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CLINICAL STUDIES

ELECTROPHYSIOLOGY

Radiofrequency Catheter Ablation for Management of Symptomatic Ventricular Ectopic Activity

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Objectives. **This study assessed the useful role of intracardiac mapping and radiofrequency catheter ablation in eliminating drug-refractory monomorphic ventricular ectopic beats in severely symptomatic patients.**

Background. **Ventricular ectopic activity is commonly encountered in clinical practice. Usually, it is not associated with life-threatening consequences in the absence of significant structural heart disease. However, frequent ventricular ectopic beats can be extremely symptomatic and even incapacitating in some patients. Currently, reassurance and pharmacologic therapy are the mainstays of treatment. There has been little information on the use of catheter ablation in such patients.**

Methods. **Ten patients with frequent and severely symptomatic monomorphic ventricular ectopic beats were selected from three tertiary care centers. The mean frequency ± SD of ventricular ectopic activity was 1,065 ± 631 beats/h (range 280 to 2,094) as documented by baseline 24-h ambulatory electrocardiographic (ECG) monitoring. No other spontaneous arrhythmias were documented. These patients had previously been unable to tolerate or had been unsuccessfully treated with a mean of 5 ± 3 antiarrhythmic drugs. The site of origin of ventricular ectopic activity was accurately mapped by using earliest endocardial activation time during ectopic activity or pace mapping, or both.**

Results. **During electrophysiologic study, no patient had inducible ventricular tachycardia. The ectopic focus was located in the right ventricular outflow tract in nine patients and in the left ventricular posteroseptal region in one patient. Frequent ventricular ectopic beats were successfully eliminated by catheter-delivered radiofrequency energy in all 10 patients. The mean number of radiofrequency applications was 2.6 ± 1.3 (range 1 to 5). No complications were encountered. During a mean follow-up period of 10 ± 4 months, no patient had a recurrence of symptomatic ectopic activity, and 24-h ambulatory ECG monitoring showed that the frequency of ventricular ectopic activity was 0 beat/h in seven patients, 1 beat/h in two patients and 2 beats/h in one patient.**

Conclusions. **Radiofrequency catheter ablation can be successfully used to eliminate monomorphic ventricular ectopic activity. It may therefore be a reasonable alternative for the treatment of severely symptomatic, drug-resistant monomorphic ventricular ectopic activity in patients without significant structural heart disease.**

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Ventricular ectopic activity in the presence of minimal or no structural heart disease is commonly encountered in clinical practice (1). Although characteristically not associated with life-threatening consequences, frequent ventricular ectopic beats can be extremely symptomatic and even incapacitating in some persons (2). For such patients, reassurance and pharmacologic therapy are currently the mainstays of treatment. The development of radiofrequency catheter ablation has provided a safe and effective method for curing most types of supraventricular tachycardias (3–10). In addition, ablation of idiopathic ventricular tachycardias has become a viable alternative to drug therapy (11–13). It remains uncertain whether isolated monomorphic ventricular ectopic activity can be effectively eliminated by catheter-delivered radiofrequency energy. To test the hypothesis that monomorphic ventricular ectopic activity has a focal origin that can be accurately localized and safely eliminated, we performed intracardiac mapping and radiofrequency catheter ablation in 10 patients with frequent and severely symptomatic monomorphic ventricular ectopic beats that failed to be suppressed by multiple medications.

Methods

Study patients. The study group comprised 10 patients recruited from patients referred for management of symptomatic ventricular ectopic activity at three tertiary care centers between January 1993 and January 1994. The selection criteria included 1) frequent symptoms that were clearly related to

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monomorphic ventricular ectopic activity and could not be relieved by reassurance; 2) inability of the patient to tolerate or unsuccessful treatment with at least three antiarrhythmic drugs; 3) no evidence of other cardiac arrhythmias; 4) no evidence of significant structural heart disease; 5) absence of electrolyte abnormalities, metabolic disorders or advanced systemic diseases. The clinical characteristics of the 10 patients are shown in Table 1. There were four men and six women, with a mean age ± SD of 46 ± 10 years (range 29 to 56). Seven patients had no apparent structural heart disease; one patient had mitral valve prolapse and mild mitral regurgitation, one patient had mild left ventricular enlargement with decreased left ventricular systolic function (left ventricular ejection fraction 48%) and one had "focal wall thinning with wall motion abnormality of the anterior right ventricle" by magnetic resonance imaging. All patients experienced frequent episodes of palpitation, fatigue, dyspnea and light-headedness. The symptoms persisted for a mean of 10 ± 10 years (range 1 to 29). Baseline 24-h ambulatory electrocardiographic (ECG) monitoring showed frequent monomorphic ventricular ectopic activity in each patient (mean 1,065 ± 631 ventricular ectopic beats/h; range 280 to 2,094). Direct correlation between the occurrence of symptoms and ventricular ectopic activity was documented by means of ambulatory electrocardiography in all patients. There were occasional ventricular ectopic couplets and triplets in some patients. No other cardiac arrhythmias were documented. The ventricular ectopic beats had a left bundle branch block pattern and inferior axis in nine patients and a right bundle branch block pattern and superior axis in one patient. Because of their disturbing symptoms, all patients had frequent clinic visits and multiple hospital admissions to initiate or adjust antiarrhythmic drugs for ventricular ectopic activity. Patients had previously received a mean of 5 ± 3 antiarrhythmic drugs (range 3 to 11), including beta-adrenergic blocking agents, calcium channel blocking agents, class IA, IB, IC and class III agents alone or in combination. These agents either could not be tolerated or did not provide adequate suppression of ventricular ectopic beats. Thus, intracardiac mapping was attempted, and radiofrequency catheter ablation was performed to eliminate the site of origin of the monomorphic ventricular ectopic activity in these patients.

Electrophysiologic study. All antiarrhythmic medications were discontinued for at least 5 half-lives before baseline electrophysiologic evaluations were performed. After written informed consent was obtained, standard electrophysiologic study was undertaken with the use of local anesthesia and mild intravenous sedation with the patient in the fasting state. Quadripolar electrode catheters (6F) were advanced from the right femoral vein and positioned in the low septal right atrium, right ventricular apex and outflow tract. A 12-lead surface ECG was monitored and recorded on a computer-based digital amplifier/recorder system with optical disk storage (ART, BARD, Biomedical Instrumentation). Bipolar intracardiac electrograms were filtered with a band-pass of 30 to 500 Hz. Intracardiac bipolar pacing was performed at twice diastolic threshold with a pulse duration of 2.0 ms. Pro-

Table 1. Clinical and Electrophysiologic Characteristics at Baseline and at Follow-Up

Pt No.	Age (yr)/ Gender	Symptoms	Duration of Symptoms (yr)	Previous Antiarrhythmic Drugs (no.)	Structural Heart Disease	VE Beats			EAT (ms)	Pace Map	RF Catheter Ablation			Follow-Up	
						No./h	Configuration	Site			No. of Applications	Guided by	Mo	VE Beats (no./h)
1	34/F	P,D	1.5	8	—	1,645	LBB/Inf	RVOT	−26	12	2	EAT	8	0
2	50/M	P,D	2.5	7	MVP,MR	711	LBB/Inf	RVOT	−22	12	3	EAT	6	2
3	29/M	P,D,PS	12	4	Mild CM	1,709	LBB/Inf	RVOT	−36	12	1	EAT	6	0
4	55/F	P,D,PS	5	3	—	1,366	RBB/Sup	LVPS	−10	10	3	EAT	14	0
5	54/F	P,FG	29	3	—	1,003	LBB/Inf	RVOT	−33	12	2	EAT	14	1
6	52/M	P,FG	5	11	—	2,094	LBB/Inf	RVOT	−30	12	1	EAT	12	1
7	46/F	P,D,PS	1	3	—	340	LBB/Inf	RVOT	...	12	3	Pace map	10	0
8	56/F	P,D,PS	7	5	—	420	LBB/Inf	RVOT	...	12	4	Pace map	7	0
9	50/F	P,FG	1.5	4	*	1,082	LBB/Inf	RVOT	...	12	5	Pace map	4	0
10	36/M	P,FG	8	3	—	280	LBB/Inf	RVOT	...	12	2	Pace map	15	0
Mean	46		10	5		1,065			−26		2.6		10	0.4
SD	10		10	3		631			9		1.3		4	

*Magnetic resonance imaging in this patient showed focal wall thining with wall motion abnormality of the anterior right ventricle. CM = cardiomyopathy; D = dyspnea; EAT = earliest endocardial activation time during ventricular ectopic activity; F = female; FG = fatigue; Inf = inferior axis; LBB = left bundle branch block configuration; LVPS = left ventricular posterior septum; M = male; MR = mitral regurgitation; MVP = mitral valve prolapse; P = palpitation; PS = presyncope; RBB = right bundle branch block configuration; RF = radiofrequency catheter ablation; RVOT = right ventricular outflow tract; Sup = superior axis; VE = ventricular ectopic; — = none; ... = not measured.

grammed electrical stimulation with up to three extra stimuli at two drive cycle lengths and rapid burst pacing was performed from the right ventricular apex and outflow tract. In seven patients, isoproterenol infusion was then initiated at 1 μg/min and increased to a maximal rate of 5 μg/min in 1-μg/min increments to achieve at least a 20% increase in heart rate. Programmed electrical stimulation and rapid burst pacing were repeated during isoproterenol infusion in these patients. The frequency of ventricular ectopic activity was compared between the baseline state and patients. The frequency of ventricular ectopic activity in the baseline state and during isoproterenol infusion at the highest dose was compared.

Mapping and ablation of ventricular ectopic foci. Detailed mapping and ablation of the ventricular ectopic focus were performed using a 7F or 8F quadripolar catheter with a deflectable tip and a 4- or 5-mm distal electrode (Webster; EP Technology; Elecath). The mapping catheter was introduced from the right femoral vein and advanced into the right ventricular outflow tract in nine patients with ventricular ectopic beats that had a left bundle branch block with inferior axis pattern. In the one patient whose ectopic beats had a right bundle branch block, superior axis configuration, the mapping catheter was introduced from the right femoral artery and advanced to the mid-left ventricular septum posteriorly. Heparin administration was limited to the latter patient. The position of the mapping catheter tip was guided fluoroscopically with the use of both right and left anterior oblique views. Mapping for the earliest site of endocardial activation during spontaneous ventricular ectopic activity was performed initially. Pace mapping was performed at endocardial sites showing early activation (local endocardial potentials earlier than the surface QRS recording) during ventricular ectopic activity. Bipolar pace mapping was performed between the distal pair of electrodes (2-mm spacing). The site of ablation was chosen at the site of earliest endocardial activation during ventricular ectopic activity or from a pace map that best reproduced the spontaneous ventricular ectopic configuration on surface 12-lead ECG recordings, or both. The power source for endocardial ablation was a commercially available electrosurgical generator that continuously delivered unmodulated radiofrequency current at 500 kHz (model RFG-3C, Radionics). The cathode was the distal electrode of the deflectable tip catheter used for mapping. The anode was an adhesive cutaneous electrocautery dispersive pad placed on the thorax. Radiofrequency energy was delivered at 20 to 30 W for 15 to 20 s during spontaneous rhythm. If ventricular ectopic beats disappeared or its frequency transiently increased, energy application was continued for up to 60 s. If a sudden increase in impedance occurred, energy delivery was terminated immediately, the catheter removed and any adherent coagulum cleaned from the tip. Successful ablation was defined as complete elimination of the spontaneous monomorphic ventricular ectopic activity with and without isoproterenol infusion for at least 30 min after the last application of radiofrequency energy.

Follow-up. After the ablation procedure, all patients were monitored in the hospital for 24 h. An ECG was obtained before discharge. Patients were given no antiarrhythmic drugs and were evaluated periodically for symptoms at clinic visits. A 12-lead ECG and 24-h ambulatory ECG monitoring were repeated at 3- to 6-month intervals or if the patient had symptoms suggestive of recurrence of ventricular ectopic activity.

Statistical analysis. Continuous variables are presented as mean value ± SD. A paired Student t test was used to analyze the statistical significance of reduction of the frequency of ventricular ectopic activity on 24-h ambulatory ECG monitoring during follow-up after successful ablation. A two-tailed t value ≤ 0.05 was considered significant.

Results

Electrophysiologic study. Spontaneous frequent monomorphic ventricular ectopic activity was present at the time of the electrophysiologic study. However, nonsustained or sustained monomorphic ventricular tachycardias were not inducible by programmed electrical stimulation in any patient. Among the seven patients who received isoproterenol infusion, the frequency of ventricular ectopic beats increased in three patients, decreased in three and was unchanged in the remaining patient. Ventricular tachycardia remained noninducible in these patients during isoproterenol infusion.

Mapping and ablation of ventricular ectopic foci. In nine patients whose ventricular ectopic activity had a left bundle branch block configuration with an inferior axis, the site of earliest endocardial activation during ventricular ectopic activity was located in the right ventricular outflow tract (Table 1). In two of the nine this site was just below the pulmonary valve and one had a focus ~1 cm superior to the His bundle. In the patient with a right bundle branch block, superior axis pattern, the site of earliest endocardial activation was in the left ventricular posteroseptal region. In five patients with right ventricular outflow tract foci, ablation was guided by endocardial activation mapping. The earliest endocardial activation at the target site was 22 to 36 ms (mean 28 ± 7 ms) before the onset of the surface ectopic QRS complex (Fig. 1). Pace mapping at that site also produced an identical match in all 12 surface leads. In the other four patients with right ventricular outflow tract foci, the ablation was guided by pace mapping. Detailed pace mapping was carried out at the endocardial sites where local activation time was earlier than the surface QRS complex during the ectopic activity. The successful site showed an identical QRS complex in all 12 surface leads (Fig. 2). In the patient with a left ventricular focus, the earliest endocardial activation was located in the posteroseptal region ~1 cm from the apex and was only 10 ms earlier than the onset of the surface QRS complex. Pace mapping at that site produced an identical configuration in only 10 of 12 leads. A fascicular potential was not observed. Delivery of radiofrequency energy at that site eliminated the ectopic activity. After the successful ablation, the tip of the ablation catheter was pulled back ~0.5 cm toward the midposteroseptal region, where a sharp deflection preceding local ventricular activation became evi-

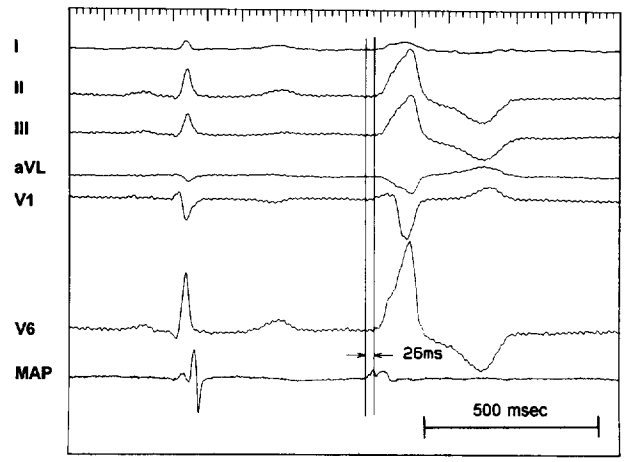

Figure 1. Patient 1. The earliest ventricular activation during ectopic activity was recorded from the distal pair of electrodes (MAP) on a quadripolar deflectable mapping catheter (2–5–2-mm interelectrode spacing) positioned in the right ventricular outflow tract. As indicated by **arrows**, the onset of local ventricular activation during ventricular ectopic activity was 26 ms earlier than the QRS complex on the surface electrocardiogram. Delivery of radiofrequency energy at this site eliminated the ectopic activity.

dent, probably representing a fascicular potential. An additional application of radiofrequency energy was administered at that site.

In three patients, it was noticed that ventricular ectopic activity disappeared transiently when the ablation catheter tip was positioned firmly at the successful site before radiofrequency energy was applied. During the delivery of energy, five patients had a sudden increase in the frequency of ventricular ectopic activity and developed nonsustained ventricular tachycardia of similar configuration lasting several seconds before the ventricular ectopic activity was permanently eliminated (Fig. 3).

The mean number of radiofrequency energy applications required for complete elimination of ventricular ectopic activity was 2.6 ± 1.3. The duration of radiofrequency application before disappearance of ventricular ectopic beats during the successful ablation was always <15 s. Energy application was then continued for 60 s. An additional application of radiofrequency energy was usually administered at the same site for 30 to 60 s after the successful ablation.

Follow-up. The follow-up data are summarized in Table 1. The ventricular ectopic activity was eliminated in all 10 patients during the procedure (Fig. 4). The immediate success rate was 100%, and no complications were associated with the procedure. During a mean follow-up period of 10 ± 4 months (range 6 to 15), no patient had a recurrence of symptomatic ectopic activity. Repeat 24-h ambulatory ECG monitoring was performed after 3 to 6 months. The frequency of ventricular ectopic activity was 0 beat/h in seven patients, 1 beat/h in two patients and 2 beats/h in one patient. The configuration of the rare ventricular ectopic beats in the latter patient at follow-up was different from that of the original ectopic activity. The

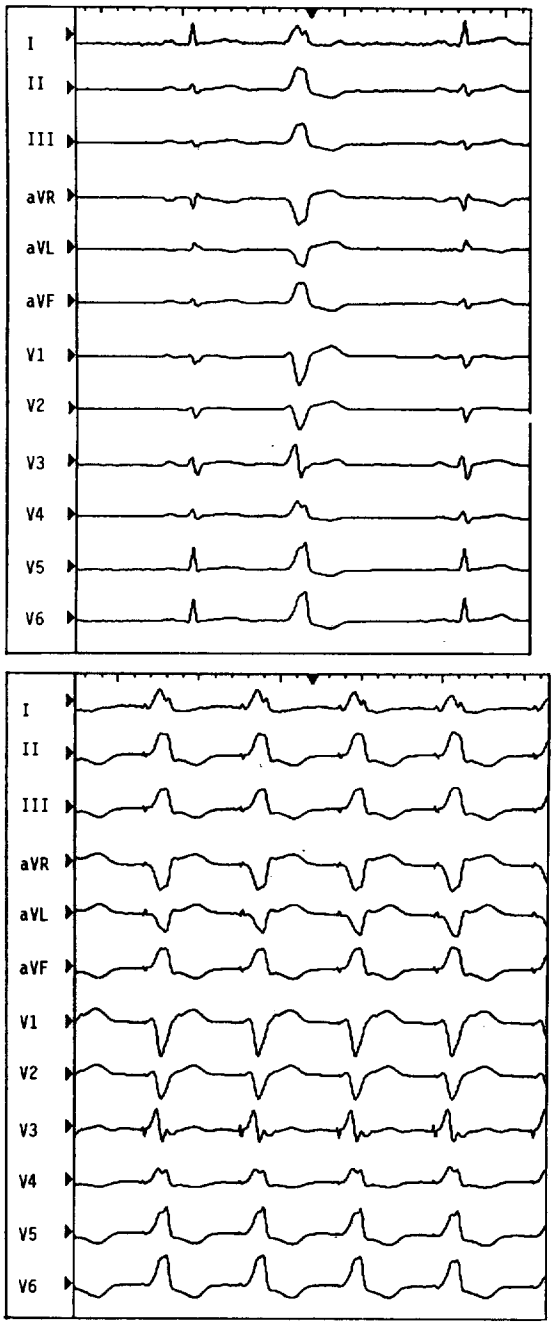

Figure 2. Patient 3. Comparison of a 12-lead electrocardiogram with spontaneous ventricular ectopic activity **(top panel)** and a pace map **(bottom panel)** at the successful ablation site in the right ventricular outflow tract. Note the nearly identical configuration of the paced QRS complexes and the ventricular ectopic activity in all leads. This was the same site at which earliest endocardial activation was recorded during ventricular ectopic activity.

average frequency of ventricular ectopic activity during follow-up (0.4 beats/h) was significantly less than that before ablation (1,065 ± 631 beats/h) ($p < 0.0001$). The patients did not report any symptoms related to the rare ectopic beats. Thus, the immediate success of ablation was maintained in the intermediate follow-up period.

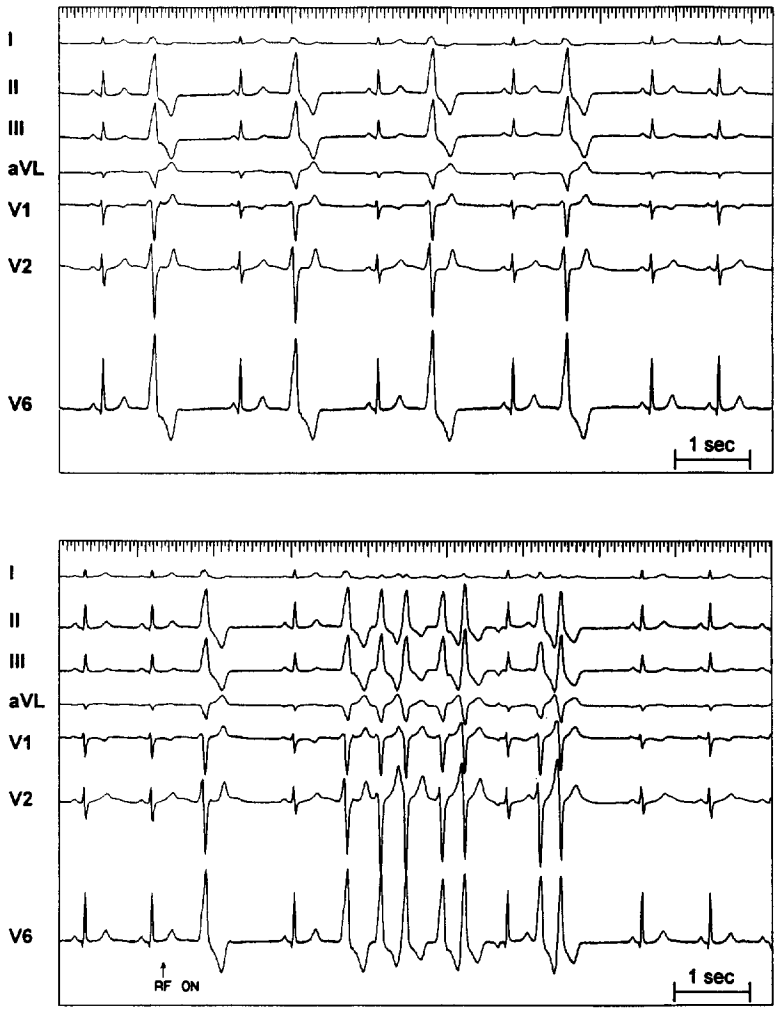

Figure 3. Patient 4. Electrocardiographic tracings. **Top,** Representative tracing of spontaneous ventricular ectopic activity before ablation. **Bottom,** During radiofrequency energy delivery (RF ON), there was a transient increase in the frequency of ventricular ectopic activity and development of nonsustained tachycardia of similar configuration followed by permanent elimination of ventricular ectopic activity.

Discussion

This study demonstrates that frequent severely symptomatic monomorphic ventricular ectopic activity in patients without significant structural heart disease originates from a focal site that can be accurately localized by intracardiac mapping and eliminated successfully by radiofrequency catheter ablation. All of our patients are free of recurrence during a mean follow-up period of 10 ± 4 months.

Ventricular ectopic activity and symptoms. The majority of monomorphic ventricular ectopic activity is probably benign, especially in younger people. Treatment usually is not neces-

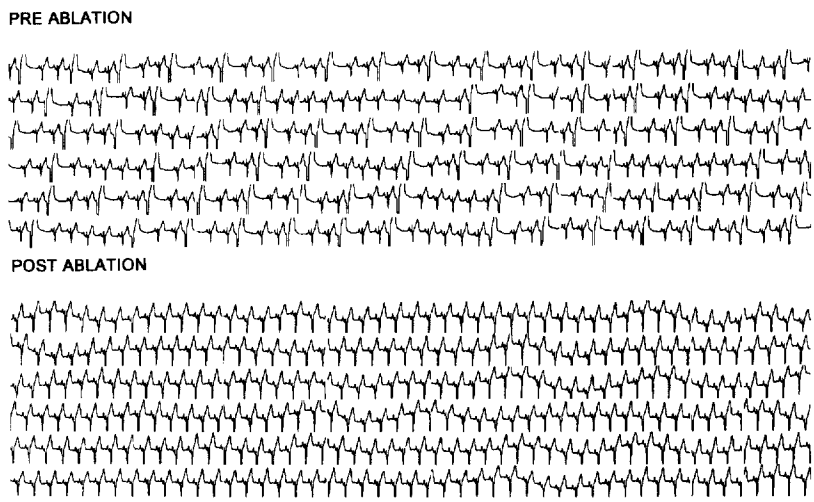

Figure 4. Ambulatory electrocardiographic tracings from Patient 1, who presented with very frequent ventricular ectopic beats. **Top tracing** is representative of spontaneous arrhythmia before ablation. After ablation **(bottom tracing),** all spontaneous ventricular ectopic beats were eliminated.

sary. When the ectopic activity is symptomatic, the symptoms should first be addressed by allaying the patient's anxiety with reassurance. If this is not successful, the frequency of the ectopic beats may be reduced by administration of beta-blockers, calcium channel blockers or other antiarrhythmic agents (14). Ventricular ectopic activity may not always be suppressible by antiarrhythmic agents. These agents may have potential side effects and may exacerbate the arrhythmias.

Although radiofrequency catheter ablation has become the therapy of choice for drug-refractory idiopathic ventricular tachycardia, its role in the management of symptomatic monomorphic ventricular ectopic activity remains uncertain. Until now, only two case reports (15,16) have described three patients in whom radiofrequency energy was successfully used to eliminate symptomatic "benign" ventricular ectopic activity. Our study demonstrated that such drug-resistant frequent monomorphic ventricular ectopic activity was effectively eliminated by catheter-delivered radiofrequency energy without major risk in a series of 10 consecutive symptomatic patients. The therapeutic end point for patients with benign but symptomatic ventricular ectopic activity should be the elimination of symptoms. Awareness of ectopic activity varies tremendously from patient to patient. Because symptoms such as palpitation, dyspnea, fatigue and dizziness can be quite nonspecific, substantiating a causal relation between the symptoms and occurrence of ventricular ectopic activity is crucial before embarking on a catheter ablation procedure. In addition, other concurrent arrhythmias, such as frequent atrial ectopic activity or paroxysmal atrial fibrillation, need to be ruled out. If these conditions are met, a high likelihood of symptomatic relief can be expected after the elimination of ventricular ectopic activity. In our series, every patient had immediate relief of symptoms after successful elimination of the ventricular ectopic focus and remained asymptomatic during an intermediate follow-up period.

Ventricular ectopic activity and idiopathic ventricular tachycardia. Idiopathic ventricular tachycardia usually originates near the right ventricular outflow tract or from the posteroseptal aspect of the left ventricle (17). The monomorphic ventricular ectopic activity in our patients originated from these similar sites as well. In general, the mechanisms responsible for the genesis of ventricular ectopic depolarizations are controversial and probably vary from patient to patient. Reentry, abnormal automaticity and triggered activity have been postulated (18). In this regard, it should be noticed that during idiopathic ventricular tachycardia, there is no evidence for isolated mid-diastolic potentials or zones of slow conduction as is characteristically found in patients with previous myocardial infarction and reentrant ventricular tachycardia (19,20). Similarly, we did not find isolated mid-diastolic potentials in any of our 10 patients with ventricular ectopic activity. The lack of mid-diastolic potentials is more consistent with a focal mechanism for these arrhythmias, such as abnormal automaticity or triggered activity, although microreentry involving very small circuits cannot be ruled out completely. The similarities regarding configuration, site of origin, electrophysiologic findings and response to therapy suggest that isolated ventricular ectopic activity and sustained ventricular tachycardia probably represent the two ends of the spectrum of idiopathic ventricular arrhythmias, sharing similar mechanisms and substrates. Conversion from one to the other has been documented (21). Although it has been previously reported (11) that ventricular ectopic beats of the same configuration as the ventricular tachycardia could be used as a target for pace mapping or localizing the earliest site of activation when clinical sustained ventricular tachycardia could not be induced, our series of patients is unique in that symptomatic frequent ventricular ectopic activity itself instead of ventricular tachycardia was the target of ablation. Our study shows that both earliest endocardial activation site and pace mapping can be used to guide the ablation of monomorphic ventricular ectopic activity. From our limited experience, it appears that the focus of ventricular ectopic activity is easier to eliminate than the focus of idiopathic ventricular tachycardia. Although it has been reported (11,13) that an average of eight applications of radiofrequency energy were required to ablate idiopathic ventricular tachycardia foci, two to three applications were usually adequate to eliminate ventricular ectopic activity in our series. This observation may imply that the idiopathic ventricular ectopic foci are smaller or located more superficially in the endocardium.

Clinical and financial implications. Ventricular ectopic activity is probably the most common arrhythmia encountered in clinical practice. In symptomatic patients, its impact on perceived quality of life can be considerable. Furthermore, symptomatic benign ventricular ectopic activity is associated with a substantial consumption of health care resources, as asserted by the frequent clinic visits, repeated ambulatory ECG monitoring and hospital admissions in our patients to initiate, adjust or change antiarrhythmic drugs. If our favorable short-term outcome of radiofrequency catheter ablation is confirmed and extended by other investigators, it may be reasonable to prospectively compare early ablation versus conventional antiarrhythmic therapy in patients with severely symptomatic monomorphic ventricular ectopic activity, using quality of life and medical costs as outcome measures.

Study limitations. The major limitations of this study are the small study group and the relatively short period of follow-up. Even though the effect of radiofrequency catheter ablation appears to be sustained in all patients for ≥6 months after the procedure, the lack of knowledge of long-term consequences in these patients necessitates continued follow-up. The small number of patients might reflect our stringent selection criteria. Although finding no failures in a small group of patients is encouraging, it does not guarantee adequate safety or efficacy statistically (22,23). With no failure in 10 patients, the 95% confidence interval for the failure rate may still extend up to 30%. Thus, the favorable outcome in our patients needs to be confirmed in a larger patient population.

Our study used no randomized control group for comparison. However, the frequency of ventricular ectopic activity as well as symptoms these patients experienced before the abla-

tion has served as an effective means of assessing the efficacy of the therapy.

Ventricular ectopic activity originated in the right ventricular outflow tract in 9 of our 10 study patients. This observation may represent a selection bias, triggered by the prior favorable experience in our centers with ablation of ventricular tachycardia originating from that region, and its overall easier accessibility. However, a predominance of right ventricular origin for ventricular ectopic activity in patients without heart disease has been well described (24,25). The true incidence of right versus left ventricular origin in patients with benign ventricular ectopic activity needs further clarification.

Conclusions. Radiofrequency catheter ablation appeared to be an appropriate management option for our 10 patients with symptomatic, drug-resistant monomorphic ventricular ectopic activity. The ventricular ectopic foci were located in the right ventricular outflow tract in the majority of patients. The mapping and ablation techniques used are similar to those used for idiopathic ventricular tachycardia. Although the procedures were performed by multiple operators from different centers, the success rate was high and no complications occurred. The symptoms related to frequent ventricular ectopic activity were resolved. Appropriate selection of patients is important to achieve an optimal clinical outcome. Until further experience is accumulated, we believe that this therapeutic approach should be performed by experienced electrophysiologists and reserved for severely symptomatic patients who do not respond to standard treatment.

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