Syncope during sports participation may serve as the first manifestation of cardiovascular disease that poses a risk for athletic training and competition. Other causes of syncope (vasovagal, dehydration) during physical activity may be more benign. The athlete who faints during sports deserves a comprehensive diagnostic evaluation that addresses the wide-ranging differential diagnosis involved. The case of a 14-year-old male with two syncopal spells during athletic training is presented to review the components of such a workup and subsequent management implications.

Few events generate more concern in the field of sports medicine than syncope during participation in athletics. Syncope implies a transient loss of effective cerebral perfusion; vigorous physical activity places the systems responsible for such perfusion under stress. It follows that syncope during exercise may serve as the first indicator of a significant cardiovascular abnormality that remains occult in the resting state. Moreover, such events may signal the presence of disease states that place the athlete at serious risk for sudden death during sports participation (16). Inquiry into a previous syncopal episode during physical activity has therefore been regarded as a key aspect of the preparticipation sports evaluation history in clearing individuals for athletic competition (18, 21).

The evaluation and management of the athlete who experiences syncope during sports requires an understanding of the broad differential diagnosis involved. Some causes of syncope relate to life-threatening conditions that clearly preclude participation in sports while others are more benign, preventable, and safely tolerated in athletic competition. This case report illustrates such a diagnostic evaluation and its subsequent management implications.

Case Report

A 14-year-old athletic, previously healthy male presented to his physician with an episode of syncope that occurred late in the morning near the end of a 5-mile
run. He had eaten a normal breakfast; the weather was not hot, but it was the first warm day of spring. He stated that he felt dizzy and tried to slow down, but before he could he collapsed to the ground. He was found by a passing motorist, who described him as pale and unconscious for several minutes. He was not incontinent of stool or urine. Upon regaining consciousness, he felt ‘‘somewhat uncoordinated’’ for the rest of the day and was brought to the emergency room later that evening and admitted to the hospital.

The patient was a well-conditioned soccer, hockey, and lacrosse player who ran during his spare time. There was no prior history of heart murmur, chest pain, dizziness, syncope, or palpitations. He denied use of drugs or medications. A normal CAT scan had been performed on him as a young child because of concern over macrocephaly. He was an adopted child, and no biological family history was available.

The patient described an unusual degree of sweating which was always exaggerated compared to his teammates. As a consequence he drank large amounts of fluid. Even on nontraining days it was not unusual for him to drink up to 10 glasses of fluid. During lacrosse camp he described himself as ‘‘drinking constantly.’’

Physical examination revealed a healthy appearing male with a regular pulse and blood pressure of 110/60. Examination of head, eyes, ears, nose, and throat was unremarkable. The lungs were clear. Cardiac evaluation revealed no lifts, thrills, murmurs, rubs, or abnormal heart sounds. The abdomen was benign, and neurologic examination was normal.

The resting electrocardiogram revealed nonspecific ST changes and T-wave flattening but was otherwise normal. The corrected QT interval was .40 seconds. During 48 hours of EKG monitoring in the hospital, two consecutive premature ventricular beats were noted. An echocardiogram was unremarkable. Specifically, right and left ventricular chamber size, wall thickness, and contractility were normal. The ventricular septum was of normal thickness, and there was no mitral valve prolapse. Both coronary arteries were found to arise normally off the base of the aorta.

During a treadmill stress test the patient completed the fifth stage of the Bruce protocol, achieving a maximum heart rate of 190 bpm. No ectopy or significant ST changes were noted. Systolic blood pressure increased to 190 mm Hg. Routine metabolic and hematologic studies as well as a urine analysis were normal, and an electroencephalogram revealed no abnormalities.

Because of the association of exercise to unexplained syncope and the patient’s strong desire to continue sports participation, a cardiac electrophysiologic study was performed. Sinus node function was normal. Wenckebach cycle length was 600 milliseconds, consistent with increased AV node vagal tone. There was no inducible ventricular tachycardia, supraventricular tachycardia, or atrial fibrillation. During isoproterenol infusion he manifested no dysrhythmias. It was concluded that the syncopal event was related to intravascular volume depletion, and given the failure to identify a cardiac etiology, the patient was discharged with approval for sports participation.

Two months later he experienced a 20- to 30-second repeat syncopal episode after he had run approximately 400 yards. The day was exceptionally hot, with temperature in the high 90s. The athlete was taken to a local hospital within 15 minutes; no abnormalities were noted. He was subsequently referred to a pediatric cardiologist, who found a normal cardiac examination. In the supine
position his heart rate was 60 bpm and blood pressure was 120/80. Upon assuming the standing position, the heart rate rose to 68 bpm and blood pressure was 110/90. The electrocardiogram was unchanged. A Holter 24-hour tracing showed no supraventricular or ventricular ectopy. A neurologic consultant could find no abnormalities. Coronary angiography revealed normal distribution of the coronary arteries, with no evidence of anomalous origin or course.

A tilt-table test was markedly positive. In the baseline state the supine blood pressure and heart rate were normal. Upon tilt to 80° he became symptomatic with dizziness related to a drop in his systolic blood pressure from 120 to 75 mm Hg. Isoproterenol was then infused at a dosage of 1 mcg/min followed by 2 mcg/min. The sinus rate increased from 80 to 120 bpm. With tilt to 80° during 2 mcg/min, his blood pressure again dropped from 130 to 75 mm Hg systolic with severe dizziness and tingling of the fingers, forcing termination of the test. When returned to the supine position, his blood pressure rapidly returned to normal, but heart rate changed from a sinus tachycardia to a junctional response at a rate of 70 bpm lasting 10–20 seconds.

A follow-up treadmill exercise test using a modified Balke protocol with a running speed of 5.5 mph revealed a maximal oxygen uptake of 55.3 ml/kg/min. No abnormal symptoms occurred, and the electrocardiogram remained free of dysrythmia and ischemic ST changes. Blood sugar was 84 and 101 mg/dl prior to and immediately after maximal exercise testing, respectively.

These testing data were felt to be indicative of vasovagal syncope, probably initiated by hypovolemia in an athlete rendered particularly susceptible by significant hyperhidrosis. He was allowed to return to competition with advice to pay strict attention to adequate hydration prior to sports participation. The use of salt tablets, 9-alpha flurocortisol, or beta blocker therapy were to be considered if his symptoms recurred.

**Discussion**

The conditions and mechanisms responsible for syncope at rest are multiple and often benign (7). When syncope occurs during or immediately after exercise, the differential diagnosis is both refined (neurologic causes become less likely) and broadened (the influences of dehydration, heat, and peripheral vasodilation need to be considered). Listed below are the etiologies that deserve consideration in this case.

Cardiac diagnosis of syncope with exercise includes the following:

- Hypertrophic cardiomyopathy
- Aortic stenosis
- Coronary artery anomalies
- Tumor
- Cardiomyopathies
- Mitral valve prolapse
- Drugs
- Pulmonary hypertension
- Prolonged QT syndrome
- Sick sinus syndrome
- Complete heart block
- Idiopathic ventricular tachyarrhythmia
Noncardiac diagnosis of syncope with exercise includes the following:

- Hyperthermia/dehydration
- Dependent venous pooling
- Hyperventilation
- Vasovagal
- Hypoglycemia
- Seizure disorder

**Cardiac Syncope**

As noted previously, syncope presenting as a manifestation of heart disease deserves primary consideration in the athlete who faints during sports participation (16). Fortunately, most of the cardiac anomalies that cause syncope are rare; unfortunately, many are also occult and their detection requires a careful diagnostic evaluation. The pathophysiology of syncope and risk of sudden death in these diseases is not well understood, but in most cases cardiac dysrhythmias (either marked bradycardia or ventricular fibrillation) are considered to be the final pathway to symptoms.

Hypertrophic cardiomyopathy (idiopathic hypertrophic subaortic stenosis, IHSS) is characterized by diffuse myocardial thickening which disproportionately involves the ventricular septum. The degree of hypertrophy in these patients can be dramatic, with obliteration of the left ventricular cavity during ventricular systole.

The condition is inherited in an autosomal dominant fashion, and a positive family history can be obtained in 20% of cases (5). The risk of sudden death is high, in some series reaching 2–4% per year. Because sudden death from IHSS is often precipitated by exercise, patients with this condition need to be restricted from vigorous sports participation (15). The mechanism of death does not relate to outflow obstruction but rather to rhythm disorders secondary to impaired diastolic filling and coronary insufficiency.

Syncope with exercise is often an early indicator of IHSS, as are symptoms of angina and shortness of breath with activity (5, 14, 15). The diagnosis is made most specifically by echocardiography upon visualization of asymmetric septal hypertrophy. The electrocardiogram is almost always abnormal as well, demonstrating left ventricular hypertrophy and ischemic ST changes. Physical examination can be deceptively benign, often with only a nonspecific systolic murmur.

Syncope with exercise can also be evidence of congenital abnormalities that create obstruction to left ventricular outflow (valvar, discrete subvalvar, and supravalvar aortic stenosis). The risk of syncope and/or sudden death in these conditions is related to the degree of outflow gradient (8). Diagnosis is more easily suspected by clinical examination in patients with aortic stenosis, who demonstrate a loud aortic outflow murmur, systolic thrill, and—with valvar stenosis—a prominent early systolic ejection click. The diagnosis can be confirmed noninvasively by two-dimensional Doppler echocardiography.

Symptomatic atherosclerotic coronary artery disease does not occur in high school athletes, but coronary insufficiency resulting from congenital anomalies of the coronary arteries is a recognized cause of sudden death during sports participation (13). The most common condition is origin of the left coronary artery from the right sinus of Valsalva, causing this vessel to course between the
aorta and pulmonary artery. The mechanism for symptoms and sudden death is unclear but may involve compression of the left coronary artery by the engorged great vessels during exercise or kinking created by the acute takeoff angle of this vessel. Other potentially significant anomalies include single coronary artery, origin of the right coronary artery from the left sinus, and anomalous takeoff of the left coronary artery from the pulmonary trunk. Intimal fibroplasia causing stenosis of a critical coronary artery branch has also been reported to be responsible for sudden death in athletes (10).

Intuitively, it would be expected that syncope and chest pain with exercise might serve as harbingers of these anomalies and alert the physician to the risk of sudden death. In most reported cases, however, sudden death occurs in a previously asymptomatic individual, even after years of competitive sports participation (24).

Nonetheless, syncope with exercise signals a need to rule out the presence of a coronary artery anomaly. The origins of the coronary arteries can usually be visualized by two-dimensional echocardiography. Treadmill testing to rule out ischemic electrocardiographic changes during exercise is important. In suspect cases, coronary arteriography will provide a definitive diagnosis.

Other cardiac conditions that predispose to dysrhythmia or depressed cardiac output with exercise need to be considered in the diagnostic evaluation. Cardiomyopathies of any etiology may trigger ventricular tachycardias under the adrenergic stimulation of sports participation. Thiene et al. recently described a high incidence of right ventricular cardiomyopathy among young Italians experiencing sudden unexpected death (22). In many (but not all) cases these myocardial diseases can be detected echocardiographically.

Mitra l valve prolapse (MVP) does not pose a risk for syncope or sudden death per se, but patients with MVP demonstrate a predisposition for ventricular and atrial dysrhythmias. McCaffrey et al. have noted that while some believe that MVP is not associated with sudden unexpected death in the pediatric age group, at least six reported cases in the literature have listed MVP as the cause of death in children (16).

The possible influence of both recreational and therapeutic drugs needs to be considered in any patient with syncope, particularly cardiotropic medications (quinidine, tricyclic antidepressants) and drugs of abuse (e.g., cocaine). Syncope with exercise can be the presenting complaint of the patient with pulmonary hypertension as well as in patients with “sick sinus syndrome,” or complete heart block (Stokes Adams attacks) (12). Patients with prolonged QT interval syndrome, as demonstrated by a corrected QT interval of over .44 seconds on the electrocardiogram, are at risk for ventricular tachycardias and sudden death that can manifest initially by syncope during exercise, when serum epinephrine levels are elevated (17). Typically these patients have a family history of similarly affected individuals. And finally, although even more unusual, unexplained ventricular tachycardia and/or fibrillation can occur in young athletes in the absence of any identifiable disease substrate (2, 4).

It is apparent from this review that in most situations a careful history, physical examination, electrocardiogram, two-dimensional Doppler echocardiogram, and routine treadmill stress test should provide sufficient information to assess the presence or absence of primary cardiac causes of syncope with exercise. Other procedures including 24-hour EKG recording, coronary angiogra-
phy, and cardiac electrophysiologic testing can be performed as indicated. In the case of the patient under discussion, a comprehensive evaluation failed to reveal any evidence of cardiac disease or predisposition to arrhythmia.

**Noncardiac Syncope**

No epidemiologic assessment of the causes of exertional syncope has been performed, but one might suspect that alterations in peripheral vascular resistance, hydration status, heat, and ventilation accompanying exercise are more often responsible for syncope during sports than primary cardiac disease is. It has long been conventional coaching wisdom, for instance, that athletes should keep moving after running competition to prevent syncope from venous pooling in vasodilated leg muscles (6). Hyperthermia and dehydration are well-recognized causes of fainting during or after competition. Hypoglycemia is not common but should be considered in the differential diagnosis, and rare cases of seizure disorders triggered by physical activity have been reported.

Buja et al. described three young athletes who demonstrated asystole with syncope secondary to voluntary hyperventilation (3). Karofsky reported 10 high school athletes who had air hunger, anxiety, or cramping (but not syncope) from hyperventilation during competition, particularly when losing (11). Since loss of consciousness can occur from hyperventilation (secondary to hypocapnia and alkalosis), it appears that hyperventilation during intense sports participation might trigger a syncopal spell.

The description of the timing and nature of the syncopal spell in this case report, coupled with the selected laboratory studies, eliminated these diagnostic possibilities. They did suggest, however, that these syncopal episodes might be vasovagal in nature.

Vasovagal syncope results from a sudden loss of vascular tone, which appears to be independent of bradycardia. Experimental evidence suggests that vasovagal reactions are paradoxically triggered by sympathetic hyperactivity, a conclusion supported by the effectiveness of beta-blocker therapy in this condition (1, 25). Animal studies by Oberg and Thoren indicate that vasodepression may be initiated by left ventricular mechanoreceptors that are responding to ventricular cavity obliteration (19). Vasovagal syncope therefore appears to be a reaction to (a) augmented cardiac contractility induced by increased sympathetic activity, and (b) reduced ventricular preload, causing cavity obliteration. These conditions are met in the exercising athlete who is volume depleted. This model conforms to the subject of this case report, an adolescent with hyperhidrosis who experienced syncope running in warm weather.

The upright tilt test with infusion of exogenous catecholamine (isoproterenol) to demonstrate symptomatic hypotension and bradycardia has been used to identify patients with vasovagal syncope (1, 20, 23). In a typical protocol, the patient is tilted on a table to 60° for 10 minutes. Blood pressure, pulse, and symptoms are assessed. The patient is then returned to the supine position and an isoproterenol infusion is started. The subject is again tilted to 60° for 5–10 minutes. If no symptoms occur, the isoproterenol dose is increased until a heart rate of 150–170 bpm is reached. A positive test is defined by the appearance of symptoms (syncope or presyncope) with bradycardia and/or hypotension. The reader is referred to other sources for details of tilt protocols (1, 20, 23).
This test therefore seeks to duplicate the pathophysiologic state of the vaso-vagal reaction: diminished left ventricular filling due to venous pooling in the legs, decreased left ventricular volume, and an increased inotropic state created by isoproterenol. It also simulates the physiologic situation found in the exercising athlete who is volume depleted.

Almquist et al. described positive tilt tests in 14 of 24 adults with recurrent syncope (vs. 2 of 18 control subjects) (1). Thilenius et al. performed tilt tests with isoproterenol infusion in 35 teenagers with a history of syncope (23). Eighty percent demonstrated a rapid onset of bradycardia within 10–15 seconds with syncope or presyncopal symptoms. The authors concluded that the tilt test should be considered the first step in evaluating the older child with syncope.

Vasovagal syncope has generally been considered benign, although prolonged asystole has been reported (9). Medical therapy has been recommended to those patients with repeated episodes or those with syncope during exercise. Beta blocker therapy (atenolol or long-acting propranolol) has proven effective in a majority of cases. As an alternative method for athletes, a combination of 9-alpha fluorocortisol and salt supplements have been used to expand blood volume (23).

**Summary**

The case of an adolescent athlete with two syncopal episodes during running was presented. An exhaustive evaluation failed to reveal any primary cardiac, neurologic, or metabolic cause. A tilt test was positive, supporting the diagnosis of vasovagal syncope. This patient described significant hyperhidrosis, a predisposing condition for volume depletion. It was concluded that hypovolemia combined with the adrenergic effects of exercise triggered vasovagal reactions that led to syncope. This case emphasizes the need for a thorough evaluation of syncope before allowing the athlete to continue sports participation.

**References**