Vasodilator Stress Echocardiography for Risk Stratification of Medically Stabilized Unstable Angina

G. Gigli*, L. Cortigiani, A. Vallebona, S. Orlandi, P. R. Mariani and C. Volterrani

Cardiovascular Units, Hospitals of Rapallo (Genova), and Lucca, Italy

Aims: The aims of this study were to assess the safety, feasibility and prognostic value of dipyridamole-atropine stress echo in patients with medically stabilized unstable angina.

Methods: The initial population consisted of 173 patients consecutively admitted at two different Coronary Care Units with class IIIB unstable angina. Of these, 56 were excluded: five had poor acoustic window, 24 did not stabilize with medical therapy and underwent urgent coronary angiography, 26 evolved in non-Q wave myocardial infarction and one patient died. The remaining 117 patients underwent dipyridamole-atropine stress echo after 48 h without symptoms or electrocardiographic evidence of myocardial ischaemia.

Results: No complications or side effects occurred. An ischaemic response was found in 61 patients. During follow-up (10 ± 9 months), three cardiac deaths, eight infarctions, 13 unstable anginas, and seven late (>3 months from stress testing) revascularizations occurred. There were 22 events (36%) in patients with, and nine events (16%) in patients without, inducible ischaemia (P=0.01). At Cox analysis peak-stress wall-motion score index (HR=5.5; 95% CI, 1.9 to 15.5; P=0.0015), and admission ST-segment depression (HR=4.2; 95% CI, 1.7 to 10.7; P=0.0022) were independent predictors of spontaneous events (cardiac death, infarction, unstable angina). The 12-month event-free survival was 69% for ischaemic and 83% for non-ischaemic group (P=0.03). In considering major events as end-points (spontaneous events, and late revascularization), again multivariate prognostic indicators were peak-stress wall-motion score index (HR=14.2; 95% CI, 2.6 to 76.6; P=0.0021), and admission ST-segment depression (HR=3.1; 95% CI, 1.4 to 6.9; P=0.0055). The 12-month event-free survival rate was 58% for ischaemic and 81% for non-ischaemic group (P=0.002). With an interactive step-wise procedure, stress echo findings were found to provide incremental prognostic contribution to that of clinical data alone.

Conclusions: With proper selection of patients, dipyridamole-atropine stress echo is extremely safe and feasible in patients with medically stabilized unstable angina, and can be useful in identification of subjects at risk for future cardiac events.

(Eur J Echocardiography 2002; 3: 59–66) © 2002 The European Society of Cardiology. Published by Elsevier Science Ltd. All rights reserved.

Key Words: Stress echocardiography; dipyridamole; prognosis; unstable angina; coronary artery disease.

Introduction

In patients with unstable angina, in whom symptoms are medically controlled and a conservative regimen is chosen, there is a high incidence of subsequent failure of medical therapy[1,2]. Several clinical[3–5], electrocardiographic[6–9], biochemical[10–12] and angiographic[13] indicators have been evaluated in an effort to predict coronary events. Diagnostic techniques employing physical[14–16] or pharmacologic stress[17,18] have been proposed for an effective risk stratification. As for vasodilator stress, dipyridamole test with radionuclide ventriculography has been recommended in these patients, and those with left ventricular functional abnormality should be referred to cardiac cath[19]. Such functional abnormalities can be assessed in a more cost-effective fashion by dipyridamole stress echo, as shown by extensive large scale experience in various patients' subset[20–24]. Data obtained with dipyridamole stress echo in patients with unstable angina are, however, conspicuously lacking to date.
The present study was designed prospectively to evaluate the safety, feasibility and prognostic value of dipyridamole-atropine stress echo in patients with medically stabilized unstable angina.

**Methods**

**Enrolment criteria**

Patients enrolled in the study were those consecutively admitted to the Coronary Care Units of Rapallo (Genova) and Lucca between November 1995 and October 1996, who met the following eligibility criteria: (1) class IIIb angina (i.e. typical chest pain at rest in preceding 48 h) (25); (2) ischaemic electrocardiographic changes and/or documented coronary heart disease. After admission patients were treated with aspirin, heparin, nitrates, beta blockers and calcium antagonists, individually or in combination, titrated on clinical and haemodynamic response. According to standardized clinical practice, cardiac enzymes were determined every 6 h for a minimum of 24 h. Myocardial infarction was diagnosed in case of rise in creatine kinase and CK-MB greater than twice the normal upper limit of laboratory. Standard 12-leads electrocardiogram was recorded twice a day or when necessary. Electrocardiographic changes were considered significant for ischaemia if a ST-segment shift ≥0·1 mV from baseline at 80 msec after the J point or a T wave pseudonormalization occurred in at least two contiguous leads. Patients who remained asymptomatic after 48 h of therapy were considered clinically stabilized and underwent stress echo. Those with severe angina (crescendo angina, pain lasting more than 20 min, angina with left ventricular dysfun ction) or with electrocardiographic evidence of recurrent myocardial ischaemia were urgently submitted to coronary angiography and excluded from the study accordingly. Patients who evolved in non-Q wave myocardial infarction as well as those with poor acoustic window, were also excluded.

**Dipyridamole-atropine stress echocardiography**

Two-dimensional echocardiography and 12-lead electrocardiographic monitoring were performed in combination with high dose (0·84 mg over 10 min) dipyridamole plus atropine (up to 1 mg) [26]. During the procedure, blood pressure and the electrocardiogram were recorded each minute. Echocardiographic images were recorded on VHS videotape in 35% of cases and/or digitally stored on magneto-optical disk in the remaining 65% for subsequent analysis. Images were evaluated independently by two expert observers. In case of disagreement a third observer evaluated the images, and its judgement was binding. In our experience, the intra- and inter-observer reproducibility in stress echo reading is 92% and 89%, respectively, as previously described [27]. Regional wall-motion was semiquantitatively assessed using a 16 segment model of the left ventricle [29]. A four-point score was assigned to each segment as follows: 1=normal; 2=hypokinesia; 3=akinesia; and 4=dyskinesia. A wall-motion score index was derived by dividing the sum of individual segment scores by the number of interpretable segments. Test was considered positive for ischaemia in case of new wall-motion abnormalities or worsening of pre-existing ones. However, akinesia becoming dyskinesia was not considered as a sign of ischaemia [29]. Positive test was defined at low or high dose when echo abnormalities appeared having infused ≤0·56 or >0·56 mg/kg of dipyridamole, respectively.

Non-echocardiographic criteria for ending the test were: peak dipyridamole + atropine dose, severe chest pain, and ST-segment depression ≥2 mm or elevation ≥1·5 mm. The test was also stopped in the case of (1) intolerable symptoms; (2) limiting side effects, including hypotension (decrease in systolic blood pressure >30 mmHg), supraventricular arrhythmias (supraventricular tachycardia or atrial fibrillation), ventricular arrhythmias (ventricular tachycardia, frequent, polymorphous premature ventricular beats), and bradyarrhythmias. Electrocardiographic changes were not taken as criteria for positivity of the test in the absence of induced new wall-motion abnormalities. However, the development of ST-segment depression ≥2 mm or elevation ≥1·5 mm was considered to be significant enough for interruption of the test.

**Follow-up data**

Follow-up data were collected following a review of the patient’s hospital chart, contact with the patient’s physician, telephone interview with the patient, and periodic visits in our outpatient clinic. Each event recorded was directly controlled by means of case sheet examination and/or medical visit. The clinical events recorded during the follow-up were cardiac death, non-fatal myocardial infarction, unstable angina requiring hospitalization, early (≤3 months) and late (>3 months) coronary revascularization (surgery or angioplasty). Death was defined as cardiac if strictly related to proved cardiac causes (such as fatal myocardial infarction, acute heart failure or malignant arrhythmias). The diagnosis of acute myocardial infarction was made on the basis of symptoms, electrocardiographic changes, and cardiac enzyme level increases. Late revascularization was considered as clinical end-point, reflecting progressive symptoms. The data were analysed for the prediction of spontaneous events (cardiac death, infarction, unstable angina) and major events (cardiac death, infarction, unstable angina, and late revascularization).

**Statistical analysis**

Values were expressed as mean ± SD for continuous variables and as frequency and percentage for...
categorical variables. Continuous variables were compared using the Student’s unpaired *t*-test, while differences of categorical variables were assessed by the chi-square test. The Kaplan–Meier curves were used for estimation of event-free survival. For survival analysis only one event was considered in each patient. Patients who underwent revascularization within 3 months from testing were censored at the time of the procedure to avoid post-test referral bias. The differences of survival curves were analysed using the log-rank test. The associations of selected variables with outcome were assessed with Cox proportional hazard models using univariate and stepwise multivariate procedures; these analysis were performed with the Statistical Package for the Social Sciences (SPSS, Chicago, Illinois). Furthermore, in an attempt to investigate the prognostic value of stress echo incremental to known clinical indicators (including patient clinical characteristics, electrocardiography and resting echo findings), an interactive stepwise procedure was performed, where variables were included into the model in the same order as in the clinical practice. Therefore, clinical data were firstly analysed and the global chi-square was calculated. Subsequently, a second step was created after addition of stress echo results to the independent predictors at first step. The incremental prognostic value of the added variables was assessed by comparison of the global chi-square at each step. At each step, a *P* value **≤ 0.01** was taken as the required level of significance for entering a variable into the model. The differences in risk were expressed as hazard ratio (HR) with the corresponding 95% confidence interval (CI). The variables included in the analysis were age (< or ≥ 65 years), sex, diabetes mellitus, hypertension, hypercholesterolaemia, cigarette smoking, previous myocardial infarction, admission electrocardiogram (ST-segment depression, ST-segment elevation, isolated T-wave changes), resting wall-motion score index, positive echo result, low-dose positive echo result, peak-stress wall-motion score index, and Δ wall-motion score index (i.e. the variation of wall-motion score index from resting condition to peak of stress). A value of *P* < 0.05 was considered statistically significant.

**Results**

**Patients**

During the study period, 173 patients were admitted at the two Coronary Care Units fulfilling the eligibility criteria. Of these, 56 were excluded: five had poor acoustic window, 24 did not stabilize with medical therapy and underwent urgent coronary angiography, 26 evolved in non-Q wave myocardial infarction and one patient died. The remaining 117 patients represented the study group and underwent stress echo on oral therapy with antianginal medications, individually or in combination (50 with beta-blockers, 62 with calcium antagonists, and 107 with nitrates), and off phylline-containing drugs for at least 24 h.

**Test result**

No major complications or side-effects requiring premature test interruption occurred. Moreover, no test was ended for ST-segment changes.

Echocardiographic positivity was detected in 61 (52%) patients, 22 at low- and 39 at high-dose (11 after atropine). Atropine was given in 54 of 56 patients without ischaemia; the peak-stress double product achieved with atropine was 15 904 ± 3873. In the remaining two patients atropine was not administered because of glaucoma (one case) and severe prostatic hypertrophy (one case). Agreement to the result of stress test was 95% with the two operators. The clinical, electrocardiographic and echocardiographic characteristics for the ischaemic and non-ischaemic group are shown in Table 1.

**Outcomes**

Follow-up data were available for all patients. During the follow-up (10 ± 9 months), there were 31 major events: three cardiac deaths, eight infarctions, 13 unstable anginas, and seven late revascularizations. Events occurred in 22/61 (36%) (two cardiac deaths, six infarctions, eight unstable anginas, and six late revascularizations) patients with and in 9/56 (16%) (one cardiac death, two infarctions, five unstable anginas, and one late revascularization) patients without inducible ischaemia (*P* = 0.01). Additionally 27 patients underwent early revascularization: 19/61 (31%) with and 8/56 (14%) without ischaemic result of test (*P* = 0.03). Furthermore, early revascularized patients were younger (63 ± 10 vs 69 ± 10 years; *P* = 0.01), more likely smokers (44 vs 22%; *P* = 0.02), and had higher peak-stress wall-motion score index (1.46 ± 0.42 vs 1.30 ± 0.34; *P* = 0.04) than those maintained on medical therapy.

**Survival analysis**

The predictors of spontaneous events at univariate analysis are listed in Table 2. At Cox analysis peak-stress wall-motion score index (HR = 5.5; 95% CI, 1.9 to 15.5; *P* = 0.0015), and admission ST-segment depression (HR = 4.2; 95% CI, 1.7 to 10.7; *P* = 0.0022) correlated independently with prognosis. The 12-month event-free survival was 69% for ischaemic and 83% for non-ischaemic group (*P* = 0.03) (Fig. 1).

The univariate predictors of major events are shown in Table 2. Independent prognostic indicators at multivariate analysis were peak-stress wall-motion score index (HR = 14.2; 95% CI, 2.6 to 76.6; *P* = 0.0021), and admission ST-segment depression (HR = 3.1; 95% CI, 1.4 to 6.9; *P* = 0.0055). The 12-month event-free survival was 58% for ischaemic and 81% for non-ischaemic group (*P* = 0.002) (Fig. 2).
In considering all events (major events and early revascularizations), the 12-month event-free survival was 38% for ischaemic and 69% for non-ischaemic group ($P=0.0001$) (Fig. 3).

### Table 1. Characteristics of the ischaemic and the non-ischaemic group.

<table>
<thead>
<tr>
<th></th>
<th>Positive stress echo (n=61)</th>
<th>Negative stress-echo (n=56)</th>
<th>$P$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>67 ± 10</td>
<td>67 ± 11</td>
<td>0.99</td>
</tr>
<tr>
<td>Males</td>
<td>40 (78%)</td>
<td>33 (60%)</td>
<td>0.46</td>
</tr>
<tr>
<td>Clinical history</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td>23 (38%)</td>
<td>17 (30%)</td>
<td>0.40</td>
</tr>
<tr>
<td>Previous revascularization</td>
<td>3 (5%)</td>
<td>5 (9%)</td>
<td>0.39</td>
</tr>
<tr>
<td>Previous effort angina</td>
<td>31 (51%)</td>
<td>16 (29%)</td>
<td>0.01</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>11 (18%)</td>
<td>10 (18%)</td>
<td>0.98</td>
</tr>
<tr>
<td>Hypertension</td>
<td>36 (59%)</td>
<td>35 (62%)</td>
<td>0.70</td>
</tr>
<tr>
<td>Hypercholesterolaemia</td>
<td>36 (59%)</td>
<td>30 (54%)</td>
<td>0.55</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>14 (23%)</td>
<td>18 (32%)</td>
<td>0.27</td>
</tr>
<tr>
<td>Admission electrocardiogram</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ST-segment depression</td>
<td>32 (52%)</td>
<td>23 (41%)</td>
<td>0.22</td>
</tr>
<tr>
<td>ST-segment elevation</td>
<td>10 (16%)</td>
<td>8 (14%)</td>
<td>0.75</td>
</tr>
<tr>
<td>Isolate T-wave changes</td>
<td>6 (10%)</td>
<td>10 (18%)</td>
<td>0.21</td>
</tr>
<tr>
<td>Normal pattern</td>
<td>13 (21%)</td>
<td>15 (27%)</td>
<td>0.49</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Resting wall-motion score index</td>
<td>1.21 ± 0.30</td>
<td>1.14 ± 0.22</td>
<td>0.15</td>
</tr>
<tr>
<td>Peak-stress wall-motion score index</td>
<td>1.52 ± 0.37</td>
<td>1.13 ± 0.22</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Data presented are mean value ± SD or number (%) of patients.

### Table 2. Predictors of spontaneous and major cardiac events at univariate analysis.

<table>
<thead>
<tr>
<th></th>
<th>Spontaneous events</th>
<th>Major events</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HR (95% CI)</td>
<td>$P$ value</td>
</tr>
<tr>
<td>A wall-motion score index</td>
<td>32.8 (5.4–197.9)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Peak-stress wall-motion score index</td>
<td>4.9 (1.8–13.7)</td>
<td>0.0022</td>
</tr>
<tr>
<td>Admission ST-segment depression</td>
<td>4.1 (1.6–10.3)</td>
<td>0.0029</td>
</tr>
<tr>
<td>Low-dose positive echo result</td>
<td>2.9 (1.2–6.7)</td>
<td>0.0154</td>
</tr>
<tr>
<td>Positive echo result</td>
<td>2.5 (1–5.8)</td>
<td>0.0381</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>0.3 (0.1–1.2)</td>
<td>0.0817</td>
</tr>
<tr>
<td>Resting wall-motion score index</td>
<td>2.7 (0.7–10.3)</td>
<td>0.1439</td>
</tr>
<tr>
<td>Admission isolate T-wave changes</td>
<td>0.2 (0.1–1.7)</td>
<td>0.1447</td>
</tr>
<tr>
<td>Female gender</td>
<td>1.7 (0.8–3.8)</td>
<td>0.1913</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1.6 (0.7–3.9)</td>
<td>0.2801</td>
</tr>
<tr>
<td>Hypercholesterolaemia</td>
<td>1.3 (0.6–2.8)</td>
<td>0.5836</td>
</tr>
<tr>
<td>Age ≤65 years</td>
<td>1.3 (0.5–3.1)</td>
<td>0.5905</td>
</tr>
<tr>
<td>Admission ST-segment elevation</td>
<td>1.2 (0.4–3.6)</td>
<td>0.6893</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td>1.2 (0.5–2.7)</td>
<td>0.7108</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.0 (0.4–2.2)</td>
<td>0.9339</td>
</tr>
</tbody>
</table>

HR=hazard ratio; CI=confidence interval.

In considering all events (major events and early revascularizations), the 12-month event-free survival was 38% for ischaemic and 69% for non-ischaemic group ($P=0.0001$) (Fig. 3).

### Incremental prognostic value of stress echo over clinical indicators

The global chi-square of the clinical model was 8.9 ($P=0.012$) for the prediction of spontaneous cardiac events. The addition of stress echo findings provided 129% incremental prognostic value (chi-square=20.4; $P<0.0001$).

When major cardiac events were primary end-points, the global chi-square of the clinical model was 18.2 ($P=0.0001$). With the inclusion of stress echo findings, 68% incremental prognostic contribution was observed (chi-square=30.6; $P<0.0001$).

### Discussion

In the acute phase of unstable angina a widely recommended management is early coronary angiography and revascularization, if clinical stabilization is not rapidly obtained with medical therapy or in patients who present at high risk for acute myocardial infarction.
crescendo angina, pain associated with myocardial dysfunction or lasting more than 20 min) [30]. About 80% of patients admitted to Coronary Care Unit with diagnosis of unstable angina, however, with appropriate medical therapy undergo clinical stabilization in 48 h [31]. This group of patients, indeed, represents the huge majority the clinical cardiologist is faced with and, in this setting, risk stratification is the most important challenge. Adverse events are common in these patients: from 14 to 57% in various studies [1, 2], similar to the 26% we described in our total group. Therefore, the identification of patients at higher risk to be treated aggressively is mandatory. On the other hand, it is particularly important to identify patients at low risk, who could be treated by conventional medical therapy without expensive and high-risk procedures and treatment. So, in practice, unstable angina (as well as many acute syndromes) needs a bimodal decision-making approach: in the acute phase it is necessary to identify high-risk patients to be treated aggressively, whereas after stabilization it is important to identify low-risk patients to be discharged early and treated by conventional medical therapy. In our work we excluded patients with non-Q wave myocardial infarction, as well as patients at high immediate risk, the resulting population being represented by low- to intermediate-risk subjects. Many clinical [3–5] and electrocardiographic variables have been identified that correlate with subsequent events: ST-segment shift, both at entry and during anginal episodes [6, 7], and ischaemic burden at Holter monitoring [8, 9]. Biochemical markers of subclinical myocardial necrosis have also been proven useful for identification of patients at high risk [10–12]. Coronary angiography seems to have limited value when used to stratify the risk of patients after an episode of unstable angina. In fact, neither the number nor the complexity of morphology of coronary lesions seems to be able to predict future events that depend mainly on plaque progression [32].

Figure 1. Cumulative survival rates free of spontaneous cardiac events (cardiac death, non-fatal infarction, unstable angina) in patients with (+) and without (−) inducible ischaemia at stress echocardiography (SE).

Figure 2. Cumulative survival rates free of major cardiac events (cardiac death, non-fatal infarction, unstable angina, late revascularization) in patients with (+) and without (−) inducible ischaemia at stress echocardiography (SE).

Figure 3. Cumulative survival rates free of cardiac events (cardiac death, non-fatal infarction, unstable angina, early and late revascularization) in patients with (+) and without (−) inducible ischaemia at stress echocardiography (SE).
previous evidence suggesting (1) the diagnostic and prognostic value of vasodilator stress echo; (2) the usefulness of cardiac stress imaging techniques in patients with unstable angina; and (3) the importance of vasodilator stress echo stratification in the space domain for a more accurate titration of risk.

Vasodilator stress echo is an established diagnostic option. For chronic ischaemic heart disease, guidelines of American Heart Association/American College of Cardiology clearly state that ‘stress echocardiography by either exercise or pharmacologic challenge (using vasodilators or dobutamine) is both sensitive and specific for detecting inducible myocardial ischaemia in patients with intermediate to high pretest probability of disease’[33]. For risk stratification, ‘when patients cannot exercise for a variety of reasons, pharmacologic stress echocardiography is a valuable alternative for evaluation of residual myocardial at risk’[33]. Our study extends to unstable angina the results previously obtained with vasodilator stress echo in other subsets of patients with coronary heart disease[20–24] and are congruent to that previously reported with other cardiac stress imaging techniques, including exercise electrocardiography[14], perfusion scintigraphy[15,17] and echocardiography combined with physical[16] or adrenergic stress[18]. This suggests that vasodilator stress echo can help in risk stratification of medically stabilized patients with unstable angina. However, the prediction allowed by vasodilator stress echo as well as by other stress testing is imperfect in this setting, and a negative stress echocardiogram does not necessarily mean a good prognosis, but implies some risk of adverse events. This is consistent with what is known on the pathophysiology of coronary events in patients with coronary heart disease and especially with unstable angina[35]. In fact, a physiologic approach, such as the one that uses stress testing, cannot predict such phenomena as fissuration, embolization, inflammation, ulceration and thrombosis, which are largely unrelated to the haemodynamic severity of the plaque. Since data obtained from specifically designed study are still lacking, our personal opinion is that patients with a negative stress echocardiogram have to be intensively medically treated and strictly followed with frequent clinical evaluation.

We found low dose positivity to have a lower univariate power in predicting major events than the positivity of the test. This finding is consistent with the fact that major events included late revascularizations, that better reflect the total atherosclerotic burden of the coronary circulation rather than the pathophysiological background of the acute phase of the illness. Although this study was not designed to specifically evaluate the extent of wall-motion abnormality and the extent and severity of coronary heart disease, our results agree with an extreme body of evidence suggesting that the extent of wall-motion dysfunction during stress echo is even more important than its presence. The higher the peak-stress wall-motion score index, the more extent and severe the underlying angiographically assessed coronary heart disease[30], and the worse the prognosis[36]. Our data demonstrate that the risk stratification with vasodilator stress echo can be obtained on the basis of peak-stress wall-motion score index also in patients with unstable angina.

Study limitations

Patient management was not standardized and the decision of medical or invasive treatment was made by the referring physician on the basis of the clinical, anatomic and stress echo findings. Consequently, a significantly greater percentage of patients with inducible ischaemia underwent early revascularization compared with those without ischaemia (P=0.03). This might have contributed to underscore the positive predictive value of the test, due to the known beneficial effects of ischaemia-guided revascularization[37]. On the other hand, this is explanatory of the relatively low 69% 12-month event-free survival (including major events, and early revascularizations) observed in patients without inducible ischaemia.

Patients were studied on antiangiial therapy. Beta blockers, nitrates and/or calcium antagonists are known to lower dipyridamole stress echo sensitivity for coronary heart disease[38], and thus might have significantly reduced the predictive value of a negative test. Nevertheless, therapy withdrawal in these patients would have been impractical, and possibly unethical.

Clinical implications

In low- to moderate-risk patients with unstable angina who stabilize under medical treatment there is no consensus as to management, since there is no evidence that an early invasive approach provides benefits in terms of prognosis compared with a conservative one[39–41]. Therefore, the prognostic stratification by non-invasive techniques is a recommended option[30]. Our results show that vasodilator stress echo performed early after a minimum of 48 h of medical stabilization in patients with unstable angina represents a possible approach to risk stratification, providing additional independent prognostic value to known clinical and electrocardiographic risk indicators. Information from pharmacologic stress echo can be combined with clinical information to assist the decision on the necessity for further investigation and coronary revascularization.

References


[34] The TIMI IIIB Investigators. Effects of tissue plasminogen activator and a comparison of early invasive and conservative
