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Atrial fibrillation: part 1

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Aim and intended learning outcomes

The aim of this article is to describe the normal anatomy and physiology of the heart and examine the mechanical and electrical events that can occur as a consequence of atrial fibrillation (AF). A good understanding of the anatomy and physiology of the heart is vital for nurses to appreciate the mechanisms that contribute to the signs and symptoms of this arrhythmia and to provide optimum care for patients. Although AF is a common rhythm abnormality, the complexities of the condition are not always fully appreciated. After reading this article you should be able to:

- Describe what constitutes normal heart function.
- Explain the signs and symptoms patients experience when normal heart function is compromised.
- Describe the link between mechanical, electrical and chemical events in the heart.
- Explain how electrolyte imbalance can influence cardiac function.
- State the normal parameters of the cardiac conduction cycle.
- Describe the techniques used to produce a good quality electrocardiogram (ECG) trace.
- Interpret a rhythm strip using basic principles.
- Describe the physiological mechanisms that can occur in AF.

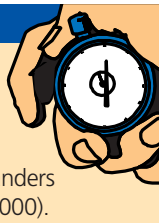
Introduction

Atrial fibrillation (AF) has been a recognised problem for many decades. The overall prevalence of AF is between 1 and 1.5 per cent in the general population, making it the most common sustained arrhythmia (Goodacre and Irons 2002). In those aged over 70 years, the prevalence increases to about 10 per cent (Goodacre and Irons 2002). The enormity of the problem and the fact that AF either complicates or causes many other medical conditions, such as stroke and heart failure, create a significant drain on healthcare resources. AF causes substantial increases in mortality and morbidity (Peters 1998); the mortality rate is double that of patients in sinus rhythm (Task Force Report 2001). It is also thought to account for up to one third of hospital admissions for cardiac rhythm problems (Task Force Report 2001).

Higher levels of mortality and morbidity have been attributable to the increased risk of arterial thromboembolism and ischaemic stroke in patients with AF (Lip *et al* 2002). In the Framingham study (Wolf *et al* 1987) risk of stroke increased with age from 1.5 per cent in the 50-59 year group to almost 25 per cent in the 80-89 year group (Task Force Report 2001). In the presence of other cardiovascular disease, the risk of stroke is estimated to be as high as 75 per cent in patients with AF (Cabin *et al* 1990). With the proportion of older people in the UK increasing, AF and the associated risks will demand even more healthcare resources (Lip *et al* 1996).

Most nurses will be familiar with AF, and have come into contact with patients with AF at some point in their career. Although a common cardiac condition, AF can be a complex and often harmful arrhythmia. Therefore, it is important to understand the normal mechanical and electrical function of the heart to improve knowledge about the characteristics and mechanisms of AF.

TIME OUT 1



Using an anatomy and physiology book of your choice, review the normal functioning of the heart and the cardiac cycle. Suggested textbooks include Scanlon and Sanders (2003), Tortora and Grabowski (2000).

In brief

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Summary
Part 1 of this two-part article on atrial fibrillation – a common cardiac rhythm abnormality – covers the mechanical and electrical functions of the heart. It also looks at cardiac monitoring and rhythm interpretation. Part two, published in next week's *Nursing Standard*, will discuss the predisposing factors, investigations and methods of treatment.

Key words
- Cardiovascular system and disorders
- Heart disorders
- Nursing

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Cardiology

Anatomy and physiology

The heart is a muscular pump about the size of the owner's fist. Positioned in the thoracic cavity between the lungs and immediately above the diaphragm (Marieb 1997), the heart acts as a double pump that serves two circulations: the pulmonary and the systemic. These circulations provide transport of oxygen and nutrients to the cells via the blood and blood vessels and remove metabolic waste products from the cells (Jowett and Thompson 2003). The pulmonary circulation is a low-pressure venous system that transports de-oxygenated blood to the lungs. The systemic circulation is a high-pressure arterial system, which transports oxygenated blood between the heart and the rest of the body. At rest, the heart beats an average of 70 to 80 times per minute (an average of 100,000 times per day), and pumps five litres of blood per minute (approximately 7,000 litres per day) (Scanlon and Sanders 2003).

The heart is composed of four chambers. The upper chambers of the heart are the left and right atria, the lower chambers the left and right ventricles. The atria are smaller and have thinner walls than the ventricles and act as low-pressure reservoirs or storage chambers, whereas the ventricles are high-pressure pumping chambers. A common wall of myocardium, known as the interatrial septum, separates the atria. The left and right ventricles have thicker walls as they have to pump blood against resistance in the pulmonary and systemic systems (Jowett and Thompson 2003). The ventricles are separated by the interventricular septum.

The cardiac cycle is a sequence of events that take place during one heartbeat. The heart contracts (systole) and then relaxes (diastole) in a sequence of volume and pressure changes to complete the cycle. The duration of the cycle is about 0.8 seconds and it occurs at an average heart rate of 75 beats per minute (bpm) (Vickers 1999a). Timings for systole and diastole are comparable at 0.4 seconds, but as heart rate increases the diastolic period decreases. As coronary perfusion occurs during diastole, rapid heart rates may severely impair blood supply to the myocardium (Jowett and Thompson 2003). The volume of blood pumped by the ventricles during each contraction is known as the stroke volume. The amount of blood ejected from the heart per minute is the cardiac output, which is calculated by multiplying the heart rate (bpm) by the stroke volume (ml/beat) (Vickers 1999a).

The action of the four heart valves is important to prevent backflow of blood and ensure that blood flows through the heart in one direction (Linden 2000). Malfunction of the heart valves can occur because of stenosis (narrowing of the valve, which restricts blood flow) or regurgitation (an incompletely closed valve) causing blood to flow backwards. The valves most often affected in the heart are the mitral and aortic. Valvular malfunction can add to the workload of the heart and make it less efficient. Without treatment this extra work can cause cardiac failure (Linden 2000).

The heart wall is another important component in heart function. It consists of three separate layers: the endocardium (inner layer), myocardium (middle layer), and outer epicardium, which is formed from the inner/visceral layer of serous pericardium.

The endocardium, made up of endothelium, lines the chambers and vessels of the heart and also extends to cover the valves (Scanlon and Sanders 2003). This smooth tissue promotes free flow of blood, preventing turbulence, blood clot formation and damage to the vessel wall. The myocardium forms the main bulk of the heart wall and consists of strong muscular tissue. There are two types of cardiac muscle cells: cells that respond to electrical impulses by contracting; and specialised muscle cells that generate and then conduct electrical impulses through the heart muscle (Levick 1995).

TIME OUT 2

Following your review of the heart, describe the generation and conduction of impulses in the heart tissues. Try to identify how this differs from normal muscular tissue.

The myocardium has the ability to depolarise (become electrically charged) and contract without stimulation from the nerves. This intrinsic ability of the heart muscle to generate an impulse spontaneously is known as automaticity or autorhythmicity (Vickers 1999b). The rate of contraction in cardiac muscle is determined by pacemaker cells in the conduction system of the heart. The sinoatrial (SA) node is known as the primary pacemaker as it is faster and more localised and therefore sets the pace for the heart. The autonomic nerves supplying the heart have the capability to alter rate and force of contraction and affect the cells' excitability and speed of conduction (Vickers 1999b).

Parasympathetic fibres (vagus nerve) slow the heart, whereas sympathetic fibres (the flight or fight response mechanism) speed up the heart. Imbalances in blood chemistry – in particular potassium, magnesium, and calcium – can under or over-stimulate cardiac cells, which encourages abnormal heart rhythms or causes the heart to speed up or slow down. Cells in the myocardium are not only connected electrically and mechanically but also chemically (Vickers 1999c). This fusion means that an impulse cannot remain in one place and, therefore,

disseminates throughout the network of cardiac cells.

The myocardium relies on adequate blood supply to fulfil its energy requirements. Without sufficient oxygen, myocardial performance can be severely impaired. The myocardium is thicker in the ventricles, especially the left ventricle, which enables it to contract more forcibly. Ventricular function is dependent on a number of factors to ensure delivery of sufficient output into the systemic circulation. The main determinant of ventricular function is cardiac output, which, as previously mentioned, is the product of heart rate and stroke volume. The autonomic nerves exert control on heart rate through the balance of sympathetic and parasympathetic influence on the sinoatrial node. Stroke volume is determined by the interaction of preload (filling of the heart during diastole), afterload (resistance against which the heart must pump), and contractility of the heart muscle (Timmis *et al* 1997).

As the ventricles fill with blood, the cardiac muscle fibres are stretched and the next contraction occurs. According to Starling's law, an increase in blood volume and subsequent myocardial stretching creates a more forceful contraction, thereby increasing the volume of blood ejected (Starling 1918). However, if the volume of blood increases beyond physiological limits, as in heart failure, the poorly functioning ventricle is unable to stretch further or contract sufficiently to increase the stroke volume. As heart failure progresses, compensatory mechanisms attempt to maintain adequate circulation. These compensatory mechanisms are often inappropriate and cause dilatation of the heart (in response to volume load), impaired myocardial contractility and reduced cardiac output, which embarrass the circulation further (Timmis *et al* 1997). In addition to ventricular dilatation and enlargement, neurohormonal activation is a major compensatory mechanism. Sympathetic nerve stimulation is one of the mechanisms that occur as a result of neurohormonal changes. This leads to increases in heart rate, myocardial contractility and vasoconstriction of blood vessels, which in turn increases preload, afterload and the oxygen requirements of the heart (Julian *et al* 2000).

The pericardium is a tough, protective fibrous sac, which encloses the heart. It has a tough fibrous outer layer that holds the heart in a fixed position and limits its movements in the thorax (Vickers 1999c). The inner serous layer consists of a parietal layer (lining the fibrous pericardium) and a visceral layer (also called the epicardium). The pericardial cavity is situated between the parietal and visceral pericardial membranes and contains 20-30ml of serous fluid. This fluid acts as a lubricant to allow friction-free movement as the heart contracts and relaxes (Vickers 1999c). The heart requires adequate blood supply to function effectively. The purpose of the coronary circulation is to supply blood directly to the myocardium, as oxygen is essential to support the metabolic demands of the heart. Oxygen is provided by the right and left coronary arteries, which are the first branches of the ascending aorta. Approximately 5 per cent (275ml/min) of cardiac output is pumped into the coronary arteries, which fill during diastole (Vickers 1999a). During systole the coronary arteries are compressed causing resistance to blood flow.

The left coronary artery arises from the left posterior sinus of the aorta. The artery divides into two branches: the left anterior descending artery and the circumflex artery. The left anterior descending artery supplies blood to parts of the left and right ventricles, the interventricular septum and apex of the heart. The circumflex artery runs in the atrioventricular groove, supplying blood to the left atrium and lateral wall of the left ventricle (Jowett and Thompson 2003). The right coronary artery arises from the right coronary sinus of the aorta. A small branch off the right coronary artery supplies the SA node. Further branches of this artery supply the conducting tissue, the right ventricle and the inferior surface of the left ventricular wall. The majority of blood reaches the myocardium during diastole. As the heart rate increases, both diastolic and systolic times shorten. Therefore, during increased heart rates the diastolic filling time of the coronary arteries and ventricles is decreased, which can seriously impair the performance of the heart in patients with underlying heart disease (Vickers 1999a). Venous drainage of the heart occurs via the coronary veins forming the coronary sinus, which opens directly into the right atrium.

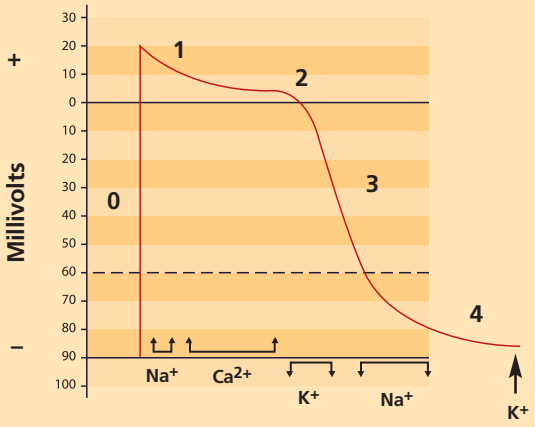
TIME OUT 3

Using a physiology textbook, review the electrolytes that are important in cardiac function and their effects on the cardiac cycle. Describe an action potential.

Cardiac conduction

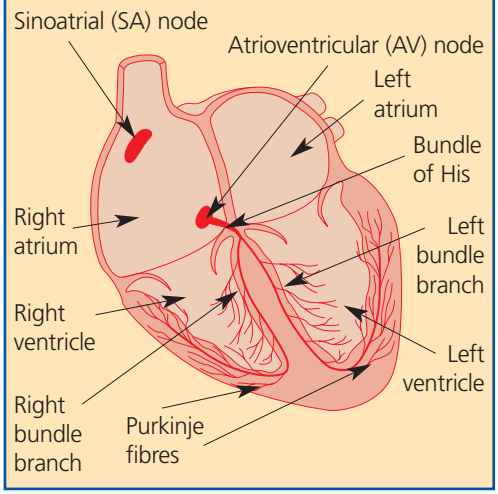
Atrial and ventricular systole and diastole are coordinated by electrical events in the cardiac cycle. The conduction system is made up of specialised muscle cells that initiate and conduct electrical impulses in the heart to produce myocardial contraction (Vickers 1999b). The conduction system of the heart is reliant on an active, energy consuming 'sodium pump'. This metabolic pump regulates the movement of positively charged sodium ($Na+$), calcium ($Ca^{2}+$) and potassium ($K+$) ions (electrolytes) in and out of the cells. As ions move in and out between the interior of the cell and the

Figure 1. Action potential in myocardial cells



Phase 0 = depolarisation (QRS)
Phase 1 = beginning of rapid repolarisation
Phase 2 = absolute refractory period (plateau phase) (ST segment)
Phase 3 = relative refractory period (T wave)
Phase 4 = polarisation (resting state)

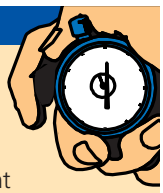
Figure 2. Electrical conduction of the heart



extracellular space, an action potential is generated (Jowett and Thompson 2003). Action potential describes the sequence of depolarisation and repolarisation in cells (Figure 1). During the cells resting (inactive) state the cells are polarised. A membrane potential difference of -90mV exists between the interior of the cell (which is negatively charged) and the extracellular space (phase 4 of the action potential). Depolarisation (phase 0) is a reversal of this electrical charge, with a marked shift in ionic concentrations (RCUK 2000). This corresponds to the QRS complex on the ECG. Positively charged Na+ ions rapidly flow into the cell until a threshold potential of -60mV is achieved. A further rapid influx of Na+ ions, followed by a more sustained flow of Ca^{2}+ ions brings the membrane potential to a slightly more positive value of +20mV. The interior of the cell is then positively charged. This positive state permits an electrical current to flow from one cell to the next, triggering contraction of myocardial cells (Julian *et al* 2000).

Repolarisation (phases 1-3) is the course by which the cell returns to its normal resting state. It occurs over three phases, with an initial slow fall of intracellular charge to +10mV (phase 1). A slow influx of Ca^{2}+ ions creates the plateau phase (phase 2), where the cell remains depolarised, followed by a marked influx of Na+ ions. This corresponds to the ST segment of the ECG. Phase 3 represents repolarisation with rapid outward movement of K+, which corresponds to the T wave on the ECG. Following repolarisation, phase 4 begins where Na+ is actively pumped out and K+ is pumped into the cell, to ensure the cell becomes repolarised. The membrane potential then returns to its resting level of -90mV and the action potential is complete (Jowett and Thompson 2003). The process is then repeated.

TIME OUT 4



Discuss the effects of high and low levels of potassium and calcium on the cardiac cells. Consider how changes in a patient's electrolyte balance might affect his or her cardiac function.

ECGs and the cardiac cycle Recording and reviewing a patient's ECG is a useful way of examining the electrical impulses generated in the heart. Under normal circumstances, depolarisation is initiated by the SA node – a group of specialised pacemaker cells situated high in the right atrium (Figure 2). The SA node has a higher rate of automaticity than other pacemaker cells and, therefore, sets the heart rate. SA node activity does not register electrically on the ECG (Bennett 1997). A wave of depolarisation then spreads from the SA node across the atrial myocardium causing the atria to contract (atrial systole). This creates the P wave on the ECG and lasts 0.06-0.10 seconds (Timmis *et al* 1997) (Figure 3). About 0.1 seconds after the P wave begins, the atria contract. The P wave is absent if atrial depolarisation fails to occur. The impulse then arrives at the atrioventricular (AV) node. A slight delay then occurs as the impulse passes through the AV node. This delay acts as a safety feature to allow atrial emptying. The electrical current travelling through the AV node is too minimal to register on the ECG and is, therefore, recorded as an isoelectric line. The time it takes to travel from the SA node to the ventricular myocardium is represented by the P-R interval on the ECG. This is measured from the beginning of the P wave to the first deflection of the QRS complex and is normally 0.12-0.20 seconds (Timmis *et al* 1997). The electrical impulse is then conducted rapidly by the bundle of His. The bundle of His

divides into left and right bundle branches and ends in the Purkinje fibres, which are firmly buried in the ventricular myocardium.

The QRS complex on the ECG represents ventricular activation (systole). Normal duration of the QRS is between 0.08-0.12 seconds (Timmis *et al* 1997). The ventricles contract shortly after the peak of the R wave. Atrial repolarisation (atrial T wave) cannot be seen as it is hidden in the QRS complex. Following ventricular depolarisation, a short pause is recorded on the ECG as an isoelectric line between the end of the QRS complex and the beginning of the T wave. This corresponds to the ST segment on the ECG. This is the absolute refractory period when no other impulse of whatever strength could be conducted should it fall here. The ST segment has particular relevance in the diagnosis of myocardial infarction and acute coronary syndromes.

The ventricles then repolarise and this corresponds to the T wave on the ECG. This period is the relative refractory period, where the cell has not yet fully recovered. If an impulse occurs at this stage it may be conducted. This is a vulnerable period in the cardiac cycle due to the instability of the cell membrane. Premature ventricular beats occurring at this stage, known as 'R on T' ectopics, can result in ventricular tachycardia or ventricular fibrillation (Bennett 1997).

The QT interval is measured from the beginning of the QRS complex to the end of the T wave and represents the duration of electrical systole. The QT interval is usually 0.35-0.45 seconds if the heart rate is 65-95bpm (Goldman 1979). Prolongation of the QT interval predisposes to ventricular arrhythmias and can occur in response to drugs such as amiodarone (Timmis *et al* 1997).

If a U wave is present it will follow the T wave. The presence of a U wave is not fully understood but may indicate an electrolyte abnormality such as hypokalaemia (Julian *et al* 2000). Theories suggest that it may be the result of slow repolarisation of the intraventricular (Purkinje) conduction system or repolarisation of the papillary muscles (Goldman 1979). Inverted U waves can occur in response to myocardial ischaemia (Timmis *et al* 1997).

TIME OUT 5

Using the diagrams on the cardiac conduction system, PQRST waves and action potential, reflect on how you would explain these electrical and chemical events to a student nurse.

Intrinsic rate Numerous cells in the cardiac conduction system are capable of achieving spontaneous electrical impulse formation. Intrinsic rates vary among the specialised pacing cells. The SA node has the highest intrinsic rate at 60-100bpm. Cells in the AV node have an intrinsic rate of 40-60bpm. The intrinsic rate is slowest in the ventricles, at 30-40bpm (Marriott 1994). The intrinsic ability of specialised cardiac cells becomes especially important when parts of the conduction system fail. If the SA node fails to fire, other areas in the heart usually act as back up pacemakers. The origin of the escape rhythm is indicated by the rate. Ventricular escape rhythms tend to be less reliable as they are much slower than atrial escape rhythms and could stop altogether (RCUK 2000).

Figure 3. ECG trace of a normal cardiac cycle

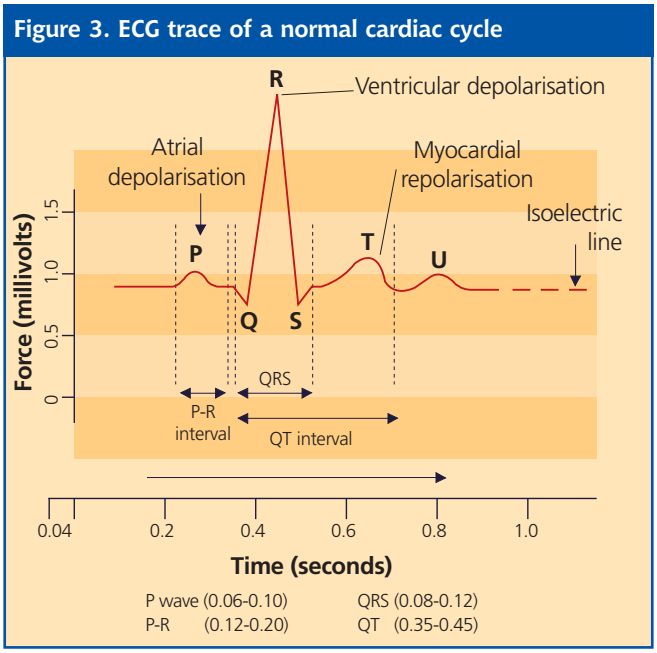
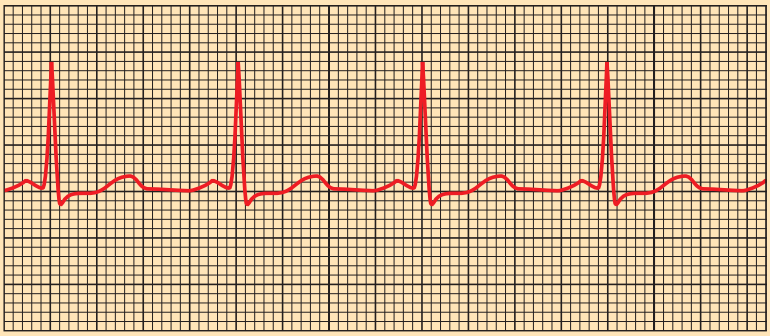


Figure 4. Sinus rhythm



Sinus rhythm Before contemplating other cardiac rhythms, it is useful to appreciate the components of sinus rhythm. The term 'sinus' denotes that the rhythm originates from the SA node as in normal conduction. The heart rate is usually 60-100bpm and the PQRST waves are within normal parameters. The rhythm is regular and a P wave precedes each QRS complex (Figure 4). A heart rate of less than 60bpm is referred to as bradycardia, and tachycardia is a heart rate exceeding 100bpm (RCUK 2000).

Box 1. Six-step approach to rhythm strip interpretation

How is the patient?
Is he or she well or unwell?

- Is there any electrical activity? Check that the leads are connected.
- What is the QRS rate?
- Is the QRS rate regular or irregular? Mark out R-R intervals on paper to confirm.
- Is the QRS normal or prolonged? Normal <0.12 seconds.
- Is there any atrial activity? Check for P waves or f waves.
- Is atrial activity related to ventricular activity? Is the P-R interval the same each time?

(RCUK 2000)

Figure 5. Lead II

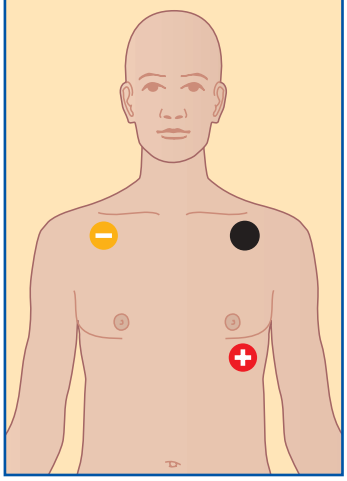
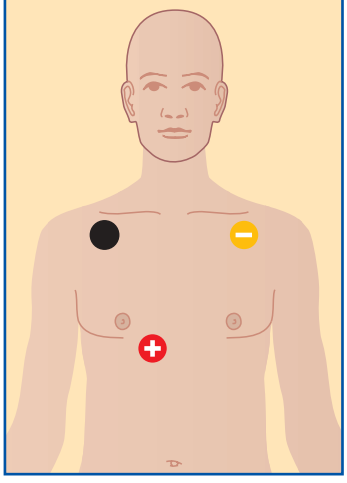


Figure 6. MCL1 (modified chest lead 1)



TIME OUT 6

Reflect on how recordings of the electrical activity of the heart are obtained and saved for interpretation in your clinical area. Before reading on make a recording of an observed rhythm for interpretation. Find out how the patient felt during the recording.

Cardiac monitoring and rhythm interpretation

Cardiac monitoring is a useful way of identifying those patients at risk of developing arrhythmias and for observing those who have confirmed rhythm problems. Correct identification of cardiac rhythms requires some experience. However, through the application of basic principles, the majority of rhythm abnormalities can be interpreted and appropriate management begun. To perform a methodical approach to rhythm interpretation the Resuscitation Council UK (2000) recommend a six-step approach (Box 1). One of the most important points in rhythm analysis is to not waste time attempting to precisely classify a rhythm. If you lack experience it is more important to recognise that the rhythm is not normal and to assess how this abnormal rhythm is compromising the patient (RCUK 2000). If the patient is unwell with the rhythm (Box 2) then more urgency is required in seeking expert help and initiating appropriate treatment. If the patient is well and is maintaining cardiac output, then more time is available to record a 12-lead ECG, interpret the rhythm more closely and instigate the prescribed treatment. It is important to remember to assess the patient and to treat his or her symptoms rather than treating the indications of the ECG trace (RCUK 2000). The same rhythm abnormality can have very different effects on different patients and the same patient may experience different symptoms during the same rhythm abnormality.

It is important to explain to patients the reasons why they are being monitored to allay any fears and anxieties and also to obtain informed consent for this procedure. The recording of an ECG may be a source of anxiety for many patients. Patients may be concerned that the equipment could harm them in some way, or that the need to obtain a recording suggests their condition is deteriorating. Good nursing intervention and sensitive and effective communication with patients and families can help to allay such anxieties.

Most ECG monitoring systems have similar features and incorporate a screen display system, adhesive electrodes that are attached to the patient's chest, and three to five ECG cables that are usually colour coded for ease of use. In a three-lead system the negative electrode (yellow) and the positive electrode (red) receive the electrical current from the cardiac muscle tissue, whereas the neutral electrode (black/green) serves merely to reduce electrical interference and has no influence on the recording. However, this will depend on whether the monitor allows for more than one lead to be selected for monitoring. Five-lead systems tend to be used more often on cardiac units. They are capable of monitoring more views of the heart with a certain degree of accuracy and are used for performing 12-lead ECGs. As with any piece of equipment, it is important that nurses familiarise themselves with the operation of the ECG monitor, for example, how to select leads and set alarms.

Monitors should be placed in clear view of the nurses' station and should be observed by someone who knows how to identify an abnormal rhythm. The optimal lead for monitoring is the lead that displays the best view of PQRST waves – commonly lead II (RCUK 2000) (Figure 5). Modified chest lead 1 (MCL1) can also be used to obtain clear views of the cardiac cycle (Figure 6). The advantage of using this lead is that the electrodes are positioned away from the locations used for the defibrillation paddles in the event of cardiac arrest (RCUK 2000).

Correct preparation of the skin and application of electrodes are essential for optimum ECG monitoring. Electrical interference is reduced if electrodes are applied over bone rather than muscle. Excess body hair should be removed from the site of application to improve electrode adherence. Skin should be wiped with alcohol or rubbed briskly with dry gauze to remove skin oils and debris (Hudak and Gallo 1994). Patients can be sensitive to the electrodes and, therefore, it is important to observe for skin irritation and change the site of the electrodes every two or three days (Hudak and Gallo 1994). Movement artifact can be improved by helping patients to relax, keeping them warm and comfortable, and replacing electrodes to ensure good skin contact, preferably over bone.

TIME OUT 7

Reflect on your experiences of cardiac monitoring and write down any medical conditions that have prompted the monitoring of a patient. Make a list of situations or conditions when cardiac monitoring would be useful and give a brief rationale for each.

Rhythm strip analysis

The rhythm strip print out from cardiac monitors cannot be relied on for the analysis of ST segment changes or advanced ECG interpretation (RCUK

2000). Ideally, if time and the patient's condition allow, a 12-lead ECG should be performed. Unlike the 12-lead ECG, which gives a view of the heart from three dimensions, the rhythm strip gives a view of the heart from one direction only. A good quality ECG recording is imperative to manage rhythm abnormalities appropriately and becomes especially important when giving treatments and analysing the patient's response (RCUK 2000).

Heart rate and regularity can be determined using standard ECG paper. The paper consists of horizontal and vertical lines, each 1mm apart. Horizontal lines denote time and vertical lines the force of contraction. At a standard paper speed of 25mm/second the 1mm square represents 0.04 seconds and the 5mm squares 0.20 seconds. On the ECG paper, 25mm is equivalent to five large squares (or 25 small squares) and represents one second, and 300 large squares represent one minute.

If the rhythm is regular, the rate can be calculated by counting the number of large squares between two consecutive QRS complexes and dividing 300 by this number. If the rate is irregular, count the number of QRS complexes that occur in 30 squares (six seconds of ECG paper) and multiply this number by ten. If the rate is fast it may be difficult to establish whether it is regular or irregular. By marking the tip of the R wave for two or three adjacent beats on a piece of paper and moving it along to another part of the rhythm strip it is possible to check if the R wave intervals match up. If the gap between each R wave interval is the same, the rhythm is regular (RCUK 2000).

TIME OUT 8

Look at the rhythm strips in Figures 7a, b and c. From what you have read so far and using the RCUK (2000) six-step approach to rhythm analysis, interpret the rhythms. For answers, see Box 3 at the end of the article (page 54).

TIME OUT 9

Using an ECG interpretation book and your experience of caring for patients with AF, describe this rhythm in relation to normal cardiac function.

Atrial fibrillation

Electrical events Atrial fibrillation is a supraventricular (occurring above the ventricle) tachyarrhythmia in which the atria quiver, rather than pump in a controlled manner. This results in decreased ventricular filling and a reduction in stroke volume. It is indicated on an ECG trace by a wavy, irregular baseline of 'f' (fibrillation) waves, as opposed to 'P' waves, and is associated with an irregular, often rapid ventricular response (Goodacre and Irons 2002). Figure 8a shows a rhythm strip with an irregular wavy base line between each QRS, no identifiable P waves, with a fast irregular ventricular response. The rhythm strip on Figure 8b shows 'fine' fibrillation waves between each QRS, and no identifiable P waves. QRS is regular (this is unusual in AF as ventricular rate is usually irregular, so this suggests evidence of heart block present with fine AF). The rhythm strip in Figure 8c shows 'coarse' fibrillation waves between each QRS. Note how the 'f' waves in this figure are more prominent than the 'f' waves in Figure 8b. The ventricular rate is irregular, which is classically seen in AF.

These fibrillation waves discharge at a rate of 300-600bpm (Julian *et al* 2000). It has been proposed that AF may be initiated in a similar fashion to ventricular fibrillation (VF), by an ectopic focus falling during the atrial recovery period (Bennett 1997). These rapid oscillations can vary in size, shape and frequency, and may be mistaken for flutter waves

Figure 7a. Rhythm strip

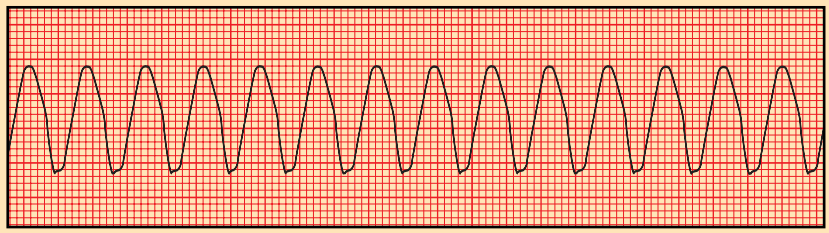


Figure 7b. Rhythm strip

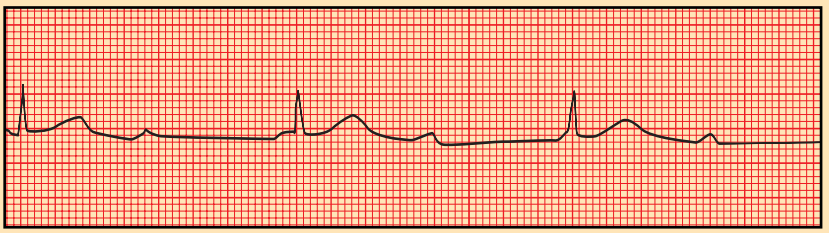
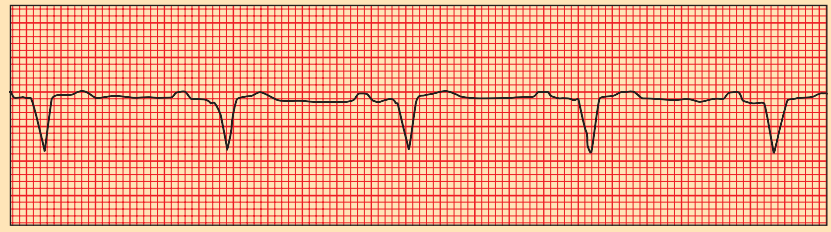


Figure 7c. Rhythm strip



Box 2. Signs and symptoms of the compromised patient

- Systolic blood pressure (BP) <90mmHg. Dependent on the patient's usual resting BP.
- Breathlessness and heart failure. Low oxygen (O^2) saturation.
- Chest pain. Increased workload on heart.
- Heart rate. Fast or slow?
- Reduced conscious level and cardiac output.

(RCUK 2000)

Figure 8a. Atrial fibrillation with rapid ventricular rate

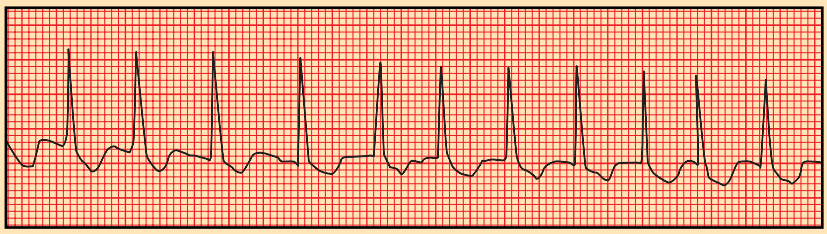


Figure 8b. 'Fine' atrial fibrillation

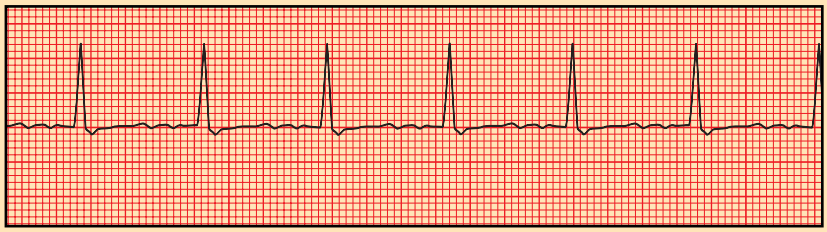


Figure 8c. 'Coarse' atrial fibrillation

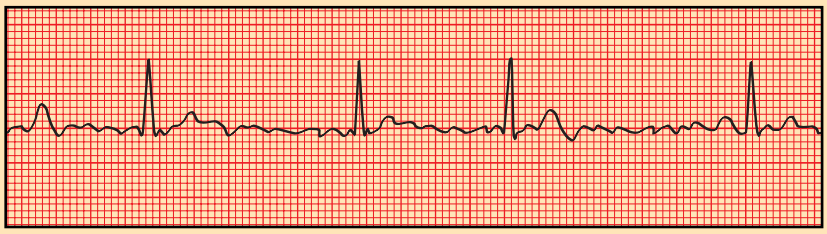
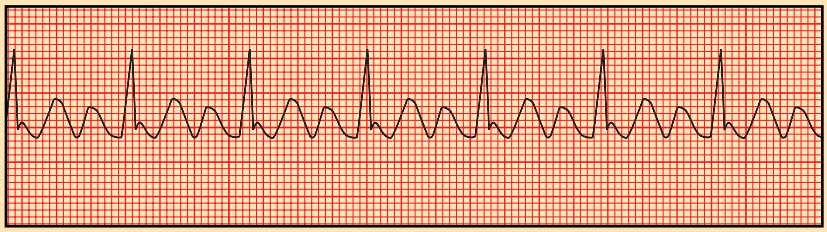


Figure 9. Atrial flutter



(Figure 9) if the amplitude is high (Goodacre and Irons 2002). Fibrillation waves can be referred to as coarse, medium or fine. It has been suggested that the size of 'f' waves may correspond to the size of the atrium – large 'f' waves occur in a large left atrium (Marriott 1994).

The number of impulses conducted from the atria to the ventricles can be variable and is dependent on the function of the AV node, the influence of the autonomic nervous system and the action of drugs (Task Force Report 2001). Generally, only a proportion of impulses are conducted slowly through the AV node. Following conduction of an impulse to the ventricles, the AV node becomes refractory to further stimulation for a short time. Impulses that attempt to penetrate the AV node at this time will fail to activate the ventricles and act as a block to succeeding impulses. This process is termed 'concealed conduction' (Bennett 1997). The result is a totally irregular ventricular response which classically occurs in AF. Ventricular rates rarely exceed 200bpm (Marriott 1994). However, if an accessory pathway – a muscle connection between the atria and the ventricles with capacity to conduct rapidly – is present, atrial impulses can be rapidly conducted, creating ventricular rates of more than 300bpm and leading to ventricular fibrillation (VF) or sudden cardiac death (Timmis *et al* 1997). The Wolff-Parkinson-White (WPW) syndrome is an example of one accessory pathway (Figure 10) and is recognised by a short PR interval (<0.12 seconds) and a delta (∂) wave when the patient is in sinus rhythm.

Regular ventricular rates can occur in AF (Figure 8b and Figure 11) due to the presence of AV block or heart block induced by medication, such as digoxin, verapamil and sotalol hydrochloride (Goodacre and Irons 2002). The QRS complex in AF is usually narrow (<0.12 seconds), but if an impulse arrives at the ventricles early, one of the bundle branches may still be refractory. If the bundle branch is unable to accept another impulse, a form of physiological block occurs. The right bundle branch tends to take longer to recover than the left bundle branch and, therefore, block in the right bundle is more common during fast heart rates (Figure 12). This form of abnormal and delayed conduction (aberrant conduction) through the ventricles produces a widened QRS complex (>0.12 seconds) or right bundle branch block pattern. A widened QRS may also indicate presence of an accessory pathway. An irregular ventricular rate with no visible P waves is diagnostic of AF, even if fibrillation waves are not visible (Bennett 1997).

Mechanical events During episodes of AF, co-ordinated contraction of the atria does not occur, as the fibrillation waves cause the atria to quiver rather than contract properly (Jowett and Thompson 2003). In the presence of AF the atria remain in diastole. Atrial contraction provides up to 10 per cent of total cardiac output (Matsuda *et al* 1983). In AF this loss of atrial systole or atrial 'kick' can compromise the efficiency of the myocardium (Lip *et al* 1996). If the atria fail to contract, blood will not empty properly from the atria into the ventricles. Blood can then pool and become stagnant within the atrial chambers, creating ideal conditions for blood clot formation. A reduction in stroke volume occurs in response to reduced ventricular filling.

Synchrony between the atria and ventricles is lost

during AF and, in combination with the irregular ventricular rate, cardiac output is reduced further (Task Force Report 2001). The intervals between each R wave vary in AF, which causes variable diastolic filling times and, therefore, varying stroke volumes. If the ventricular rate is fast, diastolic time is shortened and, as a result, cardiac output falls. This process can contribute to dilatation of the ventricles (tachycardiac-induced cardiomyopathy), although the exact mechanism is unclear (Packer *et al* 1986).

Mechanisms of atrial fibrillation

The exact mechanism of AF is still unknown. It was originally believed that AF was a completely chaotic event, with disorganised electrical impulses being randomly discharged in the atria (Goodacre and Irons 2002). However, insight into the mechanisms of AF has been enhanced through research and computerised mapping techniques, which have shown that AF can be organised. The most widely accepted theory (Moe and Abildskov 1959) is that it is caused by the presence of multiple circus movements (Figure 13).

Circus movements are described as electrical impulses that become trapped in a circular motion or re-entry of electrical current into muscle that has already been stimulated (Ellis 1998). Unlike most other arrhythmias, AF tends to have several re-entrant circuits. The critical number of circuits required for the perpetuation of AF is six (Goodacre and Irons 2002). These circuits have been located in various sites in the atrial myocardium, most commonly the right atria and superior pulmonary veins (Task Force Report 2001). These multiple circuits cause the atria to quiver/fibrillate at rates exceeding 300bpm (Goodacre and Irons 2002).

Structural abnormalities in the atria, atrial enlargement and dilatation have also been described as features of AF. It is not clear, however, whether these conditions are a cause or a consequence of AF (Task Force Report 2001). AF may be inadvertently triggered by other supraventricular arrhythmias (atrial flutter, atrial tachycardia, AV node re-entry) or premature atrial beats. It is thought that the mechanism is similar to the process of ventricular arrhythmias degenerating into VF (tachycardia-induced tachycardia). If AF is caused by this process, treating the underlying arrhythmia by radiofrequency catheter ablation can terminate the AF (Peters 1998). Atrial fibrillation may also potentially cause AF because of the changes that can occur in the atria as a consequence of AF and if AF episodes are recurrent this can trigger or sustain episodes of AF. The more AF is perpetuated, the more it can become sustained (Peters 1998). A form of electrical remodelling takes place that accommodates the re-entrant circuits, allowing the rhythm to be maintained. This process can occur in as little as 24 hours after onset of AF (Peters 1998).

Although relatively uncommon, AF may be triggered by an imbalance of nervous system control of the heart (Coumel 1989). Vagal AF occurs in response to enhanced parasympathetic tone, which slows the heart rate and shortens the atrial refractory period. AF can also be triggered by excess alcohol intake, as it causes release of adrenaline and

Figure 10. Wolff-Parkinson-White syndrome in sinus rhythm

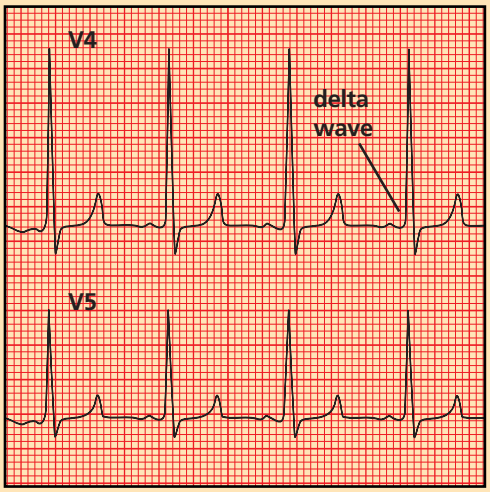


Figure 11. Atrial fibrillation with complete heart block and ventricular ectopic

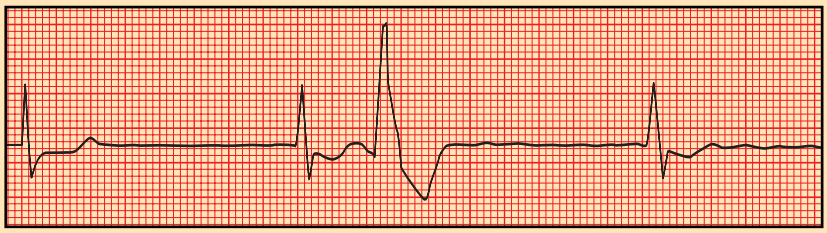
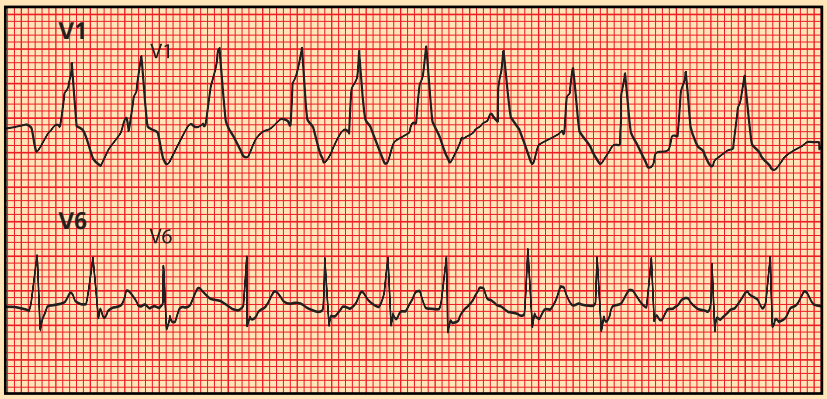


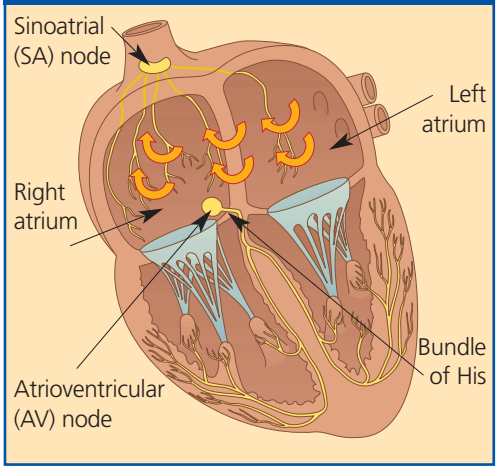
Figure 12. Atrial fibrillation with right bundle branch block (leads V^1 and V^6)



Box 3. Answers to Time Out 8

- a – ventricular tachycardia
- b – third-degree complete heart block
- c – first-degree heart block

Figure 13. Multiple re-entrant wavefronts in AF



noradrenaline. Components of alcohol can also cause slowing of the heartbeat, therefore it can increase as well as decrease the heart rate. The effect of alcohol will be discussed further in part 2 next week. Vagal AF is more common in men aged 40-50 years (Task Force Report 2001) and happens at rest or after eating. Patients tend to experience a gradual slowing of their heart rate before AF starts. Drug treatments such as beta-blockers or digoxin may worsen this form of AF (Task Force Report 2001).

Increases in sympathetic tone cause excess adrenaline to be released, which increases the heart rate and force of myocardial contraction. Unlike vagal AF, this form of AF tends to occur during the day and is often associated with exercise or stress (Task Force Report 2001). Beta-blockers may be helpful in these situations (Task Force Report 2001). The autonomic nervous system can also influence AV node conduction, causing the ventricular rate to increase or decrease.

Conclusion

Although AF is a common arrhythmia it can be complex and may cause many undesirable effects. If nurses understand the physiology and mechanisms of AF in comparison to normal heart function, they will be able to provide a more appropriate assessment and observation of patients. Nurses are in a prime position to notice subtle changes in patients because nursing care is continuous.

It is important that nurses remember to treat the patient, not the heart rhythm. Nurses do not need to be able to diagnose rhythm changes, merely to recognise if a rhythm has changed and to ask for help if they notice a change in a patient. Nurses should not forget the importance of their basic skills of assessment of, for example, pulse, blood pressure and conscious levels, and of asking the patient about his or her symptoms.

This article attempts to give greater insight into how AF may affect heart function and, inevitably, patients' activities of daily living. This should help nurses to manage this arrhythmia more holistically. Part 2 of this article will further discuss the importance of the nurse's role, particularly in relation to the causes, investigations and signs and symptoms of AF. It will also look at the treatments patients might receive

TIME OUT 10

Now that you have completed part 1 of the article, you might like to write a practice profile. Or you could read part 2 next week and base your practice profile on both articles. Guidelines to help you are on page 56.

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