Functional Pace-Mapping Responses for Identification of Targets for Catheter Ablation of Scar-Mediated Ventricular Tachycardia

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Background—Myocardial scars harbor areas of slow conduction and display abnormal electrograms. Pace-mapping at these sites can generate a 12-lead ECG morphological match to a targeted ventricular tachycardia (VT), and in some instances, multiple exit morphologies can result. At times, this can also result in the initiation of VT, termed pace-mapped induction (PMI). We hypothesized that in patients undergoing catheter ablation of VT, scar substrates with multiple exit sites (MES) identified during pace-mapping have improved freedom from recurrent VT, and PMI of VT predicts successful sites of termination during ablation.

Methods and Results—High-density mapping was performed in all subjects to delineate scar (0.5–1.5 mV). Sites with abnormal electrograms were tagged, stimulated (bipolar 10 mA at 2 ms), and targeted for ablation. MES was defined as >1 QRS morphology from a single pacing site. PMI was defined as initiation of VT during pace-mapping (400–600 ms). In a 2-year period, 44 consecutive patients with scar-mediated VT underwent mapping and ablation. MES were observed during pace-mapping in 25 patients (57%). At 9 months, 74% of patients who exhibited MES during pace-mapping had no recurrence of VT compared with 42% of those without MES observed (P=0.024), with an overall freedom from VT of 61%. Thirteen patients (30%) demonstrated PMI, and termination of VT was seen in 95% (18/19) of sites where ablation was performed.

Conclusions—During pace-mapping, electrograms that exhibit MES and PMI may be specific for sites critical to reentry. These functional responses hold promise for identifying important sites for catheter ablation of VT. (Circ Arrhythm Electrophysiol. 2012;5:264-272.)

Key Words: ventricular tachycardia • ablation • pace-mapping

Scar-mediated ventricular tachycardia (VT) is hemodynamically unstable in >70% of cases, which significantly limits mapping for diastolic activity during tachycardia. Therefore, substrate-based techniques in sinus rhythm based on scar border zone delineation and identification of abnormal electrograms (EGMs) that represent areas of slow conduction are frequently targeted for ablation. Pace-mapping can serve as a surrogate for identification of exit sites by matching VT morphology, but the most conclusive demonstration of a critical isthmus requires entrainment of VT and/or termination of VT during ablation.

Clinical Perspective on p 272

Pacing within a scar can sometimes reveal multiple exits, which can produce distinctly different QRS morphologies during pacing. Further, if slow conduction is encountered deep within a scar at the site of stimulation, reentry of the index paced beat can by itself induce VT without the need for a programmed extrastimulus. The aim of this study is to describe 2 functional responses to pace-mapping at abnormal EGM sites within scar: (1) multiple exit sites (MES), or pace-maps that exhibit >1 morphology, and (2) induction of VT by pace-mapping, termed pace-mapped induction (PMI).

We hypothesized that in patients with scar-mediated VT, those with MES identified during pace-mapping followed by ablation have improved freedom from recurrent VT, and PMI of VT at a relatively slow drive train predicts successful sites of termination during ablation.

Methods

Patient Selection

Data obtained from consecutive patients referred for catheter ablation of scar-mediated VT over a 2-year period were retrospectively analyzed. Patients with evidence of electroanatomic scar from ischemic cardiomyopathy (ICM), nonischemic cardiomyopathy (NICM), hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular (RV) cardiomyopathy, valvular cardiomyopathy caused by rheumatic heart disease, and sarcoidosis were included. The diagnosis of NICM was based on the absence of coronary artery
disease (>75% stenosis), prior myocardial infarction, or significant valvular disease. The diagnosis of ICM was established by a history of infarction with Q waves, focal wall motion abnormality, or fixed perfusion defect correlated with coronary stenosis or prior intervention. All studies were performed under general anesthesia after preprocedural transesophageal echocardiography excluded intracardiac thrombus. Epicardial mapping was performed as clinically indicated at the discretion of the operator. The Institutional Review Board at UCLA approved review of the retrospective data.

**Electroanatomic Mapping and Pace-Mapping**

Endocardial mapping was performed through a transseptal approach. Transseptal puncture was performed using a Brockenbrough needle (BRK-1), with adjunctive radiofrequency energy (RF, Valley Laboratory, PA; 30 W) applied in cases of aneurysmal or thick interatrial septal anatomy. An activated clotting time goal of 250–350 seconds was maintained throughout the procedure with intravenous heparin. Epicardial access was obtained by percutaneous subxiphoid pericardial puncture and was performed surgically in patients with previous thoracotomy, as described previously.10–12

Electroanatomic voltage mapping was performed during the baseline rhythm, which was either sinus or paced, with a CARTO ( Biosense Webster, Diamond Bar, CA) or NavX EAM system (Ensite, St Jude Medical, Minnetonka, MN) as previously described.7,13,14 Higher-density mapping was performed within scar and at border zones. Bipolar signals were filtered at 5–500 Hz and displayed at 100 mm/s.

Abnormal EGMs were defined as sites with low voltage (<1.5 mV) with fractionation, split potentials, or late potentials (LP). LP were defined as EGMs exhibiting a component with a distinct onset after the QRS. Fractionated signals (multiple high-frequency deflections) and split potentials, with >1 component separated by >20 ms isoelectric segment, were tagged. Pace-mapping was performed (10 mA at 2 ms, 400–600 ms cycle length) at these sites for comparison with the 12-lead template of the clinical or induced VT. A perfect pace-map match was defined as a 12/12 lead match and good pace-map match was defined as a ≥10/12 match.

An MES was defined as an abnormal EGM site that exhibited ≥2 distinct QRS morphologies (<10/12 match to be distinct from each other) during the same pace-mapping drive. A schematic of MES from a single pace-map site within a theoretical isthmus is shown in Figure 1. The stimulus-to-QRS (S-QRS) intervals were measured to each different morphology, and the differences between S-QRS within the same drive between different morphologies was calculated (ΔS-QRS). Catheter stability was confirmed by fluoroscopy, electroanatomic mapping position, and EGM reproducibility. The first beat of pacing was not counted as a distinct morphology to minimize the possibility of fusion. At least 2 instances of consecutive QRS complexes of a given morphology were required to minimize the chance of counting a premature ventricular beat. An MES was considered a matched MES for VT if any one of the morphologies produced from pacing exhibited a ≥10/12 match with a targeted VT (Figure 2); 2:1 conduction, or exit block, was included if consistent and reproducible (>3 conducted beats) (Figure 2, lower). Three independent observers analyzed the pace-map drive trains to confirm distinct morphologies in a given MES.

**Figure 1.** Schematic of multiple exit sites: Three exit morphologies from a single pacing site (center, marked +). Blue areas present scar, or replacement fibrosis. VT indicates ventricular tachycardia.

**Figure 2.** Upper tracing, Two morphologies with 2 different stimulus latencies seen during pace-mapping within dense scar. The second morphology matches the targeted ventricular tachycardia (VT). Lower tracing, Three distinct morphologies with 2:1 exit block from a pace-map site with the last beat matched for targeted VT.
Criteria for MES During Pace-Mapping

Criteria for MES during pace-mapping included stable catheter position at abnormal EGM site; pacing drive at 400–600 ms (10 mA, 2 ms); ≥2 distinct QRS morphologies seen during same pace-map drive; distinct QRS requires <10/12 match between 2 morphologies; first captured beat excluded; and 2 occurrences of a morphology required.

Catheter Ablation and Follow-Up

VT was induced with programmed extrastimulus testing from the RV apex and outflow tract before or after creation of the electroanatomic map. If the patient was noninducible with programmed stimulation from the RV, stimulation was performed from the left ventricle (LV). Drive cycles of 600 and 400 ms, with decrement by 10 ms until refractoriness or 200 ms, were used with up to 4 extrastimuli. Induced VTs were stored as a template; a targeted VT had at least 1 of the following characteristics: (1) similar to 12-lead of presenting VT (when available), (2) similar in cycle length to that seen on implantable cardioverter-defibrillator interrogation, and (3) reproducibly inducible. All sustained VTs seen during the procedure, either spontaneous, mechanically, or induced by programmed stimulation, were counted. Ventricular flutter or sine wave tachycardia that degenerated into ventricular fibrillation was not counted as a VT morphology or targeted.

In cases in which VT was hemodynamically tolerated, entrainment mapping was attempted at sites demonstrating diastolic activity. An isthmus was defined as a site where (1) pacing for entrainment performed 20–40 ms shorter than tachycardia cycle length (TCL) with a postpacing interval within 30 ms of TCL with concealed fusion and equivalent stimulus-QRS interval to EGM-QRS (30–70% TCL) interval or (2) VT terminated during ablation.

Cases in which VT was induced from pace-mapping (10 mA at 2 ms, 400–600 ms cycle length) were termed as PMI. A PMI was considered matched if the pace-map morphology exhibited a ≥10/12 match with the immediately induced VT. As these PMI sites were represented by abnormal EGMs in sinus rhythm, ablation was attempted in these regions regardless of whether diastolic activity was seen. Termination of VT was recorded and correlated with PMI sites.

In cases in which an induced VT was not hemodynamically tolerated, a strategy of ablating abnormal EGM sites with good pace-maps (>10/12 match), all MES identified and all LP was used. Linear lesions were also created at border zones deemed to represent exit sites by pace-mapping. This strategy was also used after a tolerated VT was successfully eliminated to achieve extensive substrate modification in all patients. If the patient was noninducible for VT, ablation of all LPs and mapped abnormal EGMs was performed. If LPs were not identified, an encircling border zone lesion set was made (see Figure 3 for mapping and ablation protocol).

Ablation was performed with an open-irrigated catheter (ThermoCool, 3.5 mm, Biosense-Webster, Diamond Bar, CA) at 30–50 W, temperature limit 45°C at 30-mL flow rate. Radiofrequency energy was applied for 60 seconds per lesion. Ablation lesions were tagged on the electroanatomic map, and lesions were considered effective if they showed at least 1 of the following: a diminution or abolishment of the local EGM, failure to capture at 10 mA at 2-ms pacing, or impedance drop >10 Ω.

After ablation was performed in the region of the targeted VT, programmed stimulation was repeated with ventricular extrastimulus testing at drive cycles of 600 and 400 ms, with decrement by 10 ms until refractoriness or 200 ms, up to 4 extrastimuli at a minimum of 2 sites. If an inducible VT had the same morphology as one seen during the case or was tolerated, it was retargeted. Acute procedural results were categorized into 3 groups: (1) complete success: noninducible after ablation for all VTs, (2) partial success: inducible for nontargeted VT, and (3) failure. Recurrence of VT after hospital discharge was assessed by patient interview and interrogation of the implantable cardioverter-defibrillator.

Statistical Analysis

All symmetrical continuous data are summarized with mean±SD and compared using unpaired Student t test. Asymmetrical continuous data are summarized with medians and ranges. Comparison of proportions and categorical variables was performed using the Fisher exact test. Time-to-event curves for VT recurrence during the follow-up period were calculated by the Kaplan-Meier method and...
Table. Patient Characteristics

<table>
<thead>
<tr>
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<th>MES+ (n=25)</th>
<th>MES− (n=19)</th>
<th>( P ) Value</th>
</tr>
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<tbody>
<tr>
<td>Age, y</td>
<td>66±11</td>
<td>60±14</td>
<td>0.15</td>
</tr>
<tr>
<td>Sex, male, n (%)</td>
<td>25 (100)</td>
<td>17 (89)</td>
<td>0.18</td>
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<tr>
<td>LVEF, %</td>
<td>31±10</td>
<td>29±14</td>
<td>0.52</td>
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<tr>
<td>Prior MI, n (%)</td>
<td>18 (72)</td>
<td>8 (42)</td>
<td>0.06</td>
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<tr>
<td>ICD, n (%)</td>
<td>25 (100)</td>
<td>18 (90)</td>
<td>0.43</td>
</tr>
<tr>
<td>Prior ablation, n (%)</td>
<td>12 (48)</td>
<td>5 (26)</td>
<td>0.33</td>
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<tr>
<td>Amiodarone, %</td>
<td>16 (64)</td>
<td>15 (79)</td>
<td>0.33</td>
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MES indicates multiple exit sites; LVEF, left ventricular ejection fraction; MI, myocardial infarction; ICD, implantable cardioverter-defibrillator.

Results

Between March 2009 and February 2011, 44 consecutive patients with scar-mediated VT (ICM=26, NICM=10, arrhythmogenic RV cardiomyopathy=2, HCM=2, valvular cardiomyopathy=2, sarcoid cardiomyopathy=2) underwent mapping and ablation. The mean age was 64±13 years, and all but 2 patients were male. The average ejection fraction was 30±12%, and 41% of patients did not have a history of myocardial infarction. Seventy percent of patients failed amiodarone therapy and 39% had prior ablation. Epicardial mapping was performed in 22 patients (50%), of which 2 were performed with surgical access due to prior cardiac surgery.

Patient characteristics are shown in the Table, stratified by the presence of MES during pace-mapping. No statistically significant differences in baseline characteristics were found between the 2 groups. There was a trend toward a higher incidence of history of myocardial infarction in the MES group, although this difference was not statistically significant.

The median number of points mapped for the LV was 442 points (range, 213–1604) on the endocardium and 637 points (range, 350–841) on the epicardium. The RV was mapped in 7 patients, with a median of 195 points (range, 114–249). Among those with ICM, infarct location delineated by electroanatomic mapping was anterior in 42% (11/26), inferior in 31% (8/26), and inferolateral in 27% (7/26). The mean mapping density in low-voltage regions (≤1.5 mV) was 2.9 points/cm² (range, 0.6–7.2 points/cm²).

Three patients presented with incessant VT and 2 patients were noninducible for VT during preablation programmed stimulation. In the remaining 39 patients, VT was induced from the RV in 90% (35/39), and 10% (4/39) required LV stimulation to induce VT. Entrainment mapping was performed in 23% of patients (10/44).

Pace-Mapping and MES

Pace-mapping was attempted at 55±33 sites with abnormal EGMs per patient. Among these, 17±15 sites failed to capture during pacing (electrically unexcitable scar), and a mean of 38±20 pace-maps were generated per patient. MES were observed during pace-mapping in 57% of patients (25/44). Of these, 1 morphology exhibited a pace-map match of ≥10/12, for a targeted VT in 40% (10/25) of patients. No differences were seen in the number of pace-maps per patient between those with MES and those without MES (39±22 versus 38±20). A typical patient with MES with 3:2 conduction out of the pacing site showing 2 distinct morphologies is shown in Figure 4.

There was no difference in the median number of VTs induced in those who did not demonstrate MES (3 VTs; range, 0–6) compared with those with MES (3 VTs; range, 0–7). The mean VT cycle length was 406±91 ms in those with MES and 397±82 ms in those without MES (\( P = 0.73 \)). Twenty-eight percent of patients with MES (7/25) also exhibited 2:1 exit block during pace-mapping. Alternation between 2 distinct morphologies, or pace-map “alternans,” was seen in 16% (4/25) of patients.

At a total of 42 MES, 31% exhibited LP, with a median onset of 21 ms (range, 0–110 ms) after the QRS. The median EGM voltage at an MES site was 0.24 mV (range, 0.04–1.5 mV). Thirty-one percent (13/42) were located in border zone tissue (0.5–1.5 mV), 50% (21/42) were in dense scar (<0.5 mV), and 19% (8/42) were <0.1 mV. The median S-QRS was 86 ms (range, 28–332 ms) at MES, and the median ΔS-QRS between 2 morphologies during the same pacing train was 22 ms (range, 2–229 ms). Concealed entrainment of an isthmus was demonstrated in 28% (7/25) of patients with MES compared with 13% (2/16) of patients who did not exhibit MES.

Termination of a targeted VT was seen in 13 of 25 (52%) patients with MES compared with 7 of 19 (37%) without MES. Acute procedural success tended to be higher in those who exhibited MES (70% versus 56% complete success, \( P = 0.35 \)). No differences in radiofrequency duration (47±21 minutes versus 40±25 minutes, \( P = 0.33 \)) and procedure time (6.4±1.3 hours versus 6.7±1.9 hours, \( P = 0.56 \)) were seen between those with MES and those without, respectively.

The overall success rate for the entire cohort was 61%, with an average follow-up of 9±8 months. At 9 months, 74% patients who exhibited MES had freedom from VT recurrence compared with 42% of those without MES observed (\( P = 0.024 \)) (Figure 5A). Among patients with ICM (n=26), those who exhibited MES (n=18) had higher freedom from VT recurrence at 9 months compared with those who did not have MES (n=8) (76% versus 38%, \( P = 0.041 \)). When comparing patients with scar not due to prior infarction (n=18; NICM, HCM, arrhythmogenic RV cardiomyopathy, rheumatic heart disease, and sarcoid), a similar trend in freedom from recurrent VT was seen between patients with MES compared with those who did not have MES, although not statistically significant (69% versus 45%, \( P = 0.267 \)) (Figure 5B). Patients who exhibited 2:1 conduction during pace-mapping had a 71% freedom from VT. No significant difference in VT recurrence was seen between those with matched MES and those who did not have any MES morphology match a targeted VT.

Pace-Mapped Induction

PMI was observed in 30% (13/44) of patients. The mean cycle length of a pace-map drive was 474±73 ms. Concealed entrainment of an isthmus was demonstrated in 46% (6/13), and termination of VT during ablation was seen in 92% (12/13) of patients with PMI (Figure 6). A perfect-matched PMI was seen in 54% (7/13), a good-matched PMI was seen in 31% (4/13), and nonmatched PMI was seen in 15% (2/13).
In 1 patient, MES was seen during PMI, and VT terminated abruptly (1.2 seconds) at this site during ablation (Figure 7). At a total of 21 PMI sites, the median EGM voltage at a PMI site was 0.2 mV (range, 0.04–1.38 mV). Twenty-nine percent (6/21) were located in border zone tissue (0.5–1.5 mV), 52% (11/21) were in dense scar (<0.5 mV), and 19% (4/21) were <0.1 mV. LP was seen in 19% (4/21) of PMI sites, with a median onset 18 ms (range, 15–40 ms) after the QRS. The median S-QRS was 102 ms (range, 28–250 ms) at PMI sites. In 19% (4/21) of the PMI sites, diastolic activity was not apparent on filtered EGM recordings, although all terminated during ablation at that site. In the remaining 17 sites, where diastolic activity was seen, 71% had an EGM-QRS during VT within 30 ms of S-QRS during pace-mapping from sinus rhythm.

Ablation was attempted at 19 of 21 PMI sites, and termination was seen in 95% (18/19) of cases at the location of induction. In 3 of these cases, termination of VT required additional radiofrequency application in the same region of the PMI. Termination of VT occurred at a mean duration of 8.2±9.3 seconds (range, 1.1–32.0 seconds) into ablation.

Although the presence of PMI was strongly associated with termination of VT, it was not predictive of clinical success. The median number of VTs induced was higher in the group in which PMI was observed (3.5 VTs; range, 1–7) compared with those without (2 VTs; range, 0–6, \(P=0.038\)). No significant differences in radiofrequency duration (50±25 versus 41±21 minutes, \(P=0.20\)) or procedure time (6.5±1.4 versus 6.6±1.6 hours, \(P=0.83\)) were seen between patients with and without PMI, respectively. Acutely, 38% of patients with PMI were rendered noninducible after ablation. Patients with PMI of VT had a 54% freedom from VT recurrence, with median follow-up of 9 months.

**Discussion**

The major findings of the present study for patients undergoing scar-mediated VT ablation are (1) identification and ablation of MES seen during pace-mapping was associated with higher VT-free survival, and (2) sites with PMI of a targeted VT strongly predicted acutely successful ablation sites.

The demonstration of multiple QRS morphologies with pacing at a fixed site identifies regions within scar that have access to \(1\) exit site. These sites may be surrogates for common conducting channels or isthmuses, and in patients in whom MES can be identified and ablated, outcomes could potentially be improved when compared with patients in whom these sites are not identified during mapping.

Definitive proof of an isthmus relies on specific criteria during entrainment mapping.\(^9\)\(^,\)\(^15\) Whereas entrainment requires hemodynamic toleration of VT, termination during ablation or mechanical “bump” have also been used as surrogates for channels critical to reentry. In the majority of cases in which VT is not tolerated, the finding of longer stimulus latency for the same matched pace-map has been applied in clinical practice to identify channel orientation and proximity to the exit site within electroanatomic scar geometry.\(^17\) Although some advocate more extensive substrate
modification to achieve scar homogenization or the complete abolition of abnormal EGMs and late activity, a method to prioritize or screen for the functional importance of an abnormal EGM identified in sinus rhythm beyond pace-map morphology would have meaningful utility.

In this cohort, even subtle alterations in QRS morphology (<10/12 match) observed were termed MES. Traditionally, pace-mapped sites that do not match the targeted VT are abandoned. Importantly, the finding of MES from a single site suggests that pace-map matching is not essential for isthmus identification. Further proof of this concept is supported by the fact that the majority of PMI where termination during ablation occurred were not matched for the VT. This finding is consistent with previously published observations.

**Figure 5.** A, Kaplan-Meier curves comparing ventricular tachycardia (VT) recurrence in patients with multiple exit sites (MES) and those without MES seen during mapping and ablation. B, Kaplan-Meier curves comparing VT recurrence in patients with MES and those without MES in ischemic cardiomyopathy (ICM, above) and non-ICM groups (NICM, below).

**Figure 6.** Upper tracing, Pace-mapped induction (solid red box) is a perfect match for the induced ventricular tachycardia (dashed red box) at a site with late potential. Lower tracing, Concealed entrainment was demonstrated at this site before ablation.
that demonstrate the limitations of pace-mapping. In a cohort of 18 patients with postinfarct monomorphic VT, Stevenson et al \(^1\) demonstrated a nonmatched pace-map during sinus rhythm in 21% (6/28) of sites where concealed entrainment was demonstrated. Further, pace-map matches were seen in 29% of site where entrainment showed manifest fusion.

Several mechanisms to account for the inherent limitations of pace-mapping have been proposed. Factors that determine which exit site shows preferential conduction during reentry are not fully understood but potentially are governed by refractory periods, anisotropic conduction, and conduction velocity.\(^{20,21}\) Importantly, pacing in sinus rhythm from a point-source with radial impulse propagation may not mimic a broad reentrant wave front emerging from an exit site (Eikonal relationship). A different QRS morphology during pace-mapping or VT may result from myocardial fusion of ≥2 wave fronts from different exits. Areas of functional block that facilitate reentry during VT may be absent during pace-mapping, resulting in a different QRS morphology.\(^{17}\) Although pacing was performed at the same output for all patients, altering pacing output may change the virtual electrode of pacing, resulting in different myocardial capture, “near” and “far.”\(^{22}\) Although not specifically examined in the present study, pacing at different rates may alter functional properties of circuit conduction and the potential site of exit.

Consistent with the observed phenomenon of MES, 25–40% of patients with postinfarct VT exhibit pleomorphism, or

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**Figure 7.**

A. Two-exit site pace-mapped induction (PMI). Proof of both multiple exit sites and PMI as isthmus surrogate. The first morphology (red solid box) is a closer match to the induced ventricular tachycardia (VT) (red dashed box) than the second morphology (blue box). Ablation at this site (red asterisk), despite the absence of overt diastolic activity, resulted in prompt termination of VT. B. Construct of 2 exits on electroanatomic map during PMI (blue and red arrows) attached to the same reentrant VT circuit in the same patient with anteroseptal infarction. Voltage threshold displayed at 0.5–1.5 mV.
spontaneous morphological changes during sustained VT. Costeas et al used activation mapping in a canine model to show that distinctly different morphologies of reentry emanated from the same region but were altered by different exit routes out of the circuit. The evidence of a “shared isthmus” was reproduced by Kimber et al, who described intraoperative mapping of patients with pleomorphic VT. In 10 of 14 episodes in which QRS morphological shifts were observed, little (<2 cm) or no change in the site of origin was observed, suggesting a switch in only the site of wave front exit and epicardial breakthrough.

Although the presence of LPS in sinus rhythm indicates an area of local conduction slowing, the present findings suggest that functional conduction information beyond a 12-lead area of local conduction slowing, the present findings suggest that more extensive substrate modification is required to improve long-term outcomes.

The induction of VT during a relatively slow pacing train (400–600 ms) was highly specific for an isthmus, as termination of VT was seen in all but 1 case when ablation was performed at these sites. Induction of scar-mediated VT characteristically requires extrastimulus programmed stimulation to facilitate unidirectional block. If sufficiently slow conduction is present at the site of stimulation, a programmed extrastimulus may not be required to initiate reentry. Consistent with MES responses to pace-mapping, sites that were non–pace-stimulus may not be required to initiate reentry. Consistent with MES responses to pace-mapping, sites that were non–pace-matched for the clinical VT that resulted in PMI were still effective targets for arrhythmia termination.

Further studies on stimulation within scar and abnormal EGMs are necessary as presently used induction protocols are performed with the RV apex and outflow tract. In our cohort, 10% of patients required LV stimulation to induce VT after being noninducible from the RV. One patient was noninducible from 2 sites in the RV and had a PMI of the clinical VT from the epicardium of the LV. The presence of PMI was not predictive of clinical success in the present study, and this probably is due to the presence of multiple VTs from a complex substrate that cannot be entirely modified by the termination of a single VT morphology. This would suggest that more extensive substrate modification is required to improve long-term outcomes.

Limitations
This cohort represents a single-center experience, and the relatively small sample size limits the ability to draw strong, generalizable conclusions. Specific to our center, general anesthesia is used in all patients, which may result in slower VT cycle lengths and unstable hemodynamic status for any given sustained VT. Additionally, epicardial mapping was implemented in 50% of patients, as 39% of patients referred had prior failed endocardial ablation and 41% did not have history of myocardial infarction. Further studies are necessary, and the present findings should be reproduced at other specialized centers.

Although the findings of paced stimulus latency to QRS onset suggests slow conduction out of a conducting channel, a critical isthmus and bystander site cannot be differentiated by pace-mapping alone. However, conduction properties during tachycardia may be different during pace-mapping, making the interpretation of S-QRS during pace-mapping in comparison with S-QRS during entrainment unreliable.

Additionally, comprehensive ablation targeting all LP and matched pace-maps at abnormal EGM sites was performed in both groups, with and without MES. The proof for specificity of MES for critical isthmuses would be stronger if ablation had been limited to only sites demonstrating MES. However, this study was analyzed retrospectively for proof of concept; prospective studies will be required. In the present study, it remains that given the same intended approach, patients with MES sites identified had significantly improved freedom from VT. To minimize bias in interpretation of MES, we used 3 independent observers to determine if distinctly different morphologies were present.

Although there were more patients with ICM in the MES group, this difference was not statistically significant. Because ablation outcomes have been shown to be superior in ICM compared with NICM patients, the differences in VT recurrence between patients with and without MES may be amplified by this trend toward a higher proportion of ICM etiology in MES. However, within the ICM cohort (n=26), the same significant differences in clinical outcomes were observed between patients with and those without MES. Additionally, the same trend was seen in the group with noninfarct scar (n=18), although this was not statistically significant.

Clinical Implications
The present findings provide further mechanistic insights into mapping of scar-mediated VT during sinus rhythm. The goal of VT ablation is the rapid and accurate identification of a critical isthmus. Whereas the proof of an isthmus traditionally requires entrainment mapping during tachycardia, the identification of such sites based on sinus rhythm observations is highly desirable. The finding of MES at a site with abnormal EGM during substrate mapping provides functional conduction characteristics within scar and may suggest a potential isthmus or common conduction channel. Additionally, the induction of VT during pace-mapping may imply an area of obligate slow conduction for a given reentrant circuit and strongly predicts arrhythmia termination during ablation.

Conclusions
PMI of VT is strongly predictive of termination during ablation, and the finding of MES is associated with higher freedom from VT recurrence. These functional responses seen during pace-mapping hold promise for identifying important sites for catheter ablation of VT.

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CLINICAL PERSPECTIVE

Ablation for scar-related ventricular tachycardias (VTs) targets isthmuses critical to the initiation and maintenance of reentry. Because the majority of VTs are hemodynamically untolerated, potential surrogates for isthmuses that can be identified in sinus rhythm during substrate-based ablation of VT are desirable. The present study introduces 2 new functional responses seen during map-guided ablation in patients with prior cardiac surgery or difficult pericardial access. Circulation. 2003;100:1288–1296.


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