The Prognosis of an Abnormal Electrocardiographic Stress Test

By Joseph T. Doyle, M.D., and Sandra H. Kinch, M.S.

SUMMARY
Treadmill walking for 10 min at 3 mph against a 5% grade has been used as an electrocardiographic (ECG) stress test as part of a prospective epidemiologic study of 2,437 men. From 1953 through 1966, 22,223 tests have been done without untoward event.

The criteria of an abnormal postexercise ECG were ischemic flattening or coving of the S-T segment, T-wave changes consistent with focal left ventricular epicardial ischemia, and paroxysmal left bundle-branch block.

Of the 2,003 men exercised two or more times, 264 developed some manifestation of ischemic heart disease (IHD) and in 75 (30%) this was an abnormal ECG response to exercise. They had higher blood pressures and were more often heavy smokers than normal responders. Body weight and serum cholesterol were similar in the two groups. Over the next 5 years there was an 85% probability that these abnormal responders would develop angina pectoris or experience a myocardial infarction.

These relatively insensitive but highly specific and reproducible ECG criteria accurately identify men with clinically silent but far-advanced coronary atherosclerosis, attested by the poor prognosis of an abnormal response. An abnormal postexercise ECG is valid evidence of IHD. A submaximal ECG stress test is useful in clinical and epidemiologic studies and might be useful in assessing the effectiveness of efforts to reduce the risk of IHD.

Additional Indexing Words:
Ischemic (coronary) heart disease  Exercise  Treadmill  Epidemiology

It is generally, although not universally, accepted that transient abnormalities of intraventricular conduction and of ventricular repolarization appearing during or after exercise are manifestations of reversible myocardial ischemia. In the absence of myocardial or valvar dysfunction, anemia, electrolyte disturbance, or digitalis, these electrocardiographic abnormalities are imputed to the inability of coronary arteries narrowed by atheroma to deliver enough blood to meet the immediate energy requirements of the increased myocardial work load. The electrocardiographic criteria of induced myocardial ischemia and their sensitivity and specificity have been long and inconclusively argued. Many types of exercise tests have been used, mostly submaximal but some maximal. Whether exercise tests can be standardized or, indeed, whether standardization is really necessary is a still unresolved question.\(^1\)\(^-\)\(^9\) This report is concerned not, however, with these problems but with the frequency with which ischemic heart disease (IHD) can be inferred from arbitrary electrocardiographic criteria in middle-aged men subjected to a moderately stiff exercise test. The topic of exercise electrocardiography has been extensively and authoritatively reviewed, most recently by Bruce and Hornsten.\(^9\)

Methods
The participants in a prospective study of degenerative circulatory disease, known as the Albany Cardiovascular Health Center, have been routinely subjected to an electrocardiographic...
stress test. The Cardiovascular Health Center project was established at the Albany Medical College by the New York State Health Department in 1952 and has been fully described elsewhere. In brief, 1,913 male employees of New York State born in the years 1898 to 1913 and working in or near Albany (89% of those eligible), have agreed to place themselves under continuous medical surveillance. On entry into the study a comprehensive lifetime medical and socioeconomic history was obtained, a complete physical examination was done, and a number of laboratory tests were performed. The tests included measurements of hemoglobin and hematocrit readings, urinalysis, anteroposterior and left lateral roentgenograms of the heart and lungs, an electrocardiogram, and the blood lipid profile. No participant was excluded because of preexisting heart disease. At least biennially, and usually annually, since he has come under observation the interim medical history of each participant has been ascertained, and the physical examination as well as the battery of laboratory tests already enumerated has been repeated. The annual attrition rate from all causes has been less than 5%. Only 33 men are completely lost to follow-up. An additional 621 men, mostly members of the New York State Legislature and appointed State officials have been gradually added to the base study and have been examined one or more times. Since this group differs in no important respect from the core group, they have been included in this report.

A major objective of the Cardiovascular Health Center has been to evaluate the worth of various diagnostic methods in the detection of preclinical IHD. An electrocardiographic stress test has, accordingly, been a part of the examination routine since the inception of the study. Although a maximal stress test should have a higher diagnostic yield, the fear of medicolegal complications has dictated the use of a submaximal test. The first 617 participants did a double two-step test exactly as specified by Master. All subsequent tests have consisted of walking for 10 min at 3 mph on a motor-driven treadmill set at a 5% grade.

Tests are performed only at the discretion of the physician who has reviewed the history, done the physical examination, and examined the resting electrocardiogram. Electrocardiographic abnormality does not exclude the subject from the exercise test. Usually, although not invariably, a well-documented history of previous myocardial infarction or of typical Heberden's angina has been considered a contraindication to the exercise test. The only other contraindication has been orthopedic disability. No attempt is made to control eating or smoking. The test is supervised by a technician although a physician is always in the immediate vicinity. The participant is cautioned to stop exercise should he develop pain, breathlessness, apprehension, or unusual fatigue. The technician is instructed to stop the test immediately if, in her judgment, the subject appears to be in trouble.

All electrocardiograms are recorded in recumbency on a direct-writing multichannel instrument at a paper speed of 50 mm/sec. The limb-lead electrodes are left in place during exercise. Suction leads are used on the chest. Immediately after exercise and again 3 min later the lateral precordial leads and leads III, II, and I are recorded. Leads taken at 6 min were found to contribute no further diagnostic information and are no longer recorded. Continuous direct-wire monitoring of the precordial electrocardiogram has revealed no changes confined to, or peculiar to, the exercise period in a small group of subjects in the study and in 100 medical students who performed a maximal exercise test. Indeed, abnormalities considered to be evidence of acute myocardial ischemia induced by the exercise tests are typically persistent for some minutes.

From February 1953 through December 1966, 2,437 of 2,534 men have had one or more exercise tests. A total of 22,223 exercise tests has been done on these men. Two hundred four tests on 154 men were incomplete. The most common reasons for stopping tests before completion included dyspnea, fatigue, leg pain or cramps, dizziness, intense anxiety and, in a very few individuals, the appearance of anginal pain. There have been no recognized complications of the test and no fatalities.

Initially, the criteria of Master were rigorously applied to the postexercise electrocardiogram. As then promulgated, by these criteria almost any change in the postexercise electrocardiogram other than an increase in heart rate was construed as evidence of coronary insufficiency. Moreover, the absence of such changes in the postexercise electrocardiogram was held to exclude coronary heart disease. Fewer than 50% of our symptomless, vigorous, and apparently healthy men could pass the Master's test so evaluated. Although it was conceded that most of these men indubitably had some degree of coronary atherosclerosis, it seemed implausible that so large a number had clinically significant impairment of coronary blood flow under the conditions of the double two-step test.

It was, therefore, decided early in 1954 to require as evidence of induced acute coronary insufficiency one or more of the following: (1) flattening or downward inclination for 0.10 sec or longer or cove-plane deformity of the S-T segment irrespective of the presence or absence of
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junctional (J) displacement; (2) a change in the direction of the T wave consistent with focal left ventricular epicardial ischemia; or, (3) paroxysmal left bundle-branch block. These criteria were admittedly arbitrary and were based on a combination of empirical observation and theoretical assumption.

Not long afterwards at a conference on March 8 and 9, 1956, at the Walter Reed Army Hospital, Mattingly and Robb presented rather similar criteria used by them for some years and validated on the basis of a prospective study of a large group of military personnel followed for 10 years.12 The QX:QT ratio has occasionally been helpful in verifying a suspected ST-segment deformity.8 Equivocal results of electrocardiographic stress tests are for clinical and statistical purposes considered to be normal. The degree of abnormality of abnormal exercise responses was also classified by the method of Robb and Marks.8 All exercise tests have been interpreted by the same reader and reviewed blind at least once. The repeatability of readings has been 96%.

The life table technique has been used to assess the prognostic implication of the response to the electrocardiographic stress tests. This is a well-known statistical device which has advantages over the commonly used cumulative or average annual incidence rate. The actual period of exposure to risk is taken into account in the analysis, a particularly important procedure since, as the period of observation increases in prospective studies, increasing numbers of the group may be lost to observation or succumb to some disease other than the one under study. As the method is used here, actual months of observation rather than the customary one-half interval estimate are used in obtaining person years at risk. Age and calendar time have been confounded on the fundamental assumption that the age-specific risk of ischemic heart disease (IHD) has remained relatively constant over calendar time. Length of follow-up is calculated by subtracting the age at entry time 0 from the year of withdrawal.

For each year of age and for cumulative periods of 5, 10, 15, and 20 years after age 40, the probabilities of developing the event under study, in the presence of the competing risk, are calculated. The primary event and the competing event are specifically defined for each comparison. This method, its assumptions, and a detailed description of the calculations appear in the technical appendix of an earlier publication.13

Two sets of probabilities have been prepared: The first sets forth the probabilities of an individual's developing an abnormal electrocardiographic response to exercise as the primary manifestation of IHD. The competing risk is any other manifestation of IHD, or loss to observation. The individual enters the life table at the time of his first exercise test. The second sets forth the probabilities of developing an additional manifestation of IHD following an abnormal exercise response. The competing risk is death from any cause other than IHD or loss to observation. The individual enters the life table experience at the time of his first abnormal response and is withdrawn at the first event of IHD or at his last visit to the Center.

To evaluate the risk factor status of the individuals with an abnormal exercise response compared with those who had no evidence of IHD, a sample of 75 controls was selected from the total disease-free population matched to the case group by date of birth and date of observation. The date of observation was the date of the first diagnosis of an abnormal electrocardiographic stress test. The variables compared were serum total cholesterol concentration, systolic and diastolic blood pressures, total body weight, and cigarette smoking. A matched-pair analysis was applied to the actual measurements for the first four variables while the proportion of current cigarette smokers in the two groups was compared.

Results

Of a total of 2,534 men examined at the Cardiovascular Health Center, 2,437 were exercised at least once. The following categories have been excluded from analysis: (1) 97 men who were not exercised, mostly because of orthopedic disabilities; (2) 45 men with antecedent IHD (myocardial infarction, 23; angina pectoris, 22); and (3) 361 men with normal postexercise electrocardiograms who continued under surveillance but were not again stress tested. Two hundred and ninety-one men of this third group were not reexamined either because they refused to return or because they had entered the study within less than 2 years of the cutoff date. The 28 men who had an abnormal electrocardiographic response to their first exercise test are considered separately. Of the remaining 2,003 men, 75 have subsequently exhibited abnormal exercise responses, 189 have had another manifestation of IHD, 99 have died from other causes, and 33 have been lost to follow-up. Severe myocardial or valvar disease, anemia, and digitalis have been excluded as possible causes of exercise-induced electrocar-
The first manifestation of IHD has been an abnormal electrocardiographic response to exercise in nearly one third of all cases. Figure 1 summarizes this enumeration.

Seventy-five men had an abnormal electrocardiographic response to exercise. Thirty-four (45%) subsequently developed other evidence of IHD in the period covered by this report. Fifty-seven had an unequivocally normal resting electrocardiogram. Twenty-four of these men (41%) subsequently developed other evidence of IHD. Only six (11%) had a postexercise electrocardiogram with a junctional depression of 2 mm or more (Robb, grade 3). The other 17 men had resting electrocardiographic abnormalities due about equally to left ventricular enlargement consequent to arterial hypertension or to nonspecific T-wave changes. One third of this smaller group had Robb grade 3 changes in the postexercise electrocardiogram and nearly two thirds later developed other manifestations of IHD.

Figure 2 shows the 5-year cumulative probabilities after age 40 of not developing an abnormal exercise response as the first manifestation of IHD. This probability declined from 998 per 1,000 persons (99.8%) during the first year to 940 per 1,000 (94.0%) over a 20-year span.

Figure 3 shows the 5-year cumulative probabilities of not developing angina pectoris or a myocardial infarction or dying from IHD following an abnormal response to exercise. The rate declines from 136 per 1,000 during the first 5 years to 6 per 1,000 within 20 years in the 75 men who developed abnormal tests while under observation. The probability of surviving IHD over the same period in the absence of an abnormal stress test declines from 985 per 1,000 in the first 5 years to 820 per 1,000 by age 59. The 28 men with an abnormal electrocardiographic response to exercise on admission also fared badly. The cumulative probability of their not developing...
ECG STRESS TEST

**Figure 3**

Probability of not developing (that is, remaining free of) another manifestation of ischemic heart disease according to electrocardiographic response to exercise.

Another manifestation of IHD over 20 years was 4%.

Data for the total 103 men with abnormal tests have not been combined since the two groups are not comparable. The 28 men with an abnormal test at entry represent survivors of a cohort whose person years of exposure to risk of IHD after an abnormal electrocardiographic response to exercise is unknown since no data on experience prior to entry are available. Furthermore, the limited numbers of person years of experience at the 5-year intervals after age 40 are so few as to make the probabilities relatively unstable. For similar reasons persons with myocardial infarction and angina pectoris at entry were excluded from the present analysis.

An attempt has been made to evaluate the relationship of the degree of abnormality to prognosis. Unfortunately, subdivision of the data results in very small frequencies in each abnormality group. Disease rates based on these are subject to large standard errors. Therefore, it is very difficult to draw reliable conclusions.

Tables 1 and 2 show the 28 individuals with an initial abnormal electrocardiographic response to exercise and the 75 with subsequent abnormal responses according to the resting electrocardiogram and the degree of abnormality of the postexercise tracing.

In table 3 are considered selected risk factors in relation to the electrocardiographic response to exercise. The mean levels and the standard deviations for serum total cholesterol, systolic and diastolic blood pressures, relative body weight, and the proportion of cigarette smokers in the two groups are displayed. The mean levels for the persons with abnormal ECG-stress tests are higher and more variable than for those of the controls. These differences are significant only for systolic and diastolic blood pressures and smoking. This finding is in accord with other data which show higher levels of these factors in diagnosed cases of IHD as compared with individuals free of IHD. There were too few cases to detect any influence on prognosis.

**Table 1**

Prognosis of Development of Additional Evidence of Ischemic Heart Disease According to Severity of Response after Entry: Abnormal Electrocardiographic Stress Test

<table>
<thead>
<tr>
<th>Degree of abnormality*</th>
<th>Abnormal ECG test after entry</th>
<th>Normal resting ECG</th>
<th>Abnormal resting ECG</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>Prognosis†</td>
<td>Total</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Total</td>
<td>75</td>
<td>34</td>
<td>45</td>
</tr>
<tr>
<td>Grade 1a</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 1a</td>
<td>40</td>
<td>15</td>
<td>37</td>
</tr>
<tr>
<td>Grade 1a</td>
<td>2</td>
<td>2</td>
<td>100</td>
</tr>
<tr>
<td>Grade 2</td>
<td>17</td>
<td>9</td>
<td>53</td>
</tr>
<tr>
<td>Grade 3</td>
<td>13</td>
<td>6</td>
<td>46</td>
</tr>
<tr>
<td>Left bundle</td>
<td>3</td>
<td>2</td>
<td>67</td>
</tr>
</tbody>
</table>

* See reference 8.
† Based on number of persons in whom additional evidence of IHD developed during study period.
resulting from a conjunction between one or more of the risk factors considered and the severity of the induced electrocardiographic abnormality rated by Robb's criteria. Nevertheless, it is reasonable to suppose that a compounding of risk factors adversely influences prognosis. The effect of increasing age was likewise obscured by the small number of cases and the multiple confounding effects of the several risk factors. There is no reason to suppose, however, that the influence of rising age, as displayed in figure 4, in increasing the likelihood of an abnormal electrocardiographic response to exercise differs in any respect from its influence on other risk factors. The longer and the more intense the exposure to risk of the susceptible subject, the greater is the likelihood of disease.

**Discussion**

Nearly one third of the diagnoses of IHD made in the Cardiovascular Health Center have rested on what has been construed as an abnormal electrocardiographic response to walking on a motor-driven treadmill at 3 mph against a 5% grade for 10 min. The very high subsequent incidence of other manifestations of IHD in men with abnormal postexercise electrocardiograms, namely, angina pectoris, myocardial infarction, and sudden death, is

![Figure 4](image-url)

**Figure 4**

Incidence of abnormal electrocardiographic response to exercise according to age; rate per 1000 person-years exposure.

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**Table 2**

**Prognosis According to Severity of Response at Entry: Abnormal Electrocardiographic Stress Test at Entry**

<table>
<thead>
<tr>
<th>Degree of abnormality *</th>
<th>Total</th>
<th>Normal resting ECG</th>
<th>Abnormal resting ECG</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Prognosis †</td>
<td>Prognosis †</td>
<td>Prognosis †</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>Prognosis</td>
<td>Total</td>
</tr>
<tr>
<td>Total</td>
<td>28</td>
<td>17</td>
<td>61</td>
</tr>
<tr>
<td>Grade 1a</td>
<td>8</td>
<td>4</td>
<td>50</td>
</tr>
<tr>
<td>1b</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>15</td>
<td>9</td>
<td>60</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
<td>4</td>
<td>100</td>
</tr>
<tr>
<td>Other</td>
<td>1</td>
<td>—</td>
<td>0</td>
</tr>
</tbody>
</table>

* See reference 8.
† Based on number of persons in whom additional evidence of IHS developed during study period.

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**Table 3**

**Level of Selected Risk Factors in Men with Abnormal Electrocardiographic Responses to Exercise Compared to Age-Matched Men with Normal Responses**

<table>
<thead>
<tr>
<th>Variable</th>
<th>ECG response to exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Abnormal (Mean ± SD)</td>
</tr>
<tr>
<td>Serum total cholesterol (mg/100 ml)</td>
<td>250 ± 43</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>150 ± 30</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>92 ± 14</td>
</tr>
<tr>
<td>Obesity (% ideal)</td>
<td>119 ± 15</td>
</tr>
<tr>
<td>Cigarette smokers (%)</td>
<td>69</td>
</tr>
</tbody>
</table>
felt to confirm the validity of induced ischemic flattening of the S-T segment as evidence of acute coronary insufficiency. Ischemic T-wave changes and paroxysmal left bundle-branch block were not useful criteria. Abnormal responders weigh more, have higher blood pressures, have higher serum total cholesterol concentrations, and are heavier cigarette smokers than men who have a normal electrocardiographic response to exercise. The prognosis for the 28 men who had an abnormal postexercise electrocardiogram on entry into the study has been similarly unfavorable.

Brody’s experience with the Master double two-step test at the Greenbrier Clinic is most nearly comparable with the present report. He tested 756 men, of whom 23 had electrocardiographic responses considered abnormal by criteria very similar to those used in this study. Seventy per cent of these men developed other manifestations of IHD in the ensuing 3 to 10 years (six had myocardial infarction and 10 “classical” angina pectoris). There is no other study with which the present observations can be cogently compared. The military population followed by Mattingly was originally stress tested because of complaints of chest pain. The insurance applicants reported by Robb and Marks were exercised in many instances because of complaints of chest discomfort.

In our opinion the observations presented justify the acceptance of an abnormal electrocardiographic response to exercise as an additional category to the nosology of IHD. The intensity of the exercise test is probably more important than the technic. Treadmills are valuable research instruments, particularly in applying work loads of progressive severity, but are not likely to be used outside the laboratory. On the other hand, the widely known and accepted two-step of Master can easily, effectively, and safely be used in any office. Even simpler is the single 12-inch step test proposed by Simonson, which could easily be utilized in field studies. The one-step test, furthermore, is readily adaptable to continuous direct-wire recording of the electrocardiogram; it is not only a cheaper but is also a more reliable technic than telemetry.

The great likelihood of developing angina pectoris, of sustaining a myocardial infarction, or of dying suddenly not long after an abnormal electrocardiographic response to exercise is evinced is clearly indicative of far-advanced atherosclerosis of the coronary arteries. Close questioning of these abnormal responders, however, has consistently failed to elicit symptoms that could in retrospect be construed as angina pectoris. The more severe transient electrocardiographic deformities induced in hypertensive men with the electrocardiographic pattern of left ventricular enlargement or with nonspecific T-wave abnormalities can reasonably be construed as evidence of advanced coronary arterial insufficiency, due either to arterial stenosis, myocardial hypertrophy or, most probably, to both factors. This interpretation is supported by the subsequent high incidence of clinically overt IHD. The lower rate of severe exercise-induced abnormalities in individuals with normal resting electrocardiograms is consistent with the view that coronary irrigation was less severely compromised in these subjects and is supported, although scarcely proved, by the apparently lower subsequent incidence of overt IHD. A priori, it seems unlikely that the abnormal reactors have coronary vasculature much different from that of men who presented initially with angina pectoris and who, incidentally, did not necessarily respond to exercise with an acutely abnormal electrocardiogram. It is possible although unprovable that the abnormal reactors had already unconsciously restricted their activity short of that which might precipitate angina. The high incidence of silent myocardial infarction, attested only by the symptomless progression of the electrocardiogram from normal to a pattern typical of intercurrent myocardial infarction, is a clear instance of the not uncommon dissociation between the subjective and the objective manifestations of IHD.
The high rate of other manifestations of IHD experienced by men with an abnormal electrocardiographic response to exercise suggests that this group might be particularly useful for testing regimens designed to lessen this risk. The elimination of cigarette smoking, the reduction of arterial blood pressure, and a program of graded exercise, for example, might be evaluated separately or concurrently by serial electrocardiographic stress tests. A significant reduction in the incidence of angina pectoris, of myocardial infarction, and of sudden death could fairly be construed as evidence for the effectiveness of these interventions.

Thus, of the 2,003 men who had at least two ECG-stress tests through December 1966, 264 men developed some manifestation of IHD and 75 (30%) of them had an abnormal electrocardiographic response to exercise as their first manifestation of IHD. Over the next 5 years there was an 85% probability that these 75 men would develop another manifestation of IHD, namely, angina pectoris, myocardial infarction, or sudden death. Men who on admission to the study had an abnormal electrocardiographic stress test also had a high subsequent incidence of IHD. Only transient ischemic flattening or coving of the S-T segment induced by exercise proved to be a diagnostically useful index of potentially inadequate coronary arterial perfusion. These men also had higher blood pressures and were more often heavier smokers than men who consistently had normal electrocardiographic responses to exercise. Relative body weight and serum total cholesterol concentration were not significantly different in the two groups. Increasing age was associated with an increasing likelihood of an abnormal electrocardiographic response to exercise.

Conclusions

It is concluded that the electrocardiographic stress test used in this study reliably identifies men who are at an exceptionally high risk of overt IHD but whose coronary atherosclerosis is still clinically silent. The test employed in this study is safe but has a low yield and is, probably, too expensive for widespread field surveys. A more strenuous exercise test would be diagnostically more productive but also more hazardous and, possibly, less acceptable. Simple step tests should be considered for clinical and epidemiologic studies. The electrocardiographic criteria used in this study are rigid, relatively insensitive but highly specific, as attested by the bad prognosis of an abnormal test. It is suggested that an abnormal electrocardiographic response to exercise should be considered a valid addition to the nosology of IHD. Individuals in this category may be particularly appropriate subjects for testing the effectiveness of risk factor intervention programs.

Acknowledgment

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