Difference in Systolic Motion Velocity of the Left Ventricular Posterior Wall in Patients with Asymmetric Septal Hypertrophy and Prior Anteroseptal Myocardial Infarction

Evaluation by Pulsed Tissue Doppler Imaging

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SUMMARY

The left ventricular (LV) posterior wall in patients with asymmetric septal hypertrophy or prior anteroseptal myocardial infarction (A-MI) frequently demonstrates normal or supernormal motion to compensate for hypokinesis of the interventricular septum. This study evaluated the systolic motion velocity of the posterior wall in these conditions using a pulsed tissue Doppler imaging system. The study population consisted of 30 patients with hypertrophic cardiomyopathy (HC) and asymmetric septal hypertrophy, 25 with prior A-MI and 30 normal controls. The systolic excursion of the posterior wall was obtained by M-mode echocardiography. The endocardial motion velocities of the posterior wall were obtained by pulsed tissue Doppler imaging. The systolic excursion of the posterior wall was significantly greater in the A-MI and HC groups than in the control group, and was significantly greater in the A-MI group than in the HC group. The peak systolic velocity of the posterior wall was significantly lower in the HC group than in the control and A-MI groups, and the time from the electrocardiographic Q wave to the peak of the systolic wave of the posterior wall was significantly longer in the HC group than in the other groups. There were rough negative and positive correlations between the LV end-diastolic pressure and the peak systolic velocity and time from the Q wave to the peak of the systolic wave, respectively. In conclusion, LV myocardial contractility in HC patients was impaired when compared to A-MI patients despite similar posterior wall motion on the M-mode echocardiogram. Pulsed tissue Doppler imaging method may provide new insights and allow
further evaluation of myocardial dysfunction. (Jpn Heart J 1998; 39: 163-172)

Key words: Tissue Doppler imaging, Myocardial systolic function, Hypertrophic cardiomyopathy, Anteroseptal myocardial infarction

The interventricular septum is characteristically hypertrophic and frequently hypo- or akinetic in patients with hypertrophic cardiomyopathy (HC) and asymmetric septal hypertrophy. Additionally, after anteroseptal myocardial infarction caused by complete obstruction of the left anterior descending coronary artery, there is also hypo- or dysskinetic motion of the left ventricular anterior wall and interventricular septum. M-mode echocardiography of the posterior wall in both conditions frequently demonstrates normal to hyperkinetic motion, and preserved left ventricular pump function. However, there have been diseases reported that have impaired myocardial shortening despite normal left ventricular ejection fraction, and much discussion has been generated about cardiac function in various diseases.

Recently, tissue Doppler imaging has allowed high amplitude of the Doppler signal from the ventricular wall to be displayed selectively after removing low amplitude of the Doppler signal from blood flow. This technique is expected to contribute substantially to clinical evaluation of myocardial function in patients with various heart diseases. In this study, pulsed tissue Doppler imaging methods were used to examine myocardial systolic function of the left ventricular posterior wall in patients with HC and asymmetric septal hypertrophy or prior anteroseptal myocardial infarction. We also discuss the clinical significance and usefulness of this technique.

METHODS

Patient population: The study population consisted of 30 untreated patients with HC and asymmetric septal hypertrophy, and 25 with prior anteroseptal myocardial infarction. The latter patients have received either isosorbide dinitrate or enalapril 5 to 10 mg twice daily, but no digitalis. The subjects with HC had no significant coronary stenosis by coronary angiography, and lacked any cardiac or systemic disease capable of producing left ventricular hypertrophy. All HC patients demonstrated hypertrophy of only the interventricular septum at end-diastole (15 mm) and normal posterior wall thickness at end-diastole (9 mm) by two-dimensional echocardiography. Left ventricular outflow obstruction was observed in 4 of the 30 HC patients. The diagnosis of 25 patients with prior anteroseptal myocardial infarction was confirmed by: 1) a history of previous myocardial infarction with complete obstruction at segment 6 or 7 of the left anterior descending coronary artery by emergent coronary angiography; 2) a
normal creatine kinase concentration; and 3) regional asynergy extending only from the left ventricular anterior wall to the interventricular septum. Six of the 25 patients had an aneurysm from the left ventricular apex to the interventricular septum. The control group consisted of 30 patients in whom cardiovascular abnormalities were suspected due to chest pain, dyspnea, arrhythmias, or heart murmurs, but in whom no abnormalities were noted on routine echocardiography and/or cardiac catheterization. The purpose of the examinations was explained and informed consent was obtained from each subject.

Study protocol: All 55 patients with HC and myocardial infarction underwent cardiac catheterization just after M-mode echocardiography and tissue Doppler imaging using a commercially available Toshiba SSA-380A (Toshiba Corp., Tokyo, Japan; 3.75 MHz probe). Left ventricular end-diastolic pressure was measured with a 6F fluid-filled pigtail Cordis catheter prior to coronary angiography and left ventriculography. The left ventricular ejection fraction was determined by the area-length method after left ventriculography.15)

M-mode echocardiography: Two-dimensional parasternal long-axis echocardiograms of the left ventricle were recorded at the level of the chordae tendineae. Left ventricular end-diastolic and end-systolic dimensions (LVDd and LVDs, respectively), and end-diastolic thickness of the interventricular septum and left ventricular posterior wall (IVSth and PWth, respectively) were measured. Percent left ventricular fractional shortening and left ventricular mass index corrected for body surface area (BSA) were calculated using the following equations:

1) % left ventricular fractional shortening (%)

\[ = \left( \frac{LVDd - LVDs}{LVDd} \right) \times 100 \]

2) left ventricular mass index (g/m²)

\[ = \left\{ \frac{1.04 \times \left[ \left( LVDd + IVSth + PWth \right)^3 - LVDd^3 \right] - 13.6}{BSA} \right\} \]

The maximal systolic excursion of the posterior wall was also measured.

Tissue Doppler imaging: Two-dimensional parasternal long-axis echocardiograms of the left ventricle were recorded and then sample volumes were set at the endocardial portions of the middle sites of the interventricular septum and posterior wall (Figure 1, left). The wall motion velocity at each point was recorded by a pulsed Doppler method, and the peak systolic velocities of both walls and the time from the electrocardiographic Q wave to the peak of the systolic wave were calculated (Figure 1, right). No patients had abnormal QRS prolongation.

Statistical analysis: Results are expressed as the mean ± standard deviation (SD). Differences in mean group values were assessed by the analysis of variance (ANOVA) and Scheffe’s F test. Linear regression coefficients were calculated to
Figure 1. Measurement method for left ventricular wall motion velocity using pulsed tissue Doppler imaging. Two sample volumes (white circles) were set at the endocardial portions of the middle sites of the interventricular septum (IVS) and posterior wall (PW) in the parasternal two-dimensional long-axis echocardiogram (left). The right panel demonstrates the wall motion velocity pattern of the IVS (top) and PW (bottom) in a normal subject. RV = right ventricle; LV = left ventricle; LA = left atrium; Ao = ascending aorta; Sw = peak systolic velocity; Q-Sw = time from the electrocardiographic Q wave to the peak of the systolic wave; Ew = early diastolic wave; Aw = atrial systolic wave; ECG = electrocardiogram; PCG = phonocardiogram.

determine the correlation among variables. A $p$ value $<0.05$ was considered significant.

**Results**

The clinical characteristics of study patients and controls are outlined in Table I. There were no significant differences in age or heart rate between the study groups. The systolic blood pressure was significantly lower in the HC group than in the other two groups (both $p<0.0001$).

**Cardiac catheterization:** The left ventricular ejection fraction was significantly lower in the anteroseptal myocardial infarction group than in the control and HC groups (both $p<0.0001$), but there was no significant difference between the HC and control groups (Table II). The left ventricular end-diastolic pressure was significantly higher in the HC group than in the anteroseptal myocardial infarction and control groups ($p<0.01$ and $p<0.0001$, respectively), and was also significantly higher in the anteroseptal myocardial infarction group than in the control group ($p<0.01$).
**Table I.** Clinical Data

<table>
<thead>
<tr>
<th></th>
<th>Number</th>
<th>Age (years)</th>
<th>Heart rate (beats/min)</th>
<th>SBP (mm Hg)</th>
<th>DBP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control group</td>
<td>30</td>
<td>59 ± 9</td>
<td>72 ± 8</td>
<td>143 ± 13</td>
<td>81 ± 10</td>
</tr>
<tr>
<td>Patient group</td>
<td>30</td>
<td>61 ± 12</td>
<td>70 ± 9</td>
<td>122 ± 15*</td>
<td>77 ± 9</td>
</tr>
<tr>
<td>HC</td>
<td>25</td>
<td>62 ± 7</td>
<td>76 ± 8</td>
<td>140 ± 12*</td>
<td>80 ± 7</td>
</tr>
</tbody>
</table>

*p < 0.0001 vs Control group, *p < 0.0001 vs HC group. HC = hypertrophic cardiomyopathy; MI = myocardial infarction; SBP and DBP = systolic and diastolic blood pressure, respectively.

**Table II.** M-mode Echocardiographic, Pulsed Tissue Doppler Imaging and Cardiac Catheterization Variables

<table>
<thead>
<tr>
<th></th>
<th>% FS (%)</th>
<th>LVMI (g/m²)</th>
<th>PVE (mm)</th>
<th>IVS</th>
<th>PW</th>
<th>EF (%)</th>
<th>LVEDP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control group</td>
<td>41 ± 7</td>
<td>110 ± 24</td>
<td>13.7 ± 0.2</td>
<td>6.6 ± 2.2</td>
<td>190 ± 34</td>
<td>7.8 ± 0.9</td>
<td>191 ± 21</td>
</tr>
<tr>
<td>Patient group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HC</td>
<td>39 ± 10</td>
<td>206 ± 52**</td>
<td>14.5 ± 0.3**</td>
<td>5.7 ± 2.5</td>
<td>210 ± 25*</td>
<td>6.1 ± 0.9**</td>
<td>232 ± 47**</td>
</tr>
<tr>
<td>anterior MI</td>
<td>28 ± 11**</td>
<td>116 ± 43***</td>
<td>16.1 ± 0.2***</td>
<td>5.4 ± 3.0</td>
<td>214 ± 20*</td>
<td>8.2 ± 0.7***</td>
<td>181 ± 26***</td>
</tr>
</tbody>
</table>

*p < 0.01, **p < 0.0001 vs Control group, *p < 0.01, ***p < 0.001, ****p < 0.0001 vs HC group. HC = hypertrophic cardiomyopathy; MI = myocardial infarction; FS = fractional shortening of the left ventricle; LVMI = left ventricular mass index; PVE = systolic excursion of left ventricular posterior wall by M-mode echocardiography; IVS = interventricular septum; PW = left ventricular posterior wall; Sw = peak systolic velocity of the left ventricular wall; Q-Sw = time from the electrocardiographic Q wave to the peak of the systolic wave of the left ventricular wall; EF = left ventricular ejection fraction by left ventriculography; LVEDP = left ventricular end-diastolic pressure.

**M-mode echocardiography:** The percent fractional shortening of the left ventricle in the anteroseptal myocardial infarction group was significantly lower than the HC and control groups (p < 0.001 and p < 0.0001, respectively), but there was no significant difference between the HC and control groups (Table II). The left ventricular mass index was significantly higher in the HC group than in the anteroseptal myocardial infarction and control groups (both p < 0.0001), but there was no significant difference between the latter two groups. The systolic excursion of the posterior wall was significantly greater in the anteroseptal myocardial infarction group than in the control and HC groups (both p < 0.0001), and the wall excursion in the HC group was significantly greater than the control group (p < 0.0001).

**Tissue Doppler imaging:** No significant difference in the peak systolic velocity of the interventricular septum was found among the three groups (Table II).
Figure 2. Correlations between the left ventricular end-diastolic pressure (LVEDP) and the peak systolic velocity (Sw) of the posterior wall (left), and the time from the electrocardiographic Q wave to the peak systolic velocity of the posterior wall (right) in patients with hypertrophic cardiomyopathy and asymmetric septal hypertrophy.

However, the time from the electrocardiographic Q wave to the peak of the systolic wave of the interventricular septum was significantly longer in the HC and anteroseptal myocardial infarction groups than in the control group (both \( p < 0.01 \)). The time from the electrocardiographic Q wave to the peak of the systolic wave of the interventricular septum was significantly shorter and longer, respectively, than that of the posterior wall in the HC and anteroseptal myocardial infarction groups (both \( p < 0.001 \)). On the other hand, the peak systolic velocity and the time from the electrocardiographic Q wave to the peak of the systolic wave of the posterior wall in the HC group were significantly lower and longer, respectively, than those of the anteroseptal myocardial infarction and control groups (all \( p < 0.0001 \)). These variables did not differ between the anteroseptal myocardial infarction and control groups. In the HC group, the peak systolic velocity and time from the electrocardiographic Q wave to the peak of the systolic wave of the posterior wall did not correlate with the percent fractional shortening of the left ventricle, left ventricular ejection fraction, systolic excursion of the posterior wall, and left ventricular mass index. The peak systolic velocity of the posterior wall showed a rough negative correlation with the left ventricular end-diastolic pressure \( (r = -0.38, p < 0.04) \). Additionally, a rough positive correlation was found between the time from the electrocardiographic Q wave to the peak of the systolic wave of the posterior wall and the left ventricular end-diastolic pressure \( (r = 0.36, p < 0.05) \) (Figure 2).

**DISCUSSION**

Hypertrophic cardiomyopathy (HC) with asymmetric septal hypertrophy is pathologically characterized by myocardial disarray and fibrosis.\(^{1,2}\) The interven-
tricular septum frequently becomes hypo- to akinetic with increasing hypertrophy. However, the posterior wall may demonstrate normal to hyperkinetic motion to compensate for the abnormal interventricular septal motion. Thus, the left ventricular ejection fraction has been reported to be normal or even supernormal. On the other hand, in anteroseptal myocardial infarction caused by complete obstruction at segment 6 or 7 of the left anterior descending coronary artery, the left ventricular anterior wall to interventricular septum which is under the control of left anterior descending coronary artery shows hypo- to dyskinetic motion. However, posterior wall shows normal to hyperkinetic motion when left circumflex or right coronary artery has a normal anatomy. Although the posterior wall in both of these conditions may have similar motion on the M-mode echocardiogram, there have been no detailed studies examining myocardial systolic function.

Several studies have found the distribution of myocardial abnormalities in patients with HC. Maron et al. have reported that myocardial damages in patients with HC and asymmetric septal hypertrophy were limited to the interventricular septum in the obstructive type, but were also found in the posterior wall in the nonobstructive type. Unverferth et al. and Tanaka et al. have emphasized that myocardial damages are present throughout the left ventricle in both the asymmetric and symmetric types of HC, and that damage density is higher adjacent to the left ventricular cavity. Takagi et al. have performed exercise thallium-201 scintigraphy in patients with HC and asymmetric septal hypertrophy and found decreased washout in all walls.

In general, myocardial circumferential shortening in the left ventricle is considered to be most prominent along the midwall among the three myocardial fiber layers. Aurigemma et al. have performed M-mode left ventricular echocardiography in hypertensive patients with moderate left ventricular hypertrophy and normal left ventricular ejection fraction, and found increased circumferential shortening of the subendocardial wall compared to the midwall. Palmon et al. and Kramer et al. have examined hypertensive patients with left ventricular hypertrophy using cine magnetic resonance imaging, and observed that both long- and short-axis myocardial shortening were decreased. O'Gara et al. have examined myocardial perfusion by thallium-201 emission computed tomography in patients with HC and found a good correlation between abnormalities of left ventricular systolic performance and reversibility of perfusion defects after exercise.

A tissue Doppler imaging technique has recently been used to measure the motion velocity of cardiac muscles allowing the ventricular wall to be quantitatively evaluated in patients with various heart diseases. Since the motion velocity of the ventricular wall is generally much less than the blood flow velocity in
the cardiac cavity throughout the cardiac cycle, its frequency shift is also low. The amplitude of tissue Doppler signals is about 40 dB greater than that of blood flow, and wall motion velocity alone can be determined by bypassing the high-pass filter which cuts low Doppler shifts.\(^{10}\)

In this study, we used pulsed tissue Doppler imaging to measure the motion velocity at the endocardial portion of the left ventricular wall, which was significantly higher than at the epicardial portion.\(^{13}\) In the patients with HC and asymmetric septal hypertrophy the peak systolic velocity and the time from the electrocardiographic Q wave to the peak of the systolic wave of the posterior wall were significantly lower and longer, respectively, than in the anteroseptal myocardial infarction and control groups. These results suggest that myocardial systolic function of the posterior wall remained normal in the anteroseptal myocardial infarction group but was impaired in the HC group. These results are supported by previous pathologic and myocardial scintigraphic studies.\(^{1,2,15,22}\) On the other hand, no significant correlation was found between the tissue Doppler imaging variables and the left ventricular mass index. The rough correlation between the variables and the left ventricular end-diastolic pressure agrees with reports suggesting that left ventricular diastolic dysfunction in HC is dependent upon myocardial abnormalities such as disarray and fibrosis rather than the magnitude of left ventricular hypertrophy.\(^{23,24}\)

It has been reported that regional wall motion asynchrony occurs in patients with unproportional hypertrophy and regional asynergy of the left ventricular wall.\(^{25-27}\) Schwammenthal et al.\(^{27}\) have examined regional wall motion abnormalities in HC by cine magnetic resonance imaging, and found the time from end-diastole to maximal wall thickening to be shorter in the hypertrophied basal region than in the nonhypertrophied apical region. In the present study, the time from the electrocardiographic Q wave to the peak of the systolic wave of the interventricular septum was significantly longer in the HC and anteroseptal myocardial infarction groups than in the control group. Additionally, the time to peak velocity of the interventricular septum was significantly shorter and longer, respectively, than that of the posterior wall in the HC and anteroseptal myocardial infarction groups. These findings suggest regional systolic asynchrony of the left ventricular wall in patients with HC and anteroseptal myocardial infarction.

**Limitations:** The influence of motion of the entire heart cannot be excluded because we only measured motion velocity at the endocardial portion without calculating the transmyocardial velocity gradient from the endocardial and epicardial velocities.\(^{11,13}\) However, the endocardial motion velocity is significantly greater than the epicardial motion velocity. Wong et al.\(^{28}\) have reported that the endocardium and subendocardium are the most stressed areas in the left ventricular wall. Our results demonstrating rough correlations between the tissue
Doppler imaging variables and left ventricular end-diastolic pressure support their view. Since the object of this study was a comparison between control and patient groups, the limitation to the motion velocity at the endocardial portion alone is not clinically important. Both short- and long-axis views are required for the complete evaluation of myocardial function of the left ventricular wall. In this study, however, only myocardial systolic function in the short-axis direction was examined. This should not affect the clinical significance of the results, because short-axis motions are more important for left ventricular contraction.\(^{29}\)

**Conclusions:** Systolic motion velocity of the left ventricular posterior wall was decreased despite a lack of the wall hypertrophy in patients with HC and asymmetric septal hypertrophy. Systolic asynchrony of the left ventricular wall also was found in patients with HC and asymmetric septal hypertrophy and anteroseptal myocardial infarction. Pulsed tissue Doppler imaging provides useful information for the detailed evaluation of myocardial systolic function at each portion of the left ventricular wall in these patients.

**References**

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