Simultaneous Biventricular Noncontact Mapping and Ablation of Septal Ventricular Tachycardia in a Chronic Ovine Infarct Model

Gopal Sivagangabalan, BSc, MBBS, FRACP; Jim Pouliopoulos, MSc; Kaimin Huang, BSc; Michael A. Barry, BSc; Juntang Lu; Stuart P. Thomas, MB, FRACP, PhD; David L. Ross, MBBS, FRACP; Aravinda Thiagalingam, MBBS, FRACP, PhD; Pramesh Kovoor, MBBS, FRACP, PhD

Background—We assessed a novel simultaneous biventricular mapping and ablation approach for septal ventricular tachycardia (VT) in a chronic ovine infarct model.

Methods and Results—In 8 sheep with inducible VT, mapping and ablation were performed 9±3 months after percutaneously induced myocardial infarction, with left ventricular ejection fraction 23±8%. Scar was identified by EnSite Dynamic Substrate Mapping plus CARTO voltage mapping. Thirty VT episodes (cycle length, 235±42 ms) were mapped with simultaneous analyses using EnSite arrays deployed in both the left ventricle and the right ventricle. Short ablation lines were created perpendicular to the breakout pathway along the scar border in the ventricle with earliest activity. If septal VT was still inducible, this line was extended before ablation in the second chamber. The end point of noninducibility of VT was achieved in all animals. The mean difference in delay in noncontact breakout timing between the ventricles was shorter for VT with (n=18) than without (n=12) septal breakout (32±7.8 ms, P<0.001). In 5 of 6 animals, after ablation in one ventricle, septal VT was still inducible with a common breakout site in the second ventricle. After septal ablation in the second ventricle, VT was no longer inducible. In the 6 animals in which septal VT had been ablated, transmural septal ablation was identified at the scar border, with overlapping left ventricular and right ventricular ablation lesions present in 5 of 6 (septal thickness 8 to 17 mm) and left ventricular endocardial ablation being transmural in 1 of 6 (6 mm).

Conclusions—Biventricular scar and VT activation mapping correctly localizes septal VT pathways, directing ablation from one or both septal endocardial aspects. Creation of a transmural septal lesion at the scar border interrupting VT exit points is highly effective at ablating septal VT. (Circ Arrhythmia Electrophysiol. 2009;2:441-449.)

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

Postinfarction ventricular arrhythmias remain a leading cause of death in survivors of acute myocardial infarction (MI) despite recent advances in reperfusion therapy.1 Implantable cardioverter-defibrillators are the gold standard for the prevention of sudden arrhythmic death in high-risk postinfarction patients.2 Therapy is limited by being reactionary (arrhythmia detection and device therapy) rather than preventive. Patients who experience implantable cardioverter-defibrillator activations, particularly shocks, often have significant psychological disturbance, which is usually underreported.3,4

Clinical Perspective on p 449
Catheter radiofrequency ablation is used as an adjunctive therapy, usually reserved for patients having multiple implantable cardioverter-defibrillator shocks, but has an emerging role in first-line therapy.5 Arrhythmias arising from the septum can present with multiple ECG morphologies, making conventional contact electrophysiological mapping difficult, particularly with hemodynamically unstable arrhythmias.

The aim of this study was to assess a novel method of simultaneous biventricular mapping to identify critical sites for ablation in an ovine model of septal ventricular tachycardia (VT).

Methods
All procedures were performed in the Westmead Hospital vivarium with approval from the institutional animal ethics committee.

MI Induction
MI was induced via a closed-chest method in 15 male sheep (Merino). Each animal was fasted overnight and then premedicated with 2 mg of IM xylazine (Ilum Xylazil, Troy Laboratories, Smithfield, NSW, Australia). Anesthesia was induced with 200 mg...
of IV propofol (Fresofyl, Pharmatel Fresenius Kabi, Hornsby, Australia) via a 7F sheath inserted under aseptic technique in the left external jugular vein. Each animal was intubated with an 8.0 endotracheal tube and anesthesia maintained with 2.5% inhaled isoflurane (Aerrane, Baxter, Deerfield, Ill). Throughout the procedure the ECG, oxygen saturation, end-tidal CO2, and arterial pressure were monitored. A 7F AL 1 or AL2 (Boston Scientific, Natick, Mass) guide coronary catheter was engaged in the left main coronary artery, and a 0.014-inch coronary angioplasty wire (PT Graphix, Boston Scientific, Natick, Mass) was selectively passed into the homonymous artery (ovine left anterior descending artery equivalent). A 3.0×20-mm coronary angioplasty balloon (Sprinter, Medtronic, Minneapolis, Minn) was deployed midway in the homonymous artery equivalent for 3 hours, as described by Reek.6 After balloon deflation, each animal was observed on the anesthetic table with full hemodynamic monitoring for 1 hour. Analgesia was given with 0.3 mg of buprenorphine IM (Temgic, Schering-Plough, Kenilworth, NJ).

Electrophysiological Study

Each animal had an electrophysiological study (EPS) to assess inducibility of VT at 5 to 8 weeks after MI, allowing time for healing and scar maturation. The sheep was premedicated, anesthetized, and monitored with the same regimen as described previously. Programmed ventricular stimulation (PVS) in an attempt to induce VT was performed with a quadripolar catheter (Blazer, Boston Scientific) deployed in the right ventricular (RV) apex. The protocol used drive trains of 8 beats followed by up to 4 extrastimuli. Two basic cycle lengths, 400 ms and 350 ms were used. Each extrastimulus was introduced at 300 ms and decremented by 10 ms until ventricular refractoriness. The end point was sustained monomorphic VT with cycle length >150 ms.

Sheep with inducible VT were commenced on oral sotalol (Sotocor, Bristol-Myers Squib, New York, NY) 40 mg bid. Animals that did not have inducible VT were not included in this study.

VT Mapping and Ablation Study

Sotalol was stopped for 7 days before the procedure. Each animal was premedicated, anesthetized, and monitored with the same regimen as described previously.

A RefStar (Biosense Webster, Diamond Bar, Calif) electrode patch was attached posteriorly in the interscapular space of each animal after the position was confirmed on fluoroscopy to overlie the heart silhouette. Bilateral femoral arterial and venous access was obtained. A left ventriculogram was performed to assess postinfarction ventricular function. An EnSite (St Jude Medical, St Paul, Minn) Multi-Electrode Array (MEA) was then advanced from the left femoral artery by a retrograde transaortic approach into the left ventricle (LV). The MEA was deployed, with the distal pigtail at the LV apex. A second MEA was positioned in the RV with the distal pigtail at the RV apex. Heparin was administered with a loading dose of 5000 IU and 1000 IU every hour thereafter.

A NaviStar (Biosense Webster, Diamond Bar, Calif) D-curve catheter was then advanced sequentially into both ventricles and used to simultaneously acquire an EnSite 3D geometry and a CARTO (Biosense Webster) 3D electroanatomic voltage map of each ventricle.

The EnSite system had a sampling rate of 1.2 kHz with filter settings 0.1 to 300 Hz. The CARTO system recorded bipolar electrograms between the distal and second electrodes of the NaviStar catheter. The interelectrode spacing was 1 mm. Unipolar filtering was set at 0.05 to 400 Hz, and bipolar filtering set at 30 to 400 Hz. Wilson central terminal (derived from leads I, II, and III) was assigned as the unipolar reference electrode. Local electrograms during sinus rhythm were recorded at each stable catheter point; the bipolar and unipolar peak-to-peak voltage amplitude was automatically calculated by the CARTO system. A bipolar peak-to-peak voltage <0.5 mV was considered dense scar, and bipolar peak-to-peak voltage >1.5 mV was considered normal myocardium. A corresponding voltage color range was applied to the geometry.

Mapping points were collected on both EnSite and CARTO systems, including pacing points, until the entire chamber had been sampled. High-density mapping was performed around regions of low bipolar voltage.

A minimum of 5 points were selected for pacing for construction of dynamic substrate maps (DSM) in both ventricles using the EnSite systems. Pacing points encompassed all aspects of both ventricles. A noncontact recording was made during pacing from each site. Pacing from the right atrium and a recording of sinus rhythm were included in DSM construction. All pacing was delivered at twice the local diastolic threshold at a cycle length of 400 ms. For each pacing site recording and from a sinus rhythm recording, zones of low electrogram voltage, <50% and <30% of the peak negative electrogram voltage (PNV), were marked. Scar was defined as an area in which the low-voltage zones from each recording overlapped. The region between the 30% and 50% PNV zones was used to identify the scar border.

PVS was performed to induce VT. Induced arrhythmias were terminated by 100 to 360 J biphasic external shock (LifePak 12, Medtronic). All VT episodes were recorded simultaneously on both the left and right EnSite systems.

A common fiducial timing marker (DaqSync, Westmead Hospital, Australia) was used to align recordings on both EnSite systems. For each VT beat, color thresholds were set from 0% to 50% PNV to aid identification of the breakout site in each ventricle. Breakout was defined as the point where the first virtual systolic electrogram crossed the 50% PNV threshold. The timing of breakout for a single VT beat was compared between chambers (Figure 1). The presystolic (prebreakout) activity was identified in both ventricles and along with the breakout point marked on both geometries.

The protocol used for ablation is presented in Figure 2. The strategy of ablation was to create a short (2-cm) line perpendicular to the noncontact VT propagation pathway leading to breakout, at border regions of scar identified by EnSite low-voltage zones, and CARTO contact bipolar electrograms (Figure 3). If the VT breakout was not associated with a region of scar, a shorter ablation line (1 to 2 cm) was created, again perpendicular to the propagation pathway leading to breakout. The chamber with earliest breakout was targeted first. Ablation was delivered by a radiofrequency generator (IBI 1500T8, St Jude Medical) with settings for irrigated tip ablation in the RV of 40 W, 50°C, and in the LV of 50 W, 50°C. The irrigation rate was 20 mL/min for all ablations. After this, PVS was repeated and if VT with a similar circuit was present, the initial ablation line was extended (up to 2 cm on either side of the initial line) to cover the propagation pathway leading to the new breakout point. If VT was still inducible despite ablation on one side of the septum, ablation was performed on the other side, targeting the propagation pathway leading to breakout from the region of low voltage.

The end point was noninducibility of VT in all animals, with PVS performed from 2 different sites (RV apex and RV outflow tract) with 2 basic cycle lengths (350 ms and 400 ms), using up to 4 extrastimuli for each induction.

At the end of the procedure, each animal was euthanized by inducing ventricular fibrillation with rapid RV pacing. The heart was excised immediately, and both ventricles were opened to identify the area of scar and ablation lesions. The septum and free walls were dissected to assess for transmurality of ablation.

Statistical Analysis

All analysis was performed using the Statistical Package for the Social Sciences for Windows (release 14.0, SPSS Inc, Chicago, Ill). A paired t test was used to compare within-sheep cycle length of VT induced at EPS and at VT mapping and ablation study. A linear mixed-effects model was used to compare mean values. Categorical variables were compared using Fisher exact test. A 2-tailed P<0.05 was considered significant.

Results

MI Induction

A 3.0×20.0-mm balloon was inflated at 8 atm for 3 hours in 15 sheep. The homonymous left anterior descending artery
Figure 1. Example of simultaneous biventricular noncontact mapping of nonhemodynamically tolerated septal VT. The same VT beat is compared in the LV (top panel, right anterior oblique view) and the right ventricle (bottom panel, left lateral view). Unipolar isopotential noncontact mapping demonstrates the breakout point in both chambers (white representing activated myocardium and purple, resting myocardium) with the color voltage range set from 0 mV to 50% PNV of the VT beat in each chamber. The red star in the center of the white breakout region indicates the position of the tracking virtual electrogram. On the LV panel, a 50% DSM marker (orange) and 30% DSM marker (red) are annotated on the geometry, demonstrating the relationship of the breakout point to the scar border. At the bottom of the LV panel, 3 surface ECG leads and 4 virtual electrograms (positioned over the breakout site) are shown; at the bottom, the fiducial timing marker is displayed. In the RV, 4 virtual electrograms at the RV breakout site and the fiducial timing marker are shown. In both chambers, the yellow vertical marker indicates the timing of breakout in each chamber. The red vertical marker is a caliper used to align the breakout timing in both chambers. In this example, the virtuals at both breakout points have QS morphology and the LV and RV breakouts are 223 ms and 237 ms, respectively, from the beginning of the timing marker, demonstrating the LV breakout 14 ms earlier than the RV breakout.
was completely occluded midway along its length. The survival rate after the MI was 87% (13/15).

**Electrophysiological Study**

The median time from MI induction to EPS was 52/8 days. Of the 13 sheep that underwent EPS, 62% (8/13) had inducible VT. VT was induced on the first induction in 5 of 8 and on the second induction in 3 of 8. All induced VT was monomorphic in morphology, with a mean cycle length of 181/31 ms, and reverted with external biphasic defibrillation (100 to 360 J).

VT mapping and ablation procedure were performed in 8 surviving sheep. The interval from MI induction to the VT ablation procedure was 39±13 weeks. The initial left ventriculogram in each animal showed areas of dyskinesis and hypokinesis involving the distal interventricular septum, apex, and anterior wall, with mean LV ejection fraction 23±8%. No change in hemodynamic parameters was observed after deployment of both noncontact multielectrode arrays in all animals.

The CARTO bipolar voltage maps and EnSite DSM showed areas of scar involving the septum, apex, and anterior walls of the LV in all animals. Septal scar was not reliably identified in the RV on either electroanatomic system in any of the sheep. The CARTO system provided spatial alignment of the RV and LV bipolar voltage maps demonstrating the relative shape and size of the septum (Figure 4).

A total of 27 VTs were induced by PVS; all were poorly tolerated, preventing entrainment or point-to-point contact mapping. A further 2 VTs occurred after ablation, and 1 VT occurred after catheter manipulation and was sustained despite catheter withdrawal from the chamber. All 30 VTs had distinct ECG morphologies (minimum of 1 lead morphology variation) and were included for analysis. The mean cycle length (ms) of all induced VT was significantly longer than the induced VT at EPS early after MI (mean difference, 64.4±20.5; P<0.001). One of the 8 animals studied did not have inducible VT at the time of the mapping procedure and was not included in further analysis.

Six of 7 sheep with inducible VT had septal breakouts at the septal scar border, as identified by noncontact mapping. All LV VT breakout points and the 50% DSM area were located within 40 mm of the EnSite MEA. There was no significant difference in the mean cycle length of VT with (n=18) and without (n=12) septal involvement (mean difference, 7.3±17.4; P=0.682). The delay in breakout timing between both ventricles was significantly shorter for VT with than without earliest breakout at the septum (mean difference, 32±7.8; P<0.001). The relationships for septal VTs between earliest breakout chamber identified by biventricular noncontact mapping, and virtual electrogram morphology at breakout site in each ventricles, and ECG bundle-branch block morphology is presented in Table 1. The ECG bundle-branch block morphology did not correspond well with the chamber of earliest breakout identified by biventricular noncontact mapping. The presence of sharp QS or slurred QS or RS morphology on the RV virtual electrogram at the site of RV breakout was able to determine which ventricle had the earliest breakout (P=0.012).

The number of VTs and the sequence of chamber ablation are presented in Table 2. All LV VT breakout sites were located in normal myocardium in close proximity to the 50% PNV DSM marker, indicating that the site of breakout occurred where the VT pathway exited the scar. All free wall VTs were successfully ablated with linear free wall ablation in the chamber of earliest breakout. One animal required separate LV and RV ablation for both LV and RV free wall VT. In 5 of 6 animals with septal VT, both LV and RV septal ablation was required to render VT noninducible. All ablations were performed between the 50% PNV and 30% PNV DSM markers, corresponding to an increase in contact bipolar peak-to-peak voltage amplitude from <0.5 mV within dense scar to ≥1 mV. All VTs (septal and free wall) were noninducible at the end of the study.

At thoracotomy, visual inspection showed epicardial scar involved the LV apex, anterior wall, and extended across the interventricular groove to involve the anterior wall of the RV. In all animals, the distal septum was densely scarred and thinned with visible scar on both the LV and RV endocardial.
surfaces. Ablation lines in the LV and RV were located at scar border regions. Free wall ablation lines were not transmural. Of the 6 sheep with septal VT circuits, transmural septal lesions were identified at the scar border zone. In 5 animals, the transmural septal lesion was the result of overlapping LV and RV septal endocardial lesions, neither being individually transmural (Figure 5). In these 5 animals, the septal thickness at the scar border ablation site ranged from 8 to 17 mm. In the sixth animal, the LV septal ablation line was transmural, with a septal thickness of 6 mm.

**Discussion**

This study demonstrated an effective method of ablation for postinfarction VT involving the interventricular septum. Simultaneous biventricular noncontact mapping of hemodynamically unstable septal VT provided both accurate substrate and VT activation maps. The strategy of creating short
ablation lines perpendicular to VT propagation before breakthrough, at the scar border, was effective in creating transmural septal ablation lesions, making VT noninducible. In 83% of the sheep with septal VT, ablation from both the LV and RV endocardial septal aspects were necessary to create transmural septal lesions.

The role of the interventricular septum in postinfarction VT has not been well described. Conventional ablation targets for VT are reentrant circuit exit points as identified by entrainment, pace mapping, or contact or noncontact activation mapping. However, reentrant circuits can be large and involve all aspects of the ventricles, with the exit point being remote from critical pathways within the circuit. Substrate-based ablation is limited to isolating areas of scar where VT is likely to originate and may require long, complex ablation lines that can extend into areas of electrically normal myocardium. Targeted radiofrequency ablation delivered at a single critical site can interrupt several VT morphologies. However, radiofrequency ablation for postinfarction VT may be ineffective because of limited lesion size even with irrigated endocardial ablation. Many reentrant circuits are located epicardially or intramurally, including deep septal sites. Combined endocardial and epicardial ablation may be required. After successful ablation of clinical VT, VT with faster cycle lengths is often still inducible, reflecting the multiplicity of postinfarction circuits.

The majority of postinfarction VT is not hemodynamically tolerated. This limits point-to-point activation mapping and the use of entrainment to locate isthmus exit sites. Simultaneous biventricular noncontact mapping in this study defined the presystolic propagation pathway to breakthrough and spread of unstable VT across both chambers, with detailed septal involvement. Eighteen of the 30 VTs in this series had noncontact earliest breakthroughs that involved the interventricular septum.

In the study by Klemm et al demonstrating the advantages of combined contact and noncontact mapping, the Dx Landmark function of the EnSite array system was used to create bipolar voltage substrate maps. This allowed noncontact propagation maps of VT to be used in conjunction with detailed contact bipolar substrate maps to identify critical borders for targeted ablation. The limitation of Dx Landmarking is that the contact maps cannot be created during initial chamber geometry acquisition and only 1 chamber can be mapped, as opposed to the CARTO system, which generates contact maps simultaneous to geometry acquisition and allows voltage mapping of multiple chambers but does not allow single-beat mapping of VT. Simultaneous use of CARTO (contact voltage maps) and EnSite (single-beat activation mapping) is feasible as used in this study. The advantage of this setup was the ability to spatially orientate the 2 ventricles and hence the size and shape of the septum on the CARTO system, whereas each of the 2 EnSite arrays provided single-beat activation mapping of VT. However, combining the individual advantages of CARTO and EnSite adds significant cost and complexity because the systems are not designed to be used together. A simpler alternative is to combine single-beat noncontact VT mapping with DSM, as used in this study. We have previously compared DSM with CARTO contact electrograms and validated the DSM scar localization with needle recordings and histology. Use of DSM allows VT activation to be viewed over low-voltage substrate marked on the electroanatomic geometry, showing the relationship of the arrhythmic circuit to areas of scar. Generation of DSM is also faster than contact voltage maps because individual points do not require verification. A major limitation of the DSM tool is that accuracy decreases beyond

### Table 1. Relationship of Surface ECG and EnSite Endocardial Virtual Electrograms to Septal VT With Earliest Breakout in the Left or Right Ventricle

<table>
<thead>
<tr>
<th></th>
<th>Septal VT, LV Breakout</th>
<th>Septal VT, RV Breakout</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV virtual morphology at breakout</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sharp QS</td>
<td>5/8</td>
<td>6/10</td>
<td>1.000</td>
</tr>
<tr>
<td>Slurred QS</td>
<td>2/8</td>
<td>3/10</td>
<td></td>
</tr>
<tr>
<td>RS</td>
<td>1/8</td>
<td>1/10</td>
<td></td>
</tr>
<tr>
<td>RV virtual morphology at breakout</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sharp QS</td>
<td>2/8</td>
<td>8/10</td>
<td>0.012</td>
</tr>
<tr>
<td>Slurred QS</td>
<td>6/8</td>
<td>1/10</td>
<td></td>
</tr>
<tr>
<td>RS</td>
<td>0</td>
<td>1/10</td>
<td></td>
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<tr>
<td>Surface ECG morphology</td>
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<td></td>
</tr>
<tr>
<td>LBBB</td>
<td>6/8</td>
<td>10/10</td>
<td>0.183</td>
</tr>
<tr>
<td>RBBB</td>
<td>2/8</td>
<td>0</td>
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</table>

Figure 4. Example of CARTO bipolar voltage mapping of the LV and RV in 1 animal. An approximate LAO (45°) view is shown, with color range <0.5 mV (red), >1.5 mV (magenta). Extensive scarring is evident in the apical half of the LV extending along the concave septal surface. The spatial orientation and curvature of the septum is seen in the space between the 2 ventricles.
from the equatorial plane of the array, an issue in large dilated chambers but not seen in the current study. The relationship of VT activation pathways to areas of scar defined by DSM was used to plan ablation lines. This strategy enabled the use of substrate localization (DSM) to be combined with activation mapping of hemodynamically unstable VT. Subsequent pathological examination confirmed that ablation lines were in fact delivered at critical scar borders.

The surface ECG morphology of VT was not helpful in identifying septal versus free wall VT or to identify which chamber had earliest septal VT breakout. However, the differences in anatomy, heart orientation in the chest cavity, and ECG lead placement may mean that ECG morphology in the ovine model does not have the same implications as seen clinically. The delay in breakout between ventricles allowed differentiation of VT with septal circuits, which, as expected, had a significantly shorter delay. All VT with septal pathways had a delay in breakout between ventricles of \(25\) ms. It may be postulated that VT with short delay between breakouts in both ventricles must involve critical channels within the septum. For hemodynamically intolerated VT, simultaneous biventricular noncontact mapping provides the only effective method of measuring this difference in ventricular breakout timing. The morphology of virtual electrograms at the brea-

### Table 2. Characteristics of VT in Each Animal and Ablation Results

<table>
<thead>
<tr>
<th>Sheep No.</th>
<th>Earliest Breakout</th>
<th>No. of VTs</th>
<th>CL of VT, ms</th>
<th>1st RFA Chamber</th>
<th>2nd RFA Chamber</th>
<th>Linear Lesions, mm</th>
<th>No. of RFA</th>
<th>Transmural Lesion (Histopathology)</th>
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<td>Septum</td>
<td>4</td>
<td>242, 254, 302, 260</td>
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<td>RV</td>
<td>20 + 22 + 18</td>
<td>22</td>
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<tr>
<td></td>
<td>Free wall</td>
<td>1</td>
<td>217</td>
<td>LV</td>
<td></td>
<td>28</td>
<td>11</td>
<td>No</td>
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<td>LV</td>
<td>RV</td>
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<td>LV</td>
<td></td>
<td>22 + 15</td>
<td>24</td>
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<td>4</td>
<td>260, 260, 247, 252</td>
<td>RV</td>
<td>LV</td>
<td>7 + 18 + 16 + 23</td>
<td>45</td>
<td>Yes</td>
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<tr>
<td>4</td>
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<td>RV</td>
<td>LV</td>
<td>24 + 34</td>
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<td>5</td>
<td>205, 257, 290, 250, 255</td>
<td>RV</td>
<td>LV</td>
<td>20 + 20 + 23 + 26 + 14</td>
<td>40</td>
<td>No</td>
</tr>
<tr>
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<td>Septum</td>
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<tr>
<td>6</td>
<td>Septum</td>
<td>1</td>
<td>254</td>
<td>LV</td>
<td></td>
<td>25 + 12</td>
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<td>No</td>
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<tr>
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<td>Free wall</td>
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<td>260</td>
<td>LV</td>
<td></td>
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<tr>
<td>7</td>
<td>Septum</td>
<td>3</td>
<td>170, 190, 182</td>
<td>RV</td>
<td>LV</td>
<td>30 + 16</td>
<td>22</td>
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**Figure 5.** Examples from 3 animals of post-mortem septal pathology, with both right and left endocardial borders. Red arrows indicate LV ablation lesion; blue arrows indicate RV ablation lesion. Normal myocardium is red; areas of scar are white. A and B. Section of the septum is in the plane of apex to base. C. Plane of the section is from anterior to posterior. All 3 panels demonstrate transmural ablation composed of overlapping right- and left-sided lesions.
kout site in the LV was not useful in determining which ventricle had the earliest breakout. This may be a consequence of the larger mass of LV myocardium, with LV breakout electrograms having sharp QS morphology in the majority of septal VT regardless of which ventricle was earliest. The RV breakout site virtual electrogram morphology was reasonably accurate at establishing whether the RV was early or late compared with the LV for septal VT.

In this study, when a deep intramural source was suspected, an ablation line was performed on the septal side with earliest breakout and extended if required. After initial ablation on 1 endocardial aspect of the septum, in 5 of the 6 sheep, different VT morphologies with a septal involvement were still inducible, with common breakout points on the opposite unablated ventricular septal surface. It may be postulated that after ablation of one endocardial septal aspect of a septal reentrant circuit, the circuit was still capable of exiting on the opposite septal endocardial surface. In this study, ablation on the opposite side of the septum rendered all septal VT morphologies noninducible.

This study suggests that transmural septal lesions may be necessary to interrupt VT with septal pathways. This study demonstrated that this is feasible with biventricular endocardial irrigated ablation targeting sites identified by noncontact mapping in both ventricles. In this series, the area of septal ablation would not have been accessible with an epicardial approach. Interestingly, in this study, VT with free wall breakouts did not require the creation of a transmural free wall lesion to render free wall VT noninducible.

Substrate maps (contact bipolar and noncontact DSM) did not reliably localize scar on the RV septal surface in this study. However, ablation perpendicular to prebreakout propagation pathways was located at scar periphery on subsequent pathological examination of the RV septum. This discrepancy probably is a result of the anatomy of the ovine RV and the density of electroanatomic mapping performed in the distal RV.

Biventricular deployment of MEAs was safe and useful in defining global septal VT activation and successful targeting of catheter ablation. The creation of transseptal lesion at the scar border zone ultimately rendered VT noninducible. The negative effect of extensive ablation along the septal scar border zones on LV function was not quantified; however, previous studies of substrate ablation with long follow-up were still inducible, with common breakout points on the opposite unablated ventricular septal surface. It may be postulated that after ablation of one endocardial septal aspect of a septal reentrant circuit, the circuit was still capable of exiting on the opposite septal endocardial surface. In this study, ablation on the opposite side of the septum rendered all septal VT morphologies noninducible.

This study suggests that transmural septal lesions may be necessary to interrupt VT with septal pathways. This study demonstrated that this is feasible with biventricular endocardial irrigated ablation targeting sites identified by noncontact mapping in both ventricles. In this series, the area of septal ablation would not have been accessible with an epicardial approach. Interestingly, in this study, VT with free wall breakouts did not require the creation of a transmural free wall lesion to render free wall VT noninducible.

The CARTO system is limited by hemodynamic stability of VT, that is, poorly tolerated VT preventing sequential point-to-point mapping. In this study, biventricular breakout sites could only be mapped using the noncontact system.

Conclusions

Biventricular scar and VT activation mapping correctly localizes critical septal VT pathways directing ablation from one or both septal endocardial aspects. Creation of a transmural septal lesion at the scar border interrupting VT exit points is highly effective at ablating septal VT.

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Disclosures

None.

References

Catheter ablation is an important therapy for recurrent ventricular tachycardia (VT) in survivors of myocardial infarction, but it is limited by anatomic complexities and intramural and epicardial arrhythmia substrate. The bilayered structure and variable thickness of the septum contribute to difficulty ablating septal VTs. We studied intraseptal VTs in a chronic infarct animal model using contact and noncontact mapping from the right and left ventricles. The simultaneous use of 2 noncontact systems allowed biventricular single-beat mapping of poorly tolerated VTs. In this model, septal VTs usually had a common intramural isthmus capable of exiting in either ventricle, with very short delay between earliest left and right ventricular activation. Initial ablation was delivered along the septum in the ventricle with earliest breakout. Failure to create a transmural septal ablation lesion at the scar resulted in persistent inducibility of septal VT and shifted earlier activation to the opposite ventricle. In the majority of animals, ablation from both sides of the septum resulted in transmural ablation that rendered septal VT noninducible. The findings illustrate difficulty of mapping and ablation for septal VT and suggest that a biventricular septal ablation strategy may be required for successful ablation of these VTs.
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