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Radiofrequency Catheter Ablation as a Cure for Idiopathic Tachycardia of Both Left and Right Ventricular Origin

DWAIN L. COGGINS, MD, FACC, RANDALL J. LEE, MD, JOHN SWEENEY, MD, WALTER W. CHEIN, MD, FACC,* GEORGE VAN HARE, MD, FACC, LAURENCE EPSTEIN, MD, ROLANDO GONZALEZ, MD, JERRY C. GRIFFIN, MD, FACC, MICHAEL D. LESH, MD, FACC, MELVIN M. SCHEINMAN, MD, FACC

San Francisco and Modesto, California

Objectives. The purpose of this study was 1) to investigate the efficacy and safety of radiofrequency energy catheter ablation as curative treatment for idiopathic tachycardia of both left and right ventricular origin, and 2) to compare the usefulness of different methods used to map the site of origin of idiopathic ventricular tachycardia.

Background. Percutaneous radiofrequency catheter ablation has been used with dramatic success in the treatment of patients with Wolff-Parkinson-White syndrome, atrioventricular node reentrant tachycardia and bundle branch reentrant tachycardia. Limited data are available on the use of radiofrequency energy catheter ablation as curative treatment for idiopathic tachycardia of both left and right ventricular origin.

Methods. Twenty-eight consecutive patients (13 to 71 years old) presenting with idiopathic ventricular tachycardia were enrolled in the study. The site of origin of both left and right ventricular tachycardia was mapped using earliest endocardial activation times during tachycardia and by pace mapping. These mapping techniques were compared.

Results. Radiofrequency ablation was successful in all eight patients (100%) with left ventricular tachycardia. Tachycardia recurred in one patient. The ablation procedure was complicated by mild aortic insufficiency in one patient. Right ventricular outflow tract tachycardia was successfully ablated in 17 (85%) of 20 patients. The success rate at follow-up was 85%. In one patient, the ablation procedure was complicated by acute ventricular perforation and death. Pace maps from successful ablation sites were better than pace maps from unsuccessful sites ($p < 0.004$). Endocardial activation times at successful ablation sites were not different from unsuccessful sites ($p < 0.13$).

Conclusions. Radiofrequency catheter ablation is an effective treatment for idiopathic ventricular tachycardia. The site of origin of tachycardia is best identified using pace mapping. Significant complications can occur and should be considered in the risk/benefit analysis for each patient.

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Radiofrequency energy has been used with dramatic success in the ablation of accessory pathway connections (1–4), the atrioventricular (AV) junction (5,6) and in patients with AV node reentrant tachycardia (7–9). Application of this technique to patients with ventricular tachycardia and structural heart disease has had only limited success (10,11), except when used for the treatment of bundle branch reentrant ventricular tachycardia (12,13). Idiopathic ventricular tachycardia, by definition, occurs in patients without known structural heart disease and usually originates near the right ventricular outflow tract or from the posteroseptal aspect of the left ventricle (14). These tachycardias have been successfully ablated using direct current shocks (15). However, because of the risk of barotrauma, the need for general anesthesia and other potential complications with direct current ablation, there is now great interest in the use of radiofrequency energy for the ablation of these ventricular tachycardias. There are at present only isolated case reports describing ablation of ventricular tachycardia arising from the septal aspect of the left ventricle or other left ventricular foci, with variable success (16,17). Klein et al. (16) and Wilbur et al. (18) recently reported the results of radiofrequency catheter ablation in patients with idiopathic right ventricular tachycardia. The current study extends their findings by comparing different mapping techniques and by providing new data on electrocardiographic (ECG) localization of tachycardia origin.

The purpose of our study was to compare different percutaneous catheter mapping techniques and to describe the results of radiofrequency energy ablation in 8 patients with

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From the Department of Medicine, Cardiovascular Research Institute and Department of Pediatrics, University of California San Francisco, San Francisco and *Doctor's Medical Center, Modesto, California. Drs. Epstein and Lesh serve on the scientific advisory board of EP Technologies, Sunnyvale, California and receive honoraria for their participation.

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Address for correspondence: Dr. Melvin M. Scheinman, Room 312, Moffitt Hospital, Box 0214, University of California, San Francisco, California 94143.

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Table 1. Clinical Characteristics of Patients With Idiopathic Left Ventricular Tachycardia

Pt No./Gender	Age (yr)	Symptoms	Exercise Induced	No. of Previous AA Drugs	Heart Disease	VT Rate (ms)	QRS (ms)
1/F	32	P, PS	No	1	None	536	160
2/M	21	P, PS	Yes	3	None	306	120
3/M	21	P	No	1	None	333	150
4/M	30	P	No	2	None	429	130
5/F	17	P, PS	Yes	3	MVP	375	130
6/F	28	P	No	2	None	353	150
7/M	13	P, PS	No	1	None	440	160
8/M	30	P, PS	Yes	2	None	340	140
Mean	24			1.8		389	143
±SD	7			1.0		75	15

AA = antiarrhythmic; F = female; M = male; MVP = mitral valve prolapse with no murmur; P = palpitations; PS = presyncope; Pt = patient; QRS = duration of QRS complex during clinical ventricular tachycardia (VT).

idiopathic left ventricular tachycardia and 20 patients with right ventricular outflow tract tachycardia.

Methods

Patients. Twenty-eight consecutive patients who presented to the University of California, San Francisco between November 1989 and December 1992 with idiopathic ventricular tachycardia defined as recurrent monomorphic ventricular tachycardia with a right bundle branch block ECG configuration and superior axis or left bundle branch block configuration and inferior axis were enrolled in this study. All patients had complete history, physical examination, baseline ECG and echocardiography, and five patients underwent magnetic resonance imaging (MRI) before electrophysiologic evaluation. The latter two imaging techniques were used to exclude patients with right ventricular dysplasia and to identify patients with segmental wall motion abnormalities due to underlying coronary artery disease. No patient had more than one configuration of ventricular tachycardia occurring either spontaneously or induced in the electrophysiology laboratory. Two patients with a normal echocardiogram and several configurations of inducible ventricular tachycardia were excluded from this study.

Eight patients presented with idiopathic left ventricular tachycardia. All patients had documented sustained ventricular tachycardia with a QRS complex during ventricular tachycardia exhibiting a right bundle branch block configuration and no apparent underlying heart disease on the basis of history, physical examination, ECG and echocardiography. Ventricular tachycardia had a superior axis in all patients, but this was not required for inclusion. The clinical presentation of each patient is shown in Table 1.

Twenty patients presented with idiopathic right ventricular tachycardia. All patients had documented ventricular tachycardia with a QRS complex during ventricular tachycardia exhibiting a left bundle branch configuration and inferior axis. Individual clinical characteristics are shown in Table 2. Seventeen patients had no known heart disease and a normal baseline echocardiogram. Magnetic resonance imaging, performed in five patients, showed no evidence of right ventricular dysplasia. No patient with a normal echocardiogram was excluded because of an abnormal MRI study.

Three patients were found to have underlying structural heart disease. One patient (Patient 9) had normal coronary arteries and mild dilated cardiomyopathy, possibly related to years of asymptomatic incessant ventricular tachycardia. One patient (Patient 18) had normal biventricular function and left main coronary artery disease treated with bypass surgery. One patient (Patient 20) had three-vessel coronary artery disease treated with bypass grafting. She had an ejection fraction of 55% with left ventricular posterobasal hypokinesis by echocardiography. None of these three patients had evidence for right ventricular disease at echocardiography. In patients with structural heart disease, our definition of idiopathic ventricular tachycardia was more stringent. None of these patients had inducible ventricular tachycardia with programmed stimulation; all had reproducible spontaneous development of left bundle, inferior axis monomorphic ventricular tachycardia during isoproterenol infusion; none had fractionated electrograms or mid-diastolic potentials observed during mapping; and all had endocardial activation times comparable to those observed in other patients with idiopathic ventricular tachycardia. In addition, premature ventricular contractions in these patients were of a single configuration, identical to that of their ventricular tachycardia.

Electrophysiologic study. Informed written consent was obtained in all patients. Patients were brought to the electrophysiology laboratory in the fasting state. Under mild intravenous sedation, standard invasive electrophysiologic study was performed. Quadripolar electrode catheters were passed from the right femoral vein and positioned in the high right atrium, low septal right atrium and right ventricular apex. Surface ECG leads I, II, aVF and V_1 were continuously monitored. Bipolar intracardiac electrograms were filtered with a bandpass of 30 to 250 Hz. Intracardiac pacing was performed at twice diastolic threshold with a pulse

Table 2. Clinical Characteristics of Patients With Right Ventricular Outflow Tract Tachycardia

Pt No./Gender	Age (yr)	Symptoms	Exercise Induced	No. of Previous AA Drugs	Heart Disease	VT Rate (ms)	QRS (ms)
9/M	50	—	Yes	0	DCM, EF 50%	364	160
10/M	40	P, PS	No	1	None	239	130
11/F	27	P, S	Yes	0	None	341	140
12/F	47	P, PS	No	3	None	300	130
13/F	52	P	Yes	3	None	273	140
14/F	22	P	Yes	4	None	375	140
15/M	44	P, PS	No	1	None	259	160
16/F	51	P, PS, D	Yes	2	None	375	120
17/F	37	P, PS, CP, D	No	0	None	343	140
18/M	54	P	No	0	LMCA, CABG	571	150
19/F	41	P, PS	Yes	2	None	214	130
20/F	71	—	Yes	0	IMI, 3v, CABG	300	135
21/F	68	S, PS	No	0	None	310	120
22/F	50	P	Yes	1	None	340	150
23/F	34	S	Yes	3	None	440	150
24/F	41	P, PS	Yes	3	None	400	140
25/F	57	P	No	0	None	400	160
26/F	57	PS	No	0	None	340	135
27/M	43	P, PS	No	1	None	285	145
28/F	42	P, PS, D	No	1	None	360	140
Mean	46.4			1.3		341	141
±SD	12.1			1.3		79	12

CABG = coronary artery bypass graft surgery; CP = chest pain; D = dyspnea; DCM = dilated cardiomyopathy; EF = ejection fraction; IMI = inferior myocardial infarction; LMCA = left main coronary artery; S = syncope; 3v = three-vessel coronary artery disease; other abbreviations as in Table 1.

duration of 2.0 ms. Programmed electrical stimulation was performed in the baseline state with up to three extrastimuli at two drive cycle lengths from two right ventricular sites. If tachycardia was noninducible in the baseline state, isoproterenol infusion was initiated at 1 μg/min and increased to a maximal rate of 5 μg/min in 1-μg/min increments to increase heart rate by 25% to 30%. Programmed electrical stimulation was performed at each new infusion rate and during the "washout" phase after discontinuation of isoproterenol. End points were development of spontaneous sustained ventricular tachycardia with or without isoproterenol infusion, induction of sustained ventricular tachycardia with pacing or extrastimulation or 5 μg/min of isoproterenol infusion. Frequent spontaneous nonsustained ventricular tachycardia or inducible nonsustained ventricular tachycardia was considered sufficient for mapping.

Mapping and ablation procedure. Endocardial activation mapping and pace mapping were performed for localization of the ventricular tachycardia focus using a steerable quadripolar ablation catheter with 5-mm interelectrode spacing and a 4- or 5-mm distal tip electrode (8F, EP Technologies; 7F, Mansfield-Webster catheters, Boston Scientific). Before mapping left-sided tachycardias 5,000 U of heparin was administered intravenously followed by maintenance doses of 1,000 to 2,000 U of heparin hourly. The ablation catheter was introduced from the right femoral vein in patients with right ventricular outflow tract tachycardia and from the right femoral artery in patients with left septal ventricular tachycardia. A transseptal approach was used successfully during a second ablation session in Patient 2 with recurrent left septal ventricular tachycardia. The ablation catheter tip was positioned fluoroscopically using both the right and left anterior oblique views. In patients with right ventricular outflow tract tachycardia, the ablation catheter was advanced across the tricuspid valve into the right ventricular outflow tract. Patients did not undergo heparinization. The tip of the catheter was then carefully passed through the pulmonic valve. This was characterized by a loss of high frequency electrograms. Initial sites of endocardial activation mapping and pace mapping were performed by slowly withdrawing the ablation catheter through the pulmonic valve. Pace mapping was performed in sinus rhythm at endocardial sites showing early activation during ventricular tachycardia. Sites of early activation were defined as those showing endocardial potentials earlier then the earliest surface recordings. In patients with idiopathic left ventricular tachycardia, the ablation catheter was passed retrograde through the aortic valve with the tip of the catheter initially positioned in the midseptum posteriorly. Pace mapping was performed in sinus rhythm at endocardial sites showing early activation during ventricular tachycardia. Pacing was bipolar and was generally performed at twice diastolic threshold at a 1.0-ms pulse duration; however, pacing thresholds were not determined at all ablation sites. For both groups, endocardial sites showing early activation and a pace map that best reproduced the spontaneous ventricular tachycardia were treated with 20 to 50 W of continuous unmodulated energy at 550 kHz (EP Technologies) delivered between the distal pole

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of the ablation catheter and a large surface-area skin patch placed over the left scapula. Energy was applied during ventricular tachycardia or in sinus rhythm for up to 100 s. If there was an abrupt impedance increase, energy delivery was terminated immediately, the catheter removed and the distal electrode cleaned. Success was defined as abolition of inducible or spontaneous ventricular tachycardia. Programmed electrical stimulation both with and without isoproterenol infusion was repeated 30 min after successful ablation. Total procedure time, defined as total time in the electrophysiology laboratory, and fluoroscopic time were recorded in all patients.

Follow-up. All patients were monitored continuously in hospital for 24 to 48 h after the ablation procedure. Electrocardiography and echocardiography were performed before discharge in all patients. Follow-up information was obtained from either the referring physician or from direct follow-up in our arrhythmia clinic. Patients who developed a clinical recurrence had their recurrent tachycardia documented by Holter monitoring or ECG. A successful long-term ablation result was defined as the absence of sustained arrhythmias or symptoms of palpitations without antiarrhythmic agents.

Statistics. All values are presented as mean value ± SD. Pace maps at successful and unsuccessful ablation sites were scored in blinded fashion by four electrophysiologists. All pace maps were given a score for the number of leads with an identical height of R wave/depth of S wave (R/S) ratio match (12 represented a perfect R/S ratio match in all 12 leads), as well as a score for fine notching in each ECG lead. The total score (R/S ratio + fine notching) from each electrophysiologist for each map was added (perfect score $24 \times 4 = 96$). The scores for all pace maps obtained from successful radiofrequency ablation sites were compared with the scores for pace maps from sites where ablation was unsuccessful. A total of 22 successful and 63 unsuccessful pace maps were scored. The pace map from the successful ablation site was not available for analysis in three patients (Patients 3, 13 and 15). Only pace maps from sites where ablation was attempted were scored. Pace maps from patients with successful ablation, as well as those from patients with failed catheter ablation were included. Pace map scores from successful ablation sites were compared with those from unsuccessful ablation sites using two-group Kolmogorov-Smirnov nonparametric testing. Endocardial activation times at successful and unsuccessful ablation sites were compared using analysis of variance. The statistical package used was Statview II, version 1.04.

Analysis of the surface ECG as a predictor of precise location of right ventricular outflow tract tachycardia was performed using the successful ablation site as evidence for tachycardia origin. Comparisons were made using two-group Kolmogorov-Smirnov nonparametric testing. Differences were considered significant if $p < 0.05$.

Results

Idiopathic ventricular tachycardia was successfully ablated in 25 (89%) of 28 patients. Radiofrequency ablation was initially successful in all eight patients (100%) with left septal ventricular tachycardia. Tachycardia recurred in two patients, and one of these patients was successfully treated at a second ablation session, for an overall success rate of 87.5% at a mean follow-up of 10.1 ± 6.7 months. Right ventricular outflow tract tachycardia was successfully ablated in the electrophysiology laboratory in 17 (85%) of 20 patients. Tachycardia recurred in one patient and was successfully treated at a second ablation session, for a success rate of 85% at a mean follow-up of 9.9 ± 7.3 months.

Electrophysiologic characteristics. Data from the electrophysiologic study, mapping and catheter ablation procedure and subsequent follow-up for patients who presented with idiopathic left ventricular tachycardia are shown in Table 3. Idiopathic left ventricular tachycardia was inducible in four patients with pacing alone, in two patients with pacing during isoproterenol infusion and in one patient with isoproterenol infusion alone. The mean ventricular tachycardia cycle length was 393 ± 89 ms. Fascicular potentials were not observed on endocardial electrogram recordings from successful ablation sites in seven of eight patients. In Patient 6, a fascicular potential was observed 10 to 20 ms before the onset of the surface QRS complex in tachycardia. This patient had recurrent tachycardia twice after apparently successful ablations and had the only long-term failure.

Data from the electrophysiologic study, mapping and catheter ablation procedure and subsequent follow-up for patients who presented with idiopathic right ventricular tachycardia are shown in Table 4. Right ventricular outflow tract tachycardia was induced in 13 patients with isoproterenol infusion alone. In no patient did pacing protocols alone induce ventricular tachycardia. The configuration of premature ventricular complexes was identical to that observed during ventricular tachycardia. The mean cycle length of induced ventricular tachycardia was 330 ± 77 ms. None of the patients with underlying cardiac disease had inducible tachycardia with programmed stimulation, and all developed spontaneous episodes of ventricular tachycardia during isoproterenol infusion.

Mapping of ventricular tachycardia. Pacing and activation mapping data from patients with either left or right ventricular tachycardias were combined because we found no differences in this variable between groups. Endocardial activation times during ventricular tachycardia at successful ablation sites varied from −10 to −45 ms before the onset of the earliest surface QRS complex. Mean endocardial activation time during ventricular tachycardia was −26.2 ± 11.6 ms at successful and −25.4 ± 11.4 ms at unsuccessful ablation sites. This difference was not significant ($p < 0.13$) (Fig. 1).

For each patient, endocardial activation time at the successful ablation site was at least as early as the endocar-

Table 3. Electrophysiologic and Ablation Data in Patients With Idiopathic Left Ventricular Tachycardia

					Successful Pace Map					Clinical Follow-Up	
Pt No.	VT Induction	VT CL (ms)	RF (no.)	EAT (ms)	R/S	Notching	VT Location in LV Septum	Complic	Success	Duration (mo)	Findings
1	Incessant	540	5	−40	11	10	MidLV/PS	—	Yes	12	—
2	S4 at RVA on Iso	255	10	−40	12	11	MidLV/PS	—	Yes	8	VT rec/success
3	320 VOD on Iso	360	9	−40	—	—	MidLV/PS	—	Yes	25	—
4	350 VOD	430	4	−40	12	9	MidLV/PS	—	Yes	8	—
5	Spontaneous on Iso	450	3	−40	12	12	MidLV/PS	—	Yes	11	—
6	300 VOD	330	7	−40	12	12	MidLV/PS	Yes	Yes	7	VT rec/failed
7	300 VOD	440	3	−30	12	11	MidLV/PS	—	Yes	8	—
8	310 VOD	340	4	−30	12	11	Anteroseptal	—	Yes	2	—
Mean		393	5.6	−37.5	11.8	10.9				10.1	
±SD		89	2.7	4.6	0.5	1.0				6.7	

Complic = complications; EAT = endocardial activation time from surface QRS complex to intracardiac electrogram; LV = left ventricular; MidLV/PS = posteroseptal midway from apex to base; Pt = patient; RF = radiofrequency ablation; Notching = number of electrocardiogram (ECG) leads on the pace map from the successful ablation site with a perfect configurational match with the clinical ventricular tachycardia; R/S = number of ECG leads on the pace map from the successful ablation site with a perfect R/S ratio match with the clinical ventricular tachycardia; VT CL = cycle length of induced ventricular tachycardia; S4 at RVA on Iso = three extrastimuli at the right ventricular apex during isoproterenol infusion; 320 VOD on Iso = ventricular overdrive pacing at 320 ms during isoproterenol infusion; VT rec = ventricular tachycardia recurred after discharge from the electrophysiology laboratory/result of second ablation session.

dial activation time at unsuccessful ablation sites. From a practical standpoint, however, many unsuccessful sites had early endocardial activation times, and the decision to perform ablation could not be based on endocardial activation time alone.

Pace mapping was analyzed with respect to both R/S ratio in each lead and fine notching in each lead. A total of 22 successful and 63 unsuccessful pace maps were scored. At successful ablation sites, at least 11 of 12 leads had identical R/S ratios compared with the 12-lead ECG of clinical ven-

Table 4. Electrophysiologic and Ablation Data in Patients With Right Ventricular Outflow Tract Tachycardia

					Successful Pace Map					Clinical Follow-Up	
Pt No.	VT Induction	VT CL (ms)	RF (n)	EAT (ms)	R/S	Notching	VT Location in RVOT	Complic	Ablation Success	Duration (mo)	Findings
9	Iso	390	2	−30	12	12	PS	—	Yes	10	PVCs × 8 h
10	Iso	230	3	−35	12	10	PS	—	Yes	9	—
11	Iso	340	11	−45	12	12	AS	RBBB	Yes	15	—
12	Iso	315	21	−30	—	—	PS	—	Yes	13	—
13	S4 at RVA on Iso	280	29	—	—	—	—	—	No	12	VT/procainamide
14	Incessant	300	7	−20	—	—	AS	—	Yes	36	—
15	Iso	250	3	−35	11	10	AS	—	Yes	13	—
16	Noninducible		3	—	—	—	—	—	No	9	VT/moricizine
17	Incessant	380	2	−40	12	12	AS	RBBB	Yes	9	—
18	Iso	560	19	—	12	8	AS	—	Yes	11	NSVT
19	Iso	280	3	−10	12	12	AS	—	Yes	9	—
20	Iso	260	10	—	12	12	AS	—	Yes	11	—
21	Incessant	320	9	—	12	12	AS	—	Yes	7	PVCs
22	Iso	280	2	−10	12	12	AL at His	—	Yes	5	VT rec/success
23	Iso	260	10	−30	12	12	AS	—	Yes	5	—
24	Iso	440	3	−10	12	12	AS	—	Yes	5	—
25	Iso	400	5	−20	12	11	AS	—	Yes	5	—
26	Iso		7	—	—	—	—	RV perf	No	—	Pt died
27	S3 at RVOT on Iso	285	5	—	11	11	AS	—	Yes	3	—
28	Incessant	360	3	−25	12	10	AS	—	Yes	1	—
Mean		325	8.4	−26.3	11.9	11.0				9.9	
±SD		89	8.5	12.1	0.4	1.3				7.2	

AL at His = anterolateral at the level of the His catheter; AS = anteroseptal; NSVT = asymptomatic nonsustained ventricular tachycardia; PS = posteroseptal; PVCs = left bundle configuration premature ventricular contractions; RVOT = right ventricular outflow tract; RV perf = right ventricular outflow tract perforation with hemodynamic collapse; other abbreviations as in Table 3.

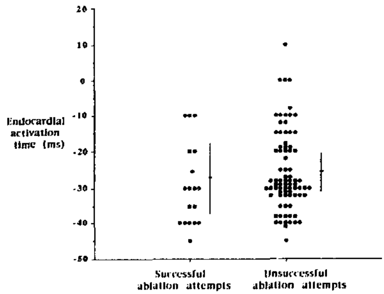


Figure 1. Endocardial activation times from sites of successful tachycardia ablation compared with activation times from sites of unsuccessful catheter ablation. Note the significant overlap between these groups ($p < 0.13$).

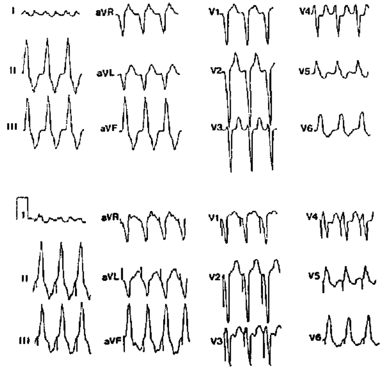
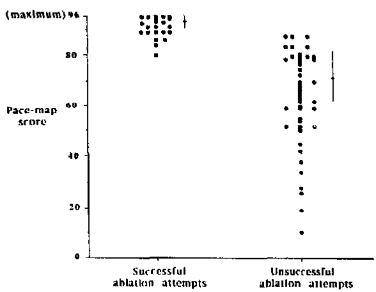


Figure 3. Comparison of a 12-lead electrocardiogram of spontaneous ventricular tachycardia and a pace map at the successful ablation site in the posteroseptal region of the left ventricle (Patient 9). Note the very close match of both R/S ratio and fine notching in all leads.

tricular tachycardia. Pace maps from successful ablation sites were significantly better than those from unsuccessful sites ($p = 0.004$) (Fig. 2). A representative pace map from a successful ablation site is shown in Figure 3. This pace map had a score of 94 (96 maximum).

Surface ECG localization of right ventricular outflow tract tachycardia. The surface ECG of tachycardia in patients with right ventricular outflow tract tachycardia was retrospectively analyzed in the 17 patients with successful radiofrequency ablation to determine whether the surface ECG could be used to more accurately localize the site of origin of right ventricular outflow tract tachycardia. Baseline ventricular tachycardia surface electrograms were assessed for

Figure 2. Comparison of pace map scores obtained from sites of successful tachycardia ablation with those from sites of unsuccessful ablation. The differences were significant ($p < 0.004$). A perfect pace map score was 96.



precordial QRS transition (first precordial lead with $R > S$) and limb lead configuration. The anatomic position of the successful radiofrequency ablation site was determined fluoroscopically from right and left anterior oblique views. Anatomic areas were defined as septal, lateral, anterior or posterior and either cranial (immediately below the pulmonic valve) or caudal (level of the His electrogram). The mean transition lead for cranial sites of ablation was 3.2 ($n = 9$, SEM $\pm$ 0.2) and 4.1 ($n = 6$, SEM $\pm$ 0.2) for caudal sites of ablation. This difference was significant ($p < 0.01$). Analysis of the limb lead QRS configuration demonstrated that all successful ablations in the septal side of the right ventricular outflow tract had a negative QRS complex in lead aVL (16 of 16), whereas successful ablation on the lateral aspect of the right ventricular outflow tract was associated with a positive QRS complex in lead aVL (1 of 1). Other limb leads offered no additional value in localizing the focus.

For left ventricular tachycardias, ECG analysis revealed either an rSR' or an RSr' in lead V_1 with a mean QRS duration of 143 $\pm$ 15 ms and a frontal plane QRS axis between 262 and −85. Analysis of limb lead configuration and transition of the R wave in the precordial leads did not offer additional information in localizing the left ventricular tachycardia focus.

Ablation results (Fig. 4 and 5). Radiofrequency ablation was successful in 8 (100%) of 8 patients with idiopathic left ventricular tachycardia at the time of discharge from the hospital. In six patients the successful ablation site was in the posteroseptal region of the left ventricle (Fig. 5). In one

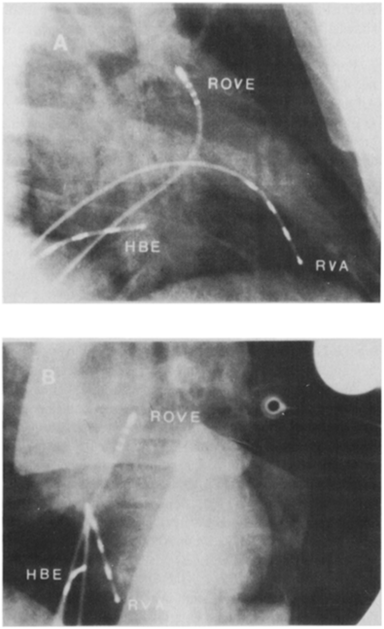


Figure 4. Right (A) and left (B) anterior oblique views of the successful radiofrequency ablation site in the anteroseptal region of the right ventricular outflow tract (Patient 11). ROVE = mapping and ablation catheter; RVA = catheter in the right ventricular apex; HBE = catheter in the His bundle position.

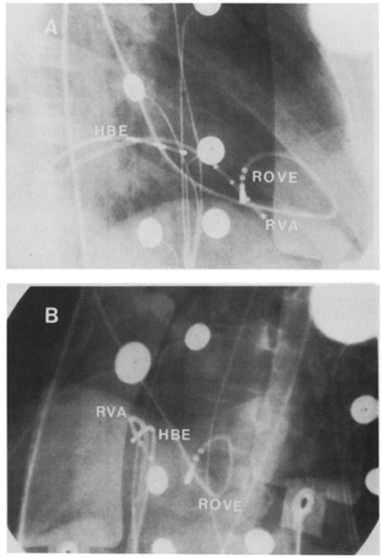


Figure 5. Right (A) and left (B) anterior oblique views of the successful radiofrequency ablation site in the posteroseptal region of the left ventricle (Patient 2). Abbreviations as in Figure 4.

patient the site of ventricular tachycardia was on the anteroseptal aspect of the left ventricle near the apex. Tachycardia in the remaining patient was ablated in the posteroseptal aspect of the left ventricle but was closer to the base than the apex.

Ablation was successful in 17 (85%) of 20 patients with right ventricular outflow tract tachycardia. The site of origin of right ventricular outflow tract tachycardia, on the basis of successful ablation, was in the anteroseptal region of the right ventricular outflow tract under the pulmonary valve in 13 patients (Fig. 4), in the posteroseptal right ventricular outflow tract under the pulmonary valve in 3 patients and in the anterolateral right ventricular outflow tract just cephalad to the His electrogram position in 1 patient.

Complications. Patient 6 who presented with idiopathic left ventricular tachycardia was noted to have a new murmur of aortic regurgitation on examination, and transthoracic echocardiogram confirmed mild aortic regurgitation. The ablation procedure in this patient was performed initially using a transseptal approach. After this failed, retrograde aortic catheterization was performed.

Permanent right bundle branch block occurred with ablation at the successful site in two patients with right ventricular outflow tract tachycardia. This did not occur as a consequence of catheter trauma, and there was no evidence for catheter dislodgment on the basis of biplane fluoroscopic imaging during ablation. One patient with idiopathic right ventricular tachycardia developed cardiac tamponade that was unrelieved by needle pericardiocentesis. Emergency thoracotomy stabilized the patient hemodynamically, but she failed to recover neurologically and died 1 month later. Transthoracic echocardiograms and Doppler studies performed after ablation were unchanged in the remaining 26 patients.

Discussion

This study confirms and extends previous reports of successful ablation in patients with idiopathic ventricular tachycardia. To our knowledge, this is this first report of a significantly large series of patients with left septal ventricular tachycardia treated by radiofrequency catheter ablation. A long-term cure was achieved in seven of eight patients.

Seventeen of 20 patients with right ventricular outflow tract tachycardia had long-term arrhythmia cure, as defined by absence of symptoms with no antiarrhythmic drug therapy. Although there have been a few recent reports describing series of patients undergoing successful radiofrequency ablation of idiopathic right ventricular tachycardia (16,17,18), the current study describes the largest series of patients with right ventricular outflow tract tachycardia reported to date. It is the first study to compare the success of different mapping techniques (endocardial activation mapping compared with pace mapping) and provides an analysis of the 12-lead ECG of right ventricular outflow tract tachycardia, which may help improve the ability to predict the location of the tachycardia focus. The report also describes a patient death that occurred as a complication of the ablation procedure. An additional point of interest was the finding that 3 of the 20 patients had associated cardiac disease.

Idiopathic ventricular tachycardia and coexisting cardiac disease. Three patients in our series had underlying heart disease (cardiomyopathy in one, previous myocardial infarction in one, coronary artery disease in one), but their clinical and electrophysiologic characteristics were otherwise typical of patients with idiopathic right ventricular tachycardia (16,19). Each of these patients presented with frequent episodes of nonsustained monomorphic ventricular tachycardia; each patient had noninducible tachycardia with programmed stimulation; and each developed spontaneous episodes of ventricular tachycardia during isoproterenol infusion. Endocardial activation time at the successful ablation site was similar to that in other patients in our series and similar to those reported by Klein et al. (16). There was no evidence for isolated mid-diastolic potentials during ventricular tachycardia, and no zone of slow conduction was observed as is characteristically found in patients with previous myocardial infarction and microreentrant ventricular tachycardia (20–22). We used the same mapping techniques to localize the site of tachycardia origin in these patients as was used for all other patients in our series. These patients emphasize that the presence of organic cardiac disease does not necessarily exclude the possibility of coexistent "idiopathic ventricular tachycardia."

Endocardial activation and mapping techniques. We found no difference in endocardial activation times during ventricular tachycardia between patients with a right ventricular outflow tract origin compared with those with left septal ventricular origin. Moreover, endocardial activation times at successful sites were similar to those reported by Klein et al. (16) and Wilber et al. (18). None of the patients showed fractionated potentials or evidence for a zone of slow conduction at the successful ablation site.

Early endocardial activation time was found to be a necessary but insufficient criterion for successful ablation. All successful ablation sites had endocardial activation times as early as or earlier than other sites in any given patient; however, many unsuccessful ablation sites had equally early activation times. In addition, the absolute value for endocardial activation time at successful ablation sites was highly variable, ranging from −10 ms in Patient 19 to −45 ms in Patient 11. In contrast to the report of Klein et al. (18), multiple unsuccessful ablation sites had endocardial activation times earlier than successful sites in other patients. We found endocardial activation time most useful as a guide to the general region of tachycardia origin. Pace mapping must then be used to identify the site of origin more accurately. During pace mapping, efforts were concentrated on matching the R/S ratio in all 12 ECG leads. If R/S ratios matched, fine notching morphology tended to follow.

Analysis of surface ECG tracings. We found that analysis of the 12-lead surface ECG provided important clues relative to right ventricular outflow tract tachycardia location. For example, more caudal locations (that is, close to the His bundle) in the right ventricular outflow tract were associated with later precordial R wave transition (leads V_4 to V_5) compared with cranial right ventricular outflow tract foci (that is, near the pulmonic valve), with transition between leads V_3 and V_4. Similarly, more lateral right ventricular outflow tract foci usually project positive forces in lead aVL.

Complications. We report the first death resulting from radiofrequency energy ablation in the right ventricular outflow tract. During the final ablation attempt in this patient, there was an impedance increase. Energy delivery was immediately interrupted. No sudden change in ablation catheter position was noted on continuous fluoroscopic imaging during the ablation attempt. On withdrawal of the catheter it was recognized that the catheter tip was adherent to the ventricular wall. With gentle manipulation the catheter was worked free and removed for cleaning. Coagulum was present on the catheter tip. Shortly thereafter, the patient's hemodynamic status deteriorated, and a large longitudinal tear in the wall of the right ventricular outflow tract was found during emergent surgical repair. We believe that this complication might have been avoided if a thermistor catheter with feedback control of catheter tip temperature had been used. Such catheters are now in clinical trials.

One patient was noted to have mild aortic insufficiency by examination and Doppler echocardiography after ablation of a left ventricular tachycardia. The cause of this abnormality is not known but is presumed to be catheter induced.

Conclusions. Our data demonstrate that radiofrequency catheter ablation can be highly successful in the treatment of idiopathic left ventricular tachycardia and support previous studies demonstrating the remarkable efficacy of ablative procedures in patients with idiopathic ventricular tachycardia arising from the right ventricle. We found that right ventricular outflow tract may coexist with other forms of cardiac disease; hence the presence of structural heart disease should not serve to discourage application of ablative procedures if the clinical and electrophysiologic findings are typical of idiopathic ventricular tachycardia. We found that analysis of the 12-lead ECG during ventricular tachycardia provided clues as to the tachycardia origin in patients with right ventricular outflow tract tachycardia. Pace mapping was clearly more accurate than endocardial activation mapping in finding successful ablation sites. Complete elimination of inducible ventricular tachycardia or spontaneous premature ventricular contractions (with configuration identical to that of ventricular tachycardia), together with perfect or near-perfect pace maps tended to predict long-term success.

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