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# Syncope in the Athletic Patient

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**Abstract** Syncope is a common but concerning event in athletic patients. As such, efforts must be made to distinguish presyncope from syncope with a critical distinction of syncope *during* exercise and postexercise syncope. Syncope most often occurs just after exercise and is usually benign; however, syncope during exercise may be a sign of pathologic structural or electrical cardiac issues. Solving this diagnostic puzzle mandates a detailed history and examination frequently augmented with diagnostic testing and imaging studies. Recommendations for treatment and potential restriction from activity also present challenging decisions to the health care provider. (Prog Cardiovasc Dis 2012;54:438-444)


*Keywords:* Syncope; Exercise; Cardiac pathology

Syncope in an athletic patient is a common symptom but should always raise concern for cardiac pathology. Although most cases of syncope in athletes are reflex syncope and considered benign,[1] even neurally mediated syncope, if it occurs when an athlete is in a physically dangerous circumstance (such as diving, motor sports, road cycling), may still be life threatening. Syncope during exertion is often caused by underlying heart disease and may be a harbinger of sudden cardiac death (SCD).[2-4] Because there is no definitive diagnostic test and a cause of syncope is only established in 50% of cases,[5] syncope in an athlete requires a thorough evaluation including a detailed and focused history and physical with the addition of case-dependent diagnostic tests. Ultimately, the purpose of this evaluation is to distinguish between life threatening and non–life-threatening causes of syncope.

## Definition

Syncope refers to the specific pathophysiology involving a transient loss of consciousness and postural tone as a result of cerebral hypoperfusion, with spontaneous and complete recovery and no neurologic sequelae.[6] There are multiple mechanisms, both traumatic and nontraumatic, which may lead to a transient loss of consciousness. Nontraumatic causes include syncope, epileptic seizures, metabolic disorders, and posterior circulation transient ischemic attacks. In contrast to true syncope, the concept of *presyncope* is defined as a feeling of lightheadedness that precedes or almost results in collapse without a loss of consciousness.[7]

### *Epidemiology*

The prevalence of syncope in the general population is high, up to 40%.[8] In a cohort of 7568 athletes undergoing preparticipation screening, there were 474 (6.2% ) cases of reported syncope within the prior 5 years.[9] Of those with syncope, most of the cases were reported as unrelated to exercise, 12% were postexertional, and 1.3% of cases were during exercise, with diagnoses of hypertrophy cardiomyopathy, right ventricular outflow tract tachycardia, and exercise-induced neurally mediated syncope. Indeed, most

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**Abbreviations and Acronyms**

**ARVC** = arrhythmogenic right ventricular cardiomyopathy

**CTA** = computed tomographic angiography

**ECG** = electrocardiogram

**LV** = left ventricular

**LVH** = left ventricular hypertrophy

**MRI** = magnetic resonance imaging

**SCD** = sudden cardiac death

syncope in the population is neurally mediated, also known as "reflex" or "vasovagal" syncope.[10] Cardiologists often use the term *neurocardiogenic* syncope, but this nomenclature should be abandoned because the initiating source of the reflex vasodilation/bradycardia is unknown and is rarely located in the heart.

### *Postexercise syncope*

Syncope that occurs *after* exercise must be distinguished from "exercise-associated collapse," where the athlete falls to the ground without true loss of consciousness or cerebral hypoperfusion.[11] In such circumstances, urgent noncardiogenic causes such as exercise-induced hyponatremia or heat illness should be considered. Exercise-associated collapse also may be a symptom of exhaustion or more commonly may be caused by neurally mediated syncope that is precipitated by standing still after exercise. For example, exercise leads to an increase in heart rate (parasympathetic withdrawal and increase in sympathetic activity), an increase in contractility and stroke volume (sympathetic activity), and balance between sympathetic vasoconstriction in inactive vascular beds and active skeletal muscle vasodilation due to metabolic, flow-mediated, and neural mechanisms.[12] Together, these exercise responses result in a marked increase in cardiac output, which is redistributed to exercising muscle. During exercise, maintenance of the increased cardiac output is preload dependent and requires peripheral muscle activity to return venous blood to the heart, hence the term *muscle pump*. When exercise is stopped suddenly, the muscle pump ceases to function, and venous return to the heart is reduced with a subsequent fall in left ventricular (LV) end-diastolic volume, stroke volume, and thus, cardiac output. Although the complete mechanism is poorly understood, neurally mediated syncope may subsequently occur through a combination of neural and hormonal mechanisms.[13] Although it does not account for all cases of reflex syncope, one such mechanism is the cardiac depressor reflex, otherwise known as the Bezold-Jarisch reflex.[14] During the acute reduction in preload and cardiac filling with the sustained elevation of catecholamines, the increased myocardial contractility may lead to activation of the LV chemoreceptors and mechanoreceptors within the LV wall and, through the cardiac depressor reflex,[13] induce hypotension and bradycardia. Regardless of the ultimate mechanism, the end result from the acute reduction of venous return is a loss of postural tone and collapse.[11] Neurally mediated syncope during exercise is less common, but still can occur, perhaps stimulated by a robust skeletal muscle vasodilation and marked "functional sympatholysis," which results in failure of sympathetic vasoconstriction leading to hypotension.[15] The physiologic adaptations to training, such as increases in vagal tone, vascular conductance, and cardiac compliance may predispose athletes to reflex syncope.[16,17]

### *Syncope during exercise*

Syncope during exercise is more concerning and has been linked with cardiovascular diseases such as hypertrophic cardiomyopathy, coronary anomalies, ion channel defects, and arrhythmogenic right ventricular cardiomyopathy (ARVC).[3,9,18-21] As such, patients who report a loss of consciousness during exercise require a focused and detailed workup for underlying cardiac pathology. It is important to note that, until a diagnosis is established or pathologic causes excluded, the athlete should be restricted from further exercise activities, although in certain highly selected cases, especially with immediate access to an automated external defibrillator, carefully supervised training may be permitted.[15]

### *History*

Evaluation of all patients having unexplained syncope during or after exercise should begin with a detailed history and physical examination as it may identify the etiology of syncope.[5] Distinguishing syncope during exercise from syncope immediately after exercise is key and often requires interviewing not only the patient but also any bystanders or family who were present during the episode of syncope; environmental factors and details of recovery are also important to establish. In fact, given the stoic nature of many young patients and often the secondary gain in withholding symptoms, having a parent present during most of the interview may increase the sensitivity of the examination. If resuscitation was performed, those records should be obtained.

Specific details of the event may also help in determining etiology, such as the presence or absence of a prodrome such as the lightheadedness and nausea of neurally mediated syncope vs chest pain, palpitations, or lack of a prodrome due to an arrhythmia. If an arrhythmia or palpitations are noted, it may be helpful to ask the patient to demonstrate their palpitations by tapping out the rhythm. Inciting factors, including loud noises, or entry into cold water would be consistent with long QT syndrome, whereas chest trauma may indicate commotio cordis. Postsyncope symptoms of confusion and bowel or bladder incontinence may be consistent with seizure activity, although patients with neurally mediated syncope often develop myoclonic jerking motions that are mistaken for seizure activity. If syncope occurs suddenly and the muscle movements occur late, a seizure is unlikely to be the primary cause, although