In treating patients with coronary artery disease, it is essential to determine the extent of myocardial damage and viability to determine which patients would benefit most from revascularization procedures.\textsuperscript{1,2} Currently used noninvasive diagnostic tests to assess the extent of myocardial necrosis (eg, nuclear imaging studies or stress echocardiography) are based on assessment of myocardial blood flow and function and/or metabolism and are performed separately from the revascularization procedure.\textsuperscript{1,2} A new 3-dimensional cardiac mapping procedure (Bio-sense) offers a unique mode of assessing myocardial function by obtaining and integrating electrical and mechanical signals from the left ventricular (LV) endocardial surface using a catheter-based navigational system.\textsuperscript{3-5} The information derived by this LV mapping system regarding cardiac mechanics and electrical properties has recently been validated in animal models.\textsuperscript{3-5} Quantitative in vivo assessment of these electromechanical signals might reflect the status of myocardial viability, because myocardial necrosis is expected to cause a decrease in both electrical and mechanical functions.

The purpose of this study is to provide preliminary animal and clinical data to support the use of such integrated electromechanical signals in distinguishing infarcted from healthy myocardial regions. The distinction between normal and infarcted myocardial regions was studied in animals and patients with prior myocardial infarction (MI) and in control subjects.

Methods
Electromechanical Mapping System
The mapping and navigation system comprises a miniature passive magnetic field sensor, an external ultralow magnetic field emitter (location pad), and a processing unit (Biosense).\textsuperscript{3-5} The catheter tip incorporates standard electrodes that allow recording of unipolar (UP) or bipolar (BP) signals and the location sensor. Signals received by the sensor are transmitted along the catheter shaft to the main processing unit. The location pad is fixed beneath the operating table (location pad), and a processing unit (Biosense). The mapping and navigation system comprises a miniature passive magnetic field sensor, an external ultralow magnetic field emitter (location pad), and a processing unit (Biosense).\textsuperscript{3-5} The catheter tip incorporates standard electrodes that allow recording of unipolar (UP) or bipolar (BP) signals and the location sensor. Signals received by the sensor are transmitted along the catheter shaft to the main processing unit. The location pad is fixed beneath the operating table (location pad), and a processing unit (Biosense). The mapping and navigation system comprises a miniature passive magnetic field sensor, an external ultralow magnetic field emitter (location pad), and a processing unit (Biosense).\textsuperscript{3-5} The catheter tip incorporates standard electrodes that allow recording of unipolar (UP) or bipolar (BP) signals and the location sensor. Signals received by the sensor are transmitted along the catheter shaft to the main processing unit.
information necessary to resolve the location and orientation of the sensor in 6 degrees of freedom. The locator pad includes 3 coils, each of which generates a magnetic field that decays as a function of the distance from the coil. The sensor measures the strength of the magnetic field, thus enabling determination of the distance from each of its sources. These distances determine the radii of theoretical spheres around each coil. The intersection of these spheres determines the location of the sensor in space.

**Electromechanical Mapping Study**

The mapping catheter was introduced through an 8F or 9F femoral sheath and placed in the left ventricle. Another reference catheter, also with a tip sensor, was taped securely to the patient’s or animal’s back. The 95% upper confidence limits of location resolution using an external location reference is <1.1 mm. The location of the mapping catheter was gated to end diastole and recorded relative to the location of the fixed reference catheter at that time, thus compensating for subject or cardiac motion. As the catheter tip is moved over the LV endocardial surface, the system continuously analyzes its location in 3-dimensional space without the use of fluoroscopy. Results were collected from both UP and BP simultaneous recordings filtered at 0.5 to 400 Hz. The stability of the catheter-to-wall contact was evaluated at every site in real time, and points were deleted from the map if 1 of the following criteria was met: (1) a premature beat or a beat after a premature beat; (2) location stability, defined as a difference of >5 mm in end-diastolic location of the catheter at 2 sequential heartbeats; (3) loop stability, defined as an average distance of >5 mm between the location of the catheter at 2 consecutive beats at corresponding time intervals in the cardiac cycle; (4) cycle length that deviated >10% from the median cycle length; (5) different morphologies of the local ECG at 2 consecutive beats; (6) local activation time differences of >5 ms between 2 consecutive beats; and (7) different QRS morphologies of the body surface ECG.

By setting a “triangle fill threshold” value, the operator chooses the minimum triangle size for which the program will close a face on the reconstructed chamber. This feature allows the operator to determine the degree to which the system will interpolate between actual data points and will ensure that a minimal level of point density will be met at each mapped region. All maps were acquired with an interpolation threshold of 40 mm between adjacent points. The 3-dimensional LV endocardial reconstruction is updated in real time with the acquisition of each new site and displayed continuously on a Silicon Graphics workstation.

**Postprocessing Analysis**

UP and BP endocardial potentials from each sampled point were recorded from the tip electrode, and electrical maps were obtained by displaying the voltage potentials on a graded color scale. An additional endocardial map was then reconstructed to represent local shortening (LS). This function calculates the fractional shortening of regional endocardial surfaces at end systole and uses color scales to highlight areas with different myocardial contractility. Two different formulas were used to compile the LS function.

1. The first LS function is based on calculation of differences in surface areas of adjacent triangles around an index point during end systole. This function is called triangular local shortening (TLS), and its equation is \( \text{TLS}(p) = \frac{2A_{\text{ED}} - 2A_{\text{ES}}}{A_{\text{ES}}} \times 100 \). In this equation, TLS(p) is the displayed LS of a point p that defines the area around that mapped point at end systole, normalized to the area around that point at end diastole (which has been arbitrarily normalized to 100%). The end-diastolic timing was determined at the peak of the R wave in the surface ECG. Ai is the surface area of the neighboring areas derived by triangular connections between neighboring points. TLS is defined as 100% for every point at end diastole. On the basis of our preliminary animal experiments in 12 normal dog hearts, the mean and upper 95% confidence limits of TLS were 67% and 79%, respectively. Therefore, the TLS was estimated as (1) <80%, the surface area around the point is normally decreasing in size during systole; (2) 80% to 100%, the surface area around the examined point is abnormally decreasing in size during systole (hypokinesis); and (3) ≥100%, no change or paradoxical increase in the surface area around the examined point during systole (akinosis or dyskinesis).

2. Additional LS function was used to assess regional contractility. This function quantifies regional wall motion by calculating the fraction of linear distances of each endocardial point from its neighboring points at end systole, relative to end-diastolic distances. This function is called linear local shortening (LLS) and uses the following equation: \( \text{LLS}(p) = 2(L_{\text{ED}} - L_{\text{ES}})/L_{\text{ED}} \times 100 \). LLE(p) denotes the average LS of a point (p) relative to all its endocardial neighboring potentials; LLE and LLE are the distances of an index point from its neighbors at the end of diastole and systole, respectively. The LLS(p) value is a ratio that becomes larger as the distance between the neighboring sites decreases during end systole. Conversely, the LLS becomes smaller (or even negative) if the contractility is reduced or has become paradoxical at the examined point. Also, to minimize potential “noise” generated by points that are too close (ie, their relative motion is smaller than the location accuracy of the system) or too distant (ie, their relative motion might be affected by more than a single region of interest), the algorithm uses a “weight function” \( W_{\text{LLS}} \) in the LLS equation to filter the impact of these points (<5 or >12 mm) from the average LS calculation. The weight function thus depends on the density of sampled points and the volume of the heart in addition to its dependency on the distances between points at end diastole. During end systole, the LLS is estimated as (1) >12%, the average distances around an examined point are normally decreasing in size; (2) 8% to <12%, mild impairment in the relative motion (ie, contractility) of neighboring points relative to the index point; (3) 4% to <8%, moderate impairment in the relative motion of neighboring points relative to the index point (ie, signifying severe regional hypokinesis or akinosis); and (5) <0%, paradoxical increase in the distances between neighboring points during systole (ie, dyskinesis).

Myocardial areas that manifested both high electrical signals (UP endocardial voltage ≥10 mV and BP endocardial voltage ≥2 mV in humans; UP endocardial voltage ≥25 mV and BP endocardial voltage ≥5 mV in dogs) and normal LS (TLS <80% and LLS ≥12% in humans and dogs) were interpreted to represent normal myocardial function. Areas with impaired electrical activity (UP voltage ≤10 mV and BP voltage <2 mV in patients; UP voltage ≤25 mV and BP voltage ≤5 mV in dogs) and impaired mechanical function (TLS ≥80% and LLS ≤8%) were interpreted to represent abnormal electromechanical properties in MI areas. If the mechanical behavior was impaired and different from what was expected from the normal electrical activity (electromechanical uncoupling), the area of interest was suspected to represent a hibernating myocardial region.

**Animal Study**

The animal study was designed to detect electromechanical changes occurring after coronary occlusion in dogs. Dogs were used for this study because they have extensive coronary collateral circulation and can tolerate myocardial ischemia and infarction. The animal model was an open-chest left anterior descending coronary artery (LAD) occlusion model. After anesthesia, a left thoracotomy and exposure of the heart obtained access to the LAD, and the infarction was produced by suture ligation of the mid-LAD, between the first and second diagonal branches. The 2 experimental groups underwent LAD ligation, and electromechanical mapping was obtained at baseline, after 24 hours (group 1; n = 6), and after 3 weeks (group 2; n = 7). In each map, UP and BP electrical voltage and TLS and LLS values were obtained by the mapping system at the occluded LAD zone (anterior and/or apical zones) and compared with the most normal functioning remote from the MI zone (usually inferior or posterior zones).

**Clinical Study**

In 24 patients (12 with documented clinical history of prior MI and 12 control patients without prior MI), electromechanical mapping was obtained after the patients had given informed consent and after...
routine diagnostic left heart catheterization. The mapping catheter was advanced under fluoroscopic guidance to the descending thoracic aorta. The catheter tip was deflected to form a J tip, and the catheter was advanced across the aortic valve into the left ventricle. Once inside the ventricle, the catheter tip deflection was released, and the initial 3 points outlining the boundaries of the left ventricle (apex, aortic outflow, and mitral valve) were acquired with fluoroscopic guidance. After these boundary points, no fluoroscopy was needed to acquire points throughout the left ventricle. The chamber 3-dimensional geometry was reconstructed in real time with 50 to 80 endocardial points until all endocardial regions (ie, anteroseptal, anterior free wall, anterolateral, inferobasal, inferoapical, and posterior walls) were represented by neighboring points on the map. After completion of the map, a thorough review of individual points was performed to obtain only stable points and eliminate “internal” points that might represent luminal or papillary muscle locations.

Comparison of Mapping Findings With Echocardiography

Twelve consecutive patients had echocardiography before the mapping procedure to assess regional contractility by echo compared with electromechanical mapping findings. Each LV map was divided into 6 regions (anteroseptal, anterior free wall, anterolateral, inferobasal, inferoapical, and posterior walls). Each region was separately analyzed for mechanical function according to the following criteria: (1) normal, with TLS <80%; (2) hypokinetic, with TLS between 80% and 100%; (3) akinetic, with TLS between 100 and 110%; and (4) dyskinetic, with TLS >110%. These regional wall motion analyses were compared with echocardiographic assessment of the corresponding regions. The echocardiographic data were obtained by 2 experienced cardiologists who were unaware of the mapping findings and who read the echo studies independently. When there was a disagreement between the 2 readings, the tapes were reviewed by both echocardiographers, and consensus was obtained.

Statistical Methods

All data are presented as mean±SD. Means of nominal values were compared among groups by ANOVA. Student’s t test was used for paired comparisons (post-LAD occlusion versus baseline values in the canine study). A regression analysis was applied to correlate between the 2 LS functions that were used to calculate regional contractility in the study. P<0.05 was considered statistically significant.
Results

Animal Study

After LAD ligation, a significant decrease in electrical activity over time was found in the LAD territory compared with baseline values (UP voltage, 32% reduction in 24 hours, 42.8±9.6 to 29.1±12.2 mV, P=0.007; 66% reduction at 3 weeks, 41.0±8.9 to 13.9±3.9 mV, P<0.0001; BP voltage, 58% reduction in 24 hours, 11.6±2.3 to 4.9±1.2 mV, P<0.0001; 78% reduction at 3 weeks, 11.2±2.8 to 2.4±0.4 mV, P<0.0001) (Figures 1 and 2, top). This was associated with impaired mechanical activity, manifested by abnormal LS values in the infarct zones at 24 hours compared with baseline (99±23% versus 64±11% at baseline for TLS, P=0.001; 1.8±1.2% versus 15.3±2.3% at baseline for LLS, P<0.0001) and after 3 weeks (100±16% versus 65±10% at baseline for TLS, P<0.001; 1.9±0.9% versus 14.9±2.1% at baseline for LLS, P<0.0001) (Figures 3 and 4, top). These values signify severe mechanical impairment (ie, regional akinesis) in the infarct territory after LAD occlusion. Both electrical voltage and mechanical LS functions were normal in the nonischemic (remote) myocardial territory at 24 hours (Figures 1 through 4, bottom). By contrast, a significant but lesser impairment in TLS and LLS was found in the zone remote from MI (inferoposterior wall) at 3 weeks after LAD ligation (76±10% versus 66±8% for TLS, P<0.01; 8.1±0.8% versus 12.7±2.3% for LLS, P<0.05) with preserved electrical activity (Figures 1 through 4, bottom). These findings together suggest that electromechanical mapping can differentiate between infarcted and healthy myocardial regions from a reduction in both electrical and mechanical activities. A representative canine voltage map before and after LAD occlusion is given in Figure 5. The decrease in voltage potentials compared with baseline in the occluded LAD zone (anterior wall) is evident from this example.

Clinical Study

Clinical and electromechanical data in 24 studied patients are presented in the Table. The voltage and LS values in the table represent the average ±SD of 7 to 12 endocardial points in each mapped region. The mean procedural time to acquire a complete electromechanical map was 31±9 minutes. Electromechanical mapping could clearly define normal myocardium, as evidenced by normal voltage (≥10 mV by UP and ≥2 mV by BP recordings) and normal LS (TLS ≥80% and LLS ≥12%). Conversely, there were areas with low voltage (≤10 mV by UP and <2 mV by BP) and abnormal mechanical activity (TLS ≥80% and LLS <8%), representing regions previously afflicted by MI. The average UP voltage in the MI region was 7.2±2.7 mV, compared with 17.8±4.6 mV in normally functioning zones remote from MI and 19.7±4.4 mV in patients without prior infarction (P<0.001 for MI values versus other zones, P=0.29 for remote versus normal values) (Table). The average BP voltage in the MI region was 1.4±0.7 mV, compared with 4.5±1.1 mV in the remote (noninfarcted) zone and 5.8±1.0 mV among patients without prior infarction (P<0.001 for MI values versus other zones, P=0.17 for remote versus normal values) (Table). Likewise, mechanical activity was impaired in the MI region compared with remote zones in all cases when examined by both TLS and LLS functions. In all cases, the mitral annulus area was defined by low electrical voltage (<10 mV by UP and <2.0 by BP recording) (Figure 5A, bottom). There were other regions with normal voltage and impaired mechanical activity (eg, remote zone of patients 5 and 8; Table). Representative voltage maps of patients with and without prior MI in different myocardial zones are shown in Figures 6 through 8.
Electromechanical Mapping in Infarcted Myocardium

**UP and BP Voltage, TLS, and LLS Values in Patients With Prior Myocardial Infarction and in Control Subjects**

<table>
<thead>
<tr>
<th>Patients With Previous MI (n=12)</th>
<th>MI Area</th>
<th>Remote From MI Area</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient</td>
<td>MI Site</td>
<td>Q Wave</td>
</tr>
<tr>
<td>1</td>
<td>Inferior</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>Anterior</td>
<td>Yes</td>
</tr>
<tr>
<td>3</td>
<td>Lateral</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>Anterior</td>
<td>Yes</td>
</tr>
<tr>
<td>5</td>
<td>Lateral</td>
<td>Yes</td>
</tr>
<tr>
<td>6</td>
<td>Anterior</td>
<td>Yes</td>
</tr>
<tr>
<td>7</td>
<td>Lateral</td>
<td>No</td>
</tr>
<tr>
<td>8</td>
<td>Lateral</td>
<td>Yes</td>
</tr>
<tr>
<td>9</td>
<td>Inferior</td>
<td>Yes</td>
</tr>
<tr>
<td>10</td>
<td>Anterior</td>
<td>Yes</td>
</tr>
<tr>
<td>11</td>
<td>Lateral</td>
<td>No</td>
</tr>
<tr>
<td>12</td>
<td>Inferior</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Mean±SD</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Patients Without Previous MI (n=12)</th>
<th>UpV, mV</th>
<th>BpV, mV</th>
<th>TLS, %</th>
<th>LLS, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>23.6±2.7</td>
<td>7.6±2.3</td>
<td>76±6</td>
<td>13.7±4.1</td>
</tr>
<tr>
<td>14</td>
<td>18.1±5.5</td>
<td>5.6±1.7</td>
<td>62±6</td>
<td>20.3±2.2</td>
</tr>
<tr>
<td>15</td>
<td>24.8±7.8</td>
<td>9.1±1.9</td>
<td>64±11</td>
<td>15.3±4.8</td>
</tr>
<tr>
<td>16</td>
<td>26.4±8.2</td>
<td>4.5±2.3</td>
<td>72±8</td>
<td>14.9±5.0</td>
</tr>
<tr>
<td>17</td>
<td>20.6±5.6</td>
<td>4.3±1.9</td>
<td>60±12</td>
<td>14.3±4.6</td>
</tr>
<tr>
<td>18</td>
<td>24.4±9.3</td>
<td>3.7±1.7</td>
<td>71±11</td>
<td>12.3±8.8</td>
</tr>
<tr>
<td>19</td>
<td>21.0±7.2</td>
<td>7.8±2.2</td>
<td>65±9</td>
<td>16.2±3.1</td>
</tr>
<tr>
<td>20</td>
<td>16.2±3.5</td>
<td>2.7±0.7</td>
<td>61±14</td>
<td>13.4±2.2</td>
</tr>
<tr>
<td>21</td>
<td>18.5±3.2</td>
<td>6.4±1.6</td>
<td>65±6</td>
<td>15.2±4.3</td>
</tr>
<tr>
<td>22</td>
<td>16.9±2.2</td>
<td>11.2±4.6</td>
<td>61±8</td>
<td>16.2±2.8</td>
</tr>
<tr>
<td>23</td>
<td>13.8±2.7</td>
<td>4.2±1.3</td>
<td>74±9</td>
<td>9.0±5.6</td>
</tr>
<tr>
<td>24</td>
<td>12.8±3.8</td>
<td>2.1±0.2</td>
<td>78±9</td>
<td>11.6±4.7</td>
</tr>
<tr>
<td><strong>Mean±SD</strong></td>
<td>19.7±4.4</td>
<td>5.8±1.0</td>
<td>67±6</td>
<td>14.3±2.7</td>
</tr>
</tbody>
</table>

*UpV indicates unipolar voltage; BpV, bipolar voltage; RCA, right coronary artery; LCx, left circumflex artery; and OM, obtuse marginal branch of LCx.*

**Comparison With Echocardiography Findings**

In 12 patients undergoing echocardiography, the correlation of regional wall motion analysis by electromechanical mapping and echocardiography was evaluated. There were 84 regions (6 regions per patient), and the concordance was 78% (56/72) between the 2 modalities. There was complete concordance in the anteroseptal and anterior free wall regions. In 10 of 72 segments (14%), the mapping showed TLS abnormality (>80%), and the echocardiography interpretation was normal. In another 6 of 72 segments (8%), the mapping showed normal TLS values and the echocardiography was read as hypokinesis. The greatest discordance, occurring in 7 of 16 segments (42%), was in the posterior wall, where the electromechanical map suggested impaired mechanical function and the echocardiogram was read as normal.

**Regression Analysis for the LS Function**

A regression analysis was applied to assess the correlation between the TLS and the LLS functions in the studied patients. Both TLS and LLS functions were evaluated in 288 segments of 24 patient (12 segments in each patient; septal, anterior, lateral, inferoposterior/apical, mid, and basal segments for each region). The regression model yielded a significant correlation ($r=0.68$, $P<0.0001$) between TLS and LLS values in the same region with the following regression equation: $\text{TLS}(%)=97.2−1.59\times \text{LLS}(%)$.

**Discussion**

Our study has made the first attempts to use catheter position in space throughout the entire cardiac cycle to calculate fractional shortening together with voltage of the intracardiac ECG to make the distinction between normal and infarcted...
myocardium. On the basis of our preliminary clinical and animal experiences using such electromechanical mapping procedures, we conclude that (1) MI can be diagnosed and differentiated from non-MI zones by a reduction in both electrical and mechanical activities and (2) such catheter-based electromechanical mapping studies might be clinically useful for real-time assessment of myocardial function in previously infarcted versus healthy myocardial zones.

Rational for Use of Voltage Potential to Assess Myocardial Function

Previous animal studies have identified profound electrophysiological alterations after experimental coronary occlusion.6–8 At the cellular level, these alterations include a marked diminution of the resting membrane potential, the action potential amplitude, the upstroke rate of depolarization, and the action potential duration.9–11 Sustained coronary occlusion induces a layer of surviving epicardial tissue overlying a core of necrotic myocardium.12 Intracellular recordings from this surviving epicardial layer showed cells with variable degrees of partial repolarization, reduced action potential amplitude, and decreased upstroke velocity. Monophasic action potentials recorded from ischemic endocardial surfaces in dogs have demonstrated a progressive and significant loss of electrical amplitude and upstroke velocity within minutes after LAD occlusion.13 Thus, electrical signals derived from ischemic and/or infarcted regions could differentiate between normal, necrotic, and ischemic myocardium and might be used as a sensitive quantitative tool for the assessment of myocardial viability.

Findings of the Study

Our animal data suggest that the extent of myocardial damage after infarction can be assessed from measurements of electromechanical endocardial signals. Myocardial necrosis is reflected by a simultaneous decrease in the measurable endocardial voltage and LS. Mechanical dysfunction could indicate irreversible myocardial necrosis as well as restorable ischemic or hibernating myocardium. The distinction between the 2 scenarios may be made by the assessment of local intracardiac voltage potentials. The existence of electromechanical uncoupling (impaired mechanical activity while electrical activity is intact) might signify retained myocardial viability. With myocardial necrosis, a decrease in electrical voltage follows the impairment in myocardial contractility, which together signify loss of myocardial viability. In our
study, initial signs of electrical impairment in dogs (as manifested by reduced endocardial voltage) were noted at 24 hours and more so at 3 weeks after coronary artery occlusion. The clinical findings showed a distinct difference in measured endocardial voltage between the infarcted and noninfarcted myocardial regions. Myocardial regions with previous MI were characterized by low electrical voltage amplitude and by impairment of regional contractility. The magnitude of electrical impairment in the infarct zones was even more apparent among patients than in the canine model. This might reflect enhanced collateral supply to the infarct zone in the canine compared with the human heart. Also, most of the human MIs were old infarcts, and the difference between the measured electrical potentials (human versus canine study) could be related to time since the infarct. Electromechanical uncoupling (preserved electrical activity and impaired mechanical activity) was observed in other regions. The significance of such findings in noninfarcted regions with impaired mechanical activity and preserved electrical function remains to be investigated. In particular, it should be determined whether such areas reflect ischemic or hibernating myocardial zones. Because this is a preliminary clinical experience, our study lacked comparative nuclear imaging data to validate those findings.

In our study, both UP and BP ECG recordings were used for analysis. Previous studies have shown that the BP recording might be more accurate in reflecting local changes in electrical activity because it is less likely to be influenced by contact stability, electrode size, and “far-field” electrical potentials. On the contrary, the magnitude of the BP signal might vary according to the orientation of the tip electrode toward the endocardial surface. In our canine study, the extent of reduction in voltage tended to be more pronounced with BP than with UP recordings. Although both UP and BP recordings could clearly identify the presence of MI in this study, it is unclear at that stage which of the 2 methods would be more accurate to detect subtle changes in local electrical activity to differentiate between normal, ischemic, and infarcted myocardium.

Limitations and Future Directions
The electromechanical paradigm for distinguishing between infarcted and healthy myocardium, with potential ability to detect myocardial viability, should undergo further validation, first, by comparison with established methods of assessing myocardial viability, such as nuclear imaging tests (eg, PET scan or thallium imaging with reinjection) or stress
echocardiography. Animal data should be correlated with histopathology in areas of infarction/ischemia versus normal zones. The electrical thresholds to distinguish between normal and abnormal viability zones should be further delineated. For example, in the posterior wall, physiological electroanatomic “abnormalities” were commonly seen as the catheter approached the mitral annulus area. This should be distinct from areas with pathological alterations due to previous infarction. According to our findings, at the present stage we cannot distinguish between normal and small infarct zones in the posterior wall near the mitral annulus (unless an extension of the MI to the inferobasal or the posterolateral areas is evident in the map). Also, electrical activity in transmural infarctions should be distinguished from nontransmural MIs. The impact of myocardial wall thickness on measurable electrical activity should also be defined. In addition, the effect of antiarrhythmic and anti-ischemic drugs on measurable electromechanical signals should be determined. Importantly, the potential for electromechanical restoration in ischemic/hibernating regions with electromechanical dissociation should be studied as the true “gold standard” for functional viability. This would necessitate larger studies with comparative electromechanical mapping before and after revascularization procedures (angioplasty or coronary artery bypass graft surgery). Finally, it remains to be determined which of the LS functions (TLS, LLS, or others) have better accuracy in detecting subtle change in regional contractility in response to acute or chronic ischemic insults.

Because LV electromechanical mapping is an invasive procedure, safety must be carefully examined from larger clinical experiences. At present, catheter design is in a rapid stage of evolution to improve torque response, tip deflection, endocardial contact stability, and tip configuration to reduce surface trauma. Ultimately, with optimized catheter design and proper physician training, this procedure should permit the routine safe acquisition of LV endocardial mapping, which may enhance our understanding of intramyocardial detection of viability among patients.

Conclusions
On the basis of our preliminary findings in animals and humans, LV endocardial mapping using a new electromechanical catheter may provide unique insights into regional and global myocardial function. Recent MI zones could be identified by a reduction in both electrical and mechanical endocardial activity and could be differentiated from normal endocardial regions. The clinical utility of such diagnostic intervention in the assessment of myocardial function and viability will require further evaluation.

Acknowledgment
This study was supported by a grant from the Cardiology Research Foundation, The Washington Cardiology Center, Washington, DC.
References

Preliminary Animal and Clinical Experiences Using an Electromechanical Endocardial Mapping Procedure to Distinguish Infarcted From Healthy Myocardium
Ran Kornowski, Mun K. Hong, Lior Gepstein, Steven Goldstein, Samer Ellahham, Shlomo A. Ben-Haim and Martin B. Leon

Circulation. 1998;98:1116-1124
doi: 10.1161/01.CIR.98.11.1116

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1998 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/98/11/1116

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/