Electromechanical Characterization of Chronic Myocardial Infarction in the Canine Coronary Occlusion Model

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Background—Defining the presence, extent, and nature of the dysfunctional myocardial tissue remains a cornerstone in diagnostic cardiology. A nonfluoroscopic, catheter-based mapping technique that can spatially associate endocardial mechanical and electrical data was used to quantify electromechanical changes in the canine chronic infarction model.

Methods and Results—We mapped the left ventricular (LV) electromechanical regional properties in 11 dogs with chronic infarction (4 weeks after LAD ligation) and 6 controls. By sampling the location of a special catheter throughout the cardiac cycle at multiple endocardial sites and simultaneously recording local electrogams from the catheter tip, the dynamic 3-dimensional electromechanical map of the LV was reconstructed. Average endocardial local shortening (LS, measured at end systole and normalized to end diastole) and intracardiac bipolar electrogram amplitude were quantified at 13 LV regions. Endocardial LS was significantly lower at the infarcted area (1.2±0.9% [mean±SEM], P<0.01) compared with the noninfarcted regions (7.2±1.1% to 13.5±1.5%) and with the same area in controls (15.5±1.2%, P<0.01). Average bipolar amplitude was also significantly lower at the infarcted zone (2.3±0.2 mV, P<0.01) compared with the same region in controls (10.3±1.3 mV) and with the noninfarcted regions (4.0±0.7 to 10.2±1.5 mV, P<0.01) in the infarcted group. In addition, the electrical maps could accurately delineate both the location and extent of the infarct, as demonstrated by the high correlation with pathology (Pearson’s correlation coefficient=0.90) and by the precise identification of the infarct border.

Conclusions—Chronic myocardial infarcted tissue can be characterized and quantified by abnormal regional mechanical and electrical functions. The unique ability to assess the regional ventricular electromechanical properties in various myocardial disease states may become a powerful tool in both clinical and research cardiology. (Circulation. 1998;98:2055-2064.)

Key Words: mechanics ■ myocardial infarction ■ electrophysiology ■ mapping

Studies of the coronary anatomy and left ventricular (LV) global and regional contractile functions are usually the basic elements of the evaluation of patients with ischemic heart disease. A major objective in the assessment of these patients is to accurately determine the presence, site, extent, and nature of the dysfunctional myocardial tissue, because these may possess important diagnostic, prognostic, and therapeutic implications. Consequently, numerous methods, such as echocardiography, angiocardiography, radioangiography, CT, and magnetic resonance imaging, have been developed throughout the years in an attempt to diagnose and quantify these damaged areas.

Common to all imaging modalities described above is their ability to assess regional mechanical function of the heart. Depression of myocardial function, whether chronic or transient, may present similarly with all of these methods. To increase our understanding of the extent and type of myocardial damage in coronary artery disease patients, we suggest evaluation of the electrical characteristics of the myocardium as well. We believe that gaining more information, in the form of combined electromechanical evaluation of the heart, may increase our understanding of the nature of various myocardial diseases.

Recently, a new nonfluoroscopic, catheter-based, magnetic mapping system that allows the generation of 3-dimensional (3D) dynamic electromechanical maps has been developed and validated in both animal and human studies. In this study, we used the ability of this new method to combine spatial, electrical, and mechanical information to evaluate the electromechanical properties of chronic LV myocardial infarction in dogs. Specifically, we planned to quantify the in vivo effects of the presence of the infarct on regional LV mechanics, electrical activation, and regional electromechanical coupling. The results of this study should be the first description of the in vivo electromechanical changes in chronic infarction.

Methods

Animal Preparation

Studies were performed on 17 mongrel dogs (6 controls and 11 dogs with chronic coronary occlusion) weighing 15 to 35 kg. The
Electromechanical Characterization of Chronic MI

The nonfluoroscopic electromechanical mapping system has been described elsewhere. In brief, the system (NOGA, Biosense) uses ultralow magnetic fields generated by an external magnetic field emitter located under the operating table to accurately determine the location and orientation of a miniature passive magnetic location sensor incorporated just proximal to the tip of a 7F deflectable-tip electrophysiological catheter (NAVI-STAR, Cordis-Webster). This information allows tracking of the tip of the mapping catheter while it is deployed within the heart without the use of fluoroscopy.

Mapping Procedure

Electromechanical mapping was performed 4 to 6 weeks after coronary ligation in the chronic infarction group and in the acute stage of the control group. Two locatable catheters (reference and mapping catheters) were introduced into the heart under fluoroscopic guidance. The location of the tip of the roving mapping catheter was recorded relative to the fixed reference catheter positioned at the right ventricular apex, compensating for both animal and cardiac movements. The location and orientation of the mapping catheter were continuously shown on the Silicon Graphics workstation, allowing the accurate 3D navigation of the catheter (Figure 1, top left).

The mapping procedure is based on sequential sampling of the location of the catheter together with the local electrogram recorded from its tip at several endocardial sites. At each endocardial site sampled, the location of the tip of the catheter was recorded at a frequency of 50 Hz. Hence, the movement of each endocardial site throughout the cardiac cycle can be traced, displayed, and analyzed (Figure 1, top right). This allowed the creation of a dynamically beating image of the 3D reconstruction of the chamber (electromechanical maps, Figure 1, bottom).

The quality of the catheter-wall contact was evaluated at each site, and points were deleted automatically 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 the difference in end-diastolic location of the catheter at 2 sequential heart beats, of >3 mm; (3) loop stability, defined as the average distance between the location of the catheter at 2 consecutive beats at corresponding time intervals in the cardiac cycle, of >3 mm; (4) cycle length that deviated >15% from the median cycle length; (5) different morphologies of the local electrogram at 2 consecutive beats; (6) local activation time difference between 2 consecutive beats of >3 ms; (7) different QRS morphologies of the body-surface ECG; and (8) marked ST elevation in the local unipolar electrogram, indicating excessive catheter pressure.

Regional Parameters

To register data between hearts, a fixed anatomic cylindrical polar reference coordinate system was defined. The center of mass of the reconstructed chamber was automatically calculated from the set of endocardial points sampled. The long axis of the ventricle was defined as a line connecting the apex with the center of mass. The long axis was divided into 3 parts (apex, midventricle, and base, consisting of 20%, 40%, and 40% of the long-axis length, respectively), and the longitudinal location of each endocardial site was determined on the basis of its projection on this axis. The midventricle and base were further divided equally into 6 different circumferential regions: anterior (π/6<θ<π/2), lateral (π/2<θ<5π/6), posterior (5π/6<θ<7π/6), inferior (7π/6<θ<3π/2), inferoseptal (3π/2<θ<11π/6), and anteroseptal (11π/6 to π/6). The circumferential coordinate parameter (θ) was defined in radians around the central axis (zero being anterior chest wall and clockwise direction being positive). In total, the endocardial surface was divided into 13 different regions for comparison.

Pathological Verification of Infarction

After termination of the mapping procedure, the animals were killed by an intravenous anesthetic overdose, and the hearts were excised. The coronary arteries were then perfused with 300 mL of TTC solution (2,3,5-triphenyltetrazolium chloride, 5 g/250 mL normal saline), and the hearts were fixed in 4% formaldehyde solution. The infarcted area was identified as the region that was not stained by TTC, the presence of fibrous scar, and myocardial thinning. The hearts were sliced transversely into sections ~5 to 7 mm in width and were later scanned. The outlines of each slice and the extent of the infarcted area were traced and measured with a special morphometric software.

The endocardial surface area of each slice was calculated by multiplying its measured endocardial circumference by the slice width. Total endocardial area (TEA) was then calculated as the sum of the surface areas of all slices. Similarly, the endocardial infarcted area of each slice was calculated by multiplying the circumference of the endocardial surface overlying infarction by the individual slice width. The endocardial infarcted area (EIA) was then calculated as the sum of these areas in all slices. The percentage of endocardial infarcted area was calculated as EIA×100/TEA.

To assess possible electrical or mechanical changes related to infarct thickness, the depth of the infarct at each slice (expressed as percentage of slice thickness) was also measured.

Correlation of Electrical Maps With Pathology

The extent of infarction as depicted from the electrical maps in the last 9 animals was correlated with the same parameter as derived from pathology. The infarcted zone was identified in the electrical
maps as the region with abnormally low voltage surrounded by a steep voltage gradient, and its surface area was calculated as the area in which the amplitude of the bipolar electrograms was lower than the threshold voltage of the margin. The threshold value of each map was defined by adding a fixed value (2.0 mV) to the median value of the 10 points with the lowest voltage values. This algorithm was based on our preliminary observation that all infarcts were characterized by a steep voltage gradient surrounding a central area of low voltage.

**Figure 1.** Electromechanical mapping of dog LV. Top left, Right lateral view of LV in an intermediate stage of mapping process (26 sampled points). Note “head and eyes” icon helping in orientation. Mapping catheter is pointing toward anterior wall. Top right, Right lateral view showing local trajectories of all sampled points throughout 2 cardiac cycles. Note that motion of each point is made of 2 identical loops, indicating good stability. Bottom left, Right lateral view of a normal LV during end diastole. Colors represent local activation times, with red indicating early activation that originates at septum. Bottom right, End-systolic view of same ventricle.
voltage and that the voltage of the border could be determined by adding a fixed value to the voltage at the center.

In 8 animals, the catheter was navigated back to the border of the scar (defined by the steepest voltage gradient), and 2 to 5 radiofrequency (RF) ablation lesions per dog were applied to sites at the suspected margin. RF ablation was performed with a 500-kHz RF generator (RFG-3C; Radionics) in a temperature-controlled mode (70°C) for up to 60 seconds. The accuracy of the ablations in identifying the margin of the infarct was then assessed by gross pathological examination.

Figure 2. Top left, LAO view of a typical LS map of LV with chronic infarction. Red represents areas with reduced LS (<4%); yellow and green, areas with mildly reduced LS; and blue and purple, areas with LS >12%. Top right, Bull’s-eye image of same ventricle, presenting average LS values in 13 LV regions. Note decreased LS value (2.8%) at infarcted territory (midanterior wall). Bottom left, LAO view of bipolar voltage map of same ventricle. Colors represent peak-to-peak amplitude of local intracardiac bipolar electrogram. Red indicates areas with abnormally low voltage (<1 mV); purple, areas with normal bipolar amplitude >7 mV; and yellow and green, areas with intermediate values. Bottom right, Regional bull’s-eye representation of same map. Note that infarcted area demonstrates abnormally low voltage (1.4 mV).
Correlation Between the Electrical and Mechanical Maps

The spatial correlation between the mechanical and electrical maps was assessed by examination of possible concordance in the regional distribution of the abnormalities by use of the fixed regional polar coordinate system. To further evaluate the spatial correlation between the maps, all points were divided into 3 groups according to their location relative to the infarcted area as determined from the

Figure 3. Top left, LAO view of left ventricular LS map of 1 control. Note that entire ventricle shows normal LS, as evident by blue and purple colors (LS >12%). Top right, Regional bull’s-eye view of same ventricle. Bottom left, LAO view of bipolar voltage map of same ventricle. Entire ventricle displays normal bipolar amplitude, as evident by blue and purple colors (voltage >6 mV) throughout map and by average regional values summarized in bull’s-eye view (bottom right).
Average LS, Bipolar, and Unipolar Amplitude Values in the Chronic Infarct and Control Groups

<table>
<thead>
<tr>
<th></th>
<th>Anterior</th>
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<th>Lateral</th>
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<th>Posterior</th>
<th></th>
<th>Inferior</th>
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<td></td>
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<td>9.2±1.5</td>
<td>7.9±0.7</td>
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<td>13.8±1.9</td>
<td>14.0±1.3</td>
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<td>14.9±1.2</td>
<td>11.7±1.9</td>
<td>14.5±1.1</td>
<td>12.5±2.3</td>
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<tr>
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<td>8.0±1.2</td>
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<td>9.2±1.3</td>
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<tr>
<td>Bipolar healthy</td>
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<td>11.1±1.3</td>
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<td>9.0±1.5</td>
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<tr>
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<td>37.2±1.8</td>
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LS indicates local shortening (%). Bipolar and unipolar voltages measured in mV.

LS infarct 1.2±0.9; LS healthy 15.5±1.2; Bipolar healthy 10.3±1.3; Unipolar healthy 39.4±2.2.

Results

Mechanical Maps

The endocardial LS maps acquired in all animals with chronic infarction displayed a similar pattern. A typical mechanical (LS) map of the LV with a chronic infarct is shown in Figure 2, top left, from a left anterior oblique (LAO) projection. Colors represent the endocardial LS values calculated at each site, with red representing areas with LS <6%, and abnormal voltage values (<threshold value), (2) normal mechanical and voltage values, (3) abnormal mechanical but normal electrical values, and (4) normal mechanical but abnormal voltage values. The LS threshold value (6%) was defined on the basis of preliminary sensitivity and specificity analysis. The percentage of EIA as determined from the electrical and mechanical maps was then calculated.

Statistical Analysis

Values are given as mean±SEM. Student’s unpaired t test was used to compare possible differences in LS and electromgram amplitude between the infarcted and healthy animals in the same group. Linear regression and Pearson’s correlation coefficient were used to correlate between the percentage of EIA as determined from the electrical maps and by pathology.

Figure 4. Average LS values in 4 different regions (mid: anterior, posterior, inferior, and interoseptal) in control (solid bars) and infarcted (open bars) groups. Note significant reduction in average LS value at infarcted region (midanterior wall) compared with noninfarcted regions in same group (†P<0.01) and with same area in controls (‡P<0.01).

Electrical Maps

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The Table and Figure 4 summarize the average regional LS data for the 11 animals with chronic infarctions that were studied and for the 6 controls. Endocardial LS was significantly lower at the LAD infarcted territory (midanterior wall, LS=1.2±0.9%, P<0.01) compared with each of the other 12 regions in the infarcted group and also with the average LS obtained at the same region in controls (15.5±1.2%).

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Electrical Maps

Figure 2, bottom left, displays a typical LAO view of an LV voltage map of 1 of the infarcted dogs. The colors represent the peak-to-peak amplitude of the sampled bipolar intracardiac electrograms, with red representing areas with low voltage (<1 mV); blue and purple, high bipolar amplitude (>6 mV); and green and yellow, intermediate values. In all cases, we found that the infarcted area, which displayed reduced LS, was also characterized by abnormally low bipolar voltage. The similarities in the regional distribution of the abnormal bipolar voltage and LS in the anterior wall can also be noted in the regional bull’s-eye views (Figure 2, top right, 2 days).
Figure 3 demonstrates a typical LAO view of a healthy LV. Note the homogeneous normal LS and bipolar voltage values (blue and purple colors) throughout the ventricle. In all hearts, we noted a decrease in voltage amplitude at the posterobasal wall (not shown in the figure), representing the mitral annulus fibrotic ring.

The Table and Figure 5 summarize the regional differences in bipolar and unipolar amplitude in the chronic infarction and control groups. The average bipolar amplitude was significantly lower (2.3 ± 0.2 mV, P < 0.01) at the infarcted area (midanteriwal wall) compared with all other LV regions in the infarcted group and also with the same area in the healthy control group (10.3 ± 1.3 mV, Figure 5). The regional distribution of the amplitude of the unipolar electrograms displayed a pattern similar to that of the bipolar one (Table, Figure 6). However, the differences between the values obtained at the infarcted and noninfarcted zones, although statistically significant, were smaller.

Correlation Between the Electrical Maps and Pathology

The accuracy of the electrical maps in delineating the presence, anatomic location, and extent of the infarcted area was evaluated as follows.

1. The percentage of endocardial infarcted area as determined from pathology was correlated with the same parameter as derived from the voltage maps. The latter value was calculated as the area in which the bipolar amplitude was lower than the threshold value at the margin. The threshold value ranged from 2.5 to 4.2 mV and averaged 3.0 ± 0.2 mV. Using this algorithm, we found excellent correlation (Pearson correlation coefficient = 0.90) between the percentage of endocardial infarcted area as determined from pathology (13.3 ± 0.9%) and from the voltage maps (12.4 ± 1.1%) (Figure 7).

2. In 8 animals, the catheter was navigated back to the margin of the infarct defined by the steepest voltage gradient, and 2 to 5 discrete point ablations per animal were delivered on the suspected border (Figure 8, left). In all cases (n = 28), the lesions were located (Figure 8, right) exactly on the margin as judged by gross pathological examination.

The thickness of the infarct was found to be relatively homogeneous in all hearts, with an average infarct depth of 65 ± 2% (range, 38% to 100%, with 1 heart displaying an infarct thickness of 100% and the rest of the hearts having slices with infarct depth ranging from 38% to 76%). Furthermore, in this series of experiments, we did not note any subendocardial infarction. Thus, because of the relatively homogeneous nature of the infarct, no significant changes were noted in the electrical or mechanical signals with respect to infarct thickness.

Correlation Between the Electrical and Mechanical Maps

In all the animals studied, we found complete concordance between the region of abnormal mechanics and low voltage...
(Table; Figures 4 and 5). In some animals, however, small spatial variations in the exact location of the abnormalities were noted. To further investigate this issue, we divided the sampled points into 3 groups: points located inside the infarcted area as determined from the voltage maps, points located outside the infarcted region, and points located in a 1-cm rim around the border. Using this method, we found that there was complete agreement in the mechanical and electrical results in the vast majority of points located outside and inside the infarct (89% and 83%, respectively). In the 1-cm border region, we found clusters of points from all types: abnormal LS and voltage values (21.5%), abnormal LS and normal voltage (12.4%), and normal LS and abnormal voltage (39.7%).

**Discussion**

The heart is characterized by 2 major functional phenomena: electrical excitation and the resulting myocardial contraction. In this study, we have tried to combine these 2 properties and to introduce a new concept in the evaluation of various myocardial diseases by simultaneous examination of the electromechanical properties of the tissue. Using the new electromechanical mapping technique that can trace and analyze the motion of individual endocardial sites throughout the cardiac cycle, we quantified regional LV mechanics by measuring local endocardial shortening. Simultaneously, we recorded endocardial potentials and generated and integrated detailed 3D electromechanical maps.

The results of the present study demonstrate that chronic myocardial infarction can be characterized and quantified by abnormalities of both the mechanical and electrical functions. Significant statistical differences in LS and in the amplitude of the local intracardiac electrograms were noted at the infarcted area compared with the noninfarcted regions. Furthermore, we also demonstrated that the 3D voltage maps can delineate with great precision both the presence and extent of the infarct.

**Electrical Maps**

The results of the present study show that chronically infarcted myocardium can be characterized by the abnormal electrograms recorded from this region. Thus, significant differences were noted between the amplitude of the electrograms recorded at the infarcted area (bipolar voltage, 2.3 ± 0.2 mV, \(P<0.01\)) compared with the noninfarcted regions in the same hearts and also with the same area in controls (10.2 ± 1.3 mV, \(P<0.01\)).
Moreover, this study has also demonstrated that a simple measurement, such as the use of the peak-to-peak amplitude of the bipolar intracardiac electrograms, can be used to delineate very accurately the presence, location, and extent of the infarct. This was indicated by the high correlation in measuring infarct size and by the precision in identifying the margin of the scar.

The observation that endocardial electrograms recorded from an infarcted area are characterized by very low amplitude and fractionated morphology is not new and is derived from the field of clinical electrophysiology. Until now, however, it has not been possible to spatially associate these electrograms. The ability of the method presented in this study to associate spatial, electrophysiological, and mechanical data allowed the accurate anatomic identification and quantification of the infarcted area.

Although both the unipolar and bipolar voltage maps could identify the presence of the scar in this study, it is widely accepted in the literature that bipolar recordings reflect more the bipolar amplitude in normal areas).

These variations were present mainly at the infarct border and may have resulted from a number of causes: (1) areas of dysfunctional but viable myocardial tissue (such as stunned or hibernating myocardium) that may be characterized by normal voltage but abnormal mechanics; (2) the fact that in the voltage maps, the value of each point is dependent on its neighboring sites, and this may result in reduction of the reduced LS area; and (3) the spectrum of mechanical abnormalities ranging from hypokinesis through akinosis to dyskinesis.

**Limitations of the New Method and Limitations of the Study**

The new method used in the present study possesses a number of technical and conceptual limitations. Because of the sequential nature of the mapping procedure, a major prerequisite is that the rhythm be monomorphic and stable throughout the procedure. By introducing filtering criteria that rejected points in which cycle length deviated >15% of the median cycle length, we were able to partially compensate for this limitation.

Another important limitation of any method that aims to measure regional mechanics is the level of noise. Several factors may possibly contribute to increasing the noise of the system in measuring regional motion: (1) the inherent noise of the system in determining the location of the catheter. The location resolution of the system was quantified previously and was demonstrated to be <1 mm in both in vitro and in vivo studies; (2) the effect of respiration not fully compensated by the reference catheter; (3) unstable catheter-wall contact; and (4) a premature beat or a beat after a premature beat, which may alter the mechanical pattern of the acquired site.

To compensate for this possible noise, several steps were undertaken. First, we introduced filtering criteria for points that failed to demonstrate stability over 2 consecutive beats. These criteria included location and loop stability parameters that examined the repeatability of the motion of each sampled site, electrical stability criteria that examined the repeatability of the local electrogram, and cycle-length stability criteria. Second, the LS value calculated for each endocardial site was averaged from all neighboring sites. Third, a weight function was added, reducing the significance of neighboring sites with low signal-to-noise ratio (very close or very far points). Fourth, regional analysis was used, which could also “average out” possible noise (false-positive or false-negative points).

Despite the obvious limitations discussed, the present study demonstrates regional correlation between decreased LS and electrogram amplitude in the infarcted territory and also shows high correlation with pathology. However, the results of the present study should be further assessed in the clinical setting, because both the pathological substrate, the size of the ventricle, interactions between the electrodes and the activation wave front, and other technical issues regarding the mapping procedure may vary from the animal model studied here. For example, because of the relatively homogeneous nature of the pathological substrate, we did not note any significant electromechanical changes related to infarct depth. Hence, future studies will have to correlate possible electromechanical changes related to different infarct models (transmural versus subendocardial, patchy versus homogeneous, etc.).

**Possible Clinical and Research Applications**

The results of this study may possess important clinical and research implications. By spatially associating mechanical and electrical information, we were able to show that the necrotic myocardial tissue could be accurately located and differentiated...
from the normal myocardium by a measurable reduction in both mechanical and electrical functions. Moreover, the ability to accurately associate on-line spatial, electrical, and mechanical data allowed us to accurately determine the location and extent of the infarct as well, which may have important diagnostic, therapeutic, and prognostic implications.

The cause of the abnormally contracting myocardial area may vary from acute ischemia, to stunned or hibernating myocardium, to irreversibly necrotic tissue. In recent years, diagnostic testing to evaluate the presence and extent of dysfunctional but viable myocardium has become an important component of the clinical assessment of patients with chronic coronary artery disease and LV dysfunction. In the past decade, several modalities have evolved to identify physiological markers of myocardial viability in regions with dysfunction. These include positron emission tomography to assess intact myocardial metabolic activity, thallium imaging to assess myocardial perfusion and membrane integrity, and dobutamine echocardiography to assess inotropic reserve.

In this study, we have tried to introduce a new concept in the characterization of dysfunctional myocardial tissue by assessment of the electromechanical properties of the tissue. Our results demonstrate that irreversibly necrotic tissue can be characterized by the coupling of abnormal mechanical and electrical activities. The concepts of the present study should now open the way to several other studies that will define the characteristic electromechanical changes in different myocardial pathological conditions, such as acute ischemia, stunning, hibernation, etc. Defining electromechanical criteria for these different entities not only may aid in accurately diagnosing the location and extent of these conditions but also may provide further insight into their nature.

Conclusions

From this study, in which electromechanical mapping was performed in the chronic infarct model in dogs, we conclude that chronic myocardial infarction could be detected, quantified, and differentiated from healthy myocardium by abnormalities of both the mechanical and electrical functions. The results of the present study also stress the importance of combining anatomic, mechanical, and electrical information for both research and clinical cardiology.

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References


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