Differentiation of Viable and Nonviable Myocardium by the Use of Three-Dimensional Tagged MRI in 2-Day-Old Reperfused Canine Infarcts

P. Croisille, MD; C.C. Moore, MD, PhD; R.M. Judd, PhD; J.A.C. Lima, MD; M. Arai, MD; E.R. McVeigh, PhD; L.C. Becker, MD; E.A. Zerhouni, MD

Background—To limit ischemic myocardial injury, it is important to differentiate viable from infarcted myocardium. Three dimensional (3D) tagged MRI has the ability to quantify myocardial 3D deformation and strain (noninvasively and precisely), and can achieve a true comparison of contraction not only from region to region, but also at different levels of function. In this study, we investigated whether regional strain mapping obtained by 3D-tagged MRI can differentiate between viable but stunned myocardium and nonviable myocardium.

Methods and Results—We examined 7 dogs 2 days after a 90-minute closed-chest left anterior descending coronary artery occlusion followed by 48 hours of reperfusion. 3D-tagged MR images spanning the entire left ventricle were acquired both at rest and during dobutamine infusion (5 μg · kg⁻¹ · min⁻¹ IV). Regional blood flow was measured with radioactive microspheres and used to define risk regions. Infarcted regions were defined as 2,3,5 triphenyltetrazolium chloride negative regions. Strains in infarcted regions were greatly impaired compared with remote regions (P<0.001) and remained unchanged during dobutamine stress. Risk regions showed a dysfunction at rest, with improved function during dobutamine infusion. Receiver operating characteristics analysis showed that radial strain was more accurate for identifying viable regions.

Conclusions—When coupled with a stress test, 3D strain mapping by the use of tagged MRI is a sensitive and noninvasive method for characterizing ischemic injury. Regional strain can be used to differentiate between viable but stunned and nonviable myocardium within the posts ischemic injured myocardium. (Circulation. 1999;99:284–291.)

Key Words: myocardial contraction □ stunning, myocardial □ inotropic agents □ ischemia □ magnetic resonance imaging
tractility occurring under low-dose dobutamine stress 48 hours after reperfusion to investigate whether regional strain mapping obtained by 3D-tagged MRI can differentiate between viable but stunned myocardium and nonviable myocardium.

Methods

The animals in this study were handled according to the animal welfare regulations of and after the approval of the animal committee of our institution. These regulations are in accordance with the Position of the American Heart Association on Research Animal Use, adopted November 15, 1984. These animals were involved in a recently published study that described the mechanisms of contrast enhancement in fast MRI of reperfused infarcts.

Experimental Preparation

We studied 7 mongrel dogs (20 to 25 kg). On day 1, the animals were anesthetized with sodium pentobarbital (35 mg/kg IV), intubated, and connected to a Harvard respirator. An intravenous femoral catheter was placed for drug administration. A catheter sheath was placed in the right femoral artery and was used to introduce a 6F pigtail catheter into the left ventricle (LV) under fluoroscopic guidance. This catheter was used for microsphere administration and for monitoring LV pressure. The sheath of this catheter was used for microsphere reference sampling from the femoral artery. A second catheter sheath was then placed in the left femoral artery and used to introduce a 7F JL 2.5 (Left Judkins 2.5-cm) guiding catheter into the left main coronary artery. A 3.5F angioplasty balloon catheter was passed through the guiding catheter and slid into the left anterior descending (LAD) coronary artery over a 0.35-mm guidewire that was positioned in the proximal left anterior descending coronary artery under fluoroscopic guidance. The balloon was then inflated, and the aVL lead of the ECG was monitored for ST-segment elevation. The balloon remained inflated for 90 minutes to produce myocardial infarction and then was deflated to allow reperfusion of the infarcted myocardium. Regional myocardial blood flow was measured immediately before LAD occlusion and 80 minutes after occlusion by LV injection of ±2 million sonicated microspheres (15±1 μm diameter) labeled with 1 of several radionuclides (153 Gd, 113 Sn, 103 K, 95 Nb, or 46 Sc; DuPont). The animals were then allowed to recover for 48 hours.

Imaging Protocol

MRI was performed 48 hours after reperfusion. The animals were again anesthetized and mechanically ventilated, and a pigtail catheter was advanced into the LV under fluoroscopic guidance to monitor LV pressure, for administration of the third and fourth sets of microspheres injected at rest, and in a subset of 3 animals during dobutamine stress. Animals were transported to the MRI facility for scanning in a whole-body 1.5T scanner (Signa; GE Medical Systems). The animals were placed in the right anterolateral position with electrodes for ECG gating, and a flexible surface coil was wrapped around the chest.

The imaging protocol is based on an ECG-triggered segmented k-space SPGR pulse sequence. Contiguous stacks of short-axis images in double obliquity were prescribed at end systole to cover the entire heart from base to apex. Six long-axis views were also prescribed from the midventricular short-axis image and were distributed in a radial fashion every 30°. Imaging parameters were as follows: 32-cm field of view; 8-mm slice thickness; repetition time, 6.5 ms; echo time, 2.3 ms; 15° tip angle; 256x110 image matrix; 1 signal acquired; and 5 phase-encoded views per movie frame (temporal resolution of 32 ms). Slab saturation achieved “black blood” in the LV cavity. To describe the 3D deformation of the heart, we used myocardial tissue tagging to encode the motion in 3 orthogonal directions (Figure 1). The tagging pulse consisted of nonselective radiofrequency pulses separated by spatial modulation of magnetization encoding gradients to achieve a tag separation of 6 mm. Tagging pulses were applied immediately before the imaging pulse and were triggered by the upslope of the QRS from the ECG. Reproducible diaphragmatic positions between image sets were obtained by stopping the respirator at end expiratory volume during acquisition time. A total of 15 (5 image planes x 3 sets) breathholds was needed to acquire a complete set of tagged images. Without removing the animal from the magnet, we administered dobutamine intravenously during continuous ECG monitoring and blood pressure recording. A dose of 5 μg · kg⁻¹ · min⁻¹ of dobutamine was infused into the intravenous femoral catheter 10 minutes before imaging and during 3D tagged imaging. The rest/stress imaging protocol lasted ~1 hour.

Definition of Infarct, Risk, and Remote Regions

After MR imaging, the hearts were arrested with KCl and excised. The LV was isolated, then sectioned from base to apex into 5 short-axis slices. The junctions of the right ventricular (RV) walls and the interventricular septum were identified to determine the position of the midseptum point on the most basal slice used as a landmark for later registration of the stack with corresponding MR images. The slices were then incubated in a 2% solution of 2,3,5 triphenyltetrazolium chloride (TTC) for 20 minutes at 37°C. Regions that failed to demonstrate brick red staining were considered to represent infarcted myocardium. For each slice, a shallow cut was made to divide the LV circumference into 12 pie-shaped sections. Each of the pie-shaped sections was further divided into 3 to 5 pieces of approximately equal thickness from the endocardial to epicardial surfaces. The exact locations of these cuts were recorded before the slices were photographed to relate TTC regions to the location from which tissue samples were taken for microsphere blood-flow counting. After the slices were photographed, myocardial samples (0.1 to 0.5 g) were obtained for microsphere counting. Each of the samples was then weighted and counted in a gamma-emission well spectrometer (model 5986, Hewlett-Packard) along with the reference blood samples at appropriate energy windows. Regional myocardial blood flow (mL/min per 100 g) was then calculated by standard methods. On the basis of TTC staining and regional myocardial blood flow findings for each animal, we assigned samples to 1 of the 3 following regions: infarcted, risk (but noninfarcted), and remote regions. Samples that were partly located in ≥1 zone were excluded from the analysis. The infarcted regions corresponded to samples failing to stain with TTC. The risk (but noninfarcted) region was defined as those noninfarcted samples (outside the TTC negative zones) in which myocardial blood flow was reduced by ≥50% during the
Data Analysis

Strain Computation

Tagged-image data consisted of sets of 120 to 180 images (5 slices × 8 to 12 phases × 3 directions). When a stress study was performed, a second set of images was acquired. Images were processed by the use of an in-house developed software program on a Silicon Graphics workstation. Contour and tag detection was performed by the use of a semiautomated detection algorithm described in detail elsewhere. Then, 3D displacement and strain throughout the LV were determined at each time point using the displacement field-fitting method at rest and at stress. This technique uses all of the available information in the 3 orthogonal tag patterns to compute the 3D deformation gradient tensor at any point in the heart wall. A material point mesh is defined within the heart wall at end diastole as the point at which 3D displacement and deformation is calculated over time. The mesh density was set to 12 points radially through the wall at each circumferential location, and at 5 levels equally spaced in the longitudinal direction. A total of 180 material points were defined that corresponded to 180 volume elements of myocardium. The inner and outer shell of points was distributed radially 2 mm into the myocardium from the fitted endocardial and epicardial surfaces, and the midwall shell of points was distributed at an equal distance from the endocardial and epicardial surfaces. The shape of the mesh was determined by the end-diastolic endocardial and epicardial contours and the density of material points was chosen to match the microsphere sampling layout for spatial registration purposes. Coordinates of the RV-LV junctions and the corresponding midseptum points were calculated on the most basal slice at end-diastole on the stack of MR images and were used as reference landmarks for strain calculation and to display reconstructed 3D strain maps. The same mesh dimensions were used for rest and stress studies allowing to match spatially the corresponding regions on the rest and stress strain maps. A data processing time of 3 to 4 hours was required to analysis a full 3D data set at rest and stress.

Circumferential registration (in-plane registration) between 3D strain maps and pathology sampling layouts was achieved by matching landmark locations. Axial registration (between-slice levels) was controlled by the use of the same number of levels (n = 5) when prescribing the stack of slices during MR imaging, when choosing the size of the reconstruction mesh of the 3D strain maps, and when sectioning the isolated heart into short-axis slices for sampling.

In this study, we report strain changes between the reference state (end diastole) and the deformed state (usually end systole) by the use of the fractional changes in length (in percent) for each of the 3 orthogonal normal strains in the radial, circumferential, and longitudinal directions (see Appendix A). Radial strain was calculated by the use of the tissue incompressibility correction that improves the robustness of wall thickening measurements (see Appendix B). We calculated in addition end-systolic first principle strain E1 (maximum thickening), and reported the vector magnitude and its direction, given by the angles between the eigenvector, the local circumferential direction (E1ac) and the longitudinal direction (E1al), respectively (Figure 2).

Accuracy of Viability Detection

To assess the accuracy of strain mapping for the detection of viable and nonviable regions, we performed receiver operating characteristic (ROC) analysis to compare observer performance to differentiate viable from nonviable regions based on the transmural extent of the infarction. Regions were considered viable when transmural extent of the infarction was equal or less than ⅓ of the wall thickness in that region. Regions were considered nonviable when transmural extent of the infarction was greater than ⅓ of the wall thickness in that region. Arrays of graphs were then reviewed independently by 3 readers in a blind fashion without knowledge of the pathology results. Observers were given an unrestricted amount of time to evaluate the cases. On the basis of the degree of contraction at rest and stress and the functional recruitment that occurred under dobutamine stress, they were asked to identify viable regions and to assign a level of confidence for that particular finding. Level of confidence was graded on a 4-point scale as follows: 4 = definitely viable, 3 = probably viable, 2 = probably nonviable, and 1 = definitely nonviable.

Statistical Analysis

All measurements are presented as mean (SEM). Differences in hemodynamic variables were compared by the use of paired t tests. Differences in microsphere blood flow measurements between regions and over time were assessed by the use of repeated-measures ANOVA. To compare strain values between regions, data were averaged by region in each animal. Differences between regions at rest were analyzed by the use of a 1-way ANOVA. To test the effect of low-dose dobutamine stress on differences in strain between regions, we used repeated-measures ANOVA. Whenever applicable, a posteriori comparisons were made according to the Tukey HSD method. The CORROC2 and LABMRMC programs (C. Metz, PhD, University of Chicago) were used to conduct the ROC analysis for this study with a maximum-likelihood estimation technique to obtain binomial ROC data. Comparison of the observed responses across readers and for the different strains was achieved with a multireader multicasus ROC ANOVA by the use of the LABMRMC algorithm. A z score test was further used to calculate, when required, the statistical significance of the difference between the areas under estimated ROC curves. Statistical significance was inferred when P < 0.05, and all reported P values were 2-tailed.

Results

Hemodynamics

There was no change in average heart rate (HR) at the end of ischemia (90 minutes) compared with baseline (130 [5] bpm versus 136 [6] bpm, P = NS), whereas systolic arterial blood pressure (SBP) decreased significantly (110 [6] versus 138 [5 mm Hg], P < 0.01). At the time of imaging (48 hours of occlusion compared with remote regions at the time of the occlusion. Remote regions were chosen on the side of the ventricle opposite the infarction.
reperfusion), HR was 129 (6 bpm) and SBP was 107 (4 mm Hg). During infusion of dobutamine, HR increased from 127 (8 bpm) to 151 (7 bpm) ($P < 0.05$), whereas SBP rose from 119 (6 mm Hg) to 136 (7 mm Hg) ($P = $NS$)$. Accordingly, the rate-pressure product increased by 35 (5%), from 15 236 (1015) to 20 502 (1340) ($P < 0.01$).

Myocardial Blood Flow
During LAD occlusion, blood flow decreased markedly in both infarcted (0.09 [0.03 mL · min$^{-1}$ · g$^{-1}$]) and risk regions (0.15 [0.04 mL · min$^{-1}$ · g$^{-1}$]) compared with remote regions (0.82 [0.07] mL · min$^{-1}$ · g$^{-1}$) ($P < 0.01$). After 48 hours of reperfusion, blood flow returned to near normal values in risk regions (0.68 [0.11 mL · min$^{-1}$ · g$^{-1}$]) compared with remote regions (0.95 [0.11] mL · min$^{-1}$ · g$^{-1}$) ($P = 0.1$), but remained decreased in infarcted regions (0.50 [0.12] mL · min$^{-1}$ · g$^{-1}$) ($P < 0.01$). Low-dose dobutamine slightly increased regional blood flow (from 0.48 [0.07] mL · min$^{-1}$ · g$^{-1}$ to 0.57 [0.12] mL · min$^{-1}$ · g$^{-1}$) in infarcted regions, and increased of a greater extent in risk regions (from [0.62 [0.10] mL · min$^{-1}$ · g$^{-1}$] to 1.05 [0.18] mL · min$^{-1}$ · g$^{-1}$) and normal regions (from [1.05 [0.2] mL · min$^{-1}$ · g$^{-1}$ to 1.46 [0.26] mL · min$^{-1}$ · g$^{-1}$]).

Strain Analysis
Overall, the size of infarcted regions (TTC-negative regions) was 6.4 (1.8%) of total LV mass and of risk regions, 23.5 (2.9%). A total of 436 of 1260 samples from all 7 dogs fulfilled the selection criteria, and each of these 436 assigned to 1 of the predefined infarcted, risk, or remote regions. Among them, there were 91 samples in the infarcted regions, 188 in the risk region, and 157 in the remote regions. The number of samples in each animal varied from 4 to 37 for the infarcted region and from 13 to 71 for the risk regions. Figure 3 shows array plots of normal strains as a function of time for 1 animal 48 hours after reperfusion at rest and under dobutamine stress. Figure 4 shows 3D strain maps of maxi-

![Figure 3. Array plots of 3D strains as a function of time at rest (black) and under dobutamine stress (gray) at multiple locations of the myocardium (midwall) in 1 animal with a 90-minute LAD occlusion imaged 48 hours after reperfusion. Each graph of the 12×5 arrays corresponds to 1 of 12 locations in the circumference at 5 levels from base to apex. **Infarcted regions; *risk (but noninfarcted) regions.](Image)
mum principal strain E1 at end systole obtained in the same animal studied at rest and then under dobutamine stress.

**End-Systolic Strains: Rest**

Radial strain exhibited at rest an overall significant difference across regions at end systole ($P<0.0001$), with a depressed radial strain in both infarcted and risk regions of 5.2 (1.3%) and 6.1 (1.5%), which contrasted with remote regions, in which wall thickening was 23.2 (1.9%) ($P<0.01$) (Figure 5). However, radial strain was not significantly different in infarcted and risk regions. Circumferential and longitudinal strains demonstrated comparable trends, even if circumferential strain failed to show overall differences across all regions. Both showed decreased negative strains in both infarcted and risk regions ($P=\text{NS}$) compared with remote regions. Maximum principal strain E1 (or greatest thickening) at end systole showed results similar to those observed with radial normal strain (Figure 6). The magnitude of E1 was reduced in both infarcted and risk regions ($P=\text{NS}$) compared with remote regions ($P<0.001$). Maximum principal strain direction changed in infarcted and risk regions compared with remote regions. Angle from the local longitudinal direction ($E_{1l}$) decreased in both regions ($P<0.01$), whereas angle from the local circumferential direction ($E_{1c}$) decreased only in risk regions ($P<0.01$).

**End-Systolic Strains: Stress**

Figure 5 summarizes end-systolic strain results grouped by region for all experiments at rest and under low-dose dobutamine infusion. Under dobutamine challenge, end-systolic radial strain increased as expected in the remote regions from 23.2 (1.9%) to 41.7 (4.2%) ($P<0.001$). Whereas end-systolic strains remained unchanged in the infarcted regions, a significant increase in end-systolic radial strain was observed in the risk regions (6.1 [1.7%] versus 20.1 [2.2%]; $P=0.002$). The difference between end-systolic radial strain in infarcted and risk regions became significant ($P=0.002$) under dobutamine stress. Again, circumferential and longitudinal strains exhibited a comparable trend, but the differences between and within regions between rest and stress were not statistically significant. Maximum principal strain E1 allowed identification of the increase of the magnitude of maximum thickening in the risk regions 14.2 (1.5%) to 36.4 (4%) ($P=0.005$), whereas the average magnitude of maximum thickening remained almost unchanged (11 [1.8%] versus 18 [2.2%]; $P=\text{NS}$) (Figure 6). Changes in direction of maximum principal strain that occurred under dobutamine stress were not statistically significant.

**Accuracy of Viability Detection**

Of a total of 420 regions analyzed ($7\times60$ midwall volume elements), 39 were categorized as nonviable regions. Figure 7...
A: Principal strain E1

<table>
<thead>
<tr>
<th>Angles (degree)</th>
<th>Infarcted</th>
<th>Risk</th>
<th>Remote</th>
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<tr>
<td>Rest</td>
<td>Stress</td>
<td>Rest</td>
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<tr>
<td>E1al</td>
<td>143(9)</td>
<td>102(14)</td>
<td>106(9)</td>
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<tr>
<td>E1al</td>
<td>81(4)</td>
<td>63(4)</td>
<td>85(4)</td>
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Figure 6. End-systolic maximum principal strain E1 (wall thickening) 48 hours after reperfusion: rest vs low-dose dobutamine stress. Regional differences between infarcted, risk, and remote regions. Results in the histogram (A) are the average magnitude of E1 within each region and between animals; B indicates the average direction of the maximum principal strain from the local circumferential direction (E1al), and from the local longitudinal direction (E1al). *P≤0.05, **P<0.001 by repeated measures ANOVA. Results are mean (SEM). %ES indicates percent change in dimension at end systole from end diastole.

Figure 7. ROC curves derived from data pooled across 3 observers. Observer performance for the detection of viable and nonviable regions are plotted by the use of SRI (radial strain calculated with the tissue incompressibility correction), Srr (normal radial strain), Scc (normal circumferential strain), and Sll (normal longitudinal strain). Numbers in parentheses give the averaged area under the curve across 3 readers. TPF indicates true positive fraction; FPF, false-positive fraction.

Discussion

This study shows that, in a closed-chest canine model 48 hours after reperfusion, 3D strain mapping obtained at rest and under low-dose dobutamine stress by the use of 3D-tagged MRI reveals viable but stunned regions and differentiates between viable but stunned and nonviable myocardium within the postischemic injured territory. Regions that are dysfunctional at rest but recover under inotropic stimulation (viable myocardium) correspond to noninfarcted (TTC positive) regions, which were at risk at the time of the occlusion but are normally perfused 48 hours after reperfusion (stunned regions). Alternatively, regions that are dysfunctional at rest and remain dysfunctional under stress (nonviable myocardium) correspond to infarcted (TTC negative) regions. Within the postischemic territory, regional strains increased significantly under low-dose dobutamine only within the noninfarcted but at risk regions, whereas regional strain remained unchanged within infarcted regions, thus allowing the identification of salvaged viable regions. 3D strain mapping quantifies accurately the extent of viable and nonviable regions. Accuracy will depend on the criteria used to define viability.

At rest, the degree of dysfunction was comparable for all strain components within the infarcted regions and the noninfarcted but at risk regions. Within the infarcted regions, end-systolic strains were severely depressed but showed on average the persistence of a residual positive thickening (+5%) and negative shortening (−2%). Because infarcted regions were circumscribed to only subendocardial layers, it appears that this residual active deformation 48 hours after reperfusion within the infarcted regions could be related to the ability of the outer wall to generate greater force than the endocardial wall.16,17

Our results suggest that radial strains differentiate viable from nonviable regions better than do longitudinal or circumferential strains. This finding confirms that radial thickening is a valid integrated measurement of regional function not only across all layers but also within a specific layer. Indeed, radial thickening accounts for the complex 3D fiber rearrangements that occur within the wall during contraction.16 Although local radial strain can be estimated directly from the radial component of the stretch tensor, its precision, which relies on the tag density orthogonal to the strain direction, is supported by the lowest density of tag data (2 to 3 tags). In contrast, in our experiments, strains involving the circumferential and longitudinal directions were the best supported with tag data that were distributed continuously across the wall. Using these 3D strain components and the incompressibility assumption to recalculate radial function, we found that SRI gave a more robust radial strain estimate.

In our study, the direction of maximum principal strain E1 showed only limited differences across regions at rest, and differences decreased, becoming not significant under dobutamine infusion. Our results are confirmed by a recent study in a reperfusion model in rabbits showing that postischemic...
dysfunction does not alter the direction of maximal shortening deformation. However, in nonreperfused infarction models, more radical changes in principal strain direction have been reported.

Experimental studies with isoproterenol or dopamine demonstrated that stunned myocardium maintains functional reserve that may be recovered after moderate isotropic stimulation without deleterious effects. Several clinical studies have subsequently shown that dobutamine stress echocardiography is a useful method to predict reversible dysfunction. Although the exact mechanism of stunning is still unknown, this observation raises the possibility that stunning is due in part to increased local mechanical stress secondary to endocardial damage that increases local afterload. In this hypothesis, stunned myocardium generates tension but no observable shortening, unless isotropic stimulation is used to reduce local afterload earlier in systole by increased contractility of the normal regions, consequently unmasking the function of the stunned myocardium. More accurate measures of contractile recruitment that can be afforded by 3D tagging can thus be of value because they provide a more accurate map of the extent of viable regions.

Our study is the first to report noninvasive measurements of 3D strain in the postischemic myocardium after reperfusion and is also the first to report strain changes that occur under low-dose dobutamine stress. Accurate measurements of myocardial deformation with implanted markers have been recently reported in postischemic injury. Implantation of markers is limited, however, to a focal region of the myocardium and is too invasive for clinical use. MR tagging has the unique ability to provide noninvasive measurements of tissue deformation. Recent experimental studies with MR tagging have reported the mechanical behavior of nonreperfused transmural infarction by the use of 2D-tagged MR. The combination of parallel-tag MR imaging and displacement field fitting has the major advantage of providing an accurate and robust method to calculate 3D deformation in a particular region of the myocardium as well as its time course. The accuracy and robustness of this method is based on its reliance on accurately identifiable tag profiles rather than poorly defined heart contours; also, it uses all the available data along the entire length of each tag line in all 3 directions rather than only points that intersect with other tags or contours. The field-fitting technique has the advantage of giving an analytical representation of LV displacement for calculation of smooth estimates of strain and of being robust on uncertainty in the tag position estimates. The reproducibility of tag position is extremely good, even with noisy images, especially when compared with the reproducibility of myocardial contours, which is lessened by the variable interpretation of the endocardium position between observers. Because 3D-tagged MRI has the ability to register strain maps spatially, a true and accurate comparison of contractility within the same region or between different regions can be achieved under varying physiological conditions (such as during a stress test) and despite conformation changes of the heart during systole.

Summary
The experimental model is an important determinant of the mechanisms for postischemic dysfunction. By using a closed-chest canine model with a 90-minute balloon catheter LAD occlusion reperfused for 48 hours, we found that not only was the invasiveness of the experimental procedure limited, but also the infarct size and the time window for tissue salvage were close to the clinical circumstances of performance of an early thrombolysis. In addition, the time of MR imaging (48 hours after reperfusion) was chosen to correspond to that at which assessment of viability is required for therapeutic planning. The results of our study form a framework on which to base ongoing and future clinical evaluations. 3D strain mapping with tagged MRI is an entirely noninvasive technique that is directly applicable clinically. This technique should constitute a diagnostic tool and an alternative to existing methods when more accurate assessment of regional mechanics is required.

Appendix A: Strain Tensor Definition
The strain tensor is solved at each point of the myocardium on the basis of the orthogonal parallel-tagged MR image sets that provide the 1-dimensional displacement field of the heart for each direction of the space. For each material point of the mesh, the local displacement gradient tensor \( \nabla U \) was computed mathematically by evaluating the directional derivatives of the local displacement field in the deformed state. From this, the lagrangian finite deformation gradient tensor \( F = (I + \nabla U)^{-1} \), where \( I \) is the identity tensor. The lagrangian finite deformation strain tensor \( E \) and linearized strain tensor \( S \) were then calculated from \( E = \sqrt{F^T F - I} \) and \( S = (F^T F)^{-2} - I \). The tensor \( S \) is useful because its axial components \( S_{rr}, S_{cc}, \) and \( S_{ll} \) represent the percent change in length from end diastole when multiplied by 100.

Appendix B: Radial Thickening Calculation
Because there were often only 2 tag lines across the heart wall, the radial component of \( S \) \( (S_{rr}) \) was the least well supported by the tag data and was subject to higher variability. For this reason, the other components of the stretch tensor, which were well supported by the tag data and the assumption of tissue incompressibility, were used to estimate the radial thickening (\( S_{rr} \)). The incompressibility correction was performed by the use of all the components of the right stretch tensor \( U \) except \( U_{rr} \), and the incompressibility constraint (determinate of \( U = 1.0 \)) to solve for the radial thickening component \( S_{rr} \):
\[
S_{rr} = \frac{1.0 + U_{cc} - U_{ll}(U_{ll} - U_{cc}) - U_{ul}(U_{ul} - U_{ll})}{U_{ll} - U_{cc}} - 1.0
\]

Acknowledgments
This study was supported in part by a Research Award from the SCBT/MR (Society of Computed Body Tomography and Magnetic Resonance), a Société Française de Radiologie grant, and NIH grants RO1HL45090 and LH45683.

References


