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Doppler-Derived Mitral Deceleration Time as a Strong Prognostic Marker of Left Ventricular Remodeling and Survival After Acute Myocardial Infarction

Results of the GISSI-3 Echo Substudy

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OBJECTIVES The goal of this study was to assess the impact of left ventricular (LV) diastolic filling on remodeling and survival after acute myocardial infarction (AMI).

BACKGROUND Little is known regarding the link between LV filling, its changes over time, and six-month remodeling and late survival in uncomplicated AMI.

METHODS Doppler mitral profile, end-diastolic volume index (EDVi) and end-systolic volume index (ESVi), ejection fraction (EF), and wall motion abnormalities (%WMA) were evaluated in 571 patients from the GISSI-3 Echo substudy at baseline, pre-discharge, and six months after AMI. Patients with baseline early mitral deceleration time (DT) 130 ms were assigned to the restrictive group (n = 147), and those with DT >130 ms to the nonrestrictive group (n = 424).

RESULTS Restrictive group patients had greater baseline ESVi and %WMA and lower EF than nonrestrictive group, and six-month greater LV dilation (EDVi, ESVi: $p < 0.001$ for EDVi and ESVi), smaller decrease in %WMA decrease ($p < 0.01$), and larger EF impairment ($p < 0.008$). Among the restrictive group, patients (n = 56) with pre-discharge persistent restrictive filling (n = 56) showed six-month greater LV enlargement ($p < 0.001$) and EF impairment ($p < 0.009$) than those (n = 91) with reversible restrictive filling. Baseline %WMA and EDVi, together with pre-discharge persistent restrictive filling, predicted severe (>20%) LV dilation. Four-year survival was 93% in nonrestrictive patients versus 88% in the restrictive group ($p < 0.06$), and 93% in pre-discharge reversible restrictive versus 79% in persistent restrictive ($p < 0.0003$). The single best predictor of mortality, by Cox analysis, was pre-discharge persistent restrictive filling (chi-square 14.88).

CONCLUSIONS Left ventricular dilation may occur even after uncomplicated AMI and may be paralleled by an improvement in LV filling. However, a baseline restrictive filling that persists at pre-discharge identifies more compromised patients at higher risk for six-month remodeling and four-year mortality. (J Am Coll Cardiol 2004;43:1646–53) © 2004 by the American College of Cardiology Foundation

In recent years, the issue of left ventricular (LV) remodeling after acute myocardial infarction (AMI) has been extensively investigated. It is now well documented that remodeling is a precursor of heart failure and a strong predictor of mortality (1–3).

Two-dimensional echocardiography is the technique of choice to assess and monitor remodeling after AMI, enabling a precise definition of magnitude and timing of the process (4). In addition, Doppler echocardiography has provided evidence that serial changes in diastolic filling pattern may parallel the evolutionary changes in LV dimensions after AMI (5,6). However, the relationship between LV filling and its changes over time and remodeling after AMI, as well as the prognostic impact of serial assessment of LV filling, have not been systematically addressed in a large population.

We therefore aimed to describe the evolutionary changes in LV filling pattern from the acute to chronic phase after AMI and to assess their impact on six-month remodeling in a large cohort of patients with uncomplicated AMI. In addition, we wanted to determine whether any echocardiographic, particularly Doppler and descriptors, including their serial changes, predicted increased risk for late (four-year) mortality.

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Abbreviations and Acronyms

AMI = acute myocardial infarction
DT = deceleration time of early filling
EDVi = end-diastolic volume index
EF = ejection fraction
ESVi = end-systolic volume index
GISSI = Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto
LV = left ventricle or left ventricular
OR = odds ratio
%WMA = extent of wall motion abnormalities

METHODS

Patients. The subset of 614 patients with confirmed AMI enrolled in the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto (GISSI)-3 Echo substudy (6,7) were recruited for this study. The protocol required serial Doppler echocardiographic studies suitable for qualitative and quantitative analysis at 24 to 48 h (baseline, mean 36 ± 8 h) from symptom onset, at hospital discharge (pre-discharge, mean 12 ± 5 days), and at six months (mean, 194 ± 17 days) after confirmed AMI, in the absence of adverse events and revascularization procedures during follow-up.

Doppler echocardiography. Two-dimensional echocardiography and Doppler ultrasound examination were obtained using commercially available instruments. Left ventricular diastolic filling pattern was obtained by placing the sample volume between the tips of the mitral leaflets in the apical four-chamber view. In each patient, multiple standard parasternal and apical view images were stored on a videotape by a 0.5-inch VHS cassette recorder for further analysis.

The method used for a centralized assessment of technical quality and for centralized quantitative analysis of LV end-diastolic volume index (EDVi) and end-systolic volume index (ESVi), ejection fraction (EF), and extent of wall motion abnormalities (%WMA) has been described elsewhere (6). Mitral regurgitation was detected using color flow Doppler graded as mild, moderate, or severe, according to previously reported criteria that took into account both the width and depth of regurgitant jets in relation to the size of the receiving chamber from multiple views (8) and the size of the jet at the regurgitant orifice (9). In cases of disagreement, the maximal grading was considered for analysis. All measurements were obtained by a single experienced blinded operator (P.L.T.) from three cardiac cycles. The intraobserver variability for all studies in the estimation of end-diastolic and end-systolic volumes by quantitative analysis was blindly assessed in 30 randomly selected patients and was 2.6 ± 2.1% and 3.7 ± 2.5%, respectively, as previously reported (6,7). On the basis of the reproducibility analysis, an increase >5% in EDVi was considered as progressive LV dilation, and a >20% increase as severe LV dilation.

From mitral Doppler tracings, the following variables were measured: peak flow velocity of early filling (E), peak

Table 1. Baseline Clinical and Doppler Echocardiographic Characteristics of the Study Population According to Baseline DT

	All Patients (n = 571)	DT ≤130 ms Restrictive (n = 147)	DT >130 ms Nonrestrictive (n = 424)	p Value
Male	471 (82%)	116 (79%)	357 (84%)	
Age	61 ± 11	60 ± 13	61 ± 11	
Killip class	1.25 ± 0.5	1.12 ± 0.9	1.57 ± 0.5	
Diabetes	83 (14%)	18 (12%)	65 (15%)	
Hypertension	162 (28%)	41 (28%)	121 (28%)	
Anterior infarct	174 (30%)	75 (52%)	99 (23%)	< 0.001
Previous AMI	63 (11%)	18 (12%)	45 (11%)	
Peak CK value	2,312 ± 2,028	3,180 ± 2,816	1,837 ± 1,548	< 0.001
Randomized treatments				
Lisinopril	289 (50.6%)	73 (49.6%)	216 (50.9%)	
Nitrate	287 (50.2%)	76 (51.7%)	211 (49.8%)	
Thrombolysis	405 (71%)	102 (69%)	303 (71%)	
Moderate-to-severe MR	32 (6%)	13 (8.8%)	19 (4.5%)	0.05
HR (beats/min)	71 ± 7	72 ± 8	70 ± 9	
EDVi (ml/m^2)	79 ± 20	81 ± 21	78 ± 19	
ESVi (ml/m^2)	42 ± 15	46 ± 17	41 ± 14	< 0.01
EF (%)	47 ± 7	44 ± 7	48 ± 7	< 0.001
%WMA	25 ± 15	32 ± 15	23 ± 14	< 0.001
Peak E (cm/s)	65 ± 18	71 ± 20	63 ± 17	< 0.001
Peak A (cm/s)	64 ± 18	60 ± 18	66 ± 18	< 0.005
E/A	1.09 ± 0.5	1.3 ± 0.6	1.0 ± 0.4	< 0.005
DT (ms)	159 ± 41	117 ± 10	173 ± 39	< 0.001

Values are mean ± SD or number (%).

AMI = acute myocardial infarction; CK = creatine kinase; DT = early mitral deceleration time; EDVi = end-diastolic volume index; EF = ejection fraction; ESVi = end-systolic volume index; HR = heart rate; MR = mitral regurgitation; WMA = wall motion abnormalities.

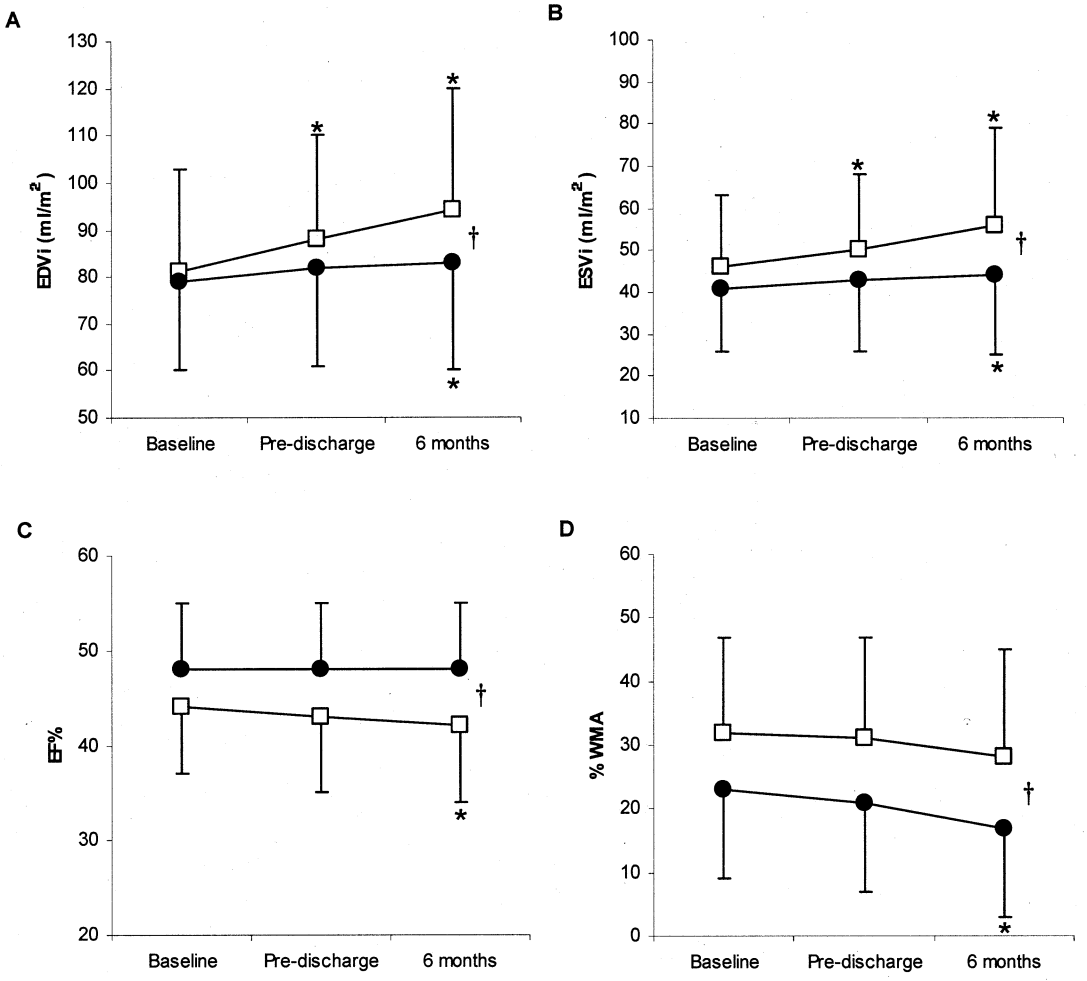

Figure 1. Changes in end-diastolic volume index (EDVi) **(A)**, end-systolic volume index (ESVi) **(B)**, ejection fraction (EF) **(C)**, and extent of wall motion abnormalities (%WMA) **(D)** during the follow-up in patients with baseline deceleration time ≤130 ms **(open squares)** and >130 ms **(solid circles)**. *$p < 0.01$ within group versus baseline; ‡$p < 0.001$ interaction time per group. Data are mean ± SE.

flow velocity at atrial contraction (A), their ratio (E/A), and deceleration time (DT) of early filling. Each Doppler profile was randomly analyzed by digital tracing by the same operator (P.L.T.), blinded to the two-dimensional data at a different time with respect to LV volumes, and measurements were calculated from three to five consecutive cardiac cycles. Intraobserver variability for all Doppler variables ranged from 1.8% to 2.4%.

According to baseline DT, irrespective of filling profile, patients were assigned to one of two groups: restrictive (with DT ≤130 ms) or nonrestrictive (with DT >130 ms). We chose this value because it unequivocally identifies patients with restrictive filling and worse prognosis after AMI (10,11,12) and also because it is very close to the value (132 ms) identified by a specifically used receiver operating characteristic curve analysis as the one predicting some degree of progressive LV dilation with the greatest accuracy.

Follow-up. The vital status at four years was sought for all patients through the census office of their towns of residence by means of prepaid return-mail questionnaire. Information on death from any cause at the four-year follow-up was available for 541 (95%) of the 571 patients representing the final study population.

Statistical analysis. All continuous data are given as mean ± SD. Differences between patients were compared by unpaired *t* testing and frequency of parameters by the chi-square test with Yates correction. Differences in Doppler-echocardiographic measurements between groups and changes over time within each group (time effect) as well as any interaction (different trends over time between groups) were assessed by repeated-measures analysis of variance (general linear model procedure of the SAS program). Multivariate logistic regression analysis, including in the model demographic, clinical, and Doppler echocardiographic variables, was performed to identify independent correlates of the changes in EDVi.

Cardiac survival curves were computed with the Kaplan-Meier method. Clinical, echocardiographic, and Doppler variables were compared for their ability to predict all-cause mortality by the Cox regression model. Variables that were significantly different between survivors and nonsurvivors by

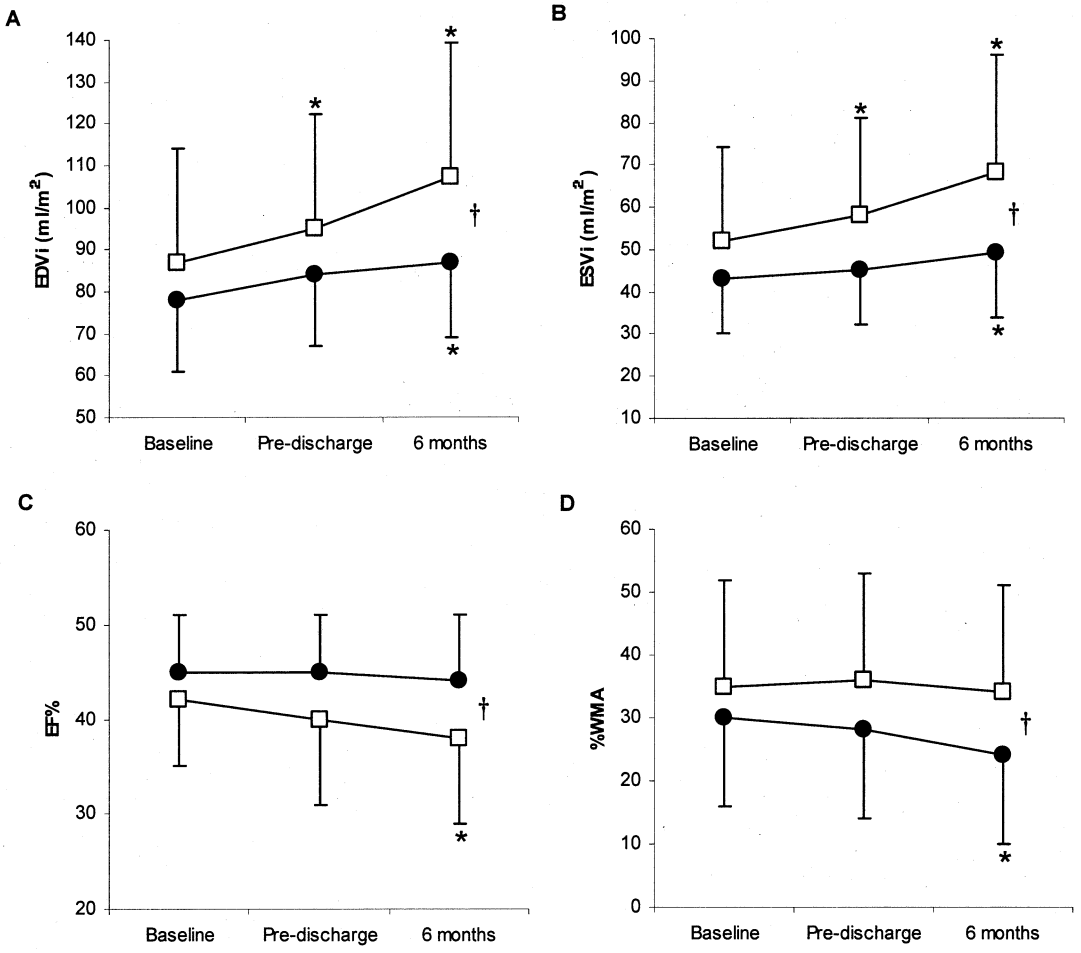

Figure 2. Changes in end-diastolic volume index (EDVi) **(A)**, end-systolic volume index (ESVi) **(B)**, ejection fraction (EF) **(C)**, and extent of wall motion abnormalities (%WMA) **(D)** during the follow-up in patients of the restrictive group with pre-discharge persistent short (≤130 ms) deceleration time (DT) **(open squares)** and reversible short (>130 ms) DT **(solid circles)**. *$p < 0.01$ within group versus baseline; ‡$p < 0.001$ interaction time per group. Data are mean ± SE.

unpaired *t* testing or by chi-square testing were included in the Cox model in stepwise fashion. Values of $p < 0.05$ were considered significant.

RESULTS

Patients with fusion of early and late mitral flow velocity (n = 19), mitral stenosis (n = 3), atrial fibrillation (n = 4), or with incomplete Doppler follow-up (n = 17) were excluded from the subset of patients enrolled in the GISSI-3 Echo substudy (6). The remaining 571 patients with echo-Doppler study suitable for analysis at baseline, at pre-discharge, and at six months after AMI represented the final study population.

There were 174 patients (30%) with anterior, 216 (38%) with inferior, and 113 (20%) with non–Q-wave AMI. Baseline clinical characteristics of the population enrolled in this substudy were comparable to those of the general population of the GISSI-3 trial discharged alive and followed-up with echocardiographic examination at six months (13).

As previously demonstrated (6), EDVi and ESVi slightly, but significantly, increased in the whole population ($p < 0.01$, for both), %WMA decreased ($p < 0.01$), and EF remained unchanged (p = NS). Early (from 64 ± 18 cm/s to 62 ± 18 cm/s) and late (from 64 ± 18 cm/s to 65 ± 19 cm/s) peak flow velocity and their ratio (from 1.09 ± 0.5 to 1.01 ± 0.5) did not change during the follow-up, while DT significantly increased from 159 ± 41 ms to 179 ± 38 ms ($p < 0.001$).

Baseline demographic, clinical, and Doppler echocardiographic characteristics of the study population according to baseline DT are reported in Table 1. There were 147 patients (26%) in the restrictive group, and 424 patients (74%) in the nonrestrictive group. There was no significant difference between the two groups with respect to age, sex, traditional risk factors, and treatments by randomization. In contrast, patients in the restrictive group, as compared with those in the nonrestrictive group, had larger infarcts, measured by creatine kinase value, and were more likely to have anterior infarct. Furthermore, they had similar baseline EDVi (p = NS) but greater ESVi ($p < 0.01$) and %WMA

Table 2. Independent Predictors of Dilation (>5%)

Variables	Odds Ratio	Wald Chi-Square	p Value
Baseline EDVi	0.96* (0.95–0.97)	35.10	0.0001
Baseline DT ≤130 ms	2.38† (1.47–3.62)	13.34	0.0003
Baseline %WMA	1.02* (1.00–1.03)	7.21	0.007

*Continuous variable; †categorical variable.
Abbreviations as in Table 1.

Table 3. Independent Predictors of Severe (>20%) Dilation

Variables	Odds Ratio	Wald Chi-Square	p Value
Predischarge persistent DT ≤130 ms	3.06† (1.56–5.97)	35.93	0.0001
Baseline EDVi	0.95* (0.93–0.96)	10.74	0.001
Baseline %WMA	1.02* (1.00–1.04)	8.07	0.004

*Continuous variable; †categorical variable.
Abbreviations as in Table 1.

($p < 0.001$), with lower EF ($p < 0.001$). Both baseline %WMA and EF were poorly correlated with DT ($r = -0.26$ and 0.26, respectively). Moreover, no relationship was observed between EDVi and DT ($r = -0.05$).

Changes in two-dimensional and Doppler variables according to baseline DT. During follow-up, EDVi and ESVi increased and %WMA decreased in both groups ($p < 0.01$, for both). However, patients with short DT showed a greater increase in EDVi and ESVi ($p < 0.001$, for both) and a smaller decrease in %WMA ($p < 0.01$) than those with ≥130 ms in DT (Fig. 1), despite a significant and greater ($p < 0.001$) prolongation in DT at six months (from 117 ± 10 ms to 156 ± 31 ms in the restrictive group; from 174 ± 38 ms to 186 ± 37 ms in the nonrestrictive group). Furthermore, a progressive impairment in EF was observed only in patients with restrictive filling ($p < 0.008$), with no changes in those with nonrestrictive filling (p = NS) (Fig. 1). Left ventricular dilation occurred in as many as 82% of patients with baseline restrictive filling versus only 40% of patients with nonrestrictive filling ($p < 0.001$). As previously reported (6), the six-week treatment assigned by randomization (lisinopril or glyceril trinitrate) did not influence LV volumes over time.

Pre-discharge LV filling patterns and remodeling. Among the 147 patients with baseline restrictive filling, 56 (38%) at pre-discharge showed a persistent short DT, whereas the remaining 91 patients (62%) showed a significant prolongation of DT (>130 ms). Baseline DT was similar in the two groups (p = NS); on the contrary, patients with persistent restrictive filling, compared with those with reversible restrictive filling, had both at baseline and at pre-discharge larger volumes (EDVi: 87± 27 ml/m vs. 78 ± 17 ml/m^2; ESVi: 52 ± 22 ml/m vs. 43 ± 13 ml/m^2, $p < 0.001$), lower EF (42 ± 7% vs. 45 ± 6 %, $p < 0.001$), and greater %WMA (35 ± 17% vs. 30 ± 14%, $p < 0.002$) (Fig. 2). Moreover, despite a subsequent significant improvement in DT in the late follow-up from 116 ± 10 to 147 ± 31, at six months they showed a far greater EDVi and ESVi enlargement ($p < 0.001$), greater impairment in EF ($p < 0.009$), and less recovery of %WMA ($p < 0.004$) than those with pre-discharge reversible restrictive filling (Fig. 2). Severe (>20%) six-month dilation occurred in 28 patients (50%) with pre-discharge persistent restrictive filling versus 30 patients (32%) with reversible restrictive filling ($p < 0.04$).

Predictors of six-month LV dilation. At multivariate analysis, among several demographic, clinical, and Doppler echocardiographic variables, baseline %WMA (odds ratio [OR]: 1.02), EDVi (OR: 0.96), and DT ≤130 ms (OR: 2.38) independently predicted progressive (>5%) six-month dilation (Table 2). Furthermore, when changes in %WMA, EF, and DT from baseline to pre-discharge were included in the analysis, baseline EDVi (OR: 0.95) and %WMA (OR: 1.02), together with pre-discharge persistent short (≤130 ms) DT (OR: 3.06), emerged as independent predictors of severe (>20%) late LV dilation (Table 3).

Outcome. During the four-year follow-up period, 47 patients died, with an estimated overall survival of nearly 91%. On univariate analysis, several demographic, clinical, and mitral Doppler variables were found to be predictive of fatal outcome (Table 4). Of note, no changes in EDVi or EF between baseline and six-month evaluation emerged as significantly associated with prognosis. When multivariate Cox analysis was performed, only pre-discharge persistent restrictive filling (i.e., persistent DT ≤130 ms), age, and baseline ESVi emerged as independent predictors of fatal outcome, with persistent restrictive filling being the single best predictor (chi-square 14.88) (Table 5). The same variables were independent predictors of mortality after removing patients with prior AMI, with persistent restrictive filling still remaining the best independent predictor (chi-square 9.40; OR, 2.75; 95% confidence interval, 1.32 to 5.73; p = 0.002). The four-year all-cause mortality rate was 12% and 7% ($p < 0.06$) in patients with baseline DT ≤130 ms and >130 ms, respectively, and 20% in patients with

Table 4. Univariate Analysis: Predictors of Fatal Outcome

Variables	Global Chi-Square	p Value
Clinical data		
Age	12.9	0.0003
Anterior AMI	0.10	0.74
Peak CK (U/l)	3.42	0.042
Baseline echocardiographic data		
EDVi (ml/m^2)	3.97	0.046
ESVi (ml/m^2)	9.81	0.0017
EF (%)	11.7	0.0006
%WMA	6.92	0.0085
DT <130 ms	4.41	0.035
Baseline vs. predischarge		
Persistent DT <130 ms	14.88	0.0001
Baseline vs. six-month		
Change in EDVi >20%	0.63	0.42
Change in EDVi >5%	0.11	0.73
Change in EF	2.48	0.11

Abbreviations as in Table 1.

Table 5. Multivariate Cox Proportional Hazard Model

Variables	Chi-Square	p Value	RR (95% CI)
Predischarge persistent vs. reversible restrictive filling	14.88	0.0001	2.96 (1.55–5.21)
Age	12.60	0.0004	2.78 (1.47–5.94)
ESVi	6.45	0.011	2.18 (1.18–4.09)

CI = confidence interval; ESVi = end-systolic volume index; RR = relative risk.

pre-discharge persistent restrictive filling versus 7% in those with reversible restrictive filling ($p < 0.0003$). Survival free of death according to both baseline DT and its evolution at pre-discharge is reported in Figure 3.

DISCUSSION

The present study in a large cohort of post-AMI patients demonstrates, first, that an early restrictive filling pattern, as expressed by a short DT, identifies patients who are ultimately candidates to progressive LV dilation and dysfunction. Furthermore, we found that the filling pattern may change within a few days after AMI and that the reversibility of a restrictive filling at pre-discharge is associated with a less pronounced six-month remodeling and a more favorable late outcome, whereas the persistence of a restrictive filling is the most powerful independent predictor of severe dilation and late mortality. Thus, we confirm that a

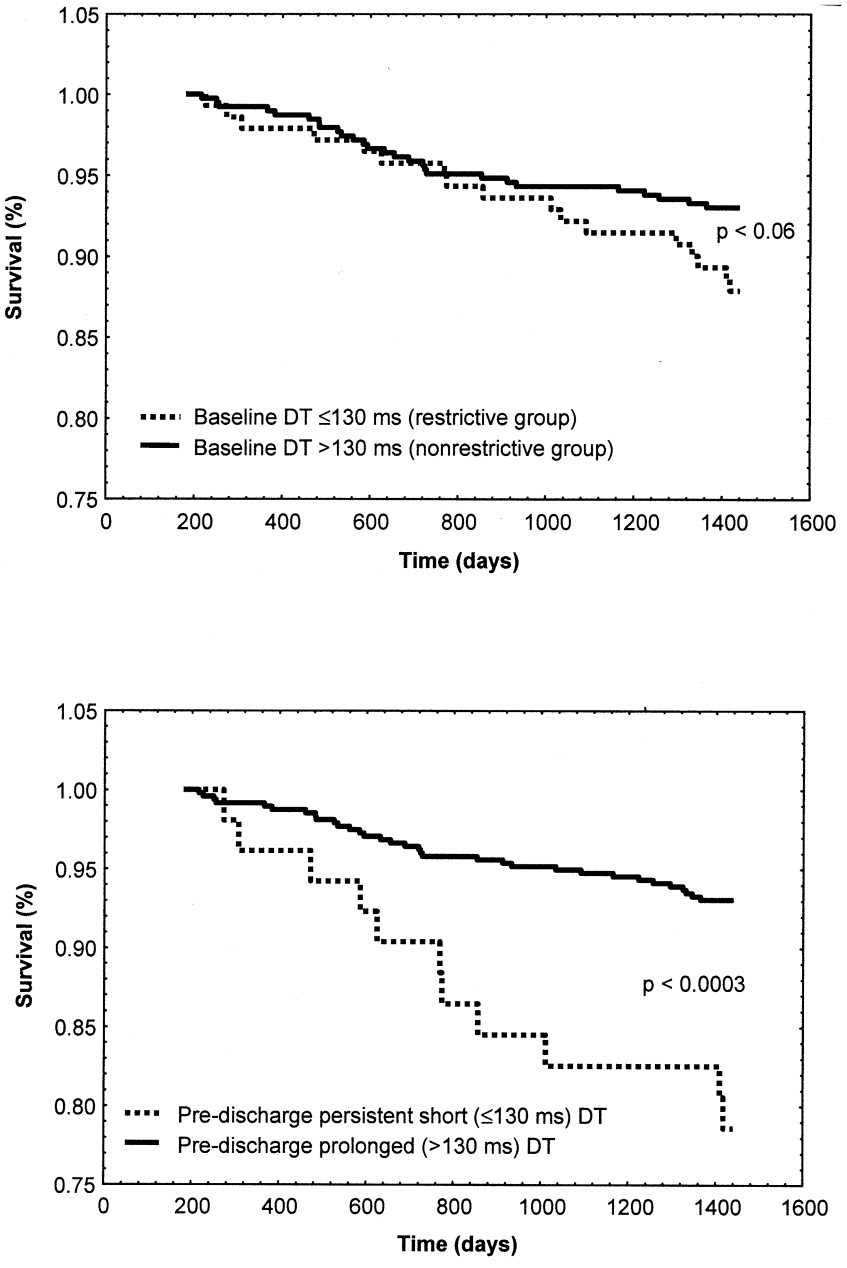

Figure 3. Cumulative survival rates for all-cause mortality according to baseline deceleration time (DT) **(top)** and pre-discharge DT **(bottom)**.

short DT at any time provides additional information, even in the setting of uncomplicated AMI and identifies patients at risk of progressive LV dilation. We also demonstrate, for the first time, that monitoring the evolutionary changes of a baseline restrictive filling pattern clearly identifies those patients at worst risk of remodeling and poor survival.

LV diastolic filling and remodeling after AMI. Recently, increasing attention has been devoted to diastolic function after AMI, and there is growing evidence indicating a strong association between diastolic dysfunction and adverse outcome (5,6,10–12,14).

Several studies have shown that a short DT, irrespective of the ratio of early and late filling velocity (E/A ratio), is strongly related to pulmonary capillary wedge pressure (15,16), and is an ideal parameter for the serial assessment of LV diastolic function in post-infarct patients (10,17). For this reason, we used DT as a means of assessing LV filling, irrespective of filling pattern, and we classified those with DT ≤130 ms as restrictive. In line with previous studies (5,10,17,18), we found that patients with baseline restrictive filling were more likely to have large infarcts, as expressed by a more extensive asynergy and LV systolic dysfunction. During follow-up, these patients showed greater LV enlargement, less recovery of %WMA, and a further impairment of EF (not seen in nonrestrictive). Of note, no relationship was observed between EDVi and DT, and both baseline %WMA and EF were poorly correlated with DT.

It has also been demonstrated that LV filling variables may change during the follow-up after AMI, particularly in patients with initial restrictive pattern (10,17,18). Indeed, in our study, as many as 91 of 147 patients (62%) with baseline restrictive filling changed significantly toward a nonrestrictive filling at pre-discharge. This favorable evolution of LV filling was paralleled by an attenuation of unfavorable remodeling in the late follow-up, not documented in patients with persistent restrictive filling. Indeed, pre-discharge persistent restrictive filling emerged as the best predictor of severe six-month dilation.

Mechanistic insight. In both animal and clinical studies, it has been demonstrated that DT is inversely related to LV filling pressure and to chamber stiffness (15,16,19) and that a short DT almost invariably indicates elevated LV filling pressure, which is a strong predictor of severe cardiovascular events (10,15,17,18). The increased wall stress after AMI (19) induces ventricular enlargement, which is ultimately aimed at reducing filling pressure. In our study, ventricular dilation was paralleled by a progressive prolongation of DT. Remodeling after AMI should then be viewed as a "compensatory" mechanism acting to preserve normal filling pressure. One could even speculate that the lower the initial DT (i.e., the higher the pulmonary capillary wedge pressure), the greater the degree of dilation required to restore normal filling pressure. Actually, in our study, EDVi, although similar at baseline in the two groups, increased more at pre-discharge in patients with short DT, and continued to increase in greater proportion. Given the aforementioned inverse relation between DT and filling pressure, a significant prolongation of a short DT would reflect the effective normalization of filling pressure; in this setting, a further "compensatory" dilation should not be required. Indeed, patients with pre-discharge reversible restrictive filling did not further dilate substantially in the late follow-up. By contrast, the persistence of restriction in the early follow-up reflects persistence of elevated filling pressure despite the initial but insufficient dilation, and represents a stimulus for further more severe and often "noncompensatory" late dilation.

Persistent restrictive filling and long-term outcome. Previous studies have shown that a restrictive filling and, specifically, a short DT is an independent predictor of adverse outcome in patients with AMI (5,12,17). In our study, a short baseline DT was not an independent predictor of death, although the four-year all-cause mortality rate was 12% and 7% ($p < 0.06$) in patients with baseline DT ≤130 ms and >130 ms, respectively. The exclusion of patients with adverse events during the six-month echocardiographic follow-up, the higher baseline systolic function in the restrictive group of our population (EF = 44%) compared with the other studies (40% or less), and our use of total mortality as the sole outcome event may account for this apparent discrepancy. On the other hand, we found the persistence at pre-discharge of a short DT (i.e., of restrictive filling) to be the most powerful independent predictor of four-year all-cause mortality. This is not completely surprising because a short DT was the best predictor of severe six-month dilation, thus providing evidence that severe remodeling is closely related to LV restrictive filling, both of which are strongly related to prognosis.

Study limitations. A successful reperfusion is important for recovery of global and regional systolic function, and could also influence the recovery of diastolic function. Routine angiographic studies to assess artery patency were not performed. In recent studies, however, patency of the infarct-related artery did not emerge as an independent predictor of either a short DT or of remodeling after AMI (7,18).

We included only uncomplicated low-risk patients, all survivors at six-month follow-up without major cardiovascular events. Indeed, the four-year mortality was <9% in this echocardiographic substudy, compared with 20.8% in the GISSI-3 study as a whole. Therefore, the results of the present study may not be applicable to all post-AMI patients but only to those optimally treated and without clinical complications and revascularization procedures. Nevertheless, it is reasonable to suppose that inclusion of unselected post-infarction patients would only have strengthened the predictive power of a short DT.

Conclusions. The present study demonstrates that LV dilation may occur even after uncomplicated AMI and may be paralleled by a significant prolongation of mitral DT, indicating an improvement in LV filling. A short baseline DT that persists at pre-discharge clearly identifies more

compromised patients at higher risk for six-month remodeling and four-year mortality. Thus, early assessment of mitral DT by Doppler echocardiography and, particularly, monitoring of its changes in the in-hospital phase add significant information to the commonly used indexes of global and regional function, and represent a very easy and cost-effective tool to accurately stratify even low-risk patients after AMI.

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