Radiofrequency Catheter Ablation of Ventricular Tachycardia in Patients With Coronary Artery Disease

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**Background.** Radiofrequency (RF) ablation of idiopathic ventricular tachycardia (VT) has been demonstrated to be highly efficacious, but the efficacy of RF ablation of VT in patients with coronary artery disease has been unknown. Therefore, the purpose of this study was to determine the feasibility of RF ablation of VT in patients with coronary artery disease.

**Methods and Results.** Fifteen consecutive patients with coronary artery disease and a history of myocardial infarction underwent an attempt at RF ablation of 16 hemodynamically stable monomorphic VTs that had been documented clinically on a 12-lead ECG and that had not been successfully managed by pharmacological or device therapy. One VT was incessant, five occurred more than 25 times, and the remainder occurred two to 20 times. An additional four VTs that had not been documented clinically also were targeted for ablation. The mean age of the patients was 68±7 years (±SD), and their mean left ventricular ejection fraction was 0.27±0.08. The mean cycle length of the 20 VTs targeted for ablation was 438±82 msec. Ablation sites were selected based on endocardial activation mapping, pace mapping, identification of an isolated mid-diastolic potential, or concealed entrainment. Sixteen of the 20 VTs (80%) were successfully ablated in 11 of 15 patients (73%), using a mean of 4.2±3 applications of RF energy, and no recurrences of the ablated VTs occurred during 9.1±3.3 months of follow-up. The mean duration of the ablation procedures was 128±30 minutes. No complications occurred in any of the patients.

**Conclusions.** The results of this study demonstrate that RF ablation of hemodynamically stable VT is feasible as adjunctive therapy in selected patients with coronary artery disease. (Circulation 1993;87:363–372)

**Key Words** • tachycardia, ventricular • radiofrequency ablation • coronary artery disease

Several studies have demonstrated that radiofrequency (RF) catheter ablation of the atrioventricular junction,1–5 atrioventricular nodal reentrant tachycardia,6–9 and accessory atrioventricular connections10–13 is highly efficacious. In contrast, the published experience with RF ablation of ventricular tachycardia (VT) has been scant and has been limited almost exclusively to ablation of idiopathic VT.14,15 Although catheter ablation of VT with direct current shocks in patients with coronary artery disease has been demonstrated to be feasible,16–19 the myocardial lesion created by RF energy is smaller than the lesion created by direct current shocks,20 and the results obtained with direct current shocks may not apply to RF energy. Therefore, the purpose of this study was to determine the feasibility of RF ablation of VT in patients with coronary artery disease.

**Methods**

**Characteristics of Patients**

The subjects of this study were 15 consecutive patients with coronary artery disease and recurrent monomorphic VT who met the following selection criteria to undergo an attempt at RF ablation at the University of Michigan Medical Center: 1) The VT was documented on a 12-lead ECG on at least two different occasions and found to always have the same configuration. The only exception was a patient who presented on two separate occasions 1 month apart with two types of monomorphic VT. 2) The VT had not been controlled by at least two antiarrhythmic drugs. 3) The VT that had been documented clinically was inducible reproducibly in the electrophysiology laboratory by programmed ventricular stimulation. 4) The VT was hemodynamically stable enough to allow catheter mapping in the electrophysiology laboratory.

The characteristics of the patients and their VTs are described in Table 1. There were 13 men and two women, and their mean age was 68±7 years (±SD). Each patient had a history of at least one myocardial infarction 6 months to 12 years before referral, and each had undergone coronary angiography that demonstrated coronary artery disease. The mean left ventricular ejection fraction as measured by contrast or radionuclide ventriculography was 0.27±0.08. Eight patients had undergone coronary artery bypass surgery, and three had undergone implantation of an internal cardioverter-defibrillator before referral.
The patients' first episode of sustained VT occurred at an interval of 19±16 months before referral (range, 1–72 months). The first onset of the recurrent VT that was targeted for ablation in this study occurred at a mean interval of 5±10 months before referral (Table 1). Among the 16 VTs with which the 15 patients in this study presented, one was incessant, five occurred more than 25 times in the 0.5–3 months before referral, and the remainder occurred two to 20 times. Eight of the VTs had a right bundle branch block configuration, and eight had a left bundle branch block configuration (Table 1). The mean cycle length of the VTs was 466±64 msec.

The symptoms associated with the VTs targeted in this study included palpitations, weakness, and light-headedness or near syncope. Although 10 of the 15 patients had a remote history of cardiac arrest or syncope in association with prior episodes of VT, none of the VTs targeted for ablation in this study caused loss of consciousness.

Two to five antiarrhythmic drugs, including amiodarone in 11 patients, were ineffective in suppressing the VTs targeted for ablation. Amiodarone was effective in two patients but was discontinued because of toxicity. At the time of the ablation procedure, 11 patients were being treated with amiodarone. This therapy was continued during and after the ablation procedure because of its efficacy in suppressing other types of VT that these patients had experienced in the past.

**Electrophysiology Tests**

Electrophysiology tests were performed in the fasting state after informed consent was obtained. The study protocol was approved by the Human Research Committee at the University of Michigan. Three 6F quadripolar electrode catheters were inserted into a femoral vein and positioned in the right atrium. His bundle position, and right ventricle. Programmed ventricular stimulation was performed with a programmable stimulator (Bloom Associates, Ltd., Reading, Pa.) using a current strength twice the diastolic threshold. Leads V1, I, II, and III and the intracardiac electrograms were displayed on an oscilloscope and recorded at a paper speed of 25–100 mm/sec.

One, two, and three ventricular extrastimuli were introduced using basic drive train cycle lengths of 350, 400, and 600 msec first at the right ventricular apex, then at the outflow tract or septum if necessary. Unless the VT was hemodynamically unstable and required immediate termination, a 12-lead ECG was recorded whenever sustained VT was induced. Sustained VT was defined as VT lasting more than 30 seconds or requiring termination by pacing or countershock because of hemodynamic instability. Nonsustained VT was defined as VT six beats to 30 seconds in duration.

**Mapping Technique**

Mapping was performed after demonstrating that the VT that had occurred clinically was reproducibly inducible by programmed stimulation. A 7F quadripolar catheter with a 4-mm distal electrode, 2- or 5-mm interelectrode spacing, and a deflectable tip (Mansfield-Webster, Watertown, Mass.) was used both for mapping and ablation. This catheter was inserted into a femoral artery and passed retrogradely across the aortic valve into the left ventricle. A 5,000-unit bolus of heparin was administered intravenously followed by 1,000 units every hour as long as the catheter was in the left ventricle.

Biplane fluoroscopy was used to identify catheter positions in the left ventricle, and mapping sites were designated according to a scheme described by Josephson et al.21 Bipolar endocardial electrograms were recorded during VT, using filter settings of 50–500 Hz. Bipolar pacing using the distal pair of electrodes of the

### Table 1. Characteristics of Patients and Ventricular Tachycardias

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>LVEF</th>
<th>Onset (mos)*</th>
<th>No. of episodes*</th>
<th>Ventricular tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>63</td>
<td>M</td>
<td>0.25</td>
<td>0.5</td>
<td>15</td>
<td>Right Superior</td>
</tr>
<tr>
<td>2</td>
<td>74</td>
<td>M</td>
<td>0.20</td>
<td>7</td>
<td>5</td>
<td>Left Inferior</td>
</tr>
<tr>
<td>3</td>
<td>74</td>
<td>F</td>
<td>0.40</td>
<td>3</td>
<td>16</td>
<td>Left Superior</td>
</tr>
<tr>
<td>4</td>
<td>71</td>
<td>M</td>
<td>0.22</td>
<td>0.5</td>
<td>10</td>
<td>Left Inferior</td>
</tr>
<tr>
<td>5a†</td>
<td>71</td>
<td>M</td>
<td>0.20</td>
<td>0.5</td>
<td>&gt;25</td>
<td>Left Inferior</td>
</tr>
<tr>
<td>5b</td>
<td></td>
<td></td>
<td></td>
<td>0.25</td>
<td>Incessant</td>
<td>Right Superior</td>
</tr>
<tr>
<td>6</td>
<td>70</td>
<td>M</td>
<td>0.30</td>
<td>3</td>
<td>6</td>
<td>Right Inferior</td>
</tr>
<tr>
<td>7</td>
<td>74</td>
<td>M</td>
<td>0.37</td>
<td>0.5</td>
<td>10</td>
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</tr>
<tr>
<td>8</td>
<td>70</td>
<td>M</td>
<td>0.34</td>
<td>2</td>
<td>6</td>
<td>Left Superior</td>
</tr>
<tr>
<td>9</td>
<td>67</td>
<td>M</td>
<td>0.30</td>
<td>3</td>
<td>&gt;25</td>
<td>Right Superior</td>
</tr>
<tr>
<td>10</td>
<td>68</td>
<td>M</td>
<td>0.19</td>
<td>36</td>
<td>5</td>
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</tr>
<tr>
<td>11</td>
<td>52</td>
<td>F</td>
<td>0.20</td>
<td>0.5</td>
<td>&gt;25</td>
<td>Left Superior</td>
</tr>
<tr>
<td>12</td>
<td>70</td>
<td>M</td>
<td>0.28</td>
<td>24</td>
<td>20</td>
<td>Right Inferior</td>
</tr>
<tr>
<td>13</td>
<td>56</td>
<td>M</td>
<td>0.40</td>
<td>1</td>
<td>2</td>
<td>Right Superior</td>
</tr>
<tr>
<td>14</td>
<td>64</td>
<td>M</td>
<td>0.16</td>
<td>0.5</td>
<td>&gt;25</td>
<td>Left Superior</td>
</tr>
<tr>
<td>15</td>
<td>77</td>
<td>M</td>
<td>0.24</td>
<td>3</td>
<td>&gt;25</td>
<td>Left Superior</td>
</tr>
</tbody>
</table>

LVEF, Left ventricular ejection fraction; BBB, bundle branch block pattern; CL, cycle length.

*Refers to the ventricular tachycardia targeted for ablation.
†This patient had two different types of ventricular tachycardia 1 month apart.
ablation catheter was performed for pace mapping or to look for concealed entrainment. Appropriate target sites for ablation were selected based on endocardial activation mapping,22 identification of an isolated mid-diastolic potential,23 concealed entrainment,19 or pace mapping.24,25 The results of a prior study in which direct current shocks were used to ablate VT in patients with coronary artery disease suggested that the efficacy was similar whether target sites were selected based on the finding of concealed entrainment or based on the other mapping techniques.19 Therefore, in the present study, priority was not given to any particular mapping technique. Ablation was attempted at sites that met the criteria of any one of the mapping techniques.

The following criteria were used to select sites for delivery of RF energy: 1) endocardial activation mapping: local endocardial activation time relative to the onset of the QRS complex during VT of at least −70 msec; 2) isolated mid-diastolic potential: the presence of a discrete potential in diastole during VT, separated by an isoelectric segment from the ventricular electrogram; if an isolated mid-diastolic potential was present, pacing maneuvers usually were performed to confirm that the diastolic potential could not be dissociated from the VT23; however, pacing was not performed if the ablation catheter did not appear to be in a stable position against the myocardium, for fear of dislodging the catheter; 3) concealed entrainment: entrainment of the VT by pacing at the target site at cycle lengths 30–100 msec shorter than the VT cycle length, with a stimulus–QRS interval longer than 90 msec and no change in QRS morphology during entrainment compared with during VT19; 4) pace mapping: very similar or identical QRS morphology during pacing (without evidence of entrainment) and during VT in at least 11 of 12 ECG leads. If none of these criteria was demonstrable, RF energy was applied to sites at which the earliest endocardial activation time or best possible pace map was present.

Each of the 16 VTs that had been documented clinically was mapped. In addition, a VT that had not been documented clinically was also mapped in each of four patients. Ablation of these VTs was attempted because they either were similar in morphology to the clinically documented VT or had a site of origin in close proximity to that of the clinically documented VT.

**Catheter Ablation**

When an appropriate target site was identified, RF energy was delivered as a continuous, unmodulated sine wave at 500 kHz (EP Technologies, Mountain View, Calif.) between the distal electrode of the ablation catheter and a large skin electrode (Valleylab, Boulder, Colo.) on the posterior chest. Applications of RF energy were delivered at a power of 30–35 W. Whenever possible, RF energy was delivered during VT. If the VT did not terminate during the first 10 seconds of the energy application, the application was discontinued, and further attempts to localize an effective target site were made; if the VT did terminate during the first 10 seconds, the energy application was continued for a total of 30 seconds. A second application of 30 seconds was delivered at successful target sites to minimize the possibility of recurrence of VT. In the event of an impedance rise, the ablation catheter was removed from the body, and the distal electrode was wiped clean of coagulum before continuing with the procedure.

The ablation procedure was considered successful acutely when the targeted VT was no longer inducible by programmed stimulation with one to three extrastimuli delivered using basic drive train cycle lengths of 350, 400, and 600 msec at the right ventricular apex and outflow tract or septum.

**Postablation Monitoring**

After the ablation procedure, patients underwent continuous ECG monitoring for 5–10 days. Creatine kinase and its MB fraction were measured every 8 hours during the first 24 hours after the procedure. A two-dimensional echocardiogram was performed 1–3 days after the procedure. An electrophysiology test was repeated 5–10 days after the ablation session, and programmed ventricular stimulation again was performed using one to three extrastimuli and basic drive train cycle lengths of 350, 400, and 600 msec at the right ventricular apex and outflow tract or septum.

**Long-term Follow-up**

The patients were examined every 2–3 months by one of the authors or referring physicians. Pharmacological antiarrhythmic therapy was modified only in the patients in whom the ablation procedure was unsuccessful. A successful long-term clinical result was defined as the absence of any signs or symptoms of the VT that had been ablated. In the three patients who had an internal cardioverter-defibrillator, the rate cutoff of the device was at least 20 beats per minute faster than the rate of the VTs that had been ablated; therefore, device discharges during follow-up were not considered indicative of recurrence of the ablated VTs.

**Statistical Analysis**

Values are expressed as mean±1 SD. Comparisons were performed using Student’s t test. A probability value less than 0.05 was considered significant.

**Results**

**Selection of Ablation Sites**

The 20 VTs that were mapped are described in Table 2. In accord with the selection criteria for patients, the 16 VTs that had occurred clinically in the 15 patients in this study were reproducibly inducible by programmed ventricular stimulation. These 16 VTs were mapped along with an additional four VTs that had not been documented clinically. The mean cycle length of the 20 VTs was 438±82 msec.

The mapping technique used to identify target sites for ablation are described in Table 2. In nine VTs, the ablation site was selected based on an early endocardial activation time (−60 to −150 msec; mean, −105±30 msec; Figure 1). The ablation site in three VTs was identified by the demonstration of concealed entrainment (Figure 2); the stimulus–QRS interval during entrainment of these VTs ranged from 150 to 300 msec (mean, 240±65 msec). In four VTs, the ablation site was identified by the presence of an isolated mid-diastolic potential (Figure 3). In the remaining four VTs, pace mapping was used to select the ablation sites (Figure 4). The number of ECG leads in which there
TABLE 2. Mapping of Targeted Ventricular Tachycardias and Acute Results of Catheter Ablation

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>BBB</th>
<th>Axis</th>
<th>CL (msec)</th>
<th>Mapping method</th>
<th>Ablation site</th>
<th>No. of RF appl.</th>
<th>Successful ablation*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Right</td>
<td>Superior</td>
<td>400</td>
<td>Pace map (11/12)†</td>
<td>4–6</td>
<td>8</td>
<td>Yes</td>
</tr>
<tr>
<td>2</td>
<td>Left</td>
<td>Inferior</td>
<td>400</td>
<td>Endo act (–120 msec)</td>
<td>4–6</td>
<td>2</td>
<td>Yes</td>
</tr>
<tr>
<td>3</td>
<td>Left</td>
<td>Superior</td>
<td>540</td>
<td>Conc entrainment</td>
<td>3–5</td>
<td>2</td>
<td>Yes</td>
</tr>
<tr>
<td>4</td>
<td>Right</td>
<td>Superior</td>
<td>330</td>
<td>Endo act (–125 msec)</td>
<td>5</td>
<td>2</td>
<td>Yes</td>
</tr>
<tr>
<td>5</td>
<td>Left</td>
<td>Inferior</td>
<td>590</td>
<td>Endo act (–125 msec)</td>
<td>5</td>
<td>6</td>
<td>Yes</td>
</tr>
<tr>
<td>6</td>
<td>Right</td>
<td>Superior</td>
<td>460</td>
<td>Endo act (–150 msec)</td>
<td>3–5</td>
<td>2</td>
<td>Yes</td>
</tr>
<tr>
<td>7</td>
<td>Right</td>
<td>Inferior</td>
<td>440</td>
<td>Endo act (–100 msec)</td>
<td>7–8</td>
<td>6</td>
<td>Yes</td>
</tr>
<tr>
<td>8</td>
<td>Right</td>
<td>Inferior</td>
<td>540</td>
<td>Conc entrainment</td>
<td>10</td>
<td>3</td>
<td>Yes</td>
</tr>
<tr>
<td>9</td>
<td>Right</td>
<td>Superior</td>
<td>350</td>
<td>Pace map (10/12)</td>
<td>6</td>
<td>5</td>
<td>No</td>
</tr>
<tr>
<td>10</td>
<td>Right</td>
<td>Inferior</td>
<td>500</td>
<td>Pace map (9/12)</td>
<td>10</td>
<td>3</td>
<td>No</td>
</tr>
<tr>
<td>11</td>
<td>Left</td>
<td>Superior</td>
<td>500</td>
<td>IMDP</td>
<td>2</td>
<td>6</td>
<td>Yes</td>
</tr>
<tr>
<td>12</td>
<td>Right</td>
<td>Inferior</td>
<td>480</td>
<td>Pace map (11/12)</td>
<td>11</td>
<td>5</td>
<td>Yes</td>
</tr>
<tr>
<td>13</td>
<td>Right</td>
<td>Superior</td>
<td>540</td>
<td>Conc entrainment</td>
<td>11–12</td>
<td>2</td>
<td>Yes</td>
</tr>
<tr>
<td>14</td>
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<td>Superior</td>
<td>320</td>
<td>IMDP</td>
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<td>2</td>
<td>Yes</td>
</tr>
<tr>
<td>15</td>
<td>Left</td>
<td>Superior</td>
<td>350</td>
<td>Endo act (–120 msec)</td>
<td>6–8</td>
<td>5</td>
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<tr>
<td>16</td>
<td>Left</td>
<td>Superior</td>
<td>420</td>
<td>Endo act (–75 msec)</td>
<td>6–8</td>
<td>2</td>
<td>Yes</td>
</tr>
</tbody>
</table>

BBB, Bundle branch block pattern; CL, cycle length; RF appl., radiofrequency application; endo act, endocardial activation mapping (endocardial activation time relative to earliest onset of QRS complex is shown in parentheses); conc, concealed; IMDP, isolated middiastolic potential.

*Defined as the inability to induce the targeted ventricular tachycardia (VT) by the end of the ablation session.

†The pace map score (in parentheses) refers to the number of ECG leads in which the QRS complexes during pacing and VT were very similar or identical.

‡This VT was ablated by the same application of radiofrequency energy that ablated the patient’s other VT.

were identical QRS morphologies during pacing and VT varied from nine to 11.

Acute Results of Ablation

Seventeen of 20 VTs (85%) targeted for ablation were no longer inducible by programmed ventricular stimulation at the end of the ablation session. Three VTs that had been documented clinically in three patients were not successfully ablated. Therefore, in 12 of 15 patients (80%), the ablation procedure was successful acutely. See Table 2 for acute results.

The mean number of RF applications per VT was $4.2 \pm 3$ (range, 2–15). The mean number of RF applications for the VTs that were successfully ablated acutely, $3.6 \pm 2$, was significantly less than for the VTs that were not successfully ablated ($7.2 \pm 6.4$, $p<0.05$). In 12 of the 17 VTs in which the acute outcome was successful, RF energy was delivered during VT, and in each case, the VT terminated within 1–8 seconds of the onset of the RF application. In one of the three VTs that were not successfully ablated, the VT terminated 5 seconds after the onset of an RF application; however, the VT was still inducible by programmed stimulation.

The ablation procedure was successful acutely for seven of seven VTs in which an isolated middiastolic potential or concealed entrainment identified the target site, four of five VTs in which the target site was selected based on the endocardial activation time, and two of four VTs in which selection of the target site was based on pace mapping. For the three VTs that were not successfully ablated acutely, no target sites could be found with an endocardial activation time during VT of earlier than $–70$ msec, an isolated middiastolic potential, concealed entrainment, or at which the pace map demonstrated a QRS configuration identical to the QRS during VT in more than nine or 10 of 12 leads of the ECG.

In patient 13 (Table 2), two VTs with a different configuration both were ablated by the same RF application at a site on the left ventricular septum. An isolated middiastolic potential was present at this site during both of the VTs.

The mean total duration of the ablation procedures, including insertion of sheaths and catheters plus mapping and ablation of VT, was $128 \pm 30$ minutes (range, 90–190 minutes).

Complications

The mean peak plasma creatine kinase concentration after the ablation procedure was $151 \pm 86$ IU/l (range, 20–290 IU/l; normal range, 30–240 IU/l), and the mean peak creatine kinase MB concentration was $6.5 \pm 5$ IU/l (range, 0–16 IU/l; normal range, 0–10 IU/l).

No complications occurred in the patients in this study. There were no episodes of ventricular fibrillation or polymorphic VT during the continuous ECG monitoring that the patients underwent for 5–10 days after the ablation procedures. Echocardiograms performed
after the ablation procedure did not demonstrate any new abnormalities attributable to the procedure.

**Follow-up Electrophysiological Testing**

Among the 12 patients who had a successful outcome acutely, the VT that had been targeted for ablation was again inducible in one patient (No. 2) during electrophysiological testing 5–10 days after the ablation procedure (see Table 3). The VTs that had been targeted for ablation were not inducible in 11 of the 12 patients. No VT could be induced in five of these 11 patients, and one or two types of monomorphic VT that either had never been documented clinically or had occurred infrequently clinically were induced in six.

**Long-term Follow-up**

The four patients in whom the outcome of ablation was unsuccessful were treated with amiodarone, quin-
dine, amiodarone plus quinidine, or by subendocardial resection. No recurrences of VT occurred during 10.8±1.5 months of follow-up.

The 11 patients in whom the targeted VTs were successfully ablated were treated with the same medications that they had been taking at the time of the ablation procedure. No recurrences of the ablated VT occurred during a mean of 9.1±3.3 months of follow-up (range, 4–13 months). Two of the three patients with an internal cardioverter-defibrillator have experienced infrequent discharges from the device.

**Discussion**

**Main Findings**

In a selected group of 15 patients with coronary artery disease and recurrent monomorphic VT, RF ablation of the VT was effective in 73% of patients. Among the 20 VTs targeted for ablation in these 15 patients, 16 VTs (80%) neither were inducible by programmed ventricular stimulation 5–10 days after ablation nor occurred spontaneously during follow-up.

The mean duration of the ablation procedures was approximately 2 hours, and no complications were encountered. Therefore, the results of this study demonstrate that RF ablation of VT is feasible in selected patients with coronary artery disease.

**Limited Role of RF Ablation of VT**

It should be noted that the patients in this report represent less than 10% of the cohort of patients with coronary artery disease who were referred for management after experiencing a cardiac arrest or VT during the time frame of this study. Therefore, the results should not be construed as representative of the success rate that might be expected in unselected patients with coronary artery disease and VT. The patients in this study represented a small subgroup of patients with recurrent VT who had only one or two types of monomorphic VT, which in most cases occurred frequently and could not be managed successfully by pharmacological therapy or an internal cardioverter-defibrillator. For example, 10 of the 15 patients had experienced...

**FIGURE 2.** Traces of radiofrequency (RF) ablation of ventricular tachycardia (VT) guided by concealed entrainment (patient 6). Panel A: 12-lead ECG of the VT induced in the electrophysiology laboratory. The VT had a right bundle branch block configuration and a cycle length of 540 msec and was identical in configuration to the VT that had been occurring spontaneously. Panel B: Pacing at a cycle length (CL) of 460 msec in the left ventricle (LV) at site 10 (posterolateral wall) during VT resulted in concealed entrainment. The pacing stimuli (S) resulted in acceleration of the VT to the pacing cycle length. The stimulus–QRS interval was 300 msec, and there was no change in the QRS configuration in leads V1 or I. RV, right ventricle. Panel C: 12-lead ECG recorded six leads at a time. The last four stimuli of a pacing train (CL, 440 msec) introduced at site 10 during VT demonstrated concealed entrainment and are shown. Note the long interval between the last pacing stimulus (S) and the last entrained QRS complex (arrow). The QRS complexes during pacing and during VT are identical in each of the 12 leads. Panel D: VT terminated approximately 3 seconds after the onset of an RF application (35 W) at site 10. The patient had undergone implantation of a permanent pacemaker several years earlier, and a ventricular paced rhythm is seen after termination of the VT. After ablation, the VT was no longer inducible by programmed stimulation.
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Incessant VT or 10 or more episodes of sustained VT during the 1-week to 3-month period before the ablation procedure. In this small but difficult-to-manage subgroup of patients with coronary artery disease and VT, RF ablation was often effective in preventing recurrences of the VTs that had been causing the clinical problem.

It is also noteworthy that only two of the 15 patients in this series were discharged from the hospital without antiarrhythmic drug therapy. This reflects the fact that patients with coronary artery disease and VT often have multiple types of VT and that catheter ablation usually was directed only at the particular VTs that were not successfully managed by drug or device therapy. In contrast to the curative role of RF ablation in patients with atrioventricular nodal reentrant tachycardia,6-9 the Wolff-Parkinson-White syndrome,10-13 or idiopathic VT,1,5 the role of RF ablation in patients with coronary artery disease and VT may more often be one of adjunctive therapy in combination with other forms of therapy that have been partially effective.
VT Pacing

VT Pacing

FIGURE 4. Pace mapping (patient 12): The ventricular tachycardia (VT) induced in the electrophysiology laboratory had a right bundle branch block configuration and a cycle length of 480 msec. Pacing in the left ventricle at site 11 (anterior wall) at a cycle length of 400 msec resulted in QRS complexes very similar or identical to the QRS complexes during VT in all leads except V4 (pace map score, 11/12). After a 35-W application of radiofrequency energy at this site, the VT could no longer be induced by programmed ventricular stimulation.

Mapping Techniques

The various mapping techniques used to select appropriate target sites for ablation of VT may identify different sites for ablation of a particular VT. For example, the sites at which an isolated middiastolic potential or concealed entrainment are demonstrated during VT usually are not the same sites at which the earliest endocardial activation time or best pace map are found, and vice versa.19,26,27 However, the results of a prior study from our laboratory suggest that these varying mapping techniques may be equally effective in identifying successful ablation sites, at least when direct

TABLE 3. Results of Programmed Ventricular Stimulation 5–10 Days After Ablation and Long-term Outcome in Patients Who Had a Successful Result Acutely

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Targeted VT</th>
<th>BBB</th>
<th>Axis</th>
<th>CL (msec)</th>
<th>Therapy</th>
<th>Months of follow-up</th>
<th>Recurrence of ablated VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No</td>
<td>...</td>
<td>None</td>
<td>...</td>
<td>Amio</td>
<td>13</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>Yes</td>
<td>...</td>
<td>None</td>
<td>...</td>
<td>Amio, quin</td>
<td>12</td>
<td>No</td>
</tr>
<tr>
<td>3</td>
<td>No</td>
<td>...</td>
<td>None</td>
<td>...</td>
<td>Amio</td>
<td>12</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>No</td>
<td>Left</td>
<td>Superior</td>
<td>400</td>
<td>Amio, mex</td>
<td>12</td>
<td>No</td>
</tr>
<tr>
<td>5</td>
<td>No</td>
<td>Left</td>
<td>Inferior</td>
<td>360</td>
<td>Amio, ICD</td>
<td>12</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>No</td>
<td>Right</td>
<td>Superior</td>
<td>350</td>
<td>Amio</td>
<td>12</td>
<td>No</td>
</tr>
<tr>
<td>8</td>
<td>No</td>
<td>Left</td>
<td>Inferior</td>
<td>580</td>
<td>Amio</td>
<td>9</td>
<td>No</td>
</tr>
<tr>
<td>11</td>
<td>No</td>
<td>Right</td>
<td>Superior</td>
<td>320</td>
<td>Amio, ICD</td>
<td>9</td>
<td>No</td>
</tr>
<tr>
<td>12</td>
<td>No</td>
<td>...</td>
<td>None</td>
<td>...</td>
<td>None</td>
<td>8</td>
<td>No</td>
</tr>
<tr>
<td>13</td>
<td>No</td>
<td>...</td>
<td>None</td>
<td>...</td>
<td>None</td>
<td>6</td>
<td>No</td>
</tr>
<tr>
<td>14</td>
<td>No</td>
<td>Right</td>
<td>Superior</td>
<td>380</td>
<td>Amio</td>
<td>4</td>
<td>No</td>
</tr>
<tr>
<td>15</td>
<td>No</td>
<td>Right</td>
<td>Superior</td>
<td>300</td>
<td>Amio, ICD</td>
<td>4</td>
<td>No</td>
</tr>
</tbody>
</table>

VT, Ventricular tachycardia; PVS, programmed ventricular stimulation; BBB, bundle branch block configuration; CL, cycle length; amio, amiodarone; quin, quinidine; mex, mexiletene; ICD, internal cardioverter-defibrillator.
current shocks are used for ablation. Therefore, in the present study, a practical approach to mapping was adopted in which RF energy was delivered at sites that met the criteria of any of four mapping techniques. Mapping was performed only with the ablation catheter, and no effort was made to obtain complete mapping data before attempting ablation. This practical approach to mapping was found to be efficient, with most of the VTs being successfully ablated by only two to six applications of RF energy.

Safety of RF Ablation of VT

There was minimal or no rise in the creatine kinase MB fraction after ablation in the patients in this study. Minimal elevation of the MB fraction also was found after RF ablation of idiopathic VT. In contrast, direct current ablation of VT uniformly results in a substantial rise in the MB fraction. The lesser degree of elevation of the MB fraction after RF ablation may reflect a smaller lesion size and a lesser degree of myocardial necrosis with RF energy compared with direct current shocks. However, Haines et al reported that RF energy may result in thermal degradation of creatine kinase, and this also may explain the lesser degree of elevation in the MB fraction after RF ablation compared with direct current ablation.

No acute proarrhythmic complications of RF ablation occurred in this study. Similarly, there was no short-term evidence of proarrhythmia after RF ablation of idiopathic VT. The results of a prior study suggest that direct current ablation of idiopathic VT may be free of long-term proarrhythmic complications; however, the long-term safety of RF ablation of VT remains to be determined.

Comparison With Ablation of Idiopathic VT

Radiofrequency ablation of idiopathic VT recently was reported to be successful in 94% of patients. The lower success rate of 73% in the present study suggests that VT that occurs in the setting of coronary artery disease may be less amenable to catheter ablation than when it is idiopathic. The presence of scar tissue, a large reentry circuit, or an intramural or epicardial location of the reentry circuit could potentially contribute to diminishing the probability of a successful outcome when VT ablation is attempted in patients with coronary artery disease. However, in the patients in this study in whom the procedure was ineffective, the reason for failure also may simply have been inadequate localization of an appropriate target site. In any case, although the efficacy of RF ablation of VT in patients with coronary artery disease may be lower than in patients with idiopathic VT, the success rate of 73% and the apparent safety of RF ablation suggest that an attempt at RF ablation may be worthwhile in selected patients with coronary artery disease.

Comparison With Direct Current Ablation of VT

Using the same mapping techniques used in the present study, the efficacy of direct current ablation of VT in patients with coronary artery disease was found to be 60%, which is similar to the efficacy of RF ablation in the present study. Therefore, although the myocardial lesion created by direct current shocks is significantly larger than the lesion created by RF energy, the efficacy of catheter ablation of VT in patients with coronary artery disease appears to be similar with both types of energy.

Although the efficacy of direct current and RF ablation of VT may be comparable, the need for general anesthesia and the potential for barotraumatic complications with direct current shocks suggest that RF energy may be preferable. However, a prospective study will be necessary to determine the relative efficacy and safety of direct current and RF ablation of VT.

Shared Zones of Slow Conduction

Fitzgerald et al demonstrated that two or three VTs could share a common zone of slow conduction, as evidenced by the presence of an isolated middiastolic potential at the same left ventricular site during each of the morphologically distinct VTs. This observation was confirmed in a patient in this study in whom two types of monomorphic VT were successfully ablated by a single application of RF energy to a site at which an isolated middiastolic potential was present during both VTs.

Limitations

Four different mapping techniques were used in this study but not in a randomized or controlled fashion. Therefore, this study provides no data regarding the relative value of endocardial activation mapping, pace mapping, identification of an isolated middiastolic potential, or concealed entrainment in correctly identifying effective target sites for ablation of VT.

Most of the VTs targeted for ablation in this study had been documented by 12-lead ECGs to occur spontaneously and therefore were known to be of clinical importance. However, four of the 20 VTs targeted for ablation had never been documented to occur spontaneously, and therefore their clinical significance was unknown. Although these four VTs appear to have been successfully ablated, it is possible that they never would have occurred spontaneously even if they had not been ablated. Therefore, these four VTs may have inaccurately inflated the apparent success rate of RF ablation to 80%. However, even if the analysis of results is limited to the 16 clinically documented VTs, the success rate was 75%.

Conclusions

The presence of multiple types of VT, the absence of documentation by a 12-lead ECG of all of the VTs that occurred spontaneously, the inability to induce the particular VTs that were clinically important, difficulty in mapping VTs that cause hemodynamic collapse, and a reluctance to rely on catheter ablation as the sole form of therapy for life-threatening VT are all factors that contribute to markedly restricting the role of catheter ablation in the management of patients with coronary artery disease and VT. Nevertheless, this study demonstrates that RF ablation of VT in patients with coronary artery disease is feasible. This technique may be particularly useful as adjunctive therapy in patients with coronary artery disease who have had several types of VT and who have partially responded to pharmacological or device therapy but who have frequent recurrences of one or two types of monomorphic VT.
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