Effects of First Myocardial Infarction on Left Ventricular Systolic and Diastolic Function with the Use of Mitral Annular Velocity Determined by Pulsed Wave Doppler Tissue Imaging

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This study was undertaken to assess the effect of a first myocardial infarction (MI) on the systolic and diastolic velocity profiles of the mitral annulus determined by pulsed wave Doppler tissue imaging and thereby evaluate left ventricular (LV) function after MI. Seventy-eight patients with a first MI were examined before discharge. Peak systolic, peak early diastolic, and peak late diastolic velocities were recorded at 4 different sites on the mitral annulus corresponding to the septum, anterior, lateral, and inferior sites of the left ventricle. In addition, the amplitude of mitral annular motion at the 4 above LV sites, the ejection fraction, and conventional Doppler diastolic parameters were recorded. Nineteen age-matched healthy subjects served as controls. Compared with healthy subjects, the MI patients had a significantly reduced peak systolic velocity at the mitral annulus, especially at the infarction sites. A relatively good linear correlation was found between the ejection fraction and the mean systolic velocity from the 4 LV sites ($r = 0.74$, $P < .001$). The correlation was also good when the mean peak systolic mitral annular velocity was tested against the magnitude of the mean mitral annular motion ($r = 0.77$, $P < .001$). When the patients were divided into 2 different groups with respect to an ejection fraction ≥0.50 or <0.50, a cutoff point of mean systolic mitral annular velocity of ≥7.5 cm/s had a sensitivity of 79% and a specificity of 88% in predicting a preserved global LV systolic function. Similar to systolic velocities, the early diastolic velocity was also reduced, especially at the infarction sites. The peak mitral annular early diastolic velocity correlated well with both LV ejection fraction ($r = 0.66$, $P < .001$) and mean systolic mitral annular motion ($r = 0.68$, $P < .001$). However, no correlation existed between the early diastolic velocity and conventional diastolic Doppler parameters. The reduced peak systolic mitral annular velocity seems to be an expression of regionally reduced systolic function. The peak early diastolic velocity is also reduced, especially at the infarction sites, and reflects regional diastolic dysfunction. Thus, quantification of myocardial velocity by Doppler tissue imaging opens up a new possibility of assessing LV function along its long axis. (J Am Soc Echocardiogr 2000;13:343-52.)

INTRODUCTION

The conventional echocardiographic methods for assessing cardiac function are based on endocardial movement and/or wall thickening and have several limitations, especially in cases of unsatisfactory echo quality. Doppler tissue imaging (DTI) is a new technique in echocardiography that records the myocardial velocities during the cardiac cycle.[1,2] Both the systolic and diastolic velocities can be recorded quantitatively by DTI and thereby provide a new way of assessing left ventricular (LV) function. Assessment of cardiac function by DTI may be more sensitive than traditional methods. However, the method is still under development, and reference values for clinical application have not yet been determined.

Most previous studies have compared DTI by color mapping with other conventional parame-

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0894-7317/2000 $12.00 + 0 **27/1/104393**

doi:10.1067/mje.2000.104393

ters.[3-6] However, a few studies have used pulsed wave DTI to assess LV contraction and relaxation velocities.[1,7] The use of pulsed wave DTI at the mitral annulus allows investigation of the contraction and relaxation velocities of the left ventricle along its longitudinal direction. A complete study of LV function by the pulsed wave DTI technique requires assessment of myocardial velocity profiles at different sites of the left ventricle. This is particularly important in the presence of regional wall motion abnormalities after an acute myocardial infarction (MI). With the use of conventional echocardiography, we previously reported that the amplitude of LV atrioventricular plane displacement (ie, the mitral annular motion) at 4 different sites (septal, anterior, lateral, and inferior) of the left ventricle can easily be used to assess LV systolic function.[8-10] A previous study from our laboratory using the same method during diastole described a feasible method of assessing diastolic function in healthy subjects.[11] It is easy to record mitral annular motion because it is devoid of trabeculae, myocardial dropouts, etc, which could otherwise prevent the assessment of LV function in a conventional way. However, little is known about the ability of pulsed wave DTI to assess LV function during ischemia and MI.[12,13] A systematic study of both systolic and diastolic LV function after an acute first MI is still missing.

The purpose of the present study is to record with pulsed wave DTI the effects of a first MI on the velocity profile of the left ventricle during systole and diastole on several sites of the mitral annulus and to compare them with standard echocardiographic parameters.

MATERIAL AND METHODS

Subjects

We started with 101 patients with acute MI. Twenty-three patients were excluded because of previous MIs, presence of left bundle branch block on the electrocardiogram (ECG), presence of LV aneurysm, or normal LV wall motion at echocardiography. Finally, 78 patients (59 men, 19 women) with a first-time acute MI were included to investigate systolic LV function. All patients were in sinus rhythm. Twenty-two patients had systemic hypertension, 12 had diabetes mellitus, and 3 patients had angina pectoris. The diagnosis of MI was based on the following criteria: (1) characteristic chest pain, (2) ECG changes, and (3) diagnostic serial changes in cardiac enzymes. Forty-nine patients were treated with thrombolysis. Fifty-five patients developed Q waves on the ECGs. Thirty-three patients showed signs of anterior MI, and 45 had inferior MI on the ECG. Infarction localization was also confirmed by echocardiographic determination of the presence of hypokinetic and/or akinetic segments at the respected areas. The mean value (SD) for maximum creatine kinase–MB was 179 (100) and for s-AST was 5.95 (5.2).

For LV diastolic studies, only 47 patients were included, that is, those who were previously healthy and without a history of systemic hypertension, valvular heart disease, cardiomyopathy, diabetes mellitus, ischemic heart disease, or other disease that might influence diastolic function. Twenty-five age-matched healthy subjects without a history of cardiac disease or systemic hypertension and who had normal findings on the rest ECG and at echocardiography served as controls.

Echocardiography

The patients were examined in the left lateral decubitus position 2 to 4 days after MI with a Hewlett-Packard Sonos 5500 (Andover, Mass) phased-array system equipped with DTI technology. Measurements of different cardiac chambers were made according to the recommendations of the American Society of Echocardiography.[14] The ejection fraction was calculated as percentage of change in LV chamber volumes between diastole and systole from apical 4- and 2-chamber views with Simpson's method.[14] The magnitude of mitral annular motion at 4 different LV sites (at the septal, anterior, lateral, and inferior walls) was also recorded by the method described earlier.[8,10]

Doppler Echocardiography

From the apical 4-chamber view, pulsed wave Doppler mitral flow velocities were recorded by placing the sample volume between the leaflet tips in the center of the flow stream.[15] Doppler gain and filters were adjusted to obtain the best spectral recordings and the transmitral peak rapid filling velocity (E), peak atrial filling velocity (A), E-wave deceleration time, and E/A ratio were measured. The pulmonary venous flow was recorded from the apical 4-chamber view by inserting the pulsed wave Doppler sample volume approximately 1 cm into the right upper pulmonary vein.[16] The pulmonary venous peak systolic (S), diastolic (D), S/D ratio, and the reverse flow velocity caused by atrial contraction were recorded at a speed of 100 mm/s. Left ventricular isovolumic relaxation time (IVRT) was recorded from the apical 4-chamber view by simultaneous recording of the mitral and aortic flows[17] at a speed of 100 mm/s.

Mitral Annular Velocity by Doppler Tissue Imaging

The pulsed wave DTI was performed by activating the DTI function in the same echocardiographic machine. Images were acquired by using a variable frequency phased-array transducer (2.0 to 4.0 MHz). The filter settings were kept low (50 Hz), and gains were adjusted at the minimal opti-

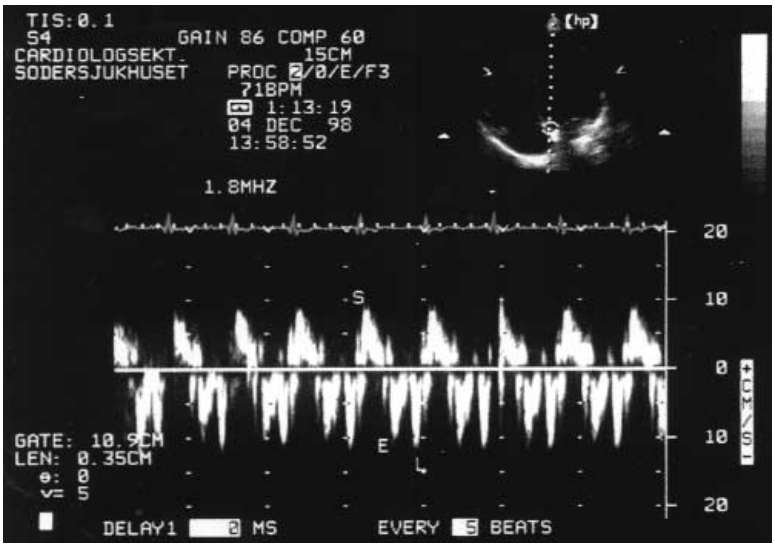

Figure 1 Two-dimensional apical 4-chamber view with the sample volume of the Doppler myocardial velocity recording at the septal border of the mitral annulus *(upper part of figure)*. The systolic *(S)* and early *(E)* and late *(L)* diastolic velocities (cm/s) are shown in the *lower part* of the figure.

mal level to minimize noise and eliminate the signals produced by the transmitral flow. A 3.5-mm sample volume was used. Four different sites at the mitral annulus were selected. In the apical 4-chamber view, the DTI cursor was placed at the septal side of the mitral annulus in such a way that the mitral annulus at the septum moved along the sample volume line. A Doppler velocity range of –20 to 20 cm/s was selected for this study. Three major velocities were recorded: the positive systolic velocity when the mitral ring moved toward the cardiac apex, and 2 negative diastolic velocities when the mitral annulus moved toward the base away from the apex (one during the early phase of diastole and another in the late phase of diastole) (Figure 1). The peak systolic and diastolic velocities were measured on-line at a sweep speed of 50 mm/s. By moving the sample volume at the lateral site of the mitral annulus, systolic and diastolic velocities of the LV lateral wall were also recorded. In a similar way, the velocities at the anterior and inferior sites of the mitral annulus were recorded in the apical 2-chamber view. A mean value from the above 4 sites was used to assess global systolic and diastolic function. A mean of 3 consecutive cycles was used to calculate all Doppler echocardiography parameters.

Statistics

Results are expressed as the mean and one SD. Comparisons of the results were made with the Student *t* test (unpaired test between healthy subjects and MI patients, and paired test between different parameters within the same group). Linear regression analysis was used for comparison of different parameters. A P value <.05 was considered statistically significant. To test intraobserver variability, measurements of systolic and diastolic DTI were made at 41 LV sites on 2 different occasions. For interobserver variability, a second investigator randomly made measurements at 33 of the above different LV sites without knowledge of other echocardiographic parameters. The intraobserver and interobserver variabilities were determined as the difference between the 2 sets of observations divided by the mean of the observations and expressed as a percentage.[7]

RESULTS

The basic clinical and echocardiographic parameters for the patients and the healthy subjects are given in Table 1. The mean mitral annular motion at 4 LV sites was 9.7 ± 1.9 mm in anterior MI and 10.7 ± 1.6 mm in inferior MI (P < .01). The intraobserver error was low: 4.6% ± 4%, 5.8% ± 4.9%, and 6.4% ± 4.9% for peak systolic, peak early diastolic, and peak late diastolic velocities, respectively. The interobserver variation for the above velocities was also low: 3.4% ± 3.5%, 6.1% ± 6%, and 6.5% ± 6%, respectively.

Systolic Myocardial Velocity

In healthy subjects, the peak systolic mitral annular velocity at the septum was significantly lower than that at the lateral and inferior sites (Table 2). Compared with healthy subjects, the MI patients had a significantly reduced peak systolic velocity at all 4 sites of the mitral annulus (Table 2). Good-quality echoes for the measurement of ejection fractions

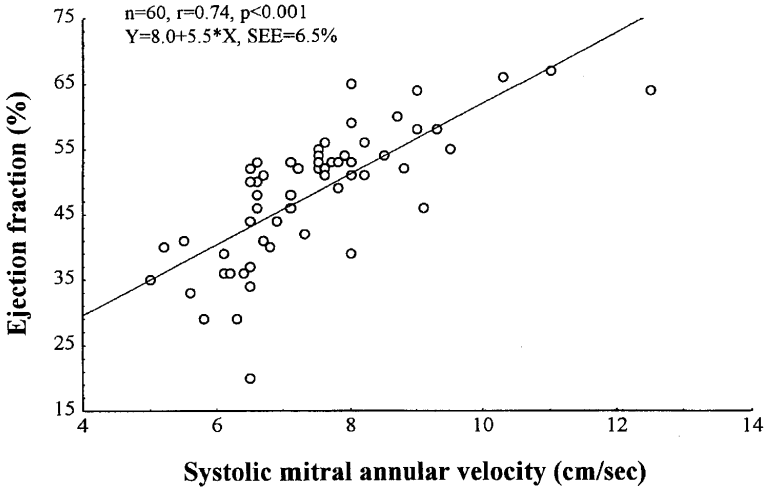

Figure 2 Correlation between left ventricular ejection fraction and the mean systolic mitral annular velocity in patients with myocardial infarction. *SEE,* Standard error of estimate.

Table 1 Basic clinical and echocardiographic parameters

	Age	Heart rate (bpm)	LV (mm)	LA (mm)	MAM (mm)
Healthy subjects (n = 25)	62 ± 6	66 ± 10	46 ± 4	34 ± 3	14.1 ± 2
Subjects with MI (n = 78)	64 ± 11	69 ± 11	51 ± 6*	38 ± 4*	10.2 ± 2†

The results are presented as the mean and SD. *LV,* Left ventricular dimension in end diastole; *LA,* left atrial dimension; *MAM,* mean value of the mitral annular motion; *MI,* myocardial infarction.
*$P < .01$ compared with healthy subjects.
†$P < .001$ compared with healthy subjects.

Table 2 Mitral annular peak systolic velocity *(S)* assessed by Doppler tissue imaging at different sites of the left ventricle

	S-Septum (cm/s)	S-Anterior (cm/s)	S-Lateral (cm/s)	S-Inferior (cm/s)	S-Mean (cm/s)
Healthy subjects (n = 25)	8.8 ± 1.3	9.1 ± 1.6	10.4 ± 1.8†	9.8 ± 1.3*	9.6 ± 1.1
Subjects with anterior MI (n = 33)	6.1 ± 1‡	5.9 ± 1‡	7.6 ± 1.2‡	7.8 ± 1.1‡	6.8 ± 1‡
Subjects with inferior MI (n = 45)	7.2 ± 1.2‡	8.7 ± 1.7	8.7 ± 2‡	6.5 ± 1.2‡	7.8 ± 1.4‡
Total for subjects with MI (n = 78)	6.7 ± 1.4‡	7.5 ± 1.9‡	8.3 ± 2‡	7 ± 1.6‡	7.4 ± 1.3‡

Results are presented as the mean and SD. *S-Mean,* Mean systolic velocity from 4 LV sites; *MI,* myocardial infarction.
*$P < .05$ compared with septum in healthy subjects.
†$P < .001$ compared with septum in healthy subjects.
‡$P < .001$ compared with healthy subjects.

were available in 60 patients (77%) with a mean (SD) value of 48% (9%). A relatively good linear correlation existed between ejection fraction and the mean systolic myocardial velocity from the 4 LV sites (r = 0.74, $P < .001$; Figure 2). Recordings of mitral annular motion and mitral annular velocities by DTI were possible in all the patients. The correlation was also good when the mean peak systolic mitral annular velocity was tested against the magnitude of the mean systolic mitral annular motion (r = 0.77, $P <$ .001; Figure 3). When the patients were divided into two different groups with respect to an ejection fraction ≥0.50 or <0.50, a cutoff point of mean systolic mitral annular velocity of ≥7.5 cm/s had a sensitivity of 79% and a specificity of 88% in predicting preserved global LV systolic function (27 true-positive, 3 false-positive, 23 true-negative, and 7 false-negative results). A previous study has shown that a mean value of mitral annular motion of 10 mm or more has high sensitivity and specificity in detecting patients with an ejection fraction of 0.50 or more.[9] When the patients were divided into 2 different groups, mitral

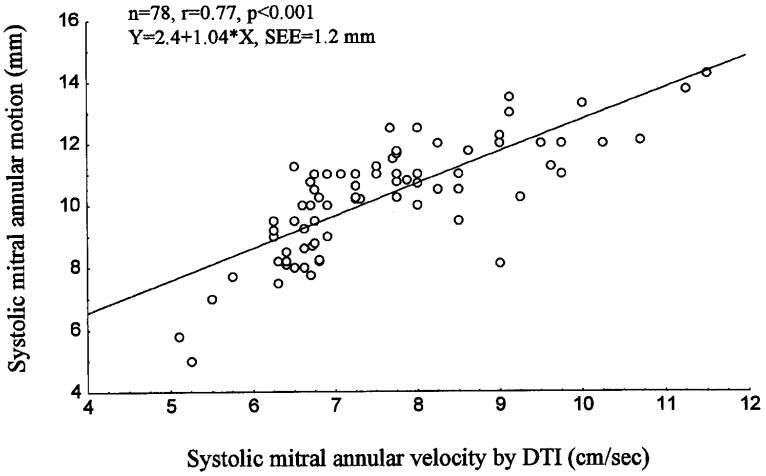

Figure 3 Correlation between the magnitude of the mean mitral annular motion and the mean systolic mitral annular velocity in patients with myocardial infarction. *SEE,* Standard error of estimate; *DTI,* Doppler tissue imaging.

Table 3 Mitral annular early diastolic velocity *(E)* assessed by Doppler tissue imaging at different sites of the left ventricle

	E-Septum (cm/s)	E-Anterior (cm/s)	E-Lateral (cm/s)	E-Inferior (cm/s)	E-Mean (cm/s)
Healthy subjects (n = 25)	10.8 ± 2.3	11.8 ± 3.4	13.2 ± 2.9*	11.5 ± 3	11.8 ± 2.5
Subjects with anterior MI (n = 18)	6.6 ± 1.7§	6.5 ± 1.4§	8.9 ± 2.1§	8.4 ± 1.4§	7.6 ± 1.3§
Subjects with inferior MI (n = 29)	7.5 ± 1.4§	10 ± 2.9†	10 ± 3.4‡	6.7 ± 1.6§	8.5 ± 2.1§

Results are expressed as the mean and SD. *E-Mean,* Mean early diastolic velocity from 4 LV sites; *MI,* myocardial infarction.
*$P < .01$ compared with septum in healthy subjects.
†$P < .05$ compared with healthy subjects.
‡$P < .01$ compared with healthy subjects.
§$P < .001$ compared with healthy subjects.

Table 4 Late diastolic mitral annular velocity *(L)* assessed by Doppler tissue imaging at different sites of the left ventricle

	L-Septum (cm/s)	L-Anterior (cm/s)	L-Lateral (cm/s)	L-Inferior (cm/s)	L-Mean (cm/s)
Healthy subjects (n = 25)	11.9 ± 2.5	11.3 ± 2.4	12.6 ± 3.4	12.6 ± 2.6	12.2 ± 2.3
Subjects with anterior MI (n = 18)	10.1 ± 1.6*	11.3 ± 2.5	10.5 ± 4.3	11.6 ± 2.8	10.9 ± 2.5
Subjects with inferior MI (n = 29)	9.9 ± 2.6†	11.4 ± 2.8	12 ± 3.2	11.2 ± 3	11 ± 2.6

Results are expressed as the mean and SD. *L-Mean,* Mean late diastolic velocity from 4 left ventricular sites; *MI,* myocardial infarction.
*$P < .05$ compared with healthy subjects.
†$P < .01$ compared with healthy subjects.

annular motion ≥10 mm and <10 mm, a cutoff point of the mean systolic mitral annular velocity of ≥7.5 cm/s had a sensitivity of 72% and a specificity of 93% in predicting preserved global LV systolic function (37 true-positive, 2 false-positive, 25 true-negative and 14 false-negative results).

When we divided the MI patients into those with anterior or inferior MIs according to ECG localization of the infarction sites, we found a reduced mitral annular velocity at nearly all sites compared with that in healthy subjects (Table 2 and Figure 4). In patients with anterior MI, the reduced velocity was more pronounced at the anterior wall than at other sites (Figure 5). In inferior MI, the mitral annular velocity at the inferior wall was significantly reduced compared with that at other LV sites (Figure 5).

Diastolic Myocardial Velocity

Two major peak velocities were found during diastole: one during early diastole and the other during late diastole. In healthy subjects, the early diastolic mitral velocity at the septum was significantly lower

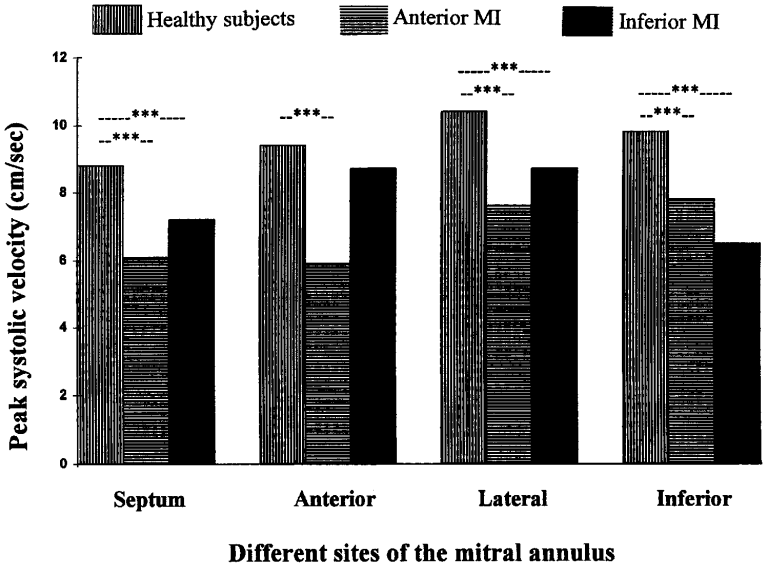

Figure 4 Peak systolic velocity determined by Doppler tissue imaging at different sites of the mitral annulus in healthy subjects and patients with anterior and inferior myocardial infarction *(MI)*. ***$P < .001$.

Table 5 Ratio of the early and late diastolic mitral annular velocities assessed by Doppler tissue imaging at different sites of the left ventricle

	Septum	Anterior	Lateral	Inferior	LV-Mean
Healthy subjects (n = 25)	0.96 ± 0.3	1.1 ± 0.5	1.1 ± 0.5	0.96 ± 0.3	1 ± 0.4
Subjects with anterior MI (n = 18)	0.66 ± 0.1†	0.65 ± 0.1†	0.85 ± 0.3	0.74 ± 0.2*	0.74 ± 0.1†
Subjects with inferior MI (n = 29)	0.76 ± 0.2	0.88 ± 0.2*	0.84 ± 0.3	0.6 ± 0.2‡	0.77 ± 0.2*

Results are expressed as the mean and SD. *LV-Mean,* Mean value of the ratio of early-to-late diastolic mitral annular velocity from 4 left ventricular sites; *MI,* myocardial infarction.
*$P < .05$ compared with healthy subjects.
†$P < .01$ compared with healthy subjects.
‡P < .001 compared with healthy subjects.

than at the lateral wall (Table 3). The early diastolic velocity was lower in both anterior and inferior MIs compared with that in healthy subjects at all sites (Table 3). Similar to systolic velocities, the reduced early diastolic velocity was more pronounced at anteroseptal sites in anterior MI and at inferior sites in inferior MI (Figure 6). The late diastolic mitral annular velocity was similar to that in healthy subjects at almost all sites (Table 4). However, compared with that in healthy subjects, the ratio between the early and late diastolic mitral annular velocity was reduced, especially at the infarction sites (Table 5).

The conventional Doppler parameters in patients and healthy subjects are given in Table 6. The peak mitral annular early diastolic velocity correlated well with both the LV ejection fraction ($r = 0.66, P < .001$, n = 47) and the mean mitral annular motion at systole ($r = 0.68, P < .001$, n = 47). However, there was no correlation between the early diastolic velocity and transmitral E wave, transmitral A wave, E/A ratio, transmitral E-wave deceleration time, IVRT, or different pulmonary venous waves (r values between 0.1 and 0.28; P value was not significant). The ratio between mitral annular early and late velocity showed no correlation with any of the transmitral flow velocities or IVRT.

DISCUSSION

Left ventricular contraction involves both a reduction of the short-axis diameter and a shortening along the longitudinal axis of the chamber.[18,19] Previously, the amplitude of the LV shortening along its long axis has been used to assess both regional and global LV systolic function.[8-10,20] The longitudinal systolic shortening of the left ventricle is reflected by the motion of the mitral annulus toward the car-

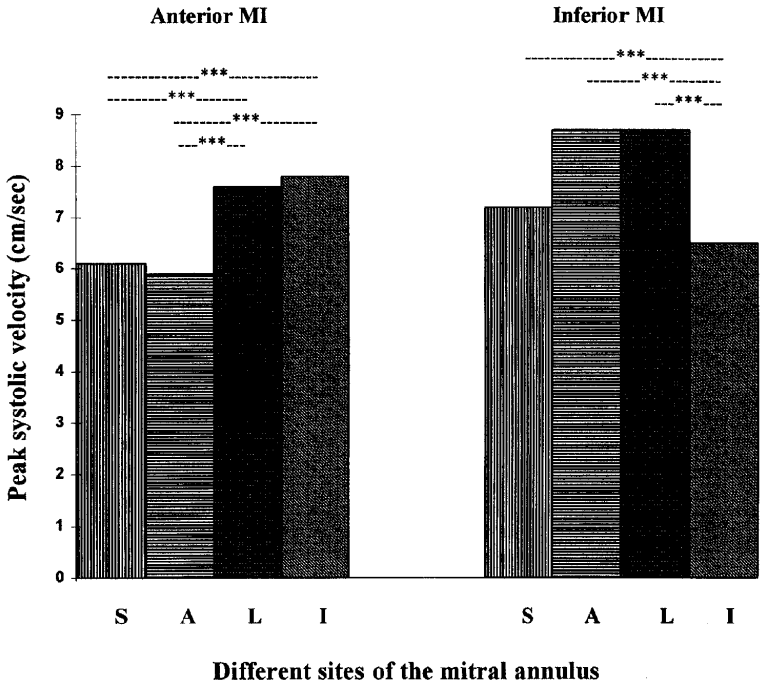

Figure 5 Peak systolic mitral annular velocity at different sites of the left ventricle in patients with anterior and inferior myocardial infarction *(MI). S,* Septum; *A,* anterior; *L,* lateral; *I,* inferior. ***$P < .001$.

diac apex in systole, whereas its recoil away from the apex is the result of diastole. As there is no appreciable motion of the apex in relation to the imaging transducer, the magnitude of the mitral annular motion reflects the extent of myocardial shortening along its longitudinal axis.[9,19,20] Recording the mitral annular motion has the advantage that it is devoid of trabeculae, myocardial dropouts, etc, and therefore is independent of echo quality. Further analysis by quantifying the myocardial velocities with the use of DTI opens up a new possibility of assessing LV function along the long axis. The major advantage of pulsed wave mitral annular velocity measurements is that the ultrasound beam is parallel to the LV contraction. Moreover, it probably measures the transmural myocardial velocity, not only the epicardial or endocardial velocities. Similar to measurements of the amplitude of mitral annular motion by conventional echocardiography, the recording of LV velocity by pulsed wave DTI at different mitral annular sites is easy. We were able to record it at all the LV sites in all the patients. In addition, the method is highly reproducible with low interobserver and intraobserver variabilities.

In the assessment of LV systolic function, the concept of global and regional function assessed by conventional echocardiography is an established factor. On the other hand, assessment of LV diastolic function has been confined to the global level with the use of different Doppler parameters.[15-17] Up to now, there has been no standard method for the assessment of regional diastolic function. Introduction of pulsed wave DTI at different mitral annular sites of the left ventricle opens up a new possibility. Thus, the pulsed wave DTI parameters may be used as an additional method that could increase the accuracy of echocardiographic LV studies.

Table 6 Conventional Doppler parameters

	Subjects with myocardial infarction (n = 47)	Healthy subjects (n = 25)
Transmitral flow		
E-wave (m/s)	0.67 ± 0.2	0.68 ± 0.1
A-wave (m/s)	0.68 ± 0.2	0.66 ± 0.1
E/A ratio	0.98 ± 0.4	1.05 ± 0.3
E-wave deceleration (ms)	198 ± 54	199 ± 30
IVRT (ms)	90 ± 22	86 ± 10
Pulmonary venous waves		
Systolic (m/s)	0.57 ± 0.2	0.59 ± 0.1
Diastolic (m/s)	0.51 ± 0.2	0.46 ± 0.1
Systolic/diastolic	1.16 ± 0.4	1.3 ± 0.2
Atrial systole (m/s)	0.31 ± 0.1	0.29 ± 0.1

Results are expressed as the mean and SD. *IVRT,* Isovolumic relaxation time.

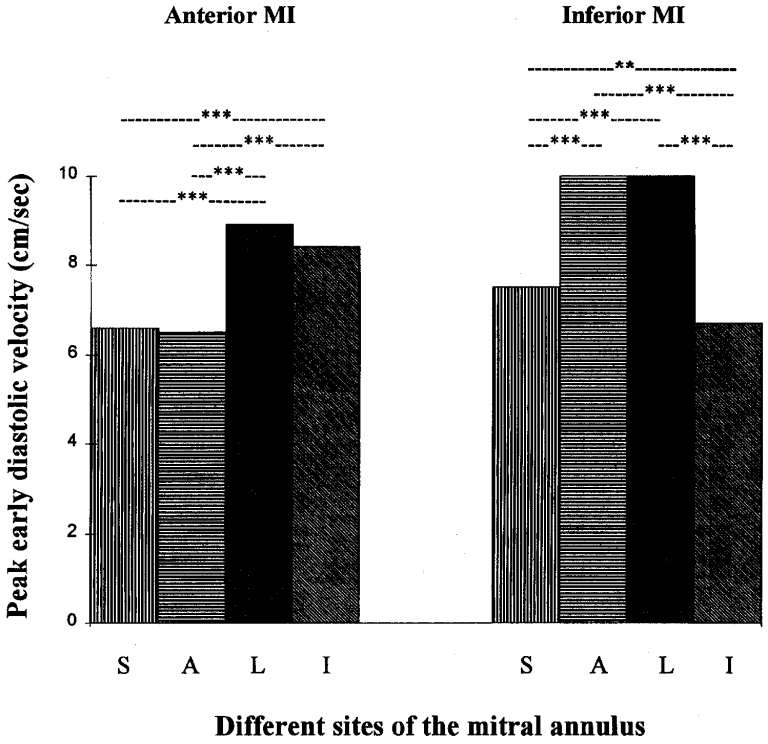

Figure 6 Peak early diastolic velocity at different sites on the mitral annulus in patients with anterior and inferior myocardial infarction *(MI)*. *S,* Septum; *A,* anterior; *L,* lateral; *I,* inferior.
***P* < .01.
****P* < .001.

Systolic LV Function by DTI in MI

In the present study, after a first MI, a significant regional difference was found in the peak systolic velocity at the mitral annulus related to the infarction site. Anterior MI patients had lower velocities at the anterior wall and septum than at other sites. The reduced velocity at the infarction site is an expression of myocardial damage after an MI. In inferior MI, the systolic velocity was not only reduced at the inferior site of the mitral annulus, there was also a significantly lower peak systolic velocity at the interventricular septum compared with the septal velocity in healthy subjects and to lateral and anterior sites within the same group of subjects with inferior MIs. This may be the result of partial damage to the septum in inferior MIs, as the posterior part of the septum is usually supplied by the right coronary artery.[21] In addition to the reduced systolic velocity at the infarction sites in both anterior and inferior MI, the systolic velocity at the noninfarction sites was also reduced compared with that in healthy subjects. It is difficult to explain this phenomenon. We do not know whether the spirally oriented myocardial fiber, which causes the shortening of the left ventricle along its long axis,[22] might have some action beyond the infarction area.

Although the myocardial velocity and ejection fraction are two different measurements, our study demonstrates a relatively good and highly significant correlation between the mean peak systolic velocity of the mitral annulus and the LV ejection fraction. This indicates that systolic velocity plays an important role in the pumping function of the left ventricle. In addition, a mean peak systolic velocity of ≥7.5 cm/s predicts a preserved LV ejection fraction (≥0.50) with relatively high sensitivity and specificity. In some previous studies using either pulsed wave DTI at lateral sites of the mitral annulus[7] or color M-mode velocity at 6 different sites on the mitral annulus,[23] a significant correlation was found between the mean peak systolic velocity and ejection fraction. However, in these studies the patients had mixed diagnoses.

Diastolic LV Function by DTI in MI

The assessment of diastolic function has clinical significance.[24,25] Although systolic abnormalities are the most common cause of congestive heart failure,

the presence of diastolic filling abnormalities may lead to inadequate cardiac output even though the ejection fraction is normal or near-normal.[26] Left ventricular diastolic dysfunction may occur after MI.[24,25] Other associated diseases (eg, systemic hypertension, valvular heart disease, cardiomyopathy, diabetes mellitus) may also influence diastolic function. To assess the effects of a first MI on the myocardial diastolic velocities, we included only patients without a history of previous disease. The peak early diastolic velocity of the left ventricle along its long axis was significantly decreased in both anterior and inferior MI compared with that in healthy subjects. Similar to the systolic velocity, the decrease in the early diastolic velocity was more pronounced at the infarction sites (ie, at the anterior site in anterior MI and at the inferior site in inferior MI). The ratio of early-to-late diastolic velocity was significantly lower, especially at the infarction sites, compared with that in healthy subjects. This is the result of a significant decrease in the early diastolic velocities in this patient population. In a previous study with old MIs or dilated cardiomyopathy and with LV dysfunction, a decreased peak early diastolic velocity was noted with the use of color-coded DTI.[5] In another study, the early diastolic velocity could also be induced during balloon occlusion at percutaneous transluminal coronary angioplasty.[13]

In our study, the peak early diastolic mitral annular velocity correlated well with the LV ejection fraction. It probably reflects the circumstance that the diastolic dysfunction is related to the systolic dysfunction after MI. However, the early diastolic mitral annular velocity did not correlate with any of the conventional Doppler diastolic parameters. The transmitral flow velocities and the IVRT are dependent on LV relaxation and on left atrial pressure.[27,28] After MI, patients may develop different degrees of diastolic dysfunction. With conventional Doppler parameters, these changes may be detected by the development of abnormal LV relaxation, pseudonormalization, or a restrictive pattern, depending on the extent of the myocardial damage. Therefore conventional Doppler parameters may fail to identify diastolic dysfunction after MI. However, the early diastolic mitral annular velocity measured by DTI has been postulated to be independent of the filling pressure.[7,29] This may be the reason for the good correlation between the early diastolic velocity determined by DTI and the LV ejection fraction, but not with the conventional Doppler parameters, in the present study. Thus, by using the mitral annular peak early diastolic velocity, it may be possible to assess the diastolic dysfunction after MI, even in the presence of pseudonormalization of the transmitral flow velocity or with an elevated LV end-diastolic pressure.

Limitation

This study analyzes the possibilities of assessing LV function by using the myocardial velocity along the long axis. No consideration was taken of the contraction of the left ventricle along its short axis caused by circumferential fiber. For this study, we divided the MI patients into anterior and inferior groups. Anterior MI with infarction only near the apex may result in a different velocity at the annulus compared with an infarction near the annulus and vice versa. Another limitation is that we used LV ejection fraction determined by echocardiography as the reference method. No isotope studies were performed to assess LV function. However, on comparing the peak systolic mitral annular velocity with LV ejection fraction, only the high-quality recordings were accepted. We also compared the peak systolic mitral annular velocity with the mean value of the mitral annular motion assessed by M-mode echocardiography. Previous studies have shown a good correlation between the LV ejection fraction determined by radionuclide angiography and the mean mitral annular motion recorded by M-mode echocardiography.[9,30] Another limitation is that coronary and LV angiography were not performed during the acute phase of the infarction to localize and evaluate the extent of LV wall asynergy. However, the infarction sites were determined by both ECG and 2-dimensional echocardiography.

Conclusion

Mitral annular velocities can readily be recorded by pulsed wave DTI after MI. The reduced peak systolic velocity seems to be an expression of regionally reduced systolic function. The mean peak systolic velocity from 4 LV sites correlated well with the LV ejection fraction. The peak early diastolic velocity is also reduced, especially at the infarction sites, and reflects regional diastolic dysfunction. Thus, quantification of the myocardial velocity by DTI opens up a new possibility of assessing LV function in patients with MI.

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