Prognostic Value of Exercise Testing, Coronary Angiography and Left Ventriculography 6–8 Weeks After Myocardial Infarction

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SUMMARY This study provides data on a consecutive series of 179 survivors of acute myocardial infarction (MI) who had symptom-limited treadmill exercise testing, coronary angiography and left ventriculography within 6–8 weeks after infarction. No patient died. The prevalence of multivessel disease was higher in the symptomatic survivors (79%) (p < 0.001). The prevalence of multivessel disease in inferior MI was 63% and in anterior MI 42% (p < 0.001). Left ventricular impairment was more severe in anterior and preexisting MI than in inferior and nontransmural MI (p < 0.005).

During a mean follow-up of 28 months, 11 cardiac deaths and 12 reinfarctions occurred. The total mortality rate was 22% (10 of 46) in patients with an ejection fraction < 30% or three-vessel disease and 1% (one of 133) in patients with an ejection fraction ≥ 30% and one- or two-vessel disease (p < 0.001). A group at high risk of mortality is thus identified by angiography. The total reinfarction rate was 9% (11 of 121) in patients with an exercise tolerance of less than 10 minutes (Bruce protocol) and 2% (one of 58) in patients with an exercise tolerance of 10 minutes or more (p < 0.1). The 58 patients who had an exercise tolerance of 10 minutes or more had a very low risk for cardiac death or reinfarction.

The identification of risk factors is of considerable importance in patient management because it may provide the opportunity to improve prognosis. Identification of high-risk patients 6–8 weeks after myocardial infarction (MI) has been attempted in this study. Little information is available that relates the extent of coronary artery disease and severity of left ventricular dysfunction to the prognostic risk of survivors.4 Although the prognostic value of exercise testing soon after MI has been established,5–11 few studies offer information about the prognostic value of coronary anatomy and left ventricular function in these patients.1–3

The aims of the present study were to define exercise test variables, coronary anatomy, and left ventricular function 6–8 weeks after infarction and to assess retrospectively the prognostic value of data obtained in defining morbidity and mortality after assessment at 6–8 weeks.

Patients and Methods

Patients

Between September 1976 and July 1978, 246 consecutive patients (age 65 years or younger) with acute MI were admitted to our coronary care unit. Our criteria for diagnosis of acute MI were a typical history of chest pain, the appearance of new Q waves or evolutionary ST-T changes and typical cardiac enzyme elevations. For admission to the series, all three criteria had to be fulfilled. Those referred from other hospitals often presented as complicated, and therefore specially selected, high-risk cases, and were excluded. Two patients with associated serious disease other than coronary artery disease were also excluded. Two hundred twenty-two patients survived the acute phase and were discharged. Of these survivors, 179 were eligible for this study.

After discharge, treatment was prescribed according to the following management directives: Asymptomatic patients were left untreated (60 patients). Those with angina were treated medically with propranolol together with long-acting nitroglycerin as necessary (89 patients). Coronary bypass surgery was offered to suitable patients with stable angina who failed to remain asymptomatic with medical treatment alone (18 patients). Surgery was also offered to those with unstable angina (six patients). Patients who had hypertension were treated with propranolol and diuretics (seven patients). Patients who had heart failure were treated with bedrest, digoxin, and diuretics (five patients). Ventricular dysrhythmias were treated with long-acting quinidine, or disopyramide as a second-line choice (six patients). No exercise rehabilitation was undertaken. Return to employment was not allowed until reassessment at 6–8 weeks.

After the patients gave informed consent, they were readmitted to the hospital for exercise testing and angiographic studies 6–8 weeks after the acute MI. A 12-lead ECG was recorded before and 24 hours after the investigations, together with serum CK.

Electrocardiography

Postinfarction electrocardiographic changes in leads I, aV_{L}, V_{1}–V_{6} were considered to represent anterior MI and those in leads II, III and aV_{F} were considered to represent inferior MI. The development of new Q waves 0.04 second long was considered diagnostic of acute transmural MI. The absence of new Q waves in the presence of characteristic serial ST-segment and T-wave changes was considered diagnostic of nontransmural MI. Patients were defined as having their first MI by the following criteria: no evidence of Q waves on initial ECG other than those related to the acute MI.
and no history of MI. Preexisting MI was based on a
documented history of infarction and the presence of Q
waves on the initial ECG.

Exercise Testing
Maximal, symptom-limited treadmill exercise test-
ing was performed according to the Bruce protocol.12
Medication was discontinued 24 hours before exercise
testing and resumed soon afterwards. The object was
to assess untreated symptoms rather than the efficacy
of treatment. Digitalis was stopped 7 days before in-
vestigation. A physician and a specially trained nurse
conducted the test. An ECG was monitored continu-
ously. Twelve-lead ECGs were recorded from adhe-
sive disc electrodes applied proximally to the trunk and
corresponding to limb lead positions.13 Ten-second
strips of the ECG were recorded on a six-channel
direct-writing recorder (mangrogh, paper speed 25
mm/sec, amplitude 1 mm = 0.1 mV) in both lying and
standing positions before exercise, at 1-minute inter-
vals during exercise, in the supine position immediate-
ly after exercise, and at 1-minute intervals after exer-
ercise. Exercise was continued until incapacitating
fatigue, dyspnea or progressive angina occurred. Exer-
cise was also terminated if ventricular tachycardia
(three or more consecutive ventricular premature com-
plexes) developed. Neither the attainment of an age-
predicted maximal heart rate nor the magnitude of ST-
segment depression was used as an end point.

Significant ST-segment depression was defined as
the development of a horizontal or downsloping ST-
segment depression $\geq$ 1 mm or $\geq$ 0.008 second in
any lead. A significant ST-segment elevation was de-
finied as the development of a ST-segment elevation $\geq$
1 mm. In patients with resting ST-segment abnormali-
ties, an additional ST-segment shift of $\geq$ 1 mm was
considered significant. Other exercise test variables
evaluated are listed in table 1. An inadequate blood
pressure response was taken as an increase of 10 mm
Hg or less in systolic blood pressure or a peak systolic
pressure of 140 mm Hg or less. The ventricular ar-
rhythmias were classified as simple (unifocal, $< 10$
beats/min) and complex ($> 10$ beats/min, multifocal,
paroxysmal, or ventricular tachycardia). Exercise tests
were reviewed independently by two cardiologists
without knowledge of clinical or angiographic details.
Disagreements in the two readings were resolved by a
third reviewer.

<table>
<thead>
<tr>
<th>Table 1. Variables Used to Predict Future Coronary Events</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exercise test</td>
</tr>
<tr>
<td>ST-segment depression</td>
</tr>
<tr>
<td>Exercise-induced angina pectoris</td>
</tr>
<tr>
<td>ST-segment elevation</td>
</tr>
<tr>
<td>Ventricular arrhythmias</td>
</tr>
<tr>
<td>Exercise tolerance</td>
</tr>
<tr>
<td>Blood pressure response</td>
</tr>
<tr>
<td>Maximal heart rate</td>
</tr>
<tr>
<td>Magnitude ST-segment shift</td>
</tr>
</tbody>
</table>

Abbreviations: LV = left ventricular; LAD = left anterior des-
cending coronary artery.

Angiography
Coronary angiography was performed in multiple
projections, including hemi-axial views, using the per-
cutaneous femoral artery technique. Left ventriculog-
raphy was done in both the right and left anterior
oblique projections (RAO and LAO). The left ventric-
ulograms and coronary angiograms were read independ-
ently by at least two cardiologists who had no know-
ledge of the clinical status of the patients. Disagreement
was resolved by a third opinion.

Each patient was considered to have three major
coronary arteries: left anterior descending (LAD),
including left main stem; left circumflex (LCx) and right
(RCA). Coronary artery narrowing were considered to
be present if the narrowing was more than 50% of the
luminal diameter in any projection. In the case of
multiple stenoses, only the most severe narrowing of a
coronary artery was recorded. Each patient was classi-
ified as having one-, two- or three-vessel coronary ar-
tery disease. For purposes of analysis, patients with
two- and three-vessel disease have been taken together
as multivessel disease. Significant narrowings in a
large diagonal branch, or obtuse marginal branch were
recorded as LAD and LCx disease, respectively.

Segmental left ventricular wall motion was analyzed ac-
cording to American Heart Association criteria.14
Using the RAO and LAO projections, seven ventricu-
lar segments were identified. These were characterized
as normal, hypokinetic, akinetic, dyskinetic or aneu-
rysmal. For interpretation, these seven segments were
classified into two main groups: the inferior wall
(formed by posterobasal, diaphragmatic and poste-
rolateral segments) and the anterior wall (formed by
anterolateral, apical and septal wall segments). The
most severe left ventricular wall motion abnormally
was recorded in each main group. The ventriculogram
was classified as showing normal, mild (hypokinetic),
and advanced left ventricular wall motion abnor-
malities (akinetic, dyskinetic or aneurysmal). Left ventric-
ular systolic and diastolic volumes were calculated
using the area-length method and the left ventricular
ejection fraction was calculated.15 The left ventricular
end-diastolic pressure was routinely measured after the
a wave or at the zenith of the R wave.

Follow-up
Patients were seen 3, 6 and 12 months after MI and
at 6-month intervals thereafter. The clinical assess-
ment and resting 12-lead ECG were obtained at each
visit. During the follow-up, all coronary events were
noted, i.e., death, recurrent MI, unstable angina pect-
oris, coronary artery bypass graft surgery and angina
pectoris. The follow-up period was terminated in case
of death or surgery.

Stable angina pectoris was defined as substernal dis-
comfort precipitated by exertion and relieved by rest or
nitroglycerin (within 3 minutes). Unstable angina pec-
toris was defined as such if either the severity, inten-
sity or frequency of the pain was rapidly progressive
despite treatment; or if the rest pain lasted for 15 min-
utes or longer and was not relieved by nitroglycerin,
and was accompanied by transient ST-T changes. The
diagnostic criteria for recurrent MI were the same as for entry into the study.

Cardiac death was proved whenever possible by autopsy. A thorough history was taken from the relatives and medical attendants to obtain an accurate description of the events and circumstances of death if it occurred outside the hospital.

Data Analysis

To retrospectively determine variables that were important in predicting forthcoming cardiac events (death, recurrence of MI and acquired angina pectoris), several subgroups were formed on the basis of exercise and catheterization variables. Discrete variables were noted as present or absent. Continuous variables were optimally dichotomized with respect to future events. The cumulative incidence in each subgroup was then calculated. This procedure was performed for time intervals of increasing length (0–6, 0–12, 0–18, 0–24, 0–30 and 0–36 months). The variables being tested are shown in table 1. High- and low-risk groups were defined with the help of the best-performing variables. Significant differences in performance and intergroup differences were calculated with the chi-square test.

Results

Patients

One hundred seventy-nine patients were eligible for the study (table 2). One hundred sixty-one were males, ages 28–64 years (mean 51 years) and 18 were females, ages 40–65 years (mean 54 years). Analysis of the available data of the 43 patients who were not evaluated were similar with respect to age, sex and site of infarction compared with the study group. Twenty-two patients (eight with anterior wall MI, 12 with inferior wall MI and two with subendocardial MI) refused further management and investigation. In five patients (two with anterior wall MI and three with inferior wall MI), technical problems prevented proper analysis of the angiograms. Two of these patients had angina pectoris and three were asymptomatic during follow-up. All patients who had bundle branch block were excluded. They did not perform an exercise test because of the known nondiagnostic ST-segment changes in these patients. Five patients had right bundle branch block and two left bundle branch block. In all of these patients, the bundle branch block was present at the time of admission. During follow-up, two patients had angina pectoris, one patient had a reinfarction and none died. Three patients died before repeat investigation at 6–8 weeks. One patient had an anterior MI and died from progressive heart failure. Autopsy revealed one-vessel disease. One patient had inferior MI and the other had a preexisting MI. Both died of fatal reinfarction and at autopsy both had two-vessel disease. Of the six patients lost to follow-up, four had inferior wall infarction and two had anterior wall infarction.

Surgery

Coronary artery bypass graft surgery was performed in 24 patients. All patients with unstable angina pectoris and those who did not respond adequately to medical therapy were operated on. One patient died perioperatively and three patients showed evidence of perioperative MI. Because the exercise test results and angiographic data may have contributed to the decision to perform surgery, no attempt was made to analyze variables predictive of future surgery.

Coronary Anatomy and Left Ventricular Function

In no case did we find entirely normal coronary arteries. In table 3, the coronary anatomy is presented in patients with anterior, inferior, nontransmural and preexisting MI.

The prevalence of multivessel disease was higher in patients with inferior MI and preexisting MI than in patients with anterior MI. The prevalence of multivessel disease was significantly higher among patients with angina pectoris at the time of investigation than in those without angina pectoris (table 4).

The presence of an ejection fraction less than 30%
TABLE 4. Correlation of Site of Myocardial Infarction and Presence of Angina Pectoris at Time of Investigation with Prevalence of Multivessel Disease

<table>
<thead>
<tr>
<th>Site of infarction</th>
<th>Total of pts</th>
<th>Prevalence of MVD No. of pts</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferior wall</td>
<td>81</td>
<td>51/81 (63%)</td>
<td>&lt; 0.025*</td>
</tr>
<tr>
<td>Angina pectoris</td>
<td>23/29 (79%)</td>
<td>2/29 (7%)</td>
<td>0.025†</td>
</tr>
<tr>
<td>No angina pectoris</td>
<td>28/52 (54%)</td>
<td>16/48 (33%)</td>
<td></td>
</tr>
<tr>
<td>Anterior wall</td>
<td>62</td>
<td>26/62 (42%)</td>
<td></td>
</tr>
<tr>
<td>Angina pectoris</td>
<td>10/14 (71%)</td>
<td>7/18 (39%)</td>
<td></td>
</tr>
<tr>
<td>No angina pectoris</td>
<td>16/48 (33%)</td>
<td>3/3 (100%)</td>
<td>&lt; 0.05†</td>
</tr>
<tr>
<td>Nontransmural</td>
<td>21</td>
<td>10/21 (48%)</td>
<td></td>
</tr>
<tr>
<td>Angina pectoris</td>
<td>6/9 (67%)</td>
<td>9/2 (5%)</td>
<td></td>
</tr>
<tr>
<td>PMI</td>
<td>15</td>
<td>11/15 (73%)</td>
<td>&lt; 0.05*</td>
</tr>
<tr>
<td>Angina pectoris</td>
<td>5/6 (83%)</td>
<td>1/17 (6%)</td>
<td>NS†</td>
</tr>
<tr>
<td>No angina pectoris</td>
<td>7/18 (39%)</td>
<td>6/9 (67%)</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Comparison with anterior wall infarction.
†Comparison with no angina pectoris.
Abbreviations: PMI = preexisting myocardial infarction; MVD = multivessel disease.

and a left ventricular end-diastolic pressure of more than 20 mm Hg occurred more frequently in patients with anterior MI and preexisting MI than in those with inferior MI and nontransmural MI (table 5).

Advanced left ventricular wall motion abnormalities were more frequent in patients with anterior infarction and preexisting MI than in those with inferior MI and nontransmural MI. The presence of advanced left ventricular wall motion abnormalities occurred more often in patients with multivessel disease (68 of 98, 69%) than in patients with one-vessel disease (34 of 81, 42%) (p < 0.001). Fifteen of 179 patients had normal ventricles at 6–8 weeks.

Coronary Events During Follow-up

The mean follow-up period was 28 months (range 13–40 months). Figure 1 is an overview of the coronary events and their interrelations after follow-up periods of 1, 2 and 3 years.

Eleven patients died of cardiac causes, six within 1 year and five in the second year of follow-up. Taking into account the three cardiac deaths within the period between discharge from hospital and admittance for investigation, the mortality in the first year was 5%, and total mortality in a mean follow-up period of 28 months was 7.3%. Two other patients died, one with a lung carcinoma and the other with a cerebrovascular accident.

Seven patients had a recurrent MI within the first year, four during the second year, and one patient during the third year. None of the patients with recurrent MI died or had surgery. Twenty-four patients had coronary vein bypass surgery.

In figures 2 and 3 and tables 6 and 7, the cumulative incidence of cardiac death, recurrence of MI, and angina pectoris acquired in follow-up for the different exercise and catheterization variables are presented. Using exercise and catheterization variables, high- and low-risk groups for mortality and recurrent MI were defined (table 8); the interrelations are shown in figure 4.

Complications

Three patients had exercise-induced ventricular tachycardia, which in all cases reverted spontaneously to sinus rhythm. Three complications resulted from cardiac catheterization. Two patients had ventricular fibrillation during angiography that successfully reverted to sinus rhythm with electroshock. One patient had transient aphasia and left arm weakness for 48 hours after the investigation.

TABLE 5. Left Ventricular Function 6–8 Weeks After Myocardial Infarction

<table>
<thead>
<tr>
<th>Site of infarction</th>
<th>No. of pts</th>
<th>EF &lt; 30%</th>
<th>LVEDP &gt; 20 mm Hg</th>
<th>Normal or mild</th>
<th>Advanced</th>
<th>Inferior wall</th>
<th>Anterior wall</th>
<th>Normal or mild</th>
<th>Advanced</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferior</td>
<td>81</td>
<td>2 (2%)</td>
<td>2 (2%)</td>
<td>37 (46%)</td>
<td>—</td>
<td>35/37 (95%)</td>
<td>2/37 (5%)</td>
<td>37/44 (84%)</td>
<td>7/44 (16%)</td>
</tr>
<tr>
<td>Anterior</td>
<td>62</td>
<td>13 (21%)</td>
<td>12 (19%)*</td>
<td>4/4 (100%)</td>
<td>0/4</td>
<td>4 (6%)</td>
<td>—</td>
<td>58 (94%)†</td>
<td>58 (94%)†</td>
</tr>
<tr>
<td>Nontransmural</td>
<td>21</td>
<td>2 (10%)</td>
<td>1 (5%) NS</td>
<td>17 (81%)</td>
<td>—</td>
<td>16/17 (94%)</td>
<td>1/17 (6%)</td>
<td>3/4 (75%)</td>
<td>1/4 (25%)</td>
</tr>
<tr>
<td>PMI</td>
<td>15</td>
<td>8 (53%)</td>
<td>6 (40%)*</td>
<td>4 (27%)</td>
<td>—</td>
<td>0</td>
<td>4/4 (100%)</td>
<td>5/11 (45%)</td>
<td>6/11 (55%)</td>
</tr>
</tbody>
</table>

Some sacrifice has been made for the sake of clarity in the analysis of left ventricular function data; "Normal" left ventricles have been taken together with those that show a mild abnormality.

*p < 0.001 vs inferior.
†p < 0.005 vs inferior and nontransmural.
‡p < 0.001 vs inferior and transmural.
Abbreviations: EF = ejection fraction; LVEDP = left ventricular end-diastolic pressure; PMI = preexisting myocardial infarction; mild = normal and hypokinesia; advanced = akinesia, dyskinesia or aneurysm.

The inferior wall motion abnormalities are subdivided into mild and advanced inferior, with coexisting anterior abnormalities except in the group with anterior infarction, in which this is reversed.
Discussion

Our study was both prospective — to evaluate coronary anatomy, left ventricular function and exercise tolerance 6–8 weeks after infarction; and retrospective — to assess the predictive accuracy of the data.

The safety and feasibility of exercise testing and cardiac catheterization soon after MI is established. Angiography was performed without deaths, although two patients had ventricular fibrillation. Exercise testing proceeded to symptom limitation regardless of ST changes, although three cases of spontaneous reverting ventricular tachycardia did occur. This method proved safe; the incidence of ventricular tachycardia was similar to that in our laboratory using symptom-limited exercise for assessing angina without previous MI (four of 554).

Coronary Anatomy and Left Ventricular Function

The prevalence of multivessel disease in our study was 55%. In three studies with similar indications for angiography, the prevalence of multivessel disease was 60–75%. These studies all included a higher prevalence of preexisting MI (22–27%) than did our study (8%), and thus could be expected to have a higher frequency of multivessel disease. Our cases with preexisting MI had a 73% prevalence of multivessel disease. The prevalence of multivessel disease after inferior MI (63%) was significantly higher than after anterior MI ($p < 0.001$). This result differs from the studies of Taylor et al. and Turner et al., who found no difference in the extent of coronary artery disease between survivors of anterior and inferior MI. Unlike survivors of transmural MI, survivors of nontransmural MI had an almost even distribution of one- and multivessel disease.

Only two patients had left main disease. They were included in the LAD group because there were too few to form a separate group. The follow-up of both patients was uneventful. The prevalence of coexisting LAD disease in patients with inferior MI was 52%. Others have also established a high frequency of coexisting LAD disease (63–82%) in symptomatic survivors of an inferior MI.

The prevalence of total coronary artery occlusion within 4 hours of infarction symptoms was found to be 87% by DeWood et al. and was 65% at 12–24 hours. At 6–8 weeks, we have found a prevalence of 71% and 50% for the RCA and LCx in transmural inferior MI and 67% for the LAD in transmural anterior MI. These data suggest that the prevalence of total occlusion rapidly decreases with time and thereafter remains fixed.

In patients with inferior MI, the left ventricular damage was moderate and almost always limited to the inferior wall. After anterior MI, left ventricular impairment is often severe and is not restricted to the anterior wall, but extends also to the inferior wall in one-third of the patients. In symptomatic patients other investigators also found a more severe left ventricular dysfunction in patients with anterior MI than in those with inferior MI.

Table:

<table>
<thead>
<tr>
<th>Time Period</th>
<th>Patients</th>
<th>Cardiac Death</th>
<th>Surgery</th>
<th>Recurrence MI</th>
<th>Angina Pectoris</th>
<th>None</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-12 months</td>
<td>179</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>43</td>
<td>69</td>
</tr>
<tr>
<td>13-24 months</td>
<td>97</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>15</td>
<td>0</td>
</tr>
<tr>
<td>25-36 months</td>
<td>35</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Figure 1. The number of coronary events at different follow-up periods. Only one event (the most serious in the following order: death, recurrent myocardial infarction [MI], surgery and angina pectoris) was allowed per patient during follow-up. The time of follow-up terminated at the time of death or surgery. The numbers of patients are those who were followed throughout the indicated period.
In survivors of a nontransmural MI, the left ventricular impairment was often moderate. Survivors of a preexisting MI often showed severe impairment of both the inferior and anterior left ventricular wall.

**Angina Pectoris**

More patients with angina pectoris after MI have multivessel disease than asymptomatic patients, regardless of infarction site. Patients with angina pectoris at the time of investigation had a 79% prevalence of multivessel disease, compared with 45% in asymptomatic patients (p < 0.001). Our results are com-

rable to those of the Framingham study, which indicated that about half of the patients surviving an infarction have angina. We found a 42% incidence of angina after 1 year. In the recent retrospective study by Amsterdam et al. of hospitalized patients, a 48% prevalence of angina after MI was found. The prospective studies of Waters et al. and Theroux et al. both report similar incidences of 61% and 42%, respectively.

**Prediction of Coronary Events**

This study retrospectively assesses the efficacy of several investigational variables to predict cardiac
Table 6A. Variables Derived from Coronary and Left Ventricular Angiography — Number of Cardiac Deaths/Number of Patients in the Subgroup Defined by the Variable

<table>
<thead>
<tr>
<th>Time interval (months)</th>
<th>0-6</th>
<th>0-12</th>
<th>0-18</th>
<th>0-24</th>
<th>0-30</th>
<th>0-36</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total group incidence</td>
<td>4/179</td>
<td>6/179</td>
<td>8/152</td>
<td>10/132</td>
<td>11/104</td>
<td>11/82</td>
</tr>
<tr>
<td>CAD subgroup incidence</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-vessel disease</td>
<td>1/26</td>
<td>1/26</td>
<td>2/21</td>
<td>4/20</td>
<td>5/16†</td>
<td>5/14*</td>
</tr>
<tr>
<td>Multivessel disease</td>
<td>2/98</td>
<td>4/98</td>
<td>5/96</td>
<td>7/77</td>
<td>8/63</td>
<td>8/51</td>
</tr>
<tr>
<td>LV function subgroup incidence</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ejection fraction &lt; 30%</td>
<td>3/25†</td>
<td>4/25†</td>
<td>5/22‡</td>
<td>5/19†</td>
<td>5/15†</td>
<td>5/14*</td>
</tr>
<tr>
<td>LV end-diastolic pressure &gt; 20 mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Advanced LV wall motion abnormalities</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data correspond to figure 2, cardiac death.

*p < 0.05.
†p < 0.01.
‡p < 0.001.

Abbreviations: CAD = coronary artery disease; LAD = left anterior descending coronary artery; LV = left ventricular.

The number of cardiac events in each subgroup increases with time, and because of the varying length of the follow-up, the number of patients in each subgroup decreases with time. Thus, the standard error of the percent also increases with time.

In our study, unlike others,5-11 exercise test variables alone or in combination were not a good predictor of subsequent mortality. Although an exercise tolerance of less than 10 minutes identified most of the patients who had a later coronary event, the high frequency of this response made this variable inefficient.

We confirm the study of Taylor et al.3 in finding a

Table 6B. Variables Derived from Coronary and Left Ventricular Angiography — Number of Recurrent Infarctions/Number of Patients in the Subgroup Defined by the Variable

<table>
<thead>
<tr>
<th>Time interval (months)</th>
<th>0-6</th>
<th>0-12</th>
<th>0-18</th>
<th>0-24</th>
<th>0-30</th>
<th>0-36</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total group incidence</td>
<td>6/179</td>
<td>7/179</td>
<td>10/152</td>
<td>12/132</td>
<td>12/104</td>
<td>12/82</td>
</tr>
<tr>
<td>CAD subgroup incidence</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-vessel disease</td>
<td>1/26</td>
<td>2/26</td>
<td>3/21</td>
<td>4/20</td>
<td>3/16</td>
<td>3/14</td>
</tr>
<tr>
<td>LV function subgroup incidence</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ejection fraction &lt; 30%</td>
<td>1/25</td>
<td>1/25</td>
<td>1/22</td>
<td>2/19</td>
<td>2/15</td>
<td>2/14</td>
</tr>
<tr>
<td>LV end-diastolic pressure &gt; 20 mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Advanced LV wall motion abnormalities</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data correspond to figure 2, recurrent myocardial infarction.

Abbreviations: CAD = coronary artery disease; LAD = left anterior descending coronary artery; LV = left ventricular.
TABLE 7A. Variables Derived from Exercise Testing — Number of Cardiac Deaths/Number of Patients in the Group Defined by the Variable

<table>
<thead>
<tr>
<th>Time interval (months)</th>
<th>0-6</th>
<th>0-12</th>
<th>0-18</th>
<th>0-24</th>
<th>0-30</th>
<th>0-36</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total group incidence</td>
<td>4/179</td>
<td>6/179</td>
<td>8/152</td>
<td>10/132</td>
<td>11/104</td>
<td>11/82</td>
</tr>
<tr>
<td>ST depression or exercise-induced angina pectoris</td>
<td>3/91</td>
<td>4/91</td>
<td>5/76</td>
<td>7/71</td>
<td>7/56</td>
<td>7/49</td>
</tr>
<tr>
<td>Exercise-induced ventricular arrhythmias</td>
<td>1/27</td>
<td>2/27</td>
<td>2/21</td>
<td>2/19</td>
<td>2/15</td>
<td>2/14</td>
</tr>
<tr>
<td>Exercise tolerance less than 10 min (Bruce protocol)</td>
<td>4/121</td>
<td>6/121</td>
<td>8/103</td>
<td>10/88*</td>
<td>11/72*</td>
<td>11/60</td>
</tr>
</tbody>
</table>

Data correspond to figure 3, cardiac death.
*\( p < 0.05 \).

TABLE 7B. Variables Derived from Exercise Testing — Number of Recurrent Myocardial Infarctions/Number of Patients in the Subgroup Defined by the Variable

<table>
<thead>
<tr>
<th>Time interval (months)</th>
<th>0-6</th>
<th>0-12</th>
<th>0-18</th>
<th>0-24</th>
<th>0-30</th>
<th>0-36</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total group incidence</td>
<td>6/179</td>
<td>7/179</td>
<td>10/152</td>
<td>12/132</td>
<td>12/104</td>
<td>12/82</td>
</tr>
<tr>
<td>ST depression or exercise-induced angina pectoris</td>
<td>2/91</td>
<td>4/91</td>
<td>6/76</td>
<td>7/71</td>
<td>7/56</td>
<td>7/49</td>
</tr>
<tr>
<td>Exercise tolerance less than 10 min (Bruce protocol)</td>
<td>4/121</td>
<td>6/121</td>
<td>8/103</td>
<td>10/88*</td>
<td>11/72*</td>
<td>11/60</td>
</tr>
</tbody>
</table>

Data correspond to figure 3, recurrent myocardial infarction.

low ejection fraction (< 30%) and the presence of three-vessel disease as the best catheterization variables for prediction of cardiac death. An ejection fraction < 30% predicted five deaths and three-vessel disease five other deaths. Left ventricular end-diastolic pressure alone or in combination with these variables did not increase the predictive accuracy. Abnormalities of wall motion after infarction are not as useful as global abnormalities (ejection fraction and left ventricular end-diastolic pressure) in predicting prognosis.

An exercise tolerance of less than 10 minutes and the presence of three-vessel disease had some value for predicting recurrent MI, although this was not statistically significant. A combination of exercise variables

![Figure 4](https://circ.ahajournals.org/)

**Figure 4.** Graphic representation of the high-risk groups. Broken line indicates high-risk group catheterization variables (46 patients); continuous line indicates high-risk group exercise test variables (121 patients). The areas are proportional to the number of patients in a group. D = one patient with cardiac death; M = one patient with recurrent infarction.
and catheterization variables did not result in a better prediction for cardiac death or recurrent MI.

Angiographic variables were better predictors for cardiac death and recurrent MI than the exercise test variables. While echocardiography and radionuclide scanning might also be used to detect low ejection fraction, angiography is still required to detect three-vessel disease.

In a few similar recent studies, the mortality at 1 year has been reported as 2–12%. In our study the first-year mortality rate (including the three patients who died in the period after discharge, and before readmittance for further investigation) was nine of 179 (5%). The Framingham study, the average annual rate of recurrence of infarction over the first 5 years was 2.9% for men and 9.6% for women. Others report a recurrence rate of 6% after the first year. We found an incidence of 4% during the first year and 7% during the mean follow-up of 28 months.

Statistically, a low prevalence of mortality and infarct recurrence in survivors of MI makes only a highly specific test able to perform well as a predictor. In the absence of such a test, it is practical only to identify high- and low-risk groups. Identification of a high-risk group was possible with both exercise and angiographic variables. The high-risk group based on exercise test variables detected all 11 cardiac deaths and 11 of 12 recurrent MIs. The high-risk group based on catheterization variables detected 10 of the 11 cardiac deaths and only five of 12 recurrent MIs (table 8). The difference in efficiency between these two high-risk groups relates to the number of patients in each group: 121 for the exercise variables and 46 for the catheterization variables. The low efficiency of the exercise tolerance group makes it adequate to define a low-risk group only. The high efficiency of the catheterization variables makes high-risk detection more reliable. Thus, an exercise tolerance criterion of 10 minutes or more results in the identification of a very low risk group for mortality and morbidity, while an ejection fraction of less than 30%, or presence of three-vessel disease identifies a high-risk group for subsequent mortality. The interrelations between both high-risk groups are illustrated in figure 4.

The conclusions of this study only apply to a selected population, because the design excluded patients who died before hospitalization or during in-hospital convalescence from MI. However, the study does show that a high-risk group can be identified in a population with a low incidence of prior MI, multivessel disease, poor left ventricular function and low post-infarction mortality.

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