La-Net

MED 606

CARDIOLOGY II

Course code: MED 606

Course title: Cardiology II

Course credit load: 2 Units

Series 1: Pathological Functional States of the Heart: Systolic and Diastolic Dysfunction, and Arrhythmias

- **Part 1.1: Heart Failure: Systolic Dysfunction**
  - Sub Part 1.1.1: Pathophysiology of systolic heart failure
  - Sub Part 1.1.2: Clinical presentation of systolic heart failure
  - Sub Part 1.1.3: Classification and grading of heart failure
- **Part 1.2: Heart Failure: Diastolic Dysfunction**
  - Sub Part 1.2.1: Pathophysiology of diastolic dysfunction
  - Sub Part 1.2.2: Clinical presentation and diagnosis of diastolic heart failure
  - Sub Part 1.2.3: Distinguishing systolic from diastolic heart failure
- **Part 1.3: Principles of Cardiac Arrhythmias**
  - Sub Part 1.3.1: Mechanisms of arrhythmia
  - Sub Part 1.3.2: Classification of arrhythmias
  - Sub Part 1.3.3: Clinical presentation of arrhythmias
- **Part 1.4: Common Arrhythmias**
  - Sub Part 1.4.1: Atrial fibrillation
  - Sub Part 1.4.2: Supraventricular tachycardia
  - Sub Part 1.4.3: Ventricular arrhythmias

Introduction

The heart can fail in genuinely two distinct ways, either through weakened contraction or through impaired relaxation and filling, and its normal rhythm can be disrupted in an equally wide variety of ways, from a benign, unnoticed irregularity to a genuinely life-threatening chaotic electrical storm. This opening Series of **Cardiology II** works through both of these fundamental pathological states, heart failure and arrhythmia, building the theoretical foundation this course rests on.

The aim of this Series is to equip you with an understanding of the pathophysiology, presentation, classification, and grading of systolic and diastolic heart failure, and the mechanisms, classification, and clinical presentation of cardiac arrhythmias, including the specific common arrhythmias of atrial fibrillation, supraventricular tachycardia, and ventricular arrhythmias.

This Series opens with **heart failure: systolic dysfunction**, moves through **heart failure: diastolic dysfunction** and **principles of cardiac arrhythmias**, and closes with **common arrhythmias**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 1.1:** describe the pathophysiology and clinical presentation of systolic heart failure
- **SLO 1.2:** classify and grade heart failure
- **SLO 1.3:** describe the pathophysiology and clinical presentation of diastolic heart failure
- **SLO 1.4:** distinguish systolic from diastolic heart failure
- **SLO 1.5:** describe the mechanisms and classification of cardiac arrhythmias
- **SLO 1.6:** describe the clinical presentation of cardiac arrhythmias
- **SLO 1.7:** describe atrial fibrillation and supraventricular tachycardia
- **SLO 1.8:** describe ventricular arrhythmias

1.1 Heart Failure: Systolic Dysfunction

1.1.1 pathophysiology of systolic heart failure

A failure of the heart's pumping capacity itself

Systolic heart failure, also termed heart failure with reduced ejection fraction, results from impaired ventricular contractility, most commonly from ischaemic heart disease or dilated cardiomyopathy, reducing the proportion of blood ejected with each contraction, and the body's compensatory response, activating the sympathetic nervous system and the renin-angiotensin-aldosterone system, initially maintains adequate perfusion but, over time, genuinely worsens the underlying condition through progressive ventricular remodelling.

This **neurohormonal activation**, while adaptive in the short term following an acute insult, becomes genuinely maladaptive when sustained chronically, driving further myocardial damage, fluid retention, and progressive decline in cardiac function, a pathophysiological insight that directly underlies the specific pharmacological treatment strategies, deliberately targeting this compensatory pathway, discussed further in the management Series that follows later in this course.

Fill in the blank: Systolic heart failure most commonly results from ischaemic heart disease or dilated ________.

1.1.2 clinical presentation of systolic heart failure

Congestion behind a failing pump

Systolic heart failure presents with symptoms reflecting fluid congestion both behind the failing left ventricle, producing breathlessness, orthopnoea, and paroxysmal nocturnal dyspnoea, and behind a failing right ventricle, producing peripheral oedema, raised jugular venous pressure, and hepatic congestion, with many patients demonstrating features of both presentations simultaneously given the frequent progression from isolated left-sided to combined biventricular failure over time.

Examination findings include a displaced apex beat reflecting ventricular enlargement, a third heart sound reflecting rapid ventricular filling into a stiff, failing chamber, and, on auscultation of the chest, bilateral basal crackles reflecting pulmonary oedema, findings that together, alongside the symptom pattern already described, allow a confident clinical diagnosis to be reached before formal echocardiographic confirmation.

Fill in the blank: A third heart sound reflects rapid ventricular filling into a stiff, ________ chamber.

1.1.3 classification and grading of heart failure

Standardised frameworks for communicating severity

Heart failure severity is graded using the **New York Heart Association functional classification**, ranging from class one, no limitation of physical activity, to class four, symptoms at rest, a widely used, simple framework allowing consistent communication of symptomatic severity between clinicians and tracking of change over time in an individual patient.

Heart failure is further classified according to **ejection fraction**, measured on echocardiography, as heart failure with reduced ejection fraction, corresponding broadly to the systolic dysfunction discussed in this Part, heart failure with preserved ejection fraction, corresponding broadly to the diastolic dysfunction discussed in the following Part, and heart failure with mildly reduced ejection fraction, an intermediate category recognised in more recent classification frameworks.

Fill in the blank: New York Heart Association class four describes heart failure symptoms present at ________.

Figure 1.1: A clinician examining jugular venous pressure and peripheral oedema during heart failure assessment.

Pilot questions 1.1

1. Describe the pathophysiology of systolic heart failure. (Linked to SLO 1.1)
2. Explain why sustained neurohormonal activation becomes maladaptive in heart failure. (Linked to SLO 1.1)
3. Describe the symptoms and signs of left-sided versus right-sided heart failure. (Linked to SLO 1.1)
4. Describe the New York Heart Association functional classification. (Linked to SLO 1.2)
5. Distinguish heart failure with reduced, mildly reduced, and preserved ejection fraction. (Linked to SLO 1.2)

1.2 Heart Failure: Diastolic Dysfunction

1.2.1 pathophysiology of diastolic dysfunction

A failure of relaxation, not contraction

Diastolic heart failure, also termed heart failure with preserved ejection fraction, results from impaired ventricular relaxation and filling despite genuinely preserved contractile function, most commonly from longstanding hypertension causing left ventricular hypertrophy and increased chamber stiffness, or, less commonly, from hypertrophic or restrictive cardiomyopathy, discussed further in a later Series of this course.

This impaired relaxation raises filling pressure required to achieve adequate ventricular filling, producing the same downstream congestive symptoms as systolic heart failure despite the ventricle's contractile function remaining genuinely normal on echocardiographic assessment, a pathophysiological distinction that, while conceptually clear, can genuinely complicate clinical diagnosis given the very similar symptomatic presentation the two conditions produce.

Fill in the blank: Diastolic heart failure most commonly results from longstanding hypertension causing left ventricular ________.

1.2.2 clinical presentation and diagnosis of diastolic heart failure

Similar symptoms, a genuinely different underlying mechanism

Clinical presentation of diastolic heart failure closely mirrors that of systolic heart failure, breathlessness, orthopnoea, and peripheral oedema, meaning symptoms and signs alone genuinely cannot reliably distinguish between the two conditions, and confirming a preserved ejection fraction on echocardiography, alongside evidence of diastolic dysfunction from specific echocardiographic measurements, is essential to establish the diagnosis with confidence.

Patients with diastolic heart failure are typically older, more commonly female, and more likely to have hypertension, atrial fibrillation, and other significant comorbidities compared with the typical systolic heart failure population, a demographic pattern that, while not diagnostic in itself, can genuinely raise clinical suspicion for this specific type of heart failure before formal echocardiographic confirmation is obtained.

Fill in the blank: Patients with diastolic heart failure are typically older, more commonly ________, and have more comorbidities.

1.2.3 distinguishing systolic from diastolic heart failure

Why the distinction genuinely matters clinically

Correctly distinguishing systolic from diastolic heart failure carries genuine treatment implications, since several of the specific pharmacological therapies with proven mortality benefit in systolic heart failure, covered in detail in a later management-focused Series, have not shown the same clear benefit in diastolic heart failure, meaning management of the latter condition focuses more heavily on symptom control and treating underlying contributing conditions such as hypertension.

Echocardiography remains the definitive investigation distinguishing the two conditions, directly measuring ejection fraction and assessing diastolic function through specific parameters, and every patient presenting with a new diagnosis of heart failure should undergo this investigation before definitive treatment planning, given how directly the distinction between systolic and diastolic dysfunction shapes the most appropriate subsequent management approach.

Fill in the blank: Echocardiography remains the definitive investigation distinguishing systolic from diastolic ________.

Table 1.1: Comparing systolic and diastolic heart failure

Feature	Systolic heart failure	Diastolic heart failure
Ejection fraction	Reduced	Preserved
Core mechanism	Impaired contraction	Impaired relaxation and filling
Typical patient	Often younger, ischaemic heart disease common	Often older, female, hypertensive

Pilot questions 1.2

1. Describe the pathophysiology of diastolic heart failure. (Linked to SLO 1.3)
2. List the common causes of diastolic heart failure. (Linked to SLO 1.3)
3. Explain why symptoms alone cannot reliably distinguish systolic from diastolic heart failure. (Linked to SLO 1.4)
4. Describe the typical demographic profile of a patient with diastolic heart failure. (Linked to SLO 1.4)

5. Explain why echocardiography is essential in every new heart failure diagnosis. (Linked to SLO 1.4)

1.3 Principles of Cardiac Arrhythmias

1.3.1 mechanisms of arrhythmia

How normal electrical rhythm goes wrong

Cardiac arrhythmias arise through three broad mechanisms: **abnormal automaticity**, where a site other than the normal sinoatrial node begins generating its own electrical impulses, **triggered activity**, abnormal electrical activity arising from oscillations in membrane potential following a normal heartbeat, and **re-entry**, where an electrical impulse travels in a continuous, self-sustaining circuit rather than extinguishing normally, the most common mechanism underlying many clinically significant arrhythmias.

Re-entry requires a specific anatomical or functional circuit with two pathways of differing conduction velocity and refractory period, allowing an impulse to travel down one pathway and back up the other in a continuous loop, and understanding this specific mechanism directly explains both the clinical behaviour of many re-entrant arrhythmias and the rationale behind certain specific treatment approaches, including catheter ablation, covered further in a later Series of this course.

Fill in the blank: Re-entry requires a specific circuit with two pathways of differing conduction velocity and refractory ________.

1.3.2 classification of arrhythmias

Sorting by rate and site of origin

Arrhythmias are classified by rate, as **bradyarrhythmias**, abnormally slow heart rhythms, or **tachyarrhythmias**, abnormally fast heart rhythms, and by site of origin, as **supraventricular**, arising above the ventricles, or **ventricular**, arising within the ventricles themselves, a classification framework that directly guides the specific differential diagnosis and the urgency of assessment a given arrhythmia presentation warrants.

Ventricular arrhythmias generally carry greater clinical concern than supraventricular arrhythmias given their closer association with underlying structural heart disease and their genuinely greater potential to produce haemodynamic compromise or degenerate into a life-threatening rhythm, though this general principle requires nuanced clinical judgement rather than blanket application, since some supraventricular arrhythmias can also produce significant haemodynamic instability in specific clinical contexts.

Fill in the blank: Tachyarrhythmias are abnormally ________ heart rhythms.

1.3.3 clinical presentation of arrhythmias

From no symptoms at all to genuine cardiac arrest

Arrhythmias present with a genuinely wide clinical spectrum, ranging from entirely asymptomatic, identified incidentally on routine examination or investigation, through palpitations, an unpleasant awareness of the heartbeat, dizziness, and syncope, to, in the most severe presentations, cardiac arrest, and the specific symptoms produced depend heavily on the arrhythmia's rate, duration, and the presence or absence of underlying structural heart disease.

Assessment of a patient with suspected arrhythmia relies heavily on **electrocardiography**, ideally captured during the actual episode of symptoms wherever practically achievable, since interpretation of the arrhythmia's specific electrocardiographic characteristics genuinely determines both the diagnosis and the most appropriate management approach, with ambulatory monitoring devices used to capture intermittent arrhythmias that do not occur during a single, brief clinic assessment.

Fill in the blank: Electrocardiography is ideally captured during the actual episode of symptoms wherever practically ________.

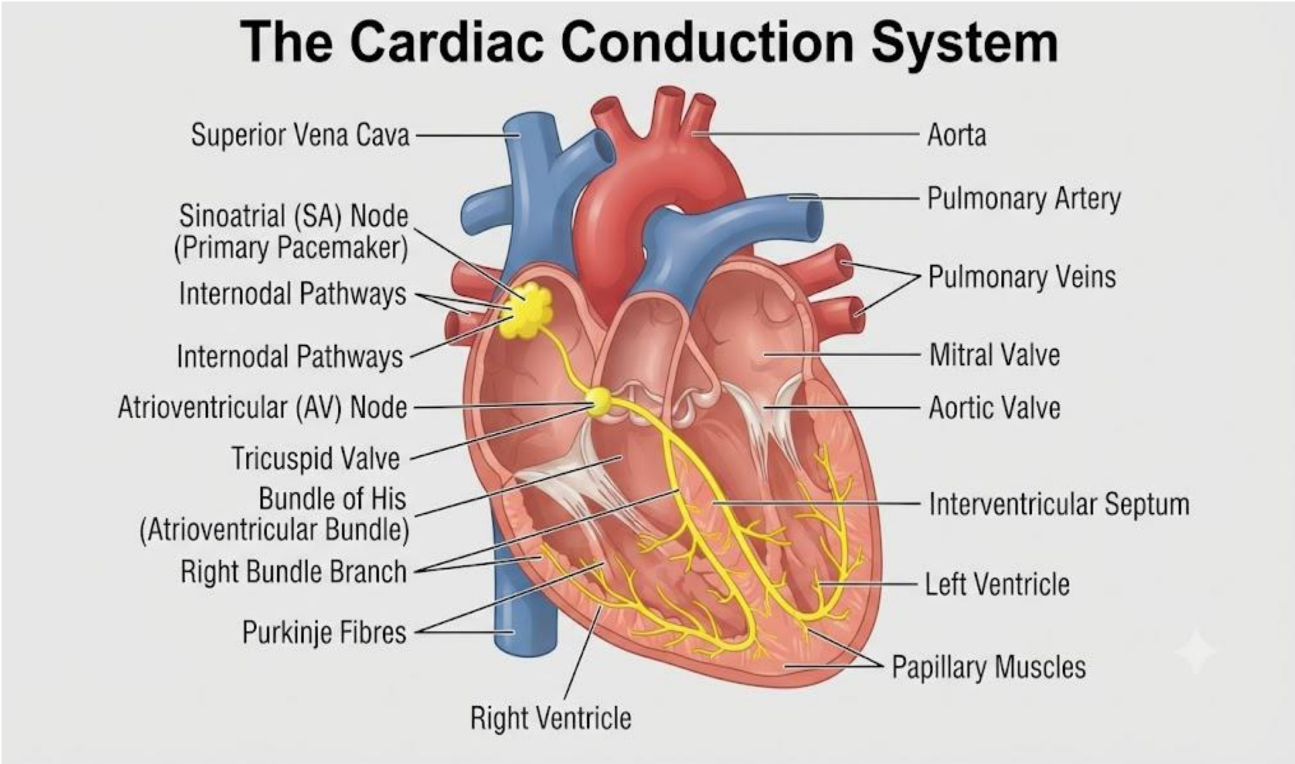

Figure 1.2: A labelled diagram of the heart's normal electrical conduction system.

Pilot questions 1.3

1. List the three broad mechanisms of arrhythmia. (Linked to SLO 1.5)
2. Describe re-entry and the anatomical requirement for it to occur. (Linked to SLO 1.5)
3. Distinguish bradyarrhythmia from tachyarrhythmia. (Linked to SLO 1.5)
4. Explain why ventricular arrhythmias generally carry greater clinical concern. (Linked to SLO 1.6)
5. Explain why electrocardiography captured during symptoms is genuinely valuable. (Linked to SLO 1.6)

1.4 Common Arrhythmias

1.4.1 atrial fibrillation

The most commonly encountered sustained arrhythmia

Atrial fibrillation, chaotic, disorganised atrial electrical activity replacing normal, coordinated atrial contraction, produces an irregularly irregular ventricular rhythm and loss of the atrial contribution to ventricular filling, and is the most common sustained arrhythmia encountered in clinical practice, with prevalence rising considerably with advancing age and in the presence of structural heart disease or hypertension.

Beyond its direct symptomatic and haemodynamic effects, atrial fibrillation carries a genuinely significant risk of **thromboembolism**, since stasis of blood within the poorly contracting, fibrillating atria predisposes to clot formation, most classically within the left atrial appendage, and subsequent embolisation, most seriously to the brain producing ischaemic stroke, a risk requiring careful, individualised assessment and, where indicated, anticoagulation, covered further in the management Series that follows later in this course.

Fill in the blank: Atrial fibrillation predisposes to clot formation most classically within the left atrial ________.

1.4.2 supraventricular tachycardia

A genuinely diverse category with a shared broad presentation

Supraventricular tachycardia, a broad term encompassing several distinct arrhythmias arising above the ventricles, including atrioventricular nodal re-entrant tachycardia and atrioventricular re-entrant tachycardia involving an accessory pathway, typically presents with sudden-onset, regular, rapid palpitations, often in an otherwise structurally normal heart, and, while genuinely alarming to the patient experiencing it, is generally not immediately life-threatening in most cases.

Acute management of a haemodynamically stable episode includes **vagal manoeuvres**, physical techniques stimulating the vagus nerve to slow conduction through the atrioventricular node, and, where these are unsuccessful, intravenous adenosine, a short-acting medication that briefly blocks atrioventricular nodal conduction, terminating many re-entrant supraventricular tachycardias by breaking the re-entry circuit already discussed earlier in this Part.

Fill in the blank: Vagal manoeuvres stimulate the vagus nerve to slow conduction through the atrioventricular ________.

1.4.3 ventricular arrhythmias

Arrhythmias carrying genuinely greater clinical weight

Ventricular tachycardia, a rapid rhythm originating within the ventricles, can be haemodynamically stable or unstable, and, particularly in the presence of underlying structural heart disease, carries genuine risk of degenerating into **ventricular fibrillation**, chaotic, disorganised ventricular electrical activity producing no effective cardiac output, a genuine cardiac arrest requiring immediate defibrillation and resuscitation given the absence of any meaningful spontaneous circulation.

Recognising ventricular tachycardia promptly and distinguishing it from supraventricular tachycardia with aberrant conduction, a genuinely important but sometimes challenging electrocardiographic distinction, matters considerably given the different management approach and generally more urgent clinical concern ventricular tachycardia warrants, and any wide complex tachycardia should generally be treated as ventricular in origin until proven otherwise given the genuinely serious consequences of a missed diagnosis in this specific clinical scenario.

Fill in the blank: Ventricular fibrillation is chaotic, disorganised ventricular electrical activity producing no effective cardiac ________.

Table 1.2: Comparing atrial fibrillation, supraventricular tachycardia, and ventricular arrhythmias

Arrhythmia	Typical rhythm character	Key clinical concern
Atrial fibrillation	Irregularly irregular	Thromboembolism risk
Supraventricular tachycardia	Regular, rapid	Generally not immediately life-threatening
Ventricular tachycardia	Regular or variable, wide complex	Risk of degeneration to ventricular fibrillation

Pilot questions 1.4

1. Describe atrial fibrillation and its typical rhythm characteristic. (Linked to SLO 1.7)
2. Explain why atrial fibrillation carries a risk of thromboembolism. (Linked to SLO 1.7)
3. Describe the acute management of a stable supraventricular tachycardia episode. (Linked to SLO 1.7)
4. Describe ventricular fibrillation and its immediate management. (Linked to SLO 1.8)

5. Explain why a wide complex tachycardia should generally be treated as ventricular until proven otherwise. (Linked to SLO 1.8)

Series Summary

This opening Series built the theoretical foundation of two fundamental pathological states of the heart. Part 1.1 covered the pathophysiology, presentation, and classification of systolic heart failure, while Part 1.2 turned to diastolic heart failure and how it is distinguished from systolic dysfunction. Part 1.3 covered the mechanisms, classification, and presentation of cardiac arrhythmias, and Part 1.4 closed with the common arrhythmias of atrial fibrillation, supraventricular tachycardia, and ventricular arrhythmias.

You should now be able to describe and classify systolic and diastolic heart failure and the common cardiac arrhythmias covered across all four Parts of this Series, meeting the outcomes set for this Series. Series 2 turns next to ischaemic heart disease and cardiomyopathies.

Glossary of Terms

- **Atrial fibrillation:** chaotic, disorganised atrial electrical activity producing an irregularly irregular ventricular rhythm.
- **Diastolic heart failure:** heart failure with preserved ejection fraction, from impaired ventricular relaxation.
- **Ejection fraction:** the proportion of blood ejected from the ventricle with each contraction.
- **New York Heart Association classification:** a framework grading heart failure symptomatic severity from class one to four.
- **Re-entry:** an arrhythmia mechanism where an impulse travels in a continuous, self-sustaining circuit.
- **Supraventricular tachycardia:** a rapid rhythm arising above the ventricles, typically regular and rapid.
- **Systolic heart failure:** heart failure with reduced ejection fraction, from impaired ventricular contractility.
- **Tachyarrhythmia:** an abnormally fast heart rhythm.
- **Thromboembolism:** clot formation and subsequent embolisation, a significant risk of atrial fibrillation.

- **Vagal manoeuvre:** a physical technique stimulating the vagus nerve to slow atrioventricular conduction.
- **Ventricular fibrillation:** chaotic ventricular electrical activity producing no effective cardiac output.
- **Ventricular tachycardia:** a rapid rhythm originating within the ventricles.

Concept Check Questions [Series 1]

Objective questions

1. Systolic heart failure most commonly results from ischaemic heart disease or dilated ________. (Linked to SLO 1.1)
2. New York Heart Association class four describes heart failure symptoms present at ________. (Linked to SLO 1.2)
3. Diastolic heart failure most commonly results from longstanding hypertension causing left ventricular ________. (Linked to SLO 1.3)
4. Re-entry requires a specific circuit with two pathways of differing conduction velocity and refractory ________. (Linked to SLO 1.5)
5. Atrial fibrillation predisposes to clot formation most classically within the left atrial ________. (Linked to SLO 1.7)

Short-answer questions

6. Describe the symptoms and signs of left-sided versus right-sided heart failure. (Linked to SLO 1.1)
7. Explain why symptoms alone cannot reliably distinguish systolic from diastolic heart failure. (Linked to SLO 1.4)
8. List the three broad mechanisms of arrhythmia. (Linked to SLO 1.5)
9. Describe the acute management of a stable supraventricular tachycardia episode. (Linked to SLO 1.7)
10. Describe ventricular fibrillation and its immediate management. (Linked to SLO 1.8)

Long-answer questions

11. Discuss the pathophysiology, presentation, and classification of systolic heart failure. (Linked to SLO 1.1, 1.2)

12. Describe diastolic heart failure and how it is distinguished from systolic heart failure. (Linked to SLO 1.3, 1.4)
13. Discuss the mechanisms, classification, and clinical presentation of cardiac arrhythmias. (Linked to SLO 1.5, 1.6)
14. Describe atrial fibrillation and supraventricular tachycardia. (Linked to SLO 1.7)
15. Discuss ventricular arrhythmias and why they carry genuine clinical significance. (Linked to SLO 1.8)

Answers to Concept Check Questions [Series 1]

Objective questions

1. Cardiomyopathy.
2. Rest.
3. Hypertrophy.
4. Period.
5. Appendage.

Short-answer questions

6. Left-sided failure produces breathlessness and orthopnoea; right-sided failure produces oedema and raised jugular venous pressure.
7. Both conditions produce a very similar symptomatic presentation despite genuinely different underlying mechanisms.
8. The three mechanisms are abnormal automaticity, triggered activity, and re-entry.
9. Management includes vagal manoeuvres and, if unsuccessful, intravenous adenosine.
10. Ventricular fibrillation is chaotic electrical activity with no cardiac output, requiring immediate defibrillation and resuscitation.

Long-answer questions

11. **Basic response:** Systolic heart failure results from impaired contractility with compensatory neurohormonal activation becoming maladaptive over time; presentation includes congestive symptoms and signs; classification uses New York Heart Association class and ejection fraction category.

    Value-added response: A value-added response notes that neurohormonal activation, while initially adaptive, drives further myocardial damage when sustained chronically.

12. **Basic response:** Diastolic heart failure results from impaired relaxation with preserved ejection fraction, commonly from hypertension; distinction from systolic failure requires echocardiography given similar symptomatic presentation.

    Value-added response: A value-added response notes that treatment benefit for several systolic heart failure therapies has not been clearly shown in diastolic heart failure.

13. **Basic response:** Mechanisms include abnormal automaticity, triggered activity, and re-entry; arrhythmias are classified by rate and site of origin; presentation ranges from asymptomatic to cardiac arrest, assessed with electrocardiography.

    Value-added response: A value-added response notes that ambulatory monitoring captures intermittent arrhythmias not present during a single clinic visit.

14. **Basic response:** Atrial fibrillation produces an irregularly irregular rhythm with thromboembolism risk; supraventricular tachycardia produces regular, rapid palpitations, managed with vagal manoeuvres or adenosine.

    Value-added response: A value-added response notes that atrial fibrillation prevalence rises considerably with advancing age and structural heart disease.

15. **Basic response:** Ventricular tachycardia can degenerate into ventricular fibrillation, a genuine cardiac arrest requiring immediate defibrillation, given closer association with structural heart disease and haemodynamic compromise.

    Value-added response: A value-added response notes that any wide complex tachycardia should generally be treated as ventricular until proven otherwise.

Answers to Pilot Questions [Series 1]

Part 1.1

1. Systolic heart failure results from impaired ventricular contractility, commonly from ischaemic heart disease or dilated cardiomyopathy.
2. Sustained activation drives further myocardial damage and fluid retention, worsening the underlying condition over time.
3. Left-sided failure produces breathlessness and orthopnoea; right-sided failure produces oedema and raised jugular venous pressure.

4. The classification ranges from class one, no limitation, to class four, symptoms at rest.
5. Reduced ejection fraction reflects systolic dysfunction, preserved reflects diastolic dysfunction, and mildly reduced is an intermediate category.

Part 1.2

1. Diastolic heart failure results from impaired ventricular relaxation and filling despite preserved contractile function.
2. Common causes include longstanding hypertension and hypertrophic or restrictive cardiomyopathy.
3. Both conditions produce very similar congestive symptoms despite different underlying mechanisms.
4. Patients are typically older, more commonly female, and more likely to have hypertension and atrial fibrillation.
5. Echocardiography directly measures ejection fraction and diastolic function, directly shaping subsequent management.

Part 1.3

1. The three mechanisms are abnormal automaticity, triggered activity, and re-entry.
2. Re-entry requires a circuit with two pathways of differing conduction velocity and refractory period, sustaining a continuous loop.
3. Bradyarrhythmias are abnormally slow, while tachyarrhythmias are abnormally fast heart rhythms.
4. Ventricular arrhythmias are more closely associated with structural heart disease and haemodynamic compromise.
5. Capturing the electrocardiogram during symptoms directly confirms the diagnosis and guides management.

Part 1.4

1. Atrial fibrillation is chaotic atrial activity producing an irregularly irregular ventricular rhythm.
2. Stasis of blood in the poorly contracting atria predisposes to clot formation and embolisation.
3. Management includes vagal manoeuvres and, if unsuccessful, intravenous adenosine.
4. Ventricular fibrillation is chaotic activity with no cardiac output, requiring immediate defibrillation and resuscitation.
5. Treating as ventricular until proven otherwise avoids the serious consequences of a missed ventricular tachycardia diagnosis.

References and Attributions [Series 1]

- Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
- Libby, P., Bonow, R. O., Mann, D. L., Tomaselli, G. F., & Braunwald, E. (2021). Braunwald's Heart Disease (12th ed.). Elsevier.
- European Society of Cardiology (2021). Guidelines for the Diagnosis and Treatment of Acute and Chronic Heart Failure. European Heart Journal.
- National Institute for Health and Care Excellence (2021). Atrial Fibrillation: Diagnosis and Management (NG196). NICE.

Figures, Plates, Tables, and Boxes

- Figure 1.1: A clinician examining jugular venous pressure and peripheral oedema during heart failure assessment. AI-generated using the prompt: "Create a realistic clinical illustration titled Heart Failure Clinical Examination, showing a clinician observing a patient's neck for jugular venous pressure and pressing gently on the patient's ankle to assess for oedema, examination room setting. Clean clinical illustration style, no text."
- Table 1.1: Comparing systolic and diastolic heart failure. AI-generated using the prompt: "Create a clean reference table titled Comparing Systolic and Diastolic Heart Failure, listing feature, systolic heart failure, and diastolic heart failure. Simple clinical reference table style with outside border."
- Figure 1.2: A labelled diagram of the heart's normal electrical conduction system. AI-generated using the prompt: "Create a clean, accurate labelled anatomical diagram titled The Cardiac Conduction System, showing the heart with the sinoatrial node, atrioventricular node, bundle of His, and Purkinje fibres clearly labelled in their normal anatomical positions. Educational anatomical diagram style, no photographic content."
- Table 1.2: Comparing atrial fibrillation, supraventricular tachycardia, and ventricular arrhythmias. AI-generated using the prompt: "Create a clean reference table titled Comparing Atrial Fibrillation, Supraventricular Tachycardia, and Ventricular Arrhythmias, listing arrhythmia, typical rhythm character, and key clinical concern. Simple clinical reference table style with outside border."

Course code: MED 606

Course title: Cardiology II

Course credit load: 2 Units

Series 2: Ischaemic Heart Disease and Cardiomyopathies

- **Part 2.1: Ischaemic Heart Disease I: Chronic Coronary Syndromes**
    - Sub Part 2.1.1: Pathophysiology of atherosclerosis and coronary artery disease
    - Sub Part 2.1.2: Stable angina: presentation and diagnosis
    - Sub Part 2.1.3: Management of stable angina
- **Part 2.2: Ischaemic Heart Disease II: Acute Coronary Syndromes**
    - Sub Part 2.2.1: Classification of acute coronary syndromes
    - Sub Part 2.2.2: Clinical presentation and diagnosis of myocardial infarction
    - Sub Part 2.2.3: Management of acute coronary syndromes
- **Part 2.3: Dilated and Hypertrophic Cardiomyopathy**
    - Sub Part 2.3.1: Dilated cardiomyopathy
    - Sub Part 2.3.2: Hypertrophic cardiomyopathy: pathophysiology and presentation
    - Sub Part 2.3.3: Management of hypertrophic cardiomyopathy
- **Part 2.4: Restrictive Cardiomyopathy and Comparison**
    - Sub Part 2.4.1: Restrictive cardiomyopathy
    - Sub Part 2.4.2: Causes of restrictive cardiomyopathy
    - Sub Part 2.4.3: Comparing the three cardiomyopathy types

Introduction

This Series turns to two of the most clinically significant categories of heart muscle disease, **ischaemic heart disease**, resulting from inadequate coronary blood supply, and **cardiomyopathy**, primary disease of the heart muscle itself, dilated, hypertrophic, and restrictive in turn. Both categories can ultimately produce heart failure through the mechanisms already established in Series 1, but each arrives at that endpoint through a genuinely distinct underlying pathological process.

The aim of this Series is to equip you with an understanding of the pathophysiology, presentation, and management of stable angina and acute coronary syndromes, and the pathophysiology, presentation, and management of dilated, hypertrophic, and restrictive cardiomyopathy, alongside the ability to compare these three cardiomyopathy types confidently.

This Series opens with **ischaemic heart disease I: chronic coronary syndromes**, moves through **ischaemic heart disease II: acute coronary syndromes** and **dilated and hypertrophic cardiomyopathy**, and closes with **restrictive cardiomyopathy and comparison**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 2.1:** describe the pathophysiology of atherosclerosis and stable angina
- **SLO 2.2:** discuss the management of stable angina
- **SLO 2.3:** classify and describe the presentation of acute coronary syndromes
- **SLO 2.4:** discuss the management of acute coronary syndromes
- **SLO 2.5:** describe dilated cardiomyopathy
- **SLO 2.6:** describe the pathophysiology, presentation, and management of hypertrophic cardiomyopathy
- **SLO 2.7:** describe restrictive cardiomyopathy and its causes
- **SLO 2.8:** compare dilated, hypertrophic, and restrictive cardiomyopathy

2.1 Ischaemic Heart Disease I: Chronic Coronary Syndromes

2.1.1 pathophysiology of atherosclerosis and coronary artery disease

A slow, progressive process narrowing the coronary lumen

Atherosclerosis, the underlying process driving coronary artery disease, develops through progressive deposition of lipid, inflammatory cells, and fibrous tissue within the arterial wall, forming a plaque that gradually narrows the vessel lumen and, once sufficiently advanced, restricts coronary blood flow, particularly during periods of increased myocardial oxygen demand such as physical exertion.

Modifiable risk factors, including smoking, hypertension, diabetes, dyslipidaemia, and physical inactivity, alongside non-modifiable factors including age, sex, and family history, together determine an individual's overall risk of developing coronary artery disease, and addressing modifiable risk factors, both before and after a diagnosis of coronary artery disease is established, remains a genuinely central component of comprehensive cardiovascular care throughout this Series.

Fill in the blank: Atherosclerotic plaque restricts coronary blood flow particularly during periods of increased myocardial oxygen ________.

2.1.2 stable angina: presentation and diagnosis

A predictable, reproducible symptom pattern

Stable angina presents with central, often crushing chest discomfort, sometimes radiating to the jaw, neck, or left arm, characteristically precipitated by exertion or emotional stress and relieved promptly by rest or sublingual nitrate, a genuinely predictable, reproducible pattern that itself carries significant diagnostic value, since a change in this established pattern, becoming more frequent, severe, or occurring at rest, raises concern for progression towards the acute coronary syndromes discussed in the following Part.

Diagnosis combines the characteristic clinical history with specific investigations, including exercise electrocardiography, functional cardiac imaging, or coronary computed tomography angiography, to confirm the presence and extent of coronary artery disease, with the specific investigation chosen depending on the patient's pre-test probability of significant coronary disease and local resource availability.

Fill in the blank: A change in the established pattern of stable angina raises concern for progression towards acute coronary ________.

2.1.3 management of stable angina

Symptom control alongside genuine risk reduction

Symptomatic management of stable angina uses sublingual nitrate for acute episodes, alongside regular anti-anginal medication, including beta-blockers and calcium channel blockers, to reduce the frequency and severity of future episodes by reducing myocardial oxygen demand, with the specific choice and combination of agents individualised according to the patient's other comorbidities and treatment tolerance.

Secondary prevention, including antiplatelet therapy, lipid-lowering medication, and comprehensive modifiable risk factor management already introduced earlier in this Part, genuinely reduces the risk of future cardiovascular events, and, for selected patients with significant coronary disease not adequately controlled with medical therapy alone, coronary revascularisation, either percutaneous coronary intervention or coronary artery bypass grafting, offers a further treatment option addressing the underlying coronary narrowing directly.

Fill in the blank: Beta-blockers and calcium channel blockers reduce anginal frequency by reducing myocardial oxygen ________.

Figure 2.1: A clinician reviewing an electrocardiogram trace while assessing a patient's chest pain.

Pilot questions 2.1

- Describe the pathophysiology of atherosclerosis. (Linked to SLO 2.1)
- List modifiable risk factors for coronary artery disease. (Linked to SLO 2.1)
- Describe the classic presentation of stable angina. (Linked to SLO 2.1)
- Explain the significance of a change in the established anginal pattern. (Linked to SLO 2.1)
- Describe the components of stable angina management. (Linked to SLO 2.2)

2.2 Ischaemic Heart Disease II: Acute Coronary Syndromes

2.2.1 classification of acute coronary syndromes

A spectrum defined by electrocardiogram and biomarker findings

Acute coronary syndrome, encompassing unstable angina, non-ST-elevation myocardial infarction, and ST-elevation myocardial infarction, results from acute plaque rupture or erosion with superimposed thrombosis, producing a spectrum of reduced coronary blood flow ranging from partial to complete occlusion, with the specific classification within this spectrum determined by electrocardiographic findings and cardiac biomarker results.

Unstable angina and **non-ST-elevation myocardial infarction** are distinguished from one another by the presence or absence of cardiac troponin elevation, reflecting genuine myocardial cell death, while **ST-elevation myocardial infarction**, identified by characteristic electrocardiographic changes, indicates complete coronary occlusion and demands the most immediate, time-critical intervention of the three categories, a classification framework directly guiding the urgency and specific management approach for each individual patient.

Fill in the blank: Unstable angina and non-ST-elevation myocardial infarction are distinguished by the presence or absence of cardiac troponin ________.

2.2.2 clinical presentation and diagnosis of myocardial infarction

Recognising the presentation, then confirming it objectively

Myocardial infarction typically presents with severe, prolonged chest pain similar in character to angina but more severe and not relieved by rest or nitrate, often accompanied by breathlessness, sweating, nausea, and a sense of impending doom, though presentation can genuinely be atypical, particularly in women, older adults, and patients with diabetes, where symptoms may be less classically dramatic or even entirely absent.

Diagnosis combines this clinical presentation with **serial electrocardiography**, identifying the specific pattern of changes reflecting the affected coronary territory, and **cardiac troponin measurement**, a genuinely sensitive and specific marker of myocardial cell death, with serial troponin measurement over several hours often required to confirm or exclude the diagnosis with confidence given the time required for troponin to rise following the onset of myocardial injury.

Fill in the blank: Serial troponin measurement over several hours is often required given the time required for troponin to ________.

2.2.3 management of acute coronary syndromes

Restoring blood flow and preventing further events

Management of **ST-elevation myocardial infarction** prioritises urgent coronary reperfusion, ideally through **primary percutaneous coronary intervention**, mechanically opening the occluded artery, performed within a genuinely narrow, time-critical window, with thrombolytic therapy used where primary percutaneous coronary intervention is not rapidly available, since the benefit of reperfusion therapy diminishes progressively the longer treatment is delayed, mirroring the same time-dependent principle already discussed for stroke thrombolysis in an earlier course.

Management of **non-ST-elevation acute coronary syndrome** combines antiplatelet and anticoagulant therapy with risk stratification determining the urgency of coronary angiography, and comprehensive **secondary prevention**, including antiplatelet therapy, lipid-lowering medication, beta-blockers, and structured cardiac rehabilitation, genuinely reduces the risk of subsequent cardiovascular events across the whole acute coronary syndrome spectrum regardless of the specific initial presentation.

Fill in the blank: The benefit of reperfusion therapy for ST-elevation myocardial infarction diminishes progressively the longer treatment is ________.

Table 2.1: Comparing the acute coronary syndrome spectrum

Category	Troponin	ECG finding
Unstable angina	Negative	May show ischaemic changes without ST elevation
Non-ST-elevation myocardial infarction	Positive	No ST elevation

ST-elevation myocardial infarction	Positive	ST elevation, indicating complete occlusion

Pilot questions 2.2

1. List the three categories of acute coronary syndrome. (Linked to SLO 2.3)
2. Distinguish unstable angina from non-ST-elevation myocardial infarction. (Linked to SLO 2.3)
3. Describe the typical presentation of myocardial infarction. (Linked to SLO 2.3)
4. Explain why serial troponin measurement is often required. (Linked to SLO 2.3)
5. Describe the priority management approach for ST-elevation myocardial infarction. (Linked to SLO 2.4)

2.3 Dilated and Hypertrophic Cardiomyopathy

2.3.1 dilated cardiomyopathy

A stretched, weakened ventricle

Dilated cardiomyopathy, characterised by ventricular dilatation and impaired systolic function in the absence of coronary artery disease, hypertension, or valvular disease sufficient to explain the degree of dysfunction observed, has a genuinely wide range of underlying causes, including genetic mutations, viral myocarditis, alcohol excess, and certain chemotherapy agents, though a specific cause remains genuinely unidentified in a meaningful proportion of cases despite thorough investigation.

Clinical presentation mirrors the systolic heart failure already discussed in Series 1, since dilated cardiomyopathy is, in essence, one of the primary causes of that presentation, and management similarly follows the same general heart failure management principles, addressing the underlying cause where genuinely identifiable and treatable, such as abstaining from alcohol in alcohol-related cardiomyopathy, alongside standard evidence-based heart failure pharmacotherapy.

Fill in the blank: Dilated cardiomyopathy is characterised by ventricular dilatation and impaired systolic ________.

2.3.2 hypertrophic cardiomyopathy: pathophysiology and presentation

A thickened, genetically determined ventricular wall

Hypertrophic cardiomyopathy, a genetic condition resulting from mutations in genes encoding cardiac sarcomere proteins, produces inappropriate left ventricular hypertrophy, often asymmetrically affecting the interventricular septum, in the absence of an identifiable secondary cause such as hypertension sufficient to explain the degree of hypertrophy observed, and represents a genuinely important cause of sudden cardiac death in young, otherwise apparently healthy individuals, particularly athletes.

Presentation ranges from entirely asymptomatic, identified incidentally or through family screening given the genetic basis of the condition, through breathlessness and chest pain, to syncope and, in the most severe presentations, sudden cardiac death, and, in a proportion of affected individuals, the hypertrophied septum can produce **left ventricular outflow tract obstruction**, a dynamic obstruction to blood flow worsening with exertion and certain medications, a specific pathophysiological feature genuinely relevant to management choices discussed in the following sub-Part.

Fill in the blank: Hypertrophic cardiomyopathy often asymmetrically affects the interventricular ________.

2.3.3 management of hypertrophic cardiomyopathy

Symptom control and, genuinely importantly, sudden death prevention

Management addresses both symptomatic control, using beta-blockers or calcium channel blockers to reduce left ventricular outflow tract obstruction and improve symptoms, and **risk stratification for sudden cardiac death**, assessing specific clinical and family history features to identify patients who would genuinely benefit from an **implantable cardioverter-defibrillator**, a device capable of detecting and treating a life-threatening ventricular arrhythmia before it proves fatal.

Family screening, given the genetic basis of hypertrophic cardiomyopathy, is a genuinely essential component of comprehensive management, identifying affected first-degree relatives who may themselves be asymptomatic but at genuine risk, allowing appropriate counselling, ongoing surveillance, and, where indicated, the same risk stratification and preventive measures already discussed for the originally identified patient, extending the genuine clinical benefit of diagnosis beyond the single individual initially presenting.

Fill in the blank: Family screening identifies affected first-degree relatives who may themselves be asymptomatic but at genuine ________.

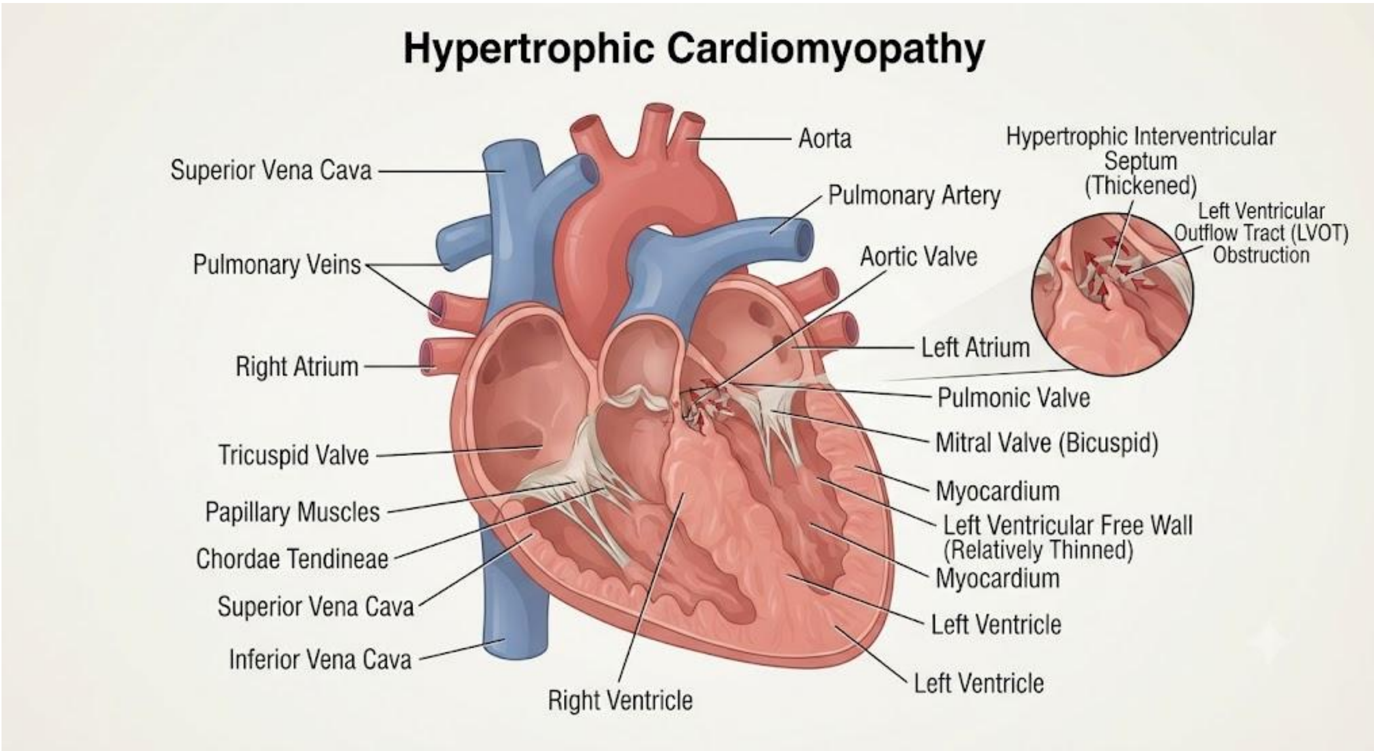

Figure 2.2: A labelled diagram of asymmetric septal hypertrophy in hypertrophic cardiomyopathy.

Pilot questions 2.3

1. List causes of dilated cardiomyopathy. (Linked to SLO 2.5)
2. Describe the clinical presentation of dilated cardiomyopathy. (Linked to SLO 2.5)
3. Describe the underlying pathophysiology of hypertrophic cardiomyopathy. (Linked to SLO 2.6)
4. Describe left ventricular outflow tract obstruction. (Linked to SLO 2.6)
5. Explain the purpose of family screening in hypertrophic cardiomyopathy. (Linked to SLO 2.6)

2.4 Restrictive Cardiomyopathy and Comparison

2.4.1 restrictive cardiomyopathy

A stiff, poorly compliant ventricle despite normal wall thickness

Restrictive cardiomyopathy, the least common of the three major cardiomyopathy types, is characterised by impaired ventricular filling from increased myocardial stiffness, with the ventricular walls typically normal or only mildly thickened, unlike the marked hypertrophy characteristic of hypertrophic cardiomyopathy, and systolic function typically preserved, at least in earlier stages of the disease, producing a diastolic pattern of dysfunction genuinely similar to that already discussed in Series 1.

Presentation reflects this impaired filling, with breathlessness, fatigue, and features of both left and right-sided congestion, often disproportionately severe right-sided features including significant peripheral oedema and ascites relative to the degree of breathlessness, a specific clinical pattern that, alongside supporting investigation, helps distinguish restrictive cardiomyopathy from the other cardiomyopathy types already discussed in this Series.

Fill in the blank: Restrictive cardiomyopathy is characterised by impaired ventricular filling from increased myocardial ________.

2.4.2 causes of restrictive cardiomyopathy

Infiltrative and other processes stiffening the myocardium

Amyloidosis, deposition of abnormal amyloid protein within the myocardium, is among the most commonly encountered causes of restrictive cardiomyopathy, alongside other infiltrative conditions such as sarcoidosis and haemochromatosis, iron deposition within the myocardium, and certain forms of endomyocardial fibrosis, each producing the characteristic restrictive physiology through a genuinely distinct underlying pathological process affecting the myocardium's normal compliance.

Identifying the specific underlying cause of restrictive cardiomyopathy genuinely matters clinically, since certain causes, particularly amyloidosis, now have specific, disease-modifying treatment options available, making thorough investigation, including cardiac magnetic resonance imaging and, in selected cases, myocardial biopsy, a genuinely worthwhile diagnostic pursuit rather than simply confirming a restrictive pattern in general terms alone.

Fill in the blank: Amyloidosis is deposition of abnormal amyloid ________ within the myocardium.

2.4.3 comparing the three cardiomyopathy types

Bringing the whole spectrum together

Comparing dilated, hypertrophic, and restrictive cardiomyopathy across their structural, functional, and clinical features provides a genuinely useful framework for confidently distinguishing between them, since **dilated cardiomyopathy** shows an enlarged, poorly contracting ventricle, **hypertrophic cardiomyopathy** shows a thickened, often asymmetrically hypertrophied ventricle with preserved or even supranormal contractile function, and **restrictive cardiomyopathy** shows normal or near-normal wall thickness with impaired filling.

Echocardiography, alongside cardiac magnetic resonance imaging in selected cases, remains the key investigation distinguishing these three cardiomyopathy types, directly visualising ventricular size, wall thickness, and functional pattern, and confidently applying this comparative framework, closing this Series on the theme of structured, comparative clinical reasoning that has run throughout this whole programme of cardiovascular study, genuinely supports accurate diagnosis and appropriately targeted management.

Fill in the blank: Hypertrophic cardiomyopathy shows preserved or even ________ contractile function despite the thickened wall.

Table 2.2: Comparing dilated, hypertrophic, and restrictive cardiomyopathy

Feature	Dilated	Hypertrophic and restrictive
Ventricular size	Enlarged	Normal or small cavity
Wall thickness	Normal or thin	Thickened in hypertrophic; normal in restrictive
Systolic function	Reduced	Preserved in both

Pilot questions 2.4

1. Describe restrictive cardiomyopathy and its typical wall thickness. (Linked to SLO 2.7)
2. List causes of restrictive cardiomyopathy. (Linked to SLO 2.7)
3. Explain why identifying the specific cause of restrictive cardiomyopathy matters clinically. (Linked to SLO 2.7)
4. Compare the ventricular size and wall thickness across the three cardiomyopathy types. (Linked to SLO 2.8)
5. Describe the key investigation distinguishing the three cardiomyopathy types. (Linked to SLO 2.8)

Series Summary

This Series covered ischaemic heart disease and the three major cardiomyopathy types. Part 2.1 covered atherosclerosis and the presentation and management of stable angina, while Part 2.2 turned to the classification, presentation, and management of acute coronary syndromes. Part 2.3 covered dilated and hypertrophic cardiomyopathy, and Part 2.4 closed with restrictive cardiomyopathy and a comparison across all three types.

You should now be able to describe and manage ischaemic heart disease and describe and compare the three cardiomyopathy types covered across all four Parts of this Series, meeting the outcomes set for this Series. Series 3 turns next to peripheral vascular disease and venous thromboembolism.

Glossary of Terms

1. **Acute coronary syndrome:** unstable angina, non-ST-elevation, and ST-elevation myocardial infarction collectively.
2. **Amyloidosis:** deposition of abnormal amyloid protein within tissue, a cause of restrictive cardiomyopathy.
3. **Atherosclerosis:** progressive lipid and fibrous plaque deposition within the arterial wall.
4. **Dilated cardiomyopathy:** ventricular dilatation and impaired systolic function without an explanatory secondary cause.
5. **Hypertrophic cardiomyopathy:** a genetic condition causing inappropriate left ventricular hypertrophy.
6. **Implantable cardioverter-defibrillator:** a device detecting and treating life-threatening ventricular arrhythmia.
7. **Left ventricular outflow tract obstruction:** dynamic obstruction to blood flow from septal hypertrophy.
8. **Primary percutaneous coronary intervention:** urgent mechanical reperfusion of an occluded coronary artery.
9. **Restrictive cardiomyopathy:** impaired ventricular filling from increased myocardial stiffness.
10. **ST-elevation myocardial infarction:** acute coronary syndrome with ECG ST elevation indicating complete occlusion.
11. **Stable angina:** predictable, exertional chest pain relieved by rest or nitrate.
12. **Troponin:** a sensitive and specific biomarker of myocardial cell death.

Concept Check Questions [Series 2]

Objective questions

- Atherosclerotic plaque restricts coronary blood flow particularly during periods of increased myocardial oxygen ________. (Linked to SLO 2.1)
- Unstable angina and non-ST-elevation myocardial infarction are distinguished by the presence or absence of cardiac troponin ________. (Linked to SLO 2.3)
- Dilated cardiomyopathy is characterised by ventricular dilatation and impaired systolic ________. (Linked to SLO 2.5)
- Hypertrophic cardiomyopathy often asymmetrically affects the interventricular ________. (Linked to SLO 2.6)
- Restrictive cardiomyopathy is characterised by impaired ventricular filling from increased myocardial ________. (Linked to SLO 2.7)

Short-answer questions

1. Describe the classic presentation of stable angina. (Linked to SLO 2.1)
2. Describe the typical presentation of myocardial infarction. (Linked to SLO 2.3)
3. Describe the priority management approach for ST-elevation myocardial infarction. (Linked to SLO 2.4)
4. Describe left ventricular outflow tract obstruction. (Linked to SLO 2.6)
5. Compare the ventricular size and wall thickness across the three cardiomyopathy types. (Linked to SLO 2.8)

Long-answer questions

6. Discuss the pathophysiology, presentation, and management of stable angina. (Linked to SLO 2.1, 2.2)
7. Describe the classification, presentation, and management of acute coronary syndromes. (Linked to SLO 2.3, 2.4)
8. Discuss the pathophysiology, presentation, and management of dilated and hypertrophic cardiomyopathy. (Linked to SLO 2.5, 2.6)
9. Describe restrictive cardiomyopathy and its causes. (Linked to SLO 2.7)
10. Compare dilated, hypertrophic, and restrictive cardiomyopathy. (Linked to SLO 2.8)

Answers to Concept Check Questions [Series 2]

Objective questions

11. Demand.
12. Elevation.
13. Function.
14. Septum.
15. Stiffness.

Short-answer questions

16. Stable angina presents with central chest discomfort precipitated by exertion and relieved by rest or nitrate.
17. Myocardial infarction presents with severe, prolonged chest pain not relieved by rest, with breathlessness and sweating.
18. Priority is urgent reperfusion, ideally primary percutaneous coronary intervention, within a narrow time-critical window.
19. It is dynamic obstruction to blood flow from the hypertrophied septum, worsening with exertion.
20. Dilated shows an enlarged cavity with thin walls; hypertrophic shows thickened walls with small cavity; restrictive shows normal walls with normal or small cavity.

Long-answer questions

21. **Basic response:** Atherosclerosis narrows coronary vessels, producing exertional chest pain relieved by rest; management combines symptomatic anti-anginal medication with secondary prevention and, where indicated, revascularisation.

    Value-added response: A value-added response notes that a change in the established anginal pattern raises concern for progression to acute coronary syndrome.

22. **Basic response:** Acute coronary syndrome spans unstable angina, non-ST-elevation, and ST-elevation myocardial infarction, distinguished by troponin and ECG findings; management prioritises urgent reperfusion for ST-elevation infarction and risk-stratified treatment for the others.

    Value-added response: A value-added response notes that comprehensive secondary prevention reduces subsequent cardiovascular events across the whole spectrum.

23. **Basic response:** Dilated cardiomyopathy causes ventricular dilatation and reduced contractility, managed as standard heart failure; hypertrophic cardiomyopathy is genetic septal hypertrophy managed with symptom control and sudden death risk stratification.

    Value-added response: A value-added response notes that family screening is essential in hypertrophic cardiomyopathy given its genetic basis.

24. **Basic response:** Restrictive cardiomyopathy causes impaired filling from increased stiffness with normal wall thickness, commonly from amyloidosis or other infiltrative conditions.

    Value-added response: A value-added response notes that identifying the specific cause matters given the availability of disease-modifying treatment for certain causes such as amyloidosis.

25. **Basic response:** Dilated cardiomyopathy shows an enlarged, poorly contracting ventricle; hypertrophic shows thickened walls with preserved function; restrictive shows normal walls with impaired filling.

    Value-added response: A value-added response notes that echocardiography remains the key investigation distinguishing all three types.

Answers to Pilot Questions [Series 2]

Part 2.1

1. Atherosclerosis develops through progressive lipid, inflammatory cell, and fibrous tissue deposition narrowing the arterial lumen.
2. Modifiable factors include smoking, hypertension, diabetes, dyslipidaemia, and physical inactivity.
3. Stable angina presents with central chest discomfort precipitated by exertion and relieved by rest or nitrate.
4. A changing pattern raises concern for progression towards acute coronary syndrome.
5. Management includes symptomatic anti-anginal medication, secondary prevention, and revascularisation for selected patients.

Part 2.2

6. The categories are unstable angina, non-ST-elevation myocardial infarction, and ST-elevation myocardial infarction.
7. Unstable angina has negative troponin, while non-ST-elevation myocardial infarction has positive troponin.
8. Myocardial infarction presents with severe, prolonged chest pain not relieved by rest, with breathlessness and sweating.
9. Troponin takes time to rise following myocardial injury, so serial measurement improves diagnostic accuracy.
10. Priority is urgent reperfusion, ideally primary percutaneous coronary intervention, within a narrow time window.

Part 2.3

11. Causes include genetic mutations, viral myocarditis, alcohol excess, and certain chemotherapy agents.
12. Presentation mirrors systolic heart failure, since dilated cardiomyopathy is a primary cause of that presentation.
13. Hypertrophic cardiomyopathy results from sarcomere gene mutations causing inappropriate left ventricular hypertrophy.
14. It is dynamic obstruction to blood flow from the hypertrophied septum, worsening with exertion and certain medications.
15. Family screening identifies asymptomatic but at-risk relatives given the condition's genetic basis.

Part 2.4

1. Restrictive cardiomyopathy has impaired filling from increased stiffness, with normal or only mildly thickened walls.
2. Causes include amyloidosis, sarcoidosis, haemochromatosis, and endomyocardial fibrosis.
3. Certain causes, particularly amyloidosis, now have specific, disease-modifying treatment options available.
4. Dilated shows enlarged, thin-walled ventricles; hypertrophic shows thickened walls; restrictive shows normal walls with small or normal cavity.
5. Echocardiography, alongside cardiac MRI in selected cases, remains the key distinguishing investigation.

References and Attributions [Series 2]

1. Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
2. Libby, P., Bonow, R. O., Mann, D. L., Tomaselli, G. F., & Braunwald, E. (2021). Braunwald's Heart Disease (12th ed.). Elsevier.
3. European Society of Cardiology (2023). Guidelines for the Management of Acute Coronary Syndromes. European Heart Journal.
4. European Society of Cardiology (2014). Guidelines on Diagnosis and Management of Hypertrophic Cardiomyopathy. European Heart Journal.

Figures, Plates, Tables, and Boxes

1. Figure 2.1: A clinician reviewing an electrocardiogram trace while assessing a patient's chest pain. AI-generated using the prompt: "Create a realistic clinical illustration titled Assessing Chest Pain with Electrocardiography, showing a clinician holding an electrocardiogram printout while speaking with a seated patient describing chest discomfort, consultation room setting. Clean clinical illustration style, no text."
2. Table 2.1: Comparing the acute coronary syndrome spectrum. AI-generated using the prompt: "Create a clean reference table titled Comparing the Acute Coronary Syndrome Spectrum, listing category, troponin, and ECG finding. Simple clinical reference table style with outside border."
3. Figure 2.2: A labelled diagram of asymmetric septal hypertrophy in hypertrophic cardiomyopathy. AI-generated using the prompt: "Create a clean, accurate labelled anatomical diagram titled Hypertrophic Cardiomyopathy, showing a cross-section of the heart with a labelled, thickened interventricular septum disproportionate to the left ventricular free wall. Educational anatomical diagram style, no photographic content."
4. Table 2.2: Comparing dilated, hypertrophic, and restrictive cardiomyopathy. AI-generated using the prompt: "Create a clean reference table titled Comparing Dilated, Hypertrophic, and Restrictive Cardiomyopathy, listing feature, dilated, and hypertrophic and restrictive. Simple clinical reference table style with outside border."

Course code: MED 606

Course title: Cardiology II

Course credit load: 2 Units

Series 3: Peripheral Vascular Disease and Venous Thromboembolism

- **Part 3.1: Acute and Chronic Arterial Disease**
  - Sub Part 3.1.1: Chronic peripheral arterial disease
  - Sub Part 3.1.2: Acute limb ischaemia
  - Sub Part 3.1.3: Investigation and management of peripheral arterial disease
- **Part 3.2: Embolic Disease**
  - Sub Part 3.2.1: Sources and mechanisms of arterial embolism
  - Sub Part 3.2.2: Clinical presentation of embolic disease
  - Sub Part 3.2.3: Management of embolic disease
- **Part 3.3: Deep Vein Thrombosis**
  - Sub Part 3.3.1: Pathophysiology and risk factors for venous thromboembolism
  - Sub Part 3.3.2: Clinical presentation and diagnosis of deep vein thrombosis
  - Sub Part 3.3.3: Management of deep vein thrombosis
- **Part 3.4: Pulmonary Embolism**
  - Sub Part 3.4.1: Pathophysiology and clinical presentation of pulmonary embolism
  - Sub Part 3.4.2: Diagnosis of pulmonary embolism
  - Sub Part 3.4.3: Management of pulmonary embolism

Introduction

This Series moves beyond the heart itself to the vascular system it supplies, working through **peripheral vascular disease**, both arterial and embolic, and **venous thromboembolism**, spanning deep vein thrombosis and its most feared complication, pulmonary embolism. Each of these conditions demands the same urgent, structured recognition already emphasised throughout this course, since delayed diagnosis in any of them can genuinely mean the difference between a limb, or a life, saved or lost.

The aim of this Series is to equip you with an understanding of chronic peripheral arterial disease, acute limb ischaemia, and their investigation and management, arterial embolism and its presentation and management, and the pathophysiology, presentation, diagnosis, and management of deep vein thrombosis and pulmonary embolism.

This Series opens with **acute and chronic arterial disease**, moves through **embolic disease** and **deep vein thrombosis**, and closes with **pulmonary embolism**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 3.1:** describe chronic peripheral arterial disease and acute limb ischaemia
- **SLO 3.2:** discuss the investigation and management of peripheral arterial disease
- **SLO 3.3:** describe the sources and clinical presentation of arterial embolism
- **SLO 3.4:** discuss the management of embolic disease
- **SLO 3.5:** describe the pathophysiology, risk factors, and clinical presentation of deep vein thrombosis
- **SLO 3.6:** discuss the diagnosis and management of deep vein thrombosis
- **SLO 3.7:** describe the pathophysiology, presentation, and diagnosis of pulmonary embolism
- **SLO 3.8:** discuss the management of pulmonary embolism

3.1 Acute and Chronic Arterial Disease

3.1.1 chronic peripheral arterial disease

Gradually reduced blood flow to the limbs

Chronic peripheral arterial disease, resulting from atherosclerotic narrowing of the peripheral arteries, most commonly affecting the lower limbs, presents with **intermittent claudication**, cramping pain in the calf, thigh, or buttock brought on by walking a consistent distance and relieved promptly by rest, and, as disease progresses, **critical limb ischaemia**, presenting with rest pain, typically worse at night, and non-healing ulceration or tissue loss.

Examination reveals reduced or absent peripheral pulses, a cool limb with delayed capillary refill, and, in chronic severe disease, hair loss and shiny, thin skin over the affected limb, findings shared in principle with the earlier course covering ulcer management, since chronic peripheral arterial disease shares the same underlying atherosclerotic risk factors already discussed in relation to coronary artery disease in an earlier Series of this course.

Fill in the blank: Intermittent claudication is cramping pain brought on by walking a consistent distance and relieved promptly by ________.

3.1.2 acute limb ischaemia

A genuine limb-threatening surgical emergency

Acute limb ischaemia, sudden interruption of arterial blood supply to a limb, presents classically with the six Ps: pain, pallor, pulselessness, paraesthesia, paralysis, and perishingly cold, a presentation constituting a genuine surgical emergency given the narrow window during which the affected limb remains genuinely salvageable before irreversible tissue and nerve damage occurs.

Acute limb ischaemia results from either **acute thrombosis** occurring on a background of pre-existing chronic arterial disease, or **embolism**, discussed in more detail in the following Part of this Series, a genuinely important distinction since it directly influences both the specific management approach and the likelihood of significant underlying chronic arterial disease elsewhere requiring further evaluation once the acute presentation has been addressed.

Fill in the blank: Acute limb ischaemia presents classically with the six Ps, including pain, pallor, and ________.

3.1.3 investigation and management of peripheral arterial disease

Confirming severity and choosing the appropriate treatment intensity

Ankle-brachial pressure index, comparing systolic pressure at the ankle and arm, remains the key bedside investigation for chronic peripheral arterial disease, with duplex ultrasound, CT angiography, or MR angiography, already discussed in an earlier course, providing further anatomical detail where intervention is being planned, following the same investigative principles already established for vascular disease elsewhere in this programme.

Management of chronic disease combines risk factor modification and supervised exercise therapy for milder disease with revascularisation, open or endovascular, for more significant or critical limb ischaemia, while acute limb ischaemia requires urgent revascularisation, whether surgical embolectomy, thrombolysis, or bypass, given the genuinely narrow, time-critical window already discussed in the previous sub-Part before irreversible limb loss occurs.

Fill in the blank: Management of milder chronic peripheral arterial disease combines risk factor modification with supervised exercise ________.

Figure 3.1: A clinician examining a patient's limb for pallor and pulse in suspected acute limb ischaemia.

Pilot questions 3.1

- Describe intermittent claudication and critical limb ischaemia. (Linked to SLO 3.1)
- List the examination findings of chronic peripheral arterial disease. (Linked to SLO 3.1)
- List the six Ps of acute limb ischaemia. (Linked to SLO 3.1)
- Distinguish thrombotic from embolic acute limb ischaemia. (Linked to SLO 3.1)
- Describe the management approach for chronic and acute peripheral arterial disease. (Linked to SLO 3.2)

3.2 Embolic Disease

3.2.1 sources and mechanisms of arterial embolism

A clot travelling from a distant source to lodge downstream

Arterial embolism, obstruction of an artery by material travelling from a distant source, most commonly originates from the heart, particularly in the presence of **atrial fibrillation**, already discussed in an earlier Series of this course given the stasis and clot formation it produces within the fibrillating atria, or from a mural thrombus following myocardial infarction, or, less commonly, from a proximal atherosclerotic plaque itself.

Emboli typically lodge at points of arterial narrowing or bifurcation, most commonly affecting the lower limb, though embolisation to the mesenteric or cerebral circulation, already discussed in relation to stroke in an earlier course, also occurs and carries its own genuinely distinct, often devastating clinical consequences depending on the specific territory affected by the embolic occlusion.

Fill in the blank: Emboli typically lodge at points of arterial narrowing or ________.

3.2.2 clinical presentation of embolic disease

A sudden, often dramatic onset, without preceding warning

Embolic acute limb ischaemia typically presents with a more sudden, dramatic onset than thrombotic acute limb ischaemia, often in a patient without a preceding history of claudication, since the affected limb has not had the opportunity to develop the collateral circulation that chronic, gradually progressive atherosclerotic narrowing typically allows to form, a genuinely important distinguishing clinical clue between the two underlying mechanisms.

A source of embolism should be actively sought in any patient presenting with acute limb ischaemia without a preceding history of claudication, including cardiac rhythm assessment for atrial fibrillation and, where indicated, echocardiography to identify a cardiac source, since identifying and appropriately treating the underlying source genuinely reduces the risk of a further, potentially even more devastating embolic event in the future.

Fill in the blank: The affected limb in embolic ischaemia has not had the opportunity to develop ________ circulation.

3.2.3 management of embolic disease

Removing the embolus and addressing its source

Acute management of embolic limb ischaemia involves urgent **embolectomy**, surgically removing the embolus, or catheter-directed thrombolysis, restoring blood flow before irreversible tissue damage occurs, following the same time-critical principle already established for acute limb ischaemia generally in the previous Part of this Series, with the specific technique chosen depending on the location and extent of the occlusion and the patient's overall clinical status.

Following successful treatment of the acute embolic event, **long-term anticoagulation** is generally indicated where a cardiac source such as atrial fibrillation has been identified, to prevent future embolic events, and this genuinely essential secondary prevention step, addressing the underlying source rather than simply the immediate embolic complication, closes the loop on comprehensive management of this specific vascular emergency.

Fill in the blank: Long-term anticoagulation is generally indicated where a cardiac source such as atrial fibrillation has been ________.

Table 3.1: Comparing thrombotic and embolic acute limb ischaemia

Feature	Thrombotic ischaemia	Embolic ischaemia
Onset	Often more gradual	Sudden, dramatic
Preceding claudication	Often present	Often absent
Collateral circulation	Often developed	Not developed

Pilot questions 3.2

1. List common sources of arterial embolism. (Linked to SLO 3.3)
2. Describe why emboli typically lodge at points of arterial narrowing or bifurcation. (Linked to SLO 3.3)
3. Explain why embolic limb ischaemia often presents without preceding claudication. (Linked to SLO 3.3)
4. Describe the acute management of embolic limb ischaemia. (Linked to SLO 3.4)
5. Explain the role of long-term anticoagulation following embolic limb ischaemia. (Linked to SLO 3.4)

3.3 Deep Vein Thrombosis

3.3.1 pathophysiology and risk factors for venous thromboembolism

Three converging factors driving clot formation

Deep vein thrombosis, clot formation within the deep venous system, most commonly affecting the lower limb, arises through the interplay of **Virchow's triad**, venous stasis, endothelial injury, and hypercoagulability, three converging factors that together create the conditions for pathological clot formation within an otherwise normally flowing venous system.

Risk factors include recent surgery or immobility, contributing to venous stasis, malignancy and certain inherited thrombophilias, contributing to hypercoagulability, and pregnancy and oestrogen-containing medication, contributing to a combination of these factors, and identifying a patient's specific risk factor profile genuinely informs both the assessed probability of the diagnosis and, once confirmed, the appropriate duration of subsequent anticoagulation treatment.

Fill in the blank: Virchow's triad comprises venous stasis, endothelial injury, and ________.

3.3.2 clinical presentation and diagnosis of deep vein thrombosis

A genuinely unreliable clinical picture requiring objective confirmation

Deep vein thrombosis presents with unilateral limb swelling, pain, warmth, and erythema, though clinical examination findings alone are notoriously unreliable for confidently confirming or excluding the diagnosis, meaning a structured **clinical probability score**, combining risk factors and examination findings, is used alongside **D-dimer** blood testing and, where indicated, **compression ultrasound** to confirm or exclude the diagnosis with genuine confidence.

A low clinical probability combined with a negative D-dimer result can safely exclude the diagnosis without further imaging, given the test's high sensitivity, though its correspondingly limited specificity means a positive D-dimer requires further imaging confirmation with compression ultrasound rather than being treated as diagnostic in isolation, a structured, sequential diagnostic pathway genuinely improving both diagnostic accuracy and resource efficiency.

Fill in the blank: A positive D-dimer requires further imaging confirmation given the test's limited ________.

3.3.3 management of deep vein thrombosis

Anticoagulation to prevent extension and embolisation

Anticoagulation, typically with a direct oral anticoagulant given its favourable convenience and safety profile compared with older agents, forms the mainstay of deep vein thrombosis treatment, preventing clot extension and reducing the genuine risk of embolisation to the lungs, the pulmonary embolism discussed in detail in the following Part of this Series, with treatment duration determined by whether the episode was provoked by an identifiable, transient risk factor or occurred without a clear precipitant.

Compression stockings and mobilisation, once anticoagulation has been established, support symptomatic recovery and may reduce the risk of the long-term complication of **post-thrombotic syndrome**, chronic limb swelling, pain, and skin changes following deep vein thrombosis, a genuinely important consideration underlining why deep vein thrombosis, despite often appearing to resolve clinically, can carry lasting implications for the affected limb well beyond the acute treatment period.

Fill in the blank: Post-thrombotic syndrome describes chronic limb swelling, pain, and skin changes following deep vein ________.

Figure 3.2: A clinician comparing limb circumference during deep vein thrombosis examination.

Pilot questions 3.3

1. Describe Virchow's triad. (Linked to SLO 3.5)
2. List risk factors for deep vein thrombosis. (Linked to SLO 3.5)
3. Describe the diagnostic pathway combining clinical probability, D-dimer, and ultrasound. (Linked to SLO 3.6)
4. Explain why a positive D-dimer requires further imaging confirmation. (Linked to SLO 3.6)
5. Describe post-thrombotic syndrome. (Linked to SLO 3.6)

3.4 Pulmonary Embolism

3.4.1 pathophysiology and clinical presentation of pulmonary embolism

A clot lodging in the pulmonary circulation

Pulmonary embolism, obstruction of the pulmonary arterial circulation by embolised material, most commonly originating from a deep vein thrombosis already discussed in the previous Part of this Series, presents with breathlessness, pleuritic chest pain, and, in more significant cases, haemoptysis and haemodynamic compromise, with the specific severity of presentation reflecting the extent and location of the pulmonary vascular obstruction.

Massive pulmonary embolism, causing significant haemodynamic compromise through acute right ventricular strain and failure, presents with hypotension and, in the most severe cases, cardiac arrest, a genuine medical emergency requiring immediate recognition and urgent treatment, in genuine contrast to smaller, haemodynamically insignificant pulmonary emboli, which can present with more subtle, easily overlooked symptoms.

Fill in the blank: Massive pulmonary embolism causes significant haemodynamic compromise through acute right ventricular ________.

3.4.2 diagnosis of pulmonary embolism

A similar structured pathway to deep vein thrombosis

Diagnosis of pulmonary embolism follows a similarly structured approach to deep vein thrombosis, combining a **clinical probability score** with D-dimer testing in patients with low or intermediate probability, and **CT pulmonary angiography**, directly visualising the pulmonary arterial tree to confirm or exclude embolic obstruction, remains the definitive investigation where clinical probability is high or D-dimer testing is positive.

In patients with significant renal impairment or contrast allergy precluding CT pulmonary angiography, **ventilation-perfusion scanning**, comparing lung ventilation with blood flow to identify a characteristic mismatch pattern, offers an alternative diagnostic approach, and, in the massive pulmonary embolism scenario already discussed, urgent bedside echocardiography can provide immediate supportive evidence of right ventricular strain even before definitive imaging can be arranged.

Fill in the blank: CT pulmonary angiography directly visualises the pulmonary arterial tree to confirm or exclude embolic ________.

3.4.3 management of pulmonary embolism

Anticoagulation, and, in the most severe cases, urgent reperfusion

Management of haemodynamically stable pulmonary embolism mirrors deep vein thrombosis management, with **anticoagulation** as the mainstay treatment preventing further clot propagation and allowing the body's own fibrinolytic system to gradually resolve the existing embolic burden over time, with treatment duration again determined by whether the episode was provoked or unprovoked, following the same principle already established for deep vein thrombosis.

Massive pulmonary embolism with haemodynamic compromise requires urgent **thrombolytic therapy**, or, where thrombolysis is contraindicated, surgical or catheter-based embolectomy, to rapidly reduce the pulmonary vascular obstruction and reverse the associated right ventricular strain, a genuinely time-critical intervention closing this Series, and the entire course, on the same theme of urgent, structured recognition and treatment that has run throughout the whole of this cardiovascular programme.

Fill in the blank: Massive pulmonary embolism with haemodynamic compromise requires urgent ________ therapy or embolectomy.

Table 3.2: Comparing stable and massive pulmonary embolism

Feature	Stable pulmonary embolism	Massive pulmonary embolism
Haemodynamic status	Preserved	Compromised, hypotension
First-line treatment	Anticoagulation	Thrombolysis or embolectomy
Urgency	Prompt but not immediate	Genuine medical emergency

Pilot questions 3.4

1. Describe the typical presentation of pulmonary embolism. (Linked to SLO 3.7)
2. Describe massive pulmonary embolism and its clinical significance. (Linked to SLO 3.7)
3. Describe the diagnostic pathway for suspected pulmonary embolism. (Linked to SLO 3.7)
4. List an alternative to CT pulmonary angiography for patients unable to have contrast imaging. (Linked to SLO 3.7)
5. Describe the management of stable versus massive pulmonary embolism. (Linked to SLO 3.8)

Series Summary

This Series covered peripheral vascular disease and venous thromboembolism. Part 3.1 covered chronic peripheral arterial disease and acute limb ischaemia, while Part 3.2 turned to arterial embolism, its sources, presentation, and management. Part 3.3 covered the pathophysiology, presentation, diagnosis, and management of deep vein thrombosis, and Part 3.4 closed with pulmonary embolism.

You should now be able to describe and manage peripheral arterial disease, embolic disease, deep vein thrombosis, and pulmonary embolism across all four Parts of this Series, meeting the outcomes set for this Series. Series 4 turns next to cardiovascular infections, congenital abnormalities, and valvular disease.

Glossary of Terms

1. **Acute limb ischaemia:** sudden interruption of arterial blood supply to a limb, presenting with the six Ps.
2. **Anticoagulation:** medication reducing blood clotting, the mainstay treatment for venous thromboembolism.
3. **Critical limb ischaemia:** severe peripheral arterial disease presenting with rest pain and tissue loss.
4. **D-dimer:** a blood test used with clinical probability to help exclude venous thromboembolism.

5. **Deep vein thrombosis:** clot formation within the deep venous system, most commonly of the lower limb.
6. **Embolectomy:** surgical removal of an embolus from an obstructed artery.
7. **Intermittent claudication:** cramping limb pain brought on by walking and relieved by rest.
8. **Massive pulmonary embolism:** pulmonary embolism causing significant haemodynamic compromise.
9. **Post-thrombotic syndrome:** chronic limb swelling, pain, and skin changes following deep vein thrombosis.
10. **Pulmonary embolism:** obstruction of the pulmonary arterial circulation by embolised material.
11. **Ventilation-perfusion scanning:** an imaging alternative for suspected pulmonary embolism avoiding contrast.
12. **Virchow's triad:** venous stasis, endothelial injury, and hypercoagulability, driving thrombosis.

Concept Check Questions [Series 3]

Objective questions

- Intermittent claudication is cramping pain brought on by walking a consistent distance and relieved promptly by ________. (Linked to SLO 3.1)
- Emboli typically lodge at points of arterial narrowing or ________. (Linked to SLO 3.3)
- Virchow's triad comprises venous stasis, endothelial injury, and ________. (Linked to SLO 3.5)
- A positive D-dimer requires further imaging confirmation given the test's limited ________. (Linked to SLO 3.6)
- Massive pulmonary embolism causes significant haemodynamic compromise through acute right ventricular ________. (Linked to SLO 3.7)

Short-answer questions

1. List the six Ps of acute limb ischaemia. (Linked to SLO 3.1)
2. Explain why embolic limb ischaemia often presents without preceding claudication. (Linked to SLO 3.3)
3. Describe the diagnostic pathway combining clinical probability, D-dimer, and ultrasound. (Linked to SLO 3.6)
4. Describe post-thrombotic syndrome. (Linked to SLO 3.6)

5. Describe the management of stable versus massive pulmonary embolism. (Linked to SLO 3.8)

Long-answer questions

6. Discuss chronic peripheral arterial disease, acute limb ischaemia, and their investigation and management. (Linked to SLO 3.1, 3.2)
7. Describe arterial embolism, its presentation, and its management. (Linked to SLO 3.3, 3.4)
8. Discuss the pathophysiology, presentation, diagnosis, and management of deep vein thrombosis. (Linked to SLO 3.5, 3.6)
9. Describe the pathophysiology, presentation, and diagnosis of pulmonary embolism. (Linked to SLO 3.7)
10. Discuss the management of pulmonary embolism. (Linked to SLO 3.8)

Answers to Concept Check Questions [Series 3]

Objective questions

11. Rest.
12. Bifurcation.
13. Hypercoagulability.
14. Specificity.
15. Strain.

Short-answer questions

16. The six Ps are pain, pallor, pulselessness, paraesthesia, paralysis, and perishingly cold.
17. The limb has not had time to develop collateral circulation, unlike gradually progressive thrombotic disease.
18. Low probability with negative D-dimer excludes the diagnosis; positive D-dimer requires compression ultrasound confirmation.
19. Post-thrombotic syndrome is chronic limb swelling, pain, and skin changes following deep vein thrombosis.
20. Stable pulmonary embolism is managed with anticoagulation; massive pulmonary embolism requires urgent thrombolysis or embolectomy.

Long-answer questions

21. **Basic response:** Chronic disease presents with claudication progressing to critical limb ischaemia; acute limb ischaemia presents with the six Ps, a genuine emergency; investigation uses ankle-brachial pressure index and imaging; management ranges from risk factor modification to urgent revascularisation.

    Value-added response: A value-added response notes that acute limb ischaemia can result from either thrombosis or embolism, with different management implications.

22. **Basic response:** Arterial embolism most commonly originates from atrial fibrillation, presenting with sudden onset ischaemia without preceding claudication; management involves urgent embolectomy or thrombolysis followed by long-term anticoagulation.

    Value-added response: A value-added response notes that identifying and treating the embolic source reduces the risk of future embolic events.

23. **Basic response:** Deep vein thrombosis arises from Virchow's triad; presentation includes limb swelling and pain; diagnosis combines clinical probability, D-dimer, and ultrasound; management uses anticoagulation with duration determined by provoking factors.

    Value-added response: A value-added response notes that post-thrombotic syndrome is a genuine long-term complication despite apparent clinical resolution.

24. **Basic response:** Pulmonary embolism results from embolisation to the pulmonary circulation, typically from deep vein thrombosis, presenting with breathlessness and chest pain; diagnosis uses clinical probability, D-dimer, and CT pulmonary angiography.

    Value-added response: A value-added response notes that ventilation-perfusion scanning offers an alternative for patients unable to have contrast imaging.

25. **Basic response:** Stable pulmonary embolism is managed with anticoagulation; massive pulmonary embolism with haemodynamic compromise requires urgent thrombolysis or embolectomy.

    Value-added response: A value-added response notes that this reflects the same time-critical urgency emphasised throughout cardiovascular emergency management in this course.

Answers to Pilot Questions [Series 3]

Part 3.1

1. Intermittent claudication is exertional pain relieved by rest; critical limb ischaemia is rest pain with tissue loss.
2. Findings include reduced or absent pulses, a cool limb, delayed capillary refill, and skin changes.
3. The six Ps are pain, pallor, pulselessness, paraesthesia, paralysis, and perishingly cold.
4. Thrombotic ischaemia often has preceding claudication and developed collateral circulation, unlike embolic ischaemia.
5. Chronic disease uses risk factor modification and exercise or revascularisation; acute disease requires urgent revascularisation.

Part 3.2

6. Common sources include atrial fibrillation, mural thrombus after myocardial infarction, and proximal atherosclerotic plaque.
7. Emboli lodge where the vessel narrows or branches, physically obstructing further passage.
8. The limb has not had time to develop collateral circulation, unlike gradually progressive thrombotic disease.
9. Management involves urgent embolectomy or catheter-directed thrombolysis.
10. Anticoagulation prevents future embolic events from the identified cardiac source.

Part 3.3

11. Virchow's triad is venous stasis, endothelial injury, and hypercoagulability.
12. Risk factors include surgery, immobility, malignancy, thrombophilia, pregnancy, and oestrogen-containing medication.
13. Low probability with negative D-dimer excludes the diagnosis; positive D-dimer requires ultrasound confirmation.
14. D-dimer has limited specificity, so a positive result requires imaging confirmation rather than being diagnostic alone.
15. Post-thrombotic syndrome is chronic limb swelling, pain, and skin changes following deep vein thrombosis.

Part 3.4

1. Pulmonary embolism presents with breathlessness, pleuritic chest pain, and, in severe cases, haemoptysis.

2. Massive pulmonary embolism causes haemodynamic compromise through right ventricular strain, a genuine emergency.
3. Diagnosis combines clinical probability, D-dimer testing, and CT pulmonary angiography.
4. Ventilation-perfusion scanning is an alternative for patients unable to have contrast imaging.
5. Stable disease is managed with anticoagulation; massive disease requires urgent thrombolysis or embolectomy.

References and Attributions [Series 3]

1. Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
2. Rutherford, R. B. (2019). Rutherford's Vascular Surgery and Endovascular Therapy (9th ed.). Elsevier.
3. National Institute for Health and Care Excellence (2020). Venous Thromboembolic Diseases: Diagnosis, Management and Thrombophilia Testing (NG158). NICE.
4. European Society of Cardiology (2019). Guidelines for the Diagnosis and Management of Acute Pulmonary Embolism. European Heart Journal.

Figures, Plates, Tables, and Boxes

1. Figure 3.1: A clinician examining a patient's limb for pallor and pulse in suspected acute limb ischaemia. AI-generated using the prompt: "Create a realistic clinical illustration titled Assessing Acute Limb Ischaemia, showing a clinician palpating the foot and ankle of a patient's pale lower limb while checking for a pulse, emergency department setting. Clean clinical illustration style, no text."
2. Table 3.1: Comparing thrombotic and embolic acute limb ischaemia. AI-generated using the prompt: "Create a clean reference table titled Comparing Thrombotic and Embolic Acute Limb Ischaemia, listing feature, thrombotic ischaemia, and embolic ischaemia. Simple clinical reference table style with outside border."
3. Figure 3.2: A clinician comparing limb circumference during deep vein thrombosis examination. AI-generated using the prompt: "Create a realistic clinical illustration titled

Deep Vein Thrombosis Examination, showing a clinician using a tape measure to compare the circumference of a patient's swollen calf against the unaffected leg, examination room setting. Clean clinical illustration style, no text."

4. Table 3.2: Comparing stable and massive pulmonary embolism. AI-generated using the prompt: "Create a clean reference table titled Comparing Stable and Massive Pulmonary Embolism, listing feature, stable pulmonary embolism, and massive pulmonary embolism. Simple clinical reference table style with outside border."

Course code: MED 606

Course title: Cardiology II

Course credit load: 2 Units

Series 4: Cardiovascular Infections, Congenital Abnormalities, and Valvular Disease

- **Part 4.1: Cardiovascular Infections I: Infective Endocarditis**
  - Sub Part 4.1.1: Pathogenesis and risk factors for infective endocarditis
  - Sub Part 4.1.2: Clinical presentation of infective endocarditis
  - Sub Part 4.1.3: Diagnosis and management of infective endocarditis
- **Part 4.2: Cardiovascular Infections II: Pericarditis and Myocarditis**
  - Sub Part 4.2.1: Pericarditis
  - Sub Part 4.2.2: Pericardial effusion and tamponade
  - Sub Part 4.2.3: Myocarditis
- **Part 4.3: Congenital Cardiac Abnormalities**
  - Sub Part 4.3.1: Acyanotic congenital heart disease in adults
  - Sub Part 4.3.2: Cyanotic congenital heart disease in adults
  - Sub Part 4.3.3: Principles of adult congenital heart disease care
- **Part 4.4: Valvular Heart Disease**
  - Sub Part 4.4.1: Aortic valve disease
  - Sub Part 4.4.2: Mitral valve disease
  - Sub Part 4.4.3: Principles of management of valvular heart disease

Introduction

This Series turns to three genuinely distinct categories of cardiac disease that together broaden the scope of this course considerably, **cardiovascular infections**, affecting the endocardium, pericardium, and myocardium in turn, **congenital cardiac abnormalities** as they present in the adult patient, and **valvular heart disease**, structural dysfunction of the heart's own valves. Each category demands its own specific pattern recognition, building genuinely on the cardiac examination and investigation skills established throughout this course.

The aim of this Series is to equip you with an understanding of the pathogenesis, presentation, diagnosis, and management of infective endocarditis, pericarditis, pericardial effusion and tamponade, and myocarditis, the presentation and principles of care for acyanotic and cyanotic congenital heart disease in adults, and the presentation and management of aortic and mitral valve disease.

This Series opens with **cardiovascular infections I: infective endocarditis**, moves through **cardiovascular infections II: pericarditis and myocarditis** and **congenital cardiac abnormalities**, and closes with **valvular heart disease**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 4.1:** describe the pathogenesis, risk factors, and clinical presentation of infective endocarditis
- **SLO 4.2:** discuss the diagnosis and management of infective endocarditis
- **SLO 4.3:** describe pericarditis and pericardial effusion and tamponade
- **SLO 4.4:** describe myocarditis
- **SLO 4.5:** describe acyanotic and cyanotic congenital heart disease in adults
- **SLO 4.6:** discuss the principles of adult congenital heart disease care
- **SLO 4.7:** describe aortic and mitral valve disease
- **SLO 4.8:** discuss the principles of management of valvular heart disease

4.1 Cardiovascular Infections I: Infective Endocarditis

4.1.1 pathogenesis and risk factors for infective endocarditis

Bacteria colonising damaged or abnormal valve tissue

Infective endocarditis, infection of the endocardial surface of the heart, most commonly affecting a heart valve, results from bloodstream bacteria adhering to and colonising damaged endothelium, typically at a site of pre-existing valvular abnormality, congenital heart disease, or a prosthetic valve, forming a **vegetation**, a mass of infected material, platelets, and fibrin adherent to the affected structure.

Risk factors include pre-existing valvular disease, congenital heart disease, prosthetic heart valves, intravenous drug use, and recent invasive procedures providing a portal for bacteraemia, and identifying a patient's specific risk factor profile genuinely informs both the index of clinical suspicion in an ambiguous presentation and the specific causative organisms most likely to be responsible in a given clinical context.

Fill in the blank: Infective endocarditis forms a vegetation, a mass of infected material, platelets, and ________.

4.1.2 clinical presentation of infective endocarditis

A genuinely wide-ranging, sometimes deceptively subtle presentation

Infective endocarditis presents with fever, malaise, and a new or changing cardiac murmur, alongside a range of specific peripheral stigmata reflecting immune complex deposition and septic embolisation, including **splinter haemorrhages** beneath the nail beds, **Janeway lesions**, painless erythematous macules on the palms and soles, **Osler's nodes**, painful nodules on the fingers or toes, and **Roth spots**, retinal haemorrhages visible on fundoscopy.

Presentation can genuinely range from an acute, rapidly progressive illness, particularly with more virulent organisms, to a genuinely subtle, subacute presentation with non-specific symptoms persisting for weeks, meaning infective endocarditis should be actively considered in any patient with unexplained fever and a heart murmur, or known risk factors, rather than dismissed as a straightforward, unrelated febrile illness.

Fill in the blank: Janeway lesions are painless erythematous macules on the palms and ________.

4.1.3 diagnosis and management of infective endocarditis

Confirming the organism, then treating for a genuinely prolonged course

Diagnosis combines **blood cultures**, ideally multiple sets taken before antibiotic therapy begins to maximise the chance of identifying the causative organism, with **echocardiography**, directly visualising a vegetation or other supporting structural evidence, and the modified Duke criteria, a structured diagnostic framework combining clinical, microbiological, and echocardiographic findings to reach a confident diagnosis.

Management requires **prolonged intravenous antibiotic therapy**, typically continued for several weeks given the relatively poor antibiotic penetration into the dense, infected vegetation, guided by the specific organism identified and its antibiotic sensitivities, and **surgical intervention**, replacing or repairing the affected valve, is indicated for significant valvular destruction, heart failure, uncontrolled infection despite antibiotics, or recurrent embolic events, a genuinely important treatment option beyond antibiotics alone in appropriately selected patients.

Fill in the blank: Prolonged intravenous antibiotic therapy for endocarditis is required given the relatively poor antibiotic ________ into the vegetation.

Figure 4.1: Splinter haemorrhages beneath the nail beds in infective endocarditis.

Pilot questions 4.1

- Describe the pathogenesis of infective endocarditis. (Linked to SLO 4.1)
- List risk factors for infective endocarditis. (Linked to SLO 4.1)
- List the peripheral stigmata of infective endocarditis. (Linked to SLO 4.1)
- Describe the diagnostic approach to suspected infective endocarditis. (Linked to SLO 4.2)
- List the indications for surgical intervention in infective endocarditis. (Linked to SLO 4.2)

4.2 Cardiovascular Infections II: Pericarditis and Myocarditis

4.2.1 pericarditis

Inflammation of the sac surrounding the heart

Pericarditis, inflammation of the pericardium, most commonly viral or idiopathic in origin, presents with sharp, pleuritic chest pain characteristically improving on sitting forward and worsening on lying flat, alongside a **pericardial friction rub**, a scratchy sound heard on auscultation independent of respiration, and characteristic diffuse ST elevation on electrocardiography distinguishing it from the more localised ST elevation of myocardial infarction already discussed in an earlier Series.

Management of uncomplicated pericarditis typically involves nonsteroidal anti-inflammatory drugs, alongside colchicine, which genuinely reduces the risk of recurrence, and the majority of cases resolve fully without complication, though a proportion of patients experience recurrent episodes, and identifying an underlying treatable cause, where genuinely present, is worthwhile given its direct relevance to preventing further recurrence.

Fill in the blank: Pericarditic chest pain characteristically improves on sitting forward and worsens on lying ________.

4.2.2 pericardial effusion and tamponade

Fluid accumulation, and a genuine emergency if it accumulates too fast

Pericardial effusion, fluid accumulation within the pericardial space, can result from pericarditis or numerous other causes, and, while a small, slowly accumulating effusion may be genuinely well tolerated given the pericardium's capacity to gradually stretch, a large or rapidly accumulating effusion can progress to **cardiac tamponade**, external compression of the heart preventing adequate filling.

Cardiac tamponade presents with the classical triad of hypotension, raised jugular venous pressure, and muffled heart sounds, alongside **pulsus paradoxus**, an exaggerated fall in blood pressure during inspiration, a genuine surgical emergency requiring urgent **pericardiocentesis**, draining the accumulated fluid, to relieve the compression before cardiovascular collapse occurs, mirroring the same urgency already discussed for other genuine cardiac emergencies elsewhere in this course.

Fill in the blank: Cardiac tamponade presents with pulsus paradoxus, an exaggerated fall in blood pressure during ________.

4.2.3 myocarditis

Inflammation of the heart muscle itself

Myocarditis, inflammation of the heart muscle itself, most commonly viral in origin, presents with a genuinely wide clinical spectrum ranging from mild, self-limiting chest discomfort and fatigue to genuinely fulminant heart failure and life-threatening arrhythmia, and, given this wide spectrum, should be actively considered in a younger patient presenting with new heart failure or significant arrhythmia without another obvious explanation.

Diagnosis combines clinical suspicion with cardiac magnetic resonance imaging, showing characteristic patterns of myocardial inflammation, and, in selected cases, endomyocardial biopsy, and management is largely supportive, addressing any resulting heart failure or arrhythmia using the same principles already established elsewhere in this course, since specific disease-modifying treatment for the underlying myocardial inflammation itself remains genuinely limited for most causes of myocarditis.

Fill in the blank: Myocarditis should be actively considered in a younger patient presenting with new heart failure or significant _________.

Table 4.1: Comparing pericarditis, tamponade, and myocarditis

Condition	Key feature	Urgency
Pericarditis	Positional chest pain, friction rub	Generally not urgent
Cardiac tamponade	Hypotension, muffled heart sounds	Genuine surgical emergency
Myocarditis	Wide spectrum from mild to fulminant	Depends on severity

Pilot questions 4.2

1. Describe the classic presentation of pericarditis. (Linked to SLO 4.3)
2. Describe the management of uncomplicated pericarditis. (Linked to SLO 4.3)
3. Describe cardiac tamponade and its classical triad. (Linked to SLO 4.3)
4. Describe the immediate management of cardiac tamponade. (Linked to SLO 4.3)
5. Describe myocarditis and its clinical spectrum. (Linked to SLO 4.4)

4.3 Congenital Cardiac Abnormalities

4.3.1 acyanotic congenital heart disease in adults

Left-to-right shunting persisting into adulthood

Atrial septal defect and **ventricular septal defect**, already discussed in an earlier course covering cardiothoracic surgery, can present for the first time in adulthood when a small defect has remained asymptomatic through childhood, or when a previously repaired defect requires ongoing surveillance, and adults with an unrepaired, significant left-to-right shunt can develop progressive pulmonary hypertension and, eventually, **Eisenmenger syndrome**, reversal of the shunt direction, over many years of untreated disease.

Coarctation of the aorta, also already introduced in an earlier course, can similarly present in adulthood, sometimes discovered during investigation of hypertension, and recognising these congenital conditions when they present later in life, rather than assuming congenital heart disease is exclusively a paediatric concern, is a genuinely important skill for any clinician managing adult cardiac patients, given the increasing number of adults now living with congenital heart disease due to improved paediatric survival.

Fill in the blank: Eisenmenger syndrome describes reversal of shunt direction following untreated pulmonary ________.

4.3.2 cyanotic congenital heart disease in adults

Surviving into adulthood with a complex repaired defect

Tetralogy of Fallot, already discussed in an earlier course, and other cyanotic congenital heart conditions, are now genuinely commonly encountered in adult practice given the considerable improvement in survival following paediatric surgical repair over recent decades, and adults with repaired cyanotic congenital heart disease require lifelong surveillance for residual lesions, arrhythmia, and progressive dysfunction of the surgically repaired structures.

Residual pulmonary regurgitation following tetralogy of Fallot repair, in particular, can produce progressive right ventricular dilatation over many years, sometimes eventually requiring pulmonary valve replacement, illustrating how the long-term follow-up needs of adults with repaired congenital heart disease genuinely extend well beyond the original successful paediatric surgical repair itself, a theme this Part's closing sub-Part develops further.

Fill in the blank: Residual pulmonary regurgitation following tetralogy of Fallot repair can produce progressive right ventricular ________.

4.3.3 principles of adult congenital heart disease care

A genuinely specialised, lifelong field of care

Adult congenital heart disease has emerged as a genuinely distinct specialist field, reflecting the growing population of adults living with repaired or unrepaired congenital heart conditions, and management requires specific expertise in the long-term natural history of these conditions, since standard adult cardiology training alone does not always adequately prepare a clinician for the specific complexities this patient population presents.

Transition of care, moving a young adult from paediatric to adult congenital heart disease services, is a genuinely important and sometimes vulnerable point in a patient's care pathway, and structured, coordinated transition, alongside lifelong specialist follow-up incorporating regular imaging surveillance and arrhythmia monitoring, genuinely improves long-term outcomes for this specific, growing patient population.

Fill in the blank: Transition of care moves a young adult from paediatric to adult congenital heart disease ________.

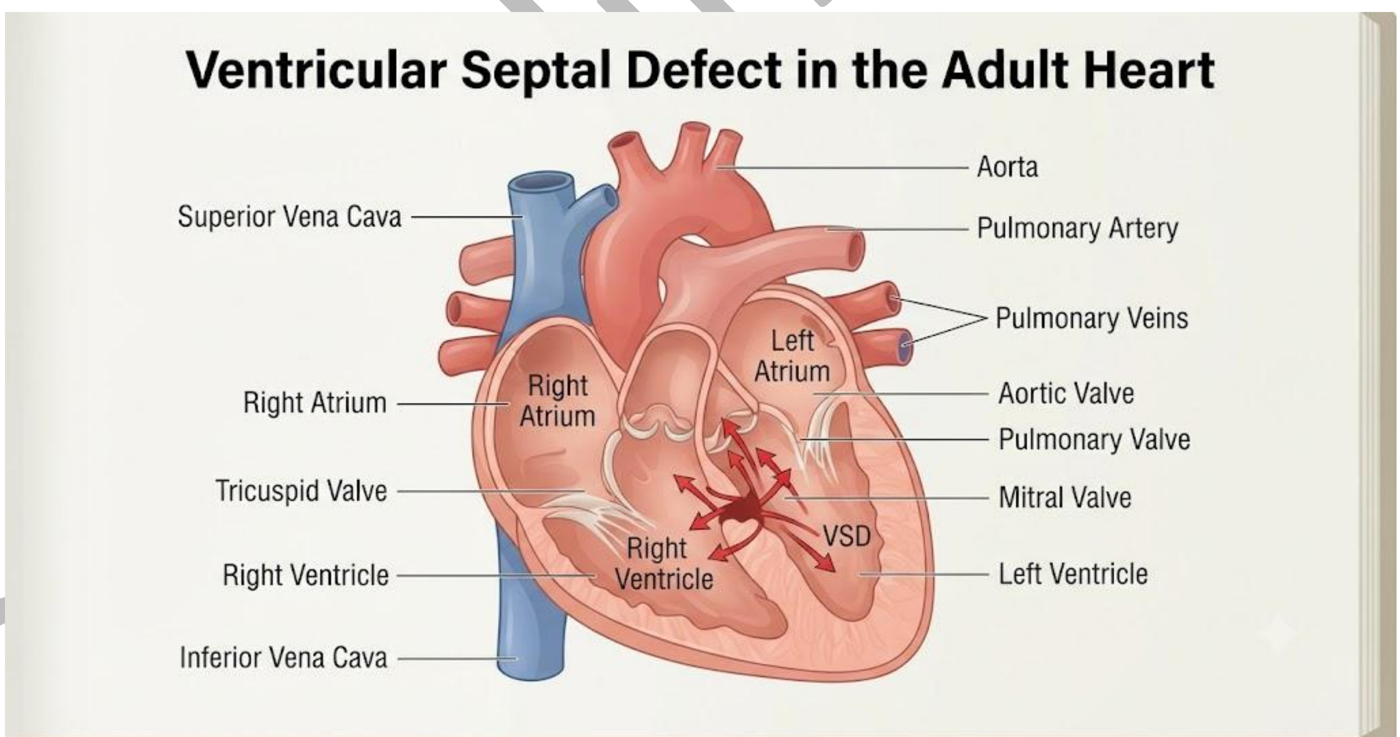

Figure 4.2: A labelled anatomical diagram of a ventricular septal defect.

Pilot questions 4.3

1. Explain how an unrepaired left-to-right shunt can progress to Eisenmenger syndrome. (Linked to SLO 4.5)
2. Describe coarctation of the aorta presenting in adulthood. (Linked to SLO 4.5)
3. Explain why cyanotic congenital heart disease is now commonly encountered in adult practice. (Linked to SLO 4.5)
4. Describe residual pulmonary regurgitation following tetralogy of Fallot repair. (Linked to SLO 4.5)
5. Describe the importance of structured transition of care in adult congenital heart disease. (Linked to SLO 4.6)

4.4 Valvular Heart Disease

4.4.1 aortic valve disease

Stenosis and regurgitation of the outflow valve

Aortic stenosis, narrowing of the aortic valve, most commonly from age-related calcific degeneration, presents with the classic triad of exertional syncope, angina, and breathlessness as the condition progresses, and produces an ejection systolic murmur radiating to the carotids, findings genuinely important to recognise given the significant symptomatic and prognostic implications of progressive, severe aortic stenosis once symptoms develop.

Aortic regurgitation, incompetence of the aortic valve allowing backflow of blood into the left ventricle during diastole, results from either primary valve pathology or aortic root dilatation, presents with a diastolic murmur and, in chronic severe cases, progressive left ventricular dilatation and eventual heart failure, and, unlike aortic stenosis, can remain genuinely well tolerated for a considerably longer period given the ventricle's capacity to compensate for the chronic volume overload.

Fill in the blank: Aortic stenosis most commonly results from age-related calcific ________.

4.4.2 mitral valve disease

Stenosis and regurgitation of the inflow valve

Mitral stenosis, narrowing of the mitral valve, most commonly resulting from previous rheumatic fever, presents with breathlessness and, given the associated left atrial enlargement, a genuinely increased risk of atrial fibrillation, and produces a characteristic mid-diastolic murmur best heard with the patient in the left lateral position, findings that, alongside the specific history of rheumatic fever where present, support the clinical diagnosis.

Mitral regurgitation, incompetence of the mitral valve allowing backflow into the left atrium during systole, results from a genuinely wide range of causes including mitral valve prolapse, ischaemic papillary muscle dysfunction, and infective endocarditis already discussed earlier in this Series, presents with a pansystolic murmur radiating to the axilla, and, like aortic regurgitation, can remain well tolerated for a considerable period before progressive left ventricular dysfunction eventually develops.

Fill in the blank: Mitral stenosis most commonly results from previous ________ fever.

4.4.3 principles of management of valvular heart disease

Watching, then intervening at the right moment

Management of valvular heart disease combines regular clinical and echocardiographic surveillance for asymptomatic or mildly symptomatic patients, since the timing of intervention genuinely matters, with definitive intervention, valve repair or replacement, whether surgical or, increasingly, catheter-based, indicated once specific symptomatic or echocardiographic thresholds of severity are reached, a decision requiring careful, individualised weighing of the specific valve lesion, symptom burden, and patient factors.

Choice between valve repair and replacement, and, where replacement is chosen, between a mechanical and biological prosthesis, already discussed in principle in an earlier course covering cardiothoracic surgery, requires the same individualised discussion of durability against anticoagulation burden, and this Series closes on the same theme of structured, individualised clinical decision-making that has run consistently throughout the whole of this cardiovascular programme.

Fill in the blank: Choice between valve repair and replacement requires individualised discussion of durability against anticoagulation ________.

Table 4.2: Comparing aortic and mitral valve lesions

Lesion	Murmur character	Common cause
Aortic stenosis	Ejection systolic, radiating to carotids	Calcific degeneration
Mitral stenosis	Mid-diastolic	Previous rheumatic fever
Mitral regurgitation	Pansystolic, radiating to axilla	Prolapse, ischaemia, endocarditis

Pilot questions 4.4

1. Describe the classic triad of aortic stenosis. (Linked to SLO 4.7)
2. Describe aortic regurgitation and its typical causes. (Linked to SLO 4.7)
3. Describe mitral stenosis and its typical cause. (Linked to SLO 4.7)
4. Describe mitral regurgitation and its typical murmur. (Linked to SLO 4.7)
5. Describe the general approach to timing intervention in valvular heart disease. (Linked to SLO 4.8)

Series Summary

This Series covered cardiovascular infections, congenital abnormalities, and valvular heart disease. Part 4.1 covered infective endocarditis, while Part 4.2 turned to pericarditis, tamponade, and myocarditis. Part 4.3 covered acyanotic and cyanotic congenital heart disease in adults and the principles of adult congenital heart disease care, and Part 4.4 closed with aortic and mitral valve disease.

You should now be able to describe cardiovascular infections, adult congenital heart disease, and valvular heart disease across all four Parts of this Series, meeting the outcomes set for this Series. Series 5 turns next to emergencies in cardiology.

Glossary of Terms

1. **Aortic stenosis:** narrowing of the aortic valve, most commonly from calcific degeneration.

2. **Cardiac tamponade:** external compression of the heart by pericardial fluid preventing adequate filling.
3. **Eisenmenger syndrome:** reversal of shunt direction following untreated pulmonary hypertension.
4. **Infective endocarditis:** infection of the endocardial surface of the heart, typically a valve.
5. **Janeway lesion:** a painless erythematous macule on the palms or soles in infective endocarditis.
6. **Mitral stenosis:** narrowing of the mitral valve, most commonly from rheumatic fever.
7. **Myocarditis:** inflammation of the heart muscle itself, most commonly viral.
8. **Osler's node:** a painful nodule on the fingers or toes in infective endocarditis.
9. **Pericardiocentesis:** drainage of pericardial fluid to relieve cardiac tamponade.
10. **Pericarditis:** inflammation of the pericardium, presenting with positional chest pain.
11. **Splinter haemorrhage:** a thin, dark streak beneath the nail bed in infective endocarditis.
12. **Vegetation:** a mass of infected material, platelets, and fibrin in infective endocarditis.

Concept Check Questions [Series 4]

Objective questions

- Infective endocarditis forms a vegetation, a mass of infected material, platelets, and ________. (Linked to SLO 4.1)
- Pericarditic chest pain characteristically improves on sitting forward and worsens on lying ________. (Linked to SLO 4.3)
- Eisenmenger syndrome describes reversal of shunt direction following untreated pulmonary ________. (Linked to SLO 4.5)
- Aortic stenosis most commonly results from age-related calcific ________. (Linked to SLO 4.7)
- Mitral stenosis most commonly results from previous ________ fever. (Linked to SLO 4.7)

Short-answer questions

1. List the peripheral stigmata of infective endocarditis. (Linked to SLO 4.1)
2. Describe cardiac tamponade and its classical triad. (Linked to SLO 4.3)
3. Describe myocarditis and its clinical spectrum. (Linked to SLO 4.4)
4. Explain why cyanotic congenital heart disease is now commonly encountered in adult practice. (Linked to SLO 4.5)

5. Describe the general approach to timing intervention in valvular heart disease. (Linked to SLO 4.8)

Long-answer questions

6. Discuss the pathogenesis, presentation, diagnosis, and management of infective endocarditis. (Linked to SLO 4.1, 4.2)
7. Describe pericarditis, cardiac tamponade, and myocarditis. (Linked to SLO 4.3, 4.4)
8. Discuss acyanotic and cyanotic congenital heart disease in adults and the principles of adult congenital heart disease care. (Linked to SLO 4.5, 4.6)
9. Describe aortic and mitral valve disease. (Linked to SLO 4.7)
10. Discuss the principles of management of valvular heart disease. (Linked to SLO 4.8)

Answers to Concept Check Questions [Series 4]

Objective questions

11. Fibrin.
12. Flat.
13. Hypertension.
14. Degeneration.
15. Rheumatic.

Short-answer questions

16. Stigmata include splinter haemorrhages, Janeway lesions, Osler's nodes, and Roth spots.
17. Cardiac tamponade is external compression preventing filling, with the triad of hypotension, raised jugular pressure, and muffled heart sounds.
18. Myocarditis ranges from mild chest discomfort to fulminant heart failure and life-threatening arrhythmia.
19. Improved paediatric surgical survival means more patients with repaired cyanotic conditions now reach adulthood.
20. Regular surveillance is combined with definitive intervention once specific symptomatic or echocardiographic thresholds are reached.

Long-answer questions

21. **Basic response:** Endocarditis results from bacteria colonising damaged valve tissue, forming a vegetation; presentation includes fever and peripheral stigmata; diagnosis uses blood cultures and echocardiography; management uses prolonged antibiotics and, where indicated, surgery.

    Value-added response: A value-added response notes that surgical intervention is indicated for significant valvular destruction, heart failure, or uncontrolled infection.

22. **Basic response:** Pericarditis presents with positional chest pain and a friction rub; cardiac tamponade is a genuine emergency requiring urgent pericardiocentesis; myocarditis has a wide clinical spectrum managed largely supportively.

    Value-added response: A value-added response notes that colchicine genuinely reduces the risk of pericarditis recurrence.

23. **Basic response:** Acyanotic conditions can present in adulthood or progress to Eisenmenger syndrome if untreated; repaired cyanotic conditions require lifelong surveillance; adult congenital heart disease is a distinct specialist field requiring structured transition of care.

    Value-added response: A value-added response notes that residual pulmonary regurgitation after tetralogy of Fallot repair can require later valve replacement.

24. **Basic response:** Aortic stenosis presents with exertional syncope, angina, and breathlessness; aortic regurgitation is better tolerated chronically; mitral stenosis follows rheumatic fever; mitral regurgitation has diverse causes with a pansystolic murmur.

    Value-added response: A value-added response notes that regurgitant lesions are generally better tolerated for longer than stenotic lesions given ventricular compensatory capacity.

25. **Basic response:** Management combines regular surveillance with definitive repair or replacement once symptomatic or echocardiographic thresholds are reached, individualised to the specific lesion and patient factors.

    Value-added response: A value-added response notes that choice between mechanical and biological prosthesis requires weighing durability against lifelong anticoagulation burden.

Answers to Pilot Questions [Series 4]

Part 4.1

1. Bacteria adhere to and colonise damaged endothelium, forming a vegetation of infected material and fibrin.
2. Risk factors include pre-existing valvular disease, congenital heart disease, prosthetic valves, and intravenous drug use.
3. Stigmata include splinter haemorrhages, Janeway lesions, Osler's nodes, and Roth spots.
4. Diagnosis combines blood cultures, echocardiography, and the modified Duke criteria.
5. Indications include significant valvular destruction, heart failure, uncontrolled infection, and recurrent embolic events.

Part 4.2

6. Pericarditis presents with pleuritic chest pain improving on sitting forward and a pericardial friction rub.
7. Management uses nonsteroidal anti-inflammatory drugs and colchicine to reduce recurrence.
8. Cardiac tamponade is external cardiac compression with the triad of hypotension, raised jugular pressure, and muffled heart sounds.
9. Immediate management is urgent pericardiocentesis to relieve compression.
10. Myocarditis ranges from mild chest discomfort and fatigue to fulminant heart failure and life-threatening arrhythmia.

Part 4.3

11. Untreated significant shunting causes progressive pulmonary hypertension, eventually reversing the shunt direction.
12. Coarctation can present in adulthood, sometimes discovered during investigation of hypertension.
13. Improved paediatric surgical survival means more patients with repaired cyanotic conditions now reach adulthood.
14. It can produce progressive right ventricular dilatation over many years, sometimes requiring later valve replacement.
15. Structured transition supports continuity of specialist care during a vulnerable point in the patient's pathway.

Part 4.4

1. The triad is exertional syncope, angina, and breathlessness.

2. Aortic regurgitation results from primary valve pathology or aortic root dilatation and can remain well tolerated for a long period.
3. Mitral stenosis most commonly results from previous rheumatic fever.
4. Mitral regurgitation produces a pansystolic murmur radiating to the axilla.
5. Regular surveillance is combined with intervention once specific symptomatic or echocardiographic thresholds are reached.

References and Attributions [Series 4]

1. Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
2. Libby, P., Bonow, R. O., Mann, D. L., Tomaselli, G. F., & Braunwald, E. (2021). Braunwald's Heart Disease (12th ed.). Elsevier.
3. European Society of Cardiology (2023). Guidelines for the Management of Endocarditis. European Heart Journal.
4. European Society of Cardiology (2021). Guidelines for the Management of Valvular Heart Disease. European Heart Journal.

Figures, Plates, Tables, and Boxes

1. Figure 4.1: Splinter haemorrhages beneath the nail beds in infective endocarditis. AI-generated using the prompt: "Create a realistic clinical illustration titled Splinter Haemorrhages in Infective Endocarditis, showing a close-up view of a patient's fingernails with visible thin, dark red streaks beneath several nail beds, clean neutral background. Clean clinical illustration style, no text."
2. Table 4.1: Comparing pericarditis, tamponade, and myocarditis. AI-generated using the prompt: "Create a clean reference table titled Comparing Pericarditis, Tamponade, and Myocarditis, listing condition, key feature, and urgency. Simple clinical reference table style with outside border."
3. Figure 4.2: A labelled anatomical diagram of a ventricular septal defect. AI-generated using the prompt: "Create a clean, accurate labelled anatomical cross-section diagram titled Ventricular Septal Defect in the Adult Heart, showing the four chambers of the heart with a labelled opening in the ventricular septum and arrows indicating the direction of blood shunting. Educational anatomical diagram style, no photographic content."

4. Table 4.2: Comparing aortic and mitral valve lesions. AI-generated using the prompt: "Create a clean reference table titled Comparing Aortic and Mitral Valve Lesions, listing lesion, murmur character, and common cause. Simple clinical reference table style with outside border."

Course code: MED 606

Course title: Cardiology II

Course credit load: 2 Units

Series 5: Emergencies in Cardiology

- **Part 5.1: Cardiac Arrest and Resuscitation**
  - Sub Part 5.1.1: Causes and recognition of cardiac arrest
  - Sub Part 5.1.2: Principles of basic and advanced life support
  - Sub Part 5.1.3: Post-resuscitation care
- **Part 5.2: Cardiogenic Shock and Acute Pulmonary Oedema**
  - Sub Part 5.2.1: Cardiogenic shock: pathophysiology and presentation
  - Sub Part 5.2.2: Management of cardiogenic shock
  - Sub Part 5.2.3: Acute pulmonary oedema
- **Part 5.3: Hypertensive Emergency**
  - Sub Part 5.3.1: Definition and pathophysiology of hypertensive emergency
  - Sub Part 5.3.2: Clinical presentation and end-organ damage
  - Sub Part 5.3.3: Management of hypertensive emergency
- **Part 5.4: Aortic Dissection**
  - Sub Part 5.4.1: Pathophysiology and classification of aortic dissection
  - Sub Part 5.4.2: Clinical presentation and diagnosis of aortic dissection
  - Sub Part 5.4.3: Management of aortic dissection

Introduction

This final Series closes the course with the genuine emergencies of cardiology, presentations where recognition and treatment within minutes, rather than hours, directly determines whether a patient survives. **Cardiac arrest**, **cardiogenic shock and acute pulmonary oedema**, **hypertensive emergency**, and **aortic dissection** each demand the same rapid, structured recognition and response already emphasised throughout this course, brought together here in their most acute, time-critical form.

The aim of this Series is to equip you with the ability to describe the causes and recognition of cardiac arrest and the principles of resuscitation and post-resuscitation care, describe the pathophysiology, presentation, and management of cardiogenic shock and acute pulmonary oedema, describe the presentation and management of hypertensive emergency, and describe the pathophysiology, presentation, diagnosis, and management of aortic dissection.

This Series opens with **cardiac arrest and resuscitation**, moves through **cardiogenic shock and acute pulmonary oedema** and **hypertensive emergency**, and closes with **aortic dissection**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 5.1:** describe the causes and recognition of cardiac arrest
- **SLO 5.2:** discuss the principles of resuscitation and post-resuscitation care
- **SLO 5.3:** describe the pathophysiology, presentation, and management of cardiogenic shock
- **SLO 5.4:** describe acute pulmonary oedema
- **SLO 5.5:** describe the definition, pathophysiology, and presentation of hypertensive emergency
- **SLO 5.6:** discuss the management of hypertensive emergency
- **SLO 5.7:** describe the pathophysiology, classification, presentation, and diagnosis of aortic dissection
- **SLO 5.8:** discuss the management of aortic dissection

5.1 Cardiac Arrest and Resuscitation

5.1.1 causes and recognition of cardiac arrest

The complete absence of effective circulation

Cardiac arrest, the sudden cessation of effective cardiac output, most commonly results from **ventricular fibrillation** or pulseless **ventricular tachycardia**, already discussed in an earlier Series of this course, though it can also result from asystole or pulseless electrical activity, a rhythm present on the monitor without any corresponding effective mechanical contraction, each requiring genuinely distinct immediate management despite the shared, universally catastrophic clinical outcome.

Recognition of cardiac arrest relies on rapid assessment of responsiveness and breathing, since a collapsed, unresponsive patient with absent or abnormal breathing should be assumed to be in cardiac arrest and resuscitation commenced immediately, without waiting for formal pulse confirmation, which is genuinely notoriously unreliable and can meaningfully delay the initiation of life-saving chest compressions in this specific, time-critical scenario.

Fill in the blank: Cardiac arrest most commonly results from ventricular fibrillation or pulseless ventricular ________.

5.1.2 principles of basic and advanced life support

A structured, algorithm-driven response

Basic life support, high-quality chest compressions and rescue breaths, alongside early defibrillation where a shockable rhythm is identified, forms the essential foundation of resuscitation, and the quality of chest compressions, adequate rate, depth, and full chest recoil between compressions, genuinely, measurably affects the likelihood of a successful outcome, making technique quality a genuine priority rather than a secondary consideration.

Advanced life support, building on this foundation with airway management, intravenous access, and specific medication including adrenaline and, for shockable rhythms, amiodarone, follows a structured, algorithm-driven approach distinguishing **shockable rhythms**, ventricular fibrillation and pulseless ventricular tachycardia, from **non-shockable rhythms**, asystole and pulseless electrical activity, since this distinction directly determines whether defibrillation forms part of the immediate treatment sequence.

Fill in the blank: The quality of chest compressions genuinely, measurably affects the likelihood of a successful resuscitation ________.

5.1.3 post-resuscitation care

The critical period after circulation returns

Following successful resuscitation and **return of spontaneous circulation**, the immediate priority shifts to identifying and treating the underlying cause of the arrest, since addressing the precipitating problem, most commonly an acute coronary syndrome already discussed in an earlier Series of this course, is genuinely essential to preventing a further, potentially fatal recurrence of the arrest.

Targeted temperature management, controlling body temperature within a specific range following resuscitation, genuinely improves neurological outcome in appropriately selected patients who remain unresponsive, and comprehensive post-resuscitation care, addressing haemodynamic stability, oxygenation, and the underlying precipitating cause together, closes the immediate resuscitation episode and transitions the patient towards longer-term recovery and rehabilitation.

Fill in the blank: Targeted temperature management genuinely improves neurological outcome in appropriately selected ________ patients.

Figure 5.1: Clinicians performing cardiopulmonary resuscitation with an attached defibrillator.

Pilot questions 5.1

- List the causes of cardiac arrest. (Linked to SLO 5.1)
- Explain why resuscitation should begin without waiting for formal pulse confirmation. (Linked to SLO 5.1)
- Describe the components of basic life support. (Linked to SLO 5.2)
- Distinguish shockable from non-shockable rhythms. (Linked to SLO 5.2)
- Describe targeted temperature management and its benefit. (Linked to SLO 5.2)

5.2 Cardiogenic Shock and Acute Pulmonary Oedema

5.2.1 cardiogenic shock: pathophysiology and presentation

The heart failing to meet the body's basic circulatory needs

Cardiogenic shock, inadequate tissue perfusion resulting from severe cardiac pump failure, most commonly following extensive myocardial infarction already discussed in an earlier Series of this course, presents with profound hypotension, cold, poorly perfused peripheries, reduced urine output, and altered mental status, a presentation reflecting the body's inadequate perfusion of vital organs despite the heart's continued, though genuinely severely compromised, mechanical activity.

Cardiogenic shock carries genuinely high mortality even with optimal modern treatment, and prompt recognition, distinguishing it from other causes of shock such as sepsis or haemorrhage through careful clinical assessment and echocardiography, directly shapes the specific, genuinely urgent treatment pathway required, given how fundamentally different the underlying management approach is for cardiogenic shock compared with these other shock categories.

Fill in the blank: Cardiogenic shock most commonly follows extensive myocardial ________.

5.2.2 management of cardiogenic shock

Supporting the circulation while treating the underlying cause

Management of cardiogenic shock requires urgent haemodynamic support, including careful, judicious fluid management, inotropic medication to support cardiac contractility, and, in selected cases, **mechanical circulatory support**, temporary devices assisting or partially replacing the heart's own pumping function while definitive treatment is arranged, alongside urgent treatment of the underlying precipitating cause, most commonly urgent coronary revascularisation for cardiogenic shock following myocardial infarction.

Coordinated, rapid multidisciplinary management, drawing on cardiology, intensive care, and, where mechanical circulatory support is required, cardiac surgery, genuinely improves outcomes given the complexity and time-critical nature of this specific presentation, and this collaborative, structured approach reflects the same theme of coordinated emergency response already emphasised throughout this Series and, indeed, this entire course.

Fill in the blank: Mechanical circulatory support assists or partially replaces the heart's own pumping ________ temporarily.

5.2.3 acute pulmonary oedema

Fluid flooding into the lungs, often alongside cardiogenic shock

Acute pulmonary oedema, fluid accumulation within the lung tissue and alveoli from acutely elevated left atrial pressure, presents with severe, sudden-onset breathlessness, orthopnoea, and, in significant cases, pink, frothy sputum, alongside widespread crackles on chest auscultation, a genuinely distressing presentation for the patient requiring prompt recognition and treatment given the significant respiratory compromise it produces.

Management includes sitting the patient upright to optimise respiratory mechanics, supplemental oxygen, and intravenous diuretic therapy to reduce fluid overload, alongside treatment of any identifiable precipitating cause, and, where acute pulmonary oedema occurs alongside cardiogenic shock already discussed earlier in this Part, management genuinely requires careful balancing of these sometimes competing therapeutic priorities, since aggressive diuresis in a shocked patient can worsen the underlying hypoperfusion.

Fill in the blank: Aggressive diuresis in a shocked patient can worsen the underlying _________.

Table 5.1: Key features distinguishing cardiogenic shock from acute pulmonary oedema alone

Feature	Cardiogenic shock	Acute pulmonary oedema alone
Blood pressure	Profoundly reduced	Often preserved or elevated
Peripheries	Cold, poorly perfused	Often warm
Primary management focus	Haemodynamic support and revascularisation	Diuresis and respiratory support

Pilot questions 5.2

1. Describe the presentation of cardiogenic shock. (Linked to SLO 5.3)
2. Explain why prompt recognition of cardiogenic shock matters clinically. (Linked to SLO 5.3)
3. Describe the components of cardiogenic shock management. (Linked to SLO 5.3)
4. Describe the presentation of acute pulmonary oedema. (Linked to SLO 5.4)
5. Explain why aggressive diuresis requires caution in a shocked patient. (Linked to SLO 5.4)

5.3 Hypertensive Emergency

5.3.1 definition and pathophysiology of hypertensive emergency

Severely elevated blood pressure with acute organ damage

Hypertensive emergency, severely elevated blood pressure associated with acute, progressive end-organ damage, is defined not by a single specific blood pressure threshold alone but by the presence of this associated organ damage, a genuinely important distinction from **hypertensive urgency**, severely elevated blood pressure without acute organ damage, which, despite the alarming numerical reading, does not carry the same immediate treatment urgency.

The underlying pathophysiology involves a failure of normal vascular autoregulation, the mechanism that would ordinarily protect end organs from excessive pressure, leading to endothelial damage and, in the most severe cases, fibrinoid necrosis of small vessels, a pathological process directly underlying the specific end-organ damage patterns covered in the following sub-Part of this Part.

Fill in the blank: Hypertensive emergency involves a failure of normal vascular ________, ordinarily protecting end organs.

5.3.2 clinical presentation and end-organ damage

A genuinely wide range of possible affected organs

End-organ damage in hypertensive emergency can affect several systems, including the brain, producing hypertensive encephalopathy with headache, confusion, and, in severe cases, seizure, the eyes, producing hypertensive retinopathy with papilloedema and haemorrhages visible on fundoscopy, the kidneys, producing acute kidney injury, and the heart, producing acute pulmonary oedema or acute coronary syndrome, already discussed elsewhere in this Series and course.

Fundoscopic examination, identifying papilloedema or specific retinal changes, provides genuinely valuable, directly visible evidence of end-organ damage and should be actively performed in any patient with significantly elevated blood pressure, since the presence of these specific fundoscopic findings, alongside the broader clinical assessment, directly confirms a hypertensive emergency rather than the less immediately urgent hypertensive urgency already distinguished in the previous sub-Part.

Fill in the blank: Fundoscopic examination should be actively performed in any patient with significantly elevated blood ________.

5.3.3 management of hypertensive emergency

Controlled, gradual reduction rather than rapid normalisation

Management of hypertensive emergency requires **intravenous antihypertensive therapy** with careful, controlled blood pressure reduction, typically no more than approximately twenty-five percent within the first hour, since overly rapid blood pressure reduction can itself cause organ hypoperfusion and worsen the underlying clinical picture, a genuinely important, sometimes counterintuitive principle given the alarming nature of the initial elevated blood pressure reading.

Treatment setting and specific medication choice depend on the predominant pattern of end-organ damage identified, with continuous blood pressure monitoring in an appropriately monitored clinical environment essential throughout the initial treatment period, and once blood pressure has been safely, gradually controlled, transition to oral antihypertensive therapy and thorough investigation for an underlying, potentially treatable cause of the hypertensive emergency completes the acute management episode.

Fill in the blank: Controlled blood pressure reduction is typically no more than approximately twenty-five percent within the first ________.

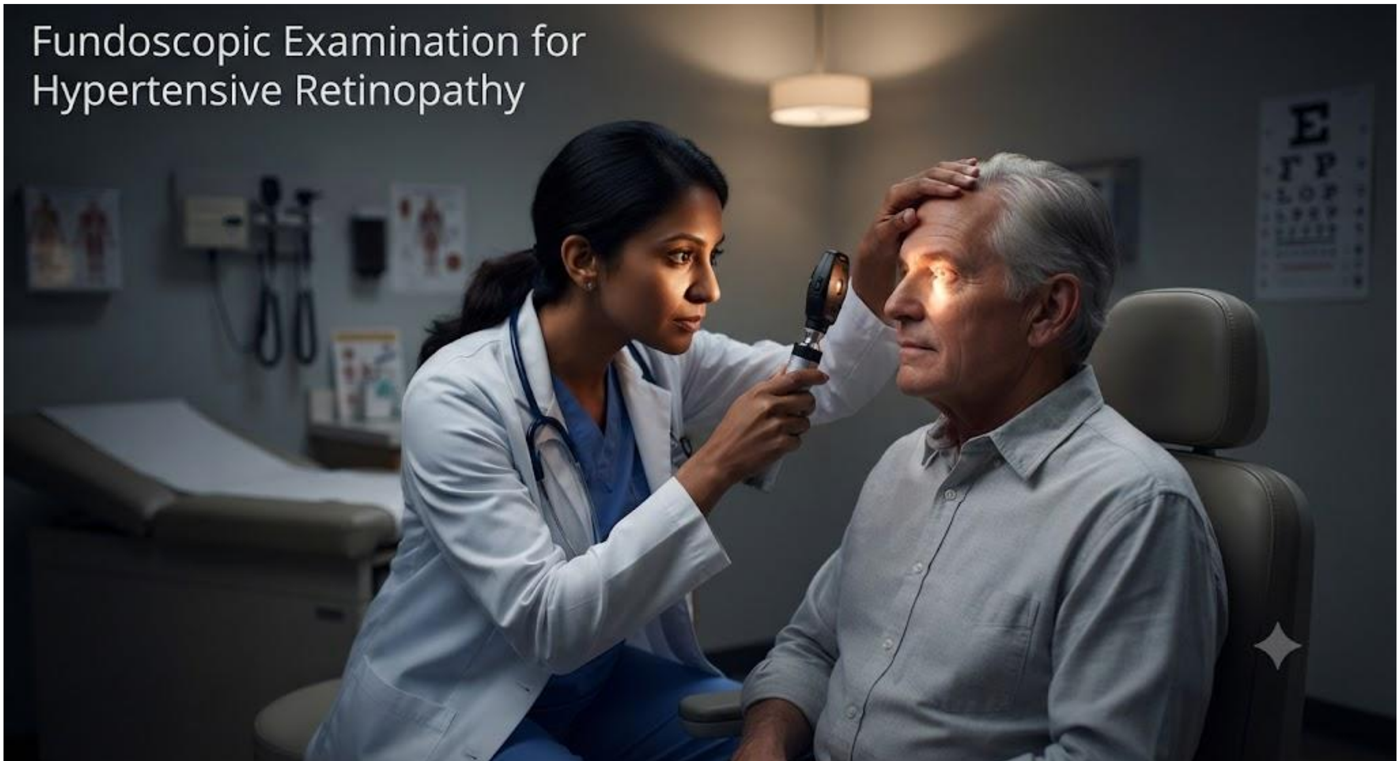

Figure 5.2: A clinician performing fundoscopic examination to assess for hypertensive retinopathy.

Pilot questions 5.3

1. Distinguish hypertensive emergency from hypertensive urgency. (Linked to SLO 5.5)
2. Describe the underlying pathophysiology of hypertensive emergency. (Linked to SLO 5.5)
3. List the organ systems that can be affected by end-organ damage in hypertensive emergency. (Linked to SLO 5.5)
4. Explain the value of fundoscopic examination in suspected hypertensive emergency. (Linked to SLO 5.5)
5. Explain why controlled, gradual blood pressure reduction is genuinely important in hypertensive emergency. (Linked to SLO 5.6)

5.4 Aortic Dissection

5.4.1 pathophysiology and classification of aortic dissection

A tear in the aortic wall creating a genuinely dangerous false channel

Aortic dissection, a tear in the inner layer of the aortic wall allowing blood to enter and separate the layers of the wall, creating a **false lumen** alongside the normal, true lumen, most commonly results from longstanding hypertension causing progressive weakening of the aortic wall, and is classified according to the **Stanford system** as **type A**, involving the ascending aorta, or **type B**, confined to the descending aorta, a distinction with direct implications for both urgency and specific management approach.

Type A dissection, given its involvement of the ascending aorta and the genuine risk of extension to compromise the coronary arteries, aortic valve, or pericardial space producing tamponade already discussed earlier in this Series, carries a considerably higher immediate mortality risk than type B dissection and is generally considered a genuine surgical emergency requiring urgent operative repair rather than medical management alone.

Fill in the blank: Stanford type A dissection involves the ________ aorta, carrying considerably higher immediate mortality risk.

5.4.2 clinical presentation and diagnosis of aortic dissection

A genuinely dramatic presentation demanding urgent recognition

Aortic dissection classically presents with sudden-onset, severe, tearing chest or back pain, often described as the worst pain the patient has ever experienced, sometimes migrating as the dissection extends along the length of the aorta, and can present with a genuinely wide range of associated features reflecting compromise of branch vessels arising from the aorta, including limb ischaemia, stroke, or, where the coronary arteries are involved, myocardial infarction.

Diagnosis relies on **CT angiography**, directly visualising the dissection flap and false lumen, and, given the genuinely broad differential diagnosis a patient with severe chest pain can present with, including the acute coronary syndrome and pulmonary embolism already discussed elsewhere in this course, maintaining a genuine index of suspicion for aortic dissection, particularly with the specific tearing pain character and any asymmetry in limb pulses or blood pressure, is essential to avoid a genuinely catastrophic missed diagnosis.

Fill in the blank: CT angiography directly visualises the dissection flap and false ________.

5.4.3 management of aortic dissection

Surgery for type A, careful medical control for type B

Type A dissection requires urgent surgical repair, replacing the affected segment of aorta, given the genuinely high risk of catastrophic complication, including aortic rupture or cardiac tamponade, if managed conservatively, while awaiting surgery, aggressive blood pressure and heart rate control genuinely reduces ongoing stress on the fragile, dissected aortic wall and the risk of further extension before definitive surgical treatment can be performed.

Type B dissection, in the absence of complication such as malperfusion of a branch vessel, is generally managed medically with careful blood pressure and heart rate control, following similar principles to the hypertensive emergency management already discussed earlier in this Series, with surgical or endovascular intervention reserved for complicated cases, and this Series, and the entire course, closes on the same theme of matching treatment urgency and intensity precisely to the specific severity and complexity of the individual presentation.

Fill in the blank: Type B dissection in the absence of complication is generally managed ________, with intervention reserved for complicated cases.

Table 5.2: Comparing Stanford type A and type B aortic dissection

Feature	Type A	Type B
Aortic segment involved	Ascending aorta	Descending aorta only
Typical management	Urgent surgical repair	Medical management unless complicated
Mortality risk	Considerably higher	Lower, but genuine risk remains

Pilot questions 5.4

1. Describe aortic dissection and the false lumen it creates. (Linked to SLO 5.7)
2. Distinguish Stanford type A from type B dissection. (Linked to SLO 5.7)
3. Describe the classic clinical presentation of aortic dissection. (Linked to SLO 5.7)
4. Describe the key investigation confirming aortic dissection. (Linked to SLO 5.7)
5. Distinguish the management of type A from type B dissection. (Linked to SLO 5.8)

Series Summary

This final Series closed the course with the genuine emergencies of cardiology. Part 5.1 covered cardiac arrest, resuscitation, and post-resuscitation care, while Part 5.2 turned to cardiogenic shock and acute pulmonary oedema. Part 5.3 covered hypertensive emergency, and Part 5.4 closed with aortic dissection.

You should now be able to describe and manage cardiac arrest, cardiogenic shock, acute pulmonary oedema, hypertensive emergency, and aortic dissection across all four Parts of this Series, meeting the outcomes set for this Series. Across the full course, Series 1 covered pathological functional states of the heart, systolic and diastolic dysfunction, and arrhythmias, Series 2 covered ischaemic heart disease and cardiomyopathies, Series 3 covered peripheral vascular disease and venous thromboembolism, Series 4 covered cardiovascular infections, congenital abnormalities, and valvular disease, and this Series has closed the course with emergencies in cardiology, together forming a comprehensive foundation in Cardiology II.

Glossary of Terms

1. **Acute pulmonary oedema:** fluid accumulation in the lung tissue from acutely elevated left atrial pressure.
2. **Advanced life support:** structured resuscitation combining airway management, access, and medication.
3. **Aortic dissection:** a tear in the aortic wall creating a false lumen alongside the true lumen.
4. **Cardiac arrest:** sudden cessation of effective cardiac output.
5. **Cardiogenic shock:** inadequate tissue perfusion from severe cardiac pump failure.
6. **False lumen:** the abnormal channel created within the aortic wall by a dissection.
7. **Hypertensive emergency:** severely elevated blood pressure with acute, progressive end-organ damage.
8. **Hypertensive urgency:** severely elevated blood pressure without acute end-organ damage.
9. **Mechanical circulatory support:** temporary devices assisting or replacing the heart's pumping function.
10. **Return of spontaneous circulation:** restoration of an effective, sustained cardiac rhythm following arrest.
11. **Stanford classification:** a system classifying aortic dissection as type A or type B.
12. **Targeted temperature management:** controlled body temperature regulation improving neurological outcome after arrest.

Concept Check Questions [Series 5]

Objective questions

- Cardiac arrest most commonly results from ventricular fibrillation or pulseless ventricular ________. (Linked to SLO 5.1)
- Cardiogenic shock most commonly follows extensive myocardial ________. (Linked to SLO 5.3)
- Hypertensive emergency involves a failure of normal vascular ________, ordinarily protecting end organs. (Linked to SLO 5.5)
- Stanford type A dissection involves the ________ aorta, carrying considerably higher immediate mortality risk. (Linked to SLO 5.7)
- Type B dissection in the absence of complication is generally managed ________, with intervention reserved for complicated cases. (Linked to SLO 5.8)

Short-answer questions

1. Distinguish shockable from non-shockable rhythms. (Linked to SLO 5.2)
2. Describe the presentation of acute pulmonary oedema. (Linked to SLO 5.4)
3. Distinguish hypertensive emergency from hypertensive urgency. (Linked to SLO 5.5)
4. List the organ systems that can be affected by end-organ damage in hypertensive emergency. (Linked to SLO 5.5)
5. Describe the classic clinical presentation of aortic dissection. (Linked to SLO 5.7)

Long-answer questions

6. Discuss the causes, recognition, and principles of resuscitation and post-resuscitation care in cardiac arrest. (Linked to SLO 5.1, 5.2)
7. Describe the pathophysiology, presentation, and management of cardiogenic shock and acute pulmonary oedema. (Linked to SLO 5.3, 5.4)
8. Discuss the definition, presentation, and management of hypertensive emergency. (Linked to SLO 5.5, 5.6)
9. Describe the pathophysiology, classification, presentation, and diagnosis of aortic dissection. (Linked to SLO 5.7)
10. Discuss the management of aortic dissection. (Linked to SLO 5.8)

Answers to Concept Check Questions [Series 5]

Objective questions

11. Tachycardia.
12. Infarction.
13. Autoregulation.
14. Ascending.
15. Medically.

Short-answer questions

16. Shockable rhythms are ventricular fibrillation and pulseless ventricular tachycardia; non-shockable are asystole and pulseless electrical activity.
17. Acute pulmonary oedema presents with severe breathlessness, orthopnoea, pink frothy sputum, and widespread crackles.
18. Hypertensive emergency involves acute end-organ damage, while hypertensive urgency does not.
19. Systems affected include the brain, eyes, kidneys, and heart.
20. Aortic dissection presents with sudden, severe, tearing chest or back pain, sometimes migrating.

Long-answer questions

21. **Basic response:** Cardiac arrest most commonly results from ventricular fibrillation or pulseless ventricular tachycardia; recognition relies on rapid assessment without waiting for pulse confirmation; resuscitation follows structured basic and advanced life support algorithms; post-resuscitation care addresses the underlying cause and targeted temperature management.

    Value-added response: A value-added response notes that chest compression quality genuinely, measurably affects the likelihood of successful outcome.

22. **Basic response:** Cardiogenic shock is inadequate perfusion from pump failure, presenting with hypotension and cold peripheries, managed with haemodynamic support and revascularisation; acute pulmonary oedema presents with severe breathlessness, managed with diuresis and respiratory support.

    Value-added response: A value-added response notes that aggressive diuresis requires caution when both conditions coexist, given the risk of worsening hypoperfusion.

23. **Basic response:** Hypertensive emergency involves severely elevated blood pressure with acute end-organ damage across the brain, eyes, kidneys, and heart, managed with controlled, gradual intravenous blood pressure reduction.

    Value-added response: A value-added response notes that overly rapid blood pressure reduction can itself cause organ hypoperfusion and worsen the clinical picture.

24. **Basic response:** Aortic dissection is a tear creating a false lumen, classified by the Stanford system as type A or type B, presenting with sudden, severe, tearing pain, diagnosed with CT angiography.

    Value-added response: A value-added response notes that type A dissection carries considerably higher mortality given its risk of coronary, valvular, or pericardial involvement.

25. **Basic response:** Type A dissection requires urgent surgical repair; type B dissection is generally managed medically with blood pressure and heart rate control unless complicated.

    Value-added response: A value-added response notes that aggressive blood pressure control reduces ongoing stress on the dissected wall while awaiting surgery in type A dissection.

Answers to Pilot Questions [Series 5]

Part 5.1

1. Causes include ventricular fibrillation, pulseless ventricular tachycardia, asystole, and pulseless electrical activity.
2. Pulse checks are unreliable and can meaningfully delay life-saving chest compressions.
3. Basic life support comprises high-quality chest compressions, rescue breaths, and early defibrillation.
4. Shockable rhythms are ventricular fibrillation and pulseless ventricular tachycardia; non-shockable are asystole and pulseless electrical activity.
5. Targeted temperature management controls body temperature within a specific range, improving neurological outcome.

Part 5.2

6. Cardiogenic shock presents with profound hypotension, cold peripheries, reduced urine output, and altered mental status.
7. Prompt recognition distinguishes it from other shock causes, directly shaping the urgent, specific treatment pathway required.
8. Management includes fluid management, inotropic support, mechanical circulatory support, and treating the underlying cause.
9. Acute pulmonary oedema presents with severe breathlessness, orthopnoea, pink frothy sputum, and widespread crackles.
10. Aggressive diuresis can worsen hypoperfusion in a shocked patient given the already compromised circulatory state.

Part 5.3

11. Hypertensive emergency involves acute end-organ damage, while hypertensive urgency does not carry the same immediate urgency.
12. Failure of vascular autoregulation leads to endothelial damage and, in severe cases, fibrinoid necrosis of small vessels.
13. Systems affected include the brain, eyes, kidneys, and heart.
14. Fundoscopic findings provide direct visible evidence of end-organ damage, confirming a hypertensive emergency.
15. Overly rapid reduction can itself cause organ hypoperfusion and worsen the underlying clinical picture.

Part 5.4

1. Aortic dissection is a tear in the aortic wall creating a false lumen alongside the true lumen.
2. Type A involves the ascending aorta, while type B is confined to the descending aorta.
3. Aortic dissection presents with sudden, severe, tearing chest or back pain, sometimes migrating as the dissection extends.
4. CT angiography directly visualises the dissection flap and false lumen.
5. Type A requires urgent surgical repair, while type B is generally managed medically unless complicated.

References and Attributions [Series 5]

1. Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
2. Libby, P., Bonow, R. O., Mann, D. L., Tomaselli, G. F., & Braunwald, E. (2021). Braunwald's Heart Disease (12th ed.). Elsevier.
3. European Resuscitation Council (2021). Guidelines for Resuscitation. Resuscitation.
4. European Society of Cardiology (2014). Guidelines on the Diagnosis and Treatment of Aortic Diseases. European Heart Journal.

Figures, Plates, Tables, and Boxes

1. Figure 5.1: Clinicians performing cardiopulmonary resuscitation with an attached defibrillator. AI-generated using the prompt: "Create a realistic clinical illustration titled Cardiopulmonary Resuscitation in Progress, showing one clinician performing chest compressions on a patient while another attaches defibrillator pads, resuscitation room setting. Clean clinical illustration style, no text."
2. Table 5.1: Key features distinguishing cardiogenic shock from acute pulmonary oedema alone. AI-generated using the prompt: "Create a clean reference table titled Key Features Distinguishing Cardiogenic Shock from Acute Pulmonary Oedema Alone, listing feature, cardiogenic shock, and acute pulmonary oedema alone. Simple clinical reference table style with outside border."
3. Figure 5.2: A clinician performing fundoscopic examination to assess for hypertensive retinopathy. AI-generated using the prompt: "Create a realistic clinical illustration titled Fundoscopic Examination for Hypertensive Retinopathy, showing a clinician using a handheld ophthalmoscope to examine a seated patient's eye in a dimly lit examination room. Clean clinical illustration style, no text."
4. Table 5.2: Comparing Stanford type A and type B aortic dissection. AI-generated using the prompt: "Create a clean reference table titled Comparing Stanford Type A and Type B Aortic Dissection, listing feature, type A, and type B. Simple clinical reference table style with outside border."