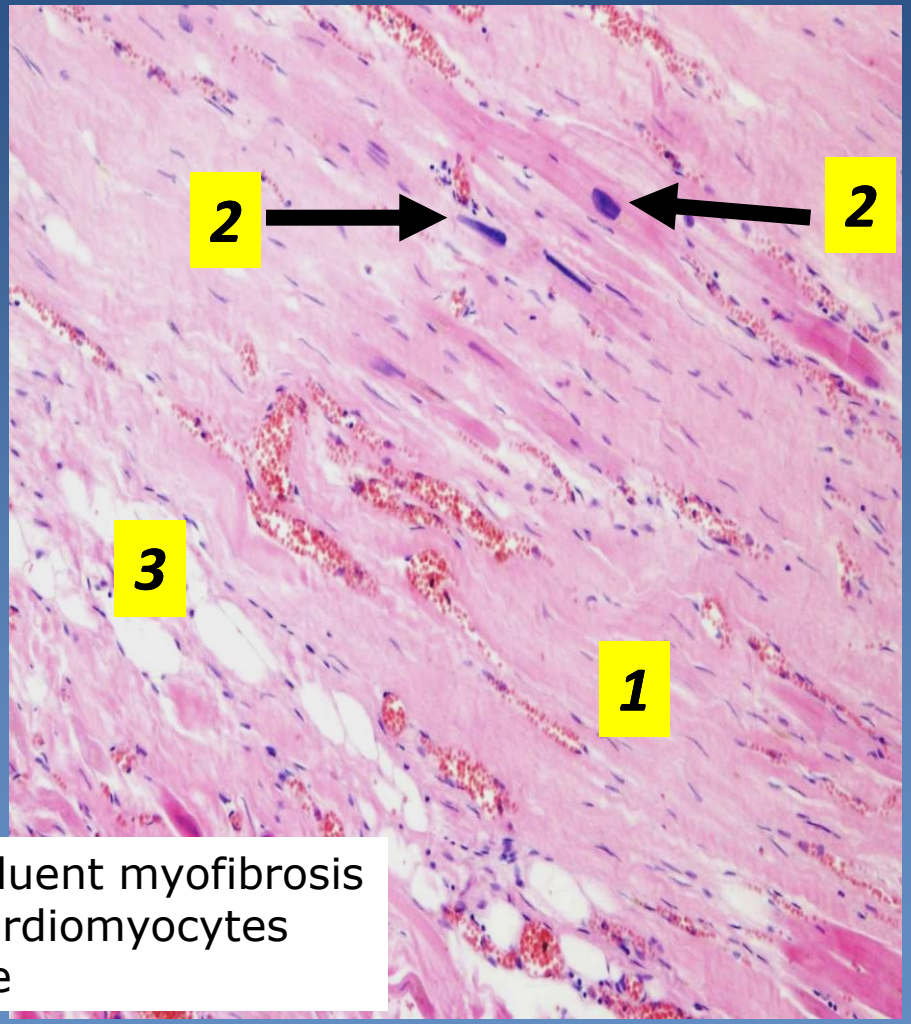
ischemic heart disease (IHS)

- Morphology of myocardial ischemia:
  - *steatosis myokardu*
  - *myomalatia (= partial necrosis – cardiomyocytes only)*
  - *dispersive /confluent myofibrosis*
  - *myocardial infarction: transmural/subendocardial (complete coagulative necrosis incl. interstitium)*



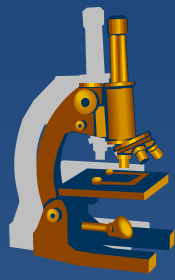
Confluent myofibrosis and myocard lipomatosis





1 bearings confluent myofibrosis
2 hypertrofic cardiomyocytes
3 adipose tissue

Angina pectoris (AP)



× **transient myocardial ischemia ➔ chest pain !!!**

1. stable (typical)

– due to increased workload, duration ≤ 15 min, relieved by rest or nitroglycerin

– no myocardial necrosis

–subendocardial LV myocardium

2. unstable

– increasing frequency / duration of pain attack, even at rest

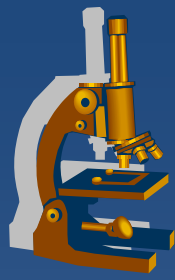
– plaque disruption + mural thrombosis, possible vasospasm

– preinfarction angina

3. variant (Prinzmetal) angina

– mostly unrelated to physical activity, coronary vasospasm - vasodilatative therapy

Myocardial infarction

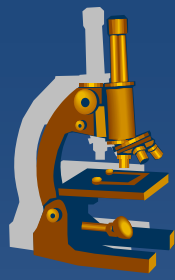


- **ischaemic coagulative necrosis**

- causes:
  - ⇨ *usually coronary thrombosis*
  - ⇨ *complicated atheromatic plaque*
  - ⇨ *event. embolism*
  - ⇨ *spasm*
  - ⇨ *inflammation*
  - ⇨ *rarely systemic causes.*

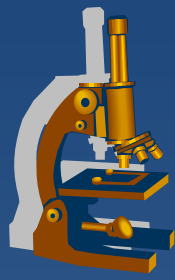
- gross
  - ⇨ ***evolution**; first signs (red, softer) after 12 hrs*
  - ⇨ ***2-3 days** established infarction (yellowish, haemorrhagic rim)*
  - ⇨ ***weeks** – formation of firm white fibrotic scar*

Myocardial infarction



- micro:
  - ⇨ *necrotic cells more red*
  - ⇨ *loss of nuclei and striation*
  - ⇨ *neutrofils*
  - ⇨ *later macrophages in stroma*
  - ⇨ *reparation by granulation tissue -> scar*

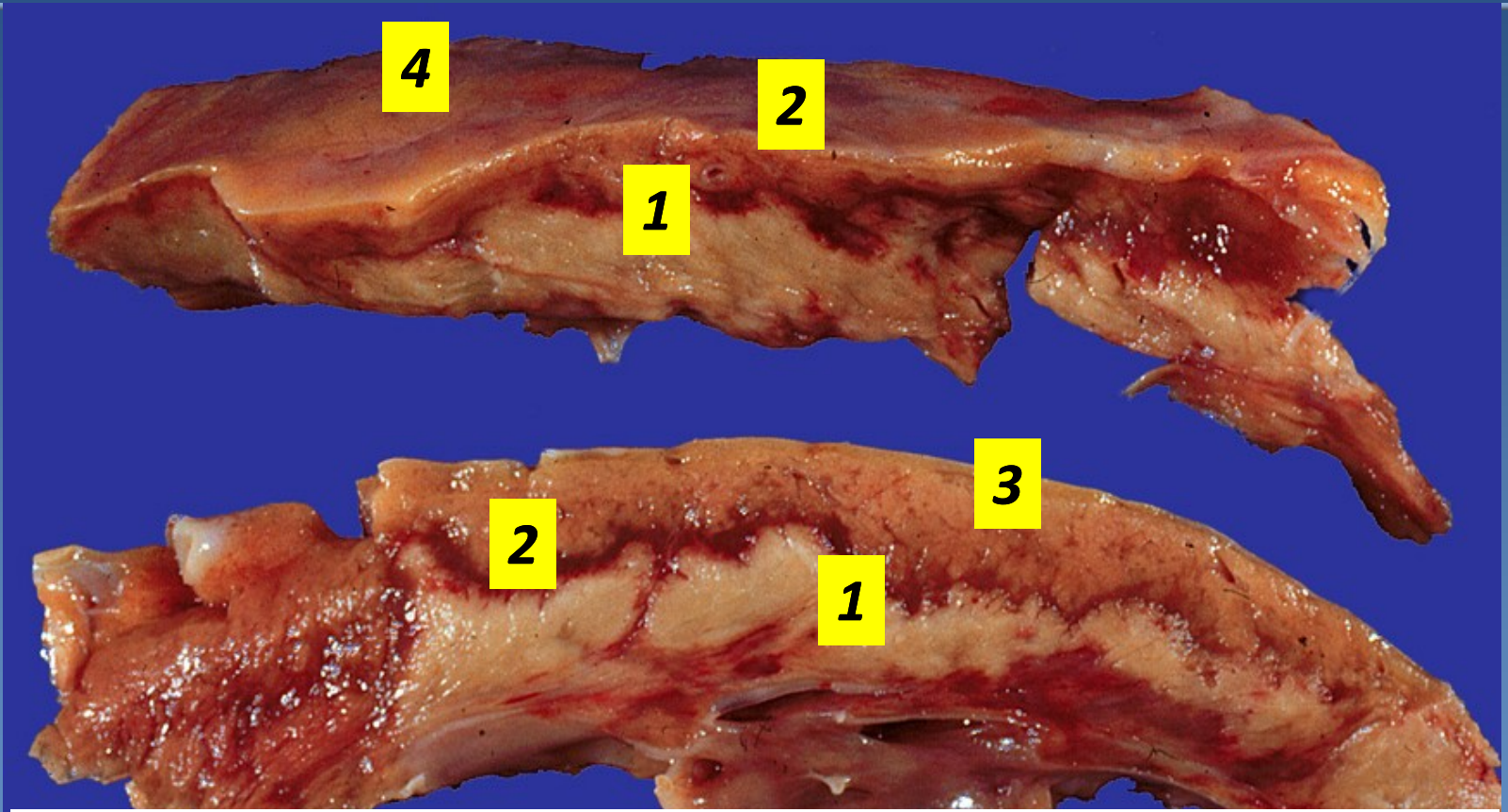
Myocardial infarction



micro:

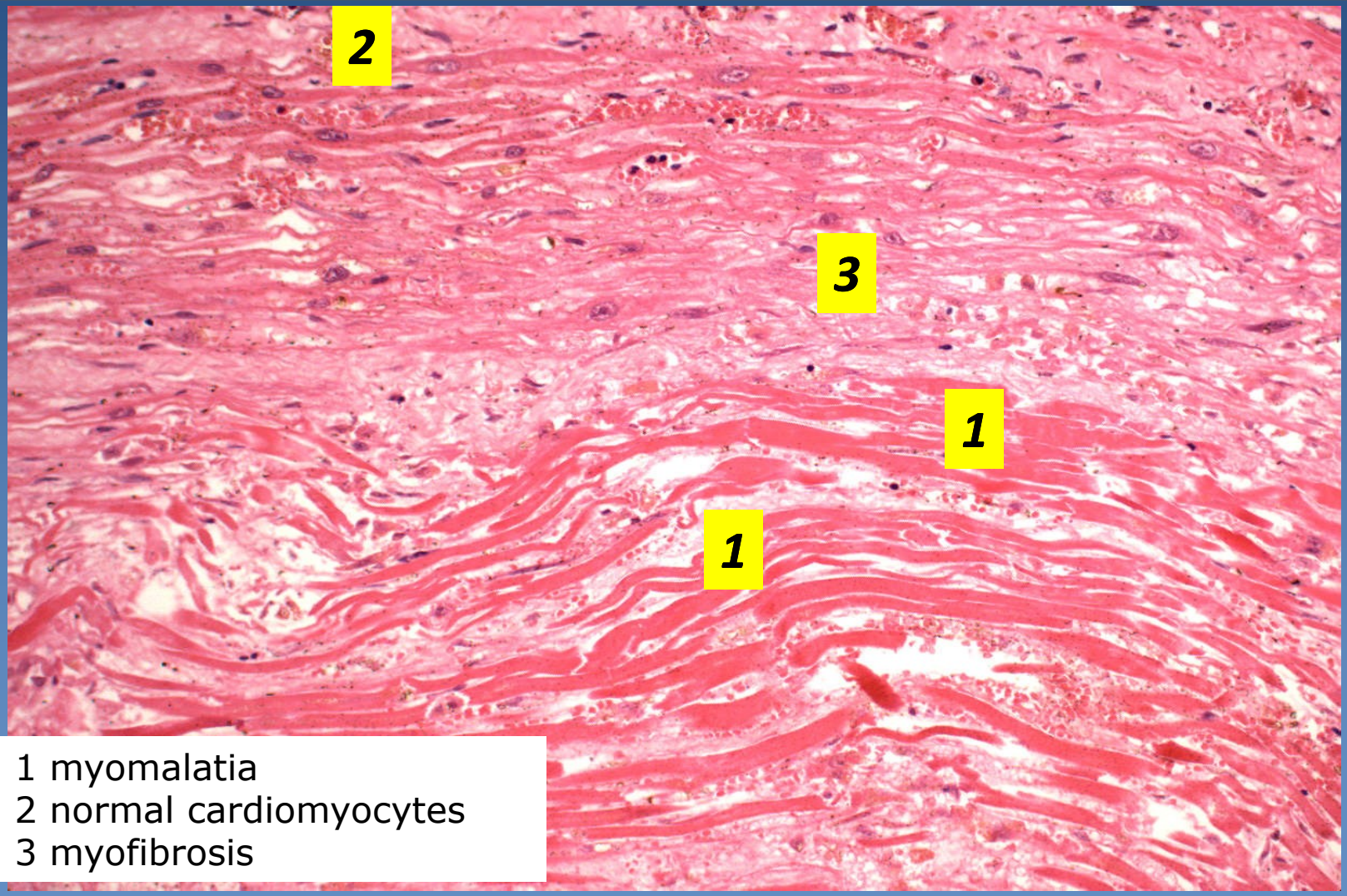
- **12-24 hr:** edema, hypereosinophilia of necrotic cells, pyknosis
- **1-3 days:** neutrophils, loss of nuclei
- **3-7 days:** macrophages at the border, desintegration of myofibers
- **1-2 weeks:** repair by granulation tissue
- **cca 2 months:** scar

Myocardial infarction



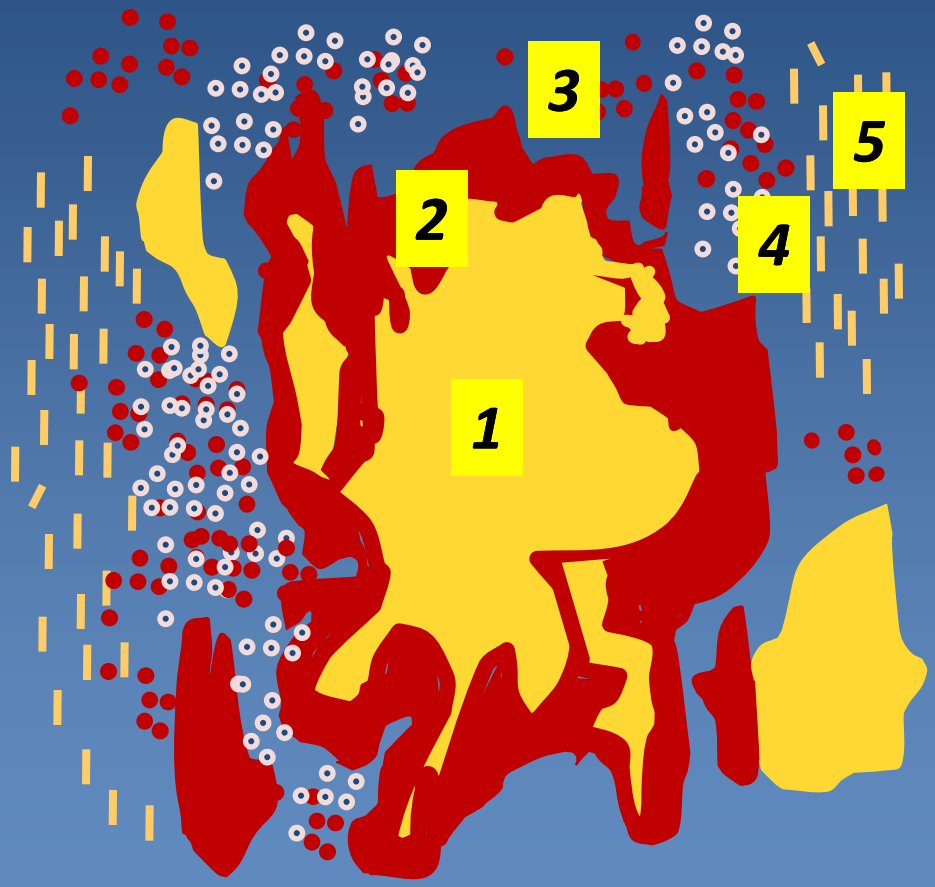
1 subendocardial coagulative necrosis 2 hyperemic rim 3 normal myocardium 4 epicardium

Myomalatia



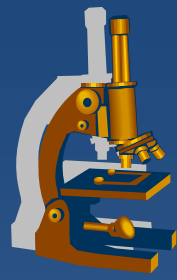
1 myomalatia
2 normal cardiomyocytes
3 myofibrosis

Microscopic changes in developed MI



1 coagulative necrosis
2 myomalatia
3 hyperemic rim
4 neutrophils
5 regressive changes

Myocardial infarction

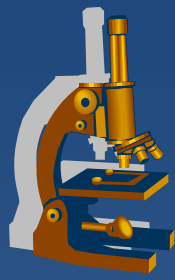


- **transmural (QIM, STEMI) - + ST elevation on ECG**
  - ≥ ¾ of wall thickness, breadth >25 mm
  - complete coronary artery obstruction

    emergency angioplasty/stenting

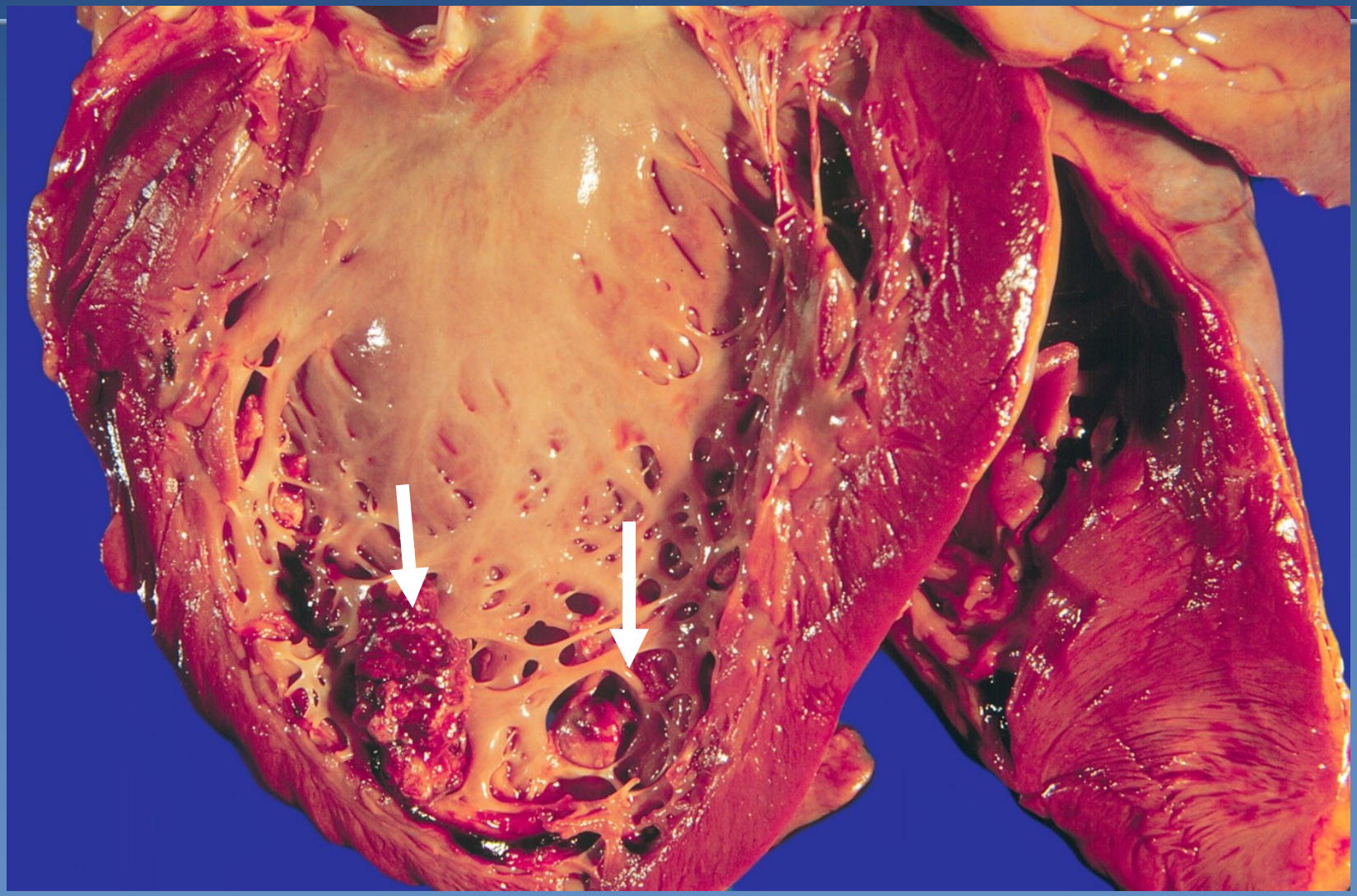
- **non-transmural (subendocardial, Non-STEMI)**
  - internal ¼ až ½ of LV wall
  - collateral bllod flow, incomplete obstruction, shorter ischemia

MI complications

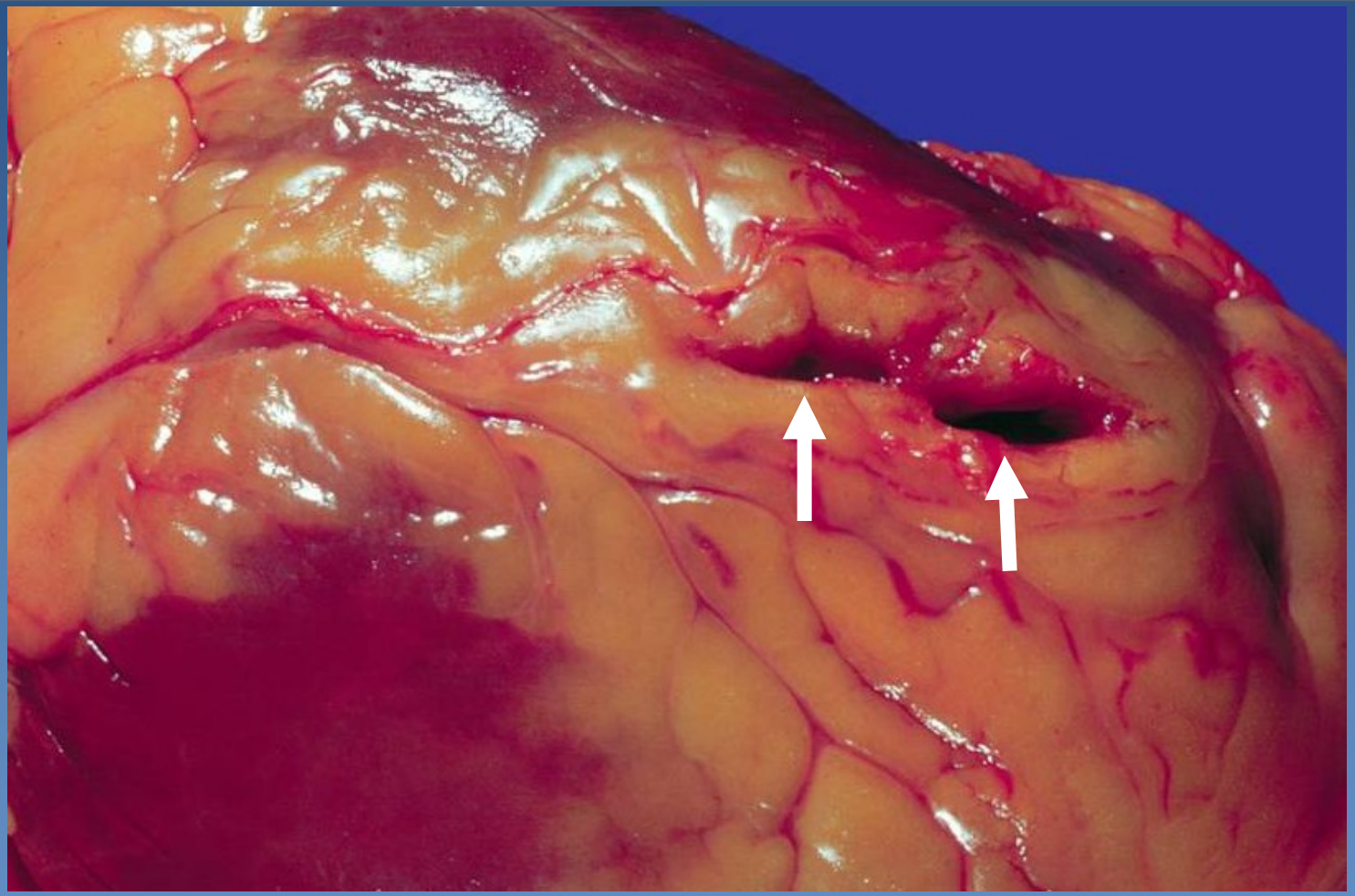


1. **sudden death (**arrythmia)
2. **cardiogenic shock** (contractile dysfunction)
3. **pericarditis epistenocardiaca**
   -> sero-fibrinous inflammation
4. **mural thrombosis**
   -> embolism into systemic circulation (-> brain, kidney, intestine, spleen infarction)
5. **ventricular aneurysm**
   *-> **acute** – risk of rupture, trhrombosis; **chronic** – LV insufficiency*
6. **cardiac rupture**
   *-> **free wall, septum, :** tamponade / acute heart failure*
7. **papillary muscle rupture**
   *-> **valvular incompetence** → acute heart failure*

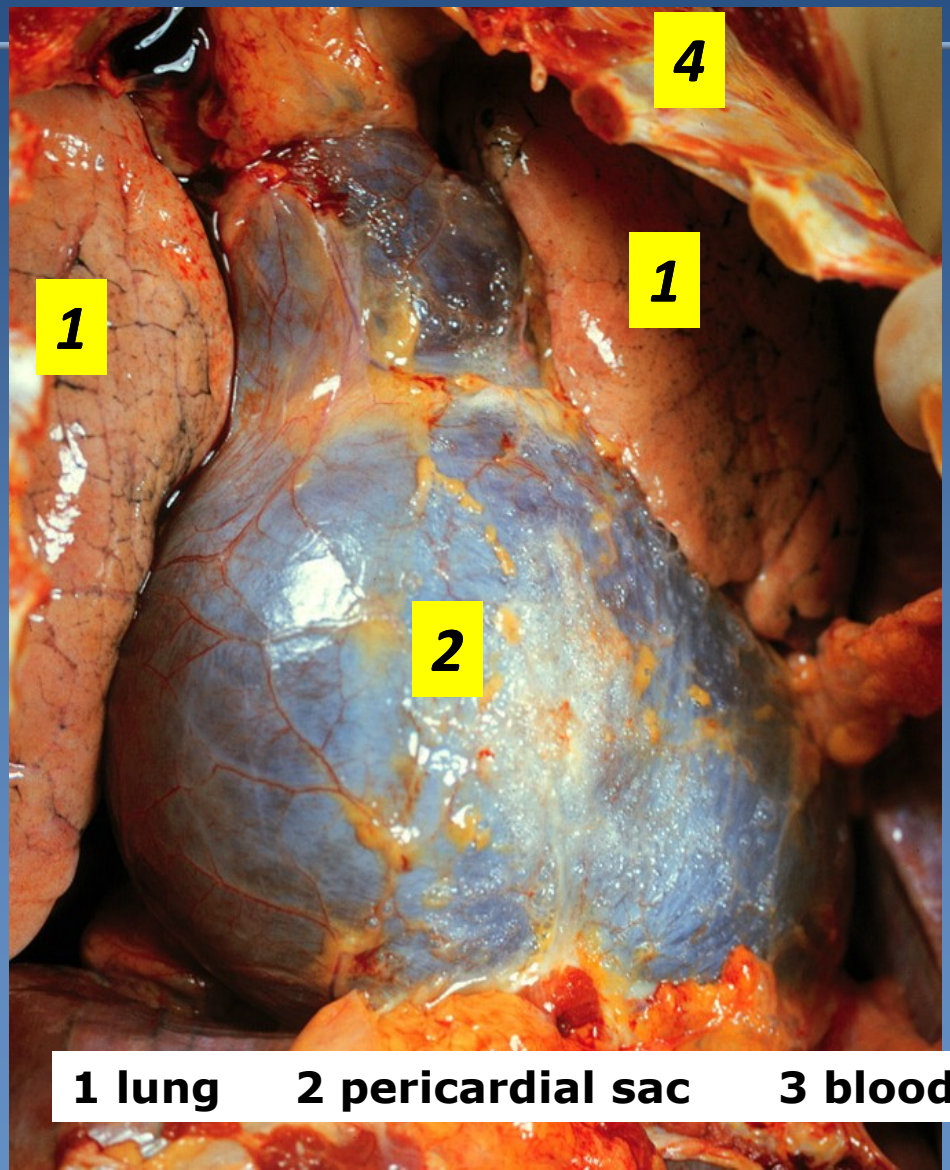
MI – mural thrombosis

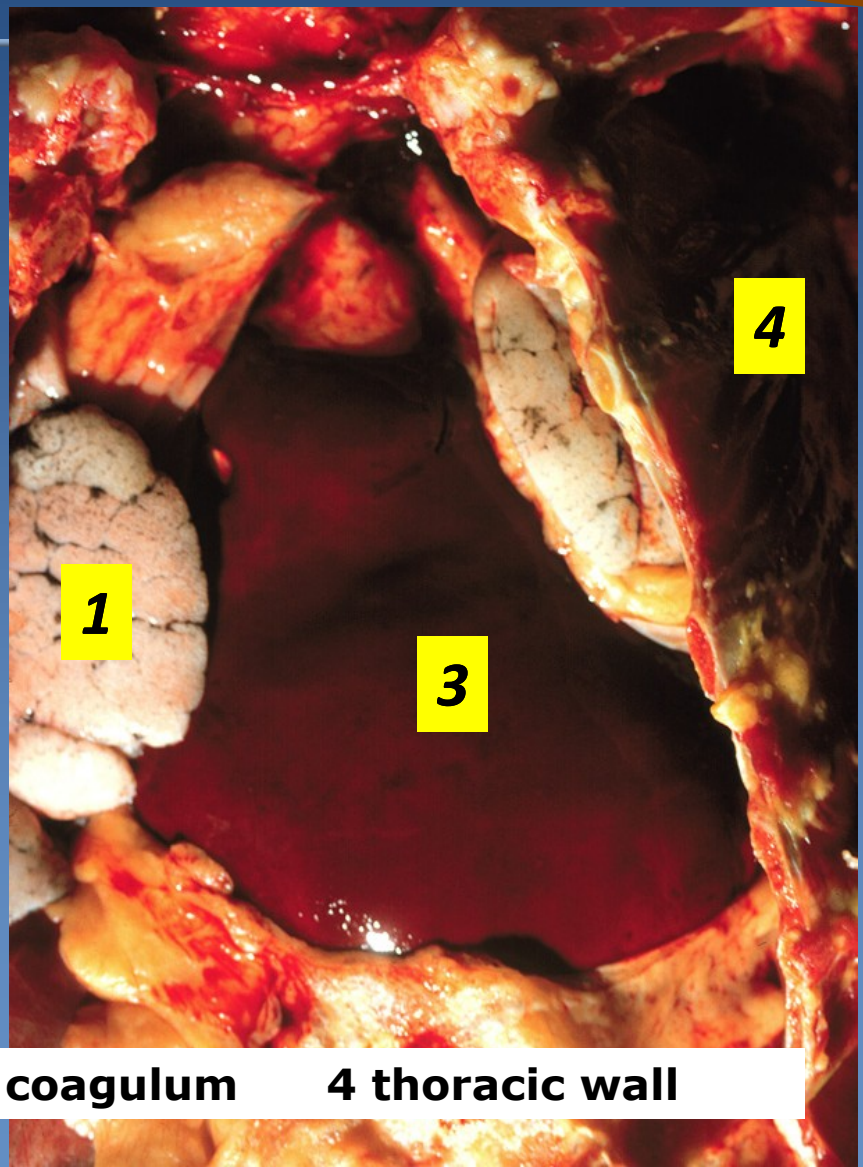


Mi – rupture



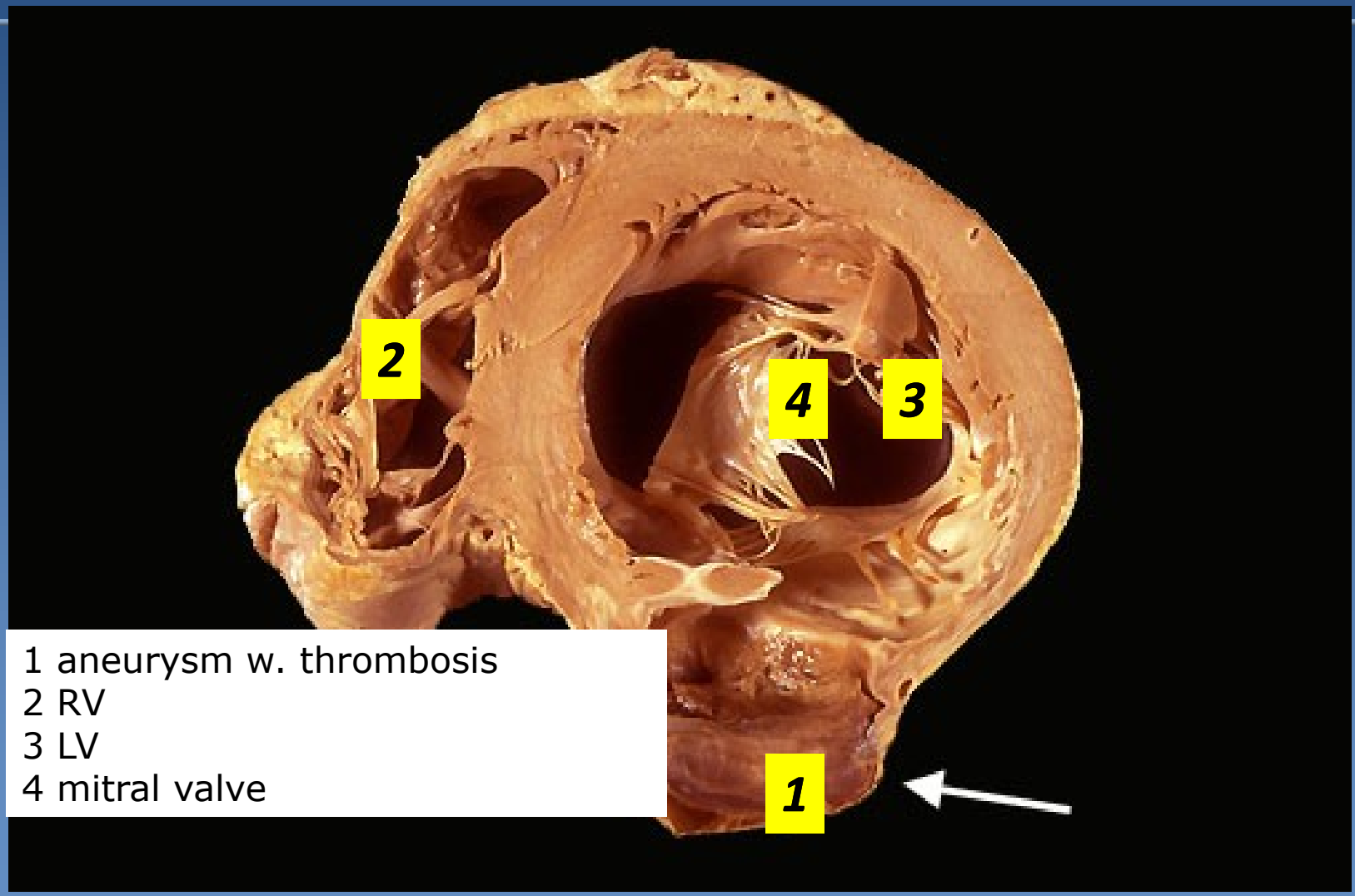
MI – rupture, tamponade





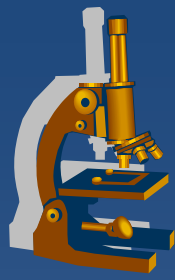
1 lung 2 pericardial sac 3 blood coagulum 4 thoracic wall

MI – LV aneurysm



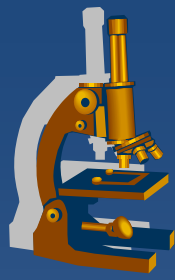
1 aneurysm w. thrombosis
2 RV
3 LV
4 mitral valve

Chronic ischemic heart disease (IHD)



- angina pectoris or MI in anamnesis
- progressive heart failure due to ischemic myocardial damage ➔ LV failure ➔ congestive RV failure
- heart hypertrophy + dilatattion, myofibrosis and/or post-MI scars
- multiple coronary arteries with significant AS stenosis
- imminent risk of MI, sudden cardiac death due to arrythmia, heart failure

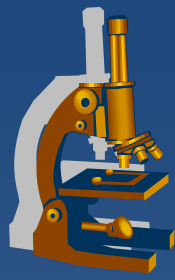
Sudden cardiac death



= unexpected death from cardiac causes, without preexisting symptoms or within 1 hr of the onset of symptoms

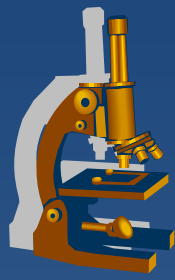
- most commonly due to lethal arrythmia (ventricular fibrillation, asystole)
- sudden collapse without signs of acute MI
- other causes:
  - ***dissecting/ruptured aortic aneurysm***
  - ***pulmonaty thrombembolism***
  - ***massive intracerebral haemorrhage***
  - ***heritable conditions incl. anatomic, electriical – channelopathies***

Endocardial / valvular diseases



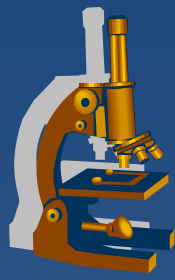
- endocarditis
  - ⇨*infectious or immune-mediated endocardial inflammation*
- degenerative diseases
  - ⇨*calcific aortic (rarely mitral) stenosis, mitral valve prolapse, annular and marginal sclerosis*
- endocrine diseases
  - ⇨*carcinoid syndrome*
- nonbacterial thrombotic endocarditis (in debilitated patients)

Infective endocarditis



- commonly by highly virulent microorganisms
  - ⇨*Strep. pyogenenes, Strep. pneumoniae, Staph. aureus, … ev. fungi*
- subacute IE – less virulent microorganisms
  - ⇨*viridans streptococci*
- predisposition:
  - ⇨*deformed valve, bioprosthesis, postcatethrization, i.v. drug addicts*
- bacteremia - endocardial damage by bacteria - trombosis = infective vegetation

Infective endocarditis

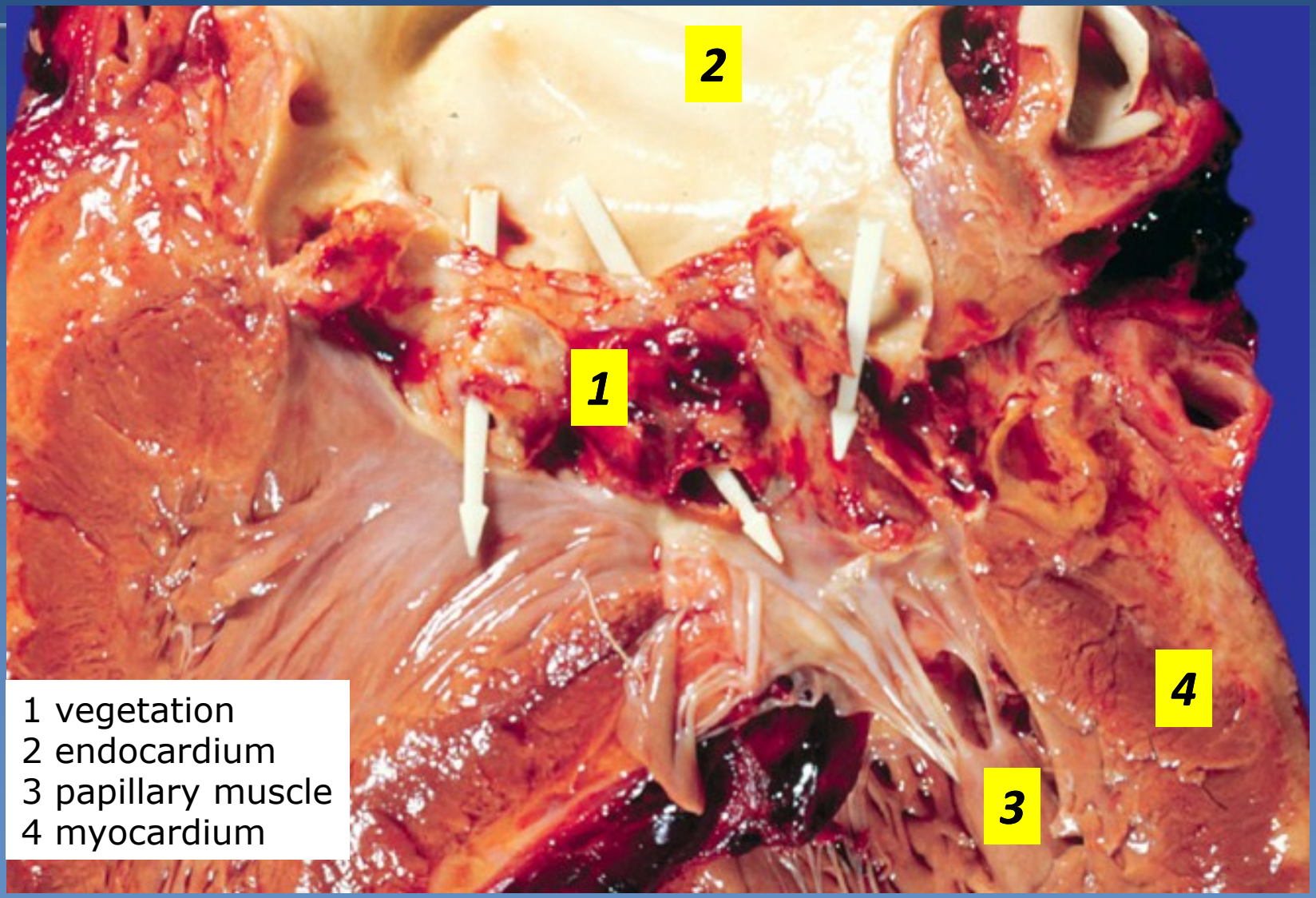


- **gross:** friable red-brown mass 0,5-2 cm on leaflets or chordae tendinae, valvular damage incl. ulceration

- **micro:**
  - ⇨ *fibrin + bacterial colonies + neutrophils (+ granulation tissue)*
  - ⇨ *Inflammation/ necrosis of the valve tissue*

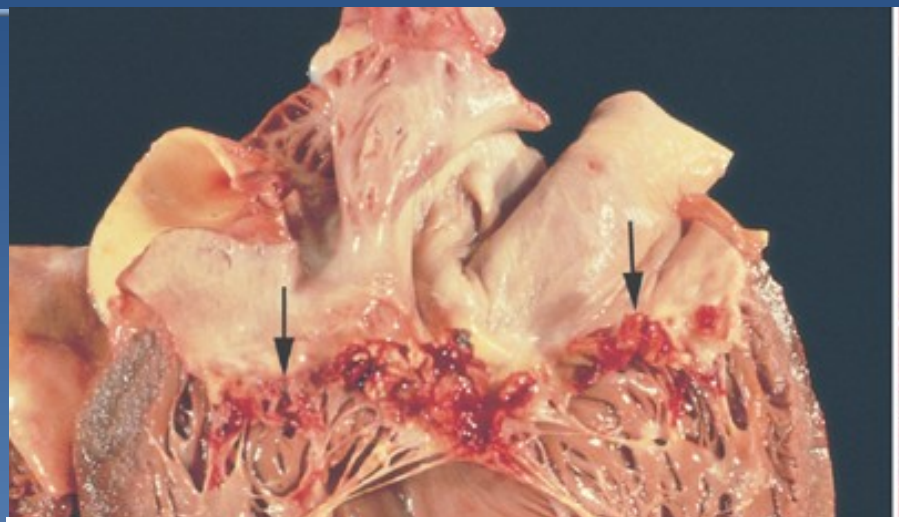
- complications:
  - ⇨ *acute: valvular damage, myocarditis + abscess, pyemia,thrombembolism*
  - ⇨ *chronic valvular disease*

Infective endocarditis–
valve destruction

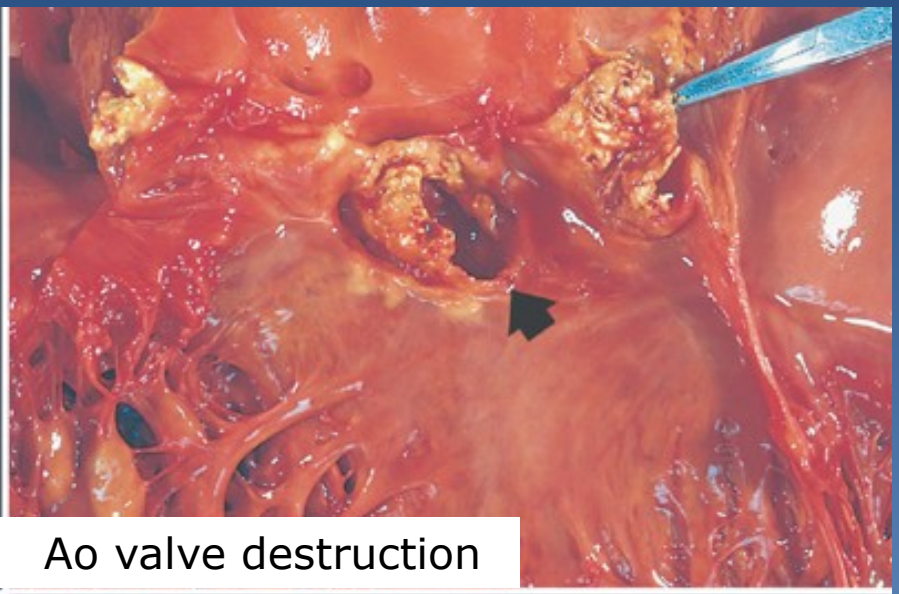


1 vegetation
2 endocardium
3 papillary muscle
4 myocardium

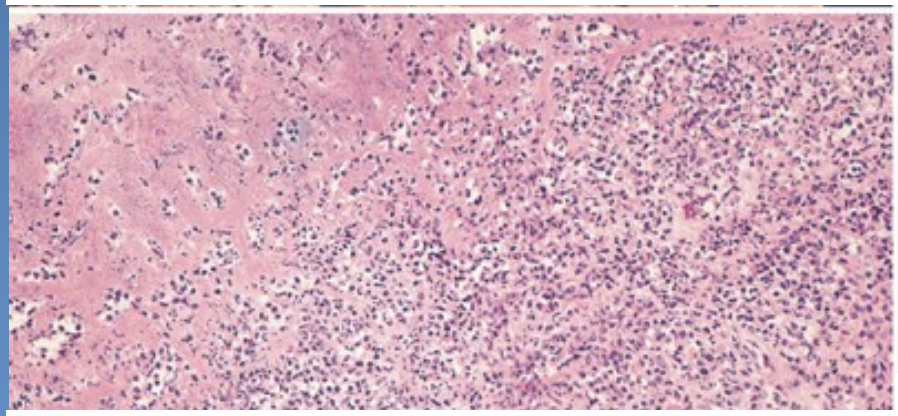
Infective endocarditis



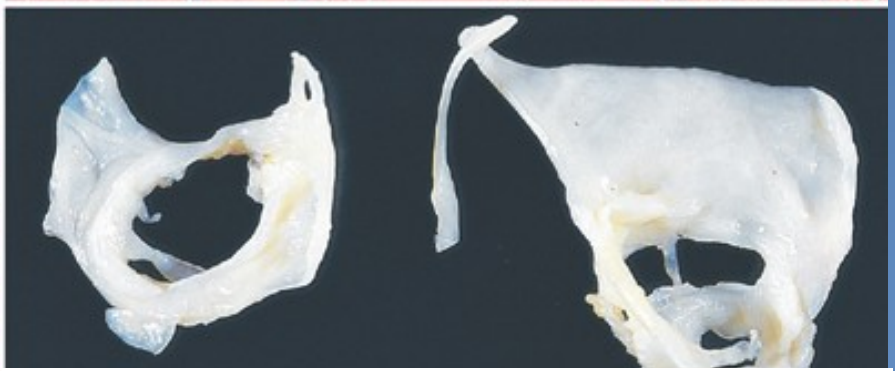
Mi vegetations



Ao valve destruction

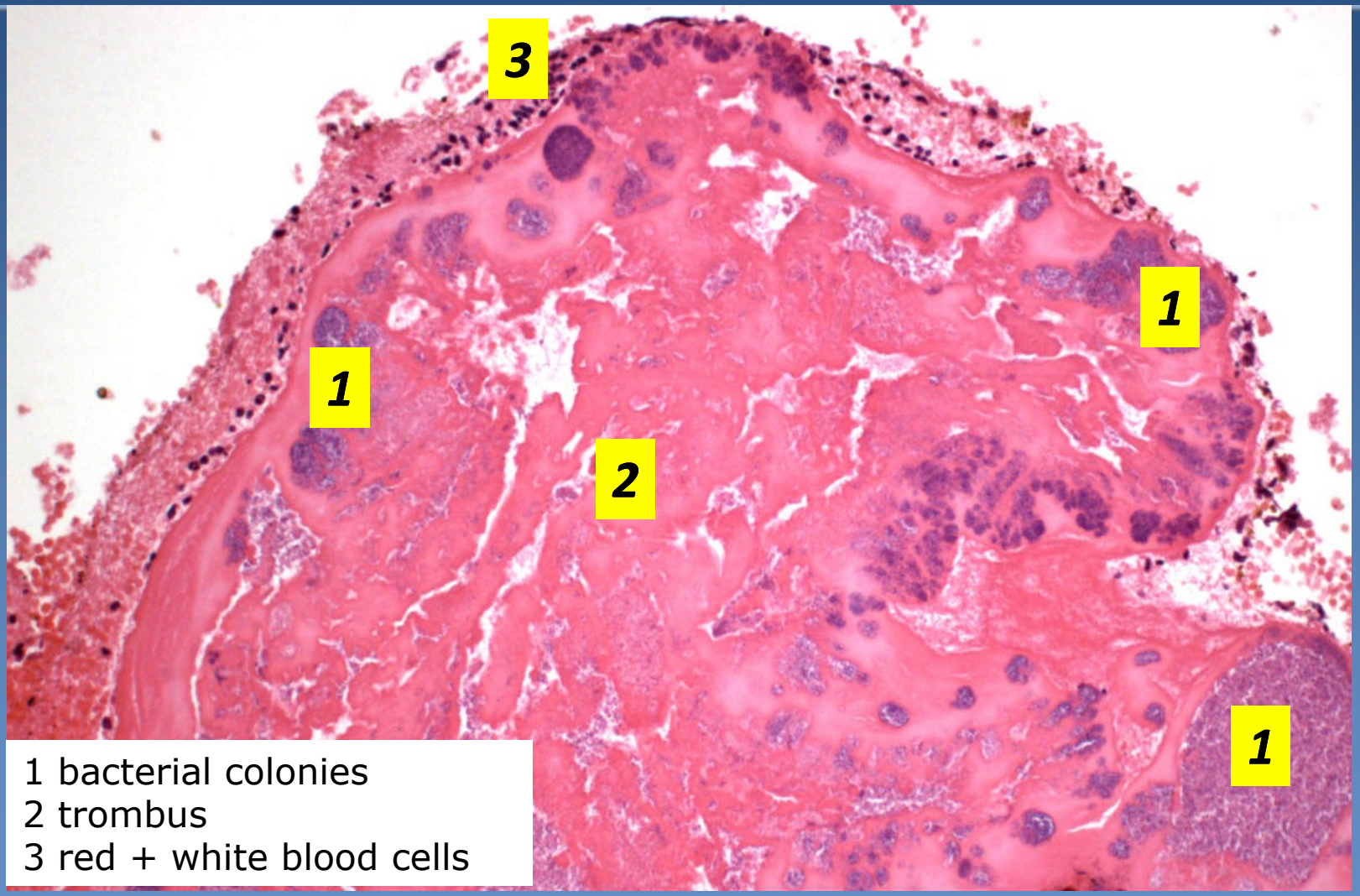


purulent inflammation



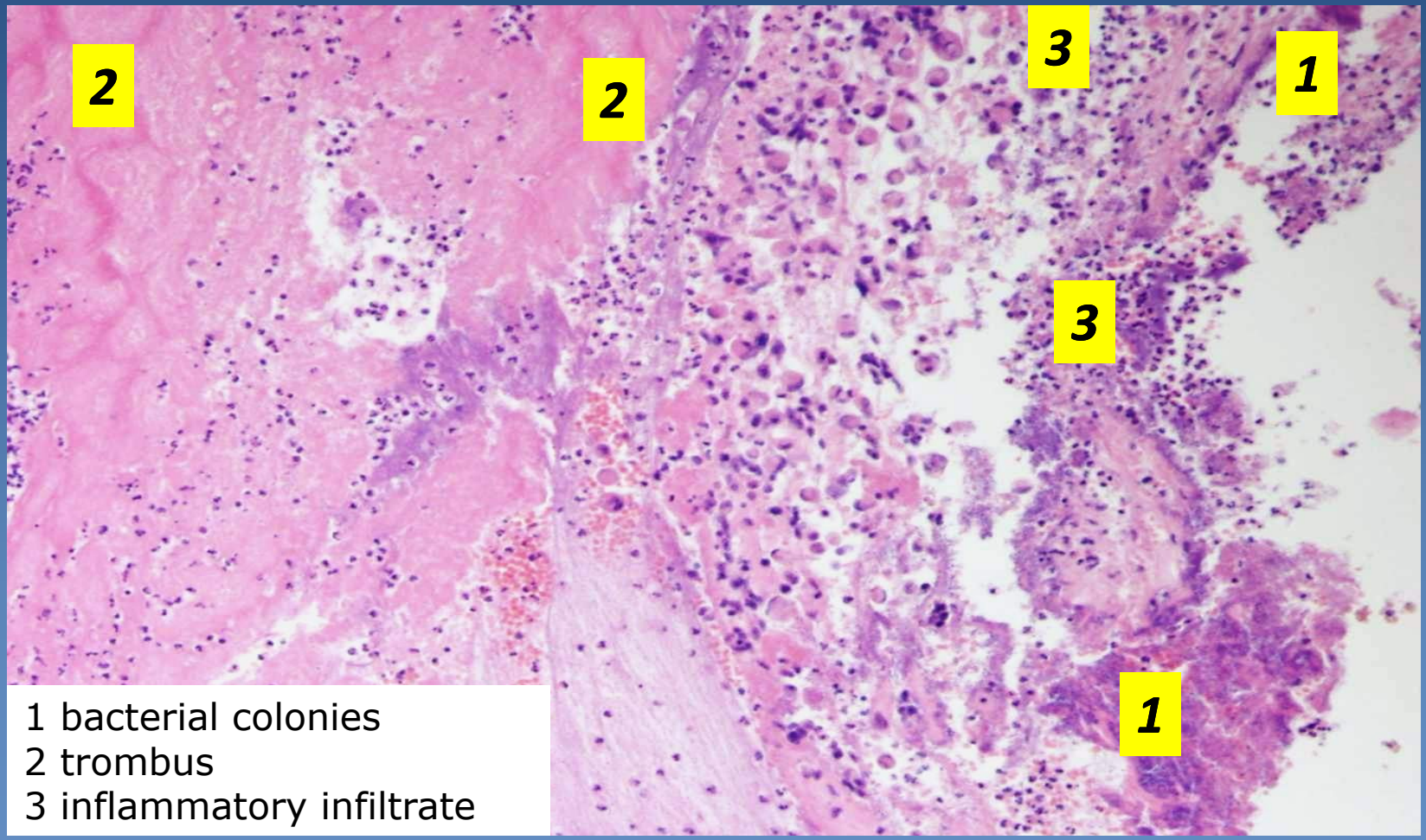
IE repair (Mi fenestration without vegetations)

Infective endocarditis - *vegetations*



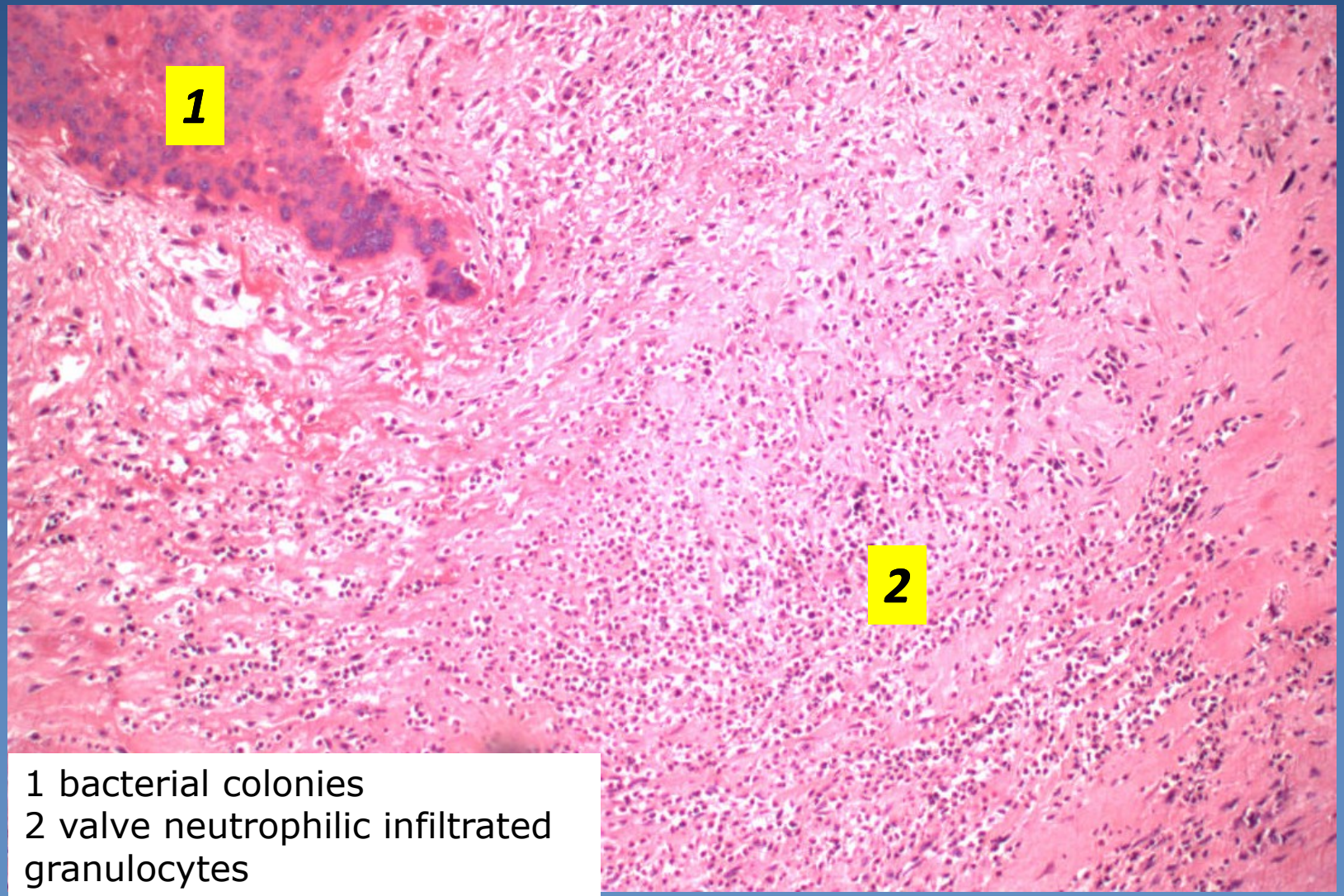
1 bacterial colonies
2 trombus
3 red + white blood cells

Infective endocarditis - vegetations



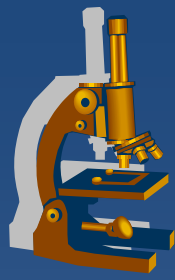
1 bacterial colonies
2 trombus
3 inflammatory infiltrate

Infective endocarditis - vegetations



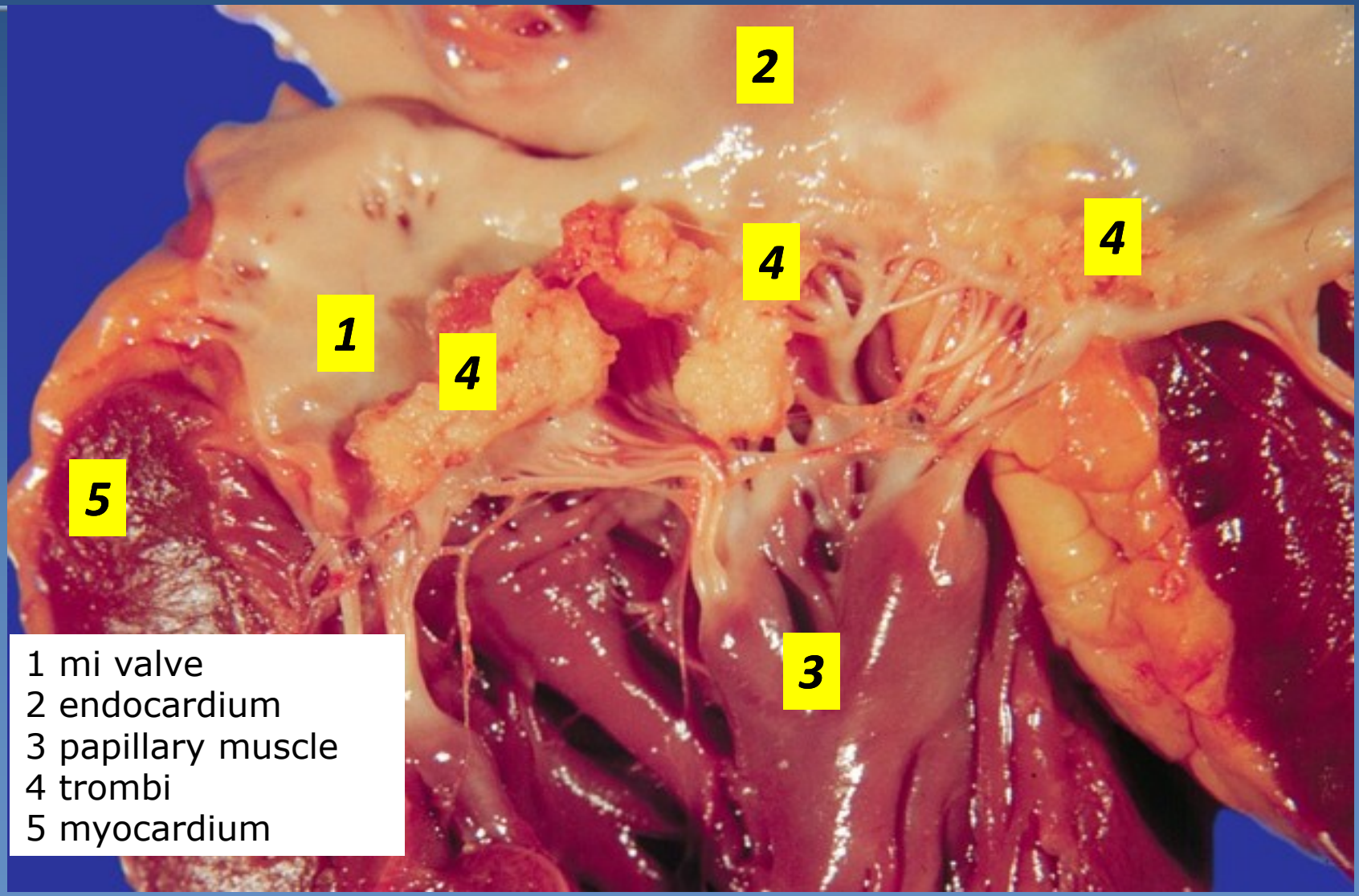
1 bacterial colonies
2 valve neutrophilic infiltrated granulocytes

Non-bacterial thrombotic endocarditis



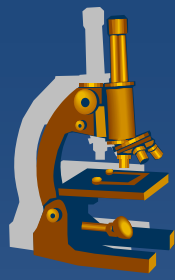
- **sterile** vegetations due to **hypercoagulative state** ⇒ concurrent venous thrombosis and lung embolization
- **in generalized malignancies**, chronic nephropathy with uremia, COPD etc.
- mostly on **mitral valve** (normal)
- micro: verrucous vegetations (single or multiple), 1-5 mm, bland thrombi
- possible source of **emboli**

Non-bacterial thrombotic endocarditis



1 mi valve
2 endocardium
3 papillary muscle
4 trombi
5 myocardium

Rheumatic fever, rheumatic heart disease

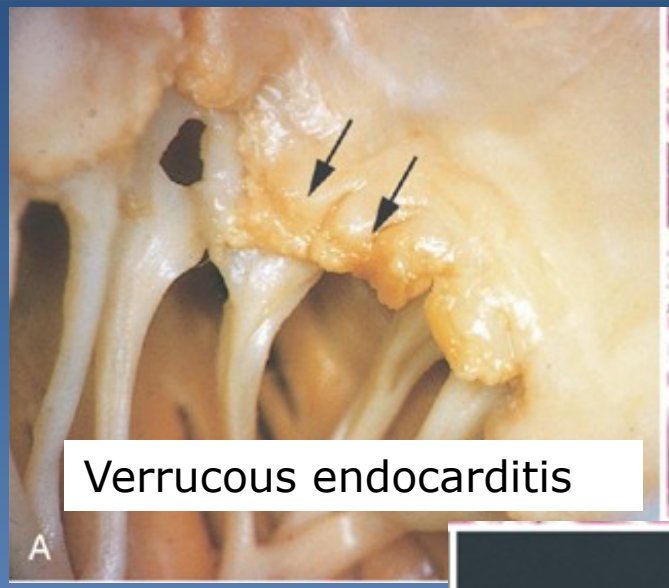


- acutenon-purulent, **imunne-mediated** systemic poststreptococcall inflammation (cross-reactive antibodies)

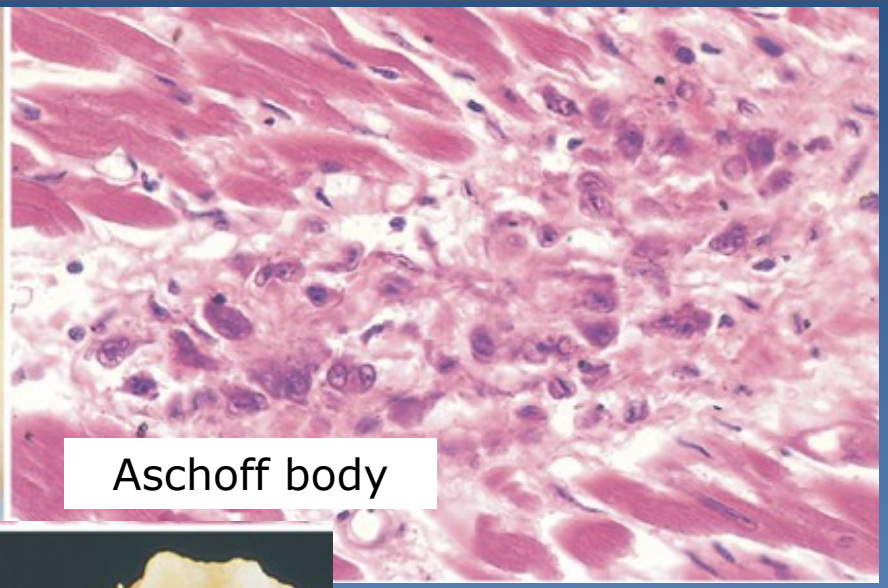
- **acute stage: PANCARDITIS**
  - ⇨*fibrinous pericarditis + myocarditis with Aschoff bodies (foci of fibrinoid necrosis + inflammatory reaction + verrucous endocarditis (small depositions of fibrin along the closure lines of Ao a Mi valves)*
  - ⇨*acute endocarditis commonly recurrent*

- **chronic stage:**
  - ⇨ *diffuse fiubrous thickening + distortion, commisural fusion → dystrophic calcifikacation - stenosis + incompetence)*

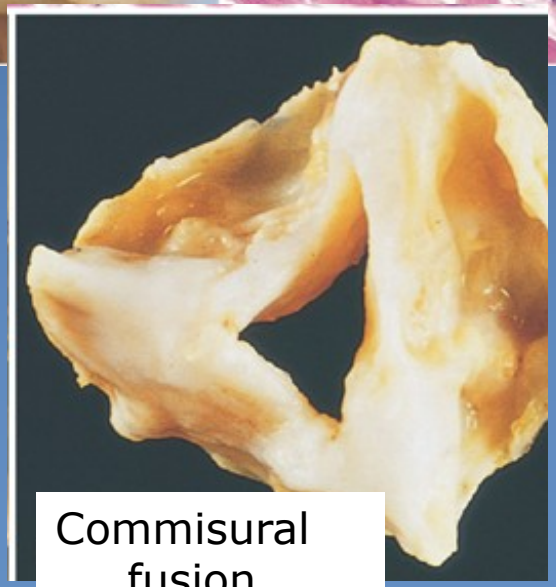
rheumatic heart disease



Verrucous endocarditis

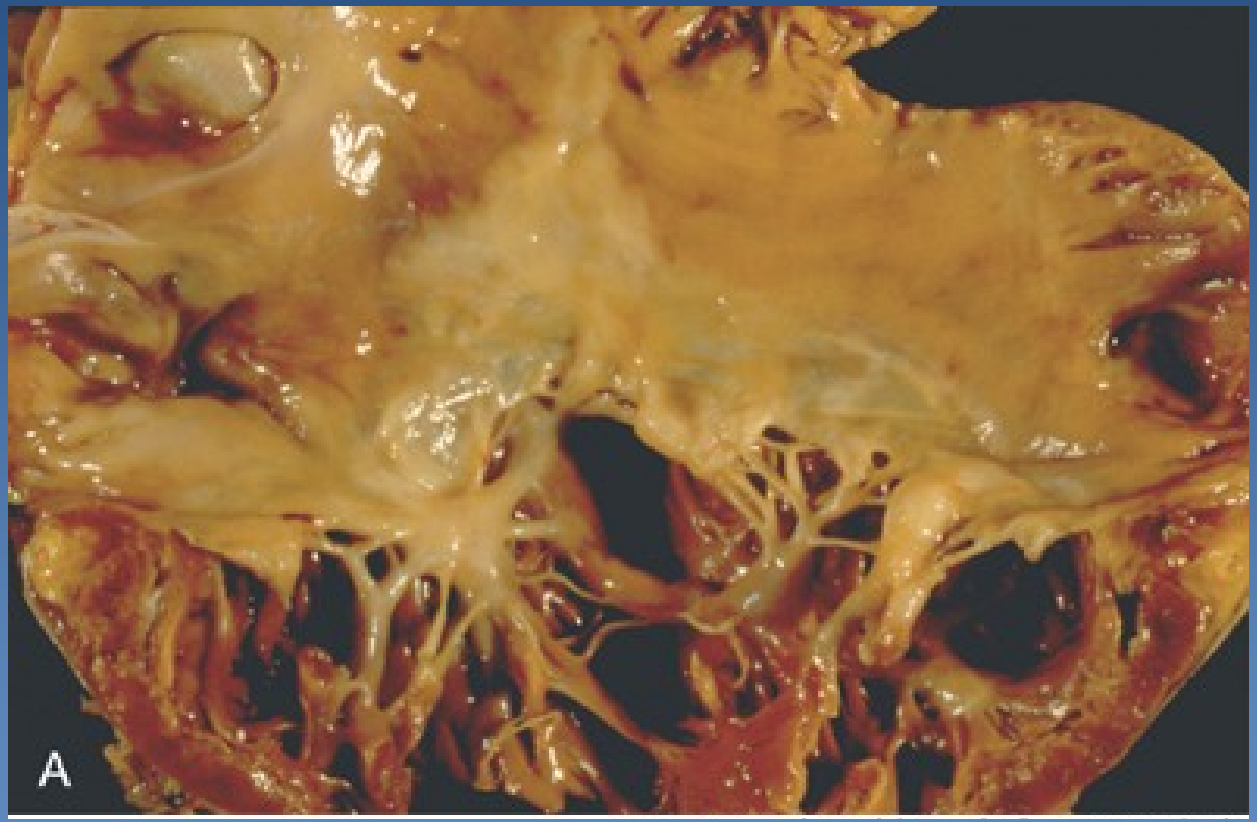


Aschoff body



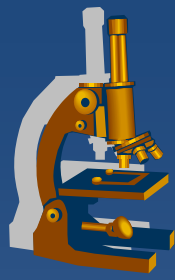
Commisural fusion

Carcinoid syndrome



endocardial fibrous plaquelike thockenings – RA, RV

Myocarditis



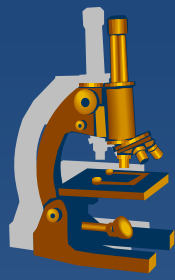
- myokardial inflammatory damage without **ischemia**

- **gross:**
  - ⇨ ***cardiac dilatation, flabby, mottled myocardium***

- **micro:**
  - ⇨***inflammatory infiltrate (acccording etiology) + cardiomyocyte regressive changes incl. necrosis***

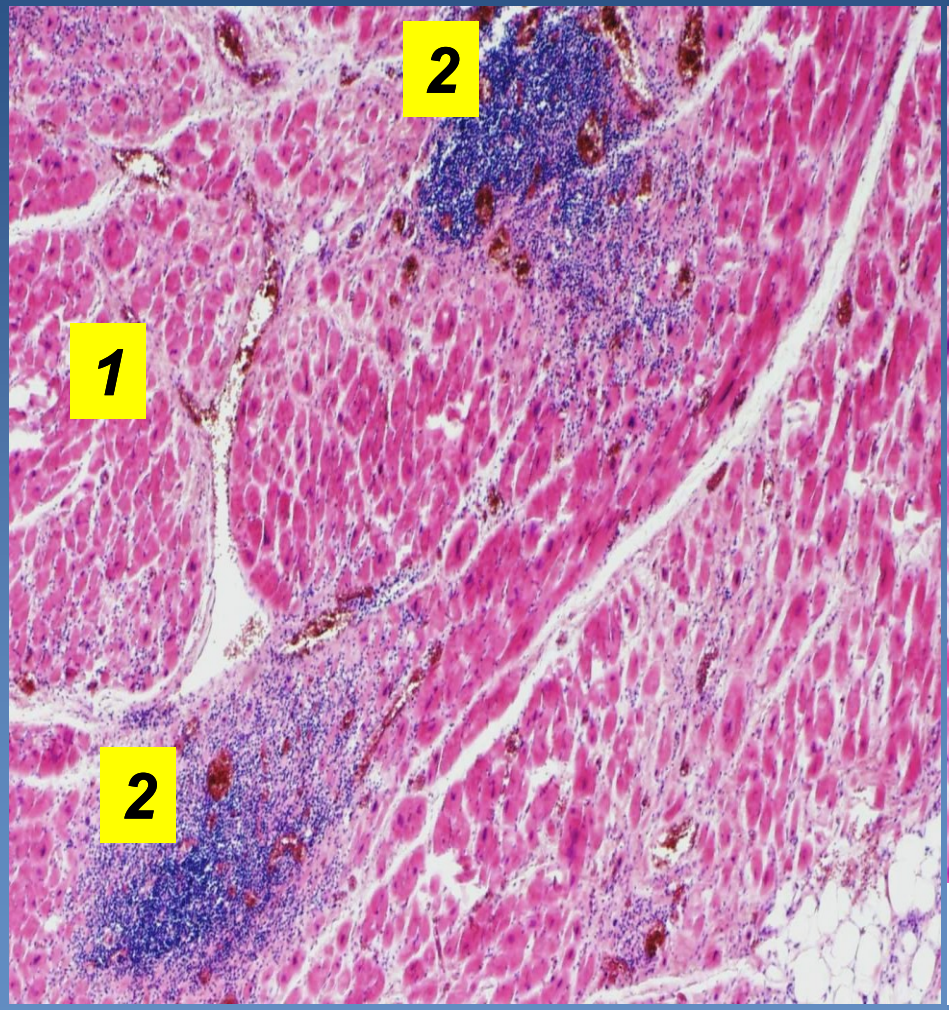
- **etiology:**
  - ⇨ viruses, ricketsia, chlamydia, bacteria (diphtheria, sepsis), fungi, protozoa (toxoplasmosis), helminths (trichinosis)
  - ⇨ immune-mediated *(drug hypersesitivity, postviral, rheumatic fever, rejection)*
  - ⇨*ionising radiation*
  - ⇨unknown *(giant-cell myocarditis*

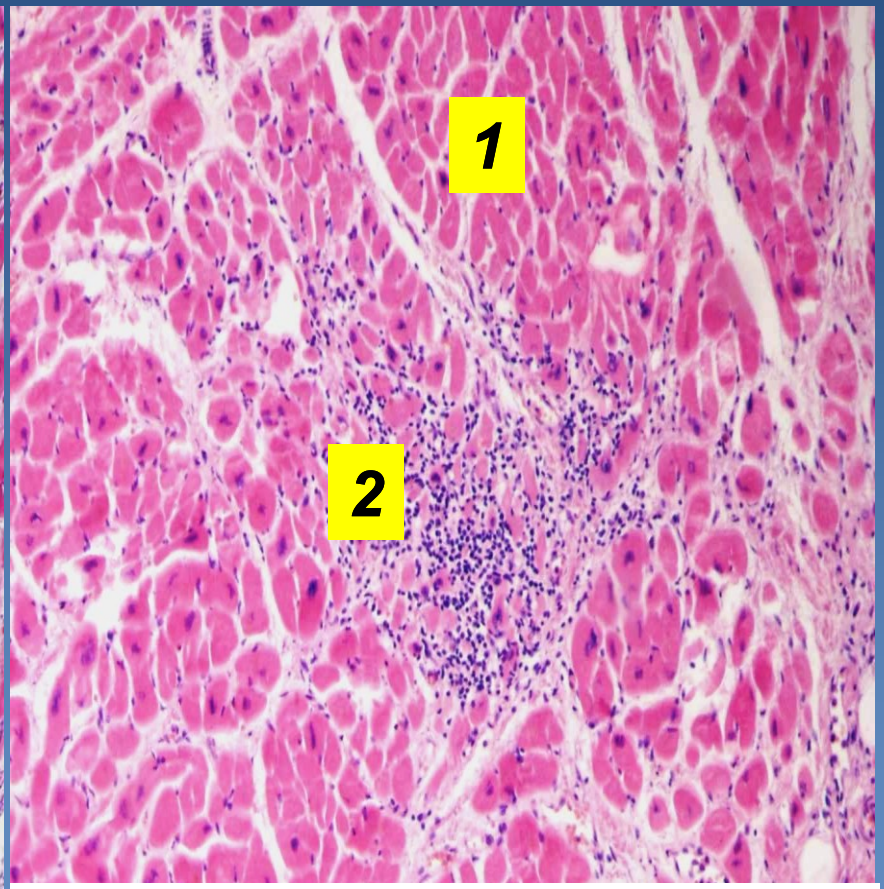
Viral myocarditis



- ***Coxsackie, parvovirus B19, influenza, EBV, CMV, HIV***
- inflammatory infiltrate: T-cells mostly
- after acute attack commonly autoimune-mediated cardiomyocytes destruction and fibrosis → dilated cardiomyopathy

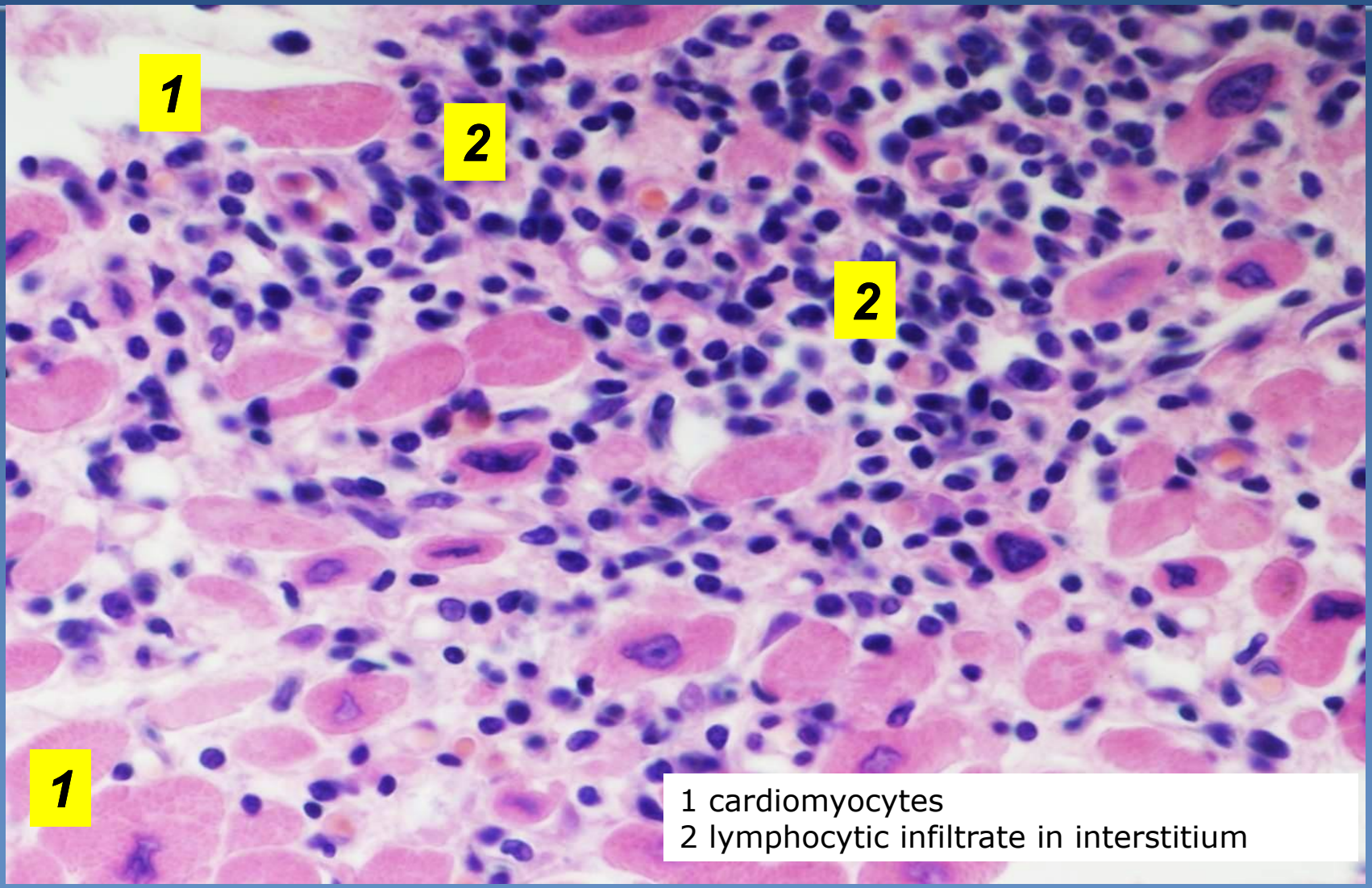
Viral myocarditis





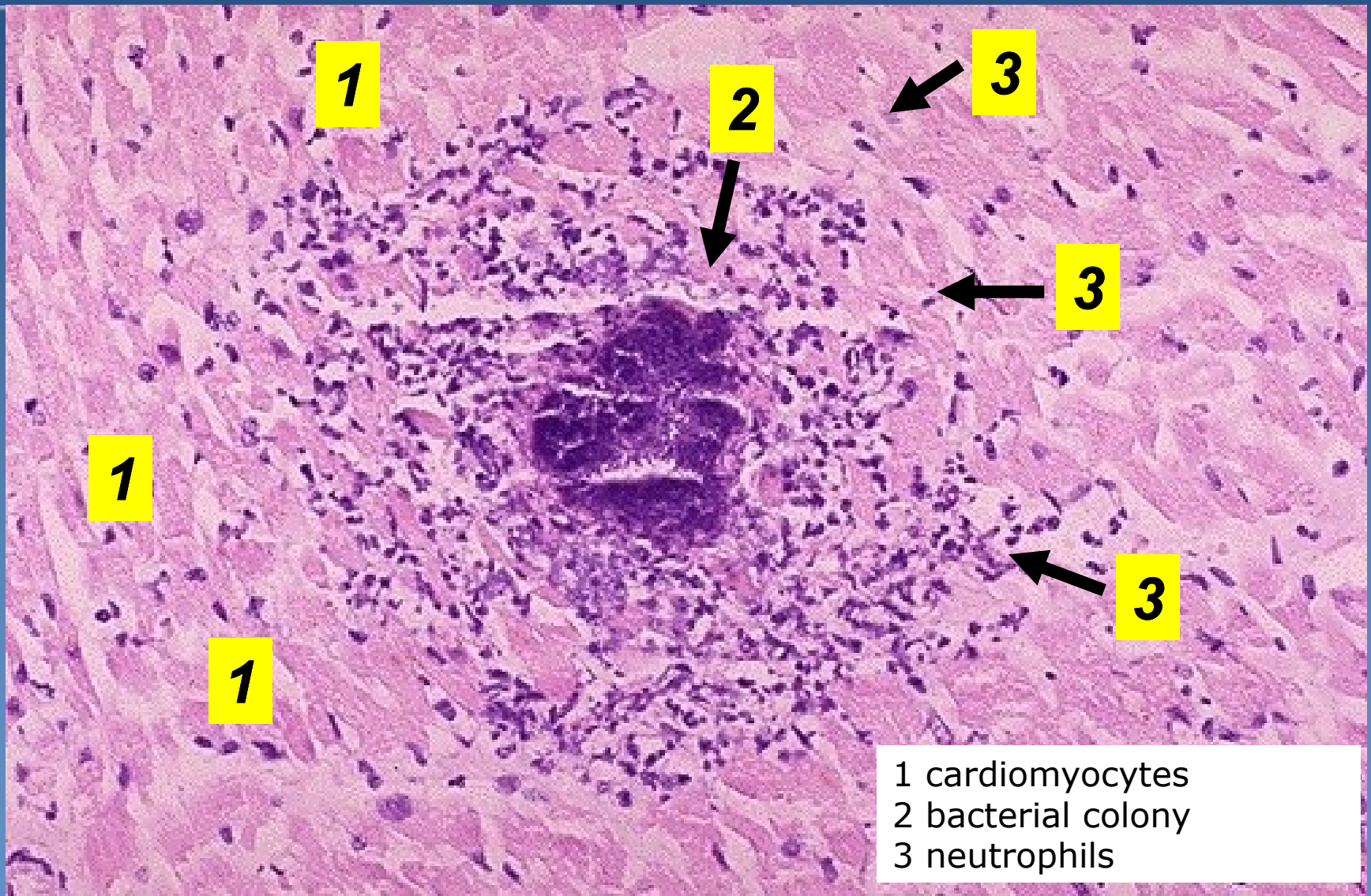
1 cardiomyocytes
2 lymphocytic infiltrate in interstitium

Viral myocarditis



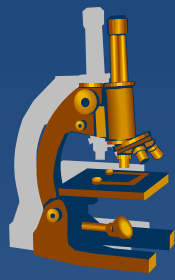
1 cardiomyocytes
2 lymphocytic infiltrate in interstitium

Septic myocarditis



1 cardiomyocytes
2 bacterial colony
3 neutrophils

Cardiomyopathies

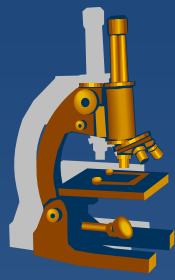


= heart disease due to myocardial abnormality, with heart dysfunction

diagnosis after exclusion of IHD, valvular disease, congenital d. or hypertension

- **heterogenous group of disorders:**
  - **dilated (DCM)**
    - *dilatation + hypertrophy, ↓ LV contraction, possible mural thrombosis; 20–50% genetic (AD); alkoholic, peripartum, myocarditis...*
  - **hypertrophic (HCM)**
    - *massive LV hypertrophy, 100% genetic, diastolic dysfunction, histologic „disarray“*
  - **restrictive cardiomyopathy**
    - *diastolic dysfunction, ↓ of compliance - ↓ filling, myocardial stiffness*
  - **specific CM**
    - *Duchenne muscle dystrophy, toxic (drugs), endocrine d., metabolic d. (hemochromatosis, amyloidosis, glykogenosis,…)*

Myocardial amyloidosis

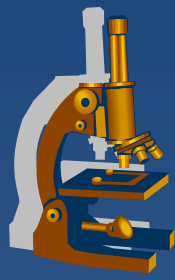


- local x systemic (mostly AL amyloidosis)
- senile amyloidosis
  - ⇨ *atrial + ventricles; amyloid protein = prealbumin (transthyretin)*
- *isolated atrial amyloidosis*
  - ⇨ *amyloid protein = atrial natriuretic peptide*
- **gross**: consistency normal - firm (rubbery)
- **micro**: variable amyloid deposits v interstitium and vessels, Congo red + polarization

Senile cardiac amyloidosis

1 normal cardiomyocytes
2 deposits amyloid

Pericardial pathology



1) Pericardial effusion

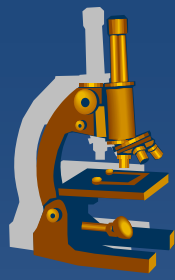
- transudate in congestive heart failura or hypoproteinemia, slow (up to 500ml – pericardial dilatation)

2) haemopericardium

– wall rupturein MI or aortic root dissection ➔ ***fatal cardiac tamponade***

diastolic filling restriction

Pericardial pathology



3) Inflammatory exudate in pericarditis:

a) non-infectious

– pericarditis epistenocardiaca, uremic, post-operative, SLE, Dressler sy (post-MI autoimmune)

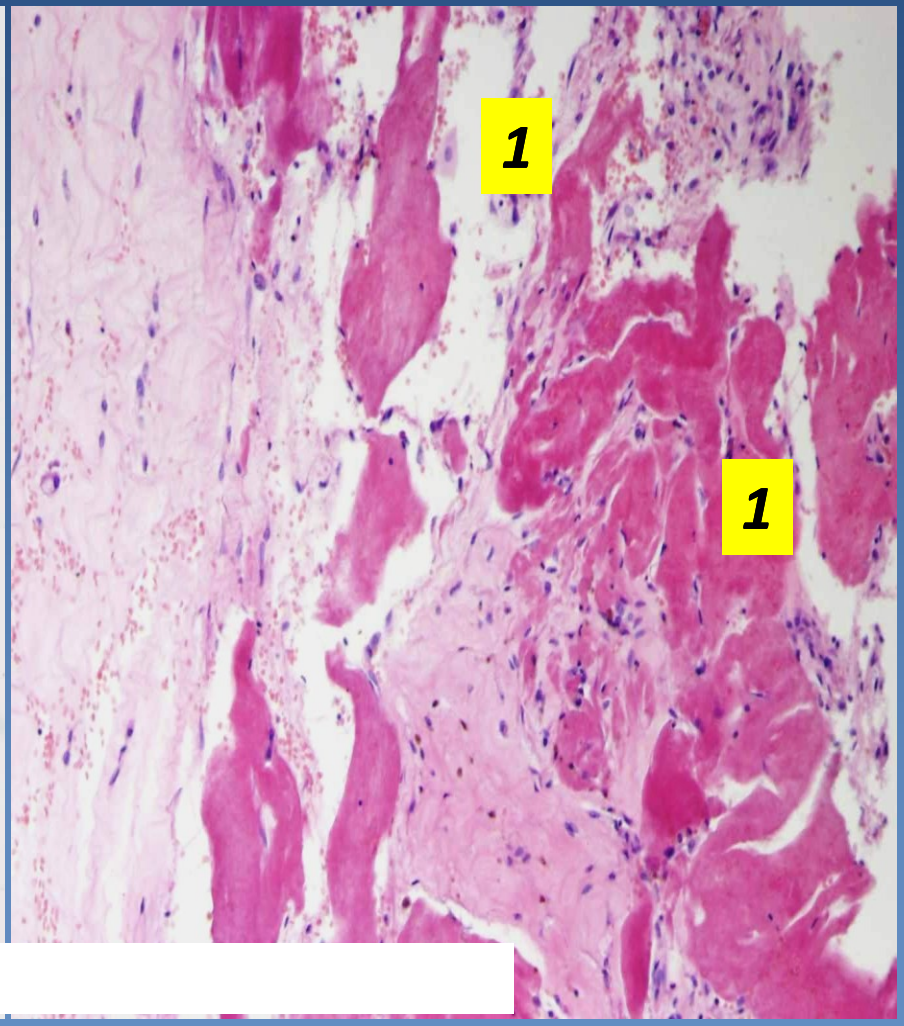
b) *infectious*

– haematogenous, direct spread, lymphogennous; variable agents

c) idiopatická

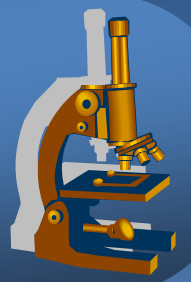
Pozn. hojení *– serózní a část. i fibrinózní exsudát ➔ vstřebávání x zbývající fibrin se organizuje ➔ perikardiální adheze /konstriktivní perikarditida (pericarditis petrosa) ➔ omezuje plnění komor*

fibrinous pericarditis

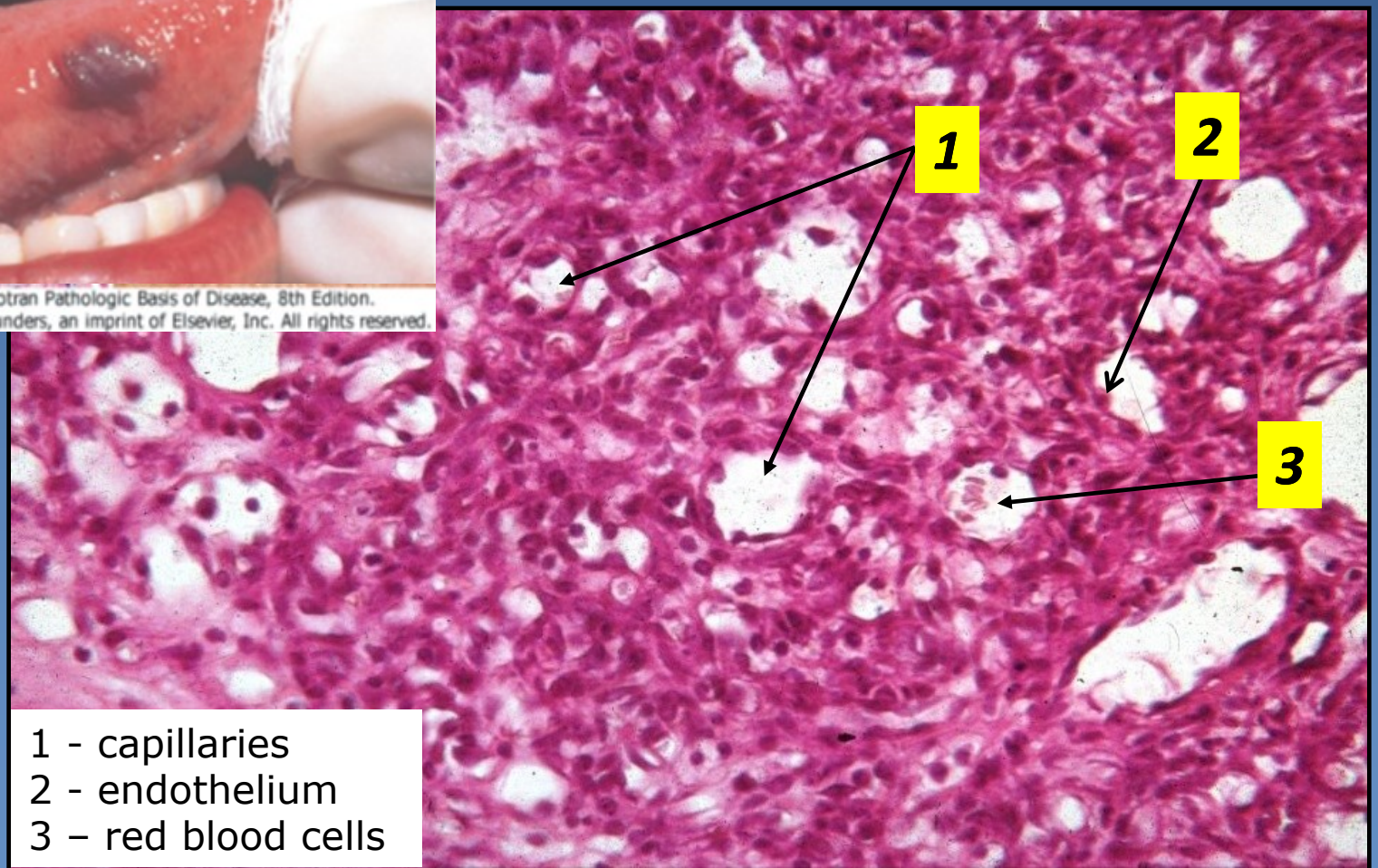


1 fibrinous exudate

Cardiovascular tumors

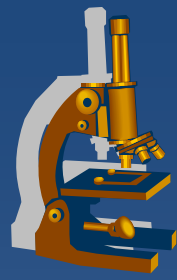


Capilary hemangioma



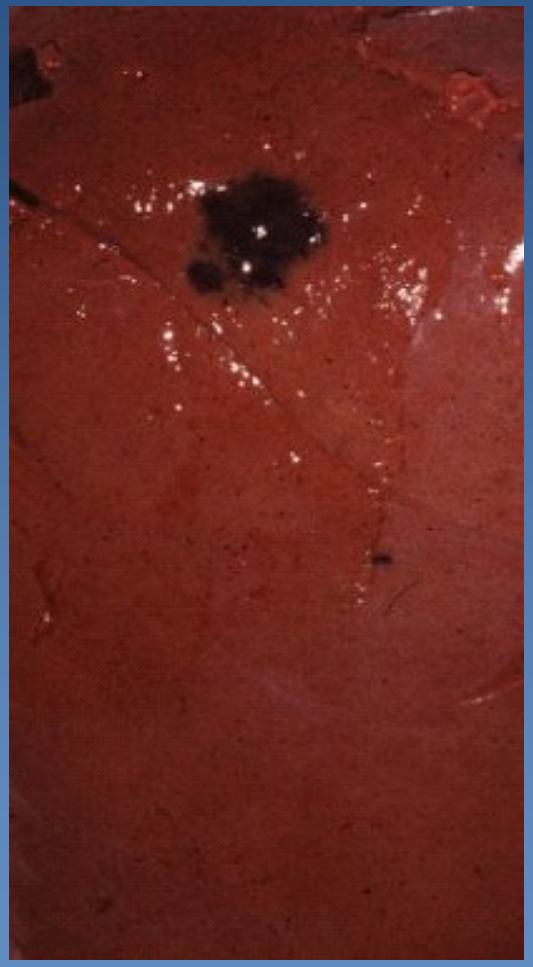
1 - capillaries
2 - endothelium
3 – red blood cells

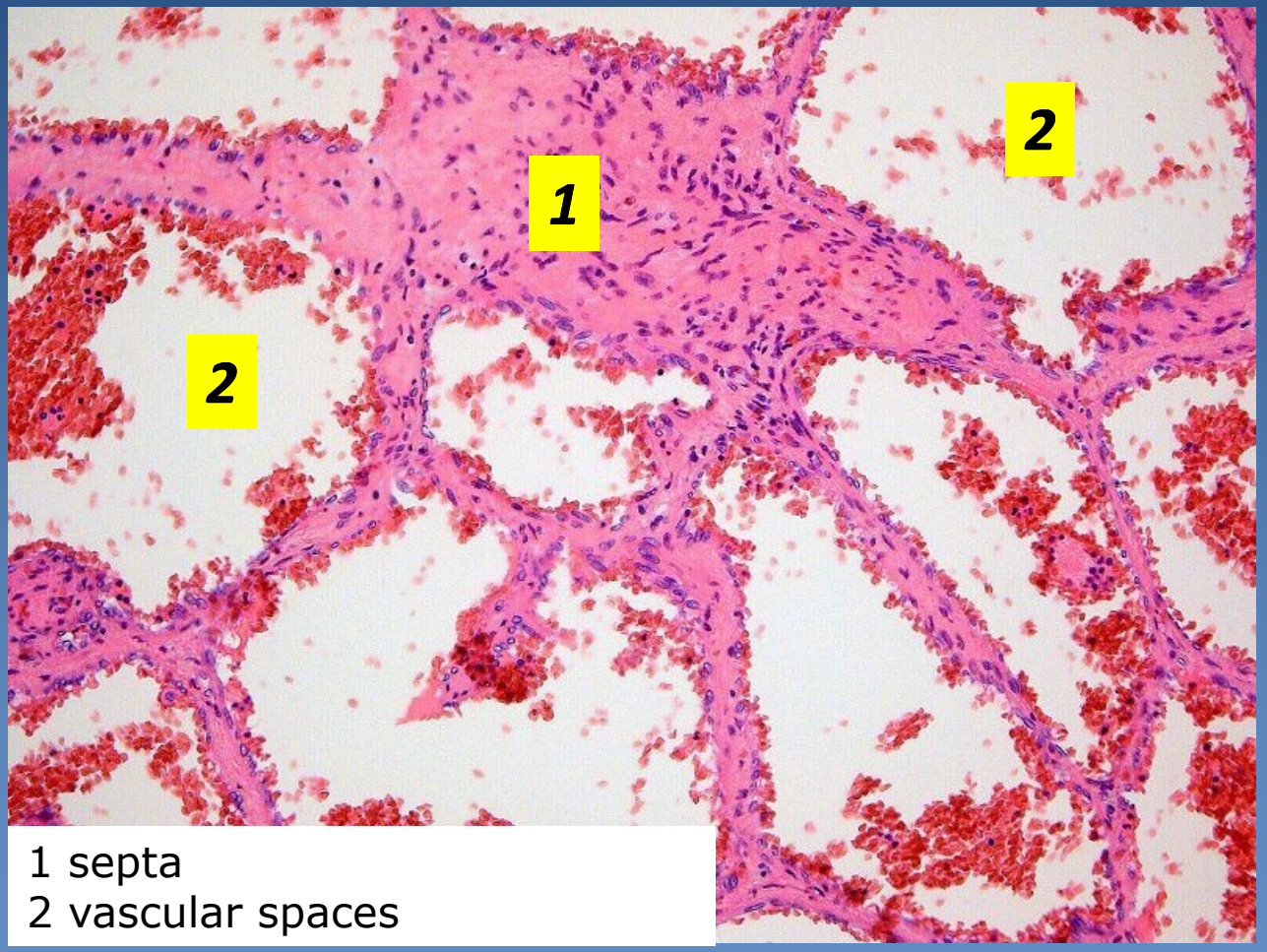
Cavernous hemangioma



- gross:
  - red -blue focus (nodular)
  - possible large size (-15 cm)
  - liver, spleen, skin; commonly multiple
- micro:
  - large blood-filled vascular spaces divided by fibrous septa

Cavernous hemangioma

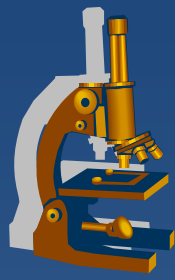




1 septa
2 vascular spaces

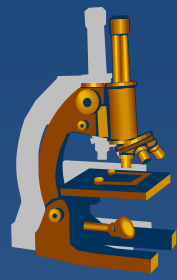
Kaposi sarcoma

Kaposi sarcoma



- **classic form**
  - ⇨ *chronic*
  - ⇨ *in mediterranean or jewish origin*
  - ⇨ *usually (90%) confined to skin*
- **endemic**
  - ⇨ *south-african children*
  - ⇨ *lymphadenopatic*
  - ⇨ *aggressive*
- **immunosuppression (transplant) associated**
  - ⇨ *– internal organs in 50%*
- **AIDS associated**

Kaposi sarcoma

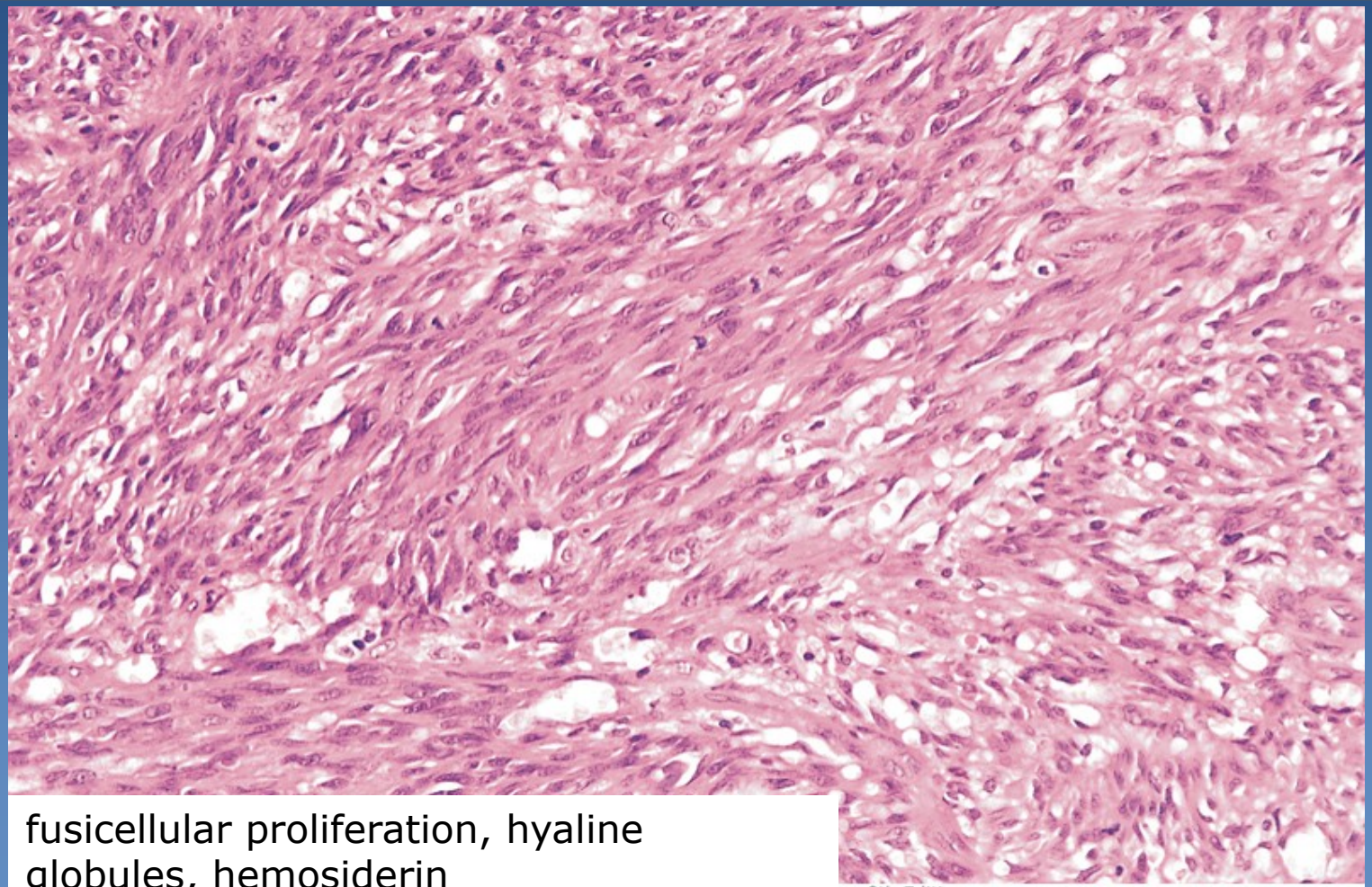


- HHV-8
- hyperproliferation of endothelial cells
- prevention of apoptosis

- **gross**:
  - ⇨ ***red to purple patches***
  - ⇨ ***raised plaques***
  - ⇨ ***nodules***

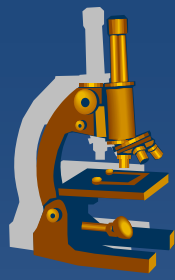
- **micro**:
  - ⇨***irregular blood spaces***
  - ⇨ ***plump atypical endothelial cells***
  - ⇨***perivascular aggregates of spindle cells***

Kaposi sarcoma



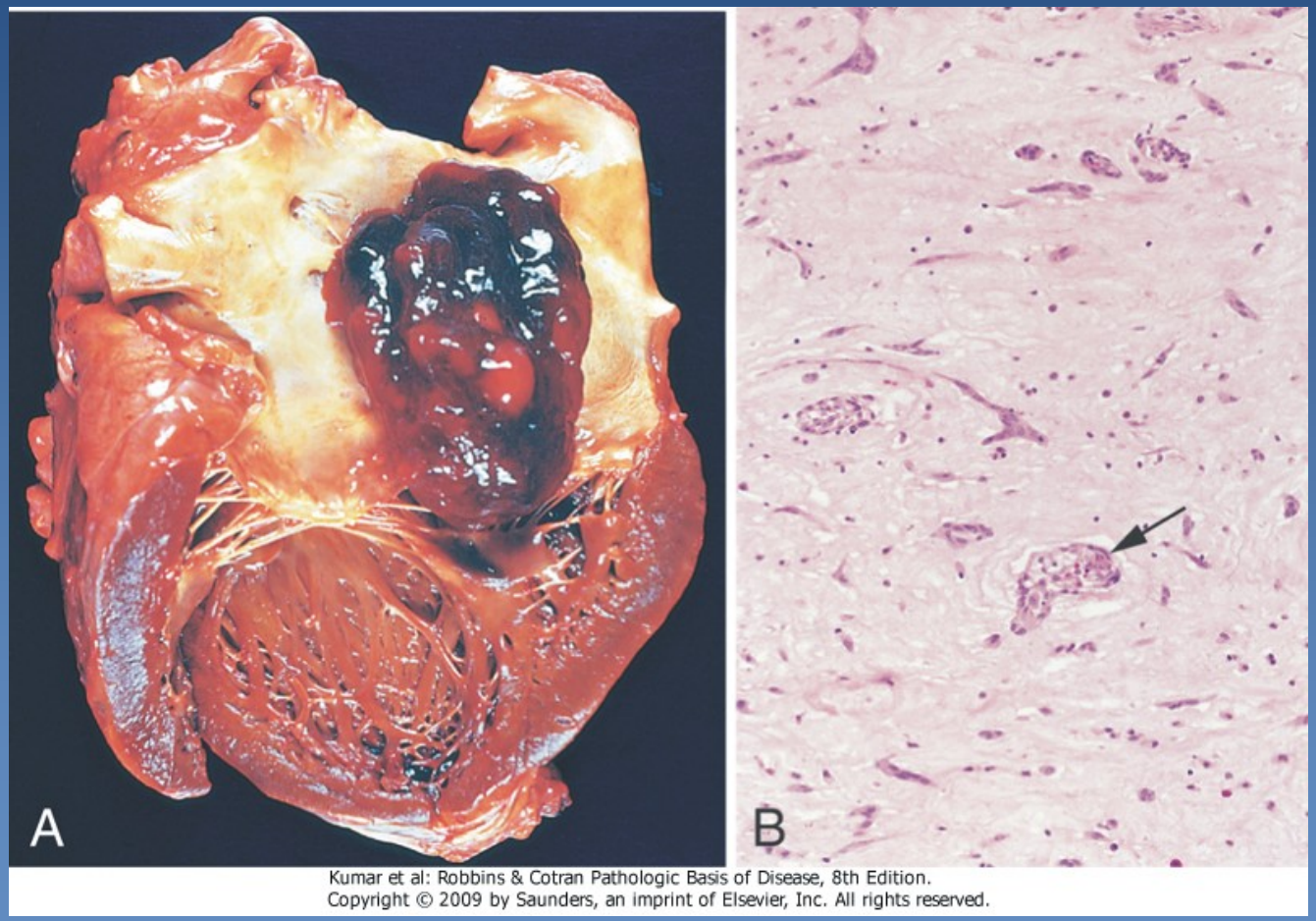
fusicellular proliferation, hyaline globules, hemosiderin

Heart tumors

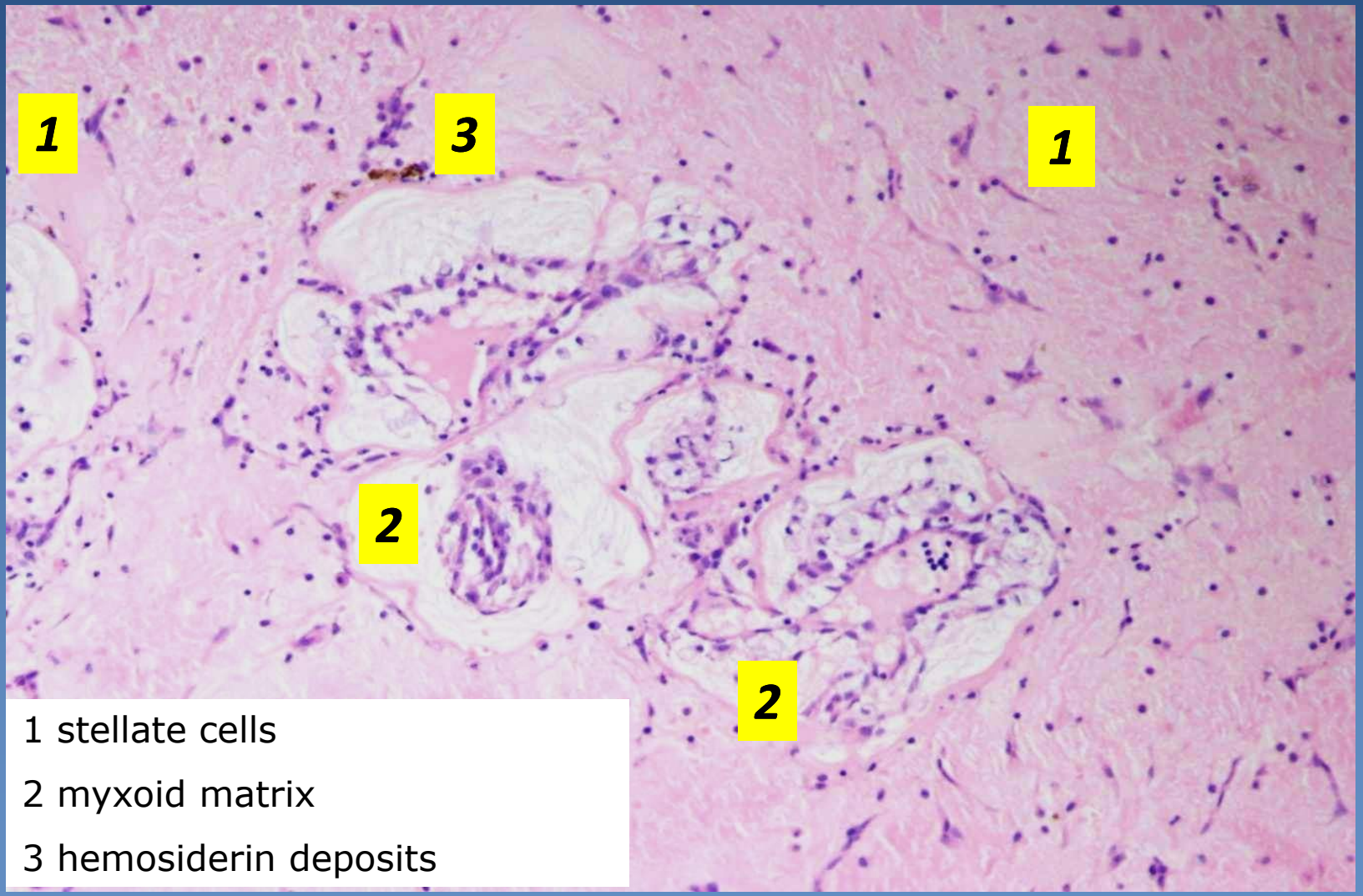


- primary tumors rare, mostly **benign myxomas**
- malignant mesenchymal (sarcomas)
  - ⇨ *leiomyo - , rhabdomyo - , hemangio - , fibrosarcoma*
- secondary tumors
  - ⇨ *20-30 x more common than primary*
  - ⇨ *metastases + infiltrates : lung, breast carcinomas, malignant melanoma, malignant lymphomas and leukemias*
  - ⇨ *direct spread (lung ca, mesothelioma, renal ca)*
  - ⇨ *pericarditis carcinomatosa – hemorrhagic effusion*

LV myxoma



Myxoma (100x)

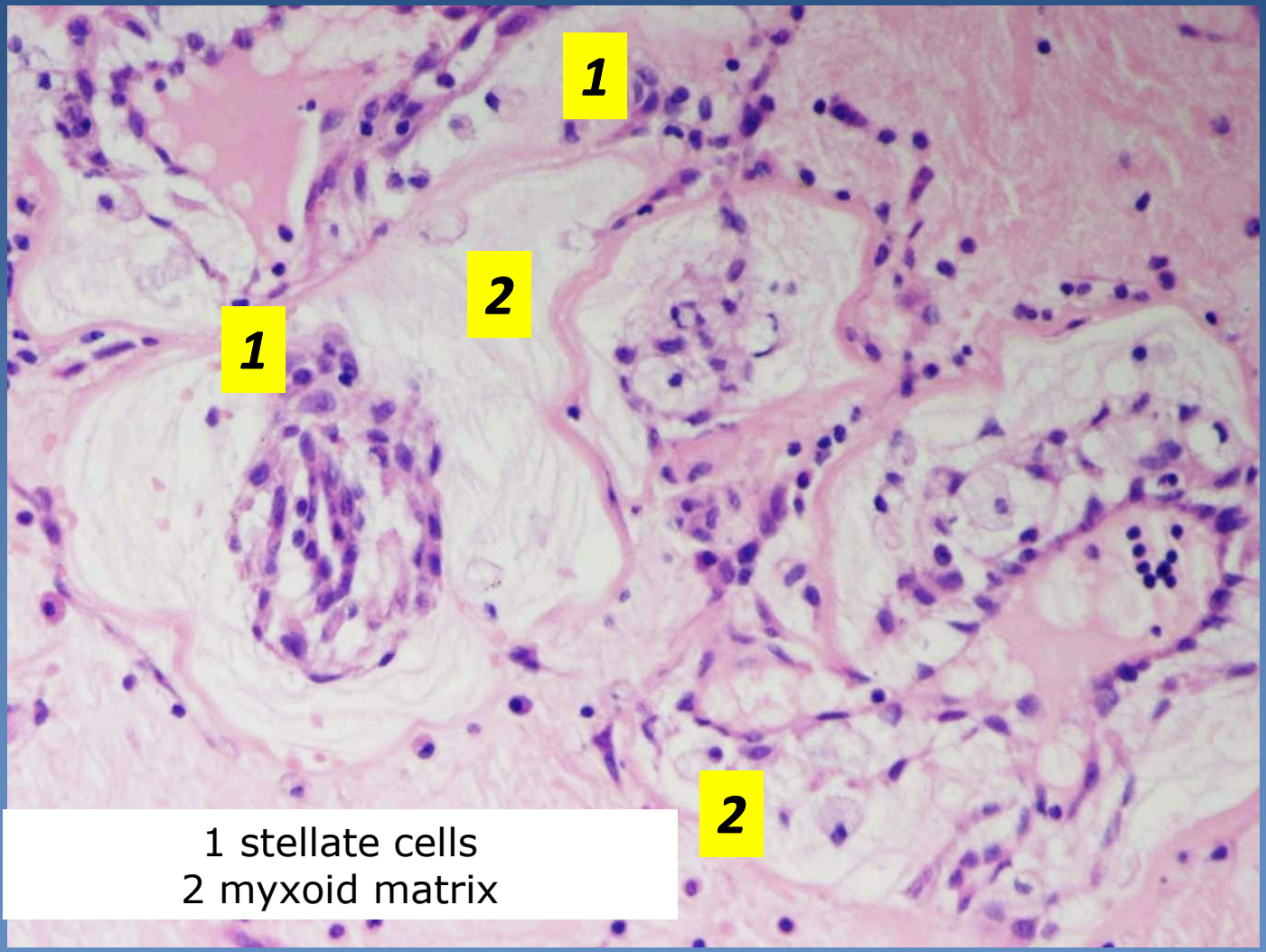


1 stellate cells

2 myxoid matrix

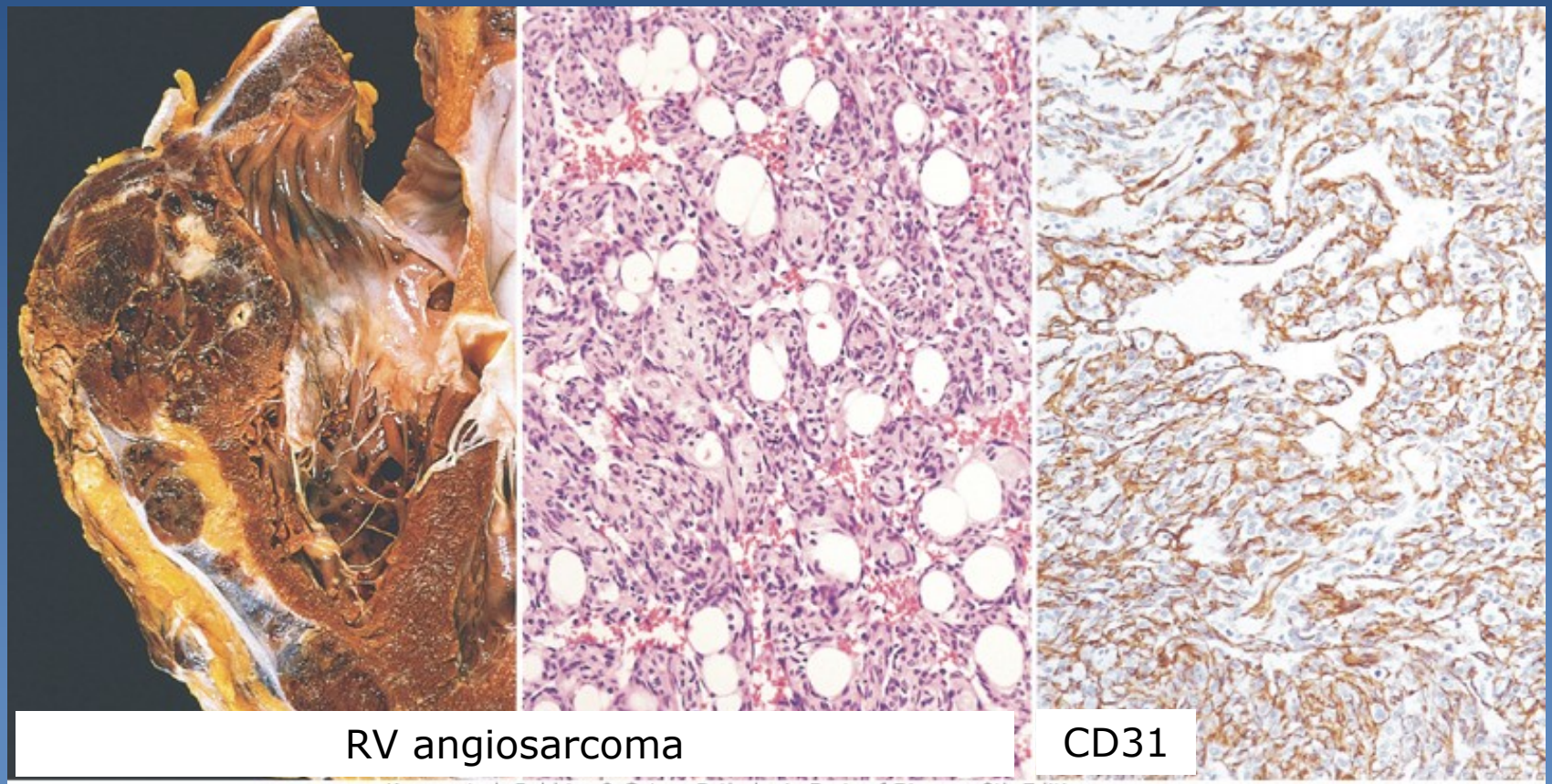
3 hemosiderin deposits

Myxoma (400x)



1 stellate cells
2 myxoid matrix

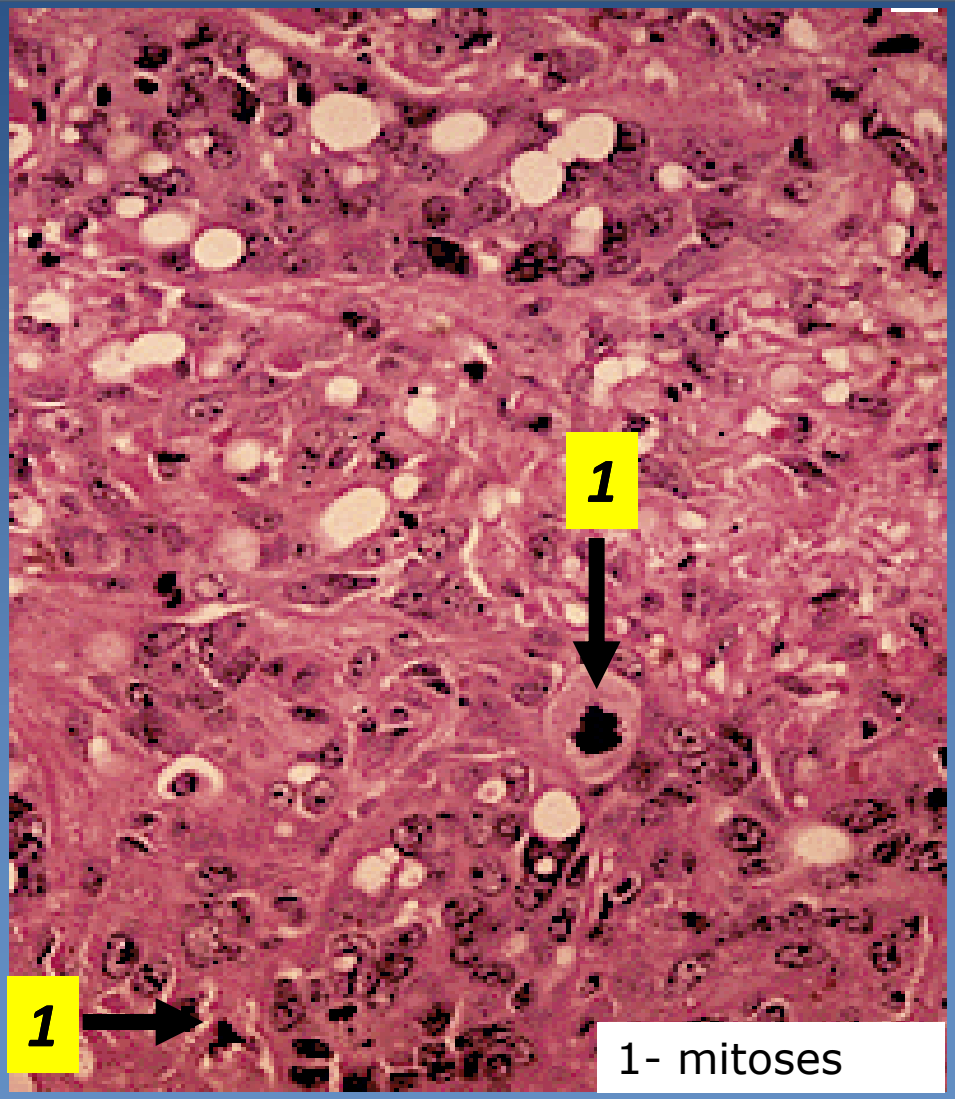
Angiosarcoma



RV angiosarcoma

CD31

Angiosarcoma



1- mitoses