Cardiovascular (Medicine)

See ECG notes

Symptoms: **Chest pain, breathlessness, palpitations, syncope, fatigue, peripheral oedema.**

- Left heart failure is the most common cardiac cause of exertional dyspnoea. Orthopnoea and paroxysmal nocturnal dyspnoea.
- Palpitations are an awareness of the heartbeat. Can be a **cardiac arrhythmia**.
- **Syncope** is a temporary impairment of consciousness due to inadequate cerebral blood flow. Faint or vasovagal attack.
- **Grade 1 - 4 cardiac status. Grade 1 uncompromised Grade 4 severely compromised.**
- **Chest pain caused by:** angina pectoris, acute coronary syndrome, pulmonary embolism, aortic dissection, pericarditis, musculoskeletal and gasto-oesophageal reflux disorder (GORD).

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<th>Central chest pain</th>
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Heart failure can cause salt and water retention leading to oedema, which in ambulant patients is more prominent over the ankles.

X-ray, ECG, stress ECG, 24-hour taped ECG, tilt testing, echocardiography, cardiac nuclear imaging, CT, MRI, cardiac catherization.

Kerley B lines indicate pulmonary oedema.

25mm/s

**P wave**

If the p wave is broad - more than 0.12s – left atrial enlargement (mitral stenosis)
If it is taller - 2.5mm – right atrial enlargement (pulmonary hypertension)
Absent in sino-atrial block
Replaced by flutter
Coronary artery bypass grafting

Left or right internal mammary artery is used to bypass stenoses in the left anterior descending or right coronary artery respectively. Sometimes the saphenous vein from the leg is anastamosed between the proximal aorta and coronary artery distal to the obstruction.

Percutaneous coronary intervention (PCI)

Small inflatable balloons are used to inflate and dilate lesions. Stent placement reduces the risk of acute vessel closure and restenosis rates. This technique is widely applied for angina resulting from isolated, proximal, non-calcified atheromatous plaques.

Acute Coronary Syndromes


1) Unstable angina
2) Non-ST-elevation myocardial infarction (NSTEMI)
3) ST elevation myocardial infarction (STEMI)

Unstable angina differs from NSTEMI in that in the latter the occluding thrombus is sufficient to cause myocardial damage and an elevation in serum markers of myocardial injury (troponin and creatine kinase). Serum troponins are taken twice. Once on admission, once after 12 hours. A normal troponin level after 12 hours suggests unstable angina.

Most severe is ST elevation myocardial infarction.

Patients may present with acute coronary syndromes without having previous anginas.

Differential: GORD, musculoskeletal pain, pulmonary embolus, aortic dissection.

Treatment:

(i) Anti-platelet therapy – aspirin (first dose 300mg, second dose 75mg) clopidogrel 300mg
(ii) Antithrombins – heparin (low molecular weight heparin is actually more efficient than unfractioned heparin) – at least for 48 hours.
(iii) Anti-ischaemia agents – Nitrates (Glyceryl trinitrate), β-blockers.
(iv) Plaque stabilisation – Statins and an ACE inhibitor

EMERGENCY:

- Oxygen
- Intravenous access
- Blood investigations (creatinine, electrolytes, glucose, lipids)
- ECG
- Glyceryl trinitrate sublingual
Mitral Stenosis

Aetiology
Usually as a result of rheumatic heart disease. Sometimes many years after the episode of rheumatic fever. Primarily affects women.

Pathophysiology
Thickening and immobility of the valve leaflets leads to obstruction of blood flow from the left atrium to the left ventricle. As a result there is an increase in left atrial pressure, pulmonary hypertension and right heart dysfunction. (Pulmonary oedema)
Atrial fibrillation is common due to the elevation of left atrial pressure and dilatation. Thrombus may form in the dilated atrium and give rise to systemic emboli.

Pulmonary arterial vasoconstriction leads to pulmonary hypertension and eventually right ventricular hypertrophy, dilatation and failure.

Symptoms
Exertional dyspnoea which becomes progressively more severe.
Haemoptysis
Atrial fibrillation
Pulmonary oedema
Right hearted side failure
Fatigue and Lower limb oedema

Basically structures that are backwards from the mitral valve are affected.

Signs
Malar flush (severe mitral stenosis)
Irregular pulse (possibly due to atrial fibrillation)
Tapping apex beat. Palpable first heart sound and left ventricular backward displacement as a result of enlarging right ventricle.
Auscultation at apex beat of a loud first sound.
Presence of a loud second sound, elevation of JVP, peripheral oedema indicates right ventricular overload.

Investigations
Chest X-ray
ECG - possibly a bifid p wave.
Echocardiography

Mechanical relief if severe pulmonary hypertension develops. Percutaneous balloon valvotomy to split the commissures. Maybe mitral valve replacement might be necessary. Latter is only performed in fulminant cases where there is associated mitral regurgitation, evidence of thrombosis in left atrium despite anti-coagulation or a badly calcified valve.

Percutaneous balloon valvotomy – apparatus inserted through femoral vein.

Mitral Regurgitation

Aetiology
Prolapsing mitral valve is the most common cause in the developed world but rheumatic heart disease continues to be a common cause in the developing world.

Causes of Mitral regurgitation: Rheumatic disease, mitral valve prolapse, Ehlers-Danlos, Infective endocarditis, rupture of chordate tendineae, rupture of papillary muscle, Marfan’s syndrome.

Symptoms
Pulmonary oedema. Dyspnoea, fatigue and lethargy (reduced cardiac output). In later stages symptoms of right heart failure also occur and eventually lead to congestive heart failure. Thromboembolism is less common than with mitral stenosis, although infective endocarditis is much more common.

Signs
First beat is soft. A pansystolic murmur is heard (palpated as a thrill), loudest at the apex beat and radiating to the axilla. A third sound may be heard as a result of rapid filling of the dilated left ventricle in early diastole.
Infective endocarditis

- Infection of endocardium or vascular endothelium of the heart
- Infection occurs in the following:
  - On valves which have a congenital or acquired defect (usually on the left side of the heart. Right sided endocarditis is more common in drug abusers.
  - On normal valves with virulent organisms such as *Streptococcus pneumoniae* or *Staphylococcus aureus*.
  - On prosthetic valves when infection may be ‘early’ (within 60 days of surgery) or ‘late’ (following bacteraemia). Infected prosthetic valves need to be replaced
  - In association with a ventricular septal defect of persistent ductus arteriosus. (Pulmonary artery to aortic artery)

**Aetiology**

Staphylococcus aureus and enterococci are most common causes. Blood cultures remain negative 5-10% in patients with infective endocarditis.

Slight pyrexia, vascular phenomena, immunological phenomena, microbiological evidence

Echocardiogram shows intracardiac mass on a valve or supporting structures or abscesses.

**Pathology**

A mass of fibrin, platelets and infectious organisms forms vegetations along the edges of the valve. Virulent organisms destroy the valve, producing regurgitation and worsening heart failure.

**Symptoms**

Systemic features of infection: night sweats, malaise, fever, weight loss, anaemia.

Valve destruction, leading to heart failure and changing heart murmors.

Vascular phenomena.
Pulmonary Heart Disease

Lung circulation offers a low resistance to flow compared to the systemic circulation and the normal mean pulmonary artery pressure at rest is **10-14mmHg** (compared to mean systemic arterial pressure of about 90mmHg).

Pulmonary hypertension = >25mmHg at rest.

**Aetiology**

Occurs as an increase in pulmonary vascular resistance or an increase in pulmonary blood flow. Hereditary, idiopathic, SLE, rheumatoid arthritis, cocaine, amphetamines, HIV infection, portal hypertension (portopulmonary hypertension), congenital heart disease.

Pulmonary hypertension also occurs secondarily to left heart disease: valvular, systolic dysfunction.

Lung disease, hypoxia eg COPD, obstructive sleep apnoea, lung fibrosis.

Thromboembolic occlusion of proximal or distal vasculature.

Sarcoidosis, glycogen storage disease.

**Clinical features**

Dyspnoea, lethargy, fatigue

Right ventricular failure, peripheral oedema and abdominal pain from hepatic congestion. On examination there is a loud pulmonary second sound, with right parasternal heave (caused by right ventricular hypertrophy).

In advanced disease there are features of right heart failure (cor pulmonale), elevated JVP, pleural effusion, ascites, pulsatile liver, peripheral oedema.
Arterial and Venous Disease

Aortic aneurysms
Permanent localized dilatation of an artery. They may be asymptomatic or cause symptoms by pressure effects or vessel rupture, occasionally with fistula formation, or they may be a source of emboli. Aortic aneurysms (vessel diameter > 3cm) are usually abdominal and most result from a degenerative process and present in elderly men. Some are the result of connective tissue disease.

Abdominal
May be asymptomatic (found as a pulsating mass on abdominal examination or as calcification on a plain x-ray), cause symptoms due to pressure effects or rupture. The later is a surgical emergency presenting with epigastric pain radiating to the back, and hypovolaemic shock. Diagnosis is by ultrasonography or CT scan. Surgical replacement of the aneurismal segment with a prosthetic graft is indicated for a symptomatic aneurysm or large asymptomatic aneurysms (>5.5cm). EVAR. Endovascular repair with stenting.

Thoracic
Cystic medial necrosis and atherosclerosis are the usual causes of thoracic aneurysms. Can be asymptomatic but can cause pressure on local structures (causing back pain, dysphagia and cough).

Dissecting aortic aneurysm
Aortic dissection results from a tear in the intima. Blood under high pressure creates a false lumen in the diseased media. Typically there is an abrupt onset of severe, tearing chest pain radiating to the back. Chest x-ray shows a widened mediastinum. Transoesophageal echocardiography or MRI.

Raynaud’s disease and phenomenon
Intermittent spasm in the arteries supplying the fingers and toes. Precipitated in cold and relieved by heat. Initial pallor followed by hyperaemia. Usually has no underlying cause. But can be caused by β-blocker therapy. Stop smoking and stop taking β-blockers. Lumbar sympathectomy + prostacyclin infusions.