Exercise Stress Testing

David Richard, MD, and Richard Birrer, MD, MPH
Danville, Pennsylvania

Exercise stress testing is used widely to evaluate the presence and extent of anatomic disease and the prognosis when significant disease has been defined. Indications for diagnostic testing include elevated blood pressure, palpitations, and chest pain. Functional testing is used to assess progression of cardiac disease, cardiac status following therapy, and physical capacity. Exercise testing must be applied to an appropriately selected group of patients. The indications, contraindications, types, results, and complications of exercise testing are presented.

Exercise stress testing has been utilized in the definition of patients with coronary artery disease since the late 1920s. Its usefulness in recent years has been expanded to include radionuclide scanning, when stress testing is equivocal, and post-infarction testing to define individuals at risk for sudden death or recurrent infarction. By combining the patient's subjective complaints with a battery of objective findings (heart rate, blood pressure, electrocardiogram [ECG] changes and test duration), the prognostic value of the exercise stress test has been increased tremendously in recent years.

INDICATIONS AND CONTRAINDICATIONS

The use of exercise stress testing (EST) in helping to diagnose patients with chest pain has been well established. It has been shown that an EST is quite likely to be positive in those patients with a subjectively convincing story for coronary artery disease, whereas in those with physician-described "noncardiac chest pain," the test is usually negative.[1] EST is especially useful to clarify the cause of pain in patients with "atypical pain." Exercise stress testing is also of use in evaluating the severity of disease in those patients with known coronary artery disease, assessing the status of the patient's current medical or surgical therapy, determining rehabilitation guidelines following myocardial infarction, and clearing patients for exercise programs.[2] Of less certainty is whether the test is beneficial in evaluating dysrhythmias, testing asymptomatic individuals (particularly in light of the recent emphasis on "silent myocardial ischemia"), and profiling so-called high-risk individuals such as airline pilots.

Certain contraindications (Table 1) must be weighed carefully prior to doing exercise stress testing on an individual. The risk of sudden death and morbidity from EST is greatly enhanced by failing to recognize these contraindications. Generally, a submaximal EST is performed in patients with these contraindications. Such an approach maximizes information concerning functional capacity, therapy, risk, and the need for further diagnostic testing while at the same time minimizing morbidity.

Submitted, revised, December 24, 1987.

From the Department of Family Practice, Geisinger Medical Center, Danville, Pennsylvania. Requests for reprints should be addressed to Dr. Richard B. Birrer, Department of Family Medicine (14-00), Geisinger Medical Center, Danville, PA 17822.

PATHOPHYSIOLOGY

The clinical value of exercise stress testing is the determination of the patient's coronary artery reserve. An inability to raise heart rate and blood pressure sufficiently during exercise is an excellent predictor of severe coronary artery disease. Ischemic changes found during EST are highly predictive of finding moderate to severe coronary artery disease on catheterization. Experimentation on dogs reveals that an 80 percent occlusion is needed before consistent evidence of coronary artery disease is demonstrated on EST.[3] The consequences of insufficient myocardial oxygenation include chest pain, electrocardiographic (ECG) changes (either ST segment elevation or depression), dysrhythmias, and, in severe cases of coronary artery disease, evidence of left ventricular pump failure (ie, fall in blood pressure during exertion).

DEFINITION OF THE TEST

Exercise stress testing requires the patient to be able to participate functionally in the procedure. The intensity

TABLE 1. CONTRAINDICATIONS TO MAXIMUM EXERCISE STRESS TESTING

Absolute
- Unstable angina pectoris
- Heart block (2nd or 3rd degree)
- Pericarditis
- Myocarditis
- Uncontrolled asthma
- Uncontrolled hypertension (>250 mmHg systolic; >120 mmHg diastolic)
- Uncontrolled or new-onset congestive heart failure
- Uncontrolled or new-onset arrhythmia
- Any new electrocardiogram changes
- Acute thrombophlebitis or embolism
- Acute febrile illness
- Respiratory failure
- Cerebrovascular disease
- Resting tachycardia (>120 beats per minute)
- Resting electrocardiogram abnormalities
- Poorly controlled diabetes
- Epilepsy

Relative
- Severe electrolyte disturbance
- Recent myocardial infarction < 6 wk
- Aortic valve disease

of exercise a patient performs is predicted from workload and is referred to in MET units (myocardial oxygen requirement at rest = 1 MET). Occupational and recreational activities in METs that can be quite useful in rehabilitative guidelines for a particular individual are displayed in the Appendix. Diagnostic yield is maximized if a patient is able to perform to test specifications (ie, closer to maximal heart rate, ECG changes, blood pressure changes, angina, or dysrhythmias). Tests that are limited by nonspecific patient complaints are of far less diagnostic value.

Exercise stress testing protocols have been developed in an attempt to elicit the most information concerning peak myocardial oxygen requirements (Table 2). These protocols, however, are plagued by a lack of standardization. Ideally the test should last from six to 15 minutes to decrease the likelihood of fatigue factors present in longer tests and to identify ischemic changes seen in tests of shorter duration.

Patient preparation is of prime importance in attaining meaningful results. The history should include current medications, as both digoxin and β-blockers can have profound interpretive implications in exercise stress testing. A 12-lead resting ECG should be obtained and the findings compared with previous tracings, if available. Most important, if the patient is prepared to anticipate fatigue and shortness of breath as normal during the procedure, the number of uninterpretable tests due to patient symptom limitations will be decreased. A post-exercise physical examination should search for any new cardiac findings (murmurs, gallops, dysrhythmias, etc).

In a maximum EST the individual attains 80 to 90 percent of his or her predicted maximum heart rate or rating of perceived exertion. The maximum heart rate is most commonly calculated from the equations (220 − age) or 210 − age (0.65) ± 10 beats per minute. Any test that does not attain the 80 to 90 percent maximum heart rate or rate of perceived exertion may be limited clinically. These criteria are modified downward in a submaximal EST.

EXERCISE FINDINGS

Electrocardiographic changes seen with exercise usually involve ST segment depression resulting from inadequate subendocardial perfusion. A positive result is understood generally as representing a 1-mm or greater ST depression at 0.08 seconds after the QRS complex (80 msec past the J point). An upsloping ST segment at the J point is gen-

TABLE 2. TYPES OF EXERCISE STRESS TEST PROTOCOLS

Test	Duration	Treadmill	Ergometer	MVO_2 Accuracy	Comments
Kasch Pulse Recovery[4]	3 min	No	No	+	Short, simple, in-office
Master 2 Step[5]	2–3 min	No	No	+	Short, simple, in-office
Bruce[5]	12 min	Yes	No	+++	Large incremental increases; difficult for disabled
Balke[6]	10 min	Yes	No	++++	Smaller graded increments; good for disabled
Naughton[6]	12 min	Yes	No	++++	Smaller graded increments; good for disabled
Scandinavian[6]	16–24 min	No	Yes	++++	Good for disabled
12-minute test[6]	12 min	No	No	+++	Very gradual protocol; good for very disabled
Triangular[6]	<20 min	No	Yes	++++	Good for very disabled; can be long
Wingate[6]	30–45 sec	No	Yes	++	Good for very disabled

MVO_2—minute oxygen consumption (myocardial)
+ = poor, ++++ = excellent

erally a normal finding (negative result), whereas if the segment is downsloping or horizontal, a positive test (ischemia) is generally understood. The degree of ST depression is proportional to the severity of ischemia in that area of myocardium; however, the duration of ST segment depression following exercise is related to the degree of obstruction in a particular vessel or vessels. Several false-positive results can occur with ST segment depression, including hypokalemia, medications (digoxin, psychotropic agents, phenothiazine tranquilizers, and lithium preparations), conduction abnormalities (left bundle branch block, Wolff-Parkinson-White), moderate to severe valvular lesions, and mitral valve prolapse.[7,8] Additionally, β-blockers, calcium channel blockers, and nitrates have a mechanism of action that automatically delays an ischemic response. False-negative ECG changes can occur with a suboptimal test (less than 80 to 90 percent of predicted heart rate), single-vessel disease, or other disease resulting in a suboptimal test (ie, pulmonary and orthopedic diseases).

ST segment elevation may also occur during exercise. Those with Prinzmetal's angina have a 10 to 20 percent rate of ST elevation with exertion.[7,9] Of a more significant nature are those with transmural ischemia, which causes ST elevation. The latter represents a critical stenosis, and immediate invasive studies may be necessary.

Mean blood pressure response with exercise should typically show an 8- to 10-mmHg increase with each stage increase in either the Bruce or Naughton protocol. Plateauing or decreasing mean blood pressure with increase in workload is an ominous sign representing left ventricular dysfunction—an inability of the heart to respond appropriately to increased myocardial demand.

Heart rate response should also increase as a direct result of increasing myocardial demand to produce an increased cardiac output. Exercise stress testing has decreased diagnostic value if the patient is unable to increase his heart rate to within 20 beats of his 90 percent predicted maximal heart rate; additionally, failure to do so represents a poor prognostic indicator if the patient is unable to appropriately increase myocardial oxygenation in the face of increased demand. Patients who are taking β-blockers or have sinoatrial nodal disease cannot be assessed accurately with respect to heart rate.

Ectopy during EST is a nondiagnostic finding when independent of other factors. When ectopy, particularly ventricular, is noted in concert with other findings (angina, blood pressure, heart rate, or ECG changes), however, it may well represent corresponding myocardial ischemia.

The patient's subjective complaints may include chest pain, fatigue, claudication, shortness of breath, lightheadedness, or confusion (secondary to decreased carotid perfusion). The most diagnostic of these complaints is typical angina pectoris in association with ECG or objective findings indicative of coronary artery disease. Unfortunately, the presence of exertional chest pain as the sole entity does not help one predict coronary artery disease without a combination of other findings. In Table 3 is a summary of the reasons to terminate exercise stress testing.

TABLE 3. REASONS TO TERMINATE EXERCISE STRESS TESTING
ST depression > 4 mm, T wave inversion, Q wave appearance
ST elevation (any)
Ventricular fibrillation
New atrial fibrillation
Sustained ventricular tachycardia
Ventricular premature beats > 25% of all beats
Heart block (2nd or 3rd degree), new bundle branch block
Blood pressure decrease > 20 mmHg after normal exercise rise
Marked angina
Severe dyspnea
Marked claudication
Syncope or near syncope
Ataxia
Confusion
Neurologic signs
Dizziness
Any fall in systolic pressure below resting value
Systolic pressure in excess of 200 mmHg or diastolic pressure in excess of 140 mmHg
Severe chest pain
Cyanosis
Sudden-onset pallor, diaphoresis

PROGNOSTIC IMPLICATIONS OF TEST RESULTS

Overall, the exercise stress test has a 55 to 60 percent sensitivity and an 80 to 85 percent specificity in patients with known coronary artery disease.[1] Its predictive value or accuracy as defined by Baye's rule varies from 10 to 85 percent depending on such variables as the amount of ST depression, age and sex of the patient, symptomatology, risk-factor analysis, and any factors associated with the prevalence of coronary artery disease.[10,11] Physician history taking alone provides invaluable data in predicting test outcomes. For example, the predictive value of a negative EST in a patient with chest pain is in the range of 60 to 65 percent.[1] Patients with "typical" exertional angina pectoris have an 88 percent likelihood of angiographic coronary artery disease (range 80 to 96 percent); in contrast, those with chest pain believed unlikely to be due to coronary ischemia have an 11 percent likelihood (range

4 to 16 percent).[1] Therefore, the likelihood of EST revealing evidence of myocardial ischemia can often be predicted from the history alone.

Further, Bruce[5] has shown that predicting coronary artery disease can be done solely on conventional risk factors (cholesterol > 6.35 mmol/L [245 mg/dL], blood pressure greater than 140 mmHg systolic or 90 mmHg diastolic, family history of a parent or sibling dying of myocardial infarction before the age of 70 years, or smoking cigarettes), as those without risk factors have a 99 percent survival at five years regardless of their exercise findings. Those with one conventional risk factor plus two exercise factors (duration less than 6 min, chest pain with exertion, failure to attain 90 percent of predicted maximal heart rate, or ST depression greater than 1 mm) dip to a 67 percent five-year coronary survival or a 33-fold increase in mortality! Those with only one exercise risk factor and one conventional risk factor have a 98.4 percent five-year survival.

Several investigators also feel that actual test duration, as an independent factor, can be highly correlated with long-term cardiac morbidity and mortality.[1,5,7,8,10,12–14] A less than 1 percent annual mortality secondary to coronary artery disease has been found in those individuals able to complete Bruce stage III exercise and attain a heart rate of 160 beats per minute.[12] McNeer et al[13] found that an inability to complete stage I of the Bruce protocol corresponded to a 59 percent two-year survival and that 55 percent of all patients in this particular subgroup had significant triple-vessel or left main coronary artery disease. Further, in these individuals, all of whom had angiographic evidence of coronary artery disease, the ability to go to Bruce stage IV revealed a 90 percent four-year survival.[13]

Electrocardiographic evidence of ST segment depression alone can hold strong correlations for later cardiac morbidity. Ellestad and Halliday,[15] who tested more than 6,000 individuals, showed that greater than 1.5-mm depression at 0.08 seconds following the QRS complex correlated with a 46 percent incidence of increased angina, myocardial infarction, or death at four years. Their eight-year follow-up revealed a 76 percent incidence of the same cardiac manifestations, approximately 10 percent per year (3.5 percent per year of myocardial infarction alone). Conversely, a negative test corresponded with a 7 percent four-year cardiac morbidity and less than 1 percent per year of myocardial infarction.

McNeer et al[13] found in subsequent cardiac catheterization that of those who had positive findings on EST at Bruce protocol stages I or II, there was a 60 percent incidence of triple-vessel disease as well as a 25 percent incidence of left main descending coronary artery disease; in comparison, only 15 percent of those individuals who went to stage IV had evidence of triple-vessel coronary artery disease. Further, the risk profile of these groups was less than 85 percent one-year survival in the former group and 99 percent in the latter. Four-year follow-up showed even more dramatic results, as those with positive results at stage I had a 98 percent incidence of coronary artery disease (73 percent triple-vessel coronary artery disease and 27 percent left main descending artery disease), while those with negative EST results who could not complete stage I still had a 52 percent incidence of coronary artery disease (21 percent triple-vessel and 10 percent left main descending coronary artery disease). Further, those who had a negative test at stage IV had a 36 percent incidence of coronary artery disease (9 percent triple-vessel coronary artery disease and 1 percent greater than 50 percent left main descending artery disease).[13] Likewise, Bartel et al[16] found that all patients with greater than 2.5-mm ST depression had abnormal coronary arteries on angiography; 90 percent had multivessel disease. Sixty percent of the cohort with negative findings on exercise stress testing, however, had normal coronary arteries. Few patients with negative or mildly positive tests had multivessel disease.[16]

Specific ECG findings are associated with increased cardiac morbidity. ST segment elevation has been associated not only with Prinzmetal's angina and ventricular aneurysm, but also with transmural ischemia.[7,8,10,12,16] When associated with the latter, severe proximal lesions were found in many individuals who were catheterized.[8,17] Further, there may be increased cardiac morbidity and mortality with T wave inversion with exercise as well as with a failure to decrease the size of R waves with exercise (believed to be a sign of left ventricular dysfunction when compared with typical decreased heart size with activity).[10]

A good correlation also exists between the length of time it takes for the post-exercise ECG to normalize and subsequent cardiac catheterization findings. Goldschlager et al[18] found that 40 percent of those whose ECG normalized within one minute after exercise had normal coronary arteries, and 90 percent of those whose abnormalities persisted at greater than nine minutes had multivessel disease.

The inability to raise systolic blood pressure above 140 mmHg is associated with 15-fold increase in sudden cardiac death compared with those with an exercise blood pressure of greater than 200 mmHg and a fourfold increase in those whose peak systolic blood pressure was 140 to 199 mmHg.[19] Seventy percent of those with a maximal systolic blood pressure of less than 140 mmHg have a greater than 70 percent incidence of coronary artery stenosis and 38 percent have a left ventricular ejection fraction of less than 40 percent.[19] There is also a correlation between exertional hypotension in medically treated individuals in five-year survival as well as the number of vessels diseased. Similarly, a lack of fall of the diagnostic blood pressure during exercise has great predictive value for poor left ventricular function secondary to coronary artery disease.[14]

The prognostic value of exercise stress testing can also be enhanced on the basis of pulse rate response with exercise. Using EST as a single variable McNeer et al[13] showed that those patients who had coronary artery disease had a 90 percent four-year survival rate with a heart rate of greater than 160 beats per minute compared with an 80 percent four-year survival for those whose peak pulse rate was 120 to 159 beats per minute and a less than 60 percent four-year survival if the participant was unable to achieve a heart rate of 120 beats per minute. Additionally, Blincoe and Lutz[14] demonstrated an increased incidence of myocardial infarction, coronary artery bypass graft, or sudden cardiac death in those patients unable to reach 90 percent of their expected maximal heart rate. Furthermore, their data increased dramatically with the addition of other EST or conventional risk factors.[8]

The predictive value of ventricular ectopy is considered tenuous when it occurs during exercise. Several workers have found no correlation of exercise ectopy when associated with normal exercise ECG results.[7,8,10,20] Califf et al,[21] however, showed that association of ventricular arrhythmias with known coronary artery disease (greater than 70 percent occlusion of major vessel) decreased patient survival. While those without exercise ectopy had a 90 percent three-year survival rate, the rate decreased to 83 percent in those with single premature ventricular contractions and 75 percent in those with paired premature ventricular contractions or ventricular tachycardia.[21]

Several investigations have shown increased survival in those patients with known coronary artery disease who are able to achieve longer EST durations, maximal heart rate (age-predicted), and fewer ST segment changes.[5,7,8,10,12-15,17,20] McNeer et al[13] showed an increase in survival from 77 to 90 percent in those with angiographic coronary artery disease at four years in these patients. Gohlke and associates[22] found a 96 percent five-year survival in those patients with two-vessel coronary artery disease and normal left ventricular function and 81 percent five-year survival in those unable to reach a maximal heart rate because of angina or fatigue. Berman and colleagues[23] showed that the predictive value of EST increased from 78 to 97 percent when ischemic ST segments were combined with two of the following variables: a double product less than 23,000, exercise duration less than six minutes, or ST segments remaining depressed longer than three minutes after completing exercise.

Bruce[5] has also looked at predicting secondary coronary events in those with coronary artery disease. Left ventricular dysfunction as indicated by an exercise duration less than three minutes, cardiomegaly, or maximum systolic blood pressure less than 130 mmHG is far more predictive than ST depression and angina. In those subjects without exertional ischemia, the annual mortality is approximately 2 percent vs 5 percent in those with ST depression. With one sign of left ventricular dysfunction, however, the annual mortality rises to 9.6 percent, with any two signs to 25 percent, and with all three signs to an astounding 88 percent.

A controversial subject is that of performing exercise stress testing on asymptomatic individuals. While some proponents exist, the majority of discussions point to an increased usefulness in those having risk factors—hypertension, age, cholesterol level, family history, and smoking history, or a worrisome personal history of angina. False-positive rates pertaining to EST alone may range from 64 to 75 percent in asymptomatic men and 70 to 85 percent in asymptomatic women in the general population, where the prevalence of coronary artery disease is low.[7,12,24,25] For those individuals with a true-positive test, however, the incidence of coronary artery disease with complications is 15-fold higher than those with negative test results (approximately 1 percent of all healthy individuals tested). Thirty-three percent of these patients will have angina, myocardial infarction, or death within the next five years.[7,26]

Abbott et al[27] reviewed the use of physician test ordering as well as the integration of the results into the patient's regimen. The authors determined that all 90 successive tests were ordered for appropriate reasons. They found that in those tests that showed depressed ST segments, physicians integrated this information into the patient's file. In those patients with negative results, however, further studies to determine the patient's problem, when it persisted, were not performed (including any further cardiac workup) by 33 percent of physicians, and some even continued antianginal medications. Further, it was shown that the likelihood of resolving a diagnostic dilemma was decreased when the initial history was questionable for angina pectoris.

INCIDENCE OF COMPLICATIONS

Complications arising from EST are exceedingly rare when the above contraindications and signals to terminate the test are heeded. Mortality figures range between 3 and 10 per 100,000, while morbidity, defined as hospital admission secondary to test-related chest pain, dysrhythmia, or myocardial infarction is about 24 in 100,000 tests.[8,12] To prevent mortality or excessive morbidity, all exercise stress testing should be physician-monitored and accompanied with adequate defibrillating equipment and cardiac medications in anticipation of a cardiac arrest. Understandably, all persons associated with performing the test should be trained in cardiopulmonary resuscitation and advanced cardiac life support.

SUBMAXIMAL STRESS TESTS

A type of exercise stress test that is shown to have tremendous predictive value is the post-infarction submaximal stress test. Its main contribution is to identify those patients at high risk for recurrent myocardial infarction or death; if the test is positive, one can proceed with invasive cardiac studies quickly, if needed. When a negative result is attained, it allows the physician and the rehabilitative team to provide the patient and family with an accurate and safe exercise regimen while providing much-needed confidence in task performance for the patient.

The test is generally run using the Naughton protocol. Some controversy has developed as to whether the test should be done immediately after myocardial infarction (8 to 12 days) or six weeks after the infarction. Both show evidence of excellent predictive value, and little interim morbidity and mortality have been seen.[28–30]

The submaximal stress test is generally run to 5 METs, or the equivalent of climbing one flight of stairs—a very practical endeavor for most people. Contraindications to running the test immediately following an infarct include angina or congestive heart failure within the previous three days, uncontrolled hypertension, profound hypotension (systolic blood pressure less than 80 mmHg), unstable ECG, or complex ventricular ectopy.[8,28,29] Likewise, the test termination threshold is equivalent to that of a routine EST, except that the test should be stopped immediately with the onset of any anginal symptoms. Maximal heart rate is considered 70 to 75 percent of expected with an absolute ceiling of 130 beats per minute.

Interpretation of the test is also similar to that of a routine EST. For those patients with abnormal ST segments prior to undergoing the submaximal stress test, an additional 2-mm ST depression is considered diagnostic of continued post-infarction myocardial ischemia.[29] Again, blood pressure and pulse response with exercise and test duration remain critical criteria in judging the patient's ultimate prognosis.

Prognostically, those patients able to perform a submaximal stress test are grouped into high and low risk. Those at high risk are individuals who are unable to exercise to more than 3 METs or who develop exercise hypotension, exercise angina, or ST depression of greater than 2 mm from the baseline ECG.[28,29] Those individuals who are unable to attain 3 METs of exercise or who develop angina (39 percent of the post-infarction group) carry a 57 percent one-year incidence of sudden cardiac death, unstable angina, or myocardial infarction.[29,30] Hence, in these individuals further evaluation at an early stage is warranted, most notably cardiac catheterization, perhaps even prior to hospital discharge. For those who show exercise blood pressure reduction, a radionuclide test after healing may be considered to determine whether any ischemic areas still exist.

In those patients who do not fit any high-risk criteria, submaximal exercise testing serves as an important baseline in determining an appropriate and safe exercise prescription as well as the beginning of a long-term rehabilitation program. It also serves to assure the physician of the adequacy of medical treatment and obviates any need for adjustment.

OTHER STRESS TESTING METHODS

Radioisotope studies have enhanced greatly the sensitivity of exercise stress testing. Although adding significantly to the cost of the study, the use of thallium radioisotope (Th 201), especially in those patients at high risk for a false-positive routine exercise study, has proven beneficial as a diagnostic tool for coronary artery disease. Specifically, radionuclide stress testing is optimal when utilized in patients with a pretest probability of disease based on age and clinical findings between 25 and 70 percent (ie, less than 85 percent maximum heart rate achieved during EST in a patient with chest pain or a patient with certain risk factors, atypical chest pain, or asymptomatic ST segment depression during exercise).[31,32]

The kinetics of the thallium isotope allow uptake to be proportional to regional perfusion. The isotope is administered intravenously at peak exercise, and uptake is measured within 10 minutes of this activity as well as three hours after activity. Any areas of fixed decrease in radioactivity represent scarring from previous infarction, whereas those areas of decreased immediate radioactivity that are adequately perfused at three hours (reversible defects) represent areas of exercise-induced myocardial ischemia.

The clinical indications for performing thallium studies in preference to a routine exercise stress test are listed in Table 4. The thallium stress test has proven to be advantageous in predicting coronary artery disease. In a compilation of studies Gibson and Beller[1] report a sensitivity of 83 percent and a specificity of 90 percent in patients with known coronary artery disease. Furthermore, thallium stress testing is very accurate in the prediction of single-vessel (80 percent) and double-vessel (83 percent) coronary artery disease as well as triple-vessel disease (96 percent). Overall, the test has a sensitivity of 85 percent and a specificity of 91 percent.[33] Because of the nature of the radioactivity, the test is useful in predicting the affected artery as well as the segment of myocardium with decreased perfusion (right coronary artery 80 percent, left

TABLE 4. POTENTIAL CAUSES OF FALSE-POSITIVE TEST RESULTS FROM EXERCISE STRESS TESTING

Women
Medications
Group I antiarrhythmic agents
Digitalis
Phenothiazines
Tricylic antidepressants
Lithium
Hypokalemia
Mitral valve prolapse
Resting electrocardiogram abnormalities
Suspected false-positive or false-negative exercise stress test results
Borderline lesions at catheterization

anterior descending 86 percent, and circumflex artery 60 percent). Multiple reversible defects tend to reflect greater than single-vessel disease, as one might expect.

A variant of the thallium stress test involves injecting intravenous dipyridamole in those individuals who are unable to exercise to a level near their predicted maximum heart rate. Dipyridamole dilates coronary arteries, which simulates the exercise state. The sensitivity, specificity, and predictive value of this test is similar to that of thallium stress testing.[34] The presence of coronary artery disease corresponds to reversible thallium defects, whereas areas of prior myocardial infarction generally are visible as a fixed defect.[29,34]

Additional stress test modalities include radionuclide cineangiography (MUGA, multi-gated acquisition study) and exercise echocardiograms. These approaches may increase the sensitivity and predictive value of EST.[35]

CONCLUSIONS

Exercise stress testing provides the physician, as well as the patient, with a noninvasive procedure for evaluating coronary artery disease. Compliance with protocol guidelines produces a very safe procedure, particularly in the office setting. Proper interpretation of EST provides a sensitive indicator of coronary artery disease, allows for an evaluation of appropriate medicinal treatment, or can alert the physician to a possible need for rapid invasive techniques. Recent advances in thallium studies have provided an additional means of testing those individuals who were previously at high risk for false-positive EST. Persantine-thallium studies allow the physician to study those patients who are unable to exercise because of other limitations. Post-infarction stress testing is also of great prognostic value to the physician in terms of patient survival or need for hurried intervention. Hence, EST should be considered as a useful tool in coronary artery disease diagnosis, treatment appraisal (surgical or medical), and post-infarction prognosis.

References

1. Gibson RS, Beller GA: Should exercise electrographic testing be replaced by radioisotope methods? In Rahimtoola SH (ed): Controversies in Coronary Artery Disease, Philadelphia, FA David, 1983, pp 1–31
2. Birrer R: Exercise prescription. Postgrad Med 1985; 77(1):219–230
3. Gould KL, Hamilton GW, Lipscomb K, et al: Method for assessing stress-induced regional malperfusion during coronary arteriography: Experimental validation and clinical application. Am J Cardiol 1974; 34:557–564
4. Kasch F: A comparison of the exercise tolerance of post-rheumatic and normal boys. J Assoc Phys Ment Rehabil 1961; 15:35–40
5. Bruce RA: Maximal exercise testing. Postgrad Med 1981; 70(6): 161–68
6. Jones NL, Campbell EJM: Clinical Exercise Testing. Philadelphia, WB Saunders, 1982, chapt 4, pp 77–88, 119
7. DeBusk RF: Techniques of exercise testing. In Hurst JW (ed): The Heart, ed 6. New York, McGraw-Hill, 1986, pp 1704–1716
8. Sheffield LT: Exercise stress testing. In Braunwald E (ed): Heart Disease, ed 2. Philadelphia, WB Saunders, 1984, pp 258–278
9. Diamond GA, Forrester JS: Analyses of probability as an aid in the clinical diagnosis of coronary artery disease. N Engl J Med 1979; 300:1350–1358
10. Nadimi M, Killam HAW: The role of exercise testing in the diagnosis of coronary artery disease: A review of its sensitivity, specificity and predictive accuracy. Cardiovasc Rev Rep 1985; 6:107–113
11. Laslett L: Management of the asymptomatic patient with an abnormal exercise EKG. JAMA 1984; 252:1744–1746
12. Stone P: Exercise testing in perspective. Primary Cardiol 1983; 9:111–138
13. McNeer J, Margolis TR, Lee KL, et al: The role of the exercise test in the evaluation of patients for ischemic heart disease. Circulation 1978; 57:64–70
14. Blincoe W, Lutz J: The prognostic value of exercise testing. Cardiovasc Rev Rep 1985; 6:347–364
15. Ellestad NH, Halliday WK: Stress testing in the prognosis and management of ischemic heart disease. Angiology 1977; 28:149–159
16. Bartel AG, Behar VS, Peter RH, et al: Graded exercise stress tests in angiographically documented coronary artery disease. Circulation 1974; 49:348–356
17. Fortuin NJ, Weiss JL: Exercise stress testing. Circulation 1977; 56:699–712
18. Goldschlager N, Selzer A, Cohn K: Treadmill stress tests as indicators of presence and severity of patients with coronary artery disease. Ann Intern Med 1976; 85:277–286
19. Irving JB, Bruce RA, DeRoue NA: Variation in and significance of systolic pressure during maximal exercise (treadmill) testing. Am J Cardiol 1977; 39:841–848
20. Froelicher V: Exercise and coronary heart disease. J Cardiac Rehabil 1981; 1:277–288
21. Califf RM, McKinnis CA, McNeer JF, et al: Prognostic value of ventricular arrhythmias associated with treadmill exercise testing in patients studied with cardiac catheterization for suspected ischemic heart disease. J Am Coll Cardiol 1983; 2:1060–1067
22. Gohlke H, Samen L, Betz P: Exercise testing provides additional prognostic information in angiographically defined subgroups of

patients with coronary artery disease. Circulation 1983; 68:979–985
23. Berman JL, Wynne J, Cohn PF: A multivariate approach for interpreting treadmill exercise tests in coronary artery disease. Circulation 1978; 58:505–512
24. Guidelines for exercise testing: A report of the Joint American College of Cardiology/American Heart Association Task Force on Assessment of Cardiovascular Procedures (Subcommittee on Exercise Testing). Circulation 1986; 74:653A–667A
25. American College of Sports Medicine: Guidelines for Exercise Testing and Prescription, ed 3. Philadelphia, Lea & Febiger, 1986
26. Bruce R, DeRouen T, Hossack KF: Value of maximal exercise tests in risk assessment of primary heart disease in healthy men: 5 years' experience of the Seattle Health Match Study. Am J Cardiol 1980; 46:371–378
27. Abbott JA, Cheitlin MD, Tedeschi BS: Graded treadmill stress testing. West J Med 1977; 126:173–178
28. Hamm LF, Stull GA, Crow RS: Exercise testing early after myocardial infarction: Historic perspective and current uses. Prog Cardiovasc Dis 1986; 28:463–76
29. Knight J, Laubach C: Low level exercise testing after myocardial infarction. Postgrad Med 1986; 79(2):123–127
30. Starling MR, Crawford MH, Kennedy VT, et al: Exercise testing early after myocardial infarction: Predictive value of subsequent unstable angina and death. AM J Cardiol 1980; 46:909–914
31. Beller GA, Gibson RS: Noninvasive testing in CAD. Prog Cardiovasc Dis 1987; 29:241–270
32. Iskandrian AS, Hakki AH, DePace NL, et al: Use of radionuclide ventriculography in the evaluation of pathophysiology during exercise in patients with heart disease. J Cardiac Rehabil 1983; 3:788–801
33. Movahed A: Radionuclides in cardiovascular diagnosis. Primary Cardiol 1987; 13(6):90–98
34. Leppo J, Boucher CA, Boucher CA, Okada RD, et al: Serial thallium-201 myocardial imaging after dipyridamole infusion: Diagnostic utility in selecting coronary stenoses and relationship to regional wall motion. Circulation 1982; 66:649–657
35. Wilson PK, Fardy PS, Froelicher VF: Cardiac Rehabilitation, Adult Fitness and Exercise Testing, Chapter 11. Lea & Febiger 1981, pp 249–286

APPENDIX

Approximate Energy Expenditure for Common Physical Activities

APPENDIX: APPROXIMATE ENERGY EXPENDITURE FOR COMMON PHYSICAL ACTIVITIES

Energy	Recreation	Activity
1.5–2 MET	Standing	Shaving, dressing, showering
4–7 mL O_2/min/kg	Card playing	Desk work
2–2.5 Kcal/min	Sewing or knitting	Electric typing, writing
	Flying, motorcycling	Automobile driving
	Walking (1–2 mph or 1.6–3.2 kph)	Dusting, light housework
	Model ship building	Calculator machine operation
	Bed exercise	Watch repair
		Light assembly work
		Hammering
2–3 MET	Golf (powercart)	Car washing
7–11 mL O_2/min/kg	Bowling, billiards	Automobile repair
2.5–4 Kcal/min	Darts, piano playing	Kneading dough
	Fishing (standing, sitting), croquet	Manual typing
	Power boating, shuffleboard	Ironing, tailoring
	Skeet shooting, light woodworking	Bartending
	Walking (2–3 mph or 3.2–4.8 kph)	Heavy lever work, dredge
	Cycling (5 mph or 8 kph)	Riding power lawn mower
	Shopping	Television, radio repair
	Kayaking, rowing, canoeing (2.5 mph or 4 kph)	Crane operator
	Horseback riding (walk)	Upholstering
	Horseshoe pitching	Using hand tools
		Cleaning, scrubbing, waxing
		Washing, polishing, sanding
3–4 MET	Calisthenics (light)	Janitorial work
11–14 mL O_2/min/kg	Fly fishing	Brick laying, plastering
4–5 Kcal/min	Archery	Twisting cables, pulling on wires
	Badminton (doubles), tennis (triples)	Raking leaves
	Sailing (small boat)	Machine assembly
	Ice boating	Hitching trailers
	Horseback riding (trot)	Cranking up dollies
	Badminton (social doubles)	Mopping, hanging wash
	Softball (noncompetitive)	Window cleaning
	Musician (energetic)	Light plumbing
	Golf (pulling bag cart)	Welding (moderate)
	Volleyball (6-man noncompetitive)	Plowing, tractor
	Walking (3–3.5 mph or 5–5.8 kph)	Trailer-truck driving
	Cycling (6 mph or 10 kph)	Light lawn mower pushing
		Wheelbarrow (100 lb or 220 kg)
		Assembly line work (light or medium parts appear at an approximate rate of 500/d or more of lifting 45-lb part every 5 minutes or so)
		Stocking shelves, packing or unpacking light or medium objects
4–5 MET	Table tennis	Heavy machine repair (farm, airplane, plumbing)
14–18 mL O_2/min/kg	Golf (carrying bag)	Using powersaw on hardwood
5–6 Kcal/min	Dancing (social)	Pushing a power mower, cart, dolly
	Tennis (doubles)	Gardening, hoeing
	Badminton (singles)	Bailing hay
	Calisthenics (moderate)	Carpentry (light)
	Swimming (light)	Painting
	Tetherball	Masonry
	Baseball (noncompetitive)	Paperhanging
	Walking (3.5–4 mph or 5.8–6.4 kph)	Interior repair, remodeling
	Cycling (6.5–8 mph or 10.4–12.8 kph)	Stair climbing (slow)
	Kayaking, rowing, canoeing (3 mph or 4.8 kph)	Putting in a sidewalk
		Carrying trays, dishes
		Gas station mechanic

Appendix continued

APPENDIX: APPROXIMATE ENERGY EXPENDITURE FOR COMMON PHYSICAL ACTIVITIES (Continued)		
Energy	**Recreation**	**Activity**
4-5 MET 14–18 mL O_2/min/kg 5-6 Kcal/min		Military marching Lifting and carrying 20–44 lb General heavy industrial labor Walking from room to room Pumping tire Plowing with a horse
5-6 MET 18–21 mL O_2/min/kg 6–7 Kcal/min	Hunting (small game) Fishing (wading) Softball (competitive) Horseback riding (posting trot) Soccer (noncompetitive) Walking (4 mph or 6.4 kph) Cycling (8.85 mph or 12.8–13.6 kph) Kayaking, rowing, canoeing (4 mph or 6.4 kph) Ice or roller skating (9 mph–15 kph)	Garden digging Shoveling light earth Stair climbing (moderate) Exterior remodeling or construction
6-7 MET 21–25 mL O_2/min/kg 7–8 Kcal/min	Backpacking (5 lb) Tennis (singles) Scuba diving (warm water) Downhill skiing (light) Water volleyball Ski touring (2.5 mph or 4 kph) Calisthenics (heavy) Water skiing Badminton (competitive) Walking (5 mph or 8 kph) Cross-country hiking Dancing (rhumba, square) Swimming (moderate)	Shoveling 10 lb or 22 kg 10 times a minute Snow shoveling Hand lawn mowing Splitting wood Using pneumatic tools Carry, lifting 46–64 lb Spading
7–8 MET 25–28 mL O_2/min/kg	Horseback riding (gallop) Snowshoeing (3 mph or 4.8 kph) Sledding, tobogganing Ice hockey Touch, flag football Paddle ball Swimming (fast) Mountain climbing Basketball (non-game) Walking (6 mph or 9.6 kph) Cycling (12 mph or 19 kph) Kayaking, rowing, canoeing (5 mph or 8 kph) Jogging (5 mph or 8 kph) Downhill skiing (vigorous)	Stair climbing (fast) Ditch digging Hand saw, ax on hardwood Carrying, lifting 65–84 lb Laying railroad track Carrying 20 lb up stairs
8–9 MET 28–32 mL O_2/min/kg 10–11 Kcal/min	Light sparring (boxing, martial arts) Fencing Scuba diving (cold water) Handball (social) Endurance motorcycling Basketball (vigorous) Soccer (competitive) Squash (social) Running (5.5 mph or 9 kph) Cycling (13 mph or 21 kph) Ski touring (4 mph or 6.5 kph) in loose snow Back packing (30 lbs)	Carrying, lifting 85–100 lb Shoveling 14 lb or 31 kg, 10 times a minute Tending furnace Using a pick Using a bar, sledgehammer Climbing a ladder Moving, pushing desks, file cabinets Stock furniture; also pushing against heavy spring tension Shoveling 16 lbs or 36 kg 10 times a minute

APPENDIX: APPROXIMATE ENERGY EXPENDITURE FOR COMMON PHYSICAL ACTIVITIES (Continued)

Energy	Recreation	Activity
10+ MET 32+ mL O_2/min/kg 11+ Kcal/min	Basketball (competitive) Hunting (big game) Heavy sparring (boxing, martial arts) Ski touring (5+ mph or 8+ kph) Running 6 mph = 10 MET 7 mph = 11.5 MET 8 mph = 13.5 MET 9 mph = 15 MET 10 mph = 17 MET Cycling 14 mph = 10 MET 15 mph = 11.5 MET 16 mph = 12.5 MET Swimming (crawl) 850 yd/18–20 min = 10 MET 950 yd/20–22 min = 11 MET 1000 yd/20–22 min = 12 MET Gymnastics Judo Spaceball Trampolining Wrestling	

At rest, 1 MET = 3.5 mL O_2/kg/min
These values are for a 70-kg person
Some activities may have a large range of metabolic activity due to excitement, stress, anxiety, or impatience