Short-Term Results of Substrate Mapping and Radiofrequency Ablation of Ischemic Ventricular Tachycardia Using a Saline-Irrigated Catheter

Vivek Y. Reddy, MD, FACC,* Petr Neuzil, MD,† Milos Taborsky, MD,† Jeremy N. Ruskin, MD, FACC*
Boston, Massachusetts; and Prague, Czech Republic

OBJECTIVES
We evaluated the safety and acute procedural efficacy of a combined electrophysiologic and anatomic approach to ablation of all inducible ventricular tachycardias (VT) during sinus rhythm using an irrigated radiofrequency (RF) ablation catheter.

BACKGROUND
Ventricular tachycardia associated with chronic myocardial infarction (MI) is frequently hemodynamically intolerable and associated with multiple electrocardiographic morphologies. Because traditional mapping techniques are contingent on hemodynamic stability for adequate VT mapping, VT ablation therapy for many patients has been disappointing.

METHODS
High-density electroanatomic mapping was performed during either sinus rhythm in 11 consecutive patients with a history of MI and ventricular arrhythmias. The RF ablation was performed using an irrigated-tip ablation catheter. All inducible VTs were targeted for catheter ablation during sinus rhythm.

RESULTS
The RF ablation lesions were placed in a linear fashion traversing the border zones of infarcted and normal tissue (mean of 3.4 linear lesions/patient). With this strategy, the target VT was eliminated in 9 of 11 patients (82%). Furthermore, when targeting all inducible monomorphic VTs, complete procedural success was achieved in 7 of 11 patients (64%). During the follow-up period (mean 13.1 ± 1.9 weeks), spontaneous VT was only noted in the two patients with no acute procedural benefit.

CONCLUSIONS
By identifying potentially arrhythmogenic tissue during sinus rhythm, substrate mapping can guide the ablation of a majority of inducible VTs using an irrigated RF ablation catheter. This emerging therapeutic paradigm may be considered in the management of patients with multiple hemodynamically unstable monomorphic VTs. (J Am Coll Cardiol 2003;41:2228–36) © 2003 by the American College of Cardiology Foundation

Malignant ventricular arrhythmias (ventricular tachycardia/ventricular fibrillation [VT/VF]) are a frequent cause of mortality in patients with a history of myocardial infarction (MI) and depressed left ventricular (LV) ejection fraction (1–3). The optimal treatment of this group of patients would be eradication of the arrhythmias. To this end, critical portions of hemodynamically stable VT circuits can be identified by transcatheter endocardial mapping during the tachycardia and eliminated by radiofrequency (RF) catheter ablation (4–8). However, only a minority (4–8%) of patients experience VT that is hemodynamically tolerated sufficiently well to allow adequate mapping, and even these patients frequently exhibit inducible hemodynamically unstable VTs with multiple electrocardiographic (ECG) morphologies (9). Thus, catheter ablation of VT has largely been relegated to an adjunctive role even at experienced centers.

The anatomic substrate for VT in these patients is predominantly located at the border zone between the normal and scarred myocardium (10). This arrhythmogenic zone has distinguishing electrogram (EGM) characteristics during sinus rhythm: low amplitude and prolonged duration (11). Using an electroanatomic mapping system (CARTO, Biosense-Webster Inc., Diamond Bar, California), it is possible to construct three-dimensional maps overlaying these EGM characteristics onto the LV chamber geometry. In a seminal study by Marchlinski et al. (12), the infarcted myocardium was identified using bipolar voltage amplitude criteria, and catheter-based RF ablation lesions were placed in a linear fashion to control drug-refractory unstable VT in nine post-MI patients. However, that study was directed to a specific population of patients with drug-refractory VT. In an effort to better define the potential of electroanatomic mapping-based substrate-ablation of VT, we examined: 1) the ability of EGM characteristics during normal sinus rhythm to delineate the scarred myocardium in patients with a history of MI and VT/VF, and 2) the use of this “substrate mapping” strategy to guide the ablation of all inducible VTs using a novel saline-irrigated RF ablation catheter.

METHODS

Patient population. Normal sinus rhythm EGM characteristics were defined using electroanatomic mapping in seven control patients with structurally normal ventricles. The 11 consecutive patients in the study population were selected based on a history of MI, and spontaneous or inducible VT/VF. The mean age of the patients was 66 ±
Abbreviations and Acronyms

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>ECG</td>
<td>electrocardiographic</td>
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<tr>
<td>EGM</td>
<td>electrogram</td>
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<tr>
<td>ICD</td>
<td>implantable cardioverter-defibrillator</td>
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<tr>
<td>LV</td>
<td>left ventricle/ventricular</td>
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<tr>
<td>MI</td>
<td>myocardial infarction</td>
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<tr>
<td>MMVT</td>
<td>monomorphic ventricular tachycardia</td>
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<tr>
<td>RF</td>
<td>radiofrequency</td>
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<tr>
<td>RV</td>
<td>right ventricle/ventricular</td>
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<tr>
<td>SWVT</td>
<td>&quot;sine wave&quot;-like ventricular tachycardia</td>
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<tr>
<td>VF</td>
<td>ventricular fibrillation</td>
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<tr>
<td>VT</td>
<td>ventricular tachycardia</td>
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7.9 years (range 54 to 77 years), and the mean LV ejection fraction was 31 ± 8% (range 20% to 40%). There were a total of six patients with anterior MIs, four patients with inferior/posterior MIs, and one patient with both anterior and inferior MIs (Table 1). The LV endocardial mapping was performed during either sinus rhythm or right ventricular (RV) pacing (between 600 and 900 ms cycle length). Mapping was performed during RV pacing if the patient was pacemaker-dependent (one patient), or pacing attenuated the degree of catheter-induced ventricular ectopy (two patients).

The subject cohort included seven patients with spontaneous sustained monomorphic ventricular tachycardia (MMVT), two patients with spontaneous VF and inducible MMVT, and two others with a history of syncope and inducible MMVT (Table 2). An implantable cardioverter-defibrillator (ICD) was present in 8 of 11 patients. This relatively heterogeneous group of patients was chosen to assess the widespread applicability of this substrate-ablation strategy. Common to each patient was a high risk for recurrent VT/VF (3,13). Patients were evaluated after written informed consent according to institutional guidelines.

Electrophysiology study. Patients were studied in a post-absorptive state with conscious sedation. Programmed stimulation was performed with up to three extrastimuli and rapid pacing from two RV and two LV sites to document all inducible VTs. Stimulation was continued until the same morphology of VT (defined as the same major axis and preordial transition) was repeatedly induced. In one patient with a history of spontaneous VT and only inducible nonsustained VT, sustained VT was inducible after ajmaline infusion. Hemodynamic instability was defined as either loss of consciousness or a decrease in the systolic blood pressure to <80 mm Hg for >10 s (prompt pace-termination or electrical cardioversion was then performed).

**Electroanatomic mapping.** Catheter access to the LV was achieved via the retrograde aortic approach, and in two patients an additional trans-septal approach. The LV electroanatomic mapping was performed using the CARTO system as previously described (14). Endocardial EGMs were recorded using deflectable Navistar catheters (BioSense-Webster Inc.) with a 4-mm-tip distal electrode. After intravenous heparin infusion, mapping was performed (using a fill threshold <20 mm) with an emphasis on fully defining the border zones of the infarcted tissue. The peak-to-peak bipolar voltages associated with each point were displayed upon the endocardial cast.

**Reference values for electroanatomic maps.** Reference values for distinguishing normal from abnormal tissue were established by mapping the LV of the seven control patients during normal sinus rhythm (mean of 69 points/patient). The mean bipolar voltage value obtained was 8.4 ± 7.8 mV (range 0.5 to 44.7 mV) and, most importantly, the 95% voltage amplitude cutoff was >1.53 mV. That is, only 5% of the points obtained were less than this value. Accordingly, normal tissue was defined as bipolar voltage >1.5 mV, and dense scar was arbitrarily defined as <0.5 mV (12). Accordingly, the color display was set to span 0.5 to 1.5 mV. As previously described, endocardial surface areas were calculated assuming multiple rectangular or trapezoidal shapes of either abnormal (<1.5 mV, bipolar voltage) or densely scarred (<0.5 mV, bipolar voltage) tissue (12). Using the CARTO electronic calipers to measure distances, the scar dimensions were estimated to best fit with either a circle,

**Table 1. Results of Substrate Mapping**

<table>
<thead>
<tr>
<th>Patient</th>
<th>LVEF (%)</th>
<th>MI Location</th>
<th>No. of VTs</th>
<th>VTCL Range (ms)</th>
<th>Rhythm During Mapping</th>
<th>No. of Pts.</th>
<th>Size of LV (cm²)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Abnormal EGM</td>
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<tr>
<td>1</td>
<td>20</td>
<td>Ant</td>
<td>7</td>
<td>245–435</td>
<td>RVp</td>
<td>370</td>
<td>51</td>
</tr>
<tr>
<td>2</td>
<td>20</td>
<td>Ant</td>
<td>1</td>
<td>278</td>
<td>SR</td>
<td>345</td>
<td>50</td>
</tr>
<tr>
<td>3</td>
<td>25</td>
<td>Ant</td>
<td>4</td>
<td>303–342</td>
<td>RVp</td>
<td>535</td>
<td>55</td>
</tr>
<tr>
<td>4</td>
<td>40</td>
<td>Post</td>
<td>3</td>
<td>348–276*</td>
<td>RVp</td>
<td>152</td>
<td>15</td>
</tr>
<tr>
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<td>311–643</td>
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</tr>
<tr>
<td>6</td>
<td>25</td>
<td>Ant</td>
<td>3</td>
<td>301–348</td>
<td>SR</td>
<td>582</td>
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<tr>
<td>7</td>
<td>35</td>
<td>Post</td>
<td>3</td>
<td>356–375</td>
<td>SR</td>
<td>163</td>
<td>27</td>
</tr>
<tr>
<td>8</td>
<td>20</td>
<td>Ant/Inf</td>
<td>3</td>
<td>272–454</td>
<td>SR</td>
<td>389</td>
<td>65</td>
</tr>
<tr>
<td>9</td>
<td>30</td>
<td>Inf/Post</td>
<td>3</td>
<td>297–344</td>
<td>SR</td>
<td>295</td>
<td>70</td>
</tr>
<tr>
<td>10</td>
<td>40</td>
<td>Inf</td>
<td>2</td>
<td>288–303</td>
<td>SR</td>
<td>237</td>
<td>15</td>
</tr>
<tr>
<td>11</td>
<td>25</td>
<td>Ant</td>
<td>4</td>
<td>258–342</td>
<td>SR</td>
<td>364</td>
<td>96</td>
</tr>
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</table>

*Sustained VT was induced only after the infusion of intravenous ajmaline.

Ant = anterior left ventricle wall; EGM = electrogram; Inf = inferior left ventricle wall; LV = left ventricle; LVEF = left ventricular ejection fraction; MI = myocardial infarction; Post = posterior left ventricle wall; Pts = number of points obtained per CARTO map; RVp = right ventricular pacing; SR = sinus rhythm; VTs = number of inducible ventricular tachycardias; VTCL = ventricular tachycardia cycle length.
RF catheter ablation. As a general strategy, pace-mapping (using bipolar pacing) was performed during sinus rhythm in the border zone between scar and normal tissue to approximate the exit site of each inducible VT. When seen, a delay in stimulus to QRS interval during pace-mapping within the scar was used to identify putative proximal sites within the VT circuit. Sequential point lesions were placed to create two bisecting linear ablation lines: one from the exit site back into the dense scar, and the other perpendicular to the first line at the exit site. These lesions typically were extended till either pacing could not capture the tissue, or the pace-maps revealed exit of the wavefront from the opposite side of the scar. The lesions were placed <10 mm apart. Potentially adverse effects on LV function were minimized by placing lesions exclusively in abnormal tissue (bipolar voltage <1.5 mV).

A few lesions (<5%) were placed using a 4-mm-tip temperature-controlled nonirrigated ablation catheter (Navistar, Biosense-Webster Inc.). Radiofrequency current was applied in 60-s intervals using a target temperature of 55°C to 60°C. However, the majority of RF lesions (> 95%) were placed using a saline-irrigated 3.5-mm-tip ablation catheter (Navistar, Biosense-Webster Inc.). This catheter is equipped with a central lumen and a “showerhead” configuration at the catheter tip. To actively cool the ablation electrode, saline (0.45% to 0.9%) was infused through the saline-irrigated catheter at 2 ml/min during catheter manipulation to maintain lumen patency, or at 15 to 30 ml/min during RF delivery. The RF lesions were placed in 60-s intervals under power control of 25 to 50 W with impedance monitoring. The activated coagulation time was maintained over 220 s. The RF energy applications were “tagged” for display on the CARTO system, and the final length of each linear lesion was measured.

Attempts were made to localize and ablate the target (and usually slowest) VT first. The target VT was defined as either the clinically spontaneous MMVT (in seven patients) or inducible MMVT(s) during electrophysiologic testing (in the remaining four patients) (Table 2). After every pair of bisecting linear lesions, programmed ventricular stimulation was repeated to induce any residual VT(s), which were in turn targeted for ablation. All patients underwent programmed stimulation using up to triple extrastimuli and rapid pacing from two RV and one to two LV sites after all ablation lesions were applied. A successful procedure was defined as either: 1) inability to induce any sustained VT; 2) induction of only a morphologically indeterminate VT with no isoelectric segment (e.g., a “sine wave”-like morphology [SWVT]); or 3) induction of only VF. Partial success was defined as elimination of one or more VTs (including the target VT) but persistence of at least one other inducible VT. Failure was defined as persistent inducibility of the “clinical” or predominant VT.

Postprocedure management and follow-up. Patients received intravenous heparin overnight, and those who underwent RF ablation were also prescribed coumadin for three months (international normalized ratio >2.0). Patients underwent echocardiography to re-evaluate LV function before discharge from the hospital. Before ablation, 7 of 11 patients were being treated with amiodarone. This drug treatment was unchanged during the follow-up period. During follow-up every three months, the ICDs were interrogated.

RESULTS

Electrophysiologic testing and substrate mapping. Programmed ventricular stimulation induced 3.7 ± 1.8 sustained monomorphic VTs per patient (range 1 to 7)—the majority of which were not hemodynamically tolerated. Only Patient #5 had hemodynamically stable VTs, but multiple other unstable VTs were also inducible.

High-density endocardial electroanatomic mapping of the LV was accomplished by acquiring 285 ± 110 discrete points/patient (range 166 to 535 points). Despite the
frequent presence of large areas of wall motion dysfunction during LV contrast ventriculography, bipolar voltage amplitude mapping identified a discrete scar and border zone in all patients. The endocardial surface area demonstrating abnormal bipolar voltage criteria was 61 ± 35 cm², and ranged from 15 to 117 cm². The area of dense scar occupied 22 ± 17 cm², and ranged from 2 to 54 cm² (Table 1). This corresponded to the area of aneurysmal dilation observed during echocardiography or contrast ventriculography.

RF catheter ablation using an irrigated-tip catheter. The results of three representative patients who underwent catheter ablation using the irrigated-tip ablation catheter are shown in Figures 1 to 3. Shown in Table 3, a mean of 37 ± 23 lesions were applied per patient (range 11 to 86) for a total of 3.4 ± 1.8 linear lesions (range 1 to 6). The average length of these lesions was 3.4 ± 1.2 cm (range 1.6 to 7.2 cm). Using noninducibility of VT as the end point, complete success was achieved in 7 of 11 (64%) patients and partial success in 2 of 11 patients. Of note, in both patients

Figure 1. Substrate mapping and radiofrequency (RF) ablation in Patient #7. The scale ranges from 0.5 to 1.5 mV in the bipolar voltage map. Purple represents normal tissue, and red represents highly scarred tissue. The localized postero-basal infarction is seen in the postero-anterior view in the bipolar voltage map. The morphologies of the two major induced ventricular tachycardia (VTs) as well as the corresponding pace-mapping sites, are shown (arrows point to sites of stimulation). Pacing between these two sites (red dot) generated a QRS morphology that alternated between VT-1 and VT-2, but with a longer stimulus-QRS latency time. This site likely represented a common pathway for the two VT circuits and was incorporated in the ablation strategy (a geometry/mesh map is shown on the bottom right). No VT was inducible postablation. Each red dot represents a 1-min RF application.
with partial success, Patients #9 and #11 (Fig. 3), all VTs inducible by RV stimulation (including the clinically spontaneous VT) were successfully ablated. However, LV stimulation after ablation induced another VT, which was not eliminated by further RF ablation in either patient. Thus, elimination of the target VT was achieved in 9 of 11 (82%) patients. Two patients were deemed failures because the target VTs remained inducible.

Complications, fluoroscopy exposure, and procedural times. Impedance increases were not observed in any of the cases, with a total of over 400 lesions applied. In Patient #11, pericardial effusion with impending tamponade occurred during an attempted transseptal puncture (Fig. 3). Pericardiocentesis was performed and the patient suffered no long-term sequelae. No other complications were observed. The total fluoroscopy times ranged from 35 to 145 min (mean 61 ± 32 min), and the total procedural time ranged from 5 to 8.5 h (mean 7.0 ± 1.3 h).

Fluoroscopy times decreased from a mean of 75 min in the first six cases to 45 min in the last five cases. There was no echocardiographic evidence of deterioration of overall or regional LV systolic function following the ablation procedure. Short-term follow-up. All patients were followed in the ICD clinic for a minimum of 12 months (mean 13.1 ± 1.9 months; range 12 to 16 months). There were no ICD therapies or detection of VT in any patient in whom either complete or partial acute procedural success was achieved. However, both patients in whom the ablation procedure failed to eliminate the target VT experienced clinical VT recurrences: 1) Patient #3 received three appropriate ICD therapies, and 2) Patient #4 developed MMVT requiring cardioversion while in the hospital, prompting ICD implantation (this patient later underwent a second ablation procedure using the substrate-based approach, and has since remained free of VT for over a year). In sum, the VT frequency decreased from a mean of 8.4 VT episodes (range 0 to 88) to 0.3 episodes (range 0 to 3) postablation. Delayed complications were not seen during follow-up in any patient.

DISCUSSION

Results of this study demonstrate that an irrigated-tip RF ablation catheter can be used safely and effectively during normal sinus rhythm to modify the arrhythmogenic substrate and render VT noninducible in patients. The substrate-based ablation strategy. The arrhythmogenic substrate for VT resides at least partly in the border zone between densely infarcted/fibrotic tissue and normal tissue. Over the past three decades, two effective surgical strategies have been developed: 1) subendocardial resec-
Figure 3. Substrate mapping and radiofrequency (RF) ablation in Patient #11. A large anterior infarction is noted in the bipolar voltage maps (right anterior oblique view in A and left anterior oblique-cranial view in B). The exit site of the clinically significant ventricular tachycardia (VT) was identified at the septal border of the scar (shown by arrow in A). This and two other VTs (not depicted) were successfully eliminated by the linear ablation strategy shown. Repeat programmed stimulation from two right ventricular sites (using triple extrastimuli and rapid pacing) failed to induce any arrhythmias, but stimulation from the left ventricle induced another hemodynamically unstable VT from the basal-lateral aspect of the scar (exit site shown by arrow in B). Adequate tissue contact could not be achieved using a retrograde aortic approach (as judged by fluoroscopic movement of the catheter within the cardiac silhouette, and by impedance monitoring). During an attempted transseptal puncture, the pericardial space was invaded and the procedure was terminated prematurely. This case was regarded a partial success.
— involving removal of the subendocardial layer containing the arrhythmogenic tissue, and 2) encircling endocardial ventriculotomy—whereby circumferential surgical lesions are placed through the border zone, presumably interrupting potential VT circuits (15–19) (Fig. 4). This experience was critical in establishing the concepts on which substrate-based ablation is based: 1) the arrhythmogenic substrate is predominantly located in the subendocardium; 2) this substrate has distinguishing EGM characteristics; and 3) removal or interruption of this arrhythmogenic tissue can abolish VT.

The advent of electroanatomic mapping systems has provided the opportunity to reconstruct and electronically manipulate endocardial casts of the LV. Studies have revealed that

![Ablation Characteristics and Outcome](image)

*Induced with three closely coupled extrastimuli during programmed stimulation from the right ventricle. †Induced with three closely coupled extrastimuli during programmed stimulation from the left ventricle. ‡Pericardial effusion during a transseptal needle puncture prompted premature termination of the procedure.

CL = cycle length; EPS = electrophysiologic study; MMVT = monomorphic ventricular tachycardia; RF lesions = total number of 1-min radiofrequency point lesions applied; SWVT = "sine-wave"-like ventricular tachycardia (indeterminate morphology with no isoelectric baseline); VT = ventricular tachycardia.

![Subendocardial Resection](image)

Figure 4. The border between the normal and infarcted/aneurysmal wall contains a stylized ventricular tachycardia (VT) circuit—predominantly endocardial, and partially intramural. The surgical procedures, subendocardial resection, and ventriculotomy are believed to either remove or transect critical endocardial portions of the VT circuit, respectively. After mapping the border zone using the electroanatomic mapping system, the putative exit site of the VT was identified with pace-mapping, and catheter-based linear lesions were placed. Although a single catheter-based linear lesion set along the scar border could potentially interrupt the circuit(s), unlike during surgery, one cannot ensure the lesion's depth and continuity. Therefore, a second linear lesion set was placed proceeding into the scarred myocardium—with the hope that this empiric lesion set would transect critical portions of the VT circuit(s). LV = left ventricular. Figure reproduced and modified with permission, courtesy of J. Miller, MD, and Williams & Wilkins Inc. (19).
the scarred substrate could be accurately identified using bipolar voltage criteria in an animal model of healed MI (20). And in a recent landmark series, Marchlinski et al. (12) demonstrated the proof-of-principle that anatomically based linear lesions extending from scarred to normal tissue were effective in suppressing drug-refractory MMVT in nine patients with ischemic heart disease.

**Localization of the VT circuit.** Using techniques similar to those previously described (12), pace-mapping was performed at the border zone of the infarct to identify the putative exit point of each induced VT. Despite the fact that ventricular activation with pace-mapping during sinus rhythm results in omnidirectional spread of activation (unlike activation during re-entrant VT, which proceeds directionally out of the scar), there was still a good match between the pace-map and VT ECG morphologies. This is likely due to the fact that the “antidromic” waveform of pace-mapping (the spread of activation proceeding into the scar) encounters diseased tissue—thereby contributing minimally to the surface QRS morphology. Points within the VT circuit were occasionally identified by pace-mapping in close proximity to the exit site but farther into the scarred myocardium. The duration of latency from stimulation to the onset of QRS complex was observed. Notwithstanding the fact that this inevitably results in the identification of irrelevant “inner loop” and “adjacent bystander” sites as well as the desired “isthmus” sites (21), this strategy nonetheless provides a general idea of the orientation of the VT circuit.

**RF catheter ablation.** The ability to identify the anatomic substrate during sinus rhythm obviates the need for hemodynamic stability during VT because mapping during sustained VT is not required. Indeed, activation mapping was not performed in this study (and entrainment mapping only performed in Patient #5). Because all inducible VTs, hemodynamically stable or unstable, were targeted for ablation, the question of how to gauge success was raised. Most previous studies had defined a successful VT ablation procedure as noninducibility of either the clinically documented VT(s) or hemodynamically stable VT(s). In our study, the only ventricular tachyarrhythmias not targeted for ablation were VF and SWVT. The definition of “sine wave”-like VT was not dependent on the rate of the VT but rather on the indeterminate nature of the rhythm’s ECG morphology. Indeed, the rates of the two SWVTs noted in this series (cycle length 301 ms and 311 ms) were actually slower than were many VTs seen in other patients that were targeted for ablation. The clinical significance of postablation inducible SWVT (as well as inducible VF) can only be assessed by prospective clinical trials.

Unlike most previous studies, programmed stimulation was also performed from the LV before a procedure was deemed successful. Indeed, the two patients labeled as partial successes may have been classified as complete successes if only RV stimulation had been performed. Despite this relatively rigorous definition of a successful ablation procedure, it was possible to ablate a majority of the VTs. In most cases, failure was due to an inability to achieve adequate electrode-tissue contact at the desired location, a limitation that should improve with further refinements in catheter design. The fluoroscopy exposure and procedure times will likely similarly diminish with these advances.

The approach used in the patient who had both hemodynamically stable and unstable VT was similar to that used in patients with only unstable VT. A sinus rhythm substrate map of the LV was created, and pace-mapping was performed to identify the putative exit site. Traditional entrainment techniques during tachycardia were then applied to identify the critical isthmus in the VT circuit. While a single point lesion at the isthmus site sufficed to eliminate the target VT, it is known that multiple morphologies of VT can originate from a common mass of tissue (22,23). Because the goal was to ablate all inducible VTs, linear ablation was performed incorporating the entrainment sites within the lesions. This strategy of combining the anatomic and entrainment data possesses several advantages: 1) identification of critical portions of the stable VT circuit is facilitated by knowledge of the location of the scar and border zones; 2) patient discomfort is minimized as most of the procedure is conducted during sinus rhythm; and 3) this strategy may eliminate not only the hemodynamically stable target VT, but all inducible VTs—which are often hemodynamically unstable.

This study also highlights the efficacy and safety of the saline-irrigated RF ablation catheter for VT ablation. This catheter was employed both because of its ability to generate deeper lesions, and because active cooling of the ablation electrode helps prevent the formation of coagulum and elevations of impedance. Notwithstanding the limited number of patients in this series, no evidence was seen of thromboembolic phenomena in this series in the over 400 RF lesions applied. This study does not address the relative efficacies of the irrigated and nonirrigated catheters—a comparison that must be evaluated in a prospective randomized fashion.

**Follow-up.** Ventricular arrhythmias did not recur in any patient whose initial procedural outcome was either a complete or partial success over a mean follow-up of 13.1 months. In both patients with failed ablations, VT recurred during the follow-up period. Despite the fact that this was a patient population at high risk for recurrent ventricular arrhythmias (3,13), these data must be interpreted with caution because of both the nonrandomized nature of this series, and the relatively short duration of follow-up. Also, because these patients were not necessarily the “typical” VT ablation candidates (i.e., patients who had previously experienced multiple recurrent VT episodes/ICD shocks), results of this study may not necessarily be applied to that population. This study would best be interpreted as provocative data to form the basis of larger prospective randomized clinical trials to fully assess the clinical utility of this approach. Also, the results of this study do not imply that patients undergoing substrate ablation procedures do not require ICDs when established indications are present. The
potential impact of this ablation strategy on both the mechanisms and risk of sudden cardiac death are unknown.

**Study limitations.** The control patient data utilized in this study as well as earlier studies were obtained during normal sinus rhythm, but some of the patients in this study were mapped during RV pacing. Although evidence shows that this does not significantly affect the EGM voltage amplitude (24), this needs to be fully studied.

The mechanisms of elimination of VT is thought to be due to interruption of critical portions of the circuit, but this remains speculative. In fact, one cannot determine from this study the number or length of the linear lesions required for successful VT ablation as programmed stimulation was not repeated after every point lesion, but rather after completion of one to two linear lesions.

Although no increases in impedance or thromboembolic phenomena occurred in any of these cases, the number of patients in whom RF ablation was performed was limited. Indeed, a stroke occurred in 1 of 16 patients in the series reported by Marchlinski et al. (12), albeit not with the theoretically safer irrigated ablation catheter, as was used in our series.

**Conclusions.** The principal advantage of substrate mapping to guide VT ablation lies in the potential application of this strategy to virtually any morphologically discernible monomorphic VT irrespective of its hemodynamic effect. This raises the possibility that most patients with a history of MI and monomorphic VT may ultimately be candidates for catheter ablative therapy. Substrate mapping may represent a catheter-based method to replicate the highly efficacious results of substrate modification by surgical techniques, but with less attendant morbidity and mortality.

**Acknowledgments**

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