A novel left ventricular (LV) mapping system (Biosense Inc) uses low-intensity magnetic field energy to determine the location of sensor-tipped catheter electrodes within the LV. On the basis of previous experimental and human studies correlating the extent of myocardial ischemia with the amplitude of electrical signals, we hypothesized that such an integrated LV electromechanical mapping system could be used to distinguish healthy from infarcted or ischemic myocardium on the basis of the extent of electromechanical endocardial signals. If this hypothesis is confirmed, the ability to detect on-line myocardial viability in the catheterization laboratory may be feasible.

The present study attempted to distinguish between infarcted, ischemic, and normal myocardium by comparing LV electromechanical mapping data with myocardial perfusion imaging studies using the dual-isotope technique in symptomatic patients with coronary artery disease.

Methods

Patients
To be included in the study, patients had to have (1) chronic ischemic coronary syndrome associated with class III or IV angina, (2) reversible and/or fixed myocardial perfusion defects on single photon emission computed tomography (SPECT) imaging, (3) significant coronary artery obstruction documented by coronary angiography, and (4) LV ejection fraction ≥40%. Exclusion criteria included peripheral vascular disease, aortic stenosis, unstable ischemic syndrome on intravenous nitrates or heparin drugs, atrial fibrillation, LV thrombus, and contraindication to adenosine administration. Informed consent was obtained from all patients before any diagnostic procedures.

Mapping System

As described previously, the electromechanical mapping system uses (1) a triangular location pad with 3 coils generating ultralow magnetic field energy, (2) a stationary reference catheter with a miniature magnetic field sensor located on the body surface, (3) a navigation sensor mapping catheter (7F) with deflectable tip and electrodes providing endocardial signals, and (4) a workstation for information processing and 3-dimensional LV reconstruction.

Mapping Procedure

The patients were heparinized (70 U/kg). The mapping catheter was advanced under fluoroscopic guidance to the descending thoracic aorta, its tip was deflected to form a J shape, and it was introduced across the aortic valve into the LV. Once inside the LV, the tip deflection was released, and the initial 3 points outlining the

Key Words: mapping ■ ventricles ■ myocardium ■ ischemia
boundaries of the LV (apex, aortic outflow, and mitral inflow) were acquired with fluoroscopic guidance. Subsequently, no fluoroscopy was needed to acquire additional sampled points throughout the LV. The operator acquired points only when the catheter tip was stable on the endocardium, as evidenced by local activation time stability, location stability, loop stability, and cycle-length stability.1–3 The system uses a triangular algorithm to reconstruct the LV anatomy, which is presented in real time on a Silicon Graphics workstation. By setting a “triangle fill threshold” value, the operator chose the size of triangles, for which the program closes a “surface” on the reconstructed chamber. This feature allowed the operator to determine the degree of the system interpolation between actual data points and ensured that a minimal level of point density was met at each mapped region. All maps were acquired with an interpolation threshold of 40 mm between adjacent points. Once all endocardial regions were represented on the map, the operator completed the reconstruction of the LV map, and the catheter was removed from the LV.

Electromechanical Data
From the electrical data, a color-coded unipolar voltage map (in mV) was generated. From the mechanical data, regional contractility was obtained by use of the local endocardial shortening (LS) formula:

\[
LS(p) = \sum \left( L_{i_{\text{ED}}} - L_{i_{\text{ES}}} / L_{i_{\text{ED}}} \right) \times 100; \]

\( L_{i_{\text{ED}}} \) and \( L_{i_{\text{ES}}} \) are the distances of an index point from its neighbors at end diastole and end systole, respectively. The LS(p) value is a ratio that becomes larger as the distance between the neighboring sites decreases during end systole. Conversely, the LS becomes smaller (or even negative) if regional contractility is reduced or becomes paradoxical.

A fixed cylindrical polar reference coordinate map (regional view map) was defined with anatomic reference points acquired at end diastole (Figure 1). 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 length, respectively. Thus, the longitudinal location of each endocardial site was determined on the basis of its projection on the long axis. The midventricular and base segments were further divided into 4 regions: anterior, septal, inferoposterior, and lateral (Figure 1). Thus, in total, endocardial sites were divided into 9 regions for comparative analysis with nuclear imaging data.

Myocardial Perfusion Imaging
All patients underwent myocardial perfusion imaging with the SPECT technique. Dual-isotope imaging was performed with 201 Tl for rest and 99m Tc sestamibi for stress imaging. Under resting conditions, 3 mCi of 201 Tl was administered intravenously. Patients were then positioned in a standard SPECT camera in the supine position, and rest imaging was performed beginning 15 minutes after thallium injection. After completion of acquisition of the rest images, subsequent imaging was performed using pharmacological stress with adenosine administration. Adenosine 140 μg · kg⁻¹ · min⁻¹ was administered with constant monitoring of heart rate and rhythm and blood pressure. 99m Tc sestamibi was administered at peak vasodilator effect (at 4 minutes of a 6-minute adenosine infusion). Heart rate, rhythm, and blood pressure were monitored for at least 7 minutes after completion of vasodilator infusion. Stress imaging was performed beginning 60 minutes after sestamibi injection. In patients

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**Figure 1.** Left anterior oblique (top left) and right anterior oblique (bottom left) views of LV voltage map. Colors represent peak-to-peak amplitudes of intracardiac electrograms, ranging from 4.9 mV (red) to 22.3 mV (purple). Same LV is displayed as a regional view (right) for segmental analysis.
Electromechanical Data in 132 Myocardial Segments Divided According to the Perfusion Status in Radionuclide SPECT Imaging

<table>
<thead>
<tr>
<th>SPECT (No. of Segments)</th>
<th>Unipolar Voltage, mV</th>
<th>Local Shortening, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal (n=56)</td>
<td>14.0±2.4</td>
<td>12.5±2.8</td>
</tr>
<tr>
<td>Reversible (n=66)</td>
<td>12.0±2.8</td>
<td>10.3±3.7</td>
</tr>
<tr>
<td>Fixed (n=20)</td>
<td>7.5±3.4</td>
<td>3.4±3.4</td>
</tr>
<tr>
<td>P (overall ANOVA)</td>
<td>0.0002</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>P (fixed vs normal)</td>
<td>0.0001</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>P (reversible vs normal)</td>
<td>0.048</td>
<td>0.067</td>
</tr>
<tr>
<td>P (reversible vs fixed)</td>
<td>0.005</td>
<td>0.001</td>
</tr>
</tbody>
</table>

with a suspected or objectively determined irreversible perfusion defect, who might have reduced perfusion at rest to viable tissue, redistribution images of the rest thallium data were performed. This was achieved by a second acquisition of thallium data at 3 to 4 hours to represent redistribution activity. After the rest-redistribution thallium study, the patient underwent stress imaging as indicated above.

The cardiac images were oriented in standard orthogonal views. Images were read by an experienced operator who was unaware of LV electromechanical mapping data. A 9-segment model was used for analysis in which the short-axis slices were selected for interpretation, representing basal, midventricular, and apical levels of the LV. The mid and basal short-axis slices were subdivided into 4 segments representing the anterior, anteroseptal, inferoposterior, and lateral regions, similar to the subdivision obtained by the LV electromagnetic mapping. A qualitative assessment for these 9 segments (normal, reversible defect, fixed defect) was performed for both the rest thallium and stress sestamibi data sets.

A comparative analysis of the myocardial perfusion images in relation to LV electromechanical data was performed using calculated average voltage and LS values in each myocardial segment defined as having normal, reversible, or fixed perfusion defects.

Statistical Methods

All data are presented as mean±SD. Means of nominal values (voltage and LS) were compared between myocardial segments with normal, reversible, or irreversibly fixed perfusion by ANOVA. Intergroup comparison was made by t test with Bonferroni correction. A value of P<0.05 was considered statistically significant.

Results

Eighteen patients (14 men, 58±12 years old) were studied. The majority (n=16) had class III angina. Six patients had previous myocardial infarction, and the majority had prior revascularization procedures (10 patients with prior angioplasty and 12 with prior coronary bypass). No procedural complications were noted during or after the mapping procedure. Of 162 myocardial segments, 132 were available for comparative analysis. In 30 segments, no definite interpretation could be made because too few (<3) sampled points were taken during the electromechanical mapping procedure. The Table summarizes the electromechanical mapping data obtained in examined segments grouped according to the results of the perfusion study. As shown in the Table, an overall significant difference in voltage potentials and LS values was found between groups (P<0.001 for each comparison by ANOVA). The average voltage potentials (14.0±2.0 mV) and LS values (12.5±2.8%) were highest when measured in myocardial segments with normal perfusion (n=56) and lowest (7.5±3.4 mV and 3.4±3.4%) when measured in myocardial segments with fixed perfusion defects (n=20). Myocardial segments with reversible perfusion defects (n=66) had intermediate voltage amplitudes (12.0±2.8 mV, P=0.048 versus normal and P=0.005 versus fixed segments) and LS values (10.3±3.7%, P=0.067 versus normal and P=0.001 versus fixed segments). A representative LV voltage map is shown in Figure 2.

Discussion

In a previous study, we found that electromechanical mapping can distinguish infarcted myocardium from noninfarcted zones by simultaneous reduction in electrical and mechanical activity. However, this preliminary experience lacked a comparison with radionuclide perfusion imaging studies to validate the assessment of myocardial viability. Thus, in the present study, we performed a comparative analysis between LV electromechanical mapping data and radionuclide perfusion imaging to differentiate between infarcted, ischemic, and normally perfused myocardium. Our results show (1) a moderate (≈15%) reduction in endocardial potentials and LS in segments with reversible perfusion defects and (2) a profound electromechanical impairment in segments with fixed perfusion defects. These results may indicate that such LV mapping may be useful for detecting myocardial viability in the catheterization laboratory on the basis of measurements and localization of electromechanical signals.

Assessment of Myocardial Viability

In treating patients with coronary artery disease, it is essential to determine the extent of myocardial damage and ischemic burden to determine which patients would benefit most from revascularization. At present, the extent and severity of myocardial ischemia in coronary artery disease are assessed by use of noninvasive myocardial imaging modalities such as nuclear scintigraphy, PET imaging, or stress echocardiography. Previous studies have identified profound electrophysiological alterations after experimental induction of acute or chronic myocardial ischemia. The combination of electrical recordings with mechanical data, using the location sensors, seems to be a novel approach for on-line quantitative assessment and localization of myocardial viability. Larger clinical studies are necessary to test whether the use of such electromechanical mapping procedures may indeed serve as a novel diagnostic tool for on-line detection and precise localization of myocardial viability.

Limitations

First, in this study, the sampled population was relatively small. Nevertheless, the segmental analysis method allowed us to obtain relatively large samples for comparative assessment. Second, the regional analysis that was performed in the present study may limit the resolution accuracy of the mapping data due to the “smearing” (ie, averaging) effect of sampled points with different characteristics within the same region. An additional limitation is the use of somewhat “oversimplified” assessment of voltage amplitudes as the only...
Figure 2. Left posterolateral (A) and right anterior oblique (B) projections of LV voltage map in patient with prior lateral wall infarction. Note infarcted area (A, arrows) manifested by low voltage amplitudes (red, <8.0 mV), unlike apical zone of ischemia (green, ~10 mV) and otherwise normal myocardium (blue and purple, >14 mV).
measure of myocardial electrical activity. Future algorithms incorporated within the LV mapping system will allow the evaluation of other electrical measures, such as the electrical signal duration and the ratio of voltage to duration. These measures may enhance the accuracy of the mapping system for detecting ischemic areas manifested by subtle changes in the electrical activity.

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References
Comparison Between Left Ventricular Electromechanical Mapping and Radionuclide Perfusion Imaging for Detection of Myocardial Viability
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