Detection and Management of Heart Disease in Athletes

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KEYWORDS

• Athlete • Cardiovascular disease • Electrocardiogram • Sudden cardiac arrest
• Preparticipation physical evaluation (PPE) • Athlete's heart • International Criteria
• Sports cardiology

KEY POINTS

- Cardiovascular disease encompasses a heterogeneous group of primary structural and electrical disorders in young athletes and chiefly coronary artery disease in older athletes.
- Sudden cardiac arrest may be the initial presentation of disease in both groups and occurs more frequently in young athletes than historical estimates would indicate.
- The traditional preparticipation evaluation is limited in its ability to detect underlying cardiovascular disease in young athletes.
- The 12-lead electrocardiogram is a more sensitive tool than the traditional preparticipation evaluation for raising suspicion of occult conditions, but its optimal implementation into screening programs remains an area of intense research and debate.
- Contemporary risk stratification and treatment guidelines may allow for safe return to sport on a case-by-case basis, with specialist consultation.

INTRODUCTION

Cardiovascular disease (CVD) remains the leading cause of death in the United States.[1] Although this statistic is perhaps not surprising in older patients with classic risk factors, such as hypertension or dyslipidemia, sudden cardiac arrest (SCA) also accounts for the majority of exercise-related deaths in young athletes.[2,3] The burden of CVD, in addition to widespread agreement that physical activity promotes and maintains cardiovascular health,[4,5] uniquely places the primary care clinician in a critical role for screening, evaluation, and management of a variety of patients and conditions to foster cardiovascular health.

Given the increasing emphasis on the benefits of sports and exercise in all ages and walks of life, the responsibilities of the primary care provider may include

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- Conducting preparticipation cardiovascular screening in pediatric athletes
- Performing the initial evaluation and management of suspected CVD in the young athlete
- Managing risk reduction in the active or newly active adult with or without diagnosed CVD

From elite athletes to weekend warriors, primary care providers have a unique opportunity to help patients of all ages engage in safe exercise and maintain an active lifestyle. This article provides a contemporary review of the epidemiology, detection, and management of CVD in athletes.

ETIOLOGY OF SUDDEN CARDIAC ARREST AND DEATH IN YOUNG COMPETITIVE ATHLETES

Overview

The diseases and conditions associated with SCA and sudden cardiac death (SCD) in the young athlete (<35 years old) comprise a heterogeneous group of congenital or acquired structural and electrical disorders. These include primary cardiomyopathies, coronary artery anomalies, channelopathies, valvular disorders, and diseases of ventricular preexcitation, among others.[2,3,6,7] Hypertrophic cardiomyopathy (HCM) is classically considered the most frequent cause of SCD in athletes, having been identified as the underlying etiology in as many as 36% of cases, a rate more than twice as frequent as the second leading cause of anomalous coronary arteries (17%).[3] More recent research, however, has identified more than 15 different etiologies of SCA/SCD in United States athletes, with HCM (16.2%), coronary artery anomalies (13.7%), and idiopathic left ventricular hypertrophy (11.1%), representing relatively smaller proportions of a diverse group of conditions[6] (**Table 1**). HCM was identified more frequently as the underlying etiology in African American athletes compared with white athletes ($P<.05$).[6] Autopsy-negative sudden unexplained death (AN-SUD), implying primary electrical disease that could not be identified postmortem, also is a common finding in SCD cases.[2] AN-SUD has been found to be the leading cause of SCA/SCD in some international studies,[8,9] whereas cardiomyopathies, such as HCM, are the leading causes in others.[10,11] Variable study populations, differing postmortem examination criteria, and lack of a standardized methodology of case identification from region to region or country to country may account for discrepancies between studies.

Certain conditions merit special attention as the most common causes of SCA/SCD that may be encountered in primary care practices.

Hypertrophic Cardiomyopathy

HCM is a condition characterized by increased left ventricular wall thickness that is usually asymmetric and involves the interventricular septum. HCM affects as many as 1 in 500 people worldwide, with more than 1400 different genetic mutations described. Abnormal wall thickness combined with systolic anterior motion of the mitral valve can lead to left ventricular outflow tract obstruction and potential symptoms of exertional dyspnea, chest pain, or syncope, but a majority of cases in young athletes are asymptomatic prior to the sentinel event of SCA. SCA stems from a disorganized myocardial architecture and arrhythmogenic foci of interstitial fibrosis.[12]

A resting 12-lead electrocardiogram (ECG) is abnormal in as many as 96% of cases of HCM.[13] Classic ECG findings suggestive of HCM include T-wave inversions (TWIs) in the lateral or inferolateral leads with concurrent ST-segment depressions (**Fig. 1**). These ECG findings are highly suggestive of cardiomyopathy and must not be

Table 1
Etiology of Sudden Cardiac Arrest and Death in Competitive Athletes: July 1, 2014, to June 30, 2016 (n = 117[a])

	Sudden Cardiac Arrest (n = 34), N (%)	Sudden Cardiac Death (n = 83), N (%)	Total, N (%)
HCM	4 (11.8)	15 (18.1)	19 (16.2)
Coronary artery anomalies	3 (8.8)	13 (15.7)	16 (13.7)
Anomalous origin LCA	—	10	—
Agenesis of LCA ostium	—	1	—
Anomalous RCA with aberrant takeoff and hypoplasia	—	1	—
Myocardial bridging	—	1	—
Idiopathic left ventricular hypertrophy/possible cardiomyopathy	—	13 (15.7)	13 (11.1)
AN-SUD	—	8 (9.6)	8 (6.8)
WPW	6 (17.6)	2 (2.4)	8 (6.8)
LQTS	4 (11.8)	3 (3.6)	7 (6.0)
Arrhythmogenic cardiomyopathy	2 (5.9)	4 (4.8)	6 (5.1)
Dilated cardiomyopathy	2 (5.9)	4 (4.8)	6 (5.1)
Aortic dissection/rupture	0 (0)	5 (6.0)	5 (4.3)
Associated bicuspid aortic valve	—	2	—
Associated Marfan syndrome	—	1	—
Myocarditis	2 (5.9)	3 (3.6)	5 (4.3)
Complications of a congenital heart defect	1 (2.9)	3 (3.6)	4 (3.4)
Ebstein anomaly	—	1	—
Left ventricular hypertrophy associated with congenital aortic stenosis	—	1	—
Transposition of the great arteries	—	1	—
Tetralogy of Fallot	1	—	—
Coronary atherosclerosis	2 (5.9)	2 (2.4)	4 (3.4)
Valvular disorder	1 (2.9)	2 (2.4)	3 (2.6)
Commotio cordis	3 (8.8)	0 (0)	3 (2.6)
Catecholaminergic polymorphic ventricular tachycardia	2 (5.9)	0 (0)	2 (1.7)
Hypertensive heart disease	0(0)	2 (2.4)	2 (1.7)
Restrictive cardiomyopathy	1 (2.9)	0 (0)	1 (0.9)
Left ventricular noncompaction	0 (0)	1 (1.2)	1 (0.9)
Fibromuscular dysplasia of the sinoatrial nodal artery	0 (0)	1 (1.2)	1 (0.9)
Paroxysmal atrial tachycardia	0 (0)	1 (1.2)	1 (0.9)
Right atrial myxoma	0 (0)	1 (1.2)	1 (0.9)
Pericarditis	1 (2.9)	0 (0)	1 (0.9)

[a] One hundred seventeen (65.4%) cases had a reported or adjudicated diagnosis.

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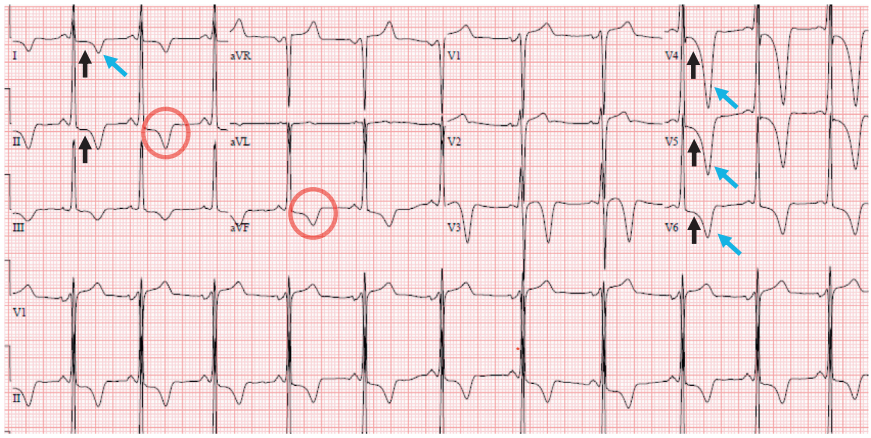

Fig. 1. Abnormal ECG findings, notably lateral lead TWIs (*blue arrows*), inferior lead TWIs (*red circles*), and lateral lead ST segment depressions (*black arrows*), that are suggestive of HCM and thus require further investigation.

discounted without appropriate follow-up testing and consultation.[14] Conversely, training-related left ventricular hypertrophy from athlete's heart does not exhibit lateral TWIs, and voltage criteria for left ventricular hypertrophy are a common finding in athletes (**Fig. 2**). Isolated left ventricular hypertrophy on ECG, when found in isolation and without other ECG abnormalities, symptoms, or family history concerns, is not a distinguishing finding for pathology and is considered a normal finding in trained athletes.[14]

Transthoracic echocardiogram (TTE) may be sufficient to make an initial HCM diagnosis, although a negative TTE does not exclude HCM involving the anterolateral wall or apex, especially when clinical suspicion is high or when markedly abnormal ECG findings are identified (see **Fig. 1**). In this case, cardiac magnetic resonance imaging (MRI) should be a routine test in the evaluation of athletes with lateral or inferolateral

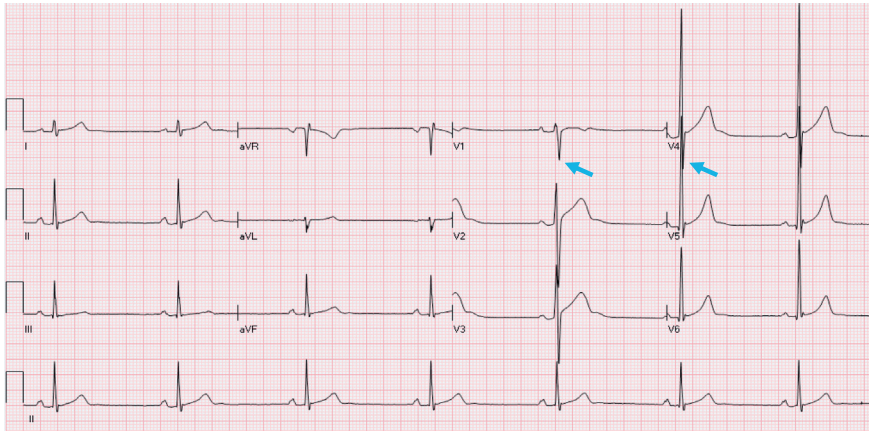

Fig. 2. Isolated voltage criteria for left ventricular hypertrophy (LVH), as evidenced by a sum of the S wave in V1 and R wave in V5 of greater than 35 mm (*blue arrows*). Note the lack of TWIs or ST depressions. LVH in isolation is considered a normal variant in trained athletes.

TWIs to exclude apical HCM, arrhythmogenic cardiomyopathy with left ventricular involvement, or nonischemic left ventricular scar.[14]

If diagnosed, management of HCM should include close consultation with a cardiologist, preferably one with experience in cardiomyopathy or sports cardiology. The athlete should be restricted from strenuous exercise and competitive sports until further work-up can be completed and the optimal care plan determined. Additional risk stratification includes assessment with exercise stress testing and 24-hour ambulatory ECG monitoring. Family screening and genetic testing also should be considered.[12] Based on current guidelines by the American Heart Association (AHA) and the American College of Cardiology (ACC), probable or unequivocal clinical expression of any degree or variety of HCM is considered a contraindication to all but low-intensity competitive sports,[15] and morbidity-reducing treatment should be pursued via specialist consultation.[16] Treatment may include pharmacologic management, implantable cardioverter-defibrillator (ICD) placement, or other procedural interventions, such as surgical myomectomy.[12] Regular specialist follow-up also can inform individual exercise regimens for fitness purposes on a case-by-case basis.[12,16]

Anomalous Coronary Arteries

Coronary artery anomalies are another leading cause of SCA/SCD in athletes. Anomalies include the left coronary artery (LCA) arising from the right sinus of Valsalva or the right coronary artery (RCA) arising from the left sinus of Valsalva. Studies suggest that fewer than 50% of athletes with SCA from an anomalous coronary artery had preexisting warning symptoms of their condition, such as exertional chest pain or syncope.[17]

Coronary artery anomalies appear to precipitate SCA/SCD as a consequence of ischemic changes arising from abnormal artery positioning or formation. For example, an anomalous LCA may be compressed by its intramural course within the wall of the aorta or during exercise as it travels between the aorta and pulmonary arteries, leading to repeat bouts of transient ischemia. These bouts may promote myocardial fibrosis, which may predispose to ventricular arrhythmias.[17]

Anomalous coronary arteries are among the more difficult conditions to detect in their preclinical state.[18] Resting and stress ECGs are frequently normal.[17,19] A high clinical suspicion must be maintained in the athlete with exertional chest pain or syncope. The coronary arteries can be satisfactorily assessed in more than 90% of athletes with a focused TTE,[20] suggesting that if they cannot be visualized in an athlete with unexplained cardiovascular symptoms, computed tomography (CT) angiography or cardiac MRI should be considered.[17]

The treatment of coronary artery anomalies is surgical. According to the AHA, athletes with an anomalous origin of the RCA may participate in sports after counseling, provided an athlete is asymptomatic with a normal exercise stress test. An anomalous LCA is a higher-risk lesion. Competitive sports participation should be restricted until at least 3 months after surgical correction of the lesion, assuming the athlete is asymptomatic with no evidence of ischemia or arrhythmia demonstrated on exercise stress testing.[21]

Wolff-Parkinson-White

Wolff-Parkinson-White (WPW) is a congenital condition characterized by 1 or more accessory conduction pathways within the heart. Classic ECG findings include a short PR interval and the presence of a delta wave (slurred QRS upstroke), signifying ventricular preexcitation (**Fig. 3**). Many patients with WPW are asymptomatic, whereas others may report symptoms suggesting arrhythmias, such as palpitations.[22] WPW

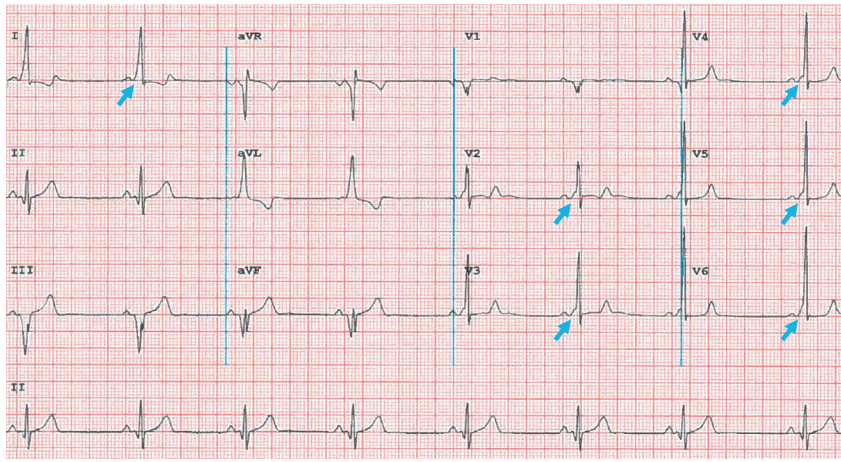

Fig. 3. ECG of a patient with WPW. Note the delta waves with short PR intervals (*blue arrows*). Other WPW findings include large Q waves in lead III and the lack of a Q wave in lead V6.

also is a known cause of SCA/SCD, accounting for 6.8% of all cases in a recent study.[6] SCA/SCD from WPW is thought to arise from the rapid conduction of atrial fibrillation across the accessory pathway, bypassing the rate-controlling atrioventricular node and thus precipitating ventricular fibrillation.[22]

Intermittent ventricular preexcitation, characterized by periodic loss of the delta wave on ECG monitoring, is considered a low-risk pathway that is unlikely to precipitate malignant arrhythmias. Persistent delta waves at rest merit further evaluation, including echocardiography, to assess for structural abnormalities associated with WPW, such as cardiomyopathies or Ebstein anomaly.[22] Exercise stress testing should be used to assess for the abrupt loss of the delta wave with exercise. Athletes who demonstrate persistent delta waves with exercise, or those who participate in moderate-intensity or high-intensity sports, are considered higher risk athletes. Such athletes should be offered consultation with an electrophysiologist for consideration of an electrophysiology study to identify high-risk pathways that may be amenable to ablation.[22,23]

Long QT Syndrome

Long QT syndrome (LQTS) is a genetic ion channelopathy that yields a prolonged QT interval on ECG and may precipitate polymorphic ventricular tachycardia, also known as torsades de pointes.[24] A prolonged QT interval in athletes is defined as a QT interval corrected for heart rate (QTc) using Bazett formula of greater than or equal to 470 ms in male athletes and greater than or equal to 480 ms in female athletes[14] (**Fig. 4**). Importantly, a single ECG with a prolonged QT interval does not equate to a diagnosis of LQTS, which requires confirmation by genetic testing, family screening, and/or paradoxic prolongation of the QT interval during the recovery phase of an exercise stress test. QT prolonging medications or electrolyte abnormalities also should be excluded. Diagnostic criteria have been recommended by the Heart Rhythm Society, the European Heart Rhythm Association, and the Asia Pacific Heart Rhythm Society. These societies recommend a formal diagnosis of LQTS with a Schwartz criteria[25] score of greater than or equal to 3.5, a confirmed LQTS genetic mutation, a QTc of greater

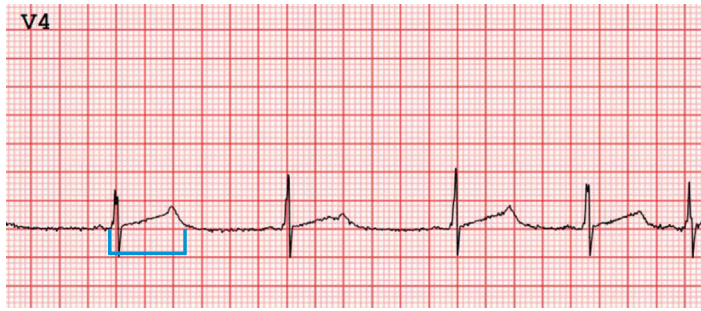

Fig. 4. LQTS. Precordial lead V4 showing a prolonged QT interval (*bracket*).

than or equal to 500 ms, or a QTc between 480 ms and 499 ms with suggestive symptoms, such as unexplained syncope.[26]

Ventricular arrhythmias may be provoked by extreme emotional stress or physical activity, especially swimming, in the setting of LQTS type I and can lead to syncope with seizure-like activity or sudden death.[24] Previously considered a contraindication to competitive sports,[27] recent observational data suggest that athletes with LQTS who undergo optimal medical management and counseling may not be at as high of risk of SCA/SCD as previously postulated.[28–30]

Updated guidelines for the management of LQTS in athletes have been published.[31,32] The medical management of LQTS includes β-blockade in most patients, including those who are asymptomatic, as well as avoiding medications that prolong the QT interval.[26] β-blockers are prohibited in certain competitive sports by the World Anti-Doping Agency.[33] ICD placement should be considered in high-risk athletes, such as those who have survived prior SCA.[26,31] Athletes with ICDs in place may consider returning to competitive sports under contemporary recommendations provided no shocks have been delivered to abort ventricular arrhythmias for at least 3 months. According to the AHA and ACC, however, "the desire of the athlete to continue athletic competition should not represent the primary indication for use of an ICD".[34]

Other Etiologies

SCA/SCD in athletes has been attributed to several other etiologies. Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a condition characterized by the progressive replacement of right ventricular myocardium by arrhythmia-promoting fibrofatty tissue.[35] Responsible for 5% of SCA/SCD in athletes in 1 study,[6] ARVC is suggested by TWIs in the anterior precordial leads (V1-V4) on ECG.[35] Given the difficulty in confirming a diagnosis, formal diagnostic criteria have been proposed.[36,37] Treatment options include β-blockade to prevent SCA and ICD implantation in high-risk patients or those who have survived aborted SCA. In patients with manifestations of ARVC-driven congestive heart failure, appropriate medical therapy should be instituted.[35] The AHA and ACC recommend restriction from competitive sports, except for possibly low-intensity sports, in all patients with possible or confirmed ARVC.[15]

Dilated cardiomyopathy causes approximately 5% of athlete SCA/SCD cases,[6] and the AHA and ACC recommend restricting symptomatic athletes from competition after diagnosis.[15] Other documented SCA/SCD etiologies include aortic dissections or rupture, myocarditis, restrictive cardiomyopathy, catecholaminergic polymorphic ventricular tachycardia, and left ventricular noncompaction cardiomyopathy.[38] SCA/SCD

related to commotio cordis, characterized by SCA after direct impact of the chest wall by a blunt object, represents another 3% of cases.[6]

INCIDENCE OF SUDDEN CARDIAC ARREST/SUDDEN CARDIAC DEATH IN THE YOUNG COMPETITIVE ATHLETE

The central goal of an effective screening protocol is to prevent the progression of preclinical disease to its clinical state. In the case of CVD in young athletes, SCA can be the initial manifestation of disease,[7,39] making the preclinical detection of pathologic conditions of paramount importance. Epidemiologic data play a crucial role in determining the appropriateness of screening and informing the optimal methods for early detection.

The annual incidence of SCA/SCD in young competitive athletes varies considerably in the published literature. An annual SCD risk of 1:200,000 in high school athletes was proposed as an estimate in the late twentieth century,[40] with rates as low as 1:417,000 also reported.[41] Contemporary research with more extensive methodology, however, has concluded that the risk is substantially higher, with minimum annual risk estimates of 1:50,000 in college athletes and 1:80,000 in high school athletes.[42] The discrepancy between historical and modern incidence estimates has been attributed primarily to research methodology differences, difficulties in identifying cases of SCD, and year-to-year variability.[43] Studies also vary regarding the inclusion of SCA with survival, as opposed to exclusively cases of SCD. Given that exercise-related SCA in competitive athletes carries a modern survival rate of 48%,[44] all major cardiovascular events, including both deaths and survivals, are important to consider when informing optimal screening protocols.

A differential risk among athletes also is present, with some athlete subgroups at considerably higher risk than others. For instance, male and African American athletes are consistently at 3-times to 5-times higher risk of SCA/SCD.[2,3,42,45] Certain sports also show a disproportionately higher risk, with more than 50% of SCA/SCD cases occurring in football and basketball.[6] Most strikingly, male Division I college basketball players have an annual SCD risk of 1:5,000.[2]

THE YOUNG ATHLETE PREPARTICIPATION PHYSICAL EVALUATION

The preparticipation physical evaluation (PPE), or sports physical, is a common encounter in the primary care office. Endorsed by numerous medical societies,[46–48] the chief goals of the PPE, as defined by the American Academy of Pediatrics *Preparticipation Physical Evaluation*, are to

1. Screen for conditions that may be life-threatening or disabling
2. Screen for conditions that may predispose to injury or illness
3. Determine general health
4. Serve as an entry point to the health care system for adolescents
5. Provide an opportunity to initiate discussion on health-related topics[42]

Although exercise has numerous health benefits, exercise can trigger SCA in athletes with an underlying heart disorder.[49] Thus, a crucial and widely practiced component of the PPE is to screen for silent CVD that may place an athlete at risk of SCA/SCD. Traditionally, preparticipation cardiovascular screening includes a standardized symptom and family history questionnaire and a targeted cardiovascular physical examination.[46,47] Positive responses to questions about symptoms, such as exertional chest pain or syncope, the identification of a heart murmur on physical examination, or a significant family history of CVD or sudden death at a young age, constitute a

positive screen and require additional investigation to exclude the presence of undiagnosed CVD.

Despite widespread endorsement and utilization of a screening history and physical examination (H&P), no outcomes data support its ability to reduce cardiovascular morbidity and mortality. In an evidence-based consensus statement, the American Medical Society for Sports Medicine (AMSSM) has recognized the limited utility of the H&P alone to detect occult CVD in young athletes.[48] Limitations of the H&P include its general, nonspecific symptom questions, yielding a high rate of positive responses, and its reliance on accurate and truthful reporting by the athlete.[50] Positive response rates to at least 1 symptom or family history question have been reported in 30% to 36% of high school and college athletes.[51–54]

Although history questionnaires yield a high number of positive responses that can be difficult to clarify, certain H&P clues must never be discounted. A history of exercise-related syncope, specifically collapse during exercise, requires a comprehensive investigation to rule out a cardiac cause. Additionally, a family history of a cardiovascular disorder or sudden death at a young age (<40 years) is suggestive of a possible heritable cardiovascular condition. Physical stigmata of Marfan syndrome or heart murmurs that suggest left ventricular outflow tract obstruction, such as in HCM, also should be carefully evaluated.[46]

THE ELECTROCARDIOGRAM AS A SCREENING TOOL

Conflicting Recommendations and Controversy

Due to the limited efficacy of the H&P in detecting CVD, as well as the growing appreciation of the higher rates of SCA/SCD in competitive athletes, the ECG has garnered interest as a tool to improve the sensitivity of cardiovascular screening. The merits and proposed methods of implementing the ECG as a screening mechanism remain a subject of considerable debate. The European Society of Cardiology (ESC) recommends the routine use of the ECG during cardiovascular preparticipation screening,[55] whereas the AHA does not support ECG screening.[47,56] The AMSSM suggests that ECG screening be considered in higher-risk athletes when accurate ECG interpretation and proper cardiology resources are available.[48] Multiple studies demonstrate that the ECG substantially increases the sensitivity to detect conditions associated with SCA/SCD compared with H&P alone.[51,54,57–59] In a meta-analysis of 15 studies and 47,137 athletes undergoing cardiovascular screening, pooled sensitivities of the screening history, physical examination, and ECG were 20%, 9%, and 94%, respectively.[60]

The Athlete's Heart and Modern Electrocardiogram Interpretation Criteria

It is well established that the hearts of highly trained athletes undergo physiologic adaptations due to increased workloads and demand. These changes include ventricular wall hypertrophy and increased vagal tone, known as the athlete's heart.[61] These physiologic adaptations can lead to distinct changes on the ECG that can be misinterpreted as pathologic findings by the untrained clinician.[14] The risk of unnecessary restriction of an athlete from participation as a result of misinterpretation, as well as unnecessary secondary testing, is a major criticism of using the ECG as a screening tool.

In order to better distinguish pathologic, disease-specific changes on the ECG from physiologic, athlete's heart changes, athlete-specific ECG criteria have been formulated. The first such criteria were published in 2010 by the ESC.[62] Further iterations have followed, including criteria from Stanford University (2011),[63] the Seattle Criteria (2013),[64] the Refined Criteria (2014),[65] and, most recently, the International Criteria

(2017).[14] Each iteration of ECG interpretation criteria has lowered the false-positive rate, or the identification of ECG abnormalities without subsequent diagnosis of disease, without compromising sensitivity to identify underlying cardiac pathology.[66–70] ECG screening by experienced clinicians using athlete-specific standards typically produces false-positive rates of less than 3% to 5%.[51,53] The International Criteria are the current standard for ECG interpretation in athletes,[14] and open access to ECG training modules based on the International Criteria are available at https://uwsportscardiology.org/e-academy/[71]

Future Screening Directions and Considerations

Screening for any disease by any method carries numerous goals, chiefly

- The detection of a disease in its preclinical course with a high degree of accuracy
- The ability to intervene in order to prevent progression to its clinical state

Although certain H&P findings may suggest the underlying presence of CVD,[46] the exclusive reliance on the H&P for cardiovascular preparticipation screening is considered limited in its ability to detect CVD that places an athlete at elevated risk of SCA/SCD.[48] The ECG provides a more sensitive screening tool, but skilled sports cardiology infrastructure to conduct accurate interpretation and the appropriate secondary investigations for abnormal ECGs remains limited in the United States. Given the benefits of exercise in young athletes and nonathletes, minimizing unnecessary restriction from participation must also remain a priority, and universal ECG screening of young patients prior to exercise is not recommended by any major medical society. Additional efforts in education and training are needed to provide more effective screening, especially of high-risk athlete subgroups. When deciding on screening methods, consideration of the available sports cardiology resources as well as the risk of an individual athlete based on sport, race, and sex, rather than utilizing an all-or-none approach, is a growing area of interest and may provide a more effective approach.[48,72] Although the ECG is a superior screening tool for the detection of disease, the efficacy of the screening ECG in preventing cardiovascular mortality remains unclear.

HEART DISEASE IN THE ADULT ATHLETE

The cardiovascular care of older athletes (>35 year old) centers on the higher prevalence of atherosclerotic coronary artery disease (CAD) and the potential for exercise-induced acute coronary syndromes stemming from atherosclerotic plaque disruption with thrombosis or fixed stenosis.[73,74] The clinical presentation of these syndromes includes acute myocardial infarction and SCA/SCD, with no preceding symptoms or warning signs in approximately 50% of cases.[75,76] In endurance athletes, oxygen supply and demand mismatch also can precipitate ischemia, infarction, or SCA secondary to a fixed stenosis from a stable plaque (so-called demand ischemia).[77]

Screening in asymptomatic older athletes should focus on CAD risk factor identification and modification and can be carried out well in the primary care setting. Exercise stress testing in asymptomatic, low-risk adults is not recommended by the AHA due to poor predictive values.[78] Furthermore, the American College of Sports Medicine (ACSM) does not recommend the need for specific medical clearance for older athletes who are already participating in moderate to vigorous intensity exercise greater than or equal to 3 days per week, are asymptomatic, and do not carry diagnoses of known cardiovascular, metabolic, or renal disease.[79] Exercise stress testing in

adults with 1 or more risk factors for CAD, however, has been shown to have better predictive value and may help inform risk factor modification and interventions.[78] The AHA recommends exercise stress testing prior to the initiation of a vigorous exercise program in men over the age of 45, women over the age of 55, patients with diabetes, and patients with CAD risk factors.[78] The ESC recommends routine exercise stress testing in patients deemed at high risk of CAD,[80] and the ACSM recommends exercise stress testing in high-risk patients prior to the initiation of moderate-intensity or vigorous-intensity exercise programs.[79]

Coronary artery calcium (CAC) scoring by CT shows significant promise in identifying subclinical CAD. Elevated CAC scores are strongly associated with future risk of a cardiovascular event, independent of classic CAD risk factors or symptoms.[81–84] CAC scoring may be appropriate to further risk-stratify patients found to be at intermediate risk of CAD based on standard risk calculators and better inform the selection of candidates for statin therapy.[81,85,86] In 1 study, asymptomatic patients with moderate (100–400) to high ($\geq$400) CAC scores without previously known CAD and who were treated with statin therapy had a lower risk of major cardiovascular events ($P<.05$).[87]

SUMMARY

Heart disease in athletes encompasses a wide variety of conditions and diseases, both congenital and acquired. In the young athlete, the large majority of cases are primary structural or electrical disorders, with SCA/SCD being the most feared, visible, and tragic clinical manifestation. SCA/SCD in young athletes is more common than historically appreciated and can be precipitated in those with any 1 of several conditions. The traditional PPE consists of a screening H&P and is limited in its ability to accurately raise suspicion of underlying CVD. Nevertheless, certain historical clues, especially exertional chest pain or sudden unexplained syncope, should not be discounted and instead prompt further investigations to rule out underlying CVD. ECG screening provides a more sensitive method for the detection of athletes at risk of SCA/SCD but requires a trained sports cardiology infrastructure to conduct accurately. Risk stratification and management strategies for many of the common disorders linked to young athlete SCA/SCD have evolved and in some circumstances allow a return to competitive sport.

In masters athletes, prevention of acute coronary syndromes and SCA precipitated by preexisting CAD is the primary objective. Exercise stress testing in low-risk, asymptomatic patients is not recommended but may be used to guide those with documented risk factors. CAC scoring represents a newer technique to quantify the burden of CAD to risk-stratify patients and may help improve patient-centered outcomes.

DISCLOSURE

The authors have nothing to disclose.

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