Chapter 3

The electrocardiogram

Electrocardiogram (ECG)

This is a graphical representation of the electrical activity of the heart and is recorded using 10 electrodes placed in specific locations on the body (Fig. 3.1). This electrical activity is displayed as 12 traces (12-lead surface ECG; see Fig. 3.2). Each deflection of the electrogram represents a particular activity in the cardiac cycle. Very minor changes to the structure or function of the heart can produce quite marked changes of the ECG, which can therefore be a powerful diagnostic tool. It is important to understand the principles behind the ECG so as to understand normality and thereafter recognize abnormality.

Electrical activity of the normal heart

The start of electrical activity in the heart occurs in an area of the right atrium where specialized pacemaker cells form the sinoatrial node (SA node). Electrical discharges from these cells produce depolarization that spreads through the atrial muscle from right to left. The only route by which electrical activity can then reach the ventricles is via further specialized pacemaker cells; the atrioventricular node (AVN). From the AVN, electrical activity then spreads rapidly down the His bundle, which subsequently divides into right and left branches; the left bundle further divides into anterior and posterior fascicles (Fig. 3.3).

Recording the ECG

The ECG is recorded with the patient lying down and relaxed. Cardiac electrical activity that is directed towards an electrode produces a positive deflection, and when directed away produces a negative deflection, on the ECG.

- Standard leads I, II and III are bipolar and oriented in the coronal plane.
- Leads aVR, aVL and aVF ('a' for augmented as instrumental augmentation is necessary because of the low potential at the extremities) are unipolar leads oriented in the coronal plane.
- Leads V_1–V_6 are unipolar leads oriented in the horizontal plane.

    The vectorial orientation of these leads is illustrated in Fig. 3.1.

Components of the normal ECG (Fig. 3.4)

- **P wave.** This represents atrial depolarization. The deflection arising as a result of repolarization is so small that it is not seen in the P wave.
- **PR interval.** This represents the time taken to conduct from the atria to the ventricles, and is taken from the onset of the P wave to the first deflection of the QRS.
- **QRS complex.** This represents ventricular depolarization. Q is the first negative deflection, R the first positive deflection and S is the first negative deflection following a positive deflection. As the left ventricle contributes most of the mass of
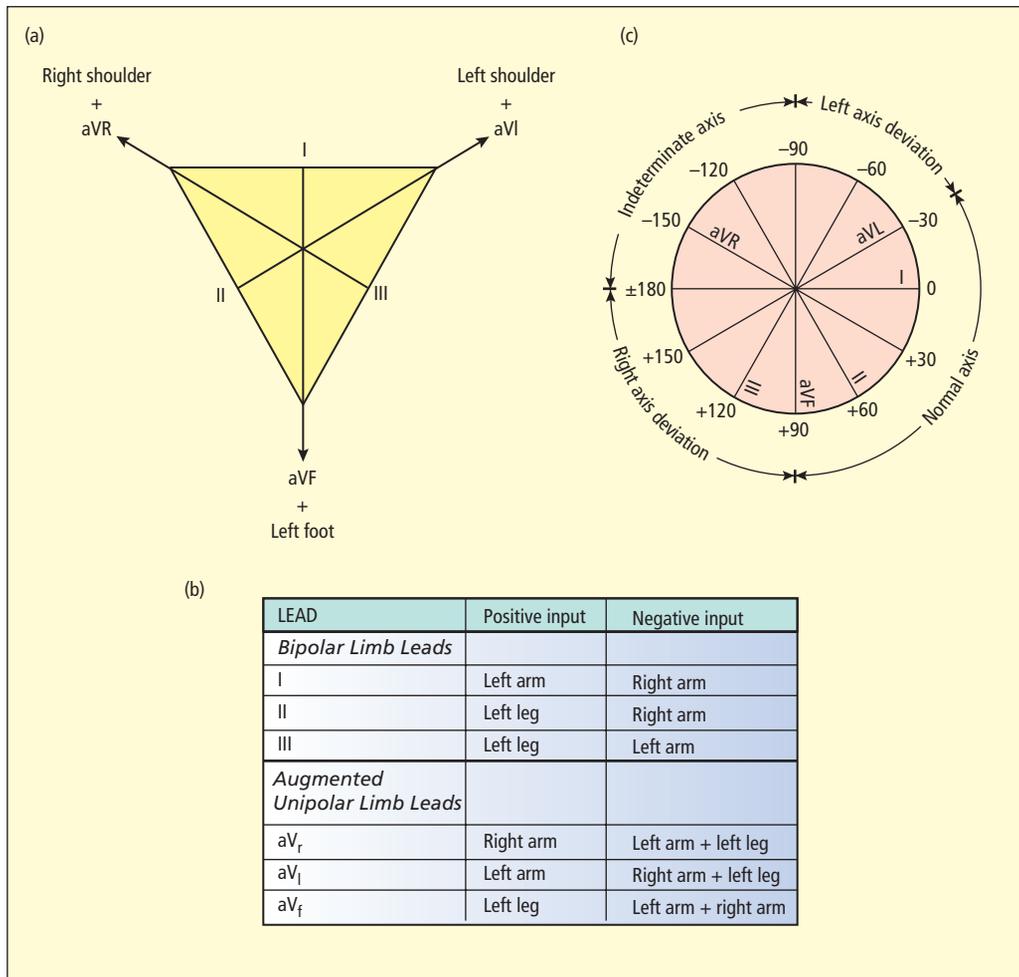
Fig. 3.1 Eindhoven's triangle: there are three 'standard' bipolar limb leads (I–III) and three augmented limb leads (aVR, aVF and aVL).

myocardium, it also contributes most of the electrical activity of the QRS component. Its duration is measured from the first deflection to the end of the QRS.

● **ST segment.** This is defined as the component of the ECG that occurs from the end of the QRS complex to the start of the T wave. The QT interval is measured from the onset of the QRS to the end of the T wave. The QT interval is affected by heart rate and so the 'corrected' QT interval (QTc) is more clinically useful.

$$QTc = QT/\sqrt{R\text{–}R'} \text{ interval}$$

Where the QT is the measured QT in seconds, and R–R' is the interval between the R waves of consecutive QRS complexes, measured in seconds.

● **T wave.** This represents ventricular repolarization.

● **U wave.** This is usually a small deflection or 'hump' seen in the end portion or after the T wave. It is not always present and the exact cause of it is unknown.
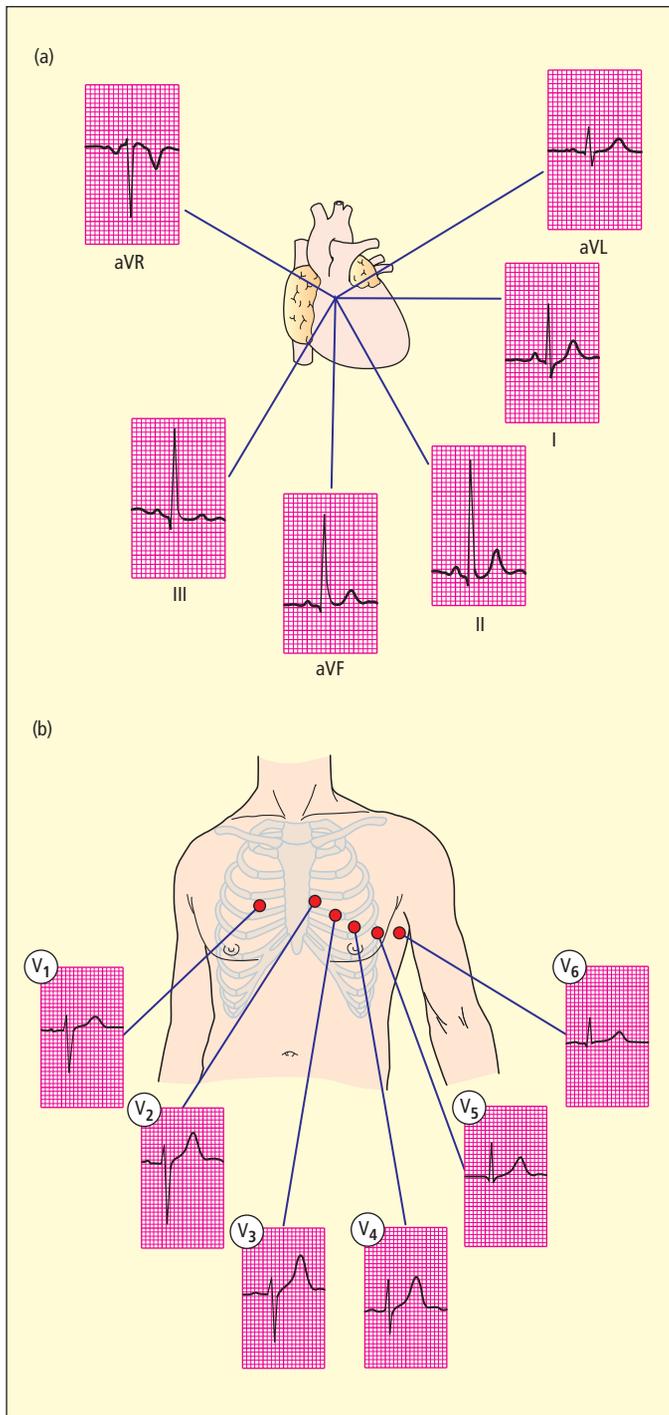
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(a)

aVR

aVL

I

III

aVF

II

(b)

V₁

V₆

V₂

V₅

V₃

V₄

Fig. 3.2 Normal 12-lead ECG. (a) Limb lead electrode positions and ECG complexes. (b) Chest lead electrode positions and ECG complexes.
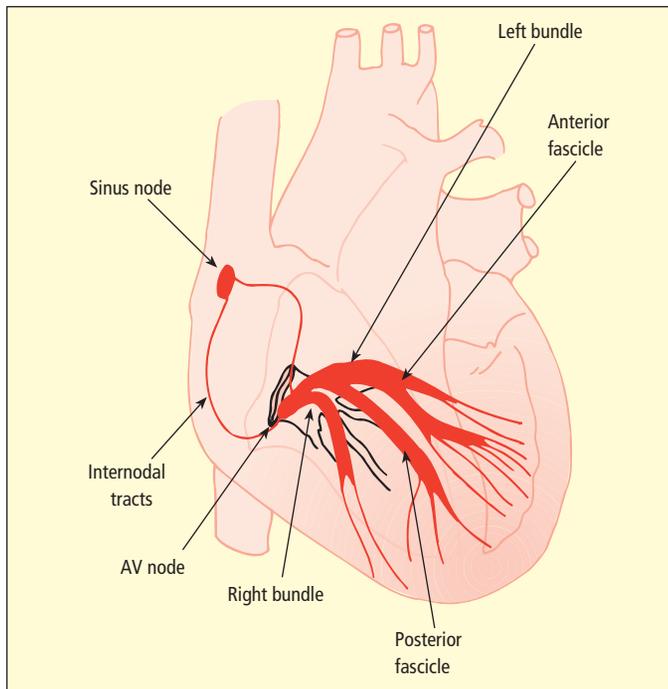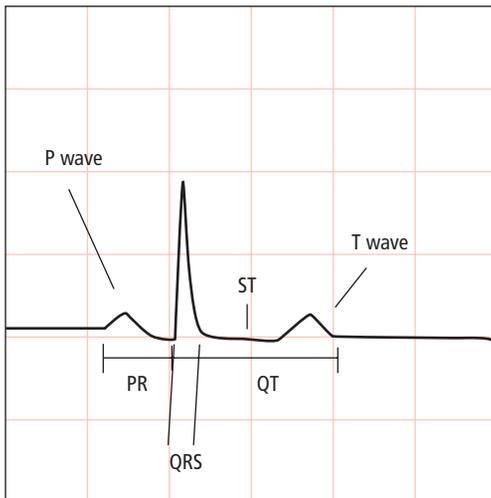
Fig. 3.3 The cardiac conduction system.

Fig. 3.4 ECG of a single cardiac cycle illustrating the different components of the ECG and relevant intervals.

Normal intervals

Each small square on the ECG paper represents 0.04 s (40 ms) and each large square 0.20 s (200 ms).

The following are normal ranges (Fig. 3.1):
- PR interval = 0.12–0.20 s
- QRS duration = <0.12 s
- QT interval = 0.35–0.45 s
- QTc interval = 0.38–0.42 s

(the QTc normal value for females is slightly longer than that for males)

Analysing the ECG

The key to accurate ECG diagnosis is to have a methodical approach to the ECG that you can apply to every ECG (Fig. 3.5). The following section identifies aspects of the ECG that require particular attention.

Cardiac rate and rhythm

In addition to the 12 leads of the ECG, most machines will display a rhythm strip. Use this to determine the rate and rhythm. The **heart rate** can quickly be calculated by counting the number

of large squares between each R–R′ interval and dividing into 300:

- 1 square = 300 beats/min (bpm)
- 2 squares = 150 bpm
- 3 squares = 100 bpm
- 4 squares = 75 bpm

Fig. 3.5 Algorithm to illustrate a logical approach to ECG analysis.

- 5 squares = 60 bpm
- 6 squares = 50 bpm

The heart rate can then be roughly defined as:

- Bradycardic <60 bpm
- Normal 60–100 bpm
- Tachycardic >100 bpm

The **cardiac rhythm** is determined by looking at the regularity or irregularity of QRS complexes and their association with each P wave. A regular rhythm where each QRS complex is preceded by a P wave (with a normal PR interval) is defined as normal sinus rhythm. If the rhythm is irregular, then this should be further qualified by identifying whether there is any regularity to the irregularity or whether it is irregularly irregular (usually atrial fibrillation (AF) or frequent ectopic beats). If the electrical baseline does not appear flat and has a 'chaotic' appearance to it, then it is most probably AF (Fig. 3.6).
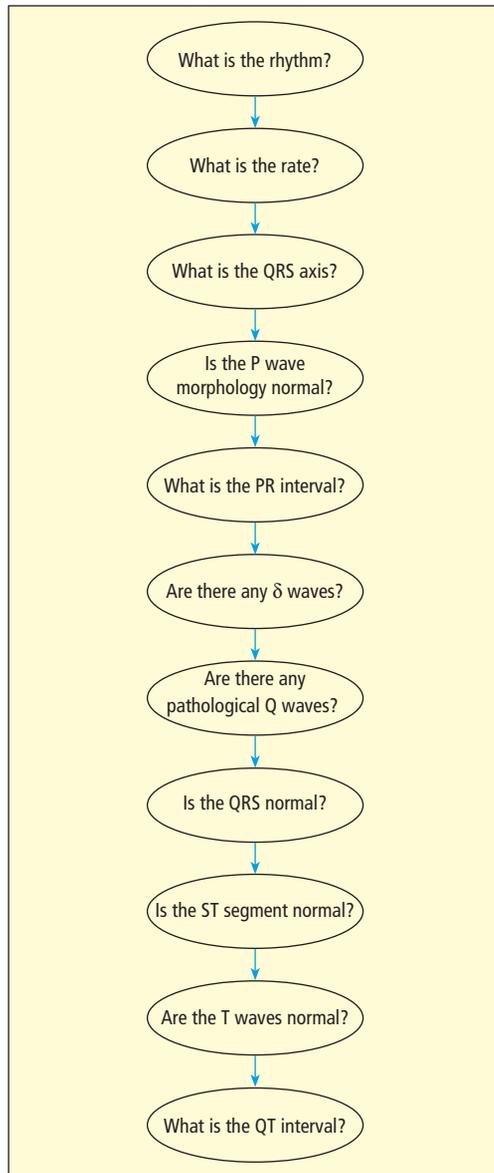
Extrasystoles

A premature depolarization of the atrium or ventricles will produce extrasystoles (ectopic beats). If they originate from the atrium, they will appear as a premature P wave which may have a different morphology and may be lost in the preceding T wave of the normal beat (Fig. 3.7a). If they originate from the ventricle, they will appear as a premature abnormal looking QRS complex (Fig. 3.7b). When normal sinus beats alternate with extrasystoles, the rhythm is called bigeminy (Fig. 3.7c).

Bradycardias/conduction abnormalities

Abnormalities of the conduction system at any level may cause bradycardias. Careful analysis of the rhythm strip should allow you to determine the level at which the abnormality exists.

Sinus bradycardia

A P wave precedes each QRS complex with a normal PR interval but the rate is slow (Fig. 3.8). This suggests abnormal sinus node function.

Junctional bradycardia

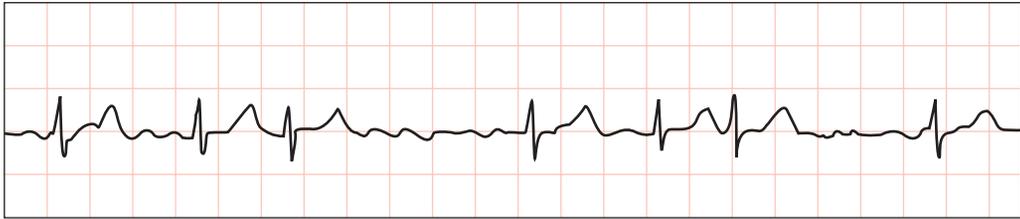No P wave is seen before a normal QRS. The level of abnormality may be just above the level of the

Fig. 3.6 Atrial fibrillation (note the irregular rhythm and chaotic baseline with no evidence of P waves).

(a)

(b)

(c)

Fig. 3.7 Rhythm strips demonstrating: (a) three sinus beats followed by three atrial extrasystoles; (b) two sinus beats followed by a ventricular extrasystole; and (c) ventricular bigeminy with alternating sinus and ventricular extrasystoles.
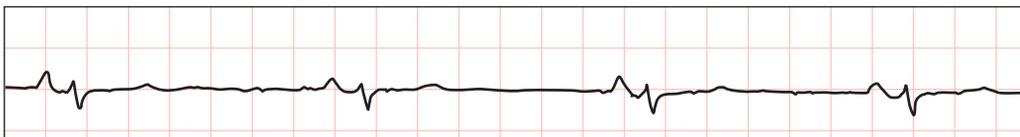

Fig. 3.8 Sinus bradycardia.

AVN and thus the P wave may be 'buried' within the QRS complex (Fig. 3.9).

Atrioventricular block

Disease of the AVN will produce differing relationships of the P waves and QRS complexes as the degree of disease and speed with which the AVN conducts (Fig. 3.10a–e). First degree block results in prolongation of the PR interval (Fig. 3.10b). Second degree block is divided into Mobitz Type I (Wenckebach) where there is progressive PR prolongation followed by a non-conducted P wave (Fig. 3.10c), and Mobitz Type II where the PR interval is more constant but P waves intermittently fail to be conducted (Fig. 3.10d). Third degree, or
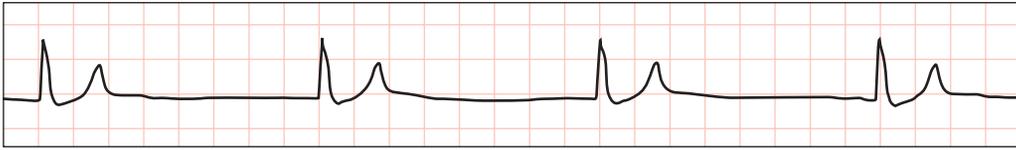
Fig. 3.9 Junctional bradycardia with no evidence of P waves.

(a)

(b)

(c)

(d)

(e)

Fig. 3.10 Varying degrees of AV block (P waves are marked with a 'p'). (a) Normal sinus rhythm. (b) First degree heart block. (c) Mobitz I (Wenckebach). (d) Mobitz II. (e) Complete heart block with broad QRS complexes and no association with the P waves.

complete, heart block results in failure of any P waves to be conducted, resulting in independent and dissociated P and QRS rhythms (Fig 3.10e).

Bundle branch block

If the abnormality exists in the His bundles, then there will be classical abnormal appearances of the QRS complexes depending on whether the right (RBBB; see Fig. 3.11) or left (LBBB; see Fig. 3.12) bundle is affected. Delay in one bundle causes the QRS to be prolonged (>0.12s) and to appear fractionated into an imperfect 'M' or 'W' configuration. Identifying the 'M' or 'W' in leads V_1 and V_6 will provide the diagnosis (see Box 3.1).

Tachycardias

If the QRS complexes are wide (>0.12s), then the tachycardia is defined as a broad complex tachycardia; if it is narrow, then it is a supraventricular tachycardia.
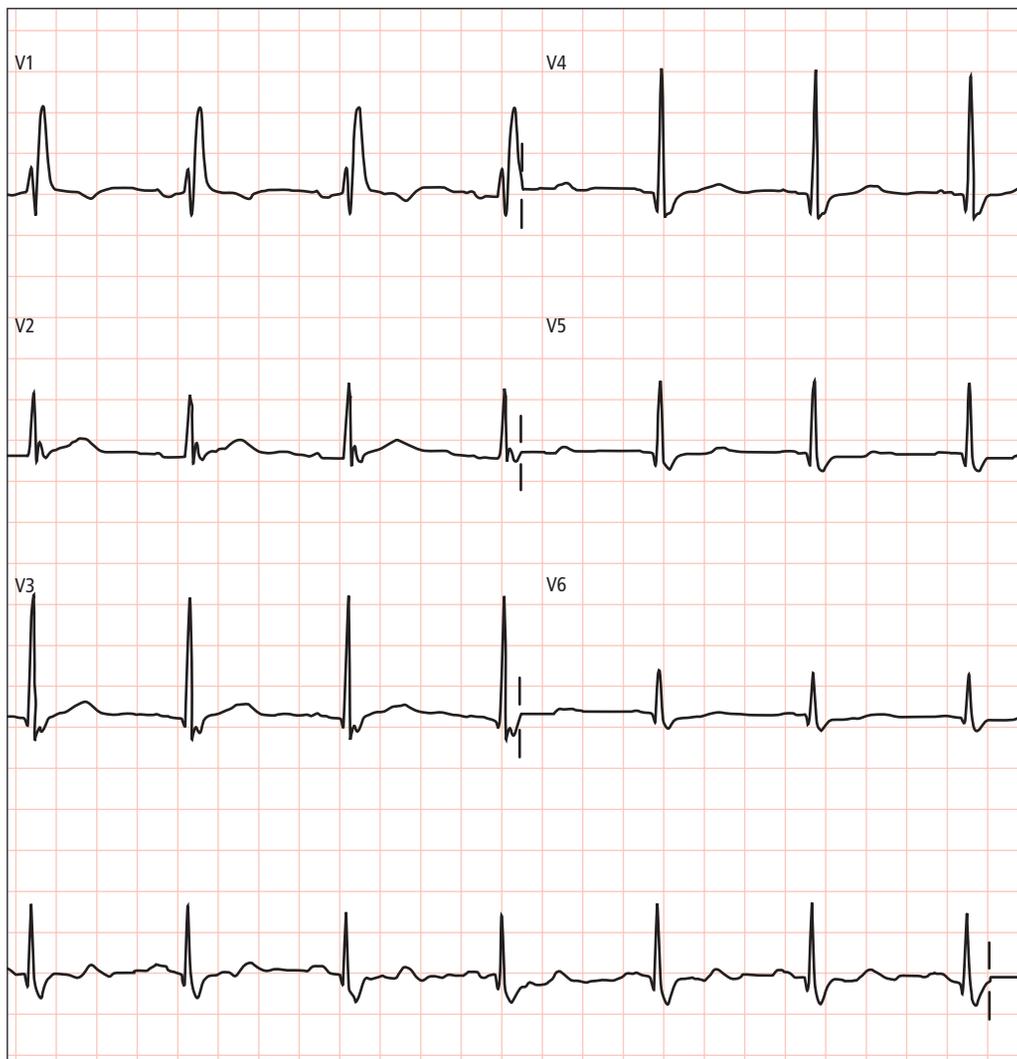

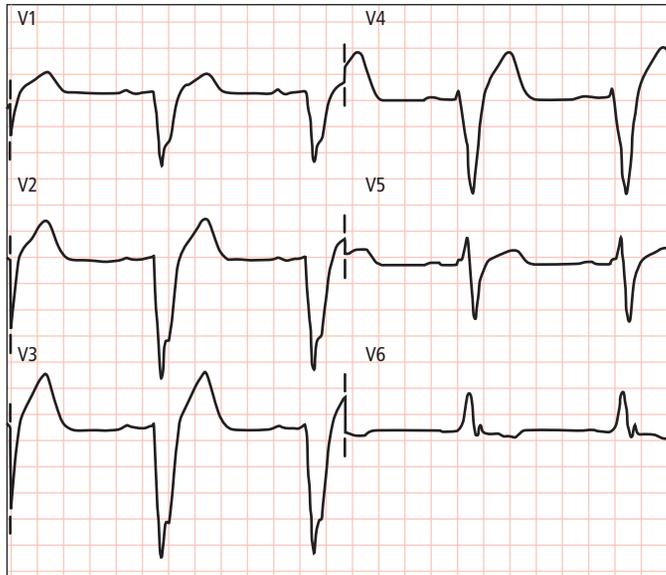**Fig. 3.11** Right bundle branch block.
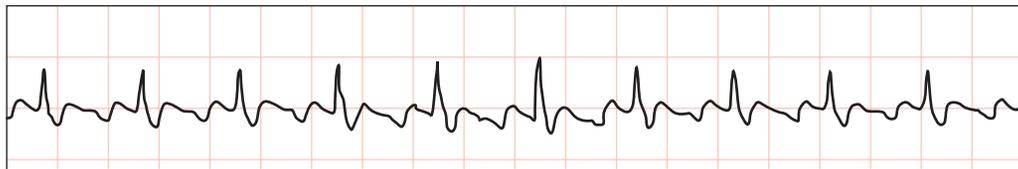
Fig. 3.12 Left bundle branch block.

Fig. 3.13 Atrial flutter (note the saw-tooth appearance of the baseline).

Box 3.1

Mnemonic to remember whether conduction abnormality is left bundle branch block (LBBB) or right bundle branch block (RBBB).

V_1 V_6

*W*i**LL**ia*M* = LBBB

*M*o**RR**o*W* = RBBB

Supraventricular tachycardias (SVTs)

These may be further defined:

• Atrial fibrillation—irregularly irregular with chaotic baseline (Fig. 3.6).

• Atrial flutter—tends to be regular with classic flutter waves that appear as a saw-tooth baseline (Fig. 3.13). The rate of the flutter waves is usually around 300 bpm giving a QRS rate of 150/min (if 2:1 ventricular conduction) or 100/min (3:1 conduction), etc.

• Atrial tachycardia, AV nodal re-entry tachycardia and AV re-entry tachycardia—all appear as a regular narrow complex tachycardia (Fig. 3.14).

Ventricular tachycardia (VT)

This is usually a regular broad complex tachycardia (Fig. 3.15). Occasionally an SVT may appear broad complex if there is associated bundle branch block. However, it is always safer in this situation to assume that a broad complex tachycardia is VT. Certain features may help distinguish one from the other (see Box 3.2).

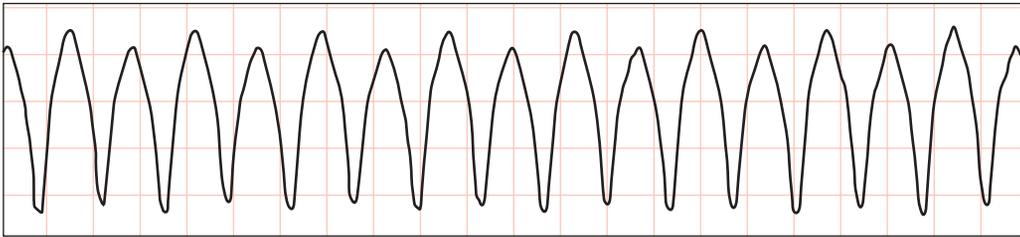**Fig. 3.14** Supraventricular tachycardia producing a narrow complex tachycardia.

Fig. 3.15 Broad complexes of ventricular tachycardia.

Box 3.2

Features that suggest a broad complex tachycardia is VT, rather than SVT with bundle branch block:

● AV dissociation (P waves seen in the tachycardia unrelated to the QRS complexes)

● Significant axis deviation compared with a pre-arrhythmia ECG

● Concordance of the chest leads (all chest lead QRS complexes are either negatively or positively directed)

● Very broad QRS complexes (>0.14 s)

● Fusion beats (occur when a P wave and QRS occur together producing a different shaped QRS)

● Capture beats (occur when a P wave transmits in the normal way to the ventricle producing a normal QRS complex)

QRS axis

The direction of spread of depolarization through the ventricles can be determined by the QRS complexes in leads I, II and III (Fig. 3.16a–c). The normal axis is defined as 0 to +90°, a leftward axis as 0 to –30° (may be normal), left axis deviation as less than –30°, and right axis deviation as more than +90°.

A change in the axis from normal to right usually occurs when strain is placed on the right side of the heart. A change from normal to left may occur as a result of left ventricular strain, but more commonly occurs with conduction abnormalities, particularly LBBB or block of the left anterior fascicle.

To calculate the direction of the QRS axis, use a 12-lead ECG as an example and refer to the vectorial diagram in Fig. 3.1. By using only the three standard limb leads (I, II, III) and the augmented unipolar limb leads (aVR, aVL, aVF), identify the lead in which the QRS is closest to having the same amount of positive and negative deflection compared with the isoelectric baseline. Having done so, the axis is 90° to this lead, being in either a rightward or leftward direction. For instance, if aVL is the 'isoelectric' lead, the direction of the QRS axis is either –120° or +60° (i.e. 90° to the left or right of aVL, which itself is positioned at –30°). If, in this example, the QRS were positive in aVR then the axis would be –120° (unusual), whereas if lead II has a positive QRS then the axis will be +60° (normal).

Abnormalities of particular components of the ECG

P wave

If P waves are absent, then consider heart block, atrial fibrillation or other abnormal rhythm as
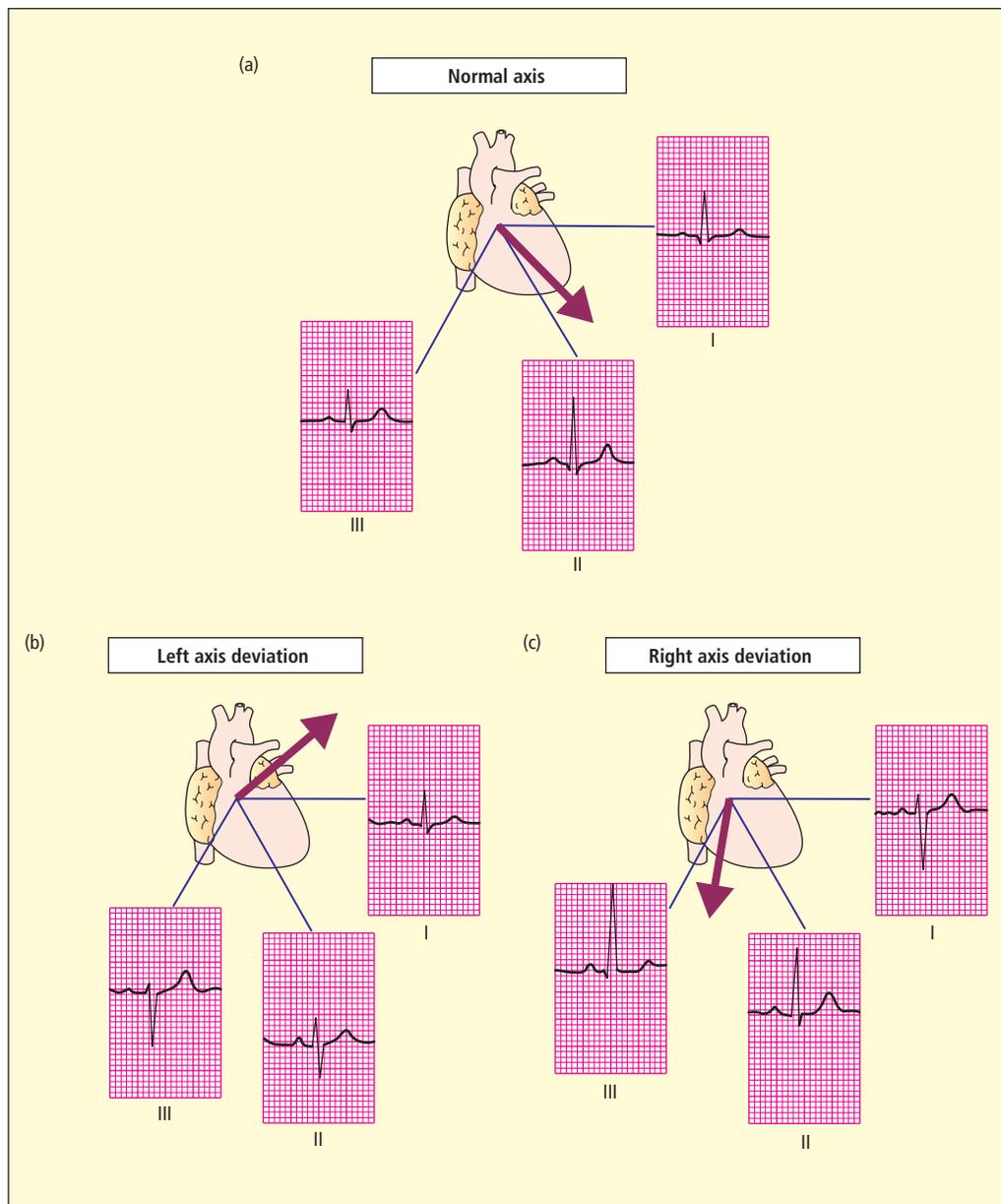
Fig. 3.16 (a) Normal axis with dominant component of QRS in I and II. (b) Left axis deviation with dominant component in lead I. (c) Right axis deviation with dominant component in II and III.

above. Tall P waves (>2.5 mm) that are peaked indicate right atrial enlargement (Fig. 3.17), whereas broad P waves (>0.08 s) that are bifid suggest left atrial enlargement (Fig. 3.18).

QRS complexes

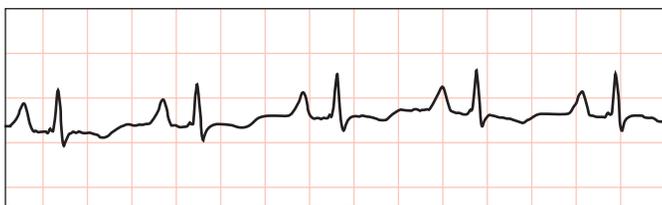A Q wave is the first downward deflection after a P wave. Some leads may have small Q waves that are

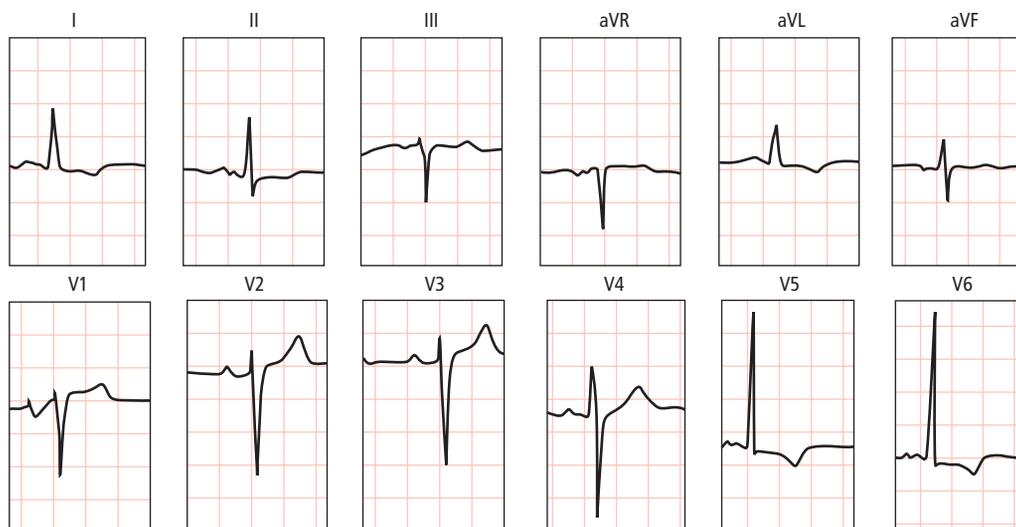**Fig. 3.17** Tall P wave of right atrial enlargement.

Fig. 3.18 Bifid P wave of left atrial enlargement.

normal, such as occurs in lead V_6 due to ventricular septal depolarization (Fig. 3.19). 'Pathological' or abnormal Q waves are defined as >2 small squares deep, or >25% of the height of the following R wave, or >1 small square wide (Fig. 3.20). Pathological Q waves are seen particularly after myocardial infarction, but also with left ventricular hypertrophy, and in bundle branch block.

Large amplitude complexes in the chest leads may indicate left ventricular hypertrophy. This is defined as an R wave in V_5 or V_6 plus the S wave in V_1 or V_2 which exceeds 35 mm (Fig. 3.21). However, in some young, thin individuals these criteria may be met but without left ventricular hypertrophy. A dominant R wave in V_1 (i.e. greater than the S wave) may indicate right ventricular hypertrophy (Fig. 3.22).

Small amplitude QRS complexes are seen in obesity, chronic obstructive pulmonary disease (COPD), pericardial effusion and severe heart failure.

ST segment

This lies between the end of the S wave and the beginning of the T wave. It is usually on the isoelectric line (baseline). However, it can be elevated (in acute myocardial infarction or pericarditis) or depressed (in ischaemia, drugs, left ventricular hypertrophy) (Fig. 3.23a–c).

T waves

These may be taller than normal (hyperkalaemia, myocardial ischaemia) (Fig. 3.24), smaller than normal (hypokalaemia, pericardial effusion, hypothyroidism) or inverted (myocardial ischaemia, myocardial infarction, ventricular hypertrophy and digoxin toxicity).
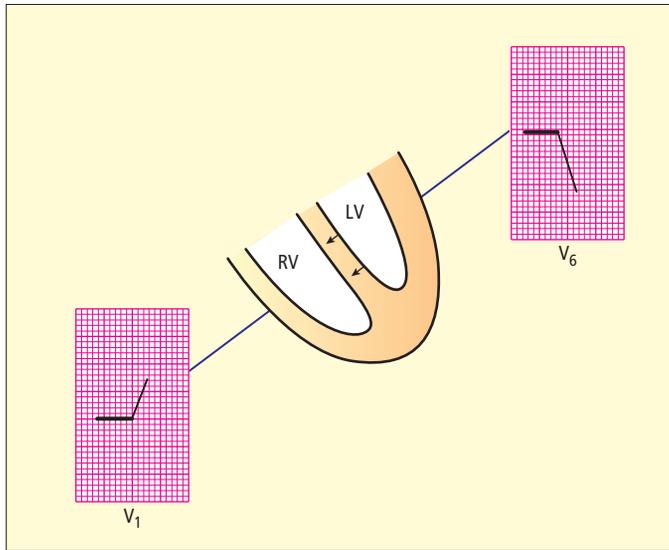
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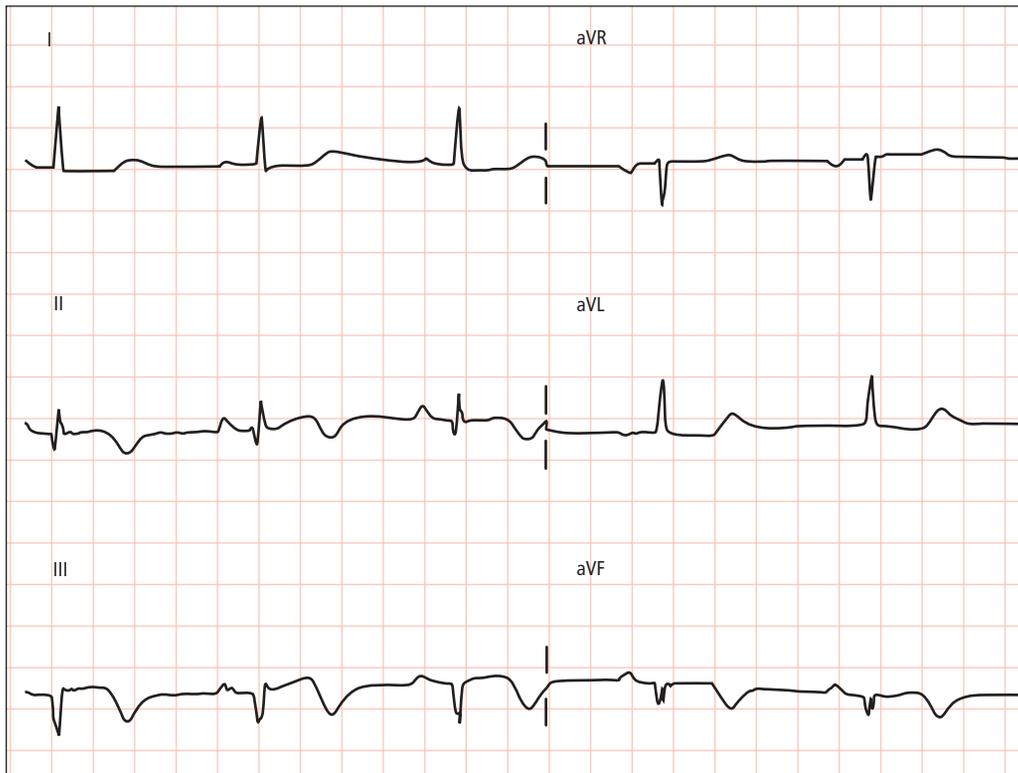**Fig. 3.19** Q wave in V_6 as a result of normal ventricular depolarization.

Fig. 3.20 Pathological Q waves in leads II, III and aVF in a patient 4 days following an inferior ST elevation myocardial infarction.
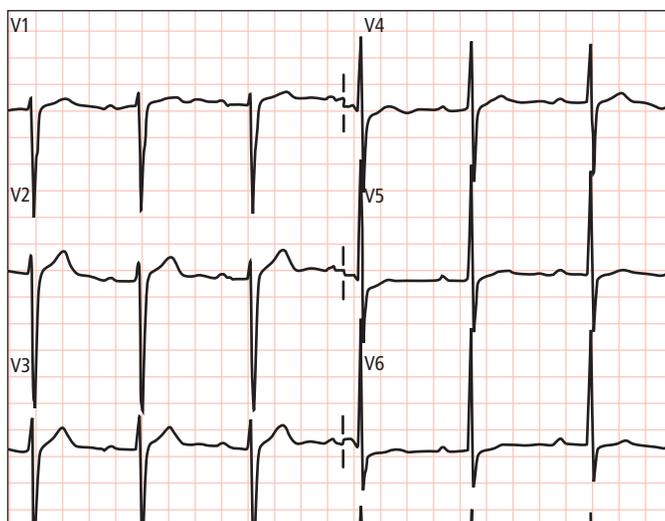
Fig. 3.21 Left ventricular hypertrophy with large QRS complexes across all of the chest leads.
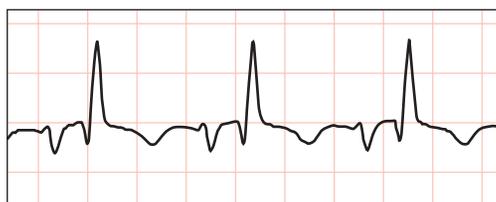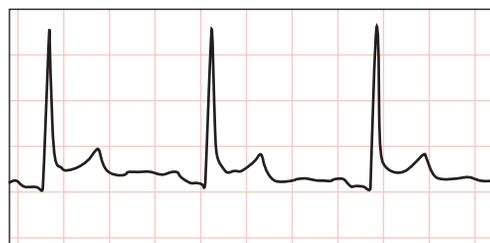

(a)

(b)

Fig. 3.22 Right ventricular hypertrophy with dominant R wave in V_1.

(c)

Fig. 3.24 Tall T waves associated with acute myocardial ischaemia.

Fig. 3.23 ST segment changes: (a) elevation associated with acute myocardial infarction; (b) depression seen with myocardial ischaemia; (c) elevation associated with acute pericarditis (note the saddle-shaped ST elevation in comparison with Fig. 3.16a).

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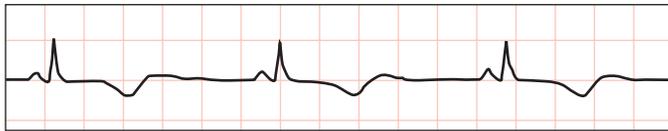
Fig. 3.25 Prolonged QT interval in patient with inherited long QT syndrome.

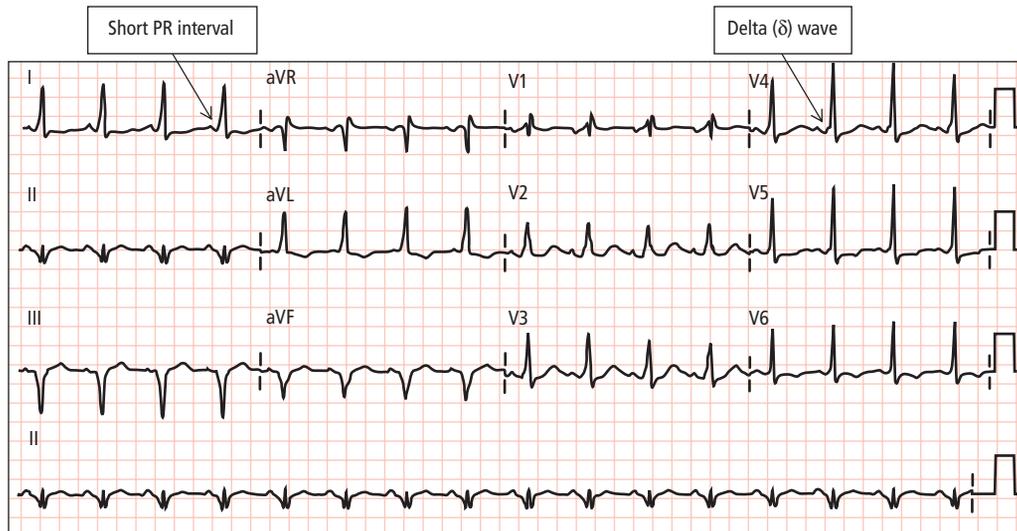Short PR interval

Delta (δ) wave

Fig. 3.26 Wolff–Parkinson–White syndrome with δ waves (small deflection at the start of the QRS), wide QRS and short PR interval.

QT interval

The QTc can be shorter than normal (hypercalcaemia and digoxin effect) or prolonged (drug induced, hypocalcaemia, inherited long QT syndrome) (Fig. 3.25).

δ Waves

These are small deflections seen at the start of the QRS complex in patients with Wolff–Parkinson–White syndrome. An accessory pathway connects the atria to the ventricles leading to a short PR interval, δ wave and a broad QRS complex (Fig. 3.26).