A Quantitative Evaluation of Refractoriness Within a Reentrant Circuit During Ventricular Tachycardia Relation to Termination

Charles D. Gottlieb, MD, Mark E. Rosenthal, MD, Nicholas J. Stamato, MD, Lawrence H. Frame, MD, Michael D. Lesh, MD, John M. Miller, MD, and Mark E. Josephson, MD

Programmed ventricular stimuli introduced during sustained monomorphic ventricular tachycardia frequently reset the tachycardia, resulting in a less than fully compensatory pause. A resetting response curve is generated when the set of return cycles is evaluated as the function of the coupling intervals of the extrastimuli delivered during the ventricular tachycardia. If the stimulated wave front encounters tissue within the tachycardia circuit that is not fully recovered, interval-dependent conduction changes should occur producing an increasing resetting response pattern. We quantified the magnitude of this interval-dependent conduction slowing in 17 morphologically distinct ventricular tachycardias. The slope of the increasing limb of the resetting response curve was determined by linear regression analysis and ranged from −0.30 to −1.14 (mean±SD, 0.70±0.25). Seven of the 17 ventricular tachycardias (41%) terminated during introduction of ventricular extrastimuli. The slope of the resetting response pattern in those ventricular tachycardias that terminated were significantly steeper than in those that did not terminate (−0.85±0.15 versus −0.61±0.21, respectively, p=0.02%). Six of the seven ventricular tachycardias terminated with programmed ventricular stimuli had a slope steeper than −0.75, whereas only one of 10 ventricular tachycardias that did not terminate exceeded this value. In conclusion, the slope of the increasing portion of the resetting response curve correlates with ability to terminate uniform sustained ventricular tachycardia by timed extrastimuli. This slope is the quantification of the magnitude of interval-dependent conduction slowing. Additionally, tissue within the reentrant circuit displaying greater degrees of interval-dependent conduction slowing may also have relatively longer effective refractory periods. (Circulation 1990;82:1289−1295)

Evidence strongly suggests that reentry is the mechanism of ventricular tachycardia occurring in a setting of a remote myocardial infarction.1–3 The reentrant pathway may be defined by anatomical, functional, or anatomical and functional characteristics of the ventricular tissue. Extra-

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stimuli introduced during sustained monomorphic ventricular tachycardia frequently reset the tachycardia, resulting in a less than fully compensatory pause.4 The ability to reset a reentrant tachycardia implies that the circuit must contain an excitable gap. Qualitative and quantitative evaluation of the functional characteristics of this excitable gap may be determined by examination of the resetting response pattern that is generated when extrastimuli are delivered over a range of coupling intervals. A resetting response curve is constructed by plotting the return cycle (the interval between the stimulated and the subsequent tachycardia complex) as a function of the coupling interval of the extrastimulus resetting the tachycardia.

Three resetting response patterns have been previously noted in sustained ventricular tachycardia,
that is, a flat response, an increasing response, and a response that is flat at relatively long coupling intervals and increasing at shorter coupling intervals. The pattern of response (flat vs. increasing) is assumed to be determined by the state of refractoriness of tissue within the reentrant circuit. Should the stimulated wave front encounter tissue within the tachycardia circuit that exhibits interval-dependent conduction slowing (i.e., the tissue is in a state of relative refractoriness), an increasing curve will be seen. We propose that the slope of the increasing portion of a resetting response curve provides an index of relative refractoriness of tissue within the reentrant circuit. Termination of ventricular tachycardia with a premature extrastimulus will occur when the stimulated impulse encounters tissue within the circuit that is in its effective refractory period.

The purpose of this investigation is to quantitatively evaluate the magnitude of interval-dependent conduction changes seen during resetting of ventricular tachycardia and to relate the steepness of the increasing response curve to the ability of extrastimuli to terminate the ventricular tachycardia. Our hypothesis is that the ventricular tachycardias that demonstrate pronounced interval conduction changes may be more likely to terminate as a result of extrastimuli introduced during resetting of ventricular tachycardia.

Methods

Patients and Tachycardias

For inclusion in this study, we required that ventricular tachycardia 1) be sustained, monomorphic, and initiated by programmed stimulation, 2) have a stable mean cycle length varying less than 20 msec over 20 consecutive beats, 3) be hemodynamically tolerated, and 4) exhibit no change in morphology or acceleration during the study protocol.

Additionally, all tachycardias included in this study demonstrated an increasing portion to the resetting response curve with double ventricular extrastimuli in which only the second interacted with the circuit. Only the data derived in response to double ventricular extrastimuli were analyzed because we have previously shown that double extrastimuli achieved greater relative prematurity and therefore delineate more of the increasing curve than do single extrastimuli. Seventeen morphologically distinct ventricular tachycardias occurring in 14 patients met these criteria and were included in the study. Nine tachycardias had a right bundle branch block morphology defined as the presence of a monophasic, biphasic, or triphasic R wave in lead V1 or a QR configuration in lead V1. Eight ventricular tachycardias had a left bundle branch block pattern defined as QS, rS, or a qRS in lead V1.

Electrophysiological Studies

All electrophysiological studies were performed with the patient in a postabsorptive state after informed, written consent had been obtained. One to five 6F quadripolar catheters with a 5-mm interelectrode distance were inserted percutaneously and positioned in the heart under fluoroscopic guidance.

Stimulation was performed with a custom-designed stimulator (Bloom Associates, Reading, Pa.). Bipolar stimulation was always performed with a distal electrode as the cathode. Stimuli were delivered as rectangular pulses 1 msec in duration and at twice late diastolic threshold. Data obtained during the procedure were displayed on a multichannel oscilloscope (Electronics for Medicine VR16, Pleasantville, N.Y.), recorded with an inkjet recorder (Siemens, Iselin, N.J.) at a paper speed of 100, 200, or 250 mm/sec, and simultaneously stored on analog magnetic tape (Honeywell 5600). At least three surface electrocardiographic leads (usually I, aVF, and V1) were recorded simultaneously along with intracardiac electrograms filtered at 30–500 Hz. To initiate ventricular tachycardia, 1–3 ventricular extrastimuli, rapid ventricular pacing, or both were performed as previously described. Twelve-lead electrocardiograms of the ventricular tachycardia were monitored to demonstrate the continued presence of the same ventricular tachycardia during the stimulation protocol.

Stimulation Protocol During Ventricular Tachycardia

All stimulation during the ventricular tachycardia was performed from the same site within the right ventricle. Single ventricular extrastimuli were delivered during the ventricular tachycardia beginning 20 msec less than the cycle length and decremented by 5–10-msec intervals until 1) resetting occurred or 2) ventricular refractoriness was encountered at the pacing site. Double ventricular extrastimuli were delivered with the first extrastimulus fixed 20 msec above the longest coupling interval, which caused resetting or 20 msec above ventricular refractoriness if no resetting occurred with the single extrastimulus. Therefore, the first extrastimulus did not interact with the tachycardia circuit. The second ventricular extrastimulus was then introduced, beginning at a coupling interval to the first extrastimulus equal to the tachycardia cycle length and then delivered in 5–10-msec decrements until termination occurred or ventricular refractoriness was encountered at the pacing site. The coupling interval of the first extrastimulus remained fixed at all times during the stimulation protocol.

Measurements and Definitions

All intervals were measured between the rapid deflection of the local electrogram recorded from the pacing catheter.

The coupling interval. The coupling interval was the interval from the ventricular tachycardia complex before stimulation to the last stimulated complex measured at the pacing site. When two extrastimuli were used, the coupling interval was then the interval from the ventricular tachycardia complex before stimulation to the second stimulated complex.
TABLE 1. Resetting Characteristics of the Ventricular Tachycardia

<table>
<thead>
<tr>
<th>VT No.</th>
<th>Underlying heart disease</th>
<th>VT morphology</th>
<th>Resetting response pattern</th>
<th>VTCL (msec)</th>
<th>Range of CIs of second extrastimuli*</th>
<th>Shortest CI normalized for VTCL (%)</th>
<th>Slope</th>
<th>Termination</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>CAD</td>
<td>LB LS</td>
<td>F+I</td>
<td>260</td>
<td>400–360</td>
<td>69.2</td>
<td>-0.38</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>CAD</td>
<td>LB LS</td>
<td>F+I</td>
<td>330</td>
<td>505–470</td>
<td>71.2</td>
<td>-0.55</td>
<td>No</td>
</tr>
<tr>
<td>3</td>
<td>CAD</td>
<td>LB RS</td>
<td>I</td>
<td>315</td>
<td>560–500</td>
<td>79.4</td>
<td>-0.59</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>CAD</td>
<td>RB RI</td>
<td>F+I</td>
<td>400</td>
<td>660–600</td>
<td>75.0</td>
<td>-0.70</td>
<td>No</td>
</tr>
<tr>
<td>5</td>
<td>CAD</td>
<td>LB LI</td>
<td>F+I</td>
<td>350</td>
<td>580–530</td>
<td>75.7</td>
<td>-0.62</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>CAD</td>
<td>RB LS</td>
<td>I</td>
<td>350</td>
<td>500–470</td>
<td>67.1</td>
<td>-0.70</td>
<td>No</td>
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<tr>
<td>7</td>
<td>Repaired ToF</td>
<td>RB RI</td>
<td>I</td>
<td>270</td>
<td>355–310</td>
<td>57.4</td>
<td>-0.67</td>
<td>No</td>
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<tr>
<td>8</td>
<td>CAD</td>
<td>RB RI</td>
<td>F+I</td>
<td>360</td>
<td>560–485</td>
<td>67.4</td>
<td>-1.04</td>
<td>No</td>
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<tr>
<td>9</td>
<td>CAD</td>
<td>LB LS</td>
<td>F+I</td>
<td>370</td>
<td>560–520</td>
<td>70.3</td>
<td>-0.50</td>
<td>No</td>
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<tr>
<td>10</td>
<td>CAD</td>
<td>LB LS</td>
<td>I</td>
<td>300</td>
<td>550–475</td>
<td>79.2</td>
<td>-0.30</td>
<td>No</td>
</tr>
<tr>
<td>11</td>
<td>CAD</td>
<td>RB RI</td>
<td>I</td>
<td>420</td>
<td>750–650</td>
<td>77.4</td>
<td>-1.14</td>
<td>Yes</td>
</tr>
<tr>
<td>12</td>
<td>CAD</td>
<td>RB RI</td>
<td>I</td>
<td>340</td>
<td>580–460</td>
<td>67.7</td>
<td>-0.88</td>
<td>Yes</td>
</tr>
<tr>
<td>13</td>
<td>CAD</td>
<td>RB RS</td>
<td>F+I</td>
<td>295</td>
<td>455–405</td>
<td>68.6</td>
<td>-0.66</td>
<td>Yes</td>
</tr>
<tr>
<td>14</td>
<td>CAD</td>
<td>LB LI</td>
<td>F+I</td>
<td>360</td>
<td>545–495</td>
<td>68.8</td>
<td>-0.80</td>
<td>Yes</td>
</tr>
<tr>
<td>15</td>
<td>CAD</td>
<td>LB LS</td>
<td>F+I</td>
<td>440</td>
<td>730–660</td>
<td>75.0</td>
<td>-0.81</td>
<td>Yes</td>
</tr>
<tr>
<td>16</td>
<td>CAD</td>
<td>RB LI</td>
<td>I</td>
<td>350</td>
<td>585–515</td>
<td>73.6</td>
<td>-0.85</td>
<td>Yes</td>
</tr>
<tr>
<td>17</td>
<td>CAD</td>
<td>RB LS</td>
<td>I</td>
<td>380</td>
<td>600–530</td>
<td>69.7</td>
<td>-0.79</td>
<td>Yes</td>
</tr>
</tbody>
</table>

*Range of coupling intervals that resulted in reset with an increasing curve.

The coupling interval normalized for tachycardia cycle length. For a single extrastimulus, the normalized coupling interval was the coupling interval of the stimulated complex divided by the tachycardia cycle length. When two extrastimuli were used, the coupling interval was divided by twofold the tachycardia cycle length because the first extrastimulus does not interact with the tachycardia circuit.

Return cycle. The return cycle was the interval from the last stimulated complex to the first nonpaced complex.

Resetting. Resetting of ventricular tachycardia in response to single or double ventricular extrastimuli was defined as having occurred if 1) the return cycle was less than fully compensatory by 20 msec or more and 2) the same ventricular tachycardia morphology and cycle length resumed after the extrastimuli were delivered.

Resetting response pattern. The resetting response pattern was defined by the set of return cycles that occurred over the range of extrastimuli coupling intervals attempted. The resetting response pattern was defined as flat if there was less than a 10-msec change in return cycles over a 30-msec decrement in extrastimuli coupling intervals. The response was defined as increasing if there was a change in return cycles greater than or equal to 10-msec over a 30-msec decrement in extrastimuli coupling intervals.

Slope of resetting response curve. The slope of the increasing portion of the resetting response curve (or the incremental change in the return cycle over a range of coupling intervals) was determined by linear regression analysis. The slopes were compared by using a Mann-Whitney U test.

Results

Characteristics of Ventricular Tachycardia

Seventeen morphologically distinct ventricular tachycardias that occurred in 14 patients were reset with double extrastimuli and displayed an increasing portion in their resetting response curve. Seven ventricular tachycardias terminated with the introduction of a second ventricular extrastimulus, whereas in the remaining 10, the ventricular effective refractory period at the pacing site was encountered before tachycardia termination. These latter 10 ventricular tachycardias were ultimately terminated by another modality, most commonly burst ventricular pacing. Eight (47%) of the ventricular tachycardias had a left bundle branch block morphology, whereas nine (53%) had a right bundle branch block morphology. A right bundle branch block morphology was seen in five ventricular tachycardias that terminated and four that did not terminate in response to extrastimuli. The mean cycle length of the tachycardias was 346 msec and was in the range of 260–440 msec (Table 1). The mean cycle length of those ventricular tachycardias that terminated was 369±46 msec, whereas those that did not terminate had a mean cycle length of 330±46 msec (p=NS). Shorter but not significantly different coupling intervals were achieved at
the pacing site in those tachycardias that did not terminate compared with those that did terminate. When normalized for tachycardia cycle length, the shortest coupling intervals were identical in both groups (Table 2).

**Characteristics of the Resetting Response Pattern**

The resetting response pattern was evaluated in 17 ventricular tachycardias reset with double extrastimuli. Nine of 17 (53%) had a flat resetting response with extrastimuli delivered at long coupling intervals and an increasing response with more closely coupled extrastimuli, whereas eight (47%) displayed only an increasing response. A fully increasing response pattern was seen in four of the seven tachycardias that subsequently were terminated with double extrastimuli versus four of the 10 that were not terminated.

**Slope of the Resetting Response Curve in Relation to Termination**

The slope of the increasing portion of the resetting response pattern was determined by linear regression analysis. The slopes ranged from −0.30 to −1.14 with a mean of −0.70±0.25. Figure 1 shows an example of the response curve for a ventricular tachycardia that was not terminated by extrastimuli. The ventricular effective refractory period was encountered at the pacing site before causing tachycardia termination.

In Figure 2, the resetting curve of a tachycardia that terminated in response to double ventricular extrastimuli is shown. The mean slope of the ventricular tachycardias that were terminated in response to the extrastimuli was significantly steeper than in those ventricular tachycardias that were not terminated, −0.85±0.15 versus −0.61±0.21, respectively (p<0.025). As shown in Figure 3, six of seven (86%) of the ventricular tachycardias terminated by double ventricular extrastimuli had a slope steeper than −0.75, whereas only one of 10 (10%) of the ventricular tachycardias that were not terminated exceeded this value.

The prematurity with which the second extrastimulus was delivered was alone not responsible for tachycardia termination. There was no statistically significant difference in the prematurity with which the second extrastimulus was delivered between the group of tachycardias that did and did not terminate (531±94 msec vs. 472±83 msec, respectively). When the shortest coupling interval achieved with the second extrastimulus was normalized for the tachycardia cycle length, there was still no significant difference between those tachycardias that terminated and those that did not (72±4% vs. 71±1%, respectively, p=NS).

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**Table 2. Effects of Cyclic Length and Extrastimulus Prematurity on Tachycardia Termination**

<table>
<thead>
<tr>
<th>Tachycardia cycle length (msec)</th>
<th>Duration of the increasing curve (msec)</th>
<th>Shortest CI achieved (msec)</th>
<th>Shortest CI achieved normalized for VTCL (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Termination</td>
<td>369±49</td>
<td>66±26</td>
<td>532±96</td>
</tr>
<tr>
<td>No termination</td>
<td>330±44*</td>
<td>51±16*</td>
<td>478±87*</td>
</tr>
</tbody>
</table>

CI, coupling interval; VTCL, ventricular tachycardia cycle length.

*p=NS compared with termination.

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**Figure 1.** Graphic plotting showing ventricular tachycardia reset but not terminated with double ventricular extrastimuli. The coupling interval of the second extrastimulus is displayed on the abscissa and the resultant return cycle on the ordinate. Note that with progressively premature extrastimuli, conduction delay occurs within the tachycardia circuit. This is demonstrated by the progressive prolongation of the return cycle in response to a decrement in extrastimulus coupling interval. Ventricular refractoriness, however, was reached at the pacing site without prior termination of the ventricular tachycardia. The slope of the resetting response curve was −0.59. The correlation coefficient was 0.95.

**Figure 2.** Graphic plotting showing resetting response curve in a ventricular tachycardia that was terminated by double ventricular extrastimuli. With increasing prematurity of the second extrastimulus, there is significant decremental conduction within the tachycardia circuit as demonstrated by progressively longer return cycles. This tachycardia terminated in response to a premature extrastimulus. The slope of this resetting response curve was −0.88. The correlation coefficient was 0.99.
Discussion

The mechanism of ventricular tachycardia occurring in association with a remote myocardial infarction is believed to be due to reentry.1-5,6 The reentrant circuit that provides the substrate for the ventricular tachycardia may theoretically be of several forms. At one extreme, reentrant tachycardias have been shown to exist in which the leading edge of the propagating tachycardia wave front is constantly impinging on tissue that has just recovered excitability.8 In this model of reentry, both the size (or wavelength) of the tachycardia circuit and its associated cycle length are critically dependent on the refractoriness of tissue supporting the circuit. Because the leading edge of the tachycardia wave front is always impinging on the "tail of refractoriness" of the preceding impulse, no excitable gap should exist within the tachycardia circuit. A premature stimulus, particularly one from a distant site, should not be able to interact with the reentrant circuit and advance the tachycardia.

At the other extreme, reentrant tachycardias have also been shown to occur around fixed anatomical barriers such as the isolated mitral and aortic valve rings.9 In such a circuit, the tachycardia cycle length is primarily dependent on the path length of the propagating wave front. Conduction velocity of the fully repolarized tissue and not refractoriness should be the major determinant of the tachycardia cycle length when the leading edge of the propagating wave front impinges on only fully excitable tissue within the circuit. Because premature stimuli have been shown to interact with and advance the tachycardia without decrement, a fully excitable gap should exist between the leading edge of the propagating wave front and tissue that remains inexitable as a result of the previous tachycardia impulse. The atrial ring model of reentry10,11 provides features of both extremes. The reentrant impulse in this model propagates around a fixed anatomical barrier, the tricuspid ring. During the tachycardia, however, there is incomplete recovery of excitability, and therefore, interval-dependent conduction changes can be observed. The cycle length of these tachycardias should be dependent on both the path length as defined by the anatomical barrier and the refractory state of the tissue supporting the reentrant impulse.10,11

Ventricular extrastimuli introduced during clinical sustained uniform ventricular tachycardia frequently results in the advancement of the tachycardia. This suggests that in those tachycardias due to reentry, an excitable gap exists within the tachycardia circuit.12 When extrastimuli are delivered over a range of coupling intervals, the response pattern can elucidate the excitable state of tissue within the reentrant circuit. A flat resetting response pattern is felt to represent engagement of the stimulated wave front with only fully excitable (or fully recovered) tissue within the tachycardia circuit.5 Therefore, as the programmed extrastimulus is delivered with increasing prematurity, its propagating wave front does not encounter tissue within the reentrant circuit that exhibits interval-dependent conduction slowing. If, on the other hand, tissue encountered by the stimulated impulse exhibits interval-dependent conduction slowing, an increasing resetting response pattern will be seen. An initial flat response at long stimulus-coupling intervals followed by an increasing curve as the extrastimulus is delivered with increasing prematurity may reflect initial engagement of fully excitable tissue followed by impingement on tissue within the reentrant circuit, which is in a relative refractory state as the extrastimulus is delivered with increasing prematurity.

The slope of the increasing portion of the resetting response curve, determined by linear regression analysis, quantifies the degree of impingement on partially excitable tissue within the reentrant circuit. There is no a priori reason why the increasing resetting response curve should be linear and not expressed more accurately as an exponential function or a higher order polynomial function. Other investigators, however, have found similar linear input-output relations while stimulating the ventricles under various circumstances.13,14 The increasing curve seen during resetting of clinical ventricular tachycardia appears to be fit by linear function with good correlation coefficients (mean, 0.95; range, 0.83-1.0).

The slopes of the increasing portion of the resetting response curves ranged from a shallow -0.30 to a steep -1.14. Those tachycardias that terminated had a significantly steeper slope as a group than those that did not terminate. Tachycardias that did and did
not terminate were comparable in terms of the closest coupled extrastimulus achieved and the prematurety of the closest coupled extrastimulus normalized for tachycardia cycle length. Those tachycardias that terminated had a slightly longer cycle length than those tachycardias that did not terminate. This difference was not statistically significant; however, the total number of tachycardias evaluated was relatively small. An additional factor that may be important for tachycardia termination is the proximity of the stimulation site to the reentrant circuit. Extra-stimuli delivered close to the reentrant circuit should engage the propagating wave front within the tachycardia circuit earlier than extrastimuli delivered at the same coupling interval relative to the onset of the surface electrocardiogram but at a site more distant from the tachycardia circuit. Unfortunately, the “site of origin” was not identified at the time that resetting was performed in these tachycardias. Therefore, an estimation of the distance from the pacing site to the tachycardia circuit was not available.

The ability of an extrastimulus to terminate a tachycardia when delivered at a shorter coupling interval than an extrastimulus that resulted in resetting is likely the result of bidirectional block within the tachycardia circuit. As the stimulated wave front enters the tachycardia circuit and travels in both directions, there will be collision of the antidromic wave front with the preexisting tachycardia impulse (antidromic collision), whereas at the same time, the orthodromic stimulated wave front will encounter fully refractory tissue or tissue unable to produce a propagating response (orthodromic block).

The slope of the increasing portion of the resetting response curve delineated a group of ventricular tachycardias that were more likely to terminate in response to double ventricular extrastimuli. It is quite possible that the site of the interval-dependent conduction slowing and site of propagation failure during tachycardia termination are the same. This may be suggested by information from spontaneous terminations in the atrial ring model of reentry where all portions of the reentrant circuit can be simultaneously recorded. If this hypothesis is correct, then the present data appear to imply that tissue exhibiting greater magnitude of interval-dependent conduction slowing may have relatively longer effective refractory periods. It is quite possible, however, that multiple levels of potential conduction delay and block may be present within the tachycardia circuit, analogous to conduction over the normal atrioventricular conduction system, given the pronounced degree of heterogeneity present in peri-infarct scarring. In the latter circumstance, the site of interval-dependent conduction slowing and the site of block during termination of the tachycardia may be different. It appears unlikely that the site of interval-dependent conduction slowing is encountered before the site of block by premature impulses. Should the area of decremental conduction be encountered by the stimulated wave front before the site of block, progressive slowing should prevent the stimulated wave front from arriving at the site of propagation failure with sufficient prematurity to impinge on its effective refractory period. Because direct recordings were not obtained from within the tachycardia circuit and more specifically at the area or areas exhibiting interval-dependent conduction delay, however, the coupling interval achieved at the site of propagation failure and of tachycardia termination is unknown.

Consideration was given to the possibility of normalizing the slope for the tachycardia cycle length. This was not performed, however, because this approach would imply that the interval-dependent conduction slowing was occurring uniformly throughout the reentrant circuit. Clinical and experimental studies have suggested that nonuniform anisotropy results in inhomogeneity of conduction of the propagating impulse within the circuit. Therefore, it is likely that only one or a few well-localized regions within the tachycardia circuit may be the site or sites of pronounced interval-dependent conduction slowing. Documentation of the site of impulse slowing during resetting and the effective refractory period at this site would require high density intraoperative mapping. Nevertheless, the resetting response curve does provide valuable information about the properties of tissue within the reentrant circuit. Because the increasing resetting response curve is believed to represent impingement on partially excitable tissue, the slope of this increasing portion may be an index of relative excitability or refractoriness of tissue within the reentrant circuit.

Limitations

Only those tachycardias that were hemodynamically tolerated, uniform in morphology and rate, and reset with an increasing curve were included. Therefore, extrapolations to more rapid hemodynamically nontolerated tachycardias, tachycardias with irregular periodicity, and tachycardias not demonstrating an increasing curve with double ventricular extrastimuli cannot be performed. The strict stability and morphology criteria for tachycardia inclusion were used in an attempt to demonstrate the path of impulse propagation and exit from the tachycardia circuit were unaltered by the premature impulse. Again, however, documentation and verification that the premature impulse traversed the identical circuit that was used by the native tachycardia wave front will require high density intraoperative mapping.

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**KEY WORDS** • ventricular tachycardia • resetting • reentry
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