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Transcatheter Electrical Shock Ablation of Ventricular Tachycardia

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Transcatheter shock ablation of ventricular tachycardia was attempted in seven patients who had drug-resistant ventricular tachycardia and in one patient in whom ventricular tachycardia was electrophysiologically induced during therapy with multiple antiarrhythmic drugs. Seven patients had previous myocardial infarction and five of them were high risk candidates for surgical therapy. One patient without organic heart disease had repetitive ventricular tachycardia manifesting two different patterns of left bundle branch block. After endocardial mapping, synchronized unipolar 250 to 300 J shocks (one to six) were delivered between the pole recording the earliest endocardial activity during ventricular tachycardia (40 to 200 ms before the onset of the QRS complex) and a body surface electrode. Immediate complications included severe but reversible cardiogenic shock (one patient), nonclinical ventricular tachycardia (two patients, requiring cardioversion in one), transient atrioventricular and intraventricular conduction disturbances (three patients) and permanent left bundle branch block (one patient). A late complication in one patient, left heart failure, occurred 3 days after delivery of five intracardiac shocks. In two patients, left ventricular ejection fraction markedly decreased and in one of them new ventricular contraction abnormalities appeared.

Clinical ventricular tachycardia did not recur in five of the seven post-myocardial infarction patients after 7 to 17 months, and it was not inducible in the four patients undergoing late electrophysiologic study. In the patient with idiopathic ventricular tachycardia, one of the configurational types of ventricular tachycardia recurred. It is concluded that transcatheter shock ablation of ventricular tachycardia is a promising technique that warrants further investigation, especially in high risk surgical candidates. The high incidence of complications, however, should temper any temptation to consider routine use of this procedure in patients with ventricular tachycardia.

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Patients with drug-refractory ventricular tachycardia are candidates for surgery, antitachycardia pacemakers or automatic defibrillators (1–3). Recently, delivery of localized direct current intracardiac shocks at the site of origin of ventricular tachycardia was shown to be effective in ablating foci of ventricular tachycardia (4). However, only a small number of patients undergoing this therapy have been reported on (4–11). In the present study, we describe our initial experience with transcatheter shock ablation of ventricular tachycardia in eight patients.

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Methods

Patients. The study group comprised eight patients, seven men and one woman, ranging in age from 21 to 66 years (mean 49 ± 16) (Table 1). Seven patients had coronary heart disease and had had a myocardial infarction 3 weeks to 5 years before the present hospitalization. Two patients (Cases 3 and 5) had a localized left ventricular aneurysm. Left ventricular ejection fraction, as determined by radionuclide ventriculography, ranged from 19 to 38% (mean 28 ± 6). One patient (Case 7) showed no demonstrable heart disease on right and left ventriculography and coronary angiography. However, her left ventricular ejection fraction was slightly depressed (45%), probably because of the cardiodepressive effect of concomitant therapy with 320 mg of propranolol and 120 mg of ajmaline bitartrate daily.

All eight patients had symptomatic ventricular tachyarrhythmias (Table 1). Patient 1 had incessant ventricular tachycardia that could not be terminated by antiarrhythmic drugs, right and left ventricular pacing techniques or repeated external cardioversions. This patient has been de-

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Table 1. Clinical and Electrocardiographic Characteristics of the Eight Study Patients

Case	Age (yr) & Sex	Spontaneous Arrhythmia	Frequency of Episodes of Arrhythmia	VT Configuration	VT Cycle Length (ms)	LVEF (%)
1	44/M	VT	Incessant	RBBB, LAD	360	30*
2	65/M	VT	3 in 1 month	RBBB, LAD	300	26
3	66/M	VT	15 in 2 weeks	LBBB, LAD	450	28
4	55/M	VT	4 in 10 months	LBBB, LAD	460	19
5	30/M	VT/VF	1 in 2 years	—	—	38
6	60/M	VT/VF	10 in 5 months	RBBB, axis −150°	450	26
				RBBB, RAD	430	
7	21/F	VT	Repetitive	LBBB, RAD	280	45
				LBBB, NA	280	
8	53/M	VT	1 in 2 months	RBBB, LAD	400	27

*Measured during ventricular tachycardia. F = female; LAD = left axis deviation; LBBB = left bundle branch block; LVEF = left ventricular ejection fraction; M = male; NA = normal axis; RAD = right axis deviation; RBBB = right bundle branch block, VF = ventricular fibrillation; VT = ventricular tachycardia

scribed elsewhere (11). Patient 2 had multiple episodes of sustained ventricular tachycardia that could be terminated only temporarily by either right ventricular pacing or external cardioversion. Three patients (Cases 3, 4 and 8) had recurrent drug-resistant sustained ventricular tachycardia. Patients 5 and 6 had, respectively, single and multiple episodes of cardiac arrest, and rapid ventricular tachycardia/ventricular fibrillation was documented at the time of resuscitation. In Patient 5, the arrhythmia was controlled by amiodarone therapy but neither amiodarone nor various antiarrhythmic agents prevented induction of rapid sustained monomorphic ventricular tachycardia by programmed ventricular stimulation. In Patient 6, who had severe angina, the tachyarrhythmias frequently occurred during episodes of ischemia and were not prevented by antiarrhythmic or antianginal drugs. Patient 7 had drug-refractory repetitive ventricular tachycardia consisting mainly of short runs (<30 seconds) and, less commonly, sustained episodes requiring pharmacologic intervention.

Twelve lead electrocardiography during spontaneous ventricular tachycardia showed a single configuration in five patients (Cases 1 to 4 and 8) and two configurations in two patients (Cases 6 and 7). In Patient 5, a single electrocardiographic lead showed a pleomorphic ventricular tachycardia.

Five patients (Cases 1, 2, 4, 6 and 8) had poor left ventricular function and were considered high risk candidates for surgical intervention. All patients gave written consent for electrophysiologic studies and ablative procedures.

Electrophysiologic studies. All patients underwent electrophysiologic studies, including programmed ventricular stimulation and catheter endocardial mapping, using standard methods previously described (12). At the time of the studies, Patients 1 to 6 were receiving amiodarone alone, Patient 7 was receiving propanolol and ajmaline and Patient 8 was not receiving any drug. Two quadripolar electrode catheters (USCI no. 6) were introduced through femoral veins and positioned in the right ventricular apex and the His bundle area. Another quadripolar electrode catheter (USCI no. 6, 1 cm interelectrode distance) was introduced into the left ventricle. In Patient 7, an additional electrode catheter was introduced into the right ventricle.

In patients with no spontaneous ventricular tachycardia at the beginning of the electrophysiologic study, programmed stimulation from the right or left ventricular apex was performed to induce ventricular tachycardia. The stimulation protocol included the delivery of one or two extrastimuli during sinus rhythm or during ventricular pacing at cycle lengths of 700 to 400 ms using stimuli of 2 ms duration and 3 or 20 mA intensity.

The site of origin of ventricular tachycardia was determined by extensive endocardial mapping of the left ventricle in the seven patients with coronary heart disease and of the right ventricle in Patient 7, in whom a right ventricular origin of the tachycardia was suspected. Precise endocardial mapping was feasible, as spontaneous or pacing-induced ventricular tachycardia was well tolerated hemodynamically in all eight patients.

Electrocardiographic leads I, II, III and V_1 and intracardiac electrograms were recorded on an eight channel Siemens-Elema Mingograph at paper speeds of 25 and 100 mm/s. Twelve lead electrocardiograms were frequently recorded during induced ventricular tachycardia and compared with electrocardiographic tracings during spontaneous ventricular tachycardia. A Medtronic 5325 stimulator was used for cardiac stimulation. Femoral artery pressure was continuously recorded throughout the procedure. Heparin (5,000 U) was administered to all the patients.

Definitions. The site of origin of ventricular tachycardia was arbitrarily defined as the earliest recorded endocardial electrical activity occurring in late diastole before the surface QRS complex during ventricular tachycardia (12). Induced ventricular tachycardia was defined as clinical when its con-

figuration and rate were similar to those of the spontaneously observed tachycardia. Induced ventricular tachycardia was defined as nonclinical when its configuration was different from that of the spontaneously occurring ventricular tachycardia.

Transcatheter shock ablation. This procedure was performed according to the technique previously described by Hartzler (4). Synchronous unipolar shocks were delivered using a standard defibrillator (Mennen-Greatbatch). The anodal output of the defibrillator was connected to a paddle placed at either the left posterolateral area or the anterior area (V_4 position) of the chest and the cathodal output was connected to the electrode recording the earliest endocardial activation during ventricular tachycardia. In Patient 1, technical difficulties prevented the administration of the shocks at the area showing the earliest endocardial activity, and therefore shocks were delivered at an adjacent site showing less early activity (11). However, for the purpose of clarity, the latter site was referred to as the site of origin of ventricular tachycardia. Except for the first two 250 J shocks given to Patient 1, all shocks delivered had an energy level of 300 J.

Protocol. The number of shocks varied in each patient, depending on effects, increasing experience and ability to induce ventricular tachycardia after the shock. However, we attempted to conform to the following protocol in most of our patients. After completion of endocardial mapping of ventricular tachycardia, the patients were anesthetized with midazolam, a benzodiazepin derivative, until a state of deep sedation was achieved. Additional intravenous boluses of midazolam were subsequently given throughout the procedure if necessary. The first intracardiac shock was delivered during ventricular tachycardia at its presumed site of origin. The intracardiac electrograms were continuously monitored before the shock to ascertain constant catheter position. After application of the shock, the patient was observed for 10 minutes. If ventricular tachycardia did not spontaneously recur during this period, induction by programmed ventricular stimulation was attempted. Whenever ventricular tachycardia could be induced, endocardial mapping of the tachycardia was again performed and a second intracardiac shock given as previously described. Five patients received a third shock. No patient received more than three shocks during a single procedure. In Patient 4, ventricular tachycardia could not be induced after the first shock and a second shock was given during sinus rhythm at the site of the first shock. In patients in whom well tolerated nonclinical ventricular tachycardia occurred or was induced by programmed ventricular stimulation after a shock, endocardial mapping of this tachycardia was accomplished. If the site of origin of the tachycardia was found to be close to that of the clinical tachycardia, an intracardiac shock was given at this site.

Four patients underwent two series of shocks within a 1 to 4 day period, and a single series of shocks was administered in the remaining four patients. At the end of the procedures, the patients were given a specific benzodiazepin receptor antagonist (RO 15-1788) that promptly restored consciousness. Both midazolam (13) and RO 15-1788 (Geller et al., unpublished data) have been shown to exert no significant cardiovascular effects.

Other procedures. After the catheter ablation procedure, the patient was transferred to the coronary care unit. A 12 lead electrocardiogram was recorded every 12 hours for the first day after the ablative procedure and daily thereafter. A chest X-ray film was obtained immediately after the ablative procedure. Serum creatine kinase and the creatine kinase-MB fraction were measured at 8 hour intervals for 3 days. In our laboratory, normal values for serum creatine kinase are less than 150 IU, and the normal creatine kinase-MB fraction is less than 5%. In seven patients, a radionuclide ventriculogram (technetium-99 pyrophosphate) was performed before and 3 to 6 days after the ablative procedures. In our laboratory, normal values for left ventricular ejection fraction are 50 ± 3%. In four patients electrophysiologic studies were performed 7 to 60 days after the ablative procedures. Programmed stimulation from the right ventricular apex, using the stimulation protocol described, was used in each patient.

Discharge antiarrhythmic regimen and follow-up. Patients 1 to 6, who were receiving amiodarone before the ablative procedures, were given the same drug after the procedure. Patients 7 and 8 were taking flecainide and no drug, respectively, when discharged. The patients were monitored by transtelephonic electrocardiographic monitoring and underwent outpatient clinic examination every 2 months.

Results

Pre-ablation electrophysiologic studies (Table 2). Of the seven patients with coronary heart disease (Cases 1 to 6 and 8), clinical ventricular tachycardia could be induced by programmed ventricular stimulation from the right or left ventricular apex in six and occurred spontaneously in one (Case 1). Endocardial mapping of clinical ventricular tachycardia showed that the earliest endocardial activation was close to the infarcted area and preceded the onset of the QRS complex by 50 to 200 ms (Fig. 1 to 3).

In Patient 7, who had no obvious heart disease, programmed atrial and ventricular stimulation failed to induce ventricular tachycardia. Mapping of one configurational type of spontaneously occurring ventricular tachycardia showed earliest endocardial activation in the high right ventricular septum, whereas mapping of ventricular premature complexes that mimicked the second type of spontaneously occurring tachycardia showed earliest activation in the right ventricular outflow tract.

Table 2. Catheter Ablation Procedures in the Eight Study Patients

	Case	First Shock	Second Shock	Third Shock	Fourth Shock	Fifth Shock	Sixth Shock
VT pattern	1	RBBB-LAD(S)	RBBB-LAD(S)	RBBB-LAD(S)	RBBB-LAD (I)	LBBB-LAD (I)	
Site of shock(T)		Inf-post LV(−60)	Inf-post LV(−70)	Inf-post LV(−85)	Inf-post LV(−80)	Inf-post LV(−120)	
Chest paddle position		Left post-lat	Left post-lat	Left post-lat	Left post-lat	Left post-lat	
Status after shock		NSR (3 min)	NSR (4 h)	NSR	NSR	NSR; frequent VPCs	
VT pattern	2	RBBB-LAD(I)	LBBB-LAD(S)				
Site of shock(T)		Inf-post LV(−50)	Inf-post LV(−90)				
Chest paddle position		Left post-lat	Left post-lat				
Status after shock		NCVT (LBBB-LAD)	Transient CAVB				
VT pattern	3	LBBB-LAD(I)	LBBB-LAD(I)	LBBB-LAD (I)			
Site of shock(T)		Inf-sept-ap LV(−75)	Inf-sept-ap LV(−75)	Inf-sept-ap LV (−90)			
Chest paddle position		Left post-lat	Left post-lat	V_4			
Status after shock		NSR + transient NCVT	NSR + frequent VPCs	NCVT→DC shock			
VT pattern	4	LBBB-LAD(I)	NSR				
Site of shock(T)		Inf-sept-ap LV(−200)	Inf-sept-ap LV				
Chest paddle position		V_4	V_4				
Status after shock		NSR, rare VPCs	NSR				
VT pattern	5	LBBB, NA(I)	LBBB, NA(I)	LBBB, NA(I)			
Site of shock(T)		Ap-sept LV(−70)	Ap-sept LV(−70)	Ap-sept LV(−70)			
Chest paddle position		V_4	V_4	V_4			
Status after shock		NSR,LBBB (hours)	Transient CAVB	NSR			
VT pattern	6	RBBB, axis −150°(I)	RBBB, axis −150°(I)	RBBB,axis −150°(I)	RBBB, RAD(I)		
Site of shock(T)		Mid-inf LV(−120)	Mid-inf LV(−100)	Mid-inf LV(−100)	Inf-post-lat LV (−95)		
Chest paddle position		Left post-lat	Left post-lat	Left post-lat	Left post-lat		
Status after shock		NSR	NSR, transient intracardiac ST depression	NSR	NSR, diffuse ischemia, LBBB, cardiogenic shock, VT/VF→DC shock		
VT pattern	7	LBBB, RAD (S*)	LBBB, RAD (S*)	LBBB, RAD(S*)	LBBB, NA (S)	LBBB, NA(S)	LBBB, NA(S)
Site of shock(T)		RVOT (−40)	RVOT (−40)	RVOT (−40)	RVS (−40)	RVS (−40)	RVS (−40)
Chest paddle position		Left post-lat	Left post-lat	Left post-lat	V_4	V_4	Left post-lat
Status after shock		NSR, transient RBBB	NSR, transient RBBB	NSR, transient RBBB + LAD, VPCs	Transient CAVB	Transient CAVB + LBBB	Transient CAVB
VT pattern	8	LBBB, LAD (I)					
Site of shock(T)		Mid inf-sept					
Chest paddle position		Left post-lat					
Status after shock		NSR, VPCs					

*Ventricular premature complexes having the same configuration as that of ventricular tachycardia. ap = apical; CAVB = complete atrioventricular block; DC = direct current; I = induced by programmed ventricular stimulation; inf = inferior; lat = lateral; LV = left ventricle; NCVT = nonclinical ventricular tachycardia; NSR = normal sinus rhythm; post = posterior; RVOT = right ventricular outflow tract; RVS = right ventricular septum; S = spontaneous; sept = septal; T = time from endocardial activity to onset of QRS complex at the site of shock(ms); VPCs = ventricular premature complexes; other abbreviations as in Table 1.

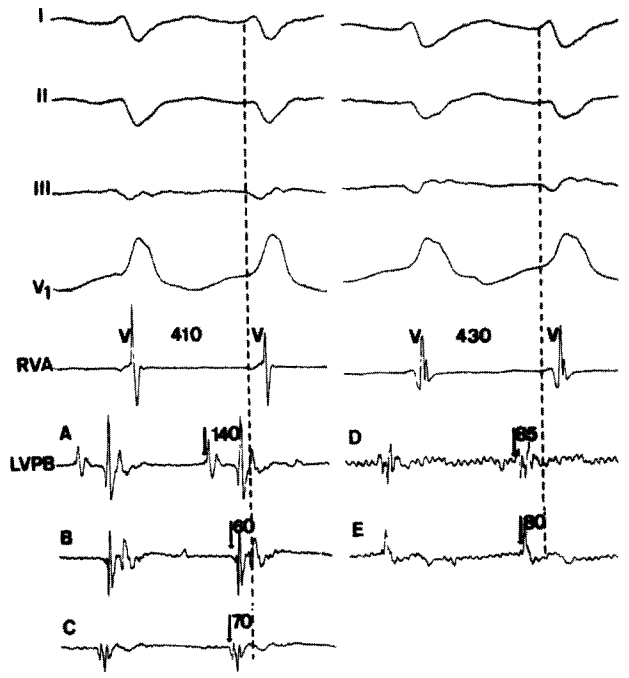


Figure 1. Patient 1. Endocardial mapping during ventricular tachycardia. Electrocardiographic leads I, II, III, V_1 and intracardiac recordings from the right ventricular apex (RVA) and the posterobasal area of the left ventricle (LVPB, **sites A** to **E**) are shown. Before catheter ablation procedures, the earliest endocardial activation is recorded in the posterobasal area of the left ventricle 140 ms before the onset of the QRS complex (**A**). Technical difficulties prevented delivery of the intracardiac shocks at this early site and therefore shocks were delivered at the endocardial sites showing less early activity. **B** and **C** represent the endocardial sites of activation where a single 250 J shock delivered during the first ablative procedure resulted in resumption of sinus rhythm for 3 minutes and 4 hours, respectively. **D** and **E** represent the endocardial sites of activation where a single 300 J shock delivered during the second ablative procedure abolished ventricular tachycardia. V = ventricular activity.

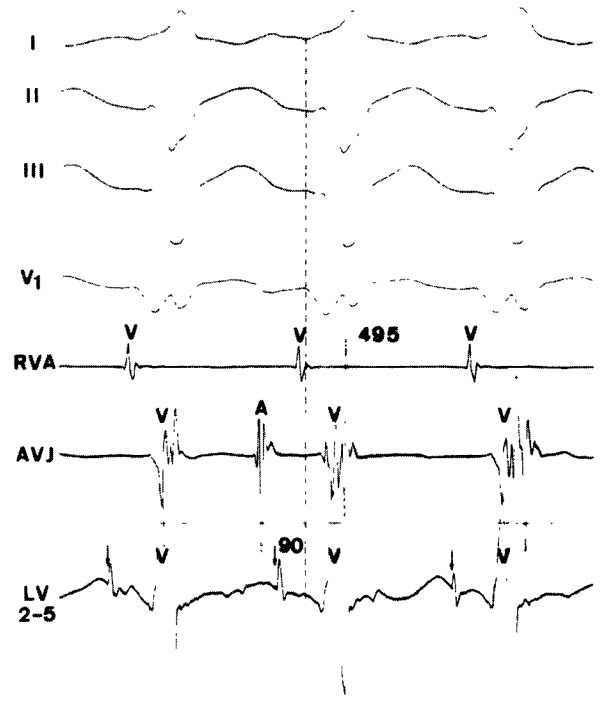
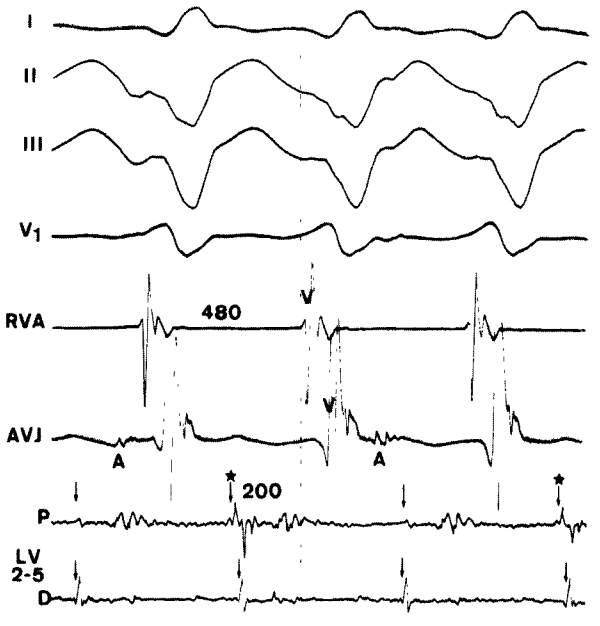


Figure 2. Patient 3. Endocardial mapping during ventricular tachycardia. Electrocardiographic leads I, II, III, V_1 and intracardiac electrograms from the right ventricular apex (RVA), atrioventricular junction (AVJ) and the inferoseptal-apical area of the left ventricle (LV) are shown. Earliest endocardial activation occurs in the latter area 90 ms before the onset of the QRS complex. A = atrial activity; V = ventricular activity.

Figure 3. Patient 4. Endocardial mapping during ventricular tachycardia. The recordings are arranged as in Figure 2; the proximal (P) and distal (D) electrograms from the inferoseptal-apical area of the left ventricle (LV) are added. The earliest endocardial activation occurs in the proximal electrogram 200 ms before the onset of the QRS complex (★). Note that a local 2:1 exit block is present in this area.



Shock ablation procedures (Table 2). A total of 26 intracardiac shocks were delivered to the eight patients: two patients received 2 shocks, two received 3 shocks and the remaining four received 1, 4, 5 or 6 shocks. Of the total of 26 shocks, 21 were delivered at the site of origin of the clinical ventricular tachycardia and 2 at the site of origin of nonclinical ventricular tachycardia close to that of the clinical tachycardia.

Effects of ablative procedures on ventricular tachycardia (Table 3). After the ablative procedures, spontaneous ventricular tachycardia did not recur in five patients (Cases 1, 2, 4, 5 and 8) during a follow-up period of 17, 16, 14, 13 and 7 months, respectively. During electrophysiologic studies performed in four of these patients 7 to 60 days after the ablative procedures, clinical ventricular tachycardia could not be induced by programmed right ventricular stimulation.

Patient 3 exhibited recurrence of ventricular tachycardia

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Table 3. Electrophysiologic Effects of Ablative Procedures in Four Patients

Case	Before Ablation	After Ablation
1	Spontaneous sustained VT	Induction of NCVT (260 beats/min) with 2 VPDs/CL 600 ms/20 mA requiring cardioversion
4	Induction of sustained CVT with 1 VPD/CL 500 ms/3 mA	No induction of VT with 1,2 VPDs/CL 700 to 400 ms/3 and 20 mA
5	Induction of sustained CVT with 2 VPDs/CL 400 ms/20 mA	No induction of VT with 1,2 VPDs/CL 700 to 400 ms/3 and 20 mA
8	Induction of sustained CVT with 1 VPD/CL 600 ms/3 mA	Induction of nonsustained (<6 seconds) pleomorphic NCVT with 2 VPDs/CL 700 to 400 ms/3 and 20 mA; no induction of CVT

CL = cycle length; CVT = clinical ventricular tachycardia; NCVT = nonclinical ventricular tachycardia; VPD = ventricular premature depolarization; VT = ventricular tachycardia.

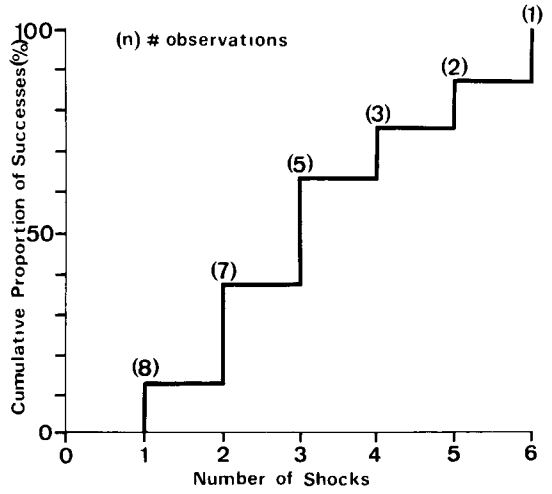
36 hours after the ablative procedure and underwent emergency aneurysmectomy with mapping-guided subendocardial resection of the arrhythmogenic area. The site of origin of tachycardia, as demonstrated during surgery, was found about 1.5 cm from the zone where three endocardial shocks were delivered. Lesions created by the shocks extended over an area of organized thrombus and consisted of small craters, 0.5 cm in diameter, that did not reach the endocardium. Ventricular tachycardia could not be induced by ventricular stimulation performed 10 days after surgery. Patient 6 developed severe myocardial ischemia with cardiogenic shock and recurrent ventricular tachycardia/ventricular fibrillation a few minutes after the second ablative procedure (see later). After prolonged resuscitative maneuvers, he recovered. While considering coronary revascularization surgery, he died suddenly 2 months later, after complaining of chest pains. Nonsustained ventricular tachycardia (<6 complexes) was frequently recorded during transtelephonic electrocardiographic monitoring after the ablative procedure. Patient 7 exhibited recurrent nonsustained ventricular tachycardia after the ablative procedures. However, only the ventricular tachycardia presumably originating from the right ventricular outflow tract was observed. This arrhythmia was subsequently well controlled by flecainide therapy.

Analysis of the relation of the number of shocks to the success of the procedure shows that 75% of the patients were successfully treated with three or fewer shocks (Fig. 4).

Immediate complications of ablative procedures. Disturbances of cardiac rhythm and conduction were frequently seen immediately after the ablative procedures (Table 2). Nonclinical ventricular tachycardia was observed after intracardiac shock in two patients (Cases 2 and 3) and required cardioversion in one. Intraventricular and atrioventricular conduction disturbances were noted in four patients (Cases 2, 5, 6 and 7); in three, these disturbances were transient, disappearing within minutes to days, but in one patient (Case 6), left bundle branch block persisted.

Severe hemodynamic deterioration was noted in one patient (Case 6). Three days after uncomplicated administration of a series of three intracardiac shocks to ablate one configurational type of clinical ventricular tachycardia, an attempt was made to ablate a second configurational type. A single 300 J shock was given during well tolerated ventricular tachycardia of 10 minutes' duration. On restoration of sinus rhythm, diffuse and marked ST-T wave changes appeared and the patient rapidly developed severe cardiogenic shock with recurrent episodes of ventricular fibrillation requiring multiple external cardioversions. After a pro-

Figure 4. Relation of the number of shocks to the success of the ablative procedure. The **abscissa** indicates the number of shocks delivered and the **ordinate** indicates the cumulative proportion of patients who successfully responded after that number of shocks.



longed resuscitative procedure, including administration of high doses of inotropic agents, the patient recovered without evident cardiac or neurologic impairment.

In one patient (Case 2), a marked but brief (<2 minutes) subendocardial injury pattern was recorded on the intracardiac electrogram close to the site where the shock was delivered. This was not associated with any significant ST-T wave changes on the surface electrocardiographic leads.

Clinical and electrocardiographic course. In Patients 1 and 7, a pericardial rub heard the day after delivery of the shocks disappeared within a few days. Patient 1 developed two episodes of left heart failure 3 days after the ablative procedure; both episodes promptly responded to intravenous administration of furosemide. During the follow-up period, the patient gave a history of two episodes of paroxysmal nocturnal dyspnea. The occurrence of congestive heart failure after the ablative procedure contrasted with its absence during a 3 day period of incessant ventricular tachycardia before the shocks. In the remaining patients, there were no significant clinical or electrocardiographic changes.

Assessment of myocardial damage. The left ventricular ejection fraction, as measured by radionuclide ventriculography before and after the ablative procedure in seven patients, significantly decreased in two patients (from 26 to 20% in Patient 2 and from 19 to 12% in Patient 4). In the remaining five patients, the left ventricular ejection fraction remained unchanged (33 ± 8% before and after ablative procedures). New development of left ventricular contraction abnormalities was observed in only one patient (Case 2), who also exhibited permanent left bundle branch block after the ablative procedure. The peak serum creatine kinase level after the ablative procedure ranged from 60 to 1,000 IU with MB fraction ranging from 3.4 to 8.7%.

Discussion

An important innovation in the management of patients with drug-refractory cardiac arrhythmias has been the ability to ablate these arrhythmias, or the sites critical to their maintenance, by endocardial shocks delivered through electrode catheters. These procedures have been extensively and successfully used for ablating the atrioventricular (AV) junction to control a rapid ventricular response during atrial tachyarrhythmias or drug-refractory AV reentrant tachycardias (14–16). In contrast, experience with the use of these procedures to ablate ventricular tachycardia is limited. To date there have been only a few studies, most of them including a small number of patients (4–11).

Previous reports on catheter ablation of ventricular tachycardia. Hartzler (4) was the first to report catheter ablation of ventricular tachycardia in three patients. Ventricular tachycardia originated in the right ventricular outflow tract in one patient with no organic heart disease and in the left ventricular septum in two patients with previous myocardial infarction. After the ablative procedure, ventricular tachycardia did not recur during a 12 month follow-up period in the patient without organic heart disease. In one of the patients with coronary heart disease, ventricular tachycardia was no longer inducible during electrophysiologic study and did not recur for 5 months. In the remaining patient, clinical ventricular tachycardia did not recur for 3 months before the patient's death from low output congestive heart failure. Puech et al. (6) described a patient with arrhythmogenic right ventricular dysplasia in whom a single endocardial shock prevented ventricular tachycardia during a 12 month follow-up period. More recently, Ruffy et al. (10) described successful ablation of a fascicular ventricular tachycardia in a patient with previous myocardial infarction. Of the 16 patients who underwent catheter ablation of ventricular tachycardia and were reported on in abstract form (7–9), six did not exhibit recurrence of ventricular tachycardia during a follow-up period of up to 12 months.

Results of the present study. In the present study, we attempted transcatheter shock ablation of ventricular tachycardia in eight patients. In seven of the eight, the tachycardia was drug-resistant and even refractory to repeated external cardioversions in one patient. In the remaining patient, spontaneous ventricular tachycardia did not recur during antiarrhythmic drug therapy, but catheter ablation of ventricular tachycardia was attempted because no antiarrhythmic drug prevented induction of sustained rapid ventricular tachycardia by programmed ventricular stimulation.

Catheter ablative procedures prevented spontaneous occurrence of ventricular tachycardia in five of our eight patients during a follow-up period ranging from 7 to 17 months. The most dramatic results were observed in Patient 1, who had incessant ventricular tachycardia refractory to repeated external cardioversions before the ablative procedure (11). In addition, in all four patients who underwent electrophysiologic studies after the ablative procedure, programmed ventricular stimulation failed to induce the clinical ventricular tachycardia, suggesting a definite cure of the arrhythmia. Interestingly, four of the five patients in whom the ablative procedure succeeded were high risk surgical candidates: one patient (Case 1) had a 3 week old myocardial infarction and ventricular tachycardia originating in the inferior wall (17); the three remaining patients had a previous myocardial infarction and severe ventricular dysfunction. Therefore, catheter ablation of ventricular tachycardia appears to represent a promising method of treatment in patients with ventricular tachycardia who are high risk candidates for surgery.

Factors affecting success in ablative procedures. Considering that catheter endocardial mapping allows localization of the origin of ventricular tachycardia with an accuracy of approximately 4 cm^2 (12), it can be assumed that the success of ablative procedures and the number of

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shocks required to ablate the arrhythmia are highly dependent on the adequate localization of the tachycardia and on administering the shock at the area that demonstrates the earliest activation. This assumption is supported by the following observations in three of our patients: in Patients 4 and 8 a very early endocardial activation during ventricular tachycardia was found (200 and 100 ms, respectively, before the onset of the QRS complex) (Fig. 3) and a single intracardiac shock was successful. However, in Patient 1, technical difficulties prevented delivery of shocks at the site showing the earliest endocardial activation during ventricular tachycardia (140 ms before the onset of the QRS complex). In this patient, four shocks were given close to the site of origin of the tachycardia (at the earliest endocardial activation of 60 to 85 ms before onset of the QRS complex) and the multiple shocks were apparently necessary to ablate the tachycardia.

Complications. The incidence of reported complications after catheter ablation of ventricular tachycardia has been variable. In a study of four patients, Hartzler and Giorgi (5) noted transient sinus bradycardia, right bundle branch block and spontaneously terminating ventricular fibrillation. No complications were reported by Puech et al. (6) and Downar et al. (9) in one and four patients, respectively. Steinhaus et al. (7) did not observe any major complication or demonstrable change in left ventricular function in six patients. Winston et al. (8) reported transient aphasia in one of their six patients. Ruffy et al. (10) noted a life-threatening new form of ventricular tachycardia in one patient during the first 24 hours after the ablative procedure. In the present study we observed a high incidence of complications that appeared to be directly related to the ablative procedure. Transient electrical instability, as manifested by nonclinical ventricular tachycardia, occurred in two patients. This has been frequently observed in dogs after endocardial ablation (18–20). Atrioventricular and intraventricular conduction disturbances were noted in four patients but were transient in three of the four. The most disturbing complication was the hemodynamic impairment observed in three patients: left-sided heart failure (one patient), and a significant decrease in left ventricular ejection fraction (two patients) with development of ventricular contraction abnormalities in one patient. In the normal canine ventricle, two discharges of 200 J have been shown to produce endocardial necrosis 9 mm in depth and 18 mm in breadth (20). Although endocardial lesions caused by shocks may be different in normal canine and scarred human ventricles, our results suggest that a substantial area of myocardial necrosis is created by multiple shocks of 300 J in the human ventricle.

Although developing immediately after the ablative procedure, the cardiogenic shock in Patient 6 was probably not related to the shock itself, but rather to myocardial ischemia caused by prolonged endocardial mapping of ventricular tachycardia. Indeed, this patient had previously had several episodes of prolonged hypotension that occurred after ischemic episodes that were not associated with ventricular tachycardia.

Conclusions. Our preliminary experience suggests that transcatheter shock ablation of ventricular tachycardia represents an important innovation in the management of patients with drug-refractory ventricular tachycardia. This procedure may be an alternative to antitachycardia surgery in high risk candidates for surgery. However, the high incidence of complications observed should temper any temptation to consider routine use of this procedure in patients with ventricular tachycardia. Because only limited experience has been reported so far, we recommend that only those patients with recurrent, sustained ventricular tachycardia refractory to antiarrhythmic drugs should be considered for this procedure. Extreme caution should prevail in patients with severely impaired left ventricular function. Future efforts should be directed to other modes of ablation, such as laser ablation, which creates controlled endocardial lesions with less energy and produces less deleterious effects than those produced by transcatheter electrode shock (19).

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