Prediction of Functional Recovery of Viable Myocardium After Delayed Revascularization in Postinfarction Patients

Accuracy of Dobutamine Stress Echocardiography and Influence of Long-Term Vessel Patency

Jean-Luc Monin, MD,* Jérôme Garot, MD,* Marielle Scherrer-Crosbie, MD,* Jean Rosso, MD,* Anne-Marie Duval-Moulin, MD,* Patrick Dupouy, MD,† Emmanuel Teiger, MD,† Alain Castaigne, MD,* Jean-Claude Cachin, MD,* Jean-Luc Dubois-Rande, MD, PhD,* Pascal Gueret, MD, FACC*
Creteil, France

OBJECTIVES

We sought to evaluate Dobutamine stress echocardiography (DSE) for predicting recovery of viable myocardium after revascularization with cineangiography as a gold standard for left ventricular (LV) function. We studied the influence of late vessel reocclusion on regional LV function.

BACKGROUND

Dobutamine stress echocardiography is a well established evaluation method for myocardial viability assessment. In previous studies the reference method for assessing LV recovery was echocardiography; long-term vessel patency has not been systematically addressed.

METHODS

Sixty-eight patients with a first acute myocardial infarction (AMI) and residual stenosis of the infarct related artery (IRA) underwent DSE (mean ± standard deviation) 21 ± 12 days after AMI to evaluate myocardial viability. Revascularization of the IRA was performed in 54 patients by angioplasty (n = 43) or bypass grafting (n = 11). Coronary angiography and LV cineangiography were repeated at four months to assess LV function and IRA patency.

RESULTS

Sensitivity and specificity of DSE for predicting myocardial recovery after revascularization were 83% and 82%. In the case of late IRA patency, specificity increased to 95%, whereas sensitivity remained unchanged. In the 16 patients with myocardial viability and late IRA patency, echocardiographic wall motion score index decreased after revascularization from 1.83 ± 0.15 to 1.36 ± 0.17 (p = 0.0001), and left ventricular ejection fraction (LVEF) increased from 0.52 ± 0.06 to 0.57 ± 0.06 (p = 0.0004), whereas in five patients, reocclusion of the IRA prevented improvement of segmental or global LV function despite initially viable myocardium.

CONCLUSIONS

Dobutamine stress echocardiography is reliable to predict recovery of viable myocardium after revascularization in postinfarction patients. Late reocclusion of the IRA may prevent LV recovery and influence the accuracy of DSE. (J Am Coll Cardiol 1999;34:1012–9) © 1999 by the American College of Cardiology

In patients with acute myocardial infarction (AMI), regional left ventricular (LV) dysfunction may represent necrotic or viable myocardium. When viable myocardium is supplied by a vessel without significant stenosis, progressive functional recovery may occur (1). However, viable myocardium supplied by a critically stenosed vessel may benefit from delayed revascularization and show functional recovery of the involved segments (2,3). Dobutamine stress echocardiography (DSE) has the potential to detect viable myocardium early after myocardial infarction (4–8) and to predict functional recovery of viable myocardium after revascularization in patients with chronic coronary artery disease (CAD) (9–16). All previous studies evaluating DSE for myocardial viability have focused on echocardiography as the reference standard to evaluate long-term improvement of segmental LV function. In these studies, echocardiography was used for assessment of inotropic reserve and as the reference for functional improvement. Furthermore, the effects of long-term patency of the revascularized vessel on functional recovery of viable segments have not been specifically addressed, whereas it has been recently demonstrated that reocclusion of the infarct-related artery (IRA)
may prevent viable myocardium from regaining contractility after revascularization (17,18).

The aim of this study was to evaluate the ability of DSE to predict regional LV function recovery after delayed revascularization of the IRA in patients after AMI using serial LV angiography as the gold standard for myocardial recovery, and to determine the influence of long-term patency of the IRA on segmental LV function and diagnostic accuracy of DSE.

METHODS

Patient population. Sixty-eight patients with a first myocardial infarction were prospectively included in this study between May 1995 and May 1997. The inclusion criteria were:

1) diagnosis of a first Q-wave AMI, with significant initial electrocardiographic ST segment elevation (>1 mm) and a significant creatine kinase MB isoenzyme elevation,

2) segmental asynergy at early LV angiography,

3) clearly identified IRA with a significant residual stenosis (>70% diameter reduction) or complete occlusion, suitable for revascularization by either percutaneous transluminal coronary angioplasty (PTCA) or coronary artery bypass grafting (CABG),

4) informed consent obtained from each patient.

Exclusion criteria were poor echogenicity, significant stenosis (>50%) of the left main coronary artery, emergency revascularization, unstable angina, atrial fibrillation or ventricular tachycardia that occurred more than 48 h after AMI.

Study protocol. Dobutamine stress echocardiography was performed between 15 and 21 days after AMI. Delayed revascularization of the IRA was attempted in all patients by PTCA or CABG regardless of DSE results. Regional and global systolic LV function were assessed by LV angiography performed immediately before revascularization and at four-month follow-up examination. Patency of the IRA was assessed by a repeat coronary angiogram at follow-up. All patients received aspirin (250 mg/day) and beta-adrenergic blocking agents if tolerated. Angiotensin-converting enzyme (ACE) inhibitors were given within the first week of AMI when the first angiographic ejection fraction was <40%. Medications were not modified after revascularization and the dosage was kept constant until the four-month follow-up examination.

DSE. All patients underwent dobutamine stress echocardiography with commercially available equipment (Acuson [Mountain View, California] 128 XP-10 c with 2.5 to 4 Mhz transducers). Beta-blockers were withdrawn for at least 48 h before DSE and reintroduced immediately after the test. Other medications (including calcium-channel blockers, nitrates, ACE inhibitors, diuretics, aspirin and anticoagulants) were continued. Four echographic views (parasternal long and short axis, apical four and two chambers) were digitized on-line with an automated acquisition system (Image Vue DCR 1.41, Nova Microsonics [Mahwah, New Jersey] at baseline, low and peak dose of dobutamine. All digitized loops were stored on 5 1/4 in. optical disks for subsequent blind analysis. After baseline imaging, continuous intravenous infusion of dobutamine was started at 5 µg/kg/min and increased by steps of 5 µg/kg/min every 5 min up to a maximum dose of 40 µg/kg/min. Atropine (0.25 to 1 mg) was added if necessary to reach 85% of maximal predicted heart rate. Electrocardiogram (ECG), blood pressure and heart rate were monitored at the end of each stage. Criteria for terminating the study were:

1) reaching ≥85% of maximal predicted heart rate,

2) worsening of wall motion in 2 contiguous myocardial segments,

3) fall of systolic blood pressure >30 mm Hg,

4) chest pain with ST segment depression ≥1 mm,

5) frequent ventricular or supraventricular ectopic beats.

Repeat rest echocardiography was performed at follow-up examination, and the same four views were digitized for off-line analysis.

Echocardiographic analysis. All DSE studies and follow-up echocardiograms were analyzed off-line by two independent observers blinded to patients’ clinical and angiographic data. The LV was divided into 16 segments for wall motion analysis according to the recommendations of the American Society of Echocardiography (19). Myocardial thickening of each segment was graded visually using a semiquantitative score (19): 1 = normal wall motion; 2 = hypokinesia (decreased systolic wall thickening); 3 = akinesia; 4 = dyskinesia (outward wall motion or systolic wall thinning). Myocardial viability in each segment was defined by a decrease of the wall motion score of >1 point, for example, from 3 to 2 (or 1), or from 2 to 1, whereas decrease

Abbreviations and Acronyms

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
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<tbody>
<tr>
<td>ACE</td>
<td>angiotensin-converting enzyme</td>
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<td>AMI</td>
<td>acute myocardial infarction</td>
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<td>ANOVA</td>
<td>two-way analysis of variance</td>
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<td>BP</td>
<td>blood pressure</td>
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<td>CABG</td>
<td>coronary artery bypass grafting</td>
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<td>CAD</td>
<td>coronary artery disease</td>
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<td>DSE</td>
<td>dobutamine stress echocardiography</td>
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<td>ECG</td>
<td>electrocardiogram</td>
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<td>IRA</td>
<td>infarct-related artery</td>
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<tr>
<td>LAD</td>
<td>left anterior descending</td>
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<tr>
<td>LV</td>
<td>left ventricle or ventricular</td>
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<td>LVEF</td>
<td>left ventricular ejection fraction</td>
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<tr>
<td>PTCA</td>
<td>percutaneous transluminal coronary angioplasty</td>
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<td>SD</td>
<td>standard deviation</td>
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<tr>
<td>WMSI</td>
<td>wall motion score index</td>
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WMSI = sum of all segment scores divided by the number of scored segments. Myocardial viability was defined for each patient by the two following criteria:

1) improvement of myocardial thickening during DSE in ≥2 adjacent segments located in the infarct zone, and
2) decrease of the LV WMSI ≥20%, as previously proposed (9).

Interobserver variability was assessed in 20 patients (1,247 segments). Differences between the two observers were resolved by consensus.

Assessment of LV function by cineangiography. The reference method for assessment of segmental and global LV systolic function was LV cineangiography performed immediately before revascularization and at four-month follow-up examination. Systolic function in the infarct zone and LV global ejection fraction were calculated from the angiographic data by one independent observer blinded to clinical and echocardiographic data. End-diastolic and endsystolic volume indexes (ml/m²) as well as left ventricular ejection fraction (LVEF) were calculated by the area-length method (20). Regional wall motion was expressed as radial shortening fraction of 13 anatomic wall segments by the center-line method in the right-anterior oblique view as previously described (18). The normal range for ejection fraction and for each radial shortening segment was determined from ventriculograms performed in 40 subjects matched for age and gender with atypical chest pain and normal coronary angiograms without other cardiovascular disease. To compare values for regional anterior or posterior wall motion with normal values and values at follow-up angiography, a standardized motion index expressed in units of standard deviation (SD) from the mean in control subjects was calculated (% shortening of patient's segment — mean of the same normal segment)/SD of the normal segment). The wall motion for an individual segment was considered normal if the value was within two SDs of the equivalent mean value in controls. Regional wall motion was considered to be hypokinetic if the wall motion of at least three segments was ≤2 SDs below the normal range. For a given territory (segments from 1 to 6 or from 7 to 13), an average of the three most hypokinetic segments was calculated and used for statistical comparisons.

Follow-up angiography. Coronary and LV angiograms were obtained at follow-up in all 52 surviving patients. The infarct vessel was considered to be reoccluded at four-months in the case of TIMI grade 0 or 1. In the case of restenosis ≥50% but TIMI grade 2 or 3, the artery was classified as patent. Surgical revascularization was considered successful if the graft did not reveal a ≥50% stenosis. Recovery of systolic regional LV function at follow-up examination was defined as an improvement of WMSI ≥1 SD for all three aforementioned initially most hypokinetic segments.

Statistical analysis. Data in the text, tables and figures are expressed as mean ± SD. Sensitivity, specificity and positive and negative predictive values of DSE were calculated according to the standard formulae. Two-way analysis of variance (ANOVA) for repeated measures was performed to assess differences in continuous variables (WMSI and LVEF) between patients with and without myocardial viability by DSE and between patients with and without IRA patency at follow-up examination. Paired Student t test was used when appropriate. Differences between proportions were assessed by chi-square analysis. Correlations were obtained by the linear regression method. A p value <0.05 was considered to indicate statistical significance.

RESULTS

Patient data. Sixty-eight patients were initially enrolled. Sixteen patients were excluded from analysis for the following reasons: 3 deaths (1 patient before revascularization and 2 patients after CABG), poor echogenicity in 2 patients, 11 patients with initially occluded IRA and failure to reopen the vessel by PTCA. Finally, 52 patients (48 men, 4 women, mean age 55 ± 10 years) completed the study. Location of AMI was anterior in 28 and inferior in 24 patients. Dobutamine stress echocardiography and revascularization were performed 21 ± 11 days and 28 ± 17 days after AMI, respectively. Forty-three patients underwent successful PTCA including three patients with stent implantation. The nine remaining patients underwent CABG without clinical, ECG or biochemical signs of perioperative myocardial infarction. All patients underwent follow-up studies 110 ± 12 days after revascularization, including a rest echocardiogram, coronary angiography and LV angiography. Medical regimens were not modified throughout the study-period.

Interobserver reproducibility for DSE and LV angiography. Interobserver reproducibility for echocardiographic wall motion score was assessed by comparing the score given by the two independent observers for each of the 16 segments in 20 randomly selected patients at baseline, low-dose 1, low-dose 2 and peak dose of dobutamine. Comparison was possible for 1,247 scored segments. The same score (from 1 to 4) was accorded by the two observers to 1,107 segments (agreement in 89% of cases). A 1-point difference in score was noted for 134 segments and a 2-point difference was noted in only six segments (interobserver disagreement in 11% of segments).

Interobserver reproducibility for the measurement of angiographic LVEF was also assessed in 20 randomly selected patients. The mean difference in LVEF between the two observers was 0.95 ± 0.59% and was not significant (p = 0.22). Linear regression showed reproducible results.
and a good correlation between the two measurements ($y = 1.01x + 0.59$, $r = 0.95$, $p < 0.0001$).

**Correlation at follow-up examination between echocardiography and cineangiography.** Follow-up echocardiographic data were directly compared with follow-up angiographic data to correlate changes in wall motion and LVEF at angiography and changes in echocardiographic WMSI. There was no correlation between changes in angiographic LVEF and changes in echographic WMSI. The correlation between the changes in regional wall motion in units of standard deviation by angiography and the changes in WMSI by echography was statistically significant: $r = 0.53$, $p = 0.0001$.

**DSE studies.** Three patterns of segmental wall motion during DSE were observed in infarct territory:

1) sustained improvement at low and high doses of dobutamine in 3 patients,
2) biphasic response in 18 patients, with improved wall thickening at low-dose dobutamine followed by deterioration at higher doses,
3) no change in segmental wall motion in 31 patients.

Biphasic response or sustained improvement at DSE were considered to be an indicator of viable myocardium. The mean peak dose of dobutamine was $25 \pm 10 \mu g/kg/min$ for the whole group. In five patients, the test was stopped at $10 \mu g/kg/min$ (frequent ectopic ventricular beats in four cases and achievement of maximal predicted heart rate in one). The reasons for terminating the study were reaching $85\%$ of maximal predicted heart rate in 21 patients, worsening of segmental wall motion in 20 patients accompanied by chest pain in 5 cases, arrhythmias in 11 patients (frequent ectopic beats in 9 patients and nonsustained VT in 2 patients). The mean dose of dobutamine at which patients exhibited improvement of systolic wall thickening was $9.0 \pm 3 \mu g/kg/min$. At this dose, there was no significant acceleration of heart rate ($68 \pm 9$ vs. $66 \pm 8$ beats/min at rest) and no significant change in blood pressure (BP) (systolic BP = $116 \pm 16$ vs. $112 \pm 13$ mm Hg).

**Reocclusion of the IRA at follow-up examination.** All grafts were patent at follow-up examination in the patients who underwent CABG. In the 43 patients who underwent PTCA, the IRA was found to be reoccluded in 12 cases (28%) at follow-up examination. The reocclusion rate was higher in patients with initial occlusion of the IRA when compared with patients in whom IRA was initially patent (respectively 9/19 vs. 3/24, $p < 0.001$).

**Diagnostic accuracy of DSE in light of vessel patency.** Myocardial viability in the infarct zone was diagnosed by DSE in 21/52 patients (40%), whereas 31 patients had no inotropic reserve. Functional recovery after revascularization was observed in 18/52 patients (35%) at follow-up angiography and 34 patients exhibited fixed dysfunction.

Among 18 patients with a biphasic response at DSE, recovery of the infarct zone at follow-up examination was present in 15 (true positives); all of them had a patent IRA at follow-up (Fig. 1). Three patients with a biphasic response showed no recovery at follow-up examination (false positives). In the three latter patients, two patients showed inotropic reserve of the inferior wall at DSE and one patient showed improved wall motion of the anterior wall and apex. All three patients had a reoccluded IRA at follow-up examination. None of the three patients with sustained improvement at DSE showed any recovery of the infarct–zone at follow-up examination (false positives). All had inotropic reserve of the anterior wall at DSE and IRA was patent at follow-up examination in 1/3 of the patients.

Among the 31 patients with no change in segmental wall motion at DSE, functional recovery occurred in three patients (false negatives) in whom the IRA was patent at follow-up. Twenty-eight patients without inotropic reserve did not show any functional improvement (true negatives) despite a patent IRA at follow-up examination in 21 patients. Thus, six patients were classified as false positives for DSE. Of these, five patients had a reoccluded IRA at follow-up procedure.

For the whole group of patients, the sensitivity, specificity and positive and negative predictive values of DSE to predict functional recovery of viable myocardium after revascularization were 83%, 82%, 71% and 90%, respectively.

In the subgroup of patients with a patent IRA at follow-up procedure, the specificity of DSE increased from 82% to 95% and the positive predictive value increased from 71% to 93%, whereas the sensitivity was unchanged (83%) and the negative predictive value slightly decreased from 90% to 82%.

All 15 patients showing a biphasic response at DSE and a patent IRA at follow-up had a functional improvement in the infarct zone at follow-up procedure. In these patients
the positive predictive value of a biphasic response was 100%.

**Influence of IRA patency at follow-up procedure on regional and global LV function.** Vessel patency and echocardiographic WMSI. Baseline WMSI was not significantly different between the patients with patent versus occluded IRA at follow-up examination and between the patients with functional recovery versus fixed dysfunction at follow-up examination (Table 1). The results of ANOVA for the evolution of WMSI (baseline vs. follow up) showed no significant effect of myocardial viability alone ($p = 0.52$) and a slight but significant effect of late vessel patency alone ($p = 0.036$); the interaction of viability and late vessel patency was highly significant ($p = 0.0089$). Wall motion score index decreased after revascularization in the 21 patients with myocardial viability at DSE (from $1.79 \pm 0.16$ to $1.44 \pm 0.22$, $p = 0.0001$, Fig. 2) and in the 16 patients with myocardial viability and a patent IRA at follow-up examination (from $1.83 \pm 0.15$ to $1.36 \pm 0.17$, $p = 0.0001$, Fig. 2). Conversely, in the five patients with viable myocardium at DSE and reoccluded IRA at follow-up examination, there was no significant change in WMSI at follow-up (from $1.68 \pm 0.14$ to $1.69 \pm 0.18$, $p = 0.9$, Fig. 2). In the remaining patients without viability at DSE, no significant change in WMSI was noted after revascularization (from $1.88 \pm 0.27$ to $1.79 \pm 0.27$, $p = 0.1$).

**Influence of vessel patency on LVEF.** Left ventricular ejection fraction at baseline was significantly lower in patients without viability when compared with patients with viability at DSE ($43 \pm 11\%$ vs. $50 \pm 9\%$, respectively, $p =$

![Figure 2](image)

Figure 2. Influence of long-term vessel patency on WMSI in viable patients after revascularization. FU = follow-up; Viable all = all patients with myocardial viability at DSE; Viable + patent = patients with viable myocardium at DSE and patent IRA at follow-up; Viable + reocc = patients with viable myocardium at DSE and a reoccluded IRA at follow-up; WMSI: echocardiographic wall motion score index. Data are shown as mean value $\pm$ SD. *$p = 0.0001$. Solid bars = baseline WMSI; open bars = follow-up WMSI.

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

<table>
<thead>
<tr>
<th></th>
<th>Patent IRA at FU</th>
<th>Occluded IRA at FU</th>
<th>$p$</th>
<th>Recovery at FU</th>
<th>No Recovery</th>
<th>$p$</th>
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<tbody>
<tr>
<td>Patients (n)</td>
<td>40</td>
<td>12</td>
<td></td>
<td>18</td>
<td>34</td>
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<tr>
<td>Age (yr)</td>
<td>$56 \pm 10$</td>
<td>$53 \pm 12$</td>
<td>NS</td>
<td>$57 \pm 9$</td>
<td>$55 \pm 11$</td>
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<td>Infarct location</td>
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<td>Anterior AMI</td>
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<td>$&lt;0.001$</td>
<td>n = 8</td>
<td>n = 20</td>
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<td>n = 10</td>
<td>n = 14</td>
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<td>Peak CK (UI/l)</td>
<td>$3786 \pm 2020$</td>
<td>$2930 \pm 1034$</td>
<td>NS</td>
<td>$2803 \pm 1178$</td>
<td>$3968 \pm 2032$</td>
<td>NS</td>
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<td>Baseline WMSI</td>
<td>$1.82 \pm 0.19$</td>
<td>$1.94 \pm 0.33$</td>
<td>NS</td>
<td>$1.77 \pm 0.16$</td>
<td>$1.89 \pm 0.26$</td>
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<td>Angiographic</td>
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<td>LVEF (%)</td>
<td>$47 \pm 9$</td>
<td>$40 \pm 15$</td>
<td>$0.03$</td>
<td>$51 \pm 7$</td>
<td>$43 \pm 11$</td>
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<td>Infarct-related vessel</td>
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<td>LAD (n)</td>
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<td>8</td>
<td>*</td>
<td>8</td>
<td>20</td>
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<td>RCA (n)</td>
<td>16</td>
<td>4</td>
<td>**</td>
<td>7</td>
<td>13</td>
<td>§§</td>
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<tr>
<td>Cx (n)</td>
<td>4</td>
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<td></td>
<td>3</td>
<td>1</td>
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<td>Initial status of IRA</td>
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<tr>
<td>Occluded (n)</td>
<td>13</td>
<td>9</td>
<td>$0.001$</td>
<td>6</td>
<td>16</td>
<td>0.004</td>
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<tr>
<td>Stenosis $\geq 70%$ (n)</td>
<td>27</td>
<td>3</td>
<td>12</td>
<td>18</td>
<td></td>
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</tr>
</tbody>
</table>

*p = 0.02 vs. RCA; p = 0.0001 vs. Cx; *p = 0.0001 vs. Cx; **p = 0.04 vs. RCA; p = 0.0001 vs. Cx; §§p = 0.0001 vs. Cx.

AMI = acute myocardial infarction; CK = creatine kinase; Cx = left circumflex artery; FU = follow-up; IRA = infarct related artery; LAD = left anterior descending artery; LVEF = left ventricular ejection fraction; RCA = right coronary artery; WMSI = wall motion score index.
In patients with fixed dysfunction or reocclusion of the IRA at follow-up procedure, LVEF at baseline was significantly lower and the IRA was more frequently the left anterior descending (LAD) (Table 1). The results of ANOVA for the evolution of LVEF (baseline vs. follow-up) showed no significant effect of myocardial viability alone (p = 0.21) and no significant effect of late vessel patency alone (p = 0.67); the interaction of viability and late vessel patency was significant (p = 0.035). When considering the whole population, revascularization of the culprit vessel led to a slight increase of LVEF (from 46 ± 11% to 49 ± 11%, p = 0.01). Left ventricular ejection fraction also increased slightly after revascularization (from 50 ± 9% to 53 ± 9%, p = 0.02, Fig. 3) for the 21 patients with viable myocardium at DSE. A greater increase in LVEF (from 52 ± 6% to 57 ± 6%, p = 0.0004) was noted at follow-up examination for the 16 patients with viable myocardium and a patent IRA at follow-up examination, whereas no significant improvement (from 42 ± 11% to 42 ± 9%, p = 0.96) occurred in the five patients with viability at DSE and occluded IRA at follow-up examination (Fig. 3).

DISCUSSION

In this study, we demonstrated that, in patients with a first Q-wave AMI, functional improvement after delayed revascularization of the IRA was accurately predicted by DSE performed two weeks after AMI with LV cineangiography as a gold standard for the assessment of functional recovery. The systematic evaluation of patency of the IRA at follow-up examination showed that regional LV function improvement, and therefore the diagnostic accuracy of DSE, may be influenced by late reocclusion of the infarct vessel. Our population fulfilled the criteria of a first Q-wave AMI with decreased global systolic function (mean angiographic LVEF at the acute phase = 46 ± 11%) and a severe residual stenosis of the IRA. In these patients, the detection of viable but jeopardized myocardium is important to select patients who can benefit from coronary revascularization, thus expecting functional improvement of segmental wall motion in the infarct zone (4,9). The need for revascularization to achieve functional recovery of viable but jeopardized myocardium after AMI has already been pointed out (4,21). In the study reported by Piérard et al. (21), most of the patients with viable myocardium but severely reduced perfusion did not show any improvement at 9 ± 7 months follow-up examination; this can be explained by the absence of revascularization of the IRA in most cases. Barilla et al. (4) also reported on myocardial viability assessed by DSE in the first week after an anterior AMI. The functional improvement of viable segments supplied by stenotic arteries was greater in the patients who underwent revascularization compared with patients who did not. Thus, our data confirm, in a homogeneous population of patients with recent myocardial infarction and viable but jeopardized myocardium at DSE, that delayed revascularization of the culprit vessel leads to a significant improvement of regional and global systolic LV function.

Reference method for segmental and global LV systolic function. All previous studies evaluating DSE to predict functional improvement of segmental myocardial asynergy have used echocardiography as the reference method for the evaluation of LV function at baseline and during follow-up examination (4–16). In the current study, global and regional LV function were assessed by cineangiography as a gold standard for functional improvement, regardless of echocardiographic data. Echocardiographic and angiographic data were analyzed by independent investigators blinded to the results of the alternative technique. The use of single-plane ventriculography to assess functional recovery could appear as a limitation by missing the recovery of segments outside the viewing plane (i.e., the septum or lateral wall). The three patients with sustained improvement...
at DSE who did not recover at follow up showed regional wall motion improvement of the anterior wall and the apex that are well imaged by the right-anterior oblique position at ventriculography. The three false positive patients with a biphasic response at DSE had inotropic reserve of the inferior (two cases) or anterior wall (one case) that are also well imaged by single-plane ventriculography. These findings may overcome the theoretical drawback that functional recovery in other segments could have been missed because of single-plane imaging.

Influence of infarct-vessel patency. Only a few studies dealing with the evaluation of myocardial viability by DSE have assessed the long-term patency of the revascularized vessels. Arnese et al. (13) reported angiographic data regarding coronary bypass graft patency in only 14/38 patients. Watada et al. (22) have evaluated the diagnostic value of DSE for predicting functional recovery of viable myocardium after AMI, but the infarct vessel was patent without residual stenosis in all cases at the time of DSE and at 25-day follow-up examination, indicating stunned rather than jeopardized myocardium in case of viability. In this study, all patients presented a severe stenosis or occlusion of the IRA at the time of DSE, and follow-up coronary angiograms were available in all cases. Our results clearly demonstrate that functional recovery after revascularization of viable but jeopardized myocardium was dependent on the status of the IRA at follow-up examination. In the patients with viable myocardium, regional systolic function as well as global ejection fraction improved after revascularization only if the IRA was patent at follow-up procedure, whereas in the case of late reocclusion of the IRA, the benefit of revascularization was lost despite the presence of initially viable myocardium at DSE. The diagnostic accuracy of DSE for predicting long-term recovery of LV function after revascularization may be influenced by late reocclusion of the IRA. In this study, we showed that in the patients with a patent IRA at follow-up procedure, the specificity and positive predictive value of DSE in predicting functional improvement increased (from 82% to 95% and from 71% to 93%, respectively), with only a slight decrease in the negative predictive value (from 90% to 82%). It is interesting to note that, in patients with a biphasic response at DSE and a patent IRA at follow-up procedure, the positive predictive value of DSE for functional recovery was 100%. These findings may suggest that, in patients with a biphasic response at DSE and a lack of subsequent improvement after revascularization, reocclusion of the IRA could explain the lack of functional recovery. Thus we suggest that the specificity and positive predictive value of DSE for predicting LV function recovery may be influenced by late reocclusion of the IRA. Previous studies have assessed the impact of late reocclusion of the IRA on regional LV function (17,18). Meijer et al. (17) evaluated the long-term patency of the IRA after thrombolysis in a subset of patients from the APRICOT study. Reocclusion of the IRA occurred in 26% of patients who showed no regional or global improvement of systolic LV function and a significant increase in LV indexed volumes. Conversely, improvement of LVEF and no significant LV remodeling occurred in patients with anterior AMI and a patent artery at follow-up examination. Garot et al. (18) have reported similar results in patients who underwent successful delayed PTCA of occluded IRA after AMI. In this study, regional and global LV systolic function improved without significant LV remodeling in patients with a patent IRA at four-month follow-up examination, in contrast with patients with a reoccluded vessel. However, despite the presence of viable myocardium, improvement of LV function was not consistently observed after revascularization because of late reocclusion of the IRA.

Frequency of myocardial viability. In this study, the presence of myocardial viability was demonstrated by DSE in 40% of the patients. In other studies (4,6), the proportion of inotropic reserve in patients assessed by DSE in the first week after AMI was higher than 70%, but in these studies the very short delay between AMI and DSE probably led to the detection of mainly stunned myocardium that is able to recover spontaneously within two weeks (23). Barilla et al. (4) reported 95% of dobutamine-responsive patients in a selected population of patients mainly with non-Q wave infarction. Our population is more likely to be compared with patients presenting chronic CAD (9,11) in whom the proportion of dobutamine-responsive patients is about 50%, which is comparable to our results.

Study limitations. Semi-quantitative analysis of segmental wall motion by WMSI during stress echocardiography could appear as a limitation. Nevertheless, the complete validation of DSE for the detection of myocardial viability is widely accepted. Furthermore, our results showed a fairly good correlation between the two observers for the echocardiographic segmental wall motion analysis. Because quantitative automated techniques for echocardiographic assessment of regional systolic function are not fully validated, semiquantitative assessment still remains the reference method in the clinical setting.

In our experience, the reocclusion rate of the IRA during follow-up procedure remains the major limitation of a strategy of delayed revascularization of the culprit vessel after AMI. In this study, the reocclusion rate at follow-up procedure in the case of occluded IRA at baseline was 47%, considering the fact that we performed mainly a standard balloon technique for the angioplasty procedure. It is likely that stent implantation may lead to a lower reocclusion rate in such patients.

CONCLUSIONS

Our results showed that DSE is a reliable tool to detect myocardial viability after AMI and to predict LV functional improvement after delayed revascularization of the culprit
vessel. When considering the long-term angiographic status of the IRA, this study demonstrates that functional recovery of viable myocardium may be prevented by the late reocclusion of the IRA, and that the diagnostic value of DSE to predict functional improvement may be influenced by the late reocclusion of the culprit vessel.

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Reprint requests and correspondence: Dr. J. L. Monin, Fédération de Cardiologie, Hôpital Henri Mondor, 51 av. Maréchal De Lattre de Tassigny, 94010 Créteil, France. E-mail: pascal.guertet@lmm.ap.hop.paris.fr.

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