4

READING AND INTERPRETING THE ELECTROCARDIOGRAM

Electrodes placed on the body's surface can detect electrical activity, which occurs in the heart. The recording of these electrical events comprises an electrocardiogram. Comparison of the information obtained from electrodes, placed in different positions on the body, enables electrical activity to be monitored and so the performance of different areas of cardiac tissue. This chapter commences with a review of the cardiovascular system and electrophysiology. This is followed by an examination of the conduction system of the heart and the electrocardiogram. The identification of normal and abnormal heart rhythms is then described.

APPLIED ANATOMY AND PHYSIOLOGY

Circulatory system

Blood flows through the body in a closed network called the circulatory system. It is pumped from the left atrium of the heart into the left ventricle, ejected into the aorta and then into other arteries. Arteries carry the blood

away from the heart. Each artery branches about 15–20 times, becoming smaller and smaller. These small arteries, or arterioles, which lead into a network of minute capillaries. Oxygen and nutrients diffuse through the thin walls of these vessels into the tissues of the body. The capillaries eventually form venules, which lead onto veins. Veins take the blood back to the right side of the heart, entering the right atrium via the vena cavae. The right ventricle then pumps this blood to the lungs where it becomes oxygenated, and then it returns to the left side of the heart.

The heart, like other organs, also requires an adequate supply of oxygen and nutrients. These are supplied from arterial branches that arise from the ascending aorta. The flow of blood that supplies the heart tissue itself is called the **coronary circulation.** The heart pumps about 380 litres of blood to its own muscle tissue every day.

Heart

The heart is positioned in the thorax underneath the sternum of the rib cage. It has four chambers and is made up primarily of muscle (**Figure 4.1**). The two atria receive blood from the veins and the two ventricles pump blood out into the arteries. The wall of the heart consists of three layers:

1 *Fibrous pericardium* – surrounds the heart giving it support and anchoring it to the diaphragm.
2 *Myocardium* – consists of muscle cells and forms the bulk of the wall of each chamber. This is much thicker in the wall of the left ventricle than the right and, therefore, enables it to develop greater pressure when it contracts. Specialized cells in this layer behave very similarly to nerve cells, generating and transmitting impulses. These cells include the sino-atrial node cells, atrio-ventricular node cells and Purkinje fibres.
3 *Endocardium* – consists of connective tissue, blood vessels and nerves, and forms the innermost layer.

(The septum, the wall separating the right and left sides of the heart, prevents blood passing from one side to the other. The atria and ventricles are separated by dense fibrous tissue.)

Valves

The mitral and tricuspid valves are the valves between the atria and ventricles on the left and right sides of the heart respectively. During ventricular contraction the valve cusps are forced together, closing off the opening and preventing blood from re-entering the atria. The aortic and pulmonary valves

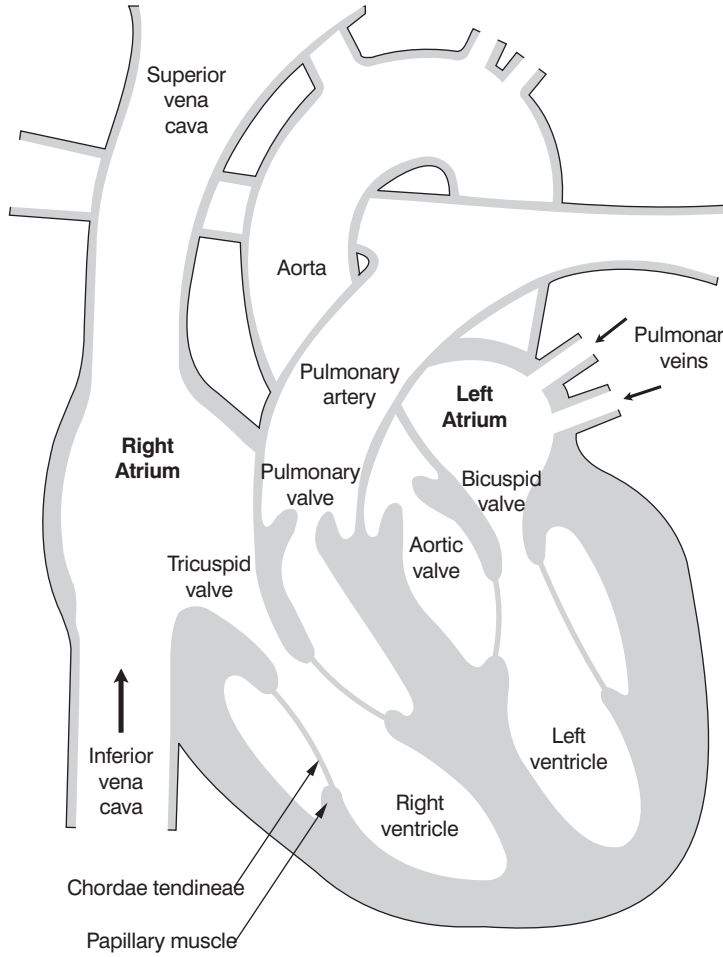

Figure 4.1 – The heart, showing chambers, valves and major vessels

are at the exits of the left and right ventricles. They are called semilunar valves because of their shape. The openings between the veins and the atria are without valves. Therefore, some blood can be forced backwards into the veins and forward into the ventricles when the atria contract.

Cardiac cycle

A single cardiac cycle is the time between the start of one heartbeat and the beginning of the next. It, therefore, includes alternating periods of contraction and relaxation. For each of the heart chambers the cardiac cycle

can be divided into two phases. During contraction, or systole, the chamber contracts and blood is pushed into an adjacent chamber or arterial trunk. Diastole follows systole. During diastole, the chamber fills with blood and prepares for the next cardiac cycle. The pressure within each chamber rises during systole and falls during diastole. The valves help to ensure that the blood flows in the correct direction. However, blood will only flow from the first to the second chamber, if the pressure in the first chamber is greater than that of the second. The correct pressure relationship is dependent on the timing of contractions. Blood movement would not occur if the atria and ventricle contacted together.

ELECTROPHYSIOLOGY

The excess of positively charged ions outside the cell membrane, and the slight excess of negatively charged ions inside the cell, causes the inside of a cell membrane to have a slight negative charge with respect to the outside. This unequal distribution of charges is due to the difference in permeability of the cell membrane to the differently charged ions. The positive and negatively charged ions, although separated by a cell membrane, are attracted to one another. A **potential difference** exists when positive and negative ions are held apart. If not separated by a cell membrane, the opposite charges would rush together. The potential difference across a cell membrane is referred to as the **transmembrane potential**. The **volt** is the unit of measurement for potential difference. The transmembrane potential in an undisturbed cell is the **resting potential**. Each cell type has a characteristic resting potential. The normal resting potential is negative. This is because the interior of the cell contains abundant negatively charged proteins, which cannot cross the cell membrane. At the normal resting potential there is a slow leakage of sodium into the cell and a diffusion of potassium out of the cell. A cell expends energy to maintain its resting potential. The sodium–potassium exchange pump stabilizes the resting potential by ejecting sodium ions from within the cell membrane and reclaiming potassium ions from the extracellular fluid.

Membrane channels enable ions to cross the cell membrane. **Passive** and **active** sodium and potassium channels exist. **Leak** or passive channels are always open and are important in maintaining the normal resting potential. Active or **gated** channels open or close in relation to specific stimuli. Three classes of gated channels exist: **chemical-, voltage- or mechanically regulated**. Voltage-regulated channels are characteristic of areas of

excitable membrane, i.e. a membrane capable of generating and conducting an **action potential**, e.g. cardiac muscle cells. Voltage-regulated channels open or close in response to changes in the transmembrane potential. The opening of voltage-regulated sodium channels is responsible for the generation of an action potential.

Graded potentials are changes in transmembrane potential that cannot spread far from the site of stimulation. When a membrane is exposed to a chemical that opens chemically regulated sodium channels, sodium ions enter the cell. The arrival of additional positive charges shifts the transmembrane potential towards 0 mV[1] This is called depolarization. The depolarization of surrounding membrane follows the movement of sodium ions across the cell membrane at one location. During this time extracellular sodium ions move towards open channels, replacing those that enter the cell. This is called a **local current**. The number of sodium channels opened by the chemical stimulus is directly proportional to the change in transmembrane potential, and the area affected by the local current. The more channels open the greater the depolarization. When the chemical stimulus is removed the transmembrane potential soon returns to normal. Repolarization is the process of restoring the normal resting potential.

Action potentials are changes in the transmembrane potential that spread across an entire excitable membrane. A graded potential initiates an action potential. A graded potential is large enough to bring an area of excitable membrane to **threshold.** Voltage-regulated sodium channels are opened and allow sodium ions to flood into the cell. There is rapid depolarization. When an action potential is stimulated, it propagates over the entire surface of the excitable membrane. During **repolarization,** sodium channels close, potassium channels open and potassium ions move out of the cell. Finally, all voltage-regulated channels close, and the membrane is back to its resting state.

For a certain period after an action potential begins the membrane will not respond to another stimulus. This is called the **refractory period**. In the **absolute refractory period** the cell will not respond regardless of how strong the stimulus is. An action potential in a cardiac muscle cell differs from that in a skeletal muscle cell due to the presence of a plateau stage between rapid depolarization and repolarization. The plateau represents calcium ion entry via slow calcium channels and prolongs the action potential. The period of active muscle cell contraction continues until the plateau ends. The absolute refractory period continues until relaxation of the cardiac muscle cell is under way. This prevents tetany occurring in cardiac muscle, which would be fatal, as a heart in tetany cannot pump blood.

Conduction system of the heart

During a single heartbeat, the entire heart contracts in a coordinated manner. Thus blood flows in the right direction at the proper time. Contractile cells, and the conducting system, are the cardiac muscle cells involved in a normal heartbeat. Gap junctions connect all heart muscle cells, including the cells of the conduction system, to each other. These gap junctions make it easier for impulses to spread between adjacent cells. So, immediately after a heart cell depolarizes, the cells around it depolarize. In this way, a wave of excitation and contraction spreads over the entire heart.

The conducting system includes:

1 *Sino-atrial (SA) node* – found in the wall of the right atrium.
2 *Atrioventricular (AV) node* – found at the junction between the atria and the ventricles.
3 *Conducting cells* – connect the two nodes and distribute the contractile stimulus throughout the myocardium. Conducting cells in the atria are in the internodal pathway. Ventricular conducting cells include those in the AV bundle, bundle branches and Purkinje fibres. These cells distribute the stimulus to the ventricular myocardium (**Figure 4.2**).

The cells of the conducting system cannot maintain a stable resting potential. As soon as repolarization has occurred, the membrane gradually drifts towards threshold. This rate of spontaneous depolarization varies in

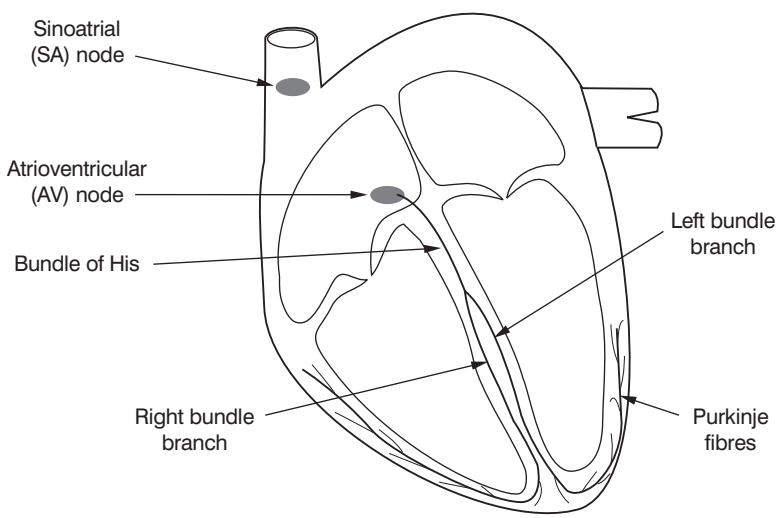

Figure 4.2 – The conduction system of the heart

different portions of the conducting system. It is fastest at the SA node. Cells of the AV node depolarize more slowly and normally most cells of the AV bundle, bundle branches and Purkinje fibres do not depolarize spontaneously. The SA node establishes heart rate as it reaches threshold first.

The SA node contains pacemaker cells, which establish heart rate. The stimulus then crosses the surface of the atria to the AV node. This takes 50 msec. At the AV node, there is a delay of 100 msec and atrial contraction begins. The time elapsed is 150 msec. The connection between the AV node and the AV bundle, or bundle of His, is the only electrical connection between the atria and the ventricles. From the AV bundle, impulses pass through the right and left bundle branches. The left bundle branch is much larger than the right bundle branch. After extending to the apex of the heart, both bundle branches turn out under the endocardial surface. Impulses are then passed on to the Purkinje fibres (time elapsed is 175 msec). The impulses are relayed through the ventricular myocardium. Atrial contraction is complete. Ventricular contraction begins. At this point the total time elapsed is 225 msec.[1]

The normal rhythm of the heart is disturbed if the conducting pathways are damaged. If the SA or internodal pathways are damaged, the AV node will take over. The heart will beat at a slower rate. If a conducting cell or ventricular muscle cell generates an action potential more rapidly than the SA or AV node, then this is called an ectopic pacemaker. This will bypass the conducting system and disrupt the timing of ventricular contraction. This will result in a reduction of the efficiency of the heart, and may be diagnosed with an electrocardiogram.

The Electrocardiogram

Electrodes attached to the surface of the body can detect the electrical changes associated with muscle contraction. An electrocardiogram is a surface recording of the electrical activity of the heart represented graphically. An electrocardiogram can be undertaken for a number of reasons, including:

1 Detection of heart rhythm disturbances
2 Provision of a baseline reading of the electrical activity of the heart
3 Determination of the effects of drugs, e.g. Digoxin
4 Identification of diseases of the conduction system
5 Detection of atrial and ventricular hypertrophy

6 Detection of myocardial infarction or ischaemia
7 Detection of the origin of an arrhythmia
8 Detection of pericarditis
9 Detection of electrolyte imbalance
10 Evaluation of the effectiveness of a cardiac pacemaker

It is important that the ECG is not used in isolation, but in conjunction with an examination of the patient. When taking an ECG, recordings are made from electrodes placed on different parts of the body. Each of the electrode systems is referred to as a lead. Recordings can be taken from limb leads, when electrodes are placed on the arms and legs, or from chest leads, when electrodes are placed on the chest. The limb leads are labelled I, II and III and the chest leads as $V_{1–6}$. Augmented limb leads are labelled as aV_l, aV_r and aV_f to donate left and right arm and foot.

The limb leads are bipolar, i.e. the electrical activity is monitored at two sites and compared by the recording equipment. By convention, the right arm is a negative pole, the left arm is a positive pole (except in lead III, where it is negative), and the left leg is a positive pole. Lead I records the potential between the right arm and the left arm. Lead II records the potential between the right arm and the left leg (when a patient is placed on a cardiac monitor the electrodes are normally placed in such a position to record this potential). Lead III records the potential between the left arm and left leg. The chest leads are unipolar, monitoring activity at one site. The lead placed on the chest is the positive pole and the limb leads form the negative pole. The augmented limb leads are unipolar and compare the differences between a given point and zero (**Figure 4.3**).

Impulses travel from negative to a positive pole. The impulses travelling towards the positive pole give a positive deflection on the ECG graph paper. The shape of the ECG varies, depending on the lead used, i.e. the waves are there, and their timing the same, but their shape and size are different. The recordings taken from a patient on a cardiac monitor, are normally taken from limb lead II.

From an electrical viewpoint, the heart can be thought of as having two chambers, the atria and the ventricles. The muscle mass of the atria is small. Therefore, their contraction and accompanying electrical changes are small. When the ventricles contract, there is a large deflection on the ECG. **Figure 4.4** is a diagram of the basic ECG waveform.

All ECG machines run at a standard rate, each using paper with standard squares. The horizontal axis represents time.

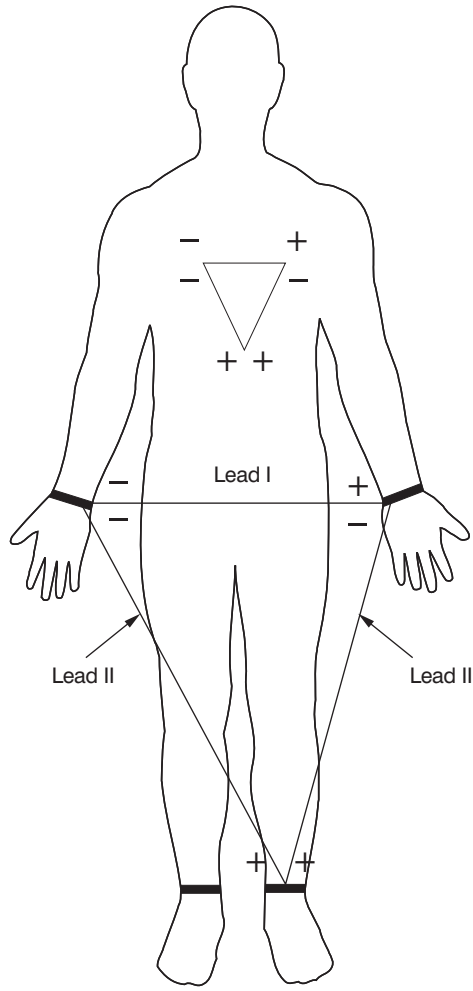

Figure 4.3 – The bipolar limb leads

1 One small square = 0.04 seconds
2 One large square = 0.2 seconds

The vertical axis represents voltage/magnitude:

1 One small square = 1 mm
2 One large square = 5 mm or 0.5 mV

It is important that the ECG machine is standardized, 1 mV giving a deflection of 10 mm (1 cm).

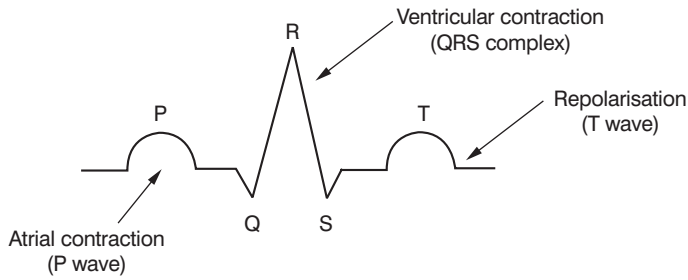

Figure 4.4 – The basic ECG waveform

Recording an ECG

Before attempting to record an ECG, the nurse must be familiar with the ECG machine and how to operate it. This will help to prevent an incorrect recording and in turn prevent an incorrect diagnosis and time wastage. The patient should be lying down and relaxed to reduce electrical interference from skeletal muscle. A good electrical contact is required, therefore, before attaching the electrodes. It may be necessary to wipe the skin with a spirit wipe and remove excess hair. The bipolar limb electrodes and unipolar chest electrodes should be correctly positioned (**Figure 4.5**). The electrodes will usually be labelled or colour-coded to assist in this process.

Before commencing recording, check that the machine is set at the correct paper speed (usually 25 mm/second) and that the calibration mark has been made such that 10 mm = 1 mV (wave height can then be readily converted into a more meaningful voltage).

Recognition of cardiac rhythms

The following describes a number of easy steps involved in the recognition of cardiac rhythm.[2]

QRS rate

The QRS complex represents ventricular depolarization. The QRS rate is classified as:

1 Normal – 60–100/minute
2 Bradycardic (slow) – < 60/minute
3 Severely bradycardic – < 40/minute
4 Tachycardic (fast) – between 150 and 200/minute

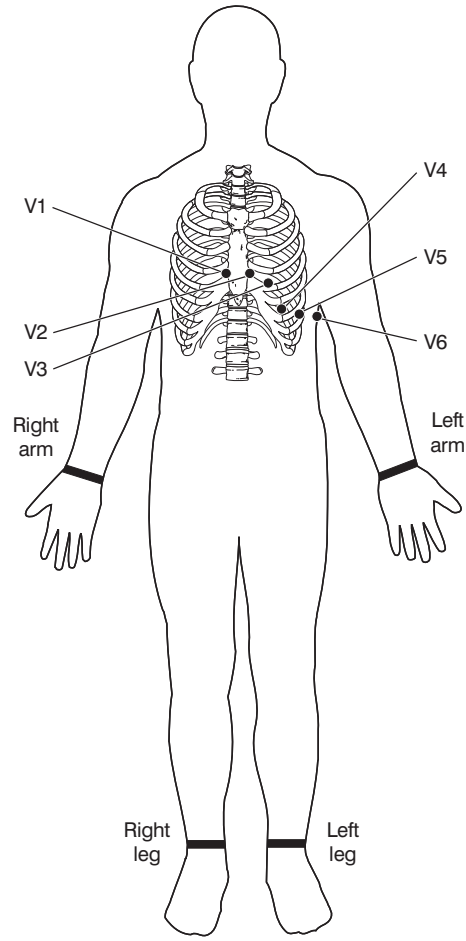

Figure 4.5 – ECG limb and chest electrodes

Heart rate can be calculated by counting the number of large 5 mm squares between consecutive R waves, and then divide this number into 300. This is demonstrated in **Table 4.1**.

QRS rhythm

By mapping out each of the R waves, it is possible to identify whether the QRS rhythm is regular or irregular.

Are there any pauses present? (A pause > 3 seconds requires rapid investigation as there is a risk of asystole).

Table 4.1 *– R-R interval and heart rate*

No. of 5 mm squares between R waves.	Heart rate (beats per min.)
1	300
1.5	200
2	150
3	100
4	75
5	60
6	50
7	45
8	40
9	35
10	30

QRS duration

Is the QRS broad or narrow? It should normally be 0.12 seconds (three small squares or less). Any abnormality of conduction takes longer and causes widening of the QRS complex. Widening of the QRS complex generally occurs when the speed of impulses are either triggered from an ectopic focus or fail to take the correct route. It takes longer for the spread of impulses to depolarize the myocardium, giving rise to a broad QRS complex.

Atrial activity

The 'P' wave represents normal atrial activity.

1 Are the P waves absent?
2 Are they all the same shape and morphology?
3 Is the pattern irregular?
4 Is there any atrial fibrillation?
5 Are there any atrial flutter waves (saw toothed in shape and at a rate of 300 per minute)?

Atrio-ventricular relationship

1 Is there 1:1 conduction (i.e. one P wave to one QRS complex), or is it 2:1 or 3:1?
2 Is the P-R interval normal, i.e. between 0.12 and 0.2 seconds (three-to-five small squares) and a constant length throughout the rhythm?

Abnormal cardiac rhythms

The conduction pathway and normal rhythm of the heart can be damaged in a number of ways, including:

1 Mechanical distortion
2 Ischaemia
3 Infection
4 Inflammation

The resulting deficit is called a heart block. In first-degree heart block the AV node and proximal part of the AV bundle slows the passage of impulses. A pause appears between the atrial and ventricular contraction. However, the heart rhythm is regular and a QRS complex follows each P wave. In mild second-degree heart block an occasional skipped beat may be seen. If the delay lasts long enough the ventricles will follow every second atrial beat. This pattern of 'atria, atria-ventricles, atria, atria-ventricles' is known as a 2:1 block. It is possible for an individual to develop a three-to-one or even a four-to-one block. During third-degree heart block or complete heart block the conduction pathway no longer functions. Although the atria and the ventricles beat, their activity is not synchronized. The atria follow the pace set by the SA node beating about 70–80 times per minute, and the ventricles follow the pace set by the AV node, beating about 40–60 times per minute.

Premature atrial contractions (PAC) can occur in healthy individuals. In PAC the normal atrial rhythm is interrupted by a surprise contraction. Stress, drugs and caffeine can all increase the incidence of PAC. In paroxysmal atrial tachycardia (PAT) a flurry of atrial activity is stimulated by a premature atrial contraction. The ventricles keep pace with the atria and the heart rate can be 180 beats per minute. During atria flutter the atria contract in a coordinated manner with frequent contractions. In atrial fibrillation the impulses travel over the atria at about 500 beats per minute. The walls of the atria quiver as opposed to producing an organized contraction. The ventricular rate remains within normal limits as they cannot keep pace with the atrial rate. Both these conditions can go unnoticed and are not considered very dangerous unless they are prolonged or associated with other cardiac damage, e.g. coronary artery disease. By contrast ventricular arrhythmias can be serious and even fatal.

As the heart's conduction system functions in one direction only, ventricular arrhythmias are not linked to atrial activity. Premature ventricular contractions (PVC) occur when a Purkinje cell or ventricular

myocardial cell depolarizes to threshold and triggers a premature contraction. The cell causing this is called the ectopic pacemaker. Ventricular tachycardia (VT) often precedes ventricular fibrillation (VF), the most serious arrhythmia. This condition is fatal as the heart stops pumping blood. Impulses are travelling from cell to cell and around the ventricular walls. A normal rhythm cannot occur because the ventricular cells are stimulating each other at such a rapid rate. A defibrillator is used to restore normal cardiac rhythm (see **Chapter 5**). It is very important not to diagnose asystole as fine VF. Asystole means there is no spontaneous electrical cardiac activity. There is an absence of ventricular activity and heart rate is zero.

References

1. Martini, F.H. *Fundamentals of Anatomy and Physiology* (4th edition). 1998; Prentice Hall International.
2. Hampden, J.R. *The ECG Made Easy* (4th edition). 1992; Edinburgh: Churchill Livingstone.

Suggested Reading

1. Hampden, J.R. *The ECG Made Easy* (4th edition). 1992; Edinburgh: Churchill Livingstone.
2. Hampden, J.R. *The ECG in Practice* (2nd edition). 1992; Edinburgh: Churchill Livingstone.
3. Houghton A.R., Gray D. *Making Sense of the ECG* (2nd edition). 1997; London: Arnold.

Review questions

1. Where does the pulmonary circulation carry blood to and from?
2. Where does the systemic circulation carry blood to and from?
3. What is the anatomical difference between the right and left ventricle?
4. What do valves prevent?
5. What is the function of the coronary circulation?
6. Where do the coronary arteries originate?
7. Starting at the vena cava, describe the direction of blood flow through the heart.

8. The left atrioventricular valve has two names, what are they?
9. What determines the resting potential of a neurone?
10. The cell membrane contains active (gated) channels and passive (leak) channels. Describe the functions of these channels.
11. Name two types of gated channels.
12. When would an action potential appear?
13. What is the role of the sodium–potassium exchange pump?
14. What are the two classes of cardiac muscle cells involved in the normal heartbeat?
15. What is the route of conduction of an electrical impulse from the SA node to the ventricles?
16. Do cardiac muscle cells need neural or hormonal stimulation to contract?
17. Which cells establish the rate of contraction?
18. Identify the following rhythms (**Figure 4.6**)

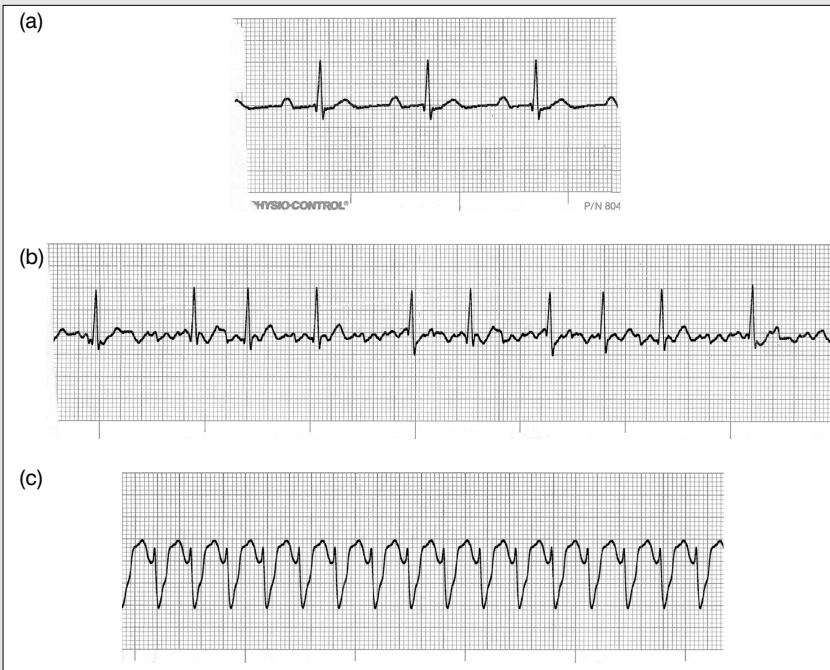

Figure 4.6 – (also continued overleaf)

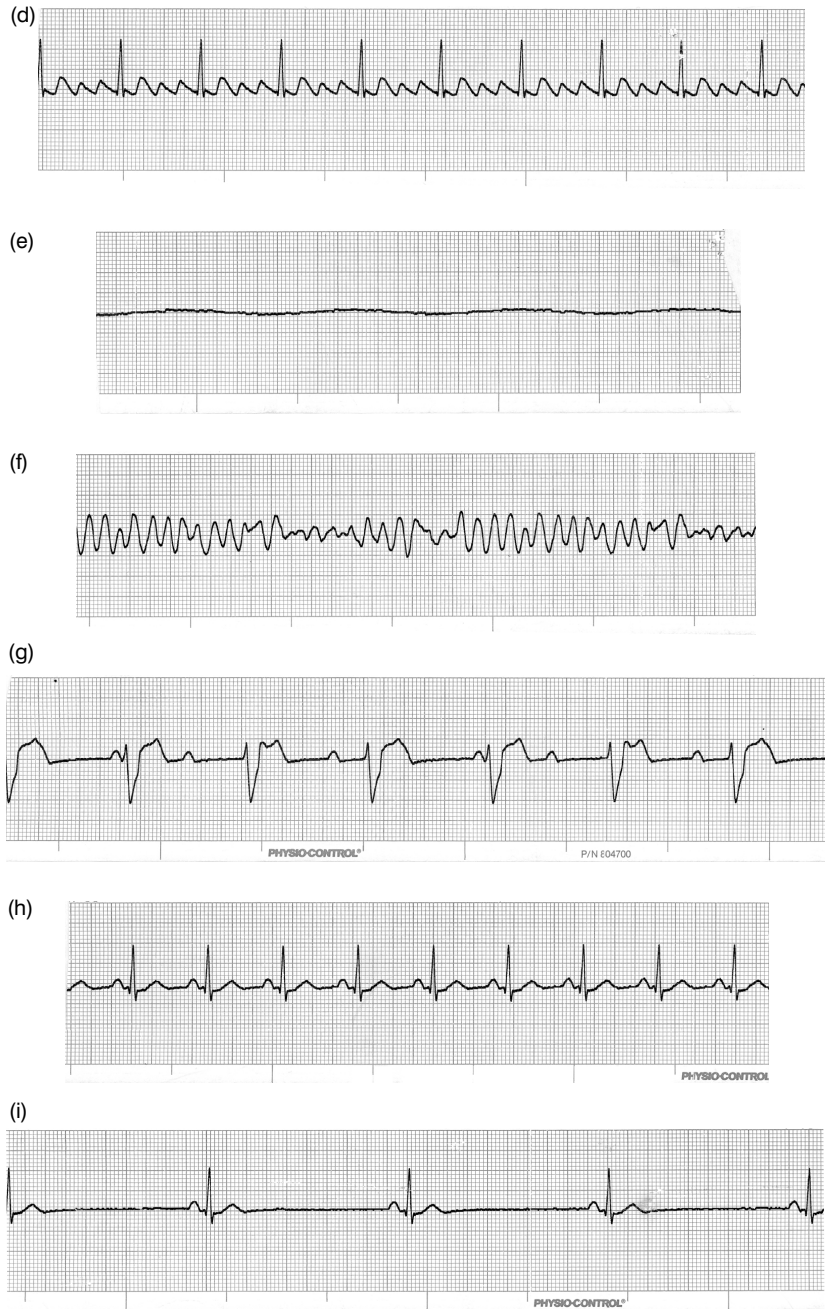

Figure 4.6 – (continued)

Review questions – answers

1. To and from the lungs
2. The rest of the body
3. The right ventricle is thin in comparison with the left ventricle, which has a very thick muscular wall
4. The regurgitation of blood
5. To meet the high oxygen and nutrient demands of the cardiac muscle cells
6. The base of the ascending aorta
7. Vena cava – right atrium – right ventricle – pulmonary circulation (pulmonary artery, pulmonary vein) – left atrium – left ventricle – aorta
8. Mitral valve and bicuspid valve
9. The membrane's permeability to potassium
10. Channels through which ions are able to pass to enter or leave a cell
11. Chemical, voltage and mechanically regulated
12. When a region of excitable membrane depolarizes to threshold
13. Maintain ion concentration (inside and outside the cell membrane) within acceptable limits
14. Contractile cells and conducting system
15. SA node – across atria – AV node – bundle of His – right and left bundle branches – Purkinje fibres – left ventricle
16. No
17. Pacemaker cells in the SA node.
18. (a) First-degree heart block
    (b) Atrial fibrillation
    (c) Ventricular tachycardia
    (d) Atrial flutter
    (e) Asystole
    (f) Ventricular fibrillation
    (g) Complete heart block
    (h) Sinus tachycardia
    (i) Sinus bradycardia