A 58-year-old man developed ventricular fibrillation and a large anterior infarction one hour after a negative exercise test. Coronary angiography post-infarction suggested the presence of a "complicated" lesion, raising the possibility that the infarction resulted from sudden occlusion of a previously nonsignificant lesion. (Chest 1988; 94:590-81)

Coronary artery disease is present in the majority of patients with sudden death, but sudden cardiac death may occur despite performance of an exercise test with negative result. This may be the result of a false negative test secondary to inadequate sensitivity of the ECG to detect myocardial ischemia or to performance of inadequate exercise to produce myocardial ischemia despite the presence of significant obstructive coronary disease at the time of the test. Alternatively, the following case report suggests that the mechanism responsible for sudden death following a negative exercise test in some patients may be development of an acute coronary arterial lesion.

**Case Report**

A 58-year-old white man complained of intermittent chest pain for one week. The pain was substernal, burning in quality, nonexertional, and lasted for several minutes before spontaneous resolution. Risk factors for coronary artery disease included smoking one pack of cigarettes/day and hypercholesterolemia.

There were no significant findings on physical examination or chest roentgenogram. Routine daily ECGs were notable for varying T-wave polarity in anterolateral leads, with no associated symptoms. There was no elevation of cardiac enzymes.

The patient was initially treated with nitrates, and later changed to therapy with diltiazem. There was no complaint of chest pain after therapy was initiated, and he was referred for exercise testing for further diagnostic evaluation. The patient exercised for 6.5 minutes (Bruce protocol), stopping secondary to fatigue. The maximum heart rate was 152 beats/minute (94 percent of age-predicted maximum heart rate) and maximum blood pressure was 180/90 mm Hg. There was no chest pain, significant ST segment shift, or arrhythmia during exercise or recovery; 12 lead ECGs were recorded following each minute of exercise, at peak exercise, and after each minute of an 8-minute recovery period.

One hour after exercise the patient complained of chest pain and
collapse. Cardiopulmonary resuscitation was started; the initial ECG revealed ventricular fibrillation. The patient was successfully defibrillated into sinus rhythm, at which time the ECG revealed marked ST elevation in leads I, AVL and V1-V6.

The patient was admitted to the coronary care unit, where sequential electrocardiograms revealed evolution of an extensive transmural anterior wall myocardial infarction; peak CPK level was 4,000 IU/L. His course in the coronary care unit was significant only for mild heart failure, which was treated with digoxin and furosemide (Lasix).

Six days following admission he again developed ventricular fibrillation and was successfully defibrillated. There was no evidence of further infarction following this event, and the patient was referred for cardiac catheterization. The ventriculogram revealed anterolateral and apical akinesis. The left anterior descending artery had a long, eccentric, irregular lesion with intraluminal lucency in the proximal third portion (Fig 1). There was no significant narrowing in the left main, right coronary, or left circumflex arteries.

**Discussion**

The majority of patients with sudden death secondary to ischemic heart disease have complicated atherosclerotic lesions. In a case similar to ours, Lintgen et al. reported a patient who died 30 minutes after a negative exercise test which included a 10-minute recovery period; autopsy revealed total occlusion of the left anterior descending coronary artery, resulting from hemorrhage into an atherosclerotic plaque and intraluminal thrombus.

Levin and Fallon compared angiographic morphology and histologic appearance of atherosclerotic plaques in patients dying of myocardial infarction or undergoing coronary bypass surgery, and found that irregular borders and intraluminal lucencies on the angiogram correlated with the presence of complicated coronary lesions (characterized by plaque rupture, plaque hemorrhage, superimposed partially occluding thrombus, or recanalized thrombus). The appearance of the lesion in the left anterior descending artery of our patient is consistent with that of a complicated lesion.

The most likely explanation for sudden death in our patient is ventricular fibrillation resulting from an acute arterial lesion at a site where the previous stenosis was not hemodynamically significant, and therefore unlikely to result in detectable ischemia at the time of exercise testing. The extent of the angiographic obstruction is probably greater than that of the plaque prior to exercise testing, owing to residual thrombus. Davies and Thomas found that patients with sudden ischemic death and coronary thrombus had occlusion at a site where the previous stenosis was <50 percent of the luminal diameter; 21/26 subjects without thrombus showed evidence of plaque fissuring or intramural thrombus, an event that may rapidly alter the occlusive nature of a lesion. Ambrose et al. showed that patients with unstable angina frequently showed progression from a previously insignificant lesion or a normal-appearing artery.

A positive exercise test depends upon the development of sufficient imbalance between myocardial supply and demand to result in myocardial ischemia, and will not usually reflect the presence of nonobstructive coronary artery lesions which do not reduce coronary flow reserve. Atherosclerotic lesions which do not limit flow or result in symptoms are usually not detected, but may be important in the genesis of sudden cardiac death. As suggested by Detrano and Froelicher, a test that indicates the presence of atherosclerotic plaque itself, rather than only plaque which results in luminal obstruction, may be useful as an adjunct to the standard ECG exercise test for identifying patients at risk for cardiac events.

**References**

3. Lintgen AB. Death from myocardial infarction after exercise test with normal result. JAMA 1976; 235:837-39