Electrogram patterns predicting successful catheter ablation of ventricular tachycardia

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ABSTRACT Ventricular tachycardia in patients with remote myocardial infarction is thought to be due to reentry. To improve the efficacy of catheter ablation, we sought to identify electrograms identifying essential components of the reentrant circuit. In this study we compared the efficacy of shocks delivered at sites of early ventricular activation during tachycardia (presumably exit sites from the reentrant circuit) with that of shocks delivered at sites recording mid-diastolic potentials that were not continuous with the main ventricular potential recorded during the QRS complex, but that always remained associated with the tachycardia during initiation, termination, and resetting with extrastimuli (presumably activation of a segment of the slowly conducting region of the reentrant circuit). A total of 20 attempts was made to ablate 14 monomorphic ventricular tachycardias in 10 patients with remote myocardial infarction with use of one to five shocks of 50 to 370 J (200 J in 70%). All seven tachycardias in which isolated mid-diastolic potentials were targeted were successfully ablated, although one required a second attempt. Twelve attempts were made to ablate seven tachycardias by delivering shocks at sites of early activation during tachycardia when mid-diastolic potentials were not identified. Only three attempts (25%) were successful. Activation preceded the QRS complex by 60, 85, and 120 msec in the three successful attempts and by 20 to 110 msec (median 55 msec) in the nine unsuccessful attempts. For the total 20 attempts, there was no significant difference between successful and nonsuccessful ablation in the number of shocks or total energy delivered. We conclude that potentials critical to the maintenance of reentry can be identified and validated by programmed stimulation techniques, and that shocks delivered to this area have a high likelihood of success.

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REGULAR monomorphic ventricular tachycardia in patients with remote myocardial infarction is presumed to result from intraventricular reentry.¹ In catheter ablation, energy has been delivered to the site of earliest ventricular activation during tachycardia under the assumption that this site is close to regions necessary for the maintenance of reentry. Complete abolition of the tachycardia has been achieved in only 27% to 53% of patients.²–⁴ The frequent failure of catheter ablation suggests that sites of early activation are not critical for the tachycardia-generating mechanism, and may simply represent exit regions to the remaining ventricular myocardium.

In animal studies of postinfarction ventricular tachycardia, large composite electrodes have identified continuous electrical activity throughout the cardiac cycle, which is thought to represent conduction through the entire reentrant circuit as well as activation of adjacent myocardium.⁵–⁷ In human studies, with the use of closely spaced bipolar endocardial electrodes, continuous electrical activity can be recorded during ventricular tachycardia,⁸ but not consistently. The reentry pathway may be too large for its entire activation to be recorded at a single bipolar electrode site. Slight changes in catheter position in regions of slow conduction may record activation at very different times (figure 1). Regions critical to the maintenance of tachycardia in animal studies, reflected by the ability to terminate the tachycardia with a cryoprobe ("ice mapping"), are often activated in early or mid-diastole.⁹

In patients undergoing catheter ablation, we sought to identify isolated mid-diastolic potentials that could not be dissociated from the tachycardia. Assuming these potentials might represent a critical component of the reentrant circuit, we delivered direct-current shocks at these sites. Failing to identify the isolated potentials,
shocks were delivered at sites of early activation. This report examines and compares the success of ablation when shocks are delivered to sites with different patterns of activation.

Methods

Patient population. Ten patients with coronary artery disease and prior myocardial infarction underwent endocardial mapping and catheter ablation for recurrent, sustained ventricular tachycardia. Time of the last infarction ranged 2 months to 22 years (median 5 years) before the attempted ablation. Ages ranged from 54 to 74 years (median 65 years). In eight patients, ventricular tachycardia with a single morphology occurred clinically, while the other two patients had spontaneous occurrences of ventricular tachycardia with two distinct QRS morphologies and cycle lengths. In each patient, at least three antiarrhythmic agents had either been unsuccessful in preventing recurrence of tachycardia or had produced intolerable side effects. Seven patients had been treated with amiodarone. Amiodarone was discontinued in five patients 9 days to 6 weeks (median 4 weeks) before study because of pulmonary toxicity (three patients) or recurrence of ventricular tachycardia (two patients). Two patients were receiving amiodarone at the time of the ablation attempt.

Endocardial mapping. After providing informed consent, patients underwent electrophysiologic study in the fasting state. Sedation and analgesia were obtained with diazepam and/or fentanyl. Programmed ventricular stimulation induced ventricular tachycardia with one to six (median three) different QRS morphologies and cycle lengths in each patient. Mapping and ablation were attempted in a total of 14 tachycardias (cycle length 250 to 530 msec, median 365 msec), including the 12 clinical tachycardias. Left and right ventricular endocardial mapping was performed during these tachycardias with either No. 6F catheters with two to eight electrodes and interelectrode spacing of 2.0 to 6.5 mm, center to center (USCI or Webster Laboratories), or a No. 7F steerable quadripolar catheter with 6.0 mm interelectrode spacing, center to center (Mansfield Scientific, Navigator model). Catheters were manipulated toward areas exhibiting progressively earlier ventricular activation during the tachycardia. Mid-diastolic activity was sought near areas of early ventricular activation. Potentials were considered "isolated" if separated from the main body of ventricular potentials associated with the QRS complex by an isoelectric segment (figure 2, A). These potentials were considered to represent activation of myocardium critically required for the maintenance of the tachycardia only if all of the following criteria were met: (1) Initiation of the tachycardia was always preceded by the appearance of the isolated potential (figure 3). (2) Ventricular extrastimuli that reset the tachycardia always advanced the isolated potential before advancing the subsequent QRS complex (figure 4, A), while extrastimuli that failed to advance the timing of the isolated potential also failed to advance the tachycardia. (3) Loss of the isolated potential was always associated with the termination of the tachycardia (figure 4, B).

If isolated mid-diastolic potentials were not identified, then the site recording earliest ventricular activation was used for shock delivery (figure 2, B). The same validation criteria were imposed on these sites to confirm an association with the tachycardia.

Shock delivery. Patients were anesthetized with methohexital. One to five direct-current shocks (damped sinusoidal waveform, Hewlett-Packard Model 78670A defibrillator) of 50 to 370 J each were delivered within a 2 min period through one or more electrodes on the same catheter used for mapping. The electrode configurations and energies used for each patient are listed in table 1. If additional shocks were required, a new catheter was used and the procedure listed as a repeat attempt. Larger energies (300 to 370 J) and smaller numbers of shocks (two) were applied in the first two patients (patients 3 and 5 in table 1). Smaller energies (200 J) and larger numbers of shocks (three to five) were applied to adjacent electrodes in the subsequent eight patients. At four left ventricular free wall sites and four of 10 septal sites, the left ventricular catheter electrode was used as the cathode and a large skin electrode was the anode. At the remaining six septal sites, shocks were delivered between catheter electrodes on either side of the interventricular septum. In three, the left ventricular electrode was used as the cathode and the right ventricular electrode was the anode. In the other

FIGURE 1. Moving a 3 mm spaced bipolar catheter electrode over a 2 cm² area of left ventricular endocardial surface during ventricular tachycardia allowed recording of isolated, low-amplitude potentials at various intervals through the diastolic period (LV tracings). V₁ = electrocardiographic lead V₁; RV = electrogram recorded at the right ventricular apex. Vertical dotted line identifies the onset of the QRS complex.

FIGURE 2. Two left ventricular electrogram patterns recorded at the site of shock delivery. A, Mid-diastolic potentials (arrows) separated by an isoelectric interval from the ventricular potential associated with the QRS complex ("isolated mid-diastolic potential"). Vertical dotted line marks onset of QRS complex. Shocks delivered at this site successfully ablated the tachycardia. B, Ventricular potential continuous with the main body of ventricular activation, beginning 110 msec before the onset of the QRS complex ("early ventricular activation"). Shocks at this site failed to ablate the tachycardia. LV = left ventricular electrogram.
three, the right ventricular electrode was used as the cathode and the left ventricular electrode as the anode\textsuperscript{11} (table 1).

Programmed ventricular stimulation was repeated 30 to 60 min after shock delivery and 5 to 7 days later. The stimulation protocol included single and double extrastimuli during right ventricular pacing at two cycle lengths from the site at which tachycardia was initiated in the preshock study. Triple extrastimuli were also used in the one patient requiring this mode of induction in the reshock study.

Five patients underwent repeat ablation attempts on six ventricular tachycardias. Two repeat attempts were performed with a new catheter during the same session, while the remaining four attempts were performed 10, 10, 15, and 42 days after the initial study.

Successful ablation was defined as both the inability to induce sustained ventricular tachycardia having the same QRS morphology and the absence of spontaneous recurrence during follow-up. Duration of follow-up ranged from 1 to 50 months (median 18 months). The significance of the difference in ablation success between the two electrogram patterns was assessed by the Fisher's exact test.

Results

The locations of shock delivery for the 14 tachycardias are shown in figure 5; there were four left ventricular free wall sites and 10 septal sites. Isolated mid-diastolic potentials were identified in seven ventricular tachycardias. Direct-current shocks were applied to the site recording the mid-diastolic potential in five tachycardias and successfully ablated four. In one of the other two, catheter contact at the site of the mid-diastolic potential terminated the tachycardia. The tachycardia could not be reinitiated during that study and ablation was not attempted. At repeat study the tachycardia was reinitiated but the mid-diastolic potential was not located. In the other tachycardia, the catheter moved away from the mid-diastolic potential site and could not be repositioned to that area. In these two tachycardias and the other seven in which isolated potentials were not initially identified, shocks were delivered at sites of early activation beginning 35 to 120 msec (median 70 msec) before the onset of the QRS complex. Only one of the nine tachycardias was successfully ablated.

A second attempt was made to ablate six of the nine tachycardias in which the initial ablation attempt was unsuccessful. During remapping, an isolated mid-diastolic potential was identified in the one tachycardia in which an unsuccessful shock was initially delivered to a site recording a mid-diastolic potential as well as in two of the five tachycardias in which only early ventricular activation had been previously identified (figure 6). The location of these isolated potentials was

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\begin{align*}
\text{FIGURE 3. Relationship between isolated mid-diastolic potential and initiation of ventricular tachycardia. Two extrastimuli (S}_2 S}_3) \text{ were delivered during right ventricular (RV) pacing at cycle length 400 msec. In panels A and B, some degree of conduction delay occurred after the extrastimuli, as evidenced by the late potentials. However, tachycardia was not initiated until the appearance of a specific isolated potential (first arrow), as shown in panel C. Each tachycardia complex is preceded by the same isolated potential (arrows).}
\end{align*}
\]
approximately 10 to 15 mm from the site at which the shock had originally been delivered. Shocks delivered at these three sites successfully ablated all three tachycardias. In the three remaining tachycardias, mid-diastolic potentials still were not identified. Shocks delivered at sites of early activation were successful in ablatting two of the three tachycardias.

A total of 20 attempts was made to ablate the 14 ventricular tachycardias. Seven of eight attempts at sites recording isolated mid-diastolic potentials were successful compared with only three of 12 attempts at sites recording early ventricular activation (p < .02).

The timing of activation for the 12 ablation attempts at sites of early activation is shown in figure 7. In the three successful attempts, shocks were delivered at sites recording ventricular activation beginning 60, 85, and 120 msec before the onset of the QRS complex during tachycardia. At seven of the nine unsuccessful sites, activation was recorded more than 40 msec before the onset of the QRS complex, and at four, activation was recorded in the same range (70 to 110 msec) as with the successful attempts (figure 7).

The number of shocks applied and total energy delivered were examined for effects on ablation success, independent of electrogram pattern. There was no significant difference between successful and unsuccessful ablation attempts with respect to either of these variables (figure 8).

Both unipolar (four attempts) and transseptal bipolar (10 attempts) electrode configurations were used for septal tachycardias. The numbers are too small for statistical analysis between the groups. However, electrogram patterns may have been a stronger predictor since all three successful unipolar attempts and three of the four successful bipolar attempts were at sites recording mid-diastolic potentials. Of the eight unsuccessful attempts in both groups, seven were at sites of early activation. Of the 10 bipolar attempts, the left ventricular electrode was cathodal in five and anodal in five, with two successes in each group.

Patient follow-up. Of the 10 patients, seven have had no recurrence of ventricular tachycardia, five of these in the absence of antiarrhythmic drugs. These five patients had successful ablation of their single (two patients) or two clinical tachycardias (three patients) and have been followed 1 to 50 months (median 18
<table>
<thead>
<tr>
<th>Patient No.</th>
<th>VT morphology/ cycle length</th>
<th>Location/ Energy</th>
<th>Cathode-anode</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>RBBB, LAD/365 msec</td>
<td>Inferolateral near apex Early activation 200 Joules</td>
<td>LV1-L lat SL</td>
<td>Failure</td>
</tr>
<tr>
<td>Repeat b</td>
<td>10 mm more basal</td>
<td>Mid-diast pot</td>
<td>LV1-L lat SL</td>
<td>Success</td>
</tr>
<tr>
<td></td>
<td>LBBB, LAD/355 msec</td>
<td>Inferior basal septum Mid-diast pot 200 Joules</td>
<td>LV1-RV1</td>
<td>Success</td>
</tr>
<tr>
<td>2</td>
<td>RBBB, RAD/525 msec</td>
<td>Superior septum midway base to apex Mid-diast pot 200 Joules</td>
<td>LV1-L ant SL</td>
<td>Success</td>
</tr>
<tr>
<td>3</td>
<td>RBBB, Ext LAD/ 300 msec</td>
<td>Inferolateral apex Early activation 370 Joules</td>
<td>LV1-L post SL</td>
<td>Failure</td>
</tr>
<tr>
<td>4</td>
<td>LBBB, N1 axis/405 msec</td>
<td>Superior midseptum Mid-diast pot 196 Joules</td>
<td>LV1-L ant SL</td>
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<td>5</td>
<td>LBBB, N1 axis/365 msec</td>
<td>Midseptum Early activation 300 Joules</td>
<td>LV1-L ant SL</td>
<td>Failure</td>
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<tr>
<td>6</td>
<td>LBBB, N1 axis/425 msec</td>
<td>Septum near aortic valve Early activation 200 Joules</td>
<td>LV1-RV1</td>
<td>Failure</td>
</tr>
<tr>
<td>Repeat</td>
<td>Right septum at same level as first attempt Early activation 200 Joules</td>
<td>LV1-LV1</td>
<td>Success</td>
<td></td>
</tr>
<tr>
<td>7</td>
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<td>Failure</td>
</tr>
<tr>
<td>Repeat</td>
<td>More basal location Mid-diast pot 200 Joules</td>
<td>LV1-LV1</td>
<td>Success</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LBBB, LAD/360 msec</td>
<td>Inferior septum midway base to apex Early activation 200 Joules</td>
<td>LV1-LV4</td>
<td>Failure</td>
</tr>
<tr>
<td>Repeat</td>
<td>Inferior right septum near tricuspid anulus Mid-diast pot 200 Joules</td>
<td>LV1-LV1</td>
<td>Success</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>RBBB, LAD/355 msec</td>
<td>Inferolateral wall near base Early activation 200 Joules</td>
<td>LV1-L lat SL</td>
<td>Failure</td>
</tr>
<tr>
<td>Repeat b</td>
<td>More apical Early activation 200 Joules</td>
<td>LV1-L lat SL</td>
<td>Success</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LBBB, LAD/360 msec</td>
<td>Inferoseptal near apex Mid-diast pot 200 Joules</td>
<td>LV1-L lat SL</td>
<td>Success</td>
</tr>
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TABLE 1
(Continued)

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<th>Patient No.</th>
<th>VT morphology/ cycle length</th>
<th>Location^A</th>
<th>Electrogram pattern</th>
<th>Energy (joules)</th>
<th>Cathode-anode</th>
<th>Result</th>
</tr>
</thead>
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<td>Basal inferior septum</td>
<td>Early activation</td>
<td>50</td>
<td>RV4-LV4</td>
<td>Failure</td>
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<td></td>
<td></td>
<td>50</td>
<td>RV3-LV4</td>
<td></td>
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<tr>
<td>Repeat</td>
<td></td>
<td>More basal location</td>
<td>Early activation</td>
<td>100</td>
<td>RV1-LV1</td>
<td>Failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>100</td>
<td>RV2-LV1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>100</td>
<td>RV3-LV1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>100</td>
<td>RV4-LV1</td>
<td></td>
</tr>
<tr>
<td>LBBB, LAD/420 msec</td>
<td>Basal inferior septum near mitral anulus</td>
<td>Early activation</td>
<td>200</td>
<td>LVI-RVI</td>
<td>Failure</td>
<td></td>
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<td></td>
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<td>200</td>
<td>LV1-RV1</td>
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<td>LV1-RV2</td>
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<td>200</td>
<td>LV1-RV3</td>
<td></td>
</tr>
<tr>
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<td>RBBB, RAD/405 msec</td>
<td>Anterolateral wall near base</td>
<td>Early activation</td>
<td>200</td>
<td>LV1-L lat SL</td>
<td>Success</td>
</tr>
<tr>
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<td>200</td>
<td>LV2-L lat SL</td>
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<td>LV3-L lat SL</td>
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<td>200</td>
<td>LV4-L lat SL</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>200</td>
<td>LV1-L lat SL</td>
<td></td>
</tr>
</tbody>
</table>

VT = ventricular tachycardia; RBBB = right bundle branch block configuration (predominant R wave in V1); LBBB = left bundle branch block configuration (predominant S wave in V1); N1 axis = normal frontal plane axis; LAD = left-axis deviation; ext LAD = extreme left-axis deviation; RAD = right-axis deviation; mid-diast pot = isolated mid-diastolic potential; LV1, LV2, LV3, LV4 = distal, second, third, and fourth electrode on left ventricular catheter, respectively; RV1, RV2, RV3, RV4 = distal, second, third, and fourth electrode on left ventricular catheter, respectively; L ant SL = large skin electrode positioned on left anterior chest; L lat SL = large skin electrode positioned on left lateral chest; L post SL = large skin electrode positioned on left posterior chest.

^AAll locations are on the left ventricular endocardium unless otherwise noted.

Repeat ablation attempt was performed during same study using a new catheter.

FIGURE 5. Schematic representation of the left ventricle in the right anterior oblique projection illustrating sites of shock delivery (darkened ovals) for the 14 tachycardias.

One of these patients had four nonclinical ventricular tachycardias inducible at electrophysiologic study both before and after ablation of the clinical tachycardia. No attempts were made to ablate the nonclinical arrhythmias, and none have occurred spontaneously in 50 months of follow-up.

FIGURE 6. Relationship between electrogram pattern and ablation success in a tachycardia requiring two ablation attempts. In the first ablation attempt, shocks were delivered to a site of early activation during ventricular tachycardia, beginning 60 msec before the onset of the QRS complex (top). In the second ablation attempt, shocks were delivered to a site recording an isolated mid-diastolic potential (arrows) that could not be dissociated from the tachycardia (bottom).
Two other patients had successful ablation of one tachycardia each. One of these patients had five inducible, but nonclinical, tachycardias for which no ablation attempt was made. He was restarted on amiodarone for atrial flutter after ablation of his clinical tachycardia and is without recurrence of that tachycardia or of any of five other nonclinical tachycardias over a 16 month period. Amiodarone had previously failed to prevent recurrences of the clinical tachycardia. The second patient had successful ablation of his clinical tachycardia, but subsequently required antiarrhythmic therapy for spontaneous occurrence of one of three inducible, previously nonclinical tachycardias.

Three patients in whom there were failed attempts to ablate four tachycardias received other antiarrhythmic therapy. One patient received amiodarone, which was previously ineffective, and has had no recurrences of ventricular tachycardia. One patient received an implantable defibrillator, had several recurrences of ventricular tachycardia of unknown morphologies, and ultimately died of left ventricular failure. The third patient subsequently died suddenly.

Discussion

These results in a small group of postinfarction patients suggest that ablation of ventricular tachycardia is likely to be successful when shocks are delivered to sites recording isolated mid-diastolic potentials during tachycardia and far less likely to be successful when shocks are delivered to sites of early ventricular activation. Successful ablation at these sites suggests that the mid-diastolic potentials selected in this study were critical for maintaining ventricular tachycardia. Other areas exhibited mid-diastolic potentials during ventricular tachycardia, but in many of these areas, ventricular extrastimuli could dissociate the potential from changes in timing of the following QRS complex. Areas in which changes in the timing of the diastolic potential consistently preceded identical changes in the timing of the QRS complex were targeted for ablation. Additional support for the relationship of the potential to the tachycardia was obtained by the appearance and disappearance of the potential on initiation and termination of the tachycardia. Since the response of the potential during initiation and termination was concordant with the resetting response to extrastimuli, the resetting criterion alone may be sufficient.

A number of additional factors may influence the success of catheter ablation, including the electrode configuration, electrode polarity, the number of shocks, and the amount of energy used. These factors were not systematically controlled in this study, and it is not possible to adequately assess their influence on ablation success independent of electrogram pattern. When the number of shocks and total energy used were compared, there was no difference in the ablation success rate, as reported by other investigators. Similarly, for septal tachycardias, successful ablation seemed to correlate better with the electrogram pattern than with unipolar vs bipolar or cathodal vs anodal shocks.

The isolated mid-diastolic potential would seem to
represent activation of a segment of the slowly conducting region of the ventricular reentrant circuit, and a critical component of the reentrant circuit. The infrequent recording of continuous electrical activation at these sites suggests that other portions of the slowly conducting region lay outside the recording range of the bipolar electrodes. These findings are consistent with studies in dogs after infarction showing reentrant circuits containing relatively narrow regions of slow conduction isolated by arcs of functional block. The reentrant impulse exits from this region in two directions to form two circuits of more rapidly conducting wavefronts (figure 9). In this model, early activation might be recorded over a relatively large exit region represented by the striped area in figure 9. Destruction of part of this area may not significantly affect the reentrant mechanism. In animal studies, application of a cryoprobe to the exit region was unsuccessful in terminating the tachycardia unless large areas were cooled. Cooling small areas in the slowly conducting region consistently terminated tachycardia. Intraoperative mapping studies in postinfarction patients have shown that sites of cryotermination may be 3 to 7 cm from sites of early activation during ventricular tachycardia. In patients with ventricular tachycardia associated with healed myocardial infarction, the region of slow conduction may be isolated or protected by an anatomic barrier in addition to or instead of the arc of functional block in the acute and subacute canine preparation of infarction.

This model may explain the failure of catheter ablation at sites of early activation (exit sites) beginning as long as 110 msec before the QRS complex. Other investigators have similarly found a poor relationship between the timing of early activation and success of catheter ablation. The high success rates for surgical ablation aimed at sites of early activation may relate to the large area of resection, which could include all or part of the slowly conducting region. With the smaller areas of necrosis resulting from catheter-delivered shocks, targeting an area of the slowly conducting region assumes greater importance.

Mid-diastolic potentials were not identified in five of the 14 tachycardias. The absence of these potentials might suggest a mechanism other than reentry or a different functional configuration of the circuit. Within the context of reentry, the area of slow conduction in these five tachycardias may have been very small or may have been located intramurally. Another possibility is that we may not have been able to maneuver the catheter to the appropriate area.

Some investigators have emphasized that mid-diastolic potentials during tachycardia may be generated by myocardium not critically related to the tachycardia circuit, and have advised caution in use of these potentials to identify the site of origin of tachycardia. In this study, isolated mid-diastolic potentials were believed to be critically related to the tachycardia only when they remained associated with the tachycardia during initiation, termination, and resetting by extrastimuli. The high ablation success rate for shocks delivered at these sites confirms the ability of programmed stimulation techniques to identify potentials that are critically related to the tachycardia.

References
2. Evans GT Jr, Scheinmann MM, and the Executive Committee of the Percutaneous Cardiac Mapping and Ablation Registry: Catheter ablation for control of ventricular tachycardia: a report of the Percutaneous Cardiac Mapping and Ablation Registry. PACE 9: 1391, 1986

FIGURE 9. Model of reentry, adapted from El-Sherif et al.,11 incorporating a slowly conducting channel (stippled area) formed by two arcs of functional block (bold vertical lines). The reentrant impulse is conducted from the exit region (striped area) over relatively normally conducting myocardium to the entrance of the slowly conducting channel. See text for discussion.

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