Heterogeneous Onset of Myocardial Relaxation in Subendocardial and Subepicardial Layers Assessed With Tissue Strain Imaging

Comparison of Normal and Hypertrophied Myocardium

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OBJECTIVES  We sought to investigate the existence of a time difference in myocardial relaxation between the subendocardium and subepicardium in patients with and without myocardial hypertrophy.

BACKGROUND  Regional differences in mechanical and electrical properties between the subendocardium and subepicardium have been described for the left ventricle in animals. However, this difference has not been well evaluated in clinical conditions.

METHODS  Time-to-peak radial strain with reference to the QRS complex was measured at the subendocardium and subepicardium at the mid-posterior wall of the left ventricle in 12 normal subjects, 14 patients with hypertensive heart disease, and 27 patients with aortic stenosis (16 with and 11 without strain electrocardiogram [ECG] pattern) using tissue Doppler-based strain imaging.

RESULTS  Time-to-peak radial strain in the subepicardium (381 ± 60 ms) was shorter than that in the subendocardium (463 ± 29 ms; p < 0.001) in normal subjects, suggesting that the subepicardial relaxation precedes subendocardial relaxation. No significant difference was found between normal subjects and patients with hypertensive heart disease (388 ± 67 ms for the subepicardium; 455 ± 36 ms for the subendocardium in hypertensive heart disease). In cases with hypertrophied myocardium due to aortic stenosis, time-to-peak radial strain in the subendocardium was shortened and that in the subepicardium was prolonged. In 10 (63%) of 16 patients with aortic stenosis and strain ECG pattern, the timing of peak strain in the subendocardium (417 ± 63 ms) preceded that in the subepicardium (452 ± 62 ms).

CONCLUSIONS  There is heterogeneous onset of myocardial relaxation in the subendocardial and subepicardial layers at the mid-posterior wall of the left ventricle. Subepicardial myocardial relaxation precedes subendocardial relaxation in normal subjects. In contrast, there is inversion of the transmural sequence of myocardial relaxation between the subendocardium and subepicardium in some patients with aortic stenosis and strain ECG pattern. (J Am Coll Cardiol Img 2009;2:701–8) © 2009 by the American College of Cardiology Foundation.
Transmural heterogeneity of the onset of myocardial contraction and relaxation has been observed in normal animal hearts using sonomicrometry and magnetic resonance imaging, and in an in vitro study using isolated myocytes (1–4). In the normal myocardium, the time-to-peak cell shortening is longer in the subendocardium than in the subepicardium. This time difference has also been described in an in vitro study using isolated myocytes with hypertrophied guinea pig hearts (3). In that study, the time difference to peak cell shortening between the subendocardium and subepicardium was less in hypertrophied myocardium than in normal myocardium. However, transmural mechanics has yet to be investigated in human hearts, and transmural mechanics in hypertrophied myocardium has not been described under clinical conditions. Investigation of transmural mechanics with a noninvasive method would provide important basic and clinical relevance concerning the pathophysiology of various myocardial diseases.

Tissue Doppler imaging enables us to measure change in regional myocardial length noninvasively over the complete cardiac cycle. With the recent development of myocardial strain imaging obtained by tissue Doppler imaging, we can estimate regional myocardial thickening over an entire cardiac cycle without being affected by cardiac translation and assess transmural distribution of myocardial strain (5). In the present study, we used myocardial strain imaging to determine whether the time difference to peak myocardial thickening between the subendocardium and subepicardium was observed in normal and hypertrophied human hearts.

**METHODS**

**Study patients.** The study subjects consisted of 27 patients with left ventricular (LV) hypertrophy caused by severe aortic stenosis, 14 patients with LV hypertrophy and a history of hypertension (Group HT), and 12 normal subjects (Group N). The subjects in Groups HT and N had no symptoms or echocardiographic findings suggestive of any cardiovascular disease other than hypertension. The patients with severe aortic stenosis were divided into 2 groups: 11 patients without strain T on electrocardiogram (ECG) (Group AS-NT) and 16 patients with strain T (Group AS-ST). Strain T was defined as asymmetric ST depression in any lead except aVR, and V1 to V3 in the absence of bundle-branch block. No significant coronary artery lesions were demonstrated by coronary angiography in any of patients with aortic stenosis. This study was conducted in accordance with institutional ethical guidelines, and all patients gave written, informed consent.

**Standard echocardiography.** Standard echocardiography and color tissue Doppler imaging were performed using a commercially available ultrasound scanner with a 2.5-MHz transducer (Aplio, Toshiba Medical Systems, Tokyo, Japan). The LV dimension, wall thickness, and transmitial inflow pattern were measured according to the guidelines of the American Society of Echocardiography (6). The LV ejection fraction was calculated using the Quinones formula (7). The LV mass was calculated using the Devereux formula (8). The severity of aortic stenosis was assessed by peak aortic valve pressure gradient and aortic valve area calculated using the continuity equation (9).

**Tissue strain imaging.** The LV short-axis image at the mid-level was obtained by color tissue Doppler imaging with a frame rate of 60 to 80 Hz and was analyzed offline using commercially available software (TDI-Q, Toshiba Medical Systems, Tokyo, Japan).

TDI-Q software enables accurate evaluation of regional myocardial strain using 2 novel techniques: the tissue Doppler tracking technique (5,10–12) and the angle-correction technique (5,12). Briefly, with the tissue Doppler tracking technique, motion of an arbitrary point on the myocardium is tracked during a cardiac cycle, based on the myocardial velocity information as follows (10). By integrating the velocity of an indexed point on the ventricular wall known from tissue Doppler imaging, we can obtain myocardial displacement and predict where the point will move next. By repeating this procedure, the system automatically tracks the motion of the point. The influence of myocardial translation can be neglected using this technique.

The angle-correction technique has been used to partly overcome the Doppler angle dependency that has been described in previous reports (5,12). To correct the Doppler incident angle, a contraction center is manually set at the center of the LV cavity in the LV short-axis view at end-systole. The myocardial velocity toward the contraction center (V motion) is automatically calculated by dividing the velocity toward the transducer (V beam) by the

**ABBREVIATIONS AND ACRONYMS**

AS-NT = aortic stenosis without strain T on electrocardiogram
AS-ST = aortic stenosis with strain T on electrocardiogram
AVC = aortic valve closure
ECG = electrocardiogram
HT = hypertensive heart disease
LV = left ventricular
N = normal subjects
TP-e = time-to-peak radial strain
cosine of the angle ($\theta$) between the Doppler beam and the direction to the contraction center:

$$V_{\text{motion}} = V_{\text{beam}}/\cos \theta$$

Using these 2 techniques, TDI-Q provides myocardial velocity, and displacement and strain as a result of velocity integration toward the contraction center without being affected by myocardial translation or the Doppler incident angle (5,10,11). In a previous experiment, the displacement data obtained by this method correlated well with true displacement ($r = 0.99$, $p < 0.0001$) (11).

Strain is defined as the change in distance between 2 myocardial points divided by the initial length ($L_0$). In clinical studies with echocardiography, myocardial strain is calculated using the following equation:

$$\left( L - L_0 \right)/L_0$$

where $L$ is the instantaneous length (13). In the present study, the distance of all 2-pixel pairs at end-diastole (equivalent to $L_0$) was set at 2 mm. Using the tissue tracking technique, we can obtain the displacement of the 2 points in the subendocardium or the subepicardium toward the contraction center. Therefore, we can obtain myocardial radial strain in the subendocardium and subepicardium separately in a cardiac cycle.

**Measurement of time-to-peak strain.** Figure 1 shows the temporal changes in myocardial radial strain in the subendocardium and subepicardium at the mid-LV posterior wall using TDI-Q. In the present study, subendocardium was defined as the inner one-third of the LV myocardium, and subepicardium was defined as the outer one-third of the LV myocardium. The myocardium contracts until myocardial radial strain reaches a peak, after which it relaxes.

We measured time-to-peak radial strain (TP-$e$) in the subendocardium and subepicardium at the mid-LV posterior wall with reference to the onset of QRS on ECG and calculated the difference of TP-$e$ between the layers. TP-$e$ was divided by the time to aortic valve closure (AVC) from the onset of QRS on ECG to correct for differences in heart rate (TP-$e$/AVC). Time to AVC was measured using pulsed-wave Doppler echocardiography of the LV outflow.

**Statistical analysis.** Data were expressed as mean ± SD. Comparison of the parameters of clinical characteristics and echocardiography among the groups

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**Figure 1. Temporal Myocardial Radial Strain Profile of the Subendocardium and Subepicardium in a Normal Subject and a Subject With Aortic Stenosis and Strain ECG Pattern**

The upper left panel shows a left ventricular short-axis image from a parasternal approach at end-diastole in a normal subject. The red dot is on the subendocardium, and the orange dot is on the subepicardium. The lower left panel shows M-mode imaging of the mid-left ventricular posterior region in the normal subject. The upper right panel shows temporal radial strain profile at the mid–left ventricular posterior region in the normal subject. The red curve in the lower panel shows the myocardial strain profile in the subendocardium; the orange curve shows that in the subepicardium. The lower right panel shows temporal radial strain profile in a subject with aortic stenosis and strain ECG pattern. The arrows indicate the peak myocardial strain in each layer. AVC — aortic valve closure; ECG — electrocardiogram; MVO — mitral valve closure.
was performed using one-way analysis of variance followed by Fisher multiple comparison tests. Categorical data among the groups were compared using the Pearson chi-square test. Unpaired Student *t* testing was used to compare the severity of aortic stenosis between patients with and without strain T and to compare the difference in TP-ε between the subendocardium and subepicardium. A `p` value of <0.05 was considered statistically significant. All statistical analyses were performed using Stat View 5.0 for Windows (SAS Institute, Cary, North Carolina).

TP-ε was measured by 2 independent observers and by 1 observer twice, a week apart, in 10 randomly selected segments to determine inter- and intraobserver variability. Variability was expressed as the absolute difference between the 2 measurements as a percentage of their mean values. Interobserver and intraobserver variability for TP-ε were 5.6 ± 6.4% and 4.8 ± 6.9%, respectively.

**RESULTS**

**Clinical and echocardiographic characteristics.** The characteristics of the patients are shown in Table 1.

There was no significant difference in age and gender among the patients and normal subjects.

Left ventricular contraction was preserved, and LV wall motion abnormality was not observed in any patients. The LV wall thickness and LV mass of patients in Group AS-NT were similar to those in Group HT. The LV wall was thicker in patients in Group AS-ST than those in Group AS-NT (`p` < 0.01). Doppler parameters of transmitral inflow suggested that diastolic function was similar among all groups, although patients in Group AS-ST had higher late diastolic inflow than those in the other groups (`p` < 0.05). In patients with aortic stenosis, aortic valve pressure gradient was significantly higher in patients in Group AS-ST than in those in Group AS-NT.

**Time-to-peak radial strain in the subendocardium and subepicardium.** Figure 2 shows the difference in TP-ε among the 4 groups. Although LV hypertrophy was observed in patients in Group HT, TP-ε in Group HT was similar to that of Group N in both the subendocardium and subepicardium. However, TP-ε in the subendocardium was shorter (`p` < 0.05) in patients with aortic stenosis than that in normal subjects, suggesting that subendocardial re-

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**Table 1. Patient Characteristics and Data of Standard Echocardiography**

<table>
<thead>
<tr>
<th>Variables</th>
<th>N (n = 12)</th>
<th>HT (n = 14)</th>
<th>AS-NT (n = 11)</th>
<th>AS-ST (n = 16)</th>
<th>P Value</th>
</tr>
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<tbody>
<tr>
<td>Patient characteristics</td>
<td></td>
<td></td>
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<tr>
<td>Age, yrs</td>
<td>61 ± 13</td>
<td>64 ± 12</td>
<td>63 ± 11</td>
<td>67 ± 13</td>
<td>0.620</td>
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<td>Male, n (%)</td>
<td>7 (58)</td>
<td>9 (64)</td>
<td>6 (55)</td>
<td>7 (44)</td>
<td>0.716</td>
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<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>127 ± 17</td>
<td>136 ± 13</td>
<td>124 ± 16</td>
<td>119 ± 16†</td>
<td>0.043</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>75 ± 9</td>
<td>79 ± 13</td>
<td>67 ± 6‡</td>
<td>66 ± 9†</td>
<td>0.002</td>
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<td>Heart rate, beats/min</td>
<td>63 ± 12</td>
<td>63 ± 13</td>
<td>64 ± 13</td>
<td>65 ± 10</td>
<td>0.949</td>
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<td>LV end-diastolic diameter, mm</td>
<td>48 ± 4</td>
<td>44 ± 4</td>
<td>46 ± 4</td>
<td>42 ± 5*</td>
<td>0.008</td>
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<tr>
<td>LV end-systolic diameter, mm</td>
<td>29 ± 5</td>
<td>27 ± 5</td>
<td>27 ± 2</td>
<td>26 ± 5‡</td>
<td>0.186</td>
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<tr>
<td>Ejection fraction, %</td>
<td>73 ± 7</td>
<td>72 ± 7</td>
<td>75 ± 6</td>
<td>73 ± 7</td>
<td>0.761</td>
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<tr>
<td>Thickness of IVS, mm</td>
<td>9 ± 1</td>
<td>12 ± 1*</td>
<td>13 ± 1*</td>
<td>14 ± 2*‡</td>
<td>&lt;0.001</td>
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<tr>
<td>Thickness of PW, mm</td>
<td>9 ± 1</td>
<td>12 ± 1*</td>
<td>12 ± 2*</td>
<td>14 ± 2*‡</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LV mass, g</td>
<td>145 ± 33</td>
<td>186 ± 35</td>
<td>216 ± 55*</td>
<td>227 ± 47*</td>
<td>&lt;0.001</td>
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<td>Mitral Doppler inflow</td>
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<tr>
<td>E-wave, cm/s</td>
<td>70 ± 17</td>
<td>67 ± 14</td>
<td>58 ± 15</td>
<td>76 ± 20</td>
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<tr>
<td>A-wave, cm/s</td>
<td>76 ± 22</td>
<td>81 ± 22</td>
<td>80 ± 13</td>
<td>103 ± 31*</td>
<td>0.011</td>
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<td>E/A</td>
<td>0.99 ± 0.40</td>
<td>0.88 ± 0.28</td>
<td>0.75 ± 0.29</td>
<td>0.78 ± 0.27</td>
<td>0.222</td>
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<td>Deceleration time of E-wave, ms</td>
<td>225 ± 23</td>
<td>263 ± 68</td>
<td>260 ± 72</td>
<td>258 ± 131</td>
<td>0.674</td>
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<td>The severity of aortic stenosis</td>
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<td>Peak pressure gradient, mm Hg</td>
<td>n/a</td>
<td>n/a</td>
<td>87 ± 36</td>
<td>120 ± 38</td>
<td>0.036</td>
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<tr>
<td>Aortic valve area, cm²</td>
<td>n/a</td>
<td>n/a</td>
<td>0.72 ± 0.32</td>
<td>0.54 ± 0.14</td>
<td>0.056</td>
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</tbody>
</table>

* `p` < 0.05 versus Group N; †`p` < 0.05 versus Group HT; ‡`p` < 0.05 versus Group AS-NT.

AS-NT = aortic stenosis without strain T ECG; AS-ST = aortic stenosis with strain T ECG; HT = hypertensive heart disease; IVS = intraventricular septal wall; LV = left ventricular; N = normal subjects; PW = posterior wall.
Myocardial Relaxation With Tissue Doppler

**Myocardial Relaxation With Tissue Doppler**

Relaxation at the mid-LV posterior wall occurs earlier in patients with aortic stenosis than in normal subjects. TP-e in the subepicardium was longer in Group AS-ST (452 ± 62 ms, p < 0.05) than in the other groups.

Figure 3 shows a comparison of TP-e/AVC among the 4 groups. For subjects in Group N and Group HT, TP-e/AVC in the subendocardium was greater than 1.0 (1.063 ± 0.050 and 1.079 ± 0.116, respectively), whereas that in the subepicardium was less than 1.0 (0.876 ± 0.140 and 0.913 ± 0.121, respectively). This indicates that myocardial relaxation in the subendocardium occurred before AVC, and that myocardial relaxation in the subepicardium occurred after AVC at the mid-LV posterior wall. Interestingly, this sequence was different in patients with aortic stenosis: subendocardial relaxation occurred before AVC in 18 of 27 patients with aortic stenosis. Subepicardial relaxation occurred after AVC in 9 of 16 patients with aortic stenosis and ECG strain pattern.

**Transmural sequence of myocardial relaxation.** We calculated the difference in TP-e between the subendocardium and subepicardium to investigate transmural discordance of myocardial relaxation at the mid-LV posterior wall (Fig. 4). In normal subjects, the timing of peak radial strain in the subepicardium (381 ± 60 ms) preceded that in the subendocardium (463 ± 29 ms, p < 0.001), indicating that myocardial relaxation at the mid-LV posterior wall occurred in the subepicardium before that in the subendocardium by about 80 ms.

In Group AS-ST, because TP-e in the subendocardium (452 ± 62 ms, p < 0.05) was shorter and that in the subepicardium (417 ± 63 ms, p < 0.05) was longer than those in Group N and Group HT, the difference in TP-e between the subendocardium and subepicardium (~35 ± 74 ms) was significantly shorter than that in Group N and Group HT (82 ± 45 ms and 67 ± 62 ms, respectively). In 10 (63%) of 16 patients with severe aortic stenosis and ECG strain T-wave, the difference was negative, indicating inversion of the transmural sequence of myocardial relaxation.

**DISCUSSION**

We demonstrated myocardial radial strain profile and transmural mechanics at the mid-LV posterior wall in humans using echocardiography for the first time. Our results indicate that in normal subjects the onset of myocardial relaxation in the subepicardium precedes that in the subendocardium at the mid-LV posterior wall, and that the transmural sequence is inverted in some patients with severely hypertrophied hearts.

**Figure 2. TP-e in the Subendocardium and Subepicardium**

Time-to-peak radial strain in the subendocardium (A) and subepicardium (B). *p < 0.05 versus Group N (normal subjects); †p < 0.05 versus Group N and Group HT (patients with hypertensive heart disease); ‡p < 0.05 versus the other groups. AS-NT = aortic stenosis without strain T on electrocardiogram; AS-ST = aortic stenosis with strain T on electrocardiogram; TP-e = time-to-peak strain from the onset of QRS on electrocardiogram; other abbreviations as in Figure 1.

**Figure 3. Ratio of TP-e to Time to AVC**

Time-to-peak radial strain /AVC in the subendocardium (A) and subepicardium (B). If the TP-e/AVC ratio is greater than 1.0, the timing of strain peak is located beyond that of AVC. *p < 0.05 versus Group N and Group HT; †p < 0.05 versus other groups. TP-e/AVC = ratio of TP-e to time to AVC from the onset of QRS on ECG; other abbreviations as in Figures 1 and 2.
Transmural mechanics assessed by echocardiography in normal human hearts. We demonstrated that the onset of subepicardial relaxation precedes subendocardial relaxation at the mid-LV posterior wall in normal human hearts. The transmural heterogeneity of myocardial strain value in human hearts has been described previously using magnetic resonance imaging (14). However, the transmural sequence of myocardial relaxation has not been previously investigated in human hearts. The transmural sequence has been studied in normal animal hearts using sonomicrometry and in isolated myocytes (1–3); these results are consistent with our data of normal human hearts assessed by echocardiography.

We also found that subendocardial contraction at the mid-LV posterior wall sustained after AVC. During isovolumic relaxation, apical untwisting is accompanied by relaxation and expansion of LV apex (1). This generates a rapid base-to-apex reversal of isovolumic intracavitary pressure and blood flow (15,16). Because volume cannot change within isovolumic relaxation, subendocardial wall thickening at the basal to middle region might sustain after AVC with the apical expansion.

Transmural mechanics assessed by echocardiography in hypertrophied hearts. We also demonstrated differences in the transmural sequence of myocardial relaxation between normal and severely hypertrophied left ventricles at the mid-LV posterior region. There was no significant difference in the transmural sequence of myocardial relaxation between Group N and Group HT, indicating that wall thickness itself may not be a major determinant of transmural difference in relaxation timing. However, the onset of subendocardial relaxation in Group AS-NT was earlier than that in Group HT. Although previous studies indicate that high LV pressure appears to affect myocardial strain value and LV segmental dyssynchrony (17,18), its effect on time-to-peak strain in the subendocardium has not been studied. The high LV pressure in end-systole may depress thickening of the subendocardium in patients with aortic stenosis resulting in a shortened duration of thickening.

The onset of subepicardial relaxation was delayed in Group AS-ST, compared with patients in Group AS-NT, and the transmural sequence of relaxation was inverted in some patients. It is known that transmural electrical heterogeneity exists in the myocardium and affects the ECG waveform (19). There are 3 types of gradient that are responsible for genesis of T-wave: 1) differences between the right and left ventricle; 2) differences between apex-base and anterior-posterior walls; and 3) transmural difference (20). In previous investigations, the presence of a repolarization gradient between the “mid-myocardial region” (M-cells) and subendocardial and subepicardial regions has been correlated with the genesis of upright T waves in ventricular wedge preparations (21). However, studies of intact hearts have failed to provide evidence for transmural differences in repolarization (22). Thus, the genesis of T-wave on surface ECG may be largely due to the base-to-apex gradient with minimum contribution from the transmural gradient (1). The presence of strain T-wave may be accompanied with significant change of the electrical property in the myocardial cells in the whole left ventricle, which generates not only base-to-apex gradient but also the transmural gradient to some extent in the hypertrophied hearts. Actually, a previous in vitro study (3) found that the action potential duration in the subepicardium and midcardium becomes longer as LV hypertrophy progresses, and that the time-to-peak cell shortening also becomes longer. In contrast, the action potential duration in the subendocardium becomes shorter and the time-to-peak cell shortening becomes shorter, although the degree is an insignificant amount. In the present study, we found that the onset of myocardial relaxation in the subendocardium and subepicardium at the mid-LV posterior wall was almost coincident, and that in some patients the onset of subendocardial relaxation preceded that of subepicardial relaxation. These findings are consistent with those of the earlier animal study (3).
**Clinical implications.** The present study is the first report regarding the transmural mechanics in normal and hypertrophied myocardium under clinical conditions. There are a lot of conditions with T-wave alteration such as myocardial hypertrophy, ischemia, and cardiac arrhythmias including the Brugada syndrome and long QT syndrome. We believe that our present method should be useful to investigate transmural electromechanical sequence of relaxation, and also contraction, and could provide new insights into the pathophysiology of these conditions.

We demonstrated the difference in the onset of myocardial relaxation between the subendocardium and subepicardium using echocardiography, without opening the chest and implanting crystals, as has been performed in some reports that focused on transmural mechanics using sonomicrometry with open-chest animals (1,2). The invasive method cannot be applied to humans. Moreover, the implantation of the crystals may cause local scarring and induce abnormal wall motion not only due to the implantation procedure, but also due to their weight and inertial properties. The thoracotomy itself can alter the temperature of the epicardial surface and change its action potential duration and T-wave polarity (23). Opening the pericardium could affect the whole cardiac motion and regional myocardial deformation. In contrast, the recent development of echocardiography has enabled the investigation of the transmural myocardial deformation in clinical settings (5,10–12).

We assumed that earlier myocardial relaxation of the subepicardium might facilitate the following relaxation of the subendocardium so that the early LV filling occurs efficiently. However, unfortunately, in this study, we could not find a significant association of the difference in TP-e between the subendocardium and subepicardium with transmural E-wave velocity ($r = 0.011$, $p = 0.940$). Further study will be needed using more sophisticated indexes of LV filling or relaxation.

**Study limitations.** In the present study, we assessed myocardial strain only at the mid-posterior wall; therefore, our results may not apply to the other LV segments. Our findings may not relate to aortic stenosis, but may reflect the consequences of LV pressure load. We did not measure actual LV pressure when we obtained the echocardiographic data. The LV pressure and wall stress affect the myocardial strain profile. The difference in the onset of myocardial relaxation among the 4 groups may be caused by differences in the hemodynamic condition rather than differences in electrical properties, especially in the subendocardium. Despite these limitations, we consider that our method is a successful noninvasive technique for demonstrating the transmural sequence of myocardial relaxation.

We set the initial length to obtain instantaneous radial strain at 2 mm. A longer length would be advantageous for obtaining peak radial strain but a smaller length would be better for obtaining regional information. In the present study, we measured time-to-peak strain, which might be less affected by noise than peak strain even with a smaller initial length.

**CONCLUSIONS**

Myocardial relaxation of the subepicardium precedes that of the subendocardium at the mid-LV posterior wall in the normal human myocardium. In the hypertrophied myocardium with aortic stenosis, the onset of myocardial relaxation in the subepicardium is earlier than that in normal myocardium at the mid-LV posterior wall, whereas the onset in the subepicardium is later than that in the normal myocardium. In some patients with severe hypertrophy and strain T-wave, there is an inversion of the transmural sequence of the myocardial relaxation, compared with that in normal subjects.

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Key Words: echocardiography ● hypertrophy ● electrocardiography.