Myocardial Strain Rate Is a Superior Method for Evaluation of Left Ventricular Subendocardial Function Compared With Tissue Doppler Imaging

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OBJECTIVES This study was performed to evaluate subendocardial function using strain rate imaging (SRI).

BACKGROUND The subendocardium and mid-wall of the left ventricle (LV) play important roles in ventricular function. Previous methods used for evaluating this function are either invasive or cumbersome. Strain rate imaging by ultrasound is a newly developed echocardiographic modality based on tissue Doppler imaging (TDI) that allows quantitative assessment of regional myocardial wall motion.

METHODS We examined eight sheep using TDI in apical four-chamber views to evaluate the LV free wall. Peak strain rates (SRs) during isovolumic relaxation (IR), isovolumic contraction (IC), and myocardial strain were measured in the endocardial (End), mid-myocardial (Mid), and epicardial (Epi) layers. For four hemodynamic conditions (created after baseline by blood, dobutamine, and metoprolol infusion), we compared differences in SR of End, Mid, and Epi layers to peak positive and negative first derivative of LV pressure (dP/dt).

RESULTS Strain rate during IC showed a good correlation with +dP/dt (r = 0.74, p < 0.001) and during IR with −dP/dt (r = 0.67, p = 0.0003). There was a significant difference in SR between the myocardial layers during both IC and IR (End: −3.4 ± 2.2 s⁻¹, Mid: −1.8 ± 1.5 s⁻¹, Epi: −0.63 ± 1.0 s⁻¹, p < 0.0001 during IC; End: 2.2 ± 1.5 s⁻¹, Mid: 1.0 ± 0.8 s⁻¹, Epi: 0.47 ± 0.64 s⁻¹, p < 0.0001 during IR). Also, SRs of the End and Mid layers during IC were significantly altered by different hemodynamic conditions (End at baseline: 1.7 ± 0.7 s⁻¹; blood: 2.0 ± 1.1 s⁻¹; dobutamine: 3.4 ± 2.3 s⁻¹; metoprolol: 1.0 ± 0.4 s⁻¹; p < 0.05). Myocardial strain showed differences in each layer (End: −34.3 ± 12.6%; Mid: −22.6 ± 12.1%; Epi: −11.4 ± 7.9%; p < 0.0001) and changed significantly in different hemodynamic conditions (p < 0.0001).

CONCLUSIONS Strain and SR appear useful and sensitive for evaluating myocardial function, especially for the subendocardial region. (J Am Coll Cardiol 2003;42:1574–83) © 2003 by the American College of Cardiology Foundation

Tissue Doppler imaging (TDI) has been used to assess regional myocardial wall motion and ventricular function (1–3). The high temporal and spatial resolution of TDI enables evaluation based on display of the myocardial wall motion in two-dimensional guided M-mode or real-time two-dimensional images, with extracted values sampled off-line. The myocardial velocity gradient has been previously described as the gradient of velocities between the endocardium and epicardium (4,5); however, these evaluations were primarily from M-mode tracings.

It is well known that the subendocardium and mid-wall of the left ventricle (LV) play important roles in ventricular function (6–10). Previously performed studies required sonomicrometry with crystals implanted in the myocardium to evaluate the subendocardial or mid-wall function. Among noninvasive methods, magnetic resonance imaging tagging has been reported to be useful for such examinations and can provide accurate three-dimensional measurements (11,12).

Strain rate imaging (SRI) by ultrasound is a newly developed echocardiographic modality based on TDI that allows quantitative assessment of regional myocardial wall motion (13–17). The concept of strain and strain rate (SR) have been well delineated in other studies: SR is defined as the difference of tissue velocities between two distinct points along the scan line of the echo beam, and strain as the integration of SR (16–19). Strain and SR also represent tissue deformation, or the changing rate of tissue deformation. Several studies have suggested that strain and SR are good indicators for evaluation of LV systolic and diastolic function (13,16–20). Measurement of SR along the mid-septum or mid-LV lateral wall has been reported to be sensitive for estimation of LV global function (16–19,21,22).

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We have observed that SR showed significant alterations, especially during isovolumic contraction (IC) and isovolumic relaxation (IR) under different hemodynamic conditions, and assumed it was because the widest pressure changes occurred during IC and IR.

The present study was performed to evaluate subendocardial function using SRI during IC and IR during different hemodynamic conditions.

METHODS

Experimental preparation. Eight sheep (weight 35 to 47 kg [mean 40.1 ± 4.2]) were studied. All sheep underwent a median sternotomy under general anesthesia induced with intravenous sodium pentobarbital (25 mg/kg body weight) and maintained by using 1% to 2% isoflurane with oxygen. The sheep were intubated and ventilated with a volume-cycled mechanical ventilator. Intracavity manometer-tipped catheters (model SPC-350, Millar Instruments, Inc., Houston, Texas) were placed in the LV through a carotid artery and in the left atrium via the appendage for pressure recording. Peak positive and negative first derivative of LV pressure (dP/dt) values were obtained. Peak +dP/dt was used for the evaluation of global systolic function and peak −dP/dt was used for the evaluation of global diastolic function (19,22–25). Another catheter was positioned in the femoral artery to monitor systemic arterial pressure and blood gases. These catheters were interfaced with a physiologic recorder (ES 2000, Gould Inc., Cleveland, Ohio) with a fluid-filled pressure transducer (model PD231D, Gould Statham, Oxnard, California). All hemodynamic data were recorded at a paper speed of 250 mm/s. Four consecutive cardiac cycles were analyzed for each hemodynamic condition. All operative and animal management procedures were approved by the Animal Care and Use Committee of the National Heart, Lung, and Blood Institute, Bethesda, Maryland.

Experimental protocol. Baseline, volume loading, dobutamine infusion, and metoprolol infusion were used to produce a total of four different hemodynamic conditions for each sheep. After a baseline recording, 500 ml blood was infused, then intravenous dobutamine (2 to 10 μg/kg per min) and 5 mg metoprolol were administered at least 1 h apart from each other. All of the hemodynamic and myocardial velocity data (described subsequently) were acquired simultaneously at each hemodynamic stage. Mechanical ventilation was interrupted briefly during echocardiographic imaging.

Echocardiographic analysis. We used a Vivid FinVe digital ultrasound system (GE/VingMed Ultrasound, Horten, Norway) for the present study. Scanning was performed longitudinally from the apex to acquire apical four-chamber views with a 5.0-MHz phased-array transducer. The TDI data were acquired with a pulse repetition frequency of 1.0 to 4.5 kHz and a frame rate of 60 to 100 frames/s to minimize the noise level, depending on the heart rate. The TDI sector angle was limited to that required to encompass the LV cavity and walls, and line density and packet size were maximized. Resulting TDI sectors were 55° to 80°, with line spacing averaging 1.5° separation of sampling directions. With these settings, no aliasing of velocities was encountered. The TDI data for the two-dimensional images for two to three cycles at each stage were stored on a magnetic optical disk, with subsequent off-line analysis of stack-based digital data. Optimized TDI and SR data for the two-dimensional images of the heart at each stage were analyzed using the EchoPac 6.3 archiving application software of the Vivid FinVe. Also, myocardial strain of each layer was obtained by integration of SR over one cardiac cycle. This software allowed us to determine and display several different locations of tissue velocity or SR simultaneously for each of the hemodynamic conditions. We divided the LV lateral wall into three layers: the inner one-third (endocardial [End]), middle one-third (mid-myocardial [Mid]), and outer one-third (epicardial [Epi]). Measurements were taken from each layer, with a sampling volume size of 3 × 3 pixels and a sampling distance of 2.5 to 3.2 mm and at 5.0 to 7.0 cm depth from the transducer, and displayed as velocity and SR–time relationships without lateral averaging. Sample points separated by distances from 2.5 to 3.2 mm were used as the basis for calculating SR. Peak negative SR during IC and peak positive SR during IR were identified at each stage. Consistently, we observed three positive SR peaks during relaxation. The second peak (Fig. 1B, open arrow) corresponds to the early diastolic wave, and the third peak (Fig. 1B, solid arrow) corresponds to the late diastolic wave (26). We measured the first peak (isovolumic relaxation wave). Isovolumic contraction was determined by the Q-wave of the ECG and the closing of the mitral valve on the two-dimensional echocardiographic cine loop. Isovolumic relaxation was determined by the opening of the mitral valve of the cine loop. Peak velocities during IC and IR were also measured at each stage by TDI.

Autotracking is a method for keeping the sampling volume over the same area of tissue despite its motion. It is very time-consuming to use and requires placing the sample...
volume at different times on the cardiac model. We used this technique in most of our sequences, especially those in which, without tracking, the sample volume moved from one region to another or out of the myocardium entirely.

Statistics. All data are expressed as the mean ± SD. One-way repeated measures analysis of variance (ANOVA) followed by Dunnett’s post hoc test was used to compare SR and tissue velocity values between the baseline, volume-loaded, dobutamine, and metoprolol states. The results are expressed as p values. The SR and TDI observations in each of the myocardial layers (Epi, Mid, and End) were compared with the intracavity LV pressure recordings and expressed as the correlation coefficient (r) and p value. Linear regression analysis was used for comparison between the SR values and peak dP/dt. All statistical analyses were performed using StatView version 5.01 (SAS Institute, Cary, North Carolina). A p value <0.05 was considered statistically significant.

RESULTS

Figure 2 shows representative SR and TDI images during IC and IR. In SRI, tissue deformation is color encoded and two-dimensionally displayed as red (compression) or blue (expansion). Green-colored areas indicate a lack of deformation. The LV lateral wall was dark red on End and gradually faded toward Epi during IC (Fig. 2A, white arrows). On the other hand, the LV lateral wall was dark blue on End and gradually faded away toward Epi during IR (Fig. 2B, white arrows). However, with TDI (Figs. 2C and 2D), LV wall velocity encoding showed no significant color change or gradient from End to Epi. Figure 1 shows the
recordings of SR (B), strain (C), and tissue velocity (D) sampled from End, Mid, and Epi. We observed a definite negative deflection during IC and a positive peak during IR on SRI. Definite differences in SR values between layers could be observed both in IC and IR. Peak of the strain over one cycle showed a definite difference between layers, and a strain gradient was observed from the endocardium to epicardium. However, on TDI, although the peak of tissue velocity during IC and IR was recognized, differences between the layers were not significant.

Table 1 shows the hemodynamics related to each stage. Cardiac output significantly increased from 1.62 ± 0.47 to 2.2 ± 0.48 l/min by blood loading and to 2.0 ± 0.48 l/min with dobutamine infusion. The heart rate also significantly changed from 100 ± 12 to 142 ± 25 beats/min with dobutamine infusion. The LV end-diastolic pressure and mean left atrial pressure did not show a statistically significant change between stages. However, both peak +dP/dt and peak −dP/dt significantly changed (p < 0.05).

Figure 2 shows representative SR curves obtained from End, Mid, and Epi during each hemodynamic condition. There were no segmental wall motion abnormalities at baseline or during subsequent hemodynamic states. Figure 3A shows the baseline data. The positive peak wave of SR can be observed in End during IR. However, this positive peak wave became smaller in Mid and even smaller in Epi.

**Table 1. Hemodynamic Parameters During Each Hemodynamic Condition**

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Blood Loading</th>
<th>Dobutamine</th>
<th>Metoprolol</th>
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<tr>
<td>Cardiac output (l/min)</td>
<td>1.62 ± 0.47</td>
<td>2.2 ± 0.48*</td>
<td>2.0 ± 0.48</td>
<td>1.54 ± 0.24</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>100 ± 12</td>
<td>106 ± 6</td>
<td>142 ± 25*</td>
<td>81 ± 8*</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>12.1 ± 4.2</td>
<td>16.2 ± 6.2</td>
<td>11.1 ± 5.9</td>
<td>15.3 ± 2.1</td>
</tr>
<tr>
<td>LAP (mm Hg)</td>
<td>8.9 ± 3.0</td>
<td>13.5 ± 4.0*</td>
<td>9.1 ± 2.5</td>
<td>10.7 ± 2.9</td>
</tr>
<tr>
<td>Peak +dP/dt (mm Hg/s)</td>
<td>1,110 ± 324</td>
<td>1,697 ± 299*</td>
<td>2,260 ± 558*</td>
<td>735 ± 250</td>
</tr>
<tr>
<td>Peak −dP/dt (mm Hg/s)</td>
<td>−1,083 ± 183</td>
<td>−1,290 ± 137</td>
<td>−1,391 ± 295*</td>
<td>−667 ± 277*</td>
</tr>
</tbody>
</table>

*p < 0.05 compared with baseline. Data are presented as the mean value ± SD.

**dP/dt** = first derivative of left ventricular pressure; LAP = left atrial pressure; LVEDP = left ventricular end-diastolic pressure.
On the other hand, the negative peak wave of SR was observed in End during IC. This negative peak wave became smaller in Mid and even smaller in Epi. The peak waves of End and Mid were increased by blood loading and dobutamine infusion, but the SR of Epi did not change significantly with these hemodynamic alterations (Figs. 3B and 3C). Conversely, peak SR during IC and IR became smaller, in parallel with decreasing LV pressure (Fig. 3D).

Figure 4 shows the comparison of SR and TDI between End, Mid, and Epi. There was a significant difference in peak SR among the three layers during both IC and IR (p < 0.0001). Peak SR of End showed the greatest absolute values of the three layers (End: -3.4 ± 2.2 s⁻¹, Mid: -1.8 ± 1.5 s⁻¹, Epi: -0.63 ± 1.0 s⁻¹, p < 0.0001 during IC; End: 2.2 ± 1.5 s⁻¹, Mid: 1.0 ± 0.80 s⁻¹, Epi: 0.47 ± 0.64 s⁻¹, p < 0.0001 during IR). However, TDI measurements of the three layers showed that although the velocity of End was greater than that of Mid and Epi, this difference was not significant. Myocardial strain also showed a significant difference between layers and a gradient from End to Epi (End: -34.3 ± 12.6%, Mid: -22.6 ± 12.1%, Epi: -11.4 ± 7.9%, p < 0.0001 by analysis of variance [ANOVA]).

Figure 5A shows the SR data during IC for each hemodynamic condition. There was a significant difference in peak SR between the four different conditions both in End and Mid during IC (End: p < 0.0001; Mid: p = 0.0004 by ANOVA). Dobutamine infusion showed the
largest absolute peak SR. However, in Epi, there was no significant difference between peak SR in different hemodynamic stages. Figure 5B shows the SR data during IR for each hemodynamic condition. There was a significant difference in peak SR between the four different conditions both in End and Mid during IR (End: p < 0.05; Mid: p = 0.03 by ANOVA). However, there was no significant difference among them in Epi. Strain also showed a significant alteration between the four different hemodynamic conditions in each layer (End: p < 0.0001; Mid: p = 0.0001; Epi: p = 0.03 by ANOVA).

Figure 6 shows the relationship between absolute peak SR of End and peak dP/dt during IR and IC. There were significant correlations between peak SR and peak dP/dt not only during IC but also IR (y = 278x + 563, r = 0.74, p < 0.0001 during IC; y = 172x + 756, r = 0.67, p = 0.0003). Correlations between SR, TDI, and peak dP/dt during IC and IR are summarized in Table 2. Peak SR of End and Mid has a significant correlation with peak dP/dt both during IC and IR. However, peak SR of Epi does not have a significant correlation with peak dP/dt. For TDI, there was no significant correlation between tissue velocity and peak +dP/dt or −dP/dt.

### DISCUSSION

The aim of the present study was to evaluate myocardial and especially subendocardial function using SR compared with an invasive global evaluation of LV function. Despite its angle dependence, echocardiographic SR measurement is superior to magnetic resonance imaging with respect to spatial and temporal resolution (19,27). The high spatial resolution available permitted us to divide the wall into three layers for study. In the default instrument settings for TDI acquisition and SR measurement, information obtained from the GE/VingMed group (Horten, Norway) specifies that lateral resolution should be <3 mm at 5 to 7 cm when the transmit focus is placed at that depth in the Octave mode. Line spacing of 1.5° for tissue Doppler sampling lines over that sector would place the lines slightly
<2 mm apart at that depth. All algorithms for lateral and axial spatial, as well as temporal averaging were disabled in the scan presets. The wall thickness we scanned for SRI ranged from 8.2 to 15.6 mm (10.4±2.8) in diastole and 9.2 to 18.7 mm (12.8±3.8) in systole. It should theoretically be possible to differentiate three layers of the myocardium for SR analysis with these resolutions and settings.

Assessment of the LV layers has been previously available only by using invasive methods (i.e., sonomicrometry implantation into the myocardium) (19,27-29). However, noninvasive discrimination for the subendocardium and mid-wall has been desirable because the analysis yields findings that are clinically applicable (6,10,28,30,31). Magnetic resonance imaging can provide accurate information on myocardial deformation. The newest implementations of TDI have incorporated improved spatial and temporal resolution. However, tethering effects of adjacent myocardium and translational heart motion can cause misinterpretation of regional wall motion (19,26,32). The concept of a myocardial velocity gradient was developed to address these problems and enables the segmental analysis of the LV independent of global heart motion (4,5,33). However, evaluation by this method has been limited to two-dimensional short-axis measurements or derived M-modes, both of which lack the spatial selectivity for comprehensive segmental survey. As shown by our data, it is possible to evaluate segmental myocardial layers, differentiating subendocardium, mid-myocardium, and epicardium. A significant difference in SR values was observed between the three layers in the baseline condition and in response to hemodynamic alterations. This is consistent with previous reports (10,28,34,35).

A number of investigators have pointed out that SR profiles tend to be more noisy than TDI data and that they are Doppler angle dependent (19,22,26); therefore, SR amplitude is a measurement with problems of reproducibility. Abraham et al. (26) recommended using the timing of the phasic change of SR for evaluation of LV relaxation instead of SR amplitude. However, Greenberg et al. (20) and Jamal et al. (23) reported that amplitudes of SR have close relationships with maximal dP/dt derived from LV pressure and reflect LV contractility as long as regional wall motion is intact, because myocardial strain reflects the deformation of tissue in response to an applied force. Peak +dP/dt and −dP/dt serve as indicators of systolic and diastolic LV function, respectively (24,25,36-38). We mea-

Figure 5. A comparison in SR of End, Mid, and Epi during IC (A) and IR (B) for each hemodynamic condition. There was a significant difference in peak SR during the four different conditions in both End and Mid during IC and IR. Abbreviations as in Figures 1 and 2.
sured the amplitude of SR during IC and IR because tissue deformation is accelerated by the dynamic pressure change of the LV, and LV contractile and relaxation properties can be observed during these periods (39,40).

The Doppler angle dependency of SR tends to show different waveforms depending on the site of the wall and the Doppler angle. The waveform of SR scanned longitudinally from the apex shows the opposite phase of waveforms scanned in short-axis planes, because longitudinal tissue deformations are in the opposite direction of radial tissue deformation (16,19). Weidermann et al. (16) reported that radial SR values were more than double the longitudinal values. However, it has been shown that regional longitudinal deformation of the LV can differentiate segments with impaired myocardial function from normal segments (15–18,21). Three-dimensional strain analysis by magnetic resonance imaging tagging reveals the detailed myocardial fiber architecture and may offer a persuasive explanation for regional longitudinal deformation of the LV. It is reported that myocardial fiber angles with respect to the short-axis plane varied linearly from $-53 \pm 20^\circ$ at the epicardium to $87 \pm 25^\circ$ at the endocardium, so that subendocardial and epicardial myocardial fibers are oriented approximately at right angles to each other (41–43). The subendocardium is predominantly composed of longitudinal myocardial fiber. Shortening of the sarcomere does not exceed 15% and relates to regional longitudinal deformation. Although thickening of fiber is only 8%, thickening of the LV wall reaches 40%, because rearrangement of subendocardial myocardial fiber architecture, such as “cross-fiber shortening,” occurs in the radial orientation (41). Therefore, longitudinal subendocardial strain and SR may directly relate to the sarcomere shortening in the subendocardium. According to the concept in the helical heart model of Torrent-Guasp et al. (44), the descending segment, which constitutes the apical loop of the myocardial band, functions as endocardium. That study revealed that contraction of descending segment was synchronized with the phasic change of LV pressure and derived dP/dt. The relative merits of the longitudinal and cross-sectional approaches for SR assessment are still being debated. Our results are concordant with a recent tissue tagging study in normal subjects from the Johns Hopkins group, which shows that endocardial deformation is greatest in the longitudinal direction and verifies the endo-epicardial gradient in normal LVs on magnetic resonance imaging (11,43).

**Study limitations.** The sampling points we measured in the present study were close to each other, and a requisite for their spatial discrimination is a good signal-to-noise ratio and good lateral resolution, so that enough wall texture is available to differentiate wall layers without the sampling point extending beyond the three wall zones during IC and IR. However, a difference in the measurements’ angulation forms scanned in short-axis planes, because longitudinal tissue deformations are in the opposite direction of radial tissue deformation (16,19). Weidermann et al. (16) reported that radial SR values were more than double the longitudinal values. However, it has been shown that regional longitudinal deformation of the LV can differentiate segments with impaired myocardial function from normal segments (15–18,21). Three-dimensional strain analysis by magnetic resonance imaging tagging reveals the detailed myocardial fiber architecture and may offer a persuasive explanation for regional longitudinal deformation of the LV. It is reported that myocardial fiber angles with respect to the short-axis plane varied linearly from $-53 \pm 20^\circ$ at the epicardium to $87 \pm 25^\circ$ at the endocardium, so that subendocardial and epicardial myocardial fibers are oriented approximately at right angles to each other (41–43). The subendocardium is predominantly composed of longitudinal myocardial fiber. Shortening of the sarcomere does not exceed 15% and relates to regional longitudinal deformation. Although thickening of fiber is only 8%, thickening of the LV wall reaches 40%, because rearrangement of subendocardial myocardial fiber architecture, such as “cross-fiber shortening,” occurs in the radial orientation (41). Therefore, longitudinal subendocardial strain and SR may directly relate to the sarcomere shortening in the subendocardium. According to the concept in the helical heart model of Torrent-Guasp et al. (44), the descending segment, which constitutes the apical loop of the myocardial band, functions as endocardium. That study revealed that contraction of descending segment was synchronized with the phasic change of LV pressure and derived dP/dt. The relative merits of the longitudinal and cross-sectional approaches for SR assessment are still being debated. Our results are concordant with a recent tissue tagging study in normal subjects from the Johns Hopkins group, which shows that endocardial deformation is greatest in the longitudinal direction and verifies the endo-epicardial gradient in normal LVs on magnetic resonance imaging (11,43).

**Table 2.** Correlation Between Strain Rate and Tissue Velocity During Isovolumic Contraction (Peak +dP/dt) or Isovolumic Relaxation (Peak −dP/dt) With Invasive Measurements

<table>
<thead>
<tr>
<th></th>
<th>During IC</th>
<th></th>
<th>During IR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SR (s⁻¹)</td>
<td>p Value</td>
<td>TDI (cm/s)</td>
</tr>
<tr>
<td>Endocardium</td>
<td>-0.74</td>
<td>&lt; 0.0001</td>
<td>-0.13</td>
</tr>
<tr>
<td>Mid-myocardium</td>
<td>-0.67</td>
<td>0.0009</td>
<td>-0.07</td>
</tr>
<tr>
<td>Epicardium</td>
<td>-0.35</td>
<td>NS</td>
<td>0.09</td>
</tr>
</tbody>
</table>

dP/dt = first derivative of left ventricular pressure; IC = isovolumic contraction; IR = isovolumic relaxation; NS = nonsignificant; SR = strain rate; TDI = tissue Doppler imaging.
was generated between End and Epi sampling positions. In the present sampling position, at 7 cm depth, the difference in transducer angulation varied from 6.2° to 10.7° (mean 8.6°) between layers with a wall thickness of 10.4 ± 2.8 mm. As already reported, SR is more angle dependent than other Doppler modalities, and hence, derived error is enlarged, especially around the apex, because there is an intermediate Doppler angle of 45° around the apex (19). To minimize such error, we adjusted the lateral wall segment for sampling so that it was as parallel as possible to the echo beam.

To track the LV wall segment instantaneously, automatic sample volume tissue tracking techniques have been developed to aid continuous sampling of the moving walls. The application of these techniques for TDI and SR measurements in our study improved the quality of the traces.

**Conclusions.** Strain and SR represent a powerful and useful modality for evaluation of subendocardial function. Detection of peak SR was possible during IC and IR, and SR amplitude during these periods correlated with peak dP/dt derived from high-fidelity LV pressure recordings.

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