Pre-participation cardiovascular screening in athletes: when and how?[1]

Saqib Ghani, Sanjay Sharma

St George's University of London, United Kingdom

Summary

Sudden cardiac death (SCD) in athletes is a rare but highly tragic event. The majority of SCDs in young athletes are attributable to inherited or congenital cardiac disorders. Athletes harbouring such potentially fatal conditions may remain asymptomatic and sudden death is frequently the first presentation. Most implicated disorders are identifiable during life, and a variety of therapeutic options are available to minimise the risk of SCD. Whereas most health professionals advocate pre-participation cardiovascular screening (PPS) on humanitarian and medical grounds, there is controversy about the most cost-effective method of screening. The American screening model encompasses a cardiovascular health questionnaire and physical examination, while in Italy a mandatory national screening programme is in existence that also incorporates 12-lead ECG. The American model is cheap and pragmatic but has poor sensitivity for the identification of sinister cardiac disorders. In contrast, the Italian model has been shown to exhibit a high sensitivity and specificity for the detection of cardiomyopathies, and has been associated with a dramatic reduction in the incidence of SCD since its inception. However, there are concerns regarding the cost-effectiveness of large-scale ECG screening programmes driven by false positive results and costs of unnecessary additive investigations to confirm or refute the presence of a serious disorder. Nevertheless, major sporting and cardiac organisations have endorsed and advocated the ECG-based screening protocol, which currently remains the most feasible and cost-effective method of pre-participation screening. More efforts are required to address some pertinent issues regarding the universal implementation of PPS programmes.

Key words: pre-participation cardiovascular screening; sudden cardiac death; electrocardiogram

Introduction

It is well established that participation in regular physical exercise is associated with reduced mortality from cardiovascular causes [1]. However, a small proportion of young (≤35 years) and apparently healthy athletes are at increased risk of sudden cardiac death (SCD) due to an unsuspected cardiac disorder [2]. Most cardiovascular causes of sudden death in young athletes are identifiable during life and several therapeutic strategies are available to minimise the risk of SCD, ranging from abstinence from exercise and pharmacotherapy to the use of implantable cardiovertor defibrillators. Although most health professionals are staunch advocates of protecting young athletes on humanitarian grounds, the feasibility of implementing widespread pre-participation cardiovascular screening (PPS) is met with resistance and remains a heavily debated topic. The low incidence of SCD and need for further investigations to identify all implicated disorders constantly challenge the efficacy and cost-effectiveness of PPS and are a persistent "Achilles heel" for proponents of such screening programmes [3]. Leading sports and cardiac organisations around the world endorse pre-participation screening in athletes to identify individuals at risk of SCD, but there is controversy over the most cost-effective method. The use of the 12-lead ECG as a screening tool is frequently contested due to concerns regarding false positive results and the need for unnecessary and costly investigations prior to clearance, as well as the remote possibility of erroneous disqualification from competitive sports. This article presents an overview of the available data on pre-participation cardiovascular screening in athletes to reduce sudden cardiac death.

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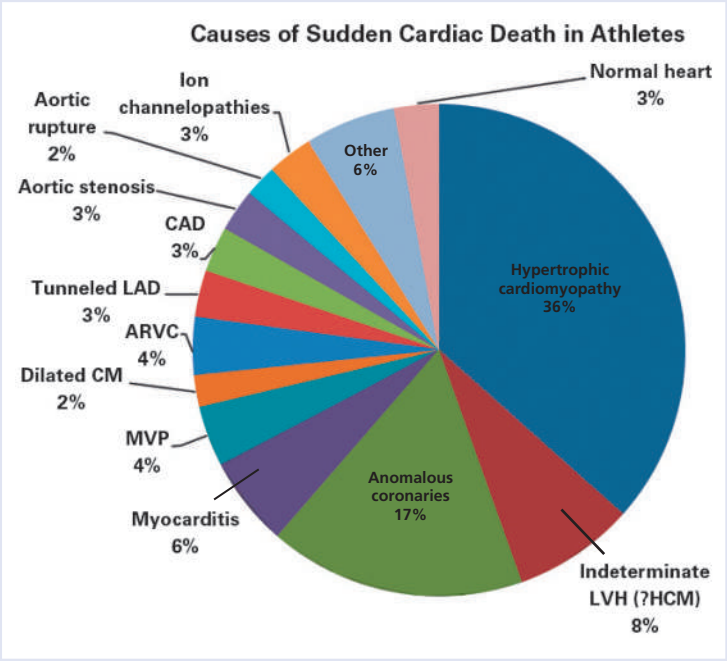
Correspondence:
Professor Sanjay Sharma, MD
Dept of Cardiovascular Sciences
St George's University of London
Cranmer Terrace
London SW17 0RE / United Kingdom
ssharma21[at]hotmail.com

Incidence of sudden cardiac death in athletes

Sudden cardiac death in athletes is a rare event albeit a highly visible one; the incidence varies between different series reported in the literature. In a prospective Italian study the incidence of SCD in athletes aged 12 to 35 was 2.3/100 000 [2]. In contrast, in US high-school and collegiate athletes the incidence of SCD was reported to be <1/100 000 [4]. An even lower incidence rate of 0.6/100 000 has been estimated from a large registry assembled over 27 years in the US [5]. The discrepancy between the Italian and US results is best explained by the fact that whereas the Italian data were prospectively gathered from a well-defined geographical area in the Veneto region, the US estimates relied on a retrospective analysis of registry and media reports. Furthermore, the Italian population comprised of relatively older athletes (12–35 years) compared to the US study (12–24 years), and constituted of a higher proportion of males than the US series (85 vs 65%). It is likely that the older age and associated ability to train harder as well as male sex contributed to a higher incidence rate of SCD in Italy.

Most SCD in athletes occur during or soon after exercise [2]. Furthermore, athletes harbouring an underlying cardiac disease are 2.8 times more likely to become victims of SCD compared with non-athletes [2], implying that exercise serves as a trigger for ventricular arrhythmia in predisposed individuals. The risk of SCD generally increases with age and males are affected 10-times more commonly than females [2, 6]. Sudden cardiac death due to cardiomyopathies and structural heart disease more commonly affects black athletes compared to white counterparts; conversely the proportion of SCD attributable to ion channelopathies is reportedly higher in white athletes [5].

Figure 1
Causes of sudden cardiac death in athletes. (Reproduced from [8]: Maron BJ, Thompson PD, Ackerman MJ, Balady G, Berger S, Cohen D, et al. Recommendation and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update. Circulation. 2007;115:1643–55. © Wolters Kluwer Health, Baltimore, USA. Reprint with kind permission.)



Causes of sudden cardiac death in athletes

The commonest cause of SCD in older athletes (>35 years) is unsuspected coronary artery disease. In contrast, over 80% of cases of SCD in younger athletes (≤35 years) are attributed to inherited or congenital cardiac disorders. Hypertrophic cardiomyopathy (HCM) is the most common cardiac disorder associated with SCD in athletes in the US [5]. In contrast, arrythmogenic right ventricular cardiomyopathy (ARVC) is the most common cardiac disorder associated with SCD in Italian athletes and accounts for approximately 25% of all cases [7]. The discrepancy in the prevalence of SCD from ARVC between the US and Italy could be explained by several factors, particularly the existence of a legal and mandatory PPS programme in Italy that has proven effective in early diagnosis of HCM with subsequent disqualification from sport. Furthermore, most data on SCD in Italian athletes is derived from the Veneto region which is internationally renowned for pathological expertise in diagnosis of ARVC. Coronary artery anomalies are the second most common cause of SCD in athletes (fig. 1). Other structural cardiac conditions include aortic root rupture (may be associated with Marfan's syndrome) and valvular heart disease (e.g., aortic stenosis, mitral valve prolapse).

The heart may be structurally normal in at least 4% of all cases of SCD [5, 7]. When a detailed pathological examination at post-mortem does not identify a structural cardiac cause, such cases are referred to as sudden arrythmogenic death syndrome (SADS). Inherited ion channelopathies or congenital accessory pathways are commonly implicated cardiac disorders in these circumstances, including long QT syndrome (LQTS), Brugada syndrome, Wolff-Parkinson-White syndrome (WPW), catecholaminergic polymorphic ventricular tachycardia (CPVT), short QT syndrome and Lenegre's disease.

Other acquired causes of SCD in athletes include myocarditis, drugs, heat stroke, electrolyte imbalance and blunt trauma to the chest wall leading to ventricular fibrillation (commotio cordis).

Role of pre-participation cardiovascular screening (PPS)

The majority of cardiac conditions implicated in SCD may remain asymptomatic and SCD is often the first presentation. Although there is a strong genetic component to most implicated cardiac disorders, a family history is identified in <25% of cases, probably because event rates in affected sedentary individuals are low.

Therefore, the most practical method for identifying young athletes at risk of potentially fatal cardiac diseases is by performing PPS in apparently healthy individuals.

The potential for preventing SCD in young athletes by abstinence from moderate to severe exercise, pharmacotherapy or implantation of cardiovertor defibrillators has prompted the medical and sporting community to recommend PPS to allow early identification of at-risk individuals. Both the American Heart Association (AHA) and the European Society of Cardiology (ESC) advocate cardiovascular screening of young athletes on ethical, legal and medical grounds [8, 9].

Current screening protocols and role of 12-lead ECG as a screening tool

An efficient screening programme should be relatively cheap and be able to identify most affected individuals whilst generating a low false negative rate [10]. Two separate models have been in practice in the US and Italy for over 30 years.

American screening protocol

The American Heart Association (AHA) screening model includes a 12-point screening protocol encompassing history relevant to cardiac symptoms, past cardiac history and family history of premature cardiac disease/death, and physical examination [11], which is aimed at identifying hypertension, valvular heart disease, co-arctation of aorta and Marfan syndrome (table 1). This approach to PPS seems pragmatic but lacks sensitivity because the majority (>80%) of individuals harbouring cardiovascular disease implicated in SCD are asymptomatic and fail to exhibit abnormal findings during physical examination. This is illustrated in a report by Maron et al. [6] of 134 cases of sudden cardiac deaths in the US in whom screening with history and physical examination led to a suspicion of an underlying cardiac disorder in only 3% cases, and to an accurate diagnosis in <1% of those evaluated. Wilson et al. [12] showed that use of health questionnaires alone when screening for cardiac disorders implicated in exercise-related SCD results in a low diagnostic yield; in a British cohort of over 2700 young athletes and physically active school children undergoing PPS using 12-lead ECG, 9 individuals were diagnosed with a cardiovascular disorder implicated in SCD however none of them were symptomatic or had a family history of note.

Table 1
The 12-element AHA protocol for pre-participation screening of competitive athletes. (Reproduced and modified from [8]: Maron BJ, Thompson PD, Ackerman MJ, Balady G, Berger S, Cohen D, et al. Recommendation and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update. Circulation. 2007;115:1643–55. © Wolters Kluwer Health, Baltimore, USA. Reprint with kind permission.)

12-element AHA recommendations for preparticipation cardiovascular screening of athletes
Medical history
Personal history
1. Exertional chest pain
2. Unexplained syncope / near-syncope
3. Excessive exertional dyspnea of fatigue
4. Prior recognition of heart murmur
5. Hypertension
Family history
6. Premature cardiac death (age <50)
7. Disability from cardiac disease in a close relative
8. Family history of certain cardiac conditions (hypertropic or dilated cardiomyopahty, long QT syndrome or other ion channelopathies, Marfan syndrome, etc.)
Physical examination
9. Heart murmur
10. Femoral pulse to exclude coarctation of aorta
11. Physical stigmata of Marfan syndrome
12. Brachial artery blood pressure

ARVC = arrhythmogenic right ventricular cardiomyopathy; CAD = coronary artery disease; HCM = indicates hypertrophic cardiomyopathy; LAD = left anterior descending artery; MVP = mitral valve prolapse.

Italian screening protocol

In Italy, individuals engaged in official competitive sports are required to undergo mandatory cardiovascular screening. The Italian model incorporates 12-lead ECG in conjunction with a health questionnaire and physical examination. Athletes with abnormalities on initial evaluation are subject to further investigations to confirm or refute the presence of a potentially serious or deleterious cardiac disorder (fig. 2). Athletes diagnosed with cardiac disorders are managed according to established guidelines which often recommend abstinence from competitive sport other than low static to low dynamic disciplines [13, 14].

In addition to permitting the diagnosis of inherited ion channelopathies and congenital accessory pathways, the 12-lead ECG is invaluable in the identification of marked repolarisation changes which are commonly found in primary cardiomyopathies. The ECG is abnormal in up to 90% cases of HCM and up to 80% cases of ARVC, thus improving the diagnostic yield for a spectrum of electrical or structural cardiovascular disorders. The efficacy of ECG in the diagnosis of HCM has been demonstrated by the Italian experience. In a large population-based study of screening outcomes in 33 735 athletes, 621 (1.8%) were disqualified because of identification of a cardiovascular disorder. Of these, 22 (0.7%) were considered to have HCM predominantly (80%) on the basis of an abnormal ECG [7]. This frequency is similar to that observed in a population-based study in the US using echocardiography, suggesting that an ECG-based screening model is as effective as echocardiography in detecting HCM [15].

The incremental role of ECG in the diagnosis of cardiac disorders during PPS was also demonstrated by

Baggish et al. in a cross-sectional study of collegiate athletes [16]. In a cohort of 510 athletes, 11 were diagnosed with cardiac disease; history and examination identified only 5 of the 11 individuals whereas additional ECG testing increased the diagnostic yield to 10 out of 11 (sensitivity of 91% with ECG compared to 45% without) and a specificity of 83%.

Figure 2
Italian pre-participation screening model endorsed by European Society of Cardiology, International Olympic Committee and Fédération Internationale de Football Association. (Reproduced from [9]: Corrado D, Pellicia A, Bjornstad HH, Vanhees L, Biffi A, Borjesson M, et al. Cardiovascular preparticipation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol. Eur Heart Journal. 2005;26:516–24. © Oxford University Press, Oxford, UK. Reprint with kind permission.)

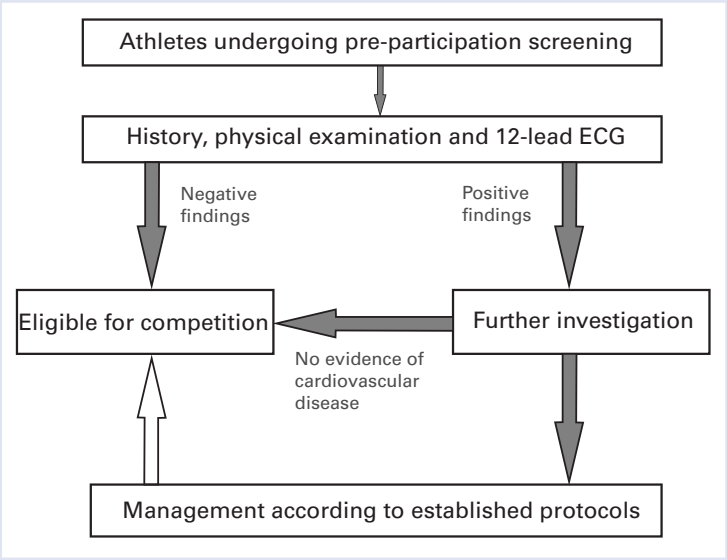


Table 2
European Society of Cardiology (ESC) classification of ECG abnormalities in athletes. (Reproduced and modified from [19]: Corrado D, Pellicia A, Heidbuchel H, Sharma S, Link M, Basso C, et al. Recommendations for interpretation of 12-lead electrocardiogram in athletes. Eur Heart J. 2010;31:243–59. © Oxford University Press, Oxford, UK. Reprint with kind permission.)

ESC classification of ECG abnormalities in athletes	
Group 1 (training-related)	**Group 2 (training-unrelated)**
Sinus bradycardia	T-wave inversions
First degree AV block	ST-segment depression
Incomplete RBBB	Pathological Q-waves
Early repolarisation	Left atrial enlargement
Isolated QRS voltage criteria for LVH	Left axis deviation / left anterior hemiblock
	Right axis deviation / left posterior hemiblock
	Right ventricular hypertrophy
	Ventricular pre-excitation
	Complete LBBB or RBBB
	Long-QT or short-QT interval
	Brugada-like early repolarization

LVH = left ventricular hypertrophy; LBBB = left bundle branch block; RBBB = right bundle branch block.

False positive rates and updates in guidelines

The participation of a young individual in regular physical exercise is associated with electrical and structural modifications which occasionally overlap with incomplete or mild phenotypic expressions of cardiomyopathies or ion channel disorders. The diagnostic conundrum is most commonly encountered during the identification of voltage criteria for chamber enlargement and early repolarisation changes on the ECG, resulting in unnecessary investigations and the potential of erroneous disqualification from competitive sports.

Several studies of athletes have revealed a high prevalence of false positive tests, based predominantly on the expression of QRS voltage criteria for left ventricular hypertrophy (LVH). False positive rates associated with ECG range between 10 and 30% [17], and have denigrated the role of ECG and fuelled arguments against its inclusion in PPS programmes [18].

Based on large population studies in a non-select group of athletes, the ESC guidelines for ECG interpretation in young athletes were revised in 2010 and ECG changes in athletes were categorised into 'group 1' which are likely to result from cardiac adaptation to exercise, and 'group 2' which are unlikely to be exercise-related and possibly representative of cardiovascular disease (table 2). In these updates, isolated voltage criteria for LVH was reclassified as a normal finding in athletes and is now included amongst the 'group 1' changes therefore reducing the need for any further evaluation [19]. In retrospect, the initial inclusion of voltage criteria for LVH as a marker of abnormality might be considered naïve since this has been regarded as a common normal variant in young athletes for many years [3]. A study by Weiner et al. comparing the performance of the 2010 ESC guidelines against the 2005 ESC recommendations in a cohort of 508 US collegiate athletes resulted in a significant reduction of false positive rates from 16 to 10%, without compromising sensitivity [20]. This reduction was solely due to the reclassification of QRS voltage criteria for LVH from abnormal to normal.

The false positive rates are likely to be even lower when athletes are evaluated in an expert setting as documented in a cohort of 2720 junior athletes and physically active children [12].

The impact of ethnicity on cardiovascular adaptation in athletes has not been studied in detail, however, there is emerging data that ECG changes including repolarisation changes similar to those seen in cardiomyopathies are more commonly seen in athletes of Afro-Caribbean origin [21–23]. A recent study on 904 black athletes with ECG and echocardiography demonstrated a significantly higher proportion of repolarisation changes, including T-wave inversions in up to 25% and ST-elevation in two-thirds of athletes [23]. Preliminary follow-up data of approximately 7 years indicates that T-wave inversions in leads V_1–V_4, which are observed in up to 13% of black ath-

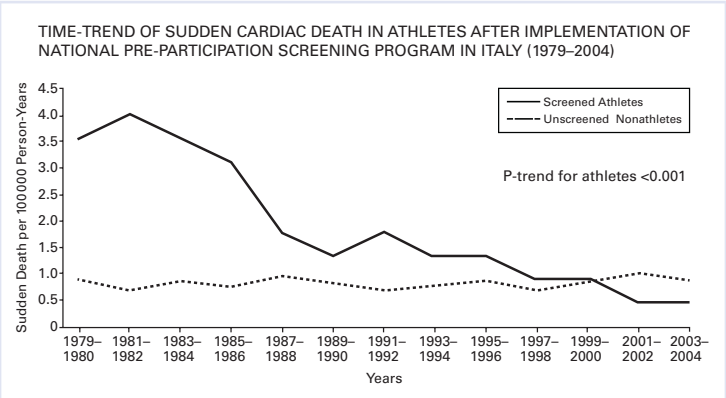
letes, are benign; however, the significance of T-wave inversions in the inferior and lateral leads is uncertain. Such variations in ECG changes in athletes of different ethnicities will have an impact on wide-scale PPS programmes in countries with multi-ethnic populations, and further studies in non-Caucasian athletes are required.

Is pre-participation screening with ECG effective in reducing sudden cardiac death in athletes?

A prospective longitudinal study evaluating the trends in SCD over a 25-year period in Italy has revealed a significant decrease in the incidence of SCD, from 3.6/100 000 per year to 0.4/100 000 per year, representing almost a 90% reduction (fig. 3). The decline in death rates was primarily from the identification and disqualification of individuals with HCM and ARVC [24]. The study was not a randomised trial raising concerns regarding reduced mortality solely being a consequence of the screening process. However, the strong cause–effect relationship between ECG screening and reduction in SCD was supported by the following findings: (1.) coincident timing between implementation of PPS and reduction in SCD; (2.) reduction of SCD was due to fewer deaths from cardiomyopathies (HCM and ARVC) and it was accompanied by an increase in the number of athletes identified with these conditions leading to disqualification; and (3.) during the screening period there was no change in the incidence of SCD in the unscreened non-athletic population.

The Italian screening model including ECG is endorsed by the European Society of Cardiology (ESC) and provides the fundamental basis for the common European protocol for prevention of sudden death in young athletes [9]. In addition the model has also been advocated by major sporting bodies including the International Olympic Committee [25], Fédération Internationale de Football Association (FIFA) and Union of European Football Associations (UEFA).

Figure 3
Annual incidence of sudden cardiac death in screened competitive athletes compared with unscreened non-athletes in Veneto region of Italy. (Reproduced from [24]: Corrado D, Basso C, Pavei A, Michieli P, Schiavon M, Thiene G. Trends in sudden cardiovascular death in young competitive athletes after implementation of a preparticipation screening program. JAMA. 2006;296:1593–601. © American Medical Association, Chicago, USA. Reprint with kind permission.)



Limitations of ECG as a screening tool

Whereas the ECG is effective in the identification of electrical disorders and primary cardiomyopathies, it is unable to detect coronary artery anomalies and premature coronary artery disease (CAD). Furthermore, a 12-lead ECG may fail to detect ARVC with incomplete phenotypic expression, concealed cases of Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia (CPVT) and congenital long QT syndrome due to variable expression. Nevertheless the ECG fulfils its main role in screening which is to identify the vast majority of cardiovascular abnormalities with few false negatives.

Psychological impact of screening

There is a conspicuous lack of published literature on psychological impact of PPS in athletes. It is reasonable to assume that athletes with false positive results will be anxious until reassured after further evaluation. However, a recent study on Norwegian football players assessed the level of distress amongst players during screening. The results suggest that almost two-thirds of athletes felt more confident when playing, and 88% were satisfied after completing screening. Approximately 16% athletes were apprehensive about the outcome of screening, and 13% were worried about disqualification. Less than 3% experienced distress during PPS [26].

Cost-effectiveness of pre-participation screening

Cost implications and unnecessary investigations due to false positive results are the drivers for the antagonists for PPS [18]. The American screening model, based on history and physical examination has a low diagnostic yield. A cost-effectiveness analysis of a prospective study involving 5615 high school athletes in the US, compared history and physical examination with ECG screening, and showed that inclusion of the ECG was more cost-effective, amounting to $44 000 per life-year saved compared to $84 000 per life-year saved with history and examination alone [27]. Similarly, Wheeler et al. analysed the cost-effectiveness of PPS and reported that the addition of ECG to history and physical examination was calculated to save 2.06 life-years per 1000 athletes at an incremental total cost of $89 per athlete and yielded a cost-effectiveness ratio of $42 900 per life-year saved compared with history and physical examination alone [28]. A more recent cost-effectiveness analysis of screening in 1473 US intercollegiate athletes demonstrated an improved diagnostic yield with addition of ECG to history and physical examination. The ECG uncovered sig-

nificant cardiac pathologies which were not identified with history and physical examination alone. The cost of history and physical examination per abnormal finding was $68 745 and the addition of ECG was associated with a marginal increase in cost to $68 893 [29]. These studies provide support that PPS using 12-lead ECG plus history and physical examination may be more cost-effective than initially assumed.

How and when to screen (personal perspective)

Data from Italian publications suggest that pre-participation screening has an important role in preventing exercise-related SCD. In a benevolent society, PPS should be offered to all young athletes participating in competitive sports as per AHA and ESC recommendations. However, there are numerous issues that need to be resolved before a de-novo screening programme can be established in other Western countries (table 3).

The authors believe that the Italian model with ECG is the most effective and has a relatively high diagnostic yield. The age of examination is an important factor in the diagnosis of cardiomyopathies. We recommend screening to be performed at the age of 14 onwards since most cases of HCM do not manifest until puberty. Conversely many young athletes aged less than 14 exhibit inverted T-waves beyond V_1 as part of the normal juvenile pattern raising unnecessary concerns about ARVC.

The authors recommend bi-annual evaluation up to the age of 21, with on-going evaluation beyond this age only in those with family history of inherited cardiac disorder or previous history of SCD in first-degree relatives. Screening should be conducted in an expert setting, by trained sports cardiologists with experience of ECG interpretation in athletes. In addition to voltage criteria for LVH, there is no evidence that isolated axis deviation or voltage criteria for atrial enlargement are of any significance and should not be considered as an indicator for further assessment in the absence of symptoms. Similarly, the presence of T-wave inversions confined to V_1–V_4 in Afro-Caribbean athletes should be considered to represent a normal ethnic variant [19, 30].

It is the authors' view that screening should not be mandatory and disqualification should not be enforced as this may be viewed as an infringement of human rights. In circumstances where there is an ongoing diagnostic dilemma, the athlete should not be labelled with a cardiac diagnosis but should be under careful annual surveillance.

Table 3
Issues regarding pre-participation screening in athletes.

Issues regarding screening in athletes
• WHO to screen?
• WHEN to screen?
• HOW often to screen?
• WHICH screening protocol?
• WHO will screen?
• WHO will pay for screening?
• WHERE is the infrastructure, personnel and expertise?
• WHO will manage the athlete with a diagnosis?

Pre-participation screening in middle-aged leisure athletes

Atherosclerotic coronary artery disease (CAD) is by far the leading cause of exercise-related SCD in individuals aged ≥35 years. The prevalence of SCD in middle-aged/senior individuals is variable, ranging from 1/50 000 to 1/100 000 in marathon runners in their mid-40s to 1/35 000 in recreational Rhode Island joggers aged 30–64 years [31]. A significant number of victims have established risk factors for CAD. A resting ECG has a low diagnostic yield for detecting silent CAD. Conventional exercise testing (ETT) will identify athletes with the most severe disease, but exhibits low sensitivity and specificity in asymptomatic individuals [32, 33]. Based on these facts, we recommend detailed assessment in symptomatic middle-aged athletes or those with risk factors. It is noteworthy that a negative ETT should not provide reassurance in individuals planning to compete in vigorous and sustained exercise, as it will only reveal ischemia in presence of luminal stenosis of >60%. Many middle-aged individuals have soft non-obstructive plaque that may rupture and cause fatal myocardial infarction whilst exercising. Ideally, CT coronary calcium is probably the best method to identify silent CAD, however, such a strategy is currently limited due to the practical inability to readily perform such an investigation in large numbers of individuals.

Alternative strategies (automated external defibrillator)

An ECG cannot identify premature coronary artery disease and anomalous coronary arteries during PPS. Athletes may also be at risk of SCD due to blunt trauma to the chest (commotio cordis), and from other acquired conditions including electrolyte imbalance, heat stroke, myocarditis and use of performance-enhancing drugs. Since the majority of cardiac arrests in athletes manifest as ventricular fibrillation, the community-based portable automated external defibrillators (AED) have emerged as an alternative strategy for secondary prevention against SCD. The most important factors in survival after cardiac arrest are the time from cardiac arrest to delivery of shock and adequate cardiopulmonary resuscitation with correct cardiac compression until AED arrives. Public access AED programmes aim to reduce this collapse-to-shock time to less than 5 minutes. A survey of 1710 US high schools with on-site AED programme demonstrated a survival benefit with early defibrillation. Of the 36 cases of cardiac arrest observed amongst young

students and older non-students, 64% survived to hospital discharge with a mean collapse-to-shock time of 3.6 minutes [34]. Public access to AED as a part of emergency response planning (ERP) has shown to be effective, and major sporting and cardiac organisations recommend implementation of such programmes in all sporting venues and fitness centres [35].

Conclusion

Pre-participation cardiovascular screening including ECG is effective in identifying cardiac disorders in young athletes, and has resulted in a reduced incidence of SCD. Major sports and cardiac organisations advocate PPS. Recent studies suggest that false positive rates are lowered in an expert setting and ECG screening is more cost-effective than contemporary evaluation with health questionnaire and physical examination alone.

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