

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 arrhythmia)
- 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, intensity, 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/modifying exercise

■ Clinical signs

- Pallor, peripheral cyanosis; cold + moist skin
- Confusion
- Resting heart rate $>130/\text{min}$ or $\leq 40/\text{min}$
- Arrhythmias (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 involvement (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)
- cardiac and/or extracardial pathologic changes

Cardial changes

- disproportion between heart function and peripheral vascular resistance
- differences 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 – unstable 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

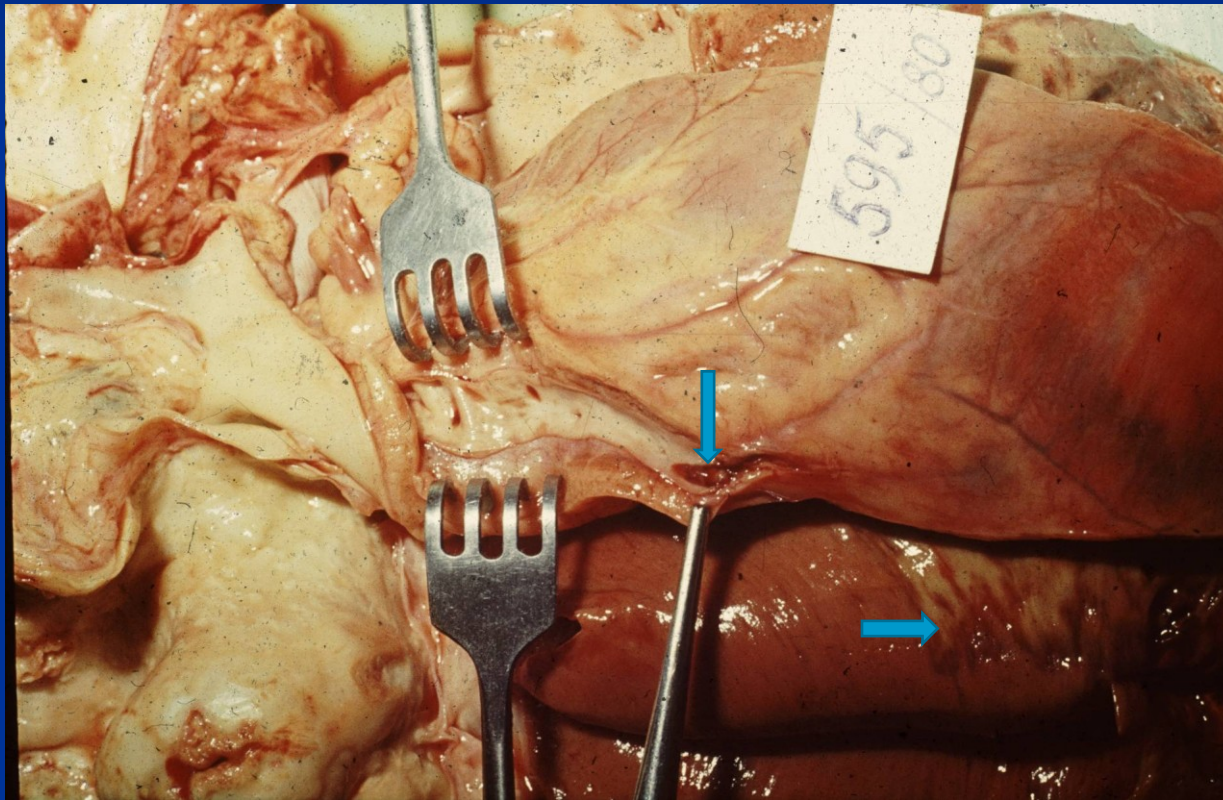
3. variant (Prinzmetal) angina

- 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

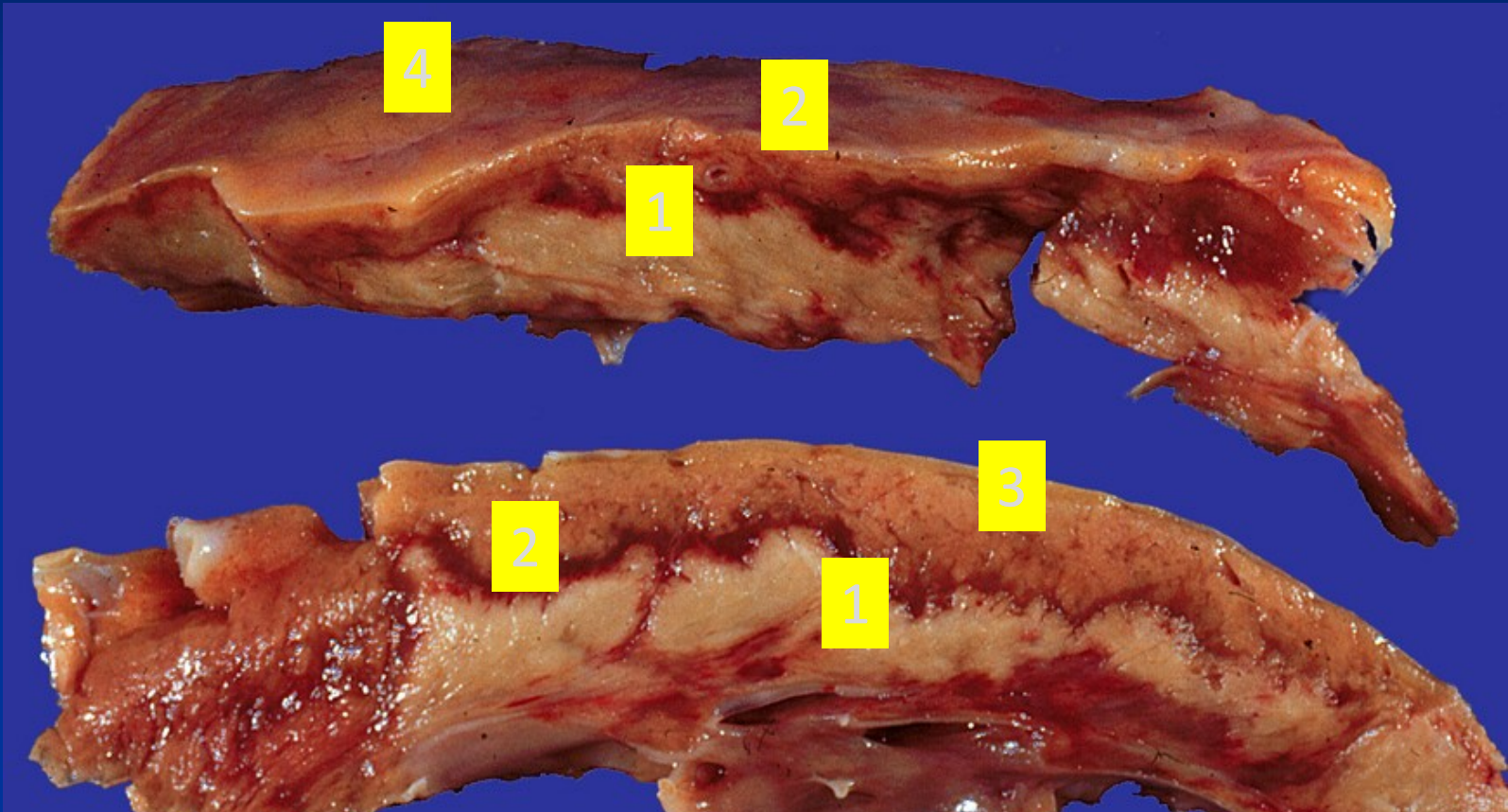
Myocardial infarction + coronary thrombosis



Myocardial infarction



Myocardial infarction



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

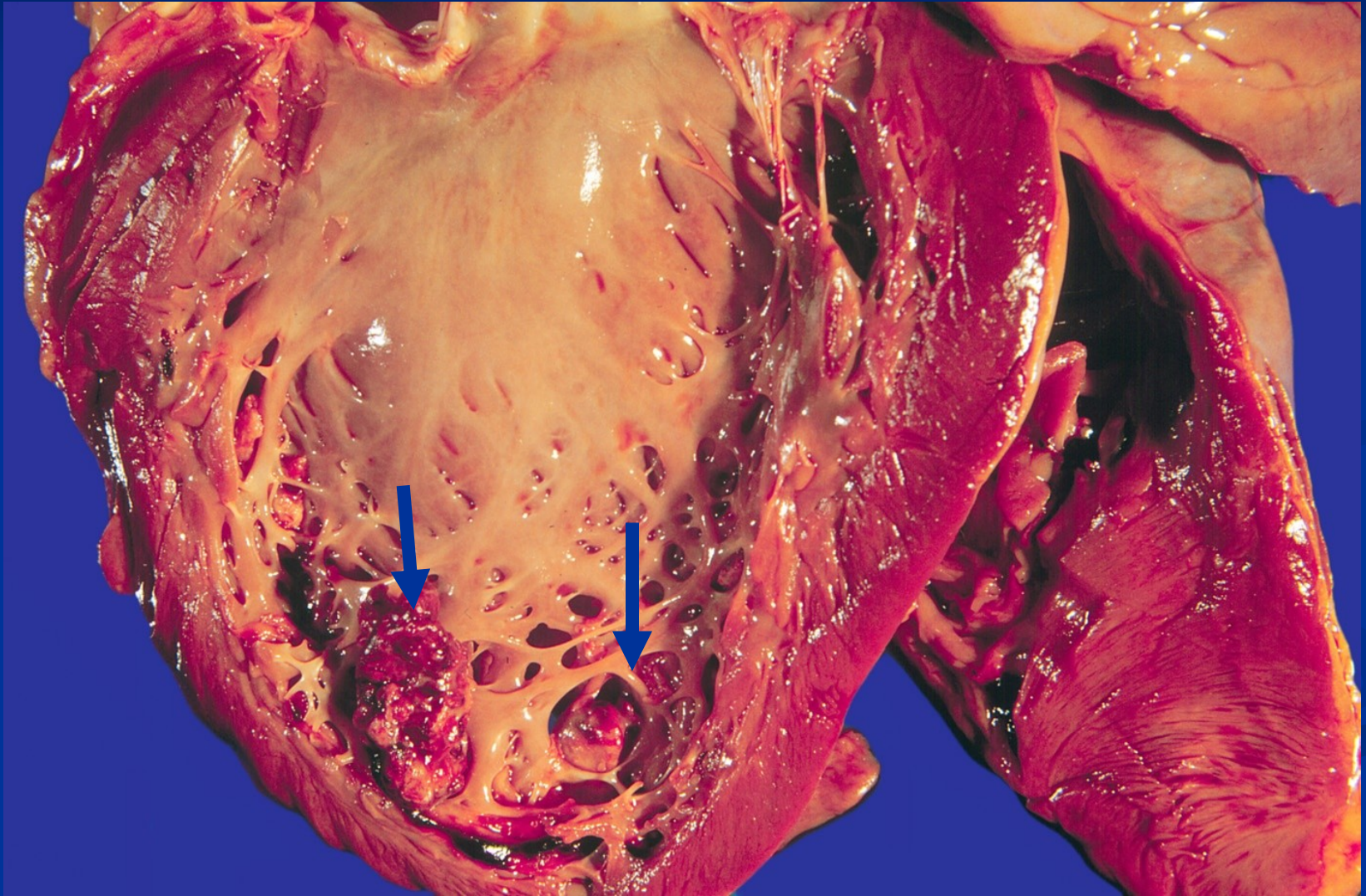
Myocardial infarction

- **transmural (QIM, STEMI) - + ST elevation on ECG**
 - $\geq \frac{3}{4}$ of wall thickness, breadth >25 mm
 - complete coronary artery obstruction
 - emergency angioplasty/stenting
- **non-transmural (subendocardial, Non-STEMI)**
 - internal $\frac{1}{4}$ to $\frac{1}{2}$ of LV wall
 - collateral blood flow, incomplete obstruction, shorter ischemia

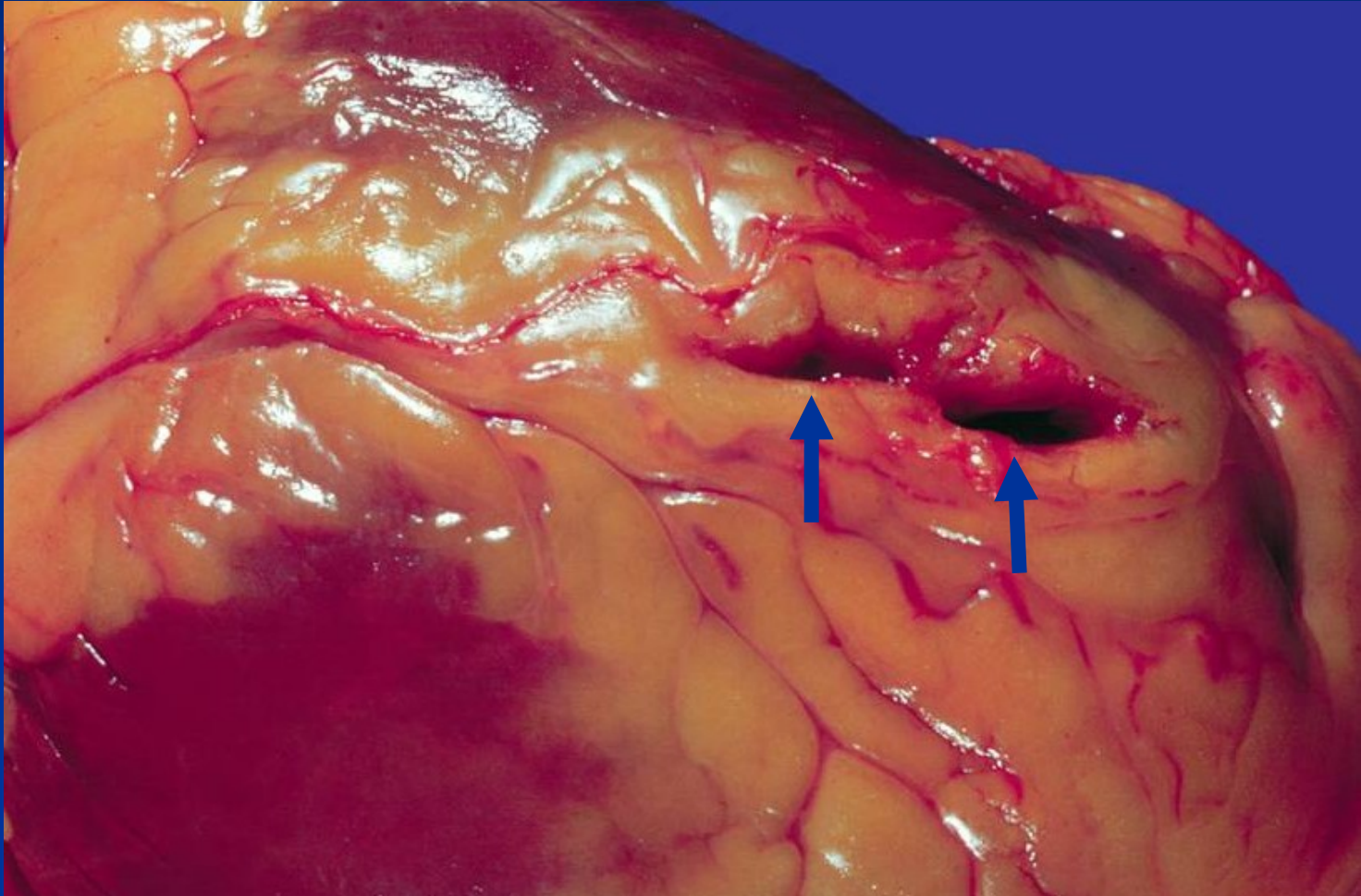
MI complications

- **sudden death (arrhythmia)**
- **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, thrombosis; chronic – LV insufficiency
- **cardiac rupture**
 - → free wall, septum, : tamponade / acute heart failure
- **papillary muscle rupture**
 - → valvular incompetence → acute heart failure

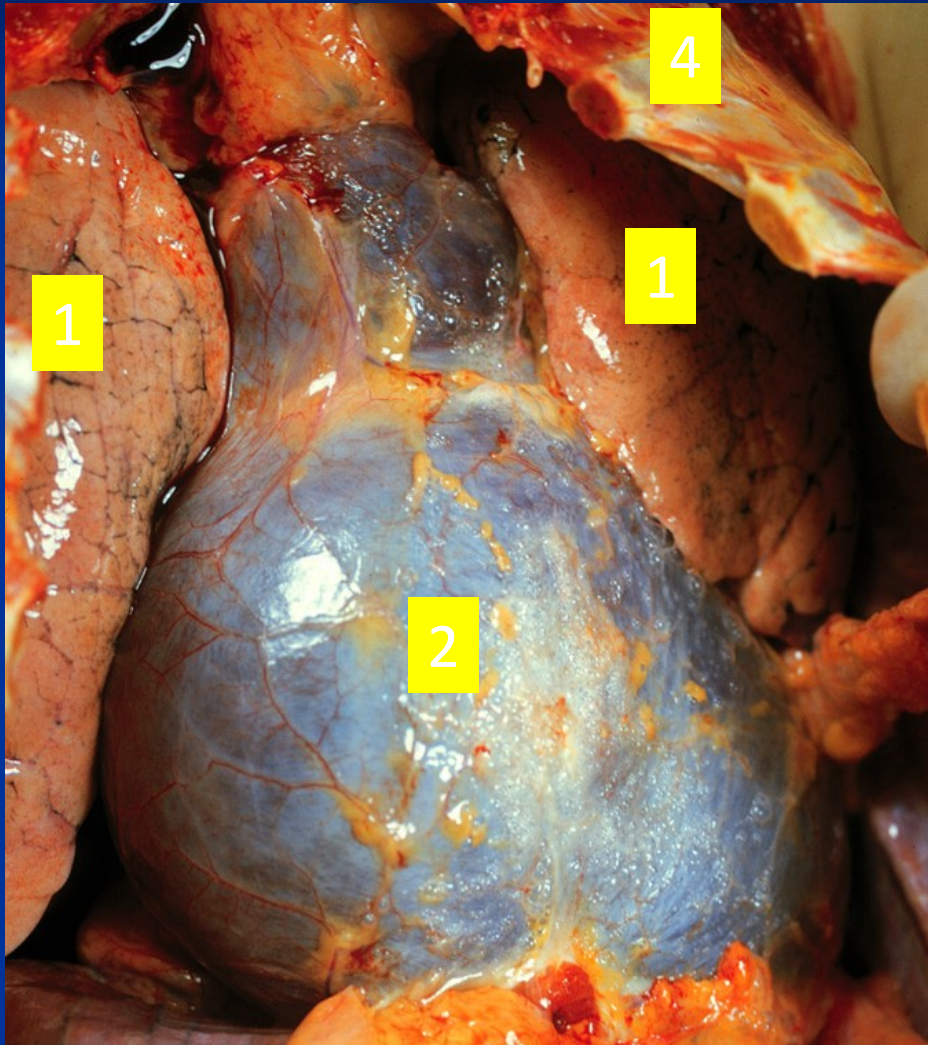
MI – mural thrombosis



Mi – rupture

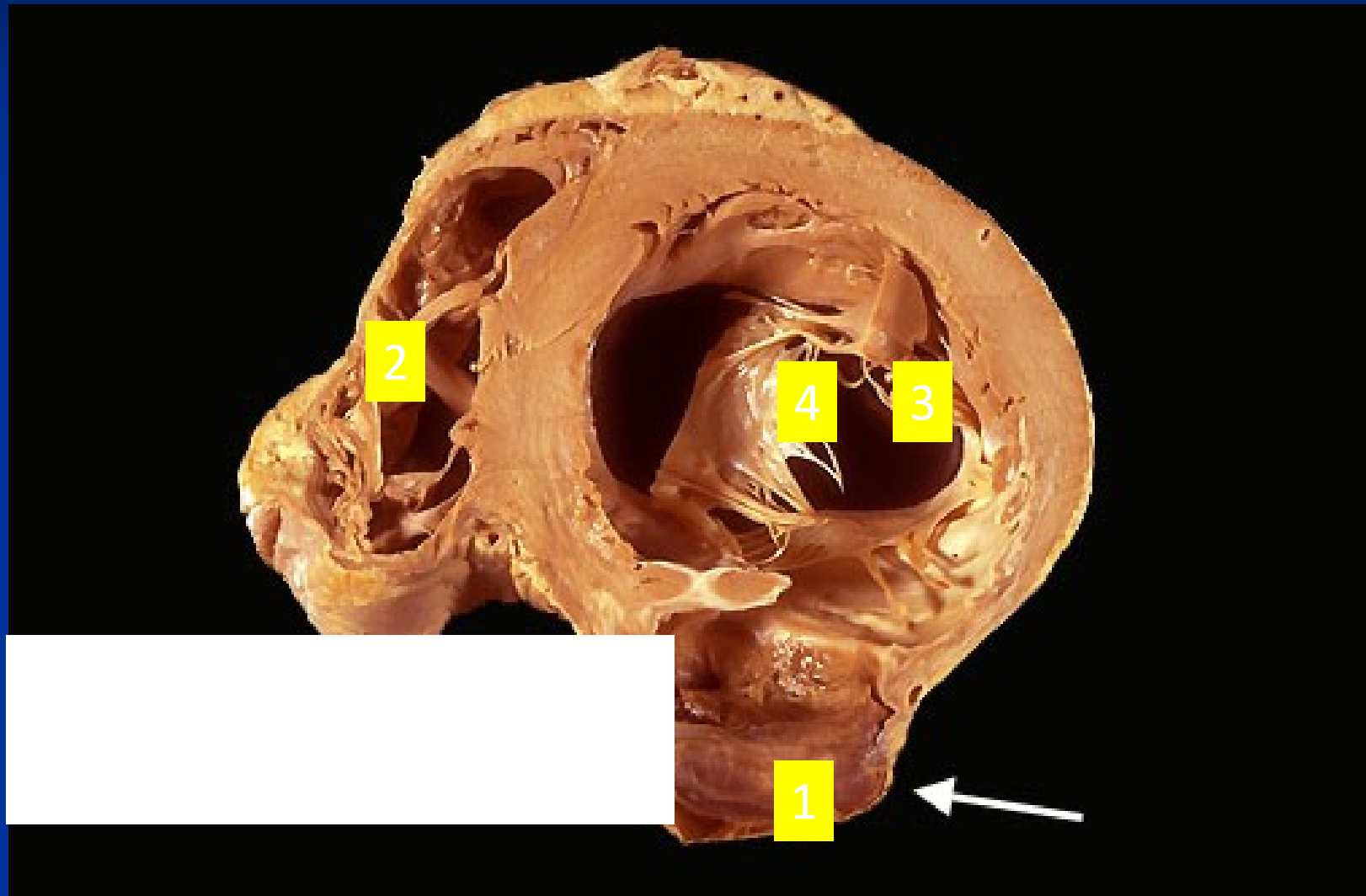


MI – rupture, tamponade



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 + 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

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 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

Myocarditis

- myocardial inflammatory damage without ischemia
- rapidly (days) progressive heart insufficiency
- **gross:**
 - cardiac dilatation, flabby, mottled myocardium
- **etiology:**
 - viruses, rickettsia, chlamydia, bacteria (diphtheria, sepsis), fungi, protozoa (toxoplasmosis), helminths (trichinosis)
 - immune-mediated (drug hypersensitivity, 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,...)

Arrhythmias

- Disturbance of heart rate and/or rhythm
- 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 arrhythmia x ventricular fibrillation (fatal without resuscitation)

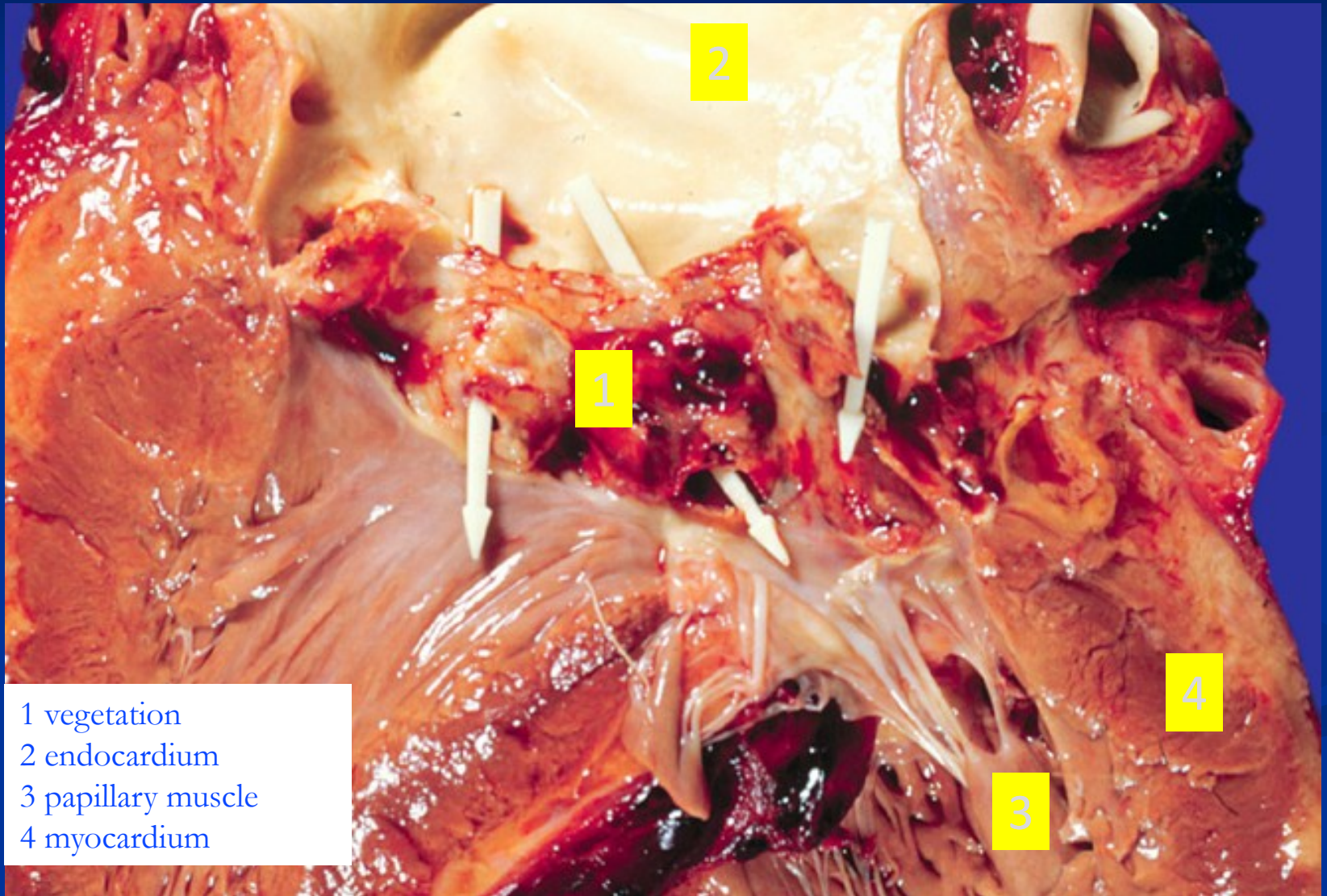
Valvular heart disease

- 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. pyogenes, Strep. pneumoniae, Staph. aureus, ... ev. fungi
- subacute IE – less virulent microorganisms
 - viridans streptococci
- predisposition:
 - deformed valve, bioprosthesis, stomatologic, surgical procedures, postcatheterization, i.v. drug addicts
 - tooth brushing, chewing (oral flora common source) in immunodeficient patient
- bacteremia - endocardial damage by bacteria - thrombosis = infective vegetation

Infective endocarditis – valve destruction



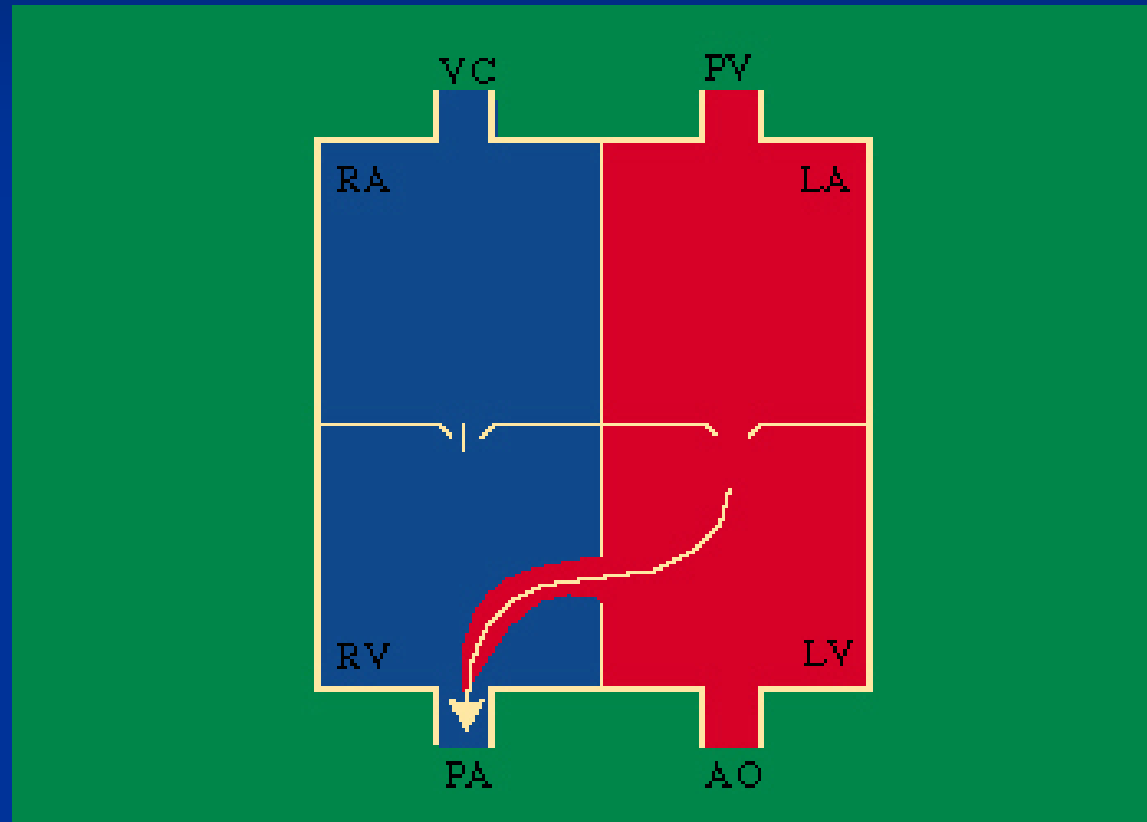
Rheumatic fever, rheumatic heart disease

- acute non-purulent, **immune-mediated** systemic poststreptococcal inflammation
(cross-reactive antibodies)
- acute stage **PANCARDITIS**
 - acute endokarditis, commonly recurrent
- chronic stage:
 - valvular calcification - stenosis + incompetence

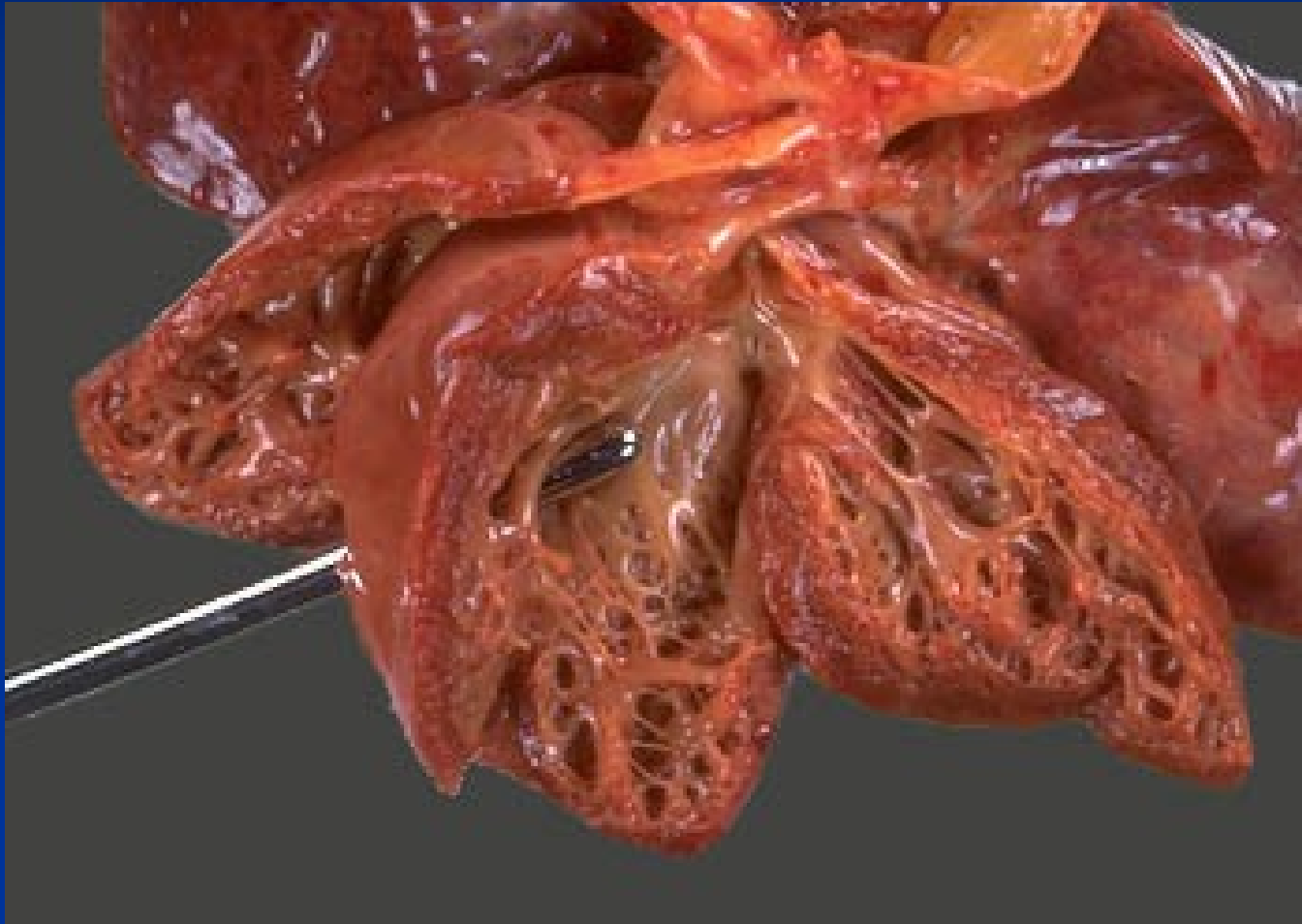
Congenital cardiovascular disease

- 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 tissue → adhesions, dystrophic calcification.

Acute fibrinous pericarditis



Atherosclerosis

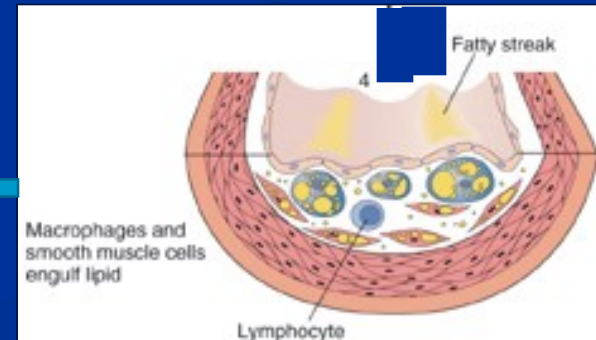
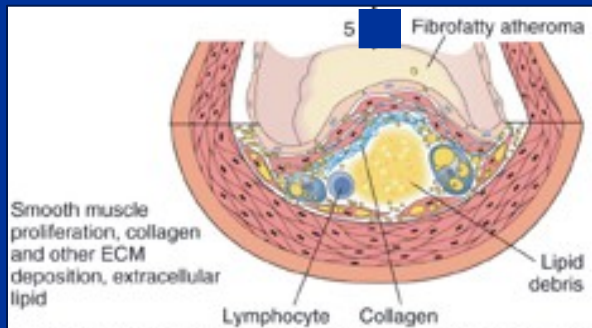
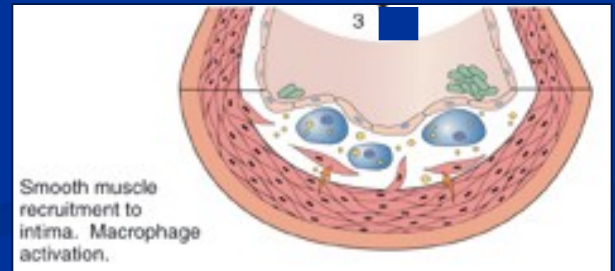
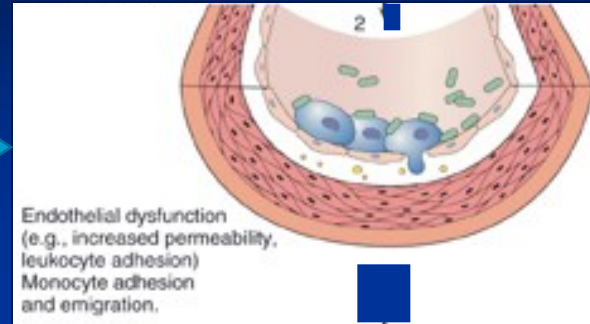
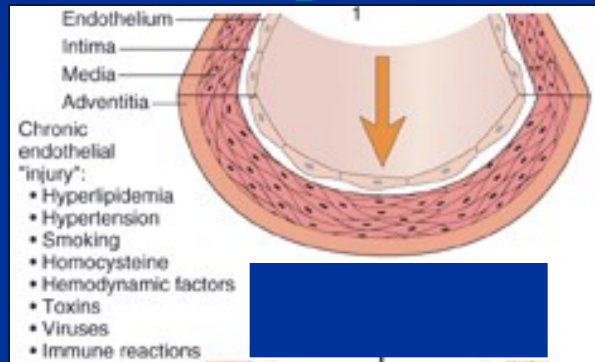
MULTIFACTORIAL COMPLEX DISEASE

- – unknown exact cause, combination of chronic inflammation, 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) ←←← hypothyroidism, nephrotic sy;
 - hypertension, diabetes mellitus, life style smoking (nicotine, CO), sedentary life, food + obesity; ↑CRP

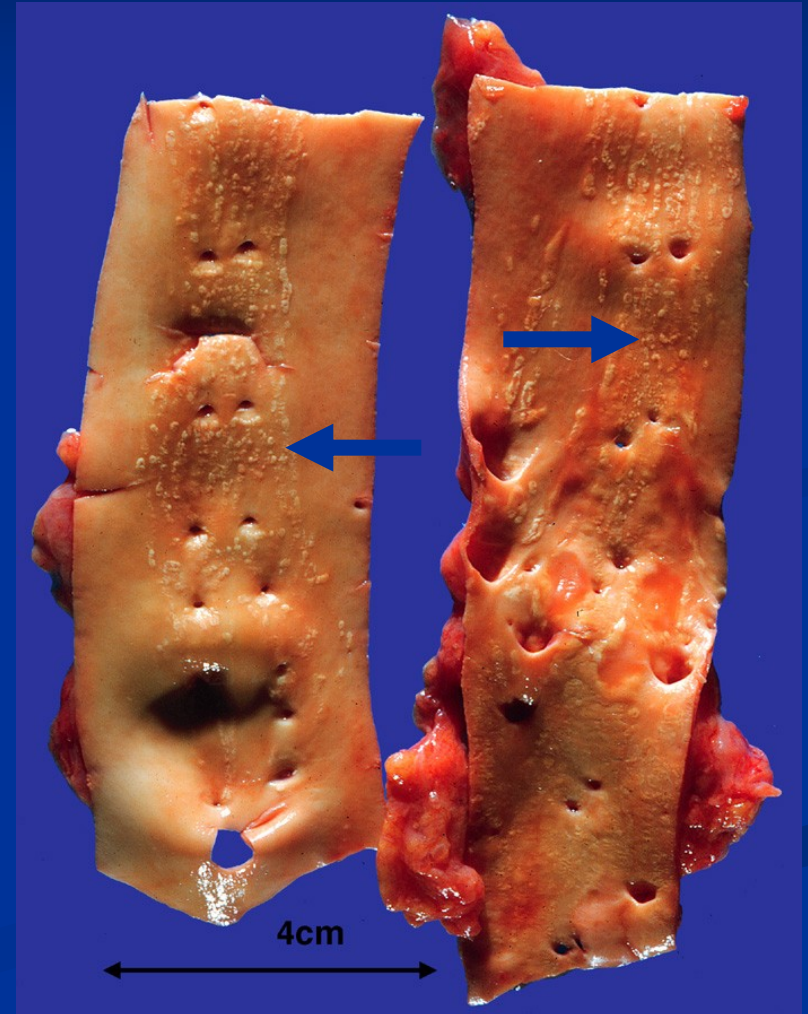
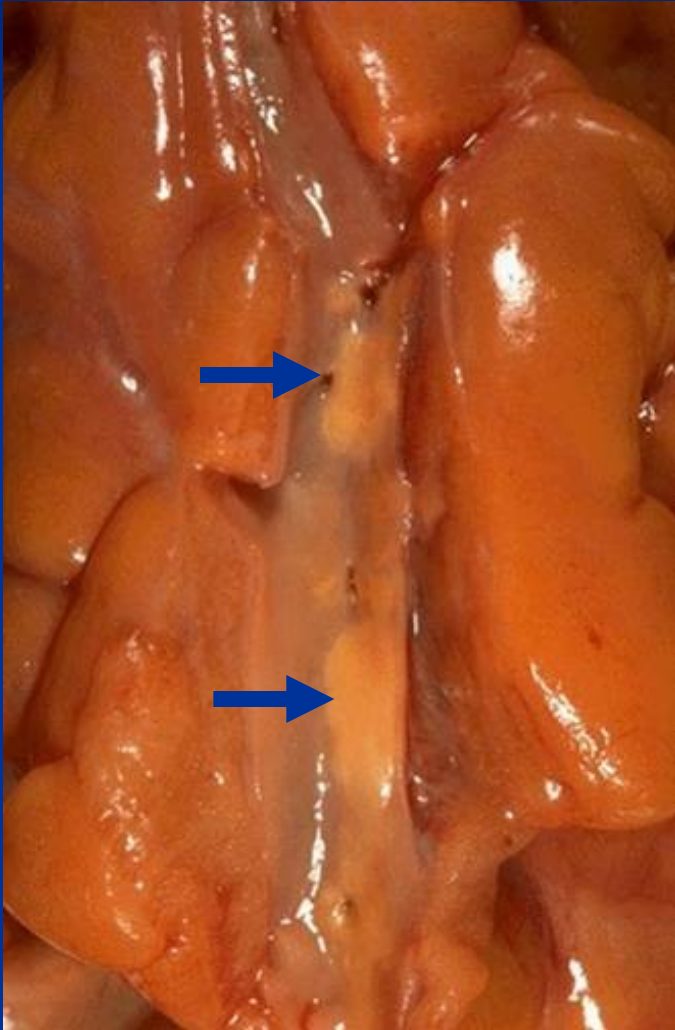
Atherosclerosis - pathogenesis



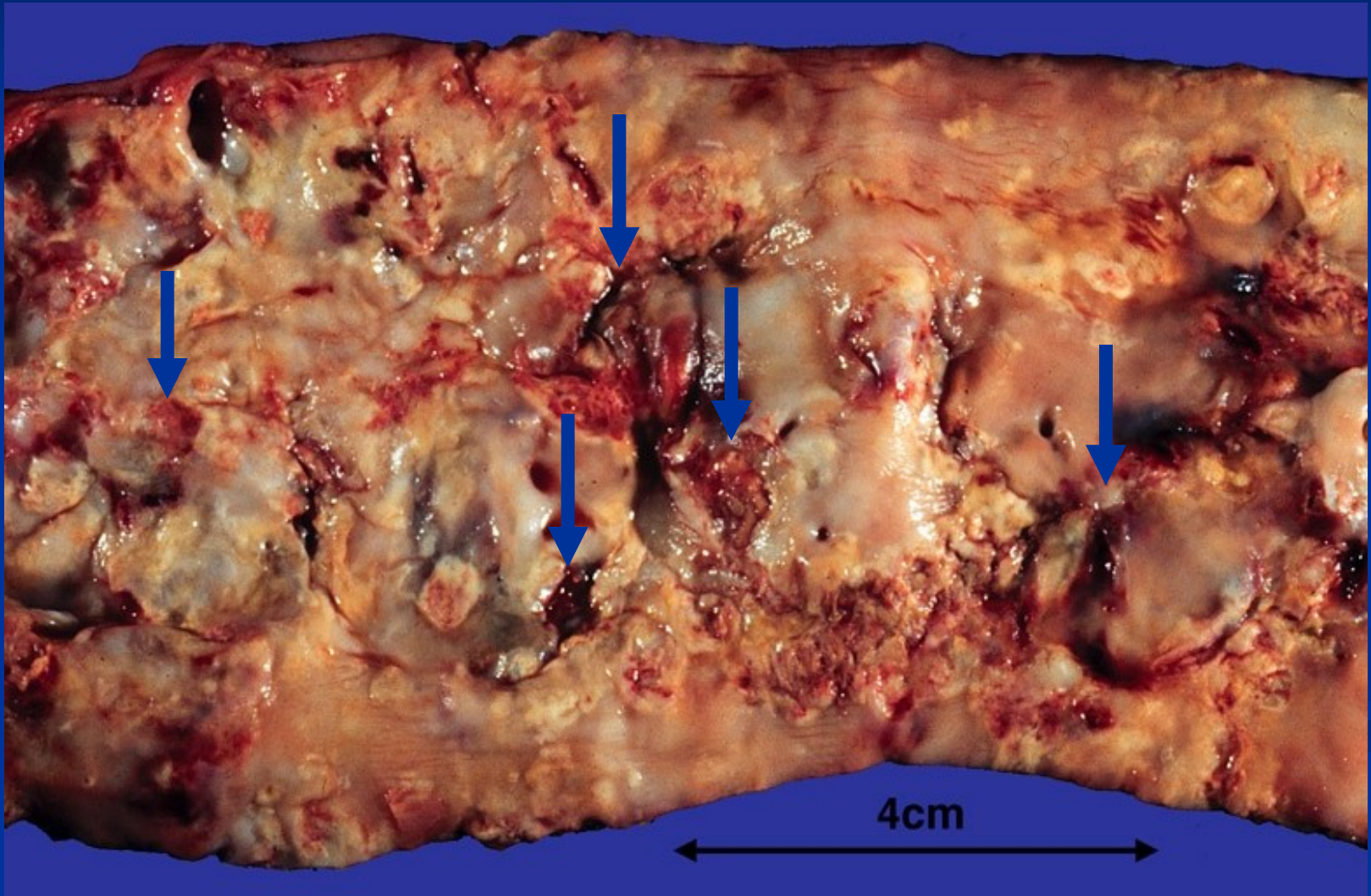
Atherosclerosis stages/changes

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

Atherosclerosis – fatty streak



Atherosclerosis – plaque ulceration, mural thrombosis



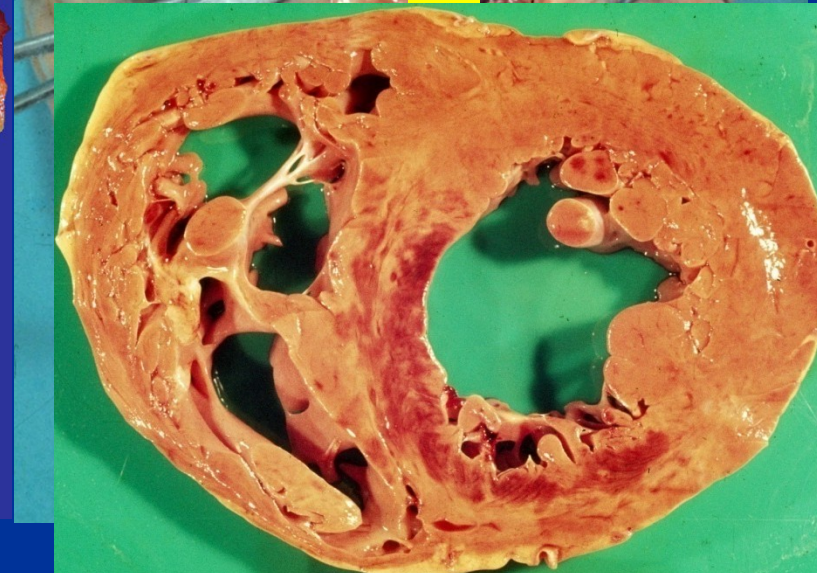
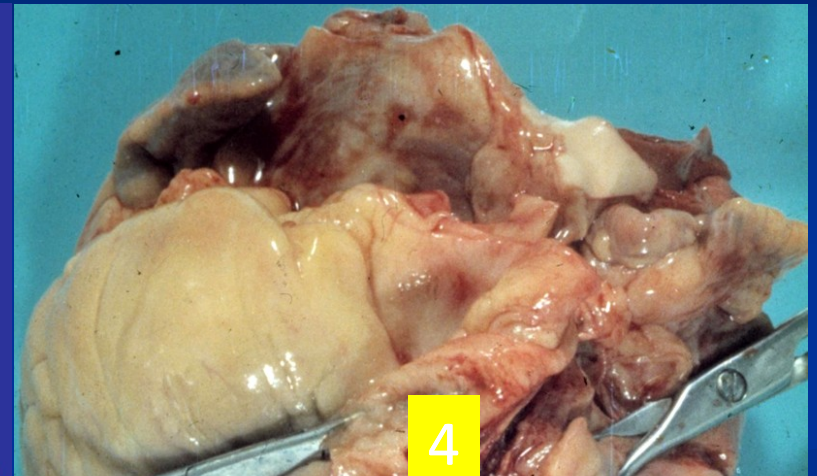
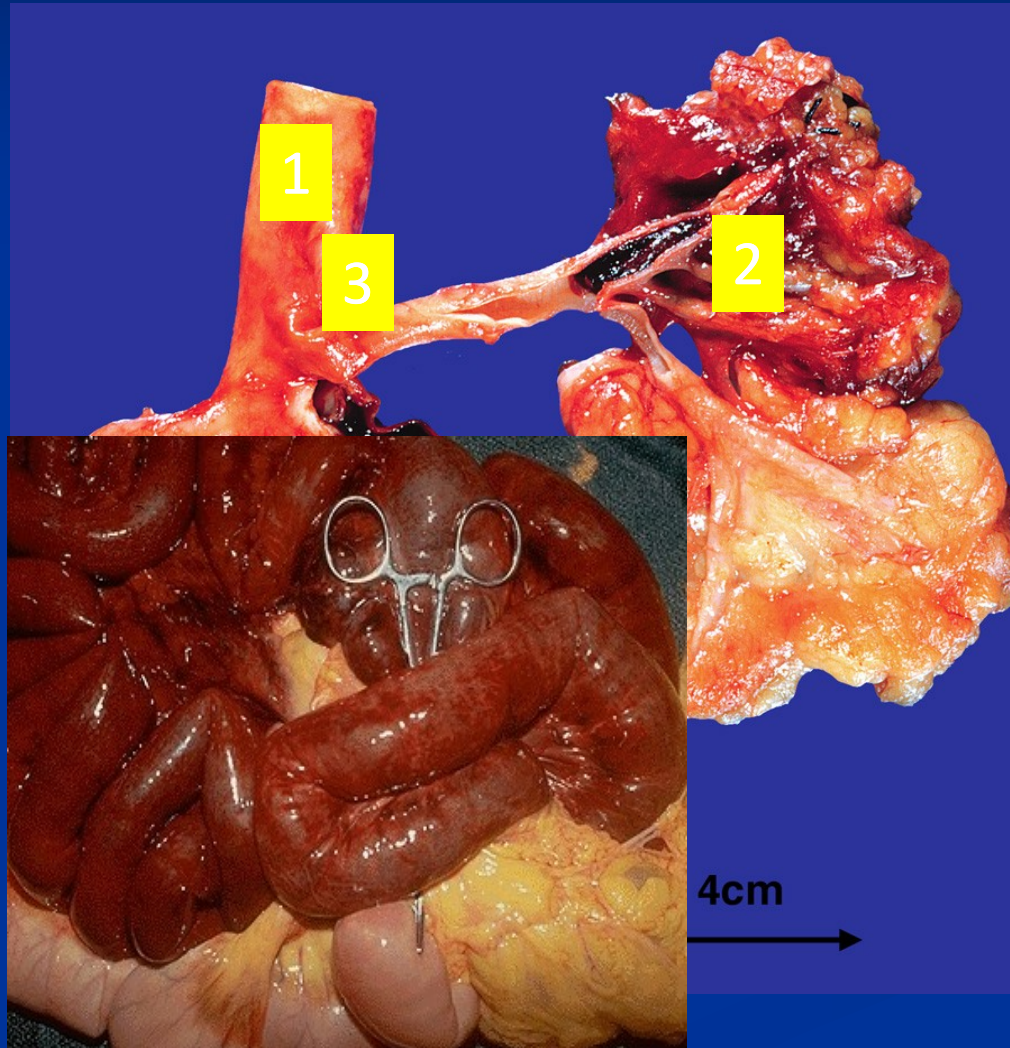
Atherosclerosis

SEQUELS: arterial occlusion in situ

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

Atherosclerosis – complications

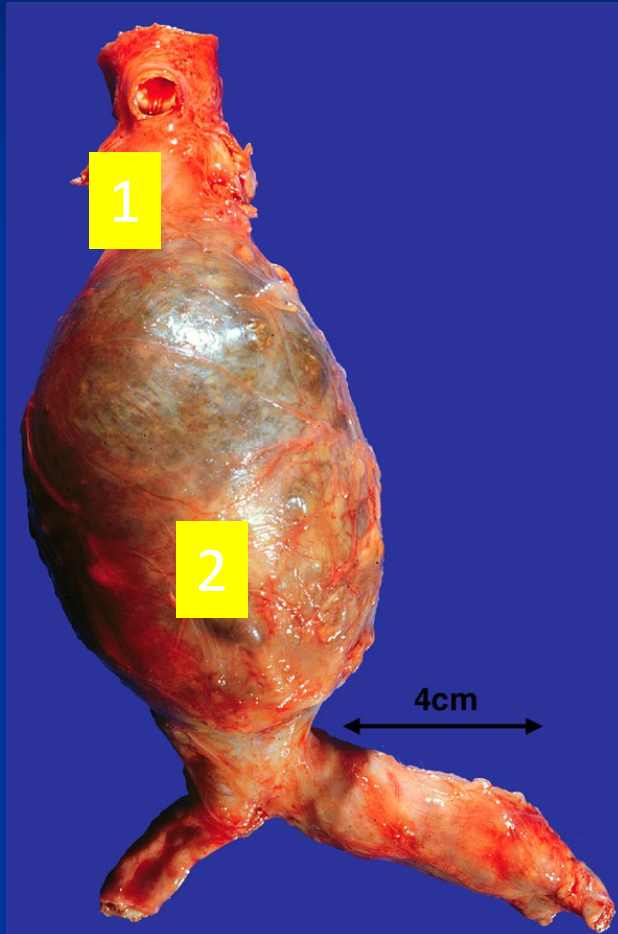
thrombosis/thrombembolia



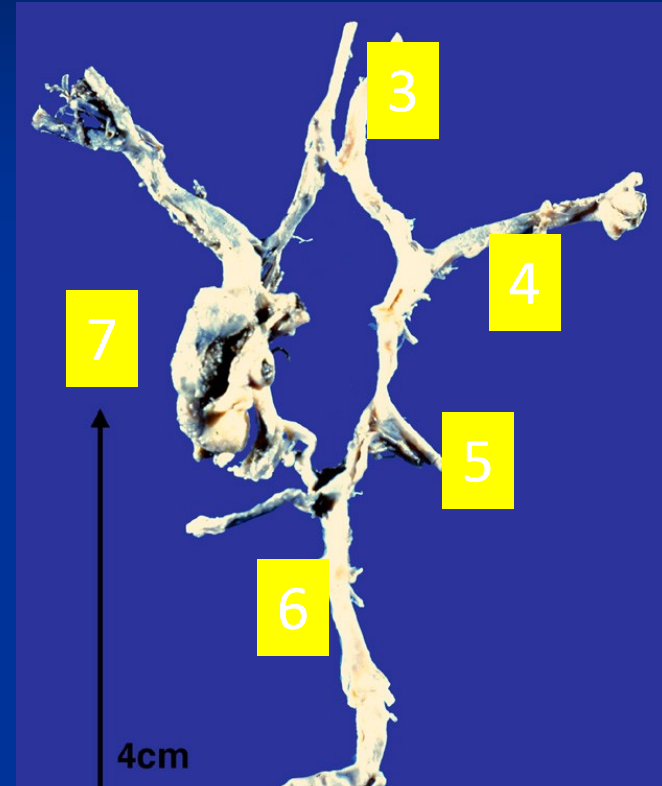
Aneurysm

- 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

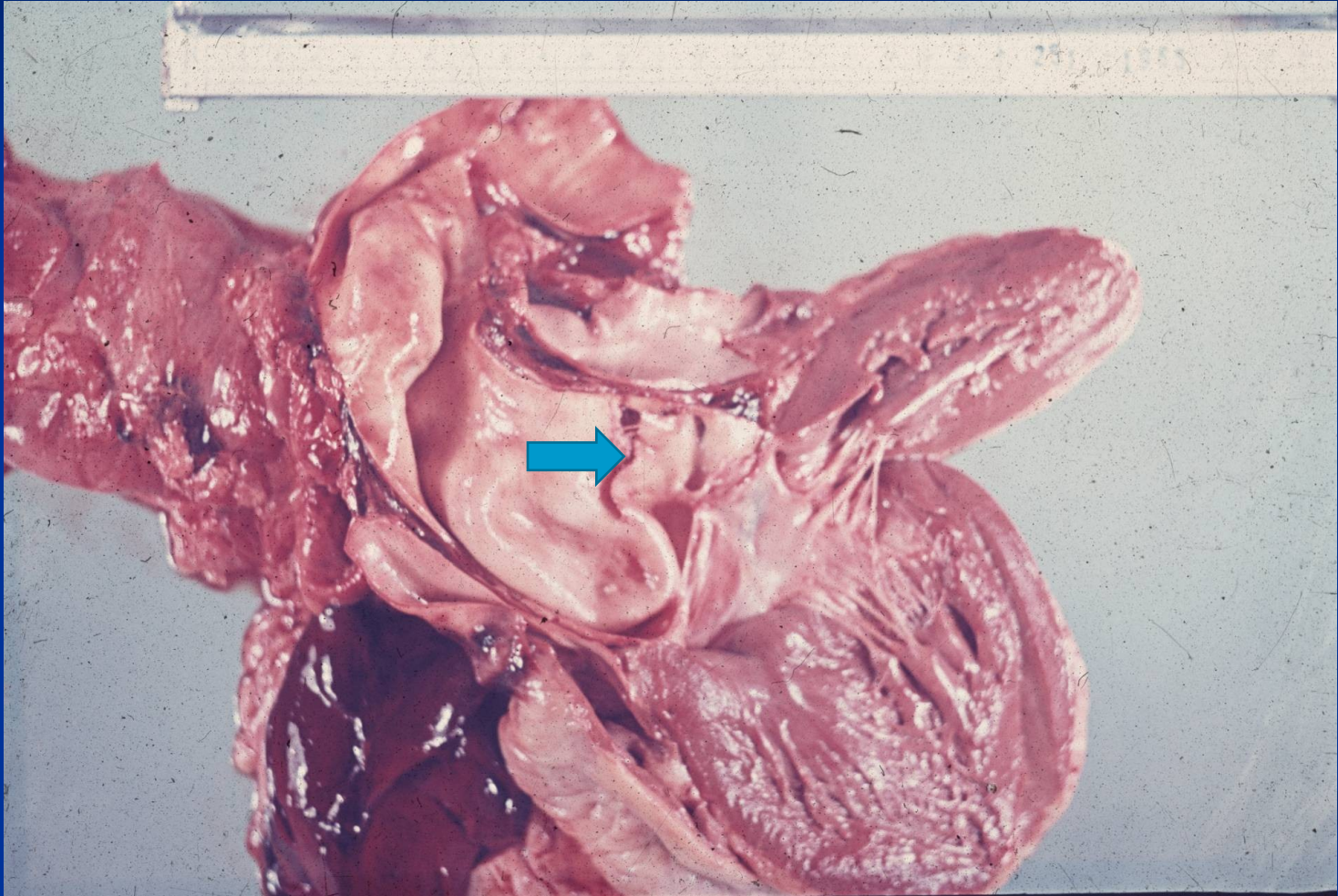


3 a. cerebri anterior
4 a. cerebri media
5 a. cerebri posterior
6 a. basilaris
7 aneurysm

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

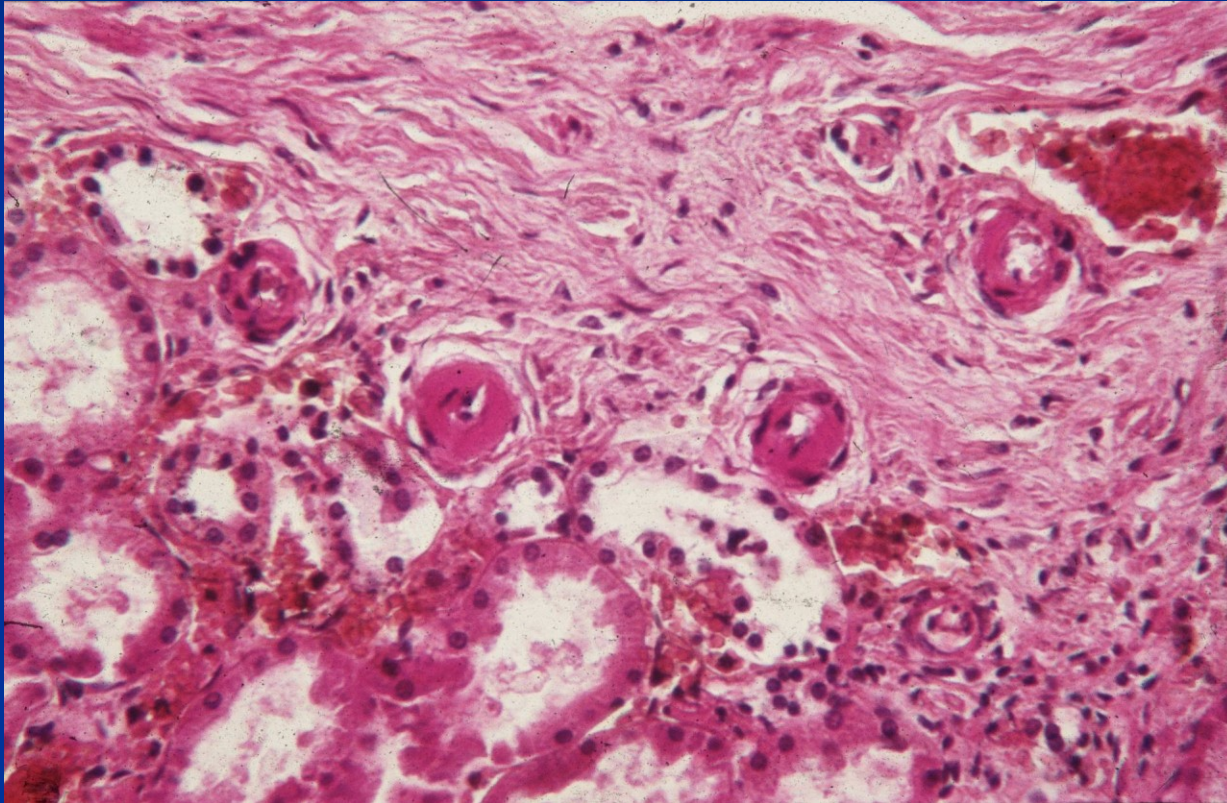
age and/or hypertension related changes

→ 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)

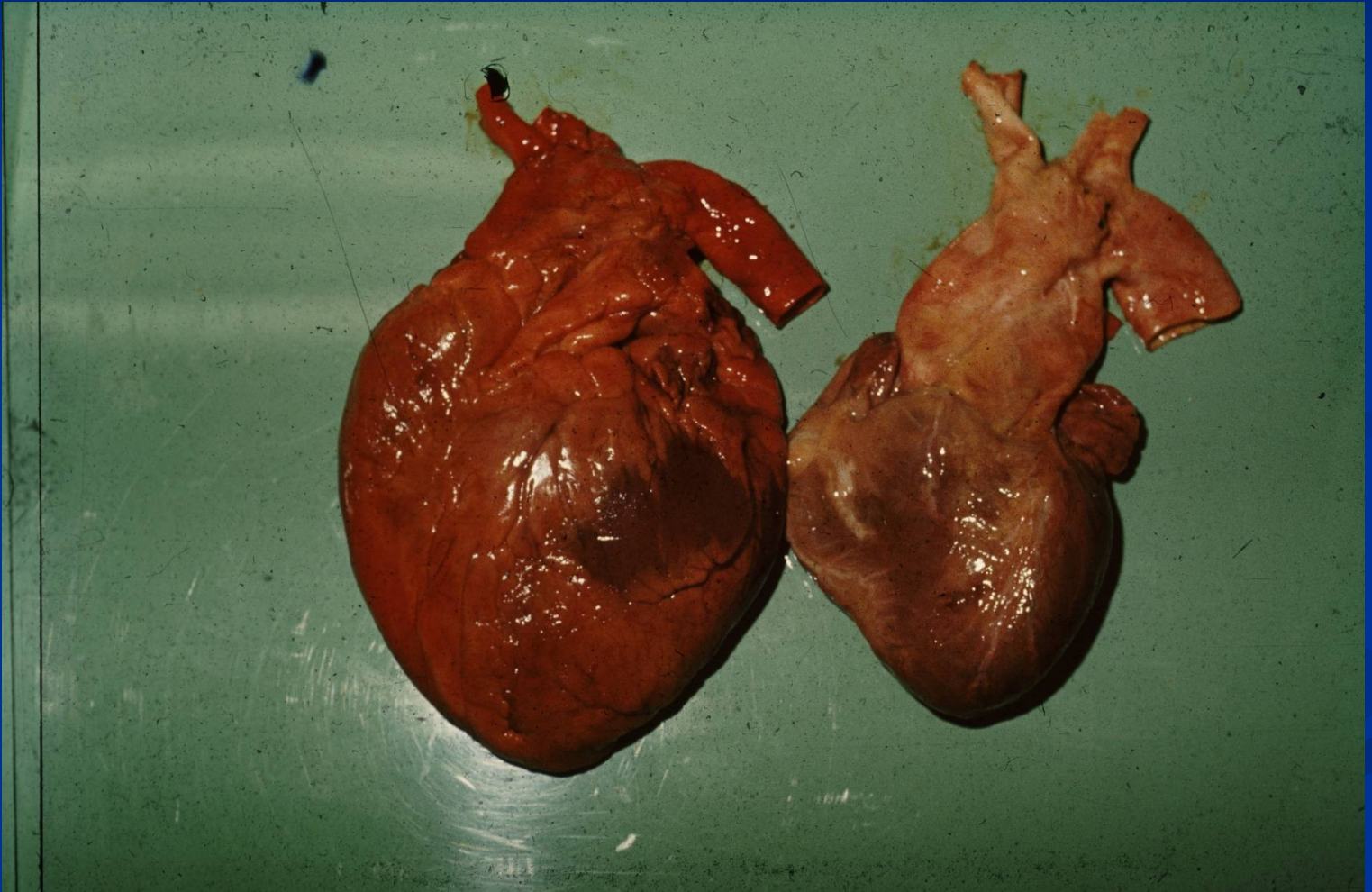
Systemic hypertension

- 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



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 immobility, 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

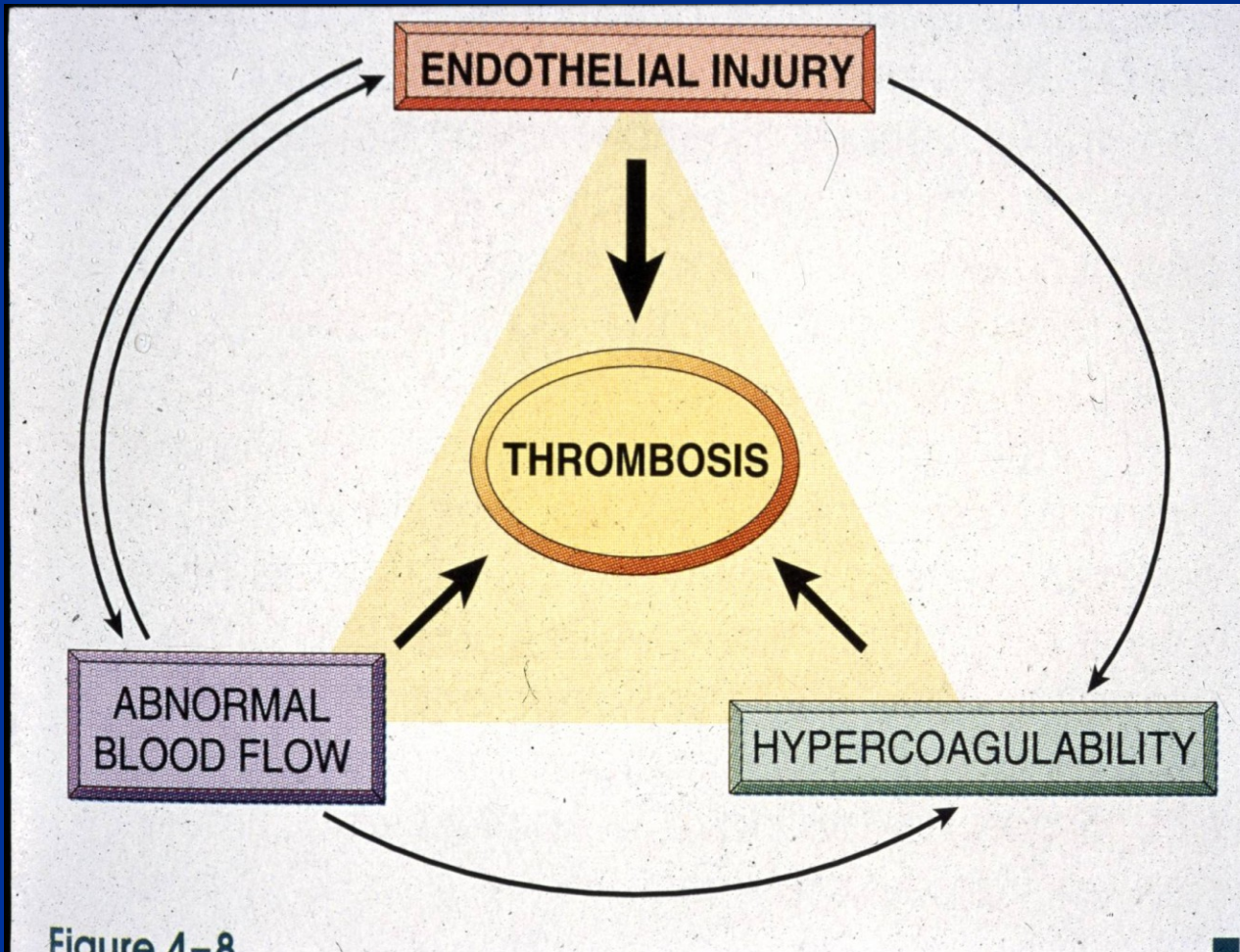
intravital intravascular pathological blood coagulation

thrombocytic aggregation, 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



Thrombosis

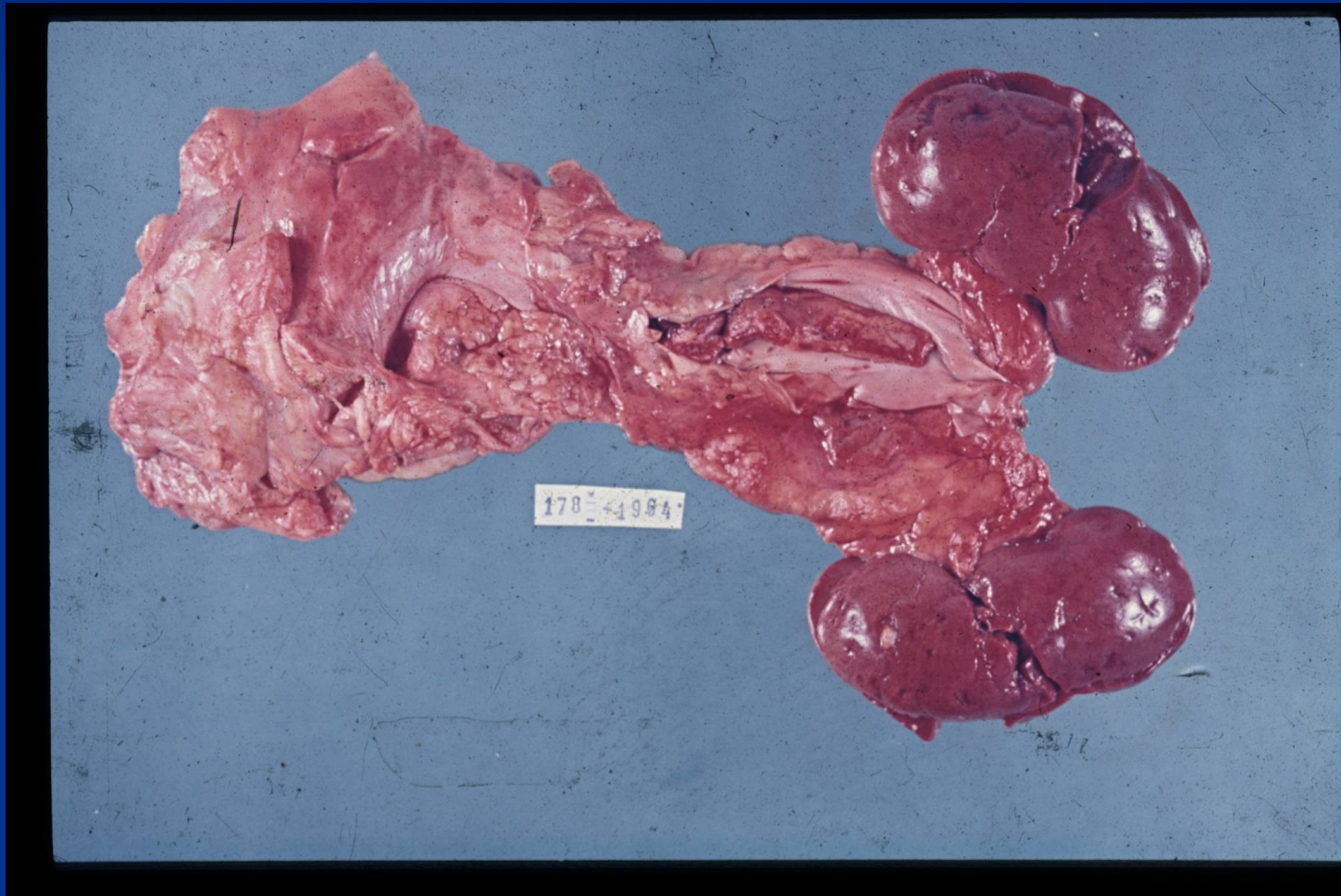
X haematoma – intravital extravascular blood clot

X cruor – postmortal intravascular blood clot

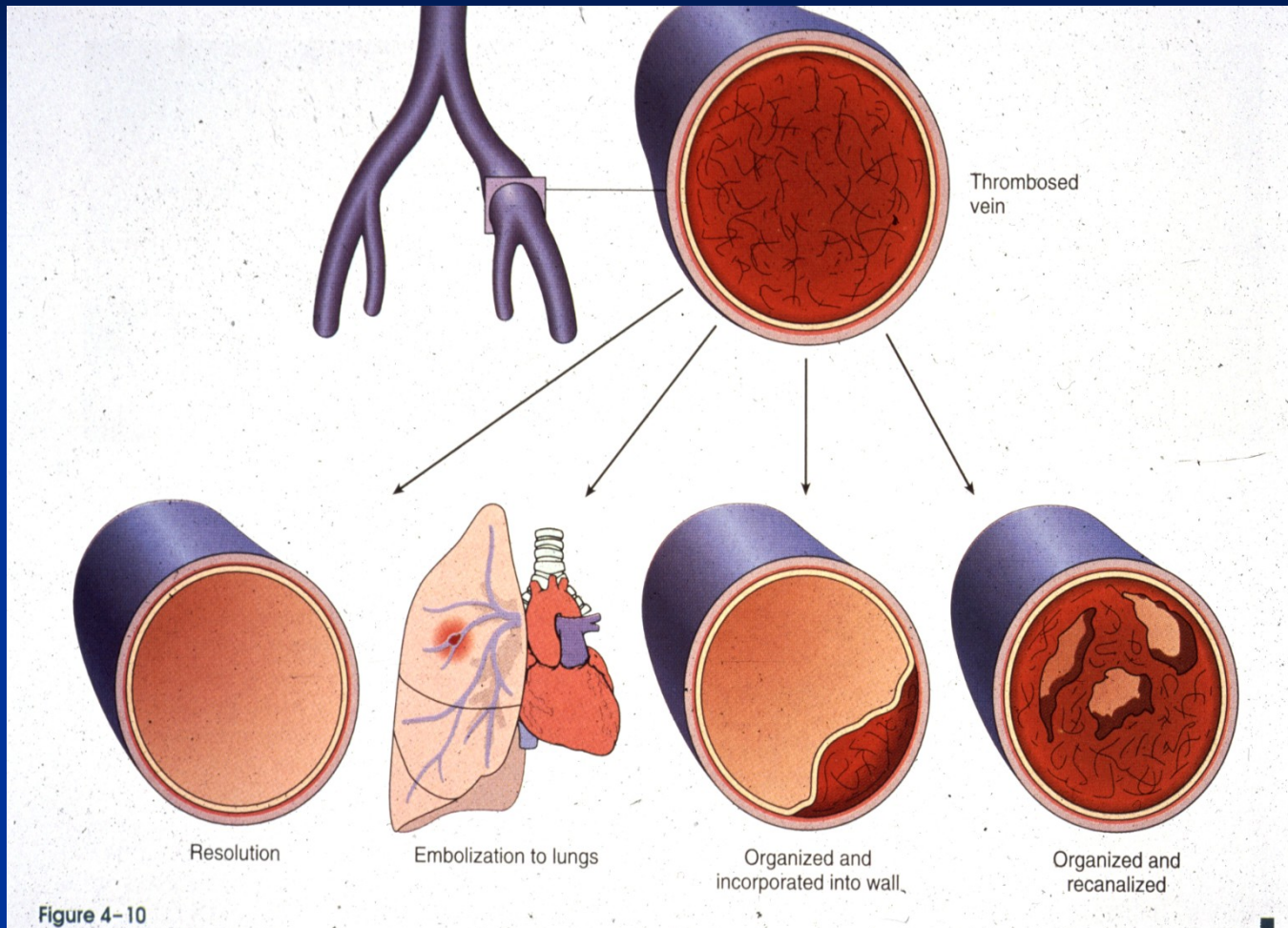
Gross:

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

Venous thrombosis



Fate of thrombi



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 (meningococcal 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 incompetence + risk of superficial 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)



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

