Identification of the Ventricular Tachycardia Isthmus After Infarction by Pace Mapping

Corinna B. Brunckhorst, MD; Etienne Delacretaz, MD; Kyoko Soejima, MD; William H. Maisel, MD, MPH; Peter L. Friedman, MD, PhD; William G. Stevenson, MD

Methods and Results—Left ventricular pace mapping during sinus rhythm was performed at 819 sites in 11 patients with VT late after infarction, and corresponding CARTO maps were reconstructed. An isthmus site was defined by entrainment and/or VT termination by ablation. Pace-mapping data were analyzed from the identified isthmus site and from sites at progressively increasing distances from this initial isthmus site. Sites where pace mapping produced the same QRS with different S-QRS delays were identified to attempt to trace the course of the isthmus. In 11 patients, 13 confluent low-voltage infarct regions were present. In all these regions, parts of VT isthmuses were identified by pace mapping. In 11 of 13 of the identified isthmus parts, the QRS morphology of the pace map matched a VT QRS. In 10 of 11 patients, radiofrequency ablation rendered clinical VTs noninducible. Successful ablation sites were localized within an isthmus identified by pace mapping in all of these 10 patients.

Conclusions—VT isthmuses can be identified and part of their course delineated by pace mapping during sinus rhythm. This method could help target isthmus sites for ablation during stable sinus rhythm. (Circulation. 2004;110:652-659.)

Key Words: tachycardia ■ mapping ■ catheter ablation ■ myocardial infarction

Sustained monomorphic ventricular tachycardia (VT) due to prior myocardial infarction is usually caused by myocardial reentry in the border zone of the infarct. VT reentry circuits are considered to be surrounded by regions of fixed or functional conduction block.1,2 In most of these reentry circuits, the myocyte discontinuities lead to formation of critical isthmuses that are desirable targets for ablation.3 Depolarization of the isthmus does not contribute to the surface ECG because of low-voltage electrograms, typical for the border zone of the infarct. The wave front leaves the isthmus at the exit from which the more normal myocardium is rapidly depolarized, which results in the formation of the QRS complex. Pace mapping during sinus rhythm at the exit of the isthmus should theoretically produce the same QRS morphology as during VT. Pace mapping at sites more proximally located in the isthmus should also produce a similar QRS complex, but with a longer stimulus-to-QRS (S-QRS) interval. Therefore, we hypothesized that part of the VT reentry circuit isthmus could be traced with pace mapping. Identification of the isthmuses would facilitate VT ablation.

Methods

Patients

Endocardial catheter mapping and radiofrequency (RF) catheter ablation were performed in 11 consecutive male patients (mean age 68±9 years) who were referred for VT ablation. All patients had a remote (>2 months) myocardial infarction. The mean left ventricular ejection fraction was 24±8%. Each patient had ≥2 episodes of sustained monomorphic VT within the preceding 6 months. The number of inducible VTs was 4.4±2.2 (range 2 to 10; Table 1). All but 1 patient had an implantable cardioverter defibrillator, which had been implanted previously. All patients had failed antiarrhythmic drug therapy.

Mapping and Ablation

Patients underwent catheter mapping and ablation according to a protocol approved by the Human Research Committee of Brigham and Women’s Hospital. After written informed consent was obtained, 6F multielectrode catheters were inserted percutaneously and positioned in the high right atrium, at the His position, and in the right ventricular apex. Left ventricular mapping used either a retrograde aortic or transseptal approach. Systemic anticoagulation with heparin was adjusted to an activated clotting time >250 seconds. Conscious sedation was achieved with intermittent administration of fentanyl and midazolam.

Catheter position was monitored and 3D anatomic reconstructions of left ventricular sites were displayed with the nonfluoroscopic mapping system CARTO (Biosense; Cordis Webster).4 A specially designed software program displayed voltage and S-QRS–interval maps and electrograms, the 12-lead ECG of each left ventricular site, and VT data. The 7F 4-mm-tip quadripolar mapping and ablation catheter has spacings of 1, 7, and 4 mm between electrodes. Bipolar electrograms from the distal electrodes of the mapping catheter were...
filtered at 10 to 400 Hz. The surface ECG leads were also stored on optical disc on a separate system (Prucka Engineering).

The initial step of the procedure included voltage mapping and pace mapping (see below) during stable sinus rhythm. The geometry of the left ventricle was defined, local electrograms were acquired, and pace mapping was performed at each site. The maximum electrogram amplitude (peak to peak) from the distal electrode pair of the mapping catheter was measured to the onset of the QRS in the ECG lead with the corresponding maps. VT was then induced by programmed stimulation. At selected sites, endocardial electrograms were acquired in VT, and entrainment mapping was used to determine whether the site was in the reentry circuit.11

For analysis, any of the following criteria defined an isthmus site; (1) pacing at the site entrained VT with concealed fusion, with an S-QRS <70% of the VT cycle length and either an S-QRS that matched the electrogram QRS (±20 ms) or a postspacing interval tachycardia cycle-length difference ±30 ms; (2) RF current application terminated VT without inducing ventricular ectopy; or (3) repeated VT termination occurred with catheter manipulation or pacing at the site, which prevented assessment of ablation during VT, and RF ablation abolished that inducible VT.

After ablation, programmed stimulation with up to 3 extrastimuli from 2 right ventricular sites using drive trains of 600 and 400 ms was performed to determine whether VTs were rendered noninducible. Successful VT ablation was defined as absence of inducible VT. VT modification was defined as absence of inducible clinical VT but with other monomorphic VTs remaining inducible.

At each stable left ventricular catheter location, pace mapping during sinus rhythm was performed. At isthmus sites, defined during VT, pace mapping in sinus rhythm was attempted after VT termination, with the mapping catheter being kept at the same location. Pace mapping was performed with unipolar stimuli (10 mA, 2 ms) from the distal electrode of the mapping catheter (cathode) and an electrode in the inferior vena cava (anode). The pacing cycle length was the same for each site in an individual patient (500 to 700 ms) and therefore was slightly above the sinus rate and slower than the rate of the induced VTs. The resulting 12-lead ECG morphology was stored from all sites with capture. Locations where pacing did not capture were marked with gray tags, which indicated dense scar. The S-QRS interval was measured to the onset of the QRS in the ECG lead with the shortest S-QRS by a custom, automated program and subsequently was adjusted by manual review. Electroanatomic maps of color-coded S-QRS intervals were reconstructed. Identified isthmus sites were marked on the map. Pace-mapping QRS morphologies were analyzed from the identified isthmus site and from sites at progressively increasing distances from this initial isthmus site. Sites where pace mapping produced the same QRS as that of the initial isthmus site with different S-QRS delays were identified to attempt to trace the course of the isthmus. A pace-mapping match was defined when 10 of 12 leads had the same morphology. The distance between the most distal site and the most proximal site of the matching isthmus sites was determined by a custom program.

### Results

In 11 patients, 819 electrograms of left ventricular sites (mean of 74±24 per patient) were recorded, and pace mapping was performed. Unipolar pacing did not capture at 48 sites (6%). In the 11 patients, 13 confluent low-voltage (≤1.5 mV) regions were present, consistent with infarcts (Figure 1A). Infarct locations were inferior or posterior in 8, anterior in 1, and in 2 discrete regions in 2 patients (posterobasal and inferoseptal in 1 and anteroapical and inferoseptal in the other). All the sites with an S-QRS >40 ms were located in the low-voltage area (Figure 1B). In each infarct region, a definite isthmus site could be identified with mapping techniques in VT (Figure 2). Starting from this initial site, part of the VT reentry circuit isthmus could be identified by pace mapping in each infarct region (Figure 3): 2 isthmuses in each of the 2 patients with 2 infarct regions and 1 isthmus in each of the 9 patients with 1 infarct region; however, in 1 infarct region, only the exit site could be determined (Table 2). The identified parts of the isthmuses had an average length of 2.4±0.6 (range 1.7 to 3.5) cm. The shortest S-QRS in each isthmus had a mean of 51±18 (range 23 to 84) ms. The longest S-QRS in each isthmus had a mean of 94±33 (range 49 to 141) ms; in contrast, the overall longest S-QRS was usually much longer than at the isthmus sites in most patients, with a mean of 160±70 (range 52 to 318) ms (P<0.05). Only

### Table 1. Baseline Characteristics: Mapping and Ablation Data

<table>
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<tr>
<th>Patient</th>
<th>Age, y</th>
<th>LVEF, %</th>
<th>Scar Location</th>
<th>Antiarrhythmics</th>
<th>PM Sites, n</th>
<th>PM CL, ms</th>
<th>VT Induced, n</th>
<th>VT Ablated, n</th>
<th>Acute Outcome</th>
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<tr>
<td>1</td>
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<td>25</td>
<td>Posterobasal/ inferoseptal</td>
<td>Amiodarone/atenolol/lidocaine/procainamide</td>
<td>109</td>
<td>600</td>
<td>3</td>
<td>2</td>
<td>Modification</td>
</tr>
<tr>
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<td>15</td>
<td>Inferobasal</td>
<td>Amiodarone/metropolol</td>
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<td>700</td>
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<tr>
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<td>Inferobasal</td>
<td>Mexiletine/carvedilol</td>
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<td>700</td>
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<tr>
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<td>Amiodarone/procainamide</td>
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<td>700</td>
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<tr>
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<td>Inferior</td>
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LVEF indicates left ventricular ejection fraction; PM, pace mapping; and CL, cycle length.
Figure 1. A, Electroanatomic bipolar voltage map of left ventricle is shown with prior inferoposterior wall infarction in posteroanterior view. Electrogram amplitude (mV) is color coded from red (≥0.2) to yellow, green, and blue to purple (≥1.5 mV). Tag 1 indicates termination of VT-1 with RF application; Tag 2, termination of VT-2 of this patient. B, Corresponding S-QRS map. S-QRS (ms) is color coded from red (≥40 ms) to yellow, green, and blue to purple (≥80 ms). Tags 1 to 8 indicate traced isthmus sites of Figure 3 with matching QRS morphology and progressively longer S-QRS intervals. Tags 9 and 10 indicate longest S-QRS intervals but with different QRS morphology. Figures 1A and 1B illustrate patient 6 in Tables 1 and 2.
2 (15%) of 13 isthmuses contained the longest S-QRS interval in an individual patient. The pace-mapping QRS morphology of the longest S-QRS interval of each patient matched an induced VT only in 3 (27%) of 11 patients. However, the pace-mapping QRS morphology at 11 (85%) of the 13 identified isthmus parts matched an induced VT (Figure 3).

Before ablation, 4.4 ± 2.2 (range 2 to 10) VTs were inducible per patient (Table 1). Of these 3.5 ± 2 (range 2 to 9) VTs were ablated, such that no VTs were inducible in 5 patients (45%) and VTs were modified in 5 (45%). One procedure was stopped prematurely because of a peripheral embolus to the foot from the insertion site in the femoral artery. Successful ablation sites were localized within an isthmus identified by pace mapping in 10 patients.

Discussion
VT late after myocardial infarction is usually due to reentry and is dependent on channels of slow conduction, the so-called isthmuses. These isthmuses are usually found at the border zone of the infarct. They are preferred targets for VT ablation because they are usually narrow and critical parts of the VT reentry circuit. However, many VTs are unmappable because of hemodynamic instability or poor reproducibility in the electrophysiology laboratory. Therefore, mapping and ablation in sinus rhythm has gained considerable interest and has been shown to be feasible.6–8 A previous study that included patients with ischemic and nonischemic cardiomyopathy and unmappable VT reported a success rate of 75% by placing extensive lines of RF lesions connecting the regions with the lowest-amplitude signals to regions with normal-amplitude signals or areas of block and crossing zones where pace mapping approximated the QRS morphology of VT.7 In the present study, we attempted to identify more limited regions for the VT substrate by tracing an isthmus with pace mapping in sinus rhythm.

Analysis of the QRS morphology during pace mapping in sinus rhythm is a well-established method for ablation of focal VTs but has been less reliable for guiding ablation of scar-related VTs.9–11 Propagation in the diseased myocardium is not homogeneous, and small differences in catheter location can cause grossly different propagation wave fronts and resulting QRS complexes.9,12–14a Regions of functional block, present during VT, may define propagation paths during tachycardia but not during pace mapping. However, the pace-mapping QRS morphology from a catheter located at or near the exit of a VT reentry circuit should usually produce a QRS morphology similar to that of VT. In the present study, a QRS match was defined as 10 of 12 leads with the same morphology.11,14a

Pace mapping in sinus rhythm also provides a measure of slow conduction, indicated by the interval between the stimulus and the QRS complex (S-QRS) exceeding 40 ms.11,14a Data in the present study showed that sites with an S-QRS delay were always in the infarct region, as identified by electrogram voltage. It is likely that pacing sites with long S-QRS delays are in a potential isthmus, adjacent to regions of conduction block.

The relationship between conduction time, pacing site location in the isthmus, and conduction block determines whether pace mapping in an isthmus produces a QRS that resembles that of VT. When pace mapping in a defined isthmus is performed, the stimulated wave front can only follow along its course, which occurs in at least 2 directions: the orthodromic and antidromic direction of the VT propagation. The wave front is only detected on the surface ECG when it leaves this protected channel. If the isthmus is long and the catheter is positioned in the distal part, near the exit, the orthodromic wave front leaves the exit and
Figure 3. Examples of pace mapping with matching QRS morphologies and progressively longer S-QRS intervals. Numbers correspond to sites in Figure 1B. Pace-map morphologies match induced VT-1. Figures 1 to 3 illustrate examples of same patient (patient 6 in Tables 1 and 2).
rapidly depolarizes the region along the infarct, colliding with and preventing emergence of the antidromic wave front from the infarct region. The resulting QRS complex is then similar to that of VT. If the isthmus is short, or the catheter is positioned more proximally, the stimulated antidromic wave front leaves the protected isthmus at the entrance, propagating to the surrounding myocardium and producing a different QRS morphology. If the orthodromic wave front reaches the exit, a fusion QRS is produced that includes depolarization from both antidromic and orthodromic wave fronts.

The presence of functional, rather than fixed, areas of block could also importantly influence these findings. If block defining an isthmus is present only during VT and is absent during pace mapping in sinus rhythm, the stimulated wave front produced by pace mapping at the isthmus site would propagate in all directions. We performed pace mapping at rates slower than the VT, which may reduce the likelihood of functional block or lines of functional block. The slower pacing rate was used because each patient had several VTs, and rapid pacing is likely to induce VT, which would prevent analysis of pace mapping and complicate the mapping procedure. Despite the difference between pacing rate and VT cycle length, we were able to identify isthmus sites with pace mapping in all the infarct areas, although only the exit of the isthmus was identified in 1 region. In 11 (85%) of these 13 isthmus segments, the QRS morphology matched 1 of the induced VT morphologies, and in the 10 with multiple isthmus parts identified, the S-QRS interval was progressively longer as the site moved along the isthmus (Figure 3), consistent with pacing progressively further from the exit.

The reentry circuit exit, which is more likely to be at the border of the infarct and close to the normal myocardium, often has no delay during pace mapping in sinus rhythm even though it is a desirable target for ablation. In 1 of the patients in the present study, only the exit site of the isthmus was identified, which had a short S-QRS of 26 ms. Pacing at surrounding sites produced different QRS morphologies, possibly indicating that the circuit dove deep to the endocardium, or functional block defined the circuit during VT and was absent during pace mapping. In 2

### TABLE 2. Pace-Mapping Data

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<th>Patient</th>
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<th>Longest S-QRS/PM-QRS=VT Match</th>
<th>Longest S-QRS Within Isthmus, ms</th>
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<td>70</td>
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(15%) of the 13 identified isthmus parts, pace mapping did not match either of that patient’s induced VTs, although a long S-QRS interval was present. These findings are consistent with pace mapping at proximal isthmus sites, with propagation away from the circuit in the antiodromic direction, as discussed above. The longest S-QRS intervals of the identified isthmus parts with a mean of 94±33 ms (excluding the one in which only the exit was identified) were much shorter than the longest S-QRS intervals of all sites in each patient, with a mean of 160±70 ms (P<0.05). The long S-QRS sites may be more proximal in isthmuses, where the antidromic wave front during pace mapping is able to propagate away from the reentry circuit, producing a QRS different from that of VT. These observations are consistent with a previous study showing that 66% of the sites at which the pace-mapping QRS complex resembled VT had an S-QRS interval <120 ms during entrainment, but in 89% of reentry circuit isthmus sites at which pace mapping did not match the VT QRS, the S-QRS during entrainment was ≥120 ms.11 This is a likely explanation for the observation that the longest S-QRS sites in most patients did not match any of the induced VTs. It is also possible that some of these long S-QRS sites were located in bystander regions of abnormal conduction. A bystander of 1 VT circuit can be a critical isthmus of another circuit, so that ablation in these areas might also be appropriate.

The observation that only 1 isthmus per infarct region was identified in patients in the present study may be due in part to the use of that isthmus for multiple VTs with different exits, such that ablation of that isthmus abolished more than 1 VT. Alternatively, extending the ablation across the isthmus to a site of inexcitable scar may have abolished other isthmuses. The remaining 13 VTs that were still inducible at the end of the procedure most likely used isthmuses that were not ablated, possibly because of an intramural or epicardial location.

In contrast to previous pace-mapping studies, the present investigation used electroanatomic mapping with exact anatomic localization of the S-QRS intervals and their morphologies and of the acquired parameters during VT. For the first time, it was therefore possible to correlate sinus rhythm pace mapping and VT data on a 3D reproduction of the left ventricle and display the precise location of part of the VT reentry circuit isthmuses.

Study Limitations

Patients in the present study were referred for catheter ablation and therefore were selected. The number of patients was relatively small. A match between S-QRS pace mapping and VT was determined as ≥10/12 and not as an exact match of 12/12, as in idiopathic VTs. Because of clinical and time restraints, sampling of sites was not homogeneous. Only limited isthmus sites were identified in VT, and the whole VT circuit was not outlined, so the pace mapping data were related only to the available VT data. The efficacy of ablation targeting the areas of isthmuses identified by pace mapping without mapping during VT is not known.

There are several caveats in interpretation of S-QRS delay with our methods. All patients in the present study were treated with antiarrhythmic drugs, and their influence on conduction could not be altered. Unipolar pacing was used to avoid changes that can be produced by capture from the proximal ring electrode when this is used as the anode.12,16 A fixed stimulus strength was used to facilitate rapid assessment during these procedures. Stimulus strength influences the size of the virtual electrode directly depolarized by the stimulus and can thus influence conduction away from the pacing site.16–18 The stimulus current was applied to a large-surface-area ablation electrode, which reduces the current density at the electrode tip compared with that observed with standard-sized electrodes.19

Conclusions

Parts of VT reentry circuit isthmuses can be traced during sinus rhythm by combining both the QRS morphology and the S-QRS delay from pace mapping in anatomic maps. Ablation within these isthmuses abolishes VT that utilizes that isthmus. This method may facilitate ablation of stable and unstable VTs during sinus rhythm, but further study is required to determine whether it is sufficiently accurate to be the sole guide for ablation.

Acknowledgment

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References


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