Doppler-Derived Mitral Deceleration Time
An Early Strong Predictor of Left Ventricular Remodeling After
Reperfused Anterior Acute Myocardial Infarction

Giampaolo Cerisano, MD; Leonardo Bolognese, MD, FESC; Nazario Carrabba, MD;
Piergiovanni Buonamici, MD; Giovanni Maria Santoro, MD, FESC; David Antoniucci, MD;
Alberto Santini, MD; Guia Moschi, MD; Pier Filippo Fazzini, MD

Background—The relation between remodeling and left ventricular (LV) diastolic function has not yet been fully investigated. The aim of this study was to determine whether early assessment of Doppler-derived mitral deceleration time (DT), a measure of LV compliance and filling, may predict progressive LV dilation after acute myocardial infarction (AMI).

Methods and Results—Fifty-one patients (aged 61 ± 11 years; 6 women) with anterior AMI successfully treated with direct coronary angioplasty underwent 2-dimensional and Doppler echocardiographic examinations within 24 hours of admission, at days 3, 7, and 30 and 6 months after the index infarction. Mitral flow velocities were obtained from the apical 4-chamber view with pulsed Doppler. End-diastolic volume index (EDVI) and end-systolic volume index (ESVI) were calculated with the Simpson’s rule algorithm. Patients were divided according to the DT duration assessed at day 3 in 2 groups: group 1 (n = 33) with DT >130 ms and group 2 (n = 18) with DT ≤130 ms. Patency and restenosis rate at 6 months were similar between the 2 groups (94% group 1 vs 89% group 2; P = 0.52; 27% group 1 vs 33% group 2; P = 0.64, respectively). LV volume indexes were similar in both groups at baseline (EDVI: 71 ± 3 mL/m² group 1 vs 70 ± 3 mL/m² group 2, P = 0.42; ESVI: 43 ± 3 group 1 vs 48 ± 3 mL/m² group 2, P = 0.13, respectively). From day 3 on, LV volume indexes progressively increased in group 2 and were significantly larger than those of group 1 at 6 months (LVEDVI 61 ± 3 group 1 vs 104 ± 6 mL/m² group 2, P = 0.00001; LVESVI 31 ± 3 group 1 vs 73 ± 6 mL/m² group 2, P = 0.00001, respectively). A significant inverse correlation was found between DT and changes in EDVI at 6 months (r = −0.68; P < 0.000001). By stepwise multiple regression analysis among several clinical, demographic, angiographic, and echocardiographic variables, DT was the most powerful predictor of EDVI changes at 6 months (P = 0.02).

Conclusions—These data suggest that early estimation (day 3) of Doppler-derived mitral DT provides a simple and accurate mean to predict late LV dilation after reperfused AMI. (Circulation. 1999;99:230-236.)

Key Words: diastole • remodeling • myocardial infarction • echocardiography

Ventricular remodeling has emerged as one of the dominant factors that determines the long-term survival of postinfarction patients.1 Several variables have been identified to predict an increase in left ventricular (LV) volume after acute myocardial infarction (AMI). These include infarct size,2 anterior location,3 transmurality of the infarct,4 and patency of the infarct-related artery.5 However, other not-yet well-defined determinants may influence and modify the remodeling process. One of the important functional aspects of remodeling is that it is accompanied by changes in diastolic properties of the left ventricle caused by scar formation, which increases stiffness, and hypertrophy of normal segments, which have delayed relaxation.6 LV dilation may occur early after infarction, probably in part because of elevated filling pressure within the ventricle.5,7,8 Doppler echocardiography has provided a rapid, feasible, and simple noninvasive method of assessing LV filling in various cardiac diseases9 in which diastolic abnormalities have been observed, including AMI.7,10–12 Among the various diastolic variables, shortening of the deceleration time (DT) of the early filling wave, indicative of a “restrictive” filling pattern, has been found to correlate with infarct size13 and to predict an adverse outcome of postinfarction patients.11,14 However, no previous study has correlated serial changes in filling patterns with serial changes in LV volumes after AMI. We hypothesized that early assessment of Doppler-derived mitral DT would predict progressive LV dilation after AMI, and serial changes in filling pattern might parallel the evolutionary changes in LV dimensions. To test this hypothesis, we performed a prospective study of patients with first anterior
AMI treated with primary coronary angioplasty. To avoid the confounding impact of infarct-related artery patency and residual stenosis on subsequent changes in LV dimensions, only patients in whom anterograde flow was fully restored without significant residual stenosis were included in the study. Serial assessments of Doppler-derived DT were obtained at days 3, 7, and 30 and 6 months after the index infarction.

**Methods**

**Patients and Study Protocol**

We prospectively studied 72 patients with anterior AMI selected among 142 patients consecutively referred to the catheterization laboratory of Careggi Hospital for emergency coronary angioplasty between April 1996 and January 1997. The study inclusion criteria were (1) confirmed first anterior AMI; (2) successful primary coronary angioplasty (defined as Thrombolysis In Myocardial Infarction Trial [TIMI] flow grade 3 and residual stenosis <30%) within 6 hours of the onset of symptoms or between 6 and 24 hours if there was evidence of continuing ischemia. Exclusion criteria were clinical signs of heart failure or cardiogenic shock in the first week after AMI, postinfarction angina, atrial fibrillation, significant valvular heart disease, and life-limiting noncardiac disease. In particular, no patient with significant (moderate to severe) mitral regurgitation was included in the study. No upper age limit was used. Of the 59 patients selected for the study, 2 (3%) were excluded because of inadequate quality of echocardiographic images and mitral Doppler tracings on the baseline echocardiographic examination. Later, further exclusions were for reinfarction (1) and death (2); an additional 3 patients did not adhere to the follow-up protocol. Thus 51 patients (45 men, 6 women, mean age 61 ± 11 years, range 34 to 89 years) were enrolled in the study. The research protocol was approved by the hospital Ethics Committee, and informed consent was obtained from each patient by one of the investigators. All patients underwent serial assessment of LV filling patterns at days 3 and 7 and LV volumes within 24 hours of admission on days 3 and 7 after the index infarction. A comprehensive echocardiographic examination and repeat coronary angiography were obtained at 1 and 6 months after primary coronary angioplasty in all patients.

**Echocardiography**

Complete M-mode and 2-dimensional echocardiography and Doppler ultrasound examination were performed with commercially available imaging systems (Aloka model SSD 870 2.5- and 3.5-MHz transducers). LV diastolic filling patterns were determined by the mitral inflow pulsed-wave Doppler examination with a 2.5-MHz transducer. In the apical 4-chamber view, the Doppler sample volume was placed in the middle of the LV inflow tract ~1 cm below the plane of the mitral annulus between the mitral leaflet tips, where maximal flow velocity in early diastole was recorded. Special care was taken to align the sample volume as close to perpendicular as possible to the mitral annular plane. Images were stored on a videotape by a 0.5-inch VHS cassette recorder (Sony SVO, 140 PA) for further analysis.

**Data Analysis**

Two investigators blinded to the clinical and angiographic data analyzed baseline and follow-up 2-dimensional echocardiograms and Doppler tracings. Discrepancies were resolved by consensus.

Two-dimensional echocardiographic images were transferred to the hard disk of a Tomtec P90 medical off-line computer analysis system and digitized. LV volumes and ejection fraction (EF) were then measured with the modified Simpson’s rule algorithm. The mean values of 3 measurements of the technically best cardiac cycles were taken from each examination. The volume indexes were obtained by dividing the volume by the body surface area at each time point. Intraobserver and interobserver variability values in the evaluation of end-systolic and end-diastolic volumes were <5%, indicating the good reproducibility of the measurements. On the basis of repeated measurements in individual patients and on the upper 95% confidence limit of the intraobserver variability, an increase in end-diastolic volume index >20% was considered LV dilation. The ratio of long-axis length to short-axis length at end diastole and end systole was measured from the apical chamber view and taken as an index of eccentricity, with a value equal to 1 for this index corresponding to a spherical shape and values of >1 representing more ellipsoid shapes.

The dimension of the left atrium was measured at end systole from an M-mode recording at the level of aortic root. LV mass was calculated by the cube formula. The left ventricle was divided according to a 16-segment model. For each segment, wall motion was scored from 1 (normal) to 4 (dyskinetic). In evaluating regional wall motion abnormalities, attention was also paid to the systolic thickening in the central portion of each segment. Anterior infarct zone was constructed, and in each patient both global and infarct zone wall motion score indexes (WMSI) were derived for baseline and follow-up 2-dimensional echocardiograms.

From Doppler spectra of 3 to 5 consecutive cardiac cycles, average values were calculated for the following diastolic variables: peak velocity of early rapid filling wave (E), peak flow velocity at atrial contraction (A), peak E/A wave velocity ratio, and DT of early filling. A digitizing pad and microcomputer were used to analyze the Doppler waveforms (leading edge). To avoid the influence of heart rate, deceleration time was calculated as the time between peak E wave and the upper deceleration slope extrapolated to zero line. Cardiac cycles with nonlinear deceleration slopes and fusion of early and late mitral flow velocity were excluded from the analysis. A DT >130 ms was classified as nonrestrictive, and ≤130 ms was defined as restrictive. This cutoff point has been shown to be consistent with restrictive hemodynamics and a powerful independent predictor of unfavorable outcome after acute myocardial infarction and idiopathic dilated cardiomyopathy. To avoid the effects of acute ischemia on LV filling patterns, we chose to measure baseline DT on day 3 after the index infarction.

**Statistical Analysis**

Continuous data are expressed as mean±SD. Echocardiographic variables are expressed as mean±SE. Baseline data were compared by means of the χ² test for categorical variables and unpaired t test for continuous variables. ANOVA with the Tukey post hoc test was used to analyze repeated measures of WMSI, EF, LV volumes, and Doppler-derived diastolic variables. Simple linear regression analysis was used to correlate DT, peak creatine kinase, and WMSI with the changes in LV end-diastolic volume index. Linear regression analysis was also used to determine intraobserver and interobserver variability. Linear and multiple regression analyses were carried out to test the relation between clinical, echocardiographic, and hemodynamic variables and DT. Univariate and multivariate regression analyses were performed to identify independent correlates of the changes in LV end-diastolic volume index. A value of P<0.05 was considered statistically significant. Statistical analysis was performed with Statistica 4.5 for Windows (StatSoft, Inc., 1993).

**Results**

**Baseline Patient Characteristics**

Patients were divided into 2 groups according to Doppler echocardiographic mitral pattern assessed at baseline (day 3) Doppler ultrasound examination. Thirty-three patients showed a DT >130 ms (group 1), and 18 had a DT ≤130 ms (restrictive LV filling pattern, group 2). All patients were receiving ACE therapy. There was no significant difference between the 2 groups with respect to age, sex, frequency of coronary risk factors, time from onset of symptoms to reperfusion, angiographic collateral grade, multivessel disease, left atrial size, LV mass index, and Doppler mitral curves as regard E, A peak velocity, and E/A ratio (Table 1).
Deceleration Time and Ventricular Remodeling

However, signs of reduced infarct size were more common among patients of group 1. These patients had a markedly lower mean peak creatine kinase and baseline (P=0.00001) among patients of group 1. These patients had a markedly lower mean peak creatine kinase and baseline WMSI and a higher EF (Table 1).

Since a sizable number of patients with a short DT had an E/A ratio between 1 and 2, to clarify if this LV inflow pattern was indicative of pseudonormalization, pulmonary venous flow velocities by transthoracic pulsed Doppler ultrasound and early diastolic velocity of mitral annulus (Ea) by Doppler tissue imaging were measured ( Aloka SSD 2002) in 8 consecutive patients with short DT (≤130 ms; mean 120±7; range 116 to 126) and an apparently normal (>1<2) E/A ratio (mean 1.2±0.2; range 1.1 to 1.43) 3 days after anterior AMI. Pulmonary venous flow reversal exceeded the duration of the mitral A wave in all patients (difference in duration 51±9 ms; range 45 to 75 ms), and the systolic fraction of pulmonary venous flow (the ratio of systolic to the sum of systolic and diastolic velocity integral) was markedly (≤0.5) decreased in all patients (mean 0.43±0.03; range 0.40 to 0.49). These data are indicative of elevated LV end-diastolic pressure and elevated LV pressure before atrial systole, respectively, despite an apparently normal E/A ratio. In agreement with pulmonary venous flow velocities recordings, the ratio of transmitral E-wave velocity to Doppler tissue imaging Ea was elevated (14±3; range 11.7 to 17.5), suggesting an increased LV end-diastolic pressure.

Linear and Multiple Regression Analysis Between Clinical Variables and DT
The correlation coefficients between DT and the clinical, Doppler echocardiographic, and hemodynamic variables are shown in Table 2. On multivariate analysis, DT was independently correlated with peak creatine kinase (P=0.006) and EF (P=0.018).

Angiographic Results
Lesion minimal lumen diameter increased from 0.19±0.30 mm at baseline to 3.18±0.42 mm after coronary angioplasty in group 1 and from 0±0 to 3.23±0.49 mm in group 2.

At 1 month, the angiographic patency rate was 100% in both groups. Lesion minimal diameter was 2.96±0.65 mm in group 1 and 3.35±0.52 mm in group 2 (P=0.016). At 6 months, patency of the infarct-related artery was 94% in group 1 and 89% in group 2 (P=0.052). No significant difference was found in minimal lumen diameter and restenosis rate (>50%) between the 2 groups.

### Table 1. Baseline Clinical, Doppler Echocardiographic, and Angiographic Characteristics of the Study Population

<table>
<thead>
<tr>
<th></th>
<th>All (n=51)</th>
<th>Group 1 DT &gt;130 ms (n=33)</th>
<th>Group 2 DT ≤130 ms (n=18)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>61±11</td>
<td>61±11</td>
<td>61±11</td>
<td>0.5</td>
</tr>
<tr>
<td>Male, %</td>
<td>88 (45)</td>
<td>91 (30)</td>
<td>83 (15)</td>
<td>0.42</td>
</tr>
<tr>
<td>Killip class</td>
<td>1.72±1</td>
<td>1.51±0.9</td>
<td>2.11±1.18</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>Diabetes, %</td>
<td>24 (12)</td>
<td>24 (8)</td>
<td>22 (4)</td>
<td>0.87</td>
</tr>
<tr>
<td>Hypertension, %</td>
<td>27 (14)</td>
<td>24 (8)</td>
<td>33 (6)</td>
<td>0.48</td>
</tr>
<tr>
<td>Dyslipidemia, %</td>
<td>22 (11)</td>
<td>21 (7)</td>
<td>22 (4)</td>
<td>0.93</td>
</tr>
<tr>
<td>Smoking, %</td>
<td>45 (23)</td>
<td>48 (16)</td>
<td>39 (7)</td>
<td>0.51</td>
</tr>
<tr>
<td>Onset of reperfusion, min</td>
<td>200±72</td>
<td>195±65</td>
<td>210±86</td>
<td>0.5</td>
</tr>
<tr>
<td>Peak CK, U/L</td>
<td>3311±2311</td>
<td>2222±1647</td>
<td>5307±2019</td>
<td>&lt;0.00001</td>
</tr>
<tr>
<td>IZ WMSI</td>
<td>2.54±0.32</td>
<td>2.43±0.33</td>
<td>2.75±0.13</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>EF, %</td>
<td>37±8</td>
<td>41±6</td>
<td>30±7</td>
<td>&lt;0.00001</td>
</tr>
<tr>
<td>LVEDVI, mL/m²</td>
<td>71±18</td>
<td>71±19</td>
<td>70±14</td>
<td>0.42</td>
</tr>
<tr>
<td>LVESVI, mL/m²</td>
<td>45±15</td>
<td>43±16</td>
<td>48±13</td>
<td>0.13</td>
</tr>
<tr>
<td>LVMI, g/m²</td>
<td>89±19</td>
<td>90±21</td>
<td>87±14</td>
<td>0.37</td>
</tr>
<tr>
<td>LADI, cm/m²</td>
<td>1.8±0.2</td>
<td>1.8±0.2</td>
<td>1.8±0.1</td>
<td>0.44</td>
</tr>
<tr>
<td>E peak, cm/s</td>
<td>57±15</td>
<td>57±13</td>
<td>59±19</td>
<td>0.32</td>
</tr>
<tr>
<td>A peak, cm/s</td>
<td>59±19</td>
<td>58±16</td>
<td>62±24</td>
<td>0.23</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>1.07±0.5</td>
<td>1.06±0.4</td>
<td>1.09±0.5</td>
<td>0.4</td>
</tr>
<tr>
<td>DT, ms</td>
<td>162±43</td>
<td>185±34</td>
<td>120±18</td>
<td>&lt;0.00001</td>
</tr>
<tr>
<td>IVRT, ms</td>
<td>117±23</td>
<td>117±24</td>
<td>119±21</td>
<td>0.4</td>
</tr>
<tr>
<td>Collaterals (grade ≥2), %</td>
<td>4 (2)</td>
<td>3 (1)</td>
<td>6 (1)</td>
<td>0.65</td>
</tr>
<tr>
<td>Multivessel CAD, %</td>
<td>39 (20)</td>
<td>36 (12)</td>
<td>44 (9)</td>
<td>0.57</td>
</tr>
</tbody>
</table>

CK indicates serum creatine kinase; IZ WMSI, infarct zone WMSI; LVESVI, left ventricular end-systolic volume index; LVEDVI, left ventricular end-diastolic volume index; LVMI, left ventricular mass index; LADI, left atrial diameter index; E wave, peak velocity of E wave; A wave, peak velocity of A wave; IVRT, isovolumic relaxation time; and CAD, coronary artery disease.

Values in parentheses are numbers of patients.
Changes in Regional and Global Ventricular Function and LV Volumes

At baseline, both global and regional contractile function were significantly better in group 1 than in group 2 (EF 41 ±1% vs 30 ±2%, P < 0.00001; WMSI: 2.43 ±0.06 vs 2.75 ±0.03, P < 0.0001). According to ANOVA, a significant progressive improvement in LVEF was observed in group 1, whereas it remained unchanged throughout the study period in group 2 (Figure 1A). Comparison between groups by ANOVA revealed that patients with a DT >130 ms (group 1) had a significantly higher improvement of global ventricular function at 6 months than did patients with a DT ≤130 ms. (Figure 1A). Similarly, the regional contractile function, expressed as WMSI, showed a higher improvement in patients of group 1 compared with those of group 2 (Figure 1B). Eleven patients (44%) with LVEF ≤40% did not have significant dilation during follow-up. Conversely, 5 (19%) patients with LVEF ≥40% had a significant increase (>20%) in chamber volume.

End-diastolic and end-systolic volume indexes were similar in both groups at baseline. From day 3 on, LV volume indexes progressively increased in group 2 and were significantly larger than those of patients of group 1 at each study point in time (Figure 1, C and D). In patients of group 1, end-diastolic and end-systolic volume decreased after 1 month (Figure 1, C and D). Finally, at baseline, eccentricity index at end diastole and at end systole were similar in both groups (group 1, 1.58 vs 1.63 group 2, P = 0.96; and 1.84 vs 1.85, P = 0.99, respectively). The eccentricity index at end diastole decreased from 1.63 to 1.49 at 6 months in group 2, indicating a more spherical shape, but increased from 1.58 to 1.63 in group 1 (P = 0.0009 for Δ group 1 vs Δ group 2). At end systole, the index decreased from 1.85 to 1.69 in group 2 but increased from 1.84 to 1.91 in group 1 (P = 0.004 for Δ group 1 vs Δ group 2).

Serial Changes of Filling Pattern

DT increased on day 7 and at 1 and 6 months in both groups (Figure 2). However, the increase in DT was significantly

| TABLE 2. Linear Correlation Between DT and Clinical and Echocardiographic Variables |
|---------------------------------|----------|----------------|
|                                  | r        | P              |
| Peak CK                         | 0.59     | <0.000002      |
| EF                              | 0.54     | <0.00002       |
| IZ WMSI                         | 0.50     | <0.00008       |
| SBP                             | 0.16     | 0.22           |
| A wave                          | 0.15     | 0.25           |
| LVESVI                          | 0.12     | 0.35           |
| Age                             | 0.12     | 0.36           |
| DBP                             | 0.11     | 0.39           |
| E wave                          | 0.078    | 0.56           |
| LVEDVI                          | 0.07     | 0.59           |
| E/A ratio                       | 0.018    | 0.89           |

CK indicates serum creatine kinase; IZ WMSI, infarct zone WMSI; SBP, systolic blood pressure; A wave, peak velocity of A wave; LVESVI, left ventricular end-systolic volume index; DBP, diastolic blood pressure; E wave, peak velocity of E wave; and LVEDVI, left ventricular end-diastolic volume index.

**Figure 1.** Time course of ejection fraction (EF; A), infarct zone wall motion score index (IZ WMSI; B), left ventricular end-diastolic (LVEDVI; C) and end-systolic (LVESVI; D) volume indexes in patients of group 1 (●●●; DT >130 ms) and group 2 (□□□; DT ≤130 ms). *P < 0.0005 within group vs day 1; §P < 0.00001 between groups. Data are mean±SE. See text for details.
higher in group 2 on days 3 and 7 than in group 1, so that the differences between the 2 groups were less pronounced at 1 and 6 months and statistically not significant (Figure 2).

Relation of DT to Changes in LV End-Diastolic Volume Index

In Figure 3, the change in LV end-diastolic volume index from baseline to 6 months was plotted against the early filling DT. A significant inverse correlation was found between the 2 variables ($r = -0.68; P < 0.0000001$). Similarly, there was a direct relation between changes in LV end-diastolic volume index and peak creatine kinase ($r = 0.66; P < 0.0000001$) and WMSI on admission ($r = 0.48, P < 0.0004$). A weaker although statistical significant correlation was found between DT and the eccentricity index at end diastole at 6 months ($r = 0.48, P < 0.0004$) and end systole ($r = 0.37, P < 0.008$).

The distribution of the DT in patients with and those without LV dilation at 6 months is shown in Figure 4. All but 2 patients with LV dilation had shortened DT ($\geq 130$ ms); in contrast, only 1 patient with a DT $\leq 130$ ms on day 3 did not develop LV dilation at 6 months.

To evaluate the independent contribution of DT to LV dilation, multiple regression analysis was performed. Variables used for analysis were as follows: age, Killip class, EF, LV volume indexes, peak creatine kinase, baseline WMSI, change in WMSI from baseline to 6 months, onset of reperfusion, collaterals, and multivessel disease. For multiple regression analysis, factors showing a $P$ value < 0.1 in univariate analysis were selected. The most important predictor of 6-month LV dilation was a DT $\geq 130$ ms ($P = 0.02$), followed by change in WMSI from baseline to 6 months ($P = 0.03$) and peak creatine kinase ($P = 0.09$).

Follow-Up

Two patients (1 of group 1 and 1 of group 2) underwent repeat coronary angioplasty for early (<24 hours) post-myocardial infarction angina. No patient was lost at 6 months of follow-up. Five patients had recurrent ischemia at 6 months (3 in group 1 and 2 in group 2). No patient developed congestive heart failure.

Reproducibility

There was an excellent agreement between DT measurements made by a single observer at 2 time points (intraobserver variability, $r = 0.93$) and between measurements made by 2 independent observers (interobserver variability, $r = 0.91$).

Discussion

The present study suggests that the assessment of LV filling pattern on Doppler echocardiography provides additional and important information in the setting of AMI, allowing identification of patients at high risk for progressive LV dilation within 6 months after AMI. A restrictive filling pattern, as expressed by a short DT, was the most powerful predictor of LV remodeling, and the degree of LV dilation was related to the severity of impairment of LV filling.

LV Diastolic Function and AMI

Although the predominant diastolic abnormality induced by transient ischemia is an impairment in relaxation, the diastolic filling pattern may change during AMI, resulting in a restrictive filling pattern. Several investigators have in fact observed an upward shift in the pressure-volume curve during AMI or ischemia as a result of an increase in resistance to LV filling or increased chamber stiffness. Furthermore, in a few experimental studies, chamber stiffness increased within 24 hours after AMI, reverting to normal after several days. Increased chamber stiffness may reflect primary
changes in the infarcted myocardium or simply the ventricular filling on a steeper portion of its pressure-volume curve.\(^6\) Therefore, it is not surprising, in view of the aforementioned effects of ischemia on LV compliance, that the filling pattern in patients with large infarcts resembles the filling behavior of those conditions, such as constrictive pericarditis and restrictive cardiomyopathies with “restrictive physiology.”

Doppler echocardiography has been used to assess diastolic function in a variety of clinical settings, including AMI.\(^7\) Doppler indexes are affected by a number of other physiological factors, including heart rate, LV systolic function, and ventricular preload and afterload.\(^8\) However, recent experimental\(^2\) data suggest that early filling DT can quantitatively assess LV chamber stiffness independent of heart rate, contractility, and afterload. Conversely, because in many patients diastolic dysfunction may involve both relaxation and chamber stiffness,\(^9\) and in view of the dynamic nature of LV filling patterns,\(^1\) the ability of the Doppler flow velocity profile as expressed by peak flow velocities (both E and A wave) and their ratio to predict diastolic abnormalities and LV filling pressure is limited.\(^9,15\) The results obtained by pulmonary venous flow velocities and Doppler tissue imaging recordings in a small subset of patients in the present study confirm that E/A ratio is less sensitive and specific than DT in assessing filling pressures.\(^2\)

Restrictive Filling Pattern and LV Remodeling
Previous studies have demonstrated that infarct size is one of the major factors that promotes LV remodeling.\(^2\) On the other hand, the size of the infarct zone has been shown to influence the diastolic filling pattern, with the large infarcts exhibiting a “restrictive” filling pattern.\(^7,13\) Therefore, a short DT, indicative of a restrictive filling pattern, might simply reflect an increasing infarct size and consequently a higher risk of LV dilation.

In agreement with the aforementioned observations, in the current study the extent of asynergy and peak creatine kinase (as estimates of infarct size) were significantly higher in patients with a short DT. Obviously, this may at least partially account for the difference in LV volumes. However, after controlling for infarct size, DT was the most significant independent predictor of LV dilation. Thus DT appears to be a powerful predictor of LV dilation independent of infarct size. It is conceivable that for comparable infarct size, the transmural extent of necrosis will influence the diastolic properties of left ventricle and the propensity to infarct expansion.\(^4\) This speculative hypothesis is supported by the finding on multivariate analysis that the absence of spontaneous recovery of wall motion in the infarct zone was the second most powerful predictor of LV dilation. An alternative hypothesis to explain the relation of DT to ventricular remodeling may not necessarily be related to the extent of myocardial abnormalities that have occurred during the index infarction. DT is inversely related to the LV filling pressure. One may speculate that the LV filling pressure itself can influence subsequent LV dilation due to the changes in wall stress that occur as consequence of the high filling pressures.

Serial Changes of Filling Pattern and LV Remodeling
Experimental and clinical studies have indicated that reperfused AMI leads to abnormal LV stiffness or relaxation that may improve with time.\(^24\) The results of these studies have suggested that reperfusion in AMI is associated with “diastolic stunning,” which is an equivalent of the well-known systolic phenomenon.\(^24\) However, experimental studies were hampered by the fact that periods of occlusion were relatively short, and this is not usually the case in the clinical setting; in the clinical studies, follow-up was performed only in a few patients, and LV volumes were not measured during the short follow-up period. In the present study, in the group of patients with a baseline short DT, the filling pattern changed from a restrictive pattern to a “normal” pattern at 6 months after AMI. The biphasic changes in LV filling pattern detected in the current study are concordant with previous experimental\(^9\) and clinical studies,\(^13\) suggesting an early increase in chamber stiffness that subsequently returns to normal. However, this evolutionary change in filling pattern may be explained by the healing and remodeling process rather than by a gradual recovery in diastolic function after reperfusion. In the early phase of AMI, the infarcted tissue is characterized by edema and cellular infiltrates, causing the tissue to be less extensible. Because of the initial LV stiffness, high left atrial pressures, and rapid increase in LV diastolic pressure during rapid filling, the diastolic filling pattern becomes “restrictive,” contributing to shortening of the DT. As healing progresses, the left ventricle becomes more compliant and dilates, inducing relevant changes in mitral flow velocities, such as prolongation of DT. Thus serial changes in filling pattern after AMI parallel the evolutionary changes in LV dimensions. These findings may provide the critical linkage between restrictive filling pattern and clinical events after AMI observed in some observational studies.\(^11,12,14\)

Study Limitations
One potential limitation of the study is the lack of simultaneous hemodynamic measurements obtained with Doppler examinations. However, we did not feel justified in performing simultaneous cardiac catheterization to correlate hemodynamic data with Doppler patterns because underlying hemodynamic features have been correlated with diastolic filling variables by others\(^23\) who showed a close inverse correlation between DT of early filling and pulmonary capillary wedge pressure irrespective of the filling pattern expressed by the E/A ratio. DT of early filling is partially dependent on age and is determined by the interaction of intrinsic diastolic properties and the alterations in hemodynamic conditions and pericardial restraint related to AMI.\(^11\) However, it is difficult to control all these factors in a clinical study. Age may be unlikely to have significant impact on our results because age distribution was similar between the restrictive and nonrestrictive groups, and patient age in our series was relatively old (average 60 years) and no correlation was found between age and DT. Although the loading conditions of patients were not characterized, in our study no patient was taking diuretic agents or digitalis, whereas all patients were receiving ACE
inhibitor therapy, and no difference in nitrate therapy was found between the restrictive and nonrestrictive group throughout the study period (19% vs 15%, respectively).

Conclusions
This study shows that early noninvasive assessment of transmirtal flow velocity by Doppler echocardiography allows identification of patients at high risk for progressive LV dilation within 6 months after anterior reperfused AMI. A restrictive filling pattern, as expressed by a short ($\leq 130$ ms) DT of early filling, is the most powerful predictor of LV remodeling, and LV dilation is related to the severity of impairment of LV filling. Larger studies are needed to determine the prognostic implications of LV filling patterns in AMI and the role of Doppler ultrasound in this setting.

References

Doppler-Derived Mitral Deceleration Time: An Early Strong Predictor of Left Ventricular Remodeling After Reperfused Anterior Acute Myocardial Infarction

Giampaolo Cerisano, Leonardo Bolognese, Nazario Carrabba, Piergiovanni Buonamici, Giovanni Maria Santoro, David Antoniucci, Alberto Santini, Guia Moschi and Pier Filippo Fazzini

Circulation. 1999;99:230-236
doi: 10.1161/01.CIR.99.2.230

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1999 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/99/2/230

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/