Catheter ablation of ventricular tachycardia in ischaemic and non-ischaemic cardiomyopathy: where are we today? A clinical review

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According to the current guidelines, patients with ischaemic cardiomyopathy (ICM) or non-ischaemic cardiomyopathy (NICM) at risk for sudden cardiac death should undergo implantation of an implantable cardioverter-defibrillator (ICD). Although ICDs effectively terminate ventricular arrhythmias, the arrhythmogenic substrate remains unchanged or may progress over time, resulting in recurrent ICD shocks. Defibrillator shocks increase mortality and worsen quality of life. Evidence from two prospective randomized trials on outcome in patients with ischaemic heart disease undergoing catheter ablation for ventricular tachycardia (VT) suggests that ablation prevents recurrence of VT and decreases the number of ICD shocks. This review will highlight the recent progress made in the ablative treatment of VT in patients with ICM and NICM.

Keywords
Ventricular tachycardia • Ischaemic cardiomyopathy • Non-ischaemic cardiomyopathy • Radiofrequency ablation • Catheter ablation • Clinical outcome • Implantable cardioverter-defibrillator

Introduction
Catheter ablation of ventricular tachycardia (VT) has made significant strides over recent years with new evidence from prospective randomized trials on outcome in patients with ischaemic heart disease. Implantable cardioverter-defibrillators (ICDs) are currently the mainstay of treatment for patients with ischaemic cardiomyopathy (ICM) or non-ischaemic cardiomyopathy (NICM), who are at risk for sudden cardiac death due to VT.1,2 However, ICDs effectively terminate VT, but do not prevent VT episodes. The arrhythmogenic substrate remains unchanged or may progress over time, resulting in new or increasingly frequent episodes of VT in a considerable number of patients. Defibrillator shocks increase mortality and worsen quality of life.3-5 β-Blocker therapy in combination with amiodarone reduces ICD shocks for some patients; however, amiodarone has significant side effects, resulting in drug discontinuation in nearly 25% of the patients.6 Catheter ablation now has an important role to control incessant VT and to reduce or prevent recurrent episodes of sustained VT.7-12 This review will focus on the progress that has been gained over recent years in the ablative treatment of VT in patients with structural heart disease.

The importance of ventricular tachycardia types, substrates, and the 12-lead surface electrocardiogram
The majority of patients who have recurrent episodes of sustained VT have an arrhythmia substrate defined by areas of ventricular scar. Scars can be due to prior myocardial infarction in those with coronary artery disease, or replacement fibrosis in non-ischaemic heart diseases, or surgical incisions, as after repair of tetralogy of Fallot. Surviving myocyte bundles within the scars characterized by diminished cell coupling and interstitial fibrosis promote slow conduction and create regions of anatomical or functional conduction block causing reentry.13 The reentry circuit includes an isthmus of slow conduction. The exit site of the critical isthmus gives rise to the QRS complex and is often the initial target during catheter ablation. The isthmus may span several centimetres.14 Furthermore, the reentry circuit needs to be appreciated as a complex three-dimensional construct that can involve the endocardium, mid-myocardium, or epicardium.15

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Ventricular tachycardia due to scar-related reentry is usually monomorphic, with each QRS complex resembling the preceding and following QRS. In monomorphic VT, the sequence of ventricular depolarization is the same from beat to beat. The QRS morphology of VT is determined by the location of the scar and the location of the reentry circuit within the scar, specifically the exit region where the reentry wavefronts propagate away from the scar to depolarize the ventricles. Typically, multiple VT morphologies can be induced in the same patient. Multiple VTs may share the same isthmus with different exit sites or depend on different isthmus within the same scar or indicate the presence of different reentry circuits in different regions of scar (Figure 1). In some patients, one monomorphic VT may initiate a second monomorphic VT causing more than one distinct QRS configuration during a single VT episode, known as pleomorphic VT. Ventricular tachycardias that occur spontaneously are commonly referred to as clinical VTs. Programmed ventricular stimulation may initiate other morphologies of VT, of less certain clinical relevance, which may not necessarily occur spontaneously.

Polymorphic VTs have a continually changing QRS morphology, indicating a changing sequence of ventricular activation. Although ventricular scar may be present, these arrhythmias are often associated with acute myocardial ischaemia, inherited ion channel abnormalities, or ventricular hypertrophy; and a fixed structural substrate, such as scar, is not required. Polymorphic VTs are less likely to have an identifiable substrate that can be targeted for ablation and warrant a different set of diagnostic and therapeutic concerns.

From these considerations, it is clear that electrocardiographic (ECG) recordings of the spontaneous VT are very helpful in guiding evaluation and therapy. Documentation of the spontaneously occurring clinical VT on the 12-lead surface ECG should always be sought if possible. The QRS morphology can serve as a guide during the mapping and ablation procedure to recognize clinical VTs and suggest the location of the VT substrate. Right bundle branch block morphology in lead V1 suggests a left ventricular (LV) origin, while left bundle branch block morphology (dominant S-wave) indicates an exit site from the right ventricle or interventricular septum. A superior axis is expected for sites emanating from the inferior ventricular wall. Cranial sites produce an inferior axis. Deep S-waves in the precordial leads V3 and V4 indicate an apical exit site, while dominant R-waves point towards an exit along the base of the ventricle. In patients with non-ischaemic cardiomyopathy (NICM), the QRS morphology can also suggest whether epicardial ablation is likely to be required, which is helpful in advising the patient about procedural risks and in optimizing the sequence of mapping and the use of anticoagulation, which is ideally avoided before attempting percutaneous epicardial access. Compared with endocardial VTs, those that originate from epicardial scar typically have a wider QRS interval since the activation wavefront traverses from the epicardial to the endocardial layer before engaging the rapidly conducting Purkinje system. The presence of a Q-wave in lead I during VT is seen if local activation spreads from the basal superior or apical superior epicardium to endocardium, while a Q-wave in the inferior leads suggests a site from the basal inferior or apical inferior epicardium. These ECG criteria have not been reliable in ischaemic cardiomyopathy (ICM).

Frequently, a 12-lead ECG of the clinical VT is not available in ICD patients. The device promptly terminates VT with either anti-tachycardia pacing or shocks. In these cases, careful analysis of stored ICD electrograms may facilitate morphological characterization of VT and, in some patients with ICM, can help recognize clinical VTs that are induced by programmed ventricular stimulation during the procedure.

**Pre-procedural considerations**

Cardiac echocardiography should be performed in all patients presenting for ablation in order to rule out mobile LV thrombus. Since cardioversion or defibrillation may be required during mapping and ablation, in patients with concomitant atrial fibrillation, transoesophageal echocardiography should be performed to screen for left atrial thrombus in patients who have not been chronically anticoagulated. If recent coronary vascular status is unknown or the patient presents with polymorphic VT, coronary angiography should be performed. The presence of significant untreated coronary artery disease may limit mapping during tachycardia due to haemodynamic compromise caused by myocardial ischaemia. If haemodynamically unstable VTs are present or anticipated, general anaesthesia should be considered. In addition, the use of a haemodynamic support system may facilitate proper mapping.
and ablation and reduce risks of haemodynamic deterioration from episodes of VT during the procedure.\textsuperscript{24}

**Procedure techniques**

Programmed stimulation is performed in order to induce VT, confirm the diagnosis, assess the VT ECG morphology, and determine whether VT non-inducibility may serve as the acute procedural endpoint (Figure 2). In the case of non-inducibility or inducibility of non-clinical VT, a substrate-based mapping and ablation approach may be chosen. If VT is haemodynamically unstable, it is immediately terminated using burst pacing or electrical cardioversion. Based on the location of the scar and the morphology of the VT, initial mapping then begins in either the right ventricle or LV, or the pericardial space. Endocardial LV mapping may be performed via a transseptal or a retrograde arterial approach. Damage to the aortic valve or coronary arteries is rare using a retrograde arterial approach.\textsuperscript{11} If retrograde aortic access is difficult or impossible, as in patients with significant peripheral vascular disease, aortic stenosis, or a mechanical aortic valve prosthesis, a transseptal approach is the preferred route of access to the LV. A transseptal approach may also be used concomitantly with a retrograde aortic access to facilitate mapping in some patients. To date, no randomized study has compared a retrograde transaortic with an anterograde transseptal approach for LV mapping and ablation. Once LV access is secured, unfractionated heparin should be administered to target an activated clotting time of $>250$ s.

**Epicardial ventricular tachycardia ablation**

Epicardial mapping and ablation is now commonly performed in experienced centres when an epicardial VT is suspected. In patients with NICM and arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D), endo- and epicardial mapping and ablation are frequently performed in combination since these patients have a higher likelihood of epicardial involvement.\textsuperscript{25} Epicardial ablation is more frequently required in patients referred after failed VT ablation.\textsuperscript{26}

There are several unique aspects to epicardial mapping and ablation. Access to the epicardium is obtained via pericardial puncture from a subxyphoid approach, which allows placement of a vascular introducer sheath into the pericardial space. The mapping and ablation catheter can then be inserted. Since an irrigated-tip ablation catheter (see below) is utilized for mapping and ablation, regular aspiration of epicardial fluid should be performed. Risks include pericardial bleeding (4.5%) that may require emergent surgery, liver laceration (3%), and damage to epicardial coronary arteries (1.2%).\textsuperscript{26,27} Coronary angiography is often needed to define the proximity of epicardial VT substrate to coronary arteries; and in some cases, overlying coronary arteries or proximity to the left phrenic nerve precludes ablation. Percutaneous epicardial access is often not possible in patients who have pericardial adhesions, as are common after prior cardiac surgery. A surgical approach to ablation is an option for some of these patients. Some operators give prophylactic steroids following the procedure in an attempt to decrease the likelihood of sterile pericarditis that may be seen in up to 11% of the patients.\textsuperscript{26} Adhesion formation may interfere with repeat attempts at epicardial access in up to 25% of the patients.\textsuperscript{26}

**Robotic mapping and ablation systems**

Systems that allow catheter manoeuvring from a console remote from the patient, with the use of magnetic fields to orient the catheter are a feasible alternative to conventional manual catheter manipulation.\textsuperscript{28} It is hoped that procedure success will be less dependent on individual operator skill, while fluoroscopy exposure to the patient and the operator is decreased. The soft-tipped magnetic catheter is less likely to result in cardiac trauma, may reduce catheter-induced ventricular extrasystoles during mapping, and may facilitate constant tissue contact during ablation. Evidence is accumulating that remote-controlled ablation of VT is feasible; however, studies are needed to compare efficacy with manual catheter mapping and ablation.\textsuperscript{29} Lastly, trials are underway to assess the feasibility of remote VT ablation using the Hansen robotic system.

**Mapping**

Scar-related VT circuits are often large. Ablation targets reentry circuit isthmuses and their exits. The ability to identify and target the VT substrate has been greatly facilitated with the use of mapping systems that permit accurate three-dimensional reconstruction of cardiac anatomy using point-by-point sampling. The position of the mapping catheter is traceable on the virtual map, reducing the need for fluoroscopy. Features of electrogram amplitude or timing can be colour-coded and displayed on the map (Figure 2B).\textsuperscript{30}

When VT is not incessant, and particularly when induced VT is unstable, initial mapping of scar-related VT focuses on locating the abnormal myocardial substrate during stable sinus or paced rhythm (substrate mapping). When VT is stable, mapping is performed during VT to delineate reentry circuit isthmuses and exits from activation mapping and entrainment mapping. Substrate mapping is also often used in combination with mapping during VT in order to minimize the time spent in VT, limiting the need for cardioversion and potential haemodynamic compromise.

**Substrate and voltage mapping**

Defining the area of scar for substrate mapping necessitates an electroanatomical mapping system (Figure 2B).\textsuperscript{30} Substrate mapping may include voltage and pace mapping and registration of characteristic electrogram features suggesting abnormal myocardium. Ablation can be guided by substrate mapping alone, but often identifies a relatively large area of potential scar and reentry circuits.\textsuperscript{31} Combining substrate mapping with short episodes of activation and entrainment mapping, even in patients with unstable VT, will increase the likelihood of successful VT ablation.\textsuperscript{32}

The amplitude (voltage) of an electrogram recorded by the mapping catheter at each site is related to the mass of the...
underlying myocardium and is reduced when the myocardium is replaced by scar. For bipolar recordings, an electrogram amplitude of <1.5 mV indicates a region of scar (Figure 3). A very low amplitude of <0.5 mV is commonly referred to as ‘dense scar’. Voltage maps correlate well with scars demonstrated by cardiac perfusion emission tomography using FDG uptake and delayed gadolinium enhancement on cardiac magnetic resonance (MR) imaging. This 1.5 mV threshold is very specific, but has limited sensitivity, and fails to detect intramural or epicardial scar. Unipolar recordings have a deeper ‘field of view’, and unipolar voltage mapping using a cut-off threshold of <8.3 mV for abnormal tissue in dilated CM and <5.5 mV in ARVC/D was able to identify epicardial scar at the time of endocardial mapping, suggesting the need to proceed with epicardial ablation. With all voltage maps, care must be taken to recognize low voltage due to poor catheter tip-to-tissue contact, rather than true scar.
During substrate mapping, other abnormal electrogram features are noted and may be marked individually on the map. At areas of slow conduction, asynchronous activation of myocyte bundles may give rise to electrograms with multiple low-amplitude components, indicating abnormal conduction, as do late potentials, defined as discrete potentials separated from the main ventricular electrogram, and that may occur after the QRS complex. Recognition of isthmuses can be accomplished by assessing the effect of pacing at the site during VT (entrainment mapping) (Figure 2D). At times, an isthmus is recognized because simple mechanical pressure from the catheter terminates VT or a stimulus that does not appear to capture terminates VT. Recognition of isthmuses can be accomplished by assessing the effect of pacing at the site during VT (entrainment mapping) (Figure 2D).

Catheter ablation

After identification of a reentry circuit isthmus, ablation is performed, either during VT with observation for VT termination (Figure 2E) or during sinus rhythm. Radiofrequency ablation is most commonly used for ablation. In contrast to ablation for most supraventricular tachycardias, large lesions are desirable for scar-related reentry circuits. Irrigated-tip RF ablation catheters are commonly used in which saline flows through pores in the ablation electrode, cooling it to allow greater energy application with less risk of scar formation to create deeper lesions. During irrigated LV endocardial and epicardial ablation, power is set to 30–50 W with a maximum temperature of 43°C and a flush rate of 17–25 mL/min. Steam pans due to explosion of steam if tissue temperature reaches 100°C, which can result in cardiac perforation in thin-walled chambers, and is rarely seen in LV scar-related VT. The volume status needs to be carefully monitored since a significant amount of irrigation fluid may be administered during mapping and ablation.

Potential complications include stroke, cardiac tamponade, valve injury, and atrioventricular block. Procedure-related death is seen in 0–3% of the patients, but is most commonly due to

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**Figure 2** Findings illustrating some of the steps in an ablation procedure for ventricular tachycardia due to prior anterior wall infarction are shown. In (A), programmed ventricular stimulation induces sustained monomorphic ventricular tachycardia. The 12-lead electrocardiographic leads are shown from the top and a recording from the right ventricular apex at the bottom of the panel. Induced ventricular tachycardia has a right bundle branch block—like configuration in V1 and a frontal plane axis directed superiorly, indicating an exit in the inferior wall of the left ventricle. Left ventricular access is obtained and a voltage map of the left ventricular created (B). The map is shown as viewed from the left anterior oblique projection. Purple indicates a normal voltage of >1.5 mV. Blue, green, yellow, and red indicate progressively lower voltages, consistent with the anterior wall infarct scar. A grey region indicates an electrically unexcitable scar, where pacing fails to capture. During creating of the voltage map, pace mapping is performed at low-voltage sites (C). At the site indicated by the arrow, pacing produces a QRS that has a right bundle branch block configuration and similar, although not identical, axis to the induced ventricular tachycardia, suggesting that the ventricular tachycardia exit is in that region of the ventricle. The stimulus to QRS interval of 90 ms is consistent with slow conduction away from the pacing site. In this case, a decision was made to induce ventricular tachycardia and assess the relation of the region identified by pace mapping to the ventricular tachycardia. (D) Findings during induced ventricular tachycardia. First, note that the ventricular tachycardia that was induced is different (V1 is isoelectric and the axis is directed more leftward) from the initial ventricular tachycardia shown in (A), possibly indicating a different reentry circuit. However, at this site, electrograms recorded from the mapping catheter electrodes (Abl d, Abl m, and Abl p) have diastolic components and double potentials, suggesting a possible isthmus. Entrainment mapping was performed at this site by pacing slightly faster (cycle length 370 ms) than the ventricular tachycardia (cycle length 385 ms) and the last three stimuli of the pacing train are shown. These capture and accelerate the ventricular tachycardia to the pacing rate without changing the QRS morphology, which is also consistent with pacing from a reentry circuit isthmus. Based on these findings, radiofrequency ablation was performed at this site during ventricular tachycardia (E). Ablation terminated ventricular tachycardia, providing further evidence that the site is in the ventricular tachycardia reentry circuit. Additional radiofrequency lesions were then placed in the region to attempt to ensure adequate ablation. Programmed ventricular stimulation was then repeated (data not shown) to determine whether this ventricular tachycardia has been rendered not inducible and determine whether other ventricular tachycardias are inducible, which may warrant further ablation. A purely substrate-based approach, ablation during sinus rhythm at all sites with late potentials or where pacing matched the induced ventricular tachycardia morphologies, could also have been considered and would have included the area identified by mapping during ventricular tachycardia.
uncontrollable VT when the procedure fails. Following successful ablation, periprocedural death may be the result of sepsis or progressive heart failure. If attempts at endocardial and epicardial ablation fail, particularly if the reentry circuit is located deep within scar involving the septal myocardium, transcoronary ethanol ablation may be used.

**Procedural endpoints**

It is important to note that to date, the best ablative strategy is unknown. No randomized trial has compared ablation during VT with a substrate-based approach. In addition, no randomized data are currently available on the best substrate-based approach, that is, comparing isthmus (channel) ablation to a linear lesion approach targeting VT exit sites vs. an encircling technique.

The common absence of a 12-lead ECG of the clinical VT and the presence of multiple inducible VTs often complicate the assessment of the effect of the ablation at the end of the procedure and definition of the ablation endpoint. If mapping is performed during VT, the minimal procedural endpoint should be non-inducibility of all clinical VTs. Many centres also target all inducible VTs that are slower than the slowest clinical VT. After ablation of the clinical VT, faster VTs may remain inducible with aggressive programmed stimulation.

While non-inducibility has been linked to better outcome, Calkins et al. reported no apparent benefit in outcome if all map-pable VTs were rendered non-inducible. In the VTACH study, successful ablation was defined as non-inducibility of any VT. In a subanalysis of the VTACH study, Wissner et al. reported that acute ablation success did not translate into greater freedom
from recurrent VT or ventricular fibrillation. In addition, inducibility of VT at the time of catheter ablation had no impact on long-term freedom from ventricular arrhythmias.

If a substrate-based mapping and ablation approach is utilized, acute procedural success can be defined as the absence of all channels inside the area of interest or linear ablation lines at target sites along the infarct scar.9

**Catheter ablation in patients with ischaemic heart disease**

**Currently available evidence from clinical trials**

To date, results of two randomized prospective multicentre studies have been published in patients with ICM and VT undergoing prophylactic catheter ablation to prevent further VT (Table 1).9,10 The SMASH-VT study, published in 2007, assessed the role of catheter ablation in patients with previous myocardial infarction and reduced LV ejection fraction (LVEF) undergoing ICD implantation for secondary prevention of sudden cardiac death.10 Patients presented with haemodynamically unstable ventricular arrhythmias, e.g. ventricular fibrillation, haemodynamically intolerable VT or syncope, and inducible VT at the time of the electrophysiology study. To foster recruitment, patients with appropriate first-time ICD shock after implantation of an ICD for primary prevention were eligible. None of the patients received Class I or III antiarrhythmic drug therapy. The control arm underwent ICD implantation only. Importantly, catheter ablation was performed utilizing a substrate-guided approach. This included mapping of the ventricular substrate in sinus rhythm without the need for VT induction. The primary endpoint was survival free from VT (appropriate anti-tachycardia pacing or ICD shock therapy). During an average follow-up period of 22.5 ± 5.5 months, there was a significant decrease in appropriate ICD therapy in the ablation group compared with the control arm (12 vs. 33%, \( P = 0.007 \)). In addition, the number of appropriate shock deliveries was reduced and there was a trend to a reduction in the number of patients with electrical storm. Catheter ablation had no significant impact on mortality. Limitations of the SMASH-VT study include the long recruitment period, no standardized anti-tachycardia pacing algorithm, and selection of only high-volume, highly experienced ablation centres.

The multicentre VTACH study, published in 2010, assessed the role of VT ablation in patients with prior myocardial infarction, reduced EF ≤50%, and haemodynamically stable VT.9 One hundred and ten patients were prospectively randomized to ICD implantation only or VT ablation at the time of ICD implantation. Ablation was guided by a combination of substrate mapping, activation mapping, and pace mapping. The primary endpoint was defined as the time to first recurrence of VT or ventricular fibrillation. The use of antiarrhythmic medication was at the discretion of the treating physician. The median time to first recurrence of ventricular arrhythmias was longer in the ablation group than the ICD only group (18.6 vs. 5.9 months). The Kaplan–Meier analysis demonstrated a significantly better rate of survival free from recurrent VT in the ablation group (47 vs. 29%, hazard ratio = 0.61, \( P = 0.045 \)). There was no difference in mortality between groups. Upon subgroup analysis, patients with an EF of ≤30% derived no benefit from catheter ablation, while patients with an EF of >30% demonstrated a statistically significant decrease in arrhythmia recurrence. Quality-of-life assessment showed no difference between groups. Further evidence is needed to assess the exact role of catheter ablation in the subgroup of patients with an EF of >30%, since these patients derived the greatest benefit from VT ablation. Although patients with an EF of ≤30% demonstrated no benefit, the results of VTACH need to be corroborated by future studies. Until then, catheter ablation of VT should not be withheld in patients with low LV function. Lastly, the significantly higher number of centres participating in VTACH compared with SMASH-VT (16 vs. 3) and the non-standardized approach to ablation used in VTACH may explain the higher rate of VT recurrence while findings may be more representative of real-world results.

Three non-randomized studies examined the role of catheter ablation in patients with previous myocardial infarction and multiple episodes of VT, despite antiarrhythmic drug therapy (Table 1).7,11,12 The recurrence rate of VT in these studies was 47, 56, and 49% during a 6-, 8-, and 12-month follow-up period, respectively. While no periprocedural death was reported in the prospective randomized SMASH-VT and VTACH studies, as well as in the prospective, multicentre Euro-VT-Study, Calkins et al.7 and Stevenson et al. reported a procedure-related mortality of 2.7 and 3.0%.9,12

A meta-analysis of trials comparing catheter ablation for VT in patients with structural heart disease to control demonstrated that VT ablation reduced recurrence of ventricular arrhythmias without significant impact on mortality.19

Electrical storm has been defined as three or more separate episodes of VT within a 24 h period and has been associated with increased mortality in patients with ICDs. In patients presenting with electrical storm, catheter ablation may serve as the only viable treatment option if antiarrhythmic therapy fails. Carbucchio et al.8 prospectively assessed the impact of catheter ablation on short- and long-term outcome in 95 patients presenting with electrical storm. Coronary artery disease was present in 76% of
Table I  Prospective studies on catheter ablation of ventricular tachycardia in patients with ischaemic cardiomyopathy

<table>
<thead>
<tr>
<th>Studies</th>
<th>Patients, ( n )</th>
<th>EF (%)</th>
<th>Substrate</th>
<th>Treatment</th>
<th>Type of VT</th>
<th>Mapping</th>
<th>Acute success, ( n ) (%)</th>
<th>FU (ms)</th>
<th>Long-term success</th>
<th>Long-term mortality</th>
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<tr>
<td><strong>Prospective randomized multicentre trials</strong></td>
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<tr>
<td>Kuck et al. 2010(^7)</td>
<td>107</td>
<td>ICM</td>
<td>VT ablation + ICD vs. ICD only</td>
<td>Only stable VT</td>
<td>Mapping during VT/substrate mapping</td>
<td>22.5 (9)</td>
<td>27 (60%)</td>
<td>47%</td>
<td>10%(^a)</td>
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<td>Active</td>
<td>52</td>
<td>34 ± 10</td>
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<tr>
<td>Control</td>
<td>55</td>
<td>34 ± 9</td>
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<tr>
<td>Reddy et al. 2007(^10)</td>
<td>128</td>
<td>ICM</td>
<td>VT ablation + ICD vs. ICD only</td>
<td>All VT</td>
<td>Substrate mapping</td>
<td>22.5 (5.5)</td>
<td>29%</td>
<td>7%(^a)</td>
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<tr>
<td>Active</td>
<td>64</td>
<td>31 ± 10</td>
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<td>Control</td>
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<td><strong>Non-randomized prospective multicentre trials</strong></td>
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<tr>
<td>Tanner 2009</td>
<td>63</td>
<td>30 ± 13</td>
<td>ICM</td>
<td>VT ablation</td>
<td>All VT</td>
<td>Mapping during VT/substrate mapping</td>
<td>51 (81%)</td>
<td>12 (3)</td>
<td>51%</td>
<td>9%(^c)</td>
</tr>
<tr>
<td>Stevenson et al. 2008(^11)</td>
<td>231</td>
<td>25(^d)</td>
<td>ICM</td>
<td>VT ablation</td>
<td>All VT</td>
<td>Mapping during VT/substrate mapping</td>
<td>113 (49%)</td>
<td>6(^e)</td>
<td>53%</td>
<td>18%(^f)</td>
</tr>
<tr>
<td>Calkins et al. 2000(^7)</td>
<td>146</td>
<td>31 ± 13</td>
<td>ICM/NICM</td>
<td>VT ablation</td>
<td>All VT</td>
<td>Mapping during VT/substrate mapping</td>
<td>59 (41%)</td>
<td>8 ± 5</td>
<td>46%</td>
<td>25%(^f)</td>
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<tr>
<td><strong>Non-randomized prospective single-centre trials(^i)</strong></td>
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<tr>
<td>Niwano 2008</td>
<td>58</td>
<td>37 ± 7</td>
<td>ICM/NICM</td>
<td>VT ablation</td>
<td>All VT</td>
<td>Mapping during VT/substrate mapping</td>
<td>43 (74%)</td>
<td>31 ± 22</td>
<td>75%</td>
<td>16%</td>
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<tr>
<td>Carbucicchio et al. 2008(^8)</td>
<td>95</td>
<td>36 ± 11</td>
<td>ICM/NICM/ARVC</td>
<td>VT ablation</td>
<td>Electrical storm</td>
<td>Mapping during VT/substrate mapping</td>
<td>85 (89%)(^h)</td>
<td>22 ± 13</td>
<td>63 (66%)</td>
<td>16%</td>
</tr>
</tbody>
</table>

ICM, ischaemic cardiomyopathy; NICM, non-ischaemic cardiomyopathy; VT, ventricular tachycardia; ICD, implantable cardioverter-defibrillator; ARVC, arrhythmogenic right ventricular cardiomyopathy; FU, follow-up.

\(^a\)P-value not given.

\(^b\)Ablation performed in SR.

\(^c\)Among 53 patients completing follow-up.

\(^d\)Median.

\(^e\)SD not given.

\(^f\)After 12 months.

\(^g\)One-year Kaplan–Meier estimate.

\(^h\)After one to three procedures.

\(^i\)Patients undergoing successful ablation; 35 ± 5 in patients with failed ablation.
<table>
<thead>
<tr>
<th>Study</th>
<th>Patients, n</th>
<th>Substrate</th>
<th>EF (%)</th>
<th>Endocardial mapping (%)</th>
<th>Epicardial access (%)</th>
<th>Acute complications (%)</th>
<th>Acute success (%)</th>
<th>Long-term complications (%)</th>
<th>Long-term success, n (%)</th>
<th>FU (ms)</th>
<th>Mortality during FU (%)</th>
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<tbody>
<tr>
<td>Multicentre experience</td>
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| Dukkipati et al. 2011       | 10          | HCM       | 57 ± 13| 10/10 (100%)            | 10/10 (100%)          | None                    | 8/9 (89%)         | 1/10 (10%)                  | 7/9 (70%)
|                             |             |           |        |                         |                       |                         |                   |                             |                          | 37.4 ± 16.9 | Not reported           |
| Sacher 2008                 | 149         | NICM      | 39 ± 16| 149/149 (100%)          | Not reported          | 12/195 (6.2%)           | 99/195 (51%)      | Not reported                | 61% after a median
|                             |             |           |        |                         |                       |                         |                   |                             | FU of 1 month              | 40 ± 29 | 26/149 (17%)           |
| Single-centre experience    |             |           |        |                         |                       |                         |                   |                             |                          |         |                        |
| Kaplan et al. 2006          | 8           | Sarcoid   | 34 ± 15| 8/8 RV, 6/8 LV          | 2/8 (25%)             | Not reported            | 2/8 (25%)         | Not reported                | 6/8 (75%) within
|                             |             |           |        |                         |                       |                         |                   |                             | first 6 ms                | (6 ms to 7 yrs) | 1/8 (13%) |
| Soejima et al. 2004         | 28          | DCM       | 30 ± 11| 20/28 (71%)             | 7/28 (25%)            | 2/28 (4%), 1 epi, 1 endo| 17/28 (61%)      | Not reported                | 17/28 (61%)               | 11.1 ± 9.3    | 1/28 (4%)              |
| Hsia et al. 2003            | 19          | NICM      | 34 ± 11| 19/19 (100%)            | None                  | Not reported            | 14/19 (74%)       | Not reported                | 5/19 (26%)               | 22 ± 12  | 4/19 (21%)            |

DCM, dilated cardiomyopathy; NICM, non-ischaemic cardiomyopathy; HCM, hypertrophic cardiomyopathy; FU, follow-up.

aExcluding one patient without attempted ablation.
bDefined as no ICD shock during follow-up.
cAccording to a total of 195 procedures performed in 149 patients.
dDefined as no VT recurrence.
the patients. Ventricular tachycardia recurred in 34% but only 8% had recurrence of electrical storm during a median follow-up of 22 ± 13 months. Two recent studies highlighted the importance of early intervention and the need for a network of collaborating medical centres to optimize ablative treatment in patients suffering from electrical storm.50,51

Catheter ablation in patients with non-ischaemic cardiomyopathy

There are no prospective, randomized studies examining the role of catheter ablation in patients with NICM or right ventricular cardiomyopathies. Non-randomized studies have demonstrated the feasibility of catheter ablation in this heterogeneous patient population25,52–54 (Table 2). The pathophysiological substrate is more varied than in ICM patients. In NICM, areas of scar containing VT circuits are often located along the base of the LV around the mitral valve annulus.52 In ARVC/D, fibro-fatty replacement of normal myocardial tissue involves the perivalvular area around the tricuspid annulus and pulmonary valve annulus as well as the right ventricular free wall and the septum, while the apex is typically spared.55 In both entities, epicardial ablation is more frequently required compared with patients with ICM.56,57 Consistent with the pathological observation that in myocardial infarction, the wavefront of necrosis initiates at the subendocardial layer spreading outward, while in patients with ARVC/D, fibro-fatty replacement progresses from the subepicardial to the endocardial layer.58,59 Applying a substrate-based mapping technique combining pacing and targeting late potentials for ablation appears to be less successful in patients with NICM than in patients with ischaemic heart disease.60 Overall, the success rate of catheter ablation for the treatment of NICM is lower than in patients with prior myocardial infarction.25,52,60 This is probably related to the progressive nature of the disease and the prevalence of epicardial and intramural reentry circuits. In patients with ARVC/D undergoing catheter ablation, freedom from VT recurrence ranged between 77 and 89% during mid-term follow-up.55,57 However, due to the progressive course of ARVC/D, late recurrence is expected.

Ventricular tachycardia originating from the Purkinje system

Occasionally, VT originates from reentry or automaticity in a diseased Purkinje system. Most commonly, this takes the form of bundle branch reentry characterized by a wide QRS complex tachycardia with a typical bundle branch block pattern. Underlying conduction abnormalities in the His–Purkinje system are frequently present with interventricular conduction delay or left bundle branch block and prolongation of the His to ventricle interval during sinus rhythm that will typically shorten during bundle branch reentrant VT.51 Slowed anterograde conduction within the left bundle branch allows a fortuitously timed ventricular extrasystole to advance retrograde up the left bundle branch, anterograde down the right bundle branch, and then through the septum, completing the reentrant circuit. Catheter ablation of the right bundle branch is curative; however, due to significant disease of the left bundle branch, severely impaired anterograde atrioventricular conduction is often present.62 Most affected patients have significant underlying heart disease and many have additional scar-related VTs, such that implantation of an ICD is often warranted.

Unresolved issues and future outlook

Therapy to prevent VT is still warranted in patients with ICDs. Although ICDs provide effective termination of VT and prevention of sudden death, episodes of VT reduce quality of life in patients with ICDs.5 Recurrent shocks often cause severe psychological impairment and post-traumatic stress syndrome. In addition, VT predicts an increased risk of death and heart failure hospitalizations in ICD recipients.63 According to the current international guidelines, catheter ablation of VT serves as an adjunct to antiarrhythmic therapy in patients experiencing appropriate ICD shocks.16 However, early referral for catheter ablation following ICD intervention has the potential to improve patient mortality as well as quality of life.64 In order to evaluate the role of catheter ablation in patients with prior myocardial infarction and recurrent VT despite antiarrhythmic drug therapy, the VANNISH study will randomize patients to continued antiarrhythmic drug therapy or catheter ablation (ClinicalTrials.gov identifier: NCT00905853). The primary endpoint is a composite of mortality and recurrent VT.

Although single-centre series often report favourable outcomes in 70–80% of the patients, in multicentre trials in patients with drug-refractory, recurrent VT, as discussed above, approximately half of the patients experience at least one episode of recurrent VT after ablation, although the frequency of VT episodes is substantially improved in the majority. Judging the true effect of catheter ablation on patient outcome is often complicated by post-procedural continuation of antiarrhythmic medication. Furthermore, the various mechanisms of VT recurrence following catheter ablation are poorly understood. Inadequate lesion formation, deep intramural reentry circuits, or previously dormant reentry circuits might contribute to recurrence.65 It is hoped that new ablation technologies including intramural needle ablation catheters may help to overcome some limitations. Advances in imaging technology may aid in the detection of appropriate ablation targets. Recently, contrast-enhanced MR imaging has demonstrated a value in delineating conducting channels within scar as potential targets for ablation.66 Furthermore, in procedural contact-force measurement may improve transmural lesion formation. An epicardial substrate may be more commonly detected in patients undergoing repeat ablation.26 To date, there is no consensus on the appropriate timing of epicardial mapping and ablation.

In patients with VT and relatively good ventricular function (LVEF ≥0.35), ICDs have not been shown to improve mortality.67 In the VTACH study, patients with an EF of >30% and stable VT demonstrated a significant reduction in recurrent ventricular
arrhythmias. Whether these patients could be safely spared ICD implantation following successful catheter ablation is not known.

Patients with recurrent VT often have relatively severe heart disease. Data regarding efficacy and risks of catheter ablation is derived largely from experienced centres. Although we believe that the present data support the earlier use of catheter ablation, prior to multiple symptomatic VT episodes that may result in severe psychological trauma, ablation should continue to be the prelude of experienced centres, appropriately equipped to manage the occasionally serious complications that can occur. With broader use, continued assessment of outcomes is warranted.

There is no evidence that catheter ablation of VT reduces mortality. There is a future, sufficiently powered, prospective randomized study using standardized ablation and follow-up protocols will need to answer this important question.

Clinical summary

Catheter ablation methods have improved substantially over the past two decades. During this time, ICDs have emerged as the major therapy for protecting patients from sudden death, but have created a cohort of patients with ventricular arrhythmias that reduce quality of life and are associated with increased mortality. Antiarrhythmic drug therapy for ventricular arrhythmias has been disappointing. In view of these considerations, catheter ablation, performed in an experienced centre, should be considered early in the management of patients with heart disease who suffer recurrent symptomomatic monomorphic VT. Selection of this therapy should be made with careful consideration of risks and efficacy, which is importantly influenced by the type and severity of the underlying heart disease.

Conflict of interest: none declared.

References


support for catheter ablation of unstable ventricular arrhythmias in high-risk patients. Herz 2009;34:545–552.

