Exercise Testing Soon after Myocardial Infarction

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SUMMARY Forty-six men under age 70, without clinical congestive heart failure or unstable angina pectoris, performed treadmill tests 3, 5, 7, 9 and 11 weeks after myocardial infarction. Patients were more frequently able to perform moderate exertion (2 mph, 14% grade) at 7 and 11 weeks than at 3 weeks following infarction. Ischemic ST-segment depression, usually unaccompanied by angina pectoris, occurred in 45% of patients and was associated with a significantly increased incidence of subsequent coronary events. The presence of exercise-induced ventricular ectopic activity provided little independent prognostic information. No serious complications occurred in 210 tests.

Exercise testing soon after myocardial infarction provides objective information concerning the capacity to resume physical activity, including return to work. Two tests, at 3–5 weeks and at 7–11 weeks, appear to provide most of the information contained in five tests performed during this time.

THERE ARE PRESENTLY NO WELL-DEFINED GUIDELINES for regulating the physical activity of individual patients following the hospital phase of acute myocardial infarction. Most studies of postinfarction patients have focused on physical activity within the hospital or that associated with formal exercise training, commencing 10–12 weeks following infarction. By contrast, there is little information concerning the natural history of changes in cardiovascular status between the time of hospital discharge and the initiation of traditional exercise training programs. This study was designed to examine the response to exercise within the early post-hospital phase, three to 11 weeks following myocardial infarction, with respect to functional capacity, electrocardiographic ST-segment response, and cardiac rhythm. Analysis of the physiologic response of the heart to exercise was felt to be potentially useful in assessing the patient's ability to undertake physical activity, including return to gainful employment.

Patients

All male patients under age 70 admitted to the Stanford Medical Center with a documented myocardial infarction were considered for this study. Patients who demonstrated any of the following at the time of hospital discharge were excluded: 1) angina pectoris at rest or of changing character, 2) clinical congestive heart failure despite medical therapy, and 3) associated conditions, including pulmonary embolization, significant valvular heart disease, hypertension (above 180/100 mm Hg), limiting musculoskeletal abnormality, or inability to return regularly to the Stanford Cardiac Rehabilitation Center. The presence of major arrhythmia, heart block, pump failure or other complications occurring earlier in the hospital course did not exclude the patient if these abnormalities were absent at the time of hospital discharge.

Our patients were classified by the Coronary Prognostic Index of Norris, in which age, prior history of myocardial infarction, and the chest X-ray on admission are combined into a weighted score which is correlated with three-year prognosis. A CPI score of 3 or less was associated with a three-year mortality of 12%, with the mortality rate increasing up to 85% when the Coronary Prognostic Index exceeded 12.

Statistical analysis was performed on an IBM 360 computer, using the Statistical Package for the Social Sciences program. Differences between proportions for responses or observations obtained under two different conditions on the same group of patients were examined using the McNemar test.

Methods

Five treadmill exercise tests were performed, at 3, 5, 7, 9, and 11 weeks following myocardial infarction. Each patient was interviewed and examined by a physician, and informed consent was obtained prior to each test, which was supervised by a physician and a specially trained nurse. Twelve-lead ECGs were recorded at rest, at the end of each 3-minute stage of exercise, and at 1, 2, 3, 5, 7 and 10 minutes of recovery. Leads V₅-V₆ were displayed continuously on a three-channel oscilloscope monitor and recorded for three minutes prior to exercise and during every minute of exercise and recovery. The ECG signal was also recorded continuously on magnetic tape.

Exercise was performed on a motor-driven treadmill using a combination of protocols described by Naughton et al. and (table 1). For this study, the patient was considered to have performed a stage of exercise if he completed at least two minutes of that stage. No patient had eaten or taken a nitric oxide compound within 90 minutes of testing. Most patients started Test 1 at Stage 3 (2 mph, 3.5% grade) or lower, depending on the clinical estimate of their capacity. The work load was increased by one stage every third minute until attainment of an arbitrary "target" heart rate of 130/minute (heart rate-limited tests) or the appearance of any of the following endpoints: 1) symptoms which increased, leading directly to termination of effort, i.e., limiting chest pain, shortness of breath, fatigue, leg cramps or dizziness (symptom-limited tests); 2) staggering gait, blank facies, fall in systolic blood pressure of at least 10 mm Hg below the peak value attained at a prior stage, or the appearance of ventricular tachycardia, i.e., three successive ventricular premature contractions (sign-limited tests). Only three tests were terminated because of lower grade arrhythmias. ST-segment depression was not an indication for terminating the test.

The target heart rate was increased to 140 and 150 beats/minute, respectively, for Tests 2 and 3. Tests 4 and 5, performed at 9 and 11 weeks, were sign or symptom-limited. The initial work load of repeat exercise tests was based on...
the performance on the prior test, and generally commenced at Stage 4 (2 mph, 7% grade), or one stage below that achieved on a prior test, whichever was less. Once a patient attained a level of effort corresponding to Stage 4, this level was used as the initial work load for all subsequent tests.

Ischemic ST-segment depression was defined as a horizontal or down-sloping displacement of the ST segment of at least 0.1 mV below the baseline. Ventricular ectopic activity (VEA) was classified by an adaptation of the method of Jelinek et al.: grade 0 = none, grade A = premature ventricular contractions (PVCs) less than three/minute, grade B = PVCs three or more/minute, grade C = couplets (two successive PVCs), grade D = ventricular tachycardia. Ventricular ectopic activity occurring during exercise and recovery was considered together, whereas VEA occurring during the three-minute initial rest period was considered separately. Grades B, C, and D were considered complex while grade A was considered simple VEA.

No patient participated in a supervised rehabilitation program between the third and eleventh week following infarction, though many engaged in regular physical activity during this time.

Results

Population Characteristics

The mean age of the 46 patients entering the study was 54.4 years (range 36 to 67). Four patients had a history of well-documented remote myocardial infarction, and another 12 had a history of angina pectoris for at least three months prior to infarction. The recent infarction was transmural and anterior in 11 patients, transmural and inferior in 28, and nontransmural in seven patients. Three patients had heart failure within the hospital. One was taking digitalis at the time of entry into the study. The Norris Coronary Prognostic Index was less than 3 in 35 patients, between 3 and 5 in seven patients, and over 6 in four patients. Mean hospital stay after infarction was 14.2 days.

Attendance to Program and Follow-up (fig. 1)

Our 46 patients performed a total of 210 treadmill exercise tests. Four of the 46 patients did not complete the entire test series. Thirty-five of 46 patients entering the study survived without major complications or surgery for a mean follow-up of 18.9 months (median = 18 months) following myocardial infarction.

Test Characteristics and Results (table 2, fig. 2)

Test performance: Serial changes in physical working capacity and hemodynamic response of the 23 patients who were symptom-limited on Test 1 are depicted in table 2 and in figure 2. Heart rate and systolic blood pressure increased significantly between the first test and any subsequent test. For patients limited by symptoms on Test 1, the mean maximal heart rate rose from 115 to 134 beats/minute by the fifth test (P < 0.001). Mean maximal systolic blood pressure rose from 157 to 178 mm Hg (P < 0.001). Heart rate at a moderate work load (Stage 4) dropped from 107 to 96 beats/minute between Test 1 and Test 5 (P < 0.001). Similar results were obtained when patients taking propranolol were excluded.

Eighteen percent of the patients completing the test series reached Stage 6 (2 mph, 14% grade) during Test 1 (4/22). Over half the patients (12/22) did so by the 7th week (P < 0.05), and 91% (20/22) did so by the 11th week after the acute event (P < 0.001).

No significant difference in peak stage attained at 11 weeks was found between subjects with respect to anterior vs posterior infarction, transmural vs nontransmural infarction, or the presence or absence of a prior history of myocardial infarction or of angina pectoris.

Eight tests were terminated due to ventricular arrhythmia, including five due to ventricular tachycardia. Limiting angina pectoris occurred on 23 tests performed in ten patients. Five patients had angina on two or more tests, while in another five it was noted only once. Patients without angina on any test reached a significantly higher stage of exertion on Test 5 than patients with angina (P < 0.01).

ST-segment Depression during and after Exercise

ST-segment elevation greater than 0.5 mm was noted prior to exercise in 30 tests. Resting, nonspecific ST-segment depression of between 0.5 and 0.9 mm was seen in 30 tests. Ischemic ST-segment depression of 1 mm or more at rest was not noted. Further ischemic ST-segment depression of 1 mm below baseline was noted in 54 tests in 21 patients and was not more frequent in patients with resting ST-segment abnormalities compared to those with normal ST segments. The incidence of ST-segment depression in Test 1 (17%) and Test 5 (31%) was not significant. Ten of 21 patients with ischemic ST-segment depression showed this finding on three or more tests. There was a significant (P < 0.005)

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**Table 1. Treadmill Exercise Test Protocol**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Speed (mph)</th>
<th>Grade (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.0</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>2.0</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>2.0</td>
<td>3.5</td>
</tr>
<tr>
<td>4</td>
<td>2.0</td>
<td>7.0</td>
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<tr>
<td>5</td>
<td>2.0</td>
<td>10.5</td>
</tr>
<tr>
<td>6</td>
<td>2.0</td>
<td>14.0</td>
</tr>
<tr>
<td>7</td>
<td>2.0</td>
<td>17.5</td>
</tr>
<tr>
<td>8</td>
<td>3.0</td>
<td>12.5</td>
</tr>
<tr>
<td>9</td>
<td>3.0</td>
<td>15.0</td>
</tr>
<tr>
<td>10</td>
<td>3.0</td>
<td>17.5</td>
</tr>
</tbody>
</table>

**Figure 1. Clinical course of 46 patients entering the program, three weeks following infarction. VF = ventricular fibrillation, CABG surgery = coronary artery bypass graft surgery.**
association between angina and ischemic ST-segment depression during a given test (fig. 3).

Ventricular Ectopic Activity (table 3)

VEA during a three-minute period prior to exercise was recorded in 19 patients. Fourteen patients had complex PVCs. All but one patient with PVCs recorded at rest also showed PVCs during or after exercise. The incidence of VEA rose from 9% to 20%, and the incidence of complex VEA rose from 4.4% to 12.5% between Tests 1 and 5 (NS).

Ventricular ectopic activity during or after exercise was seen at least once in 33 patients, while complex PVCs were noted in 27 patients. The incidence of all VEA rose from 26% on Test 1 to 60% on Test 5 (P < 0.01), while the incidence of complex VEA rose from 20% to 40% during the same period (P < 0.05). Short runs of ventricular tachycardia were noted in five tests in four patients. In all of these tests, couples were present prior to the appearance of ventricular tachycardia. Couples not progressing to ventricular tachycardia were noted eleven times in another seven patients. In 41 patients completing the test series, VEA tended to occur at approximately the same heart rate in 13 patients, while another 17 demonstrated VEA occurring in a seemingly random fashion. A significant relationship was noted between exercise-induced ST-segment depression and the presence of VEA (P < 0.001) (fig. 4). By contrast, no statistically significant relationship was noted between angina and the presence of VEA in a given test.

It is possible that the increase in heart rate between Tests 1 and 5 might itself have been responsible for the increase in frequency in VEA. To exclude this possibility, the event of interest was defined to be the occurrence of VEA at a heart rate of 120 beats/minute or less. Significantly more patients demonstrated this event at 11 than at 3 weeks (P < 0.01).

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** The highest stage of effort at 3 weeks, 7 weeks and 11 weeks after infarction is shown for the 22 patients limited by symptoms during Test 1, and who completed the test series.

![Figure 3](http://circ.ahajournals.org/)

**Figure 3.** Relationship between exercise-induced ischemic ST depression and angina.
Moreover, no significant differences were found between the mean heart rate at which VEA occurred, i.e., 101 and 104 beats/minute at 3 and 11 weeks, respectively, after infarction. Similarly, a heart rate independent increase in the frequency of complex VEA was demonstrated ($P < 0.01$).

**Other Arrhythmias; Complications**

Supraventricular premature beats were infrequent and were not computed. No episode of atrial tachyarrhythmia, second or third degree atrioventricular block or ventricular fibrillation was observed. There was no persistent chest pain and no myocardial infarction related to exercise testing. One patient with episodes of ventricular bigeminy at rest developed a short run of ventricular tachycardia during recovery from Test 3. No evidence of cardiac damage was noted following his admission to the hospital. One patient collapsed at home during heavy physical effort 18 hours after performing Test 3. He died two days later from recurrent ventricular fibrillation. One patient was successfully defibrillated following cardiac arrest one week after performing Test 1.

**Reproducibility of Ischemic ST-segment Depression and Ventricular Arrhythmias**

Test 1 identified 48% of the patients who exhibited exercise-induced ischemic ST-segment depression during any test. Likewise, 36% of patients with VEA were identified by Test 1. Test 4 or 5 identified approximately two-thirds of all patients with ischemia or VEA. The combination of Test 1 and 5 or 3 and 5 identified between 80 and 90% of all patients with ST-segment depression or VEA; other combinations yielded slightly poorer results. A substantial fraction of all patients was free of complex PVCs or ischemic ST-segment depression on any test: 25 of 46 had no complex PVCs, 25 of 46 had no ischemic ST-segment depression, and 16 of 46 had neither abnormality during the test series.

**Correlation of Test Results and Clinical Outcome**

The association between clinical coronary events and the presence of ischemic ST-segment depression and ventricular ectopic activity was examined in 42 patients who did not have coronary artery bypass graft surgery.

**Ischemic Depression (table 4)**

All patients who died suddenly (2), survived an episode of ventricular fibrillation (1), or survived a recurrent myocardial infarction (4), had ST-segment depression on at least one test. Two of these patients had experienced angina pectoris during their ordinary activities, and both experienced angina during treadmill testing. Another patient, free of angina during exercise testing, had experienced it during ordinary activities. Coronary events during the follow-up period were significantly more frequent among patients with

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**Table 3. Ventricular Ectopic Activity at Rest and During and Following Exercise**

<table>
<thead>
<tr>
<th>Tests</th>
<th>#1 (No. %)</th>
<th>#2 (No. %)</th>
<th>#3 (No. %)</th>
<th>#4 (No. %)</th>
<th>#5 (No. %)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>At Rest</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 0</td>
<td>42 91.3</td>
<td>39 90.1</td>
<td>33 78.6</td>
<td>27 77.1</td>
<td>32 80.0</td>
</tr>
<tr>
<td>Grade A</td>
<td>2 4.3</td>
<td>1 2.3</td>
<td>5 11.9</td>
<td>5 14.3</td>
<td>3 7.5</td>
</tr>
<tr>
<td>Grade B</td>
<td>2 4.3</td>
<td>3 7.0</td>
<td>4 9.5</td>
<td>3 8.6</td>
<td>4 10.0</td>
</tr>
<tr>
<td>Grade C</td>
<td>0 0.0</td>
<td>0 0.0</td>
<td>0 0.0</td>
<td>0 0.0</td>
<td>1 2.5</td>
</tr>
<tr>
<td>Grade D</td>
<td>0 0.0</td>
<td>0 0.0</td>
<td>0 0.0</td>
<td>0 0.0</td>
<td>0 0.0</td>
</tr>
</tbody>
</table>

**During and Following Exercise**

| Grade 0 | 34 73.9 | 25 58.1 | 22 52.4 | 15 42.9 | 16 40.0 |
| Grade A | 3 6.5 | 9 20.9 | 3 7.1 | 8 22.9 | 8 20.0 |
| Grade B | 8 17.4 | 7 16.3 | 13 31.0 | 7 20.0 | 12 30.0 |
| Grade C | 1 2.2 | 2 4.7 | 2 4.8 | 3 8.6 | 3 7.5 |
| Grade D | 0 0.0 | 0 0.0 | 0 0.0 | 2 4.8 | 2 5.7 | 1 2.5 |

See text for definition of ventricular activity grade.

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**Table 4. Post-MI Exercise Testing: Clinical Outcome (Patients Not Undergoing CABG Surgery)**

<table>
<thead>
<tr>
<th>Coronary events*</th>
<th>No coronary events</th>
<th>Total pts</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST depression</td>
<td>7</td>
<td>10</td>
</tr>
<tr>
<td>No ST depression</td>
<td>0</td>
<td>25</td>
</tr>
<tr>
<td>Total pts</td>
<td>7</td>
<td>35</td>
</tr>
</tbody>
</table>

*Sudden death (2), VF survival (1), MI survival (4).
an ischemic test than in those without this finding \((P < 0.01)\). Analysis of resting ST segments prior to exercise testing revealed no difference between patients with isoelectric, elevated, or depressed ST segments with respect to subsequent cardiac events.

**Ventricular Ectopic Activity (table 5)**

Of eight patients with exercise-induced couplets or ventricular tachycardia who did not undergo coronary surgery, one died during the follow-up period. Complex PVCs were not seen in one patient resuscitated from ventricular fibrillation or in one patient who died suddenly.

**Discussion**

There are no prior studies in which treadmill exercise testing has been used to estimate physical working capacity during early convalescence from acute myocardial infarction. This lack of information reflects concern with the potential hazards of exercise testing soon after myocardial infarction. These concerns may be unwarranted, however: there have been no major complications in 376 patients tested as soon as three weeks following infarction.6,9 Most investigators have employed a target heart rate of 120-130/minute and a work load of 100-900 kpm/minute.

Half of our patients tested at three weeks following infarction were symptom-limited, and one-third were heart rate-limited. As a whole, patients completing the test series performed a higher level of exercise by the seventh week than during Test 1, whether the initial test was symptom-limited or heart rate-limited. Since the initial work load was not constant for each patient or for each test, and since oxygen consumption was not directly measured, definitive conclusions concerning changes in physical working capacity cannot be drawn from this study. However, our data suggest that the physical working capacity of our patients improved substantially between the third and eleventh week following myocardial infarction.

Physical working capacity was not related to the location of infarction, a history of coronary heart disease prior to infarction, or to the extent of infarction, i.e., transmural vs nontransmural. The features of the hospital course following infarction also played a minor role in determining exercise performance in Kentala's unselected patients.10

The patient's greater familiarity with the procedure and improved confidence in his abilities probably contributed to improved performance, especially when tests were symptom-limited at 9 and at 11 weeks following infarction. While no patients engaged in supervised physical training, many exercised regularly at progressively increasing levels, and may have been encouraged to do so by the absence of ill effects on previous tests. Even nonsupervised physical activity appears to improve the physical working capacity of patients recovering from infarction.10 A "training effect" on the cardiovascular system of our patients is suggested by the progressive reduction of heart rate measured at a given stage of exercise.11-13 Studies performed in man and in animals demonstrate little or no improvement in left ventricular function during the time course of our study.15,16 suggesting that change in left ventricular performance was not a major determinant of the marked improvement in physical working capacity we noted. Treatment with antiarrhythmic medication may also have contributed to the increase in physical working capacity in a few patients by preventing the occurrence of limiting VEA during exercise.15 Treatment with beta blocking drugs also may have enhanced exercise performance, but only 5% of patients were receiving these drugs for treatment of angina pectoris.

Our patients obviously constitute a select group. For example, only four of 46 (9%) of our patients had a Coronary Prognostic Index over 6, whereas this value was present in approximately 40% of Norris' unselected patients.1 Nonetheless, the peak heart rate of our patients at a comparable time following infarction was similar to that of Kentala's and Sanne's unselected patients performing bicycle ergometry 6-8 and 12 weeks following infarction.10,16 Therefore, despite the selected nature of our patient group, there is general conformity with unselected series with respect to physical working capacity.

Most individuals utilize 30-40% of their maximum oxygen transport capacity during an 8-hour working day.19 Over half our patients reached Stage 6, demonstrating the capacity to perform occupational work, e.g., auto repair, janitorial work, as early as seven weeks postinfarction.19 Ninety-one percent did so at 11 weeks. If functional capacity were the only consideration, sedentary workers may well have returned to work even earlier.

Angina pectoris occurred at least once in ten patients (22%). However, for any of the five tests, the maximum incidence was only 14%, considerably lower than the 33% and 41% incidence reported in other series of less selected patients.10,16 Patients not limited by angina pectoris attained a significantly higher work load than those with angina. In Sanne's series of unselected patients studied 12 weeks after infarction, those limited by fatigue or dyspnea exercised to 85-90% of the physical working capacity attained by an age-matched normal population, compared to a value of 57% for those limited by angina. Exercise-induced angina pectoris was not associated with an increased incidence of VEA in our patients, whereas ischemic ST-segment depression was strongly correlated with VEA.

Despite the small size of our group, these data indicate that patients with exercise-induced ischemic ST-segment depression have a significantly higher incidence of cardiac events, i.e., sudden death, ventricular fibrillation, and infarction, than patients without exercise-induced ischemic ST-segment depression. This finding is of particular importance since four of these seven patients were free of spontaneous or exercise-induced angina pectoris.

All four of our patients undergoing coronary artery surgery demonstrated major ischemic ST-segment depression of 2 mm or more. In three of these patients, angiography revealed major obstructions in the coronary arteries supplying noninfarcted myocardium. This corroborates the observations of Savran et al. who found that patients with major

<table>
<thead>
<tr>
<th>Table 5. Post-MI Exercise Testing: Clinical Outcome (Patients Not Undergoing CABG Surgery)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary events*</td>
</tr>
<tr>
<td>Complex VEA</td>
</tr>
<tr>
<td>No complex VEA</td>
</tr>
<tr>
<td>Total pts</td>
</tr>
</tbody>
</table>

*Sudden death (2), VF survival (1), MI survival (4).
obstructions in vessels supplying noninfarcted myocardium were correctly identified by exercise testing (11 of 12 cases), while those without this pattern were correctly identified in 18 of 19 cases.18

More than half of all patients with ischemic ST-segment depression were free of angina pectoris. Likewise, nearly three-fourths of tests exhibiting ischemic ST-segment depression were unassociated with angina pectoris. This underscores the importance of exercise testing in the detection of latent myocardial ischemia. An association between exercise-induced ST-segment depression and coronary mortality has long been noted in asymptomatic patients and in patients with chronic coronary heart disease.19,20 To our knowledge, the association between exercise-induced ST-segment depression during early convalescence from myocardial infarction and an increased incidence of subsequent cardiac events has not previously been described.

A major objective of this study was to characterize VEA occurring during exercise soon after myocardial infarction. Exercise was particularly useful in demonstrating advanced grades of ventricular arrhythmia, as previously noted by Jelinek et al.8 Our patients demonstrated a 16-fold increase in the frequency of ventricular couplets and ventricular tachycardia during or following exercise. Only one high-grade ventricular arrhythmia (grade C) was detected on the three-minute resting ECG.

The incidence of all grades of exercise-induced VEA increased significantly between Test 1 and Test 5. This increase was at least partly independent of increases in heart rate and work load. Only 20% of our patients had VEA of some type on Test 5, compared with a 65% incidence in Crawford’s 60 patients studied at a mean of 17 months following acute myocardial infarction, and with 56% of Ryan’s 100 nonselected patients with chronic coronary disease.21,22 Couplets or ventricular tachycardia were noted in 10% of our patients performing Test 5, compared with an incidence of 20% in Ryan’s patients. Thus, patients in the early phase of convalescence from myocardial infarction do not appear to demonstrate an unusually high incidence of exercise-induced VEA compared to other groups of patients with ischemic heart disease. Ericsson found a higher mortality during a three-month follow-up period among patients with exercise-induced VEA at three weeks following infarction, compared with patients without VEA at this time.6 Larger series of patients are needed to elucidate the relationship between exercise-induced VEA noted soon after infarction, and subsequent mortality.

Conclusions

Exercise testing soon after myocardial infarction provides objective information concerning the capacity for physical exercise, which may be useful in planning physical rehabilitation and return to work following myocardial infarction. Two tests, performed at 3–5 weeks and again at 7–11 weeks following infarction, appear to provide most of the information concerning physical working capacity, ischemic ST-segment depression and VEA which is contained in five tests performed during this time.

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