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Chapter

Pre-Sports Participation Cardiac Screening Evaluation – A Review

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Abstract

Sudden death is associated with sports, while rare is a disastrous event. Sudden death in athletes often has a cardiac etiology. Hypertrophic cardiomyopathy and congenital coronary artery anomalies are the two most frequent causes. The existing recommendations are to perform a pre-sports participation screening consisting of full personal and family history and detailed physical examination. If abnormal findings in history or physical examination are found, additional investigations should be performed to define the nature of abnormalities. Employing an electrocardiogram, echocardiogram, or magnetic resonance imaging as a routine screening technique is not recommended in the US. The rationale of pre-participation screening is to allow as many athletes as feasible to take part in the sports.

Keywords: sudden cardiac death, athlete, pre-participation screening, electrocardiogram, echocardiogram, hypertrophic cardiomyopathy, aberrant coronary artery, Marfan's syndrome

1. Introduction

Sudden cardiac death (SCD) is defined as any death secondary to a cardiac cause within minutes to 24 h of the exercise activity [1–3]. The SCD may be associated with a rhythm disturbance or circulatory collapse. The prevalence of SCD in children is approximately 600 per annum in the USA [3]. By contrast, sudden infant death syndrome (SIDS) occurs in 7000–10,000 infants per year, and SCD in adults is seen in 300,000–400,000 subjects per year [3]. Consequently, the prevalence of SCD in childhood (and young adult) athletes is a lot less frequent than SIDS in babies and SCD in adults. The objectives of this presentation are to list the most frequent cardiac causes of sudden death in athletes, describe clinical features of more common cardiac disease entities causing SCD, and discuss methods of pre-sports participation screening. The discussion will not include SIDS and SCD without an antecedent exercise activity.

2. Causes of sudden death in athletes

Disease processes causing SCD have changed remarkably over the years. In the 1970s, aortic valve stenosis, un-palliated cyanotic congenital heart defects (CHDs), and Eisenmenger's syndrome were the major culprits. In the 1990s, hypertrophic

cardiomyopathy (HCM), congenital anomalies of the coronary arteries (CAs), premature atherosclerosis, rupture of the aorta in Marfan's syndrome, and arrhythmias were identified as major diseases entities causing SCD in athletes [2]. At the present time, HCM; congenital anomalies of the CAs; Marfan's syndrome; structural cardiac defects, namely, repaired tetralogy of Fallot, repaired transposition of the great arteries by Mustard or Senning procedures, single ventricle lesions addressed by Fontan operation; CHD without prior surgery, including aortic is responsible for SCD [2–4]. Less common causes namely, acute or chronic myocarditis; complex forms of mitral prolapse, arrhythmogenic right ventricular cardiomyopathy (ARVC), Eisenmenger's syndrome; long QT syndrome; other abnormalities of the coronary artery such as Kawasaki disease and familial hyperbeta hyperlipoproteinemia; commotio cordis; catecholaminergic polymorphic ventricular tachycardia; and Brugada syndrome were also found to be responsible for SCD [4–7]. Some of these entities will be reviewed in the ensuing paragraphs. In the US, SCD is seen more frequently following basketball and football than with other sports; this is likely to be related to the requirement for high level of physical activity in these sports [5, 6].

3. Hypertrophic cardiomyopathy

3.1 Introduction

This condition has been called asymmetric septal hypertrophy (ASH), hypertrophic obstructive cardiomyopathy (HOCM), dynamic muscular subaortic stenosis, idiopathic hypertrophic subaortic stenosis (IHSS), diffuse muscular subaortic stenosis, Brock's Disease, Teare's Disease, and others, but is now best described as hypertrophic cardiomyopathy. HCM is a primary, often inherited, disease of the myocardium affecting the sarcomeric proteins. This results in hypertrophy and disorganization of myofibrils and fibrosis of interstitia. Autosomal dominant inheritance with a high degree of penetrance is noted. Matrilineal, X-linked, and autosomal-recessive inheritance patterns have also been described. One hundred or more different mutations in 10 genes have been discovered (chromosomes 1, 14 & 15). Troponin-T, beta myosin heavy chain, and alpha tropomyosin may also be involved.

3.2 Pathology and pathophysiology

HCM is characterized by hypertrophy of the left ventricle (more often) and/or right ventricle (RV). Concentric left ventricular (LV) hypertrophy is present without an identifiable hemodynamic reason. Frequently, there is asymmetric interventricular septal hypertrophy. Diastolic dysfunction including, delayed relaxation and increased muscle stiffness are present. LV outflow obstruction, which is dynamic is seen in 25% of cases. Systolic anterior motion (SAM) of the mitral valve may contribute to the LV outflow tract narrowing. RV outflow obstruction is rare but is more frequent in infants with HCM. There appear to be areas of localized hypertrophy in the LV muscle in addition to those in the septum and serve as potential arrhythmogenic foci.

3.3 Symptoms

There are usually no symptoms in infants and children; they are most often detected because of a cardiac murmur or family history of HCM. Symptoms in infancy

usually indicate a poor prognosis. In early adulthood, the presenting symptoms are exertional dyspnea, lightheadedness, chest pain, syncope, or palpitations.

3.4 Physical examination

In the non-obstructive HCM, the findings are subtle with minimal or no increase in LV impulse. A third or fourth heart sound may be auscultated at the apex. There may either be a soft systolic murmur or no murmur. In the obstructive HCM, increased LV impulse, double or triple impulse may be felt. Pulses bisfiriens may be felt in some subjects. An ejection systolic murmur is heard at the left mid and right upper sternal borders. The murmur changes with postural maneuvers: decreases in intensity on squatting which may increase in a standing position. The murmur of HCM is usually auscultated at the left mid sternal border and increases in intensity with the Valsalva maneuver.

3.5 Chest X-ray and electrocardiogram (ECG)

Chest X-ray is of limited value; the size of the heart may be normal or cardiomegaly may be present. The size of the heart may be normal even with marked LV hypertrophy. ECG is abnormal in 90% of patients; bizarre patterns are seen, but none are characteristic of HCM. Most commonly, increased LV voltages (deep S waves in V1 and V2 and tall R wave in V5 and V6) are detected. T wave inversion, deep q waves, and enlargement of the left atrium may be identified on the ECG in some patients. Unfortunately, the ECG pattern does not discriminate between obstructive and non-obstructive types or those at risk for sudden death. However, Holter monitoring is likely to detect arrhythmia [8, 9] and may help prognosticate [9].

3.6 Echo-doppler studies

Echocardiogram is the most useful test in the diagnosis of this condition. It demonstrates LV hypertrophy (**Figure 1**), asymmetric septal hypertrophy (**Figures 1 and 2**),

Figure 1.
A. Parasternal long (A) and short (B) axis views of the left ventricle (LV) illustrating LV hypertrophy and strikingly thickened inter-ventricular septum (IVS). Ao, aorta; LVPW, LV posterior wall; RV, right ventricle. (reproduced from reference [10]).

Figure 2. Parasternal short (left) and long (right) axis views of the left ventricle (LV) illustrating distinctly thickened inter-ventricular septum (arrows in both images). Ao, aorta; LA, left atrium.

and SAM of the mitral valve (**Figure 3**). The LV cavity is completely obliterated in systole (**Figure 4**). Doppler studies will detect abnormal mitral inflow Doppler patterns and mitral insufficiency and help to quantify LV outflow tract obstruction (**Figures 5 and 6**).

Figure 3. Parasternal echocardiographic long axis views of the left ventricle (LV) and mitral valve illustrating systolic anterior motion (SAM) of the mitral valve (thick arrows in both 'A' and 'B'). Note thickened inter-ventricular septum (thin arrows), particularly noticeable in 'B'. Ao, aorta; LA, left atrium; RV, right ventricle.

Figure 4.
Parasternal long-axis views of the left ventricle (LV) of a child with hypertrophic cardiomyopathy demonstrating total obliteration of the cavity of the LV (right image) in systole (arrows).

Figure 5.
A. Parasternal long axis 2D and color doppler recording showing turbulent flow (TF) in the left ventricular (LV) outflow tract. B. Continuous-wave doppler indicates a gradient of 39 mmHg (the insert in B). The triangular pattern of the doppler recording is suggestive of subaortic narrowing. Ao, aorta. (reproduced from reference [10]).

Localized, atypical hypertrophy patterns as demonstrated in **Figures 7–9** may also be detected.

3.7 Differentiation from athlete's heart

Some athletes develop LV hypertrophy and differentiation of this LV hypertrophy from that seen with HCM maybe not easy. Criteria that help differentiate these conditions [7] are listed in **Table 1**.

3.8 Sudden cardiac death in HCM

Yearly mortality rate in subjects with HCM is in the order of 2 to 4% of the HCM cases; this is largely based on patients cared for in large tertiary care centers.

Figure 6.
Apical five-chamber view with continuous-wave doppler across the left ventricular outflow tract demonstrating a peak gradient of 75 mmHg. The triangular pattern of the doppler recording is suggestive of subaortic narrowing.

Figure 7.
Parasternal long-axis views of the left ventricle of a patient with hypertrophic cardiomyopathy showing marked inter-ventricular septal thickening (arrows). LA, left atrium.

Consequently, the true population prevalence is unknown. Examination of SCD in competitive athletes revealed that HCM is the most frequent reason for SCD and accounted for 36% of deaths [5–7]. The mortality is largely confined to the 12-to-35-year age patient group. Most of these patients are asymptomatic or mildly

Figure 8.
Subcostal view of the left ventricle (LV) of a patient with hypertrophic cardiomyopathy showing marked inter-ventricular septal thickening (arrows).

symptomatic and therefore, risk stratification of HCM is difficult. In some investigations, risk factors for SCD in HCM patients have been identified and these include, a family history of sudden death related to HCM, history of syncope or pre-syncope, history of having survived sudden death episode, massive LV hypertrophy (> 30 mm), non-sustained ventricular tachycardia, unusual blood pressure (BP) response to exercise, and high LV outflow tract gradient [1–4, 11–13].

3.9 Sudden cardiac death prevention in HCM

The mechanism by which HCM results in SCD following exercise is not clearly understood, but the development of ventricular arrhythmia, leading to ventricular fibrillation is thought to be responsible for SCD [2]. Even at the present time, we have limited knowledge as to how to prevent SCD in HCM. We continue to lack sound criteria of individual severity and relative prognostic risk, in general, and especially in athletes. Early studies showed the usefulness of beta-blockers in reducing mortality, but recent studies did not corroborate the usefulness of beta-blocker usage in the prevention of mortality. Exercise limitation may be helpful in decreasing the probability of sudden death. Indeed, the athlete with HCM should not be permitted to take part in the strenuous sports activity. If non-sustained ventricular tachycardia is present, Amiodarone has been shown to be beneficial. Intra-cardiac defibrillators (ICDs) for high-risk patients with ventricular tachycardia induced by programmed ventricular stimulation may be helpful.

Figure 9. Apical four-chamber view of the left ventricle (LV) of a patient with hypertrophic cardiomyopathy showing marked inter-ventricular septal thickening (arrows). RA, right atrium.

Parameter	Hypertrophic cardiomyopathy	Athlete’s heart
Unusual patterns of LVH on echo	Positive	Negative
LV Cavity <45 mm	Positive	Negative
LV Cavity >55 mm	Negative	Positive
Left atrial enlargement	Positive	Negative
Bizarre ECG patterns	Positive	Negative
Abnormal LV filling	Positive	Negative
Female gender	Positive	Negative
Decreased LV muscle thickness with de-conditioning	Negative	Positive
Family history of hypertrophic cardiomyopathy	Positive	Negative

ECG, electrocardiogram; LV, left ventricle; LVH, left ventricular hypertrophy.

Table 1. Criteria to distinguish athlete’s heart from HCM.

4. Congenital coronary artery anomalies

4.1 Normal coronary arteries

In normal subjects, the left and right coronary arteries (CAs) originate from left and right sinuses of Valsalva, respectively. The left main CA is short and divides into

two branches, namely the left anterior descending (LAD) and circumflex. The LAD continues in the same course as the left main CA and traverses in the interventricular groove. The circumflex traverses perpendicular to the axis of the left main CA and keeps on going in the posterior atrioventricular groove. The left CA perfuses the LV and interventricular septum [14, 15]. The right CA originates from the right sinus of Valsalva and continues in the right atrioventricular sulcus and does not branch out similar to the left CA. The right CA supplies the RV [14, 15]. The site of origin and the course of the CAs proximally can easily be demonstrated in echocardiographic studies (**Figures 10** and **11**). Color flow mapping of the CAs (**Figures 11** and **12**) should also be undertaken concurrently to ensure that the parallel lines of the transverse sinus of the pericardium are not mistaken for CAs [16].

4.2 Aberrant coronary arteries

The aberrant origin of the CA is a rare abnormality with its origin from the contralateral aortic sinus of Valsalva [16]. In aberrant left CA, the left CA originates from the right sinus of Valsalva (rarely from the right CA) with a short intramural (within the aortic wall) course and continues between the pulmonary trunk anteriorly and the aorta posteriorly. The ostium of the left CA is often Slit-like forming a potential site for obstruction to coronary blood flow. In aberrant right CA, the right coronary artery arises from the left sinus of Valsalva (less commonly from the left main CA). This is the counterpart of aberrant left CA. The right CA then traverses rightward with a short intramural course within the aortic wall and then traverses between the pulmonary artery and aorta to get to its usual course. There is adequate coronary flow at rest, but during exercise, ischemia may develop secondary to either

Figure 10.
 A short-axis view at the level of aortic sinuses demonstrating normal left and right (RC) coronary arteries and their branches. The left main coronary artery (LMC) continues in the same direction to become the left anterior descending (LAD) artery. The circumflex (CiR) and marginal (MR) branches traverse perpendicular to the axis of LMC. The aorta (AO) and pulmonary artery (PA) are also shown. Demonstration of color flow within the coronary arteries is required to ensure that these indeed are coronary arteries. (modified from reference [16]).

Figure 11. The short-axis views at the level of aortic sinuses demonstrate the origin and course of the left (LCA) (A and B) and right (RCA) (C and D) coronary arteries. The LCA continues in the same direction to become the left anterior descending artery (LAD). The circumflex (CIRC) traverses perpendicular to the axis of LCA. Color flow within the coronary arteries is demonstrated in 'B' and 'D' and is required to ensure that these indeed are coronary arteries and not parallel lines of the transverse sinus of the pericardium. Ao, aorta.

Figure 12. A. a short-axis view at the level of aortic sinuses, similar to **Figures 10 and 11**, focusing on the left main coronary artery showing its division into left anterior descending (AD) and circumflex (CIR) coronary arteries. Note the red flow in the AD and the blue flow in the CIR. B. a short-axis view at the level of aortic sinuses, similar to figure a, but focusing on to the right coronary artery (RCA) showing its origin from the right sinus with the color flow within it. AO, aorta.

partial or total occlusion of the CA [17, 18]. Several hypotheses have been proposed to elucidate the reason(s) for inadequate CA blood flow. One such hypothesis is compression of the CA (left or right) between the great arteries by the expansion of the aorta and pulmonary artery against each other during the exercise. Another hypothesis suggests that an angle is created between ostium of the CA and its main axis creating additional tension, during aortic expansion [19–21]. The intramural course of the coronary artery also contributes to the risk of SCD during exertion. Echocardiographic examples of aberrant left CA arising from the right sinus of Valsalva (**Figure 13**) and of aberrant right CA arising from the left sinus of Valsalva (**Figures 14–16**) are shown in **Figures 13–16**.

4.3 Aberrant coronary arteries and SCD

The aberrant origin of the left CA is strongly associated with SCD [19, 20]. While not as common, aberrant right CA is also seen with SCD associated with exercise [21]. Aberrant CAs constitute 17% of all SCDs following exercise [5–7] and are next only to HCM with regard to frequency.

4.4 Clinical and laboratory features of aberrant coronary arteries

Often, the first presentation of most patients with aberrant coronary arteries is SCD with exercise. A few patients may present with symptoms of angina or syncope following exercise [22]. Some patients may be detected during echocardiograms performed for some other reason. Routine physical examination is completely normal. Patients with complaints of angina or syncope following exercise should be evaluated

Figure 13.
A short-axis view of the aorta demonstrating aberrant left coronary artery from the right sinus of Valsalva, traversing between the aorta (AO) and pulmonary artery (PA). (reproduced from reference [16]).

Figure 14.
The short-axis views of the aorta (Ao) demonstrate aberrant right coronary artery (RCA) from the left sinus of Valsalva by two-dimensional (A and B) and color flow (CF) (C) imaging. The intramural (IM) course of the right coronary artery is shown in 'B'.

Figure 15.
The short-axis views of the aorta (Ao) demonstrate aberrant right coronary artery (RCA) from the left sinus of Valsalva by two-dimensional (A) and color flow (B) imaging. Color flow (CF) at the origin of RCA (B) and intramural (IM) course of the RCA (A and B) are shown. PA. Pulmonary artery.

by a cardiologist. Stress testing, while routinely used for complaints of this nature, a regular stress test may not yield abnormal results in subjects with aberrant CAs particularly if the stress test is sub-maximal [16, 17]. If the stress test with maximal activity is performed, it may precipitate a coronary event or even sudden death. Instead, the author recommends echocardiographic studies, focusing on imaging of proximal CAs [16]. Reliable echocardiographic screening methods to identify aberrant CAs (**Figure 17**) have been described [23, 24] and may be used. However, normalcy (**Figures 10–12**) of the CAs or aberrancy (**Figures 13–16**) should be documented by direct visualization of the CAs by transthoracic echocardiographic studies. With the

Figure 16.
The short-axis views of the aorta (Ao) demonstrate aberrant right coronary artery (RCA) from the left sinus of Valsalva by two-dimensional (A) and color flow (B) imaging. Color flow acceleration (FA) at the origin of RCA (B) may suggest stenosis at the origin of RCA. The intramural (IM) course of the RCA (A and B) is seen. PA. Pulmonary artery.

Figure 17.
Parasternal long-axis two-dimensional echo images of the left atrium (LA), left ventricle (LV) and aorta (AO) in a normal child are shown in A. A similar view of the heart in a child with an aberrant coronary artery (CA) is shown in B which demonstrated a cross-sectional image of the CA in the anterior wall of the AO (arrow in B). RV, right ventricle. Modified from Jureidini SB, Marino CJ, Singh GK, Balfour IC, Rao PS. J Am Society of Echocardi 2003; 16: 756–763 [23].

current state-of-the-art echocardiographic equipment, echo studies are the primary tools of investigation. If transthoracic echocardiographic images are not clear, especially in adolescents and adults with poor acoustic windows, other studies such as transesophageal echocardiography (TEE), computed tomography (CT) scan, or angiography may have to be performed to demonstrate the CA anatomy. Surgical re-implantation of the aberrant CAs along with enlarging the coronary ostium and un-roofing the intramural portion of the CA, as deemed appropriate, can be safely performed, and subsequent to recovery from surgery, participation in the full athletic activity is feasible.

5. Marfan’s syndrome

Marfan’s syndrome is a disorder of the connective tissue with multisystem involvement, particularly of the musculoskeletal, ocular, and cardiovascular systems [25, 26]. Mutations of the fibrillin-1 gene are believed to be responsible for this syndrome. The overall prevalence is one in 10,000 to 20,000 individuals [27]. The inheritance pattern is autosomal dominant with variable expression. In approximately 15% of cases, it may manifest sporadically [25]. Several reviews of SCD in young athletes disclosed aortic rupture in Marfan’s syndrome patients (2 to 3%) as a causative factor [1, 5–7, 26, 28].

Clinical manifestations of Marfan’s syndrome are tall stature with arm span >105% of the height, scoliosis, arachnodactyly, pectus deformity, hyper-extensible joints, and dislocation of the lens. They may also have glaucoma and retinal detachment. Some of the clinical features are illustrated in **Figure 18**. Cardiovascular abnormalities include mitral valve prolapse (**Figure 19**) in almost 100% of patients, some with mitral regurgitation (**Figure 19**), and aortic root dilatation (**Figures 20 and 21**) in a high percentage of patients [26, 29]. The mitral prolapse and mitral regurgitation may be appreciated on careful auscultation and confirmed by echocardiography. However, echocardiography is necessary to detect aortic root dilatation [29]; the measurements are compared with normal subjects, and Z scores are determined. The diagnosis of Marfan’s syndrome is largely on the basis of Ghent nosology criteria [27, 30] using a mixture of major and minor clinical features and the family history. In the absence of

Figure 18.
A summary of clinical features of Marfan’s syndrome.

Figure 19.
Selected video frames from apical four-chamber views demonstrating mitral valve prolapse (MP) and mitral regurgitation jet (MRJ) in a patient clinically diagnosed as Marfan’s syndrome. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

Figure 20.
Selected video frame from parasternal long-axis view demonstrating measurements of the dilated aortic root in a patient clinically diagnosed as Marfan’s syndrome. Note the measurements are listed at the left bottom. The Z scores of the dilated aortic root varied between 2.9 and 3.8.

these criteria, a scoring system is utilized [31] for the diagnosis. Spontaneous aortic dissection has been observed. To prevent such catastrophes, surgery to replace the aortic root is suggested if the aortic diameter exceeds 5.0 cm or if the rate of growth of the aorta is more than 0.5–1.0 cm/year [30]. Beta-blocking drugs, angiotensin converting enzyme (ACE) inhibiting medications and more recently, losartan (to attenuate TGF β signaling) have been used to decrease the rate of growth of the aorta. In an attempt to prevent aortic dissection and SCD, restriction from participation from sports is generally recommended [31].

Figure 21.

Selected video frames from parasternal long-axis view demonstrating measurements of the dilated aortic root in a patient clinically diagnosed as Marfan's syndrome. The measurements are listed at the left bottom in both 'A' and 'B', while the Z score of the aortic valve (AV) annulus was within normal limits, the Z score of the dilated aortic sinus was +5.5.

6. Structural heart defects

Congenital cardiac defects following repair (tetralogy of Fallot—total correction; transposition of the great arteries—Mustard or Senning operation, and single ventricle lesions—Fontan operation) in 2% and un-operated CHDs, including aortic stenosis in 3% have been reported as causative factors for SCD following exercise activity [4–7]. The clinical features of these disease entities [32–37] and their long-term sequelae [38, 39] were reviewed elsewhere [32–39] for the interested reader and will not be reviewed here.

7. Arrhythmogenic right ventricular cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a rare myocardial disorder inherited in an autosomal dominant pattern with an estimated prevalence of 1:1000–1:5000 [40]. It has been identified as a cause of SCD following sports participation in approximately 4% of cases [5–7]. Cardiomyopathic changes most frequently involve the right ventricular myocardium; however, cases involving the LV have also been reported. Exercise may be important in eliciting ventricular arrhythmias. The usual presenting symptoms are palpitations, syncope, or aborted cardiac arrest. Congestive heart failure may occur in the late stages. T-wave inversion in the right chest leads, indicative of repolarization abnormalities are seen in the ECG. However, the ECG is not sensitive in detecting ARVC [41]. The diagnosis is largely based on major and minor criteria in the echocardiographic and MRI studies, reviewed elsewhere [40, 42]. The mechanism for SCD following exercise is the development of ventricular arrhythmia [40].

8. Other disease entities

There are number of other diseases/defects that have been known to have a causal relationship with SCD following exercise; the prevalence of each of these varied

Disease entities	Percent prevalence	Comments
Myocarditis	6% [5–7]	Early studies indicated a higher prevalence ranging from 18 to 29% [28, 43, 44]
Complex Forms of Mitral Prolapse	4% [5–7]	Family history of SCD and exercise-induced arrhythmias in mitral prolapse cases are likely to be related to SCD [2]
Ion Channelopathies	3 to 4% [5–7]	Long QT syndromes (Jervell and Lange-Nielson syndrome and Romano-Ward syndrome) and Brugada syndrome
Myocardial Bridging of the Left Anterior Descending Coronary Artery	3% [5–7]	Has also been reported in other studies [16, 45, 46]
Atherosclerotic Coronary Artery Disease	3% [5–7]	Premature atherosclerotic coronary artery disease secondary to hyperbeta hyperlipoproteinemia
Dilated Cardiomyopathy	2% [5–7]	—
Wolf Parkinson White syndrome	2% [7]	—
Pulmonary Hypertension (Eisenmenger’s Syndrome), Sarcoidosis, Kawasaki Disease, Commotio Cordis	~1% each [2, 5, 6, 28, 47]	Pulmonary Hypertension (Eisenmenger’s Syndrome) [28], Sarcoidosis [5], Kawasaki Disease [2, 6], Commotio Cordis [6, 47]

ECG, electrocardiogram; SCD, sudden cardiac death.

Table 2.
Other disease entities associated with SCD following exercise.

between 1 and 6% [5–7]. These are listed in **Table 2**. A detailed discussion of these entities is beyond the scope of this paper.

9. Pre-sports participation screening

Pre-sports participation screening is commonly suggested by most medical societies [5, 48–52] including the American Academy of Pediatrics (AAP), American Heart Association (AHA), and the American College of Cardiology (ACC). Yet, the heart conditions which predispose to SCD happen in 5 in 100,000 persons and SCD occurs in 1 in 200,000 subjects. This degree of low risk makes the appraisal hard and the cost-effectiveness of screening techniques is low. The aim of any program is to discover individuals at risk for SCD during tough exercise without invasive testing and in a cost-effective manner. However, no generally established standards for screening are available at the present time. This issue was tackled differently in different countries. In the US, the pre-participation screening [5, 48, 51, 52] includes securing sports person’s personal and family history and complete physical examination (**Tables 3 and 4**) without an ECG or any other imaging studies and if abnormalities are detected in this initial screening process, further workup is pursued. In Italy [49, 53], Israel [54], and Japan [55], an ECG is also performed along with history and physical examination; this is mandated by the respective country’s laws. Screening of the athletes in other European countries is limited to the individuals participating in international, Olympic, or other professional sports [56]. Denmark, on the other hand

Personal history
1. Chest pain or discomfort during exertional activities
2. Unexplained syncope or near-syncope
3. Excessive exertional and unexplained dyspnea/fatigue or palpitations associated with exercise
4. Previously recognized heart murmur
5. Elevated systemic blood pressure
6. History of prior restriction from participation in sports
7. Results of prior testing of the heart by another physician/healthcare giver
Family history
8. History of premature death (sudden and unexpected, or otherwise) prior to age 50 years secondary to a cardiac issue in more than 1 relative.
9. Disability from heart disease in close relatives less than 50 years of age
10. History of hypertrophic or dilated cardiomyopathy, long-QT syndrome or other ion channelopathies, Marfan's syndrome, clinically significant arrhythmias, or genetic cardiac conditions in family members

Table 3.
Personal and family history [5, 48, 51, 52].

Physical examination
11. Complete physical examination including blood pressure in a sitting position *
12. Palpation of femoral pulses to exclude coarctation of the aorta
13. Look for physical stigmata of Marfan's syndrome
14. Careful auscultation to exclude left ventricular outflow tract obstruction **

**The numbering continues from Table 3.*
***Auscultation should be undertaken with the athlete in both the supine and standing positions (and with Valsalva maneuver) particularly to discover murmurs associated with HCM (dynamic left ventricular outflow tract obstruction).*

Table 4.
Pre-participation physical examination [5, 48, 51, 52].

completely rejected systematic screening for the athletes; their stated justification was a low event rate [57].

Some investigators employed screening studies utilizing ECG [58–61], echocardiogram [61–64], or MRI [65], and these investigators demonstrated that the prevalence of high-risk cardiac condition that is likely to predispose to SCD following exercise is low: ECG—0.33 to 11.5%; echocardiogram—1.26 to 5.1%; and MRI—1.25% of the athletic population screened [58–65].

The current recommendations by the AHA and ACC [5, 48, 51, 52] are to examine the personal history and family history (Table 3) and to carry out an orderly and complete physical examination (Table 4), whether it is undertaken in primary care doctors' clinic or in mass pre-participation screening programs. The rationale of the pre-participation screening is not to exclude youths from participation in sports, but to enable as many of them as possible to participate in sports to their full potential.

As mentioned above, the present AHA/ACC suggestions are that screening assessments are executed by trained examiners. This appraisal should consist of the 14-key

elements of personal and family history and physical examination (**Tables 3 and 4**). Such assessment should be carried out in a setting favorable for best auscultation of the heart. These all-inclusive screening assessments should be re-done in two years for high school athletes and in three years for the college student athletes. It is not realistic to presume that usual large-scale screening assessments are capable of excluding all clinically pertinent diseases. The writer of this chapter strongly believes that such appraisals should be undertaken by the primary care doctors (pediatricians, internists, family practitioners, and other primary care providers who care for children, adolescents and young adults) as a part of their annual regular physicals, but they should make sure that the AHA/ACC suggested 14-key elements of personal and family history and physical examination (**Tables 3 and 4**) are included.

If abnormalities are detected in the previously expressed, 14-Element AHA/ACC recommendations (**Tables 3 and 4**) for pre-participation cardiovascular screening, additional testing, as suitable, should be undertaken (**Table 5**).

9.1 Electrocardiogram

ECG is the first test that should be performed if any abnormalities are detected in the 14-element screening (Routine pre-participation screening ECG is not recommended in the US and the justification for such will be reviewed in the next section of this chapter). The ECG may identify patients with HCM because, as mentioned in the HCM section, many patients with HCM exhibit ECG abnormalities although the ECG abnormalities are not diagnostic of HCM. The value of ECG in identifying coronary abnormalities is limited. However, the ECG is useful in detecting long QT syndrome, Wolf-Parkinson-White (WPW) syndrome, atrioventricular block, and Brugada syndrome.

9.2 Echocardiogram

Echocardiogram should be performed to investigate abnormalities detected during history taking, performing a physical examination, or unexplained ECG findings. However, it is absolutely impracticable to use the echocardiogram as a screening tool for SCD [2 22]. Hypertrophic cardiomyopathy (**Figures 1–9**) and other types of cardiomyopathies can easily be diagnosed by echo-Doppler studies. Aberrant CAs should be specifically imaged in an attempt to document normalcy (**Figures 10–12**) or to establish aberrancy (**Figures 13 through 17**). Echo is also useful in quantifying dilatation of the aortic root (**Figures 20 and 21**) in Marfan's syndrome. The presence of prolapse of the mitral valve and mitral valve insufficiency (**Figure 19**) can be detected easily in echo-Doppler studies.

9.3 Other studies

A number of other studies may be useful in investigating these patients and they should focus on specific issues/concerns discovered during the screening process (**Table 5**). Exercise testing may help resolve the significance of exercise-induced symptoms. Holter or Event monitors may be used to document arrhythmias. An invasive electrophysiology study may be needed in some children. Genetic screening for HCM, Marfan's syndrome, and long QT syndrome is currently available and may be performed as indicated for a given clinical scenario. MRI and CT scans may be

1. Secure careful personal and family history (Table 3)
2. Perform methodical pre-participation physical examination (Table 4)
3. Obtain a 12-lead electrocardiogram if any abnormalities are detected during history and/or physical examination (items 1 and 2)
4. If abnormalities are detected during history taking and physical examination or if unexplained ECG findings are seen, an echocardiogram should be performed. Particular focus should be paid to address the type of abnormality detected during items 1, 2, and 3
5. If problems are detected during items 1 through 4, additional studies, specifically addressing the detected abnormalities, are in order: <ul style="list-style-type: none">• Exercise testing if exercise-induced symptoms were noted• Holter or Event monitors may be used to document arrhythmias• Genetic screening for HCM, Marfan's syndrome, and long QT syndrome, if indicated• MRI and CT scans may be performed to address issues not resolved by an echo• Cardiac catheterization and cineangiography are rarely needed
CT, Computed tomography; HCM, Hypertrophic cardiomyopathy; MRI, magnetic resonance imaging.

Table 5.
Stepwise approach to cardiac screening evaluation.

employed in specific situations, not resolved by echocardiography. Cardiac catheterization and cineangiography are rarely needed.

Given the easy accessibility of echocardiography and its utility in the detection and quantification of causes of sudden death in athletes (as reviewed in Sections 2–8 of this chapter), echocardiography has become the primary mode of investigation to address issues identified during history, physical examination and ECG screening. However, in subjects with poor echo windows, noninvasive studies such as MRI and CT scans may be utilized. These studies are particularly useful in evaluating aortic arch and pulmonary artery anomalies. Another condition in which MRI is useful is arrhythmogenic right ventricular cardiomyopathy; indeed, MRI criteria forms the basis for diagnostic confirmation of ARVC. In patients with Marfan syndrome, aortic dilatation can be quantitated and aortic dissection detected by MRI. MRI and CT scans are also useful in evaluating anomalous origin and course of the coronary arteries as well in assessing other coronary artery abnormalities listed in Table 2.

10. Routine ECG as a screening tool

The Task Force on Preparticipation Screening for Cardiovascular Disease in Competitive Athletes of AHA/ACC [5, 48, 51, 52] does not recommend routine ECGs and echocardiograms for pre-participation screening. On the other hand, the International Olympic Committee [66] and the European Society of Cardiology [67] guidelines recommend routine screening with a 12-lead ECG along with history and physical examination. Pre-participation screening ECG does not increase the diagnostic accuracy, is not practical, is not sensitive, and is not specific. In addition, both false positives and false negatives (5–20%) exist with the ECG [68]. However, it may detect patients with hypertrophic cardiomyopathy, WPW syndrome, atrioventricular block, long QT syndrome, and Brugada syndrome.

The justification for AHA/ACC guidelines for not recommending routine recording of ECGs as a part of pre-participation screening may be summarized as follows: 1. ECG is not sensitive and is not specific with false-positive ECGs taking place way

above true-positive ECGs, 2. The incidence of cardiac conditions leading to sports-related deaths is rather low (5 out of 100,000 subjects), 3. The athlete group to be screened is of the considerable size (10 million in the USA and much larger worldwide), 4. Routine ECG screening will impose a large price tag of roughly 2 Billion dollars/year, and 5. There is no adequate physician pool to do and interpret this large number of ECGs. Furthermore, the subjects with undiagnosed cardiac abnormality may present with symptoms, namely chest pain, exertional dyspnea, or syncope which may be uncovered by the screening questionnaire. Since some of the entities have a genetic and familial origin, they may be discovered by the screening protocol. Finally, studies comparing the strategies with and without ECG during screening did not demonstrate a mortality benefit in the group with routine ECGs [52, 54, 69]. Based on these and other considerations, the author is in support of not performing ECG during pre-participation screening [3] and recommends careful attention to implementing the 14-point AHA/ACC pre-participation screening protocol.

11. Summary and conclusions

Sudden death associated with sports participation often has a cardiovascular cause and the two most frequent etiologies are HCM and aberrant CAs. Clinical features of HCM, aberrant CAs, Marfan's syndrome, and ARVC were reviewed and other entities responsible for SCD were listed/tabulated. The existing recommendations are a pre-spots participation review of full personal and family history and systematic physical examination, preferably in the primary care doctors' office. Additional investigative studies should be undertaken if history or physical examination detect any abnormalities. Using ECG, echocardiogram, or MRI as routine screening techniques is contentious and is not currently recommended in the USA. The rationale of pre-sports participation evaluation is to allow as many athletes as feasible to partake instead of being excluded from sports participation.

Conflict of interest

The author declares no conflict of interest.

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