Ultrasound Strain Imaging of Altered Myocardial Stiffness
Stunned Versus Infarcted Reperfused Myocardium

Cristina Pislaru, MD; Charles J. Bruce, MD; Peter C. Anagnostopoulos, MD; Jill L. Allen; James B. Seward, MD; Patricia A. Pellikka, MD; Erik L. Ritman, MD, PhD; James F. Greenleaf, PhD

Background—In this study we evaluate the diastolic deformation of ischemic/reperfused myocardium and relate this deformation to tissue elastic properties.

Methods and Results—Farmed pigs were subjected to left anterior descending coronary artery occlusion followed by reperfusion to create either stunning (n=12) or transmural myocardial infarction (n=12). Ultrasound-derived radial strain rates (SR) and strain were measured in the ischemic and remote walls. Myocardial stiffness was estimated from diastolic pressure–wall thickness relationship obtained from preload alterations. At reperfusion, end-systolic strain (εs) was significantly reduced in both stunned and infarcted walls compared with their remote walls (3±3% versus 26±2% and 1±0% versus 33±5%, respectively; P<0.0001) or baseline values. Diastolic passive deformation (εA) and rates of deformation during early (Esr) and late (Asr) diastole were comparable between stunned and remote walls (εA: 7.3±1.6% versus 7.9±1.9%; Esr: −2.7±0.4 s⁻¹ versus −2.6±0.5 s⁻¹; Asr: −1.8±0.2 s⁻¹ versus −1.9±0.3 s⁻¹; P=NS for all) but were of significantly lower magnitude in infarcted walls versus remote walls (εA: 1.1±0.2% versus 11.4±1.9%; Esr: −0.3±0.1 s⁻¹ versus −2.4±0.4 s⁻¹; Asr: −0.3±0.1 s⁻¹ versus −2.5±0.4 s⁻¹; P<0.0001 for all). Stiffness coefficient of exponential diastolic pressure–wall thickness relation was higher for infarcted (P<0.05) but not for stunned walls (P=NS) compared with their remote walls.

Conclusions—Early after postischemic reperfusion and in the presence of severely reduced systolic deformation, diastolic passive deformation (and rates of deformation) can distinguish stiff, noncompliant, transmurally infarcted myocardial walls from those more compliant walls containing viable but stunned myocardium. (Circulation. 2004;109:2905-2910.)

Key Words: echocardiography — diastole — myocardial infarction — stunning, myocardial — myocardial stiffness

Myocardial viscoelastic properties have been studied extensively. It has been shown that myocardial stiffness increases during ischemia and recovers after reperfusion.1–4 If the ischemic period is sufficiently prolonged to create an infarct, the increase in stiffness continues further and is exacerbated with reperfusion.5 Tissue Doppler imaging and strain echocardiography are established methods to track myocardial displacement and deformation.6–9 Regional functional assessment after acute myocardial infarction (MI) has been focused on measurement of active systolic function.7,10–15 Few studies describe diastolic deformation,11–13 but none have specifically related passive diastolic deformation to myocardial stiffness and viability status. We hypothesize that passive deformation may disclose information on myocardial viability, particularly in conditions of persistent systolic dysfunction, such as stunning and acute MI. Therefore, in the present study, we relate regional diastolic deformation of stunned and infarcted myocardium to myocardial diastolic stiffness.

Methods

Animal Preparation
Experiments conformed to the Position of the American Heart Association on Research Animal Use and institutional guidelines for animal use. Pigs were anesthetized with ketamine, fentanyl, and etomidate and mechanically ventilated. After sternotomy, the heart was suspended in a pericardial cradle. Animals received heparin to maintain adequate anticoagulation. A pressure transducer catheter (Millar Instruments, Inc) was inserted via the right carotid artery into the left ventricle (LV). In 8 pigs, cotton strings were passed around the inferior vena cava to transiently reduce preload. Increases in preload were achieved by saline infusion (300 to 500 mL). Electrocardiographic and LV and aortic pressure signals were digitized online (DataQ Instruments, Inc).

Experimental Design
Acute myocardial ischemia was induced by ligating the mid-to-distal left anterior descending coronary artery for either 20 to 30 minutes (stunning group; n=12 pigs) or 90 to 160 minutes (transmural infarct group; n=12 pigs). Reperfusion was allowed for 60 minutes. Excised hearts were perfused with Evans blue for delineation of area at ischemic risk. Each heart was cut into 6 to 9 slices...
perpendicular to the LV long axis, then immersed in 2,3,5-triphenyltetrazolium chloride solution to delineate infarcted myocardium.

**Strain Echocardiography**

Digital cineloops of tissue Doppler imaging (140 to 220 frames per second) were acquired in short-axis view at low papillary muscle level. An ultrasound scanner (Vivid FiVe, GE Healthcare) and a 5-MHz transducer were used. Care was taken to align the Doppler beams with the vector of motion of the interrogated wall. Three to five cardiac cycles in sinus rhythm were acquired during steady state at baseline, at the end of occlusion, and after 30 minutes of reperfusion.

Transmural radial strain rates (SR) and strain were calculated from tissue velocities (GeMat software, GE Vingmed Ultrasound, Horten, Norway). A 3-mm sample length was used for SR calculation. The anterior (ischemic) and inferior (nonischemic) LV walls were analyzed. Timing of end-diastole was set at the onset of the QRS complex, and that of end-systole was set at the time of minimum dP/dt.16

Peak SR values were measured during systole (S\(_{SR}\)) and late diastole (A\(_{SR}\)). Natural strain was obtained by integrating SR over time with end-diastole used as reference point and then converted to Lagrangian strain.17 End-systolic strain (S\(_{sys}\)) was measured as strain deformation from end-diastole to end-systole, and postystolic strain (S\(_{ps} \)) was measured from end-systole to time of maximum diastolic strain. The time from end-systole to onset of regional S\(_{SR}\) (T\(_{TE}\)) was also measured (Figure 1).

To evaluate passive myocardial properties, we calculated the diastolic deformation \(\epsilon_0\) occurring from the onset of A\(_{SR}\) to end-diastole (Figure 1). If it is assumed that load (pressure) during late diastole is homogeneously distributed circumferentially, the ratio of \( \epsilon_0 \) of the 2 walls could be considered a measure of the relative difference in compliance: Relative Strain = \(\epsilon_0\) (ischemic segment) / \(\epsilon_0\) (normal segment)\(^{-1}\).

**Loading Alterations and Assessment of Myocardial Stiffness**

In 8 of 24 animals (stunning group, n = 4; infarct group, n = 4), cineloops of tissue Doppler imaging were also acquired during preload alterations (caval constriction and saline infusion), both at baseline and after 30 minutes of reperfusion.

Myocardial stiffness was estimated from diastolic pressure–wall thickness relationship, which is analogous to pressure–segment length relationship.18 End-diastolic wall thickness, measured from gray-scale cineloops underlying the tissue Doppler data,19 was plotted against corresponding end-diastolic LV pressure for each cardiac cycle at each level of preload. Exponential curves were fitted through the data (least-squares regression analysis) with the use of the following equation: LV pressure = \(\alpha \cdot e^{-\beta \cdot \text{wall thickness}}\), where \(\alpha\) and \(\beta\) are best-fit function coefficients. The slope of this function (\(\beta\)-coefficient) characterizes myocardial stiffness.

**Hemodynamic Parameters**

Heart rate, peak systolic and end-diastolic LV pressure, time constant of LV relaxation, and maximum and minimum dP/dt were measured at the time of each echocardiographic measurement (DataQ Instruments, Inc). Values from 3 to 5 sequential heart cycles were averaged.

**Cardiac Specimen**

The LV slice corresponding to the in vivo ultrasound imaging plane was selected on the basis of anatomic landmarks (insertion of papillary muscles and right ventricular free wall). Transmural extent of necrosis was calculated as the fraction of pixels indicating infarcted tissue from the area at ischemic risk (not stained by Evans blue). The extent of area at risk from the whole LV area was measured by summation of its extent (cm\(^2\)) in all LV slices and expressed as a percentage.

**Statistical Analysis**

The influences of time, region, and their interaction were tested with the use of repeated-measures 2-way ANOVA. Pairwise comparisons within groups were made with \(t\) tests with Bonferroni correction where appropriate. Differences between groups were tested with unpaired \(t\) tests. Statistical significance was inferred for \(P<0.05\). Values are presented as mean±SEM.

**Results**

A total of 30 pigs were used. Six animals were excluded (death, n = 4; sustained arrhythmia, n = 2). Final analysis included data from 24 complete experiments (stunning group, n = 12; infarct group, n = 12). The occlusion period averaged 27±2 minutes in the stunning group and 133±12 minutes in the infarct group. Area at risk was 26±3% and 21±2%, respectively (\(P=\text{NS}\)). Transmural extent of necrosis was 0% and 91±3%, respectively.

**Hemodynamic Variables**

Table 1 shows hemodynamic data. Mean heart rate and LV pressure did not change significantly throughout the experiment.

**Regional SR and Strain Parameters**

At baseline, deformation parameters were similar between the 2 groups (Table 2 and Figure 2). During occlusion and at reperfusion, S\(_{SR}\) and \(\epsilon_{sys}\) were markedly reduced in both stunned and infarcted walls. There was no difference in \(\epsilon_{ps}\) at reperfusion between stunned and infarcted walls (Figure 2). The S\(_{SR}\) partially recovered in some animals from the stunning group and was higher, as a mean, in stunned than in infarcted walls (\(P<0.001\)).

Conversely, both E\(_{SR}\) and A\(_{SR}\) were comparable between stunned and remote walls after reperfusion (\(P=\text{NS}\)) but were significantly smaller in infarcted walls (\(P<0.0001\) versus remote walls for both E\(_{SR}\) and A\(_{SR}\)). It is noteworthy that there was little overlap of values between stunned and infarcted walls for both E\(_{SR}\) and A\(_{SR}\) compared with systolic parameters S\(_{SR}\) and \(\epsilon_{sys}\) (Figure 2).
TABLE 1. Hemodynamic Data

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Occlusion</th>
<th>Reperfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stunning group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>79±6</td>
<td>74±6</td>
<td>77±5</td>
</tr>
<tr>
<td>LVSP, mm Hg</td>
<td>88±5</td>
<td>88±8</td>
<td>80±6</td>
</tr>
<tr>
<td>LVEDP, mm Hg</td>
<td>10.9±1.6</td>
<td>13.2±1.8</td>
<td>11.8±1.8</td>
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<td>(dP/dt_{min}), mm Hg/s</td>
<td>1285±248</td>
<td>1116±293</td>
<td>1052±205</td>
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<td>(dP/dt_{max}), mm Hg/s</td>
<td>-1109±115</td>
<td>-928±137</td>
<td>-785±79</td>
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<tr>
<td>(\tau), ms</td>
<td>49±2</td>
<td>59±3*</td>
<td>55±2†</td>
</tr>
<tr>
<td><strong>Transmural infarct group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>82±8</td>
<td>75±7</td>
<td>77±8</td>
</tr>
<tr>
<td>LVSP, mm Hg</td>
<td>108±7†</td>
<td>88±7</td>
<td>86±7</td>
</tr>
<tr>
<td>LVEDP, mm Hg</td>
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<td>12.6±0.9</td>
<td>13.0±0.7</td>
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<td>(dP/dt_{min}), mm Hg/s</td>
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<td>1306±222</td>
<td>1160±200</td>
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<td>(dP/dt_{max}), mm Hg/s</td>
<td>-1507±149</td>
<td>-1109±191</td>
<td>-969±190</td>
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<tr>
<td>(\tau), ms</td>
<td>49±3.6</td>
<td>60±3*</td>
<td>61±2*</td>
</tr>
</tbody>
</table>

LVSP indicates LV peak systolic pressure; LVEDP, LV end-diastolic pressure; and \(\tau\), time constant of LV relaxation.

*P<0.001, †P<0.01 vs baseline.

Postpysystolic strain (\(\varepsilon_{PS}\)) was higher in stunned than in infarcted walls (Table 2). The onset of \(E_{SR}\) was delayed during occlusion and at reperfusion in both stunned and infarcted walls (Table 2).

TABLE 2. Regional Functional Parameters and Wall Dimensions

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Occlusion</th>
<th>Reperfusion</th>
</tr>
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<tbody>
<tr>
<td><strong>Ischemic Segment (Anterior Wall)</strong></td>
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<tr>
<td>(S_{ps}), s⁻¹</td>
<td>1.7±0.1</td>
<td>0.3±0.1†</td>
<td>0.7±0.1*</td>
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<tr>
<td>(E_{ps}), s⁻¹</td>
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<td>-2.0±0.5</td>
<td>-2.7±0.4</td>
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<td>(A_{ps}), s⁻¹</td>
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<td>-1.7±0.4</td>
<td>-1.8±0.2</td>
</tr>
<tr>
<td>(\varepsilon_{ps}), %</td>
<td>31±3</td>
<td>-10±1†</td>
<td>3±3†</td>
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<tr>
<td>(\varepsilon_{sys}), %</td>
<td>3.0±1.2</td>
<td>29.7±3.0†</td>
<td>152.2±5.3*</td>
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<tr>
<td>(\varepsilon_{sys}), %</td>
<td>11.0±1.8</td>
<td>10.3±1.4</td>
<td>7.3±1.6</td>
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<td>(T_{pe}), ms</td>
<td>120±9</td>
<td>172±13*</td>
<td>167±13*</td>
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<tr>
<td>(WT_{tot}), mm</td>
<td>11.5±0.4</td>
<td>7.7±0.5‡</td>
<td>11.5±0.5</td>
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<tr>
<td>(SWT), %</td>
<td>37±3</td>
<td>-7±3‡</td>
<td>-2±5*</td>
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<tr>
<td><strong>Normal Remote Segment (Inferior Wall)</strong></td>
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<td>1.3±0.1</td>
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<tr>
<td>(\varepsilon_{ps}), %</td>
<td>32±3</td>
<td>29±3</td>
<td>26±2</td>
</tr>
<tr>
<td>(\varepsilon_{sys}), %</td>
<td>2.2±1.2</td>
<td>4.4±1.3</td>
<td>2.3±1.3</td>
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<tr>
<td>(\varepsilon_{sys}), %</td>
<td>10.3±1.5</td>
<td>10.8±1.3</td>
<td>7.9±1.9</td>
</tr>
<tr>
<td>(T_{pe}), ms</td>
<td>118±14</td>
<td>128±11</td>
<td>123±13</td>
</tr>
<tr>
<td>(WT_{tot}), mm</td>
<td>11.0±0.4</td>
<td>9.6±0.6</td>
<td>10.9±0.6</td>
</tr>
<tr>
<td>(SWT), %</td>
<td>35±2</td>
<td>23±3</td>
<td>27±3</td>
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</table>

Transmural infarct group

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Occlusion</th>
<th>Reperfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>(S_{ps}), s⁻¹</td>
<td>1.7±0.2</td>
<td>0.2±0.1†</td>
<td>0.2±0.1§</td>
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<tr>
<td>(E_{ps}), s⁻¹</td>
<td>-2.8±0.4</td>
<td>-0.5±0.1[]</td>
<td>-0.3±0.1[]</td>
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<td>(A_{ps}), s⁻¹</td>
<td>-2.1±0.4</td>
<td>-0.6±0.2[]</td>
<td>-0.3±0.1[]</td>
</tr>
<tr>
<td>(\varepsilon_{ps}), %</td>
<td>36±4</td>
<td>-2±1†</td>
<td>1±0†</td>
</tr>
<tr>
<td>(\varepsilon_{sys}), %</td>
<td>0.8±0.5</td>
<td>6.4±1.4[]</td>
<td>2.3±1.0§</td>
</tr>
<tr>
<td>(\varepsilon_{sys}), %</td>
<td>11.1±2.2</td>
<td>2.1±0.6†</td>
<td>1.1±0.2†</td>
</tr>
<tr>
<td>(T_{pe}), ms</td>
<td>115±6</td>
<td>138±6[]</td>
<td>143±19†</td>
</tr>
<tr>
<td>(WT_{tot}), mm</td>
<td>11.9±0.3</td>
<td>10.4±0.5[]</td>
<td>15.5±0.5[]</td>
</tr>
<tr>
<td>(SWT), %</td>
<td>39±4</td>
<td>-2±2†</td>
<td>1±2†</td>
</tr>
</tbody>
</table>

\(S\) indicates systole; \(E\), early diastole; \(A\), late diastole; \(\varepsilon_{ps}\), end-systolic strain; \(\varepsilon_{sys}\), postsystolic strain; \(\varepsilon_{sys}\), diastolic strain during late LV filling; \(T_{pe}\), time to onset of \(E_{SR}\); \(WT_{tot}\), end-diastolic wall thickness; and \(SWT\), systolic wall thickening measured from gray-scale M-mode images.

*P<0.001, †P<0.0001, ‡P<0.05 vs remote wall and baseline; §P<0.001, ||P<0.0001, ¶P<0.05 vs stunned wall.
similar LV pressure at the time of onset of $A_{SR}$ and end-diastole ($P=NS$ for all). Figure 4 illustrates examples of data from 2 animals.

**Loading Alterations and Myocardial Stiffness**

Caval constriction reduced $\varepsilon_4$ (stunned, 5.2%; remote, 5.5%; infarcted, 0.9%; remote, 3.3%) and systolic and end-diastolic LV pressure (by $\approx 35$ and 5 mm Hg, respectively). Saline infusion increased $\varepsilon_4$ (stunned, 14.5%; remote, 17.5%; infarcted, 2.4%; remote, 12.5%) and end-diastolic LV pressure (by 5 to 8 mm Hg). Consequently, changes in $\varepsilon_4$ with loading were proportional in stunned and remote walls, but $\varepsilon_4$ changed only in normal walls in hearts with infarct.

Figure 5 shows the diastolic pressure–wall thickness relationships from all tested animals. For stunned myocardial walls, diastolic pressure–wall thickness relationships shifted slightly leftward or rightward with reperfusion compared with baseline, with minor change in slope ($\beta$-coefficient: 0.38±0.09 versus 0.28±0.07 and 0.28±0.09, stunned versus remote wall and baseline, respectively; $P=NS$ for all). For infarcted walls, diastolic pressure–wall thickness relationships shifted markedly rightward at reperfusion, with a significant increase in slope, indicating increased myocardial stiffness ($\beta$-coefficient: 0.50±0.09 versus 0.19±0.03 and 0.17±0.02, infarcted versus remote wall and baseline value; $P<0.05$ for both). End-diastolic wall thickness was similar at baseline in the 2 groups but increased significantly at reperfusion in walls with infarct (Table 2).

**Discussion**

This is the first study to address the relationship between myocardial elastic properties and passive diastolic deformation measured by strain echocardiography. We demonstrate that reperfused transmurally infarcted myocardium has markedly reduced diastolic deformation (and rates of deformation) due to increased myocardial stiffness. In addition, we show that stunned myocardium has nearly normal passive diastolic deformation (and rates of diastolic deformation), consistent with preserved tissue compliance.

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**Figure 3.** A, Diastolic strain $\varepsilon_4$ at reperfusion. B, Relative strain index at baseline and reperfusion. Values of $\approx 1$ indicate that ischemic and remote walls are equally stretched. Bars indicate mean values. *$P<0.0001$ vs remote and stunned walls.

**Figure 4.** Typical example of data at reperfusion from an animal with stunning and an animal with transmural myocardial infarction. A, Top, SR images in short-axis view at time of peak SR during late diastole ($A_{SR}$). Middle, Parametric images of late diastolic strain ($\varepsilon_4$) calculated for the whole sector scan. Only myocardial walls that move parallel with the ultrasound beams and are indicated by yellow arrows can be compared correctly. Note that stunned (anterior) myocardial wall exhibits magnitude of $A_{SR}$ and $\varepsilon_4$ comparable to that of the remote (inferior) wall. Conversely, infarcted wall stretches significantly less (and with lower SR) than corresponding remote wall. Bottom, Stained cardiac specimen showing the extent of viable and infarcted myocardium. B, Corresponding transmural SR and strain of ischemic/reperfused segments (anterior wall). Although systolic parameters ($S_{SR}$, $A_{SR}$, $\varepsilon_4$) were similarly reduced, diastolic parameters ($E_{SR}$, $A_{SR}$, $\varepsilon_4$) were higher in the stunned wall.

**Figure 5.** Diastolic pressure–wall thickness relationships in all animals tested. The best-fit exponential curves are shown. Note the rightward shift and increase in slope of diastolic pressure–wall thickness relationships for infarcted reperfused walls. LVEDP indicates LV end-diastolic pressure; $WT_{ed}$ end-diastolic wall thickness.
Diastolic Strain and SR Parameters

Although systolic strain was reduced equally in both stunned and infarcted myocardium, the magnitudes of diastolic strain and SR were higher in the former. The onset of relaxation was delayed in stunned walls, caused by presence of postsystolic thickening and indicated by delayed onset of ESR; however, passive diastolic deformation and stiffness were relatively preserved, as confirmed by analysis of diastolic pressure–wall thickness relationships. In a model of stunning induced by demand ischemia, investigators found relaxation abnormalities but no change or only a slight increase in myocardial stiffness.20,21 Complete functional recovery is expected, as indicated by the lack of necrotic tissue at histochemical staining.22,23

Conversely, for transmurally infarcted myocardium, we found severely altered passive diastolic strain and SR during both early and late diastole, indicating abnormalities of both relaxation and compliance. This finding was consistent with the marked increase in stiffness shown by diastolic pressure–wall thickness relations. The increased diastolic stiffness should reduce the deformation in all cardiac phases, including systole and early diastole. Hence, in conditions of reduced late diastolic filling, peak ESR alone could convey similar information (Figure 2).

Previous studies employing strain echocardiography have demonstrated the reduction in regional systolic SR and strain in acute and chronic MI7,10–15 as well as stunning,24,25 which agrees with our findings of higher S SR (as a mean) in stunned myocardial walls. The reduction in diastolic SR has also been briefly reported.11–13 Our study is the first to validate the behavior of regional diastolic SR and strain parameters against conventional indices of myocardial stiffness.

Myocardial Stiffness and Relative Diastolic Strain

The magnitude of passive deformation was load dependent. Indices of myocardial stiffness of diastolic pressure–wall thickness relation overcome this limitation, but this approach is not realistic for clinical assessment. A surrogate approach is to compare different segments within the same heart and under the same loading conditions. Accordingly, the relative strain index disclosed the magnitude of difference in compliance between segments, relatively independent of loading condition.

Importantly, we found that reperfused infarcted myocardial walls have less than one fourth of passive deformation (therefore compliance) of remote normal walls, whereas stunned myocardial walls deform comparably to normal walls. Our results agree with previous studies, demonstrating the increase in myocardial stiffness after an MI.4,5,16,26,27 Another important finding is the large-amplitude postsystolic thickening in viable myocardial walls. An active mechanism (delayed contraction or relaxation) would be consistent with the reduced but persistent S SR and normal diastolic deformation found in stunning. A totally passive mechanism (recoil caused by the systolic stretch of elastic fibers) would also be valid. We propose that the increase in stiffness that occurs with infarction is responsible for the reduction in postsystolic thickening.

The increase in stiffness results from excessive edema,28,29 hypercontracture,30 and cellular and hemorrhagic infiltration.31 These factors explain why infarcted walls were thicker as well as stiffer than stunned walls. Mild edema may occur in animals with stunning,22 potentially causing a small increase in stiffness.29 If this was the case in our study, its effects were minor (nonsignificant) on radial diastolic strain/SR parameters and wall thickness. More studies are necessary to draw a definite conclusion on this issue. In old MI, the increase in stiffness has been attributed to collagen deposition and fibrous remodeling.31

Limitations

Open-chest preparation may reduce loading and diastolic SR,24 but this effect is minimized by comparing segments within the same heart. This approach, however, is relevant for subjects who have both injured and normal myocardium. Mitral inflow velocities were not assessed. More studies are needed to investigate longitudinal deformation. Stiffness is defined by stress-strain relationship; in our analysis, we used LV pressure as a fair approximation of wall stress.32

In this study we chose 2 extremes (stunning and infarct) to validate a concept. More studies are necessary to relate myocardial stiffness to infarct transmurality. If confirmed in patients, these results have clinical implications, for instance, to identify subjects who would benefit from revascularization. Information on tissue elasticity could complement the assessment of contractile properties with the use of strain echocardiography.

Acknowledgments

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References

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