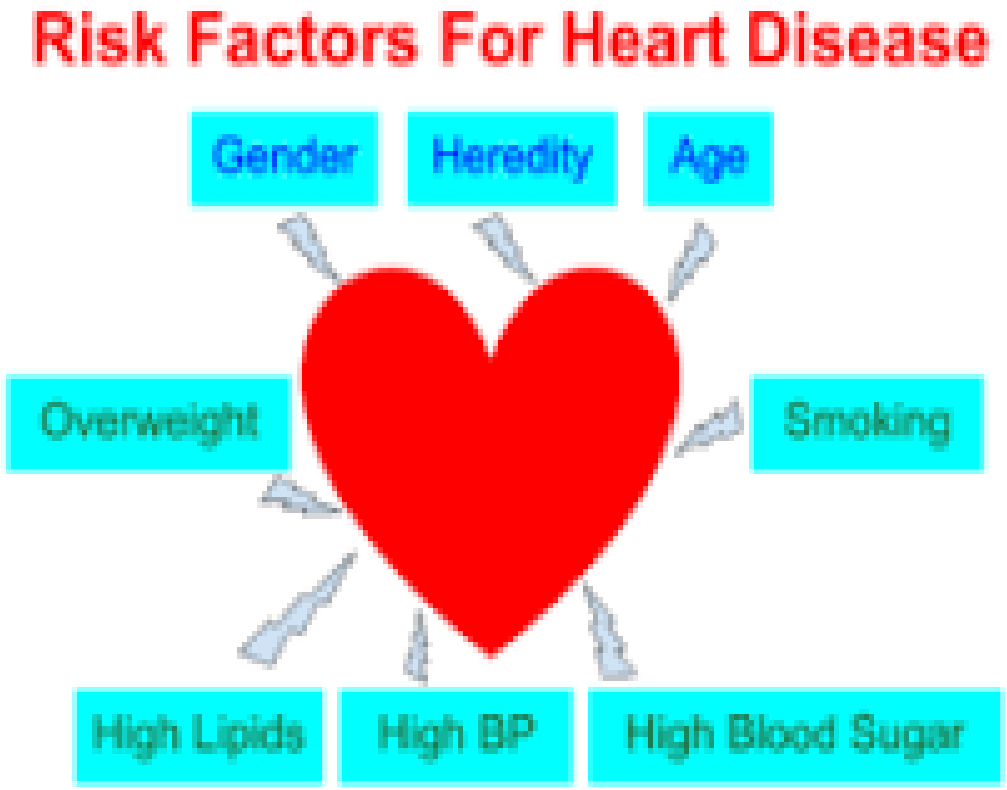
Dr abbas Hamad

pulmonologist

- المحاضرة السابعة – المرحلة الثانية
- الطب الباطني – تقنيات التخدير

- Cardiovascular diseases

Risk factors for cardiovascular diseases



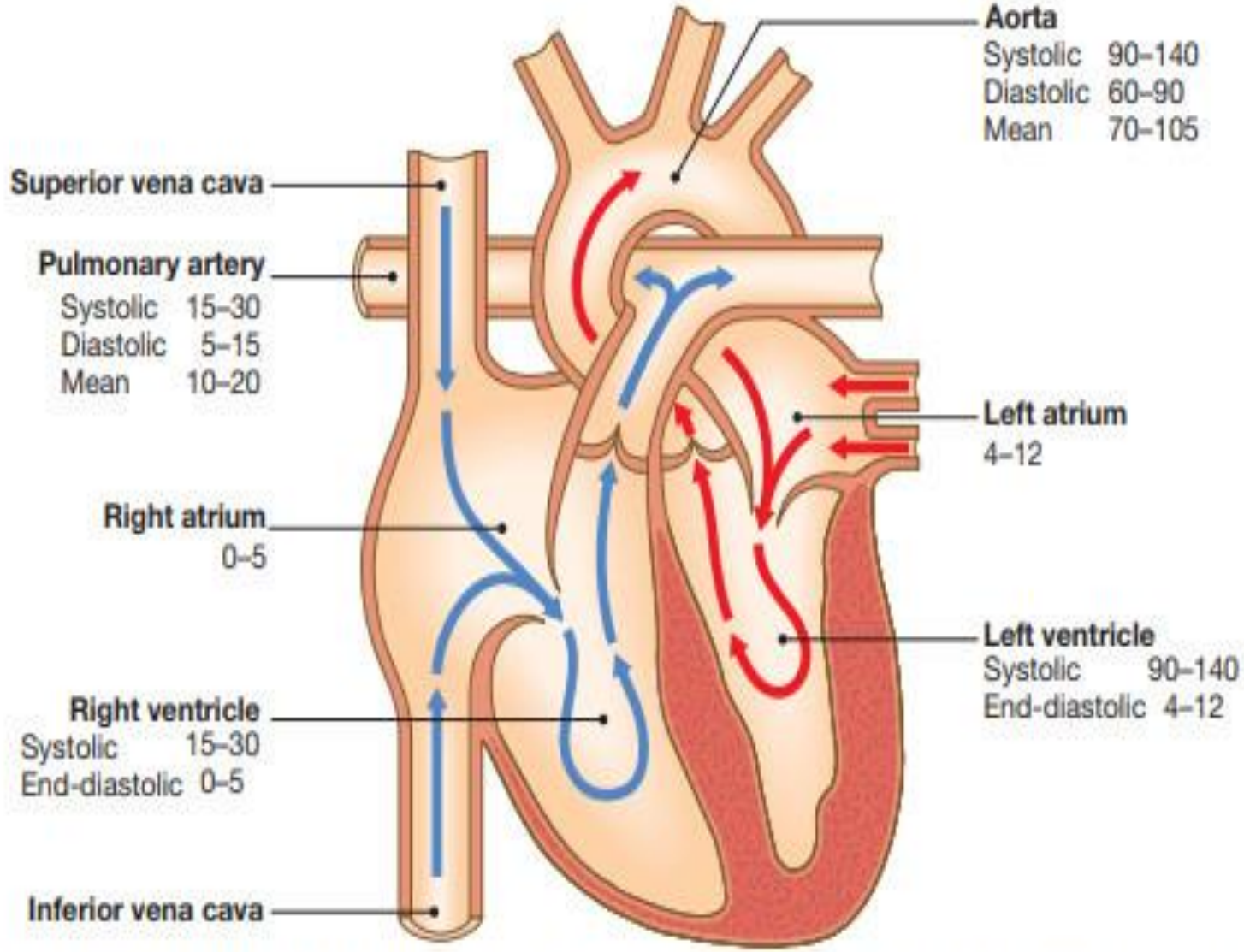


Fig. 16.1 Direction of blood flow through the heart. The blue arrows show deoxygenated blood moving through the right heart to the lungs. The red arrows show oxygenated blood moving from the lungs to the systemic circulation. The normal pressures are shown for each chamber in mmHg.

Coronary artery disease(CAD)

Coronary artery disease **(CAD)** is the most Coronary artery disease • common cause of angina and acute coronary syndrome and the most common cause of death worldwide. The World Health Organisation (WHO) has estimated that **3.8** million men and **3.4** million women die from cardiovascular disease (CVD) each year, and since 1990, more people have died from CVD than any other cause.

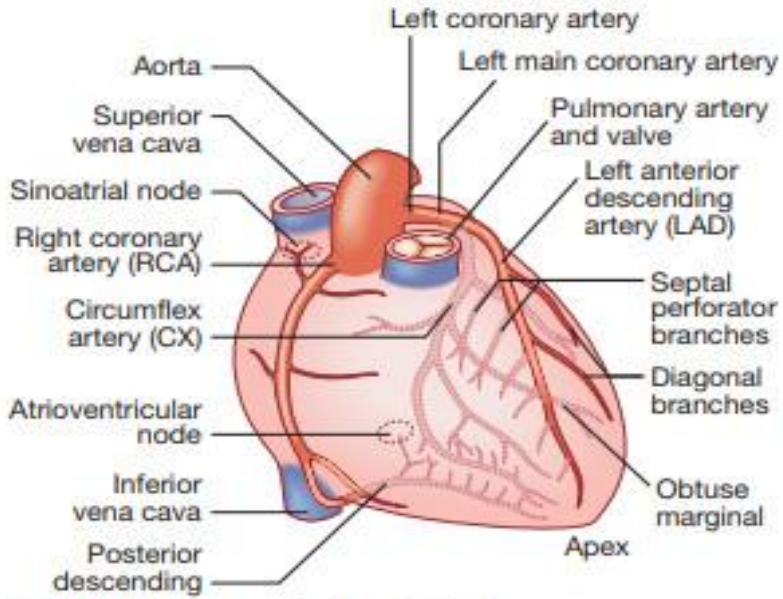


Fig. 16.3 The coronary arteries: anterior view.

16.36 Coronary artery disease: clinical manifestations and pathology

Clinical problem	Pathology
Stable angina	Ischaemia due to fixed atheromatous stenosis of one or more coronary arteries
Unstable angina	Ischaemia caused by dynamic obstruction of a coronary artery due to plaque rupture or erosion with superimposed thrombosis
Myocardial infarction	Myocardial necrosis caused by acute occlusion of a coronary artery due to plaque rupture or erosion with superimposed thrombosis
Heart failure	Myocardial dysfunction due to infarction or ischaemia
Arrhythmia	Altered conduction due to ischaemia or infarction
Sudden death	Ventricular arrhythmia, asystole or massive myocardial infarction

Angina pectoris

Angina pectoris is a symptom complex caused by transient myocardial ischaemia, which occurs whenever there is an imbalance between myocardial oxygen supply and demand

Pathogenesis **Atherosclerosis** is by far the most common cause of angina pectoris. Angina may also occur in aortic valve disease and hypertrophic cardiomyopathy, and when the coronary arteries are involved with vasculitis or aortitis.

Approximately 10% of patients who report stable angina on effort have normal coronary arteries on angiography.

Clinical features

- Stable angina is characterized by central chest pain, discomfort or breathlessness that is predictably precipitated by exertion or other forms of stress and is promptly relieved by rest
- Some patients find the discomfort comes when they start walking and that later it does not return despite greater effort (‘warm-up angina’).

Investigations

Ecg

Cxr

Echo

Blood test like s. troponin

Anti-anginal drug therapy

1- nitrates like Sublingual GTN . Oral isosorbide dinitrate. Oral • isosorbide mononitrate

2-Beta-blockers like like metoprolol and bisoprolol •

3- Calcium channel antagonists like Nifedipine 5–20 mg 3 times daily* • Nicardipine 20–40 mg 3 times daily. Amlodipine 2.5–10 mg daily Verapamil 40–80 mg 3 times daily and Diltiazem

4- Non-pharmacological treatments •

Percutaneous coronary intervention (PCI) •

Acute coronary syndrome

- **Acute coronary syndrome** is a term that encompasses both unstable angina and myocardial infarction. Unstable angina is characterized by new-onset or rapidly worsening angina (crescendo angina), angina on minimal exertion or angina at rest in the absence of myocardial damage. Myocardial infarction differs from unstable angina, since there is evidence of myocardial necrosis. The term acute myocardial infarction (MI) should be used when there is evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischaemia

16.47 Criteria for diagnosis of an acute myocardial infarction (MI)

- Detection of a rise and/or fall of cardiac biomarker values (preferably cardiac troponin (cTn)), with at least one value above the 99th centile upper reference limit (URL) and with at least one of the following:
    1. Symptoms of ischaemia
    2. New or presumed new significant ST segment–T wave (ST–T) changes or new left bundle branch block (LBBB)
    3. Development of pathological Q waves in the ECG
    4. Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality
    5. Identification of an intracoronary thrombus by angiography or postmortem

16.49 Clinical features of acute coronary syndromes

Symptoms

- Prolonged cardiac pain: chest, throat, arms, epigastrium or back
- Anxiety and fear of impending death
- Nausea and vomiting
- Breathlessness
- Collapse/syncope

Physical signs

Signs of sympathetic activation

- Pallor
- Sweating
- Tachycardia

Signs of vagal activation

- Vomiting
- Bradycardia

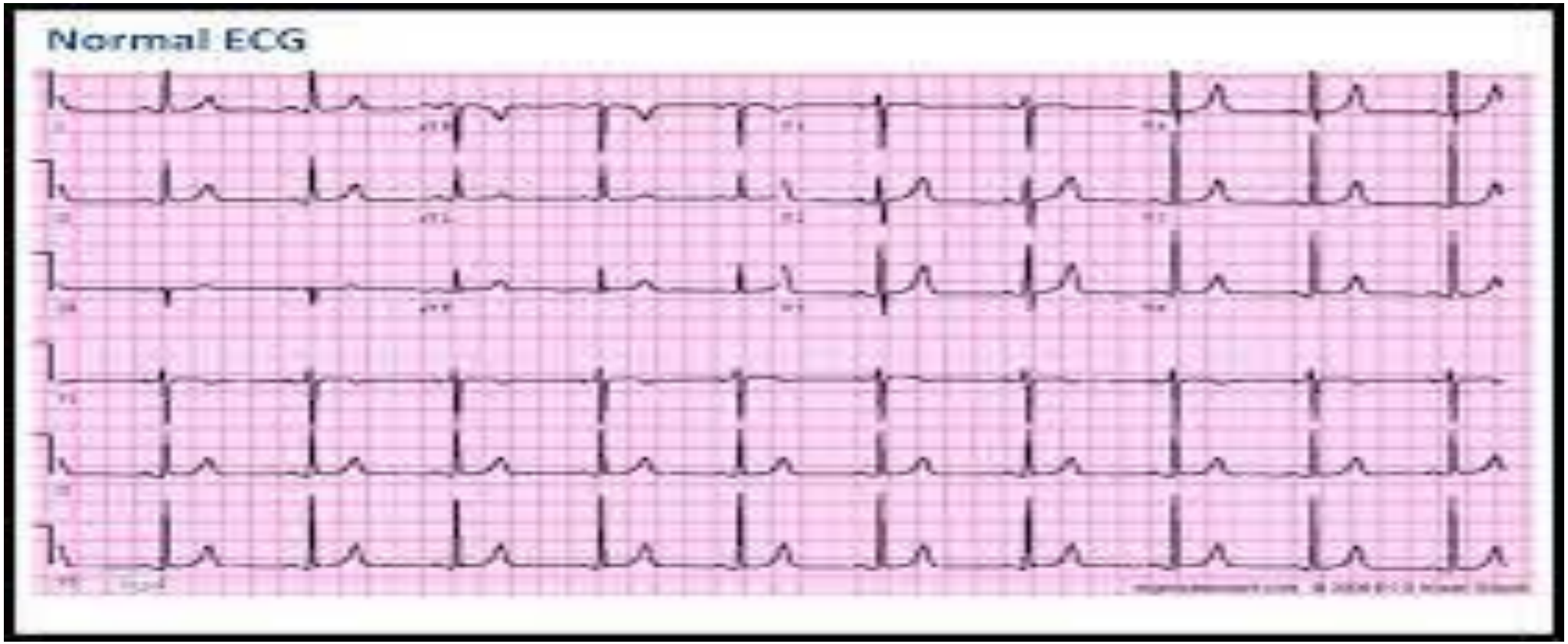
Signs of impaired myocardial function

- Hypotension, oliguria, cold peripheries
- Narrow pulse pressure
- Raised jugular venous pressure
- Third heart sound
- Quiet first heart sound
- Diffuse apical impulse
- Lung crepitations

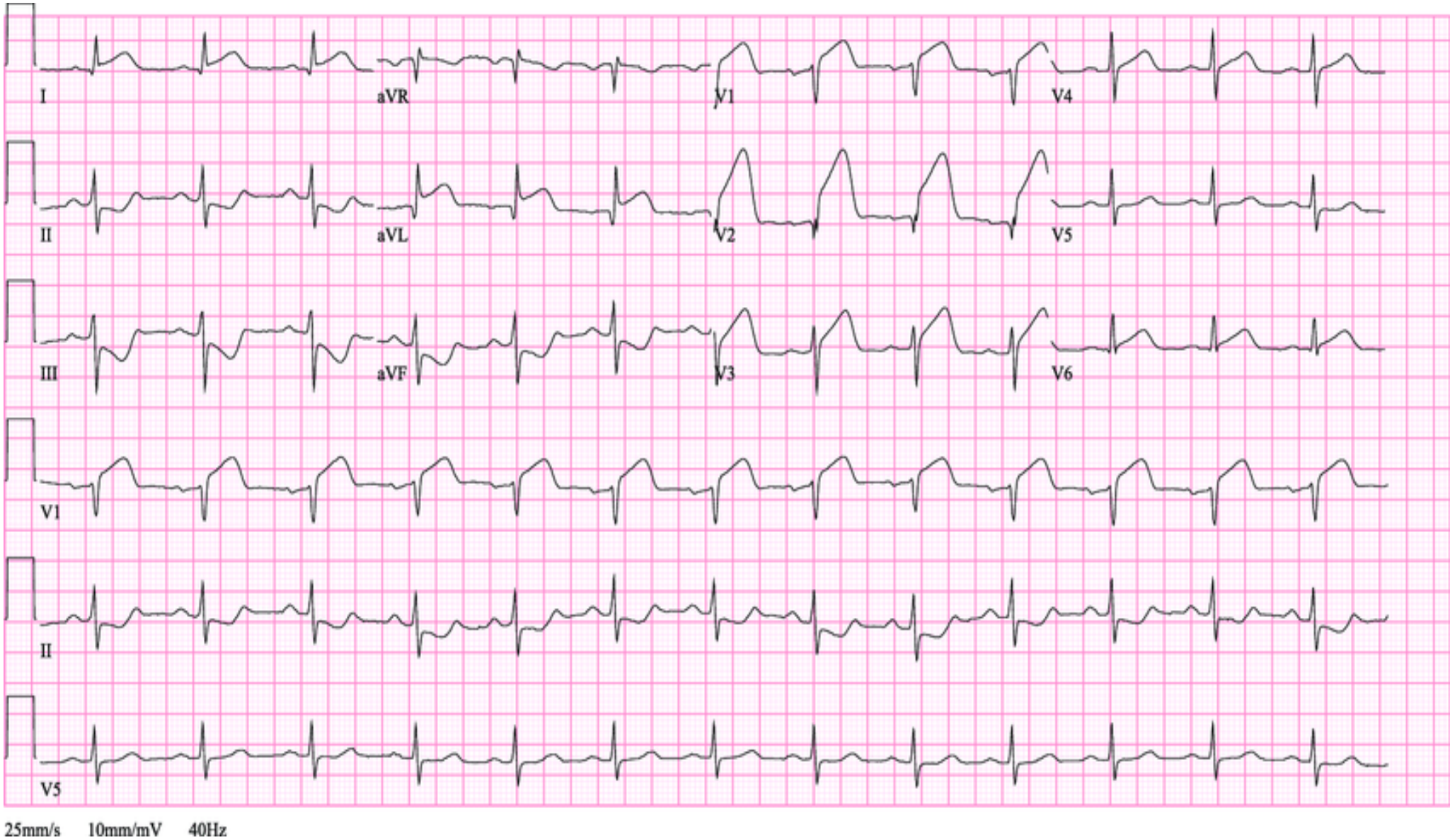
Low-grade fever

Complications

- Mitral regurgitation
- Pericarditis


Normal ECG

MI ECG



Heart failure

- **Heart failure** describes the clinical syndrome that develops when the heart cannot maintain adequate output, or can do so only at the expense of elevated ventricular filling pressure. In mild to moderate forms of heart failure, symptoms occur only when the metabolic demand increases during exercise or some other form of stress. In severe heart failure, symptoms may be present at rest. In clinical practice, heart failure may be diagnosed when a patient with significant heart disease develops the signs or symptoms of a low cardiac output, pulmonary congestion or systemic venous congestion at rest or on exercise. Three types of heart failure are recognized.

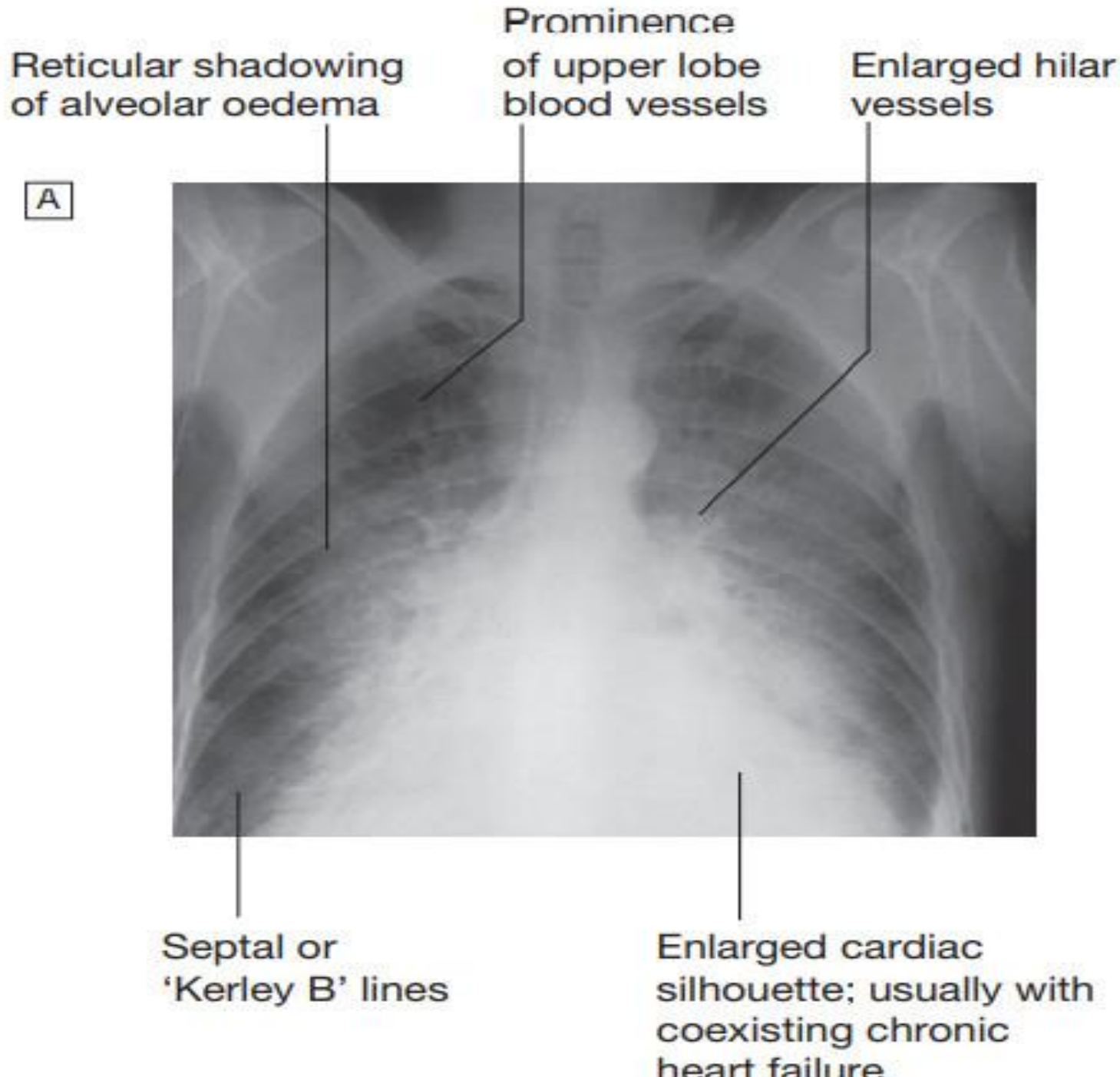
Left heart failure

- **Left heart failure** This is characterized by a reduction in left ventricular output and an increase in left atrial and pulmonary venous pressure. If left heart failure occurs suddenly – for example, as the result of an acute MI – the rapid increase in left atrial pressure causes pulmonary oedema. If the rise in atrial pressure is more gradual, as occurs with mitral stenosis, there is reflex pulmonary vasoconstriction, which protects the patient from pulmonary oedema. However, the resulting increase in pulmonary vascular resistance causes pulmonary hypertension, which in turn impairs right ventricular function.

- **Right heart failure** This is characterized by a reduction in right ventricular output and an increase in right atrial and systemic venous pressure. The most common causes are chronic lung disease, pulmonary embolism and pulmonary valvular stenosis. The term ‘cor pulmonale’ is used to describe right heart failure that is secondary to chronic lung disease.
- **Biventricular heart failure** In biventricular failure, both sides of the heart are affected. This may occur because the disease process, such as dilated cardiomyopathy or ischaemic heart disease, affects both ventricles or because disease of the left heart leads to chronic elevation of the left atrial pressure, pulmonary hypertension and right heart failure

16.12 Mechanisms of heart failure

Cause	Examples
Reduced ventricular contractility	Myocardial infarction (segmental dysfunction) Myocarditis/cardiomyopathy (global dysfunction)
Ventricular outflow obstruction (pressure overload)	Hypertension, aortic stenosis (left heart failure) Pulmonary hypertension, pulmonary valve stenosis (right heart failure)
Ventricular inflow obstruction	Mitral stenosis, tricuspid stenosis
Ventricular volume overload	Left ventricular volume overload (mitral or aortic regurgitation) Ventricular septal defect Right ventricular volume overload (atrial septal defect) Increased metabolic demand (high output)
Arrhythmia	Atrial fibrillation Tachycardia Complete heart block
Diastolic dysfunction	Constrictive pericarditis Restrictive cardiomyopathy


Reticular shadowing of alveolar oedema
Prominence of upper lobe blood vessels
Enlarged hilar vessels
A
Septal or 'Kerley B' lines
Enlarged cardiac silhouette; usually with coexisting chronic heart failure

16.15 Management of acute pulmonary oedema

Action	Effect
Sit the patient up	Reduces preload
Give high-flow oxygen	Corrects hypoxia
Ensure continuous positive airway pressure (CPAP) of 5–10 mmHg by tight-fitting mask	Reduces preload and pulmonary capillary hydraulic gradient
Administer nitrates:* IV glyceryl trinitrate (10–200 μg/min) Buccal glyceryl trinitrate 2–5 mg	Reduces preload and afterload
Administer a loop diuretic: Furosemide (50–100 mg IV)	Combats fluid overload

*The dose of nitrate should be titrated upwards every 10 mins until there is an improvement or systolic blood pressure is <110 mmHg.

THANK YOU•