Prognostic Importance of Dipyridamole-Echocardiography Test in Coronary Artery Disease

Eugenio Picano, MD, Silva Severi, MD, Claudio Michelassi, Fabio Lattanzi, MD, Michele Masini, MD, Enrico Orsini, MD, Alessandro Distante, MD, and Antonio L’Abbate, MD

We studied the value of dipyridamole-echocardiography test in comparison with clinical, resting electrocardiogram and echocardiogram variables in predicting cardiac events occurring in 539 consecutive patients referred for dipyridamole-echocardiography test from 1984 to 1987. There were 118 cardiac events: 11 cardiac deaths, 12 nonfatal myocardial infarctions, and 95 coronary revascularization (bypass or angioplasty) procedures. A Cox survival analysis identified echocardiographic positivity after dipyridamole administration as the best predictor of cardiac events (relative risk ratio, 2.7). The next most powerful predictor was angina after dipyridamole administration (relative risk ratio, 1.9). Cardiac events occurred in 14 (6%) of 253 patients with normal high-dose dipyridamole echocardiographic test results, in 21 (26%) of 82 patients with high-dose dipyridamole echocardiographic positivity (0.84 mg/kg during 10 minutes), and in 83 (41%) of 204 patients with low-dose dipyridamole echocardiographic positivity (0.56 mg/kg during 4 minutes) (p<0.0001). In a subset of 341 patients, exercise electrocardiography stress test and coronary angiography were also available. A Cox survival analysis again identified echocardiographic positivity after dipyridamole as the best predictor of cardiac events (relative risk ratio, 1.9) followed by a pathologic coronary arteriography (relative risk ratio, 1.2). We conclude that the presence and timing of a transient dyssynergy during dipyridamole stress are useful predictors of subsequent cardiac events. (Circulation 1989;80:450–457)

One of the most important roles of cardiologists in the management of coronary artery disease is to identify patients at high risk of future cardiac events so that interventions to prevent these events may be considered. Traditionally, noninvasive variables, such as patient history, electrocardiographic findings, and results of exercise testing, have been used in prognostication.1–8 However, the attainment of diagnostically useful data requires that a level of exercise be achieved that increases myocardial oxygen demand substantially. Unfortunately, many patients are incapable of sufficiently vigorous exercise. Furthermore, the electrocardiographic markers of ischemia (used in exercise electrocardiography) are severely limited in many subgroups of patients, such as hypertensives, women, patients receiving digitalis or antiarrhythmic drugs, or patients with alterations on the resting electrocardiogram.9,10

Dipyridamole testing is gaining popularity as a nonexercise-dependent method to diagnose coronary artery disease.11–17 In particular, the high-dose dipyridamole echocardiographic test has been shown to be relatively inexpensive, feasible, safe,18 highly specific for the diagnosis of angiographically assessed coronary artery disease,19–21 and to have a sensitivity similar to that of dipyridamole-thallium scanning,22 exercise-thallium scanning,23 and exercise echocardiography.24

On the basis of these observations, we hypothesized that positivity of the dipyridamole-echocardiography test (DET) would be a useful prognostic finding. Therefore, this study was undertaken to compare the ability of clinical, resting electrocardiogram, resting echocardiogram, and DET variables to predict cardiac events during a subsequent 3-year follow-up in 539 consecutive patients. Because the prognostic value of the test has not been defined up to now, patients’ outcomes were not affected by the test results with respect to

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performance of cardiac catheterization and coronary artery bypass surgery on the basis of the DET results alone. Data on exercise stress test, ejection fraction by ventriculography, and coronary angiography were also available in a subset of 341 patients, and the prognostic value of these data could be compared, in this subset, with clinical, resting electrocardiogram, resting echocardiogram, and DET variables.

Methods

Patients

The study population consisted of 539 consecutive patients who underwent DET at the Institute of Clinical Physiology from 1984 to 1987. Patients were referred to our Institute (436 patients) or seen in our out-patient clinic (103 patients) for chest discomfort or for evaluation after an infarction. Patients with overt congestive heart failure, obvious global left ventricular dysfunction by Doppler echocardiographic criteria, clinically important valvular heart disease, previous coronary artery bypass surgery, or recent acute myocardial infarction were excluded. Four hundred twenty-one patients were not taking antiangular medications at the time of DET; 118 patients were continued on their baseline regimen of antiangular medications on the day of DET. Baseline information was collected prospectively at the time of DET. The variables examined in the present study consisted of multiple descriptors of the history, results of the electrocardiogram, and results of DET, which were easily recorded in all patients.

Dipyridamole-Echocardiography Test

Two-dimensional echocardiographic and 12-lead electrocardiographic monitoring were performed in combination with dipyridamole infusion (0.56 mg/kg during 4 minutes, followed by 4 minutes of no dose and then 0.28 mg/kg during 2 minutes; the cumulative dose was therefore 0.84 mg/kg during 10 minutes).17 This infusion protocol was used because we previously showed that a higher sensitivity could be achieved with a higher dipyridamole dosage, with no loss in specificity and no apparent increase in risk.17 Aminophylline (240 mg), which promptly reverses the effect of dipyridamole, was on hand. During the procedure, blood pressure was recorded each minute with a cuff sphygmomanometer. Two-dimensional echocardiograms were continuously recorded during and up to 20 minutes after the dipyridamole administration. A commercially available wide-angle phased-array imaging system (Hewlett-Packard 77020, 2.25- and 3.5-MHz transducers) was used. In the baseline studies, all standard echocardiographic views were obtained when possible. During the test, new areas of abnormal wall motion were identified on multiple views by rapidly moving the ultrasound transducer through various positions. When the optimal position for observing the abnormal wall motion was established, the transducer was kept steady throughout the study. Segmental anatomy and wall motion were assessed in a qualitative manner as previously reported.16,17 Wall motion was graded as hyperkinetic, normal, hypokinetic, akinetic, and dyskinetic. The videotapes were analyzed by two independent observers. Positivity of the test was based on the detection of a transient asynergy of contraction, which was absent or of a lesser degree in the baseline examination. The grading and the regional localization of the asynergy were decided by consensus between the two observers. When there was disagreement on the results, a third observer reviewed the study, and his judgment was binding.

A positive electrocardiographic response was defined as an ST segment shift of 1 mm or more, both at the J point and 80 msec later.

Intraobserver variability was assessed by one of the investigators who reevaluated 100 studies, selected at random, at least 1 month after the first interpretation and without knowledge of the first evaluation.

In the 539 studies, the assessment of positivity compared with negativity was unanimous in 510. In the remaining 29 studies, a split decision occurred. The intraobserver agreement was 98% in the studies reanalyzed by the same observer. This high level of interobserver and intraobserver agreement was made possible by several factors. The quality of echocardiographic tracings was as good after dipyridamole administration as during baseline conditions. The readers agreed before the study not to record minor degrees of hypokinesia; primary reliance was placed on changes from baseline to peak dipyridamole administration, and the investigators had previous experience in joint reading.16,17

Exercise Electrocardiography

All patients performed a multistage bicycle ergometer test, with an initial load of 25 W and subsequent increments of 25 W every 2 minutes. A 12 lead electrocardiogram and blood pressure determination were performed at baseline and every minute afterward. Criteria for interrupting the test were severe chest pain, diagnostic ST segment shift, fatigue, limiting dyspnea, or maximal predicted heart rate in absence of ischemia.

Electrocardiographic tracings were considered diagnostic for myocardial ischemia when there was an ST segment shift of at least 0.15 mV, 0.08 second after the J point, compared with baseline.

Angiographic Study

All patients underwent selective coronary arteriography, with either the Judkins or the Sones technique. Multiple views of each vessel were filmed. A vessel was considered to have significant obstruction if its diameter was narrowed by 50% or more with respect to the prestenotic tract.

Two independent observers who were unaware of echocardiographic and clinical data analyzed coronary angiograms for the degree of stenosis in
the right, left main, left anterior descending (or diagonal), and left circumflex (or marginal) coronary arteries. When there was disagreement over the degree of stenosis, a third observer reviewed the study, and his judgment was binding.

The ejection fraction was calculated from left ventriculograms according to the Brown-Dodge method.

**Follow-up**

Follow-up data were obtained from at least two of four sources: review of the patient’s hospital record, personal communication with the patient’s physician and review of the patient’s chart, a telephone interview with the patient conducted by trained personnel, a staff physician visiting the patients at regular 6-month or 1-year intervals in our outpatient clinic. Follow-up data were obtained in all patients.

The outcome events were all causes of death for survival, hard cardiac events (defined as cardiac death or nonfatal myocardial infarction) for infarction-free survival, and total cardiac events (cardiac death, nonfatal myocardial infarction, and coronary revascularization procedures) for event-free survival. Only one event was considered in each patient, and any event occurring after the initial event was not considered. With regard to survival and infarction-free survival, the follow-up times of patients who had surgery were censored at the time of surgery; likewise, for infarction-free survival and event-free survival, times of patients who died for noncardiac death were censored at the time of death. There is controversy over whether or not coronary artery bypass surgery and coronary angioplasty should be considered cardiac events. They are likely to reflect the presence of severe disease. However, the decision to perform these procedures may be subjective and not by itself an adverse outcome. Therefore, the data were also analyzed with only death and myocardial infarction as adverse cardiac events.

**Statistical Analysis**

Kaplan-Meier life table estimates of survival, of infarction-free survival, and of event-free survival were used to summarize the follow-up experience in these patients and to clarify presentation. The individual effect of certain variables on survival, infarction-free survival, and event-free survival was evaluated with the use of the Cox regression model (BMDP P2L, Department of Biomathematics, University of California at Los Angeles). According to a stepwise selection process, variables were entered, or removed, from the regression equation on the basis of a computed significance probability (maximized partial-likelihood ratio).

The patients were stratified into two subgroups: those with and those without previous myocardial infarction.

Variables examined were age, sex, arterial hypertension, previous myocardial infarction, presence of electrocardiographic alterations in basal conditions, presence of resting asynergy in the basal echocardiogram, echocardiographic positivity during dipyridamole test, chest pain during the test, and ST-T changes during the test. For patients with positive DET, two subsets were also considered: patients with positivity within 4 minutes after the low-dose dipyridamole (0.56 mg/kg during 4 minutes); patients with positivity after the full-dose dipyridamole (0.84 mg/kg during 10 minutes).

For the subset of 341 patients in whom data on exercise stress test, coronary angiography, and ejection fraction by ventriculography were also available, these variables were examined and added to the variables described in the previous paragraph.

The $\chi^2$ test and the Fisher’s exact test were used for comparing discrete variables. The required level of significance was $p$ less than 0.05.

**Results**

The anamnestic and clinical baseline characteristics of the 539 patients are listed in Table 1. The angiographic and ventriculographic features of the 374 patients who underwent coronary angiography and the results of exercise electrocardiography in 401 patients are reported in Table 2.

During the follow-up, there were 13 deaths (11 cardiac and two noncardiac related), 12 nonfatal myocardial infarctions, and 95 coronary revascularization procedures (bypass or angioplasty). Coronary anatomy features in the study patients, with and without revascularization procedures are reported in Table 3. When patients with and without myocardial infarction were compared, there were proportionally more coronary revascularization procedures in the group with a previous myocardial infarction ($p<0.01$); no significant difference was found regarding death and myocardial infarction during the follow-up between the two groups (Table 4).
Table 2. Angiographic Results and Exercise Stress Test Results

<table>
<thead>
<tr>
<th>Coronary artery disease</th>
<th>Positive DET (n=227)</th>
<th>Negative DET (n=147)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ejection fraction</td>
<td>58±13</td>
<td>64±8</td>
</tr>
</tbody>
</table>

Angiographic results in 374 patients and exercise stress test results in 401 patients. Values in parentheses are percentages. DET, dipyridamole-echocardiography test; ECG, electrocardiography.

Survival Analysis

The univariate prognostic factors are shown in Table 5. The most important predictor was the result of DET; 3-year survival according to the results of DET is depicted in Figures 1, 2, and 3.

With stepwise regression, only DET was an independent predictor of death (p<0.0001; relative risk ratio, 4.2). Even when cardiac death and myocardial infarction were considered as significant cardiac events, positive DET remained the only significant predictor of future hard cardiac events (p<0.001; relative risk ratio, 2.2). Using the Cox model, when all cardiac events were considered, we found that the significant independent predictors of future cardiac events were the positivity of DET and the appearance of chest pain during the test (Table 6). The positivity of DET was the most powerful predictor of a cardiac event whether or not patients had a previous myocardial infarction. The distribution of events according to the results of DET and the presence or absence of a previous myocardial infarction is shown in Table 7.

Comparison of Dipyridamole-Echocardiography Test With Exercise Testing, Ejection Fraction, and Coronary Angiography

In the subset of 341 patients in whom cardiac catheterization and exercise stress test data were also available, the univariate analysis for all cardiac events is shown in Table 8. Because of the small number of events in this relatively limited subset, only the predictability for all cardiac events was considered statistically meaningful and therefore analyzed. The most important predictor was the result of DET, followed by a pathologic coronary arteriography (number of diseased vessels). Using the Cox model, we found that the significant independent predictors of future cardiac events were the positivity of DET (p<0.000; relative risk ratio, 1.9), a pathologic coronary arteriography (p<0.006; relative risk ratio, 1.2) and age (p=0.042; relative risk ratio, 1.0) (Table 9).

Discussion

This study shows that among a group of clinical and resting electrocardiographic and echocardiographic variables a transient myocardial dyssnergy after dipyridamole infusion is the most powerful independent predictor of future cardiac events. During the 3-year follow-up, cardiac events occurred in 41% of patients with low-dose DET positivity of DET compared with 26% of patients with high-dose DET positivity (Table 10).

Table 3. Coronary Artery Disease in Patients With and Without Revascularization Procedures

<table>
<thead>
<tr>
<th>Coronary artery disease</th>
<th>Positive DET</th>
<th>Negative DET</th>
</tr>
</thead>
<tbody>
<tr>
<td>CABG No CABG</td>
<td>CABG No CABG</td>
<td>CABG No CABG</td>
</tr>
<tr>
<td>One vessel</td>
<td>33</td>
<td>45</td>
</tr>
<tr>
<td>Two vessel</td>
<td>31</td>
<td>33</td>
</tr>
<tr>
<td>Three vessel</td>
<td>32</td>
<td>26</td>
</tr>
<tr>
<td>Left main</td>
<td>13</td>
<td>5</td>
</tr>
</tbody>
</table>

DET, dipyridamole-echocardiography test; CABG, coronary artery bypass graft revascularization procedures.

Table 4. Events According to the Presence or Absence of a Previous Myocardial Infarction

<table>
<thead>
<tr>
<th></th>
<th>Death</th>
<th>Acute myocardial infarction</th>
<th>CABG or PTCA</th>
<th>Total cardiac events</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total patients (N=539)</td>
<td>13** (2.4)</td>
<td>12 (2.2)</td>
<td>95 (17.6)</td>
<td>118 (21.5)</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No (n=346)</td>
<td>8* (2.3)</td>
<td>6 (1.7)</td>
<td>48 (14)</td>
<td>61 (17.7)</td>
</tr>
<tr>
<td>Yes (n=193)</td>
<td>5* (2.6)</td>
<td>6 (3)</td>
<td>47† (24)</td>
<td>57† (29.5)</td>
</tr>
<tr>
<td>Negative DET (n=253)</td>
<td>1* (0.4)</td>
<td>4 (1.6)</td>
<td>10 (3.9)</td>
<td>14 (5.5)</td>
</tr>
<tr>
<td>Positive high-dose DET (n=82)</td>
<td>1 (1.2)</td>
<td>4 (4.9)</td>
<td>16 (19.5)</td>
<td>21 (26)</td>
</tr>
<tr>
<td>Positive low-dose DET (n=204)</td>
<td>11* (5.4)</td>
<td>4 (1.9)</td>
<td>69 (34)</td>
<td>83 (41)</td>
</tr>
</tbody>
</table>

Values are absolute numbers and percentages (parentheses) of events.
CABG, coronary artery bypass grafting; PTCA, percutaneous transluminal coronary angioplasty; DET, dipyridamole-echocardiography test.
*Noncardiac deaths for each asterisk.
†Significantly different (p<0.01) from the value obtained in patients without myocardial infarction.
TABLE 5. Univariate Predictors of Prognosis

<table>
<thead>
<tr>
<th></th>
<th>Deaths</th>
<th></th>
<th>Hard events</th>
<th></th>
<th>All events</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( \chi^2 )</td>
<td>( p )</td>
<td>( \chi^2 )</td>
<td>( p )</td>
<td>( \chi^2 )</td>
<td>( p )</td>
</tr>
<tr>
<td>Positive DET</td>
<td>15.7</td>
<td>0.0001</td>
<td>9.9</td>
<td>0.001</td>
<td>82.9</td>
<td>0.00001</td>
</tr>
<tr>
<td>ECG changes during DET</td>
<td>11.1</td>
<td>0.001</td>
<td>4.7</td>
<td>0.03</td>
<td>27.7</td>
<td>0.00001</td>
</tr>
<tr>
<td>Pain during DET</td>
<td>8.6</td>
<td>0.003</td>
<td>4.3</td>
<td>0.04</td>
<td>37.3</td>
<td>0.00001</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td>0.15</td>
<td>NS</td>
<td>1.3</td>
<td>NS</td>
<td>10.1</td>
<td>0.001</td>
</tr>
<tr>
<td>Abnormal basal echocardiogram</td>
<td>0.9</td>
<td>NS</td>
<td>0.2</td>
<td>NS</td>
<td>8.2</td>
<td>0.004</td>
</tr>
<tr>
<td>Age</td>
<td>1.8</td>
<td>NS</td>
<td>1.91</td>
<td>NS</td>
<td>7.2</td>
<td>0.007</td>
</tr>
<tr>
<td>Abnormal basal ECG</td>
<td>0.22</td>
<td>NS</td>
<td>0.24</td>
<td>NS</td>
<td>4.4</td>
<td>0.03</td>
</tr>
<tr>
<td>Sex</td>
<td>0.52</td>
<td>NS</td>
<td>3.09</td>
<td>NS</td>
<td>3.0</td>
<td>NS</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.1</td>
<td>NS</td>
<td>0.05</td>
<td>NS</td>
<td>1.07</td>
<td>NS</td>
</tr>
</tbody>
</table>

All values are approximate and refer to the predictive value of each variable fitted separately to a Cox proportional hazard model. DET, dipyridamole-echocardiography test; ECG, electrocardiogram.

DET positivity and 6% of patients with DET negativity. DET positivity added significantly to a model for the prediction of subsequent cardiac events, which included other variables such as a previous myocardial infarction and a resting asynergy on the echocardiogram.

There may be multiple reasons for the strong prognostic stratification operated by DET. The most obvious, and probably the most important, is the high diagnostic accuracy of DET for the detection of angiographically assessed coronary artery disease. \(^{20,21}\) Furthermore, there is a marked gradient in sensitivity of DET from single to multivessel coronary disease, and the extent of coronary atherosclerosis, angiographically assessed, is known to be a powerful prognostic index. \(^{25}\)

Also, the presence of angina and ST segment shift during dipyridamole infusion showed some prognostic power but showed markedly less than echocardiographic positivity. This is not surprising because chest pain and electrocardiographic changes are markers of dipyridamole-induced ischemia but are much less specific and slightly less sensitive than regional dyssynergy. \(^{20,21,26}\) The mechanisms of dipyridamole-induced ischemia are complex and still incompletely understood. The occurrence of flow maldistribution phenomena is however likely to play a major role, whereas the increase in myocardial oxygen demand is only trivial and of minor importance in triggering ischemia. \(^{27}\) Five mechanisms that could result in a decrease in myocardial oxygen supply are suggested: passive collapse of

FIGURE 1. Cumulative survival rates in patients of group A (negative dipyridamole-echocardiography test), group B (positive high-dose dipyridamole-echocardiography test), and group C (positive low-dose dipyridamole-echocardiography test).

FIGURE 2. Cumulative survival rates in patients free of myocardial infarction of group A (negative dipyridamole-echocardiography test), group B (positive high-dose dipyridamole-echocardiography test), and group C (positive low-dose dipyridamole-echocardiography test).
the stenosis, vertical steal, horizontal steal, systemic steal, and luxury perfusion.\textsuperscript{27} Each of these mechanisms results from coronary arteriolar vaso-
dilation induced by dipyridamole because of accumulation of adenosine. The final result of all these mechanisms is a drop in the subendocardial "metabolically useful" flow. This is a prerequisite for regional contractility dysfunction, revealed as a transient asynergy by the DET.

Other features may justify the prognostic power of DET, which was even higher than that for coronary arteriography. The DET positivity is more closely linked to the physiologic significance than to the anatomic appearance of the coronary stenosis.\textsuperscript{28} This has a pathophysiologic and also a potential prognostic importance because "less anatomy/more physiology" is required for an accurate prognostic stratification in the ischemic patient.\textsuperscript{29} Finally, the DET positivity is not affected by functional factors, such as coronary tone modulations or coronary vasospasm,\textsuperscript{30–32} which may elicit the positivity of other stress procedures, such as exercise stress test.\textsuperscript{33} We found a greater incidence of cardiac events in patients with low-dose DET positivity. This finding can be explained by the results of a previous study, which showed that the earlier is the

<table>
<thead>
<tr>
<th>Variable</th>
<th>Events</th>
<th>Relative risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive DET</td>
<td>52.3</td>
<td>0.000</td>
</tr>
<tr>
<td>Pain during DET</td>
<td>6.7</td>
<td>0.009</td>
</tr>
</tbody>
</table>

Each $\chi^2$ and $p$ value is a measure of the improvement that each selected variable makes in a model containing all other significant variables. The relative risk is the independent risk of a cardiac event that is associated with a variable. The total risk is the product of the risks associated with each variable when present.

DET, dipyridamole-echocardiography test.

<table>
<thead>
<tr>
<th>Event</th>
<th>Deaths</th>
<th>Acute MI</th>
<th>CABG or PTCA</th>
<th>No events</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No previous MI (n=346)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative DET</td>
<td>1* (0.5)</td>
<td>2 (1.0)</td>
<td>5 (2.5)</td>
<td>193 (96)</td>
<td>201</td>
</tr>
<tr>
<td>Positive high-dose DET</td>
<td>0</td>
<td>2 (4.5)</td>
<td>12 (27)</td>
<td>30 (68)</td>
<td>44</td>
</tr>
<tr>
<td>Positive low-dose DET</td>
<td>7 (6.9)</td>
<td>2 (2.0)</td>
<td>31 (31)</td>
<td>61 (60)</td>
<td>101</td>
</tr>
<tr>
<td>Previous MI (n=193)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative DET</td>
<td>0</td>
<td>2 (3.8)</td>
<td>5 (10)</td>
<td>45 (86)</td>
<td>52</td>
</tr>
<tr>
<td>Positive high-dose DET</td>
<td>1 (2.6)</td>
<td>2 (5.3)</td>
<td>4 (11)</td>
<td>31 (81)</td>
<td>38</td>
</tr>
<tr>
<td>Positive low-dose DET</td>
<td>4* (3.9)</td>
<td>2 (1.9)</td>
<td>38 (37)</td>
<td>60 (58)</td>
<td>103</td>
</tr>
</tbody>
</table>

Values are absolute numbers and percentages (parentheses) of events.

MI, myocardial infarction; CABG, coronary artery bypass graft; PTCA, percutaneous transluminal coronary angioplasty; DET, dipyridamole-echocardiography test.

*Noncardiac death for each asterisk.
echocardiographic positivity during dipyridamole infusion, the lower is the ischemic ceiling on exercise stress test. In fact, the single most important item in determining prognosis of the ischemic patient is generally acknowledged to be exercise tolerance, with steady worsening of prognosis as exercise tolerance lessens.

One potential limitation of this study is the qualitative and subjective assessment of myocardial contractility with echocardiography. However, this method is very simple, reliable, not time consuming, and therefore widely adopted for clinical echocardiographic studies in ischemic heart disease. This visual assessment is generally accepted to be at least as accurate as most quantitative methods for detection of transient dyssynergy because "the eye naturally integrates space and time, and its discriminatory power is difficult to reproduce, much less surpass." Two important limitations of stress echocardiography must be recognized: the periodic failure to obtain adequate images and the present lack of automation during analysis so that accurate interpretation depends on the subjective skills of the interpreting physician. With current instrumentation, the failure rate for adequate images in resting conditions is substantially lower than 10%. During dipyridamole administration, images are of unchanged quality in comparison with the resting state; in fact, there is no hyperventilation, tachycardia, and excessive chest wall movement that severely limit the acoustic approach during other stressing procedures, such as exercise. Regarding the second limitation of subjective observer analysis, current developments in computer-assisted methods for digital image processing, creation of continuous loops and split screen analysis, image enhancement, and better endocardial border recognition are expected to further improve the assessment of wall motion abnormalities and possibly lead to establishment of more accurate methods of quantification.

In conclusion, analysis of DET results adds important prognostic information compared with other clinical, resting electrocardiographic, or echocardiographic variables. The prognostic stratification by DET appears more accurate than that by variables from other procedures, such as assessment of coronary stenosis by angiography, and other widely used stressing procedures, such as exercise electrocardiography. The presence of DET positivity, especially after the low-dose dipyridamole, places a patient at a very high risk of subsequent cardiac events and should prompt consideration of aggressive therapy.

References


KEY WORDS • atherosclerosis • myocardial ischemia • prognosis • echocardiography • coronary artery disease
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