Ablation of Idiopathic Ventricular Tachycardia

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Idiopathic ventricular tachycardia: overview

Ventricular arrhythmias are very common and can be an important prognostic indicator in patients with advanced structural heart disease. However, premature ventricular complex (PVCs), and non-sustained and sustained ventricular tachycardia (VT) may occur in the absence of structural heart disease. In this situation, VT is termed idiopathic and this designation has important prognostic and management implications. With the exception of PVC-related myopathy and the so-called “malignant” type of idiopathic VT, which will be discussed in detail below, treatment of idiopathic VT is based on symptom control, because there are no prognostic concerns. For the purposes of this review, PVCs and non-sustained and sustained VT will be considered together under the heading of idiopathic VT.

Patients with idiopathic VT are often young and healthy. In fact, there appears to be an increased incidence of idiopathic VT in endurance athletes (cyclists, rowers, runners); the cause of this is unclear, but may due to changes in autonomic tone related to training. Alternatively, idiopathic VT can occur in the setting of structural heart disease; in this case, the VT site of origin is distant from the area of damaged myocardium.¹ This is the first refinement in our definition of idiopathic VT; it is easy to recognize by the absence of heart disease, but must be identified in the presence of heart disease by its behavior and electrocardiographic signature, both of which will be discussed below.

From a pathophysiologic standpoint, the mechanism of outflow tract-related idiopathic VT is cAMP-dependent delayed after depolarizations;² the mechanism of idiopathic VT from other sites has not been well established. Understanding of this mechanism makes sense of the typical inciting events for idiopathic VT: caffeine, emotional stress, exercise—all mediated through increases in intracellular calcium. Less apparent is the underlying cause of hormone-related triggers for idiopathic VT. Not unusually, women will have increased symptoms during menses or with pregnancy; often PVCs become more frequent with the onset of menopause, only to disappear when this process is completed. The mechanism of non-outflow tract idiopathic VT is not well determined, but is generally considered to be non-re-entrant because of similar clinical behavior. One important exception is so-called left ventricular (LV) idiopathic VT, or Belhassen’s VT, which is macro-re-entrant and uses tissue adjacent to the posterior or anterior fascicle.³

The non-re-entrant mechanism of idiopathic VT influences its presentation. Although sustained VT is possible, very often the clinical presentation is isolated PVCs or bursts of repetitive monomorphic non-sustained VT, exacerbated by the situations discussed above. Symptomatic patients usually complain of rapid palpitations, but others note the pause that follows premature beats. Often this will affect exercise tolerance, which may represent the main complaint. There is a fair degree of confusion among physicians as to how to interpret this condition. All too often, symptomatic patients are subjected to investigation for structural heart disease and then not treated for the ventricular ectopy; asymptomatic patients are treated with medications because of misplaced concern over the prognostic meaning of frequent PVCs.

Clinical evaluation

Patients with idiopathic VT typically present with palpitations or incidentally detected PVCs on electrocardiogram (ECG) screening for other conditions. The first objective is to exclude structural heart disease, usually with transthoracic echocardiography. It is
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important to consider the diagnosis of arrhythmogenic right ventricular (RV) cardiomyopathy in patients who present with left bundle VT morphologies. Echocardiography is sufficient in evaluating RV size and function if performed with specific attention to this task; magnetic resonance imaging is very sensitive, but has problems with specificity. Catheter-based RV voltage mapping is the gold standard, but is obviously invasive.5 Mild or early forms of cardiac sarcoid or myocarditis are other important processes to exclude.

The evaluation of patients with PVCs in the setting of cardiomyopathy is more complex. In young patients with new-onset non-ischemic cardiomyopathy and very frequent (>20% of total beats) PVCs, PVC-related cardiomyopathy is an important consideration, as treatment of the PVCs can lead to restoration of LV function.5,6 This can be even more difficult in patients with PVCs in the setting of established cardiomyopathies, the question then being whether or not frequent PVCs are contributing to further deterioration of LV function. It is helpful to keep a high index of suspicion for this possibility, as the potential yield of treatment in well-selected patients has been substantial.7 Features that may predict a favorable response to ablation therapy include frequent PVCs, dominant or single PVC morphology, and ECG signatures compatible with recognized sites consistent with idiopathic VT.

The indications for treatment of idiopathic VT are as follows: 1) management of symptoms, 2) relief of PVC related cardiomyopathy, and 3) prevention of PVC triggers for PMVT, which may occur in normal hearts or patients with structural or electrical heart diseases.8-10 Pharmacologic therapy usually consists of β-blockers or calcium channel antagonists as first-line therapy, and small series report symptomatic control in 60-70% of patients.11,12 Class I and III antiarrhythmic drugs may also be used. For LV idiopathic VT, intravenous verapamil terminates acute episodes, and oral administration is fairly effective in preventing recurrence. My general viewpoint is that pharmacologic therapy is occasionally helpful, but is limited by side-effects particularly in young patients. Most patients with significant symptoms are probably best treated with ablation therapy.

Electrocardiography

Idiopathic VT is fairly easy to recognize on the 12-lead ECG. Because the VT arises from normal tissue, voltages are large and the QRS upstroke is brisk (except in the case of epicardial sites of origin). Idiopathic VT can arise from anywhere in the heart (Table 1), and analysis of the ECG serves as the initial localization tool. The implications for discussing risk (left versus right sided and risk of stroke, inadvertent coronary artery damage) as well as pre-procedural technical planning should be obvious.

The majority of idiopathic VTs arise from the right ventricular outflow tract (RVOT), specifically from the posterior aspect just beneath the pulmonic valve.13 This location is consistent with the ECG signature (Figure 1), which is a left bundle morphology with tall R waves in the inferior leads. In addition, because the RVOT is anterior and because the precordial leads register posterior to anterior forces, the transition (from negative to positive QRS complexes) in the precordial leads is late, typically in lead V3–V4. Another frequent site of origin is the right and left sinuses of Valsalva (Figure 2).12,14 It superficially resembles RVOT VT with left bundle morphology and positive R waves in the inferior leads; however, because the LV outflow tract is posterior (rather than leftward) to the RV outflow tract, the distinguishing feature is more positive forces in the precordial leads, an earlier precordial transition, or the presence of a broad initial R wave in V1 or V2. Continuing this series, proceeding more posterior to LV sites of origin below the valve, these will have progressively more precordial forces; at the level of the mitral annulus, VT QRS complexes are typically right bundle with positive R waves across the precordium. LV idiopathic VT also has a consistent appearance on the ECG (Figure 3), a right bundle morphology with negative forces in the inferior leads which makes sense given it association to the left posterior fascicle. The complexes do not exactly duplicate right bundle branch block/left anterior hemiblock (Figure 3), because antegrade activation of the ventricle is via the slow fiber which is close to but not within the normal Purkinje system.7 Less commonly, this syndrome can involve the anterior fascicle, in which case the QRS morphology is right bundle with positive forces in the inferior leads.

The ECG can be helpful in predicting epicardial sites of origin; in the setting of idiopathic VT, this primarily refers to sites within the coronary venous system. Although the initial algorithms have developed strategies related to the slowness of the QRS upstroke during VT (because of the lack of rapid access to the endocardial Purkinje network to coordinate activation),15 we have found the morphologic clue of unexpected q waves in focus leads (lead I for the basal anterior wall, the inferior leads for the basal inferior wall) to be more helpful.16,17

Catheter ablation

Success rates for catheter ablation in idiopathic VT range from 80% to 100% in published series,12,14,18,19 but this represents older data and does not take into account recent recognition of sites outside of the RVOT and

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**Table 1: Common sites for idiopathic ventricular tachycardia**

<table>
<thead>
<tr>
<th>Location</th>
<th>Description</th>
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<tbody>
<tr>
<td>RVOT (85%)</td>
<td>RV inflow, RV inferior wall, LV “outflow tract”</td>
</tr>
<tr>
<td>Left and right coronary cusps, coronary venous system, anterior endocardium</td>
<td>LV idiopathic VT (verapamil sensitive)</td>
</tr>
<tr>
<td>Basal LV</td>
<td>Papillary muscles (LV more common than RV)</td>
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LV: left ventricular; RV: right ventricular; RVOT: right ventricular outflow tract.
general improvements in interventional electrophysiology. In general, and as befits a non-life-threatening problem in young patients, success is expected but risk must be minimal. Success and procedural risk vary according to site of origin; many laboratories would be expected to be facile with RVOT ablation, but expertise in coronary cusp, coronary venous, or epicardial ablation is much less widespread.

**RVOT VT**

As discussed above, the vast majority of RVOT VTs arise from the posterior aspect of the RVOT, just under the pulmonary valve. This space is sometimes referred to as the “septal” RVOT, which is anatomically incorrect, as it is adjacent to the ascending aorta. We think of the outflow tract based on a nine-site mapping schema of the right anterior oblique view, but the most important of these are adjacent to the valve: site 1 = posterior (but really rightward), 2 = mid, and 3 = anterior (but really leftward). Sites 1–3 can be recognized by changes in the degree of positivity in lead I (positive at site 1, isoelectric at site 2, negative at site 3). RVOT most frequently arises from site 2–3 or 3. The most important anatomic considerations for successful ablation are 1) determining the position of the pulmonary valve (as most successful sites are just below), and 2) understanding just how leftward site 3 really is, as it is tucked into a small potential space which presumably can be ablated shut by errant first attempts. As shown in Figure 4, although the mapping catheter is in the same plane as the RV apical catheter at site 2, it breaks this plane quite a distance to the left at site 3. Attention should also be directed to the fact that the left main and left anterior descending coronary arteries are close (but usually caudal) to the perivalvular RVOT.20

Mapping can be performed with activation mapping or pace mapping. Although pace mapping has been maligned for mapping in structural heart disease, with low pacing output and attention to detail in normal tissue, it works well and is particularly useful in patients with rare PVCs (often caused by therapeutic sedation). The attention to detail is critical. The statement “12 of 12 pace map match” is not sufficiently precise. Instead, matching of the “nooks and crannies” of interesting leads is much more accurate (Figure 5).21 Ablation is performed with standard radiofrequency energy, typically around 30 Watts to achieve a temperature setting of 55°C. The affect should be nearly instantaneous, although sometimes ectopy or non-sustained VT is produced by energy application, which is a promising

![Figure 1: Twelve-lead electrocardiogram recorded during right ventricular outflow tract tachycardia. The QRS complexes have a left bundle inferiorly directed morphology with a late precordial R-wave transition; the QRS complexes only become positive at lead V4. Also note the brisk upstrokes of the QRS and large voltages, consistent with endocardial origin in healthy myocardium.](image-url)
Figure 2: Twelve-lead electrocardiogram recorded during frequent premature ventricular beats (PVCs) arising from the left coronary cusp. These QRS complexes are also left bundle inferior morphology, but compared with right ventricular outflow tract ventricular tachycardia, have an earlier transition (V3) and a broad R wave in V2. This is an important signal of its more posterior site of origin.

Figure 3: Twelve-lead electrocardiogram recorded during idiopathic left ventricular tachycardia. The QRS morphology is right bundle superior, although the right bundle pattern does not match that seen in right bundle branch block (monophasic R wave). Nonetheless, the QRS complexes are relatively narrow and normal in appearance, which often leads to confusion for SVT with aberrancy.
A healthy waiting period (30 min) is probably critical to prevent recurrence, and rechallenge with isoproterenol is helpful, particularly when this provoked PVCs or VT prior to ablation.

Atypical locations for RVOT VT include free wall sites, sites farther below the pulmonic valve, or supravalvular sites. They follow the same logic for mapping and ablation as described above. As mentioned before, subtle forms of structural heart disease may mimic idiopathic VT. By definition, the site of origin of idiopathic VT has normal endocardial bipolar voltage (>1.5 mV). Patients with ARVC, myocarditis or sarcoid may have RVOT VT sites of origin that may be difficult to differentiate from idiopathic on 12-lead ECG, but have contiguous areas of abnormal bipolar electrograms, including at the site of VT origin.

**Left ventricular outflow tract VT**

Idiopathic left ventricular outflow tract (LVOT) VTs are more difficult to map and ablate than RVOT VTs, because the anatomic space is more complex. Components of the LVOT include the sinuses of Valsalva (right and left, but rarely the non-coronary cusp can be the site of origin for VT), the subvalvular endocardium, the great cardiac vein–anterior intraventricular branch (GCV-AIV) region of the coronary venous system and the adjacent epicardium. The most frequent site among this listing seems to be the sinuses of Valsalva, but more and more we view this anatomic site as the closest approach to myocardium at a distance (within the intraventricular septum), not within the coronary cusp itself. In fact, the idea of ablating from multiple different adjacent anatomic sites (e.g. GCV + left coronary cusp + anterior endocardium) to reach sites of origin that are truly “in between” seems increasingly relevant in this application. Alternatively, sometimes relatively perfect mapping sites may be unsafe for ablation because of proximity to coronary arteries, but this situation can be rescued by ablation from an adjacent site.

Unlike mapping in the RVOT, pace mapping is unreliable during LVOT mapping, particularly as arrhythmias often arise from some distance away from mapping sites and may arise from small relatively dissociated fibers (Figure 6). These are difficult to identify except during VT and activation mapping is essential for successful ablation strategy. Another concept that is important in coronary cusp mapping is assuring that the entire semicircular extent of each cusp is explored. We have found intracardiac echocardiography extremely useful in this application (Figure 7), although other laboratories handle this by merging CT angiograms or magnetic resonance images to a mapping system, or by angiography. In any case, this anatomical principle must be well appreciated. In addition, these strategies also help with ensuring safe distance from the coronary ostium, at least from the standpoint of mapping within
the cusps. Most true coronary cusp VTs are ablated from the depths of the sinuses. Recently, we observed an electrocardiographic signature—notching on the downstroke in lead V1 that predicts an unusual site of origin—directly in between the right and left cusps at the level of the commissure between them.22 This obviously brings another dimension into sinus of Valsalva mapping.

Although not constantly necessary, we have found irrigated radiofrequency (RF) delivery helpful in sinus of Valsalva ablation for several reasons. First, remember that the ablation target is often at a distance from the catheter. Second, irrigated RF delivery is necessary for ablation within the coronary venous system, because the catheter is often wedged within the vein preventing passive cooling from blood flow. Often relatively small amounts of power (20–30 Watts) are effective, and the effect is typically rapid.

If a typical ECG morphology leads one to the right or left coronary cusp, activation mapping starts with covering the entire surface of the cusp. If presystolic sites are not found, or if initial ablation is unsuccessful, other anatomic sites must be investigated. It is probably good practice to place a coronary sinus catheter for all LVOT VT cases, delivered as distally as possible, as a point of comparison for activation mapping. Mapping and ablation in the GCV-AIV can be difficult as these veins narrow to the point that catheters are as

Figure 6: Twelve-lead electrocardiogram and intracardiac recordings during two sinus beats and a PVC which arises from the left coronary cusp. Note (arrows) the distinct late potential recorded during sinus rhythm, which precedes the ventricular tachycardia QRS and was the earliest activation site during mapping. Ablation at this site eliminated the PVC.

Figure 7: Intracardiac echocardiography during coronary cusp mapping. The aortic valve is seen in cross-section and the individual cusps are labeled. A mapping catheter (asterisk) is seen in the middle of the left coronary cusp. The extent of the left cusp is semicircular (indicated by the red arch); all of this territory must be mapped before selecting an ablation target. Note the extremely close relationship of the right coronary cusp and the right ventricular outflow tract (the attachment of the pulmonic valve is denoted by the arrow).
wide as the lumen, and because of the accompanying coronary arteries. Ablation in this area should be performed only after coronary angiography confirms a safe distance (at least 0.5 cm) between the artery and the ablation site.

LV idiopathic VT

Mapping and ablation of this macro-re-entrant VT is typically easy. The anterograde limb of the circuit, the so-called slow fiber can be difficult to map directly; the retrograde limb was always assumed to be the left posterior fascicle, but this has been called into question recently as atrial events can completely capture the ventricle with a narrow QRS during VT (by definition, using the posterior fascicle anterogradely) without disruption of the tachycardia (proving a separate retrograde limb). Nonetheless, recording of the left posterior fascicle during sinus rhythm establishes a region of interest, and ablation at the earliest fascicular potential during VT typically leads to prompt termination. Regular RF energy is sufficient, as the circuit seems very superficial. In fact, one of the pitfalls in mapping of this VT is the possibility of transient mechanical ablation with catheter movement. In this unfortunate circumstance, if waiting for recovery is unrewarding, empiric linear ablation perpendicular to the left posterior fascicle has proven successful in preventing VT recurrence.

The fascicles also represent sites of origin of focal idiopathic VTs, and are frequent sources of trigger events for polymorphic VT, as discussed above.

Papillary muscle VT

Idiopathic VT can arise from any of the papillary muscles (LV posterior medial or anterior lateral, or the single developed papillary muscle of the RV). LV papillary muscle VTs mimic the much easier LV idiopathic VT in an electrocardiographic sense, which makes sense as the papillary muscles are adjacent (in some portions apically) to the corresponding fascicles. These VTs are notoriously difficult to map and ablate, particularly because of the need for a true three-dimensional understanding of their anatomy. Tips to enhance this understanding include intracardiac echocardiography and making separate “chambers” of the papillary muscles on three-dimensional mapping systems. Ablation is also made more difficult by the high degree of motion of these structures and the difficulty stabilizing the catheter at precise locations. Irrigated RF delivery is important here, and often multiple lesions are required.

Summary

Idiopathic VT is a significant clinical problem, not in terms of prognostic implications, but because of symptoms and potential for misadventure in terms of PVC-related myopathy and, rarely, triggering polymorphic VT. Ablation is beneficial in most patients with sufficient symptoms to warrant treatment, and careful mapping informed by the intricacies of the common sites of origin is essential for success. Critical to ablation procedures is safety, particularly given the non-life-threatening nature of the problem and the common demographic of young healthy patients.


