Diseases of the Heart

- Congenital
- Acquired
- Diseases of endocardium
  - Endocarditis
  - Valvular diseases of the heart
- Diseases of myocardium
- Diseases of pericardium

Endocarditis

- Inflammation of the endocardial layer of the heart
  - Rheumatic (part of rheumatic fever)
  - Non-rheumatic
    - Non-infective
    - Atypical verrucous (Liebman–Sacks)
    - Non-bacterial thrombotic (cachectic, marantic)
    - Infective
    - Infective bacterial
    - Specific types of infective (tbc, syphilitic, fungal, viral, rickettsial)

Rheumatic fever

- A systemic non-suppurative inflammatory disease after infection (mostly upper RT) of β-haemolytic streptococcus of group A
- Immunologically conditioned disease, autoimmune reaction (2–3 weeks after infection)
- Affection of – heart, joints, CNS, skin, subcutaneous tissues

„rheumatism licks the joint, but bites the whole heart” Ernst–Charles Lasègue (French physician)
Rheumatic fever

- Children aged 5 – 15 years
- 2 – 3 weeks after streptococcal pharyngitis
- Fever with migratory polyarthritis
- Dg.: revised Jones criteria

Aschoff nodules – rheumatic granulomas

- Central fibrinoid dystrophy or necrosis of collagen fibers
- Palisading organized Anitschkow cells (macrophages) – large mononuclear cells with amphophilic cytoplasm, central round nucleus with central prominent chromatin:
  - In longitudinal section – serrated, caterpillar-like
  - In cross-section – owl-eye
- Giant multinucleated Aschoff cells – pathognomonic of RHD
- Giant cells with basophilic cytoplasm, with large multiple nuclei
- Admixture of Ly, PC, rare Neu
- Localized near to vessels

Rheumatic endocarditis – verrucous

A. Rheumatic valvular endocarditis:

1. Acute stage:
   - Macro: multiple warty verrucae (vegetations) along the line of closure of the leaflets and cusps (mostly mitral, less aortal, rare right heart valves)
   - Micro: oedema, vascularization of valves, infiltration of Ly, PC, rare Neu, Anitschkow cells, Aschoff nodules, vegetations – fibrin with superimposed platelet-thrombi

Rheumatic endocarditis – verrucous

A. Rheumatic valvular endocarditis:

2. Chronic stage:
   - Macro: deformities of affected valve/valves, stenosis of „fish mouth“, „button hole“
   - Micro: organisation of vegetations, valves – thickening – fibrous tissue, hyalinisation, calcification

Complications:

- Heart hypertrophy and dilatation
- Valvular heart diseases
- Cardiac failure
- Arrhythmias
- Thromboembolic complications
- Infective endocarditis

Causes of death in RHD:

- Cardiac failure
- Bacterial endocarditis
- Embolisation (into brain, kidneys, spleen from mural thrombus in LA in mitral stenosis)
- Sudden death as a result of massive thrombosis in LA, due to acute coronary insufficiency in aortic stenosis
Rheumatic endocarditis – verrucose

8. Rheumatic mural endocarditis
- Mostly mural endocardium in the posterior wall of LA above the posterior leaflet of the mitral valve (MacCallum’s patch)
- Macro: map-like area of thickened, roughened, wrinkled endocardium
- Mikro: oedema, fibrinoid degeneration, Ly, PC, Ma, Anitschkow cells, Aschoff nodules may be present

Rheumatic myocarditis

Macro:
- Acute stage: soft, flabby myocardium, especially LV
- Intermediate stage: small foci of necrosis, later Aschoff nodules

Micro: Aschoff nodules, later scarring in the vicinity of blood vessels

Rheumatic pericarditis

Macro:
- Fibrin deposits on the surface of both sheets of pericardium, slight amount of fibrinous exudate in the pericardial sac
- Healing with organisation – fibrous adhesions between two layers of pericardium – chronic adhesive pericarditis

Micro:
- Fibrin on the surface of serous membrane
- Infiltration of Ly, PC, Ma, rare Neu
- Aschoff nodules – later organisation, scarring

Extracardiac lesions

1. Polyarthritis (‘migratory polyarthritis’) – acute painful inflammation of synovial membranes of larger joints of the limbs, 2 or more joints involved at a time
2. Subcutaneous nodules – painless, fixed to deeper structures (ligaments, tendons, fascia), extensor surface of the wrists, elbows, ankles and knees
3. Erythema marginatum – on the trunk and prox. parts of extremities, non–pruritic erythematous rash, migratory character, erythema with central clearing, slightly elevated red margins
4. Rheumatic arteritis – coronary arteries, aorta, renal, mesenteric, cerebral arteries,…

Extracardiac lesions

5. Chorea minor – disordered and involuntary jerky movements of the trunk and the extremities, emotional instability
6. Rheumatic pneumonitis and pleuritis – pneumonitis with exsudative and proliferative changes, sero-fibrinous pleuritis
Inflammation of the endocardial layer of the heart

Rheumatic (part of rheumatic fever)

Non–rheumatic

Atypical verrucous (Liebman–Sacks)

Non–bacterial thrombotic (cachectic, marantic)

Infective

Infective bacterial

Specific types of infective (tbc, syphilitic, fungal, viral, rickettsial)

Non–infective endocarditis

Non–bacterial thrombotic (cachectic, marantic, terminal)

Patients with hypersoapable state (advanced cancer, chronic tbc, chronic sepsis, renal failure), allergy, hypovitaminosis C, endocardial trauma

Sterile vegetations on valvular endocardium (mostly mitral, less aortic and tricuspid, along the line of closure of the leaflets)

Macro: 1–5 mm vegetations, single/multiple, after healing – fibrous nodules (Lambl’s excrecences)

Micro: vegetations – fibrin with superimposed platelet thrombi, under vegetations – fibrinoid necrosis of endocardium, inflammatory infiltrate, pathognomonic Gross haematoxylin bodies

Infective bacterial endocarditis

Infection of valvular or mural endocardium

Friable infected vegetations

Depending upon the severity of infection:

1. Acute – ulcerous
2. Subacute – polypous (endocarditis lenta)

Non–infective endocarditis

Endocarditis

Non–infective endocarditis

Endocarditis

Infective bacterial endocarditis

Predisposing factors:

1. Conditions initiating transient bacteraemia, septicaemia (infection of periodontium – vigorous teeth brushing, tooth extraction,...; UGT infections – catheterisation, cystoscopy, delivery, abortion,...; GIT infections – surgery; skin infections; RT infections
2. Underlying heart disease (RHD, congenital heart diseases, prosthetic heart valves,...)
3. Impaired host defenses
Infective bacterial endocarditis

<table>
<thead>
<tr>
<th>Acute – ulcerous</th>
<th>Subacute – polypous</th>
</tr>
</thead>
<tbody>
<tr>
<td>▶ Duration &lt; 6 weeks, may be fatal</td>
<td>▶ Duration &gt; 6 weeks (months, years)</td>
</tr>
<tr>
<td>▶ S.aureus, β-hemol.strep.</td>
<td>▶ Streptococcus viridans</td>
</tr>
<tr>
<td>▶ Highly virulent bacteria</td>
<td>▶ Less virulent bacteria</td>
</tr>
<tr>
<td>▶ Usually previously normal valves</td>
<td>▶ Usually previously damaged valves</td>
</tr>
<tr>
<td>▶ Invasive, destructive, supplicative changes on valves</td>
<td>▶ Non-invasive, non-suppurative changes on valves</td>
</tr>
</tbody>
</table>

Acute endocarditis

- Macro: valvular tissue under vegetations necrotic with ulcerations

Acute endocarditis

- Micro:
  1. Outer layer – eosinophilic material – fibrin, platelets
  2. Basophilic zone – colonies of bacteria
  3. Deeper zone – valvular tissue under vegetations with non-specific inflammatory reaction (in subacute – also repair)

Systemic complications of IE

- Septic emboli
- Petechial bleeding (skin, conjunctiva)
- Osler’s nodes – painful, on the finger tips (SIE)
- Janeway’s spots – painless macular lesions on palms and soles (AIE)
- Focal necrotising glomerulonephritis (Löhlein)

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Valvular diseases of the heart

- Congenital
- Acquired
  - Causes: rheumatic heart disease (the most common), infective and non-infective endocarditis, syphilis, calcific aortic valve stenosis, calcification of mitral anulus fibrosus, myxomatous degeneration (floppy valve syndrome), carcinoid heart disease
  - Mostly mitral valve, less aortic, or both, less often right heart valves
Valvular diseases of the heart

- **Stenosis**: failure of a valve to open completely during diastole, obstruction to the forward flow of the blood
- **Insufficiency** (incompetence, regurgitation) – failure of a valve to close completely during systole, back flow of the blood

Mitral stenosis

- Mostly after RHD
- **Macro**: diffusely thickened valve leaflets by fibrous tissue/calcification
  - Deformity: „purse-string puckering“ (thickened free margins of leaflets) „button-hole“, „fish-mouth“ (severe narrowing)
- **Effects**:
  - Dilatation and hypertrophy of LA (pressure overload)
  - Normal-sized/hypertrophy of LV (reduced inflow of blood)
  - Pulmonary hypertension (lung oedema, chronic lung venostasis, hypertrophy and dilatation of RV, dilatation of RA)
- **Clin.**:
  - Exertional dyspnoea, later orthopnoea (pulmonary hypertension)

Mitral insufficiency

- Mostly after RHD
- **Macro**: deformities, retraction of the valve leaflets, fusion of commissures
- **Effects**:
  - Marked dilatation of LA (volume overload)
  - Dilatation and hypertrophy of LV (volume overload)
  - Pulmonary hypertension (lung oedema, chronic lung venostasis, hypertrophy and dilatation of RV, dilatation of RA)
- **Clin.**:
  - Exertional dyspnoea, later orthopnoea (pulmonary hypertension)
  - Weakness, fatigue (decreased cardiac output)

Aortic stenosis

1. **Calcific** – calcium deposition in scar of previously damaged valve
2. **Non-calcific** – mostly after RHD
- **Macro**: fibrous thickening, calcification of leaflets
- **Effects**:
  - Concentric hypertrophy of LV (pressure overload), later dilatation of LV (eccentric hypertrophy)
  - Pulmonary hypertension, right heart changes (similar to mitral stenosis)
- **Clin.**:
  - Exertional dyspnoea (pulmonary hypertension)
  - Angina pectoris (increased demand of hypertrophied myocardium)
  - Syncope (decreased cardiac output, brain ischemia)

Aortic insufficiency

- Mostly after RHD
- **Macro**: deformities, thickening and shortening of valve leaflets, distended deformed aortic root
- **Effects**:
  - Hypertrophy and dilatation of LV (volume overload)
  - Later changes of LA, pulmonary hypertension, changes of right heart
- **Clin.**:
  - Awareness of the heart beatings (increased output volume)
  - Corrigan’s puls (strong puls with high amplitude – caused by high systolic volume and decreased peripheral resistance)
  - Traube’s sign (systolic murmur over the femoral artery)
  - Duroziet’s sign (systolic/diastolic murmur over the femoral artery)
  - Angina pectoris (increased demand of hypertrophied myocardium)

Myocardial diseases

1. **Myocarditis** – inflammation of myocardium
2. **Cardiomyopathy** – non-inflammation myocardial diseases
   - Primary – unknown etiology
   - Secondary – known etiology
3. **Involvement of the myocardium in other diseases**:
   - Ischaemic heart disease
   - Hypertensive heart disease
   - Rheumatic heart disease
Myocardial diseases
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Myocarditis

Classifications:
- Interstitial, parenchymatous
- Specific, non-specific
- Acute, subacute, chronic

Etiologic classification:
1. Infective myocarditis
2. Idiopathic (Fiedler’s) myocarditis
3. Myocarditis in connective tissue diseases
4. Other types of myocarditis

Infective myocarditis

- **Viral (non-suppurative)**
  - Influenza virus, polyomylitis, inf. mononucleosis, etc.
  - Myocardial damage – direct viral cytotoxic effect or cell-mediated immune reaction
  - Macro: pale, flabby myocardium, chamber dilatation, necrotic areas
  - Micro: oedema, Ma, Ly, necrosis

- **Bacterial (suppurative)**
  - Hematogenous route – pyogenic bacteria (Staf.aureus, Strep.pyogenes), spread from inf. endocarditis
  - Macro: abscesses (localized), phlegmona (diffuse)
  - Micro: Neu, admixture of Ma, Ly, PC, degeneration or necrosis

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Myocarditis

1. Infective myocarditis
2. Idiopathic (Fiedler’s) myocarditis
3. Myocarditis in connective tissue diseases
   - RA, SLE, polyarteritis nodosa, dermatomyositis, scleroderma
4. Other types of myocarditis
   - Caused by physical and chemical factors, drugs, immunologic factors, metabolic disorders

Myocardial diseases

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Cardiomyopathy (KMP)

- Primary – diseases of unknown etiology
  1. Idiopathic dilated (congestive) KMP
  2. Idiopathic hypertrophic KMP
  3. Idiopathic restrictive (obliterative, infiltrative) KMP

- Secondary – diseases with known etiology
  - Nutritional disorders (chronic alcoholism, thiamine deficiency), toxic chemicals (cobalt, arsenic), drugs (cytostatics, adriamycin), metabolic diseases (amyloidosis, glykogenosis), neuromuscular diseases (muscular dystrophy), tumor infiltration, connective tissue diseases (RA, SLE, SS, dermatomyositis)

Idiopathic dilated KMP

- Macro: enlarged heavy heart (1000g), prominent dilatation of all the four heart chambers, ventricular wall hypertrophy, focal endocardial thickening, mural trombi

Idiopathic hypertrophic KMP

- = asymmetrical hypertrophy, hypertrophic subaortic stenosis, Teare’s disease
- Mostly in adults (25 – 50 years)
- Etiology unknown
- Associations with genetic factors (autosomal dominant inheritance, inherited mutations of genes for sarcomere proteins), increased circulating level of catecholamins
- Clin: often asymptomatic, becomes symptomatic due to heavy physical activity – dyspnoea, angina pectoris, congestive heart failure, sudden death
Idiopathic hypertrophic KMP
- Macro: enlarged heavier heart, ventricular volume normal/smaller, prominent myocardial hypertrophy, asymmetric interventricular septum hypertrophy
  a) Non-obstructive type – hypertrophy of apical part of septum
  b) Obstructive type – hypertrophy up to the level of mitral valve
      - obstruction of LV outflow, subvalvular, subaortic stenosis
- Micro: cardiomyocytes disorganisation in the ventricular septum, irregularly and haphazardly arranged bundles of myocardial fibres, bands of fibrous tissue, cardiomyocytes – hypertrophic with large prominent nuclei

Idiopathic restrictive (obliterative, infiltrative) KMP
- Heterogeneous group of diseases, abnormal diastolic function, restriction in ventricular filling due to reduction in the ventricular volume, rigid ventricular wall
  1. Cardiac amyloidosis
      - In any form of systemic amyloidosis / isolated heart amyloidosis
  2. Endocardial fibroelastosis
  3. Endomyocardial fibrosis
  4. Löffler’s endocarditis
  5. Other forms

Endomyocardial fibrosis
- Etiology – ?, possible anoxia, genetic disorder, pressure overload, myocardial injury, tumors of pancreas, carcinoid (serotonin production)
  - Infantile form - congenital, children under 2 years
  - Adult form
  - Macro - diffuse or focal rigid pearly-white thickening of the mural endocardium

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Ischaemic heart disease

- Acute or chronic form of cardiac disability
- Imbalance between the myocardial supply and demand for oxygenated blood
- Etiopathogenesis:
  1. Coronary atherosclerosis (atherosclerotic plaques)
  2. Complications of coronary atherosclerosis (plaque haemorrhage, fissuring, plaque ulceration, thrombosis, embolisation of atheromatous debris, local platelets aggregation - spasm of coronary artery)
  3. Non-atherosclerotic causes (vasospasm, stenosis of coronary ostia, arteritis, embolism, thrombotic disease, trauma, aneurysms, compression)

Angina pectoris

- Clinical syndrome of IHD resulting from transient myocardial ischaemia
  1. Stable (typical)
  2. Unstable (crescendo)
  3. Prinzmetal’s (variant, vasospastic)

Acute coronary syndrome

<table>
<thead>
<tr>
<th>Time</th>
<th>Macroscopy</th>
<th>Microscopy</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–6 hours</td>
<td>No change Macroreaction (TTC/NBT)</td>
<td>No significant change, stretching and waviness of myocardial fibres</td>
</tr>
<tr>
<td>6–12 hours</td>
<td>No change Macroreaction (TTC/NBT)</td>
<td>Vascular degeneration, beginning of coagulative necrosis, infiltration with neutrophils, oedema and haemorrhage</td>
</tr>
<tr>
<td>24 hours</td>
<td>Cyanotic red-purple area of haemorrhage</td>
<td>Progression of coagulative necrosis, marginal infiltration with neutrophils</td>
</tr>
<tr>
<td>48–72 hours</td>
<td>Pale, hyperaemia, yellow soft centre</td>
<td>Complete coagulative necrosis, well developed neutrophilic infiltrate, spread in the center</td>
</tr>
<tr>
<td>3–7 days</td>
<td>Hyperaemic rim, yellow soft centre</td>
<td>Decrease of neus, resorption of necrosed fibres by Ma, beginning of healing vessel and fibroblasts proliferation</td>
</tr>
<tr>
<td>10. day</td>
<td>Red-purple periphery</td>
<td>Most of necrosed fibres removed, fibrovascular reaction at periphery, pigmented Ma, Eo, Ly, PC</td>
</tr>
<tr>
<td>14. day</td>
<td>Most of necrosed fibres removed, fibrocollagenic tissue at the periphery</td>
<td></td>
</tr>
<tr>
<td>3. week</td>
<td>Necrosed debris removed, more ingrowth of fibrocollagenic tissue</td>
<td></td>
</tr>
<tr>
<td>4–6. week</td>
<td>Thin, gray-white, hard shrunken fibrous scar</td>
<td>Scar maturation, decreased vessels, Ly, PC, pigmented Ma</td>
</tr>
</tbody>
</table>

Pathologic changes in MI
Complications after MI

- Arrhythmias – sinus tachycardia, sinus bradycardia, atrial fibrillation, ventricular fibrillation
- Congestive heart failure – failure of RV, LV, both
- Cardiogenic shock – hypotension (systolic pressure less than 80 mmHg), peripheral circulatory failure, oliguria
- Mural thrombosis, embolism – intracardiac thrombosis (endocardial damage, stagnation of blood due to bradycardia), leg deep vein thrombosis – embolism
- Rupture – haemopericardium, heart tamponade
- Cardiac aneurysm – in scar tissue
- Pericarditis – aseptic fibrinous (pericarditis epistenopericardiaca)
- Postmyocardial infarction syndrome (Dressler’s sy) – pleuropericardial pain with fever, pleuritis, pericarditis, pneumonitis (autoimmune reaction?)

Chronic ischaemic heart disease

- Focal or diffuse myocardial fibrosis
- Etiopathogenesis: coronary atherosclerosis, embolism, arteritis, myocarditis
  - Healing of minute infarctions of small groups of cardiomyocytes
- Macro: foci of gray–white fibrosis in brown myocardium
- Micro: areas of diffuse myocardial fibrosis especially around small vessels, foci of myocyteysis, foci of brown myocardial atrophy, atherosclerosis of coronary arteries

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   - Rheumatic heart disease

Hypertensive heart disease

- Etiopathogenesis: pressure overload caused by long–lasting systemic hypertension → hypertrophy of LV (hypertrophy of cardiomyocytes – ↑ production of myofilaments, myofibrils, other cell organelles, nuclear enlargement)
- Macro: enlarged heart, thickening of LV, papillary muscles, trabeculae
- Concentric (without dilatation) → eccentric (with dilatation) – in decompensation
- Micro: enlargement and dystrophic changes of myocardial fibres, focal fibrosis

Pulmonary heart (cor pulmonale)

- Pathological change of RV caused by lung disorders
- Etiopathogenesis: pressure overload due to pulmonary hypertension
- Acute cor pulmonale – dilatation of RV due to massive pulmonary embolism
- Chronic cor pulmonale – hypertrophy of RV due to chronic pulmonary hypertension (lung emphysema, chron. bronchitis, pulmonary tbc, pneumoconiosis, cystic fibrosis, successive pulmonary embolism)
  - Hypertrophy of RV → dilatation of RV in decompensation

Diseases of pericardium

- Accumulation of fluid in pericardial sac – may lead to heart tamponade – impaired diastolic filling
  - Hydropericardium – non-inflammatory fluid in pericardial sac
  - Haemopericardium – blood in pericardial sac
- Pericarditis
Diseases of pericardium

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- Pericarditis

Accumulation of fluid in pericardial sac

- Hydropericardium – non-inflammatory fluid in pericardial sac
  1. Serous effusion – clear, straw-coloured
  2. Serosanguineous effusion – after CPR, trauma to chest
  3. Chylous effusion – milky
  4. Cholesterol effusion – with cholesterol crystals

- Hemopericardium – blood in pericardial sac
  - Rupture of the heart in MI, rupture of dissecting aneurysm, haemorrhagic diathesis, trauma

Diseases of pericardium

- Accumulation of fluid in pericardial sac
  - may lead to heart tamponade - impaired diastolic filling
  - Hydropericardium – non-inflammatory fluid in pericardial sac
  - Haemopericardium – blood in pericardial sac

- Pericarditis

Pericarditis

- Inflammation of both pericardial layers

- Mostly secondary to heart diseases or systemic diseases

- Primary (idiopathic) pericarditis – rare

  - Acute
  - Chronic

Acute pericarditis

1. Serous – serous exudate (more proteins, higher specific gravity than transudate)
   - viruses, RF, RA, SLE, tumors, tbc pericarditis (early stage)
2. Fibrinous and sero-fibrinous – fibrin in exudate
   - uraemia, MI (pericarditis epistenopericardiaca), RF, trauma, surgery
   - Fibrin plaques on pericardium – „hairy“ heart – healing by organisation – thickening of pericardium, adhesions
3. Purulent – creamy pus exudate
   - Pyogenic bacteria, less fungi and parasites
4. Haemorrhagic pericarditis – Ery in exudate
   - tumors, haemorrhagic diathesis, tbc

Chronic pericarditis (PK)

1. Tuberculous – granulomatous inflammation, tuberculomas
   - Healing by organisation – fibrosis, calcifications, adhesions, chronic constrictive PK
2. Chronic adhesive
   - Repair stage of fibrinous, purulent, haemorrhagic PK – formation of adhesions – obliteration of pericardial sac
   - Function of the heart is not restricted, hypertrophy and dilatation of the heart may be due to increased workload
3. Chronic constrictive
   - After tbc PK, purulent PK, haemopericardium – fibrous/fibro-calcific thickening of pericardium (0.5–1 cm)
   - Mechanically restricts the function of the heart, reduced cardiac output
   - Without cardiac hypertrophy and dilatation, heart normal/smaller
4. Pericardial plaques (milk spots) – areas of fibrous organisation, perhaps healing of preceding PK