Noninvasive Quantification of the Contractile Reserve of Stunned Myocardium by Ultrasonic Strain Rate and Strain

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Background—We sought to investigate ultrasonic strain rate and strain as new indices to quantify the contractile reserve of stunned myocardium during dobutamine infusion.

Methods and Results—Stunning of the left ventricular posterior wall was induced in 9 closed-chest pigs after 30 minutes of severe hypoperfusion followed by 60 minutes of reperfusion of the left circumflex coronary artery territory. A second group of 7 animals had no coronary occlusion and served as normal controls. An incremental dobutamine infusion protocol was used in both groups. Changes in regional radial function were monitored by use of ultrasound-derived maximal systolic radial strain rate (SR) and systolic strain (ε). In the control group, dobutamine induced an increase in both SR and maximal dP/dt, which correlated linearly (r=0.85). Conversely, ε values increased at low doses of dobutamine (2.5 to 5 μg · kg⁻¹ · min⁻¹) but decreased during higher infusion rates (10 to 20 μg · kg⁻¹ · min⁻¹). During circumflex hypoperfusion, SR and ε of the posterior wall decreased from 5.0±0.3 s⁻¹ and 63±6% to 2.9±0.3 s⁻¹ and 27±4%, respectively (P<0.01). After 60 minutes of reperfusion, SR and ε failed to fully resume because of stunning, averaging 3.6±0.2 s⁻¹ and 35±3%, respectively (P=0.12 versus ischemia, P<0.05 versus baseline). During dobutamine infusion, SR increased at 5 μg · kg⁻¹ · min⁻¹ and exceeded baseline values at 20 μg · kg⁻¹ · min⁻¹ (P<0.05), whereas ε increased only at high doses and remained below baseline levels (P<0.05).

Conclusions—The changes in regional function of stunned myocardium during inotropic stimulation could be characterized by use of ultrasonic deformation parameters. During dobutamine infusion, strain-rate values quantified the contractile reserve better than strain values. (Circulation. 2001;104:1059-1065.)

Key Words: stunning, myocardial stress echocardiography

Myocardial reperfusion after a reversible ischemic insult results in a prolonged contractile dysfunction and delayed recovery due to myocardial stunning.1,2 Stunned myocardium has been shown to retain significant contractile reserve in response to a variety of positive inotropic stimuli and pharmacological agents.3–7 The response of dysfunctional myocardium to dobutamine is widely used to identify viable myocardium during clinical stress testing monitored by echocardiography.8,9 Clinically relevant stress-induced changes in regional function, however, might occur below the threshold of visual detection.10,11 This could result in a less than optimal diagnostic accuracy of stress echocardiography for the prediction of postischemic myocardial function recovery.12,13 Regional strain rate and strain can now be quantified noninvasively from B-mode ultrasonic data acquisitions.14 The accuracy of such segmental deformation measurement has been validated during experimental ischemia by comparing ultrasound data with sonomicrometry.15 Furthermore, several reports have demonstrated the potential clinical value of this technique in the quantitative assessment of regional myocardial asynergy.16,17 On the basis of these preliminary findings, we postulated that strain rate and strain could quantify the changes in regional myocardial function during inotropic stimulation.

The aim of this study was to evaluate the ability of ultrasonic deformation indices to characterize and quantify the changes in segmental function of stunned versus normal myocardium during an incremental dobutamine challenge in closed-chest pigs.

Methods
Nineteen male crossbred pigs (27 to 32 kg) were anesthetized with intravenous propofol (0.3 mg · kg⁻¹ · min⁻¹) and fentanyl (0.5 μg · kg⁻¹ · min⁻¹), intubated, and ventilated with a mixture of air and

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measuring the spatial velocity gradient over a computation area of 4 mm. An operator-selected region of interest was positioned within the posterior and septal walls to derive regional velocity and strain-rate profiles.

The timing of end systole (aortic valve closure) and end diastole (onset of isovolumic contraction) were derived from the septal myocardial velocity profile. Both mechanical events induce identifiable notches in the velocity curve that correlate with the timing of peak negative LV dP/dt and the rapid upstroke of LV dP/dt data, respectively. Strain-rate profiles were averaged over 3 consecutive cardiac cycles with custom-made software (Speqle, K.U. Leuven, Belgium). Natural strain profile was obtained by integrating the mean strain-rate values over time with end diastole as the reference point. From the averaged strain-rate and strain data, peak systolic strain rate (SR) and systolic strain (ε) were measured.

The intraobserver and interobserver variabilities (in percentage of the mean) averaged 11.1±3% and 13.4±4% for SR and 8.1±4% and 10.2±4% for ε.

After the color myocardial velocity data had been subtracted from the clips, the underlying digital gray-scale B-mode data could be displayed in cine loop format. The myocardial wall end-diastolic (EDT) and end-systolic (EST) thickness and LV diameter were measured from an anatomic M-mode tracing. Myocardial systolic wall thickening (WT) was calculated as WT=(EST−EDT)/EDT.

**Statistical Methods**

Data are presented as mean±SEM. Multiple comparisons were performed by ANOVA with post hoc Duncan's test. Least-squares or multiple regression analysis was used to investigate the relations between echocardiographic and hemodynamic parameters. Statistical significance was inferred for P<0.05.

**Results**

Data from all animals included in the control group (n=7) were analyzed. Three animals of the stunning group were excluded because of sustained reperfusion arrhythmia (n=2) and myocardial infarction (n=1). In the remainder (n=9), no macroscopic sign of myocardial necrosis was detected by triphenyltetrazolium chloride staining after 10 days of reperfusion. At the postmortem examination, the interrogated segment of the posterior wall was within the risk region in all animals.

The hemodynamic and LV dimension and thickness data are summarized in Tables 1 and 2. In comparison with baseline, the end-systolic thickness of the stunned posterior wall decreased, whereas LV diameter increased, resulting in an increased end-systolic σ. During dobutamine infusion,
end-systolic and end-diastolic $\sigma$ of the posterior wall decreased in both groups.

**Echocardiographic Data**

**Control Group**

At baseline, SR and $\epsilon$ of the posterior wall averaged $4.7 \pm 0.3$ s$^{-1}$ and $58 \pm 5\%$, respectively. The incremental dobutamine administration resulted in a gradual increase in SR (by $30\%$ at 1 and $58\%$, respectively). The incremental dobutamine infusion of the posterior wall decreased for low infusion rates (by $18\%$ at 2.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$, $P<0.05$), then decreased for high infusion rates and was not different from baseline values at $20 \mu g \cdot kg^{-1} \cdot min^{-1}$ ($65 \pm 4\%$, $P=0.47$ versus baseline, Figure 2).

**TABLE 1. Hemodynamic Data During Dobutamine Infusion**

<table>
<thead>
<tr>
<th></th>
<th>Heart Rate, bpm</th>
<th>$\frac{dP}{dt_{max}}$, mm Hg/s</th>
<th>Maximal LVP, mm Hg</th>
<th>End-Diastolic LVP, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (n=7)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>104±3</td>
<td>1592±96</td>
<td>104±5</td>
<td>13.2±1.4</td>
</tr>
<tr>
<td>Dobu 2.5</td>
<td>122±3*</td>
<td>2472±158*</td>
<td>111±7</td>
<td>11.4±1.5</td>
</tr>
<tr>
<td>Dobu 5</td>
<td>141±3*</td>
<td>3344±190*</td>
<td>117±7</td>
<td>8.1±1.2$^*$</td>
</tr>
<tr>
<td>Dobu 10</td>
<td>160±4*</td>
<td>3969±157*</td>
<td>118±4</td>
<td>6.0±1.1$^*$</td>
</tr>
<tr>
<td>Dobu 20</td>
<td>177±5*</td>
<td>4664±160*</td>
<td>124±5*</td>
<td>5.2±1.5$^*$</td>
</tr>
<tr>
<td>Recovery</td>
<td>108±3</td>
<td>1179±143</td>
<td>90±7</td>
<td>13.6±1.2</td>
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<tr>
<td>Stunning (n=9)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>104±9</td>
<td>1637±153</td>
<td>102±5</td>
<td>12.1±1.6</td>
</tr>
<tr>
<td>Stunning</td>
<td>106±5</td>
<td>1761±185</td>
<td>106±7</td>
<td>11.7±0.9</td>
</tr>
<tr>
<td>Dobu 2.5</td>
<td>120±5</td>
<td>2597±199$^+$</td>
<td>107±5</td>
<td>8.4±1.2$^*$</td>
</tr>
<tr>
<td>Dobu 5</td>
<td>134±5$^+$$^+$</td>
<td>3344±227$^+$</td>
<td>112±4</td>
<td>6.8±1.4$^+$$^+$</td>
</tr>
<tr>
<td>Dobu 10</td>
<td>147±2$^+$$^+$</td>
<td>3875±194$^+$</td>
<td>109±5$^*$</td>
<td>5.7±1.2$^+$$^+$</td>
</tr>
<tr>
<td>Dobu 20</td>
<td>175±5$^+$$^+$</td>
<td>4952±291$^+$</td>
<td>119±5$^+$$^+$</td>
<td>4.9±1.1$^+$$^+$</td>
</tr>
<tr>
<td>Recovery</td>
<td>106±7</td>
<td>1359±168</td>
<td>94±5</td>
<td>11.1±0.7</td>
</tr>
</tbody>
</table>

*Dobu indicates dobutamine infusion ($\mu g \cdot kg^{-1} \cdot min^{-1}$).

$^*$P<0.05 vs baseline.

†P<0.05 vs stunning.

**TABLE 2. LV Diameter, Posterior Wall Thickness, and Regional Meridional Stress During Dobutamine Infusion**

<table>
<thead>
<tr>
<th></th>
<th>LVEDD, cm</th>
<th>LVESD, cm</th>
<th>EDT, cm</th>
<th>EST, cm</th>
<th>$\sigma_{ES}$, g/cm²</th>
<th>$\sigma_{ES}$, g/cm²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (n=7)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>4.19±0.06</td>
<td>2.98±0.07</td>
<td>0.53±0.02</td>
<td>0.86±0.03</td>
<td>77±6</td>
<td>34±4</td>
</tr>
<tr>
<td>Dobu 2.5</td>
<td>4.15±0.13</td>
<td>2.83±0.07</td>
<td>0.53±0.02</td>
<td>0.92±0.03</td>
<td>67±8</td>
<td>28±4</td>
</tr>
<tr>
<td>Dobu 5</td>
<td>4.01±0.09</td>
<td>2.52±0.07$^*$</td>
<td>0.56±0.01</td>
<td>1.03±0.03$^*$</td>
<td>47±4$^*$</td>
<td>17±2$^*$</td>
</tr>
<tr>
<td>Dobu 10</td>
<td>3.90±0.16</td>
<td>2.50±0.12$^*$</td>
<td>0.55±0.01</td>
<td>1.01±0.04$^*$</td>
<td>49±7$^*$</td>
<td>13±3$^*$</td>
</tr>
<tr>
<td>Dobu 20</td>
<td>3.80±0.19$^*$</td>
<td>2.40±0.11$^*$</td>
<td>0.59±0.03$^*$</td>
<td>1.04±0.04$^*$</td>
<td>50±9$^*$</td>
<td>11±3$^*$</td>
</tr>
<tr>
<td>Recovery</td>
<td>4.16±0.15</td>
<td>3.2±0.15</td>
<td>0.52±0.02</td>
<td>0.8±0.03</td>
<td>70±7</td>
<td>33±4</td>
</tr>
<tr>
<td>Stunning (n=9)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>4.10±0.04</td>
<td>2.82±0.05</td>
<td>0.54±0.03</td>
<td>0.94±0.05</td>
<td>67±5</td>
<td>32±5</td>
</tr>
<tr>
<td>Stunning</td>
<td>4.10±0.12</td>
<td>3.30±0.17$^*$</td>
<td>0.45±0.58</td>
<td>0.61±0.01$^*$</td>
<td>151±13$^*$</td>
<td>31±3</td>
</tr>
<tr>
<td>Dobu 2.5</td>
<td>3.94±0.14</td>
<td>3.20±0.18$^*$</td>
<td>0.51±0.03</td>
<td>0.68±0.02$^*$</td>
<td>126±12$^*$</td>
<td>20±2</td>
</tr>
<tr>
<td>Dobu 5</td>
<td>3.98±0.11</td>
<td>3.09±0.10</td>
<td>0.52±0.03</td>
<td>0.72±0.03$^*$</td>
<td>101±11$^+$$^+$</td>
<td>16±3$^+$$^+$</td>
</tr>
<tr>
<td>Dobu 10</td>
<td>4.07±0.08</td>
<td>2.91±0.09$^+$</td>
<td>0.54±0.02</td>
<td>0.80±0.03$^+$</td>
<td>82±9$^+$</td>
<td>13±2$^+$$^+$</td>
</tr>
<tr>
<td>Dobu 20</td>
<td>3.85±0.05$^+$</td>
<td>2.37±0.05$^+$</td>
<td>0.57±0.06$^+$</td>
<td>0.95±0.07$^+$</td>
<td>36±3$^+$</td>
<td>11±2$^+$$^+$</td>
</tr>
<tr>
<td>Recovery</td>
<td>4.28±0.11</td>
<td>3.65±0.08$^*$</td>
<td>0.49±0.01</td>
<td>0.65±0.03$^*$</td>
<td>121±12$^*$</td>
<td>27±2</td>
</tr>
</tbody>
</table>

LVEDD and LVESD indicate LV end-diastolic and end-systolic diameter; EDT and EST, posterior wall end-diastolic and end-systolic thickness; and $\sigma_{ES}$, posterior wall end-diastolic and end-systolic meridional stress.

$^*$P<0.05 vs baseline.

†P<0.05 vs stunning.
SR correlated linearly with dP/dt max (r=0.85) over the whole investigated range of contractile states and heart rates (Figure 3A). In contrast, \( \varepsilon \) increased with dP/dt max for a heart rate <135 bpm but decreased for higher heart rates, despite the increase in the contractile state (Figure 3B).

**Stunning Group**

Figure 4 shows a typical example of the changes in the radial strain profile of the ischemic posterior (red curves) and the remote nonischemic segments (blue curves) during ischemia, stunning, and subsequent dobutamine infusion. LCx hypoperfusion resulted in a delayed onset and decreased magnitude of systolic thickening of the posterior wall. After aortic valve closure, the ischemic myocardium continued to thicken during the isovolumic relaxation period (postsystolic thickening), resulting in a delayed thickening peak. After 30 minutes of total LCx reperfusion, systolic thickening partially recovered but remained abnormal because of stunning. During dobutamine infusion, deformation profile normalized gradually. After 10 days of reperfusion, segmental function of posterior wall recovered totally. Myocardial staining confirmed matching of interrogated region with area at risk of LCx and absence of myocardial necrosis. AVC indicates aortic valve closure.

**Stunning Group**

Figure 4 shows a typical example of the changes in the radial strain profile of the ischemic posterior wall (red) and remote septum (blue) during circumflex artery hypoperfusion, reperfusion, and dobutamine (Dobu) challenge. Regional ischemia resulted in a decreased systolic strain and marked asynchronous and delayed contraction of posterior wall. Abnormal strain pattern persisted on reperfusion because of stunning. During dobutamine infusion, deformation profile normalized gradually. After 10 days of reperfusion, segmental function of posterior wall recovered totally. Myocardial staining confirmed matching of interrogated region with area at risk of LCx and absence of myocardial necrosis. AVC indicates aortic valve closure.

In the remote nonischemic segments, the systolic thickening increased slightly during acute ischemia but returned to
At baseline, SR and $\varepsilon$ of the posterior wall averaged 5.0±0.3 s$^{-1}$ and 63±6%, respectively, and were comparable to the normal values derived from the control group ($P=\text{NS}$). As shown in Figure 5A, SR and $\varepsilon$ values consistently decreased in the risk region during LCx hypoperfusion, by 41% and 57%, respectively ($P<0.01$). After reperfusion, SR and $\varepsilon$ tended to increase, from 2.9±0.3 to 3.6±0.2 s$^{-1}$ ($P=0.2$ versus ischemia) and from 27±4% to 35±3%, respectively ($P=0.12$ versus ischemia), but remained lower than baseline values ($P<0.05$). During dobutamine infusion, $\varepsilon$ averaged 44±3% at 5 $\mu$g·kg$^{-1}$·min$^{-1}$ ($P=0.15$ versus stunning) and increased significantly only at 20 $\mu$g·kg$^{-1}$·min$^{-1}$, to 50±3% ($P<0.01$ versus stunning), but remained inferior to baseline values ($P<0.05$). Conversely, SR increased to 4.7±0.3 s$^{-1}$ at 5 $\mu$g·kg$^{-1}$·min$^{-1}$ ($P<0.05$ versus stunning) and 7.0±0.6 at 20 $\mu$g·kg$^{-1}$·min$^{-1}$, exceeding baseline level ($P<0.05$ versus baseline). For each of the dobutamine administration stages, however, SR values remained significantly lower than those of the remote nonischemic segment or the normal values in the control group ($P<0.05$). On recovery, SR and $\varepsilon$ returned to the levels observed in stunned myocardium before dobutamine infusion.

After 10 days of reperfusion, SR and $\varepsilon$ of the posterior wall increased to 4.8±0.3 s$^{-1}$ and 59±4%, respectively, and were comparable to baseline values ($P=\text{NS}$), suggesting a total recovery of regional function of the initially stunned myocardium.

The multiple regression analysis investigating the hemodynamic predictors of SR and $\varepsilon$ values is shown in Figure 6. In both groups, SR but not $\varepsilon$ was significantly related to heart rate ($P<0.01$) as a marker of the myocardial inotropic state during dobutamine infusion.

**Strain Versus WT**

The time course of the changes in $\varepsilon$ paralleled the changes in WT of the posterior wall in both groups (Figures 2 and 5). By use of a multiple regression model, WT values could be predicted from $\varepsilon$ and SR data (WT=0.7×$\varepsilon$+0.011×SR$+0.082$, $R^2=0.62$, $P<0.001$) and showed a strong relation between WT and $\varepsilon$ ($P<0.001$) but not SR ($P=0.1$).

Figure 5. Stunning group: changes in maximal systolic strain rate, systolic strain, and systolic thickening of at-risk posterior wall (A) and remote septum (B) during dobutamine infusion. *$P<0.05$ vs baseline; †$P<0.05$ vs stunning; ‡$P<0.01$ vs stunning.

Figure 6. Relation between SR, $\varepsilon$, and hemodynamic parameters (heart rate [HR], end-systolic stress [ves], and end-diastolic stress [ved]) by multiple regression analysis. Predicted values are plotted vs measured values in both control and stunning groups.
Discussion

Contractile Reserve of Stunned Myocardium

The finding that the contractile function of stunned myocardium can be enhanced with inotropic stimuli is not new.3–7 For the first time, however, this study has quantified, by use of SR and ε, the response of stunned myocardium to an incremental dobutamine infusion in a closed-chest animal model that mimics the clinical setting.

In contrast to stunning, acutely ischemic myocardium has a different response during inotropic stimulation characterized by a further deterioration in regional function.5,18 Dobutamine infusion, if applied to a myocardial segment with persisting acute ischemia, does not modify the maximal systolic strain rate and in fact induces a decrease in regional systolic strain with a concomitant increase in postsystolic thickening.18 Thus, the normalization of the strain curve (Figure 4) during dobutamine infusion and the increase in both strain rate and strain could differentiate between reperfused but stunned and still ischemic myocardium.

Derumeaux et al21 showed that despite persistent stunning, the maximal systolic myocardial velocity gradient (ie, strain rate) recovers significantly after reperfusion in comparison with totally ischemic myocardium. Our results are consistent with those findings and suggest that even at rest, segmental strain rate and strain could help to differentiate between postischemic reversible dysfunction and acute ischemia. In our study, however, there was an important overlap between the values of deformation parameters in subtotal ischemia and stunning. Thus, it is likely that the identification of stunning would benefit from the quantification of the contractile reserve during inotropic stimulation.

For stunned myocardium, all previous reports have investigated the regional magnitude of deformation (ie, strain) by use of microcrystals3–5,7 or echocardiography.4 Systolic strain values, however, not only are influenced by the regional contractile function but also are modified by changes in heart rate (Figure 3). The decrease in systolic strain for high heart rates could be due to the shortening of the ejection period, to the decrease in preload, or to diastolic filling.22 In contrast, maximal systolic strain rate (deformation rate), in normal myocardium, correlated linearly with maximal dP/dt, the latter being used as the gold standard for the assessment of changes in global LV function (although dP/dt may be affected by heart rate22). Furthermore, in both normal and stunned myocardium, the dobutamine-induced increase in SR preceded the increase in ε. This is in agreement with a previous study by Gorcsan et al,23 who showed that during low-dose dobutamine infusion in normal subjects, the increase in the maximal systolic myocardial velocity gradient precedes the change in myocardial thickening.

Both preload and afterload are decreased during dobutamine infusion.20 This could consistently modify regional deformation measurements.15,24 Our results showed that both systolic strain rate and strain are inversely related to the end-systolic stress used as a marker of changes in afterload (Figure 6). This load dependency should be accounted for when deformation parameters are used to identify potential changes in contractility.

Implications for Noninvasive Imaging During Stress Tests

In an attempt to overcome the limitations of visual wall motion scoring, several noninvasive quantitative imaging methods have been developed and tested during stress tests.25–28 Myocardial velocities25 and color kinesis27 investigate myocardial and endocardial motion, respectively. Such methods based on motion detection may be influenced by tethering to adjacent segments.15 In contrast, magnetic resonance tagging has the unique potential to quantify 3D myocardial deformation28 and can accurately measure peak regional myocardial strains despite a relatively low temporal resolution. During regional ischemia, however, concomitant with the reduction in systolic thickening, maximal deformation is delayed until after aortic valve closure. By measuring this latter parameter, one can underestimate the severity of an ischemic insult.18 In addition, the results of the present study suggest that maximal systolic strain-rate values might reflect the regional contractile status better than systolic strain.

Therefore, the estimation of peak strain rates and the timing of regional versus global events are necessary for the accurate assessment of ischemia-induced changes in regional function. To measure these parameters, the temporal resolution of the imaging technique becomes critical.

Study Limitations

There are several technical limitations in the postprocessing of strain rate and strain from B-mode ultrasonic data acquisitions. First, the software used in this study does not allow the transmural assessment of myocardial deformation in the different myocardial layers. Thus, we could not resolve the different patterns of functional changes in the subepicardial and subendocardial layers of normal and ischemic myocardium.21,29,30 Second, strain rate can be computed either from the spatially averaged velocity gradient as used in our study or from the slope of the linear regression line of each transmural velocity data point.23 Both approaches make the assumption of a predominantly linear distribution of transmural velocities, which might hold for normal23 but not for ischemic myocardium.21,30 Finally, the problem of angle dependency is common to all quantitative Doppler-based techniques.15 In this work, care was taken to keep the ultrasonic beam perpendicular to the interrogated segment to minimize the influence of the insonation angle.

Conclusions

Strain-rate imaging appears to be a promising technique for the quantitative assessment of dobutamine-induced changes in regional function. The combined analysis of the deformation pattern by use of strain profiles and the quantitative strain-rate measurements should be a helpful tool for the identification of reversible myocardial dysfunction.

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References
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