Prevention of ventricular tachycardia induction during right ventricular programmed stimulation by high current strength pacing at the site of origin

FRANCIS E. MARCHLINSKI, M.D., ALFRED E. BUXTON, M.D., JOHN M. MILLER, M.D., AND MARK E. JOSEPHSON, M.D.

ABSTRACT To determine whether high current strength pacing at the site of origin of ventricular tachycardia (VT) could prevent induction of VT, we studied 11 VTs in 10 patients with chronic coronary artery disease. The left ventricular site of origin of all VT was determined by endocardial catheter mapping. Reproducible VT induction from the right ventricular apex or outflow tract was demonstrated with a pacing current strength equal to twice diastolic threshold (≤2.0 mA) with single (two VTs), double (eight VTs), or triple (one VT) extrastimuli following 8 beats of a drive cycle length of 400 to 600 msec. After determination of the baseline VT induction zone (range 10 to 80 msec), repeat induction was attempted while simultaneous pacing was performed during the 8 beat drive train from the left ventricular site of origin with the use of a high current strength (10 mA [two VTs] or 20 mA [nine VTs]) and from the baseline right ventricular site with a current strength equal to twice diastolic threshold. Extrastimuli were introduced only from the right ventricular site over the same range of coupling intervals that resulted in VT initiation during the baseline state. In five of the 11 trials, no VT could be initiated; in one trial, the VT induction zone was decreased from 80 to 10 msec; in three trials, only VT of a different morphology and a distinct (>4 cm distant) site of origin was initiated; and in two trials, VT of the same morphology was initiated. In four of the five trials in which all VT was prevented by simultaneous pacing with a high current strength at the site of origin, simultaneous pacing at a lower current strength (twice diastolic threshold) at the site of origin (three VTs) or with equally increased current strength (10 to 20 mA) at nonsites of origin (two VTs) did not prevent initiation. We conclude that: (1) high current strength pacing at the site of origin during the drive train can inhibit VT induction with extrastimuli and, (2) successful prevention of VT may depend on the pacing site being the site of origin and the current strength used during pacing.


PACING TECHNIQUES in the management of ventricular tachyarrhythmias have been used almost solely as a means for arrhythmia termination.1–5 Only in the management in preventing ventricular tachyarrhythmias of patients with the long QT syndrome has pacing proven effective with any degree of consistency.4–6 The purpose of this study was to determine whether high current strength ventricular pacing at the site of origin of sustained ventricular tachycardia occurring in the setting of chronic coronary artery disease could prevent arrhythmia induction during programmed stimulation. We hypothesized that pacing with high current strength at the site of origin of the ventricular tachycardia could alter the necessary electrophysiologic substrate for reentry and prevent induction of ventricular tachycardia by extrastimuli.

Methods

Patient population. The study population consisted of 10 men ranging in age from 47 to 72 years (mean 58). All 10 patients had spontaneous episodes of sustained ventricular tachycardia that occurred in the setting of chronic coronary artery disease with documented prior myocardial infarction. All patients were candidates for catheter or surgical ablative therapy of their ventricular arrhythmia and underwent electrophysiologic study and endocardial catheter mapping of their ventricular tachycardia after giving informed written consent. Three pa-
1. FIGURE 1. Left ventricular endocardial mapping schema for ventricular tachycardia: Site 1, apex; sites 2, 3, and 4, septum; site 5, inferior wall; sites 6 and 8, posterior base; sites 7, 9, and 10, lateral wall; and sites 11 and 12, superior free wall.

2. Demonstration of the left ventricular endocardial site of origin of ventricular tachycardia. The panels are arranged with surface electrocardiographic leads of I, aVF, V1, intracardiac recordings from the right ventricular apex (RVA), right ventricular outflow tract (RVOT), left ventricular (LV) site of origin (SOO), and 10 msec time lines. LV site 2-11 demonstrated the earliest local electrical activity, which preceded the onset of the surface QRS during ventricular tachycardia endocardial catheter mapping by 80 msec. In addition the effect of pacing (entrainment) from the RVA on the recording at the site of origin is shown. Presystolic activity maintains the same relationship to the onset of the QRS complex immediately after termination of pacing (−80 msec) with the return cycle of the recording at LV-SOO 2-11 equal to the cycle length of pacing.

Patients were studied in the absence of antiarrhythmic therapy while the remaining seven patients underwent study during therapy with procainamide, amiodarone, mexiletine, tocainide, quinidine, or disopyramide alone or in combination. All patients had a sustained ventricular tachycardia of a uniform morphology that was induced reproducibly during programmed stimulation and was tolerated hemodynamically. Eleven tachycardias were studied in the 10 patients.

Catheter mapping techniques. Catheter mapping of the induced ventricular tachycardias was performed by previously described methods. One quadripolar (5 mm interelectrode distance) catheter (No. 6F USCI) was inserted percutaneously into a femoral artery and advanced to the left ventricle under fluoroscopic guidance. The left ventricular mapping schema is shown in figure 1. The site of origin of ventricular tachycardia as determined by catheter mapping was defined as the site demonstrating the earliest recorded electrical activity occurring in the latter half of electrical diastole (figure 2). The mapped site of origin and the amount by which the recorded electrical activity at this site preceded the QRS onset for each of the 11 tachycardias in the 10 patients studied are listed in table 1. During all tachycardias the earliest site of presystolic activity that had been determined by detailed endocardial catheter mapping was demonstrated to maintain a fixed relationship of local activation to the onset of the QRS after rapid ventricular pacing and/or premature ventricular extrastimuli that entrained or reset the tachycardia without terminating it (figure 2). This stimulation was performed to ensure that recorded early presystolic activity did not represent delayed activation of infarcted myocardium that was not related to the tachycardia mechanism.

Pacing protocol. For each of the ventricular tachycardias a zone of coupling intervals for the extrastimuli was defined that resulted in the reproducible (at least two consecutive initiations at each coupling interval) induction of the mapped ventricular tachycardias. Induction of ventricular tachycardia (table 1) was achieved with single, double, or triple extrastimuli during drive
TABLE 1
Characteristics of ventricular tachycardia and response to high current strength pacing

<table>
<thead>
<tr>
<th>VT No.</th>
<th>VT morphology</th>
<th>VT cycle length (msec)</th>
<th>VT site of origin</th>
<th>Pre-systolic activity (msec)</th>
<th>LV SOO threshold (mA)</th>
<th>RV site/cycle length (msec)</th>
<th>No. of extrastimuli for VT induction</th>
<th>VT zone (RV 2× threshold)</th>
<th>Response to simultaneous LV pacing</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>LBRs</td>
<td>330</td>
<td>2</td>
<td>100</td>
<td>3.0</td>
<td>RVA/500</td>
<td>D</td>
<td>270/490-270/470</td>
<td>No VT</td>
</tr>
<tr>
<td>2</td>
<td>RBLs</td>
<td>320</td>
<td>2-3</td>
<td>40</td>
<td>1.6</td>
<td>RVA/400</td>
<td>D</td>
<td>280/510-280/490</td>
<td>No VT</td>
</tr>
<tr>
<td>3</td>
<td>LBLs</td>
<td>380</td>
<td>Inf 2</td>
<td>55</td>
<td>1.4</td>
<td>RVA/400</td>
<td>S</td>
<td>290-260</td>
<td>VTC</td>
</tr>
<tr>
<td>4</td>
<td>RBRI</td>
<td>430</td>
<td>3-11</td>
<td>80</td>
<td>2.2</td>
<td>RVOT/400</td>
<td>D</td>
<td>280/500</td>
<td>No VT</td>
</tr>
<tr>
<td>5</td>
<td>RBRS</td>
<td>340</td>
<td>6-8</td>
<td>60</td>
<td>4.4</td>
<td>RVOT/400</td>
<td>S</td>
<td>280-270</td>
<td>No VT</td>
</tr>
<tr>
<td>6</td>
<td>RBRI</td>
<td>430</td>
<td>2-3-11</td>
<td>85</td>
<td>1.0</td>
<td>RVA/400</td>
<td>D</td>
<td>310/590-310/580</td>
<td>VTC</td>
</tr>
<tr>
<td>7</td>
<td>RBRI</td>
<td>360</td>
<td>8</td>
<td>80</td>
<td>1.0</td>
<td>RVA/400</td>
<td>D</td>
<td>240/420-220/400</td>
<td>VT</td>
</tr>
<tr>
<td>8</td>
<td>RBLs</td>
<td>370</td>
<td>2</td>
<td>30</td>
<td>4.8</td>
<td>RVOT/400</td>
<td>D</td>
<td>320/600-300/580</td>
<td>No VT</td>
</tr>
<tr>
<td>9</td>
<td>RBLi</td>
<td>420</td>
<td>2-11</td>
<td>80</td>
<td>1.9</td>
<td>RVA/400</td>
<td>D</td>
<td>290/490</td>
<td>VTC</td>
</tr>
<tr>
<td>10</td>
<td>RBLI</td>
<td>350</td>
<td>4-12</td>
<td>65</td>
<td>6.0</td>
<td>RVOT/550</td>
<td>D</td>
<td>300/530-270/480</td>
<td>VT induction zone</td>
</tr>
<tr>
<td>11</td>
<td>RBLI</td>
<td>400</td>
<td>3</td>
<td>40</td>
<td>6.0</td>
<td>RVA/600</td>
<td>T</td>
<td>290/580-840/290/580/820</td>
<td>VT</td>
</tr>
</tbody>
</table>

*Sites of origin that were intermediate in location are designated as Inf 2 (Inferior to site 2), 3-11 (between sites 3 and 11), etc.

VVT induction zone indicated of the S1/S2/S3/S4 coupling intervals that result in reproducible VT induction.

VT of different morphology and distant (>4 cm) site of origin.

D = double extrastimuli; LB = left bundle branch block; LI = left inferior axis-0- (+90°); LS = left superior axis-0- (-90°); LV = left ventricular; RB = right bundle branch block; RI = right inferior axis (+90°)-180°; RS = right superior axis (-90°)-180°; RV = right ventricular; RVA = right ventricular apex; RVOT = right ventricular outflow tract; S = single extrastimulus; SOO = site of origin; T = triple extrastimuli; VT = ventricular tachycardia.

cycle lengths (8 beat duration) of 400 to 600 msec from the right ventricular apex or outflow tract by use of our previously described stimulation protocol (table 1). All stimulation was performed with a programmable stimulator with an optically isolated constant current source (Bloom Associates, Reading, PA). The bipolar stimuli were 1 msec in duration, and were delivered at twice diastolic threshold (0.8 to 2.0 mA).

After definition of the zone of coupling intervals (table 1) that reproducibly initiated the tachycardia with the use of stimulation from the right ventricular site only, “preventive” pacing was attempted by the following protocol.

**Step 1.** The diastolic threshold for pacing at the site of origin was determined with bipolar stimulation with an interelectrode distance of 0.5 (two ventricular tachycardias) or 1.0 cm (nine ventricular tachycardias) and a 1 msec pulse width (table 1).

**Step 2.** Reinitiation of ventricular tachycardia was attempted with the same drive cycle length, number of extrastimuli, and zone of coupling intervals as that which initiated ventricular tachycardia in the baseline state. However, during the 8 beat drive cycle (before introduction of extrastimuli), pacing was performed simultaneously from the right ventricular site at twice diastolic threshold and from the left ventricular site of origin with a high current strength (10 mA in two ventricular tachycardias [VTs No. 1 and 2] or 20 mA in nine ventricular tachycardias) (figure 3). The pulse duration at both sites equaled 1 msec. The first beat of the drive train for both right and left ventricular pacing was triggered to begin at a coupling interval equal to the paced cycle length used in the control state, with the electromgram recorded from the right ventricular site during sinus rhythm as the recorded signal triggering the pacing. The accuracy of the stimulation (Bloom Assoc Ltd.) for the introduction of the simultaneous pacing was within 1 msec. Extrastimuli were introduced only from the right ventricular site that resulted in reproducible induction of ventricular tachycardia in the baseline state. If ventricular tachycardia was not initiated over the range of coupling intervals that reproducibly initiated ventricular tachycardia.

**FIGURE 3.** Illustration of the stimulation protocol and the effectiveness of simultaneous pacing in narrowing the zone of coupling intervals of double extrastimuli that result in induction of ventricular tachycardia. The panels are arranged and abbreviations are as in figure 2. In A, ventricular tachycardia is induced over a wide range of coupling intervals of double extrastimuli introduced from the right ventricular outflow tract after 8 beats of pacing from the right ventricular outflow tract at a cycle length of 550 msec. All stimulation is at twice diastolic threshold. The ventricular tachycardia site of origin is localized to the superior aspect of the basal septum (Site 4-12), with the maximum presystolic activity recorded at this site. In B, when left ventricular pacing at 20 mA is performed simultaneously with right ventricular outflow tract pacing at two times threshold during the drive train, extrastimuli introduced from the right ventricular outflow tract are able to initiate ventricular tachycardia only at the shortest coupling interval. In C, bipolar stimulation of the LV was performed via electrodes 1-2 (used for recording in panel A), with electrical activity at the site of origin recorded from electrode pair 3 and 4 on the quadripolar catheter.
tachycardia in the baseline state, the coupling intervals of the extrastimuli were decreased until the first extrastimulus no longer resulted in a propagated response.

Step 3. After the attempt at reinitiation during simultaneous pacing, right ventricular stimulation was repeated with the same number of extrastimuli and coupling intervals that initiated ventricular tachycardia in the baseline state. This served as an additional control for the reproducibility of ventricular tachycardia induction.

To determine whether a critical current strength for left ven-

FIGURE 3. For legend see opposite page.
tricular site of origin pacing was required to prevent ventricular tachycardia initiation, step 2 of the protocol was repeated in three patients (VTs No. 1, 2, and 4) with a current strength of twice diastolic threshold for both site of origin and right ventricular stimulation (figure 4). Finally, to assess site specificity of the protocol, step 2 was repeated in two patients by use of a site (right ventricular outflow tract or mid right ventricular septum) that was distant from the left ventricular site of origin. Repeat simultaneous pacing at different current strengths or at sites remote from the site of origin could not be performed in all patients because of (1) patient fatigue related to the duration of the protocol or (2) catheter movement that precluded assurance of repeated stimulation attempts at exactly the same site. Of note, four of the five patients (VTs No. 1, 2, 4, and 8) in whom simultaneous pacing at the left ventricular site of origin at high current strength prevented ventricular tachycardia initiation (see results) also underwent repeat simultaneous pacing at a lower current strength and/or a site remote from the site of origin.

Effect of high current strength on local refractoriness and conduction. The effects of high current strength pacing on local refractoriness and conduction in five of the 10 patients was assessed as follows:

(1) The effective refractory period at the site of origin was determined after 8 beats at a constant drive cycle length (400 or 600 msec) with the current strength during drive beats equal to diastolic threshold, twice threshold, and 20 mA. The extrastimulus was introduced from the same site at twice diastolic threshold. The extrastimulus was introduced at progressively shorter coupling intervals (10 msec decrements). The effective refractory period was defined as the longest coupling interval of the extrastimulus that failed to result in a propagated response.

Indexes of local conduction at the site of origin were measured during steady-state (at least 30 beats) pacing at a cycle length of 400 to 500 msec with current strengths equal to threshold, twice threshold, and 20 mA. Local conduction was indexed by the local activation time that was measured as the time from the stimulus artifact to the first rapid deflection recorded from electrode pair 2 and 4 (1.0 cm interelectrode distance on the quadripolar pacing catheter), while pacing from electrode pair 1 and 3. This electrode pair was chosen to mimic the interelectrode distance used during the "preventive" pacing protocol with at least one recording electrode (electrode 2) in close approximation to the ventricular endocardium. In addition, the time to completion of local activation was indexed by the total electrogram duration and was defined as the time from the stimulus artifact to the end of the local electrogram recorded at a fixed gain setting of 1 cm = 1 mV. This measurement was made as an index of local conduction time that would not be potentially masked by the decay of the signal due to the pacing stimulus. All signals were filtered at 30 to 500 Hz and were measured from recordings obtained with a constant gain setting at a paper speed of 250 mm/sec.

Data analysis. Results are expressed as the mean ± SD unless otherwise indicated. Analysis of variance was used when

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**FIGURE 4.** The influence of current strength on prevention of ventricular tachycardia induction with pacing at the left ventricular site of origin (LV-SOO). Ventricular tachycardia is induced during right ventricular apical (RVA) stimulation only (top) and when right ventricular apex and left ventricular site of origin are both paced at two times threshold during the drive train (middle). Only when pacing the left ventricular site of origin at 10 mA during the drive train was VT induction by extrastimuli from the RVA prevented (bottom). Distortion of surface electrocardiographic (ECG) recordings are observed with LV pacing and are related to poor surface ECG isolation in this patient. The panels are arranged similar to figure 2. (Recording from the right ventricular outflow tract is only demonstrated in the middle).
comparing refractory periods and conduction times at different current strengths. A value of \( p < .05 \) was considered indicative of a significant difference.

**Results**

**Characteristics of induced ventricular tachycardia.** The morphology, cycle length, and site of origin of induced ventricular tachycardias are listed in table 1. The sites of origin of six of the tachycardias were localized to the left ventricular septum, those of four to the anterior free wall adjacent to the septum, and those of two to the posterior base. Local electrical activity at the sites of origin preceded the QRS by 30 to 100 msec (mean 65). Two ventricular tachycardias were induced with single extrastimuli, seven with double extrastimuli, one with triple extrastimuli, and one with both double and triple extrastimuli. The range of coupling intervals resulting in reproducible initiation of ventricular tachycardia from the right ventricle at twice diastolic threshold are also listed in table 1. A 10 to 80 msec range of coupling intervals (tachycardia induction zone) was determined for each ventricular tachycardia.

**Effects of high current strength pacing on tachycardia inducibility (table 1).** Pacing at high current strength prevented ventricular tachycardia induction by the same number of extrastimuli as in the baseline state in five of the 11 (45%) ventricular tachycardia induction trials, and resulted in a marked decrease in the zone of coupling intervals of double extrastimuli that induced ventricular tachycardia in one additional trial (figures 3 and 5). In three patients ventricular tachycardia of a different morphology and distinct mapped site of origin (>4 cm from original site of origin) was induced during stimulation over the same range of (two ventricular tachycardias) or with closer (one ventricular tachycardia) coupling intervals when compared with those used in the baseline state (figure 6). The same ventricular tachycardias were induced over the same range of coupling intervals during simultaneous pacing as during the baseline state in two trials. In one of these trials triple extrastimuli were required for initiation of ventricular tachycardia while in the second trial double extrastimuli were always followed by 5 to 8 beats of polymorphic ventricular tachycardia before a stable unimorphic tachycardia ensued. In two of the five trials in which initiation of sustained ventricular tachycardia was prevented, one or two repetitive ventricular responses were observed. These repetitive responses had a different QRS morphology when compared with the sustained ventricular tachycardia.

**Effect of stimulation site and current intensity.** In both trials in which a distant pacing site (nonsite of origin) was used during simultaneous pacing the same ventric-

**TABLE 2**

| Effect of pacing current strength on local conduction times and refractoriness at site of origin |
|----------------------------------|----------------------------------|------------------|------------------|
| Pacing current strength          | Threshold (2.9 ± 2.3 mA)         | 2 × threshold    | 20 mA            |
| Time from pacing stimulus to rapid LV deflection (msec) | 131 ± 79 | 124 ± 83 | 108 ± 72\(^a\) |
| Time from pacing stimulus to end of local electrogram (msec) | 195 ± 23.7 | 187 ± 33 | 169 ± 24\(^a\) |
| Effective refractory period (msec) | 280 ± 24 | 280 ± 24 | 279 ± 25          |

\(^a\) \( p < .05 \) vs threshold values.

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FIGURE 5. Prevention of ventricular tachycardia induction by high current strength pacing at the left ventricular site of origin (LV-SOO) of the ventricular tachycardia. In A, ventricular tachycardia is induced during stimulation from the right ventricular apex (RVA) with extrastimuli coupling intervals ranging from 280/230 to 280/210 msec. The recording from site of origin (site 2-3) is shown in the middle tracing of panel A and demonstrates presystolic activity beginning 40 msec before the onset of the surface QRS complex during the induced ventricular tachycardia. In B, prevention of ventricular tachycardia induction is observed when the left ventricular site of origin (10 mA) and RVA (two times threshold) are paced simultaneously during the drive train (Note fusion complex on surface electrocardiogram.) Double extrastimuli, which are introduced from the RVA over an even wider range of coupling intervals than during baseline stimulation, fail to induce ventricular tachycardia.
Pacing has been used successfully to prevent ventricular arrhythmias associated with the long QT syndromes. There have also been several reports suggesting that the frequency of spontaneous ventricular ectopic activity and to a lesser extent sustained ventricular arrhythmias can be decreased or completely prevented by atrial and/or right ventricular apical pacing in the setting of acute or chronic coronary artery disease. Results have been inconsistent and atrial and/or right ventricular pacing appears to be preventive in only a highly select group of patients.

Endocardial catheter mapping is an accurate technique for localizing the site of origin of ventricular tachycardia. The electrogram at the site of origin of ventricular tachycardia determined by catheter mapping is believed to represent activation of an area near an essential part of the reentrant circuit just before exit from the circuit. Subsequently, activation of the remaining mass of the ventricles is manifest by the surface QRS complex. This concept has been supported by the behavior of the electrogram recorded from the site of origin during pacing and premature stimulation, which result in entrainment or resetting of the tachycardia. In addition, the success of localized ablation procedures (catheter fulguration or surgical excision) based on these mapping techniques also suggest that presystolic activity recorded during mapping can localize an essential arrhythmogenic area for sustained ventricular tachycardia.

The present study demonstrates that pacing at a high current strength (10 to 20 mA) at the site of origin during the drive train prevents ventricular tachycardia induction with right ventricular stimulation in 45% of ventricular tachycardia trials. A marked decrease in the coupling intervals of the extrastimuli that result in induction of ventricular tachycardia or the induction of only ventricular tachycardias having a different morphology and distant site of origin was shown in an additional four ventricular tachycardia trials. It is unknown whether induction of ventricular tachycardia of a different morphology suggests some protective effect. It is possible that the pacing technique resulted in an alteration of activity through part of the reentrant circuit and changed the site of exit and subsequent ventricular activation while maintaining the same anatomic location for the wavefront of the reentrant circuit. Given the marked disparate anatomic site of earliest presystolic activity for the morphologically different tachycardias, the reentrant “circuit” would have to be more sizeable than previously suggested.

The possibility that high current strength pacing at the site of origin of one ventricular tachycardia actually promoted or unmasked the initiation of ventricular tachycardias originating from more distant sites also
FIGURE 7. Effect of current used for pacing on local activation time. The panels are arranged and abbreviations are as in figure 2. Pacing with increasing current resulted in a progressive decrease in the local activation time as indexed by the time from the pacing artifact to first rapid deflection recorded at the left ventricular site of origin, with a marked decrease occurring when pacing at 20 mA. A decrease was also observed for the activation time of more distant right ventricular sites.

must be considered. Nevertheless, in six of the 11 ventricular tachycardias a preventive effect with regard to the ability to induce ventricular tachycardia was observed. Whether this pacing technique could be applied to prevent spontaneous ventricular tachycardia is uncertain.

Specificity of site and current strength. Although preliminary, it appears that this preventive effect of simultaneous pacing may be site specific. Initiation of ventricular tachycardia was not prevented when simultaneous pacing was performed at a site that was not the site of origin of the ventricular tachycardia. In addition, the ability to initiate a ventricular tachycardia that originated at a distant site at least 4 cm away from the original mapped site in three of the ventricular tachycardia preventive pacing trials suggests that the site of stimulation during simultaneous pacing is critical in preventing the arrhythmia. It also appears that it may be necessary to depolarize uniformly a larger area of myocardium than is depolarized by pacing at just twice diastolic threshold. All three patients who were so tested demonstrated efficacy of the preventive pacing only at higher current strengths of 10 or 20 mA but not at twice threshold.

Mechanism of prevention. Although the effective refractory period has been demonstrated to decrease when the current strength of the extrastimulus is increased, we found no effect on refactoriness as the current strength of the drive train was increased. This finding is supported by a preliminary report by Al Bitar et al., who found no change in refactoriness when altering the current strength of the drive train with stimulation performed from the right ventricular apex. This suggests that there are no significant short-
long-term effects on membrane recovery related to pacing at the described high current. Indexes of local myocardial activation, on the other hand, were altered by increasing the current strength. We noted a significant decrease in the time to local activation and the total local activation when the pacing current strength was increased. The decrease suggests more uniform local activation that may permit a longer period of recovery in areas of markedly slowed conduction before a premature stimulus is introduced. How this effect on activation at the site of origin prevents the development of ventricular tachycardia after subsequent premature extrastimuli remains speculative. Changes related to the altered effective coupling interval of the wavefront of activation of the premature stimulus at the site of origin may alter refractoriness and conduction in this surrounding myocardium, which in turn may alter subsequent wavefronts of activation or simply preclude the development of the requisite slow conduction or unidirectional block required for the development of sustained ventricular tachycardia. More investigation is necessary before any firm conclusions related to the possible mechanism of the observed preventive effect can be drawn.

Limitations. The study population was a select group of patients with coronary artery disease and ventricular tachycardia that was reproducibly inducible and well tolerated hemodynamically. One cannot conclude that the described preventive pacing technique would be effective in patients without coronary artery disease or with faster ventricular tachycardias. Second, the limited assessment of the importance of the pacing site and the current strength make conclusions with respect to the specificity of pacing site and the importance of the current strength preliminary. Confirmation of results and further study in this area are indicated. Finally, local activation time as indexed by recording of electrical activity from the second pair of electrodes on the pacing catheter can be considered only a relatively crude index of local activation. Decreases in local activation time and total electrogram duration may be a reflection of more simultaneous and therefore more rapid local activation. However, the same changes may be due to local conduction block. Confirmation and further elucidation of the effects on local activation as a result of an increase in pacing current strength and alterations that occur with subsequently introduced premature stimuli will require simultaneous recordings from multiple closely spaced electrode pairs.

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