Syncope as a Warning Symptom of Sudden Cardiac Death in Athletes

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KEYWORDS

• Syncope • Sudden cardiac death • Athletes • Cardiomyopathy

KEY POINTS

- Clinical evaluation of syncope in the athlete remains a challenge.
- Although benign mechanisms predominate, syncope may be arrhythmic and precede SCD.
- Exercise-induced syncope should be regarded as an important alarming symptom of an underlying cardiac disease predisposing to arrhythmic cardiac arrest.
- All athletes with syncope require a focused and detailed workup for underlying cardiac cause, either structural or electrical.
- Major aim is to identify athletes at risk and to protect them from SCD.
- Athletes with potentially life-threatening etiologies of syncope should be restricted from competitive sports.

INTRODUCTION

Syncope is a sudden transient loss of consciousness and postural tone with spontaneous recovery after a brief period, which does not require electrical or medical therapy.[1–9]

Loss of consciousness results from a reduction of blood flow to the reticular activating system located in the brainstem. The metabolism of the brain is strongly dependent on perfusion and, consequently, cessation of cerebral blood flow leads to loss of consciousness within approximately 10 seconds; restoration of appropriate behavior and orientation after a syncopal episode are also immediate.[5]

Syncope is an important health problem because it is common in the general population, is often disabling, may cause injury, and may represent a prelude to SCD.[2,3]

Syncope accounts for 1% of admissions to hospitals and 3% of admissions to emergency departments. In the Framingham study, in a population of 7814 individuals, the incidence of syncope was 3% in men and 3.5% in women during a 26-year follow-up.[3] The prevalence of syncope varies with age, with a peak of first faints in patients aged 10 to 30 years (47% in female patients and 31% in patients at approximately age 15).

Causes of syncope can be classified into vascular, cardiac, neurologic-cerebrovascular, psychogenic, metabolic-miscellaneous, and syncope of unknown origin.[1–5]

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Athletes with syncope presents a unique challenge for physicians. The causes of syncope in athletes range from benign neurocardiogenic episodes to life-threatening conditions, such as ventricular arrhythmias leading to SCD.

It is mandatory to identify causes and mechanisms of syncope with the aim of excluding the underlying heart disease at risk for arrhythmic cardiac arrest.

The early detection of malignant variants of syncope has important implications for prevention of fatal events during sports. Detection of cardiovascular disorders responsible for syncope has an impact on eligibility for competitive sports activity because of the increased exercise-related risk.[10]

EPIDEMIOLOGY AND CAUSES OF SYNCOPE IN ATHLETES

There are few available data on the epidemiology and causes of syncope in athletes. Colivicchi and colleagues[8] studied a large population of 7568 athletes undergoing preparticipation screening. A syncopal episode was reported in 474 (6.2%) in the previous 5 years. Syncopal episodes were exercise unrelated in 87.7% and exercise related in 13.3%, i.e. postexertional in 12.0% and exertional in 1.3%. Over follow-up, the recurrence rate of syncope was 20 per 1000 subject-years, whereas the rate of new syncopal episodes was 2.2 per 1000 subject-years. Athletes with exercise-unrelated events had a diagnosis of either vasovagal or situational syncope. Unlike postexertional syncope, 50% of syncopal episodes occurring during exertion were "cardiogenic" (2 of 4) and caused by either hypertrophic cardiomyopathy or ventricular tachycardia.[8]

NEURALLY MEDIATED SYNCOPE

In athletes, syncope is most often neurocardiogenic and seems to have a fair prognosis.[6] Specific triggers are blood draws and prolonged standing. Classic prodromal symptoms, such as warmth, nausea, and palpitations, are present in almost cases. Usually the syncopal episodes are brief (5–30 s) and patients awake nauseous. The pathophysiology of neurally mediated syncope is not completely understood.[7]

Experimental studies demonstrated that endurance athletes, having more compliant and distensible ventricles and subject to a chronic volume load during exercise, experience a decrease in their stroke volume during orthostatic position because of a reduction in their filling pressures.

Another hypothesis is that after exertion there is a rapid decrease in venous return, causing vigorous ventricular contraction that activates mechanoreceptors, causing increased afferent neural output.[7]

ORTHOSTATIC HYPOTENSION

Orthostatic hypotension may also cause syncope during sports.[4] It is defined as a 20–mm Hg drop in systolic blood pressure or a 10–mm Hg drop in diastolic blood pressure within 3 minutes of standing and results from a defect in any portion of this blood pressure control system. Orthostatic hypotension can be asymptomatic or associated with symptoms, such as lightheadedness, dizziness, blurred vision, weakness, palpitations, and syncope. These symptoms often arise in the morning or after meals or exercise. Middle-aged and senior athletes are particularly susceptible to hypotension. Orthostatic hypotension is favored by volume depletion and reduced vasomotor tone caused by long periods of training.

CARDIOGENIC SYNCOPE IN THE ATHLETE

Approximately 1% of syncope in athletes is secondary to cardiac disease.[8] Many cardiac causes of syncope are also associated with an increased risk of SCD. In adult ages (>35 years), the most common cause is atherosclerotic coronary artery disease, whereas in younger competitive athletes ($\leq$35 years), many different conditions, such as cardiomyopathies, congenital anomalies of coronary arteries, myocarditis, aortic rupture, valvular disease, preexcitation syndromes, conduction diseases, ion channel diseases, and congenital heart disease may cause cardiogenic syncope. HCM and arrhythmogenic RV cardiomyopathy (ARVC) are leading causes of fatalities in young competitors.[11]

Hypertrophic Cardiomyopathy

HCM is a heart muscle disease, usually genetically transmitted, and characterized by a hypertrophied, nondilated left ventricle (LV) in the absence of another cardiac or systemic disease capable of producing the magnitude of hypertrophy evident.[12,13] Characteristic morphologic and functional cardiac abnormalities include asymmetric LV hypertrophy with disproportionate septal thickening and reduction in LV chamber size with increased myocardial stiffness, which may critically impair diastolic LV and intramural coronary blood filling (**Fig. 1**A). Dynamic LV outflow tract obstruction is also demonstrable at rest or with exercise in a large proportion of patients. The histopathologic hallmark of HCM is myocardial disarray, with widespread, bizarre, and disordered