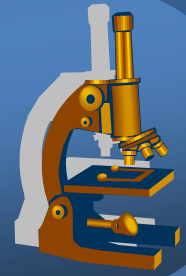


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# *Systemic Pathology*



***CARDIOVASCULAR  
system***

# ATHEROSCLEROSIS



- disease of large and medium-sizes arteries with lipid deposition into intima
- active inflammatory process
- endogenous risk factors, mostly noninfluenceables :
  - *age, MxF* (estrogen?), *familiar factors* (f. hypercholesterolemia), *hereditary homocysteinemia*
- exogenous 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* ( $\uparrow$ BP, turbulence)
- *endotoxins, immune complexes, exogenous toxins (cig. smoke),  $\uparrow$  cholesterol*

$\uparrow$  *expression of cell adhesion molecules,  $\uparrow$  permeability,  $\uparrow$  thrombogenicity*

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

## 3. Inflammation

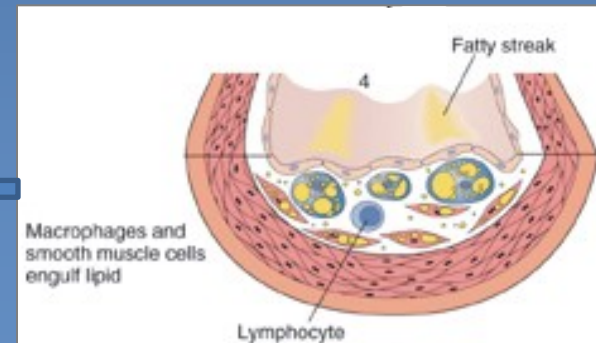
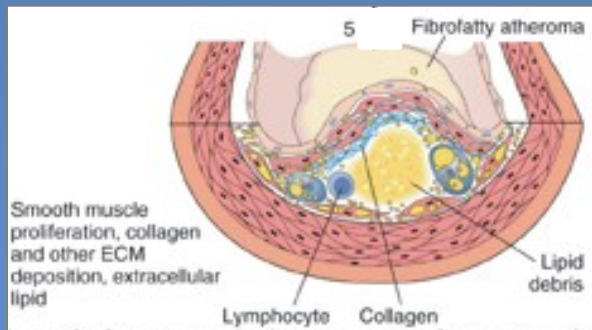
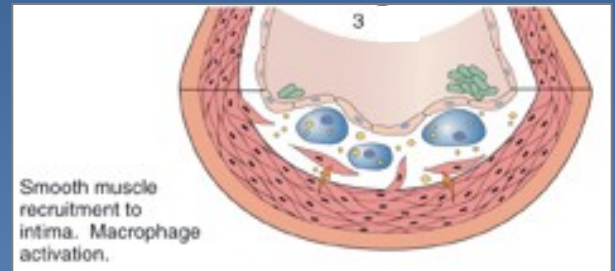
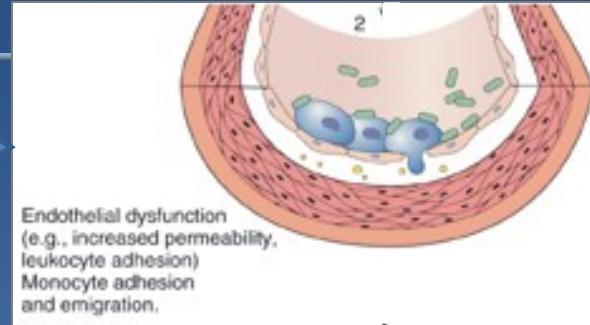
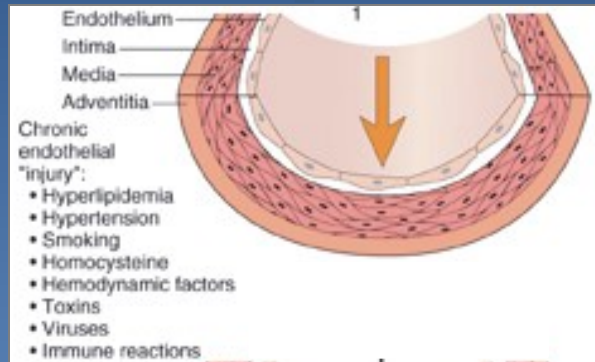
- *blood monocytes ( $\rightarrow$ foam cells), T-cells, platelets, smooth muscle cells*

## 4. Repair - proliferation of myointimal cells

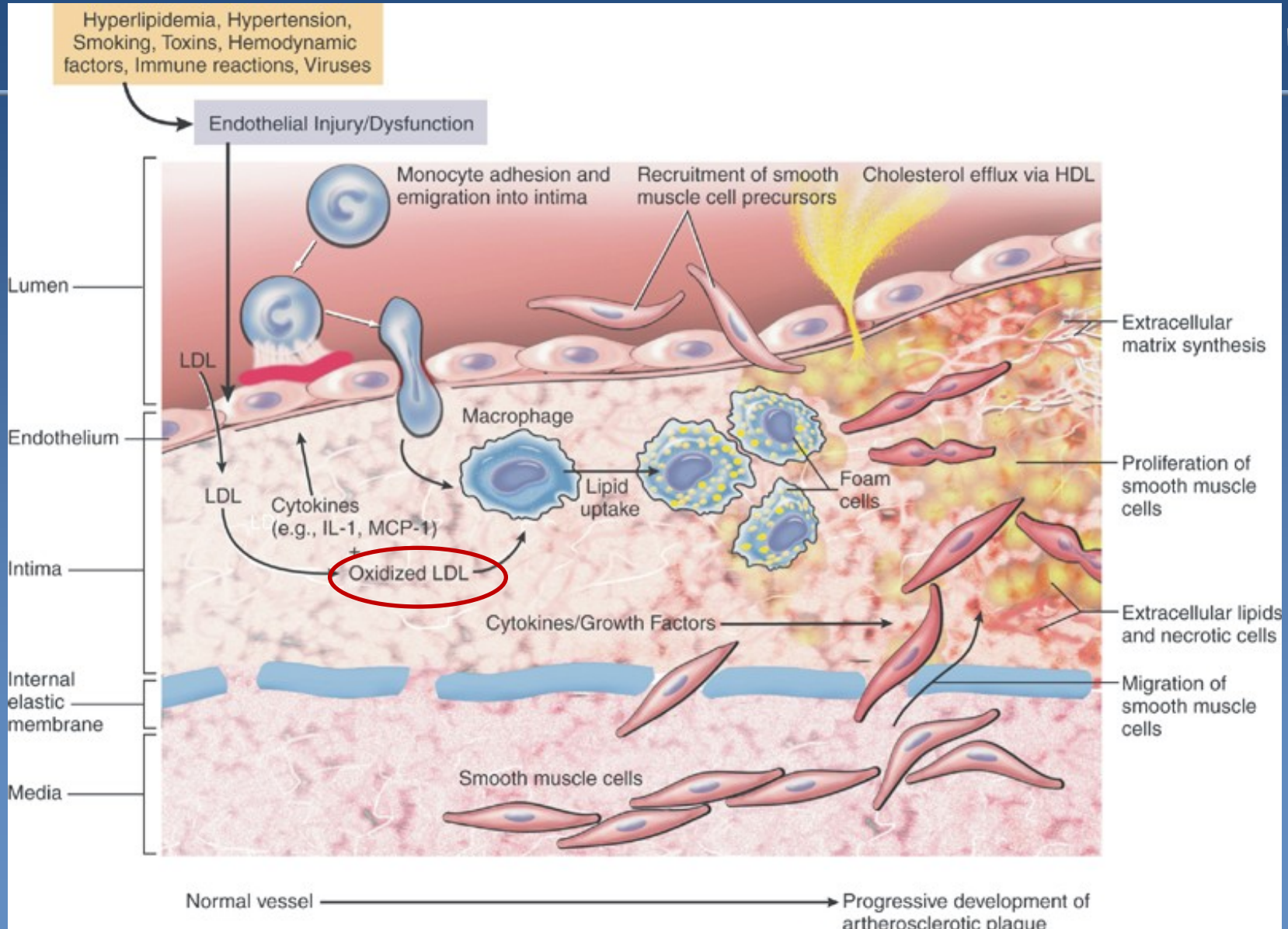
- *synthesis of collagen, elastin, proteoglycans  $\rightarrow$  **fibrotic plaque**, + lipid accumulation - **atheromatous plaque***

*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)

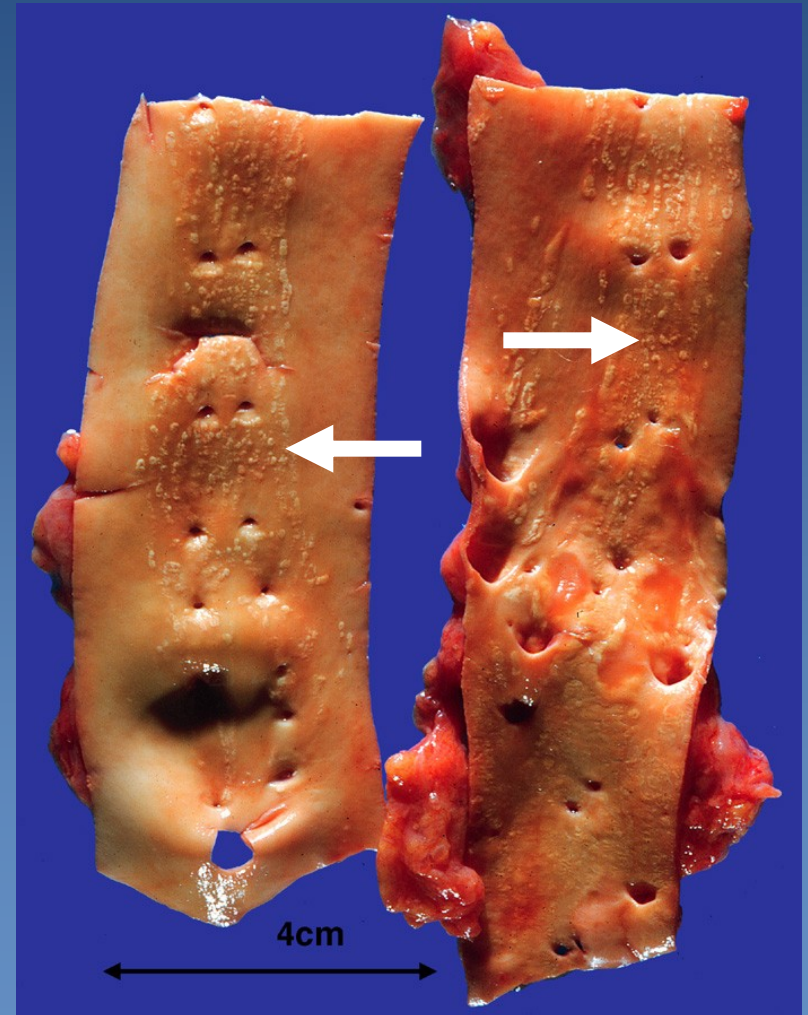
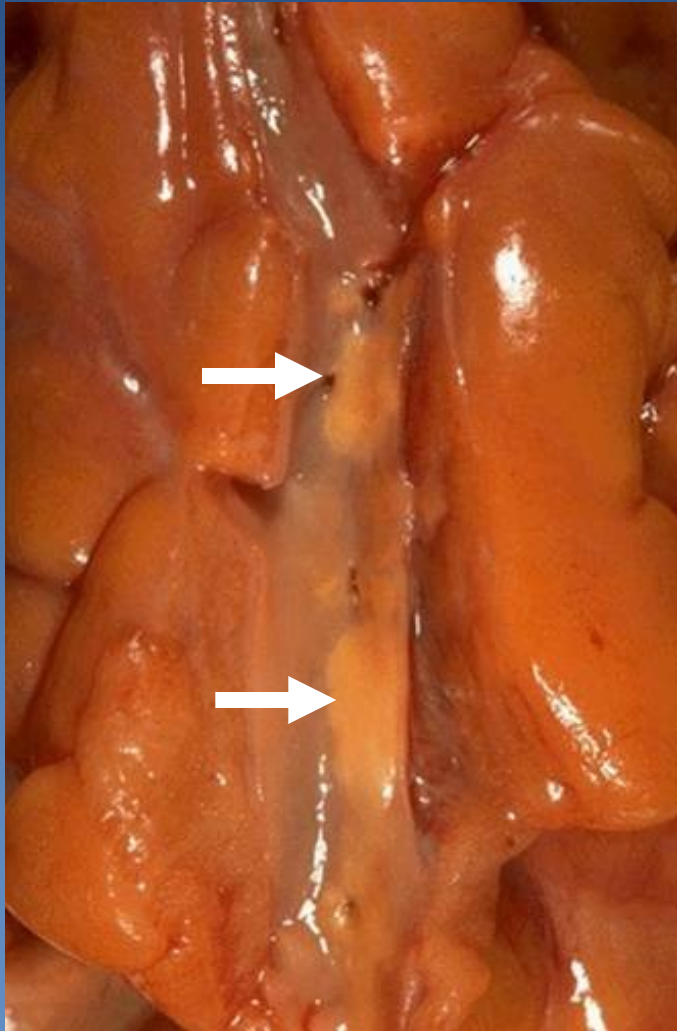
# *Atherosclerosis*



SEQUELS: arterial occlusion in situ

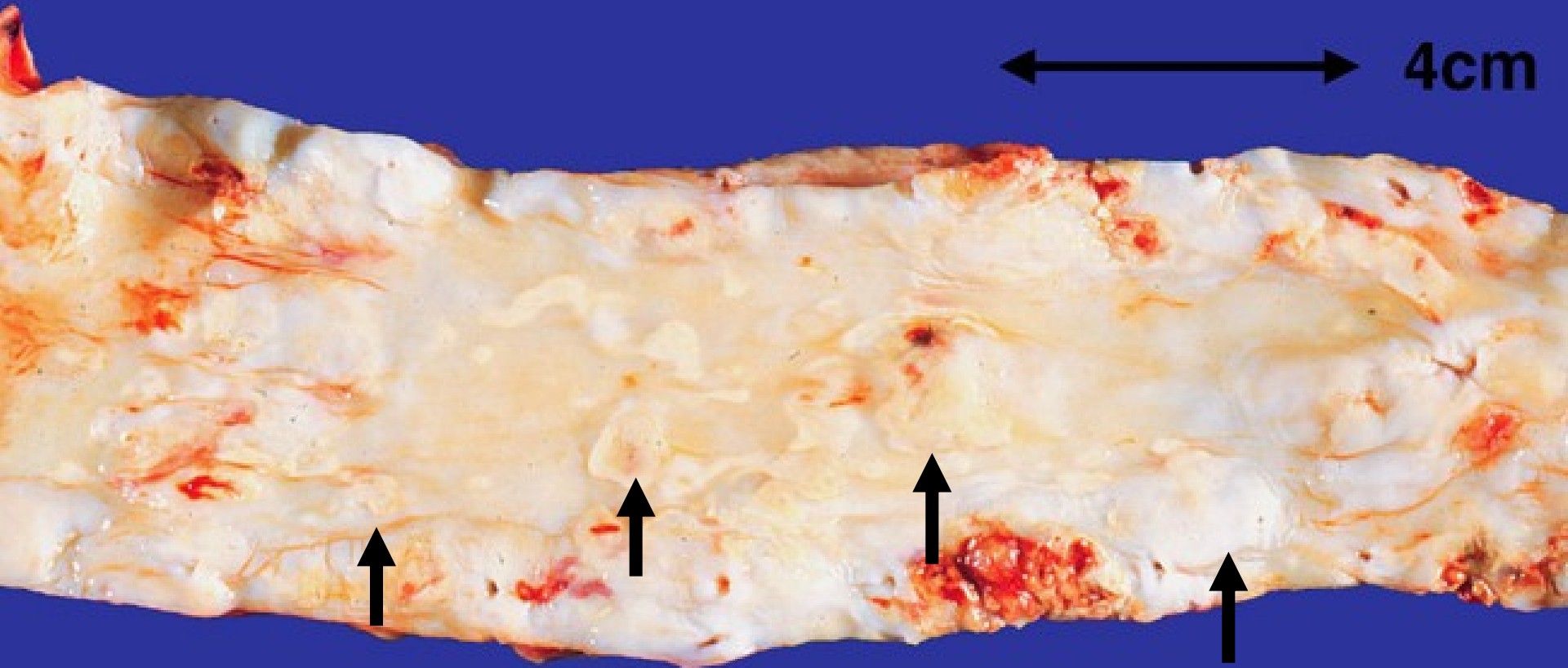
- ✘ chronic (→ hypoxia, atrophy)
- ✘ acute (→ ischemia, infarction, encephalomalatia)
- ✘ embolism (thrombus, plaque material)
- ✘ weakening of arterial wall (aneurysm), risk of rupture
- ✘ bleeding (from plaque, fissured wall)
- ✘ calcification (hypertensive factor)

# *Atherosclerosis- fatty streak*

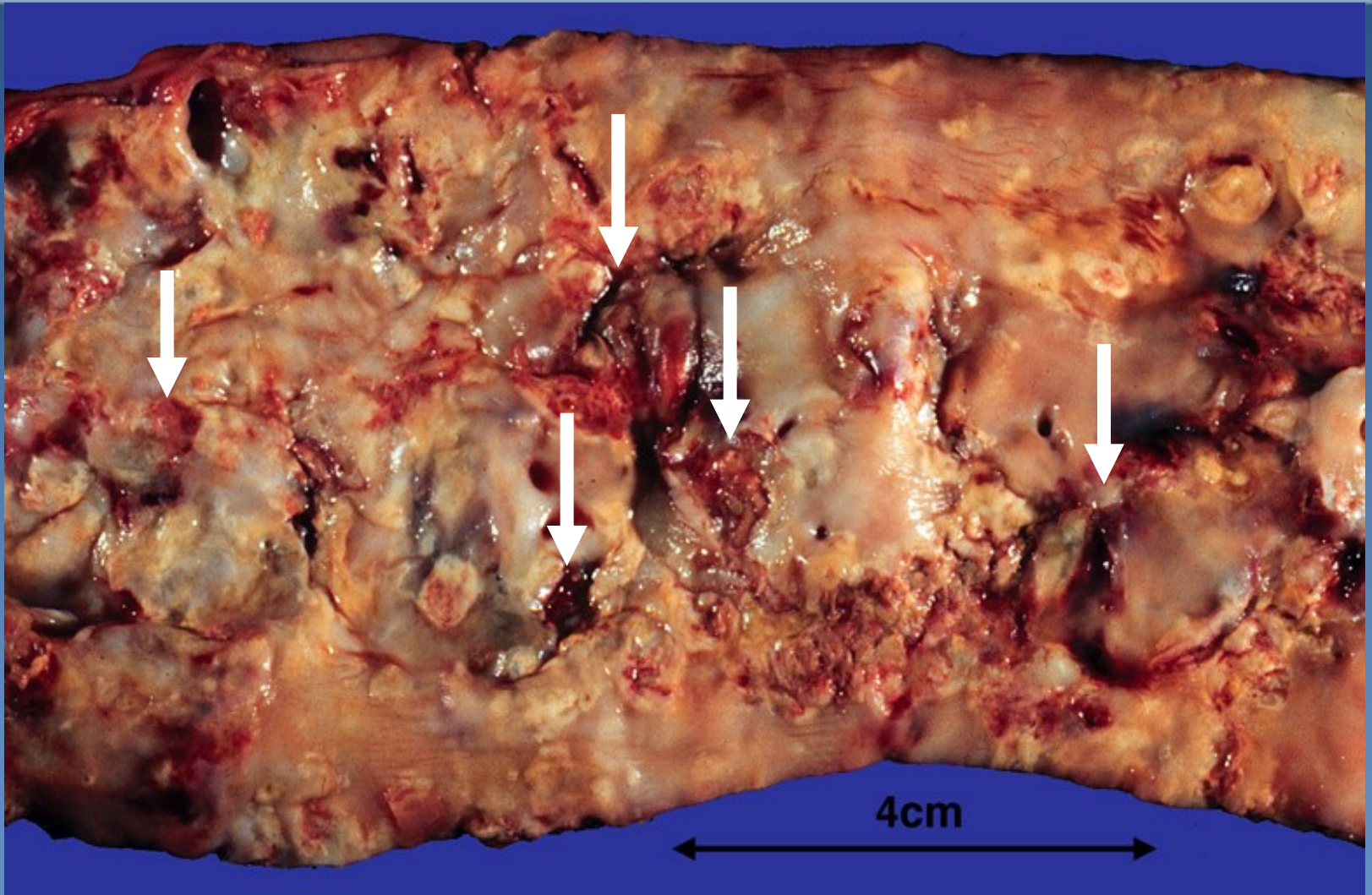
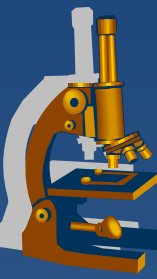




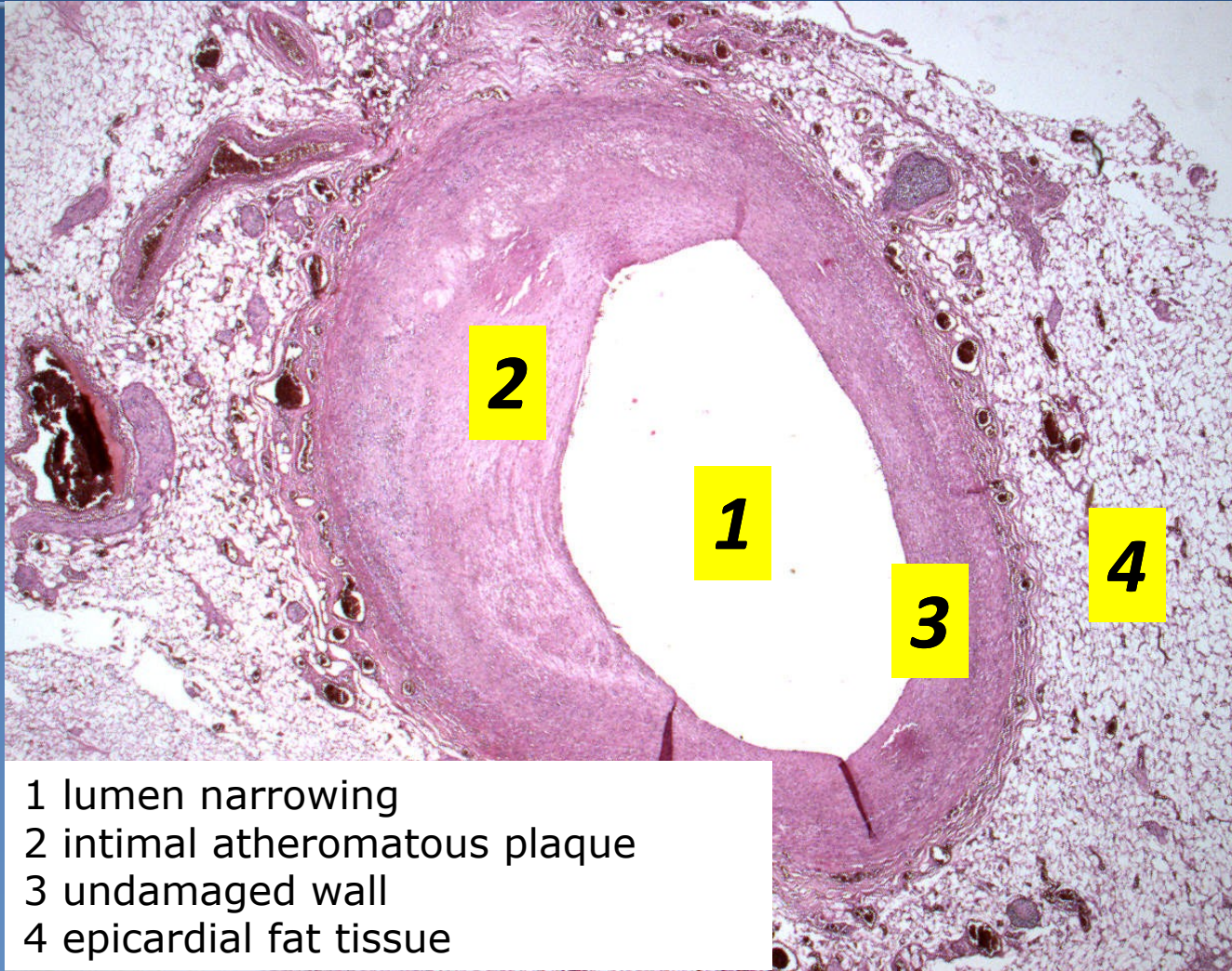
# ***Atherosclerosis - fibrous and atheromatous plaques***



# *Atherosclerosis- plaque ulceration, mural thrombosis*

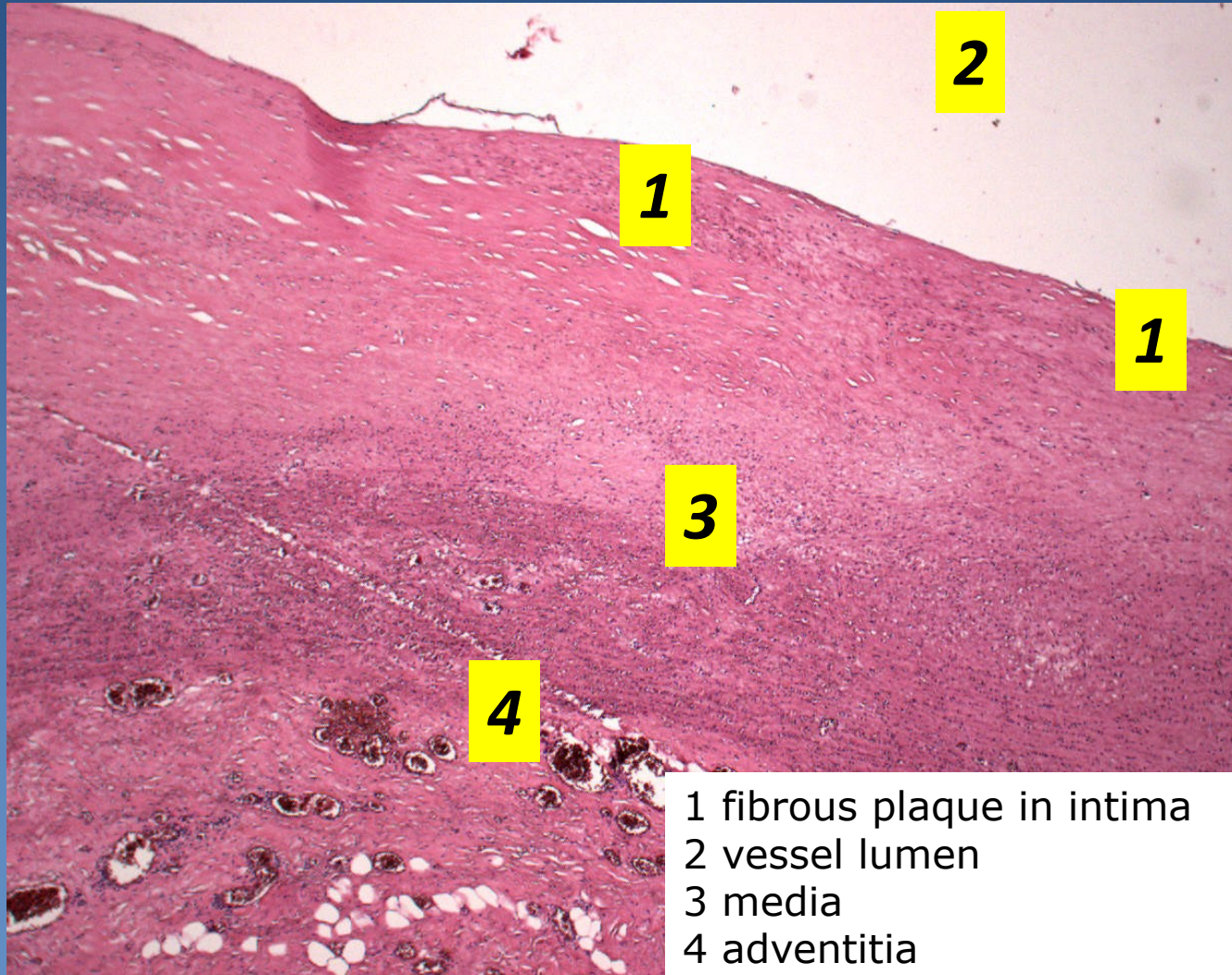


# *Atherosclerosis- coronary artery*

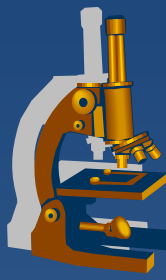


- 1 lumen narrowing
- 2 intimal atheromatous plaque
- 3 undamaged wall
- 4 epicardial fat tissue

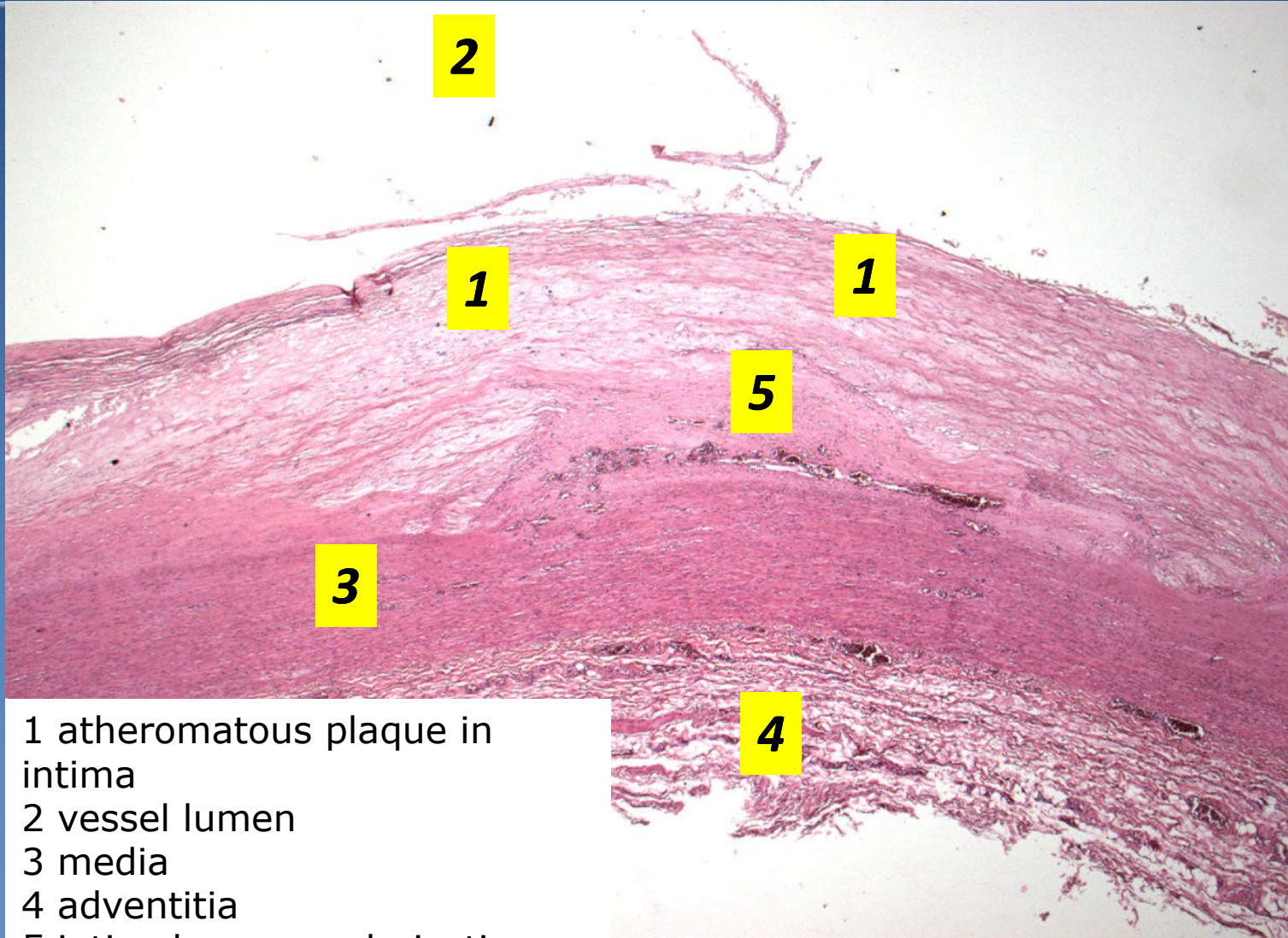
# *Atherosclerosis – fibrous plaque*



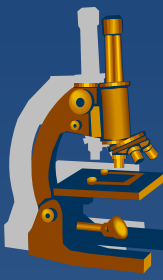
- 1 fibrous plaque in intima
- 2 vessel lumen
- 3 media
- 4 adventitia



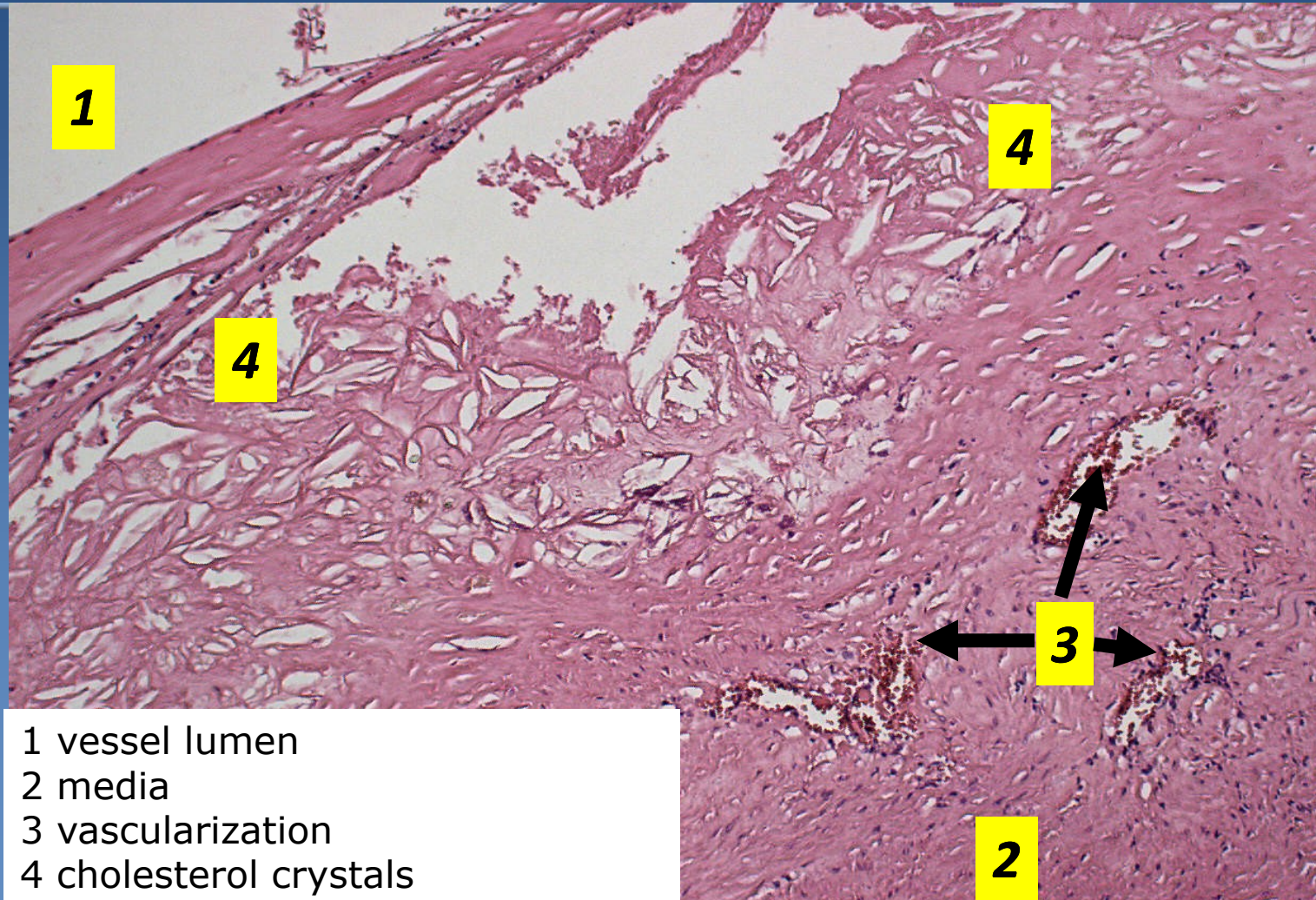
# *Atherosclerosis - atheromatous plaque*



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

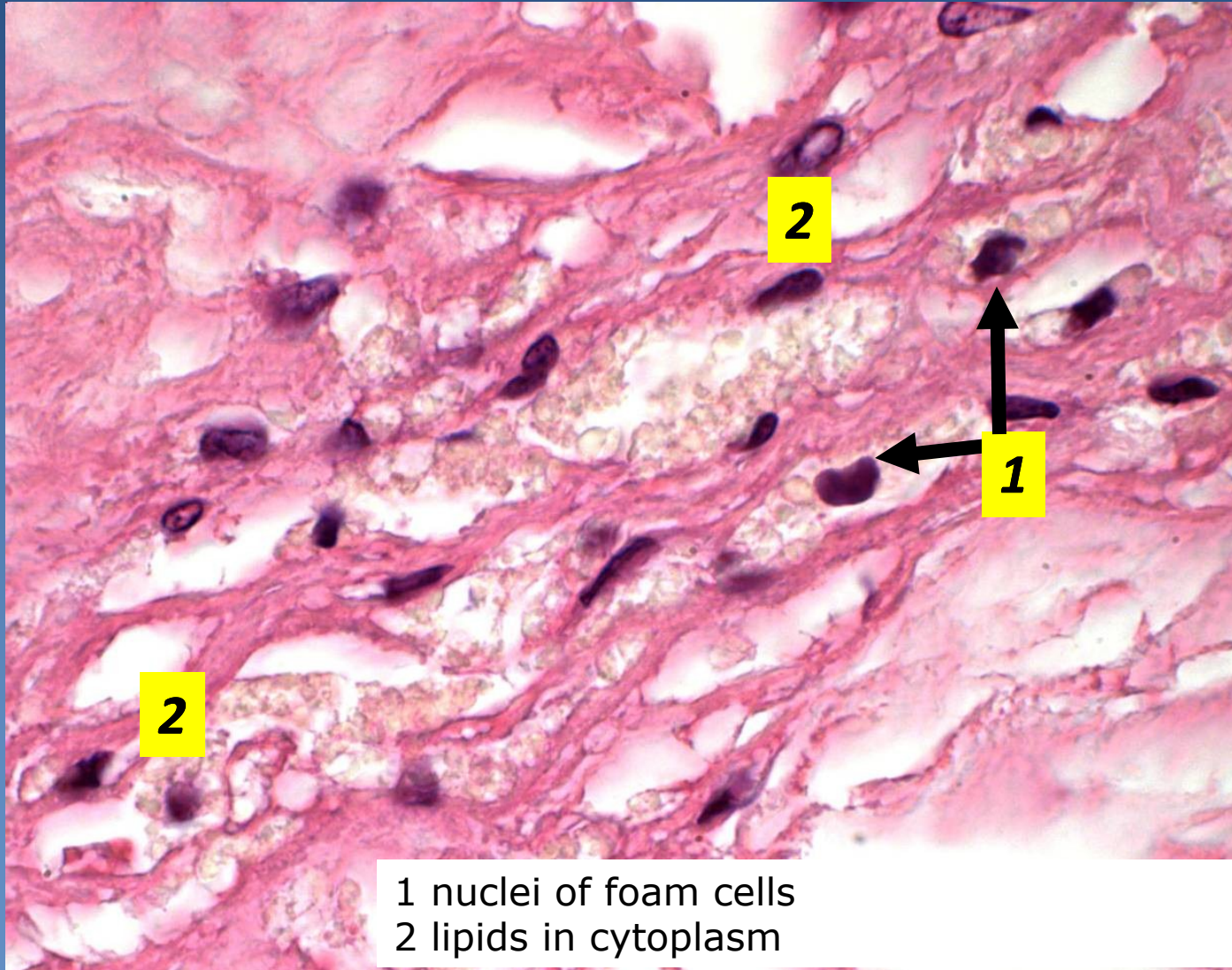


# *Atherosclerosis - atheromatous plaque, intimal neovascularization*

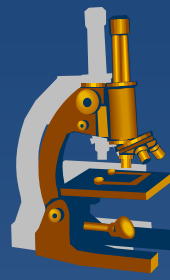


- 1 vessel lumen
- 2 media
- 3 vascularization
- 4 cholesterol crystals

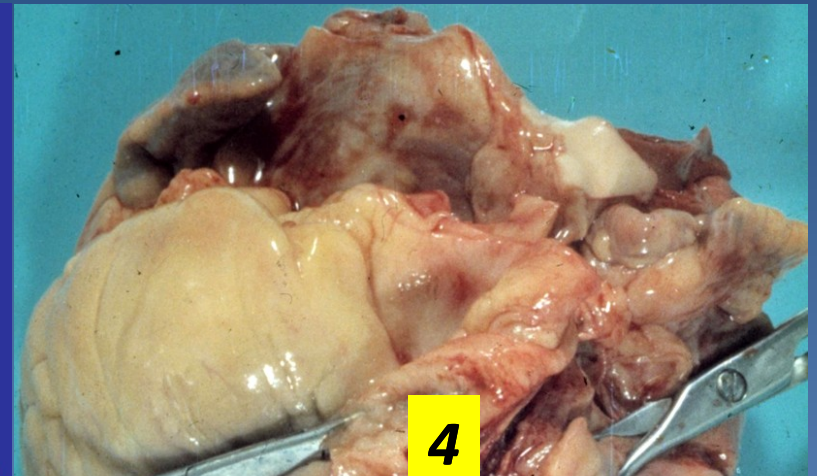
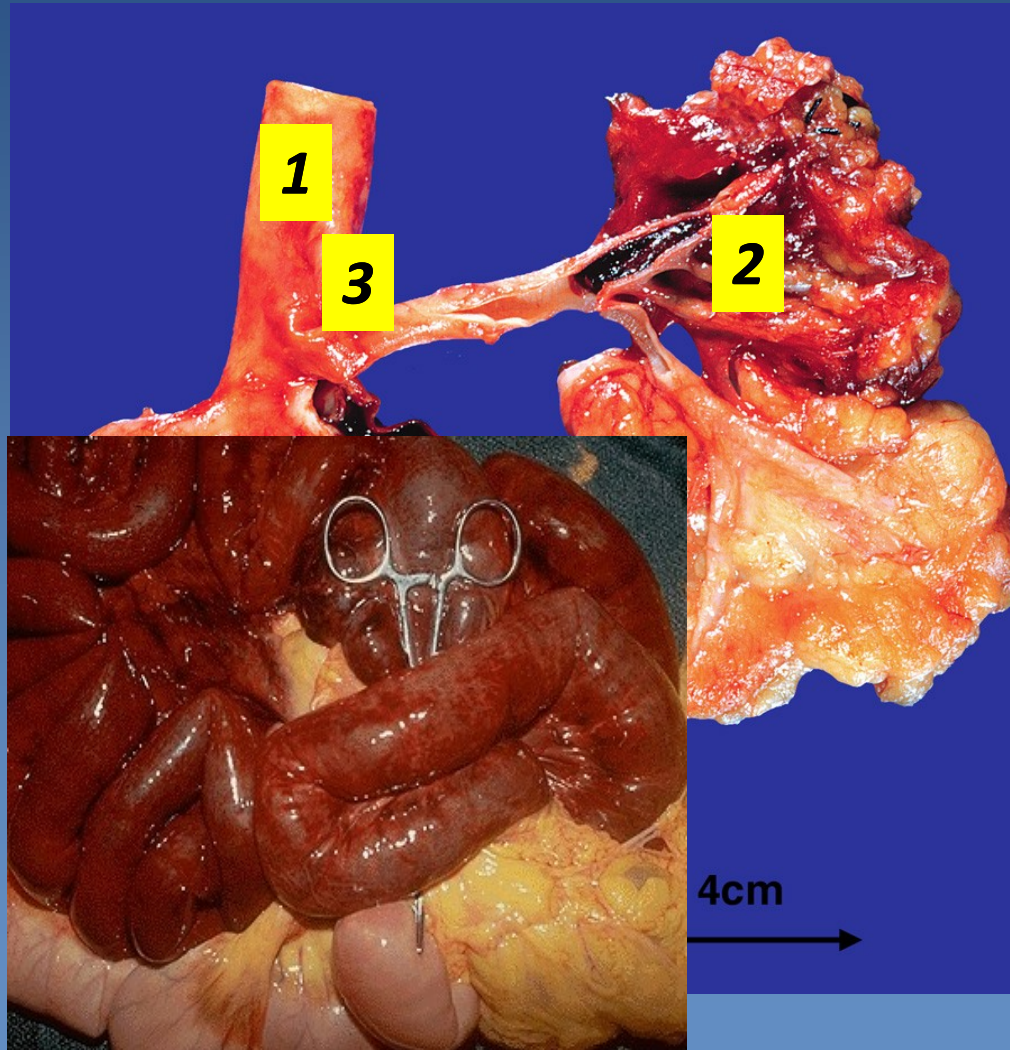
# *Atherosclerosis – foam cells in atheromatous plaque*



1 nuclei of foam cells  
2 lipids in cytoplasm



# *Atherosclerosis - complications thrombosis/thrombembolia*



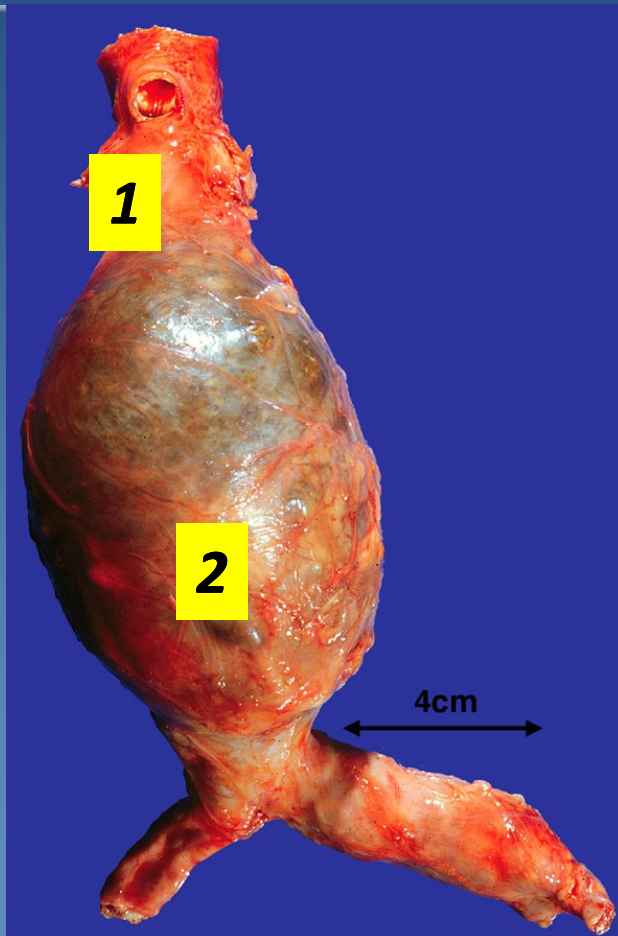


# *aneurysm*

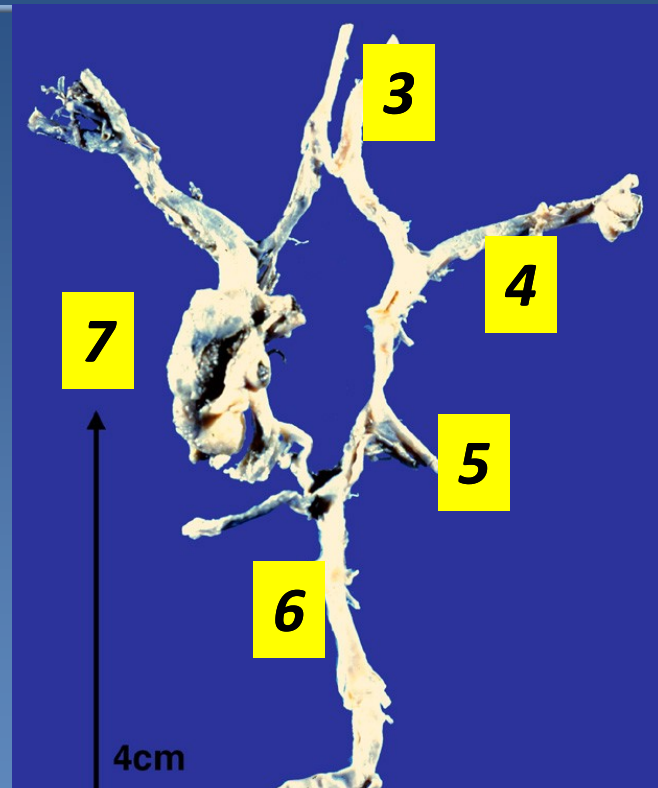


- ✗ localized, blood-filled balloon-like bulge in the wall of a blood vessel.
  - ⇒ *the circle of Willis in the brain, thoracic and abdominal aortic aneurysm*
- ✗ atherosclerotic aneurysm x syphilitic
- ✗ etiology:
  - ⇒ *hereditary defects in the structure, atherosclerosis, inflammation, disease process,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

# Atherosclerosis - complications - aneurysm



1 abdominal aorta  
2 aneurysm



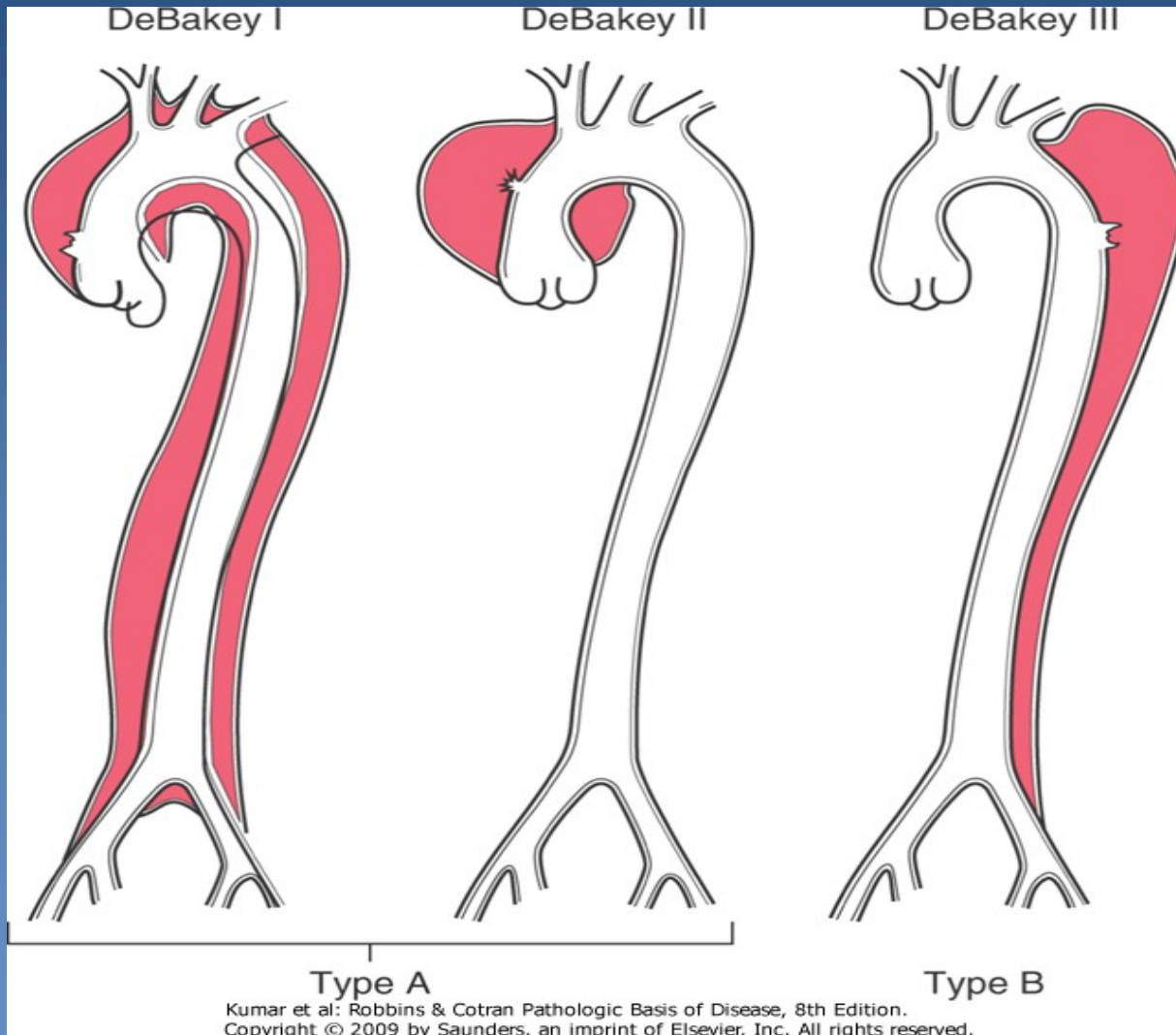
3 a. cerebri anterior  
4 a. cerebri media  
5 a. cerebri posterior  
6 a. basilaris  
7 aneurysm

# Aortic dissection

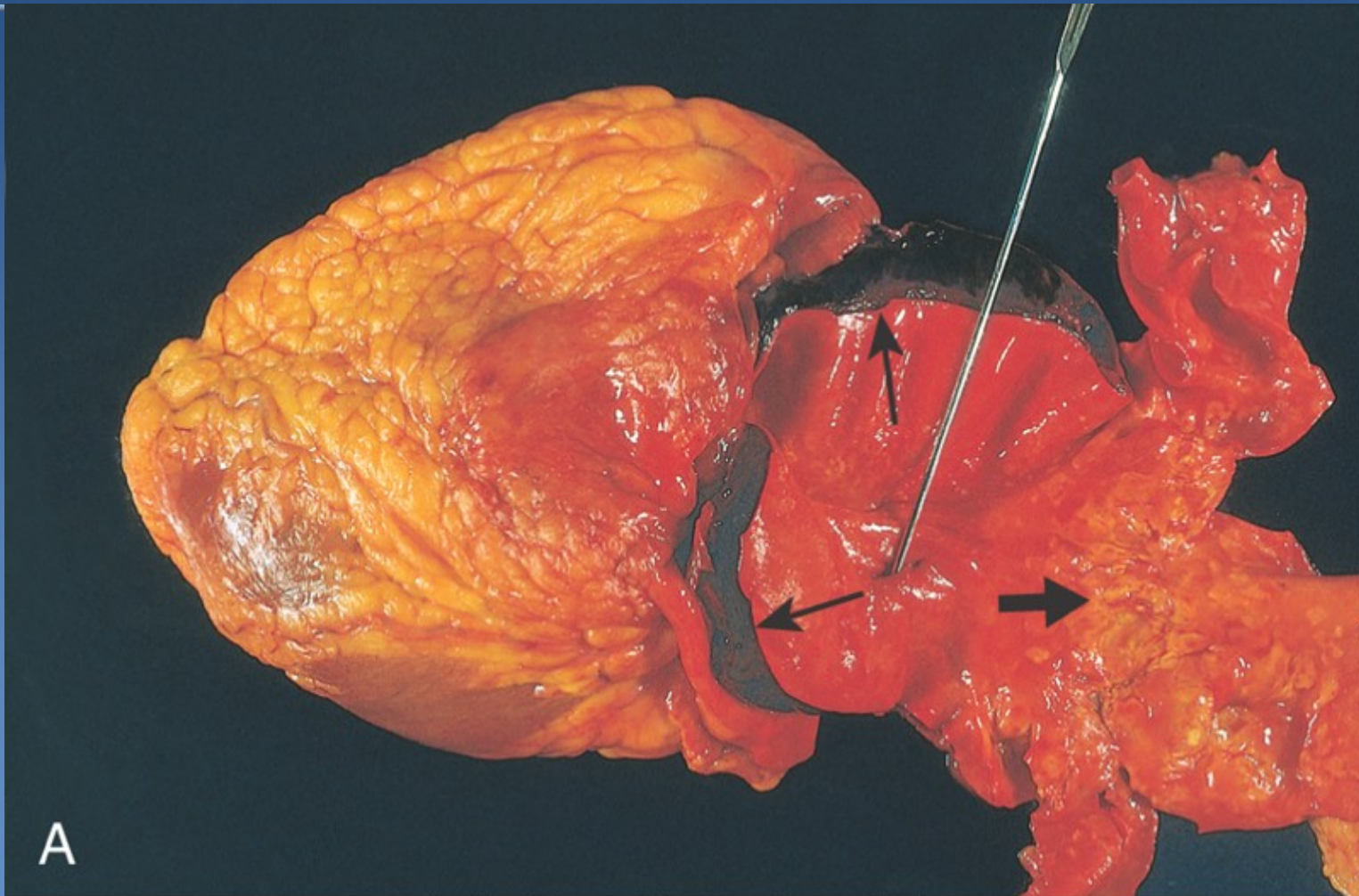


- ✗ tear in aortic intima - intramural bleeding through media, false lumen, possible „double-barreled“ aorta
- ✗ typical in ascending aort, 1–8 cm above aortic valve
- ✗ ante– and retrograde spread to the aortic root
- ✗ common thrombosis in false lumen
- ✗ risk of external rupture (→ **hemoperikardium**),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

# Aortic dissection



# *Aortic dissection*



Kumar et al: Robbins & Cotran Pathologic Basis of Disease, 8th Edition.  
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# 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<sup>+</sup> vasculitis** (dangerous, even fatal within a few years, if not recognised)

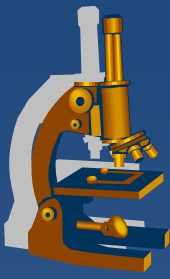
- ⇒ *Wegener granulomatosis*
- ⇒ *Churg-Strauss syndrome*
- ⇒ *microscopic polyangiitis*

× **ANCA<sup>-</sup> 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***

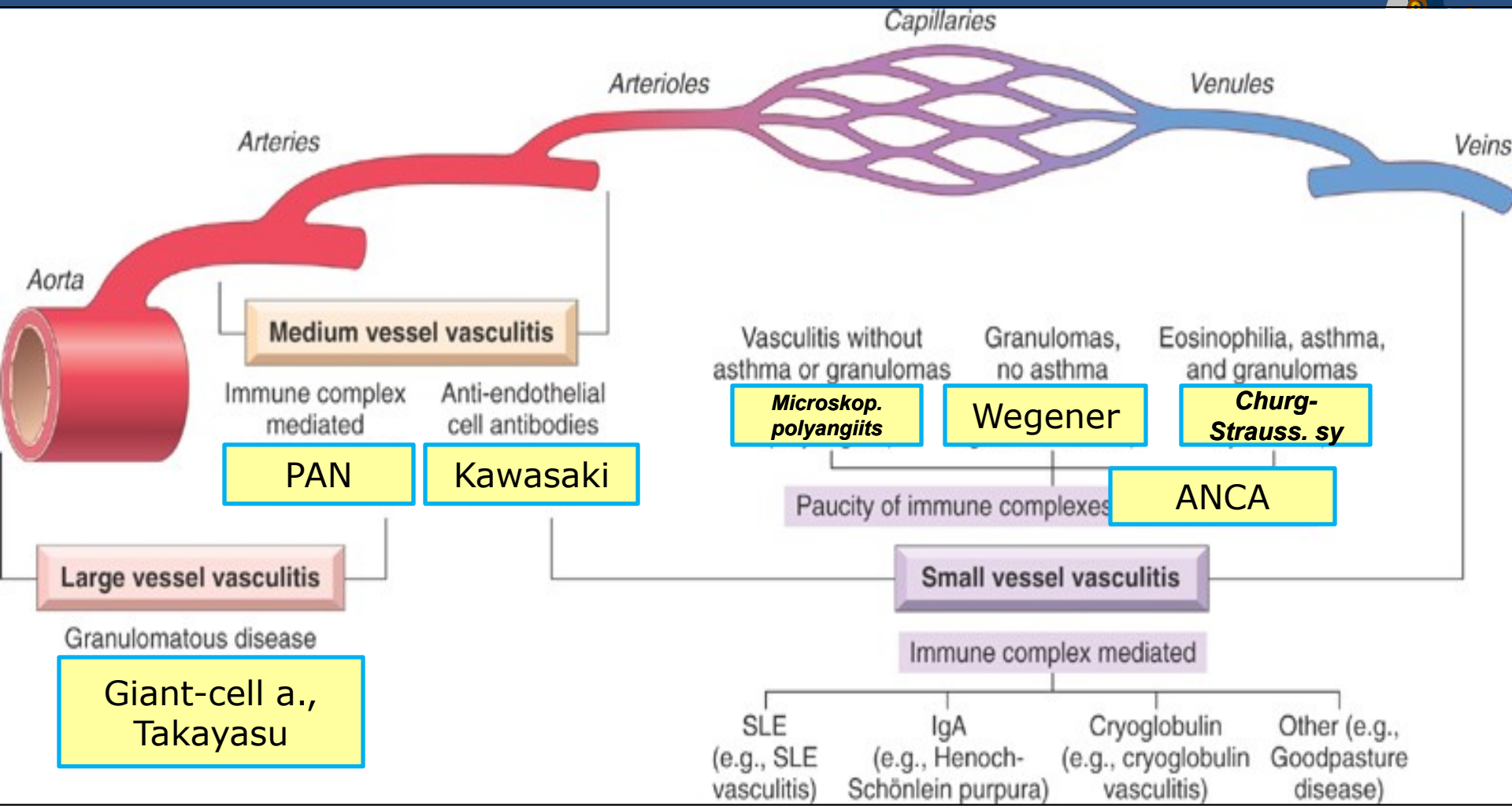
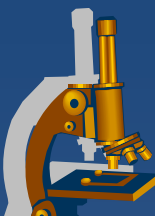
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- fever, nausea, myalgia, arthralgia
  - skin purpura
  - signs of nephritis
  - abdominal pain



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



# ANCA+ vasculitis



## × incidence ?????

⇒ *≤20/1mil. inhabitants*

⇒ *age 65+ - 53/1mil. inhabitants*

## × prognosis:

⇒ *untreated ANCA<sup>+</sup> vasculitis ≥80% fatal in 2 yrs*

⇒ *treated ANCA<sup>+</sup> vasculitis : ≥80% survives 5 yrs*

⇒ *renal failure in elders >70 yrs - in 40% due to ANCA<sup>+</sup> vasculitis*

# *granulomatosis with polyangiitis (Wegener granulomatosis)*



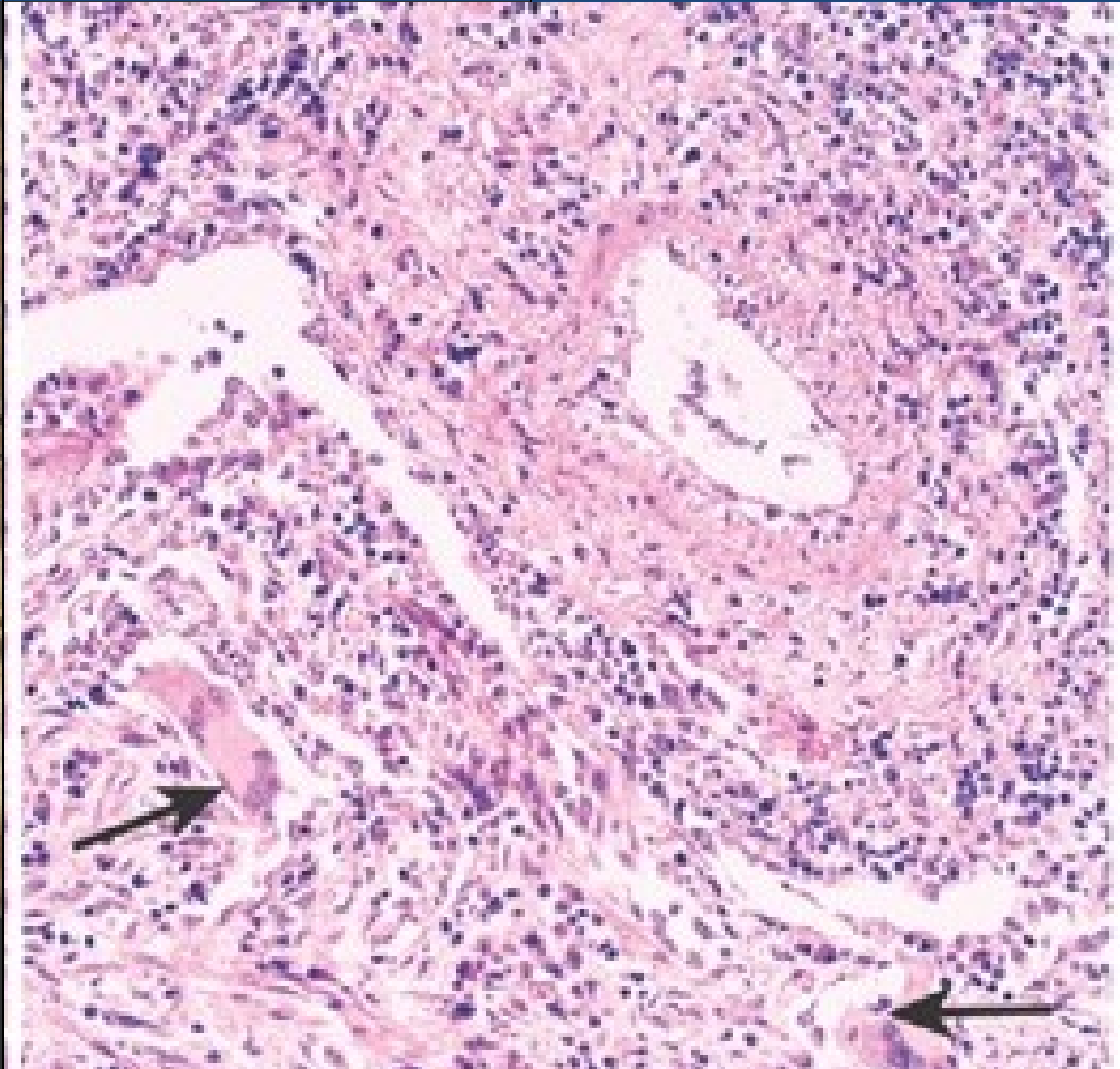
- ✗ clinically as **pneumonitis**, persistent X-ray with bilat. nodular infiltrates, **chronic sinusitis** with mucosal **ulcerations nasopharynx** (sometimes destructive axial structures), **ARI / CHRI** (focal necrosis, sickle cell GLN)

# ***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

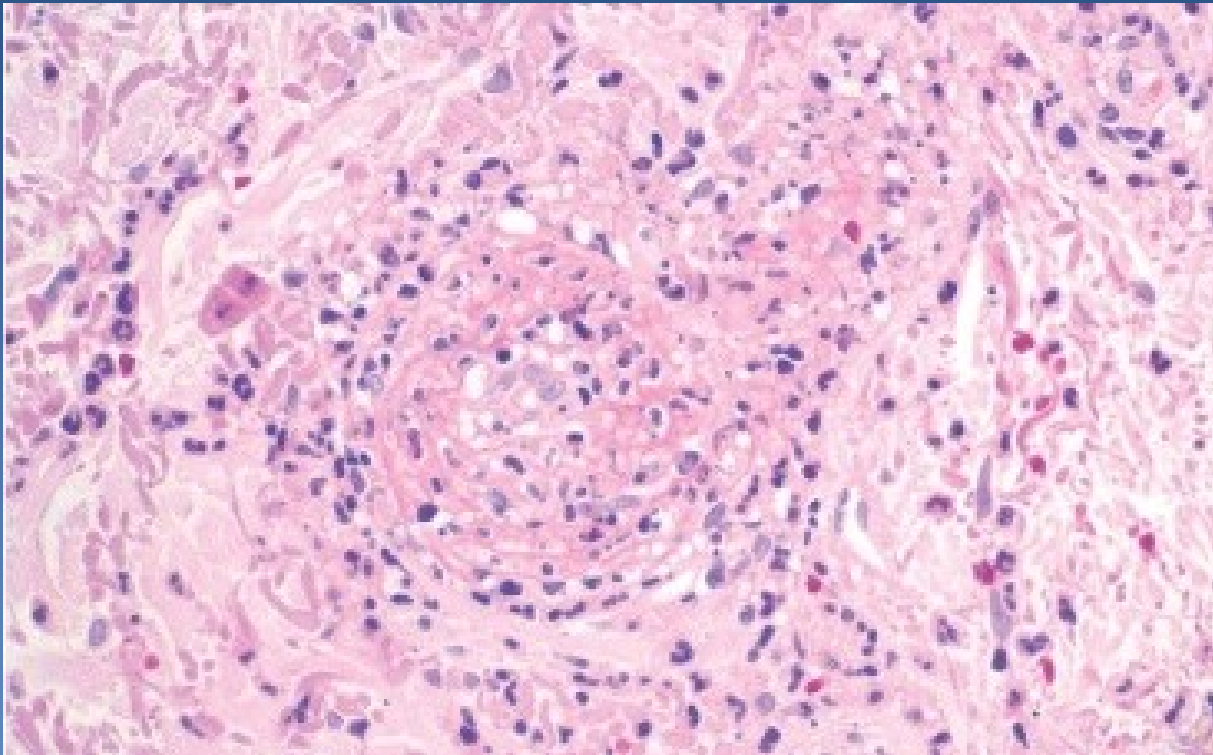
# ANCA+ VASCULITIS: microscopic polyangiitis



- ✗ 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; allergic response to exogenous antigens – bacteria, viruses, drugs)
- ✗ micro:
  - ⇒ *fibrinoid necrosis of vessel wall with neutrophils and chromatin fragments from neutrophil's nuclei - leukocytoclastic*
  - ⇒ *all lesions in the same stage of evolution* (X *polyarteritis nodosa*)

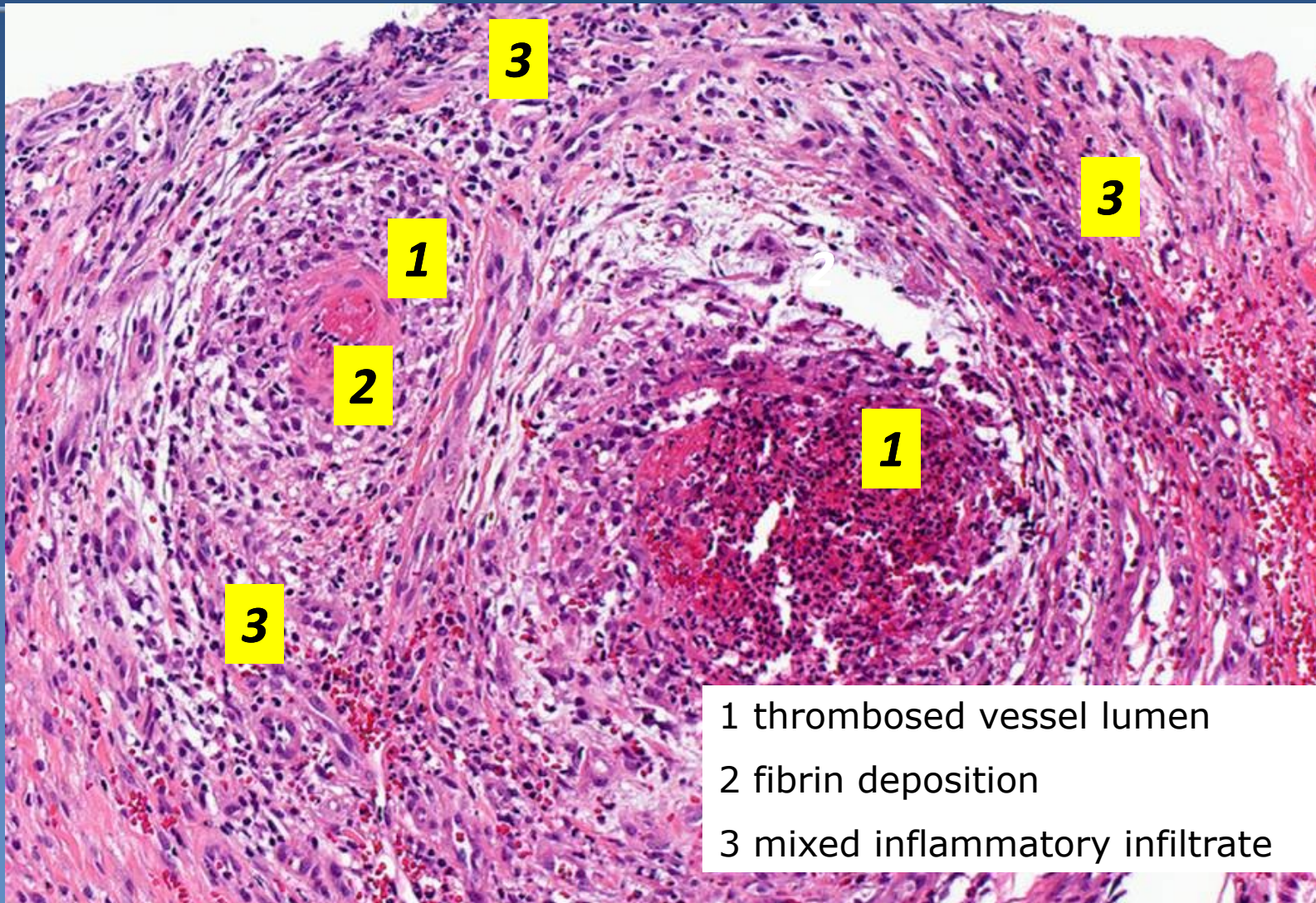


# *leukocytoclastic vasculitis*



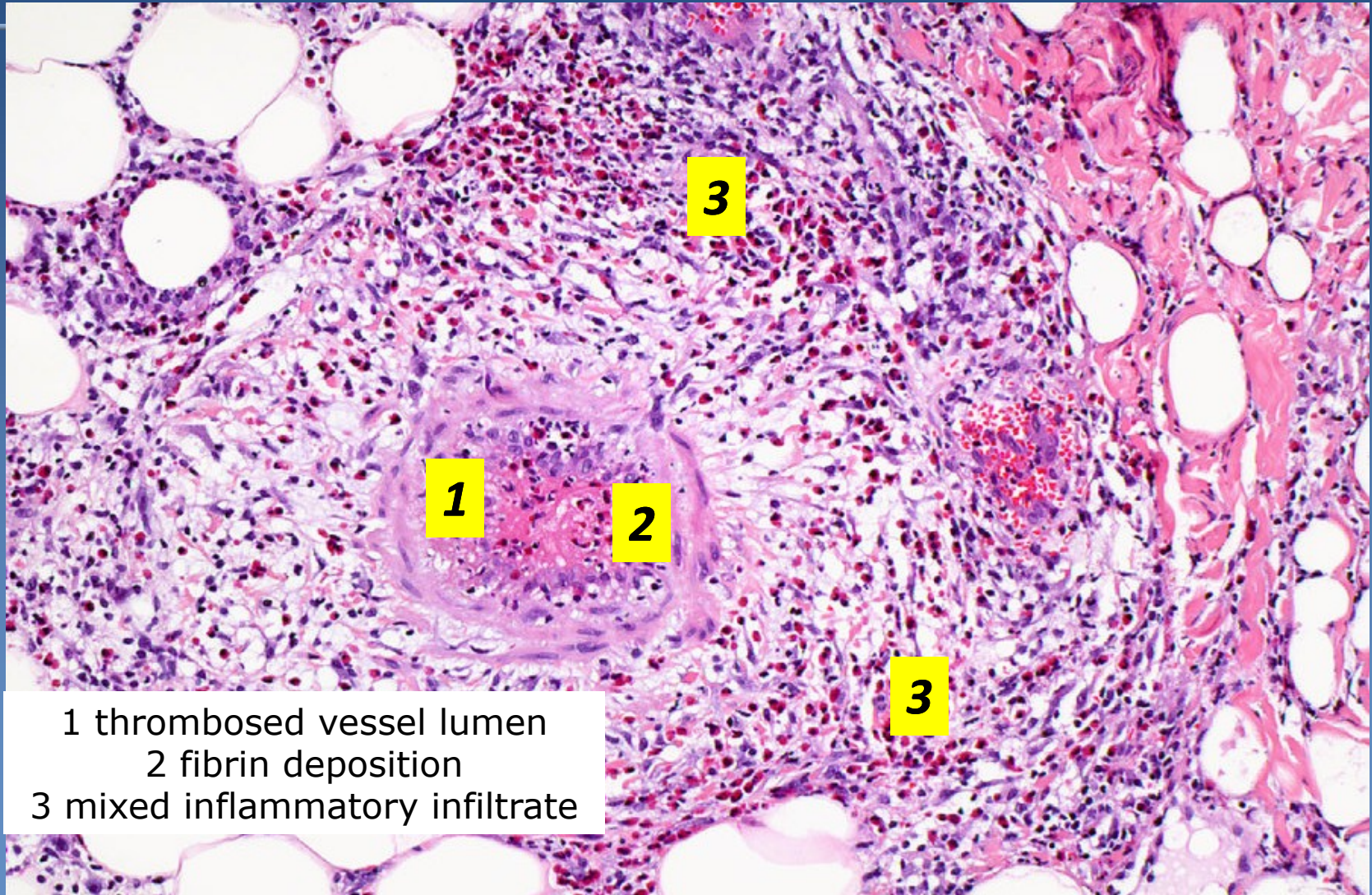
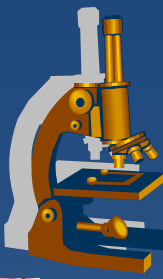
nuclear fragments from neutrophils in a small vessel wall

# *polyarteritis nodosa*



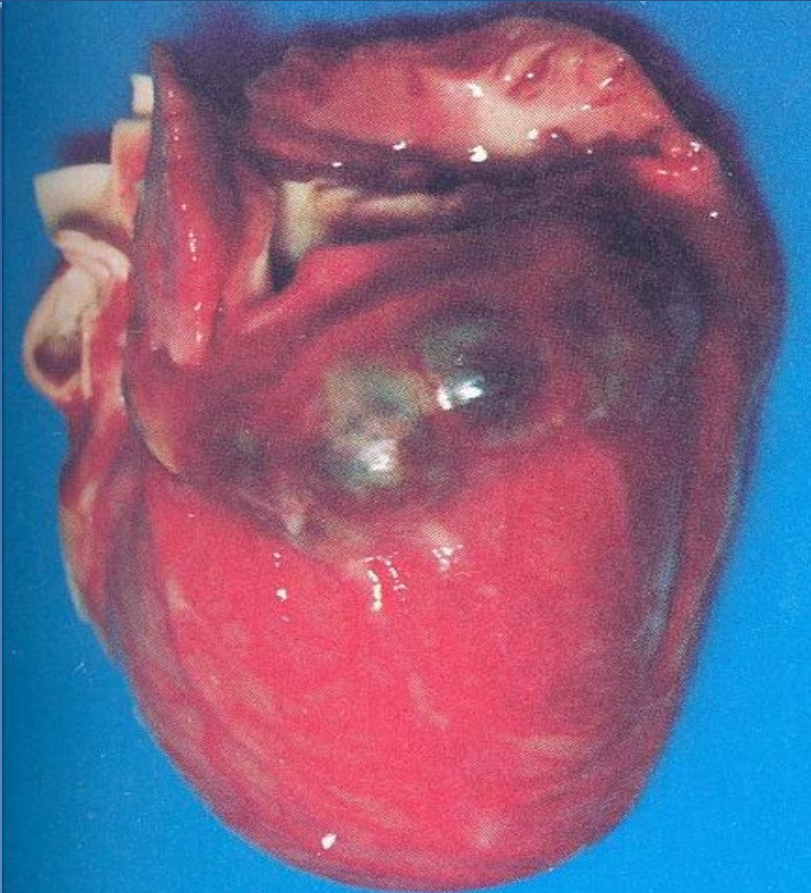
- 1 thrombosed vessel lumen
- 2 fibrin deposition
- 3 mixed inflammatory infiltrate

# *polyarteritis nodosa*

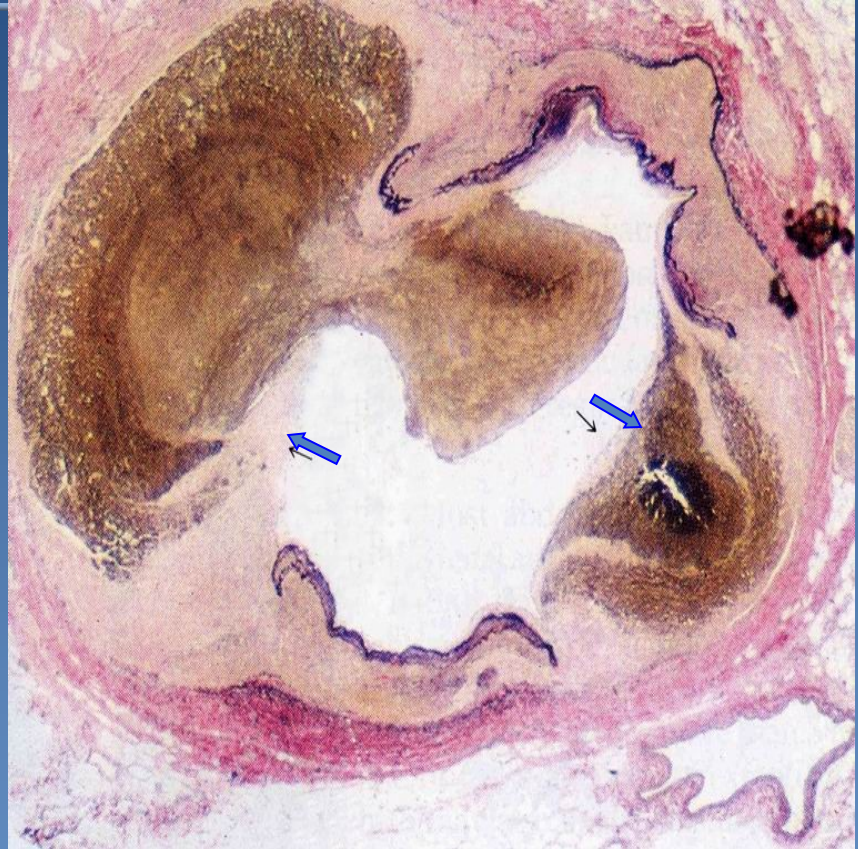


1 thrombosed vessel lumen  
2 fibrin deposition  
3 mixed inflammatory infiltrate

# *Kawasaki disease*

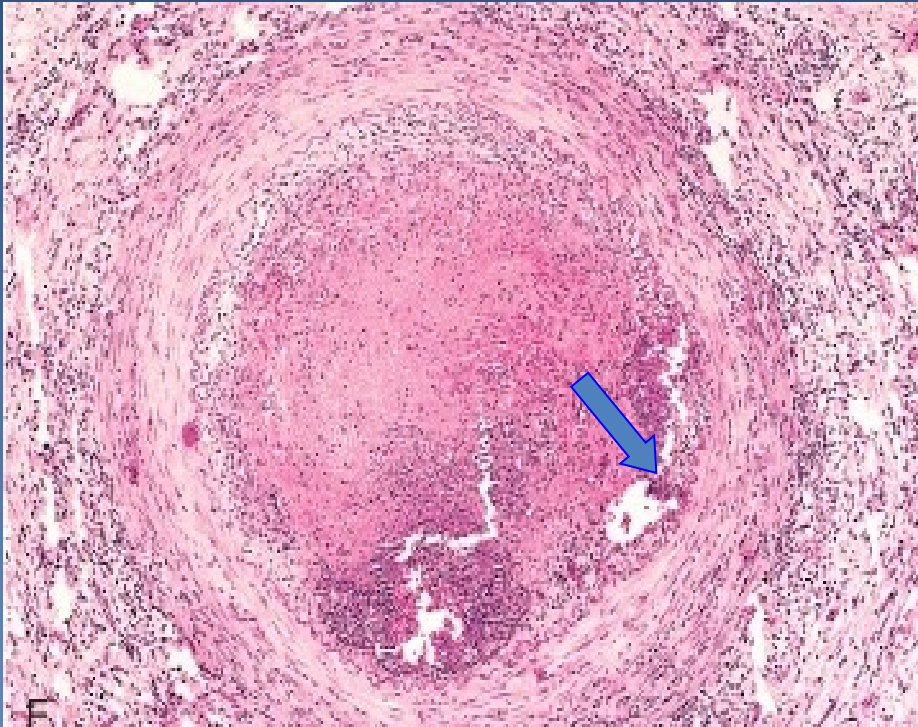


Coronary aneurysms in a child's heart



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

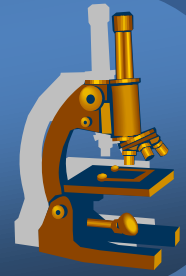


- × direct invasion of vascular wall by inf. pathogen
  
- × primary angioinvasive microorganism  
Fungi: *Aspergillus*, *Mucor* - thrombosis → ischemic necrosis
  
- × secondary vasculitis - **localized vasculitis** in focal infection
  - ⇒ purulent – meningitis
  - ⇒ pneumonia
  - ⇒ abscess, fasciitis – pyogenic bacteria
  - ⇒ granulomatous
    - obliterative endarteritis – TB tertiary syphilis, I
    - Lepra
  - ⇒ lymphocytic vasculitis – rickettsia (spotted fever, Q fever etc.)
  - ⇒ recurrent herpes, CMV
  - ⇒ necrotizing vasculitis – anthrax



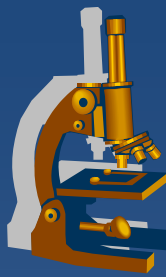
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# *Cardiac pathology*



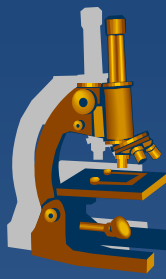


# Morphology

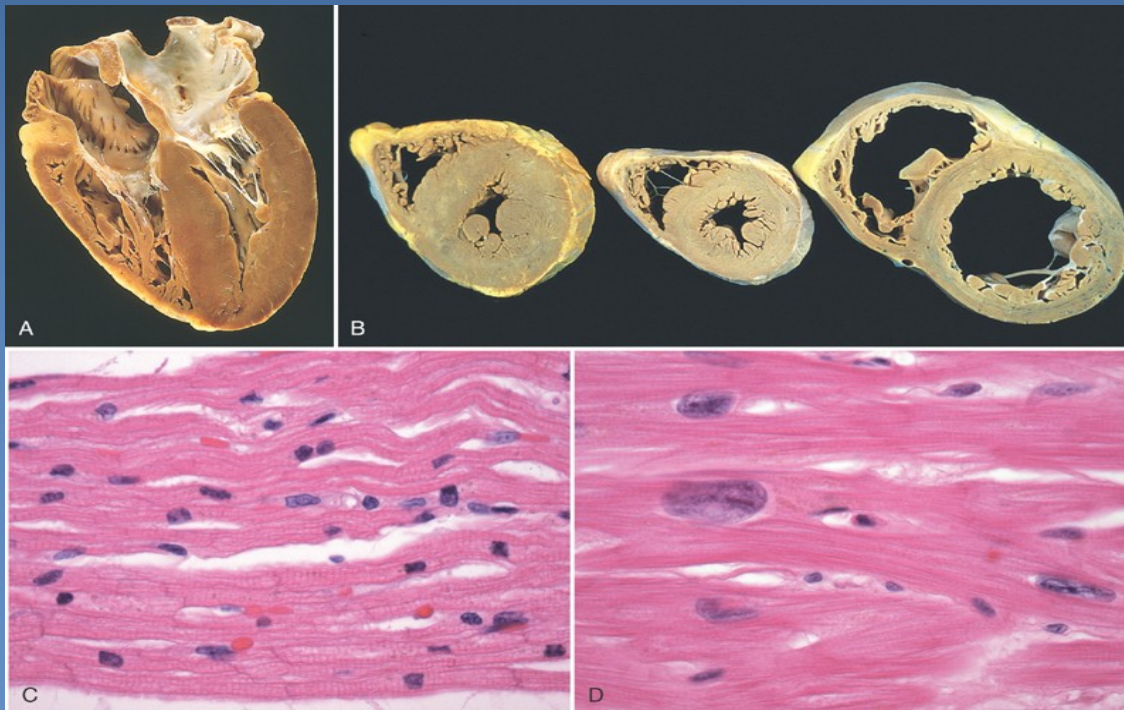


- ✗ pericardial sac – cca 30ml clear yellowish fluid
- ✗ **male = 300 – 350 g**,
  - *hypertrophy > 400g*
- ✗ **myocardium:**
  - RV 3 – 4 mm
  - LV 12 – 15 mm
- ✗ **foramen ovale**
  - *closed x opened → paradoxical embolia*

# 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



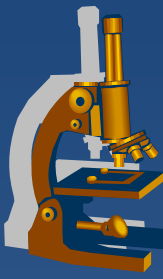
# Heart failure



- ✘ 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)
- ✘ cardiac – 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

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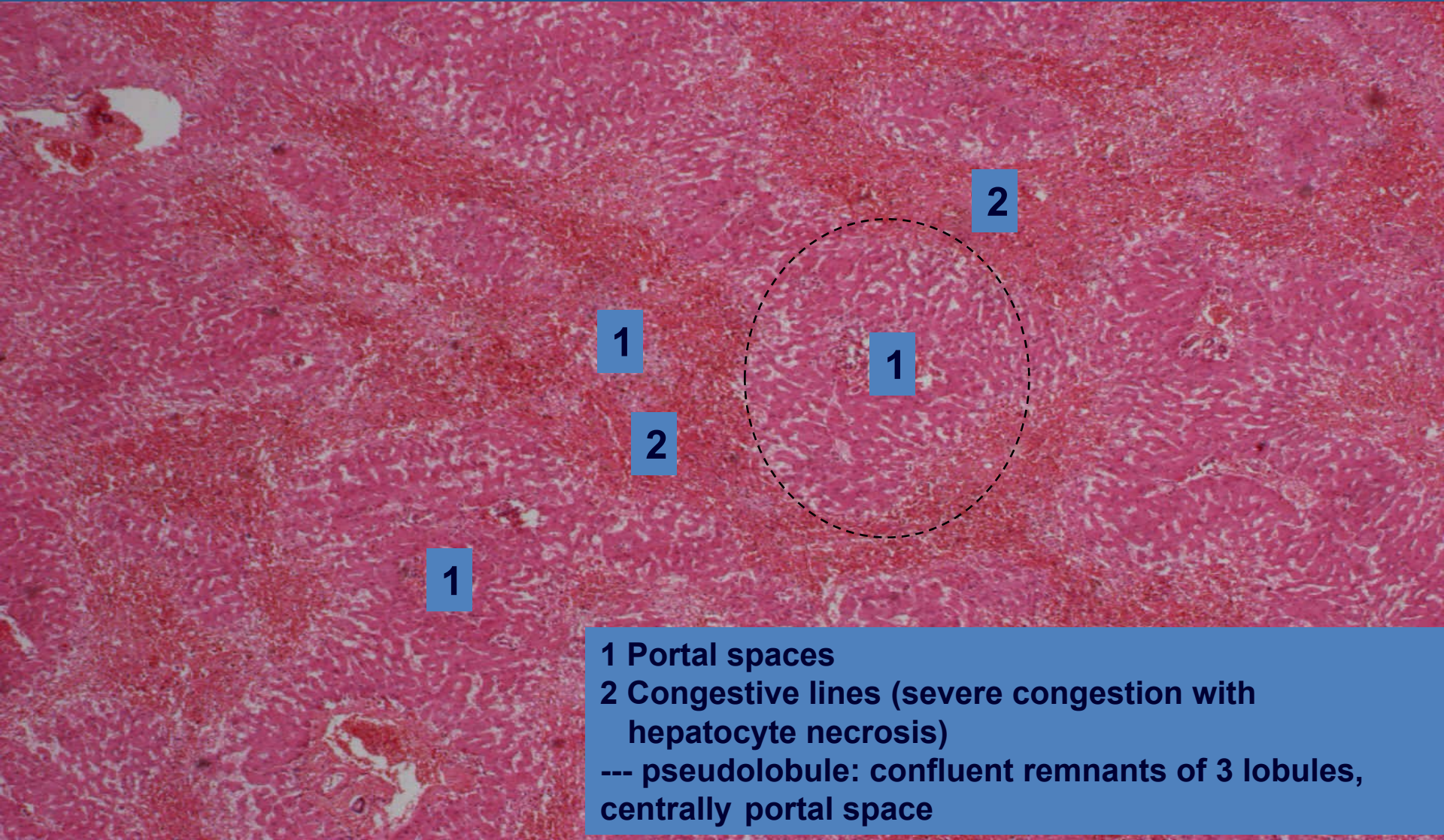


- × **venous congestion** – e.g. liver (-> *hepar moschatum*)
- × **induration** – fibroproduction (liver, spleen, kidney)
- × **oedema** –
- × **cyanosis** – visible on acral parts

***Chronic venous congestion  
(nutmeg liver - hepar moschatum)***



# Hepatic venous congestion



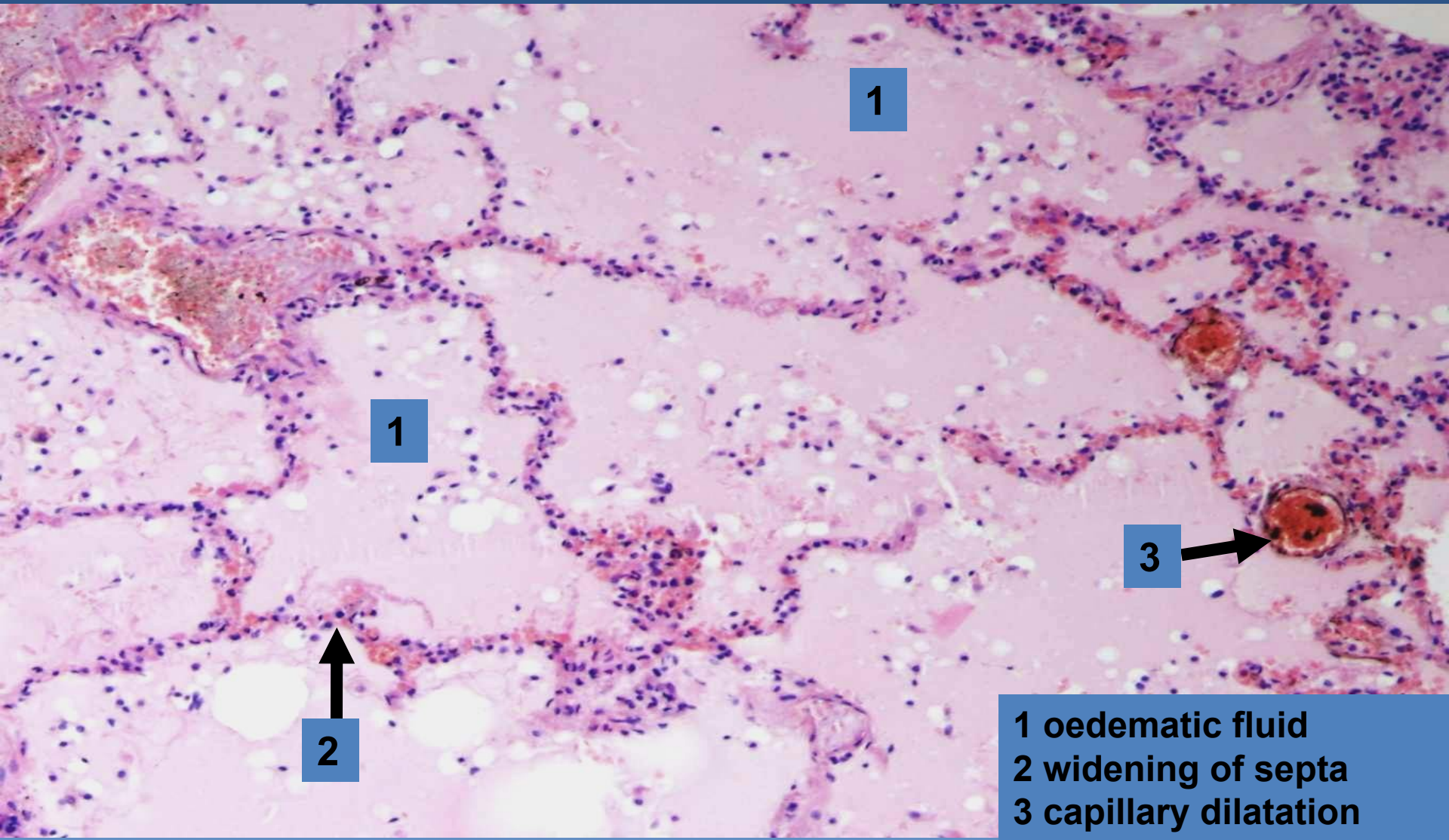
1 Portal spaces

2 Congestive lines (severe congestion with hepatocyte necrosis)

--- pseudolobule: confluent remnants of 3 lobules, centrally portal space



# *Pulmonary oedema*



1

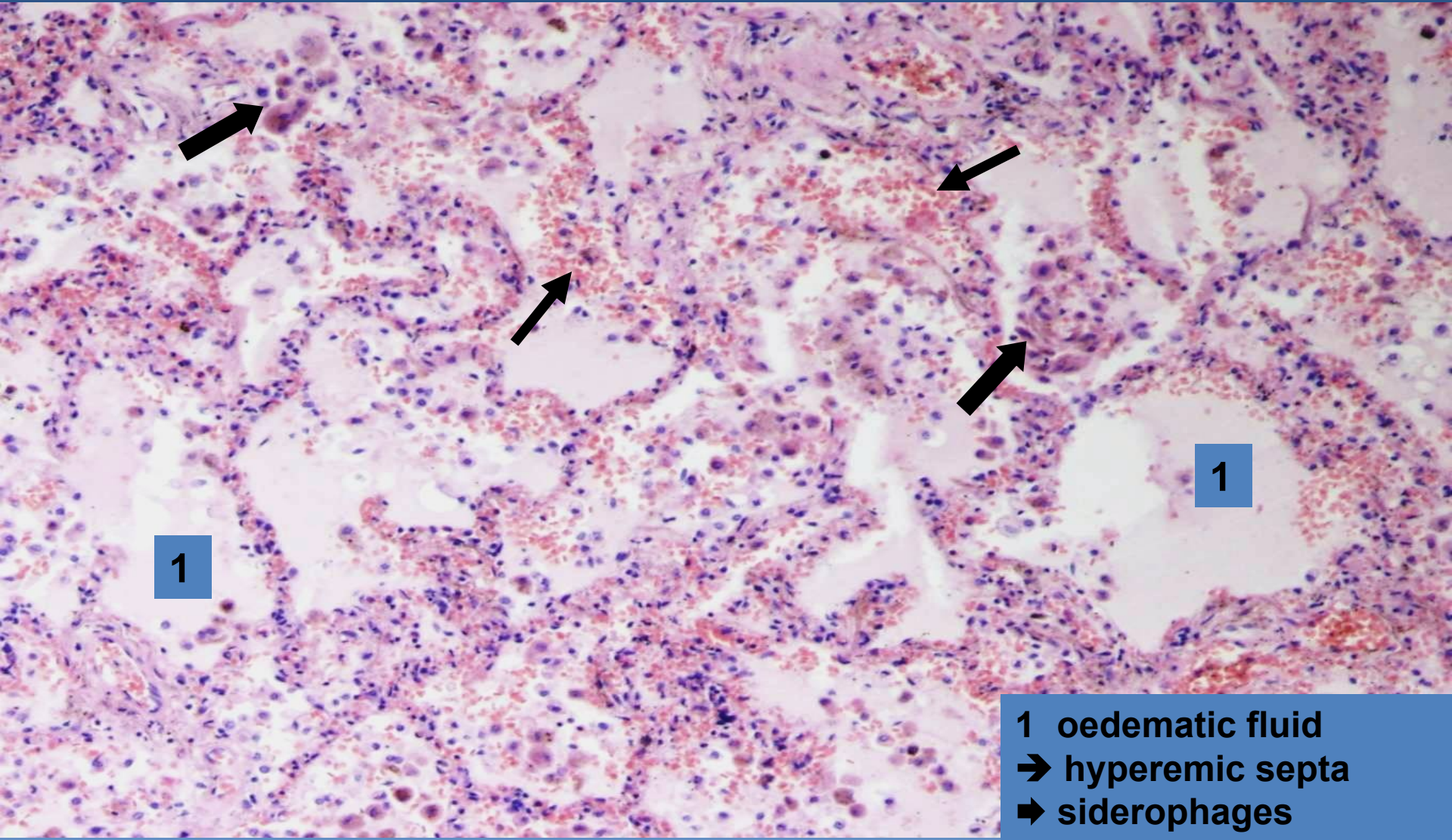
1

3

2

1 oedematous fluid  
2 widening of septa  
3 capillary dilatation

# *Chronic pulmonary venous congestion*



- 1 oedemetic 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



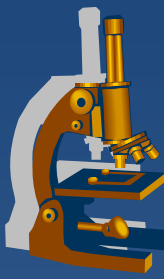
## 1) AS of coronary aa.

- commonly at a. branching
- fixed obstruction by plaque (fibrous, atheromatic)
- acute plaque change (rupture, erosion, haemorrhage, thrombosis)
- 75% stenosis – ischemia during  $\uparrow$  workload – stable angina pectoris
- 90% stenosis – ischemia even at rest – unstable angina - preinfarction

## 2) non-atherosclerotic

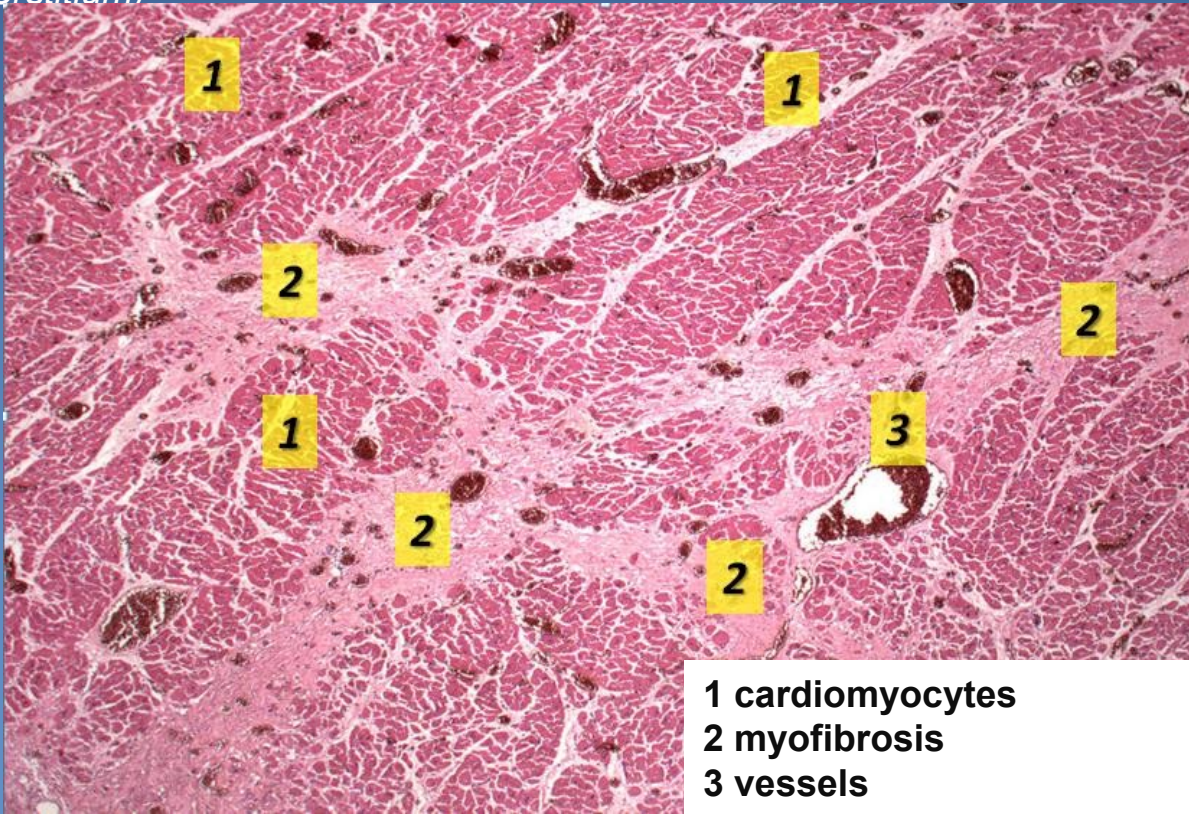
- coronary emboli – endocarditis, atrial fibrillation, mural thr., paradoxical e.
- coronary vasospasm
- aortic dissection
- coronary vasculitis
- congenital coronary aa. defects
  - hematologic disorders, amyloidosis, shock, etc.

# ischemic heart disease (IHS)



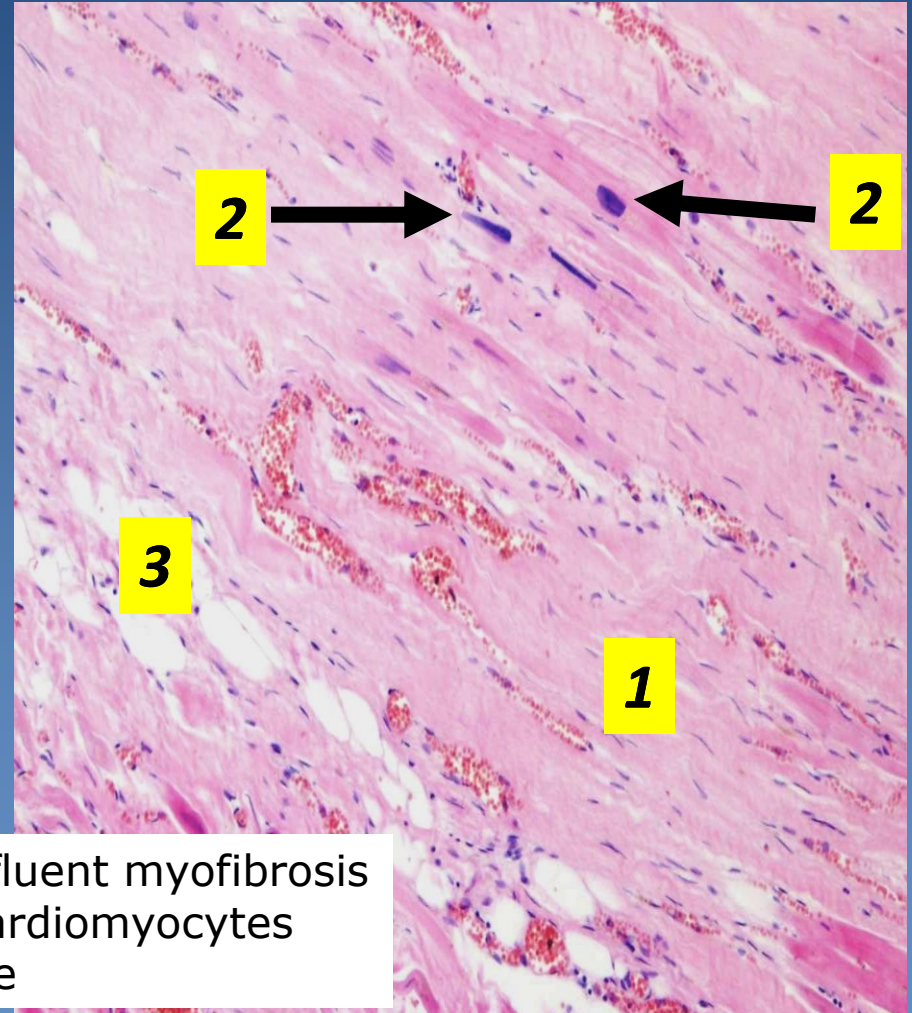
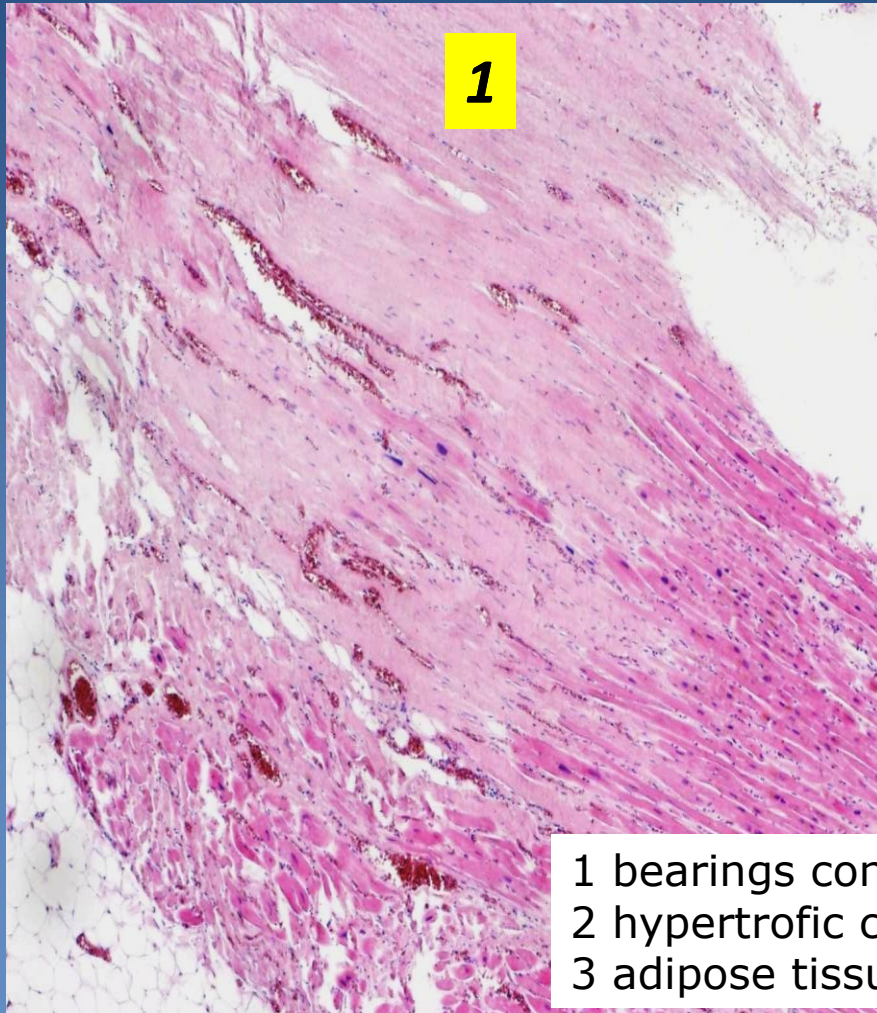
## ✘ Morphology of myocardial ischemia:

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

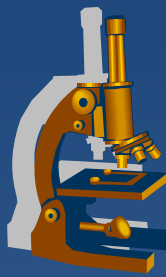


1 cardiomyocytes  
2 myofibrosis  
3 vessels

# Confluent myofibrosis and myocard lipomatosis



1 bearings confluent myofibrosis  
2 hypertrophic cardiomyocytes  
3 adipose tissue



# Angina pectoris (AP)

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

## 1. stable (typical)

- due to increased workload, duration  $\leq 15$  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*

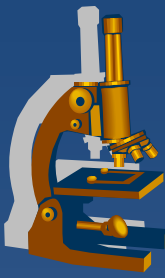


## *× micro:*

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

# *Myocardial infarction*

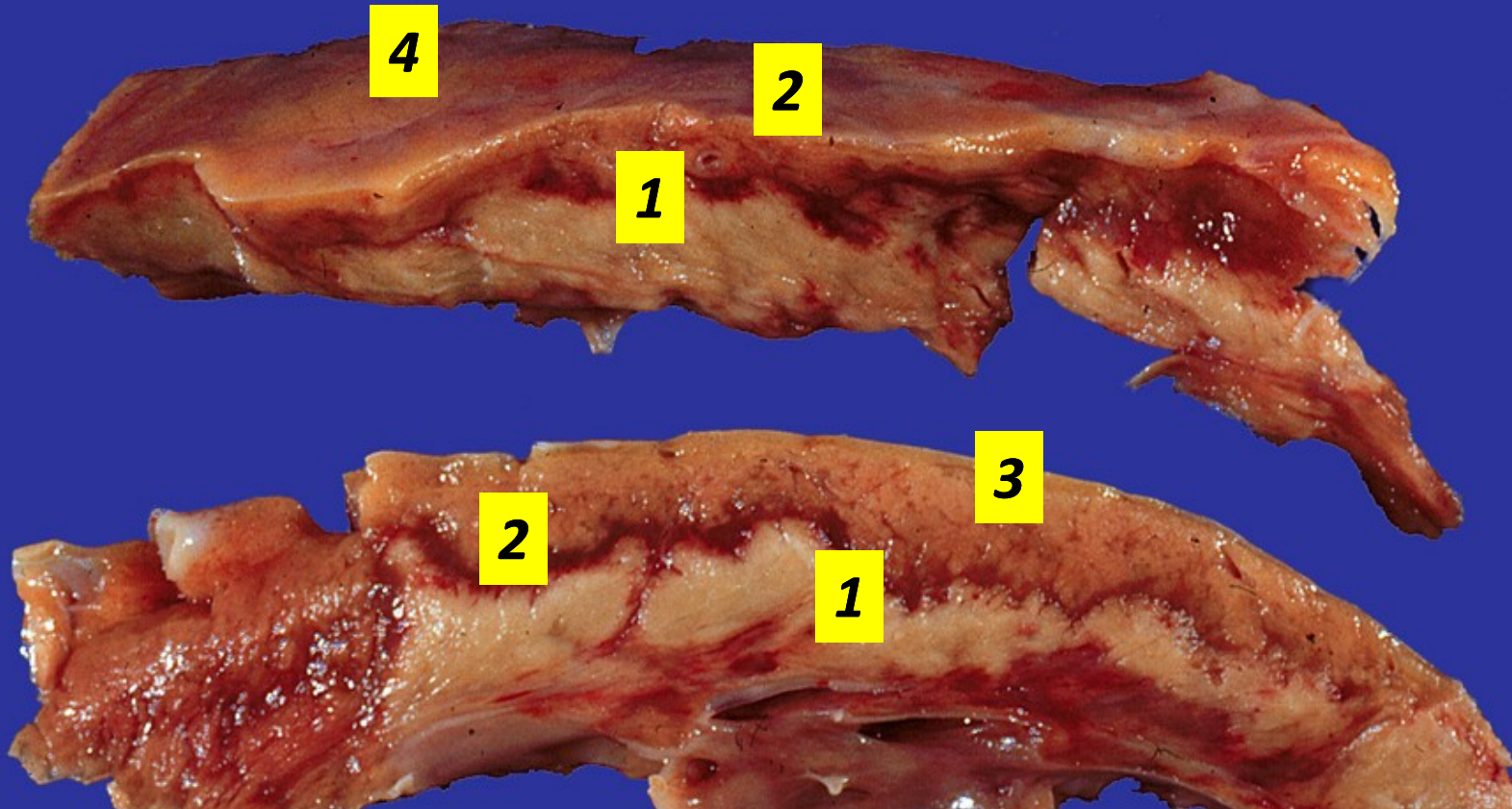
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**micro:**

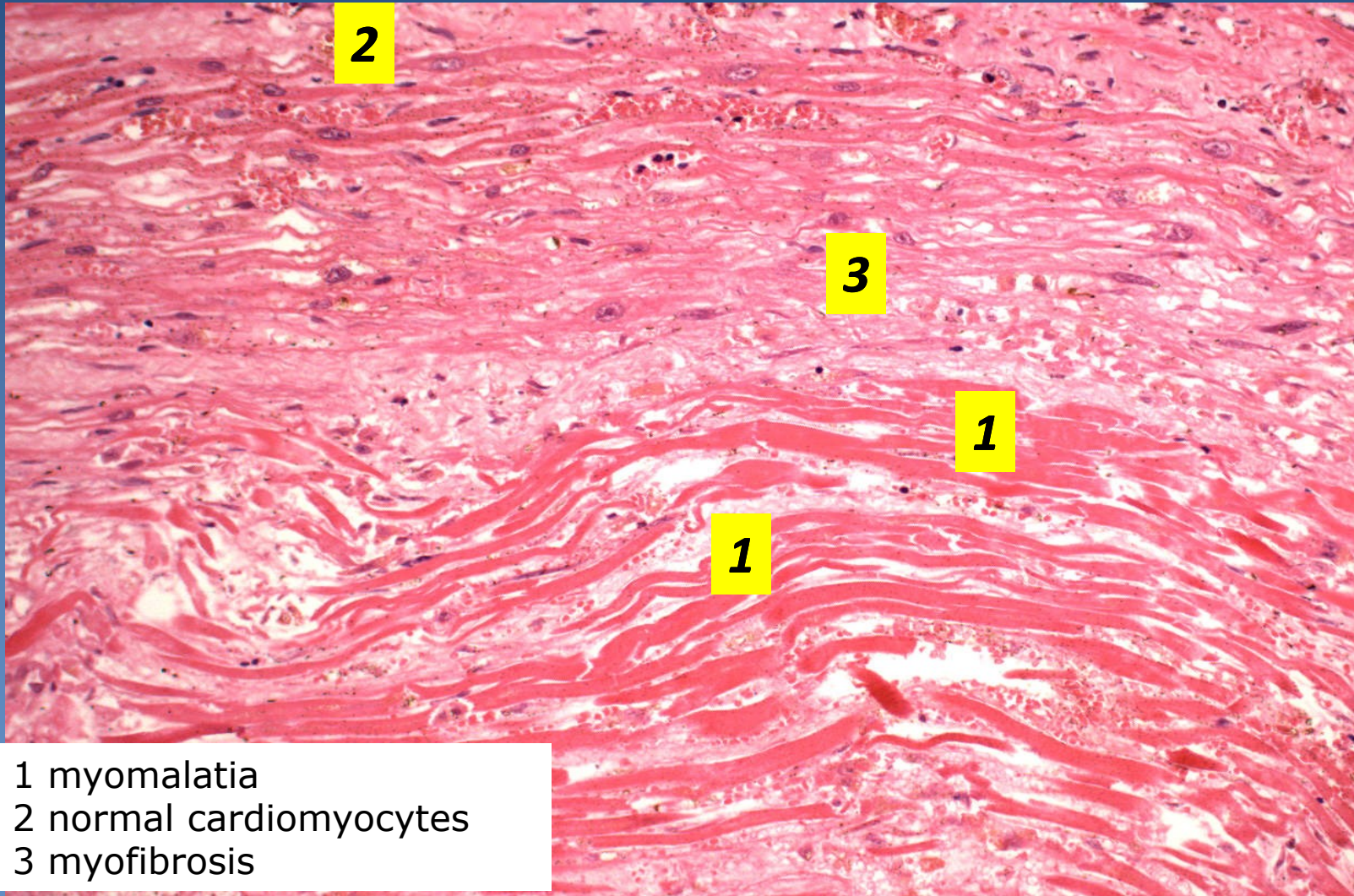
- × **12-24 hr:** edema, hypereosinophilia of necrotic cells, pyknosis
- × **1-3 days:** neutrophils, loss of nuclei
- × **3-7 days:** macrophages at the border, desintegration of myofibers
- × **1-2 weeks:** repair by granulation tissue
- × **cca 2 months:** scar

# Myocardial infarction



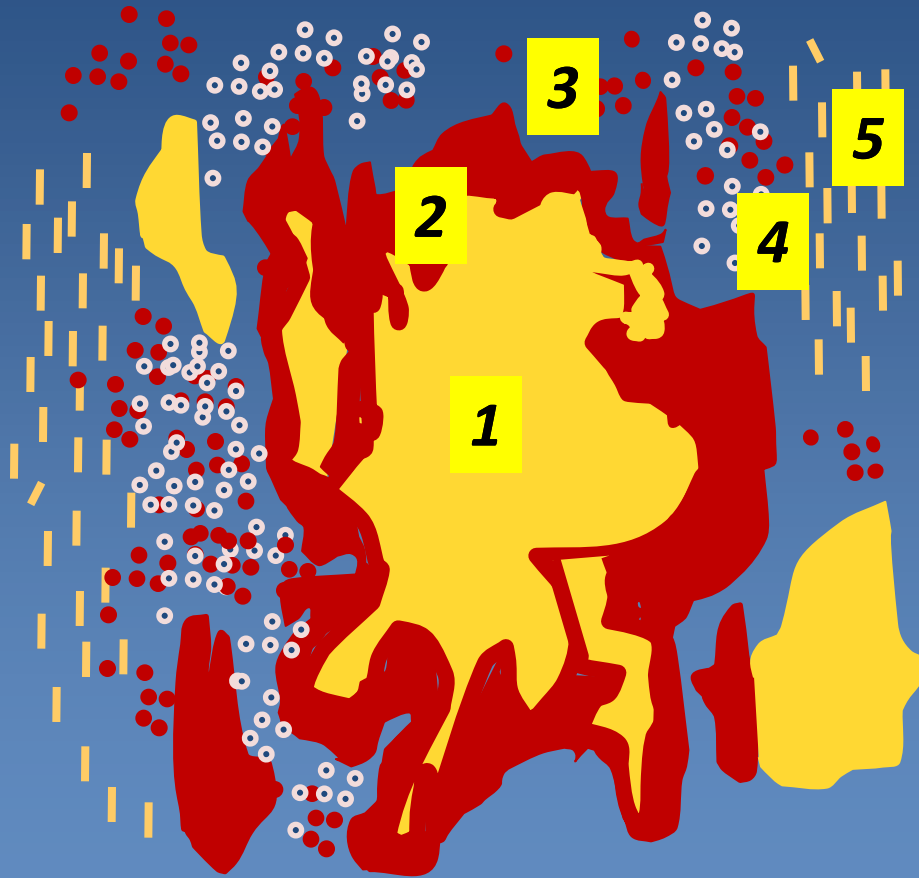
1 subendocardial coagulative necrosis 2 hyperemic rim 3 normal myocardium 4 epicardium






# Myomalatia



- 1 myomalatia
- 2 normal cardiomyocytes
- 3 myofibrosis

# Microscopic changes in developed MI



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

# Myocardial infarction

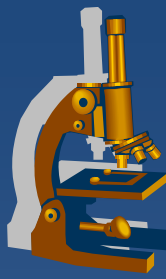


- × **transmural (QIM, STEMI) - + ST elevation on ECG**

- $\geq \frac{3}{4}$  of wall thickness, breadth  $>25$  mm
- complete coronary artery obstruction  
emergency angioplasty/stenting

- × **non-transmural (subendocardial, Non-STEMI)**

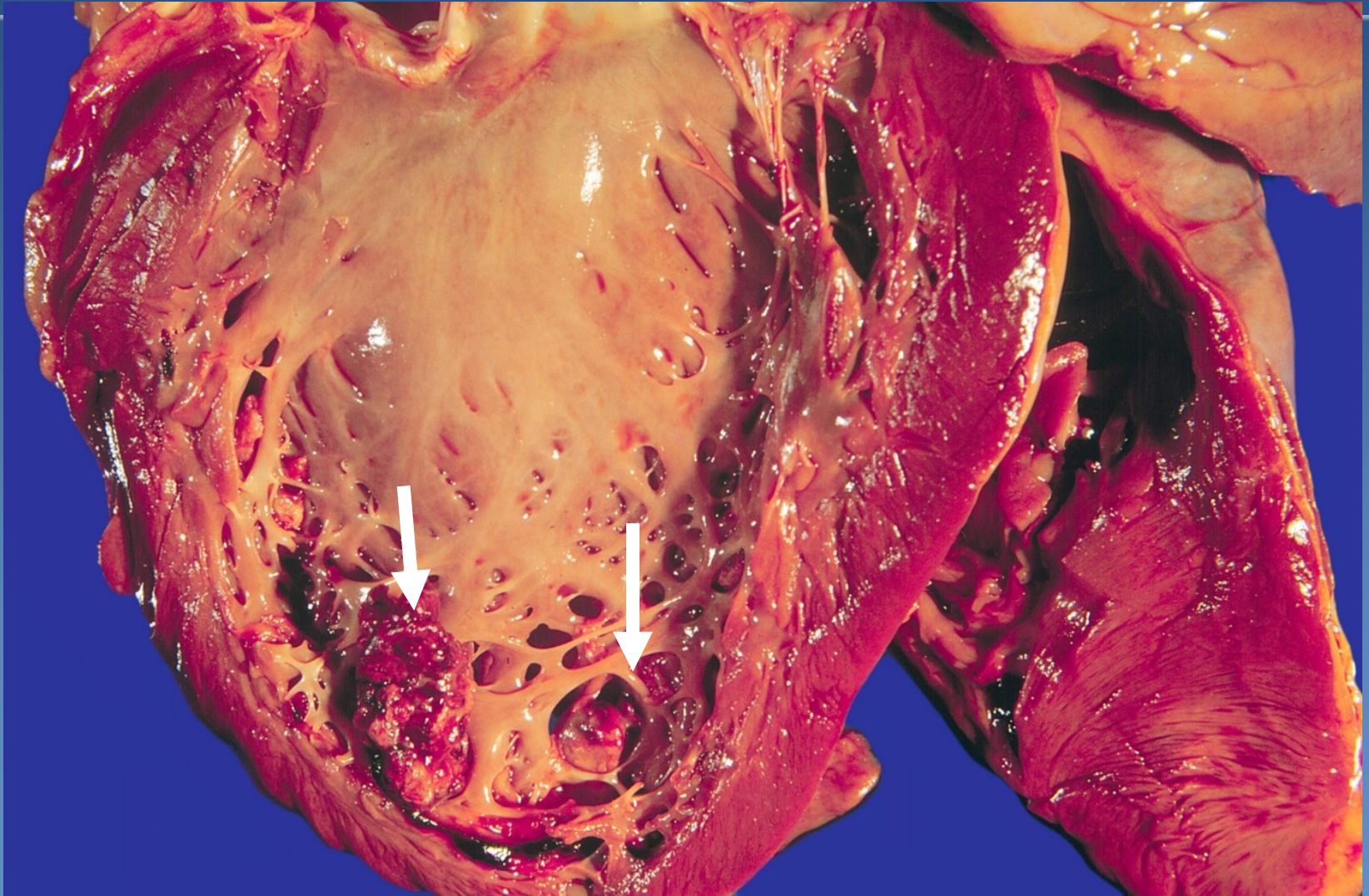
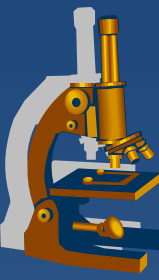
- internal  $\frac{1}{4}$  až  $\frac{1}{2}$  of LV wall
- collateral blood flow, incomplete obstruction, shorter ischemia



# **MI complications**

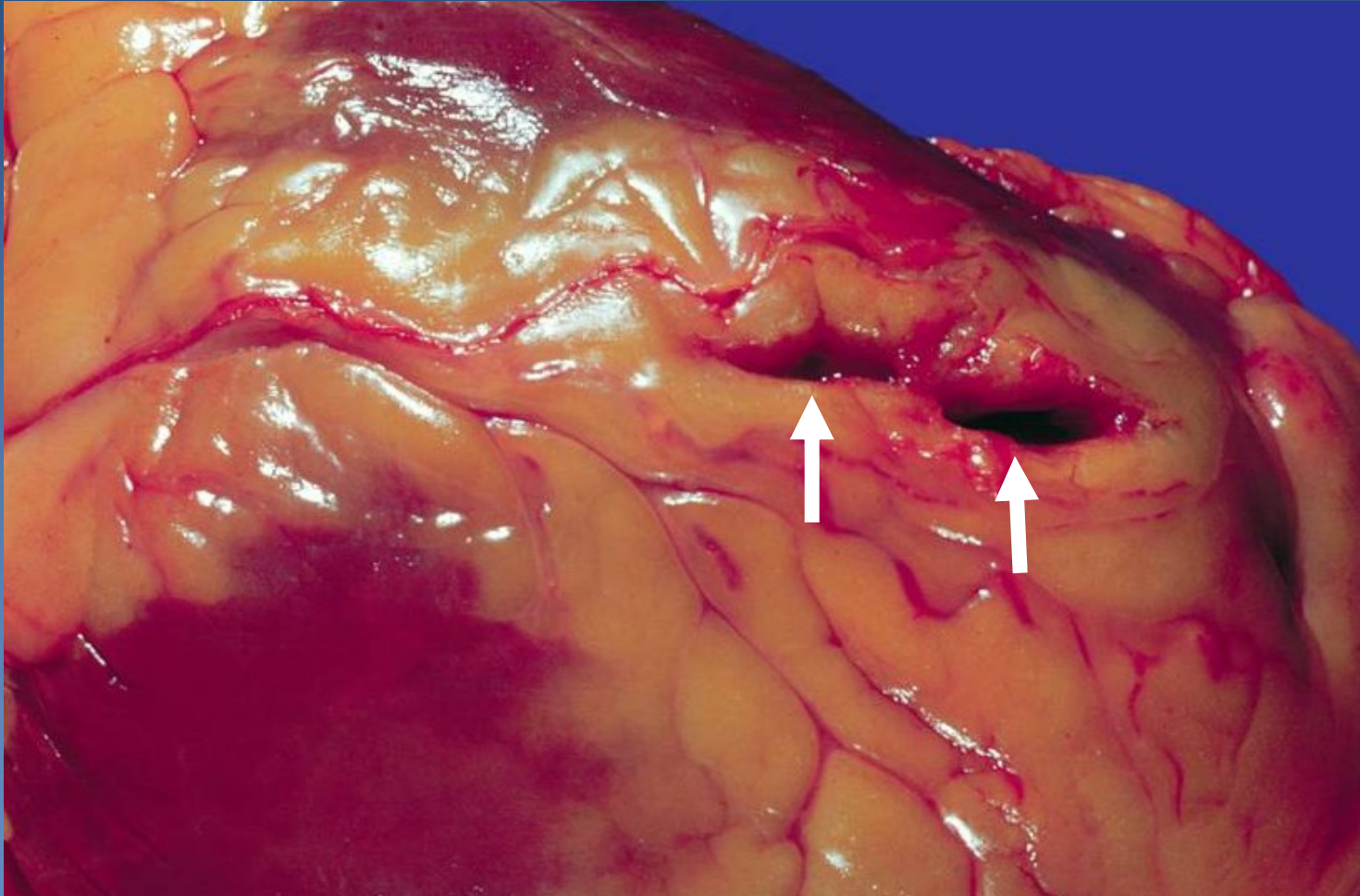
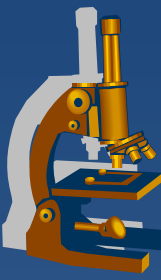
- 1. sudden death (arrhythmia)**
- 2. cardiogenic shock (contractile dysfunction)**
- 3. pericarditis epistenocardiaca**  
*-> sero-fibrinous inflammation*
- 4. mural thrombosis**  
*-> embolism into systemic circulation (-> brain, kidney, intestine, spleen infarction)*
- 5. ventricular aneurysm**  
*-> acute – risk of rupture, thrombosis; 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***

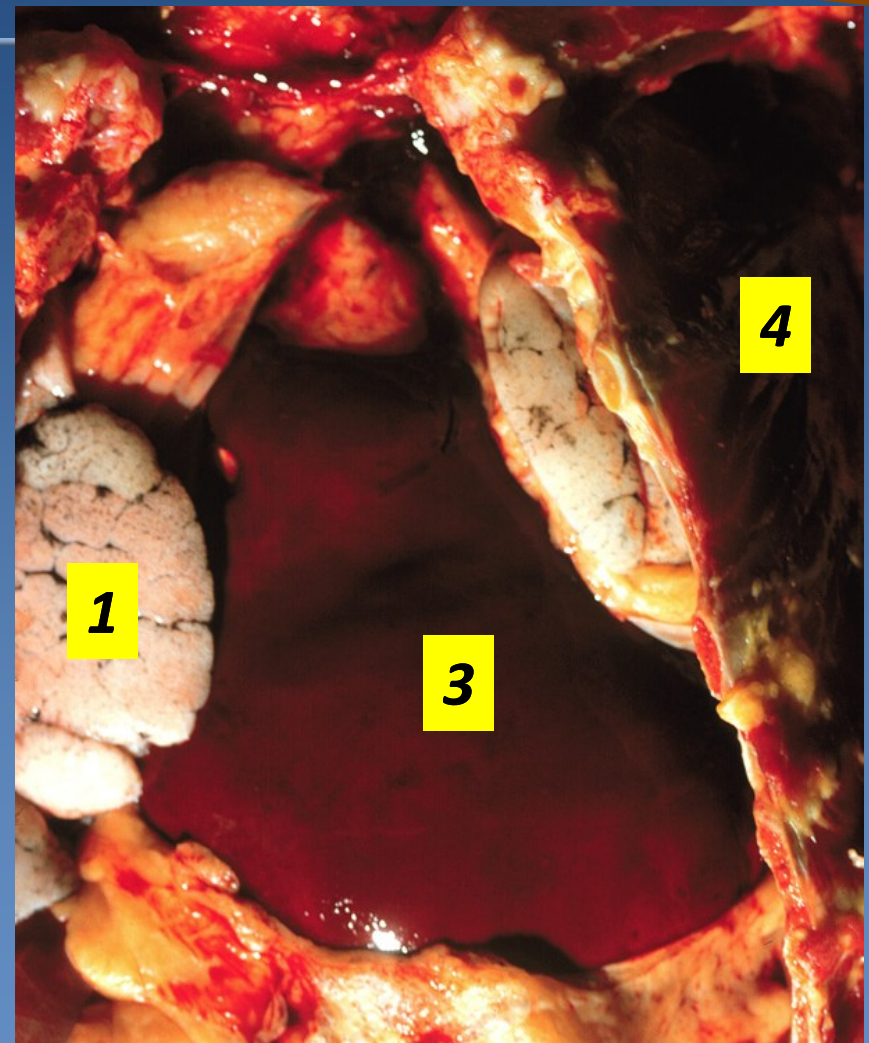
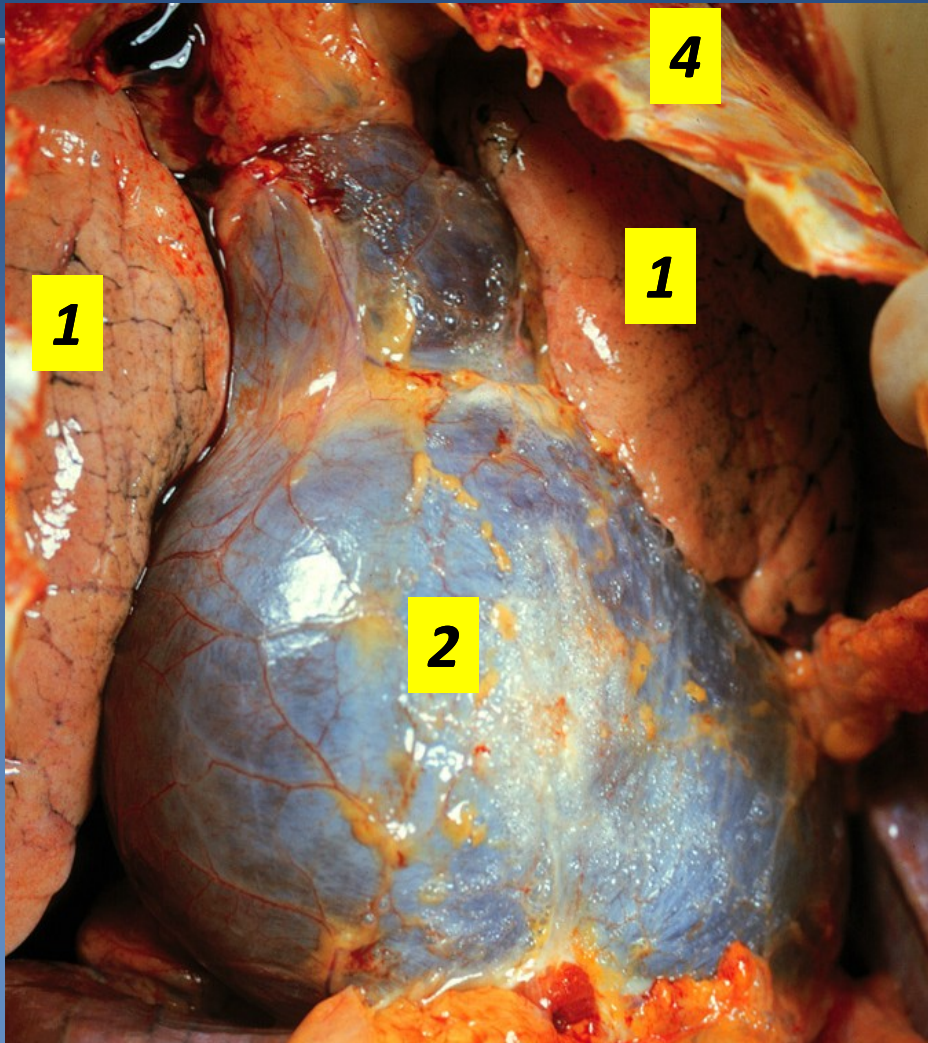
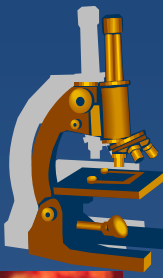




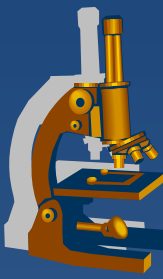
# *Mi – rupture*



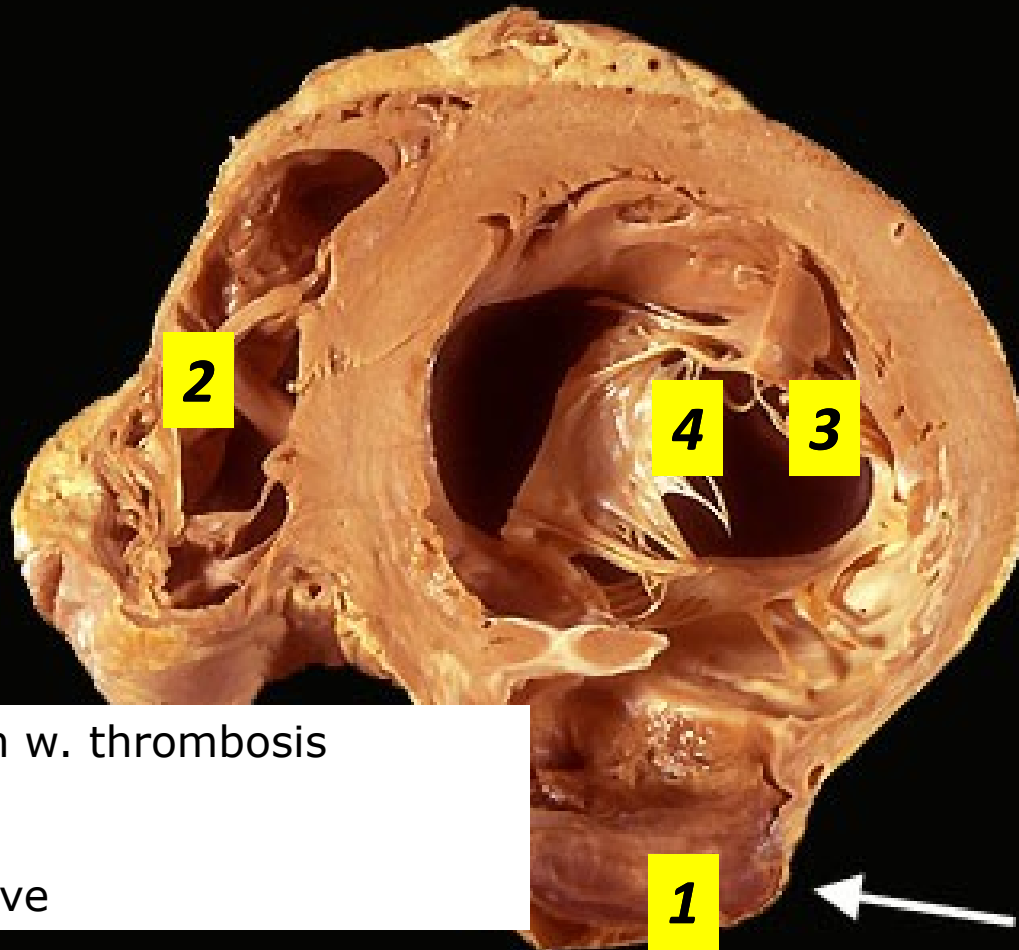
# *MI – rupture, tamponade*



**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 + dilatation, myofibrosis and/or post-MI scars
- ✘ multiple coronary arteries with significant AS stenosis
- ✘ imminent risk of MI, sudden cardiac death due to arrhythmia, 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 arrhythmia (ventricular fibrillation, asystole)

✗ sudden collapse without signs of acute MI

✗ other causes:

⇒ *dissecting/ruptured aortic aneurysm*

⇒ *pulmonary thromboembolism*

⇒ *massive intracerebral haemorrhage*

⇒ *heritable conditions incl. anatomic, electrical – 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. pyogenes, 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*

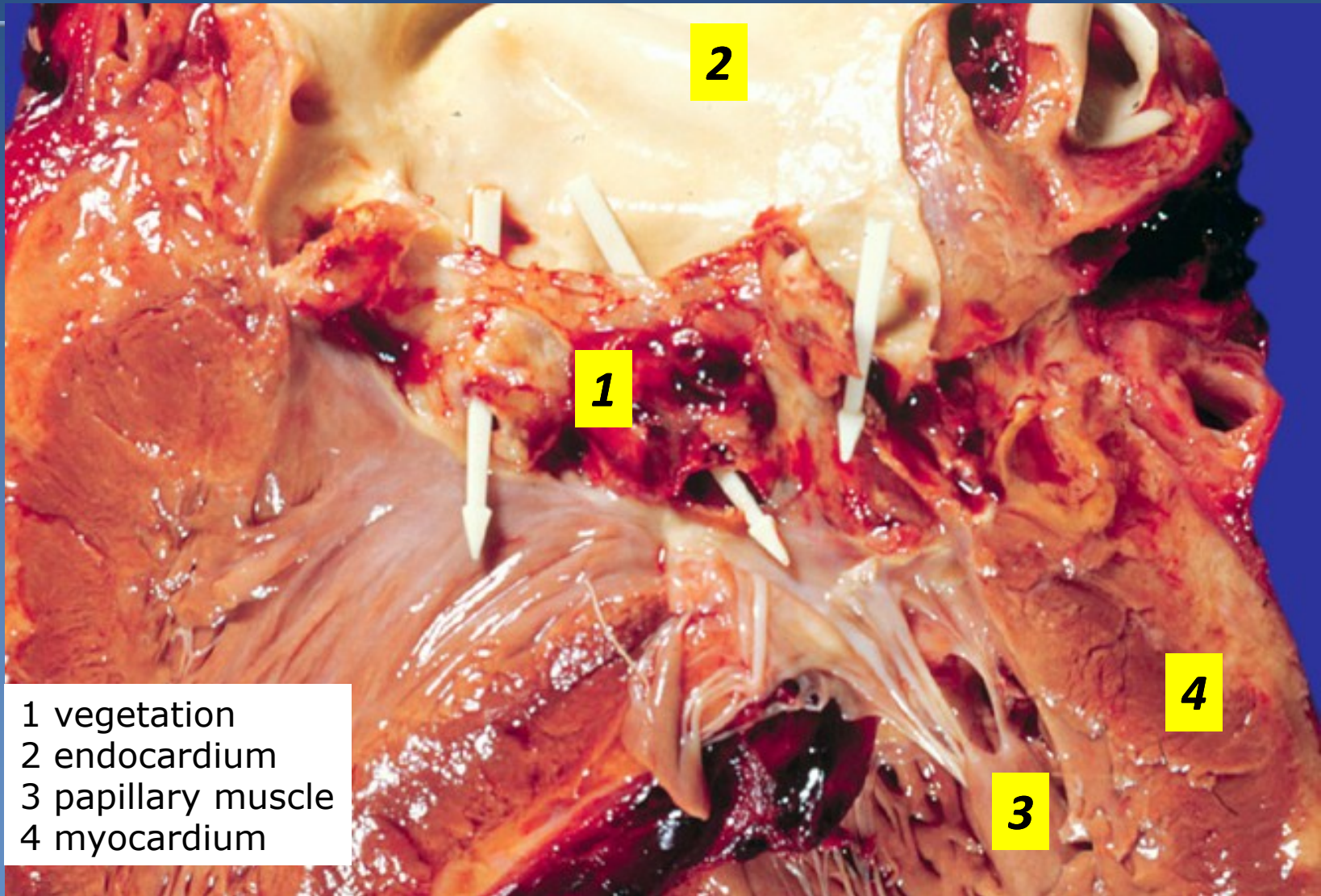
✗ **complications:**

⇒ *acute: valvular damage, myocarditis + abscess, pyemia, thrombembolism*

⇒ *chronic valvular disease*

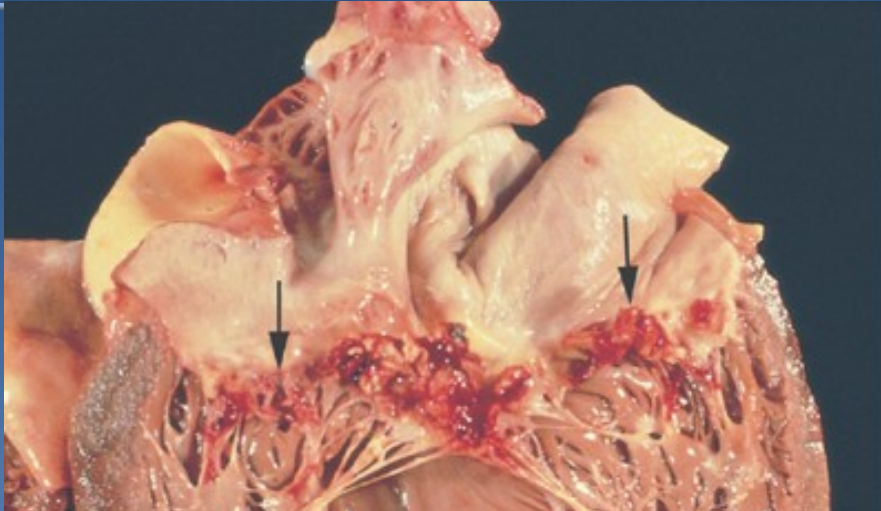


# ***Infective endocarditis- valve destruction***

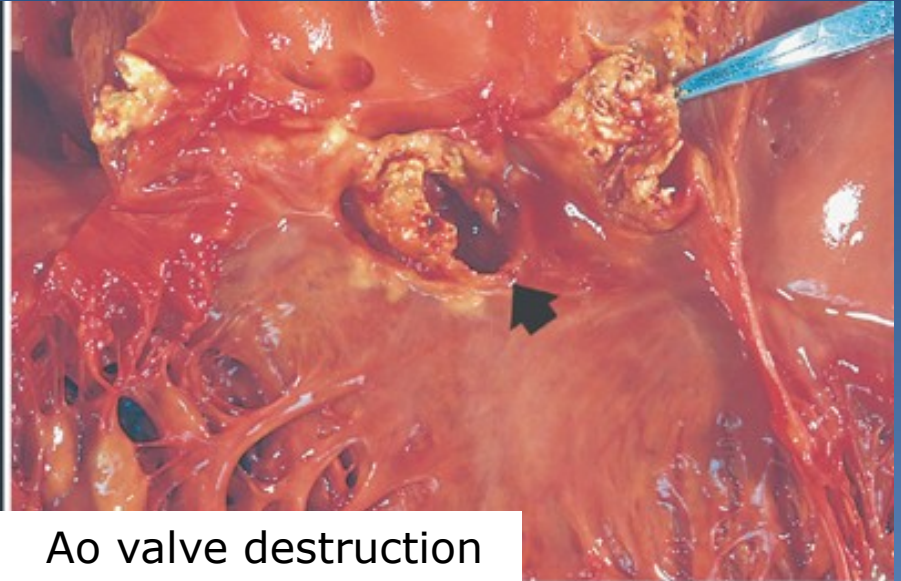


- 1 vegetation
- 2 endocardium
- 3 papillary muscle
- 4 myocardium

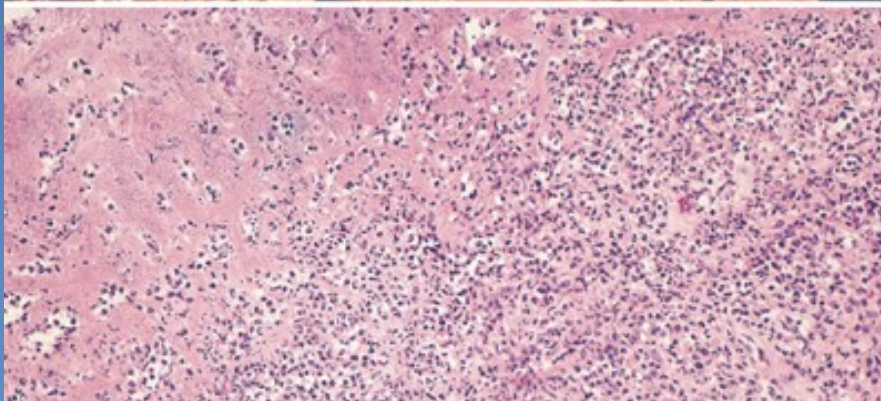
# *Infective endocarditis*



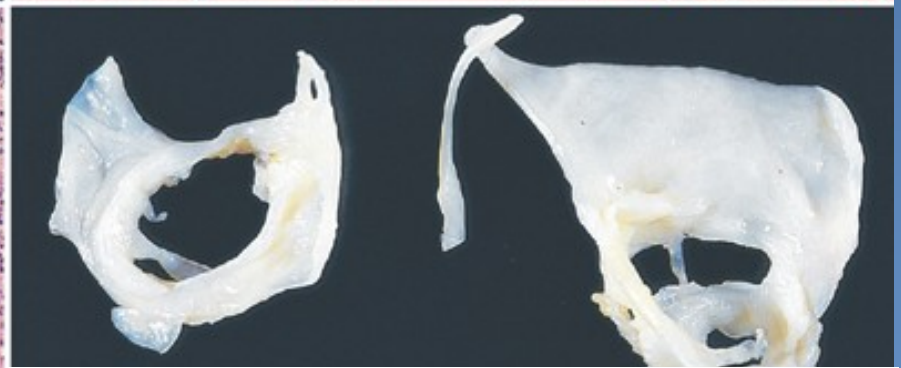
Mi vegetations



Ao valve destruction

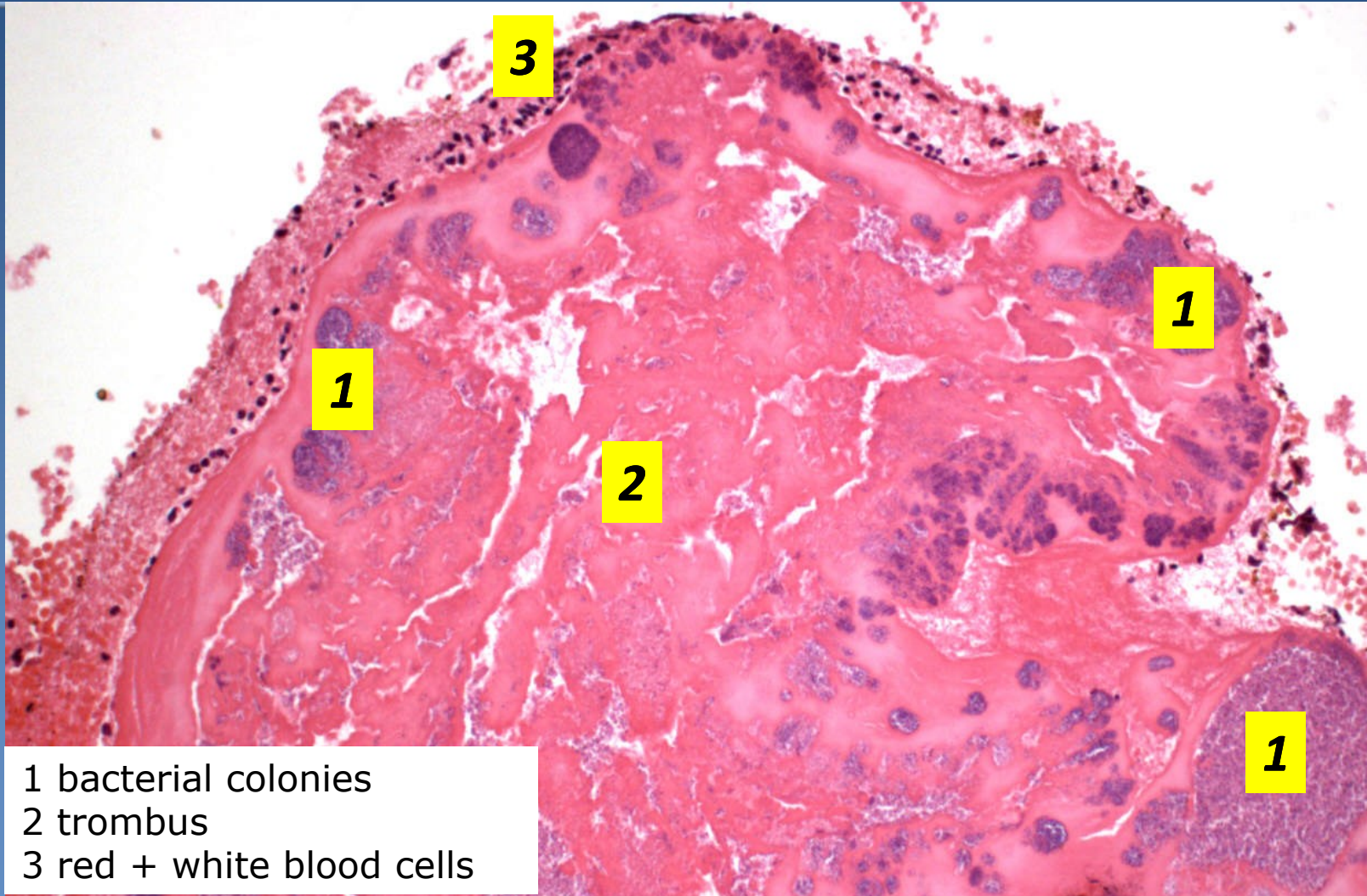


purulent inflammation



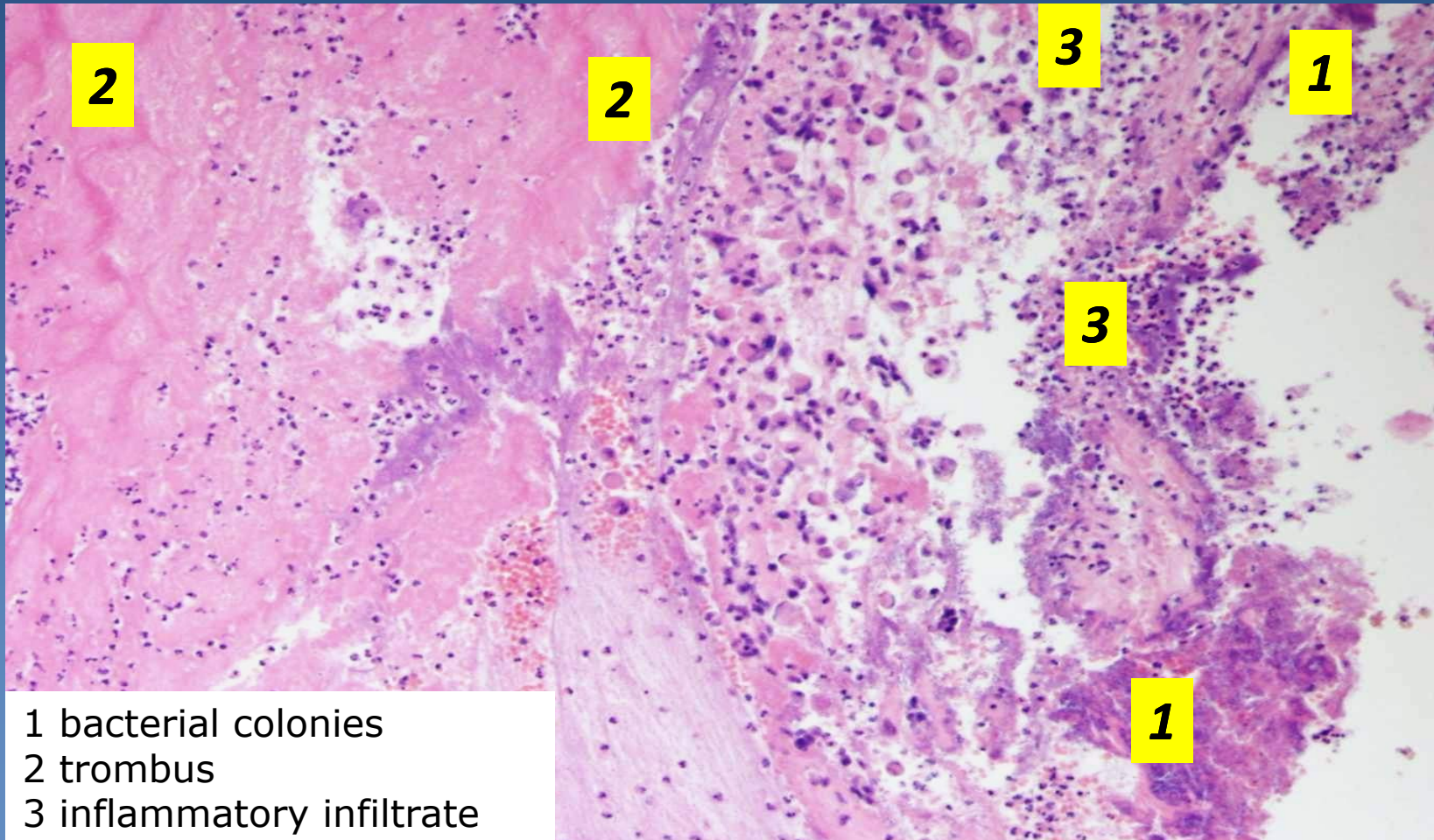
IE repair (Mi fenestration without vegetations)

# *Infective endocarditis - vegetations*



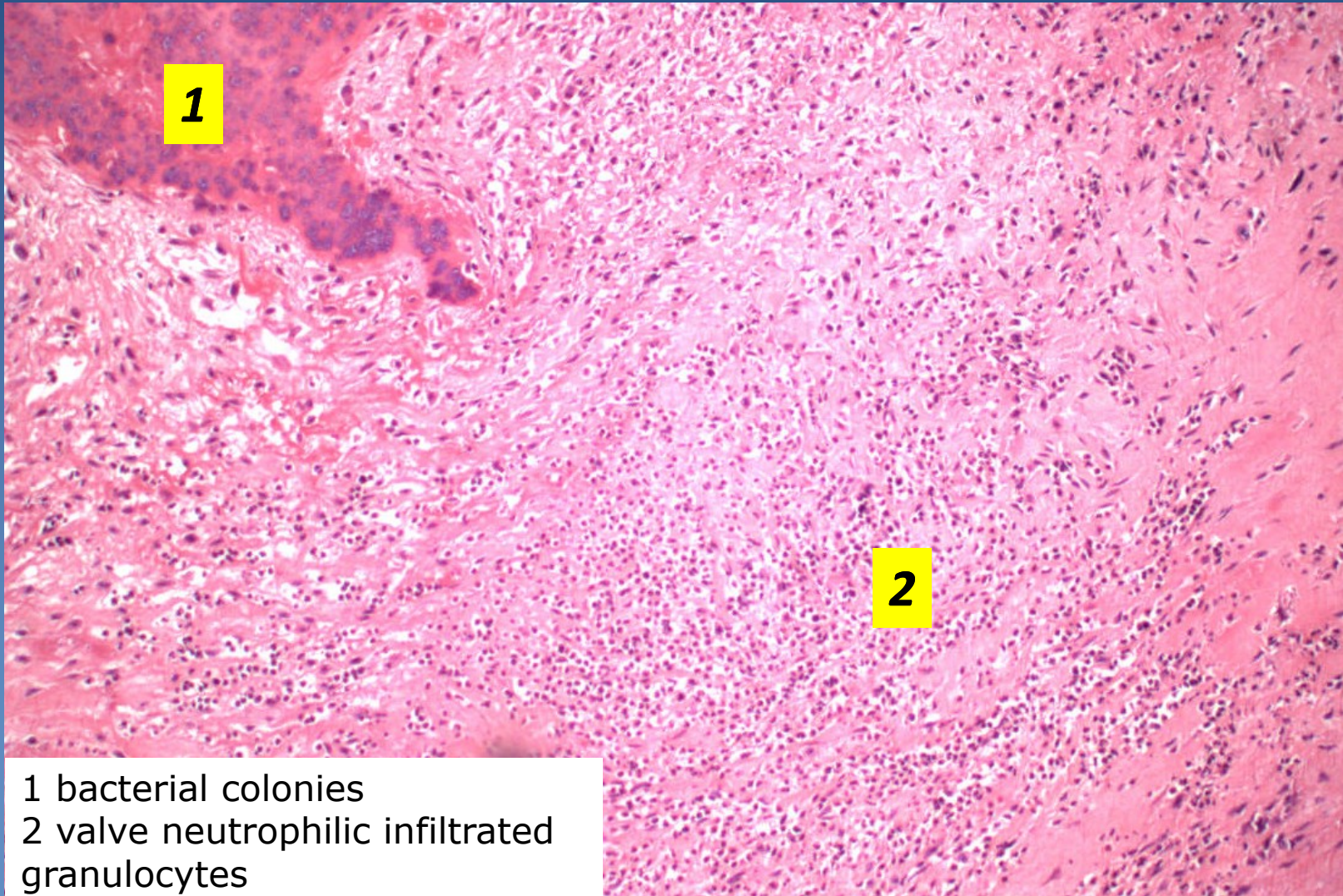
- 1 bacterial colonies
- 2 trombus
- 3 red + white blood cells

# *Infective endocarditis - vegetations*



- 1 bacterial colonies
- 2 trombus
- 3 inflammatory infiltrate

# *Infective endocarditis - vegetations*



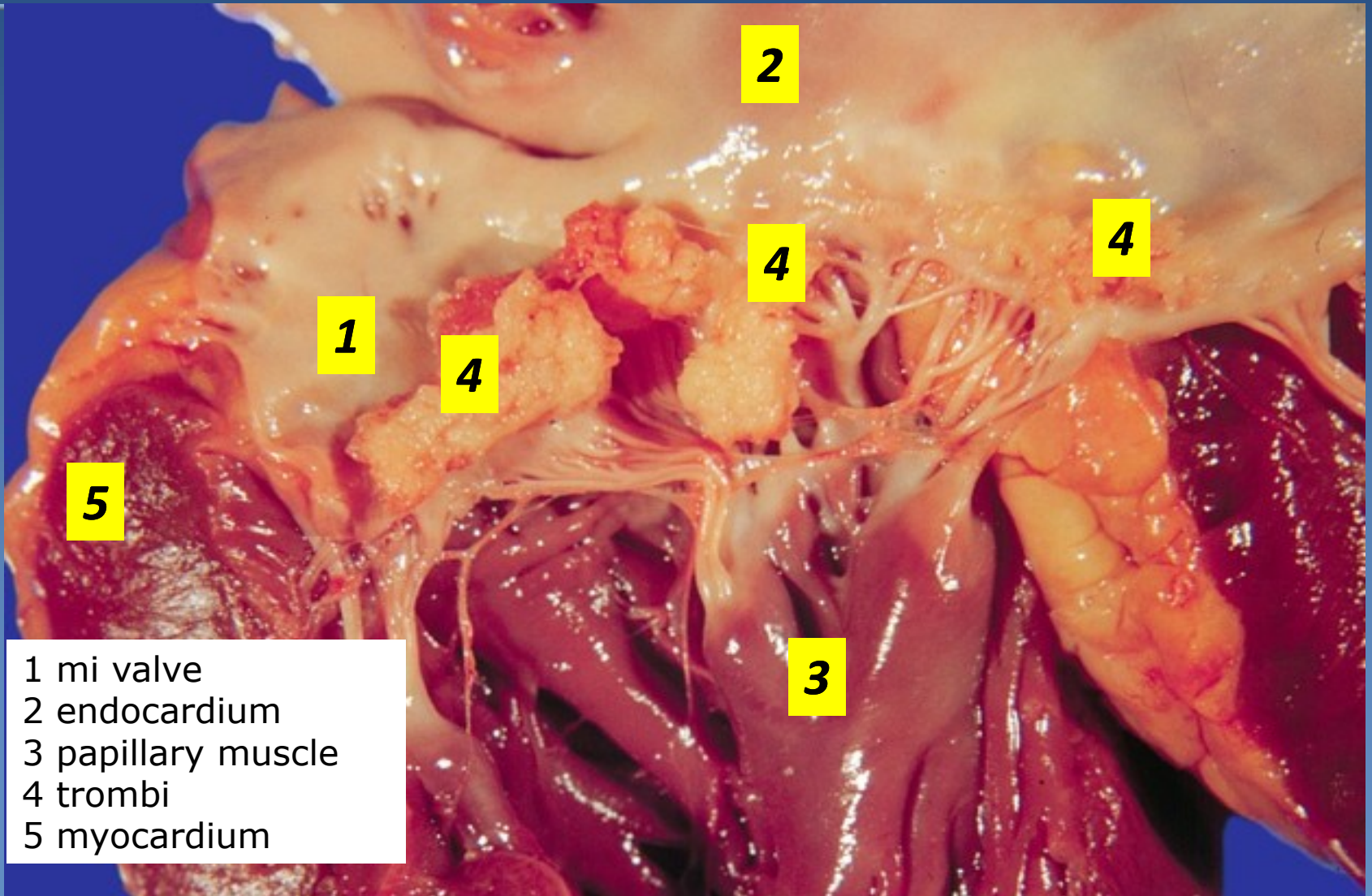
1 bacterial colonies  
2 valve neutrophilic infiltrated  
granulocytes

# ***Non-bacterial thrombotic endocarditis***



- ✗ **sterile** vegetations due to **hypercoagulative state**  $\Rightarrow$  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, **imune-mediated** systemic poststreptococcal inflammation (cross-reactive antibodies)

× acute stage: **PANCARDITIS**

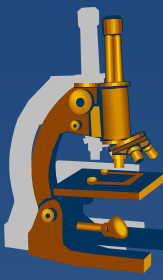
⇒ *fibrinous pericarditis + myocarditis with Aschoff bodies (foci of **fibrinoid necrosis** + inflammatory reaction + verrucous endocarditis (small depositions of fibrin along the closure lines of Ao a Mi valves)*

⇒ *acute endocarditis commonly recurrent*

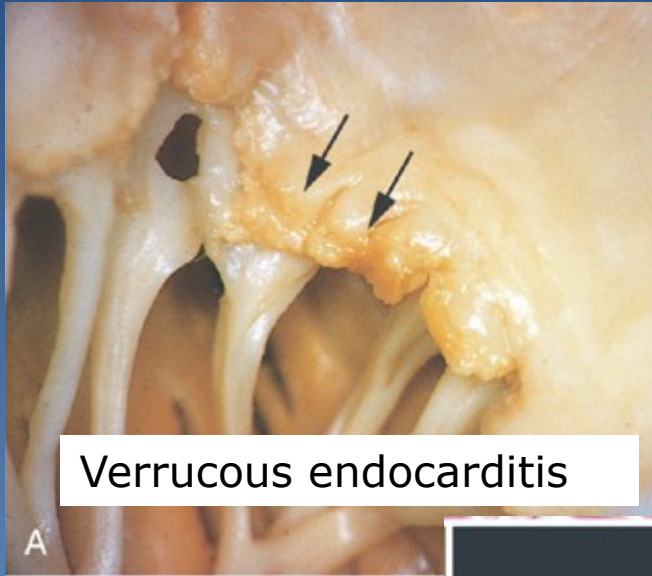
× chronic stage:

⇒ *diffuse fibrous thickening + distortion, commissural fusion → dystrophic calcification - stenosis + incompetence)*

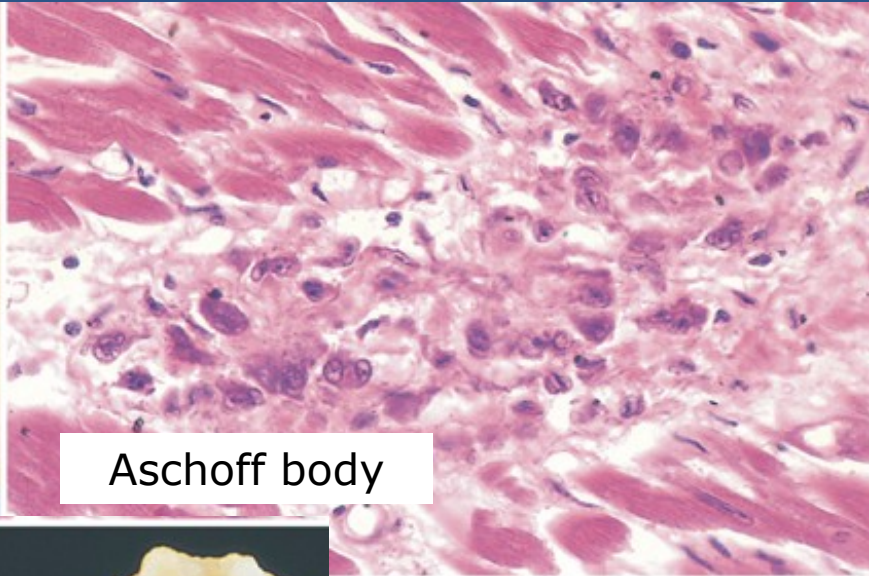




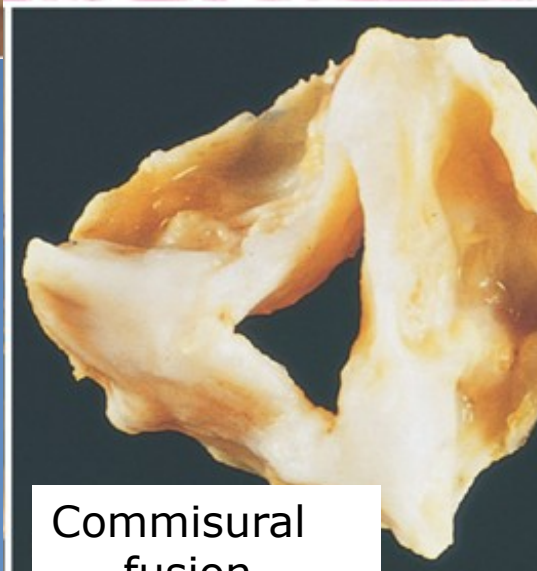
# *rheumatic heart disease*



Verrucous endocarditis

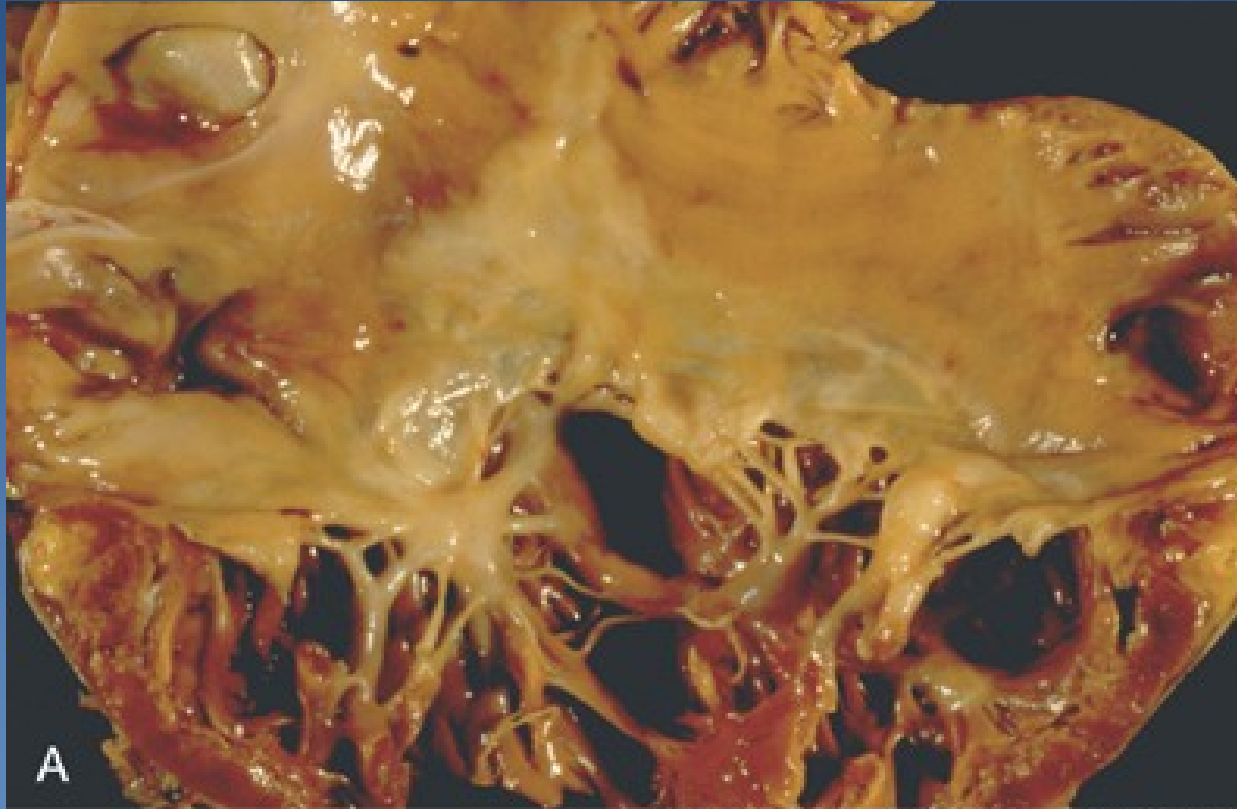


Aschoff body



Commissural fusion

# *Carcinoid syndrome*



endocardial fibrous plaquelike thickenings – RA, RV

# Myocarditis

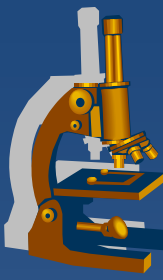


- ✗ myokardial inflammatory damage without ischemia
  
- ✗ **gross:**
  - ⇒ *cardiac dilatation, flabby, mottled myocardium*
  
- ✗ **micro:**
  - ⇒ *inflammatory infiltrate (according etiology) + cardiomyocyte regressive changes incl. necrosis*
  
- ✗ **etiology:**
  - ⇒ viruses, rickettsia, chlamydia, bacteria (diphtheria, sepsis), fungi, protozoa (toxoplasmosis), helminths (trichinosis)
  - ⇒ immune-mediated (*drug hypersensitivity, postviral, rheumatic fever, rejection*)
  - ⇒ *ionising radiation*
  - ⇒ unknown (*giant-cell myocarditis*)

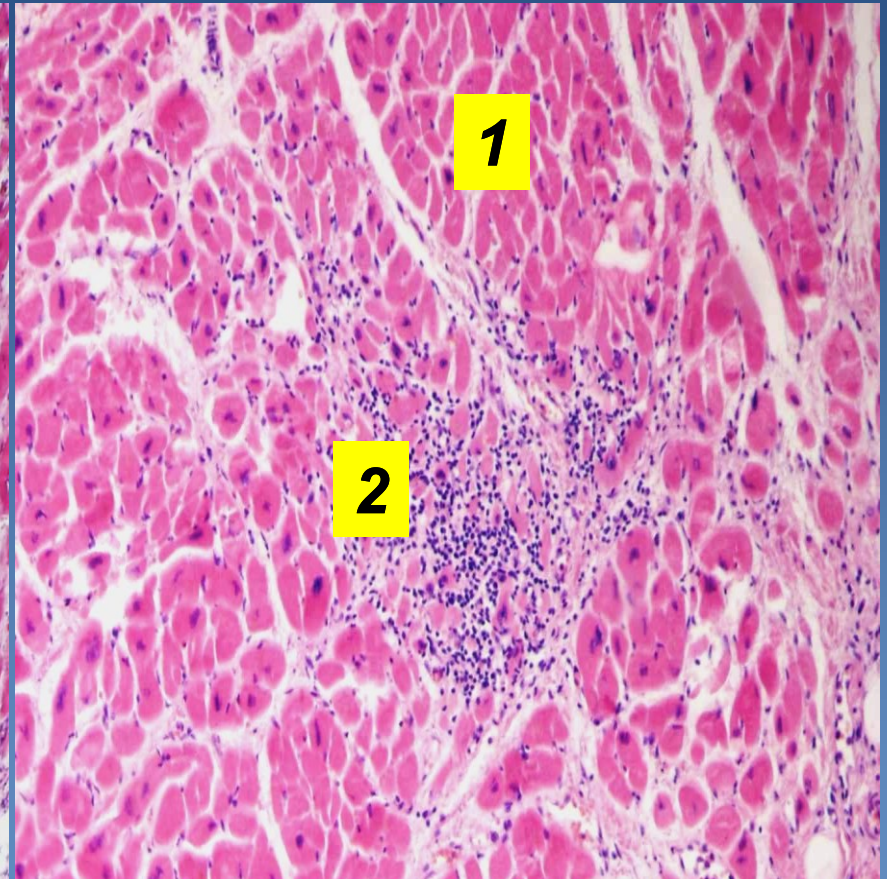
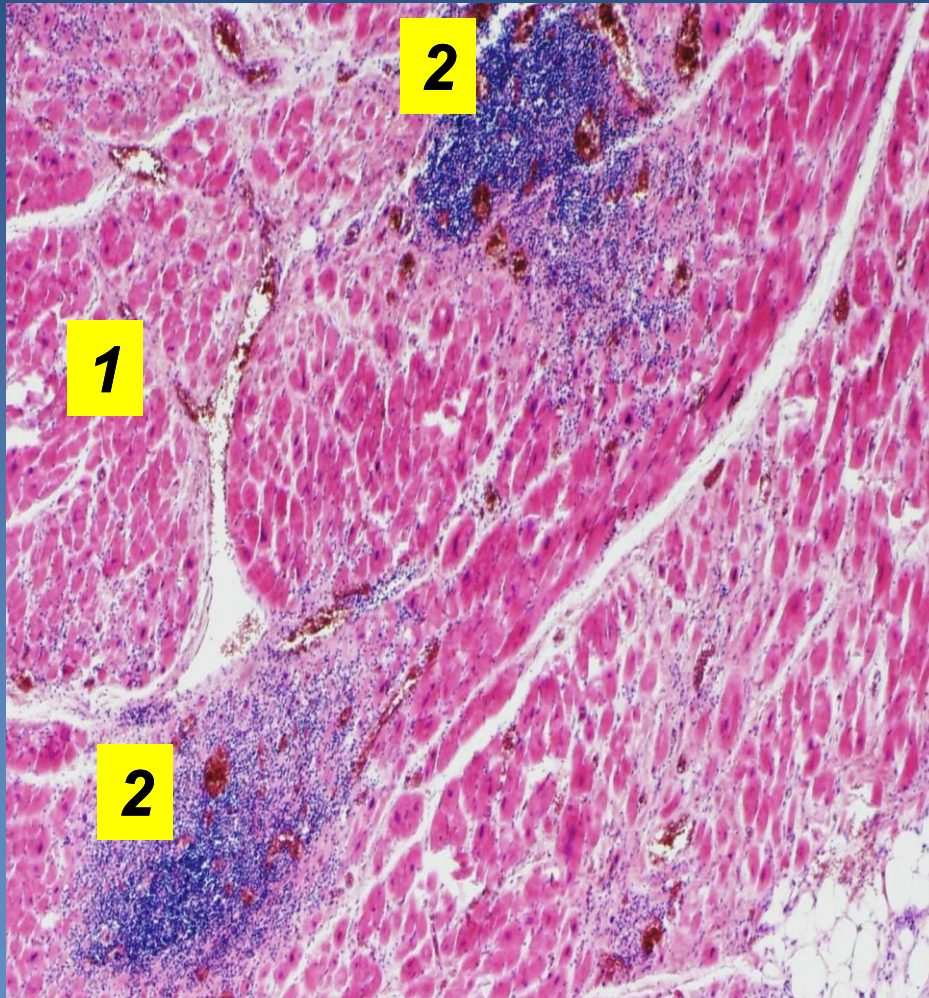
# *Viral myocarditis*



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

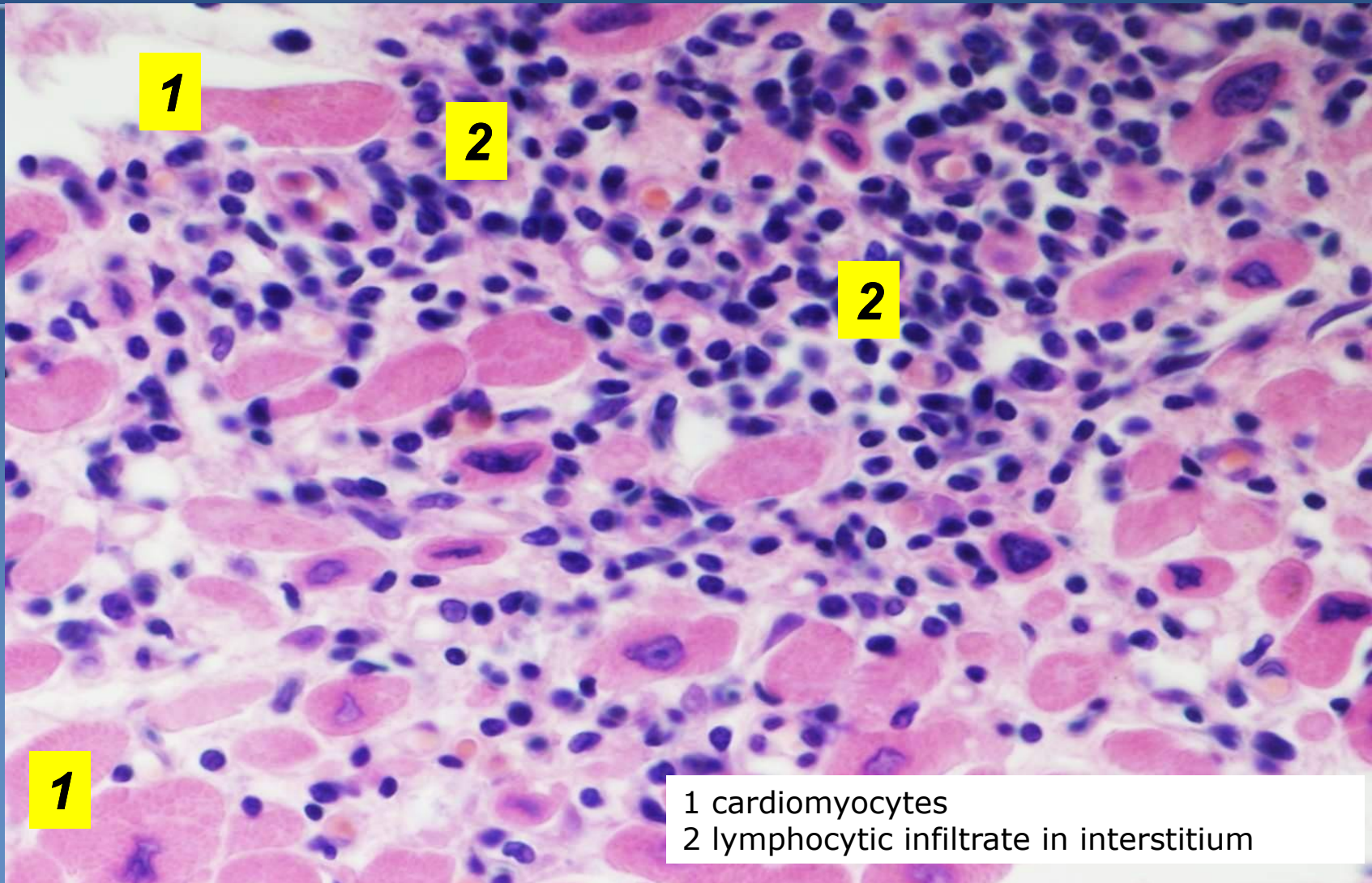
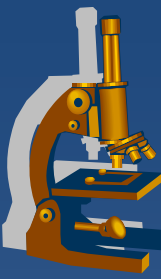


# *Viral myocarditis*



1 cardiomyocytes  
2 lymphocytic infiltrate in interstitium

# *Viral myocarditis*



1

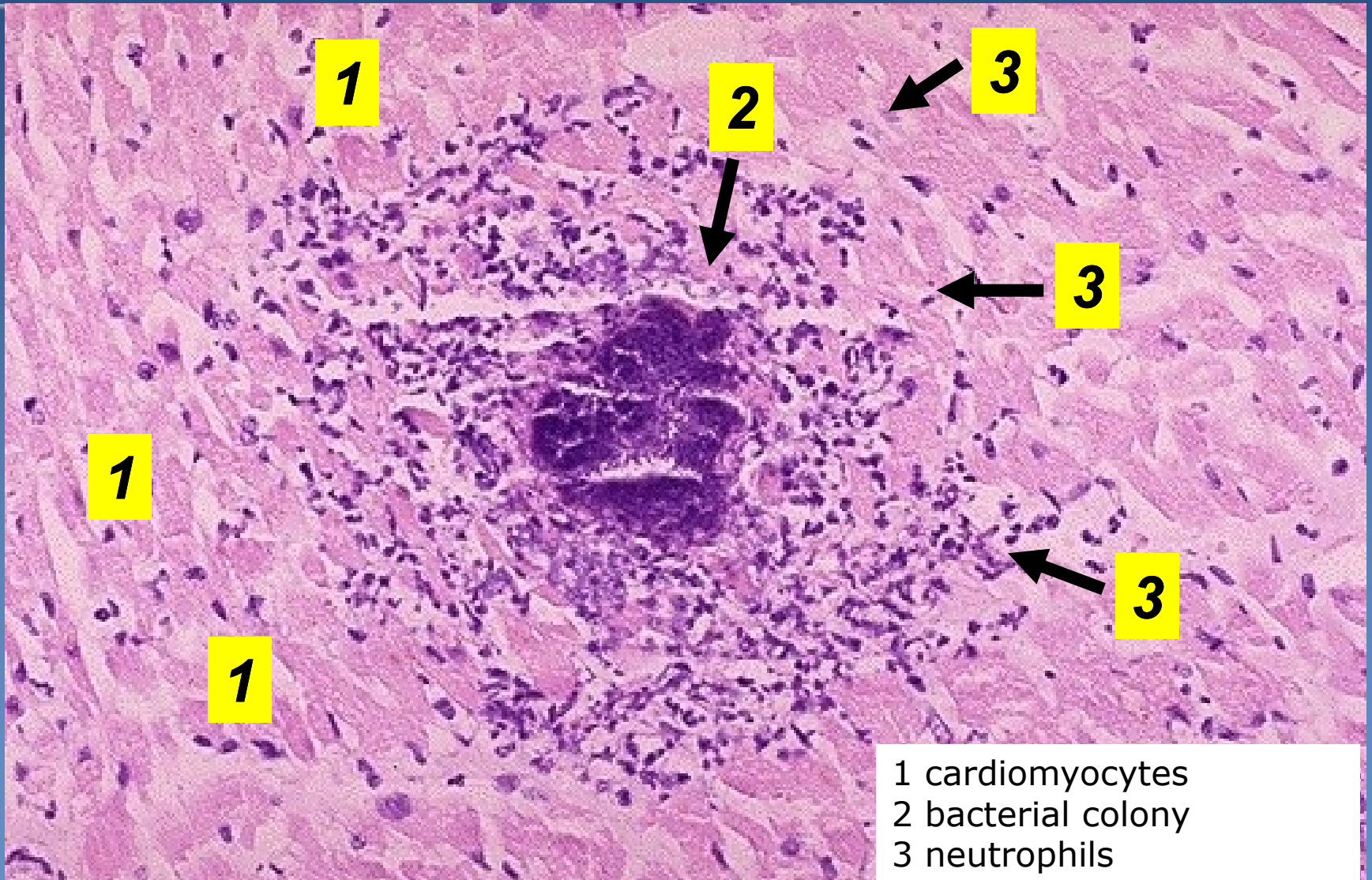
1

2

2

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);  
*alcoholic, 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, ...)

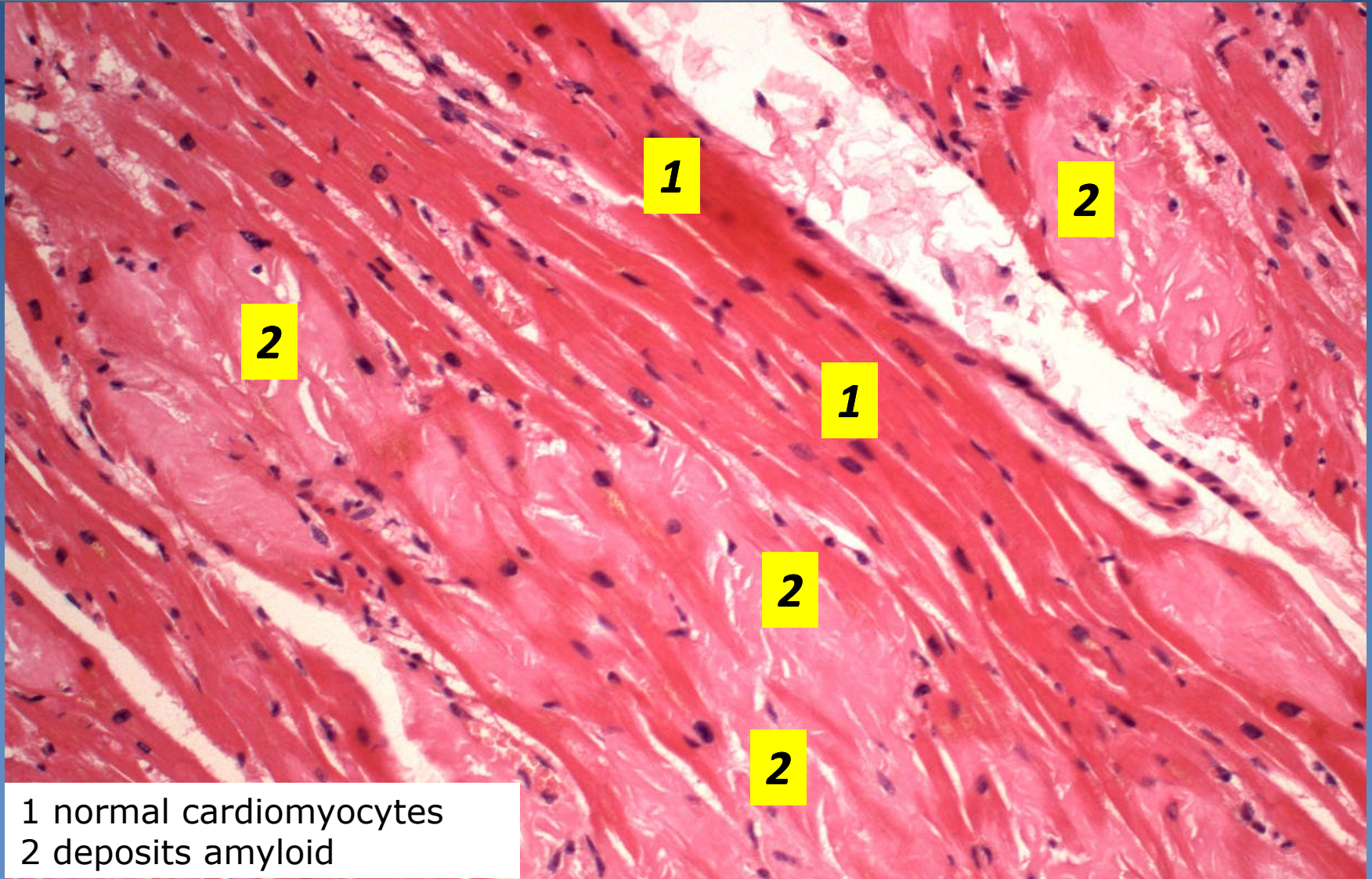
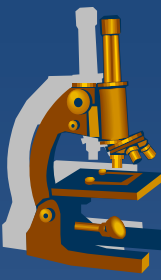


# Myocardial amyloidosis



- ✗ 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 failure or hypoproteinemia, slow (up to 500ml – pericardial dilatation)

## 2) haemopericardium

– wall rupture in 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)

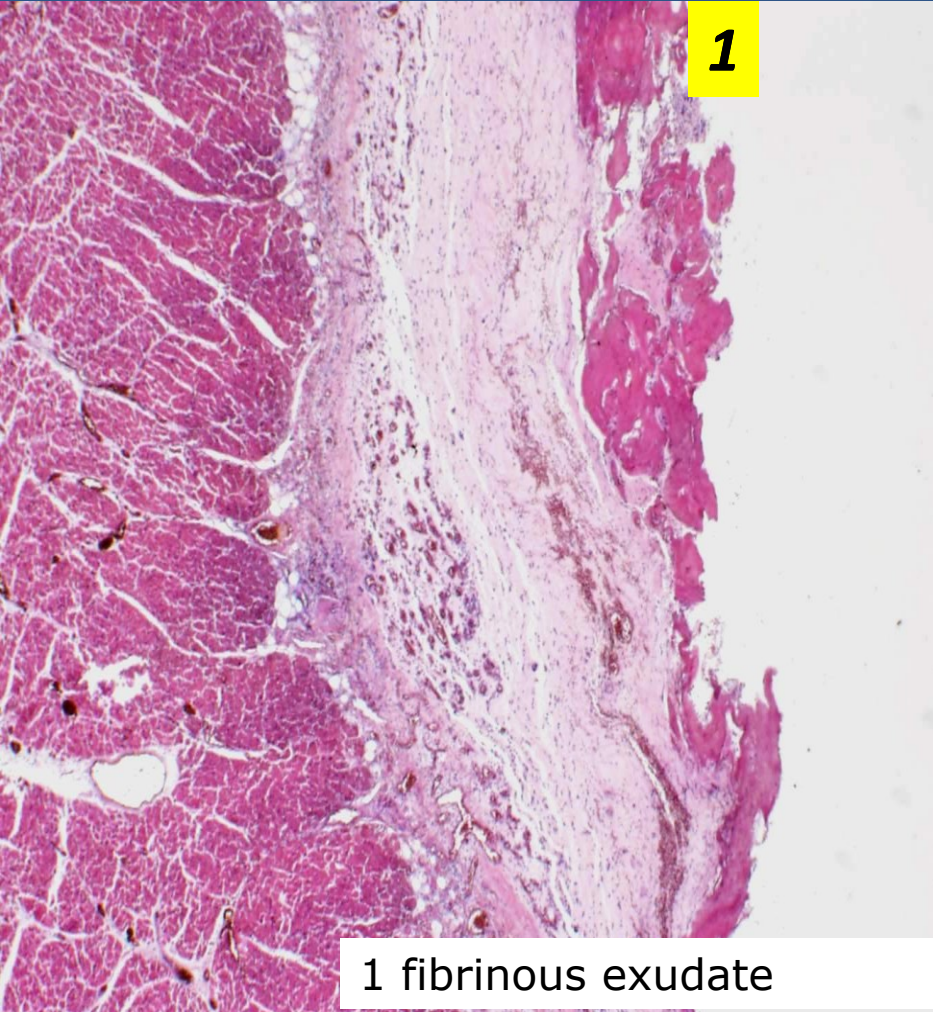
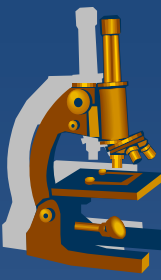
### b) *infectious*

– *haematogenous, direct spread, lymphogenous; variable agents*

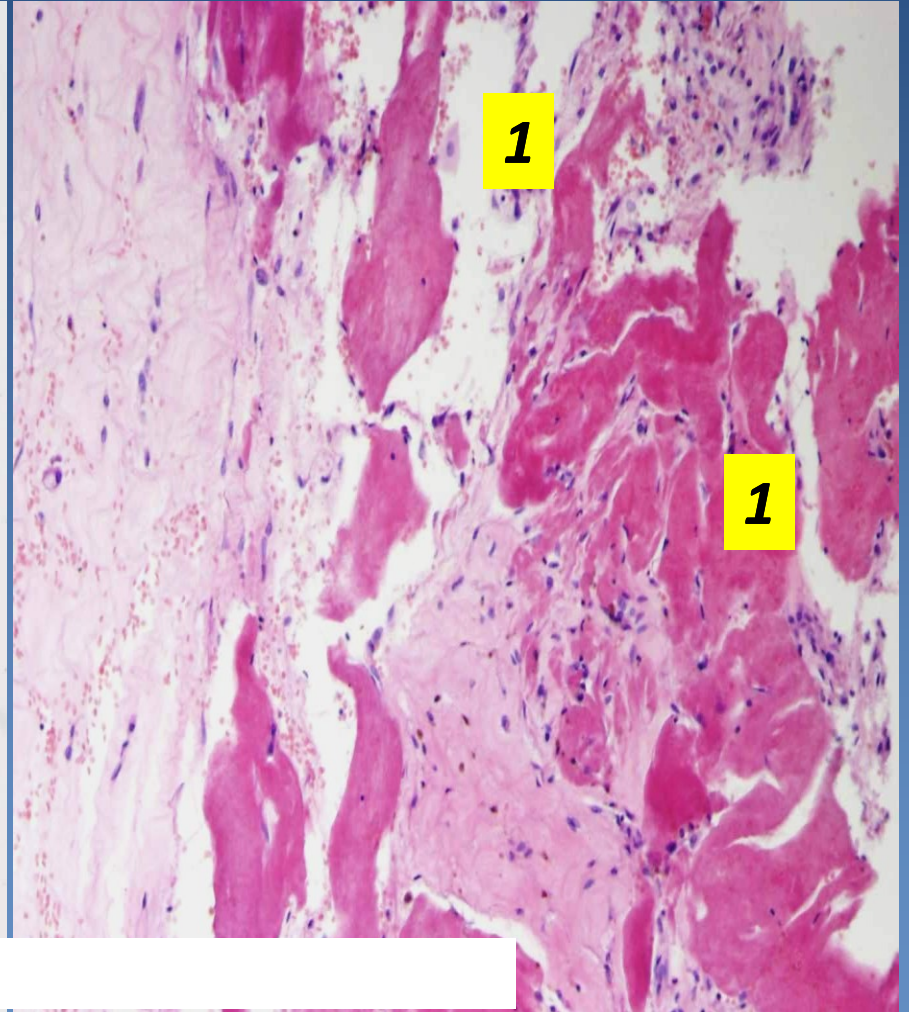
### c) *idiopatická*

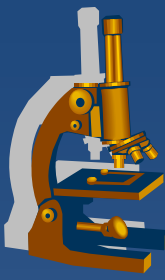
**Pozn. hojení** – serózní a část. i fibrinózní exsudát → vstřebávání x zbývající fibrin se organizuje → **perikardiální adheze /konstriktivní perikarditida** (pericarditis petrosa) → omezuje plnění komor

# *fibrinous pericarditis*



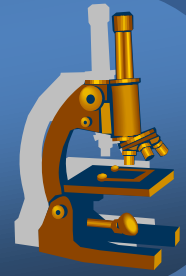
1 fibrinous exudate





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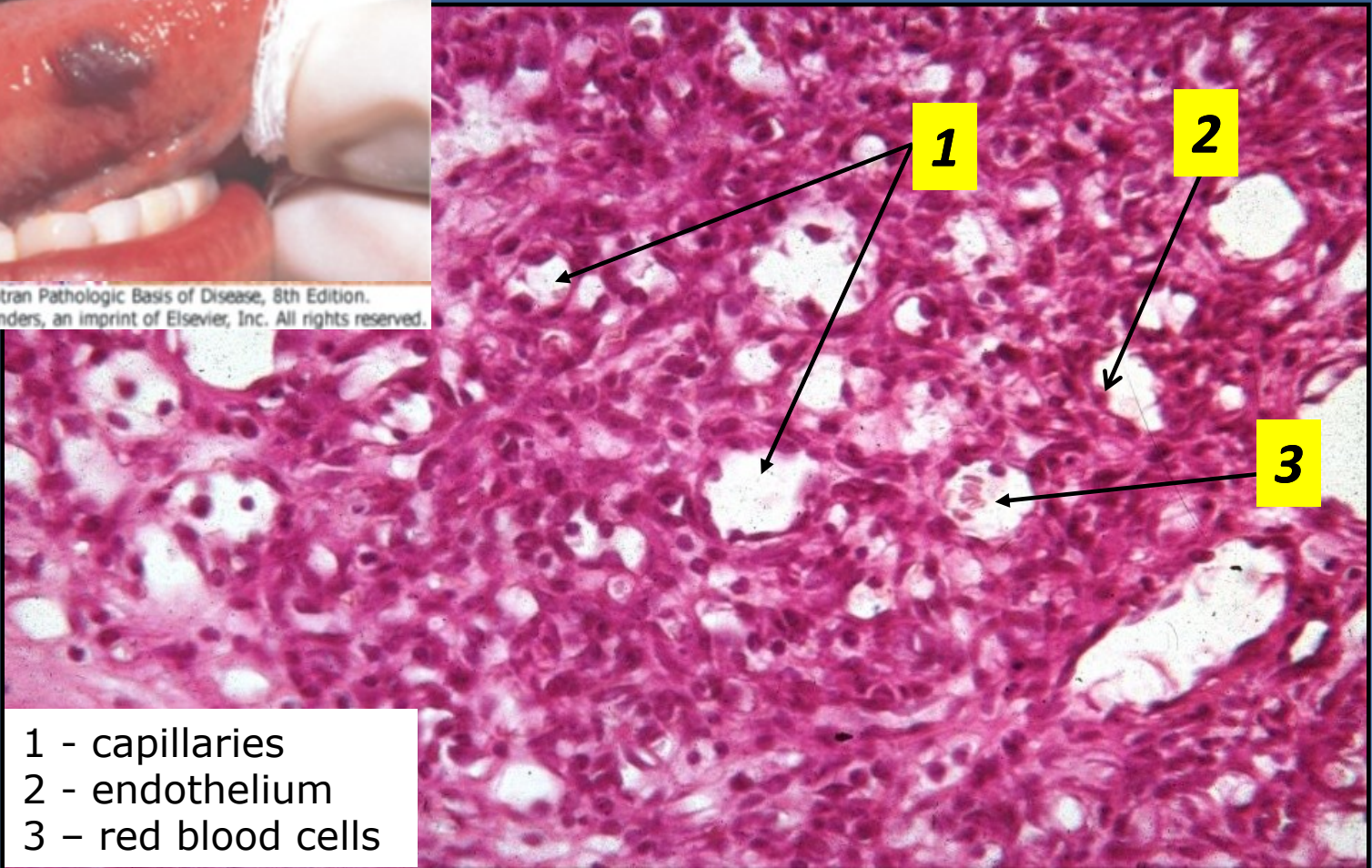
# *Cardiovascular tumors*



# Capillary hemangioma

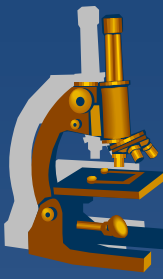


Kumar et al: Robbins & Cotran Pathologic Basis of Disease, 8th Edition.  
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- 1 - capillaries
- 2 - endothelium
- 3 - red blood cells

# *Cavernous hemangioma*



## **x**gross:

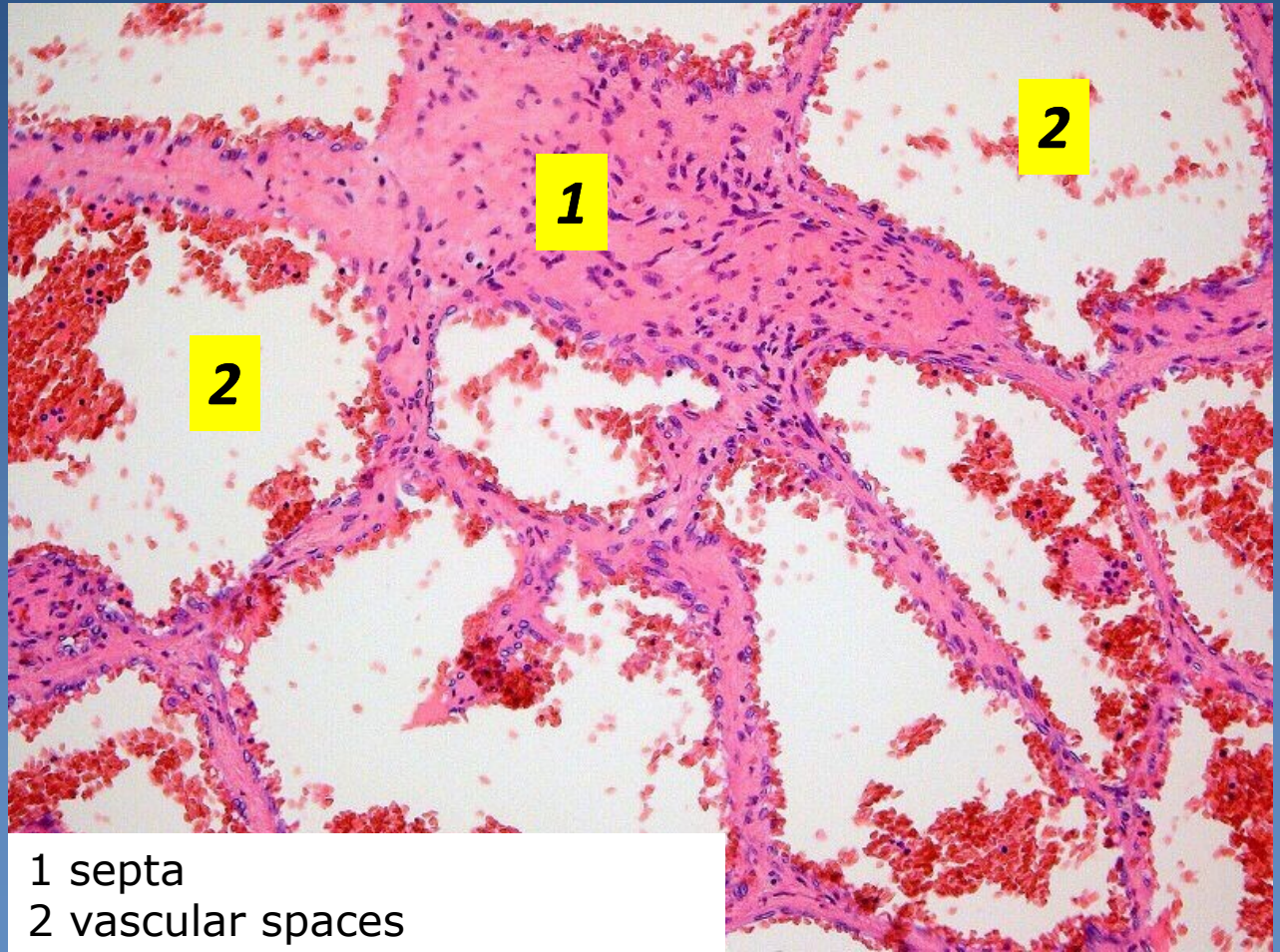
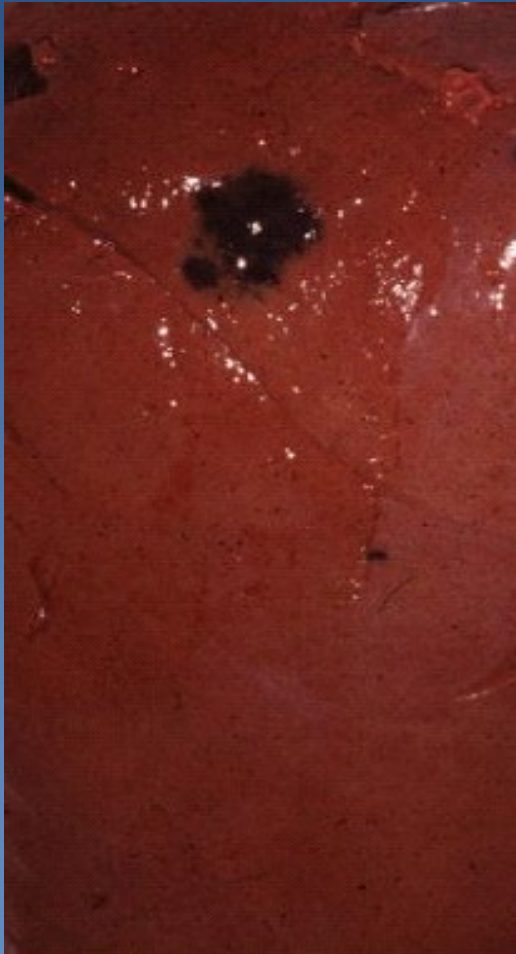
- ⇒ red -blue focus (nodular)
- ⇒ possible large size (-15 cm)
- ⇒ liver, spleen, skin; commonly multiple

## **x**micro:

- ⇒ large blood-filled vascular spaces divided by fibrous septa



# *Cavernous hemangioma*



1 septa  
2 vascular spaces

# *Kaposi sarcoma*



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# Kaposi sarcoma



## × classic form

- ⇒ *chronic*
- ⇒ *in mediterranean or jewish origin*
- ⇒ *usually (90%) confined to skin*

## × endemic

- ⇒ *south-african children*
- ⇒ *lymphadenopathic*
- ⇒ *aggressive*

## × immunosuppression (transplant) associated

- ⇒ *– internal organs in 50%*

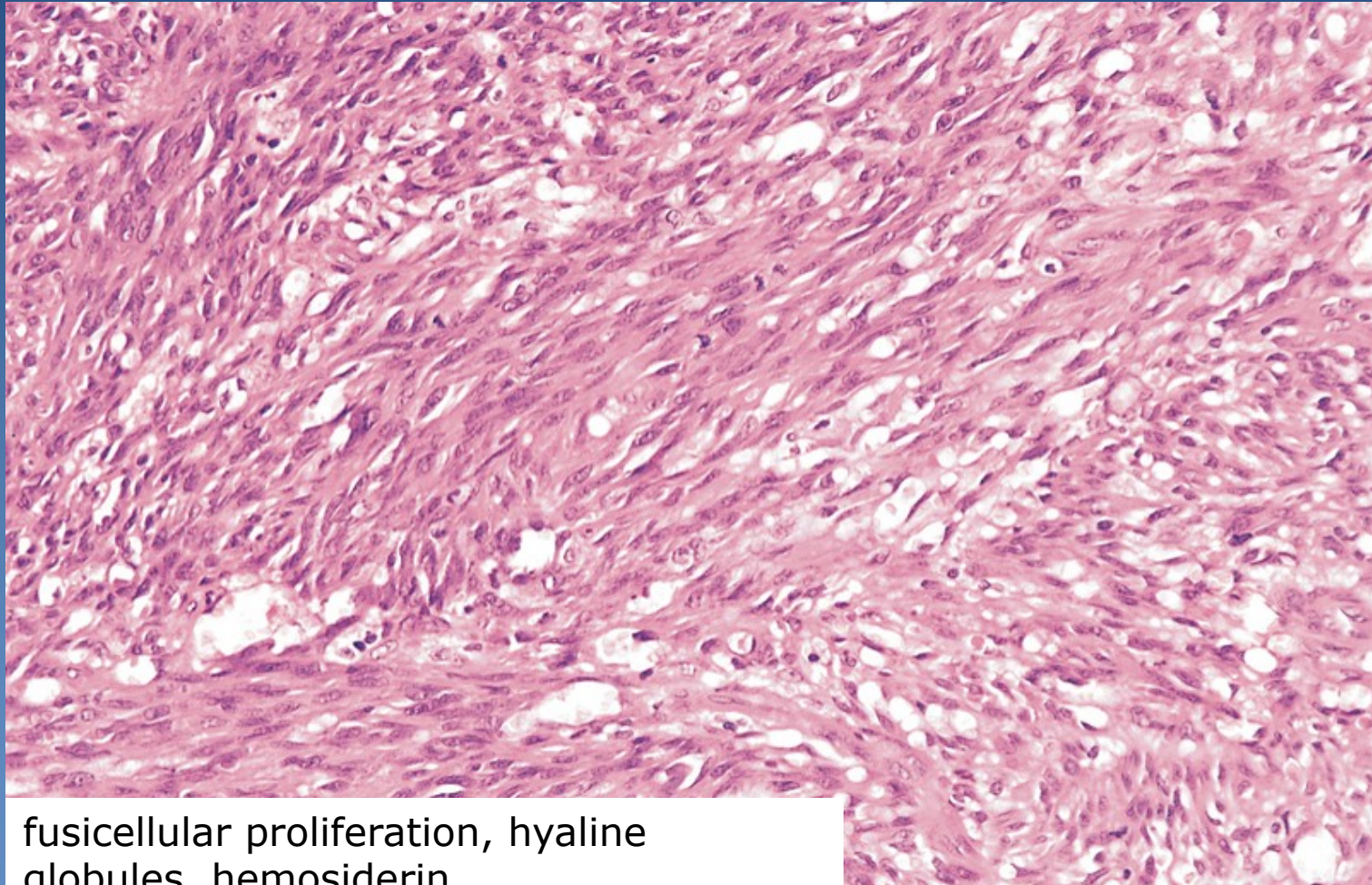
## × AIDS associated

# *Kaposi sarcoma*



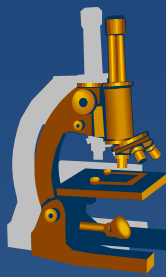
- × HHV-8
- × hyperproliferation of endothelial cells
- × prevention of apoptosis
  
- × **gross:**
  - ⇒ *red to purple patches*
  - ⇒ *raised plaques*
  - ⇒ *nodules*
  
- × **micro:**
  - ⇒ *irregular blood spaces*
  - ⇒ *plump atypical endothelial cells*
  - ⇒ *perivascular aggregates of spindle cells*

# *Kaposi sarcoma*



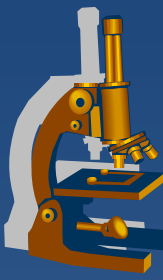
fusicellular proliferation, hyaline globules, hemosiderin

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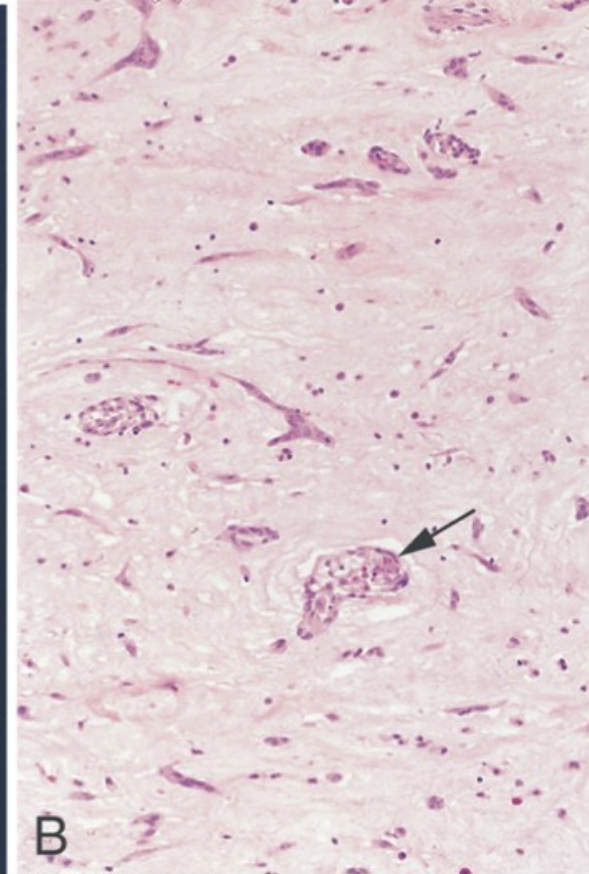
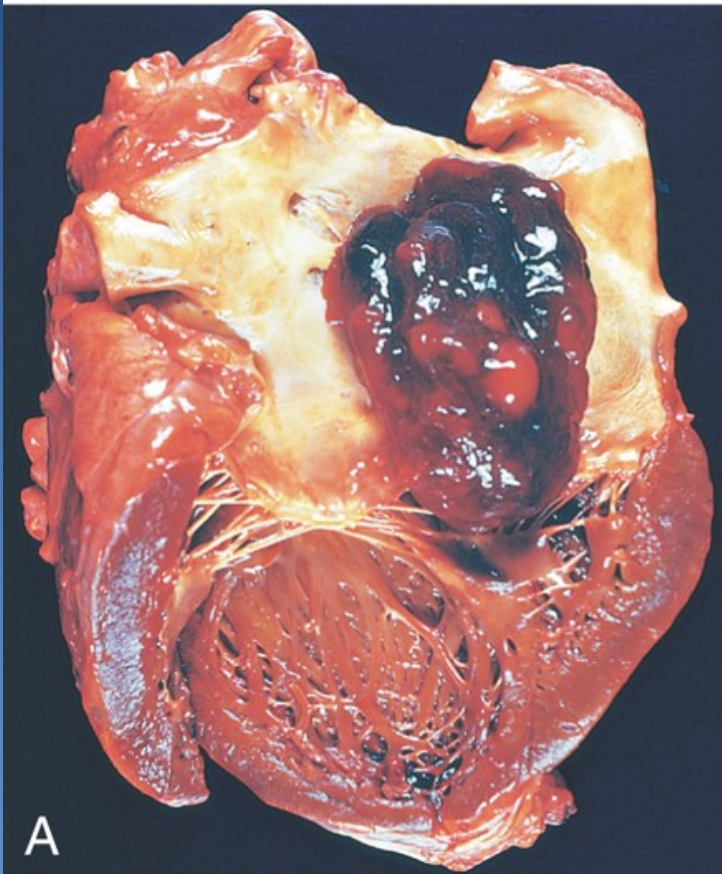


# Heart tumors

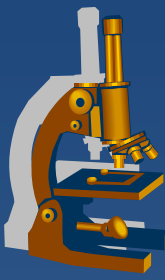
- ✗ primary tumors rare, mostly **benign myxomas**
- ✗ malignant mesenchymal (sarcomas)
  - ⇒ *leiomyo - , rhabdomyo - , hemangio - , fibrosarcoma*
- ✗ secondary tumors
  - ⇒ *20-30 x more common than primary*
  - ⇒ *metastases + infiltrates : lung, breast carcinomas, malignant melanoma, malignant lymphomas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



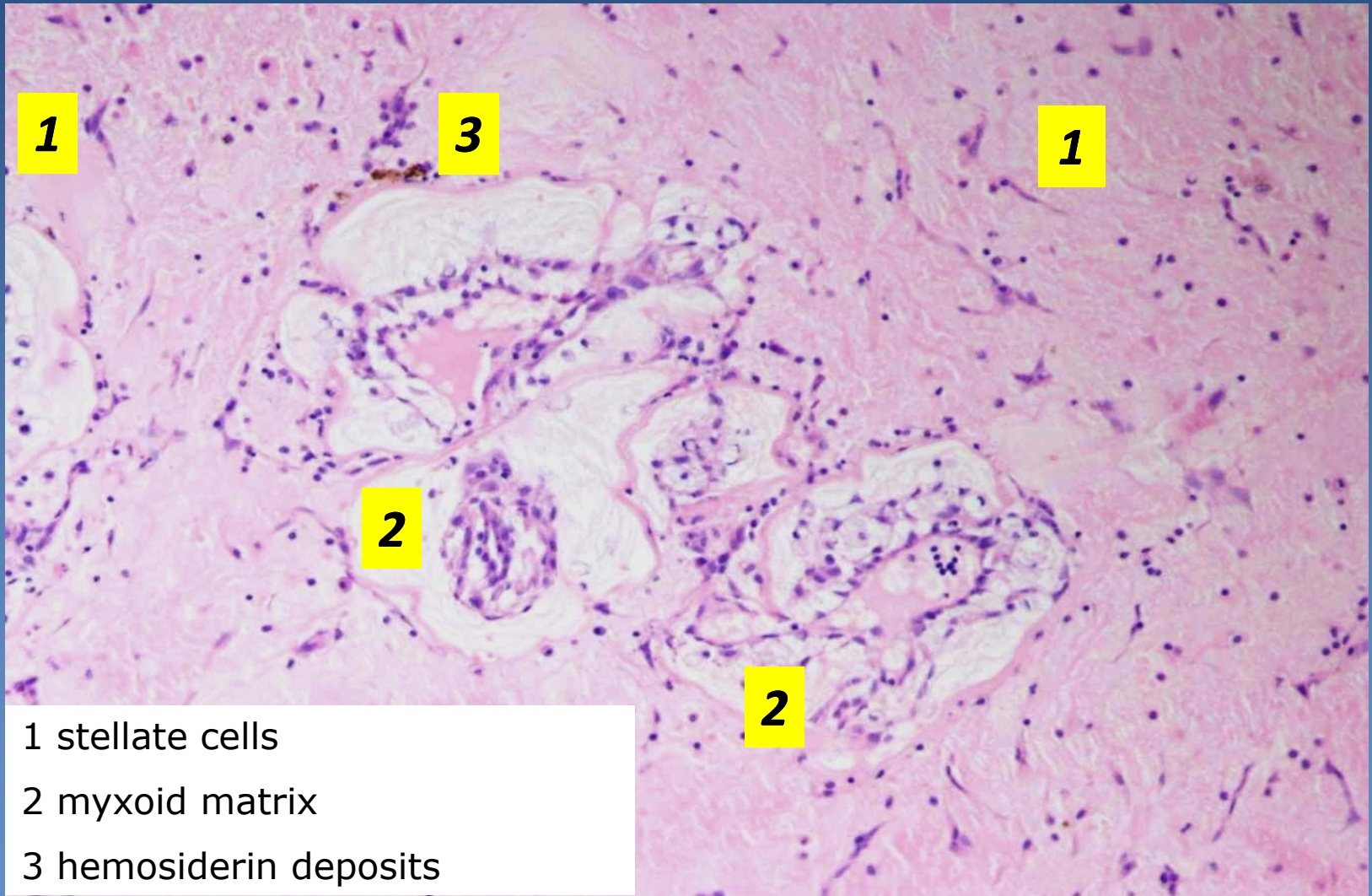
# *LV myxoma*



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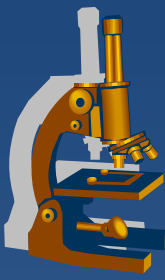


# *Myxoma (100x)*

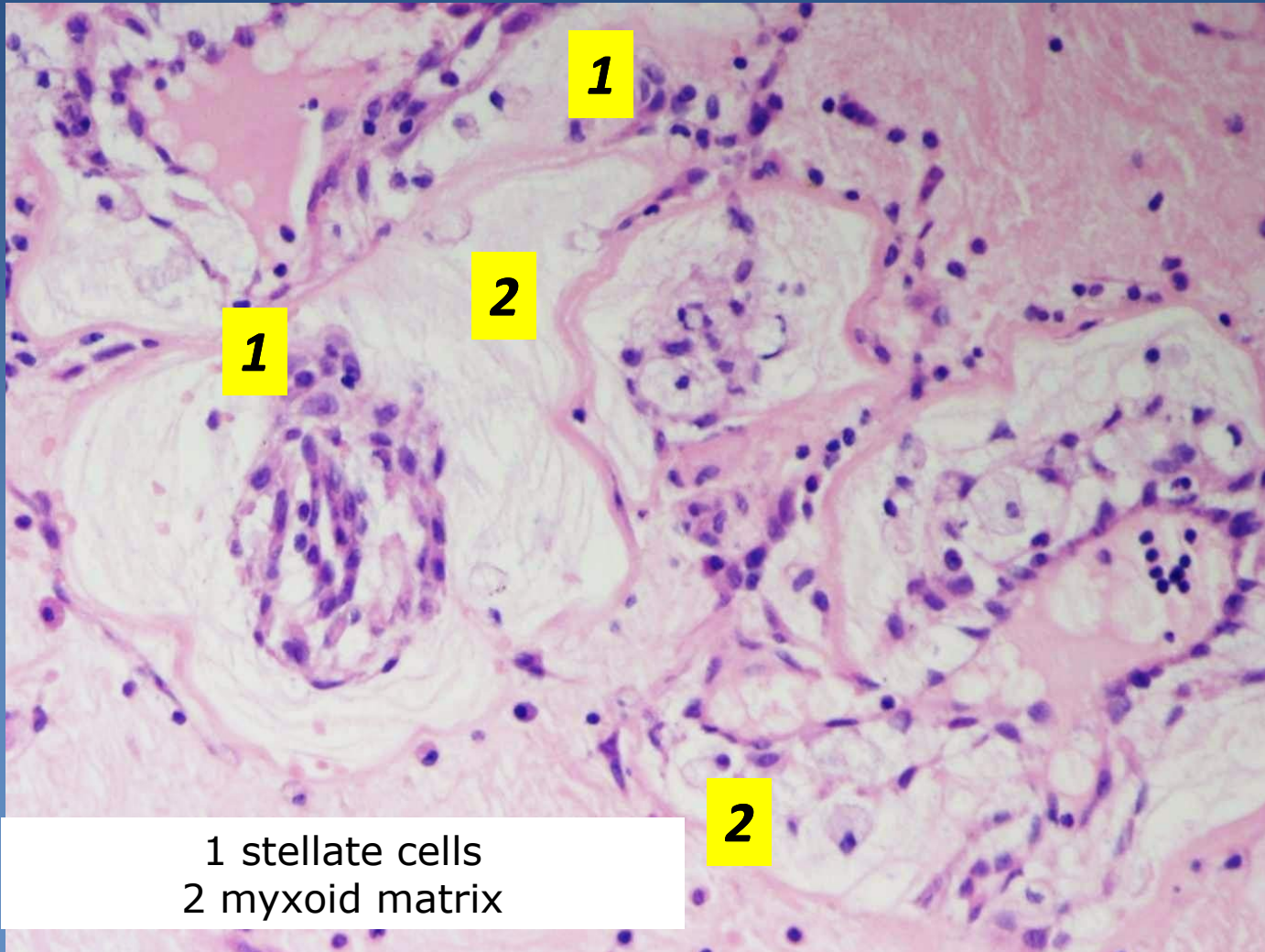


- 1 stellate cells
- 2 myxoid matrix
- 3 hemosiderin deposits

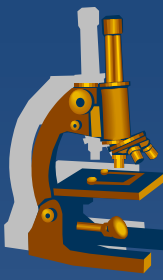




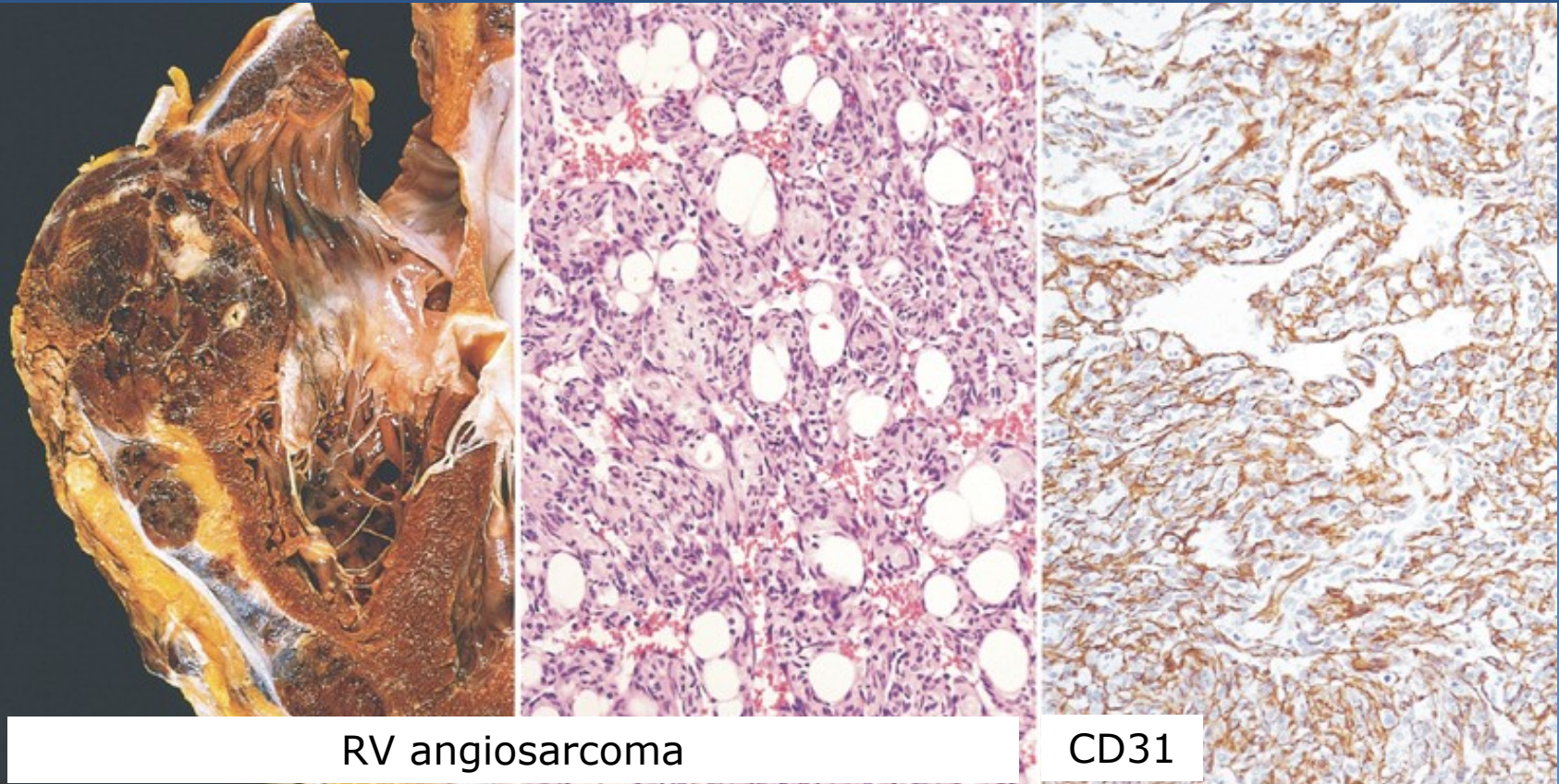
# Myxoma (400x)



1 stellate cells  
2 myxoid matrix



# Angiosarcoma



RV angiosarcoma

CD31

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# Angiosarcoma

