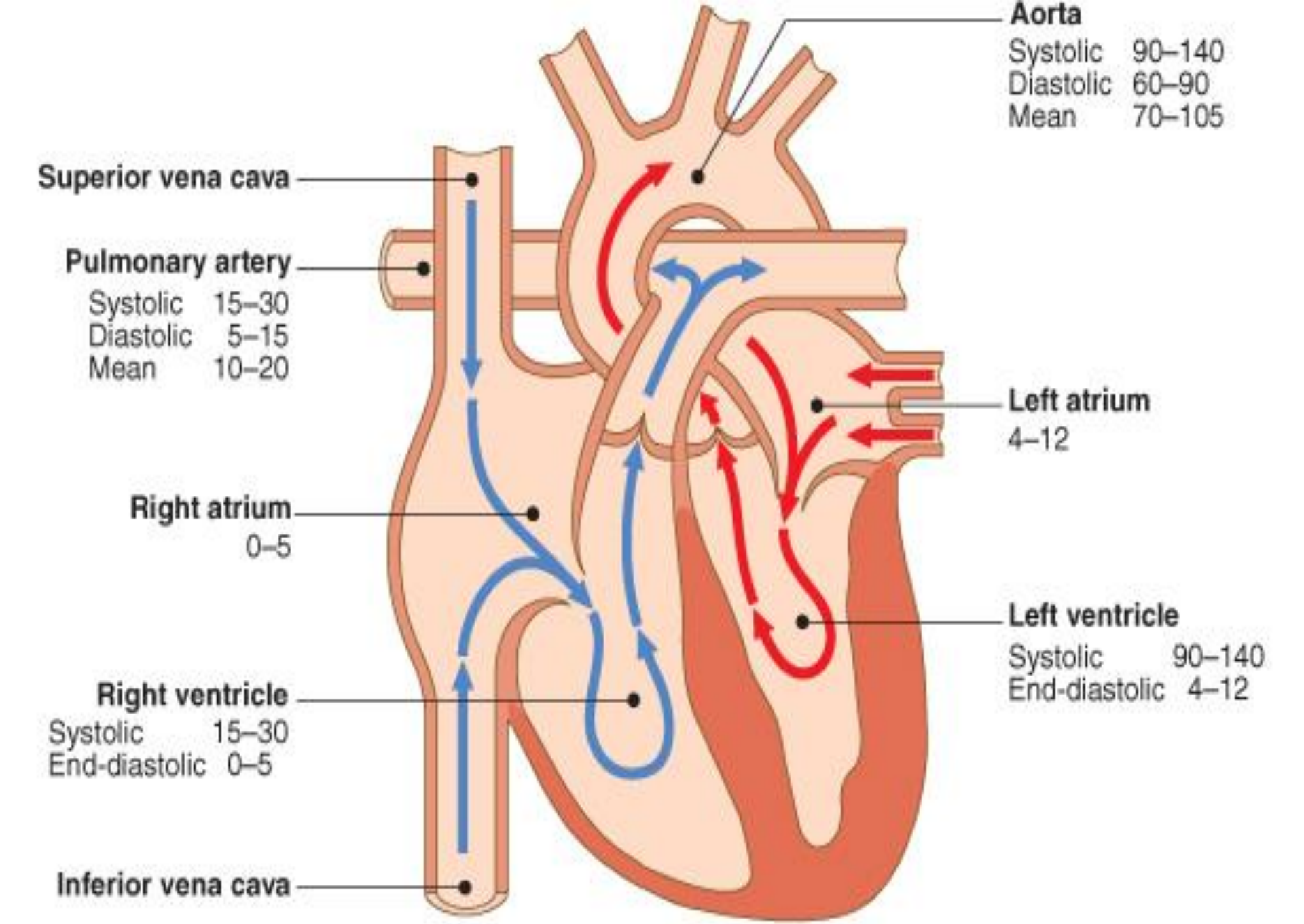
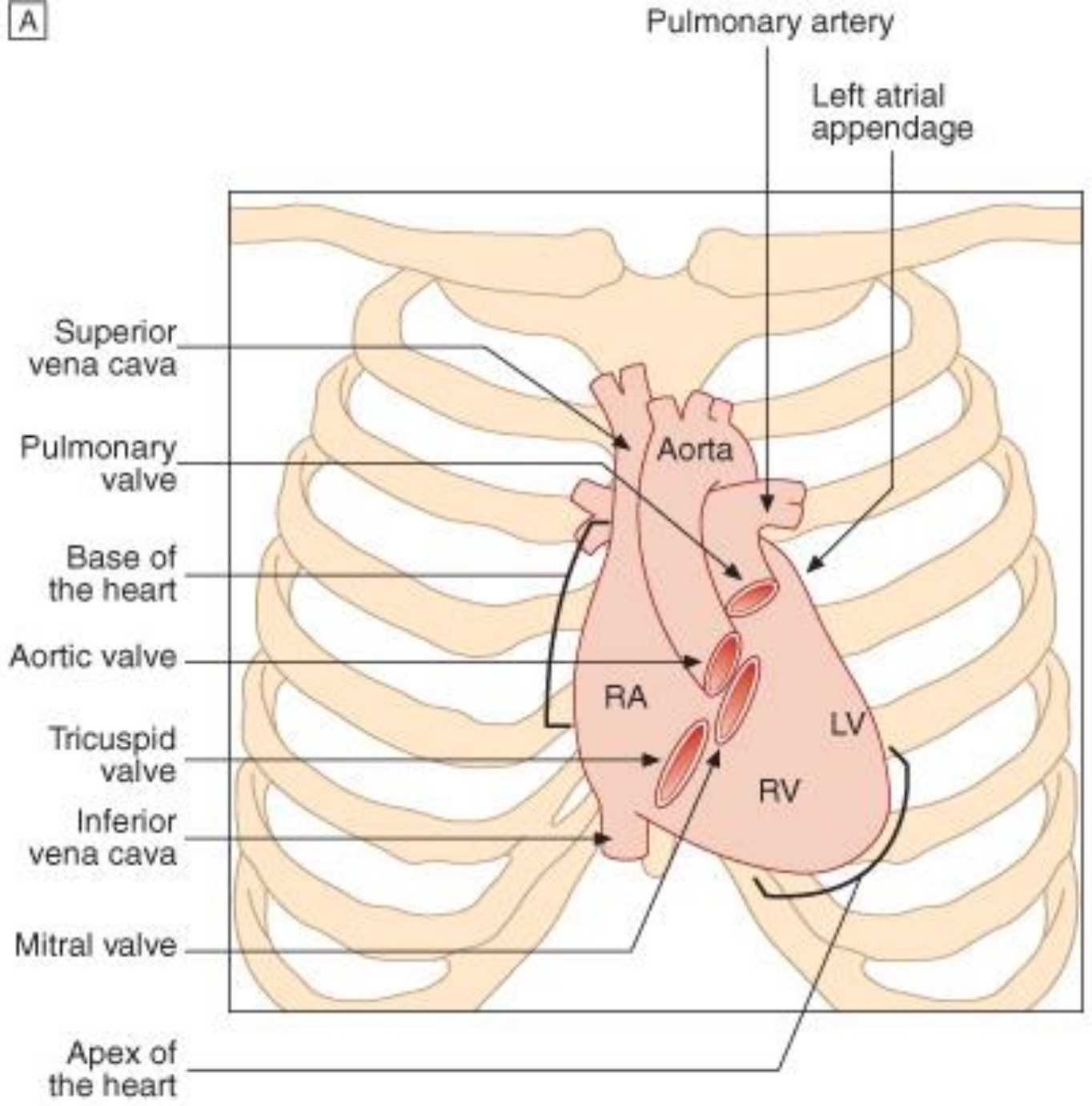
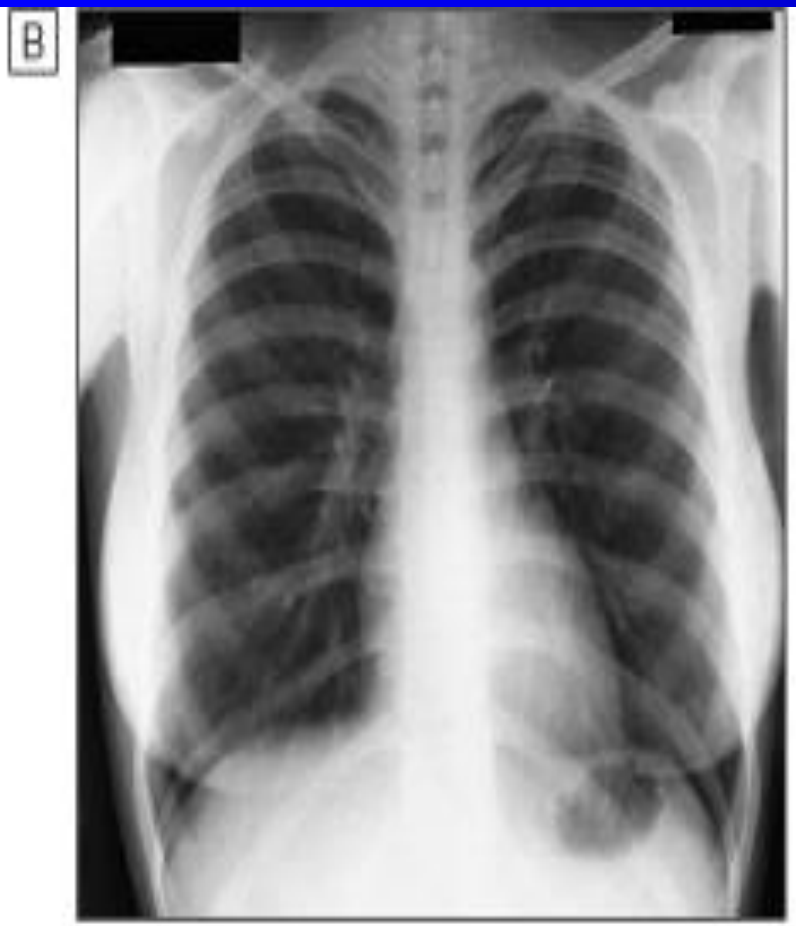






Ischaemic heart disease

Coronary component – acute, chronic restriction (arrest) of blood flow

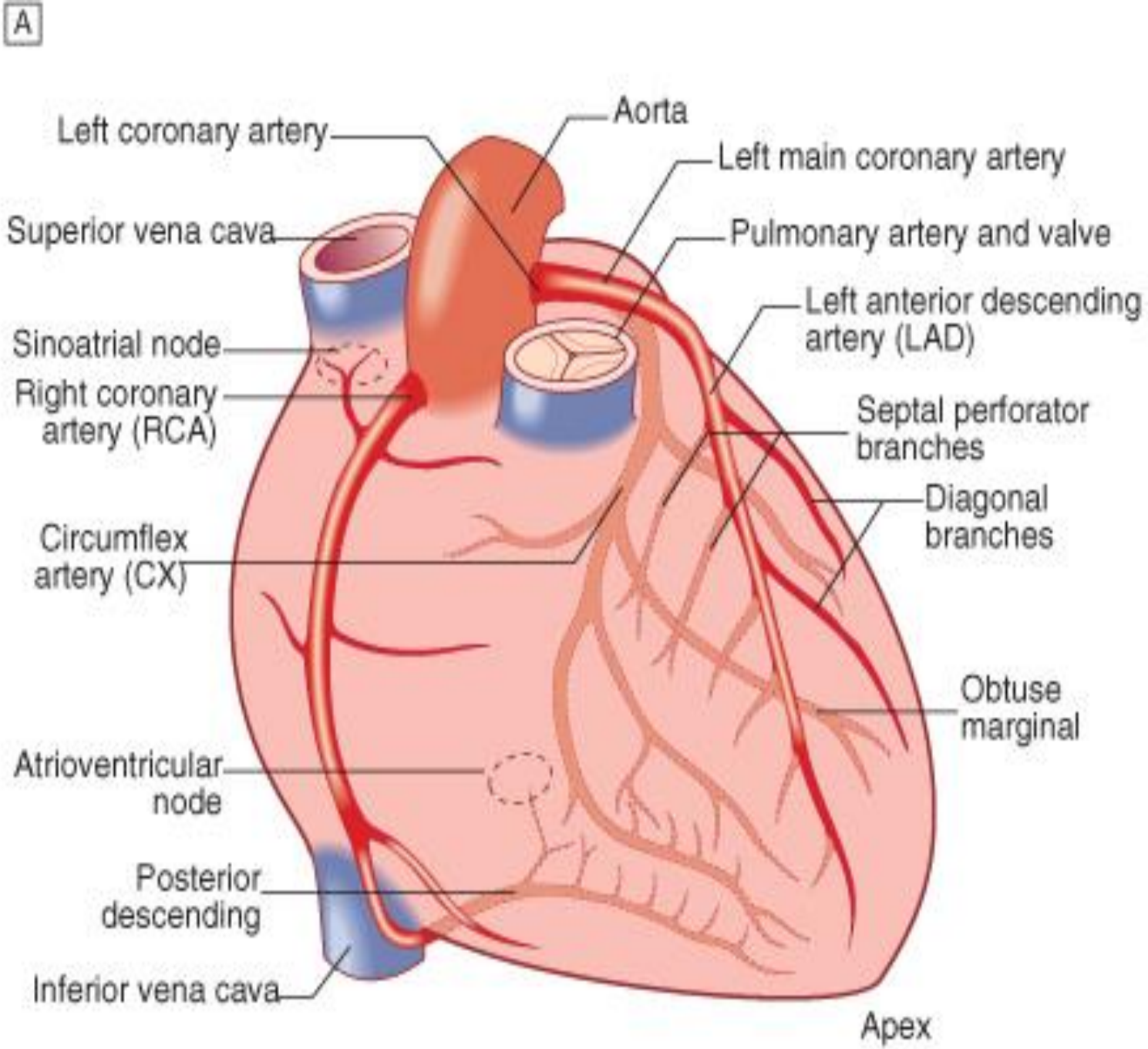
Myocardial component – ischaemia, necrosis

Aetiology

- AS affliction x collagenoses, fixed stenosis
- Spasms of coronary arteries – dynamic stenosis
- Hypertrophy of myocardium – HT, aortic stenosis, hypertrophic cardiomyopathy (HCM)
- Syndrome X – disease of small arteries

Anatomy

- A. coronaria sin. (ACS, LCA) – RIA, RC a. coron. dx (ACD, RCA)
- RIA – anterior wall of LV, septum - 50% of LV myocardium, RBB, anterior bundle of LBB
- RC – lateral and posterior walls of the heart, in 10% atrioventricular node
- ACD - RV, inferior wall of LV, in 90% atrioventr. node, dorsal part of interventr. septum, posterior bundle of LBB



Physiology

- Coronary bloodstream – 5% MSV
- Consumption of O_2 in the myocardium – 12% - extraction of O_2 from capillary blood – much higher
- Blood flow – 75% in the diastole
- Main factor of blood flow – active tonus of coronary arteries – local tension of O_2, concentration of metabolites, sympathoadrenal activity, noxae

Risk factors

Irremovable

Age

Sex

Family history

Removable

Major:

DLP, smoking, HT

Minor:

DM, obesity, physical inactivity, stress

Incidence of IHD

- 5 to 10 cases/year/1000 inhabitants
- Death rate – 2 to 3 deaths/year/1000 inhabitants
- Death rate of CV diseases: 50 to 57 % of the total death rate

Classification of IHD

Painful forms

AP – effort-associated

Acute coronary syndrome

AP - unstable

Prinzmetal AP

MI (20% painless)

Painless forms

Silent ischaemia

Heart failure

Arrhythmia

Sudden cardiac death

Painful forms of IHD

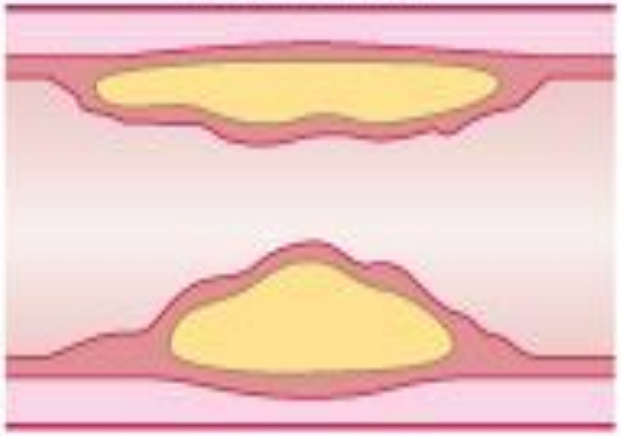
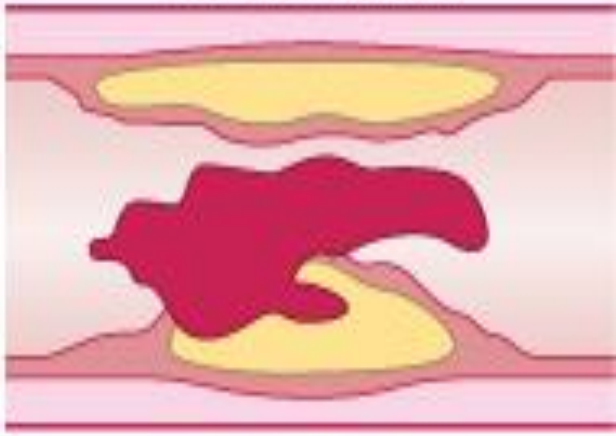
Stable AP

Pain behind sternum - exercise

CCS I – AP on more than usual exertion

II – AP on usual exertion

III –AP on less than usual exertion

	Stable angina	Unstable angina
		
Pathophysiology	• Fixed stenosis	• Dynamic stenosis
Clinical features	• Demand-led ischaemia • Related to effort • Predictable • Symptoms over long term	• Supply-led ischaemia • Symptoms at rest • Unpredictable • Symptoms over short term
Risk assessment	• Symptoms on minimal exertion • Exercise testing Duration of exercise Degree of ECG changes Abnormal BP response	• Frequent or nocturnal symptoms • ECG changes at rest • ECG changes with symptoms • Elevation of troponin

Silent myocardial ischaemia

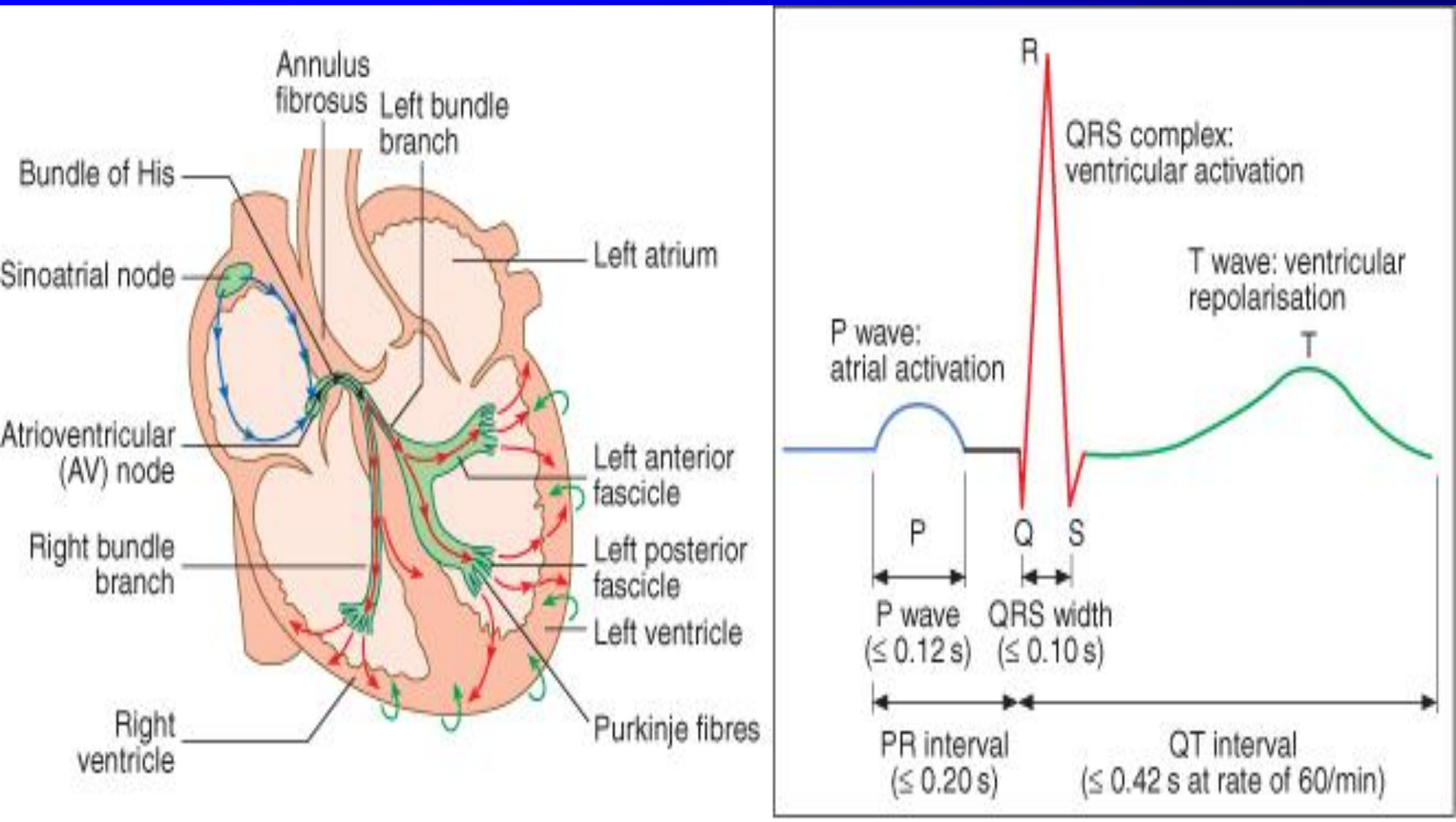
Objective proof of ischaemia
ECG, scintigraphy, echocardiography

Without subjective symptomatology

Diagnostics of chronic IHD

- Exercise tests – ECG, echocardiography, radioisotopes

Differential diagnostics of IHD
Digestive tract
Neurotic origin
Vertebrogenic
Pleural
Dissection of aortal aneurysm



Prevention of IHD

- Primary – prevent the development of IHD
- Secondary – prevent progression of the disease and development of complications
- Treatment of DLP, DM, HT, weight reduction, ban on smoking, healthy nutrition, regular physical activity

Treatment of chronic IHD

- Reduction of risk factors of IHD, change of lifestyle, and regular bodily movement
- Medicinal treatment
- Revascularization of the myocardium

Medicinal treatment

- Antiaggregants – ASA, clopidogrel
- Beta-blockers – anti-ischaemic, antianginal, antiarrhythmic effect
- Blockers of the renin-angiotensin system – ACE inhibitors, sartans (AT1-blockers) – anti-ischaemic, antianginal effect
- Statins

Revascularization

- AP of grades III to IV, grade II - younger, active people
- PCA – 1 to 2 arteries, stent implantation
- Aortocoronary bypass – multiple affliction of coronary arteries

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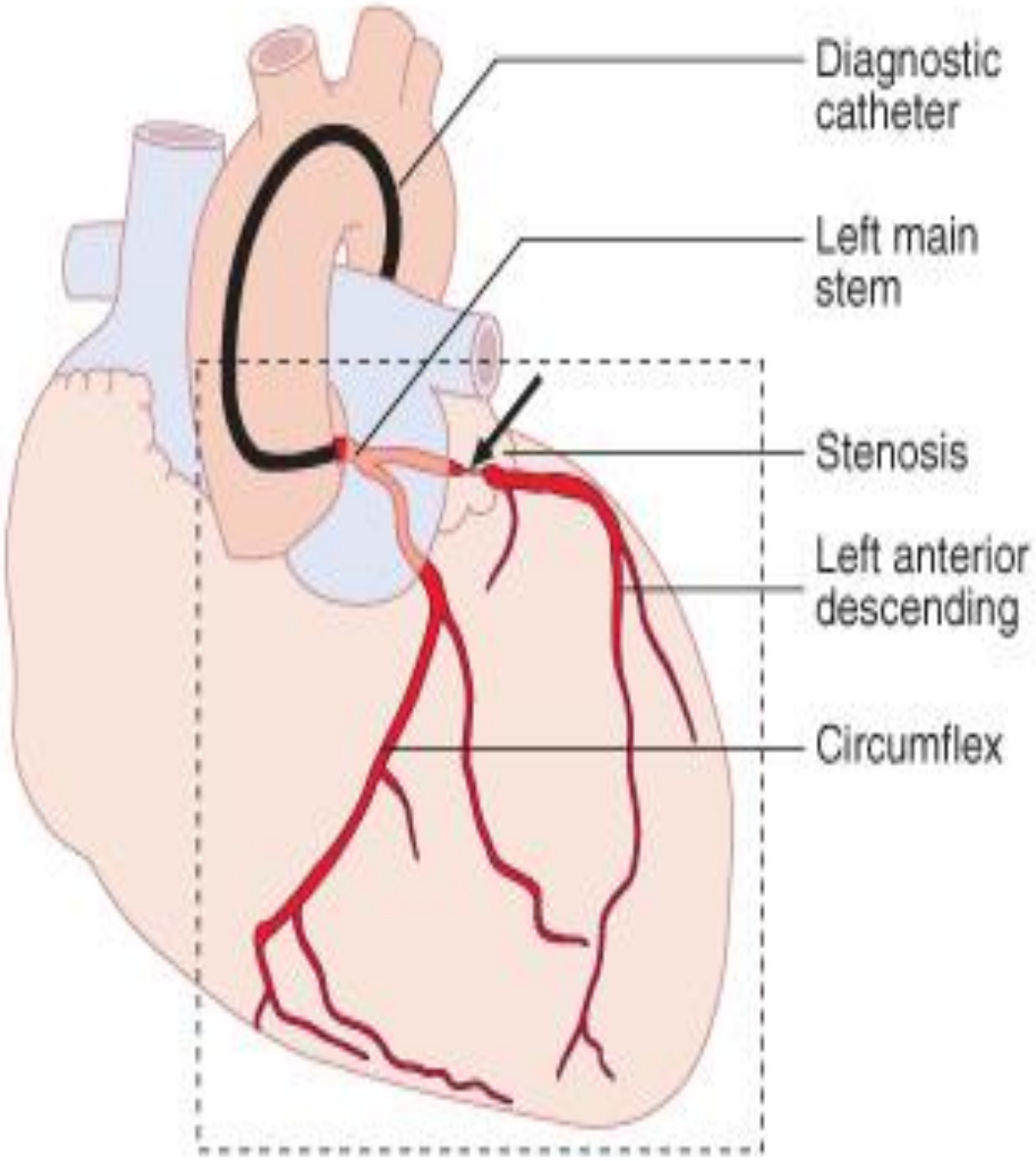

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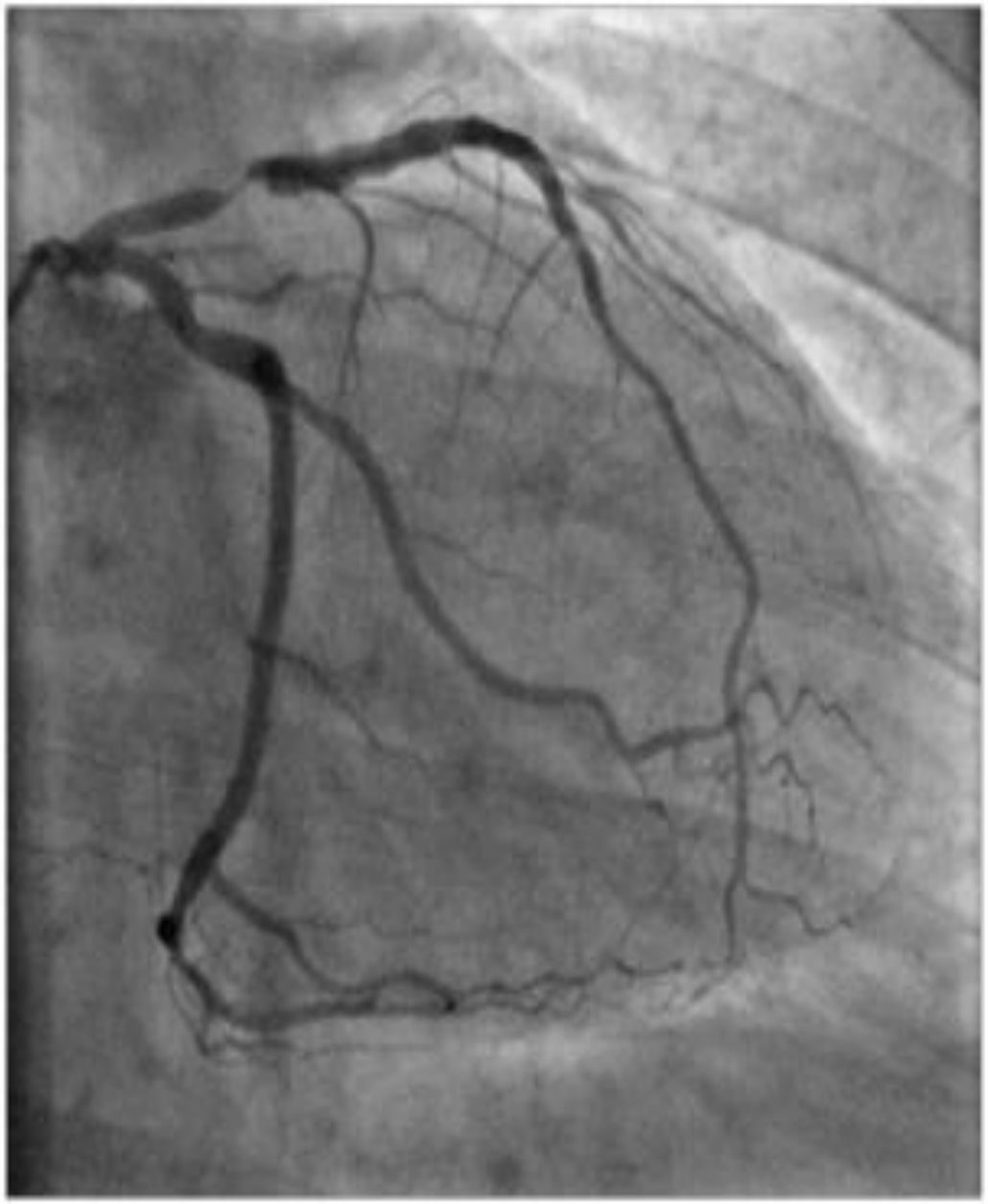
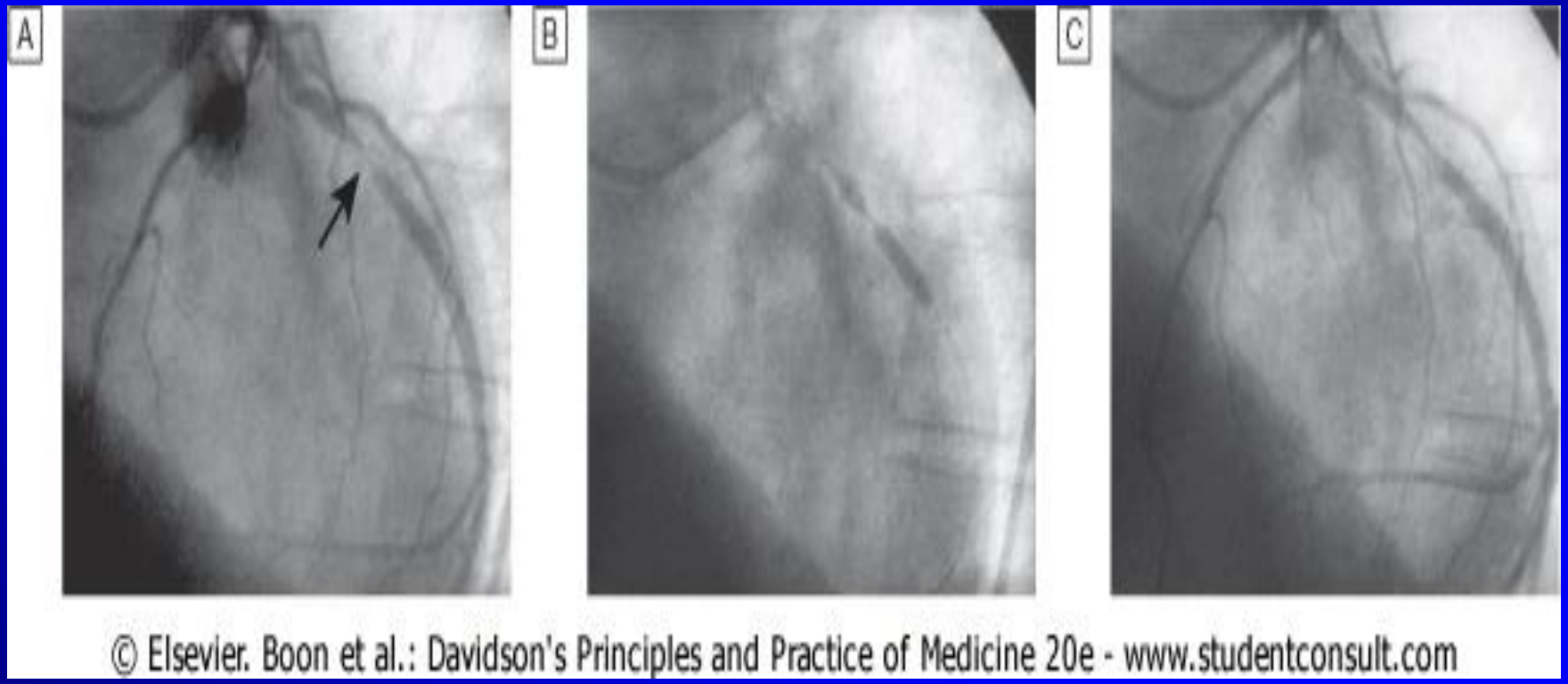
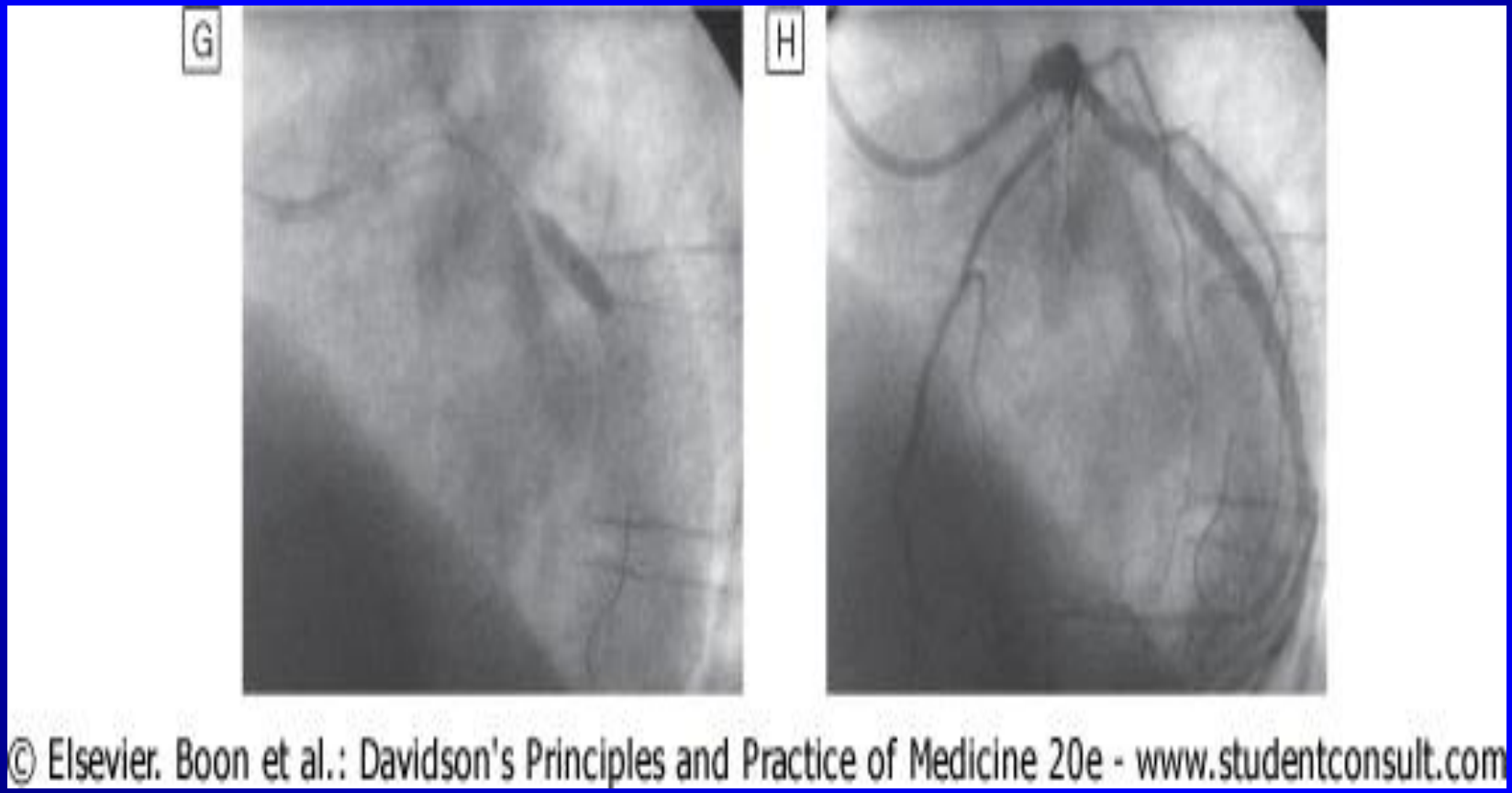
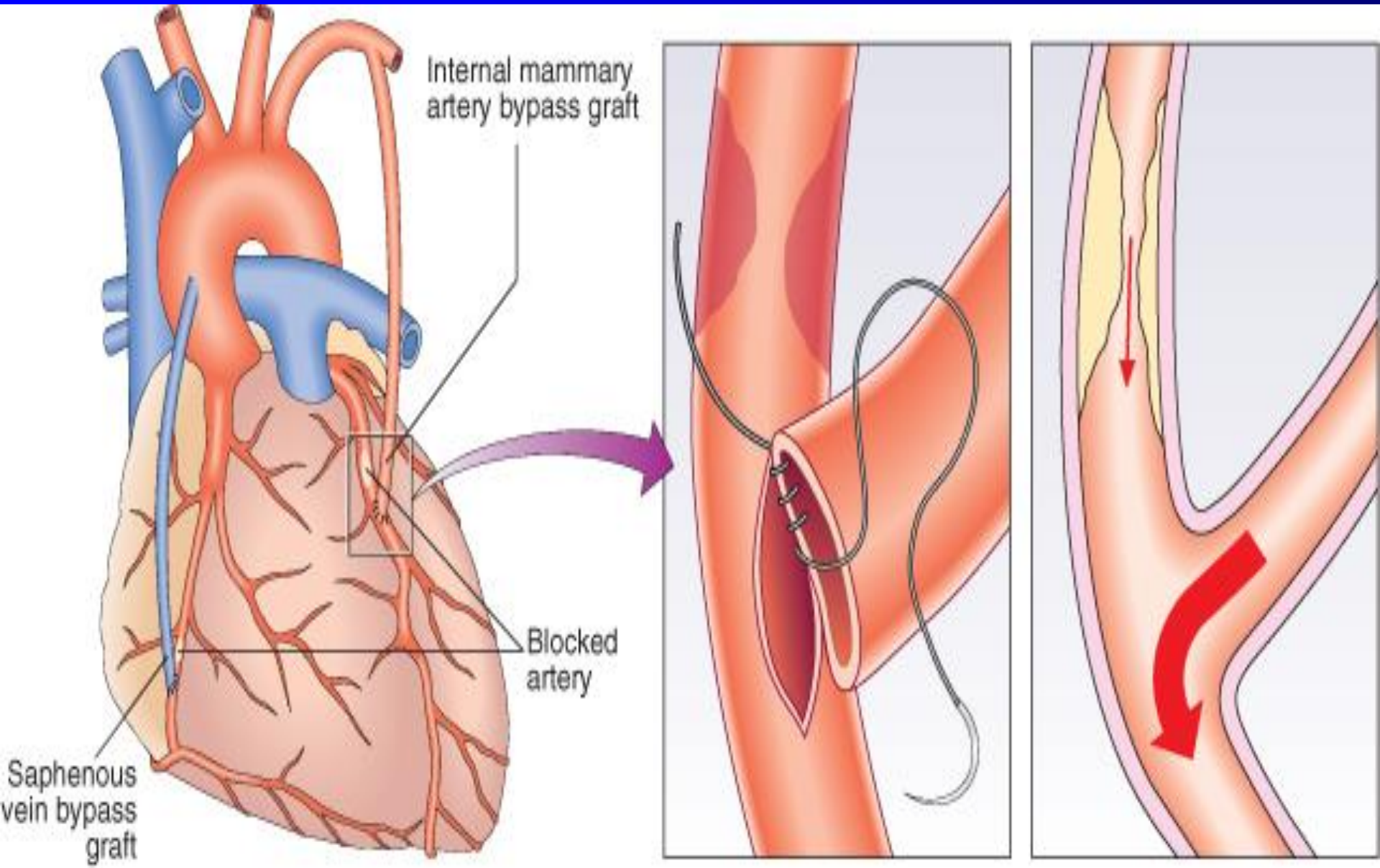








Acute coronary syndrome
Unstable AP

- Definition – disproportion in supply x O_2 utilization
- Unstable atheromatous plaque
- newly developed AP
  AP with sudden deterioration
- rest-associated AP

- Diagnostics – a history of chest pains, slower remission after nitroglycerine, pains are more frequent, of longer duration

Objective finding

- Poor or negative
- Worn out, sweaty, anxious, tachycardia, hypertensive reaction
- Differentiation between unstable AP and MI impossible at physical examination
- Urgent referral to ICU

Diagnostics

- ECG – beyond attack the ECG is normal, state after MI of more recent date, arrhythmia
- During attack – depression or elevation of ST

Laboratory – physiological values

CK, CKMB, AST, ALT

Troponin T and I

Myoglobin

Coronarography

Pharmacological and interventional treatment of unstable AP

- Analgesics including opiates, O_2 inhalation
- Anticoagulants – continuous heparin, low-molecular heparin – nadroparin, enoxaparin, dalteparin
- Antiaggregants – ASA 400 mg, clopidogrel 75 mg
- Nitrates i.v., BB, calcium blockers
- PCA + stent implantation, preferably within 24 hrs.

Acute myocardial infarction (AMI)

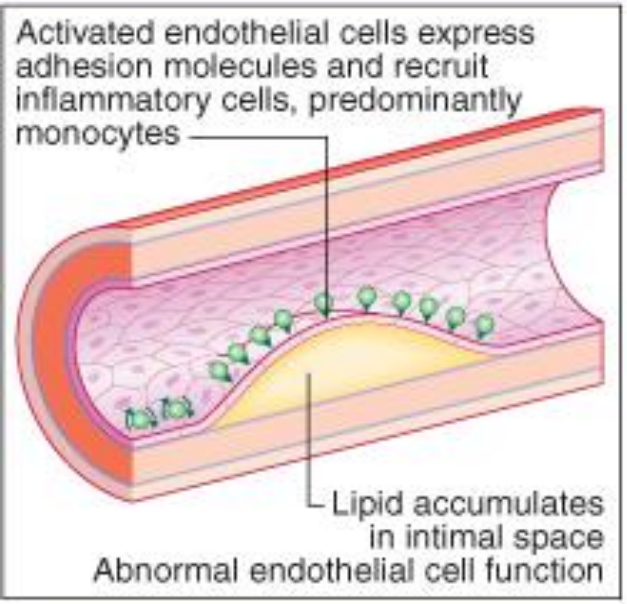
Definition

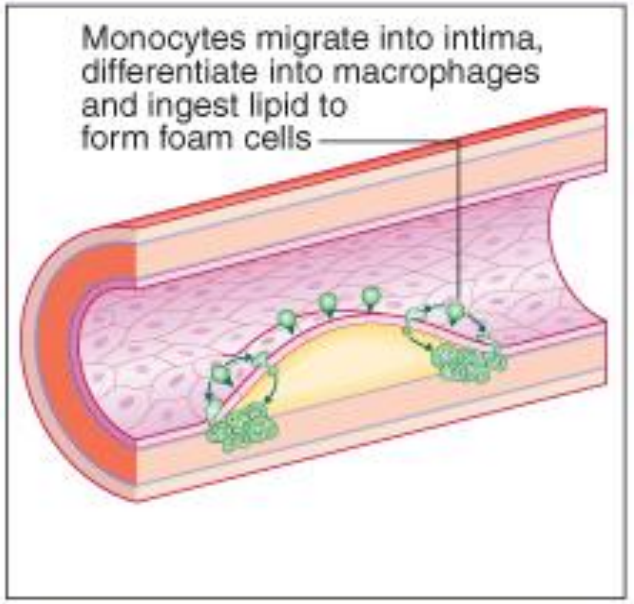
focal necrosis of the myocardium

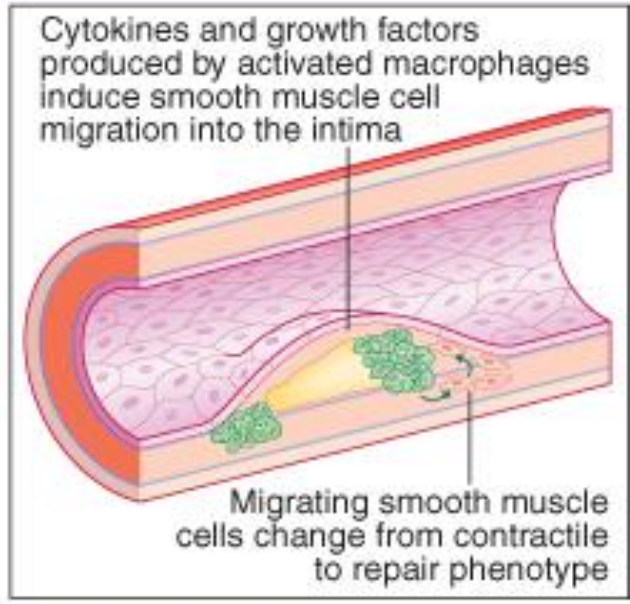
Supply of nutrients, O_2 x metabolic demands of the myocardium

- **Aetiopathogenesis**
- an acute thrombotic occlusion over the rupture of an unstable atherosclerotic plaque
- ECG: AMI with ST elevations and without ST elevations

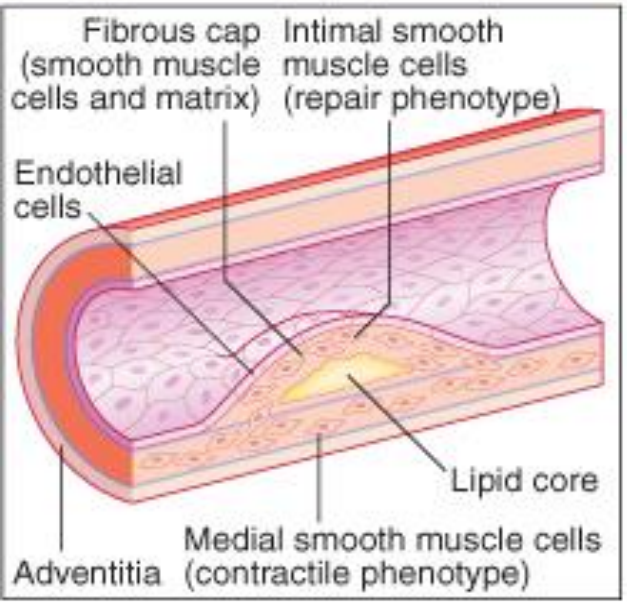
Early atherosclerosis



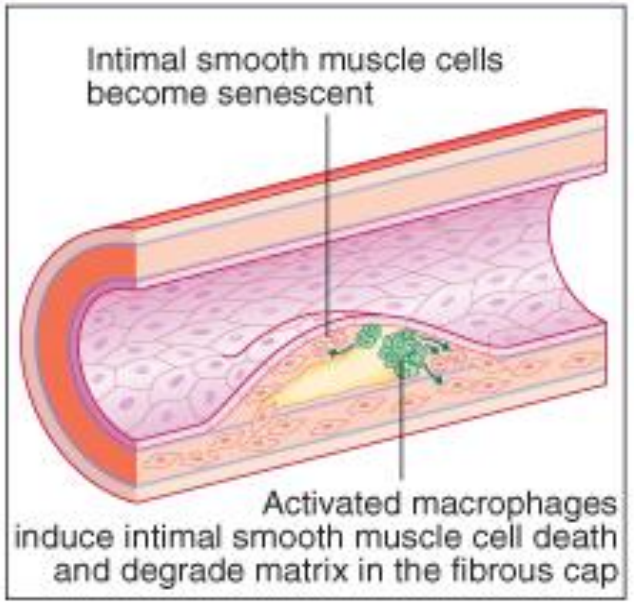




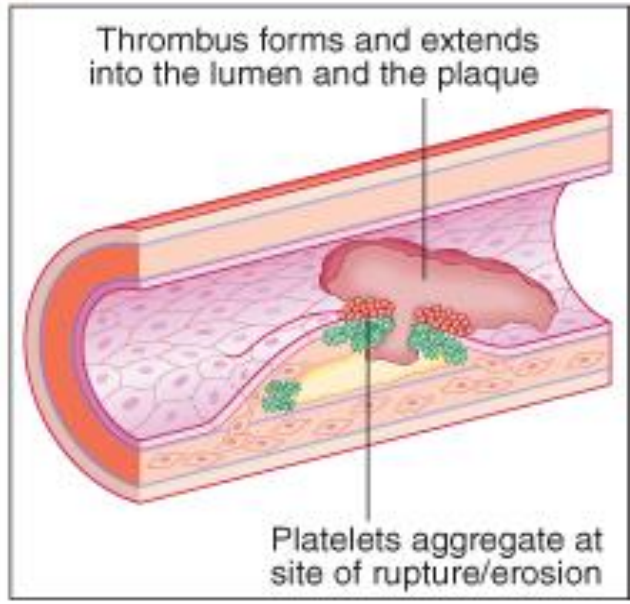
Stable atherosclerotic plaque



Advanced atherosclerosis



Unstable coronary artery disease



Diagnostics of AMI

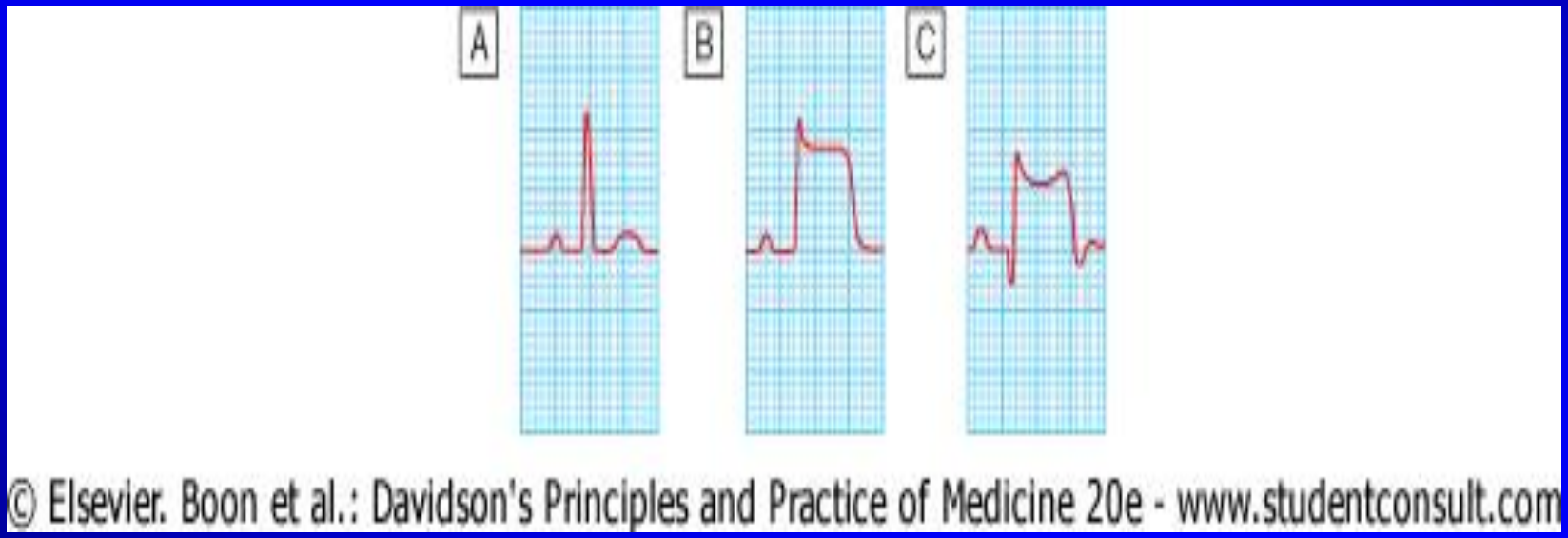
Intense, long-time pain

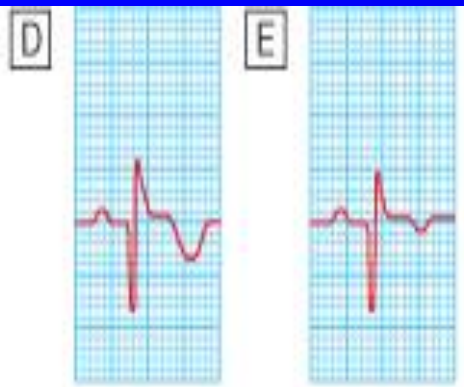
Propagation

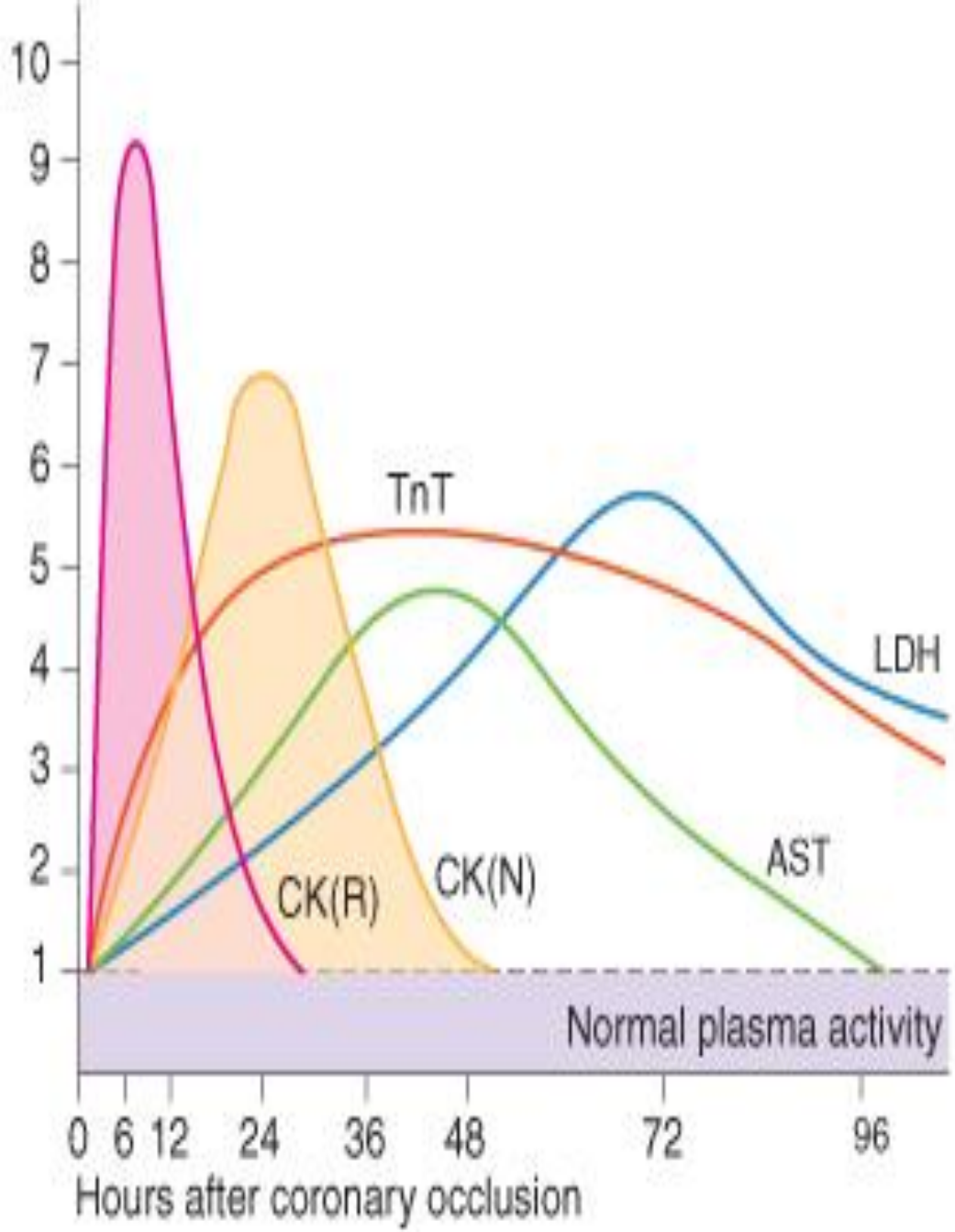
- Sudden, acute onset (60%)
- Sweating
- Tachycardia x bradycardia
- Vagal reaction – nausea, vomiting
- Anxiety

Laboratory

- Increase of cardiac enzymes - TPN,CK,CKMB,AST,LD
- ECG: peaked T, elevation of ST, Pardee wave, a coronary, symmetrically negative T







Differential diagnostics of AMI

Pain

Pulmonary embolization,

aortic dissection,

pneumothorax, pleuritis,

pericarditis,

disease of oesophagus, Tietze sy, vert. alg. sy

Pains from abdominal region

Differential diagnostics of AMI

ECG

Acute pericarditis

Acute myocarditis

Unstable AP

Hyperkalaemia

Chronic block of LBBTw

Pulmonary embolism

Biochemical changes

Myocarditis

Acute heart failure

Pulmonary embolism

Basic examination of AMI

- History
- Physical examination
- ECG
- Biochemical indicators
- Haematological indicators
- X-ray of heart and lungs
- Echocardiography

Treatment of AMI – general principles

Preservation of undamaged myocardium

Minimization of mortality and complications

Reduction of the occurrence of heart failure

Immediate transport to hospital – angioemergency line

Immediate and perfect suppression of pain

Fentanyl s.c., i.v. 2-3 ml

ASA 400-500 mg, nitrates

Treatment of AMI with ST elevations (STEMI)

Prevention of the development of extensive heart necrosis

Shortening of the prehospital phase

Already the first minutes after occlusion are decisive

The critical period lasts for abt. 48 hours

Time division of AMI treatment

Prehospital phase

Short-time acting opiates

Tranquilizers

ASA

Nitrates

Hospital phase

Opening of the occluded artery within 12 hrs. PTCA, PCI

in case of pain

ST elevation of more than 1 mm in 2 leads

New block of Tawara's branch

Primary PCI

Urgent PCI of the infarct artery in 12 hrs.

Without previous thrombolysis

ASA p.o., i.v.,

Clopidogrel 8 tabs à 75 mg

Heparin 5,000 U.

Beta-blockers unless there are contraindications

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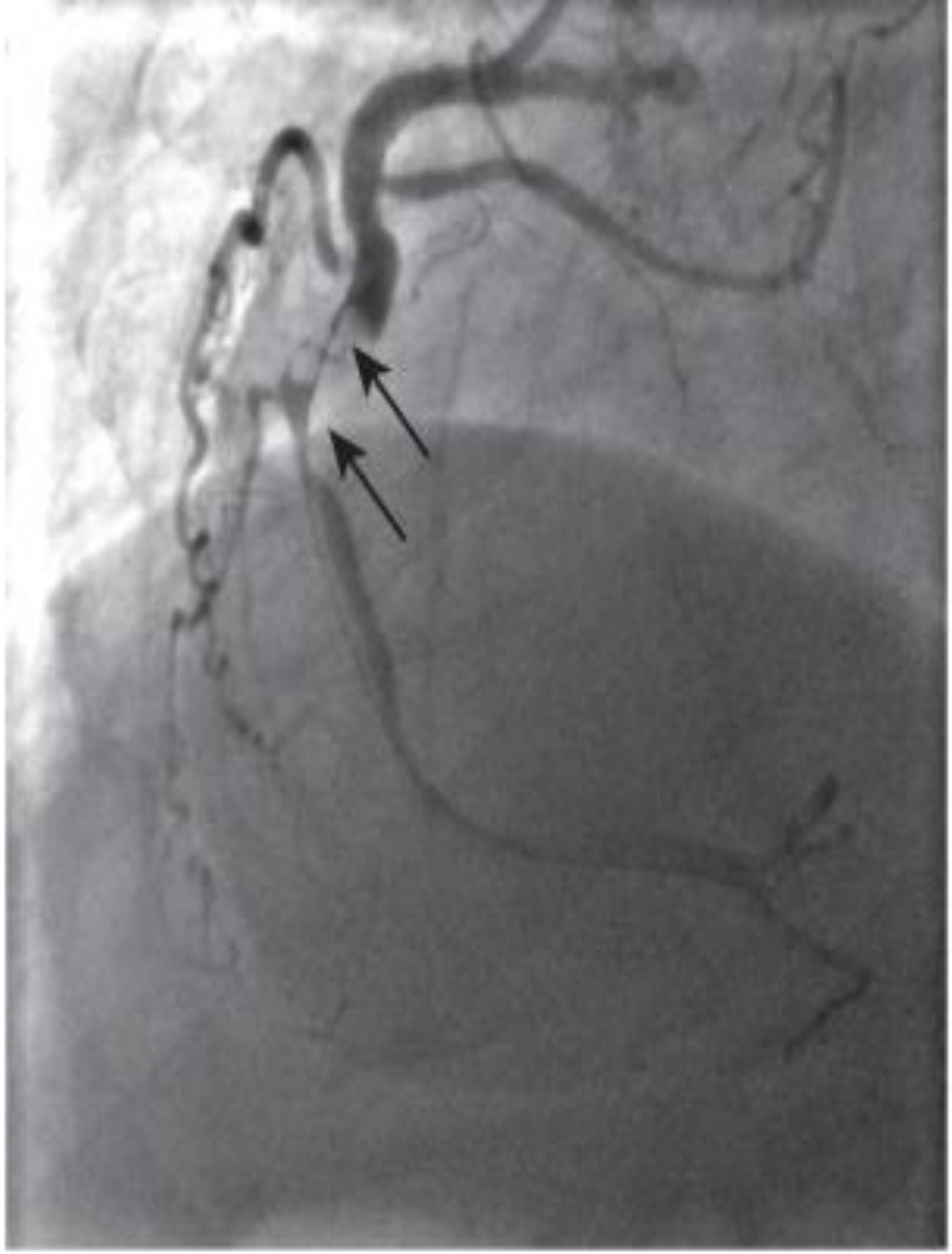

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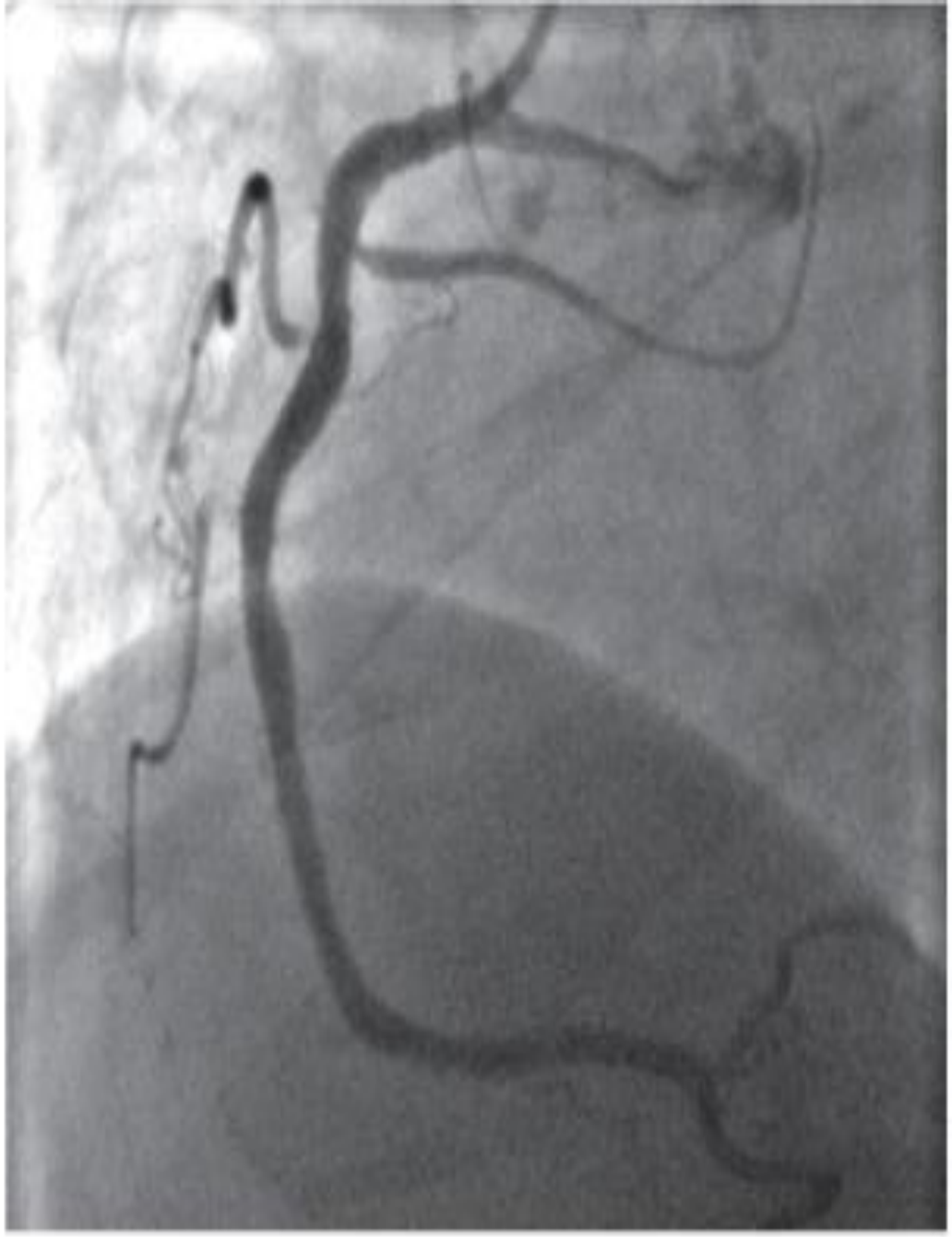


Pharmacotherapy after MI

- ACE-I prevention of LV remodelling
- Beta-blockers
- ASA 100 mg/d
- Clopidogrel 75 mg/d after PCI
- Statins

Complications of AMI

- Arrhythmia - malignant
- Sudden cardiac death
- Heart failure

- Cardiogenic shock
  - Main cause of death
  - LV dysfunction
  - Hypoxaemia
  - Lactic acidosis
  - Drop of BP under 90 mmHg, tachycardia
  - Peripheral vasoconstriction, cold sweat
  - Drop of diuresis

Complications of AMI

- Aneurysm of the heart wall – in abt. 20 to 30%
- Mural thrombi
- Acute pulmonary embolism
- Systemic embolisms
- Rupture and dysfunction of papillary muscles of the mitral valve
- Rupture of the ventricular septum
- Rupture of the free wall of LV – cardiac tamponade
- Pericarditis epistenocardiaca – in 15 %
- Postinfarction AP

After discharge from hospital

Rehabilitation

Early mobilization

Regular bodily activity, especially aerobic exercise

Secondary prevention

- ASA, clopidogrel
- ACE-I, AT1-blockers
- Beta-blockers
- Statins

Painless forms of IHD

Heart failure

Definition

Impairment of ventricular function –

- haemodynamic, neuroendocrine, and renal picture

Congestive heart failure – cardiac insufficiency + venous congestion in the pulmonary or systemic bloodstream

Causes: IHD, HT, CMP, heart defects, cor pulmonale

Acute heart failure

Causes

AMI

Acute rupture of papillary muscles

Rupture of aortic valve

Acute myocarditis

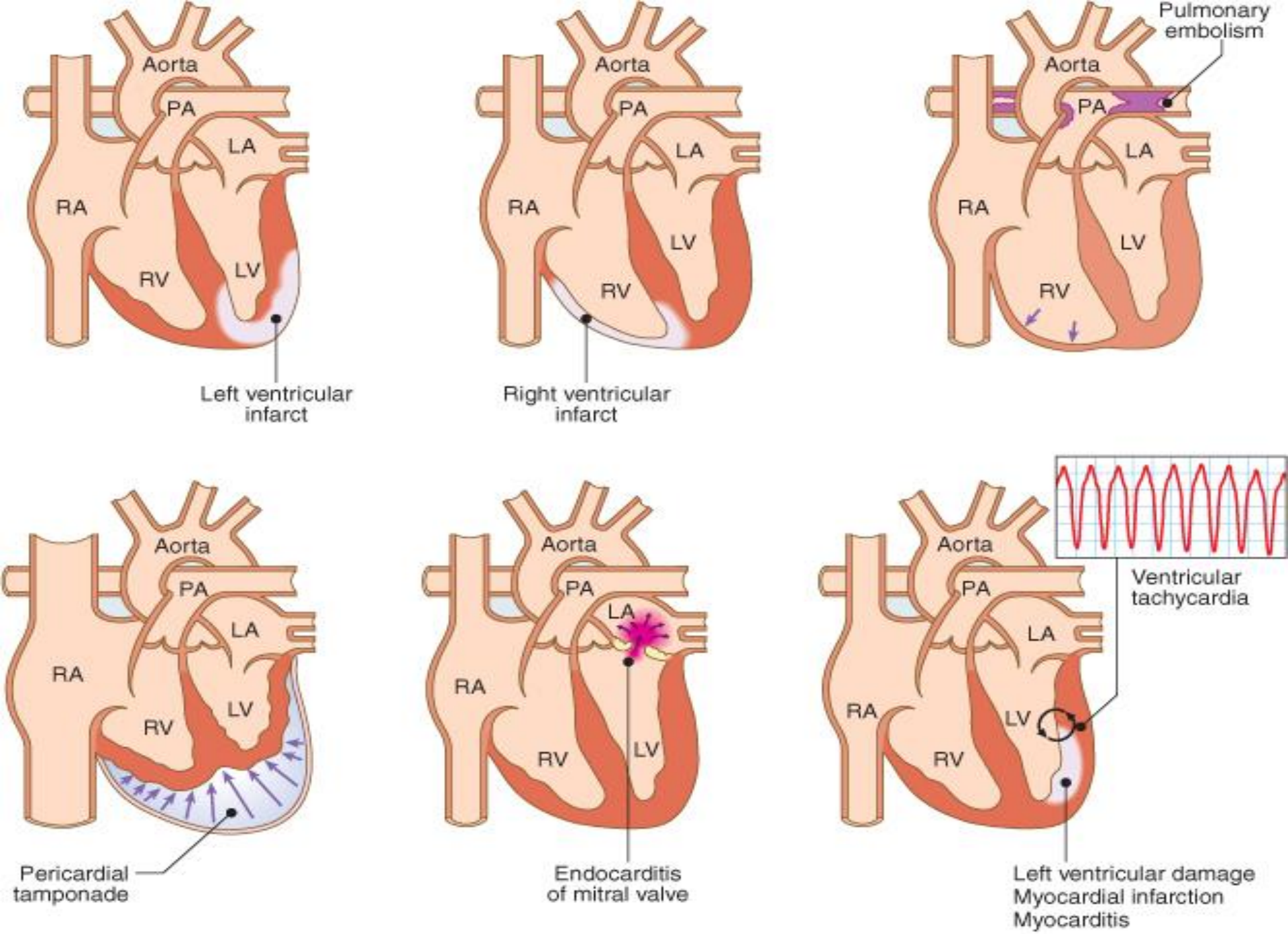
LA failure – mitral stenosis

Treatment of pulmonary oedema 1

AMI –PCI, thrombolysis

Tachyarrhythmia – electrotherapy, amiodaron, digoxin

Bradycardia – ipatropium, temporary cardiostimulation



Treatment of pulmonary oedema 2

Hypertensive crisis – decrease of BP

180-160/95-100 mm Hg - nitrates, furosemide, captopril

Suppression of the respiratory centre – morphine 3 to10 mg

Inhalation of 100% O_2 6-8 l/min

PEEP

Decrease of venous return – in a sitting position, nitrates, furosemide,

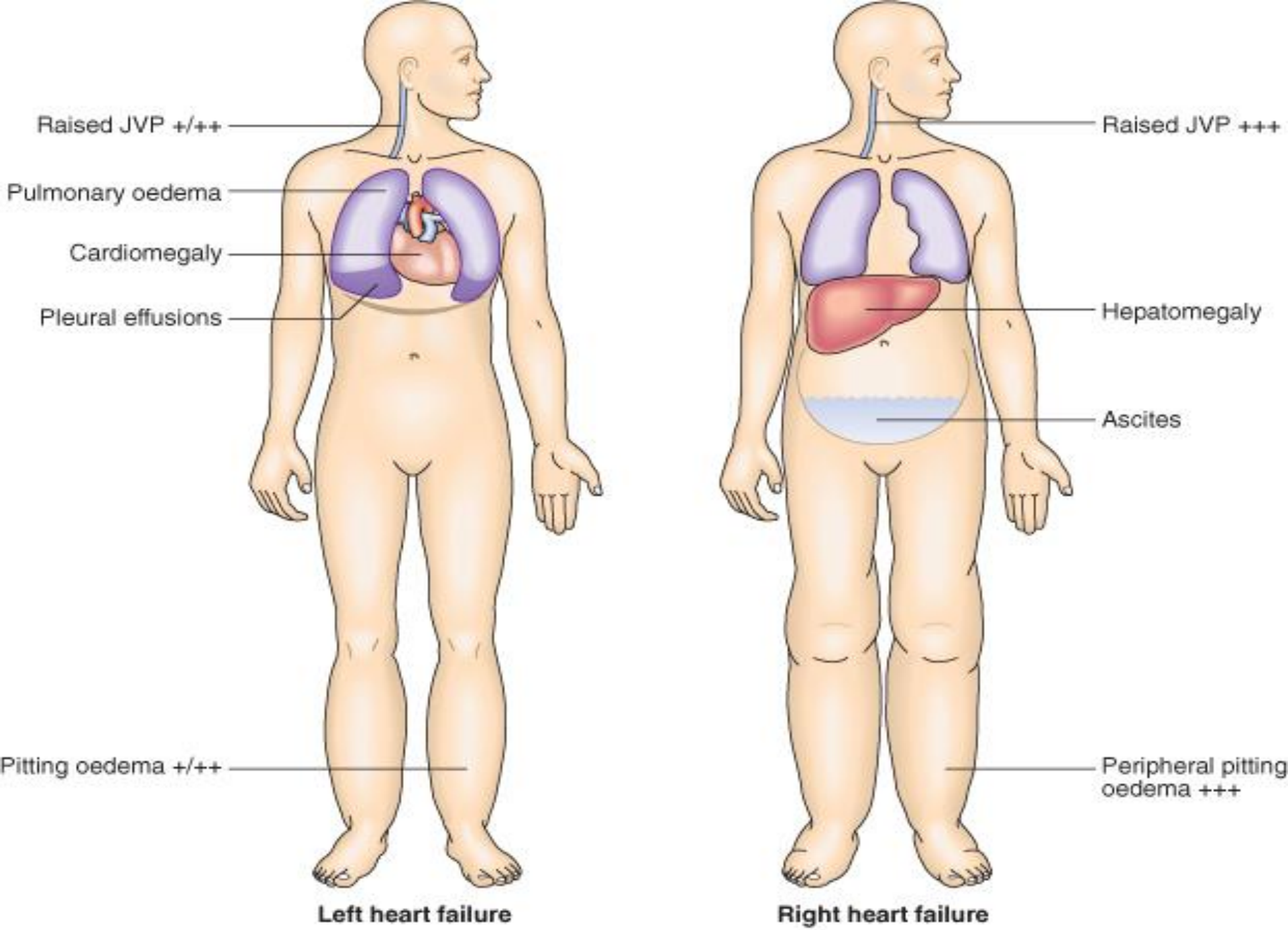
Dobutamine – positively inotropic

Noradrenaline –hypotension

Levosimendan – a calcium sensitizer

Chronic heart failure (CHF)

- Symptoms of heart failure (rest, exertion)
- Impaired function of the heart – systolic, diastolic (rest, exertion)
- Response to treatment



Left heart failure

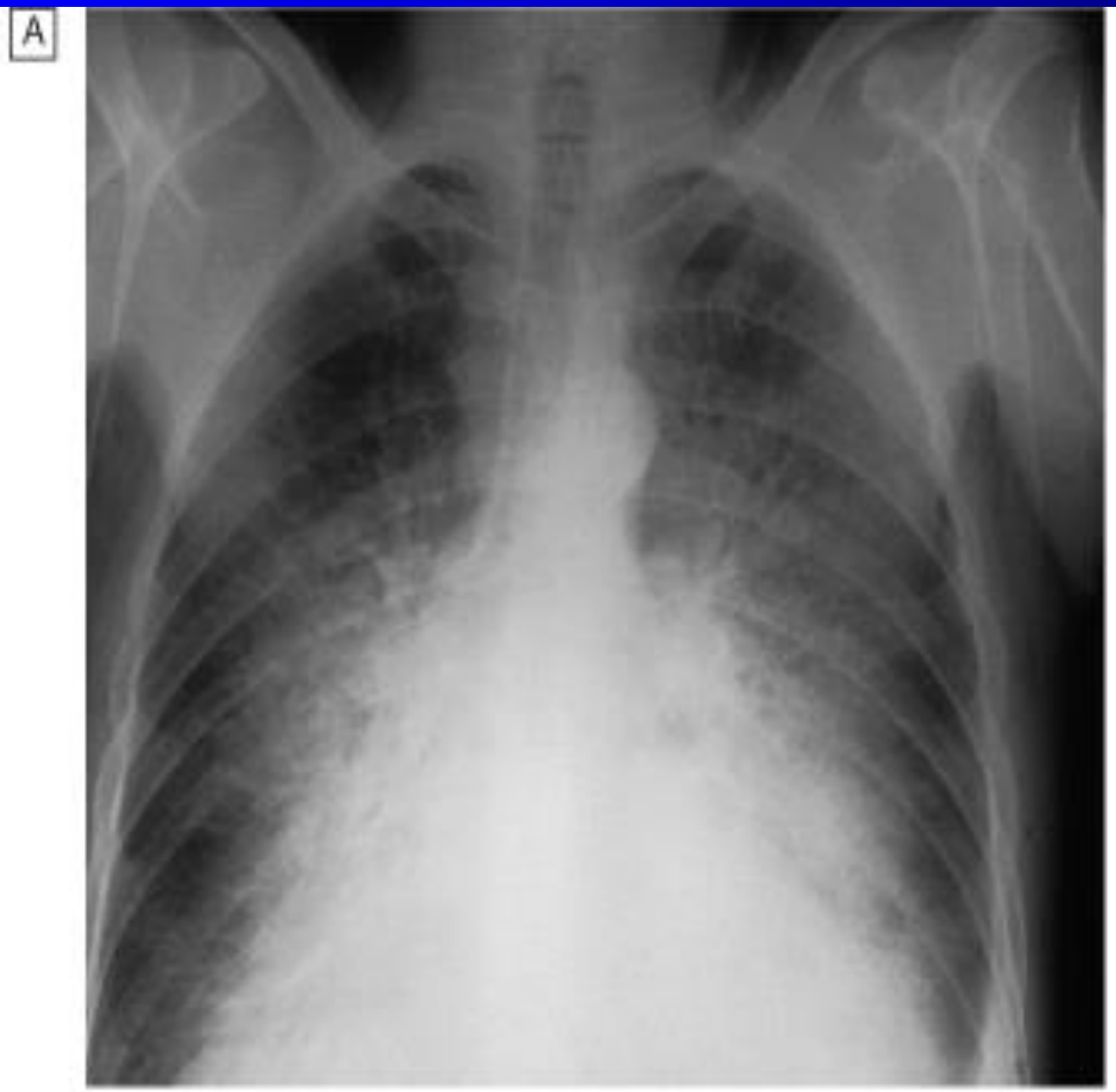
Right heart failure

CHF – subjective symptoms

- Dyspnoea – NYHA grades 1 – 4
- Fatigue
- Oedemas

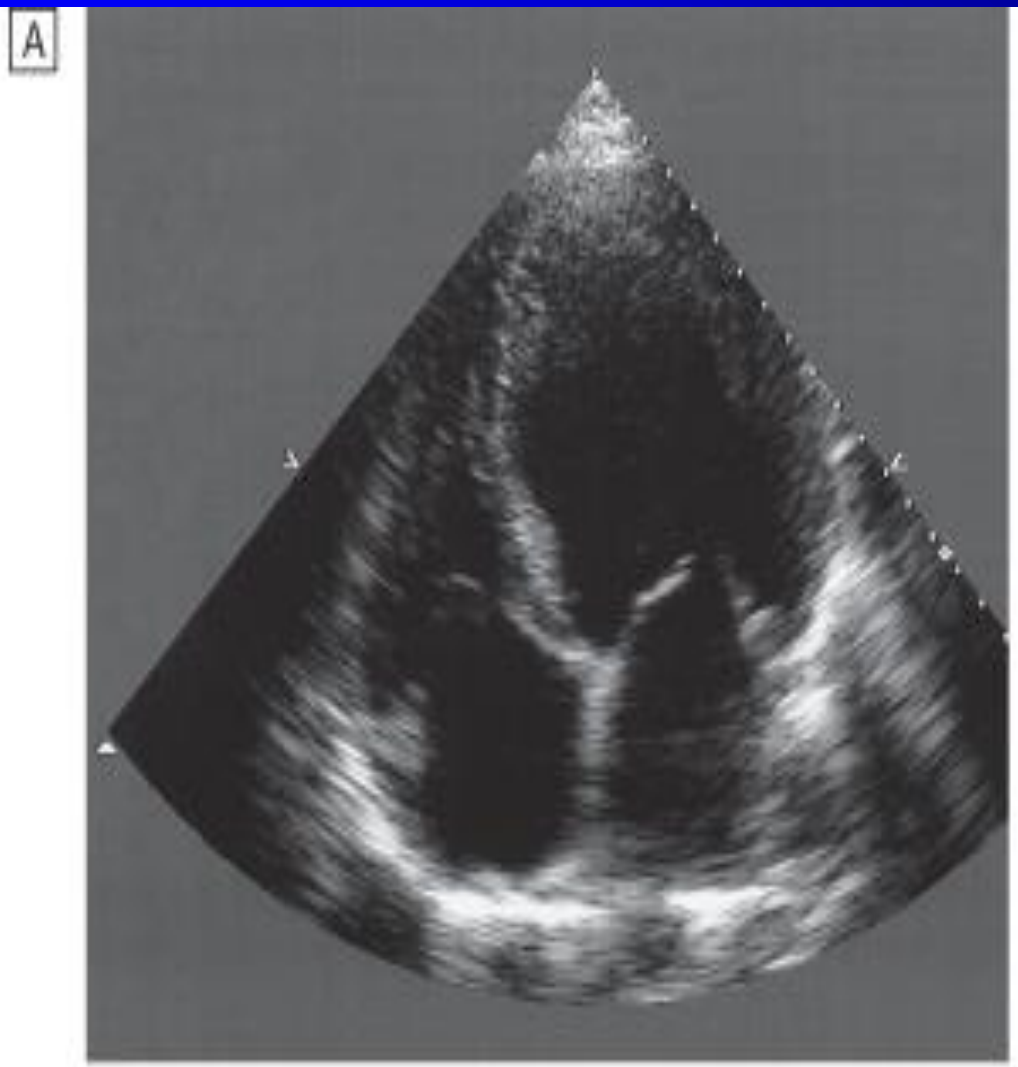
CHF – examination 1

- Physical – the heart: arrhythmia, gallop, murmurs; the lungs: non-accentuated crepitations, hydrothorax, system signs of hepatomegaly, oedemas
- XR – enlargement of the heart shadow, congestion
- ECG – state after MI, hypertrophy and overload of LV, blocks of Tawara branches, arrhythmia, ST-T non-specific changes



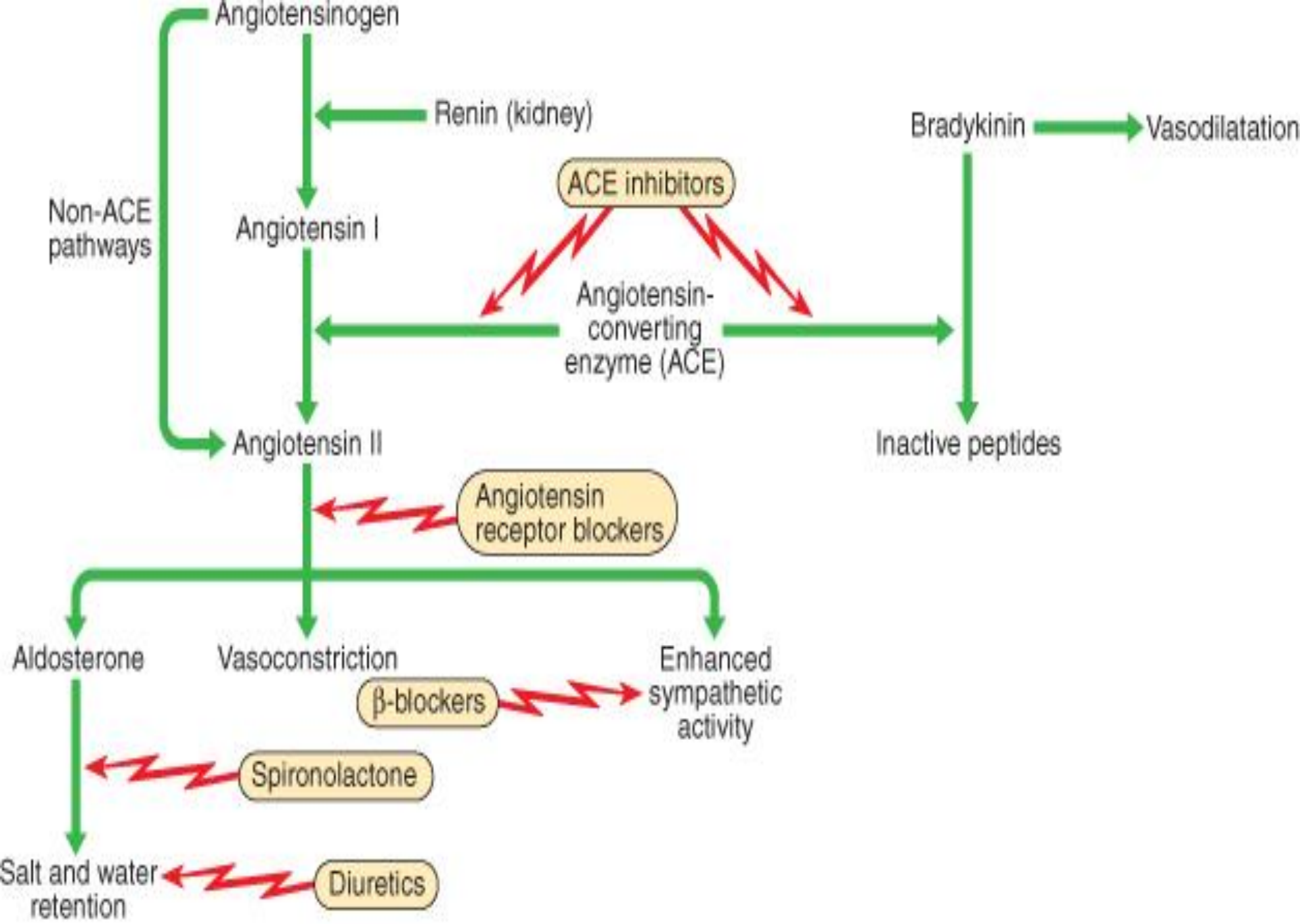
CHF – examination 2

- Echocardiography – systolic function (EF p = from 40%), diastolic function (E/A in transmitral flow rate under 0.9)
- Laboratory – anaemia, renal insufficiency, raised hepatic tests, Nt-proBNP (in acute failure over 900 pg/l, in CHF over 300 pg/l)



CHF – pharmacotherapy

- ACE-I , AT1 blockers – asymptomatic systolic dysfunction of LV, EF under 40%
- Beta-blockers
- Diuretics – thiazide d., loop d., spironolactone
- Digoxin



CHF – non-pharmacological and antiarrhythmic treatment

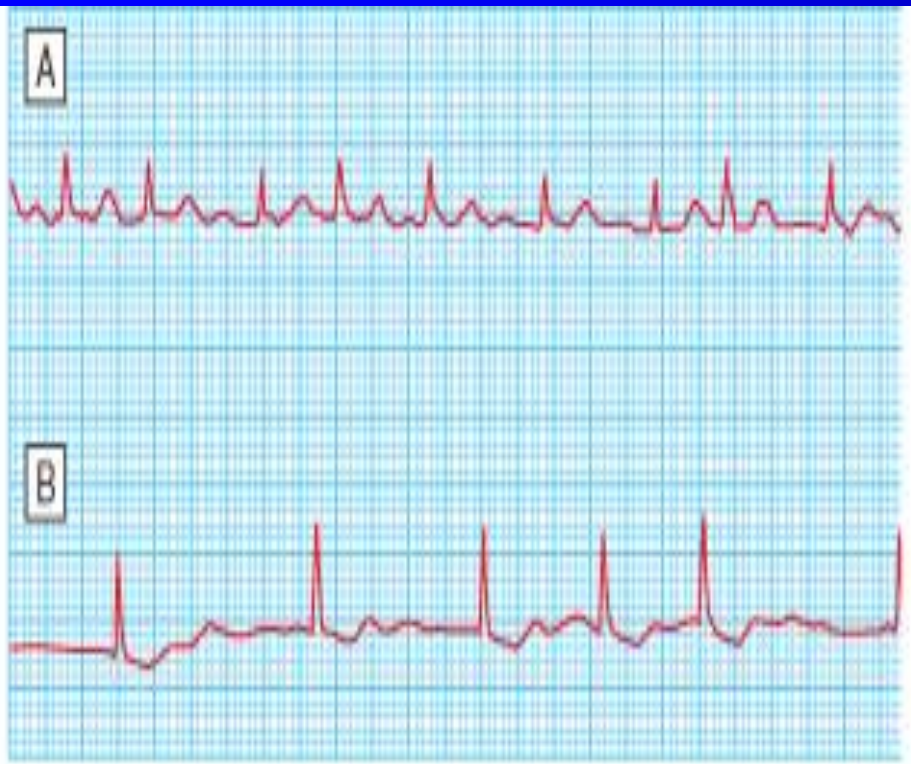
- Permanent cardiostimulation
- Implantable cardioverter-defibrillator (ICD)
- Resynchronization treatment by biventricular stimulation
- Orthotopic heart transplantation (OHT) – treatment of the terminal stages of CHF (NYHA grades III-IV), EF under 20%, adverse prognosis

Arrhythmia - classification

- Speed – bradycardia, tachycardia
- Mechanism – a disturbance in impulse production and/or conduction
- Site of origin – supraventricular a., re-entry a., ventricular a.

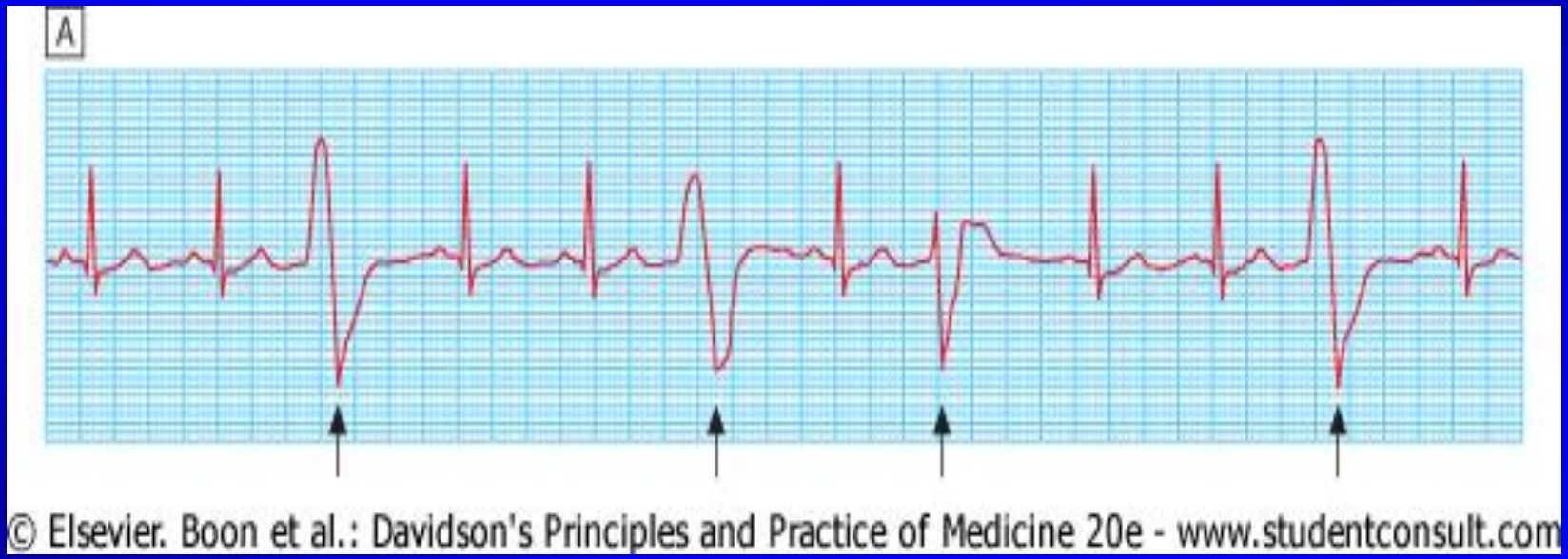
Supraventricular tachycardiac arrhythmias

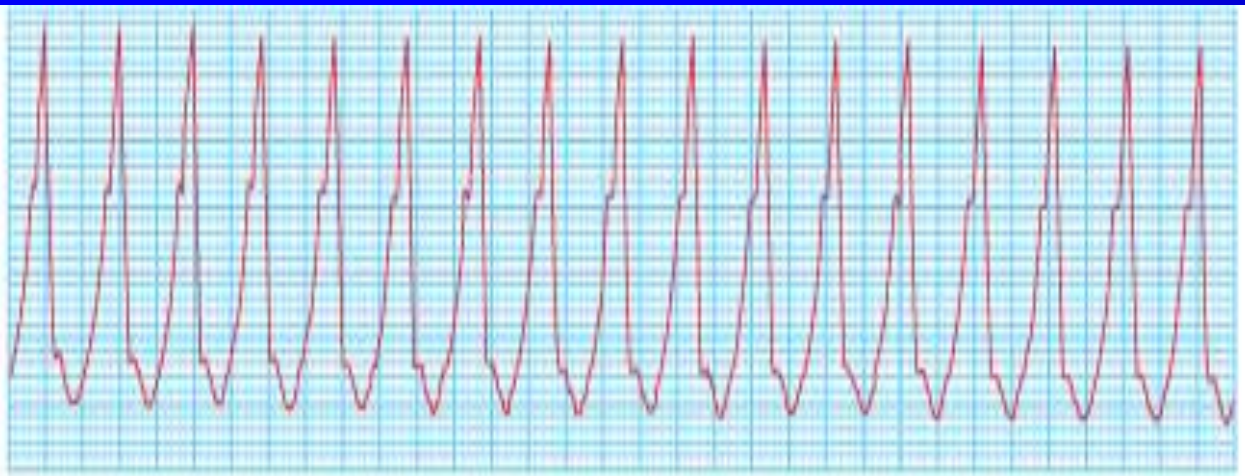
- Sinus tachycardia – over 100/min
- Atrial fibrillation – irregular chaotic atrial activity; P waves are missing
- Flutter of the atria – characteristic P waves
- Supraventricular tachycardia
- Dysfunction of the sinus node (sick sinus syndrome - SSS)

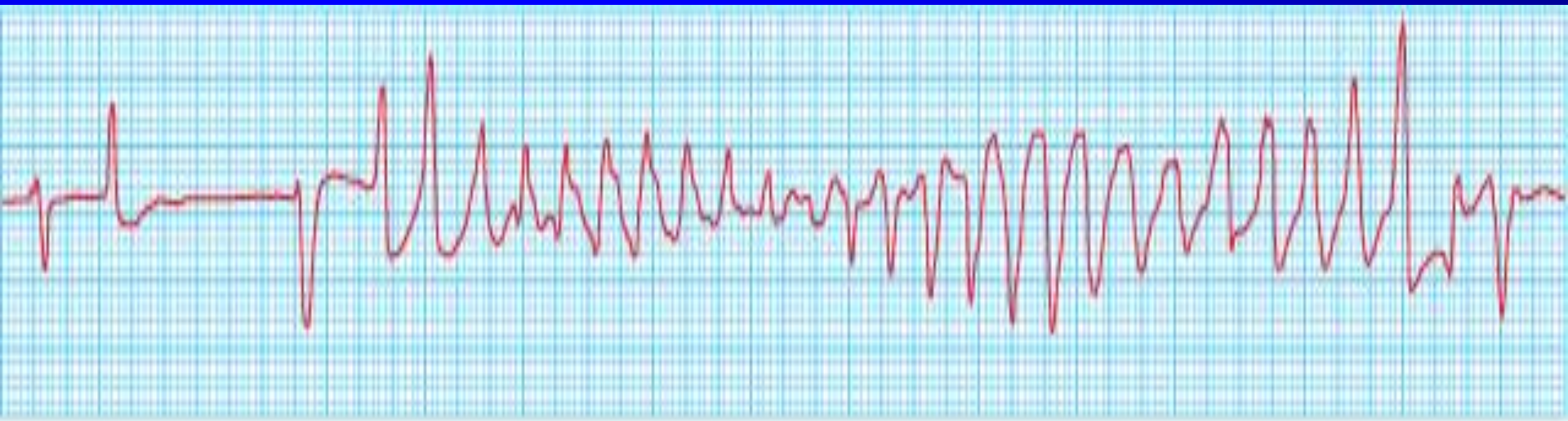


Ventricular arrhythmia

- Malignant arrhythmia
- Ventricular tachycardia
- Ventricular flutter
- Ventricular fibrillation – unconsciousness
- Early ventricular extrasystolae – R/T
- MAS-syncope (Morgagni-Adams-Stokes s.)

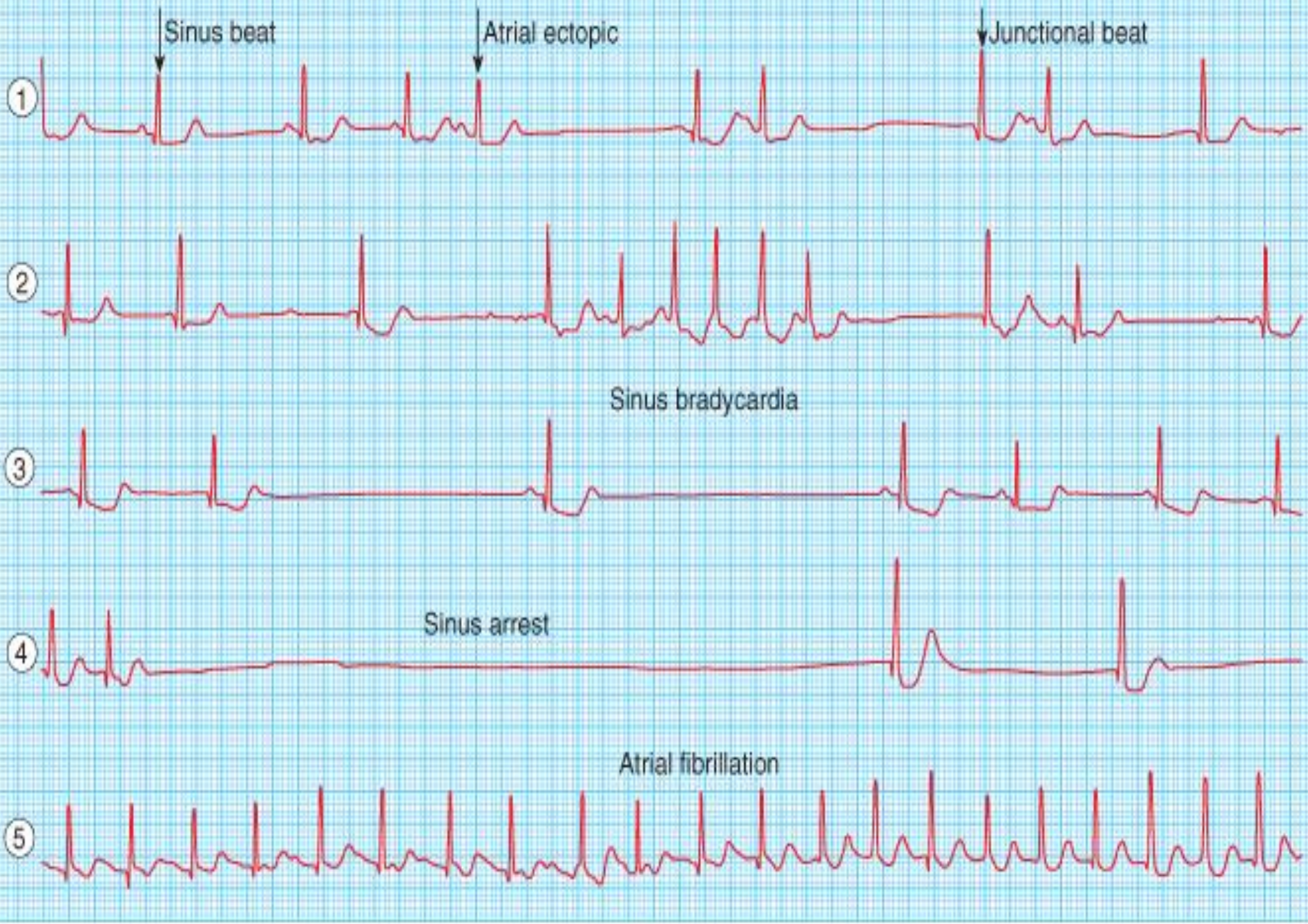






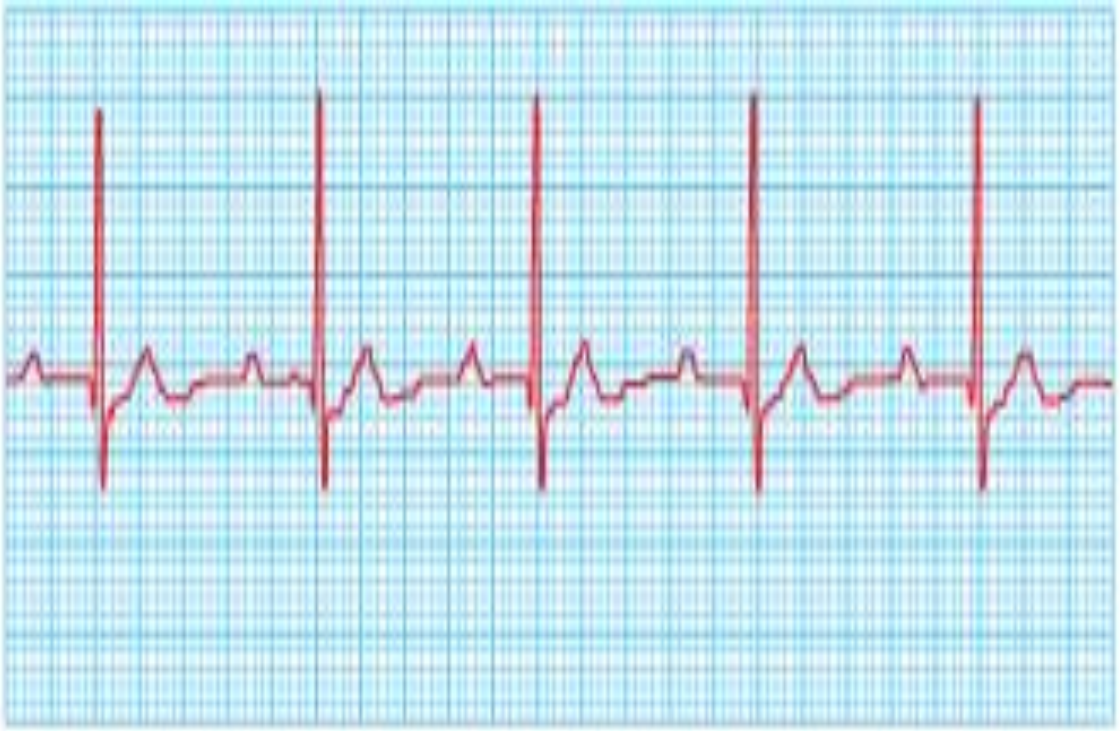
Bradycardia

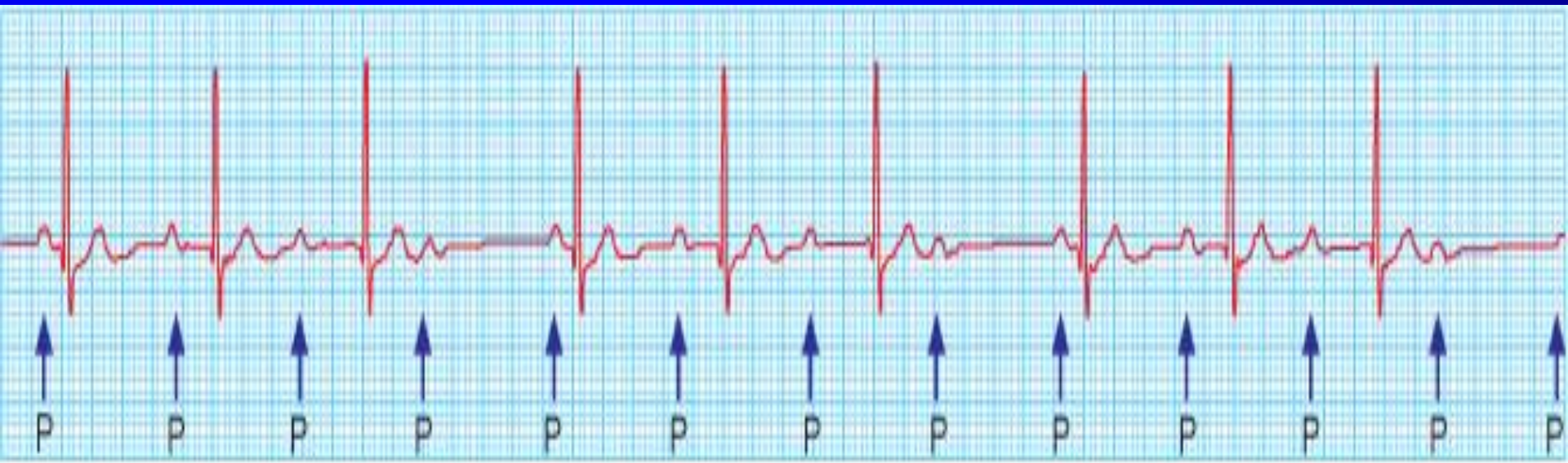
- Pulse rate under 50/min, severe under 40/min
- Sinus bradycardia
- Sinoatrial arrest – P-P is prolonged
- Atrioventricular junctional rhythm – 35 to 50/min, no P waves, QRS is slim
- Atrioventricular dissociation – rhythm from AV junction
- Idioventricular rhythm – broad QRS, 25-35/min

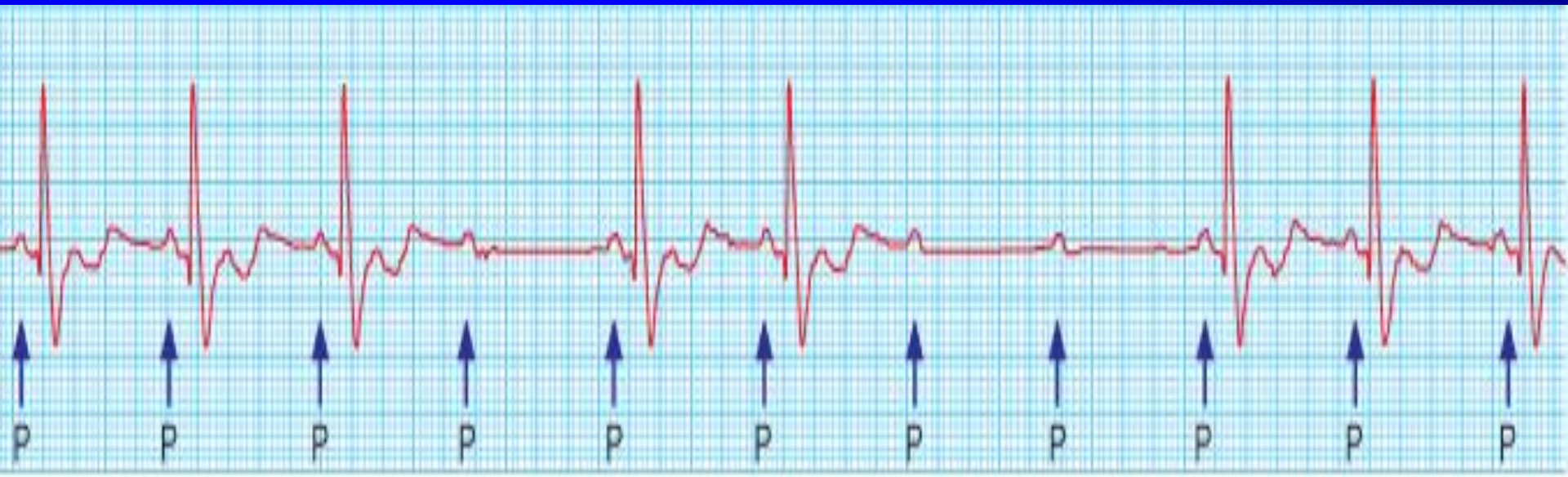


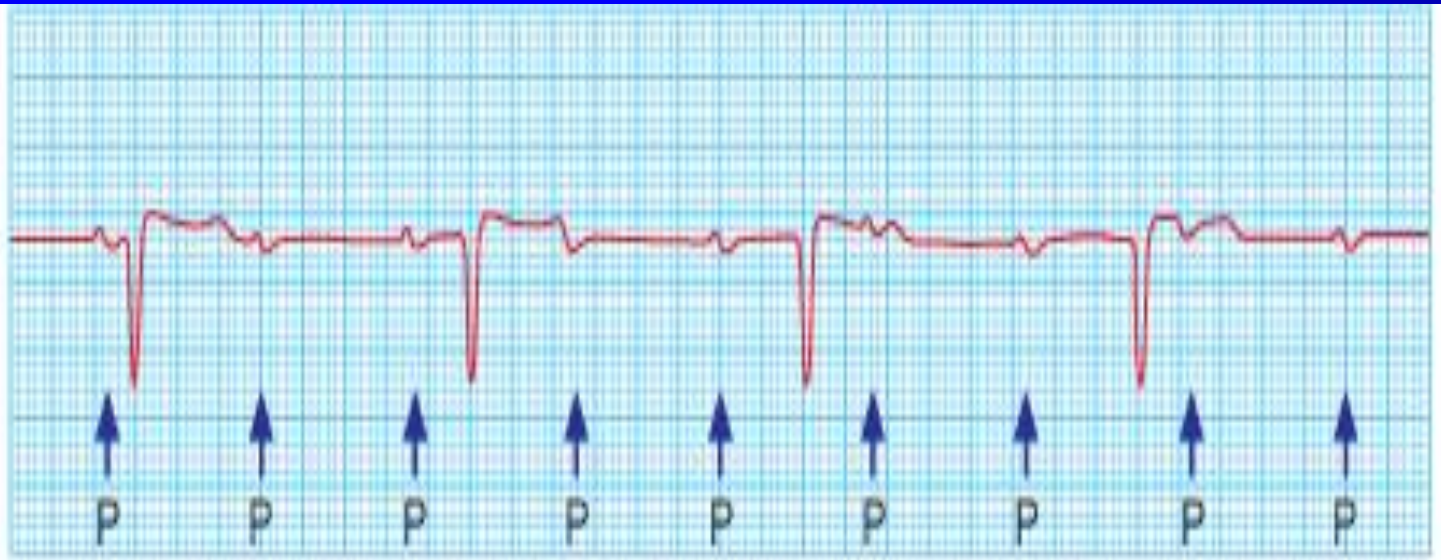
Arrhythmias from a conduction impairment

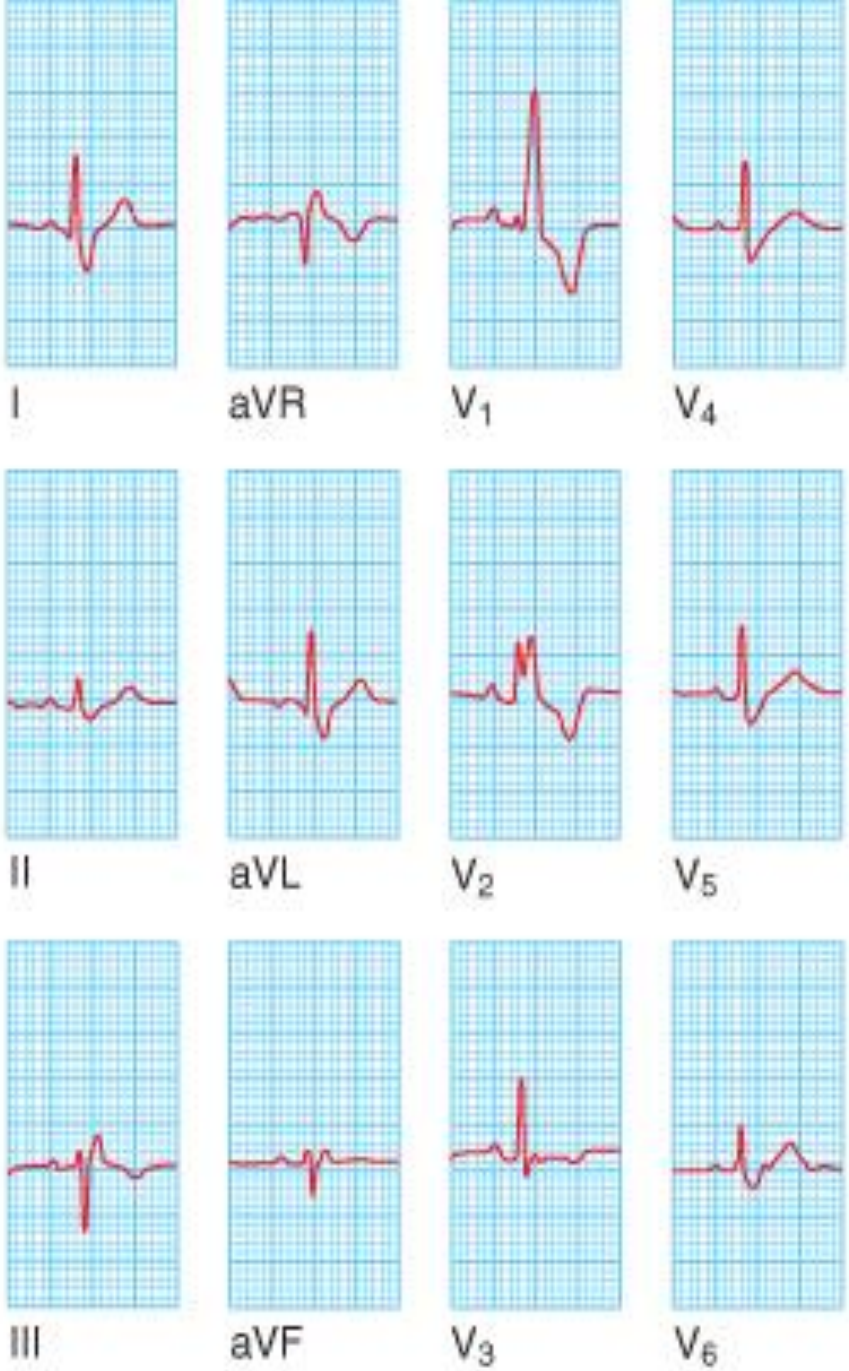
- Bundle branch blocks
- AV blocks:
- 1st degree: PQ at 0.20 s
- 2nd degree: gradual prolongation of PQ, drop-out of QRS
- 3rd degree: broad QRS 25 – 30/min

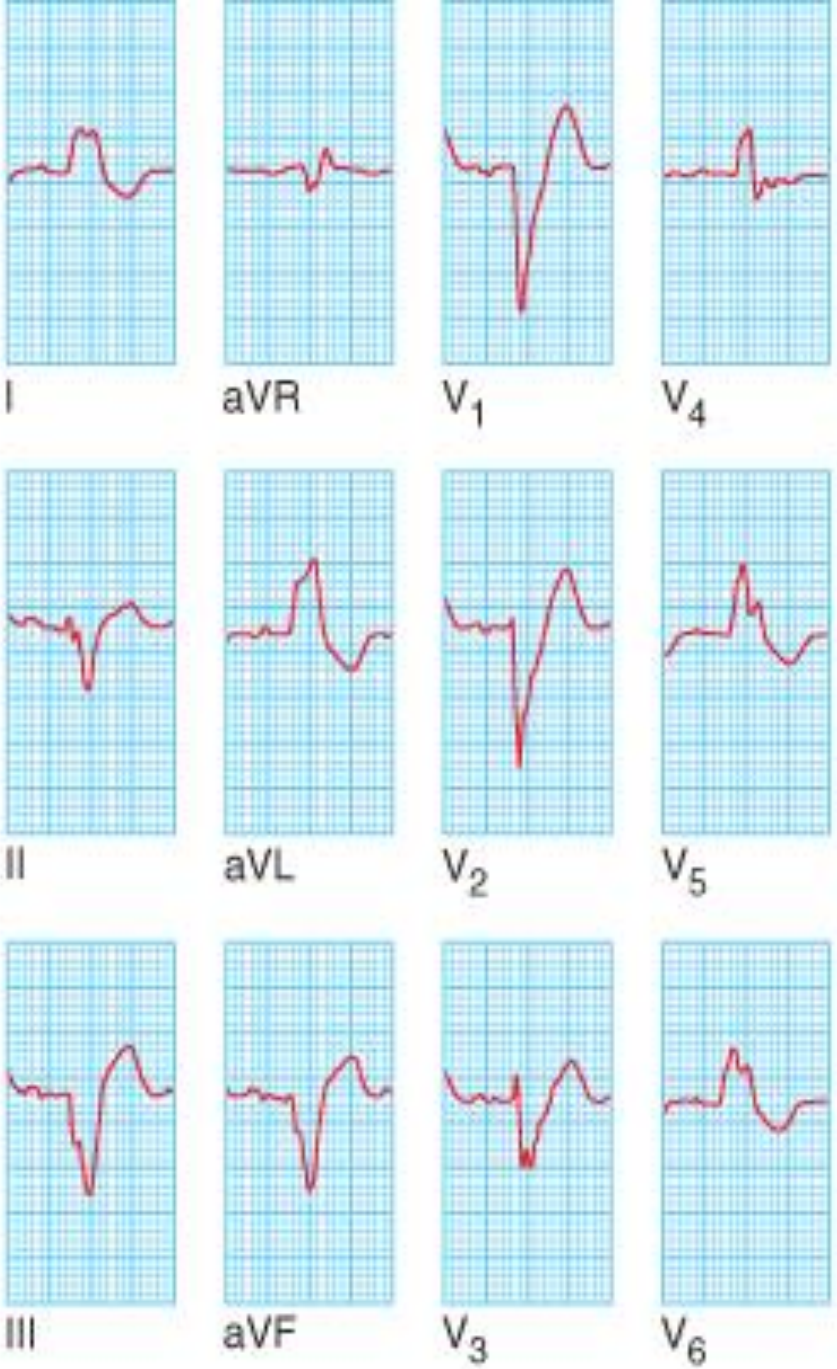












Arrhythmia – clinical picture

- Palpitations
- Dizziness
- Syncope
- Dyspnoea
- Faintness, inefficiency
- Chest pain

Arrhythmia - diagnostics

- History, physical examination
- ECG at rest
- Holter monitoring of ECG for 24-48 hrs.
- Massage of carotid sinuses with the recording of ECG – positive test – pauses above 3000 ms
- Electrophysiological examination

Arrhythmia – treatment of bradyarrhythmias

- **Aim** – suppression of symptoms, of unfavourable haemodynamic condition, improvement of prognosis
- **Pharmacological treatment** – atropine, ipratropin
- **Non-pharmacological treatment** – temporary and permanent cardiostimulation

  Pacemaker (PM) – ventricular p., atrial p., dual chamber p., biventricular p.

Arterial hypertension (HT) – prevalence, definition, classification

Prevalence – in total 20-50%, Czech Rep. (25-64 yrs.) – 35%

RF – IHD, CMP

HT – repeated increase of BP 2x over 140/90

Essential (primary) **HT** – 90%

Secondary HT

I. Simple increase of BP

II. Organic changes

III. Hypertension with severe organic changes + failure of function

HT classification by BP value in mm Hg

Category	Systolic BP	Diastolic BP
Optimal	under 120	under 80
Normal	120-129	80-84
High normal	130-139	85-89
Stage 1 HT (moderate)	140-159	90-99
Stage 2 HT (medium severe)	160-179	100-109
Stage 3 HT (severe)	180 and higher	110 and higher
Isolated systolic HT	140 and higher	under 90

HT – endothelial dysfunction

- **Mechanical effects** – increased BP, turbulent bloodstream
- **Biochemical effects** – increased A II, catecholamines, lipoproteins, smoking

HT – endothelial dysfunction

- Increased adherence and subendothelial migration of thrombocytes and monocytes
- Increased permeability for plasma components including lipoproteins
- Multiplication of local plasmatic factors with growth or modulatory effects
- Proliferation of the smooth muscles of blood vessels, storage of lipoproteins

Essential HT - pathogenesis

- **Genetic share** – polygenic type with little expressivity
- **Outer environment –** raised NaCl, insufficient supply of K, Ca, Mg?, excessive supply of food, increased consumption of alcohol, stress
- **Endogenous influences –** central and sympathoadrenal nervous system + vasoconstrictor and dilatatory humoral factors

HT - prognosis

- Height of BP – achieved during treatment
- Presence of other RFs
- Damage to target organs
- Presence of associated diseases

HT - prognosis

Cardiovascular RFs

- Stage of HT
- Age + sex – males over 55, females over 65
- Total cholesterol value
- Smoking
- DM , abdominal obesity
- Raised CRP

HT - prognosis

Damage to target organs

- **Hypertrophy of LV** – ECG, echo
- **Thickening of arterial wall** – sonography
- **Increase in serum creatinine** (males 115-130 umol/l, females 107-124 umol/l)
- **Microalbuminuria** – 30-300 mg/24 hr
- **Narrowing of renal arteries –** local or generalized

HT - prognosis

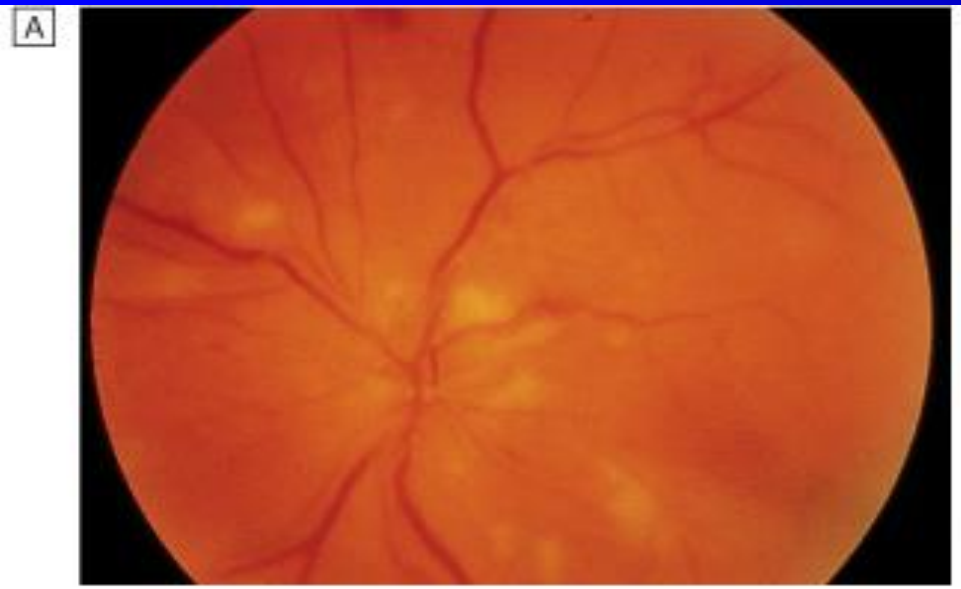
Associated diseases

- **Cerebrovascular diseases** – ischaemic CMP, cerebral haemorrhage, TIA
- **Heart affection** – MI, AP, bypass, heart failure
- **Renal failure** – diabetic and non-diabetic nephropathy with CHRI – creatinine in males over 133 and in females over 124 umol/l, proteinuria over 300 mg/24 hr

HT - prognosis

Associated diseases

- **Vascular affection –** dissecting aortal aneurysm, ischaemic disease of lower extremities
- **Advanced retinopathy –** haemorrhage or exudates, papillary oedema



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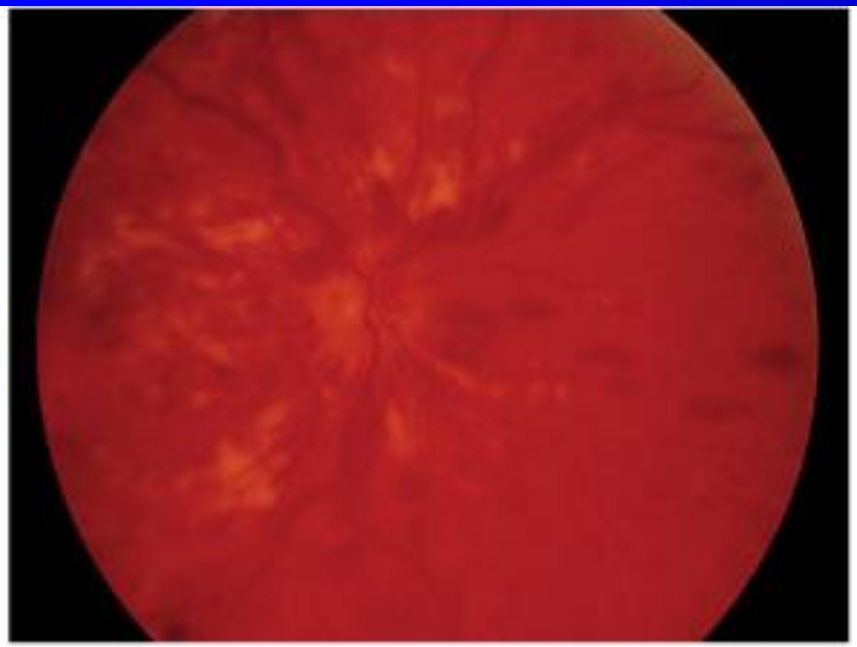


HT – clinical picture

- Stage 1: asymptomatic, headache, preoccupation, palpitation, lack of concentration, sleep disorders
- Stage 2: subjective signs similar, objective – hypertrophy of LV, microalbuminuria
- Stage 3: impairment of organic function – increasing dyspnoea during heart failure, dilatation of LV, coronary affection, hypertensive retinopathy to neuroretinopathy, cerebrovascular disease, CHRI

HT – hypertensive crisis

Acute state in any stage of HT

- **Marked rise in BP –** BPd 130-140 mm Hg
- **Rapid progressing changes in target organs –** HT encephalopathy, retino- to neuroretinopathy, failure of LV, possibility of aortal dissection, RI
- **Emergency or urgent cases** - ICU

HT - diagnostics

- **BP measured in an outpatient dept.** – 140/90 and higher
- **BP monitored over 24 hr -** 125/80 and higher
- **BP measured in domiciliary conditions** 135/85 and higher

HT - examination

Necessary in all hypertonics	Suitable in some of the groups
History incl. family h., gynaecol. h.	Echocardiography
Physical examination incl. peripheral arteries	Sonography of carotids, femoral arteries
BP in sitting and standing position, on both upper limbs	Microalbuminuria (necessary in DM)
Urine + sedimentation	Proteinuria quantitatively
Na, K, creatinine, glycaemia, uric acid in serum	Eyeground in severe HT Central BP
Lipid spectrum- total CH,HDL,TG,LDL	
ECG	

Secondary HT- suspicion

- Persons under 30 years with marked diastolic HT, in persons over 30 years with BPd over 130 mm Hg
- Resistant HT – BP above the norm at triplex combination of antihypertensives incl. diuretics
- Sudden worsening of HT, organic changes do not correspond to the length of HT
- Abnormalities in laboratory, ECG, echocg results

Secondary HT - causes

Renal c.

- **Parenchymatous –** glomerulonephritides, diabetic nephropathies, interstitial nephrities, polycystosis, HT after transplantation, obstructive uropathy, hydronephrosis
- **Renovascular –** stenoses of renal arteries, vasculitides, aneurysms, thromboses
- **Renin-producing renal tumours**

Secondary HT - causes

Endocrine c. – overproduction of pressor factors

- Pheochromocytoma – overproduction of catecholamines
- Conn sy – overproduction of aldosterone
- Cushing sy – overproduction of cortisol
- Adrenogenital sy – overproduction of deoxycorticosterone

Secondary HT - causes

Drug-induced HT

- Glucocorticoids
- Steroidal contraceptives with high content of oestrogens
- Non-steroidal antiphlogistics (NSA)
- Cocaine abuse

Secondary HT - causes

HT in pregnancy

- **Continuation of essential HT**
- **HT originated in the first months of pregnancy**
- **Pregnancy gestosis** in the last trimester – oedemas, proteinuria (more than 0.3 g/l), HT (over 140/90 mm Hg, BPs by over 30 mm Hg, BPd by over 15 mm Hg)

Secondary HT - causes

HT in sleep apnoea syndrome

HT after transplantation of organs

Aortic coarctation – HT in the upper half of the body

Neurogenic causes of HT – brain tumour, trauma, expansive inflammatory diseases, cerebral haemorrhage

HT in cardiosurgical interventions

HT – non-pharmacological treatment

- **Reduction of body mass**
- **Reduction of salt supply** – 5-6 g/d
- **Reduction of excessive alcohol consumption** – males up to 30 g/d, females up to 20 g/d
- **Quitting smoking, reduction of stressful situations**
- **Regular physical activity** – 30-45 min 3-4x/week
- **Increased consumption of fruit and vegetables, reduction of intake of saturated fats**
- **Reduction in drugs supporting retention of Na and water –** NSA, corticoids, steroidal contraceptives
-

HT – pharmacotherapy

- **BP 180/110 mm Hg and higher**
- **BP 150/95 mm Hg and higher** following 4 weeks of non-pharmacological treatment in hypertonics without associated diseases and damage to target organs
- **High normal BP (130-139/85-89) –** damage to target organs, associated diseases, DM, more than 3 RFs

HT – pharmacotherapy

- Diuretics
- Beta blockers (BB)
- ACE inhibitors (ACE-I)
- Long-term acting calcium channel blockers (CaB)
- Angiotensin II receptor antagonists (AT1-blockers)
- Substances with central and central/peripheral effects

HT – treatment - diuretics

- **Thiazide saluretics –** hydrochlorothiazide
- **D's with a lower natriuretic and a higher vasodilatory effect –** indapamide
- **Loop d's –** furosemide
- **Potassium-sparing d's –** amiloride, spironolactone
- **Combined diuretics:** HCHT + amiloride

HT – treatment - BB

- **Non-selective –** metipranolol
- **Cardioselective – Beta 1 blockers:** metoprolol, bisoprolol
- **Non-selective with ISA –** bopindolol, pindolol
- **Cardioselective with ISA –** acebutolol
- **BB of 3. generations – highly beta 1 selective + vasodilatation (NO production, positive metabolic effects)-** nebivolol
- **Combined AB and BB –** labetalol, carvedilol, celiprolol (cardioselective + ISA + alpha2 blockers)

HT – treatment – BB - contraindications

- **Asthma bronchiale, asthmoid bronchitis,COPD**
- **AVB - 2nd and 3rd degree**
- **Bradycardia**
- **Acute heart failure**
- **Unstable type 1 DM**
- **SE's –** bronchoconstriction, peripheral vasoconstriction, potency disorders, raised TG and lowered HDL

HT – treatment - CaA

- **Phenylalkylamines** – Verapamil SR
- **Benzothiazepines –** Dilthiazem SR
- **Dihydropyridines -** Amlodipine, Isradipine, Felo-, Nitrendipine

Disadvantages – negative inotropic effect, slowing of sinoatrial and atrioventricular conduction

blood flushes, erythemas, oedemas

HT – treatment – ACE-I

- Captopril
- Enalapril
- Quinapril
- Perindopril
- Ramipril
- Trandolapril
- Lisinopril
- Spirapril

- Well tolerated
- SE's: dry, irritating cough, temporary increase of urea and creatinine, hyperkalaemia
- CI's: renovascular hypertension

HT – treatment – blockade of angiotensin II receptors (AT1), sartans

- Losartan
- Valsartan
- Telmisartan
- Candesartan
- Irbesartan

- Effects and indications similar to ACE-I
- Nephroprotective effect in type 2 DM
- Lower occurrence of DM in sartan-treated HT

HT – treatment – influence of alpha-adrenergic receptors

- **Peripheral alpha-blockers** — Prazosin, doxazosin
- **Central alpha-adrenergic agonists** — Methyldopa, clonidine
- **Central agonists and peripheral antagonists** — Urapidil
- **Agonists of imidazoline receptors** — Rilmenidin, moxonidil

HT – treatment - indications

- **Thiazide diuretics**
- **Loop d's**
- **D's – aldosterone antagonists**
- **Beta blockers**
- **CaA (dihydropyridines)**

Congestive heart failure, HT in seniors, ISHT, HT in black people

Congestive heart failure, RI

Congestive heart failure, state after MI

AP, state after MI, chronic heart failure, pregnancy, tachyarrhythmia

HT in seniors, ISHT, AP, ischaemic dis. of lower limbs, AS of carotids, pregnancy

HT – treatment - indications

- CaB verapamil,diltiazem
- ACE-I
- Sartans (AT1)
- Alpha blockers

AP, AS of carotids, supraventricular tachycardia

Congestive heart failure, LV dysfunction, state after MI, non-diabetic/diabetic nephropathy, proteinuria

Congestive heart failure, LV dysfunction, state after MI, non-diabetic/diabetic nephropathy, proteinuria

Benign hypertrophy of prostate, dyslipoproteinaemia

Pulmonary embolism (PE)

Obstruction of pulmonary arteries and capillaries by blood clots, fat tissue, air, amniotic fluid

Marked rise of pressure in the pulmonary artery = **precapillary pulmonary hypertension**

PE – aetiology, RF's

- **Deep venous thrombosis of lower limbs (85%)**
- **Thrombosis of pelvic veins, renal veins, the inferior vena cava, right heart thrombi**
- **Risk factors**

Large surgical and orthopaedic interventions, traumas of the lower limbs and pelvis

Malignant tumours

Thrombosis or PE in the patient's history

Sepsis, heart failure, ictus, obesity, pregnancy, contraceptives, deficit in AT III, protein C and S, hyperhomocysteinaemia

PE – clinical picture + examination

- Degree of pulmonary bloodstream obstruction

Massive PE = sudden death in 10% of the patients

Severe PE - syncope, hypotension, cardiogenic shock – hypotension, oliguria, peripheral vasoconstriction, sweating

History – suddenly developed dyspnoea, chest pain, cough, haemoptysis, syncope

PE – clinical picture + examination

- Asymptomatically – 30%
- Clinical picture – heavy obstruction of the pulmonary bloodstream
- Tachycardia, tachypnoea, cyanosis
- Hypotension, shock
- Accentuation of 2nd murmur over the pulmonary artery, protodiastolic gallop over the right heart, pleural friction murmur

PE – clinical picture + examination

- **ECG changes** in 60% haemodynamically significant

RBBB, wave S I,aVL over 1.5 mm, front. deflect. of V5, QS III,aVF, not II, low volt. of the limb lead, inversion of T III, aVF, V1-V4

- **Laboratory**

D-dimers, lowered saturation of O_2, decrease of PO_2 and PCO_2 up to respir. alkalosis

- **Echocardiography**

Dilatation of RV, tricuspid regurgitation in PH, dilatation of the pulmon. artery stem, paradoxical movement of IVS

Proof of venous thrombosis of lower limbs – Doppler examination

PE – clinical picture + examination

- **X-ray of the heart and lungs**

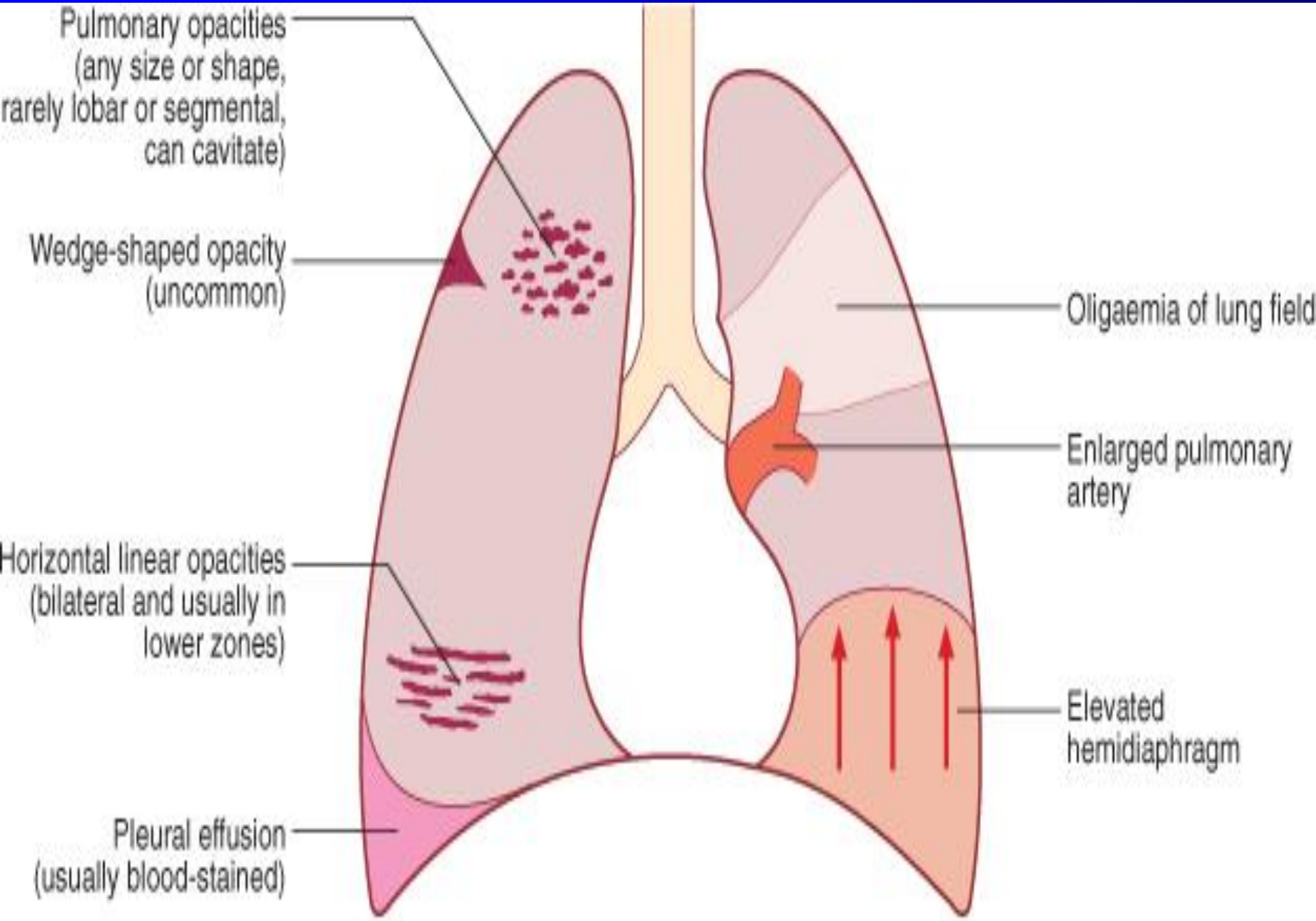
Triangular shading, pleural fluid, platelike atelectases - Fleischner sign

Right heart dilatation, thinning of the pulmonary markings – Westermark sign

50% - normal x-ray image

- **Spiral CT**

High sensitivity and specificity upon administration of the contrast medium – main stems



PE – clinical picture + examination

- **Pulmonary angiography –** "golden standard"
- **Ventilation-perfusion scan (V/Q scan)**

Perfusion scan – highly sensitive but little specific; ventilation scan – assessment of distribution, only a combination of both – sufficient diagnostic information, unavailable in service

- **Right-side catheterization**

Increased pressures in RA, RV, AP (PAP higher than 30/15 mm Hg), precapill. PH (difference between diastolic BP in the pulmonary artery and wedging higher than 8-10 mm)

PE - treatment

Acute stage

- **Thrombolytic therapy –** alteplase - Actilyse 10 mg i.v. bolus, then 90 mg in cont. inf. for 2 hr
- **Anticoagulant t.**

In severe forms after thrombolysis, in milder forms as a therapy basis

Heparin 10 000 U. as a bolus, then 750-1500 U/h according to aPTT

Low-molecular heparin

PE - treatment

Subacute stage

- Heparin for 7 to 10 days with transition to p.o. anticoagulant t. with warfarin INR 2-3
- Low-molecular heparin + transition to warfarin

Chronic stage

- Warfarin for 1 year, relapse of PE, and/or known RF's - individually

PE - prevention

- **Low risk**

Regimen measures – bandages of lower limbs, early postoperative mobilization

- **Medium, high, very high**

Medicamentous regimens – short-term, long-term

- **Primary and secondary prevention**

Low-molecular heparin 100 U/24 hr in uncomplicated surgeries, long-term prevention with warfarin, INR 2-3

Cor pulmonale

- **Dilatation of RV caused by pulmonary disease** (affection of structure or function of the lungs)
- **Cause – precapillary PH**

Restrictive, obstructive form, active with increased pulmonary vascular resistance

Cor pulmonale

- **Affection of airways and alveoli**

  Chronic bronchitis, asthma bronchiale, pulmonary fibrosis, inflammations, lung resections, hypobaric hypoxia

- **Affection of chest movements**

  Kyphoscoliosis, obesity with hypoventilation, polymyelitis

- **Affection of pulmonary vessels**

  Polyarteritis nodosa, embolism, thrombotic disease

Cor pulmonale – clinical picture

- **Symptoms of the disease conditioning PH and cor pulmonale**
- **Heart failure** – dyspnoea
- **Hypoxia and hypercapnia**

Memory disorders, irritation, restlessness, aggressiveness, or contrarily drowsiness, somnolence

- **Central cyanosis, tachycardia**

Cor pulmonale – clinical picture

- **Clinical picture of central disease**

Chronic bronchitis – prolonged expirium, barrel chest, spastic phenomena

- **Symptoms of PH**

Accentuation/split of 2nd murmur over P, diastolic regurgitant Graham Steell murmur (3rd intercostal space parasternally to the left)

- **Right heart symptoms**

Systolic regurgitant murmur over the tricuspid valve, systolic pulsation of RV

Cor pulmonale – clinical picture

- **Right heart failure**

Increased filling of jugular veins, hepatojugular reflux, hepatosplenomegaly, systolic pulsation of the liver, drumstick fingers

Cor pulmonale - diagnostics

- **Laboratory**
- **X-ray**
- **ECG**
- **Echocardiography**
- **Isotope ventriculography**

Polyglobulia, elevation of hepatic tests

Dilatation of main stems of the pulmonary artery, dilatation of the cardiac silhouette

Hypertrophy of RV

Indirect signs of PH, dilatation of right-side compartments, estimation of pressures in right-side compartments

Assessment of RV function at rest and at exertion

Cor pulmonale - treatment

- Alleviation of bronchial obstruction in COPD (= chronic obstructive pulmonary disease)
- Exclusion of smoking
- Prevention of infections (vaccination at the time of epidemics, isolation)
- Pulmonary rehabilitation
- Oxygen therapy
- Mucolytics, steroids, beta mimetics, bronchodilators, diuretics

Cor pulmonale - prognosis

- **PH class, character of the primary disease**
- **COPD – RF's**

  Age, ECG, signs of RV hypertrophy, chronic respir. insufficiency, IHD, FEV1 lower than 590 ml

Pulmonary hypertension (PH)

Increased medium pressure in the pulmonary artery (PAP) over 20 mm Hg in rest conditions

- **Latent PH**

Rest AP under 20 mm Hg; over 30 mm at exertion

- **Mild PH**

PAP at rest 20-30 mm Hg, over 30 mm at exertion

- **Severe PH**

PAP at rest over 30 mm Hg

Pulmonary hypertension (PH)

- **Hyperkinetic type of PH**

- Increased pulmonary blood flow – congenital heart defects with a left-to-right shunt, changes at first functional, later also anatomical – proliferation in the media and intima of pulmonary arteries

- Hypertrophy and failure of RV

Pulmonary hypertension (PH)

- **Postcapillary PH**

- Pathological process behind the pulmonary vascular bed
- Increased pressure in the left-sided cardiac bed – mitral stenosis, left heart failure, aortic defects, constrictive pericarditis

Pulmonary hypertension (PH)

- **Precapillary PH**

- Increased pulmonary vascular resistance
- Increased PAP, normal capillary pulmonary pressure (wedge pressure)
- Acute cor pulmonale – PE
- Chronic cor pulmonale - restrictive, hypoxic, and obstructive forms

Pulmonary hypertension (PH)

- **Primary PH**

- Pathologically high pulmonary vascular resistance and increased PAP
- Obstructive form of PH
- A rare disease, age 20-40 years, 3x more frequently in females

Primary PH

- Unknown aetiology
- Vascular spasm – hypertrophy of the media – subintimal proliferation of the fibrous tissue – microthromboses of pulmonary arterioles – necrotizing arteritis
- **Clinical picture**

  Dyspnoea, increased fatigue and faintness, syncope, Raynaud sy, haemoptysis, RV failure

Primary pulmonary PH

Diagnostics

- XR – enlargement of the main pulmonary arteries
- ECG – hypertrophy of RV
- Functional examination of the lungs –restrictive disorder + decreased diffusion capacity
- Echocardiography – dilatation of RA and RV, abnormal movement of IVS, tricuspid regurgitation

Primary pulmonary PH

- Pulmonary scintigraphy
- Pulmonary angiography – massive central pulmonary arteries with peripheral stenosis
- Right-sided cardiac catheterization – heaviness of PH, pulmonary vascular resistance, vasodilator response

Primary pulmonary PH

Treatment

- CCB's – if the vasodilator test is positive (prostacyclin, NO)
- Anticoagulant therapy with warfarin, INR ca. 2.0
- Digoxin – improved function of overloaded RV + decrease in plasmatic concentration of noradrenaline
- Diuretics, oxygen therapy
- sildenafil, bosentan, beraprost, treprostinil

Cardiomyopathy (CMP)

Diseases of the myocardium with cardiac dysfunction

3 haemodynamic types

- **Dilated CMP (90%)-** systolic contractile dysfunction
- **Restrictive CMP –** diastolic dysfunction
- **Hypertrophic CMP (HCM) –** diastolic dysfunction + supranormal contractions

Dilated CMP (DCMP)

- Dilatation + impaired contraction of LV (LV, RV)
- Genetically heterogenous disease, possibly viral infection induction
- Eccentric hypertrophy with dilatation of cavities, ball-shaped LV, myocytolysis, fibrosis, and hypertrophy of the remaining myocytes

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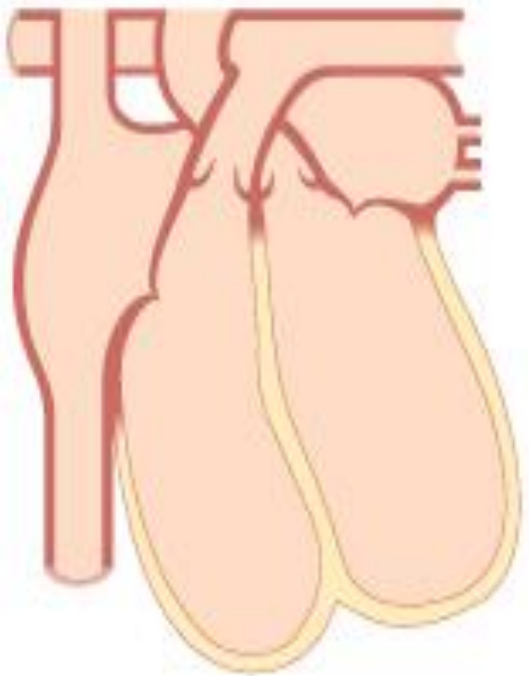


Dilated CMP (DCMP)

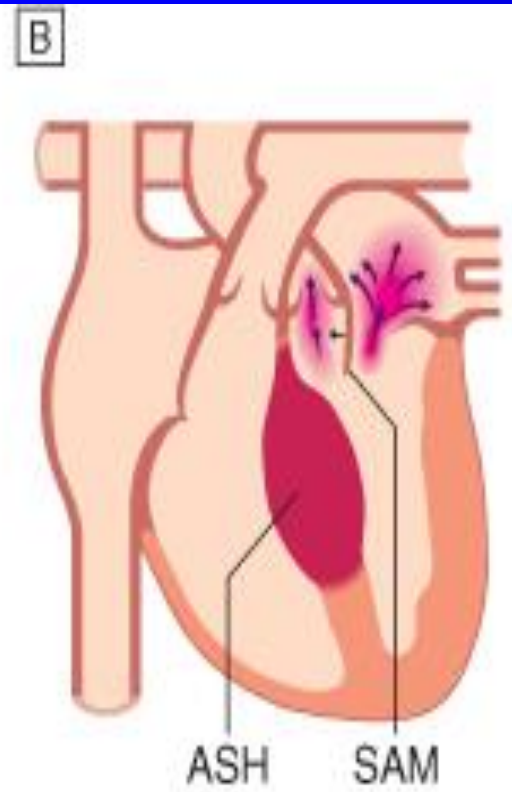
- At first asymptomatic,
- signs of left heart failure, dyspnoea, oedemas, cardiac rhythm disorders, thromboembolic accidents, sudden death
- Gallop, mitral and/or tricuspid regurgitation
- ECG – LA load, LV hypertrophy, BBB, ST-T changes, atrial fibrillation

Dilated CMP (DCMP)

- XR – enlarged heart with CTI over 0.5, pulmonary venostasis
- Echocg – dilatation of LV or even PV, non-thickened walls, diffuse hypokinesis, mitral, tricuspid regurgitation, EF of LV is decreased
- Treatment – CHF- ACE-I, AT1 block, BB, digitalis, diuretics, anticoagulant therapy

Hypertrophic CMP (HCMP)

- **Concentric hypertrophy of undilated LV (RV)**
- **Hereditary** – mutation of 7 genes - 80-90%
- **Non-obstructive HCMP**
- **Obstructive HCMP** – contraction of hypertrophic septum + abnormal movement of the frontal cusp of mitral valve = functional systolic obstruction of the outflow tract of LV (subaortic muscular stenosis)



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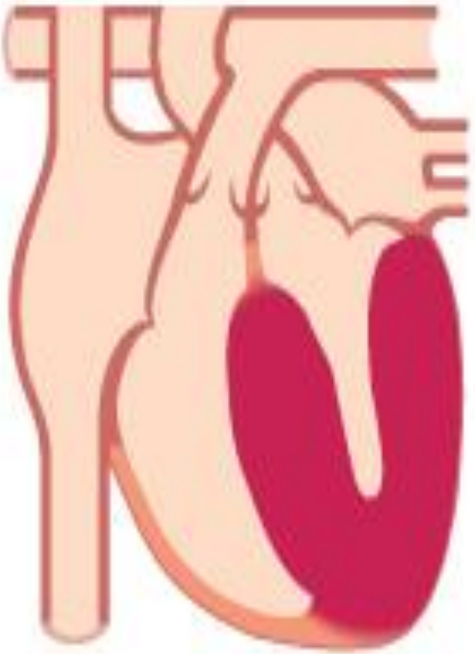

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Hypertrophic CMP (HCMP)

- Exertional dyspnoea, exertional AP, exertional syncopae, palpitation, sudden death
- Ejection murmur with a vortex parasternally to the left
- XR – non-enlarged heart
- ECG – LA load, LV hy, pathol. Q waves (hy of septum), negative T, arrhythmia, blockades

Hypertrophic CMP (HCMP)

- **Echocardiography** – asymmetrical hy of septum, (up to 20 mm), IVS is hypokinetic, the free wall of LV is hyperkinetic, EF is raised, diastolic dysfunction, obstructive HCMP - SAM, systolic gradient of LV/outflow tract up to 100 mm Hg

Hypertrophic CMP (HCMP)

Treatment

- Restriction of heavy physical loading – prevention of sudden death
- BB, CCB – verapamil, carefully diuretics, vasodilators, antiarrhythmics (amiodarone, sotalol)
- Positively inotropic substances are contraindicated even in heart failure

Hypertrophic CMP (HCMP)

Treatment

- Cardiostimulation – inverse procedure of electric activation of the heart
- Surgical interventions (myectomy, myotomy)
- PTSMA – induction of a small necrosis in the proximal part of the hypertrophic septum - ethanol into the septal branch of RIA

Restrictive CMP (RCMP)

- Restrictive filling, reduced diastolic volume of one or both ventricles, normal systolic function, increased filling pressure of LV and of pulmonary pressures
- The walls are not or only little thickened.

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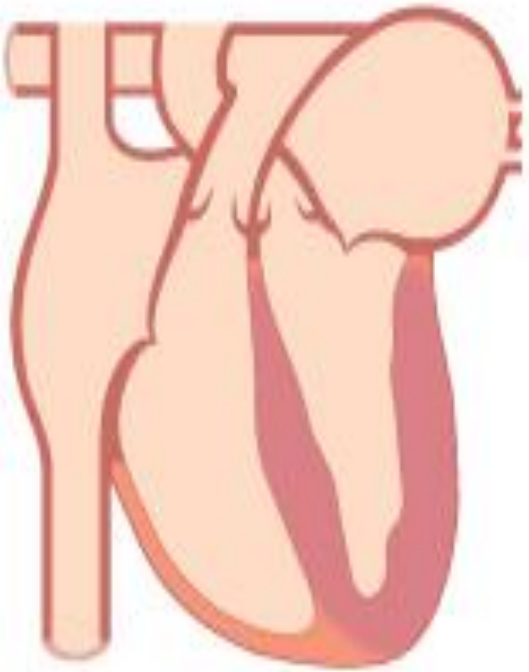


Restrictive CMP (RCMP)

- **Endomyocardial disease (fibrosis)**

Acquired, tropical areas

- **“Löffler endocarditis”**

Part of the hypereosinophilic syndrome – storing of activated eosinophils in the cytoplasm of the cardiocytes (going “rogue”)

- **Infiltrative diseases** (extracellular - amyloid)
- **Sparing diseases** (intracellular storage)
- **Carcinoid, cytostatics, irradiation**

Restrictive CMP (RCMP)

- Dyspnoea, fatigue
- Right heart failure
- Regurgitation on atrioventricular valves
- Low minute volume, SVT, low systolic volume, thromboembolic complications
- XR – non-enlarged heart, pulmonary congestion, pleural fluid

Restrictive CMP (RCMP)

- ECG – low voltage (RV), atrial fibrillation, conduction disorders, non-specific ST-T changes
- Echocardiography – mild thickening of ventricular walls, ventricles are non-enlarged, atria are enlarged, normal systolic function, restrictive filling of ventricles, mitral, tricuspid regurgitation, PV

Treatment – diuretics, antiarrhythmics, anticoagulants, KS, ICD, orthotopic transplantation in idiopathic RCMP

Arrhythmogenic right ventricular dysplasia

- Congenital, genetically conditional
- Non-homogeneous progressing replacement of RV musculature with fatty or fatty-fibrous tissue
- A frequent cause of death in young people

F

Arrhythmogenic right ventricular dysplasia

- Asymptomatically; tiredness, palpitation, dizziness, syncopae, murmur at the lower sternum, right heart failure
- Sudden death – 30-50% first symptom – severe arrhythmias, particularly ventricular fibrillation
- ECG, XR, echocg, MR, CT, right-sided catheterization, Holter monitor, programmed stimulation of ventricles
- Treatment – antiarrhythmia, ICD

Inflammatory heart affections
Infectious endocarditis

- **Valvular or mural endocardium**
- **Untreated – death**
- **Formation of vegetation –** thrombocytes, fibrin, micro-organisms
- **Rheumatic fever, postrheumatic valvular defects**
- **Odontogenic infections –** most frequent
- **Mortality 20-30%**

Infectious endocarditis

- Streptococci, staphylococci. G-negative bacteria, enterococci, HACEK, fungi
- **DG** – histology of the vegetations, positive haemoculture, echocg – a waving structure intracard., on the valves, and/or at the defect
- History of the heart defect, fever above 38 ºC, systemic or pulmonary embolization

Infectious endocarditis

DG

- Immunological phenomena – GN with proteinuria and haematuria
- Intravenous administration of medicaments, drugs
- Instrumental - stomatological intervention

Infectious endocarditis

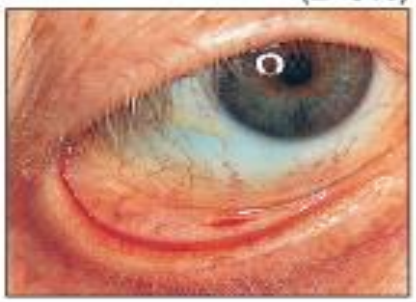
- **History**

Cardiac affection, systemic disease, i.v. application of medicaments, instrumental intervention, drug addiction

Objective examination

Fever, auscultation finding, splenomegaly, skin changes, symptoms of heart failure, neurological symptoms

Subconjunctival haemorrhages
(2–5%)

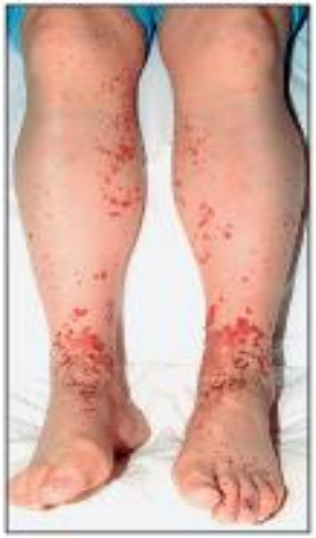


'Varying' murmurs
(90% new or changed murmur)
Conduction disorder
(10–20%)
Cardiac failure
(40–50%)

Haematuria
(60–70%)

Osler's nodes
(5%)

Petechial rash
(40–50%, may be transient)



Loss of pulses

Cerebral emboli
(15%)

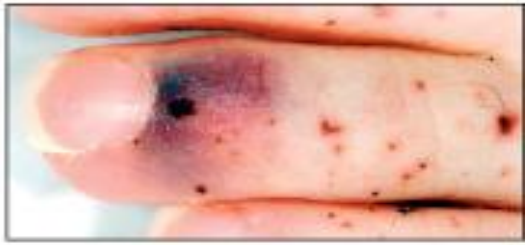
Roth's spots in fundi
(rare, < 5%)

Petechial haemorrhages on mucous membranes and fundi
(20–30%)

Poor dentition

Splenomegaly
(30–40%, long-standing endocarditis only)

Systemic emboli
(7%)
Nail-fold infarct



Digital clubbing
(10%, long-standing endocarditis only)

Splinter haemorrhages
(10%)

Infectious endocarditis

- **Laboratory + other examinations**

Haemoculture, FW, CRP, CBC, urine + sediment., ophthalmology, X-ray of heart and lungs, examination of the source – foci, i.e. ENT, urology, gynaecology, dentist's, dermatology

Echocardiography – proof of vegetation, haemodynamic affection

- **Course and complications**

Symptoms of concurrent cardiac disease, very rarely acute sepsis, frequent administration of antibiotics before dg

Infectious endocarditis - treatment

- **Antibiotics**

i.v., max. therapeutical dose, according to sensitivity, double or triple combination, six weeks

- **Cardiosurgical intervention**

Unmanageable sepsis, refractory heart failure, progressing heart defect, repeated embolizations, large waving vegetation, intracardiac abscess

Infectious endocarditis - ATB prophylaxis

- **A risk patient**

- **High risk**: artificial heart valve, bacterial endocarditis in history, cyanotic heart defects, state after surgery of congenital heart defects
- **Medium risk:** acquired valvular defects, HCMP, prolapse of the mitral valve

Infectious endocarditis - ATB prophylaxis

- **A risk patient**

- **Low risk:**
- prophylaxis is not necessary – atrial septal defect, state after surgery of atrial and ventricular septa

Infectious endocarditis - ATB prophylaxis

- **Risky intervention**

- Stomatological interventions
- Interventions in the respiratory tract
- Interventions in the GIT
- Interventions in the urogenital tract

Infectious endocarditis - ATB prophylaxis

- **Mode of ATB prophylaxis**

- p.o. 1 hr prior to intervention
- i.m. 15-30 min
- i.v. immediately
- The presumed time period of bacteraemia is less than 2 hrs, or there is no great blood loss – just one dose, otherwise over the whole time period of barrier impairment

Myocarditides - aetiology

- **Biological damage**

Viruses, bacteria, yeasts, fungi

- **Chemical damage**

Lead, arsenic, snake venoms, antibiotics, cytostatics

- **Physical damage**

Radiation

Viruses – most frequent, Coxsackie viruses B6 – 50 %

Myocarditides - aetiology

- **Other diseases**

Rheumatic fever, SLE

Hypereosinophilic syndrome

Rejection after transplantation

Myocarditides – clin. picture

- Subclinical to severe, and/or lethal termination – heart failure, arrhythmia
- Acute as well as chronic
- Acute viral myocarditides – symptoms of influenza
- Palpitations, dyspnoea, chest pain
- Exhaustion, cyanosis, tachycardia, tachypnoea, heart murmur, gallop, congestion, hypotension, shock, arrhythmias – supraventricular and also ventricular

Myocarditides - prognosis

- Complete recovery
- Decreased function of LV
- Transition to dilated CMP
- Fulminant forms – cardiogenic shock
- Sudden death - arrhythmia

Myocarditides - diagnostics

- Difficult – often unrecognized
- Isolation of infective agent from the serum, from myocardium
- Immunology – antibodies against myocardium
- FW increased in 60%, Ie in 25%, CRP
- Troponin I in 30-50%
- ECG – changes in ST-T, lengthened QT, pathol. Q, arrhythmia – SVT, AVB – rheumatic fever, Lyme borreliosis

Myocarditides - diagnostics

- XR – normal, enlarged heart shadow, signs of congestion, pleural effusion
- Echocg – changes in the size of heart chambers, disorders of both systol. and diastol. function, segment. disorders of kinetics, PV, thrombus in LV – 15%
- MR – abnormal signal from the affected myocardium
- Endomyocardial biopsy – highly specific, little sensitive

Myocarditides - treatment

- Bed rest if in acute stage
- Eradication of the caus. agent (if known)
  Antivirals, immunosuppressives – 0
- NSA – contraindicated
- ACE-I – promising results
- Treatment of arrhythmias and heart failure
- Orthotopic transplantation of the heart

Pericarditides (P)

- **Inflammation of the visceral or parietal layers of the pericardium**
- **Acute, chronic p.**
- **P. sicca, p. with effusion**

– effusion is serous, fibrinous, haemorrhagic, purulent

Pericarditides (P)

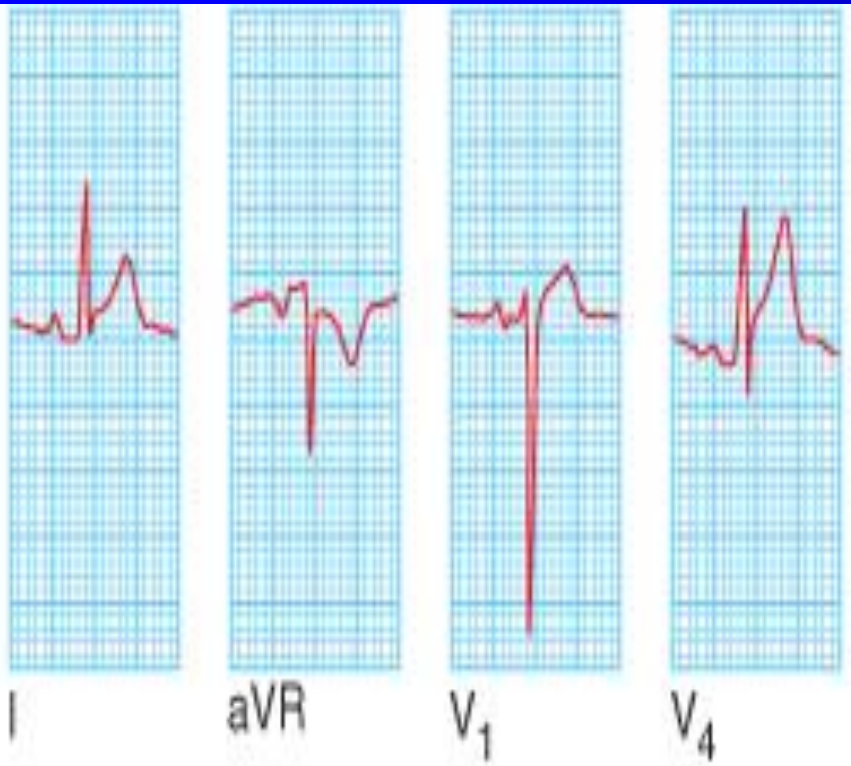
- **Aetiologically** – rheumatic, bacterial, viral, acute benign, actinomycotic, tuberculous, parasitic, uraemic, in systemic disease of the connective tissue, in MI, after a cardiosurgical intervention, traumatic, neoplastic, postirradiation p.

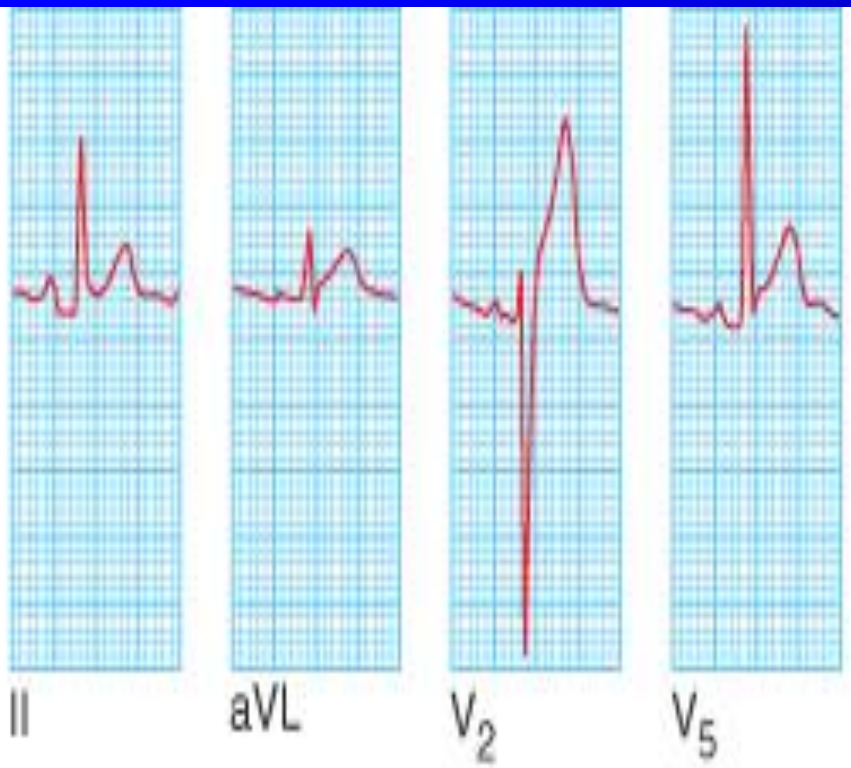
Acute p. – clinical picture

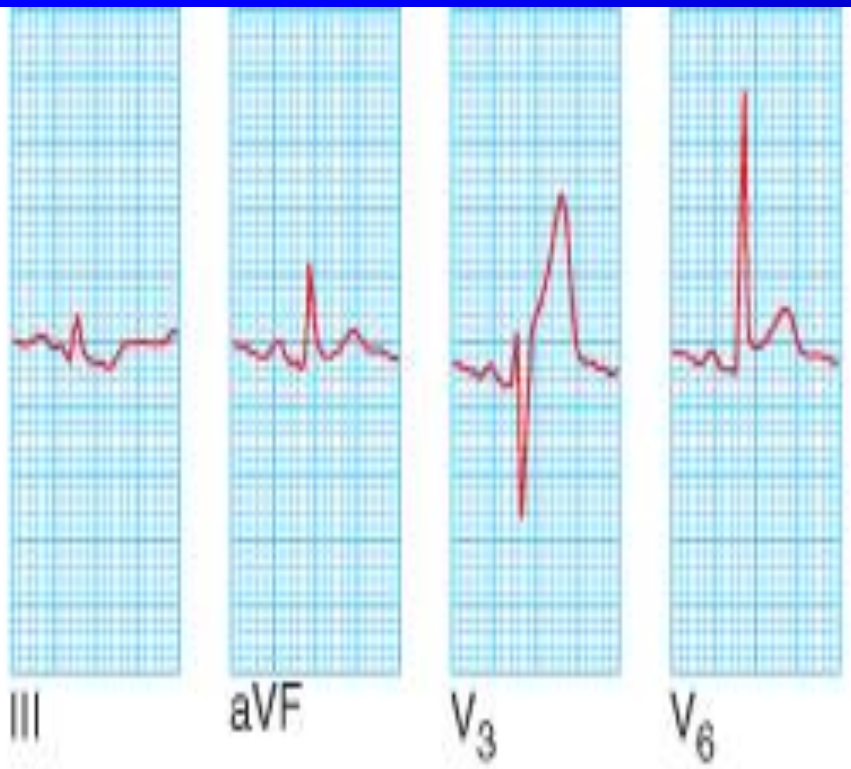
- **Chest pain** in a lying position +, in a sitting position and in forward bend –
- **Dyspnoea** – p. with effusion
- **Inflammation:** fever, chill, faintness, cough
- **Friction murmur** – gets stronger by pressing the phonendoscope against the chest, variable, disappears with exudation

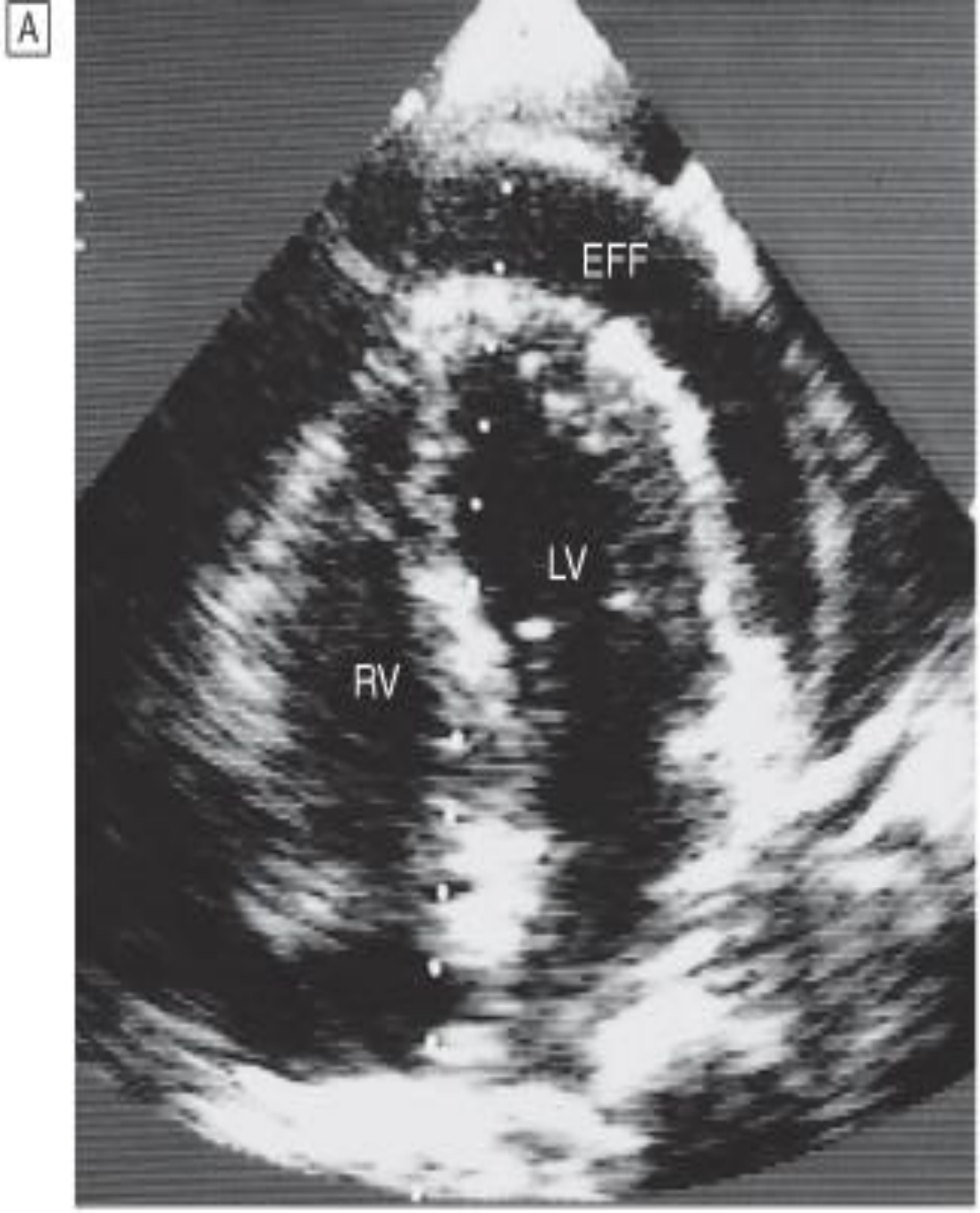
Acute p. – clinical picture

- **ECG:** elevation of ST, inversion of T, low voltage, electrical alternans
- **XR:** extension of heart shadow because of exudation
- **Echocg:** the quantity of effusion
- **CT, MR:** distinguishing of pericardial cyst, tumour
- FW, CRP, CK-MB, AST, LD, troponin









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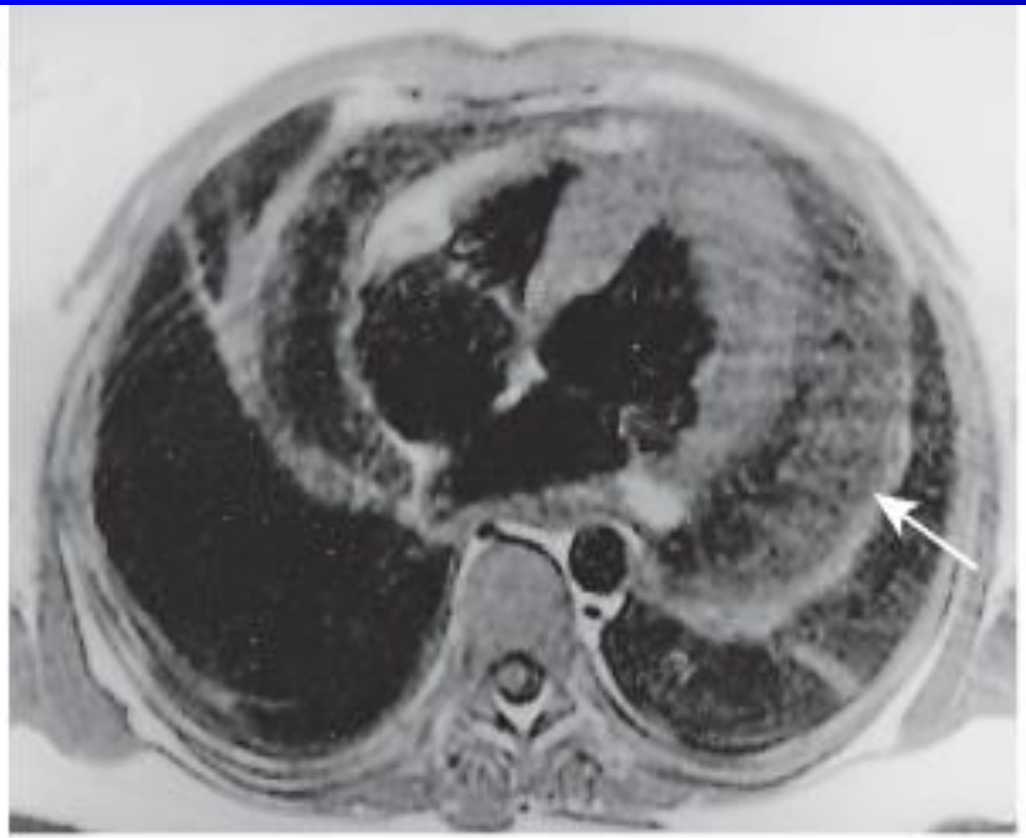


Types of pericarditides

- **Acute benign (idiopathic) p.**

In younger people, antipyretics, NSA, CS

- **Viral p.**

1 to 3 weeks after virosis, sometimes tamponade

- **Bacterial p.**

Septicaemia, or tamponade, ATB, drainage of pericardium, chronicity, high mortality

Types of pericarditides

- **Tuberculous p.**

Haemorrhagic effusion, possible tamponade, chronicity, antitubercular s

- **Uraemic p.**

Antipyretics, NSA, more intensive dialysis

- **In malignant disease**

Haemorrhagic effusion, possible tamponade

Types of pericarditides

- **In acute MI**

P. epistenocardiaca – first week, NSA

Dressler syndrome (later – 3rd week), pleuritis, pneumonitis, interruption of anticoagulant therapy

- **Pericardiotomic sy, posttraumatic, hypothyroidism, SLE**

Cardiac tamponade

- Rapid accumulation of effusion
- Congestion of blood in front of the heart
- Lungs insufficiently filled with blood, hepatomegaly
- Dyspnoea, tachycardia, hypotension, shock
- Increased filling of jugular veins
- ECG : lowered voltage, electrical alternans

Cardiac tamponade

- **Echocardiography**:

reduced volume of RV and LV, diastolic collapse of atria and the free wall of RV

- **Therapy:**

pericardiocentesis, evacuation of pericard. effusion, pericardiectomy, examination of the puncture fluid – cause + adequate treatment

Constrictive pericarditis

- Chronic inflammation, fibrous thickening of the pericardium, adhesions – restriction of diastolic filling
- TB, purulent inflammation, haemopericardium, radiation, surgical interventions
- Restriction of filling – failure of both ventricles during normal systolic function

Constrictive pericarditis

- Fatigue, dyspnoea
- Impaired function of the liver – Pick's pericarditic pseudocirrhosis
- Pulsation of cervical veins, triple sound
- Pericardiectomy

Angiology

Thromboembolic disease (TED)

- **Deep venous thrombosis (DVT)**
- **Phlebothrombosis**

Primary thrombosis of the deep venous system

- **Thrombophlebitis**

Thrombosis after primary damage to the venous wall, particularly the superficial venous system

TED - complications

- **Pulmonary embolization**
- **Chronic pulmonary hypertension (PH)**
- **Chronic postthrombotic syndrome (PTS)**

80% of the causes of chronic venous insufficiency

PTS – in 22-36% of the patients with DVT

TED – risk factors

Clinical factors

- Age over 70 years, preceding TED
- Larger surgical operation, fractures of the thigh
- Malignant tumours
- Immobilization, congestive heart weakness
- Pregnancy, contraceptives
- Venous stasis – varices, obesity
- Acquired coagulation disorders - polycythaemia

TED – risk factors

Congenital factors

- Factor V Leiden – resistance to APC
- Deficit in antithrombin III
- Deficit in protein C and protein S
- Disorder of plasminogen, dysfibrinogenaemia
- Hyperhomocysteinaemia
- HLA: antigens CW4, DR5, DQW3

TED – clinical picture + diagnosis

- **Thrombophlebitides**

A tender, reddened, rigid stripe, without raised temperature, a rather small swelling

- **Phlebothrombosis**

Warm skin, of slightly reddish colour, a rather rigid calf, tender on palpation, Homans sign, plantar sign

TED – clinical picture + diagnosis

- **Phlebothrombosis**

Phlegmasia – a massive swelling, tense skin, tenderness of the limb

- **Phlegmasia cerulea dolens** – a cyanotic to purpuric extremity
- **Phlegmasia alba dolens** – reflexive affection also of the arterial system – pale to marbly

TED – clinical picture + diagnosis

- **DVT on the upper limb**

Oedema, light cyanosis, collateral superficial venous network – traumas, iatrogenic

- **Doppler ultrasonography (DUSG) –** assessment of the blood flow
- **Venous occlusion plethysmography –** volume changes

TED – clinical picture + diagnosis

- **Duplex ultrasonography**

Two-dimensional imaging of a vessel + Doppler and colour assessment of the blood flow

- **XR contrastive phlebography**
- **CT phlebography**
- **D dimer –** a negative prediction factor

TED – treatment

- **Heparin (UFH)**

i.v. 5000 IU bolus, further continuous infusions , 750-1500 IU/h – APTT 2- 2.5x prolonged

- **LMWH**

1 mg/1 kg s.c. à 12 hrs

- **Thrombolysis, thrombectomy, interventional endovascular methods**

High femoral, ileofemoral DVT

- **P.o. anticoagulant therapy**

From day 2, INR 2.0, 3.0, Quick 20-30, 3-6 months, complications - individually

TED – treatment

- **Compression of lower limbs**

Stockings, elastic roller bandages

- **Bed rest**

24 hrs, afterwards easy walking

- **Prevention with LMWH**

Risk patients – 40 mg (0.4 ml) 1x per day

Diseases of aorta and large arteries

- **Congenital**

Coarctation, double aortic arch, right aortic arch, Marfan syndrome – later manifestations

- **Acquired**

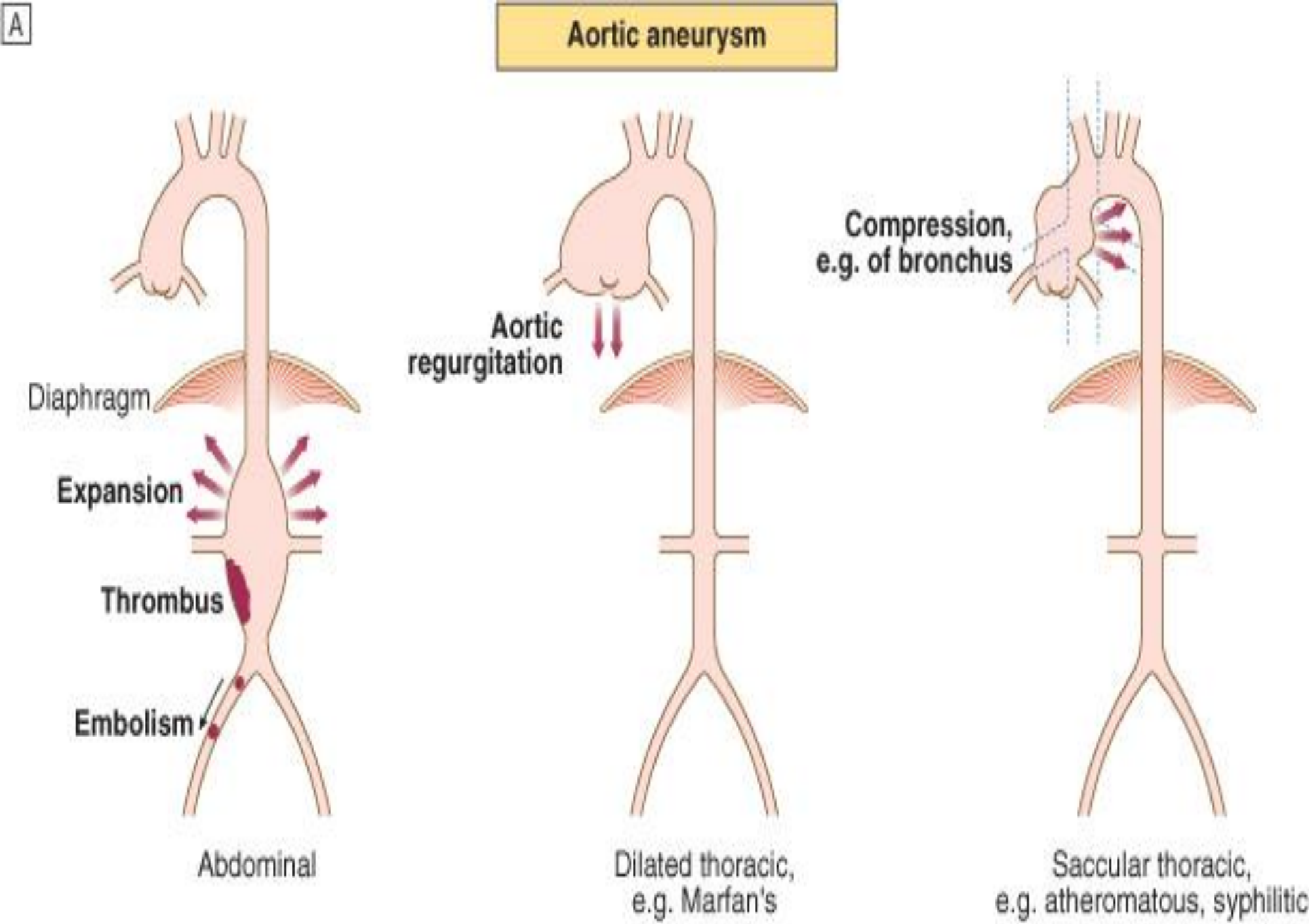
AS, aneurysm, dissection, inflammations, traumas

Aortic aneurysm

- Dilatation of the aorta by more than 50 %
- Age, more frequent in males
- X-ray examination of the chest
- Abdominal sonography, or TTE
- TEE – proof
- CT, MR, retrograde aortography
- Th: surgical (large, symptomatic, rupture)

A

Aortic aneurysm



Aortic dissection

- **Avulsion of the intima –** haemorrhage into the aortic media
- **2 lumina – right, dissection channel**
- **Severe, jerky pain** – localization and propagation depending on the site and spreading of dissection with obstruction of arteries leading from the aorta
- **Deficit of pulse** on upper limbs, lower limbs, carotids

Aortic dissection- Predisposing Factors

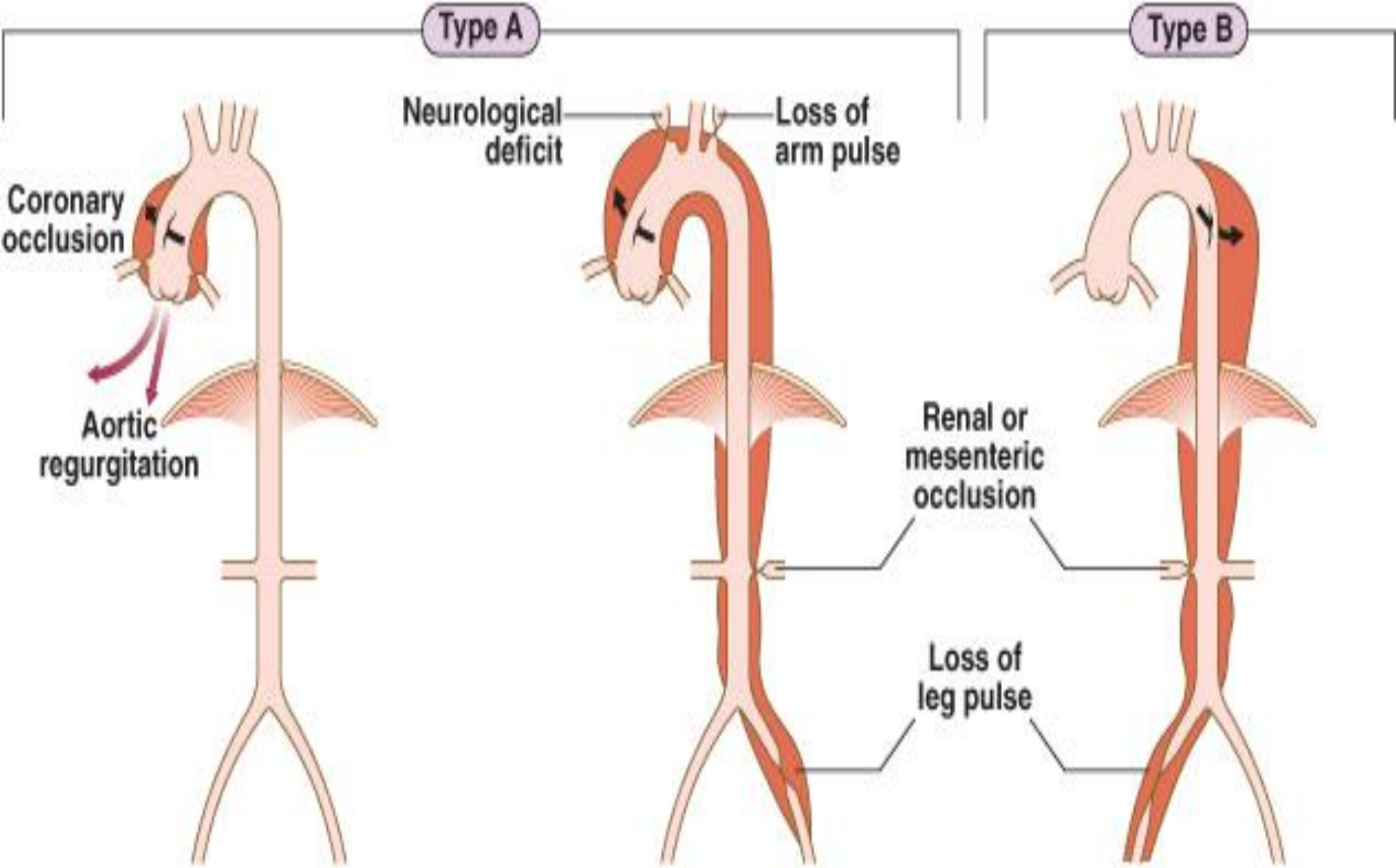
- Hypertension (80%)
- Aortic atheroclerosis
- Non-specific aortic aneurysm
- Aortic coarctation
- Collagen disorders (Marphan s sy)
- Fibromuscular dysplasia

Aortic dissection- Predisposing Factors

- **Previous aortic surgery** (CABG, aortic valve replacement)
- **Pregnancy** (usually third trimester)
- Trauma
- **Iatrogenic** (cardiac catheterization, intra-aortic ballon pumping)

B

Aortic dissection



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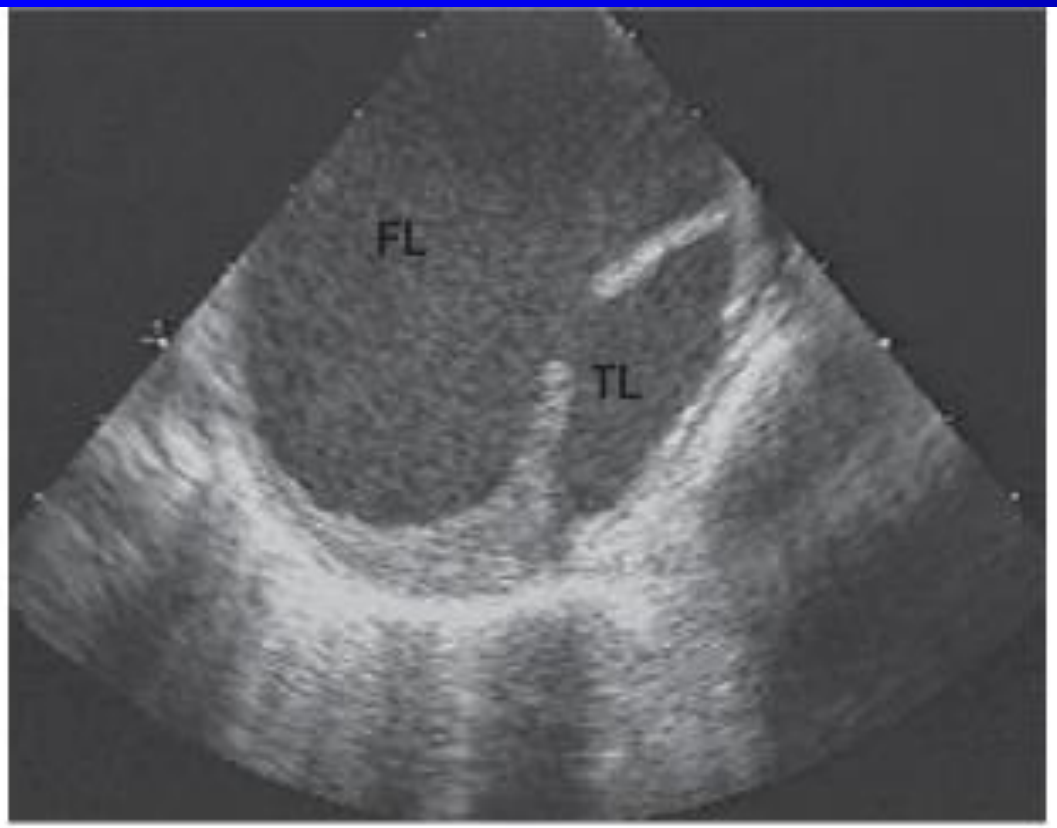

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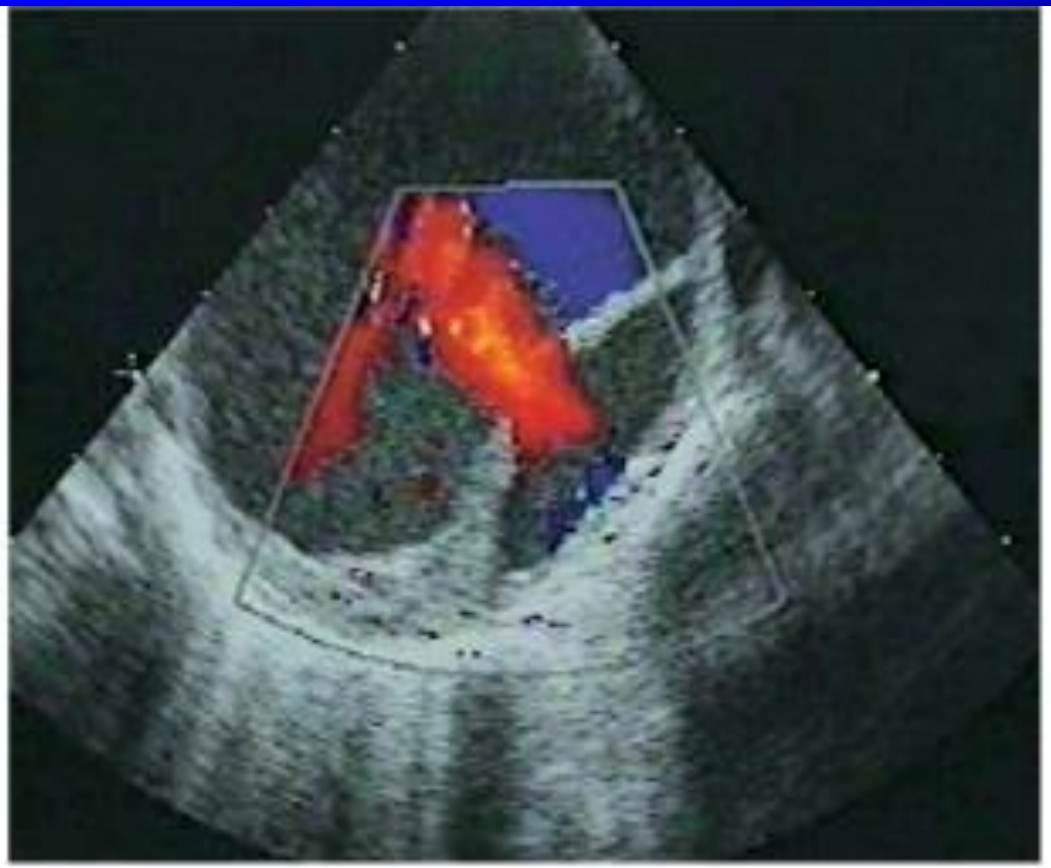
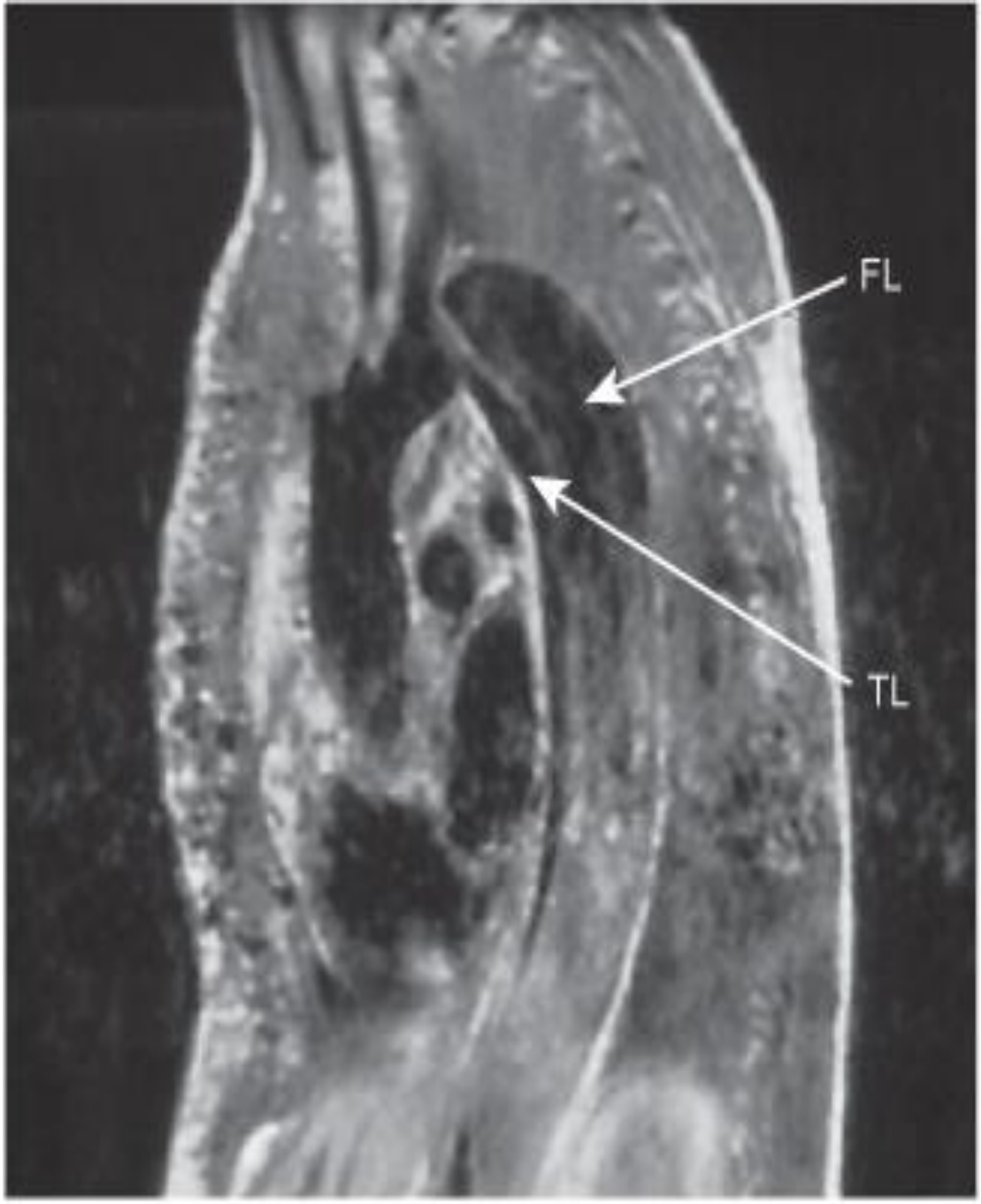


Aortic dissection

- **Ischaemic affection of organs**

CVA, AMI, renal failure, ischaemia of the extremity

- **Ao regurgitation –** affection of aortic annulus
- **Tamponade -** communication with the pericardium
- **Shock condition**
- **TEE, spiral CT – urgent operation**



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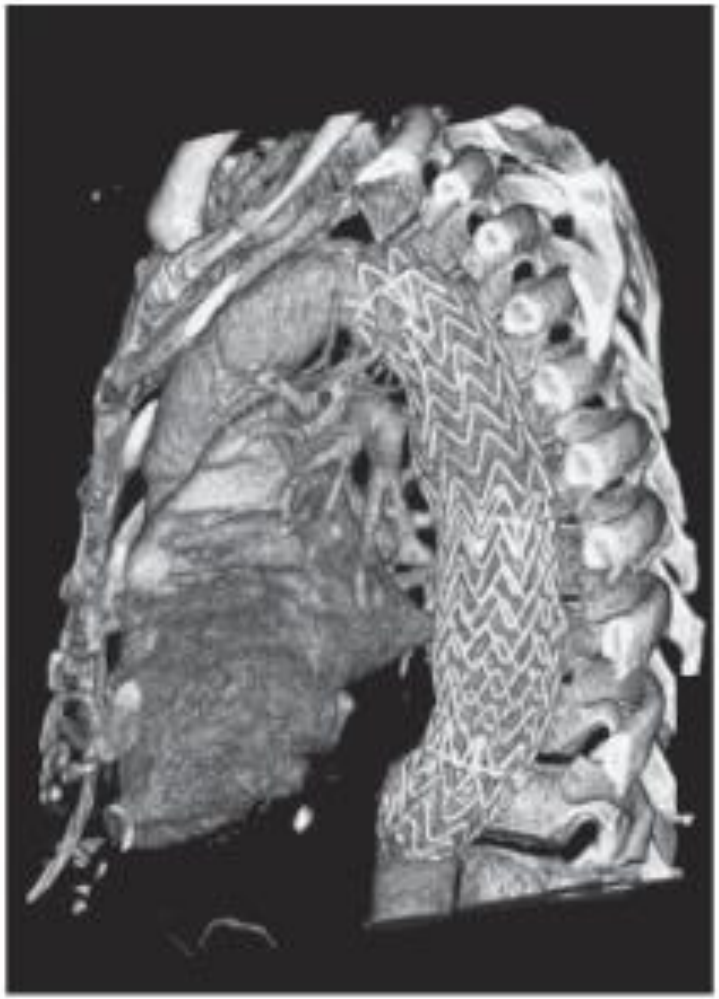


Disease of peripheral arteries (UCHPT, PAOD)

- Subrenal aorta, iliac arteries and arteries of the lower limbs, a. aortic arch, magistral cervical arteries, arteries of the upper extremities
- Ischaemic dis. of lower limbs, CHDK, CVA
- AS + subsequent thrombosis 85-90%
- 10-15% vasculitides, embolization, traumas

Factors Influencing the Clinical Manifestation of PAD – anat. site

- Cerebral circulation – TIA, VBI
- Renal arteries – HT and renal failure
- Mesenteric arteries – mesenteric angina, acute intestinal ischaemia
- Limbs (legs more often) – intermitent claudication, critical limb isch., acute isch.

Factors Influencing the Clinical Manifestation of PAD – Collateral supply

- In a patient with a complete circle of Willis, occlusion of one carotid artery may be asymptomatic
- In a patient without cross-circulation, stroke is likely

Factors Influencing the Clinical Manifestation of PAD – Speed of Oneset

- Where PAD develops slowly, a collateral supply will develop
- Sudden occlusion of a previously normal artery is likely to cause severe distal ischaemia

Factors Influencing the Clinical Manifestation of PAD – Mechanism of Injury

- **Haemodynamic** – plaque must reduce arterial diameter by 70% („critical stenosis“) to reduce flow and pressure at rest. On exertion na much lesser stenosis may become“critical“. This mechanism tends to have a relatively benign course due to collateralisation

Factors Influencing the Clinical Manifestation of PAD – Mechanism of Injury

- **Thrombotic** – occlusion of a long standingcritical stenosis may be asymptomatic due to collateralisation. However acute rupture an thrombosis of a non-haemodynamically significant plaque usually has severe consequences

Factors Influencing the Clinical Manifestation of PAD – Mechanism of Injury

- **Atheroembolic –** symptoms depend upon embolic load and size (carotid – TIA, stroke)
- **Thromboembolic –** usually secondary to atrial fibrillation

Features of Chronic Lower Limb Ischaemia

- **Pulses –** diminisched or absent
- **Bruits –** denote turbulent flow but bear no relationship to the severity of the underlying disease
- **Skin temperature –** reduced
- **Buerger‘ s sign –** pallor on elevation and rubor an dependency

Features of Chronic Lower Limb Ischaemia

- **Superficials veins –** to fill sluggishly and empty („gutter“) upon minimal elevation
- **Muscle – wasting**
- **Skin and nails –** dry, thin and brittle
- Loss of hair

Diabetic Vascular Disease – diabetic angiopathy

- **Macroangiopathy = AS**
- **Microangiopathy**

Hyaline atherosclerosis with thickening of the capillary basal membrane

Disturbance of microcirculation

- Non-enzymatic glycation of proteins
- The polyol mechanism – accumulation of sorbitol

Diabetic Vascular Disease – diabetic angiopathy

- **Microangiopathy**
- Diabetic nephropathy
- Diabetic retinopathy
- Peripheral neuropathy

DG of microangiopathy:

- Finding of proliferative retinopathy
- Examination of thickness of the arterial wall on the common carotid artery (a. car. comm.)

Diabetic Vascular Disease – diabetic angiopathy

Angiopathy

Macrovascular affection

Microvascular affection

Neuropathy

Disturbance of trophic functions

Trauma

ulceration

infection

amputation

Arterial occlusions

- **Embolization** – atheromas and thrombi from aorta, thrombi in atrial fibrillation
- **Thrombosis** - vascular damage - AS
- **Vasoneuroses** – transient disorders of blood supply

  Arterial side of microcirculation: white fingers and toes

  initial stage of Raynaud phenomenon

  Venous side: acrocyanosis

Arterial occlusions

- **Vasculitides:** inflammation up to necrosis of the vascular wall
- Focal, segmental affection of target tissues
- Damage to the target organs

Arterial occlusions - DG

Claudication pain stages	Clinical description
I	Asymptomatic
IIa	Claudication above 200 m
IIb	below 200 m
IIc	less than 50 m
IIIa	Cl. painful, ankle BP over 50
IIIb	Ankle BP under 50
IV	Defect on lower limb, devel. from II

Acute arterial occlusions - DG

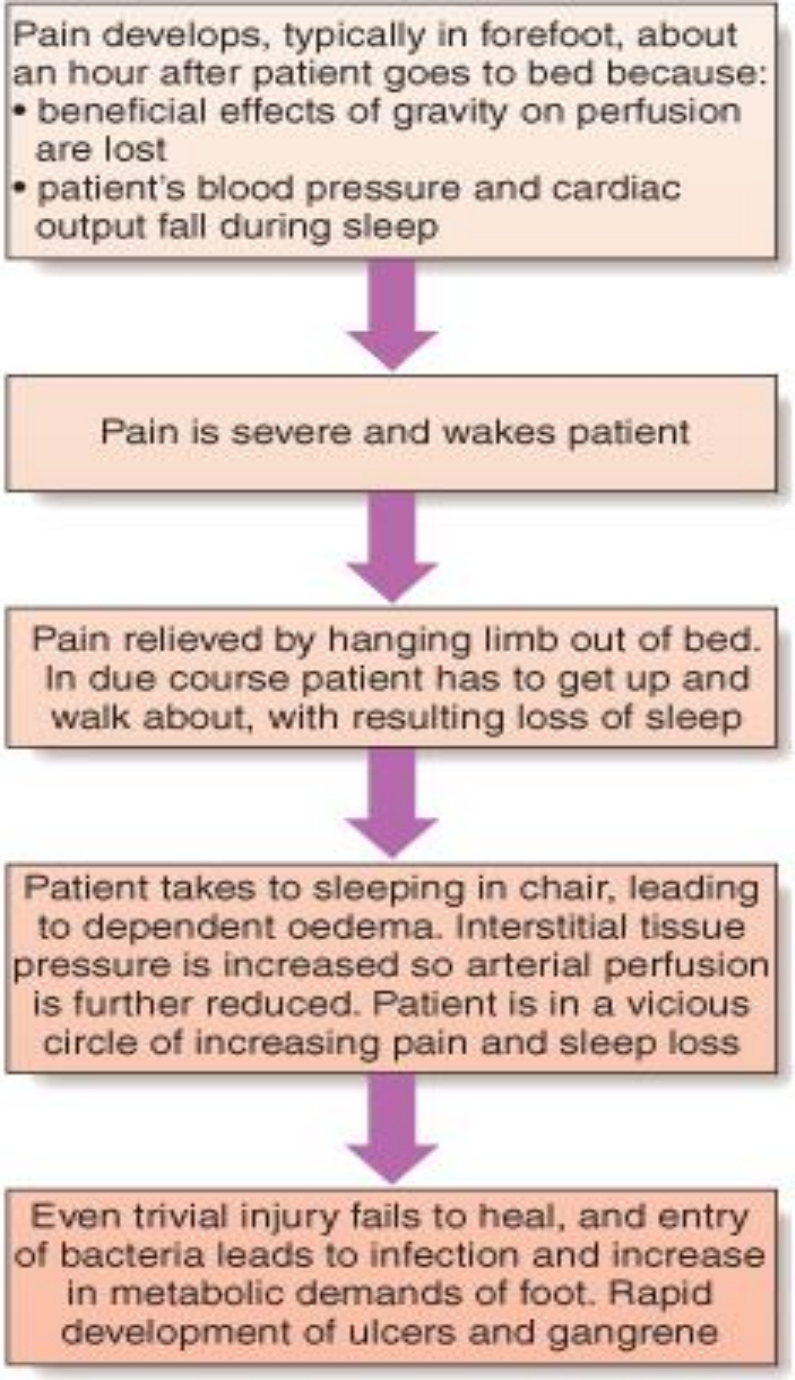
Ischaemic disease of the lower limbs

- Potency disorders
- Asymptomatic up to an advanced stage in DM angiopathies with peripheral polyneuropathy

Examination

CP compensation, murmurs, resistances in the abdomen

Lower extremity – pulsation, murmurs, trophics



Acute arterial occlusions – critical ischaemia

Advanced ischaemia – imminent loss of extremity or of its part

- **Rest pain –** analgesics
- **Partial relief after hanging down the extremity**
- **Defect on the extremity and low perfusion pressure**

Ankle BP less than 50 mm Hg, in DM the BP on the thumb is less than 30 mm Hg

Acute arterial occlusions - DG

- **Doppler ultrasonography (DUSG) –** blood flow at site of measurement

Ankle-brachial index (ABI) – normal 1.06 ± 0.15

Less than 0.9 - ischaemic disease of the lower limbs

- **Duplex USG –** a more perfect USG imaging
- **DS angiography –** golden standard
- **CT and spiral CT –** ao, abdom., pelvic tp.
- **MR AG**

Arterial occlusions - treatment

- **Dietary regimen:** elimination of risk factors
- **Physical training:** walking
- **DM angiopathy:** high-quality hygiene of the lower limbs
- **Interventional therapy:**
- **PTA, local thrombolysis**
- **Surgical therapies**

Reconstructional, endarterectomy, thrombectomy

Acute arterial occlusions - treatment

- **Pharmacotherapy**
- **Antiaggregation therapy**

ASA, ASA+dipyridamole, ticlopidine, clopidogrel

- **Anticoagulants:** after a bypass, thromboembolism
- **Vasodilators**

Pentoxifylline, naftidrofuryl, prostaglandins

Venous diseases

- **Phlebosclerosis**

Fibrosis of the intima, especially of superficial veins

- **Venous varices**

Locally varying venous diameter

In primary varices the valves are at first unaffected

Later on dilatation of the vein – venous insufficiency

Venous diseases – venous varices

- **RF's**

Age, genetics, obesity, static overloading of the lower limbs, increased intra-abdominal pressure (pregnancy)

Females/males 2-3:1

- **Clinical aspect + DG**

Pressure to pain, nightly cramps of extremities

Clin.exam., duplex USG, contrastive venography

Venous diseases – venous varices

- **Treatment**
- **Surgical techniques, sclerotization**

Selective elimination of affected veins, preservation of healthy veins

- **Physical treatment:** compressions, exercises, cold water from the foot tip proximally, not to be overexerted by long-time standing or sitting

Venous diseases – venous varices

- **Treatment**
- **Vein drugs**

Additive; modification of oedemas, of subjective feelings – remission of nightly cramps, and the like

Venous diseases – varicophlebitides, phlebitides

Inflammations of the venous wall with concomitant thrombosis

- Local striated swelling, erythema, pain on palpation
- Phlebitis of the great saphenous vein – a source of embolization into the pulmonary artery
- Local antithrombotics, antiphlogistic and antioedemic substances, bandages of lower limbs, anticoagulant therapy, vein drugs