Relation of Transmitral Flow Velocity Patterns to Left Ventricular Diastolic Function: New Insights From a Combined Hemodynamic and Doppler Echocardiographic Study

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In an effort to determine what clinically useful information regarding left ventricular diastolic function can be inferred noninvasively with pulsed wave Doppler echocardiography, mitral flow velocity patterns and measured variables were correlated with hemodynamic findings in 70 patients: 30 with coronary artery disease, 20 with idiopathic congestive cardiomyopathy, 14 with a restrictive myocardial process and 6 without significant cardiac disease. The effect of sudden changes in hemodynamics on the mitral flow velocity pattern was also investigated in a subgroup of patients who had simultaneous recording of mitral flow velocity and left ventricular pressure before and after left ventriculography. Mitral flow velocity recordings from 30 healthy adults served as a reference group.

This analysis suggests that 1) the majority of patients with these cardiac disorders demonstrate abnormal mitral flow velocity patterns or variables; 2) markedly different flow velocity patterns can be seen in patients with impaired left ventricular relaxation; 3) the different mitral patterns appear to relate more to myocardial function and hemodynamic status than to the type of disease process present; 4) certain mitral patterns suggest different filling pressures and rates of early diastolic left ventricular filling; 5) an increase in left atrial pressure can “normalize” an abnormal mitral flow velocity pattern and “mask” a left ventricular relaxation abnormality; and 6) the different patterns appear to represent a dynamic continuum with the potential to change from one to another as a result of disease progression, medical therapy or sudden changes in hemodynamics.

It is concluded that, despite the indirect method of estimation and certain limitations, mitral flow velocity recordings have clinical potential in assessing left ventricular diastolic function that merits further investigation.

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Left ventricular diastolic dysfunction is now recognized as a significant cause of cardiac symptoms even in patients with apparently normal systolic ventricular function (1-3). From left ventricular pressure recordings maximal rate of fall of pressure (−dP/dt) and the time constant (T) of an assumed exponential decline in pressure have been used to quantitate the rate of left ventricular relaxation (1,2,4-6). Because these indexes require cardiac catheterization, several noninvasive methods for indirectly assessing left ventricular diastolic function have been evaluated. These have included measurement of the left ventricular isovolumic relaxation interval (7-9), the peak rate of diastolic left ventricular wall thinning or rate of change in dimensions from M-mode echocardiograms (7,10,11) and rates of diastolic filling from contrast (4,12,13) or radionuclide angiography (9,14-16). However, only a moderate correlation between invasive and noninvasive variables of diastolic function has been reported in studies in which both have been performed (4,15).

More recently, several investigators (9,13,16-24) have attempted to indirectly assess left ventricular diastolic function by using different measurements obtained from pulsed wave Doppler ultrasound mitral flow velocity recordings. In normal subjects and patients with a variety of cardiac diseases, evaluation of left ventricular filling patterns by Doppler ultrasound has compared favorably with both angiographic (13) and radionuclide (9,16) techniques. However, because left ventricular filling rate and the mitral flow velocity pattern can be affected by factors in addition to the rate of left ventricular relaxation (25-28), caution has been advised in equating variables derived from transmitral flow...
The measurement of peak mitral flow velocity in early diastole (M1) and at atrial contraction (M2), the mitral deceleration time (DT) and the isovolumic relaxation time (IVRT) are labeled. The left ventricular pressure is recorded with a high fidelity fiberoptic catheter and the pulmonary wedge pressure with a fluid-filled catheter. The baseline for the two pressures differs as shown, but the scale is the same. Note that M1 occurs near the time of left ventricular minimal pressure, and a marked increase in diastolic pressure is seen to start simultaneously with the increase in mitral flow velocity with atrial contraction.

Velocity with specific, noninvasive or invasive variables, associated with abnormal left ventricular diastolic function (29).

In an effort to determine what clinically useful information regarding left ventricular diastolic function can be inferred noninvasively with Doppler echocardiography, this study was designed to correlate mitral flow velocity patterns and measured variables with hemodynamic findings in patients with various cardiac diseases. To investigate the precise timing of these relations and their dynamic nature, a subgroup of patients had simultaneous recording of mitral flow velocity and left heart pressures before and after left ventriculography. From these results a framework for the interpretation of mitral flow velocity patterns in the assessment of left ventricular diastolic function is proposed.

**Methods**

**Study Patients.** Mitral flow velocity recordings and hemodynamic findings were analyzed in 70 patients without mitral stenosis who were undergoing cardiac catheterization for clinical indications. Thirty patients had coronary artery disease, 20 had idiopathic congestive cardiomyopathy, 14 had a restrictive myocardial process, 5 were cardiac transplant recipients undergoing routine annual catheterization and 1 had no discernible cardiac abnormality. Simultaneous recording of mitral flow velocity and hemodynamics was performed in 21 patients: 7 with coronary artery disease, 8 with congestive cardiomyopathy, 5 cardiac transplant recipients and 1 normal subject. In 10 of these patients simultaneous recordings were repelled immediately after left ventriculography. In the patients with nonsimultaneous studies, echocardiographic examination was performed within 4 h, and in most cases within 1 h, of cardiac catheterization.

Functional status was defined as follows: class I, asymptomatic; class II, shortness of breath with moderate exertion; class III, shortness of breath with mild exertion; and class IV, shortness of breath with minimal exertion. Four patients had atrial fibrillation, two had an atrial paced rhythm and two had a ventricular paced rhythm. Mitral flow velocity recordings from 30 healthy adult volunteers were used as a control reference group for data comparison. All patients and control subjects gave informed consent to the protocol approved by the Committee for the Protection of Human Subjects at the Stanford University Medical Center.

**Echocardiography.** Two-dimensional and M-mode echocardiograms were obtained with a Hewlett-Packard model 77020A imaging system. The presence of left ventricular enlargement was assessed from parasternal (30) and apical four chamber views (31). Pulsed wave and continuous wave Doppler ultrasound examination was performed with an Irex Exemplar ultrasonograph with a 2.0 MHz transducer. Mitral flow velocity was recorded with pulsed wave technique together with the electrocardiogram (ECG) and phonocardiogram. With use of the apical four chamber view, the sample volume was placed between the tips of the mitral leaflets where the maximal flow velocity in early diastole was obtained. Peak mitral flow velocity in early diastole and at atrial contraction and the deceleration time of mitral flow velocity in early diastole were measured (Fig. 1). When atrial contraction occurred before the mitral deceleration slope had decreased to the zero baseline, the slope was linearly extrapolated to the baseline to obtain the deceleration time. Cardiac cycles with nonlinear deceleration slopes and fusion of early mitral flow velocity and flow velocity at atrial contraction were excluded from analysis. The left ventricular isovolumic relaxation time was measured from aortic valve closure on the phonocardiogram to the start of mitral flow. Recordings were made at a paper speed of 50 mm/s for velocity measurements and 100 mm/s for the isovolumic relaxation time. All values were obtained as a mean of 6 to 8 beats in patients with sinus rhythm and 10 to 15 beats in the patients with atrial fibrillation and ventricular paced rhythm.

For detecting mitral regurgitation the pulsed wave sam-
ple volume was placed at the mitral valve annulus. The maximal velocity of regurgitation was recorded with continuous wave Doppler ultrasound. Mitral regurgitation was graded as mild, moderate or severe using a combination of Doppler ultrasound criteria, which included the intensity of regurgitant signal relative to the antegrade flow signal and extension of the regurgitant jet in the left atrium. Regurgitation graded as moderate or severe was verified by left ventriculography.

In 21 patients the mitral flow velocity was recorded simultaneously with pressures by the Irex echocardiograph or a nonimaging Doppler ultrasound instrument with both pulsed and continuous wave Doppler capabilities (SD-100, Vingmed A/S, 2.0 MHz transducer).

Cardiac catheterization. Left and right heart catheterization were performed with fluid-filled catheters attached to manifold micromanometer transducers (Gould, P-50). The system shows a flat frequency response to 18 Hz and <10% deviation to 24 Hz. In 27 studies high fidelity fiberoptic catheters (Camino Laboratories, model 110-4) were used for left ventricular pressure recording and the calculation of the time constant (T) of decrease of left ventricular isovolumic pressure and the maximal positive and negative derivative of left ventricular pressure (dP/dt). T was calculated by the method of Weiss et al. (6) and dP/dt was calculated by electronic differentiation. T and dP/dt were obtained by sampling left ventricular pressure over 3 s intervals at a rate of 100 samples/s with a Hewlett-Packard 5600 B Cardiac Catheterization Computer System. Data were then resampled at 500 samples/s by Fourier interpolation. The left ventricular diastolic pressure recording was analyzed for minimal pressure in early diastole, height (in mm Hg) of the rapid filling wave, increase in pressure with atrial contraction and left ventricular end-diastolic pressure. Cardiac output was measured by the Fick method. Left ventriculograms were obtained in the 30° right anterior oblique projection in 64 of the 70 patients and coronary angiography was performed in all patients. Reduced left ventricular systolic function was defined as an ejection fraction of <50% or maximal plus positive dP/dt of <1,250 mm Hg/s.

Statistical analysis. Results are expressed as mean values ± 1 SD. Statistical analysis between groups was performed with an analysis of variance. When differences between groups were present, Scheffe's test was used to determine which means differed significantly. To compare changes in flow velocities and hemodynamic variables before and after left ventriculography, a paired Student's t test was used. To compare Doppler variables in a younger and an older age group of normal subjects, a nonpaired Student's t test was used.

Nonparametric Spearman correlation coefficients between all Doppler and hemodynamic variables were computed and tested for statistical significance (p < 0.05 overall) under the null hypothesis that the population correlation is zero. Because individual bivariate correlation coefficients do not take into account the within-group interrelations for the Doppler and hemodynamic group of variables, a multivariate canonical correlation analysis was also performed and standardized canonical correlation coefficients were computed.

Results

Study groups. The patients were classified into groups on the basis of etiology of cardiac disease and hemodynamic findings (Table I). The patients with coronary artery disease were also subclassified into a group with higher (≥16 mm Hg) or lower (<16 mm Hg) mean pulmonary wedge pressure. Because only a small number of patients with congestive cardiomyopathy (3 of 20) or a restrictive myocardial process (1 of 14) had a lower wedge pressure, these groups were not subclassified. Five cardiac transplant recipients and one other subject had no definite cardiac abnormality and were excluded from the intergroup analysis.

Clinical and echocardiographic findings (Table I). The patients with coronary artery disease were older than the normal subjects (p < 0.05). Advanced functional classes (classes III and IV) had a low prevalence in the patients with coronary artery disease who had lower pulmonary wedge pressure (11%) versus the patients in the other groups (27 to 75%). All the patients with a restrictive myocardial process...
had normal left ventricular dimensions; in the other patient
groups, left ventricular enlargement and systolic function
were variable. Mild or moderate mitral regurgitation was
prevalent in all patient groups, although severe mitral regur-
gitation was present in only five patients: three with coro-

nary artery disease who had a high pulmonary wedge pres-

sure and two with a congestive cardiomyopathy.

Table 2 shows the heart rate and Doppler mitral flow

variables in normal subjects and the patients in the study
groups. The mean heart rate was faster in the patients with
coronary artery disease who had high pulmonary wedge

pressure and in patients who had congestive cardiomyo-
apathy and a restrictive myocardial process than in the normal
subjects. The left ventricular isovolumic relaxation time
was longest in the patients with coronary artery disease who
had lower pulmonary wedge pressure and shortest in the
patients with a restrictive myocardial process. Peak mitral
flow velocity in early diastole was decreased in the patients
with coronary artery disease who had lower pulmonary

wedge pressure and smallest in the patients with coronary

artery disease who had a high pulmonary wedge pressure.

Mitral flow velocity variables in normal subjects: influence

of age. Because mitral flow velocity variables have been

reported to change with age, we classified the normal sub-

jects into a younger (mean age 38 ± 6 years) and older age

(mean age 55 ± 4 years) group of 15 individuals each and

compared all variables. This analysis revealed no statistical
difference in any of the Doppler variables between the

groups.

Hemodynamic findings (Table 3). The patients with coro-

nary artery disease who had lower pulmonary wedge pres-

sure had the most normal hemodynamic status whereas that

of the other patient groups was markedly abnormal. In the

patients with a restrictive myocardial process there was near

equalization of diastolic pressures. Left ventricular pressure

ingcrease with atrial contraction and ejection fraction were

statistically similar in all groups of patients, although the

lowest individual values in ejection fraction were seen in the

patients with congestive cardiomyopathy.

In the 27 patients who had high fidelity left ventricular

pressure recordings, mean values for T and maximal nega-
tive dP/dt were 55 ± 14 ms and 1,620 ± 305 mm Hg/s,

respectively, in the patients with coronary artery disease

who had lower pulmonary wedge pressure, 77 ± 24 ms and

1,018 ± 358 mm Hg/s in the patients with coronary artery
disease who had higher pulmonary wedge pressure and 67 ±

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>HR (beats/min)</th>
<th>IVRT (ms)</th>
<th>M1 (cm/s)</th>
<th>M2 (cm/s)</th>
<th>M1/M2</th>
<th>M3 (ms)</th>
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<tr>
<td>Normal</td>
<td>30</td>
<td>63 ± 11</td>
<td>71 ± 14</td>
<td>83 ± 16</td>
<td>60 ± 16</td>
<td>1.5 ± 0.4</td>
<td>93 ± 23</td>
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<td>CAD total</td>
<td>30</td>
<td>78 ± 12</td>
<td>82 ± 30</td>
<td>81 ± 30</td>
<td>67 ± 30</td>
<td>1.6 ± 1.4</td>
<td>197 ± 73</td>
</tr>
<tr>
<td>CAD I</td>
<td>18</td>
<td>74 ± 12</td>
<td>100 ± 27</td>
<td>80 ± 17</td>
<td>60 ± 17</td>
<td>0.9 ± 0.4</td>
<td>232 ± 58</td>
</tr>
<tr>
<td>CAD II</td>
<td>12</td>
<td>83 ± 13</td>
<td>90 ± 27</td>
<td>60 ± 20</td>
<td>20 ± 19</td>
<td>2.6 ± 1.4</td>
<td>181 ± 67</td>
</tr>
<tr>
<td>CCM</td>
<td>20</td>
<td>91 ± 16</td>
<td>76 ± 25</td>
<td>90 ± 26</td>
<td>60 ± 15</td>
<td>1.3 ± 0.9</td>
<td>130 ± 52*</td>
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<tr>
<td>RMP</td>
<td>14</td>
<td>90 ± 13</td>
<td>90 ± 27</td>
<td>82 ± 20</td>
<td>60 ± 15</td>
<td>2.3 ± 0.8</td>
<td>128 ± 33*</td>
</tr>
</tbody>
</table>

*p < 0.05 vs. normal; t p < 0.05 vs. CAD I; 1p < 0.05 vs. CAD. HR = heart rate; IVRT = left ventricular isovolumic relaxation time; M1 = peak mitral flow velocity in early diastole; M2 = peak mitral flow after atrial systole; M3 = mitral deceleration time; other abbreviations as in Table 1.

Table 3. Hemodynamic Findings in the Patient Groups (n = 64)

<table>
<thead>
<tr>
<th>Group</th>
<th>RAP Mean (mm Hg)</th>
<th>PAP Mean (mm Hg)</th>
<th>PWP Mean (mm Hg)</th>
<th>PWV Wave LVmax (mm Hg)</th>
<th>LVSP (mm Hg)</th>
<th>LVmin (mm Hg)</th>
<th>LVEDP (mm Hg)</th>
<th>EF (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAD total</td>
<td>8 ± 8</td>
<td>23 ± 15</td>
<td>15 ± 12</td>
<td>17 ± 12</td>
<td>13 ± 12</td>
<td>124 ± 19</td>
<td>5 ± 7</td>
<td>8 ± 5</td>
</tr>
<tr>
<td>CAD I</td>
<td>3 ± 3</td>
<td>15 ± 6</td>
<td>9 ± 5</td>
<td>11 ± 6</td>
<td>8 ± 6</td>
<td>127 ± 20</td>
<td>3 ± 4</td>
<td>8 ± 5</td>
</tr>
<tr>
<td>CAD II</td>
<td>15 ± 7*</td>
<td>39 ± 11*</td>
<td>26 ± 8*</td>
<td>36 ± 12*</td>
<td>24 ± 9*</td>
<td>140 ± 17*</td>
<td>11 ± 8*</td>
<td>7 ± 5</td>
</tr>
<tr>
<td>CCM</td>
<td>7 ± 7</td>
<td>29 ± 11*</td>
<td>20 ± 7*</td>
<td>25 ± 12*</td>
<td>18 ± 12</td>
<td>100 ± 17*</td>
<td>8 ± 6</td>
<td>7 ± 4</td>
</tr>
<tr>
<td>RMP</td>
<td>16 ± 4*</td>
<td>30 ± 12*</td>
<td>21 ± 51</td>
<td>27 ± 9*</td>
<td>17 ± 7</td>
<td>108 ± 18</td>
<td>8 ± 5</td>
<td>10 ± 5*</td>
</tr>
</tbody>
</table>

*p < 0.05 vs. CAD I; 1p < 0.05 vs. CAD II; 1p < 0.05 vs. CAD. EF = ejection fraction; LVmax = change in left ventricular pressure with atrial contraction; LVmin = left ventricular diastolic pressure minimum; LVEDP = left ventricular end-diastolic pressure; LVEDP = left ventricular rapid filling wave; LVSP = left ventricular peak systolic pressure; PAP = pulmonary artery pressure; PWV = pulmonary wedge pressure; PWV = PWV - LVmin = pulmonary wedge V wave - left ventricular pressure minimum difference; RAP = right atrial pressure; other abbreviations as in Table 1.
26 ms and 1,252 ± 384 mm Hg/s in the patients with congestive cardiomyopathy.

Normal ranges for mitral flow velocity variables, left ventricular diastolic pressures and T (Table 2). A normal range for the Doppler ultrasound variables was obtained from the mitral flow velocities recorded in the normal subjects: left ventricular isovolumic relaxation time of 55 to 90 ms, peak mitral flow velocity in early diastole of 60 to 112 cm/s, mitral flow velocity at atrial contraction of 40 to 100 cm/s, ratio of peak mitral flow velocity in early diastole to that at atrial contraction of 1.0 to 2.5 and a mitral deceleration time of 120 to 240 ms. Although the normal subjects in this study did not undergo cardiac catheterization, normal values for T of 22 to 46 ms (mean 35 ± 6) have been obtained in healthy adults with use of high fidelity catheters and identical methods (1,32). Values for left ventricular minimal pressure of 1.9 ± 1.7 mm Hg and for the left ventricular rapid filling wave of 1.6 ± 1.5 mm Hg have also been reported (32) in normal subjects.

Relation among mitral flow velocity patterns, hemodynamic events and results in the patient groups. Figure 1 shows the temporal relations between left ventricular diastolic pressure and mitral flow velocity in a simultaneous recording obtained with a high fidelity pressure catheter. Peak mitral flow velocity in early diastole corresponds closely in time to the left ventricular pressure minimum, and the start of mitral flow velocity deceleration corresponds to the onset of diastolic left ventricular pressure increase (the rapid filling wave). In Figure 2 a simultaneous recording of high fidelity left ventricular pressure and fluid-filled left atrial pressure shows that the onset of mitral flow corresponds closely in time to the left atrial-left ventricular pressure crossover. Both pressures decrease rapidly after mitral valve opening with a relatively short duration of transvalvular pressure difference. In mid-diastole the two pressures appear to be equal, increasing slowly together, and then both increase abruptly with atrial contraction. In the mitral flow velocity recording obtained simultaneously, forward flow throughout diastole is observed, including the period in mid-diastole when the two pressures appear equal.

The variation in mitral flow velocity patterns among the patients with coronary artery disease and the relation to hemodynamic findings are illustrated in Figure 3. In panels A and B, two patients with coronary artery disease who both had a mean pulmonary wedge pressure of 8 mm Hg are shown to have a prolonged T, suggesting a reduced rate of left ventricular relaxation. In both patients the left ventricular isovolumic relaxation and mitral deceleration time are increased and mitral flow velocity in early diastole and at atrial contraction are approximately equal. Although the left ventricular pressure minimum and mitral flow velocity at atrial contraction are similar in the two patients, the increase in left ventricular pressure with atrial contraction is markedly different. In panel C, a patient with coronary artery disease and a mean pulmonary wedge pressure of 35 mm Hg has a longer T than the patients in panels A and B, suggesting a more marked ventricular relaxation abnormality. However, the isovolumic relaxation time is shorter, mitral flow velocity in early diastole is much larger than at atrial contraction, the mitral deceleration time is short and there is an abnormally large left ventricular rapid filling wave and a minimal increase in left ventricular pressure after atrial contraction.

A similar variation in patterns was seen among patients with congestive cardiomyopathy (Fig. 4). In the three patients in the group with lower filling pressures, a normal or mildly prolonged isovolumic relaxation and deceleration time and a normal or near normal ratio of mitral flow velocity in early diastole to that at atrial contraction was seen as illustrated by the patient shown in panel A. In contrast, despite a prolongation of T, patients with a higher pulmonary wedge pressure usually had a short isovolumic relaxation time, an increase in the ratio of mitral flow velocity in early diastole to that at atrial contraction, a short mitral deceleration time and an increase in the left ventricular rapid filling wave (Fig. 4B).

The patients with a restrictive myocardial process showed less variation in mitral flow velocity patterns than...
Figure 3. Mitral flow velocity and high fidelity left ventricular (LV) pressure recordings in three patients with coronary artery disease. In Patients A and B, mitral flow velocity in early diastole is approximately the same as that at atrial contraction. The left ventricular isovolumic relaxation time (IVRT) and mitral deceleration times (DT) are prolonged compared with normal values and the rate of left ventricular relaxation is abnormally slow as assessed by T. In Patient A, left ventricular diastolic pressure continues to decrease during the early filling phase until atrial contraction occurs. In both patients most of the increase in diastolic pressure is with atrial contraction. This diastolic pressure increase is of normal amplitude in Patient A (3 mm Hg) but is increased in Patient B (8 mm Hg), despite similar flow velocities with atrial contraction and seemingly more abnormal relaxation in A. Although Patient C has the most abnormal value for T, mitral flow velocity in early diastole is much larger than flow velocity at atrial contraction, the isovolumic relaxation time is normal and the deceleration time is markedly shortened. In this patient most of the diastolic pressure rise occurs with the left ventricular rapid filling wave, and the low mitral flow velocity at atrial contraction (upper arrow) is associated with only a minimal further increase in left ventricular pressure (lower arrow).

Mitrval Flow Velocity and Hemodynamic Variables

The bivariate correlation coefficients between all Doppler and selected hemodynamic variables are shown in Table 4.

Isovolumic relaxation time. There was an inverse relation between left ventricular isovolumic relaxation time and mean pulmonary wedge pressure ($r = -0.62$), left ventricular rapid filling wave ($r = -0.64$) and left ventricular end-diastolic pressure ($r = -0.42$).

Peak mitral flow velocity in early diastole. There was a positive correlation between peak early mitral flow velocity and the pulmonary wedge V wave - left ventricular pressure minimum difference ($r = 0.59$) (Fig. 5A). Peak early mitral flow velocity also showed a positive correlation with mean pulmonary wedge pressure ($r = 0.50$) and the left ventricular rapid filling wave ($r = 0.43$). There was no correlation between peak early mitral flow velocity and cardiac output ($r = -0.20$) (Fig. 5B). Exclusion of the patients with severe mitral regurgitation did not improve this correlation.

Mitrval deceleration time. Mitral deceleration time showed an inverse relation to the size of the left ventricular rapid filling wave ($r = -0.72$). The majority of patients with a deceleration time <150 ms had an abnormal rapid filling wave >3 mm Hg (32) (Fig. 6A). The deceleration time was also inversely related to mean pulmonary wedge pressure ($r = -0.61$) and left ventricular end-diastolic pressure ($r = -0.48$).

Mitrval flow velocity at atrial contraction. Mitral flow velocity with atrial contraction was inversely correlated with mean pulmonary wedge pressure ($r = -0.57$), the left ventricular rapid filling wave ($r = -0.49$), left ventricular pressure minimum ($r = -0.38$) and left ventricular end-diastolic pressure ($r = -0.43$). There was no correlation between flow velocity and the increase in left ventricular pressure during atrial contraction. A large mitral flow velocity with atrial contraction could be associated with a small increase in left ventricular pressure; conversely, a large increase in pressure could be seen even with a low velocity at atrial contraction (Fig. 6B).

Multivariate correlation coefficients. In general, the results of the multivariate analysis confirmed the observed bivariate relations. By this analysis, the most highly correlated group of variables was the mitral deceleration time (0.60), which was inversely correlated with mean pulmonary
Changes in individual patients with angiotensin. In 10 patients mitral flow velocity recordings and hemodynamic measurements were repeated immediately after left ventriculography. Heart rate increased from 71 ± 18 to 76 ± 17 beats/min, the isovolumic relaxation time decreased from 75 ± 11 to 64 ± 12 ms and peak mitral flow velocity in early diastole increased from 73 ± 13 to 87 ± 24 cm/s after ventriculography (all p < 0.05). Mitral deceleration time and mitral flow velocity at atrial contraction were unchanged. The pulmonary wedge V wave increased from 10 ± 3 to 18 ± 5 mm Hg, the left ventricular rapid filling wave increased from 4 ± 3 to 6 ± 5 mm Hg, the left ventricular pressure rise with atrial contraction increased from 5 ± 3 to 8 ± 5 mm Hg and the left ventricular end-diastolic pressure increased from 13 ± 5 to 19 ± 6 mm Hg after ventriculography (all p < 0.05) whereas peak left ventricular systolic pressure remained unchanged.

Relation between normal and abnormal left ventricular end-diastolic pressure and mitral flow velocity variables. Left ventricular end-diastolic pressure was within normal limits (≤12 mm Hg) in 15 patients and was increased in 49 patients in the study groups. In only 4 of the 15 patients with normal end-diastolic pressure were all the Doppler variables normal. Abnormal findings in the other 11 patients included a prolonged isovolumic relaxation time in 8, increased deceleration time in 4, decreased peak early mitral flow velocity in 4 and decreased ratio of early mitral flow velocity to that at atrial contraction in 8. In 1 of the 11 patients a low peak early mitral flow velocity was the only abnormal finding; two or more variables were abnormal in the other 10. In the 49 patients with increased left ventricular end-diastolic pressure the most frequent abnormality was a shortened mitral deceleration time. In 5 of the 49 patients all mitral flow velocity variables were normal, whereas in 32 patients two or more variables were abnormal. Of the 12 patients with only one abnormal mitral variable, 9 showed a short deceleration time, 1 a short isovolumic relaxation time and 2 a prolonged isovolumic relaxation time.

Discussion

In this study we observed marked variability in the mitral flow velocity recordings within each patient group. Therefore, the relation among mitral flow velocity patterns and the hemodynamic findings will be considered across patient groups, as the different patterns observed appeared to be

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**Table 4. Bivariate Correlation Coefficients Between Doppler and Selected Hemodynamic Variables in 64 Patients**

<table>
<thead>
<tr>
<th>Variable</th>
<th>HR (beats/min)</th>
<th>PWP</th>
<th>LVSP (mm Hg)</th>
<th>LVMin (mm Hg)</th>
<th>LVRFW (mm Hg)</th>
<th>LVd (mm Hg)</th>
<th>LVEDP (mm Hg)</th>
<th>CO (liters/min)</th>
<th>EF (%)</th>
<th>dP/dt (mm Hg/s)</th>
<th>T1 (ms)</th>
</tr>
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<tbody>
<tr>
<td>IVRT (ms)</td>
<td>-0.33</td>
<td>-0.62*</td>
<td>-0.37</td>
<td>-0.23</td>
<td>-0.64*</td>
<td>0.16</td>
<td>-0.42*</td>
<td>0.25</td>
<td>0.04</td>
<td>0.42</td>
<td>0.04</td>
</tr>
<tr>
<td>MV1 (cm/s)</td>
<td>0.19</td>
<td>0.50*</td>
<td>-0.07</td>
<td>0.19</td>
<td>0.43*</td>
<td>-0.27</td>
<td>0.31</td>
<td>-0.20</td>
<td>-0.25</td>
<td>0.27</td>
<td>-0.37</td>
</tr>
<tr>
<td>MV2 (cm/s)</td>
<td>-0.2[1]</td>
<td>-0.57*</td>
<td>0.44*</td>
<td>-0.38*</td>
<td>-0.49*</td>
<td>-0.02</td>
<td>-0.45*</td>
<td>0.16</td>
<td>0.25</td>
<td>0.65</td>
<td>-0.13</td>
</tr>
<tr>
<td>MV1: MV2</td>
<td>0.30</td>
<td>0.70*</td>
<td>-0.40*</td>
<td>0.37</td>
<td>0.58*</td>
<td>-0.12</td>
<td>0.47*</td>
<td>-0.25</td>
<td>-0.32</td>
<td>-0.54</td>
<td>0.02</td>
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<tr>
<td>MV1: ts</td>
<td>-0.45*</td>
<td>-0.61*</td>
<td>0.61*</td>
<td>-0.27</td>
<td>-0.72*</td>
<td>0.03</td>
<td>-0.48*</td>
<td>0.26</td>
<td>0.46</td>
<td>0.81*</td>
<td>-0.26</td>
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*p < 0.05; n = 24 studies only. CO = cardiac output; dP/dt = maximum rate of fall of left ventricular pressure with respect to time; T1 = time constant of left ventricular isovolumic pressure fall; other abbreviations as in Tables 2 and 3.
Figure 5. Panel A shows the relation between peak mitral flow velocity in early diastole (M1) and pulmonary wedge V wave - left ventricular diastolic pressure minimum difference (PWV - LVmin). In panel B the lack of correlation between M1 and cardiac output (CO) is seen.

related more to myocardial function and hemodynamic status than to the type of disease process present.

Mitral flow velocity patterns. The majority of patients in this study could be separated into two groups that showed distinctly different mitral flow velocity patterns compared with normal subjects. **Pattern I:** Patients with this pattern had one or more of the following findings: a prolonged left ventricular isovolumic relaxation time, a decreased peak early mitral flow velocity but normal or increased mitral flow velocity at atrial contraction, a reduced ratio of peak early mitral flow velocity to that at atrial contraction and a prolonged mitral deceleration time. **Pattern II:** In this group of patients who tended to be more symptomatic and have higher filling pressures, one or more of the following findings were present: a short left ventricular isovolumic relaxation time, a normal or increased peak mitral flow velocity in early diastole, a normal or decreased mitral velocity at atrial contraction, an increase in the ratio of early mitral flow velocity to that at atrial contraction and a short mitral deceleration time. In a minority of patients the mitral flow velocity variables and pattern were normal. These mitral flow velocity patterns are illustrated schematically in Figure 7.

**Pattern I.** This pattern, most frequently seen in the patients with coronary artery disease who had lower pulmonary wedge pressure, appears to occur when there is impaired left ventricular relaxation (as suggested by a prolongation of the time constant of isovolumic relaxation) in the presence of a normal or only slightly increased left atrial pressure. Assuming that left atrial pressure at the time of mitral valve opening remains constant, a slower rate (reduced slope) of left ventricular isovolumic pressure fall would result in a later mitral valve opening, longer interval from aortic closure to mitral valve opening (isovolumic relaxation time), reduced early diastolic transmittal pressure gradient and a resultant decrease in peak early flow velocity. With less filling in early diastole, the percent of left ventricular filling with atrial contraction would likely be increased as a compensatory mechanism, perhaps aided by atrial hypertrophy or enhanced atrial systolic function. The long mitral deceleration time frequently seen in these patients may reflect a prolonged fall in left ventricular pressure associated with the impaired relaxation and the low early diastolic ventricular filling rates. This mitral flow velocity pattern was also seen in some of the patients with congestive cardiomyopathy who had lower pulmonary wedge pressure.

**Pattern II.** In contrast to pattern I, this pattern tended to be seen in patients who were more symptomatic (functional classes III and IV) and it appears to indicate the presence of increased filling pressures, an abnormally increased left
ventricular rapid filling wave and a "restrictive" left ventricular filling pattern with an abrupt, premature cessation of mitral flow velocity in early diastole. As assessed by T, the patients with coronary artery disease and congestive cardiomyopathy who had pattern II also had impaired left ventricular relaxation. However, the normal or short left ventricular isovolumic relaxation time and normal or increased peak mitral flow velocity in early diastole in these patients suggests that an elevated left atrial pressure normalized or increased the early diastolic transmirtal pressure gradient and masked the expected effect of the relaxation abnormality on this gradient and the Doppler variables. A relatively greater increase in early diastolic transmirtal pressure gradient with higher left atrial pressure may occur in part because of the characteristics of the reduction in left ventricular isovolumic pressure, which is exponential rather than linear (6). Assuming a constant T and rate of left atrial pressure decline, a higher pressure at the time of mitral valve opening will result in a larger transmirtal pressure gradient because the slope of the left ventricular pressure decrease is steeper at the higher pressure. The reason why the increase in left atrial pressure in these patients appears to be larger than the increase in left ventricular pressure minimum, which increases the early diastolic transmirtal pressure gradient is unknown.

The patients with pattern II had a short mitral deceleration time (Mdt) and the rapid filling wave in the left ventricle (LV RFW). Panel B shows the lack of correlation between mitral flow velocity at atrial contraction (M2) and increase in left ventricular diastolic pressure with atrial contraction (LV Δa).

The patients with pattern II had a short mitral deceleration time (Fig. 6A). This was inversely correlated with the left ventricular rapid filling wave, and mitral deceleration times of <130 ms were seen only in patients with rapid filling waves of >5 mm Hg. This "restrictive" left ventricular filling pattern, with its abrupt increase in left ventricular diastolic pressure and premature cessation of mitral flow, was seen in patients in all three study groups and in patients with an enlarged as well as a nonenlarged left ventricle. This pattern likely reflects a marked decrease in left ventricular chamber compliance, which is characteristic of advanced disease of various origins (32). In some patients mid-diastolic mitral regurgitation was recorded by pulsed Doppler technique near the peak of the rapid filling wave (Fig. 4B), suggesting that left ventricular pressure may have actually exceeded left atrial pressure at that time.

Most patients with pattern II had left atrial enlargement and a decrease in mitral flow velocity at atrial contraction. This decrease in velocity may be related to systolic atrial dysfunction perhaps as a result of a long-standing increase in atrial afterload from an elevated left ventricular pressure or increased viscous forces (32) or to an elevated left ventricular pressure present at the time of atrial contraction.

"Normalized" mitral flow velocity patterns. In this study some patients with a reduced rate of left ventricular relax-
Figure 7. Schematic diagram illustrating the different mitral flow velocity patterns discussed in this study and their dynamic nature. The electrocardiogram, phonocardiogram (PHONO) and left ventricular isovolumic relaxation time interval (IVRT) from aortic valve closure (AC) to mitral valve opening (MO) are labeled. A normal mitral flow velocity pattern is represented by the middle figure. Characteristics of pattern I compared with the normal pattern include increases in isovolumic relaxation time, mitral deceleration time and flow velocity at atrial contraction with decreases in peak early mitral flow velocity and ratio of peak early flow velocity to flow velocity at atrial contraction. Compared with a normal pattern, pattern II is characterized by a decrease in isovolumic relaxation time, deceleration time and flow velocity at atrial contraction and an increase in the ratio of peak early velocity to that at atrial contraction. The normalized pattern indicates that patients with impaired left ventricular relaxation and elevation of left atrial pressure may exhibit a pattern that sparesly resembles normal. The arrows indicate that these patterns represent a dynamic continuum and may change from one pattern to another with changes in hemodynamics and disease status. A = aortic valve closure; S = mitral valve closure.

Although the transvalvular pressure gradient diminishes rapidly after mitral valve opening, forward flow continues in mid-diastole presumably because of the inertial energy imparted to the blood by the early pressure gradient (36,37). The findings in this study (Fig. 2) are compatible with these reports, and a correlation was found between peak early mitral flow velocity and estimates of crossover pressure and the early diastolic transmitral pressure gradient (Fig. 5A). In the patients with simultaneous Doppler and hemodynamic recordings, we found that the left ventricular pressure minimum coincided closely in time with the peak early mitral flow velocity, whereas deceleration time was best correlated with the height and steepness of the left ventricular rapid filling wave.

In the patients with pattern I there was no correlation between mitral flow velocity and left ventricular pressure increase at atrial contraction. This finding suggests that some patients may have a reduced rate of left ventricular relaxation, but may accept volume without an abnormal increase in left ventricular pressure later in diastole after relaxation is complete. In contrast, other patients with abnormal left ventricular relaxation also have an abnormal increase in ventricular pressure with atrial contraction, perhaps because of a coexistent decrease in myocardial or chamber compliance. A separation of left ventricular filling

Determinants of left ventricular filling and the mitral flow velocity pattern. Studies in patients with mitral stenosis (33,34) have shown that the peak mitral flow velocity accurately reflects the maximal diastolic pressure difference between the left atrium and left ventricle. In the absence of valvular obstruction, the major determinant of peak early mitral flow velocity also appears to be the instantaneous left atrial to left ventricular pressure difference in early diastole (35-37). Factors postulated to affect this instantaneous pressure gradient include 1) the left atrial pressure at the time of mitral valve opening (crossover pressure); 2) the rate of relaxation of the left ventricle; 3) the myocardial and chamber compliance of the left atrium and left ventricle; 4) the end-systolic left ventricular volume; and 5) the passive viscoelastic properties of the myocardium during filling.
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Figure 8. Mitral flow velocity recorded simultaneously with left ventricular (LV) and pulmonary wedge (PW) pressures before and after left ventriculography. After ventriculography the pulmonary wedge V wave increased from 10 to 24 mm Hg, the left ventricular pressure minimum increased from 3 to 7 mm Hg, the pulmonary wedge V wave - left ventricular pressure minimum difference increased from 7 to 17 mm Hg, the left ventricular rapid filling wave increased from 2 to 8 mm Hg (short black arrows) and the end-diastolic pressure increased from 9 to 19 mm Hg. After ventriculography, the mitral flow velocity recording shows a decrease in left ventricular isovolumic relaxation time from 88 to 77 ms, increase in peak mitral flow velocity in early diastole (M1) from 93 to 144 cm/s, while the deceleration time decreased from 224 to 160 ms. Note how the increase in peak mitral flow velocity in early diastole (M1) correlated with an increase in the pulmonary wedge V wave - left ventricular diastolic pressure minimal difference and the decrease in deceleration time with an increase in rapid filling wave.

into an energy-dependent relaxation phase in early diastole and a later phase predominantly affected by intrinsic myocardial properties has been previously proposed (35,38).

Clinical interpretation of mitral flow velocity patterns: an integrated framework. The results of this study suggest that, despite the complex nature of factors affecting ventricular filling, clinically useful information regarding left ventricular diastolic pressures and function is associated with distinct mitral flow velocity patterns. We hypothesize that this occurs because certain factors influencing these patterns predominate under different circumstances. For instance, in patients with normal left atrial pressure, it appears that the reduced rate of left ventricular relaxation is the predominant factor that results in a mitral flow velocity pattern like pattern I. However, as left atrial pressure increases, the early diastolic transmirtal pressure gradient and mitral flow velocity pattern return toward normal (becoming "normalized") despite the presence of impaired left ventricular relaxation. In patients with restrictive, congestive or ischemic cardiomyopathy who have elevated filling pressures, a marked reduction in left ventricular chamber compliance appears to be the dominating influence that overwhelms the effect of a reduced rate of left ventricular relaxation and results in the mitral flow velocity pattern changing to pattern II. As shown schematically in Figure 7, we hypothesize that these patterns represent a dynamic continuum, which may change from one pattern to another as a result of disease progression, medical therapy or sudden changes in hemodynamic status.

The dynamic nature of mitral flow velocity recordings. This was illustrated in the patients studied before and after various interventions. The patients studied after left ventriculography showed an increase in pulmonary wedge and left ventricular diastolic pressures, an increase in the early diastolic transmitral pressure gradient and an increase in the left ventricular rapid filling wave (Fig. 8). These hemodynamic changes were associated with a decrease in isovolumic relaxation time, an increase in peak early mitral flow velocity and a decrease in mitral deceleration time. In the patient with congestive cardiomyopathy and low filling pressures (Fig. 9), volume loading results in partial "normalization" of the mitral flow velocity recording despite an abnormal increase in pulmonary wedge and left ventricular end-diastolic pressures. In Figure 10, dynamic changes in mitral flow velocity variables occur on a beat to beat basis in a cardiac transplant patient whose native atrial contraction is variably affecting left atrial and left ventricular diastolic pressures (39).

Comparison with previous studies. Previous studies have reported the presence of a prolonged left ventricular isovolumic relaxation time, prolonged T or reduced peak rate of early diastolic left ventricular filling in patients with coronary artery disease (1-5,8,9,12-16), congestive cardiomyopathy (1,12,13,15,16,32), left ventricular hypertrophy (40), hypertrophic cardiomyopathy (1,5,9,41) and systemic hypertension (1,8,16). In similar patient groups, studies utilizing pulsed Doppler techniques (5,9,13,17-24) have shown that many patients exhibit a mitral flow velocity pattern or variables similar to pattern I, with a reduced peak early mitral flow velocity and mitral deceleration rate, increased mitral flow velocity at atrial contraction and reduced ratio of early mitral flow velocity to that at atrial contraction. In hypertension, these changes have been seen in the absence of increased wall thickness (8) and have persisted after control of blood pressure (23). In comparative studies, this mitral flow velocity filling pattern suggesting decreased early diastolic filling has shown a strong correlation with results from angiographic (13) and radionuclide methods (9,16).
Figure 9. Electrocardiogram, phonocardiogram, mitral flow velocity and left ventricular (LV) pressure before and after volume loading in a patient with congestive cardiomyopathy and hypovolemia. In panel A, the left ventricular diastolic pressure is normal and the mitral flow velocity recording shows a long isovolumic relaxation time (AC - MO) and low velocity in early diastole. In panel B, recorded after 500 cc of volume had increased mean pulmonary wedge pressure from 1 to 20 mm Hg, the isovolumic relaxation time is shorter, peak early mitral flow velocity is slightly higher and the left ventricular end-diastolic pressure is increased (17 mm Hg). Abbreviations as in Figure 7.

These results suggest that impaired relaxation is an early sign of left ventricular dysfunction of various origins and that Doppler echocardiography may be a sensitive method in detecting such abnormalities. In patients with heart failure, a decreased atrial contribution to ventricular filling has been described (32,40) that showed an inverse correlation to pulmonary wedge pressure (40).

A mitral flow velocity pattern similar to pattern II has been reported in some patients with congestive (21) and hypertrophic (20) cardiomyopathy. In these patients, who were assumed to have a left ventricular relaxation abnormality, the masking of the relaxation abnormality was ascribed to the presence of mitral regurgitation. However, the severity of the regurgitation was not quantified and hemodynamic data were not included in these reports. In other patients with hypertrophic cardiomyopathy (42,43), the marked variation in mitral flow velocity patterns has been ascribed to other characteristics of the disease process.

Although other studies have reported that mitral regurgitation can result in an increase in mitral flow velocity (13) or mitral flow (37), there was no correlation in this study between severity of regurgitation and either the height of the pulmonary wedge Y wave or peak early mitral flow velocity except in the five patients with severe mitral regurgitation. This probably reflects the fact that multiple factors that affect peak mitral flow velocity are altered in these patients with cardiac disease. Similarly, there was no correlation between peak early mitral flow velocity and cardiac output.

Figure 10. Mitral flow velocity recorded simultaneously with respiration (resp), electrocardiogram and pulmonary wedge (PW; and left ventricular (LV) pressures in a cardiac transplant recipient. Note the beat to beat variation in size and rate of rise of the left ventricular rapid filling wave which corresponds to variation in mitral deceleration time (DT). This beat to beat variation is common in transplant recipients and is most likely the result of a dissociated native atrial contraction variably affecting left ventricular hemodynamics.

Previous studies (7,10,12,40,44) have reported that ventricular filling rates may vary with hemodynamic status and other factors in individual disease states. In patients with aortic stenosis (44), some individuals with increased pulmonary wedge pressure, and apparently more severe disease, have a more normal left ventricular filling pattern. In patients with congestive cardiomyopathy, peak left ventricular filling rate in early diastole has been reported as normal in some individuals (12), whereas other patients have been shown to have a large left ventricular rapid filling wave and elevated diastolic pressure in the presence of a prolonged T (32). In an animal study (43), the peak left ventricular filling rate in early diastole was shown to be determined by left atrial pressure at the time of mitral valve opening as well as by the rate of left ventricular relaxation. In that study there was no change in filling rate after angiotensin infusion, despite an increase in T, when left atrial pressure increased simultaneously. These studies all support the hypothesis that an elevation in left atrial pressure, and possibly other factors, can mask a left ventricular relaxation abnormality when assessed by peak rates of filling or filling patterns. Therefore, as illustrated by the patients with coronary artery disease in this study, the use of these techniques to evaluate left ventricular diastolic function may be misleading if patients with elevated left atrial pressure are analyzed together with those who have more normal pressures.

Dynamic changes in the mitral flow velocity pattern have been reported in patients with coronary artery disease after preload reduction (26) and in patients with hypertrophic cardiomyopathy after exercise and therapy with diltiazem (22). In the latter (22), a patient with congestive cardiomyo-
pathy and a mitral flow velocity pattern resembling pattern II had a more normal-looking pattern after nitroglycerin administration and a decrease in the left ventricular pressure minimum and rapid wave. We have observed similar changes in mitral flow velocity patterns in patients with coronary artery disease and congestive cardiomyopathy after medical therapy for overt congestive heart failure.

Study limitations. The assessment of left ventricular diastolic function is a complex phenomenon that is difficult to study in patients or even in a laboratory setting (38,46). Changes in inotropic state and both systolic and diastolic loading conditions have been reported to influence T and left ventricular filling in early diastole (47,48). After left ventricular relaxation is complete, the assessment of passive myocardial properties is also difficult to quantify in vivo. The mitral flow velocity variables obtained in this study do not measure, and should not be directly equated with, T, left ventricular chamber compliance, myocardial compliance or the diastolic volume or pressure of the left atrium or ventricle. Furthermore, although the flow velocity profile appears to accurately reflect the pattern of left ventricular filling (9,13,16), it does not quantitate actual transvalvular flow.

In this study it is likely that many of the bivariate correlation coefficients were heavily influenced by the patient groups studied. For instance, patients with pattern I are likely to demonstrate a positive correlation between isovolumic relaxation time and T. Conversely, patients with pattern II may demonstrate a negative correlation between these variables. Therefore, the overall correlation for the entire study group would likely depend on the characteristics of the group studied and the correlation coefficients presented in this report should not be extrapolated to other patient groups. Future work investigating these relations will also need to take into account any selection bias in their analysis.

When interpreting mitral flow velocity patterns a number of additional factors that may affect the transmirtal flow velocity variables must be considered. Despite the findings in this study, it has been reported (25,27,28) that the ratio of mitral flow velocity in early diastole to that with atrial contraction and the rate of early diastolic deceleration decrease with age in apparently healthy older adults. Conversely, mitral deceleration times and the left ventricular isovolumic relaxation time may be shorter in children because of a more rapid rate of left ventricular relaxation. By shortening diastole, sinus tachycardia may decrease the time available for early diastolic filling, increasing the proportion of filling that occurs with atrial contraction. The left ventricular isovolumic relaxation time has been reported (40) to be affected by several factors in addition to left atrial pressure at the time of mitral valve opening and the rate of left ventricular relaxation. Aortic regurgitation that results in an increase of the left ventricular rapid filling wave may also decrease deceleration time. A decrease in preload (26) and mitral regurgitation (13,20,21,37) may also affect mitral flow velocity variables.

In the present study T was not quantified in all of the patients. However, this index of left ventricular relaxation has been shown to be abnormally prolonged in nearly all patients with coronary artery disease (1-5,15,32) and congestive cardiomyopathy (15,32). Studies in patients with a restrictive myocardial disease also suggest the presence of abnormal left ventricular relaxation as assessed by T (49) or rates of early diastolic ventricular filling (50,51).

The patients with congestive or restrictive cardiomyopathy in this study tended to have advanced disease and markedly elevated pulmonary wedge pressure. It is likely that more variation in mitral flow velocity patterns would be seen in these groups if patients with less abnormal hemodynamics were included. A technical limitation in interpreting the recordings may occur in patients with a rapid heart rate or atrioventricular block in whom atrial contraction may occur at a time that causes fusion of early mitral flow velocity and flow velocity at atrial contraction.

Doppler methods. In this study, pulsed wave transmirtal flow velocities were recorded with a low filter setting and the sample volume was placed between the leaflets at a point where the largest early mitral flow velocity is recorded. A sample volume position at the anulus (52) or too far into the left ventricle can give lower velocities and different deceleration times. Deceleration time was measured instead of slope because it is independent of velocity. Continuous wave Doppler ultrasound is not adequate for these recordings and gives longer mitral deceleration times than do pulsed wave recordings. For accurate measurement of left ventricular isovolumic relaxation time a high quality mitral flow velocity recording with simultaneous phonocardiogram at 100 mm/s should be used.

Clinical implications. Despite these limitations and the oversimplification of a complex phenomenon, mitral flow velocity recordings appear to have clinical potential that deserves further investigation in assessing left ventricular diastolic properties in patients with heart disease. In this study, only 5 of 49 patients with an increased left ventricular end-diastolic pressure and 4 of 15 patients with a normal end-diastolic pressure had normal values for all variables measured from mitral flow velocity recordings. Therefore, in patients with disease processes known to affect left ventricular diastolic function, pattern I suggests a reduced rate of left ventricular relaxation with relatively normal pressures. In more symptomatic patients, pattern II appears to indicate a "restrictive" left ventricular filling pattern and an increased left atrial pressure regardless of the severity of the left ventricular relaxation abnormality, origin of disease or left ventricular size. "Normalized" patterns may be seen in patients with a reduced rate of left ventricular relaxation abnormality and moderately elevated left atrial pressure. These patients may be difficult to recognize although the
combination of an appropriate clinical history, data from other diagnostic procedures, one or more abnormal mitral flow velocity variables and serial recordings may provide important clues to their abnormality. Similarly, in patients with an abnormal pattern, serial studies may be helpful to evaluate symptoms, progression of disease or the results of medical or surgical therapy.

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