Ventricular arrhythmias and sudden cardiac death

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Key points
- Arrhythmias of ventricular origin carry the greatest risk of sudden death.
- The presence of structural heart disease is the most important risk factor in the development of malignant ventricular arrhythmias.
- Ventricular arrhythmias causing cardiac arrest within 48 hours of myocardial infarction carry the same prognosis as that of a similar sized infarct without cardiac arrest.
- Acute management focuses on resuscitation, chemical or electrical cardioversion, electrolyte restoration, and overdrive pacing.
- Implantable cardioverter defibrillators and electrophysiology catheter-based ablations now offer improved survival and quality of life, respectively.

There are 50–100 unexpected, sudden cardiac deaths (SCDs) per 100 000 population per year in Europe and USA, categorized by symptom onset to cardiac arrest time of <1 h.1 Despite the decline in coronary artery disease mortality and advancements in resuscitation services, survival from these events remains low. Unfortunately, they often occur outside the hospital environment and are associated with a survival rate of <10%. The majority occur in adults over 35 yr of age and at least half of these events can be attributed to ventricular arrhythmias, although the true incidence is unknown due to inevitable degeneration to asystole if unwitnessed.

These arrhythmias may be the presenting complaint in the emergency department and may feature throughout the perioperative period, including at preoperative assessment, during surgery and in the recovery phase, for example in intensive care. They may also be the direct target of therapy in the case of electrophysiological catheter ablation and implantable cardioverter defibrillators (ICDs). They are more frequent and carry greater risks in patients with structural heart disease, but younger patients with ion-channel abnormalities can also be susceptible.2 This review classifies the causes and significance of ventricular arrhythmias based on the presence or absence of structural heart disease and provides a simple system to aid confident distinction from supraventricular arrhythmias. Treatment goals focus on acute management, including pharmacological agents and electrical cardioversion and also the long-term role of ICDs and ablation therapies.

Pathophysiology of ventricular arrhythmias

Arrhythmia morphology

Ventricular ectopic beats
Ventricular ectopic beats increase in incidence with age, with <1% incidence in children under 11 yr to over 60% in subjects over 75 yr. While often benign in nature, hypertension with left ventricular (LV) hypertrophy risks morbidity and sudden death. The mortality risk varies with the extent of underlying disease. However, contrary to previous preoperative risk scores, in the absence of structural heart disease, even frequent and complex ectopics may be completely benign.

Ventricular tachycardia
Ventricular tachycardia (VT)3 is defined as a heart rate >100 beats min−1 with three or more consecutive beats originating from the ventricles, independent of atrial or atroventricular (AV) nodal conduction. QRS is >120 ms on ECG. They may be non-sustained (<30 s) or sustained (>30 s). Classification can be via clinical presentation, ECG, or disease entity, the latter of which is demonstrated in Table 1. Monomorphic VT has a single QRS
Ventricular arrhythmias and SCD

### Table 1 Classification of ventricular arrhythmias by disease entity

<table>
<thead>
<tr>
<th>Monomorphic</th>
<th>Polymorphic</th>
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<tbody>
<tr>
<td>1. Structural heart disease</td>
<td>1. Normal corrected QT interval</td>
</tr>
<tr>
<td>(a) Coronary artery disease</td>
<td>(a) Acute ischaemia or scar</td>
</tr>
<tr>
<td>(b) Dilated cardiomyopathy</td>
<td>(b) Catecholaminergic polymorphic (CPVT)</td>
</tr>
<tr>
<td>(c) Hypertrophic cardiomyopathy</td>
<td>(c) Brugada syndrome</td>
</tr>
<tr>
<td>(d) Valvular heart disease</td>
<td>(d) Acquired</td>
</tr>
<tr>
<td>(e) Congenital heart disease</td>
<td>(i) Drugs</td>
</tr>
<tr>
<td>(f) Infiltrative heart disease (e.g. sarcoidosis, amyloidosis, haemochromatosis)</td>
<td>(ii) Electrolyte abnormalities</td>
</tr>
<tr>
<td>2. No structural heart disease</td>
<td>(iii) Neurological injury</td>
</tr>
<tr>
<td>(a) RV outflow tract VT (fibro-fatty replacement of muscle)</td>
<td>(iv) Starvation</td>
</tr>
<tr>
<td>(b) LV outflow tract VT</td>
<td></td>
</tr>
<tr>
<td>(c) Idiopathic LV septal VT (fascicular VT)</td>
<td>2. Long corrected QT interval (&gt;440 ms men, &gt;460 ms women)</td>
</tr>
<tr>
<td>(a) Congenital</td>
<td>(a) Congenital</td>
</tr>
<tr>
<td>(b) Acquired</td>
<td>(b) Acquired</td>
</tr>
<tr>
<td>(i) Drugs</td>
<td>(i) Long QT (&gt;440 ms in men, &gt;460 ms in women) is a heterogeneous group of at least 13 forms, where the prolonged repolarization period risks Torsades de pointes VT. It is predominantly a potassium-channel abnormality.</td>
</tr>
<tr>
<td>(ii) Electrolyte abnormalities</td>
<td>(ii) Brugada syndrome is a sodium-channel gene abnormality, with normal QT interval.</td>
</tr>
<tr>
<td>(iii) Neurological injury</td>
<td>(iii) Catecholaminergic polymorphic VT, which occurs during exertion or emotional trauma, is a ryanodine receptor gene abnormality.</td>
</tr>
<tr>
<td>(iv) Starvation</td>
<td>(iv) Short QT, a rarer, heterogeneous group, where the QT is typically &lt;300 ms is typically a calcium-channel abnormality, with abnormally short repolarization.</td>
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</table>

### Cardiac risk factors for SCD

Hypertension and the consequence of LV hypertrophy are recognized arrhythmogenic substrates, as is left bundle branch block. The Framingham Study, which began in 1948, has looked into risk factors for cardiovascular disease over a long period of time and has shown that cigarette smoking, obesity, and diabetes also increase the risk of SCD. While modest exercise is protective, vigorous exercise in unfit individuals carries a burden of risk as do the social and economic stresses that influence acute coronary events.

### Catecholaminergic polymorphic VT

Catecholaminergic polymorphic VT is a rare, heterogeneous group, with normal QT interval. It occurs during exertion or emotional trauma, is a ryanodine receptor gene abnormality. Short QT, a rarer, heterogeneous group, where the QT is typically <300 ms is typically a calcium-channel abnormality, with abnormally short repolarization.

### Arrhythmogenic substrate

**Coronary artery disease**

Two arrhythmogenic mechanisms are described in patients with coronary disease:

(i) acute myocardial ischaemia, resulting in polymorphic VT, which degenerates to VF;

(ii) myocardial scarring due to ischaemic cardiomyopathy where the main arrhythmogenic events is a re-entry monomorphic VT.

Scarring increases the proportion of non-conducting tissue, for example, fibroblasts, to conducting myocytes. This changes the velocity and uniformity of action potentials passing through the scarred muscle. Slow conduction and fibrous anatomical barriers generate multiple circuits, risking re-entry of action potentials into repolarizing muscle.

Despite acute coronary artery occlusion being the main trigger for arrhythmogenic events, family history plays an important role in outcome independent of traditional risk factors, with polymorphisms discovered in multiple stages of the atheroma to plaque cascade. Importantly, in survivors of cardiac arrest due to VF with associated acute ST segment elevation myocardial infarction (STEMI), acute prognosis is not significantly different from that associated with an infarct of similar size without arrest and constitutes part of the MI process. However, the formation of scar after 48 h becomes an arrhythmogenic substrate and risks clinically significant, secondary VT or VF.

### Presence of structural heart disease

This arrhythmia group commonly carries a benign prognosis. The majority originate from the RV or LV outflow tracts and anterior/posterior fascicles (fascicular VT). The incidence of tachycardia-related cardiomyopathy and SCD is extremely low. However, there are a rare heterogeneous group of arrhythmogenic syndromes, which are strongly related to life-threatening ventricular arrhythmias and SCD, especially in the young population. Inherited gene mutations are responsible for primary electrophysiological abnormalities and disruption of cardiac cell ion channel function.

### Absence of structural heart disease

This arrhythmia group is not discussed further.
plateau, which can trigger a re-entry tachycardia. Diagnosis depends upon both characteristic coved ST elevation in V1–V3 leads, followed by a negative T wave and clinical criteria (any one):

(i) documented VF or polymorphic VT,
(ii) family history of SCD at <45 yr old,
(iii) coved-type ECG in family members,
(iv) syncope,
(v) nocturnal, agonal respiration.

The ECG signs in isolation are of questionable significance. There are three ECG subtypes for the one condition. Type 1 is the classic shape and is shown in Figure 1. Type 2 has a more saddle-shaped ST elevation and type 3 can be either shape but <2 mm ST elevation.

**Diagnosis of VT**

Eighty per cent of broad complex tachycardias are ventricular. The positive predictive value in patients with coronary artery disease, heart failure, angina, and age >35 yr is over 95%. However, supraventricular tachycardia (SVT) can be responsible. The distinction between ventricular and supraventricular is important as misdiagnosis risks inaccurate therapeutic decisions, which could be potentially fatal. However, this distinction can be difficult and the debate should not impinge on delivering safe and effective therapy. If there is doubt, one should assume the rhythm is VT.

Broad complex SVT can arise from:

(i) aberrancy (bundle branch block),
(ii) antegrade conduction through an accessory pathway (pre-excitation).

With aberrancy, conduction is still via the His–Purkinje system but with a fascicular abnormality, that is, left or right bundle branch block. The right bundle normally repolarizes more slowly than the left, thus even in health, the timing of a supraventricular beat may depolarize one bundle, while the other is still refractory. An example of pre-excitation would be that seen in Wolf–Parkinson–White syndrome.

**Clinical information**

Stable VT may be asymptomatic or present with palpitations only. Faster rhythms impact more negatively on cardiac output, producing presyncopal symptoms like dizziness and light headedness, syncope (loss of consciousness with spontaneous recovery) to sudden cardiac arrest, or SCD if not resuscitated.

Physical examination may demonstrate signs of AV dissociation, specific to VT. These signs include varying pulse volume with beat-to-beat variability of systolic arterial pressure, alternating intensity of the first heart sound and irregular cannon A waves. These large pressure waves are transmitted up the internal jugular veins when the right atrium contracts against a closed AV valve. Tachycardia termination by vagal stimulation, for example, Valsalva manoeuvre, suggests supraventricular origin but may occasionally influence VT.

**Electrocardiography**

With VT, there is often positive or negative concordance in the chest leads, with entirely positive (R) or entirely negative (QS) waves in leads V1–V6, thus concordance has no RS complexes. Criteria favouring VT can also be found in SVT with aberrancy. The supraventricular impulse is transmitted to the ventricle via an abnormal connection but with discordance in the chest leads and RS complexes present. Very broad complexes (>160 ms) increase the likelihood of VT.

The Brugada diagram is an algorithm that aids VT diagnosis from ECG (Fig. 2):

(i) The absence of an RS wave in all of V1–V6 precordial leads diagnoses VT immediately and is nearly 100% specific. An example would be VT arising from the ventricular apex as

![Twelve-lead ECG type 1 pattern in Brugada syndrome](http://lifeinthefastlane.com/ecg-library/ventricular-tachycardia/).
Ventricular arrhythmias and SCD

In the preoperative patient with ventricular ectopics or in VT, excluding structural heart disease is the primary focus. History of syncope, heart failure, or arrhythmias up to 2 months after myocardial infarction are all high-risk characteristics. Electrolyte abnormalities and use of illicit drugs should be identified and addressed. Important investigations include:

Twelve-lead ECG: looking for underlying disease like Q waves, bundle branch block, and ventricular hypertrophy and also signs of arrhythmogenic syndromes. Long-term ECG monitoring via implantable loop recorder is useful in monitoring symptoms and correlating them to arrhythmias, but as they provide no therapies, patients with structural heart disease are better protected and monitored by insertion of an ICD instead.

Exercise testing: identifies silent ischaemia in coronary artery disease but also exercise-induced arrhythmias like catecholaminergic polymorphic VT. Frequent ventricular ectopic beats during exercise risk serious cardiovascular events in patients with structural heart disease, although not specifically SCD. They are difficult to treat, with β-blockers being the only class of drug proven to be efficacious.

Echocardiography: first-line investigation for evaluation of LV and RV function and wall thickness, regional wall motion abnormalities, valvular disease and congenital abnormalities. Cardiac magnetic resonance imaging shows higher diagnostic accuracy than echocardiography in identifying and quantifying myocardial scar burden which has been directly linked to SCD; demonstrates accurate and reproducible measurements of LV ejection fraction and volumes; offers qualitative assessment of RV structure and function.

Coronary angiography: should be performed in all individuals with risk factors for coronary artery disease and inducible ischaemia on non-invasive testing.

Cardiac CT scanning: non-invasive imaging of congenital coronary malformations or those in whom MRI is contraindicated. Calcification of normal vessels can also be quantified as a ‘coronary artery calcium score’.

Electrophysiological studies: to induce VT are useful in patients with coronary artery disease to guide diagnostic evaluation and after VT ablation to assess its efficacy. They are also useful as an accessory tool in patients with syncope of unknown cause and structural heart disease to document or exclude ventricular arrhythmias.

Electrophysiological studies

In the laboratory, electrophysiological mapping may be able to detect the source of the ventricular arrhythmia, which can be subsequently ablated using radiofrequency. Mapping, via femoral access, often under local anaesthesia, can provide anatomical reconstruction, which is then correlated to the arrhythmia. Stimulation of the ventricle can evaluate inducibility of VT in at-risk patients, characterize VT pattern and assist treatment choices. Mapping catheter electrodes are usually positioned in the high right atrium, ventricle, coronary sinus (alongside mitral annulus), and His bundles (alongside tricuspid annulus). ECG, echocardiography, and MRI (if no ICD) are vital to predict region of interrogation and avoid unnecessary procedure length. Thus, a lateral infarct suggests circumflex territory and lateral LV source. The RV can be accessed via the femoral vein, the LV either across the interatrial septum or retrograde passage via the aorta.
Table 2 Morphology criteria for VT vs SVT

<table>
<thead>
<tr>
<th>Lead V1</th>
<th>Tachycardia with RBBB-like QRS</th>
<th>Lead V1-2</th>
<th>Tachycardia with LBBB-like QRS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>QRS</strong></td>
<td>VT</td>
<td><strong>QRS</strong></td>
<td>VT</td>
</tr>
<tr>
<td>$v_1$</td>
<td>$v_1$</td>
<td>$v_1$</td>
<td>$v_1$</td>
</tr>
<tr>
<td>RS</td>
<td>VT</td>
<td>$&gt; 60$ ms to nadir of S</td>
<td>VT</td>
</tr>
<tr>
<td>$v_1$</td>
<td></td>
<td>$v_1$</td>
<td></td>
</tr>
<tr>
<td>Triphasic</td>
<td>SVT</td>
<td>Monophasic R</td>
<td>VT</td>
</tr>
<tr>
<td>$v_1$</td>
<td></td>
<td>$v_1$</td>
<td></td>
</tr>
<tr>
<td>R to S ratio &lt; 1</td>
<td>VT</td>
<td>Monophasic R</td>
<td>SVT</td>
</tr>
<tr>
<td>$v_6$</td>
<td>$v_6$</td>
<td>$v_6$</td>
<td></td>
</tr>
<tr>
<td>QS</td>
<td>VT</td>
<td>Monophasic R</td>
<td>VT</td>
</tr>
<tr>
<td>$v_6$</td>
<td>$v_6$</td>
<td>$v_6$</td>
<td></td>
</tr>
<tr>
<td>Triphasic</td>
<td>SVT</td>
<td>R to S ratio &gt; 1</td>
<td>SVT</td>
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<tr>
<td>$v_6$</td>
<td>$v_6$</td>
<td>$v_6$</td>
<td></td>
</tr>
</tbody>
</table>

RBBB = Right Bundle Branch Block, LBBB = Left Bundle Branch Block
through the aortic valve. The VT QRS morphology will determine the origin of the arrhythmia, thus RBBB pattern suggests the LV as source and vice versa. The His bundle interrogation should exclude the impulses arising from a supraventricular source. Occasionally, especially with structurally normal heart VT, increased automaticity with sympathomimetic drugs, for example, isoprenaline, may be required to generate the arrhythmia.

Management of VT and prevention of sudden death

Resuscitation

In the case of a cardiac arrest, the algorithm of advanced life support is applied. The latest resuscitation algorithm for tachycardia in adults is shown in Figure 5. Haemodynamically unstable VT presents with syncope, hypotension (typically systolic pressure below 90 mm Hg), pulmonary oedema, angina, confusion, and cardiac arrest. End-organ hypoperfusion mandates prompt termination of the arrhythmia, by synchronized DC cardioversion, which is both quicker and more effective than by chemical means. Under general anaesthesia or conscious sedation, a 120–150J synchronized biphasic shock is used, which is increased in increments to a maximum of three attempts. Reversible causes like hypoxaemia, electrolyte abnormalities (in particular hypokalaemia <4 mmol litre\(^{-1}\), hypomagnesaemia, hypocalcaemia), and drug toxicity should be corrected. The Resuscitation Council advise senior help early, rather than the use of multiple pharmacological agents. In the context of acute ischaemia and primarily STEMI, immediate revascularization is indicated.

Pharmacological therapy

Historically, the main treatment of acute ventricular arrhythmias has been pharmacological, but questions exist regarding safety and efficacy of drugs. The Vaughan–Williams classification is a useful template, although many drugs have actions across classes and others do not fit into the classification at all.
Magnesium (1–2 g slow i.v.) and potassium (aim K+ >4 mmol litre⁻¹) are the safest first-line therapy in all cases and can favourably influence the electrophysiological substrate in the onset of arrhythmias. The Resuscitation Council guidelines recommend expert advice for all irregular rhythms, where the arrhythmia origin may be unclear.

**Class I sodium-channel antagonists**

Pure class I drugs have a limited role to play in VT and are useful only in structurally normal hearts for symptomatic improvement. Flecainide has been shown to increase mortality post-MI. It has a limited role, as does propafenone in idiopathic VT only. Lidocaine may be used in stable, sustained, monomorphic VT in acute MI but increases mortality if used for prophylaxis against VF. It has a rapid onset of action but is more likely to cause hypotension than amiodarone, where the onset of action is slower.

**Class II β-blockers**

In the context of STEMI, β-blockers are first-line therapy.³ They are the most successful agents in prevention of ventricular arrhythmias and arrhythmic SCD in a wide spectrum of cardiac diseases with or without underlying structural heart disease. The anti-arrhythmic efficacy stems from competitive adrenergic receptor blockade, which reduces sympathetic triggers, slows the sinus rate, and may inhibit calcium release from the ryanodine receptor. Importantly, however, they are contraindicated in Brugada syndrome as they aggravate ion current imbalances that occur during the early part of the action potential, creating a vulnerable period for an extra-systole. Unopposed vagal tone is the biggest risk factor for arrhythmias (most occur at night), thus sympatholytics and bradycardia are dangerous. In fact, pure β-agonists, for example, isoprenaline, are used for resistant VT in this group of patients.

**Class III potassium-channel antagonists**

Amiodarone and sotalol have been shown to reduce arrhythmogenic SCD but not all-cause mortality. Class III drugs block potassium repolarization currents and thus increase the threshold for re-entry. In haemodynamically stable patients, with low troponin–myocardial infarction or non-ischaemic VT, there is Class C evidence for the use of either procainamide or amiodarone to terminate confirmed or suspected VT. Procainamide has both class I and III actions and has a favourable side-effect profile but is no longer regularly available in the UK.

**Class IV calcium channel blockers**

Class IV calcium channel blockers, for example, verapamil, should never be used to terminate broad complex tachycardia of unknown origin as they may precipitate VF. They have a select role to play in confirmed fascicular VT.

**Other agents**

The role of adenosine continues to remain controversial. The 2010 ILCOR guidelines state that adenosine may aid in diagnosing VT but will not terminate it. Adenosine is considered safe only if an SVT can be confidently excluded using tools discussed, or if

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Fig 5 Resuscitation Council Algorithm for adult tachycardia (with pulse). Reproduced with the kind permission of the Resuscitation Council (UK).
SVT with bundle branch block has been previously confirmed. Otherwise, it too may precipitate VF. Amiodarone and adenosine are the only drugs recommended to be used without expert consultation in all broad complex tachycardia groups.

It is important to recognize that ventricular arrhythmias are often the final common pathway in the unstable heart. Drugs with most success long term are not those above, which act directly on conducting muscle. Instead, it is drugs like ACE inhibitors, lipid-lowering agents, and aldosterone antagonists, for example, spironolactone, that influence the course of ischaemia, fibrosis, and biochemical derangement before the arrhythmogenic substrate is set.

**Implantable cardioverter defibrillator**

The ICD is a battery-powered invasive device with four functions:

1. Sensing: the device compares atrial and ventricular rates to assign arrhythmia causation.
2. Pacing: anti-bradycardia and overdrive anti-tachycardia pacing (ATP).
3. Cardioversion: synchronized electrical current to terminate cardiac output-compatible arrhythmias.
4. Defibrillation: to terminate VF.

While implanted in much the same way as a permanent pacemaker, general anaesthesia is usually recommended as VF is induced at the end of the procedure to test function and response time.

There is compelling evidence to support their use in the presence of ischaemic heart disease, with impaired LV ejection fraction, supported by NICE guidelines, updated in 2014.

ICDs are also recommended as options for treating people with previous serious ventricular arrhythmia, that is, people who, without a treatable cause:

- have survived a cardiac arrest caused by either VT or VF, or
- have spontaneous sustained VT causing syncope or significant haemodynamic compromise, or
- have sustained VT without syncope or cardiac arrest, and also have an associated reduction in LV ejection fraction of 35% or less but their symptoms are no worse than class III of the New York Heart Association (NYHA) functional classification of heart failure.

They are also recommended for treating people who:

- have a familial cardiac condition with a high risk of sudden death, such as long QT syndrome, hypertrophic cardiomyopathy, Brugada syndrome, or arrhythmogenic right ventricular dysplasia, or

Direct comparison of ICDs with anti-arrhythmic drugs has shown a clear reduction in SCD in carefully selected patients with a high-risk profile.\(^{10}\) Importantly, anti-arrhythmic drugs such as β-blockers, amiodarone, sotalol, and mexiletine still have a role in primary and secondary prevention. They reduce the arrhythmogenic burden and consequently the number of ICD shocks delivered.

**Anti-tachycardia pacing**

ATP through a temporary pacing wire can overdrive and terminate arrhythmias successfully and more importantly is the first technique an implanted ICD is programmed to perform in the presence of wide complex tachycardia. A slow, monomorphic VT can be detected then accelerated by the device to a short-lived burst of faster ventricular pacing, which on cessation terminates the VT, without the need for general anaesthesia. If unsuccessful, a subsequent burst of fast pacing followed by a low-voltage shock may also work. High energy shocks are very effective but are both painful and are a considerable drain to the ICD battery. ATP is unlikely to work during fast, haemodynamically unstable VT and risks degeneration to VF.

**ICDs and surgery**

As the indications for ICDs increase, so does the number encountered in patients presenting for surgery. While a detailed management plan for patients with implanted electronic cardiac devices\(^{11}\) is outside the scope of this review, some important principles apply:

1. Electromagnetic interference may inappropriately trigger the device. This includes electrocautery (monopolar much greater than bipolar), nerve stimulators, evoked potential monitors, electroconvulsive therapy, radiofrequency ablation, and MRI. Succinylcholine fasciculations, shivering, and large tidal volumes should not interfere with ICD sensing.
2. All ICDs can be deactivated with a magnet. This turns off the shock therapy and anti-tachycardia modes but has no effect on pacing and anti-bradycardia modes. Removal of the magnet should restore full device function. However, reactivation to pre-magnet settings is not guaranteed, thus a full ICD check should be completed after operation.
3. Formal ICD deactivation by a cardiac technician may be required if surgery is near the box site (e.g. cardiothoracics), remembering that deactivation of the device leaves patients vulnerable to malignant arrhythmias. It is helpful to know the model type, as the interrogation consoles are manufacturer-specific.
4. External defibrillator pads are recommended in all ICD patients and the device must be deactivated at the end of surgery.
5. Anaesthetic management should focus on reducing arrhythmias. Cardiostable, non-histamine-releasing drugs are recommended. Electrolyte disturbances, particularly potassium and magnesium, should be corrected and meticulous attention should be applied to Seldinger-type insertion of intra-vascular lines.

**Ablation of ventricular arrhythmias**

Evidence for catheter ablation of VT is mostly from patients with ischaemic heart disease. It is not a simple procedure and few patients are able to tolerate VT for sufficient time to allow all the necessary mapping required to identify all possible re-entry sites. Ablation energies can be applied using radiofrequency, cryotherapy, low energy direct current, or laser.

Success rate varies between 50% and 80% with reported complication rate up to 10% depending on the urgency of the procedure. Evidence exists for both prior and post-ICD implantation with successful reduction in arrhythmic burden and ICD therapies. No direct comparison between ablation and anti-arrhythmic drugs has been performed to date. Despite successful arrhythmia treatment, it is unclear if VT catheter ablation has an effect in SCD and mortality.
A small proportion of patients with RV and LV outflow tract VT have otherwise structurally normal hearts and this procedure importantly may offer a permanent cure.

The success of ablation procedures is expanding into more experimental techniques, including transthoracic pericardial access, transcoronary chemical ablation, and also spinal cord modulation to suppress VT.

Anaesthesia for ablation procedures is covered in a previous CEACCP article.12

**Conclusion**

The most important determinant in assessing the clinical significance of ventricular ectopic beats or tachycardia is the presence or absence of structural heart disease. Benign arrhythmias are more likely in patients under 35 yr old, except in those with hereditary channelopathies. Confidence in excluding the diagnosis of SVT with aberrancy is complex and may require expert help. Treatment of acute VT is via resuscitation, electrical or chemical cardioversion, and then assessing the need for further preventive therapy. ICDs and electrophysiology catheter-based ablations now offer improved survival and quality of life, respectively. Deactivating the defibrillation and anti-tachycardia function of an ICD can be achieved with a magnet. This leaves the patient vulnerable to arrhythmias; thus, the use of external defibrillation pads is mandatory.

**Declaration of interest**

None declared.

**MCQs**

The associated MCQs (to support CME/CPD activity) can be accessed at https://access.oxfordjournals.org by subscribers to BJA Education.

**References**