Long-Term Outcomes of Combined Epicardial and Endocardial Ablation of Monomorphic Ventricular Tachycardia Related to Hypertrophic Cardiomyopathy

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Background—Monomorphic ventricular tachycardia (MMVT) is rare in patients with hypertrophic cardiomyopathy (HCM). There are limited data on the utility of catheter ablation for the treatment of MMVT in this population. This study details a series of case reports from multiple centers where combined epicardial-endocardial ablation was performed in a highly selected group of patients with HCM-related MMVT.

Methods and Results—The cohort consisted of 10 patients with HCM-related MMVT. Pericardial access was achieved using the percutaneous subxyphoid approach. Epicardial and endocardial ventricular 3D bipolar voltage maps were generated. Ablation sites were identified using a combination of entrainment, activation, late/fractionated potential, and pace mapping. Electrophysiological-identified epicardial scar was present in 8 (80%) patients, endocardial scar in 6 (60%), and no scar in 1 (10%). In the 5 patients with inducible, stable MMVT, 3 cases were successfully terminated with ablation from the epicardium and 1 from the endocardium. The case that failed catheter ablation required surgical cryoablation to abolish the incessant VT. In the remaining 5 patients, 4 underwent epicardial and endocardial ablation of sites with good pace maps and late/fractionated potentials. No ablation was performed in the remaining patient because of noninducibility and lack of identifiable scar. After 37±17 months (limits, 2 to 62 months; median, 37 months), the freedom from recurrent implantable cardioverter-defibrillator shocks was 78% (7/9 patients) in those who underwent ablation.

Conclusions—In highly selected patients with HCM, combined epicardial and endocardial mapping and ablation is a feasible and reasonably efficacious option for MMVT if refractory to aggressive trials of antiarrhythmic drugs and antitachycardia pacing. (Circ Arrhythm Electrophysiol. 2011;4:185-194.)

Key Words: ablation ■ arrhythmia ■ cardiomyopathy hypertrophic ■ epicardial mapping

In patients with hypertrophic cardiomyopathy (HCM), implantable cardioverter-defibrillators (ICDs) are the mainstay of therapy for prophylaxis against sudden cardiac death.1,2 Although the occurrence of monomorphic ventricular tachycardia (VT) is rare in these patients, the mechanism of sudden death is more commonly ventricular fibrillation and polymorphic VT.3 In instances where monomorphic VT is recurrent and refractory to antiarrhythmic drugs, catheter ablation may be useful.

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Catheter ablation has been used successfully in patients with monomorphic VT that occurs in the setting of multiple other substrates, but there are only a few anecdotal case reports about the role of catheter ablation in patients with HCM.3-6 Furthermore, although myocardial scar has been implicated in the genesis of monomorphic VT in other disease states, few data characterize the arrhythmogenic substrate in patients with HCM. However, MRI data demonstrate the presence of myocardial scar as identified by delayed enhancement imaging in patients with HCM with manifest monomorphic MMVT,7-10 suggesting that the substrate for monomorphic VT may be similar to that seen in patients postmyocardial infarction and, thus, may be amenable to catheter ablation. Additionally, because the ventricular wall in patients with HCM can be quite thick, traditional endocar-
dial mapping and ablation alone may be of limited value.\textsuperscript{10,11} In this article, we discuss in detail a multicenter experience with a combined epicardial and endocardial approach to catheter ablation in a highly selected group of patients with HCM-related monomorphic VT.

**Methods**

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agreed to the manuscript as written. The data were collected according to the policies of each center’s institutional review board.

**Patient Population**

The patient population consisted of a highly selected group of 10 patients with HCM and documented monomorphic VT in whom a combined epicardial and endocardial approach to catheter ablation was attempted. This cohort represents a collection of patients from 4 medical centers in whom this approach was attempted between December 2003 and December 2009. Patient selection criteria for this combined approach were not uniform. Most patients had antiarrhythmic drug refractory monomorphic VT with multiple ICD shocks and were believed to be appropriate candidates for catheter ablation; in cases where this was not true, the patients had hemodynamically stable monomorphic VT that was believed to be amenable to catheter ablation. Antiarrhythmic drug and antitachycardia pacing use was variable and largely depended on physician bias. The diagnosis of HCM was based on the echocardiographic documentation of left ventricular (LV) hypertrophy without chamber dilatation in the absence of any other cardiac or systemic disease that could account for such findings.

**Endocardial and Epicardial Mapping and Ablation**

Procedures were performed with either general anesthesia (7 patients) or moderate sedation (3 patients). During the procedures, intra-aortic balloon pump counterpulsation was used in 3 patients for prophylaxis against heart failure and was removed at the conclusion of the procedures.

**Table 1. Clinical Characteristics of Patients With HCM Undergoing Catheter Ablation for VT**

<table>
<thead>
<tr>
<th>Pt. No.</th>
<th>Age, y</th>
<th>Sex</th>
<th>CAD</th>
<th>HTN</th>
<th>DM</th>
<th>NYHA FC</th>
<th>ICD</th>
<th>AADs</th>
<th>History</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>69</td>
<td>Male</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>IB</td>
<td>No</td>
<td>…</td>
<td>Palpitations, MMVT, DCCV</td>
</tr>
<tr>
<td>2</td>
<td>57</td>
<td>Male</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>IB</td>
<td>No</td>
<td>…</td>
<td>Palpitations, MMVT, DCCV</td>
</tr>
<tr>
<td>3</td>
<td>53</td>
<td>Male</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>IB</td>
<td>Yes</td>
<td>A, M</td>
<td>Recurrent MMVT, ICD shocks</td>
</tr>
<tr>
<td>4</td>
<td>48</td>
<td>Male</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>IB</td>
<td>Yes</td>
<td>S</td>
<td>Recurrent MMVT, ICD storm</td>
</tr>
<tr>
<td>5</td>
<td>73</td>
<td>Male</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>II</td>
<td>Yes</td>
<td>A</td>
<td>Recurrent MMVT, ICD shocks</td>
</tr>
<tr>
<td>6</td>
<td>50</td>
<td>Male</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>IB</td>
<td>Yes</td>
<td>S</td>
<td>Recurrent MMVT, ICD storm</td>
</tr>
<tr>
<td>7</td>
<td>49</td>
<td>Male</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>II</td>
<td>Yes</td>
<td>M</td>
<td>Recurrent MMVT, ICD shocks</td>
</tr>
<tr>
<td>8</td>
<td>56</td>
<td>Male</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>II</td>
<td>Yes</td>
<td>M, S</td>
<td>Recurrent MMVT, ICD storm</td>
</tr>
<tr>
<td>9</td>
<td>66</td>
<td>Male</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>II</td>
<td>Yes</td>
<td>A, M</td>
<td>Recurrent MMVT, ICD storm</td>
</tr>
<tr>
<td>10</td>
<td>49</td>
<td>Male</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>I</td>
<td>Yes</td>
<td>A</td>
<td>Recurrent MMVT, ICD shocks</td>
</tr>
</tbody>
</table>

A indicates amiodarone; AADs, antiarrhythmic drugs; CAD, coronary artery disease; DCCV, direct current cardioversion; DM, diabetes mellitus; HTN, hypertension; ICD, implantable cardioverter-defibrillator; LVEF, LV ejection fraction; M, mexiletine; MMVT, monomorphic VT; NYHA FC, New York Heart Association functional class; PMVT, polymorphic VT; S, sotalol.

**Table 2. Two-Dimensional Echocardiogram Findings of Patients With HCM-Related VT**

<table>
<thead>
<tr>
<th>Pt. No.</th>
<th>LVEF, %</th>
<th>LVEDD, mm</th>
<th>LVESD, mm</th>
<th>IVS, mm</th>
<th>PW, mm</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>55</td>
<td>52</td>
<td>31</td>
<td>11</td>
<td>11</td>
<td>Focal midventricular and apical hypertrophy; midcavitary obstruction with gradient $= 25$ mm Hg; apical aneurysm</td>
</tr>
<tr>
<td>2</td>
<td>68</td>
<td>57</td>
<td>37</td>
<td>8</td>
<td>12</td>
<td>Apical hypertrophy; midcavitary systolic gradient $= 11$ mm Hg</td>
</tr>
<tr>
<td>3</td>
<td>57</td>
<td>47</td>
<td>27</td>
<td>16</td>
<td>12</td>
<td>Asymmetric hypertrophy; midseptal $= 26$ mm; mid-PW $= 25$ mm; midcavitary obliteration with gradient of $45$ mm Hg after Valsalva maneuver; apex akinetic/aneurysmal</td>
</tr>
<tr>
<td>4</td>
<td>63</td>
<td>51</td>
<td>31</td>
<td>14</td>
<td>11</td>
<td>Asymmetric LV hypertrophy without gradient; no wall motion abnormalities</td>
</tr>
<tr>
<td>5</td>
<td>40</td>
<td>55</td>
<td>41</td>
<td>15</td>
<td>17</td>
<td>Asymmetric LV hypertrophy; lateral wall hypokinesis</td>
</tr>
<tr>
<td>6</td>
<td>83</td>
<td>42</td>
<td>22</td>
<td>24</td>
<td>12</td>
<td>Asymmetric septal hypertrophy; midcavitary obliteration with gradient $= 23$ mm Hg</td>
</tr>
<tr>
<td>7</td>
<td>48</td>
<td>48</td>
<td>33</td>
<td>12</td>
<td>12</td>
<td>Apical akinesia/aneurysmal dilatation; organized apical thrombus; midcavitary obliteration</td>
</tr>
<tr>
<td>8</td>
<td>43</td>
<td>56</td>
<td>43</td>
<td>16</td>
<td>8</td>
<td>Asymmetric septal hypertrophy and LV dilatation; inferior wall and inferior/midseptal hypokinesis and akinosis from LV base to apex</td>
</tr>
<tr>
<td>9</td>
<td>45</td>
<td>45</td>
<td>33</td>
<td>26</td>
<td>15</td>
<td>Asymmetric septal hypertrophy without obstruction; apical and anteroseptal hypokinesis</td>
</tr>
<tr>
<td>10</td>
<td>65</td>
<td>31</td>
<td>20</td>
<td>24</td>
<td>17</td>
<td>Severe asymmetric septal hypertrophy; no LV outflow tract obstruction</td>
</tr>
</tbody>
</table>

IVS indicates interventricular septum; LVEDD, LV end-diastolic diameter; LVEF, LV ejection fraction; LVESD, LV end-systolic diameter; PW, posterior wall; RWMA, regional wall motion abnormalities.
Pericardial access was achieved using the percutaneous subxyphoid approach. LV endocardial mapping was performed using either a retrograde aortic approach or a transseptal approach. Transseptal puncture was performed using a combination of fluoroscopy and intracardiac echocardiography guidance. An 8.5-F Mullins sheath was advanced into the left atrium, with the sheath tip placed near the mitral valve plane. Intravenous heparin was given to maintain an activated clotting time ≥250 seconds. When necessary, the right ventricle (RV) was mapped using a femoral venous approach.

Multiple catheters were used for mapping and ablation, including (1) a 4-mm catheter (Navistar; Biosense Webster Inc) in 5 patients, (2) a 4-mm internally irrigated catheter (Chilli II; Boston Scientific Corp) in 1 patient who also was mapped with the Navistar catheter, (3) a 4-mm catheter with embedded magnets (Navistar RMT; Biosense Webster) in 1 patient, or (4) a 3.5-mm externally irrigated catheter (ThermoCool; Biosense Webster) in the remaining 4 patients. Programmed stimulation included up to 3 extrastimuli and rapid pacing from up to 2 RV sites to document cycle lengths and 12-lead electrocardiographic morphologies of all inducible VTs. In 9 patients, baseline 3D electroanatomical substrate maps were created using CARTO (Biosense Webster), and 1 of the mapping catheters as previously described (Navistar or ThermoCool). In 1 patient, a remotely controlled magnetic navigation system (Niobe Stereotaxis; Stereotaxis, Inc) was used with a compatible electroanatomical mapping system (CARTO RMT) and a Navistar RMT catheter to generate the baseline maps. Baseline 3D electroanatomic mapping consisted of constructing 3D bipolar voltage maps of the chambers of interest (LV, ventricular epicardial surface, and RV) during sinus rhythm or RV pacing, displaying a peak-to-peak bipolar electrogram amplitude with a fill threshold of at least 15 and 20 mm for endocardial and epicardial maps, respectively. As previously described in animal models, a bipolar electrogram voltage amplitude ≥1.5 mV was defined as normal myocardium and a voltage <1.5 mV as electrophysiological scar. Following detailed mapping to fully define the scar borders, a combination of entrainment, activation, late/fractionated potential, and pace mapping was used to identify appropriate targets for ablation. When hemodynamically stable VT was induced, standard entrainment maneuvers were used to identify and ablate the critical pathway of the VT circuit. Additionally, in a few selected patients with sustained hemodynamically stable VT, full or partial chamber activation maps were generated. In most patients, ablation of remaining putative target sites, such as late/fractionated potentials, pace map sites with a good

### Table 3. Results of VT Induction, Mapping, and Ablation

<table>
<thead>
<tr>
<th>Pt. No.</th>
<th>Arrhythmia Induced</th>
<th>No. MMVTs</th>
<th>VT Cycle Length, ms</th>
<th>Scar Location</th>
<th>Entrainment</th>
<th>Activation</th>
<th>Mapping</th>
<th>Termination During Ablation</th>
<th>Termination Site</th>
<th>Termination Time, s</th>
<th>Ablation Sites</th>
<th>Irrigated Catheter</th>
<th>Postablation VT Induced</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>...</td>
<td>0</td>
<td>...</td>
<td>Endo (LV apex)</td>
<td>No</td>
<td>No</td>
<td>...</td>
<td>...</td>
<td>Endo</td>
<td>No</td>
<td>No</td>
<td>Epi</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>PMVT</td>
<td>0</td>
<td>...</td>
<td>Endo (LV apex)</td>
<td>No</td>
<td>No</td>
<td>...</td>
<td>...</td>
<td>Endo</td>
<td>No</td>
<td>No</td>
<td>Epi</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>MMVT</td>
<td>1</td>
<td>472</td>
<td>Endo (LV apex)</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Epi</td>
<td>22</td>
<td>Endo</td>
<td>Yes</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>MMVT</td>
<td>1</td>
<td>270</td>
<td>Epi (LV lateral base)</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Epi</td>
<td>3</td>
<td>Epi</td>
<td>No</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>MMVT</td>
<td>2</td>
<td>VT1-370, VT2-280 (RVOT)</td>
<td>Epi (LV anterolateral base)</td>
<td>No</td>
<td>Yes</td>
<td>Yes-VT1</td>
<td>Epi</td>
<td>30</td>
<td>Epi</td>
<td>No</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>...</td>
<td>0</td>
<td>...</td>
<td>None</td>
<td>...</td>
<td>Yes-VT1</td>
<td>Endo-reinducible</td>
<td>Endo-12</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>MMVT</td>
<td>3</td>
<td>VT1-330, VT2-310, VT3-300</td>
<td>Endo (LV apex)</td>
<td>Yes</td>
<td>Yes</td>
<td>Endo</td>
<td>Endo</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Epi</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>MMVT</td>
<td>5</td>
<td>VT1-305, VT2-305, VT3-310, VT4-330, VT5-350</td>
<td>Endo (LV anterolateral apex)</td>
<td>Yes</td>
<td>No</td>
<td>Yes-VT1</td>
<td>Endo</td>
<td>7</td>
<td>Endo</td>
<td>Yes</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>MMVT, nonsustained</td>
<td>1</td>
<td>480</td>
<td>Endo (LV anterolateral, apex)</td>
<td>No</td>
<td>No</td>
<td>...</td>
<td>...</td>
<td>Endo</td>
<td>Yes</td>
<td>No</td>
<td>Epi</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>MMVT</td>
<td>1</td>
<td>280</td>
<td>Epi (LV apex, basal inferior)</td>
<td>No</td>
<td>No</td>
<td>...</td>
<td>...</td>
<td>Epi</td>
<td>Yes</td>
<td>No</td>
<td>Epi</td>
<td></td>
</tr>
</tbody>
</table>

Endo indicates endocardial; Epi, epicardial; MMVT, monomorphic VT; NSVT, nonsustained VT; PMVT, polymorphic VT.
Recurrent ICD shocks were defined as recurrent ICD shocks or ICD storm. ICD storm was defined before presentation. All 8 patients presented with either antiarrhythmic medications (class I to IV) and had an ICD to 73 years) (Table 1). Of the 10 patients, 8 were already on therapies that patients had been prescribed long term were either discontinued. Antiarrhythmic agents that had been recently initiated continued at the same or reduced dosage or, in selected cases, discontinued. Antiarrhythmic agents that had been recently initiated were ceased. Patients were seen in ICD clinics for device interrogation at 1 to 2 months and every 3 months thereafter. All data are represented as mean±SD.

Follow-up
VT recurrence was identified by history, by clinical symptoms, and through device interrogation. All patients received anticoagulation with warfarin or aspirin after the procedures. Antiarrhythmic drug therapies that patients had been prescribed long term were either continued at the same or reduced dosage or, in selected cases, discontinued. Antiarrhythmic agents that had been recently initiated were ceased. Patients were seen in ICD clinics for device interrogation at 1 to 2 months and every 3 months thereafter. All data are represented as mean±SD.

Results
Patient Characteristics
All 10 patients were men (mean age, 57±9 years; limits, 48 to 73 years) (Table 1). Of the 10 patients, 8 were already on antiarrhythmic medications (class I to IV) and had an ICD before presentation. All 8 patients presented with either recurrent ICD shocks or ICD storm. ICD storm was defined as ≥3 separate episodes of ICD shocks in a 24-hour period. Recurrent ICD shocks were defined as ≥2 separate episodes of ICD shocks occurring >24 hours apart and were frequent enough to warrant referral for catheter ablation by the treating cardiologists. The 2 patients without an ICD presented with hemodynamically stable VT and underwent direct current cardioversion. Only 2 of the 10 patients previously underwent an endocardial ablation.

The transthoracic echocardiogram findings of these patients are shown in Table 2. The mean LV ejection fraction was 57±13% (limits, 40% to 83%). There were 4 (40%) patients with a LV ejection fraction <50% and 3 (30%) with an apical aneurysm. Midcavity obstruction was present in 5 patients with a mid-LV gradient ranging from 11 to 45 mm Hg. All patients with an apical aneurysm also had midcavitary obstruction. The mean posterior wall thickness was 13±3 mm (limits, 8 to 17 mm), and the mean interventricular septal thickness was 17±6 mm (limits, 8 to 26 mm).

Mapping and Ablation
With programmed ventricular stimulation, 7 patients had sustained or nonsustained, monomorphic VT (Table 3). The mean number of VTs induced was 2.0 (limits, 1 to 5), and the mean cycle length was 335 ms (limits, 270 to 480 ms). The electroanatomical bipolar voltage maps identified epicardial and endocardial scar in 5 patients; epicardial scar only in 3; endocardial scar only in 1; and no scar in 1, who was also noninducible. The most common location of scar was the LV apex (7 patients). An example of a 3D bipolar voltage map in a patient with apical HCM and aneurysm (patient 1) is shown in Figure 1.

In 5 patients, radiofrequency energy was delivered during tachycardia, with termination of VT in all cases. Examples of entrainment and ablation during tachycardia are illustrated in Figures 2 (patient 3) and 3 (patient 4). Time to termination varied from 3 to 30 seconds. The site of successful termination (without recurrence) of tachycardia was epicardial in 4/5 patients and endocardial in 1/5 patients. Patient 7 (Figure 4) had termination of VT during endocardial ablation (in 12 seconds), which was subsequently reinducible; this VT was successfully and permanently eliminated by ablation from the epicardial surface (terminated in 3 seconds). Patient 8 had successful endocardial termination of a bundle branch reentrant VT1 (Table 3) (VT1 left bundle branch block and VT2 right bundle branch block) with ablation. The 1 patient (patient 6) who was noninducible and demonstrated no evidence of scar did not undergo ablation. All patients with scar underwent ablation using a combination of pace mapping and ablation of late/fractionated potentials. In the 2 patients (patients 1 and 2) who had scar but were noninducible for monomorphic VT, pace mapping was performed using the clinical VT ECG for guidance. After ablation, only 1 patient (patient 5) remained inducible; he developed incessant VT
that required surgical cryoablation for successful treatment (Figures 5 and 6). The thickness of the LV wall at the ablation site was >2 cm. This patient’s second VT was successfully ablated in the RV outflow tract.

All patients were discharged from the hospital without major complications; 2 patients did develop moderate-sized groin hematomas. During follow-up, 1 patient developed a large bloody pericardial effusion secondary to a supratherapeutic international normalized ratio of 10 after hospital discharge that required subsequent pericardiocentesis. A 2D echocardiogram that had been performed in this patient before discharge during the index hospitalization for the ablation procedure failed to reveal any significant effusion. The 2 patients who presented with VT and did not have an ICD underwent defibrillator implantation before discharge from the hospital. Thus, all patients were discharged with an ICD in place.

Follow-up
During a mean follow-up of 37.4±16.9 months (limits, 1.6 to 62.4 months; median, 37.0 months), 3 (30%) patients had ICD shocks for recurrent ventricular arrhythmias. Of these, 1 patient (patient 6) was treated medically because he was previously noninducible and had no epicardial or endocardial scar. This patient had a single ICD shock at 30 months for monomorphic VT at 235 beats/min. Following adjustment of antiarrhythmic medications, there were no further ICD therapies in the ensuing 7 months.

Repeat ablation procedures were performed in 2 patients (patients 8 and 9). Patient 8 had recurrent ICD shocks on...
sotalol and underwent repeat procedures at 4 and 8 months. During both procedures, multiple VTs were induced and mapped to the interventricular septum and failed radiofrequency ablation and transcoronary ethanol ablation. The patient was managed with an increased dose of sotalol and β-blocker. At 10 months, the patient experienced episodes of sustained, self-terminating monomorphic VT that were below the ICD detection rate. Mexiletine was added to the sotalol and β-blocker, and the patient was free of further sustained VT or ICD shocks in the ensuing 27 months of follow-up.

Patient 9 had recurrent VT (cycle length, 420 to 450 ms) at 2 months and underwent successful endocardial ablation. Subsequently, the patient stopped taking antiarrhythmic medications and was free of recurrent VT during a subsequent follow-up of 34 months.

Discussion

Monomorphic VT is an uncommon mechanism, and there are limited data on the utility of catheter ablation to treat this particular arrhythmia. Currently, data pertaining to catheter ablation of monomorphic VT in this group are limited to a few case reports that have used a strictly endocardial approach.3–6 In the present study, we report the acute and long-term results of a combined epicardial and endocardial approach to catheter ablation in a highly selected group of patients with HCM-related VT.

As in other nonischemic substrates, the VT circuits in HCM would be expected to involve the epicardium as well as the endocardium.15–18 Because of this and increased LV thickness, endocardial catheter ablation alone would be expected to have limited efficacy in HCM. This hypothesis is supported by the findings of our study. Electrophysiologically identified epicardial scar was present in 80% of patients compared to 60% with endocardial scar. Only 1 patient had endocardial scar without concomitant epicardial involvement. Furthermore, in the 5 patients with hemodynamically stable sustained monomorphic VT, 4 had successful termination of

Figure 3. A, The 12-lead ECG of the VT (cycle length, 270 ms) induced in patient 4 is shown. The morphology of the VT is right bundle branch block with a superior axis. B, The LV endocardial voltage map (left lateral view) failed to reveal an endocardial scar. However, a site with a good entrainment (postspacing interval, 282 ms) was identified (arrow). The electrogram at this site during VT (ABL) shows early activation without any diastolic electrograms seen. Because no scar was present and because of a lack of late/fractionated potentials at this site during sinus rhythm, no ablation was performed endocardially. C, The epicardial voltage map of the same patient (left lateral view) demonstrated the presence of a discrete scar in the lateral base at a site opposite to the good endocardial entrainment site. A site was identified (arrow) with a mid-diastolic potential during VT. Ablation at this site terminated the VT in 3 seconds. The area of scar also had multiple sites with late/fractionated potentials, which also were ablated. Abbreviation as in Figure 2.
VT during epicardial ablation. Even with successful termination of VT during ablation from the epicardium, it took an average of 13 seconds (limits, 3 to 30 seconds) for termination. One patient continued to have incessant VT after termination at 30 seconds during ablation. This patient subsequently required surgical cryoablation to abolish the VT and was found to have an LV thickness of $\geq 2$ cm at the site of ablation. These findings suggest that in patients with HCM-related VT, VT circuits often are located deep in the myocardium and that a strictly endocardial approach would be insufficient for successful ablation.

Interestingly, in the present series, electrophysiologically apical scar was present in 70% of patients and an apical aneurysm in 30%. The pathogenesis of the LV apical aneurysm is not fully understood. However, several etiologies have been hypothesized, including increased LV cavitary systolic pressures from midcavitary obstruction, a genetic predisposition, narrowing of intramural coronary arteries, and myocardial bridging of the left anterior descending artery promoting apical aneurysm. Whatever the mechanism, the presence of an apical aneurysm has been shown to increase the risk of arrhythmic events. The predominance of apical scar in this cohort of patients with HCM may represent the early identification of high-risk individuals who will ultimately manifest apical aneurysms. Additionally, 40% of the patients had end-stage systolic dysfunction (ejection fraction $< 50\%$), which is significantly higher than typically found in the HCM population. The high proportion of patients with apical aneurysms and end-stage systolic dysfunction in the present study are not reflective of the standard HCM population and, therefore, represents a highly selected group of individuals.

Cardiac MRI may be useful in guiding the electrophysiological search for the monomorphic VT circuit. In patients with HCM, delayed contrast-enhanced MRI can identify the size, location, and thickness of the myocardial scar and, thus, may be helpful in the electrophysiological identification of the relevant monomorphic VT circuit. Although MRI may be limited in patients with ICDs, it has been shown to be safe in patients who do not depend on a pacemaker and may become more widely available in the future.

During a mean follow-up of 37.4 ± 16.9 months (median, 37.0 months), 78% (7/9) of patients whose VTs were ablated remained free of ICD shocks. This result shows reasonable efficacy, particularly given that 100% of patients who had an ICD initially presented with recurrent ICD shocks or ICD storm before their procedures.

Figure 4. Left ventriculograms of patient 7 are shown at end diastole (A) and end systole (B). The patient had an apical akinesis and aneurysm. C, The LV endocardial bipolar voltage map was registered to the aorta and coronary arteries (LAD, LCX), and shows a large apical scar. At the site of the catheter (*), there were diastolic potentials seen, and during endocardial ablation at this site, the VT terminated in 12 seconds. However, it was subsequently reinducible even after several radiofrequency ablations at this site. D, On the epicardial surface directly opposite the endocardial site of VT termination, there were fractionated, mid-diastolic electrograms noted on the distal mapping electrodes (ABL d). E, Radiofrequency ablation at this site terminated the VT in 3 seconds and rendered it noninducible. Abbreviations as in Figure 2.
patient who was not ablated due to noninducibility and lack of identifiable scar had recurrence. These recurrences are not surprising given the increased LV wall thickness in patients with HCM and the relatively long time needed for VT termination during ablation (mean, 13 seconds), suggesting deeper myocardial VT circuits. Additionally, during the follow-up period, there is also potential for disease progression.

**Limitations**

Monomorphic VT is uncommon in HCM. Additionally, there was a higher prevalence of apical aneurysms and end-stage

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**Figure 5.** A, During VT in patient 5, the site of earliest activation is denoted (star) on the fluoroscopy image (LAO view). The site with earliest activation was in the distal coronary sinus (CS). The ablation catheter is in the pericardial space. B, Electrogram recordings from the CS catheter during VT demonstrated the earliest activation to be in the CS 5 to 6 electrodes. The diastolic electrograms at this site preceded the surface QRS by ~70 ms. C, A limited 3D bipolar voltage map of the epicardial surface and endocardial LV are shown a left lateral cranial view. There is an area of epicardial scar noted in the region of the LV anterolateral base at the site of early activation. This area was also the site of the best pace map (arrow) in sinus rhythm (D). During VT, ablation in this region terminated the tachycardia in 30 seconds. However, the VT remained inducible, and after further ablation, it became incessant, requiring surgical cryoablation.

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**Figure 6.** A, During surgery for incessant VT following catheter ablation (patient 5), the sites of epicardial radiofrequency ablation (arrows) could be identified in the basal, anterolateral LV. B, Surgical cryoablation in this region was necessary to abolish the VT. At the time of surgery, the LV thickness at this site was >2 cm.
Clinical and echocardiographic features of this patient suggest ischemic cardiomyopathy, and the effect of arterial embolism on the patients was assessed. In this study, we assessed the effect of arterial embolism on the patients using a porcine model of healed myocardial infarction. We found that arterial embolism increased the incidence of ventricular fibrillation and impaired cardiac function in a dose-dependent manner. This study suggests that arterial embolism may be an important factor in the development of ventricular tachycardia in patients with ischemic cardiomyopathy. Future studies are needed to further elucidate the mechanisms involved in the development of ventricular tachycardia in patients with ischemic cardiomyopathy. 

Conclusions
The present study demonstrates that arterial embolism increases the incidence of ventricular tachycardia in a porcine model of healed myocardial infarction. These findings support the hypothesis that arterial embolism may be an important factor in the development of ventricular tachycardia in patients with ischemic cardiomyopathy. Further studies are needed to investigate the mechanisms involved in the development of ventricular tachycardia in patients with ischemic cardiomyopathy.

Disclosures
Dr d’Avila has received consulting fees from Biosense Webster Inc. Dr Marchlinski has received research grants and honoraria from Biosense Webster Inc. Dr Reddy has received research grants and consulting fees from Biosense Webster Inc.

References
Monomorphic ventricular tachycardia (VT) is rare in patients with hypertrophic cardiomyopathy (HCM), and implantable cardioverter-defibrillators (ICDs) are the mainstay of therapy for prophylaxis against sudden cardiac death. For patients with recurrent ICD shocks despite aggressive antiarrhythmic medications and antitachycardia pacing, catheter ablation may be an option, although there are limited data regarding this. The present study details a series of case reports from multiple centers where combined epicardial-endocardial ablation was performed in a highly selected group of patients with HCM-related monomorphic VT. In these patients, the mechanism of monomorphic VT was mostly scar-related reentry. The location of scar was epicardial in 80% and endocardial in 60%. Using a combined epicardial-endocardial approach, radiofrequency ablation was effective in eliminating VT in 89% of patients acutely. After 37±17 months, the freedom from recurrent ICD shocks was 78%. These results demonstrate that a combined epicardial-endocardial approach to catheter ablation is a reasonably efficacious option for patients with HCM-related monomorphic VT if refractory to aggressive trials of antiarrhythmic medications and antitachycardia pacing.
Long-Term Outcomes of Combined Epicardial and Endocardial Ablation of Monomorphic Ventricular Tachycardia Related to Hypertrophic Cardiomyopathy
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SUPPLEMENTAL MATERIAL
**MOVIE LEGEND**

**Movie 1.** The epicardial bipolar voltage map (mesh) is shown. The map was registered to the computed tomography angiograms of the aorta, coronary arteries, and left ventricle. The large apical and lateral LV scar is seen.