Radiofrequency Catheter Ablation of Ventricular Tachycardia in Patients Without Structural Heart Disease

Lawrence S. Klein, MD; Hue-Teh Shih, MD; F. Kevin Hackett, MD; Douglas P. Zipes, MD; and William M. Miles, MD

Background. Radiofrequency energy has been used safely and successfully to eliminate accessory pathways in patients with the Wolff-Parkinson-White syndrome and the substrate for atrioventricular nodal reentrant tachycardia. However, this form of ablation has had only limited success in eliminating ventricular tachycardia in patients with structural heart disease. In contrast, direct-current catheter ablation has been used successfully to eliminate ventricular tachycardia in patients with and without structural heart disease. The purpose of this study was to test whether radiofrequency energy can safely and effectively ablate ventricular tachycardia in patients without structural heart disease.

Methods and Results. Sixteen patients (nine women and seven men; mean age, 38 years; range, 18–55 years) without structural heart disease who had ventricular tachycardia underwent radiofrequency catheter ablation to eliminate the ventricular tachycardia. Two patients presented with syncope, nine with presyncope, and five with palpitations only. Mean duration of symptoms was 6.7 years (range, 0.5–20 years). Radiofrequency catheter ablation successfully eliminated ventricular tachycardia in 15 of 16 patients (94%). Sites of ventricular tachycardia origin included the high right ventricular outflow tract (12 patients), the right ventricular septum near the tricuspid valve (three patients), and the left ventricular septum (one patient). The only ablation failure was in a patient whose ventricular tachycardia arose from a region near the His bundle. An accurate pace map, early local endocardial activation, and firm catheter contact with endocardium were associated with successful ablation. Radiofrequency ablation did not cause arrhythmias, produced minimal cardiac enzyme rise, and resulted in no detectable change in cardiac function by Doppler echocardiography.

Conclusions. Radiofrequency catheter ablation of ventricular tachycardia in patients without structural heart disease is effective and safe and may be considered as early therapy in these patients. (Circulation 1992;85:1666–1674)

KEY WORDS • ventricular tachycardia • ablation • radiofrequency current

Elimination of all or part of the anatomical substrate responsible for supraventricular tachycardia in patients with the Wolff-Parkinson-White syndrome and atrioventricular (AV) nodal reentry7–10 with the use of radiofrequency (RF) energy has been reported with a high success rate and few complications. However, equivalent success was not achieved in patients with ventricular tachycardia (VT)11–16 by using either direct-current or RF energy unless the mechanism of VT was bundle branch reentry.17,18 Failure to eliminate VT by using RF energy delivered through a catheter in patients with coronary artery disease may be due to the small size and the shallow depth created by the RF lesion,19,20 which prevents it from reaching subendocardial (or deeper) regions of the heart. Contributing factors could also include inaccurate mapping in scarred ventricles or a location of the VT focus or pathways at sites deep to the endocardium. Successful elimination of idiopathic (usually right ventricular) VT in patients without structural heart disease with direct-current countershocks has been reported.24 However, barotrauma, risk of ventricular perforation, and other potential complications associated with direct-current countershocks make this technique less desirable unless very low energies are used.

The purpose of this study was to test the hypothesis that the endocardial lesion caused by RF energy would eliminate a myocardial focus responsible for VT in patients with idiopathic VT associated with a structurally normal heart. A successful outcome would help establish one or all of the following: transmission of RF energy to the focus unimpeded by damaged endocardium, accurate mapping, and location of the focus in or near the endocardium.
Patient Population

This study comprised 16 patients: nine women and seven men. Mean patient age was 38 ± 9 years, ranging from 18 to 55 years. None of the 16 patients had identifiable structural heart disease by two-dimensional and Doppler echocardiography. Cardiac catheterization was performed in six of the 16 patients (patients 1, 3, 5, 6, 11, and 12). Patients 3 and 6 had normal endomyocardial biopsy. No patient had magnetic resonance imaging. Two patients presented with syncope, nine patients with presyncope, and five patients with palpitations only. Mean duration of symptoms was 5.9 ± 5.6 years, ranging from 6 months to 20 years. The patients had previously received a mean of 3.0 ± 2.6 antiarrhythmic drug trials, ranging from 0 to 10. All patients reported frequent palpitations and all had had multiple ECG recordings of VT before referral. (See Table 1.)

Electrophysiology Study and Ablation Protocol

Patients were sedated with midazal, fentanyl, and promethazine. Programmed electrical stimulation was performed using up to three ventricular extrastimuli at three drive cycle lengths from two right ventricular sites using twice diastolic pacing threshold (2-msec pulse width). Isoproterenol infusions were given in 1-µg/min increments to reach 1–5 µg/min in all patients in whom sustained VT was not induced in a control state. End points were spontaneous sustained VT, inducible sustained VT, or 5 µg/min isoproterenol. Programmed electrical stimulation was repeated at each isoproterenol dose. When sustained VT (greater than 30 seconds) was not induced, nonsustained VT (greater than or equal to three beats and less than 30 seconds), either spontaneous or induced, or occasionally premature ventricular contractions (PVCs), were targeted for ablation. Activation mapping and pace mapping were performed via the ablation catheter once the clinical ventricular arrhythmia was induced or present. Pace mapping was performed at high current output (5–10 mA; 2-msec pulse width) in all patients. The ablation catheter was introduced into the femoral vein (or artery) initially in all patients. When ablation was not successful with this approach, the right subclavian vein was used (patient 7). Activation mapping was performed at each catheter site before pace mapping when VT was present. Otherwise, pace mapping was occasionally performed first. Recordings were exclusively bipolar. Multiple fluoroscopic views were used to position the ablation catheter including the posterocoronal view, although no radiographic view was universally useful in all patients. RF energy was delivered through a large-tip deflectable (Polaris) electrode catheter (7F; distal electrode length 4 mm, surface area 27 mm²; Mansfield-Webster catheters, Boston Scientific, Watertown, Mass.) when the earliest site of endocardial activation was identified and a pace map was obtained that showed identical QRS complexes in at least 11 of 12 scalar ECG leads (Figure 1). The ablation catheter tip position was confirmed by using right anterior oblique, left anterior oblique, and anteroposterior radiographic views. RF current (continuous wave, 500 KHz) was generated by a conventional electrosurgical unit (Valley Laboratory, Boulder, Colo.) coupled to a device that provided real-time monitoring of root-mean-square voltage, current, and impedance. RF current was delivered at 40–60 V (usually 50–55 V) between the large-tip catheter electrode and a standard electrosurgical adhesive pad applied to the chest wall. Energy was applied during VT when possible; otherwise, it was delivered during spontaneous rhythm for 30–60 seconds but was terminated immediately when an impedance rise occurred. Programmed electrical stimulation (with and without isoproterenol infusion) was repeated 30 minutes after ablation and again 6 weeks later. Successful ablation was defined as abolition of spontaneous and inducible (premature stimulation and/or isoproterenol) VT.

Signal-averaged ECGs were obtained before the ablation procedure and again 6 weeks later; they were analyzed in the time domain using standard criteria. The two-dimensional echocardiogram/Doppler studies were obtained before ablation and 24 hours after the procedure. Mitral valve prolapse was defined as being present when a portion of the body of the mitral valve leaflet was displaced to the atrial side of a line drawn between the aortic–mitral fibrosa and the mitral posterior wall annulus in the parasternal long-axis view. Right ventricular dysplasia was defined echocardiographically as a dilated right ventricle that was equal to or larger than the left ventricle. Associated findings included areas of hypokinesis or overt thinning. Creatine phosphokinase (CPK) isozymes were obtained on three occasions, immediately after ablation and at 8-hour intervals thereafter. Only patient 4 received heparin during the procedure because the ablation catheter tip was in the left ventricle. All patients received heparin for 24–48 hours after ablation. There were no hemorrhagic complications.

Results

Presenting Arrhythmia

The mean ventricular tachycardia cycle length was 323 ± 54 msec and ranged from 250 to 400 msec. The ventricular tachycardia morphology was that of a left bundle branch block with an inferior axis in 13 patients, left bundle branch block with a rightward axis in two patients, and right bundle branch block with a superior axis in one patient. Except for patients 8 and 16, all patients had failed therapy with either verapamil or β-blockers, including patient 4 (VT originating from the left ventricle), who had had recurrent sustained VT despite oral verapamil therapy. (See Table 1.)

Results of Electrophysiology Study

Eight of the 16 patients presented with sustained VT, seven with nonsustained VT, and one patient with incessant nonsustained VT (Figure 2). Of the eight patients presenting with sustained VT, five had sustained VT at the electrophysiology study. The remaining three patients (patients 1, 8, and 10) had only nonsustained VT at the study. Of the seven patients presenting with nonsustained VT, six had nonsustained VT at the electrophysiology study and one had only PVCs at the study. The one patient (patient 12) with incessant nonsustained VT had this arrhythmia at the electrophysiology study. (See Table 1.)

Response to Isoproterenol

Of the five patients with sustained VT induced at the electrophysiology study, three had sustained VT induced...
Table 1. Characteristics of Patient Population, Presenting and Induced Arrhythmias, Electrophysiological Study Findings, and Ablation Results

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/Sex</th>
<th>VT dur (yr)</th>
<th>Presenting symptom</th>
<th>Previous AAD (n)</th>
<th>VT Morphology</th>
<th>CL (msec)</th>
<th>Location</th>
<th>Presenting</th>
<th>EPS</th>
<th>Induced by PES</th>
<th>EAT (msec)</th>
<th>RFs (n)</th>
<th>RFf</th>
<th>CPK (IU/l)</th>
<th>SAECG</th>
<th>Follow-up dur (mo)</th>
<th>Pre</th>
<th>Post</th>
<th>Success</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>43/F</td>
<td>10</td>
<td>Presync</td>
<td>4</td>
<td>LB/inf</td>
<td>400</td>
<td>RVOT/AS</td>
<td>S</td>
<td>NS</td>
<td>–</td>
<td>+</td>
<td>–</td>
<td>30</td>
<td>13</td>
<td>55</td>
<td>620</td>
<td>180</td>
<td>5</td>
<td>–</td>
</tr>
<tr>
<td>2</td>
<td>28/F</td>
<td>2</td>
<td>Presync</td>
<td>4</td>
<td>LB/inf</td>
<td>370</td>
<td>RVOT/AS</td>
<td>NS</td>
<td>NS</td>
<td>–</td>
<td>+</td>
<td>–</td>
<td>50</td>
<td>2</td>
<td>68</td>
<td>590</td>
<td>94</td>
<td>6</td>
<td>–</td>
</tr>
<tr>
<td>3</td>
<td>36/M</td>
<td>10</td>
<td>Palps</td>
<td>5</td>
<td>LB/inf</td>
<td>350</td>
<td>RVOT/AS</td>
<td>NS</td>
<td>NS</td>
<td>–</td>
<td>+</td>
<td>–</td>
<td>55</td>
<td>5</td>
<td>66</td>
<td>600</td>
<td>253</td>
<td>0</td>
<td>…</td>
</tr>
<tr>
<td>4</td>
<td>39/M</td>
<td>20</td>
<td>Palps</td>
<td>10</td>
<td>RB/sup</td>
<td>380</td>
<td>PS/M-LV</td>
<td>S</td>
<td>S</td>
<td>–</td>
<td>–</td>
<td>+</td>
<td>30</td>
<td>7</td>
<td>52</td>
<td>530</td>
<td>1014</td>
<td>5</td>
<td>…</td>
</tr>
<tr>
<td>5</td>
<td>39/M</td>
<td>0.5</td>
<td>Presync</td>
<td>3</td>
<td>LB/inf</td>
<td>340</td>
<td>RVOT/AS</td>
<td>S</td>
<td>S</td>
<td>–</td>
<td>+</td>
<td>–</td>
<td>45</td>
<td>1</td>
<td>57</td>
<td>510</td>
<td>304</td>
<td>4</td>
<td>+</td>
</tr>
<tr>
<td>6</td>
<td>24/M</td>
<td>0.85</td>
<td>Presync</td>
<td>4</td>
<td>LB/inf</td>
<td>230</td>
<td>RV/HBE</td>
<td>S</td>
<td>S</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>–35</td>
<td>19</td>
<td></td>
<td>680</td>
<td>456</td>
<td>10</td>
<td>–</td>
</tr>
<tr>
<td>7</td>
<td>18/F</td>
<td>6</td>
<td>Palps</td>
<td>0</td>
<td>LB/inf</td>
<td>300</td>
<td>RV/HBE</td>
<td>NS</td>
<td>NS</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–50</td>
<td>18</td>
<td>59</td>
<td>640</td>
<td>125</td>
<td>9</td>
<td>–</td>
</tr>
<tr>
<td>8</td>
<td>35/F</td>
<td>1.5</td>
<td>Syncope</td>
<td>0</td>
<td>LB/inf</td>
<td>280</td>
<td>RVOT/AL</td>
<td>S</td>
<td>NS</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>–40</td>
<td>4</td>
<td>54</td>
<td>560</td>
<td>125</td>
<td>9</td>
<td>–</td>
</tr>
<tr>
<td>9</td>
<td>42/M</td>
<td>4</td>
<td>Presync</td>
<td>2</td>
<td>LB/inf</td>
<td>360</td>
<td>RV/HBE</td>
<td>S</td>
<td>S</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>–50</td>
<td>43</td>
<td>…</td>
<td>700</td>
<td>6</td>
<td>6</td>
<td>–</td>
</tr>
<tr>
<td>10</td>
<td>34/F</td>
<td>2</td>
<td>Presync</td>
<td>1</td>
<td>LB/rad</td>
<td>250</td>
<td>RVOT/AS</td>
<td>S</td>
<td>NS</td>
<td>–</td>
<td>+</td>
<td>–</td>
<td>–35</td>
<td>6</td>
<td>52</td>
<td>550</td>
<td>65</td>
<td>6</td>
<td>–</td>
</tr>
<tr>
<td>11</td>
<td>44/M</td>
<td>3</td>
<td>Presync</td>
<td>2</td>
<td>LB/inf</td>
<td>300</td>
<td>RVOT/ANT</td>
<td>NS</td>
<td>NS</td>
<td>–</td>
<td>+</td>
<td>–</td>
<td>–40</td>
<td>28</td>
<td>50</td>
<td>580</td>
<td>157</td>
<td>7</td>
<td>+</td>
</tr>
<tr>
<td>12</td>
<td>47/F</td>
<td>8</td>
<td>Presync</td>
<td>5</td>
<td>LB/rad</td>
<td>400</td>
<td>RVOT/NS</td>
<td>NS-I</td>
<td>NS-I</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–35</td>
<td>1</td>
<td>51</td>
<td>480</td>
<td>37</td>
<td>0</td>
<td>(+)</td>
</tr>
<tr>
<td>13</td>
<td>43/F</td>
<td>2</td>
<td>Presync</td>
<td>3</td>
<td>LB/inf</td>
<td>280</td>
<td>RVOT/AS</td>
<td>NS</td>
<td>NS</td>
<td>–</td>
<td>+</td>
<td>–</td>
<td>–40</td>
<td>3</td>
<td>57</td>
<td>620</td>
<td>94</td>
<td>6</td>
<td>–</td>
</tr>
<tr>
<td>14</td>
<td>35/F</td>
<td>7</td>
<td>Syncope</td>
<td>4</td>
<td>LB/inf</td>
<td>330</td>
<td>RVOT/AS</td>
<td>S</td>
<td>S</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>–65</td>
<td>3</td>
<td>64</td>
<td>620</td>
<td>142</td>
<td>9</td>
<td>–</td>
</tr>
<tr>
<td>15</td>
<td>55/F</td>
<td>2</td>
<td>Palps</td>
<td>1</td>
<td>LB/inf</td>
<td>…</td>
<td>RVOT/NS</td>
<td>NS</td>
<td>PVCs</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–10</td>
<td>2</td>
<td>61</td>
<td>650</td>
<td>120</td>
<td>0</td>
<td>…</td>
</tr>
<tr>
<td>16</td>
<td>40/M</td>
<td>15</td>
<td>Presync</td>
<td>0</td>
<td>LB/inf</td>
<td>270</td>
<td>RVOT/NS</td>
<td>NS</td>
<td>NS</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–20</td>
<td>1</td>
<td>60</td>
<td>500</td>
<td>63</td>
<td>0</td>
<td>–</td>
</tr>
<tr>
<td>Mean</td>
<td>38 yr</td>
<td>5.9 yr</td>
<td>Presync, 10</td>
<td>3.0</td>
<td>LB/inf, 13</td>
<td>323</td>
<td>RVOT/AS, 10</td>
<td>S, 8</td>
<td>S, 5</td>
<td>5 mSec</td>
<td>9</td>
<td>8.1</td>
<td>58 V</td>
<td>581 mA</td>
<td>256 IU/l</td>
<td>5%</td>
<td>10.8</td>
<td>15/16</td>
<td></td>
</tr>
<tr>
<td>±SD</td>
<td>9 yr</td>
<td>5.6 yr</td>
<td></td>
<td>2.6</td>
<td>54</td>
<td>14</td>
<td>11.0</td>
<td>6</td>
<td>62</td>
<td>265 IU/l</td>
<td>3</td>
<td>3.6</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

VT: ventricular tachycardia; dur, duration; AAD, antiarrhythmic drugs; CL, cycle length; Spont, spontaneous; EPS, induced at electrophysiology study; ISO, isoproterenol: was (+) or was not (–) needed to enhance VT at electrophysiology study; EAT, endocardial activation time; RFs, radiofrequency burns delivered in an individual patient to eliminate VT; RFf, radiofrequency voltage delivered at successful ablation site; RFf, radiofrequency current delivered at successful ablation site; CPK, creatine phosphokinase; SAECG, signal-averaged electrocardiogram (Pre, before ablation; Post, after ablation); F, female; M, male; Presync, presyncopy; Palps, palpitations; LB, left bundle branch block morphology; sup, superior axis; rad, right axis deviation; RVOT, right ventricular outflow tract; AS, anteroseptal; M-LV, mid-left ventricle; RV, right ventricular; HBE, His bundle electrogram; AL, anterolateral; ANT, anterior; S, sustained; NS, nonsustained; NS-I, incessant nonsustained; PVCs, premature ventricular contractions; PES, programmed electrical stimulation; –, not inducible; +, inducible.

*Mechanism of successful ablation in this patient is not known, although presumably one of the burns subsequently resulted in scar formation that eliminated VT. It is not clear which of the burns was (were) responsible for successful ablation.

†Sustained VT was inducible immediately after ablation but not 6 weeks later despite sustained VT having been induced at all five of this patient’s previous electrophysiology studies. There has been no spontaneous recurrence of VT in this patient.

‡Although endocardial electrograms were recorded that preceded the QRS complexes during VT by 50 msec, catheter contact with endocardium was poor and was presumably responsible, at least in part, for failure of radiofrequency energy to eliminate VT.

§This patient was thought to have had successful ablation after his first study, but VT recurred 1 week later and a repeat ablation procedure was performed. Endocardial activation was found 5 msec earlier at his second study than his first study (although no difference in the pace map from the two sites could be discerned) and VT was eliminated at the second radiofrequency session.
with programmed ventricular stimulation in the absence of isoproterenol infusion, one had sustained VT induced during isoproterenol infusion, and the fifth patient had sustained VT occur spontaneously during isoproterenol infusion. Sustained VT was not inducible in the remaining 11 patients. As noted above, one of these 11 had incessant nonsustained VT (patient 12) and another also had no VT inducible (PVCs occurred spontaneously; patient 15). Of the remaining nine patients in whom only nonsustained VT was induced at electrophysiology study, isoproterenol enhanced the frequency of nonsustained VT in seven of these and had no effect on the arrhythmia in the remaining two patients. (See Table 1.)

**Ventricular Tachycardia Mapping Data**

Mapping the ventricular endocardium revealed that endocardial activation was earliest in the high right ventricular outflow tract in 12 patients (anteroseptal in 10, anterolateral in one, and anterior in one), at the right ventricular inflow tract near the His bundle (close enough to record a His potential with bipolar recordings) in three patients, and at a posteroseptal mid–left ventricular site in one patient. The ECG during VT in all three patients with right ventricular inflow tract foci (patients 6, 7, and 9) had the morphology of a left bundle branch block and an inferior axis; the ECG during VT did not predict the successful ablation site.

Activation maps were used to guide the positioning of the ablating catheter tip. The earliest endocardial activation in the 15 successful ablations was 39±14 msec (range, 10–65 msec) before the rapid onset of the QRS complex during VT. Thus, there was not a universal or predictable degree of prematurity of the ventricular electrogram predicting successful ablation. The earliest point found in the one patient whose VT was not ablated (patient 9) was 50 msec before the onset of the QRS complex. There was no correlation between early endocardial activation and inducibility of VT at the electrophysiology study. Furthermore, endocardial activation at the successful ablation site was always earlier than that at unsuccessful sites in patients requiring more than one RF pulse. (See Table 1.)

**Success Rate**

VT was eliminated in all patients whose arrhythmia arose from the right ventricular outflow tract (either anteroseptal, anterior, or anterolateral locations) (Figures 2 and 3A). Similarly, VT in the one patient with VT arising from a posteroseptal mid–left ventricular region (Figure 3C) was successfully ablated. Of the three VTs that arose from a region adjacent to the His bundle, two were ultimately successfully ablated (Figures 3B and 4–6). In patient 9, VT remained inducible and recurred spontaneously despite multiple ablation attempts. This patient was re-treated with flecainide with suppression of spontaneous VT and has since remained asymptomatic. Another patient with apparent ablation failure (patient 6) ultimately had no inducible VT at follow-up study and no spontaneous VT (see “Follow-up”). Patient 11 required two sessions to eliminate his VT (Table 1). Thus, VT was eliminated with catheter ablation by using RF energy in 15 of 16 patients, a 94% success rate. There were no complications in any of the 16 patients.

**Radiofrequency Energy Delivery**

The mean RF voltage in this series was 58±6 V and the mean current was 581±62 mA, giving a mean power of 34 W. (See Table 1.) The mean number of RF pulses delivered in the 15 successful ablations was 5.8±6, although the overall mean was 8.1±11 pulses (the patient in whom RF energy failed to eliminate VT received 43 pulses before termination of the procedure). A median of three pulses was required for successful elimination of VT in this series, possibly a more meaningful number in view of the skewed number of pulses required in the overall series. The mean duration of RF energy application before elimination of the marker arrhythmia (VT or PVCs) could be adequately assessed in six patients and averaged 8 seconds, ranging from 3 to 30 seconds. Importantly, the ablation was never successful if there was inconsistent capture during high-output pacing, probably reflecting poor catheter contact with endocardium (Figure 5).

**Cardiac Enzymes**

Among the 16 patients, the mean total CPK rise was 256±265 units, CPK-MB rise 5±3%, and CPK-MB total 18±8 units (reference range, <180 IU/l, <5%, and <9 IU/l, respectively). The maximal CPK rise occurred 8–12 hours after the ablation procedure.

**Signal-Averaged ECGs**

Ten patients had signal-averaged ECGs before and after the ablation procedure. Eight were consistently
negative and two (patients 5 and 11) were consistently positive for late potentials. Thus, no patient gained or lost late potentials because of the ablation. The presence of late potentials did not correlate with the site of origin of VT. The patient in whom RF failed to eliminate VT did not have late potentials.

**Echocardiograms**

Postprocedure transthoracic echocardiograms and Doppler studies (compared with those obtained before the ablation procedure) were unchanged in all 16 patients. No patient had evidence of right ventricular dysfunction or dysplasia. Left ventricular function was normal in all patients. Patient 3 had a thickened mitral valve without mitral valve prolapse. Patient 14 had mitral valve prolapse with a thickened mitral valve and mild mitral regurgitation.

**Follow-up**

Patients were followed for a mean of 10.8±3.6 months (range, 0.5–13 months). Over the follow-up period, 14 of the 16 patients have not had spontaneous VT, nor did they have inducible VT at follow-up study (30 minutes after the procedure and again 6 weeks later). Two patients (patients 6 and 9) had inducible VT 30 minutes after ablation. Of these two, patient 9 had recurrent sustained VT 4 weeks after the procedure and was, as previously noted, restarted on antiarrhythmic medication, and his

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** Tracings from patient 12, who presented with incessant nonsustained ventricular tachycardia. Top tracing is representative of spontaneous arrhythmia before ablation. After ablation, as displayed on the lower tracing, all spontaneous ventricular arrhythmia was eliminated.

![Figure 3](http://circ.ahajournals.org/)

**Figure 3.** Panel A: Left anterior oblique radiograph of catheter position at successful ablation site in patient 2, whose ventricular tachycardia arose from an anteroseptal region in the right ventricular outflow tract. Arrow points to the electrode used for successful ablation. Panel B: Right anterior oblique radiograph of catheter position at successful ablation site (patient 7). The patient’s ventricular tachycardia arose from an anteroseptal region just across the tricuspid valve and adjacent to the His bundle. Tip of the ablation catheter is shown (arrow), with a sharp distal loop, adjacent to the His bundle catheter; tip of this catheter was across the tricuspid valve and in the right ventricle. Panel C: Right anterior oblique radiograph of ablation catheter in the left ventricle (patient 4). Ablating tip (arrow) is in a posteroseptal region at the mid–left ventricular level; this was the successful ablation site.
arrhythmia was suppressed. Patient 6 had sustained VT inducible 30 minutes after ablation but did not have sustained VT inducible 6 weeks later despite having had sustained VT inducible at all five of his previous electrophysiology studies. This patient has not had recurrence of VT since his ablation procedure despite receiving no antiarrhythmic drug therapy. His VT is believed to have been successfully ablated. Patient 16 had PVCs (but not VT) on an ambulatory ECG recording 4 weeks after ablation. He has occasional palpitations, but his episodes of presyncope were entirely eliminated.

Discussion

New Observations

The findings described in this report demonstrate that VT in patients without structural heart disease (idiopathic VT) can be safely eliminated with RF energy delivered through a catheter with a high degree of efficacy. It did not matter whether the patient presented with palpitations, presyncope, or syncope. Furthermore, RF catheter ablation successfully eliminated VTs from several locations, including three separate regions in the right ventricular outflow tract, the right ventricular inflow tract, and in one patient, from a site in the left ventricle. Although the follow-up duration is relatively short, the frequency with which the patients in this study had spontaneous VT suggests that an early cure in these patients is likely to be a long-term cure as well. Importantly, the success rate was not affected by whether VT at electrophysiology study was sustained, nonsustained, or even if only PVCs of the same ECG morphology as the VT could be targeted for ablation. In addition, no proarrhythmic effect of RF energy delivery could be identified by programmed ventricular stimulation or signal-averaged ECG 6 weeks after ablation, and no complications occurred from the ablation procedure. Myocardial enzyme rise was minimal, and no wall motion abnormalities resulted.

Technical Considerations

We did not find it necessary in these patients to identify an area of slow conduction by trying to find regions of entrainment or concealed entrainment. 

Furthermore, fractionated electrograms or mid-diastolic potentials were not identified in any of the patients and thus were not required for successful ablation. Interestingly, the site of earliest endocardial activation time was uniformly also the site from which we obtained the best pace map. Accurate assessment of catheter contact with the endocardial wall of the heart was very difficult; electrogram size and pacing

![Figure 4](http://circ.ahajournals.org/)

**Figure 4.** Shown are a 12-lead ECG of premature ventricular contractions (PVCs) (top tracing) and a pace map from the successful ablation site (patient 7), showing nearly identical morphology of the paced QRS complexes and the PVCs. Ventricular tachycardia gradually subsided during the study, but PVCs having the same morphology as the ventricular tachycardia occurred repeatedly and were used for mapping.

![Figure 5](http://circ.ahajournals.org/)

**Figure 5.** Intracardiac recordings from patient 7. Shown are surface ECG leads I, II, III, and V1, and intracardiac recordings from the high right atrium (HRA), His bundle region (HBE), right ventricular apex (RV apex), and bipolar recordings from the proximal poles (MAPp) and from the distal poles (MAPd) of the quadripolar ablation catheter. This figure illustrates the importance of catheter contact with endocardium: On the left, an early electrogram (arrow) is recorded from the site of a good pace map, but delivery of radiofrequency energy in this region failed to eliminate spontaneous ventricular arrhythmia; on the right, an equally early electrogram is recorded from this same region. Catheter contact may be better here as evidenced by the larger amplitude of the early portion of the electrogram from this region (same recording gain). Delivery of radiofrequency energy here eliminated ventricular tachycardia. (Electrograms are redrawn for clarity.)
threshold are poor measures of catheter contact. However, as best we could determine, in addition to identifying early endocardial activation and a pace map resulting in QRS complexes that were identical in at least 11 (preferably 12) leads during pacing and VT, contact of the catheter tip with endocardium was the only other predictor of success in this series (Figure 5).36 The findings in patient 6 were instructive. Although the initial ablation attempt failed to eliminate the VT, VT was no longer inducible 6 weeks later and did not recur during follow-up. We cannot exclude the possibility that this patient’s VT may have spontaneously subsided. However, a more likely explanation is that scar tissue caused by the multiple RF burns may have interrupted a critical portion of the VT circuit leading ultimately to VT elimination.

The ablation attempts were often complicated by the fact that VT, previously frequent or sustained, was nonsustained or infrequent at the time of ablation. Eight patients presented with spontaneous sustained VT, but only five had inducible or spontaneous sustained VT at the time of ablation. In five patients (patients 7, 10, 14, 15, and 16), PVCs were the target arrhythmia at the ablation session (Figure 4). Sedation used at the time of study, catheter trauma, or other factors may have affected the VT prevalence. Fortunately, these “fragments of the arrhythmia” (nonsustained VT or PVCs) were suitable targets for ablation in these patients and did not hinder success, but they did prolong the duration of the procedure, potentially adding a small risk.

Implications for Ventricular Tachycardia Mechanism

The mechanism responsible for VT in patients without structural heart disease is not known and may be multiple.21–25,37,38 Importantly, however, it has been previously demonstrated that the presumed mechanism of VT did not alter the ablation success when direct-current energy was used.25 In the present study, attempts to delineate VT mechanism were confounded by many spontaneous terminations of sustained VT and the frequent occurrence of only nonsustained VT at the time of electrophysiology study. The heterogeneity of the presenting arrhythmias and induction protocols required for initiation of VT at the ablation sessions strongly suggests that different mechanisms of VT were operative in our patient population and that RF energy, like direct-current energy,25 delivered through a catheter, can potentially eliminate VT of any mechanism in a patient without structural heart disease.

We were able to make observations that may impact on an understanding of the mechanisms, however. Earliest endocardial activation times (mean, 39 msec before the onset of the earliest QRS) were considerably later than those found in patients with sustained VT caused by coronary artery disease (50–100 msec).13–15 This, along with the predictive accuracy for ablation success of activation mapping and pace mapping (an often unreliable predictor of ablation success in patients with structural heart disease)16,35 suggests that, unlike in patients with coronary artery disease, VT arising in patients with structurally normal hearts either may not be as critically dependent on a region of slow conduction, or the regions of slow conduction and endocardial breakthrough are geographically close or identical.25 Thus, it is likely that the VT arose in or close to the endocardium. It is possible that the RF energy in the thin-walled right ventricle burned a larger percentage of transmural myocardium compared with similar ablation in patients with coronary artery disease and VT arising from the left ventricle. However, the successful ablation in patient 5 (left ventricular VT) and the large number of VTs arising from septal regions of the right ventricle (anteroseptal location) makes this explanation less likely because a large mass of myocardium (the septum) may have been present in the region of VT in these patients. Naturally, one cannot state whether the electrical (and possibly anatomical) abnormality is congenital or acquired.

Limitations

Why a given RF pulse failed to eliminate VT in an individual patient is not identified in this study. One can speculate that the catheter is not at the critical portion of the circuit or that catheter contact is inadequate.36 The one patient in whom RF energy failed to eliminate VT (patient 9) had excellent pace maps and very early endocardial activation times. We presumed that catheter contact was inadequate, but intramyocardial or epicardial origins of his VT or suboptimal catheter location may have been responsible for the failure.

This study does not compare the relative efficacy or safety of RF and direct-current catheter ablation. A
previous study\textsuperscript{25} has suggested high efficacy and safety with the use of direct-current ablation. Our data suggest that similar results may be obtained with RF energy. Potential advantages of using RF energy versus direct-current shocks are lack of need for general anesthesia, ability to safely deliver multiple ablation applications, lack of hemodynamic embarrassment, no damage to the ablation catheter caused by energy delivery, better control of delivered energy, and a more homogeneous lesion (and thus less risk of arrhythmogenicity). The theoretical advantages of RF ablation might make it preferable to direct-current ablation in this patient population; a randomized comparison of the two techniques would be of interest. Energy sources that result in larger or more penetrating lesions (for example, direct-current countershock or microwave energy) may prove necessary for successful VT ablation in patients with structural heart disease.

Conclusions

RF catheter ablation of VT in patients without structural heart disease is feasible and safe. It is our judgment that RF ablation may be considered as early therapy in these patients and perhaps as first-line therapy in selected, highly symptomatic patients.

Addendum

Since this article was submitted, one additional patient has undergone RF catheter ablation for sustained VT. This was a 27-year-old man with normal ventricular function (by cardiac catheterization with right and left ventriculograms and coronary angiograms; a two-dimensional echocardiogram disclosed mild mitral valve prolapse) whose sustained VT occurred principally during exercise. His VT (spontaneous and induced) had the morphology of a left bundle branch block with a superior axis and a cycle length of 310 msec; it was inducible at electrophysiology study with a single ventricular extrastimulus and did not require isoproterenol infusion for induction. His signal-averaged ECG was abnormal both before and after ablation. Local endocardial activation mapped to the right ventricular free wall in a posterolateral location under the tricuspid valve. RF energy eliminated inducible VT. Therefore, this patient emphasizes that right VT can arise from sites other than the right ventricular outflow tract and can be successfully ablated with RF current.

Acknowledgments

The authors are grateful to Rose Ann Casey for expert secretarial support, the staff of the electrophysiology laboratory, David Adams, BSEE, and Gregory Ayers, PhD, for technical support, and Douglas Segar, MD, for his review of all echocardiograms.

References

Radiofrequency catheter ablation of ventricular tachycardia in patients without structural heart disease.
L S Klein, H T Shih, F K Hackett, D P Zipes and W M Miles

Circulation. 1992;85:1666-1674
doi: 10.1161/01.CIR.85.5.1666

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1992 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/85/5/1666

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation 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 is online at:
http://circ.ahajournals.org//subscriptions/