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MED 405

CARDIOLOGY I

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Course title: Cardiology I

Course credit load: 2 Units

Series 1: Evaluation of the Cardiac Patient and Common Cardiovascular Symptoms

- **Part 1.1: Approach to the Cardiac Patient**
  - Sub Part 1.1.1: Overview of cardiac history taking
  - Sub Part 1.1.2: Cardiovascular risk factor assessment
  - Sub Part 1.1.3: Functional classification of cardiac symptoms
- **Part 1.2: Chest Pain**
  - Sub Part 1.2.1: Characteristics of cardiac chest pain
  - Sub Part 1.2.2: Differential diagnosis of chest pain
  - Sub Part 1.2.3: Clinical approach to the patient with chest pain
- **Part 1.3: Dyspnoea, Orthopnoea, and Paroxysmal Nocturnal Dyspnoea**
  - Sub Part 1.3.1: Dyspnoea of cardiac origin
  - Sub Part 1.3.2: Orthopnoea and its mechanism
  - Sub Part 1.3.3: Paroxysmal nocturnal dyspnoea
- **Part 1.4: Palpitations and Other Cardiovascular Symptoms**
  - Sub Part 1.4.1: Palpitations: definition, causes, and assessment
  - Sub Part 1.4.2: Syncope and presyncope

- Sub Part 1.4.3: Oedema and other peripheral symptoms

Introduction

Cardiovascular disease is the leading cause of death globally and a major and growing burden in Nigeria. The foundation of accurate cardiovascular diagnosis is a systematic, thorough clinical assessment: a carefully taken history identifies the nature and severity of symptoms, establishes risk factors, and guides the clinician's thinking before any investigation is ordered.

This Series introduces the structured approach to evaluating the cardiac patient, covering the systematic cardiovascular history, risk factor assessment, and functional classification of cardiac symptoms. It then examines the four cardinal cardiovascular symptoms in detail: chest pain, dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea, and palpitations, with additional coverage of syncope, presyncope, and peripheral oedema.

Mastery of symptom recognition and systematic history taking is the essential first step in cardiology, enabling the clinician to generate a focused differential diagnosis and select appropriate investigations without unnecessary testing.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 1.1** describe the structured approach to cardiovascular history taking and risk factor assessment (Linked to Part 1.1),
- **SLO 1.2** explain the NYHA functional classification of cardiac symptoms (Linked to Part 1.1),
- **SLO 1.3** describe the characteristics of cardiac chest pain, its differential diagnosis, and the clinical approach to the patient presenting with chest pain (Linked to Part 1.2),

- **SLO 1.4** describe the mechanisms and clinical features of dyspnoea, orthopnoea, and paroxysmal nocturnal dyspnoea of cardiac origin (Linked to Part 1.3), and
- **SLO 1.5** describe the causes and clinical assessment of palpitations, syncope, and peripheral oedema in the cardiac patient (Linked to Part 1.4).

1.1 Approach to the Cardiac Patient

1.1.1 Overview of cardiac history taking

The cardiovascular history follows the standard medical history structure but with specific emphasis on the cardinal cardiovascular symptoms: chest pain, dyspnoea (including orthopnoea and paroxysmal nocturnal dyspnoea), palpitations, syncope, and peripheral oedema. Each symptom is explored using the SOCRATES framework (Site, Onset, Character, Radiation, Associations, Timing, Exacerbating and relieving factors, Severity) to characterise it fully.

In addition to the presenting complaint, the cardiac history must document **past medical history** of cardiovascular disease (previous myocardial infarction, angina, heart failure, rheumatic fever, hypertension, stroke), **drug history** including all prescribed and over-the-counter medications with attention to cardiotoxic agents, and **family history** of premature cardiovascular disease, sudden cardiac death, or inherited conditions such as hypertrophic cardiomyopathy.

Fill in the blank: The cardiovascular history explores the cardinal symptoms (chest pain, dyspnoea, palpitations, syncope, oedema), past medical history of cardiac disease, drug history for cardiotoxic agents, and _____ history of premature cardiovascular disease.

1.1.2 Cardiovascular risk factor assessment

Cardiovascular risk factors are conditions and behaviours that increase the probability of developing cardiovascular disease. They are classified as **modifiable** (hypertension, dyslipidaemia, diabetes mellitus, smoking, obesity, physical inactivity, excessive alcohol) and

non-modifiable (age, male sex, family history of premature cardiovascular disease, and in women, early menopause).

In clinical practice, risk factor assessment is used to calculate a patient's **global cardiovascular risk** using tools such as the **Framingham Risk Score** or the WHO cardiovascular risk prediction charts adapted for Africa. High global risk (above 20 percent 10-year risk of a major cardiovascular event) justifies aggressive risk factor modification regardless of the presence of symptoms. In Nigeria, hypertension, diabetes, and obesity are the most prevalent modifiable cardiovascular risk factors.

Fill in the blank: Cardiovascular risk factors are modifiable (hypertension, dyslipidaemia, diabetes, smoking, obesity) or non-modifiable (age, sex, family history); global risk is estimated using tools such as the _____ Risk Score.

1.1.3 Functional classification of cardiac symptoms

The **New York Heart Association (NYHA) functional classification** is the most widely used system for grading the severity of cardiac symptoms in relation to functional capacity. It comprises four classes:

Class I: no limitation of ordinary physical activity; ordinary activity does not cause symptoms.

Class II: slight limitation; comfortable at rest but ordinary activity causes symptoms.

Class III: marked limitation; comfortable at rest but less than ordinary activity causes symptoms.

Class IV: symptoms at rest; any activity causes discomfort.

Fill in the blank: The NYHA functional classification grades cardiac symptoms from Class I (no limitation of ordinary activity) to Class IV (symptoms at _____) and is used to assess symptom severity and monitor treatment response.

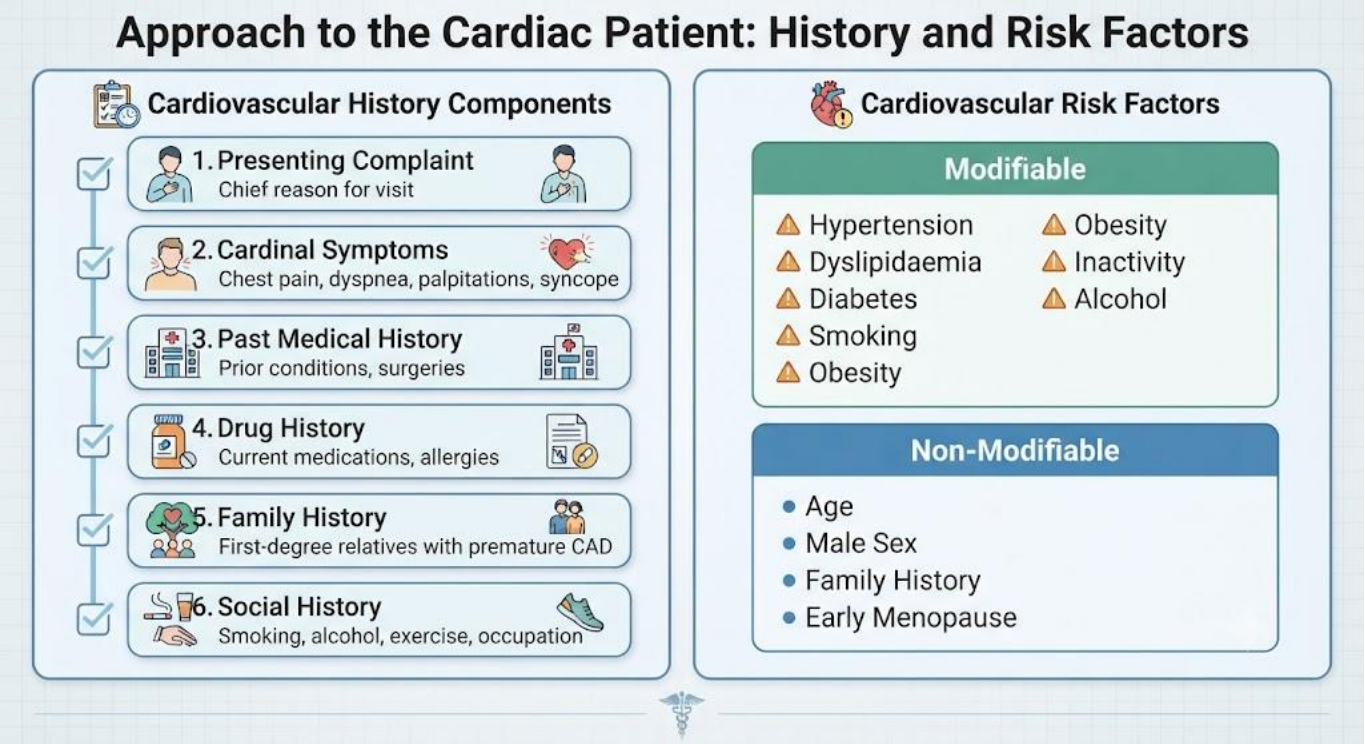

Figure 1.1: Components of the cardiovascular history and classification of cardiovascular risk factors

Pilot questions 1.1

1. Describe the components of a comprehensive cardiovascular history. (Linked to SLO 1.1)
2. Distinguish between modifiable and non-modifiable cardiovascular risk factors with examples of each. (Linked to SLO 1.1)
3. Explain what global cardiovascular risk means and how it is estimated. (Linked to SLO 1.1)
4. State and define the four NYHA functional classes. (Linked to SLO 1.2)
5. Explain how the NYHA classification is used in clinical practice. (Linked to SLO 1.2)

1.2 Chest Pain

1.2.1 Characteristics of cardiac chest pain

Cardiac chest pain most commonly arises from myocardial ischaemia and has characteristic features that distinguish it from non-cardiac causes. Classic ischaemic chest pain (angina pectoris) is typically described as a **tight, heavy, pressing, or crushing discomfort** (rather than a sharp or stabbing pain) in the central chest or retrosternal area, radiating to the **left arm, jaw, neck, or back**, precipitated by exertion or emotion, relieved by rest and glyceryl trinitrate within minutes, and lasting from 2 to 15 minutes.

Acute myocardial infarction (AMI) produces a similar but more severe and prolonged pain, typically lasting more than 20 minutes, not relieved by glyceryl trinitrate, and accompanied by diaphoresis, nausea, vomiting, and autonomic features. **Pericarditis** causes sharp, pleuritic chest pain (worse on inspiration and lying flat, relieved by sitting forward). **Aortic dissection** presents with severe, tearing pain radiating to the back, reaching maximum intensity at onset.

Fill in the blank: Classic ischaemic chest pain is central, tight or crushing, radiating to the left arm or jaw, precipitated by exertion, and relieved by rest or glyceryl trinitrate within minutes; pain lasting more than 20 minutes not relieved by GTN suggests acute _____ infarction.

1.2.2 Differential diagnosis of chest pain

The differential diagnosis of chest pain is broad and includes both cardiac and non-cardiac causes. Cardiac causes include **stable angina** (predictable exertional pain relieved by rest), **unstable angina and NSTEMI** (anginal pain at rest or with minimal exertion, representing acute coronary syndrome), **STEMI** (severe prolonged ischaemic pain with ST elevation), **pericarditis**, and **aortic dissection**.

Non-cardiac causes include **pulmonary embolism** (pleuritic pain with dyspnoea and haemoptysis), **pneumothorax** (sudden-onset pleuritic pain with dyspnoea), **pneumonia and pleuritis** (pleuritic pain with fever and cough), **oesophageal disease** (burning retrosternal pain related to food, relieved by antacids), **musculoskeletal pain** (localised, reproducible on palpation), and **anxiety and panic disorder** (often sharp, atypical, associated with autonomic features).

Fill in the blank: Cardiac causes of chest pain include stable angina, unstable angina and NSTEMI, STEMI, pericarditis, and aortic dissection; non-cardiac causes include pulmonary embolism, pneumothorax, _____ disease, and musculoskeletal pain.

1.2.3 Clinical approach to the patient with chest pain

The clinical approach to chest pain prioritises **identifying life-threatening causes** (AMI, aortic dissection, pulmonary embolism, tension pneumothorax) that require immediate management before full diagnostic evaluation. A rapid assessment includes the nature and duration of pain, associated symptoms (dyspnoea, diaphoresis, haemoptysis, syncope), vital signs, and an **ECG within 10 minutes** of presentation in any patient with possible acute coronary syndrome.

Initial investigations include **12-lead ECG** (ST elevation, depression, or T-wave changes), **cardiac troponin** (elevated in myocardial injury; serial measurement at 0 and 3 hours), **chest X-ray** (mediastinal widening in aortic dissection; pneumothorax; pulmonary oedema), and **oxygen saturation**. The history remains the most important component of the assessment; features that increase probability of a cardiac cause include typical pain character, age and sex, presence of multiple risk factors, and a prior history of coronary artery disease.

Fill in the blank: Chest pain assessment prioritises life-threatening causes; initial investigations include ECG within 10 minutes, serial cardiac _____, chest X-ray, and oxygen saturation; the history remains the most important assessment component.

Cardiac Causes	Non-Cardiac Causes
Stable Angina – **exertional pain, relieved by rest or nitroglycerin**	Pulmonary Embolism – sudden pleuritic pain, dyspnoea, risk factors for thrombosis
Unstable Angina/NSTEMI – **pain at rest, prolonged, ECG changes without ST elevation**	Pneumothorax – acute unilateral pleuritic pain, breathlessness, reduced breath sounds
STEMI – **severe crushing pain, radiates to arm/jaw, ST elevation on ECG**	Oesophageal Disease – burning retrosternal pain, related to meals or reflux
Pericarditis – **sharp pain, worse lying flat, relieved by sitting forward, pericardial rub**	Musculoskeletal – localised tenderness, reproducible with movement or palpation

Cardiac Causes	Non-Cardiac Causes
Aortic Dissection – tearing pain radiating to back, pulse differential, hypertension	Anxiety – atypical chest discomfort, associated with palpitations, hyperventilation

Table 1.1: Differential diagnosis of chest pain: cardiac and non-cardiac causes with distinguishing features

Pilot questions 1.2

1. Describe the characteristic features of ischaemic chest pain. (Linked to SLO 1.3)
2. Distinguish between the chest pain of stable angina and acute myocardial infarction. (Linked to SLO 1.3)
3. Describe the characteristic features of the chest pain of pericarditis and aortic dissection. (Linked to SLO 1.3)
4. List four life-threatening causes of chest pain and state the key distinguishing feature of each. (Linked to SLO 1.3)
5. Describe the initial clinical assessment and investigations for a patient presenting with acute chest pain. (Linked to SLO 1.3)

1.3 Dyspnoea, Orthopnoea, and Paroxysmal Nocturnal Dyspnoea

1.3.1 Dyspnoea of cardiac origin

Dyspnoea is the subjective sensation of difficult or uncomfortable breathing. Cardiac dyspnoea results from **pulmonary venous congestion**: elevated left ventricular filling pressures cause back pressure into the pulmonary veins and capillaries, increasing pulmonary capillary hydrostatic pressure, promoting fluid extravasation into the alveolar-interstitial space, reducing pulmonary compliance, and increasing the work of breathing.

Cardiac dyspnoea must be distinguished from **respiratory dyspnoea** (caused by obstructive or restrictive lung disease) and from **anaemic dyspnoea** (reduced oxygen-carrying capacity). Key distinguishing features include: cardiac dyspnoea is characteristically worsened by exertion and lying flat, associated with orthopnoea and PND, and relieved by sitting upright. It may be associated with other features of heart failure including ankle oedema, reduced exercise tolerance, and fatigue.

Fill in the blank: Cardiac dyspnoea results from pulmonary venous _____ caused by elevated left ventricular filling pressures; it is worsened by exertion and lying flat, and is associated with orthopnoea and paroxysmal nocturnal dyspnoea.

1.3.2 Orthopnoea and its mechanism

Orthopnoea is dyspnoea that occurs when lying flat and is relieved by sitting or standing upright. It is measured in terms of the number of pillows the patient needs to sleep comfortably. The mechanism involves **redistribution of blood** from the legs and dependent tissues to the pulmonary circulation when the patient adopts the supine position, increasing pulmonary venous pressure and worsening pulmonary congestion.

Orthopnoea is a hallmark feature of **left heart failure** (reduced left ventricular function with elevated filling pressures) and **mitral stenosis**. It may also occur in severe respiratory disease, bilateral diaphragmatic paralysis, and massive ascites. The clinician should ask specifically about the number of pillows used, whether the patient can lie flat, and whether they have ever woken breathless from sleep.

Fill in the blank: Orthopnoea is dyspnoea on lying flat, measured in pillows required for sleep; its mechanism is redistribution of blood to the pulmonary circulation in the supine position, worsening pulmonary congestion; it is a hallmark of left heart _____.

1.3.3 Paroxysmal nocturnal dyspnoea

Paroxysmal nocturnal dyspnoea (PND) is sudden-onset severe dyspnoea occurring at night, waking the patient from sleep, typically 1 to 3 hours after lying down. The patient typically

needs to sit up or stand, open a window, or move to a chair before symptoms improve. PND usually resolves over 15 to 30 minutes.

PND arises because fluid that has accumulated in the dependent tissues (ankles and legs) during the day is reabsorbed into the circulation when the patient lies down, increasing venous return and pulmonary venous pressure. Additionally, the normal reduction in **sympathetic tone** during sleep reduces the support previously given to cardiac output, unmasking heart failure. PND is a strong clinical indicator of **significant left heart failure** and should be actively sought in any patient with suspected cardiac disease.

Fill in the blank: PND is sudden nocturnal dyspnoea waking the patient 1 to 3 hours after lying down; it results from reabsorption of oedema fluid increasing venous return and from reduced _____ tone during sleep unmasking heart failure.

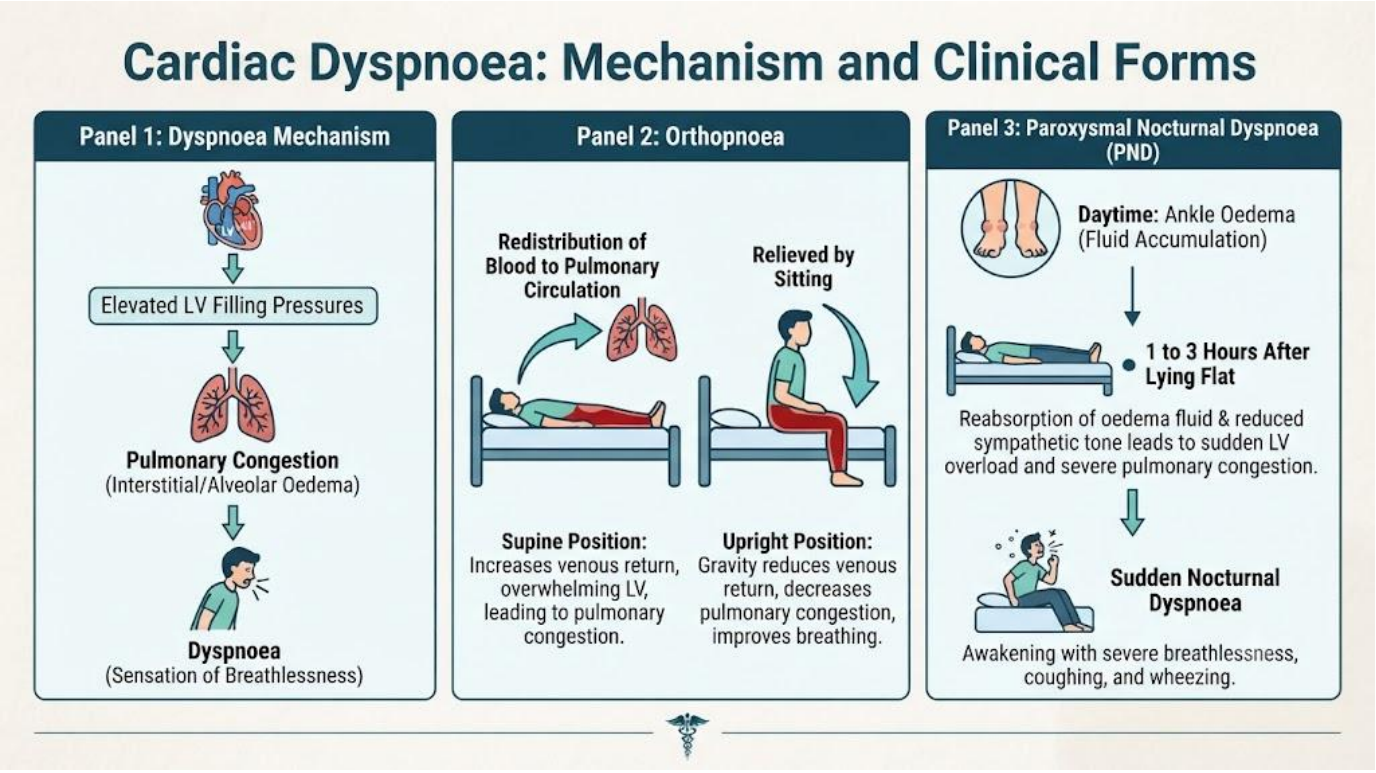

Figure 1.2: Mechanism of cardiac dyspnoea, orthopnoea, and paroxysmal nocturnal dyspnoea

Pilot questions 1.3

1. Explain the mechanism of dyspnoea in left heart failure. (Linked to SLO 1.4)
2. Describe three features that distinguish cardiac dyspnoea from respiratory dyspnoea. (Linked to SLO 1.4)
3. Define orthopnoea and explain its mechanism in left heart failure. (Linked to SLO 1.4)
4. Define paroxysmal nocturnal dyspnoea and explain why it occurs. (Linked to SLO 1.4)
5. Explain why PND typically occurs 1 to 3 hours after the patient lies down. (Linked to SLO 1.4)

1.4 Palpitations and Other Cardiovascular Symptoms

1.4.1 Palpitations: definition, causes, and assessment

Palpitations are an awareness of the heartbeat, perceived as rapid, irregular, forceful, or uncomfortable. The assessment of palpitations focuses on characterising the onset (sudden or gradual), pattern (regular or irregular), rate, associated symptoms (chest pain, dyspnoea, presyncope), and termination (spontaneous or by manoeuvres).

Common causes include **cardiac arrhythmias** (the most important to exclude): atrial fibrillation (irregular rapid heartbeat), supraventricular tachycardias (sudden-onset rapid regular palpitations, often with abrupt termination), ventricular ectopics (perception of a missed beat followed by a heavy beat), and ventricular tachycardia (rapid palpitations with haemodynamic compromise). Non-cardiac causes include **anxiety and panic disorder**, **anaemia**, **thyrotoxicosis**, **caffeine and stimulant use**, and **fever**.

Fill in the blank: Palpitations assessment covers onset, pattern, rate, associated symptoms, and termination; common causes include cardiac arrhythmias (atrial fibrillation, SVT, ventricular ectopics), anxiety, anaemia, _____, and stimulant use.

1.4.2 Syncope and presyncope

Syncope is a transient loss of consciousness due to global cerebral hypoperfusion, with spontaneous recovery. **Presyncope** is a prodrome of light-headedness, dizziness, visual disturbance, or impending loss of consciousness without complete loss of awareness. Both require evaluation to exclude a cardiac cause.

Cardiac causes of syncope (cardiac syncope) are particularly dangerous as they may predict sudden death. They include **arrhythmias** (severe bradycardia, complete heart block, ventricular tachycardia), **structural heart disease** (aortic stenosis, hypertrophic cardiomyopathy causing outflow obstruction), and **acute massive pulmonary embolism**. Non-cardiac causes include vasovagal syncope (most common, provoked by pain, prolonged standing, or emotion), orthostatic hypotension, and neurological causes. Sudden-onset syncope without prodrome, syncope during exertion, and syncope in the presence of known structural heart disease are red flags requiring urgent cardiac investigation.

Fill in the blank: Cardiac causes of syncope include arrhythmias, structural heart disease (aortic stenosis, hypertrophic cardiomyopathy), and pulmonary embolism; syncope during _____ or without prodrome are red flags requiring urgent investigation.

1.4.3 Oedema and other peripheral symptoms

Peripheral oedema in cardiac patients typically results from **right heart failure** causing elevated systemic venous pressure, sodium and water retention, and transudation of fluid into the interstitial space. Cardiac oedema is characteristically **bilateral and pitting**, worst in the ankles and legs (dependent oedema), and worsens during the day with improvement overnight after lying flat. In severe right heart failure, oedema may extend to the thighs, genitalia, and abdominal wall.

Other peripheral cardiovascular symptoms include **claudication** (calf pain on walking, relieved by rest, due to peripheral arterial disease), **cyanosis** (central or peripheral, indicating impaired oxygenation or reduced peripheral perfusion), and **fatigue** (a non-specific but often prominent

feature of heart failure resulting from reduced cardiac output and skeletal muscle hypoperfusion). Each of these symptoms may be a presenting feature of important cardiovascular disease.

Fill in the blank: Cardiac oedema results from right heart failure causing elevated venous pressure; it is bilateral, pitting, _____, worst in the ankles, and worsens during the day; claudication, cyanosis, and fatigue are additional peripheral cardiovascular symptoms.

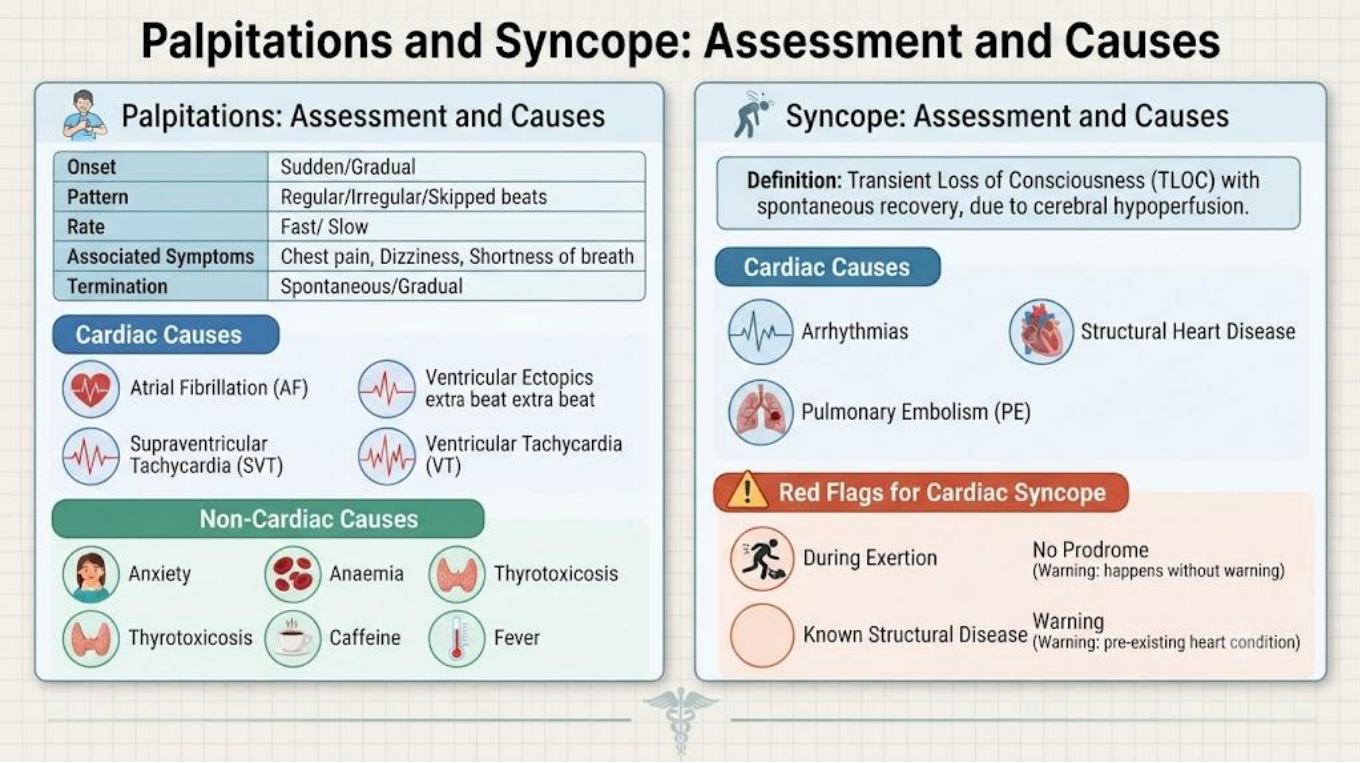

Figure 1.3: Palpitations and syncope: assessment framework, causes, and red flag features

Pilot questions 1.4

1. Define palpitations and describe the key features to establish in their assessment. (Linked to SLO 1.5)
2. Distinguish between cardiac and non-cardiac causes of palpitations. (Linked to SLO 1.5)
3. Define syncope and presyncope and list the cardiac causes of syncope. (Linked to SLO 1.5)
4. Describe the red flag features in a patient presenting with syncope. (Linked to SLO 1.5)

5. Describe the mechanism and clinical features of cardiac peripheral oedema. (Linked to SLO 1.5)

Series Summary

This Series introduced the systematic approach to evaluating the cardiac patient. The cardiovascular history covers the cardinal symptoms using SOCRATES, past cardiovascular history, drug history (including cardiotoxic agents), and family history of premature cardiovascular disease or sudden death. Cardiovascular risk factors are modifiable (hypertension, dyslipidaemia, diabetes, smoking, obesity, inactivity, alcohol) or non-modifiable (age, male sex, family history, early menopause); global risk estimation guides the intensity of risk factor management. The NYHA classification grades functional capacity from Class I (no limitation) to Class IV (symptoms at rest). Cardiac chest pain is central, heavy or pressing, radiating to the left arm or jaw, exertional, and relieved by GTN; AMI produces pain lasting more than 20 minutes not relieved by GTN; pericarditis causes sharp pleuritic pain relieved by sitting forward; aortic dissection causes tearing pain maximal at onset; and the initial approach to chest pain prioritises identifying life-threatening causes with ECG within 10 minutes and serial troponins.

Cardiac dyspnoea results from pulmonary venous congestion from elevated left ventricular filling pressures; orthopnoea is dyspnoea on lying flat due to redistribution of blood to the pulmonary circulation; and PND is sudden nocturnal dyspnoea waking the patient 1 to 3 hours after lying down due to reabsorption of dependent oedema fluid and reduced sympathetic tone during sleep. Palpitations may represent cardiac arrhythmias (atrial fibrillation, SVT, ventricular ectopics) or non-cardiac causes (anxiety, anaemia, thyrotoxicosis); assessment focuses on onset, pattern, rate, associations, and termination. Syncope is transient loss of consciousness from cerebral hypoperfusion; cardiac syncope from arrhythmias or structural disease is dangerous and syncope during exertion or without prodrome are red flags. Cardiac oedema from right heart failure is bilateral, pitting, and dependent; claudication, cyanosis, and fatigue are additional peripheral cardiovascular symptoms.

Glossary of Terms

- **Angina pectoris:** chest discomfort caused by myocardial ischaemia, typically precipitated by exertion and relieved by rest or glyceryl trinitrate.

- **Acute myocardial infarction (AMI):** myocardial necrosis due to prolonged ischaemia, presenting with chest pain lasting more than 20 minutes, elevated troponin, and ECG changes.
- **NYHA classification:** the New York Heart Association four-class grading of cardiac symptom severity in relation to physical activity.
- **Dyspnoea:** the subjective sensation of difficult or uncomfortable breathing; cardiac dyspnoea results from pulmonary venous congestion.
- **Orthopnoea:** dyspnoea that occurs on lying flat and is relieved by sitting upright; a hallmark of left heart failure.
- **Paroxysmal nocturnal dyspnoea (PND):** sudden severe dyspnoea waking the patient from sleep 1 to 3 hours after lying down; a strong indicator of significant left heart failure.
- **Palpitations:** an awareness of the heartbeat, perceived as rapid, irregular, or forceful.
- **Syncope:** transient loss of consciousness due to global cerebral hypoperfusion, with spontaneous recovery.
- **Presyncope:** a prodrome of light-headedness and near-loss of consciousness without complete loss of awareness.
- **Peripheral oedema:** fluid accumulation in the interstitial space; cardiac oedema is bilateral, pitting, and dependent, resulting from right heart failure.
- **Cardiac troponin:** a sensitive and specific biomarker of myocardial injury, elevated in acute myocardial infarction.
- **Global cardiovascular risk:** the estimated probability of a major cardiovascular event over 10 years, calculated using tools such as the Framingham Risk Score.

Concept Check Questions [Series 1]

Objective Questions

1. The NYHA functional classification grades cardiac symptoms from Class I (no limitation) to Class _____ (symptoms at rest). (Linked to SLO 1.2)
2. Classic ischaemic chest pain is central, tight or crushing, radiating to the left arm or jaw, and relieved by rest or _____ within minutes. (Linked to SLO 1.3)
3. Chest pain lasting more than 20 minutes not relieved by GTN, with diaphoresis and nausea, suggests acute myocardial _____. (Linked to SLO 1.3)
4. Cardiac dyspnoea results from pulmonary venous _____ caused by elevated left ventricular filling pressures. (Linked to SLO 1.4)
5. PND is sudden nocturnal dyspnoea waking the patient 1 to 3 hours after lying down; it results partly from reabsorption of _____ oedema fluid. (Linked to SLO 1.4)
6. Cardiac peripheral oedema from right heart failure is bilateral, pitting, and _____; it worsens during the day and improves overnight. (Linked to SLO 1.5)

Short-Answer Questions

7. State the four NYHA functional classes and define each. (Linked to SLO 1.2)
8. Distinguish between the chest pain of stable angina and acute myocardial infarction. (Linked to SLO 1.3)
9. Define orthopnoea and PND and explain the mechanism of each. (Linked to SLO 1.4)

Long-Answer Questions

10. Describe the approach to evaluating a cardiac patient, including the cardiovascular history, risk factor assessment, and NYHA classification. (Linked to SLOs 1.1 to 1.2)
11. Describe the characteristics, differential diagnosis, and clinical approach to the patient presenting with chest pain. (Linked to SLO 1.3)

Answers to Concept Check Questions [Series 1]

Objective Questions

1. IV.
2. Glyceryl trinitrate (GTN).
3. Infarction.
4. Congestion.
5. Dependent.
6. Dependent.

Short-Answer Questions

7. Class I: no limitation of ordinary activity. Class II: slight limitation; ordinary activity causes symptoms. Class III: marked limitation; less than ordinary activity causes symptoms. Class IV: symptoms at rest; any activity causes discomfort.
8. Stable angina: predictable pain precipitated by exertion or emotion, relieved by rest or GTN within 2 to 15 minutes, no troponin rise. AMI: severe prolonged pain lasting more than 20 minutes, not relieved by GTN, associated with diaphoresis and nausea, with troponin elevation.
9. Orthopnoea: dyspnoea on lying flat relieved by sitting upright; mechanism: redistribution of blood to the pulmonary circulation in the supine position increases pulmonary venous pressure. PND: sudden severe dyspnoea waking the patient 1 to 3 hours after lying down; mechanism: reabsorption of dependent oedema fluid increasing venous return, plus reduced sympathetic tone during sleep unmasking heart failure.

Long-Answer Questions

10. **Basic response:** Cardiovascular history: presenting complaint using SOCRATES for each cardinal symptom (chest pain, dyspnoea, orthopnoea, PND, palpitations, syncope, oedema); past cardiac history (MI, angina, heart failure, rheumatic fever, hypertension); drug history (cardiotoxic agents); family history (premature CVD, sudden death). Risk factors: modifiable (hypertension, dyslipidaemia, diabetes, smoking, obesity, inactivity,

alcohol); non-modifiable (age, male sex, family history, early menopause); global risk estimated by Framingham Risk Score or WHO charts; high risk above 20 percent 10-year justifies aggressive management. NYHA: Class I (no limitation), II (slight), III (marked), IV (symptoms at rest); used to assess severity and monitor treatment response.

Value-added response: The cardiovascular history is more than data collection: the skilled clinician uses it to build a probabilistic assessment of disease before any test is done; a 55-year-old hypertensive diabetic male smoker with typical exertional chest pain has a very high pre-test probability of coronary artery disease regardless of which test is subsequently ordered, and understanding this shapes the whole diagnostic and management approach.

11. **Basic response:** Characteristics: central, tight, pressing, or crushing; radiates to left arm, jaw, neck, or back; precipitated by exertion or emotion; relieved by rest or GTN within 2 to 15 minutes (stable angina); AMI: >20 minutes, not GTN-responsive, diaphoresis, nausea; pericarditis: sharp pleuritic, worse on inspiration, relieved sitting forward; aortic dissection: severe tearing, maximal at onset, radiates to back. Differential: cardiac (stable angina, unstable angina, NSTEMI, STEMI, pericarditis, aortic dissection); non-cardiac (pulmonary embolism, pneumothorax, oesophageal, musculoskeletal, anxiety). Clinical approach: identify life-threatening causes; ECG within 10 minutes; serial troponins at 0 and 3 hours; chest X-ray; oxygen saturation; history is most important component.

    Value-added response: The most dangerous error in chest pain assessment is failure to perform an ECG promptly: STEMI requires immediate reperfusion therapy and every minute of delay increases myocardial loss, making the 10-minute ECG target a patient safety imperative, not merely a guideline recommendation.

Answers to Pilot Questions [Series 1]

Part 1.1

1. Presenting complaint (cardinal symptoms using SOCRATES), past medical history of cardiovascular disease (MI, angina, heart failure, rheumatic fever, hypertension), drug history (all medications, cardiotoxic agents), family history (premature CVD, sudden cardiac death, inherited conditions), social history (smoking, alcohol, activity, occupation).
2. Modifiable: hypertension, dyslipidaemia, diabetes mellitus, smoking, obesity, physical inactivity, excessive alcohol. Non-modifiable: advancing age, male sex, family history of premature CVD, early menopause in women.
3. Global cardiovascular risk is the estimated probability of a major cardiovascular event (MI, stroke, cardiovascular death) over 10 years; estimated using tools such as the Framingham Risk Score or WHO cardiovascular risk prediction charts adapted for Africa; used to guide the intensity of risk factor management.
4. Class I: no limitation of ordinary physical activity; symptoms do not occur with ordinary activity. Class II: slight limitation; comfortable at rest but ordinary physical activity causes symptoms. Class III: marked limitation; comfortable at rest but less than ordinary activity causes symptoms. Class IV: symptoms present at rest; any physical activity causes further discomfort.
5. The NYHA classification is used clinically to document symptom severity at baseline, to monitor response to treatment (improvement in class indicates effective therapy), to guide decisions about intensity of treatment, and to stratify prognosis (Class IV carries a significantly worse prognosis than Class I or II).

Part 1.2

1. Central or retrosternal; tight, heavy, pressing, or crushing quality; radiates to left arm, jaw, neck, or back; precipitated by exertion or emotional stress; relieved by rest or glyceryl trinitrate within 2 to 15 minutes; duration typically 2 to 15 minutes.

2. Stable angina: predictable, exertional, relieved by rest or GTN within minutes, duration 2 to 15 minutes, no troponin elevation. AMI: severe, lasting more than 20 minutes, not relieved by GTN, associated with diaphoresis, nausea, vomiting, autonomic features, and elevated cardiac troponin.
3. Pericarditis: sharp, pleuritic (worse on inspiration and lying flat, relieved by sitting forward), may radiate to the left shoulder, often preceded by viral illness. Aortic dissection: sudden-onset severe tearing or ripping pain radiating to the back, maximal intensity at onset, associated with hypertension, asymmetric blood pressure, or pulse deficit.
4. AMI: prolonged ischaemic pain, ECG ST elevation, troponin rise. Aortic dissection: tearing pain to back, mediastinal widening on CXR. Pulmonary embolism: pleuritic pain, dyspnoea, haemoptysis, hypoxia. Tension pneumothorax: sudden pleuritic pain, absent breath sounds, tracheal deviation.
5. Identify life-threatening cause first; perform ECG within 10 minutes; rapid assessment of vital signs and pain characteristics; serial cardiac troponins at 0 and 3 hours; chest X-ray (mediastinal widening, pneumothorax, pulmonary oedema); oxygen saturation; history including risk factors, pain character, and prior cardiac history is the most important component.

Part 1.3

1. Elevated left ventricular filling pressures cause back pressure into the pulmonary veins and capillaries, raising pulmonary capillary hydrostatic pressure, promoting fluid extravasation into the interstitial and alveolar space, reducing pulmonary compliance, and increasing the work of breathing, producing dyspnoea.
2. Cardiac dyspnoea: worse on exertion and on lying flat, associated with orthopnoea and PND, relieved by sitting upright, associated with ankle oedema and other heart failure features. Respiratory dyspnoea: often associated with wheeze, productive cough, smoking history; not typically positional in the same way; not relieved by sitting forward (exception: COPD patients may use pursed-lip breathing).

3. Orthopnoea is dyspnoea that occurs when lying flat and is relieved by sitting or standing upright; it is measured by the number of pillows required for comfortable sleep. Mechanism: adopting the supine position redistributes blood from the legs and dependent tissues into the central and pulmonary circulation, increasing pulmonary venous pressure and worsening congestion.
4. PND is sudden severe dyspnoea waking the patient from sleep, typically 1 to 3 hours after lying down; it resolves over 15 to 30 minutes after sitting up. It results from reabsorption of fluid from dependent oedema into the circulation during recumbency increasing venous return and pulmonary venous pressure, and from reduced sympathetic support to cardiac output during sleep that unmasks decompensated heart failure.
5. During the day in the upright position, fluid accumulates in the ankles and dependent tissues due to gravity. When the patient lies flat at night, this fluid is gradually reabsorbed into the circulation over 1 to 3 hours, increasing venous return and pulmonary venous pressure to the point of triggering acute pulmonary congestion and waking the patient with severe dyspnoea.

Part 1.4

1. Palpitations are an awareness of the heartbeat, perceived as rapid, irregular, forceful, or uncomfortable. Key features: onset (sudden or gradual), pattern (regular or irregular), rate estimate, associated symptoms (chest pain, dyspnoea, presyncope, syncope), duration, and termination (spontaneous, by Valsalva, or requiring intervention).
2. Cardiac: atrial fibrillation (irregular, variable rate), supraventricular tachycardia (sudden-onset rapid regular, abrupt termination), ventricular ectopics (missed beat then heavy beat), ventricular tachycardia (rapid with haemodynamic compromise). Non-cardiac: anxiety and panic disorder, anaemia (high-output state), thyrotoxicosis, caffeine and stimulants, fever.
3. Syncope is transient loss of consciousness due to global cerebral hypoperfusion with spontaneous recovery. Presyncope is a prodrome of light-headedness, visual

disturbance, or impending loss of consciousness without complete loss of awareness. Cardiac causes: arrhythmias (severe bradycardia, complete heart block, VT), structural disease (aortic stenosis, hypertrophic cardiomyopathy), acute pulmonary embolism.

4. Syncope during exertion (suggests structural heart disease or arrhythmia triggered by exercise); syncope without prodrome (suggests arrhythmia); syncope in the presence of known structural heart disease; syncope associated with chest pain or palpitations; multiple recurrent syncopal episodes; family history of sudden cardiac death.
5. Mechanism: right heart failure causes elevated systemic venous pressure, activating the renin-angiotensin-aldosterone system and causing sodium and water retention; raised capillary hydrostatic pressure promotes transudation of fluid into the interstitial space. Features: bilateral, pitting, dependent (ankles and lower legs), worst at end of day, improves overnight when the patient lies flat, may extend to thighs and genitalia in severe cases.

References and Attributions [Series 1]

Textual References

- Kumar, P. and Clark, M. (2017). Kumar and Clark's Clinical Medicine (9th ed.). Edinburgh: Elsevier.
- Lip, G. Y. H. and Hall, J. E. (2019). Comprehensive Cardiovascular Medicine in the Community. Oxford: Wiley-Blackwell.
- Thygesen, K. et al. (2018). Fourth Universal Definition of Myocardial Infarction. European Heart Journal, 40(3), 237 to 269.
- Yancy, C. W. et al. (2017). 2017 ACC/AHA Heart Failure Guidelines. Journal of the American College of Cardiology, 70(6), e137 to e210.

Figures, Plates, Tables, and Boxes

- Figure 1.1: Cardiovascular history components and risk factor classification. AI generation prompt: "Two-panel diagram: left panel checklist Cardiovascular History Components (Presenting Complaint, Cardinal Symptoms, Past Medical History, Drug History, Family History, Social History); right panel two boxes Modifiable Risk Factors (hypertension, dyslipidaemia, diabetes, smoking, obesity, inactivity, alcohol) and Non-Modifiable Risk Factors (age, male sex, family history, early menopause); clean professional infographic style." OER search: "cardiovascular history risk factors assessment diagram modifiable non-modifiable".
- Table 1.1: Differential diagnosis of chest pain. AI prompt: "Two-column table: Cardiac Causes (Stable Angina, Unstable Angina/NSTEMI, STEMI, Pericarditis, Aortic Dissection) and Non-Cardiac Causes (Pulmonary Embolism, Pneumothorax, Oesophageal, Musculoskeletal, Anxiety); distinguishing features; alternating shading." OER search: "chest pain differential diagnosis cardiac non-cardiac table distinguishing features".
- Figure 1.2: Mechanism of cardiac dyspnoea, orthopnoea, and PND. AI prompt: "Three-panel diagram: Dyspnoea Mechanism (LV filling pressure to pulmonary congestion to dyspnoea), Orthopnoea (supine blood redistribution), PND (day oedema to overnight

reabsorption to acute congestion); clean infographic." OER search: "cardiac dyspnoea orthopnoea PND mechanism diagram pulmonary congestion".

- Figure 1.3: Palpitations and syncope assessment, causes, and red flags. AI prompt: "Two-panel diagram: Palpitations (assessment table plus cardiac and non-cardiac causes); Syncope (definition, cardiac causes, red flags); clean professional infographic." OER search: "palpitations syncope assessment causes red flags cardiac arrhythmia diagram".

Series 2: Cardiovascular System Examination and Cardiac Auscultation

- **Part 2.1: General and Peripheral Cardiovascular Examination**
  - Sub Part 2.1.1: General inspection and peripheral signs
  - Sub Part 2.1.2: The arterial pulse
  - Sub Part 2.1.3: Jugular venous pressure
- **Part 2.2: Examination of the Precordium**
  - Sub Part 2.2.1: Inspection and palpation of the precordium
  - Sub Part 2.2.2: The cardiac impulse and thrills
  - Sub Part 2.2.3: Percussion of the heart
- **Part 2.3: Cardiac Auscultation: Heart Sounds**
  - Sub Part 2.3.1: Normal heart sounds
  - Sub Part 2.3.2: Added heart sounds
  - Sub Part 2.3.3: Auscultatory areas and technique
- **Part 2.4: Cardiac Murmurs**
  - Sub Part 2.4.1: Classification and grading of murmurs
  - Sub Part 2.4.2: Systolic murmurs
  - Sub Part 2.4.3: Diastolic murmurs and continuous murmurs

Introduction

The cardiovascular examination is a structured, systematic assessment that moves logically from general inspection through peripheral signs to the precordium and cardiac auscultation. Each component provides information that either confirms or refutes the diagnostic hypotheses generated from the history, and the findings from each part of the examination build cumulatively towards a clinical diagnosis.

This Series covers the complete cardiovascular examination in sequence: general and peripheral signs including the arterial pulse and jugular venous pressure; precordial inspection, palpation, and percussion; cardiac auscultation including normal and added heart sounds; and the characterisation and interpretation of cardiac murmurs. The valvular areas, the techniques of auscultation, and the systematic approach to describing and grading murmurs are covered in clinical detail.

Competence in cardiovascular examination is acquired through repeated supervised practice at the bedside. The knowledge framework provided in this Series equips the student to make sense of what they hear and feel, converting sensory experience into clinical information.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 2.1** describe the general and peripheral signs of cardiovascular disease detectable on inspection and palpation (Linked to Part 2.1),
- **SLO 2.2** describe the characteristics of the normal arterial pulse and the jugular venous pressure, and identify abnormal variants (Linked to Part 2.1),
- **SLO 2.3** describe the findings of precordial inspection, palpation, and percussion and their clinical significance (Linked to Part 2.2),

- **SLO 2.4** describe normal and added heart sounds and explain their physiological basis (Linked to Part 2.3), and
- **SLO 2.5** classify, grade, and describe the major cardiac murmurs and state the conditions with which each is associated (Linked to Part 2.4).

2.1 General and Peripheral Cardiovascular Examination

2.1.1 General inspection and peripheral signs

General inspection of the cardiac patient begins before any hands-on assessment. The clinician notes whether the patient appears well or unwell, comfortable or distressed, and whether there are obvious abnormalities. Key peripheral signs of cardiovascular disease detectable on inspection include **central cyanosis** (blue discolouration of the lips and tongue indicating systemic hypoxaemia), **peripheral cyanosis** (blue discolouration of the fingers and toes from reduced peripheral perfusion), and **clubbing** (loss of the nail-bed angle and increase in nail curvature, associated with cyanotic congenital heart disease, infective endocarditis, and other conditions).

Examination of the hands reveals additional signs: **splinter haemorrhages** (dark longitudinal lines under the nails, a sign of infective endocarditis), **Osler's nodes** (tender nodules on the finger pads, infective endocarditis), **Janeway lesions** (non-tender erythematous macules on the palms, infective endocarditis), and **xanthomata** (cholesterol deposits in tendons or over joints, indicating familial hypercholesterolaemia). The face is examined for **malar flush** (mitral stenosis), **xanthelasma** (cholesterol deposits around the eyes), and anaemia of the conjunctivae.

Fill in the blank: Peripheral signs of cardiovascular disease include central and peripheral cyanosis, clubbing, splinter haemorrhages, Osler's nodes and Janeway lesions (infective endocarditis), xanthomata (hypercholesterolaemia), and malar _____ (mitral stenosis).

2.1.2 The arterial pulse

The radial pulse is assessed for **rate** (normal 60 to 100 beats per minute), **rhythm** (regular or irregular), **volume** (normal, reduced, or large volume), and **character** (the quality of the upstroke and waveform). The **carotid pulse** provides the most accurate assessment of pulse character due to its proximity to the heart.

Important abnormal pulse characters include **pulsus paradoxus** (an exaggerated fall in systolic BP of more than 10 mmHg during inspiration, seen in cardiac tamponade and severe asthma), **pulsus alternans** (alternating strong and weak beats of regular rhythm, indicating severe left ventricular dysfunction), **collapsing (water-hammer) pulse** (high volume with rapid rise and fall, characteristic of aortic regurgitation), and **plateau pulse** (slow rising, low volume, characteristic of aortic stenosis).

Fill in the blank: Abnormal pulse characters include pulsus paradoxus (cardiac tamponade), pulsus alternans (severe LV dysfunction), collapsing pulse (aortic _____), and plateau pulse (aortic stenosis).

2.1.3 Jugular venous pressure

The jugular venous pressure (JVP) reflects right atrial pressure and is assessed by examining the internal jugular vein with the patient positioned at 45 degrees. The JVP is measured as the vertical height of the venous pulsation above the sternal angle; the normal JVP is less than 3 to 4 cm above the sternal angle.

An **elevated JVP** indicates raised right atrial pressure, seen in right heart failure, cardiac tamponade, constrictive pericarditis, and superior vena cava obstruction. The **JVP waveform** has two main components: the **a wave** (atrial contraction, absent in atrial fibrillation, large in tricuspid stenosis and pulmonary hypertension) and the **v wave** (venous filling; large in tricuspid regurgitation). The JVP falls on inspiration (Kussmaul's sign: paradoxical rise on inspiration is seen in cardiac tamponade and constrictive pericarditis).

Fill in the blank: The JVP is measured above the sternal angle with the patient at 45 degrees; elevated JVP indicates raised right atrial pressure; the a wave reflects atrial contraction and the v wave reflects venous _____; Kussmaul's sign is a paradoxical rise on inspiration.

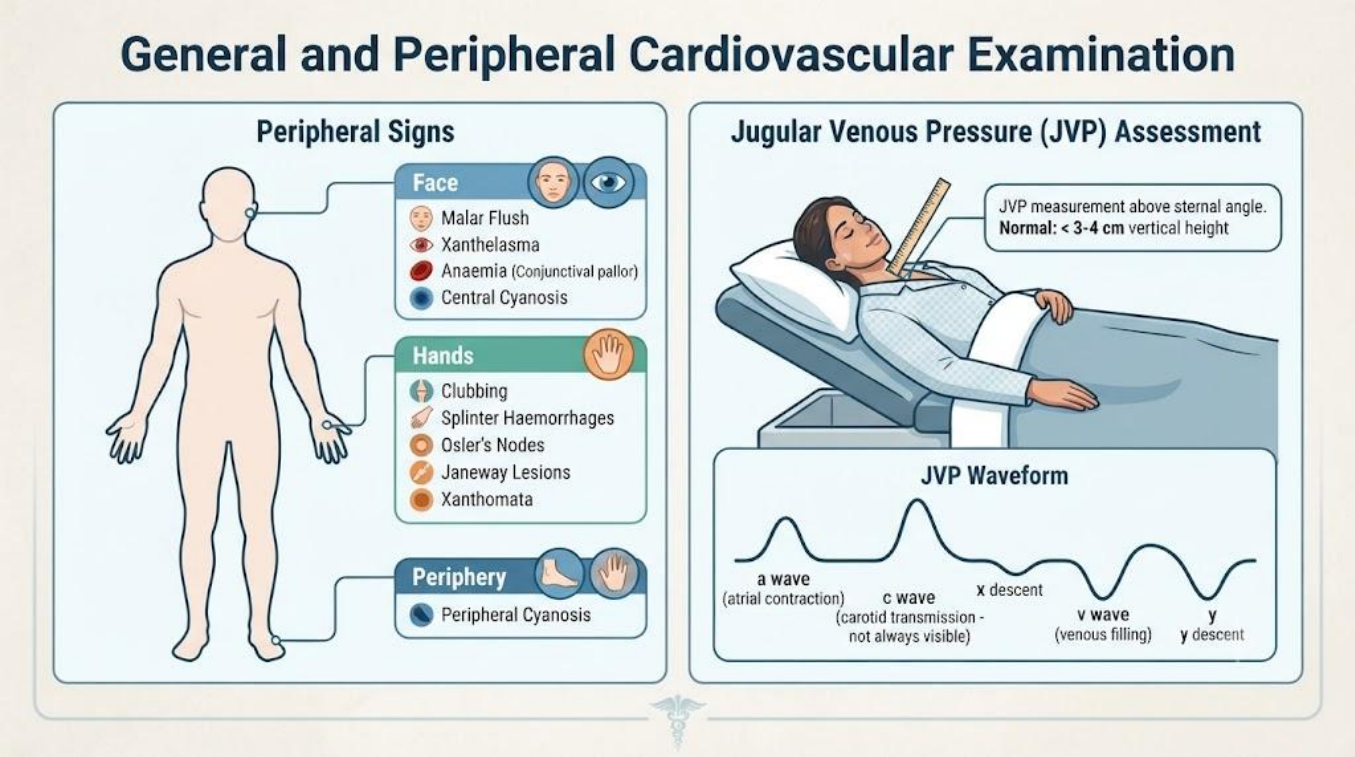

Figure 2.1: Peripheral cardiovascular signs and the assessment of jugular venous pressure

Pilot questions 2.1

1. Describe the peripheral signs of infective endocarditis detectable on inspection of the hands and face. (Linked to SLO 2.1)
2. Describe the four characteristics of the arterial pulse that should be assessed at the radial artery. (Linked to SLO 2.2)
3. Describe the collapsing pulse and plateau pulse and state the conditions with which each is associated. (Linked to SLO 2.2)
4. Describe how the JVP is assessed and state the normal value. (Linked to SLO 2.2)

5. Explain the significance of an elevated JVP and describe Kussmaul's sign. (Linked to SLO 2.2)

2.2 Examination of the Precordium

2.2.1 Inspection and palpation of the precordium

Precordial inspection involves examining the anterior chest wall for visible pulsations, deformities, and scars. A visible **apex beat** may be seen in thin patients and in conditions with increased cardiac activity. **Chest wall deformities** such as pectus excavatum (funnel chest) or pectus carinatum (pigeon chest) may compress or displace the heart. Surgical scars (midline sternotomy from previous cardiac surgery, left lateral thoracotomy) provide important past history.

Palpation of the precordium identifies the apex beat, assesses its character, and detects thrills and heaves. The **apex beat** is the lowest and most lateral point at which the cardiac impulse is felt; normally located in the fifth intercostal space in the midclavicular line. A **displaced apex beat** (lateral or inferior to its normal position) indicates cardiac enlargement. The **character of the apex beat** is assessed: a **hyperdynamic apex** indicates volume overload (aortic or mitral regurgitation); a **heaving (pressure-loaded) apex** indicates pressure overload (aortic stenosis, hypertension).

Fill in the blank: The apex beat is normally in the fifth intercostal space in the _____ line; a displaced apex indicates cardiac enlargement; a hyperdynamic apex suggests volume overload and a heaving apex suggests pressure overload.

2.2.2 The cardiac impulse and thrills

A **right ventricular heave** (parasternal heave) is felt by placing the palm along the left sternal border; a sustained lift indicates right ventricular hypertrophy or enlargement, seen in pulmonary hypertension, pulmonary stenosis, and large atrial septal defects. A **palpable pulmonary**

component of the second heart sound (P2) in the pulmonary area indicates pulmonary hypertension.

Thrills are palpable vibrations produced by turbulent blood flow through a cardiac valve or septal defect; they correspond to a **grade 4 or higher murmur** on auscultation. A systolic thrill at the base of the heart suggests aortic stenosis or pulmonary stenosis; a systolic thrill at the lower left sternal border suggests a ventricular septal defect; a diastolic thrill at the apex suggests mitral stenosis. The presence of a thrill indicates haemodynamically significant valvular or structural disease.

Fill in the blank: A parasternal heave indicates right ventricular hypertrophy; thrills are palpable vibrations corresponding to grade 4 or higher murmurs; a systolic thrill at the base suggests aortic or pulmonary stenosis and a diastolic thrill at the _____ suggests mitral stenosis.

2.2.3 Percussion of the heart

Percussion of the cardiac borders has largely been superseded by chest X-ray and echocardiography in modern practice but remains a clinical skill. The **right cardiac border** is percussed from the right side of the sternum; the **left cardiac border** follows the apex beat position. Dullness extending beyond the normal boundaries in either direction indicates cardiac enlargement or pericardial effusion.

Dullness to percussion in the cardiac area that extends laterally beyond the apex beat position, or dullness extending to the left of the normal cardiac border, may suggest a **pericardial effusion** particularly if accompanied by muffled heart sounds and elevated JVP (Beck's triad: muffled heart sounds, elevated JVP, and hypotension indicate cardiac tamponade). Percussion of the lung bases simultaneously assesses for pleural effusions, which may accompany left heart failure.

Fill in the blank: Percussion identifies cardiac borders; dullness beyond the apex beat suggests cardiac enlargement or pericardial _____; Beck's triad (muffled sounds, elevated JVP, hypotension) indicates cardiac tamponade.

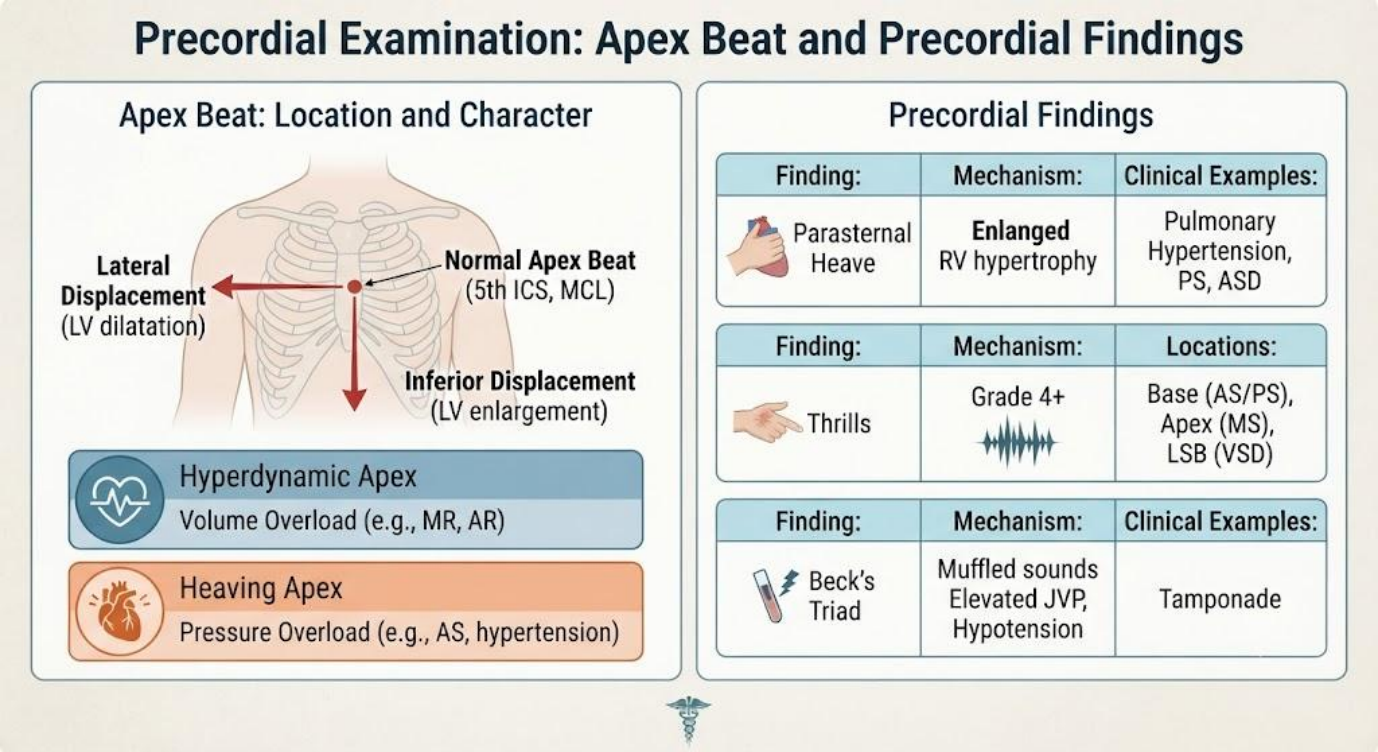

Figure 2.2: Precordial examination: apex beat assessment and additional precordial findings

Pilot questions 2.2

1. Describe the normal position of the apex beat and explain the clinical significance of apex beat displacement. (Linked to SLO 2.3)
2. Distinguish between a hyperdynamic apex and a heaving apex and state the conditions associated with each. (Linked to SLO 2.3)
3. Define a parasternal heave and state the conditions in which it is found. (Linked to SLO 2.3)
4. Define a thrill and explain its clinical significance. (Linked to SLO 2.3)
5. Describe Beck's triad and explain its clinical significance. (Linked to SLO 2.3)

2.3 Cardiac Auscultation: Heart Sounds

2.3.1 Normal heart sounds

The first heart sound (S1) is produced by closure of the **mitral and tricuspid valves** at the onset of systole. It is best heard at the apex and lower left sternal border. S1 is loud in mitral stenosis (the valve leaflets are stiff and slam shut) and soft when the mitral valve closes before significant pressure development (short PR interval, severe MR).

The second heart sound (S2) is produced by closure of the **aortic and pulmonary valves** at the end of systole. It is best heard at the base (aortic and pulmonary areas). S2 normally splits on inspiration (physiological splitting) as increased venous return delays pulmonary valve closure. **Fixed splitting of S2** (splitting that does not vary with respiration) is the hallmark of atrial septal defect. **Paradoxical splitting** (S2 splits on expiration) occurs in left bundle branch block and aortic stenosis.

Fill in the blank: S1 results from mitral and tricuspid valve closure; S2 from aortic and pulmonary valve closure; fixed splitting of S2 is the hallmark of _____ septal defect; paradoxical splitting occurs in left bundle branch block.

2.3.2 Added heart sounds

The third heart sound (S3) is a low-pitched sound heard in early diastole, shortly after S2, during rapid ventricular filling. It is normal in children and young adults but in patients over 40 indicates **left ventricular failure or volume overload** (mitral regurgitation, ventricular septal defect). The combination of S1, S2, and S3 produces a characteristic **triple rhythm or gallop rhythm** resembling the sound of a galloping horse.

The fourth heart sound (S4) is a low-pitched sound heard in late diastole, just before S1, produced by atrial contraction against a stiff (non-compliant) ventricle. It indicates **diastolic dysfunction** and is associated with hypertension, hypertrophic cardiomyopathy, and acute myocardial infarction. S4 is never normal. An **opening snap** is a high-pitched early diastolic sound that follows S2, produced by the sudden opening of a stenotic mitral valve; a short S2-to-

opening-snap interval indicates severe mitral stenosis. A **pericardial friction rub** is a scratching, to-and-fro sound heard in pericarditis.

Fill in the blank: S3 in adults indicates LV failure or volume overload; S4 indicates diastolic dysfunction and is never normal; an opening snap following S2 indicates mitral _____ and a short S2-to-snap interval indicates severe disease.

2.3.3 Auscultatory areas and technique

The four main auscultatory areas correspond to the surface projections of the cardiac valves: the **mitral area** (cardiac apex, fifth intercostal space, midclavicular line), the **tricuspid area** (lower left sternal border, fourth intercostal space), the **pulmonary area** (left second intercostal space, upper left sternal border), and the **aortic area** (right second intercostal space, upper right sternal border).

Auscultation technique requires a quiet environment and a systematic approach. The **diaphragm of the stethoscope** is used for high-pitched sounds (normal heart sounds, aortic and mitral regurgitation murmurs, pericardial rub) while the **bell** is used for low-pitched sounds (S3, S4, mitral stenosis murmur). Auscultation should be performed in all four areas and with special manoeuvres: the **left lateral decubitus position** accentuates mitral valve sounds at the apex; sitting forward and breath-holding in expiration accentuates **aortic regurgitation** at the left sternal border.

Fill in the blank: The four auscultatory areas are mitral (apex), tricuspid (lower left sternal border), pulmonary (left second ICS), and aortic (right second ICS); the diaphragm detects high-pitched sounds and the _____ detects low-pitched sounds including S3, S4, and mitral stenosis.

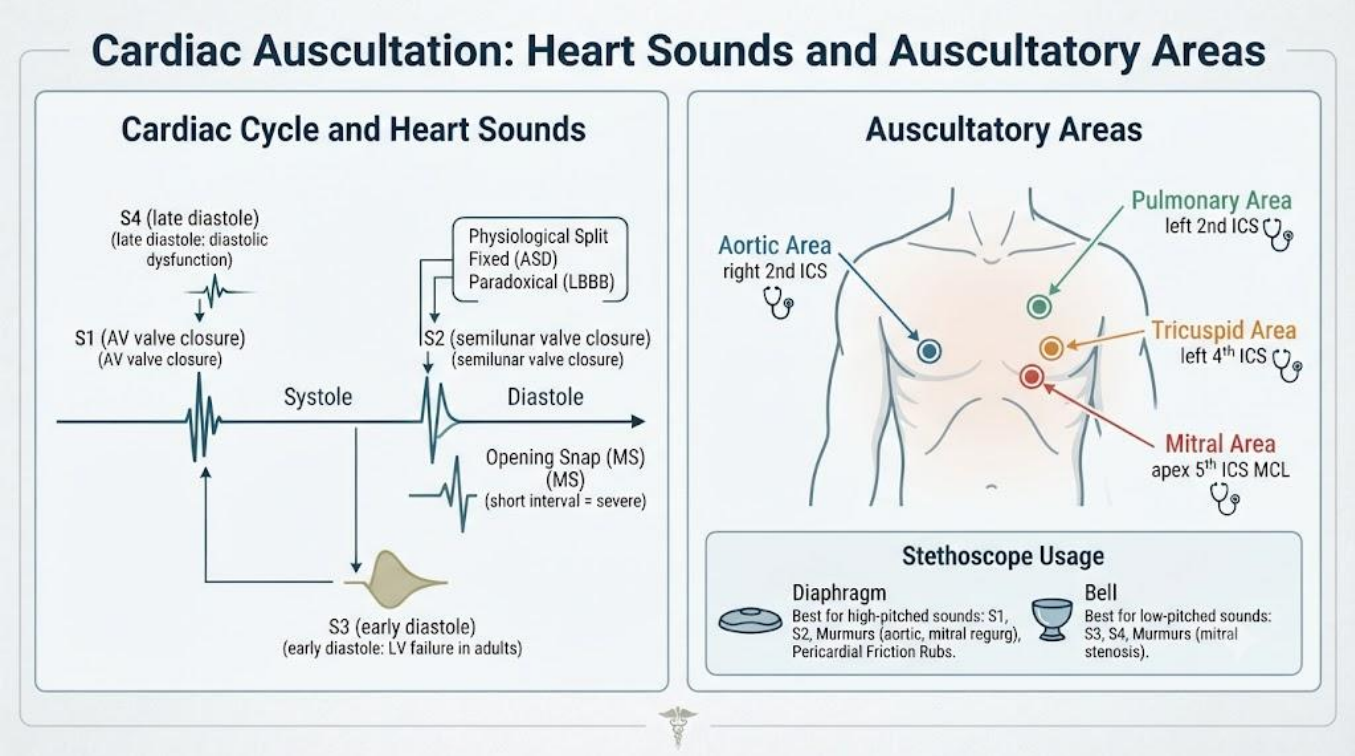

Figure 2.3: Cardiac auscultation: the sequence of heart sounds and the four auscultatory areas

Pilot questions 2.3

1. Explain the physiological basis and auscultatory characteristics of S1 and S2. (Linked to SLO 2.4)
2. Explain fixed splitting and paradoxical splitting of S2 and state the conditions associated with each. (Linked to SLO 2.4)
3. Describe S3 and S4 heart sounds, their physiological basis, and their clinical significance. (Linked to SLO 2.4)
4. Describe the opening snap and explain how it indicates the severity of mitral stenosis. (Linked to SLO 2.4)
5. Describe the four auscultatory areas and the technique for cardiac auscultation. (Linked to SLO 2.4)

2.4 Cardiac Murmurs

2.4.1 Classification and grading of murmurs

Cardiac murmurs are sounds produced by turbulent blood flow within the heart or great vessels. They are classified by their timing in the cardiac cycle: **systolic murmurs** (during systole, between S1 and S2), **diastolic murmurs** (during diastole, between S2 and the next S1), and **continuous murmurs** (heard throughout both systole and diastole).

The Levine grading system grades murmur intensity from 1 to 6: Grade 1 (very faint, heard only in ideal conditions), Grade 2 (faint but immediately audible), Grade 3 (moderately loud, no thrill), **Grade 4** (loud with a thrill), Grade 5 (very loud, heard with stethoscope partly off chest, thrill present), Grade 6 (audible without a stethoscope). Murmurs are also described by their **location**, **radiation**, **quality** (harsh, blowing, rumbling, musical), and **effect of manoeuvres** (inspiration, expiration, Valsalva, posture) on their intensity.

Fill in the blank: Murmurs are graded 1 to 6 by the Levine system; grades 4 and above are associated with a _____; murmurs are described by timing, location, radiation, quality, and effect of manoeuvres.

2.4.2 Systolic murmurs

Ejection systolic murmurs (ESM) begin after S1, peak in midsystole, and end before S2; they are produced by turbulent ejection through stenosed or high-flow outflow valves. The classic ESM is that of **aortic stenosis**: a harsh, crescendo-decrescendo murmur heard loudest in the aortic area, radiating to the carotid arteries, accompanied by a slow-rising plateau pulse and a soft or absent A2. **Pulmonary stenosis** produces a similar murmur in the pulmonary area.

Pansystolic (holosystolic) murmurs begin with S1, are uniform throughout systole, and extend to S2; they are produced by regurgitation through an incompetent valve or a ventricular septal defect throughout systole. **Mitral regurgitation** produces a blowing pansystolic murmur at the apex radiating to the axilla. **Tricuspid regurgitation** produces a pansystolic murmur at the lower

left sternal border that increases with inspiration (Carvallo's sign). **Ventricular septal defect** produces a loud harsh pansystolic murmur at the lower left sternal border.

Fill in the blank: Ejection systolic murmurs (aortic and pulmonary stenosis) are crescendo-decrescendo; pansystolic murmurs (mitral regurgitation, tricuspid regurgitation, VSD) begin with S1 and extend to S2; mitral regurgitation murmur radiates to the _____.

2.4.3 Diastolic murmurs and continuous murmurs

Diastolic murmurs are always pathological. Early diastolic murmurs begin immediately after S2 and are produced by regurgitation through incompetent semilunar valves. **Aortic regurgitation (AR)** produces a soft, high-pitched, blowing early diastolic murmur heard best at the left sternal border with the patient sitting forward and breath-holding in expiration.

Mid-diastolic murmurs occur in mid-diastole during ventricular filling. **Mitral stenosis** produces a low-pitched, rumbling mid-diastolic murmur heard best at the cardiac apex with the patient in the left lateral decubitus position, using the bell of the stethoscope, preceded by an opening snap. The murmur may be accompanied by **presystolic accentuation** (due to atrial contraction) in patients in sinus rhythm. A **continuous (machinery) murmur** is heard throughout systole and diastole in patent ductus arteriosus, loudest below the left clavicle.

Fill in the blank: Aortic regurgitation produces an early diastolic murmur at the left sternal border; mitral stenosis produces a mid-diastolic _____ murmur at the apex preceded by an opening snap; patent ductus arteriosus produces a continuous machinery murmur.

Murmur or Condition	Type and Timing	Key Features
Aortic Stenosis	Ejection systolic murmur (crescendo-decrescendo)	Aortic area → carotids, plateau pulse, soft A2
Pulmonary Stenosis	Ejection systolic murmur	Best heard in pulmonary area
Mitral Regurgitation	Pansystolic murmur	Apex → axilla, hyperdynamic apex

Murmur or Condition	Type and Timing	Key Features
Tricuspid Regurgitation	Pansystolic murmur	Left sternal border, Carvallo's sign (↑ with inspiration)
Ventricular Septal Defect (VSD)	Pansystolic murmur	Harsh, best at left sternal border
Aortic Regurgitation	Early diastolic murmur	Left sternal border, sitting forward, collapsing pulse
Mitral Stenosis	Mid-diastolic rumble	Apex, bell of stethoscope, left lateral position, opening snap
Patent Ductus Arteriosus (PDA)	Continuous machinery murmur	Below left clavicle

Table 2.1: Summary of major cardiac murmurs: type, timing, location, and key clinical features

Pilot questions 2.4

1. Describe the Levine grading system for cardiac murmurs. (Linked to SLO 2.5)
2. Distinguish between ejection systolic and pansystolic murmurs with two examples of each. (Linked to SLO 2.5)
3. Describe the murmur of aortic stenosis including its character, location, radiation, and associated signs. (Linked to SLO 2.5)
4. Describe the murmur of mitral stenosis including its character, timing, and the manoeuvres used to accentuate it. (Linked to SLO 2.5)
5. Describe the murmur of aortic regurgitation and explain how it is best heard. (Linked to SLO 2.5)

Series Summary

This Series covered the systematic cardiovascular examination. Peripheral inspection detects central and peripheral cyanosis, clubbing, splinter haemorrhages, Osler's nodes and Janeway lesions (infective endocarditis), xanthomata (hypercholesterolaemia), and malar flush (mitral stenosis). The arterial pulse is assessed for rate, rhythm, volume, and character; abnormal characters include pulsus paradoxus (tamponade), pulsus alternans (severe LV dysfunction), collapsing pulse (aortic regurgitation), and plateau pulse (aortic stenosis). The JVP, assessed at 45 degrees, is elevated in right heart failure; the a wave reflects atrial contraction and the v wave reflects venous filling; Kussmaul's sign is a paradoxical rise on inspiration. Precordial inspection identifies the apex beat, visible pulsations, and scars; palpation locates the apex (normally fifth ICS, midclavicular line), assesses its character (hyperdynamic in volume overload, heaving in pressure overload), and detects thrills and heaves; percussion identifies enlarged cardiac borders and pericardial effusion.

S1 results from mitral and tricuspid valve closure; S2 from aortic and pulmonary valve closure with physiological splitting on inspiration; fixed S2 splitting is the hallmark of ASD; paradoxical splitting occurs in LBBB. S3 indicates LV failure or volume overload; S4 indicates diastolic dysfunction; an opening snap followed by a mid-diastolic murmur characterises mitral stenosis; a pericardial friction rub indicates pericarditis. The four auscultatory areas are mitral (apex), tricuspid (lower left sternal border), pulmonary (left second ICS), and aortic (right second ICS); the diaphragm detects high-pitched sounds and the bell low-pitched sounds. Murmurs are graded 1 to 6 (Grade 4 and above have a thrill); ejection systolic murmurs (crescendo-decrescendo: aortic stenosis, pulmonary stenosis) and pansystolic murmurs (mitral regurgitation, tricuspid regurgitation, VSD) are the main systolic types; aortic regurgitation produces an early diastolic murmur; mitral stenosis a mid-diastolic rumble; patent ductus arteriosus a continuous machinery murmur.

Glossary of Terms

- **Clubbing:** loss of the nail-bed angle with increased nail curvature, associated with cyanotic congenital heart disease and infective endocarditis.
- **Splinter haemorrhages:** dark longitudinal lines under the nails, a peripheral sign of infective endocarditis.
- **Collapsing pulse:** a high-volume pulse with rapid rise and rapid fall, characteristic of aortic regurgitation.
- **Plateau pulse:** a slow-rising, low-volume pulse characteristic of aortic stenosis.
- **Pulsus paradoxus:** an exaggerated fall in systolic blood pressure of more than 10 mmHg during inspiration, seen in cardiac tamponade and severe asthma.
- **Jugular venous pressure (JVP):** the vertical height of venous pulsation above the sternal angle, reflecting right atrial pressure; normal less than 3 to 4 cm.
- **Kussmaul's sign:** a paradoxical rise in JVP on inspiration, seen in cardiac tamponade and constrictive pericarditis.
- **Thrill:** a palpable vibration produced by turbulent blood flow, corresponding to a grade 4 or higher murmur.
- **Ejection systolic murmur:** a crescendo-decrescendo murmur beginning after S1 and ending before S2, produced by turbulent ejection through a stenosed or high-flow valve.
- **Pansystolic murmur:** a uniform murmur from S1 to S2, produced by regurgitation through an incompetent valve or ventricular septal defect.
- **Opening snap:** a high-pitched early diastolic sound produced by the sudden opening of a stenotic mitral valve.
- **Beck's triad:** muffled heart sounds, elevated JVP, and hypotension; the classic triad of cardiac tamponade.

Concept Check Questions [Series 2]

Objective Questions

1. Osler's nodes, Janeway lesions, and splinter haemorrhages are peripheral signs of infective _____. (Linked to SLO 2.1)
2. A collapsing pulse is characteristic of aortic _____; a plateau pulse is characteristic of aortic stenosis. (Linked to SLO 2.2)
3. An elevated JVP indicates raised right atrial pressure; Kussmaul's sign is a paradoxical _____ of JVP on inspiration. (Linked to SLO 2.2)
4. Fixed splitting of the second heart sound is the hallmark of _____ septal defect. (Linked to SLO 2.4)
5. S3 in adults indicates LV failure or volume overload; S4 indicates diastolic _____ and is never normal. (Linked to SLO 2.4)
6. An ejection systolic murmur that is crescendo-decrescendo, loudest in the aortic area, and radiating to the carotids is characteristic of aortic _____. (Linked to SLO 2.5)

Short-Answer Questions

7. Describe the four characteristics of the arterial pulse assessed at the radial artery and name two abnormal pulse characters. (Linked to SLO 2.2)
8. Describe the normal position of the apex beat and explain the significance of displacement and abnormal character. (Linked to SLO 2.3)
9. Distinguish between ejection systolic and pansystolic murmurs with one example of each. (Linked to SLO 2.5)

Long-Answer Questions

10. Describe the systematic cardiovascular examination from general inspection through peripheral signs, JVP, and precordial examination. (Linked to SLOs 2.1 to 2.3)
11. Describe normal and added heart sounds, the cardiac auscultatory areas, and the major cardiac murmurs. (Linked to SLOs 2.4 to 2.5)

Answers to Concept Check Questions [Series 2]

Objective Questions

1. Endocarditis.
2. Regurgitation.
3. Rise.
4. Atrial.
5. Dysfunction.
6. Stenosis.

Short-Answer Questions

7. Rate (60 to 100 per minute), rhythm (regular or irregular), volume (normal, reduced, large), character (quality of waveform). Abnormal characters: collapsing pulse (aortic regurgitation) and plateau pulse (aortic stenosis).
8. Normally in the fifth intercostal space, midclavicular line. Displacement (lateral or inferior) indicates cardiac enlargement. Character: hyperdynamic apex indicates volume overload (MR, AR); heaving apex indicates pressure overload (AS, hypertension).
9. Ejection systolic (crescendo-decrescendo, begins after S1, ends before S2): aortic stenosis. Pansystolic (uniform from S1 to S2): mitral regurgitation.

Long-Answer Questions

10. **Basic response:** General inspection: well or unwell, obvious abnormalities. Face: central cyanosis (lips and tongue), malar flush (MS), xanthelasma (hypercholesterolaemia), anaemic conjunctivae. Hands: clubbing (cyanotic CHD, IE), splinter haemorrhages (IE), Osler's nodes (tender, IE), Janeway lesions (non-tender, IE), xanthomata (familial hypercholesterolaemia), peripheral cyanosis. Arterial pulse: rate (60 to 100), rhythm, volume, character (collapsing: AR; plateau: AS; pulsus paradoxus: tamponade; pulsus alternans: severe LV dysfunction). JVP: assessed at 45 degrees, measured above sternal angle, normal less than 3 to 4 cm; elevated in right heart failure, tamponade,

constrictive pericarditis; a wave (atrial contraction), v wave (venous filling); Kussmaul's sign: rise on inspiration. Precordium: inspection (apex beat, scars, deformities); palpation (apex position: 5th ICS MCL; character: hyperdynamic vs heaving; thrills; parasternal heave: RV hypertrophy); percussion (cardiac borders; dullness: enlargement or effusion; Beck's triad: tamponade).

Value-added response: A systematic approach to the cardiovascular examination, proceeding in a defined sequence from inspection through palpation and auscultation, ensures that no finding is missed; each finding contextualises the next, so that the murmur heard on auscultation is interpreted in light of the pulse character, JVP, and apex beat already established.

11. **Basic response:** S1: mitral and tricuspid closure at onset of systole; best at apex; loud in MS; soft when MV closes early. S2: aortic and pulmonary closure at end of systole; best at base; physiological splitting on inspiration; fixed splitting: ASD; paradoxical splitting: LBBB or AS. S3: early diastole, rapid ventricular filling; normal in young; LV failure or volume overload in adults. S4: late diastole, atrial contraction against stiff ventricle; diastolic dysfunction, hypertension, HCM, AMI; never normal. Opening snap: high-pitched, early diastole after S2; stenotic mitral valve; short S2-snap interval = severe MS. Pericardial friction rub: pericarditis. Auscultatory areas: mitral (apex), tricuspid (lower LSB), pulmonary (left 2nd ICS), aortic (right 2nd ICS); diaphragm for high-pitched sounds, bell for low-pitched. Murmurs: Levine grade 1 to 6 (Grade 4 = thrill); ESM: crescendo-decrescendo (AS: aortic area to carotids, plateau pulse; PS: pulmonary area); PSM: uniform (MR: apex to axilla; TR: LSB, increases with inspiration; VSD: LSB, harsh); diastolic: AR (early diastolic, LSB, sitting forward); MS (mid-diastolic rumble, apex, bell, left lateral, preceded by OS); continuous: PDA (machinery, below left clavicle).

    Value-added response: The integration of auscultatory findings with other examination signs is what makes clinical cardiology powerful: the murmur of AS is far more convincing when accompanied by a plateau pulse, a heaving apex, and a soft A2; the murmur of MS when accompanied by a loud S1, an opening snap, and malar flush;

developing this integrative thinking across multiple examination findings is the goal of bedside clinical training.

Answers to Pilot Questions [Series 2]

Part 2.1

1. Splinter haemorrhages (dark longitudinal nail lines), Osler's nodes (tender nodules on finger pads), Janeway lesions (non-tender erythematous macules on palms), clubbing (associated with IE), and in the eyes: Roth's spots (retinal haemorrhages with pale centres).
2. Rate: normal 60 to 100 beats per minute. Rhythm: regular or irregular (identify if irregularly irregular as in AF). Volume: normal, reduced (low output states), or large (high output states, AR). Character: assessed at the carotid; normal upstroke and waveform.
3. Collapsing (water-hammer) pulse: high-volume with rapid rise and rapid fall, best felt with the arm elevated; associated with aortic regurgitation, patent ductus arteriosus, and high-output states. Plateau pulse: slow-rising, low-volume, sustained; associated with aortic stenosis where LV ejection is prolonged and reduced.
4. The JVP is assessed with the patient positioned at 45 degrees; the internal jugular vein is identified medial to the sternocleidomastoid; the vertical height of the venous pulsation above the sternal angle is measured; the normal JVP is less than 3 to 4 cm above the sternal angle.
5. Elevated JVP indicates raised right atrial pressure, seen in right heart failure, cardiac tamponade, constrictive pericarditis, and SVC obstruction. Kussmaul's sign: a paradoxical rise in JVP on inspiration (normally JVP falls on inspiration); it indicates impaired right heart filling due to restriction or external compression, as in cardiac tamponade and constrictive pericarditis.

Part 2.2

1. Normally located in the fifth intercostal space in the midclavicular line. Displacement laterally (beyond the midclavicular line) or inferiorly indicates cardiac enlargement due to left ventricular dilatation or hypertrophy; the degree of displacement reflects the severity of enlargement.
2. Hyperdynamic apex: forceful, diffuse, and brief; indicates volume overload causing increased stroke volume; associated with aortic regurgitation, mitral regurgitation, and other high-output states. Heaving (pressure-loaded) apex: sustained, forceful, and not displaced; indicates pressure overload causing LV hypertrophy; associated with aortic stenosis and systemic hypertension.
3. A parasternal heave is a sustained lift of the parasternal region felt by placing the palm along the left sternal border; it indicates right ventricular hypertrophy or dilatation, which lifts the sternum during systole. Conditions: pulmonary hypertension, pulmonary stenosis, large atrial septal defect, cor pulmonale.
4. A thrill is a palpable vibration of the chest wall produced by turbulent blood flow through a cardiac valve or defect; it corresponds to a grade 4 or higher murmur on auscultation. Clinical significance: indicates haemodynamically significant valvular or structural disease; location (base: AS or PS; apex: MS or MR; LSB: VSD) guides the diagnosis.
5. Beck's triad consists of muffled (distant) heart sounds, elevated JVP, and hypotension; it is the classic clinical triad of cardiac tamponade, in which fluid in the pericardial space compresses the heart, impairs filling, reduces cardiac output (causing hypotension), and raises systemic venous pressure (elevated JVP) while muffling transmitted heart sounds.

Part 2.3

1. S1: produced by closure of the mitral and tricuspid valves at the onset of ventricular systole; best heard at the apex and lower left sternal border. S2: produced by closure of the aortic and pulmonary valves at the end of systole; best heard at the base of the

heart; normally splits physiologically on inspiration as increased venous return delays pulmonary valve closure.

2. Fixed splitting: S2 splits on both inspiration and expiration with no respiratory variation; hallmark of atrial septal defect where right ventricular filling is prolonged regardless of respiratory phase. Paradoxical splitting: S2 splits on expiration and closes on inspiration (reversed); occurs in left bundle branch block (delayed LV activation delays aortic closure) and severe aortic stenosis.
3. S3: low-pitched, early diastolic sound during rapid ventricular filling, shortly after S2; normal in children and young adults; in patients over 40 indicates LV failure or volume overload (MR, VSD). S4: low-pitched, late diastolic sound just before S1, produced by atrial contraction against a stiff ventricle; indicates diastolic dysfunction; associated with hypertension, HCM, AMI; never a normal finding.
4. An opening snap is a high-pitched, clicking early diastolic sound occurring shortly after S2, produced by the sudden tensing and opening of a rigid stenotic mitral valve. It indicates mitral stenosis. The S2-to-opening-snap interval indicates severity: a short interval (less than 60 ms) indicates severe MS because the high left atrial pressure forces the valve open rapidly.
5. Mitral area: cardiac apex, fifth ICS, midclavicular line (mitral valve sounds, MR, MS). Tricuspid area: lower left sternal border, fourth ICS (tricuspid sounds, TR). Pulmonary area: left second ICS, upper left sternal border (PS, physiological sounds). Aortic area: right second ICS, upper right sternal border (AS, AR). Technique: diaphragm for high-pitched sounds (S1, S2, AR, MR); bell for low-pitched (S3, S4, MS rumble); left lateral position for mitral sounds; sitting forward, breath-holding in expiration for AR.

Part 2.4

1. Levine grading: Grade 1 (very faint, heard only in ideal conditions by experienced auscultator); Grade 2 (faint but immediately audible); Grade 3 (moderately loud, no thrill); Grade 4 (loud with a palpable thrill); Grade 5 (very loud, heard with stethoscope partly off the chest, thrill present); Grade 6 (audible without a stethoscope).

2. Ejection systolic: crescendo-decrescendo, begins after S1, peaks in midsystole, ends before S2; produced by turbulent ejection through stenosed outflow valves. Examples: aortic stenosis (aortic area, radiates to carotids) and pulmonary stenosis (pulmonary area). Pansystolic: uniform intensity from S1 to S2; produced by regurgitation through incompetent AV valves or VSD. Examples: mitral regurgitation (apex, radiates to axilla) and VSD (lower left sternal border, harsh).
3. Aortic stenosis murmur: harsh, ejection systolic (crescendo-decrescendo), loudest in the aortic area (right second ICS), radiates to both carotid arteries; associated signs: plateau pulse, heaving apex, soft or absent A2 (aortic component of S2), systolic thrill in the aortic area.
4. Mitral stenosis murmur: low-pitched, rumbling, mid-diastolic murmur; heard best at the cardiac apex using the bell of the stethoscope with the patient in the left lateral decubitus position; preceded by an opening snap; may have presystolic accentuation in patients in sinus rhythm (due to atrial contraction). Intensity is inversely related to severity (severe MS has a short diastolic filling period and the murmur may be brief).
5. Aortic regurgitation murmur: soft, high-pitched, blowing early diastolic murmur; heard best at the left sternal border (third or fourth ICS) with the patient sitting forward and breath-holding in expiration (reduces lung volume and brings the aortic valve closer to the chest wall); accompanied by a collapsing pulse and a widened pulse pressure.

References and Attributions [Series 2]

Textual References

- Talley, N. J. and O'Connor, S. (2018). Clinical Examination: A Systematic Guide to Physical Diagnosis (8th ed.). Sydney: Elsevier.
- Bickley, L. S. (2017). Bates' Guide to Physical Examination and History Taking (12th ed.). Philadelphia: Wolters Kluwer.
- Kumar, P. and Clark, M. (2017). Kumar and Clark's Clinical Medicine (9th ed.). Edinburgh: Elsevier.
- Bonow, R. O. et al. (2014). Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine (10th ed.). Philadelphia: Elsevier Saunders.

Figures, Plates, Tables, and Boxes

- Figure 2.1: Peripheral cardiovascular signs and JVP assessment. AI prompt: "Two-panel diagram: left panel body outline with callout boxes for peripheral signs (Face: malar flush, xanthelasma, cyanosis; Hands: clubbing, splinter haemorrhages, Osler's nodes, Janeway lesions, xanthomata); right panel patient at 45 degrees with JVP measurement and waveform (a wave, v wave); clean professional infographic." OER search: "cardiovascular peripheral signs JVP jugular venous pressure examination diagram".
- Figure 2.2: Precordial examination: apex beat and precordial findings. AI prompt: "Two-panel diagram: apex beat location and displacement (left); parasternal heave, thrills, Beck's triad (right); clean professional infographic." OER search: "apex beat precordial examination thrills heave cardiac diagram".
- Figure 2.3: Cardiac auscultation heart sounds and auscultatory areas. AI prompt: "Two-panel diagram: cardiac cycle timeline with S4, S1, S2, OS, S3 (left); anterior chest wall with four auscultatory areas and diaphragm vs bell legend (right); clean professional infographic." OER search: "cardiac auscultation heart sounds auscultatory areas diagram S1 S2 S3 S4".
- Table 2.1: Summary of major cardiac murmurs. AI prompt: "Three-column table: Murmur or Condition, Type and Timing, Key Features; eight rows from Aortic Stenosis to

PDA; alternating row shading." OER search: "cardiac murmurs summary table systolic diastolic aortic stenosis mitral regurgitation".

Series 3: Hypertension — Epidemiology, Pathophysiology, and Management

- **Part 3.1: Epidemiology and Classification of Hypertension**
  - Sub Part 3.1.1: Definition and classification of hypertension
  - Sub Part 3.1.2: Epidemiology of hypertension in Nigeria and globally
  - Sub Part 3.1.3: Risk factors and causes of hypertension
- **Part 3.2: Pathophysiology of Hypertension**
  - Sub Part 3.2.1: Determinants of blood pressure
  - Sub Part 3.2.2: Mechanisms of primary hypertension
  - Sub Part 3.2.3: Target organ damage in hypertension
- **Part 3.3: Clinical Evaluation of the Hypertensive Patient**
  - Sub Part 3.3.1: History and physical examination
  - Sub Part 3.3.2: Investigations in hypertension
  - Sub Part 3.3.3: Secondary causes of hypertension
- **Part 3.4: Management of Hypertension**
  - Sub Part 3.4.1: Lifestyle modification
  - Sub Part 3.4.2: Pharmacological management
  - Sub Part 3.4.3: Hypertensive urgency and emergency

Introduction

Hypertension is the most prevalent cardiovascular risk factor in Nigeria and sub-Saharan Africa, affecting approximately 30 to 40 percent of adults. Despite its prevalence, awareness, treatment, and control rates remain low, making hypertension the leading preventable cause of stroke, heart failure, kidney disease, and premature cardiovascular death in the region. Understanding hypertension comprehensively, from its definition and epidemiology through its pathophysiology to its clinical evaluation and management, is therefore a clinical priority for every practitioner.

This Series covers the definition and classification of hypertension, its epidemiology in the Nigerian and global context, the pathophysiological mechanisms underlying primary hypertension, target organ damage, and the causes of secondary hypertension. It then describes the clinical evaluation of the hypertensive patient and provides a comprehensive account of management, from lifestyle modification through antihypertensive pharmacotherapy to the urgent management of hypertensive crises.

The skills and knowledge developed in this Series are immediately applicable in every clinical setting, as hypertension is encountered as a primary diagnosis, a comorbidity, and a risk factor in virtually every clinical specialty.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 3.1** define hypertension, state its classification, and describe its epidemiology in Nigeria and globally (Linked to Part 3.1),
- **SLO 3.2** describe the determinants of blood pressure and explain the pathophysiological mechanisms of primary hypertension and target organ damage (Linked to Part 3.2),
- **SLO 3.3** conduct a focused clinical evaluation of the hypertensive patient including history, examination, and appropriate investigations (Linked to Part 3.3),

- **SLO 3.4** describe the lifestyle and pharmacological management of hypertension (Linked to Part 3.4), and
- **SLO 3.5** distinguish between hypertensive urgency and emergency and describe the management of each (Linked to Part 3.4).

3.1 Epidemiology and Classification of Hypertension

3.1.1 Definition and classification of hypertension

Hypertension is defined as a sustained elevation of blood pressure at or above **140 mmHg systolic and/or 90 mmHg diastolic** on at least two separate readings on at least two clinic visits, in the absence of antihypertensive treatment. This threshold is used by the European Society of Cardiology and the Nigerian Hypertension Society. The 2017 ACC/AHA guidelines use a lower threshold of 130/80 mmHg.

Blood pressure is classified as: **Normal** (below 120/80 mmHg), **Elevated** (120 to 129 systolic, below 80 diastolic), **Stage 1 hypertension** (130 to 139 systolic or 80 to 89 diastolic by ACC/AHA; 140 to 159 or 90 to 99 by ESC/NHS), **Stage 2 hypertension** (140/90 and above by ACC/AHA; 160/100 and above by ESC/NHS), and **Stage 3 or severe hypertension** (180/110 and above). **Isolated systolic hypertension** (systolic above 140, diastolic below 90) is the most common pattern in the elderly.

Fill in the blank: Hypertension is defined as sustained BP at or above 140/90 mmHg (ESC/NHS) or 130/80 mmHg (ACC/AHA); isolated systolic hypertension (systolic above 140, diastolic below 90) is the most common pattern in the _____.

3.1.2 Epidemiology of hypertension in Nigeria and globally

Globally, hypertension affects approximately **1.28 billion adults** aged 30 to 79 years (WHO 2021), with the highest prevalence in low- and middle-income countries. Sub-Saharan Africa carries the greatest regional burden, with age-standardised hypertension prevalence among the

highest in the world. In Nigeria, community surveys report prevalence rates of **28 to 46 percent** in adults, translating to tens of millions of individuals with hypertension.

Despite this burden, the **awareness-treatment-control cascade** remains poor in Nigeria: only about 30 to 40 percent of hypertensive individuals are aware of their diagnosis, fewer than 30 percent are on treatment, and fewer than 10 to 15 percent achieve blood pressure control. The consequences include Nigeria's **very high burden of hypertension-related stroke**, which accounts for 45 to 77 percent of all strokes and is the leading cause of death and disability in Nigerian adults.

Fill in the blank: Hypertension affects approximately 1.28 billion adults globally; in Nigeria prevalence is 28 to 46 percent but control rates are below 15 percent, contributing to a very high burden of hypertension-related _____.

3.1.3 Risk factors and causes of hypertension

Primary (essential) hypertension accounts for approximately 90 to 95 percent of all cases and has no single identifiable cause; it results from the interaction of multiple genetic and environmental factors. Risk factors include **age** (BP increases with age), **family history**, **excess dietary sodium**, **obesity**, **physical inactivity**, **excessive alcohol**, **psychosocial stress**, and diabetes mellitus.

Secondary hypertension (5 to 10 percent of cases) has an identifiable underlying cause. The most common causes in clinical practice are **chronic kidney disease** (the most common secondary cause overall), **renovascular disease** (renal artery stenosis causing renal ischaemia and renin-angiotensin activation), **primary aldosteronism** (adrenal adenoma or hyperplasia causing excess aldosterone), **phaeochromocytoma** (catecholamine-secreting tumour), and in younger women, **oral contraceptive pill use**.

Fill in the blank: Primary hypertension (90 to 95 percent of cases) has no single cause; secondary hypertension causes include chronic kidney disease, renovascular disease, primary _____, and phaeochromocytoma.

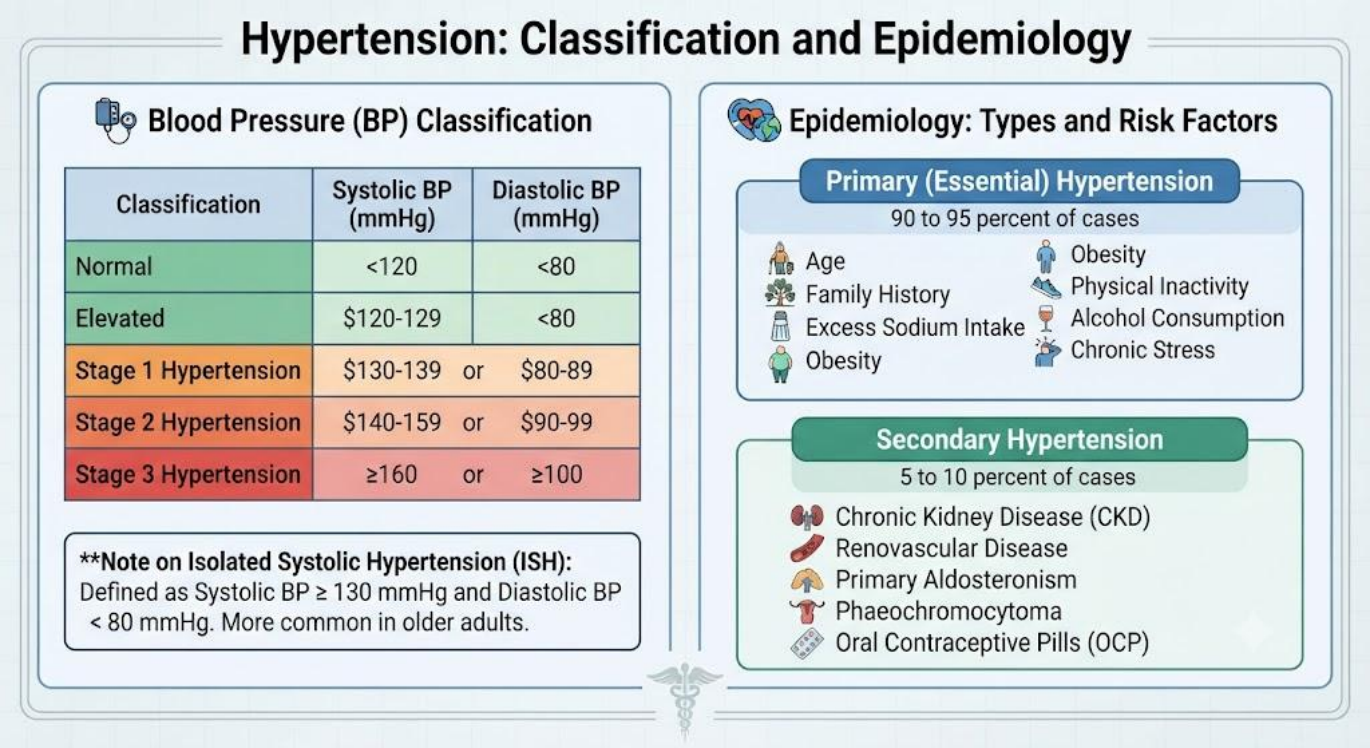

Figure 3.1: Blood pressure classification and the causes of primary and secondary hypertension

Pilot questions 3.1

1. Define hypertension and state the ESC/NHS and ACC/AHA thresholds. (Linked to SLO 3.1)
2. Describe the blood pressure classification system. (Linked to SLO 3.1)
3. Describe the burden of hypertension in Nigeria in terms of prevalence, awareness, and control. (Linked to SLO 3.1)
4. List the risk factors for primary hypertension. (Linked to SLO 3.1)
5. Name four causes of secondary hypertension. (Linked to SLO 3.1)

3.2 Pathophysiology of Hypertension

3.2.1 Determinants of blood pressure

Blood pressure is determined by **cardiac output (CO)** and **total peripheral resistance (TPR)** according to the equation BP = CO x TPR. **Cardiac output** is the product of heart rate and

stroke volume; stroke volume depends on preload (ventricular filling), contractility, and afterload. **Total peripheral resistance** is determined by arteriolar tone, which is regulated by the autonomic nervous system, vasoactive hormones (angiotensin II, aldosterone, endothelin), and local autoregulatory mechanisms.

Volume regulation by the kidneys through sodium and water handling is a critical determinant of blood pressure: increased sodium retention expands intravascular volume, increases preload and cardiac output, and raises blood pressure. The **renin-angiotensin-aldosterone system (RAAS)** is a central hormonal axis linking renal perfusion to blood pressure: reduced renal perfusion triggers renin release, angiotensin II production (causing vasoconstriction and aldosterone secretion), and aldosterone-mediated sodium and water retention.

Fill in the blank: BP equals cardiac output multiplied by total peripheral resistance; cardiac output depends on heart rate and stroke volume; TPR is regulated by arteriolar tone; the _____ system links renal perfusion to blood pressure through renin, angiotensin II, and aldosterone.

3.2.2 Mechanisms of primary hypertension

Primary hypertension develops through the interaction of multiple mechanisms. **Increased sympathetic nervous system activity** raises heart rate, cardiac output, and arteriolar tone. **RAAS overactivity** promotes sodium retention and vasoconstriction. **Impaired renal sodium excretion** (from genetic variants in renal tubular transport) causes volume expansion and elevated blood pressure. **Endothelial dysfunction** reduces nitric oxide-mediated vasodilation and promotes vasoconstriction and vascular inflammation.

Structural vascular changes develop with sustained hypertension: arteriolar **hypertrophy and remodelling** increase wall-to-lumen ratio, amplifying the pressor effect of any given stimulus and perpetuating hypertension through a vicious cycle. **Insulin resistance** (common in obese hypertensive patients) contributes through stimulation of sympathetic activity and RAAS activation. These mechanisms collectively explain why blood pressure tends to rise with age and why multiple drug mechanisms are often needed to achieve control.

Fill in the blank: Primary hypertension mechanisms include increased sympathetic activity, RAAS overactivity, impaired renal sodium excretion, endothelial dysfunction, and arteriolar _____ that amplify pressor stimuli and perpetuate hypertension.

3.2.3 Target organ damage in hypertension

Sustained hypertension damages multiple target organs through the effects of elevated pressure and accelerated atherosclerosis. **Cardiac damage** includes left ventricular hypertrophy (LVH: the heart adapts to increased afterload by thickening the LV wall, detectable on ECG and echocardiography), diastolic dysfunction, and progression to heart failure. Coronary artery disease is accelerated by hypertension-driven atherosclerosis.

Cerebrovascular damage includes ischaemic stroke (from thromboembolism in atherosclerotic vessels), haemorrhagic stroke (from rupture of Charcot-Bouchard microaneurysms in small penetrating arteries), and hypertensive encephalopathy. **Renal damage** manifests as hypertensive nephrosclerosis: arteriolar changes reduce glomerular perfusion, causing progressive CKD, proteinuria, and eventual renal failure. **Hypertensive retinopathy** causes arteriolar narrowing, arteriovenous nipping, flame-shaped haemorrhages, cotton-wool spots, and in severe cases papilloedema.

Fill in the blank: Target organ damage in hypertension includes LVH and heart failure (cardiac), ischaemic and haemorrhagic stroke (cerebrovascular), hypertensive nephrosclerosis (renal), and hypertensive _____ with arteriolar changes and papilloedema.

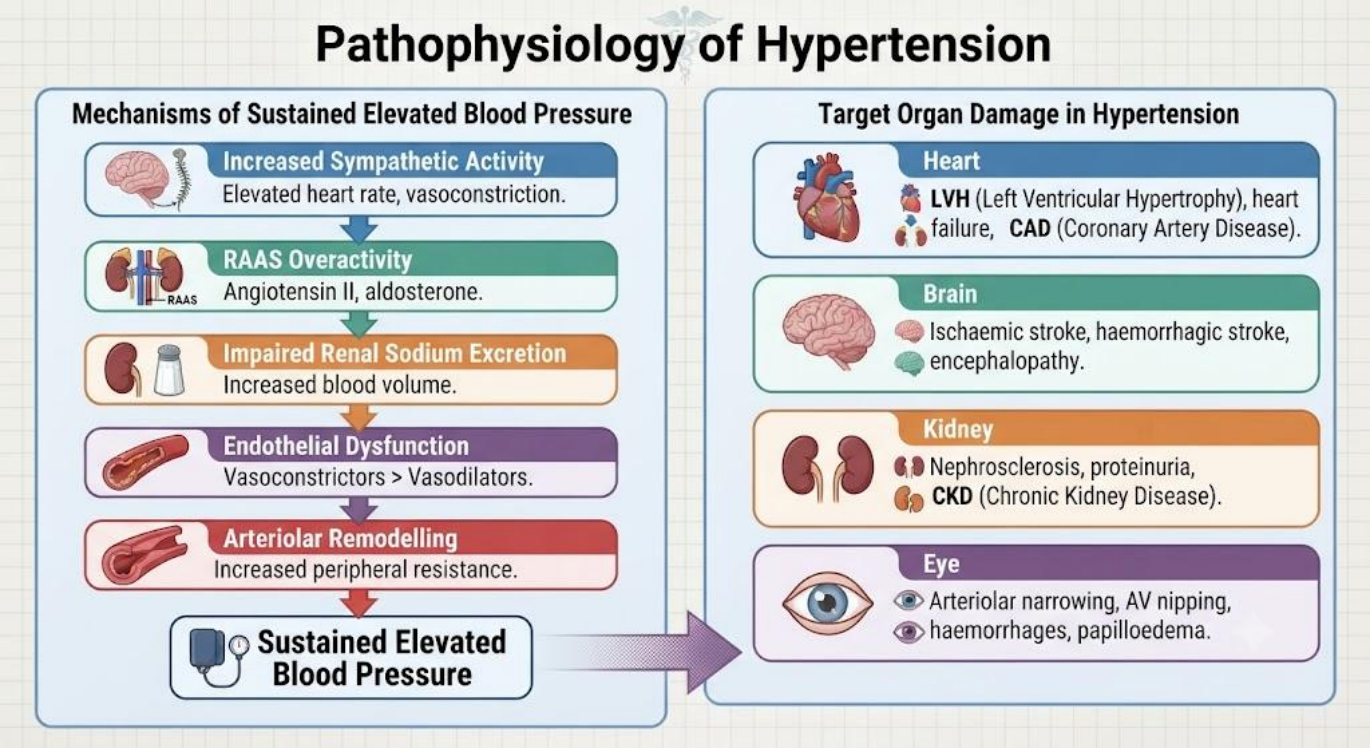

Figure 3.2: Mechanisms of primary hypertension and the four major sites of target organ damage

Pilot questions 3.2

1. State the equation for blood pressure and explain the role of cardiac output and total peripheral resistance. (Linked to SLO 3.2)
2. Explain the role of the renin-angiotensin-aldosterone system in blood pressure regulation. (Linked to SLO 3.2)
3. Describe four mechanisms contributing to the development of primary hypertension. (Linked to SLO 3.2)
4. Describe the cardiac target organ damage caused by hypertension. (Linked to SLO 3.2)
5. Describe hypertensive retinopathy and its grading. (Linked to SLO 3.2)

3.3 Clinical Evaluation of the Hypertensive Patient

3.3.1 History and physical examination

The history of a hypertensive patient aims to establish the duration and severity of hypertension, symptoms of target organ damage (chest pain, dyspnoea, neurological symptoms, visual disturbance, haematuria), current antihypertensive medications and adherence, cardiovascular risk factors, and symptoms suggesting secondary hypertension (episodic headache, palpitations, and sweating suggesting phaeochromocytoma; muscle weakness and polyuria suggesting primary aldosteronism; symptoms of renal disease).

Physical examination includes accurate **blood pressure measurement** in both arms (significant asymmetry may indicate aortic coarctation or arterial disease), measurement of **pulse rate and rhythm**, assessment of **BMI and waist circumference**, cardiovascular examination for LVH signs (heaving apex, loud A2, S4), peripheral pulses (absent femoral pulses in coarctation), fundoscopy (hypertensive retinopathy), and assessment of **target organ damage** in all systems.

Fill in the blank: History in hypertension covers duration, target organ damage symptoms, medications, risk factors, and secondary causes; examination includes BP in both arms, BMI, cardiovascular signs of LVH, peripheral pulses, fundoscopy, and assessment of _____ organ damage.

3.3.2 Investigations in hypertension

Baseline investigations in all hypertensive patients serve three purposes: to assess target organ damage, to identify cardiovascular risk factors, and to screen for secondary causes. Minimum investigations include **urinalysis and urine albumin-to-creatinine ratio** (proteinuria: renal damage), **serum electrolytes and creatinine** (renal function, hypokalaemia suggesting primary aldosteronism), **fasting blood glucose and lipids** (diabetes, dyslipidaemia), and **12-lead ECG** (LVH by voltage criteria and repolarisation changes).

Echocardiography provides a more sensitive assessment of LVH and diastolic function than ECG and is indicated when ECG LVH changes are present or when cardiac symptoms

accompany hypertension. **Renal ultrasound** is indicated when CKD or renovascular disease is suspected. Additional investigations for suspected secondary causes include **plasma aldosterone-to-renin ratio** (primary aldosteronism), **24-hour urinary catecholamines or plasma metanephrines** (phaeochromocytoma), and **renal Doppler ultrasound or MRA** (renovascular disease).

Fill in the blank: Baseline investigations include urinalysis, serum electrolytes and creatinine, fasting glucose and lipids, and ECG; echocardiography assesses LVH and diastolic function; plasma aldosterone-to-renin ratio screens for primary _____.

Investigation	Clinical Significance
Urinalysis and ACR	Detects proteinuria, marker of renal damage
Electrolytes and Creatinine	Assesses renal function, hypokalaemia (suggests secondary causes)
Fasting Glucose and Lipids	Evaluates cardiovascular risk factors
ECG	Detects left ventricular hypertrophy (LVH)

Investigation	Clinical Significance
Aldosterone-to-Renin Ratio	Screens for primary aldosteronism
Plasma Metanephrines	Detects phaeochromocytoma
Renal Doppler or MRA	Identifies renovascular hypertension
Echocardiography	Assesses LVH, diastolic function

Table 3.1: Investigations in hypertension: baseline tests and additional tests for secondary causes

3.3.3 Secondary causes of hypertension

Secondary hypertension should be suspected when hypertension presents in young patients (below 30 years), when hypertension is resistant to three or more antihypertensive drugs at

adequate doses, when there is a sudden worsening of previously controlled hypertension, or when clinical features suggest a specific secondary cause.

Chronic kidney disease is the most common secondary cause; elevated creatinine and proteinuria with reduced GFR confirm the diagnosis. **Renovascular hypertension** from renal artery stenosis (atherosclerotic in older patients; fibromuscular dysplasia in young women) causes hypertension refractory to medical therapy, with an abdominal bruit on examination. **Primary aldosteronism** presents with hypokalaemia, hypertension, and metabolic alkalosis; confirmed by elevated aldosterone-to-renin ratio. **Phaeochromocytoma** presents with episodic hypertension, headache, palpitations, and diaphoresis; diagnosed by elevated plasma metanephrines or 24-hour urinary catecholamines.

Fill in the blank: Secondary hypertension is suspected in young patients, resistant hypertension, or with specific features: CKD (proteinuria, reduced GFR), renovascular disease (abdominal bruit), primary aldosteronism (hypokalaemia), phaeochromocytoma (episodic hypertension, headache, _____, diaphoresis).

Pilot questions 3.3

1. Describe the key components of the history in a hypertensive patient. (Linked to SLO 3.3)
2. Describe the physical examination findings in a hypertensive patient with LVH. (Linked to SLO 3.3)
3. List the baseline investigations in hypertension and state the purpose of each. (Linked to SLO 3.3)
4. Describe the clinical features and diagnostic approach to primary aldosteronism. (Linked to SLO 3.3)
5. Describe the clinical features and diagnostic approach to phaeochromocytoma. (Linked to SLO 3.3)

3.4 Management of Hypertension

3.4.1 Lifestyle modification

Lifestyle modification is recommended for all hypertensive patients regardless of pharmacological treatment and can reduce systolic BP by 5 to 20 mmHg when implemented rigorously. Key interventions include **dietary sodium restriction** (below 6 g NaCl or 2.4 g sodium per day), which reduces SBP by 2 to 8 mmHg; **weight reduction** (each 10 kg of weight loss reduces SBP by 5 to 20 mmHg); and **physical activity** (30 to 45 minutes of moderate aerobic exercise at least 5 days per week, reducing SBP by 4 to 9 mmHg).

Additional lifestyle measures include **alcohol reduction** (limit to below 2 units per day for men and 1 unit per day for women), **smoking cessation** (does not directly lower BP but dramatically reduces cardiovascular risk), the **DASH diet** (Dietary Approaches to Stop Hypertension, rich in fruits, vegetables, and low-fat dairy, reducing SBP by 8 to 14 mmHg), and **stress management**. In Nigeria, reducing excessive salt use in cooking and processed foods is a particularly important public health priority.

Fill in the blank: Lifestyle modifications for hypertension include dietary sodium restriction (below 6 g NaCl), weight reduction, physical activity, alcohol reduction, smoking cessation, and the _____ diet rich in fruits, vegetables, and low-fat dairy.

3.4.2 Pharmacological management

Five major antihypertensive drug classes are recommended as first-line therapy: **ACE inhibitors (ACEi)** or **angiotensin receptor blockers (ARBs)** (reduce angiotensin II activity; preferred in diabetes, CKD with proteinuria, heart failure), **calcium channel blockers (CCBs)** (amlodipine; effective in black African patients and the elderly), **thiazide diuretics** (hydrochlorothiazide, chlorthalidone; sodium and volume reduction; effective in black patients), and **beta-blockers** (atenolol, bisoprolol; preferred with concurrent ischaemic heart disease or heart failure; not first-line in uncomplicated hypertension).

In Nigerian and African patients, **CCBs and thiazide diuretics** are generally more effective than ACEi/ARBs as monotherapy due to the low-renin, volume-dependent hypertension phenotype common in this population. Most patients require **combination therapy** to achieve target blood pressure; preferred combinations include ACEi or ARB plus CCB, and ACEi or ARB plus thiazide diuretic. The BP target is generally **below 130/80 mmHg** in patients with diabetes or CKD, and below 140/90 mmHg in uncomplicated hypertension.

Fill in the blank: First-line antihypertensives include ACEi or ARBs, calcium channel blockers, thiazide diuretics, and beta-blockers; in African patients CCBs and _____ diuretics are generally more effective as monotherapy; most patients need combination therapy.

3.4.3 Hypertensive urgency and emergency

Hypertensive urgency is severely elevated BP (typically above 180/120 mmHg) without evidence of acute target organ damage. Patients may be asymptomatic or have headache; there are no neurological deficits, no pulmonary oedema, no acute renal deterioration, and no chest pain. Management involves **oral antihypertensive therapy** with a short-acting agent (oral captopril, amlodipine, or labetalol) and close outpatient follow-up; BP should be lowered gradually over 24 to 48 hours, not rapidly.

Hypertensive emergency (hypertensive crisis) is severely elevated BP with evidence of acute target organ damage, including **hypertensive encephalopathy** (confusion, seizures, papilloedema), **acute aortic dissection**, **acute myocardial infarction**, **acute pulmonary oedema**, and **acute kidney injury**. Management requires **immediate admission to a high-dependency or intensive care unit** and **intravenous antihypertensive therapy** with short-acting titratable agents (IV labetalol, IV sodium nitroprusside, IV hydralazine); BP should be reduced by no more than 25 percent in the first hour to avoid ischaemia from sudden pressure drop.

Fill in the blank: Hypertensive urgency is BP above 180/120 mmHg without organ damage, managed with oral agents over 24 to 48 hours; hypertensive emergency includes acute organ damage and requires IV antihypertensives with BP reduction of no more than _____ percent in the first hour.

Urgency	Emergency
Definition: BP >180/120 mmHg without acute target organ damage	BP >180/120 mmHg **with** acute target organ damage (encephalopathy, dissection, AMI, pulmonary oedema, AKI)
Clinical Features: Often asymptomatic or mild headache, no organ dysfunction	Severe symptoms: chest pain, dyspnoea, neurological deficits, acute renal impairment
Investigations: Basic labs, ECG, urinalysis to exclude organ damage	Full work-up: ECG, cardiac enzymes, renal function, imaging (CT/MRI, chest X-ray, echocardiography)
Management: Oral antihypertensives, gradual BP reduction over 24–48 hours	Admit to HDU/ICU, IV antihypertensives, reduce BP by max 25% in first hour

Table 3.2: Hypertensive urgency versus hypertensive emergency: definition, features, and management

Pilot questions 3.4

1. Describe the lifestyle modifications recommended for hypertension and estimate the BP reduction achievable with each. (Linked to SLO 3.4)
2. List the five major antihypertensive drug classes and describe the preferred indications for each. (Linked to SLO 3.4)
3. Explain why CCBs and thiazide diuretics are preferred as first-line therapy in Nigerian patients. (Linked to SLO 3.4)
4. Distinguish between hypertensive urgency and hypertensive emergency. (Linked to SLO 3.5)
5. Describe the management of hypertensive emergency including the drugs used and the rate of BP reduction. (Linked to SLO 3.5)

Series Summary

This Series examined hypertension comprehensively. Hypertension is defined as BP at or above 140/90 mmHg (ESC/NHS) or 130/80 mmHg (ACC/AHA); BP is classified from normal (below 120/80) through elevated, Stage 1, Stage 2, to Stage 3 (180/110 and above); isolated systolic hypertension is the most common pattern in the elderly. Hypertension affects approximately 28 to 46 percent of Nigerian adults with control rates below 15 percent, contributing to a very high burden of stroke and cardiovascular disease. Primary hypertension (90 to 95 percent) results from interacting genetic and environmental risk factors; secondary hypertension (5 to 10 percent) has identifiable causes including CKD, renovascular disease, primary aldosteronism, and phaeochromocytoma. BP equals cardiac output multiplied by total peripheral resistance; the RAAS links renal perfusion to BP through renin, angiotensin II vasoconstriction, and aldosterone-mediated sodium retention. Primary hypertension mechanisms include increased sympathetic activity, RAAS overactivity, impaired sodium excretion, endothelial dysfunction, and arteriolar remodelling.

Target organ damage includes LVH and heart failure (cardiac), ischaemic and haemorrhagic stroke (cerebrovascular), hypertensive nephrosclerosis (renal), and hypertensive retinopathy. Clinical evaluation covers history (duration, TOD symptoms, secondary cause features), examination (BP in both arms, LVH signs, fundoscopy), and investigations (urinalysis, electrolytes, creatinine, glucose, lipids, ECG; echocardiography for LVH; specific tests for secondary causes). Lifestyle modification (sodium restriction, weight loss, exercise, DASH diet, alcohol reduction) can reduce SBP by 5 to 20 mmHg. First-line drugs include ACEi or ARBs, CCBs, thiazide diuretics, and beta-blockers; CCBs and thiazides are more effective as monotherapy in African patients; target BP is below 140/90 (uncomplicated) or below 130/80 (diabetes/CKD). Hypertensive urgency (above 180/120 without organ damage) is managed with oral agents over 24 to 48 hours; hypertensive emergency (with organ damage) requires IV therapy with no more than 25 percent BP reduction in the first hour.

Glossary of Terms

- **Primary (essential) hypertension:** hypertension with no identifiable single cause, resulting from the interaction of multiple genetic and environmental factors; accounts for 90 to 95 percent of cases.
- **Secondary hypertension:** hypertension with an identifiable underlying cause such as CKD, renovascular disease, primary aldosteronism, or phaeochromocytoma.
- **Total peripheral resistance (TPR):** the resistance to blood flow offered by the arterioles; a key determinant of blood pressure.
- **Renin-angiotensin-aldosterone system (RAAS):** the hormonal axis linking renal perfusion to blood pressure through renin release, angiotensin II production, and aldosterone-mediated sodium retention.
- **Left ventricular hypertrophy (LVH):** thickening of the left ventricular wall in response to pressure overload; a marker of hypertensive target organ damage.
- **Hypertensive nephrosclerosis:** progressive renal damage from hypertension, characterised by arteriolar changes, glomerular ischaemia, proteinuria, and declining GFR.
- **Hypertensive retinopathy:** retinal damage from hypertension, graded by arteriolar changes, haemorrhages, cotton-wool spots, and papilloedema.
- **Hypertensive urgency:** severely elevated BP (above 180/120 mmHg) without evidence of acute target organ damage.
- **Hypertensive emergency:** severely elevated BP with evidence of acute target organ damage, requiring immediate IV antihypertensive therapy.
- **DASH diet:** Dietary Approaches to Stop Hypertension; a diet rich in fruits, vegetables, whole grains, and low-fat dairy that reduces systolic BP by 8 to 14 mmHg.
- **Primary aldosteronism:** excess aldosterone secretion from an adrenal adenoma or bilateral adrenal hyperplasia, causing hypertension and hypokalaemia.
- **Phaeochromocytoma:** a catecholamine-secreting adrenal tumour causing episodic hypertension, headache, palpitations, and diaphoresis.

Concept Check Questions [Series 3]

Objective Questions

1. Hypertension is defined as sustained BP at or above _____ mmHg systolic and/or 90 mmHg diastolic by the ESC/NHS criteria. (Linked to SLO 3.1)
2. Primary hypertension accounts for 90 to 95 percent of cases; secondary hypertension accounts for 5 to 10 percent and has an _____ underlying cause. (Linked to SLO 3.1)
3. BP equals cardiac output multiplied by total peripheral _____; the RAAS regulates BP through renin, angiotensin II, and aldosterone. (Linked to SLO 3.2)
4. Left ventricular _____ is the cardiac target organ damage response to sustained pressure overload from hypertension. (Linked to SLO 3.2)
5. In Nigerian patients, calcium channel blockers and _____ diuretics are generally more effective as monotherapy due to the low-renin, volume-dependent hypertension phenotype. (Linked to SLO 3.4)
6. Hypertensive emergency requires IV antihypertensive therapy with BP reduction of no more than _____ percent in the first hour to avoid ischaemia. (Linked to SLO 3.5)

Short-Answer Questions

7. List four causes of secondary hypertension and state the key clinical feature of each. (Linked to SLO 3.1)
8. Describe the baseline investigations in a hypertensive patient and state the purpose of each. (Linked to SLO 3.3)
9. Distinguish between hypertensive urgency and hypertensive emergency. (Linked to SLO 3.5)

Long-Answer Questions

10. Describe the pathophysiology of primary hypertension and the target organ damage it causes. (Linked to SLO 3.2)
11. Describe the pharmacological management of hypertension, including drug classes, preferred indications, and BP targets. (Linked to SLO 3.4)

Answers to Concept Check Questions [Series 3]

Objective Questions

1. 140.
2. Identifiable.
3. Resistance.
4. Hypertrophy.
5. Thiazide.
6. 25.

Short-Answer Questions

7. CKD: proteinuria and elevated creatinine. Renovascular disease: abdominal bruit, refractory hypertension, renal artery stenosis. Primary aldosteronism: hypokalaemia, elevated aldosterone-to-renin ratio. Phaeochromocytoma: episodic hypertension, headache, palpitations, diaphoresis.
8. Urinalysis and urine ACR: detect proteinuria (renal damage). Serum electrolytes and creatinine: assess renal function; hypokalaemia suggests primary aldosteronism. Fasting glucose and lipids: identify diabetes and dyslipidaemia. ECG: detect LVH by voltage criteria.
9. Urgency: BP above 180/120 without acute organ damage; managed with oral agents over 24 to 48 hours. Emergency: BP above 180/120 with acute organ damage (encephalopathy, aortic dissection, AMI, pulmonary oedema, AKI); requires hospital admission and IV antihypertensives.

Long-Answer Questions

10. **Basic response:** Mechanisms: increased sympathetic activity (raises HR, CO, arteriolar tone); RAAS overactivity (angiotensin II vasoconstriction, aldosterone sodium retention); impaired renal sodium excretion (volume expansion); endothelial dysfunction (reduced nitric oxide vasodilation); arteriolar hypertrophy and remodelling (increased wall-to-

lumen ratio amplifies pressor effects). Target organ damage: Cardiac (LVH, diastolic dysfunction, heart failure, accelerated coronary artery disease); Cerebrovascular (ischaemic stroke from atherothrombosis, haemorrhagic stroke from Charcot-Bouchard aneurysm rupture, hypertensive encephalopathy); Renal (hypertensive nephrosclerosis: arteriolar changes, reduced GFR, proteinuria, progressive CKD); Retinal (arteriolar narrowing, AV nipping, haemorrhages, cotton-wool spots, papilloedema in severe cases).

Value-added response: The vascular consequences of hypertension develop over years, but the pathological process begins with endothelial dysfunction and arteriolar remodelling long before clinical events occur; this is why treating asymptomatic hypertension aggressively prevents events that are clinically silent until they are catastrophic.

11. **Basic response:** Drug classes: ACEi or ARBs (reduce angiotensin II; preferred in diabetes, CKD with proteinuria, heart failure; avoid in bilateral renal artery stenosis and pregnancy); CCBs (amlodipine; effective in African patients, elderly, isolated systolic hypertension; safe in most comorbidities); Thiazide diuretics (sodium and volume reduction; effective in African patients; avoid in gout); Beta-blockers (atenolol, bisoprolol; preferred with IHD or heart failure; not first-line uncomplicated). Preferred combinations: ACEi or ARB plus CCB; ACEi or ARB plus thiazide. In Nigerian patients: CCBs and thiazides more effective as monotherapy. BP targets: below 140/90 mmHg in uncomplicated hypertension; below 130/80 mmHg in diabetes or CKD. Adherence: simplify regimen, use once-daily drugs, fixed-dose combinations where available.

    Value-added response: Combination therapy is the norm rather than the exception in hypertension management: most patients require two or more agents to achieve target BP, and starting with low-dose combination therapy rather than monotherapy at full dose achieves faster control with fewer side effects, because the dose-response curves of antihypertensives are steep early but each drug has diminishing returns at higher doses.

Answers to Pilot Questions [Series 3]

Part 3.1

1. Hypertension is defined as sustained BP at or above 140/90 mmHg on at least two readings on at least two clinic visits (ESC/NHS threshold); the 2017 ACC/AHA guidelines use a lower threshold of 130/80 mmHg.
2. Normal: below 120/80. Elevated: 120 to 129 systolic, below 80 diastolic. Stage 1: 130 to 139 or 80 to 89 (ACC/AHA); 140 to 159 or 90 to 99 (ESC/NHS). Stage 2: 140/90 and above (ACC/AHA); 160/100 and above (ESC/NHS). Stage 3 or severe: 180/110 and above. Isolated systolic hypertension: systolic above 140, diastolic below 90; most common in the elderly.
3. Hypertension affects 28 to 46 percent of Nigerian adults; only about 30 to 40 percent of those affected are aware of their diagnosis; fewer than 30 percent are on treatment; fewer than 10 to 15 percent achieve blood pressure control; this poor cascade contributes to Nigeria's very high burden of hypertension-related stroke.
4. Age, family history, excess dietary sodium, obesity, physical inactivity, excessive alcohol consumption, psychosocial stress, and diabetes mellitus.
5. Chronic kidney disease, renovascular disease (renal artery stenosis), primary aldosteronism, phaeochromocytoma.

Part 3.2

1. BP equals cardiac output multiplied by total peripheral resistance. Cardiac output equals heart rate multiplied by stroke volume (which depends on preload, contractility, and afterload). TPR is determined by arteriolar tone regulated by the autonomic nervous system, RAAS, and local mechanisms.
2. Reduced renal perfusion triggers renin release from juxtaglomerular cells; renin cleaves angiotensinogen to angiotensin I; ACE converts angiotensin I to angiotensin II; angiotensin II causes arteriolar vasoconstriction (raising TPR) and stimulates aldosterone

secretion from the adrenal cortex; aldosterone promotes sodium and water reabsorption in the renal tubule, expanding blood volume and raising BP.

3. Increased sympathetic nervous system activity (raises HR, CO, and arteriolar tone); RAAS overactivity (promotes vasoconstriction and sodium retention); impaired renal sodium excretion (genetic variants causing volume expansion); endothelial dysfunction (reduced nitric oxide vasodilation, promoting vasoconstriction and vascular inflammation).
4. LVH (thickening of the LV wall in response to increased afterload, detectable on ECG and echocardiography), diastolic dysfunction (impaired LV relaxation), progression to systolic heart failure with further pressure overload, and accelerated atherosclerosis causing coronary artery disease and acute myocardial infarction.
5. Hypertensive retinopathy is graded: Grade I (arteriolar narrowing and increased light reflex); Grade II (AV nipping where arterioles cross over venules); Grade III (flame-shaped haemorrhages and cotton-wool spots indicating retinal ischaemia); Grade IV (papilloedema, indicating malignant hypertension with raised intracranial pressure). Grades III and IV indicate severe target organ damage requiring urgent treatment.

Part 3.3

1. Duration and level of BP; symptoms of target organ damage (chest pain, dyspnoea, neurological symptoms, visual disturbance, haematuria, ankle swelling); current antihypertensive medications and adherence; cardiovascular risk factors (smoking, diabetes, dyslipidaemia, family history); symptoms suggesting secondary causes (episodic headache, palpitations, sweating: phaeochromocytoma; muscle weakness, polyuria: primary aldosteronism; symptoms of renal disease).
2. Heaving (pressure-loaded) apex beat displaced laterally (indicating LVH and cardiac enlargement), loud aortic component of S2 (A2), fourth heart sound (S4: diastolic dysfunction from LVH), possible signs of heart failure (elevated JVP, basal crepitations, peripheral oedema) if decompensated.
3. Urinalysis and urine ACR: detect proteinuria indicating renal target organ damage. Serum electrolytes and creatinine: assess renal function; hypokalaemia suggests primary

aldosteronism. Fasting blood glucose and lipids: identify diabetes and dyslipidaemia. 12-lead ECG: detect LVH by voltage criteria (Sokolow-Lyon index) and repolarisation changes.

4. Primary aldosteronism presents with hypertension (often resistant), hypokalaemia, and metabolic alkalosis. Diagnosis: elevated plasma aldosterone-to-renin ratio (screening test); confirmed by salt loading test or fludrocortisone suppression test; adrenal CT and adrenal venous sampling to distinguish unilateral adenoma (surgical) from bilateral hyperplasia (medical treatment with mineralocorticoid receptor antagonists such as spironolactone).
5. Phaeochromocytoma presents with episodic hypertension (may be sustained), severe headache, palpitations, diaphoresis, pallor, and anxiety occurring in paroxysms. Diagnosis: elevated plasma free metanephrines (most sensitive test) or 24-hour urinary catecholamines and metanephrines; tumour localised by CT or MRI of the adrenals and abdomen; treatment is surgical after alpha-blockade (phenoxybenzamine) to prevent hypertensive crisis during resection.

Part 3.4

1. Dietary sodium restriction (below 6 g NaCl per day): reduces SBP by 2 to 8 mmHg. Weight reduction (10 kg): reduces SBP by 5 to 20 mmHg. Physical activity (30 to 45 min moderate aerobic exercise 5 days per week): reduces SBP by 4 to 9 mmHg. DASH diet: reduces SBP by 8 to 14 mmHg. Alcohol reduction: modest reduction in SBP. Smoking cessation: does not lower BP directly but dramatically reduces cardiovascular risk.
2. ACEi or ARBs: preferred in diabetes, CKD with proteinuria, heart failure; reduce angiotensin II. CCBs (amlodipine): preferred in elderly, black African patients, isolated systolic hypertension. Thiazide diuretics: preferred in black African patients, volume-dependent hypertension, elderly. Beta-blockers: preferred with concurrent IHD or heart failure; not first-line in uncomplicated hypertension. Mineralocorticoid receptor antagonists (spironolactone): used as fourth-line add-on therapy in resistant hypertension.

3. In Nigerian and African patients, hypertension is characteristically low-renin and volume-dependent; CCBs and thiazide diuretics, which act through calcium channel blockade and volume depletion respectively, are more effective as monotherapy than ACEi and ARBs, which primarily reduce angiotensin II-mediated vasoconstriction that is less prominent in this patient population.
4. Urgency: BP above 180/120 mmHg, no acute organ damage, patient may be asymptomatic or have headache; managed with oral antihypertensives (captopril, amlodipine, labetalol), gradual BP reduction over 24 to 48 hours, outpatient follow-up. Emergency: BP above 180/120 mmHg with acute organ damage (encephalopathy, aortic dissection, AMI, acute pulmonary oedema, AKI, eclampsia); immediate hospital admission required, intravenous antihypertensives.
5. Immediate HDU or ICU admission; continuous BP monitoring; IV labetalol (first-line in most emergencies), IV sodium nitroprusside (titrated drip; powerful but requires cyanide monitoring), IV hydralazine (preferred in eclampsia and pregnancy), IV glyceryl trinitrate (preferred in acute pulmonary oedema and acute coronary syndrome); BP reduction: no more than 25 percent in the first hour to prevent cerebral, coronary, or renal ischaemia from sudden pressure drop.

References and Attributions [Series 3]

Textual References

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- Kumar, P. and Clark, M. (2017). Kumar and Clark's Clinical Medicine (9th ed.). Edinburgh: Elsevier.

Figures, Plates, Tables, and Boxes

- Figure 3.1: Hypertension classification and causes. AI prompt: "Two-panel diagram: left panel BP classification table (Normal, Elevated, Stage 1, Stage 2, Stage 3, isolated systolic hypertension); right panel two boxes: Primary Hypertension (risk factors) and Secondary Hypertension (causes); clean professional infographic." OER search: "hypertension classification epidemiology causes primary secondary diagram".
- Figure 3.2: Mechanisms of primary hypertension and target organ damage. AI prompt: "Two-panel diagram: left panel five mechanism boxes feeding into Sustained Elevated BP; right panel four target organ boxes (Heart, Brain, Kidney, Eye); clean professional infographic." OER search: "hypertension pathophysiology mechanisms target organ damage diagram heart brain kidney eye".
- Table 3.1: Investigations in hypertension. AI prompt: "Two-section table: Section 1 Baseline Investigations; Section 2 Secondary Cause Tests; alternating shading." OER search: "hypertension investigations baseline secondary causes table aldosterone renin metanephrines".
- Table 3.2: Hypertensive urgency versus emergency. AI prompt: "Two-column comparison table: Urgency and Emergency; rows Definition, Features, Investigations, Management; alternating shading." OER search: "hypertensive urgency emergency comparison table management IV antihypertensives".

Series 4: Heart Failure — Mechanisms, Clinical Features, and Treatment

- **Part 4.1: Definition, Classification, and Epidemiology of Heart Failure**
  - Sub Part 4.1.1: Definition and types of heart failure
  - Sub Part 4.1.2: Classification and staging of heart failure
  - Sub Part 4.1.3: Epidemiology and aetiology of heart failure
- **Part 4.2: Pathophysiology of Heart Failure**
  - Sub Part 4.2.1: Cardiac remodelling and compensatory mechanisms
  - Sub Part 4.2.2: Neurohormonal activation in heart failure
  - Sub Part 4.2.3: Mechanisms of heart failure symptoms
- **Part 4.3: Clinical Features and Diagnosis of Heart Failure**
  - Sub Part 4.3.1: Symptoms and signs of heart failure
  - Sub Part 4.3.2: Investigations in heart failure
  - Sub Part 4.3.3: Diagnostic criteria
- **Part 4.4: Management of Heart Failure**
  - Sub Part 4.4.1: Non-pharmacological management
  - Sub Part 4.4.2: Pharmacological management of HFrEF
  - Sub Part 4.4.3: Acute decompensated heart failure

Introduction

Heart failure is a clinical syndrome in which the heart is unable to pump sufficient blood to meet the metabolic demands of the body, or can only do so at the cost of elevated filling pressures. It is a common, serious, and increasingly prevalent condition, affecting an estimated 64 million people worldwide and carrying a prognosis worse than many common cancers. In Nigeria and sub-Saharan Africa, heart failure presents characteristically in younger patients and is driven largely by hypertension, cardiomyopathies, and rheumatic heart disease.

This Series covers the definition, classification, and epidemiology of heart failure, followed by a detailed account of the pathophysiology including cardiac remodelling, compensatory mechanisms, and neurohormonal activation. It then examines the clinical features and diagnostic approach to heart failure, including the Framingham criteria, and concludes with comprehensive management including non-pharmacological measures, evidence-based pharmacotherapy for heart failure with reduced ejection fraction, and the acute management of decompensated heart failure.

Heart failure is one of the most important clinical syndromes in cardiology and medicine, and a thorough understanding of its mechanisms is essential for rational management.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 4.1** define heart failure, describe its types and classification, and outline its epidemiology and aetiology (Linked to Part 4.1),
- **SLO 4.2** describe the pathophysiology of heart failure including cardiac remodelling, compensatory mechanisms, and neurohormonal activation (Linked to Part 4.2),
- **SLO 4.3** describe the symptoms, signs, investigations, and diagnostic criteria for heart failure (Linked to Part 4.3),

- **SLO 4.4** describe the non-pharmacological and pharmacological management of heart failure with reduced ejection fraction (Linked to Part 4.4), and
- **SLO 4.5** describe the management of acute decompensated heart failure (Linked to Part 4.4).

4.1 Definition, Classification, and Epidemiology of Heart Failure

4.1.1 Definition and types of heart failure

Heart failure is defined as a clinical syndrome characterised by symptoms (dyspnoea, fatigue, ankle swelling) and signs (elevated JVP, pulmonary crepitations, peripheral oedema) resulting from structural or functional cardiac abnormality that causes reduced cardiac output or elevated intracardiac pressures at rest or with exertion.

Heart failure is classified by **ejection fraction (EF)** — the proportion of end-diastolic volume ejected with each beat (normal above 50 to 55 percent) — into three types: **HFrEF (heart failure with reduced EF)** (EF below 40 percent; systolic dysfunction predominates; the heart dilates and contracts poorly), **HFmrEF (mildly reduced EF)** (EF 40 to 49 percent), and **HFpEF (heart failure with preserved EF)** (EF at or above 50 percent; diastolic dysfunction predominates; the heart is stiff and fills poorly). HFrEF and HFpEF differ in aetiology, pathophysiology, and treatment.

Fill in the blank: Heart failure is classified by ejection fraction into HFrEF (EF below 40 percent; systolic dysfunction), HFmrEF (EF 40 to 49 percent), and HFpEF (EF at or above 50 percent; _____ dysfunction with impaired filling).

4.1.2 Classification and staging of heart failure

The **NYHA functional classification** (Class I to IV based on symptom severity, covered in Series 1) is used to assess symptoms. The **ACC/AHA staging system** provides a complementary framework emphasising disease progression: Stage A (at risk but no structural disease or

symptoms), Stage B (structural disease but no symptoms), Stage C (structural disease with current or prior symptoms), and Stage D (refractory heart failure requiring advanced therapies).

The ACC/AHA staging system is important because it emphasises that heart failure is a progressive disease that can be prevented or delayed at Stages A and B through risk factor modification (treating hypertension, diabetes, and coronary artery disease), before symptoms develop. Once symptomatic (Stage C), the goal shifts to symptom control, hospitalisation prevention, and mortality reduction. Stage D requires consideration of advanced therapies including ventricular assist devices and cardiac transplantation.

Fill in the blank: The ACC/AHA staging system classifies heart failure from Stage A (at risk, no disease) through Stage B (structural disease, no symptoms) and Stage C (symptoms present) to Stage D (refractory heart failure requiring _____ therapies).

4.1.3 Epidemiology and aetiology of heart failure

Globally, heart failure affects approximately 64 million people and carries a five-year mortality of approximately 50 percent, worse than many common cancers. In high-income countries, the most common causes are **ischaemic heart disease** (coronary artery disease causing myocardial infarction and ischaemic cardiomyopathy) and **hypertensive heart disease**. In Nigeria and sub-Saharan Africa, hypertensive heart disease is the most common cause, followed by **dilated cardiomyopathy** (often idiopathic or peripartum), **rheumatic heart disease**, and alcoholic cardiomyopathy.

Other aetiologies include **viral myocarditis** (coxsackievirus B is the most common cause), **thyroid disease** (both hypo- and hyperthyroidism), **anaemia** (high-output failure), **Chagas disease** (in Latin America; caused by Trypanosoma cruzi), and **tachycardia-induced cardiomyopathy** (reversible with rate control). Identifying the underlying cause is essential as some causes (thyroid disease, anaemia, tachycardia) are reversible.

Fill in the blank: Common causes of heart failure in Nigeria include hypertensive heart disease, dilated cardiomyopathy, rheumatic heart disease, and viral _____; identifying reversible causes (thyroid disease, anaemia, tachycardia) is essential.

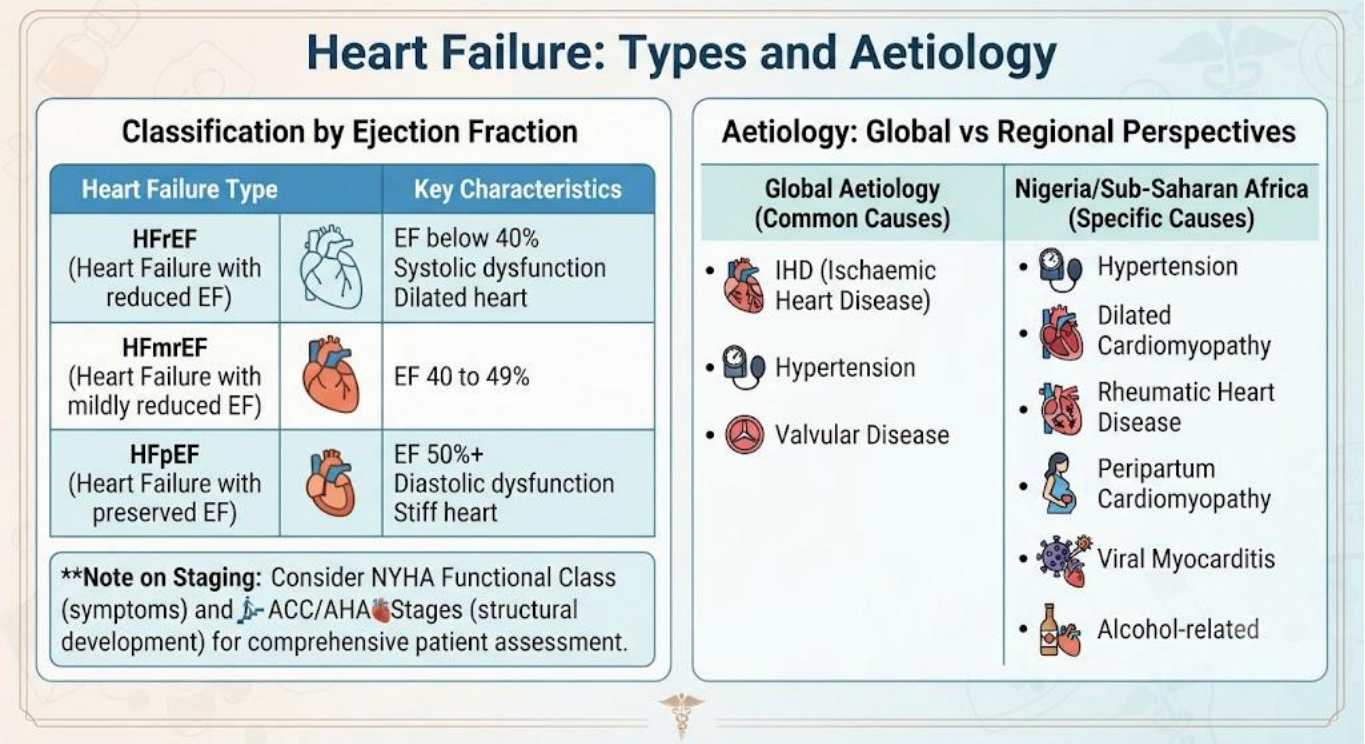

Figure 4.1: Heart failure classification by ejection fraction and the major aetiologies in Nigeria and globally

Pilot questions 4.1

1. Define heart failure and describe the three types classified by ejection fraction. (Linked to SLO 4.1)
2. Describe the ACC/AHA staging system for heart failure and explain its clinical importance. (Linked to SLO 4.1)
3. List the major causes of heart failure in Nigeria. (Linked to SLO 4.1)
4. Distinguish between HFrEF and HFpEF in terms of pathophysiology and typical patient profile. (Linked to SLO 4.1)
5. Explain why identifying reversible causes of heart failure is clinically important. (Linked to SLO 4.1)

4.2 Pathophysiology of Heart Failure

4.2.1 Cardiac remodelling and compensatory mechanisms

Cardiac remodelling refers to the changes in ventricular geometry, size, and function that occur in response to myocardial injury or chronic haemodynamic overload. In **pressure overload** (hypertension, aortic stenosis), the ventricle develops **concentric hypertrophy** (wall thickening without dilatation), which initially maintains wall stress and systolic function but eventually leads to diastolic dysfunction. In **volume overload** or after myocardial infarction, the ventricle undergoes **eccentric hypertrophy and dilatation**, with sarcomere replication in series, leading to systolic dysfunction.

Initial compensatory mechanisms maintain cardiac output in the face of impaired myocardial function: **increased preload** (Frank-Starling mechanism: a dilated ventricle generates greater force at higher end-diastolic volumes), **tachycardia** (sympathetic activation increases heart rate), and **peripheral vasoconstriction** (maintains blood pressure despite reduced cardiac output). These compensations are adaptive in the short term but become maladaptive when sustained, perpetuating the cycle of remodelling and dysfunction.

Fill in the blank: Pressure overload causes concentric hypertrophy; volume overload or MI causes eccentric hypertrophy and dilatation; compensatory mechanisms include increased preload (Frank-Starling), tachycardia, and peripheral _____.

4.2.2 Neurohormonal activation in heart failure

Reduced cardiac output activates neurohormonal systems that are initially compensatory but become progressively deleterious. The **sympathetic nervous system (SNS)** is activated, increasing heart rate, contractility, and peripheral vasoconstriction; chronic SNS activation causes myocardial toxicity, arrhythmias, and progressive LV dysfunction. The **renin-angiotensin-aldosterone system (RAAS)** is activated by reduced renal perfusion; angiotensin II causes vasoconstriction, promotes myocardial fibrosis and remodelling, and stimulates aldosterone-driven sodium retention and volume expansion.

Natriuretic peptides (BNP and NT-proBNP) are released by stretched ventricular myocardium and act as counter-regulatory hormones promoting natriuresis, vasodilation, and suppression of the RAAS and SNS. **Vasopressin (ADH)** is released by non-osmotic stimuli in heart failure, promoting water retention and hyponatraemia. **Endothelin-1** is a potent vasoconstrictor released by vascular endothelium in heart failure. The neurohormonal model of heart failure, which recognises that disease progression is driven by these activated systems rather than by reduced pump function alone, underlies the rationale for ACEi, ARBs, beta-blockers, and mineralocorticoid receptor antagonists as disease-modifying therapies.

Fill in the blank: Neurohormonal activation in heart failure involves the SNS (tachycardia, vasoconstriction, myocardial toxicity), the RAAS (vasoconstriction, sodium retention, fibrosis), natriuretic peptides (counter-regulatory), vasopressin (water retention), and _____-1 (potent vasoconstriction).

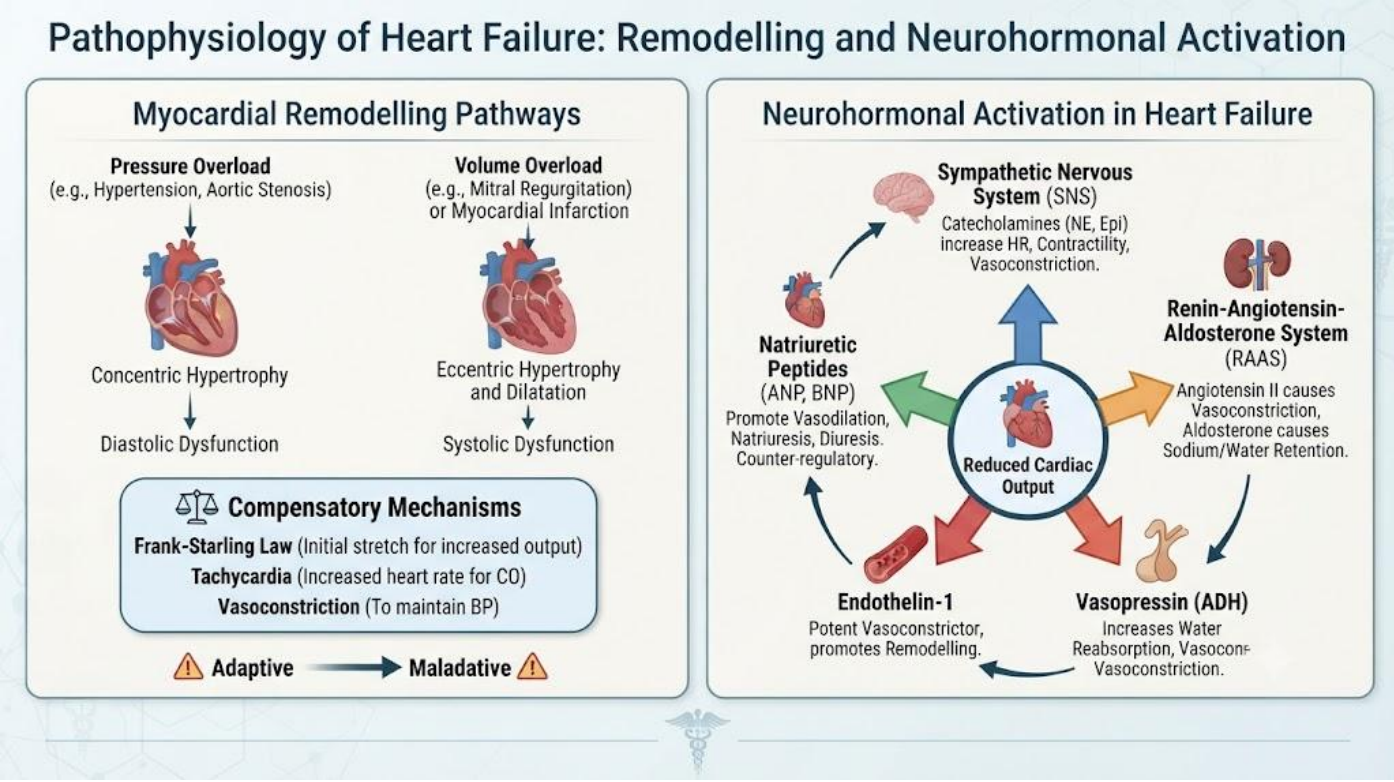

Figure 4.2: Cardiac remodelling, compensatory mechanisms, and neurohormonal activation in heart failure

4.2.3 Mechanisms of heart failure symptoms

Dyspnoea and orthopnoea result from elevated left heart filling pressures causing pulmonary venous congestion and increased work of breathing, as described in Series 1. In advanced heart failure, **cardiac cachexia** (severe involuntary weight loss with muscle wasting) develops from reduced gut perfusion, chronic inflammation, and neuroendocrine activation, and carries a very poor prognosis.

Fatigue and exercise intolerance result from reduced cardiac output causing skeletal muscle hypoperfusion and, in advanced disease, from skeletal muscle atrophy and mitochondrial dysfunction. **Peripheral oedema** results from elevated right heart filling pressures causing sodium and water retention and transudation of fluid into the interstitium, as described in Series 1. **Nocturia** is an early symptom: the supine position at night increases renal perfusion and sodium excretion relative to the daytime upright state, increasing urine production.

Fill in the blank: Dyspnoea in heart failure results from pulmonary venous congestion; fatigue from skeletal muscle _____; oedema from right heart failure and sodium retention; nocturia from increased renal perfusion in the supine position.

Pilot questions 4.2

1. Distinguish between concentric and eccentric ventricular hypertrophy and state the conditions that cause each. (Linked to SLO 4.2)
2. Explain the Frank-Starling mechanism and describe how it compensates for reduced cardiac output in heart failure. (Linked to SLO 4.2)
3. Describe the activation of the sympathetic nervous system in heart failure and explain why chronic SNS activation is harmful. (Linked to SLO 4.2)
4. Explain the role of the RAAS in heart failure and why RAAS blockade is a cornerstone of treatment. (Linked to SLO 4.2)

5. Explain the clinical significance of BNP and NT-proBNP in heart failure. (Linked to SLO 4.2)

4.3 Clinical Features and Diagnosis of Heart Failure

4.3.1 Symptoms and signs of heart failure

The classic symptoms of heart failure are dyspnoea (on exertion initially, then at rest), orthopnoea, paroxysmal nocturnal dyspnoea, fatigue, reduced exercise tolerance, ankle swelling, and nocturia. Additional symptoms include abdominal distension (ascites in severe right heart failure), anorexia (gut congestion), and palpitations.

Physical signs in left heart failure include **tachycardia**, **low blood pressure or narrow pulse pressure** in severe cases, **displaced apex beat** (cardiac enlargement), **third heart sound (S3 gallop)** (ventricular volume overload), and **bilateral basal crepitations** (pulmonary oedema). Signs of right heart failure include **elevated JVP**, **hepatomegaly** (congestive hepatopathy), **peripheral oedema** (bilateral, pitting, dependent), and **ascites**. Cardiac cachexia and pleural effusions (usually bilateral) are features of advanced biventricular failure.

Fill in the blank: Left heart failure signs include tachycardia, displaced apex, S3 gallop, and basal crepitations; right heart failure signs include elevated JVP, _____, peripheral oedema, and ascites.

4.3.2 Investigations in heart failure

Natriuretic peptides are the most valuable biomarkers for diagnosis: **BNP above 35 pg/mL or NT-proBNP above 125 pg/mL** in a patient with dyspnoea makes cardiac heart failure highly likely (high sensitivity); normal levels make heart failure very unlikely and suggest an alternative cause of dyspnoea. **Echocardiography** is the key investigation, confirming the diagnosis, measuring EF (to classify HFrEF versus HFpEF), assessing wall motion, valve function, and filling pressures.

12-lead ECG is rarely normal in heart failure: findings include LVH, left bundle branch block (LBBB, which may itself cause HFrEF: tachycardia-induced or dyssynchrony-mediated), atrial fibrillation (common in heart failure), and ischaemic changes. **Chest X-ray** may show cardiomegaly (cardiothoracic ratio above 0.5), upper lobe blood diversion, Kerley B lines (interstitial oedema), and bilateral perihilar infiltrates ('bat-wing' distribution of pulmonary oedema). Blood tests include **full blood count** (anaemia), **renal function and electrolytes**, **liver function** (congestive hepatopathy), **thyroid function** (hypothyroidism as cause or contributor), and **fasting glucose and lipids** (risk factors).

Fill in the blank: BNP above 35 or NT-proBNP above 125 pg/mL supports heart failure diagnosis; echocardiography confirms diagnosis and measures EF; chest X-ray may show cardiomegaly, upper lobe diversion, Kerley B lines, and perihilar _____ ('bat-wing' pulmonary oedema).

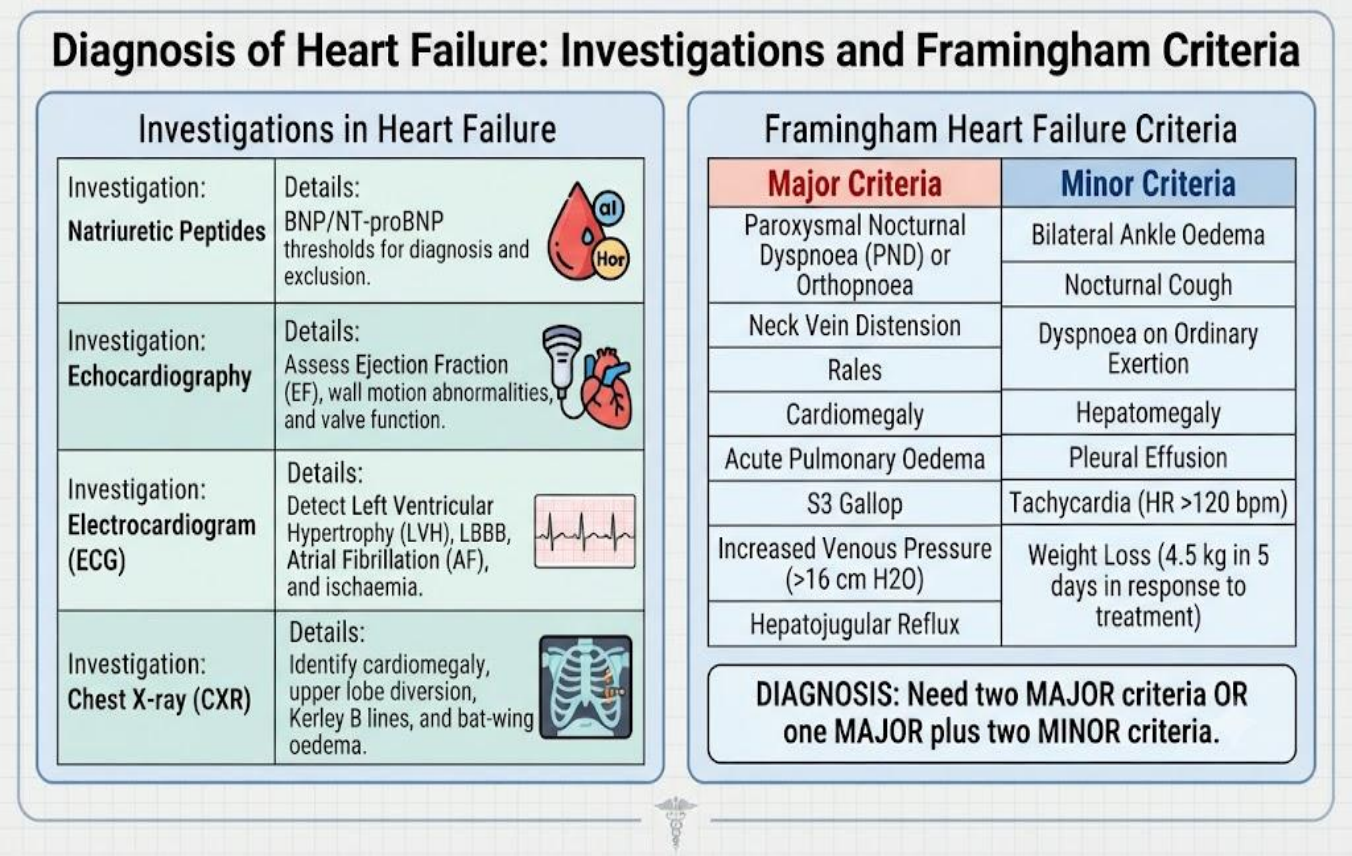

Figure 4.3: Key investigations and the Framingham criteria for the diagnosis of heart failure

4.3.3 Diagnostic criteria

The **Framingham criteria** remain the most widely used clinical diagnostic tool for heart failure, comprising major and minor criteria.

Major criteria include: paroxysmal nocturnal dyspnoea, orthopnoea, elevated JVP, pulmonary rales (crepitations), S3 gallop, radiographic cardiomegaly, acute pulmonary oedema, weight loss of more than 4.5 kg in 5 days in response to diuretic therapy, and hepatojugular reflux.

Minor criteria include: bilateral ankle oedema, night cough, dyspnoea on ordinary exertion, hepatomegaly, pleural effusion, reduction in vital capacity by one third of maximum, and tachycardia above 120 per minute.

Diagnosis requires **two major criteria** or one major plus two minor criteria, provided the minor criteria cannot be attributed to another condition. In contemporary practice, the Framingham criteria are used alongside echocardiography (which confirms the diagnosis and classifies the type) and natriuretic peptide levels.

Fill in the blank: The Framingham criteria diagnose heart failure by two major criteria or one major plus _____ minor criteria; contemporary diagnosis integrates Framingham criteria with echocardiography and natriuretic peptide levels.

Pilot questions 4.3

1. List the major symptoms and physical signs of left and right heart failure. (Linked to SLO 4.3)
2. Explain the role of BNP and NT-proBNP in the diagnosis of heart failure. (Linked to SLO 4.3)
3. Describe the echocardiographic findings expected in a patient with HFrEF. (Linked to SLO 4.3)
4. Describe the chest X-ray findings in acute pulmonary oedema. (Linked to SLO 4.3)

5. State the Framingham criteria for diagnosing heart failure. (Linked to SLO 4.3)

4.4 Management of Heart Failure

4.4.1 Non-pharmacological management

Non-pharmacological management is an essential component of heart failure care for all patients regardless of pharmacological therapy. Key measures include **fluid restriction** (1.5 to 2 litres per day in advanced heart failure with hyponatraemia), **daily weight monitoring** (an increase of more than 2 kg in 2 days indicates fluid retention and should prompt urgent medical review or diuretic adjustment), and **dietary sodium restriction** (below 2 g sodium per day to reduce fluid retention).

Exercise training (supervised cardiac rehabilitation) improves exercise capacity, quality of life, and reduces hospitalisation in stable HFrEF patients. **Alcohol abstinence** is mandatory in alcoholic cardiomyopathy and strongly advised in all heart failure patients. **Smoking cessation** reduces cardiovascular risk. Vaccination against influenza and pneumococcus prevents infections that can precipitate decompensation. Patients should be educated about symptoms of decompensation and the importance of **medication adherence**.

Fill in the blank: Non-pharmacological heart failure management includes fluid restriction, daily weight monitoring (act on more than 2 kg gain in 2 days), dietary sodium restriction, exercise training, alcohol _____, and medication adherence education.

4.4.2 Pharmacological management of HFrEF

Four drug classes have proven mortality benefit in HFrEF and are recommended as cornerstone therapy: **ACE inhibitors or ARBs** (reduce angiotensin II, decrease afterload and preload, attenuate remodelling; lisinopril, ramipril, enalapril; ARBs if ACEi not tolerated due to cough), **beta-blockers** (carvedilol, bisoprolol, metoprolol succinate; block SNS activation, reduce heart rate and arrhythmia risk, attenuate remodelling; start low, titrate slowly), **mineralocorticoid receptor antagonists (MRAs)** (spironolactone, eplerenone; block

aldosterone effects, reduce fibrosis and remodelling, reduce mortality), and **SGLT2 inhibitors** (dapagliflozin, empagliflozin; recently proven to reduce HF hospitalisation and cardiovascular death independently of diabetes).

Diuretics (loop diuretics: furosemide, bumetanide) are the mainstay of symptomatic treatment, relieving congestion rapidly but without proven mortality benefit. **Ivabradine** reduces heart rate by blocking the If channel in the sinus node; indicated when resting heart rate is above 70 bpm despite maximum tolerated beta-blocker dose. **Sacubitril/valsartan (ARNI)** combines neprilysin inhibition (enhancing natriuretic peptide effects) with ARB; superior to ACEi alone in reducing HF mortality and is the preferred agent over ACEi in patients who can tolerate it.

Fill in the blank: Cornerstone HFrEF therapies with mortality benefit include ACEi or ARBs, beta-blockers, MRAs (spironolactone), and SGLT2 inhibitors; sacubitril/valsartan (_____ inhibitor plus ARB) is preferred over ACEi where tolerated.

4.4.3 Acute decompensated heart failure

Acute decompensated heart failure (ADHF) is a life-threatening emergency characterised by acute or worsening symptoms of heart failure requiring urgent hospitalisation. Precipitants include poor medication adherence, dietary sodium excess, infection, arrhythmia (new-onset atrial fibrillation), ischaemia, and anaemia.

Initial management follows the **LMNOP mnemonic**: **L** (loop diuretics: IV furosemide to reduce congestion rapidly), **M** (morphine: reduces anxiety and dyspnoea; use with caution as it can cause respiratory depression), **N** (nitrates: IV glyceryl trinitrate for vasodilation, reducing preload and afterload; contraindicated in hypotension), **O** (oxygen: target saturation above 94 percent; avoid in patients with COPD), and **P** (positioning: sit the patient upright to reduce venous return and pulmonary congestion). **Non-invasive ventilation** (CPAP or BiPAP) is indicated for respiratory failure not responding to medical therapy.

Fill in the blank: ADHF management follows LMNOP: Loop diuretics (IV furosemide), Morphine (cautious use), Nitrates (vasodilation; avoid in hypotension), Oxygen (target above 94 percent), and _____ (sit upright to reduce venous return).

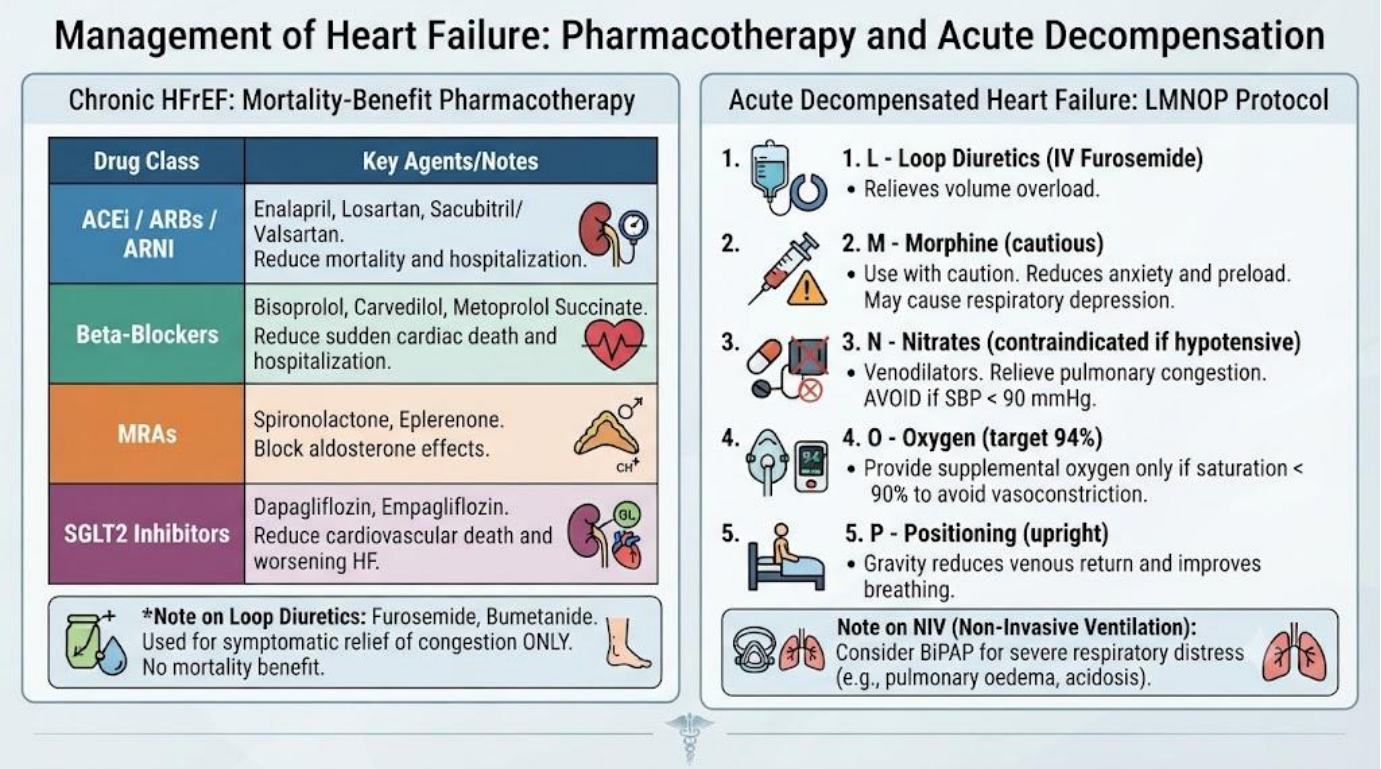

Figure 4.4: HFrEF pharmacotherapy with mortality benefit and the LMNOP protocol for acute decompensated heart failure

Pilot questions 4.4

1. Describe the non-pharmacological management of heart failure. (Linked to SLO 4.4)
2. Describe the four drug classes with proven mortality benefit in HFrEF and explain the rationale for each. (Linked to SLO 4.4)
3. Explain the role of diuretics in heart failure management. (Linked to SLO 4.4)
4. Describe the precipitants of acute decompensated heart failure. (Linked to SLO 4.5)
5. Describe the LMNOP mnemonic for the initial management of acute decompensated heart failure. (Linked to SLO 4.5)

Series Summary

This Series covered heart failure comprehensively. Heart failure is a syndrome of symptoms and signs from cardiac dysfunction; it is classified by EF into HFrEF (below 40 percent, systolic dysfunction), HFmrEF (40 to 49 percent), and HFpEF (50 percent and above, diastolic dysfunction). The ACC/AHA staging system progresses from Stage A (at risk) through B (structural disease, no symptoms) and C (symptomatic) to D (refractory). In Nigeria, hypertensive heart disease is the most common cause, followed by dilated cardiomyopathy and rheumatic heart disease. Pressure overload causes concentric hypertrophy; volume overload or MI causes eccentric hypertrophy and dilatation. Compensatory mechanisms (Frank-Starling, tachycardia, vasoconstriction) are initially adaptive but become maladaptive. Neurohormonal activation (SNS, RAAS, vasopressin, endothelin) drives disease progression and is the target of disease-modifying pharmacotherapy.

Symptoms include dyspnoea, orthopnoea, PND, fatigue, ankle oedema, and nocturia. Left heart failure signs include tachycardia, displaced apex, S3 gallop, and basal crepitations; right heart failure adds elevated JVP, hepatomegaly, oedema, and ascites. Diagnosis is supported by BNP above 35 or NT-proBNP above 125 pg/mL; confirmed by echocardiography (EF, wall motion, valves); ECG shows LVH, LBBB, or AF; CXR shows cardiomegaly, upper lobe diversion, Kerley B lines, and perihilar bat-wing oedema. Framingham criteria require two major or one major plus two minor criteria. Non-pharmacological management includes fluid restriction, daily weights, sodium restriction, exercise, and adherence. HFrEF pharmacotherapy: ACEi or ARBs (or sacubitril/valsartan preferred), beta-blockers, MRAs, and SGLT2 inhibitors all reduce mortality; diuretics relieve symptoms; ivabradine reduces heart rate when needed. ADHF: LMNOP (loop diuretics, morphine cautiously, nitrates, oxygen, positioning upright); non-invasive ventilation for refractory respiratory failure.

Glossary of Terms

- **HFrEF:** heart failure with reduced ejection fraction (below 40 percent); characterised by systolic dysfunction, ventricular dilatation, and impaired contractility.

- **HFpEF:** heart failure with preserved ejection fraction (50 percent and above); characterised by diastolic dysfunction and impaired ventricular filling.
- **Ejection fraction (EF):** the proportion of end-diastolic volume ejected with each heartbeat; normal above 50 to 55 percent.
- **Cardiac remodelling:** changes in ventricular geometry, size, and function in response to myocardial injury or chronic haemodynamic overload.
- **Frank-Starling mechanism:** the physiological principle that increased ventricular end-diastolic volume (preload) generates greater contractile force; a key compensatory mechanism in heart failure.
- **BNP/NT-proBNP:** natriuretic peptides released by stretched ventricular myocardium; elevated levels support the diagnosis of heart failure and correlate with disease severity.
- **Framingham criteria:** clinical diagnostic criteria for heart failure comprising major and minor criteria; diagnosis requires two major or one major plus two minor criteria.
- **ACE inhibitor:** angiotensin-converting enzyme inhibitor; blocks the conversion of angiotensin I to angiotensin II; reduces afterload, preload, and neurohormonal activation in heart failure.
- **Sacubitril/valsartan (ARNI):** an angiotensin receptor-neprilysin inhibitor combining neprilysin inhibition (enhancing natriuretic peptide effects) with ARB; superior mortality benefit compared to ACEi in HFrEF.
- **LMNOP:** the mnemonic for acute decompensated heart failure management: Loop diuretics, Morphine, Nitrates, Oxygen, Positioning.
- **Kerley B lines:** horizontal lines at the lung bases on chest X-ray indicating interstitial pulmonary oedema from elevated left heart filling pressures.
- **Cardiac cachexia:** severe involuntary weight loss with muscle wasting in advanced heart failure, resulting from reduced gut perfusion, chronic inflammation, and neuroendocrine activation.

Concept Check Questions [Series 4]

Objective Questions

1. HFrEF is defined as heart failure with EF below 40 percent; HFpEF is defined as EF at or above _____ percent. (Linked to SLO 4.1)
2. In the ACC/AHA staging system, Stage _____ represents structural heart disease without current or prior symptoms. (Linked to SLO 4.1)
3. The Frank-Starling mechanism compensates for reduced cardiac output by increasing contractile force in response to increased ventricular _____. (Linked to SLO 4.2)
4. BNP above 35 or NT-proBNP above 125 pg/mL supports a diagnosis of heart failure; normal levels make heart failure _____. (Linked to SLO 4.3)
5. Four drug classes with proven mortality benefit in HFrEF are ACEi or ARBs, beta-blockers, MRAs (spironolactone), and _____ inhibitors. (Linked to SLO 4.4)
6. ADHF management follows LMNOP: Loop diuretics, Morphine, Nitrates, Oxygen, and _____ (sit patient upright). (Linked to SLO 4.5)

Short-Answer Questions

7. Describe the four neurohormonal systems activated in heart failure and the consequence of each. (Linked to SLO 4.2)
8. State the Framingham major and minor criteria for the diagnosis of heart failure. (Linked to SLO 4.3)
9. Describe the chest X-ray findings in pulmonary oedema. (Linked to SLO 4.3)

Long-Answer Questions

10. Describe the pathophysiology of heart failure including cardiac remodelling, compensatory mechanisms, and neurohormonal activation. (Linked to SLO 4.2)
11. Describe the pharmacological management of HFrEF, including drug classes, mechanisms, and clinical evidence. (Linked to SLO 4.4)

Answers to Concept Check Questions [Series 4]

Objective Questions

1. 50.
2. B.
3. Preload (end-diastolic volume).
4. Very unlikely.
5. SGLT2.
6. Positioning.

Short-Answer Questions

7. SNS: tachycardia, vasoconstriction, contractility (initially adaptive; chronically causes myocardial toxicity, arrhythmias, and progressive LV dysfunction). RAAS: angiotensin II vasoconstriction and aldosterone sodium retention (fluid overload, fibrosis, remodelling). Natriuretic peptides (BNP, NT-proBNP): counter-regulatory (natriuresis, vasodilation, RAAS suppression). Vasopressin: water retention, hyponatraemia.
8. Major criteria: PND, orthopnoea, elevated JVP, pulmonary rales, S3 gallop, cardiomegaly on CXR, acute pulmonary oedema, weight loss above 4.5 kg in 5 days on diuretics, hepatojugular reflux. Minor criteria: bilateral ankle oedema, night cough, dyspnoea on ordinary exertion, hepatomegaly, pleural effusion, vital capacity reduced by one third, tachycardia above 120 per minute. Diagnosis: two major or one major plus two minor.
9. Cardiomegaly (cardiothoracic ratio above 0.5), upper lobe blood diversion, Kerley B lines (horizontal interstitial lines at lung bases), bilateral perihilar infiltrates in a bat-wing distribution indicating alveolar oedema, and pleural effusions (usually bilateral).

Long-Answer Questions

10. **Basic response:** Cardiac remodelling: pressure overload (hypertension, AS) causes concentric hypertrophy (wall thickening, maintained EF initially, diastolic dysfunction); volume overload or MI causes eccentric hypertrophy and dilatation (sarcomeres in series, systolic dysfunction, reduced EF). Compensatory mechanisms: Frank-Starling

(increased preload generates greater force), tachycardia (SNS activation increases HR), peripheral vasoconstriction (maintains BP); adaptive short-term, maladaptive when sustained. Neurohormonal activation: SNS (tachycardia, vasoconstriction, myocardial toxicity, arrhythmias); RAAS (angiotensin II vasoconstriction, aldosterone sodium retention and fibrosis, volume overload); natriuretic peptides (BNP, NT-proBNP: counter-regulatory, natriuresis, vasodilation); vasopressin (water retention, hyponatraemia); endothelin-1 (potent vasoconstriction). The neurohormonal model explains why blocking these systems (ACEi, beta-blockers, MRAs) reduces mortality.

Value-added response: Understanding the neurohormonal model is not merely academic: it directly explains why diuretics, which relieve symptoms but do not block the activated systems driving disease progression, must always be combined with ACEi, beta-blockers, and MRAs in HFrEF to address both the symptoms and the underlying mechanism of deterioration.

11. **Basic response:** ACEi or ARBs: block RAAS, reduce angiotensin II vasoconstriction and aldosterone, decrease preload and afterload, attenuate remodelling; proven to reduce mortality and hospitalisation. Sacubitril/valsartan (ARNI): neprilysin inhibition enhances natriuretic peptides combined with ARB; superior to ACEi in reducing HF mortality (PARADIGM-HF trial); preferred over ACEi where tolerated. Beta-blockers (carvedilol, bisoprolol, metoprolol succinate): block SNS activation, reduce HR, attenuate remodelling, anti-arrhythmic; must be started at low dose and titrated slowly in stable patients; proven mortality benefit. MRAs (spironolactone, eplerenone): block aldosterone, reduce fibrosis and remodelling, reduce hypokalaemia, reduce mortality. SGLT2 inhibitors (dapagliflozin, empagliflozin): reduce heart failure hospitalisation and cardiovascular death; mechanism includes osmotic diuresis, reduced cardiac fibrosis, and energy substrate effects. Diuretics (furosemide): relieve congestion rapidly; essential for symptoms; no mortality benefit. Ivabradine: reduces HR via If channel blockade; indicated when HR above 70 bpm on maximum beta-blocker.

    Value-added response: The combination of an ARNI, beta-blocker, MRA, and SGLT2 inhibitor represents current optimal pharmacotherapy for HFrEF; clinical trials have

shown that implementing this combination in all eligible patients could prevent tens of thousands of premature deaths annually, making adherence to guideline-directed medical therapy the most impactful intervention available to the clinician managing chronic HFrEF.

Answers to Pilot Questions [Series 4]

Part 4.1

1. Heart failure is a clinical syndrome of symptoms (dyspnoea, fatigue, ankle swelling) and signs (elevated JVP, crepitations, oedema) from cardiac dysfunction causing reduced output or elevated filling pressures. HFrEF: EF below 40 percent, systolic dysfunction, dilated poorly contracting ventricle. HFmrEF: EF 40 to 49 percent. HFpEF: EF at or above 50 percent, diastolic dysfunction, stiff poorly filling ventricle.
2. Stage A: at risk (hypertension, diabetes, obesity) but no structural disease or symptoms; management: risk factor modification. Stage B: structural disease (LVH, previous MI) but no symptoms; management: ACEi and beta-blockers. Stage C: structural disease with current or prior symptoms; management: full guideline-directed medical therapy. Stage D: refractory heart failure; management: advanced therapies (ventricular assist device, cardiac transplantation).
3. Hypertensive heart disease (most common in Nigeria), dilated cardiomyopathy (often idiopathic or peripartum), rheumatic heart disease, viral myocarditis (coxsackievirus B), alcoholic cardiomyopathy, and anaemia causing high-output heart failure.
4. HFrEF: EF below 40 percent, systolic dysfunction, dilated poorly contracting ventricle; typical patient: male, younger, ischaemic or dilated cardiomyopathy; responds to ACEi, beta-blockers, MRAs, SGLT2 inhibitors. HFpEF: EF above 50 percent, diastolic dysfunction, stiff non-compliant ventricle with impaired filling; typical patient: elderly woman with hypertension, diabetes, or obesity; no drug class has clearly proven mortality benefit.

5. Reversible causes include thyroid disease (hypothyroidism or thyrotoxicosis), anaemia (causing high-output failure), tachycardia-induced cardiomyopathy (resolves with rate control), alcohol (alcoholic cardiomyopathy may improve with abstinence), and peripartum cardiomyopathy (may recover with appropriate treatment); identifying these avoids lifelong therapy and may result in complete functional recovery.

Part 4.2

1. Concentric hypertrophy: pressure overload (hypertension, aortic stenosis) causes sarcomere replication in parallel, thickening the LV wall without dilatation; initially maintains wall stress and systolic function, leads to diastolic dysfunction. Eccentric hypertrophy and dilatation: volume overload or myocardial infarction causes sarcomere replication in series, chamber dilatation, and thinning; leads to systolic dysfunction and reduced EF.
2. The Frank-Starling mechanism states that increased ventricular end-diastolic volume (preload) stretches the sarcomeres, increasing their sensitivity to calcium and generating greater contractile force, thus increasing stroke volume. In heart failure, this compensates for impaired contractility by using higher filling pressures to maintain output; however, at very high filling pressures the relationship flattens and excess volume causes pulmonary congestion without further benefit.
3. Reduced cardiac output triggers SNS activation, increasing heart rate and contractility (adaptive initially); chronically, SNS activation causes myocardial catecholamine toxicity (direct myocardial injury), promotes arrhythmias (atrial fibrillation, ventricular tachycardia), upregulates beta-receptors desensitisation, and perpetuates remodelling; beta-blockers block these harmful effects and reduce mortality.
4. Reduced renal perfusion in heart failure activates the RAAS: renin release generates angiotensin II (causing arteriolar vasoconstriction increasing afterload and stimulating aldosterone), and aldosterone drives sodium and water reabsorption (expanding volume and increasing preload); angiotensin II and aldosterone also promote cardiac fibrosis and remodelling. ACEi and ARBs block angiotensin II; MRAs block aldosterone; both reduce mortality by interrupting this cycle.

5. BNP and NT-proBNP are natriuretic peptides released by stretched ventricular myocardium in response to elevated filling pressures; they diagnose heart failure with high sensitivity (normal levels make heart failure very unlikely; elevated levels support the diagnosis); they also correlate with disease severity and prognosis (higher levels predict worse outcomes); they are used to monitor response to treatment (levels fall with effective decongestion).

Part 4.3

1. Symptoms: dyspnoea on exertion, orthopnoea, PND, fatigue, ankle swelling, nocturia, anorexia. Left heart failure signs: tachycardia, low BP or narrow pulse pressure (severe), displaced apex beat, S3 gallop, bilateral basal crepitations, possible wheeze (cardiac asthma). Right heart failure signs: elevated JVP, hepatomegaly, bilateral pitting peripheral oedema, ascites.
2. BNP and NT-proBNP are released by stretched ventricular myocardium; BNP above 35 pg/mL or NT-proBNP above 125 pg/mL in a patient with dyspnoea makes heart failure the likely cause; levels below these thresholds make heart failure very unlikely and suggest an alternative diagnosis (respiratory disease, anaemia); elevated levels also correlate with severity and prognosis.
3. Echocardiographic findings in HFrEF: reduced EF (below 40 percent), globally dilated LV with impaired contractility (reduced fractional shortening), segmental wall motion abnormalities in ischaemic disease, dilated left atrium (from chronically elevated filling pressures), elevated estimated LV filling pressures (raised E/e' ratio on tissue Doppler), possible functional mitral regurgitation (dilated annulus), and if RV also affected, RV dilatation and reduced function.
4. CXR findings in acute pulmonary oedema: cardiomegaly (cardiothoracic ratio above 0.5), upper lobe blood diversion (vessels in upper zones are larger than lower zone vessels, indicating elevated left heart pressures), Kerley B lines (short horizontal lines at the lung bases: interstitial oedema in lymphatics), bilateral perihilar infiltrates in a bat-wing or butterfly distribution (alveolar oedema), pleural effusions (usually bilateral).

5. Major criteria: PND, neck vein distension (elevated JVP), pulmonary rales (crepitations), radiographic cardiomegaly, acute pulmonary oedema, S3 gallop, increased venous pressure above 16 cmH2O, weight loss above 4.5 kg in 5 days in response to treatment, hepatojugular reflux. Minor criteria: bilateral ankle oedema, night cough, dyspnoea on ordinary exertion, hepatomegaly, pleural effusion, vital capacity reduced by one third, tachycardia above 120 per minute.

Part 4.4

1. Fluid restriction (1.5 to 2 litres per day in advanced HF with hyponatraemia), daily weight monitoring (weight gain above 2 kg in 2 days requires urgent review or diuretic adjustment), dietary sodium restriction (below 2 g per day), supervised exercise training (improves exercise capacity and quality of life), alcohol abstinence (especially in alcoholic cardiomyopathy), smoking cessation, vaccination (influenza and pneumococcal), medication adherence education.
2. ACEi or ARBs: block RAAS, reduce afterload and preload, attenuate remodelling, reduce mortality and hospitalisation. Beta-blockers (carvedilol, bisoprolol, metoprolol succinate): block SNS, reduce HR and arrhythmia risk, reduce remodelling, proven mortality benefit; start low and titrate slowly. MRAs (spironolactone, eplerenone): block aldosterone, reduce fibrosis and remodelling, prevent hypokalaemia, reduce mortality. SGLT2 inhibitors (dapagliflozin, empagliflozin): reduce HF hospitalisation and cardiovascular death through multiple mechanisms.
3. Loop diuretics (furosemide, bumetanide) are the mainstay of symptomatic congestion management; they rapidly reduce preload by promoting sodium and water excretion, relieving dyspnoea, oedema, and pulmonary congestion; however, they have no proven mortality benefit and over-diuresis causes renal impairment and electrolyte disturbance; they must be combined with disease-modifying agents (ACEi, beta-blockers, MRAs).
4. Common precipitants: poor medication adherence (the most common), dietary sodium and fluid excess, infections (particularly respiratory), new-onset arrhythmias (atrial fibrillation most common), acute myocardial ischaemia or infarction, anaemia,

uncontrolled hypertension, and drugs that worsen heart failure (NSAIDs, verapamil, diltiazem, negative inotropes).

5. L: Loop diuretics (IV furosemide 40 to 80 mg IV to relieve congestion rapidly). M: Morphine (2 to 4 mg IV; reduces anxiety and sympathetic activation; reduces preload and dyspnoea; use cautiously due to risk of respiratory depression and hypotension). N: Nitrates (IV glyceryl trinitrate; reduces preload and afterload; contraindicated in hypotension below 90 mmHg systolic). O: Oxygen (target saturation above 94 percent; high-flow oxygen if hypoxic; avoid routine high-flow O2 in normoxic patients). P: Positioning (sit upright; reduces venous return and pulmonary venous congestion).

References and Attributions [Series 4]

Textual References

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- Kumar, P. and Clark, M. (2017). Kumar and Clark's Clinical Medicine (9th ed.). Edinburgh: Elsevier.

Figures, Plates, Tables, and Boxes

- Figure 4.1: Heart failure classification and aetiology. AI prompt: "Two-panel diagram: left panel three-row EF classification table (HFrEF, HFmrEF, HFpEF) with notes on NYHA and ACC/AHA staging; right panel two-column aetiology (Global and Nigeria/SSA); clean professional infographic." OER search: "heart failure classification ejection fraction aetiology causes Nigeria diagram".
- Figure 4.2: Cardiac remodelling and neurohormonal activation. AI prompt: "Two-panel diagram: left panel remodelling (concentric vs eccentric) plus compensatory mechanisms; right panel circular neurohormonal diagram (SNS, RAAS, vasopressin, endothelin, natriuretic peptides); clean professional infographic." OER search: "heart failure pathophysiology remodelling neurohormonal activation RAAS SNS diagram".
- Figure 4.3: Investigations and Framingham criteria. AI prompt: "Two-panel diagram: left panel four-row investigation table (BNP/NT-proBNP, echo, ECG, CXR); right panel Framingham major and minor criteria; clean professional infographic." OER search: "heart failure diagnosis investigations BNP echocardiography Framingham criteria diagram".
- Figure 4.4: HFrEF pharmacotherapy and LMNOP. AI prompt: "Two-panel diagram: left panel four mortality-benefit drug classes; right panel five-step LMNOP protocol; clean

professional infographic." OER search: "heart failure management HFrEF pharmacotherapy LMNOP acute decompensated diagram".

Series 5: Introduction to Electrocardiography (ECG)

- **Part 5.1: Principles and Technical Basis of the ECG**
  - Sub Part 5.1.1: Electrical basis of the ECG
  - Sub Part 5.1.2: ECG leads and their placement
  - Sub Part 5.1.3: The normal ECG waveform
- **Part 5.2: Systematic ECG Interpretation**
  - Sub Part 5.2.1: Rate and rhythm assessment
  - Sub Part 5.2.2: Axis determination
  - Sub Part 5.2.3: Intervals, waveform morphology, and ST-T changes
- **Part 5.3: ECG Patterns in Common Cardiac Conditions**
  - Sub Part 5.3.1: ECG in ischaemia and myocardial infarction
  - Sub Part 5.3.2: ECG in chamber enlargement and bundle branch block
  - Sub Part 5.3.3: ECG in arrhythmias
- **Part 5.4: Clinical Application of the ECG**
  - Sub Part 5.4.1: ECG in clinical decision-making
  - Sub Part 5.4.2: Limitations of the ECG
  - Sub Part 5.4.3: ECG as part of the cardiovascular investigation package

Introduction

The electrocardiogram (ECG) is the single most important and widely used investigation in cardiology. It is non-invasive, inexpensive, rapidly obtained, and provides immediate information about cardiac rhythm, conduction, ischaemia, infarction, chamber size, and electrolyte disturbances. Every clinical student must be able to record, systematically interpret, and clinically apply an ECG, as its findings frequently determine urgent management decisions.

This Series introduces the electrical basis of the ECG, the standard lead system and electrode placement, and the morphology of the normal ECG waveform. It then provides a systematic approach to ECG interpretation covering rate, rhythm, axis, intervals, and waveform morphology, before examining the ECG patterns associated with myocardial ischaemia and infarction, chamber enlargement, bundle branch block, and common arrhythmias. The Series concludes with the practical application of the ECG in clinical decision-making and an understanding of its limitations.

ECG interpretation is a skill acquired through practice; the systematic approach taught in this Series, applied consistently to every ECG encountered, is the foundation upon which clinical competence is built.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 5.1** describe the electrical basis of the ECG, the standard lead system, and the components of the normal ECG waveform (Linked to Part 5.1),
- **SLO 5.2** apply a systematic approach to ECG interpretation covering rate, rhythm, axis, intervals, and waveform morphology (Linked to Part 5.2),
- **SLO 5.3** identify ECG patterns associated with myocardial ischaemia and infarction, chamber enlargement, bundle branch block, and common arrhythmias (Linked to Part 5.3), and

- **SLO 5.4** explain the clinical application and limitations of the ECG in cardiovascular diagnosis and management (Linked to Part 5.4).

5.1 Principles and Technical Basis of the ECG

5.1.1 Electrical basis of the ECG

The ECG records the electrical activity of the heart from the body surface. Cardiac muscle cells are electrically polarised at rest (resting membrane potential approximately minus 90 mV). **Depolarisation** (the rapid influx of sodium ions causing membrane potential to become positive) spreads as a wavefront through the myocardium; this is the electrical event that triggers contraction. **Repolarisation** (the return of cells to their resting state through potassium efflux) follows depolarisation in each cell.

The ECG electrode detects the potential difference between two points on the body surface as the wavefront of depolarisation and repolarisation passes. A wavefront moving **towards** an electrode produces a positive (upward) deflection; a wavefront moving **away from** an electrode produces a negative (downward) deflection. The amplitude of the deflection reflects the mass of myocardium depolarising simultaneously.

Fill in the blank: ECG depolarisation produces a positive deflection when the wavefront moves _____ the electrode; repolarisation produces deflections in the opposite direction; amplitude reflects the mass of myocardium depolarising.

5.1.2 ECG leads and their placement

The standard 12-lead ECG uses 10 electrodes to generate 12 leads, each providing a different view of cardiac electrical activity. The **six limb leads** (I, II, III, aVR, aVL, aVF) are derived from electrodes on the four limbs and examine the heart in the frontal plane; the **six precordial (chest) leads** (V1 to V6) examine the heart in the horizontal plane.

Electrode placement follows standard positions: **V1** (right of sternum, fourth intercostal space), **V2** (left of sternum, fourth ICS), **V3** (between V2 and V4), **V4** (fifth ICS, midclavicular line), **V5** (anterior axillary line, same level as V4), **V6** (midaxillary line, same level as V4 and V5). Incorrect electrode placement is a common cause of ECG artefact and misinterpretation.

Fill in the blank: The 12-lead ECG uses six limb leads (I, II, III, aVR, aVL, aVF) examining the frontal plane and six precordial leads (V1 to V6) examining the _____ plane; V1 is at the right of the sternum in the fourth ICS.

5.1.3 The normal ECG waveform

The normal ECG waveform consists of a sequence of deflections corresponding to specific electrical events. The **P wave** represents atrial depolarisation (normal duration below 120 ms, amplitude below 2.5 mm). The **PR interval** (normal 120 to 200 ms) represents conduction through the AV node and His-Purkinje system from onset of atrial depolarisation to onset of ventricular depolarisation. The **QRS complex** (normal duration below 120 ms) represents ventricular depolarisation.

The QRS complex consists of: the **Q wave** (initial negative deflection before the first positive deflection), the **R wave** (the first positive deflection), and the **S wave** (the negative deflection following the R wave). The **ST segment** (from end of QRS to onset of T wave) is normally isoelectric. The **T wave** represents ventricular repolarisation; it is normally upright in most leads except aVR and V1. The **QT interval** (from onset of QRS to end of T wave; normal corrected QTc below 440 ms in men and below 460 ms in women) reflects the total duration of ventricular depolarisation and repolarisation.

Fill in the blank: The ECG waveform: P wave (atrial depolarisation), PR interval (AV and His-Purkinje conduction; 120 to 200 ms), QRS complex (ventricular depolarisation; below 120 ms), ST segment (isoelectric), T wave (ventricular _____), QTc (total ventricular electrical cycle; below 440 ms men).

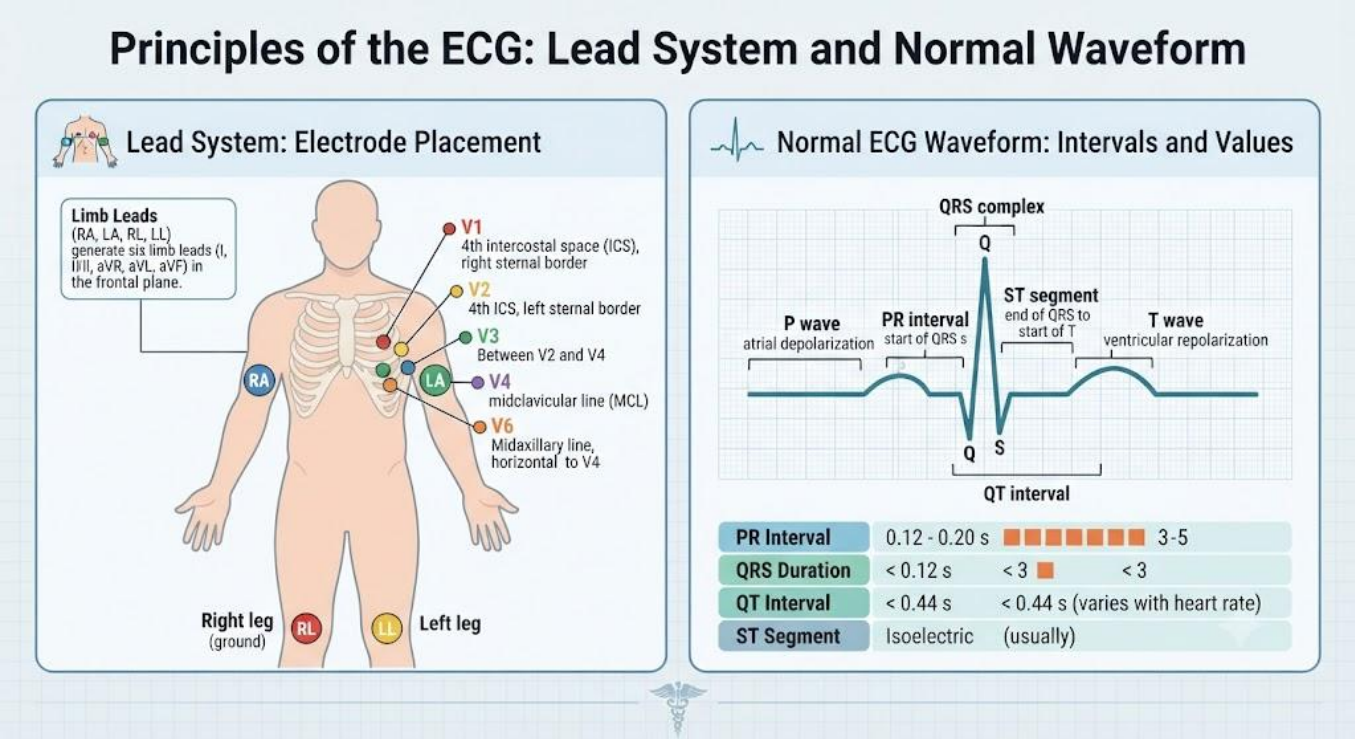

Figure 5.1: ECG lead placement and the components of the normal ECG waveform

Pilot questions 5.1

1. Explain the electrical basis of the ECG in terms of depolarisation, repolarisation, and electrode detection. (Linked to SLO 5.1)
2. Describe the placement of the six precordial ECG leads. (Linked to SLO 5.1)
3. Describe the P wave and PR interval and state the normal value for each. (Linked to SLO 5.1)
4. Describe the QRS complex and state its normal duration. (Linked to SLO 5.1)
5. Describe the ST segment, T wave, and QT interval and state the normal QTc. (Linked to SLO 5.1)

5.2 Systematic ECG Interpretation

5.2.1 Rate and rhythm assessment

A systematic approach to ECG interpretation reduces the risk of missing important findings. The recommended sequence is: rate, rhythm, axis, intervals (PR, QRS, QTc), waveform

morphology (P wave, QRS, ST segment, T wave), and clinical correlation. This sequence should be applied consistently to every ECG regardless of what is already suspected clinically.

Heart rate can be estimated by dividing 300 by the number of large squares between consecutive R waves (each large square equals 200 ms at standard paper speed of 25 mm/s) for regular rhythms. Normal rate is 60 to 100 bpm. Rhythm is assessed by examining whether the **P waves are regular and present before each QRS**, whether the **PR interval is constant**, and whether the **QRS complexes are narrow** (below 120 ms; indicating normal conduction through the ventricles) or wide (120 ms or above; indicating bundle branch block, ventricular origin, or aberrant conduction).

Fill in the blank: Heart rate is estimated by dividing 300 by large squares between R waves; rhythm assessment checks for regular P waves before each QRS, constant PR interval, and _____ (below 120 ms) or wide QRS complexes.

5.2.2 Axis determination

The cardiac axis describes the mean direction of ventricular depolarisation in the frontal plane, derived from the limb leads. Normal axis lies between minus 30 and plus 90 degrees. **Left axis deviation (LAD)** (axis between minus 30 and minus 90 degrees) is seen in left anterior fascicular block, inferior MI, and LVH. **Right axis deviation (RAD)** (axis above plus 90 degrees) is seen in RVH, pulmonary embolism, left posterior fascicular block, and tall thin individuals.

A quick method for axis estimation uses leads I and aVF: if both are predominantly positive, the axis is normal. If lead I is positive and aVF is negative, there is LAD. If lead I is negative and aVF is positive, there is RAD. If both leads I and aVF are negative, there is extreme axis deviation (northwest axis), seen in ventricular tachycardia and severe RVH.

Fill in the blank: Normal cardiac axis is minus 30 to plus 90 degrees; left axis deviation occurs in left anterior fascicular block and inferior MI; right axis deviation occurs in RVH and _____; if lead I is positive and aVF negative, axis is leftward.

5.2.3 Intervals, waveform morphology, and ST-T changes

PR interval prolongation (above 200 ms) indicates first-degree AV block (delayed AV conduction). A short PR interval (below 120 ms) may indicate pre-excitation (Wolff-Parkinson-White syndrome). **QRS prolongation** (120 ms or above) indicates bundle branch block (LBBB or RBBB) or ventricular conduction delay. **QTc prolongation** (above 440 ms men, above 460 ms women) predisposes to Torsades de Pointes ventricular tachycardia and may result from drugs (antiarrhythmics, antipsychotics, antibiotics), electrolyte disturbances (hypokalaemia, hypomagnesaemia), or congenital long QT syndrome.

ST segment elevation above the isoelectric line indicates **myocardial injury** (STEMI or pericarditis). **ST segment depression** indicates myocardial ischaemia or posterior MI. **T wave inversion** may indicate ischaemia, LVH strain pattern, bundle branch block, or pulmonary embolism. Peaked T waves may indicate hyperkalaemia. The combination of **ST changes with symptoms** of chest pain constitutes an acute coronary syndrome until proven otherwise and requires immediate clinical action.

Fill in the blank: PR prolongation indicates first-degree AV block; QTc prolongation predisposes to Torsades de Pointes; ST elevation indicates myocardial _____ (STEMI or pericarditis); ST depression indicates ischaemia or posterior MI.

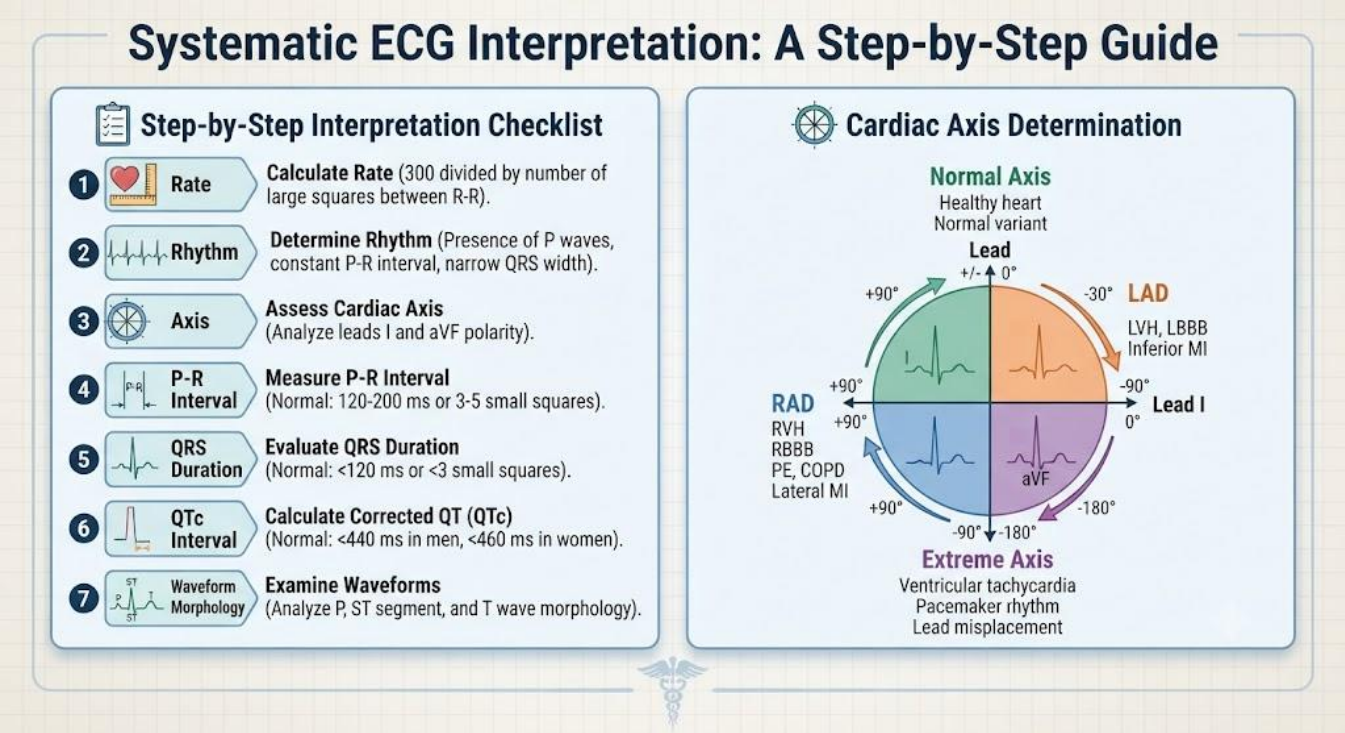

Figure 5.2: The systematic ECG interpretation sequence and axis determination using leads I and aVF

Pilot questions 5.2

1. Describe the systematic approach to ECG interpretation. (Linked to SLO 5.2)
2. Explain how heart rate is calculated from an ECG and state the normal range. (Linked to SLO 5.2)
3. Describe how cardiac axis is determined using leads I and aVF and state the causes of left and right axis deviation. (Linked to SLO 5.2)
4. Explain the significance of QTc prolongation and list three causes. (Linked to SLO 5.2)
5. Distinguish between ST elevation and ST depression and state the conditions associated with each. (Linked to SLO 5.2)

5.3 ECG Patterns in Common Cardiac Conditions

5.3.1 ECG in ischaemia and myocardial infarction

The ECG in acute myocardial infarction evolves through characteristic stages. In the **hyperacute phase** (minutes to hours after occlusion), **tall peaked T waves (hyperacute T waves)** appear in the territory of the affected artery. In the **acute phase**, **ST elevation** develops (STEMI); ST elevation in contiguous leads defines the territory of infarction: inferior MI (leads II, III, aVF), anterior MI (V1 to V4), lateral MI (I, aVL, V5, V6), and posterior MI (ST depression in V1 to V3, tall R waves in V1, confirmed by ST elevation in posterior leads V7 to V9).

As the infarction evolves, ST elevation resolves and is replaced by **T wave inversion** and development of **pathological Q waves** (duration above 40 ms, depth above 1 mm, or greater than one quarter of the R wave amplitude in the same lead). Q waves indicate **transmural myocardial necrosis** and persist permanently. In **NSTEMI or unstable angina**, the ECG shows ST depression and T wave inversion without ST elevation (non-STEMI pattern).

Fill in the blank: STEMI evolves from hyperacute T waves through ST elevation and T wave inversion to pathological Q waves indicating transmural necrosis; NSTEMI shows ST depression and T wave _____ without ST elevation.

5.3.2 ECG in chamber enlargement and bundle branch block

Left ventricular hypertrophy (LVH) on ECG is suggested by increased QRS voltage: the **Sokolow-Lyon index** (S wave in V1 plus R wave in V5 or V6 greater than 35 mm) is the most widely used criterion. LVH may also show a **strain pattern** (ST depression and T wave inversion in the lateral leads I, aVL, V5, and V6). **Right ventricular hypertrophy (RVH)** shows right axis deviation, dominant R wave in V1 (R greater than S in V1), and strain pattern in right precordial leads (V1 to V3).

Left bundle branch block (LBBB) is characterised by QRS duration of 120 ms or above, broad notched M-shaped R wave in lateral leads (I, aVL, V5, V6), no Q waves in lateral leads, and broad S wave in V1. LBBB completely changes the ST-T morphology and makes ECG interpretation for ischaemia unreliable. **Right bundle branch block (RBBB)** shows QRS of 120 ms or above, rsR' (RSR prime) pattern in V1 (giving the characteristic M-shaped appearance), and broad S wave in lateral leads I and V6.

Fill in the blank: LVH is diagnosed by Sokolow-Lyon index (S in V1 plus R in V5 or V6 above 35 mm); LBBB shows broad M-shaped R in lateral leads and absent Q waves; RBBB shows rsR' pattern in V1 and broad _____ waves in lateral leads.

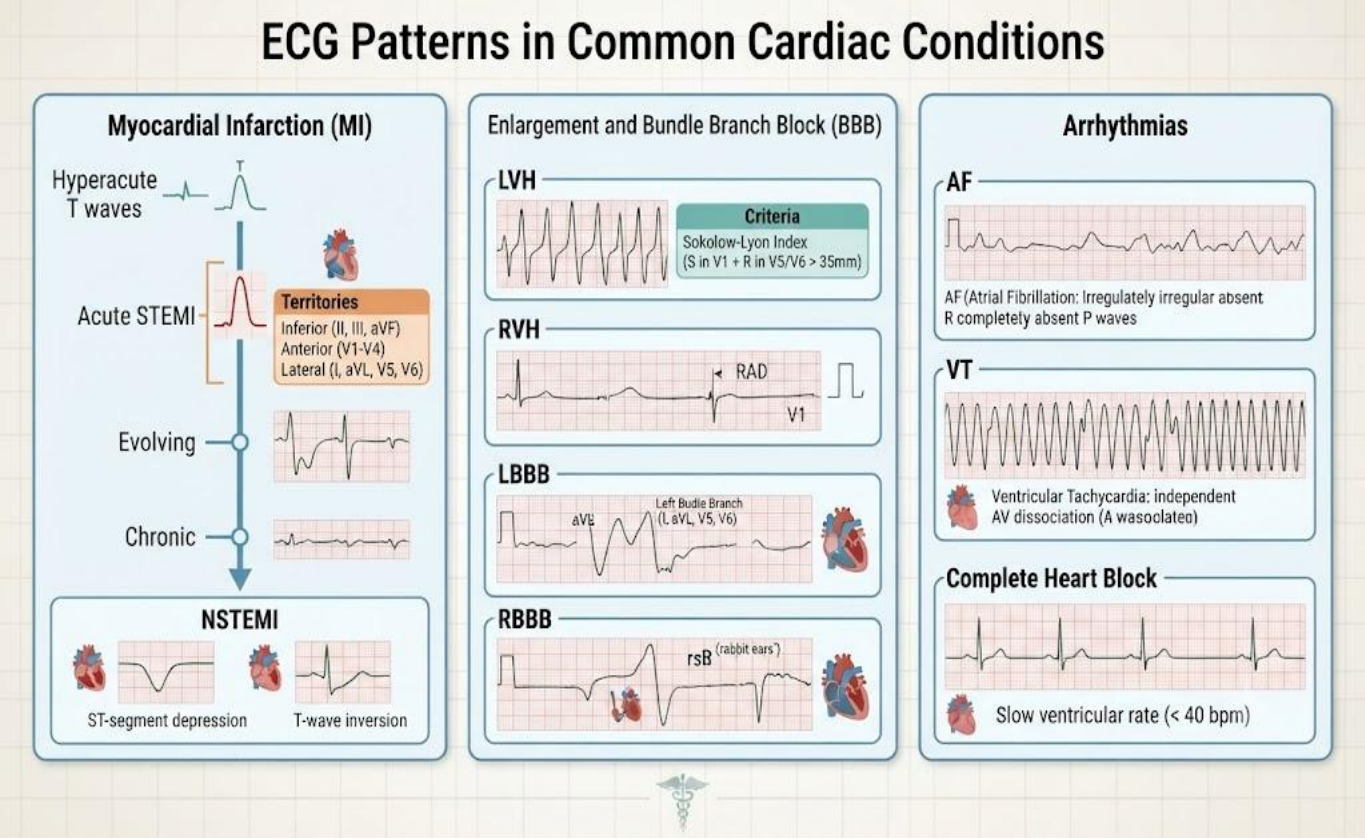

Figure 5.3: ECG patterns in myocardial infarction, chamber enlargement and bundle branch block, and common arrhythmias

5.3.3 ECG in arrhythmias

Atrial fibrillation (AF) is characterised by **absent P waves** (replaced by irregular fibrillatory baseline), an **irregularly irregular ventricular rhythm**, and narrow QRS complexes (unless bundle branch block or aberrant conduction is present). AF is the most common sustained cardiac arrhythmia and a major cause of stroke.

Supraventricular tachycardia (SVT) presents as regular narrow-complex tachycardia, typically at 150 to 250 bpm, with P waves that may be hidden in the QRS, immediately after the QRS, or occurring at a regular interval before the QRS. **Ventricular tachycardia (VT)** presents as regular wide-complex tachycardia (QRS above 120 ms), at 100 to 250 bpm, with AV dissociation (P waves and QRS complexes occurring independently), fusion beats, and capture beats — features that distinguish VT from SVT with aberrant conduction. **Complete heart block (third-degree AV block)** shows regular P waves and regular QRS complexes occurring independently (AV dissociation), with ventricular rate below 40 bpm.

Fill in the blank: Atrial fibrillation shows absent P waves, irregularly irregular ventricular rhythm; VT shows wide-complex regular tachycardia with AV _____; complete heart block shows independent P waves and QRS with ventricular rate below 40 bpm.

Pilot questions 5.3

1. Describe the ECG changes of STEMI from the hyperacute phase to the chronic phase. (Linked to SLO 5.3)
2. Describe the ECG localisation of myocardial infarction by territory. (Linked to SLO 5.3)
3. Describe the ECG criteria for LVH and LBBB. (Linked to SLO 5.3)
4. Describe the ECG features of atrial fibrillation. (Linked to SLO 5.3)
5. Describe the ECG features that distinguish ventricular tachycardia from SVT with aberrant conduction. (Linked to SLO 5.3)

5.4 Clinical Application of the ECG

5.4.1 ECG in clinical decision-making

The ECG directly determines management in several clinical scenarios. In **suspected acute coronary syndrome**, the ECG determines whether the patient has STEMI (requiring immediate primary PCI or thrombolysis) or NSTEMI (requiring antiplatelet therapy, anticoagulation, and risk stratification). In **palpitations or syncope**, the ECG may capture the causative arrhythmia or provide clues such as a long QT interval, pre-excitation (short PR and delta wave in WPW syndrome), or complete heart block.

In **breathlessness**, the ECG may show signs of pulmonary embolism (S1Q3T3 pattern: deep S in lead I, Q wave in III, T wave inversion in III; sinus tachycardia; right heart strain), heart failure (LVH, AF, LBBB), or an alternative diagnosis. In **monitoring of drug therapy** and electrolyte disturbances, the ECG detects QTc prolongation from antiarrhythmics or antibiotics and changes of hyperkalaemia (peaked T waves, widened QRS, sine wave pattern) or hypokalaemia (flattened T waves, prominent U waves).

Fill in the blank: STEMI on ECG requires immediate reperfusion (primary PCI or thrombolysis); palpitations or syncope ECG may reveal arrhythmia, long QT, or pre-excitation; hyperkalaemia produces peaked T waves, widened QRS, and _____ wave pattern.

5.4.2 Limitations of the ECG

The ECG has important limitations that the clinician must understand. A normal ECG does not exclude cardiac disease: approximately 10 percent of patients with confirmed acute MI have an initially normal or non-diagnostic ECG (particularly posterior and right ventricular MI). ECG changes may be non-specific: T wave inversions, ST changes, and axis deviations occur in many conditions and cannot be interpreted without clinical context.

ECG findings may be obscured by technical factors including incorrect electrode placement (a common error causing apparent axis deviation and ST changes), patient movement, poor skin-electrode contact, and electrical interference. **Serial ECGs** are far more informative than a single

ECG: changes that evolve over time (rising and falling ST segments, developing Q waves, resolving T wave inversions) are diagnostically highly significant and cannot be captured by a single tracing. The ECG must always be interpreted in the full clinical context of history, examination, and other investigations.

Fill in the blank: Limitations of the ECG include that a normal ECG does not exclude cardiac disease; changes may be non-specific; electrode _____ causes artefact; serial ECGs are more informative than a single tracing.

5.4.3 ECG as part of the cardiovascular investigation package

The ECG is rarely sufficient as a standalone investigation and is most valuable when combined with other investigations in a cardiovascular package. In **suspected ACS**, serial ECGs are combined with serial troponins (at 0 and 3 hours) and clinical risk scoring (GRACE, TIMI) to stratify management. In **arrhythmia investigation**, a single 12-lead ECG may be normal if the arrhythmia is paroxysmal; **24-hour Holter monitoring** or event recorders are needed to capture intermittent arrhythmias.

Echocardiography provides structural information (EF, wall motion, valves, pericardium) that the ECG cannot; **exercise stress testing** provokes ischaemia that may not be evident at rest; **coronary CT angiography or invasive angiography** defines coronary anatomy. The clinician who understands the complementary roles of these investigations selects the right test at the right time, avoiding unnecessary testing while ensuring that critical diagnoses are not missed.

Fill in the blank: The ECG is combined with serial troponins in ACS, Holter monitoring for paroxysmal _____, echocardiography for structural information, and exercise stress testing or coronary angiography for ischaemia evaluation.

Indication	Findings and Management Implications
Suspected ACS	ST elevation/depression, T-wave changes → guides reperfusion or medical therapy

Indication	Findings and Management Implications
Palpitations and Syncope	Arrhythmias (AF, VT, SVT, bradycardia) → informs antiarrhythmic therapy or pacing
Breathlessness	LVH, right heart strain, arrhythmias → supports diagnosis of heart failure or PE
Drug and Electrolyte Monitoring	QT prolongation, hyper/hypokalaemia changes → prevents arrhythmic complications
Investigation Package	Baseline assessment in hypertension, diabetes, pre-operative screening

Limitation	Clinical Note
Normal ECG not excluding disease	ACS, arrhythmias, or structural disease may still be present
Non-specific changes	ST/T changes may be due to multiple causes, reducing specificity
Technical artefact	Poor electrode placement or movement can mimic pathology
Single ECG insufficient	Serial ECGs or continuous monitoring often required for diagnosis

Table 5.1: Clinical applications, limitations, and the role of the ECG in the cardiovascular investigation package

Pilot questions 5.4

1. Explain how the ECG directly determines management in suspected acute coronary syndrome. (Linked to SLO 5.4)
2. Describe the ECG findings that suggest pulmonary embolism. (Linked to SLO 5.4)
3. Describe the ECG changes of hyperkalaemia in sequence. (Linked to SLO 5.4)

4. Describe the limitations of a single 12-lead ECG in clinical practice. (Linked to SLO 5.4)
5. Explain how the ECG fits into the cardiovascular investigation package alongside echocardiography, Holter monitoring, and coronary angiography. (Linked to SLO 5.4)

Series Summary

This Series introduced electrocardiography. The ECG records electrical cardiac activity from the body surface; depolarisation moving towards an electrode produces a positive deflection; amplitude reflects myocardial mass. The 12-lead ECG uses six limb leads (frontal plane) and six precordial leads V1 to V6 (horizontal plane); V1 is at the right sternal border in the fourth ICS and V4 at the fifth ICS midclavicular line. The normal waveform comprises: P wave (atrial depolarisation; below 120 ms, below 2.5 mm), PR interval (AV and His-Purkinje conduction; 120 to 200 ms), QRS complex (ventricular depolarisation; below 120 ms), isoelectric ST segment, T wave (ventricular repolarisation; normally upright), and QTc (below 440 ms men, below 460 ms women). Systematic interpretation follows: rate (300 divided by large squares between R waves), rhythm (P waves, PR interval, QRS width), axis (leads I and aVF; normal minus 30 to plus 90 degrees; LAD: left anterior fascicular block, inferior MI; RAD: RVH, pulmonary embolism), intervals, and waveform morphology.

ECG patterns in cardiac conditions: STEMI evolves from hyperacute T waves through ST elevation to T wave inversion and pathological Q waves; territories defined by lead groupings (inferior II/III/aVF, anterior V1 to V4, lateral I/aVL/V5/V6); NSTEMI shows ST depression and T inversion without ST elevation. LVH is suggested by Sokolow-Lyon index (S in V1 plus R in V5/V6 above 35 mm); LBBB shows broad M-shaped R in lateral leads; RBBB shows RSR' in V1 and broad S in lateral leads. AF shows absent P waves and irregularly irregular ventricular rhythm; VT shows wide-complex regular tachycardia with AV dissociation; complete heart block shows independent P waves and QRS with ventricular rate below 40 bpm. Clinical applications include immediate STEMI reperfusion decisions, arrhythmia characterisation, drug and electrolyte monitoring; limitations include a normal ECG not excluding disease, non-

specificity of changes, and the superiority of serial over single ECGs; the ECG is part of a cardiovascular investigation package including serial troponins, echocardiography, Holter monitoring, and coronary angiography.

Glossary of Terms

- **Depolarisation:** the rapid influx of sodium ions causing membrane potential to become positive; the electrical event triggering cardiac muscle contraction.
- **P wave:** the ECG deflection representing atrial depolarisation; normal duration below 120 ms, amplitude below 2.5 mm.
- **PR interval:** the interval from onset of P wave to onset of QRS complex, representing AV node and His-Purkinje conduction; normal 120 to 200 ms.
- **QRS complex:** the ECG complex representing ventricular depolarisation; normal duration below 120 ms.
- **ST segment:** the isoelectric portion of the ECG between the end of the QRS and the onset of the T wave; elevation indicates myocardial injury; depression indicates ischaemia.
- **QTc interval:** the corrected QT interval representing total ventricular electrical cycle; normal below 440 ms in men and below 460 ms in women; prolongation predisposes to Torsades de Pointes.
- **Sokolow-Lyon index:** an ECG criterion for LVH: S wave in V1 plus R wave in V5 or V6 greater than 35 mm.
- **Pathological Q wave:** a Q wave of duration above 40 ms or depth above 1 mm; indicates transmural myocardial necrosis.
- **Left bundle branch block (LBBB):** QRS duration above 120 ms with broad notched M-shaped R in lateral leads and absent Q waves; obscures ST-T analysis for ischaemia.
- **Atrial fibrillation (AF):** characterised by absent P waves, irregular fibrillatory baseline, and irregularly irregular ventricular rhythm; the most common sustained arrhythmia.

- **AV dissociation:** independent activity of the atria and ventricles; a key feature of complete heart block and ventricular tachycardia.
- **Holter monitoring:** continuous ambulatory 24-hour ECG recording used to detect paroxysmal arrhythmias not captured on a single 12-lead ECG.

Concept Check Questions [Series 5]

Objective Questions

1. A depolarisation wavefront moving _____ an electrode produces a positive deflection on the ECG; amplitude reflects the mass of myocardium depolarising. (Linked to SLO 5.1)
2. The PR interval normally measures 120 to 200 ms; a prolonged PR interval above 200 ms indicates first-degree AV _____. (Linked to SLO 5.1)
3. Heart rate on a regular ECG is estimated by dividing _____ by the number of large squares between consecutive R waves. (Linked to SLO 5.2)
4. STEMI evolves from hyperacute T waves through ST elevation to T wave inversion and pathological _____ waves indicating transmural necrosis. (Linked to SLO 5.3)
5. LBBB shows broad M-shaped R in lateral leads; RBBB shows RSR' pattern in _____ and broad S waves in lateral leads. (Linked to SLO 5.3)
6. Atrial fibrillation shows absent P waves and irregularly _____ ventricular rhythm; complete heart block shows independent P waves and QRS with rate below 40 bpm. (Linked to SLO 5.3)

Short-Answer Questions

7. Describe the placement of the six precordial ECG leads V1 to V6. (Linked to SLO 5.1)
8. Describe how cardiac axis is determined using leads I and aVF and state the conditions associated with left and right axis deviation. (Linked to SLO 5.2)

9. Describe the ECG features that distinguish ventricular tachycardia from SVT with aberrant conduction. (Linked to SLO 5.3)

Long-Answer Questions

10. Describe the systematic approach to ECG interpretation, covering rate, rhythm, axis, intervals, and waveform morphology. (Linked to SLO 5.2)
11. Describe the ECG changes in myocardial infarction, including STEMI evolution, territorial localisation, and the NSTEMI pattern. (Linked to SLO 5.3)

Answers to Concept Check Questions [Series 5]

Objective Questions

1. Towards.
2. Block.
3. 300.
4. Q.
5. V1.
6. Irregular.

Short-Answer Questions

7. V1: right sternal border, fourth ICS. V2: left sternal border, fourth ICS. V3: between V2 and V4. V4: fifth ICS, midclavicular line. V5: anterior axillary line, same level as V4. V6: midaxillary line, same level as V4 and V5.
8. If lead I and aVF are both positive, axis is normal (minus 30 to plus 90 degrees). If lead I positive and aVF negative: left axis deviation (LAD). If lead I negative and aVF positive: right axis deviation (RAD). If both negative: extreme axis deviation. LAD: left anterior fascicular block, inferior MI, LVH. RAD: RVH, pulmonary embolism, left posterior fascicular block.

9. VT: wide-complex regular tachycardia (QRS above 120 ms), AV dissociation (independent P waves and QRS), fusion beats (partially conducted sinus beat merges with VT beat), capture beats (normally conducted sinus beat interrupting VT), concordance (all precordial leads positive or all negative). SVT with aberrant conduction: narrow complex usually (unless LBBB), P waves visible before or after QRS, no AV dissociation, responds to Valsalva or adenosine.

Long-Answer Questions

10. **Basic response:** Rate: 300 divided by large squares between R waves (regular); count complexes in 6-second strip and multiply by 10 (irregular). Normal 60 to 100 bpm. Rhythm: regular P waves before each QRS (confirms sinus rhythm); constant PR interval; narrow (below 120 ms) or wide (above 120 ms) QRS. Axis: leads I and aVF; both positive = normal; I positive, aVF negative = LAD; I negative, aVF positive = RAD. Intervals: PR (120 to 200 ms; prolonged = first-degree block; short = pre-excitation); QRS (below 120 ms; wide = BBB or ventricular); QTc (below 440 ms men, below 460 ms women; prolonged = Torsades risk). Waveform: P wave (below 120 ms, below 2.5 mm; peaked or bifid = atrial enlargement); QRS voltage (high = LVH by Sokolow-Lyon); ST segment (elevation = injury/STEMI/pericarditis; depression = ischaemia); T waves (inversion = ischaemia, strain, BBB; peaked = hyperkalaemia).

    Value-added response: The systematic approach has value beyond completeness: it forces the interpreter to consider findings they might not have noticed if they read the ECG impressionistically, and a single finding found systematically (an unexpected long QTc, a subtle ST change, or an absent P wave) may change management completely.

11. **Basic response:** STEMI evolution: hyperacute phase (minutes to hours: tall peaked T waves in affected territory); acute phase (ST elevation; reciprocal ST depression in opposite leads); evolving phase (T wave inversion develops; ST elevation begins to resolve); chronic phase (pathological Q waves persist; ST normalises; T waves may remain inverted). Territorial localisation: inferior MI (ST elevation in II, III, aVF; right coronary or left circumflex artery); anterior MI (ST elevation V1 to V4; LAD territory); lateral MI (ST elevation I, aVL, V5, V6; circumflex or diagonal); posterior MI (ST

depression V1 to V3, tall R in V1; confirmed by ST elevation in V7 to V9). NSTEMI: ST depression and T wave inversion in leads of affected territory without ST elevation; elevated troponin confirms myocardial necrosis; treated differently from STEMI (no immediate reperfusion; risk-stratified management).

Value-added response: The ECG in ACS is the most time-critical investigation in medicine: STEMI requires reperfusion within 90 minutes of first medical contact (door-to-balloon time) and every 30 minutes of delay increases 30-day mortality by 7.5 percent; a systematic, rapid ECG interpretation protocol is therefore not merely an academic exercise but a skill with direct consequences for patient survival.

Answers to Pilot Questions [Series 5]

Part 5.1

1. Cardiac muscle cells are polarised at rest; depolarisation (sodium influx, membrane potential becomes positive) spreads as a wavefront through the myocardium, triggering contraction; repolarisation (potassium efflux) restores resting state. The ECG electrode detects the potential difference as the wavefront passes; a wavefront moving towards the electrode produces a positive deflection; moving away produces a negative deflection; amplitude reflects the mass of myocardium depolarising simultaneously.
2. V1: right sternal border, fourth ICS. V2: left sternal border, fourth ICS. V3: midway between V2 and V4. V4: fifth ICS, midclavicular line. V5: anterior axillary line at the same horizontal level as V4. V6: midaxillary line at the same horizontal level as V4 and V5.
3. P wave: represents atrial depolarisation; normal duration below 120 ms; normal amplitude below 2.5 mm in limb leads; best seen in lead II. PR interval: from onset of P wave to onset of QRS; represents conduction from the sinoatrial node through the AV node and His-Purkinje system to the ventricles; normal 120 to 200 ms.

4. The QRS complex represents ventricular depolarisation; normal duration below 120 ms; consists of Q wave (initial negative deflection before first positive), R wave (first positive deflection), and S wave (negative deflection after R wave); wide QRS (120 ms or above) indicates bundle branch block, ventricular rhythm, or aberrant conduction.
5. ST segment: isoelectric portion between end of QRS and onset of T wave; elevation (above isoelectric baseline) indicates myocardial injury (STEMI, pericarditis); depression indicates ischaemia or posterior MI. T wave: represents ventricular repolarisation; normally upright in most leads except aVR and V1; inversion suggests ischaemia, strain, or BBB. QTc: corrected QT interval from QRS onset to end of T wave; normal below 440 ms in men and below 460 ms in women; prolongation predisposes to Torsades de Pointes.

Part 5.2

1. Systematic sequence: Rate (300 divided by large squares between R waves; normal 60 to 100 bpm), Rhythm (P waves: regular and before each QRS? PR interval: constant? QRS: narrow or wide?), Axis (leads I and aVF), PR interval (120 to 200 ms), QRS duration (below 120 ms), QTc, P wave morphology, QRS voltage and morphology, ST segment, T waves, clinical correlation.
2. Heart rate on a regular ECG: count the number of large squares (5 mm wide, 200 ms at 25 mm/s) between two consecutive R wave peaks and divide 300 by this number. For irregular rhythms (AF): count the number of QRS complexes in a 6-second strip and multiply by 10. Normal heart rate: 60 to 100 bpm; below 60 is bradycardia; above 100 is tachycardia.
3. If lead I positive and aVF positive: normal axis (minus 30 to plus 90 degrees). If lead I positive and aVF negative: LAD (axis above minus 30 degrees). If lead I negative and aVF positive: RAD (axis above plus 90 degrees). LAD causes: left anterior fascicular block, inferior MI, LVH, LBBB. RAD causes: RVH, pulmonary embolism, left posterior fascicular block, normal in tall thin individuals.

4. QTc prolongation (above 440 ms men, above 460 ms women) predisposes to Torsades de Pointes, a form of polymorphic ventricular tachycardia that can degenerate into ventricular fibrillation and cause sudden cardiac death. Three causes: drug-induced (class Ia and III antiarrhythmics, antipsychotics, some antibiotics such as azithromycin); electrolyte disturbances (hypokalaemia, hypomagnesaemia, hypocalcaemia); congenital long QT syndrome.
5. ST elevation (above isoelectric baseline): indicates acute myocardial injury; causes: STEMI (territory-specific, with reciprocal depression), pericarditis (saddle-shaped, diffuse elevation, PR depression), LV aneurysm (persistent ST elevation months after MI), Brugada syndrome (V1 to V2). ST depression: indicates subendocardial ischaemia or posterior MI; causes: NSTEMI/unstable angina, digoxin effect (reverse tick), LVH strain, posterior MI (V1 to V3).

Part 5.3

1. Hyperacute phase (minutes to hours): tall peaked hyperacute T waves in the territory of the occluded artery. Acute phase (hours to days): ST elevation in contiguous leads; reciprocal ST depression in opposite leads; T waves begin to invert. Evolving phase (days to weeks): ST elevation resolves; T wave inversion deepens; pathological Q waves develop (duration above 40 ms or depth above 1 mm). Chronic phase (weeks to years): Q waves persist permanently; ST returns to baseline; T waves may remain inverted or normalise.
2. Inferior MI: ST elevation in leads II, III, and aVF; right coronary artery or left circumflex territory; associated with reciprocal depression in I and aVL. Anterior MI: ST elevation in V1 to V4; LAD artery territory; may extend to lateral leads (anterolateral). Lateral MI: ST elevation in I, aVL, V5, and V6; circumflex or diagonal artery. Posterior MI: ST depression in V1 to V3 with tall R waves (mirror image); confirmed by ST elevation in posterior leads V7 to V9.
3. LVH ECG criteria: Sokolow-Lyon index (S wave in V1 plus R wave in V5 or V6 greater than 35 mm); may show strain pattern (ST depression and T wave inversion in lateral leads I, aVL, V5, V6). LBBB: QRS duration above 120 ms; broad notched M-shaped R wave in

lateral leads I, aVL, V5, V6; absent Q waves in lateral leads; broad S wave in V1; LBBB obscures ST-T changes and makes ischaemia assessment unreliable.

4. Atrial fibrillation ECG: absent P waves; irregular fibrillatory baseline (fine or coarse); irregularly irregular ventricular rhythm (RR intervals vary without pattern); QRS complexes narrow unless bundle branch block or aberrant conduction is present; ventricular rate typically 100 to 160 bpm in uncontrolled AF.
5. VT features: wide-complex (QRS above 120 ms) regular tachycardia; AV dissociation (P waves and QRS occur independently, seen in about 50 percent of cases); fusion beats (sinus and ventricular complex merge, producing intermediate morphology); capture beats (normally conducted sinus beat interrupts VT, producing a narrow QRS); concordance (all precordial leads positive or all negative in VT). SVT with aberrant conduction lacks AV dissociation, may have P waves, and typically responds to Valsalva or adenosine.

Part 5.4

1. ST elevation on ECG in a patient with chest pain indicates STEMI; management: immediate reperfusion with primary percutaneous coronary intervention (PCI) if available within 120 minutes of first medical contact, or thrombolysis if PCI is not available within this timeframe; target door-to-balloon time below 90 minutes; activated immediately by ECG diagnosis without waiting for troponin results. ST depression or T wave inversion without ST elevation with troponin rise indicates NSTEMI: risk-stratified with GRACE score; no immediate reperfusion; antiplatelet and anticoagulation therapy.
2. Pulmonary embolism ECG findings: sinus tachycardia (most common); S1Q3T3 pattern (deep S wave in lead I, Q wave in lead III, T wave inversion in lead III); right axis deviation; right bundle branch block (new RBBB in the right clinical context); T wave inversions in right precordial leads (V1 to V4) indicating right heart strain; atrial fibrillation. Most findings are non-specific; normal ECG does not exclude PE.
3. Hyperkalaemia ECG changes in sequence: mild hyperkalaemia (above 5.5 mmol/L): tall peaked symmetrical T waves (tent-shaped) in precordial leads; moderate (above 6.0

mmol/L): PR prolongation, P wave flattening or disappearance; severe (above 7.0 mmol/L): QRS widening, bundle branch block pattern, sine wave appearance (QRS merges with T wave); very severe (above 8.0 mmol/L): ventricular fibrillation or asystole.

4. Limitations: a normal ECG does not exclude acute MI (approximately 10 percent of STEMI initially normal or non-diagnostic, particularly posterior and right ventricular); ECG changes are non-specific and must be interpreted in clinical context; electrode misplacement causes artefactual axis deviation and ST changes; a single ECG may miss paroxysmal arrhythmias; LBBB and paced rhythms make ischaemia assessment unreliable; serial ECGs substantially more informative than a single tracing.
5. Serial troponins (0 and 3 hours) plus serial ECGs in ACS: ECG gives immediate reperfusion decision, troponins confirm myocardial necrosis and quantify injury. Holter monitoring or event recorders: capture paroxysmal arrhythmias missed on a single ECG (palpitations, syncope). Echocardiography: provides structural information (EF, wall motion, valves, pericardium) that ECG cannot give. Exercise stress testing: provokes ST changes at peak exercise revealing ischaemia not present at rest. Coronary angiography: definitive anatomy of coronary arteries guided by ECG territory of infarction.

References and Attributions [Series 5]

Textual References

- Hampton, J. R. (2019). The ECG Made Easy (9th ed.). Edinburgh: Elsevier.
- Thaler, M. S. (2018). The Only EKG Book You'll Ever Need (9th ed.). Philadelphia: Wolters Kluwer.
- Kumar, P. and Clark, M. (2017). Kumar and Clark's Clinical Medicine (9th ed.). Edinburgh: Elsevier.
- Ibanez, B. et al. (2018). 2017 ESC STEMI Guidelines. European Heart Journal, 39(2), 119 to 177.

Figures, Plates, Tables, and Boxes

- Figure 5.1: ECG lead placement and normal waveform. AI prompt: "Two-panel diagram: left panel anterior chest and limb showing electrode positions for limb leads (RA, LA, RL, LL) and precordial leads V1 to V6 with positions labelled; right panel labelled ECG trace showing P wave, PR interval, QRS complex (Q, R, S), ST segment, T wave, QT interval with normal values; clean professional infographic." OER search: "ECG lead placement normal waveform P wave QRS T wave diagram labelled".
- Figure 5.2: Systematic ECG interpretation and axis determination. AI prompt: "Two-panel diagram: seven-step ECG checklist (left) and four-quadrant axis reference using leads I and aVF (right); clean professional infographic." OER search: "ECG systematic interpretation approach rate rhythm axis intervals waveform diagram".
- Figure 5.3: ECG patterns in common cardiac conditions. AI prompt: "Three-panel diagram: MI evolution and territorial localisation (panel 1), chamber enlargement and BBB (panel 2), arrhythmias AF, VT, complete heart block (panel 3); clean professional infographic." OER search: "ECG patterns myocardial infarction STEMI bundle branch block arrhythmia LVH diagram".
- Table 5.1: Clinical applications and limitations of the ECG. AI prompt: "Two-section table: Key Clinical Applications (ACS, palpitations, breathlessness, drug monitoring, investigation package) and Limitations (normal ECG, non-specific changes, artefact,

single ECG insufficient); alternating shading." OER search: "ECG clinical application limitations cardiovascular investigation package table".