Second Edition

# Clinical Examination

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# Cardiovascular system

chapter 3

Ischaemic heart disease and cerebrovascular disease account for almost 25% of all global deaths. In low- and middle-income countries, the rate of cardiovascular disease is increasing rapidly due to reductions in poverty and lifestyle changes. In contrast, the rates of cardiovascular disease are decreasing in developed countries due to more advanced medical care.

## 3.1 System overview

### Anatomy review

#### The heart

The heart consists of specialised cardiac skeletal muscle which contracts to deliver oxygen and nutrients to tissues and organs. Within the heart there are four main valves. The atrioventricular valves (tricuspid on the right and mitral on the left; **Figure 3.1**) separate the atria from the ventricles. These valves are open during ventricular relaxation (diastole), allowing blood to flow from the atria into the ventricles. These valves are closed during ventricular contraction (systole) to prevent backflow of blood from the ventricles into the atria. The closing of these valves at the start of systole can be heard as the first heart sound (S1) which coincides with the palpable pulse.

The pulmonary and aortic valves are semilunar (bicuspid) valves that open on ventricular contraction, allowing blood to flow into the pulmonary and systemic circulation respectively (**Figure 3.1**). The second heart sound (S2) is heard when these semilunar valves close at the end of systole. S2 is heard as a result of two valves closing, so any abnormality in the timing results in 'splitting' of the sound.

#### The peripheral vascular system

Blood is distributed to tissues by the systemic arterial circulation and then back to the heart by the venous circulation.

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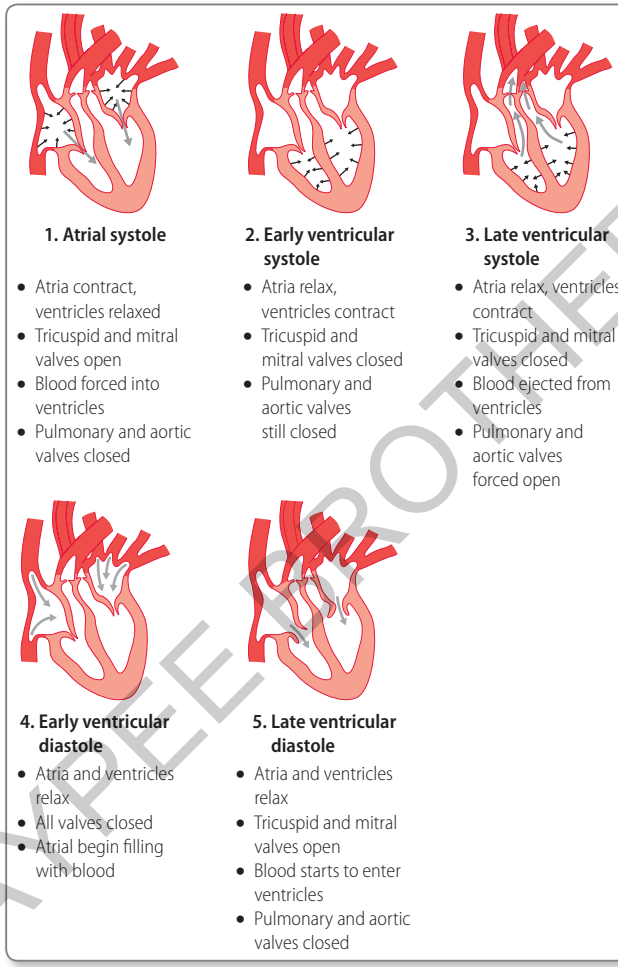


**Figure 3.1** The cardiac cycle.

The jugular veins feed into the superior vena cava and right atrium without any intervening valves, therefore any pressure change in the right atrium is seen in the jugular venous pressure.

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## Physiology review

### The cardiac cycle

There are two main phases of the cardiac cycle: systole and diastole (**Figure 3.1**). This cycle is controlled by a sophisticated system of electrical conduction, starting in the natural pacemaker, the sinoatrial node situated in the right atrium.

### Blood pressure

Systolic blood pressure is the peak blood pressure in arteries during ventricular contraction. Diastolic pressure is the minimum blood pressure during ventricular relaxation:

$$\text{Blood pressure} = \text{Cardiac output} \times \text{SVR}$$

Based on this equation, anything that affects cardiac output or Systemic vascular resistance (SVR) will affect blood pressure. Physiological mechanisms are in place to maintain normal blood pressure:

- Autonomic nervous system responses
- Capillary shift mechanisms
- Hormonal responses
- Kidney and fluid balance mechanisms

### Cardiac output

Cardiac output is the volume of blood (in millilitres) being pumped by either ventricle during 60 seconds:

$$\text{Cardiac output} = \text{Stroke volume} \times \text{Heart rate}$$

$$\text{Stroke volume} = \text{End-diastolic volume} - \text{End-systolic volume}$$

### Clinical insight

**Heart failure** is the inability of the heart to meet the body's demands for oxygen and nutrients. The primary disturbance is impairment in left ventricular function, leading to reduced stroke volume and therefore reduced cardiac output.

**Pulmonary oedema** is caused by a failure of the left ventricle to remove blood from the pulmonary circulation, resulting in 'back pressure' on the lungs. Fluid therefore overflows into the interstitial lung tissue and eventually into the air-filled spaces of the lungs.

**Peripheral oedema** is associated with right ventricular failure, with a similar back-pressure effect on the systemic venous return. Congestive heart failure is where both left- and right-sided heart failure are present in a patient; often the left-sided heart failure starts initially and causes the right heart failure.

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Starling's law dictates that the greater the volume of blood (preload) entering the ventricle during diastole, the greater the volume of blood ejected out of the heart during systole.

**Systemic vascular resistance** This is the resistance to blood flow offered by all of the systemic vasculature. The systemic vascular bed is a dynamic part of the circulatory system. Arterioles are dynamic vessels that are able to alter their diameter, thereby altering their resistance according to the body's needs. Vasoconstriction (narrowing) increases the SVR, which increases blood pressure, thus reducing cardiac output.

## 3.2 Symptoms and signs

### Symptoms

Take a full cardiac history (**Table 3.1**) using the mnemonic OPERATES+ (Table 1.2) for each presenting complaint or problem.

#### Chest pain

Chest pain is the most important symptom in cardiovascular disease, but can also be caused by non-cardiovascular disease (**Table 3.2**). Use the SOCRATES mnemonic (Table 1.3) to assess chest pain. The characteristics of cardiovascular chest pain varies according to its cause. In ischaemic heart disease it tends to be a heavy or crushing central chest pain, radiating to the left arm and/or jaw brought on by exertion and associated with nausea, sweating and breathlessness.

**Clinical insight**

**Risk factors for ischaemic heart disease:**

- age
- gender (men > women)
- family history
- smoking
- obesity (page 41)
- diabetes mellitus
- hypertension
- hypercholesterolaemia

#### Shortness of breath

Shortness of breath (SOB), also known as dyspnoea, is the subjective sensation of difficulty breathing. In cardiac pathology, breathlessness can be of three main types:

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| Component | Key points |
|---|---|
| Presenting complaint | Chest pain<br>Shortness of breath<br>Orthopnoea<br>Nocturnal dyspnoea<br>Oedema<br>Palpitations<br>Claudication<br>Collapse<br>Exercise intolerance |
| Past medical history | Cardiac disease (e.g. myocardial infarction)<br>Murmurs<br>Rheumatic fever |
| Past surgical history | Recent dental work (risk factor for infective endocarditis) |
| Drug history | Antihypertensives<br>Nitrates, e.g. glyceryl trinitrate or GTN<br>Other cardiac medications, e.g. antiarrhythmics, diuretics |
| Family history | Acquired cardiovascular disease<br>Congenital heart disease<br>Sudden death |
| Social history | Current or previous tobacco use<br>Alcohol use<br>Intravenous drug use (risk factor for infective endocarditis and deep vein thrombosis) |

**Table 3.1** The cardiac history

1. **Exertional dyspnoea:** dyspnoea during physical exertion
2. **Orthopnoea:** dyspnoea on lying flat. Orthopnoea is highly suggestive of severe heart failure. On lying flat, the venous return to the heart is increased. Pulmonary oedema occurs as a consequence of pulmonary venous congestion due to poor left ventricular function
3. **Nocturnal dyspnoea:** dyspnoea wakes the patient from sleep with a gasping sensation. Often there is pink frothy sputum after a bout of coughing

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| Origin | Examples |
|---|---|
| Cardiovascular | Coronary artery disease<br>Pericarditis or myocarditis<br>Aortic dissection<br>Valvular heart disease<br>Pulmonary embolus |
| Respiratory | Acute pleural inflammation (e.g. pneumonia)<br>Infiltration of pleura (e.g. mesothelioma)<br>Pleural malignancy (including metastases)<br>Spontaneous pneumothorax<br>Tracheal infection<br>Pleural effusion |
| Musculoskeletal | Costochondritis (Tietze's syndrome)<br>Muscle sprain<br>Rib fracture<br>Rheumatic disease |
| Gastrointestinal | Oesophagitis and/or gastro-oesophageal reflux<br>Oesophageal spasm or tear<br>Pancreatitis<br>Cholecystitis<br>Perforated gastric ulcer |
| Other | Thymoma<br>Lymphadenopathy<br>Mediastinitis<br>Herpes zoster (shingles)<br>Psychogenic |

**Table 3.2** Causes of chest pain

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### Palpitations

Palpitations are the abnormal sensation of the heart beating in the chest. Patients often describe them as irregular, missing beats, fast, fluttery, thumping or jumping. Palpitations can be found in healthy individuals or they can be a sign of a pathological arrhythmia. In healthy people, palpitations may be precipitated by anxiety, exercise, caffeine or alcohol, so ask about precipitating and relieving factors.

### Syncope

Syncope, otherwise known as fainting, is a sudden loss of consciousness immediately followed by a complete recovery.

It is a form of non-epileptic seizure, so a seizure history is useful (page 200). Syncope is caused by an interruption in the supply of oxygen-rich blood to the brain. Cardiac causes include vasovagal syncope, arrhythmia, sinus bradycardia, heart block, ventricular tachycardia, supraventricular tachycardia, aortic stenosis and hypertrophic cardiomyopathy.

### Claudication

Claudication is pain felt in the legs on walking and is relieved by rest. The pain is felt in the buttocks, thighs and/or calves. It suggests insufficient blood supply to the peripheries caused by peripheral vascular disease. Quantify the 'claudication distance', i.e. how far the patient is able to walk before the pain begins.

**Clinical insight**

**New York Heart Association (NYHA) functional classification of heart failure:**

**Class I:** no limitations; ordinary physical activity does not cause fatigue, breathlessness or palpitations

**Class II** (mild): slight limitation of physical activity; comfortable at rest; ordinary physical activity results in fatigue, palpitation, breathlessness or angina pectoris

**Class III** (moderate): marked limitation of physical activity; although comfortable at rest, less than ordinary physical activity leads to symptoms

**Class IV** (severe): inability to carry on any physical activity without discomfort; symptoms of cardiac failure are present even at rest

**Clinical insight**

All episodes of syncope should have lying and standing blood pressures measured along with an ECG to identify arrhythmias and/or prolonged QT interval.

## Signs

The key signs of cardiovascular disease are summarised in **Table 3.3**.

### Hypertension and hypotension

An abnormal blood pressure (Table 2.2) can be caused by disorders of almost any system and requires a full assessment of the patient for both cardiovascular and non-cardiovascular disease.

### Abnormal pulse rate

In adults, bradycardia is defined as <51 beats/min (bpm) and tachycardia as >90 bpm (Table 2.2). Exertion, pain, fever, anxiety,

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| | | |
|---|---|---|
| **General** | Nutritional state<br>Tendon xanthoma | Breathlessness<br>Sweating |
| **Vital signs** | High or low blood pressure<br>Fever<br>Low oxygen saturations | Tachypnoea<br>Tachycardia |
| **Pulses** | Radial rate<br>Abnormal rhythm | Abnormal character |
| **Hands** | Clubbing<br>Sweating<br>Splinter haemorrhages<br>Palmar erythema | Nicotine staining<br>Osler's nodes<br>Janeway's lesions |
| **Eyes** | Corneal arcus<br>Retinopathy | Pale conjunctiva |
| **Face and mouth** | Pallor<br>Malar flush | Central cyanosis<br>Poor dental hygiene |
| **Neck** | Raised jugular venous pressure<br>Carotid pulse character | Carotid bruits<br>Referred murmurs |
| **Precordium** | Inspect (scars, pulsations)<br>Palpation (displaced apex, heaves, thrills) | Auscultation (abnormal heart sounds, added sounds, murmurs) |
| **Chest** | Basal crackles | Dull percussion note |
| **Abdomen** | Hepatomegaly<br>Ascites | Aortic aneurysm |
| **Peripheries** | Radio-radio or radiofemoral delay<br>Sacral and/or peripheral oedema | Peripheral cyanosis<br>Absent peripheral pulses |
| **Genitalia** | Oedema | |

**Table 3.3** Key signs on cardiovascular examination

drugs, endocrine disease and arrhythmias cause tachycardia. Bradycardia is found during sleep and is also caused by drugs, endocrine disease and arrhythmias. Nonsymptomatic bradycardia may be a normal finding in individuals with a high level of fitness.

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### Crackles

Crackles, also known as crepitations, are non-musical sounds found during inspiration, caused by the reopening of collapsed or occluded alveoli. Crackles in association with other signs of cardiac disease indicate cardiogenic pulmonary oedema. Pulmonary oedema is a common and a potentially fatal cause of acute respiratory distress. Causes include left heart failure, fluid overload, severe hypertension, renal artery stenosis and renal disease.

### Oedema

Oedema is tissue swelling caused by fluid accumulation in the interstitial space. Due to gravity, the most common sites are the lower limbs and sacrum. Heart failure and vasodilator drugs are typical causes. A raised JVP and peripheral bilateral oedema suggest right ventricular failure.

## 3.3 Examination of the cardiovascular system

Position the patient at 45° to the horizontal and expose the chest (maintain dignity in women with a sheet). There are 'cardiac patterns' that can lead to a clinical diagnosis (**Table 3.4**).

### Vital signs

Assess the vital signs (Table 2.2). Pay particular attention to the pulse and the blood pressure.

### Inspection

#### General Inspection

Start with a general inspection (Chapter 2), in particular looking for breathlessness and colour. Is the patient on any oxygen? If so, how much and what effect is it having (see Chapter 4)?

Inspect the chest for scars, heaves and pulsations. Venous collaterals on the chest or shoulders are a sign of superior vena cava, axillary or subclavian vein obstruction.

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| | Aortic stenosis | Aortic regurgitation | Mitral stenosis | Mitral regurgitation |
|---|---|---|---|---|
| Pulse | Slow rising | Collapsing | Irregularly irregular (can be in AF) | |
| Peripheral findings | Low blood pressure<br>Narrow pulse pressure | Nail-bed pulsation (Quincke's sign)<br>Carotid pulsation (Corrigan's sign) | Malar flush<br>Signs of CCF | |
| JVP | | | Raised (late sign) | |
| Chest palpation | Sustained apex beat | Displaced and sustained apex beat | Tapping apex beat<br>Left parasternal heave (late on) | Displaced and sustained apex beat |
| Murmur | Ejection systolic | Early diastolic (S2 may be diminished) | Mid- to end-diastolic | Pansystolic (S2 may be diminished) |
| Primary site of murmur | Aortic area | Left sternal edge | Apex | Apex |
| Loudest heard | | Expiration at lower left sternal edge with patient sat up | Lying on left side | Lying on left side |
| Radiation | To the carotids | Along left sternal edge | None | To the axilla |
| Other findings | | Pistol shot femorals (Durosier's sign) | Opening mid-diastolic 'click',<br>Loud S1 | Bibasal crepitations secondary to LVF<br>Third heart sound |

AF, atrial fibrillation; CCF, congestive cardiac failure; JVP, jugular venous pressure; LVF, left ventricular failure; S2, second heart sound.

**Table 3.4** Cardiac patterns

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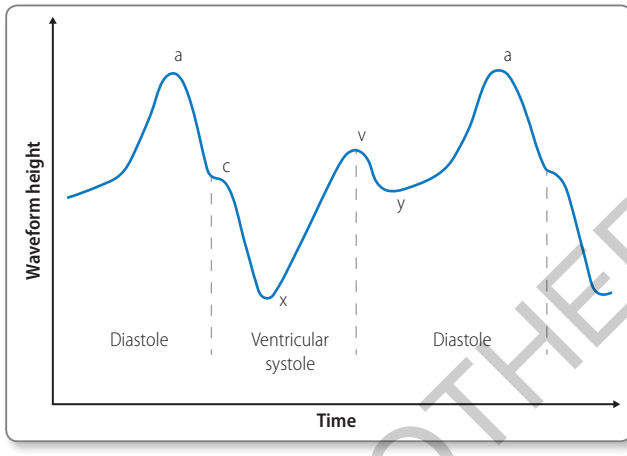


**Figure 3.2** Jugular venous pressure (JVP) waveform. a, atrium contracting, tricuspid value opens; c, right ventricular contraction; x, atrium relaxing and rapid filling; v, atrium full, tricuspid valve closed; y, atrium emptying with open tricuspid.

## Jugular venous pulse

The JVP is a visible waveform (**Figure 3.2**) in the internal jugular vein (**Figure 3.3**) rather than a palpable pulse. Due to its anatomy, the JVP reflects the pressure in the right atrium. Persistent elevation of the JVP is indicative of raised atrial pressure, so it is a reliable and early sign of right ventricular failure.

**Assessing the JVP**

- Position the patient with the trunk at 45° and rotate the head away from yourself
- Find the sternal angle (also known as the manubriosternal angle) and the internal jugular venous pulse (**Figure 3.4**)
- Confirm that it is the internal jugular vein (**Table 3.5**)
- Measure the peak vertical height of the waveform in centimetres (note: it is not the length of the pulsation along the vein but rather the vertical height that needs to be measured). A normal JVP is <3 cm

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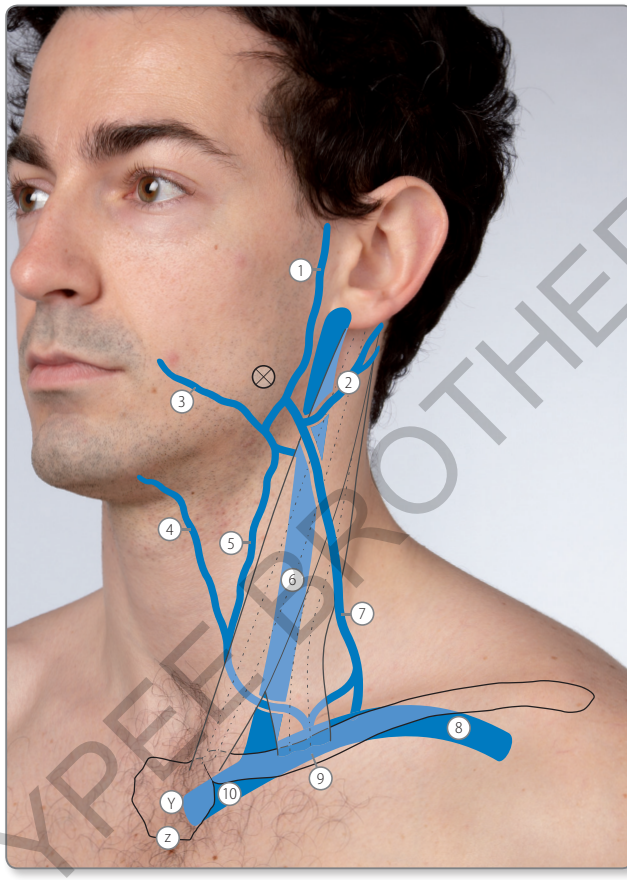


**Figure 3.3** Superficial and deep veins of the neck and face: (1), Retromandibular vein; (2), posterior auricular vein; (3), facial vein; (4), anterior jugular vein; (5), communicating vein; (6), internal jugular vein; (7) external jugular vein; (8), axillary vein; (9), subclavian vein; (10), left brachiocephalic vein; ⊗, position of the mandibular angle; (Y), manubrium of sternum; (Z), sternal angle.

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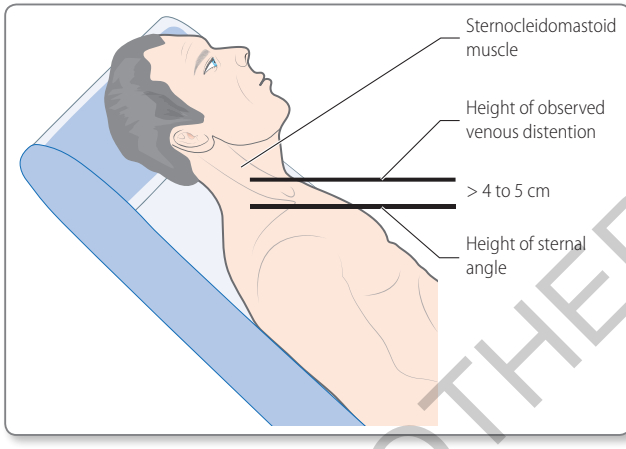


**Figure 3.4** Measuring jugular venous pressure.

| | JVP | Carotid pulse |
|---|---|---|
| Inspiration | Falls during inspiration | No effect |
| Waveforms | Double | Single |
| Pulse | Pulseless | Pulsatile |
| Effect of position | Varies | No effect |
| Abdominal pressure (hepatojugular reflex) | Rises | No effect |
| Compression distally | Obliterates waveform | No effect |

**Table 3.5** Differentiating the jugular venous pressure (JVP) from carotid pulse

**The hepatojugular reflex**

- Ensure that the patient has no abdominal pain
- Apply firm pressure to the right upper quadrant (RUQ)
- A transient elevation in JVP for a few seconds, of approximately 2 cm, is a normal finding

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# Clinical Examination

**pocket tutor Clinical Examination** is the ideal companion for medical students and junior clinicians seeking an accessible and practical guide to structuring a consultation, taking a history and examining a patient.

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