

Outlines:

- Anatomy & physiology.
- History of common symptoms.

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Introductory: **Section 1, CH.4: CVS History Taking.**

Note: This summary **contains all Macleod's important notes.**

مراجعة

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Anatomy & Physiology

- The heart comprises **two muscular pumps** working in **series**, covered in a serous sac (**pericardium**) that allows free movement with each heart beat and respiration (Fig. 4.1).
- The **right heart** (right atrium and ventricle) pumps **deoxygenated blood** returning from the **systemic veins** into the **pulmonary circulation** at relatively **low pressures**.
- The **left heart** (left atrium and ventricle) receives blood from the lungs and pumps it round the body **to the tissues at higher pressures** (Fig. 4.2).
- **Atrioventricular valves** (tricuspid on the right side, mitral on the left) separate the atria from the ventricles.
- The **pulmonary valve** on the right side of the heart and the **aortic valve** on the left separate the ventricles from the pulmonary and systemic arterial systems, respectively.
- Cardiac contraction is coordinated by **specialized groups of cells**. The cells in the **sinoatrial node** normally act as the **cardiac pacemaker**. Subsequent **spread of impulses** through the heart ensures that atrial contraction is complete before ventricular contraction (systole) begins. At the end of systole, the ventricles relax and the atrioventricular valves open, allowing them to **refill** with **blood** from the atria (**diastole**).

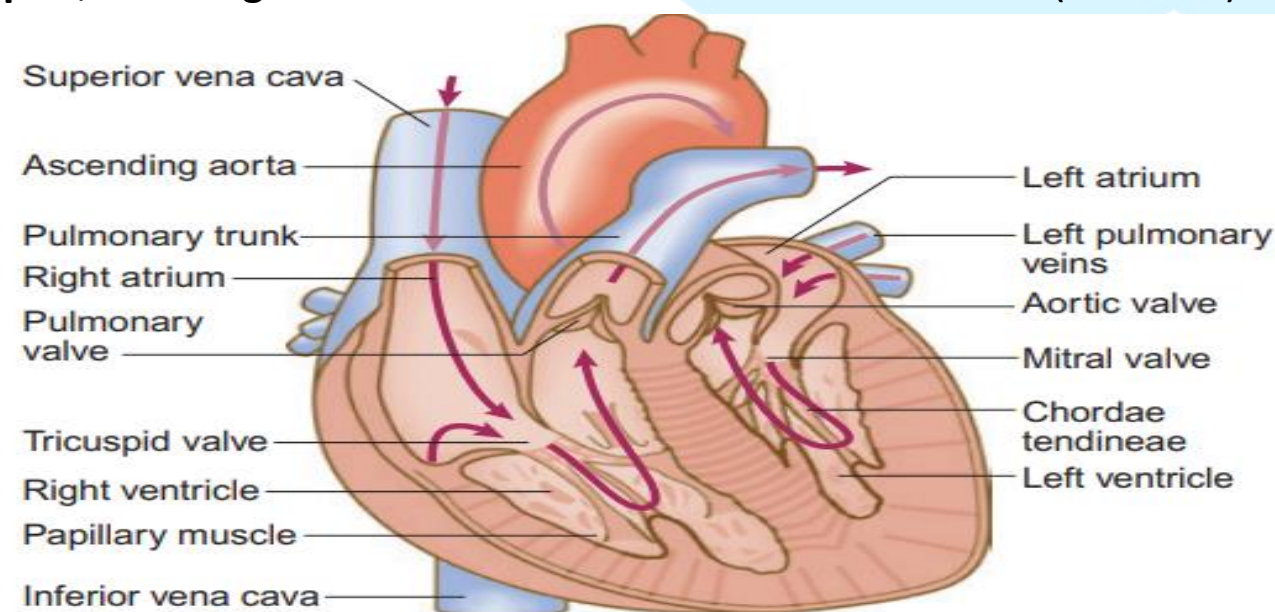


Fig. 4.1 The heart chambers and valves.

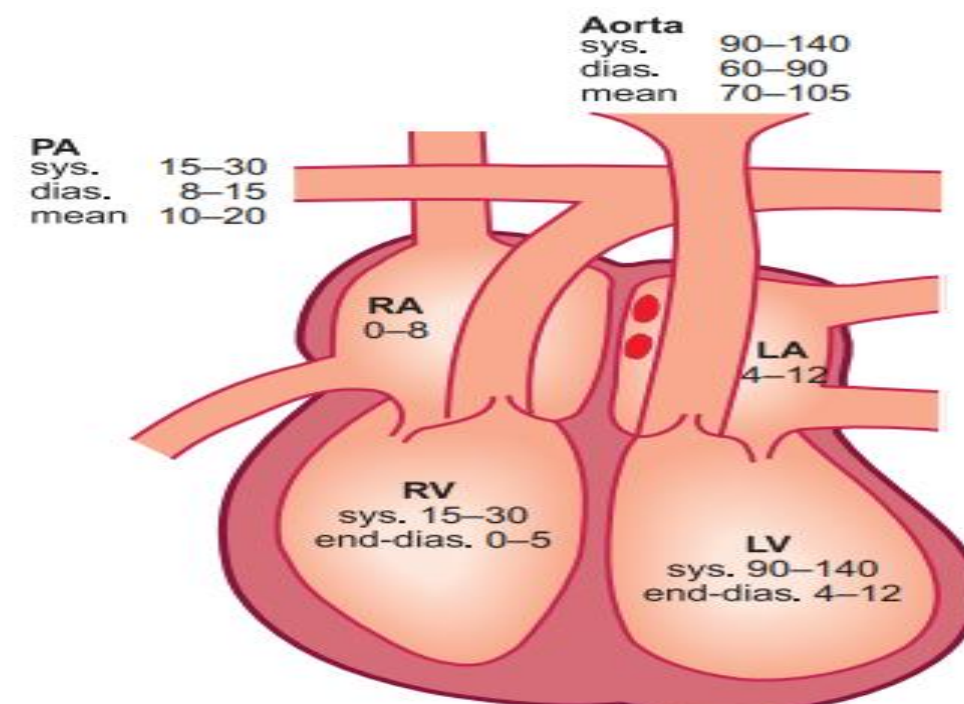


Fig. 4.2 Normal resting pressures (mmHg) in the heart and great vessels. dias., diastolic; LA, left atrium; LV, left ventricle; PA, pulmonary artery; RA, right atrium; RV, right ventricle; sys., systolic.

History

- Common presenting symptoms:

1. Chest pain.
2. Dyspnea.
3. Palpitation.
4. Syncope and presyncope.
5. Edema.

1. Chest pain:

(Types)

1- **Intermittent** (Angina Vs. Esophageal spasm).

2- **Acute:**

- Acute coronary syndrome.
- Aortic dissection.
- Pericarditis.
- Esophageal Spasm.
- Pneumothorax.
- Musculoskeletal pain.



2.2 Characteristics of pain (SOCRATES)

Site

- Somatic pain, often well localised, e.g. sprained ankle
- Visceral pain, more diffuse, e.g. angina pectoris

Onset

- Speed of onset and any associated circumstances

Character

- Described by adjectives, e.g. sharp/dull, burning/tingling, boring/stabbing, crushing/tugging, preferably using the patient's own description rather than offering suggestions

Radiation

- Through local extension
- Referred by a shared neuronal pathway to a distant unaffected site, e.g. diaphragmatic pain at the shoulder tip via the phrenic nerve (C₃, C₄)

Associated symptoms

- Visual aura accompanying migraine with aura
- Numbness in the leg with back pain suggesting nerve root irritation

Timing (duration, course, pattern)

- Since onset
- Episodic or continuous:
 - If episodic, duration and frequency of attacks
 - If continuous, any changes in severity

Exacerbating and relieving factors

- Circumstances in which pain is provoked or exacerbated, e.g. eating
- Specific activities or postures, and any avoidance measures that have been taken to prevent onset
- Effects of specific activities or postures, including effects of medication and alternative medical approaches

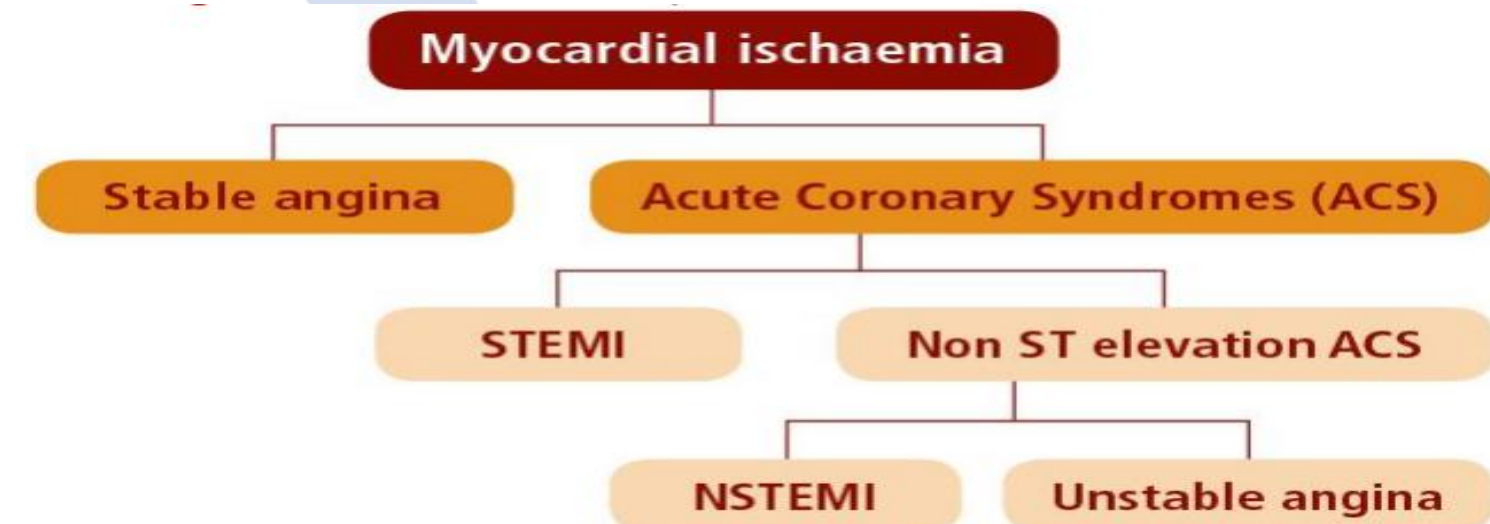
Severity

- Difficult to assess, as so subjective
- Sometimes helpful to compare with other common pains, e.g. toothache
- Variation by day or night, during the week or month, e.g. relating to the menstrual cycle

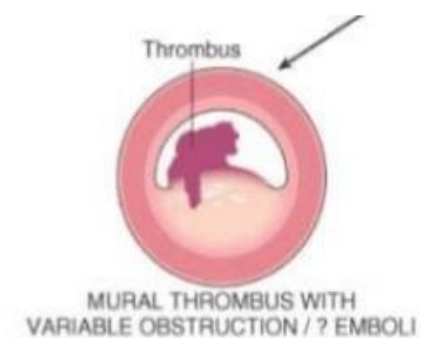
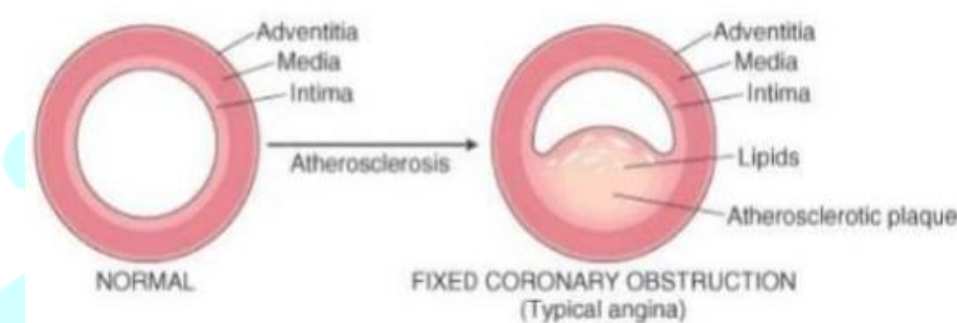
Intermittent chest pain

1- Angina:

- Chest pain due to inadequate oxygen supply to the heart muscle.
- Causes:
 1. Coronary atherosclerosis (Chronic fixed narrowing of the coronaries).
 2. Aortic stenosis.
 3. Hypertrophic cardiomyopathy.



- Types: Stable vs. unstable angina.
- Unstable angina: atherosclerotic plaque rupture with non-occlusive thrombus:
 1. Worsening in severity, frequency, less responsive to nitrates.
 2. Occur with minimal exertion or at rest.





Characteristics of Angina pain	
Site	Retrosternal
Onset	progressive , increase in intensity over 1-2 minutes
Character	Constricting, heavy
Radiation	Sometimes arm, neck, epigastrium
Associated features	Dyspnea
Timing	Intermittent, with episode lasting 2-10 minutes
Exacerbating/relieving factors	Triggered by emotion, exertion, cold, large meal Relieved by rest, nitrates
Severity	Mild to moderate

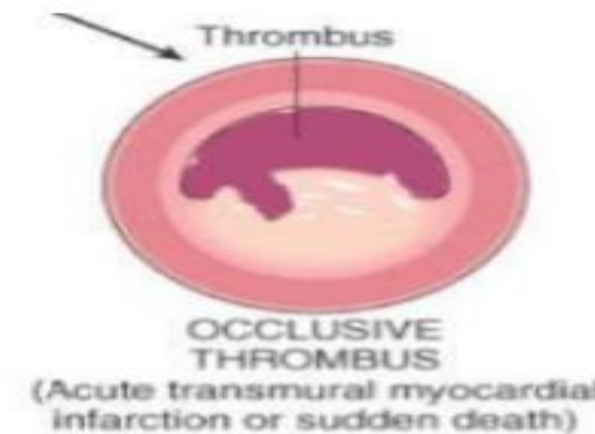
2- Esophageal spasm: (Caused by: Esophageal Spasm, GERD and Hiatal hernia).

Characteristics of Esophageal spasm	
Site	Retrosternal or epigastric
Onset	Over 1-2 minutes, can be sudden (spasm)
Character	Gripping, tight or burning
Radiation	Often to back , sometimes to arms
Associated symptoms	Heartburn, acid reflux
Timing	Intermittent, often at night-time, variable duration
Exacerbating/relieving factors	Triggered by lying flat and some food Not relieved by rest Nitrates sometimes relieve PPI/ Antacid.
Severity	Usually mild but Esophageal spasm can mimic MI

Acute chest pain:

1- ACS (Myocardial infarction)

- Atherosclerotic **plaque rupture** with **occlusive thrombus**.
- The **symptoms are more severe and prolonged** than angina.
- Positive autonomic symptoms: **nausea, vomiting, pallor, sweating**.
- Angor animi**: feeling of impending death.

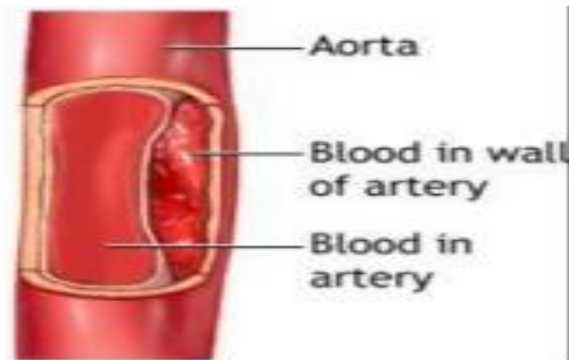


Characteristics of MI pain	
Site	Retrosternal
Onset	Rapid over a few minutes
Character	Constricting, heavy
Radiation	Often to arm(s), neck, jaw, sometimes epigastrium
Associated features	Sweating, nausea, vomiting, breathlessness, feeling of impending death (angor animi).
Timing	Acute presentation; prolonged duration.
Exacerbating/relieving factors	Stress' and exercise rare triggers, usually spontaneous. Not relieved by rest or nitrate
Severity	Usually severe



2- Aortic dissection:

- **Tear** in the **intima** of aorta.
- Associated with **profound autonomic stimulation**.
- If the tear involves the cranial or upper limb arteries, there may be associated **syncope, stroke, or upper limb pulse asymmetry**.
- Predisposing factors:
 1. HTN.
 2. CTD (Marfan syndrome).

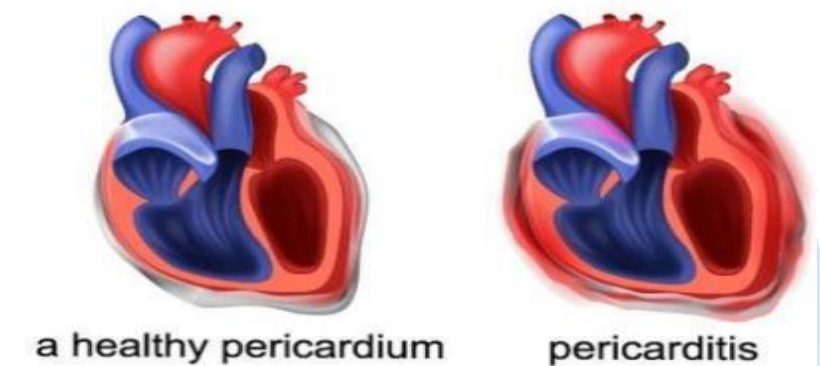


Characteristics of Aortic dissection pain

Site	Interscapular/retrosternal
Onset	Very sudden
Character	Tearing or ripping
Radiation	Back, between shoulders
Associated symptoms	Sweating, syncope, focal neurological signs, signs of limb ischemia, mesenteric ischemia
Timing	Acute presentation; prolonged duration
Exacerbating/relieving factors	Spontaneous No maneuver relieves pain
Severity	Very severe

3- Pericarditis:

- **Inflammation of the pericardium.**
- Causes:
 1. Viral infection.
 2. CTD.
 3. MI.
 4. After surgery, catheter ablation or radiotherapy.



Pericarditis Pain

Site	Retrosternal or left sided
Onset	Gradual , postural changes may suddenly aggravate
Character	Sharp, stabbing
Radiation	Left shoulder or back
Associated symptoms	Flu-like prodrome, SOB, fever
Timing	Acute presentation, variable duration
Exacerbating/relieving factors	Sitting up/lying down, inspiration worsen pain Relieved by NSAID, leaning forward
Severity	Can be severe

2. Dyspnea (breathlessness):

- Unpleasant **awareness** of breathing.
- **Acute** vs. **Chronic**.
- Causes of acute SOB:
 1. **Heart failure most common cause** (Acute or chronic).
 2. **Pulmonary embolism**.
 3. **Arrhythmias**.

4.4 Some mechanisms and causes of heart failure	
Mechanism	Cause
Reduced ventricular contractility (systolic dysfunction)	<ul style="list-style-type: none"> • Myocardial infarction • Dilated cardiomyopathy, e.g. (genetic, idiopathic, alcohol excess, cytotoxic drugs, peripartum cardiomyopathy) • Myocarditis
Impaired ventricular filling (diastolic dysfunction)	<ul style="list-style-type: none"> • Left ventricular hypertrophy • Constrictive pericarditis • Hypertrophic or restrictive cardiomyopathy
Increased metabolic and cardiac demand (rare)	<ul style="list-style-type: none"> • Thyrotoxicosis • Arteriovenous fistulae • Paget's disease
Valvular or congenital lesions	<ul style="list-style-type: none"> • Mitral and/or aortic valve disease • Tricuspid and/or pulmonary valve disease (rare) • Ventricular septal defect • Patent ductus arteriosus

• Angina equivalent:

1. **SOB caused by MI**.
2. May be accompanied with chest pain.
3. **Elderly, DM**.
4. Identical precipitant to angina and relieved with nitrate.

• Exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea:

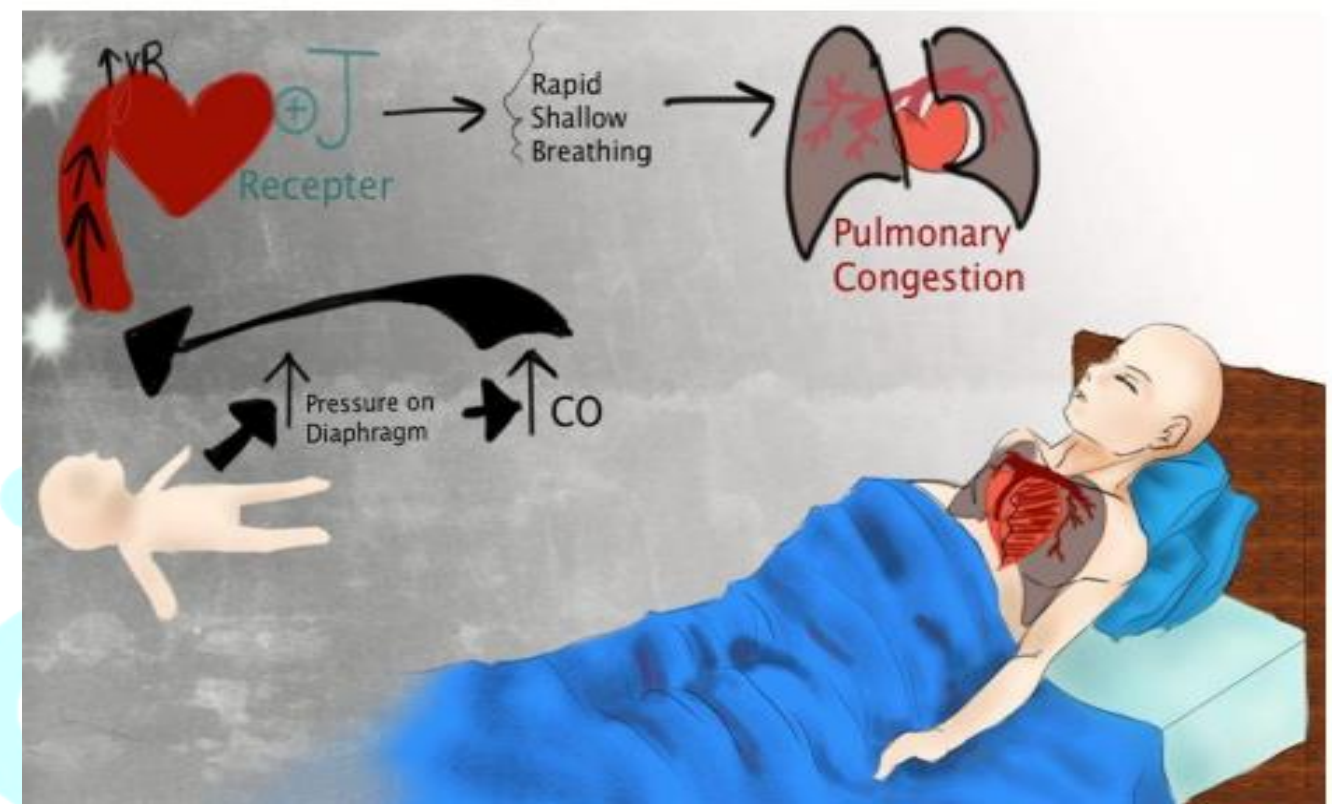
Exertional dyspnea: the **symptomatic hallmark of heart failure**.

- **NYHA grading system:** to assess the degree of **symptomatic limitation** caused by exertional SOB of heart failure.

4.5 New York Heart Association classification of heart failure symptom severity	
Class	Description
I	No limitations. Ordinary physical activity does not cause undue fatigue, dyspnea or palpitation (asymptomatic left ventricular dysfunction)
II	Slight limitation of physical activity. Such patients are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea or angina pectoris (symptomatically 'mild' heart failure)
III	Marked limitation of physical activity. Less than ordinary physical activity will lead to symptoms (symptomatically 'moderate' heart failure)
IV	Symptoms of congestive heart failure are present, even at rest . With any physical activity , increased discomfort is experienced (symptomatically 'severe' heart failure)

Orthopnea

- **Dyspnea on lying flat.**
- Mechanism: **increase venous return**.
- Severity is assessed by the **number of pillows used at night**.
- The **most severe form of dyspnea**.



Paroxysmal nocturnal dyspnea (PND)

- **SOB awaken patient from sleep.**
- Same mechanism of orthopnea.
- **Patient describe episode of choking or gasping for air, relieved by sitting.**
- **VS. Asthma attack** (which can also cause night-time dyspnea, chest tightness, cough and wheeze, but patients with **heart failure may also produce frothy white or blood-stained sputum**).

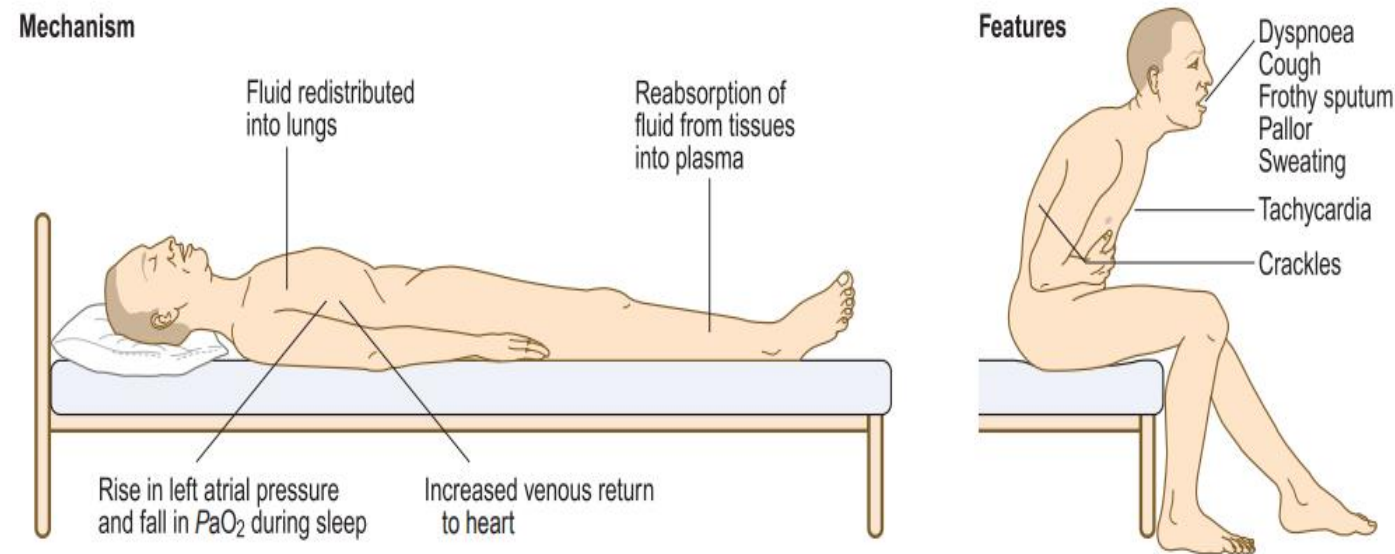


Fig. 4.3 Paroxysmal nocturnal dyspnoea.

Acute dyspnoea

Ask about:

- duration of onset
- Background symptoms of **exertional dyspnoea** and usual exercise tolerance
- Associated symptoms: **chest pain, syncope, palpitation or respiratory symptoms (cough, sputum, wheezes, haemoptysis).**

Chronic dyspnoea

• Ask about:

- Relationship between symptoms and **exertion**
- **degree of limitation caused by symptoms and their impact on everyday activities**
- **effect of posture on symptoms and/or episodes of nocturnal breathlessness**
- **associated symptoms: ankle swelling, cough, wheeze or sputum.**

3. Palpitation

- **Unexpected or unpleasant awareness of heart beating** in chest.
- Ask about:

1. **Nature of palpitation:** (Heart beats rapid, forceful, irregular).
2. **Timing of symptoms:** speed on onset and offset, frequency and duration of episodes.
3. **Precipitants for symptoms or relieving factors.**
4. Associated symptoms: **presyncope, syncope, chest pain.**
5. History of **cardiac disease.**

- In healthy people: - More common in **bed at night in slim people while lying on their left side.**

- **After exercise or in stressful situation** will be aware of their heart beating with normal sinus rhythm.

- **Extrasystoles:** - **Benign, at rest** Abolished by exercise underfilling alternating with overfilling of left ventricle.

- **Supraventricular tachycardia:** - Affects **young regular**, sudden **paroxysms.**

- **Ventricular tachycardia:** - Affects patients with **underlying cardiomyopathy, or previous MI.**

4.6 Descriptions of arrhythmias

	Extrasystoles	Sinus tachycardia	Supraventricular tachycardia	Atrial fibrillation	Ventricular tachycardia
Site	—	—	—	—	—
Onset	Sudden	★ Gradual	Sudden, with 'jump'	Sudden	Sudden
Character	★ 'Jump', missed beat or flutter	Regular, fast, 'pounding'	Regular, fast	★ Irregular, usually fast; slower in elderly	Regular, fast
Radiation	—	—	—	—	—
Associated features	Nil	Anxiety	★ Polyuria, lightheadedness, chest tightness	Polyuria, breathlessness; Syncope uncommon	★ Presyncope, syncope, chest tightness
Timing	Brief	A few minutes	Minutes to hours	Variable	Variable
Exacerbating/relieving factors	★ Fatigue, caffeine, alcohol may trigger Often relieved by walking (increases sinus rate)	Exercise or anxiety may trigger	★ Usually at rest, trivial movements, e.g. bending, may trigger Vagal manoeuvres may relieve	Exercise or alcohol may trigger; often spontaneous	Exercise may trigger; often spontaneous
Severity	Mild (usually)	Mild to moderate	Moderate to severe	Very variable, may be asymptomatic	★ Often severe

LEC 4: CVS History

High risk features for life-threatening arrhythmia

1. Previous **MI or cardiac surgery**.
2. **Associated** syncope or severe chest pain.
3. **Family hx of sudden death**.
4. **Wolff-Parkinson-white syndrome**.
5. Significant structural heart disease (**HCM, AS**).

4. Syncope/presyncope:

- **Syncope: Transient loss of consciousness** due to transient cerebral hypoperfusion.
- **Presyncope: Sensation of lightheadedness and impending loss of consciousness** without progressing to acute LOC.
- **Presyncope VS Dizziness or Vertigo** due to non-cardiovascular cause.

For syncope ask about:

- Ask about **witness**.
- **Circumstances of the event and any preceding symptoms** (palpitation, chest pain, lightheadedness, nausea, tinnitus, sweating and visual disturbance).
- **Duration of LOC, appearance of the patient while unconscious and any injuries sustained**.
- **Time to recovery** to full consciousness and normal cognition.
- **Current driving status, including occupational driving**.

For presyncope ask about:

- Exact **nature** of symptoms and **associated features** as palpitation.
- **Precipitants for symptoms** such as postural changes, prolonged standing, intense emotion or exertion.
- **Frequency** of episodes and **impact** on lifestyle.
- Possible contributing medications as **antihypertensive medications**.

Causes of syncope/presyncope

- Postural hypotension.
- Neurocardiogenic syncope.
- Hypersensitive carotid sinus syndrome (pressure over carotid sinus may lead to reflex bradycardia and syncope).
- Arrhythmia.
- Mechanical obstruction of cardiac output.
- In patients with hypersensitive carotid sinus syndrome, pressure over the carotid sinus may lead to reflex bradycardia and syncope.

Postural hypotension as a cause of syncope

- **A fall > 20 mmHg in systolic BP, > 10 mmHg in diastolic BP on standing with reflex tachycardia of 15-20 bpm increase in heart rate.**

Causes:

1. Hypovolemia.
2. Drugs.
3. **Autonomic neuropathy: Common in elderly affects one third, esp. above 65 years.**

Vasovagal syncope

- Mechanism: **abnormal autonomic reflexes produce a sudden bradycardia and/or vasodilatation.**
- In healthy people forced to **stand for a long time in warm environment or subject to painful or emotional stimuli such as sight of blood.**
- There is typically a prodrome of lightheadedness, tinnitus, nausea, sweating and facial pallor, and a darkening of vision from the periphery as the retinal blood supply (the most oxygen-sensitive part of the nervous system) is reduced. The person then **slides to the floor, losing consciousness.** When **laid flat to aid cerebral circulation** the individual **wakes up, often flushing from vasodilatation and nauseated or even vomiting due to vagal overactivity.**

The main differential diagnosis of syncope is seizure.





- **Don't hold the patient upright**, this **will worsen cerebral hypoperfusion**, leading to delays recovery and possible progression into seizure.

Arrhythmia as a cause of syncope

- Most common cause is **bradyarrhythmia**
1. **Stoke-Adams attacks**: episodic LOC secondary to sinoatrial disease or AV block.
 2. Rate limiting drugs.
 3. **Ventricular tachycardia** causes syncope more often than supraventricular tachycardia esp. in patient with impaired LV function.

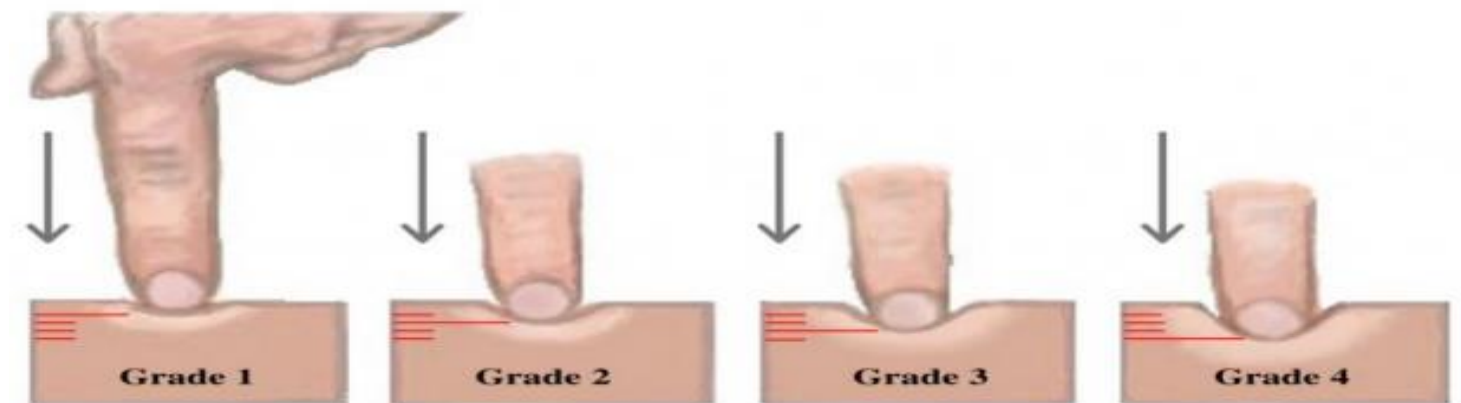
Mechanical obstruction to ventricle outflow as a cause of syncope

1. Left ventricular outflow obstruction (related to exertion).
 - **Severe aortic stenosis.**
 - **Hypertrophic cardiomyopathy.**
2. Right ventricular outflow obstruction.
3. Atrial myxoma, thrombosis of prosthetic heart valves.

4. Edema:

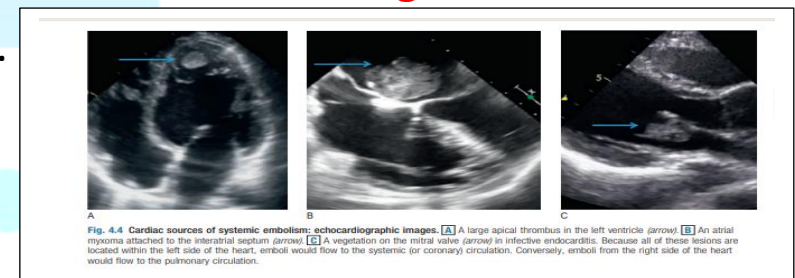
- Excess fluid in the interstitial space.
- Usually, **gravity dependent**.
- Where to look for? **Sacrum vs Ankle.**
- **Unilateral vs bilateral.**
- **Unilateral lower limb edema** may occur in **deep vein thrombosis**.
- **Heart failure** is a common cause of **bilateral lower limb edema** but other causes include **chronic venous disease**, **vasodilating calcium channel antagonists** (such as amlodipine) and **hypoalbuminemia**.
- An **elevated jugular venous pressure** strongly suggests a **cardiogenic cause of oedema**. Enquire about other symptoms of **fluid overload**, including **dyspnea, orthopnea and abdominal distension**.

Grade 1	0–2 mm indentation; rebounds immediately.
Grade 2	3–4 mm indentation; rebounds in < 15 seconds.
Grade 3	5–6 mm indentation; up to 30 seconds to rebound.
Grade 4	8 mm indentation; > 20 seconds to rebound.



Other symptoms of cardiac disease

- Non-specific symptoms; **weight loss, generalized weakness, fever, night sweats** (infective endocarditis).
- Symptoms of **stroke, acute mesenteric ischemia, acute limb ischemia** (patients with atria myxoma or infective vegetations).
- **Abdominal distension due to ascites, muscle wasting** due to **cardiac cachexia** (advanced heart failure).



Past medical history

- Ask about:
 1. Detailed record for any previous **cardiac disease, investigations, and interventions.**
 2. **Conditions** associated with **increased risk** of vascular diseases such as **hypertension, diabetes mellitus and hyperlipidemia.**
 3. **Rheumatic fever or heart murmur during childhood.**



4. Potential causes of **bacteremia** in patients with **suspected infective endocarditis** such as skin infection, recent dental work, intravenous drug use or penetrating trauma.

5. **Systemic disorders** with cardiac manifestations such as **connective tissue diseases** (pericarditis and Raynaud's phenomenon), **Marfan's syndrome** (aortic dissection) and **myotonic dystrophy** (atrioventricular block).

4.8 Key elements of the past cardiac history

	Ischaemic heart disease	Heart failure	Valvular disease
Baseline symptoms	Exertional angina? If so, ascertain functional limitation (see Box 4.2)/response to GTN spray	Dyspnoea, fatigue, ankle swelling Record usual functional status (see Box 4.5)	Often asymptomatic Exertional dyspnoea (common), chest pain or syncope
Major events	Previous myocardial infarction/unstable angina	Hospitalisation for decompensated heart failure Ventricular arrhythmias	Infective endocarditis Previous rheumatic fever
Investigations	Coronary angiography (invasive or computed tomography): presence, extent and severity of coronary artery disease Exercise electrocardiogram (or other stress test): evidence of inducible ischaemia? Exercise capacity and symptoms	Echocardiogram (\pm cardiac magnetic resonance imaging): left ventricular size, wall thickness and systolic function; valvular disease; right ventricular function	Echocardiogram (transthoracic \pm transoesophageal): nature and severity of valve lesion; ventricular size and function
Procedures	Percutaneous coronary intervention (angioplasty and stenting) Coronary artery bypass graft surgery	Implantable cardioverter-defibrillator Cardiac resynchronisation therapy	Surgical valve repair or replacement (note whether mechanical or bioprosthetic) Transcatheter valve procedures

GTN, glyceryl trinitrate.

Past drug history:

4.7 Symptoms related to medication

Symptom	Medication
Angina	Aggravated by thyroxine or drug-induced anaemia, e.g. aspirin or NSAIDs
Dyspnoea	Beta-blockers in patients with asthma Exacerbation of heart failure by beta-blockers, some calcium channel antagonists (verapamil, diltiazem), NSAIDs
Palpitation	Tachycardia and/or arrhythmia from thyroxine, β_2 stimulants, e.g. salbutamol, digoxin toxicity, hypokalaemia from diuretics, tricyclic antidepressants
Syncope/presyncope	Vasodilators, e.g. nitrates, alpha-blockers, ACE inhibitors and angiotensin II receptor antagonists Bradycardia from rate-limiting agents, e.g. beta-blockers, some calcium channel antagonists (verapamil, diltiazem), digoxin, amiodarone
Oedema	Glucocorticoids, NSAIDs, some calcium channel antagonists, e.g. nifedipine, amlodipine

ACE, angiotensin-converting enzyme; NSAIDs, non-steroidal anti-inflammatory drugs.

Family history

- Many cardiac disorders such as **cardiomyopathies have a genetic component**.
- Ask about **premature coronary artery disease in first-degree relatives** (<60 years in a female or <55 years in a male).
- Sudden unexplained death at a young age** may raise the possibility of a cardiomyopathy or inherited arrhythmia. Patients with **venous thrombosis may have inherited thrombophilia**, such as a factor V Leiden mutation. **Familial hypercholesterolemia** is associated with premature arterial disease.

Premature CAD

- In the patient:

CAD < 55 years in female, < 45 years in male.

- In the family first degree relative:

CAD < 65 years in female, < 55 years in male.

Social history

- Smoking** which is the strongest risk factor for coronary and peripheral arterial disease.
- Alcohol** can induce atrial fibrillation and, in excess, is associated with obesity, hypertension and dilated cardiomyopathy.
- Recreational drugs such as cocaine and amphetamines** can cause arrhythmias chest pain, occlusive and aneurysmal peripheral arterial disease and even myocardial infarction.
- Daily life activity and change of limitations.**
- Eligibility for certain **occupations** that have implications for public safety, such as commercial drivers and pilot.