

Sudden Cardiac Death in Adolescent Athletes



By **Matt Stark, M.D.**, pediatric pathologist at Children's Hospital and assistant professor of pediatric pathology at LSU School of Medicine. This issue of *Pediatric Review* is intended for pediatricians, family physicians and all other interested medical professionals. For CME purposes, the author has no relevant financial relationships to disclose.

OBJECTIVES

At the end of this activity, the participant should be able to:

1. Discuss the otherwise healthy adolescent athlete who dies suddenly
2. Describe the risk to young athletes of sudden cardiac death
3. Review the role of molecular diagnosis in sudden cardiac death

INTRODUCTION

Adolescent death is distressing to the family and community no matter the cause, and the sudden death of an adolescent participating in athletics is shocking. Pathologists have found that most sudden unexplained deaths (SUD) of the adolescent are associated with a hereditary condition, of which cardiovascular diseases are the most frequent. Sudden cardiac death (SCD) is the most common cause of death in the adolescent athlete and is an important unsolved challenge in the practice of pediatrics.

GENERAL HEALTH

The 2010 U.S. Census data tells there are 42,717,537 total children in the US from 10 – 19 years old, making them 14% of the population. In a national survey >80% of adolescents say they are in excellent or very good health and only about 5% of adolescents missed more than 11 days of school due to illness or injury in the previous 12 months.

What are the causes of death in otherwise healthy adolescents dying suddenly while engaged in athletics? Of the top 10 causes of death for adolescents, most are either traumatic or have an identifiable prodromal symptomology before the fatal event. Seizures, asthma, pulmonary embolism and intracranial bleeds can be mechanisms of death in athletic adolescents, but nearly two-thirds of sudden deaths in adolescents are attributable to sudden cardiac death.

EPIDEMIOLOGY

Sudden cardiac death in the adolescent is uncommon. Good numbers for adolescents only are not available, but <100 American athletes under the age of 35 have succumbed to SCD in a given year. The highest number of athlete deaths in the under 35 age group was 76 in 2006. The National Collegiate Athletic Association (NCAA) released five-year tracking numbers and found 45 SCD in 273 total deaths, making the incidence 1:43,770 participants per year. For perspective, SCD in young athletes is about as common as lightning fatalities.

SUDDEN CARDIAC DEATH

In adults, coronary artery disease (CAD) is the most common cause of SCD. "Premature CAD" is much less common in adolescents. Familial hypercholesterolemia may result in diffuse atheromas of the coronary arteries, leading to blockage and myocardial ischemia. One consultant in the United Kingdom saw an 11-year-old asymptomatic female "drop dead" after a cross country run. Subsequent investigation found a strong family history of hypercholesterolemia. Much more common in adolescent athletes are non-atheromatous coronary artery anomalies (CAA), including anomalous coronary arteries, coronary artery dissection, coronary artery vasculitis and coronary artery fistula. These anomalies are often asymptomatic with one study finding 60% of affected adolescents having no syncopal episodes or chest pain on exertion before SCD. CAA are the second most common cause of SCD in young athletes and are found in about 1% of the general population.

Cardiomyopathies are the most common cause of SCD in young athletes and hypertrophic cardiomyopathy (HCM) is the most frequent form in American athletes. In the general population, nearly one in 500 people are thought to be affected, with males and females having equal incidence. HCM is an inherited disease more widespread in African-American athletes, with the mutation found in the cardiac sarcomere. There are 11 identified genes contributing to HCM, with >1,000 identified mutations within those genes. Clinical recognition of hypertrophic cardiomyopathy is

	Genes Tested	Specimen	Cost*
ARVC	Cardiac desmosome (intercellular junction) -5 genes	EDTA (purple top) tube: <2 years: 2 – 3 ml	\$3,800 [#]
	Ion channel	>2 years: 3 – 5 ml	
	Nuclear envelope	Older Children & Adults: 5 – 10 ml	
HCM	Thick filament (Myosin related) -5 genes	EDTA (purple top) tube: 2 – 5 ml	HCM panel in a new patient: \$3375
	Thin filament (Tropomyosin, Troponin and the Actin Cytoskeleton) -6 genes		Prenatal diagnosis for a known mutation: \$2,000
	Sarcomere assembly -2 genes	Saliva: Company specific container	DNA testing of a relative for one/two/three known mutation(s): \$350 - \$700
	Organization of cytosolic structures		

Chart 1: Molecular Genetic Testing of Arrhythmogenic Right Ventricular Cardiomyopathy, Autosomal Dominant and Familial Hypertrophic Cardiomyopathy

enhanced by two-dimensional echocardiogram which will demonstrate an unexplained asymmetric left ventricular wall thickening.

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is another genetic cardiomyopathy with fibrous/fatty replacement of the heart muscle associated with ventricular tachycardia and sudden death in young people, especially athletes. ARVC principally involves the right ventricle (RV), but the left ventricle is often involved and there is support for adoption of the broad term arrhythmogenic cardiomyopathy. Fibrofatty replacement of the RV inferior, apical, and infundibular walls usually starts at the epicardium and extends toward the endocardium with many examples becoming completely transmural.

Establishment of a diagnosis of a heritably transmitted cardiovascular disease has implications for family members. Two forms of molecular diagnosis for genetic cardiomyopathies currently exist. One panel consists of polymerase chain reaction (PCR) amplification of all coding exons contained in 50 selected genes. Another panel utilizes next-generation sequencing technologies targeting 18 selected genes. A promising 56 gene next-generation sequencing panel is not yet ready for market (Chart 1).

If a pathogenic mutation is documented, relatives could be approached to test for the specific abnormal gene. This approach is termed targeted cascade screening and is thought to provide a greater rate of detection than general population screening. If possible, the living family member would undergo complete cardiological and genetic assessment by a multidisciplinary team consisting of members of genetics, cardiology and the psychosocial support system. Genetic counseling could highlight the advantages (preventative measures) and disadvantages (subsequent insurance cost

increase) of testing. Expert opinion has concluded that if no disease-associated mutation is identified, then evaluation may involve cardiologic examination only. The purpose behind this cascade screening is to reduce SCD in family members, using the successful model of the familial cancer screening programs.

Communication between outside physicians and the Children's Hospital Department of Pathology has previously resulted in the successful collection of high-quality DNA at the time of autopsy, allowing postmortem cardiomyopathy genetic testing. Regrettably, molecular diagnostic testing is performed in very few cases of SCD because of cost. Reimbursement for postmortem molecular testing is rarely covered by insurance companies regardless of consequences for relatives.

It is not clear if current youth athletic activity screening strategies are effective in identifying precursor cardiac lesions for SCD. Mass screenings of the general pediatric population have been undertaken in Japan and Taiwan, with associated high costs. In Japan, children were initially examined by history-taking and a resting 12-lead ECG, with detected abnormalities examined by a cardiologist. However, some diagnoses were missed, resulting in unexpected SCD. The Japanese estimated they spent \$8,800 per year of life saved.

No national standard for either healthcare professional certifications or screening standards for high school or college athletes exist. Thirty-nine states have a medical clearance form needing at least a history and physical (H&P) examination, meaning 11 do not even require this. Louisiana High School Athletic Association (LHSAA) Medical Evaluation Form is an H&P form that may be filled by MD, DO, Advanced Practice Registered Nurse or Physicians Assistant. Expert consensus finds pre-participation screening by H&P

Personal History	1. Chest pain/discomfort upon exertion
	2. Unexplained fainting or near-fainting
	3. Excessive and unexplained fatigue associated with exercise
	4. Heart murmur
	5. High blood pressure
Family History	6. One or more relatives who died of heart disease (sudden/unexpected or otherwise) before age 50
	7. Close relative under age 50 with disability from heart disease
	8. Specific knowledge of certain cardiac conditions in family members: hypertrophic or dilated cardiomyopathy in which the heart cavity or wall becomes enlarged, long QT syndrome which affects the heart's electrical rhythm, Marfan syndrome in which the walls of the heart's major arteries are weakened, or clinically important arrhythmias or heart rhythms.
Physical Examination	9. Heart murmur
	10. Femoral pulses to exclude narrowing of the aorta
	11. Physical appearance of Marfan syndrome
	12. Brachial artery blood pressure (taken in a sitting position)

Chart 2: American Heart Association 12-step screening for reduction of sudden death in young athletes

** Prices reflect cost before contractual discount, insurance discount.*

The ARVC panel also includes the genes in the HCM panel, can not be ordered separately at this time.

alone is not sufficient for detecting life-threatening cardiovascular anomalies in adolescent athletes, and the benefit of a 12 lead EKG is not clear, but it is not cost effective in mass screenings of American adolescents. Maron et al., representing the American Heart Association, recommend adolescents have a pre-participation examination every two years (Chart 2) and a medical history taken in the year between. Additional tests including EKG and Echocardiogram are not recommended for screening, but may be used by specialists if an abnormality is detected at pre-participation screening.

SUMMARY

Adolescent non-traumatic death frequently involves SCD. Although SCD is an uncommon event in young athletes, HCM and CAA are the leading mechanisms in that order. ARVC and HCM have a genetic component, but post-mortem molecular diagnosis is rarely performed because of cost and lack of insurance reimbursement. Mass population screening is associated with high costs and occasionally missed diagnoses.

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 - 7
 - 76
 - 760
 - 7,600
 - 7,6000
- Molecular diagnostic testing is performed in very few cases of sudden cardiac death (SCD) because of what factor?
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