Management of ventricular tachycardia

Pulseless ventricular tachycardia should be managed according to universal advanced life support algorithms (not discussed in this review). Ventricular tachycardia in the presence of haemodynamic instability or pulmonary oedema mandates urgent direct current cardioversion synchronized to the surface QRS peak (Nolan et al, 2010). This should be performed immediately if the patient is unconscious. In those who are still conscious, a 12-lead electrocardiogram should be obtained (helpful as it localizes the site of ventricular tachycardia origin in planning future ablation) and then sedation or a general anaesthetic should be administered by a trained anaesthetist before shock delivery.

Following successful cardioversion, an intravenous infusion of amiodarone into a central vein may be considered to maintain sinus rhythm – this may lead to hypotension if delivered rapidly. Patients in ventricular tachycardia without haemodynamic compromise may be treated with intravenous amiodarone alone or in combination with an oral beta blocker. All patients should be managed in a place of continuous haemodynamic monitoring. Further ventricular tachycardia should be shocked again if the patient becomes unstable, but if tolerated, may be treated with an additional infusion of lignocaine.

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Figure 1. An example of R on T phenomenon triggering ventricular tachycardia as captured on an implantable cardioverter-defibrillator. The starred complexes represent ventricular ectopics. The third ventricular ectopic occurs in the T wave of a normal QRS complex, and triggers a rapid polymorphic ventricular rhythm.

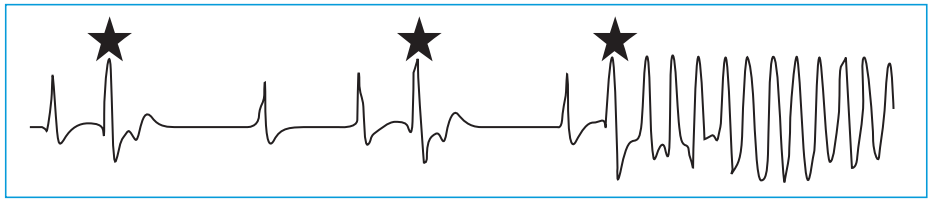


Table 1. Re-entrant ventricular tachycardias and ischaemia

Ventricular tachycardia	Polymorphic ventricular tachycardia	Monomorphic ventricular tachycardia
Substrate	Regional acute ischaemia causing acute and varying intra-myocardial electrochemical gradients	Fixed scar (old healed infarct) forming re-entry circuit
Initiator	Ectopic beats from ischaemia, electrolyte imbalance, idiopathic	Ectopic beats from ischaemia, electrolyte imbalance, idiopathic
Consequence	Ventricular tachycardia tendency resolves when ischaemia resolves	Ventricular tachycardia tendency persists despite resolution of ischaemia

Once patients are stabilized, acute reversible triggers should be excluded – these are described in detail in the 2014 European Heart Rhythm Association/Heart Rhythm Society/Asia Pacific Heart Rhythm Society consensus statement on ventricular arrhythmias (Pedersen et al, 2014). Electrolyte disorders including hypokalaemia, hypomagnesaemia and hypocalcaemia should be corrected. Ventricular tachycardia driven by acute ischaemia is usually polymorphic, and may be associated with frequent ventricular ectopic beats and R on T phenomenon (*Figure 1*). Prompt invasive assessment of the coronary circulation is needed with revascularization as required (Hollenbeck et al, 2013).

Following resolution of the ischaemia, ventricular tachycardia generally no longer occurs. Monomorphic ventricular tachycardia implies the presence of a fixed ventricular tachycardia circuit around a region of ventricular scar (see part 1 – diagnosis of ventricular tachycardia https://doi.org/10.12968/hmed.2017.78.1.C2). As the scar remains after the ischaemia resolves, the patient remains vulnerable to further ventricular tachycardia (*Table 1*).

In the context of QT prolongation associated with torsades de pointes, magnesium sulphate infusions can help during the acute arrhythmia, and isoprenaline infusions and atrial overdrive pacing (at least 90 bpm) to reduce the QTc prolongation have a role in preventing immediate recurrence once the patient is stabilized.

In cases of ventricular tachycardia storm, patients should be managed in a specialist electrophysiological centre. Ventricular tachycardia storm is defined as three or more episodes of sustained ventricular tachycardia over 24 hours, each requiring intervention. Insertion of an intra-aortic balloon pump may help to stabilize the patient as it provides diastolic counter-pulsation which maximizes coronary perfusion. A pro-adrenergic state often drives ventricular tachycardia, hence sedation or even general anaesthesia is considered to decrease sympathetic tone

and allow repeated cardioversion if needed. In the case of ventricular tachycardia storm, catheter ablation is the definitive therapy.

Implantable cardiac defibrillators

Survivors of ventricular tachycardia must be risk stratified for the likelihood of recurrence. If the ventricular tachycardia occurs as a result of acute myocardial ischaemia, no further intervention is generally required post revascularization. Short runs of non-sustained ventricular tachycardia are common immediately following primary angioplasty for ST elevation myocardial infarction as a result of myocardial reperfusion. In the long term, patients should all be managed on a standard beta blocker (Connolly et al, 2006). However, patients with an ongoing risk of further ventricular tachycardia and sudden cardiac death, particularly those with poor ventricular function or an underlying ion channel disorder, should be considered for an implantable cardiac defibrillator (Moss et al, 2002). Studies comparing implantable cardiac defibrillators with anti-arrhythmics showed that implantable cardiac defibrillators led to a greater reduction in mortality (Moss et al, 2004). National Institute for Health and Care Excellence (2014) guidelines regarding implantable cardiac defibrillator implantation are summarized in *Figure 2*.

Implantable cardiac defibrillators are specialized devices with pacemaker functionality. The implantable cardiac defibrillator consists of two elements, a pulse generator and a lead. The lead contains a pace-sense electrode as well as one or two shock coils. It is fixed within the right ventricle and attached to a subcutaneous generator containing a capacitor and battery. Electrical signals sensed from the leads are transmitted to the generator and internal algorithms detect the presence of ventricular arrhythmia, charging of the capacitor to deliver a biphasic shock across the coils through the myocardial mass to terminate ventricular tachycardia. Shocks are painful – their sudden and unpredictable nature may make them a cause of significant physical and psychological distress (Poole et al, 2008).

During slower ventricular tachycardias, the likelihood of immediate syncope, haemodynamic disturbance and cardiac arrest is less – as a consequence, rather than shocking the patient every time the device recognizes ventricular tachycardia, in the presence of slower ventricular tachycardia rates, the device attempts a different form of therapy – anti-tachycardia pacing. In the first article (https://doi.org/10.12968/hmed.2017.78.1.C2), the authors described how a re-entrant circuit formed around areas of scar tissue resulting in monomorphic ventricular tachycardia. As the wavefront revolves around the circuit, it leaves a trail of refractory tissue behind it. There is usually a dynamic area of excitable tissue within the circuit – the excitable gap. The re-entrant ventricular tachycardia can terminate by a critically timed pacing stimulus that enters the excitable gap and colliding with the ventricular tachycardia (*Figure 3*). Multiple pacing stimuli delivered in this way increase the probability of interacting with the ventricular tachycardia circuit and the likelihood of termination. Importantly, unlike shock therapy, the patient cannot feel anti-tachycardia pacing.

Anti-tachycardia pacing is not always successful. Failure to terminate ventricular tachycardia can prolong the time a

Figure 2. National Institute for Health and Care Excellence guidance (2014) on use of implantable cardioverter-defibrillators.

Secondary prevention

Patients who present, in absence of a treatable cause, with one of the following:

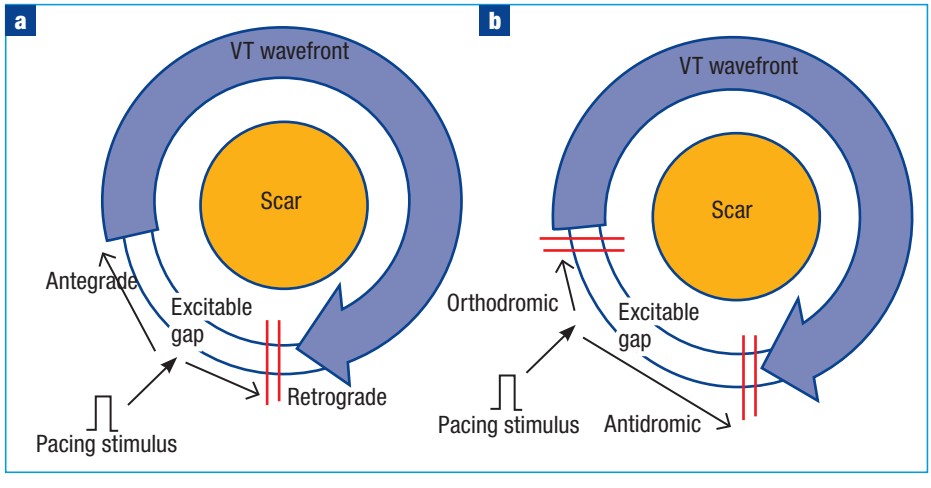
- Survived cardiac arrest caused by ventricular tachycardia or ventricular fibrillation
- Spontaneous sustained ventricular tachycardia causing syncope or haemodynamic compromise
- Sustained ventricular tachycardia without syncope or cardiac arrest and have ejection fraction ≤35% with New York Heart Association (NYHA) class ≤III

Primary prevention

- A history of heart failure (NYHA ≤III) with a left ventricular ejection fraction of <35%.
- Familial cardiac condition with a high risk of sudden death: long QT syndrome, hypertrophic cardiomyopathy, Brugada syndrome, arrhythmogenic right ventricular dysplasia, undergone surgical repair of congenital heart disease

National Institute for Health and Care Excellence implantable cardioverter-defibrillator implantation guidelines were updated in 2014. There is no longer a need to demonstrate a susceptibility to ventricular tachycardia, either on Holter monitoring or with electrophysiological testing, as many patients go on to develop ventricular tachycardia despite a negative test. Furthermore, a QRS duration of 120 ms in sinus rhythm on the resting 12-lead electrocardiogram was previously required pre-device implantation in primary prevention – this is no longer absolutely required.

Figure 3. The ventricular tachycardia (VT) wavefront (blue arrow) rotates around an island of scar (yellow circle). a. The head of the VT wavefront (blue arrow head) is followed by an area of refractoriness (blue tail). There is a gap between the head and the tail, known as the excitable gap. A critically timed pacing stimulus enters the excitable gap, and travels both towards (antidromic) and away from (orthodromic) the head of the wavefront. The antidromic activation collides with and extinguishes the VT wavefront head. The orthodromic activation 'pre-excites' the tail, continuing the wavefront. b. A more premature stimulus is displayed. This time, the stimulus encroaches on the refractory period of the preceding wavefront and is blocked from travelling around the circuit. As the antidromic wavefront again collides with the head, the result is termination.



patient remains in ventricular tachycardia, increasing the risk of patient compromise. Anti-tachycardia pacing can also accelerate the ventricular tachycardia – rather than travelling in the opposite direction and colliding with the head of the ventricular tachycardia circuit, it travels in the same direction ahead of the circuit, thus speeding it up. This can lead to very fast ventricular tachycardia and rapid degeneration to ventricular fibrillation.

What to do if the implantable cardiac defibrillator fires?

In the event of receiving a single shock, if the patient was otherwise well, he/she can be seen by the device clinic the next working day. Remote monitoring allows device interrogation from home, and early recognition by health-care professionals. Interrogation is initially directed at determining whether the shock was appropriate or inappropriate. Inappropriate therapy can occur if the device recognizes non-ventricular high rate episodes as ventricular tachycardia or registers external artefact (myo-potentials) or T waves as an independent QRS complex (T wave over-sensing). If the shock was found to be appropriate, the device can be reprogrammed to offer more anti-tachycardia pacing, and amiodarone may be considered to prevent subsequent shocks.

In the event of two or more shocks, patients are advised to call for an ambulance. The implantable cardiac defibrillator can be inhibited from delivering shocks by placing a magnet on the skin over the device. This will stop shocks but not any pacing function. These magnets are usually of a circular 'donut' shape and are usually kept on all coronary care units. 'Electrical storm' is defined as three or more appropriate shocks from an implantable cardiac defibrillator in 24 hours. Patients should receive maximal beta-blockade and amiodarone while the trigger is treated. If none is found, an ablative strategy may be required.

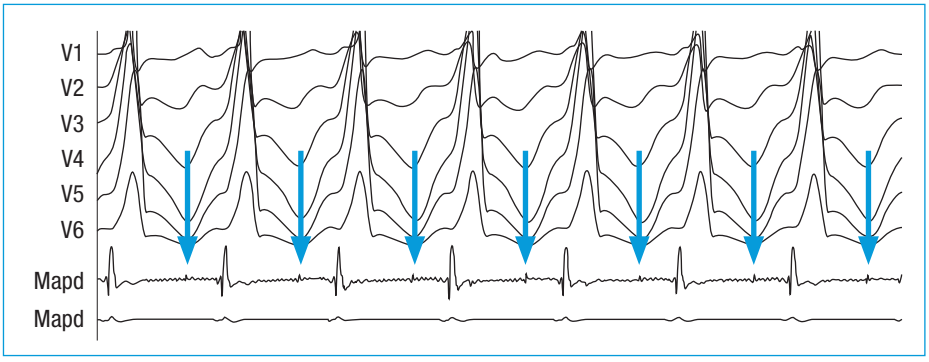
Catheter ablation of ventricular arrhythmia

Implantable cardiac defibrillators effectively terminate ventricular tachycardia, but do not prevent any subsequent recurrence. The arrhythmogenic substrate remains unchanged and may even progress over time, resulting in new or increasingly frequent episodes of ventricular tachycardia in a considerable number of patients. Ventricular tachycardia episodes predict an increased risk of death and heart failure hospitalizations in implantable cardiac defibrillator recipients – indeed there is some evidence to suggest that defibrillator shocks not only worsen quality of life but actually increase mortality (Poole et al, 2008).

In patients with recurrent symptoms associated with ventricular tachycardia without structural heart disease (e.g. outflow tract or fascicular tachycardia), catheter ablation for ventricular tachycardia offers an effective curative strategy and carries low procedural risk. Catheter ablation in patients with structural heart disease is considered as an adjunct or alternative to anti-arrhythmic therapy for patients with implantable cardiac defibrillators who receive one or more appropriate implantable cardiac defibrillator therapies for ventricular tachycardia in the absence of a reversible cause (Aliot et al, 2009). Amiodarone, the most commonly used anti-arrhythmic, is associated with several adverse effects including photosensitivity, corneal deposits, liver toxicity, pulmonary toxicity, thyroid dysfunction and peripheral neuropathy and hence is not always well tolerated.

Although there is no evidence that catheter ablation of ventricular tachycardia reduces mortality, early referral for catheter ablation following implantable cardiac defibrillator intervention has the potential to improve patient quality of life (Poole et al, 2008). Catheter ablation can be lifesaving for patients with ventricular tachycardia storm (Carbucicchio et al, 2008). The benefits of the procedure must be weighed against the risk of complications, including cardiac tamponade, stroke, heart failure and death.

Figure 4. Intra-cardiac electrogram during ventricular tachycardia – a clear 'mid-diastolic signal' can be seen (blue arrows) in the distal bipolar mapping electrode (Mapd) before the inscription of the surface QRS.

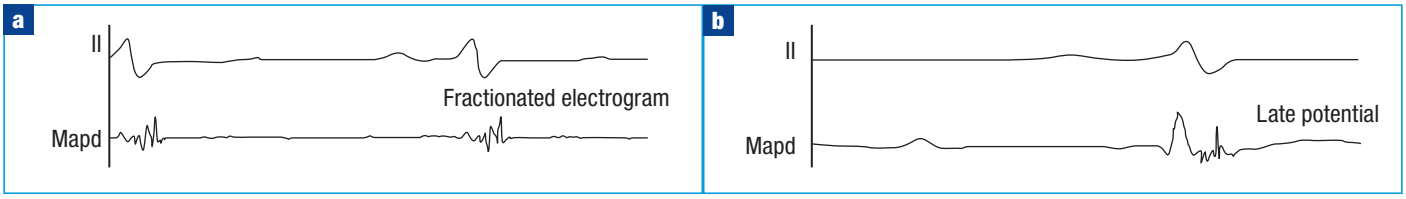


By far the most common cause of ventricular tachycardia in patients with structural heart disease is re-entry involving myocardial scar (see part 1 https://doi.org/10.12968/hmed.2017.78.1.C2). Catheter ablation aims to eliminate the 'conduction channels' within the scar that support the re-entrant circuits. These targets are identified using intra-cardiac electrograms collected by passing an exploring electrode catheter through the venous or arterial system towards the heart. In ventricular tachycardia, a small sharp signal seen by the catheter in ventricular diastole (mid-diastolic potential) can represent the site of the channel (*Figure 4*), ablation of which can terminate the ventricular tachycardia.

Ventricular tachycardia ablation is frequently limited by one thing – the patient's requirement to be in ventricular tachycardia. This is often haemodynamically poorly tolerated. With patients in sinus rhythm, fractionated electrograms and late potentials seen at the end of the QRS complex represent slow conduction through scar (*Figure 5*) and are used as surrogates for conduction channels and targeted for ablation (Zeppenfeld et al, 2005; Vergara et al, 2012).

Rather than being limited to electrogram analysis in isolation, these can now be combined with three-dimensional models of the inner (endocardial) and outer (epicardial) layers of the heart using electro-anatomical mapping systems. The maps can be used to anatomically define potential conduction channels by tagging points of fractionation or late potentials (Volkmer et al, 2006). One system (CARTO, Biosense Webster, Inc., Diamond Bar, California) uses electromagnetic fields emanating from coils beneath the patient detected by a location sensor embedded in the tip of the catheter in the patient's heart. This allows a three-

Figure 5. Intra-cardiac electrograms collected during sinus rhythm in a patient with a history of ventricular tachycardia and previous myocardial infarction. **a.** A multicomponent, 'fractionated' electrogram in the distal bipolar mapping electrode (Mapd) after the inscription of the surface QRS (lead II). **b.** A tall single sharp spike well after the surface QRS in keeping with a late potential.



dimensional reconstruction of the chamber of interest and can be colour-coded to display maps of electrical activation through the myocardium or scar.

One of the largest prospective studies assessing the effectiveness of post infarct ventricular tachycardia ablation was the multicentre 'Thermocool Ventricular Tachycardia Ablation Trial'. This enrolled 231 patients with scar-related ventricular tachycardia. During the 6-month follow-up period, 53% of patients were free of ventricular tachycardia at 6 months. Improved methods to reduce long-term ventricular tachycardia recurrence rates are still required. Procedure mortality was 3%, although in six out of these seven cases this was related to peri-procedural uncontrollable ventricular tachycardia and progressive haemodynamic compromise as opposed to the procedure itself, reflecting the severity of the underlying substrate (Stevenson et al, 2008).

Catheter ablation is also performed in patients with non-infarct related scar, such as those with other forms of cardiomyopathy. In these patients, the channels are often found on the epicardial surface of the ventricle, resulting in more complex procedures requiring pericardial access via pericardiocentesis.

Conclusions

Ventricular arrhythmias can be life-threatening. Prompt action and defibrillation can save lives. The key issue is the management following defibrillation. Implantable cardiac defibrillators can reduce mortality but are not a panacea if the underlying trigger remains. Ventricular tachycardia ablation offers a potential curative strategy but still requires future research to improve on present day outcome. **BJHM**

Conflict of interest: none.

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KEY POINTS

- Ventricular tachycardia in the presence of haemodynamic instability or pulmonary oedema mandates urgent cardioversion.
- Ventricular tachycardia driven by acute ischaemia is usually polymorphic, and may be associated with ventricular ectopic beats and secondary R on T phenomenon.
- Monomorphic ventricular tachycardia implies the presence of a fixed ventricular tachycardia circuit. After the ischaemia resolves, the patient remains vulnerable to further ventricular tachycardia and should be considered for an implantable cardioverter-defibrillator.
- Implantable cardioverter-defibrillator shocks are painful – their sudden and unpredictable nature may make them a cause of significant physical and psychological distress.
- Catheter ablation should be considered in patients with structural heart disease as an adjunct or alternative to anti-arrhythmic therapy for patients with recurrent implantable cardioverter-defibrillator therapies.