Understanding the ECG. Part 1: Anatomy and physiology

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Understanding the ECG. Part 1: Anatomy and physiology

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Cardiovascular disease is one of the main causes of death in the UK, and a common cause of hospital admission (Bhatnagar et al, 2015). It is important, therefore, that health-care practitioners develop their skills and knowledge in relation to this problem if patients are to obtain the best possible care in a timely manner. One of the most important diagnostic tests is the 12-lead electrocardiogram (ECG) (Society for Cardiological Science & Technology, 2014). Despite its widespread use, many people struggle to master the basics of ECG interpretation and errors in interpretation can lead to misdiagnosis and delays in appropriate treatment. One of the reasons for this is a limited knowledge of ECG interpretation resulting from a lack of appropriate training (Richley, 2013).

This is the first in a series of articles that will aim to:

- Explore why ECGs are important tools in the diagnosis and management of heart disease
- Explore how they can be interpreted
- Provide the reader with an understanding of the anatomy and physiology of the heart and conducting system.

This first article will explore and discuss the anatomy and physiology of the heart's electrical system. This electrical activity is fundamental in coordinating the function of the heart, and it is this activity that ECG machines record and display.

The ECG

The ECG is an important screening tool that offers practitioners a wealth of information that can be used alongside the history and clinical findings (Younker, 2011). An ECG provides a measurement of the rate and rhythm of the heart. It also provides information about the health of the electrical system, the size of the heart chambers, and the supply of blood to the heart muscle (Hampton, 2008). ECGs are pivotal in the diagnosis of cardiac ischaemia and infarction, provide the evidence for pacemaker implantation, and detect inherited abnormalities such as cardiomyopathy and long-QT syndrome (Jowett and Thompson, 2007). ECGs are also useful in detecting non-cardiac pathology, for example, pulmonary emboli and electrolyte disorders (Garcia, 2015).

From many practitioners' perspective, ECGs are perceived as complex and difficult to understand—a 'black art' understood by the few (Wetherell, 2013). However, the premise of this series is that this doesn't have to be the case. We will put forward the argument that ECGs can be understood by anyone with the time and patience to build the necessary knowledge.

Electrolytes and electricity

It is important to develop a good understanding of the way in which our cells function. A firm grounding in basic cell physiology will help readers to understand how the heart works, and to appreciate the implications of findings on the ECG. This section will introduce you to a number of terms that are important if you wish to interpret and understand an ECG, such as resting potential, repolarisation, depolarisation and action potential.

The body is composed of millions of individual cells, each enclosed by a fatty cell membrane and surrounded by extracellular fluid (Alberts et al, 2010). Both the cells and the surrounding fluid contain multiple substances including water, proteins and electrolytes. It is the concentration of electrolytes, inside and outside the cell, and their ability to cross the cell membrane, that creates electrical activity in the cell. In the heart, the most important electrolytes are sodium, potassium and calcium (Klabunde, 2012). *Figure 1* shows a typical cardiac cell.
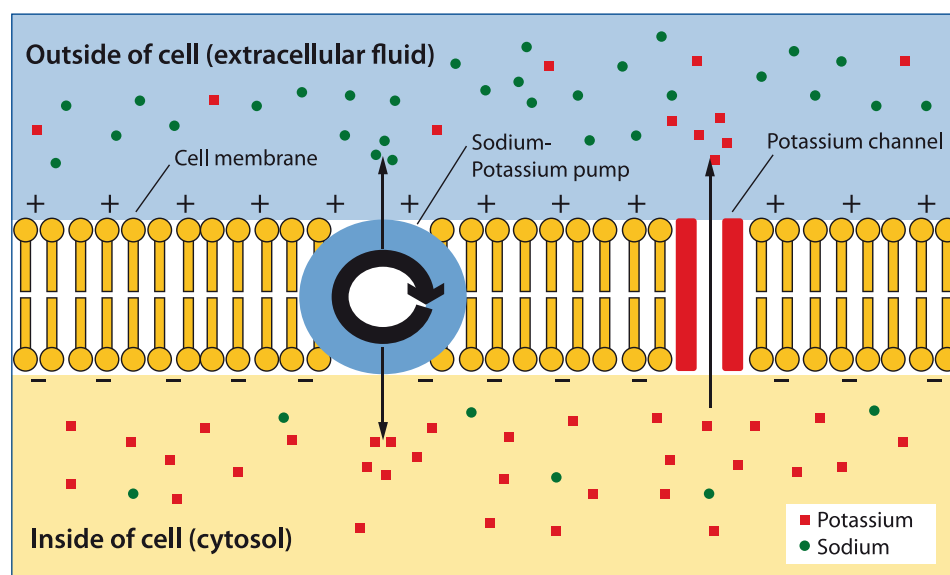

Figure 1. The cardiac cell

There is considerable interchange between the cells and the extracellular fluid. Although water and oxygen, for example, cross the cell membrane freely, the cell membrane has limited permeability to electrolytes (Marieb and Hoehn, 2015). There are two ways that the movement of electrolytes across the cell membrane is increased. Firstly, they are moved into and out of the cell by pumps embedded in the cell membrane. The most important of these is the sodium-potassium pump (Levick, 2010).

The sodium-potassium pump moves sodium out of the cell and pumps potassium in. Because it pumps continuously, potassium accumulates inside the cell, leading to a higher concentration inside than outside. In the same way, sodium concentration increases in the extracellular fluid outside the cell. The second way that electrolytes cross the cell membrane is through ion channels. Ion channels are 'doors' in the cell membrane that open and close in response to stimuli (Grant, 2009).

It is important to note that ion channels are specific to one electrolyte; for example, sodium channels only allow sodium through. When an ion channel or 'door' opens, the electrolyte moves into or out of the cell depending on the concentration gradient. In other words, it moves from the area of highest to the area of lowest concentration. When potassium channels open, potassium leaves the cell. In contrast, sodium enters the cell when its ion channels open (Pappano and Wier, 2013).

The result of these opposing concentrations of electrolytes is an electrical charge across the cell membrane. All electrolytes are electrically charged. Sodium (Na+) and potassium (K+) carry a single positive charge, while calcium (Ca++) carries two (Kaplan and Kellum, 2010). At rest, the inside of the cell membrane has a negative charge of around -90 mv, compared with the outside (Grant, 2009).

This difference in electrical charge is called the resting potential (Woods et al, 2010). The resting potential changes when ion channels open in the cell membrane, and electrolytes enter or leave the cell along their concentration gradient. This change in electrical potential is used to trigger cellular activity in many parts of the body including the nerves and skeletal muscles. In the heart, however, it triggers mechanical contraction (Marieb and Hoehn, 2015).
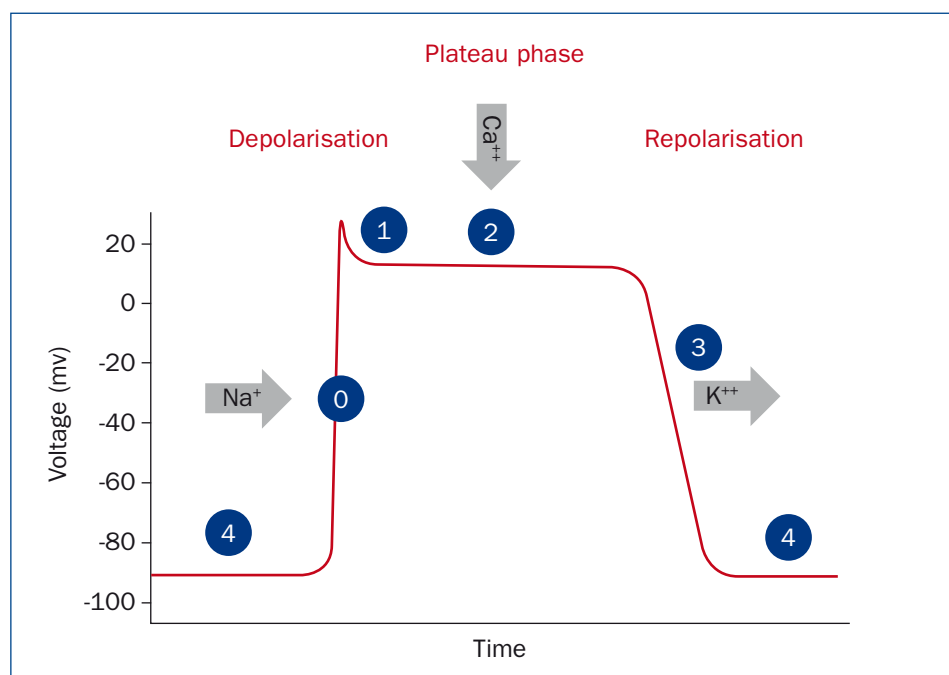

Figure 2. The action potential

Triggering contraction: the action potential

The heart is made up of the following three main tissue types (Levick, 2010):

- Fibrous
- Electrical
- Contractile.

Fibrous tissue provides a supporting skeleton to the muscle mass, and forms the heart valves. Fibrous tissue does not contract and, importantly, does not conduct electricity (Garcia, 2015). In contrast, electrical cells are highly conductive, and have pacemaker properties that will be discussed shortly. Before doing so, let us turn our attention to the final type of tissue: the contractile cells or myocytes. These provide the pumping action that moves blood through the heart, and are the commonest cardiac cell type by far (Tortora and Nielsen, 2014).

At rest, the inside of the myocyte cell membrane is slightly negative. This changes when an electrical impulse arrives at the cell membrane. The electrical impulse causes sodium channels in the cell membrane to open, allowing sodium to enter into the cell along its concentration gradient. This sudden influx of sodium alters the electrical polarity of the cell membrane—it goes from being negative on the inside to being slightly positive (*Figure 2*). This change in electrical potential is called depolarisation (Grant, 2009). Once the cell has depolarised, calcium channels open

and calcium enters the cell. This stabilises the electrical charge across the cell membrane and closes the sodium channels. Calcium flows slowly into the cell, creating an electrical plateau phase during which the electrical potential does not change (Fogoros, 2007). Calcium is also released from intracellular stores at this time. The increase in calcium concentration inside the cell triggers mechanical contraction of the cell (Aaronson et al, 2013).

When contraction is complete, calcium channels close and potassium channels open. Potassium flows briefly from the cell. This causes repolarisation; the return of resting electrical potential. The whole process of depolarisation and repolarisation is described as an action potential. The phases of the action potential are usually numbered from 0–4, as shown in *Figure 2* (Woods et al, 2010).

Once one cell depolarises, the action potential spreads rapidly from cell to cell across the heart muscle. This results in a wave of electrical activity that spreads rapidly across the tissue, swiftly followed by mechanical contraction. The wave of depolarisation continues until all cells are depolarised, or it meets non-conducting tissue (Christoffels and Moorman, 2009). A good analogy is the throwing of a stone into a pond. Once the stone hits the water, the ripples travel in all directions until they meet a solid object such as the pond edge and they then stop. The electrical wave-
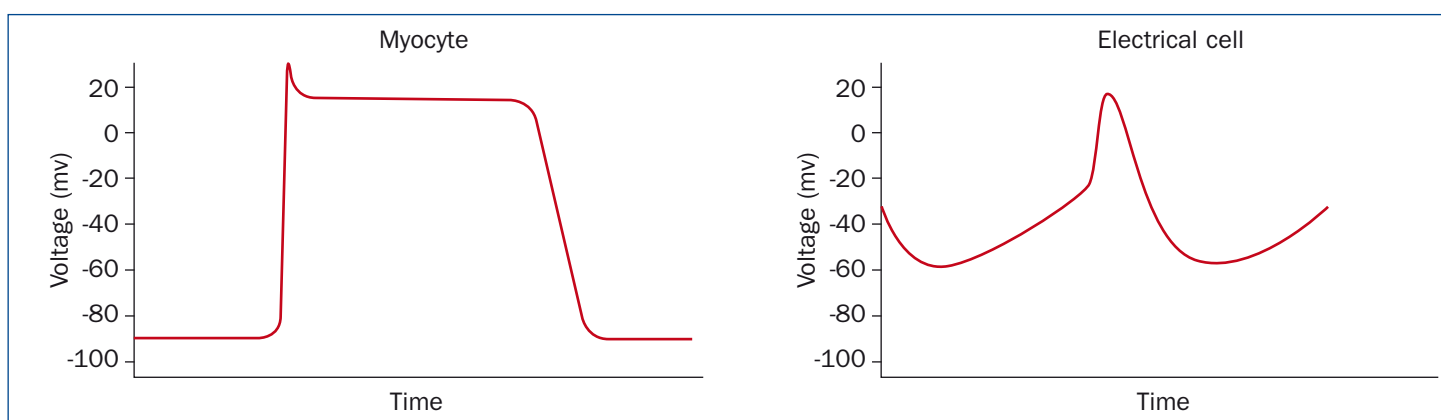
Figure 3. The action potential in myocytes and electrical cells

front travels through the heart muscle in a similar manner.

Once a cell has been depolarised, it becomes refractory for a brief time (Marieb and Hoehn, 2015). To put it simply, the cell cannot be re-stimulated until its electrical activity has returned to near resting state. Cardiac cells exhibit two types of refractory period. During the initial absolute refractory period, the cell is completely incapable of further depolarisation. Following this, a short relative refractory period exists before the full resting state is restored. During the relative refractory period, the cell may be depolarised again if a large enough stimulus is applied. The refractory period prevents the cell from being stimulated again too quickly, as can happen in skeletal muscles affected by tetany (Klabunde, 2012).

It would be fantastic if everybody's heart behaved in this textbook fashion. Unfortunately, patients are subject to disease processes and health issues that may interfere with normal electrical function. Electrolyte imbalance, ion channel abnormalities and medications can all affect the normal movement of electrolytes across the cell membrane (Grant, 2009). A good example of electrolyte imbalance is hypokalaemia (low serum potassium level). Hypokalaemia changes the normal potassium concentration gradient, and makes patients more vulnerable to ventricular arrhythmias (Resuscitation Council UK, 2010). Ion channel abnormalities are genetic mutations that change the normal function of one or more channels. This results in conditions such as long-QT and Brugada syndrome which are associated with fatal arrhythmias (Martin et al, 2012). Finally, ion channel function is affected by commonly used drugs. Calcium channel

blockers are used to treat angina, hypertension and arrhythmias (Sargent, 2006). Two drugs in this group, diltiazem and verapamil, slow the heart rate by blocking calcium channels in the sinus node. Other drugs block sodium or potassium channels, for example, flecainide (sodium) and sotalol (potassium) (Opie and Gersch, 2013). Amiodarone, one of the most commonly used anti-arrhythmic drugs, works by blocking multiple ion channels (O'Donovan, 2006).

Electrical cells and automaticity

We have described how an initial electrical stimulus depolarises a myocyte, and is then propagated across the entire muscle mass. In a skeletal muscle, the initial impulse comes to the muscle via the brain and nervous system (Marieb and Hoehn, 2015). The heart is different. In the heart, the stimulus comes from its specialised electrical cells. The sole purpose of these cells is to generate and conduct the electrical impulses that trigger contraction of the myocytes (Aaronson et al, 2013). Their principal feature is automaticity. Automaticity means that the cell is able to act as a pacemaker. It does this by spontaneously depolarising itself. In the myocyte, the cell membrane is electrically stable at rest. There is no electrical change until an impulse arrives from an adjacent cell. In contrast, there is a constant leakage of ions across the cell membrane of the electrical cell. This results in a gradual movement
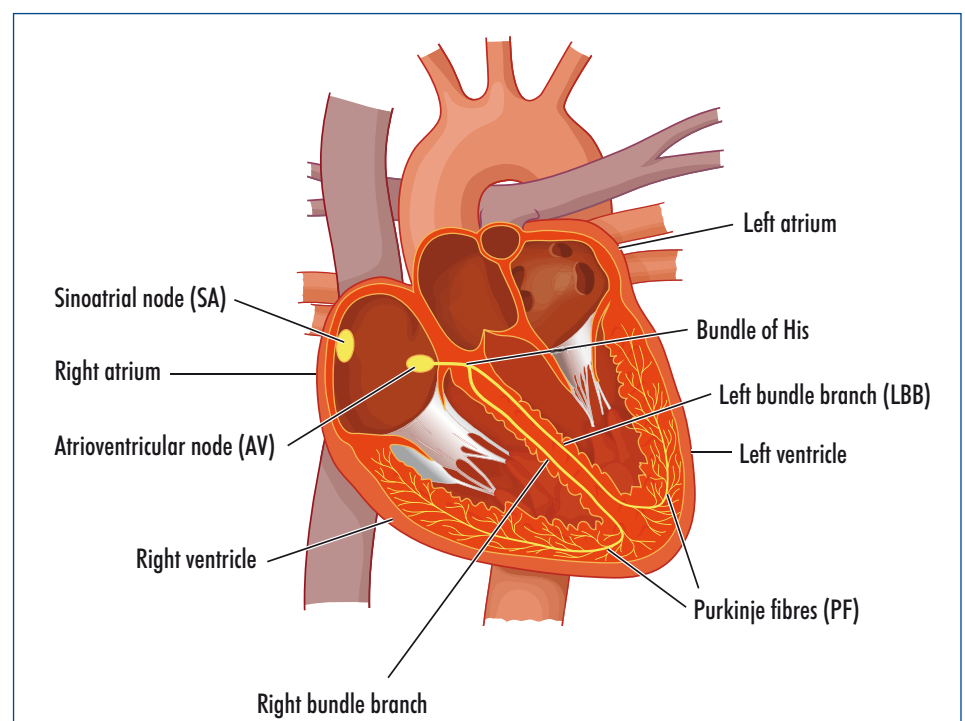
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Figure 4. The cardiac conduction system

towards depolarisation. Once a threshold is reached, calcium channels open and calcium floods into the cell, completing depolarisation. The action potential created spreads through both the electrical system and the surrounding myocytes (Fogoros, 2007). *Figure 3* compares the electrical activity in the myocyte and the electrical cell. Note that the baseline is constantly moving upward in the electrical cell, unlike the flat baseline of the myocyte. Also, the electrical cell has no plateau phase because it does not contract.

The heart's conduction system

This is a key section to understand as it provides insight into the waves recorded on the ECG and what they mean. The heart's electrical cells are arranged in a conduction system that carries the electrical impulse to every part of the organ (*Figure 4*). This ensures that the atria contract before the ventricles, and that ventricular contraction is coordinated and efficient (Hampton, 2008). The Conduction system comprises of the sinoatrial node (SA or sinus node), atrioventricular node (AV node), bundle of His, left and right bundle branches, and Purkinje fibres (Garcia, 2015).

Normally, the electrical impulse that initiates the heart beat originates in the sinus node. This small patch of electrical cells is located in the right atrium, near its junction with the superior vena cava (Christoffels and Moorman, 2009). The sinus node acts as the primary pacemaker because it has the fastest rate of depolarisation. If it slows down, or fails, the next fastest pacemaker takes over. The normal rate of depolarisation of the different parts of the conduction system is shown in *Table 1*. This is an important fail-safe feature of the system, and ensures that the heart continues to beat even if the sinus node is diseased or damaged. Sinus node disease is common in older individuals and is caused by fibrosis of the conduction tissue. The node may also be damaged by myocardial infarction, surgery or diseases of the heart muscle such as cardiomyopathy (Houghton and Gray, 2014).

From the sinus node, the electrical impulse spreads across both atria, causing the atrial myocytes to depolarise. The impulse also arrives at the AV node, located in the inferoseptal right atrium (Garcia, 2015). Conduction through the AV node is

ten times slower than through the surrounding muscle (Klabunde, 2012). This slowing of conduction allows the atria to finish contracting before the impulse passes into the ventricles. Slow conduction through the AV node also limits how many beats per minute the node can conduct. This is an important mechanism that protects the ventricles from high atrial rates during atrial arrhythmias such as atrial fibrillation (Lafuente-Lafuente et al, 2009).

From the AV node, the electrical impulse enters the bundle of His. The bundle of His penetrates the atrial wall and enters the interventricular septum. It also crosses the fibrous layer that separates the atria and ventricles. Because fibrous tissue does not conduct electricity, the AV node and bundle of His is the only route of electrical conduction from atria to ventricles in the normal heart.

The bundle of His divides within the septum to produce the left- and right-bundle branches. These carry the electrical impulse into the left and right ventricles. The bundle branches terminate in a branching network of Purkinje fibres that carry the electrical impulse to every part of the ventricle almost simultaneously, ensuring rapid and effective contraction of the chamber (Pappano and Wier, 2013). Conduction through the His-Purkinje system is extremely rapid, much faster than through the muscle cells of the ventricles.

If the system is damaged, for example, by ischaemic heart disease, conduction to the ventricles may be delayed or fail completely (Jowett and Thompson, 2007). These problems can be seen on the ECG and are referred to as heart blocks (Bennett, 2013). Complete heart block occurs when no electrical impulses are able to travel from the atria to the ventricles. This often results in severe bradycardia and is a life-threatening condition (Swift, 2013).

Extra-cardiac influences

Because of automaticity, electrical cells in the heart do not need stimulation from the

nervous system in order to depolarise. This means that the heart will continue to beat even when removed from the body, provided it is supplied with oxygen and nutrients (Marieb and Hoehn, 2015). This feature has recently been exploited to keep donated hearts alive prior to transplant, using a machine called an Organ Care System (Transmedics Inc, 2015). Despite this self-reliance, the heart is heavily innervated by the autonomic nervous system. Nerves from both sympathetic and parasympathetic branches of this system run from the brain to the heart (Levick, 2010). These nerves help to regulate the rate at which the heart beats, as well as the speed of electrical conduction and the force of contraction. This allows the brain to match cardiac output to circulatory demand.

Sympathetic nerves act as the heart's 'accelerator', increasing heart rate, speed of conduction, and contractility. Sympathetic activity increases during exercise and stress. Its opposite, the parasympathetic system, acts as the 'brakes' and slows the heart at rest. The parasympathetic nerve running to the heart is the Vagus nerve (Tortora and Nielsen, 2014).

At rest, the heart is said to exhibit vagal tone, meaning that parasympathetic tone predominates. As the demand for cardiac output rises, for example, during exertion, parasympathetic tone diminishes and sympathetic tone increases (Klabunde, 2012). A 'vasovagal reaction' occurs when a physical or emotional stimulus causes a sudden increase in parasympathetic outflow to the heart. The sudden drop in heart rate and contractility lowers blood pressure, and causing dizziness or loss of consciousness (Blanc et al, 2015).

The other important extra-cardiac influence on the heart is the endocrine system. Many hormones influence cardiac electrical activity including thyroid and growth hormones (Levick, 2010). Clinically, the most important group of hormones affecting the heart are the catecholamines, epinephrine and norepinephrine. These bind to

Table 1. Rates of depolarisation	
Area of the conduction system	**Depolarisation rate**
SA node	60–100 beats per minute
AV node	40–60 beats per minute
His-Pukinje system	20–40 beats per minute

Figure 5. The 12-lead ECG

Figure 6. ECG waveforms

receptor sites on the heart, increasing heart rate, conduction speed and contractility. Synthetic forms of epinephrine and nore-pinephrine are used to maintain cardiac output and blood pressure in critically ill people (Parry, 2012). Other drugs have the opposite effect.

Beta-blockers, for example, prevent catecholamines from binding to their receptor sites and therefore reduce their effect on the heart (Khan, 2006). Beta-blockers slow the heart rate during sinus rhythm, and are used to slow AV node conduction during atrial arrhythmias such as atrial fibrillation (Camm et al, 2010).

The ECG

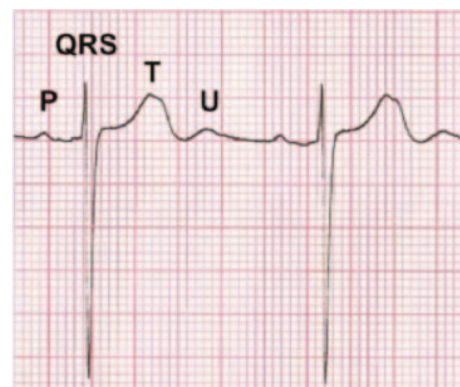The ECG is simply a recording of the heart's electrical activity (Hampton, 2008). This section will provide you with an intro-duction to the waves recorded on the ECG such as the, P-, QRS- and T-wave, which in turn will provide a firm understanding of the ECG as this series progresses.

By placing electrodes on the skin, we can record the electrical activity of the heart and the resulting waveforms. Depending on the type of machine used, and the number of electrodes that are placed, multiple views of the heart's electrical activity can be recorded. Most health-care practitioners will be familiar with the use of 12-lead ECG machines. A 12-lead ECG measures and records 12 different electrical views of the heart (*Figure 5*). In other words, it records the electrical activity as seen from 12 different points around the heart. Lead II, for example, records the electrical activity as seen from the inferior (diaphragmatic) surface of the heart. This lead is often used for rhythm monitoring (Bennett, 2013). Other types of ECG machine include bedside monitors and ambulatory ECG recorders such as Holter monitors. Electrical activity can also be recorded from electrodes inside the body; for example, through pacemaker leads or implantable loop recorders (Diemberger et al, 2015).

Three principal waveforms are recorded by the ECG (*Figure 6*):
- The P-wave
- QRS complex
- T-wave.

The P-wave is created by depolarisation of the atria, the QRS by depolarisation of the ventricles, and the T-wave by repolarisation of the ventricles (Hampton, 2008). In most people, these waveforms occur in a repeating rhythm called sinus rhythm, so called because it originates in the sinus node. In some people, a fourth waveform called a U-wave can be seen. This is usually seen at slower heart rates. The significance

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Key Points

- Contraction of the heart relies on electrical stimulation. Electrical activity is generated by the movement of electrolytes through ion channels in the cell membrane

- Depolarisation is the reversal of the electrical charge across the cell membrane, and triggers contraction of the muscle cells. Repolarisation is the return to resting electrical state. Between these two extremes is a refractory period when it is impossible, or difficult, to depolarise the cell again

- Specialised electrical cells have automaticity and act as the heart's pacemaker. The sinus node is the normal pacemaker, but all parts of the electrical system are capable of this function

- After spreading through the atria, the electrical impulse is delayed in the AV node. This allows the atria to finish emptying, and protects the ventricles from high atrial rates during atrial arrhythmias

- The electrical impulse passes from the AV node into the His-Purkinje system. This is the only electrical connection between the atria and ventricles in the normal heart, and its failure causes heart blocks

- Although the electrical system is self-contained, input from the nervous and endocrine system help to regulate cardiac activity in response to the body's needs

- Cardiac electrical activity can be recorded using skin electrodes, and displayed or printed using a variety of ECG machines. Typical waveforms are recorded; the P-wave, QRS complex and T-wave. Occasionally, U-waves are seen, although their significance remains controversial

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of the U-wave remains uncertain. Some authors think that it represents the late stages of ventricular repolarisation, while others describe it as a post-repolarisation phenomenon (Rautaharju et al, 2009). U-wave abnormalities have been described in various disease states including ischaemic heart disease (Kukla et al, 2014).

The basis of ECG interpretation is formed upon the recognition of abnormalities in the size, shape, pattern and timing of these waveforms (Gregory, 2006). Next month, we will delve into ECG waveforms in more depth, examine the timing of events, and describe sinus rhythm and its variations. We will also consider a system for rhythm interpretation, and discuss issues of accuracy.

To conclude, it is important that anyone wishing to interpret ECGs has a firm physiological foundation to help them understand why ECGs record what they do, as well as enabling them to explore the complexities of ECGs further. By providing readers with some insight into how electrolytes work, how contraction occurs, and the resulting waveforms, it is hoped that they will be in a better position to begin reading and interpreting ECGs with more confidence. **BJCN**

BHF Resources

The British Heart Foundation (BHF) is committed to sharing our knowledge to prevent heart disease devastating people's lives. We've created a range of resources that can be used by health professionals to support people with a heart or circulatory condition. Resources relevant to this article are listed below:

◆ A wealth of online information on a variety of heart conditions, tests and treatments—Find out more at **bhf.org.uk/tests**

◆ **Tests**—A booklet for adults describing the tests that are commonly used to help diagnose heart diseases or assess the current condition of people with a heart condition

◆ **Electrocardiogram**—Your quick guide. A short illustrated leaflet that helps adults understand what to expect if they're having an ECG. It explains the different types of ECG, including Holter monitoring and exercise stress tests

◆ **Your guide to heart tests and treatments**—A series of films following 18 real people's journeys through a variety of cardiac tests and treatments. By seeing the actual procedures, patients know what to expect and how to prepare. Available online at **bhf.org.uk** or on DVD, there are three films covering ECG: *Your guide to ECG*; *Your guide to 24 hour blood pressure and Holter monitoring tests*; and *Your guide to exercise ECG (stress test)*. The DVD version includes subtitles in English, Bengali, Hindi, Gujarati, Punjabi and Urdu, and optional in-vision BSL interpretation

View, download and order these items free at **bhf.org.uk/publications**

The BHF Alliance is a free membership scheme that supports professionals working with people affected by, or at risk of developing, cardiovascular disease.

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