Cardiovascular Screening of Adolescent Athletes

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KEYWORDS

• Young athletes • Electrocardiography • Sudden cardiac death
• Preparticipation screening

Preparticipation screening of competitive athletes refers to the "systematic practice of medically screening large populations of athletes before participation in sports for the purpose of identifying abnormalities that could provoke disease progression or sudden death."[1] The main objective of screening is to identify athletes who have cardiovascular risk factors so that timely evaluation and management can be initiated and appropriate decisions made about the level of physical activity or sport participation.[1–6]

Screening and prevention are the most important strategies for several reasons.[7–13] It is commonly believed that automated external defibrillators (AEDs) placed at strategic locations at athletic venues and public places will help in improving survival after a sudden cardiac arrest (SCA).[7] Results of studies done to evaluate survival after an SCA on the athletic field with timely use of an AED by reasonably trained personnel are at best equivocal.[7–13] Thirty percent to 50% of all sudden cardiac deaths (SCDs) are the first clinical manifestation of an underlying pathology.[12] Various aspects related to cardiovascular screening of young athletes and SCD are subjects of a voluminous published research and excellent reviews, commentaries, and editorials.[1–6,14–21] This article provides an overview of key aspects of cardiovascular screening currently recommended in the United States for young athletes. The main concern and impetus for such an intense focus on cardiovascular screening is the risk of SCD during sport participation.

SCD

SCD refers to "nontraumatic and unexpected sudden death that may occur from a cardiac arrest, within 6 hours of a previously normal state of health."[3] In the modern

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era of competitive sports, there have been instances of athlete deaths in almost all sports but more commonly in basketball, soccer, and football.[1–4] Sudden death in athletes is especially disconcerting because exercise has proved to decrease the risk of life-threatening cardiovascular disease.[1]

Epidemiology

Although the exact incidence of SCD during sport participation in young athletes is not known, several studies provide an estimate of the incidence and data on other epidemiologic characteristics of SCD.[1–6,22–24] The incidence of SCD is estimated to be 1 in 200,000 in high school athletes and 1 in 65,000 in collegiate athletes in the United States.[1–6,22] The incidence of cardiovascular collapse as the cause of athletic fatalities is twice that of death caused by trauma.[14,15] SCD is more common in men than in women at a ratio ranging from 5:1 to 9:1; this may be because of the higher participation rate in men in competitive sports.[1,5,16] The incidence is higher in African American athletes; the disparity could be because of a higher number of competitive athletes (almost 40%) who are African American.[1,5,23] In previous studies from Italy, the incidence of SCD in athletes was reported at 3 in 100,000.[2,25] This difference between the United States and Italy is thought to be due to the younger age of the American athletes and the inclusion of a larger number of female athletes.[2,18] In the United States, the most common sports associated with SCA are football, basketball, and ice hockey whereas in Europe it is soccer.[1–6]

Causes

Pathophysiology of SCD is explained by exercise acting as a trigger for precipitation of sometimes lethal arrhythmias in the presence of underlying structural heart diseases or other susceptibilities.[1–6,25,26] In the United States, the most common cause of SCD in young athletes (26%) is hypertrophic cardiomyopathy (HCM).[1–6,27–31] The second most frequent cause of SCD in athletes (14%) is anomalous origin of the coronary artery, most commonly the left coronary artery arising from the right sinus of Valsalva.[32,33] This group of patients may have a completely normal electrocardiogram (ECG) and exercise stress test and first manifests symptoms with exertion while playing sport.[32] Other heart-related conditions that increase the risk for SCA in young athletes are listed in **Box 1**.[1–6,10,11]

In young children, commotio cordis is an important cause of sudden death. Commotios cordis results from a blunt trauma to the chest by a fast moving projectile, such as a baseball or ice hockey puck.[10,11] The mechanism of cardiac arrest is ventricular fibrillation. The blow should be inflicted within a narrow window of time (within 10–30 milliseconds) just before the peak of the T wave during repolarization.[1,10,11] Commotio cordis accounts for 20% of SCD in children on the field.[1,10,11]

CARDIOVASCULAR SCREENING

History and Physical Examination

The current American Heart Association (AHA) recommendations for cardiovascular screening of competitive young athletes consist of a review of 12 items (**Box 2**).[1] A positive response to 1 or more of these items is considered an indication for additional cardiovascular evaluation. Information that should be ascertained in the cardiovascular screening history of young athletes is listed in **Box 3**.[1–6]

In the United States there is no mandate or law regarding preparticipation screening. The responsibility of providing screening services for student athletes rests with the institutions organizing sports. Personal physicians are expected to conduct

Box 1
Conditions affecting the heart that increase young athletes' risk for SCD

Anomalous origin of coronary artery (second most common cause in the United States)

Aortic stenosis

Aortic dissection (usually complication in Marfan syndrome)

ARVC (most common cause in Italy)

Brugada syndrome (most prevalent in those of Asian descent)

HCM

Dilated cardiomyopathy

Coarctation of aorta

Congenital heart block (Mobitz type II, complete, or third degree)

Congenital or acquired long QT syndromes

Short QT syndrome

Coronary artery disease (rare in those younger than 35)

Restrictive cardiomyopathy

Endocarditis

Ehlers-Danlos syndrome

Mitral valve prolapse

Myocarditis

Pericarditis

Postoperative congenital heart disease

Status post heart transplant

Kawasaki disease (coronary artery abnormalities)

the 12-point screening evaluation in cases of high school athletes. At higher levels of athletic participation, it is the team physician who decides appropriate medical screening procedures.

In the United States, history and physical examination (H&P) has been the standard of cardiovascular screening for competitive athletes, although several studies have questioned the efficacy of the screening H&P alone in identifying athletes at risk for cardiovascular adverse events.[1,4,18,34–38] A study of 134 cases of SCD who had undergone a screening H&P reports that only 3% of patients were suspected of having cardiac disease and eventually only fewer than 1% received an accurate diagnosis.[34] This conferred a low sensitivity to H&P as a screening tool. The investigators also reported that at the high school level, the H&P was not being administered as recommended by the AHA.[34] The low sensitivity of the H&P is partly explained by suboptimal ascertainment of information. Surveys have reported that a significant percentage of H&P forms used by high schools do not include all 12 AHA-recommended screening items (see **Box 2**). Another issue relates to who is responsible for administering the H&P. H&P is administered by professionals with different levels of qualifications. In 64% of states in the United States, nonphysicians can administer the H&P.[38] In cases of college athletics, 25% of colleges were judged to have inadequate screening H&P forms. The National Collegiate Athletic Association H&P form includes 10 of 12 items from the AHA recommendations.[36]

Box 2
The 12-element AHA recommendations for preparticipaton cardiovascular screening of competitive athletes

Personal history

1. Exertional chest pain/discomfort
2. Unexplained syncope/near syncope (judged not to be neurocardiogenic or vasovagal, of particular concern when related to exertion)
3. Excessive exertional and unexplained dyspnea/fatigue associated with exercise
4. Prior recognition of a heart murmur
5. Elevated systemic blood pressure

Family history

6. Premature death (sudden and unexplained or otherwise) before age 50 because of heart disease in 1 or more relative
7. Disability from heart disease in a close relative younger than 50
8. Specific knowledge of certain cardiac conditions in family members: HCM or dilated cardiomyopathy, long QT syndrome, other ion channelopathies, Marfan syndrome, or clinically important arrhythmias

Physical examination

9. Heart murmur (auscultation should be performed in supine and standing positions [or with Valsalva maneuver], specifically to identify murmurs of dynamic left ventricular outflow tract obstruction)
10. Femoral pulses to exclude aortic coarctation
11. Physical stigmata of Marfan syndrome
12. Brachial artery blood pressure (sitting position) preferably taken in both arms

The H&P has come under scrutiny for having low sensitivity for identifying athletes with cardiovascular risk factors. The most frequently reported causes of SCD in young athletes, HCM and anomalous origin of coronary artery, are generally asymptomatic and have normal findings on examination. The advantages of H&P include cost effectiveness, need for minimal resources, and efficiency in administration with reasonable sensitivity. Several studies have reported data substantiating the higher efficacy of ECG in identifying athletes with HCM, channelopathies, and other clinically silent cardiovascular diseases that increase the risk for SCD in young athletes.[18,20,21,25,28,39–44]

Electrocardiogram

The 12-lead ECG obtained at rest on a nontraining day has received a lot of attention as a screening tool consequent to the Italian experience showing marked decrease in SCD rates after making ECG part of standard screening.[44] The International Olympic Committee and the European Sports Council require an ECG before sports participation.[2,20,21] Japan has been subjecting all first, seventh, and tenth graders to an ECG since 1973.[40] An ECG is abnormal in 90% of patients with HCM.[1,44] Other ECG detectable diseases causing SCD include arrythmogenic right ventricular cardiomyopathy (ARVC), ion channelopathies, dilated cardiomyopathy, and Wolff-Parkinson-White syndrome (WPW).[44,45]

Corrado and colleagues[28] showed that they were able to identify 22 asymptomatic athletes (of 33,735 athletes screened) with HCM based on screening ECG, later

Box 3
Cardiovascular preparticipation screening history

Symptoms

Unusual (more than or different from others) fatigue associated with physical activities

Pain, discomfort, or feeling of pressure in chest during exercise

Presyncope or syncope (fainting) during or after exercise, emotion, or startle

Exercise-associated dizziness

Exercise-associated shortness of breath

Heart racing or skipping beats

Medical or personal history and review of systems

Unexplained seizures or seizure-like episodes

Unexplained episodes of exercise-induced asthma or asthma-like symptoms

Recent febrile illness

Detailed history of any congenital structural heart disease

Use of a cardiac pacemaker or implanted cardiac defibrillator

History of Kawasaki disease

History of rheumatic fever

Known heart murmur

Known high cholesterol or lipid disorder

Systemic hypertension

Diabetes mellitus

Thyroid disease

Any previous recommendations to restrict physical activity

Use of therapeutic medications

Use of dietary supplements or over-the-counter medications

Substance abuse or tobacco use

Use of excessive caffeine or energy drinks

Family history

Sudden or unexpected death of family members before age 50 (include deaths due to possible sudden infant death syndrome, automobile accident, or drowning)

Coronary artery disease before age 50

Family members using pacemaker or implanted cardiac defibrillator

Family history of congenital deafness

Family history of certain cardiovascular diseases, such as Marfan syndrome, cardiomyopathies, long QT syndrome, short QT syndrome, or Brugada syndrome

Family history of lipid disorders, diabetes mellitus, or systemic hypertension

Family history of primary pulmonary hypertension confirmed with an echocardiogram (ECHO). Eighty-two percent of the patients had abnormal ECG whereas 23% had some suggestions of underlying disease based on family history, a murmur, or both. In an extension of that, Corrado and colleagues[20,39] showed an 89% decrease in the incidence of SCD in athletes screened with ECG in Italy. A study of more than 5000 Nevada high school athletes showed the sensitivity of ECGs at 78% in detecting athletes at risk; this compares to 3% sensitivity for the H&P alone.[38] The specificity seems to increase when ECG changes considered grossly abnormal are pursued with additional studies.

In the United States, routine ECG is not currently recommended as part of cardiovascular screening of young asymptomatic athletes.[1,3] Reasons cited for not including ECG include cost, need for trained personnel, need for appropriate infrastructure, false-positive findings (in 10%–40%) warranting further unnecessary testing, and potential to cause anxiety in otherwise healthy athletes and their families.[1,3,4,46] Current recommendations include improving and standardizing the existing H&P forms, thus improving their yield, and creating a national data registry for reporting SCDs to better assess the epidemiology of SCD in the US athletic population. Because SCD is a rare event in young athletes, with a large number of athletes (5–10 million), it is estimated that it would cost $3.4 million to save 1 athlete's life from SCD and $330,000 to identify 1 athlete at risk for SCD.[1] The sensitivity of the screening ECG depends on appropriate interpretation by qualified professionals. The knowledge of specific ECG findings in healthy adolescents and ECG changes in athletes is imperative to determine what constitutes an abnormal and clinically significant change.[44,47–49]

There have been several questions raised regarding the Italian experience, which is cited most frequently as the basis for including ECG as a screening tool. One argument against that study is that it is an observational study and not a prospective study with a control population.[1] Also, the incidence rates that the Italians have managed to achieve after almost 25 years of ECG screening are not significantly different from the existing SCD rates in the United States.

Myerburg and Vetter[41] make several points in favor of including ECG for cardiovascular screening of competitive athletes. They contend that the false-positive rate of 10% to 25% cited in the AHA recommendations for abnormal findings on ECG is too high and according to their estimate it is approximately 4.8%.[41,42] Myerburg and Vetter[41] have suggested that all school athletes receive ECGs only twice during their school athletic career. This would mitigate the need for annual ECG for each athlete and significantly bring down the cost of such a program. With regards to personnel, the argument is that ancillary health staff with an interest in SCA should be recruited and trained to provide for the numbers required for a functional screening strategy that includes ECG. Fuller and colleagues,[38] based on their study of more than 5000 high school athletes, estimated that including an ECG in the screening protocol would cost $44,000 for each life saved. Fuller and colleagues[38] report that screening 700,000 high school athletes annually results in 1080 years of life gained when an ECG is used compared with 92 years of life gained when the AHA-recommended H&P alone is used. The average cost of screening in Japan is $8800 per life saved and in Italy a similar type of program cost $15,926 per life saved; both include screening ECG.[43,50] With government subsidy and more realistic cost assessment, it has been surmised that a screening program with ECG can be put in place at 20% to 25% of the cost presented in the AHA report.[1,41]

Currently, there is no consensus regarding whether or not routine ECG screening is prudent to apply to the US athlete population. The AHA report provides the framework for cardiovascular screening.[1,3] Large prospective studies are needed to evaluate the value of screening ECGs in the US population. In more recent publications, Maron and colleagues[1,6] have reviewed the recommendations for and against including an ECG in preparticipation screening. Maron and colleagues[1,6] note the potential usefulness of ECGs in identifying athletes at risk but also cite lack of infrastructure (including trained personnel) and the demographics of the athletic population as the principle barriers to implementing a large-scale screening program that includes ECG. Maron and colleagues[1,6,22] in their study comparing the rate of SCD in similar-sized populations in Minnesota, United States, and Veneto, Italy, reported that the rate of SCD was not significantly different between the 2 groups. The investigators, however, note that the rate of SCD has declined in Veneto, Italy, after implementation of an ECG-based screening program. In Italy, a specialist who has received training for 4 years administers the sports screening. Such a person is well trained in reading ECG of athletes, decreasing the false-positive rates and keeping the costs down. Based on many reports, including that of the AHA, inclusion of an ECG in routine screening of asymptomatic young athletes presents considerable economic and logistical challenges. Therefore, at present, ECG is indicated based on abnormal findings of H&P.

Physiology and cardiac remodeling

Knowledge of cardiovascular changes that result from regular exercise is essential in the interpretation of ECG changes in athletes.[44,51] The term, *athlete's heart*, refers to the changes that result from regular exercise, characterized by a benign increase in cardiac mass and specific circulatory and cardiac morphologic alterations that represent a physiologic adaptation to systematic training.[47,49,51–62] Endurance training (dynamic, isotonic, or aerobic), such as running or swimming, and resistance training (static, isometric, or anaerobic), such as weight training, result in different training effects on the cardiovascular system (**Table 1**).[1] In most sports, however, there is some overlap of endurance and resistance training. This is exemplified in sports, such as skiing, rowing, cycling, and triathlon. It is, therefore, expected that with regular exercise training there is some combination of cardiac chamber dilation and increase in wall thickness.[1]

The cardiac remodeling associated with regular exercise training in athletes is not a uniform phenomenon. Fifty percent of athletes may show some cardiac remodeling that includes 1 or all of the following: increase left ventricle, right ventricle, or left atrium chamber size. Ventricular wall thickness may or may not be increased. These changes are usually associated with normal systolic and diastolic function. Remodeling may occur rapidly or more gradually. Most of these changes are still within normal limits when compared with age- and gender-matched sedentary individuals.[47]

Table 1
Cardiovascular adaptation from training

Type of Training	Training Adaptations
Endurance	Increased oxygen use Decreased peripheral vascular resistance Increased stroke volume Increased cardiac output Volume overload–induced ventricular dilatation
Resistance	No change in oxygen use No change in cardiac output Increased peripheral vascular resistance Increased heart rate Pressure overload–induced ventricular hypertrophy

The degree of structural change in the heart may correlate with the type of sport that an athlete is involved in, but this is not proved conclusively.[48,49,52–54] The clinical variables that may play a role in influencing the degree of structural change in the heart include body size (50%), type of sport (14%), gender (7%), age (4%), and unknown factors (25%).[48–55] Genetics also plays a role and may be responsible for some of the unknown variables.[48–55]

Often the changes in ventricular chamber size and wall thickness seen in competitive athletes may mimic structural heart disease. A ventricular wall thickness between 13 and 15 mm and left ventricular end-diastolic size greater than or equal to 55 mm but less than or equal to 60 mm constitute the gray zone of overlap between physiologic change and structural heart disease.[1,3,48,55] In these cases, it is often difficult to make a decision whether or not to allow continued competitive sport participation. One option is to discontinue training and serially measure these variables.[27,48,62] Normalization would suggest physiologic change and an athlete might be allowed to resume competitive sport participation.[42,48,62]

Several changes are noted on an ECG due to cardiac remodeling with regular exercise training. Approximately 60% of athletes have a normal ECG whereas 15% may have ECG changes suggestive of an underlying heart disease.[44,47,48] The most commonly observed ECG changes in athletes include early repolarization, increased QRS voltages, diffuse T-wave inversion, and deep Q waves.[44,47] The most common rhythm abnormalities in athletes include bradycardia, first-degree heart block, and Wenckebach phenomenon.[44,47] Recent studies with Holter monitor have identified more complex ventricular arrhythmias, which are usually abolished on discontinuing training and may be of no clinical significance. These may be considered part of the spectrum that constitutes an athlete's heart.[44,47]

In most athletes, the changes in the heart that occur with regular training and conditioning regress with deconditioning; however, in 20% of these athletes, the changes may be permanent.[1,47] There is no evidence to show that the cardiac remodeling in an athlete has a disabling, permanent, or detrimental course.[47]

2-D ECHO

2-D ECHO is most useful for the diagnosis of structural heart disease.[45,63] Diastolic and systolic function, wall motion abnormalities, wall thickness, valve morphology, and internal chamber size can be assessed with confidence with ECHO. ECHO has also been shown to be useful in visualizing the origin of the coronary arteries.[64,65] Including contrast and tissue Doppler greatly increases the likelihood of diagnosing ARVC while using the ECHO.[63,64] Adding an ECHO to a cardiovascular screening program increases the likelihood of identifying structural abnormalities of the heart. The added cost of screening and the scarcity of resources, equipment, and personnel are significant barriers to recommend routine use of ECHO for screening. An ECHO can be considered a good confirmatory test after abnormal findings on H&P or ECG.

Genetic Testing

There have been significant advances in the identification of genetic causes for cardiovascular diseases,[66–69] leading to the question of present or future methods involving identification of genetic risk factors for cardiovascular disease using genetic testing as a screening tool. Even though progress has been made in identification of these genetic mutations, the genotype-phenotype correlation is still poor. It cannot be reliably predicted if an identified mutation will eventually lead to an expression of disease. This may lead to a high false-positive rate. With more mutations identified, the battery of tests is ever-increasing and to apply the entire set to an athlete is not prudent, which leads back to the need for a good H&P as an initial screening tool with emphasis on family history to identify individuals at risk. Once a proband is identified, an entire family can be screened for that specific mutation that has been identified.[64,68]

Other Tests

Exercise stress testing is of limited value as a screening tool but has a role in risk stratification.[45] Exercise stress testing has been recommended in athletes older than 35. This is also part of the extended Italian recommendation for athletes older than 35.[2] Maximal exercise testing is an integral component to unmask arrhythmia in ARVC, familial catecholaminergic polymorphic ventricular tachycardia, and long QT syndrome. Frequent ventricular extrasystoles during exercise indicate underlying cardiac disease.[45]

Several other tests are available for identification of certain specific conditions.[45,70] These include Holter monitoring (quantification of extrasystoles and identification of tachy- and bradyarrythmias), cardiac MRI scan (ARVC, HCM, and myocarditis), tissue Doppler (ARVC), sodium channel blocker challenge (Brugada syndrome), and adenosine challenge for pre-excitation (familial risk of WPW syndrome and supraventricular tachycardia). These tests are not appropriate for mass screening due to low yield and also due to the need for expertise to successfully obtain and interpret the results. They become important after screening has raised concerns for specific problems.

SUMMARY

Identification of young athletes at risk for an adverse cardiovascular event during sport participation is a challenging task. Even in the best of circumstances, outcome of SCA on the field remains poor; therefore, prevention is critical. Cardiovascular screening of young athletes is based on ascertaining specific history and a thorough physical examination. Although inclusion of an ECG in screening asymptomatic athletes has been shown to increase the likelihood of identifying athletes at risk, considerable logistical and economic challenges remain, including using ECG as a screening tool. Even the most ideal screening strategy may not identify all athletes at risk for SCA on the field, and unfortunately SCD may be the first clinical manifestation of some cardiac conditions.

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