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The electrocardiogram in ischaemic heart disease

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INTRODUCTION

The electrocardiogram (ECG) underpins almost every aspect of cardiology, and mastering the skills of ECG interpretations remains a core learning objective for medical students, nurses, technicians and other paramedical staff. A clear understanding of the ECG is absolutely necessary for monitoring patients undergoing a variety of procedures, and is critical for the diagnosis of many cardiac pathologies. Monitoring and identifying arrhythmias requires an appreciation of cardiac anatomy, the electrical conduction system, and ischaemic abnormalities. This chapter describes the basic components of the cardiac conduction system and reviews the standard methods for obtaining and interpreting a 12-lead ECG, with emphasis on the characteristics of ischaemia, infarction and specific arrhythmias.

ELECTRICAL ACTIVATION OF THE HEART

Anatomy

The electrical system of the heart consists of specialised muscle fibres. These are unrecognisable from contractile muscle by the naked eye and require histochemical techniques to be recognised. The components of this system are the sino-atrial (SA) node, the inter-nodal pathways, the atrio-ventricular (A-V) node, the Bundle of His and the Purkinje system (Figure 19.1). It is interesting that the discovery of the various parts of the pathway was in reverse order of activation, commencing with Purkinje in 1845 and finishing with Keith & Flack discovering the sinus node in 1907.

The S-A node is the primary cardiac pacemaker and is found high in the right atrium, just antero-lateral to its connection with the superior vena cava (SVC). It is approximately 25 mm in length and is richly supplied with autonomic nerve fibres and blood vessels. It is connected to the A-V node by three internodal tracts—the anterior, middle and posterior internodal tracts. The anterior tract passes anteriorly and to the left of the superior vena cava, entering the anterior inter-atrial band, which splits into two parts, the first passing to the left atrium (Bachmans' Bundle), the second descending anteriorly in the inter-atrial septum to the A-V node. The middle internodal tract runs in the inter-atrial septum to the A-V node, while the posterior internodal tract terminates with most of its fibres bypassing the proximal and middle portions of the AV node to enter the distal portion.

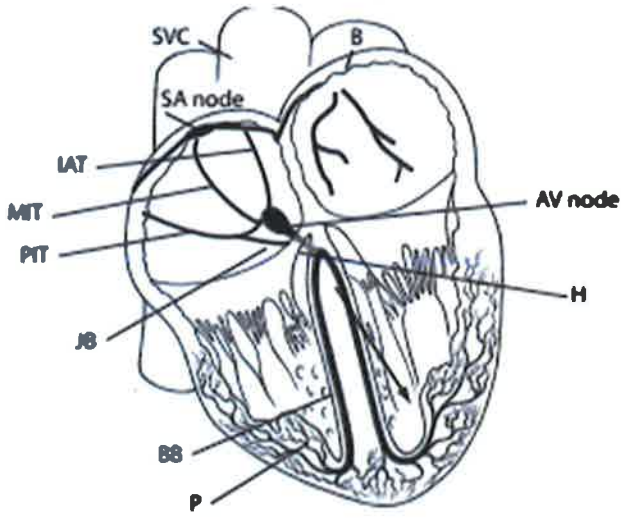

Figure 19.1 The electrical system of the heart. SVC, superior vena cava; LA, left atrium; B, Bachman's bundle of the anterior internodal tract; IAT, interatrial bundle of anterior internodal tract; MIT, middle internodal tract; PIT, posterior internodal tract; JB, James' bypass fibres; His, common bundle of His; BB, bundle branches; P, Purkinje network.

The AV node is approximately 6 × 3 × 2 mm in size and lies in the right atrium, on the right side of the inter-atrial septum, in front of the opening of the coronary sinus above the attachment of the septal cusp of the tricuspid valve. The distal "tail" of the A-V node is contiguous with the Bundle of His, which is approximately 20 × 3 mm. It bifurcates early into "branching" and "penetrating" segments. The branching segments arise immediately distal to the A-V node in the proximal His Bundle. It branches proximally to form the posterior fibres of the left bundle branch, then divides to form the anterior fibres of the left bundle branch and the right bundle branch.

The "penetrating" branch runs through the central fibrous structure and has no contact with myocardium. The right main bundle runs down the right side of the interventricular septum towards the apex. Initially it lies deep in the endocardium and has only a few branches, but on reaching the moderator band its free edge runs inwards to the base of the anterior papillary muscle of the right ventricle, where it branches to supply the whole of the right ventricular endocardium. The left main bundle emerges on the left side of the interventricular septum (IVS), just below the non-coronary cusp of the aortic valve and passes down the septum, sending branches into the septum until about a third of the way down the IVS. Here it breaks into posterior and anterior branches that pass the postero-medial and antero-lateral papillary muscles, where it branches to form the complex Purkinje network of fibres supplying the left ventricular endocardium.

Mode of activation of myocardium

The normal impulse originates in the S-A node and passes longitudinally through the atria as a "wavefront", initially activating the right atrium in a rightwards and anterior direction. This is followed by activation of the left atrium, in a left and posterior direction. The "wavefront" is rapid,

travelling longitudinally and contiguously at approximately 1000 mm/second throughout the atrial muscle.

The impulse arrives at the A-V node via the specialised internodal pathway, where it is delayed due to decremented conduction. The earliest sub-endocardial depolarisation is detected simultaneously on the left central side of the interventricular septum, and on the high anterior and infero-apico-septal regions. The wave of depolarisation then spreads transversely from endocardium to epicardium through the thickness of the left and right ventricular walls. The epicardial depolarisation is detected first on the right antero-apical region, followed by the anterior and posterior para-septal regions of the left ventricle. The lateral wall and basal septum are activated last. The Purkinje system extends to varying depths into the sub-endocardial layer in each individual, penetrating up to 3–4 mm into the free ventricular walls from the endocardium. Activation of the sub-endocardial layer (which is electrically silent) is not recorded on the surface electrocardiogram (ECG). It was originally theorised by Sodi-Pallares[1–4] that the island of Purkinje tissue acted as a "closed polarised island" where activation from the many Purkinje fibres spreads outwards in a "spherical" manner through the myocardium. It was not until activation reached the endocardium that the "closed islands" opened and net activation began to spread to the epicardium so a positive potential would be recorded (Figure 19.2). Sodi-Pallares referred to the (electrically silent) Purkinje ridge sub-endocardial layer as the "electrical sub-endocardial surface". The thickness or depth of this sub-endocardial layer is highly variable. Durrer[5] and others subsequently showed the complete sequence of ventricular activation of the heart.

On the ECG, when the wave of depolarisation moves towards a positive pole, the deflection is positive; when it moves away from the positive pole, or towards a negative pole, the deflection is negative. In an isolated muscle strip, depolarisation (an advancing wave of positive charge) and repolarisation (a wave of negative charge) both take place from the endocardium to epicardium. The result of this is that the polarity of repolarisation in the muscle strip is the opposite to that of depolarisation.

In the intact human heart, however, depolarisation also takes place from endocardium to epicardium but repolarisation takes place in the opposite direction, from epicardium to endocardium. Hence, polarity of repolarisation is the same as for depolarisation. A small number of individuals, however, especially athletes, may have their repolarisation process as in the isolated muscle strip, so in these individuals T wave inversion may be seen in many leads, particularly the precordial leads.

Thus, depolarisation begins on the left side of the interventricular septum and spreads outwards through the free walls of the ventricles. From an electrocardiographic point of view, the ventricles consist of three muscle masses, the interventricular septum and the free walls (muscle masses) of the right and left ventricles (Figure 19.3a).

The standard surface-recorded ECG is the sum of the potential electrical forces recorded in

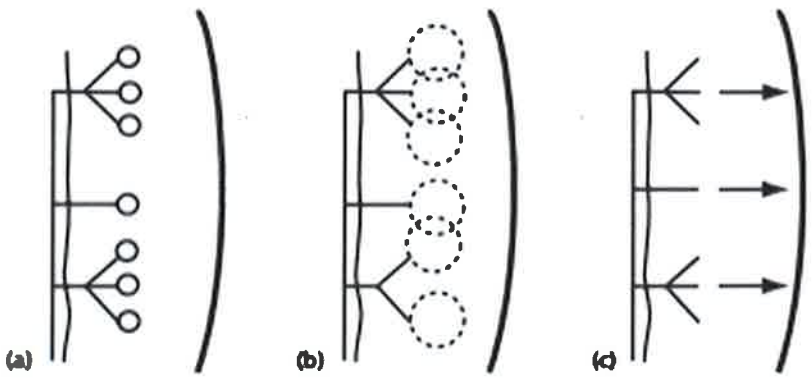

Figure 19.2 Diagrammatic representation of Sodi-Pallares' "electrical endocardial surface" concept. Electrical activation arrives at the endocardial surface via the Purkinje fibres. At first, activation is restricted to closed polarised spheres, or "closed islands" (a). The spheres coalesce and open to the endocardium (b), forming a progressive electrical wavefront spreading outwards and transversely (c) through the ventricular free wall.

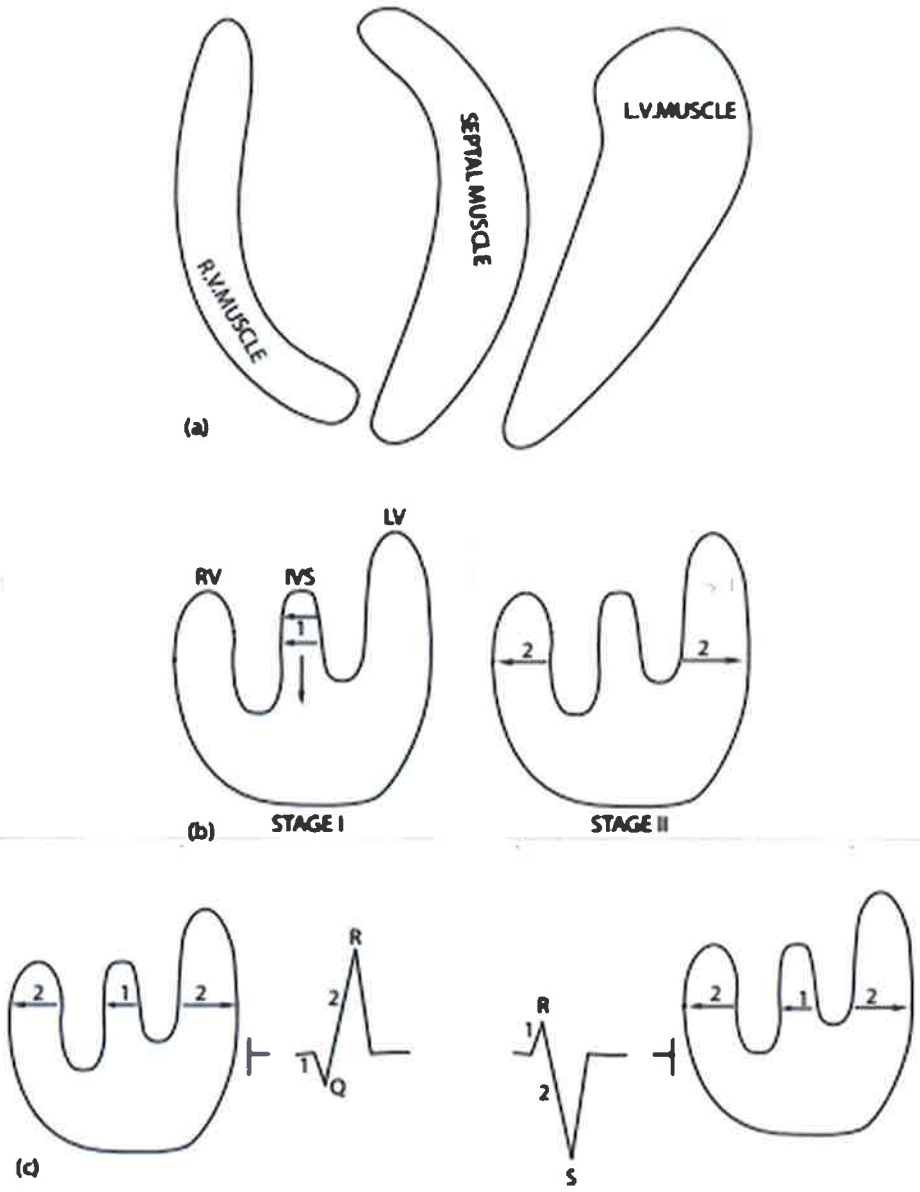

Figure 19.3 (a) The ventricle may be considered electrographically as three separate muscle masses, as shown. (b) Activation of ventricular muscle proceeds first from the interventricular septum (IVS) (1), then spreads across the rest of the muscle mass (2). RV, right ventricle; LV, left ventricle. Arrows indicate direction of signal. (c) The size of the electrical potential depends on the thickness of each of the three muscle masses. As the left ventricular wall mass is considerably thicker than that of the right ventricle, its signal is correspondingly stronger and overrides that of the right ventricle on the ECG trace. Arrows and numbers indicate sequence and direction of signal.

various planes. Activation of the interventricular septum occurs first (stage 1) and is followed by activation of the rest of the muscle mass (stage 2) (Figure 19.3b).

Because the mass of the left ventricle (LV) is much greater than that of the right ventricle (RV), the electrical potential from the LV overrides that of the RV on surface ECG electrodes (Figure 19.3c).

STANDARD ECG LEADS AND ELECTROCARDIOGRAPHIC INTERPRETATION

Einthoven's triangle (Figure 19.4a) is derived from leads placed on the right and left wrists and the left ankle. The standard ECG is made up of 12 leads: Three standard bipolar leads I, II and III derived from

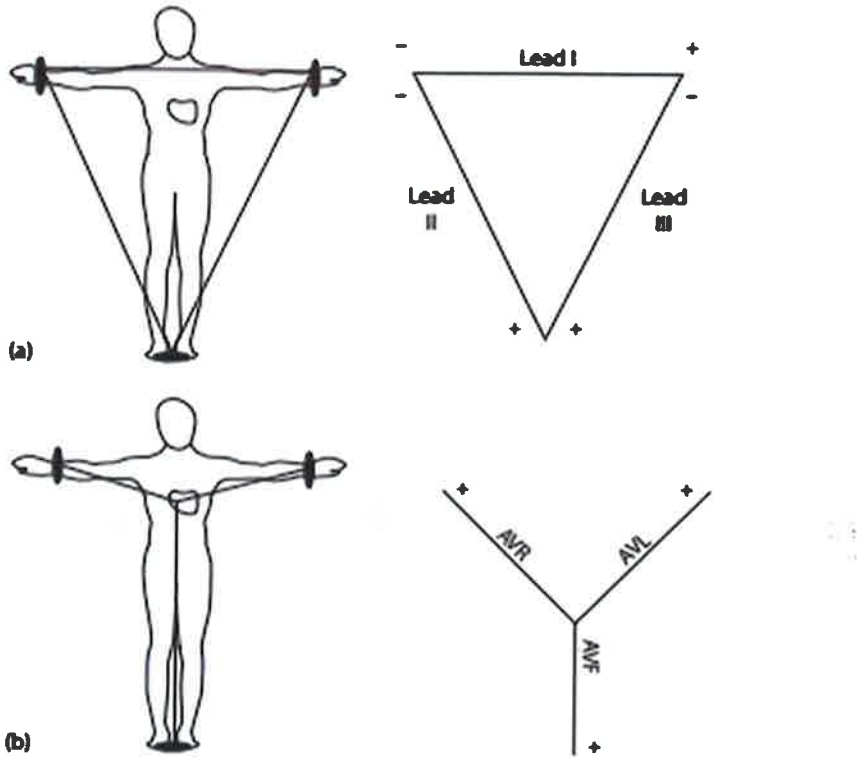

Figure 19.4 (a) Einthoven's triangle, showing the three lines of reference derived from the limb leads. (b) Three additional lines of reference formed from the augmented limb leads.

Einthoven's equilateral triangle, plus three augmented extremity leads, which are uni-polar leads and are prefixed with the letter "a", plus six chest "V" leads.

The augmented uni-polar leads on the arms and legs are seen as an extension of the torso and record actively from each peripheral electrode (Figure 19.4b). Following is a brief description of the 12 basic ECG leads:

I – Lead I connects the left and right wrists.
II – Lead II connects the right arm and left ankle.
III – Lead III connects the left arm and left ankle.
aVR – The right arm augmented unipolar lead effectively records conduction from the right shoulder.
aVL – The left arm augmented unipolar lead records conduction from the left shoulder.
aVF – The left leg augmented unipolar lead records conduction from the left thigh.
V1 – This lead is placed on the 4th right intercostal space, 2.5 cm from the midline of the sternum.
V2 – This lead is placed on the 4th left intercostal space, 2.5 cm from the middle of the sternum.
V3 – This lead is placed halfway between V2 and V4.
V4 – This lead is placed on the 5th left intercostal space in the mid-clavicular line.
V5 – This lead is placed on the anterior axillary line at the same level as V4.
V6 – This lead is placed on the mid-axillary line at the same level as V5.

The standard and augmented unipolar leads are orientated through the frontal or coronal plane of the body, whereas the precordial unipolar or V leads are orientated through the horizontal body plane. The Hexagonal reference system is an amalgamation of the orientation of the standard bipolar and augmented unipolar leads, placed through a central point of the heart, and is used to calculate the electrical axis of the heart (Figure 19.5).

PQRST complex

The PQRST complex is a recording of the electrical cardiac activity detected by skin electrodes. The P wave represents atrial activation, the QRS complex represents ventricular depolarisation, and the T wave represents ventricular repolarisation. Atrial repolarisation can occasionally be seen as a "ta" wave interposed in the QRS complex, but is generally lost in the stronger T wave.

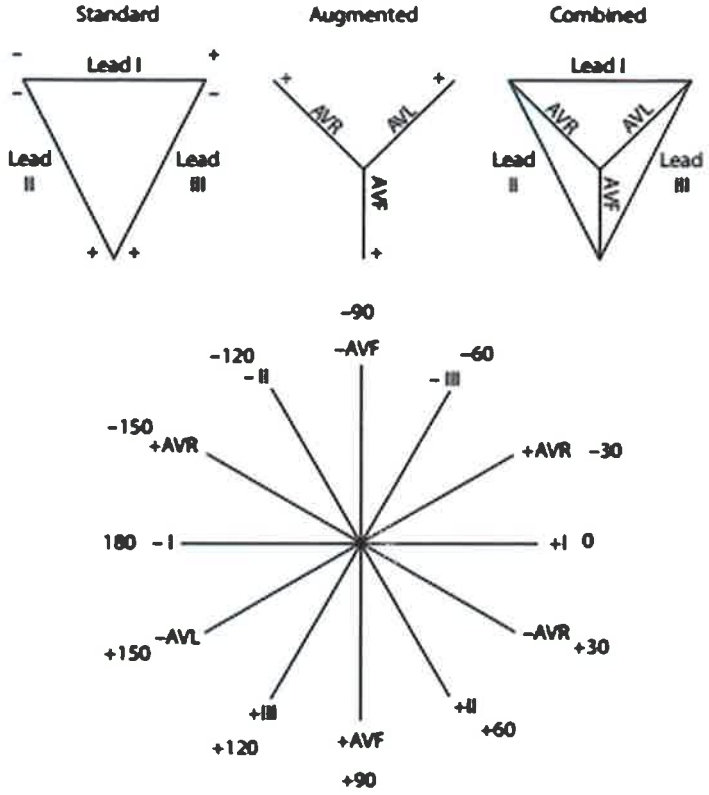

Figure 19.5 Amalgamation of the standard and augmented limb leads to form the hexagonal reference system in the frontal plane.

The PR interval (or PQ interval) represents the time from onset of atrial depolarisation to that of ventricular depolarisation, where the impulse has travelled through the atria and down the specialised internodal pathway to the A-V node. It then traverses the His Bundle, the three bundle branches, the left posterior, left anterior and right tracts, and the Purkinje system, finally activating the electrical endocardial surface of the Sodi-Pallares system.

QRS complex

The QRS complex is the net resultant electrical potential generated from ventricular depolarisation as seen by the appropriate surface electrode. Activation in the ventricle commences in the left side of the interventricular septum and spreads to the right and left surface, then activates the ventricle. Activation proceeds in the anterior and posterior regions adjacent to the septum (the resultant is electrically neutral) and then traverses the apical and lateral walls of both ventricles. The last areas activated are the lower portion of the interventricular septum and the low posterior wall adjoining it.

QRS interval

This is usually 0.06–0.10 ms in adults, tends to last longer in men and should be measured from the widest QRS complex, usually found in the mid precordial leads.

QRS axis

Axis is the direction of the dominant line of electrical excitation through the heart. It lies between −30° to +90° in adults and −30° to +110° in children and adolescents. A slight variation can occur in the same individual from time to time. Axis determines whether conduction is affected by factors such as infarction and hypertrophy and can be judged roughly by inspecting the direction of the R waves in leads I to III. Figure 19.6a shows examples of normal axis, left axis deviation and right axis deviation. To obtain a more accurate assessment

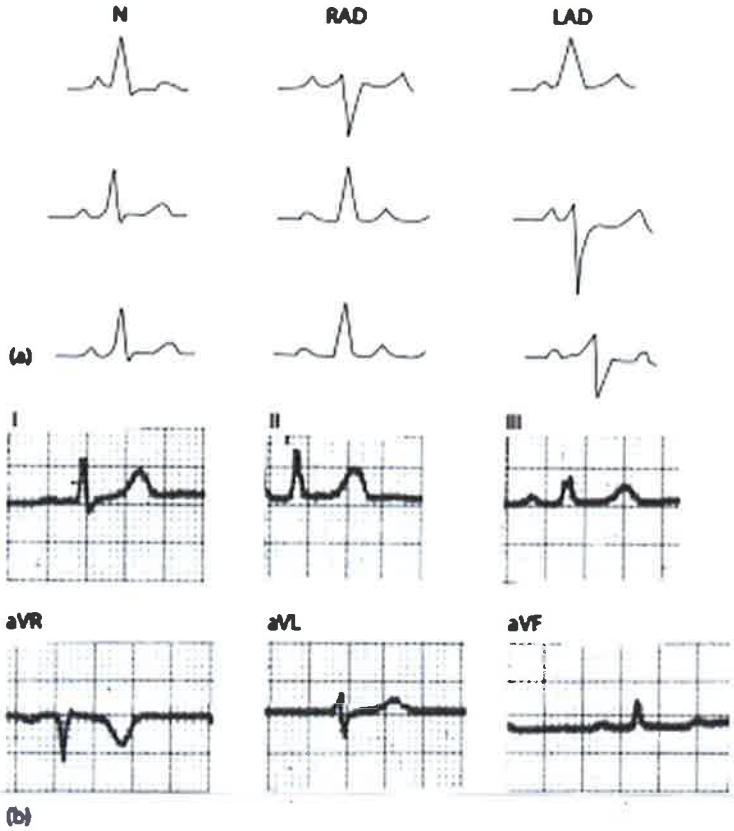

Figure 19.6 (a) Direction of the QRS complex is determined by the axis of the heart. At left is its appearance with a normal axis (N), in the middle is its appearance with right axis deviation (RAD), and at right is its appearance with left axis deviation (LAD). (b) Example of QRS direction with an axis rotated to +60°.

the hexagonal system may be used. First, the ECG lead with the greatest deflection (tallest R wave), represents the major direction of electrical axis. This is compared with the most equiphasic leads where the R and S deflections are equal. The electrical axis lies at 90° to this lead, and in the direction of the lead with the tallest R wave.

Figure 19.6b illustrates an example. In this case the lead with the tallest positive R wave is lead II, so the major direction of electrical axis is in this direction, which is +60°. The most equiphasic lead is aVL (the axis of aVL is −30°). The electrical axis of the heart lies at 90° to the aVL lead and in the direction of lead II, which is +60°.

MYOCARDIAL INFARCTION AND ISCHAEMIA

The hallmark of myocardial infarction is the development of pathological Q waves. These are waves greater than 0.04 seconds in width and more than 1/3 the height of the R wave, or complete loss of the R wave with replacement by a Q wave. Q waves are present when 40% or more of the transmural thickness of the myocardial wall is infarcted or necrotic (Figure 19.7).

Necrotic or scar tissue is electrically inactive so that an electrical "window" appears when an electrode is placed over the infarct. In Figure 19.7, the interventricular septum and right ventricular free wall are recorded from the electrode facing the left ventricular free wall. When cardiac tissue is ischaemic or injured, it becomes electrically negative, whereas the adjacent normal tissue remains positively charged. As a consequence, a negative current of injury is recorded from the injured surface by an electrode facing it (as in Figure 19.7), and a continuous positive current is recorded by an electrode facing the normal tissue adjacent to the injured tissue (as in Figure 19.8a).

In widespread sub-endocardial ischaemia or unstable angina, the whole endocardium is

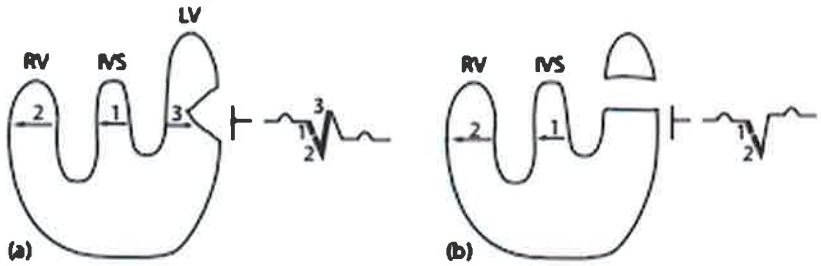

Figure 19.7 (a) An electrode placed over an infarcted segment of myocardium (3) records the current in regions (1) and (2) as moving away from the electrode, i.e. segment (3) is transparent electrically, hence forming pathological Q waves (a positive charge moving away from a positive electrode records a negative deflection on ECG). The illustration in (b) depicts a transmural infarct with only a QS complex. There is no R wave.

negatively charged. The ECG shows widespread varying ST depression due to an elevated baseline resulting from the continuous positive current (Figure 19.8b) transmitted from the normal tissue adjacent to the injured tissue. The changes occur because the continuous current related to injury affects the resting electrical baseline, but with depolarisation the true electrical baseline is re-assumed.

Myocardial infarction, however, is not usually an all-or-nothing situation. The region involved in necrosis usually contains patches of injured, ischaemic or even normal tissue. Adjacent to the most damaged portion is a surrounding area of injured tissue, and adjacent to that is ischaemic tissue (Figure 19.9).

The site of infarction relates to the coronary artery involved. Extended infarction relates to the position of the coronary lesion, such that the more proximal the lesion, generally the more extensive the infarction (taking into consideration other mitigating factors such as use of thrombolytic therapy, the extent of the luminal thrombotic occlusion, the presence or absence of diabetes mellitus, degree of collateralisation and other factors). Anterior

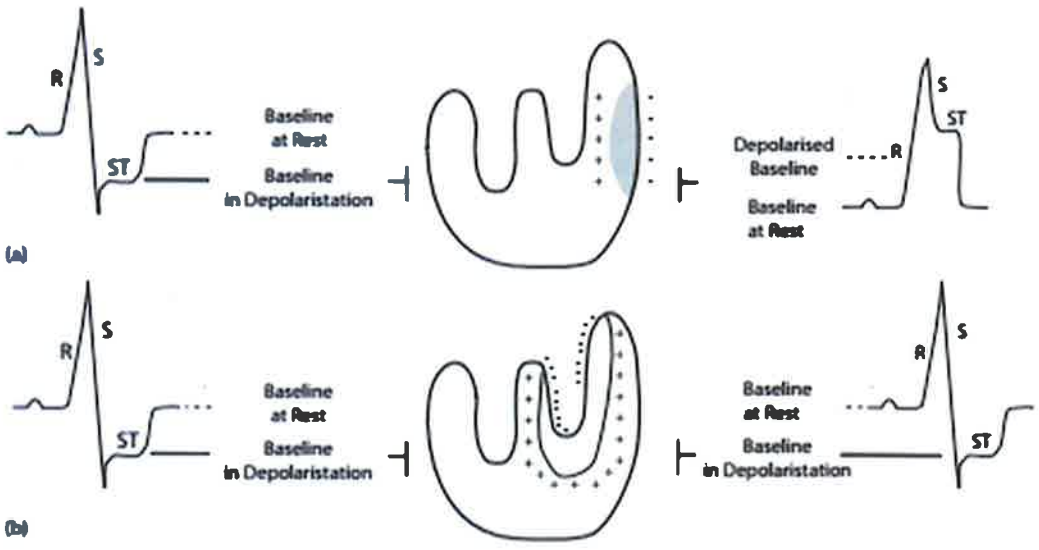

Figure 19.8 (a) Depiction of an ECG tracing of a myocardial infarct, where an electrode facing a region of infarction registers ST elevation, and an electrode away from the infarction registers ST depression ('reciprocal' change in ST level). (b) This shows an ECG tracing in myocardial ischaemia without infarction and is more widespread. An electrode over the ischaemic region and another one away from it both record ST segment depression.

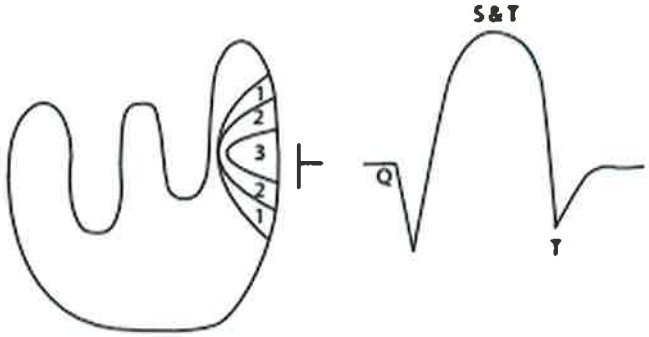

Figure 19.9 Appearance of QRS complex with a Q wave caused by an infarct. At left is the infarcted region in wall of ventricle (1 = ischaemia, 2 = injury, 3 = necrosis).

infarction usually involves the left anterior descending artery, inferior infarction usually involves the right coronary artery and posterior infarction involves the area usually supplied by the left circumflex artery. However, there are variations to this. If a diagonal branch of the left anterior descending artery occludes, T wave inversion is usually seen only in left anterior descending territory. A distal left anterior descending artery occlusion causing apical infarction usually involves T wave inversion in the apico-lateral leads. The proximal circumflex branches, when occluded, are often electrocardiographically silent on a standard ECG. To diagnose these, chest leads V7 to V12 are used. These are at the same level as lead V6 and 2 inches (5 cm) apart, coursing posteriorly around the chest.

Following is a summary of the major infarction patterns, and their association with the coronary blood supply and corresponding ECG changes.

Anterior infarction: Involves the left anterior descending artery and/or its major branches, with changes in leads V2-V4.

Septal infarction: Involves the proximal left anterior descending artery and its septal branches, with changes in leads V2-V3.

Apical infarction: Associated with rsR pattern in leads V4-V5.

High lateral infarction: Involves the obtuse marginal branch of circumflex, e.g. intermediate artery or a very proximal diagonal branch of the left anterior descending artery, with changes in leads I and aVL.

Lateral infarction: Involves the distal diagonal branches of the left anterior descending artery, distal circumflex or distal right coronary artery branches, with changes in leads V5-V6.

Inferior infarction: Usually involves the right coronary artery, but occasionally the circumflex (when dominant, in about 10% of cases) supplies the inferior surface (usually supplied by the posterior descending artery); changes in leads II, III and aVF.

Inferior infarction can show some variation in ECG pattern such that:

a. Sometime after the event Q waves may only be seen in III/aVF, whereas acutely they were present in all inferior leads; this indicates that the infarct is infero-basal, affecting the proximal portion of the inferior left ventricular wall and the interventricular septum.

b. ST depression may be noted at the same time in leads V1-V4, which is usually a mirror image of ST elevation in the posterior leads. This implies extension of the infarct upwards along the posterior wall due to:

   1. A right coronary artery with numerous postero-lateral branches extending up the posterior wall of the heart, or
   2. The circumflex artery is dominant, supplying the posterior descending branch (PDA).

There may be co-existing ST changes in V5-V6 due to involvement of the lateral wall, from a postero-lateral branch or a large PDA running to the apex.

Atrial infarction is unusual and occurs in the context of a large ventricular infarction. It is diagnosed by elevation of the PR segment, at times with a change in P wave morphology. In **acute pericarditis** there may be depression of the PR segment.

ABNORMAL RHYTHMS ASSOCIATED WITH MYOCARDIAL INFARCTION

Sinus node abnormalities

SINUS BRADYCARDIA

This may be due to:

a. Ischaemia of the SA node, causing local acidosis depressing nodal automaticity or elevation of adenosine causing a negative inotropic effect, or
b. Intense vagal stimulation which can be reversed by the administration of atropine. Bradycardia is up to three times more common in infero-posterior infarction, compared to anterior infarct. It is most likely to occur early in acute infarction (<3 hours) and is associated with a higher incidence of ventricular fibrillation, possibly as ventricular threshold is reduced with a bradycardia or because ventricular ectopics may be precipitated by long R-R intervals.

SINUS TACHYCARDIA

This is usually due to left ventricular failure but may be due to fever, coexistent pericarditis, anxiety, pulmonary embolus and other ailments. When sinus tachycardia is present there is usually a higher incidence of AV block 1° (first degree), 2° (second degree) and 3° (third degree) AV block.

SINUS ARREST

This is a rare occurrence (and is often due to S-A exit block) and occurs in inferior infarction with occlusion of the right coronary artery very proximally.

Atrial dysrrhythmias

ATRIAL ECTOPICS

These are very common in acute myocardial infarction and are due to either atrial ischaemia or atrial distension arising from heart failure with elevated LV diastolic pressure.

ATRIAL FIBRILLATION

This occurs in up to 10% of patients with acute myocardial infarction or with cardiac failure. It may be co-existent in patients with atrial disease or with damage from ischaemia, hypertension, pericarditis, chronic airways disease or other ailments. Atrial flutter and atrial tachycardia are very rare in acute myocardial infarction, when it may cause haemodynamic impairment due to rapid ventricular response. It may be treated with amiodarone (with intravenous loading), or judicious use of beta-blockers or electrical cardioversion.

Ventricular and nodal (junctional) rhythms

NODAL ESCAPE RHYTHM

These are inherently escape rhythms that occur when there are periods of sinus arrest, sinus bradycardia or abnormalities of impulse conduction with S-A block or A-V block. These rhythms are reasonably frequent in acute myocardial infarction, particularly inferior infarction as this is often associated with disorders of the S-A node causing sinus arrest, sinus bradycardia or S-A block. Nodal escape rhythm usually occurs at a rate of 50–60 beats per minute. Faster rates of AV nodal rhythm (which has automaticity) are the same as the causes of sinus tachycardia.

VENTRICULAR ECTOPIC BEATS

These occur in virtually all patients with acute myocardial infarction and they are of little prognostic significance, although it is considered that those arriving from the left ventricle are more likely to precipitate ventricular fibrillation. In the past there have been recommendations for pharmacological suppression of ventricular ectopy, but controlled trials have shown that doing so has not prevented ventricular fibrillation. Most coronary units now do not prophylactically suppress these ectopics.

VENTRICULAR TACHYCARDIA

Ventricular tachycardia consists of a series of three or more consecutive ventricular ectopic beats that occur at a rate faster than the underlying sinus rhythm. 3–10 escape beats are common in the first 36 hours after an acute myocardial infarction and are of little prognostic significance. However, rapid polymorphic ventricular tachycardia in the first 36 hours following infarction is of prognostic significance. Patients with a torsades de pointes

(TDP) pattern should have attention paid to the pro-arrhythmic effects of existing anti-arrhythmic therapy. TDP is also associated with the use of phenothiazines, tricyclic anti-depressants, erythromycin based anti-histamines, hypokalaemia and hypomagnesaemia. When polymorphic ventricular tachycardia is due solely to the myocardial infarction it usually responds well to anti-arrhythmic therapy.

IDIOVENTRICULAR TACHYCARDIA

This is frequently seen in acute myocardial infarction and often occurs following thrombolytic therapy, when re-perfusion of the coronary artery involved takes place. This rhythm rarely results in ventricular fibrillation and does not usually require treatment. However, because of loss of the atrial contraction, cardiac output can be significantly impaired by up to 20%, causing symptomatic hypotension and cardiac failure. Under these circumstances it should be treated with either anti-arrhythmic drugs or atropine.

VENTRICULAR FIBRILLATION

Prevention of death from dysrrhythmia is one of the major aims in acute coronary care. Ventricular fibrillation is often induced by electrolyte abnormalities and is more common the more extensive the myocardial infarction. The incidence decreases from 5% to ~7% of patients with acute myocardial infarction in the early 1970s to <2% in the late 1980s. This is partly due to use of beta-blockers in acute myocardial infarction, and more vigorous and effective treatment of cardiac failure and correction of electrolyte imbalances. Clinical trials have shown that ventricular premature beats are unreliable predictors of the development of ventricular fibrillation.

CONDUCTION DISTURBANCES IN ACUTE MYOCARDIAL INFARCTION

1° Atrio-ventricular block

This is a delay in conduction of the P wave from the SA node to the AV node and is evident as a prolonged PQ interval. It is seen in inferior infarction involving the AV node, where it is a benign finding, and less frequently in anterior infarction with septal involvement causing distal conduction delay. In this situation it indicates a far more extensive infarction.

2° Atrio-ventricular block

There are two main types:

Mobitz Type I, or Wenckebach block, is a progressive lengthening of the PQ interval with a subsequent "dropped" QRS beat. The mechanism is similar to that of 1° AV block. It is common to see a patient's rhythm progress from 1° AV block to Wenkebach block and then back, although Wenkebach block may be present for some time (>24 hours) before reverting to 1° block. The QRS complex usually remains narrow except in the presence of anterior infarction, when left anterior hemiblock or right bundle branch block may develop. In this situation the course may be more complicated.

Mobitz Type II block is evident as one or more "dropped" QRS beats following failure of a P wave to conduct to the AV node. It is due to ischaemia of the septal infra-nodal conduction pathway in anterior infarction and may be associated with a narrow or wide QRS complex. In the latter, due to bundle branch block, the prognosis is poor. Mobitz Type II may progress to 3° AV block and require temporary pacemaker insertion.

3° Atrio-ventricular block

This is complete conduction failure of propagation of impulse from atrium to ventricle and results in ventricular standstill, evident as a prolonged flat ECG signal. It may occur in inferior or anterior myocardial infarction. The mechanisms differ and have different prognostic implications. In inferior infarction it is the result of necrosis or ischaemia of the AV node (with or without vagal stimulation) and may progress from 1° AV block to 2° AV block (Mobitz Type I) to 3° AV block. Usually it is benign and haemodynamics are maintained, although a temporary pacemaker may at times be required. In anterior myocardial infarction the prognosis is much poorer due to the extensive nature of the infarct. Rather than having a gradual onset as in inferior infarction it may be of abrupt onset with major haemodynamic impairment. A temporary pacemaker is usually required to maintain haemodynamics and often a permanent pacemaker is required if the patient survives. Due to the large amount of left ventricular impairment associated with an anterior myocardial infarction and heart block,

an implantable defibrillator may be indicated to prevent subsequent death.

Hemiblock

Left anterior hemiblock may occur in antero-septal infarction and is of little significance unless right bundle branch block also develops. In this situation (bi-fascicular block) high grade AV block (Mobitz Type II or 3° AV block) may develop requiring a temporary pacemaker implantation.

Bundle branch block

This is failure of either the right or left bundle branch to conduct and is seen as a widened QRS complex. When occurring in the context of acute myocardial infarction it usually indicates extensive infarction with significant haemodynamic impairment.

PSEUDO-INFARCT PATTERNS ON STANDARD ECG

These are cardiac conditions where the standard resting ECG may be confused with an ischaemic pattern. Following is a brief description of the various types encountered.

Wolff–Parkinson–White syndrome

The delta wave can mimic Q waves in the inferior, lateral and posterior leads, mimicking infarction patterns.

Hypertrophic cardiomyopathy

This can occur with or without obstruction of the left ventricular outflow tract and is associated not only with cardiac failure but also with ventricular dysrrhythmias, and at times, atrial dysrhythmias. The resting ECG can show Q waves due to hypertrophy and also ST/T wave changes mimicking hyperacute infarction (ST elevation), old infarction (Q waves) or ischaemia (ST/T changes).

Chronic obstructive airways disease and right ventricular hypertrophy

These conditions may be associated with weak R wave development in the inferior and early precordial leads ('poor R wave progression'), mimicking old infarction.

Pulmonary emboli

This may be associated with T wave changes due to right ventricular strain suggesting ischaemia. At times, Q waves may develop in lead III, suggesting old infarction. If chronic recurrent pulmonary emboli occur, then right ventricular hypertrophy with clockwise precordial rotation and slow R wave development in V1–V4 may result, suggesting old antero-septal myocardial infarction.

Thoracic cage deformity

This condition, especially *pectus excavatum*, which causes flattening or compression of the heart with movement to the left, causes slow R wave development in leads V1-V4, suggesting anterior infarction.

Miscellaneous

A list of additional conditions that demonstrate pseudo-Infarct patterns on standard ECG are listed in Table 19.1, and Figure 19.10 shows some examples of pseudo-infarct patterns on ECG.

Figure 19.10a shows a pseudoinfarct pattern in Wolff–Parkinson–White syndrome. There is a Q wave in the inferior leads which is produced by pre-excitation of the posteroseptal left ventricle.

Figure 19.10b shows the ECG pattern in Brugada syndrome: This shows ST elevation in the precordial leads V_1 and V_2, which can be confused with an anteroseptal myocardial infarction.

Figure 19.10c shows the ECG pattern in hyperkalaemia: There are extensive ST segment changes and underlying heart block due to this metabolic abnormality.

The ECG in Figure 19.10d is from a patient with Q waves in leads III and aVF consistent with an old inferior infarction, Q waves in leads I, V_5 and a small RSR in V_6. She has had a previous antero-apicoseptal infarction and the RSR in V_6 indicates apical aneurysm. There are ST/T wave changes in leads I, aVL, V_5 and V_6 consistent with lateral ischaemia.

The ECG in Figure 19.10e shows this patient has atrial fibrillation and right bundle branch block but there is a small Q wave in leads V_1–V_3 as well as a Q wave in III and aVF. This patient has had

Table 19.1 Conditions that present pseudo-infarct patterns on the electrocardiogram

Cardiomyopathy	• Dilated • Hypertrophic • Takotsubo
Infiltrative cardiac disease	• Amyloid • Sarcoid • Haemochromatosis • Scleroderma • Primary and secondary cardiac tumours
Pericardial disease	• Acute pericarditis • Infective • Trauma • Post infarction
Myocarditis	
Myocardial injury/contusion	
Congenital heart disease	• Fallot's tetralogy • Primum ASD • Congenital coronary anomalies
Left ventricular hypertrophy	
Hyperkalaemia	
Cerebrovascular accident/Subarachnoid haemorrhage	
Kawasaki disease	
Pulmonary disease	• Emphysema • Spontaneous pneumothorax • Pulmonary embolus • Left sided thoracic cage deformity • Marked pectus excavatum
Neurological diseases	• Muscular dystrophy • Friedreich's ataxia
Cardiac rhythm/electrical disturbances	• Left bundle branch block • Stokes Adams with giant T wage inversion • Post tachycardia ST/T wave inversion • Pacemaker-induced T wave changes

previous inferior infarction and in the presence of right bundle branch block, also shows a previous septal infarction.

The ECG in Figure 19.10f was taken from a patient who presented to hospital with severe central pre-cordial chest pain, and was found to have normal coronary arteries. A subsequent ventilation quellum (VQ) scan showed massive pulmonary embolus. Her T wave changes in leads I, aVL, V_1–V_6 could easily be mistaken for myocardial ischaemia.

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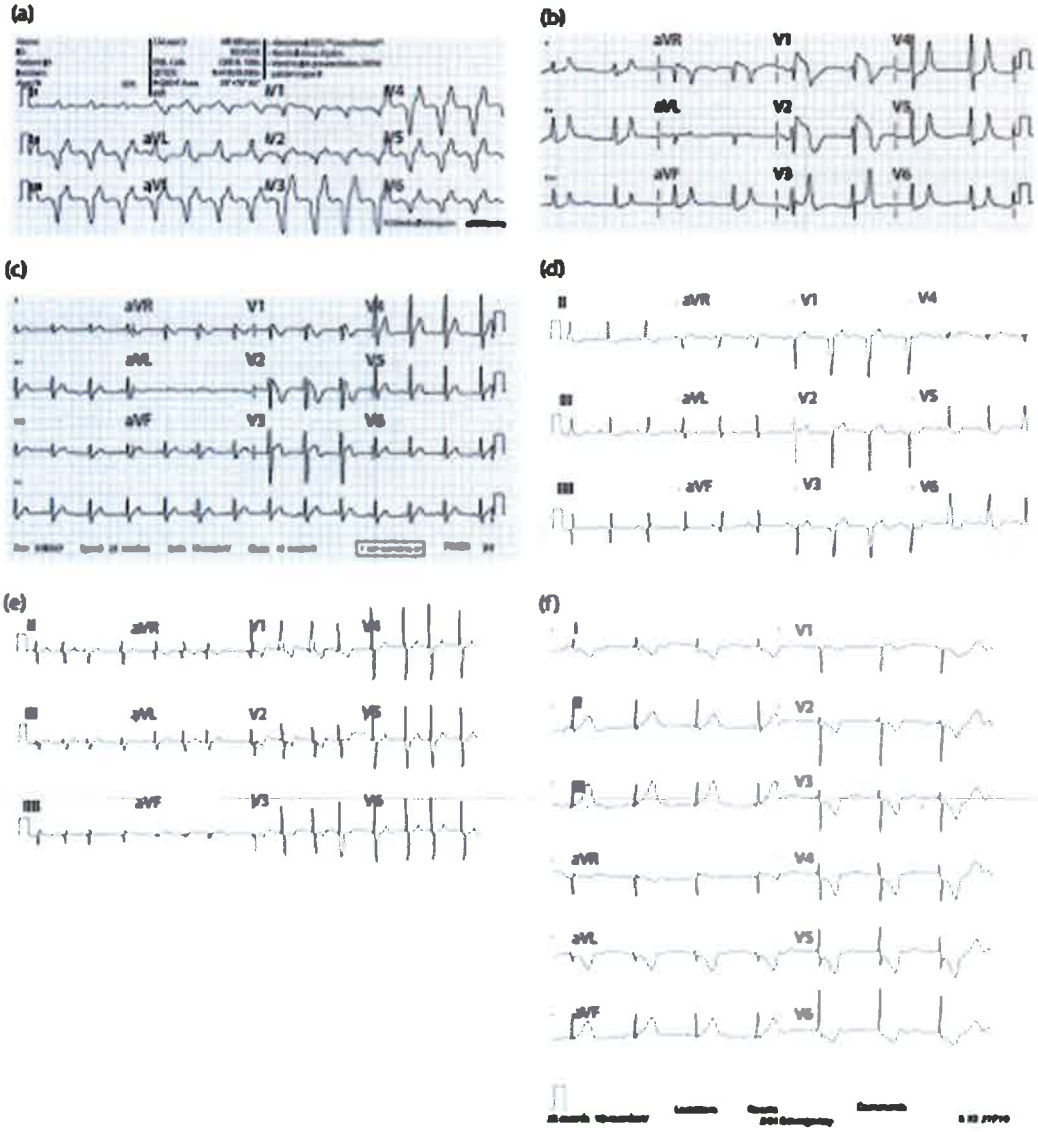

Figure 19.10 (a–f) Compilation of sample ECG tracings showing various pseudo-infarct patterns. See text for description.

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