REVIEW ARTICLE

Sports Med 1998 Nov; 26 (5): 335-350
0112-1642/98/0011-0335/$08.00/0

© Adis International Limited. All rights reserved.

Sudden Death in Athletes
An Update

Laurie G. Futterman and *Robert Myerburg*

School of Medicine, University of Miami, Jackson Memorial Medical Center, Miami, Florida, USA

Contents

Abstract

The athlete projects the ultimate image of well-being in the health status spectrum. Nevertheless, exercise-related sudden cardiac death (SCD) is an uncommon, yet tragic, occurrence. Exercise-related SCD is defined by symptoms that arise within 1 hour of participation in sport. The major mechanisms involved in exercise-related SCD are related to haemodynamic and electrophysiological changes brought about by exercise in the susceptible individual. Fatal arrhythmia seems to be the most common mechanism of death. Between 1 and 5 cases of SCD per 1 million athletes occur annually. In young athletes (<35 years old), the majority of these cases are caused by defined and hereditary cardiovascular disorders. Among other aetiologies, hypertrophic cardiomyopathy and coronary artery anomalies are most common in this group. In older athletes (>35 years old), sudden death is usually associated with atherosclerotic cardiac disease.

A problem for identifying athletes at risk for SCD is that the athlete's heart undergoes adaptive changes in response to regular physical exercise. Alterations

in cardiac function influence the physical examination, the electrocardiogram and the echocardiogram. Because of these characteristic 'abnormalities' of the athlete's heart, it is often difficult to distinguish physiological adaptations from pathophysiological processes.

Although studies and observations have helped to clarify the cardiovascular pathology responsible for SCD in young, apparently healthy individuals, effective methods for preventing SCD and identifying and screening athletes at risk remain elusive. Problems with routine comprehensive screening of athletes include the limitations inherent in the predictive value of available diagnostic procedures and the cost of testing large populations. The variation from normal cardiac physiology found within the athletic population and the rarity of SCD in athletes means that elaborate screening to determine individuals at risk is neither practical nor cost effective. A thorough assessment of pertinent family and medical histories, cardiac auscultation of young athletes, evaluation of exercise-induced symptoms and education of older athletes to the symptoms of cardiac ischaemia are all essential to primary prevention of SCD in the athletic population.

Until reliable methods can accurately identify those athletes at risk for SCD, broad recommendations are available to help guide the management and participation in sports of athletes with cardiovascular disease.

In the US and elsewhere, society perceives the athlete to be strong, fast and physically talented, projecting the ultimate image of well-being in the health status spectrum. Yet, a small proportion of athletes die suddenly and unexpectedly during competition or training. For coaches and athletes alike, this tragic scenario is a reminder of the need to examine the risks and benefits of competitive exercise. Fortunately, sudden cardiac death (SCD) is uncommon, but it attracts attention when it affects a nationally known athlete, or a young, local, talented athlete. These incidents, however infrequent, have a tragic and devastating effect on the families and the community. A case report of such an event is described here.

A 20-year-old Black student visited the college clinic after deeply lacerating his left knee during track practice. He had become light-headed while running and had fainted. He regained consciousness after only a few seconds. On physical examination, a systolic murmur was heard over the aortic area, medial to the apical impulse. Although the runner did not recall any history of a murmur, he acknowledged that both his father and uncle had died at early ages. He denied having any health problems. His injury was treated and he was released. The fainting spell was attributed to 'heat exhaustion'. The following year, he collapsed during the first heat of the one-quarter mile qualifying trials. He remained unresponsive and pulseless despite rapid initiation of cardiopulmonary resuscitation by a bystander and subsequent advanced cardiac life support intervention. Although ventricular fibrillation was observed on the electrocardiogram monitor, direct current defibrillation attempts and pharmacological intervention failed. He was pronounced dead on arrival at the University's medical centre emergency department. Autopsy revealed obstructive hypertrophic cardiomyopathy.

Sudden death syndrome, a primitive term initially used by some to describe a sudden, unexpected death, symbolised the innocence of the medical community to its exact causes.[1] The earliest description of and curiosity about sudden death dates back to the unexpected death of the Greek soldier Pheidippides, a conditioned runner, after he completed a run carrying military information from Marathon to Athens in 490BC.[2,3]

Continued research and observations have uncovered and clarified many underlying cardiovascular diseases responsible for sudden death in highly trained athletes and other apparently healthy young individuals. However, the screening and identification of athletes at potential risk for sudden death continues to elude us. Methods for pre-

© Adis International Limited. All rights reserved.

venting SCD in competing athletes remain the burden of health professionals; however, the identification of individuals at high risk for SCD is a shared responsibility of health professionals, athletic teams and schools.

SCD is defined as nontraumatic, nonviolent, unexpected death due to cardiac causes within 1 hour of the onset of symptoms (witnessed event), or within 6 hours of witnessed normal state of health (unwitnessed event).[4] Sports-related deaths are defined as those with symptoms occurring within 1 hour of sports participation.[1-19] SCD is often observed after competitive activity where a high priority is placed on excellence and achievement. In the attempt to assign a mechanism of aetiology, SCD has been classified as follows: (i) certain SCD (obvious anatomical evidence at autopsy, such as myocardial infarction or ruptured aorta); (ii) probable SCD [left ventricular (LV) hypertrophy without definite criteria for hypertrophic cardiomyopathy (HCM)]; and (iii) presumptive SCD (floppy mitral valve).[2]

1. Cardiovascular Adaptation to Exercise

The major mechanisms involved in SCD are related to haemodynamic and electrophysiological changes brought about by exercise in the susceptible individual. Changes in myocardial structure, function and blood flow can affect the cardiac response to exercise. Haemodynamic fluctuations in the normal myocardium that occur with exercise vary with the type of exercise performed. Exercise also affects the electrical function of the heart as changes in sympathetic stimulation occur.[14]

Although sports are categorised with regard to type and intensity, the mechanical action involved and the metabolism requirements inherent in each sport determine further classification as dynamic or static, and aerobic or anaerobic (table I).[5]

Dynamic exercise requires large muscle mass use and causes a marked increase in oxygen consumption as it generates a volume load on the ventricle. As a result, dynamic (isotonic) athletic activities result in larger absolute LV mass and chamber size (eccentric hypertrophy), associated with high oxygen uptake. The adaptations brought about by dynamic volume challenges to the heart include:[3,5,6]

- increased heart rate, systolic blood pressure, ventricular stroke volume, LV end-diastolic volume
- increased LV end-diastolic diameter
- decreased peripheral vascular resistance
- increased maximum oxygen uptake ($\dot{V}O_{2max}$).

LV mass-to-volume ratio is unchanged, despite ventricular wall thickening.

Static exercise, as it generates a pressure load on the ventricle, produces only small increases in oxygen consumption, cardiac output and heart rate, and no change in stroke volume. Static (isometric) athletic activities cause cardiovascular adaptations to pressure fluctuations. Since static activities produce little change in venous return, the LV end-diastolic diameter does not change.

In response to a higher afterload, the ventricular wall thickens to maintain normal wall tension and the LV mass-to-volume ratio increases. Athletes who participate in sports with a high static component generate large LV masses without an increase in chamber size (concentric hypertrophy), which is not associated with high oxygen uptake. Adaptations to isometric training include:

- increased LV mass
- no significant increase in $\dot{V}O_{2max}$
- no increase in LV end-diastolic diameter
- increased LV mass-to-volume ratio.

Aerobic and anaerobic classifications refer to the basis of metabolism involved with the activity, with most high intensity static exercise being anaerobic and most sustained, high intensity dynamic exercise being aerobic.[5]

Sympathetic stimulation, with increases in circulating catecholamines, is generated by the competitive stress and emotion, in addition to the physical exertion. These factors exaggerate cardiac responses such as blood pressure, heart rate, and myocardial contractility, which lead to increased myocardial oxygen demands. Enhanced sympathetic activity alone can promote cardiac

© Adis International Limited. All rights reserved.

Table I. Classification of sports based on peak dynamic and static components during competition (reproduced from Mitchell et al.,[5] with permission)

Low dynamic	Moderate dynamic	High dynamic
Low static		
Billiards	Baseball	Badminton
Bowling	Softball	Cross-country skiing (classic technique)
Cricket	Table tennis	Field hockey[a]
Curling	Tennis (doubles)	Orienteering
Golf	Volleyball	Race walking
Riflery		Racquetball
		Running (long distance)
		Soccer[a]
		Squash
		Tennis (singles)
Moderate static		
Archery	Fencing	Basketball[a]
Auto racing[a,b]	Field events (jumping)	Ice hockey[a]
Scuba diving[a,b]	Figure skating[a]	Cross-country skiing (skating technique)
Equestrian[a,b]	Football (American)[a]	Football (Australian rules)[a]
Motorcycling[a,b]	Rodeo[a,b]	Lacrosse[a]
	Rugby[a]	Running (middle distance)
	Running (sprint)	Swimming
	Surfing[a,b]	Team handball
	Synchronised swimming[b]	
High static		
Bobsledding[a,b]	Body-building[a,b]	Boxing[a]
Field events (throwing)	Downhill skiing[a,b]	Canoeing/kayaking
Gymnastics[a,b]	Wrestling[a]	Cycling[a,b]
Karate/judo[a]		Decathlon
Luge[a,b]		Rowing
Sailing		Speed-skating
Rock climbing[a,b]		
Waterskiing[a,b]		
Weight-lifting[a,b]		
Windsurfing[a,b]		

a Danger of bodily collision.
b Increased risk if syncope occurs.

arrhythmia or aggravate underlying myocardial ischaemia. Extreme environmental temperatures and altitude changes also have the potential to increase myocardial work. Thus, sports that generate relatively low exercise-related myocardial oxygen demand but involve emotional components (such as golf or riflery), or exposure to environmental variables (such as scuba diving and altitude climbing) may still significantly increase myocardial oxygen demand and risk of SCD in the susceptible athlete.[5]

2. Physiological Hypertrophy: The Athlete's Heart

Although the concept of the athlete's heart has been appreciated for over 100 years, it is only recently that noninvasive imaging has permitted some degree of definition and quantification.[6]

Cardiovascular adaptation to regular physical exercise leads to morphological transformation of the myocardium. These alterations influence the physical examination, electrocardiogram (ECG) and echocardiogram. Pioneering observations in

© Adis International Limited. All rights reserved.

exercise physiology suggested a difference in the hearts of wild animals when compared with their domestic counterparts and in cross-country skiers when compared with their sedentary counterparts.[8] Physiological hypertrophy was first demonstrated in 1935 and it has since been confirmed that several specific cardiovascular adaptations exist in well-conditioned athletes.[8]

Athletic heart syndrome, considered a variant of the normal heart, is quite prevalent and varies according to training regimens and type of sport. It comprises various cardiophysiological changes caused by regular exercise training and is defined noninvasively, using ECG, physical examination and echocardiography, by precise alterations in cardiac dimensions.[3,6,8] LV hypertrophy correlates with the degree of conditioning, increasing with training and decreasing with deconditioning. Unlike pathological hypertrophy, physiological hypertrophy is symmetrical and tends to plateau early after the start of a conditioning programme. Within weeks of inactivity, cardiovascular deconditioning occurs. LV mass (as well as LV end-diastolic diameter and LV wall thickness) has been reported to decrease by 20%, to become comparable to baseline sedentary controls.[8]

The ability to clearly distinguish adaptive physiology from pathophysiology of cardiovascular disease remains a constant challenge in order to assure that physiological changes of the athlete's heart are recognised. A precise diagnosis may protect a vulnerable athlete in one case, while avoiding unnecessary restriction and withdrawal from athletics in another. Unfortunately, many athletes fall into a diagnostic 'grey zone', and are subject to misdiagnosis and its consequences.[3,6]

3. Prevalence and Aetiology of Sudden Cardiac Death (SCD)

The overall incidence of SCD during athletic activity remains low. Of the 25 million competitive athletes in the US, it has been estimated that between 1 and 5 cases of SCD occur per 1 million athletes annually.[3] The most common age for SCD in athletes is in the late teens (high-school athletes), although SCD occurs in collegiate and professional level athletics as well.[7,17] The occurrence rate of athletic deaths in high-school–age athletes is not exactly known, but has been estimated to be in the range of 1 : 100 000 to 1 : 300 000.[7] The exact lesions responsible for athletic deaths differ with the age of the athlete. Although uncommon in the population, the majority of exercise-related deaths in young athletes (<35 years old) are congenital and cardiovascular in nature, the most common being HCM (46%) followed by coronary artery anomalies (19%).[1,2,6,7] The remaining deaths are caused by myocarditis (7%), ruptured aortic aneurysm (Marfan's syndrome) (5%), aortic valve stenosis (6%), dilated cardiomyopathy (6%), mitral valve prolapse (2%), arrhythmogenic right ventricular dysplasia (ARVD) (most common cause of SCD in northern Italy) [3%], myocardial bridges (5%), conduction system abnormalities [accessory atrioventricular (AV) pathways], cell membrane disease (long QT syndrome) and commotio cordis.[1,2-12] In contrast, the majority of deaths in older athletes (>35 years old) are attributed to coronary artery disease.[1,2-12]

The magnitude of SCD risk in athletes is determined by the nature and intensity of the athletic training (a potential trigger mechanism) and the status of the patient's cardiomyopathic disease.[1] Individuals with HCM, ARVD, or aortic stenosis tend to be at greater risk for SCD during and immediately after intense exercise.[13] It appears that close to 90% of athletic-field deaths occur in men. A lower frequency of deaths in females may be the result of their lower overall participation level and intensity of training. Structural cardiovascular diseases that account for SCD seem to affect women less frequently.[3]

Noncardiac causes of death have been attributed to cerebrovascular accident, heat stroke, pulmonary disease, peripheral embolism and drug abuse.[8]

3.1 Hypertrophic Cardiomyopathy

Of the difficulties in distinguishing a normal adaptive athlete's heart from one with structural heart disease, the distinction between HCM and

© Adis International Limited. All rights reserved.

physiological hypertrophy is the most challenging; distinguishing characteristics may not be clear. Differentiating physiological from pathological hypertrophy can be perplexing when athletes fall into a morphological 'grey zone' where the degree of ventricular thickness (13 to 15mm) does not reach that for HCM (>15mm) [fig. 1].[1,3,6,8] Clinical diagnosis of HCM in individuals who survive an exercise-related cardiac event is often based on history, physical examination, 12-lead ECG and 2-dimensional echocardiography with Doppler analysis.

Although HCM is infrequent (the overall prevalence is 0.1 to 0.2% in the US population), its significance is illustrated by the fact that it remains the most common (50% or more) cause of unexpected death in competitive athletes and is among the most common causes of sudden cardiac death in unselected adolescent and young adult populations.[2,3,8,13,14,18,19] Only myocarditis may be more common in selected groups. SCD is often the initial presentation of HCM, particularly in the young. HCM may be genetically transmitted; other cases occur sporadically. This primary muscle disease is associated with a number of genetic defects, the majority of which affect contractile proteins or noncontractile sarcomere proteins. A few abnormalities affect the muscle 'scaffolding' itself. LV outflow obstruction and malignant arrhythmogenesis appear to be the main mechanisms of death. Unlike SCD of non-athletes, most of the fatal events in athletes with HCM occur in the afternoon and early evening hours when activity levels are highest.[13]

In contrast to the vast majority of competitive athletes whose LV wall thickness is essentially normal, or only mildly increased, HCM is visualised echocardiographically by a markedly asymmetrical nondilated and hypertrophied (15 to 50mm wall thickness) left ventricle (in the absence of any other cardiac or systemic disease). The hypertrophied muscle reduces the volume and chamber size of the left ventricle and impairs diastolic filling (diastolic dysfunction). Some individuals with HCM experience dynamic obstruction to LV outflow from systolic anterior motion of the mitral valve leaflet, although this subset of individuals account for only a small percentage of fatal HCM cases. LV outflow tract obstruction in HCM, or myocardial ischaemia resulting from inadequate intramural coronary

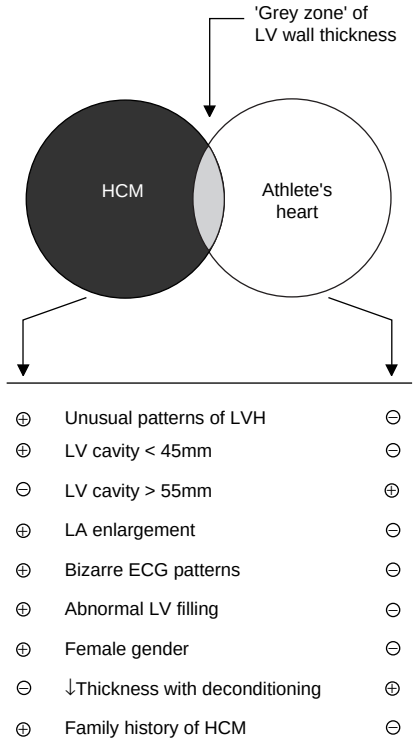

Fig. 1. Differentiating hypertrophic cardiomyopathy (HCM) from the athlete's heart: the 'grey zone' of left ventricular (LV) wall thickness. The above chart shows criteria used to distinguish HCM from the normal adapted heart of an athlete when the left ventricular wall thickness is within the shaded grey zone of overlap, i.e. consistent with both diagnoses. In this model, HCM is assumed to be in the non-obstructive form, since the presence of substantial mitral valve systolic anterior motion would confirm, *per se*, the diagnosis of HCM in an athlete. The criterion of unusual patterns of LVH may involve a variety of abnormalities including heterogenous distribution of LVH in which asymmetry is prominent and adjacent regions may be of greatly different thicknesses, with sharp transitions evident between segments. There may also be patterns in which the anterior ventricular septum is spared from the hypertrophic process and the region of predominant thickening may be in the posterior portion of septum or anterolateral or posterior free wall (reproduced from Maron et al.,[6] with permission). **ECG** = electrocardiogram; **LA** = left atrial; **LVH** = left ventricular hypertrophy; ↓ = decreased; **+** = positive criterion; **–** = not a positive criterion.

© Adis International Limited. All rights reserved.

blood flow, further increases the potential for lethal arrhythmia. Unfortunately, SCD may be the first and only symptom of HCM.[1,6,8,15,16]

3.1.1 Distinguishing between HCM and Physical Hypertrophy

The following list contrasts a number of features in HCM with those in the highly trained athlete with physiological hypertrophy.

Auscultatory Findings

- HCM: a systolic murmur at the lower left sternal border medial to the apex, increasing with manoeuvres that decrease venous return to the heart.
- Physiological hypertrophy: S1 may be normal, although soft, systolic ejection murmurs may be appreciated at the base; wide splitting of S2; third heart sound is common, owing to greater diastolic filling rates; fourth heart sound may be heard as a result of vigorous atrial contractions in individuals with thin chest walls.

Echocardiographic Findings

- HCM: marked, asymmetrical LV wall thickening (septum disproportionately greater than the LV free wall, with an average thickness of ≥20mm); diminished LV cavity size (<45mm) except in the end-stage phase of HCM; systolic anterior motion of anterior leaflet of the mitral valve; diastolic dysfunction (abnormal filling pattern).
- Physiological hypertrophy: LV wall thickening usually <12mm (but up to 16mm) not limited to the septal wall; enlarged LV end-diastolic cavity size (>55mm) is common; normal diastolic filling patterns. Isotonically trained athletes typically demonstrate increased LV cavity size and mass and septal wall thickness (usually not exceeding 12 to 15mm), whereas isometrically trained athletes do not demonstrate increased LV wall thickness beyond 12mm. Trained female athletes, as compared with male athletes, do not demonstrate typical LV wall thickening.

Electrocardiographic Findings

- HCM: dramatically increased QRS voltage suggesting LV hypertrophy with ST-T wave pattern of LV strain is most common; some patients have deep septal Q waves; deeply inverted T waves in precordial leads; ventricular tachycardia or paroxysmal atrial fibrillation.
- Physiological hypertrophy: ECG variants are chiefly related to enhanced vagal tone: (i) early repolarisation changes of ST segment elevation and T wave narrowing are seen in 50% of cases; (ii) resting sinus bradycardia (as low as 25 beats/minute) and sinus arrhythmia. Vertical axis, right bundle branch block, nonspecific T wave inversion and slight QT prolongation are also common. Although AV conduction delays (first, second and third degree) and sinus pauses have been reported in a small percentage of athletes, care should be taken to exclude underlying pathology. Horizontal or down-sloping ST segment depressions are not considered normal findings.

Generally, nonspecific variations of the 12-lead ECG in both HCM and athletic hypertrophy do not facilitate a distinction between HCM and physiological hypertrophy.

Other Assessments

Ultrasonographic tissue characterisation incorporates the use of background scatter to distinguish physiological from pathological hypertrophy. Currently its use is limited to select research centres. In HCM, there is increased intensity of ultrasonographic signal results from higher levels of cellular disarray and collagen, which creates greater background scatter.[6,8] In physiological hypertrophy, normal tissue reflectivity is demonstrated.[6]

Histological disarray of myocyte architecture, increased number of abnormal intramural coronary arteries with thickened walls and narrowed lumens are characteristic of HCM.

Electrophysiological testing is not considered reliable for predicting unselected arrhythmic events or SCD in the heterogenous HCM population.

3.2 Coronary Artery Anomalies

Coronary artery anomalies that presumably lead to myocardial hypoperfusion during exercise, are the second most common cause of exercise-induced

© Adis International Limited. All rights reserved.

SCD in young athletes (12 to 14% of SCD cases), although they are rarely diagnosed during life. Of the variety of non-atherosclerotic, congenital malformations, the most common coronary anomaly associated with SCD is an anomalous origin of the left main coronary artery from the right anterior sinus of Valsalva (fig. 2).[1,3]

The mechanism by which sudden cardiac death occurs is not understood completely, but has been attributed to the take-off angle of the left main coronary artery from the right sinus of Valsalva. It has been postulated that this acute angle creates a narrowing of the coronary ostium, which with exercise-induced aortic dilatation, further narrows the take-off angle of the left main coronary artery. This further compromises the ostial orifice and reduces coronary artery blood flow. Myocardial perfusion is also affected if the anomalous left main coronary artery traverses between the aorta and pulmonary trunk, where it may become compressed during exercise when the great vessels expand.[1,3,6,15]

Some individuals with coronary artery anomalies demonstrate tell-tale symptoms (syncope or angina) prior to an event. A high index of suspicion (using cross-sectional 2-dimensional echocardiography) may help to suggest the presence of the anomaly so that coronary arteriography can be obtained and confirm an often surgically correctable abnormality.

Single coronary arteries, coronary artery hypoplasia and pulmonary artery serving as the origin for the coronary arteries have also been associated with SCD.

3.3 Arrhythmogenic Right Ventricular Dysplasia

ARVD is a right ventricular myopathic process characterised by fatty infiltration and fibrosis, the cause(s) of which remains unclear. It is associated with structural and functional abnormalities, arrhythmias and SCD. In 25 to 30% of cases, ARVD is familial.[1,3,10,19,21] The first clinical sign of ARVD is often the occurrence of right ventricular tachycardia, frequently triggered by exercise. ARVD was reported to be an important cause of death among young athletes from a northeastern region of Italy,[6,8] where the country's cardiovascular assessment programme of athletes has effectively reduced the number of individuals with HCM competing in its athletic events. This geographical finding is thought also to correlate with a presumed genetic basis of ARVD.[6,8] ARVD is characterised by focal or widespread replacement of normal right ventricular myocardium by adipose and fibrous tissue. This process results in the thinning and dilatation of the right ventricular wall and leads to recurrent and intractable ventricular (or supraventricular) arrhythmias. Other than transient exercise-induced ventricular arrhythmia, with subsequent weakness, palpitations, or severe effort-related syncope, there may be very few symptoms.

The diagnosis of ARVD can be aided by the following findings:

- Physical findings: although the most frequent physical finding in ARVD is a widely split S2 caused by prolonged right ventricular ejection, physical findings in 50% of this population are essentially unremarkable.
- ECG changes in the majority of cases show a negative T wave from precordial leads V_1-V_3. An even larger number of individuals demonstrate re-entrant dysrhythmias manifested by polymorphic premature ventricular contractions (PVCs) [left bundle-branch block morphology].
- Chest roentgenogram may reveal right ventricular enlargement.
- Echocardiographic changes demonstrate a large, poorly contractile right ventricle with thin walls.

Diagnosis of ARVD does not usually occur until the patient develops symptomatic/sustained ventricular tachycardia. ARVD can be confirmed by endomyocardial biopsy (when specimens are acquired from the region of abnormal tissue) and right ventricular angiography.

Magnetic resonance imaging may detect abnormal fatty infiltration of the myocardium. Electrophysiological testing can help determine the site of arrhythmogenesis and efficacy of pharmacological therapy. Ultimately, an awareness of subtle changes

© Adis International Limited. All rights reserved.

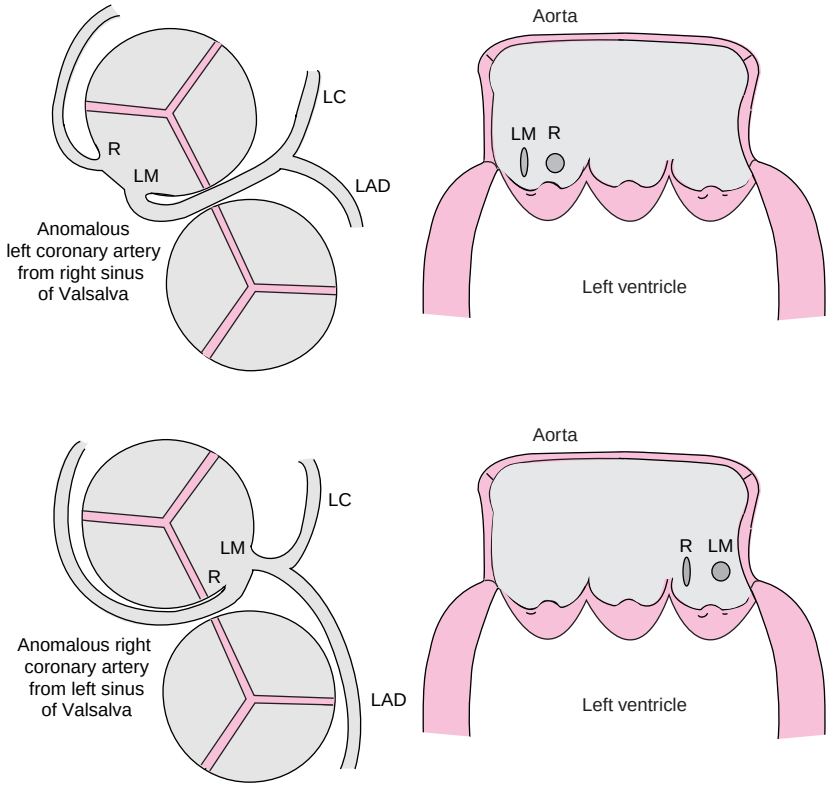

Fig. 2. Coronary artery anomalies associated with sudden cardiac death in athletes. The 2 most frequent congenital coronary artery anomalies associated with exercise-related sudden death involve the origins of both the left and right coronary arteries from the right or the left sinus of Valsalva. The coronary ostium of the anomalous vessel is slit-like compared with the normal oval shape (reproduced from Waller & Harvey,[20] with permission). **LAD** = left anterior descending coronary artery; **LC** = left circumflex coronary artery; **LM** = left main coronary artery; **R** = right coronary artery.

on the ECG and suspicion of the condition are vital for its detection.

3.4 Atherosclerotic Coronary Artery Disease

Atherosclerotic coronary artery disease and myocardial infarction is an infrequent cause of exercise-related cardiac deaths in young athletes. Atherosclerotic coronary artery disease is responsible for only 10% of SCD cases. Familial dyslipidaemia may be present.[1,3]

Although exercise dilates normal coronary arteries, it is thought to induce coronary artery spasm in atherosclerotic segments. High shear stress from physical exertion and increased systolic blood pressure, changes in cardiac dimensions during exercise, or contraction of a noncompliant atherosclerotic plaque may all trigger plaque disruption and rupture.

However, atherosclerotic disease is responsible for the majority (80%) of SCD cases in the mature athlete (>35 years old).[3] Many individuals with known risk factors for heart disease take up vigorous exercise regimens, without proper screening, as part of risk factor control programmes. Death in the older athlete is rarely caused by congenital abnormalities. The remaining cases of exercise-related cardiac death in older athletes are largely attributable to hypertrophic or dilated cardiomyo-

© Adis International Limited. All rights reserved.

pathies, or acquired valvular heart disease. Mitral valve prolapse has been suggested as a cause in some people, but this is conjectural.

3.5 Use and Abuse of Drugs

A careful clinical history may reveal a link between the use of pharmacological substances and SCD. Exposure to agents such as erythromycin, antihistamines and phenothiazines, especially during antifungal treatment with 'conazole' type agents, favours the development of torsade de pointes.[22] Many reported clinical cases of myocardial infarction have been associated with cocaine use.[9] As a cardiotoxin, effects of cocaine include impaired coronary blood flow and electrical derangement, and LV dysfunction. A link between alcohol (ethanol) and arrhythmogenic SCD has also been discussed.[22]

3.6 Aortic Rupture and Marfan's Syndrome

Aortic rupture, or rupture of an aortic aneurysm associated with Marfan's syndrome, is also a rare, but potentially preventable, cause of exercise-related cardiac death.[1-3,8,23] It is the result of an autosomal dominant disorder of connective tissue which affects several body systems. In Marfan's syndrome, the aortic media is deficient in the number of elastic fibres, which leads to weakening of the aortic wall (cystic medial necrosis) and predisposes the individual to aortic dissection and death.[24] Physical examination of these individuals may reveal long extremities, tall stature, skeletal and chest wall deformities and ocular abnormalities.[1,25]

3.7 Arrhythmic Mechanisms of SCD

Certain arrhythmias create symptoms and are dangerous despite the presence or absence of other clinical pathologies. These arrhythmias are generally associated with a very rapid or very slow heart rate that compromises haemodynamic function.[1,12,21] Of most concern are the following: atrial fibrillation associated with accessory atrioventricular (AV) pathways and pre-excitation syndromes (e.g. Wolff-Parkinson-White syndrome); long QT interval syndrome and its associated arrhythmias; rapid, sustained ventricular tachycardia; and, ventricular fibrillation. Atrial flutter or atrial fibrillation with rapid, uncontrolled ventricular rate may also be of concern in patients with disorders such as HCM, mitral valve stenosis and aortic valve stenosis and various coronary abnormalities. AV block or sinus node disease with a very slow ventricular rate rarely cause sudden death even in the presence of disease: they do, however, create risk for transient loss of consciousness, which during certain activities could lead to traumatic injury or death, e.g. tachycardia causing dizziness and transient impaired mental function during downhill skiing or auto racing.[5,23]

Primary electrical disease, in the form of idiopathic polymorphic ventricular tachycardia, has been considered responsible for some cases of sudden deaths in individuals with otherwise structurally normal hearts.[11,15] Reflex arrhythmogenesis, triggered by catecholamine release during physical or emotional stress may arise from abnormal vasodilating/cardioinhibitory responses (vasovagal, Bezold-Jarsch), placing vulnerable athletes at risk for SCD.

3.8 Myocarditis

Myocarditis is an acute inflammatory process, usually of viral aetiology (most often coxsackie B virus), that affects myocardial tissue and can compromise cardiac function.[1,3,26] It can also cause arrhythmias ranging from PVC and premature atrial contraction (most common) to atrial fibrillation or life-threatening ventricular arrhythmias (uncommon). The severity of arrhythmia does not seem to correlate with severity of myocardial dysfunction. *Chlamydia pneumoniae* myocarditis has also been noted in several SCD cases in Swedish orienteers.[27,28] Clinical and pathophysiological profiles similar to acute and healed myocarditis have been associated with long term cocaine use. The stage of the myocarditis and the timing of sudden arrhythmic death cannot always be correlated: acutely inflamed myocardium may serve as the

© Adis International Limited. All rights reserved.

arrhythmogenic nidus. There has also been suggestions that healed myocarditis with interstitial fibrosis may be arrhythmogenic. Myocarditis is not considered among the most common causes of SCD in young competitive athletes. Despite this, it has been suggested to be among the more common causes of SCD in non-athletes in similar age groups.

Most individuals with myocarditis are asymptomatic and unaware of their disease, but the disorder may be suspected when presentation includes fatigue, exercise intolerance, palpitations, or clinical evidence of arrhythmia or acute congestive heart failure. A systemic viral syndrome often precedes and may dominate myocardial symptoms. Physical examination may reveal early markers of heart failure, chest radiography may suggest cardiomegaly, and ECG may reveal sinus tachycardia with low voltage. Endomyocardial biopsy and histological examination of immunochemically stained myocardium may clarify the diagnosis, although patchy involvement may yield low sensitivity. Polymerase chain reaction gene amplification may, in the future, facilitate the identification of the viral genome from biopsy samples, especially in borderline cases.

3.9 Myocardial Bridges

Myocardial bridge refers to an area of LV myocardium that creates a tunnel completely surrounding a segment of coronary artery.[1] Although coronary artery filling takes place primarily during diastole, ischaemia may result during exertion or stress when the coronary artery (usually the left anterior descending), completely surrounded by myocardium, undergoes a critical degree of systolic compression. In addition, diastolic flow may be impaired because increased heart rate encroaches proportionately more on the diastolic filling period than on systolic flow time. This may lead to myocardial ischaemia, resulting from the increased oxygen demand of the faster-beating heart. Myocardial bridges may result in SCD on very rare occasions: bridges rarely produce clinical evidence of myocardial ischaemia and are often seen during autopsies, regardless of the cause of death.[1,5,12]

3.10 Mitral Valve Prolapse

Mitral valve prolapse is rarely associated with exercise-related cardiac deaths in athletes, despite its prevalence in the population.[3,6,16]

3.11 Aortic Valve Stenosis

Aortic valve stenosis is another congenital malformation that is considered a risk factor for SCD in athletes, although not a direct cause. Owing to its characteristically loud, harsh systolic murmur, this lesion often prompts medical attention and, depending on severity, leads to disqualification of athletes from competitive events.[1,3,6,15]

3.12 SCD in Apparently Normal Hearts/Idiopathic Ventricular Fibrillation

On autopsy, the majority of individuals with cardiac arrest demonstrate some type of cardiovascular structural abnormality; yet in 5% of cases, no evidence of structural abnormality is found. Despite careful evaluation, conditions such as focal cardiomyopathy, myocarditis or fibrosis, as well as transient electrolyte imbalances, obscure long QT syndromes, or HCM without classic phenotypical findings, may be asymptomatic and persist without notice.

As a result of the frequency of sudden arrhythmogenic death in patients with apparently normal hearts, a registry of patients surviving an episode of idiopathic ventricular fibrillation was developed.[27] Idiopathic ventricular fibrillation should be a diagnosis of careful exclusion. Idiopathic ventricular fibrillation implies the inability to establish a link between clinical and historical information and a life-threatening event when known conditions (substance use/abuse, ischaemic heart disease, cardiomyopathy, ARVD, ventricular preexcitation, myocarditis, prolonged QT interval, idiopathic ventricular tachycardia, sudden unexplained nocturnal death syndrome, infiltrative disease) have been ruled out.[3,10,12,21,22] Subtle and nonspecific findings (electrocardiographic, echocardiographic, biochemical) in survivors warrant

© Adis International Limited. All rights reserved.

investigation, although their significance remains unclear.

3.13 Commotio Cordis

Commotio cordis, or cardiac concussion, is a rare mechanism of instantaneous cardiac death that occurs in individuals free from structural cardiac disease.[16,17,29] Although the precise mechanism of death is not certain, it is thought to result from dysrhythmia (ventricular fibrillation, bradyarrhythmia) induced by an exquisitely timed, nonpenetrating precordial blow (for instance, caused by projectiles such as ice hockey pucks or baseballs) to the chest wall during an electrically vulnerable phase of ventricular repolarisation.[16,17] It is thought that mechanical energy transmitted from the projectile to the myocardium initiates an electrical impulse, sufficient to trigger a fatal ventricular arrhythmia. In contrast to other types of athletic deaths, this occurs in structurally normal hearts without injury to the myocardium, cardiac valves, or coronary arteries. Other theories linking blunt chest impact to SCD include sudden alteration in coronary blood distribution, or a transient but profound vasovagal reflex.[1,2,16,17] Commotio cordis is associated with a remarkably low rate of successful resuscitation.

Although studies have been carried out to determine the risk from individual projectiles (e.g. types of baseball), preventive coaching, recognition of the syndrome and early resuscitative measures are more likely to help avoid such catastrophic deaths.[29,30] Commotio cordis should also be considered after motor vehicle accidents when there is no other apparent injurious cause of death.[32]

3.14 Exertion-Induced Rhabdomyolysis with Sickle Cell Trait

Exertion-induced rhabdomyolysis associated with sickle cell trait has been noted in a small number of exercise-related deaths. It is thought that exertion in physically untrained individuals triggers hypoxia, lactic acidosis and red blood cell sickling.

3.15 Other Causes of SCD

Other infrequent diseases of the myocardium that may contribute to the incidence of SCD in athletes include: primary restrictive cardiomyopathy (endomyocardial fibrosis) and systemic cardiac processes such as sarcoidosis. Some cardiac diseases are associated with abnormal structure at a molecular level and cannot always be recognised by the pathologist. An example is the long QT interval syndrome which usually can be diagnosed by ECG before death and, in some instances, by genetic studies after death.[1,2]

4. Screening

Unexpected death of a competing athlete is devastating, yet prevention of SCD remains elusive. Although the risk of exercise depends on the population studied, it is accepted that athletes with known high risk cardiac abnormalities should abstain from competitive athletics. Screening provides medical clearance for competitive sport participation through methodical evaluations that elucidate relevant and pre-existing clinical or subclinical cardiovascular abnormalities. Currently there are no universal standards pertaining to the screening of high-school or collegiate athletes; however, the American Heart Association has recommended that some form of preparticipation cardiovascular screening should be mandatory for all high-school and collegiate athletes and that a standardised preparticipation medical evaluation should be created.[7]

The huge number of competing athletes in the US constitutes a major obstacle to screening strategies. Identification of high risk athletes and subsequent reduction of SCD has been the goal of many investigators. Several studies have attempted to screen large populations of young athletes, using echocardiography, ECG and medical history. These studies have largely demonstrated normal cardiovascular findings in the great majority of individuals, and at a significant cost.[1,3,8,15]

© Adis International Limited. All rights reserved.

Well-trained athletes often demonstrate abnormalities during cardiac screening that are typical of the athletic heart syndrome.

Problems in routine screening of athletes include the limitations inherent in the infrequency of SCD, poor predictive value of available diagnostic procedures and cost of elaborate testing for large populations. Although a quality history and cardiac examination can often yield pertinent findings, it has been questioned whether diagnostic screening and periodical testing of competitive athletes can identify those at risk.[1,3,15]

Unfortunately, many screening procedures and episodic evaluation protocols yield vague and uninterpretable findings. Elaborate screening tests are not cost effective and do not consistently identify athletes at risk. Researchers have demonstrated that more than 200 000 athletes would have to be screened to find 1 athlete at risk for SCD.[3] Moreover, positive results may arise because of variants of normal and do not always predict cardiac complications. Negative results, conversely, do not necessarily eliminate the possibility of exercise-related death due to coronary disease.

Although large scale screening of young, asymptomatic athletes has been determined not to be cost effective, screening of older athletes may be worthwhile. Guidelines for suggested observation, limitations and treatment practices for athletes with structural, haemodynamic and arrhythmogenic risk factors are now available for the healthcare provider.[33,34]

Entry level recommendations to identify athletes at risk for SCD usually consist of an interview designed to explore health and family history and a thorough physical examination – although there is no uniform agreement or format. A questionnaire may be used to elucidate specific areas of concern. Admitted symptoms suggestive of heart problems such as chest pain, syncope, palpitations, dyspnoea or any exercise-related symptomatology should be investigated. Recent viral infection, use of drugs and previous activity limitations should be ascertained. Screening of athletes should be performed by an appropriately trained individual who can reliably obtain a detailed cardiovascular history, perform a physical examination and identify cardiac disease. The medical history should be precise and address key symptoms of syncope or chest pain, history of hypertension or murmur, family history of premature death, cardiovascular disability, or known cardiovascular conditions.[7] The physical examination should include resting pulse rate, blood pressure in both arms while sitting and assessment of right and left femoral pulses to exclude coarctation of the aorta. Physical inspection should include a search for characteristic findings of Marfan's syndrome (e.g. long extremities, tall stature, skeletal and chest wall deformities and ocular abnormalities), especially in those who participate in sports favouring height.

Cardiac auscultation should be performed with the individual sitting or standing to maximise flow murmurs of LV outflow obstruction (HCM); Valsalva manoeuvre may be performed during auscultation to further augment such murmurs. Auscultation should also include careful assessment of the first and second heart sound and extra heart sounds and murmurs of systole and diastole. Assessment of the femoral artery pulses should exclude coarctation of the aorta.

When findings of the routine physical or historical evaluation suggest syndromes associated with high risk for SCD (e.g. HCM, coronary artery disease, Marfan's syndrome and aortic stenosis), further diagnostic investigation is necessary. This includes a 12-lead ECG and, depending upon the level of concern, an echocardiogram. Routine ECG screening in competing athletes has been proposed. Although this would undoubtedly produce some suspected or unexpected positive results indicative of cardiovascular abnormalities, the argument against routine ECG screening has centred around the very low yield and economic inefficiency.

When physiological and pathological conditions remain difficult to distinguish, a period of inactivity is suggested before repeating the athlete's evaluation.[1,3,6,16] After 6 to 8 weeks, physiological hypertrophy will resolve and the associated physical examination, ECG and echocardiogram

© Adis International Limited. All rights reserved.

findings should return to those of a normal sedentary state, but findings indicative of pathological conditions will remain unchanged. While investigative work continues towards improving risk stratification and assessment of individuals with HCM, participation in high risk sports activity should remain forbidden.[1,3,6,15]

5. Recommendations for Participation

Once a definitive cardiovascular diagnosis is made, the 26th Bethesda Conference[33] guidelines should be used to determine recommendations for entry into, or continued, athletic competition in the presence of cardiovascular disease. Although not all circumstances are clearly defined, and more research is needed for the future, some conclusions have been generally accepted.

When diagnosis of HCM is unequivocal, athletes should be restricted from most competitive sports, regardless of LV outflow pattern. Some limited low intensity activities may be permissible.[33]

Whether participation in competitive events by athletes with congenital heart disease is appropriate depends on the severity of the anomaly. Milder forms permit participation in a variety of sports, whereas strenuous exercise in severe forms can be detrimental. Periodic re-evaluations are necessary to determine alterations in haemodynamics that result from growth or changes in defect acuity.[33]

Coronary artery anomalies, when detected, should dictate the athlete's exclusion from all competitive sports. Following surgical correction, participation in sports may be permitted after 6 months' recovery time in athletes with no demonstrable exercise-induced ischaemia.[33]

Athletes with Marfan's syndrome are often characterised by several physical and clinical features. In general, athletes with Marfan's syndrome should be restricted from those sports that risk bodily collision or those associated with intense surges of autonomic activity that lead to abrupt increases in blood pressure. Overall, restriction is determined by a family history of sudden death and after assessment of aortic root and mitral valve anatomy.[33]

Involvement in sport of athletes with acquired valvular lesions depends upon the severity of the lesion and its haemodynamic consequences. In general, when valvular lesions coexist with other cardiovascular abnormalities, such as coronary artery disease or arrhythmias, participation in sports should be restricted. Athletes with echocardiographic mitral valve prolapse may engage in all sports, providing that there is no history of arrhythmogenic syncope, family history of SCD, or repetitive, sustained, or complex ventricular or supraventricular arrhythmias. When these complicating factors are present, low intensity sports only may be allowed.[33]

Athletes found to have myocarditis should be withdrawn from competitive sports for a substantial convalescent period (generally 6 months or longer if return of normal cardiac function is delayed). A thorough cardiac assessment and testing should be performed prior to resumption of training. Return to competition should be considered only when ventricular function and cardiac dimensions have normalised and relevant arrhythmias are absent on ambulatory monitoring. Endomyocardial verification of histological recovery is not justified.[33]

Although it is an infrequent cause of SCD in the US, ARVD is reported to be an important cause of SCD in young people of northeastern Italy. With the knowledge at hand, individuals with known ARVD are advised not to participate in competitive athletics.[33]

Recommendations for athletes with coronary artery disease are determined by their assigned risk group, as defined by their clinical and metabolic functions. All athletes with coronary artery disease should be made aware of prodromal symptoms and be instructed to halt the involved activity and seek medical attention if symptoms arise. Low risk groups can participate in low dynamic and low static competitive sports. Athletes in higher risk groups may be further evaluated for participation in low intensity sports, with re-evaluations every 6 months (including annual exercise stress testing). After myocardial infarction or revascularisation

© Adis International Limited. All rights reserved.

procedures, athletes should observe a period of recuperation until recovery is deemed complete.[33]

Athletes with myocardial bridging of an epicardial coronary artery with no associated evidence of myocardial ischaemia can participate in all competitive sports. In this population, when there is suspicion or objective evidence of myocardial ischaemia, the athlete should be restricted to low intensity sports.[33]

In general, all individuals with significant cardiac arrhythmias being considered for athletic competition should be screened with 12-lead ECG, echocardiography, exercise testing and extended 24-hour ambulatory monitoring. Those with long QT interval syndrome should not compete. For those with potentially life-threatening arrhythmias of any cause who do compete, standby resuscitation equipment, support staff familiar with cardiac symptoms and knowledge of basic cardiopulmonary resuscitation and an emergency plan are required.[33,35]

6. Conclusions

HCM and coronary and myocardial structural abnormalities are common pathologies that predispose young athletes to SCD. SCD from fatal arrhythmia seems to be the most common mechanism of death. In this population, however, data are insufficient to support either invasive or noninvasive approaches to clarify risk stratification for SCD. Because of the large athletic population, variants of normal cardiac physiology found within the athletic population and the rarity of SCD, screening for individuals at risk is neither practical nor cost effective. Efforts are underway to stratify athletes at risk for SCD to determine who can participate in competitive sports and who should not. Routine screening and cardiac auscultation of young athletes, evaluation of exercise-induced symptoms and education of older athletes to symptoms of cardiac ischaemia, are all essential to primary prevention of SCD in the athletic population.

Until research can accurately define variables of haemodynamic and electrical instability that permit reliable identification of athletes with HCM who are at risk for SCD, the recommendation is to disqualify athletes with confirmed HCM from moderate-to-high intensity competitive sports. Owing to the decreased risk of SCD in older athletes, individual judgement of eligibility may be used. Recommendations for participation in athletic events by individuals with other high risk cardiac conditions are now available.

For the physician, problems related to 'pressure for answers' are great and differentiating normal from abnormal physiology in the highly trained athlete is difficult. Too often, the athlete's main concern is to continue playing in the pursuit of psychological or financial rewards. Each athlete has the right to obtain proper evaluation and be able to explore therapeutic recommendations; he or she also carries the responsibility of deciding on and accepting which course to take after being fully informed of risks.[22] Although no clear legal precedent exists requiring preparticipatory screening of athletes, there is an ethical obligation of educational institutions to provide cost-effective strategies to ensure that their athletes are not placed at medical risk.[7]

Current guidelines for preparticipation screening will form the basis of proper medical care and can establish a legal standard of care once accepted and followed by healthcare professionals and judicial systems. The physician, however, is ultimately responsible for educating the athlete on matters of health and best interests and for initiating prudent efforts that identify life-threatening diseases and minimise medical risks of competitive participation – regardless of the demands made by the coaches, institutions or the athlete.

Acknowledgements

Dr Robert Myerburg is supported in part by the American Heart Association Chair in Cardiovascular Research at the University of Miami.

References

1. Maron BJ. Triggers for sudden cardiac death in the athlete. Cardiol Clin 1996; 14 (2): 195-210
2. Thompson PD. The cardiovascular complications of vigorous physical activity. Arch Intern Med 1996; 156: 2297-302

© Adis International Limited. All rights reserved.

3. Rich BS. Sudden death screening. Med Clin North Am 1994; 78: 267-86
4. Myerburg RJ. Sudden death. J Contin Educ Cardiol 1978; 13: 15-29
5. Mitchell JH, Haskell W, Raven PB. Recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities: classification of sports. J Am Coll Cardiol 1994; 24: 864-6
6. Maron BJ, Pelliccia A, Spirito P. Cardiac disease in young trained athletes – insights into methods for distinguishing athlete's heart from structural heart disease, with particular emphasis on hypertrophic cardiomyopathy. Circulation 1995; 91 (5): 1596-601
7. Maron BJ, Thompson PD, Puffer JC, et al. Cardiovascular preparticipation screening of competitive athletes. Circulation 1996; 94 (4): 850-6
8. Wight JN, Salem D. Sudden cardiac death and the 'athlete's heart'. Arch Intern Med 1995; 155: 1473-80
9. Kloner RA, Hale S, Alder K, et al. The effects of acute and chronic cocaine use on the heart. Circulation 1992; 85 (2): 407-19
10. Burke AP, Farb A, Virmani R. Causes of sudden death in athletes. Cardiol Clin 1992; 10: 303-15
11. Leonard RB, Alexander D, Byrum JE. An athlete dying young: arrhythmogenic right ventricular dysplasia. N C Med J 1994; 55 (4): 110-2
12. Rossi L. Structural and non-structural disease underlying high-risk cardiac arrhythmias relevant to sports medicine. J Sports Med Phys Fitness 1995; 35: 79-86
13. Maron BJ, Klues HG. Surviving competitive athletics with hypertrophic cardiomyopathy. Am J Cardiol 1994; 73: 1098-103
14. Myerburg RJ, Mitrani RA, Interian Jr A, et al. Identification of risk of cardiac arrest and sudden cardiac death in athletes. In: Estes NAM, Salem DN, Wang PJ, editors. Sudden cardiac death in the athlete. Armonk (NY): Futura Publishing Company Inc., 1998
15. Maron BJ, Isner JM, McKenna WJ. Hypertrophic cardiomyopathy, myocarditis and other myopericardial diseases and mitral valve prolapse. J Am Coll Cardiol 1994; 24: 880-3
16. Estes III M. Sudden death in young athletes. N Engl J Med 1995; 333 (6): 380-1
17. Maron BJ, Poliac LC, Kaplan JA, et al. Blunt impact to the chest leading to sudden death from cardiac arrest during sports activities. N Engl J Med 1995; 333 (6): 337-41
18. Van Camp SP, Bloor CM, Mueller FO, et al. Nontraumatic sports death in high school and college athletes. Med Sci Sports Exerc 1995; 27 (5): 641-7
19. Cantu RC. Congenital cardiovascular disease: the major cause of athletic death in high school and college. Med Sci Sports Exerc 1991; 24: 279-80
21. Zipes D, Garson A. Arrhythmias. J Am Coll Cardiol 1994; 24: 892-8
20. Waller BF. Exercise-related death in athletes: the most common causes. In: Waller BF, Harvey WP, editors. Cardiovascular evaluation of athletes: toward recognising young athletes at risk of sudden death. Cedar Grove (NJ): Laennec Publishing Inc., 1993: 5
22. Consensus statement of the joint steering committees of the unexplained cardiac arrest registry of Europe and of the idiopathic ventricular fibrillation registry of the United States: survivors of out-of-hospital cardiac arrest with apparently normal heart. Circulation 1997; 95: 265-72
23. Thompson PD. Athletes, athletics, and sudden cardiac death. Med Sci Sports Exerc 1993; 25 (9): 981-4
24. Sherrid MV, Mieres J, Mogtader A, et al. Onset during exercise of spontaneous coronary artery dissection and sudden death: occurrence in a trained athlete. Chest 1995; 108: 284-7
25. Futterman LG, Lemberg L. Sudden death in athletes. Am J Crit Care 1995; 4 (3): 239-44
26. Friman G, Wesslen L, Fohlman J, et al. The epidemiology of infectious myocarditis, lymphocytic myocarditis and dilated cardiomyopathy. Eur Heart J 1995; 16 Suppl. 0: 36-41
27. Wesslen L, Pahlson C, Lindquist O, et al. An increase in sudden unexpected cardiac deaths among young Swedish orienteers during 1979-1992. Eur Heart J 1996; 17: 902-10
28. Willems S. Sudden cardiac death in young athletes: orienteering on Chlamydia pneumoniae? Eur Heart J 1996; 17: 810-2
29. Curfman CD. Fatal impact – concussion of the heart. N Engl J Med 1998; 338 (25): 1841-3
30. Janda DH, Cir CA, Viano DC, et al. Blunt chest impacts: assessing the relative risk of fatal cardiac injury from various baseballs. J Trauma 1998; 44 (2): 298-303
31. Maron BJ, Strasburger JF, Kugler JD, et al. Survival following blunt chest impact–induced cardiac arrest during sports activities in young athletes. Am J Cardiol 1997; 79 (6): 840-1
32. Michalodimitrakis EN, Tsatsakis AM. Vehicular accidents and cardiac concussion – a traumatic connection. Am J Forensic Med Pathol 1997; 18 (3): 282-4
33. Maron BJ, Mitchell JH. 26th Bethesda conference: recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities. J Am Coll Cardiol 1994; 24 (4): 845-99
34. Fahrenbach MC, Thompson PD. The preparticipation sports examination: cardiovascular considerations for screening. Cardiol Clin 1992; 10: 319-27
35. Hutter AM. Cardiovascular abnormalities in the athlete: role of the physician. J Am Coll Cardiol 1994; 24 (4): 851-3

Correspondence and reprints: *Laurie G. Futterman*, ARNP, Division of Cardiology D-39, School of Medicine, P.O. Box 016960, Miami, FL 33101, USA.
E-mail: LFutterm@mednet.med.miami.edu

© Adis International Limited. All rights reserved.