Restrictive mitral inflow pattern is a strong independent predictor of lack of viable myocardium after a first acute myocardial infarction

Augusto Sestili a,*, Claudio Coletta a, Valerio Manno b, Silvia Perna a, Marco Renzi a, Patrizia Romano a, Roberto Ricci a, Vincenzo Ceci a

a Division of Cardiology, Santo Spirito Hospital, Lungotevere in Sassia 1, Rome, Italy
b Istituto Superiore di Sanità, Rome, Italy

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Abstract In patients with acute myocardial infarction (AMI) a restrictive mitral inflow pattern successfully predicts clinical outcome. The impact of myocardial viability on the mitral inflow velocities, however, is unknown. One hundred and forty-one patients with a first AMI underwent two-dimensional, Doppler and dobutamine stress echocardiography (DSE). Patients were classified into two groups based on Doppler measurement of left ventricular filling: a restrictive group (18 patients), and a non-restrictive group (123 patients).

In the non-restrictive group, myocardial viability at DSE was found in 56 patients, while in the restrictive group only three patients showed contractile reserve (46% vs. 16%, p < 0.03). The multivariate logistic regression analysis demonstrated that restrictive mitral inflow pattern was a strong independent predictor of lack of viable myocardium (OR = 12.45, p < 0.001).

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Introduction

Doppler echocardiography provides unique information that is helpful in the management of patients with acute myocardial infarction (AMI). In particular, the mitral inflow pattern accurately predicts left ventricular (LV) filling pressures, allows identification of patients at high risk for progressive LV dilation and can provide independent prognostic information over that of the assessment of LV systolic function.1–7

* Corresponding author. Via Pitide, 34-00124 Roma, Italy. Tel.: +39 06 50914714, fax: +39 06 68352458.
E-mail address: sestit@libero.it (A. Sestili).
Moreover, in patients with ischemic cardiomyopathy, the mitral inflow pattern has been found to be a potential predictor of echocardiographic and scintigraphic indices of myocardial viability and functional recovery after revascularization.⁸

Although a persistently restrictive mitral inflow pattern with a short early diastolic velocity deceleration time (DTE) despite optimal medical therapy is associated with an advanced NYHA class and poor survival, the impact of myocardial viability on the mitral inflow velocities early after a first AMI is unknown.

We postulated that in patients with AMI, the mitral inflow pattern is critically dependent on the amount of viable myocardium. Therefore, we sought to evaluate the relation between the mitral inflow pattern and the echocardiographic indices of myocardial viability.

To test this hypothesis, we performed a prospective study of patients with first acute myocardial infarction undergoing non-invasive treatment. Dobutamine echocardiography was used to determine the extent of infarct zone viability, because the degree of contractile reserve elicited by dobutamine provides an excellent assessment of the extent of viable myocardium, in the setting of myocardial necrosis coexisting with post-ischemic myocardial dysfunction.⁹

**Methods**

**Patients**

All patients with previous myocardial infarction were a priori excluded from the study. Other exclusion criteria were atrial fibrillation, cardiomyopathies, severe valvular heart disease and technically inadequate Doppler echocardiographic studies for quantitative analysis.

The final study population consisted of 141 consecutive patients (men = 121, mean age, 59.6 ± 9.1; range, 30–76 years) with a first acute myocardial infarction referred for baseline Doppler echocardiography and pre-discharge dobutamine stress echocardiography.

In our department, at the moment of the study, dobutamine stress echocardiography early after AMI was not recommended for safety reasons in patients older than 75 years, nonetheless two patients aged 76 years were referred to dobutamine stress, based on the decision of the referring physician. Dobutamine stress echocardiography (DSE) was performed on average 8 days after the onset of symptoms (range, 5–9 days).

AMI was documented by typical rise in total creatine kinase (CK) exceeding at least twice the upper limit of normal, and at least one of the following: a history of typical ischemic symptoms (>30 min), ECG changes indicative of ischemia (ST segment elevation or depression), and development of pathologic Q waves on the ECG.¹⁰

**Two-dimensional and Doppler echocardiography**

Imaging was performed in the standard parasternal and apical views with the patient in the left lateral position (Hewlett Packard Sonos 1500, 2.5- or 3.5-MHz transducer). Short-axis tomograms were acquired at the level of the mitral valve, papillary muscles, and distal third of the left ventricle. Regional function was assessed according to the 16-segment model of the American Society of Echocardiography and graded from 1 to 4 (1 = normal, 2 = hypokinesia, 3 = akinesia, and 4 = dyskinesia).

Wall motion score index (WMSI) was calculated by summing the scores for each segment and dividing by the number of segments analysed. Ejection fraction (EF) was quantified with the biplanar Simpson's rule from the apical two- and four-chamber views.¹¹

Pulsed Doppler mitral flow velocity was obtained by placing the sample volume between the tips of the mitral leaflets in the apical four-chamber view. In each patient the following variables were measured: peak early filling velocity (E); peak filling velocity at atrial contraction (A velocity); E/A ratio; deceleration time of the peak E velocity (DTE), defined as the slope from peak E extrapolated to the baseline value; and isovolumic relaxation time (IVRT). The mean values were obtained by averaging at least three consecutive beats.

Patients were assigned to one of two groups based on Doppler measurement of LV filling: a restrictive group (18 patients), with an E/A ratio ≥ 2 or E/A between 1 and 2 and DTE ≤ 140 ms; and a non-restrictive group (123 patients), with an E/A ratio < 1 or E/A between 1 and 2 and DTE > 140 ms.¹,¹²

**Dobutamine echocardiography**

Patients underwent dobutamine echocardiography while taking all prescribed medications. Wall motion analysis for any given patients was done blinded to the mitral filling pattern data.

After baseline cross-sectional echocardiography, dobutamine infusion was started at 5 μg kg⁻¹ min⁻¹ and increased at 3-min intervals to 10, 20, 30 and
40 μg kg⁻¹ min⁻¹. Atropine (0.25–1 mg) was added if necessary to reach 85% of maximal predicted rate.

Patients underwent continuous ECG monitoring, and blood pressure was recorded at the end of each stage. Standard parasternal and apical views were recorded on half inch VHS videotape at baseline and at the end of each stage of dobutamine infusion. The echocardiographic images were digitised and displayed side-by-side in quad screen format to facilitate the comparison of images. Wall motion was evaluated visually, using both endocardial motion and systolic wall thickening. The assessment was based both on the digitised images displayed in a quad screen format and on a review of images recorded on videotape. The responses of dysfunctional segments to dobutamine were classified as biphasic (improvement at low dose with worsening at high dose), worsening, no change, and sustained improvement (increased thickening without worsening later).

A patient was considered a dobutamine responder if wall motion improved by at least grade 1 in two or more dysfunctional segments during any stage of the dobutamine infusion compared with the baseline study.

Statistical analysis

All continuous data are expressed as the mean ± SD. Unpaired t- or χ²-tests were used to compare the clinical and echocardiographic data between the two groups of patients as divided by restrictive or non-restrictive filling pattern.

Univariate and multivariate logistic regression analyses were performed to identify variables’ correlates with lack of myocardial viability detected by DSE after AMI.

Wall motion score index was dichotomized into two values: WMSI ≥ 1.56 or < 1.56; the cut point was selected based on the mean WMSI value of our population. p-Values < 0.05 were considered to be statistically significant.

All the analyses were performed with STATA 8.1 statistical package.

Results

Patients

There were 68 (48%) patients with anterior AMI, 73 (52%) with inferior AMI and 101 patients (71%) were developed pathologic Q waves on the ECG (QwMI).

Restrictive LV filling was found in 18 (13%) of 141 patients, while 123 (87%) patients were showed non-restrictive filling.

Baseline demographic and clinical characteristics of the study population according to restrictive or non-restrictive filling are summarized in Table 1. There was no significant difference between the two groups with respect to age, sex, traditional risk factors (except for higher incidence of diabetes mellitus in the restrictive group: 13% vs. 38% p = 0.01), β-blocker and thrombolytic therapies.

Patients in the restrictive group, as compared with those in the non-restrictive group, had a significantly larger enzymatic infarct size, measured by creatine kinase value (p < 0.0001), and were more likely to have anterior (p = 0.01) and QwMI (p = 0.009).

Echocardiographic data

Baseline Doppler echocardiographic and DSE data of the study population are summarized in Table 1.

Diastolic filling variables

Because the study patients were grouped on the basis of E/A ratio and DTE length, peak E was significantly (p < 0.0001) higher, peak A significantly (p < 0.0001) lower and DTE significantly (p < 0.0001) shorter in the restrictive group. Finally, patients in the restrictive group had a shorter (p < 0.0001) IVRT than patients in the non-restrictive group.

The specificity of restrictive filling pattern in predicting lack of viable myocardium was very high (95%), but its sensitivity was very low (18%); analogously, its predictive positive value was quite high (83%) but its predictive negative value was low (45%).

Systolic variables

Patients in the restrictive group had significant lower ejection fraction (p < 0.0001) and higher wall motion score index (p < 0.0001) than patients in the non-restrictive group.

More specifically, restrictive LV filling was found only in patients with a wall motion score index ≥ 1.56; however, it is important to note that as many as 13 patients (72%) of the restrictive group had a wall motion score index ≥ 1.75.

Contractile reserve with Dobutamine stress echocardiography

All patients tolerate the stress test and had adequate echocardiograms. Myocardial viability was found in 59 (41%) patients and was not found in 82 (59%) patients.

Out of 123 patients in the non-restrictive group, myocardial viability was found in 56 patients,
while out of 18 patients in the restrictive group only three patients showed contractile reserve (46% vs. 16%, \(p<0.03\); Figs 1 and 2).

Although the WMSI was higher in patients with restrictive filling as a group, a majority of the patients in the non-restrictive group with high wall motion score index (\(\geq 1.75\)) exhibited myocardial viability. When the patients were divided according to the presence or absence of myocardial viability, WMSI was higher in the first group (1.6 \(\pm \) 0.21 vs. 1.5 \(\pm \) 0.27, \(p<0.01\)).

Finally patients with contractile reserve at DSE were more likely to have diabetes mellitus (22% vs. 12%, \(p<0.01\)).

### Univariate and multivariate analyses

A logistic univariate model was constructed to identify variables' correlates with lack of viability.

Four variables showed significant association at univariate model: restrictive filling pattern, diabetes mellitus, anterior myocardial infarction and WMSI \(\geq 1.56\) (Table 2). Restrictive filling pattern was the single best predictor (\(p<0.03\)).

\(\chi^2\)-test showed a correlation between anterior myocardial infarction and WMSI (\(\chi^2 = 30.31; p < 0.0001\)), therefore only WMSI \(\geq 1.56\) was chosen to be entered in the multivariate model.

The multivariate logistic regression model demonstrated that restrictive mitral filling pattern was a strong independent predictor of lack of myocardial viability (OR = 12.45, \(p<0.001\)) (Table 3).

WMSI \(\geq 1.56\) (OR = 0.35, \(p<0.008\)) and diabetes mellitus (OR = 0.25, \(p<0.012\)) showed a negative association with the lack of contractile reserve detected by dobutamine stress echocardiography.

### Discussion

Although the relation between the restrictive mitral inflow pattern and the echocardiographic and scintigraphic indices of myocardial viability has been already documented in patients with ischemic cardiomyopathy,\(^8\) the present study demonstrates for the first time such an association in patients early after a first AMI undergoing non-invasive treatment.

As shown, when a restrictive mitral inflow pattern was present, DSE indices of viability were reduced.

In large infarcts, the reduced amount of viable myocardium and consequently the increase in LV chamber stiffness may be the link that explains the presence of a restrictive mitral inflow pattern.

### Table 1: Restrictive or non-restrictive filling

<table>
<thead>
<tr>
<th></th>
<th>All patients ((n = 141))</th>
<th>Non-restrictive group ((n = 123))</th>
<th>Restrictive group ((n = 18))</th>
<th>(p)-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>59.6 (\pm) 9.1</td>
<td>60.1 (\pm) 9.3</td>
<td>56.4 (\pm) 7.1</td>
<td>ns</td>
</tr>
<tr>
<td>Male gender</td>
<td>121 (85%)</td>
<td>103 (83%)</td>
<td>18 (100%)</td>
<td>ns</td>
</tr>
<tr>
<td>Anterior myocardial infarction</td>
<td>68 (48%)</td>
<td>54 (43%)</td>
<td>14 (77%)</td>
<td>0.01</td>
</tr>
<tr>
<td>Thrombolytic therapy</td>
<td>77 (54%)</td>
<td>64 (51%)</td>
<td>13 (76%)</td>
<td>ns</td>
</tr>
<tr>
<td>QwMI</td>
<td>101 (71%)</td>
<td>83 (67%)</td>
<td>18 (100%)</td>
<td>0.009</td>
</tr>
<tr>
<td>(\beta)-Blockers(^a)</td>
<td>69 (50%)</td>
<td>60 (49%)</td>
<td>9 (56%)</td>
<td>ns</td>
</tr>
<tr>
<td>Peak creatine kinase (U/l)(^b)</td>
<td>2257 (1867)</td>
<td>1953 (1551)</td>
<td>4506 (2459)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>23 (16%)</td>
<td>16 (13%)</td>
<td>7 (38%)</td>
<td>0.01</td>
</tr>
<tr>
<td>Hypertension</td>
<td>57 (40%)</td>
<td>53 (43%)</td>
<td>4 (23%)</td>
<td>ns</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>44 (31%)</td>
<td>38 (30%)</td>
<td>6 (33%)</td>
<td>ns</td>
</tr>
<tr>
<td>Smoke</td>
<td>83 (58%)</td>
<td>72 (59%)</td>
<td>11 (64%)</td>
<td>ns</td>
</tr>
<tr>
<td>Familiality for CAD</td>
<td>34 (24%)</td>
<td>28 (23%)</td>
<td>6 (35%)</td>
<td>ns</td>
</tr>
<tr>
<td>Obesity</td>
<td>24 (17%)</td>
<td>19 (15%)</td>
<td>5 (27%)</td>
<td>ns</td>
</tr>
<tr>
<td><strong>Echocardiographic variable characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IVRT (ms)</td>
<td>118 (\pm) 22</td>
<td>121 (\pm) 21</td>
<td>96 (\pm) 15</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>WMSI</td>
<td>1.54 (\pm) 0.25</td>
<td>1.51 (\pm) 0.25</td>
<td>1.78 (\pm) 0.17</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>44.8 (\pm) 7.6</td>
<td>46.4 (\pm) 6.8</td>
<td>34 (\pm) 3.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Myocardial viability detected by DSE</td>
<td>59 (41%)</td>
<td>56 (45%)</td>
<td>3 (16%)</td>
<td>0.03</td>
</tr>
</tbody>
</table>

\(\chi^2\)-test showed a correlation between anterior myocardial infarction and WMSI (\(\chi^2 = 30.31; p < 0.0001\)), therefore only WMSI \(\geq 1.56\) was chosen to be entered in the multivariate model.

CAD, coronary artery disease; QwMI, Q wave myocardial infarction; IVRT, isovolumic relaxation time; LVEF, ejection fraction of the left ventricle (%); WMSI, wall motion score index; and DSE, dobutamine stress echocardiography.

\(^a\) \(\beta\)-Blocker therapy could be assessed in 141 patients (97%).

\(^b\) Peak creatine kinase could be assessed in 126 patients (89%).
These results can help to better understand the clinical results, as seen in some previous observational studies, that an increased LV stiffness, as measured by Doppler echocardiography, adversely affects the remodeling process and the survival after AMI.

Relation between restrictive filling pattern, myocardial viability and outcome 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.[1-7]

Cerisano et al.[3] have shown that a restrictive LV filling pattern 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, indicating that the LV remodeling process is influenced by LV diastolic dysfunction.

Moreover, other studies have shown that a restrictive LV filling after AMI is also an independent predictor of worse survival.

Nijland et al.[1] reported that restrictive filling is the single best predictor of cardiac mortality following myocardial infarction, and that it adds significantly to the predictive power of clinical and echocardiographic markers of systolic dysfunction.

Poulsen et al.[2] reported a 1-year cardiac death of 43% among patients with a pseudonormal/restrictive LV filling pattern early after AMI, while none of the patient without pseudonormal/restrictive pattern died. Recently, Møller et al.[5] have shown that the presence in the early phase of myocardial infarction of elevated LV end-diastolic pressure as assessed by an E/Vp ratio ≥ 1.5 at color M-mode Doppler, is associated with a worse prognosis (survival rate 58% vs. 98% in patients with an
However, a short (<140 ms) DTE early after AMI remained the most powerful predictor of in-hospital cardiac death.

Still more recently, Temporelli et al. have shown that LV 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 6-month remodeling and 4-year mortality.

In these studies, patients were treated with thrombolysis or conservative medical therapy and no information was provided about the perfusional status of the infarct-related artery.

Table 2  Logistic univariate model

<table>
<thead>
<tr>
<th></th>
<th>%</th>
<th>Odds ratio</th>
<th>p-Value</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restrictive mitral inflow pattern</td>
<td>12.77</td>
<td>4.18</td>
<td>0.030</td>
<td>1.15–15.17</td>
</tr>
<tr>
<td>Anterior myocardial infarction</td>
<td>48.23</td>
<td>0.52</td>
<td>0.059</td>
<td>0.26–1.03</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>16.31</td>
<td>0.49</td>
<td>0.123</td>
<td>0.20–1.21</td>
</tr>
<tr>
<td>WMSI ≥ 1.56</td>
<td>52.48</td>
<td>0.62</td>
<td>0.169</td>
<td>0.32–1.22</td>
</tr>
<tr>
<td>Age (mean ± SD)</td>
<td>59.66 ± 9.18</td>
<td>1.01</td>
<td>0.468</td>
<td>0.98–1.05</td>
</tr>
<tr>
<td>QwMI</td>
<td>71.63</td>
<td>1.29</td>
<td>0.480</td>
<td>0.63–2.64</td>
</tr>
<tr>
<td>β-Blockers</td>
<td>50.36</td>
<td>0.86</td>
<td>0.654</td>
<td>0.43–1.69</td>
</tr>
<tr>
<td>Thrombolytic therapy</td>
<td>54.61</td>
<td>1.15</td>
<td>0.676</td>
<td>0.59–2.26</td>
</tr>
<tr>
<td>Hypertension</td>
<td>40.43</td>
<td>1.11</td>
<td>0.767</td>
<td>0.56–2.20</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>31.21</td>
<td>0.92</td>
<td>0.828</td>
<td>0.45–1.90</td>
</tr>
<tr>
<td>Male gender</td>
<td>85.82</td>
<td>0.92</td>
<td>0.857</td>
<td>0.35–2.40</td>
</tr>
</tbody>
</table>

WMSI, wall motion score index; QwMI, Q wave myocardial infarction.
Finally, Cerisano et al.⁶ showed that patients with a restrictive LV filling pattern early after anterior AMI have a poor clinical outcome, even if treated with primary PTCA.

In agreement with these observations, the present study has shown that patients with a restrictive LV filling had an enzymatically larger infarctions and moderate to severe LV systolic dysfunction.

We observed, in addition, a strong inverse correlation between restrictive mitral filling pattern and the presence of viable myocardium detected by DSE, in other words, a restrictive LV filling was associated with high probability of lack of viable myocardium.

These results were derived from a population undergoing conservative therapy and may not be applicable to patients who are deemed suitable for mechanical revascularization.

Nevertheless, we believe that the associations between a restrictive filling and viability indices help elucidate the mechanism linking the mitral inflow pattern to remodeling and survival after AMI.

There is currently strong evidence that the presence and extent of myocardial viability have a significant impact on the clinical outcome of patients after AMI.

Several studies have shown that after AMI the presence of relatively large amount of viable myocardium in the infarct zone strongly contributes to maintenance of left ventricular wall shape and size, whereas absence of viability results in ventricular dilatation, particularly in large infarcts.¹³⁻¹⁵

This finding is likely to be of prognostic importance, as mortality increases with increased left ventricular volumes. Carlos et al.¹⁶ showed that both large infarct size and non-viability of the infarct zone at low dose dobutamine are most strongly predictive of adverse outcome.

Therefore, the reduced amount of viable myocardium accounts for the worse survival in those patients after AMI who exhibit a restrictive mitral inflow pattern.

Our study had a seemingly paradoxical finding: high WMSI (WMSI ≥ 1.56, OR = 0.35, p < 0.008) and diabetes mellitus (OR = 0.25, p < 0.012) showed a negative association with lack of myocardial viability (an odds ratio less than one implies that the lack of contractile reserve was less likely in these patients).

In patients with AMI (both with and without the administration of reperfusion thrombolytic therapy), reversibly injured, functionally stunned myocardium lies adjacent to infarcted myocardium. The severity of stunning is often greater in patients with high WMSI (but without a restrictive mitral inflow pattern), and full recovery may be delayed over weeks.

Diabetes does not seem to have a negative influence on the presence of viable myocardium and in the development of LV remodeling in patients with AMI. Recently Carrabba et al.¹⁷ showed that after AMI, diabetes is not an independent predictor of subsequent LV remodeling. According to these data, at 6 months, a similar incidence of LV remodeling was found in diabetic and non-diabetic patients, with similar patterns of changes in LV global and regional systolic function and in LV volumes.

### Reason for a restrictive mitral inflow pattern after AMI

Marked changes in the diastolic properties of the LV can occur in the presence of ischemic heart disease. First, acute myocardial ischemia slows ventricular relaxation and increases myocardial wall stiffness. Acute myocardial infarction causes more complex changes in ventricular pressure-volume relationships, depending on the size of the infarct and the time following infarction at which the measurements are made.

Infarcted muscle tested very early exhibits reduced stiffness (the inverse of compliance). Subsequently, the development of myocardial contracture, interstitial edema and fibrocellular infiltration contributes to stiffening of the necrotic tissue and thereby to increased chamber stiffness, with a steeper ventricular pressure—volume curve (a greater increase in pressure for any increase in volume).¹⁸

As with impairment of systolic function, the magnitude of the diastolic abnormality appears to be related to the size of infarct.

An increase in the chamber stiffness of the left ventricle will affect the transmural flow velocity in a specific manner. A high left atrial pressure at the time of mitral opening and a large left atrial-left ventricular gradient in early diastole will produce a fast acceleration in blood flow.
flow velocity curve. A high E velocity will occur on the mitral velocity curve. A rapid increase in LV pressure after its nadir will cause a rapid deceleration on the transmital flow velocity curve. There will be a lower forward velocity at atrial contraction because a relatively greater filling of the LV has occurred in early diastole. Therefore, it is not surprising, in view of the aforementioned effects of ischemia on LV chamber stiffness, 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".

Limitations

One major limitations of this study is that all possible information obtainable from Doppler echocardiography was not incorporated into assessing diastolic filling of the LV. Interrogation of pulmonary venous flow velocities may add information to the mitral inflow velocities in this assessment, moreover, recently, color M-mode and tissue Doppler have provided useful insights into the study of diastolic function. These new Doppler applications may be combined with standard Doppler flow indices to provide an accurate estimate of LV filling pressure and appear to be relatively insensitive to the effects of preload compensation, furthermore they make available prognostic information incremental to standard parameters.

However, the purpose of this study was to look specifically at the diagnostic accuracy of the mitral inflow velocity curves, routinely obtained in all echocardiographic laboratories.

The transmital flow patterns by Doppler echocardiography are partially dependent on age, heart rate, loading conditions and degree of mitral regurgitation. Age may be unlikely to have significant impact on our results because age distribution was similar between the restrictive and non-restrictive groups, and patient's age in this study was relatively old (average 60 years).

Heart rate was not characterized in this study, however, an increased heart rate, such as found in patients with restrictive filling, has been demonstrated to increase atrial filling, resulting in a non-restrictive rather than a restrictive pattern. Mitral regurgitation is another variable that has been recognized to influence left ventricular filling pattern, especially in patients with severe insufficiency. Typically, these patients have a larger E wave, which would resemble a restrictive pattern, therefore, those patients with severe mitral regurgitation were excluded from the present analysis.

Small differences in the location of the Doppler sample volume have been shown to change the contour of the mitral flow velocity curves because of the non-laminar velocity profile through the mitral valve. The velocity profile may differ in various diseases because it is related to left atrial driving pressure as well as to the rate of left ventricular relaxation and stiffness. The sample volume was placed at the tip of the mitral leaflets as they opened into the left ventricle, the location at which the velocity profile changes least throughout diastole.

Another limitation of the present study is the relatively small patient group. Specifically the number of patients with restrictive filling pattern may be too small to detect differences between groups as a result of insufficient power. Moreover, the patients group was limited to those with a first AMI and age not older than 76 years, those in sinus rhythm and those without valvular disease. Thus, our results cannot be extrapolated to the broad population with AMI.

Measurement of total CK is not recommended, at present, for the routine diagnosis of AMI, because of the wide tissue distribution of this enzyme. The most recently described and preferred biomarker for myocardial damage is cardiac troponin, or in alternative CK-MB (measured by mass assay). In the absence of availability of these last biomarkers in our hospital at the moment of the study, we employed total CK (two times greater than the upper reference limit), that, although less satisfactory than new biomarkers, demonstrated to maintain a valuable diagnostic power for the detection of myocardial infarction.

Finally, the analysis of regional function at rest and during dobutamine infusion was semiquantitative. However, this qualitative approach has been widely adopted for clinical studies with stress echocardiography in ischemic heart disease.

Conclusions

Our study shows a strong association of the mitral inflow pattern with DSE indices of myocardial viability in patients with a first AMI undergoing conservative therapy. The reduced amount of viable myocardium may be the link that explains why the restrictive mitral inflow pattern is a powerful predictor of LV remodeling and survival after AMI, as seen in some observational studies.

More important, our study identifies the mitral inflow pattern as a potential predictor of viability
and therefore of functional recovery in patients after AMI.

However, further larger studies are needed to better understand the relation between the mitral inflow pattern and the indices of myocardial viability after AMI and whether outcome, in patients with restrictive filling, may be improved by an aggressive therapeutic approach.

References