Serious cardiac events during exercise testing and during cardiac rehabilitation are rare. The risk of a cardiac fatality during medically supervised physical training is 1.3 per million patient-hours of exercise, and the risk of cardiac arrest (usually due to ventricular fibrillation) is 8.9 per million patient-hours. During treadmill exercise testing the risk of death is 0.5 per 10,000 tests, and the combined cardiac mortality-morbidity rate is 8.86 per 10,000 tests.

This report describes two cardiac arrests (one fatal) during cardiac rehabilitation, an acute myocardial infarction during exercise testing, and two instances of Prinzmetal's type of angina with ventricular tachycardia during exercise testing. Current management of such events is reviewed.

CASE REPORTS

Adverse Events During Exercise Testing

CASE 1

Acute Myocardial Infarction. A 69-year-old white man was referred for exercise testing. During the past year, he had experienced right anterolateral chest discomfort which could last a day or longer. Over the past three weeks, he had noted a "soreness" across the midchest which sometimes radiated to the neck. Four days prior to the exercise test, this discomfort had lasted for several hours. The sensation could be improved by stretching his arms.

The patient's history included hypertension, for which he received atenolol (75 mg daily) and hydrochlorothiazide (50 mg daily). He had never smoked. His mother had died of a myocardial infarction at the age of 69 years.

On cardiovascular examination, the patient's blood pressure was mildly elevated at 142/94 mm Hg, and the pulse was regular at 64 beats per minute. Findings from examination of the neck and chest were normal except for slight bilateral anterior chest wall discomfort on firm palpation. Findings from the cardiac examination were unremarkable. Laboratory studies showed a cholesterol level of 199 mg/dl and a nonfasting blood glucose level of 179 mg/dl. The resting electrocardiogram showed ST-segment depression in the inferior
and anterolateral leads, which was unchanged when compared with an ECG taken three days previously.

During the Bruce treadmill test the patient experienced mild anterior chest tightness after two minutes, which was not associated with electrocardiographic changes. The pain increased slightly at 4.7 minutes, and the test was stopped. Immediately after exercise, the pain intensified; the blood pressure fell to 74/54 mm Hg with a pulse rate of 120 beats per minute. The patient was pale and diaphoretic. The exercise ECG showed transient ST-segment elevation in lead V5 at two minutes after exercise and depression by ten minutes after exercise (Fig 1). A 12-lead ECG taken ten minutes after exercise showed extensive ischemia (Fig 2).

The patient was taken directly to the cardiac catheterization laboratory, where studies revealed total occlusion of the proximal left anterior descending (LAD) artery (with no collateralization) and of the midobtuse marginal branch of the left circumflex artery. The right coronary artery showed several areas of stenosis of 30 to 50 percent of its diameter. Intracoronary streptokinase was given, and coronary angioplasty was performed, with complete relief of the proximal LAD stenosis. The mid-LAD was noted to be diffusely diseased, with a stenosis of 50 percent of its diameter. The first diagonal branch of the LAD remained totally occluded.

An ECG obtained on the following morning showed only nonspecific T-wave abnormalities; however, because of subsequent recurrent chest pain, the patient eventually had aortocoronary saphenous vein bypass surgery.

**Case 2**

*Transient ST-Segment Elevation with Ventricular Tachycardia.* A 28-year-old white man noted a transient burning sensation in the anterior chest and dyspnea while pushing a stalled car. Several similar episodes had occurred during sexual intercourse and while awaiting a stressful interview. The medical history was unremarkable; the patient had never smoked cigarettes, exercised sporadically, and did not have a family history of early coronary disease.

On physical examination the patient's blood pressure was 130/85.

![Figure 2. Twelve-lead ECG in 69-year-old patient 10.6 minutes after exercise (case 1).](image)

**Figure 3.** Exercise ECG in 28-year-old man (case 2).
CASE 4

The patient was a 47-year-old man, asymptomatic for two years. He had a history of hypertension, for which he was taking metoprolol and a diuretic. Physical examination was normal, and laboratory data indicated a blood pressure of 140/94 mm Hg and a pulse of 100 beats per minute. Findings from the cardiopulmonary examination were normal.

Laboratory data included a total cholesterol level of 260 mg/dl and a high density lipoprotein (HDL) level of 42 mg/dl. The serum potassium level was normal at 4.6 mEq/L. The resting ECG was normal. On the Bruce treadmill test the patient had a duration of 6.5 minutes, stopping because of leg fatigue and reaching a peak heart rate of 150 beats per minute and blood pressure of 186/104 mm Hg. The exercise ECG (Fig 4) showed asymptomatic ST-segment depression.

The patient was scheduled for a follow-up thallium exercise scan. Two days before the scan, he was in the office for midscapular back pain, which had developed after walking one-half mile and subsided within a minute of rest. He would resume his walk without a recurrence. On the evening before the office visit, the discomfort had occurred while the patient was watching a football game, was associated with gas and belching, and was aggravated by lying on

CASE 5

Transient ST-Segment Elevation with Ventricular Tachycardia. A 47-year-old white man was seen in a routine corporate medical evaluation. He was taking metoprolol and a diuretic for a two-year history of hypertension. The patient had quit smoking cigarettes in 1984, after 20 pack-years. He usually walked three miles an average of five times each week, without any symptoms.

Physical examination indicated a blood pressure of 140/94 mm Hg and a pulse of 100 beats per minute. Findings from the cardiopulmonary examination were normal.

Laboratory data indicated a total cholesterol level of 260 mg/dl and a high density lipoprotein (HDL) level of 42 mg/dl. The serum potassium level was normal at 4.6 mEq/L. The resting ECG was normal. On the Bruce treadmill test the patient had a duration of 6.5 minutes, stopping because of leg fatigue and reaching a peak heart rate of 150 beats per minute and blood pressure of 186/104 mm Hg. The exercise ECG (Fig 4) showed asymptomatic ST-segment depression.

The patient was scheduled for a follow-up thallium exercise scan. Two days before the scan, he was in the office for midscapular back pain, which had developed after walking one-half mile and subsided within a minute of rest. He would resume his walk without a recurrence. On the evening before the office visit, the discomfort had occurred while the patient was watching a football game, was associated with gas and belching, and was aggravated by lying on
his back. He was given nitroglycerin and an antacid to try as diagnostic measures. He subsequently reported that the latter gave almost immediate relief.

During a thallium stress scan the patient developed the midback discomfort 5.8 minutes into the Bruce protocol. Immediately thereafter, he developed ST-segment elevation and ventricular tachycardia (Fig 5), which subsided spontaneously upon stopping the test. The thallium scan showed a perfusion defect in the anterior wall and apex, and also of the septum. Moderate redistribution was observed.

Adverse Events during Cardiac Rehabilitation

CASE 4

Ventricular Fibrillation without Infarction. A 64-year-old white man has been a long-term participant in our cardiac rehabilitation program since an anterior wall myocardial infarction in 1974 and double aortocoronary bypass grafting (with plication of an apical aneurysm) in 1981. His resting ECG showed the old anteroseptal infarct and persistent ST-segment elevation. The patient had no complaint of chest pain or dyspnea. His medication included digoxin (0.125 mg daily) and flecainide (150 mg twice daily). On prior treadmill exercise tests, he typically had developed an S3 gallop on examination after exercise and occasionally had high-grade ventricular ectopy (Fig 6).

In April 1986, midway through his usual three mile walk at the gym, the patient suddenly collapsed. Cardiopulmonary resuscitation was begun, an electrocardiographic rhythm strip revealed ventricular fibrillation, and the patient was promptly defibrillated. He was admitted to the hospital where serial ECGs and enzyme levels did not show acute myocardial injury or infarction.

Because of the patient’s chronic ventricular ectopy, the episode of ventricular fibrillation, and the ineffectiveness of or intolerance to multiple antiarrhythmic agents, he underwent electrophysiologic studies. The patient developed recurrent nonsustained ventricular tachycardia with three extrastimuli while in sinus rhythm during the baseline state. When he was loaded with 1,000 mg of intravenous procainamide, the stimulation was repeated, and he again developed ventricular tachycardia, despite a serum level of the drug of 4.6 mg/L. The patient was then loaded with oral mexiletine over three days. Ventricular tachycardia was again induced. Then oral therapy with amiodarone was tried, at a dosage of 1,000 mg daily for the first five days, gradually tapering down to 400 mg daily by three weeks. Ambulatory monitoring for 24 hours showed only rare single ventricular premature beats, as did a repeat treadmill test.

CASE 5

Fatal Myocardial Infarction. A 49-year-old white man underwent an exercise test prior to joining a corporate fitness program. His medical history indicated serum cholesterol levels ranging from 260 to 325 mg/dl, for which he was taking clofibrate. The patient smoked one pack per day and had smoked for 22 pack-years. On physical examination, his blood pressure was 124/88 mm Hg, and the pulse was 78 beats per minute and regular. His resting ECG indicated right axis deviation. On the Bruce treadmill test the patient had a duration of 10.6 minutes, stopping because of fatigue and reaching a peak heart rate of 147 beats per minute and blood pressure of 166/92 mm Hg. The exercise ECG showed borderline abnormal ST-segment depression without symptoms (Fig 7). His weight was 79.8 kg (176 lb), 21 percent of which was fat.

The patient had done well for eight months in the exercise program, walking one mile, jogging one-fourth mile, and using the stationary bicycle for 15 minutes, three times per week. At the completion of a routine exercise session, the patient had come out of the shower area complaining of dizziness, sweating, and chest discomfort. An electrocardiographic rhythm strip showed normal sinus rhythm. The patient’s dull substernal discomfort seemed similar to his “esophageal” pain, secondary to esophageal spasm diagnosed ten years earlier; however, while changing clothes, he developed an exacerbation of the chest pain. His blood pressure was difficult to obtain. Emergency treatment was initiated. The patient was transported to the hospital by ambulance, receiving cardiopulmonary resuscitation en route. He was pronounced dead at the hospital. An autopsy was not obtained.

DISCUSSION

Serious cardiac events during exercise testing are uncommon. In 1971, Rochmis and Blackburn reported one death per 10,000 tests and a combined mortality-morbidity rate of only four per 10,000 tests. In 1980, Stuart and Ellestad published the results of a national survey of exercise testing facilities (518,448
tests in 1,375 centers). The mortality was 0.5 deaths per 10,000 tests (0.005 percent), and the combined mortality-morbidity rate was 8.86 per 10,000 tests.

Three of our five case reports relate to events during exercise testing (myocardial infarction in one and transient ST-segment elevation with ventricular tachycardia in the other two). All three patients had atypical symptoms for coronary disease. One should be aware that such symptoms, when of recent onset, may reflect unstable angina pectoris, preinfarctional angina pectoris, or coronary vasospasm. Cases 2 and 3 almost certainly had the latter, with high-grade underlying proximal LAD coronary lesions.

Patients with impending myocardial infarctions or probable coronary vasospasm during exercise testing are ideal candidates for prompt invasive cardiac evaluation. Case 1 had a trial of intracoronary streptokinase, followed by percutaneous transluminal coronary angioplasty, to relieve a total LAD coronary lesion. Based on a recent report, it might be more prudent to delay angioplasty for a week or so after successful thrombolytic therapy. Cases 2 and 3 had subtotal LAD coronary lesions, which was improved with coronary angioplasty. Both are free of symptoms, with normal maximal exercise tests, 20 months and 12 months (respectively) after percutaneous transluminal coronary angioplasty.

Regarding the risks of cardiac rehabilitation, Haskell surveyed 30 programs between 1960 and 1977, noting an incidence of one cardiac arrest per 32,593 patient-hours, one fatality per 166,402 patient-hours, and one myocardial infarction per 232,805 patient-hours. A recent survey by Van Camp and Peterson reported data from 167 randomly selected programs between 1980 and 1984. The rate of cardiac arrest was only one-fourth that noted by Haskell and the rate of fatalities only one-seventh.

The fatality (case 5) occurred in a satellite facility that we help supervise and was the first we have had in 17 years. It differed from our previous experience in that the patient had an acute myocardial infarction, rather than primary ventricular fibrillation. In retrospect, the only additional medication that might have been tried is intravenous streptokinase or tissue plasminogen activator; however, the use of these medications outside of a hospital remains to be established.

Case 4, a case of ventricular fibrillation without infarction during cardiac rehabilitation, was the seventh such case (in 17 years) in our hospital-based facility, and the patient was the first to undergo electrophysiologic testing. He had been treated with flecainide, and one should be aware of the potential proarrhythmic effects of this agent, particularly during exercise. The patient's clinical response to amiodarone (based on ambulatory monitoring and exercise testing) was good, even though the drug did not prevent the induction of ventricular tachycardia in the catheterization laboratory. This discrepancy between clinical
response and electrophysiologic test response is not unusual for amiodarone. The patient's cardiac rhythm was monitored three times weekly in the supervised exercise program; only infrequent single ventricular premature beats were recorded. In view of this response, we elected not to consider an automatic implanted defibrillator.

Summary

Severe cardiac events during either exercise testing or cardiac rehabilitation are described in five patients. The three events related to exercise testing involved patients with atypical chest pain which, in retrospect, probably reflected unstable angina pectoris or coronary vasospasm (or both). Prompt coronary angiography with subsequent percutaneous transluminal coronary angioplasty was performed. Two cardiac arrests (one fatal) occurred during participation in cardiac rehabilitation programs. One was caused by primary ventricular fibrillation. Electrophysiologic testing was used in determining the efficacy of the drug. The fatality was due to an acute myocardial infarction. On-site intravenous thrombolytic therapy might prove useful in similar events in the future.

Comparisons of surveys published in the 1970s with those in the 1980s show that the mortality and morbidity during exercise testing remain low and that similar rates during cardiac rehabilitation are even lower; however, these cases exemplify that emergency situations can and do arise, necessitating prompt intervention and aggressive subsequent management.

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