Recent Advances in Cardiology



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Chapter 3

Cardiac screening for athletes

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PREPARTICIPATION CARDIOVASCULAR SCREENING IN ATHLETES

Sudden cardiac death (SCD) in young athletes is a highly emotional event when consideration is given to the youth of the individual and the circumstantial paradox. The vast majority of athletes fall victim in the absence of prodromal warning symptoms. Over the past two decades the increasing number of reports of SCD in young athletes has heightened awareness of the issue and called for measures to prevent such catastrophes.

Epidemiology, demographics and causes [1]

The true prevalence of SCD among young athletes remains a source of considerable debate but ranges between 0.5 and 2.0/100,000. Almost 90% of all SCD in young athletes occurs during, or immediately after, strenuous physical activity, indicating that exercise is a trigger for fatal ventricular arrhythmias in those athletes harbouring potentially lethal cardiac conditions. Indeed, in susceptible athletes competitive sports activity enhances the risk of sudden death by two to five folds.

There is significant male predominance (male:female ratio 9:1) of SCD, which can only be partly explained by the higher sport participation rates in males. Approximately 40% of SCDs occur in athletes under 18 years. The majority of deaths are reported in American football, basketball and soccer reflecting the popularity of these sporting disciplines in Western countries.

The cause of SCD during sport is influenced by the age of the athlete. Inherited and acquired cardiovascular abnormalities are usually responsible for SCD in athletes <35 years old, whereas atherosclerotic coronary artery disease predominates in older athletes. In young athletes, the primary cardiomyopathies, specifically hypertrophic cardiomyopathy (HCM) and arrhythmogenic right ventricular cardiomyopathy (ARVC), account for over 40% of cases. Congenital coronary artery anomalies and premature coronary artery atherosclerosis are responsible for approximately 20% of cases of SCD (**Figure 3.1**).

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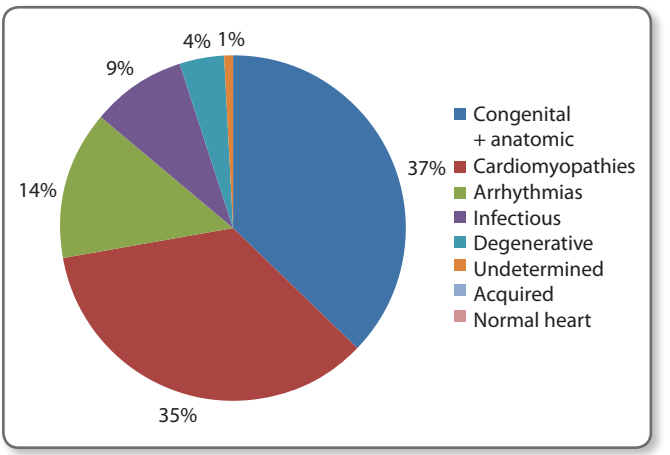

Figure 3.1 Causes of sudden cardiac death in athletes.

The heart may be structurally normal in at least 4% of all cases of SCD. In these circumstances, deaths are usually attributed to the ion channelopathies including congenital long- or short QT syndrome, Brugada syndrome and catecholaminergic polymorphic ventricular tachycardia, or congenital accessory pathways [2].

PREPARTICIPATION SCREENING

The primary aim of preparticipation screening (PPS) is to identify athletes with unsuspected cardiovascular diseases and prevent SCD through appropriate intervention. Both the American Heart Association and the European Society of Cardiology (ESC) agree that the justifications for PPS to protect athletes are compelling on ethical, medical and legal grounds [3,4]. Most countries do not have a nationally sponsored systematic PPS; however, many sporting organisations endorse such practice. In the UK, the national football and rugby league, Lawn Tennis Association and the English Institute of Sport advocate PPS in all competitors.

Although most conditions implicated in SCD can be identified during life, no single cardiac investigation will lead to the diagnosis of all conditions, given the diversity of disorders implicated. The use of multiple investigations in all athletes would be cost prohibitive considering the low incidence of SCD, therefore the most cost-effective strategies are sought to identify athletes at risk. There are currently two models of PPS in practice, one in the United States and one in Italy. The American model relies on a health questionnaire and a physical examination to identify the presence of cardiac symptoms, family history of premature cardiac disease and clinical evidence of hypertension, a cardiac murmur, coarctation of the aorta and Marfan syndrome (**Table 3.1**) [5]. This strategy is practical, relatively cheap and easy to conduct but has limited value, as 80% of athletes who die suddenly do not express prodromal symptoms, and most conditions implicated in exercise-related SCD are associated with normal physical examination. The lack of sensitivity of the American model was highlighted in a series of 115 reported cases of SCDs in young athletes who had been screened. A correct diagnosis was identified in only one athlete (0.9%) [6]. In a British cohort of over 2700 young athletes and physically active school children undergoing PPS using 12-lead electrocardiography (ECG), nine individuals were diagnosed with a

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Table 3.1 American Heart Association guidelines for preparticipation cardiovascular screening of young competitive athletes [5]

Medical history
Chest pain or discomfort on exertion
Unexplained syncope or near syncope
Excessive exertional dyspnoea or fatigue
Prior recognition of a heart murmur
Raised systemic blood pressure
Family history
Premature death (<50 years) from heart disease, in one or more relatives
Disability from heart disease in a close relative aged <50
Specific knowledge of certain cardiac conditions in family members: hypertrophic and dilated cardiomyopathy, long QT syndrome or other ion channelopathies, Marfan syndrome, or clinically important arrhythmias
Physical examination
Heart murmur
Femoral pulses to exclude aortic coarctation
Physical stigmata of Marfan syndrome
Brachial artery blood pressure (sitting position, taken in both arms)
Parental verification is recommended for high school and middle school athletes
Judged not to be vasovagal; of particular concern when related to exertion
Auscultation should be done both while supine and while standing or with Valsalva manoeuvre, specifically to identify murmurs of dynamic left ventricular

cardiovascular disorder implicated in SCD; however, none of them were symptomatic or had a family history [7]. Both studies demonstrate that a health questionnaire and physical examination alone are inadequate for identifying high-risk athletes.

Italian model

In Italy, it is mandatory for all young competitive athletes to undergo cardiovascular screening on an annual basis. The Italian model incorporates 12-lead ECG in conjunction with a health questionnaire and physical examination (**Figure 3.2**). Athletes with anomalies are investigated further to confirm or refute a cardiac disorder and those with an overt cardiac diagnosis are managed according to established guidelines [8].

Effectiveness of the ECG in diagnosing cardiac disease

Unsurprisingly, the addition of a 12-lead ECG is associated with a higher sensitivity than history and physical examination alone. A recent American study of 510 collegiate athletes identified 11 athletes with cardiovascular abnormalities [9]. History and examination alone identified only 5 of the 11 cases; however, the inclusion of the ECG detected all cases. Based on this study, the sensitivity of history and examination alone was only 45%. In contrast, the sensitivity, specificity and negative predictive accuracy when history and examination were combined with ECG were 91%, 83% and 99.8% respectively.

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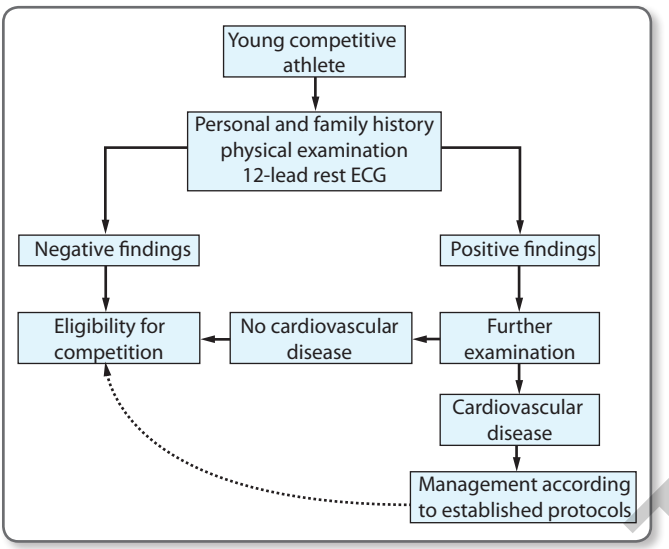

Figure 3.2 The Italian model for preparticipation screening.

The ECG is invaluable in the identification of diseases affecting cardiac conduction. Furthermore, it reveals marked repolarisation changes commonly associated in the primary cardiomyopathies [2]. The ECG is abnormal in up to 90% cases of HCM, over 80% cases of ARVC prompting further assessment with appropriate cardiac imaging tests in such individuals [10,11]. In a large population-based study of screening outcomes in 33,735 young athletes in the Veneto region of Italy, 621 (1.8%) athletes were disqualified because of the identification of cardiovascular disorders. Of these, 22 (0.07%) athletes were diagnosed with HCM, which accounted for 3.5% of all cardiovascular-related reasons for disqualification [12]. The diagnosis of HCM was made following an abnormal ECG in over three quarters (77%) of cases. The prevalence of HCM in this cohort of athletes was not dissimilar to that derived from population-based echocardiographic studies indicating that the ECG is effective in the identification of athletes with HCM.

The specificity of the 12-lead ECG-based Italian PPS program in excluding HCM has also been assessed in a study of 4450 elite athletes initially judged eligible for competition following PPS with ECG. All athletes underwent echocardiographic examination to assess previously undetected HCM. After an average 8-year follow-up period, HCM was diagnosed in only one (0.02%) athlete indicating that a normal ECG has a high negative predictive value (99.98%), excluding HCM in almost all athletes [13].

Does screening with 12-lead ECG prevent SCD in young athletes?

Although the ECG is effective in the identification of cardiac diseases in young athletes, there are few studies to assess its role in preventing SCD. The only scientifically credible data relating to the efficacy of ECG in preventing SCD are derived from prospective studies in the Veneto region of Italy, which maintains a pathologic systematic registry of SCDs in sport in addition to the country's mandatory PPS practice. These data provide compelling evidence that PPS including the ECG is associated with a reduction in SCD. A large prospective study by Corrado et al. [14] comparing the incidence of SCD during

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the prescreening era and following a 25-year period of screening showed a dramatic reduction in the incidence rate of SCD from 3.6/100,000 person-years to 0.43/100,000. This represented a 90% reduction in mortality since the implementation of PPS and were predominantly due to fewer cases of SCD from cardiomyopathies. These observations also provide the most plausible explanation for the lower incidence of HCM-related deaths in Italy compared with other countries [15].

The notion that screening with ECG reduces the risk of SCD has been challenged by some. Maron et al. [16] compared deaths rates among young athletes screened with ECG from the Veneto region of Italy and American athletes from a demographically similar population (Minnesota) who were screened using the American model between 1985 and 2005. The authors reported no significant differences in death rates in athletes between the two populations during the evaluation period, although trend analysis after 1998 clearly reveals a declining death rate in Veneto and a rising death rate in Minnesota. In contrast with the Italian data, the numerator values used to calculate the incidence of SCD in the American athletes were collated from insurance claims and media reports, which are relatively inadequate since a subsequent study of US collegiate athletes has eloquently demonstrated that reliance on such methods would only capture 20% and 56% of all SCDs respectively [17]

A more recent study by Steinvil et al. [18] from Israel investigated the incidence of SCD in young athletes 12 years after the introduction of a mandatory screening program which incorporated the ECG and compared the figures with the incidence of SCD 12 years prior to the screening program. The authors failed to demonstrate a decline in the incidence of SCD among during the two periods (2.54/100,000 vs. 2.66/100,000 person-years). The study lacked scientific rigour because identification of deaths relied solely on retrospective media searches in just two newspapers and there was no accurate data on the precise population at risk, hence the denominator value for incidence calculation was also unreliable. The authors did not provide any information about diagnostic yield following screening, athlete disposal or autopsy information causing many to be dismissive of conclusions.

Controversies relating to ECG screening

Despite the aforementioned account, the concept of early identification of potential victims remains a highly controversial and contended issue [19]. Antagonists vehemently question the cost-effectiveness of PPS and the need for multiple investigations to identify the broad and diverse spectrum of disease processes implicated in SCD. There are also concerns relating to the medical and legal implications of false positive and negative results as well as the availability of resources and practical logistics for implementing such programs.

Low incidence of events

Fortunately, the incidence of SCD in sport is low; therefore, several thousand athletes have to be assessed to identify one that may die suddenly. This raises questions regarding the cost-effectiveness of PPS, particularly since there are financial constraints on the health service. Nevertheless, such events are highly visible, particularly when high-profile athletes are involved, and the public cardiac arrest of the professional football player, Fabrice Muamba in the UK last year, is just one example of the societal impact of such tragedies.

In contrast to the wide range of estimates for the incidence of SCD, the prevalence of potentially lethal cardiovascular diseases in athletes based on ECG studies has consistently ranged between 0.2% and 3% [1], indicating that at least 1 in 500 young

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sporting individuals harbour a cardiac disorder with the potential to kill without warning. Therefore, perhaps it is the prevalence of cardiovascular conditions with the potential for SCD, rather than the incidence of SCD itself, that should influence the debate relating to the cost-effectiveness of PPS.

It could be argued that the protection of the lives of young athletes should feature at the forefront of health-care strategies to prevent cardiovascular deaths as with many deserving issues including obesity, diabetes mellitus, hypertension, coronary artery disease and heart failure. Paradoxically, the prospect of identifying a sinister genetic or congenital cardiac disorder in a young athlete is associated with disproportionately better outcomes at a much lower cost compared with the aforementioned conditions. Additionally, familial evaluation following the diagnosis of a genetic disorder provides an invaluable opportunity to identify other young relatives at risk of SCD. The early identification of a young individual with cardiac disorders also permits informed decisions relating to exercise prescription and the choice to pursue alternative career paths.

False-positive results

Athletic training is associated with electrical and structural remodelling which is considered part of the normal physiological process to sustain large increases in cardiac output for prolonged periods. Occasionally, the electrical manifestations of the athlete's heart overlap with those observed in patients with cardiomyopathy or cardiac conduction tissue disease. Concerns regarding the number of false-positive tests that necessitate further investigations, undue anxiety and the potential for erroneous disqualification continue to fuel debates against the inclusion of the ECG in the PPS process [19].Results from a prospective observational study in collegiate athletes demonstrated that the addition of the 12-lead ECG to PPS increased the sensitivity of PPS from 46% to 91%; however, there was also a corresponding 17% increase in false-positive rates [9].

The issue of false-positive ECG results is commonly exaggerated but does emphasise the need for athletes to be investigated by cardiologists experienced in the emerging discipline of sports cardiology. Most athletes deemed to exhibit ECG abnormalities against standards derived from a sedentary population demonstrate electrical manifestations of increased vagal tone and of chamber enlargement (particularly in relation to satisfying the criteria for left ventricular hypertrophy), which are normal responses to chronic physical training [20]. In a study of an unselected group of 32,652 recreational athletes, 11.8% individuals demonstrated abnormalities but only 4.8% revealed anomalies that could be considered to overlap with incomplete or morphologically mild expressions of genetic or structural cardiac disorders. Even in this small group, disqualification would only be entertained following a definitive diagnosis rather than in relation to isolated ECG findings. In response, the ESC updated guidelines to facilitate accurate interpretation of the 'athlete's ECG in 2010 [21], while minimising the risk of false-positive tests (**Table 3.2**). ECG patterns have been categorised in to group 1 (related to athletic training) and group 2 (unrelated to physical training) changes (**Table 3.2**). It is the authors' opinion that further modification of these guidelines should reduce false-positive rates to <3% [22A]. For example, there is little evidence that electrical anomalies such as axis deviation or voltage criterion for atrial enlargement predict cardiac disease in asymptomatic athletes when found in isolation or in association with other training-related changes [22B]. Although T-wave inversion (other than in leads V_1, III and aVR) definitely warrants further investigation in adult Caucasian athletes, such repolarisation changes are observed in up to 25% of athletes of African or

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Table 3.2 European Society of Cardiology (ESC) classification of ECG abnormalities in athletes [21]

ESC classification of ECG abnormalities in athletes	
Group 1 (training-related)	Group 2 (non-training-related)
Sinus bradycardia	T-wave inversions
First-degree atrioventricular block	ST-segment depression
Incomplete right bundle branch block	Pathological Q-waves
Early repolarisation	Left atrial enlargement
Isolated QRS voltage criteria for left ventricular hypertrophy	Right atrial enlargement
	Left axis deviation
	Right axis deviation
	Right ventricular hypertrophy
	Ventricular pre-excitation
	Complete left or right bundle branch block
	Long-QT or short-QT interval
	Brugada-like early repolarisation

Afro-Caribbean origin (black athletes) [23] and may overlap with both HCM and ARVC. However, recent data concerning black athletes reveals that T-wave inversion in leads V_1–V_4 that are preceded by J point elevation and convex ST segments is a normal ethnically mediated variant of the black athlete's heart [23].

On occasion, an asymptomatic male athlete may present with a combination of marked repolarisation changes and a maximal left ventricular wall thickness of 13–15 mm which raises the possibility of morphologically mild HCM. The differentiation from physiological left ventricular hypertrophy, a supposedly benign condition, is crucial but can be challenging. Generally, a systematic approach taking into account the family history, cardiopulmonary exercise test and cardiac MRI usually clarifies matters (**Figure 3.3**). Although a 4–6 weeks period of detraining demonstrating the regression of left ventricular hypertrophy is an ideal method of differentiating between the two entities, most athletes are not keen to comply as it hinders fitness and may affect team selection. Assessment of first-degree relatives is also ideal in these situations but not always practical since many national athletes compete away from their regions. The role of gene testing in such situations is controversial and beyond the scope of this chapter.

False negatives

No single diagnostic test is expected to identify all disorders implicated in SCD; however, the ECG performs exceptionally well in this regard and in a cost prohibitive financial climate is probably the most effective tool for identifying most conditions predisposing to SCD in young athletes. The ECG will identify most abnormalities directly affecting the conduction tissue or cardiac muscle, although around 5–10% of patients with HCM and approximately 20% of those with ARVC exhibit a normal resting ECG. Up to 30% of patients harbouring genetic mutations for congenital long QT syndrome have a normal QT interval; however, the vast majority of asymptomatic gene carriers with a QTc <500 ms have an excellent prognosis with event rates <0.2% per annum. Catecholaminergic polymorphic

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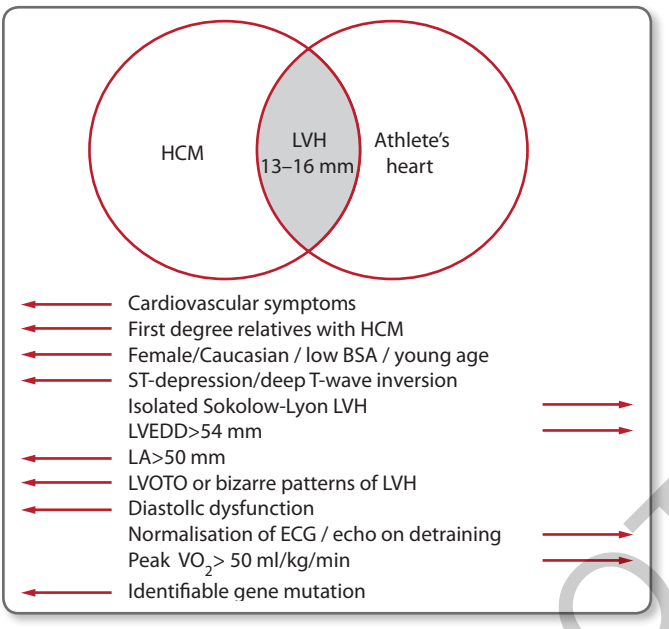

Figure 3.3 Criterion for differentiating athlete's heart from hypertrophic cardiomyopathy. Adapted from Maron et al. [24].

ventricular tachycardia will only be identified on an exercise ECG or following an epinephrine test, which should be reserved for athletes with exercise-induced palpitations or syncope. The resting ECG is ineffective in the detection of diseases affecting the coronary arteries, aorta and the valves. The Italian screening experience revealed that death rates from anomalous coronary origins remained unchanged in screened athletes and were higher than those in unscreened nonathletes (12% vs. 0.5%) [14].

Cost-effectiveness

In countries where an established PPS program is not in place, the estimated cost for a de novo program to cater for all athletes would be considerable. The American Heart Association quotes a sum of $2 billion per annum to provide ECG screening in all high school and middle school athletes in the United States; however, recent studies have revealed that PPS of young athletes using the 12-lead ECG may be cost-effective [9,25].

A US-designed computer simulation extrapolating Italian data on incidence rates of SCD was performed to compare the cost-effectiveness of PPS using history and physical examination alone versus the addition of the 12-lead ECG in athletes aged 14–22 years [25]. Estimates demonstrated that the addition of a 12-lead ECG would save 2.06 life years per 1000 athletes, yielding a cost-effectiveness ratio of US$ 42 900 per life-year saved compared with history and physical examination alone. Two other cost analysis studies in high school [26] and collegiate athletes [9] have shown similar costs of ECG screening per life saved and fall within the range that the United States and most Western European countries would be willing to pay for health-care intervention. The proponents for PPS would argue that the feasibility of implementing an ECG-based PPS for the protection of the healthiest segment of our society is commonly portrayed as an insurmountable task based on concerns relating to the cost, logistics and lack of trained personnel. The American Heart Association

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figure of $2 billion per annum to provide ECG screening in high school and middle school athletes in the United States appears impractical but sounds entirely acceptable when one considers that over $35 billion is spent each year on heart failure, a disorder associated with a 5-year survival rate of <5 years.

Most Western countries have the potential for creating an infrastructure within high schools similar to several established immunisation programs. There is evidence that teachers, coaches and even volunteering parents could be trained in recording ECGs [27]. More recently a group of experts from the United States and Europe convened to provide three impressively illustrated documents to facilitate the training of sports physicians in the interpretation of the athlete's ECG [28]. The incorporation of training in ECG interpretation in adolescent athletes and young exercising adults within cardiology and sports medicine curricula as well as the development of commercial training courses for primary care physicians should promote the number of qualified personnel required for accurate ECG reporting. Indeed the incentive should be prudent since 25–30% of the population in the Western world is aged <18 years, most exercise and at least 0.2% harbour disorders associated with premature SCD [1].

Psychological impact

Concerns have been raised about the potential impact of a false-positive test and the anxiety associated with the prospect of being diagnosed with a sinister cardiac disorder and disqualification from participating in most competitive sports. Few studies have examined the psychological impact of PPS. In a study of 441 Norwegian football players assessed for psychological stress using a Likert scale, 64% felt more confident playing and 88% were satisfied having completed the screening. The majority (77%) felt the need for screening and 84% reported they would strongly recommend it to others, 16% were afraid that the screening may have consequences for their health and 13% feared they would lose their licence to compete; however, only 3% scored high enough to be considered as experiencing stress [29].

The authors recommend that athletes judged to have abnormalities during initial evaluation be investigated promptly in an expert setting to minimise anxiety. There should be options for expert psychological support should an athlete be diagnosed with a career ending cardiac condition.

ALTERNATIVE STRATEGIES

Automated external defibrillators (AED)

Some athletes may die suddenly despite ECG screening, as a result of abnormalities of the coronary arteries or incomplete expression of cardiomyopathies and ion channelopathies. Furthermore, deaths may be due to acquired cardiac disease including commotio cordis, myocarditis, electrolyte disturbances, heat stroke and the use of performance-enhancing drugs. Therefore, a community-based portable AED should be available during athletic competition. The availability of such devices has improved survival from out of hospital cardiac arrest from 6% to over 40%. Previous data had suggested that survival from cardiac arrest during exercise was disappointingly low despite the availability of an AED. In a significant proportion of cases, the first shock was delivered by the emergency services, suggesting that lack of appropriate training in cardiopulmonary resuscitation or delays

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in defibrillation may have contributed. Subsequently, a retrospective analysis of 1710 US high schools with onsite AEDs and appropriate training of coaches reported 36 cases of sudden cardiac arrest including 14 cases in student athletes. Of the 36 cases of sudden cardiac arrest, 35 (97%) were witnessed, 34 (94%) received bystander cardiopulmonary resuscitation and 30 (83%) received defibrillation within 3 minutes [30]. A total of 23 (64%) individuals survived to hospital discharge, including 9 of the 14 (64%) student athletes indicating that prompt cardiopulmonary resuscitation and early defibrillation were associated with a significant improvement in survival rates among exercising young individuals.

SCREENING FOR LEISURE AND VETERANS ATHLETES

Atherosclerotic coronary artery disease is a leading cause of exercise-related SCD in older athletes. In this age group, the resting ECG has a poor yield for detecting silent coronary artery disease. Conventional exercise testing will only identify athletes with the most severe disease and has a role in investigating relatively sedentary individuals who are keen to embark on intensive exercise. Conventional exercise testing has a low sensitivity and specificity in asymptomatic people, and a negative exercise stress test does not preclude coronary artery disease. It is the authors' view that CT coronary calcium scoring is the investigation of choice to identify asymptomatic coronary artery disease in middle-aged individuals, keen to participate in ultra-endurance events, including the marathon. We appreciate that such practice is currently impractical due to limitations in the ability to perform the investigation in a large numbers of individuals.

Key points for clinical practice

- Sudden cardiac death in athletes is rare and usually results from inherited or congenital abnormalities of the heart
- Screening with health questionnaire, physical examination and 12-lead ECG is effective at identifying the majority of disorders implicated in sudden cardiac death in young athletes.
- Major sports and cardiac organizations advocate pre-participation screening
- Recent studies suggest false positive rates of ECG screening in athletes are lowered in expert settings.
- ECG and echocardiography changes seen in athletes heart may occasionally overlap with morphologically mild hypertrophic cardiomyopathy
- The differentiation between hypertrophic cardiomyopathy and physiological left ventricular hypertrophy in athletes can prove challenging in some but is possible in most cases

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Recent Advances in Cardiology 16

Recent Advances in Cardiology 16 is the ideal resource for keeping abreast of new developments within the field of cardiovascular medicine. Written by experts and featuring topics such as cardiovascular screening for athletes, electrocardiography of the QT interval and genetics of atrial fibrillation, each chapter highlights the latest developments of relevance to clinical practice. This book is also an invaluable revision tool for trainees preparing for postgraduate examinations.

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