University of Baghdad
College of Medicine
2024-2025

Title: cardiovascular History

Grade: third

Module: clinical History

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Date: 10/ 10/ 2024

Common presenting symptoms

4.1 Common symptoms of heart disease

Symptom	Cardiovascular causes	Other causes
Chest discomfort	Myocardial infarction Angina Pericarditis Aortic dissection	Oesophageal spasm Pneumothorax Musculoskeletal pain
Breathlessness	Heart failure Valvular disease Angina Pulmonary embolism Pulmonary hypertension	Respiratory disease Anaemia Obesity Anxiety
Palpitation	Tachyarrhythmias Ectopic beats	Anxiety Hyperthyroidism Drugs
Syncope/ presyncope	Arrhythmias Postural hypotension Aortic stenosis Hypertrophic cardiomyopathy Atrial myxoma	Simple faints Epilepsy Anxiety
Oedema	Heart failure Constrictive pericarditis Venous stasis Lymphoedema	Nephrotic syndrome Liver disease Drugs Immobility

Chest pain

➢ Intermittent chest pain :

- Chest pain due to angina is typically a dull discomfort, often described as a tight or pressing ‘band-like’ sensation akin to a heavy weight. It tends to be felt diffusely across the anterior chest and may radiate down one or both arms and into the throat, jaw or teeth.
- In stable angina episodes of pain are precipitated by exertion and may occur more readily when walking in cold or windy weather, after a large meal or while carrying a heavy load;
- the pain is promptly relieved by rest and/or sublingual glyceryl nitrate (GTN) spray, and typically lasts for less than 10 minutes.

- The degree of physical exertion required to precipitate symptoms is a better guide to disease severity than the intensity of discomfort.
- In unstable angina, there is usually an abrupt onset or worsening of chest pain episodes that may occur on minimal exertion or at rest.
- Ask about:

✓ site, onset, severity and character of the pain, and whether the pain radiates anywhere.

✓ associated symptoms such as breathlessness.

✓ aggravating and relieving factors, especially their relationship to exertion.

✓ frequency and duration of symptoms, and any recent change in pattern.

✓ degree of limitation caused by symptoms.

Acute chest pain

- Myocardial infarction causes symptoms that are similar to, but more severe and prolonged than, those of angina pectoris.Associated features include restlessness, breathlessness and a feeling of impending death, sweating, pallor, nausea and vomiting.
- Pericardial pain is typically a constant anterior central chest pain that may radiate to the shoulders. It tends to be sharp or stabbing in character, exacerbated by inspiration or lying down, and relieved by sitting forwards.

- Aortic dissection (a tear in the intima of the aorta) is usually associated with abrupt onset of very severe, tearing chest pain that can radiate to the back (typically the interscapular region) and may be 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.

4.3 Cardiovascular causes of chest pain and their characteristics

	Angina	Myocardial infarction	Aortic dissection	Pericardial pain	Oesophageal pain
Site	Retrosternal	Retrosternal	Interscapular/retrosternal	Retrosternal or left-sided	Retrosternal or epigastric
Onset	Progressive increase in intensity over 1–2 minutes	Rapid over a few minutes	Very sudden	Gradual; postural change may suddenly aggravate	Over 1–2 minutes; can be sudden (spasm)
Character	Constricting, heavy	Constricting, heavy	Tearing or ripping	Sharp, 'stabbing', pleuritic	Gripping, tight or burning
Radiation	Sometimes arm(s), neck, epigastrium	Often to arm(s), neck, jaw, sometimes epigastrium	Back, between shoulders	Left shoulder or back	Often to back, sometimes to arms
Associated features	Breathlessness	Sweating, nausea, vomiting, breathlessness, feeling of impending death (angor animi)	Sweating, syncope, focal neurological signs, signs of limb ischaemia, mesenteric ischaemia	Flu-like prodrome, breathlessness, fever	Heartburn, acid reflux

4.3 Cardiovascular causes of chest pain and their characteristics

	Angina	Myocardial infarction	Aortic dissection	Pericardial pain	Oesophageal pain
Timing	Intermittent, with episodes lasting 2–10 minutes	Acute presentation; prolonged duration	Acute presentation; prolonged duration	Acute presentation; variable duration	Intermittent, often at night-time; variable duration
Exacerbating/ relieving factors	Triggered by emotion, exertion, especially if cold, windy Relieved by rest, nitrates	'Stress' and exercise rare triggers, usually spontaneous Not relieved by rest or nitrates	Spontaneous No manœuvres relieve pain	Sitting up/lying down may affect intensity NSAIDs help	Lying flat/some foods may trigger Not relieved by rest; nitrates sometimes relieve
Severity	Mild to moderate	Usually severe	Very severe	Can be severe	Usually mild but oesophageal spasm can mimic myocardial infarction
Cause	Coronary atherosclerosis, aortic stenosis, hypertrophic cardiomyopathy	Plaque rupture and coronary artery occlusion	Thoracic aortic dissection rupture	Pericarditis (usually viral, also post myocardial infarction)	Oesophageal spasm, reflux, hiatus hernia

NSAIDs, *non-steroidal anti-inflammatory drugs.*

Dyspnoea (breathlessness)

- CV causes of dyspnea:

✓ Heart failure\pulmonary oedema(patients prefer to be upright).

✓Pulmonary embolism(patients often more comfortable lying flat and may faint (syncope) if made to sit upright.

✓arrhythmias.

✓Dyspnoea caused by myocardial ischemia is known as ‘angina equivalent’.

Some patterns of dyspnea

- Exertional dyspnea is the symptomatic hallmark of chronic 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, dyspnoea 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, dyspnoea 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, may occur in patients with heart failure, where it signifies advanced disease or decompensation. The severity can be graded by the number of pillows used at night: ‘three-pillow orthopnea’, for example.
- Paroxysmal nocturnal dyspnea PND , resulting in sudden breathlessness that wakes the patient from sleep. Patients may choke or gasp for air, sit on the edge of the bed and open windows in an attempt to relieve their distress. It may be confused with asthma, 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.

In acute dyspnoea, ask about:

- ✓duration of onset
- ✓ background symptoms of exertional dyspnea and usual exercise tolerance.
- ✓ associated symptoms: chest pain, syncope, palpitation or respiratory symptoms (such as cough, sputum, wheeze or hemoptysis).
- ✓In patients with chronic symptoms, ask about:
- ✓• relationship between symptoms and exertion.
- ○ degree of limitation caused by symptoms and their impact on daily activities.
- ○ effect of posture on symptoms and/or episodes of nocturnal breathlessness.
- ○ associated symptoms: ankle swelling, cough, wheeze or sputum.

Palpitation

- It is an unexpected or unpleasant awareness of the heart beating in the chest. Ask about:

✓nature of the palpitation: is the heart beat rapid, forceful or irregular? Can the patient tap it out?

✓ timing of symptoms: speed of onset and offset; frequency and duration of episodes

✓precipitants for symptoms or relieving factors

✓ associated symptoms: presyncope, syncope or chest pain

✓ history of underlying cardiac disease.

- High-risk features that increase the likelihood of a life-threatening arrhythmia such as ventricular tachycardia include:
- • previous myocardial infarction or cardiac surgery.
- • associated syncope or severe chest pain.
- • family history of sudden death.
- • Wolff–Parkinson–White syndrome.
- • significant structural heart disease such as hypertrophic cardiomyopathy or aortic stenosis.

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 manœuvres 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

Syncope and presyncope

- Syncope is a transient loss of consciousness due to transient cerebral hypoperfusion and episodes are typically characterized by rapid onset, short duration, and spontaneous complete recovery.
- Presyncope is a sensation of lightheadedness and impending loss of consciousness without progressing to actual loss of consciousness.
- Causes include postural hypotension, neurocardiogenic syncope, arrhythmias and mechanical obstruction to cardiac output.

In syncope ask about:

- circumstances of the event and any preceding symptoms:

palpitation, chest pain, lightheadedness, nausea, tinnitus,

- sweating or visual disturbance
- duration of loss of consciousness, appearance of the patient while unconscious and any injuries sustained (a detailed witness history is extremely helpful)
- time to recovery of full consciousness and normal cognition.
- current driving status, including occupational driving.

In patients with presyncope (lightheadedness or dizziness) ask about:

- exact nature of symptoms and associated features such as palpitation
- precipitants for symptoms, such as postural change, prolonged standing, intense emotion or exertion.
- frequency of episodes and impact on lifestyle
- •possible contributing medications, such as antihypertensive agents

- Postural hypotension, a fall of more than 20 mmHg in systolic blood pressure on standing, may lead to syncope or presyncope. It can be caused by hypovolemia, drugs or autonomic neuropathy and is common in the elderly.
- Reflex or neurocardiogenic syncope results from excessive autonomic reflexes which produce sudden bradycardia and/or vasodilatation.
- Vasovagal syncope is the most common form of reflex syncope and may be triggered in healthy people following a period of prolonged standing or a painful or emotional stimulus, such as the sight of blood.

Oedema

- Excess fluid in the interstitial space causes oedema (tissue swelling). It is usually gravity-dependent and so is seen especially around the ankles, or over the sacrum in patients lying in bed.
- Heart failure is a common cause of bilateral lower limb oedema, but other causes include chronic venous disease, vasodilating calcium channel antagonists (such as amlodipine) and hypoalbuminemia.
- Unilateral lower limb oedema may occur in deep vein thrombosis.

Non-specific symptoms of cardiac disease

- weight loss, tiredness, fever and night sweats.
- symptoms of stroke , acute limb ischemia or acute mesenteric ischemia.
- Abdominal distension due to ascites, or weight loss and muscle wasting (‘cardiac cachexia’) due to a prolonged catabolic state.

Past medical history

- Obtaining a detailed record of any previous cardiac disease, investigations and interventions is essential.
- Also ask about:

✓ conditions associated with increased risk of vascular disease such as HT, DM and hyperlipidemia

✓rheumatic fever or heart murmurs during childhood

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

✓systemic disorders with cardiovascular manifestations such as connective tissue diseases (pericarditis and Raynaud's phenomenon), Marfan's syndrome (aortic dissection) and myotonic dystrophy (atrioventricular block).

Drug history

- Drugs may cause or aggravate symptoms such as breathlessness, chest pain, oedema, palpitation or syncope .
- Ask about ‘over-the-counter medications or herbal medicines.

4.8 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.

Social history

- Smoking 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.
- cocaine and amphetamines can cause arrhythmias, chest pain, occlusive and aneurysmal PAD and even myocardial infarction.
- ask about undue stress or anxiety as these are commonly associated with cardiac-type symptoms including chest pain, Dyspnoea and palpitation.

PERIPHERAL ARTERIAL SYSTEM

- The underlying pathology in PAD is usually atherosclerosis affecting large and medium-sized vessels. PAD affects the legs eight times more commonly than the arms.
- The Fontaine classification describes the progression of symptoms that occurs as the atherosclerotic burden increases and the blood supply to the limb diminishes.

4.20 Fontaine classification of lower limb ischaemia

Stage	Description
I	Asymptomatic
II	Intermittent claudication
III	Night/rest pain
IV	Tissue loss (ulceration/gangrene)

Intermittent claudication

- It is a pain felt in the legs on walking due to arterial insufficiency and is the most common symptom of PAD. Patients describe tightness or ‘cramp-like’ pain that develops after a relatively constant distance.
- The pain disappears completely within a few minutes of rest but recurs on walking. The ‘claudication distance’ is how far patients say they can walk before the pain comes on. The ‘total walking distance’ is how far they can walk before the pain is so bad that they have to stop.

- It is important to distinguish claudication due to arterial insufficiency from other causes of lower limb pain, which include osteoarthritis, neurogenic claudication and venous claudication.

4.21 The clinical features of arterial, neurogenic and venous claudication

	Arterial	Neurogenic	Venous
Pathology	Stenosis or occlusion of major lower limb arteries	Lumbar nerve root or cauda equina compression (spinal stenosis)	Obstruction to the venous outflow of the leg due to iliofemoral venous occlusion
Site of pain	Muscles, usually the calf but may involve thigh and buttocks	Ill-defined Whole leg May be associated with numbness and tingling	Whole leg 'Bursting' in nature
Laterality	Unilateral or bilateral	Often bilateral	Nearly always unilateral
Onset	Gradual after walking the 'claudication distance'	Often immediate on walking or standing up	Gradual, from the moment walking starts
Relieving features	On stopping walking, the pain disappears completely in 1–2 minutes	Bending forwards and stopping walking Patient may sit down for full relief	Leg elevation
Colour	Normal or pale	Normal	Cyanosed Often visible varicose veins
Temperature	Normal or cool	Normal	Normal or increased
Oedema	Absent	Absent	Always present
Pulses	Reduced or absent	Normal	Present but may be difficult to feel owing to oedema
Straight-leg raising	Normal	May be limited	Normal

Night pain

- The patient is woken with pain or numbness in the affected foot due to poor perfusion. Night pain develops because on lying, the beneficial effects of gravity on perfusion are lost, and in addition, heart rate, BP and cardiac output are reduced during sleep.
- Patients may find relief by hanging the leg out of bed or by getting up and walking around.

Rest pain

- Rest pain occurs when blood flow is insufficient to meet the metabolic demands of the tissues, even at rest. Critical ischaemia is defined as rest pain (persisting for more than 2 weeks and requiring opiate analgesia) or tissue loss.
- In patients with diabetes it may be difficult to differentiate between rest pain and diabetic neuropathy, as both may be worse at night. Neuropathic pain may not be confined to the foot, is associated with burning and tingling, is not relieved by dependency and is accompanied by dysesthesia (pain or uncomfortable sensations, sometimes described as burning, tingling or numbness).

Tissue loss (ulceration and/or gangrene)

- In patients with severe lower limb PAD, perfusion is inadequate to support the tissues, and areas of tissue loss (gangrene) develop at the tips of the digits, gradually spreading proximally.

Acute limb ischaemia

- The classical features of acute limb ischaemia are the ‘six Ps’:

Pallor, pain, pulselessness and perishing cold (are relatively early signs). Paralysis (inability to move the toes/fingers) and paraesthesia (numbness or tingling over the forefoot or dorsum of the hand) are the most important and indicate severe ischaemia affecting nerve and muscle function.

- most likely underlying cause:

✓Thromboembolism: usually from the left atrium in association with atrial fibrillation or myocardial infarction. There is usually no history of claudication.

✓Thrombosis in situ: thrombotic occlusion of an already narrowed atherosclerotic arterial segment. In this situation the patient is likely to have a past history of claudication.

4.23 Acute limb ischaemia: embolus versus thrombosis in situ

	Embolus	Thrombosis
Onset and severity	Acute (seconds or minutes), ischaemia profound (no pre-existing collaterals)	Insidious (hours or days), ischaemia less severe (pre-existing collaterals)
Embolic source	Present	Absent
Previous claudication	Absent	Present
Pulses in contralateral leg	Present	Often absent, reflecting widespread peripheral arterial disease
Diagnosis	Clinical	Angiography
Treatment	Embolectomy and anticoagulation	Medical, bypass surgery, catheter-directed thrombolysis

Abdominal pain

- Mesenteric ischemia: Severe central abdominal pain typically develops 10–15 minutes after eating. The patient becomes scared of eating and significant weight loss is a universal finding. Diarrhea may also be present and the non-specific nature of symptoms may result in misdiagnosis.
- Presentation with severe abdominal pain, shock, bloody diarrhea and profound metabolic acidosis indicates infarction of the bowel, which carries a high mortality rate.
- Rarely, renal angle pain occurs from renal infarction or ischemia, and is associated with visible or non-visible hematuria.

Abdominal aortic aneurysm AAA

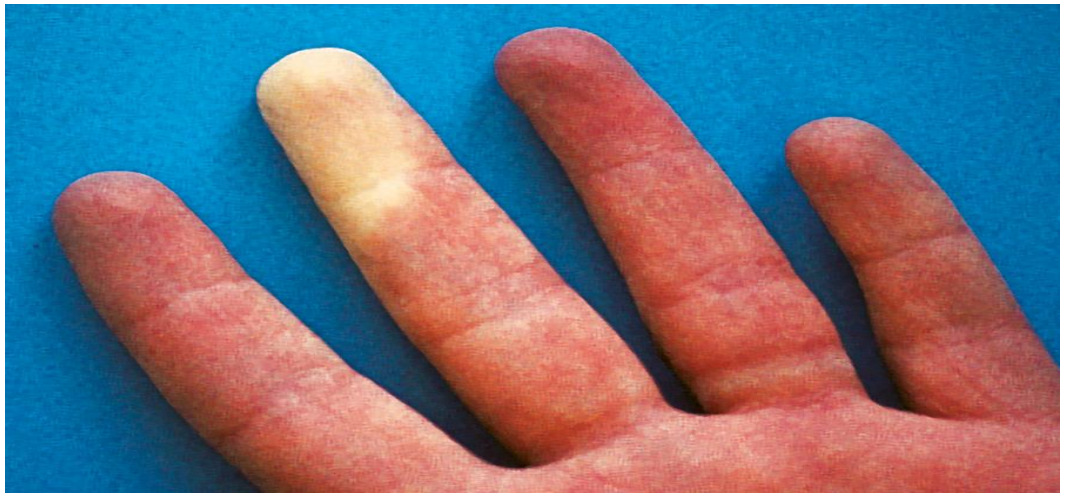
- abdominal and/or back pain, or occasionally with more subtle signs such as an awareness of abdominal pulsation. However, most patients are asymptomatic until the aneurysm ruptures.
- The classical features of AAA rupture include abdominal/ backpain, pulsatile abdominal mass, syncope and shock (hypotension).

Digital ischaemia

- Blue toe syndrome occurs when there is atheroembolism from an AAA or alternative proximal embolic source (such as popliteal aneurysm or atherosclerotic plaque). Patchy bluish discoloration appears over the toes and forefoot of one or both feet.

Vasospastic symptoms

- Raynaud's phenomenon is digital ischaemia induced by cold and
✓emotion. It has three phases (Fig. 4.32):
✓pallor: due to digital artery spasm and/or obstruction
✓ cyanosis: due to deoxygenation of static venous blood (this phase may be absent)
✓ redness: due to reactive hyperemia.



Past medical history

- peripheral vascular disease?
- Ask about previous investigations, operations or procedures.
- Is there a history of other atherosclerotic conditions such as coronary artery disease or cerebrovascular disease?
- Ask about risk factors for atherosclerotic disease, including hypertension, hypercholesterolemia and diabetes mellitus.
- Are there any other comorbidities (such as severe cardiac or lung disease).

Drug history

- Ask about medication used for secondary prevention and adherence to these: antiplatelet, lipid-lowering, antihypertensive and diabetes therapies.
- Ask about drugs for claudication, cardiac medication, anticoagulant.

Family history

- Ask about a family history of premature coronary or other vascular
- disease. There is a strong familial association for AAAs so,where relevant, a family history should be sought.

Social history

- Take a smoking history . Ask about occupation and activities of daily living. How are the patient's symptoms impacting on quality of life or employment?