Electromechanical Mapping for Detection of Myocardial Viability in Patients With Ischemic Cardiomyopathy

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Background—We evaluated the ability of electromechanical mapping of the left ventricle to distinguish between nonviable and viable myocardium in patients with ischemic cardiomyopathy.

Methods and Results—Unipolar voltage amplitudes and local endocardial shortening were measured in 31 patients (mean±SD age, 62±8 years) with ischemic cardiomyopathy (ejection fraction, 30±9%). Dysfunctional regions, identified by 3D echocardiography, were characterized as nonviable when PET revealed matched reduction of perfusion and metabolism and as viable when perfusion was reduced or normal and metabolism was preserved. Mean unipolar voltage amplitudes and local shortening differed among normal, nonviable, and viable dysfunctional segments. Coefficient of variation for local shortening exceeded differences between groups and did not allow distinction between normal and dysfunctional myocardium. Optimum nominal discriminatory unipolar voltage amplitude between nonviable and viable dysfunctional myocardium was 6.5 mV, but we observed a great overlap between groups. Individual cutoff levels calculated as a percentage of electrical activity in normal segments were more accurate in the detection of viable dysfunctional myocardium than a general nominal cutoff level. The optimum normalized discriminatory value was 68%. Sensitivity and specificity were 78% for the normalized discriminatory value compared with 69% for the nominal value (P<0.02).

Conclusions—Endocardial ECG amplitudes in patients with ischemic cardiomyopathy display a wide scatter, complicating the establishment of exact nominal values that allow distinction between viable and nonviable areas. Individual normalization of unipolar voltage amplitudes improves diagnostic accuracy. Electroanatomic mapping may enable identification of myocardial viability. (Circulation. 2001;103:1631-1637.)

Key Words: electrocardiography ■ electrophysiology ■ endocardium ■ hibernation ■ infarction

A new catheter-based mapping system, NOGA (Biosense-Webster, Haifa, Israel), uses low-intensity magnetic field energy to determine the location of sensor-tipped catheter electrodes in the left ventricle (LV) and yields information about local myocardial contractility.1–3 Simultaneous registration of the amplitude of endocardial electrical signals, which correlate inversely to the extent of myocardial ischemia,4–6 allows construction of real-time 3D electromechanical maps without x-ray fluoroscopy. Preliminary studies indicate that LV electromechanical mapping can be used to distinguish between infarcted and ischemic but still viable myocardium.7,8 However, discriminatory values for contractile and electrical function allowing separation between viable and nonviable myocardium in patients with an impaired LV resulting from ischemic cardiomyopathy have not been established.

We evaluated whether LV electromechanical mapping data can distinguish between viable and nonviable myocardial dysfunction in patients with severe LV dysfunction caused by ischemic heart disease by comparison with 3D echocardiography and perfusion and metabolism data obtained with PET.

Methods

Patients
We studied 31 patients (mean±SD age, 62±8 years; 27 men) with ischemic heart disease verified by significant coronary artery obstruction by coronary angiography and an ejection fraction <45%. Exclusion criteria included peripheral vascular disease, aortic stenosis, unstable ischemic syndrome, atrial fibrillation, and LV thrombus. Informed consent was obtained from all patients before any diagnostic procedure. The study was approved by the local ethics committee.

Study Protocol
The patients underwent 3D echocardiography to characterize wall motion as either normal or dysfunctional. Subsequently, [13N]ammo-
nia and \([^{13}\text{N}]\text{NH}_3\) PET were used to determine whether dysfunctional regions were nonviable or viable.

We studied reproducibility of electromechanical variables in a subgroup of 10 men (age, 59 ± 12 years; ejection fraction, 30 ± 12%). After finishing the electromechanical map, we sampled another 10 (true) duplicate measurements from representative parts of the myocardium identified as normal or dysfunctional by the color code from the 3D map. Each point was registered twice with the same catheter tip position. Furthermore, 10 pairs of points within a 0.5-mm distance from each other were identified randomly scattered over the myocardium (simulated duplicate measurements).

**Echocardiography**

We performed transthoracic 3D echocardiography with tissue harmonic imaging using a 2.5-MHz transducer mounted in a handheld rotation device (Vingmed System Five, GE-Vingmed Ultrasound). Coaxial rotation from the apical position was obtained with ECG-triggered recording in which each R wave initiated a 30° stepwise rotation of the transducer. A total of six 30° rotations covered the entire LV. The resulting 2D images were stored as digital cine loops in a computer for offline analysis and generation of a 3D image (Echo-Pac software, GE-Vingmed Ultrasound). From the 3D image, regional wall motion scoring was evaluated semiquantitatively as normal or dysfunctional with the 9-segment model.\(^9\) LV volumes were calculated with all 6 sections according to a volume estimation algorithm based on calculation of reconstructed polyhedrons as recently described.\(^10\) The first image after the R wave of the ECG was defined as end diastole; the smallest area just before mitral valve opening was defined as end systole.

**PET Studies**

The PET studies were conducted under a hyperinsulinemic, euglycemic (5 mmol/L) clamp (Actrapid, Novo Nordisk) 40 mU · min\(^{-1}\) · m\(^{-2}\) body surface area starting 1.5 hours before the FDG scan. All subjects were scanned in 2D with an ECAT EXACT HR whole-body scanner (CTI/Siemens) with an axial field of view of 15 cm. A 9-minute emission scan was made 10 minutes after injection of 740 MBq \([^{18}\text{F}]\text{FDG}\). A 15-minute transmission scan was then acquired. Fifty minutes after injection of \([^{13}\text{N}]\text{NH}_3\), 370 MBq FDG was injected, and 50 minutes later, a static 10-minute frame was acquired. The emission scans were corrected for scatter and attenuation. The images were reconstructed using back-projected filtering and a Hann filter with a cutoff frequency of 0.2 sinogram element, resulting in a spatial resolution of 1.9 mm. The images were resliced into 12 equally spaced short-axis images from apex to the aortic outlet. Circular regions of interest (ROIs) were defined on each short-axis plane. The ROIs were 4 pixels (7 mm) wide. Nine ROIs were defined to match the echocardiographic and electromechanical ROIs. The \([^{13}\text{N}]\text{NH}_3\) images were scaled so that the average activity was 1 in the ROI with maximal average activity. The FDG image was scaled to give the average value of 1 in this ROI. Segments with normal contractility on echocardiography were classified as normal. Segments with abnormal contractility on echocardiography were classified as nonviable when PET showed preserved metabolism (scaled average \([^{13}\text{N}]\text{NH}_3\) < 0.8 and scaled average FDG > 0.7) and as viable when PET showed preserved metabolism (scaled average FDG > 0.7) in the presence of preserved (scaled average \([^{13}\text{N}]\text{NH}_3\) ≥ 0.8) or reduced flow (scaled average \([^{13}\text{N}]\text{NH}_3\) < 0.8).

**Mapping System**

The electromechanical mapping system has been described in detail previously.\(^1\)\(^2\) The main components are (1) a triangular location pad with 3 coils placed under the patient table generating ultralow magnetic field energy, (2) a stationary reference catheter with a small magnetic field sensor located at the body surface, (3) a navigation sensor mapping catheter (7F) with deflectable tip and electrodes providing endocardial signals, and (4) a Silicon Graphics workstation for data processing and 3D reconstruction.

**Electromechanical Data**

From the mechanical data, regional contractility was obtained by the use of the linear endocardial local shortening (LS) formula: \(LS = \frac{[L_{p(ES)}/L_{ED} - L_{p(ED)}/L_{ED}]}{L_{ED}} \times 100\), where \(L_{p}(t)\) denotes the weighted average LS of a point (p) relative to all its endocardial neighboring points, and \(L_{ED} \) and \(L_{p(ED)}\) are the distances of an index point from its neighbors at end diastole and end systole, respectively. The \(L_{p}(t)\) value is a ratio that becomes smaller or even negative if regional contractility is reduced or becomes paradoxical. From the electrical data, a color-coded unipolar voltage map was generated. A fixed polar reference coordinate map was defined with anatomic reference points acquired at end diastole to match the echocardiographic and PET ROIs. The center of mass of the reconstructed LV chamber was automatically calculated by the system from the set of endocardial points sampled. The long axis of the LV was defined as the line connecting the apex (the most distal point from the center of mass) and the center of mass. The long axis was divided into 3 segments: apex, midventricle, and base, consisting of 20%, 40%, and 40% of the long axis, respectively. The midventricular and base segments were further divided into 4 regions: anterior, septal, inferoposterior, and lateral. Consequently, endocardial sites were divided into a total of 9 segments for comparative analysis with echocardiographic and PET imaging data.

End-diastolic and end-systolic volumes were calculated as the maximal and minimal volumes throughout the cardiac cycle.\(^1\)\(^3\)

**Statistical Analysis**

Data are presented as mean ± SD unless otherwise indicated. Means of nominal values (voltage and LS) were compared between myocardial segments classified as normal, nonviable, or viable by ANOVA. When appropriate, a post hoc pair-wise comparison was made with the Bonferroni modification. Normalized voltage amplitudes were obtained as the percentage of the mean value of all normal segments in each patient. To estimate the contribution of between- and within-subject variation to total variation, we used nested 2-way ANOVA. The components of variation were compared with an F test. Data were expressed as coefficients of variation (CVs), calculated by dividing the square root of the variances with the great means. The critical difference (CD) was calculated as follows:

\[
CD = Z \times \frac{\sigma}{\sqrt{n}} \times CV_{tot}
\]

where \(CV_{tot}\) denotes the total variation of the measurement in terms of CV and Z is the Z score, the number of SDs appropriate for the level of probability selected for significance.

The diagnostic performance of nominal and normalized unipolar voltage amplitudes for discrimination between nonviable and viable myocardial dysfunction was analyzed by receiver-operating charac-
teristic (ROC) curve analysis comparing nominal and normalized voltage amplitudes to distinguish between nonviable and viable myocardial dysfunction. Correlation was sought through Spearman’s test, and comparisons between 2 groups were made with a t test. A value of \( P < 0.05 \) was considered statistically significant.

**Results**

**Patient Characteristics**

Twenty-four patients (73%) had previous myocardial infarction, and 22 (67%) received treatment for congestive heart failure. Four patients (12%) had 1-vessel disease, 6 (18%) had 2-vessel disease, and the remaining 23 (70%) had 3-vessel disease. Ejection fraction was 30±9%, and LV end-diastolic pressure was 18±8 mm Hg. Normal, nonviable, and viable tissue was equally distributed between the segments except the apex; in the apex, 65% of tissue had nonviable dysfunction, 23% had viable dysfunction, and only 12% had normal function (Table 1).

End-diastolic volume was 192±68 mL by NOGA and 224±64 mL by echocardiography (\( P = 0.13 \)). End-systolic volume was also similar by NOGA and by echocardiography (147±66 and 154±60 mL, \( P = 0.49 \)).

**TABLE 1. Regional Values for LS, Nominal Unipolar Voltage Amplitudes, and Normalized Voltage Amplitudes in Nine Segments of the Heart Identified as Normal, Nonviable, or Viable**

<table>
<thead>
<tr>
<th></th>
<th>Apex</th>
<th>Septum, Middle</th>
<th>Septum, Basal</th>
<th>Anterior, Middle</th>
<th>Anterior, Basal</th>
<th>Lateral, Middle</th>
<th>Lateral, Basal</th>
<th>Inferior, Middle</th>
<th>Inferior, Basal</th>
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<tbody>
<tr>
<td>Normal</td>
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<tr>
<td>LS, %</td>
<td>9.4 ± 2.7</td>
<td>11.7 ± 4.4</td>
<td>11.0 ± 6.7</td>
<td>11.6 ± 5.5</td>
<td>13.9 ± 7.7</td>
<td>9.2 ± 3.6</td>
<td>8.6 ± 3.8</td>
<td>8.1 ± 2.7</td>
<td>9.7 ± 4.2</td>
</tr>
<tr>
<td>Nominal unipolar voltage amplitude, mV</td>
<td>11.8 ± 2.3</td>
<td>9.2 ± 1.9</td>
<td>9.8 ± 2.3</td>
<td>12.4 ± 2.6</td>
<td>12.1 ± 4.1</td>
<td>10.6 ± 3.5</td>
<td>10.4 ± 3.8</td>
<td>12.8 ± 4.4</td>
<td>9.8 ± 3.6</td>
</tr>
<tr>
<td>Normalized unipolar voltage amplitude, %</td>
<td>97 ± 14</td>
<td>88 ± 17</td>
<td>98 ± 21</td>
<td>104 ± 14</td>
<td>112 ± 22</td>
<td>93 ± 18</td>
<td>100 ± 18</td>
<td>112 ± 28</td>
<td>93 ± 23</td>
</tr>
<tr>
<td>Segments, n</td>
<td>4</td>
<td>8</td>
<td>10</td>
<td>7</td>
<td>11</td>
<td>10</td>
<td>18</td>
<td>12</td>
<td>12</td>
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<tr>
<td>Nonviable</td>
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<tr>
<td>LS, %</td>
<td>2.6 ± 3.6</td>
<td>3.1 ± 3.4</td>
<td>3.4 ± 4.1</td>
<td>3.5 ± 3.4</td>
<td>4.4 ± 3.0</td>
<td>1.1 ± 3.5</td>
<td>4.0 ± 4.7</td>
<td>3.8 ± 4.7</td>
<td>3.8 ± 4.6</td>
</tr>
<tr>
<td>Nominal unipolar voltage amplitude, mV</td>
<td>5.0 ± 2.0</td>
<td>5.1 ± 2.1</td>
<td>4.2 ± 2.3</td>
<td>5.5 ± 2.3</td>
<td>4.5 ± 2.7</td>
<td>5.1 ± 1.7</td>
<td>4.8 ± 0.5</td>
<td>4.4 ± 2.5</td>
<td>4.8 ± 1.9</td>
</tr>
<tr>
<td>Normalized unipolar voltage amplitude, %</td>
<td>53 ± 20</td>
<td>59 ± 18</td>
<td>40 ± 20</td>
<td>61 ± 18</td>
<td>54 ± 28</td>
<td>58 ± 17</td>
<td>44 ± 20</td>
<td>54 ± 18</td>
<td>48 ± 13</td>
</tr>
<tr>
<td>Segments, n</td>
<td>20</td>
<td>13</td>
<td>11</td>
<td>14</td>
<td>6</td>
<td>6</td>
<td>2</td>
<td>14</td>
<td>12</td>
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<td>Viable</td>
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<tr>
<td>LS, %</td>
<td>8.2 ± 4.4</td>
<td>5.5 ± 3.0</td>
<td>3.1 ± 2.4</td>
<td>6.2 ± 4.1</td>
<td>6.4 ± 3.3</td>
<td>5.9 ± 5.1</td>
<td>6.1 ± 3.1</td>
<td>3.5 ± 2.6</td>
<td>5.2 ± 4.0</td>
</tr>
<tr>
<td>Nominal unipolar voltage amplitude, mV</td>
<td>8.4 ± 3.3</td>
<td>7.9 ± 3.3</td>
<td>6.6 ± 1.4</td>
<td>8.4 ± 1.9</td>
<td>8.3 ± 2.4</td>
<td>9.0 ± 4.3</td>
<td>7.5 ± 2.6</td>
<td>10.3 ± 4.2</td>
<td>6.6 ± 2.8</td>
</tr>
<tr>
<td>Normalized unipolar voltage amplitude, %</td>
<td>94 ± 25</td>
<td>81 ± 26</td>
<td>71 ± 22</td>
<td>89 ± 24</td>
<td>88 ± 27</td>
<td>103 ± 42</td>
<td>76 ± 27</td>
<td>102 ± 47</td>
<td>61 ± 11</td>
</tr>
<tr>
<td>Segments, n</td>
<td>7</td>
<td>10</td>
<td>9</td>
<td>10</td>
<td>9</td>
<td>15</td>
<td>11</td>
<td>5</td>
<td>6</td>
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</table>
Procedural Characteristics
We sampled 73±19 points, of which 56±14 points fulfilled stability criteria for construction of the LV map. Of 279 myocardial segments, 272 were available for comparative analysis. In 7 segments, no definite interpretation could be made because too few (<3) points were sampled during the mapping procedure. Mapping time was 43±11 minutes.

Local Contractile Function
Figure 2 shows contractile function, LS, in segments with normal function and in dysfunctional segments classified as nonviable or viable. On average, normal segments had higher values (10.1±5%) than dysfunctional segments. Nonviable segments had lower values (3.2±3.8%) than viable segments (5.6±3.9%), but we found considerable overlap between normal and dysfunctional segments. However, visual inspection of the color-coded maps helped us identify regions with abnormal contractility (Figure 1). We found no regional differences in LS within normal, nonviable, or viable segments (Table 1).

Analysis of reproducibility revealed CVs of 108% between true duplicate measurements and 430% between simulated duplicated measurements.

Local Electrical Function
Figure 3 shows electrical function by unipolar voltage amplitudes. Normal segments had higher voltage amplitudes (10.9±3.7 mV) than nonviable (4.8±2.1 mV) and viable (8.1±3.1 mV) dysfunctional segments. On average, viable segments also had higher voltages than nonviable segments, but we noticed considerable overlap between these 2 groups. Optimum nominal discriminatory value between nonviable and viable dysfunctional myocardium was 6.5 mV at a sensitivity and specificity of 69% (Figure 4).

Analysis of reproducibility revealed a CV of 5.8% between true duplicate measurements and 26.9% between simulated duplicate measurements. The corresponding critical difference required for 95% significance of difference between 2 measurements of unipolar voltage was 2.1 and 5.9 mV, respectively.

Between-patient variability was the main component responsible for the large variability (between-patient component of variability, 17.9; within-patient variability, 9.6%; F=41.8; P<0.01). Consequently, each patient had his own individual level for electrical activity (Figure 5). We identified a statistically significant correlation between the number

Figure 2. Linear LS in segments with normal function (n=92), nonviable dysfunction (n=98), and viable dysfunction (n=82).

Figure 3. Unipolar voltage amplitudes in segments with normal function (n=92), nonviable dysfunction (n=98), and viable dysfunction (n=82).

Figure 4. Use of electroanatomic mapping with nominal voltage amplitudes to predict probability of viable myocardial dysfunction. Optimal discriminatory value between nonviable and viable myocardial dysfunction is 6.5 mV when sensitivity and specificity are 69%.

Figure 5. Individual unipolar voltage amplitudes expressed as mean value of all normal, nonviable, and viable dysfunctional segments in each patient.
of nonviable segments and average voltage amplitude in normal segments ($r=0.55$, $P<0.01$).

Normalized electrical unipolar amplitudes are shown in Figure 6. Optimum discriminatory value between nonviable and viable dysfunctional myocardium was 68% of the values observed in normal segments (Figure 7). Sensitivity and specificity were significantly higher than the sensitivity and specificity obtained with nominal discriminatory values (78% versus 69%, $P<0.02$; Figure 8). Positive and negative predictive values were also higher with normalized compared with nominal discriminatory values (Table 2). Normalized data could not be obtained in 5 patients (16%) because we were unable to identify segments with normal contraction.

We found no regional differences in nominal and normalized voltage amplitudes within normal, nonviable, or viable segments (Table 1).

![Figure 6](image1.png)

**Figure 6.** Normalized voltage amplitudes in segments with normal function and nonviable and viable dysfunction. Mean value of normal segments in each patient was set at 100%. Normalized voltage amplitudes in segments with nonviable ($n=72$) and viable ($n=60$) dysfunction were calculated as percentage of mean nominal value in normal segments, and results in nonviable and viable dysfunctional segments are given as mean value of these segments in each patient.

![Figure 7](image2.png)

**Figure 7.** Use of electroanatomic mapping with normalized voltage amplitudes to predict probability of viable myocardial dysfunction. Optimal discriminatory value between nonviable and viable myocardial dysfunction is 68% when sensitivity and specificity are 78%.

![Figure 8](image3.png)

**Figure 8.** ROC analysis representing ability of nominal (---; area under ROC curve, 0.819; SE, 0.035) and normalized (—; area under ROC curve, 0.886; SE, 0.027) voltage amplitudes to distinguish between nonviable and viable myocardial dysfunction, $P=0.02$ for paired comparison of areas under ROC curves.

### Procedural Complications

One patient had a hemorrhagic pericardial effusion in relation to the mapping procedure and was treated with pericardiocentesis. The patient suffered no permanent injury.

### Discussion

The results of the present study confirm that mechanical mapping of the LV enables reconstruction of LV anatomy. Identification of dysfunctional regions requires help from qualitative visual inspection of the color-coded mechanical maps. Nominal assessment of local contractile function with LS alone does not allow exact distinction between normal and dysfunctional myocardium in enlarged LVs with impaired contractility. Electrical mapping of LV identifies reduced electrical activity in dysfunctional myocardium. Because of a large overlap between nonviable and viable dysfunction, an exact nominal value that allows distinction between viable and nonviable myocardium is not very accurate. Normalization of electrical activity delineates a way to improve electroanatomic mapping for detection of viable myocardial dysfunction. Unipolar voltages exceeding 68% of the magnitude found in segments with preserved contractile function appear to identify viability in dysfunctional segments.

### Local Contractile Function

Calculation of LS is based on an algorithm creating a linear LS map. LS describes the shortening of the length of a line joining any 2 points in the map. All the points surrounding a particular point, p, are used in the calculation. A weighting function takes into account the density of points surrounding p, the LV volume, and the distance of each point from p. The

<table>
<thead>
<tr>
<th>TABLE 2. Diagnostic Accuracy of Normalized and Nominal Unipolar Voltage Amplitudes</th>
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<tr>
<td></td>
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<tr>
<td>Voltage Amplitude</td>
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<tr>
<td>Positive predictive value</td>
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<td>Negative predictive value</td>
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motion of each endocardial point is a summary of a complex series of motion describing that the distance between neighboring endocardial points in a normally contracting region decreases during systole as a result of segmental shortening of the contracting muscle but does not in noncontracting segments. Because the CV on duplicate measurements was very high, our results show that in its present form the algorithm is not optimal for calculation of local contractile function in enlarged LVs with globally impaired function. In contrast to the findings in patients with angina and specified regions of the LV with viable or nonviable ischemia causing local dysfunction but preserved global LV function. The difference may reflect inherent limitations of the algorithm for calculation of LS because the points used in the calculation are not independent variables. Furthermore, inclusion of the enlarged LV volume in the calculation can affect accuracy of the LS estimate. Even so, visual qualitative inspection of the color-coded mechanical maps appears to be useful for identification of dysfunctional regions because a general reduction in LS in anatomically well-defined regions separates dysfunctional areas from normally contracting areas despite a wide scatter of LS values.

**Electrical Function in Dysfunctional Myocardium**

The low CV of duplicate unipolar voltage measurements and the corresponding critical value of only 2.1 mV make the electroanatomic measurement suitable for detection of viability. However, unipolar voltage amplitudes display a wide scatter even in segment with preserved contractile function. Most of the total variation on unipolar voltage measurements is explained by the between-subject variation. Myocardial infarction and subsequent remodeling create altered electrophysiological properties. Unipolar voltage has a significant far-field component. Because we could demonstrate a correlation between the amount of nonviable dysfunctional tissue and the voltage amplitude in normal segments, we suggest that individual differences depending on heart size, wall thickness, and extent of ischemic and infarcted tissue account for the wide scatter.

The large overlap between segments with normal function and segments with nonviable and viable dysfunction makes it impossible to identify exact nominal discriminatory values for each specific condition. Our data show that individual cutoff levels of electrical activity are more accurate for detection of viable dysfunctional myocardium than a general nominal cutoff level. Depending on the threshold chosen for viability, sensitivity and specificity vary. A low threshold ascertains a high sensitivity so that almost all patients with viable dysfunctional myocardium are identified. A high threshold secures a better specificity so that false-positive results are avoided.

Dysfunctional myocardium most frequently comprises a mixture of fibrous tissue and viable myocardium. Electrical signals from chronically dysfunctional myocardium decrease in proportion to the amount of fibrous tissue. Similarly, there is a significant correlation between severity of tissue fibrosis and mechanical recovery. The threshold amount of fibrosis that differentiates myocardium with postoperative improvement from that without is 35%, implicating that ≥65% viable myocytes are necessary to ensure beneficial effect on contractile function. We suggest that our observation that a 68% preservation of electrical activity predicts functional recovery merely reflects the presence of viable myocytes in that order of magnitude.

**Clinical Implications**

At present, myocardial viability in patients with LV dysfunction is assessed by noninvasive myocardial imaging modalities such as nuclear scintigraphy, PET, and stress echocardiography. Electroanatomic mapping offers online detection of myocardial viability in the catheterization laboratory. Despite its invasive nature, it appears to be associated with few but potentially serious complications. Mapping time is modest. The advantage is that it may enable detection of myocardial viability of dysfunctional myocardium in immediate continuation of coronary angiography.

Until now, electromechanical mapping has been evaluated mostly in patients with ischemic heart disease and preserved global LV function. The technique is required only in patients with impaired global LV function resulting from regional myocardial dysfunction. Although assessment of local mechanical dysfunction may not be optimal in these patients, the system enables precise anatomical reconstruction of the LV. Its combination with measurement of local endocardial ECG amplitudes enables the construction of electroanatomic maps that appear to identify viable myocardial regions as accurately as stress echocardiography and SPECT. The technology may therefore become a single determinant of viability.

**Study Limitations**

The main limitation of the present study is the lack of follow-up to clarify whether segments classified as nonviable or viable are truly nonviable or viable after revascularization. We chose comparison with PET for this investigation because measurement of perfusion and myocardial FDG uptake by PET is considered the gold standard for assessment of myocardial viability in patients with ischemic heart disease. A limitation created by the use of normalized values for assessment of electrical activity is that a few patients may not have segments with preserved contractility; therefore, normalization is not possible.

Despite our efforts to define the boundaries of the LV for comparative analysis of mapping, echocardiography, and PET, we cannot be sure that we have identified completely identical anatomic areas by the 3 methods. The overall concordance between LV volumes determined by mapping and echocardiography indicates that the alignment is reasonable.

**Conclusions**

Electroanatomic mapping may enable identification of myocardial viability. However, endocardial ECG amplitudes display wide scatter, complicating the establishment of exact nominal values that allow distinction between viable and nonviable areas. Individual normalization of unipolar voltage amplitudes improves diagnostic accuracy of electroanatomic mapping for detection of viable dysfunctional myocardium.
Acknowledgments

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References

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