Cardiovascular disease in athletes

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Cardiovascular disease (CVD) represents the leading cause of death in the United States. In recent years, this has been reflected by an increased public awareness regarding the importance of regular physical activity in preventing CVD and death. Because athletes, in general, are considered among the most physically fit individuals, the occurrence of sudden death due to CVD in this population is quite perplexing to many.

Regular intense physical activity can cause physiologic changes to the cardiovascular (CV) system that may mimic known CVD processes. Given this, the screening of athletes for conditions that may put them at increased risk for sudden cardiac death (SCD) can be somewhat challenging. This article focuses on this problem, by discussing several CV topics in athletes including: the athlete’s heart, SCD and associated CV conditions in athletes, and preparticipation screening. Because the 26th Bethesda Conference has previously published recommendations on determining eligibility for competition in athletes with known CV abnormalities [1–7], we also review these recommendations as they relate to individual disease processes.

The athlete’s heart

In the course of systematic physical training, the heart of a well-conditioned athlete undergoes structural changes that frequently distinguish it from that of a normal individual. Commonly described as an “athlete’s heart,” these changes are often seen echocardiographically, and may include a larger left-ventricular (LV) wall thickness and end-diastolic dimension, but with preserved systolic and...
diastolic function. For unclear reasons, these changes are less pronounced in female athletes [8].

The duration and type of exercise training also appear to affect the degree and type of cardiac changes that an athlete may experience (Table 1). For example, short-term training is not associated with a change in cardiac dimension, whereas prolonged endurance training is commonly followed by LV enlargement. High dynamic exercises (eg, running) are more likely to result in an increase in absolute LV mass and chamber size (eccentric hypertrophy), whereas high static exercises (eg, weight lifting) tend to increase LV mass without increasing chamber size (concentric hypertrophy). Among athletes who participate in sports with both high dynamic and high static demands (eg, rowing, cycling), eccentric and concentric hypertrophy may be frequently seen [1,9,10].

The CV examination of an athlete may be quite distinctive. Pulse rates as low as 30 to 40 beats per minute are frequently noted and usually reflect increased vagal tone. A slightly displaced apical impulse, an atrial or ventricular gallop, and a systolic regurgitant murmur may be noted as well. Electrocardiographic (ECG)

**Table 1**

<table>
<thead>
<tr>
<th>Athlete’ heart and type of exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dynamic</td>
</tr>
<tr>
<td>Static</td>
</tr>
<tr>
<td>Alternate terms</td>
</tr>
<tr>
<td>Isotonic</td>
</tr>
<tr>
<td>Isometric</td>
</tr>
<tr>
<td>Examples</td>
</tr>
<tr>
<td>Running</td>
</tr>
<tr>
<td>Weight lifting</td>
</tr>
<tr>
<td>LV hypertrophy</td>
</tr>
<tr>
<td>Eccentric</td>
</tr>
<tr>
<td>Concentric</td>
</tr>
<tr>
<td>Volume overload</td>
</tr>
<tr>
<td>Yes</td>
</tr>
<tr>
<td>No</td>
</tr>
<tr>
<td>Pressure overload</td>
</tr>
<tr>
<td>No</td>
</tr>
<tr>
<td>Yes</td>
</tr>
<tr>
<td>Oxygen uptake</td>
</tr>
<tr>
<td>Significant</td>
</tr>
<tr>
<td>Mild</td>
</tr>
<tr>
<td>Peripheral resistance</td>
</tr>
<tr>
<td>Decreases</td>
</tr>
<tr>
<td>Increases</td>
</tr>
<tr>
<td>Blood pressure</td>
</tr>
<tr>
<td>Decreases</td>
</tr>
<tr>
<td>Increases</td>
</tr>
<tr>
<td>LV wall thickness/LV radius</td>
</tr>
<tr>
<td>Unchanged</td>
</tr>
<tr>
<td>Increased</td>
</tr>
</tbody>
</table>

*Abbreviations: LV, left ventricle.*

**Box 1. ECG patterns in athletes**

- **Sinus bradycardia**
- **Sinus pauses**
- **Sinus arrhythmia**
- **Atrial or ventricular premature beats**
  - AV blocks
  - First degree
  - Type 1, second degree
  - Advanced AV block
- **Voltage criteria of right/left ventricular hypertrophy**
- **ST elevations**
- **T-wave changes**
abnormalities may be seen in up to 40% of competitive athletes and likely result from electrophysiologic remodeling associated with physical training [11]. These are listed in Box 1.

Because of the electrocardiographic and echocardiographic changes seen in athlete’s heart, differentiating these physiologic changes from pathological conditions can be quite difficult. An abnormal ECG in an athlete may be difficult to distinguish from one seen in the CV diseases of hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy (DCM), and myocarditis [12]. In addition, an athlete with a left-ventricular wall thickness of 13 mm to 15 mm may be difficult to distinguish from a similar individual with early HCM. In light of the similarities between physiologic changes that occur in athlete’s heart and pathologic findings seen in some CV disorders, the detection of structural or electrical cardiac abnormalities in athletes should not be taken lightly.

Sudden death in athletes

Although sudden death in athletes occurs rarely, the deaths of high-profile athletes such as Reggie Lewis, Hank Gathers, and others have attracted unusual public and media attention [13]. Numerous cases have required legal action to address the issue of disqualification from competition, an issue complicated by the difficulties in appropriately identifying athletes at risk of SCD. In fact, the cases of Nicholas Knapp and Stephen Larkan involved this very issue after both were suspected of having HCM [14].

The frequency of SCD in athletes is not precisely known. Proposed estimates vary greatly and depend largely on the age of the athlete, the sampling population, the sport, and the way in which SCD was defined (Table 2) [15–18]. In general, the incidence of SCD is more common in male athletes and increases with exercise intensity.

Numerous causes of SCD in athletes have been described. Although non-cardiovascular causes exist—including heat stroke, sickle cell crisis, ruptured cerebral aneurysms, and exacerbations of bronchial asthma—CV causes, as listed in Box 2, appear to predominate. This is supported by one study that evaluated 158 deaths in trained US athletes between 1985 and 1995 and found a CV cause in 85%[19].

Table 2
Incidence of sudden cardiac death in athletes

<table>
<thead>
<tr>
<th>Population group</th>
<th>Age</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Organized high school/college athletes</td>
<td>High school/college age</td>
<td>7.5:1,000,000/year (male)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1.3:1,000,000/year (female)</td>
</tr>
<tr>
<td>U.S. Air Force recruits [16]</td>
<td>17 to 28 years</td>
<td>1:735,000/year</td>
</tr>
<tr>
<td>Rhode Island joggers [17]</td>
<td>&lt;30 years</td>
<td>1:280,000/year</td>
</tr>
<tr>
<td>Rhode Island joggers [17]</td>
<td>30 to 65 years</td>
<td>1:7,620/year</td>
</tr>
<tr>
<td>Marathon runners [18]</td>
<td>Mean age 37</td>
<td>1:50,000 race finishers</td>
</tr>
</tbody>
</table>
Cardiovascular causes of sudden death in athletes

Cardiomyopathies
- Hypertrophic cardiomyopathy
- Arrhythmogenic right-ventricular dysplasia or cardiomyopathy
- Dilated cardiomyopathy
- Idiopathic left-ventricular hypertrophy

Congenital malformation of coronary arteries
- Coronary artery aberrancies and anomalies
- Intramural coronary artery (myocardial bridging)

Coronary artery disease
- Myocarditis
- Aortic rupture
  - Marfan’s syndrome
  - Coarctation of aorta
- Valvular heart disease
  - Aortic stenosis
  - Mitral valve prolapse

Arrhythmias and conduction system abnormalities
- Long-QT syndrome
- Wolf-Parkinson-white syndrome
- Idiopathic ventricular tachycardia

Illicit drugs

Cardiovascular causes of sudden death

Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is arguably the most common cause of SCD in athletes, accounting for 36% of total deaths [19]. It is inherited in an autosomal-dominant fashion, with marked genetic heterogeneity, and probably affects about 0.2% of the general population [20].

The clinical course of HCM is quite variable, with some patients remaining asymptomatic throughout life and others developing severe symptoms of heart failure (HF) or premature death. Sudden death without antecedent symptoms is most common in children and young adults (10–30 years old) and often occurs during periods of strenuous exertion. Other clinical presentations include dyspnea, angina, arrhythmia, and syncope. Classic physical findings include a bisferious carotid pulse in the presence of LV outlet obstruction, a forceful double or triple apical impulse, and a systolic ejection murmur localized along the left sternal border, accentuated by maneuvers that decrease preload (eg, Valsalva maneuver, squatting).

Several tools are available to evaluate the patient with suspected HCM, including electrocardiography, echocardiography, and genotyping. Classic find-
ings on ECG include left-ventricular hypertrophy (LVH) or a pseudoinfarct pattern, whereas asymmetric thickening of the left ventricle in association with a nondilated chamber is typically seen on the echocardiogram. In fact, the presence of these findings in the absence of other cardiac or systemic diseases capable of causing LVH (ie, hypertension, aortic stenosis) is diagnostic for HCM [21].

Hypertrophic cardiomyopathy is typically divided into obstructive and non-obstructive forms, with the nonobstructive form being much more common [22,23]. As such, features associated with dynamic obstruction—including a systolic ejection murmur, systolic anterior motion of the mitral valve, or premature closure of the aortic valve—are not required for diagnosis. Because an increased left-ventricular wall thickness (LVWT) may be physiologic in some athletes, HCM should only be suspected in patients with a nondilated LV and an LVWT > 12 mm in men or >11 mm in women [24]. A LVWT of 13 mm to 15 mm represents a gray zone associated with diagnostic uncertainty, especially in the setting of nonobstruction. An LVWT >15 mm is highly suggestive of HCM. In patients that fall in this gray zone, features that favor a diagnosis of HCM include: unusual patterns of LVH, an LV cavity <45 mm, left atrial enlargement (LAE), an abnormal ECG pattern, abnormal LV filling, female gender, and a family history of HCM [25]. The absence of these features, along with an LV cavity >55 mm and a decrease in thickness with deconditioning, all favor a diagnosis of athlete’s heart [25].

For patients in whom the precise diagnosis of HCM is uncertain, genetic testing may be useful [26]. One such group includes young athletes with an LVWT in the gray zone or in whom there is not yet a definitive morphological expression of the disease by echocardiography [26].

The treatment of HCM varies, depending upon the presence of symptoms. For asymptomatic patients, beta-blockers or verapamil should only be used in those with significant LVH or a marked LV-outflow gradient [23]. For all other patients, the use of these medications has not been shown to protect against SCD or disease progression. Among symptomatic patients, however, a beta-blocker or verapamil is commonly used to reduce heart rate, and thus prolong diastole and passive ventricular filling. Beta-blockers also lessen myocardial oxygen demand and decrease the outflow gradient during exercise, especially when sympathetic tone is increased [23].

When atrial fibrillation, a common arrhythmia seen in patients with HCM, is present, amiodarone, beta-blockers, or verapamil can be used along with anticoagulation. Among the small subgroup of patients who have both a large outflow gradient (>50 mmHg) and severe symptoms of HF that do not respond to medical treatment, surgical options, including septal ablation, myotomy, and myomectomy, should be considered [23]. The role of dual-chamber pacing for the relief of symptoms in obstructive HCM is currently not clear; however, amiodarone or the use of an implantable cardioverter-defibrillator (ICD) should be considered for patients at high risk of SCD [23].

Among athletes with HCM, with or without symptoms of LV-outflow obstruction, participation in most competitive sports should be prohibited [4].
Older patients (>30 years) may be at reduced risk of cardiac death, and thus individual judgment in assessing eligibility should be done based on the number of potential risk factors for SCD, as listed in Box 3 [4]. Among patients who receive medical or surgical treatment, adherence to sporting restrictions is still strongly recommended.

Arrhythmogenic right ventricular dysplasia

Arrhythmogenic right ventricular dysplasia (ARVD) is a disorder of the heart muscle characterized by ventricular arrhythmias and structural abnormalities of the right ventricle (RV) due to progressive replacement of the myocardium with fibrofatty tissue. ARVD is a relatively uncommon cause of SCD in athletes, except in the Veneto region of northeastern Italy, where it predominates [27].

Patients with ARVD are frequently asymptomatic, but may present with palpitations, syncope, or SCD. The diagnosis of ARVD is based on a scoring system proposed by McKenna and colleagues, and requires that two major, one major and two minor, or four minor criteria be met, as listed in Box 4 [28].

For most cases of ARVD, treatment involves antiarrhythmic drug therapy or placement of an ICD. Because rigorous exercise may induce ventricular tachycardia (VT) and exacerbate the disease, athletes with ARVD should be limited to participation in low-intensity sports (ie, golf and bowling) [4].

Other cardiomyopathies and myocarditis

Idiopathic left-ventricular hypertrophy and dilated cardiomyopathy represent less common causes of SCD in athletes, accounting for 10% and 3% of total cases, respectively [19]. Whether the former condition is distinct or a subset of HCM is not clear. Among patients with these conditions, competitive exercise should be restricted.

**Box 3. Risk factors for SCD in patients with HCM**

- Ventricular tachycardia (sustained or nonsustained)
- Family history of SCD due to HCM (especially if at or below age 40)
- Syncope or other relevant episodes of impaired consciousness
- Severe hemodynamic abnormalities
  - Dynamic LV-outflow tract gradient (>50 mm Hg)
  - Exercise-induced hypotension
  - Moderate to severe mitral regurgitation
- Enlarged left atrium (>50 mm)
- Paroxysmal atrial fibrillation
- Evidence of abnormal myocardial perfusion.
### Box 4. Criteria for diagnosis of ARVD

**Global or regional dysfunction and structural alterations**

**Major**
- Severe dilatation and reduction of right ventricular ejection fraction with no (or only mild) LV impairment
- Localized right ventricular aneurysms (akinetiс or dyskinetiс areas with diastolic bulging)
- Severe segmental dilatation of the right ventricle

**Minor**
- Mild global right-ventricular dilatation or ejection fraction reduction with normal left ventricle
- Mild segmental dilatation of the right ventricle
- Regional right-ventricular hypokinesia

**Tissue characterization of walls**

**Major**
- Fibrofatty replacement of myocardium on endomyocardial biopsy

**Repolarization abnormalities**

**Minor**
- Inverted T waves in right precordial leads (V2 and V3) in people aged >12 years, in absence of right-bundle branch block

**Depolarization/conduction abnormalities**

**Major**
- Epsilon waves or localized prolongation (>110 ms) of the QRS complex in right precordial leads (V1–V3)

**Minor**
- Late potentials (signal-averaged ECG)
Myocarditis is felt to be an uncommon cause of SCD in athletes; however, the exact incidence is not known. Frequently caused by viral agents, myocarditis is an inflammatory disorder of the myocardium that may be largely immunologically mediated. Myocarditis should be suspected in patients that present with fatigue, exertional dyspnea, syncope, palpitations, or signs of HF on examination. An endomyocardial biopsy should be considered in athletes suspected of having myocarditis based on clinical judgment [4].

Hank Gathers, a nationally recognized basketball player at Loyola Marymount University in Los Angeles, collapsed and died while playing basketball during an intercollegiate game in 1990. He experienced a syncopal episode 3 weeks before his death and had undergone an extensive diagnostic CV evaluation that demonstrated exercise-related complex ventricular tachyarrhythmias. After excluding other causes of SCD, including HCM, myocarditis was felt to be the underlying etiology [13]. In light of this, athletes with known or suspected myocarditis should be withdrawn from all competitive sports for at least 6 months. Only after ventricular function and cardiac dimensions have returned to normal

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**Arrhythmias**

**Minor**

Left-bundle branch block type ventricular tachycardia (sustained and nonsustained) on ECG, Holter, exercise testing

Frequent ventricular extrasystoles (>1000/24 hours) (Holter)

**Family history**

**Major**

Familial disease confirmed at necropsy or surgery

**Minor**

Familial history of premature sudden death (<35 years) due to suspected right-ventricular dysplasia

Familial history (clinical diagnosis based on present criteria)

and all clinically significant arrhythmias have resolved should athletes be allowed to return to competition [4].

Coronary artery aberrancies and anomalies

Although seldom recognized and often unappreciated, coronary artery aberrancies and anomalies (CAAA) account for up to 20% of cases of SCD in young athletes [19]. Defined by their site of origin, aberrant coronary arteries arise from the aorta, but in an abnormal or atypical position [29]. Anomalous coronary arteries, however, originate from vessels other than the aorta, including the pulmonary and carotid arteries [29]. Both types of disorders may occur in isolation or in association with more complex forms of congenital heart disease.

Categorization of the numerous types of CAAA is based on the origin, course, and site of termination of the artery. Although most types produce few, if any, symptoms, others pose a much more substantial risk. Two conditions that may be associated with appreciable risk are an aberrant left main coronary artery that originates from the right coronary cusp and an aberrant right coronary artery that originates from the left coronary cusp [30]. As the arteries leave the aorta, they do so at a fairly acute angle, often leaving the ostium with a slitlike appearance. Then, as the arteries cross to their respective normal sides, some may pass between the aorta and the right ventricular outflow tract, with a potential risk of compression, especially following exercise. These consequences predispose to myocardial ischemia, syncope, or decompensated heart failure, and likely account for most cases of SCD.

Other particularly life-threatening CAAA include anomalous left coronary artery from the pulmonary artery (Bland-White-Garland syndrome) and single coronary arteries [30]. In fact, after a record-breaking, hall-of-fame career in basketball, “Pistol Pete” Maravich died at the age of 40 during a pick-up basketball game, and was later found on autopsy to have a single coronary artery.

Due to their frequent absence of symptoms, the detection of CAAA is quite difficult. Aside from a continuous systolic murmur that may be heard with an anomalous left coronary artery from the pulmonary artery, most patients have an unrevealing physical examination. Imaging modalities, including echocardiography, angiography, computed tomography, and magnetic resonance imaging have all been used to visualize CAAA; however, their routine use for screening is not indicated. In patients in whom a CAAA has been identified, however, further participation in competitive sports should be prohibited [2]. Furthermore, a prompt evaluation by a cardiothoracic surgeon should be performed for consideration of coronary artery reimplantation or coronary artery bypass grafting (CABG) to reduce the risk of SCD.

Myocardial bridging

A myocardial bridge (MB) or tunneled coronary artery is an uncommon cause of SCD in athletes. Frequently benign, this congenital condition is defined by the
presence of an epicardial coronary artery that is covered by myocardial fibers. Because it may be associated with angina, conduction system abnormalities, and SCD, patients with a symptomatic MB should be considered for treatment with beta-blockers, as well as revascularization with percutaneous coronary intervention or CABG [6].

**Coronary artery disease**

Atherosclerotic CAD is the leading cause of SCD during physical exertion in master athletes over the age of 35 years, but is rarely the cause in younger individuals [31]. Among patients who have experienced SCD due to CAD, prodromal symptoms of chest pain are frequently noted. Identification of risk factors such as elevated low-density lipoprotein (LDL) cholesterol, low high-density lipoprotein (HDL) cholesterol, hypertension, diabetes mellitus (DM), smoking, and a family history of premature CAD are important in athletes, especially over the age of 35 years. Among asymptomatic patients with moderate to high risk, screening with exercise treadmill testing can be one strategy to reduce death [31]. High-intensity competitive sports are not recommended for athletes with documented ischemic heart disease, regardless of whether the patient has symptoms, a history of previous MI, or has undergone complete revascularization [6].

**Aortic rupture and dissection**

Marfan’s syndrome, an autosomal dominant disorder of the fibrillin gene, accounts for the majority of cases of aortic rupture or dissection seen in young athletes. Flo Hyman, the famous Olympic volleyball player, died at the age of 31 from this disorder. Because of alterations in connective tissue of the aorta, root dilation with subsequent aortic regurgitation or dissection may be seen. These changes, along with aortic rupture, account for most deaths in this disorder [26,32,33].

A detailed family history will often reveal an unusual body habitus and may disclose associated CV events. Skeletal changes in Marfan’s syndrome are easiest to recognize and classically include long thin fingers, long limbs, tall stature, a funnel chest with a pigeon breast, rib deformities from bone overgrowth, a high arched narrow palate, and an excessive arm span [32]. A primary goal of management, therefore, is to detect CV manifestations early, with the intent of intervening before more significant complications arise. Surgical therapy is the mainstay of treatment for patients with these adverse CV complications. Patients with Marfan’s syndrome are advised to avoid heavy weight lifting and contact sports.

Aortic coarctation is a relatively uncommon condition that predominantly affects males and may result in aortic rupture or dissection if untreated [34]. Patients often report a history of epistaxis, headache, exertional leg weakness, or symptoms of HF. On physical examination, radial-femoral pulse delay, a differ-
ential upper limb systolic blood pressure of at least 10 mmHg (brachial>popliteal artery pressure), an interscapular crescendo-decrescendo murmur, and “cork-screw” tortuosity of retinal arterioles on fundoscopic examination may all be seen. Given the CV risks if uncorrected, surgery is the mainstay of definitive treatment.

Valvular heart disease

Valvular aortic stenosis (AS) is an uncommon cause of SCD in young athletes, occurring most commonly in patients with symptomatic disease during periods of physical exertion. Among the various causes, congenital AS (most commonly bicuspid) accounts for up to 50% of cases in patients <70 years of age [3]. Fatigue, light-headedness, dizziness, syncope, and chest pain may all be presenting features, and a constant apical ejection click and systolic ejection murmur may be heard on examination.

Unlike acquired AS, congenital AS is divided into mild (<20 mm Hg), moderate (21–49 mm Hg), and severe (>50 mm Hg) categories, based on the peak instantaneous systolic pressure gradient measured by Doppler echocardiography [2]. Current guidelines suggest that patients with asymptomatic mild AS can participate in all competitive sports. Athletes with moderate AS can participate in low to moderate static or dynamic exercises if they: (1) have no LV strain and minimal LVH on ECG, (2) lack ischemia or arrhythmias on exercise stress testing, and (3) are asymptomatic. Athletes with AS and a history of syncope or severe disease should be prohibited from participation in competitive sports [2].

Mitral valve prolapse (MVP) is a benign and common cardiac condition that is present in up to 5% of the general population [4]. Classically characterized by systolic protrusion of the mitral valve (MV) leaflets into the left atrium and a midsystolic click with or without a late systolic murmur, MVP is a rare cause of SCD in athletes. Athletes without high-risk features may safely engage in all competitive sports, whereas those with high-risk features, listed in Box 5, should be limited to lower-intensity exercise only [4].

### Box 5. High-risk features associated with mitral valve prolapse

- Arrhythmogenic syncope
- Family history of SCD in association with MVP
- Repetitive sustained and nonsustained tachyarrhythmias, including those of ventricular origin, that are exaggerated by exercise
- Moderate to severe mitral regurgitation
- Prior embolic event
**Arrhythmia and conduction abnormalities**

Most causes of SCD in athletes are arrhythmogenic, predominantly the result of ventricular tachyarrhythmias. Resulting from either structural changes (HCM, DCM, ARVD) or ischemia (CAD, CAAA), scar-mediated re-entry is likely the prevailing mechanism. A few notable exceptions to this do occur and likely account for a small number of cases of SCD in athletes. These include: congenital long-QT syndrome (LQTS), Wolff-Parkinson-White (WPW) syndrome, idiopathic ventricular tachycardia (VT), and Brugada syndrome.

With most cases resulting from mutations in the potassium and sodium channels, LQTS is a heterogeneous familial disorder with varied phenotypic expression. Syncope and SCD during or following exertion have been described in LQTS, and are most commonly due to polymorphic VT or ventricular fibrillation [35]. In this condition, beta-blockers have been shown to decrease the frequency of syncope and SCD [35]. Most believe, however, that ICD provides protection against SCD, particularly in those with syncope and recurrence of ventricular arrhythmia while the patient is undergoing beta-blocker therapy [35]. Given these risks, athletes with LQTS are advised to participate in low-intensity competitive sports only [7].

Wolff-Parkinson-White (WPW) syndrome is an uncommon condition with an estimated incidence of 0.3% [35–37]. The result of an accessory pathway between the atrium and ventricle, WPW syndrome may be clinically silent or life-threatening. With antegrade conduction down the bypass tract, the ECG may reveal a short PR interval with a delta wave and more importantly, conduction of arrhythmias at supraphysiologic rates (>300 beats per minute) may occur. Electrophysiologic evaluation has been proposed as one means for risk stratification in asymptomatic patients [36,37]. If the rate of accessory pathway conduction is >240 beats per minute or the patient is symptomatic, radiofrequency ablation (RFA) should be strongly considered [35]. Among athletes that are free of symptoms 3 to 6 months after undergoing RFA, there should be no limitation to resumption of sporting activity [7].

Idiopathic VT is an uncommon cause of SCD that usually originates from one of the ventricular outflow tracts. This arrhythmia can be effectively treated by RFA [35]. Among athletes that undergo RFA, resumption of sports may occur after 3 months [35].

Brugada syndrome, characterized by an ECG showing right-bundle branch block and ST elevation in the right precordial leads, can be a marker of malignant arrhythmias and a rare cause of SCD in athletes [38].

**Illicit drug use**

Despite the highly publicized deaths of athletes such as Len Bias and Don Rogers, the use of illicit drugs by athletes continues to be a major sporting health problem. Unlike many of the drugs abused by the general population, drugs abused by athletes are often sought because of their perceived ability to enhance
performance in competition. This has led several organizations, including the United States Olympic Committee (USOC) and the National Collegiate Athletic Organization (NCAO) to routinely test competitive athletes for illicit drugs. Narcotics, anabolic steroids, diuretics, peptide and glycoprotein hormones, alcohol, marijuana, amphetamines, corticosteroids, and beta-blockers are among some of the banned substances, many of which have CV effects (Table 3)[35]. Excessive absenteeism, poor grades, restlessness, hyperactivity, tremulousness, and pupillary abnormalities may all reflect ongoing drug use. If drug use is suspected, athletes should be limited from participating in competitive sports and should be referred for drug-treatment.

Preparticipation screening

In athletes, the main goal of preparticipation screening is to detect previously unrecognized CVD that may be associated with an increased risk of SCD. Although important, preparticipation screening is severely limited by the large number of individuals that would need to be screened in the United States (8 million) and the low overall prevalence of SCD (<0.5%) in this population [12]. Nonetheless, the impact of SCD in a young individual during competition always drives the question “what more could have been done to identify this person?”

The effective use of screening tests demands thorough epidemiological evaluation, not merely acceptance as a result of public outcry or consensus. Based on previous studies, it has been estimated that as many as 200,000 competitive, asymptomatic athletes would need to be screened to identify a single athlete who would die as a result of competition [39]. Even if we had a tool to screen for SCD with a sensitivity and specificity of 99%, the low prevalence of disease in this population would yield a positive predictive value of only 0.05%.

Because HCM is the most common cause of SCD in young athletes, most studies have focused screening efforts on the detection of this disease. In addition to a thorough history and physical examination, echocardiography has regularly been proposed as an effective screening tool, because of its high reported sen-

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**Table 3**

Illicit drugs and their adverse cardiovascular effects

<table>
<thead>
<tr>
<th>Illicit drug</th>
<th>Adverse cardiovascular effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anabolic steroids</td>
<td>Premature CAD, MI, CM, SCD</td>
</tr>
<tr>
<td>Human growth hormone</td>
<td>Premature CAD, CM</td>
</tr>
<tr>
<td>Amphetamine</td>
<td>Coronary artery spasm, MI, SCD, CM, ventricular arrhythmias</td>
</tr>
<tr>
<td>Ma huang and ephedra alkaloids</td>
<td>MI, SCD, arrhythmias, CVA</td>
</tr>
<tr>
<td>Cocaine</td>
<td>Arrhythmias, coronary artery spasm, MI, HTN, dilated CM, myocarditis, pulmonary edema, ruptured aortic aneurysm</td>
</tr>
</tbody>
</table>

Abbreviations: CAD, coronary artery disease; CM, cardiomyopathy; CVA, cerebrovascular accidents; HTN, hypertension; MI, myocardial infarction; SCD, sudden cardiac death.
sensitivity and specificity. Although several studies have applied echocardiography to screening large athletic populations, most have been disappointing, with a significant associated cost. In fact, in one study of 501 college athletes screened using personal and family medical history and a 12-lead ECG, 90 subsequently underwent echocardiography. At a cost of approximately $45,000 for the echocardiograms, only 3 athletes were identified as having interventricular septal thickening, none of whom were restricted from sporting participation [40].

Guidelines

Despite the limitations of athletic screening, the American Heart Association recommends that some form of preparticipation CV screening be performed for high school and collegiate athletes [41]. This process primarily focuses on the identification of historical and physical features associated with increased risk by an experienced health care worker. These features are listed in Box 6. The screening process should be repeated every 2 years in high school athletes, with a

<table>
<thead>
<tr>
<th>Box 6. AHA consensus panel recommendations for preparticipation screening [31,41]</th>
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<tbody>
<tr>
<td><strong>Personal history</strong></td>
</tr>
<tr>
<td>Exertional chest pain</td>
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<tr>
<td>Heart murmur</td>
</tr>
<tr>
<td>Easy fatigability</td>
</tr>
<tr>
<td>Syncope</td>
</tr>
<tr>
<td>Exertional dyspnea</td>
</tr>
<tr>
<td>Systemic hypertension</td>
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<tr>
<td><strong>Family history</strong></td>
</tr>
<tr>
<td>Premature sudden death</td>
</tr>
<tr>
<td>Heart disease in close relatives younger than 50 years of age</td>
</tr>
<tr>
<td><strong>Physical examination</strong></td>
</tr>
<tr>
<td>Heart murmur (precordial auscultation in supine and standing</td>
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<tr>
<td>positions to identify heart murmurs consistent with dynamic</td>
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<td>left-ventricular outflow obstruction)</td>
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<td>Femoral pulses</td>
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<tr>
<td>Stigmata of Marfan’s syndrome</td>
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<tr>
<td>Blood pressure measurement</td>
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more thorough history and blood pressure measurement performed in each of the subsequent 3 to 4 years for college athletes.

Summary

Although disturbing, SCD in athletes fortunately remains an infrequent event. Numerous CV diseases have been associated with increased risk; however, the correct identification of these conditions remains difficult. Screening methods, including echocardiography, are currently available, but remain limited largely by cost and the low incidence of these disorders in the screened population. Therefore, a thorough history and physical examination should be regularly performed on athletes, with meticulous evaluation of any abnormal findings. In addition, as more sensitive tests become available to identify at-risk individuals, regular testing should be performed to assess their utility.

References


