Long-term Results of Catheter Ablation of Idiopathic Right Ventricular Tachycardia

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Ten consecutive patients with recurrent episodes of symptomatic, idiopathic, sustained monomorphic ventricular tachycardia (VT) originating in the right ventricle underwent an attempt at catheter ablation of the ventricular tachycardia. There were seven women and three men, with a mean age of 39±14 years (±SD). None of the patients had any evidence of structural heart disease. The VT had a left bundle branch block configuration and an inferior axis in each patient, and the mean cycle length was 313±75 msec. Based on the methods of induction of the VT and the response of the VT to verapamil, the VT mechanism was presumed to be reentry in six patients, triggered activity in three patients, and catecholamine-sensitive automaticity in one patient. Sites for ablation were guided by pace mapping, and an appropriate target site was identified in the right ventricular outflow tract in each patient. From one to three shocks of 100–360 J (mean total, 336±195 J) were delivered from a defibrillator between the tip of the ablation catheter (cathode) and a patch electrode on the anterior chest (anode). An electrophysiology test 7–9 days after ablation demonstrated that VT was still inducible in only one patient, who was treated with amiodarone. One other patient had a recurrence of VT 3 weeks after ablation and was treated with verapamil. Eight of 10 patients were not treated with antiarrhythmic medications and had no episodes of symptomatic VT during 15–68 months of follow-up (mean follow-up, 33±18 months). There were no acute or long-term complications. In conclusion, long-term success in preventing VT is achievable safely and in a high percentage of patients who have idiopathic right VT originating in the right ventricle. (Circulation 1990;82:2093–2099)

Several studies have demonstrated that catheter ablation of ventricular tachycardia (VT) with electric shocks may be effective in preventing recurrences of VT.1–7 The majority of patients who have been the subjects of these studies have had structural heart disease; in most cases, the VT originated in the left ventricle. Among patients in whom VT originated in the right ventricle, the majority have had arrhythmogenic right ventricular dysplasia.1,2,5 Therefore, very little information has been available on the results of catheter ablation of idiopathic VT originating in the right ventricle.8

The purpose of this study was to describe the long-term results of catheter ablation of idiopathic right VT in 10 patients who were followed for 15 months to 5.5 years after ablation.

Methods

Patient Characteristics

The study population comprised 10 consecutive patients with recurrent episodes of sustained, monomorphic VT of one configuration who did not have any evidence of structural heart disease and who underwent an attempt at catheter ablation in the right ventricle. Characteristics are given in Table 1.

There were seven women and three men, with a mean age of 39±14 years (±SD). The first episode of sustained VT occurred at a mean of 48±49 months before the catheter ablation procedure. The number of clinical episodes of sustained VT preceding the ablation procedure was four or five in four patients and 10 or more in six patients. In each patient, the VT was associated with symptoms of rapid palpitations, lightheadedness, and chest pain or dyspnea, and one patient experienced loss of consciousness as a result of the VT.

The VT had a left bundle branch block configuration and an inferior axis in each patient. The mean
rate of the clinically documented episodes of VT was 190±40 beats/min. Between three and five antiarrhythmic medications (including amiodarone in two patients) had been either not tolerated or ineffective in controlling the VT on a clinical basis or during electropharmacological testing.

No patient had any evidence of structural heart disease. Each patient had a normal physical examination, electrocardiogram, and echocardiogram. The possibility of arrhythmogenic right ventricular dysplasia was ruled out by the absence of any right ventricular abnormalities on the echocardiogram and by the presence of a normal right ventricular ejection fraction by miltigated radionucleide angiography. A contrast left ventriculogram and coronary angiograms were normal in each patient. In addition, no histological abnormalities were present in five patients who underwent right ventricular myocardial biopsies. Two of the patients in the series were also included in a previous report.4

Electrophysiology Study Protocol

Electrophysiology tests were performed in the fasting, unsedated state after informed consent had been obtained. Three 6F quadrupolar electrode catheters (USCI, Billerica, Mass.) with 0.5- or 1-cm interelectrode spacing were inserted into a femoral vein and positioned in the right atrium, across the tricuspid valve to record the His bundle electrogram, and in the right ventricle. A previously unused catheter was used for right ventricular mapping and to deliver the ablative shocks. A 6F quadripleurode catheter with 0.5-cm spacing inserted into a femoral artery was used for left ventricular mapping. Leads V1, I, and III and the intracardiac electrogrodes were recorded on a Siemens-Elema Mingograf 7 (Solna, Sweden) recorder at a paper speed of 100 mm/sec. A 12-lead electrocardiogram was recorded each time VT was induced and during pace mapping. Pacing was performed with a programmable stimulator (Bloom Associates, Ltd., Reading, Pa.) using stimuli that were 2 msec in duration and had a current strength twice the diastolic threshold (always <1 mA). Sustained VT was defined as VT lasting more than 30 seconds, and nonsustained VT was defined as VT from six beats to 30 seconds in duration.

It is realized that the mechanism of VT often cannot be definitively established in a clinical electrophysiology laboratory. Nevertheless, a presumptive diagnosis of mechanism may be appropriate based on certain observations.9 The mechanism of VT was classified as reentry if the VT was provokable by programmed stimulation with one to three ventricular extrastimuli and was not suppressed by 10–20 mg i.v. verapamil.9–11 Triggered activity was considered to be the possible mechanism of VT if the VT was inducible by atrial and ventricular pacing at a critical range of cycle lengths and if the induction of VT was suppressed by 10–20 mg verapamil administered intravenously.9–11 The mechanism of VT was considered to be catecholamine-sensitive automaticity if the VT was not provokable by atrial or ventricular pacing or programmed stimulation but was inducible by infusing isoproterenol and if the VT could not be terminated by pacing but was terminated within several minutes after discontinuation of the isoproterenol infusion.9–11

Mapping of Ventricular Tachycardia

In light of the fact that the VT had a left bundle branch block configuration in each patient and that none of the patients had structural heart disease, the site of origin of the VT was presumed to be in the right ventricle, and detailed left ventricular mapping was not performed. Because the VT had an inferior axis in each patient, mapping efforts were concentrated in the right ventricular outflow tract.

The primary mapping technique was pace mapping.12 Pacing was performed either during sinus rhythm at a rate similar to the VT rate or during VT at a rate slightly faster than the VT rate. The QRS morphology in each of the 12 leads of the electrocar-
VT, CL 340 ms

Pacing, CL 300 ms, RVOT

FIGURE 1. A pace map (patient 1) in which pacing at a site in right ventricular outflow tract (RVOT) resulted in QRS complexes that closely matched QRS complexes during ventricular tachycardia (VT). VT cycle length (CL) was 340 msec, and pacing cycle length was 300 msec. Top panel: Frontal plane leads. Bottom panel: Precordial leads.
diagram was compared with the QRS morphology during VT. If pacing was performed in the setting of VT, the pace-map was considered valid only if there was no evidence of entrainment of the VT. Typical entrainment was ruled out by the absence of QRS fusion during incremental pacing rates, and concealed entrainment was ruled out by the presence of a short stimulus latency (<40 msec) during pacing. Mapping was directed toward identifying a site at which pacing resulted in the closest possible match between the QRS complexes during pacing and during VT. Pace-maps were graded on a scale of zero to 12, based on the number of leads in which there was a close match between pacing and VT (Figure 1).

The endocardial activation time relative to the QRS was recorded at each mapping site during VT using the distal pair of electrodes of the mapping catheter. The bipolar electrograms were recorded at a filter setting of 50–500 Hz.

**Catheter Ablation Protocol**

The catheter ablation protocol was approved by the human subjects committee. After an appropriate target site was identified, the distal electrode of the catheter that had been used for mapping was connected to the cathodal output of a defibrillator (Airshields, Hatboro, Pa.), and a 16-cm patch electrode (R2 Corp., Niles, Ill.) positioned on the left anterior chest wall was used as the anode. Sodium methohexitol was used for general anesthesia. Shocks of 100–360 J that had a damped sinusoidal configuration were delivered. The inducibility of VT was tested 5–10 minutes after delivery of the shock; if VT was still inducible, another shock was delivered after additional mapping. When VT was no longer inducible, an additional shock was delivered to the same site to minimize the risk of recurrent VT. A maximum of three shocks were delivered.

The patients underwent continuous monitoring for 7 days after the ablation procedure. Creatine kinase (CK)-MB fractions were measured every 8 hours during the first 24 hours after ablation. Right and left ventricular function was assessed by echocardiography 3 or 4 days after the procedure.

The inducibility of VT was tested in the electrophysiology laboratory 7–9 days after ablation. The stimulation protocol consisted of incremental atrial pacing to the point of atrioventricular block, incremental ventricular pacing between cycle lengths of 600 and 250 msec, and programmed ventricular stimulation with one to three extrastimuli using at least two basic drive cycle lengths between 600 and 400 msec at the right ventricular apex and right ventricular outflow tract or septum. The entire stimulation protocol was repeated during an infusion of 1–3 μg/min isoproterenol titrated to attain a sinus rate of 110–120 beats/min.

**Follow-up**

The patients were seen on a regular basis every 3–6 months by one of the authors or by the referring physicians. To obtain follow-up information for this study, each patient was interviewed in person or by telephone by one of the authors.

**Statistics**

Values are given as mean±1 SD. Statistical analyses were performed using Student's t test or linear regression analysis. A probability value of less than 0.05 was considered significant.

**Results**

**Mechanisms of Ventricular Tachycardia and Results of Mapping**

Monomorphic VT that had the same left bundle branch block configuration and inferior axis as the patients' spontaneous VT was inducible in every patient. The VT was sustained in nine patients and nonsustained in one patient. The mean cycle length of the induced VT was 313±75 msec. The method of induction and the presumed mechanisms of VT are described in Table 1. The mechanism of VT was presumed to be reentry in six patients, triggered activity in three patients, and catecholamine-sensitive automaticity in one patient.

The mapping results are also described in Table 1. In each patient, the best pace-map achievable was at a site in the right ventricular outflow tract. The pace-map score was 12 in three patients and 11 in seven patients.

The endocardial activation time during VT at the sites of the best pace-map ranged from 0 to −45 msec. In each patient, the site of the best pace-map was also the site at which the earliest endocardial activation was recorded. The mean endocardial activation time in the three patients who had VT presumed to be caused by triggered activity (−22±10 msec) was not significantly different than the mean endocardial activation time in the six patients presumed to have reentrant VT (−22±18 msec).

**Short-term Results of Catheter Ablation**

The mean number of ablative shocks was 2±0.7 and the mean total energy delivered was 336±195 J (Table 2). Ten to 15 minutes after the last shock was delivered, VT was not inducible in any of the patients. The VT became noninducible after delivery of one shock in seven patients and after delivery of two shocks in three patients.

During the 7 days of postablation monitoring, none of the patients had any episodes of spontaneous VT. Electrophysiologic testing 7–9 days after ablation demonstrated sustained monomorphic VT that had the same configuration as the baseline VT and a cycle length of 270 msec in one patient (Table 2); the VT was induced by triple ventricular extrastimuli during infusion of isoproterenol. The electrophysiology test demonstrated no inducible VT in the other nine patients.

**Complications**

The mean maximum CK concentration after ablation was 1,056±1,092 IU/l (normal range, 30–225
**TABLE 2. Technique and Results of Catheter Ablation of Ventricular Tachycardia With Shocks Delivered in Right Ventricular Outflow Tract**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Shocks (n)</th>
<th>Total J (N1&lt;225 IU/l)</th>
<th>Max CK (N1&lt;10 IU/l)</th>
<th>Maximum CK-MB (N1&lt;10 IU/l)</th>
<th>EPS 7–9 days after ablation</th>
<th>Discharge medications</th>
<th>Follow-Up (mo)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1</td>
<td>150</td>
<td>237</td>
<td>50</td>
<td>No VT</td>
<td>None</td>
<td>68</td>
<td>No VT</td>
</tr>
<tr>
<td>2</td>
<td>1</td>
<td>150</td>
<td>557</td>
<td>68</td>
<td>No VT</td>
<td>None</td>
<td>49</td>
<td>No VT</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
<td>500</td>
<td>1,668</td>
<td>156</td>
<td>No VT</td>
<td>None</td>
<td>48</td>
<td>VT (3 wk), verapamil†</td>
</tr>
<tr>
<td>4</td>
<td>2</td>
<td>200</td>
<td>1,000</td>
<td>51</td>
<td>No VT</td>
<td>None</td>
<td>38</td>
<td>No VT</td>
</tr>
<tr>
<td>5</td>
<td>2</td>
<td>200</td>
<td>384</td>
<td>21</td>
<td>VT, CL 270 msec*</td>
<td>Amiodarone</td>
<td>28</td>
<td>No VT</td>
</tr>
<tr>
<td>6</td>
<td>2</td>
<td>400</td>
<td>1,344</td>
<td>75</td>
<td>No VT</td>
<td>None</td>
<td>27</td>
<td>No VT</td>
</tr>
<tr>
<td>7</td>
<td>2</td>
<td>200</td>
<td>318</td>
<td>15</td>
<td>No VT</td>
<td>None</td>
<td>23</td>
<td>No VT</td>
</tr>
<tr>
<td>8</td>
<td>2</td>
<td>400</td>
<td>955</td>
<td>92</td>
<td>No VT</td>
<td>None</td>
<td>16</td>
<td>No VT</td>
</tr>
<tr>
<td>9</td>
<td>3</td>
<td>760</td>
<td>3,833</td>
<td>518</td>
<td>No VT</td>
<td>None</td>
<td>16</td>
<td>No VT</td>
</tr>
<tr>
<td>10</td>
<td>2</td>
<td>400</td>
<td>266</td>
<td>13</td>
<td>No VT</td>
<td>None</td>
<td>15</td>
<td>No VT</td>
</tr>
<tr>
<td>Mean</td>
<td>2</td>
<td>336</td>
<td>1,056</td>
<td>106</td>
<td>No VT</td>
<td>None</td>
<td>33</td>
<td>No VT</td>
</tr>
<tr>
<td>±SD</td>
<td>±0.7</td>
<td>±195</td>
<td>±1,092</td>
<td>±150</td>
<td>No VT</td>
<td>None</td>
<td>±18</td>
<td>No VT</td>
</tr>
</tbody>
</table>

*VT was induced by triple extrastimuli during infusion of isoproterenol and had same configuration as baseline VT.
†Recurrence of sustained VT 3 weeks after ablation; patient has been treated with verapamil and has had no further episodes of symptomatic VT.

CK, creatine kinase; EPS, electrophysiology study; VT, ventricular tachycardia; CL, cycle length; S, double ventricular extrastimuli; A, atrial; V, ventricular; Iso, isoproterenol; Ss, triple ventricular extrastimuli; TA, triggered activity.

IU/l) and the mean maximum CK-MB fraction was 106±150 IU/l (normal range, 0–10 IU/l) (Table 2). There was a significant correlation between total energy of the ablative shocks and maximum CK-MB fraction ($r^2=0.7$, $p<0.01$).

No electrocardiographic or echocardiographic abnormalities were noted after ablation in any of the patients. Continuous electrocardiographic monitoring demonstrated occasional premature ventricular depolarizations in four patients. No patient had VT or ventricular fibrillation.

**Long-term Results**

Patient 5, who had inducible sustained VT 1 week after ablation, was treated with amiodarone and has had no recurrences of symptomatic ventricular tachycardia during 28 months of follow-up (Table 2).

Patient 3 had an episode of symptomatic, sustained VT 3 weeks after ablation. She was treated with verapamil and has had no further recurrences of symptomatic VT.

The remaining eight patients were not treated with any antiarrhythmic drugs after ablation and have had no episodes of symptomatic VT. The mean duration of follow-up has been 33±18 months (range of follow-up, 15–68 months) (Table 2).

**Discussion**

**Main Findings**

The results described in this report demonstrate that idiopathic right VT may be caused by reentry, triggered activity, or catecholamine-sensitive automaticity and that regardless of the mechanism it is possible to achieve long-term success in eliminating the VT by catheter ablation with electric shocks. Eighty percent of the patients in this series were not treated with antiarrhythmic medications after ablation and have had no recurrences of symptomatic VT during a minimum of 15 months of follow-up. Our results also suggest that successful catheter ablation of right VT can be achieved safely, with a low risk of complications. In this series, no complications, and specifically no late proarrhythmic events, have been observed up to 5.5 years of follow-up.

**Mapping Technique**

The site of origin of VT conventionally has been identified by activation mapping, which involves searching for the site of earliest endocardial activation relative to the QRS complex during VT. In patients with coronary artery disease, left ventricular sites at which endocardial activation precedes the QRS by 50–100 msec often can be found. In contrast, in this study of patients with idiopathic VT, endocardial activation times earlier than 20–30 msec before the onset of the QRS often could not be identified. This was the case regardless of whether the presumptive mechanism of idiopathic VT was reentry, triggered activity, or catecholamine-sensitive automaticity. In the case of triggered activity and automatic VTs, in which there is not continuous depolarization of some portion of a reentrant circuit during diastole, endocardial activation times that are very early relative to the QRS complex would not be expected. However, the explanation for earlier endocardial activation times during reentrant VT in patients with coronary artery disease than in patients with idiopathic VT is unclear. It is possible that the
earlier endocardial activation relative to the QRS in patients with coronary artery disease may reflect differences in size, spatial configuration, conduction properties, or anatomical location of the reentry circuit compared with patients with idiopathic VT.

These types of differences in the VT reentry circuit may also explain why concealed entrainment could be demonstrated in more than 50% of VTs in patients with coronary artery disease and a history of prior myocardial infarction but could not be demonstrated in any patients with idiopathic VT in this series. The phenomenon of concealed entrainment, in which pacing during VT results in acceleration of the VT rate to the pacing rate, in a long stimulus-to-QRS interval (>100 msec), and in no change in the QRS morphology, may indicate that the pacing site is within a zone of slow conduction of the reentry circuit. The inability to demonstrate concealed entrainment in patients in the present study suggests that the reentry circuit of idiopathic VT differs significantly in nature or location from the reentry circuit of VT in patients with prior myocardial infarction.

A site at which there is a close match between the QRS complexes during pacing and during VT presumably is near the site of origin of VT caused by triggered activity or an automatic mechanism or close to the exit site of the reentry circuit when the VT is caused by reentry. Although the precise spatial resolution of pace mapping in identifying the site of origin of VT or the exit site of a reentry circuit remains to be determined, our results suggest that pace mapping is clinically useful in identifying an appropriate target site for ablation of VT, regardless of whether the presumptive mechanism of VT is reentry, triggered activity, or automaticity. Furthermore, it is clear that a perfect match between pacing and VT is not necessary to achieve a successful outcome, because several of the patients in this series had no recurrences of VT after delivery of shocks to target sites at which the pace-map score was 11 instead of 12. However, because shocks were not delivered to sites at which the pace-map score was less than 11, the minimum number of leads in which a close match between VT and pacing is necessary to achieve successful ablation of VT remains unknown.

Triggered Activity

In the present study, three of 10 patients with idiopathic VT were found to have VT possibly due to triggered activity, and the outcome of catheter ablation has been successful in two of these three patients during 38–49 months of follow-up. Although the abnormality that is responsible for the occurrence of idiopathic VT caused by triggered activity is unknown, the long symptom-free interval after ablation suggests that the abnormality may be localized and amenable to focal ablation compared with a diffuse process that could result in recurrent episodes of VT from other foci after the original focus is ablated. Our results suggest that the same may be true of VT caused by catecholamine-sensitive automaticity. However, additional experience with catheter ablation of both of these types of VT will be necessary before the suitability of a focal ablative technique for these two mechanisms of VT can be established.

Complications of Catheter Ablation

Electric shocks delivered to the endocardium of the right ventricle resulted in myocardial necrosis, as reflected by release of CK-MB. The amount of enzyme release varied widely from patient to patient and correlated with the total energy of the ablative shocks. However, postablation echocardiograms did not demonstrate any right ventricular wall motion abnormalities, and none of the patients has developed signs of right ventricular failure during follow-up. Therefore, catheter ablation in the right ventricle with shocks totaling 150–800 J in strength does not appear to result in clinically significant right ventricular dysfunction. Nevertheless, because as little as one shock of 150 J can result in the long-term suppression of VT and because the amount of myocardial necrosis that results from the ablative shocks is directly related to the amount of energy delivered, it may be preferable to avoid the use of shocks of more than 200 J.

It has been unclear whether the delivery of electric shocks to the endocardium of patients with idiopathic VT might create arrhythmogenic foci that could result in new ventricular arrhythmias several months or years after ablation. Although the results of the present study cannot rule out the possibility that this may occur many years later, it is at least somewhat reassuring that no evidence of late arrhythmic complications has been observed up to 5.5 years after ablation.

Limitations

Target sites for ablation were selected by pace mapping, and although the endocardial activation times recorded at the target sites were always the earliest endocardial activation times that had been recorded from all of the mapping sites, we cannot rule out the possibility that an earlier endocardial activation time might have been recorded at a site that was not mapped. The present study was not designed to compare the clinical value of pace mapping with that of activation mapping in identifying appropriate target sites for ablation, and no conclusions can be drawn regarding which mapping technique is more accurate.

Another limitation of the present study is that a standardized protocol for delivering ablative shocks was not used in the patients. The shock strength varied from 100 to 360 J, depending on the preference of the attending physician in charge of the ablation procedure. Because the number of patients receiving shocks of a particular strength was small, the optimal shock strength cannot be identified based on the limited data in this series.

A third limitation is that the small number of patients does not allow an accurate determination of the risk of complications. The absence of any clinically
significant complications in 10 patients suggest that the risk is low; however, the possibility of rare complications cannot be excluded. Additional experience in a larger number of patients will be required to determine the actual morbidity of catheter ablation of VT arising in the right ventricular outflow tract.

Finally, it should be noted that the ablation site in each of the patients was the right ventricular outflow tract. Therefore, the results obtained in this series may not necessarily apply to patients who have idiopathic VT originating in the inflow tract or at the apex of the right ventricle. However, all patients with idiopathic right VT who underwent catheter ablation at our institutions were included in this series, and there was no selection bias based on the particular site of origin within the right ventricle; the fact that the VT originated in the right ventricular outflow tract in each patient strongly suggests that this is by far the most common site at which idiopathic right VT arises.

Conclusions

Long-term success in preventing recurrences of symptomatic VT is possible by catheter ablation in patients who have idiopathic sustained monomorphic VT of one configuration arising in the right ventricle, regardless of whether the VT is caused by reentry, triggered activity, or catecholamine-sensitive automaticity. The 80% success rate in this series suggests that successful ablation of right VT can be achieved in a high percentage of patients when target sites for ablation are selected based on pace mapping. Furthermore, our experience indicates that ablation with electric shocks of idiopathic VT arising in the right ventricle can be achieved safely, with low risks of clinically significant right ventricular dysfunction and early or late proarrhythmic complications. Another factor that favors the catheter ablation approach to idiopathic right VT is the relative ease with which an electrode catheter can be manipulated within the right ventricle compared with within the left ventricle.

Given these considerations, it may be appropriate to attempt catheter ablation not only in patients with idiopathic right VT who cannot be effectively managed with antiarrhythmic medications but also in patients who may prefer to avoid chronic pharmacological therapy, particularly with drugs that have significant long-term risks of toxicity, such as amiodarone.

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References


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