Treatment of Ventricular Tachycardia by Transcatheter Radiofrequency Ablation in Patients With Ischemic Heart Disease

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Background Recurrent sustained ventricular tachycardia (VT) is not responsive to antiarrhythmic drugs in the majority of patients, who therefore need therapy with nonpharmacological methods. We evaluated prospectively the feasibility, safety, and efficacy of transcatheter radiofrequency (RF) ablation of VT in 21 selected patients with ischemic heart disease and VT.

Methods and Results Twenty-one patients with ischemic heart disease and recurrent, drug-refractory VT documented by 12-lead ECG were selected who had sufficient hemodynamic tolerance of VT to undergo transcatheter mapping. Documented clinical VT was reproduced by programmed cardiac stimulation (PCS), and the site of origin was localized by a combination of techniques, including pace mapping, activation-sequence mapping, recordings of middiastolic potentials, and application of resetting and entrainment principles. RF current at 55 V was applied (3.8±3.1 applications per patient) for as long as 30 seconds at a time to target sites. Twenty-four distinct clinical VTs (mean cycle length, 445±52 milliseconds) were mapped and ablated in 21 patients. In 17 of 21 patients (81%), the procedure was acutely successful, and the target clinical VT could no longer be induced by PCS after the procedure, whereas in 4 patients, clinical VT remained inducible. By contrast, VTs with shorter cycle length and different QRS morphology than the ablated VT could still be induced by PCS in 12 of 21 patients. One patient died in intractable congestive heart failure 10 days after the procedure, and the remaining 20 are alive at the end of the follow-up period. The majority of the patients continued to be treated with at least one additional mode of antiarrhythmic therapy; 12 patients were still taking antiarrhythmic drugs, and 9 patients received an implantable cardioverter/defibrillator. During a mean follow-up period of 13.2±5.0 months, 9 of 20 patients (45%) had recurrent VT. In 4 patients, the recurrent VT was different than the previously ablated one. Clinical VT recurred in all 4 patients in whom RF ablation had been acutely unsuccessful. Four patients with recurrent VT underwent repeat RF ablation procedures that were acutely successful and had no further recurrence.

Conclusions Transcatheter RF ablation is feasible but has only moderately high efficacy in a small, selected group of patients with ischemic heart disease and drug-refractory, highly frequent, hemodynamically tolerated, sustained VT. In the majority of the patients, this treatment technique is palliative rather than definitive, and many of the patients continue to require other methods of antiarrhythmic therapy.

Keywords: • tachycardia • radiofrequency • ablation • ischemia • heart disease

Therapeutic options in patients with recurrent, sustained ventricular tachycardia (VT) include antiarrhythmic drug therapy, electrophysiologically guided ventricular surgery, transcatheter electrical ablation, and automatic implantable cardioverter/defibrillator (ICD) therapy. Many patients have drug-resistant VT or develop intolerance to the antiarrhythmic drugs that effectively suppress their arrhythmias, thus requiring nonpharmacological methods of therapy. Map-guided ventricular surgery may be highly effective in carefully selected patients, but the risk of ventricular surgery is high; in addition, many patients are not surgical candidates because of severely depressed ventricular function or the presence of advanced disease in other organ systems. Transcatheter DC ablation of the site of VT origin has been reported to result in low-to-moderate success in suppressing recurrent VT, but there are growing concerns about its immediate and latent morbidity and mortality.

Recently, radiofrequency (RF) energy has been used for transcatheter ablation of accessory pathways, modification of the atrioventricular node for treatment of supraventricular tachycardias, and elimination of ectopic atrial foci. However, data on transcatheter ablation of VT by using RF energy are very limited, particularly in patients with structural heart disease. The purpose of this investigation was (1) to evaluate prospectively the feasibility, safety, and efficacy of transcatheter RF ablation for treatment of recurrent, sustained, monomorphic VT in patients with ischemic heart disease and previous failure of one or more established methods of antiarrhythmic therapy and (2) to describe the limitations of the transcatheter RF ablation technique and to clarify its role as a treatment method for VT in patients with ischemic heart disease.

Methods

Selection Criteria
The study population consisted of 21 patients with ischemic heart disease who were selected from a group of 307 patients referred to Massachusetts General Hospital for management of VT between January 1991, when a protocol was prepared for prospectively evaluating the safety and efficacy of RF
Table 1. Clinical Characteristics of Patients, Their Induced Ventricular Tachycardias, and Follow-up

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<th>LVEF</th>
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<th>PRE</th>
<th>POST</th>
<th>ICD PRE</th>
<th>POST</th>
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<th>REC</th>
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Table shows number (n) of antiarrhythmic drugs (AADS) tried before ablation, drugs patient was taking at admission (PRE), drugs that were administered after ablation (POST), patients who had implantable cardioverter/defibrillators (ICDs) implanted before radiofrequency (RF) ablation, those who had ICDs implanted after RF ablation, response to programmed cardiac stimulation (clinical VT [CLIN], nonclinical VT [NON], or no VT) after RF ablation, VT recurrence (REC), and follow-up (F/U). Figures in parentheses in F/U column represent duration of follow-up after second ablation. VT indicates ventricular tachycardia; D, disopyramide; Dx, diagnosis; LVEF, left ventricular ejection fraction; CAD, coronary artery disease; Pro, procainamide; Quin, quinidine; Ami, amiodarone; M, mexiletine; Eth, ethmozine; S, sotalol; N, no; and Y, yes.

Ablation of VT, and December 1992. Selection criteria included previous clinical failure of other methods of antiarrhythmic therapy; absence of hemodynamic collapse during clinical VT; documentation of the "clinical," spontaneous, sustained, monomorphic VT with a 12-lead ECG recording; and reproducible induction of this documented VT by programmed cardiac stimulation (PCS). Each patient had to have at least one clinical VT recurrence during chronic antiarrhythmic drug therapy optimized by serial electrophysiological testing. Patients who experienced syncope or hemodynamic collapse during VT were excluded, as were those with acute myocardial infarction, unstable angina pectoris, untreated heart failure, left main coronary artery disease, aortic stenosis, or any contraindication to right- or left-side heart catheterization. Fifty of the 307 patients (16%) presenting with VT met the inclusion criteria stated above and underwent PCS with attempted ventricular endocardial catheter mapping. Ventricular mapping could not be adequately performed or completed, and therefore transcatheter RF ablation could not be applied. In 20 patients because of poor hemodynamic tolerance of the induced VT over periods long enough to complete mapping or because of technical problems associated with catheter mapping. Of the remaining 30 patients (10%) who underwent ventricular mapping and transcatheter RF ablation, 21 constituted a homogenous group with coronary artery disease and chronic myocardial infarction. The results of the transcatheter RF ablation in these 21 patients are presented.

Clinical Characteristics

There were 1 female and 20 male patients with a mean ± SD age of 67 ± 8 years (range, 50 to 81 years) (Table 1). All patients underwent right- and left-side heart catheterization and coronary angiography. Two-dimensional echocardiography was performed before and after the ablation procedure in all patients.

A mean of 3.4 antiarrhythmic drugs (range, 1 to 7) per patient had been tried and had proved to be ineffective with clinical VT recurrence during chronic therapy despite compliance by the patient (Table 1). The antiarrhythmic drugs that the patients were taking at the time of hospital admission that was necessitated by recurrent VT are shown in Table 1. Three patients presented with incessant VT (patients 10, 12, and 1 [second VT]; Table 2). Before transcatheter RF ablation, 6 patients had already undergone ICD implantation (Table 1), 3 patients had undergone left ventricular (LV) aneurysmectomy and directed subendocardial resection, and 1 patient had undergone transcatheter ablation using DC energy for drug-refractory and frequently recurrent VT. Sixteen patients had a single surface ECG QRS morphology documented during spontaneous VT, and 5 patients had two distinct VT surface ECG morphologies documented clinically. In patients with an ICD, spontaneous VT could be documented with a 12-lead ECG recording either because the VT rate was lower than the programmed detection rate or when VT recurred in the
hospital while the cardiac rhythm was being continuously monitored after inactivation of the ICD.

**Electrophysiological Testing**

Transcatheter RF ablation for treatment of VT was considered investigational, and a prospective protocol was submitted to the institutional review board. Electrophysiological studies were performed with the patients in the fasting, postabsorptive state after informed written consent was obtained. Antiarrhythmic drugs had been discontinued before the procedure in 10 patients. In 9 patients in whom drug therapy had slowed the rate of recurrent VT, rendering it hemodynamically tolerable without suppressing it, the electrophysiological mapping and catheter ablation were performed while the patients continued to take their antiarrhythmic drugs. These drugs included amiodarone in 4 patients, procainamide in 1 patient, quinidine in 1 patient, sotalol and mexiletine in 1 patient, and procainamide and mexiletine in 2 patients.

Two 6F quadripolar electrode catheters were inserted percutaneously into a femoral vein; one was positioned at the right ventricular (RV) apex, and the other was positioned at the RV outflow tract, His bundle position, or right atrium as needed during mapping. A 7F quadripolar deflectable electrode catheter was inserted percutaneously into the right or left femoral artery for LV stimulation and mapping. PCS consisted of one, two, and three extrastimuli during ventricular pacing at two drive cycle lengths (600 and 400 milliseconds) from the RV apex and RV outflow tract. LV PCS using single and double extrastimuli at two drive cycle lengths was performed if the clinically documented, sustained VT could not be reproduced by stimulation from two RV sites.

**Definitions**

Electrically induced VT was considered to be the same arrhythmia as the “clinical” VT when it was identical in surface ECG configuration (same QRS duration, same bundle branch block mimicry, same frontal and horizontal axes) to the documented, spontaneous VT and did not differ in cycle length by more than 50 milliseconds. An induced VT that differed in surface ECG morphology (different bundle branch mimicry or a change in either frontal or horizontal axis by more than 45°) from the documented VT was considered “nonclinical.” Whenever VTs with multiple distinct surface ECG configurations were induced, the one that matched the documented clinical VT in surface ECG morphology was chosen as the target arrhythmia for mapping and ablation. Nonclinical VTs induced by PCS were not ablated unless they occurred spontaneously later and were mapped and ablated during a second study. The recording site that manifested the earliest local electrogram relative to a fiducial point on the surface

### Table 2. Characteristics of 25 Ventricular Tachycardias in 21 Patients

<table>
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<tr>
<th>Patient</th>
<th>Induction Mode</th>
<th>VT-CL (Clinical)</th>
<th>No. of Distinct Morphologies</th>
<th>Earliest LAT</th>
<th>Site of Origin</th>
<th>MDP</th>
<th>Concealed Entrainment</th>
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<td>+</td>
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VT indicates ventricular tachycardia; CL, cycle length; LAT, local activation time relative to a surface ECG fiducial point; MDP, middiastolic potential; RV, right ventricle; PCS, programmed cardiac stimulation; NA, not available; and LV, left ventricle.

*Patients who underwent a second ablation for a second, "new" VT.
†One patient who underwent a second ablation for the same VT.
ECG during VT was called “the site of origin” of VT. This was frequently, but not uniformly, the same as the site of RF current application (vide infra).

**Ventricular Mapping**

RV and LV mapping was carried out with 7F deflectable quadripolar electrode catheters (Mansfield, Inc) introduced into the femoral vein or femoral artery percutaneously. Catheter positions were identified fluoroscopically in the right and left oblique positions using a three-coordinate system described previously. The position of the electrode catheter was determined as a first approximation by pace mapping using a previously described ECG algorithm applied to VT and pace-mapped surface ECG morphology. Beyond this approximate localization, all higher-resolution mapping was based on findings observed during endocardial activation-sequence mapping and resetting and entrainment during sustained, monomorphic VT induced by PCS.

The two independent criteria used in selecting the site of RF current application were the local activation time during VT and the presence of isolated middiastolic potentials, low-amplitude, high-frequency electrograms separated from the major local electrogram by an isoelectric segment (Fig 1) with components that could be reset or entrained whenever VT was reset or entrained. When a recording site manifesting such a diastolic potential was encountered, synchronized resetting and entrainment were attempted first by stimulating a remote site (eg, RV outflow tract for a middiastolic potential recorded from the left ventricle). If the middiastolic potential could be reset and entrained whenever VT was reset and entrained, then pacing at multiple cycle lengths was attempted from the site manifesting the middiastolic component, in an attempt to demonstrate concealed entrainment. These middiastolic potentials that could be reset and entrained and the recording sites from which concealed entrainment could be demonstrated were used as the primary targets in selecting RF ablation sites. Otherwise, the earliest recorded presystolic local electrogram was used as the site of RF current application.

During VT induction and ventricular mapping, the femoral arterial pressure was monitored continuously. Heparin was administered intravenously at an initial dose of 4000 U, followed by 1000 U every additional hour during the electrophysiological procedure. Surface ECG, intracardiac electrograms, and the arterial pressure were displayed on an oscilloscope and recorded at a paper speed of 100 to 200 mm/s using a Midas (PPG Industries, Inc) system throughout the procedure.

**Catheter Ablation Techniques**

RF current at a frequency of 500 kHz was delivered as a continuous, unmodulated sine-wave output from an RF current generator (model RFG-3C, Radionics). The RF current was delivered between the 4-mm tip (27 mm² surface area) of a 7F deflectable quadripolar electrode catheter and an indifferent dispersive patch electrode (129 cm²) positioned on the thorax. RF current was delivered in applications of up to 30 seconds' duration at each ablation site while maintaining an output potential between 50 and 60 V. The current was turned off earlier than 30 seconds in case of an abrupt rise in impedance or catheter-tip displacement observed by fluoroscopy.

PCS was repeated if VT terminated during RF current application. A complete PCS protocol using as many as three extrastimuli from two RV sites and an additional LV site was performed 30 minutes later if no VT was inducible by PCS immediately after RF energy delivery. If the target VT was still inducible by PCS, ventricular mapping resumed, followed by repeated attempts at RF ablation.

After ablation, the cardiac rhythm of each patient was monitored continuously in a cardiac telemetry unit for a minimum of 3 days. Serial serum creatine kinase (CK) levels and MB fractions were measured during the first 48 hours, and intravenous heparin was administered for 3 days after ablation procedure. An electrophysiological study with PCS was repeated 2 to 4 days after RF ablation in all except two patients—one in whom the ablation was acutely successful and whose worsening clinical heart failure, despite absence of VT, precluded further invasive studies, and another in whom first RF ablation was acutely unsuccessful and who was reluctant to undergo another study. Oral warfarin therapy was started for prevention of thromboembolism and maintained for at least 30 days after discharge.

**Follow-up**

All patients had ambulatory ECG studies in addition to continuous cardiac rhythm monitoring before discharge.
Short-term suppression of VT was defined as no spontaneous and no induced occurrence of the clinical target VT before hospital discharge. After discharge, follow-up information was obtained by one of the investigators by direct interview with the patient and with the referring physicians. In patients with an ICD, additional follow-up data were acquired through regular interrogation of the implanted device.

Statistical Analysis

Parameters are presented as mean±SD. Comparisons for univariate analysis were performed using Fisher’s exact test. A value of P<.05 was considered significant. The actuarial VT-free survival after hospital discharge was described using Kaplan-Meier curves.

Results

A total of 30 ventricular mapping and transcatheter RF ablation procedures were performed in 21 patients to eliminate 25 distinct target VTs (Table 1). In 3 patients, ablation of the clinical VT was accomplished in two back-to-back mapping and ablation sessions during the same hospitalization, and in 1 patient (patient 17 in Table 1), three consecutive sessions were performed to ablate two different VTs during the same hospitalization. Three patients underwent two separate hospitalizations for transcatheter ventricular mapping and RF ablation for two different VTs, both of which had been documented clinically and both of which could be reproduced by PCS (patients 1, 3, and 9 in Table 1). One patient (patient 10 in Table 1) underwent a second ablation session for the same target VT after recurrence of his tachycardia 1 month after discharge from the hospital.

Electrophysiological Study and Mapping

VT was incessant in 3 patients (VTs 1B, 10, and 12 in Table 2) and inducible by PCS in all others. The incessant or inducible VTs manifested only one surface ECG morphology in 8 patients; nine patients had two distinct, 3 patients had three distinct, and 1 patient had four distinct induced monomorphic VT surface ECG morphologies (Table 2). At least one of the induced VTs matched the clinically documented VT in surface ECG morphology in every patient who underwent transcatheter RF ablation. Twenty-four distinct induced VT surface ECG configurations, all previously documented clinically, were mapped. The mean cycle length of these target VTs was 445±52 milliseconds (range, 310 to 550 milliseconds) (Table 2).

The ventricular locations of the specific sites of origin for all mapped VTs and other characteristics of the VTs are shown in Table 2. During 20 of 25 VTs mapped in 16 of 21 patients, isolated middiastolic potentials were recorded (Table 2). In 8 patients, ventricular pacing from these sites manifesting a middiastolic potential during VT, using a stimulus strength 1 mA above capture threshold and decrementing the pacing cycle length by 20 milliseconds at a time starting with VT cycle length, resulted in findings consistent with concealed entrainment. During the remaining VTs, similar pacing at decrementing cycle lengths resulted in acceleration of the VT rate to the ventricular pacing rate with minimal changes in surface QRS configuration (minimal fusion activation) and with varying degrees of latency (80 to 210 milliseconds) relative to pacing artifact. Rarely, suprathreshold pacing from these sites resulted in VT termination.

During five VTs, isolated middiastolic potentials could not be recorded despite extensive searching, and a combination of criteria, including earliest local activation times and VT entrainment with no change in surface QRS morphology with maximal stimulus to QRS duration, was used to select target sites for RF ablation.

Ablation Procedure and Its Immediate Effect

A mean of 3.8±3.1 applications of RF current were delivered per patient (range, 1 to 10), for a cumulative RF current duration of 87±39 seconds (range, 32 to 230 seconds). The mean power was 29 W (range, 26 to 48 W). In 14 of the 21 patients, VT terminated during RF current application. At the end of the procedure, 21 of the 25 clinical target VTs (84%) were no longer inducible in 17 of 21 patients (81%), and 4 of 25 target VTs (16%) were persistently inducible in 4 patients (19%) (Table 1). In 6 of 17 patients with no inducible target VT, no sustained VT of any kind could be induced by PCS (Table 1). In the remaining 11 patients, although the target VT could no longer be induced after RF ablation, the stimulation protocol induced one or two nonclinical VTs (Table 1) that manifested different surface ECG configuration and shorter cycle lengths (mean cycle length, 320±60 milliseconds; range, 240 to 380 milliseconds; compared with a mean cycle length of 445±52 milliseconds for ablated VTs) than the target VTs. The majority of these faster VTs could not be mapped because of poor hemodynamic tolerance. Few VTs that could be mapped uniformly showed a site of origin different from the previous site of ablation by at least two coordinate differences. Our policy was not to approach these VTs, previously undocumented clinically, by mapping and RF ablation.

Complications

One patient died in the hospital 10 days after transcatheter RF ablation. Before RF ablation, he had had clinical heart failure, and his LV ejection fraction was 20%. In this patient, RF ablation was undertaken to eliminate frequent spontaneous VT that contributed to cardiac deterioration. He had no further recurrent VT after the RF ablation procedure but died of progressive congestive heart failure associated with sepsis. The patient had undergone four RF current applications, nearly the same as the mean number per patient, and the highest serum CK value documented after his ablation was 93 U/L, suggesting that RF ablation had not resulted in substantial CK myocardial injury. Nevertheless, the contribution of RF ablation to this patient’s death cannot be ruled out; therefore, the procedure-related mortality was 4.8%. The remaining 20 patients remained hemodynamically stable after the procedure.

The mean maximal serum CK measured was 88±45 U/L (range, 31 to 410 U/L; normal range, 20 to 400 U/L) with a mean MB fraction of 9±5% (range, 5% to 19%; normal range, 0% to 3%). CK-MB measurements showed a mean maximal value of 26.7±14.9 ng/mL (range, 6.8 to 70.0 ng/mL; normal range, 0 to 7.5 ng/mL).

One patient developed ST segment elevation in inferior leads during energy delivery that resolved over 1 hour; the highest serum CK value measured in this patient was 47 U/L. Another patient developed a
transient pericardial rub that resolved without other complications.

Suppression of VT and Other Therapies

Of the 19 patients who underwent follow-up PCS before discharge, the results of PCS were concordant with those observed at the end of the RF procedure: clinical VT was not inducible in 15 of 19 patients, but faster, nonclinical VTs (electrically induced during the RF ablation session but not previously documented as spontaneous VT) remained inducible in 10 of 19 patients. In 3 of 19 patients, the clinical VT was persistently inducible. Thus, clinical VT was not inducible during the follow-up study before hospital discharge in 15 of the 19 patients (79%) studied.

Five of the 6 patients with no spontaneous or inducible postablation VT of any kind were discharged from the hospital off antiarrhythmic drugs (Table 1). One patient who had been on amiodarone for years and who underwent LV mapping and RF ablation while receiving this drug remained on the same regimen after ablation (Table 1). Nine of the 11 patients with rapid nonclinical VT induced after transcatheter RF ablation were treated with antiarrhythmic drugs, and one underwent ICD implantation (Table 1). Two of the patients with persistently inducible clinical VT were treated with amiodarone and sotalol, respectively, in addition to having had ICDs implanted before RF ablation, and two underwent new ICD implantation (Table 1). All of the three ICDs implanted de novo after RF ablation were third-generation devices capable of storing information during detected events. Thus, a total of 12 of 21 patients were still taking antiarrhythmic drugs after RF ablation, with 6 of 12 taking amiodarone (Table 1). However, the antiarrhythmic drug at discharge was new or different from the antiarrhythmic drugs previously taken in only 3 of the 21 patients (Table 1).

Follow-up

We classified the 20 patients who were discharged from the hospital according to their response to last PCS; 16 patients left the hospital with the ablated VT no longer inducible, and 4 had persistently inducible VT (Fig 2). All 20 surviving patients discharged from the hospital were alive at the end of a mean follow-up period of 13.2±5.0 months (range, 5 to 21 months). During this period, 9 patients (45%) had recurrent VT, resulting in 55% long-term efficacy (Fig 2). The actuarial VT-free survival in the 20 patients discharged from the hospital is represented by the graph in Fig 3. VT recurred early (1 to 3 months) after discharge in all except one patient, who had recurrent VT 11 months after RF ablation (Table 1).

Fig 2 describes the follow-up data in graphic format. All 4 patients who were discharged from the hospital with their clinical VT persistently inducible after the ablation procedure had recurrent VT 1 to 3 months after RF ablation, documented to be their clinical VT by surface ECG (Fig 2). Five of the 16 patients in whom clinical VT could not be induced after RF ablation had documented recurrent VT (Fig 2). In 3 of these 5 patients (patients 1, 3, and 9 in Table 1), the recurrent VT was shown to be different than the previously ablated VT by surface ECG morphology and by repeat ventricular mapping that identified a site of origin distinct from the previous RF ablation site (Table 2). These 3 patients underwent RF ablation of the new VT, after which the second ablated VT was rendered no longer inducible acutely and at the predischarge study. These 3 patients have had no recurrence since the second ablation (Fig 2). In another patient (patient 14 in Table 1) with successful ablation of the clinical VT, in whom rapid VTs other than the ablated VT had been induced by PCS, recurrent VT resulted in a countershock from his ICD with a programmed detection rate of 188 beats per minute. Because the cycle length of the previously ablated VT was 500 milliseconds, this recurrent VT, terminated by the ICD countershock, was considered different than the previously ablated one.

Fig 3. Plot of actuarial ventricular tachycardia (VT)-free survival after radiofrequency ablation in 20 patients discharged from the hospital. The solid line represents recurrence-free survival for any VT, and the broken line represents recurrence-free survival for previously ablated “clinical” VTs.
In summary, in 4 patients, the recurrent VT was different than the ablated VT, whereas recurrent VT was the same arrhythmia as the previously documented and targeted clinical VT in the remaining 5 patients (Fig 2). The recurrence of the ablated clinical VT is represented by the broken line in Fig 3. Therefore, the actual recurrence rate of the ablated VT was 25% during the follow-up period, and in 4 of these 5 patients, failure of RF ablation had been immediately demonstrable by the persistence of the PCS-induced clinical VT before hospital discharge. Thus, clinical VT recurred in only one of the 16 patients who had their ablated VT no longer inducible before discharge from the hospital.

One patient with acutely unsuccessful ablation (patient 10) presented in incessant VT with the same morphology as before and underwent a second ventricular mapping and transcatheter RF ablation procedure for the same VT, which this time was acutely successful, and this patient has not experienced recurrent VT since the second ablation (Fig 2).

Predictors of Recurrence

Univariate analysis failed to identify LV ejection fraction, VT cycle length, and the characteristics of the local electrograms recorded from the ablation site (local activation time, presence of middiastolic potentials) as correlates of immediate success or of VT recurrence during follow-up period. Although the presence of middiastolic potentials recorded from the ablation site predicted VT interruption during RF current application, this finding did not correlate significantly with clinical VT recurrence.

Clinical VT recurred in 4 of the 4 patients (100%) in whom the clinical VT remained inducible after RF ablation. By contrast, in 16 patients whose clinical VT could not be induced after RF ablation, the ablated VT recurred in 1 (7%). The persistence of PCS-induced clinical VT after RF ablation strongly correlated positively with clinical recurrence of the ablated VT after discharge (P=.0012). By contrast, the presence of any inducible VT, including the nonclinical ones other than the target VT, did not predict VT recurrence.

Discussion

The experience with RF ablation for VT is limited. Favorable results have been reported but only in small groups of patients with limited follow-up, and two of these most recent reports included the results of RF ablation in small numbers of patients without coronary artery disease. Our study describes the results observed in 21 highly selected patients with recurrent VT of ischemic heart disease. These results demonstrate that in patients with hemodynamically tolerated VT of ischemic heart disease, transcatheter RF ablation is a feasible procedure with relatively few complications, low (5%) mortality, and modest long-term efficacy. The high recurrence rate of VT over a short period of time in patients whose procedures were acutely unsuccessful (Fig 3) underscores the high frequency of recurrent VT in this selected group of patients. Our results also show that in ischemic heart disease where multiple morphologically distinct VTs are often seen in the same patient, a different form of VT may arise after successful elimination of one form. Thus, transcatheter RF ablation remains palliative rather than curative and does not obviate the need for treatment with other antiarrhythmic methods.

Comparison With Previous Results

Our acute success rate of 84% (21 of 25 targeted VTs no longer inducible immediately after ablation) in patients with VT of coronary artery disease is similar to the acute success rate of 85% previously reported in patients with ischemic heart disease by Morady et al. The same investigators reported that the targeted VT did not recur in any of the 12 patients who had an acutely successful RF ablation during a mean follow-up of 9 months. In the present study, targeted VT recurred in 1 patient in the group with acutely successful RF ablation, whereas recurrence of any VT, including new forms that had not been mapped and ablated, was more common. Other differences between the present study and the previously published report include differences in mapping technique, during follow-up period, and in the incidence of amiodarone therapy after ablation.

RF Ablation and Other Methods of Therapy

Compared with the previously published results of high-voltage DC catheter ablation, the short-term efficacy of RF ablation appears to be better. More focused energy delivery in the RF technique may be responsible for its higher success rate compared with transcatheter DC shock. However, it is also true that the mapping techniques used to identify the site of origin of VT have become more refined over the past 5 years, and the greater success of RF ablation may reflect this improvement in mapping and precise localization rather than the differences between the two energy-delivery techniques. It is reasonable to expect a higher safety record for RF ablation because it avoids the barotrauma commonly associated with transcatheter DC shocks. However, longer follow-up in many more patients is needed before the safety of the RF ablation technique can be adequately assessed and compared with the short- and long-term complications associated with transcatheter DC ablation.

It is difficult to compare RF ablation with other methods of antiarrhythmic therapy, especially because only a small, selected minority of all patients with VT are candidates for mapping and RF ablation. Antiarrhythmic drug therapy is usually disappointing in treatment of recurrent sustained VT of ischemic heart disease because less than one third of the patients have their recurrent VT suppressed by drugs. ICD therapy is universally applicable and highly effective for VT with rapid rates. However, ICD may be inappropriate for patients with very frequent or incessant VTs with relatively slow rates. As shown by our own results, it is not uncommon for recipients of ICD to require other methods of antiarrhythmic therapy in an attempt to suppress recurrent VT resulting in frequent countershocks. Map-guided ventricular surgery with endocardial resection is the only other method of directed, focused antiarrhythmic therapy for VT. Although the long-term efficacy of this technique in survivors of surgery is higher than of transcatheter ablation, many patients with recurrent VT are poor surgical candidates because of markedly depressed ventricular function,
and perioperative mortality and morbidity are substantially higher in all reported series compared with transcatheter ablation.

Efficacy of RF Ablation and Myocardial Injury

The volume of myocardium injured irreversibly depends on the total energy delivered and the number of sites ablated. Our use of RF current for treatment of VT was conservative, and the average number of applications was similar to those previously reported. Reflecting this policy was the modest increase in the serum CK-MB fraction associated with RF ablation in this series. Even with limited injury, one death occurred 10 days after the procedure as a result of heart failure in a patient who died, but these two were not in clinical heart failure and they survived RF ablation without any complications. More definitive safety guidelines need to be defined, and until then, transcatheter RF ablation must not be undertaken in patients with heart failure and precarious ventricular function unless there is reasonable certainty that ventricular dysfunction is secondary to incessant VT and potentially, even if partially, reversible.

The significance and clinical relevance of nonclinical VT induced in the electrophysiological laboratory constitute a topic of controversy. Available data acquired in patients undergoing map-guided ventricular surgery suggest that inducible VT, even if different than the previously documented forms, may recur during the follow-up period. In our series, 4 patients had recurrent VT manifesting rates and surface ECG configurations different than the previously observed ones during their ablated clinical VTs. These 4 patients had already had different VTs induced by PES documented during the ablation session. By contrast, in 8 patients with successfully ablated clinical VT but still inducible VT of another morphology, there was no recurrence (Table 1). Ventricular mapping and RF ablation for all distinct VTs inducible in every patient are probably neither feasible nor safe.

The absolute need for limiting injury leads necessarily to the search for highly specific markers for identifying effective ablation sites. Most of the models of reentry postulate the presence of a zone of unidirectional block and slow conduction, and participation of such pathways in VT mechanism has been convincingly demonstrated in animal models of experimental myocardial infarction. Experimental data suggest that proximal segments of such pathways are the optimal sites for successful ablation. The hallmark of such pathways in VT of ischemic heart disease is believed to be the low-amplitude, isolated, middiastolic to early diastolic signals recorded during VT, which can be reset or entrained simultaneously with VT in the clinical electrophysiological laboratory. Such recording sites have been described as effective catheter ablation sites in previous reports. In our series, the middiastolic potentials could be recorded in the majority, and their presence at the ablation site predicted VT interruption during RF current application but did not guarantee long-term success. Thus, the presence of the middiastolic potentials may be necessary but not sufficient for durable effect, and additional criteria may have to be met for long-term success of RF ablation.

Technique Limitations

There are major, important limitations of the use of transcatheter RF ablation for treatment of VT. Our selection criteria were conservative but similar to those described in previous reports. With these criteria, only a small minority of all the patients with ischemic heart disease and sustained VT are suitable candidates for ventricular mapping and transcatheter RF ablation. In fact, the patients with the most life-threatening forms of VT—combining rapid VT rates and compromised ventricular function resulting in hemodynamic collapse—are not suitable candidates for ventricular mapping. These patients cannot be treated by transcatheter RF ablation and need therapy with antiarrhythmic drugs, ICD, map-guided surgery, or a combination of these techniques.

The second important limitation is related to the presence of multiple spontaneous as well as induced VT morphologies commonly observed in patients with advanced coronary artery disease. Although usually one clinical form predominates, as shown by a few of the patients in our study, after successful ablation, another form with a different surface ECG configuration may occur. Even if these “different” VTs are mechanically related, mapping shows their sites of origin to be disparate, and in practical terms this means applying more RF energy to different sites if the intention is to eliminate all VTs. Performing repeated ablations for multiple VTs in the same patient may result in an unacceptable amount of cumulative myocardial injury, with the potential to further reduce function in ventricles already compromised by ischemic injury. Therefore, in many patients with coronary artery disease, RF ablation remains palliative and does not represent definitive treatment for all VTs. This important limitation of the transcatheter RF ablation technique in patients with ischemic heart disease is clearly reflected by our results; 15 of 21 patients (71%) were being treated with antiarrhythmic drugs, ICD, or both after RF ablation.

Study Limitations

In our study, only 21 patients with ischemic heart disease and VT met the stringent selection criteria necessary to undergo RF ablation during a 2-year period. In addition, 14 of the 20 other patients in whom ventricular mapping was undertaken during this time period with the intention to perform RF ablation, which could not be done for various difficulties, had ischemic heart disease. Thus, if an intention-to-treat analysis were to be used, the acute success rate would decrease from 17 of 21 (81%) to 17 of 35 (49%), with even a lower rate for long-term success.

The concomitant use of antiarrhythmic drugs in our series is an important limitation that interferes with the accurate evaluation of the efficacy of transcatheter RF ablation. However, although 12 patients continued to take antiarrhythmic drugs after RF ablation, a new drug was added or the previous drug regimen was changed in only 3 patients. The remaining 9 patients remained on the antiarrhythmic drugs that had decreased VT rate without suppressing it (Table 1). Even though these drugs had proven to be ineffective as the only method of
therapy before RF ablation, freedom from VT after RF ablation is a measure of the efficacy of combined therapy with RF ablation and antiarrhythmic drug therapy rather than of RF ablation alone in these patients. Unlike antiarrhythmic drug therapy, concomitant ICD therapy may be an advantageous resource rather than a limitation during post–RF ablation follow-up as long as the implanted devices are capable of storing data that can be retrieved and analyzed later.

Another limitation of our study involves the criteria used for the selection of target sites. Although middiastolic signals were recorded from the ablation sites in the majority of the VTs, criteria satisfying concealed entrainment were present in only a minority (8 of 21, or 38%). More frequent use of this criterion for selection of the RF current sites may improve short- as well as long-term success of the procedure.

Clinical Implications

Transcatheter RF ablation is feasible in carefully selected patients with ischemic heart disease and frequently recurrent or incessant sustained monomorphic VT that is hemodynamically tolerated. It can be performed safely with relatively few complications, but in patients with most severely compromised ventricular function and a history of heart failure, it should be undertaken with great caution and only after all alternatives have been tried. The majority of the VTs can be ablated successfully provided that careful and detailed ventricular mapping can be performed. However, RF ablation remains an investigational technique that is at best a palliative form of therapy in patients with VT and advanced coronary artery disease, most of whom continue to require other forms of therapy mainly because of the presence of multiple distinct ventricular arrhythmias with different characteristics.

References


Treatment of ventricular tachycardia by transcatheter radiofrequency ablation in patients with ischemic heart disease.

Y H Kim, G Sosa-Suarez, T G Trouton, S S O’Nunain, S Osswald, B A McGovern, J N Ruskin and H Garan

_Circulation_. 1994;89:1094-1102
doi: 10.1161/01.CIR.89.3.1094
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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