Inverse Solution Mapping of Epicardial Potentials
Quantitative Comparison With Epicardial Contact Mapping

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Background—Catheter ablation of ventricular tachycardia (VT) is still one of the most challenging procedures in cardiac electrophysiology, limited, in part, by unmappable arrhythmias that are nonsustained or poorly tolerated. Calculation of the inverse solution from body surface potential mapping (sometimes known as ECG imaging) has shown tremendous promise and can rapidly map these arrhythmias, but we lack quantitative assessment of its accuracy in humans. We compared inverse solution mapping with computed tomography–registered electroanatomic epicardial contact catheter mapping to study the resolution of this technique, the influence of myocardial scar, and the ability to map VT.

Methods and Results—For 4 patients undergoing epicardial catheter mapping and ablation of VT, 120-lead body surface potential mappings were obtained during implantable defibrillator pacing, catheter pacing from 79 epicardial sites, and induced VT. Inverse epicardial electrograms computed using individualized torso/epicardial surface geometries extracted from computed tomography images were compared with registered electroanatomic contact maps. The distance between 3D substrate mapping to study the resolution of this technique, the influence of myocardial scar, and the ability to map VT.

Conclusions—Inverse solution maps can identify sites of epicardial pacing with good accuracy, which diminishes over myocardial scar or over slowly conducting tissue. This approach can also identify epicardial VT exit sites and ventricular activation sequences. (Circ Arrhythm Electrophysiol. 2012;5:1001-1009.)

Key Words: ablation ■ electrocardiography ■ electrophysiology mapping ■ mapping ■ ventricular tachycardia

Catheter ablation of scar-related ventricular tachycardia (VT) is still one of the most challenging procedures in clinical cardiac electrophysiology. The majority of these arrhythmias are poorly hemodynamically tolerated, may be difficult to induce, and frequently transform to other tachycardias during catheter mapping. Three-dimensional (3D) substrate mapping has added substantial capability and insight into catheter ablation of VT, but the procedure is restricted by the limitations of point-by-point catheter mapping. Methods for rapidly identifying VT circuit and exit sites are needed to facilitate the procedure.

Clinical Perspective on p 1009

Body surface potential mapping (BSPM), the acquisition of electrocardiographic recordings from multiple thoracic sites, can be combined with patient-specific geometry to estimate, by inverse solution, epicardial electric events from body surface recordings. This methodology, also known as ECG imaging, elegantly described by Ghosh et al and Jia et al, has been correlated to biventricular pacing, sites of successful ablation of accessory pathways, open-chest mapping, and spontaneous ventricular ectopy in a case report and in a series of patients who subsequently underwent electrophysiological study with catheter mapping.

ECG imaging shows tremendous promise, although its spatial accuracy in humans has not yet been completely determined. We quantitatively assessed the accuracy of BSPM with inverse solution mapping to localize paced epicardial sites of activation by comparing it with simultaneously acquired computed tomography (CT)–registered 3D electroanatomic epicardial contact maps created during catheter mapping/ablation procedures for patients with VT. Then, we applied the technique to gain insight into the tachycardia circuit and exit sites.

Methods

Four consecutive consenting patients undergoing epicardial catheter mapping and ablation of VT were enrolled. In a protocol approved by the Capital Health Research Ethics Board, BSPM was performed during the clinical procedure. Patients underwent CT scanning before the procedure for correlation with electroanatomic mapping and generation
of patient-specific geometry, and surface electrodes were applied on the patient’s torso according to our previously published methodology.

**Clinical Methods**

BSPM electrodes were applied before the procedure, which was performed using standard techniques. The pericardial space was entered percutaneously and mapped using an electroanatomic nonfluoroscopic system (Carto, Biosense Webster, Inc, Diamond Bar, CA). Point-by-point epicardial and endocardial mapping was performed using an irrigated 3.5-mm-tip deflectable electrode catheter (Navistar Thermocool, Biosense Webster Inc) during sinus or paced rhythm. Pacing was performed with stable catheter position at multiple epicardial sites at minimum pacing output (<10 mA) to ensure consistent focal myocardial capture. BSPM recordings were acquired during pacing at each site. The location of each pacing site provided by the electroanatomic mapping system was noted for later off-line comparison.

**BSPM and CT**

Body surface electrodes (Foxmed, Idstein, Germany) were applied immediately before beginning the clinical procedure using our previously published schema. Consistent lead position was assured by the use of bony landmarks and prefabricated electrode strips, with fixed interelectrode spacing of 50 mm. Recordings were made using a 128-channel acquisition system, with 1000-Hz sampling rate (Mark 6, BioSemi, Amsterdam, the Netherlands). Axial CT (0.8–3 mm) was performed within 24 hours before the procedure (Siemens Sonata, Erlangen, Germany), and images were analyzed to create patient-specific torso and epicardial surfaces using commercial software (Amira 4.1, Mercury Computer Systems, Chelmsford, MA).

**Modeling and Data Processing Methods**

The bioelectric problem was modeled based on the epicardial potential approach. The linear model relating epicardial potentials ($\phi_e$) and measured body surface potentials ($\phi_b$) through the transfer matrix $A$ at every time instant can be expressed by the following equation:

$$ A\Phi_e = \Phi_b $$

(1)

Transfer coefficients of matrix $A$ were calculated using a homogeneous isotropic realistic torso model and constant interpolating functions between triangle centroids. The ill-posed system of equations was solved for epicardial potentials ($\phi_e$) using Tikhonov regularization according to:

$$ \min \|A\Phi_e - \Phi_b\|^2 + \lambda^2 \|\Phi_e\|^2 $$

(2)

where $\| \cdot \|$ denotes the norm, $\lambda$ is the regularization (smoothing) parameter, and $B$ is the regularizing operator. A second-order (Laplacian) operator was used, and the regularization parameter was determined using the L-curve method. BSPM signals were processed to interpolate leads with considerable noise or missing data to produce a single averaged ECG complex for each recorded lead. Computational routines were implemented in MATLAB (The Mathworks Inc, Natick, MA) for processing and analysis of data. MAP3D visualization software was used to display and inspect potential distributions on heart and torso surfaces.

**Data Analysis**

The electroanatomic (Carto) epicardial map was registered to the CT, and points were projected to the nearest nodes of the discretized CT epicardial surface and used as the localization gold standard. The Carto map was registered manually to the CT data (Figure 1) to permit quantitative comparison of pacing sites, with sites of earliest activation identified by the inverse solution map. As a measure of accuracy, the Euclidean (shortest 3D) distance was calculated for each recording between the earliest computed potential minimum and the actual pacing site projected on the epicardial surface. In addition, the geodesic distance (the shortest path connecting the 2 points along the surface) was estimated by solving for the shortest path on the discretized epicardial surface. For analysis of pacing site localization, the sites were grouped into categories based on anatomic substrate at the pacing location: sites were characterized as scar, scar margin, or no scar on the basis of the filtered (50–400 Hz) bipolar signal amplitude. Sites with bipolar peak-to-peak signal amplitude <1.5 mV were classified as scar, whereas sites within 10-mm Euclidean distance of a point with signal amplitude <1.5 mV were classified as scar margin. Sites also were classified by the presence or absence of delay >40 ms between stimulus and QRS onset.

Electroanatomic (Carto) pacing sites were compared with the centroid of areas of calculated early negative potentials. The centroid was identified visually within the earliest discrete area of calculated negative isopotentials by investigators blinded to the corresponding electroanatomic maps. In 3 cases, recordings also were made during pacing from the endocardial right ventricular (RV) apex using the implantable cardioverter-defibrillator lead. This site was localized easily on the CT scan, providing a readily identifiable pacing site for comparison. For 2 of these, epicardial point-by-point activation mapping was performed to qualitatively compare patterns of activation identified by contact mapping with those identified by ECG imaging. Local activation was determined from the inversely computed electrograms at the point of steepest descent (minimum dV/dt) during depolarization. Epicardial potential distributions of VTs for which an exit site was identified were computed. Epicardial isochrones were calculated as described above. Sites of early calculated potential minima were compared qualitatively with isochronal maps and sites of successful ablation.

**Statistical Analysis**

Statistical analysis was performed with the SAS package (SAS Institute Inc, Cary, NC), taking into account the relationship among the 6 groups of measurements, each characterized by delay factor and scar factor and incorporating these factors into the model. Because data are unbalanced and correlated within subjects, the generalized estimating equation approach was used (the GLIMMIX procedure of SAS), with the natural logarithm of the Euclidean distance as the outcome variable. From the GLIMMIX procedure options, within-subject analysis was chosen, with a constant correlation between all pairwise repeated measurements (referred to as compound symmetry or CS model). The significance level was $P<0.05$; post hoc analysis used Sidak-protected $P$ values for multiple pairwise comparisons. For all groups of measurements, we calculated the least-squares mean values with SE and 95% CIs for natural logarithm of Euclidean distance, as well as back-transformed geometric mean values and their 95% CIs. Differences of least-squares means of natural logarithm values (ratios of back-transformed values) were tabulated, and their significance was assessed by Sidak $P$ values, adjusted for multiple comparisons.

**Results**

Four patients, all men, age 60±20 years, with scar-related VT were included (Table 1). Inverse solution maps created from recordings during pacing at epicardial sites demonstrated an initial negative potential surrounding the pacing site. Figure 2 illustrates a typical example of both recorded body surface potentials and calculated epicardial potentials at multiple time instants after pacing at a basal inferior epicardial site. An early minimum potential correlates with the site of pacing. During early repolarization, a maximum appears in close proximity to the pacing site.

**Accuracy of Localization of Pacing Sites and Influence of Myocardial Scar**

Comparison of computed inverse solution sites of activation with registered electroanatomic maps yielded good accuracy when pacing at sites without scar and without stimulus-QRS delay. The results of pacing site localization in relation to the
The anatomic substrate are summarized in Table 2 and Figure 3. The 6 groups in Table 2 and Figure 3 are not independent, but each is defined by 2 factors: presence of delay (pacing producing no delay/delay) and presence of or proximity to scar (pacing sites with no scar/in scar margin/within scar region). The test of fixed effects showed that there is no significant interaction between these factors and that the delay factor is not statistically significant ($P=0.467$). Figure 3 graphically depicts the differences between the groups. The greatest accuracy is at sites where there is neither stimulus-QRS delay nor local scar. The presence of nearby scar significantly reduces the accuracy of the technique. The significance of differences of least-squares means of natural logarithm of Euclidean distance (ratios of back-transformed values) was assessed by $P$ values adjusted for multiple comparisons as follows: no scar versus scar margin ($P=0.014$); no scar versus scar ($P=0.013$); scar margin versus scar ($P=0.076$).

Euclidean distances $<50$ mm were closely matched by geodesic distances (distances over the curvature of the heart surface). There were no differences in estimates of surface alignment (registration of electroanatomic to CT surface) accuracy among the groups.

In the 3 cases for which maps were created during pacing from the patient’s implantable cardioverter-defibrillator, an area of positive potential was observed early over the RV apex (thought to represent endocardial to epicardial spread), followed by a potential minimum that increased in negative magnitude throughout the depolarization phase. The calculated distances between the RV apical pacing lead tips and the centroids of the areas of earliest negative potentials were 12, 25, and 24 mm, respectively, for the 3 cases. Inverse solution mapping of RV endocardial pacing is illustrated in Figure 4.

Ventricular Activation Sequence: Contact Mapping Versus Inverse Solution

Contact mapping of the epicardium during pacing from the RV apical endocardium revealed activation patterns that were highly congruent with inverse solution maps (Figure 5). The site of earliest activation was accurately identified by both techniques at the RV epicardium over a relatively large area of epicardial breakthrough. In case 3, breakthrough was at the anterior RV apex, from which activation spread radially over the RV free wall, with latest activation at the basal inferior left ventricle (LV). This pattern was nearly replicated by inverse solution mapping. The area of late activation at the basal inferior wall of the LV correlated closely with a large area of endocardial scar, which was only minimally represented on the epicardial contact map (not shown). In case 4, the site of epicardial breakthrough was identified by both techniques at the apical inferior wall of the LV. Activation spread across the inferior wall, with late activation occurring at the basal anterolateral LV. This patch of late activation was identified as being more inferiorly distributed on inverse solution maps, whereas it was more superolateral on the Carto map. The site of late activation correlated with a large basal lateral infarction scar, where late, fragmented, low-amplitude signals were recorded from the endocardium.
Localization of VT Exit Sites

VT ablation or exit sites were localized clinically for 6 VTs (Table 1), of which 2 were epicardial. VT1 was recorded from a 30-year-old man with nonischemic cardiomyopathy. Epicardial contact mapping revealed a confluent patch of posterior and lateral basal low-amplitude signals. This VT appeared to exit from the superior margin of the basal lateral scar and was ablated successfully at a site proximal to the superior scar margin (noted by asterisk in Figure 6, first row). The successful ablation site was within scar, where a late potential was recorded and where pacing resulted in stimulus-QRS delay and a morphological match for VT; near match without delay was observed pacing higher on the postero basal LV epicardium. This correlated with an area of early epicardial potential minimum on the inverse solution map. Computed epicardial isochrones suggested a basal superior origin, with propagation across the basal anterior wall, constrained by a line of slow conduction (crowding of isochrones) along the basal midanterior wall, with breakout just superior to

Table 2. Localization of Epicardial Pacing Sites: Cumulative Results for All 4 Cases

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (NS-ND)</th>
<th>Group 2 (SM-ND)</th>
<th>Group 3 (S-ND)</th>
<th>Group 4 (NS-D)</th>
<th>Group 5 (SM-D)</th>
<th>Group 6 (S-D)</th>
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<tr>
<td></td>
<td>n=17</td>
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<td>n=14</td>
<td>n=6</td>
<td>n=23</td>
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<tr>
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<td></td>
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<td></td>
<td></td>
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<tr>
<td>Euclidean distance</td>
<td>13±9</td>
<td>43±11</td>
<td>28±27</td>
<td>28±26</td>
<td>50±47</td>
<td>67±37</td>
</tr>
<tr>
<td>Median (range)</td>
<td>10 (30)</td>
<td>44 (33)</td>
<td>22 (87)</td>
<td>16 (77)</td>
<td>24 (97)</td>
<td>63 (115)</td>
</tr>
<tr>
<td>Minimum time</td>
<td>27±15</td>
<td>58±36</td>
<td>31±11</td>
<td>46±21</td>
<td>76±24</td>
<td>68±29</td>
</tr>
<tr>
<td>Alignment distance</td>
<td>3±3</td>
<td>3±2</td>
<td>4±2</td>
<td>5±4</td>
<td>3±6</td>
<td>7±5</td>
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<td>Measured</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Delay time</td>
<td>30±10</td>
<td>25±12</td>
<td>32±9</td>
<td>57±15</td>
<td>71±26</td>
<td>73±28</td>
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<tr>
<td>Bipolar signal amplitude</td>
<td>4.2±2.8</td>
<td>2.2±0.9</td>
<td>0.9±0.4</td>
<td>4.1±1.7</td>
<td>2.0±0.4</td>
<td>0.8±0.4</td>
</tr>
</tbody>
</table>

NS/SM/S indicates pacing sites with no scar/in scar margin/within scar region; D/ND, pacing producing delay/no delay; minimum time, time (ms) when the first discernible minimum appears in calculated epicardial potential distribution; Euclidean distance, 3-dimensional distance (mm) between pacing site and estimated location of that pacing site; alignment distance, distance (mm) between actual Carto point and point projected onto the epicardial surface (registration error); delay time, time (ms) from stimulation spike to the earliest QRS onset of surface ECG; bipolar signal amplitude, peak-to-peak bipolar signal amplitude (mV) measured by Carto system at pacing site during sinus rhythm.

Values presented as mean±SD unless otherwise indicated.
the site of successful ablation. VT2 was recorded in patient 2, a 70-year-old man with prior inferior myocardial infarction. Computed epicardial potential maps are shown in Figure 6, second row, at 2 time instants during ventricular activation in VT: 40 ms after QRS onset, an early minimum potential is recorded at the mid-inferolateral LV. This area of negative potentials intensifies and enlarges by mid-QRS (77 ms after QRS onset). The site of early minimum correlates approximately to the site of successful ablation (which was an isthmus site within scar, not at the breakout site). Computed isochronal maps are highly consistent with these findings, demonstrating earliest activation at the mid-inferolateral LV, followed by propagation across the inferior wall, constrained by a line of slow conduction surrounding infarct scar (represented by isochronal crowding), with breakout over the apical junction of the 2 ventricles.

**Discussion**

**Localization of Epicardial Pacing Sites**

The accuracy of the inverse solution has been reported previously using canine heart preparations in a torso-shaped tank\(^9\) and in humans with implanted pacing devices.\(^7\) It also has

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**Figure 3.** Box plot of natural logarithm of Euclidean distances between sites of pacing as identified by Carto electroanatomic maps and sites of early negative potentials identified from epicardial inverse solution maps, grouped by presence or absence of stimulus-QRS delay >40 ms and by whether pacing was over myocardial scar (signal amplitude <1.5 mV), within 1 cm of scar (scar margin), or over myocardium with normal signal amplitude. **Heavy line** indicates median, **boxes** represent interquartile ranges, and **error bars** represent range.

**Figure 4.** Right ventricular apical pacing. Top, Computed tomography–derived epicardial surface and site of right ventricular apical pacing lead. Bottom, Inferior views of calculated epicardial maps demonstrating an area of positive potentials over the site of the pacing leads (left), possibly representing endocardial to epicardial spread, which rapidly becomes an area of negative potential 6 ms after the pacing impulse. Subsequent maps showed propagation of negative amplitude away from the pacing site.
been compared in clinical cases with identify an epicardial focus \(^\text{14}\) and, more recently, clinical ventricular ectopy and origins of VT. \(^\text{15}\) We are not aware of previous studies comparing simultaneous BSPM with epicardial 3D contact mapping nor of closed-chest human studies quantitatively comparing contact mapping with inverse solution mapping. The inverse solution calculates epicardial potentials, and thus performing BSPM during epicardial catheter mapping/ablation

Figure 5. Contact electroanatomic epicardial activation isochronal maps (left) and computed inverse solution epicardial isochronal maps (right) for case 3 (top row) and case 4 (bottom row) during right ventricular (RV) endocardial pacing. Activation spreads radially from RV pacing site to left ventricular (LV) posterolateral wall. Qualitatively, similar patterns of activation are reconstructed by the inverse solution. Contact mapping was more sensitive to late, low-amplitude potentials, demonstrating later activation in the superobasal LV segment in case 4 than was detected by the inverse solution map.

Figure 6. Inverse epicardial potential maps and isochrones of activation for cases 1 and 2 during ventricular tachycardia (VT) 1 and VT2. Asterisk (*) indicates the ablation site that terminated VT or the clinically identified VT exit site. On the left are calculated epicardial isopotential maps at time instants after QRS onset, as shown, with views as noted. On the right are calculated epicardial isochronal maps of the entire QRS with views as noted. First row: VT1. An early minimum potential is seen at the mid-superolateral left ventricle; isochronal maps demonstrate propagation from the superobasal left ventricle across the anterior wall. Second row: VT2. Inferior views of potential maps at 40 ms and 77 ms after QRS onset are shown. An early inferolateral minimum potential is seen just apical to the ablation site that was within myocardial scar. Isochronal maps are consistent with myocardial breakout near this site and propagation across the inferior wall. See more detailed discussion in text.
procedures represents an ideal circumstance to estimate the accuracy of this technique to identify sites of epicardial activation. The Carto system provides excellent and reproducible positional localization and thus supplied a good gold standard against which to compare localization by inverse solution mapping. We compared epicardial pacing sites localized by Carto with computed localization using inverse solution maps generated from body surface potential maps, registering the cardiac surface mapped using Carto with the epicardial surface determined from a preprocedure CT scan, which also was used to provide patient-specific torso and cardiac geometry for performing the inverse solution. We found that epicardial pacing from sites that were not within or close to myocardial scar, in the absence of significant stimulus to QRS delay, was accurately mapped using BSPM with inverse solution computation. The mean distance between sites of earliest activation determined by Carto and those determined by the inverse solution was only 13 ± 9 mm. This accuracy is markedly reduced, however, in the presence of myocardial scar, which increased the estimated discrepancy to 28 ± 27 mm, or the presence of nearby scar, which was associated with a discrepancy of 43 ± 11 mm. The reasons for this detraction in localization accuracy are not completely clear, but there are several likely contributors. The presence of myocardial scar was not factored into the calculation of epicardial potentials by the inverse solution, and the inhomogeneity of myocardial substrate may thus significantly violate the assumptions used to calculate epicardial potentials. Pacing from within myocardial scar, even in the absence of substantial stimulus-to-QRS delay, may result in relatively early activation of myocardium, which is not immediately below the pacing catheter but rather within the ventricular wall or on the endocardial side so that the usual pattern of epicardial activation (early potential minimum with radial spread of activation) is not seen. Similarly, because scar is a 3D structure, it is likely that the scar margin identified by contact mapping of the epicardial surface does not accurately identify the full extent of myocardial scar, possibly contributing to the reduced accuracy seen when pacing near myocardial scar. Stimulus to QRS delay was defined as that which exceeded 40 ms, in keeping with clinical practice. Latency between stimulus and QRS onset is typically thought to represent activation of a channel or isthmus, usually within scar, which is too small to generate a significant representation on the surface ECG. The duration of the delay is determined by the distance from the pacing site to a larger volume of myocardium (the breakout site), which generates the earliest portion of the QRS, as well as the conduction velocity within the isthmus. Sites with delay may exit close to the pacing site if local conduction velocity before breakout is low but may also exit remotely. Indeed, this was reflected in our data, in which the presence of stimulus-to-QRS delay increased the variability of distances assessed (Figure 3). It is interesting to note that we observed clustering of calculated early potential minima sites when pacing within scar or near scar margin in the presence of stimulus-QRS delay. In case 1, the early minima clustered at the high anterobasal LV (5 sites) and high basal lateral segments (6 sites), possibly correlating with exit from the margins of scar that were mapped to this area. In case 2, clustering occurred at the midinferior LV (7 sites), correlating with the basal inferior scar. In case 3, clustering occurred on the high basal lateral LV (5 sites) and, in case 4, 3 disparate pacing sites within the inferior scar resulted in calculated early potential minima clustered at the midinferior LV at the margin of scar. It is possible that this clustering represents activation of myocardial isthmuses with common exits at the scar margin, although this cannot be confirmed from the available data. It is intriguing, however, that sites of clustering of calculated early activation correlate moderately well with sites where VT was ablated successfully.

Ventricular Activation Sequence: Contact Mapping Versus Inverse Solution Qualitative comparison of the activation sequence determined by point-by-point contact mapping with that determined by inverse solution mapping yielded similar patterns, including the demonstration of areas of slow conduction or functional block where isochronal crowding was observed. Because of the complexity of the analysis and the small sample size, we did not attempt quantitative comparisons. We used the minimum dV/dt of the calculated epicardial potential to identify local activation. This has been used in unipolar contact mapping and is supported by experimental and theoretical studies. The derivative calculated from a few successive samples is vulnerable to measurement noise in body surface potentials or to slight changes in the regularization parameter that controls the amount of smoothing in the inverse calculation. This problem was mitigated in part by spatial smoothing of inverse electrograms using moving average spline interpolation. Rapid activation over large regions and lines of abrupt jumps in the calculated activation time, suggesting lines of conduction delay/block, also have been observed in figures published by other investigators. Differences in overall activation time between contact maps and calculated epicardial activation maps were observed (Figure 5). These discrepancies may be due, in part, to limitations of the inverse solution method used, which can produce only unipolar-like electrograms, which inherently have reduced ability to resolve changes in activation time with respect to neighboring sites and which contain superposition of both near- and far-field potentials (epicardial and transmural contributions) in comparison with contact bipolar recordings. Not surprisingly, contact mapping had better ability to resolve low-amplitude late potentials, seen in Figure 5, case 4, at the high posterobasal LV.

Localization of VT Exit Sites This study attempts to correlate sites of early activation during epicardial VTs determined by BSPM and inverse solution calculation, with sites of myocardial breakout or critical isthmuses identified during catheter ablation procedures. This comparison is necessarily limited by several important factors: the site of successful ablation may be remote from the site of epicardial exit; diastolic components of VT are not mappable using this methodology; and myocardial scar is 3D but is mapped on the endocardial and epicardial surfaces only using catheter techniques. Despite these challenges, BSPM with inverse solution mapping approximately colocalized the site of earliest epicardial activation with the site of successful ablation.
ablation in the 2 VTs that were mapped to the epicardium. The exact activation sequence during VT was not mapped using contact catheters because of hemodynamic instability or for clinical expediency; thus, the location of VT exit was inferred and not necessarily precisely located for all cases. Computed isochronal maps of VT showed earliest activation in the region at or close to the site where the early potential minimum was identified. Similar to the recordings during apical RV pacing, regions of isochronal crowding were observed, suggesting sites of functional block. Not surprisingly, sites of conduction block were seen at similar locations during both RV apical pacing and VT in cases 3 and 4 (data not shown). Our findings confirm prior modeling studies performed in canines, as well as human studies, which have demonstrated the ability of this technique to delineate cardiac activation.

Study Limitations
Geometric inaccuracies in the representation of the heart surface have been identified as contributing the most substantial errors to estimates of epicardial potentials. We attempted to minimize these inaccuracies by constructing customized torso and cardiac geometries for each patient from CT scans obtained the day before the procedure and with the use of the L-curve method for identification of a regularization parameter, which has been thought to be more robust in the presence of geometric errors. It is possible that differences in body position, intravascular volume, or other factors may have changed the geometry between the imaging and mapping studies. Similarly, we were unable to control error introduced by respiration (which may change both thoracic conductance and relative cardiothoracic position) or other changes in patient position during the procedure, nor was it possible to control for error introduced by cardiac motion during ventricular activation, particularly during VT. These potential inaccuracies may have affected both the inverse solution map and the gold standard electroanatomic map, artificially increasing discrepancies between the 2. The challenges of epicardial contact mapping include the anatomic map, artificially increasing discrepancies between both the inverse solution map and the gold standard electroanatomic map. This study provides human, closed-chest validation that inverse solution maps derived from body surface potential maps can identify sites of epicardial pacing with good accuracy, which diminishes when pacing is performed over myocardial scar or over a slowly conducting isthmus. Cardiac magnetic resonance imaging was precluded by the presence of implantable cardioverter-defibrillators, and we did not endeavor to incorporate scar location data from echocardiography or other imaging modalities.

Conclusions
This study provides human, closed-chest validation that inverse solution maps derived from body surface potential maps can identify sites of epicardial pacing with good accuracy, which diminishes when pacing is performed over myocardial scar or over a slowly conducting isthmus. Cardiac magnetic resonance imaging was precluded by the presence of implantable cardioverter-defibrillators, and we did not endeavor to incorporate scar location data from echocardiography or other imaging modalities.

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References
Inverse solution mapping is a technique for inferring cardiac activation from body surface potential maps (multisite ECG recordings from the thoracic surface). This study provides the first human, closed-chest, quantitative assessment of the accuracy and potential limitations of this methodology by recording body surface potential maps during contact catheter mapping of the epicardium in patients undergoing catheter ablation of ventricular tachycardia. The calculated sites of ventricular activation were compared with 3-dimensional electroanatomic maps of the actual sites, correlated by registering both images with a computed tomography scan of the heart and thorax. Inverse solution maps identified sites of epicardial activation within 13±9 mm of the site as identified by catheter mapping over normal myocardium, but the presence of local scar adversely influenced this accuracy. Mapping of ventricular tachycardia and paced activation sequences is also shown to be feasible. These promising results suggest that inverse solution mapping can permit noninvasive assessment of cardiac activation, with potential applications for catheter ablation of ventricular tachycardia, targeted permanent pacing, and other interventional and diagnostic procedures.
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