**Novel therapeutic concepts**

**The role of catheter ablation in the management of ventricular tachycardia**

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The treatment strategy for ventricular tachycardia (VT) is guided by patient symptoms, the risk of sudden death estimated by VT mechanism and underlying cardiac structure, and the risk vs. benefit ratio of potential therapies. Over the last few decades, catheter ablation has emerged as the primary treatment of idiopathic VT and become an important management strategy in reducing VT burden in patients with structural heart disease. This article reviews the technique and outcomes of catheter ablation for ventricular arrhythmias as well as potential future directions for this procedure.

**Keywords**

Catheter ablation • Ventricular tachycardia

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**Introduction**

Ventricular arrhythmias represent a clinical spectrum ranging from premature ventricular complexes (PVCs) to monomorphic ventricular tachycardia (VT) and polymorphic VT/ventricular fibrillation (VF). These arrhythmias can occur in the presence and absence of structural heart diseases (SHD). In patients with a structurally normal heart, ventricular arrhythmia can be subcategorized as idiopathic and those resulting from an inherited ion channel abnormality. Catheter ablation of ventricular arrhythmias has evolved considerably since first described >3 decades ago, especially in management of idiopathic VT and VT with SHD. The role of catheter ablation in inherited arrhythmias remains limited. Therefore, this review article will focus on describing the techniques and outcomes of catheter ablation of idiopathic VT, and VT in patients with SHD. We will also briefly review the history of catheter ablation for ventricular arrhythmias and attempt to predict how this procedure will evolve over the next decade.

**Historic perspective**

The first electrophysiology evaluation of VT was performed in 1972 and was rapidly followed by reports of further characterizations of VT with programmed electrical extra-stimulation and the development of endocardial mapping.²,³ The early reports describing the technique and outcomes of surgical VT ablation began appearing in the literature in 1978.⁴–⁶ Table 1 shows a series of reports of surgical ablation of VT published between 1981 and 1988.⁴–¹³ Between 72 and 97% of patients were rendered non-inducible and the recurrence rates ranged from 5 to 33%. Although these were impressive results, the operative mortality was high, ranging from 8 to 17%. It was for this reason that surgical ablation of VT was ultimately abandoned in favour of catheter-based approaches.

The field of catheter ablation was launched in 1982, shortly after the appearance of initial reports of high-energy DC ablation (fulguration) of the atrioventricular node.¹⁴–¹⁶ This ablation strategy was initially applied to a series of three patients with refractory VT in 1983.¹ The report described delivering 300 J shocks for ablation of refractory VT in the right ventricular outflow tract (RVOT), left ventricular septum, and mid right ventricular septum of a patient without SHD and two patients with prior myocardial infarction, one of whom had failed a prior surgical VT ablation procedure. Fontaine et al. was also an early pioneer in DC shock ablation of VT, reporting both a high clinical efficacy of this procedure (49% success, 43% partially success, and 92% clinical efficacy) and a high complication rate including an 8.5% mortality during 3-month of follow-up.¹⁷ Despite high clinical efficacy, fulguration was associated with barotrauma and arcing, resulting in rupture of cardiac structures, myocardial dysfunction, arrhythmias, and death.¹⁸ Within 6 years of these early reports of using DC shock ablation for treatment of...
VT, radiofrequency energy emerged on the scene and was quickly determined to be a safer and more controllable energy source for catheter ablation procedures. Thus, DC shock was quickly abandoned in favour of radiofrequency catheter ablation.

**Technique and role of catheter ablation for treatment of idiopathic ventricular arrhythmias**

**Overview**

Catheter ablation of idiopathic ventricular arrhythmias using RF energy was first reported in the late 1980s. This procedure was shown not only to be technically feasible but associated with a low incidence of complications and high acute efficacy for achievement of long-term cure. The procedure quickly gained worldwide acceptance and is one of the most common types of ablation procedures performed today.

**Idiopathic ventricular tachycardia**

Idiopathic VT is defined based upon its occurrence in a patient without evidence of SHD and without evidence of a genetic arrhythmia syndrome. Patients with idiopathic VT most commonly present with palpitations, but more severe symptoms including syncope are rarely observed. Patients with idiopathic VT have a low risk of sudden death, and placement of an ICD is generally not warranted. Idiopathic VT can be sub-classified based on several criteria such as the site of origin, mechanism, response to pharmacologic therapy, and arrhythmia pattern. Figure 1 and Table 2 demonstrate the different clinical characteristics and electrocardiographic (ECG) findings associated with idiopathic VT arising from the most common sites in the right and left ventricle: (1) outflow tract VT, (2) fascicular VT, (3) intra-cavitary VT, (4) perivalvular VT, and (5) epicardial VT.

**Outflow tract ventricular tachycardia**

Right ventricular outflow tract VT is the most common form of outflow tract VTs, accounting for 70–80% of patients. Less commonly, outflow tract VTs originate from the LVOT, aortic cusps, and aorto-mitral continuity. While pre-cordial R-wave transition (from rS to Rs) typically occurs at V3 or later in RVOT VT, the transition normally occurs at or before V3 in LVOT VT. Because the transition in V3 could be either RVOT or LVOT, the V2 transition ratio \[ R / (R + S)_{LVOT} \] can be used to determine whether the site of origin is from RVOT, or LVOT. A V2 transition ratio \[ \geq 0.60 \] predicted LVOT origin with 91% accuracy. Additionally, VT pre-cordial transition occurring later than sinus rhythm pre-cordial transition has been reported to exclude an LVOT origin with 100% accuracy. Outflow tract VT is often induced with isoproterenol infusion and burst pacing and can be terminated with high-dose adenosine, consistent with cathecholamine-induced c-AMP-mediated delayed afterdepolarizations and triggered activity as mechanism.

**Fascicular ventricular tachycardia**

The term ‘fascicular VT’ can be applied to four different entities: (i) verapamil-sensitive left fascicular VT, (ii) focal Purkinje VT (propranolol-sensitive automatic VT), (iii) Purkinje fiber-mediated VT post-infarction, and (iv) bundle branch reentry VT and inter-fascicular reentry. Some of these can be broadly considered to be a form of idiopathic VT, whereas others, like bundle branch reentry, are almost always observed in patients with SHD. The mechanisms also differ. While the mechanism of focal Purkinje VT is abnormal automaticity and/or triggered activity, reentry is the mechanism of the other types. In structurally normal hearts, the most common is verapamil-sensitive left fascicular VT. Focal Purkinje VT is usually observed in patients with ischaemic heart disease but may also be observed in patients with structurally normal hearts.

Idiopathic fascicular VT or verapamil-sensitive left fascicular VT is the most common form of idiopathic VT arising from the left ventricle. It was first described in 1979 as a diagnostic triad: (i) induction with atrial pacing, (ii) right bundle branch block pattern (RBBB

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**Table 1** Surgical treatment in ventricular tachycardia

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>N</th>
<th>CAD (%)</th>
<th>EF (%)</th>
<th>Guiding technique</th>
<th>Follow-up duration (months)</th>
<th>Acute success (%)</th>
<th>Recurrence rate (%)</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Guiraudon et al.</td>
<td>1981</td>
<td>23</td>
<td>0</td>
<td>NA</td>
<td>Mapping</td>
<td>28 ± 23</td>
<td>79</td>
<td>33</td>
<td>17</td>
</tr>
<tr>
<td>Josephson et al.</td>
<td>1982</td>
<td>60</td>
<td>100</td>
<td>27 ± 10</td>
<td>Mapping</td>
<td>19 ± 11</td>
<td>70</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>Moran et al.</td>
<td>1983</td>
<td>68</td>
<td>96</td>
<td>NA</td>
<td>Mapping</td>
<td>20.4</td>
<td>89</td>
<td>6</td>
<td>12</td>
</tr>
<tr>
<td>Miller et al.</td>
<td>1984</td>
<td>100</td>
<td>100</td>
<td>28 ± 9</td>
<td>Mapping</td>
<td>23 ± 17</td>
<td>72</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>Platia et al.</td>
<td>1986</td>
<td>28</td>
<td>100</td>
<td>25</td>
<td>Mapping</td>
<td>25</td>
<td>82</td>
<td>10</td>
<td>11</td>
</tr>
<tr>
<td>Krafchek et al.</td>
<td>1986</td>
<td>39</td>
<td>100</td>
<td>32 ± 11</td>
<td>Mapping (90%)</td>
<td>22 ± 17</td>
<td>97</td>
<td>9</td>
<td>10</td>
</tr>
<tr>
<td>Garan et al.</td>
<td>1986</td>
<td>36</td>
<td>97</td>
<td>29 ± 9</td>
<td>Mapping</td>
<td>25 ± 15</td>
<td>66</td>
<td>7</td>
<td>17</td>
</tr>
<tr>
<td>Swerdlov et al.</td>
<td>1986</td>
<td>105</td>
<td>93</td>
<td>34 ± 11</td>
<td>Mapping (79%)</td>
<td>23 ± 21</td>
<td>68</td>
<td>22</td>
<td>16</td>
</tr>
<tr>
<td>Ostermeyer et al.</td>
<td>1987</td>
<td>93</td>
<td>100</td>
<td>28 ± 14</td>
<td>Mapping</td>
<td>26 (4–96)</td>
<td>87</td>
<td>10</td>
<td>11</td>
</tr>
<tr>
<td>Haines et al.</td>
<td>1988</td>
<td>45</td>
<td>100</td>
<td>34 ± 12</td>
<td>Mapping</td>
<td>19 ± 12</td>
<td>85.3</td>
<td>11</td>
<td>9</td>
</tr>
</tbody>
</table>

N, number of patients; CAD, coronary artery disease; EF, ejection fraction.
like morphology and left superior-axis configuration with relatively narrow QRS (120–140 ms), and (iii) manifestation in patients without SHD. Subsequently, in 1981, Belhassen et al. demonstrated the pharmacologic characteristic of verapamil sensitivity as the fourth main feature.34,35 Normally, this form of VT presents as paroxysms of sustained VT, often exercise related, and responds to carotid sinus pressure and Valsalva maneuver. Age at presentation is typically 15–40 years and affected individuals are predominantly men (60–80%). The clinical presentation includes palpitations, lightheadedness, and less frequently, syncope.

The mechanism of verapamil-sensitive VT is reentry, likely incorporating abnormal Purkinje tissue with slow, decremental, calcium-dependent conduction serving as the antegrade limb and fascicle (most commonly posterior) as the rapid retrograde limb. The electrocardiographic findings vary depending on the exit site to the myocardium, which is associated with the retrograde limb. The
<table>
<thead>
<tr>
<th>Sites of origin</th>
<th>Prevalence</th>
<th>Mechanism</th>
<th>ECG findings</th>
<th>Presentation</th>
<th>Medication therapy</th>
<th>Ablation target</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Outflow tract VT</td>
<td>70–80% of idiopathic VT</td>
<td>DAD-related triggered activity</td>
<td>LBBB inferior axis Delayed R wave transition (at or after V3, or later than sinus rhythm) V2 transition ratio* &lt; 0.6</td>
<td>Exercise-induced sustained VT Incessant NSVT PVCs Tachycardia-related cardiomyopathy</td>
<td>Adenosine β-Blocker Verapamil/ diltiazem IA, IC, III AAD</td>
<td>Presystolic signal Perfect pace match with short S-QRS QS at unipolar</td>
</tr>
<tr>
<td>RVOT</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVOT, aortic cusps, AMC</td>
<td>16% of outflow tract VT</td>
<td>DAD-related triggered activity</td>
<td>LBBB inferior axis Early R wave transition V2 transition ratio ≥ 0.6 Sometimes presents with &gt; 1 morphology</td>
<td>Same as RVOT Idiopathic VF (PVC-induced VF) (LVOT)</td>
<td>Adenosine β-Blocker Verapamil/ diltiazem IA, IC, III AAD</td>
<td>Presystolic signal Perfect pace match with short S-QRS QS at unipolar (except cusp or great vessel foci)</td>
</tr>
<tr>
<td>2. Fascicular VT</td>
<td>Verapamil-sensitive VT 10–15% of idiopathic VT</td>
<td>Macro-reentry</td>
<td>Relatively narrow QRS LPF: RBBB, LAD LAF: RBBB, RAD</td>
<td>Same as RVOT</td>
<td>Verapamil</td>
<td>During VT: diastolic potential, anterograde Purkinje potential During sinus: diastolic potential or linear lesion at mid-inferiorseptum, perpendicular to the long axis of LV</td>
</tr>
<tr>
<td>Focal Purkinje VT (Propanolol-sensitive automatic VT)</td>
<td>Rare</td>
<td>Enhanced automaticity</td>
<td>Relatively narrow QRS. Depending on origin: LBBB (from RV distal Purkinje) or RBBB (from LV distal Purkinje)</td>
<td>Exercise or cathelcolamine related. Incessant NSVT Polymorphic/ monomorphic VT Idiopathic VF (PVC-induced VF)</td>
<td>β-Blocker Lidocaine Adenosine</td>
<td>The earliest presystolic Purkinje potential</td>
</tr>
<tr>
<td>3. Intra-cavity VT</td>
<td>Papillary muscle 2.4–5.2% of idiopathic VT</td>
<td>Enhanced automaticity vs. DAD-related triggered activity</td>
<td>APM: RBBB (q in V1), inferior axis PPP: RBBB (q in V1), left superior axis. RV PM: LBBB, more variable</td>
<td>Same as RVOT Idiopathic VF (PVC-induced VF) (LVOT)</td>
<td>Same as outflow tract VT, not well studied</td>
<td>Same as RVOT</td>
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<tr>
<td></td>
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<td></td>
<td>ICE can be helpful for catheter navigation and positioning. May require wide area ablation or ablation at both sides of papillary muscle</td>
</tr>
<tr>
<td></td>
<td>Moderator band 2.5% of idiopathic VT</td>
<td>Enhanced automaticity vs. DAD-related triggered activity</td>
<td>LBBB with pre-cordial transition later than V4 or later than sinus rhythm. Left superior axis Relatively narrow QRS</td>
<td>NSVT or monomorphic VT Idiopathic VF (PVC-induced VF)</td>
<td>Same as outflow tract VT, not well studied</td>
<td>Same as RVOT</td>
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<td></td>
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<tr>
<td>4. Perivalvular VT</td>
<td>Tricuspid annulus 8% of idiopathic VT</td>
<td>Enhanced automaticity vs. DAD-related triggered activity</td>
<td>LBBB R or r in lead I, R in V6 A negative component (QS, qs, Qr, or qr pattern) in lead aVR</td>
<td>Same as RVOT</td>
<td>Same as outflow tract VT, not well studied</td>
<td>Same as RVOT</td>
</tr>
</tbody>
</table>

Continued
<table>
<thead>
<tr>
<th>Sites of origin</th>
<th>Ablation target</th>
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<th>Mechanism</th>
<th>ECG findings</th>
<th>Presentation</th>
<th>Medication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral annulus</td>
<td>Same as RVOT</td>
<td>Same as RVOT</td>
<td>Enhanced automaticity</td>
<td>RBBB, early transition in V1 or between V1 and V2</td>
<td>Same as RVOT</td>
<td>Same as RVOT</td>
</tr>
<tr>
<td>Epicardial VT</td>
<td>Same as outflow tract VT, not well studied</td>
<td>Same as outflow tract VT, not well studied</td>
<td>Enhanced automaticity vs. DAD-related triggered activity</td>
<td>LBBB, inferior axis, abrupt loss of R waves in V2</td>
<td>Same as RVOT</td>
<td>Same as RVOT</td>
</tr>
</tbody>
</table>

VT, ventricular tachycardia; RBBB, right bundle branch block; LBBB, left bundle branch block; PVCs, premature ventricular complexes; AAD, anti-arrhythmic drugs; NSVT, non-sustained ventricular tachycardia; DAD, delayed after-depolarization; LAD, left-axis deviation; RAD, right-axis deviation; RVOT, right ventricular outflow tract; LVOT, left ventricular outflow tract; LAD, left anterior descending; RAD, right anterior descending. *AIV/GCV: anterior interventricular vein/greater cardiac vein.*

Table 2 continued

<table>
<thead>
<tr>
<th>Sites of origin</th>
<th>Ablation target</th>
<th>Ablation therapy</th>
<th>Mechanism</th>
<th>ECG findings</th>
<th>Presentation</th>
<th>Medication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epicardial VT</td>
<td>Same as outflow tract VT, not well studied</td>
<td>Same as outflow tract VT, not well studied</td>
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most common subtype (90–95%) is ‘left posterior fascicular (LPF) VT’, following by ‘left anterior fascicular (LAF) VT’. The rarest form is ‘upper septal fascicular VT’ presenting as a narrow complex rhythm that involves simultaneous anterograde activation of LAF and LPF with retrograde conduction over a separate fascicle inserting into the left bundle branch. However, RBBB and left bundle branch block pattern (LBBB) like morphologies have also been reported.

**Papillary muscle ventricular tachycardia**

Approximately 7% of idiopathic ventricular arrhythmias originate from left ventricular papillary muscles (PAP), especially the posteromedial PAP. Recent reports have also described idiopathic VT arising from the right ventricular septal PAP. The main mechanism of this arrhythmia is triggered activity. The anterolateral PAP originates from the mid anterolateral LV, whereas the posteroseptal PAP arises from the mid-inferior LV. The triggered PAP VT foci are generally mapped to the base of the PAP muscle and appear to involve the distal Purkinje fibre system. The ECG morphology of PAP VT is RBBB like with superior axis when arising from the posteromedial PAP and inferior axis when arising from the anterolateral PAP. When compared with fascicular VT, those arising from a PAP have a broader QRS complex (150 ± 15 vs. 127 ± 11 ms), and do not exhibit an rsR’ pattern in lead V1.

**Technique of catheter ablation in idiopathic ventricular tachycardia**

Because idiopathic VT is a focal arrhythmia, caused predominantly by triggered activity and increased automaticity (except for verapamil-sensitive fascicular VT), activation mapping and pace mapping are the major approaches used to identify an appropriate ablation site. In general, abolishing of inducible VT or PVCs is the end point for the procedure. In outflow tract VT, localization of the precise source is sometimes challenging because of the complex structural relationships of the RVOT, the LVOT, and the aortic root. A stepwise approach is useful, especially when 12-lead ECG does not provide clear criteria to guide ablation. In the setting of the common LBBB-like inferior axis idiopathic VT, mapping should start from the RVOT. Subsequent mapping of the distal coronary sinus can add useful information and occasionally enable the ablation of an epicardial origin. If activation and pace mapping suggest the origin outside these RVOT and distal coronary sinus, mapping of the LVOT and aortic sinus of Valsalva via retrograde arterial access is the typical next step. If all previously described approaches to mapping and ablation fail, epicardial mapping via percutaneous pericardial access should be considered.

‘Activation mapping’ is the method of choice for mapping idiopathic VT and PVCs. The ability to induce the clinical arrhythmia in the EP laboratory is a very important factor for the success of ablation. Rapid burst pacing, isoproterenol infusion, aminophylline, calcium infusion, or atropine may facilitate arrhythmia induction.31

Earliest local activation on the bipolar electrogram from the responsible focus typically precedes the QRS onset by 15–45 ms during VT or PVCs with a characteristic QS complex in the unipolar signal.41

‘Pace mapping’ refers to pacing at a particular site in the ventricle in an attempt to replicate the exact 12-lead ECG morphology observed during spontaneous ventricular arrhythmias. This approach
to mapping is used to confirm the results of activation mapping and can be of value when VT is difficult to induce and/or clinical PVCs are infrequent, which severely limit the ability to perform activation mapping. Pace maps with identical or near-identical matches of VT morphology in all 12-lead ECG are indicative of the site of origin of the VT/PVCs. The specificity of pace mapping in isolation appears to be lower than that of activation mapping. When possible, it is optimal to rely on both pace mapping and activation mapping. Figure 2 shows a representative electroanatomic map, and merged computed tomography (CT), obtained at the time of ablation of a PVC focus in the anterior RVOT. The ablation lesion was applied at the area of 12/12 ECG pace match and earliest activation.

Ablation of fascicular VT in the absence of SHD depends upon VT mechanism. Idiopathic fascicular VT is often used to describe verapamil-sensitive reentrant VT. However, focal Purkinje VT with a triggered mechanism can also be seen in the absence of SHD. In addition, both of the former should be distinguished from interfascicular reentry, an arrhythmia which typically occurs in the setting of SHD. For focal Purkinje VT, the ablation target is the earliest pre-systolic Purkinje potential during VT. For verapamil-sensitive reentrant VT, two ablation strategies have been described. The traditional approach uses the anterograde Purkinje potentials or diastolic potentials during VT as a target. Verification of target sites can be achieved with entrainment mapping demonstrating concealed
Catheter ablation of idiopathic VT/PVCs is the presence of symptoms. Another common indication is for treatment of presumed PVC-induced cardiomyopathy. Catheter ablation can be performed as first-line treatment. Table 4 shows the indications for VT ablation as delineated in the 2009 EHRA/HRS expert consensus on Catheter Ablation of Ventricular Arrhythmias.

**Outcomes and safety**

Table 2 summarizes the reported outcomes of catheter ablation of idiopathic VT/PVCs. The outcomes are categorized as those associated with ablation of idiopathic VT/PVCs from different locations. Among 815 patients who underwent ablation of outflow tract VT, the acute efficacy varied from 76 to 100%, the recurrence rate was 0–23%. In fascicular VT (42 patients), acute efficacy varied from 85 to 100% and the recurrence rate ranged from 0 to 25%. The intra-cavitary PVC/VT group (96 patients) had not only a high acute success rate ranging from 89 to 100% but also a high recurrence rate with long-term efficacy of 0–58 and 40% after single ablation in PAP and moderator band VT, respectively. In PVCs/VT arising from septal tricuspid and mitral annulus (81 patients), the acute success of catheter ablation varied from 66 to 100%, with the lowest success rate in those arising from septal tricuspid annules. Epicardial idiopathic PVCs/VT ablation carried some major limitations by locations including inability to access the earliest site of activation, inadequate power delivery in the coronary sinus, and proximity to coronary arteries. The acute success in this group varied from 44 to 100% with recurrence rate up to 25% in the apical crux. When evaluating the overall success rate of idiopathic VT, a recent large multicenter study including 1185 patients reported an acute success rate of 84% and a long-term success rate of 71%, in the absence of anti-arrhythmic medications. The complication rate was low with 2.4% incidence of major complications. The only predictor of long-term success is RVOT PVC location. Complications of catheter ablation of idiopathic VT/PVC ablation are rare, with a reported complication rate of 3–5%. Access site vascular complications are most frequent. Less common complications include cardiac tamponade/hemopericardium thromboembolic events, AV block, and coronary artery injury.

The common causes of failed ablation in idiopathic PVCs/VT include imprecise mapping, inability to reach the site of origin, especially intramural and epicardial locations, limited power delivery, inadequate catheter stability, and paucity of PVCs during the procedure. Several methods have been used to overcome these limitations including a stepwise approach for outflow tract VT ablation, multi-electrode array sampling multiple sites simultaneously, using ICE to navigate the catheter in PAP PVCs/VT, adjusting the level of sedation along with isoproterenol or calcium infusion to increase intra-procedural PVCs, epicardial ablation or using contact-force ablation catheters.

**Indications for ablation of idiopathic ventricular tachycardia**

The primary indication for catheter ablation of idiopathic VT and PVCs is the presence of symptoms. Another common indication is for treatment of presumed PVC-induced cardiomyopathy. Catheter ablation can be performed as first-line treatment. Table 4 shows the indications for VT ablation as delineated in the 2009 EHRA/HRS expert consensus on Catheter Ablation of Ventricular Arrhythmias.

**Pathophysiology of ventricular tachycardia in patients with structural heart disease**

Ventricular tachycardia in patients with SHD most commonly results from reentry involving a region of ventricular scar. The scar is commonly caused by a prior myocardial infarction. Other causes of ventricular scar include non-ischaemic cardiomyopathy (NICM), surgical incisions, and other less common conditions such as arrhythmogenic right ventricular cardiomyopathy (ARVC), sarcoidosis, and Chagas disease. On pathologic analysis, ventricular scar consists of regions of dense fibrosis with collagen and regions of fibrosis interspersed with surviving myocyte bundles. The dense fibrosis creates conduction block, whereas the regions of fibrosis interspersed with viable myocytes lead to slow conduction and provide a fertile substrate for reentry. Figure 3 shows a schematic of the components of the VT circuit as described by Stevenson et al. These circuits are often complex and demonstrate variable involvement of the endocardium, mid-myocardium, and epicardium. The scar area in SHD is often extensive, contains false isthmuses (bystanders), and typically has multiple potential reentry circuits or multiple exit sites with shared isthmus. The critical isthmus consists of a proximal part (entrance), a central part, and an exit. The exit site of the critical isthmus gives rise to the QRS complex. Therefore, it is very important to have a 12-lead ECG of clinical (spontaneous) VT available to help identify and target the clinical VT. In patients with an implantable cardioverter defibrillator (ICD) who do not have a 12-lead ECG of the VT available, the VT cycle length, and ICD far-field electrogram morphology during spontaneous or induced VT can be helpful.

**Catheter ablation of ventricular tachycardia in patients with structural heart disease**

**Technique of ventricular tachycardia mapping of ventricular tachycardia in structural heart disease**

The main goal of VT mapping in patients with SHD is identification of the site of origin for triggered activity or the critical isthmus for reentrant VT. Figure 4 shows the mapping and ablation technique employed for catheter ablation of VT in patients with SHD, based on haemodynamic stability during tachycardia, stability of VT re-entry circuit (stable morphology and cycle length), and inducibility during the procedure.
Table 3 Outcomes of catheter ablation in idiopathic ventricular tachycardia

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>N</th>
<th>Age (years)</th>
<th>Prevalence</th>
<th>PVC locations</th>
<th>Follow-up (months)</th>
<th>Acute success</th>
<th>Recurrence</th>
<th>Major complications</th>
</tr>
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<tbody>
<tr>
<td>Outflow tract VT</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>O'Donnell et al.</td>
<td>2002</td>
<td>33</td>
<td>39±9</td>
<td>Study only in RVOT VT</td>
<td>100% RVOT</td>
<td>54±17</td>
<td>97%</td>
<td>6%</td>
<td>NA</td>
</tr>
<tr>
<td>Vestal et al.</td>
<td>2003</td>
<td>91</td>
<td>49±16</td>
<td>Study only in RVOT VT</td>
<td>100% RVOT</td>
<td>56±31</td>
<td>85%</td>
<td>23%</td>
<td>NA</td>
</tr>
<tr>
<td>Ito et al.</td>
<td>2003</td>
<td>168</td>
<td>53±15</td>
<td>Study only in outflow tract VT</td>
<td>69% RVOT, 31% LVOT</td>
<td>21±10</td>
<td>100%</td>
<td>0%</td>
<td>None</td>
</tr>
<tr>
<td>Sekiguchi et al.</td>
<td>2005</td>
<td>148</td>
<td>56±15</td>
<td>100% of idiopathic VT</td>
<td>72% RVOT, 19% PA, 9% LVOT</td>
<td>29±18</td>
<td>86%</td>
<td>0%</td>
<td>None</td>
</tr>
<tr>
<td>Joshi et al.</td>
<td>2005</td>
<td>72</td>
<td>NA</td>
<td>Study only in outflow tract VT</td>
<td>100% RVOT</td>
<td>51</td>
<td>99%</td>
<td>3%</td>
<td>NA</td>
</tr>
<tr>
<td>Iwai et al.</td>
<td>2006</td>
<td>122</td>
<td>51±15</td>
<td>Study only in outflow tract VT</td>
<td>82% RVOT, 22% LVOT</td>
<td>NA</td>
<td>90% in RVOT, 76% in LVOT</td>
<td>NA</td>
<td>None</td>
</tr>
<tr>
<td>Krittayaphong et al.</td>
<td>2006</td>
<td>144</td>
<td>42±11</td>
<td>Study only in RVOT VT pt.</td>
<td>100% RVOT</td>
<td>72±28</td>
<td>92%</td>
<td>12%</td>
<td>None</td>
</tr>
<tr>
<td>Bala et al.</td>
<td>2010</td>
<td>37</td>
<td>53±16</td>
<td>16% of outflow tract VT</td>
<td>51% R–L com, 19% LCC, 11% RCC, 19% LVOT*</td>
<td>13±10</td>
<td>100%</td>
<td>0% in R–L com, 6% in the others</td>
<td>3%</td>
</tr>
<tr>
<td>Fascicular VTb</td>
<td></td>
<td></td>
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<tr>
<td>Nakagawa et al.</td>
<td>1993</td>
<td>8</td>
<td>26±10</td>
<td>Study only in fascicular VT</td>
<td>110% LPF</td>
<td>11 (1–67)</td>
<td>100%</td>
<td>12%</td>
<td>1 Mitral regurgitation from chordae rupture</td>
</tr>
<tr>
<td>Ciggins et al.</td>
<td>1994</td>
<td>8</td>
<td>24±7</td>
<td>29% of idiopathic VT</td>
<td>88% LPF, 95% LPF</td>
<td>10±7</td>
<td>100%</td>
<td>25%</td>
<td>None</td>
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<tr>
<td>Wen et al.</td>
<td>1994</td>
<td>20</td>
<td>28±8</td>
<td>Study only in fascicular VT</td>
<td>5% LAF</td>
<td>7±8</td>
<td>85%</td>
<td>0%</td>
<td>None</td>
</tr>
<tr>
<td>Lin et al.</td>
<td>2005</td>
<td>6</td>
<td>17–46</td>
<td>12% of idiopathic VT</td>
<td>100% LPF</td>
<td>16±8</td>
<td>100%</td>
<td>0%</td>
<td>None</td>
</tr>
<tr>
<td>Intra-cavitary VT</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Doppalapudi et al.</td>
<td>2008</td>
<td>7</td>
<td>47±23</td>
<td>2.4% of idiopathic VT</td>
<td>100% PPM, 42% LPF, 20% MA, 17% PPM, 11% LAF</td>
<td>8.9±5.3</td>
<td>100%</td>
<td>0%</td>
<td>None</td>
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<tr>
<td>Yamada et al.</td>
<td>2010</td>
<td>71</td>
<td>53±15</td>
<td>44.6% of idiopathic LV VT</td>
<td>9% APM, 42% LPF, 20% MA, 17% PPM, 11% LAF</td>
<td>21±9</td>
<td>89% in PPM, 71% APM</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Santoro et al.</td>
<td>2015</td>
<td>8</td>
<td>42±13</td>
<td>5.2% of idiopathic VT</td>
<td>100% RPM</td>
<td>8±4</td>
<td>100%</td>
<td>NA</td>
<td>None</td>
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<tr>
<td>Sadek et al.</td>
<td>2015</td>
<td>10</td>
<td>45±14</td>
<td>2.5% of idiopathic VT</td>
<td>100% Moderator band</td>
<td>22±12</td>
<td>100%</td>
<td>60%</td>
<td>None</td>
</tr>
<tr>
<td>Perivalvular VT</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Tada et al.</td>
<td>2005</td>
<td>19</td>
<td>61±12</td>
<td>5% of idiopathic VT</td>
<td>100% MA</td>
<td>21±15</td>
<td>100%</td>
<td>0%</td>
<td>None</td>
</tr>
<tr>
<td>Tada et al.</td>
<td>2007</td>
<td>38</td>
<td>61±14</td>
<td>8% of idiopathic VT</td>
<td>100% TA</td>
<td>31±7</td>
<td>66%</td>
<td>0%</td>
<td>None</td>
</tr>
</tbody>
</table>

Continued
<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Age (years)</th>
<th>Prevalence (%)</th>
<th>PVC locations</th>
<th>Follow-up (months)</th>
<th>Acute success</th>
<th>Recurrence</th>
<th>Major complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Van Herendael et al.</td>
<td>2011</td>
<td>24 ± 16</td>
<td>10.4%</td>
<td>Basal RV VT</td>
<td>28%</td>
<td>100%</td>
<td>19%</td>
<td>None</td>
</tr>
<tr>
<td>Kawamura et al.</td>
<td>2012</td>
<td>12 ± 15</td>
<td>1.8%</td>
<td>Basal Crux</td>
<td>40%</td>
<td>50%</td>
<td>11%</td>
<td>1.0 mV</td>
</tr>
<tr>
<td>Doppalapudi et al.</td>
<td>2009</td>
<td>4 ± 12</td>
<td>1.8%</td>
<td>Basal Crux</td>
<td>30.7 ± 15</td>
<td>100%</td>
<td>0%</td>
<td>None</td>
</tr>
<tr>
<td>Mountantonakis et al.</td>
<td>2015</td>
<td>47 ± 15</td>
<td>9.2%</td>
<td>Basal Crux</td>
<td>40%</td>
<td>70%</td>
<td>70%</td>
<td>None</td>
</tr>
</tbody>
</table>

VT, ventricular tachycardia; RVOT, right ventricular outflow tract; LVOT, left ventricular outflow tract; RV, right ventricle; GCV, greater cardiac vein; AIV, anterior interventricular vein; LCX, left circumflex coronary artery; RCC, right coronary cusp; LCC, left coronary cusp; R–L com, right–left coronary cusp commissure; LPF, left posterior fascicle; LAF, left anterior fascicle; PPM, posterior papillary muscle; APM, anterior papillary muscle; MA, mitral annulus; RPM, right papillary muscle; TA, tricuspid annulus; RV, right ventricle; GCV, greater cardiac vein; AIV, anterior interventricular vein; LCX, left circumflex coronary artery.

a Include only LV endocardium outflow tract, not aortic cusps.

‘Substrate mapping’ for reentrant VT allows the mapping procedure to be performed in sinus rhythm and often is used in combination with activation mapping and entrainment mapping to minimize the time spent in VT. The mapping provides delineation of the scar based on abnormal local electrogram amplitude and abnormal local electrogram configuration. For abnormal endocardial electrogram amplitudes, the dense scar is defined by a voltage amplitude < 0.5 mV endocardially and normal is defined by an amplitude > 1.5 mV in bipolar recording. Somewhat different parameters are used during epicardial mapping, with the low-voltage area defined as a bipolar signal amplitude < 1.0 mV. Because epicardial fat may decrease signal amplitude, low-voltage areas during epicardial mapping should also show abnormal electrogram configuration. Measurement of unipolar voltage is also helpful in substrate mapping. Epicardial unipolar voltage mapping can be used to predict the epicardial arrhythmia substrate. The unipolar voltage cut off in normal tissue has been reported to be ≥ 8.27 mV in the left ventricle of patients with a dilated cardiomyopathy and ≥ 5.5 mV in the right ventricle of patients with ARVC. For abnormal local electrogram configuration, several characteristics have been described including fractionated electrograms, isolated delayed components, multipotential electrograms, and sharp high-frequency ventricular potentials that are sometimes only identified by pacing. These abnormal configurations are considered indicative of local electrical activity arising from pathological tissue. However, voltage mapping may be challenging to identify septal or mid-myocardial substrates. Regions of late gadolinium enhancement on cardiac MRI have been successfully integrated within electroanatomic mapping systems to define the presence of, subendocardial, transmural, mid-myocardial or epicardial scar, and to identify septal substrates for ablation in both NICM and ischaemic cardiomyopathy (ICM). Prior studies have documented a strong association between low-voltage regions and VT reentry circuit isthmus sites with core scar sites as identified by late gadolinium enhancement on MRI. A major limitation of MRI as a tool for VT ablation is the presence of an ICD in many patients undergoing this procedure. Fortunately, techniques have been developed to safely image patients with ICD systems, and a wideband late gadolinium enhancement technique has been developed to eliminate hyper-intense artefacts from the ICD generator.

‘Pace mapping,’ which can be used for identification of the arrhythmia source in triggered VT and for identification of the VT exit site in reentry, is another methodology that enables mapping during sinus rhythm. An identical paced 12-lead vs. VT 12-lead ECG morphology match suggests proximity to the exit or isthmus of the VT reentry circuit, or the source of triggered foci. During pace mapping of reentrant VT, a long stimulus-to-QRS interval (≥ 40 ms) during pacing indicates regions of slow conduction. A pacing threshold ≥ 10 mA has been used to define unexcitable scar, provided electrode-tissue contact is adequate.

‘Entrainment mapping’ during reentrant VT is performed during VT to identify the functional involvement of each pacing site within the reentry circuit. Post-pacing interval (PPI), QRS morphology during entrainment and stimulus to QRS interval are the three major characteristics used during entrainment mapping to characterize the functional involvement of each pacing site within the reentry circuit as demonstrated in Figure 3. The PPI, measured from the last stimulation pulse that captures myocardium, and entrains or resets...
the tachycardia to the next depolarization at the pacing site, represents the conduction time from pacing site to the reentry circuit, through the circuit and back to the pacing site. Therefore, the difference between PPI and tachycardia cycle length (PPI–TCL) indicates the conduction time from the pacing site to the circuit. The presence of 12-lead QRS morphology match during entrainment is indicative that the pacing site is in the inner loop of the circuit. A suitable site for ablation during reentrant VT is indicated by critical isthmus characteristics: PPI–TCL ≤ 30 ms, stimulus to QRS interval < 70% of TCL, and concealed entrainment. 

‘Activation mapping’ can be performed during triggered and reentrant VT. The focus of activation mapping during reentrant is to identify sites with bipolar electrogram evidence of continuous diastolic activity, isolated diastolic potentials, or both. Unlike idiopathic VT, there is no earliest or latest point of activation in reentry. It is important to note that mid diastolic sites can be part of abnormal slow conduction unrelated to the VT circuit. To localize the critical isthmus, it is important to confirm that electrograms of interest cannot be dissociated from VT and participate as an active component of the critical circuit. Therefore, activation mapping in isolation is not a reliable method for reentrant VT in SHD but can be a useful adjunct in conjunction with other mapping modalities.

### Ablation strategies

Current ablation strategies (Figures 4 and 5) are guided by identification of potential reentry circuit isthmuses and exit sites based on substrate, pace, and entrainment mapping. A schematic drawing showing several commonly employed ablation strategies is presented in Figure 5. These techniques can be used together, especially for complex substrates with multiple exit sites. In the setting of stable sustained VT, targeted isthmus ablation (Figure 5A) at sites with the three entrainment criteria (PPI–TCL ≤ 30 ms, stimulus to QRS interval < 70% of TCL, and concealed entrainment) has the highest success rate with positive predictive value of 100% and negative predictive value of 96% for prediction of VT termination with RF application at that site. However, almost 80% of induced VT episodes in SHD are either non-sustained and if sustained are haemodynamically intolerable, thus precluding proper entrainment mapping. As a result, pace and substrate-mapping strategies are the main stay approach for VT ablation in most patients with SHD.

Substrate ablation is developed from the concept of surgical ablation by removal of arrhythmogenic tissue. In the setting of unmappable VT, various technique have been proposed. Scar homogenization (Figure 5B) by RF ablation of all sites with abnormal electrograms has been shown to increase freedom from VT in SHD patients. Creation of a line of block by creating a linear RF lesion is also widely used. Ablation lines can be applied parallel to the scar border within the low-voltage area encompassing the exit region (Figure 5C), placed perpendicular to all defined isthmuses between islands of unexcitable tissue (Figure 5D), or extend perpendicular to the scar border from the area of dense scar, across the border zone and connecting out to an anatomical barrier (Figure 5D). Soejima et al. suggested less extensive ablation that resulting in lower inducibility of VT by applying of short ablation lines at the reentry isthmus and additional lesions extending parallel to the border zone of infarction until pacing failed to capture or
mitral annulus was reach (Figure 5A). These different substrate ablation strategies have not been directly compared in clinical trials. VISTA trial (ablation of clinical ventricular tachycardia vs. additional of substrate ablation on the long-term success rate of VT ablation) is a recent open-label, randomized, parallel-group, and multicenter study comparing isthmus ablation alone with additional substrate ablation approach in patients with ICM presenting with stable VT. The investigator found that the 12-month freedom of recurrent VT in substrate ablation group was higher than isthmus ablation group (84.5 vs. 51.7%, P < 0.001) without differences in mortality rate and complication rate.

Local abnormal ventricular activity (LAVA) is a high-frequency abnormal ventricular signal representing near-field signals of slowly conducting tissue and hence potential VT isthmuses. Local abnormal ventricular activity can be found in 90% of patients with NICM, ICM, and ARVC. It is sometimes hidden in the far-filed signal and can be unmasked by pacing from RV, at potential of interest, or during local ectopics. This LAVA ablation can be applied both endo- and epicardially. The VT ablation endpoint as LAVA elimination, additional to non-inducibility, has showed to be associated with reduction in recurrent VT or death during long-term follow-up and become the better predictor of success in substrate ablation.

Nonetheless, substrate and pace mapping is limited in patients with advanced heart failure, unstable VT with acute decompensated heart failure or VT storm whom the extensive ablation may result in worsen cardiac dysfunction. External haemodynamic support devices, including intra-aortic balloon and percutaneous left ventricular assist devices, have been successfully used to provide haemodynamic support during mapping of induced VT episodes and allow activation and entrainment mapping to be performed in these patients. However, there was no improved short-term success or greater long-term freedom from VT when haemodynamic support is used for delineation of the critical isthmus and targeting of VTs. Therefore, the use of these devices has been primarily restricted to patients who failed substrate-based ablation or have recurrence of rapid VTs and may be considerable in patients with very poor LV function.

The best end point for the ablation has not been well defined. However, complete VT non-inducibility was found to be associated with reduction of the likelihood for all-cause mortality in patients with NICM and be associated with longer longevity and lower

Figure 3 A schematic drawing of the components of the ventricular tachycardia circuit. The main components include entrance, central isthmus, and exit sites. Characteristics of entrainment mapping according to the site of pacing are summarized in the algorithm (PPI, post-pacing interval; TCL, tachycardia cycle length; S-QRS, stimulus to QRS interval).
VT/VF recurrence in post-infarction VT. Therefore, a non-inducibility of any VT is a reasonable end point for VT ablation.90,91

**Outcome and safety**

Table 5 summarizes the reported outcomes of catheter ablation of VT in patients with SHD. The outcomes are categorized based on the ventricular substrates; those associated with ICM and those with NICM. In the setting of ICM, the utility of catheter ablation has been examined both as primary as well as secondary treatment after failed medication therapy. The SMASH-VT and VTACH trials were randomized controlled trials of catheter ablation as primary treatment for VT. Both studies showed that catheter ablation significantly reduced arrhythmia recurrence but had no impact in mortality, when an ICD has been implanted. The rate of complications was ~5% and no peri-procedural mortality was observed.93,94 In contrast, when VT ablation is performed in patients with previous myocardial infarction selected after failure of prior anti-arrhythmic therapy, acute success is 49–89% and long-term recurrences occur in 34–57% of patients. Induction of multiple morphologies of VT and unstable VT at baseline appear to be associated with a higher risk of VT recurrence.84,100 The procedure-related mortality reaches 3% and the complication is ~5–10%, slightly higher than VT ablation as primary treatment. These results may be biased by selection bias related to enrolment.

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**Figure 4** The algorithm shows mapping strategies based on haemodynamic stability during tachycardia, stability of VT reentry circuit (stable morphology and cycle length), and inducibility during procedure (VT = VT).

**Figure 5** Different ablation strategies and abnormal electrograms are shown. These strategies can be used together. (A) Isthmus ablation. (B) Scar homogenization. (C) Ablation lines applied parallel to the scar border within the low-voltage area encompassing the exit region. (D) Ablation lines placed perpendicular to all defined isthmuses between islands of unexcitable segment or extend perpendicular to the scar border from the area of dense scar, across the border zone and connecting out to normal myocardium or anatomical barrier (e.g. mitral valve).
Table 5  Outcomes of ventricular tachycardia ablation in patients with structural heart disease

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Design</th>
<th>N</th>
<th>Age (years)</th>
<th>ICM/ NICM</th>
<th>LVEF VT episodes preablation</th>
<th>Failed amiodarone</th>
<th>Ablation</th>
<th>FU period (months)</th>
<th>Acute success</th>
<th>Recurrence</th>
<th>Long-term mortality</th>
<th>Complications</th>
</tr>
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<tr>
<td></td>
<td></td>
<td><strong>VT ablation in ICM</strong></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Primary treatment (without failed medication therapy)</td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>Reddy et al. (SMASH-VT)</td>
<td>2008</td>
<td>Prospective, multicenter, randomized, open</td>
<td>128 (64 in ablation gr)</td>
<td>67</td>
<td>100/0</td>
<td>31 ± 10°</td>
<td>None</td>
<td>Substrate ablation</td>
<td>23 ± 6</td>
<td>NA</td>
<td>12%</td>
<td>9%</td>
<td>5%</td>
</tr>
<tr>
<td>Kuck et al. (VTACH)</td>
<td>2010</td>
<td>Prospective, multicenter, randomized, open</td>
<td>107 (52 in ablation gr)</td>
<td>66 ± 8</td>
<td>100/0</td>
<td>34 ± 10</td>
<td>None</td>
<td>Mapping and Substrate ablation</td>
<td>23 ± 9</td>
<td>60%</td>
<td>41%</td>
<td>10%</td>
<td>4%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>After failed medication therapy[^a]</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Calkins et al. (cooled RF multicenter)</td>
<td>2000</td>
<td>Prospective, multicenter, non-randomized</td>
<td>146</td>
<td>65 ± 13</td>
<td>82/18</td>
<td>31 ± 13</td>
<td>25 ± 31</td>
<td>40%</td>
<td>Mapping ablation</td>
<td>8</td>
<td>75%</td>
<td>46%</td>
<td>25%[^c] 8%</td>
</tr>
<tr>
<td>Stevenson et al. (thermocool VT ablation trial)</td>
<td>2008</td>
<td>Prospective, multicenter, non-randomized</td>
<td>231</td>
<td>68 (59–72)</td>
<td>90/10</td>
<td>25 (20–35)</td>
<td>11 (5–32)</td>
<td>70%</td>
<td>Mapping ablation</td>
<td>6</td>
<td>49%</td>
<td>47%</td>
<td>18%[^c] 7%</td>
</tr>
<tr>
<td>Carbacicchio et al.</td>
<td>2008</td>
<td>Prospective, single-centre, non-randomized</td>
<td>95</td>
<td>64 ± 13</td>
<td>75/25</td>
<td>36 ± 11</td>
<td>NA</td>
<td>94%</td>
<td>Mapping and Substrate ablation</td>
<td>22 ± 13</td>
<td>89%</td>
<td>34%</td>
<td>16%</td>
</tr>
<tr>
<td>Tanner et al. (Euro-VT)</td>
<td>2009</td>
<td>Prospective, multicenter, non-randomized</td>
<td>63</td>
<td>64 ± 9</td>
<td>100/0</td>
<td>30 ± 13</td>
<td>55 ± 19</td>
<td>49%</td>
<td>Mapping and Substrate ablation</td>
<td>12 ± 3</td>
<td>81%</td>
<td>37%</td>
<td>9%</td>
</tr>
<tr>
<td>Dinov et al. (HELP-VT)</td>
<td>2014</td>
<td>Prospective, single-centre, non-randomized</td>
<td>227</td>
<td>NICM 59 ± 13</td>
<td>72/28</td>
<td>33 ± 11</td>
<td>NA</td>
<td>37%</td>
<td>Mapping ablation</td>
<td>NICM 20</td>
<td>NICM 67%</td>
<td>NICM 77%</td>
<td>NICM 13% 11%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>VT ablation in NICM</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Hsia et al.</td>
<td>2003</td>
<td>Observational, single-centre</td>
<td>19</td>
<td>61 ± 16</td>
<td>0/100</td>
<td>34 ± 11</td>
<td>NA</td>
<td>63%</td>
<td>Mapping and Substrate ablation</td>
<td>22 ± 12</td>
<td>73%</td>
<td>52%</td>
<td>20%</td>
</tr>
<tr>
<td>Soejima et al.</td>
<td>2004</td>
<td>Observational, single-centre</td>
<td>28</td>
<td>54 ± 14</td>
<td>0/100</td>
<td>30 ± 11</td>
<td>NA</td>
<td>43%</td>
<td>Mapping and Substrate ablation</td>
<td>11 ± 9</td>
<td>61%</td>
<td>39%</td>
<td>4%</td>
</tr>
<tr>
<td>Sacher et al.</td>
<td>2008</td>
<td>Retrospective, multicenter</td>
<td>149</td>
<td>52 ± 15</td>
<td>0/100</td>
<td>39 ± 16</td>
<td>NA</td>
<td>59%</td>
<td>Mapping and Substrate ablation</td>
<td>40 ± 29</td>
<td>51%</td>
<td>39% (1 month)</td>
<td>17%</td>
</tr>
<tr>
<td>Tokuda et al.</td>
<td>2012</td>
<td>Retrospective, single-centre</td>
<td>226</td>
<td>52 ± 14</td>
<td>0/100</td>
<td>38 ± 17</td>
<td>NA</td>
<td>65%</td>
<td>Mapping and Substrate ablation</td>
<td>53 ± 40</td>
<td>55%</td>
<td>23%[^c] 22%</td>
<td>5%</td>
</tr>
<tr>
<td>Dinov et al.</td>
<td>2015</td>
<td>Observational, single-centre</td>
<td>102</td>
<td>58 ± 15</td>
<td>0/100</td>
<td>33 ± 12</td>
<td>NA</td>
<td>NA</td>
<td>Mapping and Substrate ablation</td>
<td>24</td>
<td>61%</td>
<td>53%</td>
<td>21%</td>
</tr>
</tbody>
</table>

\[^a\] Ablation group only.
\[^b\] Cool RF multicenter, thermocool VT, Carbacicchio et al., and HELP-VT studies included patients with NICM as a minor portion as noted in column 5.
\[^c\] At 1 year.
\[^d\] In patients presenting with VT storm.
of patients with prior failed anti-arrhythmic therapy and referral to a single tertiary care centre.

In NICM, there are no prospective, randomized studies examining the role of catheter ablation. The majority of studies are single-centre reports. The success rate varies from 51 to 73% and the recurrence rate ranges from 23 to 77%. Overall, when compared with VT ablation in patients with ICM, the success rate appears to be lower with a higher recurrence rate and comparable complication rates. The HELP-VT study was a single-centre study that compared ablation in NICM and ICM. This trial reported that catheter ablation of VT in patients with an NICM was associated with a lower success rate and higher recurrence rate when compared with patients with prior infarction. The additional epicardial mapping and ablation in this population has been found to increase the number of acute success and lower recurrent rate. Yokokawa et al. demonstrated that patients with recurrent VT have larger scar and that most recurrent VT circuits were new, representing a changing substrate, but in close proximity to prior ablation lesions (6 ± 3 mm).

For the patients who presented with VT storm, VT ablation can be a life-saving procedure and has been evolving as the standard of care. A meta-analysis including 471 patients from 39 publications showed an acute success rate of 72%, recurrence rate of 6% and procedure-related mortality rate of 0.6%. After 61 ± 37 weeks follow-up, 17% of patients died with majority of those occurred during the first year after the procedures.

Unlike idiopathic VT, the acute success of catheter ablation in VT with SHD is limited. Post-ablation attempts to reinduce VT are often not performed due to haemodynamic instability. When the clinical VT can be induced during the procedure, the acute failure of catheter ablations often reflects the presence of an epicardial origin, intramural focus, or the proximity of lesions to His/coronary artery/phrenic nerve. Therefore, advanced approaches including epicardial ablation, including transcoryonary ethanol ablation, and surgical ablations serve as alternative ablation strategies. Surgical ablation/hybrid procedure is however rarely performed and reserved for some specific cases, e.g., cases with thrombus overlaying the substrate, cases with mechanical aortic and mitral valves that preclude access or cases with failed epic- and endocardial ablation. Overall, complications rate of VT ablation in SHD is 8%. Similar to idiopathic VT, the most frequent complication is access related. Other more serious complications include cardiac tamponade and thromboembolic events. The independent predictors of complications include age, renal insufficiency, depressed LVEF, and operator experiences.

Despite reduction of VT recurrence after catheter ablation, the overall long-term mortality of VT patients with SHD remains high, varying from 4 to 22% (Table 5). A meta-analysis of trials comparing catheter ablation for VT in patients with SHD to controls confirms the findings that ablation reduces VT recurrence without significant impact upon mortality when an ICD is present. However, 1-year freedom from VT recurrence is associated with improved transplant-free survival, independent of heart failure severity. The substantial mortality is consistent with the severity of heart disease and association of spontaneous VT with mortality and heart failure. Worsening heart failure resulting from catheter ablation can be a concern. However, a single high-volume centre study has revealed no echocardiographic evidence of deterioration in left ventricular function when ablation is performed by an experienced operator.

### Indications

Acceptable indications for catheter ablation of VT in patients with SHD continue to evolve. The indications proposed in the 2009 HRS/EHRA Consensus document on catheter ablation are summarized in Table 4. In contrast to the most recent guidelines, when catheter ablation was considered a last resort option after failure of multiple anti-arrhythmic agents, current guidelines indicate that under specific circumstances VT ablation can be considered as an acceptable alternative to amiodarone or even prior to the use of anti-arrhythmic medications (Table 4).

### Predictions for the future

In contrast to catheter ablation in the setting of idiopathic VT, catheter ablation in patients with SHD represents the ‘frontier’ of catheter ablation. Although the techniques and outcomes for catheter ablation of VT have improved over the past decade, they are currently suboptimal. The long-term efficacy of the procedure is relatively low and the complication rates are high.

Fortunately, there is now an increased intensity of focus on the field of catheter ablation of VT in patients with SHD. Greater effort is being placed on developing prospective trials to better define the true safety and efficacy of the procedure in a variety of settings. Because of the recent observation that the outcomes of VT ablation are improved in patients with less severe disease, studies are being designed which enrol patients earlier in their VT course. Table 6 shows a list of ongoing trials of VT ablation in patients with SHD. While VT ablation vs. enhanced drug therapy (VANISH), medical anti-arrhythmic treatment or radiofrequency ablation in ischaemic ventricular tachyarrhythmias (MANTRA-VT), and anti-arrhythmic therapy vs. catheter ablation as first-line treatment for AICD shock prevention (AVATAR) are comparing catheter ablation and anti-arrhythmic medication early in the course of the disease after several VT episodes, substrate targeted ablation using the flexibility ablation catheter system for the reduction of VT (STAR-VT), and substrate modification study in patients getting an implantable cardioverter defibrillator (SMS-ICD) are investigating the role of catheter ablation at the time of ICD implantation, after only one episode of ventricular arrhythmia and/or non-invasive programmed stimulation, which induces monomorphic VT. The BERLIN (home monitoring based early ICD intervention study) and PARTITA (does timing of VT ablation affect prognosis in patients with an ICD?) studies will also investigate the appropriate timing for catheter ablation. One compares VT ablation at the time of implant and after the third shock and another compares VT ablation after first ICD shock and after VT storm occurs. The Indian trial of endocardial ventricular substrate ablation to prevent recurrent VT events (INTERVENE) trial is a unique trial conducted in India where there is a limitation of ICD implant. The trial compares the results of medications treatment with and without adjunctive VT ablation in a situation where resources preclude ICD implantation.

Research is ongoing to develop ablation tools that achieve more effective and deeper ablation lesions to address intra-myocardial
substrates. Bipolar RF ablation, alcohol ablation, irrigated needle ablation, high-frequency focused ultrasound, and electroporation are under investigation. Bipolar RF ablation, alcohol ablation, irrigated needle ablation, high-frequency focused ultrasound, and electroporation are under investigation. Bipolar RF ablation, alcohol ablation, irrigated needle ablation, high-frequency focused ultrasound, and electroporation are under investigation. Bipolar RF ablation, alcohol ablation, irrigated needle ablation, high-frequency focused ultrasound, and electroporation are under investigation. Bipolar RF ablation, alcohol ablation, irrigated needle ablation, high-frequency focused ultrasound, and electroporation are under investigation.

Research is also underway to allow a more precise understanding of the arrhythmogenic substrate. Cardiac magnetic resonance may be of value in identifying the arrhythmogenic substrate and allow more precise catheter ablation. The ancillary study of intervention mechanisms for ventricular tachycardia, an ancillary STAR-VT study, will examine the CT substrate for VT and the impact of ablation upon that substrate. Moreover, an imaging study combined with 252-lead ECG has been used to developed non-invasive mapping, guiding catheter ablation in both idiopathic VT and defining substrate in ischaemic VT.

Another area of active research is evaluating sympathetic modulation as a therapy for patients with refractory VT. Bilateral cardiac sympathetic denervation was found to have benefit in continued freedom from ICD shocks and a significant reduction in ICD shocks, extending beyond the acute post-sympathectomy period (mean follow-up of 367 ± 251 days post-procedure). The PREVENT-VT (cardiac denervation surgery for prevention of ventricular tachyarrhythmias) trial will determine the role of this treatment in refractory VT. Additionally, Remo et al. reported a case series of renal artery denervation for treatment of refractory VT in the NICM and ICM patients with maximized anti-arrhythmic drug and prior ablation. The case series showed promising results in terms of safety and effectiveness as an adjunctive therapy. Additionally, the ongoing renal sympathetic denervation to suppress ventricular tachyarrhythmias trial will provide more insights regarding benefits from renal denervation.

### Conclusion

In the more than three decades since first described, VT catheter ablation as progressed as a ‘tale of two cities’. The techniques and outcomes of catheter ablation of idiopathic VT has rapidly matured, with compelling demonstration of a safe, effective, and curative procedure with the exception of some rare idiopathic VT locations, e.g.,
anterior PAP, moderator band, and epicardial VT that may carry relatively higher recurrence rate. In contrast, VT catheter ablation in patients with SHD has followed a very different trajectory, and remains in its infancy. Although progress has been made by the improved tools for mapping and ablation, the outcomes of this procedure remain poor with <50% success rates and excess risk of major complications including stroke and death. Because of this, the adoption of this procedure has been slow. Fortunately, a new intensity and focus has been placed on catheter ablation of VT in patients with SHD. New technologies for mapping and ablation are in development and multicenter clinical trials are underway. We are hopeful that these efforts with bear fruit and that the long-term efficacy of this procedure will soon exceed 70% with a reduction in major complication rates to an acceptable level.

Conflict of interest: S.N.: Consultant: Biosense Webster, Medtronic and CardioSolv; Research Grant: Biosense Webster and St. Jude Medical. H.C.: Consultant: Medtronic and St. Jude Medical; Research Grant: Boston Scientific and St. Jude Medical.

References


Catheter ablation in VT


