Open Access Journal of Sports Medicine

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REVIEW

Cardiac Screening in Athletes: Current Status and Future Challenges

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Abstract: Pre-participation screening (PPS) is a primary-prevention strategy used in competitive sports to identify athletes at risk for cardiac diseases that can present with malignant arrhythmias and sudden cardiac death (SCD). Although compelling evidence supports the ability of PPS to lower SCD rates among athletes, no universal protocol has been adopted. Over time, however, key elements have emerged as common denominators: medical history, physical examination, and a 12-lead electrocardiogram (ECG). Research on ECG interpretation continues to evolve in search of the most accurate criteria for detecting cardiac disease, yet significant challenges persist. A major difficulty is the overlap between physiological remodeling in well-trained hearts and early manifestations of cardiomyopathy. In addition, some subgroups (female athletes, master athletes, and pediatric athletes) remain underrepresented in current studies. To ensure broad and equitable implementation, PPS must be supported by public-health policies and recognized as cost-effective, with the expectation that early identification of at-risk individuals will ultimately reduce the burden of advanced treatments on healthcare systems. Achieving this goal requires improving the sensitivity and specificity of existing screening tools to minimize misdiagnosis and avoid unnecessary secondary investigations or unwarranted disqualification from competition. This review summarizes the current status of cardiac screening in athletes, highlights persistent challenges, and outlines future directions to enhance its effectiveness and long-term sustainability.

Keywords: preparticipation evaluation, sudden cardiac death, electrocardiography, sports cardiology, risk stratification, cost-effectiveness

Introduction

Screening is a preventive medical strategy that aims to identify subjects at risk for specific diseases before clinical manifestation of them, with the aim of enabling early treatment, shortening recovery time, and improving outcomes. Nevertheless, an ideal test of screening should balance sensitivity and specificity, in order to minimise the risk of false positives.[1]

In the context of young competitive athletes, this measure is mandatory as some cardiovascular diseases (CVDs) can be triggered by physical effort, with the first onset sometimes being malignant arrhythmias.[2] Both the European Society of Cardiology (ESC) and the American Heart Association (AHA) agree that systematic cardiovascular evaluation of competitive athletes is ethically and medically justified.[3,4] The rationale behind pre-participation screening (PPS) lies in the possibility of preventing sudden cardiac death (SCD) through targeted intervention such as lifestyle modifications, reduction of trigger factors, or adoption of prophylactic measures like implantation of a cardiac defibrillator to prevent or treat malignant arrhythmias. Moreover, recent experience with SARS-CoV-2 infection in athletes has contributed to refining contemporary approaches to cardiovascular risk assessment in sport, emphasizing the importance of proportionate, evidence-based cardiac screening to support safe sport participation and informed clinical decision-making.[5–7] This review provides a critical appraisal of existing cardiac screening strategies in competitive athletes, addressing unresolved issues in electrocardiogram (ECG) interpretation, special populations, and healthcare sustainability, while outlining priorities for future improvements.

https://doi.org/10.2147/OAJSM.S523274
Received: 24 September 2025
Accepted: 3 February 2026
Published: 19 February 2026

Epidemiology of Sudden Cardiac Death in Athletes

Sudden cardiac death (SCD) is an emotionally and socially impactful event, particularly when it concerns young people, as these individuals appear healthy. Although rare, its reported incidence in the literature ranges widely, from 0.2 to 6.8 cases per 100,000 athlete-years.[8–11]

Different factors could impact on individual susceptibility to SCD, such as sex, ethnicity, age or type of sport practiced. With regard to sex, studies consistently indicate that male athletes have a two- to ten-fold higher risk of sudden cardiac arrest (SCA) and SCD compared to female athletes.[11–14] It is probably due to the higher training loads, more pronounced cardiac remodelling, and possible different disease expression.

Ethnic disparities have also been documented: Black athletes, as compared to white and Asian ones of same age and sex, are subject to a five-fold increased risk of events. Indeed, in a large United Kingdom (UK) registry, it has been reported that cardiomyopathies are more common in Black athletes, particularly arrhythmogenic cardiomyopathy (ACM) with a higher predominancy of a biventricular pattern in this subpopulation.[15]

Furthermore, the risk of SCD increases with age, as different etiologies can be observed: for example, in master athletes (MAs, older than 35 years), the main cause, though not exclusive, of SCD is coronary artery disease.[16,17] Instead, in younger athletes, cardiomyopathies and congenital heart defects are more prevalent.[18] About this, over time the epidemiology of SCD has evolved, likely due to the implementation of PPS and improved detection of early-stage CVDs, and while historically hypertrophic cardiomyopathy (HCM) accounted for over one-third of SCD cases in the U.S.[19] and arrhythmogenic right ventricular cardiomyopathy (ARVC) was responsible for approximately one-fourth of cases in the Veneto region of Italy,[10] more recent studies have identified idiopathic left ventricular hypertrophy and idiopathic left ventricular fibrosis as increasingly frequent causes of SCD:[20] in particular, the so-called isolated non-ischemic left ventricular scar (NILVS) has been recognized as a critical substrate for sustained ventricular tachycardia (VT)[21] and as the most common identifiable cause of sports-related SCD.[22]

The type of sport also impacts the risk of SCD, given that the training load and the aerobic component can vary greatly between different activities: certain sports, including football, basketball, American football, and cycling, have also been linked to an increased risk of SCA and mortality for unclear reasons.[11]

In addition, improved molecular insights and post-mortem analyses have revealed that a subset of athletes dying with a structurally normal heart actually harbour primary electrical disorders, including long and short QT syndromes, Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia (CPVT).[13,17,23] At last it deserves to be mentioned, the phenomenon known as “commotio cordis”, that is a blunt, non-penetrating blows to the precordium that may alter the electrical stability of the myocardium, triggering ventricular fibrillation (VF) without structural injury to the ribs, sternum, or heart itself, and that can be a real issue in a dynamic context as a playing field.[24]

State of the Art of Preparticipation Screening

There is a substantial divergence between European and American guidelines concerning the specific PPS protocols recommended by cardiologists and sports medicine specialists.[25–28] Moreover, the resources available across countries vary considerably, reflecting differences in healthcare systems and the level of economic funding allocated.[29]

Historically, the AHA recommended that competitive athletes undergo annual PPS with an assessment based on 14 key elements including targeted personal and family history and a physical examination. It was demonstrated a limited power (<10%) of the AHA model to detect potentially lethal cardiovascular abnormalities,[4,30] because of the latency of symptoms in certain cardiac conditions.

Over the years, the European Football Union (UEFA), FIFA and the International Olympic Committee (IOC) have all recommended including a 12-lead ECG in the PPS.[31–33] Indeed, ECG pathological features, as repolarisation abnormalities, indirect signs of overload, pre-excitation patterns or pathognomonic patterns, may raise suspicion of underlying arrhythmogenic substrate such as cardiomyopathies, pre-excitation syndromes or cardiac ion channel disorders.[3] Williams et al conducted a prospective study on high school athletes (13–19 years old) to compare the effectiveness of the AHA 14-element screening versus 12-lead ECG interpreted with the Seattle Criteria.[34] The AHA 14-point evaluation identified abnormalities in 43.8% of athletes with an SCD-risk condition.

In contrast, the ECG detected 93.8% of these cases, emphasizing the superior ability of ECG to identify athletes with cardiovascular conditions.[27] In a large Italian study involving 22,324 young athletes undergoing PPS, a condition associated with SCD risk was identified in 29% of them by a positive family history, symptoms, and/or abnormal physical examination, while in 59% by abnormalities on a resting 12-lead ECG.[2]

Recently, the AHA and the American College of Cardiology (ACC) published a scientific statement that marks a significant shift in the debate on ECG screening in athletes.[28] While the AHA previously opposed mandatory ECG screening due to concerns about logistics, manpower, financial burden, and false positives, the new scientific statement acknowledges the benefits of ECG in detecting life-threatening cardiac conditions, underlining how, with expert interpretation, it is possible to cut down misdiagnoses. This change reflects growing evidence that ECG screening significantly improves the detection of at-risk athletes.

The clinical impact of such programs is supported by long-term population data. In the Veneto region of Italy, for example, the introduction of mandatory nationwide PPS was followed by an almost 90% reduction in SCD among young competitive athletes, while rates in unscreened individuals remained stable.[10] Although observational in design, the close temporal association between the start of screening, the rise in early diagnoses of conditions such as ARVC and HCM, and the subsequent fall in fatal events provides strong evidence of a protective effect.

The sensitivity of PPS to detect CVDs is influenced by the prevalence of these conditions in the athletic population: available data suggest that the prevalence of CVDs responsible for SCD ranges from 0.04% in young athletes aged 8–15 years to a maximum of 0.7% in individuals aged 12–35 years.[2,10,13,35–37] However, in older athletic populations, the prevalence increases significantly, to 11.8% in MAs aged 35–81 years.[38–40]

Pitfalls of Athlete's ECG

Interpreting an athlete's ECG requires dedicated criteria, as exercise-induced cardiac remodeling can mimic changes seen in cardiomyopathies, making differential diagnosis challenging. Since the first European classification in 2000,[41] subsequent updates, most notably the ESC criteria of 2010, the Seattle Criteria (2013),[34] and the Refined/CRY update (2014),[36] have progressively improved accuracy, culminating in the current International Criteria of 2017.[42] These guidelines group ECG findings into three categories: normal, reflecting training-related adaptations that require no further testing when consistent with the athlete's sport, exercise intensity, and absence of symptoms; borderline, changes that may occasionally be associated with pathology and warrant investigation when two or more appear on the same tracing; and abnormal, patterns not attributable to athletic remodeling and therefore requiring systematic evaluation to exclude underlying disease.

A further revision is now being planned in response to the growing body of new evidence. Recent studies, for example, have questioned the fixed 16-year threshold for T-wave inversion (TWI), suggesting pubertal status as a more reliable marker;[43–45] highlighted the diagnostic importance of low QRS voltages, rare in athletes but often linked to cardiomyopathy;[46,47] and indicated that even a single premature ventricular beat in an ECG trace might merit investigation, rather than the two-beat threshold set by the current criteria.[48] Moreover, although the criteria are formally intended for athletes aged 12–35, they are largely derived from studies in young males, raising concerns about their validity in other groups. In children and adolescents, incomplete maturation and lower training loads increase the risk of misclassification,[45,49] while MAs over 35 show distinct exercise-related remodelling (according to the age at which they start practising intense sport)[50] and face a higher risk of coronary artery disease without dedicated ECG standards. Female athletes are likewise underrepresented in validation studies and, despite similar training levels, display fewer structural and electrical adaptations, with anterior TWI more often a benign finding.[51,52] Together, these observations reinforce the need for future updates to incorporate age- and sex-specific interpretation criteria and tailored screening strategies.

Screening Resource Challenges

While the clinical benefit of PPS is well established, worldwide implementation remains disparate. The main barriers are organizational and economic: healthcare systems differ widely in their ability to fund large-scale screening, and costs must be balanced against other public health priorities. Italian data show that a nationwide, publicly funded program can be delivered at a relatively modest per-athlete cost, whereas estimates from the United States project a much higher

 https://doi.org/10.2147/OAJSM.S523274

financial burden in a largely private healthcare context. These discrepancies highlight the need for strategies that ensure equitable access and sustainability, making the economic dimension itself one of the key future challenges of cardiac screening.[53,54]

In low- and middle-income countries, where universal ECG-based PPS may not be financially or logistically feasible, alternative and scalable organizational models have been suggested to improve access. These include task-shifting approaches in which trained non-physician healthcare providers are involved in initial screening steps, with referral of abnormal findings to specialist centers.[55] However, in the specific context of athlete screening, cost-effectiveness is critically dependent on the accuracy of ECG interpretation.[56] Reducing false positives and avoiding unnecessary downstream investigations requires both expertise and continuous training of the interpreting physician. Accordingly, any application of alternative organizational models should be approached with caution, as reduced interpretative expertise may paradoxically increase false-positive rates and overall costs.[56] In this context, telemedicine-based ECG interpretation hubs, allowing centralized expert review of tracings acquired locally or through mobile ECG services, represent a potentially promising strategy to preserve diagnostic accuracy while improving access.[57] Emerging technologies, including AI-assisted ECG analysis, may further support such models in the future, although their role in athlete screening has not yet been sufficiently validated.[58] Alongside primary prevention strategies, investment in secondary prevention measures, particularly the widespread availability of automated external defibrillators (AEDs) and cardiopulmonary resuscitation (CPR) training in sports environments, represents a highly effective and immediately implementable approach to reduce sports-related mortality, especially in settings where economic or organizational constraints limit the development of comprehensive screening programs and where these life-saving skills can be disseminated across the general population.[59]

Secondary Prevention

Even the best screening programs cannot identify every individual at risk, as some lethal arrhythmogenic disorders present with a structurally normal heart or remain clinically silent. For this reason, secondary prevention is essential to limit the consequences of unexpected cardiac arrest. Over the last two decades, the combination of widespread availability of AEDs and well-rehearsed emergency action plans has proven lifesaving.[59] In US collegiate athletes, a 70% reduction in SCD rates has been linked to improved access to AEDs and standardized CPR protocols, while in Italy the “Balduzzi law” markedly increased the presence of AEDs in sports facilities and boosted survival after out-of-hospital cardiac arrest.[13] International registries, including the FIFA Sudden Death Registry, consistently report far higher survival when defibrillation is provided promptly on site.[60,61]

These findings reinforce that primary and secondary prevention are complementary: systematic screening can identify many high-risk individuals before symptoms arise, while rapid response systems, public access defibrillation, trained personnel, and clear emergency pathways provide a critical safety net for those who inevitably escape detection.

Conclusion

Cardiac screening of athletes has evolved from simple history and physical examination to structured PPS programs with expert ECG interpretation, demonstrating a clear capacity to identify individuals at risk for SCD and, when implemented at a population level, to reduce mortality. Nevertheless, important challenges remain. Diagnostic criteria require further refinement to account for pubertal development, sex, ethnicity, and the growing participation of MAs. Equitable access and long-term sustainability, particularly in countries with limited healthcare resources or predominantly private systems, are additional barriers that must be addressed. Finally, no screening strategy can eliminate all risk, underscoring the essential role of secondary prevention through widespread availability of AEDs, trained responders, and robust CPR protocols.

An integrated approach that combines high-quality primary PPS with effective secondary prevention offers the best path to safeguarding athlete health while meeting the practical realities of diverse healthcare settings. Despite the progress achieved, substantial work is still required to refine criteria, expand accessibility, and ensure consistent global implementation of effective screening strategies.

Abbreviations

ACC, American College of Cardiology; ACM, Arrhythmogenic cardiomyopathy; AED, Automated external defibrillator; AHA, American Heart Association; AI, Artificial intelligence; ARVC, Arrhythmogenic right ventricular cardiomyopathy; CPR, Cardiopulmonary resuscitation; CPVT, Catecholaminergic polymorphic ventricular tachycardia; CVD, Cardiovascular disease; ECG, Electrocardiogram; EHRA, European Heart Rhythm Association; ESC, European Society of Cardiology; FIFA, Fédération Internationale de Football Association; HCM, Hypertrophic cardiomyopathy; IOC, International Olympic Committee; LV, Left ventricle/ventricular; MA, Master athlete; NILVS, Non-ischemic left ventricular scar; PPS, Pre-participation screening; RV, Right ventricle/ventricular; SCA, Sudden cardiac arrest; SCD, Sudden cardiac death; TWI, T-wave inversion; UEFA, Union of European Football Associations; UK, United Kingdom; US, United States; VF, Ventricular fibrillation; VT, Ventricular tachycardia.

Disclosure

The authors report no conflicts of interest in this work.

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