In their discussion, the authors state that exercise duration of 3 minutes or less was the only significant predictor of subsequent CHD in women. However, the reader is not told how many potentially predictive variables were considered (and tested) by the authors. They describe access to SPSS and UCLA Biomedical Computer Programs, including crosstabs and contingency table analyses; these provide the capability of testing associations between a large number of variables. The authors state that they processed a maximum of 20 information bits for each of the original 1077 subjects. They do not describe how many variables were tested, nor how the variables were chosen (e.g., why exercise duration was dichotomized at 3 minutes for women and at 5 minutes for men).

We are not told if age of the women was predictive. Unless all 311 women were the same age, we would expect that older women would have a higher probability of developing CHD. We might also expect that older women would have a shorter exercise duration, the variable found "most predictive" by the authors. The covariance of age and exercise duration was not specified.

Another potential predictor for CHD incidence is the reason for exercise cessation, or end point. The relative frequencies of various exercise end points in women were not given. For example, "marked dyspnea" after only 3 minutes of the Long Beach protocol might be predictive of subsequent CHD.

Finally, we were not told how many asymptomatic persons tested during the 10-month study developed arrhythmias, conduction defects or symptoms suggesting angina pectoris, and thus were excluded from the present study. The exercise results in these persons could be analyzed together with data from the 1077 persons reported in the present study to give a clearer indication of the predictive value of testing asymptomatic persons.

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References


The authors reply:

To the Editor:

The mean age of the 300 women at study entry who did not develop CHD within 5 years was 44.6 ± 12.3 years, compared with 54.1 ± 7.9 years for the 11 women at study entry who developed CHD within 5 years (p < 0.01). Of the 11 women who developed CHD, one developed myocardial infarction and 10 developed classic angina pectoris.

The 20 information bits processed for the subjects were age, sex, ST pattern, amount of ST-segment depression, 10 variables obtained from questioning the subjects or their physicians to determine whether the subjects had developed classic angina pectoris, myocardial infarction, or sudden cardiac death, exercise duration, and five variables dealing with the R wave. The duration of treadmill exercise using the Ellestad protocol that discriminated between the presence or absence of CHD within 5 years was 3 minutes or less in women and 5 minutes or less in men.

The coefficient of correlation between age and duration of exercise in our asymptomatic women was −0.36. Age did not predict the presence or absence of CHD in women within 5 years as well as did an exercise duration of 3 minutes or less.

The asymptomatic women exercised until they reached 100% of their predicted maximal heart rate, overwhelming fatigue, or marked dyspnea with a plateau of the heart rate. None of these end points discriminated between the presence or absence of CHD within 5 years.

We do not have follow-up data on the asymptomatic persons we excluded from the study because they had major ST-segment changes at rest, or developed a marked arrhythmia, a conduction abnormality, chest pain or any symptoms suggestive of angina pectoris during or after the maximal treadmill stress test.

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Reproducibility of Supine Exercise Testing

To the Editor:

Thadani et al. give an excellent discussion of poor reproducibility of supine exercise testing in individual patients. Upton et al. found similar results in normals with repeated exercise several days apart. The problem goes deeper; this technique does not reproduce physiology and habits of normal living and may give misleading results. We present a case in point.

A 51-year-old, very active housewife, who regularly played five or six sets of singles and doubles of tennis a day, was admitted with an acute anterolateral myocardial infarction, documented by ECG changes and enzyme elevations. She had been in apparently excellent health.

Her hospital course was complicated by chest pain, which was controlled on propranolol, 320 mg/day in divided doses. She was intolerant of nitrates. After discharge, on this dose of propranolol, she suffered chest pain two or three times per week while making beds or ironing. Resting blood pressure and pulse were 90/60 mm Hg and 58 respectively. An exercise treadmill test done 6 weeks after infarction on propranolol demonstrated severe chest pain that radiated to the left arm and pseudonormalization of the right precordial T waves after 7 minutes (stage III) of the Bruce protocol, at a heart rate of 97 beats/min. All symptoms and signs resolved during recovery.

A supine bicycle exercise radionuclide angiogram was done. After 3½ minutes at 200 kpm the test was stopped for leg weakness and shortness of breath. She had neither chest pain nor ECG changes. Baseline, peak exercise and recovery ejection fractions were 76%, 86% and 78%, respectively (average of two independent observers). She had anterolateral hypokinesia at rest.

Because the patient's lifestyle had been unacceptably altered by her symptoms and she was refractory to medical therapy, cardiac catheterization and aortocoronary bypass were performed. Angiography and coronary arteriography confirmed the mild anterolateral hypokinesia and showed 100% occlusion of the left anterior descending artery at the 1st septal branch, with distal collateral filling from the right and circumflex arteries. There was also a 60% occlusion of the right coronary artery in its midportion. She received saphenous vein grafts to the right coronary artery and left anterior descending artery. The ECGs were unchanged postoperatively.

Thirteen weeks after surgery, she was completely asymptomatic in her activities of daily living and household chores. Her only medication was propranolol, 10 mg four times daily for a perioperative arrhythmia. On a treadmill, she went to 11 minutes (stage IV) of the Bruce protocol and to a heart rate of 170 beats/min. The test was stopped for leg weakness. Mild chest discomfort was noted 4 minutes into the test, but it resolved spontaneously at 9 minutes. She returned to her previous lifestyle, except that she now confines her singles tennis to inferior opponents.

A postoperative supine bicycle exercise radionuclide angiogram was done at 14 weeks. The patient went only 3 minutes at 200 kpm.
Reproducibility of supine exercise testing.
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