Electrical activity is a basic characteristic of the heart and is the stimulus for cardiac contraction. Disturbances of electrical function are common in heart disease. Their registration as an electrocardiogram (ECG) plays an essential role in the diagnosis and management of heart disorders.

**THE GENESIS OF THE ELECTROCARDIOGRAM**

Pathways of conduction and the electrocardiogram

The sinus node is situated in the right atrium close to the entrance of the superior vena cava. The atrioventricular node lies in the right atrial wall immediately above the tricuspid valve. The fibres of the AV bundle (of His) arise from the atrioventricular node and run along the posterior border of the septum between the ventricles (Fig. 1.1). On reaching the muscular part of the septum, they split into right and left bundle branches and then spread out in the subendocardium of the ventricles as the Purkinje system. The right bundle is a slender, compact structure. The left bundle soon splits into two or more divisions or fascicles, one of which proceeds anteriorly, sharing the same blood supply as the right bundle, and another is directed posteriorly.

In the usual sequence of events, the electrical impulse arises in the sinus node and spreads across the atria to reach the atrioventricular node. It can then only reach the ventricles by passing into the rapidly conducting atrioventricular bundle and its branches.

The first part of the ventricles to be activated is the septum, followed by the endocardium. Finally, the impulse spreads outwards to the epicardium.

The spread of the cardiac impulse gives rise to the main deflections of the electrocardiogram: P, QRS and T waves (Fig. 1.2):

- **The P wave** represents atrial depolarization.
- **The PR interval** represents the time taken for the cardiac impulse to spread over the atrium and through the AV node and His–Purkinje system.
- **The QRS complex** represents ventricular depolarization.
- **The T wave** represents ventricular repolarization.

**Electrodes and leads**

A conventional ECG consists of tracings from 12 or more leads. The term ‘lead’ refers to the ECG obtained as a result of recording the difference in potential between a pair of electrodes.
The bipolar (standard) leads
In these leads, the electrodes are attached to the limbs. In lead I the positive electrode is attached to the left arm and the negative to the right arm. In lead II the positive electrode is attached to the left leg and the negative to the right arm. In lead III the positive is attached to the left leg and the negative to the left arm. They may thus be depicted as:

- **lead I** = left arm minus right arm (LA–RA)
- **lead II** = left leg minus right arm (LL–RA)
- **lead III** = left leg minus left arm (LL–LA).

It can be deduced from these equations that lead II should be equal to the sum of leads I and III.

The position from which the heart is viewed by each of these leads is shown in Figure 1.3.

Unipolar leads
These have an exploring electrode placed on a chosen site linked with an indifferent electrode with a very small potential. In an attempt to obtain a
central terminal with ‘zero potential’, Wilson connected all three limb electrodes through 5000 Ω resistances to form the indifferent electrode.

**Unipolar chest leads**

When unipolar leads are recorded from the chest wall, the exploring electrode is connected to the positive pole of the ECG and the negative to the central terminal of Wilson. By convention, the following sites are normally selected (Fig. 1.4):

- **V1**, the fourth intercostal space just to the right of the sternum
- **V2**, the fourth intercostal space just to the left of the sternum
- **V3**, midway between V2 and V4
- **V4**, the fifth intercostal space in the midclavicular line
- **V5**, the left anterior axillary line at the same horizontal level as V4
- **V6**, the left midaxillary line at the same horizontal level as V4.
Additional leads can be taken from V3R and V4R, sites on the right side of the chest equivalent to V3 and V4. Occasionally, leads may be placed at higher levels, for example the second, third or fourth intercostal spaces or further laterally (V7 and V8).

**Unipolar limb leads**

In these leads, the exploring electrode is placed on one limb, and the negative pole is connected to Wilson’s central terminal, modified by the omission of the connection from the limb under study to the central terminal. This modification augments the voltage of the ECG, and the leads so derived are referred to as ‘a’ leads. They are designated as follows:

- aVR, right arm lead
- aVL, left arm lead
- aVF, left foot lead.

The resulting lead orientations and their relation to the standard bipolar leads are presented in Figure 1.15 on page 15.

**THE NORMAL ELECTROCARDIOGRAM**

Normally, ECGs are recorded at a rate of 25 mm/s and the ECG paper is printed with thin vertical lines 1 mm apart and thick vertical lines 5 mm apart (Fig. 1.5). The interval between the thin lines represents 0.04 s and that between two thick lines 0.20 s. If the heart rhythm is regular, the rate can be counted by dividing the number of small squares between two consecutive R waves into 1500 or large squares into 300.

There are also thin horizontal lines at 1-mm intervals and thick horizontal lines at 5-mm intervals. An ECG recording is standardized so that 1 mV gives a deflection of 10 mm on the paper. The height of a deflection therefore indicates its voltage.
The P wave

The normal P wave (Fig. 1.6A) results from the spread of electrical activity across the atria (the activity of the sinus node itself cannot be detected in the ECG). Because the impulse spreads from right to left, the P wave is upright in leads I, II and aVF, is inverted in aVR and may be upright, biphasic or inverted in lead III, aVL and V1. It should not be higher than 3 mm in the bipolar leads or 2.5 mm in the unipolar leads, or greater than 0.10 s in duration.

When abnormal, the P wave may become:

- **inverted** (i.e. negative in the leads in which it is usually positive). This indicates depolarization of the atria in an unusual direction, and that the pacemaker is not in the sinus node, but is situated either elsewhere in the atrium, in the AV node or below this; or there is dextrocardia

- **broadened and notched**, due to delayed depolarization of the left atrium when this chamber is enlarged (P mitrale) (Fig. 1.6B). In V1, the P wave is...
then usually biphasic with a small positive wave preceding a deep and broad negative one
- tall and peaked, exceeding 3 mm, as a result of right atrial enlargement (P pulmonale) (Fig. 1.6C)
- absent or invisible due to the presence of junctional rhythm or sinoatrial block
- replaced by flutter or fibrillation waves.

**PR interval**

This is measured from the beginning of the P wave to the beginning of the QRS complex (i.e. to the onset of the Q wave if there is one, and to the onset of the R wave if there is not). This interval corresponds to the time taken for the impulse to travel from the sinus node to the ventricular muscle. There is an isoelectric segment between the end of the P wave and the beginning of the QRS, whilst the impulse is passing through the AV node and the specialized conducting tissue, as an insufficient amount of tissue is being electrically stimulated to produce a deflection detectable on the body surface.

The PR interval varies with age and with heart rate. The upper limit in children is 0.16, in adolescents 0.18 and in adults 0.20 s, although it may be even longer in a few normal individuals. The faster the heart rate the shorter is the PR interval. It is regarded as abnormally short if it is less than 0.10 s. A shortened PR interval is seen when the impulse originates in the junctional tissue and in the Wolff–Parkinson–White syndrome (see p. 165). The PR interval is prolonged in some forms of heart block (see p. 181).

**The QRS complex**

The QRS complex represents depolarization of the ventricular muscle. The components of the QRS complex are defined as follows (Fig. 1.7):

- The R wave is any positive (upward) deflection of the QRS. If there is more than one R wave, the second is denoted R'; an R wave of small voltage may be denoted r.
- A negative (downward) deflection preceding an R wave is termed Q.

![Fig. 1.7 Variations in the QRS complex (see text).](image)
• A negative deflection following an R wave is termed S.
• If the ventricular complex is entirely negative (i.e. there is no R wave), the complex is termed QS.

The whole complex is often referred to as the QRS complex irrespective of whether one or two of its components are absent.

Ventricular depolarization starts in the middle of the left side of the septum and spreads across to the right (phase 1 of ventricular depolarization) (Fig. 1.8). Subsequently, the main free walls of the ventricles are activated, the impulse spreading from within outwards and from below upwards. Because of the dominating bulk of the left ventricle, the direction of the vector of phase 2 is to the left and posteriorly. Finally, the base of both ventricular walls and the interventricular septum are depolarized. The appearances of the QRS in different leads can be largely explained by the major vectors of these phases as is seen in Fig. 1.8. In leads facing the left ventricular surface, there is a small Q wave due to septal depolarization and a large R wave due to left ventricular depolarization. On the right side of the heart, as seen from V1, there is usually an r wave due to septal depolarization and a large S wave due to left ventricular forces directed away from the electrode.

Pathological Q waves

As mentioned, small, narrow Q waves are normally to be found in leads facing the left ventricle (e.g. lead I, aVL, aVF, V5 and V6). These Q waves do not normally exceed 2 mm in depth, or 0.03 s in width. It should be noted that QS waves are normal in aVR, and are common in V1. Abnormally broad and deep Q waves are often a feature of myocardial infarction (see p. 110). Q waves in lead III are difficult to evaluate but can be ignored if there are no Q waves either in lead II or in aVF, or if they do not exceed 0.03 s. Usually, a ‘normal’ Q wave in lead III diminishes or disappears on deep inspiration because of an alteration in the position of the heart, whilst the ‘pathological’ Q wave of infarction persists.
The QRS complex should not exceed 0.10 s in duration, and usually is in the range 0.06–0.08 s. Broad QRS complexes occur in bundle branch block (p. 12), in ventricular hypertrophy and in ventricular ectopic beats.

The T wave

The T wave is due to repolarization of the ventricles. If repolarization (the T wave) occurred in the same direction as depolarization (the QRS complex) the T wave would be directed in an opposite way to that of the QRS. In fact, depolarization takes place from endocardium to epicardium, whereas repolarization takes place from epicardium to endocardium. Because of this, the T wave usually points in the same direction as the major component of the QRS complex. Thus, the T wave is normally upright in leads I and II as well as in V3 to V6, is inverted in aVR, and may be upright or inverted in lead III, aVL, aVF and V1 and V2.

The T waves are usually not taller than 5 mm in standard leads and 10 mm in precordial leads. Unusually tall and peaked T waves may be seen in hyperkalaemia and in early myocardial infarction. Flattened T waves are seen when the voltage of all complexes is low, as in myxoedema, as well as in hypokalaemia and in a large number of other conditions in which it may be regarded as a nonspecific abnormality. Slight T wave inversion is also often non-specific, and may be due to such influences as hyperventilation, posture and smoking. The most important causes of T wave inversion are:

- myocardial ischaemia and infarction
- ventricular hypertrophy
- bundle branch block.

Detailed descriptions of T wave changes will be found in the subsequent section on abnormalities of the ST segment, and also under the subheadings dealing with ventricular hypertrophy, bundle branch block and myocardial infarction.

The QT interval

The QT interval represents the total time from the onset of ventricular depolarization to the completion of repolarization. It is measured from the beginning of the Q wave (or the R wave if there is no Q wave) to the end of the T wave. Its duration varies with heart rate, becoming shorter as the heart rate increases. In general, the QT interval at heart rates between 60 and 90 beats/min does not exceed in duration half the preceding RR interval. The measurement of the QT interval is often difficult as the end of the T wave cannot always be clearly identified, and the relationship between heart rate and duration of the QT is a complex one. Tables are available in textbooks of electrocardiography giving normal QT intervals. In practice, the main importance of a prolonged QT interval is that it is associated with a risk of ventricular tachycardia (particularly torsades de pointes, p. 179) and sudden death. A long QT is sometimes an inherited abnormality but may result from such drugs as quinidine, procainamide, disopyramide, amiodarone and tricyclic antidepressants.
The ST segment

The ST segment is that part of the ECG between the end of the QRS complex and the beginning of the T wave (Fig. 1.9). The point of junction between the S wave and the ST segment is known as the J point. The ST segment occurs during a period of unchanging polarity in the ventriles, corresponding with the plateau of the action potential. The normal ST segment is situated on the isoelectric line but curves upwards.

Displacements of the ST segment and variations in its shape are of great importance in electrocardiographic diagnosis. The characteristic abnormalities of the ST segment are illustrated in Fig. 1.9. In some normal individuals, particularly young people of African descent, slight ST elevation is seen. This may be up to 1 mm in standard leads and 2 mm in the right precordial leads. Depression of more than 0.5 mm is abnormal. When ST elevation occurs in normal individuals, it is often preceded by a slight notch on the downstroke of the R wave:

- Acute myocardial infarction. The ST segment is elevated with a curve which is convex upwards in the leads facing the infarct. At a later stage ST segment elevation becomes less pronounced as T wave inversion develops. These changes are considered in more detail on p. 110.
- Pericarditis. This also causes ST elevation, but the ST segments are concave upwards and the changes are widespread rather than localized as in myocardial infarction.
• **Digitalis therapy.** This depresses the ST segment, particularly in leads II and III, so that there is a gentle sagging, but the T wave remains upright or flattened.

• **Ventricular hypertrophy.** ST segment depression may occur in leads facing the relevant ventricle and be accompanied by asymmetrical T wave inversion. This contrasts with the symmetrical T wave inversion seen in myocardial infarction and ischaemia.

• **Acute myocardial ischaemia.** The ST segment is horizontally depressed or slightly downward sloping from the J point onwards.

• **Sinus tachycardia.** There may be ST depression which slopes upwards from the J point.

• **Hypothermia.** There is a prominent J wave (the junction of the S wave and the ST segment) (Fig. 1.10).

### The U wave

The U wave is a broad, low-voltage wave present in most normal ECGs. Its cause is unknown; it may become unusually prominent in hypokalaemia and with digitalis therapy.

### ABNORMAL ELECTROCARDIOGRAM PATTERNS

#### Left ventricular hypertrophy (Fig. 1.11)

Hypertrophy of the left ventricle increases the amplitude of R waves in left chest leads and S waves in right chest leads. Where there is septal hypertrophy, deep but narrow Q waves are seen in left chest leads. When left ventricular hypertrophy becomes advanced, the T wave may become flattened in the leads in which the R wave is tall; eventually ST depression and T wave inversion may occur.

Many efforts have been made to lay down criteria for the diagnosis of left ventricular hypertrophy. None is satisfactory as many factors contribute to the amplitude of ECG waves, including the thickness of the chest wall and the age of the patient. The following criteria have gained wide acceptance:

• R in V5 or V6 plus S in V1 greater than 35 mm. This criterion applies only in individuals over 25 years of age. In younger persons, R in V5 or V6 plus S in V1 should exceed 40 mm before the diagnosis of left ventricular hypertrophy can be made.
Right ventricular hypertrophy (Fig. 1.12)

When the right ventricle becomes hypertrophied, the leads facing the right ventricle (particularly in V1, V3R and V4R) show dominant R waves instead
of the usually dominant S wave. The diagnostic criterion for right ventricular hypertrophy is:

- R wave in V1 equal to or greater than the S wave and at least 5 mm tall.

As with left ventricular hypertrophy, ST depression and T wave inversion may develop in the leads with tall R waves.

**Left bundle branch block (Fig. 1.13)**

When the left branch of the bundle is blocked, the interventricular septum is activated from the right instead of from the left side and the initial vector (phase 1) is directed to the left. Because of this, the normal initial q wave in the left ventricular leads is lost, being replaced by a small r wave. Right ventricular depolarization, which follows, produces an r in V1 and an s in V6. The left ventricle is finally depolarized resulting in an R' in V6 and a broad S in V1. The QRS duration is increased to 0.12 s or more.

The abnormal left ventricular depolarization sequence in left bundle branch block causes secondary repolarization changes. Consequently, the ST segment and T wave are abnormal. This prevents interpretation of other factors causing ST and T wave changes, such as ischaemia and infarction.

**Right bundle branch block (Fig. 1.14)**

In this disorder, the right branch of the bundle is blocked, but the septum is activated from left to right, as in the normal heart. The left ventricular q wave is preserved, as is the initial r wave over right chest leads. The left ventricle is then depolarized, producing an S wave in right chest leads and an R wave in left chest leads. Finally, depolarization reaches the right ventricle, and so produces an R' in the right chest leads and a deep broad S wave in the left chest leads. An M pattern is thus seen in the right chest leads, such as V1. It is also common to see T wave abnormalities in leads V2 and V3.

**The mean frontal QRS axis**

The total electrical activity at any one moment of time can be summed and represented by a single electrical force of a certain magnitude and in a certain direction, termed the instantaneous vector. All the instantaneous vectors occurring during the inscription of the QRS complex can be averaged, the direction of the vector so derived being called the mean QRS axis. It is customary to measure this only in the frontal plane, based on the orientation of the limb leads (Fig. 1.15). The limb lead with the tallest R wave will be closest to the QRS axis.

An alternative method of deriving the mean frontal QRS axis is to find in which of the leads I, II, III, aVR, aVL and aVF, the deflections of the QRS above and below the line are most nearly equal. The mean frontal QRS axis is at right angles to this lead.

Left axis deviation is present when the axis is less than −30° and right axis deviation when the axis is greater than +110°.
Calculation of the mean frontal QRS axis is of limited value except in a few conditions such as the differentiation of ostium primum from ostium secundum atrial septal defects (see p. 278).

Left axis deviation is often due to block in the anterior division (fascicle) of the left bundle branch, and when associated with right bundle branch block is a frequent precursor of complete heart block.

Right axis deviation commonly accompanies right ventricular hypertrophy, but may be due to block of the posterior fascicle of the left bundle.

Fig. 1.13  Left bundle branch block. (A) The initial vector is abnormal in being from right to left across the septum, thus producing an initial r wave in V6 and a q wave in V1. (B) 12-lead ECG demonstrating features of left bundle branch block.
ELECTROCARDIOGRAM INTERPRETATION

ECG interpretation is largely a matter of experience and pattern interpretation. However, while building experience, it is useful to develop a method of ‘systematic’ ECG analysis. This is most easily performed by asking oneself a number of questions in a logical sequence about P, QRS and T waves in turn. A simple system is presented in Box 1.1.

Fig. 1.14 Right bundle branch block. (A) The septum is depolarized normally from left to right and hence a small q is preserved in left ventricular leads and a small r in right ventricular leads. Left ventricular depolarization produces an s wave in V1 and an R wave in V6. Late depolarization of the right ventricle results in a prominent R’ wave in V1 and broad S wave in V6. (B) 12-lead ECG showing features of right bundle branch block.
CHAPTER 1

The electrical activity of the heart: the electrocardiogram

Fig. 1.15  Hexaxial reference system. The orientation of the limb leads in the frontal plane is shown, together with the normal range for the mean frontal QRS axis.

Box 1.1  A system of ECG interpretation

Rate and rhythm
What is the rate (see p. 4)?
Is it regular or irregular?

P wave
Are P waves present?
Is the P wave axis normal (p. 5)?
Is there evidence of left or right atrial enlargement (p. 6)?

PR interval (normal range 0.12–0.20 s)
Is the PR interval normal?
Is each QRS complex preceded by a P wave?
Is there evidence of a slurred QRS upstroke (delta wave) (p. 165)?

QRS complex
Is the QRS duration within normal limits (0.08–0.11 s)?
Is there evidence of bundle branch block (p. 12)?
Is the QRS axis normal (p. 12)?
Are pathological Q waves present (p. 7)?
Is there a normal R wave progression across the chest leads (p. 7)?

ST segment and T wave
Is there abnormal ST elevation or depression (p. 9)?
Are the T waves upright (except aVR and V1)?

QT interval
Is the QT interval normal (in general less than 0.44 s)?
FURTHER READING

