Assessment of Mitral Annulus Velocity by Doppler Tissue Imaging in the Evaluation of Left Ventricular Diastolic Function

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Objectives. This study assessed the clinical utility of mitral annulus velocity in the evaluation of left ventricular diastolic function.

Background. Mitral inflow velocity recorded by Doppler echocardiography has been widely used to evaluate left ventricular diastolic function but is affected by other factors. The mitral annulus velocity profile during diastole may provide additional information about left ventricular diastolic function.

Methods. Mitral annulus velocity during diastole was measured by Doppler tissue imaging (DTI) in 59 normal volunteers (group 1); 2) in 20 patients with a relaxation abnormality as assessed by Doppler mitral inflow variables (group 2) at baseline and after saline loading; 3) in 11 patients (group 3) with normal mitral inflow (E) to late mitral inflow (A), in 38 consecutive patients (group 4) undergoing cardiac catheterization in whom mitral inflow velocity and tau as well as mitral annulus velocity were measured simultaneously.

Results. In group 1, mean ± SD peak early and late diastolic mitral annulus velocity was 10.0 ± 1.3 and 9.5 ± 1.5 cm/s, respectively. In group 2, mitral inflow velocity profile changed toward the pseudonormalization pattern with saline loading (deceleration time 311 ± 84 ms before to 216 ± 40 ms after intervention, p < 0.001), whereas peak early diastolic mitral annulus velocity did not change significantly (5.3 ± 1.2 cm/s to 5.7 ± 1.4 cm/s, p = NS). In group 3, despite a significant change in mitral inflow velocity profile after nitroglycerin, peak early diastolic mitral annulus velocity did not change significantly (9.5 ± 2.2 cm/s to 9.2 ± 1.7 cm/s, p = NS). In group 4, peak early diastolic mitral annulus velocity (r = −0.56, p < 0.01) and the early/late ratio (r = −0.46, p < 0.01) correlated with tau. When the combination of normal mitral inflow variables with prolonged tau (>50 ms) was classified as pseudonormalization, peak early diastolic mitral annulus velocity <8.5 cm/s and the early/late ratio <1 could identify the pseudonormalization with a sensitivity of 88% and specificity of 67%.

Conclusions. Mitral annulus velocity determined by DTI is a relatively preload-independent variable in evaluating diastolic function.

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Left ventricular diastolic dysfunction has been recognized as an important primary cause of heart failure (1,2). Doppler examination of mitral inflow has been most widely used to evaluate left ventricular diastolic function. However, because the mitral inflow velocity profile is affected by several factors including volume status, left atrial pressure and rate of myocardial relaxation (3–5) it is desirable to have additional variables to complement mitral inflow velocity in evaluating diastolic function. The mitral annulus velocity profile during diastole reflects the rate of changes in the long-axis dimension and in left ventricular volume. It has been shown (6,7) that the ratio of mitral annulus motion during atrial systole to the total diastolic annular motion is increased when relaxation is abnormal. Characteristic mitral annulus motion was also reported in restrictive cardiomyopathy. Therefore, in this study we evaluated the clinical value of using Doppler tissue imaging (DTI) to measure mitral annulus velocity during diastole in the assessment of diastolic function.

Methods

Study subjects. We studied four groups of subjects. Group 1 included 59 normal volunteers (30 men, 29 women; mean age 45 ± 14 years) with no cardiovascular symptoms and normal findings on two-dimensional echocardiography. Group 2 comprised 20 patients (7 men, 13 women; mean age 64 ± 6 years) with a relaxation abnormality as assessed by mitral inflow variables (deceleration time >240 ms and ratio of early mitral inflow (E) to late mitral inflow (A) <1). Patients with valvular disease, a regional wall motion abnormality or a history of myocardial infarction were excluded. Group 3 consisted of 11 patients (7 men, 4 women; mean age 35 ± 8 years) with normal left ventricular systolic and diastolic function.
Echocardiography was initially performed for nonspecific electrocardiographic abnormalities or atypical chest pain. Group 4 consisted of 38 consecutive patients (26 men, 12 women; mean age 54 ± 10 years) undergoing diagnostic coronary angiography or electrophysiologic study. Coronary angiography showed coronary artery disease in 28 patients. Patients with myocardial infarction or significant valvular disease were excluded.

Echocardiography. Echocardiograms were obtained by using an Acuson XP/10 with a 2.5-MHz transducer. Sample volume (size 2 mm) of the pulsed wave Doppler was placed between the tips of the mitral leaflets in the apical four-chamber view. Early (E) and late (A) transmitial flow velocities, the ratio of early to late peak velocities (E/A) and deceleration time of E velocity were obtained.

Pulsed wave DTI was performed by activating DTI function in the same machine. Sample volume was located at the septal side of the mitral annulus. Early (E') and late (A') diastolic mitral annulus velocities and the ratio of early to late peak velocities (E'/A') were obtained.

Doppler echocardiograms were recorded on a strip chart recorder with a sweep speed of 100 mm/s; the mean values of three different cardiac cycles were obtained.

Saline and nitroglycerin infusion protocol. In group 2, 500 to 700 ml of normal saline solution was rapidly infused. In group 3, nitroglycerin was infused at an initial rate of 30 μg/min that was increased by 10 μg/min every 2 min until the heart rate was 10 beats/min above the heart rate at rest. The mean rate of nitroglycerin infusion was 99 ± 48 μg/min (range 50 to 180).

Measurement of tau. Left ventricular pressure was measured before angiography. An 8F pig-tail catheter was placed in the left ventricle and a 3F Millar transducer (Millar Instruments) was introduced into it. Care was taken to avoid premature ventricular contraction. Pressure wave forms were recorded on a Sony digital audio tape recorder with a sampling rate of 600 Hz for later analysis and simultaneously into the echocardiograph while obtaining Doppler signals. Tau was calculated according to the method proposed by Weiss et al. (8). The pressure decrease from the peak negative first derivative of left ventricular pressure (dP/dt) to 5 mm Hg above minimal left ventricular diastolic pressure was applied to the exponential decay:

\[ P = \exp(At + B), \]

where \( P \) is pressure, \( A \) is the slope of ln P versus t, t is the time after the peak negative dP/dt, and tau is represented by \(-1/A\).

Statistics. Results are expressed as mean value ± SD. Paired t tests were used to assess differences before and after alteration of loading condition. Statistical relations were assessed by linear regression analysis. A p value <0.05 was considered statistically significant.

Results

Normal values. Normal values for mitral annulus velocity were obtained from 59 normal volunteers between the ages of 20 and 69 years (group 1). Mean peak E' velocity and peak A' velocity were 10.0 ± 1.3 cm/s and 9.5 ± 1.5 cm/s, respectively, with an E'/A' ratio of 1.1 ± 0.2. As with peak E and A velocity of mitral inflow, peak E' velocity decreased and peak A' velocity increased with age (Table 1). The mean E/A ratio reversed (i.e., was <1) in patients aged 60 to 69 years, whereas the E'/A' ratio reversed earlier in patients aged 40 to 49 years.

Load dependence. In group 2, mitral inflow velocity profile changed toward the pseudonormalization pattern with saline loading (deceleration time 311 ± 84 ms before intervention to 216 ± 40 ms after intervention, \( p < 0.001; \) E/A ratio 0.7 ± 0.1 to 0.9 ± 0.1, \( p < 0.001 \)). In contrast, peak E' velocity and E'/A' ratio did not change significantly (peak E' velocity 5.3 ± 1.2 cm/s to 5.7 ± 1.4 cm/s, \( p = \text{NS}; \) E'/A' ratio 0.5 ± 0.1 to 0.5 ± 0.1, \( p = \text{NS} \)) (Fig. 1). In group 3, deceleration time increased significantly with nitroglycerin infusion (165 ± 16 ms to 211 ± 37 ms, \( p < 0.001 \)) and E/A ratio decreased significantly (1.4 ± 0.3 to 1.0 ± 0.2, \( p < 0.01 \)). In contrast, peak E' velocity and E'/A' ratio did not change significantly (peak E' velocity 9.5 ± 2.2 cm/s to 9.2 ± 1.7 cm/s, \( p = \text{NS}; \) E'/A' ratio 1.2 ± 0.4 to 1.2 ± 0.4, \( p = \text{NS} \)) (Fig. 2).

Mitral annulus velocity and tau. In 38 patients (group 4) mitral annulus velocities as well as mitral inflow variables and pulmonary venous flow velocities were measured simultaneously with tau at the catheterization laboratory. Among the mitral annulus velocities, peak E' velocity (\( r = -0.56, p < 0.01 \)) and E'/A' ratio (\( r = -0.46, p < 0.01 \)) correlated with tau (Fig. 3). There were 17 patients who showed normal mitral inflow variables (deceleration time <240 ms and E/A ratio >1). Of these 17 patients, 8 showed prolonged tau (≥50 ms) and can be classified as having a pseudonormal mitral inflow pattern. When the combination of mitral annulus velocity with a peak E' velocity <8.5 cm/s and E'/A' ratio <1 was classified as indicating a relaxation abnormality, this mitral annulus velocity pattern could identify the underlying relaxation abnormality (based on tau ≥50 ms) with a specificity of 88% and specificity of 67% (Fig. 4).

Discussion

Mitral annulus motion. Motion of the mitral annulus represents changes in left ventricular long-axis dimension as the cardiac apex is relatively fixed during the cardiac cycle (9–11). In the absence of gross distortion of the ventricular shape or severe regional wall motion abnormalities, the changes in long-axis dimension could reflect left ventricular
volume changes. However, in evaluating diastolic function, assessment of the rate of change, rather than absolute change, in left ventricular volume is desirable. Pulsed wave DTI provides the capability of recording the low velocities of the moving wall structure with a high sampling rate. In this study we measured mitral annulus velocity at the septal side. This side of the mitral annulus, in contrast to other sides (lateral, anterior or inferior), moves in a direction more parallel to the ultrasound beam and is less affected by the translational movement of the heart; thus, there is a narrow band of spectral velocity and angle correction is not needed.

Mitral annulus motion shows two distinct movements toward the atrial side during diastole in patients with sinus rhythm. Early diastolic movement (E') begins simultaneously with the beginning of mitral inflow, but its peak velocity precedes the peak velocity of mitral inflow and ends well before the end of mitral inflow (12) (Fig. 5). Mitral inflow after the end of left ventricular lengthening in the long-axis dimension.

Table 1. Mitral Inflow, Pulmonary Venous Flow and Mitral Annulus Velocity in 59 Normal Volunteers

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>20–29 (n = 12; M/F 6:6)</th>
<th>30–39 (n = 12; M/F 6:6)</th>
<th>40–49 (n = 12; M/F 6:6)</th>
<th>50–59 (n = 12; M/F 6:6)</th>
<th>60–69 (n = 11; M/F 6:5)</th>
<th>Total (n = 59; M/F 30:29)</th>
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<tbody>
<tr>
<td>Mitral flow</td>
<td>Peak E velocity (cm/s)</td>
<td>85 ± 14</td>
<td>79 ± 13</td>
<td>70 ± 15</td>
<td>66 ± 11</td>
<td>65 ± 19</td>
</tr>
<tr>
<td></td>
<td>Peak A velocity (cm/s)</td>
<td>45 ± 9</td>
<td>49 ± 10</td>
<td>51 ± 13</td>
<td>55 ± 11</td>
<td>66 ± 11</td>
</tr>
<tr>
<td></td>
<td>E/A ratio</td>
<td>1.9 ± 0.4</td>
<td>1.7 ± 0.4</td>
<td>1.4 ± 0.4</td>
<td>1.3 ± 0.3</td>
<td>1.0 ± 0.2</td>
</tr>
<tr>
<td></td>
<td>DT (ms)</td>
<td>133 ± 29</td>
<td>144 ± 33</td>
<td>158 ± 37</td>
<td>180 ± 27</td>
<td>193 ± 25</td>
</tr>
<tr>
<td></td>
<td>IVRT (ms)</td>
<td>76 ± 8</td>
<td>85 ± 16</td>
<td>87 ± 10</td>
<td>95 ± 12</td>
<td>100 ± 12</td>
</tr>
<tr>
<td>Pulmonary venous flow</td>
<td>Peak systolic velocity (cm/s)</td>
<td>46 ± 8</td>
<td>47 ± 7</td>
<td>50 ± 10</td>
<td>53 ± 12</td>
<td>53 ± 6*</td>
</tr>
<tr>
<td></td>
<td>Peak diastolic velocity (cm/s)</td>
<td>52 ± 10</td>
<td>51 ± 8</td>
<td>46 ± 8</td>
<td>43 ± 7</td>
<td>42 ± 5*</td>
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<td>Systolic/diastolic ratio</td>
<td>0.9 ± 0.2</td>
<td>0.9 ± 0.1</td>
<td>1.1 ± 0.3</td>
<td>1.3 ± 0.3</td>
<td>1.3 ± 0.2*</td>
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<td>Peak A velocity (cm/s)</td>
<td>21 ± 6</td>
<td>23 ± 5</td>
<td>21 ± 4</td>
<td>26 ± 5</td>
<td>32 ± 16*</td>
</tr>
<tr>
<td>Mitral annulus velocity</td>
<td>Peak E' velocity (cm/s)</td>
<td>11.8 ± 1.4</td>
<td>13.0 ± 1.9</td>
<td>9.2 ± 1.4</td>
<td>8.5 ± 1.9</td>
<td>7.5 ± 1.6</td>
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<td>Peak A' velocity (cm/s)</td>
<td>8.6 ± 1.6</td>
<td>8.7 ± 1.8</td>
<td>9.2 ± 1.4</td>
<td>10.2 ± 1.5</td>
<td>10.7 ± 2.2</td>
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<tr>
<td></td>
<td>E'/A' ratio</td>
<td>1.4 ± 0.2</td>
<td>1.5 ± 0.5</td>
<td>1.0 ± 0.2</td>
<td>0.8 ± 0.2</td>
<td>0.7 ± 0.1</td>
</tr>
</tbody>
</table>

* n = 9; M/F = 5:4. A = late mitral inflow; A' = late diastolic mitral annulus motion; DT = deceleration time of early mitral inflow; E = early mitral inflow; E' = early diastolic mitral annulus motion; IVRT = isovolumetric relaxation time; M/F = male/female ratio.

Figure 1. Mitral inflow pattern (upper panel) and mitral annulus velocity pattern (lower panel) before and after saline loading. After loading, the mitral inflow pattern showed a trend toward pseudonormalization, whereas there was no significant change in the mitral annulus velocity pattern. DT = deceleration time of early mitral inflow.
sion, whether it is driven by pressure gradient or inertia, would lead to an increase in left ventricular volume in the short-axis dimension, which would mean higher compliance of the ventricular chamber in the short-axis dimension (13). Therefore, it may be assumed that a relaxation abnormality reflected in the long-axis dimension could potentially be evident earlier than clinical manifestation of global left ventricular relaxation abnormality. In our normal volunteers peak E’ velocity was higher than peak A’ velocity, mimicking the pattern of normalmitral inflow pattern (upper panel) and mitral annulus velocity pattern (lower panel) before and after intravenous nitroglycerin (NG) infusion. There were significant changes in the E velocity and deceleration time, but peak E’ velocity and E’/A’ ratio did not change significantly.

Figure 2. Mitral inflow pattern (upper panel) and mitral annulus velocity pattern (lower panel) before and after intravenous nitroglycerin (NG) infusion. There were significant changes in the E velocity and deceleration time, but peak E’ velocity and E’/A’ ratio did not change significantly.

Figure 3. Correlation between tau and deceleration time of early mitral inflow (DT), E/A ratio, peak E’ velocity and E’/A’ ratio.
mitral inflow. However, reversal of the E/A ratio in mitral inflow occurred in subjects in their 60s whereas reversal of the E'/A' ratio occurred in subjects in their 40s, a younger age group, thus supporting the preceding assumption.

The practical advantage of assessing the mitral annulus velocity is that this variable can be measured even in patients whose echocardiographic images are of poor quality in contrast to obtaining pulmonary venous flow pattern or assessing short-axis area change using acoustic quantification.

**Load dependence.** Mitral inflow variables are load dependent, and patients with a relaxation abnormality may show a normal pattern with elevated left atrial pressure. This pattern can occur because mitral inflow variables are velocity data determined by the pressure difference between the left atrium and left ventricle during diastole. Assessment of volume change has the theoretic advantage of being less preload dependent than mitral inflow variables. García et al. (14) observed that peak E' velocity correlates poorly with peak E velocity, suggesting the relative preload independence of peak E' velocity. In this study we demonstrated that, in contrast to mitral inflow velocity, peak early mitral annulus velocity did not change significantly after alteration of preload with infusion of saline solution or nitroglycerin.

**Mitral annulus velocity in the diagnosis of diastolic dysfunction.** Because it is less dependent on preload, the mitral annulus velocity profile should correlate better with tau than does deceleration time of mitral inflow. However, the mitral annulus velocity profile did not correlate very well with tau, partly because mitral annulus velocity reflects only relaxation in the long-axis dimension. Of our 17 patients with normal mitral inflow variables, 8 had prolonged tau (pseudonormalization). Peak E' velocity <8.5 cm/s and E'/A' ratio <1 best discriminated a pseudonormal from a normal filling pattern (sensitivity 88%, specificity 67%). The pulmonary venous flow pattern has been used to differentiate pseudonormal from normal
diastolic function. However, atrial reversal was <0.3 m/s and systolic/diastolic velocity was >1 in all of our eight patients with pseudonormalization. Thus, the pulmonary venous flow pattern was not helpful in detecting pseudonormalization in this subgroup of our study.

Our study results indicate that peak E\textsuperscript{9} velocity is reduced, resulting in a reversal of the E\textsuperscript{9}/A\textsuperscript{9} ratio, in patients with a relaxation abnormality. In contrast to mitral inflow velocity, mitral annulus velocity is relatively preload independent. The mitral annulus velocity profile in patients with restrictive physiology was previously described (14) to be associated with a decrease in E\textsuperscript{9} and A\textsuperscript{9}. Hence, peak early mitral annulus velocity decreases from normal to restrictive physiology, whereas mitral inflow shows a pseudonormal pattern when a relaxation abnormality changes to restrictive physiology (Fig. 6). Therefore, measurement of early mitral annulus velocity should be useful in distinguishing a pseudonormal from a normal diastolic filling pattern.

**Limitations of the study.** Our study has several limitations. 1) Because we excluded patients with a regional wall motion abnormality, we are not convinced that our results can be applied to such patients, especially those with a basal septal wall motion abnormality. 2) We measured mitral annulus velocity at the septal side for the reasons mentioned. However, this approach has the potential disadvantage of being affected by right ventricular function; thus, our results cannot be applied to patients with significant pulmonary hypertension or impaired right ventricular function. 3) All patients in our study had sinus rhythm; the application of our findings to patients with atrial fibrillation should be studied in the future.

**Conclusions.** Mitral annulus motion is less load dependent than conventional mitral inflow variables. Its assessment by DTI appears to be useful for evaluating diastolic function, especially in the detecting a pseudonormalization pattern of mitral inflow.

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**References**

3. Choong CV, Herrmann HC, Weymann AE, Fifer MA. Preload dependence


