Late Potentials Are Unaffected by Radiofrequency Catheter Ablation in Patients with Ventricular Tachycardia

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TWIDALE, N., ET AL.: Late Potentials Are Unaffected by Radiofrequency Catheter Ablation in Patients with Ventricular Tachycardia. *Reentrant ventricular tachycardia is dependent on an area of myofibers, embedded in scar tissue, which exhibit slow conduction. Late potentials recorded by signal-averaged electrocardiography appear to correspond to these zones of slow conduction and frequently are present in patients with VT. We hypothesized that elimination of inducible VT by catheter-mediated ablation of critical areas of slow conduction would alter late potentials. Four patients underwent catheter ablation in which radiofrequency current was delivered to zones of slow conduction exhibiting isolated mid-diastolic potentials that could not be dissociated from the tachycardia. The four patients had developed VT (cycle length 382 ± 50 msec; mean ± SEM) 13–180 months after inferior myocardial infarction. Late potentials were present in each patient before catheter ablation was attempted. Although VT was not inducible in any patient immediately after ablation, late potentials were still present in all four patients and there was no significant difference in the QRS duration (136.5 ± 4.0 msec postablation; 135.7 ± 4.5 msec preablation), root mean square voltage in the terminal 40 msec of the QRS (10.0 ± 1.0 μV postablation; 5.9 ± 0.4 μV preablation), or in the duration of the low amplitude signal (69.2 ± 2.0 msec postablation; 62.7 ± 3.4 msec preablation). At follow-up electrophysiology study performed 14 ± 7 days after ablation, one of the four patients had inducible VT. In conclusion, late potentials persist even after successful radiofrequency catheter ablation and do not appear to be useful for predicting results of follow-up electrophysiology study. (PACE, Vol. 17, February 1994)*

ventricular tachycardia, late potentials, signal-averaged electrocardiography, radiofrequency current, catheter ablation

Introduction

The usual mechanism of sustained monomorphic ventricular tachycardia in patients with healed myocardial infarction is reentry, which appears to be dependent on a zone of myofibers, interdigitated by scar tissue and exhibiting the slow conduction required to sustain reentry.[1,2] Direct endocardial recordings during ventricular tachycardia show continuous low amplitude multiphasic signals that bridge successive ventricular complexes.[3] At these same sites, endocardial electrograms recorded in sinus rhythm are fractionated and continue beyond the entire normal ventricular depolarization.

Using high resolution recording equipment and advanced signal averaging techniques, the resulting low amplitude signals (LAS), or late potentials can be detected from the body surface.[4] As the finding of late potentials appears to reliably identify patients with a history of ventricular tachycardia, it is presumed that late potentials re-

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sult from late activation of myofibers within the region producing ventricular tachycardia.[5,6] However, among asymptomatic patients recovering from acute myocardial infarction, the presence of late potentials has a relatively low specificity and positive predictive accuracy for inducible and clinical ventricular tachycardia.[7,8] These findings suggest that late potentials could arise from conduction through damaged ventricular myocardium, which although exhibiting slow conduction, is incapable of sustaining reentrant ventricular tachycardia.

We have previously demonstrated that mid-diastolic potentials recorded from the endocardium during ventricular tachycardia may represent activation of myocardium critically required for the maintenance of reentry.[9] Thus, initiation of ventricular tachycardia is always preceded by the appearance of the isolated mid-diastolic potential, ventricular extrastimuli that reset the tachycardia always advance the isolated potential before advancing the subsequent QRS complex, and termination of the tachycardia is always preceded by loss of the isolated potential. The finding that direct current shocks delivered to these sites frequently eliminates ventricular tachycardia lends additional support for the critical role of these regions.

We hypothesized that elimination of inducible ventricular tachycardia by catheter-mediated ablation of critical areas of slow conduction would alter late potentials. As radiofrequency current requires close proximity of the ablation electrode to the target site, absence of late potentials after successful ablation of ventricular tachycardia would reasonably be assumed to suggest that late potentials correspond only to critical regions of slow conduction zone. Conversely, their persistence after successful ablation would suggest that late potentials may arise, on occasion, from nonarrhythmogenic regions.

Methods

Patients

Four patients with coronary artery disease and prior myocardial infarction underwent endocardial mapping and catheter ablation for recurrent, sustained monomorphic ventricular tachycardia (Table I). The four patients had developed sustained ventricular tachycardia 13–180 months (mean 97 ± 41 months; mean ± standard error of the mean [SEM]) after myocardial infarction. Their age ranged from 55–69 years (mean 58 ± 2 years). In all four patients the clinical ventricular tachycardia had a single QRS morphology with a mean cycle length of 382 ± 50 msec (range 280–520 msec). Each patient had failed between 3–6 antiarrhythmic drug trials before their attempted ablation. Antiarrhythmic drugs were discontinued at least five elimination half-lives before attempted ablation. None of the patients had bundle branch block on a standard 12-lead ECG.

Endocardial Mapping

After providing written informed consent, patients underwent electrophysiology study in the

Table I.

Clinical Characteristics and Electrocardiographic Features of Sustained Ventricular Tachycardia

Patient	Age/Gender	EF (%)	MI-VT (mths)	VT		Unsuccessful Drug Trials
				Morphology	CL (msec)	
1	61/M	33	156	RBBB, LAD	520	3
2	56/F	36	180	RBBB, LAD	360	3
3	69/M	52	13	RBBB, RAD	280	4
4	55/M	29	42	RBBB, LAD	350	6
Mean ± SEM	58 ± 2	37 ± 5	97 ± 41		382 ± 50	4 ± 0.7

LAD = left axis deviation; MI-VT = interval between myocardial infarct and onset of ventricular tachycardia; RAD = right axis deviation; RBBB = right bundle branch block configuration.

fasting state and under heavy sedation with fentanyl (50–150 μg/hour IV) and midazolam (2–4 μg/hour IV). Programmed ventricular stimulation induced ventricular tachycardia with a single QRS morphology in three patients (1, 2, and 4), while patient 3 had inducible ventricular tachycardia that had two distinct QRS morphologies and cycle lengths. Mapping and ablation was attempted for all five tachycardia morphologies (cycle length 270–520 msec). Right ventricular endocardial mapping was performed during these tachycardias with a 6-French catheter with four electrodes and interelectrode spacing of 2 mm, center-to-center (Mansfield-Webster, Watertown, MA, USA). For left ventricular mapping and catheter ablation, a 7-French quadrapolar catheter with large tip-electrode (4-mm long; 27 mm^2 surface area; Mansfield-Webster Catheters) was inserted percutaneously into the right femoral artery and advanced into the left ventricle. The catheter was deflectable and controlled by a device at the base of the catheter. During left ventricular mapping and ablation, the patient was heparinized with 10,000 units IV, followed by a further 1,000 U/hour for the duration of the procedure.

The mapping catheter was manipulated toward areas exhibiting progressively earlier ventricular activation during ventricular tachycardia. Mid-diastolic activity was sought near areas of early ventricular activation and were considered to represent activation of myocardium, which were critically required for maintenance of the tachycardia if they could not be dissociated from the subsequent tachycardia complex.[9] In addition, evidence of slow conduction within or near the tachycardia circuit was confirmed in some patients by pacing at sites recording mid-diastolic potentials and producing identical tachycardia QRS morphology and long pacing latency.[10]

Catheter Ablation Procedure

Radiofrequency current (continuous wave, 550–750 kHz) was generated using a custom designed electrosurgical device (Liz 88, American Cardiac Ablation Corp., Foxboro, MA, USA). The unit was coupled to a device that provided real-time monitoring of root mean square (RMS) voltage, current and impedance (Boston Scientific, Watertown, MA, USA). Radiofrequency current was delivered at 45–60 V (usually 55 V) between the large-tip catheter electrode and a standard adhesive electrosurgical dispersive pad applied to the chest wall. The application of energy was terminated immediately if there was an increase in impedance or displacement of the catheter electrode.

Postablation Management

Programmed ventricular stimulation was repeated 30–60 minutes after the final application of radiofrequency current and again 7–21 days later (mean 14 ± 4 days). The stimulation protocol included single, double, and triple ventricular extrastimuli during two pacing cycle lengths (600 and 400 msec) from two ventricular sites (right ventricular apex and right ventricular outflow tract). Heparin administration was ceased at the completion of the ablation procedure. Plasma concentrations of creatine kinase (CK) and CK-MB isoenzyme were measured every 8 hours for 24 hours after ablation.

Signal-Averaged Electrocardiography

Signal-averaged electrocardiography (ECG) was performed with a Corazonix Predictor (Corazonix Inc., Oklahoma City, OK, USA). The electrocardiogram was recorded during sinus rhythm in all patients using standard bipolar, uncorrected X, Y, and Z leads, a mean of 7.5 ± 2.2 days before ablation was attempted. Signals from 200–300 beats/min were amplified, digitized, averaged, and then filtered with a bidirectional filter with high bandpass frequency filter of 25 Hz. The filtered leads were combined into a vector magnitude, $\sqrt{x^2 + y^2 + z^2}$, and QRS duration, the duration of the LAS < 40 μV and the RMS voltage of the signals in the last 40 msec of the filtered QRS were calculated. Recordings with a noise level > 0.3 microvolts were rejected. The signal-averaged ECG was considered to he abnormal if the filtered QRS duration was > 114 msec, and either a LAS was present (RMS-40 < 20 μV) or this potential (late potential) had a duration > 38 msec. Patients with bundle branch block on the standard 12-lead surface electrocardiogram were excluded from study.

Left Ventricular Function

Left ventricular ejection fraction was obtained in the 45° left anterior oblique projection before and after catheter ablation, by radionuclide angiography at rest during sinus rhythm. Left ventricular aneurysm was defined as the presence of regional paradoxical systolic wall motion.

Statistical Analysis

All variables are expressed as mean ± SEM. The significance of differences between mean values of continuous variables were assessed using paired two-tailed Student's *t*-test. A probability value of < 0.05 was considered significant.

Results

Results of Radiofrequency Catheter Ablation

The radiofrequency variables required to eliminate inducible ventricular tachycardia in all four patients are shown in Table II. A mean of 9.7 ± 0.8 (range 8–12) applications of radiofrequency were required at a mean power of 30.5 ± 4 watts delivered for a mean of 40 ± 9.3 seconds. An example of the ablation procedure (patient 1) is shown in Figure 1.

Isolated mid-diastolic potentials were recorded at the site of successful ablation in all four patients (Table II). At these sites, the earliest intracardiac ventricular activation (measured in relation to the QRS onset recorded on standard surface electrocardiographic leads I, II, or V_1) ranged between −30 and −70 msec (mean −47 ± 10 msec). In patient 3, two ventricular tachycardia morphologies were inducible before ablation was attempted (1: right bundle branch block configuration, right axis deviation, cycle length 280 msec; and 2: right bundle branch block configuration, left axis deviation, cycle length 270 msec). Delivery of radiofrequency current during a sustained episode of the second ventricular tachycardia terminated the tachycardia and prevented induction of both ventricular tachycardias 30 minutes following delivery of energy.

Ventricular tachycardia was not inducible in any of the four patients 30–60 minutes following the last application of radiofrequency current.

Table II.

Inducible Ventricular Tachycardia and Radiofrequency Variables Used for Ablation

Patient	VT Morphology/ Cycle Length (msec)	Location	Electrogram Pattern	Radiofrequency Power (W)/Duration (sec)	Acute Result	Follow-Up EPS Result
1	RBBB, LAD/520	Inferobasal	Mid-diastolic pot present Local V-QRS −60 msec	31/65	Success	−ve
2	RBBB, LAD/360	Inferobasal	Mid-diastolic pot present Local V-QRS −30 msec	41/25	Success	−ve
3	RBBB, RAD/280	Lateral basal	Mid-diastolic pot present Local V-QRS −30 msec	26/20	Success	+ve (CL = 265 msec)
	RBBB, LAD/270	Lateral basal	Mid-diastolic pot present Local V-QRS −40 msec	26/20*	Success*	
4	RBBB, LAD/350	Inferobasal	Mid-diastolic pot present Local V-QRS −70 msec	24/50	Success	−ve

* Ablation of the VT with RAD, also eliminated this VT as well.
EPS = electrophysiology study; LAD = left axis deviation; Local V-QRS = earliest local ventricular activation relative to onset of QRS during VT; mid-diastolic pot = mid-diastolic potential; RAD = right axis deviation; RBBB = right bundle branch block configuration; −ve = no inducible ventricular tachycardia; VT = ventricular tachycardia.

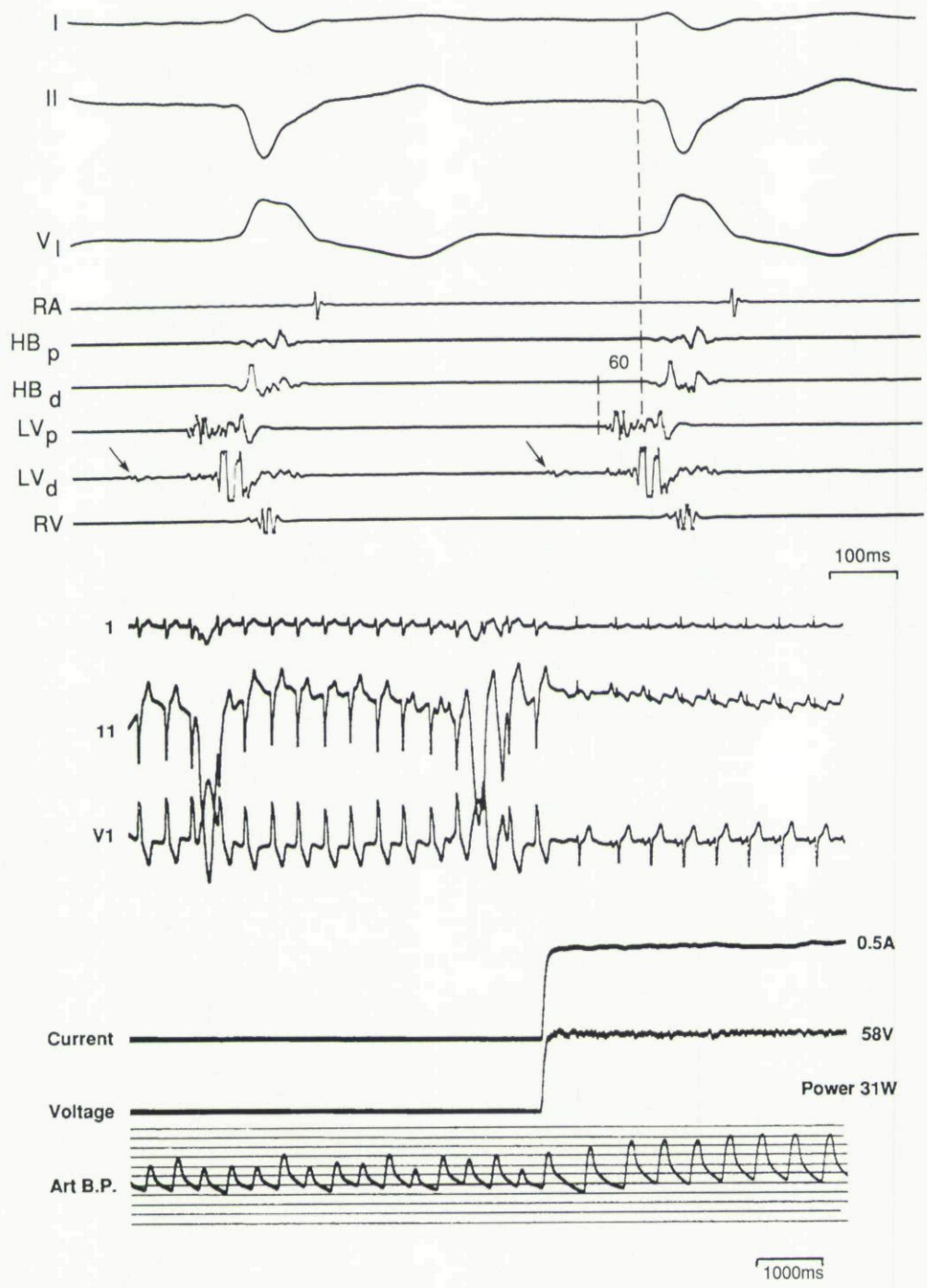

Figure 1. *Top panel: The recordings were obtained from the ablation electrode during ventricular tachycardia. From the top the tracings are surface ECG lead I, II, V_1, and electrograms recorded from the right atrium (RA), the proximal His-bundle region (HB_p) and distal His-bundle region (HB_d), the ablation electrode (LV), and the right ventricular apex (RV). Mid-diastolic potentials (arrows) are separated by an isoelectric interval from the ventricular potential associated with the QRS complex. Vertical dotted line marks the onset of the QRS complex. The ventricular potential, continuous with the main body of ventricular activation, begins 60 ms before the onset of the QRS complex. Bottom panel: Radiofrequency current was applied to the large tip electrode on the left ventricular catheter at 31 watts. Ventricular tachycardia terminated as radiofrequency current was applied. Art. B.P. = arterial blood pressure.*

After the ablation procedure, total CK was elevated in one patient (1,162 IU/L; normal < 289 IU/L); however, CK-MB isoenzyme was not elevated in any patient. Follow-up electrophysiology study was performed in all patients 14 ±7 days after ablation. Programmed ventricular stimulation induced sustained monomorphic ventricular tachycardia in one patient (3) using S_2–S_3 delivered from the right ventricular apex. The cycle length was slightly shorter than the clinical tachycardia (265 vs 280 msec, respectively) but the QRS morphology was identical.

Effect on the Signal-Averaged ECG

An example of the effects of radiofrequency catheter ablation on the signal-averaged ECG of one patient (1) and composite data for all patients are shown in Figures 2 and 3. Before ablation, all four patients had late potentials recorded with the mean QRS duration being 135.7 ± 4.5 msec, the RMS-40 voltage was 5.9 ± 0.4 μV, and the LAS duration was 62.7 ± 3.4 msec. At a mean of 19.5 ± 4.6 days after ablation, the mean QRS duration was 136.5 ± 4.0 msec, the RMS-40 voltage was 10.0 ± 1.0 μV, and the LAS duration was 69.2 ± 2.0 msec. These values were not statistically significantly different from those obtained before ablation.

Effect on Left Ventricular Function

The mean left ventricular ejection fraction in the patients before ablation was 37.5 ± 5% (range 29%–52%) and three patients had left ventricular aneurysms present. The patients' left ventricular ejection fraction, measured 1–3 months after the ablation procedure, was 46.5 ± 4% (range 36%–55%). Ejection fraction increased in all patients by 1%–22%, but the increase did not reach statistical significance.

Clinical Outcome

During a mean clinical follow-up of 16.7 ± 6.5 months, no patient has had recurrence of ventricu-

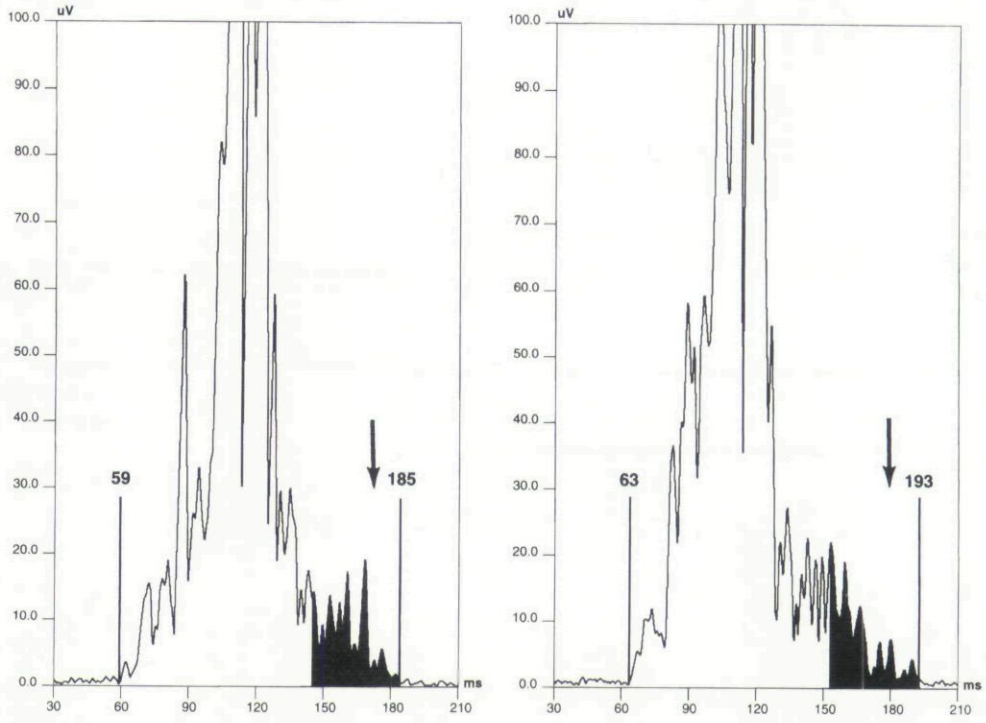

Figure 2. *Example of signal-averaged electrocardiography in patient 1. The vector magnitude tracings of the filtered QRS complex are shown before ablation (left panel) and after ablation (right panel). The terminal 40 ms of the filtered QRS is shaded black. A late potential (arrow) persists after successful ablation.*

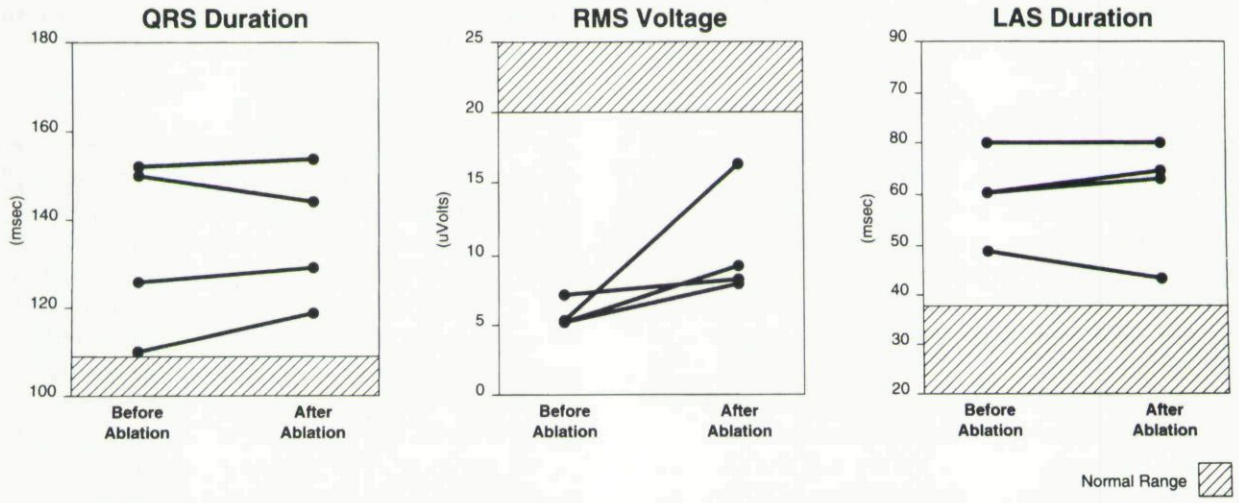

Figure 3. *Effect of radiofrequency catheter ablation on QRS duration (left panel), root mean square voltage of the terminal 40 msec of the QRS (RMS) voltage (middle panel), and low amplitude signal (LAS) duration (right panel).*

lar tachycardia and none has received antiarrhythmic drug therapy. The one patient who had inducible ventricular tachycardia at his postablation electrophysiology study received an implantable cardioverter defibrillator but has had no discharges from the device up to 18 months after implantation.

Discussion

Late potentials recorded by signal-averaged ECG in patients with coronary artery disease and sustained ventricular tachycardia persisted after successful radiofrequency catheter ablation. There was no apparent change in the pattern or duration of late potentials that could predict the efficacy of catheter ablation. These results would suggest that radiofrequency catheter ablation that targets middiastolic potentials may abolish ventricular tachycardia by disconnecting the critical segment of slow conduction from exit points. The persistence of late potentials after successful ablation of this critical slow zone implies that late potentials may result from conduction through other regions of slow conduction that do not support ventricular tachycardia. This conclusion is supported by recordings made in the experimental canine infarction model showing that regions that produce late potentials during sinus rhythm may not always correspond with sites recording slow reentrant activation during ventricular tachycardia.[11] The lack of correlation in this model was explained by the fact that the critical sites during reentry were more dependent on the location and extent of arcs of functional conduction block and not solely upon sites exhibiting delayed activation.

Similarly, Hood et al.[12] have recently shown in patients with sustained ventricular tachycardia, that up to 95% of the abnormal signals produced within the myocardium critical for ventricular tachycardia occur before the terminal 40 msec of the QRS. Thus conventional signal averaging techniques may not be suitable to detect changes in activation of tissue targeted by ablation. The use of more sophisticated signal-averaged ECG techniques before and after catheter ablation of ventricular tachycardia, including two-dimensional frequency analysis and spectral turbulance analysis, might permit examination of the entire QRS complex.[13] In contrast with the failure of late potential to be eliminated by radiofrequency catheter ablation, Breithardt et al. [14] have shown that late potentials are frequently absent after successful surgical ablation in which all regions of slow conduction are removed by resection of scar tissue. Similarly, tissue necrosis resulting from transcoronary infusion of ethanol may modify late potentials in some patients with ventricular tachycardia.[15]

It might be argued that the ventricular lesions produced by repeated applications of radiofrequency current might provoke new regions of slow conduction resulting in delayed activation and late potentials. However, the lesions produced by radiofrequency current are small (approximately 0.5 cm in diameter) and have clear demarcated edges.[16] By contrast, endocardial sites recording low-amplitude, delayed activation signals are typically found in the border zone of infarcted tissue which morphologically exhibits ragged and mottled edges.[17]

Supportive evidence from this study that persistent late potentials after catheter ablation was not due to damage produced during the ablation procedure was that cardiac enzymes (CK-MB isoenzyme) were not elevated in any patient and that left ventricular ejection fraction was improved after catheter ablation. The latter finding probably reflects reversal of deleterious effects upon withdrawal of antiarrhythmic drug therapy, and the untoward effects of recurrent ventricular tachycardia upon left ventricular function. Reversal of severe left ventricular dysfunction after surgical ablation has been previously reported.[18]

The results of this study have clinical implications for the interpretation of detecting late potentials in patients without documented ventricular tachycardia who are recovering from acute myocardial infarction or have had unexplained syncope. The finding that late potentials persisted after successful elimination of ventricular tachycardia is consistent with the finding that late potentials have a relatively low specificity and poor positive predictive value for induction of ventricular tachycardia. Thus, while late potentials may indicate areas of damaged myocardium exhibiting delayed conduction, only a subset of these patients also have central regions of block, which is required for reentry to occur.[19]

The ability to predict the efficacy of catheter ablation performed for ventricular tachycardia, perhaps obviating the need for a follow-up electrophysiology study using a noninvasive test, has obvious clinical appeal. Unfortunately, the findings of the present study show that persistence of late potentials after catheter ablation do not predict the results of follow-up electrophysiology study, and, therefore, a follow-up study is recommended to assess the efficacy of ablation. This conclusion is supported by the findings of Borggrefe et al.[20] who found signal-averaged ECG was not helpful in predicting successful outcome of direct current catheter ablation of ventricular tachycardia.

This study has obvious limitations including the small number of observations made, which mean that our failure to find any significant change in signal-averaged ECG recordings after ablation certainly does not exclude the possibility that a larger sample size might have detected one. In addition, because of the need to perform endocardial mapping of sustained ventricular tachycardia in a relatively expeditious manner, complete validation of mid-diastolic potentials and pacing to entrain the tachycardia was not always done in each patient.

In conclusion, the results of this study demonstrate that late potentials recorded by signal-averaged ECG in patients with ischemic heart disease and ventricular tachycardia persists, even after successful catheter ablation to prevent recurrent tachycardia.

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