Surgical Ablation of Refractory Ventricular Tachycardia in Patients With Nonischemic Cardiomyopathy

Elad Anter, MD; Mathew D. Hutchinson, MD; Rajat Deo, MD; Haris M. Haqqani, MBBS; David J. Callans, MD; Edward P. Gerstenfeld, MD; Fermin C. Garcia, MD; Rupa Bala, MD; David Lin, MD; Michael P. Riley, MD; Harold I. Litt, MD; Joseph Y. Woo, MD; Michael A. Acker, MD; Wilson Y. Szeto, MD; Erica S. Zado, PA-C; Francis E. Marchlinski, MD; Sanjay Dixit, MD

Background—The surgical approach for the treatment of ventricular tachycardia (VT) has been largely replaced by percutaneous, catheter-based techniques. However, some VT circuits, particularly in patients with nonischemic cardiomyopathy, remain inaccessible to percutaneous ablation. Surgical therapy of these VTs is an alternative approach; however, its methodology has not been well defined. The purpose of this study was to evaluate the efficacy of preoperative electroanatomic and electrophysiological characterization of the VT substrate and circuit to guide surgical ablation.

Methods and Results—Eight patients with recurrent sustained VT refractory to antiarrhythmic drugs underwent endocardial and/or epicardial ablation procedures. Electroanatomic mapping was performed, and the VT substrate and circuit(s) were defined using voltage, activation, entrainment, and pace mapping. All 8 patients underwent detailed endocardial mapping; 6 patients also underwent epicardial mapping. Radiofrequency ablation was performed with the use of an open-irrigation catheter. After the unsuccessful percutaneous approach, surgical cryoablation was applied to the sites previously identified and targeted during the percutaneous procedure. There were no significant perioperative complications. During a mean follow-up period of 23±6 months (range, 15 to 34 months), 6 patients had significant reduction in VT burden as evident by a reduced number of implantable cardioverter-defibrillator shocks after ablation (6.6 to 0.6 shocks per patient; P=0.026). Two patients died, one of progressive heart failure and one of sepsis.

Conclusions—VT circuits inaccessible to percutaneous ablation techniques are rare but can be encountered in patients with nonischemic cardiomyopathy. These VTs can be successfully targeted by surgical cryoablation guided by preoperative electroanatomic and electrophysiological mapping. (Circ Arrhythm Electrophysiol. 2011;4:494-500.)

Key Words: ventricular tachycardia ■ catheter ablation ■ surgical ablation ■ cryoablation

Catheter-based radiofrequency ablation has become an important therapeutic option for treatment of ventricular tachycardias (VTs). Ablation therapy is considered an effective strategy for the treatment of idiopathic and scar-related VTs. However, in the latter category, and especially in patients with a nonischemic substrate, the success of radiofrequency ablation is not uniform. The likely reason for this discrepancy is the nature and/or distribution of scar. We have previously shown that in patients with nonischemic cardiomyopathy, the scar burden is often perivalvular and frequently epicardial. Although critical components of the VT circuit in these locations can still be successfully ablated with a conventional approach, in some patients a catheter-based approach is ineffective. In these cases, surgical ablation of the arrhythmia substrate may be the only alternative. However, the existing approach to surgical VT ablation is largely based on the experience reported more than 2 decades ago, when this procedure was performed exclusively in patients with healed myocardial infarction. Such an approach may not be applicable in the nonischemic patient population manifesting heterogeneous scar burden.

Editorial see p 429
Clinical Perspective on p 500

In the present study, we report our experience of surgical ablation, guided by detailed preoperative electroanatomic and electrophysiological characterization of the underlying substrate in patients with nonischemic cardiomyopathy who have drug-refractory VT and had failed percutaneous catheter radiofrequency ablation.

Methods

Study Population
Over a 3-year period (2007 to 2009), 527 patients underwent 644 VT ablation procedures at our center. Structural heart disease was...
Endocardial Mapping
A retrograde transaortic approach was used to access the left ventricle (LV) in all cases. A detailed electroanatomic map of the chamber of interest was created during sinus rhythm or right ventricular (RV) pacing. All mapping was performed with a 3.5-mm, open-irrigation–tip catheter (Navistar Thermocool, Biosense Webster, Diamond Bar, CA), maintaining a fill threshold of 15% to ensure adequate representation of the entire sampled surface area. The details of the contact electroanatomic mapping system (CARTO, Biosense Webster) have been described previously. Bipolar signals were also acquired on the Prucka (GE) recording system and analyzed separately.

Epicardial Mapping
Epicardial access was obtained with the technique originally described by Sosa et al. Briefly, under general anesthesia, a Tuohy needle was introduced through a subxiphoid approach to gain access to the pericardium. An 8F sheath was then advanced over a wire into the pericardial space. Mapping was performed, using the same methodology as described above.

Reference Values for Voltage Mapping and Areas of Voltage Abnormalities
The reference values for identifying low-amplitude endocardial bipolar electrograms were defined as per previously established criteria. A signal amplitude $>1.5$ mV was categorized as normal. The most abnormal signal amplitude ($<0.5$ mV) comprised “dense scar.” The reference values for defining abnormal electrograms in the epicardium have been established recently. For this study, normal epicardial electrograms were defined as signal amplitudes $\geq1.0$ mV, recorded at a distance $\geq1$ cm from large epicardial coronary arteries and/or valves. To adequately distinguish abnormal epicardial locations from fat mimicking scar, these areas were also required to demonstrate abnormal electrograms (ie, fractionated, split, and/or late potentials). The extent of abnormal epicardial and epicardial bipolar voltage signals was quantified by measuring contiguous areas of abnormal electrograms, using the “area calculation” tool available in the 3D mapping system.

Electrophysiological Study and VT Ablation
During the electrophysiology study, programmed electric stimulation was performed from the RV at 2 drive cycle lengths, using up to 3 extrastimuli. The ECGs of all spontaneous and induced VTs were analyzed on the Prucka recording system. When the VT was stable and tolerated, endocardial and/or epicardial activation and entrainment mapping were used to characterize the critical components of the circuit and/or the site of origin (if the underlying mechanism was thought to be nonreentrant). If the VT was not well tolerated or not reproducibly initiated, detailed characterization of the underlying substrate was performed by (1) marking areas manifesting late, split, or fractionated potentials and (2) pace mapping to mimic the VT morphology. The combination of abnormal electrograms during sinus rhythm, delayed stimulus to QRS during pace mapping, and a good (10/12 or better) QRS match of pace map were used as surrogates of the VT circuit. Radiofrequency ablation was performed, extending from the border zone to the dense scar, while transecting critical components of the VT circuit. When these regions were in close proximity to anatomic boundaries (mitral or aortic valve), the lesion sets were extended to incorporate these inert areas. Typical settings during lesion creation were power range of 20 to 50 W and maximum temperature of 45°C, for a total duration of 60 to 180 seconds to achieve an impedance drop of 10 to 18 Ohms.

Cardiac Imaging
Intracardiac echocardiography (Acuson AcuNav, 8F ultrasound catheter) was used in all ablation procedures. The transducer was placed in the RV, and abnormal echogenic area, representing scar in the endocardium and/or subepicardium, was identified. Cardiac MRI was performed before the ablation procedure in 4 of the 8 patients. We have previously described the feasibility and safety of MRI in patients with defibrillators. In brief, informed consent was obtained and cardiac MRI was performed at 1.5 T. The defibrillator was interrogated before the study, and tachycardia detection and therapy were disabled. Pacing was programmed to VVI mode at 40 bpm in these nondependent patients. Cardiac MRI sequences were adapted to minimize energy deposition and image artifact. After cardiac MRI, defibrillators were reinterrogated and returned to previous settings. No clinically relevant changes were seen in any parameters in these 4 patients. Standard criteria in the form of delayed enhancement using a phase-sensitive inversion recovery sequence after gadolinium-DTPA administration were used for identifying ventricular scar.

Surgical VT Ablation Technique
Surgery was performed by means of a median sternotomy with cardiopulmonary bypass. Cryothermy was applied under cold cardioplegia. The relevant VT surfaces (endocardial, epicardial, or both) were carefully inspected, and correlation to the preacquired electroanatomic map was performed. Endocardial exposure was performed through a transaortic valve approach. Moreover, in all patients, the previously placed radiofrequency ablation lesions were identified. No additional mapping was performed during the surgical procedure. Cryothermy to the endocardial and/or epicardial locations previously identified was applied using the Surgifrost Surgical Cryoablation System (Medtronic CryoCath LP, Quebec, Canada). This consists of
a flexible metal probe with an adjustable insulation sheath that can be molded to conform to the cardiac contours. The system uses Argon gas to achieve rapid cooling to a temperature of $-150^\circ$C. During a 3-minute application time, this creates an ablation lesion as deep as 60 mm (Figure 1).

**Follow-Up**

After completion of the surgical ablation, patients recovered in the hospital as per standard open-heart surgery protocol. They were subsequently followed through regular clinic visits (first month and every 3 months thereafter), during which arrhythmia recurrences were assessed by patient interview, 12-lead ECG, and device interrogation. In the event of no arrhythmia recurrence, modification/discontinuation of the antiarrhythmic regimen was left to the discretion of the treating electrophysiologist.

**Statistical Analysis**

Continuous variables are expressed as group mean±1 standard deviation. Comparisons of continuous variables between groups were analyzed with the Wilcoxon signed rank test. Categorical variables expressed as proportions in different groups were compared by the $\chi^2$ test. A probability value of <0.05 was considered statistically significant.

**Results**

**Patient Population**

Baseline characteristics of the 8 patients are listed in Table 1. There were 7 men and 1 woman, with a mean age of 58±11 years. The median LV ejection fraction was 35%, and all patients had nonischemic cardiomyopathy (6 patients with dilated cardiomyopathy and 2 patients with hypertrophic cardiomyopathy). Each patient included in this series had failed medical therapy with at least 1 antiarrhythmic drug (AAD) and had multiple appropriate implantable cardioverter-defibrillator (ICD) shocks (median, 6; range, 3 to 16) in the preceding 3 months before surgical ablation. Patients underwent a median of 1.5 (range, 1 to 3) endocardial and 1.0 (range, 0 to 2) epicardial radiofrequency ablation procedures before the surgical ablation.

**Substrate Characterization**

The details of electroanatomic mapping are presented in Table 2. The LV endocardium was mapped in all 8 patients (average of 398±228 sites per subject). Additionally, in 4 patients manifesting a clinical VT morphology consistent with RV origin (left bundle-branch block pattern), electroanatomic mapping of the RV was also performed. Endocardial regions of dense scar occupied small areas, ranging from 0% to 12% of the entire LV endocardium (mean, 3.7±2.7%). Consistent with our prior observations, these low-voltage zones were predominantly in the perimetal annulus distribution. Two patients with hypertrophic cardiomyopathy (patients 3 and 8) also demonstrated scar along the base of the heart.

### Table 1. Baseline Patient Characteristics

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, y</th>
<th>Sex</th>
<th>LVEF, %</th>
<th>NICM</th>
<th>ICD</th>
<th>No. Failed AADs</th>
<th>No. ICD Shocks in Preceding 3 Months</th>
<th>No. of Prior Endocardial Procedures</th>
<th>No. of Prior Epicardial Procedures</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>65</td>
<td>M</td>
<td>15</td>
<td>Yes</td>
<td>Yes</td>
<td>3</td>
<td>8</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>50</td>
<td>M</td>
<td>70</td>
<td>Yes</td>
<td>Yes</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>54</td>
<td>M</td>
<td>58</td>
<td>Yes</td>
<td>Yes</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>4</td>
<td>51</td>
<td>F</td>
<td>25</td>
<td>Yes</td>
<td>No*</td>
<td>1</td>
<td>1</td>
<td>External shock</td>
<td>1</td>
</tr>
<tr>
<td>5</td>
<td>51</td>
<td>M</td>
<td>30</td>
<td>Yes</td>
<td>Yes</td>
<td>2</td>
<td>12</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>6</td>
<td>74</td>
<td>M</td>
<td>30</td>
<td>Yes</td>
<td>Yes</td>
<td>3</td>
<td>16</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>7</td>
<td>74</td>
<td>M</td>
<td>50</td>
<td>Yes</td>
<td>Yes</td>
<td>2</td>
<td>6</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>8</td>
<td>48</td>
<td>M</td>
<td>40</td>
<td>Yes</td>
<td>Yes</td>
<td>2</td>
<td>4</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

LVEF indicates left ventricular ejection fraction; NICM, nonischemic cardiomyopathy; ICD, implantable cardioverter-defibrillator; and AAD, antiarrhythmic drugs.

*ICD was implanted after ablation procedure.

### Table 2. Endocardial and Epicardial Substrate Characteristics

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Sinus Rhythm LV Points</th>
<th>LVZ, cm²</th>
<th>% Dense Scar</th>
<th>LP in Dense Scar, %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Endocardial</td>
<td>Epicardial</td>
<td>Endocardial</td>
<td>Epicardial</td>
</tr>
<tr>
<td>1</td>
<td>299</td>
<td>N/A</td>
<td>30.0</td>
<td>N/A</td>
</tr>
<tr>
<td>2</td>
<td>277</td>
<td>542</td>
<td>19.2</td>
<td>96</td>
</tr>
<tr>
<td>3</td>
<td>264</td>
<td>219</td>
<td>3.0</td>
<td>12</td>
</tr>
<tr>
<td>4</td>
<td>217</td>
<td>377</td>
<td>5</td>
<td>32</td>
</tr>
<tr>
<td>5</td>
<td>492</td>
<td>604</td>
<td>28.9</td>
<td>28.9</td>
</tr>
<tr>
<td>6</td>
<td>622</td>
<td>826</td>
<td>34.0</td>
<td>65</td>
</tr>
<tr>
<td>7</td>
<td>826</td>
<td>N/A</td>
<td>0</td>
<td>N/A</td>
</tr>
<tr>
<td>8</td>
<td>186</td>
<td>379</td>
<td>3.6</td>
<td>88</td>
</tr>
</tbody>
</table>

LV indicates left ventricle; LVZ, low-voltage zone defined as bipolar signal amplitude $\leq$1.5 mV in endocardium and $\leq$1.0 mV in the epicardium; dense scar was defined as a bipolar signal amplitude $\leq$0.5 mV in both the endocardium and epicardium; and LP, late potentials.
septum. Epicardial mapping was performed in 6 patients. A mean of 491/213 sites (range, 219 to 826 per patient) were sampled to create the RV/LV epicardial voltage map. In 2 patients, detailed epicardial mapping was not performed: One patient had undergone multiple prior valve replacement procedures, and so we anticipated a lack of potential pericardial space; in the second patient, difficult pericardial access resulted in RV perforation requiring emergent surgery.

**Electrophysiological Characterization**

A total of 24 spontaneous or induced VTs were observed (median, 2 per patient; range, 1 to 8), with a mean cycle length of 334±186 ms (Table 3). Four patients had stable and well-tolerated VTs that allowed characterization of the circuit. In the other 4 patients, the VT was not hemodynamically tolerated, and the substrate was characterized by identifying surrogates of the potential VT circuits. The clinical VT (defined as either identical to a 12-lead ECG obtained during spontaneous arrhythmia or matching electrogram morphology and rate, as recorded by the ICD during spontaneous VT) was induced and characterized in all patients. In 5 patients, at least 1 component of the VT circuit was mapped and targeted from the endocardium. Additional endocardial lesions were performed with intention to eliminate all induced VTs. A mean of 51±49 minutes of radiofrequency ablation time was applied during the endocardial ablation procedure. In the 6 patients undergoing epicardial VT mapping, the abnormal substrate was usually opposite to the endocardial ablation lesions but was separated by thick myocardium that ranged from 15 to 32 mm (as measured by intracardiac ultrasound and/or MRI). Moreover, all 4 patients who underwent cardiac MRI exhibited a midmyocardial scar (Figure 2). The midmyocardial scar was identified in the septum in 3 cases and in the inferior wall in 1 case. These MRI findings of midmyocardial scar correlated with lack of endocardial or epicardial scar at these locations on the bipolar voltage map. However, in 1 patient with basal-septal midmyocardial scar, scar was identified on the epicardial surface.

At the conclusion of the last percutaneous radiofrequency ablation procedure, 4 patients had no inducible VT, 3 patients were inducible for the clinical VT, and in 1 patient inducibility could not be assessed because of urgent surgical interven-

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>No. of Induced VT</th>
<th>No. of VTs Targeted</th>
<th>No. of RF Lesions</th>
<th>VT Mapping/Target Identification</th>
<th>Inducible at End of Most Recent Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Endocardial</td>
<td>Epicardial</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>1</td>
<td>1/1</td>
<td>N/A</td>
<td>28</td>
<td>N/A AM/EM/PM/LP</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>0/2</td>
<td>2/2</td>
<td>0</td>
<td>15 PM/LP</td>
</tr>
<tr>
<td>3</td>
<td>2</td>
<td>1/2</td>
<td>2/2</td>
<td>41</td>
<td>37 AM/EM/PM/LP</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
<td>2/4</td>
<td>1/4</td>
<td>12</td>
<td>18 AM/EM/PM/LP</td>
</tr>
<tr>
<td>5</td>
<td>2</td>
<td>1/2</td>
<td>1/2</td>
<td>21</td>
<td>52 EM/PM/LP</td>
</tr>
<tr>
<td>6</td>
<td>8</td>
<td>8/8</td>
<td>8/8</td>
<td>182</td>
<td>53 PM/LP</td>
</tr>
<tr>
<td>7</td>
<td>1</td>
<td>0/1</td>
<td>0</td>
<td>0</td>
<td>0 PM/LP</td>
</tr>
<tr>
<td>8</td>
<td>4</td>
<td>4/4</td>
<td>4/4</td>
<td>114</td>
<td>0 PM/LP</td>
</tr>
</tbody>
</table>

VT indicates ventricular tachycardia; RF, radiofrequency; AM, activation mapping; EM, entrainment mapping; LP, late potentials; and PM, pace mapping.

**Surgical Ablation**

The median interval between the last percutaneous radiofrequency ablation procedure and surgical cryoablation was 2±4 weeks (range, 0 to 12 weeks). Cryothermy was applied at the previously identified critical VT circuit locations. In 5 patients, cryothermy was applied from both the endocardium and epicardium, and in 3 patients from the epicardium alone. The details of the surgical ablation procedure are shown in Table 4. Importantly, in 2 patients (subjects 3 and 8), the clinical VT originated from the thickened (>20 mm) basal septum where MRI demonstrated midmyocardial scar (Figure 2). In this location, endocardial and epicardial cryoablation lesions were applied adjacent to the base of the LV septum near the aortic valve, taking care to spare the left anterior descending coronary artery. In 2 patients (subjects 4 and 5), the clinical VT was mapped to the epicardial surface and was

![Figure 2. Cardiac magnetic resonance (CMR) image shows a 4-chamber view of the heart obtained using a phase-sensitive inversion recovery sequence approximately 15 minutes after gadolinium-DTPA administration, which demonstrates a mid-myocardial late enhancement in the basal septum (arrow). RV indicates right ventricle; LV, left ventricle.](image-url)
in close proximity (<1 cm) to the left anterior descending and circumflex arteries. During surgical exploration, these locations were targeted with cryoablation after ensuring a minimum separation of ≥0.5 cm from the coronary vessels. In 1 patient, surgical ablation was performed after complication of the percutaneous ablation procedure (perforation of the RV). This patient had a normal endocardial voltage map, and the circuit of the clinical VT did not appear to be endocardial. When the heart was exposed, epicardial scar was evident on the anterolateral LV surface, consistent with the VT morphology. This location was targeted by cryotherapy. Overall, a median of 5 cryothermal applications (range, 7±4) were made per subject, with a mean cryoablation time of 18±6 minutes per patient. Of note, in 2 patients (subjects 1 and 4), surgical cryoablation was combined with valve repair (Table 4). For patients who only underwent a surgical cryoablation procedure, the mean surgical procedure duration was 112±28 minutes (range, 82 to 164 minutes).

### Short- and Long-Term Outcomes

The follow-up data are presented in Table 5. Two patients died during the index hospitalization (at 6 and 10 weeks after the surgical ablation): one from progressive heart failure and second from sepsis. In the remaining 6 patients, the median time from surgery to discharge was 7 days (range, 5 to 11 days). Noninvasive programmed stimulation via the RV defibrillator lead was performed in 4 patients before discharge. VT was induced only in 1 patient. Patients were discharged on the preablation AAD regimen: 4 patients were discharged on a single AAD and 1 patient on 2 AADs. One patient was discharged of AAD therapy. Over a mean follow-up of 23±6 months (range, 15 to 34), 4 patients remained free of VT, 1 patient had a single VT episode resulting in 1 ICD shock, and 1 patient had 3 VT episodes, all occurring during the first 3 postoperative months, but none over the remainder 12 months of follow-up (without modification of medical therapy). Overall, there was a significant reduction in burden of VT and ICD therapies. The number of ICD shocks per patient declined from 6.6 shocks in the preceding 3 months before surgery to 0.6 during the first 3 postsurgical months (P=0.026).

### Discussion

We report our surgical ablation experience in patients with recurrent symptomatic VT that is refractory to medical therapy and percutaneous ablation procedures. The surgical ablation procedure was guided by detailed electrophysiological and electroanatomic mapping obtained a priori during the percutaneous ablation procedure. The salient findings of this study include (1) VTs refractory to percutaneous ablation were more common in patients with a nonischemic substrate, (2) the underlying substrate frequently involved a midmyocardial or epicardial scar in close proximity to a coronary vessel, and (3) detailed a priori electroanatomic and electrophysiological characterization of the arrhythmia/substrate was useful to identify the surgical targets of ablation.

It should be emphasized that this series represents one end of the spectrum: patients with frequent symptomatic VTs that are resistant to AAD therapy and percutaneous ablation techniques. Surgical ablation for these patients was the last resort. In these cases, electroanatomic and electrophysiologically guided surgical cryoablation may be useful. Despite the excellent outcome in 6 patients (75%), 2 patients died during the follow-up period. These deaths were not directly related to the ablation procedures, because both patients had a normal uncomplicated recovery from their surgical procedure. It is possible, however, that these procedures, along with their associated hospital stay, may indirectly increase the risk for complications.

The initial experience with surgical VT ablation was described in patients with healed myocardial infarcts and primarily involved endocardial resection of the visible scar. However, this led only to a modest long-term arrhythmia control. Subsequent work by Miller et al\(^6\) showed that the outcome of surgical VT ablation in the setting of healed infarction could be improved by detailed preoperative and intraoperative mapping. Seminal work by Josephson et al\(^11\–14\) resulted in the development of subendocardial resection, which further enhanced the procedural efficacy of surgical VT ablation in this population, with long-term arrhythmia control rates of ≈80%. With improvements in catheter-based

---

### Table 5. Follow-Up Data

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Time From Surgery to Discharge, Days</th>
<th>NIPS Before Discharge</th>
<th>No. of AADs at Discharge</th>
<th>No. of ICD Shocks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>11</td>
<td>Not performed</td>
<td>2 (quinidine, mexiletine)</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>5</td>
<td>Noninducible</td>
<td>1 (sotalol)</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>8</td>
<td>Not performed</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>Died</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>5</td>
<td>7</td>
<td>Noninducible</td>
<td>1 (amiodarone)</td>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>Died</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>7</td>
<td>6</td>
<td>Noninducible</td>
<td>1 (sotalol)</td>
<td>0</td>
</tr>
<tr>
<td>8</td>
<td>7</td>
<td>MMVT inducible</td>
<td>1 (mexiletine)</td>
<td>1</td>
</tr>
</tbody>
</table>

NIPS indicates noninvasive programmed stimulation; AADs, antiarrhythmic drugs; and ICD, implantable cardioverter-defibrillator.
ablation technology, success rates for percutaneous VT ablation have improved, and VT arrhythmia surgery has become uncommon. However, in some patients, particularly those with a nonischemic substrate, catheter ablation may still fail, and a surgical approach remains the only alternative. Despite this, studies on the surgical ablation experience in this patient population are lacking.

As our experience shows, patients with nonischemic cardiomyopathy may have midmyocardial and/or basal LV epicardial scars that are in close proximity to major coronary arteries. Thus, radiofrequency energy may not be effective or safe in these locations. For this subgroup of patients, surgical VT ablation can be beneficial. However, in our opinion, to accomplish successful surgical cryoablation the following are important: (1) detailed characterization of the underlying arrhythmia mechanism/Substrate, using percutaneous electrophysiological/electroanatomic mapping, (2) cardiac imaging (intracardiac ultrasound/MRI), (3) full sternotomy for complete visualization of various cardiac surfaces, and (4) cardiopulmonary bypass with cold cardioplegia. Additionally, precise surgical ablation is also facilitated by identifying and targeting locations manifesting prior radiofrequency lesions. In our experience, these sites are easily recognized in the endocardium but may not be as evident in the epicardium. In the latter location, they appear as small punctate spots (Figure 3B) and may be indistinguishable from epicardial fat or scar. This appearance of epicardial lesions may be a reflection of the inadequacy of catheter-based radiofrequency energy to create effective epicardial lesions by means of the percutaneous technique.

Limitations

The major study limitations are as follows: (1) The study population is heterogeneous, consisting of patients with dilated as well as hypertrophic cardiomyopathy. Thus, the arrhythmia mechanism(s)/substrate may not be uniform. (2) Although we performed detailed electrophysiological and electroanatomic characterization of the substrate endocardially in all patients, in 2 patients the epicardium was not well characterized. Nevertheless, in these 2 subjects, surgical ablation was still guided by the information collected during detailed endocardial mapping. (3) Because intraoperative mapping was not performed, we cannot comment on its utility in our study population; however, a hybrid surgical approach with mapping during surgery is also likely to be useful. (4) Programmed stimulation was not performed routinely in all patients before discharge, and therefore assessment of acute success is based on clinical outcome. (5) Patients included in this series had incessant VT resistant to AAD therapy and percutaneous ablation attempts, and therefore outcome cannot be extrapolated to patients in whom less aggressive measures were not fully utilized.

Conclusion

VT refractory to percutaneous catheter ablation is rare. However, when encountered, it is primarily seen in patients with a nonischemic substrate. Such VTs can be successfully targeted by surgical cryoablation guided by prior detailed percutaneous electroanatomic and electrophysiological mapping.

Disclosures

Drs Dixit and Callans received speaking honoraria from Biosense-Webster. Dr Marchlinski received speaking honoraria and research grants from Biosense-Webster. Drs Hutchinson and Gerstenfeld received research grants from Biosense-Webster.

References


**CLINICAL PERSPECTIVE**

Catheter ablation is an important option to control recurrent ventricular tachycardia (VT). Success rates continue to improve as our understanding of VT physiology is growing and better ablation tools are introduced. However, some VT circuits are still inaccessible to percutaneous ablation. In the majority of these cases, the VT circuit involves a midmyocardial scar, which precludes effective energy delivery or is in close proximity to an epicardial coronary vessel, limiting the ability to intervene. In these patients, surgical ablation is an alternative approach. Our study shows that surgical ablation guided by preoperative electroanatomic and electrophysiological mapping can be effective. Therefore, when these VTs are encountered, surgical ablation should be considered.

---


Surgical Ablation of Refractory Ventricular Tachycardia in Patients With Nonischemic Cardiomyopathy


_Circ Arrhythm Electrophysiol._ 2011;4:494-500; originally published online June 14, 2011; doi: 10.1161/CIRCEP.111.962555

_Circulation: Arrhythmia and Electrophysiology_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2011 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-3149. Online ISSN: 1941-3084

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circep.ahajournals.org/content/4/4/494

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation: Arrhythmia and Electrophysiology_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation: Arrhythmia and Electrophysiology_ is online at:
http://circep.ahajournals.org//subscriptions/