Michael Sampson

Understanding the ECG. Part 10: Pacing, drugs, and electrolytes

British Journal of Cardiac Nursing, June 2016, Vol 11, No 7, p. 376–388.

Introduction

Over the past nine months, this series has examined the most important topics in ECG interpretation. These include cardiac anatomy and physiology, arrhythmias, heart blocks, pre-excitation, the cardiac axis, chamber enlargement, and myocardial ischaemia (Garcia, 2015). We have also discussed many of the key principles of electrocardiography, and emphasised the need for a structured approach to ECG evaluation (Gregory, 2006).

Inevitably in such a large subject, there are topics that we have not touched on. Some of these are less relevant to cardiac nurses, and some more so. In this last instalment of the series, we address three issues that have particular relevance to practitioners caring for cardiac patients. These are

- The recognition of cardiac pacing and pacing problems
- The ECG in the management of cardiovascular drugs
- And the diagnosis of electrolyte disturbances.

As with previous articles in the series, we will place the ECG in the context of clinical practice, and consider important aspects of patient management.

Cardiac pacing

A pacemaker is essentially a device that delivers an electrical stimulus to the heart, resulting in depolarisation of the myocardium, and cardiac contraction (Davies, 2009). Early devices could do little more than this basic function, and were large and unreliable (Ward et al, 2013). Electricity was delivered to the myocardium regardless of intrinsic electrical activity, creating the possibility of competition between native and paced rhythms (Furman, 2003). While this was life-saving treatment in patients with complete heart block, there was considerable potential for complications and pacemaker mediated arrhythmias (Bennett, 2013).

In the modern era, pacemakers are small, reliable, and considerably more sophisticated (Harper & Morris, 2009). They are able to sense and interpret intrinsic electrical activity, and are usually programmed to deliver electrical stimulation only when this is absent, or when the rate of depolarisation is too slow (Davies, 2009). Pacing may be delivered in the atrium, the ventricle or in both chambers sequentially, depending on underlying electrical function (Kalahasty et al, 2014). Complex computer algorithms allow customisation of pacemaker function, and ensure that the

pacemaker responds appropriately to changing physiological conditions. Pacemaker-mediated problems are less common, although they have not disappeared altogether (Wiper et al, 2008).

According to the National Institute for Cardiovascular Outcomes Research (NICOR) and the British Heart Rhythm Society (BHRS), the number of patients receiving a pacemaker has increased year on year, with around 50 000 new systems implanted in England in 2013–14 (Murgatroyd et al, 2014). The management of individuals with pacing systems is highly relevant to cardiac nursing (Davies, 2009) as these patients are cared for by cardiac nurses. Temporary pacing systems are also commonly used on cardiac wards and critical care units, especially in patients recovering from myocardial infarction or cardiac surgery (McNaughton, 2013). The ability to evaluate the ECG in the paced patient, and to recognise common problems, is therefore an important skill for cardiac nurses to have.

Modes of pacing

The mode of pacing used will depend on the indication for pacing, the presence or absence intrinsic electrical activity, and the number of leads placed in the heart (Bennett, 2013). A number of different modes are possible, of which the most commonly used are atrial demand, ventricular demand, and dual chamber pacing. These can be described as AAI, VVI and DDD, using the code devised by the North American Society of Pacing and Electrophysiology (NASPE) and British Pacing and Electrophysiology Group (BPEG) (Bernstein et al, 2002) (*table 1*).

First letter **Chamber paced**	*Second letter* **Chamber sensed**	*Third letter* **Response to sensing**	*Fourth Letter* **Rate modulation**	*Fifth letter* **Multisite pacing**
O = None	O = None	O = None	O = None	O = None
A = Atrium	A = Atrium	T = Triggered	R = Rate modulation	A = Atrium
V = Ventricle	V = Ventricle	I = Inhibited		V = Ventricle
D = Dual (A+V)	D = Dual (A+V)	D = Dual (T+I)		D = Dual (A+V)

Table 1. NASPE / BPEG pacing code (Bernstein et al, 2002)

Atrial demand pacing (AAI)

In atrial demand pacing, an electrical impulse is sent to the right atrium only when the heart rate falls below a level programmed into the pacemaker, typically between 50 and 70 beats per minute (Harper & Morris, 2009). The electrical impulse spreads through the atria, causing them to depolarise and contract, and is then conducted to the ventricles via the AV node and distal conduction system (Bennett, 2013). On the ECG, atrial pacing can be recognised by a small pacing spike in front of the P-wave (Houghton & Gray, 2014) (*figure 1*). The pacing spike is a small ECG

deflection created by the electrical energy delivered by the pacemaker (Garcia, 2015). Provided that the AV node and distal conduction system are normal, the P-wave will be followed by a normal, narrow QRS (Hampton, 2013).

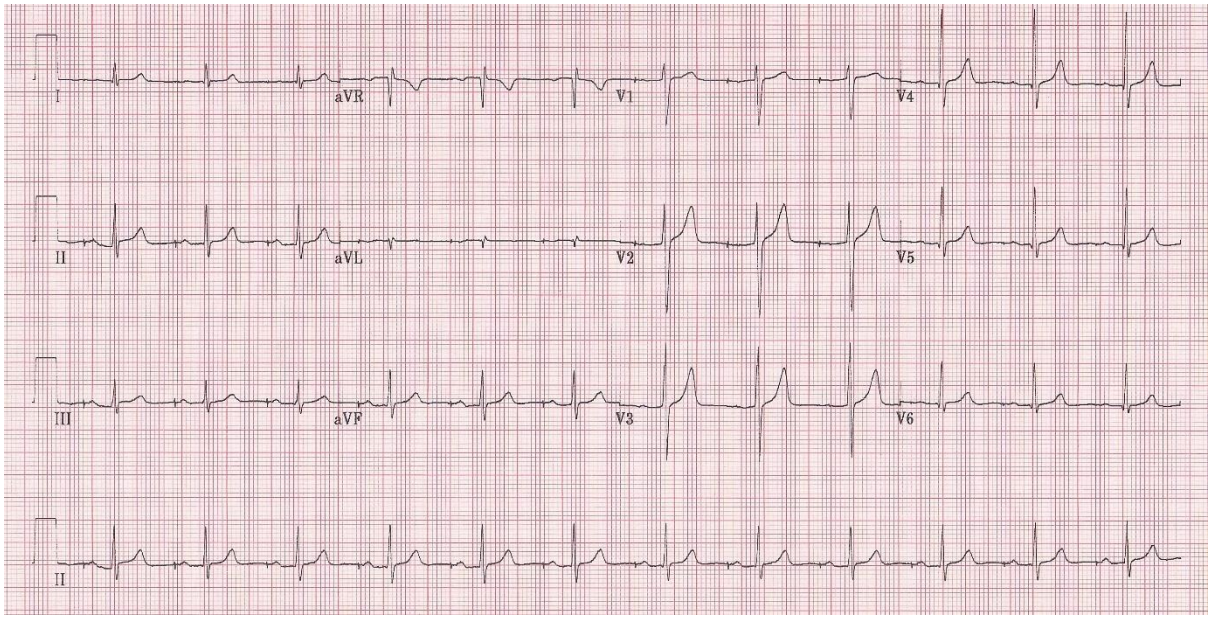

Figure 1. Atrial pacing. Note the pacing spike in front of the P-wave, and the normal QRS complex. In this example, pacing spikes are most obvious in leads II, III, aVL, aVF, V1, V2, but difficult to determine in the other leads. This is a common finding with permanent pacing systems.

The most common indication for atrial demand pacing is sick sinus syndrome (Brignole et al, 2013). In this disease, the sinus node may fail to depolarise intermittently, or fibrous tissue may prevent the impulse from exiting the node (Ewy, 2014). This results in pauses in the sinus rate, which may cause dizziness or loss of consciousness (Jensen et al, 2014). The amount of pacing that an individual receives will depend on how slow their underlying rhythm is, the number of pauses that occur, and the minimum rate set on the pacemaker (Kalahasty et al, 2014). The atrial lead is commonly placed in the right atrial appendage, and may be the only lead placed if AV conduction is normal (Bennett, 2013). The disadvantage of this approach is that ventricular pacing is not possible; if atrioventricular (AV) block develops, the patient will have to undergo re-operation to implant an additional lead. A recent study demonstrated a two-fold risk of re-operation in people receiving an atrial versus a dual chamber system, suggesting that this is not an insignificant risk (Nielsen et al, 2011).

Ventricular demand pacing (VVI)

In VVI pacing, an electrical impulse is sent to the right ventricle, stimulating ventricular contraction if heart rate falls below a pre-set value (Hampton, 2013). On the ECG, this results in a pacing spike in front of the QRS complex (Garcia, 2015). Because conduction through the ventricles bypasses the normal His-Purkinje system, the QRS complex that follows is broad and bizarre, with a discordant T-wave (Houghton & Gray, 2014) (*figure 2*). There is often QT prolongation (Harper & Morris, 2009).

Historically, ventricular leads have been placed in the right ventricular (RV) apex (Hampton, 2013). There is emerging evidence that this can result in ventricular dyssynchrony, and an increased risk of heart failure (Shimony et al, 2012). As a result, there is a trend away from this site, and towards lead placement in the RV outflow tract (RVOT) or interventricular septum (Bennett, 2013). When the pacing lead is in the RV apex, the spread of electricity is from apex to base, and from right to left. This results in an ECG with left axis deviation, and a left bundle branch block (LBBB) type pattern (Harper & Morris, 2009) (*figure 2*). If pacing is from the RVOT, ventricular activation is still right to left, however the impulse sweeps down from base to apex, resulting in right axis deviation but the same LBBB configuration (Bennett, 2013).

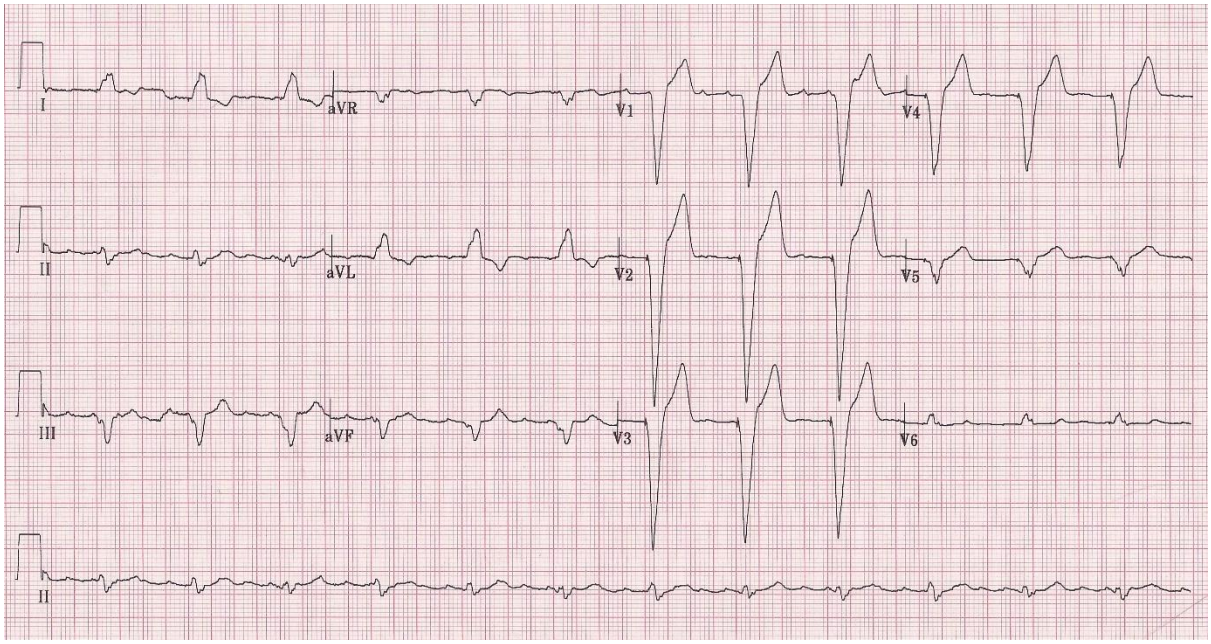

Figure 2. Ventricular pacing. Note the broad QRS with an LBBB configuration. There is left axis deviation, suggesting that the lead is in the RV apex. The underlying rhythm is atrial tachycardia (best seen in lead V1).

In the past, VVI pacing was commonly used in patients with intermittent bradycardia, however its use has declined in recent years (Harper & Morris, 2009). This is largely due to concerns about pacemaker related symptoms. In ventricular pacing, the atria are not stimulated, so they contract out of sequence and their contribution to ventricular filling is lost (Klabunde, 2012). The normal chronotropic response to exercise is also lost, meaning that heart rate does not increase during exertion (Bennett, 2013). These physiological effects reduce cardiac output and exercise capacity, and may cause breathlessness, fatigue and hypotension; this problem is described as 'pacemaker syndrome' (Wiper et al, 2008). In the modern era, the only common indication for VVI pacing is permanent atrial fibrillation; atrial pacing and sensing are ineffective in this condition, leaving VVI as the only choice (Camm et al, 2010).

Dual chamber pacing (DDD)

In most individuals, a dual chamber system is implanted, with leads in both the right atrium and right ventricle (Brignole et al, 2013). This overcomes many of the problems inherent in single chamber pacing; the system can act as an atrial or ventricular pacemaker, or both chambers can be paced sequentially. This ensures that intrinsic rhythm and conduction are used as much as possible, and that when pacing is delivered, normal AV synchrony is maintained (Bennett, 2013). This usually prevents the symptoms associated with pacemaker syndrome, providing that programming is appropriate (Wiper et al, 2008). This type of pacing is described as DDD because both chambers are sensed, both are paced, and the response to sensing can be either inhibition or triggering (Kalahasty et al, 2014).

On the ECG, dual chamber pacing combines the features of atrial and ventricular pacing, provided that both chambers are being paced (Houghton & Gray, 2014). A pacing spike precedes both the P-wave and the QRS complex, and the QRS is broad as previously described (Garcia, 2015) (*figure 3*).

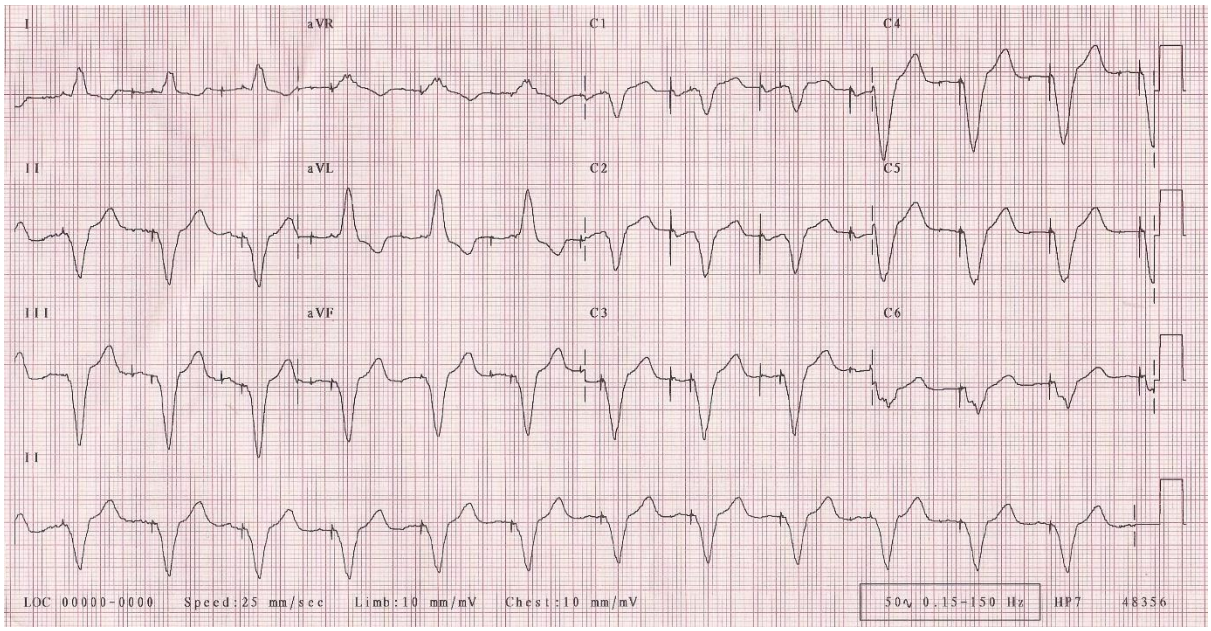

Figure 3. Dual chamber pacing. Note the pacing spikes in front of both the P-wave and QRS complex (not visible in every lead). The P-wave is quite flat, and hard to see in many of the leads.

Pacing problems

From an electrical viewpoint, pacing problems are either sensing-related or pacing-related (Bennett, 2013). They can be further divided into undersensing, oversensing, failure to pace, and failure to capture (Harper & Morris, 2009). These problems can be detected on the ECG, and should be sought during evaluation of the patient, especially if the heart rate is unexpectedly high or low. Problems are more common in temporary systems because lead insertion is often undertaken in emergency conditions, and by less senior clinicians (Bennett, 2013). Temporary leads are also more vulnerable to damage or displacement because they are not designed for permanent fixation in the heart, and are connected to a pacing box that is outside of the body (Jowett & Thompson, 2007).

Undersensing

A pacemaker will only withhold pacing if it detects intrinsic electrical activity. In undersensing, the pacemaker fails to detect this activity, and delivers inappropriate pacing stimuli (Harper & Morris, 2009). This can be detected on the ECG by pacing spikes despite an adequate intrinsic rate (Hampton, 2013) (*figure 4*). These unnecessary pacing spikes may or may not capture the myocardium (i.e. result in depolarisation of the chamber), depending on where they fall within the cardiac cycle (Davies, 2009).

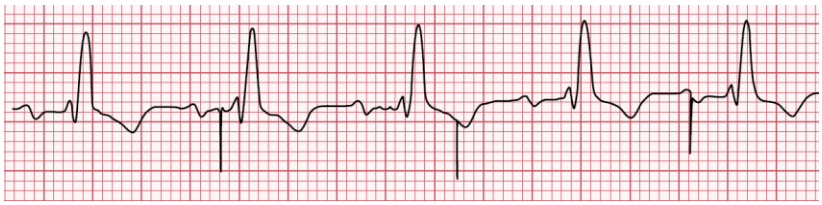

Figure 4. Undersensing. Pacing spikes can be seen despite an adequate intrinsic rhythm; they have not captured the myocardium.

Although undersensing is often a relatively benign occurrence, there is the potential for serious complication (Bennett, 2013). A pacing impulse falling on the latter part of the T-wave can trigger depolarisation during the relative refractory period, at which time the myocardium is only partially repolarised (Fogoros, 2007). The heart is vulnerable to arrhythmia formation at this point; an 'R on T' pacing impulse can initiate a ventricular arrhythmia, potentially causing cardiac arrest (Chemello et al, 2010). The sensing threshold can be adjusted on both permanent and temporary pacing generators; lowering the threshold allows the device to 'see' more electrical activity, and can rectify undersensing (Bennett, 2013). Care must be taken to avoid too low a threshold, as this may result in oversensing.

Oversensing

Oversensing occurs when extraneous electrical activity is sensed by the pacemaker, and incorrectly interpreted as chamber depolarisation (Aehlert, 2011). This can result in inappropriate inhibition of pacing; the pacemaker fails to deliver pacing when it is in fact needed (*figure 5*). If interference is prolonged, severe bradycardia or asystole are possible (Furrer et al, 2004). Causes of oversensing include myopotentials from skeletal muscle contraction, electromagnetic interference, electrical activity from other chambers, and lead damage or failure (Harper & Morris, 2009). Modern implantable devices are shielded from most sources of electromagnetic interference, making this a rare cause of malfunction in permanent systems (Misiri et al, 2012).

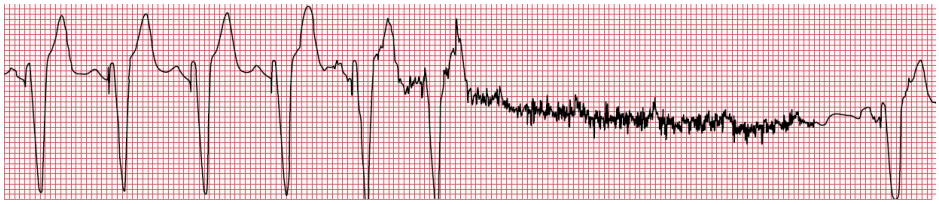

Figure 5. Oversensing. Pacemaker activity has been inhibited by myopotentials from skeletal muscular activity, resulting in a brief period of asystole.

Failure to pace

Failure to pace occurs when the pacing system fails to deliver an electrical stimulus to the heart (Davies, 2009). Causes include power failure as well as disconnection, fracture or displacement of the lead (Aehlert, 2011). Failure to pace can be recognised when the heart rate on the ECG is below the rate set on the pacemaker, but no pacing spikes are visible (Harper & Morris, 2009). In other words, the heart rate is too low, but the pacemaker has failed to respond. Intermittent failure is possible, in which case some pacing spikes will appear as expected, while other will be absent (Davies, 2009) (*figure 6*). Careful evaluation of the system for power supply or connection faults should be undertaken if pacing failure occurs (McNaughton, 2013).

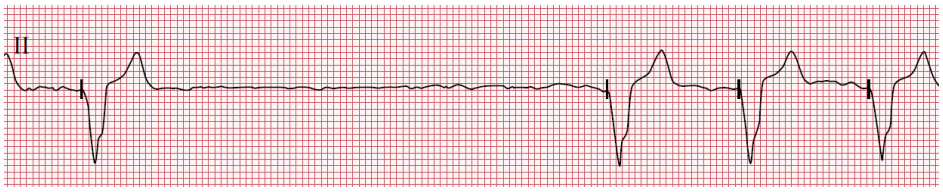

Figure 6. Intermittent failure to pace. There is a single ventricular paced beat, followed by a long pause. Pacing resumes after approximately three seconds. This is a dangerous situation, as the patient appears to have no underlying rhythm; complete pacing failure will result in asystole.

Failure to capture

Capture refers to myocardial depolarisation following a pacing impulse (Houghton & Gray, 2014). Capture fails if the pacing stimulus occurs while the heart tissue is refractory, or if the stimulus is not large enough to trigger depolarisation (Aehlert, 2011). Failure to capture is relatively simple to recognise on the ECG; pacing spikes will be seen as expected, however they will not be followed by P-waves (in atrial pacing) or QRS complexes (in ventricular pacing) (Harper & Morris, 2009) (*figure 7*).

The most common cause of failure to capture is displacement of the pacing lead; the lead may pull back from the heart wall, losing adequate contact, or push forward, perforating the heart wall (Aehlert, 2011; Harper & Morris, 2009). Other causes include battery depletion, device or lead failure, and an increase in stimulation threshold (Aehlert, 2011). Stimulation threshold is the minimum voltage required to capture the myocardium consistently (Bennett, 2013). An increase in threshold can occur over time, especially if oedema or fibrous tissue develop around the lead tip (McNaughton, 2013). Hyperkalaemia, acidosis and drug therapy (for example flecainide) can also increase the threshold (Kalahasty et al, 2014). Increasing the pacing output (the voltage delivered to

the heart) so that it exceeds the stimulation threshold will restore capture in this situation (Aehlert, 2011). The increased output should include a safety margin; it is common practice to set the output at several times the threshold on temporary pacing systems (McNaughton, 2013).

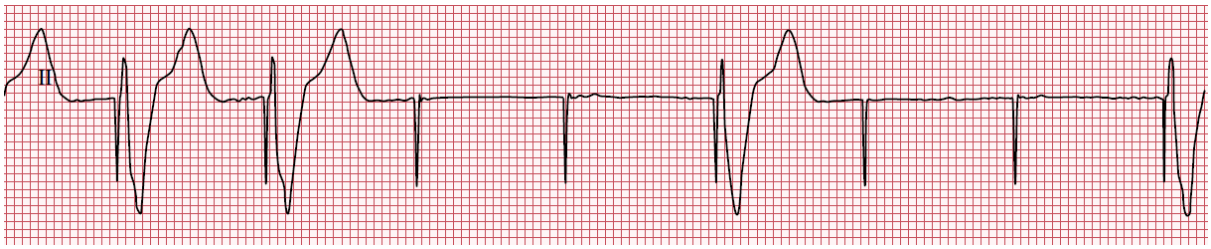

Figure 7. Failure to capture. There are regular pacing spikes, however only the first, second, fifth and eighth are followed by QRS complexes. The remaining pacing impulses have failed to capture the myocardium.

Management of pacing problems

The management of pacing problems depends to some degree on the type of system. Permanent pacemakers can be interrogated by placing a sensor over the pacing generator, attached to a portable computer (Bennett, 2013). Sensing and pacing thresholds can be checked and adjusted, and lead impedance measured (Kalahasty et al, 2014). Pacing function can be evaluated from current performance, as well as from information stored in the memory of the device (Harper & Morris, 2009).

Temporary systems lack this sophistication, and the investigation of problems relies on evaluation of the patient, ECG, and device settings (McNaughton, 2013). Power supply and lead connections should be checked, alongside sensing and pacing parameters (Aehlert, 2011). Unless a simple problem is found, for example a loose connection, expert help should be sought from a cardiologist. If lead displacement is suspected, chest x-ray is useful in determining lead position; posterior-anterior and lateral chest films are typically needed (Torres-Ayala et al, 2014). Lead repositioning or replacement may be necessary, and may be urgent if the patient is compromised, or if there is perforation of the heart (Seegers et al, 2009).

Cardiovascular drugs

Some cardiovascular drugs have little or no direct effect on cardiac electrical activity (Bunker, 2014). Others, however, have profound effects on sinus rate, conduction speed through the heart, and the repolarisation of myocytes (Fogoros, 2007). These electrical changes are reflected in the ECG, and are useful in guiding drug therapy (Garcia, 2015). Understanding how drugs influence the ECG allows practitioners to predict how safe it is to prescribe or administer these medications (Opie & Gersh, 2013). It also ensures that drug effects are not misconstrued; this is especially important when they alter the ST-segment or T-wave, mimicking the effects of ischaemia (Houghton & Gray, 2014). Among the most commonly used cardiovascular drugs affecting the ECG are beta-blockers, digoxin, flecainide, sotalol and amiodarone (Bennett, 2013).

Beta-blockers

Beta-blockers are among the most widely used drugs in the treatment of cardiovascular disease (Khan, 2006). They bind to beta-adrenergic receptors on multiple organs including the heart, attenuating the effects of the sympathetic nervous system and circulating catecholamines (Fogoros, 2007). This makes them useful in conditions such as angina, chronic heart failure, and hypertension, where a reduction in sympathetic stimulation is desirable (Joint Formulary Committee, 2016). They also reduce the ventricular response during atrial arrhythmias, making them the first choice in the management of atrial fibrillation (AF) (Camm et al, 2010).

Beta-blockers slow the rate of sinus node discharge, as well as conduction speed through the AV node (Opie & Gersh, 2013). During sinus rhythm, this may result in sinus bradycardia and an increase in the PR interval (Bennett, 2013). During AF, the ventricular rate is decreased both at rest and during exercise (Lafuente-Lafuente et al, 2009). The effect of beta-blockers on heart rate can be hard to predict; in some individuals it may be minimal despite large doses, while in others even small doses can result in profound bradycardia (Erdmann, 2009). This is especially so in individuals with latent conduction system disease, in whom beta-blockers may cause asystolic pauses or AV block (Volgler et al, 2012) (*figure 8*). The ECG is a useful tool in evaluating this response, although an ambulatory recording (e.g. a 24-hour Holter monitor) is often necessary to uncover the true extent of any drug effect (Bennett, 2013). Drug withdrawal may be necessary if there is extreme bradycardia, long pauses, or high level AV block. If stopping the drug is undesirable, a pacemaker can be implanted to permit ongoing drug treatment (Camm et al, 2010).

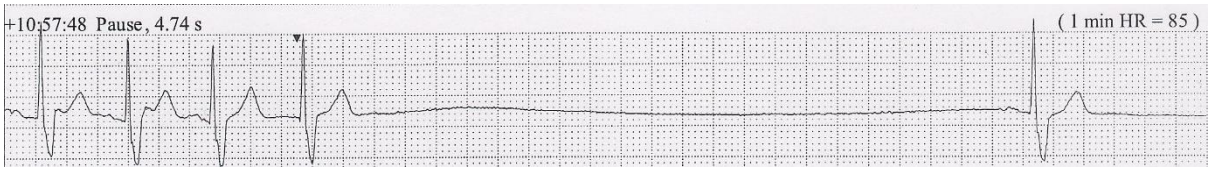

Figure 8. An asystolic pause of almost five seconds in a patient taking bisoprolol for paroxysmal atrial fibrillation; the patient reported dizzy spells, but had not lost consciousness.

Digoxin

The principal use of digoxin is to control the heart rate during atrial fibrillation; the drug is also used as an adjunct in chronic heart failure (Camm et al, 2010; McMurray et al, 2012). Digoxin binds to the sodium-potassium pump within the cell membrane, causing increases in intracellular sodium and calcium (Chung, 2006). This results in two significant effects. Firstly, parasympathetic stimulation to the heart is increased, slowing sinus rate and AV conduction (Opie & Gersh, 2013). Secondly, the increase in calcium availability within myocardial cells results in a mild, positive inotropic effect (Fogoros, 2007).

On the ECG, the effects of digoxin are similar to those of beta-blockers (Bennett, 2013). In sinus rhythm, heart rate falls and the PR interval may increase. During AF, ventricular response is slowed, although this effect is seen only at rest (Panchmatia, 2010). The failure of digoxin to control the heart rate during exercise explains why the drug is not recommended as monotherapy in AF, except in sedentary individuals (National Institute of Health and Care Excellence (NICE), 2014). As with beta-blockers, patients should be monitored for profound bradycardia, pauses and AV block (Joint

Formulary Committee, 2016). Digoxin at therapeutic doses also causes changes to the ST-segment and T-wave, especially in the lateral leads (Houghton & Gray, 2014). Down-sloping ST-segment depression commonly occurs, in association with T wave inversion (*figure 9*). This may be difficult to distinguish from other causes of repolarisation abnormality, for example ischaemia, however the presence of AF on the ECG, and a current prescription for digoxin, strongly suggests a drug effect (Hampton, 2013).

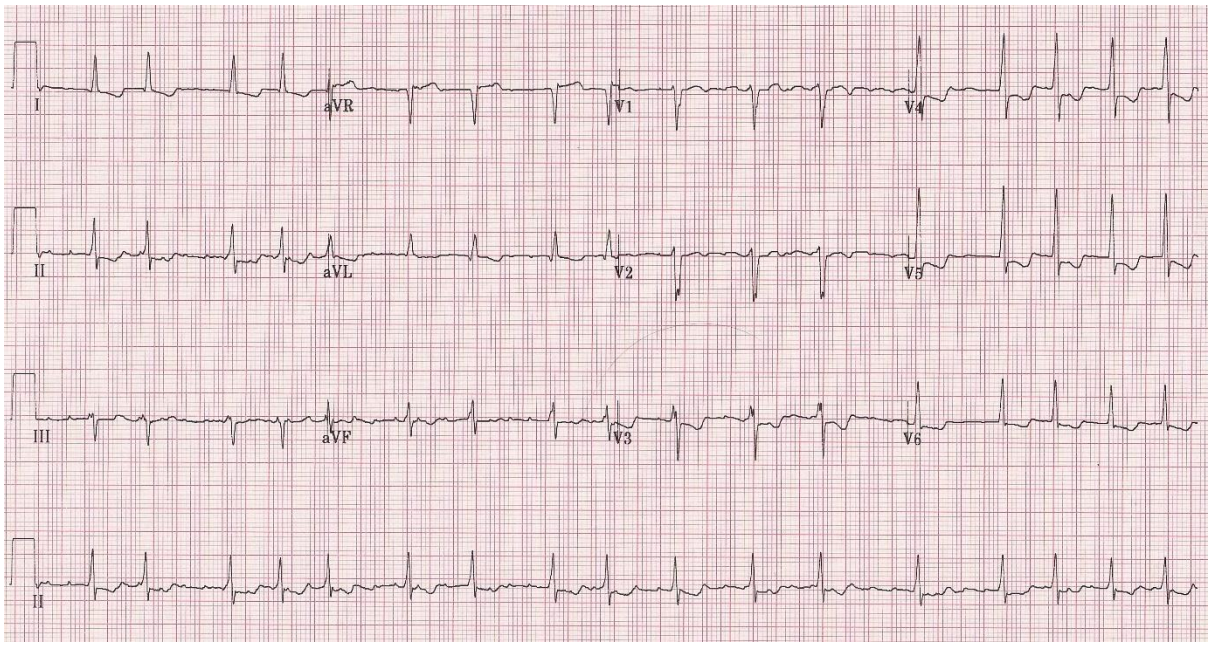

Figure 9. Digoxin effect. There is widespread down-sloping ST depression and T-wave inversion, most obviously in leads V3-V6. Note that the rhythm is atrial fibrillation; the patient had been taking digoxin for several months.

Flecainide

Flecainide is a class IC antiarrhythmic drug, used in the management of both supraventricular and ventricular arrhythmias (Joint Formulary Committee, 2016). Its most common use is the management of paroxysmal atrial fibrillation (PAF); orally, it reduces the frequency of attacks, while an acute episode can be terminated by an infusion of the drug (Opie & Gersh, 2013). Flecainide works by blocking sodium channels within cardiac myocytes; this slows the speed of depolarisation, and therefore conduction of electrical activity through the myocardium (Bennett, 2013). On the ECG, flecainide may prolong the QRS and PR intervals, although it has little effect on the QT interval or heart rate (Fogoros, 2007). The effect on the ECG is dose-dependent; at higher doses, greater prolongation of conduction intervals occurs.

Like all antiarrhythmic drugs, flecainide is capable of pro-arrhythmia. This was demonstrated most clearly in the Cardiac Arrhythmia Suppression Trial (CAST), in which there was higher mortality in heart attack survivors taking the drug compared to placebo (CAST Investigators, 1989). The mechanism of death was thought to be ventricular arrhythmia provoked by the drug. This finding has limited our modern use of the drug to patients without structural heart disease, in whom the drug is relatively safe (Aliot et al, 2011). Flecainide can exacerbate conduction disorders, and

precipitate asystolic pauses and AV blocks (Joint Formulary Committee, 2016). If these occur, or there is extreme widening of the QRS or PR intervals, the drug may need to be stopped.

Sotalol

Although sotalol is a beta-blocker it also has class III antiarrhythmic properties, meaning that it prolongs cardiac repolarisation (Joint Formulary Committee, 2016). It does this by blocking potassium channels within cardiac tissues (Fogoros, 2007). Like flecainide, sotalol is widely used in the management of PAF, although it can also be used for other supraventricular or ventricular arrhythmias (Camm et al, 2010). On the ECG, the usual effects of beta-blockers on heart rate and AV conduction are observed (Opie & Gersh, 2013). In addition, there may a significant increase in the QT interval, reflecting the prolongation of repolarisation (Hampton, 2013) (*figure 10*). This results in an increased risk of torsade de pointes, with the level of risk directly proportional to the length of the QT interval (Nachimuthu et al, 2012). If the QTc reaches 500ms, the drug should be stopped (Bennett, 2013).

As well as the length of the QT interval, several other factors should be considered when using sotalol. Firstly, the risk of torsade de pointes is further increased by hypokalaemia; care should be taken when using potassium wasting diuretics (Opie & Gersh, 2013). Secondly, sotalol is excreted primarily by the kidney, and will accumulate if renal function is significantly reduced (Joint Formulary Committee, 2016). The drug should therefore be avoided in patients with renal impairment, or the dose reduced. Finally, although sotalol can be used with relative safety in patients with normal hearts or uncomplicated coronary artery disease, it should be avoided in patients with structurally abnormal hearts (Bennett, 2013). This recommendation stems from the SWORD trial, which demonstrated increased mortality in individuals with left ventricular systolic dysfunction who were taking sotalol (Waldo et al, 1996).

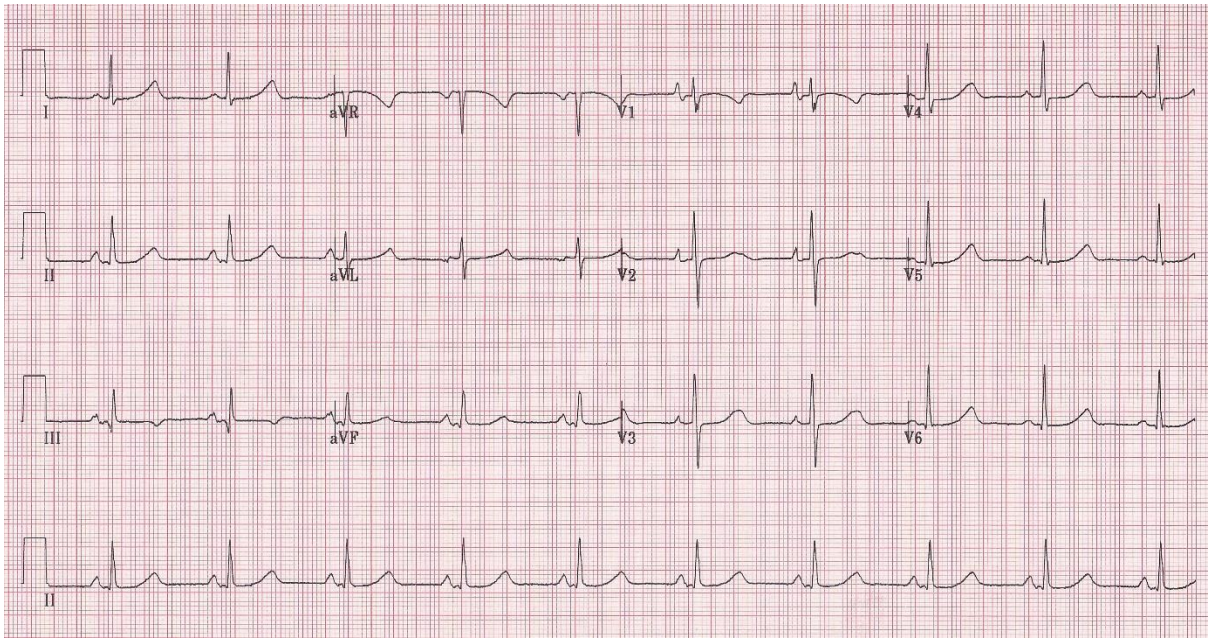

Figure 10. QT prolongation in a patient taking sotalol. The QTc calculated by the ECG machine was 506ms; the drug was stopped.

Amiodarone

Amiodarone is also considered a class III antiarrhythmic drug, because its principal effect is to prolong repolarisation by blocking potassium channels (O'Donovan, 2006). Uniquely, however, it also blocks sodium and calcium channels, and exerts a degree of beta blockade (Fogoros, 2007). It therefore has properties from of all of the major antiarrhythmic drug groups. This makes it a very versatile drug in clinical practice; it is highly effective for both atrial and ventricular arrhythmias, has little proarrhythmic potential, and can be used in individuals with structurally abnormal hearts (Opie & Gersh, 2013). Unfortunately, this impressive performance comes at a price; amiodarone is associated with serious complications including liver, thyroid and lung toxicity (Siddoway, 2003). These problems occur most often during prolonged treatment; long term use is therefore limited to dangerous arrhythmias such as ventricular tachycardia, or to arrhythmias resistant to other drugs (Bennett, 2013).

Amiodarone slows the heart rate during sinus rhythm as well as during atrial arrhythmias (Garcia, 2015). It may also prolong the PR, QRS and QT intervals. Because of the long loading period needed to achieve therapeutic levels of the drug, ECG effects typically develop gradually over a period of months (Fogoros, 2007). Although bradycardia and AV block can be problematic, QT prolongation is rarely an issue as, unlike sotalol, amiodarone is not associated with a significant risk of torsade de pointes (Siddoway, 2003). In addition to a regular ECG, patients taking amiodarone should undergo regular blood testing for liver and thyroid function, as well as evaluation of lung function (Dixon et al, 2013).

Electrolyte disturbance and the ECG

The contraction of cardiac myocytes depends on electrical activity created by the movement of electrolytes across the cell membrane (Grant, 2009). Normal levels of potassium, sodium and calcium are important in maintaining normal function; abnormalities can result in arrhythmias, cardiac dysfunction or death (Kaplana & Kellum, 2010). The role of the ECG in detecting electrolyte imbalance is limited; changes in serum sodium have little effect on the ECG, while hypokalaemia, hypercalcemia, and hypocalcaemia result in minor and non-specific ECG changes (Garcia, 2015). These are described in *table 2*. The most dangerous electrolyte problem, hyperkalaemia, does result in significant electrical changes, making the ECG an important tool in its detection and management (Alfonzo et al, 2014).

Electrolyte imbalance	Possible ECG findings	Notes
Hypokalaemia	• Mild ST-segment depression • Mild decrease in T-wave amplitude • Minimal prolongation of the QRS complex • Prominent U-wave	ECG changes are usually seen only in severe hypokalaemia, e.g. below 2.7mmol/l (Slovis & Jenkins, 2009). Hypokalaemia encourages the development of ectopic beats, and can contribute to the development of arrhythmias such as AF and flutter
Hypercalcemia	• Shortened QT interval owing to ST segment shortening	
Hypocalcaemia	• QT prolongation caused by lengthening of the ST segment	

Table 2. ECG findings in hypokalaemia and calcium disturbance

Hyperkalaemia

In a normal individual, serum potassium is kept between 3.5 and 5.0mmol/l (Klabunde, 2012). Blood levels are regulated by the kidneys, with excess potassium excreted in the urine (Marieb & Hoehn, 2015). Hyperkalaemia, defined as a serum potassium of 5.5mmol/l or more, occurs when renal excretion is inadequate, and may be exacerbated by excessive intake of potassium in the diet or the release of intracellular potassium into the bloodstream (Alfonzo et al, 2014). Renal failure is the most common cause of hyperkalaemia; others contributory factors include drugs that cause potassium accumulation, for example ACE inhibitors, the use of potassium supplements, acidaemia, and cell damage due to burns or crush injuries (Aehlert, 2011). Hyperkalaemia can be classified as mild, moderate or severe (Soar et al, 2010) (*table 3*).

Level	Serum potassium
Mild	5.5 – 5.9mmol/l
Moderate	6 – 6.4mmol/l
Severe	≥6.5mmol/l

Table 3. Levels of hyperkalaemia (Soar et al, 2010)

On the ECG, progressive change occurs as serum potassium rises; this makes the ECG a useful tool in the detection of hyperkalaemia, and in predicting its severity (Alfonzo et al, 2014). The first change, seen in mild to moderate hyperkalaemia, is an alteration in the size and shape of the T-waves (Houghton & Gray, 2014). On a normal ECG, T-waves are asymmetrical and their height does not exceed 6mm in the limb leads, and 12mm in the precordials (Garcia, 2015). In hyperkalaemia, the T-waves become taller, and may exceed the height of the R-wave (Slovis & Jenkins, 2009). This gives them a peaked or tented appearance (Hampton, 2013). In one in five individuals, the T-waves also develop a narrow, symmetrical shape which contrasts shapely with their usual width and asymmetry (Garcia, 2015). T-wave changes can occur in any ECG lead, but most are most commonly seen in II, III and V2 to V4 (Slovis & Jenkins, 2009). Care must be taken to exclude differentials, which include

the hyperacute T-waves of ST elevation myocardial infarction, raised intracranial pressure, and normal variant (Houghton & Gray, 2014). If hyperkalaemia is suspected, immediate assessment of serum potassium using arterial or venous blood gas analysis is recommended (Guthrie, 2010).

As serum potassium climbs above 6.5mmol/l, the PR interval prolongs, and the P-wave progressively flattens and widens until it disappears altogether (Garcia, 2015) (*figure 11*). The QRS starts to widen, and gradually merges with the T-wave, until a rhythm resembling a sine-wave is seen (Garcia, 2015) (*figure 12*). This is a pre-terminal finding, leading rapidly to asystole, ventricular fibrillation, or a wide, pulseless idioventricular rhythm (Slovis & Jenkins, 2009). ECG progression can be rapid, and even small rises above 6.5mmol/l can result in rapid deterioration from tall T-waves to cardiac arrest (Alfonzo et al, 2014). Urgent action to prevent clinical deterioration is therefore paramount; initial treatment can be divided into three initial stages

- Stabilisation of cardiac electrical activity
- The movement of potassium out of the blood and into the cells
- The removal of excess potassium from the body

Methods to achieve these three steps are considered in *table 4*, and should be complemented by continuous monitoring of ECG and vital signs, regular assessment of serum potassium (at least hourly initially), and frequent blood glucose sampling if insulin and glucose are given (Alfonzo et al, 2014). Once treatment has been initiated, steps to prevent recurrence should be taken, for example managing renal function and reviewing the drug chart for medications that may be contributory (Houghton & Gray, 2014).

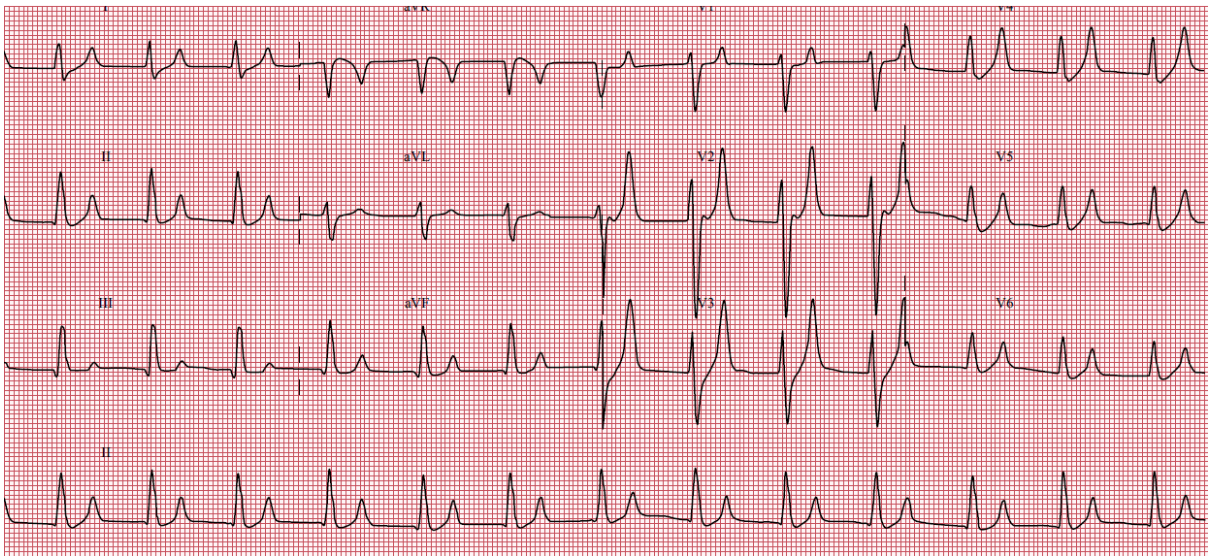

Figure 11. ECG from a patient with a serum potassium of 7.1mmol/l. T-waves in lead II and the precordials are tall, narrow, symmetrical and peaked. P-waves have disappeared, and the QRS is starting to widen. Urgent action to stabilise the heart, and to correct serum potassium, is required.

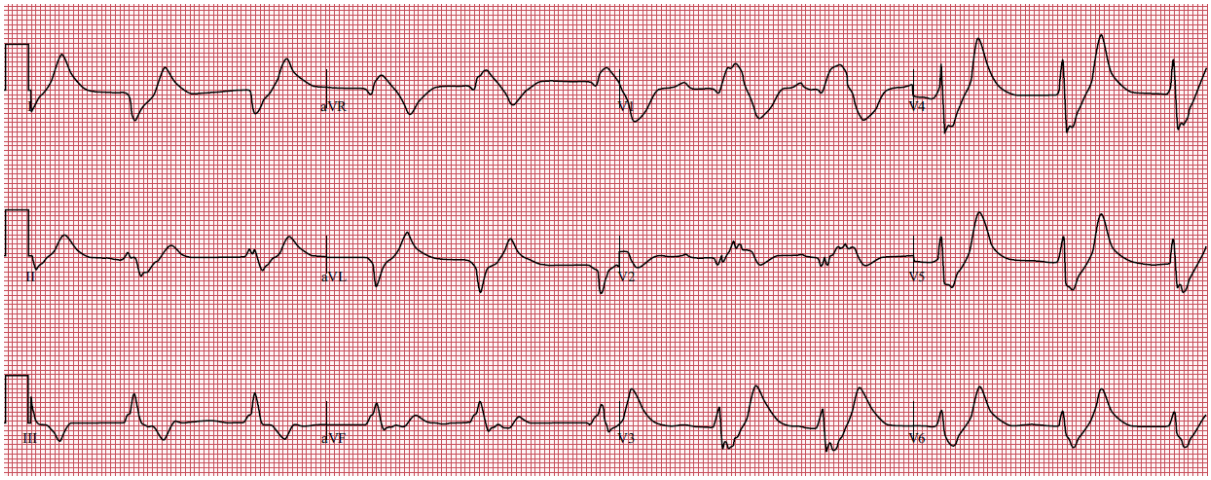

Figure 12. ECG from a patient with a serum potassium of 9mmol/l. The QRS has broadened and merged with the T-wave, creating a pattern resembling a sine-wave. This is an ominous finding; cardiac arrest is likely without rapid intervention.

Step and purpose	**Treatment**	**Notes**
1. Stabilise cardiac electrical function	Intravenous calcium chloride or calcium gluconate	10-20ml of calcium gluconate 10% by slow injection is commonly used.
2. Shift potassium into the cells	5-10 units of fast acting insulin (e.g. Actrapid) given with 50ml of 50% glucose over 5-10 mins 10-20mg of salbutamol given by nebuliser Sodium bicarbonate may be useful if there is acidosis	Blood glucose should be checked at 0, 15, 30, 60, 90, 120, 180, 240, 300 and 360 minutes after insulin administration, and then as required. Salbutamol should only be used in additional to, not instead of, insulin and glucose. Sodium bicarbonate is not recommended for routine use
3. Remove potassium from the body	Ion exchange resins (e.g. calcium resonium) Haemodialysis	Treatment will depend on the cause of hyperkalaemia, as well as its severity. Ion exchange resins are only indicated in mild to moderate hyperkalaemia.

Table 4. Management of hyperkalaemia (source: Alfonzo et al 2014; Joint Formulary Committee, 2016)

Conclusion

Pacing, cardiovascular drugs, and electrolyte disturbance produce characteristic ECG features that can alert practitioners to potential problems, and guide patient management (Houghton & Gray, 2014). In the paced patient, careful evaluation of the relationship between pacing spikes and the P-waves or QRS complexes allows the ECG reader to determine whether the atrium or ventricle is being paced, or both chambers sequentially (Davies, 2009). Pacing problems such as undersensing or failure to capture can be determined by studying this same relationship (Aehlert, 2011). The detection of pacing problems should trigger an evaluation of the system for faults, with the involvement of a cardiologist as necessary.

The ECG is also useful in evaluating the effect of drugs on cardiac electrical function. Effects include a decrease in heart rate, widening of the PR interval or QRS duration, as well as prolongation of the QT interval (Fogoros, 2007). Profound bradycardia, asystolic pauses, and AV blocks can develop, and should be sought using ambulatory monitoring if necessary. In drugs that prolong repolarisation, especially sotalol, careful evaluation of the QT interval is necessary because of the risk of torsade de pointes; if the QTc exceeds 500ms, drug discontinuation is recommended (Bennett, 2013).

Finally, most electrolyte disturbances cause minimal and non-specific change on the ECG (Garcia, 2015). The exception is hyperkalaemia, which results in progressive change including tall, peaked T-waves, loss of the P-wave, and QRS widening (Slovis & Jenkins, 2009). This dangerous condition can rapidly result in cardiac arrest, especially when serum potassium rises above 6.5mmol/l (Alfonzo et al, 2014). Urgent action to stabilise cardiac electrical activity, and reduce serum potassium level is essential (Joint Formulary Committee, 2016).

Key points

- Pacing can be recognised by the presence of pacing spikes in front of the P-wave or QRS complex. Ventricular pacing produces a QRS complex that is broad, and has a left bundle branch block configuration.
- Problems with pacing include under- and over-sensing, as well as failure to pace or capture. Examination of the ECG for the presence of pacing spikes, and their relationship to the P-waves and QRS complexes permits diagnosis of these problems. Simple measures to correct problems in temporary systems include checking generator settings, power supply, and lead connections.
- Cardiovascular drugs that affect the ECG include beta-blockers, digoxin, flecainide, sotalol and amiodarone. Changes include slowing of the heart rate, widening of the PR interval or QRS duration, and prolongation of the QT interval.
- All rate slowing and antiarrhythmic drugs can cause profound bradycardia, asystolic pauses, and AV blocks. Sotalol and amiodarone prolong the QT interval, which increases the risk of torsade de pointes. Sotalol is especially dangerous in this respect; if the QTc exceeds 500ms the drug should be stopped.
- Most electrolyte disturbances cause minimal change to the ECG. The exception is hyperkalaemia, in which tall, peaked T-waves progress to loss of the P-wave, and QRS widening as serum potassium rises. Urgent action to prevent cardiac arrest is required; cardiac electrical activity must be stabilised and serum potassium level reduced.

References

Aehlert B (2011) *ECGs made easy*, fourth edition, Maryland Heights: Mosby Elsevier.

Alfonzo A, Soar J, MacTier R et al (2014) *Treatment of acute hyperkalaemia in adults,* clinical practice guideline from the UK Renal Association, available at http://www.renal.org/docs/default-source/guidelines-resources/joint-guidelines/treatment-of-acute-hyperkalaemia-in-adults/hyperkalaemia-guideline---march-2014.pdf?sfvrsn=2 [accessed 07/07/2016].

Aliot E, Capucci A, Crijns HJ, Goette A, Tamargo J (2011) Twenty-five years in the making: flecainide is safe and effective for the management of atrial fibrillation, *Europace*, 13(2):161-73.

Bennett DH (2013) *Bennett's Cardiac Arrhythmias: Practical notes on interpretation and treatment*, 8th edition, London: Hodder Arnold.

Bernstein AD, Daubert JC, Fletcher RD et al (2002) The Revised NASPE/BPEG Generic Code for Antibradycardia, Adaptive-Rate, and Multisite Pacing, *Pacing and Clinical Electrophysiology*, 25(2):260-4.

Brignole M, Auricchio A, Baron-Esquivias et al (2013) 2013 ESC guidelines on cardiac pacing and cardiac resynchronisation therapy, *European Heart Journal*, 34, 2281–2329.

Bunker, J (2014) Hypertension; diagnosis, assessment and management, *Nursing Standard*, 28(42), 50-59.

Camm AJ, Paulus Kirchhof P, Lip GYH, Schotten U, Savelieva I, Ernst S, Van Gelder IC, Al-Attar N, Hindricks G, Prendergast B, Heidbuchel H, Alfieri O, Angelini A, Atar D, Colonna P, De Caterina R, De Sutter J, Goette A, Gorenek B, Heldal M, Hohloser SH, Kolh P, Le Heuzey JY, Ponikowski P & Rutten FH (2010) Guidelines for the management of atrial fibrillation, *European Heart Journal*, 31, 2369–2429

Cardiac Arrhythmia Suppression Trial (CAST) Investigators (1989) Preliminary report: Effect of encainide and flecainide on mortality in randomized trial of arrhythmia suppression after myocardial infarction, *N Engl J Med*, 32I, 406-4I2.

Chemello D, Subramanian A, Nanthakumar K (2010) Cardiac arrest caused by undersensing of a temporary epicardial pacemaker, *Canadian Journal of Cardiology*, 26(1):e13-4.

Chung, C (2006) The use of digoxin in the treatment of arrhythmias and heart failure, Br J Card Nurs, 1(9), 442-447.

Davies A (2009) Permanent pacemakers; An overview, *British Journal of Cardiac Nursing,* 4(6), 262-269.

Dixon K, Thanavaro J, Thais A, Lavin MA (2013) Amiodarone Surveillance in Primary Care, *The Journal for Nurse Practitioners,* 9(1), 46-54.

Erdmann E (2009) Safety and tolerability of beta-blockers: prejudices and reality, *Eur Heart J Suppl.* 11(Suppl A):A21-5.

Ewy GA (2014) Sick sinus syndrome: synopsis, *J Am Coll Cardiol,* 64, 539-40.

Fogoros RN (2007) *Antiarrhythmic drugs, a practical guide*, 2nd edition, Oxford, Blackwell Publishing.

Furman, S. (2003) The early history of cardiac pacing, *Pacing and Clinical Electrophysiology*, 26(10): 2023-2032.

Furrer M, Naegeli B, Bertel O. (2004) Hazards of an alternative medicine device in a patient with a pacemaker, *New England Journal of Medicine,* 350(16):1688-90.

Garcia TB (2015) *12-lead ECG: The Art of Interpretation.* 2nd Edition. Burlington, Ma : Jones and Bartlett

Grant AO (2009) Cardiac ion channels, *Circulation: Arrhythmia & Electrophysiology*, 2, 185-194.

Gregory J (2006) An analysis tool for 12-lead ECG interpretation, *British Journal of Cardiac Nursing*, 1(5), 216-221.

Guthrie, K (2010) Hyperkalaemia, available at http://lifeinthefastlane.com/hyperkalemia/ [accessed 07/07/2016].

Hampton JR (2013) *The ECG in practice,* 6th edition, London: Churchill Livingstone.

Harper R & Morris F (2009) Pacemakers and electrocardiography, in Morris F, Brady WJ & Camm J (eds) *ABC of Clinical Electrocardiography*, 2nd edition, Oxford:Blackwell publishing, p. 66-69.

Houghton AR & Gray D (2014) *Making sense of the ECG: A hands-on guide*, 4th edition, Boca Raton: CRC Press.

Jensen PN, Gronroos NN, Chen LY, Folsom AR, deFilippi C, Heckbert SR, Alonso A (2014) Incidence of and Risk Factors for Sick Sinus Syndrome in the General Population*, J Am Coll Cardiol,* 64, 531–8.

Joint Formulary Committee (2016) British National Formulary, 71st edition, London: BMJ Group and Pharmaceutical Press.

Jowett NI & Thompson DR (2007) *Comprehensive coronary care*, 4th edition, London: Bailliere Tindall.

Kalahasty G, Kapa S, Cooper J (2014) Components of a pacing and ICD system: basic concepts of pacing, in Ellenbogen KA & Kaszala K (eds) *Cardiac Pacing and ICDs*, 6th edition, Oxford; Wiley Blackwell, p. 34-81.

Kaplana LJ & Kellum JA (2010) Fluids, pH, ions and electrolytes, *Current Opinion in Critical Care,* 16:323–331

Khan E (2006) Beta blockers: Types and clinical implications, *Br J Card Nurs*, 1, 132-138

Klabunde RE (2012) C*ardiovascular physiology concepts*, 2nd edition, Baltimore, MD; Lippincott Williams & Wilkins.

Lafuente-Lafuente C, Mahé I & Extramiana F (2009) Management of atrial fibrillation, *BMJ,* 339:b5216

Marieb EB & Hoehn KN (2015) *Human Anatomy & Physiology*, 10th edition, Cambridge: Pearson

McMurray JJ, Adamopoulos S, Anker SD, Auricchio A, Böhm M, Dickstein K, Falk V, Filippatos G, Fonseca C, Gomez-Sanchez MA, Jaarsma T (2012) ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure, *European journal of heart failure*, 1;14(8):803-69.

Misiri J, Kusumoto F, Goldschlager N. (2012) Electromagnetic interference and implanted cardiac devices: the nonmedical environment (part I), *Clinical cardiology*, 35(5):276-80.

McNaughton A (2013) Temporary pacing; clinical indications and techniques, *British Journal of Cardiac Nursing, 289-292.*

Murgatroyd F, Linker N, Cunningham D et al (2014) *National audit of cardiac rhythm management devices 2013-14*, available at https://www.ucl.ac.uk/nicor/audits/cardiacrhythm/documents/annual-reports/CRM_National_Annual_Report_2013-14 (accessed 28/06/2016).

Nachimuthu S, Assar MD & Schussler JM (2012) Drug-induced QT interval prolongation: mechanisms and clinical management, *Therapeutic Advances in Drug Safety*, 3(5), 241–253.

National Institute of Health and Care Excellence (2014) *Atrial fibrillation: the management of atrial fibrillation. NICE clinical guideline 180.* Available at https://www.nice.org.uk/guidance/cg180 [Accessed 12/07/2016].

Nielsen JC et al (2011) A comparison of single-lead atrial pacing with dual-chamber pacing in sick sinus syndrome, *Eur Heart J*, 32, 686-696.

O'Donovan K (2006) Amiodarone as a class III antiarrhythmic drug, *Br J Card Nurs*, 1(11), 530-539.

Opie L & Gersch B (2013) *Drugs for the Heart*. 8th edition. Philadelphia: W.B.Saunders.

Panchmatia S (2010) Atrial fibrillation and the use of antiarrhythmic drugs, *Nurse Prescribing*, 8 (1), 20-27.

Seegers J, Luthje L, Zabel M & Vollman D (2009) Loss of capture after right ventricular pacing lead revision; what is the mechanism? *Clin Res Cardiol*, 98(8), 517-520.

Shimony A, Eisenberg MJ, Filion KB, Amit G (2012) Beneficial effects of right ventricular non-apical vs. apical pacing: a systematic review and meta-analysis of randomized-controlled trials, *Europace,* 14(1):81-91.

Siddoway LA (2003) Amiodarone: guidelines for use and monitoring, *Am Fam Physician*, 68:2189-96.

Slovis C & Jenins R (2009) Conditions not primarily affecting the heart, in Morris F, Brady WJ & Camm J (eds) *ABC of Clinical Electrocardiography*, 2nd edition, Oxford:Blackwell publishing, p. 53-56.

Soar J, Perkins GD, Abbas G et al. (2010) European Resuscitation Council Guidelines for Resuscitation 2010 Section 8. Cardiac arrest in special circumstances: Electrolyte abnormalities, poisoning, drowning, accidental hypothermia, hyperthermia, asthma, anaphylaxis, cardiac surgery, trauma, pregnancy, electrocution, *Resuscitation,* 81: 1400-1433.

Torres-Ayala SC, Santacana-Laffitte G, Maldonado J (2014) Radiography of cardiac conduction device: apictorial review of pacemakers and implantable cardioverter defibrillators*, J Clin Imaging Sci*, 4(74).

Vogler J, Breithardt G & Eckardt (2012) Bradyarrhythmias and conduction blocks, *Rev Esp Cardiol*, 65, 656-667.

Waldo AL, Camm AJ, deRuyter H et al for the SWORD investigators (1996) Effect of d-sotalol on mortality in patients with left ventricular dysfunction after recent and remote myocardial infarction, *Lancet,* 348: 7–12.

Ward C, Henderson S, Metcalfe NH (2013) A short history on pacemakers, *International journal of cardiology*, 169(4):244-8.

Wiper A, Roberts DH, Jenkins NP (2008) Pacemaker syndrome – a forgotten diagnosis? *British Journal of Cardiology*, 5(1), 46-7.