Magnetic Resonance–Based Anatomical Analysis of Scar-Related Ventricular Tachycardia
Implications for Catheter Ablation


Abstract—In catheter ablation of scar-related monomorphic ventricular tachycardia (VT), substrate voltage mapping is used to electrically define the scar during sinus rhythm. However, the electrically defined scar may not accurately reflect the anatomical scar. Magnetic resonance–based visualization of the scar may elucidate the 3D anatomical correlation between the fine structural details of the scar and scar-related VT circuits. We registered VT activation sequence with the 3D scar anatomy derived from high-resolution contrast-enhanced MRI in a swine model of chronic myocardial infarction using epicardial sock electrodes (n=6, epicardial group), which have direct contact with the myocardium where the electrical signal is recorded. In a separate group of animals (n=5, endocardial group), we also assessed the incidence of endocardial reentry in this model using endocardial basket catheters. Ten to 12 weeks after myocardial infarction, sustained monomorphic VT was reproducibly induced in all animals (n=11). In the epicardial group, 21 VT morphologies were induced, of which 4 (19.0%) showed epicardial reentry. The reentry isthmus was characterized by a relatively small volume of viable myocardium bound by the scar tissue at the infarct border zone or over the infarct. In the endocardial group (n=5), 6 VT morphologies were induced, of which 4 (66.7%) showed endocardial reentry. In conclusion, MRI revealed a scar with spatially complex structures, particularly at the isthmus, with substrate for multiple VT morphologies after a single ischemic episode. Magnetic resonance–based visualization of scar morphology would potentially contribute to preprocedural planning for catheter ablation of scar-related, unmappable VT. (Circ Res. 2007;101:939-947.)

Key Words: ventricular tachycardia • catheter ablation • MRI

Catheter ablation of scar-related monomorphic ventricular tachycardia (VT) is a promising therapy that may reduce morbidity and mortality associated with this condition.¹ Substrate voltage mapping allows ablation of scar-related VT during sinus rhythm in patients with hemodynamically unstable VTs where conventional activation mapping is difficult.² This electroanatomical mapping technique defines scar in the endocardium or the epicardium during sinus rhythm, and the infarct border zones are ablated.

However, scar detection using the voltage-based substrate mapping has several limitations. First, it is essentially 2D because it defines the extent of scar on the surface, either endocardial or epicardial, and does not provide complex 3D anatomy of the scar. Second, spatial resolution of the voltage-based scar definition is limited by the number of points studied by the catheter operator. Lastly, electrically defined scar may not necessarily be identical with anatomical scar. For example, anatomically scarred myocardium with hypertrophy may be electrically defined normal.³

To elucidate the 3D anatomical correlation between the fine structural details of the scar and scar-related VT circuits, we registered VT activation sequence with the 3D scar anatomy derived from high-resolution contrast-enhanced MRI in a swine model of chronic myocardial infarction (MI). To achieve precise registration between the electrode locations and the MRI-derived 3D scar anatomy, we used epicardial sock electrodes (n=6 animals), which have direct contact with the myocardium where the electrical signal is recorded. Because not all VTs were expected to show epicardial reentry, we used endocardial basket catheters in a separate study to evaluate scar-related endocardial VTs where conventional mapping is difficult.
group of animals (n=5 animals) to assess the incidence of endocardial reentry in this infarct model. The electrical and scar data in this group were not spatially registered because the electrodes in the basket catheter are not in direct contact with the myocardium.

Materials and Methods
All studies were performed according to the Position of the American Heart Association on Research Animal Use. All protocols were approved by the Animal Care and Use Committee of the Johns Hopkins University School of Medicine.

Experimental Protocol
In 11 domestic swine (27 to 31 kg), the mid–left anterior descending coronary artery was occluded for 150 minutes using a balloon angioplasty catheter (2.7 Fr) via a carotid artery to create MI under general anesthesia. Ten to 12 weeks after MI, bipolar electrograms were recorded from 300 to 380 sites over the ventricular epicardium using a multielectrode epicardial sock placed via median sternotomy.

Table 1. Characteristics of VT Morphology in the Epicardial Group

<table>
<thead>
<tr>
<th>Animal</th>
<th>Weeks Post-MI</th>
<th>VT Morph</th>
<th>CL (ms)</th>
<th>Earliest Activation</th>
<th>Latest Activation</th>
<th>Overall Pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>11.9</td>
<td>1</td>
<td>218</td>
<td>Ant MI border</td>
<td>Ant RV</td>
<td>Heterogeneous</td>
</tr>
<tr>
<td>2</td>
<td>10.4</td>
<td>2</td>
<td>241</td>
<td>Ant MI border</td>
<td>Posterior</td>
<td>Centrifugal</td>
</tr>
<tr>
<td>3</td>
<td>10.9</td>
<td>6</td>
<td>177</td>
<td>Ant MI border</td>
<td>Posterior</td>
<td>Centrifugal</td>
</tr>
<tr>
<td>4</td>
<td>11.0</td>
<td>8</td>
<td>199</td>
<td>Lat RV</td>
<td>Lat LV</td>
<td>Heterogeneous</td>
</tr>
<tr>
<td>5</td>
<td>9.9</td>
<td>13</td>
<td>219</td>
<td>Posterior</td>
<td>Ant MI border</td>
<td>Centrifugal</td>
</tr>
<tr>
<td>6</td>
<td>10.1</td>
<td>17</td>
<td>184</td>
<td>Lat LV</td>
<td>Lat RV</td>
<td>Centrifugal</td>
</tr>
</tbody>
</table>

In case of reentry, the time reference was arbitrarily determined at the time of electrical activation at the isthmus, so that both the earliest and the latest activations were located at the isthmus. Morph indicates morphology; CL, cycle length; Ant, anterior; Lat, lateral.

Table 2. Characteristics of VT Morphology in the Endocardial Group

<table>
<thead>
<tr>
<th>Animal</th>
<th>Weeks Post-MI</th>
<th>EF (%)</th>
<th>VT Morph</th>
<th>CL (ms)</th>
<th>ECG Characteristics Spatial Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>10.0</td>
<td>38</td>
<td>1</td>
<td>187</td>
<td>Neg Concord RSAD Anteroseptal Anteroseptal Endocardial reentry</td>
</tr>
<tr>
<td>2</td>
<td>11.4</td>
<td>26</td>
<td>2</td>
<td>316</td>
<td>RBBB LAD Anteroseptal Anteroseptal Endocardial reentry</td>
</tr>
<tr>
<td>3</td>
<td>11.6</td>
<td>32</td>
<td>3</td>
<td>164</td>
<td>LBBB Normal Anteroseptal Anteroseptal Endocardial reentry</td>
</tr>
<tr>
<td>4</td>
<td>9.9</td>
<td>26</td>
<td>4</td>
<td>198</td>
<td>Pos Concord LAD Anteroseptal Anteroseptal Endocardial reentry</td>
</tr>
<tr>
<td>5</td>
<td>12.0</td>
<td>32</td>
<td>5</td>
<td>218</td>
<td>Pos Concord LAD Anteroseptal Anteroseptal Endocardial reentry</td>
</tr>
<tr>
<td>6</td>
<td>216</td>
<td></td>
<td></td>
<td></td>
<td>Neg Concord LAD Anteroseptal Anteroseptal Endocardial reentry</td>
</tr>
</tbody>
</table>

In case of reentry, the time reference was arbitrarily determined at the time of electrical activation at the isthmus, so that both the earliest and the latest activations were located at the isthmus. EF indicates left ventricular ejection fraction; Neg Concord, negative concordance; RBBB, right bundle branch block; LBBB, left bundle branch block; Pos Concord, positive concordance; RSAD, right superior axis deviation; LAD, left axis deviation; n=5 animals.
(n=6 animals, epicardial group) at a minimum sampling rate of 1000 Hz for 10 seconds. A pair of bipolar stimulating electrodes were attached to the ventricular myocardium to induce sustained monomorphic VT using a swine programmed electrical stimulation protocol. Sustained VT was defined as monomorphic VT lasting >15 seconds confirmed in multiple leads. After each VT induction, the stimulating electrodes were moved to another electrode location, and the programmed electrical stimulation was repeated for a total of 10 to 20 arbitrary locations. Because epicardial sock data were obtained in an open-chest setting, surface ECG was not recorded in the epicardial group. After completion of the programmed electrical stimulation, ex vivo MRI was conducted to visualize infarct geometry. Briefly, after intravenous administration of heparin 5000 IU and Gd-DTPA (gadolinium diethylene triamine pentaacetic acid 0.20 mmol/kg), the animals were euthanized, and the hearts were removed and filled with vinyl polysiloxane. As markers for registering the MR and the epicardial sock data, eight to fifteen 10×1 mm glass tubes filled with Gd-DTPA (5 mmol/L) were placed in the myocardium, and the locations of the sock electrodes and the glass tubes were digitized (Microscribe 3DLX, Immersion, San Jose, Calif). The sock electrodes were then removed from the heart, and the heart was scanned in a 1.5-T MR scanner with a 3D gradient echo sequence to visualize the infarct and to locate the glass tube markers (bandwidth, ±130 Hz/pixel; flip angle, 20°; echo time/repetition time, 4.02/9.7 ms; field of view, 100×100 mm; image matrix, 256×256; spatial resolution, 0.39×0.39×0.39 mm).

The remaining 5 animals (endocardial group) underwent in vivo MRI in a 1.5-T scanner for assessment of cardiac function, using an ECG-gated, cine steady-state free precession sequence (bandwidth, ±977 Hz/pixel; echo time/repetition time, 1.9/3.9 ms; readout flip angle, 45°; field of view, 300×300 mm; image matrix, 256×256; spatial resolution, 1.2×1.2×8.0 mm). Subsequently, bipolar electrograms were recorded from 176 sites in the left ventricular cavity using a multielectrode basket catheter placed via a carotid artery in a closed-chest setup. The same swine programmed electrical stimulation protocol was conducted to induce VT using a pacing catheter (7 Fr) advanced to the right ventricular apex through a femoral vein.

**Data Analysis**

Left ventricular volumes were calculated from in vivo MRI using MIPAV (NIH). Custom programs in MATLAB (Mathworks Inc) were used to visualize isochronal maps on the epicardial sock and the endocardial basket during VT. For the epicardial group, the normal ventricular myocardium and infarct were extracted from the ex vivo MR images using a signal intensity threshold to visualize a volumetric image of infarct in the ventricles. Candidate hyperenhanced

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**Figure 1.** A, High-resolution contrast-enhanced MRI (spatial resolution, 0.39×0.39×0.39 mm). The region of high intensity (indicated by white arrows) is the infarct. The myocardium in the infarct region is substantially thinner than the remote myocardium, and a thin rim of the viable myocardium on both the septal and the endocardial sides of the infarct was observed. B through D, Basal (B), anterior (C), and posterior (D) views of the 3D infarct geometry reconstructed from MRI. The infarct regions are represented by dark gray, and the normal ventricular myocardium (RV and LV) regions are shown in pink. The scar exhibited variable wall thickness, with occasional branching of the infarct structure at the periphery. There are islands of infarct within the viable myocardium and also islands of viable myocardium within the infarct. The scar involved the RV in the apical region.
areas were identified as infarct if hyperenhancement was seen in >1 slice and the mean intensity of the hyperenhanced region was >6 SD above the mean intensity of the remote region. The locations of the glass tube markers were determined from the ex vivo MR images, and the electrodes were spatially referenced to the MR images using rigid-body transformation.

Electrical activation was defined as the first peak of the derivative of QRS in the regional bipolar deflection. For the epicardial group, epicardial reentry was defined as a visually confirmed macroreentry circuit during VT where the activation pathway was accounted for by sock recordings and the electrode locations of the earliest and the latest activation were immediately adjacent to each other. In case of reentry, the time reference was arbitrarily determined at the time of electrical activation of the isthmus, so both the earliest and the latest activation were located at the isthmus. For the endocardial group, the definition of endocardial reentry was the same as that of epicardial reentry except that the activation was recorded by the endocardial basket catheter.

**Statistical Analysis**

Values are means±SD. Parameters were compared between the epicardial and endocardial groups using 1-way ANOVA. If a statistically significant result was obtained, then individual locations were compared by a 2-tailed t-test. A value of P<0.05 was considered statistically significant. Statistics were performed with SigmaStat 3.0 (SPSS, Chicago, Ill).

**Results**

All animals survived the MI procedure, which resulted in anteroseptal MI (n=11 animals). The animals underwent VT induction 10.7±0.7 weeks after MI in the epicardial group (n=6 animals) and 11.0±1.0 weeks after MI in the endocardial group (n=5 animals), and there was no significant difference between the two groups (P=NS; Tables 1 and 2). At the time of VT induction, left ventricular end-diastolic volume was 61.2±14.9 mL, left ventricular end-systolic volume was 42.0±9.3 mL, and left ventricular ejection fraction was 30.8±5.1% (endocardial group; Table 2). Sustained monomorphic VT was reproducibly induced in all 11 animals. The cycle length was 204±32 ms in the epicardial group (n=39 VTs) and 217±53 ms in the endocardial group (n=6 VTs), and there was no significant difference between the 2 groups (P=NS). All VTs were hemodynamically unstable.

**Scar Geometry**

High-resolution contrast-enhanced MRI revealed a 3D complex structure of the scar in this occlusion/reperfusion MI model. The myocardium in the infarct region is substantially thinner than the remote myocardium, and there was a thin rim of the viable myocardium on both the septal and the endocardial sides of the infarct (Figure 1A). The scar exhibited variable wall thickness with occasional branching of the infarct structure at the periphery (Figure 1B through 1D). There are islands of infarct within the viable myocardium, and also islands of viable myocardium within the infarct. The scar involved the right ventricle (RV) in the apical region.

**Epicardial Reentry and Scar Geometry**

In the epicardial group (n=6 animals), a total of 76 sites were stimulated (12.7±3.0 per animal), and 21 VT morphologies (3.5±1.6 per animal) were induced at 39 sites (6.5±4.6 per animal, Table 1). Four (19.0%) of 21 VT morphologies induced at 7 sites were epicardial reentry (Figure 2 and Table 1). The epicardial reentry circuits were mostly of the classic figure-of-8 type, and the central common pathway, or the isthmus, was located at the infarct border zone or over the
infarct in all 4 epicardial reentry morphologies. The scar geometry at the isthmus often contained multiple three-dimensionally intricate structures. In VT no. 5 (Table 1), the isthmus was located at the posteroapical segment of the infarct border zone. The scar geometry at the isthmus was characterized by scar tissue interspersed with multiple canals of viable myocardium (Figure 3A). Electrical activation initially spread longitudinally toward the base, split into two opposite lateral directions, then returned longitudinally and apically to the posteroapical region. The isthmus of VT no. 12 was located at the anterior irregular surface of the scar (Figure 3B). This irregular surface consists of thin interwinding layers of viable myocardium (see MR image in Figure 3B). There were two distinct epicardial reentry circuits in the same heart with different isthmus locations (VT nos. 18 and 19). In VT no. 18, the isthmus was located at the anterior right ventricular insertion where a small volume of viable myocardium bound by the infarct tissue is protruding into the RV (Figure 4A). The isthmus of VT no. 19 was located at a small volume of viable myocardium surrounded by the infarct tissue at the anteroapical infarct border (Figure 4B).

The remaining 17 VT morphologies induced at 32 sites did not show epicardial reentry, but mostly showed the centrifugal pattern where unidirectional electrical propagation spread from the breakthrough point to other regions of the heart (Figures 5 and 6 and Table 1). In VT no. 2, the epicardial breakthrough was located at the infarct border zone in the anterior left ventricle (LV) and spread toward the RV and the lateral LV, ending at the latest site in the posterior segment (Figure 5A). The infarct geometry at the breakthrough regions was characterized by multiple islands and protrusions of viable and scar myocardium, where electrical activation exits from the endocardial side of the myocardium. In VT no. 4, the propagation pattern was centrifugal and similar to that of VT no. 2 but spread in the opposite direction, from the posterior segment to the anterior LV (Figure 5B). The latest activation site was the normal myocardium at the infarct border in the anterior LV. In VT no. 7, the breakthrough points were located in the viable myocardium in the RV, and the electrical wave propagated to the latest activation site in the viable myocardium in the anterior LV (Figure 6A). In VT no. 17, the breakthrough was located in the viable myocardium in the lateral LV, and the electrical wave propagated to the latest activation site in the infarct border zone (Figure 6B). Some VT activation sequences showed a heterogeneous
Endocardial Reentry

In the endocardial group (n=5 animals), a total of 7 sites were stimulated (1.4±0.5 per animal), and 6 VT morphologies (1.2±0.4 per animal) were induced at 6 sites (1.2±0.4 per animal; Table 2). Four (66.7%) of 6 VT morphologies were endocardial reentry (the Figure in the online data supplement at http://circres.ahajournals.org), and the reentry circuits were mostly of the classic figure-of-8 type. The isthmus of all 4 VT morphologies was located at the anteroseptal aspect of the left ventricular endocardium, where the scar was located. The 2 remaining VT morphologies did not show endocardial reentry; 1 showed the centrifugal pattern where unilateral electrical propagation spread from the anterior to the septal LV, and the other morphology was of heterogeneous pattern without global propagation pattern (data not shown).

Discussion

The present study combined electrical measurements and contrast-enhanced MRI to examine the 3D anatomical correlation between the scar and VT reentry circuits in chronic MI. To interpret the data accurately, we focused on studying only macroreentry circuits visually confirmed on the epicardial sock recordings. As anticipated, most VT activation sequences in the epicardial group did not show epicardial reentry but the centrifugal or heterogeneous patterns (Table 1). This does not mean that those epicardial “nonreentry” patterns arose from a triggered or automatic mechanism. Rather, all of the VTs induced in the epicardial group were most likely reentry because they were reproducibly induced by programmed stimulation.14 It is possible that the electrical activation propagated back to the site of origin by a pathway undetectable by surface recordings.15 The relatively high incidence of reentry in the endocardial group (66.7%) strongly suggests that at least some of those epicardial “nonreentry” patterns are accounted for by endocardial reentry. In addition, heterogeneous patterns in the epicardial group likely represent both spatially and temporally heterogeneous arrival of propagation wavefront from endocardial or intramural reentry.

Scar Geometry

Contrast-enhanced MRI with high spatial resolution (0.39×0.39×0.39 mm) minimized the partial volume effect7 and visualized the fine details of the 3D infarct structure that are not easy to grasp from the 3D tomographic images (Figure 1). Our results clearly demonstrate that the scar morphology is not just transmural or nontransmural but much more complex than previously thought. The occlusion/reperfusion MI model in the present study reflects a clinical scenario in which acute coronary artery occlusion is followed by early revascularization, and our data indicate that only a single
ischemic episode without baseline atherosclerosis can create 3D intricate scar anatomy that could independently function as a substrate for VT. This is consistent with the fact that sustained VT can be induced in patients who receive thrombolytic therapy after the first MI.16

Electroanatomical Correlation During VT

Histologically, restoration of coronary blood flow by reperfusion within 2 to 3 hours rescues some of the ischemic cells from cell death,17–19 resulting in heterogeneous necrosis in the infarct region.20–25 This creates multiple regions of nonuniform anisotropy26 where the electrical impulse conduction can be significantly delayed.27 Contrast-enhanced MRI revealed that both the isthmus of the reentry circuits and the epicardial breakthrough of the centrifugal circuits were characterized by 3D complex structures of the viable and scar myocardium in the periinfarct region. Thus, MR-guided morphological assessment of the scar may help narrow the focus to the potential locations of ablation targets, rather than targeting as much of the infarct border zone as possible. However, there are no apparent anatomical characteristics that distinguish between these 2 structures; thus it is not clear whether all of such anatomical structures should be targeted to successfully prevent recurrence of VT.

The epicardial breakthrough was mostly at the infarct border (Figure 5A and 5B) but was often located at the viable myocardium (Figure 6B). This indicates that the epicardial breakthrough of the centrifugal circuits is not the site of origin but simply a surface exit of a continuous circuit. This is consistent with the clinical observation that a sizable minority of the successful VT ablation sites are in the viable myocardium.3 Furthermore, the same anatomical structure at the infarct border was found to serve as the isthmus and a centrifugal pathway in different VT morphologies (Figure 4B and Figure 6B). Therefore, it appears valid to target the complex anatomical structures in the peri-infarct region in any VT circuits.

Clinical Implications

It has been shown that MR-derived scar geometry can noninvasively predict the presence of VT substrate in patients in vivo.11,28 This study further advanced the concept and presents a possibility to predict locations of potential VT circuits and isthmus from scar geometry that could be noninvasively obtained by MRI before ablation procedures. Given the marked complexity of scar geometry, MR-based scar analysis would potentially play a complementary role in catheter ablation of unmappable VT, rather than replacing the current approach of detailed mapping. This would be particularly important for preprocedural planning for some fraction of scar-related VT circuits that are of epicardial origin3,29 or involve both the epicardial and endocardial layers,29,30 where successful ablation requires epicardial delivery of radiofrequency energy. Given the noninvasive nature of MRI, the MR-based scar analysis could play a role not only in preprocedural planning but also could be repeated after failed procedures until successful ablation is accomplished. MRI can visualize the spatial and temporal extent of ablation lesions,31–33 which can also be incorporated in the MR-based scar analysis. Detailed description of scar geometry would facilitate electroanatomical mapping of VT substrate34 to target all inducible and spontaneous monomorphic VTs to

Figure 5. Epicardial centrifugal pattern of VT registered with MR-derived scar. A possible electrical propagation at the infarct border zone is indicated by dashed red arrows. A, VT no. 2. The epicardial breakthrough (circumscribed by a broken red line) was located at the infarct border zone in the anterior LV and spread toward the RV and the lateral LV, ending at the latest site in the posterior segment. The infarct geometry at the breakthrough regions is characterized by multiple islands and protrusions of viable and scar myocardium, where electrical activation exits from the endocardial side of the myocardium. B, VT no. 4. The propagation pattern was centrifugal and similar to that of VT no. 2 but spread in the opposite direction, from the posterior segment to the anterior LV. The latest activation site (circumscribed by a broken red line) was the normal myocardium at the infarct border in the anterior LV.
reduce VT recurrences and implantable cardioverter-defibrillator firing.\textsuperscript{1,2,3} The spatial resolution of cardiac MRI has improved dramatically over the past several years, and high-resolution scar imaging used in the present study will likely become available in vivo with high-speed imaging techniques.

**Limitations**

In the epicardial group, we were able to study anatomical correlation between scar geometry and VT circuits for a fraction of inducible VTs. In the endocardial group, the electrical and scar data were not registered to characterize the scar at the VT isthmus because the electrodes in the basket catheter are not in direct contact with the myocardium and precise registration cannot be conducted in vivo. Therefore, scar characteristics of the isthmus described in this study apply to only epicardial reentry circuits in this model. Because we did not conduct simultaneous acquisition of endocardial and epicardial signals, we could not study 3D complex VT circuits that travel both the endocardium and the epicardium with reference to the infarct geometry.\textsuperscript{29}

**Conclusions**

MRI revealed a scar with spatially complex structures, particularly at the isthmus, with substrate for multiple VT morphologies after a single ischemic episode. The reentry isthmus was characterized by a relatively small volume of viable myocardium bound by the scar tissue at the infarct border zone or over the infarct. MR-based visualization of scar morphology would potentially contribute to preprocedural planning for catheter ablation of scar-related, unmap-pable VT.

**Acknowledgments**

We thank Edward J. Ciaccio, PhD, for valuable input on electrophysiological data analysis and Kenneth Rent for excellent technical assistance.

**Sources of Funding**

This work was supported by National Heart, Lung, and Blood Institute grants R01-HL64795 (to H.R.H.), PO1-HL077180 (to D. A. Kass and A.C.L.), and Z01-HL004609 (to E.R.M.) and the Donald W. Reynolds Cardiovascular Research Foundation.

**Disclosures**

None.

**References**


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Circ Res. 2007;101:939-947; originally published online October 4, 2007;
doi: 10.1161/CIRCRESAHA.107.158980

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Print ISSN: 0009-7330. Online ISSN: 1524-4571

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Supplemental Figure 1. Left panel (A-D): Endocardial reentry. Right panel (A-D): Surface ECG of respective VT circuits. The endocardial reentry circuits were mostly of the classic figure-of-eight type. The isthmus of all 4 VT morphologies was located at the anteroseptal aspect of the LV endocardium, where the scar was located.
Supplemental Figure