COMMON PRESENTING SYMPTOMS OF HEART DISEASE

The common symptoms of heart disease are chest pain, breathlessness, palpitations, syncope, fatigue and peripheral oedema. Careful history taking is important as there is overlap between symptoms arising from cardiovascular disease and those from other pathology. The severity of anginal pain, dyspnoea, palpitations or fatigue may be classified according to the New York Heart Association grading of ‘cardiac status’ (Table 9.1).

**Chest pain (K&C 6e p. 732)**

Acute central chest pain or discomfort is a common presenting symptom of cardiovascular disease and must be differentiated from non-cardiac causes. The site of pain, its character, radiation and associated symptoms will often point to the cause (Table 9.2).

**Dyspnoea (K&C 6e p. 733)**

Dyspnoea is an abnormal awareness of breathlessness. The causes are discussed on page 487. Left heart failure is the most common cardiac cause of exertional dyspnoea.

*Orthopnoea (K&C 6e p. 733)* refers to breathlessness on lying flat, as a result of gravitational redistribution of blood leading to increased pulmonary blood volume. *Paroxysmal nocturnal dyspnoea* occurs when there is an accumulation of

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<th>Grade</th>
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<tr>
<td>Grade 1</td>
<td>Uncompromised</td>
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<tr>
<td>Grade 2</td>
<td>Slightly compromised</td>
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<td>Grade 3</td>
<td>Moderately compromised</td>
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<td>Grade 4</td>
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fluid in the lungs at night causing the patient to awake suddenly from sleep.

**Palpitations** *(K&C 6e p. 733)*

A palpitation is an awareness of the heartbeat. The normal heartbeat is sensed when the patient is anxious, excited, exercising or lying on the left side. In other circumstances it usually indicates a cardiac arrhythmia, commonly ectopic beats or a paroxysmal tachycardia (p. 412).

**Syncope and dizziness** *(K&C 6e p. 734)*

Syncope means a temporary impairment of consciousness due to inadequate cerebral blood flow. There are many causes, but the most common is a simple faint or vasovagal attack (p. 697). The cardiac causes of syncope are the result of either very fast (e.g. ventricular tachycardia) or very slow heart rates (e.g. complete heart block) which are unable to maintain an adequate cardiac output. Attacks occur suddenly and without warning. They last only 1 or 2 minutes,
with complete recovery in seconds (compare with epilepsy, where complete recovery may be delayed for some hours). Obstruction to ventricular outflow also causes syncope (e.g. aortic stenosis, hypertrophic cardiomyopathy), which typically occurs on exercise when the requirements for increased cardiac output cannot be met.

**Other symptoms**

Tiredness, lethargy and exertional fatigue occur with heart failure and result from poor perfusion of brain and skeletal muscle. Heart failure also causes salt and water retention, leading to oedema, which in ambulant patients is most prominent over the ankles. In severe cases it may involve the genitalia and thighs.

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### INVESTIGATIONS IN CARDIAC DISEASE

**The chest X-ray** *(K&C 6e p. 741)*

This is usually taken in the postero-anterior (PA) direction at maximum inspiration. A PA chest film can aid the identification of cardiomegaly, pericardial effusions, dissection or dilatation of the aorta, and calcification of the pericardium or heart valves. Cardiomegaly is indicated by a cardiothoracic width ratio $>50\%$ (maximum transverse diameter of the heart compared to maximum transverse diameter of the thorax measured from the inside of the ribs). Examination of the lung fields may show signs of left ventricular failure (Fig. 9.1), valvular heart disease (Fig. 9.2) or pulmonary oligaemia (reduction of vascular markings) associated with pulmonary embolic disease.

**The electrocardiogram** *(K&C 6e p. 744)*

The electrocardiogram (ECG) is a recording from the body surface of the electrical activity of the heart. Each cardiac cell generates an action potential as it becomes depolarized and then repolarized during a normal cycle. Depolarization of cardiac cells proceeds in an orderly fashion in the normal situation, beginning in the sinus node (lying in the junction between superior vena cava and right atrium) and then spreading sequentially through the atria, AV node (lying beneath the right atrial endocardium within the lower interatrial septum), and the His bundle in the interventricular
septum, which divides into right and left bundle branches (Fig. 9.3). The bundle branches spread throughout the subendocardial surface of the right ventricle and left ventricle respectively. The main left bundle divides into an anterior superior division (the anterior hemi-bundle) and a posterior inferior division (the posterior hemi-bundle).

The standard ECG has 12 leads:

- Chest leads, $V_1$–$V_6$, look at the heart in a horizontal plane (Fig. 9.4).
- Limb leads look at the heart in a vertical plane (Fig. 9.5). Limb leads are unipolar (AVR, AVL and AVF) or bipolar (I, II, III).

**Fig. 9.1** The chest X-ray in left ventricular failure. A, Kerley B lines; B, hilar haziness; C, fluid in the right horizontal interlobar fissure; D, upper lobe venous engorgement; E, cardiomegaly.
Investigations in cardiac disease

The electrocardiogram

LA = left atrium
RA = right atrium

Pulmonary venous hypertension
Large LA appendage
Large LA
Calcified mitral valve
Pleural effusions

Fig. 9.2  Schematic representation of the chest X-ray in mitral stenosis. LA, left atrium; RA, right atrium; TR, tricuspid regurgitation.

Widened carina
Large RA in 2nd TR
Kerley B lines (interstitial oedema)

LA = left atrium
RA = right atrium

Fig. 9.3  The normal cardiac conduction system. In normal circumstances only the specialized conducting tissues of the heart undergo spontaneous depolarization (automaticity) which initiates an action potential. The sinus (SA) node discharges more rapidly than the other cells and is the normal pacemaker of the heart. The impulse generated by the sinus node spreads first through the atria, producing atrial systole, and then through the atrioventricular (AV) node to the His–Purkinje system, producing ventricular systole.

Right atrium
SA node
AV node
Right ventricle
Right bundle branch
Left atrium
Left atrium
Left ventricle
Left ventricle
Bundle of His
Left bundle branch
Left bundle branch
Fig. 9.4  ECG chest leads. (a) The V leads are attached to the chest wall overlying the intercostal spaces as shown: V₄ in the mid-clavicular line, V₅ in the anterior axillary line, V₆ in the mid-axillary line. (b) Leads V₁ and V₂ look at the right ventricle, V₃ and V₄ at the interventricular septum, and V₅ and V₆ at the left ventricle. The normal QRS complex in each lead is shown. The R wave in the chest (precordial) leads steadily increases in amplitude from lead V₁ to V₆ with a corresponding decrease in S wave depth, culminating in a predominantly positive complex in V₆.
The ECG machine is arranged so that when a depolarization wave spreads towards a lead the needle moves upwards on the trace (i.e. a positive deflection), and when it spreads away from the lead the needle moves downwards.

**ECG waveform and definitions** (Fig. 9.6)

The *heart rate*. At normal paper speed (usually 25 mm/s) each ‘big square’ measures 5 mm wide and is equivalent to 0.2 s. The heart rate (if the rhythm is regular) is calculated by counting the number of big squares between two consecutive R waves and dividing into 300.

The *P* wave is the first deflection and is caused by atrial depolarization. When abnormal it may be:
■ Broad and notched (> 0.12 s, i.e. 3 small squares) in left atrial enlargement (‘P mitrale’, e.g. mitral stenosis)
■ Tall and peaked (> 2.5 mm) in right atrial enlargement (‘P pulmonale’, e.g. pulmonary hypertension)
■ Replaced by flutter or fibrillation waves (p. 414)
■ Absent in sinoatrial block (p. 409).

The QRS complex represents ventricular depolarization:
■ A negative (downward) deflection preceding an R wave is called a Q wave. Normal Q waves are small and narrow; deep (> 2 mm), wide (> 1 mm) Q waves (except in AVR and V₁) indicate myocardial infarction (p. 443).
■ A deflection upwards is called an R wave whether or not it is preceded by a Q wave.
■ A negative deflection following an R wave is termed an S wave.

Ventricular depolarization starts in the septum and spreads from left to right (Fig. 9.3). Subsequently the main free walls of the ventricles are depolarized. Thus, in the right ventricular leads (V₁ and V₂) the first deflection is upwards (R wave) as the septal depolarization wave spreads towards...
those leads. The second deflection is downwards (S wave) as the bigger left ventricle (in which depolarization is spreading away) outweighs the effect of the right ventricle (see Fig. 9.4). The opposite pattern is seen in the left ventricular leads (V_s and V_6), with an initial downwards deflection (small Q wave reflecting septal depolarization) followed by a large R wave caused by left ventricular depolarization.

**Left ventricular hypertrophy.** The increased bulk of the left ventricular myocardium in left ventricular hypertrophy (e.g. with systemic hypertension) increases the voltage-induced depolarization of the free wall of the left ventricle. This gives rise to tall R waves (> 25 mm) in the left ventricular leads (V_5, V_6) and/or deep S waves (> 30 mm) in the right ventricular leads (V_1, V_2). The sum of the R wave in the left ventricular leads and the S wave in the right ventricular leads exceeds 40 mm. In addition to these changes there may also be ST-segment depression and T wave flattening or inversion in the left ventricular leads.

**Right ventricular hypertrophy** (e.g. in pulmonary hypertension) causes tall R waves in the right ventricular leads.

The **QRS duration** reflects the time that excitation takes to spread through the ventricle. A wide QRS complex (> 0.10 s, 2.5 small squares) occurs if conduction is delayed, e.g. with right or left bundle branch block, or if conduction is through a pathway other than the right and left bundle branches, e.g. an impulse generated by an abnormal focus of activity in the ventricle (ventricular ectopic).

**T waves** result from ventricular repolarization. In general the direction of the T wave is the same as that of the QRS complex. Inverted T waves occur in many conditions and, although usually abnormal, they are a non-specific finding.

The **PR interval** is measured from the start of the P wave to the start of the QRS complex whether this is a Q wave or an R wave. It is the time taken for excitation to pass from the sinus node, through the atrium, atrioventricular node and His–Purkinje system to the ventricle. A prolonged PR interval (> 0.22 s) indicates heart block (p. 410).

The **ST segment** is the period between the end of the QRS complex and the start of the T wave. ST elevation (> 1 mm above the isoelectric line) occurs in the early stages of myocardial infarction (p. 443) and with acute pericarditis. ST segment depression (> 0.5 mm below the isoelectric line) indicates myocardial ischaemia.
The QT interval extends from the start of the QRS complex to the end of the T wave. It is primarily a measure of the time taken for repolarization of the ventricular myocardium, which is dependent on heart rate (shorter at faster heart rates). The QT interval is therefore corrected for heart rate (QTc) and normally is $\leq 0.44$ s. The long QT syndrome may be congenital or acquired (hypokalaemia or hypomagnesaemia, drugs e.g. quinidine, sotalol, chlorpromazine) and is associated with an increased risk of torsades de pointes ventricular tachycardia (p. 420) and sudden death.

$$QT_c = \frac{QT \text{ interval}}{\sqrt{\text{RR interval}}} \text{ (in seconds)}$$ (QC 9.1)

The cardiac axis refers to the overall direction of the wave of ventricular depolarization in the vertical plane measured from a zero reference point (Fig. 9.7). The normal range for the cardiac axis is between $-30^\circ$ and $+90^\circ$. An axis more negative than $-30^\circ$ is termed left axis deviation whereas an axis more positive than $+90^\circ$ is termed right axis deviation. A simple method to calculate the axis is by inspection of the QRS complex in leads I, II and III. The axis is normal if leads I and II are positive; there is right axis deviation if lead I is negative and lead III positive, and left axis deviation if lead I is positive and leads II and III negative. Left axis deviation occurs due to a block of the anterior bundle of the main left bundle conducting system (p. 399), inferior myocardial infarction and the Wolff–Parkinson–White syndrome. Right axis deviation may be normal and occurs in conditions in which there is right ventricular overload, dextrocardia, Wolff–Parkinson–White syndrome and left posterior hemiblock.

**Exercise electrocardiography** (K&C 6e p. 746)

This is a technique used to assess the cardiac response to exercise. The 12-lead ECG is recorded whilst the patient walks or runs on a motorized treadmill, and the test should be performed according to a standardized method (e.g. the Bruce protocol). Myocardial ischaemia provoked by exertion results in ST segment depression ($> 1$ mm) in leads facing the affected area of ischaemic cardiac muscle. During an exercise test the blood pressure and rhythm responses to exercise are also assessed. Exercise normally causes an increase in heart rate and blood pressure. A sustained fall in
Fig. 9.7 Cardiac vectors. (a) The hexaxial reference system, illustrating the six leads in the frontal plane, e.g. lead I is $0^\circ$, lead II is $+60^\circ$, lead III is $120^\circ$. (b) ECG leads showing the predominant positive and negative deflection with axis deviation.