"chest pain" may lead to a negative response in a patient experiencing angina pectoris. When taking a history aimed at discerning angina pectoris, one needs to seek answers to these more nuanced descriptions of symptoms. In addition to chest discomfort, patients may have associated discomfort in the arm, throat, back, or jaw. They also may experience dyspnea, diaphoresis, or nausea associated with angina pectoris.

There is a good deal of variability in the expression of symptoms related to myocardial ischemia, although each person tends to have a unique signature of symptoms. Some have no chest discomfort but only radiated arm, throat, or back symptoms; dyspnea; or abdominal discomfort. Myocardial ischemia can also manifest in a "silent" form, particularly in the elderly and in patients with long-standing diabetes mellitus. The duration of angina pectoris varies, probably depending on the magnitude of the underlying myocardial ischemia. Exertion-related angina pectoris, the hallmark of stable obstructive CAD, typically resolves with rest or with decreased intensity of exercise. In stable angina pectoris, the duration of events is usually in the range of 1 to 3 minutes. Prolonged symptoms in the 20- to 30-minute range are indicative of a more serious problem such as NSTEMI or STEMI.

The physical examination of patients with CAD is typically normal. However, if the patient is physically examined during an episode of myocardial ischemia, either at rest or after exertion, significant changes may be present. As with any form of discomfort, there may be a reflex increase in heart rate and blood pressure. Elevated heart rate and blood pressure may act to sustain the duration of angina by increasing myocardial oxygen demand in the face of supply-limiting coronary stenosis. Acute mitral regurgitation can develop if the distribution of myocardial ischemia includes a papillary muscle, the supporting structure of the mitral valve. The physical examination in such cases would demonstrate a new systolic murmur consistent with mitral regurgitation. If severe enough in degree, this mitral regurgitation will cause decreased LV compliance and, consequently, an acute elevation in left atrial and pulmonary vein pressure leading to pulmonary congestion. In this setting, the patient will have not only the symptom of angina pectoris but also the symptom of dyspnea and the physical finding of rales. Ischemia-induced increases in LV filling pressure due to diminished compliance also can occur independently of ischemia-induced mitral regurgitation. Decreased LV compliance can produce the abnormal heart sound S_4; in the case of severe diffuse myocardial ischemia causing LV systolic dysfunction, an S_3 may also be perceived. Resolution of myocardial ischemia results in not only a cessation of angina pectoris but also a return to the patient's baseline physical examination status.

Diagnosis and Differential Diagnosis

Three basic forms of testing have played major roles in assessing patients with chest discomfort possibly due to CAD. All of these tests capitalize on the effect of myocardial ischemia on various aspects of cardiac physiology. First, myocardial ischemia induced by exercise or by spontaneous coronary occlusion results in subendocardial ischemia, which appears on an ECG as diffuse ST depression (Fig. 8-2). Once ischemia resolves, the ECG returns to normal. Second, myocardial ischemia typically affects a segment of heart muscle, and that territory develops a wall motion abnormality that can be detected by either echocardiography or nuclear scintigraphy. Third, the basis for myocardial ischemia is a decrease in coronary and myocardial blood flow. This abnormality can be detected by assessing the distribution of radioactive tracers such as thallium 201 or technetium sestamibi using specialized detectors for imaging myocardial perfusion. All stress test techniques used in diagnosing patients with possible CAD rely on these means of detecting the impact of myocardial ischemia on cardiac electrical activity, mechanical function, or myocardial perfusion.

Stress testing in its various forms frequently plays a pivotal role in the assessment of patients with possible CAD. In using stress testing, it is important to understand the significance of pretest probability of CAD in interpreting the results of any stress test method. For a patient with a high pretest probability of CAD, a positive test is highly predictive of underlying CAD, and a negative test carries the weight of being falsely negative. The opposite is true in a patient with a low pretest probability of CAD: A negative test is associated with a high negative predicative value for the presence of CAD, but a positive test is likely to be falsely positive. These factors play into a clinician's interpretation of test results and must always factor into decision making regarding the need for additional testing.

Stress testing is useful not only as a diagnostic tool but also in the long-term management of established CAD. Exercise stress testing, through its ability to quantify exercise capacity, can monitor the effectiveness of medical therapy directed at reducing myocardial ischemia. The findings of an exercise stress test also have predictive value in that patients with ischemia induced at low workloads are more likely to have extensive multivessel disease, whereas those who achieve high workloads are less prone to ischemic complications of CAD. A higher risk for poor outcomes related to CAD is implied by (1) ECG changes of ST depression early during exercise and persisting late into recovery; (2) exercise-induced reduction in systolic blood pressure; and (3) poor exercise tolerance (<6 minutes on the Bruce stress test protocol).

Patients with a normal resting ECG can reliably be assessed by standard exercise stress testing with ECG monitoring (Fig. 8-3). The specificity of ST changes with exertion is significantly reduced in the face of baseline ECG abnormalities related to LV hypertrophy, left bundle branch block (LBBB), pre-excitation, or use of digoxin. Various imaging techniques (echocardiography, nuclear scintigraphy, magnetic resonance imaging) have been developed to overcome the impact of baseline ECG abnormalities on the validity of stress testing. Because women also have lower specificity for ECG changes during exercise testing than men, an imaging technique is frequently used in the assessment of women. Overall, the addition of an imaging technique to stress testing significantly improves the sensitivity, specificity, and predictive value of the stress test but also greatly increases its cost.

Radionuclide stress testing is a common form of imaging-based stress test. Near peak exertion, a radionuclide tracer (thallium 201, technetium 99, or tetrofosmin) is administered intravenously. The tracer is distributed to the myocardium in a quantity directly proportional to blood flow. This type of image testing relies on a disparity of tracer uptake to detect an area of ischemia. Thallium 201 redistributes over 4 hours to viable