

## 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

## 4.2 Canadian Cardiovascular Society: functional classification of stable angina

Grade	Description
1	Ordinary physical activity, such as walking and climbing stairs, does not cause angina. Angina with strenuous, rapid or prolonged exertion at work or during recreation
2	Slight limitation of ordinary activity. Walking or climbing stairs rapidly, walking uphill, walking or climbing stairs after meals, in cold, in wind, or when under emotional stress, or only during the few hours after awakening
3	Marked limitation of ordinary physical activity. Walking 1–2 blocks on the level and climbing less than one flight in normal conditions
4	Inability to carry on any physical activity without discomfort; angina may be present at rest

### 4.3 Cardiovascular causes of chest pain and their characteristics

	Angina	Myocardial infarction	Aortic dissection	Pericardial pain	Oesophageal pain
<u>Site</u>	Retrosternal	Retrosternal	Interscapular/retrosternal	Retrosternal or left-sided	Retrosternal or epigastric
<u>Onset</u>	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)
<u>Character</u>	Constricting, heavy	Constricting, heavy	Tearing or ripping	Sharp, 'stabbing', pleuritic	Gripping, tight or burning
<u>Radiation</u>	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
<u>Associated features</u>	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
<u>Timing</u>	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
<u>Exacerbating/relieving factors</u>	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 manoeuvres relieve pain	Sitting up/lying down may affect intensity NSAIDs help	Lying flat/some foods may trigger Not relieved by rest; nitrates sometimes relieve
<u>Severity</u>	Mild to moderate	Usually severe	Very severe	Can be severe	Usually mild but oesophageal spasm can mimic myocardial infarction
<u>Cause</u>	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*.

#### 4.4 Some mechanisms and causes of heart failure

Mechanism	Cause
Reduced ventricular contractility (systolic dysfunction)	Myocardial infarction Dilated cardiomyopathy, e.g. genetic, idiopathic, alcohol excess, cytotoxic drugs, peripartum cardiomyopathy Myocarditis
Impaired ventricular filling (diastolic dysfunction)	Left ventricular hypertrophy Constrictive pericarditis Hypertrophic or restrictive cardiomyopathy
Increased metabolic and cardiac demand (rare)	Thyrotoxicosis Arteriovenous fistulae Paget's disease
Valvular or congenital lesions	Mitral and/or aortic valve disease Tricuspid and/or pulmonary valve disease (rare) Ventricular septal defect Patent ductus arteriosus

#### 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)

## 4.6 Descriptions of arrhythmias

	<b>Extrasystoles</b>	<b>Sinus tachycardia</b>	<b>Supraventricular tachycardia</b>	<b>Atrial fibrillation</b>	<b>Ventricular tachycardia</b>
<u>S</u> ite	–	–	–	–	–
<u>O</u> nset	Sudden	Gradual	Sudden, with 'jump'	Sudden	Sudden
<u>C</u> haracter	'Jump', missed beat or flutter	Regular, fast, 'pounding'	Regular, fast	Irregular, usually fast; slower in elderly	Regular, fast
<u>R</u> adiation	–	–	–	–	–
<u>A</u> ssociated features	Nil	Anxiety	Polyuria, lightheadedness, chest tightness	Polyuria, breathlessness Syncope uncommon	Presyncope, syncope, chest tightness
<u>T</u> iming	Brief	A few minutes	Minutes to hours	Variable	Variable
<u>E</u> xacerbating/ 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
<u>S</u> everity	Mild (usually)	Mild to moderate	Moderate to severe	Very variable, may be asymptomatic	Often severe

## 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, $\beta_2$ 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.*

## 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 <a href="#">Box 4.2</a> )/response to GTN spray	Dyspnoea, fatigue, ankle swelling Record usual functional status (see <a href="#">Box 4.5</a> )	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*.

## 4.9 Causes of abnormal pulse rate or rhythm

Abnormality	Sinus rhythm	Arrhythmia
Fast rate (tachycardia, > 100 bpm)	Exercise Pain Excitement/anxiety Fever Hyperthyroidism Medication: Sympathomimetics, e.g. salbutamol Vasodilators	Atrial fibrillation Atrial flutter Supraventricular tachycardia Ventricular tachycardia
Slow rate (bradycardia, < 60 bpm)	Sleep Athletic training Hypothyroidism Medication: Beta-blockers Digoxin Verapamil, diltiazem	Carotid sinus hypersensitivity Sick sinus syndrome Second-degree heart block Complete heart block
Irregular pulse	Sinus arrhythmia Atrial extrasystoles Ventricular extrasystoles	Atrial fibrillation Atrial flutter with variable response Second-degree heart block with variable response



## 4.10 Haemodynamic effects of respiration

	Inspiration	Expiration
Pulse/heart rate	Accelerates	Slows
Systolic blood pressure	Falls (up to 10 mmHg)	Rises
Jugular venous pressure	Falls	Rises
Second heart sound	Splits	Fuses

## 4.11 Common causes of atrial fibrillation

- Hypertension
- Heart failure
- Myocardial infarction
- Thyrotoxicosis
- Alcohol-related heart disease
- Mitral valve disease
- Infection, e.g. respiratory, urinary
- Following surgery, especially cardiothoracic surgery

## 4.12 Causes of increased pulse volume

### Physiological

- Exercise
- Pregnancy
- Advanced age
- Increased environmental temperature

### Pathological

- Hypertension
- Fever
- Thyrotoxicosis
- Anaemia
- Aortic regurgitation
- Paget's disease of bone
- Peripheral atrioventricular shunt

## 4.13 British Hypertension Society classification of blood pressure (BP) levels

BP	Systolic BP (mmHg)	Diastolic BP (mmHg)
Optimal	<120	<80
Normal	<130	<85
High normal	130–139	85–89
<b>Hypertension</b>		
Grade 1 (mild)	140–159	90–99
Grade 2 (moderate)	160–179	100–109
Grade 3 (severe)	>180	>110
<b>Isolated systolic hypertension</b>		
Grade 1	140–159	<90
Grade 2	>160	<90

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## 4.14 Clinical clues to secondary hypertension

Clinical feature	Cause
Widespread vascular disease Renal bruit	Renovascular disease, including renal artery stenosis
Episodes of sweating, headache and palpitation	Phaeochromocytoma
Hypokalaemia	Primary aldosteronism
Cushingoid facies, central obesity, abdominal striae, proximal muscle weakness Chronic glucocorticoid use	Cushing's syndrome
Low-volume femoral pulses with radiofemoral delay	Coarctation of the aorta
Bilateral palpable kidneys	Adult polycystic kidney disease (p. 243)

## 4.15 Differences between carotid artery and jugular venous pulsation

<b>Carotid</b>	<b>Jugular</b>
Rapid outward movement	Rapid inward movement
One peak per heart beat	Two peaks per heart beat (in sinus rhythm)
Palpable	Impalpable
Pulsation unaffected by pressure at the root of the neck	Pulsation diminished by pressure at the root of the neck
Independent of respiration	Height of pulsation varies with respiration
Independent of the position of the patient	Varies with the position of the patient
Independent of abdominal pressure	Rises with abdominal pressure

## 4.16 Abnormalities of the jugular venous pulse

Condition	Abnormalities
Heart failure	Elevation, sustained abdominojugular reflux > 10 seconds
Pulmonary embolism, tamponade	Elevation
Pericardial effusion	Elevation, <del>prominent</del> flattened 'y' descent
Pericardial constriction	Elevation, Kussmaul's sign, prominent 'y' descent 'x' descent
Superior vena cava obstruction	Elevation, loss of pulsation
Atrial fibrillation	Absent 'a' waves
Tricuspid stenosis	<del>Giant</del> 'a' waves Large, prominent
Tricuspid regurgitation	Giant 'v' or 'cv' waves
Complete heart block	'Cannon' waves

## 4.17 Cardiac auscultation: the **best sites** for hearing an abnormality

Site	Sound
Cardiac apex <ul style="list-style-type: none"> <li>• Midsystolic of M prolapse</li> <li>• Pansystolic of MR</li> <li>• Opening snap of MS</li> </ul>	First heart sound Third and fourth heart sounds Mid-diastolic murmur of mitral stenosis
Lower left sternal border  Apex ←	Early diastolic murmurs of aortic and <u>tricuspid</u> <span style="margin-left: 600px;">↓</span> <span style="margin-left: 600px;">pansystolic</span> <del>Opening snap of mitral stenosis</del> Pansystolic murmur of ventricular septal defect
Upper left sternal border $S_2$ splitting	Second heart sound Pulmonary valve murmurs
Upper right sternal border	Systolic ejection (outflow) murmurs, e.g. aortic stenosis, hypertrophic cardiomyopathy
Left axilla	Radiation of the pansystolic murmur of mitral regurgitation
Below left clavicle	Continuous 'machinery' murmur of a persistent patent ductus arteriosus

## 4.18 Abnormalities of intensity of the first heart sound

### Quiet

- Low cardiac output
- Poor left ventricular function
- Rheumatic mitral regurgitation
- Long P–R interval (first-degree heart block)

### Loud

- Increased cardiac output
- Large stroke volume
- Mitral stenosis
- Short P–R interval
- Atrial myxoma (rare)

### Variable

- Atrial fibrillation
- Extrasystoles
- Complete heart block



## 4.19 Abnormalities of the second heart sound

### Quiet

- Low cardiac output
- Calcific aortic stenosis
- Aortic regurgitation

### Loud

- Systemic hypertension (aortic component)
- Pulmonary hypertension (pulmonary component)

### Split

#### **Widens in inspiration (enhanced physiological splitting)**

- Right bundle branch block
- Pulmonary stenosis
- Pulmonary hypertension
- Ventricular septal defect

#### **Fixed splitting (unaffected by respiration)**

- Atrial septal defect

#### **Widens in expiration (reversed splitting)**

- Aortic stenosis
- Hypertrophic cardiomyopathy
- Left bundle branch block
- Ventricular pacing

## 4.20 Grades of intensity of murmur

Grade	Description
1	Heard by an expert in optimum conditions
2	Heard by a non-expert in optimum conditions
3	Easily heard; no thrill
4	A loud murmur, with a thrill
5	Very loud, often heard over a wide area, with thrill
6	Extremely loud, heard without a stethoscope

## 4.21 Causes of systolic murmurs

### Ejection systolic murmurs

- Increased flow through normal valves:
  - Severe anaemia, fever, athletes (bradycardia → large stroke volume), pregnancy
  - Atrial septal defect (pulmonary flow murmur)
  - Other causes of flow murmurs (increased stroke volume in aortic regurgitation)
- Normal or reduced flow through a stenotic valve:
  - Aortic stenosis
  - Pulmonary stenosis
- Subvalvular obstruction:
  - Hypertrophic obstructive cardiomyopathy

### Pansystolic murmurs

- Mitral regurgitation
- Tricuspid regurgitation
- Ventricular septal defect
- Leaking mitral or tricuspid prosthesis

### Late systolic murmurs

- Mitral valve prolapse

#### 4.23 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

## 4.22 Fontaine classification of lower limb ischaemia

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

## 4.24 Signs of acute limb ischaemia

- Pallor
- Pulselessness
- Perishing cold
- Paraesthesia
- Pain (worse when muscle squeezed)
- Paralysis

## 4.25 Acute limb ischaemia: embolus versus thrombosis in situ

	<b>Embolus</b>	<b>Thrombosis</b>
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

## 4.26 Signs suggesting vascular disease

Sign	Implication
<b>Hands and arms</b>	
Tobacco stains	Smoking
Purple discoloration of the fingertips	Atheroembolism from a proximal subclavian aneurysm
Pits and healed scars in the finger pulps	Secondary Raynaud's syndrome
Calcinosis and visible nail-fold capillary loops	Systemic sclerosis and CREST (calcinosis, Raynaud's phenomenon, oesophageal dysfunction, sclerodactyly, telangiectasia)
Wasting of the small muscles of the hand	Thoracic outlet syndrome
<b>Face and neck</b>	
Corneal arcus and xanthelasma	Hypercholesterolaemia
Horner's syndrome	Carotid artery dissection or aneurysm
Hoarseness of the voice and 'bovine' cough	Recurrent laryngeal nerve palsy from a thoracic aortic aneurysm
Prominent veins in the neck, shoulder and anterior chest	Axillary/subclavian vein occlusion
<b>Abdomen</b>	
Epigastric/umbilical pulsation	Aortoiliac aneurysm
Mottling of the abdomen	Ruptured abdominal aortic aneurysm or saddle embolism occluding aortic bifurcation
Evidence of weight loss	Visceral ischaemia

## 4.28 Clinical features of venous and arterial ulceration

Clinical feature	Venous ulceration	Arterial ulceration	Neuropathic ulceration
Sex	More common in women	More common in men	Equal in men and women
Risk factors	Thrombophilia, family history, previous deep vein thrombosis, varicose veins	Known peripheral vascular disease or risk factors for atherosclerotic disease, e.g. smoking, diabetes, dyslipidaemia, hypertension	Diabetes or other peripheral neuropathy (loss of sensation, loss of intrinsic foot muscle function, autonomic dysregulation)
Pain	Often painless but some patients have some pain that improves with elevating the leg	Severe pain, except in diabetics with neuropathy; improves on dependency	Painless or neuropathic pain
Site	Gaiter areas; 80% medial (long saphenous vein), 20% lateral (short saphenous vein)	Pressure areas (malleoli, heel, fifth metatarsal base, metatarsal heads and toes)	Pressure areas, sole of foot, tips of toes
Appearance	Shallow, irregular margin Slough on granulating base	Regular, 'punched out' Sloughy or necrotic base	Macerated, moist white skin surrounded by callus, often on load-bearing aspects (motor neuropathy)
Surrounding skin	Lipodermatosclerosis always present Oedema	Shiny, hairless, trophic changes	Dry due to reduced sweating (autonomic neuropathy)
Veins	Full and usually varicose	Empty with 'guttering' on elevation	Normal
Temperature	Warm Palpable pulses	Cold Absent pulses	Warm or cold due to autonomic neuropathy Palpable pulses



## 6.42 Features of deep vein thrombosis of the lower limb

Clinical feature	Non-occlusive thrombus	Occlusive thrombus
Pain	Often absent	Usually present
Calf tenderness	Often absent	Usually present
Swelling	Absent	Present
Temperature	Normal or slightly increased	Increased
Superficial veins	Normal	Distended
Pulmonary embolism	High risk	Low risk



## 4.29 Risk factors for deep vein thrombosis

- Obesity
- Smoking
- Recent bed rest or operations (especially to the leg, pelvis or abdomen)
- Recent travel, especially long flights
- Previous trauma to the leg, especially long-bone fractures, plaster of Paris splintage and immobilisation
- Pregnancy or features suggesting pelvic disease
- Malignant disease
- Previous deep vein thrombosis
- Family history of thrombosis
- Inherited thrombophilia, e.g. factor V Leiden
- Recent central venous catheterisation, injection of drug
- Use of oral contraceptive or hormone replacement therapy



## 6.38 Diseases associated with secondary Raynaud's syndrome

- Connective tissue syndromes, e.g. systemic sclerosis, CREST (calcinosis, Raynaud's phenomenon, oesophageal dysfunction, sclerodactyly, telangiectasia) and systemic lupus erythematosus
- Atherosclerosis/embolism from proximal source, e.g. subclavian artery aneurysm
- Drug-related, e.g. nicotine, beta-blockers, ergot
- Thoracic outlet syndrome
- Malignancy
- Hyperviscosity syndromes, e.g. Waldenström's macroglobulinaemia, polycythaemia
- Vibration-induced disorders (power tools)
- Cold agglutinin disorders



## 6.41 Clinical features of venous and arterial ulceration

Clinical feature	Venous ulceration	Arterial ulceration
Age	Develops at age 40–45 but may not present for years; multiple recurrences common	First presents in over-60s
Sex	More common in women	More common in men
Past medical history	Deep vein thrombosis (DVT) or suggestive of occult DVT, i.e. leg swelling after childbirth, hip/knee replacement or long bone fracture	Peripheral arterial disease, cardio- and cerebrovascular disease
Risk factors	Thrombophilia, family history, previous DVT	Smoking, diabetes, hypercholesterolaemia and hypertension
Pain	One-third have pain (not usually severe) that improves with elevating the leg	Severe pain, except in diabetics with neuropathy; improves on dependency
Site	Gaiter areas; usually medial to long saphenous vein; 20% are lateral to short saphenous vein	Pressure areas (malleoli, heel, fifth metatarsal base, metatarsal heads and toes)
Margin	Irregular, often with neopithelium (appears whiter than mature skin)	Regular, indolent, 'punched out'
Base	Often pink and granulating under green slough	Sloughy (green) or necrotic (black), with no granulation
Surrounding skin	Lipodermatosclerosis always present	No venous skin changes
Veins	Full and usually varicose	Empty with 'guttering' on elevation
Swelling (oedema)	Usually present	Absent
Temperature	Warm	Cold
Pulses	Present, but may be difficult to feel	Absent