Striking Effect of Left Ventricular High Filling Pressure with Mitral Regurgitation on Mitral Annular Velocity During Early Diastole. A Study Using Colour M-mode Tissue Doppler Imaging

N. Ohte, H. Narita, S. Akita, K. Kurokawa, J. Hayano and G. Kimura

Third Department of Internal Medicine, Nagoya City University Medical School, Nagoya, Japan

Aims: To evaluate the effect of considerably high left ventricular filling pressure with mitral regurgitation on mitral annular velocity during early diastole.

Subjects: Two hundred and forty-three patients who underwent cardiac catheterization for evaluation of chest pain.

Methods: Mitral annular velocity during early diastole was measured by colour M-mode tissue Doppler imaging. Patients were divided into the following three groups according to the cardiac catheterization data. Group A (n=147): patients having left ventricular relaxation time constant \( r < 46 \) ms and left ventricular end-systolic volume index \( < 38 \) ml m\(^{-2}\); group B (n=88): patients having \( r \geq 46 \) ms and/or end-systolic volume index \( \geq 38 \) ml m\(^{-2}\); group C (n=8): patients having mean pulmonary capillary wedge pressure \( \geq 16 \) mmHg in addition to \( r \geq 46 \) ms and end-systolic volume index \( \geq 38 \) ml m\(^{-2}\).

Results: Mitral annular velocity during early diastole was significantly less in group B \( (4.8 \pm 1.4 \text{ cm s}^{-1}) \) than in group A \( (7.7 \pm 1.9 \text{ cm s}^{-1}) \). However, there was no significant difference between groups A and C \( (8.3 \pm 0.8 \text{ cm s}^{-1}) \). A transmitral E/A >1:0 was observed in 12/147 patients of group A, 10/88 of group B, and 8/8 of group C. The incidence of \\( \geq \)Sellers’ grade II mitral regurgitation was higher in group C than the others.

Conclusions: A paradoxically faster mitral annular velocity during early diastole is found in patients having left ventricular dysfunction with moderate to severe mitral regurgitation and considerably high left ventricular filling pressure. Attention should be paid to an interpretation of mitral annular velocity during early diastole regarding left ventricular early diastolic performance in patients having mitral regurgitation with an E/A >1:0 in their transmitral flow.

Key Words: left ventricular function; mitral annular velocity; tissue Doppler imaging; mitral regurgitation.

Introduction
It has been reported that mitral annular velocity during early diastole is a reliable parameter of left ventricular early diastolic performance\(^{[1-9]}\). Furthermore, several investigators, including ourselves, have demonstrated that the parameter is relatively independent of left ventricular filling pressure\(^{[1-4,6]}\). A considerably high left ventricular filling pressure, however, accelerates the early diastolic filling flow velocity, producing a condition called pseudonormalization or a restrictive pattern of left ventricular filling\(^{[7,8]}\). In such a condition, the left ventricles with remodelling due to their dysfunction may undergo early diastolic filling volumes without a rapid movement of the mitral annulus along the left ventricular long axis. In fact, however, we found several patients who had relatively faster mitral annular velocity during early diastole with a high left ventricular filling pressure.

Address for correspondence: Nobuyuki Ohte, MD, Third Department of Internal Medicine, Nagoya City University Medical School, 1 Kawasumi, Mizuho-cho, Mizuho-ku, Nagoya 467-8601, Japan. Tel: +81-52-853-8221; Fax: +81-52-852-3796; E-mail: ohte@med.nagoya-cu.ac.jp

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and mitral regurgitation, although they had severely impaired left ventricular systolic and early diastolic performance because of coronary artery disease. Thus, we hypothesized that the left ventricular early diastolic filling volume may be received by longitudinal distention of the left ventricles in patients with a considerably high left ventricular filling pressure with mitral regurgitation. Accordingly, we investigated this issue in a relatively large number of patients suffering from coronary artery disease.

Subjects and Methods

We screened 248 consecutive patients who underwent cardiac catheterization for evaluation of chest pain. A total of 201 patients was defined as having coronary artery disease. Among these patients, 154 were diagnosed as having coronary artery disease with prior myocardial infarction and 47 as those without prior myocardial infarction. The other 47 patients were diagnosed as having atypical chest pain; their electrocardiograms, echocardiograms, and left ventriculograms were normal; their coronary arteries did not meet the criteria for significant coronary stenosis. Patients with an acute myocardial infarction and those with primary valvular heart diseases were excluded from the study. Patients whose Doppler echocardiographic imaging was of poor quality were also excluded. Thus, the final study population consisted of 45 patients with atypical chest pain and 198 patients with coronary artery disease (153 with and 45 without prior myocardial infarction). They consisted of 202 males and 41 females, and their mean age was 62·3 ± 8·0 years. Among the patients with prior myocardial infarction, 97 were diagnosed as having anterior wall myocardial infarction and 56 as inferior wall myocardial infarction on the basis of the extent of left ventricular wall motion abnormality. However, 14 patients had a history of repeated myocardial infarction, being classified into either anterior or inferior myocardial infarction based on the extension of the asynergic region. Twenty-eight of the patients with coronary artery disease had mitral regurgitation due to papillary muscle dysfunction and/or left ventricular enlargement. Except for sublingual nitroglycerin and diuretics, all heart medications were routinely discontinued at least 3 days before cardiac catheterization. All subjects gave written informed consent for participation in the study. The study was performed according to the approval by the Ethical Guidelines Committee of Nagoya City University Medical School.

Colour M-mode Tissue Doppler Imaging and Pulsed Doppler Echocardiography

Colour M-mode tissue Doppler imaging was obtained at the endocardium of the posterolateral corner of the mitral annulus using an echocardiograph (PowerVision 6000, Toshiba Co., Tokyo, Japan) with two-dimensional image guidance of the apical long-axis view. In our study, the posterolateral corner of the mitral annulus was the most clearly and consistently visualized place on the two-dimensional images in the patients, so that we chose this place for the measurement of mitral annular velocity during early diastole. Imaging was performed with a 3·7 MHz transducer with a pulse repetition frequency of 4·5 KHz. On this occasion, care was taken to align the M-mode cursor so that the Doppler angle of incidence was as close to 0° as possible with mitral annular motion. The region of interest for measuring the wall motion velocity was placed at the endocardium, which had the brightest blue colour mingling with white on colour M-mode tissue Doppler tracings (Fig. 1). Appropriate maximal velocity limits for displaying the wall motion velocity were used to clearly reveal the phase of its top speed on colour M-mode tracings. As upper velocity limits, one of 4·80 cm s⁻¹, 6·48 cm s⁻¹, 7·20 cm s⁻¹, 9·72 cm s⁻¹, or 14·4 cm s⁻¹ was employed in each patient. Thus, mitral annular velocity during early diastole was measured as the highest velocity of the endocardium that moved away from the apex. Mitral annular velocity during early diastole was measured on three consecutive cardiac cycles, and their mean was used for statistical analysis. The size of the square region of interest consisted of nine pixels, each of which was 0·409 mm in height and 4·17 ms in width on M-mode tracings.

Immediately after tissue Doppler examination, pulsed Doppler echocardiography was conducted to obtain the E/A ratio of the transmural flow at the mitral tip level in the left atrium. A mean of the E/A ratio obtained from three consecutive cardiac cycles was used for the analysis. Reproducibility in these measurements was reported elsewhere.⁴⁻⁵. The heart rate of each subject was obtained from the electrocardiogram. Contiguously after the Doppler examination, we measured arterial blood pressure with a sphygmomanometer.

Cardiac Catheterization

Diagnostic cardiac catheterization was performed within 2 h after the echocardiographic study in the patients. As we previously reported⁴⁻⁵, pulmonary capillary wedge pressure was obtained by a flow-directed pulmonary artery catheter. Left ventricular pressure was then obtained with a micromanometer-tipped catheter (SPC-464D, Millar Instrument Co., Houston, U.S.A.). From the recorded pressure waves, a time constant ‘τ’ of the fall in left ventricular pressure was computed by applying a monoexponential fitting with zero asymptote to the left ventricular pressure decay⁹. Just after the pressure measurement, biplane contrast left ventriculography was performed. Left ventricular end-systolic volume and left ventricular end-diastolic volume were then calculated using the method of Chapman et al.⁹. The left ventricular ejection fraction was then determined. The left
ventricular end-systolic volume was divided by the body surface area of each patient, being expressed as an left ventricular end-systolic volume index. Mitral regurgitation was semi-quantitatively graded according to the criteria proposed by Sellers et al.[11].

Classification of Patients

Subjects were divided into the following three groups according to the haemodynamic data obtained by cardiac catheterization. Group A: patients with relatively better left ventricular performance ($\tau < 46$ ms and left ventricular end-systolic volume index $< 38$ ml m$^{-2}$); Group B: patients with relatively worse left ventricular performance ($\tau \geq 46$ ms and/or left ventricular end-systolic volume index $\geq 38$ ml m$^{-2}$); Group C: patients with considerably high mean pulmonary capillary wedge pressure in addition to worse left ventricular performance (mean pulmonary capillary wedge pressure $\geq 16$ mmHg with $\tau \geq 46$ ms and left ventricular end-systolic volume index $\geq 38$ ml m$^{-2}$). The threshold values of discriminating the patients with relatively better left ventricular performance from those with relatively worse were derived from each mean value of the $\tau$ and left ventricular end-systolic volume index in the patients with coronary artery disease. The patients having a considerably high left ventricular filling pressure were determined as those with mean pulmonary capillary wedge pressure higher than the mean plus 2 SD (standard deviation) of the measured pressures in the coronary artery disease patients ($8.9 \pm 3.5$ mmHg).

Statistical Analysis

Results are presented as mean $\pm$ SD. Differences in parameters between two groups were compared using Student’s unpaired $t$-test and those in parameters among three groups were compared using the analysis of variance with a Bonferroni adjustment. Relationships between parameters in two groups were evaluated by univariate linear regression analysis. Stepwise multivariate linear regression analysis was performed to determine which independent variables would significantly affect the mitral annular velocity during early diastole. The independent variables were age, heart rate, mean blood pressure, left ventricular end-systolic volume index, time constant $\tau$, and mean pulmonary capillary wedge pressure. Differences in incidences of the E/A
Table 1. Clinical characteristics and cardiac catheterization data of the patients in each group.

<table>
<thead>
<tr>
<th></th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>147</td>
<td>88</td>
<td>8</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>116/31</td>
<td>78/10</td>
<td>8/0</td>
</tr>
<tr>
<td>Age (years)</td>
<td>62·7 ± 7·9</td>
<td>61·8 ± 8·0</td>
<td>60·8 ± 7·8</td>
</tr>
<tr>
<td>Heart rate (beats m⁻¹)</td>
<td>66·0 ± 10·3</td>
<td>66·2 ± 12·1</td>
<td>73·1 ± 6·9</td>
</tr>
<tr>
<td>Mean blood pressure (mmHg)</td>
<td>90·8 ± 11·3</td>
<td>90·1 ± 11·6</td>
<td>85·7 ± 9·8</td>
</tr>
<tr>
<td>Time constant τ (ms)</td>
<td>40·7 ± 4·5</td>
<td>49·6 ± 5·8*</td>
<td>58·5 ± 2·9†</td>
</tr>
<tr>
<td>LV end-systolic volume index (ml m⁻³)</td>
<td>23·8 ± 8·4</td>
<td>48·2 ± 21·3*</td>
<td>71·1 ± 14·6†</td>
</tr>
<tr>
<td>LV ejection fraction (%)</td>
<td>68·3 ± 9·1</td>
<td>53·3 ± 11·5*</td>
<td>35·1 ± 4·0†</td>
</tr>
<tr>
<td>Mean pulmonary capillary wedge pressure (mmHg)</td>
<td>7·7 ± 2·5</td>
<td>9·1 ± 3·0*</td>
<td>20·1 ± 1·6†</td>
</tr>
</tbody>
</table>

Data are presented as mean ± SD.
*P<0.001 vs group A, †P<0.001 vs group B.

Group A: patients having LV relaxation time constant τ < 46 ms and left ventricular end-systolic volume index < 38 ml/m².
Group B: patients having τ ≥ 46 ms and/or left ventricular end-systolic volume index ≥ 38 ml/m².
Group C: patients having mean pulmonary capillary wedge pressure ≥ 16 mmHg in addition to τ ≥ 46 ms and left ventricular end-systolic volume index ≥ 38 ml/m².

>1·0 and of mitral regurgitation between groups were compared using the χ²-test. Statistical significance was accepted when P<0.05.

Results

One hundred and forty-seven patients were classified into group A, 88 into group B, and eight into group C (Table 1). According to the classification, there were clear differences in time constant τ, left ventricular end-systolic volume index, left ventricular ejection fraction, and mean pulmonary capillary wedge pressure among the three groups. No significant differences were observed in age, heart rate, or mean blood pressure among the three groups. Mitral annular velocity during early diastole was significantly greater in group A (7·7 ± 1·9 cm s⁻¹) than in group B (4·8 ± 1·4 cm s⁻¹) (P<0.001). Mitral annular velocity during early diastole in group C (8·3 ± 0·8 cm s⁻¹) was also significantly greater compared with that in group B (P<0.001). However, no significant difference was found in mitral annular velocity during early diastole between groups A and C (Fig. 2). In patients other than those with inferior myocardial infarction, mitral annular velocity during early diastole was also faster in group A (7·8 ± 1·9 cm s⁻¹, n=119) and group C (8·3 ± 0·9 cm s⁻¹, n=7) than in group B (4·5 ± 1·4 cm s⁻¹, n=62) (P<0.001). No significant difference was observed between groups A and C. In patients with inferior myocardial infarction, mitral annular velocity during early diastole was faster in group A (7·0 ± 1·9 cm s⁻¹, n=28) than in group B (5·3 ± 1·2 cm s⁻¹, n=26) (P<0.005). Only one patient with inferior myocardial infarction was assigned to group C, and its mitral annular velocity during early diastole was 8·2 cm s⁻¹.

Univariate linear regression analysis showed that time constant τ and left ventricular end-systolic volume index were both significantly correlated with mitral annular velocity during early diastole, although the correlation coefficients were not so high (Figs 3 and 4). As shown in these figures, the group C patients had relatively preserved left ventricular systolic function despite the fact that they had severely impaired left ventricular relaxation. In contrast, mean pulmonary capillary wedge pressure, age, heart rate, or mean blood pressure were not significantly correlated with mitral annular velocity during early diastole (Table 2). Stepwise multivariate

Figure 2. Comparison of mitral annular velocity (MAV) during early diastole among the three classified groups. Mitral annular velocity during early diastole was faster in groups A and C than in group B. No significant difference was observed between groups A and C. Abbreviations as in Table 1.
III. The incidence of accompanying mitral regurgitation

patients had Sellers’ grade II and three others had grade

patients in group C had mitral regurgitation; five

had Sellers’ grade III regurgitation. In contrast, all

mitral regurgitation of Sellers’ grade II but no one

observed in group A. Three patients in group B had

mitral annular velocity during early diastole (Table 3).

regression analysis demonstrated that in addition to left

ventricular end-systolic volume index and time constant τ, mean pulmonary capillary wedge pressure and other

clinical factors had partial but significant effects on mitral annular velocity during early diastole (Table 3).

An E/A > 1.0 in transmitral flow was observed in 12 of

147 group A patients (8.2%), in 10 of 88 group B

patients (11.4%), and in all 8 group C patients (100%).
The incidence of an E/A > 1.0 was significantly higher in

patients (11.4%), and in all 8 group C patients (100%).

It has been reported that Doppler tissue velocities of the

left ventricular longitudinal direction during early diastole at the mitral annulus or at the myocardium close to it are a useful tool for the assessment of left ventricular early diastolic performance[1–3]. We have reported that mitral annular velocity during early diastole had a significant relationship with the left ventricular end-systolic volume index, as well as with left ventricular relaxation, providing important information regarding left ventricular behaviour from end-systole to early diastole where left ventricular early diastolic performance is determined[4–5]. From the viewpoint of the effect of preload on mitral annular velocity during early diastole, Nagueh et al.[6] showed that mean pulmonary capillary wedge pressure did not affect the mitral annular velocity during early diastole. Sohn et al.[7] indicated that mitral annular velocity during early diastole was not changed significantly by altering the preload with an infusion of saline solution or nitroglycerin. Farias et al.[8] also reported that mitral annular velocity during early diastole reflected myocardial dysfunction in patients with heart diseases even though their transmitral flow shows a pseudonormal or restrictive pattern. Furthermore, Oki et al.[9] demonstrated that peak early diastolic myocardial velocity at the left ventricular posterior wall correlated well with the left ventricular relaxation constant τ in patients with heart diseases regardless of their left ventricular filling pressure. 

Our previous study[10] of a relatively small population with coronary artery disease also showed no significant correlation between the mitral annular velocity during early diastole and mean pulmonary capillary wedge pressure. Without rapid mitral annular motion, the enlarged left ventricles with their dysfunction could receive a relatively large early diastolic filling volume accompanied by high left ventricular filling pressure. However, in this series of subjects, we actually recognized that in patients who had high left ventricular filling pressure with mitral regurgitation as well as relatively worse left ventricular function, mitral annular velocity during early diastole reflected early diastolic filling flow velocity as a mirror image, being similar to that observed in healthy normal subjects[11]. We therefore examined the mechanisms of this phenomenon that is similar to the ‘pseudonormalization’ originally observed in transmital flow velocity waveforms[12].

≥ Sellers’ grade II was also higher in group C than in the other groups.

Discussion

The present study demonstrates that mitral annular velocity during early diastole is paradoxically faster in patients having considerably high mean pulmonary capillary wedge pressure, although they had severely deteriorated left ventricular function. In such patients, mitral regurgitation ≥Sellers’ grade II was commonly observed.

Figure 3. Relationship between the time constant τ of left ventricular relaxation and mitral annular velocity during early diastole. A significant negative correlation was observed. The circles (arrow) indicate the patients with mean pulmonary capillary pressure ≥ 16 mmHg.

Figure 4. Relationship between the left ventricular end-systolic volume index and mitral annular velocity during early diastole. A significant negative correlation was found. Patients with mean pulmonary capillary wedge pressure ≥ 16 mmHg are shown as circles (arrow).

Mitral regurgitation ≥Sellers’ grade II was not observed in group A. Three patients in group B had mitral regurgitation of Sellers’ grade II but no one had Sellers’ grade III regurgitation. In contrast, all patients in group C had mitral regurgitation; five patients had Sellers’ grade II and three others had grade III. The incidence of accompanying mitral regurgitation
Mean pulmonary capillary wedge pressure did not significantly correlate with mitral annular velocity during early diastole in the univariate regression analysis in the present investigation, as it did not in previous studies[1–4]. However, it has become apparent that mean pulmonary capillary wedge pressure does somewhat affect mitral annular velocity during early diastole after adjusting for the influences of other parameters in the multivariate regression analysis. Thus, it is possible that considerably high mean pulmonary capillary wedge pressure may have accelerated mitral annular velocity during early diastole by overcoming the effect of left ventricular dysfunction which reduces mitral annular velocity during early diastole. All patients with high mean pulmonary capillary wedge pressure had mitral regurgitation/Sellers’ grade II in this investigation. The left ventricles could eject blood easily to the left atrium and reduce their volumes with a greater magnitude of systolic displacement of the mitral annulus to the apex in patients with mitral regurgitation than in those without mitral regurgitation, because the left atrium is a low-pressure chamber compared with the aorta[13]. Thus, in moderate to severe mitral regurgitation, the mitral annulus travels the relatively longer path to its pre-systolic position during diastole. Alam et al.[12] indicated that mitral annular velocity during early diastole was well correlated with the M-mode recorded magnitude of mitral annular motion during early diastole. Consequently, in patients with mitral regurgitation, rapid and large early diastolic left ventricular filling produced by high left ventricular filling pressure may speed up the early diastolic motion of the mitral annulus[14], although left ventricular elastic recoil and left ventricular relaxation of the impaired left ventricles would not be sufficient to do so.

Mean pulmonary capillary wedge pressure did not significantly correlate with mitral annular velocity during early diastole in the univariate regression analysis in the present investigation, as it did not in previous studies[1–4]. However, it has become apparent that mean pulmonary capillary wedge pressure does somewhat affect mitral annular velocity during early diastole after adjusting for the influences of other parameters in the multivariate regression analysis. Thus, it is possible that considerably high mean pulmonary capillary wedge pressure may have accelerated mitral annular velocity during early diastole by overcoming the effect of left ventricular dysfunction which reduces mitral annular velocity during early diastole. All patients with high mean pulmonary capillary wedge pressure had mitral regurgitation/Sellers’ grade II in this investigation. The left ventricles could eject blood easily to the left atrium and reduce their volumes with a greater magnitude of systolic displacement of the mitral annulus to the apex in patients with mitral regurgitation than in those without mitral regurgitation, because the left atrium is a low-pressure chamber compared with the aorta[13]. Thus, in moderate to severe mitral regurgitation, the mitral annulus travels the relatively longer path to its pre-systolic position during diastole. Alam et al.[12] indicated that mitral annular velocity during early diastole was well correlated with the M-mode recorded magnitude of mitral annular motion during early diastole. Consequently, in patients with mitral regurgitation, rapid and large early diastolic left ventricular filling produced by high left ventricular filling pressure may speed up the early diastolic motion of the mitral annulus[14], although left ventricular elastic recoil and left ventricular relaxation of the impaired left ventricles would not be sufficient to do so.

As shown in Figs 2 and 3, the correlations of the left ventricular end-systolic volume index and the left ventricular relaxation time constant to the mitral annular velocity during early diastole were significant but the correlation coefficients were not so high. These findings suggest that determinants of mitral annular velocity during early diastole were multifactorial, as shown in the multivariate regression analysis in this study. The patients with high mean pulmonary capillary wedge pressure and mitral regurgitation had relatively preserved left ventricular systolic function in contrast to severely impaired left ventricular relaxation as shown in these Figs 2 and 3. This may also have made the mitral annular velocity during early diastole faster in these patients.

Several investigators have reported that in patients with diffuse myocardial diseases such as hypertrophic, dilated and hypertensive cardiomyopathies, myocardial velocities during early diastole deteriorate together with left ventricular diastolic dysfunction, even if they have pseudonormalized or restrictive filling[2,15,16]. In contrast, our study was conducted in patients with coronary artery disease who had at least some normal parts of left ventricular myocardium, even though they had severe global systolic and/or diastolic left ventricular dysfunction. This may also explain why our result is somewhat different to that of previous studies.

### Table 2. Univariate linear regression analysis for mitral annular velocity during early diastole.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Correlation coefficient</th>
<th>P values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>r = -0.11</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rate</td>
<td>r = -0.11</td>
<td>NS</td>
</tr>
<tr>
<td>Mean blood pressure</td>
<td>r = 0.04</td>
<td>NS</td>
</tr>
<tr>
<td>Time constant r</td>
<td>r = -0.42</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>LV end-systolic volume index</td>
<td>r = -0.48</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>Mean pulmonary capillary wedge pressure</td>
<td>r = -0.08</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS, not significant; other abbreviations as in Table 1.

### Table 3. Stepwise multivariate regression analysis for mitral annular velocity during early diastole.

<table>
<thead>
<tr>
<th>Variables</th>
<th>β</th>
<th>Partial R² (%)</th>
<th>F value</th>
<th>P value</th>
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</thead>
<tbody>
<tr>
<td>LV end systolic volume index</td>
<td>-0.35</td>
<td>23</td>
<td>26.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Time constant r</td>
<td>-0.37</td>
<td>4</td>
<td>25.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mean pulmonary capillary wedge pressure</td>
<td>0.22</td>
<td>3</td>
<td>12.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Age</td>
<td>-0.16</td>
<td>2</td>
<td>9.18</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Mean blood pressure</td>
<td>-0.11</td>
<td>1</td>
<td>4.33</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Heart rate</td>
<td>-0.12</td>
<td>1</td>
<td>4.16</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

Abbreviations as in Tables 1 and 2.
Limitations

The location of myocardial infarction may affect mitral annular velocity during early diastole obtained at the posterolateral corner of the mitral annulus. However, quite similar results were observed regarding the difference in mitral annular velocity during early diastole among the three classified groups, even in patients with or other than those with inferior myocardial infarction. The mitral annulus is made of fibrous tissue, and is harder than the myocardium. Therefore, in the chronic stage of myocardial infarction, global left ventricular performance rather than local asynergy should have been reflected in its motion. Nagueh et al[1] also reported that evaluation of left ventricular diastolic function using mitral annular velocity during early diastole at the lateral corner of the mitral annulus is applicable even in patients with local asynergy caused by coronary artery disease.

Although the number of patients who belonged to group C was small, the invasively obtained haemodynamic characteristics of those were quite typical. We believe that to clarify the clinical significance of mitral annular velocity during early diastole in such patients is important in their management.

Conclusions

Attention should be paid to interpreting the haemodynamic significance of mitral annular velocity during early diastole regarding left ventricular early diastolic performance in patients having mitral regurgitation with the E/A >1·0 on their transmitral flow. Mitral regurgitation may disrupt the involvement between the reduced mitral annular velocity during early diastole and left ventricular early diastolic dysfunction.

References


