Cardiovascular system

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Cardiovascular diseases

- Major cause of morbidity and mortality (1/3 of deaths)
- Leading risk factors: ↑ serum cholesterol level, smoking, hypertension (CV disease itself), obesity/metabolic syndrome, sedentary lifestyle

Common clinical signs

- General
  - Weakness
  - Fatigue
  - Weight change (↑ due to edema)
  - Poor exercise tolerance
  - Cyanosis

Common clinical signs

- Musculoskeletal
  - Muscular fatigue / pain
  - Chest, shoulder, neck, jaw, arm pain / discomfort
  - Peripheral edema
  - Intermittent claudication (leg pain / cramps / discomfort)

Common clinical signs

- Dizziness (abnormal blood pressure, cardiac arrythmia)
- Headache (arteritis, hypertension)
- Loss of vision (retinopathy, transient ischemic attack)
- Chest pain (myocardial ischemia/infarction, pulmonary embolism, aortic dissection)
- Palpitations (ischemic heart disease, valvular disease)

Common clinical signs

- Cough (left ventricular failure, antihypertensive drugs – ACE inhibitors)
- Acute dyspnea (acute left ventricle failure, pulmonary embolism)
- Chronic dyspnea (congestive cardiac failure, aortic valve disorder, congenital heart disease)
- Swollen ankles (congestive cardiac failure, venous insufficiency)

Implication for the therapist

- Evaluation of possible cardiac signs
- Assessment of possible risks of adverse cardiac event
- Evaluation of type/degree of organ impairment, level of disability, functional limitations
- Individual exercise program (mode, duration, intensity, frequency) commonly necessary

Implication for the therapist

Exercise

- Primary and/or secondary prevention of cardiovascular diseases
- Increase of CV functional capacity, ↓ of myocardial oxygen demand
- Adjunctive therapy for lipid management (endurance exercise)

Indications for discontinuing/modifying exercise

- Symptoms
  - New-onset or easily provoked anginal chest pain
  - ↑ episodes, intenstity, duration of angina (unstable angina)
  - Discomfort in the upper body
  - Fainting, dizziness
  - Sudden severe dyspnea
  - Severe fatigue
  - Nausea, vomiting
  - Back pain during exercise

Indications for discontinuing/modyfying exercise

- Clinical signs
  - Pallor, peripheral cyanosis; cold + moist skin
  - Confusion
  - Resting heart rate >130/min or ≤40/min
  - Arrythmias (irregular heartbeats, palpitation)
  - Blood pressure (BP) abnormalities: fall in systolic BP during increasing workload; rise of systolic BP >250 mm Hg and/or diastolic >115 mm Hg
  - Inability to converse during activity
  - Signs of CNS involvment (confusion, delirium, stroke, …)
  - Recent myocardial infarction (within 48 hours)
  - Acute infection or fever >37,8 C

Morphology

- pericardial sac – cca 30ml clear yellowish fluid

- heart size – approx. the person‘s closed fist
  - **male = 300 – 350 g**,
    - hypertrophy > 400g

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

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

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 and/or extracardial pathologic changes

Cardial changes

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

- differencies due to rapidity of development:

  – sudden → *acute dilatation*

  – chronic → *adaptation* → → →

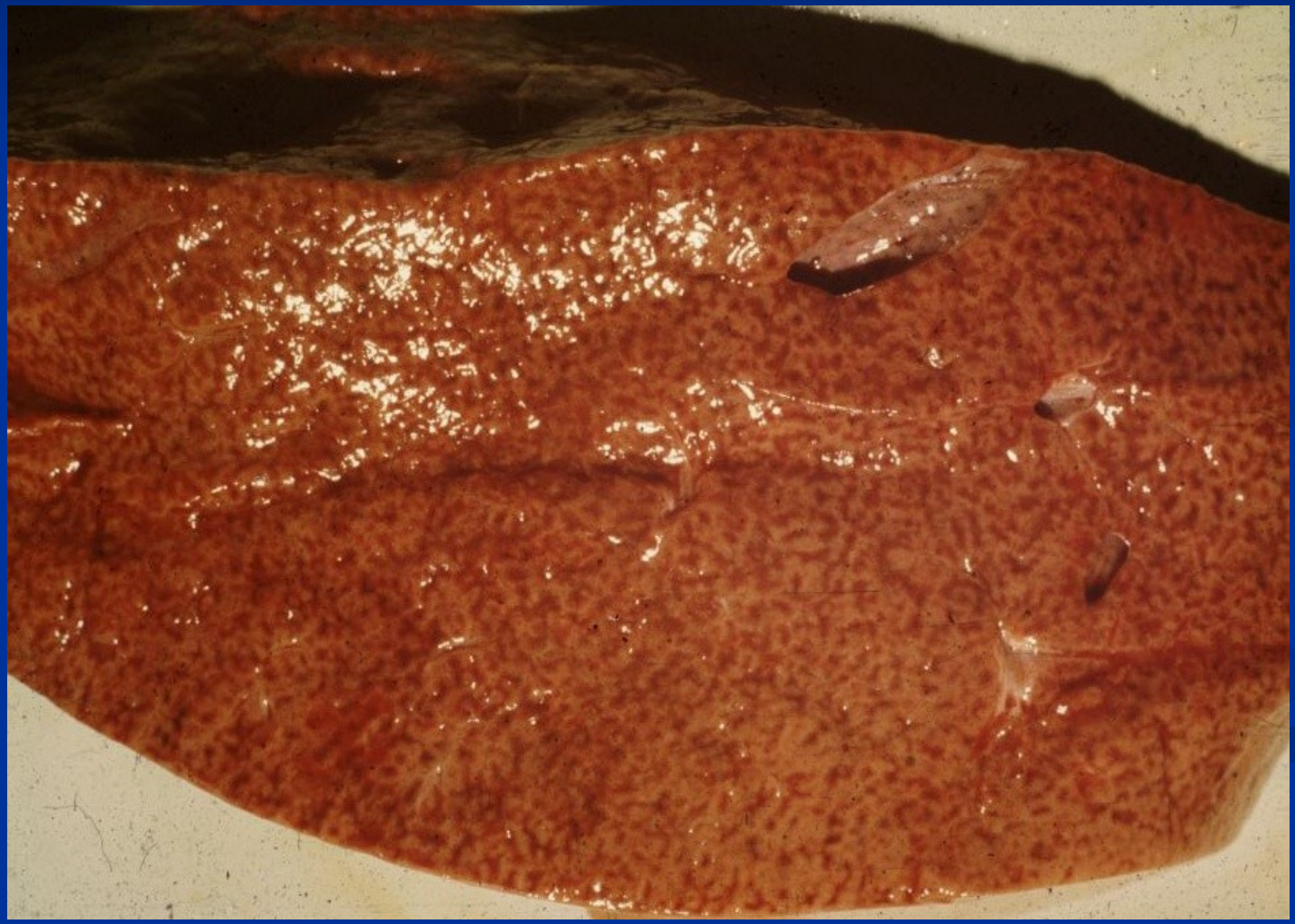
  myocardial hypertrophy (↑ nutritional demands) +/- ventricular dilatation (enhanced contractility), + activation of neurohumoral systems (norepinephrin, renin-angiotensin system, atrial natriuretic peptide)

  if not treated → progression into heart failure

Extracardial changes

- **venous congestion** (filled vessels)– blood stays ahead of the heart, *e.g. liver (→ hepar moschatum)*
- **induration** (firmer consistency) – decreased oxygen + nutrients → loss of functional cells + fibroproduction *(liver, spleen, kidney)*
- **oedema** – congestion + outflow of fluid from capillaries *visible /palpable in soft tissues*
- **cyanosis** (bluish discoloration) – increased level of deoxygenated hemoglobin *visible on acral parts*

Chronic venous congestion (nutmeg liver - hepar moschatum)



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

- **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
- **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.

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

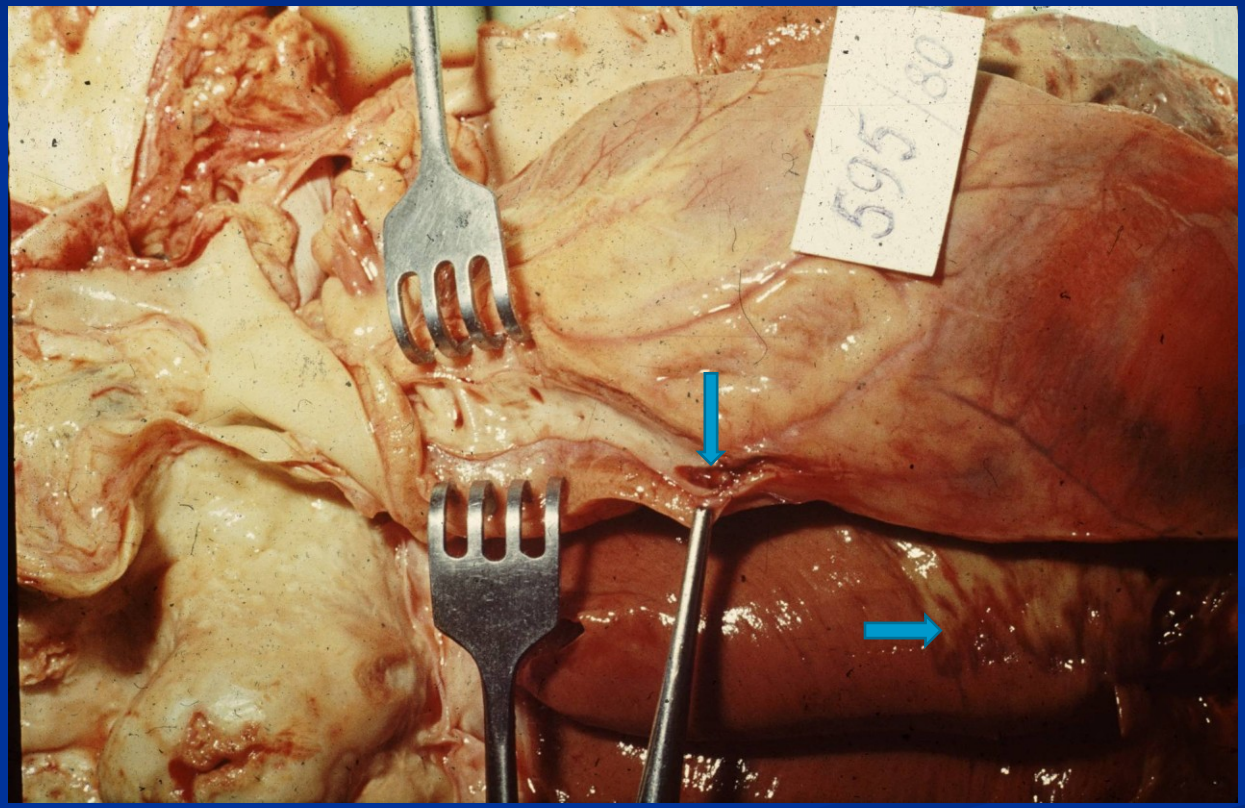
Myocardial infarction

- **ischaemic coagulative necrosis**

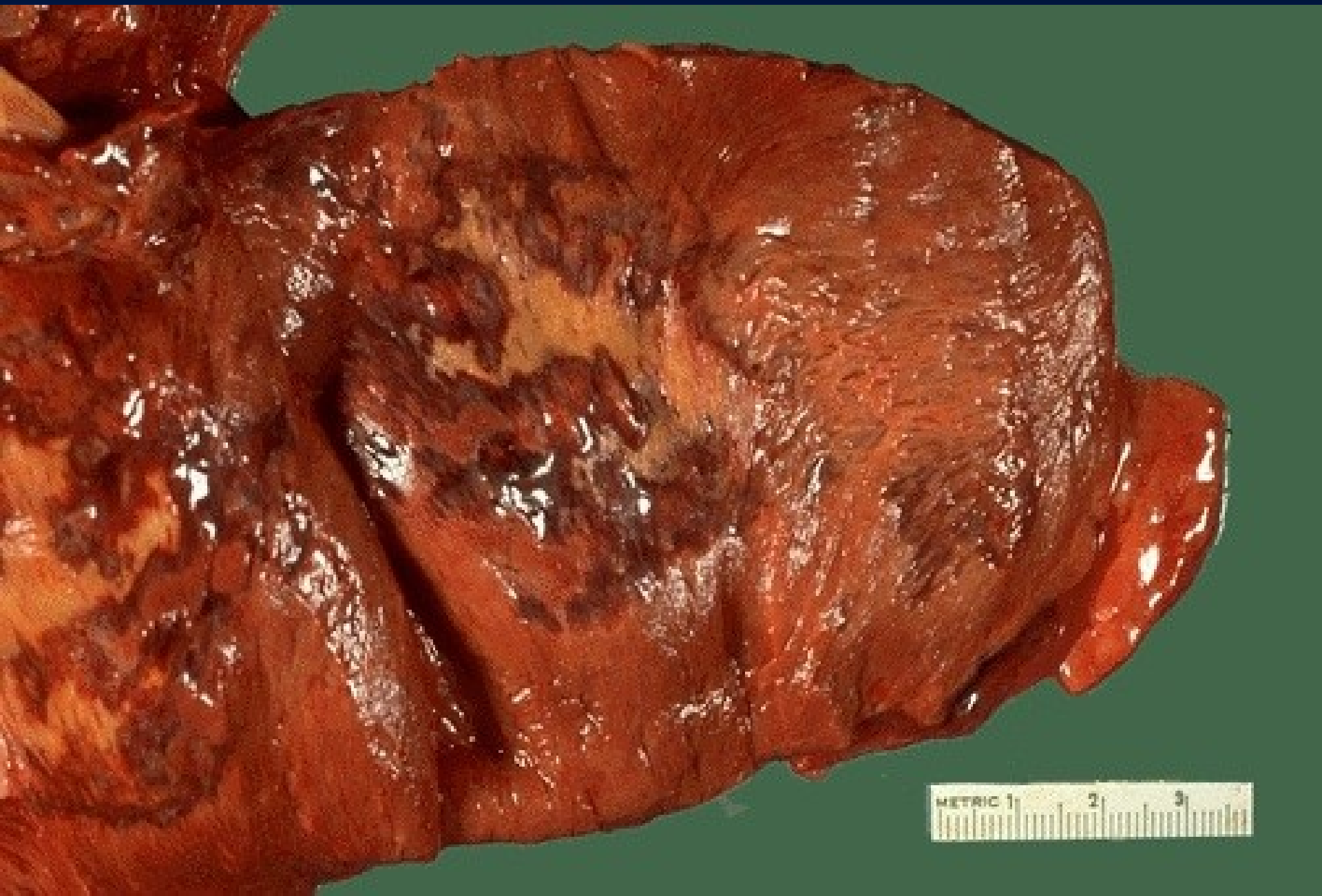
- causes
  - usually coronary thrombosis
  - complicated atheromatic plaque
  - event. embolism
  - spasm
  - inflammation
  - rarely systemic causes.

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

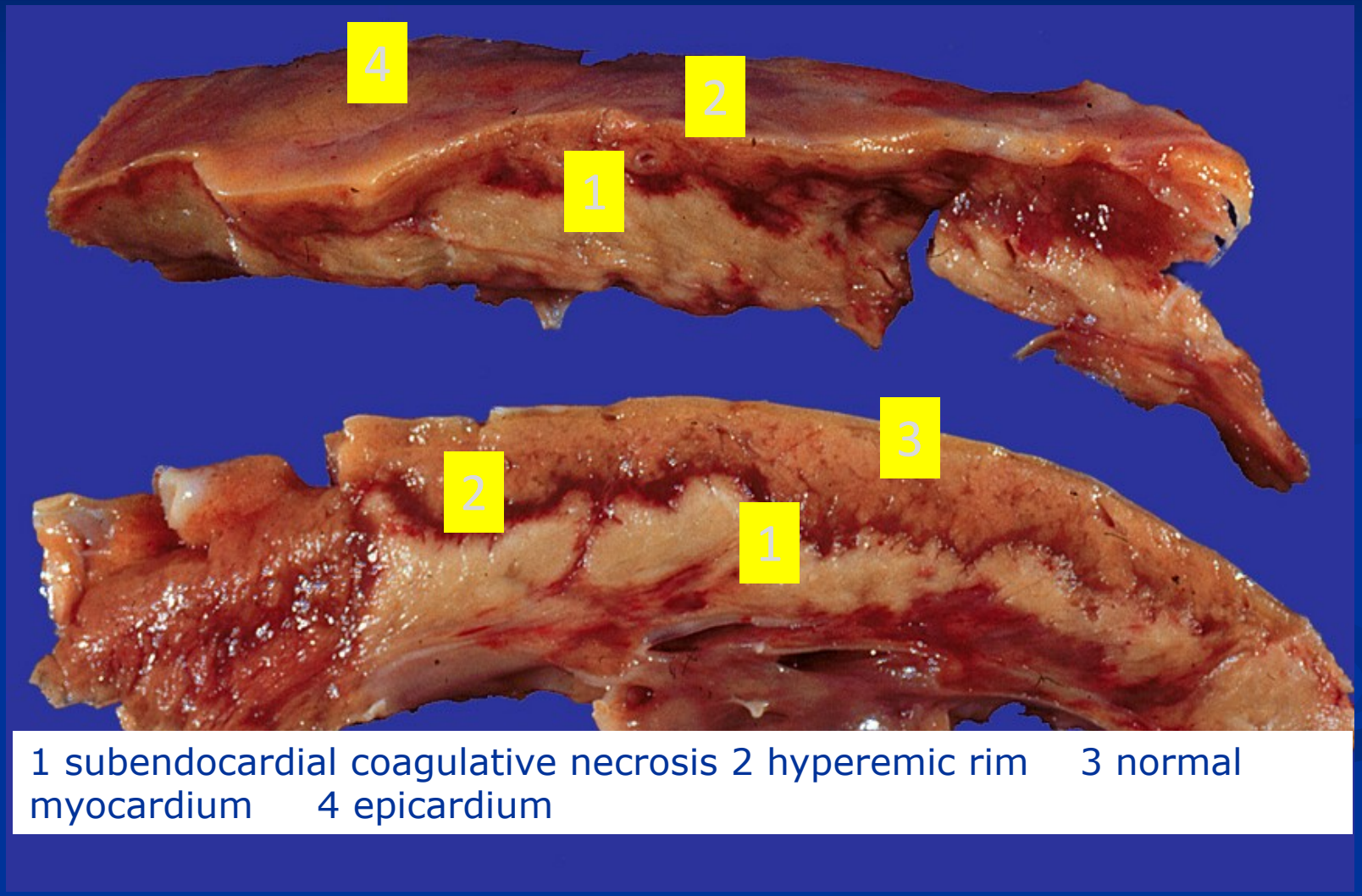
Myocardial infarction + coronary thrombosis



Myocardial infarction



Myocardial infarction



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

Myocardial infarction

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

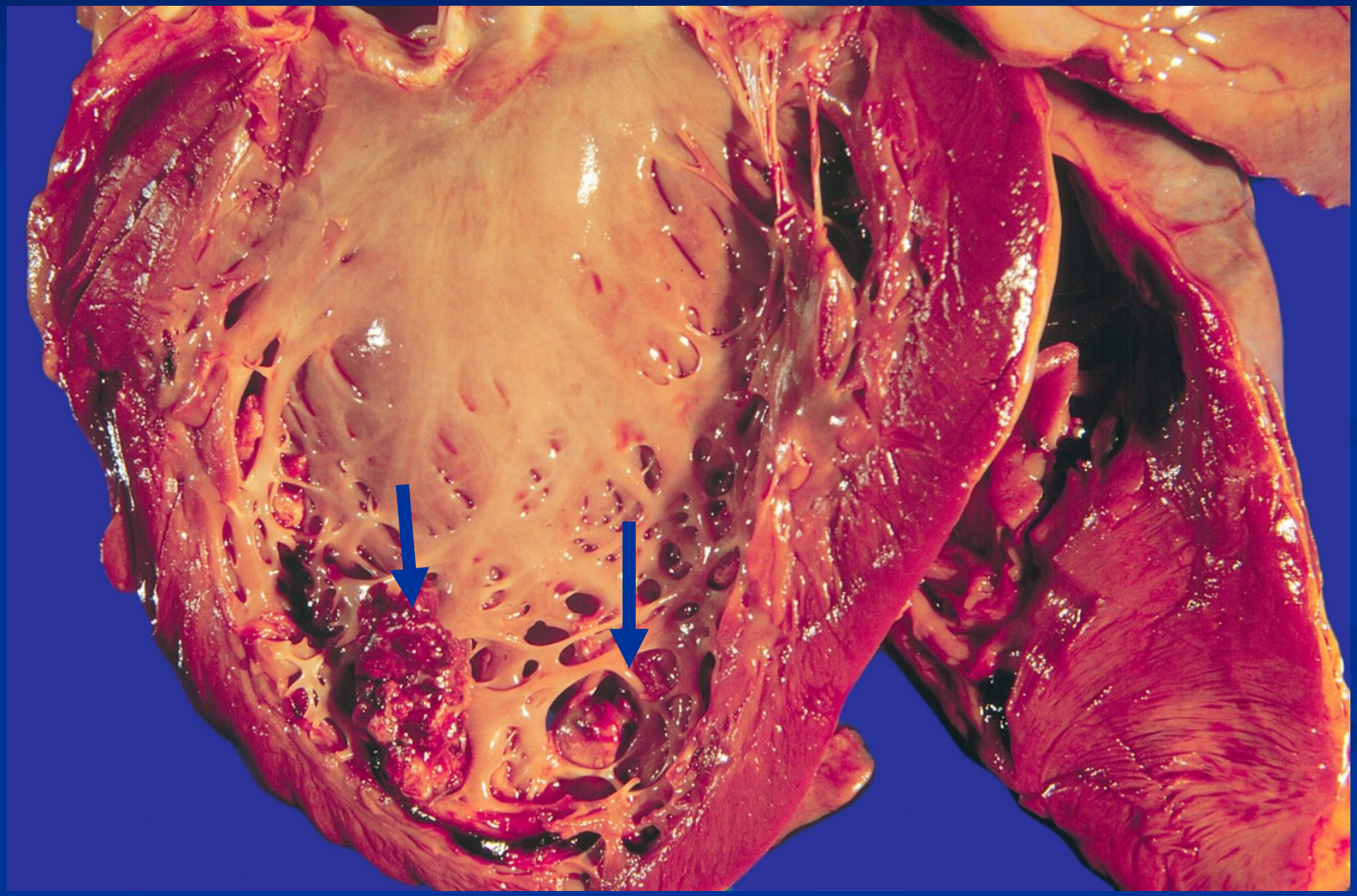
    emergency angioplasty/stenting

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

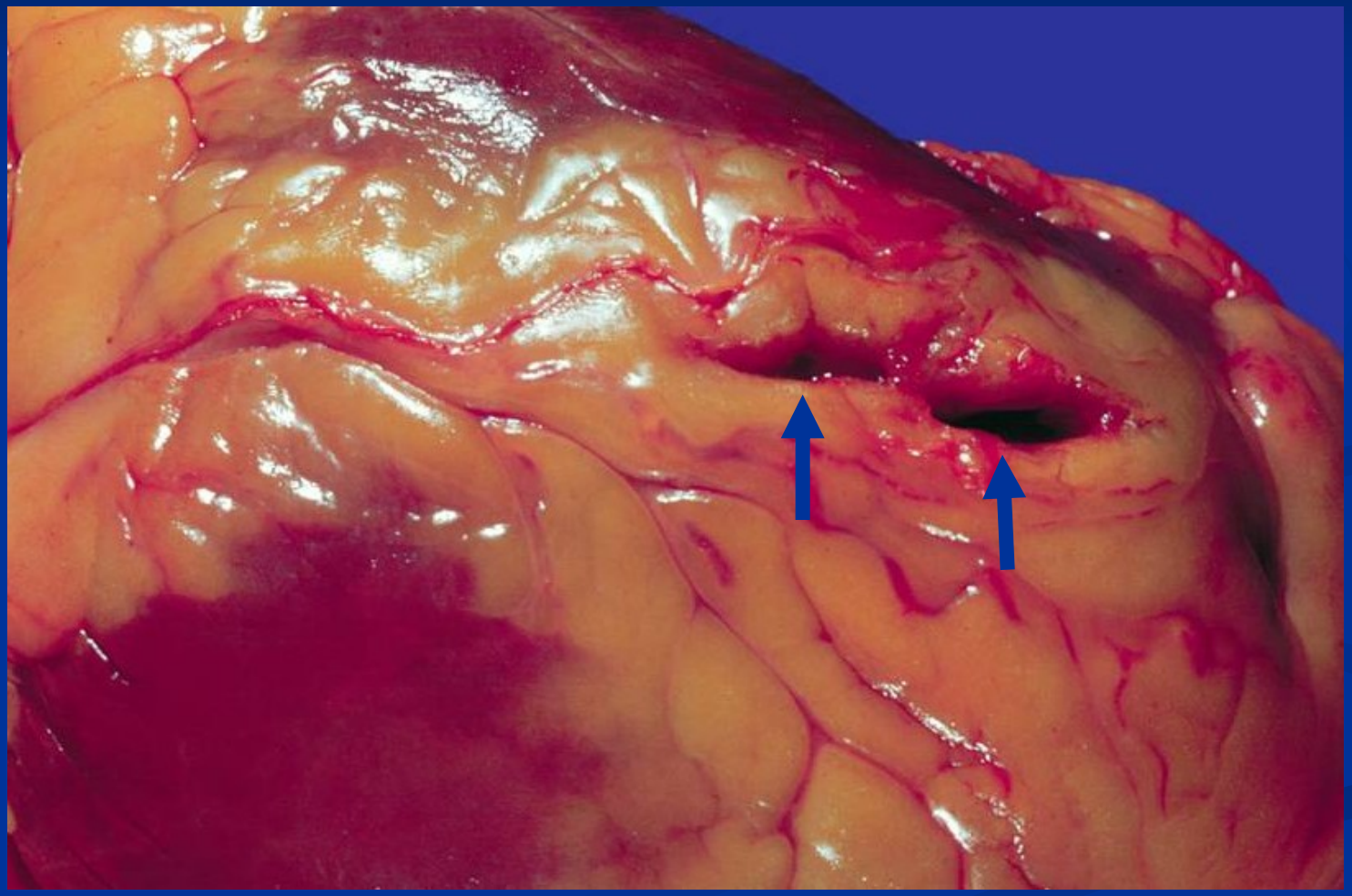
MI complications

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

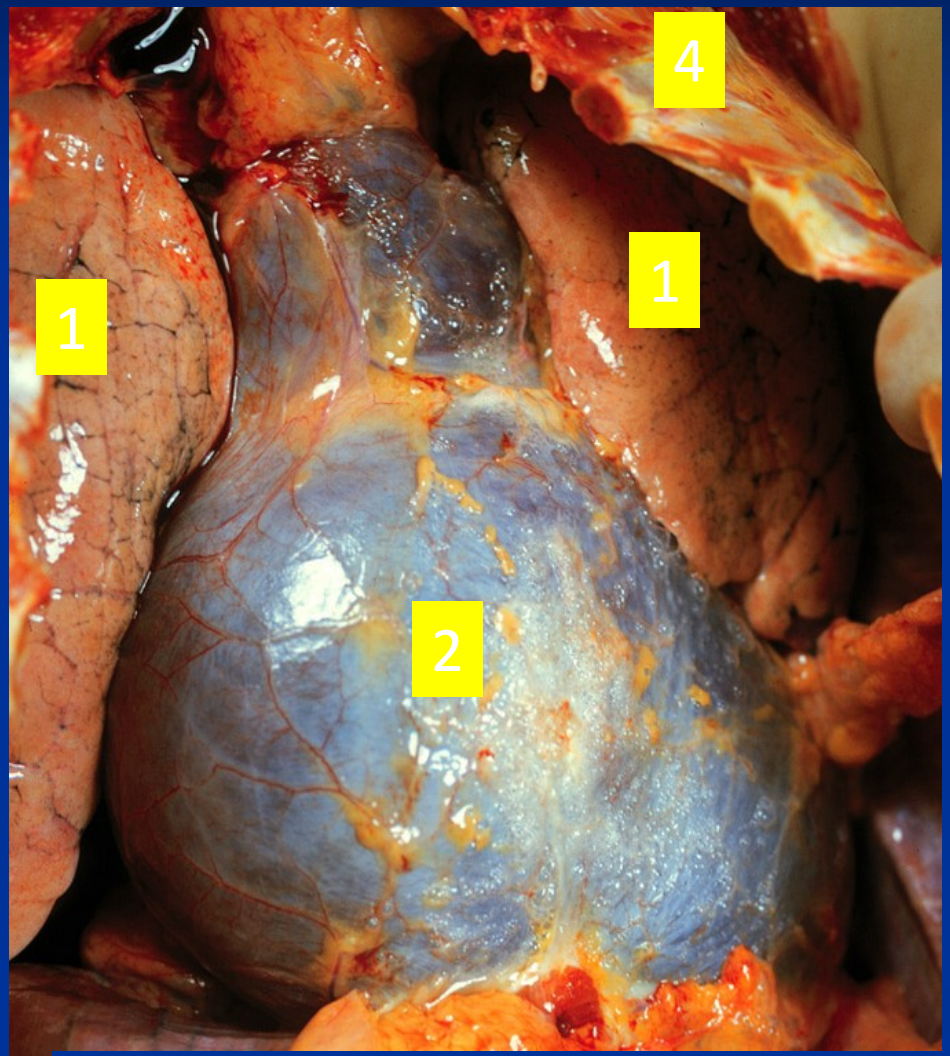
MI – mural thrombosis

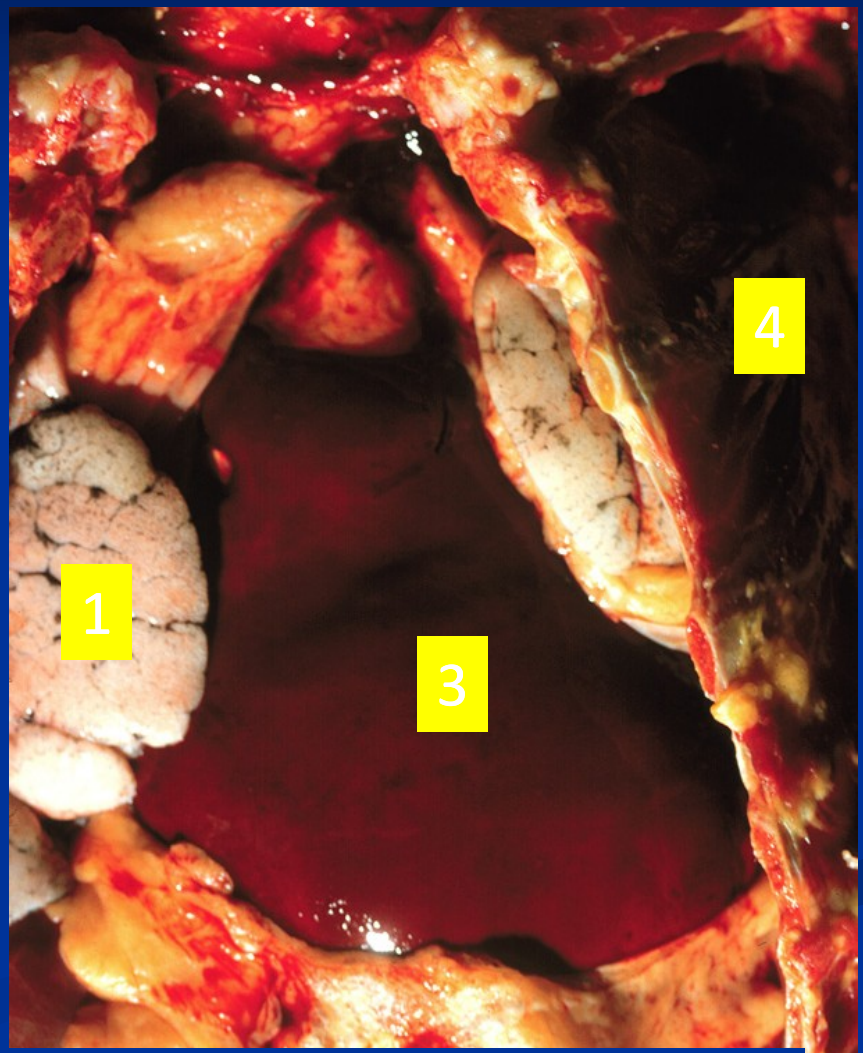


Mi – rupture



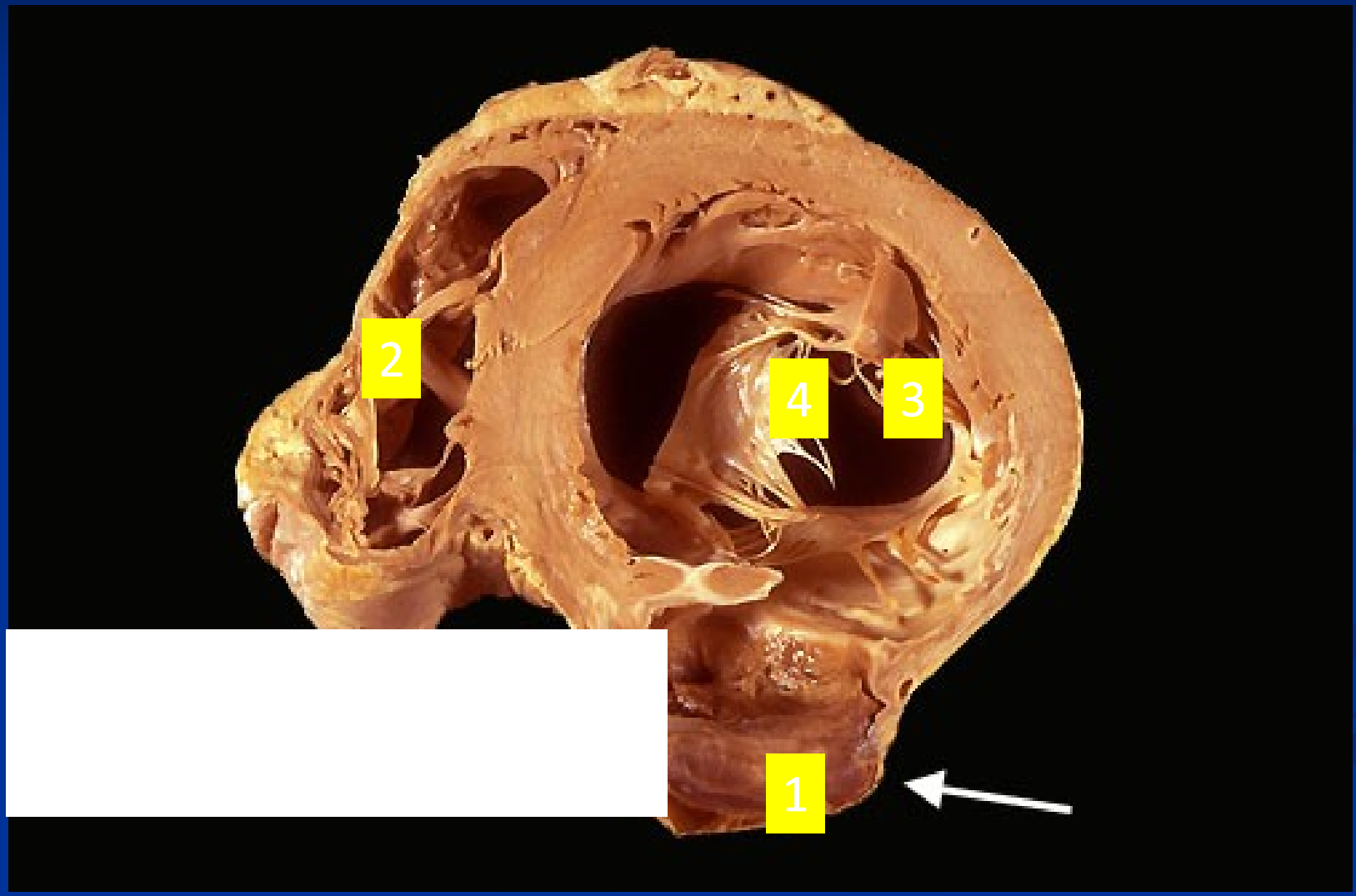
MI – rupture, tamponade





1 lung 2 pericardial sac 3 blood coagulum 4 thoracic wall

MI – LV aneurysm



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

Disperse myofibrosis of the heart

- Repeated multiple microinfarcts („unstable angina pectoris“)
- Repair by scarring
- Disperse scars – small whitish foci in myocardium

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, electrical – channelopathies

Myocarditis

- myocardial inflammatory damage without **ischemia**
- rapidly (days) progressive heart insufficiency

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

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

Cardiomyopathies

= heart disease due to myocardial abnormality, with heart dysfunction

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

Possible cause of sudden death in younger people

- **heterogenous group of disorders:**
  - dilated (DCM) most common

  – dilatation + hypertrophy,↓ LV contraction, possible mural thrombosis; 20–50% genetic (AD); alcoholic, peripartum, myocarditis...

  - restrictive cardiomyopathy: diastolic dysfunction, ↓ of compliance - ↓ filling, myocardial stiffness
  - hypertrophic (HCM)
    – massive LV hypertrophy, 100% genetic, diastolic dysfunction

  - specific CM
    – Duchenne muscle dystrophy, toxic (drugs), endocrine d., metabolic d. (hemochromatosis, amyloidosis, glycogenosis,…)

Arrythmias

- Disturbance of heart rate and/or rhytm
- Pathologic changes in cardiac conductive system
- Ventricular or atrial
- Tachycardia (↑ heart rate) or bradycardia (↓ heart rate)
- Different patterns (ECG)
- Different clinical significance – benign respiratory sinus arrythmia x ventricular fibrillation (fatal without resuscitation)

Valvular heart disease

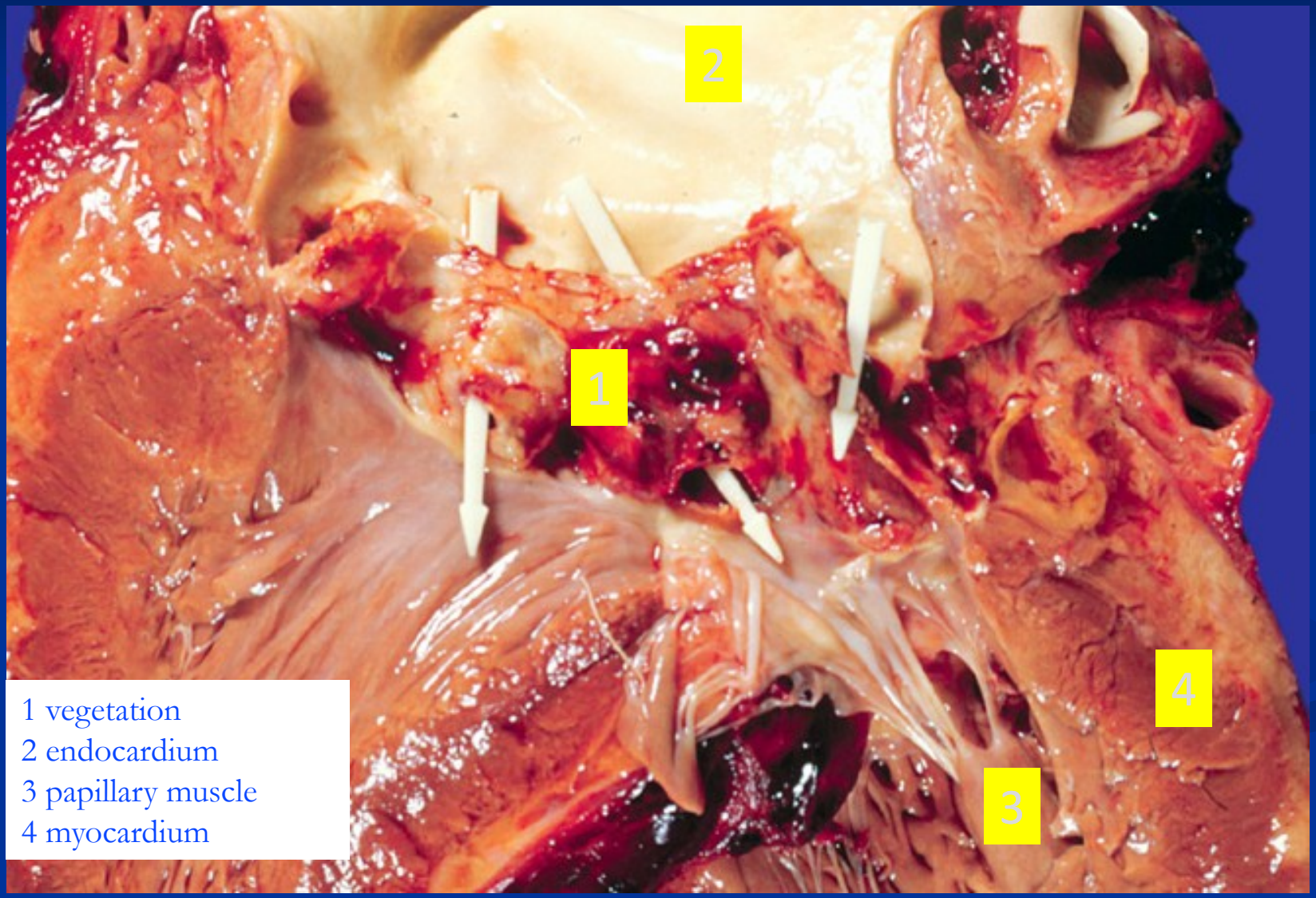
- congenital defects
- endocarditis (rheumatic – immune-mediated, infective, thrombotic non-infective, in SLE)
- degenerative changes (mucoid, calcification, fibrosis in ischemic heart disease)
- dilatation of the ventricles (relative incompetence)

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, stomatologic, surgical procedures, postcatethrization, i.v. drug addicts
  - tooth brushing, chewing (oral flora common source) in immunodeficient patient

- bacteremia - endocardial damage by bacteria - trombosis = infective vegetation

Infective endocarditis – valve destruction



1 vegetation
2 endocardium
3 papillary muscle
4 myocardium

Rheumatic fever, rheumatic heart disease

- acute non-purulent, **immune-mediated** systemic poststreptococcal inflammation (cross-reactive antibodies)

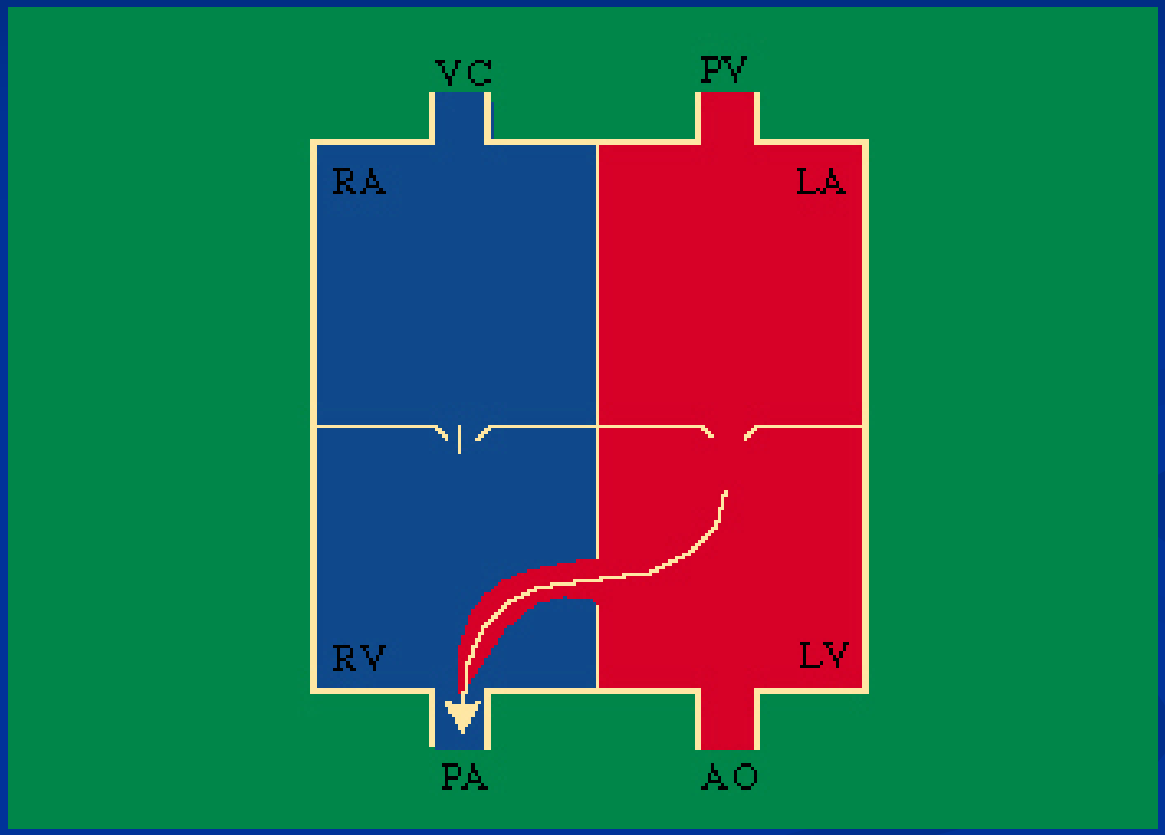
- **acute stage** **PANCARDITIS**
  - acute endokarditis, commonly recurrent

- **chronic stage:**
  - valvular calcification - stenosis + incompetence

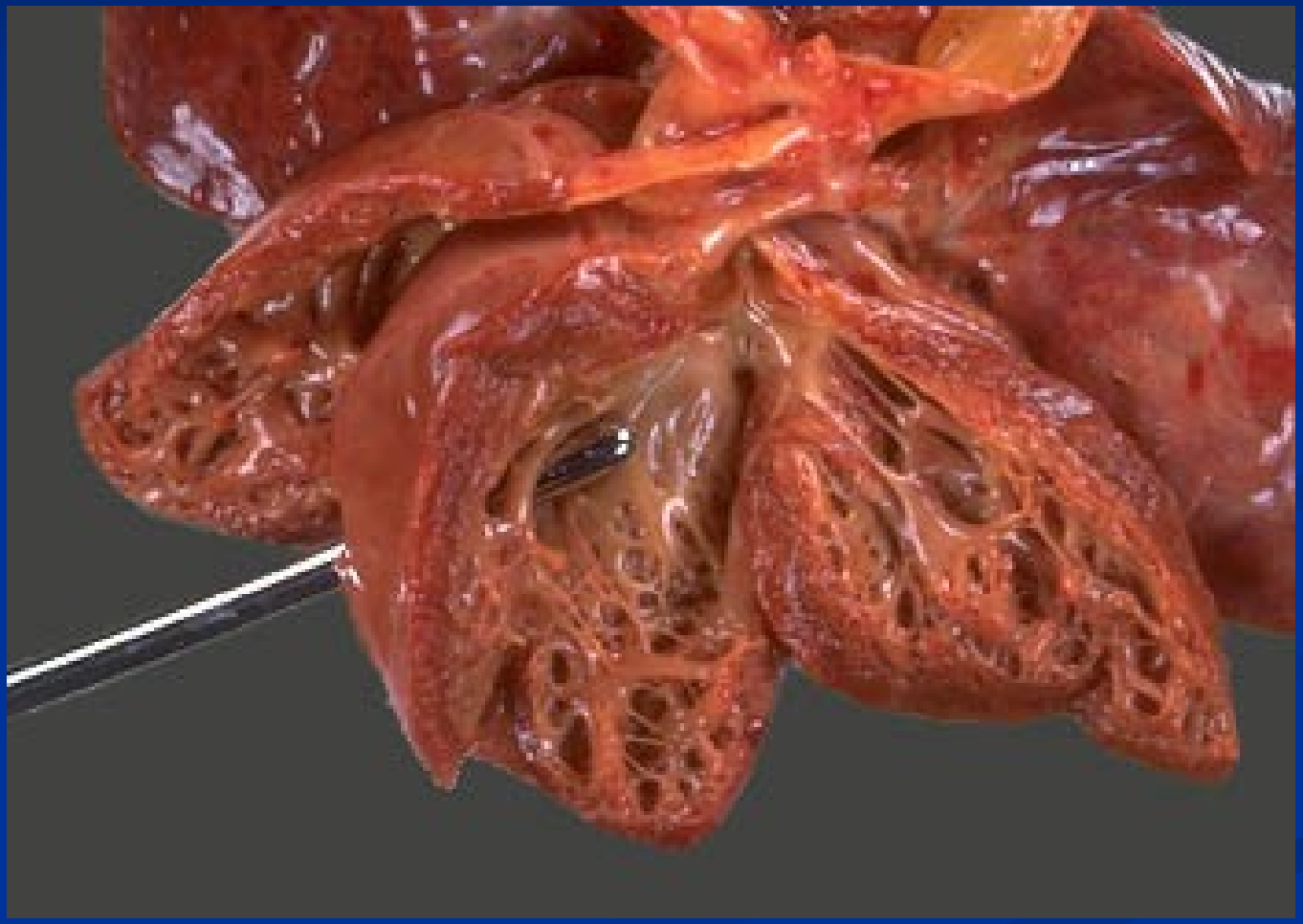
Congenital cardiovascular disease

- Approx. in 1% of newborns
- Usual cause of heart failure in children
- Variable types :
  - Pathological shunts – open communication between spaces which should be closed (septa)
  - Congenital stenoses
  - Complex congenital defect – combination of multiple malformations

Ventricular septal defect



Ventricular septal defect



Pericardial pathology

Pericardial effusion

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

hemopericardium

– wall rupture in MI or aortic root dissection → **fatal cardiac tamponade**

diastolic filling restriction

Pericardial pathology

Inflammatory exudate in pericarditis:

- non-infectious

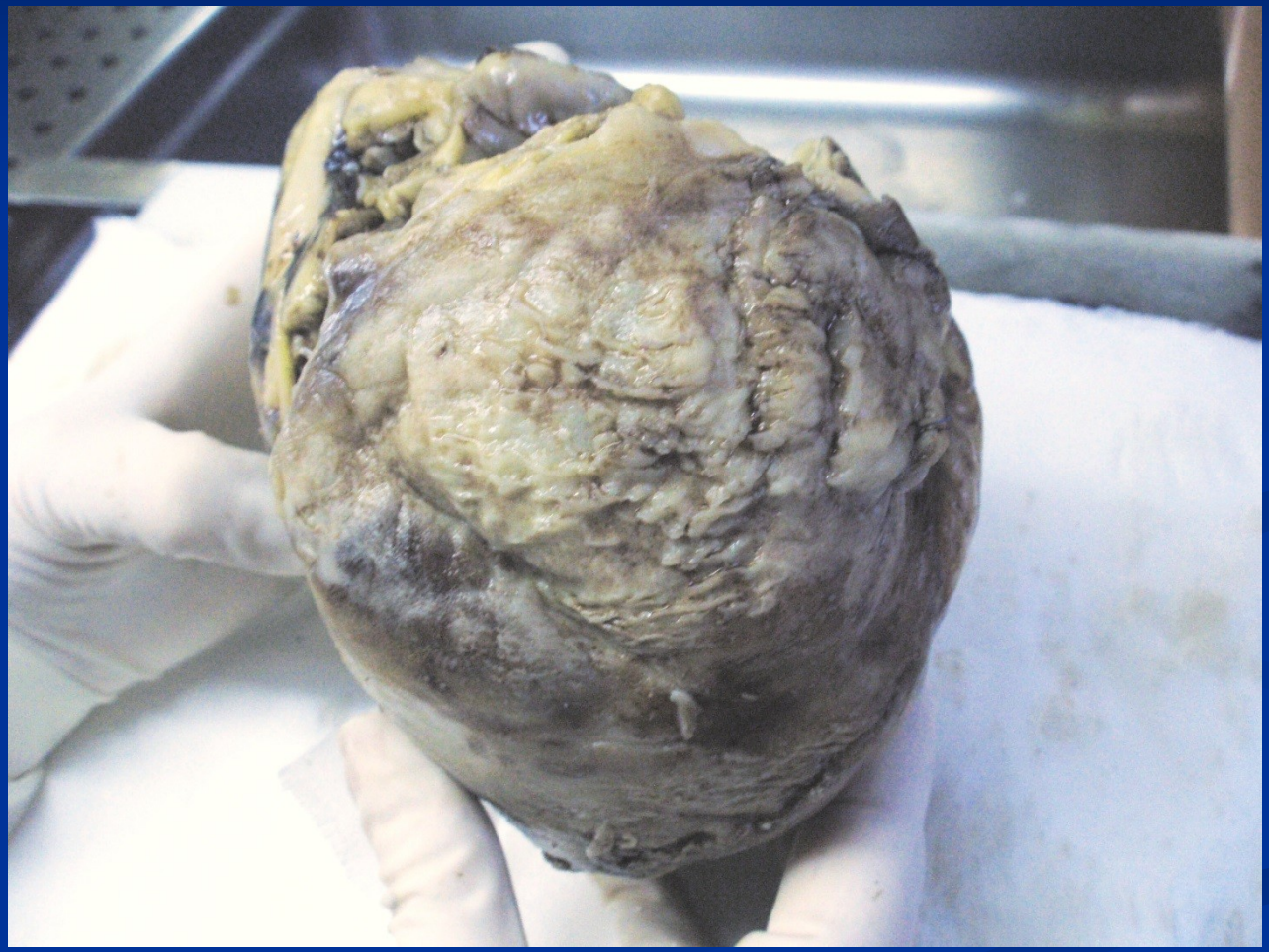
  pericarditis epistenocardiaca (post-MI) uremic, post-operative,

- infectious

  – hematogenous, direct spread, lymphogenous; variable agents

Healing: may be complicated. Fibrinolysis x organisation by granulation tisssue → adhesions, dystrophic calcification.

Acute fibrinous pericarditis



Atherosclerosis

MULTIFACTORIAL COMPLEX DISEASE

- – unknown exact cause, combination of chronic inflamation, fibrosis, lipid deposition

Atherosclerosis

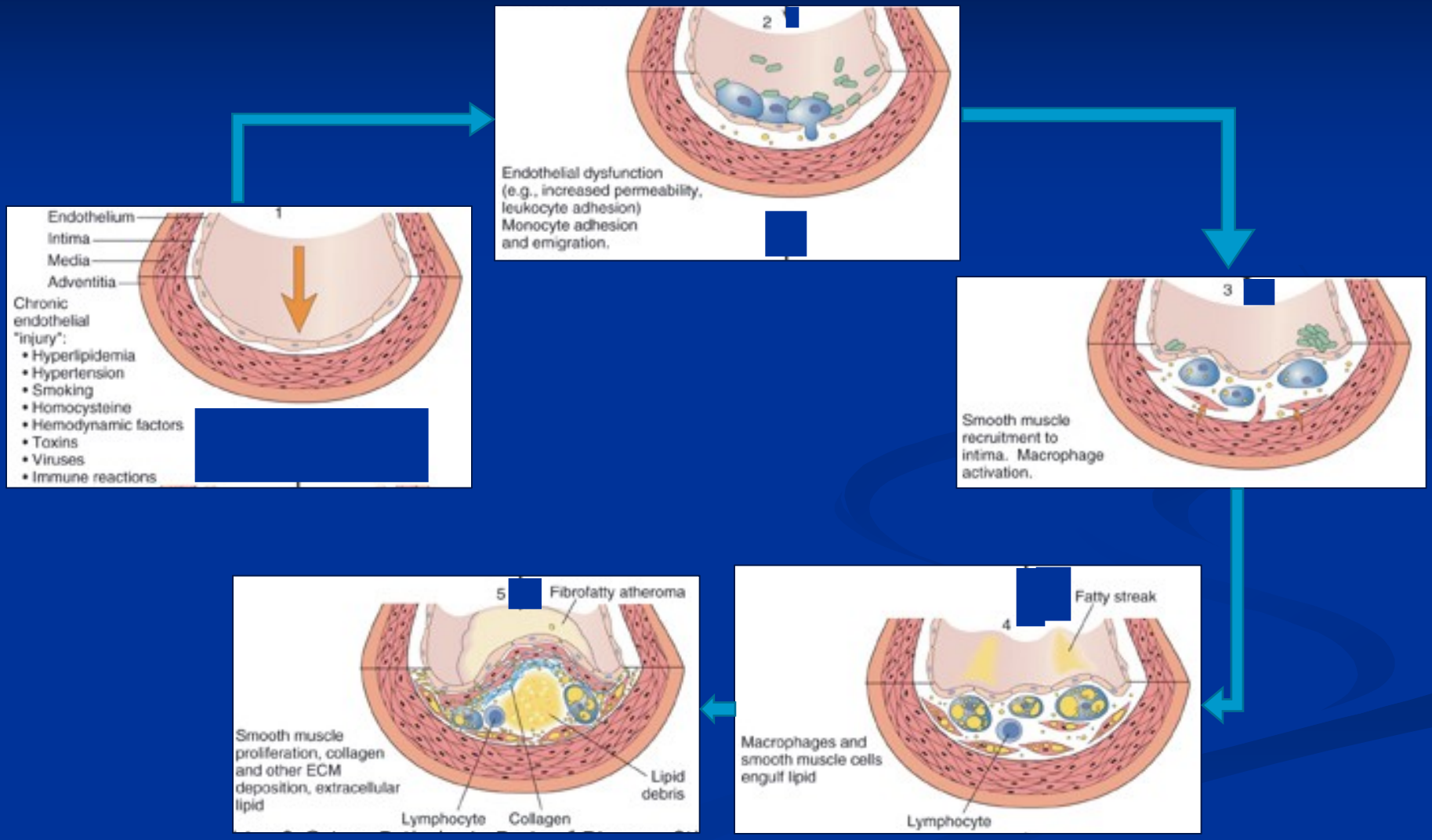
- disease of large and medium-sizes arteries with lipid deposition into intima

active inflammatory process

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

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

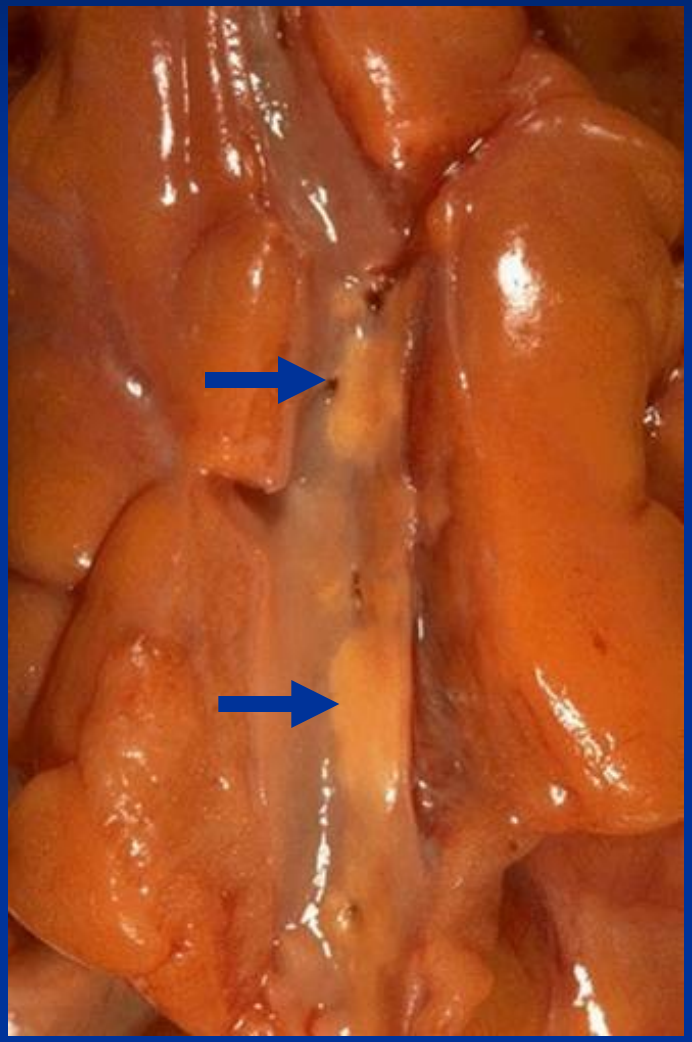
Atherosclerosis - pathogenesis

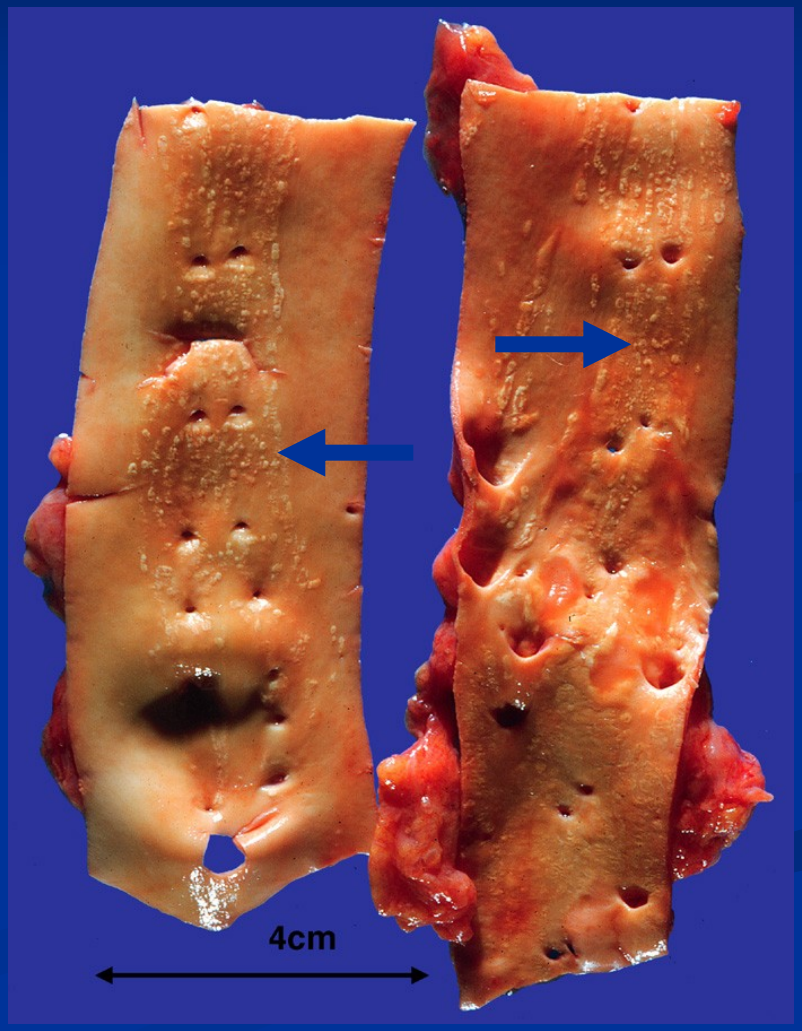


Atherosclerosis stages/changes

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

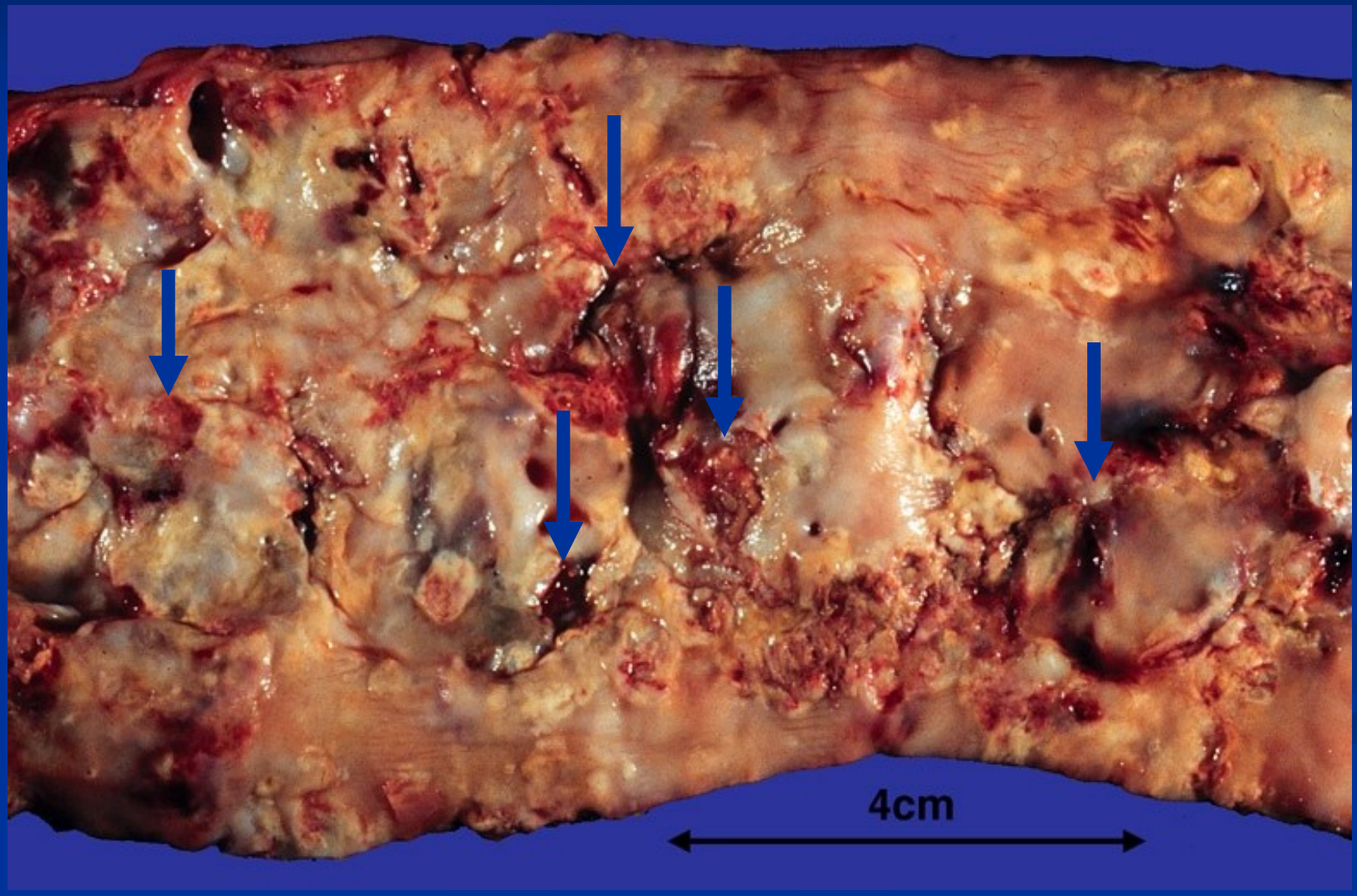
Atherosclerosis – fatty streak





Atherosclerosis –

plaque ulceration, mural thrombosis

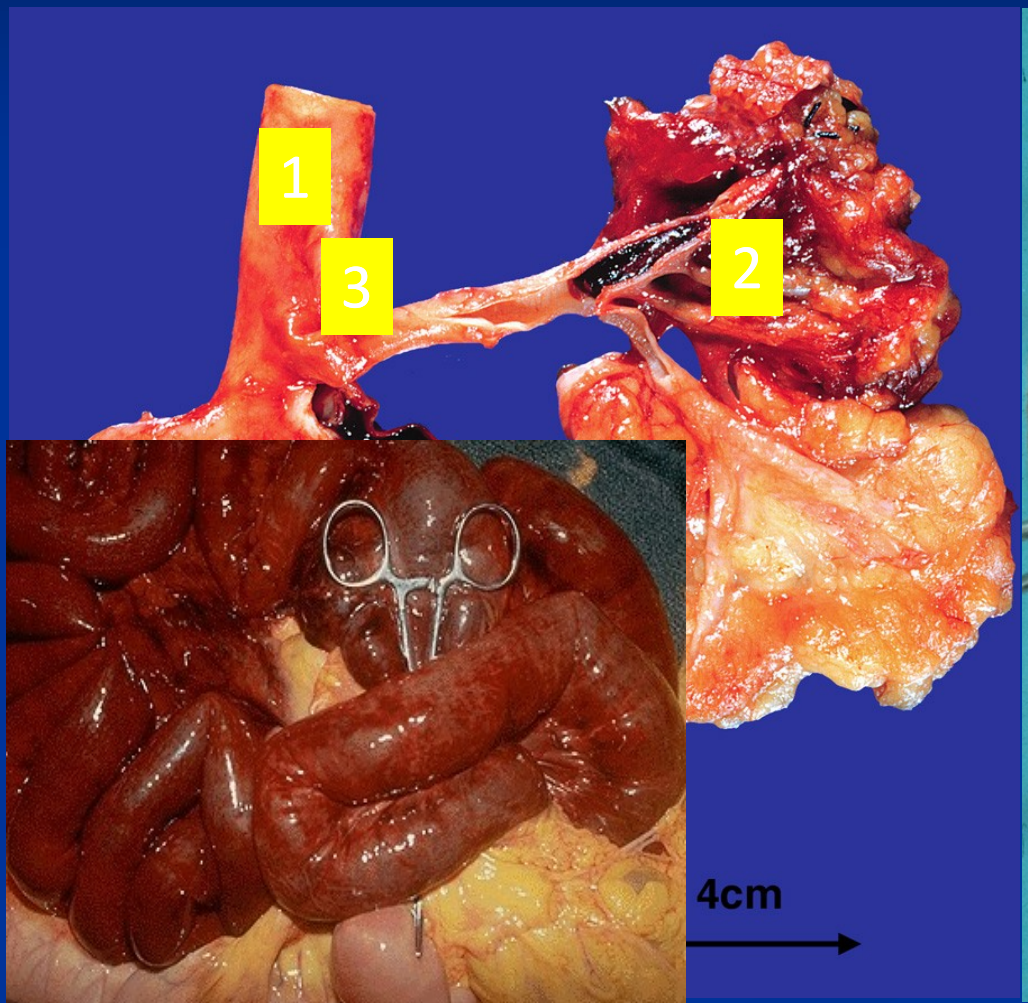


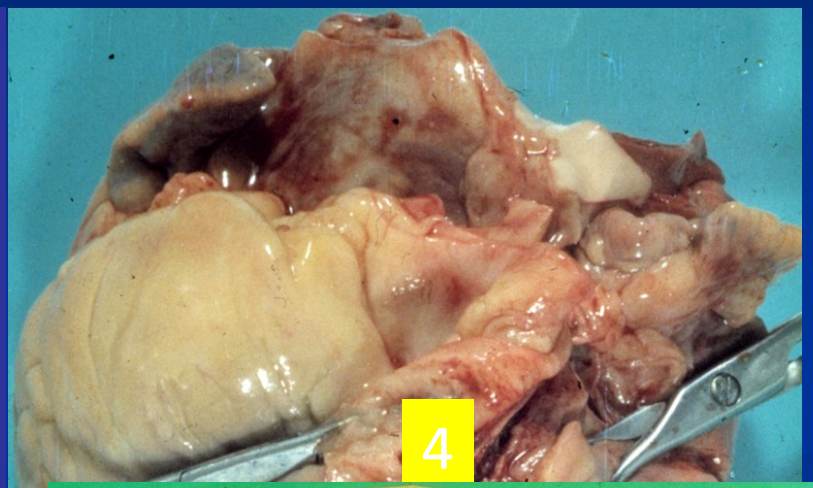
Atherosclerosis

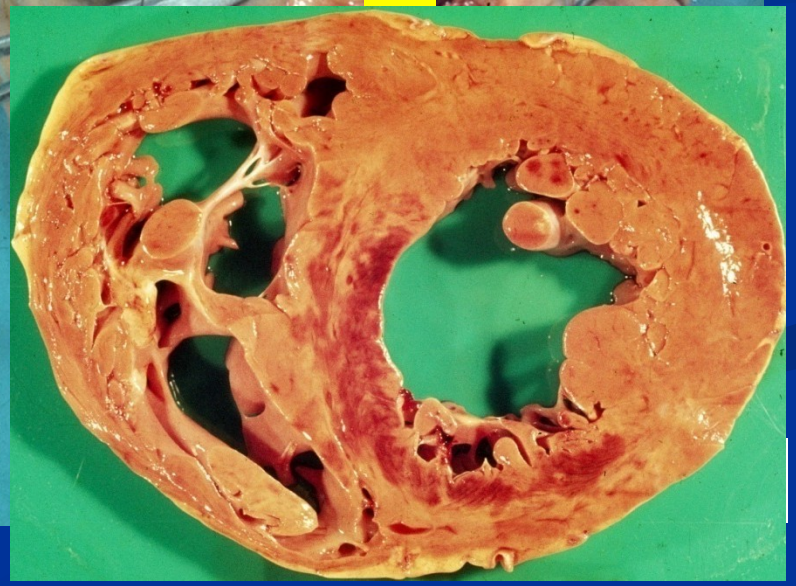
SEQUELS: arterial occlusion in situ

- 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 – complications
thrombosis/thrombembolia



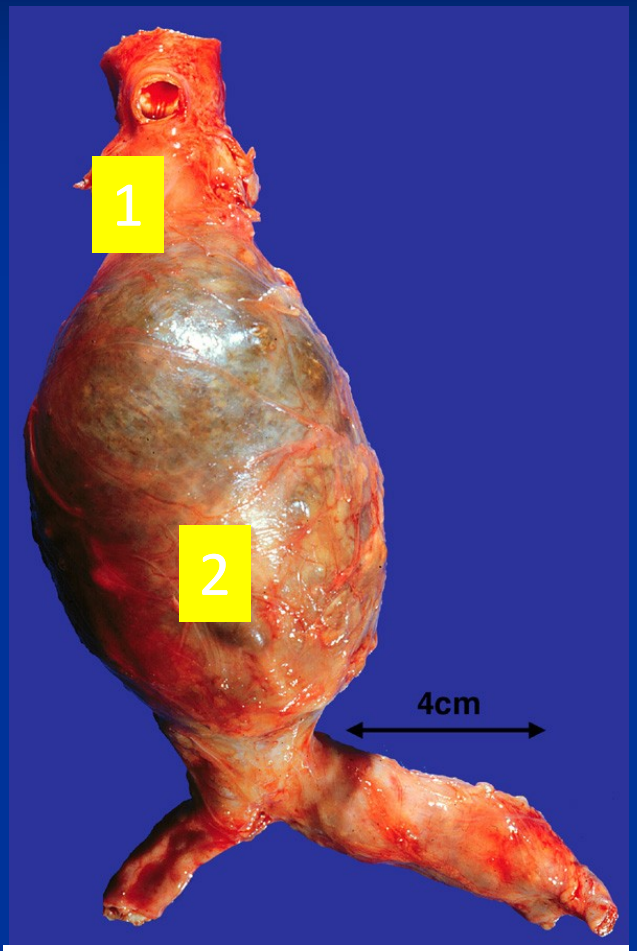




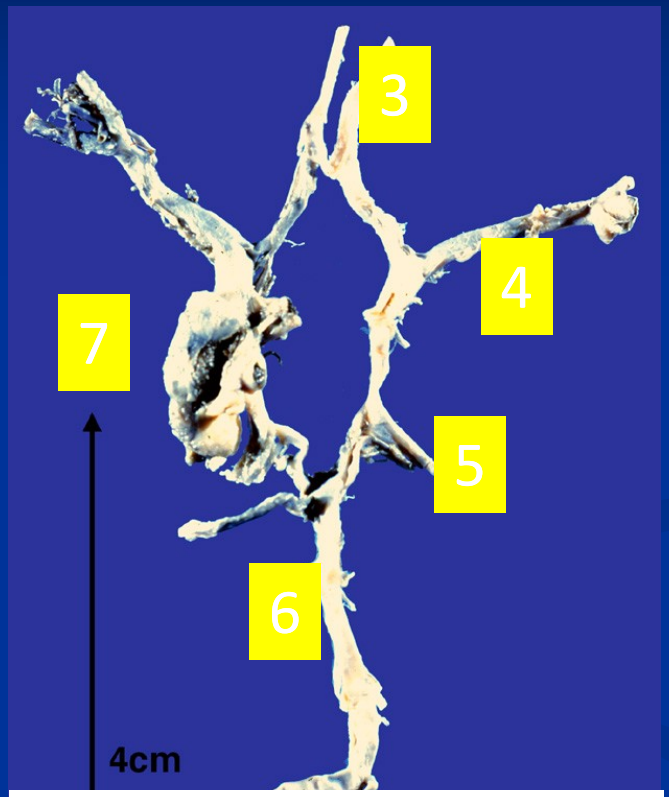
Aneurysm

- 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



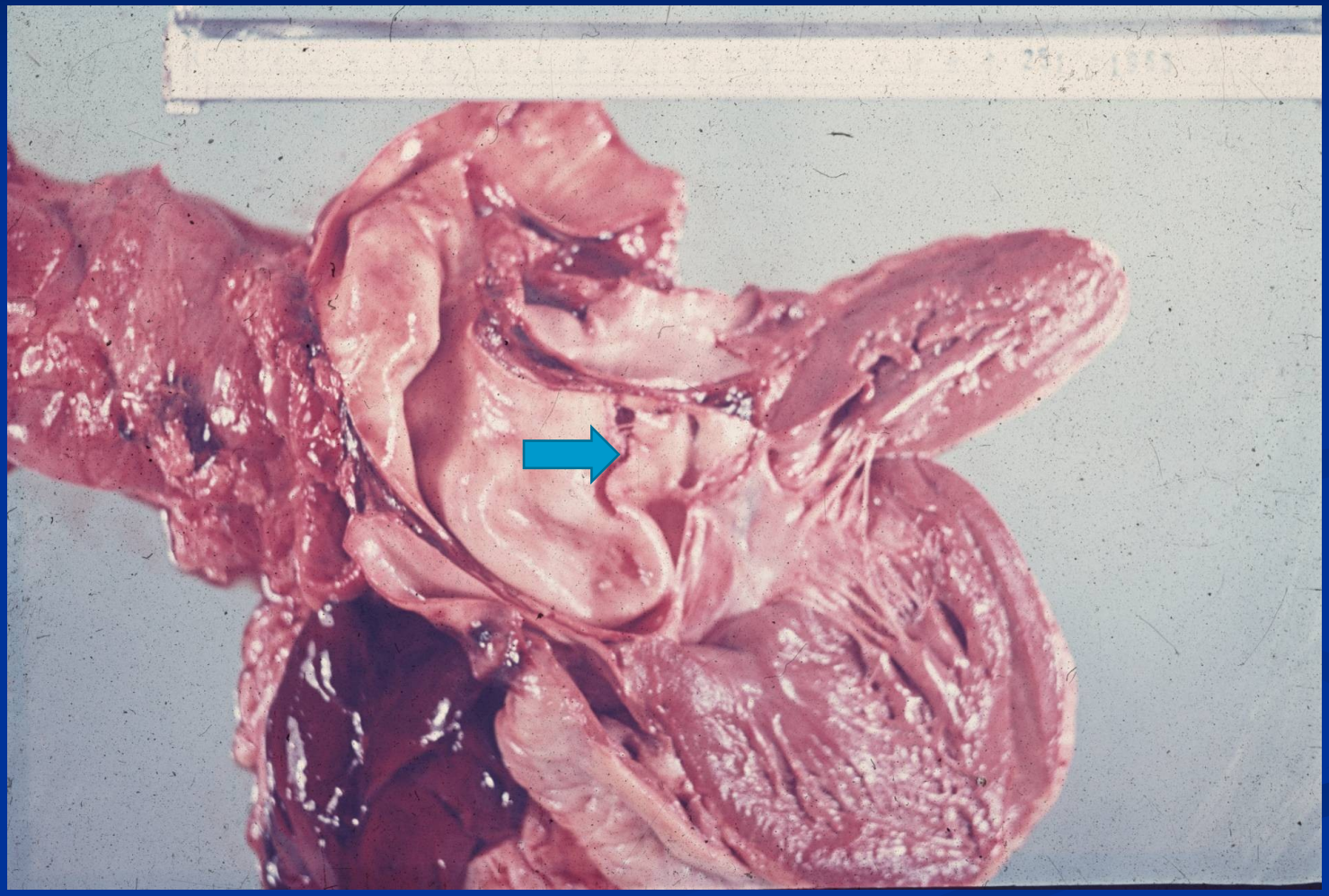
3 a. cerebri anterior
4 a. cerebri media
5 a. cerebri posterior
6 a. basilaris
7 aneurysm

Aortic dissection

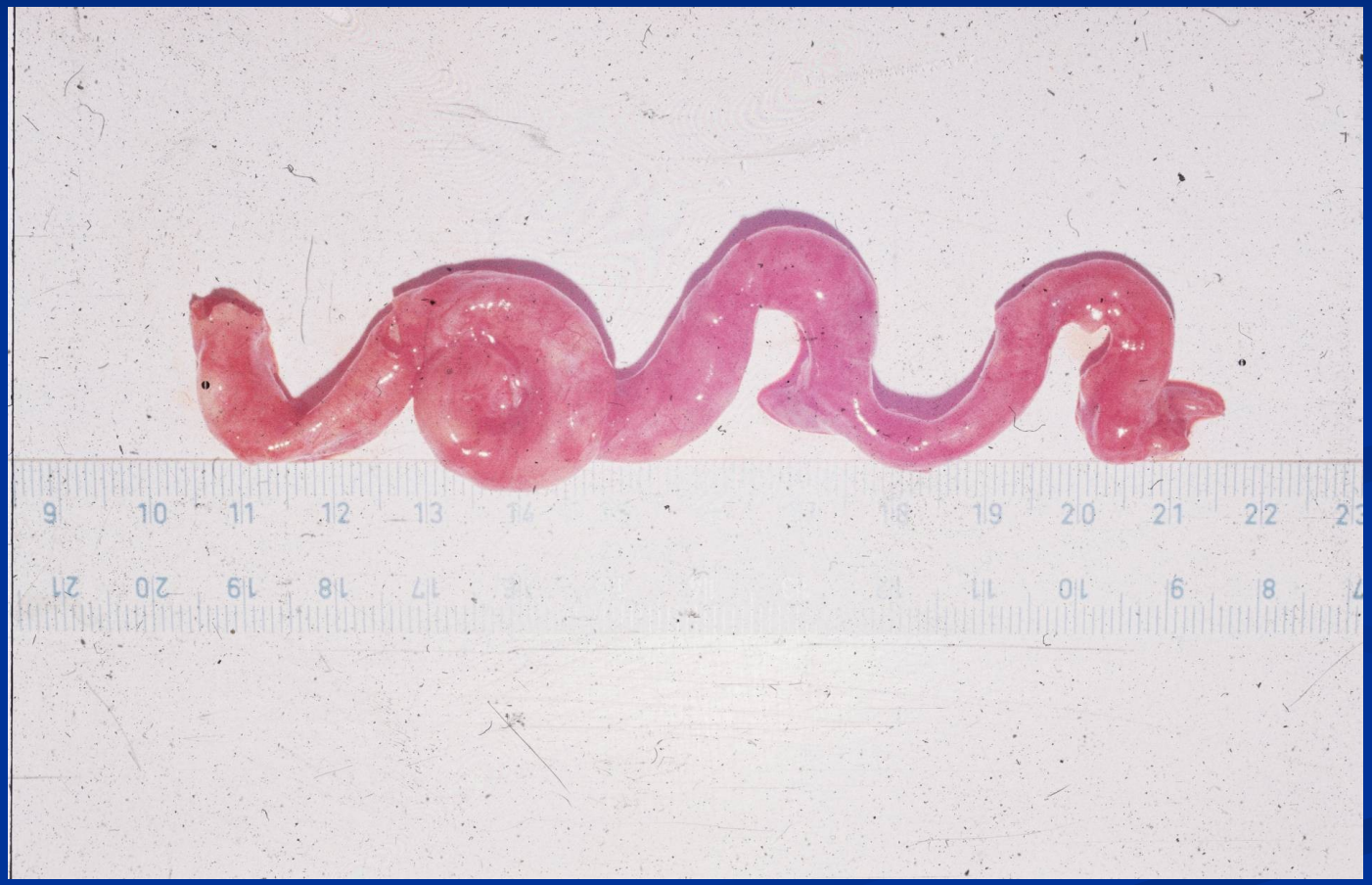
- tear in aortic intima - intramural bleeding through media, false lumen, possible „double-barreled“ aorta
- typical in ascending aorta, 1–8 cm above aortic valve
- ante– and retrograde spread to the aortic root
- common thrombosis in false lumen
- risk of external rupture (→ hemopericardium), progression at the aortic branches (→ variable organ ischemia), heart failure

- predisposition – hypertension, Marfan sy, cystic medial necrosis, …

Dissecting aneurysm



Serpentine aneurysm - a. lienalis



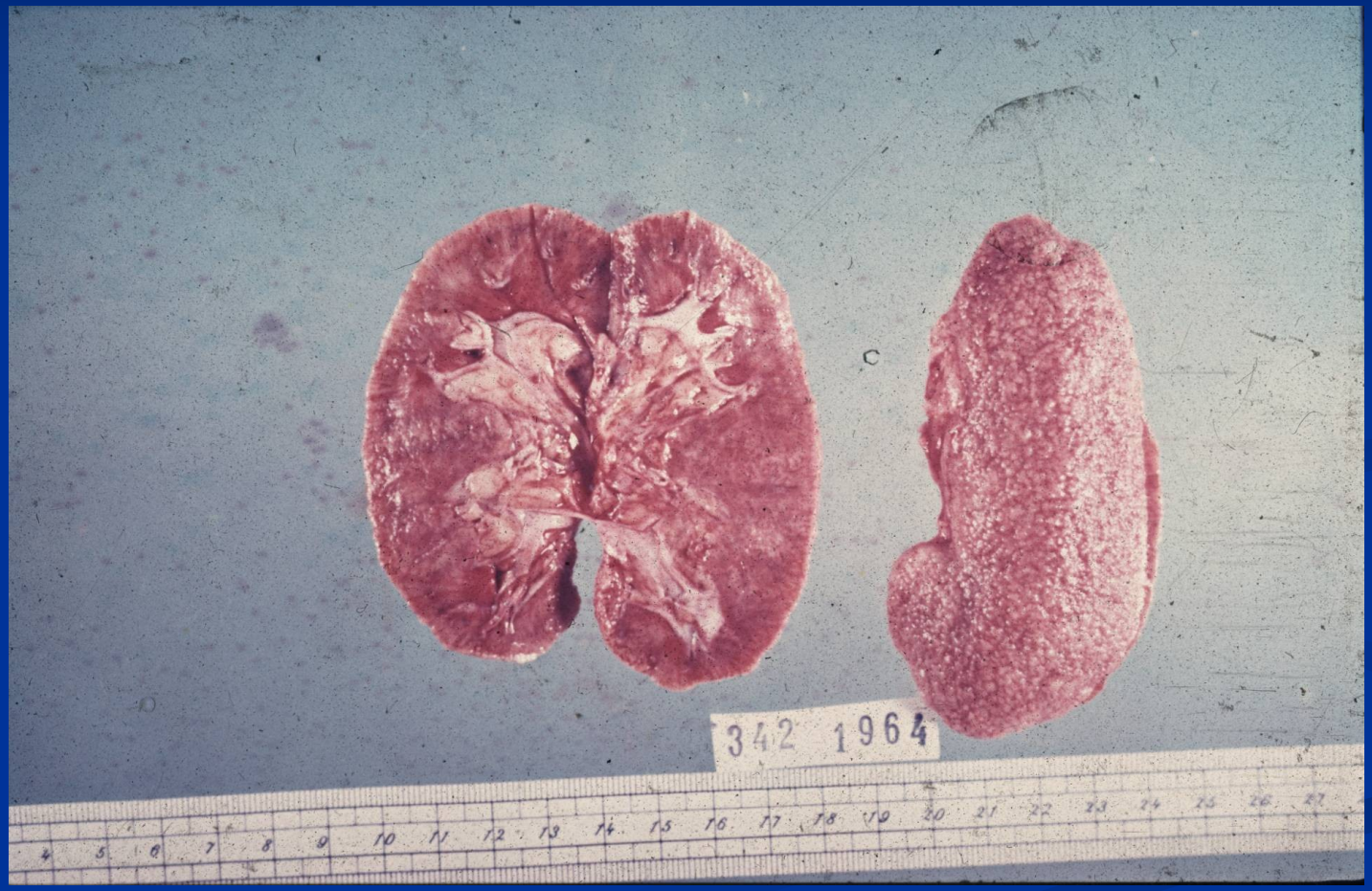
Arteriosclerosis

- in muscular arteries (middle sized)
- smooth muscle hypertrophy
- intimal fibrosis
- collagenisation of elastic membrane
- hyalinisation

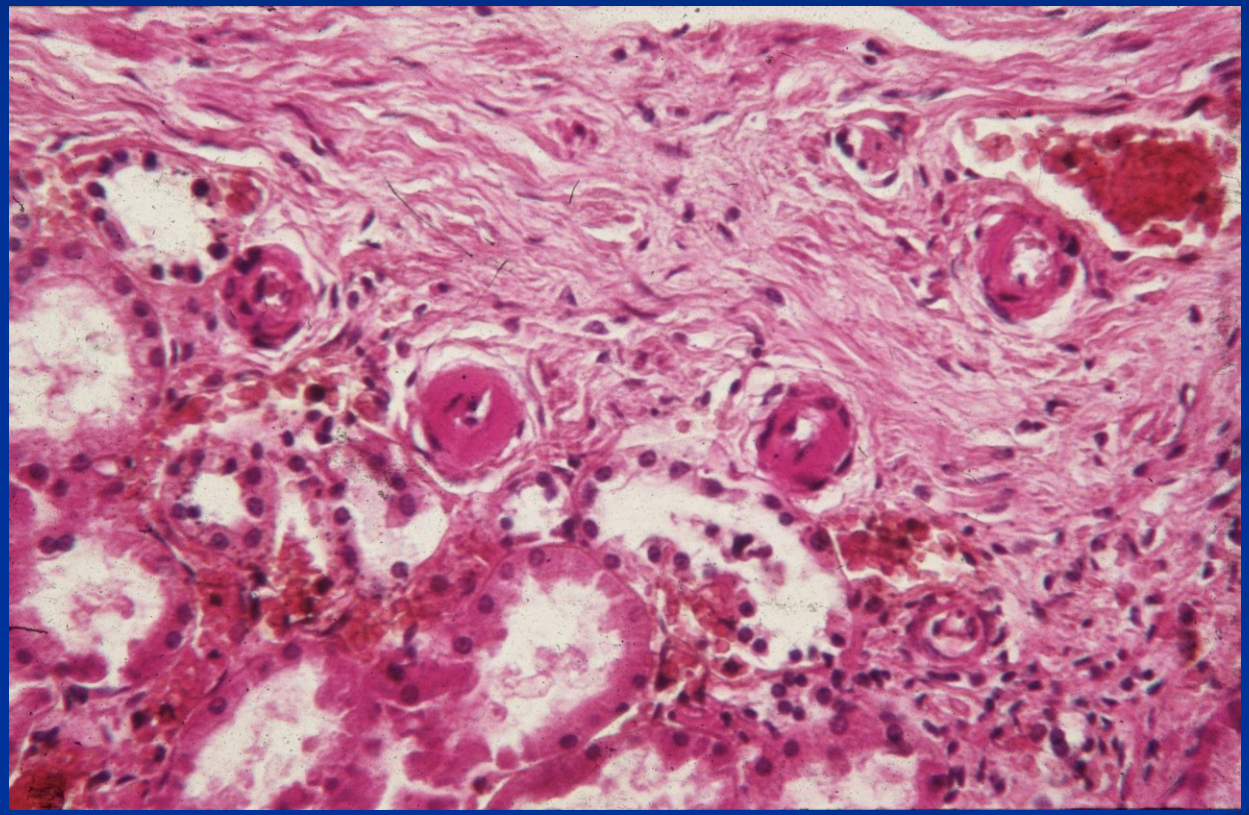
age and/or hypertension related changes

→ nephrosclerosis (→ shrinkage of kidneys, decreased function), cerebral ischemia, …

Renal arteriosclerosis - nephrosclerosis



Renal arteriosclerosis



Systemic hypertension

- Increase in total peripheral vascular resistance
- Primary (essential) hypertension (heritable basis, acquired risk factors – sympathetic overactivity incl. stress, high salt intake, …)
- Secondary hypertension (renal, endocrine hyperfunction, aortic coarctation, drug induced)

Systemic hypertension

- Benign hypertension – gradual (years – decades) progression of organ damage

- Malignant (accelerated) hypertension – severe, often acute damage
  - Renal (→ renal insufficiency)
  - Heart (→ cardiac failure)
  - Brain (→ stroke, usually brain haemorrhage)
  - Retina (→ blurred vision, blindness)

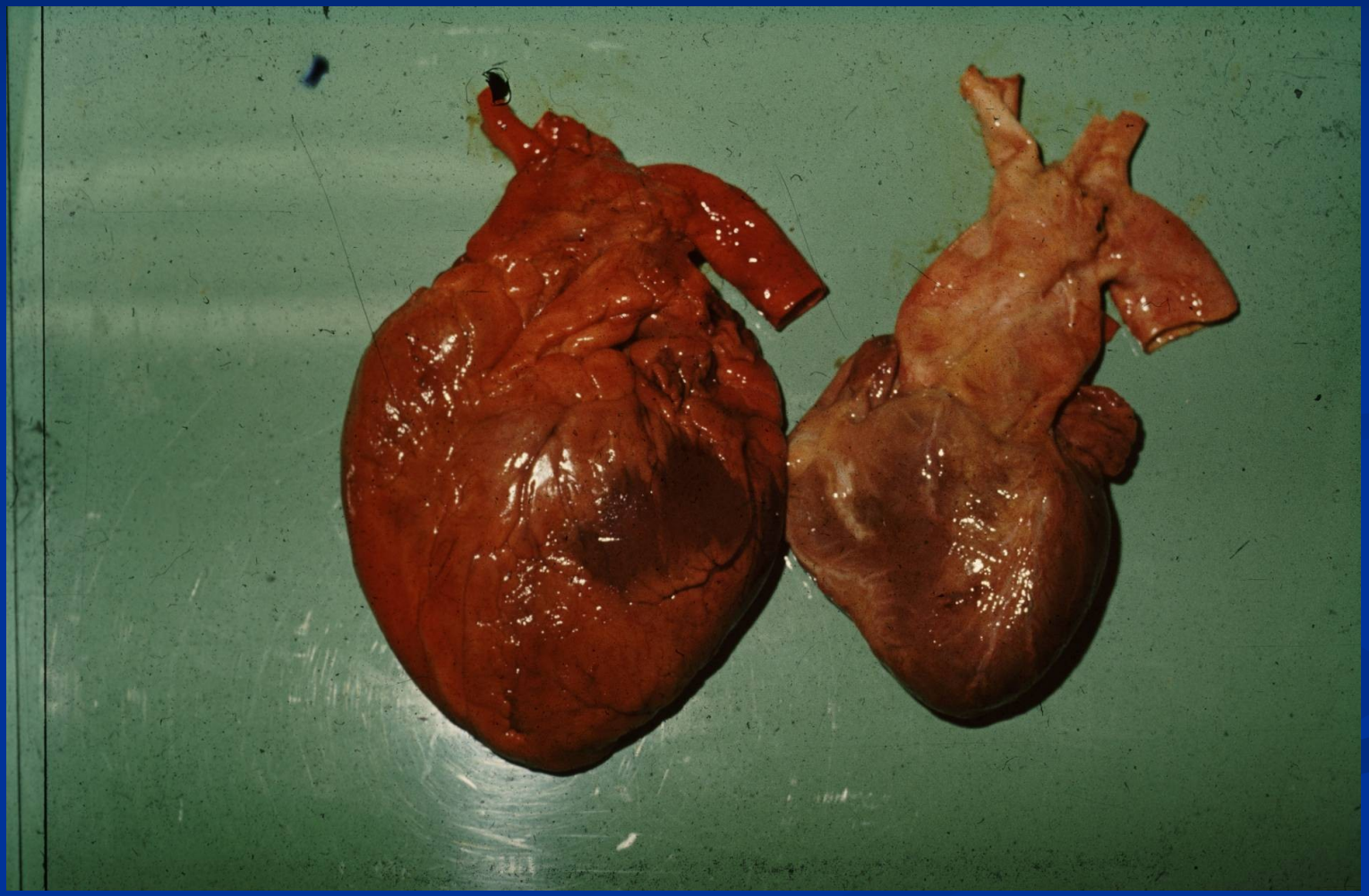
Systemic hypertension and heart

- 90–95% essential , major risk factor for AS, ischemic heart disease
- Adequate control (life style changes, medication) necessary

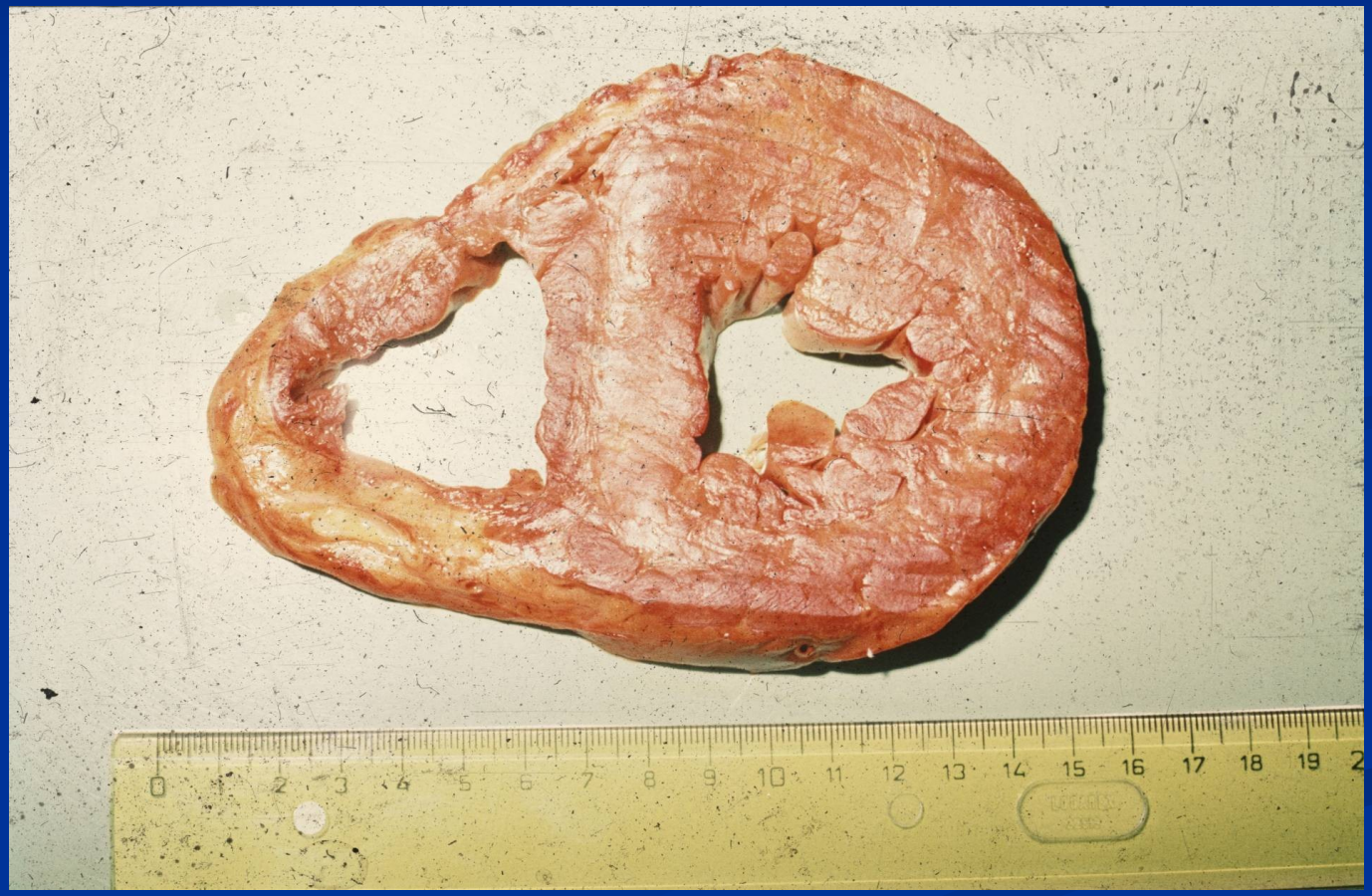
- work overload → LV adaptation to ↑ peripheral resistance = **cor hypertonicum** (concentric LV hypertrophy) → limited compensatory mechanisms → **cor hypertonicum decompensatum** (dilatation of hypertrophic LV)

- → heart insufficiency (+relative coronary incompetence)

Cor hypertonicum

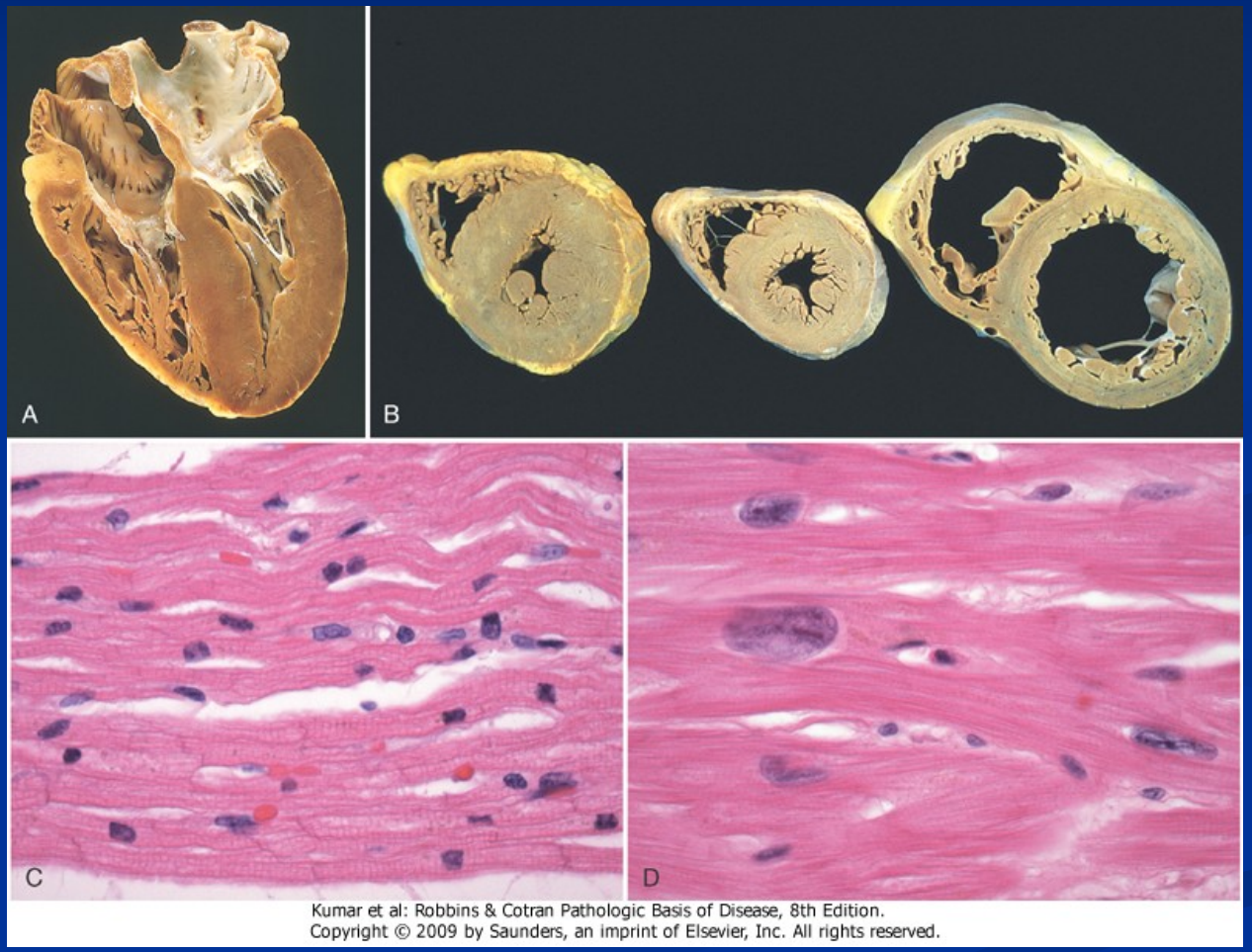


Cor hypertonicum



LV hypertrophy

Cor hypertonicum - evolution



Kumar et al: Robbins & Cotran Pathologic Basis of Disease, 8th Edition.

Orthostatic hypotension

- Postural hypotension – drop in systolic (20 mm Hg) or systolic + diastolic (10 mm Hg) blood pressure with concomitant pulse increase (15 beats/min) on standing from a supine or sitting position
- Acute or chronic
- Common in older adults
- Syncope, fall, organ ischemia (MI, brain transient ischemic attack)
- Autonomic nervous dysfunction
- Variable other causes (blood volume depletion, prolonged imobility, malnutrition, alcoholism, antihypertensive drugs)

Vasculitis

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

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--)

Thrombosis

Main cause of local blood flow disorders

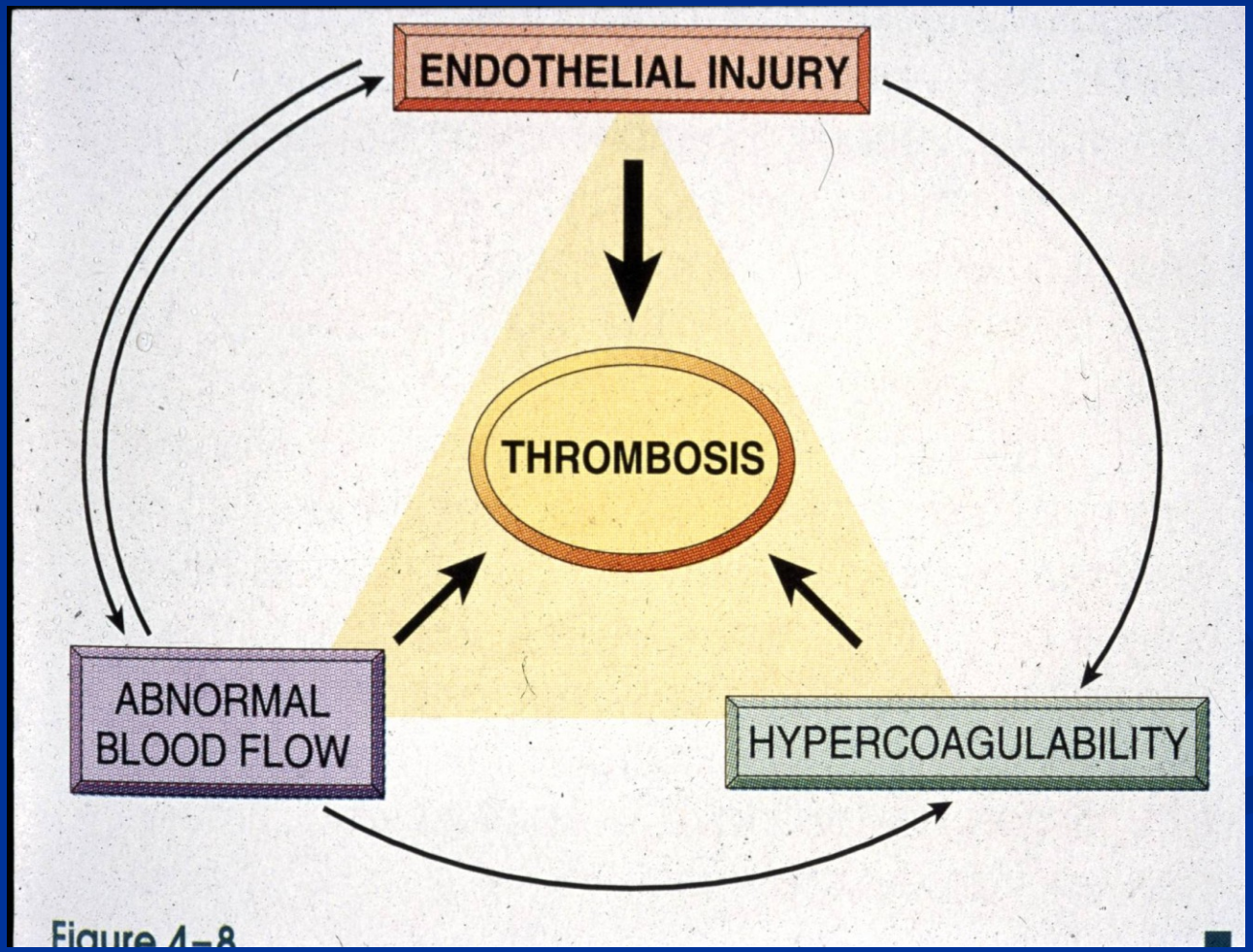
intravital intravascular pathological blood coagulation

trombocytic agregation, fibrinogen transformation → fibrin, thrombus formation

Thrombosis

- *Endothelial injury* most important (trauma, AS, microorganisms, toxins, inflammation) – coagulation factors activation
- *Stagnation:* turbulent non-laminar blood flow, adhesion, common in veins
- *Coagulation disorders:* ↑ coagulability or ↓ fibrinolysis
- a) inborn defects: F V (Leiden) genetic mutation, …
  b) acquired: oral contraceptives, disseminated tumors, DIC

Thrombosis


ENDOTHELIAL INJURY
THROMBOSIS
ABNORMAL BLOOD FLOW
HYPERCOAGULABILITY

Thrombosis

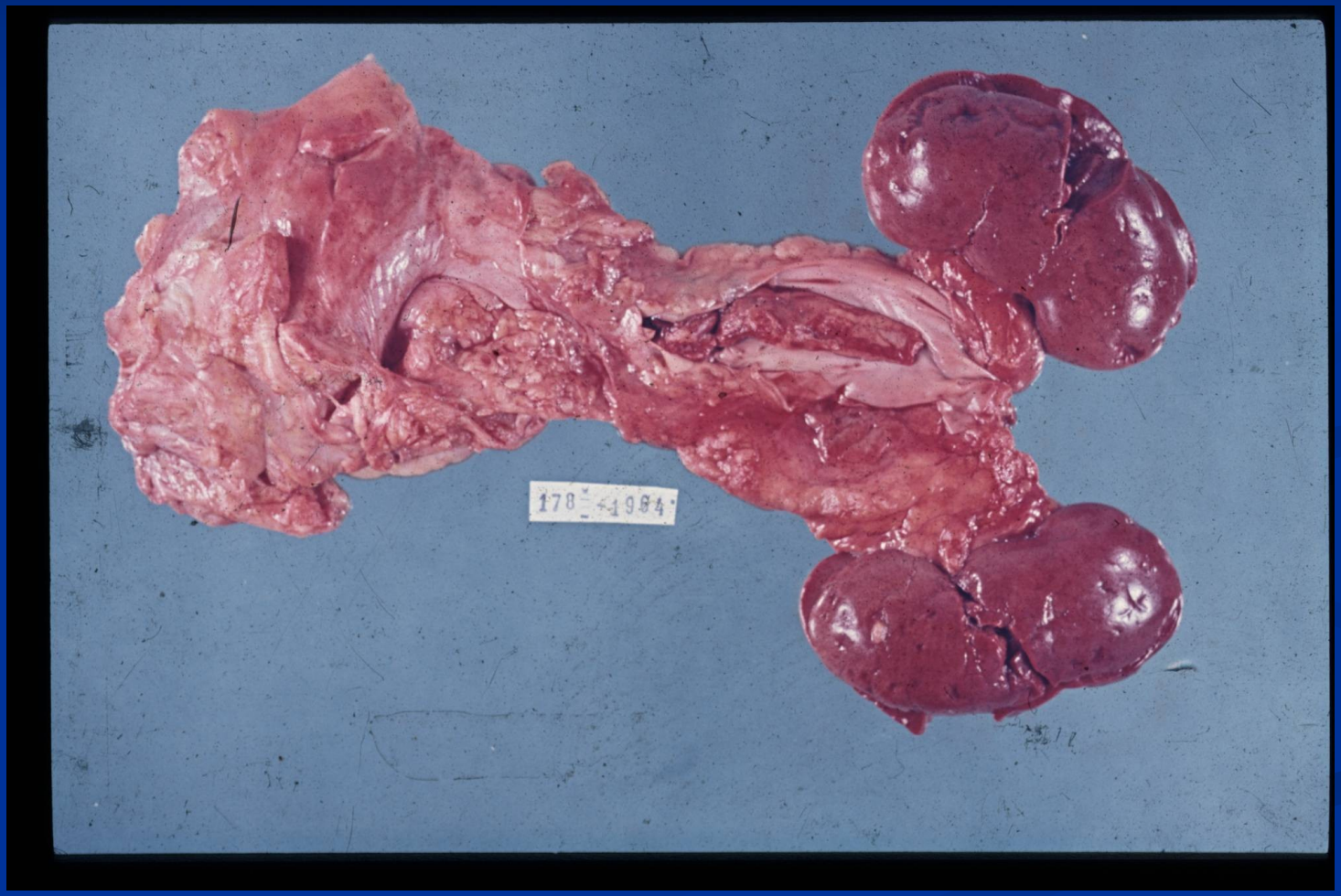
X haematoma – intravital extravascular blood clot

X cruor – postmortal intravascular blood clot

Gross:

- mural (usually heart, arteries)
- obturating (veins)

Venous thrombosis



Fate of thrombi

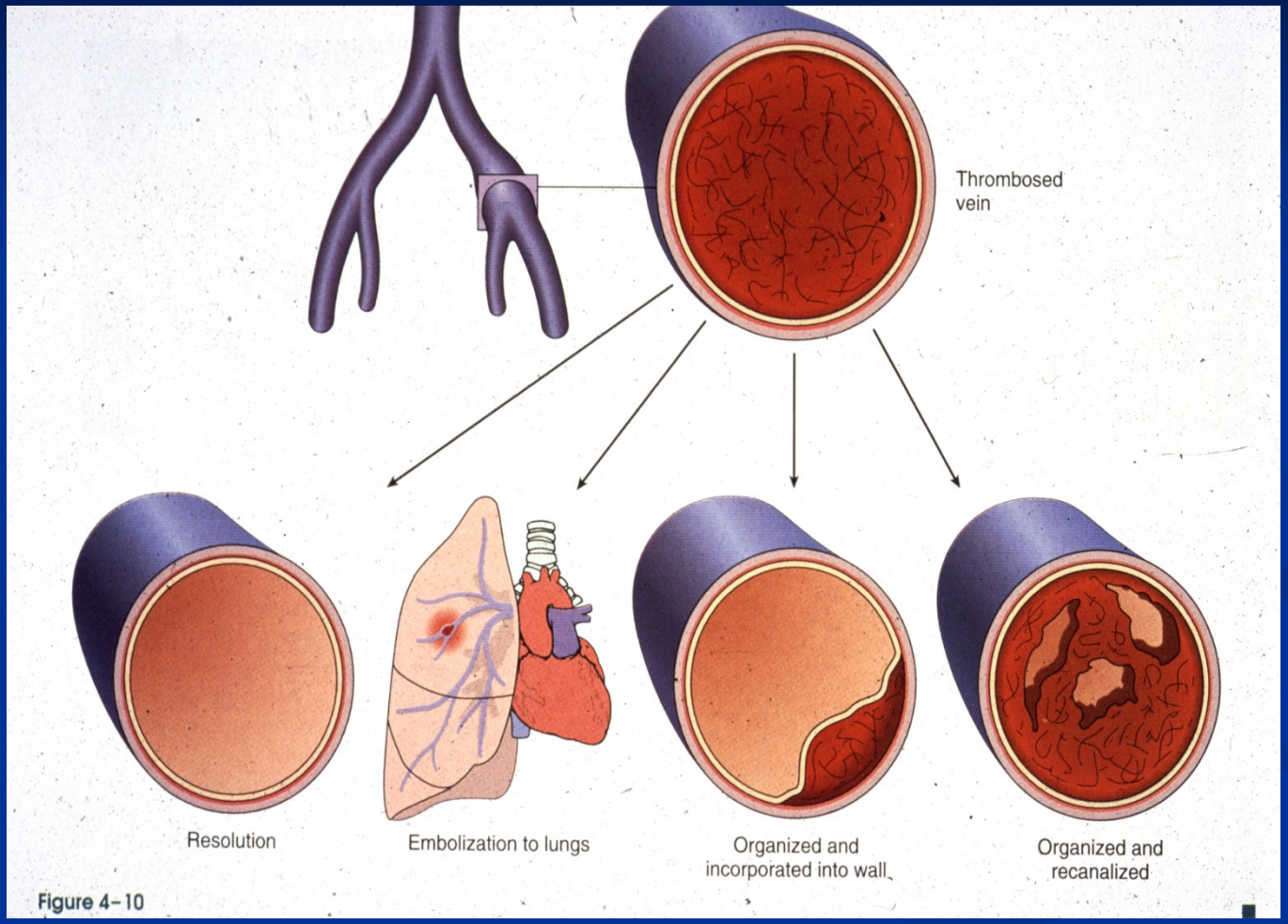


Figure 4–10

Thrombosis

Fate of thrombi

- **Propagation –** growth in the direction of blood flow
- **Organisation** – fixation to the vessel wall, reparation through fibroproductive inflammation, retraction, recanalisation
- **dissolution** - resolution
- **embolism**

Thrombus organisation

- reactive changes in the thrombus, growth of young immature fibrotic (granulation tissue), later collagen production

- Thrombus retraction, resorption by granulation tissue, recanalisation, surface covered by endothelium

Disseminated intravascular coagulation

- DIC
- Acquired coagulopathy, 40% mortality
- Widespread endothelial damage or release of tissue thromboplastin (part of cell membranes) into circulation in:
  - major tissue trauma
  - obstetric complication: protracted labor, placental abruption (amniotic fluid embolism)
  - infection (menigococcal sepsis)
  - neoplasms, liver disease, etc.

DIC

- 1. phase: diffuse activation of coagulation in microcirculation (brain, lungs, liver, kidney, heart) – ischaemia, organ failure

- 2. phase: coagulation factors consumption, activation of fibrinolysis → hemorrhagic diathesis

  hemolytic anemia

Deep vein thrombosis (DVT) and pulmonary embolism (PE)

- Occlusion of a vein by a thrombus with secondary inflammatory reaction in the wall of the vein (thrombophlebitis)
- Risk of thrombus detachment, lung thrombembolism
- Venous thrombembolism – VTE, anticoagulation therapy necessary
- Significant health problem
- Due to:
  - Immobility (venous stasis)
  - Trauma (venous damage)
  - Lifestyle – smoking, DM, obesity, hormonal status (oral contraceptives, …)
  - Hypercoagulation incl. genetic factors

Varicose veins

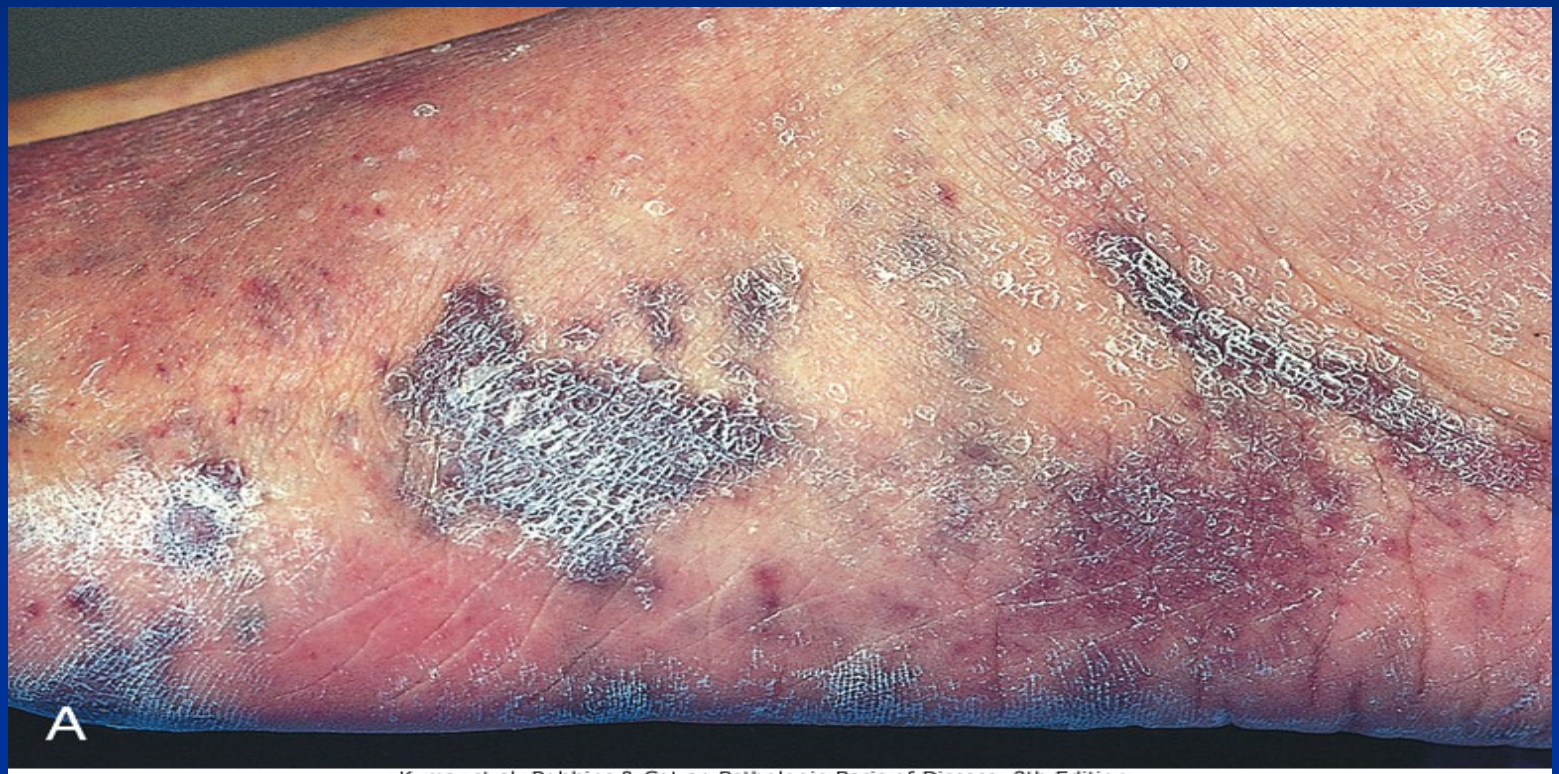
- Abnormal dilatation of veins + valve inkompetence + risk of superfitial thrombosis
- Women > men
- Usually lower extremities
- Inherited trait + high venous pressure (prolonged standing, sitting; hormonal changes, obesity, heart failure)
- Preventive measures + exercise

Cardiovascular tumors

- Cardiac tumors: rare, mostly benign
- Hemangioma: benign vascular (endothelial) tumor, any localisation possible, common on skin, mucosa
  - Red-blue focus
  - Size mm – 15 cm
- Hemangiosarcoma: malignant vascular tumor, any localisation possible, rare, very aggressive, fatal
  - red to purple patches
  - raised plaques
  - nodules

Kaposi sarcoma

special type of angiosarcoma, in immunodeficiency (HIV)



Kaposi sarcoma