Strain Rate Imaging for Assessment of Regional Myocardial Function

Results From a Clinical Model of Septal Ablation

Theodore P. Abraham, MD; Rick A. Nishimura, MD; David R. Holmes, Jr, MD; Marek Belohlavek, MD, PhD; James B. Seward, MD

Background—Regional myocardial function assessment is essential in the management of coronary artery disease (CAD). Tissue Doppler imaging (TDI) by depicting local myocardial motion can potentially quantify regional myocardial function. Strain rate imaging (SRI) that depicts regional deformation is less susceptible to cardiac translation and tethering and may be superior to TDI for regional function analysis. We examined regional myocardial function using TDI and SRI in a unique clinical model of a small, discrete myocardial infarction.

Method and Results—Ten patients with severely symptomatic septal hypertrophy underwent basal septal ablation via intracoronary alcohol injection and had TDI and SRI pre- and postablation. Invasive hemodynamics showed no appreciable change in global function. Peak systolic strain rate was significantly lower postablation versus preablation (−0.5 versus −1.2 s⁻¹, P<0.001) and when comparing infarct and noninfarct areas (−0.5 versus −1.5 s⁻¹, P<0.001). In contrast, peak systolic tissue velocities were similar pre- and postablation (3.9 versus 2.9 cm/s, P=0.16) and between infarct and noninfarct areas (2.9 versus 2.2 cm/s, P=0.13). SRI analysis demonstrated reduced systolic function in the peri-infarct zone and preserved systolic function in the remote nonischemic zone.

Conclusion—In the clinical setting of a small, discrete infarct unaccompanied by changes in global function, SRI accurately depicted changes in regional function. These data suggest that SRI may be the optimal method for objective, quantitative assessment of regional myocardial dysfunction. (Circulation. 2002;105:1403-1406.)

Key Words: imaging • tissue • myocardium

There is currently no optimal method for quantifying regional myocardial systolic dysfunction by echocardiography. Tissue Doppler imaging (TDI)1,2 depicts local myocardial motion and may enable quantitation of myocardial function. Because TDI is influenced by cardiac translational motion and myocardial tethering, it may be imprecise when discrete areas of the myocardium are involved. Strain rate imaging (SRI), which describes myocardial deformation, is theoretically less susceptible to translation or tethering and is potentially superior to TDI in regional myocardial function assessment.3,4 The optimal model to compare the ability of TDI and SRI to assess regional myocardial function requires a localized infarction that does not change global function. Septal ablation for treatment of patients with hypertrophic obstructive cardiomyopathy (HCM) results in a discrete area of infarction. We evaluated regional function assessment by TDI and SRI in this unique clinical model of a myocardial infarction.

Methods

Patients
The study group consisted of consecutive patients undergoing septal ablation for severe symptomatic HCM (resting outflow tract gradient >50 mm Hg) from January 2000 to September 2000. Ten healthy volunteers served as controls.

Cardiac Catheterization
Alcohol ablation was performed as described elsewhere.5 Patients who came to the catheterization laboratory were fasting and under light sedation. A temporary pacemaker was placed in the apex of the right ventricle in all patients. The target septal perforator artery was selected via coronary angiography (Judkins technique) and contrast echocardiography (Optison, Mallinckrodt Inc). A septal perforator artery was selected if the myocardial segment enhanced by the contrast injection into that artery was the site of contact from the systolic anterior motion of the mitral valve leaflets. One to 3 mL of absolute alcohol was injected into the distal end of the inflated balloon in the septal perforator artery.

Invasive Hemodynamics
Pressures were measured in the left ventricle (LV), aorta (AO), and left atrium using high fidelity manometer-tipped pressure transducer catheters (Millar Instruments) inserted through a transseptal approach as previously described.6 Pressures were directly recorded in a digital format at 5 ms intervals during held end expiration, before and after the ablation.
Echocardiography

Apical imaging, before and after ablation, was performed using a 2.5 MHz phased array probe with a System FiVe ultrasound machine (GE Vingmed). Single walls were imaged using a narrow sector angle with the wall parallel to the ultrasound beam.

Image Analysis

Analysis was performed by 2 independent, blinded observers using custom software (TVI 16.3b, GE Vingmed). Myocardial motion was coded such that in TDI, motion toward and away from the transducer was coded red and blue, respectively, and in SRI, yellow-red indicated shortening and blue-white indicated lengthening (Figure 1a and 1c, respectively). Color M modes and peak systolic velocities (TV) and strain rates (SR) were obtained by placing the cursor at the midmyocardial level in each wall. Tissue velocities were also normalized to end diastolic ventricular length. The infarct zone was the area highlighted by intramyocardial contrast. To examine changes in myocardial function around the infarct, SR was measured in the peri-infarct zone (midseptum) and in a nonischemic zone (apical septum). All measurements were taken from an acceptable tracing, and the final value was the average of 3 measurements.

Statistics

Continuous variables are expressed as mean ± SD. Pre- and postablation hemodynamic, TDI, and SRI variables are compared using a 2-tailed independent student’s t-test. A P value < 0.05 was considered significant. Peak values within 2 SD of the mean for normals were set as the cutoff for sensitivity, specificity, and accuracy calculations.

Results

Septal infarct was characterized by a reduction in LV outflow tract gradient and septal artery occlusion on angiography. All invasive indices of systolic and diastolic function remained unchanged (Table 1). Peak systolic SR was lower post versus preablation, whereas pre- and postablation peak systolic TV (raw and normalized) were similar (Table 1). Also, SR was lower in the infarct segments versus normal segments (0.5 versus 1.5 s⁻¹; P < 0.01). Peak systolic TV was similar in infarct and normal segments (raw: 3.0 versus 2.4 cm/s, P = 0.13; normalized: 0.30 versus 0.32, P = 0.4). Sensitivity, specificity, and accuracy of SRI and TDI are presented in Table 2. Systolic SR indicated reduced function in the peri-infarct zone (−0.7 ± 0.2 s⁻¹) and normal function in the nonischemic zone (−1.2 ± 0.14 s⁻¹; Figure 2). No postinfarct increase in systolic SR was observed in the normal zone (preinfarct versus postinfarct: −1.3 versus −1.2 s⁻¹, P = 0.09).

Discussion

This study demonstrates the ability of SRI to accurately depict regional myocardial function in a clinical model of a small myocardial infarction. Infarct segments demonstrated lower SR and absence of a shortening pattern postablation. SRI analysis also demonstrated an area of systolic dysfunction surrounding the infarct with no augmentation of contractile function in the normal zone. The myocardial infarction in this study was of a size that measurements were not confounded by global changes in systolic or diastolic function.

There has been considerable interest in the ability to noninvasively diagnose and quantify regional myocardial function around the infarct, SR was measured in the peri-infarct zone (midseptum) and in a nonischemic zone (apical septum). All measurements were taken from an acceptable tracing, and the final value was the average of 3 measurements.

![Figure 1. Color M-mode images of the septum.](image-url)

**TABLE 1.** Hemodynamic, TDI, and SRI Data

<table>
<thead>
<tr>
<th></th>
<th>Preablation</th>
<th>Postablation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean BP, mm Hg</td>
<td>89 ± 9</td>
<td>100 ± 11</td>
</tr>
<tr>
<td>LVEDP, mm Hg</td>
<td>22 ± 9</td>
<td>19 ± 10</td>
</tr>
<tr>
<td>dp/dtmax, mm Hg/s</td>
<td>1677 ± 263</td>
<td>1333 ± 46</td>
</tr>
<tr>
<td>τ, ms</td>
<td>0.064 ± 0.014</td>
<td>0.066 ± 0.016</td>
</tr>
<tr>
<td>Normalized TV, s⁻¹</td>
<td>0.43 ± 0.11</td>
<td>0.34 ± 0.09</td>
</tr>
<tr>
<td>Obs 1</td>
<td>3.9 ± 1.0</td>
<td>2.9 ± 0.8</td>
</tr>
<tr>
<td>Obs 2</td>
<td>4.3 ± 0.8</td>
<td>3.5 ± 0.9</td>
</tr>
<tr>
<td>Preablation</td>
<td>−1.2 ± 0.14</td>
<td>−0.5 ± 0.2</td>
</tr>
<tr>
<td>Postablation</td>
<td>−1.5 ± 0.2</td>
<td>−0.6 ± 0.25</td>
</tr>
</tbody>
</table>

BP indicates blood pressure; LVEDP, left ventricular end diastolic pressure; dp/dtmax, peak positive dp/dt; τ, time constant of relaxation; TV, peak systolic tissue velocity from infarct area; normalized TV, TV normalized to end diastolic ventricular length; SR, peak systolic strain rate from infarct area; and Obs 1 and 2, blinded observers.
TABLE 2. Sensitivity, Specificity, and Accuracy of SRI and TDI

<table>
<thead>
<tr>
<th>Observer</th>
<th>Sensitivity, %</th>
<th>Specificity, %</th>
<th>Accuracy, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-peak</td>
<td>60</td>
<td>100</td>
<td>65</td>
</tr>
<tr>
<td>2-peak</td>
<td>40</td>
<td>100</td>
<td>70</td>
</tr>
<tr>
<td>1-color</td>
<td>30</td>
<td>97</td>
<td>78</td>
</tr>
<tr>
<td>2-color</td>
<td>30</td>
<td>86</td>
<td>83</td>
</tr>
</tbody>
</table>

Peaks indicate peak systolic velocity and strain rate; color, color M mode (visual).

contraction. In TDI, the high frequency, low amplitude blood pool echoes are filtered out, resulting in selective measurement of myocardial velocities. Peak systolic TV has been used as a quantitative measure of systolic function. In SRI, however, is often unable to reliably distinguish active contraction from an akinetic segment that is “pulled” by the adjacent normal myocardium, thus erroneously demonstrating motion with respect to the transducer. Measurement of local tissue deformation (strain) may overcome these limitations of TDI. Strain is the change in length corrected for the initial length (ie, % deformation), and strain rate is the rate of normalized change. Strain was initially proposed as a measure of myocardial stiffness. In SRI, the difference in TV obtained from 2 points along the ultrasound beam is divided by the intervening distance to yield the spatial gradient of velocity, which is the strain rate. Absolute strain is calculated by integrating the strain rate signal. Peak systolic SR and strain may accurately reflect local systolic function because they measure myocardial deformation, not displacement. Doppler-derived strain rates have been validated in vitro and in vivo.

Prior clinical studies of TDI and SRI did not correct for changes in global systolic and diastolic function, and only indirectly assessed the area of infarction. In this study, the infarction did not cause changes in global hemodynamics, and the infarct was delineated by contrast echocardiography. We demonstrated the relative superiority of SRI over raw and normalized TDI in depicting changes in regional myocardial function after a small infarct. Furthermore, SRI analysis reveals reduced function peri-infarct and normal function in the remote non-ischemic myocardium. These data indicate that the region of myocardial dysfunction extends beyond the area of the infarct, there is a graded decrease in myocardial function from a remote nonischemic area toward the infarct, and there is no augmentation of systolic function in the non-ischemic myocardium. These findings are concordant with other studies of regional deformation in ischemia.

Limitations

Strain rate signal can be noisy and is influenced by the angle of insonation. The intra-operative study design precluded independent confirmation of strain rates using MRI.

In conclusion, TDI and SRI are novel techniques that enable quantitation of regional myocardial function. Using a unique clinical model of a small myocardial infarction, we demonstrated that assessment of myocardial deformation (SRI), not displacement (TDI), is an accurate indicator of regional function. In addition, SRI allows analysis of myocardial function in the peri-infarct and non-ischemic zones. Our data suggest that SRI may provide optimal objective assessment of regional myocardial function in coronary artery disease, although evaluation in more standard clinical settings will help determine its role in clinical practice.

References


Figure 2. Representative image illustrates clear delineation of the infarct segment by intramyocardial contrast (A) compared with the normal echodensity of the peri-infarct (B) and nonischemic segments (C). Mean systolic SR was low-absent in A, reduced in B, and normal in C. Mean systolic TV was similar in A and B and C. † indicates systole.


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