Linear Ablation Lesions for Control of Unmappable Ventricular Tachycardia in Patients With Ischemic and Nonischemic Cardiomyopathy

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Background—Conventional activation mapping is difficult without inducible, stable ventricular tachycardia (VT).

Methods and Results—We evaluated 16 patients with drug refractory, unimorphic, unmappable VT. All patients had implantable defibrillators and had experienced 6 to 55 VT episodes during the month before treatment. Patients underwent bipolar catheter mapping during baseline rhythm. The amount of endocardium with an abnormal electrogram amplitude was estimated using fluoroscopy in 3 patients and a magnetic mapping system (CARTO) in 13 patients. For the magnetic mapping, normal endocardium was defined by an amplitude \( >1.5 \text{ mV} \); this measurement was based on sinus rhythm maps in 6 patients who did not have structural heart disease. Radiofrequency point lesions extended linearly from the “dense scar,” which had a voltage amplitude \( <0.5 \text{ mV} \), to anatomic boundaries or normal endocardium. To limit radiofrequency applications, 12-lead ECG during VT and pacemapping guided placement of linear lesions. No new antiarrhythmic drug therapy was added. The amount of endocardium demonstrating an abnormal electrogram amplitude ranged from 25 to 127 cm\(^2\). A total of 8 to 87 radiofrequency lesions (mean, 55) produced a median of 4 linear lesions that had an average length of 3.9 cm (range, 1.4 to 9.4 cm). Twelve patients (75%) have been free of VT during 3 to 36 months of follow-up (median, 8 months); 4 patients had VT episodes at 1, 3, 9, and 13 months, respectively. Only one of these patients had frequent VT.

Conclusions—Radiofrequency linear endocardial lesions extending from the dense scar to the normal myocardium or anatomic boundary seem effective in controlling unmappable VT. (Circulation. 2000;101:1288-1296.)

Key Words: tachycardia • ablation • defibrillators, implantable

The presence of multiple morphologies, hemodynamic intolerance, and noninducible ventricular tachycardia (VT) have limited the widespread applicability of catheter ablative therapy.\(^1-3\) An anatomically based approach deployed in sinus rhythm might eliminate the requirement for mapping all ventricular arrhythmias and extend the applicability of ablative therapy.

On the basis of prior experience with sinus rhythm electrogram mapping and surgical ablative therapy, we hypothesized that in patients with unmappable, unimorphic VT, (1) the abnormal endocardium can be defined using detailed sinus rhythm voltage mapping, and (2) linear ablation lesions that repeatedly and/or selectively interrupt the border zone of abnormal endocardium could control VT.

Methods

Patient Population
This study included 12 men and 4 women who had recurrent VT that induced shocks from their implantable defibrillator. Nine patients had coronary disease and a prior infarction, and 7 patients had nonischemic cardiomyopathy. Four of the 7 patients had primarily right ventricular (RV) involvement, with marked RV dilatation and marginally depressed left ventricular (LV) systolic function (Table 1). Two patients had idiopathic cardiomyopathy, with dilatation of both ventricles. The last patient with nonischemic cardiomyopathy had biventricular involvement in the setting of rheumatic valvular disease. Despite drug therapy, all patients had frequent VT, which was documented by electrograms stored in the implantable defibrillator (Table 1). Thirteen of the 16 patients had received long-term (>2 months) therapy with amiodarone, and 6 had prior ablation attempts. Most patients had evidence of multiple VT morphologies, as based on 12-lead ECG or stored electrograms. All documented, spontaneous VT was unimorphic. The 16 patients came from a pool of 48 patients who were referred to our hospital for catheter ablation.

The study patients were evaluated after informed consent was obtained. All procedures were done following the institutional
guidelines of the Allegheny University of the Health Sciences. A total of 15 patients were also treated with antiarrhythmic agents during the study (Table 1). Antiarrhythmic agents were not withdrawn because (1) 14 patients were being treated with amiodarone and (2) all patients were experiencing recurrent VT. All patients underwent programmed stimulation through triple extrastimuli at 2 RV sites to document the mode of induction, the cycle length, and the configuration of the induced VT (Table 1). At the time of evaluation, VT was not mappable because of hemodynamic collapse with VT, changes in QRS morphology with attempted mapping, or the inability to induce sustained VT (Table 1).

Table 1. Patient Characteristics

<table>
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<th>Patient No.</th>
<th>Age, y</th>
<th>Sex</th>
<th>VT Substrate</th>
<th>LVEF, %</th>
<th>Failed AAD</th>
<th>No. of VT Episodes in Prior Month</th>
<th>No. of VT Morphologies at EPS</th>
<th>No. of Inducible VT Episodes</th>
<th>VTCL, range</th>
<th>Reason VT not Mapped</th>
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</table>

AAD indicates antiarrhythmic drugs; Am, amiodarone; BB, β-blockers; Br, bretylium; CAD, coronary artery disease; CM, change in morphology with mapping; F, female (under Sex); F, flecainide (under AAD categories); L, lidocaine; LVEF, left ventricular ejection fraction; M, male (under Sex); M, mexiletine (under AAD categories); Ni, VT not inducible; NICM, nonischemic cardiomyopathy; NT, initiated VT poorly tolerated; Pr, procainamide; P, propafenone; Q, quinidine; S, sotalol; D, disopyramide; VTCL, cycle length for induced VT; EPS, electrophysiological study; and IV, intravenous.

Figure 1. Voltage maps of RV in anterior (AP) and posterior (PA) views. Color range for bipolar electrogram maps is identical for all subsequent figures. Purple represents normal endocardium (amplitude $<1.5$ mV); red, dense scar (amplitude $>0.5$ mV); and range between purple and red, border zone (signal amplitudes between 0.5 and 1.5 mV). Top, RV map from patient without heart disease is represented almost entirely by color purple. Bottom, RV map from patient with VT and RV cardiomyopathy with abnormal endocardium over superior RV free wall and septum. Linear ablation lesions, identified by contiguous dark circles, extend from dense scar to normal endocardium. APEX indicates location of RV apex.

Figure 2. Voltage maps of LV in right and left anterior oblique (RAO and LAO) views. Color range is as described in Figure 1. Top, LV map from patient without heart disease is represented by color purple. Bottom, LV map from patient with VT and coronary disease shows apical dense scar, as indicated by color red, and border zone that extends over septum and anterior LV. Linear ablation lesions, identified by contiguous dark circles, extend across border zone.
Sinus Rhythm Mapping

Detailed endocardial mapping was performed during supraventricular rhythm (14 patients) or, if the patients were pacemaker-dependent, paced rhythm (2 patients). A total of 72 to 430 sites were recorded per chamber. Only the LV was mapped in patients with coronary disease. In patients with nonischemic cardiomyopathy, both ventricles were mapped. Access to the LV was via a retrograde aortic approach in 14 patients and a transseptal approach in 2 patients. During all LV mapping and ablation, heparin was administered to achieve and maintain an activated coagulation time >250 s.

Reference Values for Sinus Rhythm Bipolar Electrogram Mapping

In the first 3 patients, all mapping and ablation was performed using a 7-F extended-curve thermistor catheter (SteeroCath-T, Electrophysiology Technologies, Inc) that had a 4-mm tip electrode. The catheter provided temperature and impedance monitoring during energy delivery, as well as bidirectional steerability. All recordings were bipolar, with an interelectrode distance of 2 mm. Signals were filtered at 30 to 500 Hz and were displayed at 100 to 200 mm/s on a physiological recording system (Prucka, Inc). Electrogram amplitudes were measured with electronic calipers. Sites with an electrogram amplitude $<3.0$ V were considered abnormal.

In the next 13 patients, we used the CARTO ( Biosense, Inc) magnetic mapping system with the Navistar catheter. Navistar catheters were 7 or 8 Fr, unidirectionally deflectable, and provided impedance but not temperature monitoring. The Navistar bipole consists of a 4-mm-tip electrode and a 2 mm-ring electrode separated by 1 mm of spacing. Electrograms were filtered at 10 to 400 Hz and displayed at 100 mm/s; peak-to-peak amplitude was measured automatically.

Reference values for distinguishing normal and abnormal electrograms with this system were established by mapping the RV (4 patients) and/or LV (4 patients) in 6 patients who did not have structural heart disease. A total of 71 to 168 sites were recorded per normal ventricle. None of these subjects was taking cardioactive drugs. Five of these subjects were men, and their mean age was 37±12 years.

Reference Values of Magnetic Mapping System

The mean bipolar electrogram amplitude recorded from the normal RV was $3.7\pm1.7$ mV (range, 0.4 to 11.5 mV). Of note, 95% of all bipolar electrogram signals recorded from the RV were $>1.44$ mV. The mean bipolar electrogram amplitude recorded from the LV was $4.8\pm3.1$ mV (range, 0.6 to 20.5 mV). Of note, 95% of all LV electrograms were $>1.55$ mV. Using these data, we defined normal endocardium using the CARTO-Biosense system as that demonstrating a bipolar electrogram of $>1.5$ mV. On the basis of our previous experience with catheter and intraoperative mapping, we then arbitrarily designated a value of $<0.5$ mV as consistent with “densely scarred” endocardium.
Figure 4. Electrocardiograms of VT and pacemap identifying appropriate regions for linear ablation in patient with coronary disease. A, Left bundle-branch block VT mimicked during pacing from LV septum (pacing site 2-3).9,10 Inset shows location of linear lesion that crossed site of pacemap. Two other left bundle VT morphologies were present; thus, additional septal linear lesions existed. B, Two additional right bundle VT morphologies mimicked by pacemapping. Mesh figure of LV in left anterior oblique (LAD) projection shows relationship of pacemap sites to nonseptal linear lesions. C, Voltage maps projected to display septal, inferior, and anterolateral LV to show all linear lesions crossing border zones, with mesh figure as in B. Linear nature of multiple point lesions is emphasized by solid lines.
Figure 5. Linear lesions crossing all aspects of border zone of inferior infarction in patient with multiple unmappable VTs. Voltage maps projected display septal, inferior, and lateral LV and show extent of abnormal recordings and linear lesions. Voltage map color range is as defined in Figure 1. Seven linear ablation lines interrupt border zone. Twelve-lead ECGs were not available for spontaneous VTs warranting a more extensive, non-guided approach.

Voltage Map Color Display and Technique for Estimating Extent of Abnormal Endocardium

The magnetic mapping system includes a magnetic sensor in the catheter tip that can be localized in 3D space using the ultralow magnetic field generators placed under the fluoroscopic table. The electrogram amplitude recorded from the catheter at different endocardial locations can be shown on a computer display as a voltage map. The color display for illustrating abnormal myocardium was set with a color range of 0.5 to 1.5 mV to highlight the border zone (Figures 1 through 6). The CARTO system can also calculate the anatomic distance between any 2 designated points.5,6 Thus, by assuming a rectangular or trapezoidal shape of any abnormal segment, the extent of the endocardium demonstrating abnormal (<1.5 mV) and densely scarred (<0.5 mV) voltage amplitudes could be estimated (Tables 2 and 3).

Ablation Technique and Location of Linear Lesions

Linear lesions were placed using 3 guiding principles. (1) Lesions would extend across the borders of the endocardium that demonstrated abnormal bipolar electrogram voltage. (2) Lesions would typically extend from the areas demonstrating the lowest amplitude signals (<0.5 mV) to areas demonstrating a distinctly normal signal (>3 mV for the first 3 patients and >1.5 to 2.0 mV for the last 13 patients) or to a valve continuity. (3) Lesions would cross through the border zones at sites where mapping approximated the QRS morphology of VT. Twelve-lead ECG recordings of spontaneous and induced VT were analyzed to regionalize the site of origin of the VT using standard criteria.9,10 The mapping catheter was then placed along the appropriate border of the scar, and pacemapping was performed to create a 12-lead ECG that approximated the VT (Figures 3 and 4). This modification to a purely anatomic ablation approach was essential because (1) the extent of electrogram abnormalities was large (Tables 2 and 3) and (2) the size and 3D character of the VT circuit was not established. In 5 of the 16 patients, the electrograms stored in the defibrillator did not match the electrograms of induced VT, and 12-lead ECGs of spontaneous VT were unavailable. In these patients, linear lesions were created at 3- to 5-cm intervals along all aspects of the border zone. (Figure 5).

Sequential point lesions created the linear lesions. For each point lesion, radiofrequency energy was applied for 90 to 120 s using a power output to achieve a targeted temperature of 60°C (StereoCath-T catheter) or a targeted impedance drop of 6 to 10 ohms (Navistar catheter). Power output started at 5 W and was titrated over 30 s to a maximum output of 50 W or until the targeted temperature or impedance was achieved. To avoid an impedance rise, output was decreased manually whenever the impedance drop exceeded 10 ohms or temperature exceeded 60°C to 65°C. For the first 3 patients, the linear lesion location and size were estimated using fluoroscopy. This process was facilitated using 3D magnetic mapping (CARTO-Biosense) in the last 13 patients.9,10 Radiofrequency energy applications were “tagged” for display, and the length of the linear lesion was documented (Tables 2 and 3).

In 8 of the 16 patients, programmed stimulation was repeated after making linear lesions on one end of the endocardium that demonstrated abnormal electrograms to assess the inducibility of specific, targeted tachycardia morphologies. In all but one of the 16 patients, stimulation that resulted in VT in the baseline state (and that included triple extrastimuli) was performed after creating all linear lesions. In the one patient in whom this was not done, prolonged periods of hemodynamic instability followed the prior episodes of VT.

Follow-Up

VT recurrence was identified via device interrogation and a report of clinical symptoms. No new antiarrhythmic drug therapy was added (Table 4). In addition, we assessed the effects of the linear lesions on the LV by using gated nuclear blood pool scanning in 6 patients with ischemic cardiomyopathy. A policy regarding anticoagulation after the procedure was established after the first 2 left-sided ablation procedures. Coumadin was prescribed for 3 months to achieve an international normalized ratio >2.0.

Statistical Analysis

Results are presented as mean ± SD unless otherwise indicated. Comparisons of total procedure and fluoroscopy time between patients with coronary disease and those with nonischemic cardiomyopathy were made using an unpaired Student’s t-test. A compar-
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The extent of LV endocardium subtended by contiguous bipolar recordings demonstrating abnormal electrograms in patients with coronary artery disease was 65 ± 24 cm² and ranged from 51 to 110 cm² (Table 2 and Figures 2, 5, and 6). The dense scar (< 0.5 mV) ranged from 5 to 44 cm² (Table 2).

Endocardial Voltage Maps in Patients With Dilated Cardiomyopathy
In all patients with dilated cardiomyopathy, abnormal endocardial electrograms were recorded from the RV septum and free wall (Figures 1 and 6). In contrast, abnormal electrograms were recorded from the LV in only 3 patients. Only one of these patients had extensive LV septal and free wall abnormalities (Table 3). The extent of the RV endocardium subtended by contiguous bipolar recordings demonstrating abnormal electrograms was 60 ± 36 cm² and ranged from 25 to 127 cm² (Table 3). The abnormal endocardium in the single patient with extensive LV involvement measured 45 cm². Dense scar (< 0.5 mV) was also identifiable in all patients with nonischemic cardiomyopathy. The estimated size of the dense scar ranged from 8 to 50 cm² (Table 3).

Linear Radiofrequency Ablation Lesions
A total of 8 to 87 (median, 55) radiofrequency lesions were applied per patient. Point lesions created 1 to 9 linear lesions (median, 4). The average length of the linear lesion was 3.9 cm, and the range was 1.4 to 9.4 cm (Tables 2 and 3).

Programmed Stimulation After Ablation
In 6 of the 8 patients in whom programmed stimulation was repeated after making lesions on one end of the scar, only VT morphologies consistent with an origin from the opposite end of the scar were induced (Table 4). In response to stimulation at the completion of all ablation lines, 7 of the 15 patients had no inducible VT. Of the 8 patients with persistent inducible VT, 5 had rapid VTs inducible with double (2 patients) or triple (3 patients) extrastimuli. These VTs did not match the cycle length or morphology of the spontaneous arrhythmias. In the remaining 3 patients, a slower tachycardia was still inducible. In 1 of these 3 patients, the induced VT did not match spontaneous VT. In the second patient, the VT matched a clinical VT morphology but was slower in rate. In

### Table 2. Mapping and Ablation Characteristics in Patients With Coronary Artery Disease

<table>
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<tr>
<th>Patient No.</th>
<th>No. of RV Sites</th>
<th>No. of Endo Sites</th>
<th>Size of LV, cm²</th>
<th>Size of RV, cm²</th>
<th>No. of RF</th>
<th>Length of Linear Lesions, cm</th>
<th>Procedure, hr</th>
<th>Fluoroscopy, min</th>
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Egs indicates electrograms; Endo, endocardial; NA, not applicable because total number of sites recorded using fluoroscopy alone did not document estimated number >100/patient; ND, not done because accuracy of estimate of endocardial involvement using fluoroscopy was thought to be limited; and RF, radiofrequency applications.

### Table 3. Mapping and Ablation Characteristics in Patients With Nonischemic Cardiomyopathy

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<th>Size of RV, cm²</th>
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RF indicates radiofrequency applications; Egs, electrograms.
the last patient, no ECG information was available for the spontaneous VT.

**Duration of Procedure and Fluoroscopy Exposure and Complications**

The total procedure time ranged from 6.0 to 13.5 hours (mean, 8.8±1.9 hours). The total fluoroscopy time ranged from 60 to 196 minutes (mean, 121±38 minutes). The total procedure time (10.8±2.1 versus 8.1±2.1 hours; P<0.05) and the total fluoroscopy time (156±32 versus 90±27 minutes; P<0.01) were greater for patients with VT who had nonischemic rather than ischemic cardiomyopathy.

None of the 6 patients with LV ejection fraction measurements before (mean, 24±6%) and after (mean, 23±9%) ablation demonstrated a deterioration (>5%). One patient experienced a cerebral vascular accident with right-sided hemiparesis at the end of the procedure. Residual arm weakness persisted at 6 months. In this patient, an impedance rise occurred during radiofrequency energy application. When the ablation catheter was removed through a transseptal sheath, residual coagulum was noted.

**Follow-Up**

All patients have been followed for ≥3 months (range, 3 to 36 months; median, 8 months; Table 4). Three patients died at 3, 4, and 8 months after the ablation procedure from refractory heart failure (baseline LV ejection fraction was 18%, with associated severe mitral regurgitation), pneumonia, and complications of abdominal surgery. Fifteen patients were free of VT during the initial follow-up month (Figure 7), and 12 patients (75%) have been free of any recurrence of VT during the entire duration of follow-up. Only 1 of the 4 patients with VT during follow-up had frequent recurrences. Recurrent VT in this patient has been slow and amenable to pacing therapy.

![Figure 7. Outcome after linear lesions for unmappable VT. All patients had frequent VT during month before ablation. A total of 12 patients remained arrhythmia-free during follow-up. Isolated VT occurred at 3, 9, and 13 months in 3 patients, with frequent VT in only one patient.](image)
Discussion
The current study describes a new approach for the ablation of unmappable, unimorphic VT associated with ischemic and nonischemic cardiomyopathy. The study documents that (1) the extent and voltage characteristics of the abnormal endocardium can be defined using sinus rhythm, bipolar electrogram, voltage mapping, and (2) catheter-based ablative therapy that creates linear lesions targeting the border zone can control recurrent VT.

Electroanatomic Substrate for VT
The magnetic mapping system (CARTO) provided a tool for creating a 3D bipolar electrogram voltage map. In patients with both ischemic and nonischemic cardiomyopathy, the extent of abnormal electrogram recordings was large, averaging >50 cm² (Tables 2 and 3). Cassidy and colleagues noted a comparable degree of endocardial electrogram abnormalities in patients with monomorphic VT in the setting of ischemic and nonischemic cardiomyopathy. Of note, sampling was typically limited to <20 sites in that study. By sampling from hundreds of sites, we could characterize the electroanatomic substrate in more detail. Almost uniformly, contiguous areas of very abnormal signals with an amplitude <0.5 mV were identified in patients with ischemic and nonischemic cardiomyopathy. This dense scar was typically surrounded by large “border zone” areas with signal amplitudes between 0.5 and 1.5 mV, which transitioned into normal myocardium (Figures 1 through 6).

Linear Lesion Deployment Based on Surgical Experience
Subendocardial resection guided by the presence of the endocardial scar is associated with a 70% to 80% arrhythmia cure rate. Such surgical therapy is performed when uniform, sustained VT cannot be initiated at the time of surgery. This extensive surgical experience served as the basis for creating linear lesions that connected the dense scar area to the normal endocardium; we used electrogram recordings during sinus rhythm as a guide. The magnetic electroanatomic mapping system assisted in creating contiguous lesions through the defined border zone by documenting the location of the catheter tip and its relationship to the endocardial anatomy.

Use of 12-Lead ECG During VT and Pacemapping to Guide Placement of Linear Lesions
Eliminating all of the border zone endocardium using current catheter-based ablative techniques would be impractical and possibly unsafe. The placement of linear lesions was guided by an interpretation of the 12-lead ECG during VT and pacemapping in the border zone. Linear lesions were created in regions that crossed the border zone and intersected the best pacemap site. We hypothesized that the best pacemap site approximated the exit site of the VT circuit and that the described endocardial linear lesion would likely interrupt a portion of a reentrant circuit (Figure 8). This described modification to a purely anatomic approach allowed us to target specific regions of the border zone in most patients.

Limitations
Because of the variability in the frequency of VT, it is difficult to ascertain whether a good clinical response is causally related to any ablation procedure. We validated the assumption of causality by (1) documenting the frequency of VT in the month before ablation, (2) using diagnostic information from the implantable defibrillator to document VT recurrence, (3) not adding new antiarrhythmic drug therapy, and (4) including only patients followed for ≥3 months. Because of these strict criteria, we are confident that arrhythmia control after ablation occurred in most patients.

Although linear lesions were created using repeated point radiofrequency applications, we do not wish to imply that we created conduction block. Our technique is descriptive, and the mechanism of efficacy is speculative. Each patient had unmappable tachycardia that was documented. We do not know whether a similar ablation technique would be effective in patients with polymorphic VT. In addition, given the average duration of the procedure and the duration of fluoroscopic exposure, the technique as described might not be practically applied in many institutions.

Finally, although we think that we have validated the efficacy of the described technique, we cannot be as certain about its safety. Indeed, we found no evidence of deterioration in LV function with repeat radionuclide scans. Nevertheless, a cerebral vascular accident occurred in one of our patients. The use of magnetic mapping catheters with temperature-monitoring capabilities may decrease the risk of thromboembolic phenomena.

References


Linear Ablation Lesions for Control of Unmappable Ventricular Tachycardia in Patients With Ischemic and Nonischemic Cardiomyopathy
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