Mapping and Ablation of Epicardial Idiopathic Ventricular Arrhythmias From Within the Coronary Venous System

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Background—The prevalence of epicardial idiopathic ventricular arrhythmias that can be ablated from within the coronary venous system (CVS) has not been described.

Methods and Results—In a consecutive group of 189 patients with idiopathic ventricular arrhythmias referred for ablation, the site of origin (SOO) of ventricular tachycardia and/or premature ventricular contractions was determined by activation mapping and pace mapping. Mapping was performed within the CVS if endocardial mapping did not reveal an SOO. Venography of the CVS and coronary angiography were performed before ablation in the CVS. In 27 of 189 patients (14%±5%; 95% confidence interval), the SOO of the ventricular arrhythmia was identified from within the coronary venous system, either in the great cardiac vein (n=26) or the middle cardiac vein (n=1). The mean activation time at the SOO was −29±8 ms. Twenty of 27 patients (74%) underwent successful ablation within the CVS. Epicardial ventricular arrhythmias displayed a broader R wave in V1 compared with arrhythmias in the control group (85 ms [interquartile range, 40] versus 65 ms [interquartile range, 95]; P<0.01). Two patients had recurrent premature ventricular contractions within 2 weeks after ablation, and no recurrences occurred in the remaining patients during a median follow-up of 13 months (range, 25). In the 7 patients with unsuccessful ablation, failure was because the ablation catheter could not be advanced to the SOO within the great cardiac vein (n=4), inadequate power delivery at the SOO (n=1), proximity to the phrenic nerve (n=1), or proximity of the SOO to a major coronary artery (n=1). Transcutaneous epicardial ablation was effective in 1 of 2 patients in whom it was attempted.

Conclusions—Almost 15% of idiopathic ventricular arrhythmias have an epicardial origin. ECG characteristics help to differentiate epicardial arrhythmias from endocardial ventricular arrhythmias. The SOO of epicardial arrhythmias can be ablated from within the CVS in approximately 70% of patients. (Circ Arrhythm Electrophysiol. 2010;3:274-279.)

Key Words: ventricular arrhythmias ■ ablation ■ coronary venous system ■ mapping

The prevalence and results of radiofrequency catheter ablation of idiopathic ventricular arrhythmias originating in the epicardium have not been systematically studied. The purpose of the present study was to evaluate the prevalence of epicardial idiopathic ventricular arrhythmias and their ECG characteristics as well as the feasibility of radiofrequency ablation of these arrhythmias from within the coronary venous system (CVS).

Methods

Patient Characteristics

The subjects of this study were a consecutive group of 189 patients with idiopathic premature ventricular contractions (PVCs) or ventricular tachycardia (VT) referred for catheter ablation. Twenty-seven of the 189 patients were found to have an epicardial site of origin (SOO) and are the focus of this study.

Eighteen of the 27 patients had frequent PVCs, and 9 patients had sustained VT. The indications for an ablation procedure included highly symptomatic palpitations (n=24) or left ventricular dysfunction (n=3). There was no evidence of structural heart disease in these patients based on echocardiography, stress testing, and cardiac MRIs. A 24-hour Holter monitor was performed before ablation in all patients. These 27 patients were compared with a randomly chosen series of 27 patients with idiopathic ventricular arrhythmias in whom the site of origin was in the right ventricular outflow tract (n=13), an aortic cusp (n=8), the left ventricular outflow tract (n=5), or the mitral annulus (n=1). The study subjects and the control group were compared statistically and found to be similar in age and sex.

Electrophysiology Study and Mapping

After informed consent was obtained, multipolar electrode catheters were inserted into a femoral vein and positioned in the right ventricular apex, the His bundle position, and the high right atrium. Subsequently, 3000 U of heparin was administered. If mapping in the left ventricle or aortic cusps was necessary, the patient received additional heparin to achieve an activated clotting time of 250 to 300 seconds.

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Programmed atrial and ventricular stimulation was performed with up to 4 extrastimuli to assess for the inducibility of VT. If no ectopy was present at baseline, isoproterenol was infused at rates up to 20 μg/min.

In the presence of frequent spontaneous ectopy, activation mapping was performed using a 4-mm- or 3.5-mm-tip catheter (Navistar or ThermoCool, Biosense Webster, Diamond Bar, Calif). In the single patient without frequent PVCs, pace mapping was used to identify the SOO. Pace mapping was performed at a pacing cycle length equal to the coupling interval of the spontaneous PVCs. In all patients, a 3D electroanatomic mapping system was used for activation mapping (Biosense Webster). The SOO was considered to be epicardial if the earliest site of activation and a matching pace map were identified from within the CVS (Figure 1).

If the PVCs had a left bundle-branch block morphology, activation mapping first was performed in the right ventricular outflow tract followed by the coronary venous system, the aortic cusp, and the left ventricle. If the PVCs had a right bundle-branch block morphology, activation mapping of the aortic cusps and the left ventricular endocardium first was performed using a retrograde aortic approach. If the earliest activation time from the endocardium/aortic cusp was <10 ms before the onset of the QRS complex, a 2.5F multipolar catheter was introduced into the CVS (Pathfinder, CARDIMA, Inc, Fremont, Calif; Figure 2). Venography of the coronary venous system was performed (Figure 3, left panel). The 2.5F multipolar catheter was positioned in different coronary venous branches until the earliest site of local endocardial activation was detected. Pace mapping was performed at this site.

Radiofrequency Ablation Within the Coronary Venous System

Radiofrequency ablation was performed at the site of earliest activation in the CVS with the 3.5-mm open-irrigated-tip catheter (Thermocool, Biosense Webster). If this catheter could not be maneuvered to the SOO, a 6F or 7F Blazer catheter (Boston Scientific, Natick, Mass) or a 5F Mariner catheter (Medtronic Inc, Minneapolis, Minn) was used. Once the catheter was positioned at the SOO, a coronary angiogram was performed to assess the distance of the catheter tip to a major epicardial coronary artery (Figure 3, right panel). If this distance was <4 mm, ablation was not attempted. To avoid phrenic nerve injury, ablation also was not attempted if there was diaphragmatic capture when pacing at an output of 20 mA with the ablation catheter.

Radiofrequency energy was delivered with the irrigated-tip catheter at a power of 15 to 25 W for up to 60 seconds. If radiofrequency energy delivery resulted in a temperature >45°C, a 6-mm Cryocatheter (Medtronic Inc) was used for cryoablation, with a target temperature of −30°C. For nonirrigated-tip catheters, the target temperature was set to 55°C with a power of 15 to 25 W. Impedance was assessed during these applications. After ablation of PVCs, isoproterenol was administered at rates up to 20 μg/min to assess for inducible PVCs. Coronary angiography was repeated after ablation to assess for patency of the coronary arteries in the vicinity of the SOO. Programmed stimulation was repeated after ablation.

Imaging of the Coronary Venous System

An occlusive angiogram of the CVS was performed to assess the CVS anatomy (Figures 2 and 3). The great cardiac vein was divided into 2 segments: proximal and distal (Figures 3 and 4). The proximal segment was defined as the segment from the beginning of the great cardiac vein to the site where the vein assumes an anterior course along the left anterior descending artery in the interventricular groove. The segment beyond this point was defined as the distal segment (Figure 4).

ECG Analysis

Sixteen of the patients with idiopathic epicardial ventricular arrhythmias had a left bundle-branch block PVC morphology compared with 17 patients in the control group. All but 1 had an inferior axis. R-wave width and S-wave width in leads V1 and V2 as well as the maximal deflection index (interval measured from the beginning of the QRS complex to the maximum deflection in the precordial leads divided by the QRS duration) were assessed for the targeted arrhythmias (Figure 5). ECG data were analyzed by 2 observers blinded to the site of origin of the targeted arrhythmia. Discrepancies were resolved by a third observer.

Follow-Up

Therapy with antiarrhythmic medications was discontinued if ablation was acutely successful. Patients were seen in an outpatient clinic 3, 12, and 18 months after the ablation procedure and underwent 24-hour Holter monitoring at 3 months. The median duration of follow-up was 13 (range, 25) months. Successful ablation was defined as an 80% decrease in PVC burden at 3 months’ follow-up. The PVC burden was defined as the percentage of PVCs relative to the total number of QRS complexes on a 24-hour Holter monitor.

Statistical Analysis

Continuous variables were expressed as mean±1 standard deviation. Procedural details among patients with and without a successful ablation
procedure were compared using the Student t test. Procedural and ECG comparisons of those patients with epicardial arrhythmias (n=27) versus a control group (n=27) were performed using the Wilcoxon rank sum test. Categorical variables were compared with the χ² test. If the expected sample size was smaller than 5 cells, the Fisher exact test was used. Paired-sample t tests were used to compare PVC burden before and after ablation. A probability value <0.05 was considered statistically significant. SPSS 17.0 software (SPSS Inc, Chicago, Ill) was used for calculations.

Results

Patient Characteristics

Twenty-seven of the 189 patients (14%±5%; 95% confidence interval) with idiopathic PVCs or VT referred for catheter ablation were found to have an epicardial SOO. Patients with arrhythmias originating in the epicardium (10 men; age, 51±17 years) more often were on antiarrhythmic medications than were the patients in the control group (14 men; mean age, 55±16 years; 8 of 27 versus 2 of 27; P=0.04). Nine of the 27 patients with epicardial arrhythmias had VT; the remaining patients had frequent PVCs. All but 1 of the patients in both groups had an inferior axis morphology, consistent with an outflow tract origin of the arrhythmia. In the control group, 6 patients had VT and the remaining patients had frequent PVCs. The mean PVC burden before catheter ablation was 22±11%, with a range of 2.1% to 42%. The PVCs were monomorphic in 17 patients and pleomorphic in 10 patients. The median duration of palpitations was 3.5±4.6 years. All VTs in patients with epicardial arrhythmias had a right bundle-branch block and inferior axis morphology, with a mean cycle length of 357±81 ms.

![Diagram of the heart showing the great cardiac vein (GCV) and its proximal and distal segments.](image)

Figure 3. Left, Venogram of the great cardiac vein (GCV). The great cardiac vein is divided in a proximal (prox.) and distal segment. The catheter is located at the end of the proximal segment (asterisk). In the distal segment, the great cardiac vein changes its circular course around the atrioventricular junction to an anterior course following the left anterior descending artery. Right, The ablation catheter is located at the site of the earliest activation during ventricular ectopy. A coronary angiogram illustrates the proximity to both left anterior descending coronary artery and left circumflex coronary artery.

![Diagram of the anterior aspect of the heart showing the course of the great cardiac vein in relation to the left anterior descending coronary artery (LAD) and the left circumflex coronary artery (Cx).](image)

Figure 4. CT angiography of the anterior aspect of the heart illustrating the course of the great cardiac vein in relation to the left anterior descending coronary artery (LAD) and the left circumflex coronary artery (Cx).

![ECG tracings showing PVCs ablated in the right ventricular outflow tract and epicardial PVCs.](image)

Figure 5. Left, ECG of a PVC that was ablated in the right ventricular outflow tract. Note the sharp R wave in V₁ (40 ms) compared with middle panel. The S wave is broader in V₁, compared with that in the middle panel. Middle, ECG of an epicardial PVC that was ablated within the proximal great cardiac vein. The R wave in V₁ measures 100 ms and the S wave 57 ms. Right, ECG of an epicardial PVC where the earliest activation was within the distal great cardiac vein. Ablation within the distal great cardiac vein was only temporarily effective. Note the narrow R wave in lead V₁ (65 ms), consistent with a site of origin in the right ventricular outflow tract.
ECG Characteristics

Epicardial arrhythmias had a distinct morphology, depending on the SOO along the great cardiac vein. The epicardial arrhythmias that had an SOO in the proximal segment of the great cardiac vein had a right bundle-branch block morphology. When the SOO was more distal, the ventricular arrhythmias had a left bundle-branch block morphology. Epicardial arrhythmias had a broader R wave and a smaller S wave in lead V1 compared with endocardial arrhythmias. In patients with epicardial left bundle-branch block ventricular arrhythmias, the R wave in V1 was broader than 75 ms, whereas 12 of 16 patients with epicardial left bundle-branch block arrhythmias had an R wave ≥75 ms. Also, the maximal deflection index was higher in epicardial than endocardial arrhythmias. The arrhythmias with an SOO in the great cardiac vein that were more distal had a broader S wave (median, 60 ms [interquartile range, 25]) compared with 36 ms (IQ range 63) in the control group (P<0.0001; Figure 6). Only 1 patient in the control group had an R wave in V1 broader than 75 ms, whereas 12 of 16 patients with epicardial left bundle-branch block arrhythmias had an R wave ≥75 ms. Also, the maximal deflection index was higher in epicardial than endocardial arrhythmias.

Compared with left bundle-branch block arrhythmias originating in the coronary cusps, the R wave in V1 was broader in epicardial arrhythmias compared with cusp arrhythmias (85 ms [IQ range, 20] versus 35 ms [IQ range, 60]; P=0.01).

The maximal deflection index was no different when cusp arrhythmias were compared with epicardial arrhythmias (0.69 [IQ range, 0.16] versus 0.65 [IQ range, 0.19]; P=0.2).

### Table 1. Comparison of ECG Characteristics in Patients With Epicardial Arrhythmias Compared With a Control Group

<table>
<thead>
<tr>
<th></th>
<th>Epicardial SOO</th>
<th>Control Group</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample size, n</td>
<td>27</td>
<td>27</td>
<td></td>
</tr>
<tr>
<td>QRS, ms</td>
<td>140 (20)</td>
<td>145 (20)</td>
<td>0.88</td>
</tr>
<tr>
<td>Arrhythmia morphology</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LBBB/RBBB</td>
<td>16/11</td>
<td>17/10</td>
<td>0.42</td>
</tr>
<tr>
<td>Axis: IA/SA</td>
<td>26/1</td>
<td>27/0</td>
<td>0.32</td>
</tr>
<tr>
<td>R-wave width in V1, ms</td>
<td>85 (40)</td>
<td>65 (95)</td>
<td>0.02</td>
</tr>
<tr>
<td>S-wave width in V1, ms</td>
<td>40 (43)</td>
<td>80 (93)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>R-wave width in V2, ms</td>
<td>80 (53)</td>
<td>70 (78)</td>
<td>0.40</td>
</tr>
<tr>
<td>S-wave width in V2, ms</td>
<td>50 (35)</td>
<td>80 (68)</td>
<td>0.23</td>
</tr>
<tr>
<td>Time to maximum deflection, ms</td>
<td>100 (20)</td>
<td>85 (19)</td>
<td>0.002</td>
</tr>
<tr>
<td>MDI</td>
<td>0.69 (0.16)</td>
<td>0.59 (0.15)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

LBBB indicates left bundle-branch block; RBBB, right left bundle-branch block; and MDI, maximal deflection index. Values are median (interquartile range).

### Table 2. Procedural Details in Patients With and Without an Acutely Successful Ablation Procedure

<table>
<thead>
<tr>
<th></th>
<th>Total (n=27)</th>
<th>Ablation Effective (n=20)</th>
<th>Ablation Ineffective (n=7)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Procedure time, min</td>
<td>296±56</td>
<td>292±55</td>
<td>303±63</td>
<td>1.0</td>
</tr>
<tr>
<td>Fluoroscopy time, min</td>
<td>57±15</td>
<td>56±16</td>
<td>60±14</td>
<td>1.0</td>
</tr>
<tr>
<td>Radiofrequency ablation time, min</td>
<td>6.2±7.6</td>
<td>5.5±4.7</td>
<td>6.3±6.8</td>
<td>0.78</td>
</tr>
<tr>
<td>QRS width V1, ms</td>
<td>147±20</td>
<td>152±23</td>
<td>137±9</td>
<td>0.11</td>
</tr>
<tr>
<td>R-wave width in V1, ms</td>
<td>82±48</td>
<td>113±40</td>
<td>76±7</td>
<td>0.03</td>
</tr>
<tr>
<td>S-wave width in V1, ms</td>
<td>61±43</td>
<td>39±24</td>
<td>61±8</td>
<td>0.03</td>
</tr>
<tr>
<td>Activation time at SOO, ms</td>
<td>−29±8</td>
<td>−30±7</td>
<td>−29±6</td>
<td>0.65</td>
</tr>
<tr>
<td>Location of the SOO within GCV distal/proximal</td>
<td>1/20</td>
<td>6/7</td>
<td></td>
<td>0.001</td>
</tr>
<tr>
<td>Distance from coronary artery, mm</td>
<td>8.1±3.6</td>
<td>8.7±3.3</td>
<td>6.8±4.3</td>
<td>0.94</td>
</tr>
<tr>
<td>Coronary sinus diameter at SOO, mm</td>
<td>5.6±2.5</td>
<td>5.5±2.4</td>
<td>5.7±3.0</td>
<td>0.99</td>
</tr>
</tbody>
</table>

GCV indicates great cardiac vein.

### Angiography Results

The SOO was located a mean of 8.1±3.6 mm from the closest major coronary artery (the circumflex artery in 11 patients and the left anterior descending artery in 16 patients). One of 27 patients had an SOO within 4 mm of a major coronary artery. The mean coronary sinus diameter was 5.6±2.5 mm at the SOO of the epicardial arrhythmias (Table 2).

### Catheter Ablation

The epicardial PVCs were acutely abolished in 20 of 27 (74%) patients by radiofrequency ablation within the CVS. Activation mapping was used as the primary mapping technique in 26 of 27 patients. Three of 27 patients required isoproterenol for PVC induction and 1 patient had PVCs only during isoproterenol washout. One of 27 patients had infrequent PVCs, and pace mapping was used to identify the site of origin in this patient (Table 2).
At the ablation sites in the CVS, the mean activation time of the PVCs was $-29 \pm 8$ ms. There was no significant difference in activation time between the effective ablation sites ($-30 \pm 7$ ms) and the presumed SOOs in the patients with an unsuccessful outcome ($-29 \pm 9$ ms, $P=0.6$).

An open irrigated-tip catheter was used in all but 1 patient. However, we were unable to position the irrigated-tip catheter at the SOO in 8 patients. In 4 of 8 patients, the SOO was reached with a conventional nonirrigated-tip ablation catheter. Ablation was effective in 3 of 4 of these cases.

In the 7 patients in whom the ventricular arrhythmias were not successfully ablated, failure was due to inability to advance the ablation catheter to the SOO in the distal segment of the great cardiac vein in 4 patients; in these 4 patients, the SOO could be reached with the 2.5F multipolar catheter. In 1 patient each, the reason for failure was inadequate power delivery at the SOO, diaphragmatic capture during high-output pacing at the SOO, or proximity to a major coronary artery. In the patient in whom power delivery was inadequate, a conventional 4-mm-tip 7F catheter was used for delivery of radiofrequency energy. The maximal power that was delivered was $<15$ W. This site could not be reached with an irrigated-tip catheter. In the patient with a major epicardial coronary artery within 4 mm of the SOO, radiofrequency energy was not delivered at this site and cryoablation was not attempted.

Unsuccessful procedures were more frequent when the SOO was located in the distal segment than in the proximal segment of the great cardiac vein. Transeptal epicardial ablation was effective in 1 of 2 patients in whom it was attempted. In this patient, the effective ablation site was earlier in the epicardium than in the CVS. In the other patient in whom transeptal epicardial ablation was attempted, the ablation failed despite an activation time of $-30$ ms that was as early as in the CVS. Ablation lesions were delivered at both sites without eliminating the targeted PVCs. The remaining patients with failed ablations refused a transeptal epicardial approach.

There was no significant difference of the diameter of the CVS at the SOO in patients with acutely successful versus unsuccessful procedures. The distance between the coronary arteries and the SOO did not differ significantly between patients with acutely successful versus unsuccessful procedures. In patients with ineffective ablations within the coronary venous system due to the distal location of the SOO, the R wave in $V_1$ was narrower compared with arrhythmias that could be ablated from within the CVS ($76 \pm 7$ ms versus $113 \pm 40$ ms; $P=0.03$).

No complications occurred during the mapping or ablation procedure or during follow-up.

Ablation Data
An average of $6.2 \pm 5.8$ radiofrequency energy applications were delivered within the CVS, with a mean total radiofrequency energy duration of $6.2 \pm 7.6$ minutes. The mean procedure time was $296 \pm 56$ minutes and the mean fluoroscopy time was $57 \pm 15$ minutes. Cryoablation was effectively used for ablation within the great cardiac vein in 1 of the 27 patients because of high temperatures when using radiofrequency energy. A total period of 8 minutes of cooling to $-80^\circ$C was used in this patient. Coronary angiography showed no acute abnormalities in the coronary artery closest to the ablation site in any patient (Table 2).

Follow-Up
The mean PVC burden significantly decreased from $21 \pm 11\%$ at $2.3 \pm 1.4$ months before ablation to $6 \pm 9\%$ at $4.3 \pm 1.4$ months after ablation ($P=0.007$). The mean PVC burden at 3 months was $0.6 \pm 1\%$ in patients with a successful outcome, compared with $18 \pm 9\%$ in patients with an unsuccessful outcome ($P=0.004$). Two patients had recurrent PVCs within 2 weeks after an initially effective procedure. No recurrences occurred in the remaining patients during a median follow-up of $13 \pm 11$ months.

Discussion
Main Findings
Approximately 15% of patients with idiopathic PVCs were found to have an epicardial SOO identified within the CVS, most often in the great cardiac vein. Successful long-term ablation of these arrhythmias was achieved from within the CVS in approximately 70% of patients. ECG characteristics differentiated ventricular arrhythmias with an epicardial origin from ventricular arrhythmias from other sites of origin.

Great Cardiac Vein Anatomy
The great cardiac vein forms the base of the triangle of “Brocq and Mouchet,” along with the left anterior descending and left circumflex arteries (Figure 3 and 4), as it leaves the anterior interventricular groove and courses laterally to the atrioventricular junction. Not surprisingly, the SOO of epicardial arrhythmias was found to be close to either the circumflex artery or the left anterior descending coronary artery. However, in only 1 patient was the SOO considered to be too close (<4 mm) to a coronary artery to allow the safe application of radiofrequency energy. Patency of the coronary artery closest to the ablation sites was confirmed after ablation in all patients in whom radiofrequency energy was delivered within the CVS, establishing the acute safety of this approach. There are no long-term safety data available. Computed tomography of the coronary arteries during follow-up might help to answer this question.

Anatomic Constraints
The outcome of the ablation procedure was closely related to the location of the SOO within the CVS. In the 7 patients with an ineffective procedure, all but 1 had an SOO in the distal segment of the great cardiac vein, and in 4 of these 7 patients, the ablation catheter could not be maneuvered to the SOO that had been identified with the 2.5F diagnostic catheter.

In a study by El-Maasarany et al., 15% of patients were found to have a well-developed Vieussens valve after the great cardiac vein crossed the circumflex artery. There was a decrease in the diameter of the great cardiac vein at the site of this valve. It is conceivable that in 1 or more of our patients, the inability to advance the ablation catheter was due to a Vieussens valve. However, there was no evidence of a valve in the venograms of the great cardiac vein. Of note is that
only 1 patient had an acutely effective procedure in the distal segment of the CVS. However, this patient had recurrent PVCs 12 hours after the procedure.

Transcutaneous epicardial ablation was attempted in 2 of 7 patients with a failed ablation procedure from within the CVS. Ablation within the pericardial space was effective in 1 of these 2 patients.

The ability to deliver sufficient ablative energy at the SOO appeared to determine whether or not ablation was effective. Despite the anatomic constraints, ablation via the coronary venous system appears to be at least as effective as with a percutaneous transhauracic approach, which has a reported success rate of approximately 70%. Using the coronary venous system for mapping and ablation might result in a lower complication rate because damage to the right ventricle or extracardiac structures is less likely to occur.

**ECG Features**

Different criteria have been described for identifying an epicardial origin of a ventricular arrhythmia. These criteria include a pseudo delta wave or a delayed upstroke of the precordial QRS complex. Site-specific criteria also have been described.

The ECG morphology of epicardial idiopathic arrhythmias depends on their SOO along the great cardiac vein. In the proximal segment they display a right bundle-branch block morphology and in the distal segment they have a left bundle-branch block morphology. This may be explained by a location within the basal-lateral myocardium for the former and a more antero-basal location for the latter arrhythmias. If the SOO is in the proximal segment of the great cardiac vein, the initial vector is directed toward lead V1, accounting for the right bundle-branch block morphology. As the SOO moves toward the distal part of the great cardiac vein, closer to the anteroseptal myocardium, the initial vector is directed away from V1, resulting in a left bundle-branch block morphology.

Idiopathic arrhythmias originating in the epicardium most often had a left bundle-branch block morphology with an inferior axis. Right ventricular outflow tract arrhythmias and arrhythmias from the coronary cusp share this feature. An R-wave width >75 ms in V1 is useful for differentiating epicardial idiopathic arrhythmias from endocardial arrhythmias. The broader R wave is explained by a more posterior position relative to lead V1 compared with the right ventricular outflow tract. However, because of a more anterior position relative to lead V1, the epicardial arrhythmias that are located within the distal part of the great cardiac vein have a narrow R wave in V1, making them impossible to distinguish from right ventricular outflow tract arrhythmias.

**Clinical Implications**

Idiopathic ventricular arrhythmias often originate from the epicardium and can be reached from within the epicardial venous system. When idiopathic PVCs or VT with left bundle-branch block/inferior axis morphology have an R wave in V1 >75 ms and when the earliest endocardial activation time in the right ventricular outflow tract activation is <10 ms, an epicardial SOO should be suspected. A 2.5F diagnostic catheter within the great cardiac vein can identify whether the SOO can be reached via the coronary venous system.

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**Disclosures**

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**References**


**CLINICAL PERSPECTIVE**

We report on the prevalence of idiopathic epicardial ventricular arrhythmias and the results of radiofrequency catheter ablation from within the coronary venous system. In 27 of 189 patients (14%), an epicardial site of origin of the ventricular arrhythmia was identified from within the coronary venous system, either in the great cardiac vein (n = 1) or the middle cardiac vein (n = 1). Twenty of 27 patients (74%) underwent successful radiofrequency ablation within the coronary venous system. In 7 of the 27 patients, the ablation failed mainly for inability to reach the site of origin with the mapping catheter. Epicardial ventricular arrhythmias displayed a distinct ECG morphology compared with other sites of origin.
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