Localization of the Slow Conduction Zone During Reentrant Ventricular Tachycardia

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**Background**—Reentrant ventricular tachycardia is sometimes difficult to treat effectively because localizing the slow conduction zone (SCZ) for catheter ablation may be problematic. It was hypothesized that a linear relationship exists between activating wave-front acceleration and deceleration in the SCZ and, respectively, contractions and expansions of the far-field extracellular signal, which could be used for SCZ localization.

**Methods and Results**—To test the hypothesis, a model was developed to approximate SCZ location on the basis of the time interval between activation at the recording site and shifts in electrogram far-field deflections. Electrograms were recorded during reentry from 196 to 312 epicardial sites (canine model, 8 episodes). Activation maps of reentry were constructed to determine wave-front velocity, and piecewise linear adaptive template matching (PLATM) measured time shifts in far-field electrogram deflections. Linear trends of cycle length change often occurred during tachycardia (mean trend, $+15$ ms/96.8 cardiac cycles; $r^2=0.92$). Alteration in the time interval for activation through the SCZ approximated the change in tachycardia cycle length (mean correspondence, 75.7%). The beginning and end times of far-field extracellular waveform time shifts measured by PLATM predicted the time from recording site activation to activation at the SCZ proximal and distal edges, respectively (mean absolute error with respect to activation mapping, 20.3 ms).

**Conclusions**—During reentry, PLATM estimates the time interval from activation at any recording site near the circuit to SCZ activation. PLATM time intervals are convertible to arc lengths along the circuit for potentially more rapid and accurate update of a hand-held probe toward the SCZ for catheter ablation. (Circulation. 2000;102:464-469.)

**Key Words:** ablation ■ reentry ■ tachycardia

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**Ventricular tachycardia (VT)** is an important health concern that can be life threatening. There is abundant evidence that many VTs occurring in humans are caused by reentry.1–5 Initiation of a reentrant circuit requires a slow conduction zone (SCZ) and a zone of unidirectional block. In large circuits such as those arising from infarct scars, areas of slow conduction in and around the scar should be targeted for ablation.2 Clinical studies suggest that targeting this region will interrupt the circuit and stop tachycardia without recurrence.1,4,5 In a canine study, application of cryothermy terminated reentrant VT only when the circuit was interrupted within the central isthmus,6 where the SCZ is located.

An effective method to determine arrhythmogenic areas for ablation is to isolate sites that, when paced during tachycardia, exhibit entrainment with concealed fusion.3 Such sites are believed to reside within the reentrant circuit isthmus, which is bounded by lines of anatomic or functional block. However, ablation will fail if lesions are too small or too far away from the reentry pathway to completely interrupt the circuit.7 Reentrant tract geometry can be complex and extensive with multiple entrances and exits,8 so it is sometimes difficult and time consuming, and occasionally impossible, to locate all arrhythmogenic sites.3,7,8 Entrainment with concealed fusion also occurs by pacing from bystander pathways.3 At a bystander pathway, activation proceeds from the main circuit loop but is constrained by block lines having the shape of a cul-de-sac; ablation there does not terminate reentry.3 Site-by-site activation mapping also identifies arrhythmogenic sites for ablation, but besides the fact that it is tedious and time consuming to time synchronize data and create isochrone maps, this process is subjective when multiple electrogram deflections are present.9 Basket-type catheters in which electrograms are recorded simultaneously from many sites are also used for mapping; however, good electrode contact at all sites on the endocardium is difficult to ensure because of irregularities in the ventricular surface, so areas crucial to reentry may not be recorded.10 Basket catheters also have limited torque capabilities, which hampers correct placement, and they may abrade the endocardium.10

Quantitative analysis of dynamic, cycle-by-cycle changes in electrogram shape is a promising new tool for localizing reentry features, including arcs of block bounding the isthmus, as has been demonstrated in a canine model of reentrant VT with a figure-8 pattern of conduction.11 From previous
observations of changing conduction in the SCZ,\textsuperscript{11} it was hypothesized that a linear relationship exists between activating wave-front acceleration and deceleration in the SCZ and, respectively, contractions and expansions of the far-field extracellular signal, which could be used for SCZ localization. Because similar trends in cycle length change have also been observed in human VT,\textsuperscript{12} an approach based on this hypothesis, described herein, is of potential relevance to clinical ablation therapy.

## Methods

### Data Collection and Activation Mapping

A canine model of VT caused by reentrant excitation that has been described elsewhere\textsuperscript{13} was used for creation of activation maps and measurement of cycle length. Bipolar electrograms from 196 to 312 sites on the epicardial border zone in the anterior left ventricle of the heart are digitized at 1 to 2 kHz and recorded simultaneously.\textsuperscript{11,13} For each of 8 reentry episodes (7 experiments), cycle length was computed from onset to termination on the basis of the ECG R-R interval and plotted with respect to cycle number. When a trend in cycle length, defined as a general tendency for linear cycle length increase or general tendency for linear decrease, occurred over \( \geq 50 \) cycles, 2 of the cycles 25 and 50 cycles apart were selected for further analysis. Activation maps of these cycles were constructed to determine the time interval for the activating wave front to propagate from the proximal to the distal edge of the SCZ and the temporal relationship between activation at each recording site and SCZ activation for comparison with electrogram shape analysis measurements described below.

### Time-to-Distance Model

A simple model was used to approximate electrogram deflection alterations occurring when conduction velocity changes over an arc of the circuit (Figure 1A). Let the SCZ, denoted by the shaded region in the isthmus, cover a 30-mm arc. The remaining arc, shown for the right-hand loop, is 150 mm in length (180-mm total length of the loop). Let conduction velocity be 0.5 mm/ms in the SCZ and 1.0 mm/ms elsewhere during cycle k of tachycardia. The time for the activating wave front to propagate from the SCZ proximal to distal edge is \( 30 \text{ mm}/(0.5 \text{ mm/ms}) = 60 \text{ ms} \) and from the distal to proximal edge is \( 150 \text{ mm}/(1.0 \text{ mm/ms}) = 150 \text{ ms} \). The tachycardia cycle length is therefore \( 60 + 150 = 210 \text{ ms} \). For an arbitrary site three fourths of the way along the arc of the SCZ referenced at 0 ms (solid black circle in Figure 1A), the time between activation at the site and activation at the SCZ proximal edge is \(-45 \text{ ms}\) and to activation at the distal edge is \(+15 \text{ ms}\). Now suppose that conduction velocity decreases to 0.3 mm/ms in the SCZ and remains at 1.0 mm/ms elsewhere during cycle \( k + n \) of tachycardia (Figure 1B). Then the new time for the wave front to traverse the SCZ is \( 30 \text{ mm}/(0.3 \text{ mm/ms}) = 100 \text{ ms} \), and the cycle length is \( 100 + 150 = 250 \text{ ms} \). For the arbitrary site referenced at 0 ms (solid black circle in Figure 1B), the time between activation at the site and activation at the SCZ proximal and distal edges is now \(-75 \text{ ms}\) and \(+25 \text{ ms}\), respectively.

As a first approximation, the only change at the source that generates the electrogram deflections during SCZ activation, from cycle \( k \) to \( k + n \), can be considered to be a deceleration of the process by \( 100 \text{ ms}/60 \text{ ms} = 1.67 \). Then the electrogram deflections caused by SCZ activation during cycle \( k + n \) will appear exactly the same as during cycle \( k \), except that they will be expanded by 1.67 along the time axis. This is illustrated in Figure 1C for the arbitrary site in Figure 1A and 1B. The 2 electrograms (cycle \( k \), thick trace, and cycle \( k + n \), thin trace) are time aligned at the point of activation at the recording site, which is taken as the extremum point (negative electrogram peak at time 0 ms denoted by solid vertical line). The time interval for activation of the SCZ is shown by thick vertical dotted lines (cycle \( k \)) and by thin vertical dotted lines (cycle \( k + n \)). The decreased conduction velocity in the SCZ, which increases the time for the activating impulse to traverse the region by 40 ms, causes the electrogram of cycle \( k + n \) (thin trace) to expand by 40 ms with respect to the electrogram of cycle \( k \) (thick trace). The phase lag or shift resulting from expansion, defined as the relative position in time of corresponding electrogram deflections of cycle \( k \) versus cycle \( k + n \), changes by \(-30 \text{ ms} \) from activation at the recording site (solid vertical line; Figure 1C) to activation at the SCZ proximal edge (left dotted lines) and changes by \(+10 \text{ ms} \) from activation at the recording site to activation at the SCZ distal edge (right dotted lines; Figure 1C). Outside the SCZ activation interval, no further change in phase lag occurs; it remains \(-30 \text{ ms} \) at times before SCZ activation and \(+10 \text{ ms} \) at times after SCZ activation (see also phase lag trace in Figure 1C, top). At any recording site in the circuit, electrogram far-field deflections occurring during the SCZ activation interval are affected in the same way as the arbitrary site of Figure 1. However, the time interval from activation at the recording site to the point where phase lag changes will differ at each site, depending on its distance along the loop of the circuit with respect to the SCZ.
mapping. However, the number printed at each recording site was not the activation time but rather the PLATM-estimated time interval from activation at the site to activation at the SCZ. Separate maps were created to show activation time intervals from recording sites to the SCZ proximal edge and to the distal edge. Also, in separate tests, electrograms from template/input cardiac cycles separated by 25 and 50 cycles were used for PLATM analysis. The mean error of PLATM estimates with respect to activation mapping was tabulated.

Results

Relationship of Tachycardia Cycle Length to the SCZ

In Figure 2, cycle length change with respect to cycle number is given for 2 monomorphic VT episodes. In each episode, there was an approximately linear trend in cycle length over many cardiac cycles. In episode 1A (pace termination), there were alternating upward and downward trends in cycle length (labeled i through iv), whereas in episode 2 (spontaneous termination), the only trend in cycle length was upward. During a given trend, 1- to 5-ms oscillations occurred. These examples were typical of trends of cycle length change in the other reentry episodes; cycle length tended to prolong (mean trend, +15 ms/96.8 cardiac cycles; \( r^2 = 0.92 \)). Cycle length change was caused mostly by alterations in SCZ conduction velocity as is shown in Figure 3 for reentry episode 1A. In Figure 3A, the activation map is shown for cycle 61. The isthmus is located near the map center, and arrows denote wave-front direction. In Figure 3B through 3D, electrograms from selected sites in the isthmus, circled in Figure 3A, are shown for cycles 11 and 12, 61 and 62, and 111 and 112, respectively. The SCZ is considered to extend between sites 58 and 54, where slow conduction occurs. There is an approximate correspondence of cycle length disparity between cycles 11 and 61 (20 ms, Figure 3B and 3C) and between cycles 61 and 111 (−12 ms, Figure 3C and 3D), with the change in activation time through the SCZ and, more precisely, through isthmus sites 58 to 52 (104−82−22 ms and 87−104 = −17 ms, respectively). During all trends, an approximate correspondence existed between cycle length disparity and alteration in SCZ activation interval.

Changes in Electrogram Deflections

In Figure 2A (episode 1A), cycle length increases gradually over \( \approx 50 \) cardiac cycles and then decreases gradually over \( \approx 50 \) cycles. Changes in electrogram deflections occurring...
during these gradual trends in cycle length are shown in Figure 4 for 2 representative electrograms (site 55 in Figure 4A and 4C and site 74 in Figure 4B and 4D). Site 55 was located in the SCZ, and site 74 was near the isthmus entrance (Figure 3A). Figure 4A shows overlapped electrograms extracted from 50 cardiac cycles of the site 55 recording, aligned with respect to the extremum point (cycles 11 to 60 when cycle length prolonged; see Figure 2A). The electrograms of the first 12 cardiac cycles are red; the next 12 to 13 are yellow and then green and blue. The electrograms of Figure 4A expand over 50 cycles in approximately the same way as in the model (Figure 1C). The 50 electrograms extracted from cycles 11 to 60 of site 74 (Figure 4B) show similar electrogram expansions; however, expansions begin about 20 ms after activation at the recording site, in accordance with the position in the circuit of site 74 with respect to the SCZ. In Figure 4A and 4B, the total change in phase lag between the deflections is ≈20 ms, in conformity with cycle length change over the interval. Figure 4C and 4D shows 50 electrograms of sites 55 and 74, respectively, extracted from cycles 61 to 110 of episode 1A (Figure 2A). The time intervals and extent of phase lag changes between deflections are similar to those in Figure 4A and 4B but are reversed in direction because cycle length decreased by ≈12 ms from cycle 61 to 110 and therefore SCZ conduction velocity increased in approximate proportion. These gradual cycle-to-cycle changes in far-field electrogram deflections, of approximately linear proportion to the change in SCZ conduction velocity and change in tachycardia cycle length, were typical of those at all other sites in the same and in other episodes of tachycardia.

**PLATM Maps of SCZ Boundaries**

PLATM maps of time intervals from activation at each of 196 recording sites to activation of SCZ. Top, Zero line is SCZ proximal edge; Bottom, zero line is SCZ distal edge. LAD indicates left anterior descending coronary artery; LL, left lateral margin.

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Figure 5. PLATM maps (episode 2) of time interval from activation at each of 196 recording sites to activation of SCZ. Top, Zero line is SCZ proximal edge; Bottom, zero line is SCZ distal edge. LAD indicates left anterior descending coronary artery; LL, left lateral margin.
between PLATM time intervals and corresponding activation map intervals was 20.3±1.6 ms over all sites for the 10 trends. PLATM measurements were not manually corrected for errors.

**Discussion**

**Possible Clinical Method to Determine Optimal Ablation Sites**

Limitations in existing clinical methods to determine optimal ablation sites led us to consider the potential of beat-to-beat electrogram measurements for identifying reentrant circuit features because circuits are often dynamic, not static, entities, with variation in the reentrant path and conduction velocity, both of which cause alterations in electrogram shape. Unlike activation mapping, PLATM measurement of electrograms at a single recording site suffices to determine the time interval from activation at that site to activation of the SCZ. Unlike entrainment methods, bystander areas are unlikely to significantly affect isthmus and SCZ localization because they did not show dramatic conduction velocity changes in these experiments and therefore would not be expected to significantly influence changes in far-field deflections. Furthermore, no complex instrumentation is needed to adapt clinical mapping systems to include PLATM analysis. PLATM might therefore be useful as an adjunct to existing clinical entrainment and mapping procedures to more rapidly and accurately “home in” on the best site to ablate.

**Model of Conduction Velocity Change**

The model described in Figure 1 was used to estimate alterations in electrogram deflections caused by SCZ conduction velocity changes. Although conduction velocity changes during reentry in the same canine model have been reported to occur in other areas of the circuit such as at wave-front pivot points, our observations suggest that the bulk of the alteration in conduction velocity occurs within the SCZ. Conduction velocity and changes in conduction velocity were approximated as being uniform throughout the SCZ. However, real conduction velocity varies, as in Figure 3B through 3D, in which the activation time intervals between sites in the SCZ are not uniform although the sites are approximately equidistant (5-mm spacing). The lowest conduction velocity and the largest conduction velocity change over many cycles occur toward the SCZ center between sites 55 and 56. Increased conduction velocity and smaller changes between cycles occur toward the SCZ proximal and distal edges. This pattern, observed in all tachycardia episodes of this study, may be related to isthmus width and shape as suggested by a theoretical model and an experimental study. An improved model of SCZ conduction velocity change would include these factors.

When SCZ conduction velocity decreased, a concomitant increase in conduction velocity was often observed in the region just distal to its distal edge toward the exit of the isthmus; note the decrease in the activation interval between sites 54 and 52 from 16 ms (Figure 3B) to 8 ms (Figure 3C). This region can be considered a rapid conduction zone (RCZ) with properties opposite to the SCZ.
Acceleration of the wave front in the RCZ when cycle length prolongs, which may be due to increased time for recovery there, results in advance rather than delay of phase lag between electrogram deflections. This can be observed during time 60 to 120 ms, just after activation of the SCZ distal edge, for the electrograms of site 55, episode 1A (Figure 4A). The electrogram deflections on successive cardiac cycles advance (shift to the left) rather than delay during time 60 to 120 ms. There is deceleration of the wave front in the RCZ when cycle length shortens, and electrogram deflections on successive cardiac cycles delay slightly (shift subtly to the right) during time 60 to 120 ms (Figure 4C). For simplicity, the RCZ was not modeled in this study, but it accounts for much of the discrepancy between cycle length disparity and change in SCZ activation interval. The relationships between conduction velocity and changes in amplitude and baseline level of the extracellular signal, which require further elaboration, also were not modeled.

PLATM and the conduction velocity model of Figure 1 might be useful for determining the arc length along the circuit from recording site to the proximal and distal SCZ edges. If, for example, the PLATM interval from recording site to SCZ proximal edge activation is 20 ms, as at site 74 (Figure 4B and 4D), then arc length to the edge would be estimated as 20 ms x 1 mm/s = 20 mm (approximately correct for site 74 based on 5-mm electrode spacing; see Figure 3A). If a quadrupolar catheter with specialized electrode configuration were available, wave-front velocity (speed and direction) could be automatically calculated with the Sobel operator. The catheter line could then be dragged along the endocardium toward the SCZ, using the angle of the propagating wave front with respect to the recording site as a guide while taking PLATM measurements at regular intervals to gauge SCZ distance. When PLATM measurements showed the probe to reside within the SCZ, then the heart would be ablated.

Influence of Far-Field Potential on the Extracellular Waveform

It was considered that the extracellular signal during 1 cardiac cycle represented primarily events occurring at the activating wave-front leading edge. Large directional changes in the leading edge as it pivoted around obstacles to conduction were observed to correspond in time to peaks in far-field electrogram deflections. Therefore, areas along the circuit loop at which peaks in far-field deflections will be generated include the ends of arcs of block around which the wave front pivots to enter or exit the isthmus, as well as other obstacles to conduction such as spurs protruding from arcs of block, isolated block lines, and the characteristic curvature of the isthmus itself. Bipolar electrodes, used in these recordings and often used clinically to record from the ablation catheter tip, sense far-field activity differently from unipolar electrodes (decreased amplitudes and slopes in bipolar recordings). However, this factor would not be expected to significantly influence PLATM measurements, which are relative, not absolute, and therefore independent of slope and peak absolute magnitudes.

Study Limitations and Future Directions

Erroneous PLATM measurements can result from flawed weight initialization and from noise effects on weight convergence, which are subjects of further research. Because there is an inverse relationship between field strength of the extracellular potential and distance to site of origin, increased voltage resolution and higher signal-to-noise ratio systems may also increase accuracy. Occasional presence of T waves might be distinguished in part from far-field activation by use of the timing constraint imposed by the repolarization process.

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References

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