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# Prognostic Implications of Restrictive Left Ventricular Filling in Reperfused Anterior Acute Myocardial Infarction

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**OBJECTIVES** We sought to assess the relative prognostic role of a restrictive left ventricular (LV) filling pattern after a first anterior acute myocardial infarction (AMI) in patients treated with primary percutaneous transluminal coronary angioplasty (PTCA).

**BACKGROUND** In thrombolized patients, a short Doppler-derived mitral deceleration time (DT) of early filling is a powerful independent predictor of heart failure and death. However, it is still unknown whether the outcome of patients with AMI with a short DT may be improved by a more aggressive treatment.

**METHODS** In 104 patients, two-dimensional and Doppler echocardiograms were obtained three days after the index AMI. Coronary angiography was performed in all patients one and six months after PTCA. The patients were classified into two groups according to the DT duration: group 1 (n = 34) with DT ≤130 ms and group 2 (n = 70) with DT >130 ms. All patients were followed-up for a mean (±SD) period of 32 ± 10 months.

**RESULTS** During the follow-up period, 14 patients (13%) were admitted to the hospital for congestive heart failure, and 9 patients (9%) died. All cardiac deaths (n = 7) occurred in group 1. The survival rate at mean follow-up was 79% in group 1 and 97.2% in group 2 (p = 0.003). Multivariate Cox analysis showed that only age and restrictive filling were independent predictors of event-free survival. Furthermore, when survival with no cardiovascular events was analyzed, a short DT still emerged as the most powerful independent predictor.

**CONCLUSIONS** Patients with a restrictive LV filling pattern early after anterior AMI have a poor clinical outcome, even if treated with primary PTCA. (J Am Coll Cardiol 2001;37:793–9) © 2001 by the American College of Cardiology

Several variables to predict a poor prognosis after acute myocardial infarction (AMI) have been proposed. Among these, the extent of left ventricular (LV) systolic dysfunction and LV end-systolic volume are two major predictors of survival after AMI (1–3). However, the contribution of abnormal LV filling to long-term prognosis has not been well defined, and the prognostic impact of early assessment of LV diastolic filling in the very early phases of AMI has been poorly investigated (4–7). Doppler echocardiography has provided a rapid, feasible, simple and noninvasive method of assessing LV diastolic filling (8). 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 predict an adverse outcome in various cardiac diseases (9). Preliminary studies suggest that Doppler variables allow identification of patients at high risk of progressive LV dilation after AMI (10), providing independent prognostic information also in the setting of thrombolyzed AMI (4–7). However, no information is available on diastolic filling and long-term outcome in patients with AMI treated with a more aggressive treatment, such as early mechanical reperfusion. Furthermore, there are few data on the relation between LV filling pattern, LV remodeling and prognosis after MI (6). Thus, the primary end point of this prospective study was to determine the prognostic significance of the early occurrence of a LV restrictive filling pattern in a consecutive series of patients with anterior AMI successfully treated with primary percutaneous transluminal coronary angioplasty (PTCA). A secondary end point was to examine the long-term (two years) serial changes of LV systolic function and the geometry of patients with restrictive LV filling.

## METHODS

**Patients and study protocol.** We prospectively studied 104 patients with a first anterior AMI selected among 241 patients consecutively referred to the catheterization laboratory of Careggi Hospital for emergency PTCA between May 1996 and June 1997. The study inclusion criteria were: 1) a confirmed first anterior AMI; 2) successful primary PTCA (defined as Thrombolysis in Myocardial Infarction [TIMI] flow grade 3 and residual stenosis <30%) within 6 h of the onset of symptoms or between 6 and 24 h if there was evidence of continuing ischemia. Exclusion criteria included atrial fibrillation, significant valvular heart disease and life-limiting noncardiac disease. No upper age limit was used. Of the 125 patients selected for the study, 14 (11%)

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**Abbreviations and Acronyms**

| | |
|---|---|
| A | = peak velocity of late diastolic filling wave |
| AMI | = acute myocardial infarction |
| DT | = (early filling) deceleration time |
| E | = peak velocity of early diastolic filling wave |
| LV | = left ventricular |
| LVEF | = left ventricular ejection fraction |
| PTCA | = percutaneous transluminal coronary angioplasty |
| TIMI | = Thrombolysis In Myocardial Infarction trial |
| WMSI | = wall motion score index |

were excluded because of an inadequate quality of the echocardiographic images and mitral Doppler tracings at baseline; an additional 7 patients (6%) did not adhere to the follow-up protocol. Thus, 104 patients (79 men and 25 women, mean age 61 ± 12 years) were enrolled in the study. The hospital's Ethics Committee approved the research protocol, and informed consent was obtained from each patient by one of the investigators. All patients underwent echocardiographic assessment of LV filling patterns, volumes and systolic function at three days, and repeat coronary angiograms were obtained one and six months after primary PTCA, or earlier if there was a clinical indication. In a subgroup of 48 patients, a serial echocardiographic bi-dimensional examination was obtained at 1, 6 and 24 months. Details pertaining to acquisition and analysis of Doppler echocardiographic and angiographic data are reported elsewhere (10,11).

**Definitions and outcome measures.** A DT >130 ms was defined as nonrestrictive, and ≤130 ms was defined as restrictive. This cutoff point has been shown to be consistent with restrictive hemodynamic data and a powerful independent predictor of unfavorable outcome after AMI and idiopathic dilated cardiomyopathy (4–8). To avoid the effects of acute ischemia on LV filling patterns (12), we chose to measure baseline DT on day 3 after the index AMI.

The outcome events were death from any cause, hospital admission for congestive heart failure, nonfatal myocardial infarction and total events (all of the aforementioned). All deaths were considered to be of cardiac origin unless a noncardiac etiology was established at autopsy. The diagnosis of heart failure was based on the presence of at least two of the following criteria: bibasilar pulmonary rales, dyspnea, a third heart sound or radiographic evidence of pulmonary congestion (13). The diagnosis of AMI was established by the presence of a clinical episode of prolonged chest pain and a rise in serum cardiac enzymes levels to at least twice the upper limit of normal, or by the appearance of one or more new pathologic Q waves. For purposes of analysis, only one event (the most serious) was tabulated for each patient. After hospital discharge, patients were referred to their private physician, who regulated therapy. No attempt was made to standardize therapy. All patients were asked to return to our outpatient clinic for evaluation by one of the investigators three months after discharge and annually thereafter. For those patients not returning to the clinic at the designated time, follow-up information was collected by telephone interview.

**Statistical analysis.** Continuous data are expressed as the mean value ± SD. Baseline data were compared by means of the chi-square test for categoric variables and the unpaired *t* test for continuous variables. Analysis of variance with the Tukey post hoc test was used to analyze repeated measures.

Event-free survival curves for all major adverse cardiac events, and specifically for mortality, were constructed by using the Kaplan-Meier method. Statistical differences between curves were assessed with the log-rank or Mantel-Haenszel test.

Multivariate analysis with the Cox proportional hazards regression model was used to test for the potential role of LV restrictive filling on clinical outcome. The variables examined included age, gender, diabetes, hypertension, time from onset of symptoms and treatment, multivessel disease, collateral flow (Rentrop grade 2 or 3), enzymatic infarct size, heart rate and systolic blood pressure at hospital admission and cardiogenic shock. The echocardiographic variables included LV volume indexes, LV ejection fraction (LVEF) and wall motion score index (WMSI). The odds ratio and relative risk with 95% confidence intervals were calculated. A p value <0.05 was considered to be statistically significant in all cases. Finally, we also analyzed the data according to a modified stepwise procedure in which the individual factors were included in the model in the same order in which they are actually considered in clinical practice. Statistical analyses were performed with Statistica version 4.5 for Windows (StaSoft, Inc., 1993) and with SPSS version 7.0 for Cox regression analysis.

## RESULTS

**Patient characteristics.** The clinical, angiographic and echocardiographic characteristics of the patients with (group 1; n = 34) and without (group 2; n = 70) restrictive filling are shown in Table 1. Patients with restrictive filling had a longer time from the onset of symptoms to reperfusion, a higher heart rate on hospital admission and a significantly larger enzymatic infarct size, and they more often showed signs or symptoms of LV heart failure during the hospital course. In addition, they had larger LV end-systolic and end-diastolic volume indexes, a lower LVEF and a higher LV mass and WMSI, as compared with patients without restrictive filling.

**Angiographic results.** The minimal lumen diameter of the lesion increased from 0 ± 0.2 mm at baseline to 3.2 ± 0.4 mm after PTCA in group 1 and from 0 ± 0.2 to 3.1 ± 0.3 mm in group 2. At one month, the angiographic patency rate (TIMI flow grade 3) was 97% in group 1 and 98% in group 2. The minimal diameter of the lesion was 3.1 ± 0.4 mm in group 1 and 2.9 ± 0.6 mm in group 2 (p = 0.08).

**Table 1.** Baseline Characteristics of the Study Group

| | Total Group (n = 104) | DT ≤130 ms (n = 34) | DT >130 ms (n = 70) | p Value |
|---|---|---|---|---|
| Age (yrs) | 61 ± 12 | 62 ± 12 | 61 ± 12 | 0.56 |
| Male | 79 (76%) | 26 (76%) | 53 (76%) | 1 |
| Diabetes mellitus | 21 (20%) | 7 (20%) | 14 (20%) | 1 |
| Hypertension | 32 (31%) | 10 (29%) | 22 (31%) | 0.83 |
| Hyperlipidemia | 29 (28%) | 7 (20%) | 22 (31%) | 0.24 |
| Killip class | 1.8 ± 1 | 2.3 ± 1 | 1.5 ± 1 | 0.04 |
| Heart rate (beats/min) | 81 ± 16 | 89 ± 16 | 77 ± 15 | 0.0003 |
| Onset of reperfusion (min) | 166 ± 87 | 190 ± 93 | 153 ± 82 | 0.0001 |
| Peak CK (U/liter) | 3,516 ± 2,863 | 5,467 ± 2,938 | 2,583 ± 2,319 | 0.04 |
| Multivessel CAD | 50 (48%) | 17 (50%) | 33 (47%) | 0.77 |
| Collateral vessels (grade ≥2) | 8 (8%) | 2 (6%) | 5 (7%) | 0.89 |
| EDVI (ml/m$^2$) | 73 ± 20 | 81 ± 18 | 69 ± 16 | 0.0008 |
| ESVI (ml/m$^2$) | 43 ± 13 | 56 ± 13 | 38 ± 13 | 0.0001 |
| Ejection fraction (%) | 40 ± 9 | 32 ± 7 | 45 ± 7 | 0.0001 |
| Wall motion score index | 2.25 ± 0.4 | 3 ± 0.2 | 2 ± 0.4 | 0.0001 |
| Peak E-wave velocity (cm/s) | 59 ± 5 | 62 ± 19 | 59 ± 15 | 0.38 |
| Peak A-wave velocity (cm/s) | 63 ± 18 | 64 ± 21 | 62 ± 17 | 0.60 |
| E/A ratio | 1 ± 0.4 | 1 ± 0.6 | 1 ± 0.4 | 1 |
| E-wave DT (ms) | 162 ± 16 | 119 ± 16 | 187 ± 37 | 0.0001 |
| Left atrium (mm) | 33 ± 8 | 34 ± 4 | 31 ± 7 | 0.12 |
| Mass index (g/m$^2$) | 86 ± 18 | 92 ± 16 | 83 ± 20 | 0.02 |

Data are presented as the mean value ± SD or number (%) of patients.
CK = creatine kinase; CAD = coronary artery disease; DT = deceleration time; EDVI = end diastolic volume index; ESVI = end systolic volume index.

At six months, the patency of the infarct-related artery was 92% in both groups. No significant difference was found in minimal lumen diameter and restenosis rate (>50%) between the two groups. Likewise, no significant difference in the distribution of TIMI flow grade 3 at one- and six-month follow-up was found between the two groups.

**Outcome.** Patients were followed up for a period of 32 ± 10 months (range 1 to 43). During this period, one patient had a nonfatal myocardial infarction, 14 (13%) were admitted to the hospital for congestive heart failure and 9 (9%) died. Of the deaths, seven were cardiac (7%) and two noncardiac (2%). Of the seven patients with cardiac deaths, five died from refractory congestive heart failure and two had sudden death. Overall, there were 24 hard events (23%) (all-cause mortality, repeat AMI or congestive heart failure).

Cardiac death occurred only among patients with a restrictive filling pattern. Hospital readmission for heart failure occurred in 11 patients (32%) with and 3 patients (4%) without a LV restrictive filling pattern ($p < 0.0001$). Reinfarction occurred in only one patient in group 1. Furthermore, target vessel revascularization for recurrent ischemia was performed in two patients and additional revascularization procedures for multivessel disease were performed in five patients in group 2, but in none of the patients in group 1. Overall, cumulative events (i.e., death, reinfarction or congestive heart failure) were significantly higher in group 1 than in group 2 (38% vs. 7%, $p = 0.001$). In addition, major cardiac adverse events (i.e., death, reinfarction and target vessel revascularization) differed significantly between the two groups (24% vs. 6%; $p < 0.008$). The two-year cumulative mortality rate was 21% in the restrictive group and 3% in the nonrestrictive group ($p = 0.003$). Moreover, the cumulative two-year incidence rate of all hard events was 39% in group 1 and 6% in group 2 ($p = 0.00004$) (Fig. 1).

On univariate analysis, age, time to reperfusion, end-systolic volume, LVEF and restrictive filling were all predictive of a fatal outcome (Table 2). When multivariate Cox analysis in a forward stepwise regression procedure was performed, only age and restrictive filling emerged as independent predictors of survival (Table 2). Furthermore, when survival with no cardiovascular events (death, reinfarction and hospital admission for heart failure) was analyzed, a short DT still emerged as the most powerful independent predictor (Table 2).

Multivariate Cox analysis was also performed according to a modified procedure to assess the incremental value of restrictive filling with respect to clinical, echocardiographic and angiographic variables for predicting fatal outcome. Four different models were used (Table 3): model 1 includes only the clinical variables. The addition of echocardiographic data reflecting systolic dysfunction (model 2) improved the global chi-square value (an index of predictive power) from 8.3 to 19.3. The addition of angiographic variables (model 3) did not improve this value. In contrast, the addition of restrictive filling (model 4) significantly improved the global chi-square value from 20.1 to 28.5. A similar trend was observed when the same procedure was applied for predicting cumulative cardiac events (Table 3).

**Serial changes in LV systolic function and geometry.** The subgroup of 48 patients (15 with and 33 without a restrictive LV filling pattern) who underwent serial echocardiographic studies had baseline, clinical, echocardiographic and angiographic characteristics similar to those of

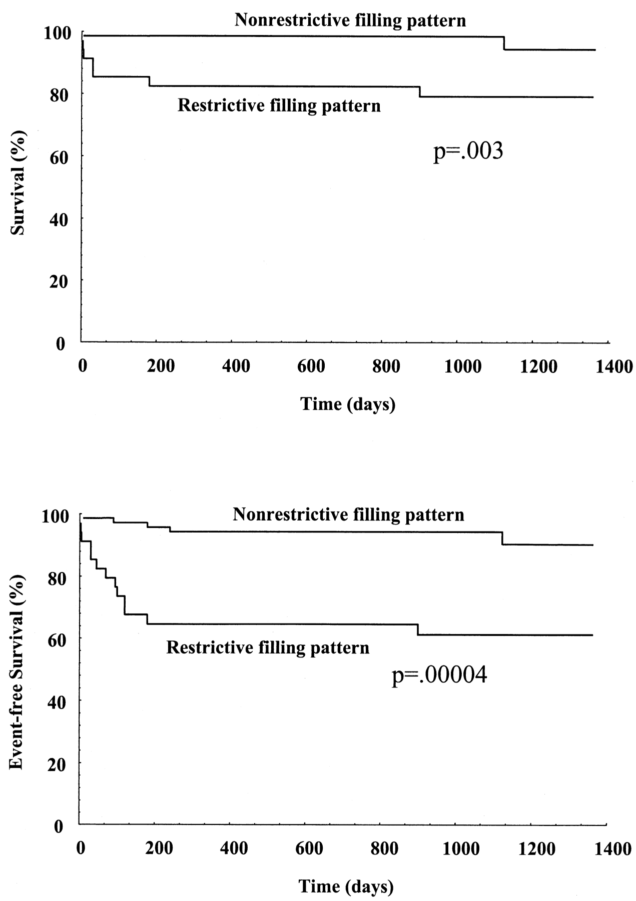


**Figure 1.** Cumulative survival rates for cardiac mortality **(top graph)** and for all major cardiac events (death, hospital admission for heart failure or myocardial infarction) **(bottom graph)**.

the total study group (Table 4). A significant progressive improvement in WMSI and LVEF was observed in patients with nonrestrictive filling (LVEF: from 44 ± 6% to 50 ± 13%; WMSI: from 2.2 ± 0.4 to 1.6 ± 0.6; $p < 0.001$), although they remained unchanged throughout the study period in patients with restrictive filling (LVEF: from 28 ± 8% to 27 ± 7%; WMSI: from 2.7 ± 0.3 to 2.7 ± 0.4). End-diastolic and end-systolic volumes were significantly higher in patients with restrictive filling than in those with nonrestrictive filling at day 3, and progressively increased in the restrictive group up to six months (Fig. 2). However, from 6 to 24 months, patients with restrictive filling did not show any further increase of LV volumes, nor a worsening of regional and global function. Similarly, paralleling serial changes in LV volumes and diastolic and systolic eccentricity indexes did not show any further increase or, in other words, any further transition of LV to a further more spherical shape (Fig. 2).

**Table 2.** Multivariate Cox Proportional Hazard Models

| Variable | p Value | RR (95% CI) |
|---|---|---|
| Ranked independent predictors of cardiovascular mortality | | |
| Age | 0.004 | 1.14 (1.04–1.25) |
| Restrictive pattern | 0.022 | 8.65 (1.35–55.3) |
| Ejection fraction | 0.424 | 1.03 (0.94–1.13) |
| Time to reperfusion in hours | 0.489 | 1.20 (0.70–2.05) |
| EDVI | 0.158 | 1.04 (0.98–1.11) |
| Ranked independent predictors of cumulative hard events* | | |
| Restrictive pattern | 0.007 | 8.38 (1.78–39.3) |
| Age | 0.021 | 1.07 (1.01–1.13) |
| Shock | 0.067 | 2.66 (0.93–7.60) |
| Wall motion score index | 0.226 | 0.33 (0.05–1.95) |
| EDVI | 0.280 | 1.02 (0.98–1.06) |
| Peak CK | 0.324 | 1.00 (0.99–1.00) |
| Blood pressure <100 mm Hg | 0.738 | 1.23 (0.38–4.28) |
| Ejection fraction | 0.745 | 1.01 (0.93–1.09) |

*Including death, myocardial infarction and hospital admission for heart failure.
CI = confidence interval; RR = relative risk; other abbreviations as in Table 1.

## DISCUSSION

The major finding of the present study was that patients with a restrictive filling pattern, as expressed by a short DT, early after anterior AMI had a poor clinical outcome, even if treated with an aggressive therapeutic approach, such as primary PTCA, and despite a persistent patency of the infarct-related artery. Cardiac death occurred only in patients with a short DT (≤130 ms). Furthermore, a short DT proved to be the most important predictor of cumulative cardiac events. Thus, the finding of restrictive LV filling in the early phase of an evolving AMI adds significant prognostic information to indicators of systolic dysfunction (ejection fraction and end-systolic volume), which are currently recognized as the most important predictors of a poor outcome after AMI (1–3), allowing the identification of a subgroup of patients at very high risk of cardiac events among patients with anterior myocardial infarction and moderate to severe systolic dysfunction.

**Restrictive filling pattern and AMI.** Recently, the utility of Doppler assessment of LV filling has been examined in the setting of AMI. Although the predominant diastolic abnormality by transient ischemia is impairment in relaxation (14), the diastolic filling pattern may change during AMI, resulting in a restrictive filling pattern as a result of an increase in resistance to LV filling or increased chamber stiffness (15–17). 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 found in those conditions with restrictive physiology.

Several studies have shown that, irrespective of the ratio of early transmitral flow velocity to atrial flow velocity (E/A ratio) changes, a short DT in postinfarction patients is strongly associated with an elevated LV filling pressure and larger infarctions (10,15,18). Recently, we have also shown that restrictive LV filling is the most powerful predictor of post–myocardial infarction LV remodeling, and that the degree of LV dilation is related to the severity of impairment of LV filling (10), indicating that the LV remodeling process is influenced by LV diastolic dysfunction. In agreement with these observations, the present study has shown that patients with restrictive LV filling had an enzymatically

**Table 3.** Multivariate Cox Model Analysis

| Model | Variables | Cardiac Death: Global Chi-Square Value | Cardiac Death: p Value | Event-Free Survival: Global Chi-Square Value | Event-Free Survival: p Value |
|---|---|---|---|---|---|
| 1 | Age, gender, diabetes, Killip class | 8.34 | 0.079 | 9.59 | 0.047 |
| 2 | EDVI, ejection fraction, wall motion score index | 19.31 | 0.007 | 14.95 | 0.036 |
| 3 | Multivessel CAD, collateral vessels (Rentrop grade >2) | 20.10 | 0.017 | 14.99 | 0.059 |
| 4 | Restrictive pattern | 28.49 | 0.001 | 23.76 | 0.004 |

Abbreviations as in Table 1.

large infarctions, moderate to severe LV systolic dysfunction and a higher prevalence of LV remodeling. These findings may provide the critical link between a restrictive filling pattern and clinical events after AMI, as observed in previous studies (4–7) and in the present one.

**Mechanical reperfusion, late patency and prognostic implications of a restrictive filling pattern.** A few preliminary studies have shown that a restrictive filling pattern is an independent predictor of adverse outcome in patients with AMI. Nijland et al. (5) reported that the survival rates at one and three years after AMI were only 50% and 22%, respectively, in patients with a short DT. Poulsen et al. (6) reported a one-year cardiac death rate of 43% among patients with a pseudonormal/restrictive LV filling pattern early after AMI. Still more recently, Moller et al. (7) showed that a short (<140 ms) DT early after AMI was the most powerful predictor of in-hospital cardiac death. However, in these studies, patients were treated with thrombolysis or conservative medical therapy, and no information was provided about the perfusion status of the infarct-related artery. In the present study, we enrolled only patients with an anterior AMI successfully treated with primary PTCA, and, to the best of our knowledge, ours is the first investigation of the prognostic contribution of Doppler transmitral early DT in a subset of patients treated with such an aggressive therapeutic approach. All patients underwent repeat coronary angiography at one and six months, which documented a high late patency rate in both patient groups. Our findings indicate that a short DT observed in an early phase of AMI still retains its prognostic significance, even after optimal recanalization of the infarct-related artery and late persistent patency. However, compared with previous studies (5–7) performed mainly in thrombolyzed patients, the outcome rate was lower, although no selections based on age, eligibility for thrombolysis or hemodynamic instability were done that could possibly exclude high risk patients. Thus, it

**Table 4.** Baseline Characteristics in the Total Group and Echocardiographic Subgroup

| | Total Group (n = 104) | Echocardiographic Subgroup (n = 48) | p Value |
|---|---|---|---|
| Age | 61 ± 12 | 60 ±10 | 0.61 |
| Male | 79 (76%) | 40 (89%) | 0.30 |
| Diabetes mellitus | 21 (20%) | 8 (17%) | 0.60 |
| Hypertension | 32 (31%) | 11 (23%) | 0.31 |
| Killip class | 1.8 ± 1 | 1.7 ±1 | 0.56 |
| Heart rate (beats/min) | 81 ± 16 | 80 ± 15 | 0.71 |
| Onset of reperfusion (min) | 166 ± 87 | 149 ± 78 | 0.25 |
| Peak CK (U/liter) | 3,516 ± 2,863 | 3,645 ± 2,366 | 0.78 |
| Multivessel CAD | 50 (48%) | 21 (44%) | 0.61 |
| Collateral vessels (grade ≥2) | 8 (8%) | 4 (8%) | 0.89 |
| EDVI (ml/m$^2$) | 73 ± 20 | 71 ± 18 | 0.55 |
| ESVI (ml/m$^2$) | 43 ± 13 | 44 ± 17 | 0.69 |
| Ejection fraction (%) | 40 ± 9 | 39 ± 10 | 0.54 |
| Wall motion score index | 2.25 ± 0.4 | 2.37 ±0.4 | 0.87 |
| Peak E-wave velocity (cm/s) | 59 ± 5 | 58 ± 16 | 0.56 |
| Peak A-wave velocity (cm/s) | 63 ± 18 | 58 ± 18 | 0.11 |
| E/A ratio | 1 ± 0.4 | 1 ± 0.5 | 0.19 |
| E-wave DT (ms) | 162 ± 45 | 160 ± 38 | 0.78 |
| Left atrium (mm) | 33 ± 8 | 32 ± 7 | 0.45 |
| Diastolic eccentricity index | 1.56 ± 0.1 | 1.57 ± 0.1 | 0.55 |
| Systolic eccentricity index | 1.82 ± 0.2 | 1.83 ± 0.2 | 0.77 |
| Mass index (g/m$^2$) | 86 ± 18 | 85 ± 22 | 0.56 |

Data are expressed as the mean value ± SD or number (%) of patients.
Abbreviations as in Table 1.

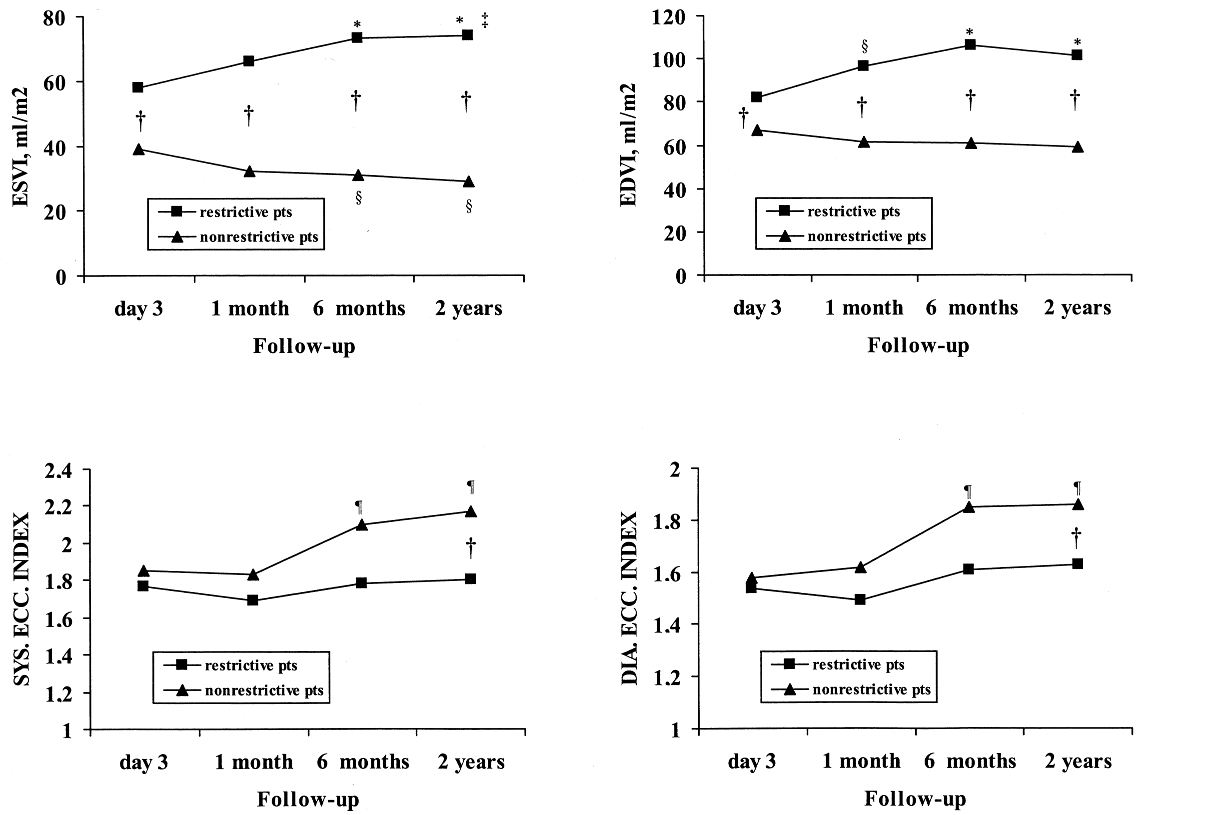


**Figure 2.** Time course of left ventricular (LV) end-systolic **(top left graph)** and end-diastolic **(top right graph)** volume indexes (ESVI and EDVI, respectively) in the restrictive and nonrestrictive filling groups. Time course of LV systolic (SYS.) **(bottom left graph)** and diastolic (DIA.) **(bottom right graph)** eccentricity (ECC.) indexes in the restrictive and nonrestrictive filling groups. Analysis of variance: *$p < 0.0005$ vs. baseline within groups; †$p < 0.001$ between groups; ‡$p < 0.05$ vs. one month within groups; §$p < 0.05$ vs. baseline within groups; ¶$p < 0.005$ vs. baseline and one month within groups.

could be hypothesized that there is a relative long-term protective effect of mechanical reperfusion and persistent late patency on clinical outcome in some patients, as indirectly confirmed by the serial changes of LV geometry and function in the subgroup of 48 patients who underwent repeat echocardiography up to 24 months. Although LV volumes increased at six months among patients with restrictive LV filling, as compared with patients with normal LV filling, they remained unchanged from 6 to 24 months, as did LVEF and diastolic and systolic eccentricity indexes. Thus, it is reasonable to speculate that mechanical reperfusion and persistent late patency may have blunted the remodeling process or at least attenuated its effects on LV shape and geometry, and therefore may have exerted a positive influence on the progression of heart failure and prognosis.

**Study limitations.** One potential limitation of the study is the lack of simultaneous hemodynamic measurement. However, this kind of correlation has been performed by other investigators (18), who showed a close inverse correlation between DT and pulmonary capillary wedge pressure, irrespective of the filling pattern expressed by the E/A ratio.

Deceleration time is partially dependent on age and is determined by the interaction of intrinsic diastolic proprieties and alterations in hemodynamic conditions and pericardial restraint related to AMI (4). However, it is difficult to control all these factors in a clinical study. Age may be unlikely to have a significant impact on our results, because its distribution was similar between the restrictive and nonrestrictive groups. Furthermore, medical treatment at hospital discharge was not different between the two patient groups.

The patient group is relatively small and limited to those with a first anterior AMI, those in sinus rhythm and those without valvular disease. Thus, our results cannot be extrapolated to the broad population with AMI.

**Conclusions.** In patients with an anterior myocardial infarction successfully treated with primary PTCA, a restrictive LV filling pattern, as assessed by Doppler-derived transmitral early DT at day 3 from the index infarction, is a powerful independent predictor of an adverse outcome. However, compared with thrombolysis, mechanical reperfusion seems to exert more favorable effects on early LV filling abnormalities after AMI, and consequently on prognosis, probably because of an anti-remodeling effect of the open infarct-related artery.

Preliminary studies performed in patients with dilated cardiomyopathy or congestive heart failure showed that reversibility of restrictive filling is associated with a better outcome (19). Thus, large-scale studies utilizing simple and easy to measure Doppler indexes of LV filling, such as mitral DT, are needed to assess the efficacy of medical therapy in patients with early LV diastolic filling abnormalities during AMI.

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