Fundamental Differences in Electrophysiologic and Electroanatomic Substrate Between Ischemic Cardiomyopathy Patients With and Without Clinical Ventricular Tachycardia

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**Objectives**
The aim of this study was to compare the electrophysiologic substrate in ischemic cardiomyopathy (ICM) patients with and without sustained monomorphic ventricular tachycardia (SMVT).

**Background**
Despite the universal presence of potentially arrhythmogenic left ventricular (LV) scarring, it is not clear why the majority of ICM patients never develop SMVT.

**Methods**
Detailed electroanatomic mapping of the LV endocardium was performed in 17 stable control ICM patients (16 males) without clinical SMVT. They were compared with 17 ICM patients (15 males) with spontaneous SMVT. Standard definitions of low-voltage zones and fractionated, isolated, and very late potentials were used.

**Results**
There were no significant baseline differences between the groups in terms of LV diameter, ejection fraction (27% vs. 28%), infarct territory, or time from infarction. However, control patients had smaller total low-voltage area (30% of surface area vs. 55%, p < 0.001); smaller very low-voltage area (7.3% vs. 29%, p < 0.001); higher mean voltage of low-voltage zones; fewer fractionated, isolated, and very late potentials with lower density of these scar-related electrograms per unit low-voltage area; and less SMVT inducibility. Potential conducting channels within dense scar and adjacent to the mitral annulus were more frequent in SMVT patients.

**Conclusions**
Compared with ICM patients with SMVT, an otherwise similar control group demonstrated markedly smaller endocardial low-voltage zones, lower scar-related electrogram density, and fewer conducting channels with faster conduction velocity. These findings may explain why some ICM patients develop SMVT and others do not.

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SMVT with that of an otherwise matched group of patients with spontaneous clinical SMVT. We hypothesized that differences in endocardial substrate characterized by a high-density electroanatomic map would account for the difference in SMVT incidence between the groups.

**Methods**

**Patient population.** We studied a control group of stable ICM patients who had no history of spontaneous clinical SMVT. They were enrolled prospectively from a population referred for prophylactic implantable cardioverter-defibrillator (ICD) insertion based on accepted primary prevention criteria (9). Inclusion criteria were as follows: 1) earlier MI(s) with more than 3 months elapsed since the most recent MI; 2) moderate-to-severe left ventricular (LV) systolic dysfunction with ejection fraction (EF) ≤40%; and 3) no history of clinical ventricular arrhythmias, syncope, cardiac arrest, or undiagnosed palpitations. These patients comprised the control group and are referred to as Group 1.

The control group was compared with a group of ICM patients with a history of symptomatic SMVT (Group 2). Inclusion criteria for these patients were similar to those for Group 1 except they were also required to have had at least 1 clinical SMVT episode, including those terminated by ICD therapy. The majority of this group (15 of 17 patients) was enrolled from consecutive patients undergoing substrate-based catheter ablation of SMVT causing frequent ICD therapies. Members of this group were also enrolled from patients undergoing electrophysiologic studies after presenting with SMVT before ICD insertion.

Exclusion criteria were: 1) angina, acute coronary syndrome, or MI within the last 3 months; 2) surgical or percutaneous revascularization within the last 3 months; 3) decompensated cardiac failure within the last month; 4) intracardiac thrombus; 5) uncontrolled hypertension; 6) active cerebrovascular disease; 7) severe aorto-iliac peripheral vascular disease; 8) any contradiction to anticoagulation; and 9) inability to provide informed consent.

All patients provided informed consent, and the protocol was approved by the Human Research Ethics Committee of Melbourne Health.

**Protocol.** Programmed ventricular stimulation and electroanatomic LV endocardial mapping was performed in both groups.

**PROGRAMMED VENTRICULAR STIMULATION.** Patients were studied in the post-absorptive state, after echocardiography excluded LV thrombus. A hexapolar catheter was advanced to the right ventricle. Stimulation was performed at twice the diastolic threshold from the right ventricular apex and right ventricular outflow tract. Four extrastimuli were delivered during 400- and 350-ms basic drive cycle lengths, with simultaneous stepwise 10-ms decrements in the coupling intervals of all 4 extrastimuli until the first extrastimulus reached a coupling interval of 200 ms or was refractory. Induced ventricular tachycardias were analyzed for cycle length, morphology, axis, hemodynamic and morphological stability, and induction and termination characteristics. All induced SMVTs in Group 2 ablation patients were targeted for ablation.

**ELECTROANATOMIC MAPPING.** After programmed stimulation, endocardial LV electroanatomic mapping was performed in the basal rhythm using a retrograde approach via the right common femoral artery. Systemic anticoagulation with intravenous unfractionated heparin was initiated, aiming for an activated clotting time of ≥250 s.

A 3.5-mm tip electroanatomic mapping catheter with 2-mm ring electrode and 1-mm interelectrode spacing (Navistar, Biosense Webster, Diamond Bar, California) was used. A 3-dimensional shell of the chamber geometry was constructed and electrograms were systematically recorded from the entire LV endocardium. Fluoroscopy and signal characteristics, in addition to electroanatomic stability, were used to verify stable endocardial catheter contact before points were acquired. Bipolar electrograms were filtered at 0.5 to 400 Hz and displayed at 200 mm/s on the CARTO (Biosense Webster) mapping system. Valvar locations were defined by electrograms and fluoroscopy. Mapping density was uniform across the LV endocardium and was sufficient to allow a fill threshold of 15 mm. An effort was made to obtain equal scar sampling density in all patients.

Acquired bipolar electrograms were analyzed for stability, amplitude, duration, morphology, and timing relative to the surface QRS complex and were displayed as isopotential maps on the CARTO system. The incorporated software was then used to measure chamber volume and surface area, as well as low-voltage areas. The total low-voltage zone (TLZ) was defined as the area displaying bipolar voltage of ≤1.5 mV and very low-voltage zones (VLZ) had bipolar voltage <0.5 mV (7). Points with bipolar voltage between 0.5 and 1.5 mV were labeled intermediate low-voltage zones (ILV) (7).

Electrograms were classified by 2 independent observers according to standard criteria (10) and modified for use with the recording system (11) into 1 of the following groups:

1. Normal electrograms with 3 or fewer sharp intrinsic deflections from baseline, amplitude ≥3 mV, duration <70 ms, and/or amplitude/duration >0.046.
2. Fractionated potentials with multiple intrinsic deflections, amplitude ≤0.5 mV, duration ≥133 ms, and/or amplitude/duration ≤0.005.

**Abbreviations and Acronyms**

- **EF**: ejection fraction
- **ICD**: implantable cardioverter-defibrillator
- **ICM**: ischemic cardiomyopathy
- **ILV**: intermediate low-voltage zone
- **LV**: left ventricle
- **MI**: myocardial infarction
- **SMVT**: sustained monomorphic ventricular tachycardia
- **TLZ**: total low-voltage zone
- **VLZ**: very low-voltage zone
3. Isolated potentials were those displaying an additional signal separated from the local ventricular electrogram by a >20-ms isoelectric interval (11).

4. Very late potentials were electrograms with an isolated component occurring ≥100 ms after the QRS.

5. Any electrograms not fitting into 1 of these categories were classified as nonfractionated abnormal electrograms.

The presence of potential conducting channels associated with the VLZ was inferred from observation of 1 of the following characteristics: 1) a corridor of preserved voltage component occurring ≥100 ms after the QRS.

Statistical analysis. All continuous data are presented as mean ± 1 SD. Baseline and mapping data were analyzed by the chi-square test (or Fisher exact test where appropriate), and with the Mann-Whitney U test. A value of p < 0.05 was considered statistically significant.

Results

Baseline characteristics. Baseline characteristics of the patients are presented in Table 1. Twenty-one patients with no history of clinical ventricular arrhythmias were enrolled in Group 1 but 4 were excluded due to LV thrombus or peripheral vascular disease. The remaining 17 patients who underwent LV endocardial mapping were compared with a group of 17 consecutive post-infarction patients who had spontaneous SMVT (Group 2).

Overall, the groups were similar (Table 1), notably, in terms of LV size and systolic function, time since myocardial infarction, revascularization, infarct distribution, and New York Heart Association functional class. All patients had undergone recent angiography or stress perfusion scintigraphy to exclude active ischemia. Background cardiac failure medical therapy was excellent in both groups. Group 1 control patients were significantly younger on average (mean age 59 ± 11 years vs. 67 ± 6.1 years, p = 0.03). No control patients were on amiodarone or other membrane active antiarrhythmic drugs, but there was an almost uniform use of antiarrhythmic drugs in the 17 SMVT patients to treat their index arrhythmias at the time of presentation (Table 1). These drugs included sotalol (mean daily dose 213 mg) in 6 patients, procainamide (maintenance intravenous infusion) in 2 patients, quinidine (mean daily dose, 1.2 g) in 3 patients, flecainide (mean daily dose, 250 mg) in 2 patients, and mexilitene (mean daily dose, 675 mg) in 8 patients, and these were ceased before the mapping procedure. Sixteen of 17 SMVT patients were being treated with maintenance doses of amiodarone at the time of presentation and this was continued in all patients at the time of mapping.

LV voltage mapping. The results of electroanatomic mapping are presented in Table 2. The mean number of tip points sampled was comparable between the groups (240 ± 96 points in Group 1; 234 ± 42 points in Group 2; p = NS). There were no significant differences between the 2 groups in terms of LV volume or total mapped endocardial surface area.

The areas of both the TLZ (bipolar voltage ≤1.5 mV) and the VLZ (<0.5 mV) were significantly smaller in Group 1 control patients compared with Group 2 SMVT patients (Table 2). An example of the endocardial voltage map from patients in each group is shown in Figure 1A. Differences in voltage persisted when controlled for LV endocardial surface area (Fig. 1B), and were not due to different point sampling.

Table 1 Baseline Characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Group 1: Control Patients</th>
<th>Group 2: SMVT Patients</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>17</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>Mean age, yrs</td>
<td>59 ± 11</td>
<td>67 ± 6.1</td>
<td>0.03</td>
</tr>
<tr>
<td>Male</td>
<td>16</td>
<td>15</td>
<td>NS</td>
</tr>
<tr>
<td>Anterior infarct</td>
<td>11</td>
<td>8</td>
<td>NS</td>
</tr>
<tr>
<td>Inferior infarct</td>
<td>6</td>
<td>9</td>
<td>NS</td>
</tr>
<tr>
<td>Mean time since infarct, months</td>
<td>93 ± 87</td>
<td>110 ± 55</td>
<td>NS</td>
</tr>
<tr>
<td>Ejection fraction, %</td>
<td>26 ± 6</td>
<td>28 ± 9</td>
<td>NS</td>
</tr>
<tr>
<td>LV diastolic dimension, mm</td>
<td>68 ± 7</td>
<td>65 ± 9</td>
<td>NS</td>
</tr>
<tr>
<td>Acute reperfusion therapy</td>
<td>13</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>Coronary bypass surgery</td>
<td>8</td>
<td>8</td>
<td>NS</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>2</td>
<td>2</td>
<td>NS</td>
</tr>
<tr>
<td>NYHA functional class 1/2/3</td>
<td>9/8/0</td>
<td>6/9/2</td>
<td>NS</td>
</tr>
<tr>
<td>ACE inhibitor or ARB</td>
<td>17</td>
<td>15</td>
<td>NS</td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>15</td>
<td>14</td>
<td>NS</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>0</td>
<td>16</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Other antiarrhythmic drugs</td>
<td>0</td>
<td>13</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Left bundle branch block</td>
<td>7</td>
<td>7</td>
<td>NS</td>
</tr>
<tr>
<td>QRS duration, ms</td>
<td>132 ± 31</td>
<td>144 ± 32</td>
<td>NS</td>
</tr>
</tbody>
</table>

Data are given as n or mean ± SD. ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; LV = left ventricular; NYHA = New York Heart Association; SMVT = sustained monomorphic ventricular tachycardia.

Table 2 Electroanatomic Mapping Data

<table>
<thead>
<tr>
<th>Data</th>
<th>Group 1: Control Patients</th>
<th>Group 2: SMVT Patients</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tip points, n</td>
<td>240 ± 96</td>
<td>234 ± 42</td>
<td>NS</td>
</tr>
<tr>
<td>LV volume, ml</td>
<td>247 ± 60</td>
<td>245 ± 97</td>
<td>NS</td>
</tr>
<tr>
<td>LV surface area, cm²</td>
<td>258 ± 37</td>
<td>237 ± 64</td>
<td>NS</td>
</tr>
<tr>
<td>TLZ (&lt;1.5 mV) area, cm²</td>
<td>78 ± 30</td>
<td>132 ± 64</td>
<td>0.009</td>
</tr>
<tr>
<td>VLZ (&lt;0.5 mV) area, cm²</td>
<td>19 ± 15</td>
<td>70 ± 31</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ILZ (0.5–1.5 mV) area, cm²</td>
<td>58 ± 22</td>
<td>62 ± 41</td>
<td>NS</td>
</tr>
<tr>
<td>VLZ as percent of TLZ area, %</td>
<td>23 ± 13</td>
<td>55 ± 15</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>VLZ sampling density, points/cm²</td>
<td>2.3 ± 1.8</td>
<td>1.5 ± 0.6</td>
<td>NS</td>
</tr>
</tbody>
</table>

Data are presented as mean ± SD.

ILZ = intermediate low-voltage zone; TLZ = total low-voltage zone; VLZ = very low-voltage zone; other abbreviations as in Table 1.
density (Table 2). In addition, the proportion of the TLZ that was composed of very low voltage was significantly less in the control group (Table 2). There was no significant difference between the groups with respect to the area of the intermediate low-voltage zone (ILZ) (bipolar voltage 0.5 to 1.5 mV) (Table 2). This zone was uniformly found surrounding the VLZ and most likely represents the scar-border zone. Within both the TLZ and VLZ, the mean bipolar voltage was significantly lower in Group 2 SMVT patients than in Group 1 control patients (Fig. 1C).

**Electrogram and channel characteristics.** When considering the entire endocardial map, fractionated and isolated potentials were significantly more prevalent in the Group 2 (SMVT) patients than in the control group (Fig. 2B). Very late potentials were essentially seen in the SMVT group only. Within the total, intermediate and very low-voltage zones there was also a significantly higher prevalence of fractionated, isolated, and very late potentials in Group 2 (SMVT) patients despite equal low-voltage zone point sampling density (Table 3). These differences persisted for fractionated and very late potential prevalence when controlled for TLZ surface area (Fig. 2C). However, the difference in isolated potential density did not reach statistical significance.

There was an increased prevalence of putative conducting channels in the SMVT group (Table 4). More Group 2 patients displayed mitral isthmus channels and voltage channels, and very late potentials (reflecting electrically conducting regions within dense VLZs) were largely found in this group alone. Examples of these are depicted in Figure 3.

**Programmed ventricular stimulation.** The results of programmed stimulation are summarized in Table 5. Group 2 patients with clinical tachycardia were more likely to be inducible and had a greater number of induced SMVTs. Of the total 68 induced SMVTs in Group 2, 30 were of left bundle branch block morphology and 38 had right bundle branch block configuration. Four induced tachycardias in Group 1 were of left bundle branch block pattern and 2 were of right bundle branch block pattern. A greater propor-
tion of the induced SMVTs were mappable in Group 2 compared with Group 1, although a high prevalence of unmappable SMVT was seen in both groups. The majority of inducible patients in both groups had no mappable SMVT.

The 4 inducible Group 1 control patients had no demonstrable substrate differences compared with the 13 noninducible control patients. The TLZ (38.3 ± 10.5% of LV surface area in the inducible patients vs. 27.5 ± 9.5% in the noninducible patients, p = NS) or VLZ (12.0 ± 6.1% vs. 5.8 ± 5.1%, respectively, p = NS) proportionate areas did not significantly differ between the inducible and noninducible control patients, nor did the prevalences of fractionated (4.2 ± 1.5% vs. 3.3 ± 2.6%, p = NS), isolated (8.5 ± 6.2% vs. 4.4 ± 3.3%, p = NS), or very late potentials (0.05 ± 0.1% vs. 0.03 ± 0.08% respectively, p = NS).

**Discussion**

This study showed significant differences in the endocardial LV electrophysiologic substrate between patients with and without clinical SMVT (rather than inducible SMVT) and...
expanded on previous observations. We demonstrated that when compared with ICM patients with SMVT, a control group with similar infarct size, EF, and time since infarct demonstrated: 1) markedly smaller endocardial low-voltage zones; 2) a lower prevalence of fractionated, isolated, and very late potentials; 3) fewer putative conducting channels; and 4) different low-voltage zone characteristics with more preserved voltages, and lower fractionated, isolated, and very late potential density. These observations may in part explain the difference in SMVT incidence between the groups.

**Earlier studies.** Two earlier studies examined the myocardial substrate in patients with previous MI who did not have clinical ventricular arrhythmias. In a landmark study, Cassidy et al. (10) used a nondeflectable catheter with 1-cm bipolar spacing to sample from a mean of 11 LV endocardial sites in a group of ischemic and dilated cardiomyopathy patients. Their study included 9 patients with previous MI and no clinical SMVT. At the 11 sites sampled, these patients demonstrated less endocardial fractionation than patients with clinical ventricular arrhythmias. However, a more detailed

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**Table 5** Inducibility With Programmed Stimulation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group 1: Control Patients</th>
<th>Group 2: SMVT Patients</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with inducible SMVT, n (%)</td>
<td>4 (24)</td>
<td>15 (88)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SMVTs induced, n (range)</td>
<td>6 (1-2)</td>
<td>68 (1-12)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mean cycle length, ms (range)</td>
<td>265 (250-290)</td>
<td>360 (270-610)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mappable SMVTs, n (%)</td>
<td>0 (0)</td>
<td>28 (41)</td>
<td>0.05</td>
</tr>
<tr>
<td>Patients with mappable SMVT, n</td>
<td>0</td>
<td>6</td>
<td>NS</td>
</tr>
<tr>
<td>Patients with PVT or VF, n</td>
<td>4</td>
<td>1</td>
<td>NS</td>
</tr>
</tbody>
</table>

PVT = polymorphic ventricular tachycardia; SMVT = sustained monomorphic ventricular tachycardia; VF = ventricular fibrillation.
assessment of the nature of the substrate could not be performed in that early study. In addition, they had less severe cardiomyopathy than the control patients in the present study (mean EF of 40 ± 18% compared with 26 ± 5%). Wiener et al. (16) also sampled a limited number of endocardial points (16 to 40 points) using a nondeflectable catheter in 11 patients having surgery for ventricular aneurysms, including 5 patients with no history of SMVT. They focused mapping to the scar-border zone and observed that patients without SMVT had less fractionation in this region of the aneurysm than patients with SMVT. The extent of global endocardial low-voltage and the presence of channels were not assessed with this limited mapping. Although these early studies provide some initial insights into the mechanism of post-infarct SMVT, they do not provide the detailed electroanatomic substrate analysis that has become the cornerstone of different ventricular tachycardia ablation strategies (5,7,17,18).

**Mechanism of post-infarct ventricular tachycardia and ablation targets.** The mechanism of post-infarct SMVT is scar-related re-entry in the majority of patients (1). Inhomogeneous scarring with varying degrees of subendocardial myocardial fiber preservation within dense zones of fibrosis leads to slowed conduction, nonuniform anisotropy, and the potential for channels within the scar zone—conditions necessary for the development of re-entry (2,3). In the ablation era, high-density electroanatomic mapping has been used to characterize the electrical correlates of this well-described pathologic substrate, allowing for development of substrate modification strategies (7) to target unappable SMVTs (19). Channels within a scar (isolated potentials [6,11], very late potentials) or regions at the scar-border zone (putative exit sites) (18,20) appear to represent critical components of the re-entrant circuit and have all been targeted with successful long-term ablation outcomes.

The current study compares these key markers of SMVT substrate by creating high-density maps of the left ventricle (mean of 240 contact points) with detailed sampling from within the low-voltage regions in ICM patients with and without previous clinical ventricular arrhythmias. Our data suggest that it is both the extent of the VLZ and the presence of numerous channels within this zone that are critical to the development of ventricular arrhythmias. Although the border zone region of the scar (ILZ) did not differ in area between the 2 groups, in the SMVT patients, this zone also had a significantly higher prevalence of putative conducting channels. It is noteworthy that earlier pathologic (21) and radiologic (22) studies have also suggested that a higher burden of fibrosis implies a greater coexisting substrate for development of SMVT (and hence an increased chance of spontaneous clinical arrhythmias), but until now detailed clinical mapping data from the dense scar region have been lacking.

**Electrograms, SMVT substrate, and channels.** Whether fractionated, isolated, or very late potentials are the most important markers of critical SMVT substrate cannot be answered by the current study. All 3 of these electrogram types demonstrated a higher prevalence in SMVT patients and a higher density within the scar than in controls. This suggests a fundamentally different scar composition (more “arrhythmogenic”) in the Group 2 SMVT patients. Notably, very late potentials were observed almost exclusively in patients with clinical SMVT. Recent observations (W. Jackman, MD, unpublished data, 2007) have highlighted very late potentials as a possible ablation target. In sinus or paced rhythm, very late potentials represent electrically viable sites that undergo very delayed activation by slow, serpiginous impulse propagation within scar. Although a proportion of these potentials may represent cul-de-sacs within scar, during SMVT the same sites may be an important zone of constrained diastolic isthmus conduction.

In addition, channels between scar and the mitral annulus (12,13), and scar-to-scar channels (14,15) may form a protected isthmus that can facilitate re-entry either by slow conduction or functional block. The current study also demonstrated that patients with clinical SMVT had a greater number of these putative channels than patients without SMVT.

**Discrepancy between infarct size and extent of low-voltage zones.** The finding of markedly smaller low-voltage zones in the control group despite similarly large previous MI(s) and severe LV systolic dysfunction suggests a significant degree of electromechanical uncoupling in the scarred LV regions in these patients. It is not clear why the 2 groups should demonstrate such significant differences in endocardial low-voltage zone extent and characteristics. Both groups had a similar proportion of patients who had undergone revascularization and acute reperfusion, and the mean time since index infarction was not significantly different. No patients had any evidence of active myocardial ischemia. It is interesting to speculate that increased subendocardial preservation in the acute infarction phase, reduced post-infarct remodeling, or differing healing responses (perhaps genetically determined) to MI explain the markedly reduced burden of endocardial low voltage seen in Group 1 (control) patients. Indeed the higher mean voltage observed within the low-voltage zones of Group 1 compared with those in Group 2 patients also suggests fundamental differences, not only in the extent of the scar but also in its composition.

**Clinical implications.** Recent studies have demonstrated that SMVT in patients with ICM can be successfully ablated by targeting channels within (11) or between areas of apparently dense scar (14,15), or between scar and the mitral annulus (12,13). The current study demonstrates that these channels are not universally present in patients with ICM and that the extent of dense scarring varies markedly. In particular, patients without clinical ventricular tachycardia had a significantly lower prevalence of channels and an almost complete absence of very late potentials within a much smaller dense scar body. This, despite the fact that patients were otherwise similar with respect to several key factors (infarct size, EF, time from infarct), suggests that not all ICM patients will inevitably develop the substrate critical to development of SMVT.
In patients with ischemic cardiomyopathy, tachycardias causing sudden death include SMVT, polymorphic ventricular tachycardia, or ventricular fibrillation. Whether the risk of sudden death is related to substrate features that favor SMVT is not clear but is likely to be the case. The present study has identified multiple differences in the LV substrate between patients with and without a history of clinical SMVT. This should stimulate further studies to determine whether these differences may also translate into differing risks of sudden cardiac death. Noninvasive techniques to determine dense scar extent and distribution might also be evaluated in this context.

**Study limitations.** By slowing conduction velocity and altering refractoriness, clinical antiarrhythmic drug exposure could potentially affect electroanatomic mapping findings. Differences in antiarrhythmic drug use between the 2 groups were unavoidable given the characteristics of the study groups. However, in the context of multiple reports of ventricular tachycardia ablation techniques targeting the SMVT substrate with successful outcome, it is unlikely that the markedly discrepant findings related to low-voltage zone area, scar density, and electrogram density between the groups simply represent an artifact of amiodarone or other antiarrhythmic drug use. We believe it is more likely that differences observed were due to the nature of the myocardial substrate.

Mapping data in this study were acquired with the use of a single commercially available electroanatomic mapping system and its associated proprietary software. We cannot exclude the introduction of a systematic bias related to this, but we feel this is unlikely given the widespread use of this system in SMVT ablation.

**Conclusions**

Despite equally severe LV dysfunction as well as similar infarct age and distribution, patients without clinical SMVT had significantly smaller endocardial low-voltage areas, fewer scar-related electrograms, and fewer potential conducting channels compared with similar ICM patients with spontaneous SMVT. These differences in endocardial electrophysiologic substrate may play an important role in SMVT arrhythmogenesis in the chronic post-infarct context.

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**REFERENCES**


**Key Words:** cardiomyopathy • tachycardia • myocardial infarction • electrophysiology.