Idiopathic Epicardial Left Ventricular Tachycardia Originating Remote From the Sinus of Valsalva

Electrophysiological Characteristics, Catheter Ablation, and Identification From the 12-Lead Electrocardiogram

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Background—Despite the success of catheter ablation for treatment of idiopathic ventricular tachycardia (VT), occasional patients have been reported in whom VT could not be ablated from the right or left ventricular endocardium or from the aortic sinus of Valsalva (ASOV).

Methods and Results—In 12 of 138 patients (9%) with idiopathic VT referred for ablation, an epicardial left ventricular site of origin was identified 10 mm from the ASOV. Coronary venous mapping demonstrated epicardial preceding endocardial activation by 10 ms (41±7 versus 15±11 ms before QRS onset; P<0.001). VT induction was facilitated by catecholamines and terminated by adenosine. Ablation through the coronary veins or via percutaneous transpericardial catheterization was successful in 9 patients; 2 required direct surgical ablation as a result of anatomic constraints. No ECG pattern was specific for epicardial VT. However, slowed initial precordial QRS activation, as quantified by a novel metric, the maximum deflection index, was more useful. A delayed precordial maximum deflection index ≥0.55 identified epicardial VT remote from the ASOV with a sensitivity of 100% and a specificity of 98.7% relative to all other sites of origin (P<0.001).

Conclusions—Although clinically underrecognized, idiopathic VT may originate from the perivascular sites on the left ventricular epicardium. The mechanism is consistent with triggered activity. It is amenable to ablation by transvenous or transpericardial approaches, although technical challenges remain. Recognition of a prolonged precordial maximum deflection index and early use of transvenous epicardial mapping are critical to avoid protracted and unsuccessful ablation elsewhere in the ventricles. (Circulation. 2006;113:1659-1666.)

Key Words: ablation • electrocardiography • pericardium • tachycardia • ventricles

I diopathic left ventricular tachycardia (ILVT) has diverse mechanisms and sites of origin. Most ILVT originates from the left ventricular endocardium, and the responses of these tachycardias to pharmacological agents, programmed electrical stimulation, mapping, and catheter ablation have been defined.1–6 An inability to successfully ablate some ILVT from the endocardium led to the recognition of epicardial sites of origin, particularly adjacent to the aortic sinus of Valsalva (ASOV).7–9 However, occasional examples of epicardial ILVT have been reported that arise remote from the aortic root and are not amenable to ablation via the ASOV.10–14 This latter group of tachycardias has not been systematically characterized.

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The purpose of this study was to define the electrophysiological features, anatomic substrate, and approach to catheter ablation of epicardial idiopathic ILVT not originating adjacent to the ASOV. Because early identification of this form of idiopathic VT would allow more directed mapping and potentially abrogate prolonged and unsuccessful ablation attempts elsewhere in the ventricles, we also developed a novel metric derived from the 12-lead ECG to facilitate the recognition of this arrhythmia.

Methods

Study Population

The study population consisted of 12 patients (8 women, 4 men) with a mean age of 44±21 years (range, 11 to 74 years). These patients were derived from a series of 138 consecutive patients undergoing electrophysiological evaluation and ablation of idiopathic ventricular tachycardia. The remaining patients had right ventricular endocardial outflow tract tachycardia (n=86), left ventricular endocardial tachycardia (n=33), or ASOV tachycardia (n=7). On presentation, study patients had 9±7 months of highly symptomatic tachycardia.
Electrophysiological Evaluation, Mapping, and Ablation

All patients were studied in the absence of antiarrhythmic drug therapy. Two multipolar catheters were positioned in the right atrium and/or right ventricle for pacing and recording. A 2.5F 16-pole microwire with 2-6-2-mm spacing (Pathfinder, Cardima, Inc, Fremont Calif) was initially positioned along the distal great cardiac vein (GCV) and proximal anterior interventricular vein (AIV). This catheter, or a smaller 1.5F 8-pole version, was subsequently moved to other areas of the coronary venous system as dictated by the ECG and findings of endocardial mapping. Careful attention was given to the proper placement of surface ECG electrodes. Surface electrocardiograms and intracardiac signals were recorded and displayed digitally and stored for offline analysis (Cardiolab, Prucka Engineering, Houston, Tex).

Programmed stimulation was performed from the right ventricular apex and right ventricular outflow with up to 3 extrastimuli and burst pacing. If VT was not induced, stimulation was repeated during infusion of isoproterenol (1 to 8 μg/min), epinephrine (5 to 30 μg/min), or phenylephrine (50 to 200 μg/min). If sustained VT was induced, adenosine (75 to 250 μg/kg) was administered intravenously through a central line. Mapping was performed during VT or premature beats if tachycardia could not be induced. Once VT was induced, right and left ventricular endocardial activation maps were sequentially through a central line. Mapping was performed during VT or premature beats if tachycardia could not be induced. Once VT was induced, right and left ventricular endocardial activation maps were sequentially performed in each patient using standard electroanatomic techniques (CARTO, Biosense/Webster, Diamond Bar, Calif). In all patients, mapping of the right ventricular outflow tract included recordings from within the pulmonary artery. However, pulmonary angiography and intracardiac echocardiography were not routinely performed. In patients with earliest endocardial activation in the outflow tract, a catheter was positioned above the aortic valve, and activation mapping was performed in each ASOV. Pacing was performed at sites with the earliest endocardial and epicardial activation times during tachycardia at cycle lengths 20 ms shorter than the tachycardia cycle length or during sinus rhythm at the tachycardia cycle length if sustained VT was not induced.

In selected patients who had not previously undergone endocardial ablation, an attempt was made to ablate the tachycardia from the endocardial site with earliest activation time with the use of a large-tip catheter (NAVI-STAR DS, Biosense Webster, Inc, Diamond Bar, Calif). Epicardial ablation was then performed at the site of earliest epicardial activation and the best pace-map match. Ablation was initially attempted within the coronary vein with a 6F 4-mm-tip deflectable catheter (Blazer, Boston Scientific, Natick, Mass) and power titrated to a maximum temperature of 60°C. If the venous approach was not successful or if an ablation catheter could not be advanced to the site of earliest venous activation, ablation was performed by a percutaneous transpericardial approach. Ablation in the pericardial space was performed with a standard 4-mm-tip catheter (NAVI-STAR, Biosense Webster, Inc), a 4-mm irrigated-tip electrode (Chilli, Boston Scientific), or a cryocatheter (Freezor Xtra, Cryocath, Montreal, Quebec). Regardless of the ablation approach, coronary arteriography was performed immediately after ablation and 30 minutes after the final energy application. When relevant, pacing at maximum output was performed at the site of ablation immediately before ablation to identify the potential risk of injury to the phrenic nerve.

ECG Analysis

Simultaneous 12-lead ECGs during VT were recorded digitally at high speed (100 to 200 mm/s) in all patients for offline analysis. Intervals were measured with electronic calipers. QRS duration, axis, preexcitation zone transition, voltage in every lead, and a new metric, the maximum deflection index (MDI), were calculated for each tracing. For comparison, ECGs also were analyzed from patients with idiopathic VT arising from other sites. ECGs were reviewed by 2 investigators blinded to the site of origin; discrepancies were adjudicated by a third investigator.

The MDI was calculated as follows. The QRS duration was measured as the interval between the earliest rapid deflection of the ventricular complex in any of the 12 simultaneous leads to the latest offset in any lead. Time to maximum deflection was measured from the onset of the QRS complex to the maximum deflection in each preexcitation lead; the maximum deflection was defined as the largest amplitude deflection either above or below the isoelectric line. The time to maximum deflection was divided by the QRS duration to obtain the MDI. The MDI was calculated for each preexcitation lead separately and as the shortest of any preexcitation lead (preexcitation MDI; Figure 1A).

Statistical Analysis

Data are reported as mean±SD or as frequencies. Continuous data were analyzed by paired or unpaired t test or ANOVA with Bonferroni post hoc testing for multiple comparisons when appropriate. Receiver-operating characteristic curves were used to determine the MDI cutoffs for optimal sensitivity and specificity for the identification of non-ASOV epicardial ILVT.

The authors had full access to the data and take full responsibility for its integrity. All authors have read and agree to the manuscript as written.

Results

Electrophysiological Characteristics

VT was incessant at baseline in 1 patient and required catecholamine facilitation in the remaining 10 (2 spontaneous, 3 programmed ventricular extrastimuli, 5 burst pacing). VT was sustained in 10 patients and nonsustained in 1 patient. The mean cycle length was 390±62 ms (range, 290 to 510 ms). Burst pacing terminated VT in all 10 patients with sustained tachycardia. Adenosine terminated VT in 8 of 9 patients with sustained tachycardia. Entrainment of the tachycardia was not observed in any patient. In 1 patient, only frequent premature beats with a morphology similar to that of the clinical sustained VT were initiated during catecholamine infusion.

Mapping and Ablation

In patients with epicardial ILVT, the earliest site of activation was the AIV-GCV junction in 3, the proximal AIV in 2, and the middle cardiac vein (MCV) in 3 (Table and Figure 2). The earliest activation time on the epicardium always preceded the earliest activation on the endocardium (−41±7 ms versus −15±11 ms; P<0.001; the Table). In the 7 patients with earliest activation in the AIV or AIV-GCV junction, activation of the left ASOV occurred >10 ms later. In contrast, of 7 patients successfully ablated from the left ASOV, activation in the sinus preceded earliest activation in the coronary vein by a minimum of 10 ms. By angiography, the shortest distance between the left aortic cusp and the mapping catheter at the AIV-GCV junction was at least 1 cm in all patients. Exact pace-map matches were never obtained.
from the endocardium. The best pace-map match always was recorded from the epicardium.

Endocardial ablation was unsuccessful in all 10 patients in whom it was attempted, despite the use of a large-tip or irrigated-tip catheter. In contrast, 11 of 12 patients underwent successful epicardial ablation using a variety of techniques (Table). The initial approach was transvenous in 6 patients (Figure 3 and Figures I and II in the online Data Supplement) and was successful in 5 (2 proximal MCV, 2 proximal AIV, 1 GCV). In the remaining 6 patients, an ablation catheter could not be advanced to the site of earliest coronary venous activation. A percutaneous transpericardial approach was used as the initial procedure in 6 patients and after unsuccessful transpericardial ablation in 1 patient (Figure 4). The site of earliest activation recorded transpericardially was within 5 mm of the site of earliest venous activation in all 7 patients, confirming the perivascular origin of this arrhythmia. In 4 of 7 patients, the transpericardial approach was successful (2 AIV-GCV junction, 1 GCV, 1 distal MCV). In all 3 patients with unsuccessful transpericardial ablation, VT arose from the proximal AIV or AIV-GCV junction. Two of these patients ultimately underwent direct surgical cryoablation as a result of continued episodes of highly symptomatic tachycardia unresponsive to drug therapy. Both patients undergoing surgery had a thick layer of epicardial fat (3 to 5 mm) over the site of earliest activation adjacent to the AIV. One of these patients also had an intramural course of both the artery and vein at the site of earliest epicardial activation.

Discrete high-frequency potentials and/or multicomponent electrograms were recorded from the coronary veins at the site of earliest epicardial activation in 8 of 12 patients (Data Supplement Figure IIB), including 4 of 5 successfully ablated from the vein. Similar potentials were recorded on the epicardium in 3 of 7 patients undergoing transpericardial mapping (Figure 4C), including 3 of 4 successfully ablated by this approach. In 1 of the patients requiring surgical ablation, an initial high-frequency potential was recorded from the vein, while a simultaneous recording from the pericardial space directly over the site of earliest venous activation demonstrated only a low frequency and somewhat later electrogram (Data Supplement Figure III). This finding may reflect the insulating effect of the thick layer of epicardial fat found at surgery.

Coronary arteriography performed 30 minutes after ablation and exercise perfusion imaging at 3 months were normal in all patients. One patient had pericarditis associated with a small pericardial effusion after transpericardial ablation that resolved spontaneously after 1 week of therapy with indomethacin. There were no other short- or long-term complications. All patients were discharged without antiarrhythmic therapy. During a mean follow-up of 23±15 months, 1 patient with initially successful ablation and the patient with unsuccessful ablation had recurrent VT treated with antiarrhythmic therapy. The remaining 10 patients (83%) are free of recurrent symptoms and tachycardia without drug therapy.

**ECG Characteristics**

There were no significant differences in QRS duration, frontal plane axis, and lead voltage when epicardial ILVT arising remote from the ASOV was compared with other sites of origin. All patients with AIV tachycardia had a left bundle pattern and a precordial transition beyond V2. In the 3 patients in whom the tachycardia arose at the AIV-GCV junction, the R/S amplitude index in V1 or V2 was ≥0.5, and the R-wave duration index was ≥0.3, similar to that reported for ASOV tachycardias8,13 (Figure 4A). In the remaining 4 patients in whom the tachycardia arose more distally in the AIV, R waves were smaller and narrower, and the ECG was indistinguishable from tachycardia originating from the right ventricular septal endocardium (Figure 3A and Data Supplement Figure III). In all 7 patients, there were tall peaked R waves in the inferior leads (usually >15 mV). Lead I...
The precordial MDI (0.61; 95% CI, 0.59 to 0.63) was demonstrated very low amplitude (≤0.2 mV) in 3 patients and an rS or QS pattern in the remainder.

The precordial MDI (0.61; 95% CI, 0.59 to 0.63) was significantly longer in patients with epicardial ILVT relative to other sites of idiopathic VT origin: endocardial ILVT, 0.40 (95% confidence interval [CI], 0.38 to 0.42; P<0.001); endocardial right ventricular outflow tract tachycardia, 0.46 (95% CI, 0.37 to 0.54; P=0.001) (Figure 1B). The receiver-operating characteristic curve identified an MDI ≥0.55 as the optimal cut point, with 100% sensitivity and 98.7% specificity for detection of non-ASOV epicardial origin. Only 1 ASOV tachycardia had a long MDI. Among MDIs calculated from individual precordial leads, the V5 MDI provided the best discrimination (Data Supplement Figure IV) but had greater overlap between groups.

### Discussion

#### Characterization of Epicardial ILVT of Perivascular Origin

In this study, epicardial ILVT arising remote from the ASOV was not uncommon, occurring in ∼9% of all patients referred for catheter ablation of idiopathic VT. These tachycardias share several common features suggesting a unique clinical entity: perivascular origin, catecholamine enhancement, and adenosine sensitivity. The last 2 characteristics, combined with the inability to entrain the tachycardia, are most compatible with triggered activity as the underlying mechanism.19 The anatomic substrate for this arrhythmia, particularly the propensity for perivascular sites, is unclear. Percutaneous transpericardial mapping in 7 patients confirmed that the close proximity of the tachycardia origin to the coronary vasculature was not simply a result of limited epicardial mapping via the transvenous approach. Similarly, elimination of the tachycardia with low-power radiofrequency energy application from within the vein suggests a close relationship to the site of origin. Participation of muscular coats enveloping the proximal pulmonary veins in the genesis of atrial arrhythmias is widely recognized.20 However, the muscular coat of the coronary venous system is limited to the coronary sinus, the proximal few millimeters of the GCV, and rarely the proximal few millimeters of the MCV.21 The tachycardias described in this study arise more distally, suggesting little role for perivenous muscular coats. The media of coronary veins do contain myocytes embryologically derived from primordial right atrial tissue, but the potential role of such cells in arrhythmogenesis is unknown.22 Further work is

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**Results of Mapping and Ablation**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age, y</th>
<th>ECG*</th>
<th>Location</th>
<th>Epicardial AT, ms</th>
<th>Endocardial AT, ms</th>
<th>Ablation Approach†</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>26</td>
<td>LBBB, 90</td>
<td>AIV-GCV</td>
<td>−43 (SOV 5)</td>
<td>−28 (LV)</td>
<td>Successful CV RF (15 W, 210 s)</td>
</tr>
<tr>
<td>2</td>
<td>70</td>
<td>LBBB, 90</td>
<td>AIV-GCV</td>
<td>−49 (SOV −26)</td>
<td>−10 (LV)</td>
<td>Successful TP catheter cryoablation (4 applications§)</td>
</tr>
<tr>
<td>3</td>
<td>11</td>
<td>LBBB, 90</td>
<td>AIV-GCV</td>
<td>−32 (SOV −15)</td>
<td>−20 (LV)</td>
<td>Successful TP RF irrigated tip (45 W, 240 s) and catheter cryoablation (4 applications§)</td>
</tr>
<tr>
<td>4</td>
<td>45</td>
<td>LBBB, 120</td>
<td>Prox AIV (1.5 cm)‡</td>
<td>−35 (SOV −5)</td>
<td>−15 (RV)</td>
<td>Successful TP RF irrigated tip (50 W, 295 s)</td>
</tr>
<tr>
<td>5</td>
<td>70</td>
<td>LBBB, 120</td>
<td>Prox AIV (1 cm)‡</td>
<td>−41 (SOV −5)</td>
<td>−30 (RV)</td>
<td>Successful CV RF 4-mm tip (10 W, 184 s)</td>
</tr>
<tr>
<td>6</td>
<td>34</td>
<td>LBBB, 120</td>
<td>Prox AIV (2.5–3 cm)‡</td>
<td>−45 (SOV −3)</td>
<td>0 (RV)</td>
<td>Successful TP RF irrigated tip (50 W, 276 s)</td>
</tr>
<tr>
<td>7</td>
<td>47</td>
<td>LBBB, 120</td>
<td>Prox AIV (1.5 cm)‡</td>
<td>−48 (SOV 0)</td>
<td>−18 (RV)</td>
<td>Successful CV RF 4-mm tip (15 W, 175 s)</td>
</tr>
<tr>
<td>8</td>
<td>58</td>
<td>LBBB, 100</td>
<td>GCV</td>
<td>−44</td>
<td>−15 (LV)</td>
<td>Successful CV RF 4-mm tip (5 W, 60 s)</td>
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<tr>
<td>9</td>
<td>74</td>
<td>LBBB, 150</td>
<td>GCV</td>
<td>−53</td>
<td>−20 (LV)</td>
<td>Successful TP RF 4-mm tip (35 W, 390 s)</td>
</tr>
<tr>
<td>10</td>
<td>50</td>
<td>LBBB, −30</td>
<td>Prox MCV</td>
<td>−32</td>
<td>10 (RV)</td>
<td>Successful CV RF (20 W, 210 s)</td>
</tr>
<tr>
<td>11</td>
<td>37</td>
<td>LBBB, −30</td>
<td>Prox MCV</td>
<td>−37</td>
<td>−20 (RV)</td>
<td>Successful CV RF 4-mm tip (15 W, 150 s)</td>
</tr>
<tr>
<td>12</td>
<td>34</td>
<td>RBBB, −115</td>
<td>Distal MCV</td>
<td>−38</td>
<td>−10 (LV)</td>
<td>Successful TP RF 4-mm tip (40 W, 180 s)</td>
</tr>
</tbody>
</table>

LBBB indicates left bundle-branch block; LV, left ventricle; CV, coronary venous; RF, radiofrequency; TP, transpericardial; Prox, proximal; AT, activation time; and RBBB, right bundle-branch block.

*Precordial lead pattern, ORS axis.
†Maximum radiofrequency power in watts; duration in seconds.
‡Distance from AIV-GCV junction.
§Cryoapplications given for 4 minutes; minimum temperature <−80°C.
required to clarify the anatomic basis for the distribution of these arrhythmias.

Epicardial ILVT arising remote from the ASOV may be more common than previously recognized. Ito and colleagues\(^{13}\) reported 8 of 80 patients with idiopathic outflow tract tachycardias that could not be ablated from the right or left ventricular endocardium or the ASOV and were of presumed epicardial origin. Coronary venous mapping suggested an origin near the proximal AIV in several patients.\(^{12}\) However, additional epicardial mapping and catheter ablation were not attempted. Tanner et al\(^{14}\) reported 5 of 33 patients with idiopathic outflow tract tachycardia not amenable to ablation from the left or right ventricular endocardium or the ASOV. Three patients underwent successful ablation via a coronary venous approach and 2 by a transpericardial approach. Additional details about the site of origin and electrophysiological characteristics were not provided. Stellbrink et al\(^{10}\) described successful ablation of an incessant adenosine-sensitive idiopathic ILVT successfully ablated from a marginal branch of the GCV after unsuccessful endocardial ablation. The present study extends these observations in a larger series of patients, indicates a wider distribution of perivascular sites of origin, provides detailed information about mapping and ablation, and presents evidence for a common mechanism.

**Relationship to ILVT Originating From the ASOV**

ILVT successfully ablated from the ASOV appears to arise from the epicardium subjacent to the valve leaflets, most commonly the left coronary cusp.\(^{7-9}\) Crescents of ventricular myocardium are incorporated into the base of the right and left coronary sinuses at the VA junction.\(^{23}\) Kanagaratnam and colleagues\(^{7}\) indicated that even earlier activation times could be identified in the anterior epicardial space between the atrial appendage and the aortic root. However, these sites had large atrial electrograms, and only the atrium could be captured during attempts at pace mapping. Simultaneous recording of potentials from the AIV was not performed. In our series, all patients with AIV tachycardia had venous activation times preceding that in the ASOV by at least 10 ms (Figures 3D and 4C). In patients undergoing successful ablation from the ASOV, the converse was true. Although it is possible that some of the tachycardias mapped to the AIV-GCV junction in this study are similar to those arising from the ASOV and could be ablated from that site, the distance between the left ASOV and the junction (\(>1\) cm) suggests that this possibility is unlikely. The ECG appearance of ILVT arising near the AIV is similar to those originating from the ASOV when earliest venous activation is at the AIV-GCV junction and resembles tachycardias arising from the right ventricular septal endocardium when earliest activation arises somewhat more distally in the AIV. These observations underscore the importance of simultaneous coronary venous mapping during electrophysiological evaluation of problematic outflow tract tachycardias. Finally, ventricular tachycardia also may arise from the main pulmonary artery above the valve, with ECG patterns occasionally similar to those arising from the AOV or AIV.\(^{24,25}\) However, activation in the pulmonary artery was not early in any of the study patients.

**Approaches to Catheter Ablation**

As demonstrated in the present series and in previous case reports, ablation via a transvenous approach is feasible and

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**Figure 3.** A, ECG of a proximal AIV tachycardia. B, Electroanatomic endocardial activation maps of VT. Earliest activation was on the septal right ventricular tract 18 ms before QRS onset, when endocardial ablation with an 8-mm electrode catheter failed to terminate VT (pink dots). C, Recordings from a mapping microcatheter positioned in the distal GCV and AIV demonstrating earliest epicardial activation 48 ms before QRS onset (arrow). D, Right anterior oblique radiograph immediately before ablation demonstrating the positions of the ablation electrode in the proximal AIV (large arrow), a Judkins catheter positioned at the left coronary artery ostium (small arrow), and an diagnostic catheter in the right ventricular outflow tract at the earliest site of endocardial activation (double arrows). E indicates epicardial venous mapping catheter; LVS, left ventricular septum; OT, right ventricular outflow tract; and RVA, right ventricular apex.
often effective. With conventional 4-mm electrodes, delivery of \( >10 \) to \( 15 \) W seldom is possible because of limited electrode cooling, leading to a rapid rise in electrode temperature. However, even low power often is sufficient. Currently available ablation catheters often are difficult to advance beyond the distal GCV or the proximal portions of the MCV because of the tortuosity, sharp angulation, and small caliber of the distal veins.

The percutaneous transpericardial approach permits mapping access to the entire epicardial surface and the deployment of a greater range of ablation catheters and energy sources. Despite the versatility of this approach, barriers to effective energy delivery adjacent to the coronary vessels remain. In 2 patients undergoing surgery after unsuccessful transpericardial ablation of AIV tachycardias, extensive epicardial fat in both and an intramural location of the coronary vessels in 1 patient may have contributed to the failure of this method. In the experimental study of d’Avila and colleagues,\textsuperscript{26} epicardial ablation with an irrigated electrode produced deeper lesions (mean, \( 6.7\pm1.7 \) mm) than conventional electrodes. However, no myocardial lesions were produced when the layer of overlying fat was \( >3.5 \) mm. In addition, coronary blood flow may shield and cool the subepicardium beneath the vasculature.\textsuperscript{27} Alternate energy sources potentially could be more effective. In this study, catheter cryoablation was successful in 1 patient when used as initial therapy but not in a second patient after unsuccessful irrigated-tip radiofrequency ablation. In the normal canine heart, Lustgarten and colleagues\textsuperscript{28} found that epicardial catheter cryoablation with a 6-mm electrode produced a mean lesion depth of \( 1.6\pm0.7 \) mm with a maximum depth of \( 2.4 \) mm.

Ablation adjacent to the coronary artery carries significant potential for arterial injury. D’Avila et al\textsuperscript{29} demonstrated that the risk of damage during radiofrequency ablation directly over the artery, including thrombosis, medial necrosis, and hyperplasia, was inversely proportional to vessel diameter; there was little evidence of injury when vessel diameter exceeded \( 0.5 \) to \( 1.0 \) mm. Similar findings were reported for cryoablation.\textsuperscript{28} We found no clinical evidence of arterial

![Figure 4. A, A 12-lead ECG of a tachycardia arising at the AIV-GCV junction. B, Surface and intracardiac electrograms from the same patient. Recordings from a coronary venous mapping catheter positioned with the proximal poles (AIV, 15 to 16) at the AIV-GCV junction demonstrate earliest activation at the junction, preceding the surface QRS by 43 ms (large solid arrow). Activation of the left ASOV, recorded simultaneously, does not begin until after QRS onset (large open arrow). Atrial electrograms can be seen from the proximal electrodes of the venous mapping catheter (small arrows). C, Electroanatomic map of epicardial activation recorded by a percutaneous transpericardial approach, shown in a superior (cranial) view. The grid scale is 1.85 cm. The pink dot represents the site of successful ablation; orange dot, earliest activation in the left sinus of Valsalva, \( >2 \) cm from the successful ablation site; and dashed line, the approximate location of the left coronary artery. Inset, Electrogram (\( \sim0.20 \) mV) at the site of successful ablation \( \sim45 \) ms before QRS onset. E indicates epicardial venous mapping catheter; LSV, left sinus of Valsalva; LAO, left anterior oblique; and RVA, right ventricular apex.](image)
injury in any patient in this study. Although high flow through larger epicardial vessels may provide some protection from arterial injury, careful monitoring of electrode position relative to the artery, postprocedural assessment of arterial patency, and restriction of the procedure to highly symptomatic patients remain prudent.

**ECG Correlations**

In this study, a long precordial MDI, reflecting delayed initial activation of the left ventricle, reliably discriminated epicardial ILVT from other sites of origin with high sensitivity and specificity. This observation is consistent with slower spread of activation from a focus on the epicardial surface relative to the endocardium and delayed global ventricular activation resulting from later engagement of the His-Purkinje network.\(^\text{30,31}\) Rodriguez and colleagues\(^\text{12}\) suggested that a pseudo “delta wave” at QRS onset suggested an epicardial focus; this finding was present in several patients with unsuccessful ablation of apparent right ventricular outlet tract tachycardia. Recently, Berreuzo et al\(^\text{13}\) reported that a delayed intrinsicoid deflection in lead V\(_2\) identified patients with an epicardial site of origin with a sensitivity of 87% and a specificity of 90%. This analysis excluded patients with structurally normal hearts or with left bundle configuration tachycardia. Our findings extend these observations to patients with ILVT, with a technique independent of specific QRS configuration. Prospective evaluation is required to confirm the utility of the MDI for identifying epicardial ventricular tachycardia, particularly in patients with structural heart disease.

**Clinical Implications**

Epicardial ILVT arising remote from the ASOV appears to be a catecholamine-enhanced, adenosine-sensitive tachycardia consistent with cAMP-mediated triggered activity in most cases. These tachycardias arise adjacent to epicardial coronary vasculature. Despite technical challenges, they are amenable to either transvenous or percutaneous transpericardial catheter ablation. The MDI, derived from a 12-lead ECG, is enabling to either transvenous or percutaneous transpericardial catheter ablation. The MDI, derived from a 12-lead ECG, is enabled to either transvenous or percutaneous transpericardial catheter ablation. This study, a long precordial MDI, reflecting delayed initial activation of the left ventricle, reliably discriminated epicardial ILVT from other sites of origin with high sensitivity and specificity. This observation is consistent with slower spread of activation from a focus on the epicardial surface relative to the endocardium and delayed global ventricular activation resulting from later engagement of the His-Purkinje network.\(^\text{30,31}\) Rodriguez and colleagues\(^\text{12}\) suggested that a pseudo “delta wave” at QRS onset suggested an epicardial focus; this finding was present in several patients with unsuccessful ablation of apparent right ventricular outlet tract tachycardia. Recently, Berreuzo et al\(^\text{13}\) reported that a delayed intrinsicoid deflection in lead V\(_2\) identified patients with an epicardial site of origin with a sensitivity of 87% and a specificity of 90%. This analysis excluded patients with structurally normal hearts or with left bundle configuration tachycardia. Our findings extend these observations to patients with ILVT, with a technique independent of specific QRS configuration. Prospective evaluation is required to confirm the utility of the MDI for identifying epicardial ventricular tachycardia, particularly in patients with structural heart disease.

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CLINICAL PERSPECTIVE

Ventricular tachycardia (VT) in the absence of structural heart disease is a common clinical entity. Drug therapy often is ineffective or poorly tolerated. Idiopathic VT generally arises from the right or left ventricular endocardium or, less commonly, from the aortic sinus of Valsalva (ASOV) or pulmonary artery. Catheter ablation has been highly effective in eliminating this form of VT, with a reported success rate of ≥90%. In the present study, we report a series of patients with idiopathic VT that could not be ablated from these sites. These tachycardias arose from the left ventricular epicardium in close proximity to the coronary vasculature. Coronary venous or percutaneous transpericardial mapping facilitated identification of an epicardial site of origin in all patients. Most of these tachycardias were successfully ablated, although technical challenges related to access and the insulating effects of epicardial fat remain. An epicardial location of idiopathic VT can be suspected on the basis of delayed precordial ECG lead activation as quantified in a new ECG metric developed from these patients. These tachycardias are not rare (9% of a consecutive series of 138 patients with idiopathic VT presenting for ablation) and may be a common cause of failed ablation. In this group of patients, careful evaluation of the ECG and early use of epicardial mapping can help avoid prolonged and unsuccessful ablation attempts at other ventricular sites.