MAKING SENSE

MAKING SENSE of the ECG

A hands-on guide

Fifth edition

Andrew R Houghton

CRC Press
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Preface to the fifth edition

The primary aim of this fifth edition of *Making Sense of the ECG* remains the same as all its predecessors – to provide the reader with a comprehensive yet readable introduction to electrocardiogram (ECG) interpretation, supplemented by clinical information about how to act upon your findings.

Each chapter has been revised and updated, incorporating current guidelines and research. The text has been clarified and/or expanded where necessary, new ECGs have been included, and the references and suggestions for further reading at the end of each chapter have been updated throughout.

A new chapter has been added on ECG interpretation in athletes, with reference to the latest guidelines in this field. New material has been included on Brugada syndrome and on ECG findings in arrhythmogenic right ventricular cardiomyopathy. A glossary of ECG terminology has also been added as an appendix.

Once again, I am grateful to everyone who has taken the time to comment on the text and provide ECGs from their collections. Finally, I would like to thank all the staff at CRC Press who have contributed to the success of the Making Sense series of books.

Andrew R Houghton
2019

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Chapter 1

Anatomy and physiology

The heart is a hollow muscular organ that pumps blood around the body. With each beat, it pumps, at rest, about 70 millilitres of blood and considerably more during exercise. Over a 70-year life span and at a rate of around 70 beats per minute, the heart will beat over 2.5 billion times.

The heart consists of four main chambers (left and right atria, and left and right ventricles) and four valves (aortic, mitral, pulmonary and tricuspid). Venous blood returns to the right atrium via the superior and inferior vena cavae, and leaves the right ventricle for the lungs via the pulmonary artery. Oxygenated blood from the lungs returns to the left atrium via the four pulmonary veins, and leaves the left ventricle via the aorta (Figure 1.1).

The heart is made up of highly specialized cardiac muscle comprising myocardial cells (**myocytes**), which differs markedly from skeletal muscle because heart muscle:

- Is under the control of the autonomic nervous system
- Contracts in a repetitive and rhythmic manner

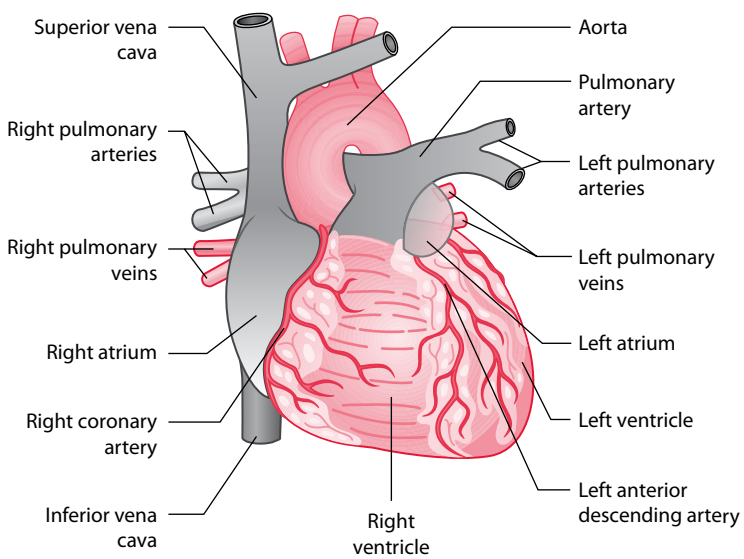

Figure 1.1 Cardiac anatomy.

Key point: • The heart and major vessels.

- Has a large number of mitochondria which make the myocytes resistant to fatigue
- Cannot function adequately in anaerobic (ischaemic) conditions

CARDIAC ACTIVATION

Myocytes are essentially contractile but are capable of generating and transmitting electrical activity. Myocytes are interconnected by cytoplasmic bridges or syncytia, so once one myocyte cell membrane is activated (depolarized), a wave of depolarization spreads rapidly to adjacent cells.

Myocardial cells are capable of being:

- *Pacemaker cells*: These are found primarily in the sinoatrial (SA) node and produce a spontaneous electrical discharge.
- *Conducting cells*: These are found in:
  - The atrioventricular (AV) node
  - The bundle of His and bundle branches
  - The Purkinje fibres
- *Contractile cells*: These form the main cell type in the atria and ventricles.

All myocytes are self-excitable with their own intrinsic contractile rhythm. Cardiac cells in the SA node located high up in the right atrium generate action potentials or impulses at a rate of about 60–100 per minute, a slightly faster rate than cells elsewhere such as the AV node (typically 40–60 per minute) or the ventricular conducting system (30–40 per minute), so the SA node becomes the heart pacemaker, dictating the rate and timing of action potentials that trigger cardiac contraction, overriding the potential of other cells to generate impulses. However, should the SA node fail or an impulse not reach the ventricles, cardiac contraction may be initiated by these secondary sites ('escape rhythms').

THE CARDIAC ACTION POTENTIAL

The process of triggering cardiac cells into function is called *cardiac excitation–contraction coupling*. Cells remain in a resting state until activated by changes in voltage due to the complex movement of sodium, potassium and calcium across the cell membrane (Figure 1.2); these are similar to changes which occur in nerve cells.

Phase 4: At rest, there is little spontaneous depolarization as the Na^+/K^+/ATPase pump maintains a negative stable resting membrane potential of about –90 mV. Some cardiac cells display automaticity or spontaneous regular action potentials, which generates action potentials in adjacent cells linked by cytoplasmic bridges or syncytia, so once one myocyte cell membrane is activated (depolarized), a wave of excitation spreads rapidly to adjacent cells; the SA node, whose cells are relatively permeable to sodium resulting in a less negative resting potential of about –55 mV, is usually the source of spontaneous action potentials.

Phase 0: There is rapid opening of sodium channels with movement of sodium into the cell, the resulting electrochemical gradient leading to a positive resting membrane potential.

Phase 1: When membrane potential is at its most positive, the electrochemical gradient causes potassium outflow and closure of sodium channels.

Phase 2: A plateau phase follows, with membrane potential maintained by calcium influx; membrane potential falls towards the resting state as calcium channels gradually become inactive and potassium channels gradually open.

Phase 3: Potassium channels fully open, and the cell becomes repolarized.

Phase 4: Calcium, sodium and potassium are gradually restored to resting levels by their respective ATPase-dependent pumps.

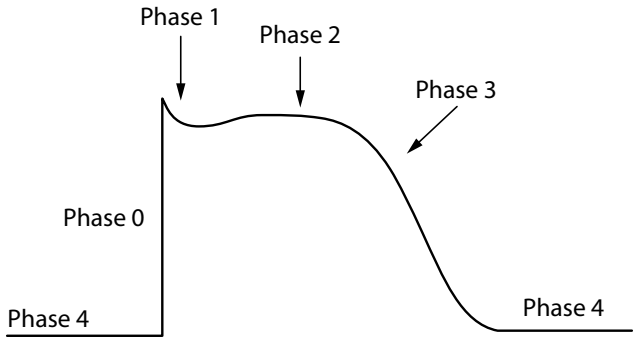

Figure 1.2 The cardiac action potential.

Key point: • The different phases of the cardiac action potential.

The SA node is susceptible to influence from:

- The parasympathetic nervous system via the vagus nerve, which slows heart rate
- The sympathetic nervous system via spinal nerves from T1 to T4 – these increase heart rate and can increase the force of contraction
- Serum concentration of electrolytes, e.g. hyperkalaemia, which can cause severe bradycardia (note that hypokalaemia can cause tachycardia)
- Hypoxia, which can cause severe bradycardia

Cardiac drugs can also affect cardiac rate, some acting through the SA node, others through the AV node or directly on ventricular myocytes:

- Negative chronotropes reduce cardiac rate
  - Such as beta blockers and calcium channel blockers
- Positive chronotropes increase cardiac rate
  - Such as dopamine and dobutamine
- Negative inotropes decrease force of contraction
  - Such as beta blockers, calcium channel blockers and some anti-arrhythmic drugs such as flecainide and disopyramide
- Positive inotropes increase force of contraction
  - Such as dopamine and dobutamine

THE CARDIAC CONDUCTION SYSTEM

Each normal heartbeat begins with the discharge ('depolarization') of the SA node. The impulse then spreads from the SA node to depolarize the atria. After flowing through the atria, the electrical impulse reaches the AV node, low in the right atrium.

Once the impulse has traversed the AV node, it enters the bundle of His which then divides into left and right bundle branches as it passes into the interventricular septum (Figure 1.3). The right bundle branch conducts the wave of depolarization to the right ventricle, whereas the left bundle branch divides into anterior and posterior fascicles that conduct the wave to the left ventricle.

The conducting pathways end by dividing into Purkinje fibres that distribute the wave of depolarization rapidly throughout both ventricles. Normal depolarization of the ventricles is therefore usually very fast, occurring in less than 0.12 s.

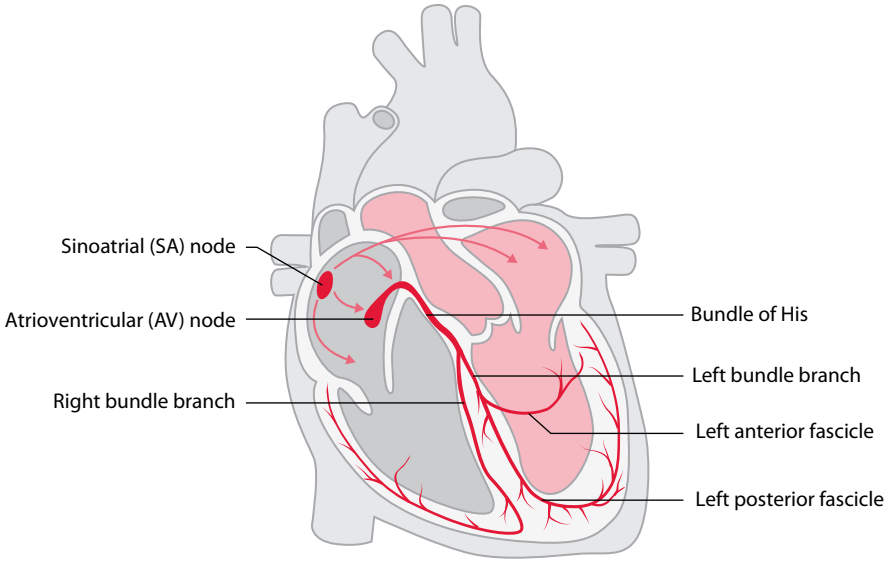

Figure 1.3 The cardiac conduction system.

THE CARDIAC CYCLE

The events that occur during each heartbeat are termed the cardiac cycle, commonly represented in diagrammatic form (Figure 1.4). The cardiac cycle has four phases:

1. Isovolumic contraction
2. Ventricular ejection
3. Isovolumic relaxation
4. Ventricular filling

These phases apply to both the left and right heart, but we will focus on the left heart here for clarity. Phases 1–2 correspond with ventricular systole and phases 3–4 with ventricular diastole.

Isovolumic contraction begins with closure of the mitral valve, caused by the rising left ventricle (LV) pressure at the start of ventricular systole (which coincides with the QRS complex on the ECG). After the mitral valve has closed, pressure within the LV continues to rise but the LV volume remains constant (hence 'isovolumic') until the point when the aortic valve opens.

Ventricular ejection commences when the aortic valve opens and blood is ejected from the LV into the aorta.

Isovolumic relaxation commences with closure of the aortic valve. Pressure within the LV falls during this phase (but volume remains constant), until the LV pressure falls below left atrium (LA) pressure. At this point, the pressure difference between LA and LV causes the mitral valve to open and isovolumic relaxation ends.

Ventricular filling begins as the mitral valve opens and blood flows into the LV from the LA. This phase ends when the mitral valve closes at the start of ventricular systole. Towards the end of the ventricular filling phase, atrial systole (contraction) occurs, coinciding with the P wave on the ECG, and this augments ventricular filling.

As shown in Figure 1.4, the pressures within the cardiac chambers vary throughout the cardiac cycle. A pressure difference between two chambers causes the valve between them to open or close. For example, when LA pressure exceeds LV pressure the mitral valve opens, and when LV pressure exceeds LA pressure the mitral valve closes.

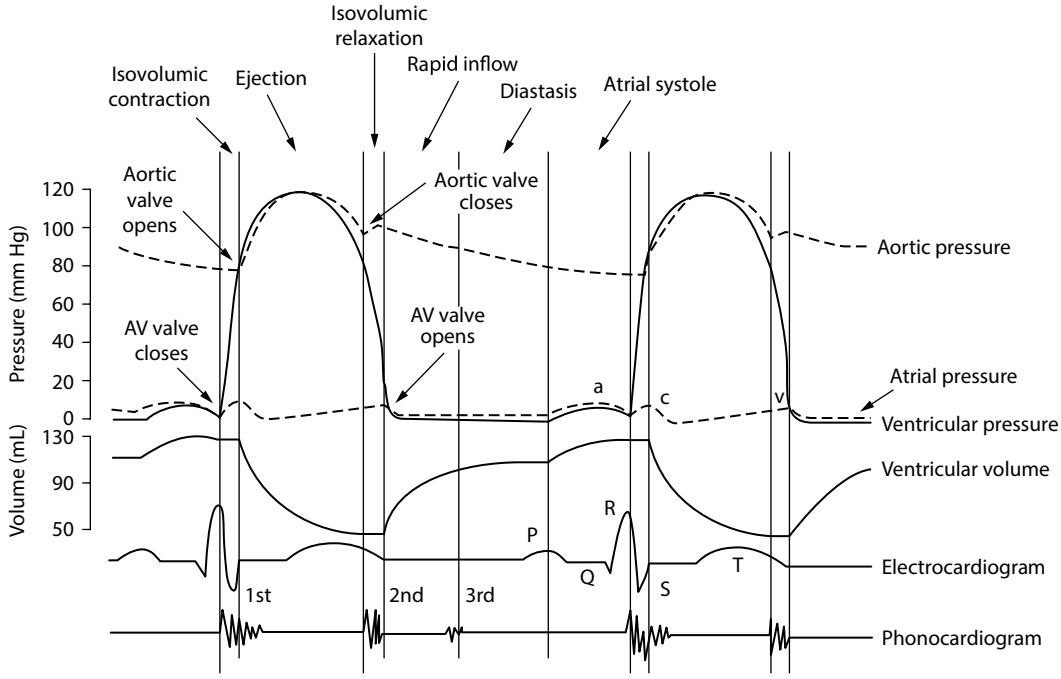

Figure 1.4 The cardiac cycle.

Key point: • The different phases of the cardiac cycle.

Further reading

Amin AS, Tan HL, Wilde AAM. Cardiac ion channels in health and disease. *Heart Rhythm* 2010; 7: 117–126.

Cabrera JA, Sánchez-Quintana D. Cardiac anatomy: What the electrophysiologist needs to know. *Heart* 2013; 99: 417–431.

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Chapter 2

PQRST: Where the waves come from

The electrocardiogram (ECG) is one of the most widely used and useful investigations in contemporary medicine. It is essential for the identification of disorders of the cardiac rhythm, extremely useful for the diagnosis of abnormalities of the heart (such as myocardial infarction) and a helpful clue to the presence of generalized disorders that affect the rest of the body too (such as electrolyte disturbances).

Each chapter in this book considers a specific feature of the ECG in turn. We begin, however, with an overview of the ECG in which the following points are explained:

- What does the ECG actually record?
- How does the ECG 'look' at the heart?
- Where do each of the waves come from?

It is recommended you take some time to read through this chapter before trying to interpret ECG abnormalities.

WHAT DOES THE ECG ACTUALLY RECORD?

ECG machines record the electrical activity of the heart. They also pick up the activity of other muscles, such as skeletal muscle, but are designed to filter this out as much as possible. Encouraging patients to relax during an ECG recording helps to obtain a clear trace (Figure 2.1).

By convention, the main waves on the ECG are given the names P, Q, R, S, T and U (Figure 2.2). Each wave represents depolarization ('electrical discharging') or repolarization ('electrical recharging') of a certain region of the heart. This is discussed in more detail in the rest of this chapter.

The voltage changes detected by ECG machines are very small, being of the order of millivolts. The size of each wave corresponds to the amount of voltage generated by the event that created it: the greater the voltage, the larger the wave (Figure 2.3).

The ECG also allows you to calculate how long an event lasted. The speed at which ECG paper moves through the machine is standardized at a constant rate of 25 mm/s, so each small (1 mm) square on the ECG represents 0.04 s, and each large (5 mm) square represents 0.2 s. This means that by measuring the width of a wave, you can calculate the duration of the event causing it. For example, the P waves in Figure 2.4 are 2.5 mm wide, so we can calculate that atrial depolarization lasted for 2.5×0.04 s, or 0.10 s.

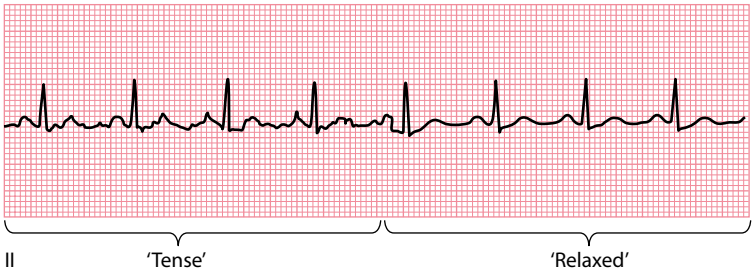

Figure 2.1 Skeletal muscle artefact.

Key points:
- An ECG from a relaxed patient is much easier to interpret.
- Electrical interference (irregular baseline) is present when the patient is tense, but the recording is much clearer when the patient relaxes.

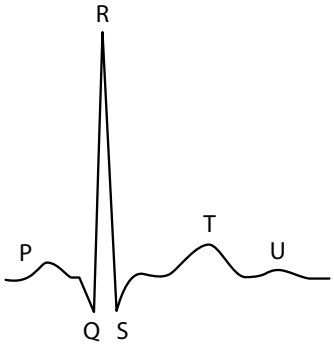

Figure 2.2 Standard nomenclature of the ECG recording.

Key point:
- The waves are called P, Q, R, S, T and U.

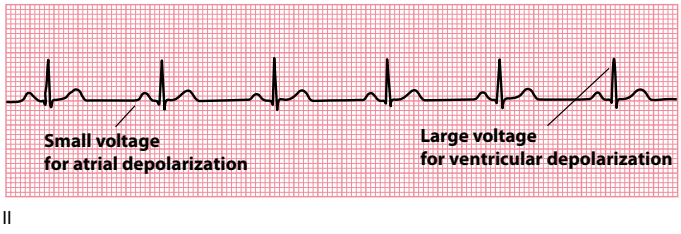

Figure 2.3 The size of a wave reflects the voltage that caused it.

Key point:
- P waves are small (atrial depolarization generates little voltage); QRS complexes are larger (ventricular depolarization generates a higher voltage).

HOW DOES THE ECG 'LOOK' AT THE HEART?

To make sense of the ECG, one of the most important concepts to understand is that of the 'lead'. This is a term you will often see, and it does *not* refer to the wires that connect the patient to the ECG machine (which we will always refer to as 'electrodes' to avoid confusion).

In short, 'leads' are different *viewpoints* of the heart's electrical activity. An ECG machine uses the information it collects via its four limb and six chest electrodes to compile a comprehensive picture of the electrical activity in the heart as observed from 12 different viewpoints, and this set of 12 views or leads gives the 12-lead ECG its name.

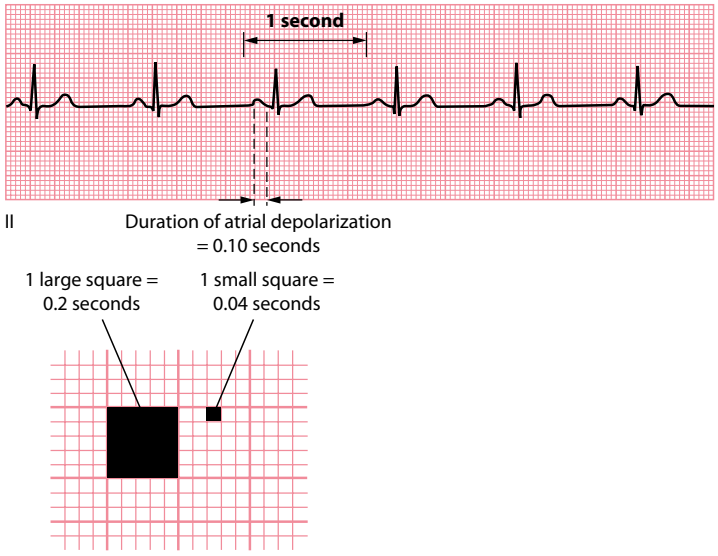

Figure 2.4 The width of a wave reflects an event's duration.

Key points:
- The P waves are 2.5 mm wide.
- At a paper speed of 25 mm/s, atrial depolarization therefore took 0.10 s.

Each lead is given a name (I, II, III, aVR, aVL, aVF, V_1, V_2, V_3, V_4, V_5 and V_6) and its position on a 12-lead ECG is usually standardized to make pattern recognition easier.

So what viewpoint does each lead have of the heart? Information from the four limb electrodes is used by the ECG machine to create the six limb leads (I, II, III, aVR, aVL and aVF). How the machine does this is discussed in Chapter 3. For now, you just need to know that each limb lead 'looks' at the heart from the side (the frontal or 'coronal' plane), and the view that each lead has of the heart in this plane depends on the lead in question (Figure 2.5).

ECG LEAD NOMENCLATURE

There are several ways of categorizing the 12 ECG leads. They are often referred to as limb leads (I, II, III, aVR, aVL, aVF) and chest leads (V_1, V_2, V_3, V_4, V_5, V_6). They can also be divided into bipolar leads (I, II, III) or unipolar leads (aVR, aVL, aVF, V_1, V_2, V_3, V_4, V_5, V_6).

Bipolar leads are generated by measuring the voltage between two electrodes – for example, lead I measures the voltage between the left arm electrode and the right arm electrode. Unipolar leads measure the voltage between a single positive electrode and a 'central' point of reference generated from the other electrodes – for example, lead aVR uses the right arm electrode as the positive pole and a combination of left arm and left leg electrodes as the negative pole.

As you can see from Figure 2.5, lead aVR looks at the heart from the approximate viewpoint of the patient's right shoulder, whereas leads I and aVL have a left lateral view of the heart, and leads II, III and aVF look at the inferior surface of the heart.

The view that each limb lead has of the heart is more formally represented in the hexaxial diagram (Figure 2.6), which shows the angle that each limb lead has in relation to the heart. This diagram is invaluable when performing axis calculations, and how to use the diagram is described during the discussion of the cardiac axis in Chapter 10.

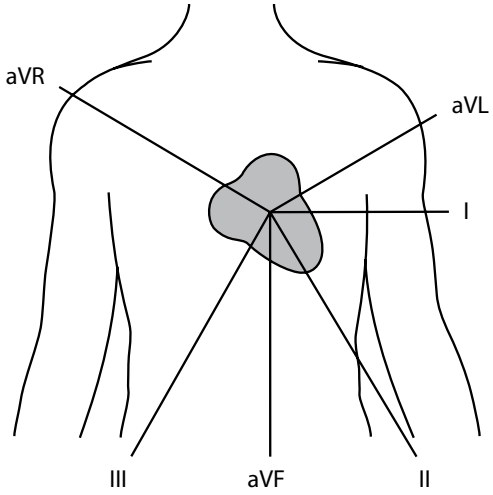

Figure 2.5 The viewpoint each limb lead has of the heart.

Key point:
- The limb leads 'look' at the heart in the frontal (or 'coronal') plane, and each limb lead looks at the heart from a different angle.

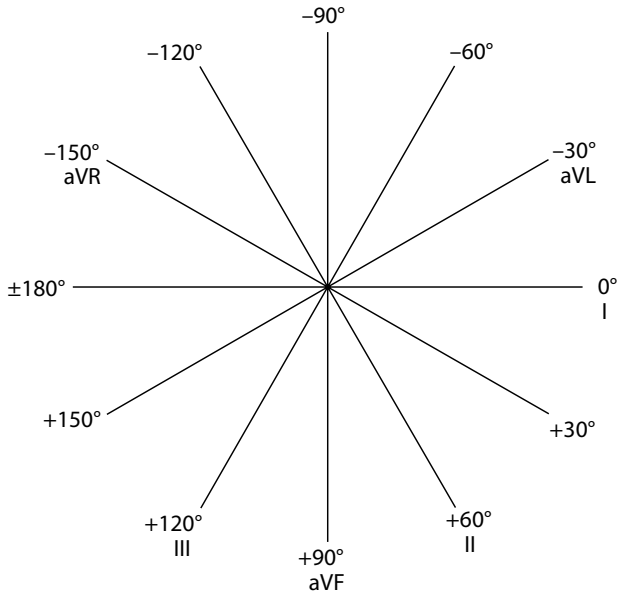

Figure 2.6 Hexaxial diagram.

Key point:
- This shows the angle of view that each limb lead has of the heart.

The six chest leads (V_1–V_6) look at the heart in a horizontal ('transverse') plane from the front and around the side of the chest (Figure 2.7). The region of myocardium surveyed by each lead therefore varies according to its vantage point – leads V_1–V_4 have an anterior view, for example, whereas leads V_5–V_6 have a lateral view.

Once you know the view each lead has of the heart, you can tell whether the electrical impulses in the heart are flowing towards that lead or away from it. This is simple to work out, because electrical current flowing towards a lead produces an upward (positive) deflection on the ECG, whereas current flowing away causes a downward (negative) deflection (Figure 2.8).

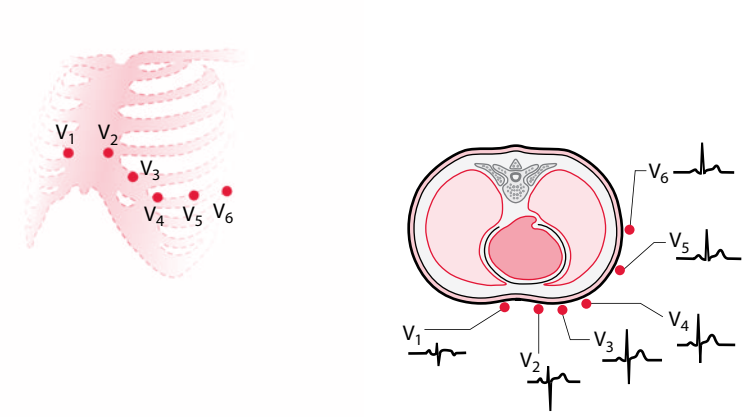

Figure 2.7 The viewpoint each chest lead has of the heart.

Key point:
- Each chest lead looks at the heart from a different viewpoint in the horizontal ('transverse') plane.

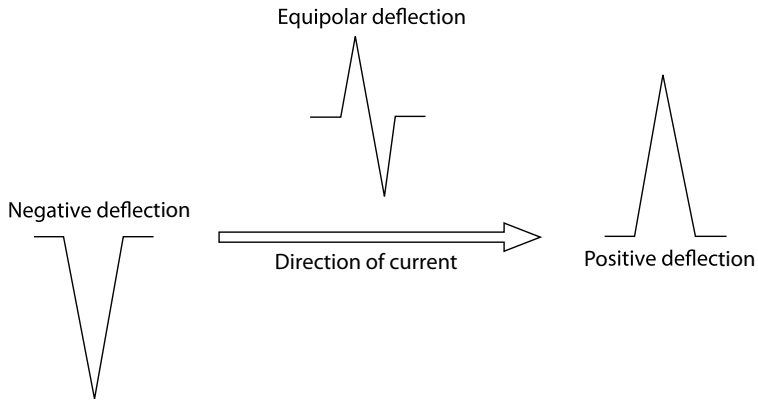

Figure 2.8 The direction of an ECG deflection depends on the direction of the current.

Key point:
- Flow towards a lead produces a positive deflection, flow away from a lead produces a negative deflection and flow perpendicular to a lead produces a positive then a negative (equipolar or isoelectric) deflection.

The origin of each wave will be discussed shortly, but just as an example consider the P wave, which represents atrial depolarization. The P wave is positive in lead II because atrial depolarization flows towards that lead, but it is negative in lead aVR because this lead looks at the atria from the opposite direction (Figure 2.9).

In addition to working out the direction of flow of electrical current, knowing the viewpoint of each lead allows you to determine which regions of the heart are affected by, for example, a myocardial infarction. Infarction of the inferior surface will produce changes in the leads looking at that region, namely leads II, III and aVF (Figure 2.10). An anterior infarction produces changes mainly in leads V_1–V_4 (Figure 2.11).

WHERE DO EACH OF THE WAVES COME FROM?

As we saw in Chapter 1, each normal heartbeat begins with the discharge ('depolarization') of the sinoatrial (SA) node, high up in the right atrium. This is a spontaneous event, occurring 60–100 times every minute. Depolarization of the SA node does not cause any noticeable wave on the standard ECG (although it can be seen on specialized intracardiac recordings). The first detectable wave appears when the impulse spreads from the SA node to depolarize the atria (Figure 2.12). This produces the **P wave**.

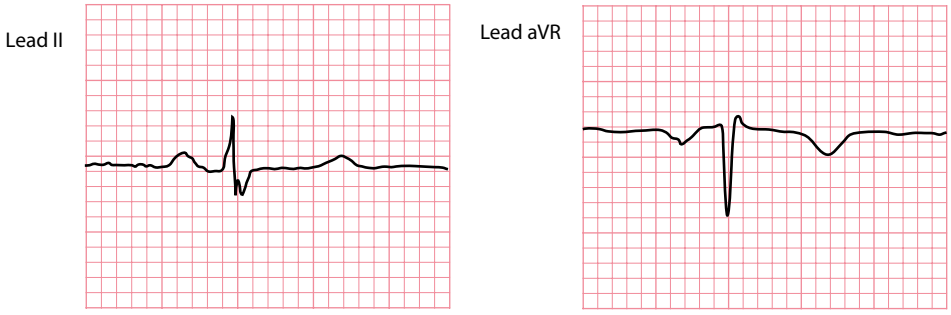

Figure 2.9 The orientation of the P wave depends on the lead.

Key point:
- P waves are normally upright in lead II and inverted in lead aVR.

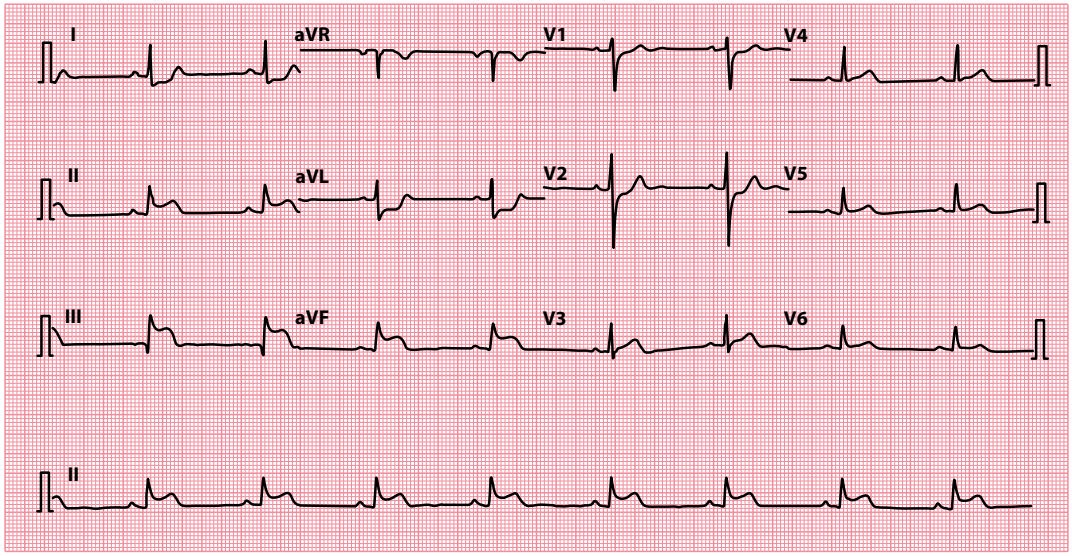

Figure 2.10 An inferior myocardial infarction produces changes in the inferior leads.

Key points:
- Leads II, III and aVF look at the inferior surface of the heart.
- ST segment elevation is present in these leads (acute inferior myocardial infarction).
- There is also reciprocal ST segment depression in leads I and aVL.

The atria contain relatively little muscle, so the voltage generated by atrial depolarization is relatively small. From the viewpoint of most leads, the electricity appears to flow *towards* them, and so the P wave will be a positive (upward) deflection. The exception is lead aVR, where the electricity appears to flow *away*, and so the P wave is negative in that lead (see Figure 2.9).

After flowing through the atria, the electrical impulse reaches the atrioventricular (AV) node, low in the right atrium. Activation of the AV node does not produce an obvious wave on the ECG, but it does contribute to the time interval between the P wave and the subsequent Q or R wave. It does this by delaying conduction, and in doing so acts as a safety mechanism, preventing rapid atrial impulses (for instance during atrial flutter or fibrillation) from spreading to the ventricles at the same rate.

The time taken for the depolarization wave to pass from its origin in the SA node, across the atria, and through the AV node into ventricular muscle is called the **PR interval**. This is measured from the beginning of the P wave to the beginning of the R wave and is normally between 0.12 s and 0.20 s, or 3 to 5 small squares on the ECG paper (Figure 2.13).

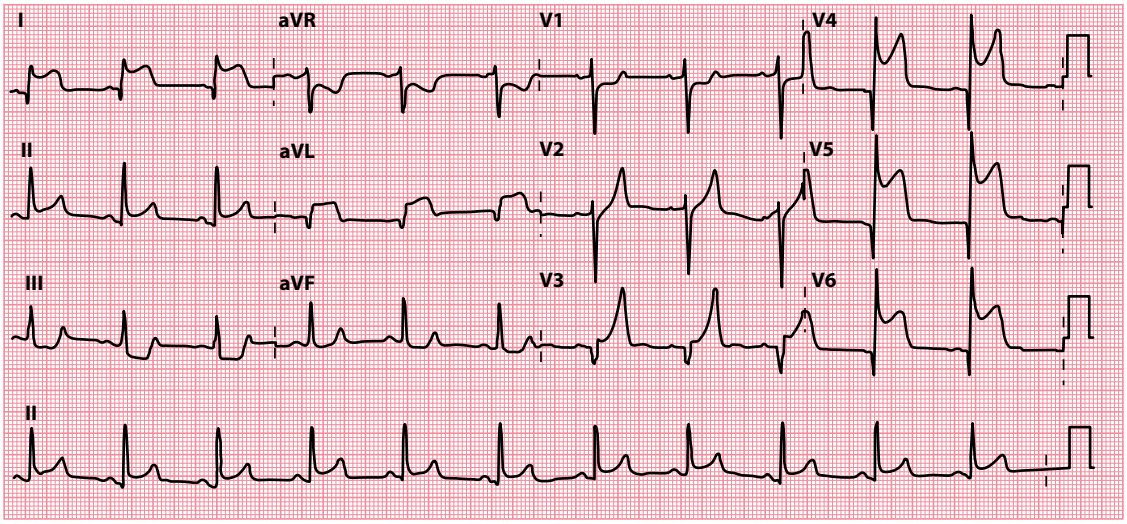

Figure 2.11 An anterolateral myocardial infarction produces changes in the anterolateral leads.

Key points:
- Leads V_3–V_6, I and aVL look at the anterolateral surface of the heart.
- ST segment elevation is present in these leads.

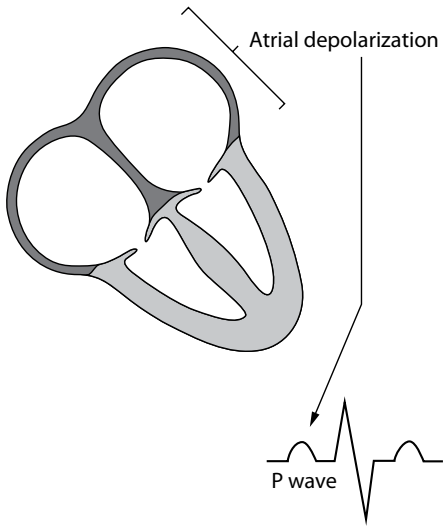

Figure 2.12 The P wave.

Key point:
- The P wave corresponds to atrial depolarization.

Once the impulse has traversed the AV node, it enters the bundle of His which then divides into left and right bundle branches as it passes into the interventricular septum (Figure 2.14). Current normally flows between the bundle branches in the interventricular septum, from left to right, and this is responsible for the first deflection of the **QRS complex**. Whether this is a downward deflection or an upward deflection depends on which side of the septum a lead is 'looking' from (Figure 2.15).

By convention, if the first deflection of the QRS complex is downward, it is called a **Q wave**. The first upward deflection is called an **R wave**, whether or not it follows a Q wave. A downward deflection after an R wave is called an **S wave**. Hence, a variety of complexes is possible (Figure 2.16).