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Understanding the ECG. Part 3: Arrhythmias

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Introduction

Heart rhythm disturbances, or arrhythmias, are a common problem encountered in clinical practice, and their recognition is a core skill in many areas of nursing (Bench and Brown, 2011; Jowett and Thompson, 2007). Evaluating heart rhythm is also one of the first steps in interpretation of the 12 lead ECG (Gregory, 2006). In normal sinus rhythm, the electrical impulse that causes the heart to beat arises in the sinus node, and is conducted to the ventricles by the specialised cardiac conduction system (Marieb and Hoehne, 2015). An arrhythmia is any deviation from this normal system of electrical impulse formation and conduction. In other words, arrhythmias occur when the electrical impulse arises outside of the sinus node, or is conducted abnormally through the electrical conduction system (Khan, 2006).

In this third article of the *British Journal of Cardiac Nursing's* ECG series, we will examine arrhythmias caused by abnormal impulse generation. The intention is not to explain every possible rhythm disturbance, but rather to review the most common and clinically relevant arrhythmias, and describe how they can be recognised on the ECG.   We will also place each arrhythmia in clinical context by briefly discussing why it occurs, and how it is treated. Hopefully this process will illustrate the mechanisms that cause arrhythmias, and deepen the reader's understanding of the electrical function of the heart. The rhythm disturbances that we will cover in this article are as follows:

- Ectopic beats
- Atrial fibrillation (AF) and flutter
- Supraventricular tachycardia (SVT)
- Ventricular tachycardia (VT)
- Ventricular fibrillation (VF)

Rhythm evaluation

In the previous article in this series (Sampson and McGrath, 2015a), we discussed the features of sinus rhythm, and identified the steps required to evaluate a rhythm. Briefly, these are to examine the heart rate, regularity of the rhythm, appearance of P-waves and the duration of the PR and QRS intervals. These are dealt with more fully in B*ox 1*. You may find it useful to review these important steps before reading further.

Box 1. Key steps in systematic rhythm evaluation

Ectopic beats

In the first part of this series, we discussed how electrical activity is generated within pacemaker cells in the sinus node (Sampson and McGrath, 2015b). We also described how many other parts of the heart are capable of generating an electrical impulse. This useful feature ensures that the heart does not stop beating if the sinus node fails. It also means, however, that beats can (and do) arise spontaneously from other parts of the heart and transiently interrupt normal sinus rhythm (Garcia, 2015). These beats are referred to as ectopic beats.

Ectopy is a medical term meaning outside of the normal place or position (Oxford University Press, 2015). Ectopic beats are single electrical impulses arising outside of the sinus node. They may originate in the atria, atrioventricular (AV) junction (the AV node and bundle of His) or the ventricles (Bennett, 2013). On their own, single ectopic beats are a normal phenomenon and do not require medical treatment. Frequent ectopic beats, however, can cause significant symptoms, and those arising from the ventricles can cause a deterioration in cardiac function (Lee et al, 2012). Ectopic beats are also common triggers for sustained arrhythmias in susceptible people (Fogoros, 2007).

Ectopic beats have a number of ECG features that help us to recognise them (Bennett, 2013)

- They are normally seen during sinus rhythm, which causes it to appear irregular at times
- All ectopic beats occur earlier than the next sinus beat would be expected
- Atrial ectopics are preceded by a P-wave with a different shape to the sinus beats. The P-wave may distort the T-wave of the previous beat if the ectopic is very early. The QRS complex is narrow.
- Junctional ectopics have an inverted P-wave. This may occur before or after the QRS complex, or may be hidden by it. As with atrial ectopics, the QRS is narrow.
- Ventricular ectopics are not preceded by a P-wave. The QRS complex is wide and bizarre. The T-wave is discordant; in other words, it travels in the opposite in direction to the QRS.

Figure 1 and *figure 2* compare atrial and ventricular ectopic beats.

Figure 1: The third and fifth beats are atrial ectopics. Note the P-wave buried in the T-wave, and the narrow QRS complexes.

Figure 2: The fourth and sixth beats are ventricular ectopics. Note the absence of P-waves, the wide QRS and the inverted T-wave.

Atrial fibrillation

AF is the most common arrhythmia, affecting around 2% of the UK population (National Institute for Health and Care Excellence (NICE), 2014a).   Prevalence increases with age, and numbers are widely expected to double as Western populations age (Colilla et al, 2013; Krijthe et al, 2013).   The problems associated with AF include a substantial increase in stroke risk, heart failure caused by uncontrolled heart rate, and unpleasant symptoms including palpitation, dizziness and shortness of breath (Camm et al, 2010).   Although a minority of patients who develop AF have no identifiable precipitant, the disease is strongly associated with conditions that cause left atrial enlargement or fibrosis. These include hypertension, coronary heart disease, and mitral valve disease. Other conditions classically associated with AF are thyrotoxicosis, alcohol abuse and chest infection (Andrade et al, 2014).

AF is a disease of the left atrium. The arrhythmia is triggered by ectopic beats arising from the pulmonary veins where they enter the left atrium (Haisseguerre et al, 1999). Impulse generation is fast and chaotic, and results in multiple electrical wavefronts sweeping across the atria. These collide, extinguish and reform, creating electrical chaos in the atria, and bombarding the AV node with up to 600 impulses per minute (Bennett, 2013). Fortunately, the AV node is unable to conduct every impulse, and thus only a fraction are conducted to the ventricles. The ventricular rate in AF is often rapid, but even without medication some patients will present with slowly conducted AF. This is due to slow AV node conduction caused by fibrosis of the conduction tissues (Camm et al, 2010). Drugs such as beta blockers, diltiazem and digoxin are used to slow AV node conduction when the ventricular rate is too fast (Lafuente-Lafuente et al, 2009).

On the ECG, AF has a number of key features that make it easy to recognise (Elliott, 2014):

- P-waves are replaced by fibrillation waves. These distort the baseline of the ECG in a constant, uneven pattern

- The rhythm is completely irregular, in other words there is no regular interval between the QRS complexes
- The QRS complex is narrow, unless conduction in the ventricles is abnormal (*e.g.* as a result of bundle-branch block)
- In some patients, fibrillation waves are very small or cannot be seen; AF can still be diagnosed by the lack of P-waves and completely irregular rhythm.

Figure 3 and *figure 4* show examples of atrial fibrillation.
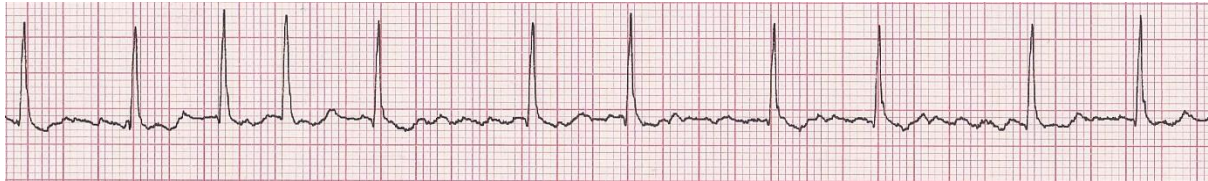
Figure 3: Atrial fibrillation. Note the irregular rhythm, absence of P-waves and jagged baseline.

Figure 4. Atrial fibrillation. In this example, the fibrillation waves are very fine so the baseline appears to be flat. AF is diagnosed by the irregular rhythm and lack of P-waves.

The treatment of AF depends on presentation, symptoms and individual patient factors. All patients should be assessed for stroke risk, and anticoagulation commenced if risk factors are present (Lip et al, 2010). Treatment of the rhythm itself aims to slow the ventricular rate as discussed above, or to terminate the rhythm using a co-ordinated electric shock through the chest wall (direct-current (DC) cardioversion), or antiarrhythmic drugs, such as amiodarone or flecainide (NICE 2014a). In longer term management, catheter ablation is rapidly gaining acceptance as an effective treatment, although success rates are variable and there is a small risk of serious complications, such as stroke, tamponade, and phrenic nerve injury (Cappato et al, 2010).

Atrial flutter

Atrial flutter is closely related to AF, and is often seen in the same patients. Although the mechanism is very different, the same underlying conditions are associated with flutter (*e.g.* hypertension and coronary heart disease) (Camm et al, 2010).

Impulse generation in atrial flutter is much more regular than in AF. The arrhythmia is commonly initiated by an ectopic beat which conducts abnormally through the atria (Gong et al, 2007). The impulse starts to circle continuously around the annulus of the tricuspid valve, travelling in an anticlockwise direction up the interatrial septum and then down the lateral wall of the right atrium (Feld et al, 2008). The impulse sweeps around the right atrium approximately 300 times a minute. As with AF, the AV node is unable to conduct every impulse. Unlike AF, in which conduction through

the AV node has a random pattern, there is often a regular ratio of conducted to blocked impulses. For this reason the rhythm is often regular, or regularly irregular (irregular but with some regular intervals). The ventricular rate is often a multiple of 300 (*e.g.* 150 beats per minute) (Bennett, 2013).

On the ECG, the characteristic features of atrial flutter are (Page et al, 2015)

- P-waves are replaced by regular flutter waves. These look similar to P-waves in the right sided precordial leads (V1 to V3), but in the inferior leads (II, III and aVF) have a characteristic 'saw tooth' pattern.
- As described above, the rhythm may regular or regularly irregular.
- As with AF, the QRS is narrow unless conduction in the ventricles is abnormal.

Figure 5 shows an example of atrial flutter, seen in lead II.
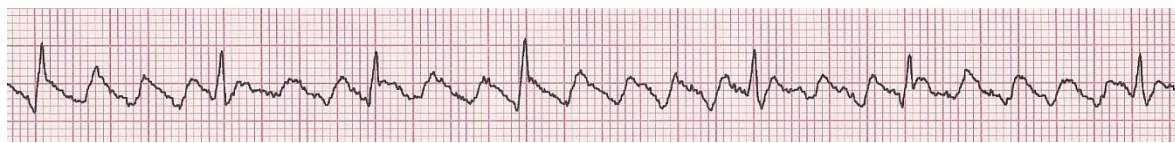
Figure 5. Atrial flutter. Note the saw tooth flutter waves.

The initial treatment of atrial flutter is identical to that of AF, namely anticoagulation and control of heart rate or rhythm. In specialist centres, catheter ablation is routinely offered to patients diagnosed with flutter because the procedure is simpler, safer and more successful than ablation for AF (Perez et al, 2009).

Supraventricular tachycardia

The literal meaning of SVT is a rhythm arising above the ventricles with a heart rate exceeding 100 beats per minute. By this definition, SVT includes AF, flutter and even sinus tachycardia (Marine, 2007). Although some authors (rather confusingly) use SVT in this broad sense, most clinicians involved in arrhythmia management use the term to refer to a more specific type of rhythm. This rhythm is fast, regular, has a narrow QRS complex, and shows no visible P-, flutter- or fibrillation-waves. The most common mechanism for this type of rhythm is re-entry (Page et al, 2015).

Re-entry describes a situation where two distinct pathways of conduction exist, with different conduction properties. An electrical impulse conducts down one, and then returns via the other to re-excite the area that it originated from (Fogoros, 2007). In SVT, re-entry occurs when the electrical impulse travels from the atria to the ventricles through the AV node, and is then conducted back into the atria. If the timing is right, the electrical impulse travels around this abnormal circuit continuously, causing a rapid tachycardia. The re-entry circuits that cause SVT are found either in the AV node itself (dual AV-node physiology) or involve an additional electrical connection between the atria and ventricles (accessory pathway). They are congenital conditions, although symptoms may not develop until later in life (Mullord and Sargent, 2011).

In dual AV-node physiology, the AV node has two electrical pathways leading into it instead of one (Marine, 2007). Re-entry occurs when an atrial ectopic beat enters one pathway while the other is still refractory, and therefore unable to conduct. If the second pathway has recovered by the time

the impulse has arrived in the AV node, it exits the node not only via the bundle of His but also via the additional pathway, travelling upwards into the atria (Lee and Linker, 2014). This causes a type of SVT known as atrioventricular nodal re-entrant tachycardia (AVNRT). This is commonest cause of SVT (Specter et al, 2009).

An accessory pathway is the second most common cause of SVT. An accessory pathway is a strand of myocardium that crosses the fibrous barrier that electrically isolates the atria from the ventricles (Page et al, 2015). Re-entry over an accessory pathway is again initiated by an ectopic beat. The electrical impulse travels to the ventricle via the AV node as usual, but is conducted back to the atria by the accessory pathway, establishing a re-entry circuit. This type of SVT is called atrioventricular re-entrant tachycardia (AVRT) (Lee et al, 2013).

On the ECG, the features that identify SVT are (Bennett, 2013)

- A regular tachycardia, usually between 130 and 250 beats per minute
- P-waves are often not seen. Where they are visible, they occur after the QRS complex
- The QRS complex is narrow, unless ventricular conduction is abnormal

An example of SVT can be seen in *figure 6*.

Figure 6. SVT. Note the regular rhythm, narrow QRS and absence of P-waves.

SVT is generally benign, although it can cause intrusive symptoms and result in diminished quality of life (Page et al, 2015). Sufferers may be young and healthy. Episodes are usually paroxysmal, meaning that they occur intermittently and terminate spontaneously. If episodes fail to terminate within a few hours, patients will often come to the A&E department for treatment (Appelboam et al, 2015). Re-entrant SVTs rely on split second timing, and will terminate if conduction through part of the circuit is slowed. This can be achieved by transiently slowing conduction through the AV node. Methods to slow AV node conduction include Valsalva manoeuvres, carotid sinus massage, and the administration of adenosine (Pitcher and Nolan, 2015). Beta-blockers and verapamil are also effective, although they are less commonly used. Re-entrant SVTs are highly amenable to catheter ablation, which has a success rate greater than 90% (Specter et al, 2009).
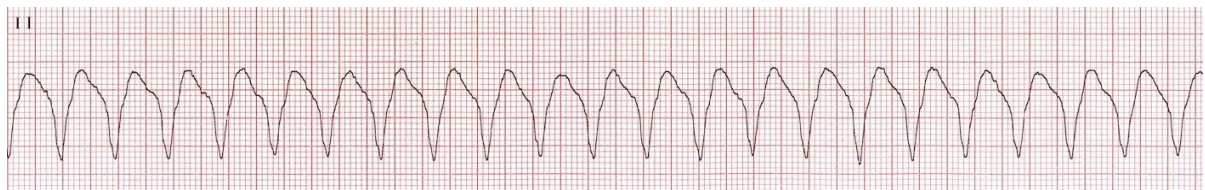
Ventricular tachycardia

As its name suggests, VT arises from a site within the ventricles. It may occur in short bursts that cause few symptoms, or in a sustained arrhythmia that can cause severe hypotension, loss of consciousness or cardiac arrest (Bennett, 2013). It is a much more alarming finding on the ECG than the rhythms we have considered so far. VT can be divided into two types according to ECG appearance: monomorphic and polymorphic.

Monomorphic VT

Monomorphic VT is the commonest type, and the most likely to be encountered in clinical practice. Monomorphic means having the same appearance; in monomorphic VT, the QRS complexes all have the same shape (*figure 7*). The key ECG findings in monomorphic VT are (Bennett, 2013)

- The rhythm is fast and regular
- The QRS complex is wide and bizarre
- T-waves are discordant
- P-waves do not precede the QRS complexes
- The heart rate can range from 120 to 250 beats per minute

Figure 7. Monomorphic VT. Note the broad QRS complexes and absence of P-waves.

A similar broad complex tachycardia occurs if SVT is conducted to the ventricles abnormally, for example in a patient with bundle branch block. There are various ECG features that may be seen in VT that help distinguish it from aberrantly conducted SVT. These include dissociated P-waves buried in the T-waves, and capture or fusion beats (Swift, 2013). These features occur because the sinus node often continues to fire during VT. The P-waves produced may be seen distorting the T-waves in VT. Capture beats are occasional normal beats seen during VT. They occur when a sinus node impulse manages to conduct to the ventricles between beats produced by the ventricular focus. Fusion beats occur in a similar way, but the ventricle is activated partly by the impulse from the sinus node, and partly by the ventricular focus, giving a 'fused' QRS (*figure 8*) (Houghton and Gray, 2014).

Figure 8. Monomorphic VT. The sixth beat is a fusion beat

The most common cause of monomorphic VT is structural heart disease, especially coronary artery disease. The mechanism is often a small, localised re-rentry circuit within the ventricle (Fogoros, 2007). In ischaemic heart disease, electrical function is normal in unaffected areas of the ventricle, but slow in areas that are ischaemic. An ectopic beat may travel through the ventricle and find an ischaemic area refractory despite the recovery of conduction in the surrounding tissue. The electrical impulse flows around the ischaemic area, but not through it. If the ischaemic area has

recovered conduction by the time that the impulse has circumvented it, the impulse is conducted back up through the ischaemic area, and a re-entry circuit is created (Wissner et al, 2012).

Less commonly, monomorphic VT occurs in structurally normal hearts, in which case the mechanism is usually a rapidly firing ectopic focus (Prystowsky et al, 2012). This is a common mechanism for VT in critically ill people, in whom ischaemia, electrolyte imbalance and increased sympathetic tone may all contribute to enhanced automaticity in parts of the ventricle (Bennett, 2013).

Polymorphic VT

In polymorphic VT the appearance of the QRS complex is not consistent, but changes progressively over a number of beats. The most common cause of polymorphic VT is QT interval prolongation, either due to drug therapy or inherited long QT syndrome. In patients with QT prolongation, polymorphic VT is also referred to as Torsade de Pointes (Bennett, 2013).

Figure 9 shows an example of Torsade de Pointes. As with monomorphic VT, the QRS complex is wide and bizarre; however, unlike monomorphic VT, the shape of the QRS complex is variable and appears to "twist" around the baseline. It is this appearance that gives the arrhythmia its name, from the French word 'torsader', meaning to twist (Dessertenne, 1966).
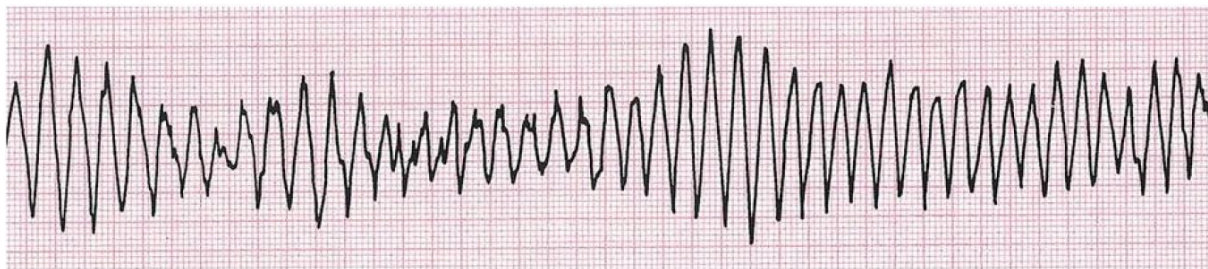
Figure 9. Torsade de pointes tachycardia. Note the constantly changing QRS morphology.

Treatment of VT

Seen on a bedside monitor, ventricular tachycardia constitutes a medical emergency and warrants immediate assessment and treatment (Swift, 2013). If the patient is unresponsive and pulseless, immediate defibrillation and cardiopulmonary resuscitation (CPR) are indicated (Soar et al, 2015). Patients who are responsive, but haemodynamically unstable, may require DC cardioversion; although stable patients can be managed with intravenous anti-arrhythmic drugs, typically amiodarone (Pitcher and Nolan, 2015). The treatment of potential precipitants is also important (*e.g.* correcting electrolyte imbalance). Patients should also be assessed for underlying heart disease, especially if they have no previous history of heart problems (Swift, 2013).

Longer term management depends on the risk of sudden cardiac death. Patients with normal hearts are at low risk, and are usually managed with medication or catheter ablation. Ablation has a success rate of around 90% in this group of patients (Ling et al, 2014). Patients with structural heart disease are at higher risk, especially when left ventricular function is significantly impaired. This group of patients may be offered an implantable cardioverter defibrillator (ICD) in addition to drug therapy (NICE, 2014b).

There is growing interest in ablation for VT due to structural heart disease, although success rates are lower than in normal heart VT, and the procedure does not seem to improve survival (Ling et al, 2014).   In long-QT syndrome, the management of polymorphic VT depends on which sub-type of the syndrome is present.   ICD implantation may be recommended if the history and clinical presentation suggest a high risk of sudden death (John et al, 2012). QT-prolonging drugs should be avoided (Nachimuthu et al, 2012).

Ventricular fibrillation

In VF, there is no organised rhythm at all. Electrical activity in the ventricles is chaotic and rapid. Very rarely, short self-terminating episodes of VF occur, but in most cases the rhythm is sustained and causes complete loss of cardiac output, and cardiac arrest (Bennett, 2013). The cause of VF is often acute ischaemia due to coronary heart disease. However, it can also occur in other forms of structural heart disease, as well as in inherited ion-channel disorders such as Brugada syndrome (John et al, 2012).

VF can be recognised on the ECG by its chaotic appearance. There are no recognisable QRS complexes (*figure 10).* The only treatment for VF is immediate defibrillation and CPR (Soar et al, 2015). Survivors of a VF arrest should be considered for an ICD (NICE, 2014b).
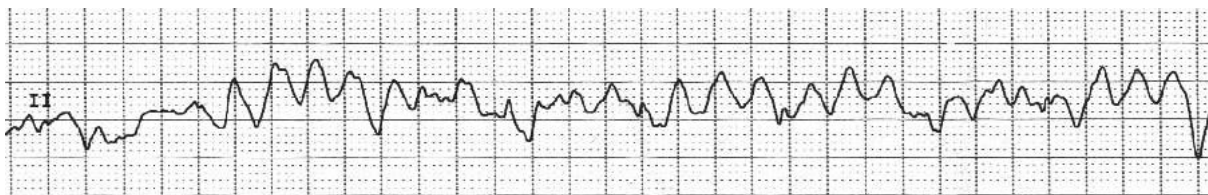
Figure 10. Ventricular fibrillation. Electrical activity is totally disorganised. There are no recognisable complexes.

Conclusion

Arrhythmias are a common cause of illness, and affect people with normal hearts as well as those with underlying structural or electrical abnormalities. Some arrhythmias have little haemodynamic consequence, while others may cause cardiac arrest. Recognising the clinical relevance of the arrhythmia is therefore important.

In this review, we have examined the most common arrhythmias that cardiac nurses are likely to encounter in clinical practice, and identified their ECG features. We have also put them in clinical context, and given a very brief overview of their treatment. We hope to have increased the reader's understanding of this important aspect of ECG interpretation, however given the complexity of the subject matter, readers are encouraged to explore it further. Next week we conclude our look at heart rhythm disturbances by examining arrhythmias caused by abnormal conduction of the electrical impulse.

Key points

- Ectopic beats are individual electrical impulses arising outside of the AV node. They are generally benign, although they frequently act as the triggers for sustained arrhythmias. On the ECG they are seen as single, early beats. Ectopics from the atria and AV junction have a narrow QRS, while ventricular ectopics are wide and bizarre in appearance.

- Atrial fibrillation (AF) is the most common arrhythmia and is associated with older age and cardiovascular disease. It is triggered by ectopics from the pulmonary veins, resulting in chaotic and uncoordinated atrial activation.   Key ECG features of AF are the loss of P-waves, and a totally irregular rhythm. The QRS is normally narrow. Initial treatment goals are to reduce stroke risk, and slow the heart rate. In the longer term, drugs and ablation are moderately successful in controlling the arrhythmia.

- Atrial flutter is closely associated with AF. It is caused by a regular electrical wavefront that sweeps around the tricuspid valve annulus in the right atrium. On the ECG it is often regular and can be recognised by sawtooth flutter waves in the inferior leads. The QRS is usually narrow. Initial treatment mirrors that of AF. Catheter ablation is highly successful in preventing recurrence of flutter and should be routinely offered.

- Supraventricular tachycardia (SVT) refers to a fast, regular rhythm with a narrow QRS complex. P-waves are often not visible, although sometimes they can be seen after the QRS. SVT is commonly caused by additional electrical pathways in the heart that are present from birth. Slowing AV-node conduction with vagal manoeuvres, carotid sinus massage or adenosine terminates the arrhythmia. The rhythm is not dangerous but can be intrusive. Catheter ablation is highly successful.

- Ventricular tachycardia is a more dangerous rhythm that can cause cardiac arrest. It is associated with structural and electrical heart disease, although it occasionally occurs in normal hearts. It can be recognised by wide, bizarre QRS complexes that are not preceded by P-waves. It may be monomorphic or polymorphic in appearance. Acute treatment of VT may require defibrillation, DC cardioversion or amiodarone infusion. In longer term management, ablation may be curative if the heart is normal, but in abnormal hearts an ICD may be required to prevent sudden death.

- Ventricular fibrillation is almost always sustained, and causes cardiac arrest. On the ECG it appears as disorganised electrical activity with no discernible rhythm. Immediate CPR and defibrillation are required. Survivors of VF arrest should be considered for an ICD.

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