Symptom-limited vs Heart-rate-limited Exercise Testing Soon After Myocardial Infarction

Robert F. DeBusk, M.D., and William Haskell, Ph.D.

SUMMARY To develop guidelines for exercise testing soon after uncomplicated myocardial infarction, 93 men completed a heart-rate-limited (HRL) protocol and 107 completed a symptom-limited (SXL) protocol 3 weeks after the acute event. In the HRL protocol, effort terminated at a heart rate of 130 beats/min in the absence of a limiting symptom, exertional hypotension or ventricular tachycardia. Peak heart rate was not an end point in the SXL protocol. Despite a higher peak heart rate and workload in patients who completed the SXL protocol, the prevalence of exercise-induced ischemic ST-segment depression and ventricular ectopic activity was similar in the two groups. No complications occurred with either protocol. Twelve patients (6%) had cardiac events within the next 2 months. Regardless of the test protocol used, early events were more common in patients with ischemic ST-segment responses (15%) than in patients without ischemic responses (3%) ($p < 0.01$). In contrast, exercise-induced ventricular arrhythmias were not predictive of early events. Eleven weeks after infarction, when all tests were SXL, the prevalence of exercise-induced ischemic ST-segment depression and premature ventricular complexes was similar to that at 3 weeks. We conclude that SXL and HRL exercise test protocols reveal a similar prevalence of ischemic ST-segment depression and ventricular ectopic activity soon after uncomplicated myocardial infarction.

TREADMILL EXERCISE testing performed soon after myocardial infarction aids in assessing prognosis and provides guidelines for physical activity. Few guidelines concerning the appropriate timing and intensity of such testing are available to the clinician. Moreover, there are few data concerning the reproducibility of exercise-induced ischemic and arrhythmic abnormalities observed soon after infarction. In particular, few comparisons are available between exercise tests performed soon after infarction (e.g., 3 weeks) and those performed at a later phase of convalescence (e.g., 11 weeks).

The present study had two major objectives: 1) to compare heart-rate-limited and symptom-limited test protocols with respect to safety, magnitude of cardiovascular response, prevalence of ischemic and arrhythmic abnormalities and prognostic value in patients with uncomplicated infarction, and 2) to compare test responses 3 weeks after infarction with those at 11 weeks in order to determine the reproducibility of ischemic ST-segment depression and premature ventricular complexes (PVCs) disclosed by the two protocols.

Methods

Two hundred males, mean age 54 ± 6 years, who were hospitalized at the Stanford Medical Center for treatment of acute myocardial infarction were selected for this study. Myocardial infarction was documented by a history of prolonged chest pain and the appearance of new Q waves or evolutionary ST changes and characteristic elevation of cardiac enzymes. Patients usually began ambulation 5–7 days after infarction. The average duration of hospitalization was 10 ± 2 days. No formal exercise conditioning program was provided between the time of hospital discharge and treadmill exercise testing 3 weeks after infarction.

Patients who had one or more of the following characteristics 3 weeks after hospital admission were not included: 1) rest angina or unstable angina; 2) clinical congestive heart failure or the presence of a ventricular diastolic gallop; 3) associated medical conditions such as significant valvular heart disease, hypertension > 180/100 mm Hg, limiting pulmonary disease or musculoskeletal abnormalities.

Three weeks after infarction, 7% of patients took propranolol and 6% of patients took quinidine or procainamide. Eleven weeks after infarction, 10% of patients took propranolol and 6% of patients took quinidine or procainamide. These medications were discontinued 16–24 hours before exercise testing and were resumed after testing. At 3 and 11 weeks, 3% of patients took digitalis though none had clinical heart failure.

All tests were performed on outpatients between 1:00 p.m. and 5:00 p.m. at least 2 hours after eating or smoking. Before testing, patients were interviewed and examined by a physician and informed consent was obtained. A physician and a specially trained nurse conducted the tests. Twelve-lead ECGs were recorded at rest, at the end of each 3-minute stage of exercise and at 1, 3, 5, 7 and 10 minutes of recovery. In addition, leads V4 to V6 were continuously displayed on a three-channel oscilloscopic monitor and were recorded on magnetic tape for 3 minutes before exercise and during the entire period of exercise and recovery. Exercise was performed on a motor-driven treadmill using a combination of protocols described by Naughton et al. (table 1).

In the heart-rate-limited exercise test protocol, effort commenced at a work load equivalent to 3 multiples of resting energy expenditure (mets). Work loads were added every 3 minutes until the appearance
of any of the following end points: 1) limiting symptoms of chest pain, dyspnea, fatigue, leg cramps or dizziness; 2) hypotension, i.e., a fall in systolic blood pressure of 10 mm Hg or more from the peak value attained earlier during exercise; 3) ventricular tachycardia, i.e., ≥ 3 PVCs; or 4) a heart rate of 130 beats/min in the absence of other end points.

In the symptom-limited exercise test protocol, effort was discontinued only in the event of limiting symptoms, exertional hypotension or ventricular tachycardia, without respect to peak heart rate.

In neither test protocol was the magnitude of ischemic ST-segment depression used as an end point for testing. Ischemic ST-segment depression was said to be present when flat or downsloping ST segments were depressed 0.1 mV or more below the PQ line 0.08 second after the J point. If resting ST-segment depression was noted, at least 0.1 mV of additional ST-segment depression was required for the diagnosis.

Responses to the two exercise protocols 3 weeks after infarction are shown in figure 1. Patients were grouped on the basis of the test protocol completed 3 weeks after infarction: heart-rate-limited (group 1) or symptom-limited (group 2). Patients in group 1 could manifest a heart-rate-limited or symptom-limited response, but group 2 patients manifested only symptom-limited responses. All patients undergoing exercise testing at 11 weeks also completed a symptom-limited protocol.

Between 3 and 11 weeks 15 patients were dropped from the study for medical reasons: 12 due to cardiac events and three due to development of an S₃ gallop sound. An additional eight patients were dropped for nonmedical reasons: two developed orthopedic problems, four moved from the area and two declined to participate further.

Statistical analyses were performed using programs from the Statistical Packages for the Social Sciences. Discrete variables were compared using contingency tables. The reproducibility of exercise-induced PVCs and ischemic ST-segment depression was expressed by kappa coefficient (κ). Changes in the prevalence of exercise-induced ventricular ectopic activity and ischemic ST-segment depression were analyzed with the McNemar test. Comparisons between and within groups for continuous response variables were analyzed by independent group or matched-pairs  \( t \) tests. Two-tailed tests of statistical significance were used and \( p \) values < 0.05 were considered significant.

### Results

#### Clinical Characteristics

Group 1 and 2 patients did not differ significantly in the clinical characteristics shown in table 2.

#### Test End Points (fig. 1)

Of the 93 patients in group 1, 42 (45%) stopped solely because of the heart rate limit of 130 beats/min. Of the remaining 51 patients, 33 (35%) stopped because of fatigue or dyspnea, eight (9%) because of exercise-induced hypotension and 10 (11%) because of angina pectoris. Of the 107 patients in group 2 who completed a symptom-limited test 3 weeks after infarction, 48 stopped at a heart rate of 130 beats/min or less and 59 at a heart rate exceeding 130 beats/min. The distribution of end points in these two subgroups was not significantly different: 70% stopped because of

### Table 2. Clinical Characteristics

<table>
<thead>
<tr>
<th>Group 1 (n = 93)</th>
<th>Group 2 (n = 107)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>52 ± 9</td>
</tr>
<tr>
<td>Norris score</td>
<td>2.7 ± 1.9</td>
</tr>
<tr>
<td>Max SGOT</td>
<td>116 ± 74</td>
</tr>
<tr>
<td>Max CPK</td>
<td>840 ± 714</td>
</tr>
<tr>
<td>Hospital complications</td>
<td>51%</td>
</tr>
<tr>
<td>Site of MI</td>
<td></td>
</tr>
<tr>
<td>Inferior</td>
<td>66%</td>
</tr>
<tr>
<td>Anterior</td>
<td>25%</td>
</tr>
<tr>
<td>Nontransmural</td>
<td>10%</td>
</tr>
</tbody>
</table>

Abbreviation: MI = myocardial infarction.

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**Table 1. Treadmill Test Protocol**

<table>
<thead>
<tr>
<th>Speed (mph)</th>
<th>Incline (%)</th>
<th>Work load (mets)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.0</td>
<td>3.5</td>
<td>3</td>
</tr>
<tr>
<td>2.0</td>
<td>7.0</td>
<td>4</td>
</tr>
<tr>
<td>2.0</td>
<td>10.5</td>
<td>5</td>
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<tr>
<td>2.0</td>
<td>14.0</td>
<td>6</td>
</tr>
<tr>
<td>2.0</td>
<td>17.5</td>
<td>7</td>
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<tr>
<td>3.0</td>
<td>12.5</td>
<td>8</td>
</tr>
<tr>
<td>3.0</td>
<td>15.0</td>
<td>9</td>
</tr>
<tr>
<td>3.0</td>
<td>17.5</td>
<td>10</td>
</tr>
<tr>
<td>3.0</td>
<td>20.0</td>
<td>11</td>
</tr>
<tr>
<td>3.0</td>
<td>22.5</td>
<td>12</td>
</tr>
</tbody>
</table>

Abbreviations: mph = miles per hour; mets = multiples of resting energy expenditure.

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**Figure 1. Treadmill tests 3 weeks after myocardial infarction. HR = heart rate (beats/min); GPA = group 1; GPB = group 2.**
fatigue or dyspnea, 19% because of exercise-induced hypotension and 11% because of angina pectoris. At 11 weeks, when all patients completed a symptom-limited protocol, end points in group 1 and group 2 patients were similar: dyspnea and fatigue in 68% and 72%, hypotension in 15% and 21% and angina pectoris in 10% and 18% of tests, respectively. The distribution of end points at 11 weeks was similar for group 1 patients who were originally limited to a heart rate of 130 beats/min and for those originally limited by symptoms at a heart rate less than 130 beats/min.

Cardiovascular Measurements at Peak Effort

Cardiovascular measurements at peak effort 3 weeks after infarction were significantly higher in group 2 than in group 1 patients (table 3). By 11 weeks there was no longer a significant intergroup difference in peak double product. Between 3 and 11 weeks, both groups had a significant ($p < 0.001$) increase in cardiovascular variables at peak effort.

Cardiovascular Measurements at Onset of Ischemic ST-segment Depression

Cardiovascular measurements at the onset of ischemic ST-segment depression (table 4) were not significantly different between groups 1 and 2 3 weeks after infarction. At 11 weeks, heart rate at the onset of ischemic ST-segment depression was significantly ($p < 0.05$) higher in group 2 than in group 1 patients. Work load at the onset of ischemic ST-segment depression increased significantly ($p < 0.001$) in both groups between 3 and 11 weeks. Although heart rate and double product at the onset of ischemic ST-segment depression increased between 3 and 11 weeks, this increase was not statistically significant in either group. Ischemic ST-segment depression occurred at a work load of 75–80% of the peak work load at both 3 and 11 weeks. Similarly, ischemic ST-segment depression occurred at a double product of 80–89% of the peak double product at 3 and 11 weeks. The magnitude of ischemic ST-segment depression did not change significantly between 3 and 11 weeks.

### Table 3. Cardiovascular Measurements at Peak Effort

<table>
<thead>
<tr>
<th></th>
<th>3 weeks</th>
<th>11 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group 1 (n = 93)</td>
<td>Group 2 (n = 107)</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>123 ± 12</td>
<td>134 ± 21*</td>
</tr>
<tr>
<td>HR (% of age-predicted)</td>
<td>73 ± 7</td>
<td>81 ± 12*</td>
</tr>
<tr>
<td>Double product</td>
<td>197 ± 42</td>
<td>220 ± 53*</td>
</tr>
<tr>
<td>Work load (mets)</td>
<td>4.8 ± 1.5</td>
<td>6.2 ± 1.7*</td>
</tr>
</tbody>
</table>

* $p < 0.001$ vs group 1.
† $p < 0.01$ vs group 1.

Abbreviations: HR = heart rate; mets = multiple of resting energy expenditure.

### Table 4. Cardiovascular Measurements at Onset of Ischemic ST-segment Depression

<table>
<thead>
<tr>
<th></th>
<th>3 weeks</th>
<th>11 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group 1 (n = 17)</td>
<td>Group 2 (n = 22)</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>114 ± 10</td>
<td>121 ± 17</td>
</tr>
<tr>
<td>Double product</td>
<td>176 ± 31</td>
<td>180 ± 52</td>
</tr>
<tr>
<td>Work load (mets)</td>
<td>3.8 ± 1.1</td>
<td>4.6 ± 2.0</td>
</tr>
<tr>
<td>Max STI (mV)</td>
<td>0.23 ± 0.10</td>
<td>0.19 ± 0.12</td>
</tr>
</tbody>
</table>

* $p < 0.05$ vs group 1.

Abbreviations: mets = multiples of resting energy expenditure; max STI = maximal ST-segment depression.

Premature Ventricular Complexes

The prevalence of any exercise-induced PVC, defined as the proportion of patients with any PVC, was not significantly different between group 1 and 2 patients 3 or 11 weeks after infarction (fig. 2). The prevalence of any exercise-induced PVC increased significantly between 3 and 11 weeks in both groups of patients.

Antiarrhythmic therapy appeared to have little bearing on the results of this study: not only was antiarrhythmic usage relatively infrequent, but patients who had received antiarrhythmic therapy in the 16–24 hours before testing had a prevalence and frequency of PVCs similar to that of patients not receiving antiarrhythmic therapy.

The peak frequency of exercise-induced PVCs did not correlate significantly with peak heart rate at 3 or 11 weeks. Exercise-induced PVCs were no more common in patients with exercise-induced ischemic ST-segment depression than in patients without ischemia.

The reproducibility of PVCs was defined as the tendency of a test result, i.e., presence or absence of
PVCs on the 3-week test, to be found on the 11-week test. Group 2 patients had a reproducibility of PVCs significantly above the chance level (κ 0.376, p < 0.001). Seventy-nine percent of group 2 patients with PVCs at 3 weeks also had PVCs at 11 weeks, and 64% of group 2 patients without PVCs at 3 weeks had no PVCs at 11 weeks. In contrast, the reproducibility of exercise-induced PVCs in group 1 patients was at the chance level (κ 0.143, NS). Sixty-four percent of group 1 patients with PVCs at 3 weeks also had PVCs at 11 weeks, and 52% of group 1 patients without PVCs at 3 weeks had no PVCs at 11 weeks.

Three group 1 patients and one group 2 patient had exercise-induced coupled PVCs without ventricular tachycardia at 3 weeks. At 11 weeks, coupled PVCs were noted in five group 1 and in six group 2 patients. The only episode of ventricular tachycardia, a three-beat run, occurred at 11 weeks in a group 1 patient. There were no complications of exercise testing with either protocol.

Ischemic ST-segment Depression

Flat or downsloping ST-segment depression at rest was found in the following proportion of patients 3 weeks after infarction: none, 58%; 0.0–0.05 mV, 30%; 0.06–0.09, 9%; and ≥ 0.1 mV, 3%, without significant differences between the two patient groups. A similar proportion of tests showed resting ST-segment depression 11 weeks after infarction. The prevalence of exercise-induced ST-segment depression ≥ 0.1 mV, defined as the proportion of patients with this abnormality, was not significantly different between the two groups at 3 or 11 weeks (fig. 3). Between 3 and 11 weeks, the prevalence of exercise-induced ischemic ST-segment depression increased significantly in group 1 (p < 0.01) but not in group 2 patients. Neither the prevalence nor the magnitude of ischemic ST-segment depression were significantly correlated with peak heart rate or work load in either group of patients 3 or 11 weeks after infarction.

Exercise-induced ischemic ST-segment depression was reproducible (p < 0.001) in both patient groups: 94% of group 1 and 76% of group 2 patients with exercise-induced ischemic ST-segment depression at 3 weeks also had ischemia at 11 weeks and 79% of group 1 and 85% of group 2 patients without ischemic ST-segment depression at 3 weeks were still free of ischemia at 11 weeks. There was no significant difference in the reproducibility of exercise-induced ischemic ST-segment depression in the two groups (κ 0.347 for group 1 and κ 0.542 for group 2).

Exercise-induced Angina Pectoris

The prevalence of exercise-induced angina pectoris in group 1 patients was 12% and 21% 3 and 11 weeks after infarction. The prevalence of exercise-induced angina pectoris was similar in group 2 patients: 22% and 18%, respectively. Anginal responses were highly reproducible in both groups (group 1: κ 0.622, p < 0.001; group 2: κ 0.413, p < 0.001). Seventy percent of group 1 and 60% of group 2 patients with exercise-induced angina 3 weeks after infarction also had angina 11 weeks after infarction and 86% of group 1 and 93% of group 2 patients free of angina 3 weeks after infarction were still free of angina 11 weeks after infarction. There was no significant difference in the reproducibility of exercise-induced angina pectoris in the two groups.

Exercise-induced Angina Pectoris vs Exercise-induced Ischemia

Exercise-induced ischemic ST-segment depression was usually painless: 74% of group 1 and 63% of group 2 patients with ischemic ST-segment depression had no angina pectoris 3 weeks after infarction. A similar relationship between ischemic ST-segment depression and angina pectoris was noted in both groups 11 weeks after infarction. Ischemic ST-segment depression was seen in half of group 1 and group 2 patients with exercise-induced angina pectoris 3 and 11 weeks after infarction.
Cardiac Events 3–11 Weeks After Infarction

One group 1 patient and two group 2 patients developed S₃ gallop sounds 3–11 weeks after infarction, but did not develop clinical heart failure during this interval. These patients were classified as medical dropouts. Four of 93 group 1 patients (4%) and eight of 107 group 2 patients (7%) had cardiac events 3–11 weeks after infarction. One group 1 patient died suddenly, one had ventricular fibrillation with resuscitation, one died from myocardial infarction and one had coronary artery bypass graft surgery for unstable angina pectoris. Four group 2 patients had unstable angina pectoris, one of whom underwent coronary artery bypass graft surgery. The one group 1 patient and the three group 2 patients who underwent coronary artery bypass graft surgery had triple-vessel disease on coronary arteriography.

Cardiac events 3–11 weeks after infarction were more frequent among patients with ischemic ST-segment depression ≥ 0.1 mV 3 weeks after infarction (15%) than in patients without ischemic responses 3 weeks after infarction (3%) (p < 0.01). Seven patients with early events had ischemic ST-segment responses 3 weeks after infarction. Ischemia appeared at a heart rate of 130 beats/min or less in six of these seven patients. All three group 1 patients who experienced sudden death, ventricular fibrillation with resuscitation or fatal myocardial infarction between 3 and 11 weeks had ischemic ST-segment depression ≥ 0.2 mV 3 weeks after infarction.

Early cardiac events were no more frequent among patients with any exercise-induced PVC 3 weeks after infarction (10%) than among patients without exercise-induced PVCs (6%). This was true even when complex PVCs, i.e., bigeminy, couplets, ventricular tachycardia or frequency > 3 PVCs/min were considered separately.

Discussion

The major conclusion of the present study is that symptom-limited and heart-rate-limited treadmill exercise test protocols are equally safe and effective in eliciting ischemic ST-segment depression and ventricular ectopic activity soon after uncomplicated myocardial infarction. Moreover, the prevalence of ischemic ST-segment depression and ventricular ectopic activity were not related to the peak heart rate attained 3 or 11 weeks after infarction. Therefore, the distinction between heart-rate-limited testing and symptom-limited testing soon after infarction appears to be largely arbitrary if the heart rate limit is sufficiently high. In fact, the diagnostic yield of exercise-induced ischemic and arrhythmic abnormalities may be more closely related to the extent of coronary heart disease than to the protocol used. Because our patients were free of clinically significant left ventricular dysfunction, caution is required in generalizing these results to populations in which more extensive left ventricular dysfunction is present.

Safety considerations are paramount in the performance of exercise testing soon after myocardial infarction. Because we felt that a heart-rate-limited test might prove safer than a symptom-limited one 3 weeks after infarction, we initially used a peak heart rate of 130 beats/min as an end point, based on the study of Ericsson et al.7 Few of our first 93 patients who completed a heart-rate-limited protocol actually had complex ventricular arrhythmias such as coupled PVCs. In fact, among the first 93 patients, coupled PVCs were more often observed in those who failed to attain the heart rate limit of 130 beats/min than in those who did so.

Of the next 107 patients who completed a symptom-limited protocol 3 weeks after infarction, none had couplets or ventricular tachycardia. Peak heart rates in both groups of our patients were similar to the 80–84% age-predicted peak heart rates noted by Ibsen et al.,4 who performed symptom-limited exercise testing 3 weeks after infarction. We therefore concluded that the use of a protocol in which tests were terminated solely because of attainment of the arbitrary peak heart rate of 130 beats/min afforded a false sense of security regarding test safety. In fact, there was no safe heart rate at which complex PVCs were absent, and the heart rate limit was therefore discarded.

The appearance of 0.1 mV of ischemic ST-segment depression is often used as an end point for exercise testing. While this is done partly in the interest of safety, there are few data to substantiate the greater safety of such a policy compared with that of testing to a symptom limit regardless of the extent of ischemic ST-segment depression. Moreover, in our patients, marked ischemic ST-segment depression > 0.2 mV was as often noted in patients with a peak heart rate less than 130 beats/min as in patients with a higher peak heart rate.

Thus, the safety of treadmill exercise testing soon after infarction may be more dependent upon the criteria used to select the patient for testing than upon the protocol used to test the patient. Our policy of withholding exercise testing from patients with an S₃ gallop 3 weeks after infarction may prove overly conservative. Other investigators have uneventfully performed treadmill exercise testing 3 weeks after infarction in patients with clinically significant left ventricular dysfunction, many of whom were taking digitalis or diuretics.1, 7, 8

A similar prevalence of ischemic ST-segment depression was seen in both groups at 3 and 11 weeks. Moreover, neither the double product at the onset of ischemic ST-segment depression nor the maximal extent of ischemic ST-segment depression were significantly different between the two groups at 3 or 11 weeks. Further, the double product at which ischemic ST-segment depression occurred bore a strikingly constant relationship to peak double product in both groups of patients who completed exercise tests 3 and 11 weeks after infarction. Thus, for the diagnosis of ischemic ST-segment depression, there appears to be little advantage in exceeding the heart rate of 130 beats/min 3 weeks after infarction.

Is a single treadmill exercise test performed soon
after infarction sufficient to elicit arrhythmic and ischemic abnormalities or should multiple tests be used? Ventricular ectopic activity was more reproducible in our patients who completed a symptom-limited protocol than in those who completed a heart-rate-limited protocol, but this appeared to be of little practical consequence because exercise-induced PVCs were not significantly related to early recurrent cardiac events in either group.

Ischemic ST-segment depression was highly reproducible with either test protocol: over 75% of patients with this abnormality at 3 weeks also had the abnormality at 11 weeks, and over 75% of patients without ischemia at 3 weeks were still free of it at 11 weeks. Thus, a single test at 3 weeks is nearly as effective as a second test at 11 weeks in detecting or in excluding arrhythmic and ischemic abnormalities.

Because the safety of exercise testing as soon as 3 weeks after infarction has been well documented, there appears to be no advantage in delaying this evaluation until a later phase of convalescence. Ventricular function studied by radioisotope methods appears to change little between the time of hospital discharge and several months after infarction. Therefore, most of the improvement in ventricular function — a reflection of tissue healing — appears to occur 3 weeks after clinically uncomplicated infarction. Moreover, some patients will experience recurrent cardiac events 3–11 weeks after infarction. If a single test is to be performed, it should therefore be carried out 3 weeks rather than 11 weeks after infarction.

Of the 12 patients (6%) who experienced a cardiac event between 3 and 11 weeks, seven had ischemic ST-segment depression ≥ 0.1 mV at 3 weeks. Six of these seven patients had this abnormality at a heart rate of 130 beats/min or less. All three patients experiencing fatal or nearly fatal events between 3 and 11 weeks had ischemic ST-segment depression ≥ 0.2 mV at a peak heart rate of 130 beats/min or less on the initial test. These results confirm the prognostic importance of marked ischemic ST-segment depression at a low work load documented by Elgestad et al. in patients with chronic ischemic heart disease. Thus, for the detection of patients at high risk for early recurrent coronary events, heart-rate-limited testing and symptom-limited testing appear to be equally effective.

We have previously documented the lack of prognostic significance of exercise-induced PVCs in patients without clinical heart failure soon after uncomplicated myocardial infarction. The extent of left ventricular dysfunction is known to influence the prognostic significance of ventricular ectopic activity. It is thus not surprising that authors who have evaluated patients with clinically significant left ventricular dysfunction have found exercise-induced PVCs to have prognostic significance.

Aside from the clarification of prognosis, exercise testing performed soon after infarction aids in establishing individualized guidelines for physical activity during early convalescence. Concern with physical activity is one of the most frequent sources of anxiety after infarction. Access to an optimal level of physical activity may alleviate anxiety and depression after infarction. The absence of significant ischemic ST-segment depression, angina pectoris and ventricular ectopic activity during exercise testing provides some assurance that these abnormalities will also be absent during the lesser intensity of effort encountered by patients during their usual activities.

Conclusions

Symptom-limited and heart-rate-limited treadmill exercise tests performed 3 weeks after uncomplicated myocardial infarction are equally safe and effective in detecting ischemic and arrhythmic abnormalities. A single exercise test performed 3 weeks after infarction detects the great majority of ischemic and arrhythmic abnormalities found with testing 11 weeks after infarction.

Exercise-induced ischemic ST-segment depression 3 weeks after infarction that is predictive of early recurrent cardiac events usually occurs at a heart rate of 130 beats/min or less.

References

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