Thirty-Month Follow-up of Maximal Treadmill Stress Test and Double Master’s Test in Normal Subjects

By Wilbert S. Aronow, M.D.

SUMMARY

One hundred normal subjects who had a double Master’s test and a maximal treadmill stress test (MTST) were clinically evaluated 30 months later. Ninety-nine of these 100 subjects (99%) had a follow-up treadmill test at 30 months. One of four subjects (25%) with initially an abnormal double Master’s test developed coronary heart disease within 30 months. Three of 96 subjects (3.1%) with initially a normal double Master’s test developed coronary heart disease within 30 months. Three of 13 subjects (23.1%) with initially an abnormal MTST developed coronary heart disease within 30 months. The MTST correlated better than the double Master’s test in predicting subsequent coronary heart disease.

Additional Indexing Words: Electrocardiography Exercise Treadmill

MAXIMAL TREADMILL stress testing has been used by some investigators to detect latent subclinical myocardial ischemia due to coronary artery disease.1-3 We have previously reported3 that 13 of 100 clinically normal subjects (13%), mean age 51 ± 6 years, had ischemic S-T segment depression of at least 1 mm following a maximal treadmill stress test. Four of these 100 normal subjects (4%) had at least 1 mm of ischemic S-T segment depression following a double Master’s two-step test.4 All four normal subjects who had an abnormal double Master’s test also had an abnormal maximal treadmill stress test.3

Follow-up data from asymptomatic people tested with the maximal treadmill stress test need to be obtained to determine the value of this stress test in predicting latent coronary artery disease. Therefore, we are reporting 30-month follow-up clinical data from our 100 previously asymptomatic subjects who had a maximal treadmill stress test3 and a double Master’s test.4 In addition, 99 of these 100 subjects (99%) had a repeat maximal treadmill stress test at their 30-month follow-up.

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Subjects and Methods

The 100 clinically normal subjects who previously had a double Master’s two-step test4 and a maximal treadmill stress test3 were followed for 30 months. They included 98 men and two women who were between ages 40 and 67 years, with a mean age of 53.5 ± 6 years, at the 30-month follow-up. They were hospital personnel or their friends. All normal subjects had a normal 12-lead resting electrocardiogram and blood pressure below 140/90 mm Hg at the time of their initial treadmill stress test. None of them was on any medication.

Ninety-nine of the 100 subjects (99%) had a treadmill stress test at the 30-month follow-up. Ninety-five asymptomatic subjects had a maximal treadmill stress test as previously reported.3 Four subjects who now had angina pectoris due to angiographically documented significant coronary artery disease with at least 75% narrowing of one or more major coronary vessels performed a submaximal treadmill stress test. None of the 99 subjects was on any medication at the time of the treadmill stress test. The treadmill stress test was performed at least 2 hours after a light meal.

The treadmill stress tests were performed as previously described.3 Leads I, aVF, and V5 were recorded in the supine and standing positions before exercise. A multistage uninterrupted treadmill stress test similar to that described by Doan and associates1 was then performed. The patients were monitored with lead V5 throughout exercise with an oscilloscope. Leads V5, aVF, and I were recorded in that order each minute during exercise, continuously after 75% of the predicted maximal heart rate3 was reached, immediately after exercise in the upright and supine positions, and in the supine position every minute after exercise for at least 6 min. Blood pressures were recorded before and after exercise.
The asymptomatic subjects were exercised until they reached 100% of their predicted maximal heart rate or exhaustion. Three of the four subjects with angina pectoris exercised until the onset of angina; the fourth subject with angina pectoris exercised until he reached 90% of his maximum predicted heart rate.5

The criterion for an abnormal exercise test was 1.0 mm or more of ischemic S-T segment depression below the resting level, with either the S-T segment extending horizontally for at least 0.08 sec or with downward sloping of the S-T segment.

The electrocardiograms were reviewed by the author after the study was completed. He did not know from whom the tracings under review were obtained.

Results

Ninety-six of the 100 normal subjects (96%) experienced no clinical manifestations of coronary artery disease during the 30-month follow-up period. Four of the 100 normal subjects (4%) developed angina pectoris during the 30-month follow-up period. Two of these four subjects had significant three-vessel disease visualized by coronary angiography, and one of these two subjects had experienced a transmural myocardial infarction. One subject with angina had significant two-vessel disease visualized by coronary angiography. The fourth subject with angina had a 75% occlusion of his left anterior descending coronary artery visualized by coronary angiography.

Table 1 shows that one of the four subjects (25%) who initially had at least 1 mm of ischemic S-T segment depression following a double Master's two-step test developed angina pectoris due to angiographically documented significant coronary artery disease within the 30-month follow-up period. This subject also had experienced a transmural myocardial infarction. Three of the 96 subjects (3.1%) who did not have at least 1 mm of ischemic S-T segment depression following the double Master's two-step test developed angina pectoris due to angiographically documented significant coronary artery disease within the 30-month follow-up period.

The data in table 1 were analyzed using a chi-square test. Normal subjects with an initially abnormal double Master's test were more likely to develop coronary heart disease within 30 months than normal subjects with a normal double Master's test (chi-square = 4.79; P < 0.03).

Table 2 indicates that three of the 13 subjects (23.1%) who initially had at least 1 mm of ischemic S-T segment depression following a maximal treadmill stress test developed angina pectoris due to angiographically documented significant coronary artery disease within the 30-month follow-up period. One of these three subjects also had experienced a transmural myocardial infarction. One of the 87 normal subjects (1.1%) who initially had a normal maximal treadmill stress test developed angina pectoris due to angiographically documented significant coronary artery disease. This normal subject also initially had a normal double Master's test.

The data in table 2 were analyzed using a chi-square test. Normal subjects with an initially abnormal maximal treadmill stress test were more likely to develop coronary heart disease within 30 months than normal subjects with a normal maximal treadmill stress test (chi-square = 14.16; P < 0.001).

Of interest, two of the four subjects who were asymptomatic and had at least 1 mm of ischemic S-T segment depression following their initial double Master's test and maximal treadmill stress test went on a diet low in saturated fats, sugars, and calories, stopped smoking, and went on an exercise program. These two subjects remained asymptomatic and had normal maximal treadmill stress tests at their 30-month follow-up. One of the four subjects remained asymptomatic and had an abnormal maximal treadmill stress test at his 30-month follow-up.

Of the 13 normal subjects who initially had an abnormal maximal treadmill stress test, three

Table 2

Incidence of Coronary Artery Disease Developing within 30 Months in Normal Subjects with Initially Abnormal or Normal Maximal Treadmill Stress Test

<table>
<thead>
<tr>
<th>Incidence of coronary artery disease</th>
<th>Normal subjects</th>
<th>No.</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormal maximal treadmill stress test</td>
<td>13</td>
<td>3</td>
<td>23.1</td>
<td></td>
</tr>
<tr>
<td>Normal maximal treadmill stress test</td>
<td>87</td>
<td>1</td>
<td>1.1</td>
<td></td>
</tr>
</tbody>
</table>

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subjects (23.1%) developed coronary heart disease and had an abnormal follow-up maximal treadmill stress test. Four of these 13 subjects (30.8%) remained asymptomatic and had a normal follow-up maximal treadmill stress test. Six of these 13 subjects (46.1%) remained asymptomatic and had an abnormal follow-up maximal treadmill stress test.

Of the 87 normal subjects who initially had a normal maximal treadmill stress test, one subject (1.1%) developed coronary heart disease and had an abnormal follow-up maximal treadmill stress test. Three of these 87 subjects (3.5%) were asymptomatic and had an abnormal follow-up maximal treadmill stress test. One subject remained asymptomatic and did not have a follow-up maximal treadmill stress test. Eighty-two of the 86 subjects (95.4%) were asymptomatic and had a normal follow-up maximal treadmill stress test.

Discussion

Ischemic S-T segment depression in asymptomatic people provoked by a double Master’s test,6-10 by a submaximal treadmill stress test,11 and by a near-maximal treadmill stress test12 has been correlated with an increased probability of developing subsequent overt coronary heart disease.

Bruce and his associates13 found no evidence of coronary events in a 3-year period following detection of an abnormal maximal treadmill stress test. However, at the 5-year follow-up period, Bruce and McDonough14 reported that three of 22 normal people (13.6%) who initially had an abnormal maximal treadmill stress test developed clinical evidence of coronary heart disease. Only two of 199 normal people (1%) who initially had a normal maximal treadmill stress test developed clinical evidence of coronary heart disease.

Our data show that more asymptomatic people who develop clinically apparent coronary heart disease within 30 months will have initially an abnormal maximal treadmill test than an abnormal double Master’s test. Biostatistical analysis of our data also shows that the maximal treadmill stress test correlated better than the double Master’s test in predicting subsequent clinically apparent coronary heart disease. However, there was a greater number of false-positive maximal treadmill stress tests than double Master’s tests in our asymptomatic subjects. In addition, the probability of developing subsequent clinically apparent coronary heart disease was similar for those asymptomatic individuals who had an initially abnormal double Master’s test or an initially abnormal maximal treadmill stress test.

Therefore, our data confirm that the maximal treadmill stress test and the double Master’s test are useful in predicting the subsequent development of clinically significant coronary heart disease. Asymptomatic individuals with an abnormal maximal treadmill stress test or an abnormal double Master’s test should be considered as individuals who have a high risk for developing clinical coronary heart disease and should be treated intensively with a program aimed at reduction of risk factors for coronary heart disease.

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