Characteristics of Wavefront Propagation in Reentrant Circuits Causing Human Ventricular Tachycardia

Anthony W.C. Chow, MRCP; Richard J. Schilling, MD; D. Wyn Davies, MD; Nicholas S. Peters, MD

Background—We investigated the relationship between wavefront curvature and slowing of conduction both within and outside the diastolic pathway of circuits causing ventricular tachycardia (VT) in the infarcted human heart.

Methods and Results—Propagation was determined around the reentrant circuits of 11 VT (cycle length, 348±75 ms) in 8 patients undergoing high-resolution noncontact mapping. The diastolic pathway had a mean wavefront velocity of 0.82±0.49 m/s and occupied 68±7% of VT cycle length. Significant changes (>5 degrees/mm) in trajectory of propagation occurred in 8 diastolic pathway segments (10.1±3 degrees/mm) in which wavefront propagation slowed to 0.41±0.11 m/s compared with the segments immediately preceding (0.91±0.16 m/s, P<0.05) and following (1.07±0.33, P<0.05) the change in trajectory. At the turning points of entry (9.3±3.9 degrees/mm) and exit (9.0±4.8 degrees/mm) of the diastolic pathway propagation, velocity slowed at entry from 1.23±0.4 to 0.6±0.26 ms (P<0.001) and was more rapid at exit turning points (0.8±0.25 m/s) (P<0.05). There was an inverse relationship between wavefront curvature and velocity, both within and outside the diastolic pathway (r=0.46, P=0.0001), and VT cycle length correlated with total curvature multiplied by length of the diastolic pathway (P<0.01).

Conclusions—Slowing of propagation in circuits causing VT in the infarcted human heart occurs over regions of wavefront turning, with an inverse relationship between wavefront curvature and velocity, both within and outside the diastolic pathway. Conduction is slower at entry than exit turning points of the diastolic pathway but is slowest during turns within the diastolic pathway. (Circulation. 2002;105:2172-2178.)

Key Words: tachycardia ■ waves ■ ventricles ■ mapping

Myocardial architecture and the passive and active membrane properties of cardiac myocytes combine to determine the conduction characteristics of propagating wavefronts.1,2 Mapping studies of the infarcted human left ventricle (LV) have confirmed that ventricular tachycardia (VT) is dependent on slowing of conduction through a discrete, largely endocardial diastolic pathway bordered by lines of conduction block;3-7 the precise location and mechanism of conduction slowing within the diastolic pathway are unknown. Slow conduction is necessary to maintain reentry but also occurs in regions prone to conduction block.9 Understanding the mechanisms of slow conduction in human VT will assist in recognition and localization of regions of circuits most prone to conduction block and termination of tachycardia.

Experimental studies indicate that curvature of the propagating wavefront is an important determinant of both the conduction velocity and likelihood of conduction block,10-13 but the role of wavefront curvature remains to be determined in the human heart. In the present study, we used noncontact endocardial mapping of VT to investigate the relationship between wavefront curvature and slowing of conduction both within and outside the diastolic pathway of circuits causing VT in the infarcted human heart.

Methods

Patients

Eight male patients aged 61±5.3 years with structural heart disease and documented stable monomorphic VT undergoing radiofrequency ablation guided by a noncontact mapping system were studied (Table 1). Seven patients had a past history of myocardial infarction, and I had idiopathic dilated cardiomyopathy. All patients had dilated LVs with impaired function (ejection fraction 36±5%) and had recurrent sustained and hemodynamically stable VT inducible by programmed stimulation.

Mapping Procedure

Patients were studied in the postabsorptive state, having given written informed consent, and the study had local ethics committee approval. A 6F quadrupolar catheter was positioned at the right ventricular apex for programed stimulation. Two deflectable 7F mapping/ablation catheters were introduced into the LV by a retrograde transaortic route and via a transeptal puncture. Patients were heparinized, and the noncontact catheter was deployed.

Noncontact Mapping

The technique of noncontact mapping has been described previously.14-16 The noncontact system (Endocardial Solutions Inc) consists of a catheter-mounted 64-wire multielectrode array (MEA) woven around an 8-mL balloon.
The MEA collects raw far-field endocardial potentials that are recorded, amplified, and processed by the system to reconstruct unipolar electrograms,14 the integration of which produces isopotential maps with activation represented as the leading edge of advancing negative potential. All recorded data are stored on optical media and, for the present study, were examined offline.

A locator signal emitted from a roving catheter can define its position in 3-dimensional space relative to the MEA. Using this, an anatomic representation of the LV endocardium is reconstructed.

**Distance and Turning Measurements**

The noncontact system gives the 3-dimensional Cartesian coordinates of endocardial points using the center of the MEA as reference. The distance between 2 endocardial points can be calculated from the following coordinate geometry equation:

$$D = \sqrt{(x_1-x_2)^2+(y_1-y_2)^2+(z_1-z_2)^2}$$

where \(x_1, y_1, z_1, x_2, y_2,\) and \(z_2\) are the Cartesian coordinates of 2 points on the endocardium relative to the center of the MEA and \(D\) is the straight-line distance between the points. The change of angulation between 2-directional vectors can be calculated from Cartesian coordinates using 3-dimensional vector geometry17 using the following equation:

$$\theta = \cos^{-1} \left( \frac{[(x_1-x_3)\times(y_1-y_3)+[(y_1-y_3)\times(z_1-z_3)]...]{(x_1-x_3)^2+(y_1-y_3)^2+(z_1-z_3)^2}}{[(x_2-x_3)^2+(y_2-y_3)^2+(z_2-z_3)^2]} \right)$$

where \(x_1, y_1, z_1, x_2, y_2, z_2, x_3, y_3, z_3\) represent the Cartesian coordinates and direction vector from point 1 to point 2 and \(x_2, y_2, z_2, x_3, y_3, z_3\) are the coordinates and direction vector from point 2 to point 3 (Figure 1). The change in direction between the 2 vectors is expressed as \(\theta\) in radians, and values are converted to degrees of turn for final analysis.

**Definitions**

Diastolic pathways were defined as regions of LV diastolic activation that were protected from systolic activation. Entry and exit turning points of the diastolic pathway were defined as regions with acute change in wavefront trajectory around the ends of the lines of block bordering the diastolic pathway. Between the exit and entry turning points, the systolic portion of the circuit is referred to as the outer pathway.

The pathway taken by the leading edge of the activation wavefront around the lines of block bordering the diastolic pathway was determined for the entire identifiable portion of the circuit using a series of consecutive short straight-line measurements between adjacent points along the line of trajectory of and perpendicular to the advancing wavefront. The change in 3-dimensional trajectory of propagation between 2 consecutive straight-line measurements was calculated using 3-dimensional vector geometry, expressed as degrees of turn per millimeter, and a series of such consecutive straight-line measurements was used to determine turning and velocity change throughout the VT circuit.

A significant turn was defined as a change in direction of propagation greater than the estimated maximum geometric curvature of the LV endocardial surface calculated from the 2-dimensional echocardiographic measurements from each patient based on modified Simpson’s rule18. The conical apical portion (the terminal 0.5 cm of the LV cavity long axis) was excluded from the calculation and proved not to be involved in any of the VT circuits being studied. By this method, the estimated minimum circumference of the short-axis cross-section (360 degrees) was 82 ± 9 mm (range, 71.3 to 94.8 mm), which equates to an estimated maximal possible change in

**Table 1. VT Characteristics of Patients**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, y</th>
<th>IHD</th>
<th>LVEF, %</th>
<th>VT Cycle Length, ms</th>
<th>Proportion of DP Mapped, %</th>
<th>DP/CL, %</th>
<th>Length of DP, cm</th>
<th>Mean DP Velocity, m/s</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>65</td>
<td>No</td>
<td>35</td>
<td>455</td>
<td>100</td>
<td>74</td>
<td>16.9</td>
<td>0.87</td>
</tr>
<tr>
<td>2</td>
<td>60</td>
<td>Yes</td>
<td>35</td>
<td>485</td>
<td>67</td>
<td>0.76</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>64</td>
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<td>40</td>
<td>320</td>
<td>100</td>
<td>68</td>
<td>11.2</td>
<td>0.98</td>
</tr>
<tr>
<td>4</td>
<td>69</td>
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<td>30</td>
<td>310</td>
<td>100</td>
<td>64</td>
<td>8.5</td>
<td>0.96</td>
</tr>
<tr>
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<td>55</td>
<td>Yes</td>
<td>35</td>
<td>338</td>
<td>100</td>
<td>76</td>
<td>1.07</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>64</td>
<td>Yes</td>
<td>30</td>
<td>325</td>
<td>40</td>
<td>1.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>56</td>
<td>Yes</td>
<td>45</td>
<td>425</td>
<td>45</td>
<td>0.71</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>61</td>
<td>Yes</td>
<td>38</td>
<td>286</td>
<td>100</td>
<td>48</td>
<td>6.9</td>
<td>0.78</td>
</tr>
</tbody>
</table>

IHD indicates ischemic heart disease; LVEF, left ventricular ejection fraction; DP, diastolic pathway; and CL, VT cycle length. The mean DP wavefront velocity is an average of all velocities measured within the DP for each VT.
trajectory of 4.4 degrees/mm for simple planar propagation. Therefore, in the present study, we defined a significant change in the trajectory of a turning wavefront in a VT circuit as a region with ≥5 degrees/mm of turn.

Statistical Analysis
Data are expressed as mean±SD. Statistical analysis of continuous variables was by the Wilcoxon paired test. Spearman’s rank coefficient was used to calculate correlation. A value of \( P<0.05 \) was considered significant.

Results
In 8 patients, 11 sustained monomorphic VTs (cycle length, 348±75 ms) were induced and mapped. Of these, the entire circuit was identified in 7 VTs, and continuous activation could be identified over 57±17% of the cycle length in the remainder (Table 1). The VTs that were fully defined had a diastolic path length of 9.8±3.8 cm (range, 4.7 to 16.9 cm) and accounted for 68% of the total VT cycle length (range, 48% to 80%). The mean distance between adjacent endocardial points measured was 4.6±1.8 mm (range, 1.3 to 8.9 mm). Wavefront velocity within the diastolic pathway showed considerable variation (Figure 2). Mean diastolic pathway velocity varied between patients from 0.38 to 1.07 m/s (mean, 0.82±0.2 m/s) (Table 1). This was significantly different when compared with the outer pathway wavefront velocity of 1.3±0.28 m/s (\( P<0.001 \)).

Wavefront Turning Within the Diastolic Pathway
Wavefront turning within the diastolic pathway during VT is illustrated by a sequence of isopotential maps in Figure 3. Reconstructed electrograms corresponding to points A through H on the virtual endocardium are shown in Figure 4. The virtual endocardium has been cut along the posterior wall and laid open, and points B through E are equally spaced along the length of the diastolic pathway. The green arrows represent the direction of wavefront activation. Resting endocardial potential is seen as purple that changes to white on activation. Frame 1 shows the end of systole before activation enters the diastolic pathway. Diastolic activation then propagates in a lateral direction (frame 2), with low-amplitude diastolic potentials seen on the electrograms. Significant slowing of wavefront velocity at the point of turning within the diastolic pathway (frame 3) corresponds with the longest delay seen between electrograms C and D. Activation then propagates in a basal direction to the end of the diastolic pathway (frame 4), breaking out as systolic activation (frame 6) with clockwise and anticlockwise wavefronts to complete a figure-8 reentrant circuit (frames 7 and 8).

Wavefront velocities along the course of 4 different diastolic pathways in 3 patients are shown with the corresponding endocardial locations (right inset). Refer to text for details.
associated with significant slowing of wavefront propagation to 0.41±0.11 m/s compared with 0.91±0.16 m/s in the region before \( P<0.05 \) and 1.07±0.33 m/s after \( P<0.05 \) the change in trajectory (Table 2). In regions of the diastolic pathways without significant (<5 degrees/mm) angulations, wavefront velocity was 0.96±0.52 m/s.

**Turning at Entry and Exit TP**

Of 44 entry and exit turning points in 11 circuits, detailed activation of 20 entry turning points and 21 exit turning points was identified and mapped. The rate of turn at entry was 9.3±3.9 degrees/mm. Wavefront velocity significantly slowed from 1.23±0.4 m/s in the outer pathway to 0.6±0.26 m/s \( P<0.001 \) at entry turning points and remained unchanged (0.53±0.28 m/s) on entering the most proximal diastolic pathway. The rate of turn at exit from the distal diastolic pathway was 9±4.8 degree/mm, during which wavefront velocity (0.8±0.25 m/s) was unchanged from the immediately preceding portion of diastolic pathway (0.77±0.28 m/s) \( P=0.31 \). On leaving the region of the exit turning point, wavefront velocity increased to 1.6±0.27 ms \( P=0.0001 \).

When the wavefront velocities of all regions of the circuit with significant turn (>5 degrees/mm) were compared, entry

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**Figure 3.** Sequence of isopotential maps during VT with a turn within the diastolic pathway. Sept indicates LV septum; Post, posterior wall; Lat, lateral LV wall; and Apex, LV apex. Refer to text for details.

**Figure 4.** Reconstructed electrograms corresponding to endocardial points A through H in Figure 3. Diastolic potentials are shown by the green arrows and systolic activation by blue arrows. Refer to text for details.
turning point wavefront velocity (0.6±0.26 m/s) was slower than exit turning point (0.8±0.25 m/s) (P<0.01), but slowest velocity occurred during turning within the diastolic pathway (0.41±0.1 m/s) (P<0.05 versus entry or exit turn) despite similar rates of turning in all 3 regions (9.3±4, 9±4.8, and 10.1±2.9, degrees/mm respectively) (P=0.57).

**Correlating Propagation Velocities With Rates of Turn**

There was an inverse correlation between degrees of turn and wavefront velocities during angulation with those before and after angulation. *P* values are derived by comparing the degrees of turn and wavefront velocities during angulation with those before and after angulation. *P*<0.05.

**Discussion**

Nonuniformity of conduction with localized regions of slow conduction is considered critical for reentry.1-7 Detailed mapping to characterize and localize slow conduction during VT has been limited by the resolution of clinical mapping technologies and complexities of the anatomy of the infarcted human heart.

Various animal, in vitro, and computer models1,2,10,11 indicate factors that determine cardiac conduction velocity, include tissue excitability, cellular architecture, cellular coupling, the degree of structural and functional anisotropy, wavefront curvature, changes in tissue dimensions, and current-to-load relationship.1,2,8,19 Mapping studies performed on intraoperative patients20 and explanted hearts21,22 and during catheter mapping3-7 indicate that although tissue excitability and the cellular action potential may be normal in the infarcted human heart,23 regions of myocardium forming the diastolic pathway have abnormal conduction properties, but these studies lack the resolution to show which regions of the circuit are responsible for the greatest slowing of conduction. Although anisotropy,19 wavefront turning,10,13 and other factors have been shown to affect conduction in animal studies, which of these factors predominate in causing slowing of conduction in human VT remains uncertain.

**Curvature and Conduction Velocity**

Using the noncontact mapping system, we have characterized the conduction properties of the diastolic pathway of the intact human heart, demonstrating myocardial conduction velocities similar to those measured in previous studies,22 with greatest slowing of conduction at regions with significant angulations and an inverse relationship between wavefront curvature and velocity, both within and outside the diastolic pathway.

**Conduction at Entry and Exit Turns**

Experimental models have shown that conduction through a small isthmus that expands to activate a larger mass of cardiac tissue is associated with greatest slowing just beyond the point of exit or expansion, where there is greatest curvature and greatest current-to-load mismatch.24 On leaving the diastolic pathway, abrupt expansion from a narrow isthmus to a large volume of excitable myocardium results in pronounced wavefront convexity, which has been implicated in slowing of activation and possibly conduction block in experimental models.10,11,24 The converse would be expected on entering an isthmus. In the present study, however, at entry turning points of human VT circuits, wavefront velocity slows to approximately half that in the outer pathway, with little additional change over the proximal portion of the diastolic pathway. Furthermore, wavefront velocity at the exit turning points did not change significantly from the distal diastolic pathway despite expanding to activate the ventricular myocardial mass from a narrow isthmus. As a consequence, velocity at entry was slower than exit, but degrees of turning were similar. The same observation has been noted in canine infarct experiments.25

This lack of concordance with experimental models that have used preparations of cultured or intact healthy myocardium may result from differences in the conduction properties of the diseased myocardium of the diastolic pathway21,22 and
the consequent change in the functional properties of the myocardium on entering and exiting the diastolic pathway.\textsuperscript{2,21,22} Conduction at entry and exit may therefore be determined not only by wavefront curvature but also by the combination of the change in the dimensions of the conduction pathway and the changing conduction characteristics of the constituent myocardium. Data from the early healing phase of the canine infarct model have shown that the epicardial border zone, where diastolic pathways are frequently located,\textsuperscript{26} constitutes regions with characteristic disorganization of gap junction distribution (also seen at the healed infarct border zone of human hearts), altered excitability, and conduction characteristics.\textsuperscript{26–28}

Isthmus Size and Conduction

A variation in the isthmus size along the course of the diastolic pathway may account for some of the heterogeneity of conduction, although there is evidence that isthmus size may have little influence on conduction velocity.\textsuperscript{10} The true 3-dimensional size of the diastolic isthmus cannot be determined by noncontact or epicardial mapping, because only the endocardial or epicardial surface is mapped. There is good pathological evidence from postmortem specimens of diseased hearts of the highly variable size and orientation of the surviving strands of myocardial tissue in the region of the diastolic pathway associated with infarct scar,\textsuperscript{21–23} thus providing additional structural explanation for nonuniformity of wavefront conduction within the diastolic pathway.

Clinical Implications

Failure to suppress VT safely by drugs that act on ion channels to modify the action potential has increased the focus on alternative strategies to block conduction in the treatment of VT.\textsuperscript{29} Understanding the mechanisms of slow conduction in human VT will help recognize and localize regions of the circuit most prone to conduction block and therefore guide treatments aimed at terminating and preventing tachycardia by promoting conduction block at vulnerable targets. This insight may also enable better interpretation of diagnostic maneuvers, such as resetting, entrainment, and pace mapping, during mapping and ablation of human VT.

Limitations of the Study

It is possible to map only endocardial portions of VT circuits using the noncontact system; subendocardial or intramural activation cannot be delineated. Tissue anisotropy may have accounted for some variation in wavefront velocity, but the exact orientations of myocardial fibers, particularly in scarred infarcted ventricles, could not be determined in this study.

The locations of entry and exit points of the diastolic pathway were determined by assessing the lines of block that formed the borders of the pathway, with activation sequences that funneled into (entry) and expanded out from (exit) the diastolic pathway. This means of determining the ends of the diastolic pathway has been validated previously by demonstrating concealed entrainment\textsuperscript{3} and was confirmed in 2 patients in the present study.

Any errors in creating the LV geometry would have resulted in some inaccuracies of endocardial distance measurements. To limit this error, large numbers of endocardial points at all LV segments were collected during the study to give a more precise endocardial geometry matrix.

Although errors in endocardial distance measurements will have arisen from using a series of short straight-line measurements between points on the endocardial geometry matrix, these will have been minor and will have affected the results uniformly. It is also known that although remaining acceptable, there are minor inaccuracies of electrogram reconstruction when MEA-to-endocardial distances are >34 mm in humans\textsuperscript{14} but that the error remains small to <50 mm. In the present study, 70% of all measurements were <35 mm and >90% were <45 mm.

Conclusions

Slowing of conduction in circuits causing VT in the intact infarcted human heart occurs over regions of turn of the wavefront, with an inverse relationship between wavefront curvature and velocity, both within and outside the diastolic pathway. Conduction is slower at entry than exit turning points of the diastolic pathway but is slowest during turns within the diastolic pathway. Characterization of conduction in human VT has important clinical implications for mapping and delivery of therapy aimed at blocking conduction.

Acknowledgments

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References

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